rent proficiency and competence in this area. Because it is a voluntary process that exceeds the minimum entry-level requirements for practice within a profession, it should be distinguished from a license to practice diabetes education (1). The CDE examination, like most certification programs, has as its main objective to see that the public receives quality care by upgrading management practices above the minimum licensure set by each health discipline. It must be taken every 5 yr to retain the title of CDE. Nevertheless, some concern has arisen that the examinations are too basic and may convey a false sense of confidence about areas for which groups of educators have not been adequately trained (e.g., a nurse providing complete nutritional counseling) (2).

The introduction of the CDE comes at the same time that the endocrinology fellowship population is declining. Of the nine traditional internal medicine subspecialties, only endocrinology and nephrology experienced a decrease in fellows between 1976 and 1988 (3). The number of first-year endocrine fellows decreased by 24%, whereas the number of all endocrine fellows decreased by 10% during this 12-yr period. By contrast, total fellows increased in infectious disease (43%), cardiology (40%), pulmonary diseases (25%), gastroenterology (14%), rheumatology (12%), and hematology and oncology (4%) (3). Although one could speculate about the etiologies for the decline in endocrinology fellows, certainly a major reason is the relatively poor reimbursement for the cognitive-oriented medical subspecialties (4). Because the prevalence of diabetes appears to be increasing (5), it seems clear that an increasing number of nonendocrinologists, including nonphysicians, such as qualified nurse practitioners, nutrition specialists, and health educators, will need to participate in the care of this patient population.

Therefore, 6 yr and 6200 CDE certificates later, we feel that it is appropriate to assess the progress of this new niche of educators. Unfortunately, we do not have any data to report. However, we are quite concerned about the common theme of complaints expressed by diabetes educators across the country, namely, that they are having a difficult time interacting with the referring physicians. In particular, many doctors feel intimidated or threatened by nurse or nutrition educators who, after passing the CDE examination, are often better trained in certain aspects of diabetes education and care than the well-intentioned physician. The result of this phenomenon is frustration among the educators, because they are qualified to provide a greater portion of the diabetes management than they are currently allowed to perform. Furthermore, it is likely that overall diabetes care would improve with increased input from the educator. It is time to move the concept of the diabetes team out of the large diabetes center and toward the primary-care physicians who manage over 90% of the diabetic patients in this country.

What are some possible solutions to these problems? First, and probably most important, we need to educate those doctors who are currently underusing the CDE. This could be achieved through articles in primary-care journals or continuing medical education programs, such as Clinical Education Program III sponsored by the American Diabetes Association. The National Certification Board for Diabetes Educators is now assisting the new or recertified educator by informing their employers of CDE certification, but perhaps this program could be expanded to include a greater number of physicians and administrators. Another problem, for which no simple solutions exist, is how to adequately reimburse educators for their highly skilled expertise. Most diabetes educators/specialists are paid less than nurse-managers who are on equal levels of the career track. This is especially true of those working in private physician's offices.

The introduction of the CDE comes at a critical period of diabetes health care in this country. It is now time to better use these talented health-care educators and specialists, because they are definitely here to stay as permanent and prominent members of the health-care team.

RUTH FARKAS-HIRSCH, MS, RN, CDE
IRL B. HIRSCH, MD

FROM THE UNIVERSITY OF WASHINGTON SCHOOL OF MEDICINE, SEATTLE, WASHINGTON.

ADDRESS CORRESPONDENCE TO RUTH FARKAS-HIRSCH, MS, RN, CDE, UNIVERSITY OF WASHINGTON MEDICAL CENTER, 1959 NE PACIFIC STREET, RC 99 SEATTLE, WA 98195.

CDE, CERTIFIED DIABETES EDUCATOR.

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Diabetic Ketoacidosis in Pregnancy

Another atypical case

I read with interest the report by Maislos et al. (1) describing an episode of DKA associated with intrauterine fetal death and urinary tract infection in a

multigravida woman with past history of macrosomia. She remained normoglycemic postpartum but then developed gestational diabetes in a subsequent pregnancy. After the latter pregnancy, she had normal plasma glucose off insulin for 18 mo. As suggested by the authors, it is likely that the infection led to DKA. Herein is a report of an atypical case of acute DKA in a normal pregnancy associated with intrauterine fetal death, followed by rapid normalization of glucose tolerance.

