Treating Hypertension in the Elderly—Whom to Treat, When, and with What?

Marvin Moser, MD; William Cushman, MD; Suzanne Oparil, MD; Stephen Glasser, MD

Following a symposium on hypertension in Tampa, FL on February 23, 2000, sponsored by the National Heart, Lung, and Blood Institute, a panel was convened to discuss the treatment of hypertension in the elderly and review some recent data on management. Moderating the panel was Dr. Marvin Moser of Yale University School of Medicine, New Haven, CT. Serving on the panel were Dr. William Cushman of the VA Medical Center, Preventive Medicine Section, Memphis, TN; Dr. Suzanne Oparil of the Vascular Biology and Hypertension Research Program, University of Alabama at Birmingham, Birmingham, AL; and Dr. Stephen Glasser of the University of Minnesota School of Public Health, Minneapolis, MN.

DR. MOSER: Let us first define what we mean by hypertension in the elderly; explain some of the physiologic differences in the elderly compared to the young, especially in patients over 75 or 80 years of age, that might influence treatment decisions; then discuss the influence of comorbidities on therapy choices; and finally, discuss when and how to treat this rapidly growing segment of our population. Is monotherapy appropriate? Is combination therapy appropriate? Which approved drug classes are effective and which do not work? What should we expect when we treat the elderly hypertensive? Are results conclusive in terms of reduction of morbidity and mortality, or do we not have enough data thus far to make firm recommendations?

Dr. Oparil, what about hypertension in the elderly? Please define it—both isolated systolic and systolic/diastolic hypertension.

DR. OPARIL: Hypertension is hypertension. The standards do not change...the definitions of hypertension in adults do not change with different ages. Systolic/diastolic hypertension is defined as a systolic blood pressure (SBP) greater than 140 and a diastolic blood pressure (DBP) greater than 90 mm Hg. Isolated systolic hypertension (ISH) is defined as a systolic pressure equal to or greater than 140 mm Hg with a diastolic pressure of less than 90 mm Hg. This is the Joint National Committee (JNC) VI definition.

DR. MOSER: Are the Europeans and the World Health Organization wrong in saying that we should continue to define ISH as above 160 mm Hg? What data do we have with pressures between 140 and 160 that risk is increased and that reducing pressure is beneficial?

DR. GLASSER: Implicit in your question is exactly why there is a difference of opinion between the Europeans and others. I do not think it is a matter of right or wrong. We know that there is a graded, continuous, increased risk of cardiovascular events as SBP climbs above 140 mm Hg. But what we do not know for the group with pressures between 140 and 159 mm Hg is whether treatment reduces that risk. We have good data for treatment of SBP above 160 mm Hg, in terms of reducing risk. We can confirm increased risk but lack sufficient information about reducing the risk in this stage 1 group. At present there are limited drug treatment data that risk can be reduced between 140 and 159 mm Hg. Those who support this approach cite indirect evidence.

DR. MOSER: Dr. Cushman, when we define ISH as above 140 mm Hg and implicitly inform practitioners that these levels should be treated, are we correct?

DR. CUSHMAN: It depends on whether we consider the epidemiologic risk, in which case 140–159 mm Hg (stage 1 systolic hypertension) carries a 50% greater risk for a cardiovascular event compared to a SBP of <140, or whether we seek answers from prospective clinical trials on results of therapy. In terms of defining hypertension, it is fine to use 140 mm Hg and above for a definition of ISH. We should certainly initiate lifestyle changes in all patients in the stage 1 range.

However, in light of prospective clinical trials, I might be inclined to change the number to 150 mm Hg. If you note the SBP levels in the placebo group in the Systolic Hypertension in the Elderly Program (SHEP), for example, they averaged in the 150s,

whereas in the treatment group they averaged in the 140s. Subjects had a greatly reduced risk of cardiovascular events in the lower blood pressure (BP) group (140s). I extrapolate from these data that above 150 mm Hg we should treat with drugs.

On the other hand, a great number of our older population have diabetes and other comorbid conditions, and I interpret the prospective clinical treatment trials to indicate that BP even lower than 140 mm Hg should be the goal in these patients. We have no data that treating diabetics with SBP below 140 mm Hg at entry is beneficial. We need more data. My expectation is that lower will be better, but I will credit the evidence and suggest medication at >150 mm Hg for most hypertensives and at >140 mm Hg for diabetics.

