Bradley C, Pierce MB, Hendrieckx C, Riazi A and Barendse S (1998) Diabetes Mellitus. In M Johnston and DW Johnston (Eds) *Health Psychology,* Vol. 8 in AS Bellack and M Hersen (Eds) *Comprehensive Clinical Psychology,* Oxford: Elsevier Science, 277-304.

COMPREHENSIVE CLINICAL PSYCHOLOGY

VOLUME: HEALTH PSYCHOLOGY

CHAPTER 4: DIABETES MELLITUS

CLARE BRADLEY,

Royal Holloway, University of London, Egham Surrey, UK,

MARY B. PIERCE,

Department of Public Health and Primary Care, Charing Cross and Westminster Medical School, 4th Floor, Chelsea and Westminster Hospital, 369 Fulham Road, London SW10 9NH.

CHRISTEL HENDRIECKX

Diabetes Research Center,Vrije Universiteit Brussel, Laarbeeklaan 103, 1090, Brussels, Belgium.

AFSANE RIAZI,

Royal Holloway, University of London, Egham Surrey, UK,

and

SHALLEEN BARENDSE,

Royal Holloway, University of London, Egham Surrey, UK,

4.2.1. Risk Factors in IDDM

4.2.2. Prevention of IDDM

4.2.2.1.Primary prevention

4.2.2.2.Secondary prevention

4.2.2.3.Tertiary prevention

4.3 NIDDM: ETIOLOGY, RISK FACTORS AND PREVENTION

4.3.1. Risk Factors in NIDDM

4.3.2. Prevention of NIDDM

4.3.2.1.Primary prevention

4.3.2.2.Secondary prevention

4.3.2.3.Tertiary prevention

4.4. IMPACT OF THE CONDITION ACROSS THE LIFE SPAN ON PERSON AND FAMILY

4.5. PSYCHOLOGICAL ASPECTS OF SYMPTOMS

4.6. SPECIFIC ISSUES OF ADHERENCE

4.7. PSYCHOLOGICAL INTERVENTIONS IN THE MANAGEMENT OF DIABETES

4.7.1. Blood Glucose Awareness Training (BGAT)

4.7.2. Identification of Stress Reactivity

4.7.2.1.Mechanisms involved in stress and diabetes control

4.7.2.2.Acute stress and blood glucose reactivity

4.7.2.3.Life stress and blood glucose reactivity

4.7.3. Stress Management Training

4.7.4. Monitoring of Psychological Outcomes and Processes

4.7.4.1. Measurement of well-being and satisfaction

4.7.4.2. Measurement of quality of life

4.7.4.3. Measurement of knowledge, beliefs and other cognitions.

4.7.5. Patient Empowerment

4.7.6. Weight Management

4.7.6.1.Eating disorders and withholding of insulin

4.1 INTRODUCTION TO DIABETES MELLITUS

4.1.1 The Conditions

Diabetes Mellitus is not a single disorder but a heterogeneous group of disorders. All forms of diabetes mellitus are characterized by hyperglycemia and disturbances of carbohydrate, fat and protein metabolism which are associated with absolute or relative deficiencies of insulin action and/or insulin secretion. The World Health Organization (WHO) in 1985 developed a now widely accepted classification of the disorder, largely based on clinical characteristics (see table 1) (WHO Study Group on Diabetes Mellitus, 1985).

Insulin is needed to allow glucose to pass from the blood into most of the body cells. Only the cells of the brain and central nervous system can use glucose from the blood in the absence of insulin. Without insulin, most body cells metabolize substances other than glucose for energy. However, fat metabolism in the absence of glucose metabolism, creates ketone bodies which are poisonous and their build up is associated with hyperglycemic coma. In the absence of sufficient insulin, unmetabolized glucose builds up in the blood. Water is drawn from body cells by osmosis to dilute the highly concentrated blood, and is then excreted along with much of the glucose, once the renal threshold for glucose (usually 10mmol/L) is exceeded. Dehydration follows.

The characteristic presenting symptoms of diabetes are fatigue, extreme thirst, excessive urination, unexplained itch, recurrent infections (e.g. boils and thrush) and otherwise unexplained weight loss**.** These symptoms recur if treatment is not adequately controlling the condition.

Complications of diabetes are responsible for considerable morbidity and mortality. The acute complications of diabetes are hypo- and hyperglycemic coma and infections. The chronic complications include microvascular complications such as retinopathy and nephropathy, and the macrovascular complications of heart disease and stroke. Diabetes mellitus is the commonest cause of blindness and of renal failure in the UK and the USA. Other common complications include autonomic and peripheral neuropathy. A combination of vascular and neuropathic disturbances result in a high prevalence of impotence in men with diabetes. Peripheral neuropathy causes lack of sensation in the feet which can cause minor injuries to go unnoticed, become infected and, with circulatory problems obstructing healing, ulceration and gangrene are serious risks and amputation is not uncommon. Recent evidence from metaanalysis of studies of the relationship between glycemic control and microvascular complications (Wang, Lau & Chalmers, 1993), and from the longitudinal multi-centre Diabetes Control and Complications Trial in the USA (DCCT Research Group, 1993), have established a clear relationship between improved blood glucose control and reduction of risk of retinopathy and other microvascular complications in IDDM. It is likely that there would be similar findings for NIDDM though the studies did not include NIDDM patients. However, the DCCT included highly selected, well-motivated, well-educated and well-supported patients, cared for by well-staffed diabetes care teams involving educators and psychologists as well as diabetologists and diabetes specialist nurses.

The challenge of obtaining the goals of the intensively treated patients in the DCCT with the vast majority of people with diabetes given the more limited health care resources typically available in routine practice, is considerable. If diabetes control can be improved without significant damage to quality of life the economic, health and quality of life savings associated with a

reduction in complications in later life will be vast. Although some people who have had poorly controlled diabetes over many years do not develop complications, complications commonly arise after 15-20 years of diabetes and individuals in their 40's or even 30's may develop several complications in rapid succession. However, 15 years ago patients had no way of monitoring their own blood glucose levels at home. Urine glucose monitoring only told them when their blood glucose had exceeded the renal threshold of approximately 10mmol/L (i.e. was far too high), without being able to discriminate between the too high levels of 7-10 mmol/L or the hypoglycemic levels below 4mmol/L. Clinics relied on random blood glucose testing and there were no measures of average blood glucose over a longer period. Now there are measures of glycosylated hemoglobin (GHb or HbA_1 or HbA_{1c}) which indicate average blood glucose over a 6 to 8 week period and measures of glycosylated protein, fructosamine, which indicates average blood glucose over a two week period. Blood-glucose meters for patients were first introduced in the early 1980s and the accuracy and convenience of the meters and the reagent strips they use has improved dramatically since the early models. Now blood-glucose monitoring is part of the daily routine for most people using insulin in developed countries. Blood-glucose monitoring is less often prescribed for tablet- and diet-alone-treated patients, financial reasons probably being allowed to outweigh the educational value of accurate feedback in improving control long term. The reduced risk of hypoglycemia and diabetic keto-acidosis in NIDDM patients not using insulin means that acute crises rarely arise in these patients though their risk of long-term complications is at least as great as in IDDM and might be expected to be reduced if feedback from blood-glucose monitoring were provided.

4.1.2 Insulin Dependent Diabetes Mellitus (IDDM)

Insulin-dependent diabetes mellitus (IDDM), also known as juvenile-onset diabetes, ketosis-prone diabetes, or Type 1 diabetes, is the commonest form of diabetes in children and young adults of European origin. Age at clinical onset of the condition is usually under the age of 30 years.

People who develop IDDM lose the ability to produce insulin altogether. Diagnosis and treatment with exogenous insulin is often followed by a 'honeymoon' period when endogenous insulin production restarts and the exogenous insulin doses required are reduced and, occasionally, can be discontinued. This is, however, always only a temporary respite. Thereafter, exogenous insulin, usually delivered by subcutaneous injection, will be needed continuously throughout the lifetime of a person with IDDM. If insulin therapy is discontinued, even for a few days, ketotic coma will develop and, if not treated, will result in death.

The onset of IDDM is abrupt, with severe thirst, excessive urination and dramatic weight loss. Individuals usually present to the doctor with one or more of these symptoms and an elevated blood glucose level. They will also have glucose, and possibly ketones, in their urine. This combination indicates diabetic keto-acidosis and is a potentially fatal medical emergency. Diagnosis is made clinically and confirmed on the basis of an elevated blood glucose concentration (>6.7mmol/L).

IDDM can be managed by balancing injected insulin, carbohydrate intake, and energy expenditure. The goal of treatment is to maintain blood glucose levels as close to the normal range as possible (i.e. 4-6mmol/L) in order to reduce the risk of chronic complications, while also avoiding the dangers of blood glucose levels falling to hypoglycemic levels. To achieve this goal, people with IDDM will usually need to measure their blood glucose levels several times daily. In the 1980's and early 1990's, two injections a day were commonly used by IDDM patients. However, recent evidence has shown that more intensive therapy, with up to five injections per day, is associated with reduced risk of chronic complications (DCCT Research Group, 1993; Wang et al. 1993) though at the price of three-fold increases in occurrence of severe hypoglycemic episodes (DCCT). Intensive insulin regimens have become more commonly used in the 1990s and this trend has been facilitated by the development of insulin delivery devices such as pen-injectors which simplify the task of administering insulin, and continuing improvements in blood glucose monitoring methods (Bradley, Christie & Home [Eds.], 1991) The dangers of hypoglycemia, which tend to be increased with the intensity of the

insulin therapy, give increasing cause for concern. Severe hypoglycemic episodes cause acute behavior changes with behavior often becoming aggressive, and unless action is taken to increase the blood glucose levels, collapse and coma follow rapidly. Insulin-dependent individuals need to carry with them at all times glucose tablets or some other form of rapidly absorbed glucose so that they can respond to early signs of hypoglycemia by increasing their blood glucose levels and thus prevent a severe hypoglycemic episode.

4.1.3 Non-insulin Dependent Diabetes Mellitus (NIDDM)

Non-insulin-dependent diabetes mellitus (NIDDM), also known as maturity-onset diabetes or Type 2 diabetes, is the commonest form of diabetes world-wide (85% of cases in the developed world). Age at clinical onset of the disorder is usually over the age of 40 years.

People with NIDDM continue to produce insulin, but it is inadequate either in amount or in action to maintain their blood glucose levels in the normal range. With the development of the pathological process involved, patients first have asymptomatic glucose intolerance and then go on to develop diabetes mellitus. Unlike IDDM, the process of development of NIDDM is insidious. Individuals may have NIDDM for years before it is diagnosed.

Diagnosis may result from patients presenting with classical symptoms, or with one or more of the complications of diabetes, or diabetes may be found on routine medical examination (confirmed by oral glucose tolerance testing). Dietary management is the first choice of treatment for NIDDM. A low fat weight-reducing high fiber diet will be recommended to those who are overweight and if weight can be reduced and maintained close to ideal weight it may be the case that no further treatment is required. The diet recommended is usually high in complex carbohydrates and fiber and low in refined carbohydrates in order to avoid rapid absorption of glucose and to maintain blood glucose at a more even level without peaks of hyperglycemia. Reduction in fats may also be recommended (particularly if the individual has abnormal lipid

levels) with a view to lowering and maintaining triglyceride and cholesterol levels and thereby reducing the risks of cardiovascular disease which accompany NIDDM.

If glucose levels cannot be controlled adequately with diet alone, oral hypoglycemic agents are added to treatment. These tablets do not contain insulin which, being a protein, would be digested if taken orally, but rather act to stimulate production of endogenous insulin or to enhance the effectiveness of existing levels of insulin. A variety of oral hypoglycemic drugs are available, such as sulphonylureas and biguanides. These differ in their modes of action and side effects, for example, sulphonylureas may increase the risk of hypoglycemia. If adequate control of blood glucose cannot be achieved with diet and tablets, insulin is likely to be recommended. It is important to recognize that NIDDM patients using insulin are just as prone to severe hypoglycemia as IDDM patients. However, because in NIDDM there is limited endogenous insulin secretion, sufficient to allow some glucose metabolism, ketoacidosis and hyperglycemic coma are unlikely to develop if exogenous insulin is reduced or discontinued.

Although NIDDM is often misguidedly referred to as 'mild diabetes', the risk of chronic complications is no less than that associated with IDDM. People with NIDDM are two or three times as likely to develop cardiovascular disease than people without diabetes and 75% die of cardiovascular disease. The life expectancy of people with NIDDM is, on average, reduced by a decade as a result of the disease. Although the later onset of NIDDM might be expected to reduce the risk of complications which are usually associated with raised blood glucose levels over 15 or more years, the insidious onset of NIDDM delays diagnosis, and chronic hyperglycemia over many years probably accounts for the many cases which are only diagnosed after the onset of complications.

4.2.IDDM: ETIOLOGY, RISK FACTORS AND PREVENTION

IDDM is caused by the autoimmune destruction of insulin-producing beta-cells in the pancreas. The pathological process is probably initiated several years, sometimes even decades, before clinical onset by an interaction of genetic predisposition and environmental triggers (Zimmet, 1995). The incidence of IDDM has been reported to change with age, rising to a peak around puberty (Laakso & Pyörälä, 1985) with a second peak later in life (Krolewski, Warram, Rand & Kahn, 1987). Altogether more cases of IDDM are diagnosed in adulthood rather than in childhood (Vandewalle et al.1993) and, in some studies, a higher incidence has been found in males than in females (Blohmé et al. 1992; Bruno et al.1993). Marked geographical variation in IDDM incidence has been observed, varying from 35.3 per 100,000 inhabitants in Finland to 1.9 per 100,000 in Japan (Karvonen, Tuomilehto, Libman & LaPorte, 1993). Differences in incidence between neighboring countries have led to the assumption that diet and lifestyle are important determinants of the disease (Green, Gale & Patterson, 1992), but differences in the frequency of 'diabetogenes' may also play a role. Over the past 20 years the incidence of IDDM has doubled in Europe (Bingley & Gale, 1989) and is steadily increasing worldwide (Krolewski et al, 1987). However, most of the incidence data are only available for those with early onset before the age of 20 years. Since most cases are diagnosed during adulthood, it may be that the apparent doubling in incidence is due to a shift towards earlier clinical onset of diabetes rather than to an increase in the number of people developing IDDM at some point in their lives (Bingley & Gale).

4.2.1. Risk Factors in IDDM

A genome-wide search has revealed that more than 12 gene loci contribute to genetic susceptibility for IDDM (Todd, 1995). The genetic component of the condition is indicated by the 5% prevalence of IDDM in first degree relatives, compared with less than 1% in the general population (Bingley, Bonifacio & Gale, 1993; Gorus, Anselmo & Keymeulen, 1993), and the higher concordance rate for IDDM in identical than in non-identical twins (Leslie & Elliott, 1994) Nevertheless, 90% of the new cases occur in individuals without a family history of diabetes (Bingley et al.). These individuals, however, also carry certain predisposing genotypes.

Genetic susceptibility is not sufficient to cause diabetes (Karvonen et al. 1993) since a majority of people with disease-associated alleles do not develop IDDM. Environmental agents appear to have an important role. The way in which the environment affects the pathogenesis is still unclear. It has been hypothesized that viral infection (Krolewski et al. 1987; Zimmet, 1995) and proteins in the diet (Gerstein, 1994) are important factors. Psychological stress may also have a role in increasing vulnerability to viral infection or impairing defense mechanisms against such infection, thereby facilitating the progression of the hidden pathological process. Clear evidence for such an effect is difficult to obtain though suggestive evidence is available to support the notion that stress may contribute to the development of IDDM (see Bradley, 1988 for review). There is stronger evidence to suggest that psychological stress can trigger the onset of symptoms in a person in whom islet beta-cell damage is at an advanced stage (Bradley, 1988; Clayer, Bookless-Pratz & Harris, 1985). Blood glucose levels already raised as a consequence of diminishing insulin production can be further elevated by neuroendocrine responses to psychological stress and tipped over the renal threshold to precipitate the overt symptoms of diabetes which would have appeared in due course without the occurrence of the stress.

