CABG in Canada

In 2 recent *CMAJ* articles[1,2] we reported a favourable national trend of decreasing risk-adjusted rates of death after coronary artery bypass graft (CABG) surgery over 4 fiscal years — 1992/93 through 1995/96. The 17% relative decline in adjusted death rates over this period was judged to be comparable to the declines seen over similar periods in various regions of the United States.[3–6] We now briefly report results of more recent analyses that include data from 2 additional fiscal years, 1996/97 and 1997/98.

The updated results (Table 1) were generated using hospital discharge data from the Canadian Institute for Health Information. This data source provides information on CABG surgery cases from all provinces except Quebec. We performed risk adjustment analyses to control for differences in average severity of illness across years using the methods described in our original articles.[1,2]

These data demonstrate a steady increase in the number of CABG procedures performed each year and continuing improvement in both observed and adjusted death rates after CABG surgery, despite a concomitant increase in expected death rates. The latter suggests that, on average, patients selected for CABG surgery are becoming sicker and their cases more complex, and yet they are more likely to experience favourable short-term outcomes after surgery. The 29.5% relative decline in risk-adjusted death rates seen over 6 years is unlikely to be the result of "upcoding" of severity of illness, because the coding of hospital discharge data is not done by the clinicians who care for the patients and because the observed death rates (which are not subject to coding biases) also dropped considerably.

Although the reasons for the improved outcomes are not clear from our analyses, we would nonetheless propose that collective congratulations ought to be extended to the community of cardiologists, intensivists, anesthetists and cardiac surgeons who care for these patients.

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References

1. Ghali WA, Quan H, Brant R. Coronary artery bypass grafting in Canada: national and provincial mortality trends, 1992-1995. *CMAJ* 1998; 159(1):25-31.
2. Ghali WA, Quan H, Brant R. Coronary artery bypass grafting in Canada: hospital mortality rates, 1992–1995. *CMAJ* 1998;159(8):926-30.
3. Peterson ED, Jollis JG, Bebchuk JD, Delong ER, Muhlbaier LH, Mark DB, et al. Changes in mortality after myocardial revascularization in the elderly. *Ann Intern Med* 1994;121:919-27.
4. Hannan EL, Kilburn H Jr, Racz M, Shields E, Chassin MR. Improving the outcomes of coronary artery bypass surgery in New York State. *JAMA* 1994;271:761-6.
5. O'Connor GT, Plume SK, Olmstead EM, Morton JR, Maloney CT, Nugent WC, et al, for the Northern New England Cardiovascular Disease Study Group. A regional intervention to improve the hospital mortality associated with coronary artery bypass graft surgery. *JAMA* 1996;275:841-6.
6. Ghali WA, Ash AS, Hall RE, Moskowitz MA. Statewide quality improvement initiatives and mortality after cardiac surgery. *JAMA* 1997; 277:379-82.

Table 1: Risk-adjusted Canada-wide rates of death for coronary artery bypass grafting (CABG) for fiscal years 1992/93 through 1997/98

Fiscal year	No. of cases	Observed death rate, %	Expected death rate, % *	Observed/ expected ratio	Adjusted death rate, %†
1992/93	11 895	3.60	3.09	1.17	3.97
1993/94	12 094	3.89	3.19	1.22	4.16
1994/95	12 956	3.54	3.37	1.05	3.59
1995/96	13 412	3.36	3.46	0.97	3.31
1996/97	14 786	3.29	3.66	0.90	3.07
1997/98	15 926	2.93	3.57	0.82	2.80

*The expected death rate was calculated by averaging the predicted probabilities of death (from the logistic regression model used for risk adjustment) for CABG cases performed in a given year.
†The risk-adjusted death rate was calculated by multiplying the observed/expected ratio by the death rate for the entire 6-year period, 3.41%.

Does treating hypertension protect against dementia?

