

Expert Panel Discussion

Prehypertension—What Is It and Should It Be Treated?

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In May 2006, a panel of experts was assembled during the meeting of the American Society of Hypertension, Inc to discuss the diagnosis and treatment of prehypertension. Is it a real entity? Does it pose a risk? Should it be treated entirely with lifestyle interventions or should some patients be placed on pharmacologic therapy? The panel was chaired by Dr Marvin Moser, Clinical Professor of Medicine at the Yale University School of Medicine, New Haven, CT, and Editor in Chief of The Journal of Clinical Hypertension. Panelists included Dr Thomas Giles, Professor of Medicine at Louisiana State University, New Orleans, LA; Dr Joseph Izzo, Jr, Professor of Medicine at SUNY Buffalo School of Medicine, Buffalo, NY; and Dr Henry Black, Roberts Professor of Preventive Medicine at the Rush University School of Medicine in Chicago, Chicago, IL. (J Clin Hypertens. 2006;8:812–818) ©2006 Le Jacq

DR MOSER: Let me start first by asking Dr Giles, the immediate past president of the American Society of Hypertension, Tom—what is prehypertension? If you have it, are you really at increased risk for a cardiovascular (CV) event?

DR GILES: Prehypertension is defined as a systolic blood pressure (BP) range from 120 to 139 mm Hg and a diastolic BP between 80 and 89 mm Hg. It was a classification made in the Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7). This was based on the fact that people who have BPs in that range are at a greater risk for CV disease than people with BPs <120/80 mm Hg. This concept was based on the Lewington study that was published in *The Lancet* a few years ago that reported a continuous linear relationship between BP and CV risk starting around a level of about 115/75 mm Hg and data from the National Health and Nutrition Examination Survey (NHANES) and Framingham study. The concept I believe alerted the practicing physician to the fact that people whose systolic BPs were in the range of 120–139 mm Hg should be examined more carefully because they might, in point of fact, be at some increased risk.

DR MOSER: Tom, please clarify this. How much is the risk increased in a person who is thin, has no other CV factors, and has a BP of 132/82 mm Hg, compared with someone with a pressure of 120/80 mm Hg.

DR GILES: The first factor, of course, is age. If the person you just described is 80 years old and the person with whom they are being compared is 40, then the relative risk is probably 30–40 times higher. So there are factors other than BP that are important. One can look lean but have a visceral fat increase, in which case they would be at increased risk. Or, one might have certain other biomarkers or some genetic predisposition for cardiac disease—like a family history. So what I'm saying is that in people with BPs from 120–139 mm Hg, the degree of CV risk probably has to be assessed in a global way and will not be dependent on threshold values for any one parameter, including BP, blood glucose, or blood lipids.

DR MOSER: Henry, the British Hypertension Society said that JNC 7 was wrong and that BPs designated as prehypertension might not pose an added risk and might be normal in a person with no other risk factors. As Tom noted, there are some hidden risk factors, but without other obvious risk factors, such as dyslipidemia, an abnormal blood



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glucose level, and obesity, are these people at great enough risk to warrant labeling and intervention? Prehypertension in a middle-aged person like this may be normal, but prehypertension in a 45-year-old obese person with diabetes might have stage 1 hypertension and a totally different prognosis. Do you distinguish that or are we going to use the term prehypertension based on numbers?

DR BLACK: I certainly understand the issue. When the JNC 7 reclassified hypertension, it was to clarify the previous complex definitions. We were impressed with the Lewington data, to which Dr Giles alluded. Not only was risk gradually increased for BP levels from 115/75 mm Hg, but when they adjusted for other risk factors (lipids, glucose, diabetes, weight), that risk remained. The increase in risk associated with a rising BP, even in the prehypertensive range, was the same for all ages but, of course, age is probably the most significant risk factor and one we cannot reverse.

So the issue is, as I see it, how to translate epidemiologic information from more than a million subjects followed on average for 12 years into useful recommendations for providers of care and their patients. The designation that someone is prehypertensive means only that. It doesn't necessarily mean that therapeutic interventions are justified. We do not have a trial that has demonstrated that reducing BP in people in the prehypertensive BP range will reduce their event rates. We need that to really validate any decision to treat them, especially with drugs. In fact, we don't even have a trial in people with systolic BPs from 140–149 mm Hg that validates the need to treat them. At present, however, treating hypertension in the lower levels of stage 1 just seems to make sense.

