HYPERTENSION Canada

Published by the Canadian Hypertension Society

March 2005
Bulletin No. 82

2005 CHEP Recommendations

# Canadian Hypertension Education Program Recommendations: 2005 Update

2005 marks the sixth consecutive year for which the Canadian Hypertension Education Program (CHEP) has released updated recommendations for the management of hypertension. This year, the CHEP Evidence-based Recommendations Task Force focused on the evidence supporting expedited assessment of the hypertension-related risk of atherosclerotic disease as well as a more global atherosclerotic risk assessment. In addition, the 2005 recommendations support the increasingly held belief that, in the choice of antihypertensive drugs, the consideration of blood pressure (BP) control effectiveness supersedes the consideration of "pleiotropic" effects for the five major antihypertensive classes.

*Continued on page 2*

# A Brief History of Canadian Hypertension Recommendations

*by Norm RC Campbell, Denis Drouin and Ross Feldman*

Canada has a long history of developing hypertension recommendations, specifically evidence-based recommendations. The use of a systematic evidence-based approach has been both critiqued and heralded, but is recognized as the distinguishing feature of our recommendations.

The first national recommendations were developed by a committee consisting of Drs. Kuchel, Mahon, McKenzie and Ogilvie. The recommendations were sponsored by Health and Welfare Canada. They reviewed evidence up to 1977 for the usefulness of pharmacotherapy and the stepped-care approach. Subsequently, the Canadian Hypertension Society sponsored the creation of several sets of recommendations. The 1984 recommendation process involved a large multidisciplinary committee chaired by Dr. Alexander Logan. The process addressed three major issues: to determine if all hypertensive patients without target organ damage should

*Continued on page 5*

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 fourth quarter 2004. 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 2005. Canadian Publications Mail Sales Product Agreement #40063348.

## 2005 CHEP Recommendations

Continued from page 1

### New Key Messages

The new key messages identified in the 2005 recommendations are:

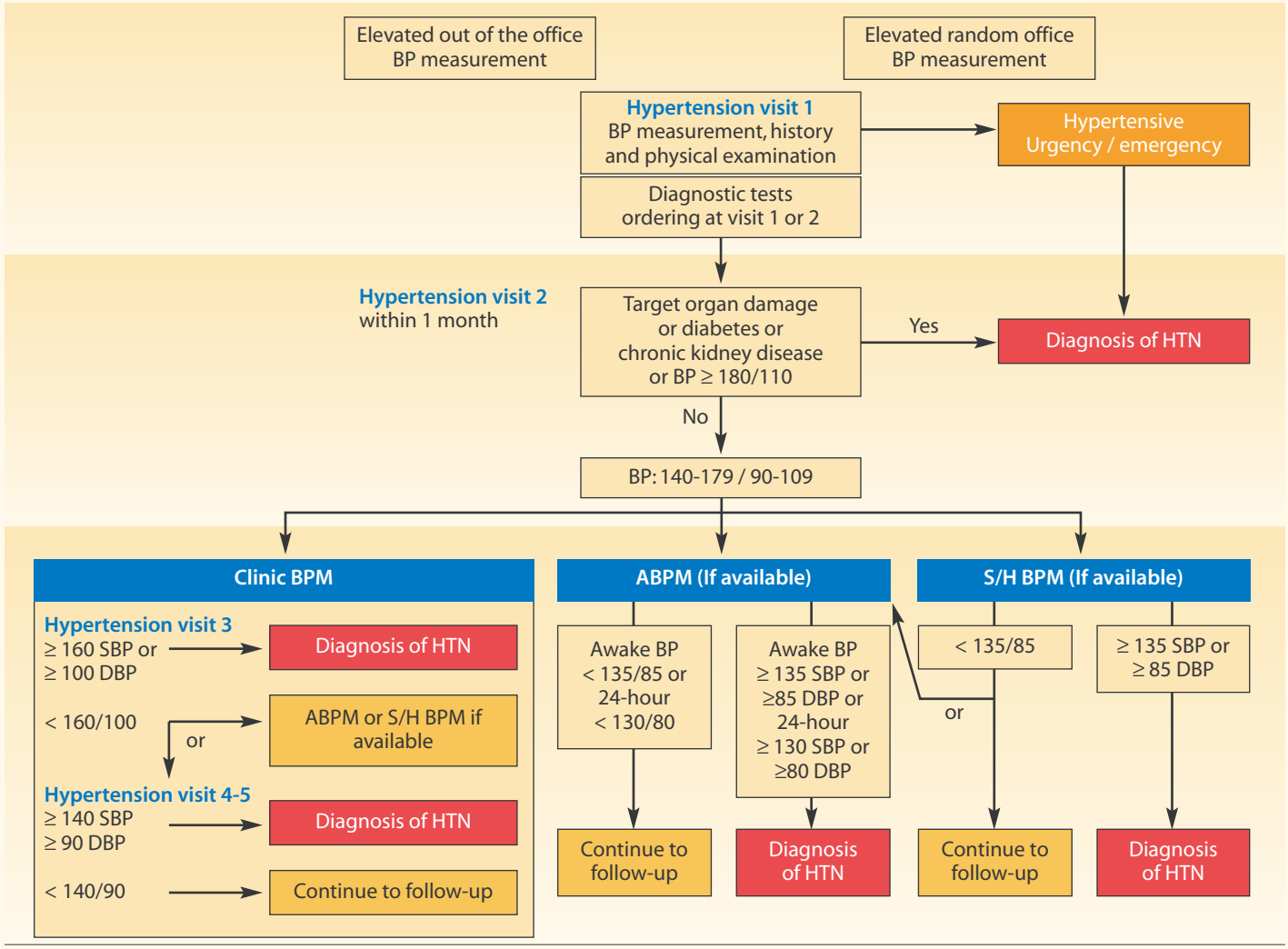
- The diagnosis of hypertension should be expedited (especially in the setting of increased risk). Based on our analysis of recent studies, the time to diagnosis (and treatment) can be significantly shortened (Figure 1).
- Practitioners can utilize any of the three validated technologies to diagnose hypertension. Office, ambulatory and self/home measurements should all be considered first-line technologies with which to diagnose hypertension.
- Reducing hypertension-related complications in the general population of patients with hypertension depends more on the extent of BP lowering achieved than on the choice of any specific first-line drug class. This year, non-dihydropyridine calcium channel blockers (verapamil and diltiazem) have been added to the list of first-line agents.