DR. MOSER: That is an interesting point. Maybe we can agree with this modified definition. People are at greater risk above 140 mm Hg, but at present we do not have sufficient data for a blanket recommendation to treat people who have no other risk factors and a DBP of <90 mm Hg. Should we say that 150 mm Hg is the cut point to add medication, on the basis of better data than we have with lower BP cut points? Or do we advise adding medication if BPs stay above 140 mm Hg and/or 90 mm Hg after a suitable period of lifestyle modification? Obviously, in people with other risk factors, i.e., diabetics, smokers, and people with hyperlipidemia or known coronary heart disease, specific treatment should be started earlier. But perhaps drug therapy should not be tried unless the SBP is >150 mm Hg in a relatively healthy 75–80-year-old.

DR. CUSHMAN: It is not because I disbelieve that patients in the 140–150 mm Hg group would benefit, but we do not yet know the long-term benefits of treatment.

DR. MOSER: Other comments?

DR. OPARIL: If you leave people untreated at 150 mm Hg and wait a few years, they will be at 160 mm Hg and then they will qualify for treatment regardless of the definition.

DR. CUSHMAN: If BP is hovering around 150 mm Hg, I would be more prone to initiate drug treatment.

DR. GLASSER: I would tend to agree with that, but as Dr. Moser noted, I would also tend to take into account other target organ damage or comorbidities. We should encourage lifestyle modifications in this borderline BP group and consider specific therapy if there is no response.

DR. MOSER: But is there a problem for an 80-year-old man to be told not to eat a steak and to cut sodium intake?

DR. GLASSER: It depends on the degree to which you recommend that.

DR. CUSHMAN: I would certainly tell him to go out and walk 30–60 minutes every day.

DR. MOSER: Now, should these things we are talking about apply to people younger than 80 as well as over 80 years of age? Are we going to make any distinctions in our approach to the very old?

DR. GLASSER: Clearly the data on people over 80 are much less abundant. We simply do not have the evidence yet to be dogmatic about recommendations in this age group.

DR. MOSER: I just reviewed the data on a total of about 1500 people over the age of 80 in the clinical trials. A reduction in cardiovascular events was demonstrated in treated patients with systolic/diastolic as well as isolated systolic hypertension. Life may not be significantly prolonged, but strokes and heart failure are reduced. Therapy appears to be quite worthwhile.

DR. GLASSER: I believe that additional studies are needed. I suspect that it would be beneficial to treat 80-year-old individuals who are physiologically younger. One must take into consideration what one is trying to achieve, given the person's overall state of health.

DR. MOSER: Of course, in these patients you do not want to use any treatment that may decrease the quality of life. In the SHEP trial there were about 650 people over 80 years of age; treating those people with medication achieved benefit—mostly a reduction in nonfatal strokes and heart failure. Certainly the prospect of fewer strokes and episodes of heart failure is a reasonable objective.

DR. CUSHMAN: In a recent study of nursing home patients, whom we often considered beyond needing treatment, there were fewer falls and fewer adverse effects in those treated for their hypertension, with better control of their BP, especially if they were on diuretics. Better control of BP may actually decrease postural hypotension. Regardless of whether your goal is to prevent strokes, heart attacks, or heart failure, even short-term benefits may actually be seen in the very old if BP is lowered.

DR. MOSER: You may even prevent further osteoporosis with the use of a diuretic.

Dr. Oparil, there are many physiologic changes in the elderly. Do these really affect management? What does the clinician have to know other than that cardiac output and renal function are decreased and that baroreceptor sensitivity is also blunted? How do these changes impact treatment?

DR. OPARIL: There are a few things that happen in the elderly that are important. We lose nephrons as we get older, whether we are normotensive or hypertensive. If we are hypertensive, we lose nephrons faster and it is hard to judge the status of renal function because it is not very precisely reflected in the serum creatinine. Early on, a relatively large change in renal function might result in an insignificant change in serum creatinine levels and may be missed by the clinician. Conversely, once serum creatinine begins to increase in the elderly, there is already so much renal damage that specific antihypertensive treatment can push the patient into renal failure.

