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# Refractive Error Changes in Diabetes Mellitus

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## INTRODUCTION

An estimated 1.8 million people in the United Kingdom have diabetes mellitus (DM), which reflects around 3% of the population (Watkins 2003). Statistics of the World Health Organisation suggest that there were around 171 million diabetic patients worldwide in 2000 (an estimated prevalence of 3%). It is predicted that this number will increase to around 366 million patients worldwide by the year 2030 (an estimated prevalence of 5.2%) (WHO 2004). These figures suggest that the number of diabetic patients (either diagnosed or undiagnosed) attending optometric practice is also likely to increase significantly over the next two decades. It has been well documented that fluctuations in refractive power often accompany changing blood glucose levels (Okamoto *et al.* 2000; Giusti 2003; Sonmez *et al.* 2005); however, the nature and the aetiology of refractive fluctuations in diabetes mellitus is poorly understood. The aim of this paper is to review the scientific literature relating to refractive error changes associated with diabetes and to discuss the implications of these changes for the optometrist.

Multiple organs or organ systems are affected by the disease process of diabetes mellitus. Some effects are acute (e.g. ketoacidosis, hypoglycaemia), and other effects are more chronic, taking a minimum of five years to develop (e.g. retinopathy). The chronic effects of diabetes on the retina are well known and are seen by ophthalmoscopic inspection and fluorescein angiography. The effects of diabetes on the anterior segment of the eye are less well reported in the literature but the effects of diabetes on the cornea and crystalline lens are thought to play a key role in the refractive error changes sometimes associated with the disease.

Investigators have observed both myopic (Giusti 2003) and hyperopic (Okamoto *et al.* 2000; Sonmez *et al.* 2005) refractive shifts in diabetic patients, which have been related to changing plasma glucose concentration. It would be helpful for the eye care practitioner to understand how blood glucose concentration could potentially affect the ocular parameters that contribute to the refractive power of the eye. If such relationships could be

established, then eye care practitioners would be better placed to advise patients on expected visual signs and symptoms during periods of altering diabetic metabolic control.

In the first part of this review, the effects of diabetes on the ocular structures that contribute to refractive power will be described. The second part of the paper reviews the scientific literature relating to refractive power shifts in diabetes. The third part considers the main hypotheses as to how myopic and hypermetropic shifts could occur with changing blood glucose concentration. The final part of the paper considers the implications for the optometrist in practice.

## **THE ANTERIOR OCULAR MANIFESTATIONS OF DIABETES**

### **Cornea**

#### *Curvature and thickness*

The cornea contributes approximately two-thirds of the refractive power of the human eye. Variations in curvature of either the anterior or the posterior corneal surface or changes in corneal thickness can alter the corneal refractive power.

Diabetes can lead to structural changes in corneal epithelial cells (Tsubota *et al.* 1991a; Tsubota *et al.* 1991b; Hosotani *et al.* 1995; McNamara *et al.* 1998) and endothelial cells (Schultz *et al.* 1984; Larsson *et al.* 1996). However, changes in central corneal curvature do not appear to be associated with altering blood glucose levels in diabetic patients (Saito *et al.* 1993; Okamoto *et al.* 2000). Saito *et al.* (1993) and Okamoto *et al.* (2000) investigated the degree of change in corneal curvature associated with the rapid correction of hyperglycemia. In a study of 28 eyes of 14 diabetic patients, Okamoto *et al.* found no change in corneal curvature with changing blood glucose concentration. Saito *et al.* investigated 10 eyes of 5 patients and came to similar conclusions.

Planten and co-workers carried out several studies to investigate the cause of refractive error changes in newly-diagnosed diabetic patients. Twenty-three patients had reported that they experienced fluctuations in their vision. The authors speculated that the refractive changes occurring in these patients was due to a change in the refractive index of the cornea or crystalline lens as previous studies had found no significant acute changes in lens curvature or position (Planten 1975; Planten *et al.* 1979; Planten 1981).

In several studies of corneal thickness in Type I diabetic patients, corneal thickness was found to be increased compared to that of age-matched controls (Busted *et al.* 1981; Pierro *et al.* 1993; Weston *et al.* 1995; Larsson *et al.* 1996). This is in contrast with the findings of other workers (Schultz *et al.* 1984; McNamara *et al.* 1998). Generally researchers have failed to demonstrate significant differences in corneal thickness between Type II diabetic patients and age-matched control subjects (Schultz *et al.* 1984; Larsson *et al.* 1996).

