of uncontrolled diabetes, but metabolic control is well accepted as a fundamental part of the preventive program in avoiding fungal infection and active tuberculosis, as well as bacterial infection. Perhaps the most common and easily demonstrable example clinically is the persistence of Candida infections producing vulvovaginitis in the female and balanitis in the male, responding poorly to specific therapy such as nystatin or gentian violet but dramatically improving with the cessation of or marked improvement in excessive glycosuria and hyperglycemia. Such improvement is readily shown to occur in the diabetic patient responsive to oral hypoglycemic therapy, with rapidity of improvement comparable to that obtained with insulin. Similar responses can be obtained in certain patients with nearly normal endogenous insulin reserve upon application of diet and, of course, with the administration of insulin.

To date there has been no indication in patients responsive to oral hypoglycemic agents, when properly combined with reasonable dietary adherence, that new infection or aggravation of existing infection has occurred as a result of the use of oral rather than insulin therapy.

### Neuropathy

No controlled studies have proved that metabolic control prevents the development of neuropathy. However, neuropathy, particularly the more severe types such as amyotrophy, anesthetic feet, Charcot joints, and so forth, classically develops in the adult who seems to have had a short duration of diabetes, but who was unknowingly hyperglycemic for a period of time, or in the patient with known diabetes of longer duration in whom therapy was inappropriate or inadequate. Although diabetic patients may at times have an exacerbation of symptoms due to neuropathy following treatment of any kind, and although on occasion hypoglycemia may itself induce neuropathy, the *usual* clinical observation is that following improved metabolic control by whatever means, many manifestations of neuropathy improve sooner or later.

Recent biochemical data demonstrating the presence of the polyol pathway in nerve suggest a mechanism by which increased ambient glucose concentrations in the Schwann cell activate the formation of sorbitol,[14] so that nerve function may be compromised. To date the critical circulating blood glucose levels for activation of this pathway have not been clearly demonstrated, but its possible major metabolic role in the production of neuropathy provides further evidence for the desirability of keeping blood glucose levels as close to normal as is readily obtainable.

### Cataracts

At least two morphologic types of cataract occur in diabetes. These are: (1) the snowflake, flocculent, or metabolic cataract, occurring mainly in juveniles with grossly uncontrolled diabetes; and (2) the senile cataract due to sclerosis of the lens nucleus, indistinguishable from that seen in the nondiabetic and the commonest type observed in adult diabetic patients. The more rapid maturation of senile cataract in the diabetic than in the nondiabetic has recently been re-