I enjoyed reading the guidelines from the Canadian Consensus Conference on Dementia[1] as well as the excellent physician's guide to using the recommendations.[2] Having taken part in the conference, I can attest to the fact that the material published in your pages accurately reflects the deliberations at the meeting.

Despite my overall support for the guidelines, I must disagree with the statement that "the treatment of hypertension reduces the incidence of not only vascular dementia but also Alzheimer's disease" in scenario 4 of the physician's guide.[2] The authors cite an article by Forette and colleagues,[3] who make claims that are far too optimistic given the evidence they present. Forette and colleagues describe the Systolic Hypertension in Europe (Syst–Eur) trial, a secondary goal of which was to reduce the incidence of vascular dementia by the treatment of isolated systolic hypertension. Only 2 cases of vascular dementia occurred, far fewer than had been originally predicted. It was also found that treatment of hypertension apparently reduced the incidence of Alzheimer's disease and mixed and vascular dementias by 50%. Although this finding is interesting, it should be pointed out that the confidence interval around the estimate of a 50% reduction included 0. Thus, the data are also just as compatible with no treatment effect. Forette and colleagues' results constituted a post-hoc analysis, suggesting the possibility of false-positive results.

Of interest as well is the fact that the only comparable study (the Systolic Hypertension in the Elderly Program [SHEP]), while using different drugs than those used in the Syst–Eur trial, found no protection against dementia with control of systolic hypertension.[4] A colleague and I have pointed out our objections to Forette and colleagues' overly optimistic claims in more detail elsewhere.[5]

Further trials are needed to determine whether treatment of hypertension will in fact reduce the incidence of vascular dementia and Alzheimer's disease, especially in the frail elderly suffering from comorbidity. In the meantime, we must continue to approach the decision to treat systolic hypertension in the very elderly with great care, lest we do more harm than good in treating them.

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References

1. Patterson CJS, Gauthier S, Bergman H, Cohen CA, Feightner JW, Feldman H, Hogan DB. Management of dementing disorders: conclusions from the Canadian Consensus Conference on Dementia. *CMAJ* 1999;160(12 Suppl):S1-15.
2. Patterson CJS, Gauthier S, Bergman H, Cohen CA, Feightner JW, Feldman H, Hogan DB. Canadian Consensus Conference on Dementia: a physician's guide to using the recommendations. *CMAJ* 1999;160(12):1738-42.
3. Forette F, Seux ML, Staessen JA, Thijs L, Birkenhäger WH, Babarskiene MR, et al. Prevention of dementia in randomised double-blind placebo-controlled Systolic Hypertension in Europe (Syst–Eur) trial [see comment]. *Lancet* 1998;352:1347-51. Comment in: *ACP J Club* 1999;130:57.
4. SHEP Cooperative Research Group. Prevention of stroke by antihypertensive drug and treatment in older persons with isolated systolic hypertension. Final results of the Systolic Hypertension in the Elderly Progam (SHEP). *JAMA* 991;265:3255-64.
5. Clarfield AM, Paltiel O. Prevention of dementia in randomised double-blind placebo-controlled Systolic Hypertension in Europe (Syst–Eur) trial [comment]. *ACP J Club* 1999;130:57. Comment on: *Lancet* 1998;332:1347-51.

[Two of the authors respond:]

We acknowledge that our interpretation of the Syst–Eur trial was overly optimistic. Many criticisms have been levelled at this trial as reported by Forette and colleagues.[1–3] In the intention-to-treat analysis the incidence of all dementia was apparently reduced by 50%. Although this reduction, from 7.7 to 3.8 cases per 1000 patient-years, was statistically significant ($p = 0.05$), the 95% confidence interval (CI) was 0%–76%. In the per-protocol analysis, reduction was 60% ($p = 0.03$) and the 95% CI was 2%–83%.[4] It has been pointed out that there appeared to be a large number of subjects not completing the protocol and that assignment of as few as 2 of these subjects could change the conclusions.[2] However, Forette and colleagues reported that when subjects who could not be traced before publication were subsequently included in the analysis, the original conclusions were validated.[5]

