EDITORIAL

ORIGINAL ARTICLES
• Kidney Disease in the Pregnant Woman with Diabetes
• The impact of pregnancy and diabetes on the eye
• Non Alcoholic Fatty Liver Disease
• Nutrition in Pregnancy
• Mood disorders during and after pregnancy

FEATURE ARTICLE
• Should They or Shouldn’t They?

CPD ACCREDITED DIABETES TRAINING
EDITOR’S comment

This wintertime issue of the Journal is essentially dedicated to pregnancy. We have included a diverse range of topics that should be of interest and use to most practitioners with an interest in diabetes. With the adoption of The International Association of the Diabetes and Pregnancy Study Groups (IADPSG) criteria for the diagnosis of gestational diabetes mellitus (GDM) by The Society for Endocrinology, Metabolism and Diabetes of South Africa (SEMDSA), the number of women diagnosed with this condition will increase. Does your clinic have the scope and facilities for such an increase?

Diagnosing GDM has profound implications for both the soon-to-be mother in the context of this and any future pregnancies, as well as for her offspring. Much research is looking at the notion of whether or not GDM might be one cog in the wheel of the alarming rate of increase in the incidence of type 2 diabetes. One certainty captured throughout all the contributions that follow is that timeous attainment of optimal glycaemic control is central to the successful reduction of adverse pregnancy outcomes. We include two clinically-orientated pieces covering the common microvascular manifestations complicating diabetes and pregnancy.

When managing these cases, we should also bear in mind that it is easy to focus only on the numbers. Mood disorders in the intra and post-partum periods, however mild, are also associated with adverse outcomes and it is refreshing that Rosemary Flynn offers us an insight into these disorders. Being honest in acknowledging that they are underdiagnosed is a good start for future vigilance. Diabetes in pregnancy is thus another ideal opportunity for all healthcare providers to work within a collaborative environment.

Finally, post delivery, the team should up efforts to endorse an informed and supportive ‘breast is best’ philosophy to assist as many moms as possible to consider breast feeding as a first option. Our unit has seen good value by including a lactation specialist within the wider team.

As customary at this time of the year, I would like to remind you of two important topical matters.

Firstly, with the winter season at our doorstep, we have included a short 2015-specific update on the public health issues surrounding seasonal influenza and the preventative value of vaccination for this troublesome virus.

Secondly, please remember the 17th CDE Postgraduate Forum in Diabetes Management, which will take place at the Emperors Palace and Convention Resort, 64 Jones Road, Kempton Park, Gauteng, from Friday 14 to Sunday 16 August 2015.

This CPD-Accredited event is the largest annual diabetes-specific meeting in Sub-Saharan Africa and provides the primary gathering point for clinicians from over 210 affiliated CDE Centres of Excellence nationwide, over 650 contracted general practitioners, other interested clinicians and representatives of the Pharmaceutical Industry and Medical Funders who are interested in the provision of quality practical diabetes care solutions. Registration is now open. Further details on the event, costs, the Programme and Venue accommodation are available on the CDE Website (www.cdecentre.co.za).

Dr Stan Landau
Editor
email: Stan@cdecentre.co.za
“Truthfully, being pregnant is changing me as a person. Each day is part of this amazing journey that has completely shifted the focus of my life and made me reevaluate my personal and professional goals.”

Holly Madison

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With the onset of pregnancy, a number of dramatic related physiological changes have resulting effects in most organ systems. The kidneys are often placed under tremendous pressure during gestation; so much so that new diseases can occur, or occult pathology can be unmasked.

This article will focus on kidney pathology during pregnancy and particularly on diabetes related problems.

**Normal Physiological Changes in Pregnancy**

In the early weeks of a normal pregnancy, the blood volume increases as a result of sodium and water retention. This is accompanied by a 30-50%, increase in cardiac output and a decrease in peripheral vascular resistance. The latter accounts for the fall in blood pressure normally observed at this time.

These changes continue into the second trimester, where their effects peak, and then stay relatively stable until delivery.

**Kidneys:** The kidney’s size can increase by as much as 30% in pregnancy as a result of the increase in renal vasculature and an increase in the interstitium. This is accompanied by an increase in the glomerular filtration rate (GFR), caused mainly by an increase in cardiac output, which increases renal blood flow. These changes are evident within one month of conception and can result in a measured increase in GFR by as much as 50% during the second trimester and represent marked hyperfiltration. Renal blood flow can increase by as much as 80% over the same period. These increases in blood flow are accompanied by a decrease in vascular resistance, as is found elsewhere in the body.

It is not surprising then, that this increase in the GFR results in a substantial fall in the serum creatinine, which is further lowered by haemodilution secondary to increased sodium and water retention. In fact, normal serum creatinine levels in pregnancy range from 35-79 μmol/l. Values above this reflect considerable kidney dysfunction.

