

tised in a variety of settings. To exclude those who practise “less than” tertiary care medicine from our community is self-defeating. I believe that we will gain much more strength as a larger, more unified community.

I would encourage CAEP and our EM community at large to engage in discussions about consolidated training, and wouldn't argue against “fellowships” and other recognitions of distinction in training. I believe that our EM colleagues from smaller centres will continue to be interested in educational opportunities and research initiatives designed by those with more training and with experience in high acuity/high volume centres. I propose that we look at a broader educational strategy to support our whole EM community rather than attempt to define a one-size-fits-all definition of an emergency physician that cleaves the majority from our ranks.

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Reference

1. Lund A. I AM Emergency Medicine [letter]. *Can J Emerg Med* 2006;8(1):4-5.

Nalidixic acid overdose and metabolic acidosis

To the editor: A 15-year-old boy was recently brought to our emergency centre with lethargy and agitation after ingesting about 60 tablets of nalidixic acid (NA) in a suicide attempt. Shortly after arriving, he suffered a tonic-clonic seizure requiring intravenous diazepam. In addition to altered mentation, he was tachypneic with a respiratory rate of 40 beats/min. Cardiovascular, gastrointestinal and neurologic examinations were normal, and blood glucose was 100 mg/dL. Arterial

blood gas analysis revealed a pH of 7.31, PCO_2 of 11.3, $[HCO_3^-]$ of 5.7, PO_2 of 98.7 and oxygen saturation of 96%. Because of the profound metabolic acidosis, he was treated with 44 mEq of sodium bicarbonate ($NaHCO_3$). With supportive care, the boy recovered uneventfully after 12 hours.

Nalidixic acid poisoning may cause altered mentation, psychosis, convulsions and, rarely, metabolic acidosis.¹⁻⁴ Quinolones inhibit GABA (gamma-aminobutyric acid) receptors in the human central nervous system, potentially precipitating seizures.² Nalidixic acid interferes with lactate metabolism and may cause acidosis in predisposed patients,^{3,4} although, in this case, the acidosis may have been in part due to seizure and agitation. Sodium bicarbonate was administered intravenously because of concerns about acidosis-related myocardial and central nervous system dysfunction,⁵ but it is unclear whether this therapy was beneficial. Nalidixic acid poisoning is an uncommon problem, but should be considered in the differential diagnosis of patients presenting with unexplained metabolic acidosis and seizures. NA should not be prescribed in patients who have a propensity to develop acidosis, particularly patients with poor perfusion, sepsis or liver disease.

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References

1. Christ W, Lehnert Ulbrich B. Specific toxicology aspects of the quinolones. *Rev Infet Dis* 1988; 10 (suppl 1):141-6.
2. Tsujii A, Sato H, Kume Y, et al. Inhibitory effects of quinolone antibacteriocidal agents on GABA binding to receptors sites in rat brain membranes. *Antimicrob Agents Chemother*

1988;32:190-4.
3. Suganthi AR, Ramanan AS, Pandit N, et al. Severe metabolic acidosis in nalidixic acid overdosage. *Indian Pediatr* 1993;30:1025-6.
4. Sharma S. Lactic acidosis. Available: www.emedicine.com/med/topic1253.htm (accessed 9 Feb 2006).
5. Priestley MA, Lieh-Lai MW. Acidosis, metabolic. Available: www.emedicine.com/PED/topic15.htm (accessed 9 Feb 2006).

Public understanding of prolonged ED waiting times

To the editor: Physicians and health care leaders struggle with the growing problem of emergency department (ED) waiting times,¹ but little is known about the public understanding of these waits. We conducted a waiting room survey to assess public perceptions regarding the causes of prolonged ED waiting times at Lions Gate Hospital, a 240-bed community hospital and trauma centre in North Vancouver, BC, that has 45 000 ED visits per annum.

A trained surveyor (K.S.) invited patients and families in the ED waiting room to respond to the question, “What do you feel is most responsible for the long wait to be treated in the ED?” There were a total of 201 responses from patients who waited between 0.5 to 12.5 hours. The most common response was “Not enough doctors working” ($n = 61$; 30%). Other responses included: “Too few hospital beds, resulting in a back-up of admitted patients in the ED” ($n = 34$; 17%); “Not enough nurses working” ($n = 30$; 15%); “The ED is too small” ($n = 26$; 13%); “Too many people come to the ED for minor problems” ($n = 24$; 12%); “the ED is not operating efficiently” ($n = 22$; 11%); and “Other” ($n = 4$; 2%).

The causes of prolonged ED waiting times have been well described, and the Joint Position Statement by the Canadian Association of Emergency Physicians and the National Emergency