Hypertension in Older Adults

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The high prevalence of hypertension in older persons (nearly one of two subjects aged 60 years and older) suggests that the recognition and treatment should be a priority for physicians. Although diastolic blood pressure is regarded as an important risk factor, it is now clear that isolated systolic hypertension and elevated pulse pressure also play an important role in the development of cerebrovascular disease, congestive heart failure, and coronary heart disease, which are the major causes of cardiovascular morbidity and mortality in the population aged older than 65 years. Controlled, randomized trials have shown that treatment of systolic as well as systolodiastolic hypertension decreases the incidence of cardiovascular and cerebrovascular complications in older adults. The question of whether treatment of hypertension should be maintained in very old persons, those older than 80 years, is still undecided.

General Considerations on Hypertension in Elderly Persons

Definitions

The definition of hypertension does not change with age: a systolic blood pressure (SBP) of $>140$ mm Hg and/or a diastolic blood pressure (DBP) of $>90$ mm Hg. The Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNCVI) and the World Health Organization/International Society of Hypertension guidelines subcommittees have agreed that both SBP and DBP should be used for the classification of hypertension (1,2). Systolodiastolic hypertension is diagnosed when SBP is $\geq 140$ mm Hg and DBP is $\geq 90$ mm Hg. Isolated systolic hypertension (ISH) refers to an SBP of $\geq 140$ with a DBP of $<90$ mm Hg.

Epidemiology

Although rise in blood pressure (BP) is not a normal part of aging, the incidence of hypertension in the elderly population is high. After the age of 69, the prevalence of hypertension rises to 50\% (3). In the 1988–1991 National Health and Nutrition Examination Survey, which evaluated an ambulatory population aged 65 to 74, the overall prevalence was 49.6\% for stage 1 hypertension (140–159/90–99 mm Hg), 18.2\% for stage 2 (160–179/100–109 mm Hg), and 6.5\% for stage 3 hypertension ($>180/110$ mm Hg) (3).

Both SBP and DBP are higher in blacks than in whites (4), and in women than in men (5), after the age of 30. Between the fifth and the sixth decade of life, the prevalence of hypertension (6) is higher in women (40\%–50\%) than in men (30\%–40\%).

The prevalence of ISH is about 7\%, 11\%, 18\%, and 25\% in age classes 60 to 69, 70 to 79, 80 to 89, and older than 90 years, respectively (7). ISH is also more frequent in women than in men. In the Framingham study, ISH, defined as an SBP of $>160$ mm Hg with a DBP of $<95$ mm Hg, accounted for 57\% of cases of hypertension in men and for two thirds of cases in women between the ages of 65 and 89 (8). The Systolic Hypertension in the Elderly Program (SHEP) study found that the prevalence of ISH, defined as an SBP of $>160$ mm Hg with a DBP of $<90$ mm Hg, increased from about 8\% among subjects in their 60s to 22\% by the age of 80 (9).

Physiopathology

Both SBP and DBP increase with age (3). SBP rises progressively until the age of 70 or 80, whereas DBP increases until the age of 50 or 60 and then tends to level or even decline slightly (3,5). This combination of changes probably reflects stiffening of the blood vessels and reduced arterial compliance and leads to a large increase in pulse pressure with aging (3). Pulse pressure has been shown to be the best predictor of structural alterations in the arteries (10,11).

The mechanisms for hypertension in elderly persons are still unclear. The major effects of normal aging on the cardiovascular system involve alterations of the aorta and of the systemic vasculature. Aortic and large-artery wall thickness increases and vessel elasticity decreases with age. These changes induce a decline in aortic and large-artery compliance and an elevation of SBP (12). The reduction of vessel elasticity results in an increase in peripheral vascular resistance. Baroreceptor sensitivity is modified with age. Alterations in baroreceptor reflex mechanisms may explain the variability of BP revealed by continuous monitoring (13). Decreased baroreceptor sensitivity results in an impairment of postural reflexes, making elderly hypertensive individuals more sensitive to orthostatic hypotension (14). Changes in the balance between $\beta$-adrenergic vasodilation and $\alpha$-adrenergic vasoconstriction are in favor of vasoconstriction that increases peripheral vascular resistance and BP. Sodium retention due to increased intake and decreased excretion could also contribute to hypertension. A fall in plasma renin with increasing age has been demonstrated (15). The renin response to salt intake is more reduced with age in hypertensive than in normotensive elderly subjects.
(15); however, the renin-angiotensin system is not regarded as playing a major role.

