HYPERTENSION Canada

Published by the Canadian Hypertension Society

March 2003
Bulletin No. 74

ALLHAT: Unexpected Results?

by Frans Leenen

Large-scale randomized clinical trials performed in the 1970s and 1980s established that antihypertensive drug treatment reduces rates of strokes to the extent one would anticipate based on the observed decreases in blood pressure (BP). However, decreases in CHD death or nonfatal myocardial infarction (MI) were less than expected. One of the possible explanations put forward for this relative failure was that adverse effects of the study drugs—particularly the negative metabolic effects of diuretics—offset the potential benefit of BP reduction.

In the 1980s, new classes of antihypertensive drugs emerged, including angiotensin-converting enzyme (ACE) inhibitors, calcium-channel blockers (CCBs) and alpha$_1$-blockers with putative cardiovascular (CV) protective, anti-atherosclerotic actions. Despite the absence of trial evidence for better cardiac outcomes with these new agents, many physicians switched their management strategies for hypertension from diuretic-based regimens to ACE inhibitors and/or CCBs, particularly in older hypertensive patients at high risk for CV events.

A statement from the CHEP 2002 Recommendations Committee regarding ALLHAT and its impact on the 2002 Recommendations review process appears on page 8.

The Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) was designed in 1993/1994 to directly test the rationality of this shift in management strategies. Its primary hypothesis was that the combined incidence of fatal CHD and nonfatal MI would be lower in hypertensive patients randomized in a double-blind design to: 1) a calcium antagonist (amlodipine); 2) an ACE inhibitor (lisinopril); or 3) an alpha-adrenergic blocker (doxazosin) as first-line therapy than in those in whom a similar degree of BP control is achieved using a thiazide-like diuretic (chlorthalidone) as first-line therapy. To maximize statistical power, 1.7 times as many patients were assigned to the diuretic arm compared to each of the other three arms.

Continued on page 2

Table of contents

Published by the

Canadian Hypertension Society

 Hypertension Canada is published four times a year, in an English and a French edition, by STA HealthCare Communications Inc, 955 Boul. St. Jean, Suite 306, Pointe-Claire, Quebec H9R 5K3. Subscriptions are available upon request to anyone interested in hypertension. All articles published in Hypertension Canada are the sole responsibility of the authors, and do not necessarily reflect the opinions of the Canadian Hypertension Society or STA HealthCare Communications Inc. Legal delivery first quarter 2003. Articles may be submitted, in either English or French, to the Editorial Office, Hypertension Canada, The Toronto Western Hospital, University Health Network, 399 Bathurst St, Toronto, Ontario, M5T 2S6. FAX: (416) 603-7919. Deadline for submissions for the next issue is April 15, 2003. Canadian Publications Mail Sales Product Agreement #40063348.

ALLHAT

Continued from page 1

Secondary outcomes included all-cause mortality, stroke, combined CHD, combined CVD (including fatal, hospitalized or treated non-hospitalized CHF), progression of renal dysfunction and cancer, as well as the "safety outcomes" of hospitalization for gastrointestinal (GI) bleeding and angioedema. Pre-specified subgroups included older (≥ 65 years of age) patients, black patients, women and patients with diabetes. To evaluate differences in effects on CHD/CVD between the various first-step drugs, ALLHAT was designed with a large sample size (9,000-15,000 participants/intervention arm) and long follow-up (4-8 years). The doxazosin arm of the trial was terminated early when chlorthalidone was found to be superior in preventing CV events.

ALLHAT was designed as a practice-based, randomized clinical trial in high-risk hypertensive patients, of whom about half are women and half are ethnic minorities (especially black patients, who represented 35% of the study population). Main eligibility criteria included: age of 55 years or older; systolic or diastolic hypertension; and at least one other risk factor for CHD (evidence for atherosclerotic disease or type II diabetes), while exclusion criteria included a recent (*i.e.,* within past 6 months or less) MI or stroke, symptomatic CHF and/or ejection fraction < 35%. To achieve goal BPs of < 140/90 mmHg, first-line therapies were to be increased to 10 mg/day (amlodipine), 40 mg/day (lisinopril) and 25 mg/day (chlorthalidone), followed by the addition of open-label step-two (reserpine, clonidine, or atenolol) or step-three (hydralazine) drugs.

