retinopathy with cystoid macular edema, cotton-wool spots, and hemorrhages. Visual acuity was 20/30 (right eye) and 20/60 (left eye). She was treated initially with furosemide for the retinal edema, but visual acuity continued to deteriorate and was 20/60 (right eye) and 20/80 (left eye) at 19 wk gestation. Panretinal photocoagulation was performed with 2000 laser burns to each eye, and there was some improvement in visual acuity to 20/30 in both eyes by 27 wk gestation.

At 28 wk gestation, the patient developed a marked increase in peripheral edema associated with hypertension (160/110 mmHg) and progression of nephropathy. Blood pressure was controlled aggressively with antihypertensive agents, but there was a deterioration in her retinopathy with the development of extensive proliferative lesions, for which she received further laser treatment in the left eye at 30 wk gestation.

The patient gave birth to a healthy child at 33 wk gestation by lower-segment cesarean section, at which point visual acuity was 20/80 (right eye) and 20/60 (left eye). Urinary protein secretion was now 5.6 g/24 h, creatinine clearance 41 ml/min, plasma creatinine 152 M, and albumin 24 g/L.

Postpartum, her retinopathy progressed in both eyes with further neovascularization, fibrosis, and preretinal hemorrhages on the right eye despite additional photocoagulation. She received a total of 4000 laser burns to the left eye and 3800 laser burns to the right eye.

Eight months postpartum, the patient detached her right macula, necessitating vitreoretinal surgery. Her subsequent visual acuity was 20/80 (right eye) and 20/30 (left eye), and retinopathy was now quiescent. Metabolic control during and after pregnancy was excellent, with HbA_{1c} values all within the normal range.

This case history illustrates a type of severe retinopathy shown to regress postpartum in several studies (2–4). However, in this case, the retinopathy was only just contained by laser treatment during pregnancy, and in contrast to the expected course, it continued to deteriorate after delivery. Some researchers suggest a more judicious use of laser treatment in the pregnant woman with retinopathy and demonstrate a high rate of regression of the type of lesions seen in our patient toward the end of pregnancy and postpartum (3,4).

In 1982, Moloney and Drury (2) reported progressive changes in background retinopathy in pregnant diabetic women that completely regressed to control levels 6 mo after delivery. New lesions occurring in a group of women with proliferative retinopathy also regressed, although not back to control values. Particular mention was made of a marked increase of streak and blob hemorrhages and soft exudates that completely resolved in the postpartum period.

Ohrt (3) noted increased soft exudates, hemorrhages, and retinal edema in 23 of 100 pregnancies, all of which regressed postpartum. In contrast, although regression occurred in the cases of new proliferations, this was not nearly as complete.

The preliminary report from a prospective study in Denmark supports these findings (4). Retinopathy in women at the onset of pregnancy deteriorated in 50% of the cases, but postpartum regression was common. Also, the development of proliferations was uncommon and also regressed by 6 mo postpartum. It was suggested that treatment with photocoagulation during pregnancy be restricted and close ophthalmological supervision be undertaken by a person familiar with the spontaneous course of retinopathy during diabetic pregnancy.

Our case showed a poor response to photocoagulation during the pregnancy with further deterioration postpartum. Although evidence suggests that a more limited use of laser treatment in such cases might be a reasonable option, there are not yet any methods of predicting which patients will do well or means of selecting those who warrant more aggressive treatment. Until then, withholding laser treatment must be attendant with anxiety.

MEL CONWAY, MRCP
JANET BALDWIN, MRCOG
EVA M. KOHNER, FRCP
W. EDWARD SCHULENBURG, FRCS
JOSEPH CASSAR, MD

From the Department of Endocrinology and Medicine, West Middlesex University Hospital, Isleworth, Middlesex, United Kingdom.
Address correspondence and reprint requests to Dr. Joseph Cassar, West Middlesex University Hospital, Isleworth, Middlesex, TW7 6AF U.K.

