Acuity Variations in Diabetes Mellitus

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The purpose of this paper is to briefly review the factors involved in acuity changes in diabetes mellitus and to report findings suggesting a new concept, namely, diurnal fluctuations in acuity that are not associated with hypoglycemic episodes.

Decreased acuity in diabetes may be due to:

A. Non-diabetic causes common to the general population

B. Factors secondary to diabetes:

1. refractive changes secondary to blood glucose changes
2. cataracts, juvenile or senile
3. diabetic retinopathy
4. hypoglycemia
5. diurnal fluctuations, probably due to blood glucose changes

A sudden onset of myopia should always arouse a suspicion of diabetes mellitus (2). The changes may be as great as two to three diopters or sufficient to change acuity from 20/20 to 20/200 or less. The most dramatic changes usually occur in undiagnosed diabetes and may cause serious diagnostic confusion as summarized by the editor's comment on D. M. Watkin's article (7), "Diabetes Mellitus in an Internist":

"Myopia was a presenting complaint only 10 days after a. . . normal postprandial blood sugar. The significance of the myopia was not appreciated by the ophthalmologist who prescribed glasses. The neurologist interpreted the leg cramps as a sign of multiple sclerosis, and the otolaryngologist somehow favored a diagnosis of brain tumor." Typically, the acute myopia develops during the onset of the disease and may progress over several months with intermittent exacerbations. Hypermetropia typically occurs during the treatment period and tends to clear over a period of three to four weeks (6). Myopia therefore results from increased blood sugar, and hypermetropia results from returning the blood glucose level toward normal. These refractive changes may occur suddenly but invariably resolve slowly over several weeks or months.

Theories to explain the refractive variations have included changes in the axial length of the globe, changes in the refractive index of the aqueous or vitreous, accommodative paresis, and accommodative spasm. Rosenstein showed that atropine cycloplegia does not change the refractive findings (quoted by Mattos), (4). That the axial length, or vitreous or aqueous alterations were not involved was proven by Elschnig who reported on myopia in a diabetic with unilateral aphakia (quoted by Mattos), (5). He found no refractive changes in the aphakic eye, thus excluding axial length, vitreous or aqueous changes as etiological factors. Duke-Elder first explained the lens alterations that occur. With hyperglycemia the osmotic pressure of the aqueous decreases and fluid infiltrates into the lens making it more spherical and resulting in myopia. With decreases in the blood sugar the flow of fluid is reversed.

The cataracts that occur in diabetes may be of the juvenile or snowflake type or typical senile. The snowflake cataracts appear suddenly in early uncontrolled juvenile diabetics and resolve with good diabetic control. The acuity changes with cataracts are slow, and when snowflake cataracts clear, the acuity improvement is slow.

Advanced diabetic retinopathy may not result in any decreased acuity, and conversely, minimal diabetic retinopathy involving the macula may seriously impair the vision. Decreased acuity in diabetic retinopathy may be due to macular edema, macular

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exudates, macular hemorrhages, gliotic masses overlying the macula, retinal detachment, vitreous hemorrhages, or a combination of these factors. Generally, there is no correlation between the severity of the retinopathy and the visual acuity. In any case there are no rapid fluctuations of acuity with diabetic retinopathy, even though the disease is characterized by remissions and exacerbations in its early stages.

Acuity decreases secondary to hypoglycemia are usually associated with dizziness, diplopia, micropsia, macropsia, and central scotomas (1). The blood glucose is usually 60 mg% or less, and the disturbance may be secondary to the cerebral effects of hypoglycemia. The onset occurs suddenly and responds in minutes after the ingestion of glucose.

In following diabetics in the out-patient ophthalmology clinic, it was observed that some patients complained of decreased visual acuity at certain consistent times of the day, with a duration of 1 to 3 hours. Some stated they could not read in the morning. Others noted their acuity was decreased in the afternoon. A few stated they would not drive at certain times of the day because of visual difficulties. History and chart review did not reveal any correlation with poor control or hypoglycemic episodes. The following findings were tabulated from patients with this complaint of fluctuating acuity.

These findings were tabulated from approximately 150 diabetics questioned over a year-and-a-half period. Most of the patients had diabetic retinopathy of various stages. The incidence of confirmed fluctuating acuity was 8% or one in every 12 diabetics. Only those with an elicited history of daily acuity changes were examined for this finding. It is possible that others have this variation, but the change is not sufficient to be a complaint for their acuity needs. Being aware of the variation will also depend on whether the patient's activities such as reading, sewing, and so forth, require good acuity. The incidence might well be higher if all diabetics were examined for this finding regardless of their history. In some patients the second daily examination may not have coincided exactly with the time of greatest acuity deficit.