Study outcomes were assessed at regular (every 3-4 months) follow-up visits and reported to the Clinical Trials Center. For hospitalized events, copies of death certificates and hospital discharge summaries were requested (which were obtained in 99% of all cases of CVD events). In addition, biennial study electrocardiograms (ECGs) were done, as were searches of relevant databases for

Continued on page 4

Blood Pressure and Cognitive Function: Emerging Evidence for Added Benefits of Hypertension Control

by J. David Spence

Treatment of hypertension is the most effective way of preventing stroke; effective blood pressure (BP) control reduces the risk of stroke by about half. Recently, it has become apparent that effective treatment of hypertension also reduces cognitive decline. While a great deal of distinction was made in the past between vascular dementia and Alzheimer's dementia, it appears that this distinction is becoming blurred.

High BP itself causes damage to the small resistance vessels at the base of the brain, leading to hyaline degeneration and fibrinoid necrosis. This, in turn, leads to lacunar infarctions and intracerebral hemorrhages in the internal capsule, basal ganglia, thalamus, brainstem and cerebellum. These strokes can be virtually eliminated by effective control of BP.[1] In the North American Symptomatic Carotid Endarterectomy Trial (NASCET), intracerebral hemorrhages were reduced to 0.4% of strokes by careful attention to BP control.[2]

The Nun Study[3] followed about 700 nuns in a religious order in the Midwestern United States. Subjects agreed prospectively to have an autopsy at the time of death, and underwent annual testing of their cognitive function. When the results were reported, 102 autopsies had been carried out in women aged 76 to 100 years. Of these, 61 had Alzheimer's disease (AD) and 41 did not. Not all of the women who had pathological evidence of AD had been demented during life, but if they had even one or two small lacunar infarctions, the odds ratio for dementia was 20.7 (95% CI; 1.5-288). Among those without AD, dementia was only weakly associated with brain infarctions.

Subsequently, the Systolic Hypertension in Europe (Syst-Eur) trial showed that treatment of isolated systolic hypertension reduced AD dementia by half, from 7.7 to 3.8 cases per 1,000 patient-years.[4]

Recently, the Study on Cognition and Prognosis in the Elderly (SCOPE)[5] showed that, among elderly patients with mild to moderate hypertension, candesartan reduced non-fatal strokes and progression of cognitive decline among patients whose baseline mini-mental-state examination (MMSE) score was between 24 and 28. In the Perindopril Protection Against Recurrent Stroke Study (PROGRESS)[6] of perindopril ± indapamide in 6,105 individuals with previous stroke or transient ischemic attack (TIA), active treatment was associated with a 28% relative reduction in risk (compared to placebo) for subsequent cerebrovascular events. Individuals who experienced a cerebrovascular event while receiving active treatment had a 34% relative reduction in risk (compared to subjects receiving placebo who experienced a subsequent cerebrovascular event) for the development of dementia and a 45% relative reduction in risk for severe cognitive decline.

Thus, effective control of BP prevents not only stroke, but also dementia. Since the elderly fear dependence more than death, it does our elderly patients no favour to leave their BPs uncontrolled.

High BP itself causes damage to the small resistance vessels at the base of the brain, leading to hyaline degeneration and fibrinoid necrosis. This, in turn, leads to lacunar infarctions and intracerebral hemorrhages in the internal capsule, basal ganglia, thalamus, brainstem and cerebellum. These strokes can be virtually eliminated by effective control of BP.

J. David Spence, MD, FRCPC, Stroke Prevention & Atherosclerosis Research Centre, Robarts Research Institute, London, Ontario.