### Old But Still Important Messages

These new messages need to be incorporated into what remain the "older but still really important" considerations for the management of the patient with hypertension, namely:

- The management plan for patients with hypertension must be based on their global cardiovascular risk. A patient's global cardiovascular risk (and recognition of risk factors beyond hypertension) has important implications in terms of the management of those other risk factors, as well as the

Figure 1
**The Expedited Assessment and Diagnosis of Patients with Hypertension: Focus on Validated Techologies for Blood Pressure Assessment**




HTN = hypertension; ABPM = ambulatory bloob pressure measurement; S/H BPM = self/home blood pressure measurement
SBP = systolic blood pressure; DBP = diastolic blood pressure
BP measured in mmHg

management of the patient's hypertension. The patient's global risk impacts on their target BPs (Table 1) as well as in the choice of specific drug therapies (Table 2).

- Lifestyle modifications are the cornerstone of antihypertensive and anti-atherosclerotic therapy. It is understood that it is difficult to implement lifestyle changes, given the factors in our society that discourage physical activity and healthy eating. Notwithstanding, even brief healthcare professional interventions increase the probability of a patient adhering to some lifestyle changes.
- Combinations of therapies (pharmacologic and lifestyle) are generally necessary to achieve target BPs. Most patients require more than one antihypertensive drug to achieve recommended BP targets (Table 3). This is also true in the

Table 1
**Target Values for Blood Pressure**

| Condition | Target (SBP/DBP mmHg) |
|---|---|
| Diastolic ± systolic hypertension | < 140/90 |
| Isolated systolic hypertension | < 140/90 |
| Diabetes | < 130/80 |
| Renal disease | < 130/80 |
| Proteinuria > 1g/day | < 125/75 |

context of combining pharmacologic and lifestyle modification interventions and in the consideration of "global" strategies for atherosclerotic risk reduction

- Focus on adherence. Failure to achieve this adaptation is probably the most important factor leading to our ongoing challenge to control BP and to reduce the epidemic of hypertension-related morbidity and mortality (Table 4).

