SS 1

Complications and Mortality of Diabetes in Australia

Associate Professor Jonathan Shaw

International Diabetes Institute, Melbourne, Australia

Diabetes represents a growing problem in both developed and developing countries, with most of the healthcare burden arising from diabetic complications. The recent AusDiab study provides a national population-based sample of over 11,000 adults from the general population of Australia from which estimates of the prevalence and impact of diabetes and its complications can be derived.

Diabetic retinopathy, which is the leading cause of visual loss in working aged people in Australia, was found to be present in 21.9% of people with previously diagnosed type 2 diabetes. The major risk factors for retinopathy were poor glycaemic control, hypertension and duration of diabetes, with retinopathy being identified in 57% of those with a diabetes duration of over 20 years. Over 75% of people with type 2 diabetes reported having an eye examination in the previous 2 years.

Peripheral arterial disease and diabetic neuropathy are the reasons that diabetes accounts for approximately 50% of all lower limb amputations. Foot examinations undertaken in the AusDiab study showed that these complications were each present in 13-14% of those with type 2 diabetes, and that 24.1% of those with previously diagnosed diabetes, and 15.3% of those with newly diagnosed diabetes had at least one significant risk factor for future foot ulceration. Only 50% of those with diabetes reported that they had had their feet examined in the previous 12 months, and even amongst those with 'at-risk' feet, only 57% reported a foot examination in the previous year.

Diabetes is now the biggest single cause of end-stage renal disease in Australia. Diabetes was the primary renal disease for 26% of all those going onto dialysis or a renal transplant program in 2003, compared to 16% in 1993. Over this 10 year period, the numbers of people with diabetes registered with end-stage renal disease tripled. Within the AusDiab data, microalbuminuria was present in 25% of those with previously diagnosed diabetes. Even after adjusting for other risk factors, those with impaired fasting glucose and impaired glucose tolerance had a 20-40% increased risk of having microalbuminuria, compared to those with normal glucose tolerance.

Economic data have shown significant increases in costs associated with the presence of diabetic complications. The mean annual cost of healthcare increased from AUS$4025 in those without complications to $7025 in those with microvascular complications, $9055 in those with macrovascular disease, and $9645 in those with both types of complications.

Mortality data published from the Dubbo Study, relating to elderly people in a single Australian town, showed that diabetes was associated with a 60-90% increased mortality over 15 years, and that in this elderly population, diabetes was associated with 18 months shorter survival. Data from five other Australian and New Zealand cohorts showed that over approximately 5 years, diabetes led to an approximate doubling of cardiovascular mortality. In the AusDiab study, mortality data show that compared to those with normal glucose tolerance, total mortality over 4 years was increased by 40%, 70%, 60% and 140% in IGT, IFG, newly diagnosed and previously diagnosed diabetes, respectively.

Overall, these data show the significant health burden and financial costs associated with diabetic complications in Australia. Perhaps most concerning is the increased mortality risk associated with IGT and IFG over only four years.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 2

Making the Diagnosis of Gestational Diabetes: Now and in the Futere

Boyd E Metzger, MD

Division of Endocrinology, Metabolism & Molecular Medicine,
Northwestern University Feinberg School of Medicine, Chicago, IL, USA

There has been longstanding controversy about the diagnostic criteria for GDM, about the cost-effectiveness of strategies for its detection and about therapies that are appropriate and effective. Healthcare providers and policy makers receive conflicting advice. Over the past decade, a number of professional groups and individuals have concluded that, to date, data available from published studies do not provide conclusive evidence of benefit from the detection and treatment of GDM. This has lead some experts to recommend that all routine, systematic testing for GDM be deferred pending resolution of the issues. On the other hand, in recent years, several studies have provided evidence of adverse perinatal consequences associated with mild, untreated maternal hyperglycemia of a lesser degree than meeting traditional criteria for GDM. As a result, some experts feel that criteria presently used for the diagnosis of GDM are too restrictive and that detection efforts should be increased and diagnostic criteria expanded. In addition, data from animal models, epidemiological studies and clinical follow-up observations indicate that intrauterine exposure to the metabolic environment of diabetes or GDM can increase the risk of obesity and alterations of glucose metabolism and type 2 diabetes in the next generation. The importance of resolving such controversy is amplified by the fact that obesity, type 2 diabetes and gestational diabetes mellitus (GDM) are increasing globally at rapid rates and dealing with these epidemic changes has major implications and potentially puts enormous pressure on medical and public health resources.

*The international, multicenter, HYPERGLYCEMIA & ADVERSE PREGNANCY OUTCOME (**HAPO**) study that is currently ongoing has as its primary objective determination of the independent associations between maternal glycemia and outcome in a large, diverse cohort. It is likely that in future years, results of the **HAPO study** will help resolve the controversies about screening and diagnosis. The design of the **HAPO study** will be reviewed in detail. It is also anticipated that some ongoing clinical trials of therapy of GDM will effectively address the issues regarding benefits and costs of treatment. Indeed, a recently reported study that employed randomized assignment to intervention or no intervention found reduction of such adverse events by treatment.*

*What options can be recommended in the interim? As discussed above, one alternative is to be very conservative and defer efforts to screen for, diagnose, and treat GDM pending the availability of results of the **HAPO study** and previously mentioned therapeutic clinical trials that are underway. However, it is much easier to make the recommendation to defer all present efforts for GDM detection as an intellectual statement than it is to implement it in a medically acceptable manner. It is true that in the majority of instances, GDM is "mild" and does silently represent the first appearance of glucose intolerance in a woman. However, type 2 DM is also usually asymptomatic for years after its onset, even at a level of hyperglycemia severe enough to convey risk of birth defects and fetal loss. There is broad consensus that type 2 DM should not go undetected during pregnancy, as might be the case if no surveillance for GDM is systematically performed. The recommendations of the 4th International Workshop Conference on GDM for detection and diagnosis provide a useful approach to putting this problem into perspective. In many venues, the cost and complexity of detection of GDM by the large-scale administration of either a one-step or two-steps screening/diagnostic protocol are critically important considerations. However, strategies that employ blood glucose testing only in the presence of traditional risk factors for GDM have limited sensitivity and specificity.*

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 3

Obstetric Care for Gestational Diabetes – Prevention of Perinatal Morbidity

Moshe Hod, MD

Perinatal Division and WHO Collaborating Center for Perinatal Care, Dept. of Obstetrics & Gynecology, Rabin Medical Center, Beilinson Campus, Petah Tikva, and Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv, Israel

The metabolic changes that take place in the mother during pregnancy have both immediate effects on the fetus in utero and long-lasting effects on the offspring, starting from the early days after birth and extending through infancy, childhood, and even adulthood. This paper presents all the known perinatal risks and complications in offspring of mothers with gestational diabetes mellitus (GDM) and describes the headway made in understanding their underlying pathophysiology and the manner in which hyperglycemia affects these processes. This review demonstrates that in most instances, stringent control of glucose levels for several months before conception can decrease complications during pregnancy and birth, even to rates within the accepted range in the general population. Part of the recent improvement in the outcome of GDM pregnancies derives from updated knowledge and advances in monitoring techniques and treatment in perinatal (maternal fetal medicine) departmenys, neonatal units and pediatric practices. However, most of it is attributable to maternal metabolic balance. Women today have the ability to monitor and balance their blood glucose, and clinicians have the ability to closely follow their control by strict 24-hour measurements of blood glucose levels and monthly measurements of glycosylated hemoglobin (HbA1c) levels. Thus, with good patient-physician cooperation, optimal balance can be achieved and offspring morbidity and mortality minimized. It is important that all physicians be made aware of the dangers of GDM to ensure that all women at risk who are planning pregnancy are referred to the appropriate clinics and informed about the importance of proper glucose control already before pregnancy, and during the course of pregnancy and delivery.

The best evidence-based indication for CSII is frequent, unpredictable hypoglycemia, in spite of best attempts with multiple daily insulin injections (MDI), and this is now incorporated into several national guidelines.

Earlier studies published by our department have described in depth the clinical and investigational aspects of congenital abnormalities in offspring of diabetic mothers [(1)] and the long-term consequences of GDM into childhood and adolescence [(2)].

The present review focuses mainly on the perinatal period: the morbidity and mortality that occur around the time of delivery and are associated with pregnancy and birth, and early neonatal morbidity.

Perinatal Mortality

The advances made in the knowledge and practice of good metabolic control in recent years have been accompanied by a reduction in the perinatal mortality rate of infants of diabetic mothers (IDM). However, it still remains high relative to the nondiabetic population. A Swedish study conducted from 1982 to 1985 [(3)] reported a mortality rate of 1.3% for this patient group, or four times the national average. Nevertheless, this is a considerable improvement over the 19% reported for 1958-1971. These studies show that the better the balance of glucose level, the lower the perinatal mortality rate [(4)].

Some researchers differentiate the mortality of IDM that is associated with congenital malformations - still the leading cause - and mortality due to perinatal complications, such as asphyxia. The risk of asphyxia among IDM is 27%, and even higher when the mother has nephropathy or hyperglycemia before delivery [(4)]. Congenital abnormalities account for 50% of all deaths of IDM, compared to 20 to 30% in infants of nondiabetic mothers. Sudden intrauterine death towards the end of pregnancy used to be very prevalent, and continues to occur even today. Although perinatal mortality is decreasing significantly, the ratio of fetal-to-neonatal

deaths remains constant at 2:1. The cause of death is still unclear. Tyrala [5] found that hyperglycemia and hyperinsulinemia lead to hypoxemia and acidosis in the fetus, decreasing its ability cope with hypoxic states.

Complications of Pregnancy and Birth

Macrosomia

Along with the decrease in neonatal mortality and congenital malformations, the greater awareness of the need for strict metabolic control in GDM has led to a significant drop in the rate of macrosomia (birth weight >4000 g). Studies released in the 1980s reported a 20% to 50% incidence of macrosomia in IDM [6]. The macrosomia is selective and affects mainly subcutaneous adipose levels. Clinically, macrosomic infants appear large, flabby and plethoric. Fat pads may be noted in the upper back and lower jaw, conferring a cushingoid appearance. As the heart, liver, kidneys and brain are undisturbed by the hyperglycemia, the ratio of the head circumference to the abdominal circumference is high. Ballard [8] measured the ponderal index (weight/length^3) in 179 IDM and 510 infants of nondiabetic mothers matched for age, sex, country of origin, and year of birth. Macrosomia was found in 45% of the study group compared to only 8% of the controls, and it was disproportionate in 19% and 1%, respectively. In 1954, Pedersen [7] suggested that fetal macrosomia is due to hyperinsulinemia consequent to an overabundance of maternal glucose (Fig. 2).

Fetal macrosomia is associated with complications at birth, including shoulder dystocia, brachial plexus injury and asphyxia. Shoulder dystocia is found 2-5 times more often in IDM, and is even more prevalent in the presence of high birth weight. Other problems include cephalohematoma, facial nerve paralysis, and clavicular fractures. Infants after traumatic delivery are at higher risk of distress, low Apgar score, and asphyxia, and may find it more difficult to adapt to the extrauterine environment. To avoid these complications, macrosomia should be prevented to the extent possible. Clinicians must be able to identify it and to apply the most appropriate mode of delivery in each case.

Prevention of macrosomia. *Already in the early 1990s many studies showed that the higher the glucose level, the higher the rate of macrosomia. However, when tight glucose control was maintained during pregnancy, macrosomia decreased, even to rates equal to those in the nondiabetic population. Langer [8] and others [9] showed that the optimal ratio of small-for-gestational-age (SGA) infants (below 10th percentile for age) to large-for-gestational age (LGA) infants (above 90th percentile for age) is achieved when average blood glucose levels range between 86 and105 mg/dl (4.8-5.8 ml/l), i.e., the norm for nondiabetic women. If levels decrease to below 86 mg/dl (4.8 ml/l), the proportion of SGA infants rises; if they increase to above 105 mg/dl (5.8 ml/l), the proportion of LGA infants rises [11]. Therefore, to obtain a rate of macrosomia equal to that in the general population (5-10%), glucose control needs to be tight, but not inordinately, so that levels remain stable and not veer from the norm. Hod et al. [12] demonstrated that by adopting a protocol of optimal balance during the course of pregnancy and inducing labor around the 38th week of gestation for fetuses identified by ultrasound as LGA, clinicians can achieve a 5% macrosomia rate without a significant increase in the rate of cesarean section.*

Mode of delivery *The rate of cesarean section for macrosomia may be as high as 47%. For prenatal diagnosis, sonographic measures of abdominal circumference (over 90th percentile for gestational age) and shoulder soft tissue width (over 12 mm) are superior to measures of biparietal diameter and femur length [13], but they are still not sufficiently correlated for good reliability[14]. Rates of both macrosomia and cesarean section have been found to be lower in diabetic women treated by diet and insulin than in diabetic women treated by diet alone [4,5].*

Intrauterine Growth Restriction

Approximately 20% of IDM have intrauterine growth restriction (IUGR) compared to 3-7% of the general population. Several factors are responsible for this difference.

1. An abrupt balance in glucose level; like amino acids and glucose, can interfere with fetal growth.

2. Preeclampsia can disturb placental blood flow and transfer of metabolites; the risk rises with an increase in HbA1c [15].

3. Diabetes-induced vascular disease may decrease placental perfusion.

4. The high rate of congenital malformations may slow growth rate.

Using sonographic measurements of crown-rump length, Pedersen et al. [16,17] showed that growth deceleration begins already in the first weeks of pregnancy. These fetuses have a greater likelihood of congenital abnormalities.

Early Perinatal Morbidity

Offspring of diabetic mothers are susceptible to many early perinatal complications. Early complications are more prevalent in pregestational diabetes mellitus than in GDM, and some, particularly polycythemia and hyperbilirubinemia, are more prevalent in macrosomic than nonmacrosomic IDM [18-20]. The frequency with which these complications occur depends on the severity of the diabetes [6].

Hypoglycemia

The less well balanced the maternal glucose level, the more likely the presence of hypoglycemia in the infant after birth. When the mother is hyperglycemic in the last trimester, and especially at delivery, placental metabolism increases, and the fetus too becomes hyperglycemic. The hyperglycemia induces increased insulin secretion from the fetal pancreas. After birth, the high insulin supply from the mother stops (fetal blood levels are 70-80% those of the mother), but the hyperinsulinemia remains, leading to a rise in the insulin-to-glucose ratio [5]. As a consequence, glycogenolysis and lipolysis are delayed, glucogenic enzymes are not produced, and the liver produces low glucose levels. In addition, the insulin heightens glucose uptake by the surrounding tissues, such as striated muscle, so that the available glucose is utilized immediately. The combination of high tissue uptake and low liver production result in hypoglycemia, which could last as long as 24-72 hours, until insulin levels adjust to the metabolic state.

Blood glucose levels above 90 mg/dl (5.02 ml/l) in the mother during delivery are correlated with an increased occurrence of hypoglycemia in the infant [6]. When insulin balance is strict, the rate of maternal hypoglycemia rises threefold [21]. Neonatal hypoglycemia is defined as a glucose level below 40 mg/dl (2.6 ml/l) in the first 2-3 days of life. The incidence of hypoglycemia in the general population is 0.5-4% in term newborns and 67% in preterm newborns. The incidence in IDM is 20-40%, with those with disproportionate macrosomia being at the highest risk [8].

IDM who are asymptomatic at birth undergo routine glucose measurement at 1-2 hours of life. If levels are below 40 mg/dl, feeding is started. In symptomatic infants (poor tonus, pallor, restlessness, events of apnea or respiratory distress), IV dextrose is injected immediately. Feeding is also begun early to prevent hyperglycemia, which could delay the activity of the glucogenic enzymes. The chronic intrauterine hyperinsulinemia depresses the ability of the fetal liver to release glycogen (decreased hepatic phosphorolysis), placing the infant at risk of hypoglycemia not only in the first hours after birth but also for the next few days. Kinnala et al. [22] compared oxygen consumption and carbon dioxide release during treatment for hypoglycemia between IDM and SGA infants and found differences in the type of metabolic disturbances. Some authors have suggested a below-normal level of activity of the sympatho-adrenal axis in IDM. This assumption was supported by the finding of decreased urinary catecholamine levels in IDM with severe hypoglycemia. Furthermore, the injection of IV epinephrine in IDM caused a rise in plasma glucose levels and free fatty acids and a drop in insulin. In women who maintain tight metabolic balance during pregnancy and delivery, this complication is avoided, and their children show no difference in metabolic behavior from non-IDM controls [5].

Hypocalcemia and Hypomagnesemia

Tsang et al. [23-25] demonstrated already in the 1970s that IDM, like preterm or asphyxiated infants, are at risk of hypocalcemia (<7 mg/dl in preterm infants and >8 mg/dl in term infants). The incidence of hypocalcemia in IDM is high, reaching up to 50% in the first 3 days of life. This is true even when preterm birth and presence of asphyxia – which occur at higher rates in IDM than in healthy infants – are accounted for. The degree of hypocalcemia depends on the severity of the maternal diabetes and is related to the diabetes-induced loss of urinary magnesium in the mother, which in turn causes magnesium loss in the fetus. Lack of

magnesium can adversely affect pituitary hormone secretion in the infant and cause hypocalcemia. Of 156 infants examined in the study of Tsang et al. [23-25], 21 had magnesemia in the first 3 days of life.

Polycythemia and Hyperbilirubinemia

Polycythemia (venous hematocrit over 70% at 2 hours of life and over 65% at 6 hours) occurs in about 3% of infants born above sea level, and about 5% of infants born in higher altitudes. In IDM, the incidence ranges from 12% to 40% in different studies [4-6]. The literature suggests two explanations for polycythemia, which is induced by hypoxemia: one involves high erythropoiesis, and the other, a change in placental-fetal blood distribution.

1. A study in a sheep model demonstrated that hyperglycemia, hyperketonemia, and hyperinsulinemia are associated with increased oxygen need and decreased arterial oxygen capacity [5]. Hypoxemia causes an increase in the production of erythropoietin and, thereby, secondary erythropoiesis. IDM have increased concentrations of plasma erythropoietin at birth, which is correlated with glucose and insulin levels in the amniotic fluid and cord blood [26].

2. Intrauterine placental-fetal blood volume is about 110 mg/kg, with the placental compartment accounting for 35% and the fetal compartment, 65%. When pregnant sheep were allowed continuous inspiration of 10% oxygen, the hypoxemia in the fetus caused a shift in the blood distribution between the compartments, so that only 25% of the blood volume remained in the placenta with the remainder going to the fetus [5]. This mechanism has not yet been studied in depth, but it is apparently associated with changes in blood vessel resistance.

Polycythemia should be carefully managed in order to prevent hyperviscosity syndrome in the infant. Hyperviscosity syndrome may be accompanied by cyanotic spells, worsening of respiratory distress or hypoglycemia, oliguria, necrotizing enterocolitis, or renal vein thrombosis.

