private and public drug plans. That's good public policy.

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A recent *CMAJ* editorial[1] expresses concern that the reclassification of levonorgestrel 0.75 mg (Plan B) as a "behind the counter" product represents a "needless barrier to access."

The National Association of Pharmacy Regulatory Authorities strongly believes that incorporating pharmacists' counselling in the provision of emergency contraceptives benefits women and the health care system. Pharmacists can play a key role in educating women on the risk of infection associated with unprotected sex, the correct use of barrier and hormonal contraception and the management of side effects of this medication. Women will have the option of visiting a physician or a pharmacist and thus will be able to make their own decision on the initial point of care.

Given the experience in British Columbia[2] of a "dramatic rise" in the total use of emergency contraceptives "resulting mainly from pharmacy dispensing" (to quote the *CMAJ* editorial), it is difficult to understand how consultation with the pharmacist presents a barrier to access. Licensed pharmacists possess the knowledge, skills and professionalism needed to sensitively supply emergency contraception.

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Physicians and advocacy

It is evident that providing responsible advocacy for patients, individually and collectively, is an obligation for Canadian physicians, as was discussed in a *CMAJ* editorial[1] earlier this year. The College of Family Physicians of Canada has distributed a Declaration of Commitment, dated Nov. 25, 2004, that states "we are a resource to our practice populations — promoting health to prevent illness, providing and explaining health information, collaborating with and facilitating access to other caregivers, and advocating for patients throughout the health care system." The Educating Future Physicians for Ontario project identified "advocate" as one of the roles patients expect from their physician. Similarly, the Royal College of Physicians and Surgeons of Canada's CanMEDs roles include the role of "advocate."

Even if such advocacy makes administrators uncomfortable, physicians must judge what is in the best interests of their patients and behave accordingly. We must strive for communication within institutions that ensures that medical staff can make good judgments about how best to exercise their responsibility for advocacy, but we must never stifle their voices. There are too many historical examples of suppression of information when patients would have benefited from prompt disclosure.

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A novel mutation in a patient with pantothenate kinase–associated neurodegeneration

Pantothenate kinase–associated neurodegeneration is an autosomal recessive disorder characterized by accumulation of iron mainly in the basal ganglia.[1,2] In about half of these cases, patients have an identifiable mutation in the *PANK2* gene.[1]

We previously described a 13-year-old boy who showed the "eye of the tiger" sign on a T_2-weighted magnetic resonance (MR) image[3] that is highly specific not only for this disease but also for a mutation in the *PANK2* gene.[1] Here we report on our screening for mutations of the *PANK2* gene conducted on the genomic DNA of the patient and his family (Fig. 1).

DNA was isolated from peripheral blood using a phenol-chloroform reference protocol. All exons of the gene were amplified by polymerase chain reaction (PCR),[4] and the amplified prod-

ucts were subjected to cycle sequencing using an ABI PRISM 377 (Applied Biosystems) DNA sequencer. A novel mutation was identified in exon 2 resulting in a change of histidine to tyrosine at amino acid position 173 (H173Y), which also creates an *Rsa*I restriction site. To better visualize the restriction products, exon 2 of the gene was reamplified using custom-designed primers (5′-ACCTGACCTCCAATGTGG-3′) and (5′-AGTGTGGAGACTCGAGAAG-3′). Amplified products (176 base pairs) were subjected to restriction endonuclease analysis by using *Rsa*I; 0.5 units of enzyme were used for 10 mL of PCR reaction products. After a 3-hour incubation at 37°C, electrophoresis was carried out on a 2% agarose gel. Fig. 2 shows the restriction analysis for all the family members. The patient is homozygous for this mutation, whereas both the siblings and subjects 1, 2, 5 and 6 are heterozygous.

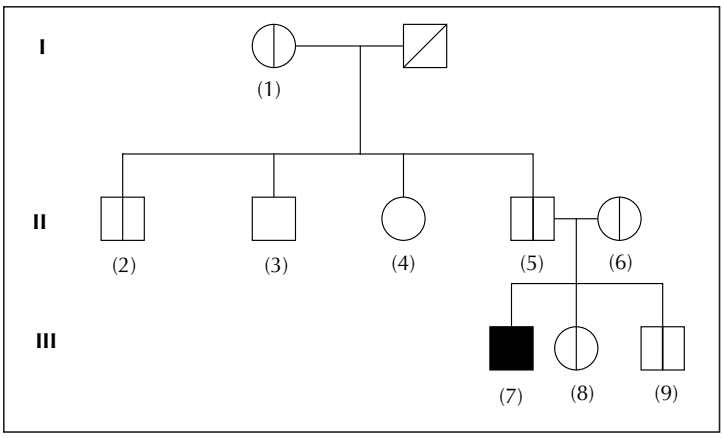

Fig. 1: Pedigree of our patient with pantothenate kinase–associated neurodegeneration. Circles represent females and squares, males. The square with an oblique line through it represents a deceased grandparent. The patient (homozygous for the mutation H173Y in the *PANK2* gene) is represented by a black box. Heterozygotes for the mutation are shown with half-cut boxes. Homozygotes for the wild-type genotype are shown with blank boxes. Numerical codes for all subjects are given in parentheses.

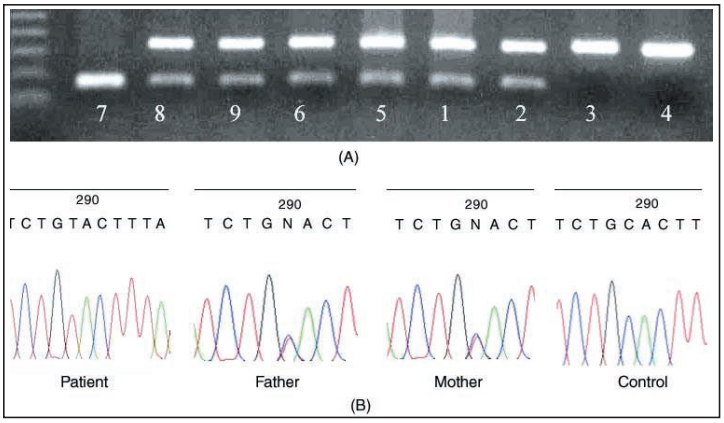

Fig. 2: The mutation H173Y (C to T) creates an *Rsa*I restriction site. (A) A 2% agarose gel shows restriction analysis by the *Rsa*I enzyme. The amplified product (176 base pairs) of exon 2 generated by custom-designed primers was digested by the enzyme at 37°C for 3 hours. The numbers below each lane represent numerical codes of subjects in Fig. 1. Subjects 3 and 4 carry the wild-type genotype and, thus, their amplified products remained undigested, showing only 1 band in the top panel. Both the siblings (8 and 9) and subjects 1, 2, 5 and 6 are heterozygous for this mutation, with 1 allele carrying the mutation and the other carrying the wild-type gene; thus, the top band shows the wild-type allele and the bottom one shows the mutant allele. Patient (7) is homozygous for the mutation, with both the alleles having the mutation, thus, displaying only 1 lower band. (B) Electrophoretogram of patient, father, mother and control. A transition from "C" in control to "T" in the patient can be seen, whereas the 2 parents are heterozygous.

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DOI:10.1503/cmaj.1050096

Correction

A correction[1] that appeared in a recent issue of *CMAJ* should have also referred to the print version of the relevant article.[2]

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