was not conducted, and subjects were brittle type I diabetic subjects with possible cerebral dysfunction due to hypoglycemic episodes (3–5). In type II diabetic subjects, a defect in memory retrieval has been shown (6), although the study design was different from this investigation, in that it was cross-sectional and testing was conducted at one time point. It is not clear if the defect reported is related to glucose concentrations, a feature of diabetes other than hyperglycemia, or the effect of chronic illness.

In this study, no changes in vocabulary recall or abstract logic were demonstrated as a function of spontaneous variations of serum glucose and osmolalities encountered. It is possible that other types of psychological testing or wider ranges of glucose concentration and osmolality might be necessary to demonstrate any effects of moderate hyperglycemia. However, if higher glucose concentrations are studied, they would have to be examined in healthy subjects to avoid the complicating effects of stress and chronic illness. Therefore, it appears possible that symptoms of dysmentation reported by diabetic subjects with moderate hyperglycemia may be referable to elevated glucose concentrations, osmolalities, or other associated metabolic derangement, but the degree of abnormality necessary to cause cognitive dysfunction is probably greater than that reported in this study.

LAWRENCE PARKER, MD
CHANG-LIM KIM, MD
EDWARD HESS, MD
JOSE RABELO, MD, PhD
RICHARD CHARTER, PhD

From the Endocrinology Section, Medical Service, and Psychology Service, University of California at Irvine-Long Beach Medical Program, Long Beach, California.

Address correspondence and reprint requests to Lawrence Parker, MD, Endocrinology Section, Veterans Administration Medical Center, 5901 East Seventh Street, Long Beach, CA 90822.

REFERENCES

1. Shipley W: A self-administered scale for measuring intellectual impairment and deterioration. *J Psychol* 9:371–77, 1940
2. Wing RR, Epstein LH, Lamparski D, Hagg SA, Nowalk MP, Scott N: Accuracy in estimating fasting blood glucose levels by patients with diabetes. *Diabetes Care* 7:476–78, 1984
3. Franceschi M, Cecchetto R, Minicucci F, Smizne S, Baio G, Canal N: Cognitive processes in insulin-dependent diabetes. *Diabetes Care* 7:228–31, 1984
4. Skenazy J, Bigler E: Neuropsychological findings in diabetes mellitus. *J Clin Psychol* 40:246–58, 1984
5. Holmes C: Neuropsychological profiles in men with insulin-dependent diabetes. *J Consult Clin Psychol* 54:386–89, 1986
6. Perlmuter L, Hakami M, Hodgson C, Ginsberg J, Katz J, Singer D, Nathan D: Decreased cognitive function in aging noninsulin-dependent diabetic patients. *Am J Med* 77: 1043–48, 1984

Nesidioblastosis Versus Islet Cell Hyperplasia

In the February issue of *Diabetes Care,* Fong et al. (1) presented three cases of nesidioblastosis occurring in adults and added these to seven previously reported cases. They speculated, based on their own experience (30% of patients) and from other literature (2), that cases of nesidioblastosis may be underreported as a cause of hyperinsulinemic hypoglycemia.

We reported a case in 1986 that met all the criteria for nesidioblastosis in an adult but titled the paper "Islet Cell Hyperplasia" as a way of making some important points (3). Our review of the literature showed that many authors had used the term *nesidioblastosis* to describe different pathological processes. Furthermore, many reports had stressed other hyperplastic changes in hyperinsulinism in addition to the budding of endocrine cells from ducts. We proposed the less restrictive definition of islet cell hyperplasia to identify the diffuse proliferation of endocrine cells with different morphological patterns and different proportions that vary from case to case.

Clearly, by conducting a literature search for adult cases of nesidioblastosis, Fong et al. easily overlooked our report. That fact reinforces the need for more discussion of our proposal to redefine the entity of hyperinsulinemic hypoglycemia where no discrete adenoma is found. The editorial by Rahier (4) emphasized the lack of specificity of nesidioblastosis in association with hyperinsulinism. We agree but stress that by avoiding the restrictive term *nesidioblastosis* and instead including all patterns of islet cell hyperplasia together, a more consistent histological diagnosis will be used. We suspect the true incidence will be ~6–7% of all cases of hyperinsulinemic hypoglycemia found by Stefanini et al. (5) in their series of 1067 cases.

As indicated by Fong et al., the diagnostic and therapeutic problems presented by hyperinsulinemic hypoglycemia remain difficult. Their observation that proinsulin values in the normal range may help to differentiate adenomas from a diffuse process may be useful. Routine serum insulin and C-peptide determinations have largely replaced proinsulin levels in the workup of insulinomas in the last several years. Especially in patients with no definite preoperative localization by radiological and pancreatic vein sampling techniques, a proinsulin measurement should now be considered. Unfortunately, because of the rarity of the condition, it will require several years for comparison data to prove the true usefulness of this measurement as a discriminating and predictive factor. Until then, physicians and surgeons involved in the preoperative management of these patients must continue to be prepared for an intraoperative decision about the extent of surgical resection.

