exanthemata, including chicken pox, were often followed temporally by a marked increase in the number of cases of juvenile diabetes.

I conclude with two apt quotations: "The more things change, the more they remain the same;" and "Those who forget history are doomed to repeat it."

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Blood Glucose Monitoring for the Visually Impaired Person

I would like to thank you for publishing the extremely informative article by Bernbaum et al. (1), on the "Effectiveness of Glucose Monitoring Systems Modified for the Visually Impaired" in your October 1993 issue.

Since its publication, however, two articles (2,3) have appeared in the literature that have successfully addressed the problem Bernbaum noted in the article—that one of the monitors lacked a way of applying "a sufficient blood sample to the reagent strip independently because the system lacked . . .landmarks for tactile feedback." Both of these articles have provided techniques that can overcome this problem and have been published under the auspices of the American Association of Diabetes Educators (AADE).

For more information on meeting the special needs of people with both diabetes and visual impairment, your readers are welcome to contact AADE's Visually Impaired Persons Specialty Practice Group at 444 North Michigan Avenue, Suite 1240, Chicago, IL 60611-3901 or call 1-800-338-3633.

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Intraperitoneal Insulin Administration Does Not Modify Plasminogen Activator Inhibitor 1 Levels in IDDM Patients

Plasminogen activator inhibitor 1 (PAI 1) is the main regulator of the fibrinolytic process, and elevated plasma level is considered a risk factor of atherothrombosis (1). In vitro, insulin stimulates PAI 1 synthesis by liver cells (2). In vivo, PAI 1 levels are strongly correlated with fasting insulin values, and obese and non-insulin-dependent diabetes mellitus patients with endogenous hyperinsulinemia have high PAI 1 values (3). However, despite peripheral hyperinsulinemia, insulin-dependent diabetes mellitus (IDDM) patients exhibit normal PAI 1 values (4). This phenomenon may be due to under-insulinization of the liver by the subcutaneous route of insulin administration. Insulin administered intraperitoneally, using implantable pumps, is almost entirely absorbed into the portal circulation (5), reproducing the physiological concentration gradient between portal and peripheral circulation. Such a route of administration, by increasing portal insulin concentration, could eventually have an unfavorable effect on PAI 1 levels.

We studied 11 IDDM patients (six men) with a mean age of 34.4 years (range 21–48), a mean duration of diabetes of 22.3 years (range 5–31), and a normal body weight (body mass index [BMI]: 22.7 ± 0.3 kg/m^2; mean ± SE) who were well controlled by means of a continuous subcutaneous insulin infusion (CSII) for at least 3 months. Then they were equipped with an implantable programmable insulin infusion pump (Infusaid model 1000, $n = 6$, Minimed model MIP 2001, $n = 5$) delivering insulin intraperitoneally. Blood samples were obtained before and 1, 3, and 10 months after implantation at 0800 after a 12-h fast while the patients received only the basal insulin rate from the pump. Twenty age-matched subjects served as a control group.

As shown on Table 1, diabetes control estimated by means of monthly blood glucose (4 or 5 daily measurements) and HbA_{1c} (Bio-Rad, Hercules, CA) was fair on CSII and did not change after switching the route. Lipid parameters remained in the normal range. Insulin requirements and BMI did not change. Fasting plasma free in-

Table 1—*Results for the patients treated by subcutaneous, then intraperitoneal insulin, and control group*

	Subcutaneous insulin	Intraperitoneal insulin			Control group
	Month 0	Month 1	Month 3	Month 10	
PAI 1 activity (U/ml)	5.1 ± 0.8	8.1 ± 1.9	4 ± 1.1	6.6 ± 1.3	9.76 ± 1.2
Free insulin (mU/l)	17 ± 3.9	16 ± 2.7	16.5 ± 2.1	14.4 ± 3.1	8.1 ± 0.66
Mean monthly blood glucose (mg/dl)	151 ± 9.3	142 ± 7.8	151 ± 7.8	146 ± 5.5	—
HbA_{1c} (%)	6.9 ± 0.3	6.5 ± 0.3	6.8 ± 0.3	6.3 ± 0.3	5.5 ± 0.2
Plasma triglycerides (mM)	0.83 ± 0.10	0.85 ± 0.09	0.88 ± 0.08	0.83 ± 0.10	1.03 ± 0.04
Plasma cholesterol (mM)	5.03 ± 0.38	4.95 ± 0.42	4.74 ± 0.38	4.92 ± 0.69	5.30 ± 0.2
BMI (kg/m^2)	22.7 ± 0.3	22.7 ± 0.3	22.7 ± 0.3	22.7 ± 0.3	22 ± 0.5
Insulin requirements (U/day)	40.5 ± 4.4	40 ± 3.4	41.6 ± 3.9	43.8 ± 4.8	—

Data are means ± SE.

sulin levels (CIS Bio Industries, Gif-Sur-Yvette, France) were also unchanged. This apparent paradox is in accordance with previously published studies showing that patients on long-term intraperitoneal insulin administration have lower integrated insulin concentrations but fasting values similar to those observed with the subcutaneous route (6). PAI 1 (Biopool, Umea, Sweden) levels were in the low normal range (0–17 U/ml) and lower than those of the control group ($P < 0.05$), but, in fact, values in these control subjects were slightly higher than those we have published (4). PAI 1 levels were not modified 1 to 10 months after switching the route of insulin administration.

These negative results do not contribute to the understanding of the relationship between insulin and PAI 1 secretion. In fact, there are several candidates for stimulating PAI 1 secretion in vivo, which are insulin itself, proinsulin, very-low-density lipoprotein, and unidentified factors associated with insulin resistance (7). These results do not support the hypothesis of a direct role of insulin on PAI 1 secretion by the liver, but they are important in practice because they show that intraperitoneal insulin administration by implantable insulin infusion devices in IDDM patients, providing good metabolic control and quality of life, does not have an unfavorable effect on this risk factor of atherothrombosis, as was feared.

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Alteration of the Risk Factor Paradigm for Discontinuance of Insulin Pump Therapy

In a recent paper in *Diabetes Care*, Floyd et al. (1) reported a discontinuation rate of insulin pump therapy of 49% after an average of 9.9 months of treat-