References

1. Spence JD. Cerebral consequences of hypertension. In: Laragh JH, Brenner B (eds). Hypertension: Pathophysiology, Diagnosis, and Management. Raven Press, New York, 1995. pp. 741-53.
2. Barnett HJ, Taylor DW, Eliasziw M, et al. Benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. N Engl J Med 1998; 339(20):1415-25.
3. Snowdon DA, Greiner LH, Mortimer JA, et al. Brain infarction and the clinical expression of Alzheimer disease: The Nun Study. JAMA 1997; 277:813-7.
4. Forette F, Seux ML, Staessen JA, et al. Prevention of dementia in randomised double-blind placebo-controlled Systolic Hypertension in Europe (Syst-Eur) trial. Lancet 1998; 352(9137):1347-51.
5. Hansson L, Lithell H, Skoog I, et al. Study on Cognition and Prognosis in the Elderly (SCOPE): baseline characteristics. Blood Press 2000; 9(2-3):146-51.
6. PROGRESS Collaborative Group. Randomized trial of a perindopril-based blood-pressure-lowering regimen among 6105 individuals with previous stroke or transient ischemic attack. Lancet 2001; 358:1033-41.

ALLHAT

Continued from page 2

study outcomes. ALLHAT did not have a formal adjudication of study events; rather, an endpoints subcommittee was established for validation of physician's diagnoses in a random (10%) subset of hospitalized CHD and stroke events. A *post-hoc* validation for all (first event) hospitalized and fatal cases of CHF is underway.

Changes in BP

At baseline, 90% of patients were receiving antihypertensive treatment, but only 27% had adequately controlled BP (defined as systolic BP < 140 mmHg and diastolic BP < 90 mmHg). Control rates improved to 55% after one year and to 66% after five years in the study. In Canada, adequate control of BP was present in only 22% of participants at baseline, but improved markedly during the study to 61% after one year and 75% after five years. This improvement in control rates was due to a progressive increase in the number of antihypertensive drugs used (Figure 1). These findings are of major significance, demonstrating that physicians are able to dramatically improve BP control in a structured setting with regular feedback on BP control in their own versus other practices. This is made even more remarkable given that recommended treatment strategies were rather restrictive (see above).

The baseline BP average of ALLHAT patients was 146/84 mmHg (note that the study did not include a wash-out period). During the study, the average BP decreased progressively over time to reach 134-136/75-77 mmHg after three to four years. Systolic BP remained the highest in the lisinopril arm: higher than the chlorthalidone arm for all lisinopril patients by about 2 mmHg, for older lisinopril patients by 3 mmHg and for black lisinopril patients by 4 mmHg. Overall BP control was best in the chlorthalidone arm (68.3% adequately controlled), followed by the amlodipine arm (66.3%) and the lowest in the lisinopril arm (61.2%). Persistence of double-blind steps or equivalent treatment was high in the chlorthalidone and amlodipine arms (80.5% and 80.4%, respectively, after five years) and clearly lower in the lisinopril arm (72.6%).

Figure 1

Number of Antihypertensive Drugs Used and BP Control (< 140/90 mmHg) Over Course of ALLHAT

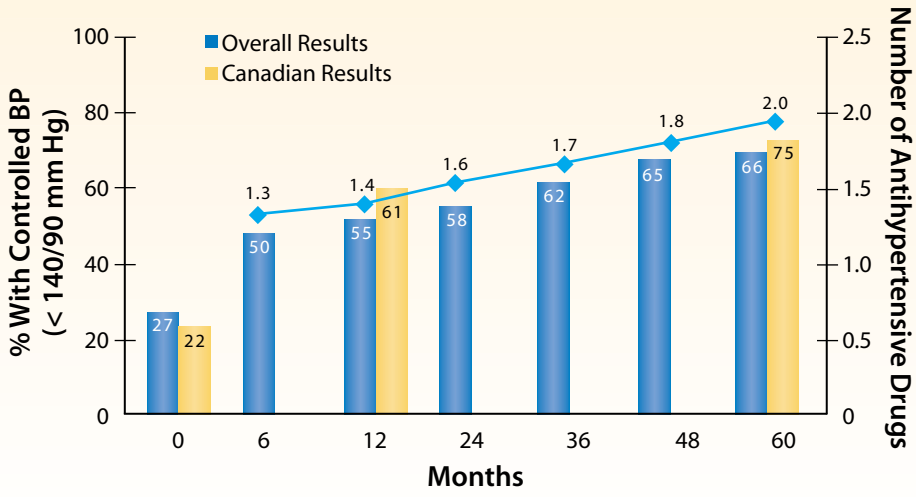
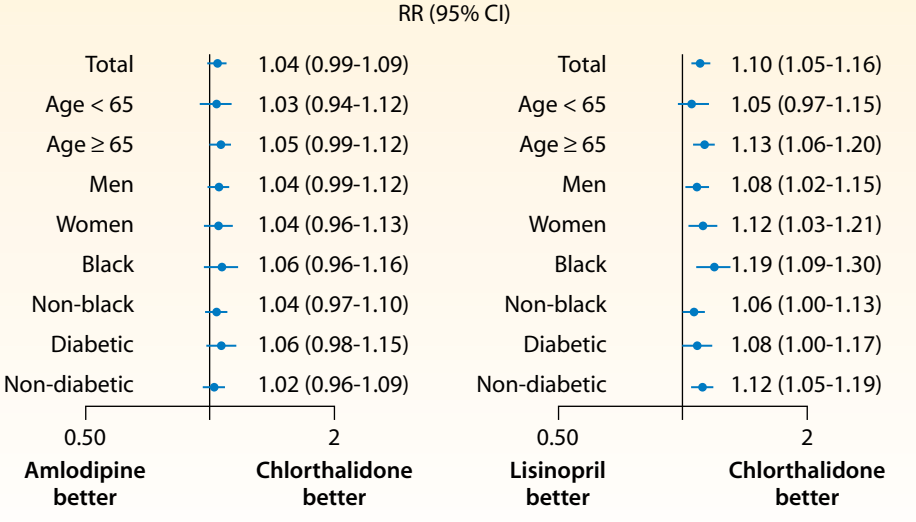