Current knowledge about the incidence of IDDM has developed as a result of national and regional registries (Karvonen et al. 1993). Standardized data are collected on previous exposure to environmental factors and occurrence of metabolic and genetic markers for new IDDM cases (Gorus et al. 1993). The long-term follow-up of at-risk families allows comparative studies of the cause and pathogenesis of the disease and can help clarify temporal, regional and environmental variations in the incidence of diabetes (Diabetes Epidemiology Research International Group [DERIG],1988).

4.2.2. Prevention of IDDM

Presently, there is no cure for IDDM. Despite evidence that improved blood glucose control reduces the risks of complications and despite the development of increasingly effective insulins and more convenient delivery systems and monitoring devices, the complications of diabetes

continue to present major health problems with massive personal, social and economic costs. The possibilities of preventive strategies are therefore attracting considerable attention.

4.2.2.1. Primary prevention

The aim of primary prevention is to avoid the initiation of the hidden pathological process leading to IDDM. This strategy requires identification of genetically predisposed individuals and protection against avoidable environmental factors (e.g. nutritional or viral). Because of the current lack of specific knowledge about the nature of these triggering factors, large-scale initiatives could lead to treatment of many people who might never develop IDDM (Palmer & McCulloch, 1991). The role of the diabetes registries is crucial in improving understanding of the interaction between genetic and environmental factors. A first attempt at primary prevention has been initiated in Finland (Akerblom et al.1993), where the role of cows' milk proteins in the diet of young children is under investigation.

4.2.2.2. Secondary prevention

The preclinical phase of IDDM is accompanied by humoral, immune and metabolic abnormalities. During the past decade, much knowledge has been accumulated about the diabetes-predictive value of several of these early markers (Dotta, Dionisi, Farilla & DiMario, 1996). Refining the use of these marker combinations would allow early diagnosis of diabetes and offers the possibility of secondary prevention with the aim of halting or delaying the progression of further destruction of the beta-cell mass by pharmacological intervention at an early stage of the pathological process.

The presence of antibodies against various diabetes-associated islet-cell autoantigens is currently considered to be one of the best indicators of a latent autoimmune disease process in the islets (Dotta et al. 1996), although the relation of these antibodies to the pathological changes is presently poorly understood. Data obtained in a representative sample recruited through diabetes registries show that at least one of these immunological markers is present in approximately 90% of the individuals at clinical diabetes onset and also in the vast majority of relatives in the

preclinical stage (Gorus et al.[in press]; Verge et al.1996). The presence of more than one type of autoantibody greatly increases the risk of IDDM (Eisenbarth, Verge, Allen & Rewers, 1993). Metabolic markers can be detected in a later phase of the preclinical stage. A decreased early-insulin response to an intravenous glucose challenge and increased proinsulin levels can indicate impending clinical onset of IDDM. There are now indications that combined presence of these biological markers can also be helpful in identifying preclinical IDDM in the general population (Hagopian et al.1995). The early diagnosis of a hidden disease process now creates the theoretical possibility of influencing the process pharmacologically in such a way that the clinical disease manifestation is prevented. Pilot studies in animal models and on a small scale in humans suggest that such an approach is feasible. Large-scale intervention trials have been initiated with nicotinamide and with insulin. It is expected from preliminary studies that nicotinamide (vitamin B3) could delay the onset of IDDM (Elliott & Chase, 1991; Pozzilli & Andreani, 1993), but that the preventive effect could vary from individual to individual (Herskowitz, Jackson, Soeldner & Eisenbarth, 1989). Multi-centre programs were launched to investigate the effect of this vitamin on the pathological process and on the biological markers in first degree relatives **(**Nordenfelt, 1996). A second approach has been the prophylactic use of insulin to allow the remaining beta-cell mass to rest or to induce tolerance against beta-cell autoantigens. A clinical trial has begun in the USA (Keller, Eisenbarth & Jackson, 1993).

4.2.2.3. Tertiary prevention

Tertiary prevention aims to prevent long-term complications despite the onset of diabetes. One approach has been pancreatic organ transplantation which has been shown to restore normoglycemia and to halt the progression of chronic complications (Robertson, 1992). However, the intervention is by no means a cure, and the required immunotherapy can cause serious unwanted effects, including increased risk of cancers. For these reasons, solitary pancreas transplantation is not a suitable treatment for diabetes. Pancreas transplantation is, however, sometimes performed in combination with a kidney graft in individuals with severe chronic complications. Over the last 20 years, transplantation of insulin producing islets has been studied as an alternative for pancreatic organ transplantation (Pipeleers, Keymeulen & Korbutt, 1994).

Several clinical trials have been initiated worldwide (Sutherland et al.1996). Although many obstacles have yet to be overcome, transplantation of allogeneic (including human to human) or xenogeneic (including pig to human) donor tissue has the potential to treat IDDM in the early stages with a view to avoiding complications (Pipeleers et al).

4.3. NIDDM: ETIOLOGY, RISK FACTORS AND PREVENTION

4.3.1. Risk Factors in NIDDM

NIDDM appears to be a complex multigenic-environmental disorder, with a strong genetic component. Current theories about its pathogenesis implicate intrauterine malnutrition (Hales & Barker, 1992) and a common (as yet little understood) metabolic disorder Syndrome X (Reaven, 1988) which leads in differing circumstances to NIDDM, obesity, hypertension, or hyperuricaemia (gout).

Familial clustering is a characteristic feature of NIDDM. Monozygotic twin studies show a concordance rate of over 90% (Newman et al.1987; Pyke, 1979). The US National Health and Examination Survey (Harris, 1984) showed that in people aged 35 to 74, about 35% of those with a medical history of diabetes had one or both parents with diabetes. Other studies have shown an even greater concordance for NIDDM in sibling pairs than parents and children (Beaty, Neel & Fajans, 1982). The literature on risk of NIDDM in the offspring of affected people has been reviewed by Pierce and colleagues (Pierce, Keen & Bradley, 1995). They concluded that having a parent with NIDDM increases offspring risk by between two- and four-fold, but a family history of NIDDM does not increase risk of IDDM. NIDDM risk increases if the affected parent is the mother rather than the father, or if both parents are affected, and with increasing age, and in certain racial groups.

NIDDM is more common in those who are overweight, people who take little exercise, and in racial groups other than Northern European Whites. The association between obesity and risk of NIDDM is strong, graded, and consistent. This literature has been comprehensively reviewed by Felber, Acheson and Tappy (1992). The distribution of obesity, central or peripheral, has been shown to be a better predictor of later diabetes than the degree of adiposity. People with 'central' or 'male' type obesity are at much higher risk than those with 'peripheral' obesity.

Epidemiological studies have shown a relationship between inactivity and NIDDM (Helmrich, Ragland, Taylor, Leung & Paffenbarger, 1991; Taylor, Ramm, Zimmet, Raper & Ringrose, 1984), although these studies have been cross-sectional rather than prospective and hence caution is needed in deducing causality.

People with impaired glucose tolerance progress to NIDDM at a rate of between 2% and 6% per year, largely dependent on race (Jarret, Keen, Fuller & McCartney, 1979; Saad et al.1988). However, it is not yet possible to predict who will progress to NIDDM and who will revert to metabolic normality. Gestational diabetes can be regarded as an extreme form of impaired glucose tolerance in some pregnant women in response to the metabolic strain of pregnancy. After the baby is born, the diabetes may disappear, but it is likely to reappear in later life.

Certain racial groups, including Micronesians (e.g. Nauruans), Native Americans (e.g. Pimas), Asian Indians, Mexican Americans, and Australian Aborigines, seem to be particularly prone to NIDDM, a fact which may be due to their genotype (Neel, 1982). Their susceptibility manifests itself when they move from a 'traditional' lifestyle to a more 'modern' one with increased availability of food and with reduction in the demand for physical exertion (Zimmet, 1992). Possibly, the genetic make-up of these people is adapted to survival in the face of variable supplies of food. This 'thrifty genotype' (Neel, 1982) lays down fat stores very quickly when food is available and so facilitates survival during periods of famine. Such a genetic make-up would have clear evolutionary advantages in a 'traditional' lifestyle such as that of the Australian Aboriginal hunter-gatherers. However, when these people move to a life-style characterized by a

continuously plentiful food supply, they readily become overweight and develop NIDDM. Alternatively Hales and Barker (1992) have hypothesized that infants who are malnourished in utero, develop physical changes in their pancreas and other organs, which in later life cause a decreased ability to cope with excess calories and so these people become overweight and develop NIDDM. This 'thrifty phenotype' hypothesis could also account for these racial groups being particularly prone to NIDDM in the face of westernization.

4.3.2. Prevention of NIDDM

4.3.2.1. Primary prevention

There have been several studies testing the possibility of primary prevention of NIDDM using either pharmaceutical agents or behavioral methods (Erikksonn & Lindegarde, 1991; Jarret et al, 1979; Keen, Jarret & McCartney, 1982; Knowler, Sartor & Schersten, 1987; Sartor, Schersten, Carlstrom, Melander & Persson, 1980;). These have given somewhat contradictory results. However, recently Pan et al. (1994) reported the results of a randomized controlled trial of diet and exercise alone, or in combination, suggesting that all of these interventions reduced the rate of progression to NIDDM in people with impaired glucose tolerance in mainland China. In the light of these findings, a large multi-centre primary prevention trial is being planned in the USA. The interventions are likely to include hypoglycemic drugs and/or behavioral methods aimed at the reduction of obesity and the promotion of exercise. The challenges of designing such a trial are considerable as potential recruits are likely to have preferences for drug or lifestyle intervention which will make a simple randomized controlled trial design inappropriate (Bradley, 1993).

4.3.2.2. Secondary prevention

The secondary prevention of NIDDM is currently under investigation by the UK Prospective Diabetes Study Group (UKPDS) who are investigating the effects of early treatment regimens for NIDDM (UK Prospective Diabetes Study Group VIII, 1991).

4.3.2.3. Tertiary prevention

Tertiary prevention of NIDDM involves the detection and treatment of complications of NIDDM, and the reduction of associated risk factors. Retinopathy (American Diabetes Association, 1992), neuropathy, and early signs of nephropathy (Viberti, Walker & Pinto, 1992) can be detected by annual screening. Retinopathy can usually be treated effectively by laser therapy to prevent progression to blindness if detected in the early stages (Diabetic Retinopathy Study Group, 1981). Treatment of established neuropathy has been described by the St. Vincent Guidelines as 'unsatisfactory' (Krans, Porta, Keen & Staehr Johansen, 1995), though there is evidence from the DCCT that improved blood glucose control reduces risk of nerve damage (DCCT research group, 1993). There has been considerable interest in the possibilities of using drugs, particularly aldose reductase inhibitors, as neuropathy prevention therapy, though their value is far from clear (Krans et al.). The St. Vincent Guidelines suggest that the best hope for preventing neuropathic foot problems developing is through the education of patients and professionals and the organization of foot care services (Krans et al.). Incipient nephropathy can be detected by testing the urine for small amounts of albumin (microalbuminuria), and at this stage the process is potentially reversible by improved management of blood pressure and metabolic control. Avoidance of smoking (Mulhauser, 1990) and reduction of any modifiable risk factors for cardiovascular disease is particularly important for people with NIDDM, because of their elevated risk of myocardial infarction and stroke.

As with IDDM, psychological stress may trigger the manifestation of symptoms in NIDDM probably by a similar mechanism (Bradley, 1988; Clayer et al.1985). Evidence for a causal role of stress in initial onset of the pathological process is again less clear. Mechanisms by which stressful events may play a causal role will vary depending on the kind of NIDDM where, unlike IDDM, there is not always a reduction in insulin production. Indeed in individuals with NIDDM who are not overweight, insulin resistance may be involved where insulin production may increase as its effectiveness decreases. There is some evidence to suggest that stressful events may be causal in the onset of insulin resistance with stress-induced increases in adrenaline and

cortisol secretion reducing the effectiveness of an individual's insulin (Rizza, Mandarion & Gerich, 1982; Smith, 1984). If repeated or prolonged stressful events are experienced, insulin resistance over a period of months or years may result in excessive compensatory insulin production and, eventually, to exhaustion of pancreatic islet cells and manifestation of diabetes.

4.4 IMPACT OF THE CONDITION ACROSS THE LIFE SPAN ON PERSON AND FAMILY

With increasing knowledge of the familial risks of diabetes, diabetes may have a psychological impact on the outlook on life of an at-risk individual well before onset or without the actual onset necessarily occurring. People with diabetes may be concerned about the possibilities of onset of diabetes in their children. Indeed, the fact that a proportion of parents of at-risk children are prepared to enter them in clinical trials of the use of daily insulin injections in the hope of preventing diabetes onset (Keller et al.1993) provides an indication of the level of concern and lengths to which some are prepared to go in order to reduce the risk of diabetes onset.

When diabetes is diagnosed, the demands of diabetes and its treatment vary considerably with the type of diabetes (IDDM usually being more demanding in terms of insulin treatment required and the increased need for monitoring of blood glucose levels). Within any one type of diabetes there is considerable individual variation in the effort needed and difficulty experienced in achieving blood glucose levels which approximate normoglycaemia. The need to manage diabetes can add to the psychological demands of meeting the commonly experienced challenges throughout the life-cycle, such as leaving home, marriage, and pregnancy. Pregnancy, and preparing for pregnancy, are especially demanding times for women with diabetes when the need for normoglycaemia is emphasized as essential for the health of the baby and recommended prior to conception through to birth. The success which many women achieve in obtaining near-normal blood glucose levels at this time is sometimes attributed by their physicians to the hormonal changes of pregnancy rather than to the efforts of the woman herself. Such attributions can be demotivating and depressing. The challenges of a first pregnancy are, however, dwarfed

by the challenges of a second pregnancy where the needs of the first child are often incompatible with the needs of the developing second child. Pregnant women with diabetes attempting to achieve normoglycaemia are particularly prone to hypoglycemia and this risk carries new dangers when the pregnant woman is also in sole charge of a young child. Fear of hypoglycemia, a concern to most people with insulin-requiring diabetes (Irvine, Cox & Gonder-Frederick, 1994) is likely to be heightened in pregnant women, especially those with young dependent children.

Many people with insulin-requiring diabetes are sufficiently fearful of hypoglycemic reactions to avoid tight control of blood glucose levels and the associated increase in risk of hypoglycemia especially if their warning signs of hypoglycemia are unreliable (see sections below on psychological aspects of symptoms and on Blood Glucose Awareness Training). Fear of hypoglycemia may lead individuals to aim for higher blood glucose levels which increase their risk of long-term complications. Others, anxious to reduce their risks of other complications may be experiencing numerous, potentially dangerous, hypoglycemic episodes.

When NIDDM patients are unable to achieve adequate diabetes control with tablets and diet, it is usually recommended that insulin be introduced. Indeed the prospect of insulin injections is often used by doctors as a stick to motivate greater dietary efforts and more reliable use of sulphonylurea tablets. As a result, the need for insulin may be taken as a sign of failure or a sign of progression from 'mild' to increasingly 'severe' diabetes. Many patients also fear the process of injection, prior to the experience. In practice, the injections are usually found to be an obstacle soon overcome and incorporated into daily life with little difficulty. Injections allow greater flexibility of timing and content of meals as well as improved blood glucose control. Improved satisfaction with treatment following changeover from tablets to insulin has been found in several studies (Jennings et al. 1991; Taylor, Foster, Kyne-Gzebalski & Vanderpump, 1994). Such findings could be used by health professionals to inspire their patients who are reluctant to change. Interestingly the aspect of quality of life widely felt by people with diabetes to be impaired by their diabetes and rated as very important, is enjoyment of food (Bradley, 1995a). After decades of attempting to persuade patients to modify the timing and content of their food

intake to fit in with fixed insulin doses, and to use food manipulation to compensate for changes in energy expenditure, there is now growing recognition that a more rewarding approach is to manipulate insulin doses to fit in with food and other lifestyle factors (Howorka, 1996).

4.5. PSYCHOLOGICAL ASPECTS OF SYMPTOMS

Individual variation in the nature and extent of symptoms of hypoglycemia is considerable. Symptoms may include autonomic symptoms such as irritability or anxiety, shaking or sweating associated with adrenaline released in response to the falling blood glucose level. Symptoms may also include neuroglycopenic symptoms such as loss of concentration or memory loss, due to the shortage of glucose in the brain (Deary, Hepburn, Macleod & Frier, 1993). Hunger is also a common, though by no means universal symptom.