These changes are responsible for decreased cardiac output, decreased heart rate, decreased myocardial contractility, left ventricular hypertrophy, and diastolic dysfunction. They induce an impairment of renal function with decreased renal perfusion and reduced glomerular filtration rate (16–18).

**BP as a Risk Factor for Cardiovascular and Cerebrovascular Morbidity and Mortality**

Hypertension stands out as the major risk factor for cardiovascular morbidity and mortality in the elderly population (19,20). The risk from hypertension has been demonstrated for stroke, left ventricular hypertrophy, congestive heart failure, coronary and peripheral artery diseases, vision impairment, end-stage renal disease, cognitive impairment, and dementia. Both SBP and DBP are established risk factors, but, with advancing age, SBP becomes a better predictor than DBP in men and women (21,22).

**BP and Cerebrovascular Morbidity**

Hypertension increases the risk for transient ischemic attacks (23), as well as the incidence of any type of strokes (24–26), including ischemic strokes and focal intracerebral hemorrhages (27). The risk of stroke rises proportionately with increasing BP (23,28,29). SBP and DBP, as well as combined hypertension, have been proven to increase substantially (30). In the last 25 years, emphasis has been on the deleterious influence of elevated DBP (27). However, as stressed by Black (18), SBP has been proven for more than 25 years to be a stronger predictor of cardiovascular diseases than DBP (22,31,32). The association between SBP and the incidence of strokes was demonstrated in a prospective study that included 2772 elderly persons aged 65 to 74 years (33) and still persists at highly advanced ages as shown in a 10-year longitudinal study of 191 women aged 61 to 101 years (21). Because, at any fixed SBP level, mortality increases with decreasing DBP, pulse pressure is bound to be considered as a risk factor in its own right and should be examined as such (34).

Data from the Framingham study have shown that, apart from the risk of stroke, elevated SBP is a major risk factor for all cardiovascular diseases, including congestive heart failure, ischemic cardiopathy, and peripheral artery diseases (20). The risk of left ventricular hypertrophy (LVH) increases with age (35). However, hypertension, especially systolic hypertension (36), and obesity are independent risk factors for LVH in men and women of all ages. LVH is more frequent in American blacks and in women. Data from the Framingham study have shown that LVH demonstrated by echocardiography is a major predictor of cardiovascular disease and all-cause mortality, most likely because it is a major predictor for heart failure (37). In a report of the Framingham cohort who developed heart failure, these elderly people tended to have primarily systolic hypertension (38). The incidence of ischemic cardiopathy is 3.5 times higher in hypertensive than in normotensive elderly persons. Hypertension is the major risk factor for ischemic cardiopathy other than age, excessive weight, hypercholesterolemia, or hyperglycemia (20). Hypertension, along with diabetes mellitus, accounts for the majority of new cases of end-stage renal disease (39). In addition, renal disease may be a cause and a consequence of hypertension.

A recent review (40) shows that data from cross-sectional studies are conflicting, but most longitudinal studies demonstrate a positive correlation between BP levels and the development of cognitive abnormalities and dementia years later.

High mid-life BP has been shown to be a strong and independent predictor of later cognitive impairment (41–46). However, some studies that included very old patients reported a J-shaped curve profile with a higher cognitive impairment in subjects with low BP (47–50). The decrease in BP may be due to some pathological processes also affecting cognitive functioning or, alternatively, may be a consequence of dementia. As underlined by Glynn and colleagues (51), the relationship between cognition and BP is complex, and different factors should be taken into account. Age and education, duration of follow-up, hyperinsulinemia, and other cardiovascular risk indicators, such as diabetes mellitus or hypercholesterolemia, may potentiate the negative effects of high BP on cognitive functions (52). The mechanism by which chronic hypertension alters cognitive functions remains to be elucidated. Chronic hypertension leads to vascular remodeling with narrowing of the lumen and wall thickening (53). This might affect cerebral blood flow and disturb cerebral metabolism and structure. The negative effect of high BP levels on intellectual performance could also be linked to alterations in the cerebral white matter (54,55).