The intention of Forette and colleagues' study was to determine whether antihypertensive treatment would reduce the incidence of vascular dementia.[4] The finding of only 2 incident cases of vascular dementia among 32 cases of all dementia is not surprising. Although vascular dementia is considered to be much less common now than in the past, its distinction from Alzheimer's disease is less clear than once thought. Not only do they share common risk factors,[6] but there is mounting evidence that the occurrence of strokes in people with Alzheimer's neuropathological changes may precipitate or exaggerate the clinical manifestations of dementia.[7]

These observations provide a plausible rationale for the findings of Forette and colleagues. However, the SHEP study failed to demonstrate that antihypertensive treatment reduced the incidence of dementia.[8] These apparently conflicting results might be explained by differences in the 2 study populations; for example, the mean pretreatment diastolic blood pressure was 10 mm Hg lower in the SHEP study than in the Syst–Eur trial. It has been suggested that the use of a calcium-channel blocker in the Syst–Eur study could explain the apparent difference in findings.[4,9] However, data from the Canadian Study of Health & Aging indicate that people with hypertension who were taking calcium-channel blockers were more likely to experience a decline in cognitive performance than those taking other antihypertensive medications.[10]

We thank Mark Clarfield for his observations and hope that this debate will be settled by a definitive trial using all causes of dementia as an end point. Indeed, such a study is already underway, the Study on Cognition & Prognosis in the Elderly (SCOPE).[11] In the meantime we hope that systolic hypertension will be appropriately recognized and treated in older people, as this will reduce the incidence of stroke. Whether such treatment will also decrease the incidence of dementia remains to be seen.

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References

1. Clarfield AM, Paltiel O. Prevention of dementia in randomised double-blind placebo-controlled Systolic Hypertension in Europe (Syst–Eur) trial [comment]. *ACP J Club* 1999;130:57. Comment on: *Lancet* 1998;332:1347-51.
2. Pahor M, Somes GW, Franse LV, Shorr RI, Applegate WB. Prevention of dementia: Syst–Eur trial [letter]. *Lancet* 1999;353:235.
3. Zuccalà G, Pedone C, Cocchi A, Bernabei R, Carbonin P. Prevention of dementia: Syst–Eur trial [letter]. *Lancet* 1999;353:235-6.
4. Forette F, Seux ML, Staessen JA, Thijs L, Birkenhäger WH, Babarskiene MR, et al. Prevention of dementia in randomised double-blind placebo-controlled Systolic Hypertension in Europe (Syst–Eur) trial [see comment]. *Lancet* 1998;352:1347-51. Comment in: *ACP J Club* 1999;130:57.
5. Forette F, Staessen JA, Thijs L, Birkenhager WH. Author's reply [letter]. *Lancet* 1999; 353:236-7.
6. Skoog I. The relationship between blood pressure and dementia: a review. *Biomed Pharmacother* 1997;51:367-75.
7. Snowdon DA, Greiner LH, Mortimer JA, Riley KP, Greiner PA, Markesbury WR. Brain infarction and the clinical expression of Alzheimer's disease: the NUN study. *JAMA* 1997;277:813-7.
8. SHEP Cooperative Research Group. Prevention of stroke by antihypertensive drug and treatment in older persons with isolated systolic hypertension. Final results of the Systolic Hypertension in the Elderly Program (SHEP). *JAMA* 1991; 265:3255-64.
9. Kazuomi K, Pickering TG. Calcium antagonist and prevention of dementia in elderly people [letter]. *Lancet* 1999;353:1184.
10. Maxwell CJ, Hogan DB, Ebly EM. Calcium-channel blockers and cognitive function in elderly people: results from the Canadian Study of Health and Aging. *CMAJ* 1999;161:501-6.
11. Hansson LH, Skoog I, Banki CM, Baro F, Bretcler M, et al (for the SCOPE Investigators). Study on Cognition and Prognosis in the Elderly (SCOPE). *Blood Press*. In press.