Individuals prone to hypoglycemia include anyone taking insulin and individuals taking certain kinds of oral hypoglycemic agent (particularly sulphonylureas). Individuals usually come to recognize the idiosyncratic but fairly reliable symptoms which for them herald the onset of hypoglycemia, and learn to take action to raise the blood glucose levels before they are rendered incapable of doing so by increasingly severe symptoms which impair rational action and precede the unconscious state of hypoglycemic coma. However, some individuals lose the ability to recognize the symptoms of hypoglycemia and become highly vulnerable to severe hypoglycemia as a result. Loss of warning of hypoglycemia, or hypoglycemia unawareness, may result from autonomic neuropathy, one of the microvascular complications of diabetes, which becomes increasingly common with duration of diabetes of 15 or more years. Damage to the autonomic nervous system impairs adrenal responses to low blood glucose levels. In the absence of adrenal symptoms, neuroglycopenic symptoms may not be noticed until the cognitive effects are too debilitating to take action to raise the blood glucose levels. However, onset of hypoglycemic unawareness is not always associated with autonomic neuropathy and there has been much concern among people with diabetes, clinicians and researchers to identify the causes of loss of warning with a view to preventing this loss or restoring awareness.

It has commonly been assumed that hypoglycemic unawareness develops when symptoms of hypoglycemia occur below the level of blood glucose at which cognitive impairment begins. It is also widely assumed that the detection of hypoglycemia is primarily dependent on autonomic symptoms. However, Cox et al. (1994) have argued that neuroglycopenic symptoms, such as difficulty in concentrating and lack of coordination, are as prevalent as autonomic symptoms and occur at similar levels of blood glucose. Furthermore, people with IDDM have reported using neuroglycopenic symptoms to recognize hypoglycemia, sometimes in preference to autonomic symptoms. Cox and colleagues also cited evidence to suggest that an episode of hypoglycemia leads to a reduction in strength of autonomic but not neuroglycopenic symptoms associated with any subsequent hypoglycemia within 24 hours. In line with these findings are reports from elsewhere (Cranston, Lomas, Maran, Macdonald & Amiel, 1994) that neuroglycopenic symptoms provided earlier warning of impending hypoglycemia than did autonomic symptoms and may indeed be more useful and more reliable cues to low blood glucose than has generally been assumed. Cox and colleagues at the University of Virginia have developed a Blood Glucose Awareness Training program (Cox, Gonder-Frederick, Julian & Clarke, 1992) which has been successfully used to regain lost awareness of hypoglycemia, and Amiel's research group at the University of London (Cranston et al.) and others (Fanelli et al. 1993) have also reported success using more traditional medical approaches. These interventional approaches are reviewed below.

In the late 1980's there was a nationwide move in the UK to switch from animal insulins extracted from cattle and pigs to the genetically engineered 'human' insulin. Most clinicians appear to have assumed that the changeover would have little effect on blood glucose levels. However, many people with insulin-treated diabetes were profoundly upset by the changeover as indicated by the influx of letters to the British Diabetic Association (Posner, 1992). Problems of rapid onset of severe hypoglycemia occurring without warning were reported, some with devastating and even fatal consequences. Although undoubtedly some of the problems were coincidental to the changeover to human insulin, it is by no means clear that all the problems would have occurred anyway (Alexander, 1993). The problems were further exacerbated by an

unwillingness of some doctors to accept the possibility that human insulin may have caused the problems, and their refusal to accede to their patients' requests to change back to animal insulin. Many patients continue to suffer from the aftermath of the changeover to human insulin, since new developments in insulin delivery systems are usually only developed for use with human insulin and thus are not available to those who changed back to animal insulins. The British Diabetic Association (BDA) continues to campaign for the provision of animal-insulin alternatives to the now standard human-insulin cartridges used in pen-injector devices, so that patients using animal insulins may also benefit from the convenience of using these devices (North, 1991).

A major reason for anxiety about hypoglycemia is the increasing concern that hypoglycemia may lead to chronic forms of cognitive dysfunction. It has long been recognized that severe hypoglycemia involving coma can cause chronic impairments of cognitive function. More recently, attention has been directed to the chronic effects of less severe, but recurrent hypoglycemia (Gold et al. 1994). It is difficult to demonstrate a causal association but the available evidence (including uncontrolled case reports and retrospective studies) does suggest that repeated and severe hypoglycemia may lead to chronic cognitive dysfunction. Ryan (1994) has provided a useful overview of work assessing cognitive impairments related to hypo- and hyper-glycemia which assesses the pros and cons of various measures of cognitive function in determining the nature and extent of cognitive impairments associated with different levels of blood glucose.

In contrast to the marked and troublesome nature of symptoms of hypoglycemia, hyperglycemia is quite often asymptomatic and this in itself creates challenges. Indeed some individuals who have adapted to raised blood glucose levels feel uncomfortable, when initially reducing their blood glucose levels. However, adaptation to the experience of blood glucose levels closer to normal levels soon follows. Resistance to tightening control of blood glucose levels may be reduced if such patients appreciate that any discomfort is likely to be temporary.

Most of the early signs of chronic complications of diabetes are also asymptomatic. High blood pressure, microalbuminuria indicative of early signs of kidney problems, and early signs of retinal damage are usually, if not always, without symptoms, and require medical screening to allow for early diagnosis and treatment. Peripheral neuropathy causes numbness in the extremities and a real risk that damage to the feet will go unnoticed. Infections and ulcers, once developed can be difficult to clear up in the face of circulatory problems which usually accompany the neuropathy and may lead to gangrene and the subsequent need for amputation. These serious problems currently depend for their solution on patient education, vigilant self-care, and rapid access to skilled chiropody and medical attention when minor damage to the feet is first noticed.

4.6. SPECIFIC ISSUES OF ADHERENCE

Adherence, or compliance, has been defined as the extent to which a person's behavior coincides with health advice (Haynes, Wang & Gomes, 1987). The concept implies that specific recommendations can be made which, if followed, are likely to lead to health benefits. Certainly the measurement of adherence requires that the recommendations have to be specified, and then the extent to which those recommendations have been implemented needs to be assessed. Such measurement may be practical if considering whether or not a patient had been adherent in taking a course of antibiotics. In this case, the recommendations can be specified in detail and the behavior monitored in various ways to establish the extent of match between recommendations and behavior. Where diabetes is concerned, potentially effective recommendations are likely to be complex, to vary from patient to patient and, for individual patients, to vary from day to day. 'Simple' recommendations to take the same amount and types of insulin at the same time each day and eat the same amount and type of food at the same times to balance that insulin while somehow keeping energy expenditure constant are unlikely to be effective in achieving adequate diabetes control. If such recommendations did lead to improved metabolic control they would be likely to cause serious damage to patients' psychological well-being and quality of life. More

complex regimens involve balancing the various manipulable components in the equation (eg timing and dose of the next injection of insulin, timing and content of the next meal or snack and voluntary exercise) to deal with those components of the equation which are either not immediately controllable or cannot be controlled without undesirable costs (e.g. family or work commitments, action of the previous insulin injection). Such regimens require sufficient education and understanding to allow problem solving appropriate to the occasions that arise and cannot be specified in detail in advance. 'Adherence', under these circumstances is an unmeasureable and unsuitable concept.

Some authors have referred to measures of diabetes control as 'proxy measures of adherence'. Such a practice is highly misleading as it assumes that if the patient had followed whatever recommendations had been made then the diabetes control would be maximal. Use of HbA1 or other measures of diabetes control as a measure of adherence denies the possibility that the recommendations made by the health professional may have been inadequate to the task of controlling that individual's diabetes. There is no good reason to attribute all departures from optimal outcomes to patient 'non-adherence' and there are many good reasons for not doing so. In particular, it is depressing and discouraging for patients who are trying to deal with the demands of diabetes management to have poor outcomes attributed to their actions (or lack of them), particularly when they have been struggling to follow recommendations which were not adequate to the task. The common tendency to take credit for successes and blame other factors for failure is one that affects health professionals as much as other groups (Gamsu & Bradley, 1987; Gillespie & Bradley, 1987) and it undoubtedly serves to protect their own self esteem. However, such an ego-defensive stance is not only discouraging for patients and their families but also serves to reduce the perceived need for interventions to improve the organization and functioning of the health-care system (Bradley, 1989). It would be more supportive of patients' self-esteem to recognize patients' efforts and achievements and to adopt a collaborative problem-solving approach when outcomes need to be improved.

There are many different ways of managing diabetes. If the patient is not able to achieve satisfactory diabetes control with one approach, it may be that some aspect of the approach is deficient in some way. The education provided for the patient may not have resulted in the necessary knowledge and understanding required. Barriers to care may be obstructing application of adequate education and may include social pressures and competing demands, fatalistic or unrealistically optimistic beliefs about outcomes of diabetes and the risk of complications (Glasgow, 1994; Lewis & Bradley, 1994). Knowledge, understanding and beliefs may all be compatible with self-care but the recommendations made may create conflicts with other priorities in the individual's life which may best be dealt with by changing the recommendations made without necessarily compromising the level of diabetes control that can be achieved.

Thus, in the complex field of diabetes management, the notion of self-care behavior is far more useful than the concept of 'adherence'. It can be useful and constructive to investigate self-care behavior and to explore the reasons for actions taken and consider the extent to which the actions were likely to result in the intended outcomes (and avoid unwanted outcomes) given the circumstances concerned. While it is neither necessary nor helpful to ask whether the actions were 'adherent' it can be useful to assess self-care activities and efforts have been made to do so (Toobert & Glasgow, 1994).

A useful review of the complexities in attempting to apply the concept of 'compliance' to diabetes management is provided by Glasgow (1991).

4.7. PSYCHOLOGICAL INTERVENTIONS IN THE MANAGEMENT OF DIABETES

4.7.1. Blood Glucose Awareness Training (BGAT) and Other Approaches to Regaining Hypoglycemia Awareness

Cox, Gonder-Frederick and their colleagues at the University of Virginia developed a program of Blood Glucose Awareness Training (BGAT) to train individuals with diabetes to recognize their level of blood glucose. The BGAT program and associated manual (Cox et al. 1992) is based on blood glucose monitoring together with monitoring of physical symptoms, mood and environmental cues associated with each blood glucose reading in order to identify the symptoms and cues which predict high blood glucose levels and those which predict low blood glucose levels for each individual. Cox and his colleagues developed an error grid analysis to identify those errors made in estimating blood glucose which matter clinically. Efforts are then focused on reducing the errors that would be clinically important such as failing to detect extreme blood glucose levels, or mistaking an acceptable blood glucose level for one that is too high or too low.

BGAT has been shown to be effective in teaching individuals with IDDM to improve their accuracy in recognizing blood glucose levels. Associated improvements in diabetes control have also been found with improved glycosylated hemoglobin results in BGAT patients compared with untrained control patients (Cox et al. 1991). Long-term follow-up has shown that participants in the BGAT training program had had fewer episodes of severe hypoglycemia and fewer car crashes than controls at five-year follow-up (Cox, Gonder-Frederick, Julian & Clarke, 1994). More recently, BGAT-2 has been developed to increase sensitivity to low blood glucose levels, and benefits have included fewer motoring offenses and car accidents, and fewer nocturnal episodes of severe hypoglycemia (Cox, Gonder-Frederick Kovatchev, et al. 1995). A multi-center trial of BGAT-2 used by clinicians other than those in the original research team developing the program, suggested that BGAT-2 offers a useful strategy for treating the disabling problem of hypoglycemia unawareness. BGAT-2 improved detection of low blood glucose levels in individuals who had reported impaired hypoglycemia awareness on recruitment to the study (Cox, Gonder-Frederick, Polonsky, et al.1995). There is increasing evidence that patients who have lost their warning symptoms of hypoglycemia can learn to attend to new signs of hypoglycemia which may be physical, cognitive or mood symptoms or may be circumstantial indicators that hypoglycemia is likely (e.g. running for a bus when late for a meal).

Another approach to treating loss of awareness of hypoglycemia has been to avoid hypoglycemia by diet review, advice about exercise and redistribution of insulin in patients with hypoglycemic unawareness (Cranston et al. 1994; Fanelli et al. 1993). After at least three clear weeks without hypoglycemia, patients became aware of symptoms of hypoglycemia. Adrenaline responses were more marked at higher blood glucose levels than was previously the case, and at levels higher than those at which cognitive impairment was indicated by a reaction-time task (Cranston et al.) It has been suggested (Bradley & Gamsu, 1995) that in attempting to avoid hypoglycemia, participants in such studies may learn to become more sensitive to neuroglycopenic symptoms at higher levels of blood glucose than they were before. Thus, the intervention to avoid hypoglycemia, may act as an indirect form of BGAT. It is also possible that the success of BGAT in increasing awareness of hypoglycemic symptoms may in part be due to a reduction in occurrence of hypoglycemia.

4.7.2. Identification of Stress Reactivity

4.7.2.1.Mechanisms involved in stress and diabetes control

Stress may affect metabolic control in diabetes in at least two ways (Barglow, Hatcher, Edidin & Sloan-Rossiter, 1984):

(i) a direct psychophysiological effect via sympathetic and pituitary activity which results in the elevation of catabolic hormone levels and the suppression of anabolic hormones. In people with diabetes, this may result in increased blood glucose levels, although, for a small minority, less readily understood decreases in blood glucose levels result.

(ii) a behavioral mechanism whereby stress leads to behavioral changes capable of disrupting self-care behavior. (Barglow et al. 1994). For example, time urgency may make blood glucose monitoring and well-balanced meals impractical leading to disruptions in metabolic control. Glycemic fluctuations themselves can contribute to the behavioral changes via impaired

cognitive function (Holmes, Hayford, Gonzalez & Weydert, 1983; Ryan, 1994; Wang et al. 1993), which, for example, may cause poor self-care responses to feedback from blood glucose monitoring. A two way causal link between stress and diabetes control has been suggested (Bradley, 1988) whereby life events cause disruption in diabetes control which in turn causes increases in the number of life events.

4.7.2.2. Acute stress and blood glucose reactivity

Several seemingly well-controlled acute stress studies conducted in the 1980's reported no significant changes in blood glucose control in response to potential stressors such as mental arithmetic and public speaking (Edwards & Yates, 1984; Kemmer et al.1986; Naliboff, Cohen & Sowers, 1985). However, this work overlooked the possibility that the commonly observed individual differences in response to stress might be real and interesting, and not a reflection of methodological inadequacies (Bradley, 1988). More recent studies of experimental stress have examined physiological mechanisms hypothesized to mediate the relationship between psychological stress and blood glucose control. There is evidence that reduced blood flow to the insulin injection site (Hildebrandt, Mehlsen, Sestoft & Nielson, 1985) and insulin resistance over several hours (Moberg, Kollind, Lins & Adamson, 1994) may cause increased blood glucose levels in individuals with IDDM in response to acute laboratory stressors. It has been noted elsewhere (Bradley & Gamsu, 1995) that laboratory studies with a short time frame are likely to underestimate such long-lasting effects of stress on blood glucose.

The effects of a laboratory stressor (Stroop test) on changes in blood flow at the insulin injection site have been studied in IDDM patients (Greenhalgh, Jones, Jackson, Smith & Yudkin, 1991).

The results showed that mean levels of blood glucose rose in some patients and fell in others in response to acute stress, and that these changes were largely explicable in terms of changes in free insulin levels, which in turn were partially mediated by changes in injection-site blood flow. The authors proposed that vasodilation at the subcutaneous insulin injection site may in some cases lead to a paradoxical hypoglycemic effect during acute stress via an increased rate of absorption of insulin, an effect which will be counterbalanced to a greater or lesser degree by increases in counter-regulatory hormones. In other individuals, absorption of insulin may fall during stress and contribute to hyperglycemia. Again these findings underline the importance of looking at individual differences.

Idiosyncratic blood glucose responses which were reliable across a 12-week time period within individuals with IDDM have been reported in response to caffeine and to the competitive playing of a video game (Carter, Gonder-Frederick, Cox, Clarke & Scott, 1985). A more recent study from this research group (Gonder-Frederick, Carter, Cox & Clarke, 1990) has reported similar results**.** Such studies suggest that the inconsistent findings of previous research may have resulted because of differences in the stress-responsiveness of the individual patients recruited.