DR MOSER: So your belief is that the BP level alone, even in the absence of other recognizable risk factors, is enough to indicate that a patient is at increased risk and that they have prehypertension. Is that fair?

DR BLACK: Yes, compared with someone whose BP is lower, and other things being equal. Now, you talked about what both the Europeans and British said about JNC 7. JNC 7 did not stratify risk as in JNC VI. Because drug treatment was recommended for everyone with consistent and validated BPs above 140/90 mm Hg, there was no need for such stratification since it was not changing therapeutic recommendations. Prehypertensives were not stratified in JNC 7 and perhaps they should have been. My preference now would be to take the A, B, and C stratification that was used in JNC VI and apply it to the prehypertensive range

of BPs. But it should always be remembered that classification and stratification systems should be evolutionary, not revolutionary. Making changes that are too drastic will not be adopted by practicing physicians and other clinicians. I believe that we ought to take the prehypertensives whose risk is clear and classify them in a way that doctors can use. So if you are classified as having *prehypertension stage A* (no risk factors or target organ damage), one set of treatment recommendations would emerge. If you have *prehypertension stage B* (there are other risk factors present) or *prehypertension stage C* (with demonstrable target organ damage or CV events), a different set of treatment recommendations would be appropriate. Using the term prehypertension in JNC 7 was designed to get the attention of the public and providers. It worked. Now it will be important to further clarify that group to provide risk/benefit and cost/benefit ratios for patients. A stratification or classification system that is not tied to treatment is of very little value in my estimation.

DR MOSER: Joe, we always try to define increased risk and we can talk about the relative risk of a BP of 180/100 mm Hg as being 3 or 4 times that of a person with 120/80 mm Hg, but what about the absolute risk? Do we have specific numbers on the absolute risk difference, excluding other factors, of a BP of 135/85 mm Hg compared with a BP of 120/80 mm Hg? I know there is a relationship of the rise in BP to risk, but is it enough to justify interventions other than lifestyle modifications?

DR IZZO: First of all, let me build on what my colleagues have said, but differ slightly. The meta-regression study that has been referred to from the Prospective Studies Collaboration clearly demonstrated a logarithmic, not a linear, risk of BP; and it's very mathematically precise. For every 20-mm Hg increment in systolic pressure or 10-mm Hg increment in diastolic pressure, relative risk doubles for stroke or ischemic heart disease.

DR MOSER: Put that in absolute risk terms, not relative risk, if you can.

DR IZZO: Well, I want to point out again that we're talking about a fairly wide range of BPs that goes all the way from 115 to 185 systolic. Even in the range that we're talking about, prehypertension, the answer in relative terms is that you cut the risk in half by going from an SBP of 135 to, say, 115. Now what does that really mean? It is hard to give you a term that encompasses absolute risk because of the interaction of hypertension with other risk factors. Although, if you take a

look at the Multiple Risk Factor Intervention Trial (MRFIT), 30% of all heart attacks occurred in what we now call the prehypertension group. In fairness, the prehypertension group in MRFIT comprised about half of the study participants, which is double the estimated one fourth of the US population estimated to have prehypertension. Thus, it could be argued that about 15% of all heart attacks in the United States would be expected to occur in the prehypertension group.

DR MOSER: But the participants in MRFIT were a high-risk group—they had other risk factors.

DR IZZO: On the other hand, they had prehypertensive BPs.

DR MOSER: I'm trying to get a feeling of what the absolute risk of prehypertensive BPs would be without other risk factors. We can talk about doubles and triples, but is it 3 people per 1000 in 5 years who would have a CV event or 10 people or how many? Can we be specific at this time?

DR IZZO: The overall heart attack rate is about 0.5% of the US population per year. Maybe the absolute risk is important in terms of public policy, but the relative risk is most relevant to the treatment of an individual patient.

DR MOSER: I'm trying to get some answers because we're going to talk about treatment. To justify some specific treatments, that might be costly or have adverse effects, it might be helpful to know just how beneficial the treatment would be—or maybe this isn't important.

DR IZZO: Again, let's talk about the relative risk of age and BP. At age 40, a systolic pressure of roughly 120 mm Hg is a relative risk of 1, and at age 80, 120 mm Hg is a relative risk 64 times greater. So with a doubling of age, we have substantially more risk to deal with. The absolute risk rates will indeed depend on the number of other risk factors present in the patient or population to be treated, but every patient with any degree of BP above the optimal pressure of 115–120 mm Hg ought to have systolic BP lowered by 20 mm Hg, irrespective of the absolute risk. I think JNC 7 should have made the recommendation that anyone with a pressure above 140/90 mm Hg (or 130/80 mm Hg in those with diabetes or chronic kidney disease) should receive treatment to lower systolic BP by at least 20 mm Hg AND to a level below 140 (or 130) mm Hg; BP control by far is number one on the disease prevention hit parade and ought to be controlled aggressively, period.