#### *Refractive index and corneal endothelial cells*

In the human eye, the refractive index of the corneal epithelium is 1.401, the underlying stroma is approximately 1.38 and the corneal endothelium is 1.372 (Patel *et al.* 1995). There are relatively few experimental measurements of human corneal refractive index described in the literature. These values tend to be based on histological and mathematical data (Fatt and Harris 1973; Laing *et al.* 1976). Nevertheless, corneal refractive index may be an important parameter to consider in diabetes, since the influx of metabolites (including glucose) from the aqueous humour may change the corneal refractive index significantly and thus affect corneal refractive power. Using the formula:

$$F_{\text{cornea}} = (n_{\text{cornea}} - 1) * r_{\text{cornea}}^{-1};$$

where  $F_{\text{cornea}}$  is corneal power,  $n_{\text{cornea}}$  is corneal refractive index, and  $r_{\text{cornea}}$  is the radius of curvature of the central cornea (Ellis *et al.*) and assuming Gullstrand constants, where  $n_{\text{cornea}} = 1.376$  and  $r_{\text{cornea}} = 7.7\text{mm}$ : a change of approximately 0.0075 in refractive index will induce a 1 D change in corneal

power. Additionally, a change in central corneal curvature of 0.2mm would give a change of approximately 1 D in corneal power.

Recent evidence linking elevated glucose concentration to reduced corneal endothelial Na<sup>+</sup>, K<sup>+</sup>-ATPase activity is of interest since this enzyme is an important component of the endothelial pump which is thought to be responsible for maintaining corneal hydration control (Whikehart *et al.* 1993). In light of this finding, it is perhaps not surprising that corneal thickness and time to recover from induced oedema are often increased in diabetic humans and animal models (Herse 1990a, 1990b). Oedema occurs when there is a significant reduction in the amount of oxygen reaching the cornea. The total corneal swelling induced by contact lens induced hypoxia has been shown to decrease with increasing blood glucose concentration in diabetic patients. (McNamara *et al.* 1998).

## **Crystalline lens**

### *Thickness*

Lens thickness is often greater in diabetic patients than in non-diabetic subjects, mainly due to cortical thickening (Huggert 1953; Brown and Hungerford 1982). Other biometric changes include steepening of the anterior and posterior surfaces of the lens and shallowing of the anterior chamber (Sparrow *et al.* 1990). After adjusting for age, these changes are more pronounced in Type I diabetic patients and in patients with increased disease duration. Age-related increases in axial lens thickness, cortical thickness and nuclear thickness are accelerated after the onset of diabetes (Sparrow *et al.* 1990). These findings could explain the increased prevalence of myopia found in diabetic patients. The expansion of the sagittal width of the lens is also accelerated in diabetic patients (Sparrow *et al.* 1990). Neither age nor disease duration appears to be responsible for these changes; however, the presence of diabetic retinopathy in Type 1 patients does seem to have an effect.

In the crystalline lens, refractive index has been found to be highest at the centre and lowest at the periphery of the lens (Gullstrand 1909; Smith 2003).

It may be that small changes in refractive index in the centre of the lens could induce an acute shift in refractive error in diabetic patients. We would expect a 3D change in refraction with a 0.02 change in refractive index (Planten 1981).

The size of the expected change in the crystalline lens in diabetes mellitus can be calculated with the help of Le Grand's theoretical eye (Wyszecki and Stiles 1982). To obtain a change in refraction of 2D with an anterior chamber depth of 3.1mm, the anterior surface of the lens would have to shift by 0.5mm, and change its radius from 10.2 to 14.1mm. Alternatively the entire lens would need to shift by approximately 1.5mm to obtain a 2D change in refraction. However, only a small change in refractive index (e.g. from 1.42 to 1.41) would be required for the same refractive change, making refractive error changes more likely to explain these shifts (Planten *et al.* 1979).

#### *Accommodation*

An aspect of ocular performance that seems to be affected in young diabetic patients is accommodation. The amplitude of accommodation in Type I patients is lower than that of age-matched control subjects (Moss *et al.* 1987; Mantyjarvi and Nousiainen 1988; Braun *et al.* 1995). It appears that even when there is no detectable retinal damage, the amplitude of accommodation in Type I patients is lower than that of age-matched healthy subjects. Furthermore, it appears that the latency of the response is unusually long (Braun *et al.* 1995).