Table 2
**Considerations in the Individualization of Antihypertensive Therapy**

| | Initial Therapy | Second-line Therapy | Notes and/or Cautions |
|---|---|---|---|
| Hypertension without other compelling indications | Thiazide diuretics, beta-blockers, ACE inhibitors, ARBs, or long-acting CCBs (consider ASA and statins in selected patients) | Combinations of first-line drugs (Table 3) | Alpha-blockers are not recommended as initial therapy. Beta-blockers are not used as monotherapy over 60 years of age. Hypokalemia should be avoided by using potassium-sparing agents in those who are prescribed diuretics as monotherapy. ACE inhibitors are not recommended in black patients. |
| Isolated systolic hypertension without other compelling indications | Thiazide diuretics, ARBs or long-acting DHP CCBs | Combinations of first-line drugs | Hypokalemia should be avoided by using potassium-sparing agents in patients who are prescribed diuretics. |
| Diabetes mellitus with nephropathy | ACE inhibitors or ARBs | Addition of thiazide diuretics, cardioselective beta-blockers, long-acting CCBs or use an ARB/ACE inhibitor combination | If the serum creatinine level is >150 mmol/L, a loop diuretic should be used as a replacement for low-dose thiazide diuretics if volume control is required. |
| Diabetes mellitus without nephropathy | ACE inhibitors, ARBs or thiazide diuretics | Combinations of first-line drugs or addition of cardioselective beta-blockers and/or long-acting CCBs | |
| Angina | Beta-blockers (strongly consider adding ACE inhibitors) | Long-acting CCBs | Avoid short-acting nifedipine. |
| Prior myocardial infarction | Beta-blockers and ACE inhibitors | Combinations of additional agents | |
| Heart failure | ACE inhibitors, beta-blockers and spironolactone (ARBs if ACE-inhibitor intolerant) | ARBs or hydralazine/isosorbate dinitrate or loop diuretics as additive therapy | Avoid nonDHP CCBs (diltiazem, verapamil). |
| Past cerebrovascular accident or TIA | ACE inhibitor/diuretic combinations | | BP reduction reduces recurrent cerebrovascular events. |
| Renal disease | ACE inhibitors (diuretics as additive therapy) | Combinations of additional agents | Avoid ACE inhibitors if bilateral renal artery stenosis. |
| Left ventricular hypertrophy | ACE inhibitors, ARBs, DHP CCBs, diuretics, (beta-blockers for patients under 55 years of age) | | Avoid hydralazine and minoxidil. |
| Peripheral arterial disease | Does not affect initial treatment recommendations | Does not affect initial treatment recommendations | Avoid beta-blockers with severe disease. |
| Dyslipidemia | Does not affect initial treatment recommendations | Does not affect initial treatment recommendations | |

## 2005 CHEP Recommendations

Continued from page 4

Table 3
**Useful Antihypertensive Drug Combinations**

| Column 1 | Column 2 |
|---|---|
| Thiazide diuretic<br>Long-acting CCB | Beta-blocker*<br>ACE Inihibitor<br>ARB |

*For additive hypotensive effect in dual therapy, combine an agent from Column 1 with any in Column 2.*

** Caution should be exercised in combining a non-DHP-CCB and a beta-blocker*

Table 4
**Recommendations to Improve Adherence to Antihypertensive Prescriptions**

**Adherence can be improved by a multi-pronged approach:**

- adherence to pharmacologic and nonpharmacologic therapy should be assessed at every visit
- simplify medication regimens to once-daily dosing and utilizing electronic medication compliance aids
- tailor pill-taking to fit patients' daily habits
- encourage greater patient responsibility/autonomy in monitoring their BP and adjusting their prescriptions
- coordinate with work-site healthcare givers to improve monitoring of adherence with pharmocologic and lifestyle modification prescriptions
- educate patients and patients' families about their disease/treatment regimens

## History of Canadian Recommendations

Continued from page 1

be treated (including a look at cost effectiveness), to review proper measurement of blood pressure (BP) and diagnosis of hypertension, and to address relative effectiveness and cost effectiveness of treatment with diuretics, beta blockers, sodium restriction and weight loss.

In 1985, Dr. Jacques de Champlain chaired and Dr. Pierre Larochelle coordinated recommendations on hypertension management in the elderly. In 1989, the process was chaired by Dr. Martin Myers. In creating the recommendations, the 1989 process considered the reduction of cardiovascular events, BP lowering, adverse effects and costs. While early recommendations processes created the template for the current recommendation process, the methodology was not published. The processes were largely funded by national and provincial grants.

