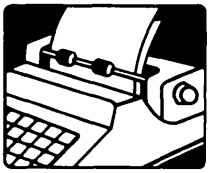
Editorial



A Parting Word . . .

In June 1976, Don Etzwiler, then assuming duties as President of the American Diabetes Association, offered me the challenging assignment of developing a new, as-yet-unnamed journal for the ADA, to focus on clinical advances in diabetes. With some trepidation, and against the advice of several close colleagues and friends, I opted to accept the opportunity offered me. The prepublication days were aided by the efforts of a great many people, most notably Caroline Stevens, Leonard Elliott, and Jack Dugan of the ADA staff; Frank Williams and his Publications Committee; Bob Kroc and Don Whedon who had the conviction to invest Kroc Foundation and NIH funds that provided fiscal resources to launch such a publication; and Don Etzwiler, Dan Mintz, Mort Bogdonoff, and Sallie Muller who provided advice, encouragement, and ideas on how a journal should be begun. By January 1978, our 19-month gestation period had ended, and DIABETES CARE was born.

Our readers must judge for themselves whether or not DIABETES CARE meets their needs and whether it has been a success. By conventional publication criteria, e.g., circulation growth, advertising support, manuscript submission rate, manuscript rejection rate, etc., we have succeeded. I suspect, however, that the real reason for any success that DIABETES CARE has enjoyed lies in the fact that 1978 marked the beginning of a new modern era in diabetes, and that DIABETES CARE was at the right place at the right time to chronicle that era.

Consider the advances that have occurred in diabetes since the inception of this journal. Understanding of the etiology and pathogenesis of diabetes has improved such that it has been possible to divide the disease into two entities—type I (IDDM) and type II (NIDDM). Universal diagnostic criteria have been adopted. Glycosylated hemoglobin has become a useful clinical test to document glycemic control, and nonenzymatic glycosylation has emerged as a potential mechanism to explain how hyperglycemia directly can inflict damage on a multitude of tissues and biologic processes. Patient self-monitoring of blood glucose, insulin infusion pumps, and intensive conventional therapy have markedly altered the treatment of type I diabetes. Glucose sensor-controlled automated infusion systems and continuous glucose monitoring systems have permitted a better understanding of glucose-insulin relationships.

An outpouring of new information has brought to clinical awareness many of the psychosocial problems encountered by diabetic patients, and new clinical strategies have emerged to deal with these. The birth of recombinant DNA technology has resulted in a clinically useful product—human insulin. And so much data have emerged on the relationship between the metabolic aberrations of diabetes and the microvascular complications that the controversy surrounding this issue has ended. Meanwhile, the controversy concerning oral agents, engendered by the UGDP study, has also waned with the development of a better understanding of type II diabetes, including both receptor and postreceptor defects, as well as relative insulin deficiency. The retardation of glucose absorption from the gut, via dietary fiber or pharmacologic agents, has altered dietary practice. The management of diabetes in pregnancy has been revolutionized. The Diabetic Retinopathy Study has established specific high-risk characteristics for laser photocoagulation. And the list goes on. Clearly, any book written about diabetes prior to 1978 is obsolete, as that year marked the beginning of a new era of diabetes management.

Parallel advances have been made in the basic sciences with regard to diabetes. The mechanism of insulin action has become more clearly understood, via the identification of the subunits of the insulin receptor, insulin mediators, and glucose transporters that can be recruited from cytoplasm to cell membrane. Islet microanatomy and paracrine functions have become better understood. The insulin gene has been localized to the short arm of chromosome 11, its nucleotide sequence identified, and nucleotide insertions flanking the insulin gene have been suggested as possible control points that may be altered in individuals predisposed to type II diabetes. Islet cell surface antibodies and cytotoxic antibodies, and other observations of the immune system, have emerged as important in the genesis of type I diabetes. Here, too, the list goes on.

What is responsible for this veritable explosion of knowledge and understanding of diabetes that has resulted in a complete change in our treatment of this disease? I believe that credit can be given to the National Diabetes Commission of 1975 and the National Diabetes Act of 1976. The concrete changes seen in diabetes are a direct result of the vastly expanded national research effort that followed as a consequence of the National Diabetes Act. The development of DIABETES CARE has facilitated the scientific advances being rapidly translated into clinical practice.

Particularly in our current situation of difficult financial times, our governmental leaders must be made aware of the magnitude of change in the science of diabetes that has occurred over the last several years as a consequence of earlier legislative wisdom. Now is the time to renew those efforts and assure that diabetes does not become a victim of shortsighted fiscal restraint.

As I retire from my duties as Editor of DIABETES CARE, I have many emotions. I am happy with the progress of this child, which has served to record the excitement and the enormous progress of the last 5 years. I am somewhat fearful to see that child grow up and leave home, to face the world at a time when progress could be slowed unless expanded resources are made available to fund diabetes research. I am optimistic that we can generate those resources and eventually make the need for DIABETES CARE obsolete by wiping out this disease. I am grateful to all those authors who entrusted their manuscripts to us. I am sympathetic to the frustrations of authors whose papers did not pass our editorial scrutiny and were not published. I am humbled by the awesome responsibility to make decisions (acceptance or rejection) concerning the product of the work of a fellow scientist/scholar. I am appreciative that so many others shared that burden in their capacity as peer reviewers. I am indebted to the staff of DIABETES CARE that makes things happen—Roberta Fineman, Maureen Clark, Rob Dinsmoor, Dorothy Segal, Martha Puente, and Caroline Stevens, as well as their predecessors who were with us at various steps along the way. I am thankful for having had the magnificent opportunity to launch this journal.

It is a wise policy that precludes an Editor from serving an ADA journal more than five years. One grows weary and rusty. New Editors bring fresh looks, new ideas, and undaunted enthusiasm. I could easily have been tempted to stay on, if such were permitted. For me, DIABETES CARE has been a labor of love. Yet, there is only so much one individual can contribute to any particular effort. I leave now to face new challenges, new opportunities. Yet, I feel confident that DIABETES CARE has grown to the point that its survival is assured. So, I bid you all farewell. And many thanks for putting up with me.

Adieu.

JSS