#### *Cataract*

The intracellular accumulation of sorbitol (a by product of glucose metabolism) during poor metabolic control (hyperglycemia) will not only create an osmotic gradient leading to the uptake of water and lens swelling (Gabbay 1973), but will also cause a change in solubility (precipitation) of lens proteins which can lead to metabolic cataract formation (Kinoshita and Merola 1964; Caird *et al.* 1969; Ehrlich *et al.* 1987).

Cataract is more commonly found among diabetic patients, particularly in women and in patients with longer duration disease (Harding *et al.* 1993;

Rotimi *et al.* 2003; Hennis *et al.* 2004). Hypoglycemia induced by strict control of blood glucose in diabetic patients may induce cataract, since glucose is the major energy source of the lens (Vinding and Nielsen 1984). Cataract formation is associated with lens swelling, vacuole formation, and increased membrane permeability. Different forms of cataract in diabetic patients are juvenile cataract (opacity shaped like a snowflake, affecting the anterior and posterior cortical layer of the lens in young diabetics) (Nielsen and Vinding 1984) and age-related cataract (nuclear, cortical, and posterior subcapsular) cataract (Leske *et al.* 1991). Studies of young type I diabetic patients (with no detectable cataract on slit lamp) suggest that there is increased light scatter in diabetic patients that appears to correlate with HbA1c and the degree of diabetic retinopathy present (Kato *et al.* 2000; Kato *et al.* 2001).

### **Aqueous humour**

Patients with type 1 diabetes have reduced aqueous flow rates (Hayashi *et al.* 1989). This does not appear to occur in patients with type 2 diabetes (Larsson *et al.* 1995). One of the determinants of aqueous humour production is ocular blood flow (Bill 1975), which has been shown to increase with acute elevations in glucose concentration in type 1 diabetic patients (Bursell *et al.* 1996). Therefore, an increase of blood flow might be expected to induce an increase in aqueous flow. Lane and co-workers studied aqueous flow, ocular blood flow, and intra-ocular pressure in type 1 diabetic patients and control subjects, controlling for glucose and insulin concentration (Lane *et al.* 2001). They found a decrease in aqueous flow in type I patients versus fasting controls; however, aqueous production did not appear to be affected by insulin or glucose concentrations.

## **REFRACTIVE ERROR AND DIABETES**

### **Axial Length**

The results of a recent study suggested that axial length is reduced in diabetic patients compared with control subjects (Pierro *et al.* 1999). In this study axial length was negatively correlated with the degree of diabetic retinopathy (mean axial length was decreased in the subjects with background and proliferative



retinopathy compared to the group without retinopathy). No difference in axial length was found between the diabetic patients without retinopathy and the control subjects. Refractive power was not measured during this study.

### **Refractive error**

Since the 19th century, it has been recognised that changes in blood glucose concentration can influence vision in patients with diabetes (Da Costa 1890; Duke-Elder 1925). Optometrists are trained to consider the possibility of undiagnosed diabetes if a patient complains of a bilateral, unexpected or rapid change of vision or prescription. If diabetes is suspected, the eye care practitioner may postpone prescribing spectacles until the refractive error has stabilised, which generally occurs when the patient's diabetes is better controlled. Refractive changes associated with diabetes can be both acute (transient) and chronic (sustained). However, relatively little is known about the biochemical changes, which accompany these refractive events.

Chronic refractive changes reported in diabetic patients include an increase in myopia (Fledelius 1983, 1986, 1987; Mantyjarvi 1988). These refractive changes tend to be of a low magnitude and present during adulthood. One author studied the frequency of myopia among 1,416 Danish adults (including 381 adults with diabetes), and found a significantly higher prevalence of myopia among diabetic compared to non-diabetic subjects (38% in diabetic subjects versus 27.5% in non-diabetics) (Fledelius 1983). Myopia patterns (age of onset and degree) were investigated in another study, including 80 diabetic and 80 non-diabetic, Danish, myopic adults. The author found that the mean refractive values of the two groups were  $-2.00D$  and  $-3.00D$  respectively, but late-onset myopia was more prevalent among diabetic patients (40.0% compared to 22.5% in non-diabetics). However, this study was based on self-reported data (Fledelius 1986). As stated above an increase in lens thickness (Brown and Hungerford 1982) or in surface curvature (Sparrow *et al.* 1990) could explain the increased prevalence of late-onset myopia in diabetic patients.