Because older people have less muscle mass, antihypertensive treatment should be initiated at lower doses. As a ballpark figure, treatment should be initiated with one half the dose usually given to younger patients.

There are also problems with stiffness of blood vessels. This correlates not only with increased pulse pressure but also with impaired baroreflex function. When older people stand up, a reflex increase in heart rate and cardiac output may not occur as early or as reliably as in the young. Therefore, a major problem with postural hypotension and medication intolerance may be noted. However, pretreatment postural hypotension should not deter the physician from treating the patient's hypertension; it just mandates caution. BP should be followed with standing measurements, as well as the usual sitting BPs.

DR. MOSER: This is one reason why α blockers should not be used as primary treatment. To amplify Dr. Oparil's comments, cardiac output decreases in older people, but by lowering BP we may actually increase it, and that is a beneficial effect. Certainly renal function is something we have to pay attention to because we see elderly people with creatinine levels of 1.2 or 1.3 and if we give them a medication that decreases renal function even a little bit more, creatinine levels could rise to 3.0 or 4.0. These are some things one has to worry about.

But how clinically important are these concerns? Is it enough to advise just using one half the dose of what you would ordinarily use, and take the BP with the patient standing up? The half-dose concept is not based on much evidence.

DR. GLASSER: The last point—taking BPs in the standing position—is important. We all know this but I do not see it done often. Certainly it also makes sense to titrate slowly. The half-dose phenomenon, I must agree, was just pulled out of the air. I am not sure that I know of any studies that have specifically examined that.

DR. MOSER: Is it reasonable?

DR. GLASSER: Clinically, it has been.

DR. CUSHMAN: I am not sure that all drugs are the same, or that the half-dose phenomenon is valid in all situations. We found that with diuretics—doses of up to 50 mg a day of hydrochlorothiazide (HCTZ), for example—were as well or better tolerated in the elderly than in young people. What we tended to find is that a dose of 50 mg of HCTZ is more likely to cause biochemical changes in a young person and less antihypertensive effect than in the elderly, who tolerate it quite well.

We actually were comparing 50 mg of HCTZ to 100 mg and found that 100 mg caused more biochemical adverse effects in everybody, but had no greater efficacy in either young or old. We had actually started titration at 25 mg/day.

Studies show that many physicians stop at a too-low dose of an angiotensin-converting enzyme (ACE) inhibitor, as well as other antihypertensive agents. There is little good evidence that we should not use full doses of ACE inhibitors or other drugs in the elderly if these are needed to reduce BP to goal levels.

DR. MOSER: Years ago, when we did not know much about drugs and dosage effects, we just continued to titrate upward. If 1 mg was not effective we kept doubling the dosage; we thought the more the better. We now know that most of the antihypertensive drugs are almost fully effective at the lower dosage range. Dr. Cushman, please give examples of average doses in the elderly with a diuretic, a ß blocker, an ACE inhibitor, and a calcium channel blocker.

DR. CUSHMAN: With HCTZ, for example, I would start with 12.5 mg and usually go to 25 mg and, even though we have demonstrated that above these levels this is a safe drug, I would not increase it to 50 mg/day until I was using three drugs and not achieving goal BPs.

DR. MOSER: After 25 mg/day would you add a ß blocker or an ACE inhibitor or another agent?

DR. CUSHMAN: Exactly. If I chose an ACE inhibitor, I would have to consider that the elderly might be like black Americans: They need higher doses of ACE inhibitors. If I used a drug like lisinopril or enalapril or quinapril, I would consider that 10–20–40 mg is the normal dosage range. I do not use 5 mg, for example, even though some of these agents have been approved by the FDA at that dose.

DR. MOSER: You don't think they work at the lower dosages?

DR. CUSHMAN: I think they rarely work and I am not concerned about the risk unless somebody is

volume-depleted. If the patient is on a diuretic I would tend to start at the lower dosage.

DR. MOSER: What about postural hypotension, and is the cough dose-related? I have seen quite a few elderly people experience postural changes if dosages are not carefully titrated.

DR. CUSHMAN: I don't think the cough is dose-dependent. I do frequently become a hero when I stop an ACE inhibitor in somebody who has a cough. This is probably two or three times more common in women. If the cough is no different when you stop the ACE inhibitor, then you can go back to using it.