4.7.2.3. Life stress and blood glucose reactivity

Studies of major life events and diabetes control have suggested that increased life events are associated with raised blood glucose levels (Chase & Jackson; 1981; Barglow et al. 1994). However, within-subject variations in stress and in blood glucose values would have been obscured by the methods used which correlated aggregated retrospective reports of life events with GHb or HbA1 measures of averaged blood glucose, thereby oversimplifying the picture. Recent research has begun to look at the relationship between minor daily events and diabetes control (using serial blood glucose measurements) in order to overcome the methodological limitations of the earlier studies of life events - blood glucose relationships.

Investigations of daily stressful events are providing more interpretable results with studies reflecting individual differences in response to stress that were also seen in some of the laboratory based acute stress studies. Although the overall picture suggests that increased daily stress correlates with increased blood glucose levels in IDDM (Hanson & Pichert, 1986) and in NIDDM (Goetsch, Abel & Pope, 1994), examination of individual's blood glucose response to stress indicates that some people display stress-reactivity and some people do not. Halford, Cuddihy and Mortimer (1990) found that approximately half the sample of 15 IDDM participants had significant associations between stress and blood glucose levels, and that this association was independent of the effects of diet and exercise self-management.

Within those people who do display stress-reactivity, most show an increase in blood glucose while a few show a decrease in blood glucose (Aikens, Wallander, Bell & McNorton, 1994; Riazi, Pickup & Bradley, 1996). Aikens and colleagues studied twenty-five women with IDDM who monitored daily stress and blood glucose for 30 consecutive days. Blood glucose was found to be higher on high-stress days than on low-stress days, with 8 of the 25 subjects showing significant positive associations between stress and same-day blood glucose. Stress showed little relation to next-day blood glucose, although two subjects showed associations (one positive and one negative). In a recently conducted study of 54 men and women with IDDM which measured daily stress and blood glucose levels over a three week period (Riazi et al. 1996), individual differences were marked in the magnitude, direction and duration of blood glucose stress reactivity. Those with higher levels of HbA1 indicating poorer control of diabetes, were more stress-reactive than those with lower levels of HbA1.

These findings show the individual differences in response to stress that can be seen in laboratory based acute stress studies which investigate individual differences but are not seen in studies which limited the analysis to group differences or in studies of life events using average measures of blood glucose. Although increases in blood glucose levels are the most common form of stress reactivity, decreases are not uncommon and there is a need for individuals to

discover empirically how their own blood glucose levels respond to different kinds of stress in order to take appropriate action to prevent or correct disruptions in diabetes control.

4.7.3. Stress Management Training

The overly simple model of life stress causing raised blood glucose levels has inspired the use of stress management training, particularly relaxation training, as an aid to diabetes control. Relaxation is thought to decrease adrenocortical activity (Jevning, Wilson & Davidson, 1978; DeGood & Redgate, 1982) as well as circulating levels of catecholamines (Mathew, Ho, Kralik, Taylor & Claghorn, 1980; Mathew, Ho, Kralik, Taylor, Semchuk, et al. 1980) and hence is expected to prevent stress-induced increases in blood glucose levels.

The research to date supports the view that stress management techniques may be valuable to aid diabetes management for some people but not with others. It has been suggested (Bradley, 1994a) that relaxation techniques are unlikely to do harm except when blood glucose is already tightly controlled (Seeburg & DeBoer, 1980) and the insulin dosage is not appropriately reduced to balance the effects of relaxation on insulin requirements (which may well be reduced) and/or when used at a time when blood glucose is already low (<4 mmol/L) and there is a risk of hypoglycemia. It is important to take the precaution of measuring blood glucose immediately before all relaxation training or practice sessions. Twenty minutes of relaxation can lead to a drop in blood glucose as great as 3 mmol/L. Such a substantial fall is only likely if blood glucose is well above the normal range of 4-6 mmol/L to start with and probably results from a suppression of catecholamine secretion. It is recommended that relaxation training should not be conducted at blood glucose levels below 4 mmol/L because of the risk of hypoglycemia. Relaxation training might be continued if the anticipated reduction in blood glucose were counteracted with pre-training intake of slow release carbohydrate (e.g. an apple). A further precaution of measuring the blood glucose after a relaxation session, before leaving the therapist's office, is also recommended to avoid the risk of hypoglycemia while traveling home.

Several studies have reported some success with improvements in diabetes control following relaxation training (usually involving EMG biofeedback) in NIDDM and IDDM patients (McGrady, Bailey & Good, 1991; McGrady & Gerstenmaier, 1990; Rosenbaum, 1983; Surwit & Feinglos, 1983). There is evidence to suggest that relaxation training is least useful for those subjects whose glycemic control was good to start with and most useful when used by subjects who not only had poor control of their diabetes but who felt that stress disrupted their diabetes control and who were currently experiencing stressful events (Bradley, Moses, Gamsu, Knight & Ward, 1985; Lammers, Naliboff & Straatmeyer, 1984).

In contrast, Feinglos, Hastedt and Surwit (1987) found no significant effect of relaxation training in people with poorly controlled IDDM who reported stress-induced hyperglycemia. After 6 weeks in which the trial group practiced relaxation techniques at home, measures of glucose tolerance, GHb levels (which reflect blood glucose levels over 6-8 weeks), and insulin requirements did not differ between relaxation and control groups. Unfortunately, the authors did not provide any information about the variability of the individual patients.

In a more recent study, Lane, McCaskill, Ross, Feinglos & Surwit (1993) investigated 38 people with NIDDM who were treated with intensive conventional diabetes therapy (including diabetes education and dietary interventions) with half of the subjects assigned at random to receive biofeedback-assisted relaxation training. Both groups demonstrated significant improvements in GHb level after 8 weeks, but not in glucose tolerance. No added benefit of relaxation training was seen on either measure though it was found that those subjects who showed improvements in glucose tolerance after pretreatment with the anxiolytic drug alprazolam also showed improvements in glucose tolerance after relaxation training. It was also found that subjects who responded best to relaxation training were those scoring higher on measures of trait anxiety. Overall, the authors concluded that relaxation may be useful for the treatment of neurotic, anxious, and autonomically reactive individuals but may be ineffective for other patients. Lack of

selection of patients likely to benefit from relaxation training may have been responsible for the nonsignificant finding of relaxation of the group as a whole.

What is emerging from the studies of relaxation training on people with diabetes, is that it is most useful for those who show stress-related disturbance of blood glucose control or for those who are anxious and autonomically reactive. The degree to which these groups overlap is as yet unclear though there is some evidence to suggest that the groups may be separate. Self-reported anxiety did not correlate with blood-glucose reactivity to stress in the studies of Riazi et al (1996). Assessment of subjects' stress-reactivity before embarking on relaxation training in future studies will help in the identification of patients most likely to benefit from relaxation training. To date, most studies have incorporated biofeedback into the relaxation training. Relaxation training on its own would be more practical and less costly. There is limited evidence that biofeedback is an unnecessary addition for most people (Bradley et al. 1985). Evaluation of low-technology forms of relaxation training with patients shown to be stress reactive would be a useful next step.

4.7.4. Monitoring of Psychological Outcomes and Processes

Just as measurement of blood glucose is essential to the task of achieving and maintaining tight control of diabetes, the measurement of psychological outcomes is central to protecting and improving the quality of life of people with diabetes. However, while measurement of blood glucose is standard practice in diabetes management and research, measurement of psychological outcomes is, as yet, common only in research settings. Quality of life measurements are now increasingly recognized as essential parameters in clinical trials evaluating new treatments for diabetes and improvements in aspects of quality of life are often among the primary outcomes to be investigated. Treatments including insulins and insulin delivery systems are being developed to be more convenient and flexible and compatible with quality of life. There is growing recognition that the desired biomedical outcomes are more likely to follow if treatment regimens

are convenient and flexible and can be integrated into individual's lives without impairing the quality of life. Although measurement of psychological outcomes in routine clinical practice is far from standard practice, recent initiatives are encouraging such developments. The St. Vincent Declaration Action Programme for diabetes care in Europe has recommended the routine measurement of psychological well-being and patients' satisfaction with treatment in the monitoring of diabetes management (Bradley & Gamsu, 1994; Krans et al. 1995), and many national initiatives are now underway including a national study of monitoring of diabetes care in the UK (Wilson et al.1993), and an initiative in the Netherlands to evaluate the effects of routine monitoring of the psychological outcomes recommended by the St. Vincent Guidelines F.J. Snoek and colleagues, personal communication, 1996).

4.7.4.1. Measurement of psychological well-being and patient satisfaction

The measures recommended in the St. Vincent Guidelines are the Well-being Questionnaire (Bradley, 1994b, Bradley & Lewis, 1990) and the Diabetes Treatment Satisfaction Questionnaire (Bradley, 1994c) which were designed and developed specifically for people with diabetes. The Well-being Questionnaire was first designed in the early 1980's for diabetes clinical trials because it was recognized that generic instruments to measure depression or anxiety are unsuitable for people with diabetes. Measures such as the Beck Depression Inventory (Beck, Ward, Mendelson, Mock & Erbaugh, 1961), developed for the general population, include items concerned with somatic symptoms which may well be indicative of depression in the population as a whole but in a diabetic sample are more likely to result from inadequate blood glucose control. For example, symptoms of fatigue, appetite disturbances, weight loss, sleep disturbances, irritability and loss of sexual interest can all be symptoms of high blood glucose levels. At around the same time and for the same reasons, Zigmond and Snaith (1983) developed the Hospital Anxiety and Depression (HAD) scale to focus on cognitive symptoms and avoid somatic symptoms. The HAD was intended to be appropriate for use with hospital patients and encouragingly, recent work with diabetic populations has confirmed the original factor structure of the HAD (McColl et al. 1995).

The Well-being Questionnaire has subscales to measure depression and anxiety which are similar to those of the HAD but, unlike the HAD, also has subscales to measure energy and positive well-being. These additional subscales are more sensitive to change than the depression and anxiety (Bradley, 1994b) and the Well-being Questionnaire is likely to be more sensitive than the HAD to positive benefits of new treatments for diabetes. If only depression and anxiety scales are used, the optimal state measurable is an absence of depression or anxiety and floor effects occur if, as usually happens, in diabetes research, patients are not depressed or anxious when baseline measures are taken. The World Health Organization (WHO) has recommended wider use of the Well-being Questionnaire with other patient groups in addition to those with diabetes, in a context of continuing development (WHO Regional Office for Europe Consensus Meeting on Quality Assurance Indicators, Stockholm, August 1993 and November 1995). As with the HAD scale, care is needed when using the instrument with a different population of patients as, despite efforts to reduce confounding of depression scores with symptoms of poorly controlled diabetes, different illnesses may influence responses in different ways. Efforts to improve and evaluate translations of the Well-being Questionnaire in European and other languages have been associated with the widespread use of the instrument in multi-national clinical trials and in the St. Vincent European monitoring initiative and work is underway to meet the demand for a shortened version of the 22-item Well-being Questionnaire which will facilitate use of the instrument in routine clinical practice (Bradley,1996a).

The Diabetes Treatment Satisfaction Questionnaire (DTSQ) (Bradley, 1994c; Bradley & Gamsu, 1994; Krans et al. 1995), is a single-page instrument that has been widely used in clinical trials and is valued for its sensitivity to change in response to alterations to diabetes treatment regimens. A broader, though less detailed, single-page measure of satisfaction with the diabetes clinic service (The Diabetes Clinic Satisfaction Questionnaire, DCSQ) has since been developed to meet the need to audit patients' satisfaction with the service as a whole as well as, more specifically, with their particular treatment regimen (Wilson et al. 1993). The DCSQ is designed to identify sources of dissatisfaction so that efforts to improve the service can be well informed.

This instrument is being used in the UK national study of audit of diabetes care steered by the Diabetes Information Managment and Audit Committee of the British Diabetic Association. (Wilson et al.).

The use of diabetes-specific instruments such as the Well-being Questionnaire, DTSQ and DCSQ allows for instruments which focus on issues of particular relevance to the diabetic population. The instruments can be short because issues of little concern in diabetes can be excluded. Although generic instruments are less likely than diabetes-specific instruments to be sensitive to changes following changes in treatment for diabetes, there is some demand for generic instruments which comes particularly from health service managers and health economists who want to compare outcomes in different patient groups. The reader is referred elsewhere for a review which considers the use of generic measures with diabetic populations (Bradley, 1996b).

4.7.4.2. Measurement of quality of life

The phrase 'quality of life measures' is often indiscriminately used to cover health status measures, well-being and satisfaction measures and even measures of self-reported symptoms as well as measures of how good or bad life is felt to be. Focusing here on the latter group of measures, an important distinction can be made between individualized measures and those which are not tailored to the individual.

With most measures of quality of life, diabetes-specific or generic, the aspects of life deemed to be relevant to quality of life are specified by the designers of the questionnaire. Although the choice of aspects of life may have been informed by prior research with samples of people with diabetes and by input from health professionals involved in diabetes care, there is no scope for the individual respondent to decide if any one particular aspect of life asked about is applicable to them and, if it is, whether it is important to their quality of life. The Diabetes Quality of Life (DQOL) measure used in the Diabetes Control and Complications Trial includes 56 items which

all receive equal weight regardless of their relative importance to the individual (Jacobson et al. 1994). It has been suggested that the lack of differences reported between DQOL scores of those who intensified their treatment in the trial and those who did not may be attributable to the limitations of the measure rather than to any real lack of impact of intensification of treatment on quality of life (Bradley, 1994d). Thus an item concerned with immediate and pervasive worries about hypoglycemia which may markedly impair an individual's quality of life will have no more impact on the DQoL total score than an item concerned with satisfaction with time taken to determine blood glucose levels or an item about how often the individual has to explain what it means to have diabetes. The DCCT research group reported that the DQoL total scores showed no impact of intensified treatment on quality of life (DCCT, 1993). This was surprising given the considerable increase in severe hypoglycemia experienced by the intensified treatment group in the DCCT, but not surprising when the nature of the quality of life measure used is examined.

Individualized measures of quality of life ask the individual respondent which domains are important to his or her quality of life. An increasingly valued measure of individual quality of life is the Schedule for Evaluation of Individual Quality of Life (SEIQoL) interview method (Hickey et al.1996; McGee, O'Boyle, Hickey, O'Malley & Joyce, 1992; O'Boyle, McGee, Hickey, O'Malley & Joyce, 1991). The SEIQoL establishes in interview the domains of life important to a particular individuals quality of life, elicits ratings for each domain from 'as good as it could possibly be' to 'as bad as it could possibly be', determines weights for each domain using a method of judgment analysis. The philosophy underpinning the SEIQoL has influenced the design of a questionnaire measure for Audit of Diabetes Dependent Quality of Life (ADDQoL) (Bradley, Todd, Gorton, Plowright & Symonds, 1995; Todd, Bradley & Symonds, 1993). The ADDQoL includes a series of domains identified in pilot work to be commonly affected by diabetes and its treatment. Working life and family life are two of the standard domains included together with worries about the future, enjoyment of food and other items more specifically relevant to diabetes. Each domain is first rated for the impact of diabetes on that domain and then rated for the importance of that domain for quality of life. Domain ratings

are then multiplied by importance ratings such that a domain which is unimportant will score zero regardless of how much it is affected by diabetes while ratings other than zero for an important domain will receive greater weight. Any domain that is said to be inapplicable is ignored. The weighted ratings of all applicable domains are then summed and divided by the number of applicable domains to give scores which vary from the same maximum (+9) to the same minimum (-9) for all individuals. In this way, the ADDQoL, like the SEIQoL, focuses on applicable domains that are important to the particular individual. Unlike the SEIQoL, the ADDQoL is specifically concerned with the effects of diabetes and its treatment on quality of life. The ADDQoL provides a framework which is readily modified to be suitable for use with other patient groups and is currently being adapted for use with renal patients. There is now considerable interest in patient-centered measures of quality of life and although other measures aim to be relevant by ensuring that item design is informed by qualitative work with patients, few instruments are patient-centered in providing opportunities for patients to indicate which items are relevant to them personally and which are not and to provide their own weightings of importance for each relevant item (Greenhalgh, 1996; Bradley, 1996b).