Authors who have investigated the effect of more acute changes in plasma glucose concentration have reported that an acute decrease in plasma glucose levels causes a (transient) hyperopic change (Keller 1973; Marmor 1973; Gwinup and Villarreal 1976; Fledelius and Miyamoto 1987; Saito *et al.* 1993; Willi 1996). Conversely other authors report the possibility of increasing hyperopia under *hyperglycemic* conditions (Huggert 1954; Rosen 1956; Vaughan and Asbury 1980). Rosen (1956) described a case-report where a hyperopic change was observed with increasing blood glucose levels, and Huggert (1954) studied the crystalline lens of 23 diabetic subjects. Ten subjects were myopic and 14 subjects were hyperopic (in one subject hyperopia was preceded by myopia, and was therefore counted twice). Vaughan and Asbury (1980) described both myopic and hyperopic refractive error changes in diabetes with changing blood glucose concentration.

Thus both myopic and hyperopic shifts have been reported in diabetic patients and the underlying mechanism responsible remains to be established. Table 1 summarises the results of various studies published in the scientific literature, describing acute refractive error changes with induced changes in blood glucose levels.

| <b>Author</b>           | <b>Year</b> | <b>No of subjects</b> | <b>Key findings</b>  |
|-------------------------|-------------|-----------------------|--|
| Huggert                 | 1954        | 23                    | Myopia before and hyperopia after the onset of treatment       |
| Gwinup <i>et al.</i>    | 1976        | 10                    | Myopia after induced hyperglycaemia                            |
| Fledelius               | 1987        | 72                    | Myopia and hyperopia (before and after the onset of treatment) |
| Saito <i>et al.</i>     | 1993        | 5                     | Hyperopia after the onset of treatment                         |
| Furushima <i>et al.</i> | 1999        | 7                     | Myopia after induced hyperglycaemia in non-diabetic subjects   |
| Steffes                 | 1999        | 1                     | Myopia after induced hypoglycaemia                             |
| Okamoto <i>et al.</i>   | 2000        | 14                    | Hyperopia after the onset of treatment                         |
| Guisti                  | 2003        | 20                    | Myopia after the onset of treatment                            |
| Sonmez <i>et al.</i>    | 2005        | 18                    | Hyperopia after the onset of treatment                         |

*Table 1. Conflicting results presented in the literature comparing acute refractive changes in the eye to changing blood glucose levels.*

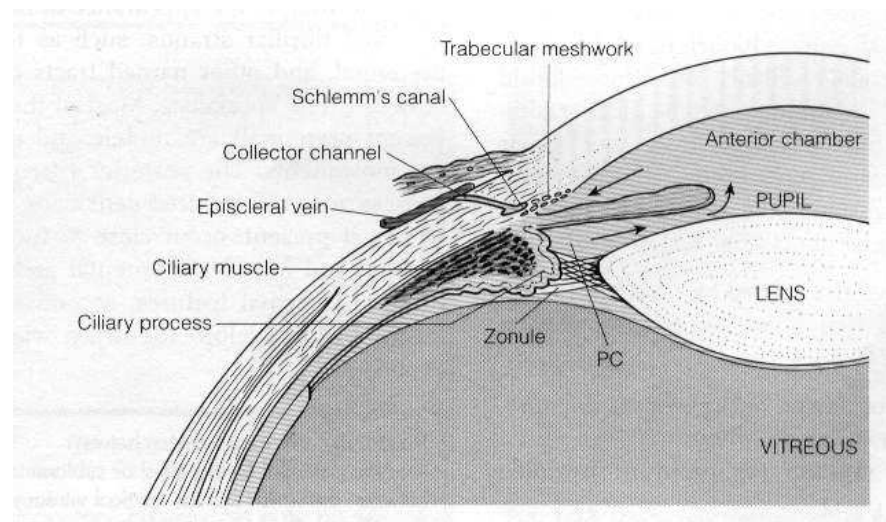
Different approaches have been adopted in the above studies, which may explain the lack of agreement in findings. It is important to define the words 'acute effect', since investigators describe the refractive shifts at different time points after the commencement of the study. The majority of investigators have been interested in the response of the eye when elevated blood glucose levels were reduced to near normal values. This reduction could take several weeks to months to occur and might be considered a sub-acute response rather than a true acute response.