Hyperbilirubinemia seems to be a result of several factors, not all of which are known. They include hemolysis, increased red blood cell mass, inefficient erythropoiesis, prematurity, trauma, and delayed hepatic enzyme production [6]. IDM are at higher risk of hyperbilirubinemia [8], and about 50% of affected infants require phototherapy. The need for exchange transfusion is rare.

Hyaline Membrane Disease

The risk of hyaline membrane disease at any given gestational age before week 38 is 5 to 6 times higher in IDM than in infants of nondiabetic mothers [5]. Glucose balance has an effect on the incidence of the disease. In the past, hyaline membrane disease was a major risk factor for morbidity and mortality because labor was induced early in diabetic women. Even today, though, the disease remains widespread and researchers are seeking ways to prevent it. Clinicians tend to find hyaline membrane disease more often (about 20% frequency) after cesarean section than after vaginal delivery (10%) [4]

Several studies have attempted to explain the mechanism of hyaline membrane disease. Hawden and Aynsely-Green [4], in an investigation of type II pneumocytes in rats and rabbits, showed that insulin inhibits the cortisol-dependent production of phosphatidylcholine (lecithin), apparently as a consequence of the inhibited production of one of the prerequisites of phosphatidylcholine, fibroblast-pneumocyte factor. In rats, high glucose levels block the transformation of choline to phosphatidylcholine, and butyrate blocks the translation of mRNA into surfactant proteins [4]. Insulin may also have an opposite, dose-dependent stimulatory effect on the transformation of choline to phosphatidylcholine.

In another study in pregnant rats, streptozotocin-induced diabetes led to hyperglycemia without hyperinsulinemia [5]. The fetuses exhibited inhibited surfactant production, inhibited glycogen metabolism, and morphologically immature lungs.

Diabetic pregnancy in humans is associated with delayed attainment of the phosphatidylcholine/sphingomyelin ratio in amniotic fluid. Even a ratio above 2.0 does not guarantee lung maturation. This finding has as yet no explanation.

Before birth, fetal lung maturity is determined by the presence of phosphatidylglycerol. In all cases of early induction, the mother should be given steroids. There is no proof that steroids interfere with the metabolic balance over the long term [5].

When the diabetes adversely affects the vascular system, there is a higher occurrence of fetal lung prematurity [5]. The introduction of the ultrasound nonstress test has improved the ability of clinicians to identify fetuses at risk and thereby prevent their early birth.

Hypertrophic Cardiomyopathy

Cardiomyopathy secondary to the anabolic effect of insulin is more prevalent in macrosomic newborns (8.3% vs. 1.8% in neonates of appropriate size) [27,28]. High insulin levels act on both insulin and IGFII receptors, which are found in high density mainly in the ventricular septum [5]. Therefore, most IDM have hypertrophy of the ventricular septum, which in turn leads to subaortic stenosis [4,6]. Five percent of affected infants will acquire cardiac insufficiency consequent to left ventricular obstruction. In one study, investigators compared the cardiac state between infants born after diabetic pregnancy whose mothers began strict follow-up early (first trimester) or later (second, third trimester) [5]. They found that both groups had more cardiac hypertrophy than controls. However, none of the early-follow-up group required oxygen after birth whereas 19% of the late-follow-up group did so. Usually, the cardiomyopathy resolves after 6 months without sequelae. In very severe cases, there may be cardiac insufficiency and signs of respiratory distress, such as tachypnea, increase in oxygen consumption, or defective feeding.

Very little is known about the long-term effects of hyperinsulinemia on cardiosclerotic diseases in adults. Long-term hyperinsulinemia causes pathologic arterial smooth muscle changes which have been related to arteriosclerosis. Moreover, hyperinsulinemia, regardless of the presence of arteriosclerosis, may cause hypertension [29,30].

Sonographic Assessment in Diabetic Pregnancy

Antenatal ultrasound plays an important role in monitoring diabetic pregnancies. The main issues associated with sonographic assessment of these pregnancies include the following:

1. *Assessment of gestational age*
2. *Detection of congenital anomalies*
3. *Surveillance of growth*
4. *Dynamic assessment of fetal status (BPS, Doppler)*

The ultrasound evaluation should take into consideration the differences between GDM (Gestational Diabetes Mellitus) and PreDM(Pre-existing Diabetes Mellitus), and therefore, the sonographic approach must be tailored accordingly.

1. Gestational age determination

Evaluation of gestational age is extremely important for accurate monitoring of the advancing pregnancy. Estimation of gestational age should be performed in the first trimester of pregnancy, preferably, using TVS (trans-vaginal sonography). CRL is the best parameter for this purpose.

2. Congenital anomalies

With current care, perinatal mortality of the IDDM has been drastically reduced. The main contributor to perinatal mortality and morbidity in these patients is congenital malformations of the fetus.

Abnormalities commonly affecting IDDM's include CNS, Heart, Skeletal, Genitourinary and GIT malformations. The lesion most associated with diabetic embryopathy, the caudal regression syndrome, is actually less common, with an incidence of 1.3 per 1000 diabetic pregnancies. Detection of congenital anomalies should be started in the first trimester of pregnancy and repeated in the second trimester. If possible, early anomaly scan using TVS (transvaginal sonography) may be helpful (14-16 weeks). A basic examination is mandatory in the second trimester of pregnancy and the following organs should be observed: cranium and brain, spine, stomach, bladder, kidneys and insertion of the umbilical cord. The four chamber view of the heart must be obtained, however, a detailed fetal echocardiography performed by a skilled pediatric cardiologist is preferred.

3. Fetal growth monitoring

Monitoring fetal growth continues to be a challenging and highly inexact process. Although today's tools, which involve serial plotting of fetal growth parameters, are superior to earlier clinical estimations, accuracy is still +/- 15%, using the most sophisticated ultrasound equipment. The most important task is to detect fetal macrosomia and IUGR. Since fetal macrosomia is the most frequent fetal complication of pregnant diabetic patients, a particular effort should be directed toward its diagnosis and management. Thus, unless the patient is not obese and periodic fundal measurements are normal, all pregnant diabetic patients should undergo ultrasound growth assessments of the fetus every several weeks, starting at around 20 weeks of pregnancy for preGDM's and time of diagnosis for GDM's. The macrosomic fetus, at some time will be above the 95th percentile for one or more parameters, most frequently, the abdominal circumference. The positive predictive value for the diagnosis of macrosomia exceeds 90% when the abdominal circumference or the estimated fetal weight is above the 95th percentile. In IDDM's, macrosomia is more apparent in some fetal structures: liver, subcutaneous fat, soft tissues of arm, thigh and cheeks. These variables (selective organomegaly) are measurable and may aid in predicting early development of macrosomia. IUGR is associated with conditions that predispose to uteroplacental insufficiency, and therefore is most likely to appear in DM complicated by severe vasculopathy. In most centers decision making process regarding time and mode of delivery takes place at around 37-38 weeks of gestation, therefore EFW should be performed at that time.

4. Assessment of fetal well-being

Dynamic assessment diabetic pregnancies implies two types of investigations: BPS (Biophysical Score) and Doppler studies. The fetal BPS is often applied to evaluate the significance of a nonreactive NST. It may serve as an important tool for fetal surveillance, especially in order to prevent unnecessary early interventions, thereby allowing prolongation of pregnancy beyond 37 weeks. In diabetic gravidas, uteroplacental insufficiency may be difficult to detect by ultrasound assessment of fetal growth, since fetal weight gain can be excessive due to fuel metabolism even when uteroplacental circulation is compromised. Doppler umbilical artery velocimetry has been proposed as a clinical tool for Antepartum fetal surveillance in pregnancies at risk for placental vascular disease. The data is conflicting, and several large studies have now confirmed that ranges for umbilical artery waveforms indices are not different in a diabetic population without pregnancy complications than in the normal controls.

Proposed ultrasound work-up in DM complicating pregnancy

A. GDM patients

Ultrasound evaluation should start immediately following diagnosis.

2nd and 3rd trimesters:

a. *Fetal growth and weight estimations starting at diagnosis and continuing at 3-4 weeks intervals.*
b. *Fetal weight estimation at 37-38 weeks.*
c. *BPS at weekly intervals starting at 34 weeks only for insulin treated and/ or patients with poor compliance and control.*

B. PreDM patients

1st trimester:

a. *8-10 weeks – TVS dating of pregnancy (CRL).*
b. *12 weeks – Nuchal translucency (optional).*

2nd trimester:

a. *15 weeks – Transvaginal first detailed anatomical survey of the fetus (optional).*
b. *22 weeks – Second detailed anatomical survey of the fetus (abdominal).*
c. *20-24 weeks – Fetal echocardiography*

3rd trimester:

a. *Fetal growth and weight estimations starting at 20 weeks, at 3-4 weeks Intervals.*
b. *Fetal weight estimation at 37-38 week*
c. *BPS at weekly intervals starting at 32-34 week*

In all examinations a thorough assessment of all fetal growth parameters is mandatory (BPD, OFD, HC, AC, FL).

a) Level II evaluation of fetal congenital anomalies is performed at 14–15 weeks and repeated at 20–22 weeks of gestation.

b) Fetal heart echocardiography at 20–22 weeks of gestation.

c) Assessment of fetal growth combined with the evaluation of fetal biophysical profile, every 2–4 weeks until 38 weeks.

- Umbilical blood flow evaluation.

- Weekly visits at the clinic allow close follow-up and early detection of any signs of fetal stress or distress.

Conclusion

It is well recognized today that strict diabetic control before conception, during pregnancy, and during delivery decreases the rate of perinatal mortality, the rate of cesarean section and complicated birth, and the early and late morbidity in the offspring. Nevertheless, not all diabetic women are being referred to specialized clinics before pregnancy, and many of those who are continue to find it difficult to maintain optimal glucose balance while continuing their regular lifestyle. The genetics of diabetes and the precise mechanisms underlying the many changes observed in clinical trials remain for the most part unknown. More data are also still needed on the effects of diabetes on the adult and on diseases of adulthood, such as hypertension and coronary heart disease.

References

1. *Mor A, Bar J, Hod M. Congenital abnormalities in offspring of diabetic mothers – clinical and research. Harefuah 1998; 135: 545-551.*
2. *Ferber A, Ben-Rafael Z, Hod M. Intrauterine metabolism and fetal growth – long-term consequences. Harefuah 1999; 136: 554-559.*
3. *Hanson U, Persson B. Outcome of pregnancies complicated by type I insulin dependent diabetes in Sweden: acute pregnancy complications, neonatal mortality and morbidity. Am J Perinatol 1993; 10: 330-333.*
4. *Hawdon JM, Aynsley-Green A. Prenatal complications, including hypoglycemia. Diabetes and Pregnancy: An International Approach to Diagnosis and Management, edited by Dornhorst A, Hadden DR, 1996, John Wiley & Sons, 303-318.*
5. *Tyrala E. The infant of the diabetic mother. Obstet Gynecol Clin North Am 1996; 23: 221-241.*
6. *Weintrob N, Karp M, Hod M. Short and long range complications in offspring of diabetic mothers. J Diabetes Complications 1995; 9: 1-7.*
7. *Pedersen J. Weight and length at birth of infants of diabetic mothers. Acta Endocrinol 1954; 16: 330-342.*
8. *Ballard JL, Rosenn B, Khoury JC, et al. Diabetic fetal macrosomia: significance of disproportionate growth. J Pediatr 1993; 122: 115-119.*
9. *Langer O. Prevention of macrosomia. Ballieres Clin Obstet Gynaecol 1991; 5: 333-347.*
10. *Landon MB, Gabbe SG, Piana R, et al. Neonatal morbidity in pregnancy complicated with diabetes mellitus: predictive value of maternal glycemic profiles. Am J Obstet Gynecol 1987; 156: 1089-1095.*
11. *Langer O, Levy J, Brustman L, et al. Glycemic control in gestational diabetes mellitus: how tight is tight enough: SGA vs. LGA? Am J Obstet Gynecol 1989; 161: 646-653.*
12. *Hod M, Bar J. Timing and mode of delivery in gestational diabetes. Diabetes Care 1998; 21: 113-117.*
13. *Neiger R. Fetal macrosomia in the diabetic patient. Clin Obstet Gynecol 1991; 35: 138-150.*
14. *Mintz MC, Landon MB, Gabbe SG, et al. Shoulder soft tissue width as a predictor of macrosomia in diabetic pregnancies. Am J Perinatol 1989; 6: 240-243.*
15. *Hanson U, Persson B. Epidemiology of pregnancy-induced hypertension and pre-eclampsia in type I insulin dependent diabetic pregnancies in Sweden. Acta Obstet Gynecol Scand 1998; 77: 620-624.*
16. *Pedersen JF, Molsted-Pedersen L. Early growth delay detected by ultrasound marks increased risk for congenital malformations in diabetic pregnancy. Br Med J 1981; 283: 269-271.*

17. *Pedersen JF, Molsted-Pedersen L, Mortensen HB. Fetal growth delay and maternal hemoglobin A1c in early diabetic pregnancy. Obstet Gynecol 1984; 64: 351-352.*
18. *Mountain KR. The infant of the diabetic mother. Baillieres Clin Obstet Gynaecol 1991; 5: 413-442.*
19. *Berk MA, Mimouni F, Miodovnik M, et al. Macrosomia in infants of insulin-dependent diabetic mothers. Pediatrics 1989; 83: 1029-1034.*
20. *Ogata SE. The infant of the diabetic mother: pregnancy as a "tissue culture experience". Isr J Med Sci 1991; 27: 524-531.*
21. *The Diabetes Control and Complications Trial Research Group. The effect of intensive treatment of diabetes on the development and progression of long term complications in IDDM. N Engl J Med 1993; 329: 977-986.*
22. *Kinnala A, Manner T, Nuutila P, et al. Differences in respiratory metabolism during treatment of hypoglycemia in infants of diabetic mothers and small-for-gestational-age infants. Am J Perinatol 1998; 15: 363-367.*
23. *Tsang RC, Chen I, Atkinson W, et al. Neonatal hypocalcemia in birth asphyxia. J Pediatr 1974; 84: 428-433.*
24. *Tsang RC, Kleinman L, Sutherland JM. Hypocalcemia in infants of diabetic mothers: studies in Ca, P and Mg metabolism and in parathyroid hormone responsiveness. J Pediatr 1972; 80: 384-**??***
25. *Tsang RC, Light JL, Sutherland JM. Possible pathogenetic factors in neonatal hypocalcemia of prematurity. J Pediatr 1973; 82: 423-429.*
26. *Widness JA, Teramo KA, Clemons GK, et al. Direct relationship of antepartum glucose control and fetal erythropoietin in human type I (insulin-dependent) diabetic pregnancy. Diabetologia 1990; 33: 378-383.*
27. *Mountain KR. The infant of the diabetic mother. Clin Obstet Gynecol 1991; 5: 413-442.*
28. *Cooper MJ, Enderlein MA, Tarsoff H, et al. Asymmetric septal hypertrophy in infants of diabetic mothers: fetal echocardiography and the impact of maternal diabetic control. Am J Dis Child 1992; 146: 226-229.*
29. *Stuot RW. Insulin and atheroma: 20 years perspective. Diabetes Care 1990; 13: 631-654.*
30. *Reaven GM, Hoffman BB. Hypertension as a disease of carbohydrate and lipoprotein metabolism. Am J Med 1989; 87: 2s-6s.*
31. *Hod M, Diamant YZ. The offspring of a diabetic mother – short- and long-range implications. Isr J Med Sci 1992; 28: 81-86.*
32. *Silverman BL, Rizzo T, Cho NH, et al. Long-term effect of the intrauterine environment. The Northwestern University Diabetes in Pregnancy Center. Diabetes Care 1998; 21: 142-149.*
33. *Silverman BL, Rizzo T, Green OC, et al. Long-term prospective evaluation of offspring of diabetic mothers. Diabetes 1991; 40: 121-125.*
34. *Metzger BE, Silverman BL, Freinkel N, et al. Amniotic fluid insulin concentration as a predictor of obesity. Arch Dis Child 1990; 65: 1010-1052.*
35. *Silverman BL, Metzger BE, Cho NH, et al. Impaired glucose tolerance in adolescent offspring of diabetic mothers. Relationship to fetal hyperinsulinism. Diabetes Care 1995; 18: 611-617.*
36. *Warram JH, Krotewski AS, Gottliev MS, et al. Differences in risk of insulin-dependent diabetes in offspring of diabetic mothers and diabetic fathers. N Engl J Med 1984; 311: 149-152.*
37. *Warram JH, Martin BC, Krolewski AS. Risk of IDDM in children of diabetic mothers decreases with increasing maternal age at pregnancy. Diabetes 1991; 40: 1679-1684.*
38. *Pettitt DJ, Aleck KA, Baird RH, et al. Congenital susceptibility to NIDDM: Role of intrauterine environment. Diabetes 1988; 37: 622-628.*
39. *Silverman BL, Cho NH, Metzger BE. Impaired glucose tolerance (IGT) in adolescent offspring of diabetic mothers. Diabetes 1993; 42: 87A.*
40. *Rizzo T, Metzger BE, Burns WH, et al. Correlations between antepartum metabolism and intelligence of offspring. N Engl J Med 1991; 325: 911-916.*
41. *Petersen MN, Pedersen SA, Greisen G, et al. Early growth delay in diabetic pregnancy: relation to psychomotor development at age 4. Br Med J 1988; 296: 598-600.*
42. *Vladorova D. Children of diabetic mothers from the aspects of the neurologist. Cesk Neurol Neurochir*

1982; 45: 94-98.
43. *Hod M, Levy-Shiff R, Lerman M, et al. Developmental outcome of offspring of pregestational diabetic mothers. J Pediatr Endo Metab 1999; 11, 867-872.*

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 4

Insulin and Oral Hypoglycemic Agents in Gestational Diabetes

Mary Anne Lim-Abrahan, MD, FPCP, FPSEM

Professor, Endocrinology, Diabetes & Metabolism, Dept of Medicine, UP College of Medicine

Gestational diabetes develops because pregnancy increases requirements for insulin secretion while increasing insulin resistance. In gestational diabetes, there is impaired pancreatic â,-cell compensation for insulin resistance. Gestational diabetes increases the risk of adverse perinatal outcomes and increased risk of later childhood obesity and T2DM.

The traditional treatment approach to gestational diabetes includes diet, exercise, careful blood glucose monitoring & treatment of hyperglycemia with insulin achieving targets.

Novel approaches in the treatment & prevention of gestational diabetes with glyburide (known in Asia as glibenclamide) and metformin are emerging.

Glyburide is a second-generation sulfonylurea that may not cross the placental barrier. This has been attributed to its extensive plasma protein binding and short elimination half-life. In a randomized head-to-head comparison of glyburide vs insulin showed no difference for mean glucose levels, % of LGA infants, macrosomia, neonatal lung complications, hypoglycemia, and admission to a neonatal ICU or fetal anomalies.

