

INSULIN DEPENDENT DIABETES MELLITUS
PSYCHOSOCIAL, EDUCATIONAL AND
LIFESTYLE IMPLICATIONS

CENTRE FOR NEWFOUNDLAND STUDIES

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PSYCHOSOCIAL, EDUCATIONAL AND LIFESTYLE IMPLICATIONS**

By

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A paper folio submitted to the School of Graduate Studies in partial fulfillment of the requirements for the degree of Master of Education.

Faculty of Education
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January, 2000

St. John's

Newfoundland



FOREWORD

This paper folio is, for the most part, written for educators, school counsellors, school administrators and allied professionals who work, or might in the future, work with school aged children with Insulin-Dependent Diabetes Mellitus. A particular focus is placed on understanding and working with adolescents who have been diagnosed with this disease.

This topic is an important area of study to this writer as I had served as an educational consultant in a pediatric hospital as a member of the diabetes clinic team. As a member of this team for eight years until 1996, I had worked with hundreds of school aged children, mostly adolescents, and their families who have been dealing with this dreadful, often frightening and life altering disease. The emotional and financial cost for families dealing with diabetes is often figuratively and literally draining.

During this eight year period, I had close interpersonal relationships with students of all ages and their parents and endeavoured to understand and help them with many of their immediate and long-term concerns and fears which often accompanied a child's diabetes. It became quite evident, during my stay at the Janeway Child Health Centre, how important it is to have a dedicated, comprehensive diabetes team. I was very fortunate to have worked with a team that consisted of three pediatricians, a nurse, dietician, social worker, dental hygienist, educational consultant and clinical psychologist.

While working with this team and with parents and students, I experienced the joys

of seeing the great majority of these children meet success in school, athletics, university and the maintenance of their diabetes. I also witnessed the emotional lows and the sense of helplessness and hopelessness in some families in which depression was manifested in eating disorders, substance abuse and suicidal ideation was experienced. As a teacher and counsellor, I have witnessed how serious this disease is and can be.

It must be emphasized, however, that diabetes need not limit the quality of one's life. With good diabetic control and the support of family, friends, school and medical personnel, people with diabetes may experience few, if any, restrictions in their life experiences and career choices.

This paper folio is divided into three sections. The first section will discuss what Insulin-Dependent Diabetes Mellitus is, its prevalence and incidence as well as diabetic complications and life style implications for people with IDDM. Relevant research with its implications for present and future care will be addressed.

The second section will consist of a literature review of psycho educational and psychosocial research in relating to students with IDDM. This section will focus on learning patterns and cognitive functioning in these students as well as psychosocial issues such as coping strategies, family functioning, peer relationships and its inter-relationship with diabetes maintenance.

The third section is directed towards school personnel who work with students with diabetes. Guidelines and suggestions will be given to classroom teachers, physical education teachers, counsellors and administrators to help them provide for the needs of students with diabetes. Legal implications for schools will also be addressed. It is very important for school personnel to appreciate the burden that IDDM can place on children as well as their families. The financial and emotional burden of IDDM will therefore be discussed. Typical treatment plans for children with diabetes will be reviewed as well as the composition of a comprehensive team, which is essential in providing services to this population.

In this work, this writer appreciates license or leeway to discuss at times, personal perspectives and/or personal experiences that may be relevant to this topic.

IDDM: NATURE AND LIFESTYLE IMPLICATIONS

Foreword

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IDDM: Nature of IDDM and Life Style Implications

Introduction

There are four general types of diabetes which will be identified and discussed. The major focus in this paper, however, will be IDDM which develops predominantly in children and young adults but may be present in all age groups. This type of diabetes therefore is most seen by school personnel in working with students on a daily basis. In order to do justice to this topic, however, we will discuss the four general types of diabetes.

Insulin-Dependent Diabetes Mellitus (IDDM) is the classical life threatening form of diabetes, which only became treatable with the discovery of insulin by Banting and Best in 1921. This type of diabetes is the form of the disease most well known to the public even though it accounts for less than ten percent of the total diabetic population. By definition Insulin-Dependent Diabetes Mellitus identifies patients who cannot survive without insulin treatment. The most common cause is auto immune destruction of the B cells of the islets of Langerhans which produces insulin. Insulin deficiency is so profound that ketoacidosis will develop and death will occur without insulin replacement.

Classification System of Diabetes

TYPE I - Insulin-Dependent Diabetes Mellitus - IDDM

Type I, also called Insulin-Dependent Diabetes Mellitus was previously named juvenile onset diabetes. It is the type of diabetes that develops most often in childhood but may be

experienced by adults thirty years old or even older. It occurs in people who are usually thin or average weight and all people with this type of diabetes must be treated with insulin.

The beta cells in the pancreas are the only cells in the human body that produces insulin. These beta cells, as well as others, which are located in the islets of Langerhans, make several other hormones which in turn make digestive enzymes (Davidson, 1998). In individuals with IDDM, the B cells of the pancreas are destroyed by autoimmune processes whereby virtually no insulin is produced. Autoimmune destruction is caused when the body's immune system, which is responsible for combatting and destroying body invaders such as viruses and bacteria, "thinks" that its own pancreatic B cells are foreign. The immune system therefore initiates a response to destroy these cells. When this happens, this type of diabetes, in the absence of insulin replacement cause ketoacidosis, and ultimately death (Pickup, 1997).

As the term implies, ketoacidosis involves ketones which are the chemical by-product of fat breakdown. Some amount of fat breakdown is always taking place in the body and consequently there is always some normal amount of ketones in the blood. The amount of ketones in the blood increase, especially if a person does not eat for 12 to 18 hours and the body tends to use stored fat as fuel. The problem of ketoacidosis develops when the pancreas of people with IDDM is not producing insulin and there is not enough insulin by injection to burn sugar. Insulin is needed to burn sugar in the body. In a non-diabetic person, as soon as the blood sugar rises, as it does after normal meals including carbohydrates, the body uses the sugar as fuel and stores away fat for later use when carbohydrates are not available. In the normal situation,

no carbohydrate is eaten, the body has a relatively low blood sugar, insulin production by the pancreas decreases and the body turns to fat for energy. In people without diabetes, ketones increase a small amount in the blood from fat breakdown and may spill over into the urine. In people with untreated or poorly treated Type I diabetes or IDDM, sugar builds up in the bloodstream but without insulin can not be used for fuel. The body then turns to fat stored for energy which in turn can cause high levels of ketones in the blood. In this case, ketones, which is an acid may spill over into the urine but can also cause the whole body to become extremely acid. Diabetic keto acidosis (DKA) is therefore caused by having far too little insulin in the blood which may be due to neglect or cases of denial or anger (Saudek, 1997). DKA can cause major sickness, damage to internal organs, including the brain, or it can be fatal. Symptoms of DKA include extreme thirst, frequent urination, fatigue, dehydration and overall physical illness. Diabetic ketoacidosis has been dangerously mistaken as a bad influenza. Although ketoacidosis is generally considered a complication of IDDM, DKA is a possibility in all types of diabetes where continued hyperglycaemia (high blood sugars) are untreated or poorly treated.

Hypoglycaemia occurs from a relative excess of insulin in the blood and results in excessively low blood glucose levels. The level of glucose that produces symptoms of hypoglycemia varies from person to person and for the same person under different circumstances. Hypoglycemia usually occurs gradually and is generally associated with typical warning signs which may include rapid heartbeat, perspiration, shakiness, anxiety and hunger. When symptoms occur, preventive action can be taken by eating carbohydrates. If warning signs are absent or ignored and blood glucose levels continue to fall, hypoglycemia may lead to

confusion, stupor and finally unconsciousness. Most people, however, never suffer such severe hypoglycemia. Hypoglycemic reactions are not ordinarily associated with loss of consciousness (Cryer, 1994), except in individuals who suffer from diabetic neuropathy, those who are intoxicated by drugs or alcohol, or in children who may not understand or tend to ignore the warning signs. Hypoglycaemia does not occur in people who only require medical nutrition therapy and exercise, and is rare in people treated with oral glucose lowering agents (Type II)

Hypoglycaemia, or low blood sugar, will be discussed separately as this most common complication has special consideration and implications for educational personnel who are working with students on a daily basis.

Features of Type I Diabetes (IDDM)

The age of onset is usually but not always in people who are less than forty years old, the most common age group being childhood. There is a relatively quick onset with symptoms developing over a period of weeks and months. The patient usually complains of constant thirst, frequent urination, weight loss and possibly a progression to ketoacidosis. Quite often or usually there is no known history of Type I diabetes in the family. Risk is increased if there is a strong family history of IDDM and it is mildly increased if a single family member has been diagnosed with the disease (Davidson, 1998).

In the United States, according to Saudek (1997) the risk for a person to be diagnosed with IDDM when the mother is Type I is between two and three percent, while it is six percent if

the father is Type I. The risk is higher if either parent had developed diabetes before the age of eleven. If a sibling has diabetes, the probability for another sibling is between three and fifteen percent. In identical or monozygotic twins, the risk factor if one twin develops IDDM is between thirty-five and fifty percent chance that the other twin will develop the disease.

With Type I diabetes, insulin replacement is always needed to prevent ketoacidosis. Typically, it is quite difficult to keep blood sugars in ideal range as it is very sensitive to small changes in diet, exercise and insulin dose. During the honeymoon period, or within a few weeks or months of onset, diabetes may be easier to control or stabilize. In many cases very little insulin or none at all is needed and patients have sometimes realized a false sense of security or a feeling that the diabetes is going away or is getting better. This honeymoon phase may last from a few months to a year. As a general rule, however, people with diabetes need two or more insulin injections per day (Brink, 1997).

Etiology of IDDM

According to Atkinson and McLaren (1994), autoimmune destruction of the B cells accounts for over 90 % of the cases in Western countries. However, the etiology is multifactorial and imperfectly understood. IDDM is the result of an unfavourable interaction between environment factors and an inherited predisposition to the disease. Pickup and Williams (1997) suggest that based on research with monozygotic twins that the genetic component accounts for 30-40% of the total risk of developing IDDM. Inheritance of susceptibility to IDDM does not depend on a single gene but a number of genes at different loci or locations.

Let us further consider the hereditary factor. The best way to learn if heredity is a factor in the development of a disease is to see if it tends to run in families. Secondly, we determine if the disease occurs more often in monozygotic twins than dizygotic twins. If the condition were 100% genetic or hereditary, in the event that one identical twin had IDDM, then the other twin having the identical genes would have it also. As mentioned earlier, the concordance rate in identical twins is high, between 35-50%, where one has IDDM, the other twin does too. The concordance rate is not 100% however, which shows that IDDM is not entirely genetic. It is also important to note that although the risk does increase somewhat when a parent or sibling has IDDM, it is unusual for several people in a family to develop Type I diabetes.

Studies suggest that something in the environment plays a role in the development of IDDM (Davies, 1994). It is not known however, what environmental factors may trigger B cell destruction in genetically susceptible people. This trigger may be a virus, although no particular virus has been found in people with Type I diabetes. A susceptible person may be exposed to a toxin in food or the air but again evidence is lacking. There is no evidence that emotions, psychological or physical stress causes IDDM and to dispell an old wives tale: IDDM is not caused by excess sugar intake.

In conclusion, we can only say that IDDM is an autoimmune disease caused by the destruction of B cells in the pancreas. It occurs in people with a genetic susceptibility but requires something in the environment to trigger its development.

TYPE II - Non-Insulin Dependent Diabetes Mellitus (NIDDM)

Type II diabetes accounts for over 90% of all diabetes, where Type I accounts for less than 10%. Although Type II is capable of causing all the symptoms and complications of Type I, it is a very different form of diabetes. Saudek (1997) defines Type II diabetes as “that form of diabetes that does not require insulin treatment to avoid ketoacidosis”. Except under unusual stress, people with Type II should not have ketoacidosis even when insulin treatment is withdrawn. The reason for this is fairly simple. The pancreas of a person with Type II diabetes does make some insulin, just not enough. Even small amounts of insulin made can restrain the uncontrolled breakdown of fat that causes DKA.

Unlike Type I diabetes, the typical person diagnosed with Type II is older, overweight, with a strong family history of diabetes. Usually the presentation of the disease is characterized by a slow onset; with symptoms developing over a period of months to many years. Thirst, frequent urination and weight loss may not be noticed and hence Type II diabetes can be described as silent disease. Unlike IDDM, Type II has well defined risk factors, as mentioned earlier, that place people at increased risk of this disease.

People with Type II diabetes may experience very high blood sugars. Sometimes low blood sugars are seen, but in most cases it is easier to control without the extreme highs and lows that are seen in Type I. Treatment usually starts with prescribed diet and exercise. If the disease progresses or is under poor control, pills may be introduced by a physician.

