

Introduction

Diabetes mellitus (DM) is a chronic disorder of carbohydrate metabolism caused by abnormal insulin function or insulin deficiency, which results in elevated blood sugars. The influence of DM on human health is enormous and, unfortunately, is increasing steadily, in terms of overall health, mortality and economic impacts (Center for Disease Control and Prevention, 2002). World-wide prevalence rates have risen dramatically over the past 10 years in every age, sex, race, and education category (Crooks, Buckwalter, & Petitti, 2003) and this trend is continuing in epidemic proportions (Wild, Roglic, Green, Sicree, & King, 2004). DM ranks first in US direct healthcare costs compared to any other chronic disease (American Diabetes Association, 1998). It is the fifth leading cause of death in the US (Center for Disease Control and Prevention).

Types of DM

Type 1 DM (T1DM) is characterized by a lack of insulin production. Type 2 DM (T2DM) is characterized by gradual insulin resistance. Defective insulin production leads to chronically high blood glucose levels (hyperglycemia). If insulin is not produced, carbohydrates cannot be metabolized, which results in high glucose levels in the blood stream; while the body, particularly the brain, cannot utilize the glucose it “starves” (Desrocher & Rovet, 2004). T1DM affects individuals at a young age and requires that exogenous insulin be injected several times a day. Individuals with T2DM acquire insulin resistance over several years and may be many can regulate blood glucose levels through weight control, medication and exercise.

DM and Neurogenic Communication Disorders: Shared Etiologies and Concomitant Diagnoses

People with diabetes represent a significant proportion of those diagnosed with neurogenic conditions, especially stroke. For many, the etiology underlying the diagnosis of DM is the same or similar to etiologies that lead to neurogenic communication impairments. This is not surprising, given that DM is associated with long-term complications that affect almost every part of the body, often leading to high cholesterol, heart and blood vessel disease, peripheral cardiovascular disease, hypertension, obesity, retinopathy, blindness, nephropathy and renal failure, poor or absent teeth, depression, arthritis, decubitus ulcers, and required amputations (National Institute of Diabetes and Digestive and Kidney Diseases, 2007).

People with diabetes have significantly higher incidence than others of numerous conditions likely to affect cognition and communication. These include stroke, focal lesions not attributable to stroke, accelerated brain atrophy, atherosclerosis, proliferative lesions of cerebral vessels, peripheral and autonomic neuropathies, vascular dementia, Alzheimer’s disease (de Groot, Anderson, Freedland, Clouse, & Lustman, 2001; Guthrie & Guthrie, 2004; Logroscino, Kang, & Grodstein, 2003; Peila, Rodriguez, & Launer, 2002; Vanhanen et al., 1999; Worrall, Chaulk, & Moulton, 1996).

Numerous authors have reported that individuals with diabetes are more likely than those without diabetes to have problems with:

- Memory (Tun, Nathan, & Perlmutter, 1990), including problems associated with constructs of verbal memory (Cosway et al., 2001; Elias, D’Agostino, Elias, and Wolf, 1995; Greenwood, 2003; Hassing et al., 2004), visual memory (Elias et al.), short-term memory (den Heijer et al., 2003; Vanhanen et al., 1999; Worrall, Moulton, & Briffett,

1993), 15-minute delayed recall (den Heijer et al., 2003), working memory (Arvanitakis, Wilson, Bienias, Evans & Bennett, 2004), episodic memory (Arvanitakis et al; Hassing et al., 2004; Wahlin, Nilsson, & Fastbom, 2002), and semantic memory (Arvanitakis, et al, 2004; Worrall et al., 1993);