Metformin, a pregnancy category "B" class drug, appears to be safe, effective and non-teratogenic during pregnancy. Metformin use leads to a 10-fold reduction in gestational diabetes in women with PCOS. It also has been shown to decrease early pregnancy loss in women with PCOS by 10-fold. There are no birth defects or neonatal hypoglycemia, nor were there reports of lactic acidosis in the mother. Metformin does not adversely affect infants' birth weight or length or growth, motor and social development in the first year of life.

Thiazolidinediones, a PPAR agonist, a new class of insulin sensitizers increase insulin sensitivity by increasing muscle glucose uptake and decreasing hepatic gluconeogenesis. Currently available TZDs rosiglitazone and pioglitazone have both been classified as pregnancy category "C" drug and should not be used in the treatment of gestational diabetes or T2DM during pregnancy.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 5

Using Diabetes Education Research in Clinical Prac

Professor Trisha Dunning, AM

Department of Endocrinology and Diabetes St. Vincent's Health Melbourne and The University of Melbourne

Diabetes education and clinical practice are influenced by research from a range of methods: quantitative and qualitative studies as well as quality management and evaluation research. Diabetes educators need to understand the various research methods in order to be able to assess the implications of the findings from each paradigm, and use them appropriately and effectively in clinical care.

The aim of diabetes education is to assist the person with diabetes to actively participate in their care by developing their knowledge and problem-solving skills so they can adequately self-care and integrate diabetes into their lives. In achieving this aim diabetes educators practice in a climate of evidence-based care, therefore they must accept they have a responsibility to be research active. Evidence-based care refers to the conscious, explicit and judicious use of current best evidence to make decisions about the care of individuals. It encompasses five main stages: recognising a clinical issues, being able to formulate a relevant answerable question, searching for the evidence to answer the question, evaluating the evidence, implementing relevant findings, and monitoring the outcomes. Significantly, the diabetes educator is often the interface between the evidence and clinical practice (the point at which evidence enters practice) in that they interpret the research for colleagues and people with diabetes and help them incorporate relevant findings in their individual practices or lives. Thus research is a significant core component of the diabetes educator role.

There is no doubt there are barriers to undertaking research in clinical practice. In order to participate in and use research diabetes educators require research knowledge, critical appraisal skills, and a supportive leadership, management and professional organisational infrastructure that encourages and supports research. Currently, research is not a major component of most diabetes educator education curricula. Research training through research courses, higher degrees and mentoring could help diabetes educators utilise research findings more critically and effectively as well as encouraging them to collaborate in or undertake research to enhance the evidence base for and the value of diabetes education to clinical care.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 6

The Culture and Context Based Education: A Key Factor in the Success of Diabetic Education at the Community Level

Dr. Supattra Srivanichakorn

Director, Institute of Community based Health Care Research and Development, Thailand

Health services for diabetic patients and risk group in Thailand mostly are hospital based. They are crowded and not patients friendly. The chance of patients to get information or relevant knowledge is very limited. " Glycemic level " is the target of health professionals to tackle at every visit rather than the patients' health and self-management ability of patients and family. Around 50 % of the diabetic patients are uncontrolled and their behaviors are unfavorable. They don't really know the specific and clear process to save them well. Mostly know the broad information.

Decentralized the health services to primary care has been developed, the patients are cared by primary care providers (nurses, paramedics under doctors' supervision) nearby their residences. The services are more friendly and have more opportunities to discuss about their health. The diabetic education and self-help group is implementing in the health service setting or at home or in the community. But again the information and knowledge given are still broad. They start to know the gaps' knowledge that are not specific and not relevant to life styles. The patients and general people still have some mis-understand and not adequate understanding to let them early detection and prevent the complications.

The given information is not culture sensitive and not relevant to life styles and local contexts. The education is provided separately according to the diseases, not integrated as risk factors/ behaviors. People cannot link between the diabetic risk and other cardiovascular risks.

After the primary care providers got trained on detail and specific knowledge on updated diabetic physiology, nutrition and exercise including people centered approach, they have realized the gap and start to learn the detail belief and behaviors of people to understand people. These introduce the starting points of more relevant education, mutual understanding among patients and providers and learn from each others.

We have learned that the new education is not focus only on methodologies, but the relevant/ accurate content is the key.

"Creativity is nice, but an accurate understanding of your target audience is even better.
Your million dollar asset is your knowledge of your target audience."

Identify relevant alternatives is also an important way for start changing behaviors. People participation is one strategy to make diabetic education easy, simple and relevant to people. Role models is another success method. Linkage with community leaders and village health volunteers is another ways to start for community prevention programs.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 7

Patient Education: Are We Wasting Our Time?

Kan Ching Yee, Eva, Alice Ho Miu Ling Nethersole Hospital, HKSAR

Diabetes patient education was recognized as an important element of diabetic management since the time insulin therapy was discovered. The important role of patient education to enhance self-care by patients is irrefutable. Every body of the diabetic team is an educator to different extent, and every interaction with the patient can be regarded as an educational act.

As educators, however, we do question ourselves on the value of education from time to time. The author will bring the audience to a reflective journey to reconsider our practice of patient education. Firstly, issues on roles, abilities and limitations of diabetes educator in the context of helping patients to live with the diabetic illness will be discussed. Secondly, real life struggles of diabetes educators on value of educating will be presented. Case scenarios will be used to illustrate both patient and educator factors which commonly intimidate the perception of value in patient education. Thirdly, the author will assert on the position that the only situation that can invalidate patient education itself is when all patients can perfectly self-manage their diabetes. Being "ineffective" is not equivalent to being "valueless". Ineffective education can cause much frustration but it does not imply patient education being "valueless". It only challenges us to pursue new direction in patient education to improve our effectiveness. The science and art of health education and health behavior are eclectic, rapidly evolving, and reflective of an amalgamation of approaches, methods, and strategies from social and health sciences. To maximize effectiveness, diabetes educators need to synthesize large and diverse literatures and apply them to practice. Finally, the importance of appropriate goal setting and right means to achieve goals in patient education will be illustrated by day to day examples contributed by experienced diabetes educators.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 8

Some Insights from the IDF WPR Childhood and Adolescence Diabcare 2003 Study

Warren Lee on behalf of the Steering Board

The IDF WPR Childhood and Adolescence Diabcare (ChildDiab) project was an audit and benchmarking project on the management of diabetes management in children and adolescents in the IDF Western Pacific Region. It was set up as a collaborative effort between Novo Nordisk (International Operations Clinical Development Centre), Bio-Rad Laboratories and 56 paediatric centers in Australia, China, Hong Kong, Taiwan, Indonesia, Japan, Malaysia, Philippines, Singapore, South Korea and Thailand, appointed to participate in the project by the respective national diabetes associations. The 2003 study specifically focussed on the problem of Type 2 diabetes mellitus in childhood and adolescence. Data Collection started 14 Jan 2003 and was completed on 9 Feb 2004. Data was recorded on paper forms and included anthropometric indices, frequency of clinic visits and centralised HbA1c measurements. The survey forms also covered details of the date of onset or discovery, family medical history of parents and siblings with type 2 diabetes, presence of diabetes complications and details of current diabetes management. Each centre contributed all the data that they had available for the patients. The study population included all patients with a duration of diabetes of a minimum of 12 months and whose age was below 18 years on the date of the visit. Centralised HbA1c samples were tested in a centralized laboratory using a automatic high-pressure liquid chromatography method (Bio-Rad VARIANT, Bio-Rad Laboratories, Hercules, CA). All data handling and statistical analysis were performed by Novo Nordisk. A total of 346 data collectionforms (DCFs) were obtained from 56 centres in the 11 countries/territories, out of which 15 (4.3%) were excluded. The mean age of patients in the study was 14.7 +/- 2.2 yrs, the mean duration of diabetes 2.8 +/-1.9 years, and age of onset was 11.9 +/- 2.3 years, respectively, with a slight preponderance of females (55% of cohort) to males (45%). About 43.6% of patients had at least one parent with type 2 diabetes, but the majority (88.4%) of patients did not have any siblings with T2DM. Acanthosis nigricans is a well-established marker of insulin resistance and has been reported to occur in up to 60-90% of North American children with type 2 diabetes but was noted in 37.6% of our study cohort. The large majority (96.7%) of patients were diagnosed by clinical judgment. Approximately three in five patients were diagnosed by autoantibody negativity (56.0%) and elevated C-peptide/insulin(60.7%). The mean BMI of patients was 26.5 +/- 6.6 kg/m2 and (40.5%) of patients were found to be overweight.The regional mean HbA1c of patients was 8.0 +/- 2.8% while 40.4% had a HbA1c > 7.5 %. The mean fasting plasma glucose (FPG) was 9.1 +/- 4.9 mmol/l, while 54% of patients with had an FPG > 7.0 mmol/l. The regional mean Fasting Total Cholesterol (TC) was 4.8 +/- 1.1 mmol/l., mean fasting HDL-C of patients was 1.3 +/- 0.4 mmol/l, regional mean fasting LDL-C of patients was 2.9 +/- 1.0 mmol/l and the regional mean fasting triglyceride was 1.6 +/- 1.1 mmol/l. The proportion of patients having optimal control of total cholesterol, HDL-cholesterol, triglycerides and LDL-cholesterol was 43.2%, 74.1%, 59.5% and 53.4%, respectively. The proportion of patients in the region treated with anti-hyperlipidaemia medication was 2.6%. Although 10.2% had poor blood pressure control, only 4.1% of patients were treated with anti-hypertensive medication. The most frequent diabetes complication was microalbuminuria (8.0%), followed by neuropathy (1.2%).The class of oral antidiabetic drugs (OAD) most frequently prescribed in the region were the biguanide (53.5%), followed by the sulphonylureas (14.2%).Insulin was prescribed to 26.7% of patients, at a mean dosage of 36.0 +/-26.0 units/kg 0.6 +/- 0.4 units/day/kg. Overall, almost half (48.9%) of patients in the region were treated only with OAD, as compared to 15.4% of patients who were treated with a combination of insulin and OAD and 10.9% who were treated with only insulin. Overall, 59% of patients had more than four clinic visits within 12 months prior to the date of the visit. Blood glucose and urine glucose monitoring was performed in 64.6% and 4.9% of patients in the region. About three in five (63.5%) patients used a glucose meter to monitor their blood glucose levels. The frequency of blood glucose and urine glucose monitoring was 32.2 and 38.7 times/month, respectively. This study provided a much needed clinical and demographic overview of

the phenomenon of type 2 diabetes mellitus among children and adolescents in the Western Pacific region, and further leads us to conclude that glycemic control of these patients needs improvement.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 9

School Intervention of Type 2 Diabetes in Japan

Kaichi Kida

Department of Pediatrics, Ehime University School of Medicine, Shitsukawa, Toon-city, Ehime 791-0295, Japan

Type 2 diabetes is explosively emerging in children in Asia-Pacific region according to a rapid change of their lifestyles such as Westernization of eating habit and decreased physical activities. In fact, the incidence of childhood type 2 diabetes(age 6-15yrs) has been 4 times increased in parallel with a change of their lifestyles for these 30 years in Japan, which is now significantly higher than that of childhood type 1 diabetes. It is further demonstrated that chronic complications of the eye and kidney develop in children with type 2 diabetes as fast as or even faster than in children with type 1 diabetes. Furthermore, it is to be recognized that children in this region have the genetic insulin-resistance leading to type 2 diabetes compared with children in Caucasian populations. That is why interventions to prevent the development of type 2 diabetes or suppress the progress of chronic complication in children are needed, in this region. The intervention-strategies in Japan are (1) to detect type 2 diabetes at the early stage and avoid the progress of complications by screening of type 2 diabetes for all school children and (2)to prevent the development of type 2 diabetes in children with its risk such as obesity by screening of risk factors and lifestyle-interventions in school

In Japan, all school children undergo the diabetes screening with the urine test once a year according to the Law of School Health since 1992. The incidence of newly diagnosed type 2 diabetes is 5-7 to one hundred thousands of school children. The prevalence of eye and kidney complications is several time higher in those who drop out from the treatment, which indicates that the early detection of type 2 diabetes by the diabetes screening is significantly beneficial for school children. The cost to find a new case of type 2 diabetes by the urine screening followed by OGTT is US$28,000 while the total expense for the medical care of complications and pension for one case is estimated to be US$118,000 and US$90,000 are saved for one case by the early detection.

Many of local governments in Japan run the health promotion program for school children to prevent the lifestyle-related diseases including type 2 diabetes. Risk factors for lifestyle-related diseases including obesity, dyslipidia and hypertension are screened and lifestyle-interventions are provided to school children with any of these risk factors by school nurses and school dietitians. Obesity, a major risk factor for type 2 diabetes, is significantly improved in 50% of school children by the lifestyle-interventions in school. School- and community-based lifestyle intervention could be a way to stop the explosively increasing trend of type 2 diabetes in children, particularly, in this region.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 10

New Insulins

Philip Home

Newcastle Diabetes Centre and University of Newcastle upon Tyne

Prior to the last decade, the last really new insulins were introduced in the 1940s and 1950s, though purity was significantly enhanced in the 1970s, and the minor advance of the introduction of human insulin occurred in the early 1980s. However that last innovation, which was allowed by the genetic engineering of insulin, also opened the door to entirely new insulins.

The effects of genetically engineered insulin have not been entirely predictable – in some insulin receptor interactions were very significantly changed in ways that adversely affected the safety profile of the insulin. This is now much better understood and seems to be associated with prolonged signalling from the insulin receptor. Other concerns have concerned enhanced IGF-1 activity.

But in other such insulin analogues the predictable changes in hexamerization properties have produced useful and safe insulins which are more physiological in terms of absorption profile. Proof of concept in control of post-prandial glucose excursions is well established. By themselves, when used with conventional basal insulin (NPH insulin), the other important effect of these insulins is to reduced nocturnal hypoglycaemia, and this effect has meant that improvements in HbA_{1c} are clinically small, though measurable. These rapid-acting insulin analogues are now regarded as safe for use in pregnancy, as well as in routine clinical practice, and have spawned new biphasic (pre-mix) insulins which seem to echo the gains in post-prandial glucose control and amelioration of hypoglycaemia.

Amino acid substitution can produce insulins with other properties however. One approach is to produce an insulin soluble in mildly acid solution, but which precipitates at the injection site, and gradually redissolves again to give prolonged effect. The first of these was unsuccessful, but subsequently insulin glargine has shown itself able to cover 24 h of basal insulin supply in the majority of people with diabetes, clinically giving improved basal blood glucose control and/or reduced nocturnal hypoglycaemia compared to NPH insulin in both major types of diabetes.

Another approach to producing an insulin with prolonged action is that of insulin detemir. Duration of action, not as long as insulin glargine, is prolonged mainly by binding of a fatty acid adduct on the insulin to albumin in the interstitial fluid of the injection site. Such binding also occurs in the circulation and probably accounts for this insulin's reduced variability of effect. This combination of improvements leads to improvements of basal blood glucose control and particularly of nocturnal hypoglycaemia. This insulin also has intriguing effects on body weight control.

As blood glucose control is a mixture of control of post-prandial and basal concentrations, it is not surprising that combined use of rapid- and prolonged-acting analogues gives synergistic effects. This has been demonstrated in people with Type 1 diabetes, and while people with Type 2 diabetes starting insulin appropriately early require only one injection of a basal insulin (with continued oral drugs), as their own islet B-cell function wanes meal-time analogues (separately or as a biphasic preparation) are again indicated.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 11

New Insulin Receptor Sensitisers

Professor Ken Shaw

Academic Department of Diabetes and Endocrinology, Queen Alexandra Hospital, Portsmouth, UK

Insulin resistance (IR) is a significant component of the T2 Diabetes/Metabolic Syndrome and adversely impacts on achieving optimal glycaemic control with current therapies. IR pathogenesis includes abnormal insulin receptor responsiveness and post-receptor insulin signalling, with disturbance of the tyrosine kinase/phosphorylation enzyme cascade, compounded by increased inflammatory cytokine activity.

Targeting IR and improving insulin sensitivity (IS) by overcoming insulin action pathway disturbance is a fundamental treatment objective. Exercise, weight reduction ± anti obesity agents and metformin are well established means of improving IS. PPAR gamma receptor activation reduces IR, with first generation thiazolidinediones (TZDs) also now established as effective treatment for T2DM.

New drug development to enhance IS aims to potentiate pathways of insulin action either by insulin receptor activation or via post-receptor signalling and stimulation of the phosphorylation cascade. Several agents including minerals (magnesium, chromium, vanadium, dopamine agonists, PKC inhibitors) have the potential to improve IS, but much current interest still derives from study of PPAR sub types expressed in different tissues, enabling novel pharmacological intervention.

PPAR gamma activation, as with TZDs, has regulatory effect on adipocytes, modulates glucose homeostasis and influences vascular function. PPAR alpha activation regulates genes involved in lipoprotein metabolism as well as inhibiting inflammatory responses (? vasculo protection). Reducing lipotoxicity is likely to improve IS. PPAR delta activation stimulates fatty acid oxidation in muscle and adipose tissue, and may be useful for treatment of obesity, IR and vasculo protection.

A number of PPAR gamma agonists, both TZD and non-TZD, are under investigation, but there is particular interest in dual PPAR agonists and possibly pan PPAR activation. Fibrates have significant PPAR alpha activity, which influences lipoprotein metabolism, reduces lipotoxicity, enhances IS and independently improves glycaemic control. Two interesting new dual PPAR agonists, Tesaglitazar and Muraglitazar, combine potential benefits of gamma activation, promoting fatty acid uptake and improved glucose metabolism with alpha activation addressing lipid homeostasis, and with possible advantage in terms of weight control and reducing CVS risk.

Tesaglitazar and Muraglitazar are currently undergoing phase III clinical trials. Preliminary reports indicate a dose-dependent metabolic improvement with similar outcomes for both compounds, including reduced triglycerides, raised HDL cholesterol and lower glucose levels. The prevalence of metabolic syndrome in insulin resistant patients may be lessened. Side effect profiles have yet to be fully evaluated, whilst the therapeutic application of these new agents in the context of existing drugs has still be determined.

Recent knowledge of the regulatory role of the endocannabinoid (EC) system for body weight and various metabolic processes, via central and peripheral mechanisms, offers further possible opportunity of enhancing insulin action. Over activation of the EC system is associated with increased food intake and obesity, and also nicotine dependence. Rimonobant, which blocks CB1 receptors, has been subjected to a number of clinical trials (the "RIO" Studies). Pooled data has shown significant reduction in body weight and in particular central obesity with improved metabolic profile: lower HbA1C levels, raised HDL cholesterol, reduced triglycerides and lower blood pressure, both systolic and diastolic. Glycaemic improvement is only partly explained by weight reduction. The STRATUS-US study showed the odds of quitting cigarette smoking were doubled and post cessation weight gain was markedly lessened. Stopping smoking alone would improve insulin sensitivity.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 12

Incretins

Professor David R Matthews

Oxford Centre for Diabetes, Endocrinology and Metabolism, Oxford , UK

Some decades ago the observation was made that glucose absorbed from the gut seemed to be more efficient than intravenous glucose in stimulating insulin secretion. This observation led to the isolation and characterisation of two hormones that were signalling directly to the beta cell and augmenting glucose mediated insulin secretion. The hormones were GLP-1 and GIP.

