Table 1. *Joslin Clinic Standard for Blood Glucose Control in Diabetic Patients Treated with Oral Hypoglycemic Compounds: Standards of Control* and Degree of Control†*

RELATION TO FOOD	GOOD		FAIR		POOR
	Blood Sugar‡ (mg./100 ml.)	*Urine Sugar (per cent)*	*Blood Sugar‡ (mg./100 ml.)*	*Urine Sugar (per cent)*	
Fasting	110	Trace	130	0.1	All
1 hr. p.c.	150	0.3	180	0.5	other
2 hr. p.c.	130	0.1	150	0.3	cases
3 hr. p.c.	110	Trace	130	0.1	

*For purpose of classification as to degree of control 70 per cent or more of values must conform with standards listed in the table.
†These standard values are the highest acceptable.
‡Glucose as determined by the Somogyi-Nelson procedure.

more conclusive than the data of 20 years ago. Meanwhile the practicing physician is busily and assuredly attempting to lower blood cholesterol levels, but is not certain how assiduously to work toward lowering blood glucose levels, and if so, how much.

A major concern has been the possibility that these oral agents might produce or allow the earlier development of the following: (1) islet cell failure with consequent decompensation of endogenous insulin function and greater activity of the diabetes; (2) infections; (3) neuropathy; (4) cataracts; (5) onset or more rapid progression of microangiopathy; and (6) greater morbidity and/or mortality from accelerated macroangiopathy, particularly that involving the coronary, cerebral, and peripheral vasculature. Because no good evidence had ever been presented to suggest that oral hypoglycemic agents were "antidiabetic," which would mean that they were inherently capable of delaying or preventing these more serious problems, the only reasonable benefit to be expected would be consequent to a net increase in the effectiveness of endogenous insulin, as indicated by lowering of the blood glucose level over and above that obtainable with diet. Thus, if significant lowering of blood glucose levels could not be attained and then maintained, there would be no basis for using any of the currently available oral hypoglycemic agents. The extent to which oral hypoglycemic agents have succeeded or failed with regard to these potential problems will be briefly reviewed.

Islet Cell Function

That sulfonylureas lower blood glucose levels primarily by increasing insulin secretion from the pancreas has been definitely proved.[6] However, it has long been contended by some investigators that extrapancreatic effects of sulfonylureas, particularly those related to hepatic glucose release, contribute to the effects upon glucose.[6] Recently considerable data have confirmed extrapancreatic actions, which are demonstrable in the absence of insulin at cellular sites and with con-