Some authors have suggested that hypertension, particularly high SBP, was the main risk factor for white-matter lesions (56–59), although others have reported either no or an inverse association with BP (47,60,61).

In addition, hypertension appears to be the strongest risk factor for all forms of vascular dementia (53,57,58,62–67). Although clinical criteria (International Statistical Classification of Diseases, Tenth Revision, and Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition) used in most research studies to establish the diagnosis of probable Alzheimer’s disease specifically exclude cases with symptoms or signs of cerebrovascular diseases (68), the issue of a possible vascular component in this disease was addressed recently. Some authors have found an inverse relationship between the occurrence of Alzheimer’s disease and hypertension (47,69,70), while others have suggested that hypertension may play a role in the development of Alzheimer’s disease (64,71). Alzheimer’s disease has been reported to be associated with other vascular risk factors (58,72), including coronary heart disease (73), atrial fibrillation (74), diabetes mellitus (75), white-matter lesions (76), and indicators of atherosclerosis (55).

**Hypertension and Vascular Mortality**

A correlation between mortality and high BP has been extensively proven for those in late life until around the age of 80 years. Data from epidemiological surveys in populations aged 65 and older have shown that global cardiovascular mortality is 2.5- to 3-fold higher in hypertensive than in normotensive subjects (77,78). This excess of mortality is mainly correlated with SBP (33,78,79).

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However, in very old populations, as reviewed by Forette (80, 81), several studies have shown paradoxical relationships between BP values and mortality. Indeed, some authors have reported either no or an inverse association between BP and mortality in people aged older than 80 years (82, 83). In people older than 75 years, a 10-year survey showed that low initial DBP was predictive of higher mortality (84, 85). In the Adventist Health Study, the subjects were all 84 years and older. Cardiovascular and all-cause mortality were registered in an 11,828 person-year follow-up study. The apparent effects of hypertension were small unless subjects were currently taking antihypertensive medication (86). In the Helsinki Aging Study, where subjects were 80 years and older, there was an inverse relation between BP and 5-year mortality, after controlling for age, gender, and clinically significant diseases (87).

Yet, the association between elevated BP and reduced cardiovascular as well as all-cause mortality in the oldest-old individuals has been challenged. Higher DBP might be needed to provide an appropriate blood flow in the heart, brain, and coronary arteries during diastole because of severe atherosclerosis. Alternatively, low BP might be the result of underlying illness (88–92). In a population of 3657 elderly residents of East Boston, higher SBP predicted a linear increase in cardiovascular and total mortality after adjustment for confounding variables (including frailty and disorders such as congestive heart failure and myocardial infarction) and after exclusion of deaths within the first 3 years of the 10.5-year follow-up. After the same adjustments, higher DBP predicted linear increases in cardiovascular but not in total mortality (90, 91). In the Framingham study, subjects aged older than 75 years were divided into two groups (92). In those with cardiovascular disease at the biennial examination, there was in men a distinct U-shaped curve of cardiovascular mortality rate in relation to SBP, with a substantial increase in mortality rate below an SBP of 120 mm Hg for both men and women. In the sample free of cardiovascular disease, there were increasing cardiovascular morbidity and mortality rates with increasing BP levels for both men and women. The conclusion was that the excess mortality rate reported for low BP levels in persons older than 75 years derives from the inclusion in the sample of the substantial proportion of this age group who have cardiovascular disease.

These explanations, however, are not totally convincing. The populations they are based on are not strictly limited to old-old individuals. To explore the possible reasons of the paradoxical survival of hypertensive elderly persons, all-cause and cardiovascular mortality were analyzed in 795 persons aged 75 to 96 years and followed prospectively for 3 years in the Rancho Bernardo Chronic Disease Study (85). Survival analyses showed a significant trend for improved survival with increasing DBP in men aged 80 years and older versus all-cause mortality and cardiovascular mortality. Results were not explained by a wide range of biological and historical factors, such as differences in the use of antihypertensive medication, pulse pressure, history of hypertension, history of coronary heart disease, isolated systolic hypertension, interval change in DBP over an average of 12 years, or by cholesterol, triglycerides, fasting plasma glucose, smoking, or body mass index.