The observation remained only of academic interest until the ability to synthesise peptides became a reality. It transpired that GIP had little effect in type 2 diabetes but GLP-1 markedly augmented insulin secretion. Moreover it became apparent that this was a glucose dependent secretagogue so that there was little activity near normoglycaemia. Thus incretins held out the hope that here was a family of "smart" agents which would not have the adverse effects of clinical hypoglycaemia. This crucial observation meant that one had an agent which would lower raised blood sugar but would not cause clinical hypoglycaemia. GLP-1 has a very short half life and so therapeutically it is only useful when infused continuously. Nevertheless, the early data from such infusions demonstrated clearly the effects on hyperglycaemia without the induction of clinical hypoglycaemia. However, GLP-1 analogues – of which there are now several – can be given once or twice daily and have prolonged effects on the beta cell. These include are exendin-4, a peptide originally isolated from the saliva of the hela monster, liraglutide, a GLP-1 analogue with a fatty acid side chain. The disadvantage of these agents is that they need to be injected and an alternative strategy emerged which was the use of inhibitors of the peptidases which normally break down active GLP-1. This is the family of DPP-iv inhibitors. The advantage of these inhibitors is that they are small molecules which can therefore be given orally.

Incretins are know to have effects that are widespread outside the pancreatic axis as well. They seem to have central effects on appetite, slow gastric emptying alter hepatic glucose handling and may have effects directly on adipocytes. In clinical practice it was uncertain which effects would be apparent. Initial doses of GLP-1 and analogues suggested that nausea would be a problem, but it soon became apparent that this was very transient as a side effect, and doses could be quickly titrated upwards. The effects on appetite were measurable, and weight neutrality at least, and perhaps weight loss, was apparent with liraglutide. So another unwanted feature of diabetes therapy – namely weight increase – seemed to be addressed with incretin therapy.

A further possible limitation to GLP analogue use was the fear that tachyphylaxis (or down-regulation) would occur. Most peptides show this phenomenon. However over many weeks the incretins continue to exert their effects, and there seems to be no decline in efficacy with time.

Much more excitement has been generated with the incretin axis research because murine models imply that beta cell apoptosis is inhibited and neogenic process signalled by PDX-1 and IDX-1 are enhanced. At its most optimistic, projections for incretin use in type 2 diabetes might mark a step forward in prevention of progressive beta cell failure which is currently the hallmark of type 2 diabetes.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 13

Modified Diabetic Food to Suit Different Culture: Taiwan's Experience

Chwang Leh-Chii, DrPH, RD

Department of Food and Nutrition,Taipei Veterans General Hospital

Diabetes in Taiwan is the most rapidly growing chronic disease among adults. The prevalence of diabetes in general population is 4%. Its prevalence is 12.7% (1 in 8) among those who are over age 45, and 15.1% (1 in 7) among senior citizens (age > 65yrs). The estimated number of diabetic patients in year 2000 was approximately 1 million and the projected patient number will reach 1.4 million in year 2010. Around 2 decades ago, diabetes ranked the 11th in the leading causes of death, and its mortality rate was 7.91/100,000 in 1980. The mortality drastically increased to 39.26/100,000 in 2002, and diabetes has become the 4th leading cause of death since then.

The Status of Diabetes Management

The Diab Care (Taiwan) study groups surveyed 5,005 patients in 2002 and reported that the majority of patients had poor glycemic control. The mean HbA1c was 8.9±2.26%, only 1/5 (20.9%) of the patients had HbA1c < 7.0%. Nearly 1/2 of the patients (47.6%) were over weight, 2/3 (64.9%) had hypertension, 1/2 (52.6%) had dyslipidemia (high triglyceride and high total cholesterol), and 2/5 (40.7%) had hyperuricemia.

Diabetic patients accounted for 22.1% of total inpatient days. The cost for treating diabetic patients as revealed by the National Insurance Bureau was 11.5% of the total health care expenditure, which was 4.3 times higher than the average cost for non-diabetic patients. Diabetes treatment has become a major issue in health care.

Nutrition Intervention for Diabetes in Practice

Findings of these statistics raise concerns on the importance of glycemic management of the diabetic patients. Nutrition is a fundamental element in prevention and treatment for diabetes; considerable efforts have been undertaken by dietetic professionals in healthcare settings to conduct nutrition education and intervention for glycemic control.

Taiwanese diet mainly originated from Fu-jiang Province of South China and also has fused Japanese, western and various regional Chinese cuisines. Traditional Chinese medicine has been practiced for centuries. It is therefore not surprising to see Taiwanese, who share a similar culture, often going to Chinese medicinal halls despite the many well-established western medical facilities, or continuing to choose foods based on traditional hot/cold concepts.

The Chinese medicine named diabetes as "Shiao Keh Symptom", delineating the wasting and thirsty characteristics of the disease. Ancient Chinese tested diabetes by observing whether ants were attracted to a person's urine. Although Chinese medicine has attributed the disease to indulgence in gourmet savories and abnormality in pancreases since antiquity, much of the present diet therapy for diabetes practiced in Taiwan mainly adopts the ADA principles established in late 20th century, and is modified to suit local dietary culture.

General Dietary Principle for Diabetic Patients

- *Discuss with dietitians and work out a diet plan according to each individual's dietary habit, life style and medication.*
- *Diabetic diet is a balanced meal, composed of 6 basic food groups.*
- *Carbohydrate and total calories are distributed to each meal to avoid fluctuation in blood sugar. (Carbohydrate count is stressed for patients who have type 1 DM or eat out frequently).*
- *Choose high fiber foods, such as whole grain, legumes and vegetables to increase bulky*

feelings.

- *Prepare foods by steaming, boiling, poaching and roasting, instead of frying.*
- *Avoid pickled, salted and highly seasoned foods.*
- *Avoid sugar concentrated foods, desserts and drinks.*
- *Keep food record, bring it to follow-up counseling sessions and discuss with dietitian periodically.*

Steps of Diet Plan:

1. Estimation of calorie requirement is based on IBW or Adjusted BW for obese patients.

(1) IBW

- *BMI (22) x Ht2*
- *Male : (Ht – 80) x 0.7 ± 10%, Female : (Ht – 70) x 0.6 ± 10%*

(2) Adjusted BW = IBW + (Actual Wt – IBW / 4)

2. Calorie Requirement = IBW or Adj BW x Kcal/BW Kg

Activity	*Kcal /Kg BW*
Sedentary	*20*
Light	*25*
Moderate	*30*
Heavy	*35*

3. Distribution of energy intake by Protein: Fat: CHO ratio = 15~20%: 30~35%: 50~55%

4. Selection of foods by exchange list. Sample daily exchange and menus are shown in APPENDIX.

Exchange List

1. Rice and starches (2 g protein, 15 g carbohydrate and 70 Kcal)

Like many other Asian countries, rice is the most popular staple in Taiwan. Rice is central to Taiwanese food culture. The most common term for "eat" is "chi fan" (literally, eat rice) and one of the most common greetings is "chi koa fan le ma?" (Have you eaten rice yet?).

A typical Taiwanese meal is consisted of plain steamed rice, 1 main dish (braised meat or fried fish), 1-2 side dishes (meats and /or soy products stir-fried with vegetables, or plain vegetables) and soup. Noodles topped with meats and vegetables are also widely consumed. Rice porridge, steamed bun, with or without fillings bread and sandwiches are common breakfast items.

Each rice exchange contains about 15 grams of carbohydrates, 2 grams of protein, and a trace of fat for a total of 80 calories, serving size may vary. One rice exchange = 1/4 bowl of cooked rice = 1/2 bowl of rice porridge or cooked cereal = 1/2 bowl of noodle (mee, mian), kuay teow or vermicelli made from rice or wheat flour, or glass noodle (tung fen) made from mung bean starch = 1/4 package of instant noodle = 1 slice of bread = 1/4 steamed bun (bao).

Sticky rice and long-grain rice are usually processed to make rice cake or dumpling used for light meals, desserts or festival foods. One rice exchange = 1/2 bowl of rice cake = 13 small round dumplings = 1/5 sticky rice wrapped in bamboo leaves.

Wheat products, in addition to noodles of different shapes, include wonton and dumpling. One rice exchange = 2 spring roll wrapper = 4 dumpling wrapper = 7 wonton wrapper (thin) = 3 wonton wrapper (thick).

Starchy root plants, e.g. sweet potato, and legumes, seeds, nuts of low fat content, e.g. mung bean, lotus seed, can be served alone or prepared with other ingredients to make a dish or dessert. These foods also belong to rice exchange group. One rice exchange = 1/2 potato, sweet potato, or taro = 1/2 corn = 1/4 bowl of mung bean, azuki bean (small red bean), or broad bean
= 5 chestnut = 7 water chestnut = 32 lotus seed.

It is interesting to find that the serving size of rice exchange varies in different rice-consuming communities. In Taiwan, 1 rice exchange = 50g (1/4 bowl) cooked rice (70 Kcal). In Hong Kong, 1 rice exchange = 30g (1 T or 1/6 bowl) cooked rice (45Kcal). In China, 1 rice exchange = 25g raw rice (90 Kcal). In Japan, 1 rice exchange = 55g cooked rice (80 Kcal).

2. Vegetables (1 g protein, 5 g carbohydrate, and 25 Kcal)

Taiwanese meal would not be complete without a plate of leafy vegetables, usually quickly stir-fried with garlic cloves in wok over high flame, or poached in boiling water, then topped with chopped shallot and pork sauce. A wide variety of vegetables make appearance in everyday Taiwanese cuisine. Vegetables are also stir-fried with slices of meat to make side dish, or braised with chunk of meats to make entrâe, or simmered with other ingredients to make soup.

One vegetable exchange = 1/2 bowl of cooked leafy vegetables, squash, bamboo shoot, mushroom, bean sprout, turnip or carrot = 1 bowl of raw vegetables. Each group contains 5 grams carbohydrates, 2 grams of protein, and 25 Kcal.

3. Fruits (15 g carbohydrate and 60 Kcal)

Taiwan locating at the semi-tropical zone has abundant production of fruits all year round. Imported fruits from other parts of the world are also available at affordable price. Fruit stalls and juice bar are everywhere. Fresh fruits are usually served at the end of a meal as dessert. Fruit juices of every kind are popular beverages.

Fruit exchange has 15 grams of carbohydrates and 60 Kcal. The serving size varies with kind of fruits, e.g. 1 medium or 1/2 banana is one exchange; it also varies with the increased sugar content as well as the enlarged size of the new variety. One fruit exchange = 1 medium orange, mango (local variety, small, green), guava (local verity), star fruit, apple, pear, peach, plum, apricot, or kiwi = 1 slice watermelon (300g) = 1 slice pineapple (120g) = 1/2 grape fruit, guava (Thai variety, large), melon, or banana = 1/2 C orange juice = 1/3 pomelo, or cantaloupe = 1/4 papaya, mango (new variety, large) = 1/4 clove of durian = 2 wax apple = 5 mangosteen (manggis) = 9 lychee or cheery = 12 grape, or longan (dragon's eye fruit).

4. Meats and Soy Products

The exchange groups for meat are categorized by lean meat (30~50 Kcal), medium-fat meat and soy product substitutes (75 Kcal), and high fat meat (100 Kcal). Exchange sizes on the meat list are generally one Taiwan ounce (1 liang = 37.5 g) of cooked meats, e.g. pork, beef, poultry, fish, egg; or one piece of soybean products or gluten products.

- *Meats High-Fat (7 g protein, 8 g fat, and 100 Kcal)*

= 1 liang (37.5g) ham, sausage, cured meat, belly pork. (Not recommended)

- *Meats Medium Fat (7 g protein, 5 g fat, and 75 Kcal)*

= 1 liang (37.5g) lean meat (pork, beef, poultry, or fish) = 2/3 chicken drum stick without skin
= 2 T dried pork or fish hash = 1 egg (avoid salted or preserved duck egg) = 6 quail eggs

- *Meats (lean) (7 g protein, < 3 g fat, and 30~55 Kcal)*

= 1 liang (37.5g) chicken breast without skin = 2 liang (75g) squid = 8 shrimp = 4 prawn
= 1 T dried fish = 1 piece sea cucumber = 1 piece jelly fish = 8 oyster (small) = 22 clam
= 1/2 crab

Soybean products and gluten products extracted from wheat flour have meant vegetarian meat to people in China for centuries. The different varieties of tofu, soybean curd made from soybean milk, have played a dominant role in Chinese cooking. Acquiring cholesterol-free nature as well as rich protein and unsaturated fat contents, soy product is not just a meat substitute, but rather an essential protein food in daily meal plan for diabetic patients.

- *Soy and gluten products (7 g protein, 5 g fat, and 75 Kcal)*

= 1 tofu = 1/2 box tofu = 1 black dry tofu = 2 five-spice dry tofu = 2 fried tofu = 3/4 soy pouch
= 1 bowl soy milk = 1 T dried soybean = 1/2 bowl fresh soybean = 1/2 gluten product

5. Milk and milk products

Milk group is usually one cup or 8 oz. In order to reduce fat intake, skim milk and low-fat milk are recommended to replace whole milk. Dairy products are rich source of protein, calcium and vitamin B2, but they are not popular food items in Taiwan, since many people have lactose intolerance.

- *Whole milk (8 g protein, 8 g fat, 12 g carbohydrate, and 150 Kcal)*

= 1 C (milk fat > 3.5%) = 3 T whole fat milk powder (not recommended)

- *Low fat milk (8 g protein, 4 g fat, 12 g carbohydrate, and 120 Kcal)*

= 1 C (milk fat < 1.5%) = 3 T low fat milk powder

- *Non-fat milk (8 g protein, trace of fat, 12 g carbohydrate, and 80 Kcal)*

= 1 C (milk fat < 0.5 %) = 3 T non-fat milk powder = 3/4 slice low fat cheese

= 200ml low fat yogurt

6. Oil and fat

An oil exchange is usually 1 teaspoon of oil or fat, and some oil-rich nuts, seeds. Avoid saturated fats and choose polyunsaturated or monounsaturated fats instead.

- *Oil and fat (5 g fat and 45 Kcal)*

= 1 t vegetable oil (soybean, corn, peanut, sesame seed, canola, olive, or rice bran oil)

= 1 t animal fat (lard, butter, dripping), not recommended

= 1 t satay sauce, chili sauce

= 1 t margarine, cheese, peanut butter, or salad dressing

= 1/2 sausage

= 2 walnuts = 4 macadamia nuts = 5 cashew nuts = 15 peanuts

= 27 pumpkin seed (15 g) = 65 watermelon seed (45 g) = 105 sunflower seed (16 g)

7. Free foods

Foods of very low calories are free to choose, e.g. bottled water, plain tea, black coffee, sugar free carbonated soft drinks, sugar substitutes, gelatin, konniyaku, senchou (dark jelly made from cincau plant), and raw or poached greens.

Cooking and Seasoning

Diabetic patients usually have complication of over weight, hypertension, and hyperlipidemia, therefore food preparation emphasizes on cutting down fat, salt and sugar. Stir-frying is one of the most common ways of everyday cooking. The use of fat as cooking medium adds richness of flavor and also adds fat content. It is advised to use vegetable oil instead of animal fat for cooking, and also shift to water as cooking medium, e.g. boiling, steaming, poaching, stewing, and simmering.

The use of soy sauce and other seasonings high in sodium needs to be reduced. Sodium equivalents of seasonings are as follows: 2000mg Na=1 t salt = 5 t MSG = 5 t worcestershire sauce = 6 t soy sauce = 7t oyster sauce =10t miso or soy sauce paste = 12 t ketchup = 15t barbecue sauce. Pickled or salted vegetables and soy products having strong flavor are appetizing and favorable to the elderly who are losing taste acuity, should be limited too. An alternative approach to enhance food flavor is by using spring onion, ginger, garlic, parsley, basil, chili, medlar, pepper, or five-spice powder (mixture of cassia bark, star anise, clove, Sichuan pepper, and dried tangerine peel).

Tonic and Taboo Foods

People nowadays are still embedded with traditional folklore food beliefs. Sick and weak people are usually at the "cold" status, and need to ingest "warm" and "tonic" foods to maintain body in a state of harmony. Hospital menus therefore tend to exclude "cold" foods (e.g. duck), "toxic" foods (e.g. eggplant, pumpkin), or other taboo foods upon request. Beef is seldom served in hospital wards, because some patients consider buffaloes used to be farmers' working partners, and should be treated as friends not as food materials; some others do not eat beef simply due to religious reason. A number of patients turn into vegetarians after getting sick. Organ meats (liver, brain, kidney...) and fish roe, though the general public regards them as tonic delicacies, are not included in hospital menu due to rich contents of cholesterol.

Challenges to Nutrition Care for Diabetes

With the trend toward eating more meals outside the home, it is found that weighing food in restaurants is impractical. Since Taiwanese cuisine often contains several ingredients mixed in one dish, which makes judging serving sizes even more difficult. Although packed foods with nutrition labeling has nutrition

information on calories, protein, fat, carbohydrate and sodium, however, nutrition labeling is not mandatory to all foods. Therefore instruction of evaluating food consumption is a task for both patients and dietitians. Lack of compliance and motivation of patients further weakens the efficacy of nutrition education.

The Bureau of Health Promotion has taken initiatives to build up an island-wide Diabetes Care Network by collaborating joint efforts of physicians, dietitians, pharmacists and nurses since year 2000. Another strategy is changing the health insurance policy to reimburse hospital payments only after patients have completed 4 visits with diabetic education sessions. Under these circumstances, referrals to dietitians have indeed increased. Around 80,000 patients were enrolled in the program.

Dietitians engaged in the Network must be certified diabetic educators. Functions of dietitians in the diabetic care team are:

- *Monitoring anthropometrical measurements and lab data,*
- *Evaluating dietary habit and life style, and assessing food consumption,*
- *Implementing nutrition education on food choices and diet plan, and*
- *Diagnosing nutritional status.*

It should be noted that the 2004 survey also indicated only 1.3% of patients had HbA1c < 7%, BP <130/80 mmHg and LDL-C < 100 mg/dl, in other words, diabetic patients together with health care team members have to make more efforts to combat the threat of the disease.