The appearance of proteinuria during pregnancy (commonly occurring after 20 weeks gestation) can occur in patients with normal glomeruli. A number of causes are possible. The increased GFR results in increased...
protein delivery to the tubules; it may well be that the normal tubular reabsorption of protein is overwhelmed, resulting in its appearance in the urine. There also appears to be increased permeability of the glomeruli. Anything up to 300 mg of protein excretion per day in this context is considered to be normal.

Estimating GFR in pregnancy: The accurate assessment of GFR in pregnancy can be rather difficult for various reasons. However, it is probably more important to compare relative changes over time rather than rely on an absolute isolated value. Timed collections (traditionally over 24 hours) are often inaccurate in both pregnant and non-pregnant patients due to the inconvenience associated with emptying the bladder into a bottle over an entire day. This frequently results in over- and under-collections, making the true assessment of creatinine clearance (a surrogate for GFR) difficult. The situation may be aggravated in pregnancy due to urinary tract stasis associated with the changed hormonal milieu. This means that the collected urine may well have been formed prior to the timed period as it lay stagnant in the sluggish, dilated urinary tract.

The various formulas used in non-pregnant women (e.g. Cockcroft-Gault and Modification of Diet in Renal Disease [MDRD]) have not been as extensively studied in pregnant women. However, there is some indication that these formulas both underestimate ‘true GFR’ (as measured using the inulin clearance method as a gold standard). Many laboratories in South Africa quote the MDRD formula when reporting results and it must be pointed out that this formula is notoriously inaccurate when the GFR is > 60 ml/minute (usually the case in pregnant subjects). As mentioned above, the charting of changes in serum creatinine may well be more important than a single GFR evaluation.

Estimating proteinuria in pregnancy: The estimation of proteinuria has been greatly simplified over the past ten years with the use of the spot urine protein (or albumin): creatinine ratio. This, together with GFR calculations using serum creatinine, has meant that timed 24-hour urine collection is more or less a thing of the past.

It is, however, not clear how accurate spot urine collections are when it comes to pregnant women. Several studies have shown that the association with a 24-hour timed collection and a spot urine protein: creatinine ratio is poor. Nevertheless, as with the evaluation of GFR in pregnancy, it is perhaps the changes over time and not the absolute value that is important. For this reason, many nephrologists and obstetricians use spot urine samples during pregnancy as well.

It is important to monitor the changes in the patient who has pre-existing proteinuria as her pregnancy develops. It also becomes important to quantify new onset proteinuria after 20 weeks of gestation to diagnose pre-eclampsia. This is of course in conjunction with regular blood pressure measurements.

Ureters and bladder: Ultrasound studies of the urinary tract during pregnancy usually give the appearance of hydronephrosis or obstruction with ureteric and renal pelvic dilatation. This has a number of causes, including the high levels of progesterone, which causes ureteric dilatation and loss of peristalsis. Additionally, as the uterus enlarges, it tends to result in a degree of obstruction. The proximity of the iliac arteries and ovarian veins can also cause some obstruction of the ureters. These changes result in a dilated and sluggish urinary tract that can accommodate as much as 300 ml of urine. This increased volume can become infected, contributing to the increased incidence of pyelonephritis in pregnancy.

The bladder itself appears inflamed and swollen on cystoscopy, whilst progesterone also causes dilatation and increased bladder volume initially. However, with the increasing size of the uterus, bladder volumes tend to decrease during the course of the pregnancy. Increased ‘floppiness’ of the bladder can lead to incompetence of the vesico-ureteric valves. This can cause reflux and contribute to increased infection risk.

Pregnancy and Diabetes

Given the large increase in GFR (hyperfiltration) that occurs during pregnancy, it is not surprising that in many instances, pregnancy can aggravate underlying kidney pathology. In a recent prospective study, Imbasciati et al revealed an accelerated rate of GFR loss after delivery in women who’s GFR was <40 ml/min or whose proteinuria was > 1 g/day before falling pregnant. This
study was confined to women without diabetes, but there are indications that the degree of kidney dysfunction is important and not the underlying kidney pathology.

It is reassuring to note that in a few small studies, in patients with no evidence of kidney disease (i.e. normal GFR with normal albuminuria) prior to falling pregnant, no evidence was seen of an increased risk of developing diabetic nephropathy in the long term after single or even repeated pregnancies. However, it would seem that if a patient developed pre-eclampsia during the pregnancy, there was a far higher rate of developing diabetic nephropathy later on (41.9% versus 8.9%).

In most developed regions where the degree of diabetes care has improved, it is not uncommon to encounter women with diabetes in their reproductive years. Amongst these will be cohorts who have existent pre-pregnancy diabetic nephropathy. This latter group will have the highest rates of adverse maternal and foetal outcomes. For example, rates of preterm delivery can range from 29-91%, small for gestational age infants from 14-45% and pre-eclampsia from 27-69%.