DR GILES: Can I just chime in here for a second? You might get the impression that we are all agreeing on a universal concept of prehypertension. But

in the paper published in *The Journal of Clinical Hypertension*, we offered an expanded definition of hypertension, which basically did not depend on the threshold value of BP. The concept is that among people who have BPs in the prehypertensive range, there are some normal people and we can tell who they are. This is not rocket science. A thoughtful physician in his or her office can do a risk assessment with the BP as part of the equation. If you watch the BP increase from 120 to 130 mm Hg, something is happening to the CV system. You can't just ignore that, even if the increase is only in the *prehypertensive* range.

The Comparison of Amlodipine vs Enalapril to Limit Occurrences of Thrombosis (CAMELOT) trial showed a relationship of lowering BP to a reduction in coronary atheroma volume in people whose BPs were in the so-called normotensive range. The Perindopril Protection Against Recurrent Stroke Study (PROGRESS) trial, which was a secondary prevention trial, enrolled people with prior strokes and BPs at levels that might not have been treated in the past and lowered them by 10 mm Hg systolic. Recurrent stroke was reduced by $\geq 30\%$ when an angiotensin-converting enzyme inhibitor and diuretic were used.

DR MOSER: Again, these were people with known vascular disease and were not just treated because of their BP levels.

DR GILES: Correct. But the BP reduction resulted in a better outcome even in the prehypertensive range. So I would take the position that what we ought to be doing for people who are prehypertensive is to try and distinguish those who already have a disease—hypertension—from those who do not. That way you can offer a patient an escape from the prehypertensive classification. Who wants to be labeled as having a disease if they are normal? On the other hand, if you have a disease, more than likely you need some treatment, maybe pharmacologic or nonpharmacologic.

DR MOSER: Henry, we all agree that an increased BP is clearly a CV risk factor and that we'd all be better off if we had so-called optimal pressure below 120/80 mm Hg. Give us your sense of how much we want to intervene in someone's life by labeling them prehypertensive instead of high-normal. Are we justified in doing this? Are we ready to say that if changes in lifestyle don't work we should use antihypertensive drugs in some of these patients? First take the prehypertensive with just one risk factor and then discuss your approach to some of the patients that Tom was referring to—the prehypertensive who already has evidence

of ischemic heart disease, microproteinuria, or previous vascular disease.

DR BLACK: I wanted to first get back to your estimate of absolute risk and how much we can change it. The logic is clear—if someone is at low risk, the treatment that you have to provide must be absolutely safe. If your risk is low, any treatment must not increase that risk. On the other hand, if we are able to more precisely estimate risk using more than simply the BP, we should be able to distinguish those whose risk is low from those whose is high or highest. In the individuals with high risk (but with prehypertensive levels of BP), it is likely that there will be dramatic reductions in events with treatment. The benefits of treatment in diabetic patients with hypertension affirms that notion.

The term prehypertension was coined because when people heard “high normal” as their BP classification, all they heard was *normal*. They didn’t hear *high*. JNC 7 wanted to get people’s attention that this wasn’t necessarily a level of BP that should be ignored—providers should understand that, and patients should understand as well. In JNC 7, lifestyle modification only was clearly recommended in prehypertensives. When we suggest treating hypertension, there are 2 ways to do it: with or without drugs. Both are *treatments*. Lifestyle modification (no drugs) is probably the safest approach to hypertension treatment. Since JNC 7 unambiguously recommended lifestyle modification for prehypertensives, treatment was advocated, although *not* drug treatment. At that time, there were no data to support a recommendation for pharmacologic therapy in people in that range. There was *no* mention of using pharmacologic agents in these patients unless, perhaps, they had evidence of end-organ damage, diabetes, or chronic renal disease—a recommendation also included in JNC VI. We now have new data that we should pay attention to and integrate into our thinking. The Trial of Preventing Hypertension (TROPHY), which studied people in the higher range of prehypertension (130–139 mm Hg systolic), was able to show that a single untitrated dose of a safe and well-tolerated agent brought BP down about 10/6 mm Hg, which theoretically reduces risk. I think that’s very important for us to recognize.