- Attention (Ack, Miller, & Well, 1961; Klein & Waxman, 2003; Rovet, Ehrlich, & Hope, 1988; Ryan, 2001; Ryan et al., 1992; Weinger & Jacobson, 1998; Worrall, et al, 1993; Meneilly, Cheung, Tessier, Yakura, & Tuokko, 1993; Nilsson, Fastbom, & Wahlin, 2002);
- Psychomotor skills (Hassing et al., 2003; Ryan, Geckle, & Orchard, 2003; Ryan, Williams, Orchard, & Finegold, 1992; Weinger & Jacobson, 1998);
- Visuospatial and visuoconstructional ability (Arvanitakis, Wilson, Bienias, Evans & Bennett, 2004; Franceschi et al., 1984; Ryan, 2001; Ryan, Vega, and Drash, 1985; Ryan, Williams, Orchard, and Finegold, 1992);
- Orientation (Ciotti et al., 1986; Worrall, Moulton, & Briffet, 1993; Croxson & Jagger, 1995; Lowe, Tranel, Wallace, & Welty, 1994; Zelinski, Crimmins, Reynolds, and Seeman, 1998);
- Word finding, verbal fluency, and generative naming (Helkala, Niskanen, Viinamaki, Partanen, & Uusitupa, 1995; Kanaya, Barrett-Connor, Gildernorin, and Yaffe, 2004; Lowe, Tranel, Wallace, and Welty, 1994; Perlmutter, Tun, and Sizer, 1987; Wahlin, Nilsson, and Fastbom, 2002);
- Problem-solving (Ryan, 1988; Klein & Waxman, 2003; Meneilly, Cheung, Tessier, Yakura, & Tuokko, 1993; Ryan, Vega, And Drash, 1985), including problems associated with constructs of visuospatial problem-solving (Meneilly et al.), abstract reasoning (Franceschi et al., 1984; Klein & Waxman, 2003; Skenazy & Bigler, 1984); and
- Executive functions (Helkala, Niskanen, Viinamaki, Partanen, & Uusitupa, 1995; Strachan, Deary, Ewing, & Frier, 1997).

Additionally, increased incidence of hearing loss and the effects of diabetic conditions on hearing are well documented (Cullen & Cinnamon, 1993; Dalton, Cruickshanks, Klein, Klein, & Wiley, 1998; Parving, 1991).

Recent research demonstrating that thyroid autoimmunity is increased in type 1 and type 2 diabetics, and that hyperglycemia can contribute to an exaggerated immune response, supports increasing evidence that T2DM is an immune-related disease (Autoimmune Disease Coordinating Committee, 2005). Likewise, there is a growing body of research linking vascular disease to autoimmune disease. Even in the absence of events that are common in people with DM and that have well-known associations with memory and language deficits, such as cerebrovascular events, uncontrolled hyperglycemia may be related to cognitive changes resulting from vascular defects in the blood-brain barrier, hypertension, and peripheral or autonomic neuropathy (Biessels, 1999; Ryan, Williams, Orchard, & Finegold, 1992). A great deal remains to be learned about connections between underlying etiologies of DM and neurogenic deficits in cognition and communication (Libby & Ridker, 2004).

Despite robust evidence of diabetes-associated cognitive and communicative deficits identified through research involving psychometric testing, little is known about the degree to which patients are aware of and affected personally by such deficits. Hallowell (2007) surveyed 2,608 individuals with diabetes (with diagnosis validated via glucose and HbA1c measures) to study

relationships between the diagnosis of diabetes and self-reported communication problems. After removing all participants with a history of stroke or brain injury and adjusting for age, diagnosis of diabetes remained a significant independent predictor of overall communication scores as well as each of the six communication indices studied. In further research, it will be crucial to include patient-generated observations about these cognitive-communicative issues, not simply a priori categories derived from analyses of assessment battery results.

Research Needs Related to Diabetes and Clinical Aphasiology

Given the paucity of studies offering empirical data on the effects of diabetes on speech and language disorders, further investigation of this area is sorely needed. A critical first step for ensuring the clinical relevance and social validation of qualitative and quantitative research in this area is to explore whether and how individuals with diabetes indicate concerns about or awareness of problems with aspects of communication. Additionally, it is critical to know whether and to what degree specific difficulties in cognition and communication are due to acute changes in glucose levels or, rather, to more chronic etiologies associated with the long-term metabolic effects of erratic glucose control, hyperglycemia or hypoglycemia. Thus, further empirical study of diabetic participants involving thorough communication evaluations, fasting blood glucose and glycosylated hemoglobin tests, with age-matched non-diabetic controls, are also needed.

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