Approximately 30% of people with Type II diabetes are prescribed insulin to manage their blood sugars. This does not transform the person's diabetes into Type I or insulin dependent but is rather a medical decision or chosen method to manage the patient's diabetes.

Etiology of Type II Diabetes

A striking feature of Type II diabetes is the genetic component which is much greater than in IDDM. Martin (1992) states that the concordance rate in identical twins is as high as 90% while the lifetime risk is up to 40% by having a first degree relative with the disease. The particular genetic factors involved are unknown, however. Excessive food intake and sedentary lifestyles of western-style societies greatly favour the development of NIDDM in that these factors predispose to obesity. It has been found that the risk of developing Type II diabetes is reduced by significantly regular daily exercise (Helmrick, 1991). This makes good sense in that regular exercise is important in reducing or negating obesity. The importance of obesity in causing Type II diabetes cannot be over emphasized. In the body most sugar breaks down in the muscle tissues. Although researchers do not know why, it is clear that the muscle cells of obese people are less responsive to insulin than thinner people. This condition is called insulin resistance. If a person is obese and his/her pancreas is not capable of making large amounts of insulin required because of insulin resistance, Type II diabetes can occur.

As with Type I diabetes, the combination of genetic susceptibility and environmental factors are considered important in inducing Type II diabetes to develop in individuals.

Gestational Diabetes Mellitus

Gestational diabetes mellitus is diabetes first diagnosed during pregnancy. It is usually a signal that Type II diabetes may develop later in the woman's life. Gestational diabetes is by far the most common form of diabetes in pregnancy occurring in up to 5% of women. During pregnancy the hormones made by the placenta counteract insulin, causing insulin resistance, as discussed earlier. If the female is also obese this can be an additional problem with insulin resistance. In most cases, however, the pancreas is strong enough to increase output of insulin as required. If not however, gestational diabetes will occur.

According to Sauder (1997) in three out of four cases, gestational diabetes mellitus disappears when its cause, pregnancy, is terminated. This form of diabetes, however, is a signal to the mother that her pancreas is not as strong as normal and she is at higher risk for Type II diabetes later in life.

Other Types of Diabetes

All other types of diabetes are those in which the cause is known. As discussed earlier, the pancreas is the only organ that produces insulin in the body. Upon severe abdominal injury, tumour or other disease, the pancreas may need to be removed, which will automatically induce diabetes. There are also drugs and medications used to treat other disorders that can induce diabetes. In some cases, the person may be predisposed to diabetes and the drug acts as a catalyst or agent that induces diabetes in the patient.

Finally, there are some uncommon diseases that cause diabetes by counteracting the effect of insulin. There are also rare cases where the cells of the body lack receptors to insulin as well as other conditions and syndromes that have a high positive correlation with diabetes. We will not delve further into these types of diabetes as the major focus of this paper is Type I diabetes.

Epidemiology of IDDM

The epidemiology of diabetes is a relatively new field of study. Scattered epidemiologic observations have been recorded for centuries but few systemic studies have been reported before 1960 that did not have major flaws in their design (Brink, 1987). It also stands to reason that since non-insulin dependent diabetes mellitus (NIDDM) accounts for more than 90% of people diagnosed with diabetes, that most of the research has been focused on this area. IDDM on the other hand, being relatively uncommon has had comparatively few epidemiological studies worldwide.

In order to provide clarity for this topic, it will be necessary to briefly discuss the terms epidemiology prevalence and incidence. West (1978) defines epidemiology as the study of the distribution and determinants of disease. The definition incorporates two main areas of investigation. The first describes the distribution of health status in terms of sex, age, race, geography, etc. The second involves the explanation of those observed distribution patterns in

terms of etiology. Frequencies of disease are expressed as rates to account for differences in population. In epidemiologic terms, a useful rate must include the following components: 1) the number of people affected, 2) the population among which the affected persons were observed, and 3) the time at which or during the cases were observed (Brink, 1987).

The two rates that appear in epidemiologic studies are incidence rate and prevalence rate. Incidence is the number of new cases of a disease that come into being during a particular period of time. The incidence rate is the number per specified unit of population. For example, three people per one hundred thousand in Canada may develop a particular disease each year. Prevalence, on the other hand is the frequency of the disease at any given point in time. Prevalence rate is the proportion of a given population that manifests the disease at any given point (Berger, 1999), irrespective of the length of time elapsed from the onset of the illness to the time that the prevalence is measured.

Insulin dependent diabetes mellitus develops predominantly in children and young adults. With the exception of some native populations, IDDM occurs in populations on all continents. However, the incidence differs enormously between populations, countries and ethnic groups. Although research into the epidemiology is intensifying, distribution is known only for approximately 5% of the world population, most studies being performed in the northern hemisphere, more particularly Europe and North America (Derig, 1988).

The prevalence of IDDM in the pediatric and adolescent population is generally found to

be less than 2 per 1,000. According to Brink (1997) the best estimate of prevalence of IDDM in the under 18 year old population in the United States is 1.4 per 1,000. According to epidemiological convention, the incidence of IDDM can be described in terms of place, time and personal specific characteristics.

Epidemiology According to Place

IDDM in Europe

Overall, Europe encompasses more than a ten-fold difference in incidence, ranging from 35 cases in Finland to three new cases per 100,000 children up to age 14 annually (Karvonen, 1993). A major feature in reviewing IDDM incidence in Europe is the high incidence level in Nordic countries, especially Finland, as compared with the rest of Europe. For example, Estonia, although it is very similar to Finland in ethnic and cultural background, has an incidence of only one-third of that in Finland. Iceland, a country founded centuries ago by migration from Norway has lower incidence rates than Norway. There is evidence of geographical variation within Germany itself where south Germany has 11 per 100,000 as compared to 7 per 100,000 in Eastern Germany (Pickup, 1997). This considerable incidence variance among and within countries with homogeneous composition, strongly suggests that environmental factors contribute significantly to the geographical variation (Joner, 1989).

Epidemiological research of European populations estimates that approximately 10 children per 100,000, ages 0 - 14 years will develop IDDM each year (Pickup, 1997). Table 1 gives reported incident rates (per 100,000 per year in Europe; age group 0 - 14 years.)

Table 1

Country (region)	Period	Boys	Girls
<i>Nordic countries</i>			
Finland			
Whole nation	1983-88	36.9	31.6
Norway			
Whole nation	1978-82	23.3	20.7
Sweden			
Whole nation	1977-83	24.2	22.0
Denmark			
Two countries	1989-90	21.5	21.4
Iceland			
Whole nation	1980-89	10.5	11.1
<i>Baltic region</i>			
Estonia			
Whole nation	1983-88	10.7	10.0
Latvia			
Whole nation	1983-88	6.4	6.7
Lithuania			
Whole nation	1983-88	6.5	7.0
<i>British Isles</i>			
England			
Oxford	1989-90	17.8	14.9
Yorkshire	1978-90	13.7	13.3
Scotland			
Whole region	1977-83	20.1	19.6
Tayside	1980-83	21.5	18.2
Northern Ireland			
Whole region	1989-90	17.8	15.4
<i>Central Europe</i>			
The Netherlands			
Whole nation	1978-80	9.9	9.5
Five countries	1989-90	11.2	10.8
Belgium			
Antwerp region	1989-90	9.2	10.4

Table 2

Country (region)	Period	Boys	Girls
France			
Four provinces	1989-90	9.2	10.4
Luxemburg			
Whole nation	1989-90	12.1	12.6
Germany			
Former GDR	1982-84	7.4	7.4
Austria			
Whole nation	1979-84	7.9	7.3
Switzerland			
Whole nation	Cohorts 1970-72	7.2	(Males only)
Hungary			
Eighteen countries	1989-90	7.7	7.5
Slovenia			
Whole nation	1989-90	5.2	7.7
<i>East Europe</i>			
Russia			
Novosibirsk district	1983-89	4.6	4.9
Poland			
Nine western provinces	1989-90	5.3	5.8
Three cities	1989-90	5.7	6.0
Romania			
Bucharest region	1989-90	4.6	5.7
<i>South Europe</i>			
Italy			
Turin	1984-86	7.8	9.1
Lombardia	1989-90	7.6	5.9
Lazio	1989-90	7.2	5.8
Eastern Sicily	1989-90	11.2	9.0
Sardinia	1989-90	33.5	26.9
Portugal			
Three districts	1989-90	10.1	4.9
Greece			
Athens region	1989-90	10.9	7.7
Thessaloniki region	1989-90	5.3	3.8
Spain			
Madrid	1985-88	11.8	10.9
Catalonia	1989-90	10.5	10.6

*The standard population assumes sex and age classes (0-4;5-9;10-14 years)or equal sizes.

IDDM outside Europe

The incidence level in North America is comparable to that in the United Kingdom (16 per 100,000), but rates of non-whites are somewhat lower than whites (Karvonen, 1993). In Prince Edward Island, Canada, a very high incidence level was found as compared to the rest of North America. The rate of incidence was comparable to Nordic countries. (See table 2)

Tull et al (1991) found a very low incidence in non-whites (6 per 100,000) as compared to whites (29 per 100,000) in the Virgin Islands. Karvonen's research (1993) suggests that incidence levels in South America are below 7 per 100,000. In Austria and New Zealand, the general incidence among the population of European ancestry is comparable to central Europe (see Tables 1 and 2). As can be seen in Table 2, there is very low incidence of IDDM in Asian populations. IDDM is also quite rare in many native populations. For example in the circumpolar region as well as the black African population.

Epidemiology According to Time

Studies by Patterson (1983), Green (1992), Nystrom (1990) and others have provided well established evidence of increasing incidence of IDDM during the last two to three decades in Scotland, Denmark, Finland and The Netherlands. Green (1992) has found a steady increase in incidence during the last few decades, where Bingley (1989), in Europe has found some instances of doubling in incidence per generation. Increasing incidence has predominantly been reported by Europe and some Caucasian populations in North America. A recent study in Pennsylvania has shown evidence that IDDM has increased sharply during the last few years. (Dokheel, 1993)

In the northern hemisphere, it has been consistently found that more cases of IDDM are diagnosed during autumn and winter, than spring and summer. The same seasonality trend was identified in Chile (Levy-Marchal, 1995). Some have concluded, according to Pickup, (1997) that this seasonal trend is seen particularly in children around puberty and is less evident in the first years of life. Pickup elaborates further by stating: "as the destruction of B cells of the pancreas may begin several years before the clinical onset of the disease, this pattern may reflect seasonal variations in individual susceptibility or certain environmental precipitating factors." In his study with subjects without diabetes, Macdonald (1987) found that glucose tolerance in non-diabetic subjects deteriorates slightly during the winter months. This may indicate an increasing demand for increased production of insulin during the winter months, thereby precipitating diabetes in people with partially destroyed B cells.

Epidemiology according to person-related variables

As discussed earlier, studies have estimated that risks of siblings and children of IDDM patients developing the disease is between 5-10% compared to 5% in the general population. The risk may be smaller for children of diabetic women than for offspring of diabetic men. Some research has shown that the risk of IDDM in children increases with increasing maternal age (Flood, 1982).

Attempts have been made to characterize inheritance patterns of IDDM. Although no definite models have been confirmed (Pickup, 1997), it seems that simple dominant inheritance is unlikely. It should be stressed that only a limited proportion of genetic susceptibility can be

explained at present and that relatively few people who have genetic markers associated with IDDM actually develop the disease. It should also be kept in mind that although there is apparent strong genetic susceptibility to IDDM, about 80-90% of newly diagnosed children will not have close relatives with the disease (Dahlquist, 1985).

Until recently it has been consistently found that boys have a slightly higher risk of IDDM than girls. The influence of gender, however, apparently interacts with other determinants which are most likely non-genetic because advanced genetic analysis has failed to demonstrate any gender difference in susceptibility (Pickup, 1997). Staines (1993) found that the incidence of IDDM peaks at preschool age and later around puberty for both sexes. This age dependency profile may be related to increased susceptibility and/or exposure to environmental factors at certain ages.

Non-genetic factors or determinants of IDDM are of particular importance as these are possibly modifiable in the aim of preventing the disease. Most research has focused on exposure to infectious diseases, immunizations, nutrition, socio-economic status and stressful life events (Pickup, 1997).

Several studies have focused on the possibility that dietary factors play a role in the etiology of IDDM. The association between IDDM and breastfeeding has been studied. It was found that there is an association between short duration of breastfeeding during infancy and the risk of the children developing IDDM (Dahlquist, 1989). This association, however, is described

as weak and even, if causal in nature, would only explain a few cases.