APPENDIX-1: Sample Daily Exchange

Exchanges from each food group for the listed calories

Kcal	1200	1500	1800	2000	2200
Vegetables	4	4	4	4	4
Fruits	2	2	2	2	2
Rice	7	9	12	13	15
Meat	3	3	3	4	4
Soy	1	2	2	2	3
Oil	6	7	7	8	9
Milk	1	1	1	1	1

Exchanges of meal distribution for 1500 calories

	Breakfast	Lunch	Snack	Dinner	Snack
Vegetables	1	1.5	0	1.5	0
Fruits	0	1	0	1	0
Rice	2	2.5	1	2.5	1
Meat	1	1	0	1	0
Soy	0	1	0	1	0
Oil	1	3	0	3	0
Milk	0	0	0	0	1

Exchanges of meal distribution for 1800 calories

	Breakfast	Lunch	Snack	Dinner	Snack
Vegetables	1	1.5	0	1.5	0
Fruits	0	1	0	1	0
Rice	2	4	1	4	1
Meat	1	1	0	1	0
Soy	0	1	0	1	0
Oil	1	3	0	3	0
Milk	0	0	0	0	1

APPENDIX -2: Sample Menu

Day 1

Breakfast	Lunch	Snack	Dinner	Snack
Fish hash	Bamboo shoot w/ sliced pork	Mung bean w/ pearl barley	Beef w/ black pepper sauce	Cookies w/ low fat milk
Egg-tofu	Onion w/ jelly fish		Soy pouch w/ basil	
Braised kelp	Vegetable		Greens	
Rice porridge	Steamed rice		Steamed rice	

Day 2

Breakfast	Lunch	Snack	Dinner	Snack
Pork hash	Chicken w/ celery	Boiled taro cake	Steamed cod fish	Purple sticky rice
Braised gluten	Shrimp tofu		Stir-fried egg & tomato	
Stir-fried cabbage	Greens		Vegetable	
Rice porridge	Steamed rice		Steamed rice	

Full text. e-Journal: *http://www.medassocthai.org/journal*

References

1. Health Statistics, National Health Department, Taiwan, 2005, available at http://www.doh.gov.tw/statistic/index.htm
2. Chuang LM, Tsai ST, Huang BY, Tai TY. The current state of diabetes management in Taiwan. Diabetes Res Clin 2001; 54: Suppl 1:S55-S65.
3. Lin T, Chou P, Lai MS, Tsi ST, Tai TY. Direct cost-of-illness of patients with diabetes mellitus in Taiwan. Diabetes Res Clin Pract 2001; 54: Suppl 1:S43-6.
4. Chwang LC. Nutritional support and education for diabetic patients in Taiwan. Presented at 10th PENSA Congress, 2004, Thailand.
5. Nutrition handbook for diabetes, Department of Food & Nutrition, Taipei Veterans General Hospital, 2004.
6. Food exchange lists, Department of Food & Nutrition, Taipei Veterans General Hospital, 2004.
7. Tung TY, Chwang LC. Diet and nutrition for diabetes nephropathy. Clinical Medicine 2003; 52:316-320.
8. Lin CS, Chwang LC. Diet principles for diabetes. Clinical Medicine 2003; 52:321-324.
9. Shu YF, Chwang LC. Medical nutrition therapy for diabetic patients with complications. Clinical Medicine 2003; 52:325-328.

SS 14

The Challenges of Optimizing Diet Among People with Diabetes – The Malaysian Experience

Mary Easaw-John

Chief Dietitian and Senior Manager, Dietetics and Food Services, National Heart Institute, Kuala Lumpur, Malaysia

The global burden of people with diabetes is estimated to reach 300 million in 2025 (King, 1999) with type 2 diabetes. Type 2 diabetes is common in the adult population but in the last two decades it has emerged as a problem among the children and adolescents. This is due to an increase in the prevalence of overweight, obesity and a sedentary lifestyle.

In Malaysia, the Second National Health and Morbidity Survey conducted in 1996 reported a rise in the prevalence rate of 8.3% (NHMS 2, 1996) as compared to 6.3% in the 1986 (NHMS1, 1985). Prevalence of diabetes mellitus varies amongst ethnic groups with 16% among the Indians and 4.7% among the Malays. (Khalid BAK et al. 1990). There is limited data on the prevalence of diabetes mellitus among the Chinese in Malaysia. Pockets of study have been done on effectiveness of dietary counseling among non insulin dependent diabetes mellitus patients in the community. Majority of the patients are seen in the hospitals.

According to American Diabetes Association 2004, nutrition recommendations and principles for people with diabetes are

1. *Attain and maintain optimal metabolic outcomes including*
    a. *Blood glucose levels in the normal range or as close to normal as is possible to prevent or reduce the risk of complications of diabetes.*
    b. *A lipid and lipoprotein profile that reduces the risk for macrovascular disease*
    c. *Blood pressure levels that reduce the risk for macro and microvascular complications.*
2. *Prevent and treat the chronic complications of diabetes. Modify nutrient intake and the lifestyle as appropriate for the prevention and treatment of obesity, dyslipidemia, cardiovascular disease, hypertension and nephropathy.*
3. *Promote better health through healthy food choices and physical activity taking into consider ation individual needs and cultural preferences while respecting the individual's willingness to change.*
4. *For pregnant and lactating women, to provide adequate energy and nutrients needed for optimal obstetric outcomes.*
5. *For children and adolescents, to provide adequate energy and nutrients for optimal growth and development.*
6. *For older adults, to provide for the nutritional and psychosocial needs of an aging individual.*
7. *For individuals treated with insulin or insulin secretagogues, to provide self management education for treatment and prevention of hypoglycaemia and exercise related blood glucose fluctuations.*

Dietary advice for people with diabetes has been a controversial issue for many years. Many would also resort to traditional medicals and herbs as a part of their treatment. People with diabetes need to understand that the diet has to help them to maintain weight or loss weight reasonably, reduce blood glucose and blood lipid levels and delay complications of the diabetes. (Franz MJ Diabetes Care 1994). These goals are most effectively met through a combination of exercise, drug therapy and nutrition therapy. Diet therapy is an essential and lifelong component of treatment for all diabetics, regardless of the client's weight, blood glucose levels, or use of medication.

Advising the Malaysian with diabetes

The goal of diet counseling is to facilitate behavior change, not merely to pass along the information. Patients who are told to make numerous dietary changes may feel overwhelmed and resentful especial if their diet is already modified for other chronic diseases. The changes of success maybe greatly improved by setting only one goal rather than completely overhauling the diet. Learning goals should be mutually developed. Reinforcement and feedback are useful in helping the patient achieve his dietary targets. Diet modifications should be made sequentially rather than simultaneously.

In my experience as a dietitian with 20 years of counseling experience I have noted the following with our patients.

• When a patient is newly diagnosed for diabetes refrain from using terms such as mild or borderline diabetes. The patients will think that diabetes is still in control and can enjoy any food, as the diabetes is 'mild'.

• We need to tell them that diet, exercise and medical management is important. Exercise is another important aspect of treatment for both type I and type II diabetes, regardless of weight status, unless contraindicated for other medical reasons

• Meal spacing should be an important consideration for type 2 diabetes. Eating meals for 4 - 5 hours apart allows post prandial glucose level to return to base line. Type 2 diabetes will be able to maintain to appropriate blood glucose level and maximize drug effectiveness by eating meal of approximately the same composition at approximately the same time every day

• Tell them the timing of meals is very important.

• The total carbohydrate intake for the day must be consistent from day to day. This means that the same amount of carbohydrate should be taken for each breakfast, each lunch, each dinner and each snack. In another words, the portion of food must be consistent.

• Sample meal planning must be discussed with them. As Malaysia is a multi racial country the cultural and food beliefs must be considered.

• Inform them about hidden sugars in the food. Some common Malaysian food and drinks are:

	(Food and drinks)	**(Better to choose)**
1.	Teh tarik kurang manis	Teh see (tea with evaporated milk)
2.	Local sweet kuih	Savoury kuih
3.	Sweet sour sauces	Masak assam pedas or soup or curry
4.	Carbonated drinks or sweetened drinks or cordial, air bandung	Plain barley water, Chinese tea and Ω cup freshly squeezed fruit juices
5.	Kuah rojak or kuah satay	Eat small amount
6.	3 in 1 beverages and sweetened cereals	Go for plain or individually served

1. The Glycemic Index

People with diabetes have been advised to eat a high carbohydrate diet for many years. In fact, health professionals now recommend the approximately half, to two thirds of all the food we eat come from foods that are high in carbohydrate. But all carbohydrate foods are not the same.

Traditionally, carbohydrate-containing foods have been classified as either containing simple sugars, like fruit, dairy foods and table sugar, or complex carbohydrates, like breads, cereals, legumes and starchy vegetables. This was based on the physical structure of the carbohydrate in the food. It was assumed that complex carbohydrates were slowly absorbed into the blood, while simple sugars were thought to be rapidly absorbed.

Glycaemic index or GI is a measure of how quickly or slowly the body metabolises the carbohydrate in foods into glucose in comparison to a reference food. Low GI foods (GI range below 55) are foods that are slowly digested and absorbed while high GI foods (GI range more than 70) are foods quickly digested and absorbed. Intermediate GI food has a GI range between 55 and 70 (Brand Miller et al, 1996). High GI foods may not necessarily contain the most sugar or the least fiber. Sugar (sucrose) is classified as an intermediate GI food.

Use of GI concept in diabetes management

• Primary nutrition intervention which focuses on educational approaches such as reduced energy intake, modest weight loss and basic carbohydrate counting has been shown to be more effective in glycaemic control than the low GI approach on its own (i.e. reduction of 1-2 unit (%) HbA_{1c} as compared to 0.5 unit (%) HbA_{1c} respectively) (Brand Miller, 2003)

• Choosing low-GI foods in place of conventional or high-GI foods has a small (~0.4% unit HbA_{1c} reduction) effect on medium-term glycaemic control (Evidence A) (Brand-Miller, 2003). However, there is insufficient evidence from large long-term studies (ADA, 2004)

• Low-GI diet is not recommended as a primary strategy in meal planning (ADA, 2004)

• If the concept of GI in dietary advice is chosen, GI must be used to complement established dietary concerns such as the amount and type of fat, fiber, salt content with emphasis on total amount of carbohydrate in meals and snacks. Some foods low in GI but high in fat e.g. ice cream need to be limited whereas some foods with high GI but high in nutrients e.g. carrots are still encouraged.

• It is not necessary to avoid all high-GI foods. Exchanging at least two meals per day (or > 50% total carbohydrate) from high to low GI will lower the GI of the whole diet by about 15 units. This is sufficient to bring about clinical improvement in glucose metabolism in people with diabetes (Brand, 1991)

Rule of thumb

High GI food + Low GI food = Intermediate GI Food

Refer Appendix 12 for glycaemic index of selected foods (Foster-Powell et al., 2002). There are limitations in using the list as there is scarcity of data on local foods. More foods need to be tested for GI using standardized methods. It is recommended that self-blood glucose monitoring should be done before meals and 2HPP to evaluate blood glucose response to different types of common foods to improve selection (Evidence C).

Glycaemic load (GL)

Glycaemic load or GL is calculated by multiplying the amount of carbohydrate contained in a specified serving size of the food by the GI value of that food (with the use of glucose as the reference food), which is then divided by 100.

Glycaemic Index represents the quality of the carbohydrate but does not take into account the quantity of the carbohydrate consumed. Total amount of available carbohydrate in meals or snacks is more important than the source or type (low or high GI) (Evidence A). The higher the GL, the greater the expected elevation in blood glucose and in the insulinogenic effect of the food. There is still insufficient data to determine the efficacy of GL in diabetes management.

How to apply the GI to their eating plan?

To incorporate the Glycemic Index into their eating plan here are some tips.

• Have at least 3 low GI foods throughout the day, ideally one at each meal

• All carbohydrates that you eat do not need to be low GI

• You do not have to avoid all high GI foods, but try and eat them with low or intermediate GI foods whenever possible. This will bring down the average GI of the meal

• Include low fat, high carbohydrate foods with each meal and snack

• Try to spread the amount of carbohydrate you eat evenly throughout the day.

2. Understanding what's on the labels – for patients with diabetes

Patients need to be aware of other terms, which mean literally sugar in bombastic words! These are:

• Corn syrup, high fructose corn syrup, maple syrup

• Fruit juice concentrate

• Honey, molasses

• Raw sugar

• Dextrose, lactose, maltose, sucrose

In well control diabetes, sucrose (sugar) can be taken in very small amounts about 1-2 teaspoons

daily in place of a carbohydrate as a part of a meal plan. However, foods with sugar are usually nutrient poor and maybe in high in fat. In the Malaysian scenario, most food is prepared with sugar especially in sambals*, sweet sour sauce etc. People with diabetes are therefore advised to consume sugar judiciously.*

3. Use of artificial sweetener for patients with diabetes

Artificial sweeteners are divided into

• Nutritive – these are similar to sucrose (sugar) and sugar alcohol such as sorbitol, mannitol and xylitol. Such alcohol can caused a smaller increased in glucose than sucrose and carbohydrates do, but they may caused abdominal gas, discomfort, and osmotic diarrhea when consumed in large amount. It better to ask patient to use no sugar added jam than 'diabetic jam'.

• Non nutritive – Saccharin, aspartame, and acesulfame – K are approved for use and maybe safely used by people with diabetes

4. Alcohol Intake and Diabetes

Moderate use of alcohol in well control diabetics does not effect blood sugar level. However, alcohol intake should be

• limited to less than 2 drinks per day. As doctors, you need to emphasize that they cannot drink the alcohol accumulated, for e.g. 7 drinks one go during the weekends.

• consumed with food and not between meals, because it can cause hypoglycemia. This is usually difficult for a person who drinks alcohol and to eat the food simultaneously.

• not taken by type 1 diabetic to compensate calories from their food intake.

• avoided during pregnancy and lactation.

• discouraged by people who are trying to loose weight.

• not taken by people with high triglyceride levels.

• avoided by people who have a history of alcohol abuse.

5. Encourage patients to eat 20-30 grams of fiber daily preferably from a wide variety of food

Soluble fiber can delay glucose absorption but fiber itself has other benefits such as

• increasing the volume of the diet without increasing the calorie (plus for weight management),

• providing vitamins and minerals, preventing constipation and reducing serum cholesterol.

• the Malaysian fruits and vegetables provide varying amount of fiber to the diet. Whole meal bread, rye bread, wholegrain breakfast cereals, capattis*,* putu *made from atta flour are some examples of whole grains.*

• 5 servings of fruits and vegetables per day, 1 serving of beans and pulses and 1 serving of whole grains food per day should be able to provide the recommended fiber intake per day.

6. Limit cholesterol intake to 300 milligrams or less to decrease the risk cardiovascular diseases

• You need to emphasize to your patients especially those on lipid lowering drugs that the drugs will not do the trick if they continue to eat a high fat and cholesterol diet.

• Limit organ meats, egg yolks, butter, fish roe etc

• Reduced intake of fat especially total saturated fat

• Emphasis on that cholesterol is found only in animal foods.

7. Fats Intake for person with diabetes

The recommendation for total fat is 30% from total calories of the meal plan.

• This amount is appropriate for those with normal lipid levels and normal weight.

• However, diets containing 25% calories from fat maybe appropriate for obese patients trying to loose weight.

• Diabetes with high level of triglycerides may see a greater improvement their lipid, glucose and insulin levels by consuming a total fat of 30% with increase fat coming from polyunsaturated or monounsaturated fat. The calories coming from the carbohydrates can be decrease by 5% to control the triglycerides.

• *Santan (coconut milk) based food is allowed in small portion once a week.*

• *Trans fatty acids are usually found in margarine and bake products and can be a source to raise blood cholesterol levels. Trans fatty acids are formed when a liquid based margarine is hydrogenated to formed solid and improved shelf life of the margarine. Margarine containing trans may be used sparingly but not to replace cooking oil. Palm oil based margarine do no contain trans fatty acids*

8. Protein intake for patients with diabetes

• *Protein should provide 10-20% of total calories from the meal plan for the day*

• *There is lack of evidence to support either a higher or lower protein intake helps the patients*

• *However if the patients has an indication of renal disease that i.e. the glomerular filtration rate (GFR) begins to fall a reduction in protein intake from 0.8 g/kg (normal recommended dietary allowance) to 0.6g/kg may help slow the rate of GFR decline.*

• *Lean protein such as lean red meat, chicken without skin and fat, fish, and vegetable protein such as bean and pulses are recommended.*

• *Seafood such as squids, crabs, prawns and other shellfish can be consumed in small quantities once in a week.*

9. Sodium Intake for person with diabetes

For hypertensive with diabetes, less than 6g salt per day is allowed. However they should avoid salt rich foods such as belacan, cincaluk, budu*, salted fish, pickles, soya sauce, oyster sauce, commercial seasonings and allowed* Ω *teaspoon salt in cooking per day.*

10. Eating out challenges

Eating out is always a challenge. In Malaysia in the last 10 years, there is a mushrooming of 24 hours restaurants. This means that you are tempted to eat out at any time of the day or night. Education based on the following should be emphasis

• *Control or share the food portion.*

• *Ask questions & make request for changes.*

• *Scan the menu for small servings.*

• *Choose healthy choices e.g. noodle soups,* assam pedas*,* yong tauhu, chappatis, thosai, ikan bakar

• *If there are extras after a meal ask for a doggie bag.*

***11. Diabetes Management during the Fasting month of* Ramadan**

Fasting during the month of Ramadan *is one of the five pillars of Islam hence it is obligatory for all healthy adult Muslims. The* Ramadan *fast can last up to 14 hours from dawn to sunset in the day during which the individuals abstain from taking food, drink and oral medication. There is a tendency to consume a large meal in the evening at the break of fast and a variable amount of food before dawn, which may adversely affect glycaemic control.*

Adjustment of the diet protocol for Ramadan fasting

• *Never skip* sahur *(dawn meal). Sahur should consist of a balanced meal with adequate carbohy drate taken as late as possible just before* Imsak(beginning of the fast) *to avoid unnecessary prolonged fasting.*

• *Do not delay "berbuka" i.e. the breaking of the fast at sunset, also known as Iftar. Limit intake of high-sugary foods e.g. kuih. However, 1-2* kurma *(dates) at start of berbuka according to* Sunnah *may be taken as part of the carbohydrate exchange. The main meal is encouraged after* Maghrib *(dusk) prayers.*

• *Supper after* Tarawih *(special prayers during Ramadan) can be taken as replacement of the pre-bed snack.*

• *Include fruits and vegetables at both* sahur *and* berbuka. *High fibre carbohydrates are encouraged at all meals.*

• *Limit fried or fatty foods.*

• *Limit intake of highly salted foods to reduce risk of dehydration.*

• *Sufficient fluid must be taken to replenish fluid loss during the day. Aim for 8 glasses a day. Choose sugar-free drinks. Drink adequately at* sahur. *Have a drink after each visit to the toilet to replace loss. Dietary indiscretion during the non-fasting period with excessive gorging, or compensatory consumption of carbohydrates especially sweetened and fatty foods contributes to the risk of hyperglycaemia and weight gain.*

Monitoring

Blood glucose levels may be erratic during Ramadan. *Frequency of blood sugar monitoring should be increased to adjust medication especially if on insulin. Monitoring should be performed before sahur, 2-4 hours after sahur, mid to late noon, before berbuka, and 2-4 hours after berbuka.*

Patients with blood glucose < 3.0 mmol/l or symptoms of hypoglycaemia should break their fast (batal puasa) immediately and be managed appropriately.