A 34-yr-old black woman, gravida 3, para 2, presented at 20 wk of pregnancy because of vaginal bleeding and ruptured membranes. She also noted polyuria, polydipsia, polyphagia, and nocturia of several days duration. She received no prenatal care until 3 wk before this admission when a plasma glucose 1-h postoral 50-g glucose load was 6.6 mM. Her past history was significant for one normal spontaneous vaginal delivery and one cesarean section for fetal distress. She had no previous history of diabetes or macrosomia. Laboratory findings included: FBG, 17.8 mM; Na^+, 126 mM; K^+, 5.4 mM; Cl^-, 93 mM; CO_2, 3 mM; BUN, 3.9 mM; and plasma ketones 3+ undiluted. Arterial blood gases showed pH 7.10, PO_2 148 mmHg, and PCO_2 9.2 mmHg. Blood and urine toxicology were negative. No evidence of infection was seen.

DKA was treated with fluids and intravenous insulin. Later, dead fetus and placenta were delivered. Glycemic response was quick, and 5 days later insulin was discontinued. FBG was 5.3 mM and 2-h postprandial was 10.8 mM. She then was referred to the Diabetes Care Center where she weighed 178.5 lb, at a height of 5′ 2.5″, and her blood glucose profile was FBG, 5.9; prelunch, 4.6; 1500, 6.0; h.s., 4.4 mM. A month later, while on no medications, she had normal lipids, normal thyroid function tests, negative islet cell and thyroid antibodies, and the results of her 75-g oral glucose tolerance test are shown in Table 1.

Table 1—*Results of 75-g oral glucose tolerance test*

Min	Blood glucose (mM)	C-peptide (nM)
0	5.1	0.66
30	6.5	1.19
60	6.8	1.48
120	7.2	1.82

The patient remained euglycemic during the subsequent year postpartum and was then lost to follow-up.

This 20-wk pregnant woman had a normal diabetes screening 3 wk before presenting with DKA and a stillborn delivery, and subsequently became normoglycemic with elevated C-peptide levels. The cause of this atypical presentation is unknown, and the relationship between fetal death and this acute DKA remains unclear. The woman had no history of macrosomia. Atypical diabetes presentations have been described previously but not during pregnancy (2). The elevated C-peptide with normal blood glucose may indicate a baseline insulin resistance probably secondary to obesity. Superimposed on that, an unknown factor has precipitated the DKA in this seemingly normal pregnancy.

Nicolas N. Abourizk, MD

From The Diabetes Care Center, Department of Medicine, Saint Francis Hospital and Medical Center, Hartford; and The University of Connecticut School of Medicine Farmington, Connecticut.

Address correspondence to Nicolas Abourizk, MD, St. Francis Hospital and Medical Center, 114 Woodland Street, Hartford, CT 06105–1299.

DKA, diabetic ketoacidosis; FBG, fasting blood glucose.

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Renal Failure in Wolfram's Syndrome

Dr. Don J. Wolfram, who died in March 1991, was a fellow in medicine at the Mayo Foundation in 1936 when he reported the simultaneous occurrence of diabetes mellitus and optic atrophy in 4 of 8 siblings. No mention was made of deafness or any polyuria exceeding that expected with their diabetes (1).

Since 1936, Wolfram's syndrome, now often referred to as the DIDMOAD syndrome, has been expanded by the inclusion of diabetes insipidus, deafness, and neurological and genitourinary abnormalities, including atony of the bladder and higher urinary tracts (2). An autosomal recessive inheritance, not HLA linked as is the common form of IDDM, is presently considered the mode of inheritance of Wolfram's syndrome (3).

When Marquardt and Loriaux (4) reported 2 cases and reviewed the literature in 1974, they were able to identify 43 patients. They suggested the neurogenic bladder found in 6 patients, including their 2, was consequent to diabetes insipidus with resultant bladder atony, hydroureter, and reflux rather than diabetic neuropathy. One of Khardori's (5) 2 patients with bladder dysfunction since the age of 8 yr, two years after his diagnosis of diabetes, never had diabetes insipidus but showed unilateral dilatation of his right ureter and renal pelvis at the time of his death