That is not to say that everyone in the prehypertension range, regardless of what else they have, ought to be treated with drugs; *not at all*. What it means is that we now have some evidence that we can effectively lower BP in these subjects and we can do it safely. If we use the epidemiologic information to predict benefit, an admittedly risky

proposition, treating such subjects and lowering their BP this much (10/6 mm Hg) might have a large impact on outcomes. As my colleagues have mentioned, many, if not the majority of heart attacks and strokes, occur in people with BP <140/90 mm Hg. Personally, I wouldn’t give medication to someone with prehypertensive levels of BP who have nothing else wrong with them. And, it is likely that a substantial number of people with BPs in the prehypertension range would not experience a BP decrease with medication. But it seems to me that if BP does go down without adverse consequences, considerable benefit could be achieved from the probable reduction in stroke, heart attack, and renal and heart failure risk. In 2006, there is no solid information to base any recommendations other than lifestyle modifications, which we know are only modestly effective, but certainly safe. One last comment: some have criticized the *labeling* of patients. They fear that designating someone as prehypertensive would prevent them from getting jobs and possibly increase their insurance premiums. This is not an issue we should concern ourselves with since the insurance companies are better actuaries than we are. If the insurance industry, that first demonstrated the increased risk above 140 mm Hg and 90 mm Hg, think that risk is elevated below our arbitrary definition of hypertension, they will price the policies accordingly.

DR MOSER: So your bottom line is if you have a patient with prehypertensive BP levels of about 130/80–85 mm Hg, with no other risk factors, you would encourage lifestyle changes but would probably not use specific pharmacotherapy, at least at present. But if that person had one or more other risk factor, you probably would?

DR BLACK: Yes, I think that if you have a safe drug that is well tolerated, and with a simple clinical assessment you find that the risk is high enough and the possible benefit of lowering BP is great enough, I would recommend pharmacologic therapy if lifestyle modification had failed. This might even be recommended at the outset. There is reason to believe that nonpharmacologic therapy will fail and that benefit will outweigh risk. Right now I believe that lifestyle modification must be unambiguously encouraged in prehypertensives until we have more data on the safety and efficacy of drugs.

DR MOSER: Alright, Joe, I come in to see you in your office. I am a perfectly healthy 45-year-old person with no worries and a good insurance policy. You take my BP several times and it’s 130/80–85 mm Hg; you tell me that you would like to see me

in several months. No specific diagnosis is given. I come back and my BP is still about 130/85 mm Hg. Now you tell me that I am prehypertensive. Does that have any effect on my psyche? Isn't it possible that I will walk out of your office with at least a perception that I have a disease? Forget the insurance policy worries, think about the psychologic impact of a label of a disease on me and on my family. Am I wrong?

DR IZZO: No, you are not. But the whole concept of prehypertension is also really about the lifetime burden issue, the age-related increase in BP, and the concept that even if you make it to middle age and are still normotensive, your risk is over 90% of becoming hypertensive if you live another 20 or so years.

DR MOSER: Yes, but what do you do about the psychologic impact of labeling? I personally don't think it's a red herring. I think it's real.

DR IZZO: I believe the issue is an early warning and that's the intent. We have ignored this for too long and have generally missed a golden opportunity to prevent future disease. As Tom mentioned, there are many markers of increased risk that are being investigated. Prehypertension is not an entirely benign condition.

DR MOSER: So we encourage physicians to do something. We say to a patient that they are prehypertensive but don't have a disease as we now define it. We can lower the BP and possibly prevent some of the complications that used to occur with elevated BP. If we can explain to people what this is all about and get physicians to explain it, then the burden of having a disease label is going to be lessened (this may only take about 2 minutes). If a patient is just told that they have prehypertension without clarification and that they are going to be treated, their attitude will be completely different. Again, am I wrong?

DR IZZO: No, but another factor here that needs to be discussed is the attitude of the patient about this problem. There are people who are extraordinarily proactive about their health and there are people who don't seem to care. Of those who are proactive, I commonly get questions such as "Should I be doing something?" or "What can I do?" When we lay out the menu of possibilities for our hypertensive patients, believe it or not, some will actually adopt lifestyle modifications, but most will *say* they will try lifestyle modifications but do nothing. More and more, patients are asking "Should I be taking medications?" So patients are now confronting doctors with this choice, and these questions may increase based on

the TROPHY study because there are some savvy consumers out there.