DR. MOSER: Let's go back to dosage again. Assuming that many elderly patients, especially the very old (>80 years of age), do not respond well to an ACE inhibitor by itself, why not add a small dose of a diuretic to the low, 5-mg dose of the ACE inhibitor, rather than increasing the dosage? A better effect is obtained than with either drug alone, with a high degree of safety and little or no increase in side effects.

DR. CUSHMAN: Obviously, what I am saying has to be put in context. I put everybody over the age of 60 on a diuretic unless there is some absolute contraindication. Whenever I use an ACE inhibitor or other drug, such as a ß blocker, in an older individual, it's almost always with a background of at least a low-dose diuretic.

DR. OPARIL: In the case of ACE inhibitors, many patients—not only the elderly—are undertreated. The importance of administering an adequate dose has to do with the duration of ACE inhibition. All ACE inhibitors, even the very short-acting ones like captopril, will completely inhibit ACE for a few hours. The duration of action of the drug, then, depends in part on the dose (higher doses lead to more sustained enzyme inhibition) and in part on the specific drug.

DR. CUSHMAN: Would you agree that you virtually cannot overdose an ACE inhibitor?

DR. OPARIL: Yes.

DR. MOSER: I have to disagree. In heart failure I believe that the doses of an ACE inhibitor are usually too low, but I would not push an ACE inhibitor above 15–20 mg/day, especially in the elderly and especially those who are on a diuretic. I have seen too many instances of dizziness and postural hypotension. And what about renal function?

DR. OPARIL: In patients who are on furosemide and an ACE inhibitor, we have to worry about precipitating renal failure, but this isn't very common.

DR. MOSER: I think you agree that in patients with relatively normal renal function you would use a longer-acting thiazide, not a shorter-acting loop diuretic like furosemide.

DR. OPARIL: I agree.

DR. MOSER: Dr. Glasser, would you use a ß blocker with a diuretic in this population?

DR. GLASSER: Let me first add one comment about the ACE inhibitors. Renal artery stenosis is a bit more common in the elderly and because of this I tend to go lower and slower with the ACE inhibitors, just to protect the few patients who might incur trouble.

Regarding ß blockers, I was more enthusiastic about these agents as monotherapy 5 years ago. From a pathophysiologic point of view, if one thinks of arterial stiffness and plaque rupture related to pulsatile pressure and pulse pressure, one would almost think that a ß blocker would be the premier agent to use. Yet studies seem to support the use of diuretics to a greater extent in the elderly. The JNC VI recommended a diuretic as initial therapy in the elderly population and a ß blocker with a diuretic if this is not effective.

Certainly in post-myocardial infarction patients and in patients with hypertension and angina, ß blockers are first choices. Also, the newer studies indicate that ß blockers reduce morbidity and mortality in patients with heart failure, when added to conventional therapy. However, as monotherapy in an elderly hypertensive I am not convinced that this is my first choice. Combination therapy is a different issue.

DR. MOSER: It was surprising to many people that ß blockers were not as effective in the elderly in reducing coronary heart disease morbidity and mortality. Often forgotten, however, in the arguments put forth by some of our colleagues against using these agents in the elderly, is that ß blockers significantly reduced strokes and heart failure. These are two major events we want to prevent. There are also data in post-myocardial infarction patients who are at high risk and in patients with heart failure, whether or not they are hypertensive, to indicate that morbidity and mortality are reduced despite some theoretic considerations about the effect of these agents on insulin resistance. Some of the benefits of ß blockers can be accounted for by their reduction of the activity of the renin-angiotensin-aldosterone system. These agents may also increase the availability of vasoactive peptides. The JNC VI recommendation for the use of a ß blocker plus a diuretic in the elderly makes good sense.

None of us has brought up the use of calcium channel blockers (CCBs) in the elderly. Where do these fit into the treatment algorithm of the elderly patient with or without comorbid conditions? I guess we can assume that about 60%–70% of the elderly have some comorbid disease.

DR. GLASSER: There is a long-term hypertensive trial in the elderly based on the use of nitrendipine, a reasonably long-acting dihydropyridine CCB. This trial, the Systolic Hypertension-Europe (Syst-Eur) study, reported a reduction in strokes and overall cardiovascular events in treated compared to control subjects, so a long-acting CCB may be an alternative should a diuretic be insufficient alone or not well tolerated. There is controversy about the CCBs increasing cardiovascular events; my feeling is that these adverse events occur predominantly with short-acting dihydropyridine CCBs and not with the longer-acting ones or other classes of calcium antagonists—the non-dihydropyridine CCBs, such as verapamil or diltiazem.

