There Should Be a National American Diabetes Association Program for Diabetes Screening

The two articles in the May 1994 issue of *Diabetes Care* about screening for diabetes were excellent and very appropriate (1, 2). A national program for screening is long overdue.

Why is there no national program? The background is very unfortunate in my mind. Many affiliates were carrying out varied screening programs in the 1960s and 70s, resulting in a quasi-national program, albeit disorganized and diverse. Some of the programs were good; others not so. Certain members of the American Diabetes Association (ADA) evaluated the poor programs and on this basis, in the early 80s, bitterly denounced all screening on two bases: *1*) there were too many false-positives, which generated unwarranted fear in those tested, and 2) the yield was too low to justify the man power and expenditures required. (This was because some affiliates screened indiscriminately rather than emphasizing high-risk groups.)

Considering the present "state of the art" compared with that of over 30 years ago, I feel that our efforts (3) indicate that good screening was being carried out in the past.

There should be a national program for screening, and it is long overdue. But where would the man power come from? The program could be carried out at the local level by ADA chapters, provided that four criteria are met: *1*) there must be standard procedures and well-defined glucose values (the earlier work by Harris et al. [4] in 1979 is a milestone in this respect); 2) adequate professional supervision must administer and oversee the program; 3) compilation of results and conclusions should be carried out by a coordinating body of the national ADA; and 4) the program should be under the able guidance of Harris and Modan.

The plea by Harris and Modan (1, 2) for a national ADA screening program should be followed up.

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Osler and His Students

The "sweet pea" episode

In the introduction to his book *A Year With Osler*, Joseph H. Pratt, who relates his experiences with William Osler during the 1896–1897 academic year, alludes briefly to the fact that the latter's students once wore sweet peas to a diabetes lecture (1). Unfortunately, Pratt's few words fail to convey the lighthearted collegiality that made this brief episode so memorable. The reader may even wonder why he mentions it at all.

Luckily, however, Dr. Pratt's is not the only extant description of the "sweet pea episode." In a recently discovered letter written in 1952, Dr. Richard Rand, a 1900 graduate of Johns Hopkins who went on to serve as house officer there (2), relates the same story to a friend. We learn from Rand's account that the final laugh was not on Osler, but on the chairman of surgery, William Halsted:

> [The lecture] was held in the surgical amphitheatre—all the class was there, and all wore sweet peas. Dr. Osler came in, on time, looked around, and had a good laugh.
>
> Just then Dr. Halsted drifted in, looked around, and this colloquoy [sic] followed:
>
> Dr. Halsted: "What's going on Osler?"
>
> Dr. Osler: "We are having a symposium on diabetes."
>
> Dr. Halsted: "Why all the flowers?"
>
> General laughter—exit Dr. Halsted.
>
> I know because it was my class; so I remember the flowers—but not much of diabetes! (R. Rand, unpublished letter, 16 June 1952)

This little story held such tremendous personal meaning for Rand that he remembered it with fondness over 50 years later. Perhaps the fact that the rather taciturn Halsted was the butt of the joke made it especially memorable.

The "sweet pea episode" provides a glimpse at the early days of the Johns Hopkins Medical School and the mutual love and respect that pupil and teacher held for each other. No doubt Osler, as Chairman of Medicine, set the tone for this camaraderie (3), rare, then as it is now. Would that we, as diabetes educators, conveyed an atmosphere in our own institutions equally as jovial!

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Lipoprotein(a) Levels in Type II Diabetic Patients Are Not Influenced by Metabolic Control or by Microalbuminuria

Lipoprotein(a) [Lp(a)] is a cardiovascular risk factor in the general population, but its role, as such, in diabetic patients has yet to be established. Microalbuminuria is a risk factor for cardiovascular diseases in type II diabetic patients, but the mechanism(s) accounting for this association is as yet, largely unknown. In microalbuminuric type II diabetic patients, the Lp(a) levels have been reported normal (1,2) or increased (3). Conflicting results have been reported about the relation between Lp(a) levels and the degree of metabolic control in type II diabetic patients (4–7).

We examined a group of 71 consecutive type II diabetic patients and a group of 20 (8 men, 12 women) age-matched nondiabetic control subjects to establish whether Lp(a) levels are related to the presence of microalbuminuria (albumin excretion rate [AER] between 20 and 200 μg/min) or to the degree of metabolic control. Diabetic patients without microalbuminuria were 19 men and 28 women; 26 treated with diet only and 21 with diet plus oral agents. Diabetic patients with microalbuminuria were 11 men and 13 women; 9 treated with diet only and 15 with diet plus oral agents. None of the diabetic patients or control subjects was on hormonal or insulin treatment.

Exclusion criteria were antihypertensive treatment with drugs other than angiotensin-converting enzyme inhibitors or calcium antagonists; treatment with β-blockers, diuretics, or hypolipidemic drugs; recent history of vascular events; acute or chronic systemic diseases; and AER >200 μg/min.

Lipoprotein(a) was evaluated by an enzyme-linked immunosorbent assay method (8), HbA_{1c} by high pressure liquid chromatography, and urinary albumin by radioimmunoassay at least three times over a period of a week in 24-h urine collections. Comparisons among groups were performed with the Kruskall-Wallis test of variance, the Mann-Whitney (*U*) test, or the analysis of variance and Student's *t* test when appropriate. Pearson's correlation coefficient was used for correlation analysis. Triglyceride, urinary albumin excretion, and Lp(a) values were logarithmically transformed before inclusion in this analysis.

Lp(a) levels are not significantly different between control subjects (median 13, range 2–59 mg/dl) and diabetic patients with (9, 1–51) or without (12, 2–32) microalbuminuria. Analysis of correlation shows that among diabetic patients, Lp(a) levels are not related to plasma lipid levels, AER GHb, or duration of diabetes.

The frequency distribution of Lp(a) isoforms in diabetic patients is the same as in the nondiabetic population (9). Since serum Lp(a) levels are largely (>90%) genetically determined by Lp(a) isoforms (10), it would seem that in diabetic patients, Lp(a) levels should not differ from those in nondiabetic subjects because of a genetic component.

In our study, Lp(a) levels are not significantly affected by the presence of microalbuminuria and are not related to the degree of metabolic control. Our observations are cross-sectional, and we cannot establish whether modifications of the AER or of the degree of metabolic control may affect Lp(a) levels.

Since high Lp(a) levels are an independent vascular risk factor in the general population, it seems important to establish the role of Lp(a) as a vascular risk factor in diabetic patients and to identify possible relations among Lp(a) levels and microalbuminuria or metabolic control. In type II diabetic patients, microalbuminuria and a poor metabolic control are associated with an increased risk of vascular complications, but from our study, this association does not seem to be mediated by increased Lp(a) levels.

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