In 1990, the Canadian Hypertension Society and the Canadian Coalition for High Blood Pressure Prevention and Control (CCHBPPC) co-sponsored the development of nonpharmacologic hypertension management recommendations. The recommendations were developed in a panel consensus approach chaired by Dr. Arun Chockalingam. In 1992, the Canadian Hypertension Society updated the recommendations, subsequently published in 1993 in a process co-chaired by Drs. George Carruthers and Pierre Larochelle. This was the first process where the evidence-based medicine process was clearly articulated. Four panels (diagnosis chaired by Dr. Brian Haynes, pharmacotherapy chaired by Dr. Richard Ogilvie, hypertension in the elderly chaired by Dr. Richard Reeves, and diabetic hypertension chaired by Dr. Pavel Hamet) reviewed the evidence and developed recommendations for adjudication by the entire group. In 1997, Drs. Simon Rabkin and Robert Burrows co-chaired a recommendations process on the management of hypertension in pregnancy, sponsored by the Canadian Hypertension Society and the Society of Obstetrics and Gynecology. The process also followed the CHS evidence-based approach to grading evidence.

In 1999, two separate processes reported. The first was sponsored by the Canadian Hypertension Society and the CCHBPPC, co-chaired by Drs. Ellen Burgess and Norm Campbell and focused on lifestyle modification to prevent and control hypertension. The second process reporting in 1999 was an update of the 1993 management recommendations and was sponsored by a broad multidisciplinary group of national organizations headed by the Canadian Hypertension Society with Dr. Ross Feldman chairing. Both processes followed explicit evidence-based methodology similar to the 1993 process.

Not only was the Canadian Hypertension Society active in developing national hypertension recommendations, but the CCHBPPC also developed hypertension management recommendations to guide practitioners in the use of self-measurement of BP, BP measurement and follow-up and adherence to medication. Further, the Canadian Guide to Periodic Health Examination also produced national hypertension management recommendations.

Between 1985 and 1992, the Canadian Heart Health Surveys were conducted. The survey estimated that 21% of adult Canadians were hypertensive and that only 13% of those with hypertension were treated and controlled. The NHANES survey from the U.S. in 1994 found that 20% of adult Americans had hypertension and 25% were treated and controlled. The recognition of the poor treatment and control rate in Canada compared to that of our southern neighbors was seen as an

*Continued on page 7*

# Salt and Hypertension: The Debate Continues

*by Alain Vanasse and Gérard Plante*

Dr. Alexander Logan recently published some commentaries[1] following the publication in *Hypertension Canada*[2] of our critical analysis of the study by Tuomilehto[3] et al. In order to proceed with a constructive debate about salt and arterial hypertension, we propose to differentiate the information provided, depending on whether it comes from fundamental studies, clinical studies or population studies. These different types of studies are of a distinct epistemological nature and this should be taken into account when analyzing the results. Before proceeding, we must correct certain statements.

Contrary to what Dr. Logan asserts, we proposed this commentary as independent scholars and scientists. We do not have a bone to pick with the food industry. We have never stated that the article by Tuomilehto tipped the balance "in favour of a health policy of universal dietary salt restriction." Instead, we suggested that readers analyse whether this study, together with the results from previous tests, is convincing enough. We also questioned the relevance of adopting measures similar to those in force in Finland, as well as the necessity of legal advisers and Canadian scientists to take a stance on this issue.