Conflicting results have been found in studies that have attempted to co-relate the risk of IDDM and socio-economic status. Pickup (1997) contends, however, that opposite trends have been found in these studies, case control techniques were questionable, and the associations found were positive but quite weak.

Some studies have addressed the possibility that stressful life events may contribute to the pathogenesis of IDDM. It is possible that stressful events and psychological distress, through elevated stress hormones increase the demand for endogenous insulin production and therefore precipitates IDDM in susceptible individuals (Hagglof et al, 1991). Hagglof et al found some confirmatory evidence but the association is weak.

Acute and Long-term Complications of IDDM

In this section we will discuss two broad categories of complications: acute or immediate and long-term. Acute complications are directly caused by high blood sugar and may be experienced within minutes or hours after blood sugar increases and may disappear very quickly as blood sugar decreases. These complications include thirst, frequent urination, blurred vision, fatigue, weight loss and in the extreme ketoacidosis and/or coma. Long-term complications usually occur over many years or decades and are often irreversible. These complications can negatively affect blood vessels, eyes, kidneys, nerves, skin, or essentially total bodily function.

This section will initially address the acute complications of the disease and later address long-term complications. We need to keep in mind that complications in IDDM and NIDDM can be quite similar. Ketoacidosis which is the hallmark of Type 1, or IDDM was discussed earlier, and hypoglycaemia (low blood sugar) which is a particular concern for educators will be discussed separately.

Acute Complications

Thirst and Frequent Urination

When there is a high concentration of glucose, or in other words a high blood sugar, an individuals' blood tends to thicken. The brain registers this as a sign of dehydration and sends messages to the body to drink fluids in order to dilute blood back to normal. Depending on the level of hyperglycaemia, thirst and frequent urination (polyuria and polydipsia) can be quite severe. It is not uncommon for people to urinate every half hour or more and to continually drink fluids in enormous amounts. This situation of course can be intensified if the person drinks fluids that contain sugar.

Blurred Vision

Blurred vision is often the major symptom that leads a person with diabetes to seek medical help. Permanent visual impairment can be caused by diabetes and therefore it is often scary for people with diabetes who experience problems with their vision. In contrast with retinopathy, a severe complication of the eye, blurred vision often occurs as an immediate effect of high blood sugar and thankfully can be quickly reversed when steps are taken to bring blood

sugar levels back to normal.

The lens is a focusing device which is located towards the front of the eye. It focuses our vision and changes shape depending on whether we are focusing on objects nearby or at a distance. If the blood sugar is high for a number of days, the lens tends to swell, lose its elasticity and therefore cannot change shape easily. The result is blurred vision. Fortunately with stabilized blood sugar, most people experience improved vision within a few days or less.

Fatigue

It is important to remember that without insulin, carbohydrates cannot be converted to energy by cells in the body. Fatigue is generally caused by a lack of insulin which is essential to help convert these carbohydrates into a usable energy source. Fatigue from high blood sugar can be mild or severe. When it is mild it may be ignored or misinterpreted as being caused by overwork, age, etc. In the extreme, however, a person may fall asleep during a conversation, while eating, or when operating a vehicle. Fatigue is also a symptom of low blood sugar which will be discussed later.

Weight Loss

Weight loss is also caused, like fatigue, by a lack of insulin or insulin function in the body. When blood sugar rises in the blood and is not converted to energy because of the lack of insulin, it spills into the urine. In effect, the body rids itself, through urine, of many calories that it would normally use for energy. Consequently people with high blood sugars, especially over

an extended period of time often experience fatigue as well as weight loss. For example, a person may ingest 2,000 calories which is required to maintain his/her body weight, but 300 calories may be passed through the urine. Weight loss is inevitable.

Infections

The most common infection seen in patients with poorly managed diabetes is caused by a fungus called candida or monilia. This is often seen in females with diabetes in the form of vaginal yeast infections, but men are also susceptible to yeast infections in the groin or other areas of the skin. Other common infections include urinary tract infections, gum infections, infections of wounds, and infections of the extremities, especially the feet. People with poorly controlled diabetes are also susceptible to secondary bacterial infections, such as bronchitis which may complicate viral respiratory infections such as the flu.

The reason why high blood sugar or hyperglycaemia increases the risk of infections is two-fold. The first reason is that in a high sugar environment in the blood, fungus and bacteria that are causal agents for infection, thrive. Secondly, in the presence of high blood sugar, our immune system which is responsible for fighting infections is depressed and therefore less effective.

Personal Perspective

In my work with children and adolescents in clinical and classroom settings, I have all too often witnessed students experiencing these complications as well as listened to students

describe problems they experience in school because of these symptoms. Problems in school, associated with fatigue, focusing attention, feeling disinterested and emotionally depressed, can all be associated with ongoing hyperglycaemia. Students have also told me how they often feel moody and irritable when their blood sugars are high. These symptoms have social implications, in regard to how they are seen by students and teachers alike and can have a negative impact on social interaction.

Difficulty with visual focusing and blurred vision is an acute complication that has significant negative consequences in the classroom. Moving the student with hyperglycaemia nearer to the blackboard may not be helpful. The acute complications and symptoms of hyperglycaemia contribute to a general feeling of unwellness and it is my experience that this translates into absenteeism and/or academic underachievement.

Long-term Complications of Diabetes Mellitus

Although, there are many long-term or chronic complications associated with diabetes, the section will focus on three major areas of concern, diabetic eye disease (retinopathy), diabetic kidney disease (nephropathy), and nerve damage caused by diabetes (neuropathy). While these serious complications of diabetes will only briefly be addressed in this paper, they cause immense concern in individuals with diabetes and health care professionals.

Diabetic Eye Disease

Diabetic eye disease is among the most feared long-term complications of diabetes. It is the most common cause of blindness in adults aged 20-74 years, according to the Wisconsin Epidemiologic Study of Diabetic Retinopathy. This study also found that 3.6% of patients with Type I diabetes and 1.5% of patients with Type II diabetes were legally blind. In the Type I group, 85% of blindness was attributable to diabetic retinopathy, while in the Type II group, one third was found to be attributable to the disease. It is further noted that prevalence of retinopathy is strongly related to the duration of diabetes. After twenty years, nearly all patients with Type I diabetes and more than 50% of patients with Type II diabetes had some degree of retinopathy.

Retinopathy is a major cause of visual impairment in people with diabetes. The retina is a photographic plate that transmits visual images to the brain. In order for images to focus properly on the retina, however, they must pass through the cornea, the pupil, the lens and the vitreous. If the image passes successfully through these systems, it will need to hit on a specific area of the retina, called the macula, in order to ensure focused vision.

The first sign of diabetes negatively affecting the eye is called background retinopathy. It usually starts 5-10 years after diagnosis. It is extremely common in people with diabetes and in itself is usually not dangerous. Background retinopathy is seen by ophthalmologists as red spots on the retina called micro aneurysm, larger spots called micro-haemorrhages or yellow deposits called exudates. When this originates, it is not a major immediate concern, but instead an indication that it should be watched closely and that good diabetes management must be

followed so that it does not progress to the next stage which is labelled proliferative retinopathy. In this stage, tiny new blood vessels form in the eye which are fragile and can easily haemorrhage, causing bleeding into the vitreous and therefore causing loss of vision in the eye, as images cannot be transmitted to the macula. Over a period of weeks or months, the haemorrhaged blood will often drain from the eye and vision will return. Bleeding can reoccur, however; scars and blood clots can form in the eye and this process can cause retinal detachment. Without surgical intervention and good diabetic management, permanent visual impairment and blindness can follow.

On a positive note, it should be emphasized that with recent advances in laser surgery techniques and early detection of diabetes retinopathy, visual impairments can be greatly reduced or prevented.

Diabetic Kidney Disease (Nephropathy)

A major role of the kidneys is to clean the blood of waste products as it flows through this organ, which consists of millions of tiny filters called nephrons. Another important role is to regulate the salt and water balance in the body. By controlling water and salt content and by helping to rid the body of excess liquids, well functioning kidneys maintain appropriate fluid levels (hydration) and blood pressure.

Kidney damage, due to diabetes, is classified, as is diabetic eye disease, as a micro vascular or small blood vessel complication of diabetes. In diabetic nephropathy, the nephrons

develop thickened borders and in end stage renal disease (ESRD) eventually clog up completely. The first indication that the kidney's filtering system is ineffective and that nephropathy is developing is the appearance of protein in the urine (proteinuria).

There are five stages in diabetic nephropathy, the fifth stage being called end stage renal disease whereupon the patient needs kidney dialysis to survive. Although many conditions may cause kidney failure, a disproportionate share of people with diabetes have EDRS. Although approximately 3% of the population have diabetes, nearly 50% of patients starting dialysis have renal failure due to diabetes (Davidson, 1998).

Although this is a serious complication of diabetes, less than one third of people with IDDM never show signs of kidney involvement (Saudek, 1997). The figure is similar for Type II diabetes.

The Diabetes Control and Complications Trial (1993) has shown that the rate of development and progression of diabetic nephropathy is related to glycemic control. However, many IDDM patients develop proteinuria and kidney problems in spite of strict glycemic control (Pickup, 1997). According to Pickup, it appears that although hyperglycaemia may be necessary to cause diabetic renal damage, other factors are evidently needed for nephropathy to develop such as genetic predisposition, hypertension, etc.

Diabetic Neuropathy

Diabetic neuropathy is another long-term complication of diabetes, especially if the disease is poorly controlled. In simple terms, diabetic neuropathy is nerve damage which is associated with or caused by diabetes. This section will discuss the two major types of diabetic neuropathy: peripheral neuropathy and autonomic neuropathy.

Peripheral Neuropathy

Peripheral neuropathy is the earliest, most widely recognized and most common form of diabetic neuropathy (Davidson, 1998). This type of neuropathy occurs in IDDM, NIDDM as well as secondary forms of diabetes mellitus, the overall prevalence being 25-35% (Young, 1993). Twenty percent of newly diagnosed Type II patients have peripheral neuropathy (Davidson, 1998), while 1-2% of newly diagnosed Type I patients are affected (Pickup, 1997). People with IDDM, seem to be at particular risk, however, as it has been reported by Fraser (1977) that the prevalence increases from 4% after one year to 28% after five years.

Peripheral neuropathy is a generalized sensorimotor neuropathy of gradual onset and is usually progressive. The legs are almost always affected before the hands. Since gradual nerve damage first affects nerves with the longest axons (Saudek, 1997), it usually targets areas of the body furthest from the spine - feet and toes. Symptoms usually begin with numbness and tingling of the toes, progressing gradually, over a period of years to the ankle and leg. Some people may complain of pain, over sensitivity or numbness in their toes or soles of the feet. When the peripheral neuropathy is severe, people experience a great deal of discomfort or

numbness. When decreased sensation is experienced, the foot can be injured, even without the person knowing, causing ulceration and infections. The combination of ulceration and poor circulation can cause serious consequences. According to Davidson (1998), 50-75% of total lower extremity non-traumatic amputations in the United States are in diabetic patients with peripheral neuropathy.

Autonomic Neuropathy

As with peripheral neuropathy, nerve damage caused by diabetes and long standing hyperglycaemia can negatively affect the nerves that carry messages to the autonomic nervous system. The autonomic nervous system controls functions such as heart rate, bowel function, breathing, body temperature and in men erectile function. According to Davidson (1998), signs and symptoms of autonomic neuropathy almost always appear after peripheral neuropathy is established.

While this paper will not discuss at length the specific complications involved in autonomic neuropathy. If we consider the many different functions controlled by autonomic nerves it is not surprising that many problems can arise from autonomic neuropathy. Common problems experienced are: 1) stomach involvement (gastro paresis), 2) intestinal involvement (enteropathy), 3) urinary retention, 4) sweating abnormalities, and 5) impotence.

The epidemiology of autonomic neuropathy has not been studied systematically on a large scale (Pickup, 1997). He does state however, that patients with IDDM appear to be at

higher risk and that this risk rises with lengthening duration of diabetes.

IDDM and Lifestyle Implications

Aside from ongoing life-long adaptations people must make in order to successfully manage their diabetes on a daily basis, there are numerous lifestyle implications of this disease placing limitations on quality of life as well. This section will discuss diabetes and its implications regarding driving motor vehicles, employment and obtaining insurance. Further discussion will also centre around the use of tobacco and alcohol.

Driving

The main problems for drivers with IDDM is hypoglycaemia and visual impairment resulting from cataracts or retinopathy. Hypoglycaemia during driving can cause visual changes which in turn can cause poor steering, increased swerving, poor road positioning and compensatory slow driving (Cox, 1993). Despite these problems, however, no increase was found in accident rates of diabetic drivers (Songer, 1988).