REFERENCES

1. Cassar J, Kohner EM, Hamilton AM, Gordon H, Joplin GF: Diabetic retinopathy and pregnancy. *Diabetologia* 15:105–11, 1978
2. Moloney JB, Drury MI: The effect of pregnancy on the natural course of diabetic retinopathy. *Am J Ophthalmol* 93:745–56, 1982
3. Ohrt V: The influence of pregnancy on diabetic retinopathy with special regard to the reversible changes shown in 100 pregnancies. *Acta Ophthalmol* 62:603–16, 1984
4. Serup L: Influence of pregnancy on diabetic retinopathy. *Acta Endocrinol Suppl* 227:122–24, 1986

Hypoglycemia After Successful Pancreas Transplantation in Type I Diabetic Patients

One purported advantage of successful pancreas allograft transplantation over intensive insulin therapy in insulin-dependent (type I) diabetic patients is the achievement of physiological normoglycemia with consequent improvement in quality of life and potential stabilization of diabetic complications. Hypoglycemia, a very frequent occurrence in patients receiving an intensive insulin regimen, has not been reported after successful pancreas transplantation in type I diabetic recipients. We were therefore surprised to observe recurrent fasting and postprandial hypoglycemia (serum glucose <2.8 mM) in a subset of our pancreas transplant recipients. To examine this unexpected finding, we compared serum glucose, insulin, and C-peptide levels

before and after mixed-meal and intravenous glucose stimulation in five hypoglycemic patients with 10 nonhypoglycemic recipients of heterotopic, whole cadaveric pancreas-kidney transplants who demonstrated normal allograft function and insulin independence for at least 6 mo. The clinical characteristics of the hypoglycemic and nonhypoglycemic groups, including prednisone, azathioprine, and cyclosporine doses; mean ± SE age (38 ± 5 vs. 33 ± 2 yr); weight (59 ± 5 vs. 68 ± 7 kg); body mass index (22.6 ± 1.4 vs. 23.1 ± 1.5 kg/m^2); duration of diabetes (23 ± 3 vs. 21 ± 2 yr); and time posttransplantation (12 ± 6 vs. 6 ± 1 mo), were not significantly different. All patients, including the hypoglycemic group, had normal liver, renal, and cardiac function and normal nutritional status.

Mean ± SE serum glucose concentrations were significantly lower ($P < 0.05$) in the hypoglycemic versus nonhypoglycemic group at baseline and poststimulation nadir during all stimulation tests. Four of five hypoglycemic patients had fasting hypoglycemia, with glucose levels ranging from 2.2 to 3.1 mM. Hypoglycemia occurred post–mixed-meal stimulation in two of five patients, and after intravenous glucose administration in all five patients in the hypoglycemic group. Three patients were symptomatic, experiencing shakiness, dizziness, and sweating with serum glucose levels of 1.6–2.6 mM. The other two patients were asymptomatic despite glucose levels of 1.3–2.7 mM. None of the patients without hypoglycemia experienced any of these symptoms. Six months after the initial studies, all hypoglycemic patients continued to manifest low fasting (2.4–2.7 mM) and poststimulation nadir (1.3–2.7 mM) serum glucose levels. In contrast, serum insulin, C-peptide, and glucagon levels at baseline and after stimulation were not significantly different between the two groups. Baseline insulin was 115 ± 29 vs. 208 ± 43 pM in hypoglycemic versus nonhypoglycemic patients, whereas corresponding serum C-peptide was 1.1 ± 0.1 vs. 1.6 ± 0.2 nM, respectively. Post–mixed-meal and intravenous glucose stimulation, serum insulin, C-peptide, and glucagon concentrations were similar in the two groups. After intravenous glucose administration, the mean glucose disappearance rate (K_G) was not different between the hypoglycemic versus nonhypoglycemic groups (2.86 ± 1.00 vs. 2.77 ± 0.03%/min).

