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Editor's note: In the course of preparing a response to Harold Merskey's letter, Andrew Malleson contacted Harald Schrader, the lead author of a study of Lithuanian drivers involved in rear-end collisions, and shared Dr. Merskey's letter with him. Dr. Schrader and his colleagues prepared their own response to the initial letter, and we have included that letter here, along with Dr. Malleson's.

Acute hepatitis associated with levofloxacin in a patient with renal insufficiency

Jon-David Schwalm and Christine Lee[1] reported a case of acute hepatitis in a hemodialysis patient taking oral levofloxacin. We observed profuse epistaxis and an acute rise in hepatic enzyme levels, particularly alkaline phosphatase, with levofloxacin therapy in a 63-year-old patient with mitral valve disease, coronary artery disease and chronic renal insufficiency, which resolved with discontinuation of the drug. The hepatic enzyme levels rose again when another drug in the same class, ciprofloxacin, was initiated. A full description of this case is available as an eletter on *eCMAJ* (www.cmaj.ca/cgi/eletters/168/7/847).

Coagulopathy associated with use of a fluoroquinolone and warfarin, as observed in this patient, is relatively well established.[2] An increase in hepatic enzymes is less well established, although it has been observed with other drugs in the same class.[3] Delayed hepatotoxicity can occur with accumulation of amiodarone (used to manage atrial fibrillation in this patient) but is usually heralded by a rise in alanine aminotransferase months after initiation of therapy, unless the reaction is idiosyncratic and occurs within the first 4 weeks.[4] The initial rise in hepatic enzymes in this patient occurred within days of initiation of levofloxacin and of the rechallenge with the second fluoroquinolone (ciprofloxacin). This patient had acute-on-chronic renal failure, as did the patient described by Schwalm and Lee.[1] The creatinine level was 212 mol/L on initiation of levofloxacin, peaking at 407 mol/L at the time of presentation with epistaxis and decreasing to 177 mol/L 4 days after discontinuation of levofloxacin. Nephrotoxicity and allergic nephritis have been linked to levofloxacin.[5] Renal dysfunction might have been the underlying problem, with altered renal clearance increasing the potential for hepatoxicity. The creatinine level did not increase with the ciprofloxacin rechallenge.

Physicians should be alert to the possibility of fluoroquinolone-associated hepatotoxicity. Comorbidities such as renal failure may increase the potential for such toxic effects.

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Violence in Liberia

Contrary to the caption below the photo of a Liberian child with an intravenous drip in his scalp,[1] children who die of cholera in Monrovia are indeed "felled by violence."

Civil wars kill more civilians than soldiers. Most of these deaths are not sustained at the front lines, but they are still a direct result of the violence. Wars ruin all the structures of civil society, immunization and health care often being among the first to go. While I was volunteering for Doctors Without Borders/Médecins Sans Frontières (MSF) in southern Sudan, many children under our care died violent deaths, mostly from malnutrition, pneumonia and dehydration. Watching a baby rigid with tetanic spasms, I was struck by the thought that this seemed a particularly violent way to die.

As physicians, a privileged and educated elite, we have a duty to be informed about how war affects health and to advocate always for peace.

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Reference
1. Not all victims of Liberia's brutal war were felled by violence [photo caption]. *CMAJ* 2003; 169(4):328.

Correction

A death notice for Dr. James E. Dimmick of Parksville, BC, who assures us that he is alive and well, was wrongly submitted to *CMAJ* and appeared in a recent edition.[1] We apologize for its publication.

Reference
1. Deaths. *CMAJ* 2003;169(6):639.