In most developed countries, including Canada and the United States, diabetic drivers must declare their diabetes to the regulatory authority. In the UK, a drivers licence is issued for a maximum of three years but can be renewed following completion of a medical questionnaire. Drivers with insulin treated diabetes in Canada and the United States, as well as many other countries have limitations imposed upon them in regard to vocational licences. In many countries, drivers who have insulin treated diabetes are disqualified from being professional

truck drivers. In the UK, they are also disqualified from operating passenger vehicles with more than 17 seats (Pickup, 1997). This legislation can restrict employment prospects in the transportation industry as well as associated occupations such as repairing or testing large commercial vehicles.

In Canada, however, people with diabetes are not disqualified as professional truck drivers or heavy equipment operators. All drivers with diabetes, must however, be given medical clearance every three years.

Employment

The risk of acute hypoglycaemia is the main potential problem that limits people with diabetes in regard to employment and career choices (Pickup, 1997). An individual is generally restricted from employment in a particular area, if hypoglycaemia poses a risk to the worker, to his or her colleagues or the general public. People with IDDM are not usually permitted to work alone in isolated or dangerous areas or at unprotected heights. In the UK for example, people with IDDM or insulin treated diabetes are disqualified from careers in civil aviation, emergency services (police force and armed forces, etc.), offshore oil-rig work, as well as occupations requiring one to work at heights.

According to the Canadian Diabetes Association, Canadian residents are not automatically disqualified from the above stated career choices. Limitations are only put in place on an individualized basis due to related medical problems such as visual impairment, etc.

Another consideration is the employability of people with diabetes. In the United States, Songer (1989), found that reduced employment in workers with IDDM was seven times higher than a non-diabetic sibling group and was principally due to diabetic complications. This writer is unable to find similar data in the Canadian population. Discrimination may play a factor in hiring practices according to Songer. Job applicants who told their prospective employers of their diabetes were more likely to be refused employment as compared to non-diabetic siblings or individuals who did not declare that they had diabetes.

Despite significant medical and technological advances made in managing diabetes, discriminations in employment against people with diabetes still occurs. Often this discrimination is based on misinformation or lack of up-to-date information or knowledge about diabetes. It is likely that the greatest concern is that hypoglycemia will cause sudden unexpected incapacitation. .

In summary, diabetes, especially IDDM can have significant consequences and implications in regard to career choices and employment. It is hoped that with further education and continued research, the negative impact of inappropriate discrimination and limitations in career opportunities will be reduced.

Insurance

Diabetes constitutes a medical risk that prospective insurers assess before entering into contractual agreements with people who have diabetes. Several factors are considered when

underwriting life and health insurance policies, including the duration and severity of the diabetes as well as the co-existence of diabetic complication and concurrent medical disorders (Pickup, 1997). There does not appear to be any standardized guidelines; some companies will not accept applicants with diabetes on any terms, while others will with financial penalty or with particular restrictions. According to Pickup, life insurance premiums are unaffected if agreed upon before diabetes is diagnosed, but if accepted, people with diabetes can expect premiums to be increased by 10-40%, even if they are free of complications. He also states that although traffic accidents are no more frequent amongst people with diabetes, many companies quote higher premiums.

Smoking

It is well known that smoking is a major cause of ill health and premature death. Coronary heart disease has been shown to be eight times more frequent in people with diabetes overall and the risk of myocardial infarction was increased in those diabetics who smoked more than 15 cigarettes per day (Willet, 1987). Muhlhauser (1986) implicates smoking with diabetic complications such as retinopathy, nephropathy and cardiovascular problems. Mykkanen (1992) contends that 35% of those with IDDM die from cardiovascular problems, while Pickup (1997) says that mortality rates are over twice as high for people with diabetes as compared to non-diabetic non smokers. Although more research may be worthwhile in regard to IDDM and smoking, it is quite clear that smoking enhances complications and mortality inflicted by diabetes.

Alcohol

Research on the consequences of alcohol consumption in people with diabetes is quite limited. Alcohol seems to impede the recovery from insulin induced hypoglycaemia but this has not been proven (Pickup, 1997). In a study by Walsh (1974), alcohol was thought to be a contributing factor in 19% of hypoglycaemic episodes but no new research is available at present. The incidence of retinopathy and neuropathy may be associated with excessive alcohol consumption but very little research is available and no recent studies have been published. It is therefore, quite evident that alcohol and its effect on diabetes is a topic that requires much study. At present it is generally agreed that people with diabetes should follow the general guidelines recommended for the non-diabetic population in regard to daily intake of alcohol. They should avoid, however, sweet wines, liqueurs and sweetened mixers.

IDDM - Research and Future Care

Diabetes Control and Complications Trial

Results found recently in the Diabetes Control and Complications Trial (DCCT) have significant and important implications for present and future care of people with IDDM. This landmark study was designed to compare the impact of intensive and conventional diabetes therapy on the progression of micro vascular and neuropathic complications of IDDM (Diabetes Control and Complications Trial, 1993).

Conventional therapy consisted of one or two daily insulin injections, daily self-monitoring of blood glucose levels, clinic visits every three months and a comprehensive programme of

education about diabetes. It did not include or encourage daily adjustments in the insulin dose in response to self-motivating data. Intensive therapy involved the administration of insulin three or more times daily, self-monitoring of blood glucose four times daily, weekly telephone calls and monthly clinic visits. Insulin dosage was adjusted according to self-monitoring results.

The DCCT was terminated after an average follow-up of 6.5 years. The results of the large study which had a sample of 1,441 patients and 9,300 patient-years of follow-up may be extremely beneficial to people with IDDM.

The two treatment groups had similar results until approximately three years, when the cumulative incidence curves of complications began to separate. After five years of treatment, the cumulative incidence of retinopathy was approximately 50% less in the intensive therapy group. Intensive therapy reduced the risk of retinopathy by 76% and the reduction of risk became more pronounced with time. Intensive therapy reduced the appearance of neuropathy at 5 years by 69% and also decreased the risk of nephropathy by 34%.

Research in the prevention and cure of IDDM

Others areas of research in diabetes are ongoing and may be instrumental in the management and eventual prevention and cure of IDDM. Genetic research is ongoing and always striving to isolate genes that make people susceptible to diabetes. We know that the concordance rate among monozygotic twins is 35-50%, and therefore a strong genetic association. The problem, however, is that it is almost certain that there is no single gene

responsible for causing IDDM or susceptibility to the disease. Ongoing research may, however, provide new insights, prevention or cure for the disease.

Research into the immune processes causing IDDM is a very important area of study today. Although immunology as a science has progressed rapidly, it is fair to say that the autoimmune process that attacks the pancreas in IDDM is poorly understood (Saudek, 1997). If the cause of the autoimmune attack was found or if a particular antibody was identified as the offender, a blocking antibody may be developed to neutralize this antibody. At present, a large clinical trial, called the Diabetes Prevention Trial, Type I is underway. The purpose of this trial is to screen large numbers of high risk people for IDDM to discover those with positive antibodies who are likely to develop the disease. Particular antibodies have been found to be common in people who are about to get IDDM but are rare in the general population (Saudek, 1997). In these individuals, prevention approaches, such as injecting low dose insulin before they actually acquire the disease is being implemented. One theory is that by doing this, the beta cells are not required to secrete as much insulin, therefore reducing the strain on the cells, which may in turn reduce the possibility of B cell destruction (Pickup, 1997). This promising trial will probably be completed by the turn of the century.

Pancreas transplantation has been the object of extensive research over the last two decades. The process of transplanting a whole pancreas is recommended and reserved only for diabetic patients who, due to renal failure, need kidney transplantation. In this case, a combined kidney-pancreas transplantation is performed. The surgical risks, postoperative risks and the

chance that the pancreas will fail or be rejected is a greater risk than controlling diabetes by traditional methods (Saudek, 1997) and therefore pancreas transplantation is not considered as a routine method or procedure in treating diabetes.

Islet cell transplantation, which would require less extensive surgery has also been an area researched extensively but has only been successfully completed a few times (Pickup, 1997). This procedure involves transplanting non-human cells into the pancreas of people with diabetes. Although barriers to this type of transplantation are formidable and have not met great success, this research has led to new research into the development of artificial pancreas.

New Approaches in Normalizing Blood Sugar

This paper has discussed at length the importance of good maintenance of normal blood sugars in reducing or eliminating the complications caused by diabetes. A huge amount of research has been directed toward investigating ways to help people with IDDM monitor and normalize their blood sugars more effectively.

According to Saudek, new types of insulin are being developed which hopefully will be more reliable and consistent in their absorption. This in turn should translate into better glycemic control. Implantable insulin pumps is another area of intensive research. Roughly the size of a hockey puck, these pumps are surgically inserted under the skin of the abdomen. The pump is electronically controlled by an external radiotelemetry device, similar to a television remote control. Between meals and overnight, the pump delivers a basal rate of insulin

continuously and before each meal the person tests the blood glucose and sends a signal to the pump to deliver the required amount of insulin. Therefore insulin injection by needle is unnecessary. It is expected that implanted insulin pumps will be generally available in the very near future. The next major step would be to link such a device with an automatic measure of the blood glucose so that the correct amount of insulin could be released automatically throughout the day. The problem, however, is that technology has not been developed to continuously monitor blood glucose without a constant blood withdrawal system. Research into the development and perfection of alternate procedures of glucose sensing and monitoring are ongoing however, with very encouraging results. One particular development is the introduction of a near infrared light beam that will yield a spectrum that may be analysed to determine blood glucose level (Saudek, 1997). This technique may be available in the very near future and will therefore make obsolete the practice of pricking one's finger up to four times a day to extract blood in order to monitor blood glucose.

Psychosocial research which involves the study of many areas such as family functioning and peer relationships is also a very important part of the general effort of helping individuals with IDDM in the proper maintenance of diabetes. This area will not be addressed at this time but will be addressed at length in the next section of this paper folio.

SUMMARY

To this point, this paper has provided information on insulin dependent diabetes mellitus along with descriptions of other forms of diabetes. Prevalence and incidence of the disease was discussed as well as associated complications of the disease. Further consideration was given to lifestyle implications for people with IDDM. This area will be further addressed in the next two sections of this paper with particular emphasis on adolescents.

Recent developments in research for the prevention, cure and management of diabetes was also discussed and how this may positively affect the quality of life for people with IDDM. It seems abundantly clear that extensive research is presently ongoing in the area of diabetes mellitus with very promising implications.

This writer's intention is to offer educational personnel a medical background in diabetes. Hopefully this paper will be of value to educators striving to meet the needs of students with IDDM and their families.

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IDDM: EDUCATIONAL AND FAMILY IMPLICATIONS

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PART ONE

INTRODUCTION

Insulin dependent diabetes mellitus (IDDM) is a chronic condition requiring a lifelong process of compliance with health regimens. Lack of compliance is a major problem in adolescents with diabetes, with up to 50% not complying with care regimens (Rapoff, 1991). Adolescents appear to be less compliant than younger children because adolescents tend to strive to distance themselves from parents and authority figures in order to develop independence (LaGreca, 1994). This need for independence may lead to problems with compliance since neglect of care may be used as a way of asserting independence and resisting the authority of parents and health care professionals.

Managing IDDM is a difficult task at any age, but particularly in adolescence. Throughout childhood, satisfactory diabetes management is often maintained by well-structured home and school environments and regular clinical supervision. This well-maintained structure may diminish, however, as the child enters adolescence and spends more time with peers and less time under the watchful eye of parents and teachers. Often the control of the diabetic regimen may be abruptly shifted into the hands of an adolescent who may not be ready to assume and handle such a responsibility. In order to maintain good metabolic control, the adolescent would be responsible for balancing insulin dosage, diet, and exercise as well as the emotional changes that accompany adolescence. Diabetic management is a formidable task for most adults and therefore extremely complicated for adolescents, who by their very nature, experience many physical, social and psychological changes.

In this paper we will review the literature in regard to psychosocial variables and their relationship with diabetes management during adolescence. Although psychosocial variables may not directly affect metabolic control, they may affect health care adherence, which in turn affects metabolic control. This section will discuss a most important psychosocial domain, family functioning, and its relationship with the management of diabetes during adolescence.

IDDM and the Family

Management of insulin-dependent diabetes mellitus places a large burden on adolescents and their families who must integrate its demands into their daily lives (Travis, 1987). These demands include daily multiple injections and blood glucose tests, a prescribed diet, daily exercise, as well as attending to acute problems of high and low blood glucose levels. These pervasive demands require extraordinary family co-operation and communication on a consistent basis in order to safeguard against the long-term consequences of IDDM.