Individuals should be advised that neither the finger-prick for self-monitoring of blood glucose nor injecting oneself with insulin will break the fast (batal puasa) (Omar, 1984).

After Ramadan, *the patient's therapeutic regime will revert to the regime prior the fasting month. An overall evaluation will also be required especially with regard to glycemic control and change in weight.*

11. Main challenge in nutrition therapy among the Malaysians with diabetes are as follows:

• *Are they consuming hidden or refine sugars?*
• *Are their meals on time?*
• *Are their portions of carbohydrate the same at each meal?*
• *Are they working on shift?*
• *Are they taking too many servings of fruits as snacks?*
• *Are their medication taken on time?*
• *Are they regular meal skippers?*
• *How much alcohol are they consuming?*
• *Monitoring their meals and blood control during the* Ramadan?

The presentation will discuss the above at the Congress.

In conclusion diet therapy is the cornerstone in the treatment of all forms of diabetes and must be followed permanently, along with medications even when no symptoms appear

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 15

Diabetic Nephropathy – from Epidemiology to Management

Juliana CN Chan

Professor, Department of Medicine and Therapeutics, The Chinese University of Hong Kong, The Prince of Wales Hospital, Shatin, NT., Hong Kong

There is now a global epidemic of diabetes and metabolic syndrome with Asia in the forefront. In contrast to the west where the main increase of diabetes will occur in the elderly, that in Asia will mainly occur in the young to middle aged group. In developed countries, despite there being a gradual decline in the incidence o f coronary heart disease, that of chronic kidney disease is rapidly increasing with diabetes and hypertension accounting for more than 70% of all new cases of renal replacement therapy. In this connection, there is growing evidence pointing to the high risk for renal disease in Asians. In a large scale survey involving more than 6600 Asian type 2 diabetic patients with hypertension, 40% were found to have micro and 20% have macroalbuminuria. With the onset of chronic kidney disease, there is further perturbation of the metabolic milieu characterized by micro-inflammation, acidosis, anemia, and abnormal bone metabolism. All these factors contribute to a 5-8 fold increased risk of cardiovascular disease in patients with chronic kidney disease compared to those with preserved renal function. Given the growing population of young to middle aged Asian diabetic patients and their predilection for renal and thus cardiovascular disease, the burden of these complications on both health care utilization and societal productivity will be phenomenal in Asia in the years to come.

The pathogenesis of diabetic kidney disease is multifactorial and involves genetic, metabolic, hemodynamic and growth factors. Both twin and family studies have demonstrated the familial clustering of diabetic kidney disease suggesting either genetic predisposition or shared environment. In Chinese type 2 diabetic patients, variants encoding the genes for angiotensin converting enzyme, hepatic lipase, apo E, aldose reductase have all been shown to be associated with increased risk of diabetic nephropathy. In support of the importance of these metabolic pathways, both epidemiology and interventional studies have confirmed the beneficial effects of blood pressure and blood glucose control in slowing the rate of deterioration of renal function in both type 1 and type 2 diabetic patients. Inhibition of the renin angiotensin system using either ACE inhibitors or AII receptor blocker (ARB) has also been shown to reduce the risk of progression of albuminuria. In particular, both the RENAAL and IDNT studies have confirmed the renoprotective effects of ARB in type 2 diabetic patients with overt nephropathy and renal impairment. While there is experimental evidence suggesting the importance of lipotoxicity on renal function, definitive evidence regarding the beneficial effects of lipid lowering on renal function is still awaited.

Given the complexity of the pathogenesis of diabetes and its complications, there is now increasing acceptance that protocol driven care using a multidisciplinary team may be a cost-effective strategy to reduce the risk of these devastating complications. Not dissimilar to the Steno 2 study, we have previously demonstrated that patients with overt diabetic nephropathy managed by a pharmacist-diabetologist team had 50% risk reduction in developing ESRD compared to patients treated by usual care. Taken together, identification of high risk subjects for nephropathy possibly using genetic factors may further increase the cost effectiveness of these structured care protocols. On the other hand, it is noteworthy that at least 10% of patients with overt nephropathy continue to develop ESRD yearly despite use of ACE inhibitors or ARB and optimal control of all risk factors. Thus, there is a need to explore novel therapy to retard the progression of renal function. To this end, there are ongoing studies to evaluate the use of dual blockade of the renin angiotensin system, aggressive lipid lowering and inhibitors of advanced glycation endproducts on development of diabetic nephropathy.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 16

Diabetic Retinopathy

Apichart Singalavanija, MD, FICS

Department of Ophthalmology, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand

Diabetic retinopathy is one of the major complications of diabetes. It is the leading cause of blindness and visual disability. Ten years after diagnosis of DM, the prevalence of retinopathy is 40-50%. After 20 years the prevalence is 60-90%. It is estimated that more than 2.5 million people worldwide have visual loss due to diabetic retinopathy. Diabetic retinopathy is classified into two main stages A) nonproliferative DR which is characterized by microaneurysms, retinal hemorrhages, intraretinal microvascular abnormalities, venous beading, cotton wool spots and hard exudates. B) proliferative DR which shows neovascularization of optic disc, retina and/or iris. The visual loss from DR is mainly caused by diabetic macular edema, vitreous hemorrhage, fibrovascular proliferation and tractional or rhegmatogenous retinal detachment. The management of diabetic macular edema is grid or focal laser photocoagulation, intravitreal triamcinolone injection or cutting the vitreous on macula. Vitreous hemorrhage and fibrovascular proliferation can be treated by pars plana vitrectomy, elimination of vitreous traction and removal of membrane- induced surface traction.

Screening for diabetic retinopathy is beneficial for early detection of the disease. Screening by ophthalmoscopy is simple and inexpensive. Nowadays digital fundus cameras are used and automatic screening by computerized programs are under experiment. Laser photocoagulation, control of blood glucose, blood pressure, normalization of lipids, and exercise will prevent the disease progression and reduce the risk of blindness.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 17

Update in the Management of Small Fiber Diabetic Neuropathy

Tae Sun Park, MD, PhD
Hong Sun Baek, MD, PhD, Ji Hyun Park, MD, PhD

Division of Endocrinology and Metabolism, Chonbuk National University Medical School

Small fiber diabetic neuropathy (SFDN) usually occurs early in the course of diabetes mellitus and often is present without objective signs or evidence of nerve damage. It is a subtype of sensory neuropathy, which manifests as painful feet and hyperalgesia may be associated with autonomic dysfunction. In parallel with increased understanding of the pathogenesis of diabetic neuropathy (DN), there must be refinements in our ability to measure quantitatively the different types of defects that occur in this disorder, so that appropriate therapies can be targeted to specific fiber types. Clinical examination may reveal a decrease in thermal and pinprick sensation, but muscle strength, proprioception, vibration sense and tendon reflexes are usually normal. In the diagnosis of SFDN, quantitative sensory test (QST), cardiovagal and adrenergic autonomic testing, assessment of intraepidermal nerve fibers (IENFs), and skin blood flow measurement can be used in the clinical setting. IENFs assessment is quantitative and highly sensitive and specific, so it can be used for serial testing in clinical setting. Other noninvasive methods for assessing skin blood flow have enabled serial clinical measurements of the effects of drugs on microvascular perfusion in SFDN patients. These tests must be validated and standardized to allow comparison between studies and a more meaningful interpretation of results.

Although, the pathogenesis of DN is complex and involves multiple pathways, the early diagnosis and management can delay and/or reverse the progression of SFDN. But the treatment for symptomatic relief is polypharmacy with adverse effects and drug interactions and only moderately effective. So early therapeutic intervention with combined therapies aimed at modulation and/or blocking of aberrant pathways might prove to be beneficial in the prevention and/or reversal of SFDN. Disease modifying treatment aimed at underlying pathophysiologic hypotheses based on polyol pathway activation, protein kinase C activation, oxidative stress. Recently clinical trials for safe and effective disease-modifying agents targeting the underlying pathologies have been accomplished.

Hyperglycemia induced metabolic change lead to dysregulation of cytokine control such as IL-1, IL-6, and TNF-alpha. Especially, the mechanisms of enhanced TNF-alpha production may be ascribed to macrophage stimulation by high glucose itself, hyperglycemia-induced oxidative stress, or exposure to advanced glycation end products. TNF-alpha has a possible role in neuropathic pain and correlations between tissue levels of TNF-alpha and pain and hyperalgesia. TNF-alpha has also been linked to the generation and maintenance of neuropathic pain. Studies in rats and mice showed that blocking TNF-alpha reduces hyperalgesia in models of SFDN. Thus, suppressants of TNF-alpha can be used for alleviating signs and symptoms of SFDN. Some drugs such as nicotinamide, ACE inhibitors, calcium channel blockers, gliclazide, and alpha 1 receptor blocker have inhibitory effects on TNF-alpha. One of the antioxidants known as the TNF-alpha suppressants, alpha-lipoic acid has a beneficial effect on SFDN patients. Accumulating evidence supports as the use of antioxidant and TNF-alpha suppressant in the symptomatic treatment of SFDN.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 18

Difficuties in Building a Diabetes Team : An Indonesian Experience

Aris Wibudi

President of Indonesian Diabetes Educator Association

It is well known that the rate of increase of diabetologists in Indonesia is not at par with that of type-2 diabetes incidence in the country. In order to overcome this difficulty, since 1991, the Indonesian Endocrinology Society has started an initiative to implement a Diabetes Educator (DE) Program, which was founded by Dr. Sidartawan Soegondo.

However, during the implementation of this program, several difficulties appeared as follows:

1. Inconsistencies in the understanding and appreciation of the DE concept as well as the minimal knowledge requirements that must be known by DEs. In order to overcome these problems, a diabetes guide book has been printed and published regarding the Integrated Management of Diabetes Mellitus in 1995. In 1999, a teaching methodology for DE and a training for basic DE trainers have been standardized in a book called the Diabetes Mellitus Educator Training Module. This book is a result of our collaboration with WHO.

2. Human resource limitation, i.e. the number of competent DEs. This enforces us to centralize the trainings to solely Jakarta.

3. These Jakarta-centralized trainings could not fulfill the needs of having many regional DEs.

4. Big funding is needed to educate DEs. Prospective DEs are not expected to bear the costs associated with trainings, in addition to the transportation and accomodation costs while they are in Jakarta. This problem has been solved by having sponsors as well as decentralized trainings by implementing Train the Trainers program. However, the decentralized trainings can only be done on the basic level, while those of the advanced level are expected to be conducted centrally in Jakarta.

5. DEs in Indonesia are not at the moment certified by the Indonesian Department of Health. Results of such trainings can not be applied towards professional credits for physicians, nurses, nor nutritionists. As such, frequently it is found that tranined DEs move to other departments in order that their organization ranks be elevated. Solutions are yet to be found to circumvent this problem.

6. Indonesia covers a wide area, causing communication problems and others related to supervising the DEs' activities in their respected regions. We have thought to solve the problem via internet, however, it is a fact in Indonesia that not all regional hospitals have access to the internet.

7. A uniformed understanding and appreciation regarding the importance of DEs have not be borned either by internists and endocrinologists in managing Diabetes Mellitus. We have done frequent socialization to physicians in order for them truly understand the important roles of DEs.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 19

A Model for Improving Diabetes Care in Adolescent with Diabetes

Jeerunda Santiprabhob, MD

Department of Pediatrics, Faculty of Medicine Siriraj Hospital, Mahidol University, Bangkok, Thailand

Type 1 diabetes is a very demanding medical condition for patients and others involved, including parents, family members, and medical personnel. It requires extensive behavioral changes, frequent glucose monitoring, and daily insulin injection. It is even more difficult for patients to adapt their behaviors to diabetes when they become adolescents. Adolescents are struggling to find their own identity, and a number of behavior adjustments required by diabetes can interfere with their drive for independence and peer acceptance. As a result, suboptimal adherence to the diabetes regimen in adolescents often leads to poor glycemic control. In this context, adolescents with diabetes need to be treated with the following goals: 1) promotion of physical and psychological well-being, 2) achievement of good glycemic control to prevent long-term microvascular complications, 3) integration of patients into the normal school, social, and working life of people in their age group, and 4) helping patients learn to be more responsible for their diabetes care.

A model for improving diabetes care for adolescents with diabetes should consist of the following:

1. Multidisciplinary team approach

The multidisciplinary team approach is a key concept in managing patients with diabetes. The multidisciplinary team should consist of pediatric endocrinologists, diabetes nurse educators, nutritionists, psychologists, and social workers. The team should provide psychological support and knowledge in diabetes management skills and assist adolescents with diabetes to become more independent in their diabetes care.

2. Out of clinic activities

2.1 Diabetes camp

The missions of the diabetes camp are to teach self-management skills to children and adolescents with diabetes and to allow them to meet other patients with a view to sharing their experiences. The Diabetes Education Program, Faculty of Medicine Siriraj Hospital has organized a diabetes camp once every other year since 1990. Each camp lasts 5 days covering both educational and social programs, and consists of around 60-70 patients. Because of the camp's friendly atmosphere, relationships can be easily developed among patients and with medical staffs. The camp gives medical staffs opportunities to understand patients' thoughts and behaviors, identify problems related to diabetes management, and adopt strategies to deal with them. It has also proven to be an invaluable experience for patients to learn diabetes self-management skills and form friendships with others.

2.2. Diabetes group support

Most teenage patients appreciate opportunities to socialize with their peers to discuss diabetes related issues and develop their own support structures. Teenagers are often more receptive to experiences and advice given by their peers than those provided by medical staffs. To this end, a Thai Diabetic Child and Adolescent Support Club was formed by parents and patients with diabetes. The website of the organization can be located at WWW.thaidiabetes.com.

3. Transfer of care to adult diabetes clinic

In most cases, adolescents with diabetes do not want to be transferred to adult diabetes care because they have developed close relationships with pediatric team members. Transition to adult care providers should be planned ahead and its steps discussed between the patient, family, pediatric diabetes team, and adult care providers.

It is important for health care providers to understand nature and needs of adolescents with diabetes and to provide them with sufficient support. The ultimate goal is to incorporate each adolescent patient to be a responsible, fully functioning member of his or her own health care team.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 20

From Model of Diabetes Center to the Prevention of Diabetes at the Grass Roots

Professor Thep Himathongkam, MD, FACP, FACE

Theptarin Hospital, Bangkok, Thailand

"Theptarin Diabetes and Endocrine Center" was founded in 1985 to create a diabetes care team model for Thailand. The Center's ultimate goal is, however, to raise the standard of diabetes care, which now focuses on prevention throughout Thailand. Theptarin has taken three steps to realize this vision: build a model, create awareness, and act as a philanthropic organization.

Step 1 – Create a Diabetes Team Model

In 1985, a modern diabetes team did not exist and was unheard of in Thailand. In order for it to be replicated throughout the country, a Thai model had first to be created.

The first obstacle in building a model was the availability of personnel. Then, no institution produced the required team members, e.g. dietitians or diabetes educators. Staffs at Theptarin were introduced to the new professions and those interested received in-house apprenticeship training.

The second obstacle was lack of demand for diabetes education. Patients did not see the value of prevention, and viewed consulting dietitians or diabetes educators as being a waste of time and money; they only wanted to see physicians, to be cured. Only after 6 years of forced education did Theptarin patients start to recognize dietitians and diabetes educators as professionals and see the importance of education, enabling organized, formal diabetes classes to start.

Today, the Theptarin Diabetes Team has grown in number, through continuous in-house trainings. Consulting and educational services for patients have expanded and become much more systematic. Advanced techniques and services, e.g., distal bypass surgery and foot clinic, have been added. Several team alumni have moved on and made great contributions to diabetes service at other health care facilities. The team is now the model to be adapted for replication throughout Thailand and the region.

Step 2 – Create Awareness

Creating awareness of new diabetes team approach must be done among three target groups: medical personnel, patients, and the public at large.

The Theptarin team constantly raises the awareness of diabetes team approach among all audiences. Among most frequently used methods is traditional classroom lectures and discussions to medical personnel and students. The team also host numerous student interns in various fields especially nutrition.

The most effective strategy with sustainable results is to form an entity to bring personnel who work with diabetes patients together. Thus, with the Theptarin team as the driving engine, the Thai Society of Diabetes Educators was established in 1998.

The concept of diabetes team approach must be introduced to patients to create demand for service. After demand for service has become apparent, medical personnel will demand to acquire training. At Theptarin, all new diabetes patients are routed to see diabetes educators prior to seeing physicians. Physician's referral is the key to building up credibility for diabetes educators and dietitians; thus, physicians work in teams with the latter two professions, referring patients to them. Theptarin also uses enjoyable activities, eg., a diabetes camp or a diabetes club, to locate interested persons. These groups can be our best means of promoting the diabetes team approach among their peers.

Also, Theptarin sends messages about diabetes care team and prevention to the public through mass media and through large systems, such as government agencies. Theptarin has also pulled resources from pharmaceutical companies for publishing advertorials warning the public about diabetes risks and introduc-

ing diabetes educators and dietitians as professions assisting physicians in helping patients control diabetes. Diabetes is now one of the government's top agenda items. Theptarin is one of the government's main resources for knowledge, with Theptarin staff often giving talks to audiences of different levels, e.g., government employees, teachers, or village leaders.

Step 3 – Act as a Philanthropic Organization

To raise the standard of diabetes care throughout Thailand and move into prevention at the grass roots, key sectors, i.e., universities, professional associations, private enterprises, and the government, must coordinate and share resources and expertise. Theptarin, as a private institution, had stepped in and taken this coordinating role. Examples of philanthropic activities Theptarin has accomplished include coordinating with Department of Allied Science at Chulalongkorn University to draw up Thailand's first curriculum for bachelor degree in dietetics, or coordinating with the Ministry of Public Health in organizing diabetes training for medical personnel in primary care units across Thailand.

Currently, the diabetes team approach is well-known and widely-accepted as being best practice in Thailand. The government recognizes the need to and has steered its attention towards treatment and prevention of chronic care diseases. The most apparent holes that Thailand must close are lack of up-to-date knowledge for preventing and controlling chronic diseases, and the production of diabetes educators and dietitians to support the building of care teams.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 21

Differential Gene Expression During Adipogenesis to Understanding Pathogenetic Mechanism of Insulin Resistance and Metabolic Syndrome

Lee-Ming Chuang, MD, PhD

Department of Internal Medicine & Graduate Institute of Clinical Medicine, National Taiwan University Medical College, Taipei, Taiwan

Differentiation of 3T3-L1 preadipocytes to adipocytes is a well-characterized cellular model for studying molecular mechanism of adipogenesis and insulin sensitivity. With mRNA differential display, we have isolated 50~60 genes that were confirmed with Northern blot analyses to show a different level of expression during this process. About one third of these genes were regulated by rosiglitazone. Among the genes that we have cloned, some of them, such as adipoQ and HMG1c, have been demonstrated to be important in the pathogenesis of obesity and type 2 diabetes by other groups. Here we demonstrated some of the genes of transcriptional factors, signaling molecules, adipocytokines and enzymes that might regulate insulin sensitivity and relate to obesity and type 2 diabetes. Validation of these candidate genes were done by clinical genetic studies, by correlating tissue expression levels and/or circulating protein concentrations, and by functional studies in the cellular systems.