Figure 2

Combined CVD in ALLHAT: Subgroup Comparisons



95% CI; p = 0.04 for interaction

Main Outcomes

After up to seven years of follow-up (average 4.9 years), cumulative event rates for the primary outcome (fatal CHD and non-fatal MI) were the same for the three treatments: relative risk (RR) 0.98 (0.90-1.07) for amlodipine compared to chlorthalidone and RR 0.99 (0.91-1.08) for lisinopril compared

to chlorthalidone. All-cause mortality rates also were the same. In contrast, fatal and non-fatal stroke rates tended to be lower in the amlodipine group (RR 0.93, 0.81-1.06) and were significantly increased in the lisinopril group (RR 1.15, 1.02-1.30). Event rates for heart failure were increased in the amlodipine group (RR 1.38, 1.25-1.52) and the lisinopril group (RR 1.19, 1.07-1.31) compared to the chlorthalidone group. Cancer-rates were similar for the three treatment-group, while GI bleeding rates tended to be lower in the amlodipine group (RR 0.92, 0.82-1.03) and higher in the lisinopril group (RR 1.11, 0.99-1.24) compared to the chlorthalidone group. For none of the outcomes, nor for any of the patient subgroups (including diabetics, patients with CHD at baseline, and patients older or younger than 65 years of age) did lisinopril outperform chlorthalidone. Figure 2 compares the incidence of combined CVD between ALLHAT's pre-specified subgroups in each treatment arm. In younger (< 65 years of age) and non-black patients, chlorthalidone remained similar to or better than lisinopril, even with nearly equal BP control (see above).

Conclusions

ALLHAT showed that amlodipine and lisinopril have effects equivalent to chlorthalidone on CHD and total mortality in older patients with mild to moderate hypertension and CHD risk factors (*i.e.,* it is a primary and secondary prevention trial). The results strongly refute concerns raised about the safety of dihydropyridine (DHP) CCBs: amlodipine was not associated with increases in cancer or GI bleeding, nor with excess MI rates in any subgroup (including patients with diabetes or CHD at baseline). The trial also did not show advantages for lisinopril over chlorthalidone for CV protection; on the contrary, stroke rates were significantly higher in the lisinopril group (only in black patients), as were combined CVD rates (in black and, to a lesser extent, non-black patients).

Heart failure rates were significantly lower in the chlorthalidone group compared to the amlodipine and lisinopril groups. The diagnosis of heart failure in ALLHAT was based on criteria (see Table 1) similar to those used in the Systolic Hypertension in the Elderly Program (SHEP). This represents a rather "soft" diagnosis, confounded by possible side-effects of amlodipine (*e.g.,* ankle edema) or lisinopril (*e.g.,* nocturnal cough/dyspnea). All cases of hospitalized CHF are being reviewed for validation.

