

positions (because of the formula for provincial allocation of funds for post-graduate 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 article² 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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References

1. Steiner IP, Yoon PW, Kelly KD, Diner BM, Donoff MG, Mackey DS, et al. Resident evaluation of clinical teachers based on teachers' certification. *Acad Emerg Med* 2003;10:731-7.
2. Steiner IP. Emergency medicine practice and training in Canada [editorial]. *CMAJ* 2003;168(12):1549-50.

Clarifying my letter

During the editing process, a shift occurred in content of my letter to the editor¹ 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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Reference

1. Abbass A. Drug marketing priorities [letter]. *CMAJ* 2003;168(2):149.

The heart of the matter

Sandeep Arora and associates¹ 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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