There are features of IDDM that are especially important in respect to their impacts upon, and likely disruption of family life (Hauser, 1993). For example, the fact that dietary control is a basic aspect of diabetes management means that a key family ritual, meals, may be disrupted by

this illness in the family. Ordinarily in adolescence, parental involvement in dietary control, personal hygiene and bodily care diminishes (Hauser, 1993) but with the advent of diabetes, parental involvement in the affairs of the adolescent's body is often heightened. Often there is the parental necessity to monitor the adolescent's metabolic control, and therefore testing or monitoring urine and/or blood glucose one or more times per day. A conscientious parent may also monitor their teenagers' physical activity outside the home to be sure he or she is getting enough exercise. Complicating matters further, the parent will often, with greater vigilance than usual, enforce restrictions regarding alcohol, tobacco and fast foods (Robin, 1989). During adolescence, independence is a salient concern. Adolescents with diabetes must negotiate through the normal developmental tasks with the additional burden of parental involvement in the daily management of their diabetes (Safyer, 1993), which may be viewed as stifling this independence.

According to Wertlieb (1991), family environment is one of the most influential factors which affect teenagers' adjustment to diabetes. Two lines of work can be distinguished in research on family functioning and insulin dependent diabetes mellitus. One area of study highlights the ways that the family may influence the adjustment and metabolic control of the diabetic adolescent. The other area of study investigates the impacts of the child's or adolescent's diabetes on the relationship between parents, the relationship of parent and child, and overall aspects of family functioning.

In this paper, emphasis will be placed upon family functioning and its influence on the adolescents adjustment to IDDM. This writer does acknowledge, however, that these two areas of research are interrelated and therefore the impact of diabetes on the family unit will be briefly discussed.

A number of studies have examined the relationship between diabetes control and family cohesion and the emotional bond that family members have with one another (Anderson et al, 1981). In Anderson's study, adolescents in good control reported more cohesion and less conflict among family members; parents encouraged them to behave more independently, to express their feelings directly and to act openly. Adolescents in good control described family members as more committed, helpful and supportive of each other than did adolescents in fair or poor control. In contrast, adolescents in poor control reported that they were treated differently than their siblings and that family members were critical, distrustful or indifferent about their diabetes management.

Other studies by Anderson (1990) and Hauser (1993) provide support for the association between family cohesion and good metabolic control of adolescents with IDDM, although some evidence may be attenuated by disease duration. For example, Hanson (1989), found that despite age, the longer the adolescent had IDDM the weaker the association between family cohesion and disease control. This finding may suggest that the family environment may be particularly important as the adolescent first learns to deal with diabetes. Early adjustment also

has been found to be predictive of regimen adherence in later adolescence, while families who are supportive early are more likely to assist the teenager to attain good control without interfering with the normal developmental process of adolescence.

Kovacs et al (1993), studied the influence of family support on adherence across nine years of adolescence but failed to find any relationship between baseline support characteristics and later regimen adherence. According to Hauser (1993) this is not surprising from a developmental perspective, as effective social support for a 12 year old adolescent is qualitatively different from effective support for an older adolescent.

Hauser et al (1993), focused on two aspects of family environment; cohesion and organization. He defines cohesion as commitment, help and support that family members provide for one another. Organization is the structure and planning of family activities and responsibilities. Hauser's findings reconfirmed his previous results (Hauser et al, 1989), which suggests that successful initial adaptation to diabetes depends upon a family environment that is highly cohesive and organized. In pre-adolescent children, diabetes adjustment was more strongly associated with family cohesion, in contrast with adolescents. These results suggest the possibility that younger children benefit from close parental relationships with respect to adjustment to their chronic illness.

It appears that in seeking greater independence from families, adolescents with diabetes

distance themselves from specific facets of the family environment (e.g. cohesion) but continue to be receptive to and benefit from effective family organization (Hauser, 1993).

In a four year longitudinal study Hauser et al (1990), found that cohesion, organization and family conflict were strongly associated with adherence levels in children and adolescents, one year after diagnosis. The strongest prediction of longer term adherence was family conflict. Findings in this study are consistent with previous studies, (Hanson et al, 1987), (Hauser et al, 1990) which cite the importance of family support for the favourable adjustment and adherence of children and adolescents with diabetes. Family support was found to be positively correlated with both short and long-term adherence to diabetic regimen.

A specific finding of this study is that family conflict adversely influences adherence. When more conflict was perceived by parents and their children, this was associated with lower levels of adherence. Miller-Johnson et al (1994) found similar results, in that both parents and child ratings of conflict were consistently related to diabetes management and adherence. This consistent finding has also been noted by others, such as (Anderson et al, 1980) and (Wysocki, 1992). When conflict does develop, it places a child (Miller-Johnson, 1994) at risk of poor diabetic management and poor adherence. This may provide some explanation why management of diabetes becomes less adequate during adolescence, when increased parent-child conflict is common (Emery, 1992).

Lorenz and Wysocki (1991) concluded that aspects of family functioning such as conflict, communication, problem solving and cohesion are important modulators of the efficacy or IDDM treatment. Their most consistent finding was that family conflict is associated with poor diabetic control. According to Wysocki (1992) family conflict takes multiple forms such as marital discord, disputes with extended family, sibling rivalry and teen parent arguments and it is unlikely that these would be equally potent in disrupting treatment adherence or diabetic control. A more detailed examination of specific types or sources of family conflict may yield data that could lead more directly to the development of pertinent interventions (Wysocki, 1992).

Robin and Foster (1989) characterize teen-parent conflict as a developmentally normal manifestation of the clash between adolescents' need for autonomy and the parent's needs to maintain family stability and control. They hypothesize that parent-adolescent conflict is a function of three factors:

1. The family's skills in problem solving communication and their ability to negotiate conflict situations (e.g. Arguments about a teenagers choice of friends).
2. The extent to which family members hold counterproductive or irrational beliefs about one another's behaviour and motives. (e.g., An adolescents inference that her parents do not want her/him to have friends)
3. The degree to which the family is characterized by functional or structural anomalies that subvert normal lines of authority or influence within the family. (e.g. an adolescents' involvement in the parent's marital problems).

Wysocki concludes that the previously mentioned conflicts occur very frequently in

diabetic and non-diabetic adolescents, often surrounding mundane daily events such as homework, chores and clothing, etc. Since treatment of IDDM requires many daily routines such as injections, blood glucose tests and diet, it would not be surprising if adolescents with IDDM have even more frequent and/or intense conflicts with parents. As a reaction to anxiety and conflict, families may develop counterproductive attitudes and beliefs that could obstruct communication about IDDM. Anxiety over short and long-term effects and complications of diabetes may predispose parents to exaggerated reactions to minor fluctuations in diabetic control or behavioural transgressions by the adolescent (Wysocki, 1992).

According to Wysocki, adaptation to IDDM may also affect family structure and role functions. Management of IDDM may impose a burden on families that require extraordinary co-operation and planning. It often forces parents to be involved in aspects of their teenagers' lives where in normal circumstances they would not. It, therefore, would not be surprising that the demands of IDDM negatively impacts on family functioning which in turn may negatively influence an adolescents' adherence and diabetic management.

Although there is much evidence for the relationship between family support and regimen adherence, it is important to note that most research has been conducted with white middle class adolescents from intact families. It is therefore, questionable whether present findings can be generalized to single parent families or minority groups.

It is reasonable to conclude that family environment and functioning is significant in the treatment and management of diabetes in adolescents. There is evidence that families with high cohesion, organization and low conflict are positively co-related with better adolescent adherence and diabetic control. The research discussed in this section has implications for teachers and school counsellors, which will be addressed in the next paper in this paper folio.

PART TWO

INTRODUCTION

Insulin-dependent diabetes mellitus (IDDM) is the most common endocrine/metabolic disorder of childhood, affecting 1 in 500 children under the age of 18. IDDM results from a deficiency of endogenous insulin, a hormone produced by the beta cells of the pancreas, which is essential for glucose utilization by the body's major organs, including the brain. An exogenous supply of insulin is essential to maintain proper metabolic control. Exogenous insulin doses, which are typically injected two to four times per day, are large amounts of hormone that do not mimic the minute and continuous insulin secretions of a normally functioning pancreas (Sperling, 1990). It is not uncommon then, for a child with diabetes to experience wide fluctuations in blood glucose levels during the day in response to the timing, type and dose of insulin administration, the type and amount of food ingested, and the amount of exercise (Sperling, 1990). There is recent evidence that even stress can affect blood glucose levels (Gonder-Frederick et al, 1990). States of relative glucose depletion (i.e., hypoglycaemia) and glucose excess (i.e. hyperglycaemia) often occur routinely within the course of a day for individuals with diabetes, compared to the relatively stable glucose concentrations of persons without diabetes whose levels remain in the normal glycemic range.

Because the brain and other neural structures are critically dependent upon glucose as fuel for normal neural functioning, large imbalances in glucose which occurs in hyper or hypoglycaemia can have physiologically and neurologically adverse effects (Gonder-Frederick et al, 1990).

Evidence of disrupted central nervous system functioning has been found in diabetic children with electrophysiological studies. Eeg-Olofsson and Petersen (1966) studied children between the ages of two and 15 years and detected abnormal electroencephalograms (EEG's) in 35% of the diabetic sample versus 13% of the nondiabetic children studied. There is evidence that abnormal EEGs may result from both severe hyperglycaemia and recurrent hypoglycaemia. (Tsalkian et al, 1990) reported an increase in the incidence of abnormal EEGs in children who exhibited severe hyperglycaemia resulting in diabetic ketoacidosis (DKA), which is also characterized by electrolyte imbalance (i.e. a disruption in the sodium/potassium balance of the body) and acidosis. Although many of the EEGs returned to normal after stabilization of metabolic status, many remained abnormal. In addition to hyperglycaemia, recurrent acute hypoglycaemia also has been shown to cause a slowing of EEGs and degeneration of ganglion cells (Haumont et al, 1979). EEG evidence of long-term neurophysiological disruption may be due to demyelination from hyper or hypo-glycaemia (Vlassara, Brownlee, & Cerami, 1983).

Besides the neurophysiological effects of glucose imbalances, cognitive functioning is also affected in diabetes. Decrements in decision making ability, sustained attention and mental flexibility have been observed in studies of induced hypoglycaemia in adults (Blackman et al 1990). Evidence in adults further indicates that decrements in attention and mental efficiency occur at higher blood glucose levels than the onset of physical symptoms (e.g. trembling, dizziness, etc.) that can alert an individual to hypoglycaemia. In other words, cognitive decrements in response to hypoglycaemia precede physical warning signs of impending

hypoglycaemia in diabetic adults, who have been studied more extensively than children.

Studies with diabetic children similarly have found decreases in decision-making efficiency and attention during hypoglycaemia, whether experimentally induced or observed naturalistically in a diabetes summer camp setting (Ryan et al, 1990). Furthermore, these effects were found in diabetic children even at relatively “mild” levels of hypoglycaemia, (Ryan et al, 1990). Such blood glucose levels are fairly common within the course of daily functioning. Ryan et al, also reported a disassociation between hypoglycaemic physical symptoms and cognitive disruption, again suggesting that a critical physiologic warning system may be ineffective in avoiding the impact of cognitive disruption. Of particular note for children who may experience mild, undetected hypoglycaemia in the classroom, Ryan found a delay in the return to normal cognitive functioning for up to an hour after blood glucose levels had returned to the normal glycaemic range. These findings highlight the importance of allowing school-age diabetic children to maintain mid-morning and mid-afternoon dietary snacks to help avoid transient episodes of hypo-glycaemia during classroom instruction.

These hypoglycaemic effects on cognitive performance calls into question whether repeated hypoglycaemic episodes can lead to cumulative or enduring cognitive deficits. Preliminary evidence by Golden et al, (1989) indicates that they may.

IDDM and Intelligence

The question of whether diabetes has chronic effects on cognitive functioning in children has been of concern since the 1920's. Studies of intelligence with samples of diabetic children initially contended that these children do not differ from non-diabetic peers. This conclusion has since been refuted because of biased sampling from private hospitals, and because of over generalization across subgroups of diabetic children (Ack et al, 1961). Ack, et al (1961) were the first investigators to evaluate subgroups of diabetic children, such as those with early age of disease onset, as possibly at risk for lower intellectual functioning. Because children who acquire diabetes early, before five years of age, are still undergoing rapid brain growth and development, it is thought that the intellectual functioning of these children may be particularly vulnerable to glucose disruption during the maturational process. Indeed, Ack, et al (1961) found that children with disease onset before the age of five, obtained Stanford-Binet IQ scores that were 10 points lower on average than children with later disease onset. The scores of children with later onset did not differ from those of controls and no effect of disease duration was found.