DR MOSER: Tom, you label me as a prehypertensive but you are a concerned, caring, and sensitive doctor. You tell me that this is just an indication that my pressure is a little higher than it should be and that I've got to do something. "If you do," you tell me, "we can prevent what used to happen to people who had hypertension." You suggest lifestyle modification and give me some material to read. What are the statistical odds of my reducing the BP from 130/85 mm Hg down to the 120/80-mm Hg range with weight loss if appropriate, moderate exercise, sodium restriction, and alcohol moderation, if necessary?

DR GILES: Oh, if you really go at it, for example, with a personal trainer, it is possible to achieve a 5- or 10-mm/Hg decrease. But let me get back to this issue because, as you know, I'm not fond of that term. We keep saying prehypertension and I keep contending that everybody is *pre*-something. To utilize an overworked cliché, we are all *pre-dead*.

DR MOSER: What should we label them? Can we coin a new term for this finding?

DR GILES: Well, we did that. In the TROPHY trial, approximately 45%–50% of the subjects had the metabolic syndrome. So this was a group at increased risk for CV disease. By the expanded definition, these people have stage 1 hypertension and we're going to treat it. And we are going to help prevent stroke, heart attack, and end-stage renal disease.

DR MOSER: So maybe we should go back to high-normal if you don't have any other risk factors and stage 1 hypertension if you have prehypertension levels plus other risk factors.

DR GILES: Well, I don't know about the high-normal part. If you have normal BP, that's optimal, and most people are willing to accept a level of 120/80 mm Hg although, for a small woman who weighs 90 pounds, her optimal BP might be 90/50 mm Hg. BPs that are above optimal for a person indicates that the CV system is not behaving in a normal physiologic fashion. As the BP continues to increase, the pressure itself begins to contribute to the ongoing disease process. So if you've got the metabolic syndrome and your BP has increased continually by 5 or 10 mm Hg, treatment is necessary, either nonpharmacologic or pharmacologic.

DR MOSER: Okay, so the bottom line, again, is that with these BPs, with or without other risk factors, we certainly should try lifestyle modification. That is going to work in a small number of people. In a person with pressures between 120–135/80–85

mm Hg or so, with no other risk factors, this is appropriate. It is also okay to try lifestyle changes even if they have other risk factors. A small number of people will have their pressures reduced to optimal BP levels. How long do you try before specific medical therapy? Three months, 6 months, longer?

DR GILES: You might wait 3–6 months, provided their other global risks aren't great.

DR MOSER: After, let's say, 4 months, I come back in to see you and I've really tried as hard as I can with or without a trainer to reduce my weight and sodium, etc. I haven't controlled the BP. Now, are you ready to use antihypertensive drugs in this relatively low-risk person with perhaps minor dyslipidemia on the basis of the one study that we've just mentioned?

DR GILES: In the appropriate patient, and this might be one, I might very well do that. I think your admonition about first do no harm is a good one. I believe, at this point in time, we do have enough data to offer patients a way to lower their BPs pharmacologically with total assurance that benefit clearly overrides risk.

DR MOSER: Tom, tell us about the TROPHY study. It's the only trial we have at present that has looked at possible prevention of hypertension or lowering of BP by pharmacologic means in this group of people.

DR GILES: This trial, which I found fascinating, actually looked at what they called prehypertension. As I've already indicated, these people were, in my view, at risk because of their other conditions but, the question was if you lower BP, could you prevent the inexorable rise in BP so that people wouldn't become hypertensive by the formal definition of 140/90 mm Hg? The trial showed a marked difference between those individuals who were left on placebo treatment and whose BPs continued to go up and those who got active treatment, with the angiotensin receptor blocker candesartan.

What I think is remarkable is that the "prehypertensive" individuals had a decrease of 10 mm Hg in systolic BP with treatment.

DR MOSER: So the first 2 years of the study when people were on a specific medication, in this case an angiotensin receptor blocker, the group on the drug noted a decrease of 10 mm Hg SBP compared with the placebo group. Baseline BPs were in a range of 120–139 systolic. Then, when the drug was stopped and both groups were continued on placebo in addition to lifestyle modifications, BPs remained lower in the group originally treated. The BP difference was only about 2 mm Hg at the end of the study, but there was still some benefit in the

prevention of a BP increase. So you think the data are clear enough to give guidance to practitioners at this stage that the benefit outweighs the risk.

DR GILES: Well, some doctors are going to require more data than others. I have a sort of Bayesian approach to this. I think we've got enough data right now that, personally, if I was in that group, I would take some medication.

There will be trials forthcoming that will add to the data that we already have.