Although our knowledge about the time required for glucose to enter the eye after blood plasma glucose concentration is increased is limited to animal models, it is thought that the latency is between 5 minutes (Cameron *et al.* 2001) and 30 minutes (Chou and Lin 2000).

Researchers (Caird *et al.* 1969; Saito *et al.* 1993) have examined the refractive shifts occurring after instigating (insulin) treatment for the first time. This may not reflect blood glucose changes as they occur on a day-to-day basis in treated diabetic patients. To investigate refractive error shifts in diabetic patients, the conditions require more 'regular' glucose fluctuations. The response of the eye to the body's first experience with oral diabetes medications or injected insulin has not been thoroughly investigated. Insulin acts on cells throughout the body to stimulate the uptake, utilisation and storage of glucose. A sudden drop in extra-cellular glucose levels or an increase in intra-cellular glucose (as would occur in undiagnosed diabetes) might change the refractive index more significantly than when this change occurs on a hourly basis.

It is possible that in diabetic patients, the membrane of the crystalline lens permits an influx of water into the lens when blood glucose levels fall. During hypoglycaemia, the glucose concentration in the aqueous humour will

decrease (Figure 1). As a consequence, the osmotic balance along the lens capsule or epithelium will increase the influx of water from the aqueous humour into the lens, and induce a refractive shift. This effect may be dramatic when treatment for diabetes mellitus is first initiated and may be different to the sorts of changes that occur when the diabetes is under control.



*Figure 1. Aqueous humour is produced by the ciliary body and secreted into the posterior chamber. It flows through the pupil into the anterior chamber, where it is drained by the trabecular meshwork and Schlemm's canal (Reprinted from Forrester et al. 2002, with permission of Elsevier).*

Gwinup and Villarreal (1975) investigated the effects of chronic and acute changes in blood glucose levels on refractive error. In a group of ten subjects with diabetes they demonstrated that a decrease in blood glucose concentration of 100mg/dl (5.6mM/l) altered the refractive error by about 0.50D over a period of few weeks. Furthermore, they demonstrated an acute change in refraction with changing blood glucose concentration in six out of ten of the diabetic subjects (Figure 2). In all six patients, the refraction became more myopic in both eyes within 15 minutes after an injection of glucose. The authors suggested that the accumulation of by-products of glucose metabolism within the lens, followed by the accumulation of water, had caused the lens to swell resulting in myopia (Gwinup and Villarreal 1976).

The initial blood glucose concentration in all 6 subjects was below 150mg/dl (8.4mM/l), but no measurements of blood glucose concentration were taken afterwards.

Two of the diabetic subjects were aphakic and both aphakic subjects became more hypermetropic (rather than more myopic) after blood glucose concentration increased. These data also appear to suggest that the cause of the myopic refractive shift in the phakic subjects was likely to be due to the crystalline lens. The data from the aphakic subjects suggest that the cornea and the posterior lens capsule also appear to have an influence on refractive error fluctuations. However, since the refractive change was opposite in sign in the phakic versus the aphakic eyes, the anterior lens capsule, subcapsular epithelium and lens fibres may act to reverse any change induced by the cornea or by the lens capsule. Additionally, the data support the theory that the latency of refractive changes after blood glucose change is small.

