will be referred to the MCC Executive Board at its meeting in October 2003.

We expect that this example and other "disconnects" in licensure and immigration policies of the "federation of partners" will be studied, so that when the anticipated recommendations of the task force are made public, they can be acted upon by the MCC and other bodies in a coordinated and timely manner.

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Reference
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A university's name

In contrast to the information in Table 1 of Patrick Sullivan's article about medical students' debt on graduation,[1] the correct name for our university is Memorial University of Newfoundland.

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Reference
1. Sullivan P. Mortgage-sized debt the new normal for medical students. *CMAJ* 2003;169(5):457-8.

SARS in health care workers

I wondered if Monica Avendano and associates[1] were planning a follow-up report on the 14 health care workers who were treated for severe acute respiratory syndrome (SARS) at the West Park Healthcare Centre. At the time of publication of that report, all of the patients had recovered sufficiently to go home, but only one had returned to work.

I am interested and concerned as to how these patients have progressed in the past few months.

Gordon Farrow
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Reference
1. Avendano M, Derkach P, Swan S. Clinical course and management of SARS in health care workers in Toronto: a case series. *CMAJ* 2003; 168(13):1649-60.

[The authors respond:]

We have continued to follow the patients described in our article[1] after their discharge from the SARS unit. They have undergone chest radiography, pulmonary function testing, chest CT, sleep studies and graded exercise tests. By the eighth week after discharge, the results of chest radiography were normal for all patients. However, CT of the chest showed abnormalities in some patients for up to 6 months after discharge. Convalescent serum antibody tests have been performed for all patients, but the results are not yet available.

Most of the patients have returned to work, the initial group going back 2 months after the onset of acute illness. Fatigue, dyspnea on exertion and insomnia are the most common persisting symptoms. Most of the patients have demonstrated symptoms indicative of the psychological impact of SARS. We are planning a follow-up review for next spring, 1 year after the onset of illness.

Monica Avendano
Peter Derkach
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Reference
1. Avendano M, Derkach P, Swan S. Clinical course and management of SARS in health care workers in Toronto: a case series. *CMAJ* 2003; 168(13):1649-60.

Ziprasidone — not an option for serotonin syndrome

A recent article concerning serotonin syndrome[1] contained an inaccuracy that might result in clinicians attempting a misguided, if not fatal, treatment option. While correctly noting the presumed role of 5-HT_{1A} receptor activation in the pathophysiology of the syndrome, the authors twice surmise that ziprasidone, an atypical antipsychotic, might warrant study as a therapeutic option because of its potent blockade of 5-HT_{1A} receptors.

The reference that the authors use as the pharmacologic basis for this assertion does acknowledge the potent binding of ziprasidone at the 5-HT_{1A} receptor;[2] however, the high affinity of the drug for this receptor is as an agonist, not as an antagonist.[3,4] Other effects of ziprasidone on the serotonergic system include potent antagonism of 5-HT_{1D}, 5-HT_{2A} and 5-HT_{2C} receptors, as well as moderate inhibition of serotonin reuptake.[3,4]

The net result of ziprasidone on serotonergic neurotransmission makes it an inappropriate candidate for treating serotonin syndrome. Aside from the overt problem of directly stimulating 5-HT_{1A} receptors, there is also the more subtle, yet still concerning, matter of indirectly stimulating these same receptors via antagonism of 5-HT_{2A} receptors and inhibition of serotonin reuptake. In fact, there have been reported cases of serotonin syndrome precipitated by the use of other atypical antipsychotics, which are also 5-HT_{2A} receptor antagonists, in combination with serotonergic drugs.[5]

Thus, the use of ziprasidone for treatment of serotonin syndrome seems ill-advised and could prolong or worsen the patient's symptoms. In cases in which the clinician seeks treatment with serotonin antagonists, purported options include methysergide, cyproheptadine and propranolol.[6]

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References
1. Birmes P, Coppin D, Schmitt L, Lauque D. Serotonin syndrome: a brief review. *CMAJ* 2003;168(11):1439-42.
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3. Green B. Focus on ziprasidone. *Curr Med Res Opin* 2001;17:146-50.
4. Carnahan RM, Lund BC, Perry PJ. Ziprasidone, a new atypical antipsychotic drug. *Pharmacotherapy* 2001;21:717-30.
5. Duggal HS, Fetchko J. Serotonin syndrome and atypical antipsychotics. *Am J Psychiatry* 2002;159:672-73.
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Competing interests: None declared.