Conversely, in a 5-year Dutch longitudinal study of community residents aged 85 years and older, the observed inverse correlation between BP and mortality disappeared after adjustment for indicators of poor health (93). After adjustment for health status, neither SBP nor DBP was correlated with all-cause mortality, although a significant positive relation was observed between higher DBP and an increased risk of dying from stroke and all cardiovascular causes.

In conclusion, a positive relationship between BP and survival of subjects aged 85 years and older has been demonstrated in several longitudinal studies. To date, the role of potential confounding factors that might explain this relationship is differently appreciated, but it is clear that relationships between hypertension and survival are less straightforward in oldest-old individuals than in other age groups.

**Diagnosis of Hypertension**

In all ages, the diagnosis needs repeated measurements in resting conditions, without anxiety, coffee, alcohol, or tobacco. However, misdiagnosis may be more frequent in elderly persons, especially in women, due to several factors. The length of the cuff may be inappropriate in case of obesity or very low weight. Baroreceptor reflex sensitivity is reduced with age; therefore, fluctuations in BP, such as postural hypotension, may occur more commonly in elderly individuals (10). Fluctuations with anxiety (white coat effect) and physical training are also more frequent in this population (94). Older patients may have rigid arteries as a result of atherosclerosis that increase the measured BP. However, a pulseless palpable artery is not as reliable an indicator of pseudohypertension as previously recognized (95, 96). The difficulties in assessing BP should encourage the development of ambulatory measurement methods in elderly patients (97).

Essential hypertension is the most common cause of hypertension in this population. However, secondary hypertension is more prevalent in elderly than in younger subjects. Renovascular hypertension is common in older people and should be considered in those whose hypertension appears refractory to multiple-drug therapy, and in patients with renal insufficiency of unknown cause (98).

**The Benefits of Treatment in Elderly Persons**

The following randomized, double-blind placebo-controlled intervention studies provide strong evidence in favor of treating hypertension in elderly patients: the Veterans Administration Study (99), the Management Australian therapeutic trial (100), the Hypertension Detection and Follow-up Program (79), the Medical Research Council (MRC) trial (101), the Swedish Trial in Old Patients with Hypertension (STOP-Hypertension) (102), the European Working Party on High Blood Pressure in the Elderly (EWPHE) report (103), the Syst-Eur Vascular Dementia Project (Syst-Eur) (62), and Syst-China trial (104). First-outcome trials that focused attention on systolodiastolic hypertension show a reduction in cardiovascular and cerebrovascular morbidity and mortality in patients aged older than 65 years. In the EWPHE trial, 840 hypertensive patients aged older than 60...
years were assigned to diuretic treatment or placebo. The EWPHE trial showed that active treatment was associated with a 27% reduction in cardiovascular mortality, a 60% reduction in fatal myocardial infarctions, a 52% reduction in strokes, and a significant reduction in the incidence of severe congestive heart failure. In the STOP-Hypertension trial, treatment with a diuretic or a β-blocker was compared with a placebo in more than 1600 hypertensive individuals between the ages of 70 and 84. There was a 47% reduction in strokes, a 73% reduction in stroke mortality, and a 43% reduction in total mortality. In the MRC trial, which enrolled 4000 hypertensive patients aged between 65 and 74 years, there was a 31% reduction in strokes, a 44% reduction in coronary events, and a 35% reduction in all cardiovascular events.

