netite, singly or in combination (that is, fractional crystallization), cannot account for the observed chemical changes in the lava series. The compositions assumed for the isomorphous minerals include generous ranges on either side of those inferred from optical measurements, yet the graphical tests show that no reasonable combination of added or subtracted representatives of the intratelluric minerals could yield oxide trends satisfactorily near those of the lava series itself. When, however, xenolithic material is included as one of the variables in the graphical tests with olivine and plagioclase, a remarkably good conformance of test trends to actual trends is obtained, and it is concluded that such a reasonable combination of bulk assimilation and fractional crystallization could have produced the chemical characters of the series.

Energy from the heat of crystallization of the particular mass of magma being contaminated would be entirely inadequate to accomplish the amount of assimilation implied by the graphical results. But an abundant supply of heat would be furnished by incremental crystallization of a magma that was circulating by thermal convection in a cupola above a major basaltic magma body, as suggested by S. Holmes [*Geol. Mag.* 68, 241 (1931)] and, in fact, several lines of evidence make it appear likely that the roots of the young volcanoes of the Paricutin region are cupolas rather than laccolithic masses. The Paricutin eruption is visualized as sampling the more contaminated magma of the upper part of a cupola through a tap that perhaps is offset somewhat from the apex of the cupola to provide a pressure-storage arrangement and account for the unusually steady and prolonged eruption.

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Six-Minute Responsiveness Test to Insulin

In 1935, H. P. Himsworth, of London, found that diabetic individuals could be classed as being either sensitive (responsive) to insulin or insensitive to the hormone. From a practical viewpoint, such classification would have greatly facilitated establishing a correct rationale concerning treatment in a given patient. Unfortunately, workers in the U.S.A. and Canada were unable to confirm Himsworth's findings, presumably, it now appears, because unlike European commercial insulin, the insulins available in this hemisphere are not free of the hyperglycemic-glycogenolytic "glucagon" factor. The presence of glucagon seriously interferes with the efficiency of a responsiveness test to insulin.

Using a glucagon-free insulin, the writer and his group at The Brooklyn Hospital were able to substantiate the findings of Himsworth in 100 unselected diabetic patients. After a small dose (3 units) of such insulin by vein, blood-glucose determinations (venous) were made after 2, 4, and 6 min. The resulting glucose curves served to array most of these diabetic individuals in the two distinctive categories propounded by Himsworth. Moreover, serial curves during the course of treatment of the diabetes demonstrated the fact that the patients who were originally unresponsive or poorly responsive to the insulin could in many instances by adequate treatment of the diabetes be reverted to normal responsiveness to the hormone. The 6-min test, accordingly, furnishes a much better yardstick for clinical improvement in the patient than do casual blood-glucose determinations. Casual blood-glucose readings may actually have been reduced to normal by massive dosage of insulin from without rather than as a result of improvement in the individual's own intrinsic efficiency in utilizing insulin. The test adequately makes this differentiation.

An extended study in 50 nondiabetic persons suggests that a most useful aspect of the test will be that of indicating which members of a diabetic family are destined ultimately to become clinical diabetics. Some of the obese individuals in the study, in spite of normal glucose-tolerance tests and normal casual blood-glucose readings, registered in the 6-min test a lack of responsiveness to glucagon-free insulin. This resistance to insulin in certain obese human beings correlates rather well with Mayer's findings of extreme insulin resistance in hereditary obese mice. It is reasonable to conjecture that such persistent defective response to insulin must result in a decreased insulin-efficiency and an increased production-demand on the body for insulin. In the presence of the well-recognized supernormal demands for insulin production already imposed by the obesity alone, such strained production-demand would be expected ultimately to result in "bankruptcy" of the intrinsic insulin-producing mechanism.

The work of Bornstein, Evelyn Anderson, and others suggests that the obese individual who develops diabetes usually suffers predominantly from a "relative" insufficiency of insulin rather than from an intrinsic inability of the organism to produce insulin. This deficiency is relative to exorbitant demands for insulin which the body cannot adequately meet. Whereas many diabetics give an antecedent history of obesity, only strikingly few of the large number of nondiabetic obese individuals actually do develop diabetes, despite their well-established increased demand for insulin. It would, accordingly, be of clinical importance to learn just which of these nondiabetic obese individuals show defective inability to respond to insulin and this well before clinical diabetes develops. The 6-min test furnishes one clue in this direction.

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