Irrespective of the CHF validation, ALLHAT highlights three major points relating to high-risk, older (≥ 55 years) hypertensive patients:

- thiazide-like diuretics remain unsurpassed as first-step therapy for BP control and prevention of CV events;
- once-daily DHP CCBs are similar to thiazide-diuretics (except, it is likely, for CHF), with no safety concerns; and
- ACE inhibitors have no special cardioprotective effects and, if anything, are somewhat less effective in this regard than chlorthalidone.

Table 1

ALLHAT Criteria for HF Diagnosis

The diagnosis of heart failure (HF) in ALLHAT was based on criteria similar to those used in the Systolic Hypertension in the Elderly Program (SHEP). Diagnosis required that patients exhibited at least one sign or symptom from each category below:

Category A

Paroxysmal nocturnal dyspnea
Dyspnea at rest
NYHA classification III
Orthopnea

Category B

Rales
Ankle edema
Tachycardia
Cardiomegaly by CXR
CXR characteristic of CHF
S_3 gallop
Jugular venous distention

Adapted from: Piller LB, Davis BR, Cutler JA, et al. Curr Control Trials Cardiovasc Med 2002; (1):10.

Continued on page 8

Special Announcement

Canadian Federation of Biological Societies: 46th Annual Meeting

Major theme: Cardiovascular & Metabolic Diseases
Date: June 11-14, 2003
Location: Ottawa Congress Centre
Ottawa, Ontario, Canda
Abstract Deadline: March 17, 2003
Contact: CFBS, 305-1750 Courtwood Crescent
Ottawa, Ontario, Canada K2C 2B5
Tel: 613-225-8889
Fax: 613-225-9621
Email: wantonious@cfbs.org

Format/Features:

- 11 symposia
- 4 plenary lectures
- 6 award lectures
- Employment Fair, Scientific Exhibits, Poster Sessions, Internet Café, Receptions, Societies' Banquets

Program information and online forms available on the CFBS website: *http://www.cfbs.org*

Real Risks or Made-up Myths? The Truth About Hot Tubs and Saunas for Hypertensive Patients

Effects of Immersion in a Hot Tub on Blood Pressure in Hypertensive Patients

by Tae Won Shin, Merne Wilson, and Thomas W. Wilson

As a medical resident at the University of Saskatchewan (and a swimmer), Dr. Tae Won Shin noticed signs at local swimming pools warning people with high blood pressure (BP) to "check with a physician" before using the hot tubs. There is little evidence in available literature on which to base such advice. Therefore, we decided to conduct this study ourselves.

We hypothesized that the reason for excluding hypertensives from hot tubs was heat-induced vasodilation, causing relative hypotension with organ dysfunction. We estimated that, upon hot-tub immersion, normotensive subjects would show a systolic BP fall of 10% and that hypertensives would experience twice this degree of decline. A sample size of 18 people per group gave us 90% power to detect this difference.

We recruited 23 normotensive subjects aged 19 to 83 years and 21 hypertensive subjects aged 47 to 78 years. Our local city-owned aquatic facility, The Harry Bailey Centre, provided the venue for the study. The Centre has two large (12-person) air-jet-fed hot tubs, each maintained at a temperature of 40°C.

Systolic and diastolic BPs and heart rate (HR) were measured with an Omron HEM 711-IC oscillometric device for 10 minutes at baseline, during 10 minutes of immersion in a hot tub, and for 10 minutes post-immersion. The results of these measurements are summarized in Table 1.

Changes in BP were evident within the first minute of immersion, and reached a plateau at between five and 10 minutes of immersion. No subject experienced more than a 30% decrease in systolic BP at any time. The HR response was slightly lower in the hypertensive group, possibly due to reduced baroreceptor function. Only two of the hypertensive subjects were taking beta-adrenergic blockers; removing them from the analysis did not negate the difference in HR response between the two groups.