PAUL MARGULIES, MD
ELLEN KAHN, MD

Downloaded from http://diabetesjournals.org/care/article-pdf/12/10/751/438276/12-10-751.pdf by guest on 09 August 2022

From the Departments of Medicine and Laboratories, North Shore University Hospital, and Cornell University Medical College, Manhasset, New York.
Address correspondence and reprint requests to Paul Margulies, MD, 444 Community Drive, Manhasset, NY 11030.

REFERENCES

1. Fong TL, Warner NE, Kumar D: Pancreatic nesidioblastosis in adults. *Diabetes Care* 12:108–14, 1989
2. Schein PS, Delellis RA, Kahn CR, Gorden P, Kraft AR: Islet cell tumors: current concepts and management. *Ann Intern Med* 79:239–57, 1973
3. Weinstock G, Margulies P, Kahn E, Susin M, Abrams G: Islet cell hyperplasia: an unusual cause of hypoglycemia in an adult. *Metabolism* 35:110–17, 1986
4. Rahier J: Relevance of endocrine pancreas nesidioblastosis to hyperinsulinemic hypoglycemia. *Diabetes Care* 12:164–66, 1989
5. Stefanini P, Carboni M, Patrassi N, Basoli A: Beta-islet cell tumors of the pancreas: results of a study in 1,067 cases. *Surgery* 75:597–609, 1974

Readability of Diabetes Self-Report Measures

The recent proliferation of self-report measures designed to assess patients' knowledge, attitudes, and behavior regarding diabetes suggests a need for evaluation of the readability of these instruments. This information could be useful to both clinicians and researchers in the selection of measures appropriate to their respective patient populations, and it could serve a sensitizing function for those involved in the future development of such instruments. Consequently, we analyzed the readability of 26 diabetes self-report instruments with the Flesch reading ease formula (1,2). This is a well-validated and reliable method that yields a reading ease score and a grade equivalent based on calculation of average sentence length and average number of syllables per word. Table 1 shows the reading ease and grade equivalent

TABLE 1
Flesch reading ease scores for directions and stimulus items of 26 diabetes self-report measures

Measure	Refs.	Directions		Stimulus items	
		Flesch reading ease score	Grade equivalent	Flesch reading ease score	Grade equivalent
Self-Efficacy for Diabetes Scale	3	87.0	6	87.2	6
Diabetes Opinion Survey-R4	S.B. Johnson, unpublished observations	90.6	5	78.2	7
Diabetes Educational Profile 1980 version, part A	4	78.0	7	77.0	7
Sullivan Diabetic Adjustment Scale	5	96.0	5	77.0	7
Barriers to Adherence Questionnaire	6	80.1	6	76.2	7
Hypoglycemic Fear Survey	7	75.9	7	75.9	7
Diabetes-Specific Perceived Social Support	8	55.0	10–12	75.3	7
Diabetes Care Profile 1984 version	9	90.0	5	75.0	7
Diabetes Regimen Adherence Questionnaire	10	58.0	10–12	74.0	7
Parents' Diabetes Opinion Survey-R4	S.B. Johnson, unpublished observations	81.2	6	71.7	7
How Do You Feel About Diabetes and Its Treatment?	8	90.3	5	71.5	7
Test of Diabetes Knowledge R2	11	54.9	10–12	71.0	7
Insulin Dependent Patient Questionnaire	12	67.0	8,9	69.0	8,9
DKN Scales	13			69.0	8,9
Diabetes Self-Care Behaviors	8	42.9	13–16	68.0	8,9
Diabetes Health Belief Scale	14			68.0	8,9
Diabetes Educational Profile	15	95.6	5	67.6	8,9
Diabetes Quality-of-Life Scale	16			67.0	8,9
Attitude Scale Statements	17			65.6	8,9
Non-Insulin Dependent Patient Questionnaire	12	67.0	8,9	64.0	8,9
ATT39	18			62.0	8,9
Diabetes Knowledge and Management Skills Questionnaire	10	54.0	10–12	61.3	8,9
Health Belief Questionnaire	10	58.0	10–12	59.0	10–12
Diabetes Family Behavior Checklist	19	77.9	7	53.5	10–12
Diabetes Knowledge Test	20	71.0	7	51.0	10–12
Diabetes Information Test	21	47.0	13–16	48.0	13–16

Downloaded from http://diabetesjournals.org/care/article-pdf/12/10/751/438276/12-10-751.pdf by guest on 09 August 2022