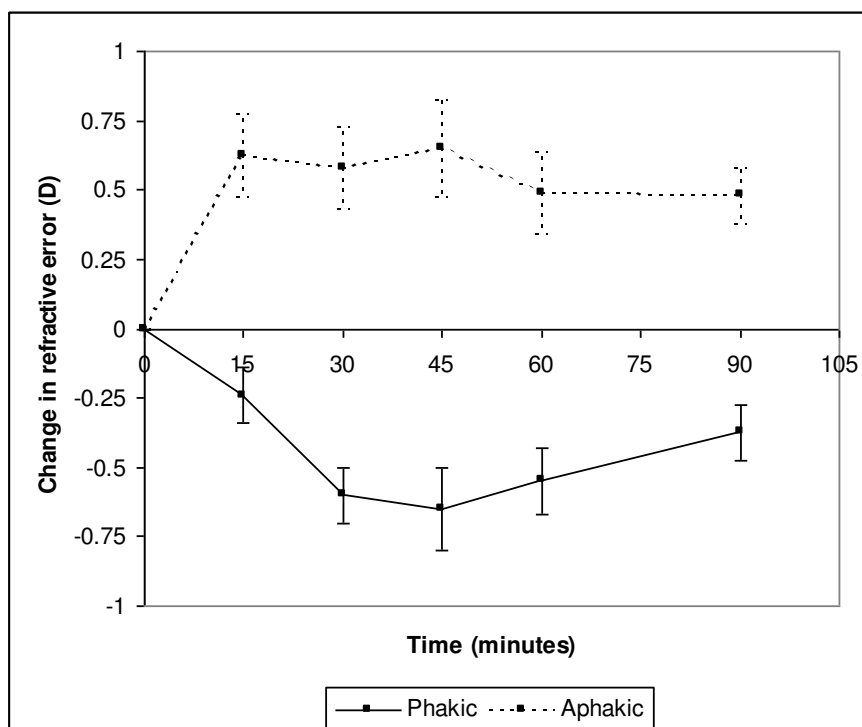


Figure 2. Changes in refraction of the eyes of aphakic diabetic subjects ( $n=4$ ) and diabetic with intact lenses ( $n=12$ ) following induced hyperglycemia (mean  $\pm$  SD) (Adapted from Gwinup and Villarreal 1976).

Steffes (1999) obtained similar results to those of Gwinup and Villarreal (1976). In a fasting Type I diabetic patient with a blood glucose concentration of about 95mg/dl (5.3mM/l), the administration of insulin caused the blood glucose concentration to reduce to about 50mg/dl (2.8mM/l) (Figure 3).