Table 1

BP and Heart Rate (Initial Average Values and Maximal Changes)

Group	Initial SBP (mmHg)	Initial DBP (mmHg)	Initial HR (BPM)	Max ΔSBP (mmHg)	Max ΔDBP (mmHg)	Max ΔHR (BPM)
HT (*n* = 21)	145±4*	93±2*	83±2	-29±3	-26±1	+11±2*
NT (*n* = 23)	132±3	83±2	75±4	-27±3	-24±2	+16±0

HT = hypertensive subjects; NT = normotensive subjects; *p < 0.05 HT vs. NT

After leaving the hot tub, BP and heart rate returned towards baseline over 10 minutes for all subjects. No subject complained of any adverse clinical symptoms, such as dizziness, headache, nausea, palpitations or chest pain.

Therefore, in treated hypertensive patients and normotensive subjects alike, systolic BP falls by about 20% during 10 minutes of bathing in a hot tub. In a study done in the 1970s, Strandgaard[1] lowered BP acutely with ganglionic blockers and nitrates in groups of treated and untreated hypertensives as well as in normotensives. He found that all three groups could tolerate a 25% drop in systolic (or mean) arterial pressure before autoregulation of cerebral blood flow was impaired. Furthermore, a fall of 50% was necessary before symptoms occurred.

We conclude that most of our treated hypertensive patients can be reassured that hot tubs are safe.

Tae Won Shin, MD; Merne Wilson, RN, MSc; and Thomas W. Wilson, MD, FRCPC, Department of Medicine and Cardiovascular Risk Factor Reduction Unit, University of Saskatchewan, Saskatoon.

Reference

1. Strandgaard S. Autoregulation of cerebral blood flow in hypertensive patients. The modifying influence of prolonged antihypertensive treatment on the tolerance to acute, drug-induced hypotension. Circulation. 1976;53:720-727.

Benefits and Risks of Sauna Bathing

by R.I. Ogilvie

The sauna, or Finnish bath, has been a tradition in many parts of Canada for almost a century. A recent review by Hannuksela and Ellahham[1] summarizes the physiology and medical risks of sauna bathing. Unlike the Turkish bath, the sauna has dry air and a high temperature (80 to 100°C at the level of the bather's face and 30°C at floor level), with a relative humidity of 10 to 20%. Humidity is temporarily increased by throwing water on the hot rocks of the sauna heater. A good sauna has efficient ventilation, with air changing three to eight times per hour. A common ritual consists of several short stays of 5 to 20 minutes interspersed with cooling-off periods and followed by oral intake of fluids. Skin temperature rapidly increases to about 40°C; sweating begins early and reaches its maximum at about 15 minutes with an average loss of 0.5 kg per session. Skin blood flow becomes 50 to 70% of the total cardiac output while blood flow to internal organs decreases. Cardiac output increases by 60 to 70%, with an increase in heart rate (HR), while stroke volume is unchanged. The effect on blood pressure (BP) is variable. Frequent sauna bathing improves heat tolerance and reduces the magnitude of the changes. Sudden exposure to cold after the sauna activates the sympathetic nervous system, causing constriction of skin blood vessels. HR and stroke volume are decreased; BP and cardiac work are increased. Normal values are attained within a few hours.

Sudden Death, CHD and Heart Failure

According to the review by Hannuksela and Ellahham,[1] the risk of myocardial infarction (MI), coronary death and sudden death are lower during sauna bathing than with other daily activities. Of 6,175 sudden deaths over one year in Finland, 102 (1.7%) occurred within 24 hours of a sauna bath. One third of the deaths were accidental; the majority of non-accidental deaths were due to acute MI in which alcohol was an important contributing factor. In a study of 1,631 acute heart attacks or sudden deaths in Helsinki, 29 (1.8%) occurred within three hours of a sauna bath. In another prospective study (of 12,310 Finns), only two of 77 sudden coronary deaths over six years occurred in a sauna. Sauna bathing is well-tolerated by individuals with stable coronary disease, with fewer and more moderate ECG changes or perfusion defects than were observed with exercise. Alcohol consumption, however, can increase the risk of hypotension, fainting, arrhythmia and sudden hyperthermic death. Severe aortic stenosis, unstable angina pectoris or a recent MI are contraindications for sauna bathing. Cardiac arrhythmias are considered to be a relative contraindication to sauna bathing.

Sauna bathing has been generally contraindicated for patients with chronic heart failure. There have been studies, using lower temperatures, reporting improved vascular endothelial and cardiac function with repeated exposure in patients with chronic heart failure.[2] The effect of a stroke or transient ischemic attactk (TIA) on the vascular response to sauna bathing has not been studied. Sauna bathing should be avoided until neurological and cardiovascular systems have stabilized.