Holmes and Richman (1985) subdivided a sample of diabetic children into four Onset x Duration groups to evaluate the interrelation of these disease variables on IQ scores. Diabetic children with early disease onset (less than 7 years) and long disease duration (greater than 5 years), obtained the lowest WISC-R Performance IQ (PIQ) scores compared to the other three onset/duration groups of children. Further, the early onset-long duration group had lower scores

on each of the PIQ subtests, indicating a pervasive finding. Generally, slower responding on the timed subtests which comprise the PIQ scale may account for the lower scores of the early onset-long duration group. Although this group exhibited two disease risk factors, correlations revealed a stronger relation between the age of onset variable and PIQ scores ($r = .34$) than disease duration and PIQ, where there was no statistical relation. Despite lower PIQ scores, the early onset-long duration group had higher Verbal IQ (VIQ) scores that did not differ from the other groups, suggesting a specific impact of early disease onset on non verbal spatial difficulties.

In another study of the cognitive development of newly diagnosed diabetic children, Rovet et al (1990) documented comparable IQ scores for diabetic children and sibling controls on the Griffiths IQ test, the WPPSI, or the WISC-R at study entry and one year later. There was only a trend for the early onset children to have lower PIQ scores at diagnosis, but after one year these scores significantly declined compared to the late onset children. Episodes of ketonuria, defined as high blood glucose levels and ketones in the urine, were associated with poorer spatial abilities over one year. Diabetic ketoacidosis (DKA) at diagnosis, an acute state of severe hyperglycaemia, ketonuria, and acidosis, was marginally related to lower spatial and overall IQ scores one year post diagnosis. No effect of mild or severe hypoglycaemia was found, possibly because these effects may be diminished in the first year of the disease during a "honeymoon period" in which glucose fluctuations are moderated by partial insulin secretion from residual beta cell functioning (Tsalikian, 1990). In contrast to the poorer PIQ's of early onset children, at one year post diagnosis, those with later disease onset had poorer VIQ's than

children with early disease onset. Late disease onset, after the age of 5, coincides with school entry and a time when crystallized cognitive skills are undergoing most rapid growth and development; a period of greater vulnerability (Ryan, 1990).

Lower VIQ's associated with later disease onset may reflect disruption in the frontal and inferior parietal lobes which myelinate later (Rovet et al, 1990). These differential Verbal and Performance IQ findings of up to 10 points are clinically and theoretically important (even though they are in the average range) because such a discrepancy can be indicative of learning problems with a unique pattern of skills related to either early versus late disease onset (Rovet et al, 1990).

IDD and Neuropsychological Skills

In addition to standardized intelligence measures, neuropsychological measures also have been used to study the cognitive performance of diabetic children. Ryan, (1985) found that adolescents with early disease onset (less than 5 years) performed more poorly than either controls or children with later disease onset on five clusters of tests measuring verbal intelligence, visuospatial processes, learning and memory, mental and motor speed, and school achievement. Similar to other studies, children's scores were in the average range, however, these investigators went beyond the group averages and determined that 24% of the early onset group compared to 6% of the late onset group evidenced clinically impaired performance (i.e. two or more standard deviations below normative expectation) on three or more tests. This data

suggested that although mean scores for the diabetic group were in the average range, a substantial proportion of children nevertheless may have experienced difficulties. Regression analyses revealed that age at onset was associated with primarily right hemisphere visuospatial brain skills. In contrast, duration of diabetes best predicted performance on measures of left hemisphere verbal skills, consistent with findings of newly diagnosed children (Rovet et al, 1990).

Using different measures, Rovet, Ehrlich and Hoppe (1988) also found decrements in visuospatial performance in their early onset (less than 4 years) group. On memory measures, Hagen (1990) found that children with early diabetes onset obtained lower Digit Span scores than controls and exhibited deficient use of strategies to organize and recall information. Kovas (1994) also found evidence that diabetic adolescents had poorer working memory on a short-term memory test and poorer verbal associative learning that was one half a standard deviation below local norms, although memory for information in meaningful context was age-appropriate.

Early onset disease effects may relate to a higher risk of hypoglycaemia for younger children with diabetes due to a greater difficulty in controlling their diet, less predictable patterns of exercise, and to their increased sensitivity to insulin (Ternand et al, 1982) Children who acquire diabetes before age five often experience more hypoglycaemic complications than children with later disease onset. For example, Rovet et al (1988) found children with early disease onset to have twice as many episodes of hypoglycaemic convulsions (3 incidents/child)

than a later onset group (1.5 incidents/child). Hagen et al (1990) found 53% of their early onset group had severe hypoglycaemia resulting in either loss of consciousness or requiring hospitalization compared to 13% of a later onset group. In fact, Ryan (1990) observed that the early onset effect described in the literature likely is an artifact of hypoglycaemic complications which are more frequent in younger children and may have a greater impact on the rapidly developing brain.

In contrast, later disease onset and longer disease duration have been associated with reduced left hemisphere verbal skills. Kovas (1992) found diminishing verbal skills with longer disease duration. When the WISC-R Vocabulary subtest was administered to newly diagnosed children every year for up to 8 years post diagnosis, lower Vocabulary scores were found to be significantly associated with longer disease duration ($r = .47$). A follow-up study provided partial support for the hypothesis that memory dysfunction may have mediated diminishing longitudinal scores (Kovas et al, 1994). Diabetic adolescents with better working short-term memory experienced less decline in Vocabulary scores over time. However, given that verbal memory skills are also left hemisphere related, it is unclear if diminished memory is an indicator of the same disruption affecting vocabulary scores. In addition to providing more support of left hemisphere disruption with longer disease duration, these findings may begin to explain the interrelations among affected skills.

IDDM and Academic Achievement

The evidence to date suggests that subgroups of diabetic children with early or late disease onset may be at differential risk of decrements in Performance IQ and/or Verbal IQ, respectively. The occurrence of these cognitive weaknesses and/or deficits place children at an increased risk for school achievement problems. In addition, children with diabetes possess another risk factor because they are absent from school about twice as often as their healthy peers (14 days per year versus 7, on average) (Kovas et al, 1992). In fact, Ryan et al (1985) found that math, reading and spelling differences between diabetic and control children on the WRAT were primarily due to more missed schooling for diabetic children, based upon parental estimates of absences over the preceding three years. Kovas et al (1992) found a relation between grade point averages (GPAs) which declined over the course of eight years, and yearly school absenteeism ($r = .25$), such that children with diabetes who missed more school had lower GPAs.

Although several studies have found that diabetic children have more academic difficulties and missed more schooling than non-diabetic children, there is a disagreement about the role of school absences in mediating this effect. For example, Fowler et al (1985) evaluated California Achievement Test scores in groups of children with ten different chronic illnesses. Diabetic children obtained a mean achievement score around the 45th percentile which was lower than the state-wide average 63rd percentile and lower than the scores of six other disease groups, even though diabetic children had far fewer school absences than many of the illness

groups with higher achievement levels (e.g. chronic bowel disorder and hemophilia). Across all illness groups, school absence was not significantly related to school achievement, unlike the general school population where there is such an association (Kovas et al, 1992). The illness groups with the lowest achievement scores, like diabetes, tended to be those illness groups with central nervous system involvement (i.e., spina bifida and epilepsy), including hypoxia (i.e. cystic fibrosis).

Children with early onset diabetes have been found to perform more poorly on the Wide Range Achievement Test (WRAT) reading test compared to late onset and control children (Ryan, 1985) and on the WRAT Arithmetic test compared to late onset children (Rovet et al, 1988). Hagen et al (1990) found late onset children scored lower on the reading subtest of the Peabody Individual Achievement Test (PIAT) than controls, consistent with lower VIQ estimates for these children (Rovet et al, 1990, Rovet et al, 1988) also found that diabetic males had lower spelling scores than females and that chronically poorer metabolic control, as measured by a glycosylated haemoglobin measure, was related to lower math scores.

Consistent with intelligence test results, the academic achievement of diabetic children is generally in the average range, although there is evidence that a significant number of children experience more school difficulties than these averages would suggest. Specifically Rovet, (1993) found 24% of children with diabetes had received special school services versus 13% of control children. In another study Hagen et al (1990) found 53% of early onset children had received special services compared with 27% of late onset children and 17% of controls.

In order to evaluate the presence of learning disabilities in their sample of diabetic children, Rovet et al (1993) reanalysed their data obtained from 1983 to 1985 and classified children according to the Rourke and Strang (1983) learning disability subtypes. In the Rourke and Strang (1983) procedure, a cutoff score of less than 90 (less than 25th percentile) on a WRAT subtest is used to determine a learning disability, with scores greater than 94 (greater than 34th percentile) used to determine adequate achievement. Using this procedure, 30% of early onset children had a math disability, 22% had a combined reading/math disability, and 44% had no disability compared to 12%, 15% and a no disability rate of 58% for late onset children. Seventeen percent of late onset children also had an isolated reading disability compared to none in the early onset group. Thus, 56% of early onset versus 42% of late onset children would be classified as LD with this technique. These percentages are consistent with the evidence of the greater vulnerability of early onset children to neurocognitive difficulty. Further, significantly more early onset children fell into the Arithmetic Disabled category, consistent with early onset children having more visuospatial problems. However, while the Rourke and Strang (1983) learning disability classification technique has been useful for research purposes, in part because the relatively narrow four point difference between the presence or absence of a learning problem allows most children to be classified with little lost data, its clinical application is probably more limited because of these liberal criteria (Holmes et al, 1985).

Rovet et al (1993) also reanalysed their data, gathered after 1985, with newly diagnosed diabetic children using the same LD classification technique. In this study, children with

learning problems had more disease risk factors present than children without learning problems. Specifically learning problems were associated with more episodes of ketonuria, poorer metabolic control as assessed by glycosylated hemoglobin values, and more episodes of mild asymptomatic hypoglycaemia. Equally important, this second set of data helps to illustrate an emerging trend in the more recent literature of fewer diabetic children exhibiting cognitive difficulties. For example, in the second data set, only 42% of the early onset group were identified as LD compared to 56% in the pre-1985 data. Similarly, for the late onset children, the post-1985 data revealed that only 32% were identified as LD versus 42% in the pre-1985 data. However, regardless of the time period selected, the pattern remains of more early onset children having learning problems than later onset children. Finally, when the early and late onset groups are combined in both Rovet studies, just 35% of the post-1985 sample, versus 50% of the pre-1985 sample, were identified with learning problems, indicating a significant decrease in the incidence of learning problems in children over time from the same research centre.

What could account for a decrease in the incidence of learning problems? According to Rovet (1993), sample based difference may explain the apparent decrease because enrolled children (post-1985 data) only had diabetes three years when classified into LD groups, versus a disease duration of 5.7 years on average for the pre-1985 children. It simply may take longer than three years for the effects of diabetic metabolic abnormalities to become manifest. Alternatively, a new cohort of children with diabetes may exist; a group whose health care providers manage their disease differently to avoid severe hypoglycaemia based upon earlier findings of hypoglycaemia related cognitive impairments (Holmes et al, 1992). In addition to

changes in knowledge over time, this new cohort also has had the advantage of new advances in disease management technology such as lancets and glucometers which facilitate more readily obtained and accurate blood glucose readings to better avoid hypoglycaemia. Only 2% of Rovet et al's (1990) sample experienced severe hypoglycaemic episodes in the first year after diagnosis compared to 45% of their pre-1985 sample (Rovet et al, 1988). This newer, smaller proportion is more in keeping with other sample estimates of severe hypoglycaemia of around 7% (Bergada et al 1989).

IDDM and Gender Differences

With changes in the care of diabetic children to avoid hypoglycaemia, traditional risk factors for learning problems may become more apparent. For example, the learning disability literature indicates boys are more cognitively vulnerable to learning problems, and boys with the added risk factor of a chronic illness that can affect CNS functioning may be more at risk for learning problems than non-diabetic boys or diabetic girls. The impact of diabetes on the different genders has not received much attention, although several studies suggest that an interaction may exist. Holmes (1992) found disease by gender interactions, such that diabetic boys obtained lower Perceptual Organizational and Freedom from Distraction factor scores than control boys. In addition, boys with high risk disease characteristics of early onset or long duration obtained lower factor scores than boys with later onset or short disease duration. No such disease associations were found for the scores of girls, providing further evidence of greater male vulnerability to the effects of diabetes. Forty percent of diabetic boys had either received

special services programming at school or had been retained a grade compared to 16% of diabetic girls or 7% of the control boys. Holmes also found that high SES diabetic boys experienced signs of greater reading disruption than same SES diabetic girls in that they read significantly slower, made more time consuming errors, and made more total deviations from print. With a relatively brief disease duration of 5 years, it is not known if these difficulties might intensify with additional time.