DR. MOSER: There is also a small Japanese study in the elderly, with about 200 people in an isradipine group compared to about 200 people on a diuretic. Cardiovascular events were equal with isradipine but not fewer than with a diuretic. At least we now have some data confirming that the CCBs are probably safe in the elderly, but thus far no data that they are any better than other therapies. That leads to the question, should we use them preferentially? They are widely used in the elderly. Dr. Cushman, should physicians continue this practice?

DR. CUSHMAN: No, I don't think they should. We have a number of trials that are going to address the issue. Several studies in diabetics with only a few hundred patients showed that more cardiovascular events occurred with long-acting CCBs than with ACE inhibitors, but these studies are too small to draw definitive conclusions. It is of some concern to me that in the Swedish Trial in Old Patients (STOP)-2 elderly hypertension study, conventional therapy with a diuretic and/or a ß blocker was as effective as therapy with an ACE inhibitor or CCB, but myocardial infarctions and heart failures were more frequent with CCBs than with ACE inhibitors. CCBs also have not been shown to be as beneficial in these two areas in other trials. This raises enough concern that I would be hesitant to use a CCB as monotherapy in an elderly person. I have no qualms about using it in a multidrug regimen in nonresponsive patients. CCBs are very effective at lowering BP in the elderly, so if a patient is taking a diuretic, ß blocker, or ACE inhibitor, or a combination, and the BP is not controlled, then a CCB would be a good choice to add. It is probably not a good idea, however, to use a nondihydropyridine CCB, such as verapamil, and a ß blocker together because of concerns about cardiac conduction and bradycardia.

DR. MOSER: I agree with Dr. Cushman. CCBs are effective blood pressure lowering agents in the elderly, perhaps even more so as monotherapy than a ß blocker or an ACE inhibitor, but all of the data I have seen, in either primary or secondary prevention trials, have failed to convince me that they are as effective or more effective in reducing morbidity and mortality than other agents. I believe that it is important to emphasize data from the large STOP-2 study. As Dr. Cushman noted, diuretics/ß blockers were just as effective as ACE inhibitors or CCBs in reducing morbidity and mortality in elderly hypertensive patients. However, there was a significantly greater occurrence of myocardial infarctions and congestive heart failure events, with the CCB as compared to ACE inhibitors. My first choice in the treatment of the elderly hypertensive is still a diuretic and, based on the Syst-Eur data, I would use a CCB as an alternative in ISH. I agree with Dr. Cushman that these agents are indicated as add-on therapy in a patient not responsive to other therapy. Who disagrees with that?

DR. GLASSER: I totally agree.

DR. OPARIL: I also agree with that, with one caveat: Most of the trials that have been mentioned have been carried out in Europe, with 99% Caucasian populations. I think that for the black American elderly, and there are many, CCBs clearly do have a place if a diuretic by itself is not effective.

The other interesting concept is that there is quite a bit of evidence that if an ACE inhibitor is combined with a CCB, some of the adverse effects of the CCB are overcome, such as activation of the sympathetic nervous system and peripheral edema. There is a place there for this type of combination therapy.

DR. MOSER: You are right, of course. However, as Dr. Cushman's group demonstrated, if you use any medication with a diuretic, you obtain a better result than if you use a combination that does not include a diuretic. Is that correct, Dr. Cushman?

DR. CUSHMAN: In general that is true. I have to qualify that by saying that we compared all regimens that included a diuretic vs. all those that did not. So we lacked the power to compare a diuretic/ACE inhibitor vs. a CCB/ACE inhibitor. There are larger studies that suggest that the CCB/ACE inhibitor combination is quite effective at lowering BP.

