Response to Meriden

We are writing in response to the letter from Terry Meriden, MD, FACP, regarding the position taken by the American Diabetes Association during the health care debate of the past 2 years. Dr. Meriden faults the Association for having taken a position on health care reform that, he asserts, "specifically endorsed the Clinton plan."

This principal assertion of Dr. Meriden, that the American Diabetes Association specifically endorsed all components of the Clinton health care plan, is simply incorrect. While it is true that much discussion took place about whether the Association should or should not endorse the specifics of the Clinton plan, in the end no such endorsement was made. The Principles on Health Care Reform, developed by the Association and endorsed by a number of other health care organizations involved with diabetes, were principles that the Association believed had to be incorporated in *any* health care reform package.

Let us be clear. We will continue to be advocates for the enactment of these principles, which would seek to ensure universal access to quality diabetes treatment; prohibit pre-existing condition exclusions; provide coverage for prescription drugs and insulin, as well as for diabetes-related supplies, equipment and education; and achieve a mandate for community rating. As with other issues, at times our advocacy will involve asking our constituents to promote the passage of a particular piece of legislation that provides significant benefits for people with diabetes.

Dr. Meriden also faults the Association for making "a political decision." In the overheated atmosphere of last year's health care debate, virtually any decision (including a decision not to take a position) could be labelled "political." To avoid taking a position because an issue is "too sensitive" and may have "negative consequences" because of changes in Washington's "political winds," as Dr. Meriden suggests, is no less "political" and far more calculating.

Finally, the mission of the Association does include, as Dr. Meriden asserts, finding a prevention and cure for diabetes. These goals correctly require our strong support for continued diabetes-related research. But that is only half of our mission. The other half is to improve the lives of people with diabetes. This requires us to be advocates for better health care delivery, more comprehensive insurance coverage, and for bringing an end to discriminatory laws and policies based on outdated information.

The Principles on Health Care Reform reflected precisely where the Association should have been during last year's debate. To have remained silent during this debate would have been to abdicate our responsibility to be advocates for people with diabetes.

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Elevation and Fluctuation of Serum CA19-9 and SPAN-1 Levels After Administration of Sulfonylurea in a Diabetic Patient With Lewis(a−,b−) Blood Phenotype

It has been reported that poorly controlled diabetic subjects often show elevated serum CA19-9 levels in spite of there being no evidence of malignant diseases or other pathological conditions (1,2). However, Lewis (Le) blood phenotype is an important factor because type I carbohydrate antigens have the same structure as sialyated Lewis antigens (3). When serum CA19-9 concentrations are measured in diabetic subjects, the levels in Le(a+,b−) subjects are usually higher than those in Le(a−,b+) subjects (2,4,5). On the other hand, Le(a−,b−) subjects usually show normal levels of CA19-9, even when they have malignant tumors (6). In this report, we describe a rare case of a diabetic patient with Le(a−,b−) blood phenotype whose serum CA19-9 and SPAN-1 levels were markedly elevated and fluctuated after the administration of sulfonylurea.

HISTORY AND EXAMINATION — A 60-year-old woman with a 3-year history of NIDDM was referred to our hospital in June 1991. In January 1991, her glycemic control became aggravated and she was treated with insulin. After glycemic control was restored with daily administration of 12 U human NPH insulin and urinary excretion of C-peptide returned to normal (61 μg/day), insulin therapy was discontinued. This occurred 3 months before her visit to our hospital. Her height was 144 cm, body weight was 55 kg, and body mass index was 26.5 kg/m^2. No abnormal findings were recorded upon physical examination. Optic fundi were normal and peripheral neuropathy was not noted.

INVESTIGATIONS — Laboratory data showed a fasting plasma glucose (FPG) level of 102 mg/dl and HbA_{1c} of 5.3%. The serum CA19-9 level was elevated at 350 U/ml, but the serum carcinoembryonic antigen (CEA) level was 2.4 ng/ml. The serum amylase level was normal at 94 U. Liver function test, electrolytes, blood urea nitrogen, creatinine, and