Anaphylaxis treatment: the details

Having read the review article by Anne Ellis and James Day,[1] I have several questions about drug therapy for anaphylaxis.

Ellis and Day[1] report that patients seen in their unit are usually discharged with a 4-day prescription for prednisone and diphenhydramine, a relatively common approach. However, given that many patients must drive or go to work, I wonder why the authors do not advocate one of the newer nonsedative antihistamines. Similarly, would it be appropriate to recommend the addition of ranitidine for 48 hours, on the basis of the experimental evidence presented by Ellis and Day[1] and given the risk of a biphasic reaction? Since the second-phase reaction may be more severe than the primary reaction,[1] this approach might be safer, although it is as yet unproved. I also wondered what dosage of prednisone is recommended for postdischarge therapy and whether the dose should be tapered.

Ellis and Day[1] mention the cross-reactivity between cephalosporin and penicillin, but there have been conflicting recommendations as to whether this applies to the third-generation cephalosporins. Kelkar and Li[2] recommended against prescribing third-generation cephalosporins to patients allergic to penicillin, but their review was based on extrapolation and inference. Anne and Reisman[3] concluded that it is safe to administer cephalosporin antibiotics, especially third-generation drugs, to penicillin-allergic patients. Pumphrey and Davis[4] reported 6 anaphylactic deaths after a first cephalosporin dose, which occurred over a 5-year period in the United Kingdom. Three of these patients had a penicillin allergy, but the generation of the cephalosporins in these cases was not indicated. In my own experience, many physicians in France are not reluctant to use third-generation cephalosporins, when indicated, for penicillin-allergic patients (in the hospital environment).

Finally, prescribing epinephrine as volumes of a 1:1000 solution is a potentially dangerous dosing system. Administering epinephrine measured in micrograms (or milligrams), as pumped from clearly labelled ampoules, might avoid inadvertent ventricular tachycardia.

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Competing interests: None declared.

In their excellent review of the diagnosis and management of anaphylaxis, Anne Ellis and James Day[1] mention that anaphylactic patients who use β-blockers should be given glucagon. I was not aware of this use of glucagon.

In my own experience as a family physician, the most significant case of anaphylaxis that I remember involved a patient who had not previously been seen in our clinic and whose medical history was unknown to us. He walked into the clinic, bypassed the receptionist and entered an examination room, where he lost consciousness. Resuscitation required multiple intravenous doses of epinephrine. The patient's condition was eventually stabilized in hospital with administration of corticosteroids.

We later learned that this patient, who was taking β-blockers and who had not previously been aware of any allergies, had been stung by an insect while walking along a street leading toward the clinic. Fortunately, he was able to reach the clinic before losing consciousness.

Although this incident happened 20 years ago, it remains applicable, reminding us that patients with anaphylaxis often do not present to their own physician, and a history of β-blocker therapy may not be evident. In this situation, would Ellis and Day recommend a combination of epinephrine and glucagon?

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Reference
1. Ellis AK, Day JH. Diagnosis and management of anaphylaxis. *CMAJ* 2003;169(4):307-12.

Competing interests: None declared.

[The authors respond:]

Axel Ellrodt raises several questions regarding discharge therapy after anaphylaxis. The first relates to alternatives to diphenhydramine prophylaxis. Diphenhydramine has been established as an effective agent in the treatment and prevention of anaphylactic and anaphylactoid reactions, where its sedative properties are an advantage.[1] Given orally at doses of 25 to 50 mg every 4 to 6 hours, it remains the antihistamine of choice to prevent and manage these episodes. A second-generation antihistamine could be substituted if sedation were a concern. However, because biphasic reactivity may be delayed for up to 24 hours, the patient should be advised to minimize activity (including driving) during this interval, and sedative effects may therefore be unimportant. After this interval, treatment with