DR. MOSER: I think we all agree that: 1) Hypertension in the elderly, even in people over 80, should be treated. Some of us might wish to hedge on the definition of isolated systolic hypertension, but it can be reasonably defined as a SBP of >140

or >150 mm Hg and a DBP of <90 mm Hg, with or without comorbidities. More importantly, if there are comorbidities, and most elderly people have some, specific treatment should be instituted earlier than in lower-risk subjects. 2) Dosages should be smaller—about one half the starting dose in a younger persons is reasonable. 3) There are physiologic changes, such as a reduction in cardiac output, renal function, and baroreceptor sensitivity, that may play a role in choosing medications and dosages, but we can probably treat most (but not all) elderly people satisfactorily. One caveat: take the BP in the standing position and use this measurement to guide treatment.

We have just summarized which drugs we would use in the elderly. But let's be more specific. If you use a CCB or a ß blocker, what dosages would you start with and when would you stop titrating upward and add a diuretic?

DR. GLASSER: I tend to be on the conservative side and start at lower doses to protect the few patients who might have an adverse reaction.

For atenolol or metoprolol I would start at 25 mg/day and consider going up to 50 mg/day. I would start nadolol at 20 mg/day and go to 40 mg/day if necessary.

I tend not to push dosages higher than these. I used to push to the highest dose of one drug before adding another, but we no longer do that; we tend to add a second drug—usually a diuretic.

DR. MOSER: What about dosages of CCBs?

DR. OPARIL: The most commonly used CCB is amlodipine. The usual starting dose in younger people is 5 mg, but I would start an older person with 2.5 mg/day.

DR. MOSER: And how high would you go before adding a diuretic, for example?

DR. OPARIL: Probably to the middle of the dose range, about 5 mg/day.

DR. MOSER: Does any of you agree with the JNC VI recommendations that combination therapy is appropriate as initial therapy? We based that on data that monotherapy works in only about 50% of people and that combinations that affect different physiologic parameters are effective in about 70-plus percent of patients. How many agree? Dr. Cushman? You have a lot of experience with combinations.

DR. CUSHMAN: I think it is an appropriate concept, but I most often use one drug, increase the dosage, and then add other drugs. I do this slowly in the elderly. Many physicians are resistant to going beyond using a single drug and this is one reason for failure to achieve goal pressures. The first thing I tell my patients, for example, is that most hypertensive patients need two or three drugs. If I can achieve results with one pill, i.e., a combination of two drugs, then that's great.

DR. MOSER: Why not save time? Titration is easier and faster with a combination than with monotherapy. Also, many elderly patients are already taking four or five or more pills a day. Adding one pill is different from adding two or three. There are a lot of psychological advantages to starting a combination when you expect a success rate of about 75% or 80%.

DR. CUSHMAN: But I would like to have more data, especially on adherence and safety, before I start with two-drug therapy, even though intuitively I consider it a good idea.

DR. GLASSER: I have a somewhat different perspective. I start patients on one drug, usually a diuretic. If goal pressure is not achieved and I decide to add a second drug, that is when I use a combination tablet. This is not really adding a second drug most of the time, but rather adding a combination in a single dose as the second step.

DR. OPARIL: We have not discussed tolerability of therapy in the elderly. I have a referral practice and most of the patients who come to me initially have a lot of complaints about the drugs they have been treated with. I try to sort out the complaints; are they secondary to the disease process or to the therapy? That is why I am reluctant to use a fixed-dose combination as primary therapy, particularly in the elderly.

DR. MOSER: That sounds reasonable, but I have found that small doses of two different agents produce few side effects. There is some reluctance to accept the JNC VI recommendations regarding the use of fixed-dose combinations as initial therapy. The sense of this panel is that they might be useful, maybe not as initial therapy but as treatment if a first drug doesn't work.

What about data comparing fixed-dose combinations to higher-dose monotherapy? We have some good data with ß blocker/diuretic, ACE inhibitor/diuretic, and angiotensin receptor blocker (ARB)/diuretic combinations in low doses, compared to higher-dose monotherapy with CCBs and ACE inhibitors. Combinations are as effective or more effective, with relatively few side effects. Despite these data, the medical world may not be ready for this approach.

DR. CUSHMAN: A comment about this: before I would push to 100 mg of atenolol, 10 mg of amlodipine, or 50 mg of HCTZ, I would use a second drug or a combination. On the other hand, I would not stop with a tiny dose of a medication and say, "Well, the patient has had a trial of a single drug."

I would go to an average JNC VI dose of a drug before concluding that there is no response. We do see increased response with increased doses of an ACE inhibitor or a CCB.