Evidence that males may be more vulnerable to metabolic insult is also provided by another line of research describing the gender effects of experimentally induced hypoglycemia in adults. Gonder-Frederick (1994) found the cognitive and motor performance of men deteriorated twice as much as that of women at mild levels of hypoglycemia. If young boys are similarly more cognitively disadvantaged than girls by hypoglycemia, then greater male sensitivity to metabolic insult in addition to or concomitant with a period of greater maturational vulnerability may be the mechanism(s) that results in diabetic boys experiencing more learning problems than diabetic girls (Holmes et al, 1992).

SUMMARY

Patterns in the cognitive functioning of children with diabetes have been identified based on a growing number of studies. Despite differing methodologies, the following consensus has emerged: children at greatest risk for learning problems are those with early disease onset, and those who have experienced severe fluctuations in metabolic control resulting in either hypoglycaemic seizures or unconsciousness, or diabetic ketoacidosis (DKA). Either of these risk factors may interact with traditional cognitive risk factors such as gender, placing boys at relatively greater risk for learning problems than girls (Holmes, 1992). Children from these high risk groups, particularly those who have multiple risk factors, warrant monitoring of their academic achievement and may require academic or special classroom intervention. Because children with early disease onset experience particular cognitive and metabolic vulnerability compared to later onset children, intensive preschool programming or early intervention programs may be beneficial for children under the age of five (Kovas, 1992). Finally, results suggest that when evaluating children with diabetes, if an abbreviated WISC-R or WISC-III is used, it may be best to avoid prorated Performance of Full Scale IQ scores that rely heavily on the Picture Completion and Picture Arrangement subtests, because initial evidence indicates that these may not act as traditional measures of nonverbal intelligence in diabetic children.

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IDDM: GUIDELINES FOR SCHOOL PERSONNEL

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Introduction

The guidelines offered in this paper were specifically developed for educational personnel, but are considered useful for all people who work with children and youth who have Type 1 diabetes. At this time in Newfoundland, our education system endeavours to develop an ISSP (Individual Student Services Plan) to meet the needs of each child with special needs including those with health needs such as IDDM. An ISSP may be developed, with the input of families, the student, educators, medical care team, as well as community service personnel who work with the child. If the school is going to meet the needs of students with diabetes, it is imperative that educational personnel have the necessary information about IDDM. It is also extremely important for educational personnel to be aware of the student's health care needs and therefore lend support to the student and his/her family. This paper will provide information on the most common health care needs of a student with diabetes. Recommendations and suggestions are provided to assist school personnel to meet the needs of students with diabetes.

As compared to the previous two papers in this paper folio, this paper is written in a less formal style and is intended to be more accessible to readers. The information is often quite general and often cannot be attributed to a particular source or research paper.

General Information

What is Diabetes Mellitus - Type 1?

Diabetes affects approximately 1 out of every 500 children. It is a chronic disease which means that without new research to develop a cure, it lasts a lifetime. With Type 1 diabetes, the pancreas does not produce enough insulin. This form of diabetes is treated by daily injection of insulin, monitoring of blood glucose levels, diet and exercise. Insulin is required by the body to help glucose (sugar) from food to be distributed into all the cells of the body. When a person's pancreas does not produce enough insulin, the cells cannot use all the glucose from the food that is eaten and therefore extra glucose builds up in the blood. The kidneys attempt to expel this extra glucose through the urine but have to produce extra urine to do so. It is this cycle that causes children with diabetes to urinate frequently which in turn causes dehydration and unusual thirst. Frequent urination in itself is not a symptom of diabetes, but an indicator of undiagnosed or poor controlled diabetes.

What are Causes of IDDM?

Although researchers are not certain, it appears that two factors need to be present to cause Type 1 diabetes. First, there needs to be a genetic predisposition towards one inheriting diabetes. Second, there needs to be an environmental trigger to cause the condition. It is believed that a virus is often the trigger but this is not definite. What is clear, however, is that you cannot "catch" diabetes and that it is no one's fault when a person develops the disease.

How are Children Affected by Diabetes?

Because their bodies do not produce any or enough insulin, children with diabetes cannot break down glucose which is necessary to provide energy to all the cells of the body. If the child does not receive insulin, his or her body will attempt to acquire energy from another source. The body begins to break down fats to replace the energy that it should be getting from glucose. The byproducts from breaking down these fats are called ketones and when they reach a high level in the blood, this causes a severe condition called ketoacidosis. Stress or illness in a person with diabetes can increase the tendency toward ketoacidosis. This situation may result in the diabetic child feeling nauseated and vomiting. When a diabetic person is feeling sick because of influenza, infection, colds, etc, it is extremely important for him/her to continue to take insulin in order to prevent ketoacidosis. A person with ketoacidosis needs immediate medical treatment as this condition may lead to diabetic coma, organic damage or even death.

Physical Problems Related to Diabetes

Physical problems related to diabetes can be minimized when blood sugar levels are kept in good control. Changes often occur in people who have had diabetes for many years. These changes include vision problems, changes in touch sensation and changes in blood vessels. In people with poor diabetes control, the problems can occur sooner and cause kidney failure, blindness, heart disease, stroke and amputations due to poor blood circulation. Medical personnel should be contacted regularly and should watch for early signs of these problems. As

noted, good diabetes control can delay or prevent these complications.

Families may be able to help prevent most complications by keeping the child's diabetes in good control. Diabetes control is a balance of diet and stress management combined with insulin and exercise. It must be kept in mind that control of a child's diabetes will vary at different developmental levels, during times of illness, and also because of individual differences.

The potential effects of diabetes on students vary because of several factors including:

- the amount of insulin produced by the body varies. This varies between individuals.
- the response to insulin in the body varies
- the different activity levels of children
- psychological and social stresses experienced, and
- children's individual response to stress

Team Approach to Care for Children and Adolescents with Diabetes

As mentioned earlier it is very important for the family, health care providers and school personnel to work together as a team in order to co-ordinate a treatment plan for students with diabetes. In the past, I had worked as an educational consultant with the diabetes team at the Janeway Child Health Centre. Our clinic functioned as a family centred health care team where all members, including school personnel, took an active part in planning health care for the child

or adolescent. It is my hope that in the near future all students with diabetes have a faculty member designated as a part of this team. This faculty member, whether he or she be a teacher, school nurse, counsellor, etc. can be the liaison for home, diabetic clinic team and the school so that important information and communication can be enhanced.

The team approach discussed in this section may be viewed as the ideal situation where the child with diabetes and his/her family has easy and regular access to all members of the multi-disciplinary team. In many small communities in Canada, all of these services may not be available.

If we take into account classroom time, recess and lunch breaks, extra-curricular activities and homework assignments, at least half of a student's day is related to school activity. Diabetes control is affected by diet, exercise, stress and insulin; all components may be related to school scheduling, activities, academic workload and peer relationships.

Below is information about members of a family centred health care team and their respective tasks. Goals of treatment and recommendations to meet health care and developmental needs are also discussed.

Child or Youth and his or her family -

Tasks:

- To provide personal and family health history,

- To observe and describe symptoms and problems,
- To record blood glucose levels to determine insulin needs,
- To work with team to develop health care plan, and
- To communicate with team about changes in child's condition and effectiveness of treatment.

Family Physician and/or Pediatrician -

Tasks:

- To provide for physical examinations, including vision and hearing checks,
- To monitor child growth and development,
- To check blood glucose levels and urinalysis,
- To monitor glyco saturated haemoglobin or Hb A1C for overall diabetes control,
- To communicate with parents, teachers and other professionals involved in the child's care, and
- To monitor family strengths, emotional well being and stress level.

Pediatric Endocrinologist -

Tasks:

- To diagnose diabetes,
- To help provide control and give examinations, and looking for complications,
- To recommend treatment to the family and family physicians, and
- along with other physicians to make any needed referrals to ophthalmologists, dermatologists, etc.

Diabetes Nurse Educator* -

Tasks:

- To discuss insulin types and help the child and family to adjust insulin dosages,
- To provide information about diabetes supplies such as glucose meters, injectors, etc.
- To suggest how to balance insulin and exercise with diet and stress management, and
- To teach families how to manage diabetes.

*In my experience, the diabetes nurse educator plays an extremely important part on the team and has a great deal of contact with the family, especially when a child's diabetes is in poor control.

Diabetes Nutrition Educator/Dietician -

Tasks:

- To determine adequacy and quality of the child's diet,
- To set guidelines on caloric needs for the child's optimum growth and development,
- To suggest how to balance diet with exercise, and
- To develop a nutrition care plan.

Social Worker/Counsellor/Psychologist -

Tasks:

- To help the family learn to balance family life with the child's care,
- To help the family deal with parenting and behaviour concerns, and
- To provide referral to community resources when necessary,

Education Consultant -

Tasks:

- To monitor academic progress and how this may be related to stress management,
- To communicate with the team how school related concerns such as achievement, peer interaction, behavioural issues may be correlated with diabetes control,
- To liaise between school and the rest of the team in order to facilitate good communication, and
- along with other members of the team, to provide for workshops or information on diabetes to be given to school personnel.

Treatment Goals for Children with Diabetes

Using a family-centred health care team approach to treating children and youth with diabetes can help health and education professionals and the family arrange for a comprehensive treatment plan. The goals of treatment for school-aged children with diabetes include the following:

- normal growth and development of the child,
- prevention of complications of diabetes,

- nearly normal blood glucose levels,
- normal lifestyle including participation in the usual activities of childhood,
- knowledge of diabetes and good self-care skills, and
- good school attendance and adjustment with minimal absenteeism due to diabetes complications.

These goals may be accomplished by:

- careful management by its health care team,
- educating the child, family and personnel working with the child about diabetes,
- monitoring the child's overall physical and mental health and watching for complications,
- providing a balanced and nutritious diet,
- giving appropriate insulin dosages and meals at correct times,
- monitoring blood glucose levels,
- balancing diet and stress management with insulin and exercise,
- encouraging and helping the student develop self-care abilities, and
- providing a consistent and predictable home and school life to make controlling diabetes easier.

Medical Care Guidelines for School Personnel

The management of diabetes in children is quite different from diabetes management in adults. Children usually require two or more insulin injections a day. This may interfere with the school routine, especially if the school day is extended because of extracurricular activities,

etc. While in school, most youngsters will need snacks two or three times per day as well as a consistent lunchtime in order to keep good blood glucose control and to guard against hypoglycaemia (low blood sugar).

Recognition and Treatment of Hypoglycaemia

Hypoglycaemia, also known as low blood sugar or insulin reaction, is the most common medical emergency encountered by school personnel dealing with a child who has diabetes. Most students who have had diabetes for a while or are in upper elementary or junior high can readily recognize the symptoms and will request treatment. Younger children or those with low cognitive levels, attention problems, etc. may not recognize or may ignore these important signs. Hypoglycaemia requires prompt recognition and immediate treatment. The most likely times to suspect low blood sugar would be close to a meal or snack time or following physical activity such as gym, band, recess, etc.

Early, more easily recognized, warning signs of low blood sugar are:

- shakiness and tremors,
- pale appearance,
- rapid and strong pulse,
- sweating, and
- unusual hunger.

More subtle signs of a lack of sugar to the brain are:

- sudden mood swings or inappropriate behaviour,

- drowsiness or sleepiness or inability to concentrate,
- headache,
- confusion, and
- yawning or lethargy.

Severe reactions include:

- loss of consciousness and
- convulsions or seizures.

Treatment

Students should be treated as soon as symptoms are recognized. This should be done in the classroom and without disruption when possible. If a teacher is in doubt as to whether the student is experiencing low blood sugar, it is always best to offer treatment. Low blood sugar may be treated by one of the following:

1. 4-6 oz of fruit juice or soda (not diet),
2. 1 tablespoon of honey or sugar,
3. Administering a glucose tablet. Parents often will provide these to the school.

When symptoms of hypoglycaemia have been detected, the educator should also keep in mind that:

1. If symptoms do not subside in 10 minutes, repeat treatment
2. If a meal or snack is not scheduled within the next 15 minutes, follow initial

treatment with a protein and carbohydrate snack such as milk and crackers with cheese or peanut butter.

3. Never leave a person alone during a low blood sugar reaction.
4. You cannot harm a child with diabetes by giving sugar if the blood sugar is not low. It will only increase the blood sugar for a short period of time.
5. If the child is unable to swallow liquids or food, is unconscious or having a seizure, Glucagon (a prescription medication) may be given through injection by a nurse or trained individual.
6. If Glucagon is given or if a child does not respond to oral treatment or is unconscious, designate a person to call 911 and the child's parent or guardian.
7. Notify parents of all incidents of low blood sugar reaction. The most likely times for incidents are following physical activity or close to meal or snack times. It should not be necessary for parents to come to school for every reaction.

