Ceux-ci ne devraient pas réagir à de telles situations par la censure, mais plutôt fonder leurs décisions sur des considérations plus globales témoignant d’une vision à long terme, les amenant ainsi à générer d’authentiques débats intellectuels, professionnels et sociaux autour de questions et d’enjeux importants pour la santé.

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Accountability in CMAJ

One of the questions arising from the recent firing of CMAJ’s editor and senior deputy editor is “To whom should the Journal and its editorial board be accountable?” If the CMAJ is directly accountable to its publisher, CMA Media Inc., then it effectively does not have editorial independence, despite claims to the contrary. However, I believe that CMAJ should not be directly accountable to any one organization, even its owner. The CMAJ is accountable to CMA Media to prepare a journal for publication every 2 weeks — that is all. In fact, it is CMA Media and the Canadian Medical Association that are responsible to Canadian physicians, the Canadian public, the world medical community and medical scholarship in general to ensure that we have an independent medical journal.

The CMAJ should first and foremost be accountable to its principles, and these principles must be clearly stated. In addition to articles on research and clinical medicine, the journal should be committed to publishing news and commentary about trends in Canadian and world medical practices and policies, as well as environmental issues and international health. The intersection of arts, medicine and life is also a relevant topic.

CMAJ should also be accountable to its readers, especially CMA members (after all, it is “our” journal), to the Canadian public, and to the community of world medical scholarship and publications.

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Chronic fatigue

Jacques Cornuz and colleagues,1 in Box 2 of their case report, list 11 initial laboratory tests for patients with prolonged or chronic fatigue. Given that hypogonadism is one of the most frequently reported abnormalities of patients with chronic fatigue syndrome,2-5 it is surprising that none of the available tests for assessing cortisol production2-5 was included. The importance of this assessment is especially evident in light of the virtually complete recovery of patients with chronic fatigue who are treated with low-dose hydrocortisone.6

Another rationale for assessing cortisol production in patients with chronic fatigue is the fact that this condition shares 43 clinical features with Addison’s disease,7,8 including hypocortisolism, chronic fatigue, and all of the symptoms listed in the diagnostic criteria for chronic fatigue.7 This impressive clinical overlap between 2 distinctly named diseases suggests that in practical terms, chronic fatigue should be regarded as a mild form of Addison’s disease.7

Although Cornuz and colleagues, in Table 1 of their paper, correctly mention Addison’s disease as one of the major underlying causes of fatigue, they should have remarked that “pigmentation in skin creases, scars and buccal mucosa”9 is far from being a constant feature of Addison’s disease.10 Therefore, the absence of such pigmentation in patients with chronic fatigue should not mislead general practitioners to exclude hypocortisolism as a possible cause of that unremitting symptom.

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Recent reports indicate that in addition to viral infections,1 chronic bacterial infections may also lead to chronic fatigue syndrome. For example, chronic fatigue syndrome has been di-
agnosed in patients with Lyme disease, which is caused by intracellular *Borrelia* infections. In addition, patients with chronic fatigue may have intracellular *Chlamydia* spp. or mycoplasmal infections. Brucellosis and tuberculosis are also important causes of chronic fatigue.

Patients with symptoms of fatigue should be screened for these bacterial infections.

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**[The authors respond:]**

We agree with Riccardo Baschetti that hypocortisolism is one of the reported laboratory abnormalities of patients with chronic fatigue syndrome and that hypoactivity of the hypothalamic–pituitary–adrenal axis is a potential pathophysiologic mechanism. However, as recently stressed by Cho and colleagues, the question of whether such hypoactivity is a cause or a consequence of chronic fatigue syndrome remains unanswered. Our review was intended to cover the diagnostic approach to chronic fatigue, rather than treatment, but we can mention here that the results of trials evaluating hydrocortisone therapy have been inconsistent. The first trial assessing this pharmaceutical approach showed only a modest benefit at the expense of adrenal suppression. To our knowledge, the promising results of a second randomized trial assessing lower dose have never been replicated.

As noted by Prasanta Padhan, the workup for chronic fatigue should be adapted to the local context according to the prevalence of diseases causing fatigue, including bacterial infections. Fatigue has been described in both early and chronic Lyme borreliosis. In one prospective study, fatigue was present in more than half of patients with early Lyme disease, and fatigue was more frequent than arthralgia, myalgia or headache. In addition, Lyme disease may lead to a post-Lyme borreliosis syndrome including fatigue. However, the appropriateness of serological testing depends on the probability of disease. Although such testing would be appropriate for a patient with fatigue and a history of erythema migrans (i.e., high pretest probability), it could be inappropriate for a patient suffering fatigue with no objective signs (i.e., low pretest probability) because of the high risk of false-positive results. The pretest probability should also be considered for brucellosis and tuberculosis testing. *Chlamydia* and *Mycoplasma* have been found in patients with chronic fatigue and might be associated with the severity of symptoms.

David Barton is correct that most test results for patients with fatigue will be normal, and we agree that physicians must avoid performing diagnostic procedures simply to address their anxiety about a lack of diagnosis. Because our teaching case report represented an amalgamation of cases, there is no follow-up information. However, in a trial involving women with fatigue (on which one of us [B.F.] was a coauthor) celiac disease was diagnosed by serologic testing and then small-intestine biopsy in 2 patients. One of these patients was lost during follow-up but the other underwent a gluten-free diet and was followed clinically after diagnosis for 12 months. After a few months, she felt better and her fatigue subsided. She underwent a second biopsy 12 months after the diagnosis, which showed no histologic abnormalities.

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