We confirmed that some of the genes were involved in the generation of insulin resistance and clinical diseases. Of the transcriptional factors, molecular variant of the PPAR??is shown to associate with body build, insulin sensitivity and the risk to type 2 diabetes. Two of the target genes of PPAR?, i.e. APM1/ACDC/ADIPOQ and SORBS1, have been demonstrated to involve insulin sensitivity and associate with human obesity, type 2 diabetes and coronary artery disease. More importantly, we demonstrated a genetic interaction between the PPAR?? and APM1/ACDC/ADIPOQ on determination of insulin sensitivity in our population. In addition, we have also demonstrated the correlation of adipose tissue expression of these genes and circulating level of adiponectin with various metabolic phenotypes of insulin resistance syndrome. Besides, using SNPs of all the candidate genes that we identified in the differential display for adipogenesis, we further found that HMGA2 was significantly associated with type 2 diabetes in our population. Finally, we found a novel gene that might regulate prostaglandin metabolism and PPAR? activity.

We believe that pathway-based differential display is a feasible approach to isolate important genes in controlling body weight and insulin sensitivity. Future development for characterizing of these target(s) might be benefited by a combined approach with molecular genetics, animal models construction, protein structure analyses and ligand identification, which might result in potential drug design for treatment of the subjects with metabolic syndrome in the future.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 22

Mitochondrial Gene and Diabetes in China

Jianping Weng, MD

Professor of Internal Medicine, The First Affiliated Hospital of Sun Yat-Sen University
Vice-president of Chinese Diabetes Society
President, Guangdong Diabetes Research Center, Guangzhou, China 510080

Mitochondrial and Diabetes

Diabetes is regarded as disturbance of fuel metabolism. The mitochondrion is the intracellular powerhouse. Mitochondrial dysfunction will affect the energy supply leading to a disease state. The relationship between the mitochondrial function and the pathophysiology of diabetes became the burning question. Mitochondrial defects play a critical role in two prominent features of diabetes: insulin resistance and pancreatic B cell dysfunction. Defects in mitochondrial fatty acid oxidation leads to increases in intracellular fatty acid metabolites that disrupt insulin signaling in muscle and liver. On the other hand, normal oxidative mitochondrial metabolism which generates ATP is required for glucose sensing in islet B cell. Mitochondrial dysfunction makes B cells do not sense glucose properly and therefore do not release appropriate amounts of insulin. After frank diabetes occurs, the excess ROS generated in mitochondria by hyperglycemia and higher concentration of FFA accelerated B cell apoptosis.

Mitochondrial gene and Diabetes

Mitochondria are present in most eukaryotic cells, varying in number from hundreds to thousands. Not only its function but also its number and distribution in the cells altered in diabetes. All of these are influenced by genetics and environmental factors. Interestingly, mitochondria had there own DNA (mtDNA) which is independent of nuclear DNA. Only a few mitochondrial components are encoded by mtDNA, most of the mt-proteins are nuclear DNA encoded. Remarkably, the majority of the known mutations leading to a mitochondrial disease have been identified in mtDNA rather than in nuclear DNA.

The human mtDNA is a 16,569-bp-long, encodes for 13 proteins involved with oxidative phosphorylation as well as 22 tRNAs and 2 rRNAs involved in synthesis of the mitochondrial complexes. The mtDNA is transcribed and translated within the mitochondrion. It is maternally inherited because of the non-persistence of sperm mtDNA in the zygote after fertilization. The mitochondrial genome exists in multiple copies in every cell. It is highly susceptible to mutation as, in contrast to nuclear DNA, mtDNA consists only of coding sequences and its repair mechanisms are poor. So it is particularly sensitive to oxidative stress. Moreover, it is juxtaposed to the respiratory chain, which generates mutagenic oxygen derivatives. Consequentlyÿthe mtDNAs have a high mutation rate, 10"20 times faster than nDNA. The mutated mtDNA co-exit in the cell with wild mtDNA and will accumulated for its replication is faster than the wild type. If the mutation reached the upper threshold level beyond which the mitochondrial population collapse with a concomitant decrease in ATP. This decrease in ATP results in the phenotypic expression of disease. The mutation and wild type DNA co-existed in the cell were called heteroplasmy.

The mitochondrial genome has been implicated in the pathogenesis of diabetes mellitus by several mechanisms: point mutation in coding region; common polymorphism in regulatory region; and reduced amount of mtDNA content.

The most common mitochondrial diabetes is the A3243G mutation which interferes with the rate of charging of the tRNA(Leu, UUR). This mutation leads to mitochondria with reduced function as reflected by a 70% reduction in oxygen consumption in mitochondria carrying high heteroplasm levels for the A3243G mutation. Many other point mutations, mostly in tRNA genes, have been identified to represent a risk factor for diabetes, Their frequency is much lower compared to the A3243G mutation.

Approximately 85% of carriers of the A3243G mutation will develop diabetes before the age of 70. The mutation is strictly maternally transmitted and mostly all children from an affected mother will become

carriers of the mutation. The first described large pedigree with diabetes and impaired hearing co-inherited in association with an A3243G mutation in mitochondrial DNA in 1992. The syndrome

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 23

Epidemiology of Pad in Asian Diabetes

Tong-Yuan Tai, Yi-Der Jiang

National Health Research Institutes and National Taiwan University Hospital, Zhunan and Taipei,Taiwan

Diabetic subjects report an increase in the number of patients developing PAD many fold greater than their non-diabetic subjects. A WHO (World Health Organization) international study on people with diabetes aged 35-54 years identified Tokyo and Hong Kong as the cities with the lowest PAD prevalence in Asia. In the Edinburgh Artery Study, PAD (ABI < 0.9) prevalence in diabetic patients reaches 20.6%, nearly 4 times higher than the one found in Hong Kong Study. PAD was a frequent risk factor of foot lesions in Germany (48%), but it was far less common in India (13%) and Tanzania (12%).

Diabcare-Asia 2001 was conducted at the primary healthcare institutions of 9 Asian countries, including China, Indonesia, Korea, Malaysia, Philippines, Singapore, Taiwan, Thailand and Vietnam. The prevalence rates of absent foot pulse and acute ulcer/gangrene were respectively 4.51% and 0.84% for type 1 diabetes, and 2.83% and 1.28% for type 2 diabetes. Diabcare-Asia 2003 launched a similar survey at the secondary/tertiary care levels in 9 countries. The prevalence rates of absent foot pulse and acute ulcer/gangrene read respectively 2.30% and 1.24% for type 1 diabetes, and 3.29% and 1.35% for type 2 diabetes.

PAD SEARCH was a multinational cross-sectional study performed in 2003 in China, Hong Kong, Indonesia, Korea, Philippines, Taiwan and Thailand to investigate the prevalence of PAD in diabetic patients with the risk factors (aged 50 years and older, with one or more risk factors including smoking, hypertension and dyslipidemia) and to investigate the PAD risk factors. The diagnostic criterion for PAD was ABI less than 0.9. The highest PAD prevalence was observed in Hong Kong with 31.5% and the lowest in Korea with 11%. Thailand, on the other hand, showed the lowest odds ratio for PAD and Indonesia the highest one after smoking status, lipid profiles, duration of diabetes-hypertension, sex, age, BMI, previous history of PAD, cerebrovascular disease, ischemic heart disease and systolic blood pressure were adjusted. In this study, the PAD rate of high risk Taiwanese diabetics was 17.5%, which was 3 times higher than that of type 2 diabetics aged 40 or over, who were followed up at the diabetic clinic of the National Taiwan University Hospital (NTUH).

Up to December 2002, baseline screening for PAD had been performed in 3632 type 2 diabetics aged 40 or over at NTUH. Up to June 2005, 1574 patients of the above cohort had received at least one follow-up screening for PAD. The prevalence of PAD (ABI < 0.9) was 5.95% and the incidence rate 1.14%. ABI < 0.9 was an independent risk factor for all-cause and cardiovascular mortality in subjects with type 2 diabetes. Old age, smoking, poor glycemic control, poor blood pressure control, and dyslipidemia were significant risk factors for PAD prevalence, incidence and mortality. Hypoadiponectinemia (<3.75mg/ml) was associated with new PAD onset for those aged over 60. Incident cases, mild PAD (ABI 0.7-0.9 vs. <0.7), and better blood pressure control, higher education level, and higher BMI were salutary factors for PAD prevention or recovery. Among the modifiable risk factors, glycemic, lipid and blood pressure control are important to prevent new PAD. Moreover, once a low ABI is detected, aggressive treatment should be instituted because one third or more patients might have a chance to return to normal ABI before they progress to a severer degree of PAD with ABI < 0.7.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 24

A Human Replacement Tissue (Dermagraft), in Treatment of Diabetes Foot Ulcer with Peripheral Arterial Disease, Experience in Thai Patients at Theptarin General Hospital.

Julee Jatuvorapat, MD, Thep Himathongkum, MD

Theptarin General Hospital, Bangkok, Thailand

Normal healing of skin wounds consists of 4 phases that are regulated by the integrated action of chemokines, cytokine, growth factors, and proteases. The failure of some acute wound to complete these phases of healing lead to molecular imbalances of key regulator molecules and consequently wound becomes chronic. The molecular environment of chronic wounds characteristically contains elevated levels of inflammatory cytokine, and proteases, low level of mitogenic activity, and cell that are often senescent and response poorly to growth factors. The advancement of tissue-engineering has made possible the manufacturing of living human dermal fibroblast cultured in vitro onto a bioabsorbable mesh to produce a living, metabolically active tissue. This tissue delivers normal human dermal matrix proteins, cytokines and growth factors to the wound bed (Dermagraft, Advanced Tissue Sciences, Inc.), fourteen cases of diabetic foot ulcers whom had PAD with non healing wounds for more than 4 weeks were enrolled in the study. Age range from 64 to 88 years old, 4 males and 10 females. Ulcer duration range from 4 to 48 weeks, wound site were at lower leg to tip of toes (2 lower leg wounds, 2 planta wounds at heel, 4 dorsal foot wounds and 6 toe wounds), size were 1 to 15 cm2, All wounds need HBOT to promote granulation before implanted Dermagraft. Eleven cases use HBOT alone. Two cases were revascularization for 1 year prior. One case was received angioplasty 1 month before HBOT and dermagraft. Wound bed was prepared before implanted of Dermagraft by keeping wounds moist, clean and free of infection. The complete wound closure were observed at 4 –8 weeks with healing rate of 0.122 cm2/day. In conclusion Dermagraft improved wound healing and shorten hospital stay. This may be important factor to prevent adverse events associated with prolonged bed rest in elderly patients and prevent amputation.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 25

Surgical Management of Charcot Foot Deformity

Daniel K Lee

Director, Foot and Ankle Service, Department of Orthopaedic Surgery,
University of California, San Diego, USA

Surgical reconstruction of the diabetic Charcot foot and ankle presents significant challenges as deformity, instability, and ulceration are present in a high percentage of patients who have Charcot arthropathy. Certainly not all patients with Charcot deformities require reconstruction. Traditionally, bracing devices, orthoses, shoe modifications and accommodations are effective conservative treatments in providing support and in preventing further deformity. In the presence of wounds, the traditional means of treatment consist of debridement, antibiotics, and immobilization with limited weight bearing. Regardless of which treatment choice is undertaken, the goals of Charcot treatment still remain the same: correction of deformity as well as elimination of infection, with production of a stable, plantigrade foot. Achieving these goals should help prevent and/or control ulceration and infection, thereby avoiding amputation as the most devastating complication of the diabetic Charcot foot and ankle.

According to the American Diabetes Association 2002 report, more than 60% of nontraumatic lower-limb amputations in the United States occur among people with diabetes. From 2000 to 2001, about 82,000 nontraumatic lower-limb amputations were performed each year among people with diabetes. Charcot foot occurs most often in people with diabetes mellitus, and about 60–70% of people with diabetes develop peripheral nerve damage that can lead to Charcot foot. In most cases, onset occurs after the age of 50 and after the patient has had diabetes for 15 to 20 years. The cost of diabetes in the United States is over $130 billion. The prevalence of total diabetes in the United States, all ages, is 18.2 million people - 6.3% of the population - have diabetes, with 1.3 million people aged 20 years or older of new cases diagnosed per year.

When conservative measures fail and surgery is indicated for Charcot foot and ankle deformities. The main indications are (1) unbraceable deformity; (2) recurrent ulceration secondary to deformity, instability, or both; and (3) Charcot arthropathy with pain that is unresponsive to conservative measures. Reconstruction of the Charcot foot is targeted towards the rebuilding of the normal architecture of the foot and ankle by removing the high-pressure areas commonly in the medial and lateral column of the foot. The high rates of infection (25%) with internal fixation techniques and improved external fixation devices and techniques have proven external fixation as a viable alternative.

Ilizarov External fixation allows gradual and controlled correction of the Charcot foot and ankle deformities in a new way. These severe deformities can now be corrected with percutaneous wire techniques and circular rings with Ilizarov methods for gradual correction. These wire and ring fixators allow the surgeon to adjust or manipulate the Charcot foot during and after surgery and the patient can perform early weightbearing in 10 to 14 days; thus decreasing morbidity and non-compliancy. Furthermore, this method allows for (1) single or stage correction with acute or chronic ulcerations, (2) revisions for previous reconstructions, (3) acute treatment of Charcot-type fractures and dislocations, (4) arrest of progressive degeneration of Charcot's foot dislocations, (5) access for simultaneous advance wound healing treatments, and (6) plastic surgery techniques. Adjunctive surgical procedures can also be performed concomitantly with this method; such as, tendo Achilles lengthening, application of an external/internal bone stimulator, minimally-invasive screw and plate fixation.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 26

Critical Issues and Standards for Diabetes Education Globally

Marg McGill

Royal Prince Alfred Hospital, Sydney, Australia

In recognition of the importance of diabetes education the International Diabetes Federation (IDF) created the Consultative Section on Diabetes Education with the remit of improving the lives of people with diabetes through assisting health professionals, professional organisations and member organisations to provide high quality diabetes care worldwide. Reflecting the Section's philosophy, that education is the responsibility of all team members, its membership is multidisciplinary and globally representative.

A major activity has been to conduct leadership workshops in 5 of the 7 IDF Regions. From these workshops common critical issues impacting on diabetes education were identified. These included :

1. too many patients for the number of staff,

2. lack of integration of clinical care and education,

3. lack of health professional education programs and

4. lack of standards for the provision of high quality diabetes education.

5. hiding the diagnosis as society does not understand

Examining the first critical issue of too many patients for the number of trained staff, there is no question that the world is facing an epidemic of diabetes and its complications. Simply seeing more and more patients in the traditional manner, (which is largely based on acute care models), results in shorter and shorter consultations with little or no access to some specialist services, diabetes and nutrition education. Undoubtedly the future demand for care of people with diabetes will increase significantly. With the best intentions on the part of ministries of health, diabetes health professionals and organisations it is unlikely that sufficient resources will ever be available to cope. Thus different models of care such as the Chronic Care Model need to be considered. A key feature of this Model is care by truly integrated multidisciplinary teams. Unfortunately in many countries this concept is not well known and/or accepted. In this situation roles of the different health professionals are demarcated and there is no blurring of professional boundaries. Moreover, the nurse often does education at a different site from the dietitian and the doctor provides clinical care at yet another site. This separation is not only geographical in nature but also philosophical resulting in no standardized approach to the patients, no communication about the patient, no common plan developed with the patient for their target goals and outcomes. The Section together with WHO is working on a collaborative program to lead change in implementing better models that facilitate improved quality of care and prevention strategies at every level.

As a first step towards implementing a model of care that is underpinned by skilled multidisciplinary teams the Section developed the International Curriculum for Health Professional Education. The Curriculum has been widely disseminated and feedback on its usefulness has been very positive. Contributions to the writing of Curriculum reflect global needs. It is very comprehensive, is suitable for different health disciplines at different levels and can be adapted for short workshop or post- graduate diploma or masters programs. Despite this detail some regions have indicated that without content to underpin the curriculum they would still find it difficult to develop and deliver a health professional education program. To this end the Section is currently working on developing web-based and paper-based resources for the curriculum which will be available in early 2006.

Another significant activity has been the development of the International Standards for Diabetes Education which can assist individuals and institutions in the development of high quality diabetes education services. Standards cover areas of leadership, communication between team members and about patients, documentation of clinical care, ongoing professional education of all team members, physical require-

ments needed to provide a diabetes education service, evaluation of the program and research activities. These standards serve as a benchmark by which organisational and individual educators can be evaluated.

There is no doubt that in light of the growing epidemic of diabetes we cannot continue to provide care in the "old way". New models of care that integrate team members, and integrate clinical care and education are some solutions to this important issue. Ultimately we hope that in the future each country of the world will have a Centre of Excellence for Diabetes adapted to the local culture, professional organisation/s for all team members, high quality standards for care and education, programs for certifying diabetes educators and to assist in the provision of clinical care the acceptance of the concept of nurse practitioner/clinician.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 27

The Joys and Tears of Establishing a Professional Organization

Anne-Marie Felton (Chairman FEND)

Federation of European Nurses in Diabetes

The Federation of European Nurses in Diabetes (FEND) was established in December 1995. It is a pan European, non-governmental and not-for-profit organisation. It is managed by an Executive committee on a voluntary basis without remuneration. Membership is exclusive to nurses working in the specialty of diabetes (clinical practice, research, education) at primary and secondary specialist level.

Mission:

- *To promote the delivery of evidence-based care for people with diabetes throughout Europe.*

Aims:

- *To develop and promote the professional role of the diabetes nurse in Europe.*
- *To influence European health care policy relevant to diabetes care and research.*
- *To promote acceptable standards and equity of care for people with diabetes throughout Europe.*
- *To co-operate and collaborate with national and international health care organisations.*

FEND has established a unique voice for nurses working in the field of diabetes and exerts a positive political influence at European Union level in relation to policy development and implementation in relation to diabetes care, research, education and service delivery. This presentation will share the process of development and evolution of FEND since it's establishment and will indicate it's future direction over the next decade.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 28

Role of Nurses in Diabetic Control: Is It Visible?

