### LETTERS & CORRESPONDANCE

I have to disagree with Lussier and Richard about the education of emotions. I doubt whether the much simpler emotions of animals can help us understand the complex emotions of compassion and pity. To respond to suffering with compassion requires an act of imagination in which one can visualize oneself in the situation of the sufferer. It also requires enough identification with the sufferer to understand the nature of his or her suffering, while avoiding personal involvement that could cloud one's judgment and harm the patient.

One's own emotions must be appropriate to the situation, and so medical education should include an education of the emotions, which is to say a moral education. I do not think this is unattainable. Some non-Western cultures seem to do it much better than we do. Compassion is seen as an emotion that can be nurtured and cultivated. There is evidence that in India the families of schizophrenics are more successful in dealing with emotional tensions and are able to achieve lower relapse rates than Western families. This suggests a higher level of emotional development. Martha Nussbaum's book, "Poetic justice,"<sup>5</sup> expresses these notions very elegantly. To use Dan Goleman's<sup>6</sup> term, we could say that our profession-and indeed our whole culture-lacks "emotional intelligence." In their illness narratives, our patients seem to be telling us that we often fail in our response to suffering.

Again, I would like to thank Lussier and Richard for their comments and invite others to join the debate.

— Ian R. McWhinney, MD, FRCGP, CCFP, Professor Emeritus London, Ont

#### References

- 1. Lussier M-T, Richard C. Thoughts on the importance of being different [letter]. *Can Fam Physician* 1997;43:1046-7.
- 2. McWhinney IR. The importance of being different. Part 1: The marginal status of family medicine [editorial]. *Can Fam Physician*

1997;43:193-5 (Eng), 203-5 (Fr).

- 3. McWhinney IR. The importance of being different. Part 2: Transcending the mindbody fault line [editorial]. *Can Fam Physician* 1997;43:404-6 (Eng), 414-7 (Fr).
- 4. Stewart M, Brown JB, Weston WW, McWhinney IR, McWilliam CL, Freeman TR. Patient-centered medicine: transforming the clinical method. Newbury Park, Calif: Sage; 1995.
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# Pharmacotherapy debate on alcohol withdrawal syndrome

I would like to respond to Dr Doug Bates' letter<sup>1</sup> in the March issue with regard to the use of benzodiazepines for treating alcohol withdrawal. I would not argue with using diazepam in the manner he describes for most cases, but would like to issue a strong word of caution against its use in cases of severe liver disease. In our hospital, a diazepam loading dose of 40 mg contributed to liver failure and death of a patient. This is because diazepam is metabolized in the liver and lorazepam in the kidneys. We use the latter drug initially in all cases until we have obtained results of liver function tests.

I would like to respond also to another letter in the same issue from Dr C.A. McNeill,<sup>2</sup> in which he describes the use of chlordiazepoxide as a treatment for alcohol withdrawal. In our hospital, we long ago discontinued using this drug for treating acute alcohol withdrawal because of its lipophilic character, that is, rapid storage and slow release of fat. We found it ineffective for treating acute symptoms.

> — Paul Gawthrop Nanaimo, BC

### References

- 1. Bates D. Another perspective on management of withdrawal [letter]. *Can Fam Physician* 1997;43:421-4.
- 2. McNeill CA. Article on alcohol withdrawal practical [letter]. *Can Fam Physician* 1997;43:420.

## Response

Dr Gawthrop provides an important caveat for pharmacologic treatment of alcohol withdrawal syndrome (AWS). One must use loading doses of diazepam cautiously in patients with severe liver disease.

Whether one benzodiazepine is superior to the others is a matter of debate.<sup>1</sup> The liver is the primary organ of metabolism for all benzodiazepines. Patients older than 60 years and patients with severe liver disease have impaired hepatic oxidative capacity; they are at increased risk for excessive accumulation and toxicity for most benzodiazepines, excepting lorazepam and oxazepam. Lorazepam and oxazepam undergo glucuronic conjugation only and are less likely to lead to toxicity (**Figure 1**<sup>1,2</sup>).

Liver problems, in the form of elevated aminotransferase enzymes, are common among alcoholics. Although lorazepam appears to be safer than longer acting agents, short-acting agents like lorazepam need to be given more frequently and can lead to rebound phenomenon when finally stopped.<sup>3</sup> This is important because benzodiazepines with short half-lives result in rapidly changing blood levels and require frequent administration to avoid abrupt fluctuations in blood levels that might increase risk of seizures.<sup>4</sup> Long-acting agents, on the other hand, provide smoother pharmacokinetics, improve patient comfort,<sup>5</sup> and interfere less with cognitive function.<sup>6</sup> Long-acting agents should be used when possible.

Hoey et al compared long and short-acting benzodiazepines for inhospital treatment of AWS.<sup>7</sup> They