Asian physician pursuing graduate studies in a Canadian institution, and the online availability of the latest medical literature through my university's subscription has opened up a new world for me, helping me to improve the quality of my research and my understanding of the issues. I am already dreading the loss of this privilege when I return home.

Medical schools and research centres in developing countries often cannot pay for the high cost of online journal access, and subscriptions to print versions are limited. In many cases, researchers have access only to abstracts (through PubMed [www.ncbi.nlm.nih.gov/entrez/query.fcgi] and, more recently, Google Scholar [www.scholar.google.com]). It is difficult for residents on limited stipends to buy even single articles, which cost anywhere from US$10 upward. Furthermore, Internet access is limited, and safe online banking and credit card use are not available. As a result, residents and scientists use outdated sources for their research, which is reflected in the final quality and scientific rigour of their work.

The initiatives promoting open access that have been undertaken by *CMAJ*, BioMed Central (www.biomedcentral.com/), SciDev.Net (www.scidev.net/) and the Public Library of Science journals, among others, are laudable. However, the practice of making authors pay for online publication of their articles, as described in the *CMAJ* editorial,[1] might dissuade researchers in developing countries from sharing their research results in international journals. Special discounts will need to be worked out, and journals will need to continue exploring innovative ways to support progress in open access and offset their costs.

CMAJ's experience has shown the advantages of an open-access policy.[1] I hope that the journal continues its leadership in promoting equal opportunities and access in the global medical community.

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Reference

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DKA and thrombosis

Josephine Ho and associates[1] report an unfortunate case of a 6-year-old girl with diabetic ketoacidosis (DKA) and thromboembolic stroke. Although the authors do a credible job of describing the diverse causes of pediatric stroke and the controversies surrounding treatment of children, there was little emphasis on the danger of extreme hyperosmolar states and risks of thrombosis. More information about the initial presentation of the patient, with specific reference to the concentration of serum sodium and serum osmolarity, would have been helpful in determining her risks of thrombosis.

Diabetes is associated with a prothrombotic state through a number of mechanisms.[2] The mostly adult entity of hyperosmolar nonketotic coma has had various degrees of association with thrombosis,[2,3] as has extreme hypernatremia in breast-feeding neonates.[4] Recent evidence has also demonstrated that among children with DKA, there is a higher incidence of deep venous thrombosis with femoral central venous lines.[5,6] Serum glucose and sodium concentrations and hence effective plasma osmolarity were significantly higher in those patients with blood clots.[5]

Although there is no direct evidence for its efficacy, our practice has been to use prophylactic anticoagulation in patients with DKA who are in a significant hyperosmolar state, as well as to eliminate the use of femoral catheters in patients with these risk factors. There is significant controversy surrounding the dose of anticoagulant therapy, specifically whether the efficacy of dosages for prophylaxis of deep venous thrombosis outweighs the risks associated with full systemic anticoagulation.[7] As with most clinical issues, particularly in pediatric critical illness, this controversy lends itself well to a clinical trial in patients with extreme hyperosmolar states, including those with DKA.

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[Three of the authors respond:]

Jeff Burzynski raises an interesting point about the danger of the hyperosmolar state and risk of thrombosis. In the patient that we described (a 6-year-old girl with DKA and stroke),[1] the initial serum sodium level was 132 mmol/L and initial blood glucose, 43.4 mmol/L. The corrected sodium level was 144 mmol/L with a calculated serum osmolarity of 331 mOsm/L. We agree that patients with DKA have hyperosmolarity because of hyperglycemia and hypernatremic dehydration, and we[1] and others[2,3] have suggested that the hyperosmolarity contributes to the prothrombotic tendency of children with DKA.

Worly and associates[2] described 3 patients aged 14–18 months with DKA and calculated serum osmolarity of 291–356 mOsm/L who experienced deep venous thrombosis associated