Recognition and Treatment of Diabetic Ketoacidosis

Ketoacidosis is the result of prolonged high blood sugar and insulin deficiency. In contrast to hypoglycaemia, the symptoms have a gradual onset and take hours or days to develop. If ketoacidosis is severe enough and is uncorrected, it can lead to coma or even death. This condition requires prompt medical attention and treatment.

Warning signs of Ketoacidosis are:

- excessive thirst,
- excessive urination,

- dry skin,
- flushed skin,
- very deep rapid breathing,
- breath that smells fruity,
- eyes that appear sunken,
- nausea/vomiting,
- abdominal pain.

Treatment for Ketoacidosis includes:

- contacting parents, and
- call 911 for transportation to a medical facility.

The following table compares the signs and symptoms of insulin reaction (low blood sugar) and symptoms of ketoacidosis (high blood sugar).

	High Blood Sugar	Low Blood Sugar
Onset	Gradual	Sudden
Illness prior to onset	Frequently	No
Fever	Sometimes	No
Appearance	Extremely ill	Very weak
Skin	Dry & Flushed	Moist & Pale
Mouth	Dry	Wet
Thirst	Intense	Absent
Hunger	Absent	Maybe Present
Vomiting	Common	Rare
Fruity Breath	Yes	No
Blood Glucose	High - usually more than 200 mg/dl	Low - less than 70 mg/dl

Suggestions for School Personnel to Enhance School Adjustment

The following suggestions should help school personnel provide for the needs of students with diabetes. Although needs will vary at different developmental levels, the following may be used as general guidelines for all students with diabetes:

1. Meet with parents and students at the beginning of the school year to discuss concerns.
2. Develop a care plan including arrangements for blood glucose testing and/or insulin injections. These arrangements may include:
 - a private space,
 - hand washing facilities,
 - containers for trash and needle disposal,
 - adult support, monitoring or assistance as indicated,
 - material to record results, and
 - food sources with readily absorbable sugar.
3. Notify appropriate school personnel, as well as substitute teachers, about children with diabetes.
4. Because most children with diabetes require snacks during the day, make arrangements for snacks to be taken with the least disruption to the child's school day. It is important that meals and snacks be eaten on time to prevent low blood sugar. Arrangements for the child to bring snacks from home to school should be made during the meeting with parents at the beginning of the school year.

5. Allow minor low blood sugar reactions to be treated in the classroom. This should be done calmly and efficiently. Most children with diabetes are taught at an early age to carry some form of sugar for use in an emergency. Often children recognize the symptoms and prefer to treat themselves unobtrusively. As a backup, however, teachers should have glucose preparations, juice, etc. to treat reactions. Arrangements should be made so all reactions are recorded.
6. Since students with diabetes should avoid foods with a lot of sugar, parties or special events should include foods that a person with diabetes can eat. Use non-food items for rewards or treats for all students, whenever possible.
7. Students with diabetes have no restrictions on physical activity and should be encouraged to participate in sports, physical education, etc. As mentioned earlier, extra snacks may be necessary during this time especially if the activity is before meal times.
8. Some children enjoy demonstrating blood glucose monitoring or insulin injection techniques during health education classes or for science projects. In a unique way, it allows teachers and other children to understand the complexities of living with diabetes and it may also give the student with diabetes a sense of feeling important. The decision to take part in these activities must be an individual one.
9. Above all, it is most important that students with diabetes be treated the same as other students in school.

Consideration for School Counsellors

Insulin dependent diabetes mellitus may present a variety of psycho-educational, physical and psychological concerns for counsellors. It poses many challenges for counsellors who may endeavour to develop and implement a therapeutic intervention program for affected students. Although no specific psychological problems are found in all children with diabetes, the nature of the disorder may lead to adverse psychological reactions (Garner, 1981).

According to Spirito (1989), teenagers with chronic medical problems have a rate of suicidal attempts nearly twice as high as expected per capita. Spirito also states that the initial impact of IDDM, the daily stresses of management and its frequently negative long-term prognosis, may render adolescents with diabetes more vulnerable to suicidal ideation and behaviours. Furthermore, youths with diabetes have knowledge of and ready access, to a potential means of suicide, namely, insulin.

According to Sclar and colleagues (1999), a national survey in the United States, 1990-1995, has shown that the prevalence of depressive illness in people with diabetes is nearly threefold the observed rate in the national average. This finding, although specific to the adult population, is food for thought for counsellors working with diabetic youth. Gold and colleagues (1993), in a 12 year longitudinal study of school aged children aged 8 to 13 years, found that youth newly diagnosed with diabetes had a slightly elevated rate of suicidal ideation.

Those who did attempt suicide, often used diabetes-related methods. Additionally, non compliance with the medical regimen was related to suicidal ideation over the course of IDDM. Suicidal ideation was also found to be related to depressive symptoms experienced at that time.

Two further areas of concern for counsellors are substance abuse and eating disorders in adolescents with IDDM. Gold and colleagues (1993) found that 50% of adolescents aged 12-16 years reported using tobacco or alcohol in their lifetime. In their study, one quarter of diabetic teens were deemed to drink alcohol dangerously. There was a high correlation between teens who use drugs and alcohol and a positive family history of alcohol and substance abuse. Few teenagers perceived alcohol or drug abuse to affect their diabetic control, and the majority believed their control to be good or excellent, thus demonstrating the impact of denial in diabetic adolescent substance abuse.

Eating disorders pose a particularly serious risk to health in young people with insulin dependent diabetes mellitus (Pollock, 1995). Pollock states that the rate of bulimia has been reported as high as 38% and as low as 0% in this population. According to Khan and Montgomery (1996), prevalence studies show that young females with diabetes are at increased risk of developing an eating disorder, particularly bulimia. Insulin omission occurs at a high rate in young females with diabetes, providing them with a unique method of losing weight or purging. Without the administration of insulin, teenagers, male or female, may eat food high in sugar content that will not be metabolized or stored in the body as fat. The teenager finds that he or she initially can eat what they want and actually lose weight. This maladaptive method to

weight management however will ultimately lead to ketoacidosis.

The information provided for counsellors in this section is only a guide to the range of possible problems they may face in their everyday practice. It is appreciated that while the research reviewed is neither comprehensive or exhaustive. Persons studying research in the area of adolescents with diabetes and counselling issues must appreciate the research in this area is quite limited, controversial and inconclusive.

Yousef (1993) provides suggestions for counsellors who are preparing to work with children and youth with IDDM. According to Yousef, this preparation process should involve the following:

1. Become aware through pre and in-service training programs of the wide range of needs encountered by the child with diabetes and his/her family. This may be achieved by having a meeting with members of the diabetic team at your regional hospital.
2. Establish a trusting relationship with the child and family. The counsellor can build such a relationship by: being sensitive to their needs, offering reassurance, encouraging the expression of concerns and fears in a comforting atmosphere, exhibiting genuine interest and caring attitudes, attending to the various stages of the adjustment process (denial, anger, bargaining, depression and acceptance), and reinforcing their appropriate responses.
3. Work within the framework of a multidisciplinary team. The multidisciplinary

team approach has received more emphasis in recent years. Such a team may consist of physicians, nurses, social workers, counsellors, teachers nutritionists and others.

The counsellor's roles include conducting parental conferences, making referrals to available support services, facilitating the functioning of parent self-help groups or special diabetic camps and conducting home visits when needed.

4. Evaluating the diabetic child's emotional status and social maturity. Counsellors should use appropriate methods for evaluating the child's adjustment. When social or emotional problems are observed, the counsellor should consult with parents and significant others to promote social and emotional development. The counsellor can also play a key role in encouraging teachers and peers to maintain a facilitative learning environment for the child with diabetes.
5. Providing early vocational counselling services. The counsellor should identify the characteristics of diabetic children related to future employment skills and assist in vocational preparation and development.
6. Helping the parents organize their home lives. Like other chronic illnesses, diabetes may lead to family disorganization. The counsellor can help by conducting individual and group counselling in order to ease existing concerns, providing adequate reassurance and support, and helping parents understand and cope with problem situations.
7. Encouraging the development of self-regulation on the part of the child with diabetes. The counsellor can also play an important role in maintaining the diabetic child's health and general well-being by enhancing self-management of the disorder and

encouraging treatment adherence. Several behavioural procedures (e.g., reinforcement, contingency contracting and self-monitoring) have proven successful in improving treatment compliance. The counsellor can also contribute by alleviating the psychological discomfort experienced by the child in following the medical regimen (i.e. diet, insulin injections, urine testing).

8. Counsellors may be instrumental in conveying to students that diabetes need not be the focal point of their lives. Students with diabetes can enjoy full, completely normal lives with few, if any, restrictions. A goal of counselling may be to assist the adolescent to view diabetes as one of life's challenges which may have positive spin-offs, such as sense of determination, responsibility and a deep appreciation of life in general.

Conclusion

During my ten years at the Janeway Child Health Centre, I have been blessed with the opportunity to work with many children, youth and their families dealing with this disease. I have also been blessed with the opportunity to work with and learn from so many knowledgeable and caring health professionals. One regret that I have is not researching the area of IDDM more fully during my tenure as an educational consultant to the diabetic team at the Janeway. I realize now that my lack of knowledge was remediated by the fluid interaction and integration of a viable multi-disciplinary team. Based on my past experiences, I would now encourage any counsellor or professional in a position or situation similar to mine to develop a rich background knowledge of this critical area.

I have worked with some adolescents with diabetes who have experienced a number of psychological and family problems. Then again, I have worked with many other adolescents without diabetes probably at a relative morbidity ratio, with the same problems stated above. In fact, I am not aware of any research which shows that people with diabetes have a higher prevalence of psychological, behavioral or family problems. Research to date is inconclusive in regard to the effect of IDDM on adolescent psychological adjustment and educational abilities and achievement. The only research I am aware of that is conclusive and incontrovertible is that insulin induced hypoglycaemia and ketoacidosis is unique to IDDM, and can be dangerous. I have counselled students who have nearly died from these complications. As educators, our foremost task is to ensure the safety of our students and therefore it is necessary for school

personnel to be given in-service training when a student with diabetes is enrolled in school.

Simplicity should rule in this three step plan, as follows, when students with IDDM are enrolled:

1. When a student is diagnosed with diabetes the principal of the school is to be contacted by a diabetes team member.
2. A school representative is designated to meet with a member of the medical clinic, parent and student.
3. Pertinent information from this meeting be disseminated to school staff.
ie. The designated school representative would be the liaison.

In conclusion, it is my hope that the information in this paper folio will be useful to school personnel who are now, or who in the future will work with students with diabetes. A knowledgeable school staff in the area of IDDM undoubtedly will have a very positive impact on the daily lives of students and their families.

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Glossary of Terms

Autoimmunity -

A process in which the body's immune system attacks a part of the body. Cells that produce insulin may be destroyed by autoimmunity.

Beta Cells -

Small groups of cells in the pancreas that produce insulin.

Blood Glucose Level -

A measurement of the amount of sugar in the blood.

Blood Glucose Monitoring -

Checking the level of blood sugar several times each day by placing a drop of blood on a test strip.

Cells -

The building blocks of the body that use glucose for energy.

Chronic condition/disease -

A condition that affects a person for a long time or a lifetime.

Dehydration -

Excessive water loss from the body.

Diabetes Mellitus, Type 1 -

Insulin dependent diabetes: formerly called juvenile onset diabetes. Type 1 always requires insulin therapy.

Genetic Predisposition -

An inherited tendency to develop a condition.

Glucagon -

A hormone produced by the pancreas to raise blood sugar levels. Glucagon can also be given by injection.

Glucose - A common form of sugar that is produced when food is digested.

Glycosylated Haemoglobin (Hb A1C) -

A blood test that gives a good indication of overall diabetes control over the last few months.

Hyperglycaemia -

High blood glucose.

Hypoglycaemia -

Low blood glucose.

Insulin -

A hormone produced by the pancreas to make glucose available to cells of the body.

Insulin Dependent Diabetes Mellitus -

IDDM or Type 1 diabetes.

Insulin Reaction -

Low blood glucose, also called insulin shock and hypoglycaemia.

Ketoacidosis -

A condition where blood glucose and ketone levels are very high.

Ketones -

A waste product of the body that is produced when fat is burned for energy.

Multidisciplinary Team -

A group of professionals from different fields who work together to give specialized services to the family.

Nephropathy -

A complication of diabetes in which nerves are damaged and cause numbness and tingling.

Pancreas - A gland in the abdomen that produces insulin.

Pediatric Endocrinologist - A doctor for children with diabetes or other gland problems.

Retinopathy -

A complication of diabetes in which the blood vessels of the back of the eye are damaged.