More recent trials specifically addressed the treatment of pure isolated hypertension. The SHEP trial (9) assigned individuals with ISH, and whose average age was 72 years, to either diuretic therapy (plus a β-adrenergic blocker or reserpine, if needed) or placebo. A 37% reduction in nonfatal strokes, a 32% decrease in cardiovascular events, a 33% decrease in nonfatal myocardial infarctions, and a 55% reduction in heart failure were observed in the treated group in comparison with the placebo group. The analysis by Kostis and colleagues (105) shows that heart failure was reduced in the SHEP cohort, whether or not subjects had a history or electrocardiographic evidence of prior myocardial infarctions. In the Syst-Eur trial, patients aged 60 years and older with ISH received the calcium blocker nitrendipine as a first-line treatment. Compared with placebo controls, patients receiving active treatment experienced a 42% reduction in the total number of strokes, a 44% reduction in nonfatal strokes, a 31% decrease in fatal and nonfatal cardiovascular endpoints, and a 26% decrease in fatal and nonfatal cardiac endpoints, including sudden death. However, there was only a nonsignificant 30% reduction in fatal and nonfatal myocardial infarcts (32). A meta-analysis by Staessen and colleagues (34) shows that in 15,693 patients with ISH, collected from eight trials, including those aged in their 80s, antihypertensive treatment reduced the incidence of strokes by 30%. Total mortality was also decreased by 13%, cardiovascular mortality by 18%, all cardiovascular complications by 26%, and coronary events by 23%. Treatment was more effective to prevent strokes than coronary events. Because SBP at entry was correlated with strokes and total mortality while DBP was not, it was suggested that the benefit of treatment was essentially attributable to the reduction in SBP (Table 1).

The SHEP study, the MRC trial, and the Syst-Eur trial show that reducing SBP in older persons with stage 2 and 3 ISH (SBP >160 mm Hg and DBP <90 mm Hg) reduced morbidity and mortality. It has yet to be determined whether treatment in patients with stage 1 ISH (SBP 140–159 mm Hg and DBP <90 mm Hg) will also be beneficial.

**Should Very Old People Be Treated?**

The value of antihypertensive treatment is well established when pooling patients aged 60 or 65 years and older (106–109). The results are more discordant when focusing on the upper age class, 80 or 84 years and older (103). The EWPHE study failed to demonstrate a significant benefit after the age of 80 (110,111). In the STOP-Hypertension trial (102), treatment produced less reduction in stroke, myocardial infarction, and other cardiovascular deaths in older patients, and the upper age limit was 84 years. In contrast, in the SHEP trial (9), the effect of active treatment compared with placebo on the relative risk of stroke increased with age and reached its maximum in the group of patients aged 80 years or older. The Syst-Eur trial evidences a probable reduction in morbidity, but not in mortality, in the oldest patients (32,112). An important finding in the Syst-Eur trial (113) is that active treatment significantly reduced by half the incidence of dementia, from 7.7 to 3.8 per 1000 persons. In a nonrandomized 3-year follow-up of 1810 persons aged 75 and older (mean age 83 y), subjects taking antihypertensive treatment had a significantly (relative risk [RR] = .7) reduced incidence of dementia (114). On the contrary, no reduction in dementia is shown in the SHEP study. The meta-analysis of data from 1670 patients aged 80 years and older (115) suggests that treatment prevented 34% of strokes (95% confidence interval, 8%–52%). Rates of major cardiovascular events and heart failure were significantly reduced in the treated group relative to the placebo group.

### Table 1. Overview of Controlled Trials on the Effects of Antihypertensive Treatment in Elderly Persons

<table>
<thead>
<tr>
<th>Trials (Reference)</th>
<th>Type of Trial</th>
<th>Duration of Follow-up (y)</th>
<th>No. Patients (Treated/Placebo)</th>
<th>Stroke Risk Reduction (% Treated vs Control)</th>
<th>All Cardiovascular Disease Reduction (% Treated vs Control)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EWPHE (110)</td>
<td>Multicentric, randomized, double-blind vs placebo</td>
<td>4.6</td>
<td>840 (416/424) ≥60</td>
<td>36</td>
<td>29</td>
</tr>
<tr>
<td>STOP-Hypertension (102)</td>
<td>Multicentric, randomized, double-blind vs placebo</td>
<td>2</td>
<td>1627 (812/815) 70–84</td>
<td>47</td>
<td>40</td>
</tr>
<tr>
<td>MRC Working Party (101)</td>
<td>Randomized, blind vs placebo</td>
<td>5.8</td>
<td>4396 (2183/2213) 65–74</td>
<td>25</td>
<td>17</td>
</tr>
<tr>
<td>SHEP Cooperative Research Group (9)</td>
<td>Multicentric, randomized, double-blind vs placebo</td>
<td>4.5</td>
<td>4736 (2365/2371) ≥60</td>
<td>33</td>
<td>32</td>
</tr>
<tr>
<td>Syst-Eur (32)</td>
<td>Multicentric, randomized, double-blind vs placebo</td>
<td>2</td>
<td>4695 (2398/2297) ≥60</td>
<td>38</td>
<td>26</td>
</tr>
<tr>
<td>Syst-China (104)</td>
<td>Multicentric, randomized, double-blind vs placebo</td>
<td>2</td>
<td>2394 (1253/1141) ≥60</td>
<td>38</td>
<td>37</td>
</tr>
</tbody>
</table>

