positions (because of the formula for provincial allocation of funds for postgraduate training positions in family medicine and specialties). Emergency medicine is already short of training slots, and such a loss of positions would be disastrous. The solution to the issues raised by MacDonald is to improve the existing educational tracks.

The Commentary format of my article[2] precluded discussion of the topics that Alan Drummond has raised. Indeed, the quality of emergency care in Canada is negatively affected by all of the factors he describes. I would welcome a comprehensive strategy that would alleviate these problems. I also maintain that the quality of emergency medicine training is a crucial issue. The credibility of the specialty is based on our ability to advocate for patients and on our capacity to develop high-quality clinicians, educators, researchers and administrators.

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Clarifying my letter

During the editing process, a shift occurred in content of my letter to the editor[1] that I wish to correct. The statement that we need to stop "pharmaceutical companies from controlling information about treatments" suggests the onus is on industry to present balanced education. My submitted title, which was changed during editing to "Drug marketing priorities," was "Where is the marketing for effective and cost effective psychotherapies?"

The emphasis of my letter is that we, as administrators, educators and clinicians are responsible for providing and learning about a balanced psychological, social and biological approach to patient care. Given strong evidence for brief psychotherapies in a broad range of conditions, physicians should be afforded equal opportunity to learn about these treatments side by side with pharmacotherapy options. Moreover, patients should be aware of and have access to these cost-effective and safe therapies where they choose. The onus is on us who provide programs, edit journals or coordinate medical faculties to be certain we are facilitating this balance in medical education and practice.

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The heart of the matter

Sandeep Arora and associates[1] recommend extracardiac biopsy and other diagnostic modalities instead of heart biopsy for diagnosis of cardiac amyloidosis. However, endomyocardial biopsy remains an excellent method of demonstrating this problem, and false-negative results are uncommon in patients with heart failure.[2]

Immunohistochemical typing of the amyloid may be prognostic. Primary (amyloid light-chain [AL]) amyloidosis with resultant heart failure is associated with a poor prognosis, and up to 40% of such patients die of heart disease.[3] In contrast, senile amyloidosis, which is common, is often uncomplicated, and treatment with cytotoxic agents may not be required.[4,5]

Algorithms have been proposed to diagnose amyloidosis.[5,6] Depending on the amyloid type, the results of extracardiac staining may not accurately indicate the presence of cardiac amyloid.[3] Furthermore, in a study of patients with AL-type amyloidosis who had positive results on endomyocardial biopsy, the extracardiac biopsy results were not always positive.[3]

In patients with severe heart failure, biopsy-proven extracardiac amyloid site, characteristic electrocardiographic findings and characteristic echocardiographic changes, most clinicians feel confident in attributing cardiac dysfunction to amyloidosis.[3] However, doing so may underestimate or overestimate cardiac involvement, depending on the patient population. Endomyocardial biopsy may be the only way to diagnose amyloidosis if it is confined to the heart. Immunotyping of the biopsy specimen may add prognostic information. Heart biopsy is also useful in distinguishing restrictive myocardial abnormalities from constrictive pericardial disease.

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Nouveau mécanisme de présentation des lettres

Le site amélioré des cyberlettres du *JAMC* est désormais le portail de réception de tous les textes destinés à la chronique Lettres. Pour rédiger une cyberlettre, consultez un article sur le site www.jamc.ca et cliquez ensuite sur le lien «Lettres électroniques : répondre à cet article», dans la boîte en haut à droite de l'article. Toutes les cyberlettres seront étudiées pour une éventuelle publication dans le journal imprimé.

Les lettres répondant à un article publié dans le *JAMC* sont plus susceptibles d'être acceptées pour publication imprimée si elles sont présentées dans les deux mois de la date de publication de l'article. Les lettres acceptées pour publication imprimée sont révisées en fonction du style du *JAMC* et raccourcies au besoin (elles doivent habituellement compter au maximum 250 mots).

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[The authors respond:]

We agree that endomyocardial biopsy remains the ideal test to confirm cardiac amyloidosis, especially in cases of isolated cardiac amyloidosis. However, in cases of systemic amyloidosis with suspected cardiac involvement, the need for endomyocardial biopsy can be obviated by the combined findings of low-voltage QRS complexes on electrocardiography, a typical restrictive pattern on transmitral Doppler blood flow imaging and the classic "granular sparkling" appearance of the myocardium with 2-dimensional echocardiography.[1] These findings, together with the results of biopsy of subcutaneous fat, the rectum or gums, or the bone marrow (all of which are safer and less difficult and require less expertise than endomyocardial biopsy), allow cardiac amyloidosis to be diagnosed with great certainty. Radionuclide scintigraphic scanning with iodine 123-labelled serum amyloid P, which localizes signals to organs with amyloid deposits, is another noninvasive test that can be used in certain patients.

Endomyocardial biopsy is limited in its ability to identify any cardiac abnormality that is not diffuse, insofar as only a few biopsy samples are typically obtained and evaluated. The question is whether this method is warranted when an accurate diagnosis can be made on the basis of results of noninvasive testing. We suggest that endomyocardial biopsy be used as a confirmatory test rather than as a screening method.

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1. Arora S, Arora A, Makkar RPS, Monga A. Stiff heart syndrome. *CMAJ* 2003;168(13):1690-1.