4.7.4.3. Measurement of knowledge, beliefs and other cognitions.

Many different measures of knowledge of diabetes were designed and developed in the 1980's but more recently there has been widespread disenchantment with such measures (Glasgow, Toobert, Hampson & Wilson, 1995). This has resulted in part from the abstract content of some knowledge measures and partly from the inappropriate expectation that knowledge of diabetes will correlate with measures of blood glucose control (Beeney, Dunn & Welch, 1994). When no correlation is found the validity of the knowledge questionnaire has been questioned. However, there is no logical reason to expect that responses to many of the items on knowledge questionnaires will correlate with blood glucose levels (e.g. items concerned with foot care). In addition, validation of knowledge items is complicated by the fact that patients often acquire considerable knowledge after they develop a problem, thereby leading to counterintuitive associations between knowledge and outcomes in cross-sectional surveys. Care and ingenuity is needed to select appropriate methodologies to develop knowledge measures for particular

purposes (Bradley, 1995). In the light of continuing evidence of inadequate levels of knowledge, with patients with NIDDM being particularly disadvantaged (Anderson, Hiss, Stepien, Fitzgerald & Funnel, 1994), the measurement of knowledge and provision of remedial education as required is likely to result in marked improvements in biomedical and psychosocial outcomes of diabetes care.

Other measures of psychological processes which can be valuable in understanding difficulties with diabetes management include measures of health beliefs, and perceptions of control, or locus of control, of diabetes. A variety of reliable diabetes-specific measures have been validated for various purposes (Bradley, 1994e; Kohlmann et al. 1993; Lewis & Bradley, 1994). Measures of health beliefs and, particularly measures of patients' attributions or locus of control, have proved useful in understanding patients preferences for injection treatments or continuous subcutaneous insulin infusion (CSII) pumps, and in understanding why some patients developed the dangerous acute complication, diabetic ketoacidosis (DKA), during pump use (Bradley, Gamsu, Knight, Boulton & Ward, 1986; Bradley et al.1987).

In a feasibility study involving all insulin treated patients at the Royal Hallamshire Hospital in Sheffield (Knight et al. 1984), patients were offered the choice of CSII pumps, intensified injection treatment or conventional injection treatment. An illustrated lecture informed patients about the treatments available and patients were told that "the evidence suggests that CSII controls blood glucose levels better than injections". Unfortunately, only with hindsight was it realized that patients needed to know, but had not been told, that the effectiveness of CSII depended on the actions of the individual user and that CSII required at least as much, if not more, thought, effort and precautions to use it effectively and safely, than was needed with injection therapies. Those attracted to CSII tended to be those who were seeking a medical solution for their condition and who had less sense of personal control over their diabetes (as measured by the perceived control of diabetes scales). Sadly, it was those with the strongest sense of medical control and least sense of personal control who did least well with CSII pumps both in terms of blood glucose control at 12 months (Bradley et al. 1987), and occurrence of

DKA during the study (Bradley et al. 1986). A massive increase in rates of diabetic ketoacidosis occurred among CSII users in the first two years of the study, and the only variables to discriminate between those who developed DKA and a matched group of those with no DKA were the psychological variables, perceived control and health beliefs.

Although questionnaire measures of health beliefs and cognitions are somewhat cumbersome for clinical use, experience with the tools in a research context and in detailed assessment of individuals presenting with particular problems can be valuable. The concepts underlying the measures can usefully guide clinical interviews seeking to establish sources of difficulty with diabetes management with a view to tailoring the treatment to suit the individual patient and identifying inappropriate beliefs and expectations of treatments which need to be addressed.

The Handbook of Psychology and Diabetes (Bradley, 1994f) provides ready access to most of the questionnaire measures described above and provides reference to yet more measures. Despite the availability of such measures and increasing evidence for their value in clinical as well as research contexts, there is resistance among some clinicians to monitoring psychological outcomes (Barendse, 1995). The unfamiliarity of the tools leads many physicians to feel that they would need training in use of the instruments before they would feel able to use them. There is also concern about how they would deal with cases of depression and anxiety which may be brought to light as a result of using the questionnaires. These are both understandable concerns which may be dealt with by involvement of psychologists to support use and interpretation of the psychological measures and in helping to deal with psychological problems identified. Interestingly one source of resistance to using the instruments was the reported concern that patients would not want to have psychological outcomes considered as part of routine diabetes care though all the available evidence suggests that quite the reverse is the case: people with diabetes and parents of children with diabetes have long complained about the excessive emphasis on their blood glucose levels in diabetes clinics while little attention is paid to the effects of diabetes management on their lifestyle and quality of life. Clinical psychologists have an important role to play in encouraging and supporting diabetes care teams in measuring

psychological outcomes and in taking action to protect or improve the psychological outcomes of people with diabetes.

4.7.5. Patient Empowerment

With diabetes, as much if not more than with other chronic illnesses, optimal management can only be achieved by means of complex behavioral changes on the part of the patient. It is not enough for the clinician merely to give instructions. Health professionals are increasingly working towards holistic approaches to treatment, which include an emphasis on providing the education, support and encouragement to enable patients, where possible, to manage their own diabetes. Indeed, the 2nd edition of the St. Vincent Declaration Action Programme to improve diabetes care in Europe (Krans et al. 1995) now includes specific guidelines to facilitate patient empowerment.

The construct of empowerment has been in use in other domains of psychology for some time, and has at times been both ill-defined and misunderstood. In the context of diabetes care, a useful overview of patient empowerment has been provided by Anderson (1995).

The 'empowerment' of individual patients is a complex goal. It requires first that patients have a level of authority and responsibility to be able to make decisions and manage their own treatment. Secondly, it requires that they be willing to accept that responsibility and to achieve the necessary skills and knowledge to make them capable of managing their diabetes. And thirdly, the environment of care needs to be such that policies and practices facilitate self-management of diabetes care. Central to the achievement of each of these elements is the health care professional. He or she must accept patients as active and responsible self-carers, educate them both in knowledge of diabetes and self-care choices, and facilitate the setting of individual goals of self care through an assessment of individual preferences. Patently, this becomes even more vital in an age of technologically advanced health-care systems, where the tendency may be to alienate patients from active participation in their own medical treatment (Stabler, 1993).

Whilst numerous accounts of research into patient empowerment may be found, the literature specific to diabetes care is as yet limited, but growing (Anderson, 1995; Anderson, Funnell, Barr, Dedrick & Davis, 1991; Anderson, Funnell, Butler, Arnold & Feste, 1995; Doherty et al, 1996; Feste, 1992; Kinmonth et al. 1996). In looking to the future for a more widespread adoption of attitudes and practices which facilitate patient empowerment, it is clear that certain barriers need to be overcome. Some patients may be reluctant to accept responsibility for their own diabetes management, and need to be encouraged with the knowledge that they are best placed to make decisions which impact on the day to day running of their lives. They then need access to professional support, education in the knowledge of their condition, self-care skills and choices available to them. Health care professionals may also misunderstand patient empowerment. They may see this as an unethical suggestion that they should abdicate responsibility for care and relinquish clinical control and decision making. At the other end of the spectrum, some may see patient empowerment as a license to divest themselves of the more intractable problems associated with diabetes care. In reality, however, patient empowerment involves active participation of the health professional in the role of facilitator, enabling patients to set individual goals for diabetes management and to achieve optimal outcomes in both medical and psychosocial health.

There is early evidence for the success of patient empowerment interventions. Anderson et al. (1995) reported a randomized waiting-list control group trial of patient empowerment education. Despite some problems associated with drop-out rates and the combination of data from randomized and non-randomized participants for analysis, the results suggested a modest improvement in blood glucose control, along with gains in self-efficacy and attitudes toward diabetes.

Greenhalgh, Chowdhury & Helman (1996) evaluated a patient empowerment model with a sample of British Bangladeshis with diabetes, and identified a variety of culturally-related factors which may create obstacles to patient empowerment including ignorance about diabetes and

available services, poverty, fatalism, and religious convictions. These findings underline the need for individual patient focus in empowerment programs if those programs are to be appropriate to patients needs. Quatromoni, Milbauer, Posner, Carballeira, Brunt & Chipkin (1994) described a study which used focus groups to explore culture-specific information that could be useful in empowerment interventions with Caribbean Latinos with NIDDM. Amongst the issues they identified were "feelings of social isolation, little understanding of long-term consequences of diabetes, fatalism regarding the course of the disease, barriers to diet and exercise interventions, skepticism regarding the value of preventive health behaviors, and a clear need for culturally-sensitive health-care provision" (Quatromoni et al. 1994, p869). Similarly, the Haida Gwaii Diabetes Project (Herbert, 1996) described an approach which uses community-based research to identify a culturally-sensitive approach to diabetes prevention and management. Outcomes suggest an empowerment benefit to individuals with diabetes, their families, and community. Moreover, there were reported benefits to health care professionals, in that they also felt empowered to implement diabetes care in ways which were more pertinent to patients.

Early evidence suggests that patient empowerment offers a promising way forward in diabetes care. It is perhaps worthwhile, however, to sound a cautionary note. Should empowerment interventions fail to show significant benefits in the future, it may be that the interventions themselves have not been adequate to the task, either in that health professionals did not receive adequate training to enable them to move to more participative care, or that the empowerment goals were not sufficiently focused on the cultural or individual needs of patients.

4.7.6. Weight Management

Weight management is a problem both for people with IDDM, and NIDDM in rather different ways. People with IDDM need to balance their carbohydrate intake, insulin and exercise in order to control their blood glucose levels. Current dietary recommendations tend to be less restrictive than was the case in the 1980s though typically patients would be advised to avoid foods

containing simple sugars (sweets, cakes, sugary drinks etc.) and foods with a high fat content. Paradoxically if they develop hypoglycemic symptoms they need to take instantly available glucose to avoid a severe hypoglycemic episode which can lead to coma. The consequent rise in blood glucose may overshoot and result in hyperglycemia. If their carbohydrate intake is too high, an increase in insulin dose will be needed to maintain metabolic control and, as with people who do not have diabetes, carbohydrate excess will lead to weight gain. However, in people with diabetes, crash diets to deal with unwanted weight may result in metabolic chaos and a careful weight reduction program is needed which balances reduced carbohydrate intake and/or increased exercise with reduced insulin doses.

4.7.6.1. Eating disorders and withholding of insulin

Adolescent girls with IDDM tend to be heavier than matched non-diabetic girls for reasons which are not understood, and are likely to be dieting more intensely in order to control their weight. In cross-sectional and case-controlled studies (Fairburn, Peveler, Davies, Mann & Mayou, 1991; Peveler, Fairburn, Boller & Dunger, 1992), eating disorders have been found to be as prevalent but not more common in young women with IDDM than those without. Nine per cent of adolescent girls in the latter study met the diagnostic criteria for 'eating disorder not otherwise specified' in the ICD (International Classification of Diseases) classification. Other studies have indicated that adolescents and young women with diabetes are at greater risk of eating disorders than those who do not have diabetes (Rodin, Daneman, Johnson, Kenshole & Garfinkel, 1985; Rosmark et al.1986). However, care needs to be taken over the methods used to identify eating disorders in people with diabetes. Generic eating disorder self-report assessments, such as the Eating Attitudes Test (EAT) or Eating Disorder Inventory (EDI) are likely to yield different scores from people with diabetes because of the emphasis on diet and dietary restraint in diabetes education. Items such as I “avoid foods with sugar in them” and “engage in dieting behavior” on the EAT and “I eat sweets and carbohydrates without feeling nervous” on the EDI may be useful indicators of eating disorders in the general population, but in people with diabetes, responses will be affected by the diabetes treatment regimens commonly recommended. Efforts have been made to control for this bias by excluding from the EAT and EDI those items

which would be answered significantly differently by those with diabetes (Cantwell & Steel, 1996; Steel, Young, Lloyd & Macintyre, 1989). However, further evaluation is needed before these modified measures can be used with confidence in studies involving people with diabetes. Rodin and Daneman (1992), in a review of this literature suggested that the reliability of prevalence rates for 'caseness' is improved when based on structured interviews and standardized diagnostic criteria such as ICD-9, rather than self-report questionnaires which are often used in prevalence studies.

However disordered eating has been found to be common even in more methodologically rigorous studies I addition to more commonly recognized eating disorders and Peveler and colleagues (1992) also reported that 15% of their sample were omitting or reducing their insulin in order to reduce their weight. Insulin withholding has also been found to be associated with current or past symptoms of anorexia or bulimia nervosa **(**Biggs, Basco, Patterson & Raskin, 1994). When insulin is withheld, blood glucose levels will rise until they reach the renal threshold (10mmol/L) and then glucose will be excreted in the urine taking with it water which would otherwise have been retained. Thus, withholding of insulin is a means of effortless weight loss though not without potentially serious consequences. In particular, the chronic hyperglycemia of a person who habitually withholds insulin to lose weight, will put the individual at risk of diabetic ketoacidosis requiring hospitalization in the short term (if they omit insulin for too long a period) and, in the longer term, the risk of microvascular complications of diabetes is markedly increased. Pediatricians responsible for the care of adolescents with diabetes may well downplay concerns about future complications for fear of alarming young patients and their parents and it can therefore be quite possible for young people with diabetes to be ignorant of the now well-established causal link between chronic hyperglycemia and the serious microvascular complications which are the reasons for concern that motivate the clinicians to attempt to improve diabetes control. Where young people with IDDM are well informed about the risks of microvascular complications and the importance of blood glucose control for reducing those risks, the attractions of quick and easy weight loss may nevertheless outweigh the more distant concerns of complications in 15 years or so and by injecting some insulin

occasionally, the dangers of diabetic ketoacidosis and associated coma may be avoided. Withholding of insulin thus presents a diabetes-specific form of eating disorder which can have much sought after immediate effects but with serious short- and longer-term consequences.

4.7.6.2. Weight reduction programs in NIDDM

In NIDDM, obesity is more prevalent than in the general population. Dietary therapy is the mainstay of treatment of NIDDM. A significant proportion of people who follow the dietary recommendations will regain metabolic control without having to take oral hypoglycemic tablets or insulin injections. In the overweight, calorie restriction is particularly important for metabolic control and weight loss is associated with improved glycemic control. Many studies have shown that it is possible to induce weight loss in NIDDM patients using a variety of pharmaceutical methods, e.g. appetite suppressants, behavioral methods, exercise, conventional diets or very low calorie diets (VLCDs). However, longer term studies typically show that subjects lose weight for the first 6 months of a program and thereafter regain the weight (Wing & Anglin, 1996). The UK Prospective Diabetes Study Group (1990**)** examined the responses of more than 3000 newly diagnosed NIDDM patients to diet therapy and showed that 16% achieved near-normal fasting glucose after 3 months (with an average weight loss of 8 kg). However in the second 3 months the blood glucose increased a little in spite of continued but decelerated weight loss. Metabolic control appears to be related more to calorie intake than body weight per se. This study shows the value of diet therapy in the successful management of NIDDM but the weight loss required for adequate diabetes control may be considerable and oral hypoglycemic agents may be needed as well.

The evidence suggests that that even when dietary change is achievable in the short term, it is not sustainable at the same level over a longer period. Perhaps more attention needs to be given to developing diets which are more acceptable to the individual and, hence, more sustainable. Diets may be unacceptable for economic, psychological or cultural reasons. A diet that may be affordable and tolerable in the short term with the support of therapist and fellow patients on a weight loss program may be unsustainable without such support. There is marked individual

variation in weight loss achieved in weight loss programs. In one intervention study, outcomes varied from a weight loss of 29.5kg to a gain of 7.7kg from pre-treatment to follow-up (Wing, Epstein, Nowalk, Koeske & Hagg, 1985). An understanding of the reasons for the variability may well be helpful in determining how to improve the success rate. In particular, consideration of patients' preferences for different weight loss programs may be of value in facilitating successful weight loss. It is likely that a group program will suit some but not others and different kinds of exercise program will suit different people. It is important to bear in mind that a conventional randomized clinical trial of particular weight-loss programs will have recruited only those individuals prepared to follow any of the programs under study and will be likely to have failed to recruit those patients with a strong preference for one program if the program (or a similar program) was available outside the trial. Where a new program is only available within a trial, the patients are likely to have marked preferences for that program, and disappointment will follow if randomization allocates the non-preferred treatment. Subsequent responses, which may include dropping out of the trial, will undermine the assumptions of a randomized trial. Such trials, attempting to answer the, perhaps inappropriate, question, 'Which weight loss program works best?' may be less useful in informing routine clinical practice than preference trials or partially-randomized preference trials, where patients with preferences are encouraged to follow the treatment they prefer and only those without preferences are invited to accept allocation of treatment by randomization. In such preference trials the question of interest is 'Which weight loss program works best for which patients?' (Bradley, 1993).