*Note: EWPHE = European Working Party on High Blood Pressure in the Elderly; STOP-Hypertension = Swedish Trial in Old Patients with Hypertension; MRC = Medical Research Council; SHEP = Systolic Hypertension in the Elderly Program.*
decreased by 22% (RR = .77, p = .03) and 39% (RR = .58, p = .01), respectively. On the other hand, there was no reduction in cardiovascular deaths or total mortality. Large-scale specific trials are needed for definite conclusions on the benefit of treatment in very old patients, although the favorable results on morbidity may argue against a threshold beyond which hypertension should not be treated.

More information is needed. We hope it will be provided by the Hypertension in the Very Elderly Trial (HYVET) study, which is currently assessing the ability of antihypertensive drug treatment to reduce the risk of stroke and other cardiovascular endpoints in patients aged more than 80 years (116). Subsidiary projects in the trial, which examine quality of life and cognitive function, will enlighten the overall benefits and risks for this age group in which quality is as important as quantity of life. Until we have the results of the HYVET and other studies in large groups of subjects aged older than 80 years, available data suggest that, even if it does not prolong life expectancy, antihypertensive treatment in very old patients might preserve the quality of life by preventing nonfatal strokes. In the absence of certainty, each particular case must be carefully assessed, and the possible benefits of antihypertensive treatment weighed against the risks, which tend to increase with age.

The safety, as well as the benefit, of treating elderly patients with stage 1 ISH (SBP 140–159 mm Hg and DBP <90 mm Hg) remains to be determined by ongoing and planned trials.

What Should Be the Goal BP Levels in Elderly Persons?
Controversy still exists on the goal BP level. Indeed, according to the J-shaped curve hypothesis, a major reduction in DBP (<65 mm Hg) might be associated with an increase in mortality, especially in elderly patients and in patients with ischemic cardiopathy. In hypertensive elderly individuals, lowering DBP might jeopardize appropriate blood flow in the brain, heart, and kidney during the diastole. However, low DBP was not responsible for an increase in mortality in the SHEP study (117).

The JNCVI goal for BP control (SBP <140 mm Hg and DBP <90 mm Hg) seems too strict to achieve in elderly individuals. It has been recommended either to decrease SBP to <160 mm Hg and DBP to <90 mm Hg as an intermediate goal BP (6), or to obtain a 20-mm Hg decrease under the initial BP, as proposed in the Syst-Eur trial (32).

The Hypertension Optimal Treatment (HOT) study also tried to determine the ideal BP values in 19,000 hypertensive patients who were given a felodipine-based regimen. The most favorable BP level in this study was 139/86 for mortality and 139/83 for overall morbidity (118).

Treatment
Nonpharmacological Intervention
Older hypertensive patients can also benefit from nonpharmacological interventions to lower BP (1,6). Weight control is important because the volume associated with obesity contributes to the development of left ventricular hypertrophy and congestive heart failure. Other interventions should include reduction of excessive alcohol consumption, smoking cessation (119), and exercise training. It has been shown that a walking program lowered DBP by 12 mm Hg on average and was more effective than more intensive exercise training (120). Any reduction of dietary sodium should be approached with caution in older people (121,122), because this can induce a reduction in food intake.

Influence of the Class of Drugs
The majority of the trials used diuretics and β-blockers as first-line drugs. The efficacy and the safety of these two classes of drugs have been demonstrated in elderly individuals (8,101,102,110,123,124). The benefit of calcium antagonists (32,104,125–127) and angiotensin-converting enzyme (ACE) inhibitors (127,128) has been evidenced more recently in the prevention of cardiovascular and cerebrovascular complications in older patients.