Our study demonstrated that, despite successful heterotopic, whole cadaveric pancreas transplantation, a subset of type I diabetic recipients are at risk for spontaneous and poststimulation hypoglycemia. Potential mechanisms that may contribute to hypoglycemia after pancreas transplantation include *1*) hyperinsulinemia, *2*) increased insulin sensitivity, or *3*) abnormal counterregulation. Hyperinsulinemia often occurs in heterotopic pancreas transplant recipients as a consequence of systemic venous drainage (1,2) and perhaps insulin resistance secondary to prednisone therapy (2–4). Although our heterotopic pancreas transplant recipients were hyperinsulinemic as expected, serum insulin and C-peptide profiles were similar in the hypoglycemic and nonhypoglycemic patients; thus, apparent differences in insulin secretion could not explain the occurrence of hypoglycemia in some of our patients. Regarding insulin sensitivity, Luzi et al. (5) reported that hepatic insulin sensitivity is normalized, whereas peripheral insulin sensitivity is moderately improved in type I diabetic allograft recipients. Because we observed no significant difference in incremental integrated serum insulin and glucose areas and K_G between hypoglycemic and nonhypoglycemic groups, we are tempted to conclude that no clinically significant difference in peripheral insulin sensitivity existed between the two groups in our study. Second, because our patients experienced fasting hypoglycemia, we can infer that the hypoglycemic group probably had an increased hepatic insulin sensitivity or reduced gluconeogenic fluxes to the liver. Finally, the hypoglycemia could also represent a manifestation of defective counterregulatory responses in our patients who had long-standing diabetic complications (6). However, further studies are necessary to elucidate the mechanism of hypoglycemia in pancreatic allograft recipients.

Hypoglycemia in type I diabetic patients who otherwise have successful pancreas transplantation has significant clinical implications in terms of cognitive impairment or even death. During the follow-up, we observed that the patients who developed hypoglycemia did so repeatedly, even to physiological mixed-meal ingestion over 1 yr posttransplantation. We conclude that hypoglycemia can occur in a subset of type I diabetic recipients after successful pancreas transplantation. Although the mechanism and scope of this problem are uncertain, it is important that physicians be aware of this potentially serious metabolic complication, which has not been previously addressed in the literature of heterotopic whole cadaveric pancreas allograft recipients.

DARYL A. COTTRELL, MD
MITCHELL L. HENRY, MD
THOMAS M. O'DORISIO, MD
RAYMOND J. TESI, MD
RONALD M. FERGUSON, MD
KWAME OSEI, MD

From the Departments of Medicine and Surgery, The Ohio State University Hospitals, Columbus, Ohio.

Address correspondence and reprint requests to Kwame Osei, MD, 485 McCampbell Hall, 1581 Dodd Drive, Columbus, OH 43210.

ACKNOWLEDGMENTS

Health Grant GCRC-RR-34.

This work was presented as an abstract at the National American Federation for Clinical Research meeting, Washington, DC, May 1990.

REFERENCES

1. Ostman J, Bolinder J, Gunnarsson R, Brattstrom C, Tyden G, Wahren J, Groth CG: Effects of pancreas transplantation

on metabolic and hormonal profiles in IDDM patients. *Diabetes* 38 (Suppl. 1):88–93, 1989
2. Diem P, Abid M, Redmon B, Sutherland D, Robertson RP: Systemic venous drainage of pancreas allograft as independent cause of hyperinsulinemia in type I diabetic recipients. *Diabetes* 39:534–40, 1990
3. Rizza R, Mandarino L, Genest J, Baker B, Gerich J: Production of insulin resistance by hyperinsulinaemia in man. *Diabetologia* 28:70–75, 1985
4. Robertson RP, Abid M, Sutherland D, Diem P: Glucose homeostasis and insulin secretion in human recipients of pancreas transplantation. *Diabetes* 38 (Suppl. 1):97–98, 1989
5. Luzi L, Secchi A, Facchini F, Battezzati A, Staudacher C, Spotti D, Castoldi R, Ferrari G, DiCarlo V, Pozza G: Reduction of insulin resistance by combined kidney-pancreas transplantation in type I (insulin-dependent) diabetic patients. *Diabetologia* 33:549–56, 1990
6. Amiel S, Tamborlane W, Simonson D, Sherwin R: Defective glucose counterregulation after strict glycemic control of insulin-dependent diabetes mellitus. *N Engl J Med* 316:1376–83, 1987

Role of Diabetologist in Evaluating Diabetic Retinopathy

Nathan et al. (1) compared the accuracy of ophthalmologists with diabetologists in screening for diabetic retinopathy with seven-field color stereoscopic fundus photography. Although we agree with the final conclusion of this study that well-trained diabetologists can make ophthalmology referral decisions, we found the methodology and some results worthy of remark.