Some studies have begun to look at reasons for success and failure in weight loss. National survey data in the USA were used to investigate self-reported weight loss and it was found that intentional weight loss in people with diabetes was associated with having been hospitalized twice in the previous year, and having seen a dietitian (Will, 1995) suggesting that a strong motivation to lose weight is important for success. Wing has shown that men lost weight faster than women but found no association between weight loss and whether or not subjects were taking insulin, nor between weight loss and duration of diabetes (Wing, Shoemaker, Marcus, McDermott & Gooding, 1990). Thus use of insulin and diabetes of long duration do not appear to

present particular obstacles to weight loss. In a later study, Wing and colleagues found smaller weight losses in blacks than whites related to faster regaining of weight in blacks (Wing & Anglin, 1996). Pierce and Armstrong (1996) found that in Afro-Caribbean diabetic patients the prescribed diets were held in contempt and largely ignored as being so mean as to be incompatible with a healthy life. Moreover the subjects profoundly disagreed with the medical notion of a healthy body weight. Large body size was perceived as healthy and attractive within this group and valued as such.

4.7.7. Treatment of Sexual Dysfunction

The vascular and neuropathic complications of diabetes are associated with a higher incidence of sexual problems among men with diabetes than among non-diabetic men. Women with diabetes may also experience more sexual difficulties than non-diabetic women though not all studies have found differences between women with and without diabetes (Jensen, 1981). It has been erectile failure in men with prevalence of 35-59% (Bancroft & Gutierrez, 1996) which has attracted the attention of clinicians, researchers and pharmaceutical companies. The potential contribution of psychologists in dealing with the sexual problems of men with diabetes have been little recognized. The increased likelihood of an organic component to erectile dysfunction associated with diabetes has led to a disproportionate emphasis on drug (self-administered papaverine injections to induce erection), mechanical (vacuum tumescence), and surgical interventions (penile implants) vigorously promoted by the manufacturers. Although diabetic men are at least, if not more, likely than other men to have impotence of psychogenic origins, the ready availability of funding for trials of drug and surgical interventions has encouraged and facilitated such approaches while funding for trials of psychological therapies is far less readily available and such studies are few and far between. Nevertheless, there is evidence that good results can be achieved with psychological approaches. Veves, Webster, Chen, Payne & Boulton (1995) reported that psychosexual counseling resulted in successful intercourse in 17 (60%) of the 24 men opting for this treatment. A further 7 (25%) in whom the marital relationship had

previously been unstable, went on to medical treatment after marital therapy. The success rate for psychosexual interventions compared favorably with the success rate of (55%) for those opting for papaverine injections and did not have the unwanted consequences experienced by 15% of men using papaverine which included priapism, pain at injection site, and dizziness. However, the modest success rates of psychosexual counseling with unselected patients reported by McCulloch, Hosking & Tobert (1986), have been widely quoted as reason for pursuing biomedical approaches to treatment. Increasing experience of the failure of surgical implants and complications following surgery have led to the cautious view that surgery should be viewed as a 'last resort' (Alexander, 1990; Dunsmuir & Holmes, 1996). Furthermore there is increasing emphasis on the importance of psychosexual counseling prior to biomedical interventions (Dunsmuir & Holmes).

Several authors have noted an unwillingness among their patients to view their erectile dysfunction as psychogenic in origin, and the limited enthusiasm for psychosexual counseling (Alexander, 1990; McCulloch et al.1996; Veves, et al. 1995). How much of this resistance is due to physicians' emphasis on biomedical approaches and limited appreciation of psychological approaches is unclear. An editorial in the British Medical Journal recommended that patients with erectile impotence "should be told that impotence in diabetes has a physical cause and will not improve without a physical treatment" (Price, 1993). Others have taken issue with this view (Bancroft & Gutierrez, 1996), but it is clear that this widespread exclusive focus on erectile dysfunction as an organic problem in diabetes will present a challenge to those wishing to encourage psychological approaches.

Meisler, Carey, Lantinga & Krauss (1989) have offered guidelines for a multidisciplinary approach to assessment of sexual dysfunction. Further development and wider use of psychological interventions has the potential for improved psychosexual functioning and prevention of the often irreversible complications of biosurgical interventions.

4.8. CULTURAL ISSUES

Cultural issues have been referred to at various points throughout the chapter along with life span, age and gender issues where each has been particularly relevant. Sections of the chapter concerned with risk factors for diabetes review evidence showing diabetes to be less common in white western populations than in other populations. The high incidence of diabetes among racial minorities in Western society present particular challenges to diabetes care services when language barriers pose particular problems of management for a disorder which requires complex self-management tasks and dietary change. Good communication between health professionals and their patients is central to the development of self-management skills and appropriate practical solutions need to be found to facilitate communication across languages with multi-lingual health professionals or use of translators familiar with diabetes and its management. Clashes between cultural beliefs, lifestyle and recommendations for diabetes management, especially dietary recommendations, create particular difficulties where weight reduction is an important component of the recommended management plan for overweight people with NIDDM (see section 4.7.6).

4.9. PROFESSIONAL ISSUES

In the USA, involvement of psychologists in diabetes care teams is now standard practice. In the UK it is becoming increasingly common to find psychologists attached to diabetes care teams for two or more sessions per week although this is still the exception rather than the rule. A recent Briefing Paper from the British Psychological Society (British Psychological Society, Division of Clinical Psychology, 1994) on services for people with diabetes mellitus recommended one whole time equivalent post for each local diabetes service allocated between adult services, services to the elderly and services to children and young people. It was recognized that clinical psychologists who work with adults will not normally have the skills and knowledge to work effectively with children and their families and that special expertise may also be required for

some older people. Such recommendations are realistic rather than ideal. This level of input is likely to be mostly taken up with crisis intervention and problem solving rather than preventive work to improve communication, knowledge and skills in diabetes management across the service as a whole which in the longer term would be of most benefit.

There is considerable scope for clinical psychologists to make valuable contributions to diabetes management, providing assessment and treatment and conducting research and evaluation as well as teaching other health professionals and patients. Several specific psychological therapies which clinical psychologists may be able to offer include stress management and relaxation training, weight management interventions and management of eating disorders and sexual dysfunction. More general psychological skills will be valuable in helping patients to identify and overcome barriers to self care and in facilitating the process of helping patients and their advisors to negotiate manageable treatment regimens which are tailored to individuals' lifestyles and preferences.

For clinical psychologists to be most effective in diabetes care, detailed understanding is required of diabetes and the varieties of management possible. The management regimens recommended in a particular clinic are likely to be only a small minority of those which could be made available. There are many diabetologists who continue to recommend particular doses and timing of insulin which then dictate the timing of meals and snacks and require patients to manipulate their diet and exercise to fit in with the regimen prescribed. Initially it may well be more demanding to teach patients how to manipulate their insulin appropriately to fit in with preferred meal times and varying levels of energy expenditure but in the longer term it is a great deal less demanding and more convenient for the patient, their family, friends and other associates if they adjust their insulin dose rather than their life (Howorka, 1996). Problems with poor diabetes control which diabetologists may construe as 'compliance' problems may more productively be construed as inflexible treatment recommendations and/or inadequate education in diabetes management and self-care skills in adjusting insulin doses and timing to accommodate a flexible lifestyle. By developing an understanding of how diabetes can be managed physiologically and

by appreciating the range of different medical approaches to diabetes management, clinical psychologists are better placed to apply their psychology to the task of improving diabetes management while at the same time protecting, and even improving, the psychological well-being and quality of life of people with diabetes.

Table 1 WHO (1985) Classification of Diabetes Mellitus

1. Insulin-dependent diabetes mellitus (IDDM)
2. Non-insulin-dependent diabetes mellitus (NIDDM)
    a. Non-Obese
    b. Obese
3. Malnutrition-related disorders
4. Other types of diabetes associated with certain conditions and syndromes including: pancreatic disease; diseases of hormonal etiology; drug induced or chemical-induced conditions; abnormalities of insulin or its receptors; certain genetic syndromes.
5. Gestational diabetes mellitus

NIDDM and IDDM are the most common forms of diabetes in first-world countries with gestational diabetes also being common.

REFERENCES

Aikens, J.E., Wallander, J.L. Bell, D.S.H. & McNorton, A. (1994). A Nomothetic-Idiographic Study of Daily Psychological Stress and Blood Glucose in Women with Type 1 Diabetes Mellitus. Journal of Behavioral Medicine, 17, 535-548.

Akerblom, H.K.,Savilahti, E., Saukkonen, T.T., Paganus, A., Virtanen, S.M., Terami, K., Knip, M., Ilonen, J., Reijonen, H., Karjalainen, J., Vaarala, O. & Reunanen, A. (1993). The case for elimination of cows' milk in early infancy in the prevention of Type 1 diabetes: the Finnish experience. Diabetes/Metabolism Reviews, 9, 269-278.

Alexander, W. D. (1990). The diabetes physician and an assessment and treatment program for male erectile impotence. Diabetic Medicine, 7, 540-543.

Alexander, W.D. (1993). Human insulin: lessons from the UK? The Medical Journal of Australia, 159, 75-76.

American Diabetes Association. (1992). Screening for diabetic retinopathy. Diabetes Care, 15, (Suppl. 2), 16-18.

Anderson, R.M. (1995). Patient empowerment and the traditional medical model: A case of irreconcilable differences? Diabetes Care, 18, 412-415.

Anderson, R.M., Funnell, M.M., Barr, P.A., Dedrick, R.F. & Davis, W.K. (1991). Learning to empower patients: Results of professional education program for diabetes educators. Diabetes Care, 14, 584-90.

Anderson, R.M., Funnell, M.M., Butler, P.M., Arnold, M.S., Fitzgerald, J.T. & Feste, C.C. (1995). Patient Empowerment: Results of a randomized controlled trial. Diabetes Care, 18, 943-949.

Anderson, R.M., Hiss, R.G., Stepien, C.J., Fitzgerald, J.T. & Funnel, M.M. (1994). The diabetes education experience of randomly selected patients under the care of community physicians. Diabetes Educator, 20, 399-405.

Bancroft, J. & Gutierrez, P. (1996). Erectile dysfunction in men with and without diabetes mellitus: a comparative study. Diabetic Medicine, 13, 84-89.

Barendse, S. (1995). Doctors' intentions to use psychological measures in diabetes clinics: a field evaluation of the theory of reasoned action. Project Report. Department of Psychology, Royal Holloway, University of London.

Barglow, P., Hatcher, R., Edidin, D.V. & Sloan-Rossiter, M. (1984). Stress and metabolic control in diabetes: psychosomatic evidence and evaluation of methods. Psychosomatic Medicine, 46, 127-144.

Beaty, T.H., Neel, J.V. & Fajans, S.S. (1982). Identifying risk factors for diabetes in first degree relatives of non- insulin-dependent diabetes. American Journal of Epidemiology, 115, 380-397.

Beck, A.T., Ward, C.H., Mendelson, M., Mock, J. & Erbaugh, J. (1961). An inventory for measuring depression. Archives of General Psychiatry, 4, 561-71.

Beeney, L.J., Dunn, S.M. & Welch, G. (1994). Measurement of diabetes knowledge: the development of the DKN scales. In C . Bradley (Ed.) Handbook of Psychology and Diabetes: a guide to psychological measurement in diabetes research and practice. 159-191. Chur, Switzerland: Harwood Academic Publishers.

Biggs, M.M., Basco, M.R., Patterson, G., & Raskin, P. (1994). Insulin withholding for weight control in women with diabetes. Diabetes Care, 17, 1186-9.

Bingley, P.J., Bonifacio, E. & Gale, E.A.M. (1993). Can we really predict IDDM? Diabetes, 42, 213-220.

Bingley, P.J. & Gale, E.A.M. (1989). Rising incidence of IDDM in Europe. Diabetes Care, 12, 289-295.

Blohmé, G., Nyström, L., Arnqvist, H.J., Lithner, F., Littorin, B., Olsson, P.O., Schertén, B., Wibell, L. & Östman J. (1992). Male predominance of Type 1 (insulin-dependent) diabetes mellitus in young adults: results from a 5-year prospective nationwide study of the 15-34 year age group in Sweden. Diabetologia, 35, 56-62.

Bradley, C. (1988). Stress and diabetes. In S. Fisher & J. Reason (Eds) Handbook of life stress, cognition and health. Chichester: John Wiley, 383-401.

Bradley, C. (1989). Psychosocial influences and adjustment: family and health care systems. In R, Larkins, P. Zimmet & D, Chisholm (Eds.) Diabetes 1988. Elsevier Science Publishers, B.V.

Bradley, C. (1993). Designing medical and educational intervention studies: a review of some alternatives to conventional randomized controlled trials. Diabetes Care, 16: 509-518.

Bradley, C. (1994a). The contribution of psychology diabetes managment. British Journal of Clinical Psychology, 33, 11-21

Bradley, C. (1994b). The Well-being Questionnaire. In: C. Bradley (Ed.) Handbook of Psychology and Diabetes: a guide to psychological measurement in diabetes research and practice. pp89-109. Chur Switzerland: Harwood Academic Publishers,.

Bradley, C. (1994c). The Diabetes Treatment Satisfaction Questionnaire (DTSQ). In C Bradley (Ed.) Handbook of Psychology and Diabetes: a guide to psychological measurement in diabetes research and practice. pp.111-132. Harwood Academic Publishers, Chur Switzerland,

Bradley, C. (1994d). What about quality of life? Balance, 140, June/July, Special issue on the implications of the Diabetes Control and Complications Trial, ppVIII-IX.

Bradley, C. (1994e) Measures of perceived control of diabetes. In C . Bradley (Ed.) Handbook of Psychology and Diabetes: a guide to psychological measurement in diabetes research and practice. 291-331. Chur, Switzerland: Harwood Academic Publishers.

Bradley, C. (Ed.) (1994f) Handbook of Psychology and Diabetes: a guide to psychological measurement in diabetes research and practice. Chur, Switzerland: Harwood Academic Publishers.

Bradley, C. (1995). Health beliefs and knowledge of patients and doctors in clinical practice and research. Patient Education and Counseling, 26, 99-106.

Bradley, C. (1996). Measuring quality of life in diabetes. In S.M. Marshall, P.D. Home & R.A. Rizza (Eds.) The Diabetes Annual, 10, 207-224. Amsterdam: Elsevier Science B.V.

Bradley, C. (1996). The WHO (Ten) Well-being Index: A critique. Psychotherapy and Psychosomatics, 65, 331-333.

Bradley, C. (1997). Design of a renal-dependent individualised quality of life questionnaire. Advances in Peritoneal Dialysis, 13, 116-120.

Bradley, C., Christie, M. & Home, P. (Eds.) (1991). The Technology of Diabetes Care: Converging Medical and Psychosocial Perspectives. Chur, Switzerland: Harwood Academic Publishers.

Bradley, C., & Gamsu, D.S. (1994). Guidelines for encouraging psychological well-being. Report of a working group of the World Health Organization regional office for Europe and International Diabetes Federation European St. Vincent Declaration action program for diabetes. Diabetic Medicine, 11, 510-516

Bradley, C. & Gamsu, D.S. (1995). Diabetes: psychosocial issues in adolescents and adults. Current Opinion in Psychiatry, 6, 400-406.

Bradley, C., Gamsu, D.S., Knight, G., Boulton, A.J.M. & Ward, J.D. (1986). Predicting risk of diabetic ketoacidosis in patients using continuous subcutaneous insulin infusion. British Medical Journal, 293, 242-243.