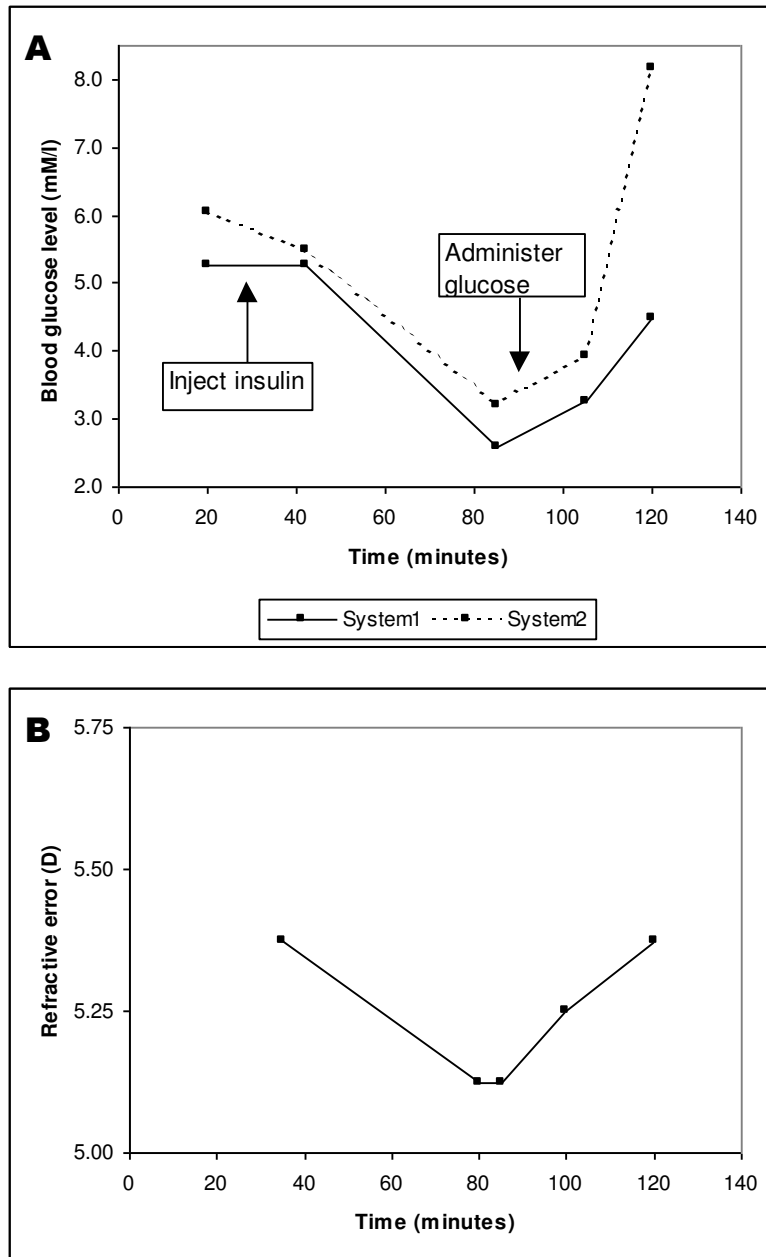


Figure 3. Comparison of (A) finger prick measurements of whole blood glucose taken with two different systems (circles and squares); and (B) measured refractive error during induced hypoglycemia (Adapted from Steffes 1999).

Upon administration of dextrose (glucose), the patient's blood glucose increased to about 5mM/l with system 1 and >8.0mM/l with system 2. Concurrent measurements of refractive error were made, and the changes in refractive error were closely tracked those of the blood glucose concentration. To the best of our knowledge, this is the only *in vivo* evidence in the literature of refractive error changes due to induced *hypoglycaemia*. The results of this study concur with Gwinup and Villarreal's finding that the latency is short.

A novel approach to the question of whether myopic or hypermetropic changes are caused by hyperglycemia was reported by Furushima *et al.* (Furushima *et al.* 1999). Somatostatin, which is known to suppress insulin secretion, was administered to a group of seven non-diabetic volunteers (mean age 23 years), with normal vision. Together with a glucose-load, the somatostatin induced hyperglycemia in all subjects. A statistically significant average change of  $-1.93 \pm 0.39$  D in refractive error was observed 150 minutes after the glucose load despite the instillation of cycloplegic eye drops ( $p < 0.01$ ). The subjects' refractive error changed concurrently with their blood glucose level, which increased from  $3.9 \pm 0.6$  mM/l to  $15.6 \pm 2.3$  mM/l. The time taken to reach the maximum refractive power was the same as the time taken to reach the maximum blood glucose level. Ultrasound measurements showed significant changes in the dimensions of both the anterior chamber depth which became shorter, and the crystalline lens, which became thicker. The axial length of the eye remained unchanged.

### **Aetiology of refractive error shifts in diabetes**

Thus there is still some uncertainty about how refractive error in humans is influenced by acute and chronic changes in blood glucose concentration.

Both hypermetropic and myopic refractive shifts could occur from morphological changes in the cornea, crystalline lens, axial length or retinal thickness. The last two possibilities have been investigated previously, and no change in either axial length or retinal thickness was found with changing blood glucose concentration in 28 eyes (Okamoto *et al.* 2000).

### *Myopia*

A myopic shift with decreasing blood glucose concentration in diabetes could be explained by the accumulation of metabolites such as sorbitol. The intracellular glucose levels in the crystalline lens are not regulated by insulin, and therefore the concentration of glucose within the lens increases with increasing blood glucose concentration (Olansky 2004).

Aldose reductase is an enzyme that converts any excess of intracellular glucose into sorbitol, which can then be further metabolised into fructose. Cell membranes are relatively impermeable to sorbitol allowing this compound to accumulate within the lens. This is paralleled by an influx of water from the aqueous humour, producing lenticular swelling (Gabbay 1973). This leads to an increase of lens curvature and induced myopia.

### *Hyperopia*

A decrease in blood glucose concentration in the aqueous humour could also cause a transient difference in osmotic pressure between the aqueous humour, crystalline lens and vitreous body. As a result, a decrease in refractive index might develop in the lens, leading to a hyperopic shift. A decrease in lenticular refractive index from 1.42 to 1.40 has been shown to produce a hyperopic change of 3.20D (Planten 1981). Therefore, small changes in refractive index could produce significant changes in refractive power as stated above.