With regard to the results of the Tuomilehto study and the content of commentaries published in *Hypertension Canada*, we refer the reader to the original publications. We agree with Dr. Logan that an observational study is not sufficient to infer causality between salt and arterial hypertension. It is important to point out that it is often difficult, if not impossible, for logistical or ethical reasons, to conduct randomized studies where the results would be likely to produce the conclusive data sought. In the absence of such data, we have to turn to population studies and experimental data that come from studies on animals. This being said, these studies support the possible causality between sodium chloride and the pathogenesis of hypertension. These studies also recommend a threshold effect that would be between 50 and 100 mmol of sodium per day. In his response, Dr. Logan suggests that "it is highly unlikely that sodium has a direct toxic effect since it is the mainstay in body fluid regulation and overall health." If we follow this simplified logic, nicotine is also not harmful because of a "natural" regulating neurohormone necessary to transfer the nerve impulse to the synaptic level. Nonetheless, we totally agree that other mechanisms involved in the vascular toxicity of sodium, potassium, calcium and magnesium cations also have to be taken into account. Considering the important role of phosphate and sulphate anions in hypertension found in renal failure, why not also look at the potentially harmful effects on the vascular wall?[4] This type of research could likely fill the knowledge gap about sodium and the precocious remodelling of the vessel wall, perhaps even before blood pressure rises. We have also identified the critical role that proteoglycans play in the interstitial immobilization of sodium,[5] as well as the impact of this phenomenon on the stiffness of main arterial trunks and resistance arteries.[6] At the European Society of Hypertension convention, Professor Hugh de Wardener, renowned pioneer in the field of hypertension and sodium, recently pointed out the direct impact of natremia, within a variation limit range of 3 mmol/L, on the hypothalamus, the thirst centre and sodium intake.[7] This new approach is starting to win over scientists interested in exploring physiopathological issues that have remained vague for too long. Although he seems to consider the issue a nuisance, we acknowledge the interesting scientific contribution of Dr. Logan and his collaborators to understanding sodium/insulin resistance, and insulin/natriuretic peptide interactions.[8,9]

On a clinical level, it is important to distinguish between two types of intervention: preventive intervention and curative intervention. With respect to the curative intervention, the sodium/heart/vessel/hypertension debate has been ongoing for over a century and has contributed to the development of diuretics, the first antihypertensive agents, which are still rightly considered beneficial for the treatment of hypertension.[10] The highest authorities on the subject (ISH, WHO, JNC, Canadian Coalition for Hypertension Control, Hypertension Society of Quebec) rely on the findings of randomized studies to unanimously recommend a reduction in salt intake as one of the nonpharmacologic methods to treat arterial hypertension.

Let us now examine the problem based on prevention logic and public health. Did Dr. Logan not realize, in the meta-analysis published by his group in 1996, that a direct relationship exists between the intake of sodium and arterial hypertension in the elderly?[11] The most recent DASH studies illustrate a significant decrease in the arterial pressure of obese individuals who reduce their dietary salt intake.

With regards to public health, failing to take action can have adverse effects. These adverse effects would be very difficult to justify to the general public with inappropriate academic rhetoric. Even

with the lack of conclusive data, it could prove to be necessary to take action, as in the case of tobacco or drinking and driving. If we acknowledge that hypertensive, obese and elderly patients would benefit from a reduction in their sodium intake as a means to prevent cardiovascular disease, we can concede that a large percentage of the general public would be affected by such measures. The challenge comes in translating these measures into effective actions. Should we adopt a behavioural or an ecological approach? The modern mainstream health promotion and prevention institutes[12-15] recommend a global approach. How can hypertensive, obese or elderly patients reduce their consumption if we do not provide them with the means to estimate their sodium intake? Would explicit labelling of the sodium content on food products sold in Canada not be a factor that would make choosing appropriate food easier?

We must recognize the pivotal role of dietary salt in the development of cardiovascular diseases. Taking legitimate measures to inform the public on the salt content of the food products they consume represents one of the appropriate interventions, based on the best knowledge available, in accordance with the best practices in public health. Far from wanting to attack the agro-food industry, as Dr. Logan alleges, it is a question of exercising our fundamental role as citizens, physicians and scholars. We disclaim any conflict of interest in this debate.

