OBSERVATIONS

Assessing Dependency in Insulin-Treated Patients With Diabetes Supported by a Community Nursing Service

Patients with insulin-treated diabetes deemed incapable of self-care present community nurses in the U.K. with a significant work load. Factors determining such dependency are unreported. We undertook an audit to identify the care received by such patients with the aim of giving back or increasing independence wherever possible.

Patients visited by the Wolverhampton Community Nursing Service to assist with their insulin were identified, and dependency was evaluated using uniform criteria. The potential for increased self-care and independence was assessed by community nurses. Diabetes nurse specialists were subsequently involved further to determine whether a decrease in dependency was possible.

There were 88 patients aged 76 ± 10 (40–95) years (mean ± SD [range]) who were identified. Of these, 28 patients (32%) were on once- and 60 (68%) on twice-daily insulin. Visits occurred twice daily in 24 patients (27%), once daily in 50 patients (57%), and less frequently in 14 patients (16%) (36,950 visits/year). Procedures carried out were as follows: insulin drawing up (75 patients), injecting (46), technique checking (24), blood glucose monitoring (7), and others (7).

Initial dependency factors were as follows: state of mind (n = 50, 57%; 28 patients with dementia, 9 refusing self-care, 8 with debilitating anxiety, and 5 with psychotic disorders), visual impairment (n = 53, 60%), poor dexterity (n = 30, 34%), stroke (n = 18, 20%), and social problems (n = 36, 41%), mainly social isolation (n = 31). Each category constituted the single major dependency factor in 50 (57%), 14 (16%), 18 (20%), 6 (7%), and 0 patients, respectively. The community nurses considered 40 (45%) patients capable of further independence and 48 (55%) not. For those not capable, there was total agreement by the diabetes nurse specialists. Of those seen as capable of greater independence (n = 40), only five became independent, and dependency was reduced in seven (n = 12; nine [75%] patients with visual problems that were overcome by the use of preloaded insulin pens and three with dexterity problems). There were 28 patients who did not proceed to increased independence; in 21, this was due to psychological/psychiatric problems. Reevaluation of patients falling into the finally dependent group (n = 76) showed the following barriers: state of mind (n = 50, 66%), dexterity (n = 15, 20%), stroke (n = 6, 8%), and visual problems (n = 5, 7%; all compounded by dexterity problems). The workload reduction for the community nurses after this process was 4,361 visits/year (11.8%).

Insulin-treated diabetic patients dependent on community nursing present a significant caseload. Overcoming visual and dexterity problems made a small but important contribution both to establishing patient independence and to reducing nursing workload. Greatly underestimated and previously unrecognized was the contribution of state of mind to dependency. No patient falling into this category gained independence. Our study is the first to document this finding, and it provides data for assessing dependency and suggests the need for a close liaison between community services and specialist diabetes services in assessing the need for continuing care in community-dependent patients with insulin-treated diabetes.

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Transient Hypoglycemic Hemiparesis in Children With IDDM

Focal neurological deficits complicating hypoglycemia are well-described in adults (1). However, they are uncommon in children. Although the mechanism of the neurological deficit remains unclear, the selectivity of the neurological damage caused by hypoglycemia implies that different areas of the brain have different degrees of vulnerability (2). Cerebrovascular disease may be implicated in the pathogenesis of hemiparesis in adults, but this is most unlikely to be the case in children.

We observed nine episodes of acute transient hemiparesis following nocturnal hypoglycemia in five young children with IDDM.

In all five children, IDDM had been diagnosed at an early age, between 1 and 4 years. They were receiving a combination of intermediate and short-acting insulin, twice a day, at a dose of 0.7–1.0 U · kg^{-1} · day^{-1}. At the time of the episode, HbA_1 varied between 6.5 and 11.4% (nondiabetic range 4.7–7.9) in seven cases. In the other two episodes, HbA_{1c} was 7.0 and 8.2%, respectively (nondiabetic range 4.0–5.8).

There was no past medical or family history of migraine. Although two children had a previous history of generalized convulsions attributed to hypoglycemia, none had a history of a seizure disorder. In all cases, the severe hypoglycemic episode occurred in the early hours of the morning, usually when the child was still asleep. Capillary blood glucose checked in eight out of nine episodes ranged between 1.9 and 3.0 mmol/l.

All of the children presented with confusion and with weakness on one side of their body. Two children also had dysphasia, and one of them had ataxia.