DR. MOSER: In the elderly, in whom expectations of goal BPs with an ACE inhibitor, an ARB, or a ß blocker as monotherapy are not great, why not start patients with a small dose of a ß blocker/diuretic or an ACE inhibitor/diuretic right from the start, and get a good response?

DR. CUSHMAN: I agree, but of course I already qualified my comments by saying I would never start therapy without a diuretic.

DR. MOSER: Dr. Glasser, what do you expect from treatment in the next 100 people over the age of 65? In what percentage do you guess that you are going to be able to lower BP to a goal of <140? Or do you set a goal of 150 mm Hg and settle for that level?

DR. GLASSER: It depends on the patient's starting BP levels. For the typical elderly hypertensive with a SBP of 170–180 mm Hg, it will take multiple drugs to bring the pressure down and it may be reducible to only 150–155 mm Hg without incurring trouble. But remember, even a 20-mm Hg reduction reduces the risk of heart failure or a stroke. We may have to settle for less than perfect BPs, but in some patients goal pressures can be achieved.

DR. MOSER: Dr. Oparil, have you reached the same conclusion?

DR. OPARIL: I agree with that. Only in a minority of elderly patients do you reach 140 mm Hg systolic, particularly if you start at a high level, such as 200 mm Hg systolic. Also, if we believe in the concept of beneficial vascular remodeling, it is possible that over a period of years you may get progressively better control.

DR. MOSER: Dr. Cushman, I know you agree with that concept, too, and I am going to ask you the last question. Why do you think physicians are reluctant to treat patients with BPs of 170/70 mm Hg or 180/75 mm Hg? Is it the concern about decreasing the DBP too much as you reduce the SBP? Since coronary arteries fill during diastole, does decreasing the DBP result in increased coronary events in patients with ischemic heart disease—the so called "J curve" effect? How much does this interfere with the decision to treat systolic hypertension?

DR. CUSHMAN: About a decade ago this "J curve" concept had a lot greater impact. I think that much of the inertia about treating systolic hypertension in the presence of normal DBP is the lack of appreciation that systolic elevations pose a much higher risk than diastolic elevations. The reluctance may be based on a century-long focus on DBP.

I don't worry about getting the DBP too low when treating to a SBP of <150 mm Hg. There are data to suggest that a diastolic drop to below 55 mm Hg, for example, predicts higher risk, but I'm not sure that it has anything to do with the treatment itself.

DR. MOSER: But if the patient's SBP decreases from 170 to 150 mm Hg and the DBP goes from 75 to 55 mm Hg, does that mean we should be careful about decreasing the systolic pressure any further?

DR. CUSHMAN: I would hold the course at that point. On the other hand, if the systolic drops to 140 mm Hg and the diastolic is 50 mm Hg, as of today I would probably back off a little bit.

DR. MOSER: Then there is a cut point at which we should pay attention, and it is around the 55–60 mm Hg range.

DR. CUSHMAN: That is right, although my belief is that it is really a marker of wide pulse pressure, stiff arteries, and high risk and that the treatment is really not doing any harm. However, in the absence of safety data from treating to that level of DBP, I think we need to err on the side of safety for the patient.

DR. OPARIL: Yes, I agree, but let's also recognize that it often takes a great deal of effort to reduce SBP to below 160 mm Hg.

DR. MOSER: To summarize briefly: elevated BP in the elderly—either systolic/diastolic or ISH—increases cardiovascular risk. Ideally, if SBP is elevated to >140 mm Hg, it should be treated and reduced to <140 mm Hg, but this is often difficult. Medications should be given at lower doses and titrated upward slowly to avoid side effects, especially postural hypotension. BP should be followed in the standing position. Data clearly show benefit in reducing both cerebrovascular and cardiovascular events in elderly hypertensive patients following lowering of BP, even in patients >80 years of age. Choice of therapy includes a diuretic and, if this is ineffective, the use of other agents, i.e., ß blockers, ACE inhibitors, ARBs, and CCBs. Combination therapy is reasonable in this age group. Some physicians still are convinced that this should not represent initial therapy despite the JNC VI recommendations that combination therapy is appropriate. Numerous effective combinations of ß blockers/diuretics, ACE inhibitors/diuretics, ARBs/diuretics, and ACE inhibitors/CCBs are available.