Hypertension

Most patients with hypertension apparently tolerate sauna bathing well, and experience hemodynamic changes similar to those observed in healthy subjects. Non-randomized studies of hypertensive patients have reported BP reductions of approximately 20/10 mmHg with regular sauna bathing. This is similar to or greater than the response that has been reported with regular aerobic exercise. There is less information about the effect of antihypertensive drugs. Older individuals taking multiple antihypertensive drugs who have orthostatic hypotension may be at higher risk for adverse events with sauna bathing. They may have a higher risk of syncope after the sauna session. Hypertensive pregnant women should not use the sauna bath, as uterine vascular resistance is increased.

Sauna bathing is safe and may be beneficial for most individuals with hypertension or stable coronary artery disease. Moderation in duration of sessions and of temperature, with habituation of regular repeated bathing, is recommended. There should be strict prohibition of alcohol, and a strong emphasis on adequate rehydration (with other liquids) after the session.

R.I. Ogilvie, MD, FRCPC, FACP, Hypertension Unit, Toronto Western Hospital, Toronto.

References

1. Hannuksela ML, Ellahham S. Benefits and risks of sauna bathing. Am J Med 2001;110:118-26.
2. Kihara T, Biro S, Imamura M et al. Repeated sauna treatment improves vascular endothelial and cardiac function in patients with chronic heart failure. J Am Coll Cardiol 2002;39:754-9.

ALLHAT

Continued from page 5

For the management of older hypertensive patients, the results from ALLHAT imply that thiazide-like diuretics should be the "preferred" drug of choice for first-step therapy. Once-daily DHP CCBs and, to a lesser extent, ACE inhibitors, are appropriate alternatives. However, the results of ALLHAT also strongly indicate that most hypertensive patients require more than one medication for BP control. Although the most optimal drug combinations remain to be established, diuretics should generally be part of such antihypertensive regimens based on current information.

Frans HH Leenen, MD, PhD, FRCPC, Principal Investigator for ALLHAT Region 9 (Canada), University of Ottawa Heart Institute.

References

1. Pill LB, Davis BR, Cutler JA, et al, for the ALLHAT Collaborative Research Group. Validation of Heart Failure Events in the Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) Participants Assigned to Doxazosin and Chlorthalidone. Curr Control Trials Cardiovasc Med 2002; 3(1):10.
2. Cushman WC, Ford CE, Cutler JA, et al, for the ALLHAT Collaborative Research Group. Success and Predictors of Blood Pressure Control in Diverse North American Settings: The Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT). J Clin Hypertens 2002; 4(6):393-404.
3. The ALLHAT Officers and Coordinators for the ALLHAT Collaborative Research Group. Major outcomes in high-risk hypertensive patients randomized to angiotensin-converting enzyme inhibitor or calcium channel blocker vs diuretic. The Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT). JAMA 2002; 288:2981-97.

Statement from the CHEP 2002 Recommendations Committee

The release of the ALLHAT results presented a special challenge for those involved in the yearly revisions of the Canadian Recommendations for the Management of Hypertension. With the appreciation that the ALLHAT results would be made available before the release of the newest revisions of the 2002 recommendations (but after the point at which the recommendations were ratified), the CHEP steering committee authorized a re-appraisal of those recommendations which would potentially be impacted by the results of ALLHAT. These recommendations included those related to:

- the management of hypertension in patients without other compelling indications for therapy;
- the management of patients with hypertension and diabetes; and
- the management of patients with hypertension and concurrent cardiovascular diseases.

This process of re-appraisal and re-voting on amended recommendations is expected to be completed by April 2003. The entire package of 2002 recommendations is expected to be released within several days following final ratification, via the Canadian Hypertension Society (CHS) website (*www.chs.md*).

Ross Feldman, MD, FRCPC, University of Western Ontario, London, Ontario.

The Canadian Hypertension Society has established an Internet home page at:

www.chs.md

Readers of Hypertension Canada are invited to visit the homepage,
and to submit suggestions on how its effectiveness may be improved.

HYPERTENSION Canada

is published by the

Canadian Hypertension Society