Bradley, C., Gamsu, D.S., Moses, J.L., Knight, G., Boulton, A.J.M., Drury, J. & Ward, J.D. (1987). The use of diabetes-specific perceived control and health belief measures to predict treatment choice and efficacy in a feasibility study of continuous subcutaneous insulin infusion pumps. Psychology and Health, 1, 133-146.

Bradley, C. & Lewis, K.S. (1990). Measures of psychological well-being and treatment satisfaction developed from the responses of people with tablet-treated diabetes. Diabetic Medicine, 7, 445-51.

Bradley, C., Moses, J.L., Gamsu, D.S., Knight, G. and Ward, J.D. (1985). The effects of relaxation on metabolic control of Type 1 diabetes; a matched controlled study. Diabetes, 34, 17A.

Bradley, C., Todd, C., Gorton, T., Plowright, R.E.A. & Symonds, E. (1995) A patient-centered measure of the impact of a chronic disorder on quality of life: design and development of the ADDQoL. International Conference and Exhibition: Scientific Basis of Health Services. London.

British Psychological Society, Division of Clinical Psychology.(1994) Purchasing clinical psychology services: services for people with diabetes mellitus. Briefing paper No. 4. British Psychological Society.

Deleted: 2

Deleted: 32

Bruno, G., Merletti, F., Vuolo, A., Pisu, E., Giorio, M. & Pagano, G. (1993). Sex differences in incidence of IDDM in age group 15-29yr. High risk in males in Province of Turin, Italy. Diabetes Care, 16, 133-136.

Cantwell, R., & Steel, J.M. (1996). Screening for eating disorders in diabetes mellitus. Journal of Psychosomatic Research, 40, 15-20.

Carter, W.R., Gonder-Frederick, L.A., Cox , D.J., Clarke, W.L. & Scott, P. (1985). Effects of stress on blood glucose in IDDM. Diabetes Care, 8, 411-412.

Chase, H.P. & Jackson, G.G. (1981). Stress and sugar control in children with insulin-dependent diabetes. Journal of Pediatrics, 98, 1011-1013.

Clayer, J.R., Bookless-Pratz, C. & Harris, R.L. (1985). Some health consequences of a natural disaster. The Medical Journal of Australia, 143, 182-184.

Cox, D.J., Gonder-Frederick, L., Anderson, R., Schlundt, D., Jacobson, A., Marerro, D. & Clarke, W. (1994) Professionals' beliefs about useful symptoms of hypoglycaemia. Diabetes Care, 17, 776-777.

Cox, D.J., Gonder Frederick, L.A., Julian, D.M. & Clarke, W.L. (1992) Blood Glucose Awareness Training Manual. Charlottesville: University of Virginia.

Cox, D.J., Gonder-Frederick, L.A., Julian, D & Clarke, W. (1994). Long-term follow-up evaluation of blood glucose awareness training. Diabetes Care, 17, 1-5.

Cox, D.J.,Gonder-Frederick, L.A., Julian, D.M., Cryer, P., Lee, J.A., Richards, F.E. & Clarke, W.L. (1991). Intensive versus Standard Blood Glucose Awareness Training (BGAT) with insulin-dependent diabetes: mechanisms and ancillary effects. Psychosomatic Medicine, 53, 453-462

Cox, D., Gonder-Frederick, L., Kovatchev, B., Polonsky, W., Schlundt, D., Julian, D. & Clarke, W. (1995). Reduction of severe hypoglycaemia (SH) with blood glucose awareness training (BGAT-2). Diabetes, 44 (Suppl), 27A.

Cox, D., Gonder-Frederick, L., Polonsky, W., Schlundt, D., Julian, D. & Clarke, W. (1995). A multi-center evaluation of blood glucose awareness training - II. Diabetes Care, 18, 523-528.

Cranston, I., Lomas, J., Maran, A., Macdonald, I. & Amiel S.A. (1994) Restoration of hypoglycaemia awareness in patients with long-duration insulin-dependent diabetes. Lancet, 344, 283-287.

DCCT Research Group 1993. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. New England Journal of Medicine, 329, 977-986.

Deary, I.J., Hepburn, D.A., Macleod, K.M., & Frier, B.M. (1993). Partitioning the symptoms of hypoglycemia using multisample confirmatory factor analysis. Diabetologia, 36, 771-777.

DeGood, D.E. & Redgate, E.S. (1982). Interrelationships of plasma cortisol and other activation indices during EMG biofeedback training. Journal Behavioral Medicine, 5, 213-223.

DERIG (Diabetes Epidemiology Research International Group) (1988). Geographic Patterns of Childhood Insulin-Dependent Diabetes Mellitus. Diabetes, 37, 1113-1119.

Diabetic Retinopathy Study Group. (1981). Photocoagulation for proliferative diabetic retinopathy. Clinical application of Diabetic Retinopathy Study (DRS) findings. DRS report No. 8. Opthalmology, 88, 583-600.

Doherty,Y., Hall, D., James, P.T., & Roberts, S.H. (1996). Teaching change counseling skills to a multidisciplinary diabetes team: lessons learnt. Diabetic Medicine, 13, (suppl 7), S4.

Dotta, F., Dionisi, S., Farilla, L. & DiMario, U. (1996). The use of immunological markers in IDDM after more than 20 years from ICA description. Diabetes/Metabolism Reviews, 12, 121-126.

Dunsmuir, W.D. & Holmes, S.A. (1996). The aetiology and management of erectile, ejaculatory, and fertility problems in men with diabetes mellitus. Diabetic Medicine, 13, 700-708.

Edwards, C. & Yates, A.J. (1985). The effects of cognitive task demand on subjective stress and blood glucose levels in diabetic and non-diabetic subjects. Journal of Psychosomatic Research, 29, 59-69.

Eisenbarth, G.S., Verge, C.F., Allen, H. & Rewers, M.J. (1993). The design of trials for prevention of IDDM. Diabetes, 42, 941-947.

Elliott, R.B. & Chase, H.P. (1991). Prevention or delay of Type 1 (insulin-dependent) diabetes mellitus in children using nicotinamide. Diabetologia, 34, 362-365.

Erikksonn, K.F. & Lindegarde, F. (1991). Prevention of Type II (non-insulin-dependent) diabetes mellitus by diet and physical exercise. The 6-year Malmo feasibility study. Diabetologia, 34, 891-898.

Fairburn, G.C., Peveler, R.C., Davies, B., Mann, J.I., & Mayou, R.A. (1991). Eating disorders in young adults with insulin-dependent-diabetes mellitus: a controlled study. British Medical Journal, 303, 17-20.

Fanelli, C.G., Epifano, L., Rambotti, A.M., Pampanelli, S., Divincenzo, A., Mondarelli, F., Lepore, M., Annibale, B., Ciofetta, M., Bottini, P., Porcellati, F., Scionti, L., Santeusanio, F., Brunetti, P., & Bolli, G.B. (1993) Meticulous prevention of hypoglycaemia normalises the glycaemic thresholds and magnitude of most neuroendocrine responses to, symptoms of, and cognitive function during hypoglycemia in intensively treated patients with short-term IDDM. Diabetes, 42, 1683-1689.

Feinglos, M.N., Hastedt, P. & Surwit, R.S. (1987). Effects of relaxation therapy on patients with Type I Diabetes Mellitus. Diabetes Care, 10, 1,72-75.

Felber, J.P., Acheson, K.J. & Tappy, L. (1992). From Obesity to Diabetes. Chichester: Wiley.

Feste, C. (1992). A practical look at patient empowerment. Diabetes Care, 15, 922-925.

Gamsu, D.S. & Bradley, C. (1987). Clinical staff's attributions about diabetes: scale development and staff vs patient comparisons. Current Psychological Research and Reviews, 6: 69-78.

Gerstein, H.C. (1994). Cow's milk exposure and Type I Diabetes Mellitus. Diabetes Care, 17, 13-19.

Gillespie, C.R. & Bradley, C. (1988). Causal attributions of a doctor and patients in a diabetes clinic. British Journal of Clinical Psychology, 27, 67-76.

Glasgow, R.E. (1991). Compliance to diabetes regimens: conceptualization, complexity and determinants. In J.A. Cramer & B. Spilker (Eds.) Patient compliance in medical practice and clinical trials. New York: Raven Press.

Glasgow, R.E. (1994). Social-environmental factors in diabetes: barriers to diabetes self-care. pp.335-349. In C . Bradley (Ed.) Handbook of Psychology and Diabetes: a guide to psychological measurement in diabetes research and practice. Chur, Switzerland: Harwood Academic Publishers.

Glasgow, R.E., Toobert, D.J. Hampson, S.E. & Wilson, W. (1995). Examples of programmatic research in behavioral medicine. Annals of Behavioral Medicine, 17, 32-40.

Goetsch,V.L., Abel J.L. & Pope, M.K. (1994). The effects of stress, mood and coping on blood glucose in NIDDM: A prospective pilot evaluation. Behavioral Research and Therapy, 32, 503-510.

Gold, A.E., Deary, I.J., Jones, R.W., O'Hare, J.P., Reckless, D.J. & Frier, B.M. (1994). Severe deterioration in cognitive function and personality in five patients with long-standing diabetes: a complication of diabetes or a consequence of treatment? Diabetic Medicine, 11, 499-505.

Gonder-Frederick, L.A., Carter, W.R., Cox , D.J. & Clarke, W.L. (1990). Environmental stress and blood glucose control in IDDM. Health Psychology, 9, 503-515.

Gorus, F.K., Anselmo, J. & Keymeulen, B. (1993). Type 1 (insulin-dependent) diabetes mellitus: an autoimmune, predictable and preventable disease? Lessons from national registries and new challenges to clinical biology. Acta Clinica Belgica, 48, 96-108.

Gorus, F.K., Goubert, P., Semakula, C., Vandewalle, C.l., De Schepper, J., Scheen, A., Christie, M.R., Pipeleers, D.G., et. al. for the Belgian Diabetes Registry (1997). IA-2-autoantibodies complement GAD65-autoantibodies in new-onset IDDM patients and help predict impending diabetes in their siblings. Diabetologia, 40, 95-99.

Green, A., Gale, E.A.M. & Patterson C.C. for the Eurodiab Ace Study Group. (1992). Incidence of childhood-onset insulin-dependent diabetes mellitus: the Eurodiab Ace Study. Lancet, 339, 905-909.

Greenhalgh, J. (1996). (The role of) User outcomes within diabetes care. Outcomes Briefings, 8, 10-16. U.K. clearing house on health outcomes. Leeds.

Greenhalgh, P.M., Jones, J.R., Jackson, C.A., Smith C.C.T. & Yudkin, J.S. (1991). Changes in injection-site blood flow and plasma free insulin concentrations in response to stress in Type 1 diabetic patients. Diabetic Medicine, 9, 20-29.

Greenhalgh, T., Chowdhury, M. & Helman, C. (1996). Applying the patient empowerment model to non-white ethnic groups: a qualitative survey of health beliefs, illness experience, and barriers to change in British Bangladeshis with diabetes (Abstract). Diabetic Medicine, 13, supp7, S5

Hagopian, W.A., Sanjeevi, C.B., Kockum, I., Landin-Olsson, M., Karlsen, A.E., Sundkvist, G., Dahlquist, G., Palmer, J. and Lernmark, Å. (1995). Glutamate decarboxylase-, insulin- and islet-cell antibodies and HLA typing to detect diabetes in a general population-based study of Swedish children. Journal of Clinical Investigation, 95, 1505-1511.

Hales, C.N. & Barker, D.J. (1992). Type 2 (non-insulin-dependent diabetes mellitus): the thrifty phenotype hypothesis. Diabetologia, 35, 595-601.

Halford, W.K., Cuddihy, S. & Mortimer, R.M. (1990). Psychological stress and blood glucose regulation in Type 1 diabetic patients. Health Psychology, 9, 516-528.

Hanson , S.L. & Pichert, J.W. (1986). Perceived stress and diabetes control in adolescents. Health Psychology, 5, 439-452.

Harris, M.I. (1984). for the National Diabetes Data Group. Unpublished data from the 1976-80 US National Health and Nutrition Examination Survey, National Center for Statistics.

Haynes, R.B., Wang, E. & Gomes, M.D. (1987). A critical review of interventions to improve compliance with prescribed medications. Patient Education and Counseling, 10, 155-166.

Helmrich, S.P., Ragland, D.R., Taylor, D.W., Leung, R.W., & Paffenbarger, R.S. Jnr. (1991) Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. New England Journal of Medicine, 325, 147-52.

Herbert, C.P. (1996). Community-based research as a tool for empowerment: The Haida Gwaii Diabetes Project Example. Canadian Journal of Public Health, 87, 109-112.

Herskowitz, R.D., Jackson, R.A., Soeldner, J.S. & Eisenbarth, G.S. (1989). Pilot trial to prevent Type 1 diabetes: progression to overt IDDM despite oral nicotinamide. Journal of Autoimmunity, 2, 733-737.

Hickey, A.M., Bury, G., O'Boyle, C.A., Bradley, F., O'Kelly, F.D. & Shannon, W. (1996). A new shorot-form individual quality of life measure (SEIQoL-DW): application in a cohort of individuals with HIV/AIDS. British Medical Journal, 313, 29-33.

Hildebrandt, P., Mehlsen, J., Sestoft,L. & Nielson,S.L. (1985). Mild mental stress in diabetes: changes in heart rate and subcutaneous blood flow. Clinical Physiology, 5, 371-376.

Holmes, C.S., Hayford, J.T., Gonzalez, J.L., & Weydert, J.A.(1983) A survey of cognitive-functioning at different glucose levels in diabetic persons. Diabetes Care, 6, 180-185.

Howorka, K. (1996) Functional Insulin Therapy. Principles, teaching approach, and practice. (2nd ed.). Berlin, Germany: Springer Verlag.

Irvine, A., Cox, D. & Gonder-Frederick, L. (1994). The Fear of Hypoglycaemia Scale. In C . Bradley (Ed.) Handbook of Psychology and Diabetes: a guide to psychological measurement in diabetes research and practice. 133-155. Chur, Switzerland: Harwood Academic Publishers.

Jacobson, A.M. and the DCCT Research Group. (1994). The Diabetes Quality of Life Measure. In C . Bradley (Ed.) Handbook of Psychology and Diabetes: a guide to psychological measurement in diabetes research and practice. 65-87. Chur, Switzerland: Harwood Academic Publishers.

Jarret, R.J., Keen, H., Fuller, J.H., & McCartney, M. (1979) Worsening of diabetes in men with impaired glucose tolerance borderline diabetes. Diabetologia, 16, 25-30.

Jennings, A.M., Lewis, K.S., Murdoch, S., Talbot, J.F., Bradley, C. & Ward, J.D. (1991). Randomized trial comparing continuous subcutaneous insulin infusion and conventional insulin therapy in Type II diabetic patients poorly controlled with sulphonylureas. Diabetes Care, 14, 738-744.

Jensen, S. B. (1981). Diabetic sexual dysfunction: a comparative study of 160 insulin treated diabetic men and women and an age-matched control group. Archives of Sexual Behavior, 10, 493-504.

Jevning, R., Wilson, A.F. & Davidson, J .M. (1978). Adrenocortical activity during meditation. Hormones and Behavior, 10, 54-60.

Karvonen, M., Tuomilehto, J., Libman, I. & LaPorte, R., for the World Health Organization DIAMOND Project Group. (1993). A review of the recent epidemiological data on the worldwide incidence of Type I (insulin-dependent) diabetes mellitus. Diabetologia, 36, 883-892.

Keen, H., Jarret, R.J., & McCartney, M. (1982). The ten-year follow-up of the Bedford survey 1962-72: glucose tolerance and diabetes. Diabetologia, 22, 73-78.

Keller, R.J., Eisenbarth, G.S. & Jackson R.A. (1993). Insulin prophylaxis in individuals at high risk of Type I diabetes. Lancet, 341, 927-928.

Kemmer, F.W., Bisping, R., Steinberger, H.J., Baar, H., Hardtmann, F., Schlaghecke, R. & Berger, M. (1986). Psychological stress and metabolic control in patients with Type 1 diabetes mellitus. New England Journal of Medicine, 314, 1078-1084.

Kinmonth, A-L., Woodcock, A..J., Griffin, S., Campbell, M., Spiegal, N., Burgess, H. (1996). Diabetes care from diagnosis: development of a patient-centered approach to primary care of newly diagnosed non-insulin dependent diabetes mellitus for evaluation in a randomized controlled trial. Diabetic Medicine 13, (suppl 7), S13.