Nathan et al. compared two different examination methods. The diabetologists used direct ophthalmoscopy through undilated pupils, and the opthalmologists used indirect ophthalmoscopy through dilated pupils with only occasional use of slit-lamp biomicroscopy. Indirect ophthalmoscopy gives a small magnification (20 dpt × 3) and is not sensitive enough to detect subtle diabetic lesions such as microaneurysms, small hemorrhages, and early new vessels. To screen effectively, it is therefore essential to perform direct ophthalmoscopy or slit-lamp biomicroscopy through a dilated pupil. The predominant use of indirect ophthalmoscopy in the study explains the poor results of the ophthalmologists. Moss et al. (2) showed that ophthalmologists using indirect supplemented with direct ophthalmoscopy concurred with seven-field color stereoscopic fundus photography 85.7% of the time, significantly better than in the study by Nathan et al.

We were disappointed by the decision of Nathan et al. not to dilate the pupil before direct ophthalmoscopy. Klein et al. (3) found this to be both an insensitive and nonspecific method. Even when pupillary dilation was used, Sussmann et al. (4) found a serious error rate of ~50% for diabetologists.

The impression from these studies is that if diabetologists make ophthalmology referral decisions for their patients, appropriate training is indispensable. If this is not guaranteed, regular detailed examination by an ophthalmologist is necessary.

JUTTA WIEK, MD
RICHARD NEWSOM, MBBS, BSc
EVA KOHNER, MD, FRCP

From the Diabetic Retinopathy Unit, Royal Postgraduate Medical School, London, United Kingdom.

Address correspondence and reprint requests to Jutta Wiek, MD, Diabetic Retinopathy Unit, Royal Postgraduate Medical School, Du Cane Road, London W12 OHS, UK.

REFERENCES

1. Nathan DM, Fogel HA, Godine JE, Lou PL, D'Amico DJ, Regan CDJ, Topping TM: Role of diabetologist in evaluating diabetic retinopathy. *Diabetes Care* 14:26–33, 1991
2. Moss SE, Klein R, Kessler SK, Richie KA: Comparison between ophthalmoscopy and fundus photography in determining severity of diabetic retinopathy. *Ophthalmology* 92:62–67, 1985
3. Klein R, Klein B, Neider MW, Hubbard LD, Meuer SM, Brothers RJ: Diabetic Retinopathy as detected using ophthalmoscopy, a nonmydriatic camera and a standard fundus camera. *Ophthalmology* 92:485–91, 1985
4. Sussman EJ, Tsiaras WG, Soper KA: Diagnosis of eye disease. *JAMA* 247:3231–34, 1982

Patient-Oriented Educational Material on Diabetes

In diabetes, much of the success of treatment depends on patient education. Therefore, educational material must be oriented toward the average patient's ability to understand sometimes complicated medical concepts. The following is offered as an example of **p**atient-**o**riented **e**ducational **m**aterial (POEM) that patients and professionals alike may find both comprehensive and comprehensible.

I

Diabetes has two forms,
(Exceptions can defeat the norms)
The one's acute and in a child,
The other initially seems mild,
Its onset in adults is stealthy,
Though the patient feels quite healthy,
Till the sugar in the blood
Has started up a urine flood
That courses in the night at first,
And then all day. A nagging thirst
So difficult to satisfy,
Can leave you feeling limp and dry.
Although there's sugar in excess,
The cells are starving nonetheless.
And so you eat a lot and still
You're hungry, weak, fatigued, and ill.
Infections such as furuncles