Seven of the patients had decreased afternoon acuity and five had decreased morning acuity. The change varied from one Snellen line to five lines in three patients with an average change of three lines. Seven of the patients wore refractive corrections. Since the pinhole acuity was in most instances the same as the best corrected acuity, it can be assumed that the change that occurs in these patients is in the lens. None of the patients could relieve symptoms by the ingestion of glucose, and none had associated symptoms of hypoglycemia such as dizziness, diplopia, micropsia, macropsia, or scotomas. Also, the symptoms usually lasted from 1 to 3 hours and sometimes occurred only a short time after a meal.

It is a well-documented fact that the acute refractive changes that occur in early diabetes are usually of weeks or months in duration and that the changes are secondary to fluid flow into or out of

	Age	D. D. Years	A. M. Acuity	P. H.	P. M. Acuity	P. H.	Diabetic Control
M. M.	62	10	20/20	20/20	20/40	20/20	good
N. L.	69	13	20/200	20/100	20/100	20/100	poor
H. F.	50	2	20/50	20/50	20/100	20/50	good
C. S.	59	21	20/100	20/50	20/50	20/40	good
F. G.	62	19	20/20	20/20	20/100	20/30	good
A. W.	52	8	20/100	20/80	20/30	20/30	good
S. V.	52	6	20/20	20/20	20/40	20/20	good
V. F.	56	7	20/30	20/30	20/50	20/30	fair
F. J.	65	1	O. D. CF	CF	CF	CF	good
			O. S. 20/20	20/20	20/50	20/20	
E. H.	67	9	20/50	20/50	20/100	20/50	good
L. B.	66	12	O. D. 20/200	20/200	20/200	20/200	good
			O. S. 20/50	20/30	20/30	20/30	
E. H.	69	23	O. D. HM	HM	HM	HM	good
			O. S. 20/100	20/30	20/30	20/30	

DD Disease duration
PH Pinhole acuity
CF Count fingers
HM Hand motion
Four patients in which this complaint was elicited failed to show any A. M. to P. M. variation.

the lens. No reports could be found in the literature referring to diurnal variations in acuity in well-controlled diabetics. The only exception found that was not associated with actual hypoglycemia was O. Lippmann's correspondence (3) on D. M. Watkin's article (7) in which he reports on myopic changes in a 38-year-old brittle diabetic: "Her myopia increased and decreased within minutes after blood sugar variations. She used the variation of her visual acuity as a substitute for a blood sugar test for many months. Her visual observations were confirmed several times by blood sugar tests." Even a well-controlled diabetic obviously does not have the same level of blood glucose throughout the day. The glucose levels rise after meals and may fall almost to hypoglycemic levels at other times of the day, producing a marked difference between high and low glucose levels, even though two-hour postprandial determinations are in a range classed as good control. It is conceivable that this acceptable variation between high and low levels could cause sufficient fluid shifts in the lens to produce diurnal variations in acuity. It was interesting that the patients in this series stated that the time of decreased acuity was consistent from day to day. The capsule of the lens in a normal healthy state maintains fairly stable molecular concentrations, and fluid changes are normally slow. When, however, the lens is repeatedly subjected by changing osmotic pressures to fluid shifts, it is reasonable that normal diffusion and active transport of fluid mechanisms are stressed and that fluid shifts could then occur more rapidly.

It is important that ophthalmologists be aware of this diurnal variation of acuity in diabetics and its probable incidence. If a patient is examined at a time of best acuity, no refractive correction would be recommended. Sugar ingestion to relieve symptoms might be inadvertently and erroneously advised. Conversely, if the patient is examined at a time of greatest acuity deficit, refractive correction would probably be prescribed, and a reversal of his diurnal variation from A.M. to P.M., or P.M. to A.M. would result. Examination by different ophthalmologists at different times of the day could result in markedly contradictory findings.

In summary, the factors involved in acuity changes in diabetics are briefly reviewed with emphasis on the rate of acuity variations. Twelve patients with a consistent history of diurnal acuity variations were examined, and an average variation of three Snellen lines was found. The incidence of this confirmed complaint was 8% of the number of patients questioned. The mechanism is assumed to be rapid fluid changes in the lens secondary to daily glucose fluctuations, in contradistinction to the slow fluid shifts causing myopia and hyperopia in early diabetics. It is postulated that the rapid changes occur due to damage of the normal lens mechanisms by repeated stress or insult inflicted by recurrent adjustments to osmotic pressure changes. The clinical importance of the entity is emphasized.

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