Alternative theories have evolved from animal studies, when the response of the lens to changing glucose levels has been investigated (Jacob and Duncan 1982). The crystalline lens was placed in solutions with different glucose concentrations, causing a hyper-osmotic and norm-osmotic shock. A hyper-osmotic (or hypertonic) situation is created when the solution has a higher glucose concentration than the crystalline lens. The initial response in a frog lens to hyper-osmotic shock was a decrease in lens volume. A second response was gradual lens swelling, due to the net entry of glucose and water during high aqueous humour glucose levels. On return to norm-osmosis



(when the solution has an almost equal glucose concentration to the lens), the already swollen lens experienced hypo-osmotic shock and swelled still further with increased K<sup>+</sup> permeability. This increase in K<sup>+</sup> efflux (outward flow) is presumably used by cells to increase their volume in response to osmotic stress (Beebe *et al.* 1990).

| <b>Author</b>           | <b>Mean change in blood glucose concentration (mM/l)</b> | <b>Mean change in refractive error (D)</b> | <b>Comments</b>  |
|-------------------------|--|--|--|
| Gwinup and Villareal.   | + 8.4  | -0.50                                      | None   |
| Furushima <i>et al.</i> | + 11.2   | -2.00                                      | Cycloplegic refraction, insulin suppression, non-DM subjects |
| Steffes                 | - 2.8  | -0.25                                      | One subject  |

*Table 2. Results from three studies showing refractive error changes after induced hypo- or hyperglycemia.*

Table 2 summarises the results of the three human studies described above. It is perhaps surprising that cycloplegic refraction results in such a large refractive change. However, insulin secretion was completely suppressed in these subjects, resulting in very high blood glucose levels. Glucose would have been building up into the aqueous humour and extra-cellular space of the cornea and crystalline lens. As a result, the refractive index of the aqueous humour would be increased significantly causing a large refractive change.

### **Implications for the optometrist**

Hyperglycemia often goes unnoticed by diabetic patients, whereas there may be several undesirable symptoms associated with acute hypoglycemia. A rapid decline in blood glucose concentration is associated with sweating, trembling, anxiety, weakness, hunger, nausea and vomiting. However, prolonged hypoglycemia can include more severe symptoms like visual

disturbances, restlessness, irritability, inability to concentrate, mental confusion, and personality changes among others (National Diabetes Information Clearinghouse 2003). Thus diabetic patients may take immediate action to correct for hypoglycaemia (usually with an intake of glucose), and any associated refractive error shifts may therefore be avoided. Consequently, the response of the crystalline lens to untreated hyperglycaemia may be of more immediate concern to the optometrist involved in carrying out eye examinations on diabetic patients.

Thus, refractive error change can be influenced by fluctuating blood glucose concentration. Therefore it seems appropriate to enquire about blood glucose concentration when carrying out eye examinations on diabetic patients. If blood glucose concentration is uncharacteristically high or low at the time of refraction, then it may be wise to repeat the refraction prior to prescribing, as well as advising patients about the possible implications for diabetic retinopathy and other complications associated with the disease.

In hyperglycemia, glucose can accumulate in the lens causing an increase in curvature and a shift towards myopia (Mantjarvi 1988). However, hyperopic changes have also been shown to occur during hyperglycemia and this is thought to be due to a decrease in refractive index in the crystalline lens. If the geometric effect dominates (swelling of the lens altering lens curvature), the refraction will shift towards myopia. If the refractive effect dominates, the refraction will shift towards hypermetropia.

## **CONCLUSIONS**

The underlying mechanism governing the relationship between plasma glucose concentration and refractive error is not fully understood. There is no clear answer as to whether the refractive changes seen are due simply to changes in refractive index of the cornea, aqueous humour or lens or whether swelling of the lens occurs causing changes to its curvature, position or size. It is possible that both the refractive index and surface curvature change,

in which case either a myopic or hyperopic response may occur, depending on the individual's physiology. However, from a clinical point of view, fluctuating blood glucose concentration has an influence on short-term changes in refraction, and these changes may be large enough to measure. In the majority of cases, people with diabetes appear to become more myopic as their blood glucose concentration increases, but a significant minority become more hypermetropic. Where acute changes have been reported in the literature, the latency of these changes appears to be short, being of the order of a few minutes. This implies that a hypo- or hyperglycemic state could influence refractive findings during the eye-examination. When taking a diabetic patient's history, it seems appropriate to enquire about the most recent blood glucose concentration, or even suggest taking a finger stick test before the eye-examination. It should be borne in mind that (consistently) high blood glucose readings could induce transient myopia.

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