Pornsri Sriussadaporn

Faculty of Nursing, Mahidol University, Bangkok 10700, Thailand

The prevalence of diabetes mellitus is increasing throughout the world including Thailand. At present, diabetes mellitus is one of the ten major chronic health problems that cause Thai people to go to hospitals. Although most of diabetic patients have received diabetes education from health care personnel, poor glycemic control is still present in a large number of patients. It was found that only 3 percent of patients had good glycemic control. This situation causes an impact on health care delivery since it is the responsibility of nurses who work as health care personnel and also diabetes-care teams to solve this problem. This presentation is therefore aimed to illustrate the role of nurses in diabetic control that is described as diabetes nursing. Diabetes nursing comprised of nursing care service for diabetic patients and people who are at risk and nursing interventions to educate and encourage diabetic patients and their families to perform self-management to achieve good diabetic control with well-being lifestyle and without complications. The roles of nurses in diabetes nursing include:

- Promote healthy lifestyle for prevention of disease
- Screen people who are at risk of having diabetes for early diagnosis and appropriate treatment
- Plan and implement cares for the hospitalized patients
- Collaborate with other multidisciplinary teams to provide special and appropriate care
- Provide continuing care to enhance self-care management in diabetic patients and their families
- Evaluate the effectiveness of glycemic control
- Develop and improve quality of diabetes nursing by using and conducting nursing research.

Diabetes nursing is available at all levels of health care service settings such as home care (HC), primary care unit (PCU), secondary and tertiary care hospital, private clinics and hospitals. There are 3 groups of nurses who are responsible for providing care for the diabetic patients: 1) general nurses (GN), 2) diabetes nurse (DN), and 3) advanced practice nurses (APN) in diabetes.

General nurses is classified in to 3 levels according to the duration of nursing study program including registered nurses (4-year program), technical nurses (2-year program), and practical nurses (1-year training program). Most of the health care services in diabetes nursing practice are provided by general nurses.

Diabetes nurses are general nurses who were trained through the diabetes educator program. Most of the diabetes nurses work in diabetic clinics both in PCU and OPD. Some of the diabetes nurses were assigned to be case managers or primary nurses for hospitalized diabetes patients.

Advanced practice nurses are the graduated nurse certified by The Thai Nurses Council. They work as an advanced clinician, a practitioner, a manager, an educator, an administrator, a leader, a consultant, a collaborator, and a researcher. The advanced practice nurses in diabetes have professional competencies in managing diabetes patients with complex health problems, providing social and psychological support and education on how to cope with diabetes. Most of the APN in diabetes practice in university and tertiary care hospitals.

In conclusion, the most important role of nurses in managing diabetic control is to enable diabetic patients and their families to cope with diabetes and perform good self-diabetes management. To achieve this role, it is necessary for nurses to obtain good and advanced knowledge about diabetes. The government through the Ministry of Public Health and the Nurses Council should cooperate to initiate not only a National Standard of Diabetes Nurse Educator Program for nurses who responsible for giving health care service to diabetes patients in all setting, but also establish a standard diabetes educators training program for APN especially who work in PCU where there are very few diabetologists on duty.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 29

Significance of Islet Autoantibodies in Type 2 Diabetes

Eiji Kawasaki*, Katsumi Eguchi**

*Department of Metabolism/ Diabetes and Clinical Nutrition, Nagasaki University Hospital of Medicine and Dentistry, Nagasaki, Japan
**The First Department of Internal Medicine, Graduate School of Biomedical Sciences, Nagasaki University, Nagasaki, Japan

Type 2 diabetes is characterized by combinations of decreased insulin secretion and decreased insulin sensitivity (insulin resistance), and the mass of pancreatic beta cells and their function are preserved to some extent, and insulin injection is seldom needed to sustain life. Typically, type 2 diabetes develops after middle age without hyperglycemic symptoms, and majority of patients are obese or have been obese in the past. Furthermore, anti-islet autoimmunity is not fundamentally involved in its pathogenesis. However, there is a subgroup of diabetic patients with islet autoantibodies (autoantibodies to GAD, IA-2, and insulin) who are diagnosed as having type 2 diabetes at disease onset. These patients are referred as "latent autoimmune diabetes in adults (LADA)" or "GADAb-positive type 2 diabetes", which is regarded as a slow-onset from of type 1 diabetes.

*There are several longitudinal nation-wide studies on natural history of GADAb-positive diabetic patients in Japan. The prevalence of GADAb in adult-onset Japanese patients with type 2 diabetes is 3 - 4 %, and is higher in the patients with shorter duration of diabetes (~ 5 years) compared to patients with longer duration of diabetes. Furthermore, the frequency of GADAb in insulin-treated and/or insulin deficient (ID) patients is higher than that in patients treated by diet and/or OHA. The GADAb-positive ID patients are characterized by young age at onset of diabetes, low BMI, low maximum BMI, and high HbA1c levels. The prevalence of IA-2Ab and anti-thyroid autoantibodies in the GADAb-positive ID patients are higher than those in the GADAb-positive non-ID patients. In the genetic studies, GADAb-positive ID patients are associated with type 1 diabetes high-risk HLA such as DRB1*0405-DQB1*0401 or *0901-*0303, whereas only DRB1*0405-DQB1*0401 is associated with GADAb-positive non-ID patients. Furthermore, GADAb-positive patients with protective HLA, DRB1*1501-DQB1*0602 or *1502-*0601, are characterized by older age at onset of diabetes, higher BMI, resistance to the ID state, low titer of GADAb. Type 2 diabetic patients with GADAb often progress toward an ID state within several years after diagnosis. Although high levels of GADAb have a high predictive value for future insulin requirement, there are a certain number of patients with high titer of GADAb who do not progressed to insulin dependency for many years and the predictive value of GADAb positivity for future insulin requirement is estimated about 67% by Baye's theory. Thus accurate predictive strategies of future insulin deficiency in type 2 diabetic patients with GADAb using autoantibody epitope analysis, genetic determination, or T cell assay are required for the effective immune intervention.*

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 30

Fulminant Autoantibody-Negative Type 1 Diabetes in Japan – Is It a Distinct Entity?

Toshiaki Hanafusa, Akihisa Imagawa

First Department of Internal Medicine, Osaka Medical College, Takatsuki, Japan

Type 1 diabetes mellitus results from a marked depletion of insulin-secreting pancreatic-cells. Type 1 diabetes is divided into two subtypes; i.e. autoimmune type 1 (type 1A) diabetes and idiopathic (type 1B) diabetes. Autoantibodies against various constituents of pancreatic ?eta-cells are hallmarks of type 1A diabetes mellitus. Although type 1A accounts for a major part of type 1 diabetes, a considerable part of patients with type 1 diabetes are type 1B. Our previous clinical and immunopathological research indicated the presence of a few type 1B patients whose diabetes develops and progresses very rapidly into a total deficiency of insulin-secreting capacity.

To establish this type of diabetes as a new and distinct clinical entity, we first performed a hospital-based research on such patients. Consequently, we could establish "fulminant type 1 diabetes mellitus" as a novel subtype of type 1 diabetes. This type of diabetes shows many unique characteristics including 1) remarkably abrupt onset of the disease, 2) very short duration of diabetic symptoms, e.g. polyuria, thirst, and body weight loss, 3) ketoacidosis at diagnosis, 4) negative status of islet-related autoantibodies, such as GADAb, 5) virtually no C-peptide secretion, 6) elevated serum pancreatic enzyme levels, and 7) presence of T lymphocytes in the exocrine pancreatic tissue (Imagawa A et al., N Engl J Med 2000).

Following this first description, the Japan Diabetes Society organized a committee for research on fulminant type 1 diabetes and performed a multi-center study and a nation-wide survey to clarify the more detailed clinical and immunologic characteristics of fulminant type 1 diabetes (Imagawa A et al., Diabetes Care, 2003). As a result, a total of 161 patients with fulminant type 1 diabetes mellitus were collected from all over Japan. Fulminant type 1 diabetes accounted for nearly 20% of acute-onset type 1 diabetes mellitus with ketoacidosis. At onset, compared with 137 patients with autoimmune type 1A diabetes, patients with fulminant type 1 diabetes had significantly shorter duration of hyperglycemic symptoms, higher plasma glucose level, lower HbA1c level, lower serum C-peptide level, lower arterial pH, higher prevalence of preceding flu-like symptoms and epigastralgia, more frequent association with pregnancy, and higher prevalence of elevated serum pancreatic enzyme levels. GAD antibody was virtually negative. One patient with fulminant type 1 diabetes died and another patient suffered from cardiac and respiratory arrest at onset. None of type 1A patients suffered from such episodes.

Based on these results, we would like to conclude that fulminant type 1 diabetes is a distinct subtype within type 1 diabetes. Metabolic derangement is more severe in this subtype than in type 1A diabetes. Disregard or oversight of this rapidly-progressing diabetes would result in a death of the patient, and thus all clinicians must keep this fulminant type 1 diabetes in mind.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 31

The Challenges of Establishing Foot Clinics and Foot Education Around the World

AVM Foster

Honorary Consultant Podiatrist, The Diabetic Foot Clinic, King's College Hospital, London SE5 9RS, United Kingdom

The diabetic foot is a major public health problem of our time, and people with diabetes are at greatly increased risk of undergoing major amputation of a leg. The increasing global incidence of diabetes brings a corresponding increase in diabetic complications including those affecting the foot. Currently there are up to one million amputations of a leg per year in people with diabetes. Throughout the world up to 70% of leg amputations happen to people with diabetes.

In order to address the problems of the diabetic foot, various models of diabetic foot care programmes have been set up throughout the world over the past two decades. Some of these regional and national foot programmes have been successful and may provide useful models for improving diabetic foot care in other areas.

Much has been learned about the aetiopathology of the diabetic foot and the special needs of diabetic foot patients with neuropathy and neuroischaemia, and it seems likely that around 85% of diabetic amputations are preventable.

To achieve reduction of amputations it will be necessary to establish more diabetic foot clinics around the world and to offer diabetic patients footcare education programmes, with special emphasis on avoiding local risk factors. However, in many parts of the world there is ignorance about risk factors for foot ulceration, and failure to provide the best preventive foot care and optimal treatment of diabetic foot problems. There is great need for a concerted effort to increase awareness of the diabetic foot among people with diabetes and health care professionals, and to provide diabetic foot clinics where preventive foot care and treatment of existing problems can be obtained.

Every thirty seconds a lower limb is lost to diabetes somewhere in the world. The majority of these amputations begin with a foot ulcer. Worldwide, the most important risk factors for foot ulceration are peripheral neuropathy, peripheral vascular disease, foot deformities, walking barefoot or wearing unsuitable shoes, plantar callus, trauma, and infection. Peripheral neuropathy can be prevented or reduced with optimal control of diabetes, but this is not easy to achieve. Peripheral vascular disease can be prevented or reduced by good control of hyperglycaemia, hypertension and hyperlipidaemia and smoking cessation, but again, it is not always possible to achieve this. However, even feet with severe neuropathy and/or ischaemia can be successfully managed. Foot deformities may be congenital or acquired and will be less likely to lead to foot ulceration if accommodated within suitable footwear. High pressure points leading to callus and ulceration can be debrided and there are numerous off-loading techniques, including insoles, crutches, casts and braces. Prophylactic surgery may have a role in treating some types of foot deformity. Provision of suitable footwear is essential for high-risk diabetic feet and some patients may need bespoke (tailor-made) shoes. Common causes of trauma, such as walking barefoot leading to burns and puncture wounds, can be addressed by teaching the patient how to look after his feet. Callus can be removed by sharp debridement and closure of wounds and ulcers can often be achieved by a combination of debridement and off-loading. If infection is detected early and treated with antibiotics then gangrene can often be prevented. Vascular techniques such as angioplasty and bypass can save limbs.

These techniques are best offered within the forum of a multi-disciplinary diabetic foot clinic. The work of the diabetic foot clinic is aimed at preventing many foot problems and offering early treatment and regular follow-up of foot problems. Treatments can be categorised under mechanical control, metabolic control, microbiological control, vascular control, wound control and educational control, all of which need to be addressed in the diabetic patient with foot ulcers.

The aim of education programmes is to impart awareness that they are at risk to diabetic patients and to persuade them to check their feet every day, report problems early, adhere to treatment programmes, and attend the clinic regularly for follow-up.

No one person is able to fill all the needs of the diabetic foot patient: it takes a team, and the most successful diabetic foot clinics have employed multidisciplinary teams who work together in joint clinics. Teams usually include physician, nurse, podiatrist (or other health care professional trained in podiatry techniques), orthotist/shoe maker, and surgeon. The best-known foot clinics were created a step at a time, beginning with a basic model and gradually developing into centres of excellence. However, there are many barriers which need to be overcome when setting up diabetic foot clinics and educational programmes, including the following:

The diabetic foot may be regarded as a "dirty" disease, and health care professionals may be reluctant to care for smelly, ulcerated, or infected, feet.

Foot problems are common in leprosy (Hansen's disease) and some patients may be reluctant to attend a clinic for ulcerated feet for fear of being stigmatised.

Emotional conflicts are common. Patients are asked to rest their feet but can only do this at the expense of work and family commitments.

Patients may prefer to turn to traditional medicine and traditional remedies as an alternative to orthodox medicine and the diabetic foot clinic.

There may be no recognised profession of podiatry (only 19 countries are affiliated to the international federation of podology), so patients may find it hard to gain access to preventive footcare.

In developing countries where infectious disease is a great problem and per capita spending on health is very limited, it may be difficult to find funding to set up a diabetic foot clinic. Good diabetic foot care is cost effective but is often perceived as less important than, for example, treating malaria or water-bourn diseases. Furthermore, in areas where poverty is rife it may be impossible for the individual to pay for private health care in a diabetic foot clinic, and the cost of suitable preventive footwear may be a barrier to effective care.

Forming a cohesive team may be difficult in countries where the physician is perceived to be "the boss" and is unaccustomed to working closely with other health care professionals and sharing responsibility with them. Forming an enthusiastic team with joint aims and objectives may therefore be difficult. In some health care systems, doctors lose revenue when they refer patients to the hospital or to another health care professional. Avoiding inter-departmental rivalries and fighting within the foot clinic team may be difficult.

In some developing countries there may be a desire on the part of medical staff to provide expensive high-tech equipment and treatments instead of offering very basic, inexpensive care which could significantly reduce diabetic foot problems.

The scale of the problem of the diabetic foot is enormous. In India alone there are 33 million people with diabetes. However, work by the International Working Group on the diabetic foot, the World Diabetes Foundation, and national organisations is gradually improving the outlook for diabetic foot patients. The International Working Group has published guidelines and consensus documents. A "Step by Step" programme, funded by the World Diabetes Foundation and piloted in India and Tanzania, has endeavoured to overcome some of the barriers to setting up effective diabetic foot services. Teams of doctors and healthcare professionals have been provided with practical training in basic techniques for preventing, assessing and healing foot ulcers, unique educational materials aimed at patients and healthcare professionals, surgical instruments and text books. The patients' educational materials do not depend upon the written word to impart their messages. At the end of the "Step by Step" programme the participants have the resources and equipment to set up their own diabetic foot clinics and spread the word about diabetic foot care to other workers. If successful, this programme could be rolled out to other developing countries and help to meet the challenges of setting up diabetic foot clinics and footcare education programmes.

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 32

Therapeutic Footwear

Somkiet Mahaudomporn

Raj Pracha Samasai Institute, Samutprakarn,Thailand
Theptarin General Hospital, Bangkok,Thailand

Introduction

Therapeutic Footwear is an importance tool in managing diabetic foot problem. Footwear should be worn by all patients who are at risk from ulceration, infection, gangrene and ultimately amputation. The appropriate designed footwear can have a positive effect on the clinical outcomes for these groups of patients. The specific features of the footwear will be described. Therapeutic footwear is effective in preventing and healing the ulcers but only if the patient is compliant, however our experiences have shown that patient dissatisfaction with footwear leads to non-compliance. It is known that one of the main reasons for patients, non-adherence to footwear use as a therapeutic intervention is that the foot wear is visually unattractive and heavy. However, patient experience of previous footwear and a good understanding of their foot problems may also play a role in the complex picture of what makes a good shoe.

General considerations

In the context of this information that footwear can be of benefit, but we have no record of what patients believe to good and bad shoes. All patients with Neuropathic feet should wear therapeutic footwear throughout their lifetime. This is true whether or not they have ulcers. So, the therapeutic footwear should include the following features;

A soft insole
A wide and tough under sole
A well-fitting and adjustable fastener
A big and wide toes box
Soft& smooth linings
Style acceptable to the customer
Cost
Weight of the shoe

Develop a footwear design criteria for patients with an at risk foot, based on available clinical experience and the opinions of patient.

Type of Therapeutic Footwear.

The practical points of prescription is based on the risk factor for ulcerations. The practitioner must take into consideration the patient's social, work and personal need. The University of Texas Foot Classification System was devised the patient from a high-risk category into the lowest possible category. Category 0 to 3 are risk factors for ulceration and the proper shoes should be. They are;

Category 0 : No pathology	*Possible shoe accommodations*
Category 1 :Neuropathy without deformity	*Sport shoes/Simple sandal*
Category 2 :Neuropathy with deformity	*Extra depth shoe accommodation*
Category 3 :History of pathology	
-History of ulceration[Scars are soft & mobile]	*Extra depth shoe accommodation with arch support*
-Foot is not deformed with numerous scars	*Extra depth shoe accommodation with Insole*
-History of charcot's joint	*Custom molded with rocker shoes and Insole*

Conclusion

Therapeutic footwear with specific features and design will be effective in management of risk foot in

diabetes. The design criteria can be use as a basis for future work investigating the clinical effectiveness of combinations of footwear in the patients with specific pathologies.

References:

1. *Armstrong DG, Lavery LA,Harkless LB: Treatment based classification system for assessment and care of diabetic feet.J am Podiatr Med Assoc 86:311-316,1996*
2. *Lavery LA, Armstrong DG, et al:Choosing the practical screening instrument to identify patients at risk for diabetic foot ulceration. Arch Intern Med158:157-162,1998.*
3. *Reiber GE, Smith DG, Wallace C et al.Effect of therapeutic footwearon foot reulcerationin patients with diabetes: a randomized control trial. JAMA 2002:187(19),2552-8.*
4. *Knowles EA, Boulton AJ. Do people with diabetes wear their prescribed footwear? Diabet Med 1996:13(12),1064-8*

Full text. e-Journal: *http://www.medassocthai.org/journal*

SS 33

Helping Patients Cope with Painful Neuropathy

Lea Sorensen

Diabetes Centre, Royal Prince Alfred Hospital and Discipline of Medicine, University of Sydney, Sydney, Australia

The insensate form of neuropathy is a common manifestation of diabetic foot disease. However, approximately one in ten patients suffer from painful symptoms which are often extremely debilitating and significantly affect quality of life. Curiously, pain in the feet may be present with no objective clinical signs of neuropathy and thus, the condition is all-too-often under-recognised and under-treated.

It was previously thought that painful symptoms were part of a continuum of neuropathy that eventually resulted in complete loss of sensation. However, current research supports the notion of insensate (large nerve fibre) and painful (small nerve fibre) neuropathy existing as two separate conditions, each requiring specific clinical and educational management.

This overview presents findings on the development of painful diabetic neuropathy, diagnosis, and a systematic approach to easing the pain for those living with this condition.

Full text. e-Journal: *http://www.medassocthai.org/journal*