References
1. Logan A. Sodium sensitivity, not level of sodium intake, the culprit in cardiovascular disease. Hypertension Canada 2004; 78:7.
2. Vanasse A, Turgeon G. The true relationship between sodium and cardiovascular disease, Hypertension Canada 2003; 76:3-7
3. Tuomielhto J, Jousilahti P, et al. Urinary sodium excretion and cardiovascular mortality in Finland: a prospective study. Lancet 2001; 357:848-51.
4. Safar ME, London GM, et al. Arterial stiffness and kidney function. Hypertension 2004; 43:163-7.
5. Et-Taouil K, Lacasse S, et al. Glycosaminoglycans determine structural properties of the large arteries. J Hypertension 2004; 22(suppl 2):S57.
6. Et-Taouil K, Schiavi PI, et al. Sodium intake, large artery stiffness, and proteoglycans in the spontaneously hypertensive rat. Hypertension 2001; 38:1172-6.
7. He FJ, Markandu ND, et al. Plasma sodium – Ignored and underestimated. J Hypertension 2004; 22(suppl 2):S11.
8. Feldman RD, Logan AG, et al. Dietary salt restriction increases vascular insulin resistance. Clin Pharmacol Therap 1996; 60:444-51.
9. Miller JA, Abouchacra S, et al. Atrial natriuretic factor counteracts sodium-retaining actions of insulin in normal men. Am J Physiol 1993; 265:R584-90.
10. Lanthier L, Nawar T, et al. Résultats de l'étude ALLHAT : un traitement uniformisé de l'hypertension artérielle. Médecine/Sciences 2003; 19:377-80.
11. Midgley JP, Matthew AG, et al. Effect of reduced dietary sodium on blood pressure: a meta-analysis of randomized controlled trials. JAMA 1996; 275:1590-7.
12. Haddon W, Baker S. Injury control (Chapter 8). Injury control. In: Clark DW, McMahon B (ed.). Preventive and community medicine.. Little Brown, Boston, 1981, pp. 109-140.
13. Walsh JME, McPhee SJ. A systems model of clinical preventive care: An analysis of factors influencing patient and physician. Health Education Quarterly 1992; 19(2):157-175.
14. Bandura A. Self-efficacy: Toward a unifying theory of behavioural change. Psychological Review 1977; 84:191-215.
15. Green LW, Kreuter MW, et al. *Health Education planning: A diagnostic Approach*. Mayfield, Palo Alto, 1980, 306 p.

*Alain Vanasse, MD, PhD,*
*Department of Family Medicine,*
*University of Sherbrooke.*
*Gérard Plante, MD, FRCP,*
*Departments of Medicine (Nephrology),*
*Physiology, Pharmacology,*
*University of Sherbrooke.*

## History of Canadian Recommendations

Continued from page 5

opportunity to reduce cardiovascular morbidity and mortality in Canada during the mid-1990's. Discussions between the CCHBPPC (Dr. Campbell), Health Canada (Dr. Taylor) and the Heart and Stroke Foundation of Canada (Dr. Wilson) centered on why the Canadian Hypertension Guidelines were not improving BP control (relative to the U.S.) and what would be required to improve the process to result in better hypertension treatment and control. The consensus was that, to be successful, the recommendations would have to be evidence-based, up-to-date, associated with credible organizations (Canadian Hypertension Society and College of Family Physicians of Canada) and, most important, associated with an extensive, sustained implementation program. Thus the Canadian Hypertension Education Program (CHEP) program was initiated in 2000, attached to the 1999 management update.

The CHEP process was developed to be more systematic and was structured to reduce bias, to increase transparency and to value rigorous research design (internal and external validity of results) and patient outcomes. In particular, a committee of clinical epidemiologists with expertise in evidence-based medicine was created to review all recommendations, to ensure a consistent application of the systematic approach to the literature review and recommendation development. A librarian was hired to develop and run sensitive and specific literature search strategies. However, the most unique aspect of CHEP was the commitment to an annual update of the recommendations and to a distinct and extensive implementation program. Once it was determined to be sustainable, a third component was added to the CHEP process to assess the impact of the program on hypertension treatment, control and complications.

In the future, a CHEP research network will contribute to optimizing patient management by replacing opinion and lower-quality evidence with knowledge. The CHEP process is a very extensive volunteer effort by most of the clinical members of the hypertension community to reduce the burden of hypertension in Canada. It is hoped that this process will lead to better health outcomes for Canadians and contribute to improved BP control in the Canadian population. Updated CHEP material is on the CHS website: *www.hypertension.ca*.

*Continued on page 8*

# The First Cases of Pheochromocytoma

*by E.C. Abbott*

The title of this article is misleading, because pheochromocytomas must have been discovered by pathologists in patients at autopsy, many years prior to the first reports of successful surgical removal in 1926. Indeed, the first pathologic description was in 1886 by Frankel. The clinical features would have been confusing and the pathophysiology a mystery. Dr. William Young Jr., Head of Endocrinology at the Mayo Clinic, presented the clinical course of their first case at the annual meeting of the Atlantic Endocrine Society in 2002.[1,2] A 1982 paper[3] from the Surgical Department at the Mayo Clinic had pointed out that their first successful case was later than a case reported from Switzerland that same year. Dr. Cesar Roux of Lausanne was the surgeon who reported the first case.

