cally caused deficiencies of vitamins B6 and B12 and folic acid, the condition we associate with McCully, from his extensive studies on children with homocystinuria.[2,3] Multiple strokes because of premature, extensive vascular disease resembling arteriosclerosis were noted throughout the body tissues of those children. In addition, the early, progressive cognitive decline seen in the patient described by Simon and associates[1] closely resembles the cognitive decline we are seeing as a previously unrecognized side effect of statin therapy.[4,5] Was this patient receiving any statin drugs?

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[Two of the authors respond:]

Cerebral autosomal-dominant arteriopathy with subacute infarcts and leukoencephalopathy is a phenotypically heterogenous disease. Although the parents of the patient we described[1] were asymptomatic by history, one of them could well have harboured the same R182C mutation in the Notch3 gene as did the proband. We suggested a new mutation as a possibility only.

We did not test the patient for fasting serum homocysteine levels because the result would not have changed management. To date, it has not been shown that a reduction in homocysteine levels reduces clinical events in terms of stroke or any other vascular disease.[2] Nevertheless, we routinely counsel all stroke patients to eat sufficient fruits and green leafy vegetables to ensure that they receive enough B vitamins including pyridoxine, folic acid and cobalamin.

The patient was not initially treated with a statin agent. Since diagnosis, we have observed elevation of his total cholesterol and have begun treatment with a statin. In our opinion, it is more likely that this therapy will result in slowing, rather than acceleration, of the dementing process.[3]

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Competing interests: None declared.

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Genetics and insurance

I was pleased to read the commentary by Bartha Knoppers and Yann Joly[1] and the accompanying article by the Canadian Genetics and Life Insurance Task Force.[2] I agree that there is an urgent need to systematically address the issues of genetic risk and insurability in the Canadian context. Although 90% of people are insured at standard premiums,[1] there is a growing perception among many of those who undergo predictive (presymptomatic) testing that their test results may have negative consequences.

Consider the following recent clinical scenarios: a health care provider who works full-time for an insurance company requests anonymous testing for *BRCA1* (a gene associated with higher risk of breast and ovarian cancer); a man at risk for a neurodegenerative disease registers in a genetics clinic under an assumed name and pays out-of-pocket for genetic counselling and DNA testing; a specialist uses a code word indicating a positive result on a genetic test when referring a patient for prophylactic surgery; women with positive test results for *BRCA1* or *BRCA2* request that no copies of test results or medical recommendations be sent to their physicians; other women at risk for *BRCA1* or *BRCA2* either decline to undergo testing or refuse to disclose the results of testing to relatives (including children).

Fear of insurance discrimination is adversely affecting medical care in this country because patients in such situations perceive that the results of genetic testing could compromise their ability to obtain insurance. Whether the risk is real or imagined, the *perception* of risk is preventing these patients from accessing appropriate care. I wholeheartedly support a moratorium on the use of genetic information by insurance companies and the formation of an independent body to study the issue and make recommendations.

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Competing interests: None declared.

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Correction

In an article about cervical cancer mortality in Canada by Edward Ng and associates,[1] the 3-year moving mortality averages were based on data obtained from the International Agency for Research on Cancer. The agency name was given incorrectly in the Methods section and in the cited reference (reference 9 in the original article). The correct information appears in the second reference[2] in this correction.

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