Blacher and colleagues (129) have highlighted the role of pulse pressure as a major cardiovascular risk, and they have emphasized the need for randomized trials with antihypertensive drugs acting differently on the pulsatile component of BP. Those authors have suggested that vasopeptidase inhibitors and nitric oxide donors may possibly increase the distensibility of large arteries and reduce pulse pressure. Smulyan and Safar (130) also underline the interest of new drugs that could increase aortic distensibility and decrease SBP without substantially reducing DBP.

At least 36 trials are now in progress (131) and should help to give further information on some still unanswered questions.

Pharmacological Strategies
Because age- and disease-associated factors affect the metabolism and distribution of pharmacological agents, these factors should be considered when selecting antihypertensive therapy. Low doses of medication should be given and slowly adjusted. However, despite alterations in metabolism, most elderly patients tolerate medications without a significant increase in adverse events compared with younger patients or control groups. First-line treatment should be diuretics or β-blockers (JNCVI). In ISH, diuretics and calcium antagonists are recommended (8,32,112).

Concomitant diseases may influence the choice of therapy. In patients with coronary artery diseases, β-blockers may be useful, but peripheral artery disease, heart failure, or obstructive bronchopathy may limit their use at advanced age (132). The advantage of calcium antagonists in older patients with coronary artery diseases is still questioned (133,134). In cardiac dysfunction and congestive heart failure, diuretics, ACE inhibitors, or both are appropriate initial choices. In recent German studies, a low-dose reserpine-thiazide combination appeared to be cost effective, compared with ACE inhibitors or calcium-channel-blocker conventional monotherapy, but the groups were small, the follow-up duration was very short (6 wk), and the mean age of patients was only 58 years (135,136). Reserpine was extensively used in early hypertension trials, but its central side effects of sedation and severe depression in persons given high doses has led to the drug being seldom prescribed today.
An older hypertensive individual is also more likely to have impaired renal function than a younger patient. A careful adjustment of BP may be necessary to avoid major reductions of renal blood flow, which may further impair renal function.

Fixed-dose therapy has an advantage for elderly persons in that it may help to increase compliance, achieve a higher response rate, and reduce the cost of treatment (98).

Management of Hypertension in General Populations

Although clinical trials provide evidence that treating patients with elevated SBP can yield significant benefits in terms of reduced morbidity and mortality, management of hypertension in the community is disappointing. Only around 15% of the patients are adequately treated (137). Less than 30% of patients on hypertensive drugs attain the JNCVI goal for BP control (SBP <140 mm Hg and DBP <90 mm Hg), either in the United States or in Europe (3,118,138). Indeed, achieving the DBP goal is within reach, but reaching the SBP goal is much more difficult (18). A survey in the United Kingdom found that physicians were less likely to treat elevated SBP than DBP at any patient age (139). The HOT trial shows that physicians can successfully reduce DBP to <90 mm Hg in more than 90% of patients when they participate in a clinical trial. SBP is less likely to be lowered, even with the use of a three-drug regimen (118). The same conclusion is shown by the Anti-hypertensive and Lipid Lowering Treatment to Prevent Heart Attack trial (140) and the Controlled Onset Verapamil Investigation of Cardiovascular Events (CONVINCE) trial (141). Black (18) insisted on shifting our focus from DBP to SBP when determining therapeutic goals. Moreover, the new indication of pulse pressure as the most powerful risk factor must lead physicians to consider SBP rather than DBP when determining treatment goals, and the development of drugs that more adequately lower SBP should be promoted (18,34,130).

Acknowledgments

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108. RIGAUD AND FORETTE


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Editor Nominations

Journal of Gerontology: Biological Sciences

The Gerontological Society of America’s Publications Committee is seeking nominations for the position of Editor of the *Journal of Gerontology: Biological Sciences.*

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Nominations and applications may be made by self or others, but must be accompanied by the candidate’s curriculum vitae and a statement of willingness to accept the position. **All nominations and applications must be received by May 1, 2001.** Nominations and applications should be sent to the GSA Publications Committee, Attn: Jennifer Campi, The Gerontological Society of America, 1030 15th Street, NW, Suite 250, Washington, DC 20005-1503.