An added interest for Canadians is that the Mayo Clinic patient was a 30-year-old Roman Catholic nun from Chatham, Ontario, who had been referred there because of unexplained complaints. Mother Joachim described episodes of sudden-onset back pain with occipital headaches, gas, nausea and vomiting. She was admitted to St. Mary's Hospital in Rochester, N.Y. on June 3, 1926 and remained there until December 13 of that year—a suitable case for today's Length of Stay Committees in any hospital. Mother Joachim was observed on many occasions to be cold and clammy with pallor, tachycardia and tachypnea during her headaches. She looked anxious, had dilated pupils and blood pressure (BP) as high as 300/160 mmHg. When asymptomatic, her BP was as low as 100/70 mmHg. Numerous medications were tried without success.[2]

Because of persistent lumbar pain and a suspicion there were "toxins evidently intermittently discharged affecting the sympathetic," a laparotomy was performed on October 11, with a view to sympathectomy. In a little over an hour, a round, lemon-sized tumour had been removed from the left adrenal. Postoperatively, her spells disappeared and her systolic BP never exceeded 130 mmHg again. The tumour was thought to be histologically malignant. There are no details of her intra-operative BP responses during removal of this pheochromocytoma, without alpha- or beta-blockade, but it was likely that alarming rises in BP created a nightmare for the anesthesiologist and surgeons.

Mother Joachim did not die from metastatic tumour. She lived for another 18 years. A convent friend, Mother Gertrude, wrote to tell Dr. Charles Mayo in 1944 that "coronary thrombosis took her while she slept." She never had the advantages of biochemical confirmation, the imaging and nuclear scanning techniques we have available. Yet cases of pheochromoctoma are not always suspected clinically today and diagnostic challenges still exist.

References
1. Mayo CH. Paroxysmal hypertension with tumor of retroperitoneal nerve: report of a case. JAMA 1927; 89:1047-50.
2. Young FW Jr. Pheochromocytoma: 1926-1993. Trends in Endo & Met 1993; 4:122-7.
3. van Heerden JA. First Encounters with Pheochromocytoma: The story of Mother Joachim. Am J Surgery 1982; 144:277-9.

*E.C. Abbott, MD, FRCPC,*
*Division of Endocrinology,*
*Department of Medicine,*
*Dalhousie University*

## History of Canadian Recommendations

Continued from page 7

Suggested Reading
1. Kuchel OG, Mahon WA, McKenzie JK, et al. Approach to drug therapy for hypertension. CMAJ 1979; 120:565-70.
2. Larochell, P, Bass MJ, Birkett NJ, et al. Recommendations from the consensus conference on hypertension in the elderly. CMAJ 1986; 135:741-5.
3. Myers M, Carruthers SG, Leenen FH, et al. Recommendations from the Canadian Hypertension Society Consensus Conference on the Pharmacologic Treatment of Hypertension. CMAJ 1989; 140:1141-6.
4. Chockalingam A, Abbott D, Bass M, et al. Recommendations of the Canadian Consensus Conference on Non-pharmacological Approaches to the Management of High Blood Pressure. CMAJ 1989;142:1397-409.
5. Carruthers SG, Larochelle P, Haynes RB, et al. Report of the Canadian Hypertension Society Consensus Conference. CMAJ 1993; 149: 289-93.
6. Helewa ME, Burrows RF, Smith J, et al. Report of the Canadian Hypertension Society Consensus Conference. CMAJ 1997; 157: 715-25.
7. Campbell NRC, Burgess E, Choi BCK, et al. Methods and an overview of the Canadian recommendations. CMAJ 1999; 160: S1-S6.
8. Feldman RD, Campbell N, Larochelle P, et al. 1999 Canadian recommendations for the management of hypertension. CMAJ 1999; 161: S1-S17.
9. Zarnke KB, Campbell NRC, McAlister FA, et al. A novel process for updating recommendations for managing hypertension: Rationale and methods. Can J Cardiol 2000; 16: 1094-102.
10. Campbell NRC, Nagpal S, Drouin D. Implementing hypertension recommendations. Can J Cardiol 2001; 17: 851-3.

*Norm RC Campbell, MD, FRCPC,*
*Faculty of Medicine, University of Calgary.*
*Denis Drouin, MD,*
*CME Office, Université Laval.*
*Ross Feldman, MD, FRCPC,*
*Professor of Medicine, Physiology, Pharmacology and Toxicology,*
*University of Western Ontario.*