Knight, G. Boulton, A.J.M., Drury, J., Gamsu, D.S., Moses, J.L., Bradley, C. & Ward, J.D. (1984). A feasibility study of the use of continuous subcutaneous insulin infusion in a diabetic clinic: Patients' choice of treatment. Diabetic Medicine, 1, 267-272.

Knowler, W.C., Sartor, G. & Schersten, B. (1987). Effects of glucose tolerance and treatment of abnormal tolerance on mortality in Malmohus County Sweden. Diabetologia, 30, 541A.

Kohlmann, C.W., Schuler, M., Petrak, F., Kustner, E., Kronhne, H.W. & Beyer, J. (1993). Associations between type of treatment and illness-specific locus of control in Type 1 diabetes patients. Psychology and Health., 8, 383-391.

Krans HMJ, Porta M, Keen H & Staehr Johansen K (1995) Diabetes Care and Research in Europe: the St Vincent Declaration action program. Diabetologia, 15, 40-7.

Krolewski, A.S., Warram, J.H., Rand, L.I., & Kahn, C.R. (1987). Epidemiologic Approach to the Etiology of Type I Diabetes Mellitus and its Complications. New England Journal of Medicine, 317, 1390-1398.

Laakso, M. & Pyörälä, K. (1985). Age of onset and type of diabetes. Diabetes Care, 8, 114-117.

Lammers, C.A., Naliboff, B.D. & Straatmeyer, A.J. (1984). The effects of progressive relaxation on stress and diabetic control. Behavior Research and Therapy, 22, 641-650.

Lane, J.D., McCaskill, C.C., Ross, S.L., Feinglos, M.N. & Surwit, R.S. (1993). Relaxation Training for NIDDM: Predicting who may benefit. Diabetes Care, 16, 1087-1094.

Leslie, R.D.G. & Elliott, R.B. (1994). Early Environmental Events as a Cause of IDDM. Diabetes, 43, 843-850.

Lewis, K.S. & Bradley, C. (1994). Measures of diabetes-specific health beliefs. In C . Bradley (Ed.) Handbook of Psychology and Diabetes: a guide to psychological measurement in diabetes research and practice. (pp. 247-289). Chur, Switzerland: Harwood Academic Publishers.

Mathew, R.J., Ho, B.T, Kralik, P., Taylor, D. & Claghorn, J.L. (1980). Catecholamines and migraine: evidence based on biofeedback induced changes. Headache, 20, 247-252.

Mathew, R.J. , Ho, B.T., Kralik, P., Taylor, D., Semchuk, K., Weinman, M. & Claghorn, J.L. (1980). Catechol-O -methyltransferase and catecholamines in anxiety and relaxation. Psychiatric Research, 3, 85-91.

McColl, E., Steen, I.N., Meadows, K.A., Hutchinson, A., Eccles, M.P., Hewison, J., Fowler, P. and Blades, S.M. (1995). Developing outcome measures for ambulatory care: an application to asthma and diabetes. Social Science and Medicine, 41, 1339-48.

McCulloch, D.K., Hosking, D.J. & Tobert, A. (1986) A pragmatic approach to sexual dysfunction in diabetic men: psychosexual counseling. Diabetic Medicine, 3: 485-9.

McGee, H.M. O'Boyle, C.A., Hickey, A., O'Malley, K.M. & Joyce, C.R.B. (1991). Assessing the quality of life of the individual: the SEIQoL with a healthy and a gastroenterology unit population. Psychological Medicine, 21, 749-759.

McGrady, A., Bailey, B.K. & Good, M.P. (1991). Controlled study of biofeedback - assisted relaxation in Type 1 diabetes. Diabetes Care, 14, 360-365.

McGrady, A. & Gerstenmaier, L. (1990). Effect of biofeedback assisted relaxation training on blood glucose levels in a Type 1 insulin-dependent diabetic: A case report. Journal of Behavior Therapy and Experimental Psychiatry, 21, 69-75.

Meisler, A.W. Carey, M.P., Lantinga, L.J. & Krauss, D.J. (1989). Erectile dysfunction in diabetes mellitus: a biopsychosocial approach to etiology and assessment. Annals of Behavioral Medicine, 11, 18-27.

Moberg, E., Kollind, M., Lins, P.E., & Adamson, U. (1994). Acute Mental Stress Impairs Insulin Sensitivity in IDDM patients. Diabetologia, 37, 247-251.

Mulhauser, I. (1990). Smoking and diabetes. Diabetic Medicine, 7, 10-15.

Naliboff, B.D., & Cohen, H.J. & Sowers, J.D. (1985). Physiological and metabolic responses to brief stress in non-insulin dependent diabetic and control subjects. Journal of psychosomatic research, 1985, 29, 367-374.

Neel, J.V. (1982). The thrifty genotype revisited. In J. Kobberling & R. Tattershall (Eds.) The genetics of diabetes mellitus. London: Academic Press.

Newman, B., Selby, J.V., King, M.C., Slemenda, C., Fabsitz, R. & Friedman, G.D. (1987). Concordance for type 2 (non-insulin-dependent) diabetes mellitus in male twins. Diabetologia, 30, 763-68.

Nordenfelt, L. (1996). Prevention and Ethics in Medicine: The case of diabetes prevention. Journal of Pediatric Endocrinology and Metabolism, 9, 381-386.

North, J. (1991) The pen-injector's impact on self-management. In C. Bradley, M. Christie and P. Home (Eds.) The Technology of Diabetes Care: converging medical and psychosocial perspectives. Pp 84-88. Chur, Switzerland: Harwood Academic Publishers.

O'Boyle, C.A., McGee, H.M., Hickey, A. O'Malley, K. & Joyce, C.R,B, (1992). Individual quality of life in patients undergoing hip replacement surgery. Lancet, 339, 1088-1091.

Palmer, J.P. & McCulloch, D.K. (1991). Prediction and prevention of IDDM - 1991. Diabetes, 40, 943-947.

Pan, X., Guanwei, L., Zuoxin, A., Jixing, W., Bennett, P.H. & Howard, B.V. (1994). Effect of dietary and/or exercise on the incidence of diabetes in subjects with IGT- The Da Qing IGT and Diabetes Study. Paper given at the 15th International Diabetes Federation , Kobe, Japan.

Peveler, R.C., Fairburn, G.C., Boller, I., & Dunger, D. (1992). Eating disorders in Adolescents with IDDM. Diabetes Care, 15, 1356-1360.

Pierce, M. & Armstrong, D. (1996). Afro-Caribbean lay beliefs about diabetes: an exploratory study. In K. Kelleher & S. Hillier (Eds.) Researching Cultural Differences in Health. Pp 97-102. London: Routledge.

Pierce, M., Keen, H. & Bradley, C. (1995). Risk of diabetes in offspring of parents with non-insulin-dependent diabetes. Diabetic Medicine, 12, 6-13.

Pipeleers, D., Keymeulen, B. & Korbutt, G. (1994). Islet Transplantation. In S.M. Marshall & P.D. Home (Eds.) The Diabetes Annual/8, pp 299-230. Amsterdam: Elsevier Science B.V.

Posner, T.R. (1992). Draft report to BDA LOW taskforce on letters about the changeover to human insulin. British Diabetic Association. London.

Pozzilli, P. & Andreani D. (1993). The Potential Role of Nicotinamide in the Secondary Prevention of IDDM. Diabetes/Metabolism Reviews, 9, 219-230.

Price, D.E. (1993). Managing impotence in diabetes. British Medical Journal. 307, 275-276.

Pyke, D.A. (1979). Diabetes : the genetic connections. Diabetologia, 17, 333-343.

Quatromoni, P.A., Milbauer, M., Posner, B.M., Carballeira, N.P., Brunt, M. & Chipkin, S.R. (1994). Use of focus groups to explore nutrition practices and health beliefs of urban Caribbean Latinos with diabetes. Diabetes Care, 17, 869-873.

Reaven, G.M. (1988). The role of insulin resistance in human disease. Diabetes, 37,1595-1607.

Riazi, A., Pickup, J. & Bradley, C. (1996). Blood glucose reactivity to stress: individual differences in magnitude, timing, and direction of responses. Diabetic Medicine, 13, Suppl.7, S5-6.

Rizza, R.A. Mandarion, L.J. & Gerich, J.E. (1982). Cortisol induced insulin resistance in man: impaired suppression of glucose utilization due to a postreceptor defect of insulin action. Clinical Endocrinology and Metabolism, 54, 131-138.

Robertson, R.P. (1992) Pancreatic and islet transplantation for diabetes - cures or curiosities? New England Journal of Medicine, 327, 1861-1868.

Rodin, G.M. & Daneman, D. (1992). Eating disorders and IDDM: a problematic association. Diabetes Care, 15, 1402-1412.

Rodin, G.M., Daneman, D., Johnson, L.E. Kenshole, A. & Garfinkel, P. (1985). Anorexia nervosa and bulimia in female adolescents with insulin dependent diabetes mellitus: a systematic study. Journal of Psychiatric Research, 19, 381-384.

Rosenbaum, L. (1983). Biofeedback-Assisted Stress Management for Insulin-Treated Diabetes Mellitus. Biofeedback and Self-Regulation, 8, 519-532.

Rosmark, B., Berne, C., Holmgren, S., Lago, C., Renholm, G. & Sohlberg, S. (1986). Eating disorders in patients with insulin dependent diabetes mellitus. Journal of Clinical Psychiatry, 47, 547-550.

Ryan, C. (1994). Measures of cognitive function. In C . Bradley (Ed.) Handbook of Psychology and Diabetes: a guide to psychological measurement in diabetes research and practice. Pp 191-219. Chur, Switzerland: Harwood Academic Publishers.

Saad, M.F., Knowler, W.C., Pettitt, D.J., Nelson, R.G., Mott, D.M. & Bennett, P.H. (1988). The natural history of impaired glucose tolerance in the Pima Indians. New England Journal of Medicine, 319, 1500-1506.

Sartor, G., Schersten, B., Carlstrom, S.,Melander,, A., & Persson, G.(1980). Ten-year follow -up of subjects with impaired glucose tolerance: prevention of diabetes by tolbutamide and diet regulation. Diabetes, 29, 154-157.

Seeburg, K.N. & DeBoer, K.F. (1980). Effects of EMG biofeedback on diabetes. Biofeedback and Self-regulation, 5, 289-293.

Smith, U. (1984). Insulin resistance in obesity, Type II diabetes and stress. Acta Endocrinologica, 105, suppl. 262, 67-69.

Stabler, B. (1993). On the role of patient. Journal of Pediatric Psychology, 18, 301-312.

Steel, J.M., Young, R.J., Lloyd, G.G. & Macintyre, C.C.A. (1989). Abnormal Eating attitudes in young insulin-dependent diabetics. British Journal of Psychiatry, 155, 515-521.

Surwit, R.S. & Feinglos, M.N. (1983). The effects of relaxation on glucose tolerance in non-insulin-dependent diabetes. Diabetes Care, 6, 176-179.

Sutherland, D.E.R., Gores, P.F., Hering, B.J., Wahoff, D., McKeehen,D.A. & Gruessner W.G. (1996). Islet transplantation: An Update. Diabetes/Metabolism Reviews, 12, 137-150.

Taylor, R., Foster B., Kyne-Gzebalski &Vanderpump, M. (1994) Insulin regimens for the non-insulin dependent: impact on diurnal metabolic state and quality of life. Diabetic Medicine, 11, 551-557.

Taylor, R., Ramm, P., Zimmet, P., Raper, L.R. & Ringrose, H. (1984). Physical activity and prevalence of diabetes in Melanesian and Indian men in Fiji. Diabetologia, 27, 578-582.

Todd, J.A. (1995). Genetic analysis of Type 1 diabetes using whole-genome approaches. Proceedings of the National Academy of Science, USA. 92, 8560-8065.

Todd, C., Bradley, C. & Symonds, L. (1993). Psychosocial measurement in the audit of diabetes services. Audit Trends, 1,141-143.

Toobert, D.J. & Glasgow, R.E. (1994). Assessing diabetes self-management: the Summary of Diabetes Self-Care Activities Questionnaire. 351-375. In C . Bradley (Ed.) Handbook of Psychology and Diabetes: a guide to psychological measurement in diabetes research and practice. Chur, Switzerland: Harwood Academic Publishers.

UK Prospective Diabetes Study Group VII (1990). Response of fasting plasma glucose to diet therapy in newly presenting type II diabetic patients. Metabolism, 39, 905-912.

UK Prospective Diabetes Study Group VIII (1991). Study design, progress and performance. Diabetologia, 34, 877-890.

Vandewalle, C.L., Decraene, T., Schult, F.C., De Leeuw, I.H., Pipeleers, D.G. & Gorus, F.K. (1993) Insulin antibodies and high titre islet-cell antibodies are referentially associated with the HLA DQA1*0301-DQB1*0302 haplotype at clinical onset of Type 1 (insulin-dependent) diabetes mellitus before age 10 years, but not at onset between age 10 and 40 years. Diabetologia, 36, 1155-1162.

Verge, C.F., Gianani, R., Kawasaki, E., Yu, L., Pietropaolo, H., Jackson, R.A., Chase, H.P. & Eisenbarth, G.S. (1996). Prediction of Type 1 diabetes in first-degree relatives using a combination of insulin-, GAD- and ICA512/IA-2 autoantibodies. Diabetes, 45, 926-933.

Veves, A. Webster, L, Chen, T.F. Payne, S. & Boulton, A.J. (1995). Etiopathogenesis and management of impotence in diabetic males: 4 years experience from a combined clinic. Diabetic Medicine, 12, 77-82.

Viberti, G.C., Walker, J.D. & Pinto J.D. (1992). Diabetic nephropathy. In K.G.M.M. Alberti, R.A. DeFronzo, H. Keen & P. Zimmet (Eds.) International textbook of diabetes mellitus. London: John Wiley, 1267-1328.

Wang, P.H., Lau, J. & Chalmers, T.C. (1993). Metaanalysis of effects of intensive blood-glucose control on late complications of Type I diabetes. Lancet, 341, 1306-1309.

Will, J.C. (1995). Self-reported weight loss in adults with diabetes: results from a national health survey. Diabetic Medicine, 12, 974-978.

Wilson, A., Home, P.D., Bishop, A,, Bradley, C., Brown, K.J.E., Hargreaves, B. & other members of the working group of the Research Unit of the Royal College of Physicians and British Diabetic Association (1993). A dataset to allow exchange of information for monitoring continuing diabetes care. Diabetic Medicine, 10, 378-390.

Wing, R.R. & Anglin, K. (1996). Effectiveness of a behavioral weight control program for blacks and whites with NIDDM. Diabetes Care, 19, 409-418.

Wing, R.R., Epstein, L.H., Nowalk, M.P., Koeske, R. & Hagg, S. (1985). Behavior change, weight loss, and physiological improvements in type II diabetic patients. Journal of Consulting and Clinical Psychology, 53, 111-122.

Wing, R.R., Shoemaker, M., Marcus, M.D., McDermott, M. & Gooding, W. (1990). Variables associated with weight loss and improvements in glycemic control in type II behavioral weight control programs. International Journal of Obesity, 14, 495-503.

World Health Organization (1985) WHO Study Group on Diabetes Mellitus. Second Report. (WHO Technical Report Series, No 727). Geneva: World Health Organization.

World Health Organization Regional Office for Europe (1993). Consensus Meeting on Quality Assurance Indicators in Mental Health Care. Stockholm, August 1993.

World Health Organization Regional Office for Europe (1993). Consensus Meeting on Quality Assurance Indicators. Stockholm, November 1995.

Zigmond, A.S. & Snaith, R.P. (1983). The Hospital Anxiety and Depression Scale. Acta Psychiatrica Scandinavica, 67, 361-70.

Zimmet, P.Z. (1992). Kelly West Lecture 1991: Challenges in diabetes epidemiology - from West to the rest. Diabetes Care, 15, 232-252.

Zimmet, P.Z. (1995). The Pathogenesis and Prevention of Diabetes in Adults. Diabetes Care, 18, 1050-1065.