There exists strong evidence that interdisciplinary stroke rehabilitation leads to better functional outcome than does usual care.³ Although there is less evidence regarding the timing of rehabilitation, the need for such services must be determined during acute care to avoid missing this important component of overall stroke care.

We therefore propose that an additional indicator be included for optimal stroke care: timely assessment for rehabilitation when appropriate.

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DOI:10.1503/cmaj.1050085

[Two of the authors and a colleague respond:]

We agree with the comments of Andrew Dawson and colleagues. In fact, organized care in an inpatient stroke unit — which includes early mobilization and rehabilitation — is the first key indicator in our paper¹ and is effective in improving outcomes after stroke.^{1,2} We recognize that there is an overemphasis in the literature on hyperacute stroke treatments relative to the benefits of several rehabilitation therapies, which apply for the majority of stroke survivors.

The Canadian Stroke Quality of Care Study, from which the acute care indicators emerged, is an ongoing multiphase study. Our goal is to build a comprehensive evaluation framework

that measures patients' access to appropriate stroke care, according to their particular symptoms, as well as the flow of patients receiving such care. This model will include indicators that reflect care within each sector along the continuum of care; more importantly, the current work will build the critical indicators reflecting true integration between the points along the continuum, including transition from acute care to rehabilitation, and from rehabilitation to community care and recovery.

We and many other researchers are at work on the development of stroke rehabilitation indicators. This research suggests that tools such as the Functional Independence Measure, the Barthel Score and the Orpington Score may be used to facilitate referral and to measure the transition between acute care and rehabilitation, but the ideal tools for tracking patients from inpatient care into the community are still unclear.

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DOI:10.1503/cmaj.1050116

Are children with type 1 diabetes immunocompromised?

In their clinical report of a 4-year-old child with leukemia and an enlarging arm lesion that proved to have been caused by an opportunistic fungus, Ahmed Mater and associates¹ state that "[these i]nfections generally occur in immunocompromised patients with conditions such as neutropenia, diabetes or hematologic malignant disease."¹ This statement implies that all patients with type 1 or type 2 diabetes mellitus are immunocompromised. Our interest is children (up to 18 years of age) with type 1 diabetes, and we challenge the accuracy of the statement in this context.

Mater and associates¹ cite 2 papers^{2,3} that listed "diabetes," specifically diabetes complicated by ketoacidosis, as a risk factor for opportunistic infections. However, those articles did not provide evidence to support this claim in children with type 1 diabetes. Is there any evidence to show increased rates of infection or prolonged recovery from infection in children with type 1 diabetes? In-vitro data have demonstrated impaired immune function due to hyperglycemia and/or hypoinsulinemia in association with type 1 diabetes.^{4,5} However, those studies did not show that the differences in cell-mediated and humoral immune function translate into significant morbidity or mortality in the clinical setting. In fact, the humoral response to influenza vaccine in patients with type 1 diabetes is no different from that of controls with respect to protection rates.⁶ The incidence of candidal infection is greater

among patients with type 1 diabetes, but the reason for this is unclear.^{7,8} It may be due to a genetic polymorphism in the gene encoding β -defensin 1.⁸ However, there is no evidence that this genetic difference leads to an immunocompromised state allowing invasive fungal disease to occur. There have been case reports of patients with type 1 diabetes and diabetic ketoacidosis in whom severe opportunistic infections have developed.⁹ The increased susceptibility may be attributed to the short-term acidic environment of diabetic ketoacidosis, which is ideal for certain opportunistic pathogens.

In summary, there is insufficient evidence to conclude that children with type 1 diabetes mellitus are immunocompromised. The evidence indicates that an immunocompromised state occurs only in the context of poor glycemic control with severe complications such as diabetic ketoacidosis or in adults with vasculopathy and peripheral neuropathy. Fortunately, with modern standards of care and education of families to manage intercurrent illness in their children with type 1 diabetes mellitus, hospital admission for diabetic ketoacidosis is now rare.

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DOI:10.1503/cmaj.1050095

Effect of thiazolidinediones on lipid profile

In their review of oral hypoglycemic therapy in type 2 diabetes mellitus, Alice Cheng and George Fantus¹ mention the effect of thiazolidinediones on high-density lipoprotein (HDL) cholesterol; I would like to add some comments about the effects of these agents on low-density lipoprotein (LDL) cholesterol and triglycerides.

In fact, the effect of thiazolidinediones on serum lipids and lipoproteins varies with the agent used (pioglitazone or rosiglitazone). As noted by Cheng and Fantus, HDL levels increase with either of these 2 drugs.²⁻⁷ However, LDL cholesterol levels remain unchanged with pioglitazone monotherapy or a combination of pioglitazone with other oral hypoglycemic agents or insulin.²⁻⁴ In contrast, LDL cholesterol levels increase with rosiglitazone monotherapy or combination therapy.⁵⁻⁷ Although pioglitazone has been associated with a decrease in triglyceride levels,²⁻⁴ the effects of rosiglitazone on triglycerides have been variable, ranging from a 2% increase to a 19% decrease.^{6,7}

Studies directly comparing the 2 agents are scant, and the cause of this variation in lipid levels is unknown.

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Competing interests: None declared.

DOI:10.1503/cmaj.1050034

[The authors respond:]

We appreciate Pankaj Madan's supplementary information to our article on oral antihyperglycemic therapy.¹ As Madan has correctly outlined, the studies comparing pioglitazone (monotherapy or combination therapy) with placebo have demonstrated no changes in LDL cholesterol,²⁻⁴ whereas studies comparing rosiglitazone (monotherapy or combination therapy) with placebo have demonstrated an increase, ranging from 8% to 19%, in LDL cholesterol.⁵⁻⁷ In clinical practice, this elevation may have a small impact, if any, for patients with diabetes mellitus using lipid-lowering therapy (statins) to achieve target LDL levels.^{8,9}

The lack of direct-comparison studies makes it difficult to draw definitive conclusions regarding the lipid differences between the 2 medications. Simi-