Three of five cases had more than one episode of severe hypoglycemia. This confirms previous reports that children with severe hypoglycemia are likely to have a history of similar episodes (3). The episodes of hypoglycemic hemiparesis were transient, lasting between 15 min and 2 days. The timing of neurological recovery may be related to the length and severity of the hypoglycemic insult. No signs of any residual neurological dysfunction and no evidence of any cognitive impairment could be noted.

All five children were right-handed, and in four of them, the affected side was the left, as previously reported (4). This might suggest that the nondominant hemisphere is more susceptible to hypoglycemia. However, other reports have shown it to be the right side that is more commonly affected. In those children who had recurrent episodes, the affected side

was always the same. In three of the cases, cerebral Doppler blood flow studies, performed between 2 and 6 months after the episodes, were normal. Computed tomography (CT) scan and magnetic resonance imaging (MRI) of the brain were normal in the two cases in which these investigations were performed. Glucose and counterregulatory hormone profile (adrenaline, cortisol, glucagon, growth hormone) were studied in three cases, and no abnormalities were seen. However, two of the cases did not become hypoglycemic during the period of overnight monitoring. In the third case, the blood glucose fell to 2.7 mmol/l, but this was probably not low enough to trigger an autonomic response. The threshold for this response is determined by the level of prevailing glycemic control. In fact, those with tight control need to reach a lower blood glucose before activating their autonomic response.

An electroencephalogram (EEG) in one child, 7 days after an episode of transient hemiparesis, showed a significant abnormality, with persistent multifocal spikes and predominantly slow activity in the right posterior quadrant. Two months later, when the EEG was repeated, there was considerable improvement of the right posterior focus, but there were still foci of sharp and slow waves more marked on the left. EEG abnormalities in diabetic children seem to be related to young age, early onset of diabetes, and a history of severe hypoglycemic episodes (5).

The Diabetes Control and Complications Trial (DCCT) demonstrated that intensive insulin treatment increases the incidence of severe hypoglycemia in adult patients with IDDM. This has implications in the choice of the range of glycemic control that is appropriate for young children. Children receiving more than one injection of insulin per day may experience more severe hypoglycemia and may have a greater susceptibility to hypoglycemia because of strict glycemic control (6).

The mechanism underlying hypoglycemia-induced hemiparesis is unclear. Current hypotheses include cerebral vasospasm, impaired cerebral autoregulation, selective neuronal vulnerability (2), and underlying cerebrovascular disease. In adults with long-standing diabetes, there is an increased frequency and severity of cerebrovascular atherosclerosis, which may contribute to transient hemiparesis. However, such cerebrovascular complications are unlikely in children.

Different regions of the nervous system demonstrate variable responses to systemic insult such as anoxia, intoxicant, and metabolic disorders. The selectivity of damage caused by hypoglycemia in the nervous system is of considerable interest. The neurones of the middle layer of the cerebral cortex are most affected, the basal ganglia and anterior thalamus are the next most sensitive, while the brainstem and the spinal cord are the most resistant. Vascular spasm and impairment of cerebral autoregulation may also be implicated (2). Previous studies have shown that hypoglycemia causes a symmetrical increase in cerebral blood flow (7); however, a recent study has shown that mild hypoglycemia is associated with an increase in cerebral blood flow, which is greater in the gray matter and in the right hemisphere (8). This may explain the laterality of neurological deficits observed during severe hypoglycemic episodes.

In conclusion, we think that children who are diagnosed to be affected by IDDM before the age of 5 years are particularly at risk of transient hemiparesis associated with episodes of hypoglycemia. These signs are more likely to recur in the same children. The consequences of recurrent episodes of transient hemiparesis on cognitive function and neurological development have yet to be studied, and the long-term risk for these children remains unknown. Tight glycemic control to prevent long-term complications is likely to increase the frequency and severity of hypoglycemic episodes and may carry risks in young children.

Severe hypoglycemia is associated with EEG abnormalities whose clinical relevance is not known. Diabetic children with documented EEG abnormalities may be at increased risk of brain injury during recurrent hypoglycemic states. Future neurological follow-up of these patients is important as the significance of these episodes is unclear.

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No Differences in Attentional Functioning Between Type 1 Diabetic Patients With and Without a History of Severe Hypoglycemia

In past years, increasing concern has arisen about the long-term detrimental effects of recurrent severe hypoglycemia on neurocognitive abilities in type 1 diabetic patients (1,2). There is convergent evidence that in children, recurrent episodes of severe hypoglycemia contribute to a slowed mental development and may reduce eventual IQ level (3). There is less agreement about the effects of hypoglycemia on cognitive functioning in adults with type 1 diabetes. Evidence gathered from different studies suggests that