DR MOSER: Joe, what would you do right now? A patient doesn't respond to nonpharmacologic therapy, you've got the TROPHY data and you've got your common sense and experience to guide you. Are you going to use medication?

DR IZZO: Well, the acid test for me, Marv, is what I would do if I were sitting across the table from myself, knowing what I know.

DR MOSER: Okay.

DR IZZO: I'd take the medication. And the reason is, and I want to emphasize this for a lot of reasons, that BP is the number one CV risk factor. Yes, we'll have to treat fewer people to prevent a single event if we treat only high-risk people, but in a given patient, the risk is zero or one. Which would you prefer to have? A better chance or a lesser chance? And, the answer is if you prefer a better chance and most importantly we can give you a strategy, be it nonpharmacologic or pharmacologic, that has virtually no risk attendant to the therapy itself and some potential benefit, then I'd choose specific therapy. Are you going to take the pill or not, providing that you have the means to do so? It also has to do with your own attitude toward health and health intervention as well as socioeconomic factors. What worries me most is that there is so much cynicism in the community about the role of the pharmaceutical industry, physicians, health maintenance organizations, and so forth, that we're getting away from what our fundamental mission should be: to prevent disease. If we've got effective strategies that prevent disease, in the end they're going to cost us less money and improve the overall quality and length of life. It is a slam dunk, a black and white issue, as far as I'm concerned. You treat the BP aggressively.

DR MOSER: Let me summarize. The designation *prehypertension* evoked a great deal of criticism because people felt that labeling would be disadvantageous and a disservice to patients. Some believed that many physicians would simply label a patient without a follow through; however, there is evidence that BPs of 120–139/80–89 mm Hg, do increase risk, not to the degree as a BP of 150 or 160 mm Hg,

but to some degree. Especially as Drs Giles, Izzo, and Black pointed out, most of these people have some other risk factors. They may not have dramatic increases in lipid levels or diagnosable increases in blood glucose levels or microalbuminuria, but they have some other risk factors. Basically they are at higher risk than another person with no risk factors.

I think we would all agree that lifestyle interventions should be the first method tried, except in a person who has obvious evidence of vascular involvement, such as microalbuminuria, evidence of left ventricular hypertrophy, or vascular change. If that doesn't work, and based on very preliminary data from the TROPHY study, specific pharmacologic therapy might be tried. This study suggests that by blocking the renin-angiotensin system with a relatively safe medication, you cannot only reduce BP in people who are not hypertensive by previous definitions but you probably can prevent hypertension from developing, at least over the course of 2, 3, or 4 years. During this time, it is highly possible that there might be some vascular injury from an elevated BP that we couldn't detect clinically. Is that a fair summary of the subject?

DR IZZO: Fabulous, Marv, fabulous.

DR GILES: I would second that. I would only say that in the future we may not use the term prehypertension at all. This was borrowed a little from prediabetes and, as we know, these people have almost 100% certainty of going on to type 2 diabetes. As Joe noted, BP risk is impossible to define with a threshold.

DR BLACK: I agree.

DR MOSER: Well, we had to have some number so that when we talked about hypertension, we would know what we were talking about—

140/90 mm Hg is certainly an artificial cut-off point. This does not imply that people at 138/88 mm Hg are fine and those at 142/92 mm Hg are sick.

DR GILES: I know, but what I'm saying now is that BP is a physical force. Hypertension is a disease. And although the two are clearly related, they're not the same thing. And as long as we use those terms interchangeably, we're stuck with this concept of a BP threshold, and there isn't one.

DR IZZO: May I add one more comment? BP is highly variable, and we have not come to grips with this. It's extremely important for everyone and it's especially important for the conversation we're having today. There are people with reactive BP changes in a doctor's office, in the workplace setting, and to stress situations in general. We need to categorize this separately. I would suggest that this be termed *reactive hypertension*. It's been called *white coat hypertension* among other things but it must be differentiated from sustained hypertension because the medications we use to lower BP today have little effect on the BP reactivity—not even β -blockers. There are also many disease classification issues. We have made the assumption that the BP number is a stable indicator like serum sodium, but it's not. We have to be very mindful that the number we're talking about is a little suspect and situationally dependent. We must make that clear and then wrap that into guidelines for the future.

DR MOSER: And despite the fact that we all think that this is a recent finding, we can go back 30, 40, and 50 years and realize that people who were hyperreactive to the cold pressor test or to emotion developed hypertension much more frequently than those who were nonreactors.

Thank you.