The natural history of diabetic nephropathy is characterized by initial hyperfiltration, as evidenced by an increase in GFR, followed by the appearance of microalbuminuria (30-300 mg/day of urinary albumin excretion). Next, overt nephropathy follows as urinary albumin excretion rises above 300 mg/day with the appearance of hypertension and an inevitable decrease in GFR. The development of overt diabetic nephropathy takes roughly 5-10 years. The sequence outlined above is merely the typical pattern, but there are many variations in real practice. Although much progress has been made recently in terms of the prevention and retardation of diabetic nephropathy, the many evidence-based trials are often restricted to non-pregnant patients. Some of the treatment strategies are even contraindicated in the pregnant, for example the use of renin-angiotensin pathway inhibitors. Thus, most of the current literature in pregnant women with diabetes consists of case studies.

Although much progress has been made recently in terms of the prevention and retardation of diabetic nephropathy, the many evidence-based trials are often restricted to non-pregnant patients.

Unfortunately, this group carries an increased risk of pre-eclampsia (7-10% versus 2-5%) and also a slightly increased risk for the development of postpartum proteinuria. There is also an increased risk for the development of type 2 diabetes in these patients, even years beyond the delivery of the infant. Following delivery, the GFR usually reverts to normal.

Women with pre-existent diabetes and normal kidney function: In those women who have normal levels of proteinuria and normal GFR’s prior to falling pregnant, there is often a 3-4 fold increase in urinary albumin excretion, above a level of 300 mg/day, 20 weeks into gestation. It is not clear what the cause of the increased albumin excretion is, but it may well be that these women overwhelm normal tubular resorption. In spite of the dramatic rise in proteinuria in this group, it tends to reverse back to normal upon delivery.

In woman with preceding microalbuminuria only, the mean increase in excretion can be 10-fold and 20-30% of patients go on to develop nephrotic range (>3g/day) proteinuria. In spite of this exaggerated rise in proteinuria during the pregnancy, it still tends to return to normal levels postpartum.

Women with pre-existent diabetes with overt nephropathy and preserved GFR: In this group, where there is evidence of clinical disease preceding pregnancy, the GFR often does not increase as it does in normal non-diabetic patients. The serum creatinine tends to remain unchanged or decreases only to some extent in the initial two trimesters. Once again, the proteinuria can rise to nephrotic range and then revert to pre pregnancy levels upon delivery. It is unusual for the GFR to decrease in these patients.

Women with overt nephropathy and reduced GFR: these women may also develop a marked increase in urine protein excretion above nephrotic range. Although this often reverts back to baseline upon delivery, certain women develop deteriorating GFR during the pregnancy which does not always return to pre pregnancy levels.

It would seem that GFR levels below 60ml/min prior to pregnancy are predictive of a failure to show an increase in GFR during gestation. These patients are
also more likely to show a further decline in GFR post partum. However, it is still not entirely clear why some pregnant patients experience a complete return in renal function following delivery and others do not.

**Pre-eclampsia in Patients with Diabetes**

Often patients with diabetes can develop proteinuria and hypertension, as discussed above, without developing full-blown pre-eclampsia. This means that many diagnoses of pre-eclampsia in this group may be because of misclassification. In spite of this, the incidence of true pre-eclampsia rises from 2.5% in women without diabetes to 7-10% in those with GDM and 15-20% in those with pre-existing diabetes. In those with established diabetic nephropathy, the risk is as high as 27-69%. The reasons for the increasing levels of pre-eclampsia are unclear, although the disease does seem to have something to do with microvascular dysregulation. It may well be that the abnormalities involved with insulin resistance and abnormal glucose metabolism aggravate the vasculopathy associated with pre-eclampsia.

Recent studies have found an association with increasing HbA1c levels and the risk of pre-eclampsia. In those patients who develop gestational diabetes, appropriate treatment with nutritional therapy or insulin results in a decreased incidence of pre-eclampsia. Whilst some studies have found an association with higher urinary albumin excretion in the first trimester, others have not. There may also be abnormal placental attachment because of abnormal diabetic microvasculature that further contributes towards the development of pre-eclampsia.

As mentioned previously, the distinction between the proteinuria and hypertension of diabetic nephropathy versus pre-eclampsia is often difficult. Nevertheless, the distinction is an important one as the best treatment for pre-eclampsia is delivery. Recently a range of proteins which may be involved in the vasculopathy of this disease have been discovered and it is hoped that these may eventually be used to assist with diagnosis. Examples of these anti-angiogenic proteins are soluble forms like tyrosine kinase 1 and endoglin. Trials are underway assessing the diagnostic accuracy of these proteins when they are raised.

**Blood pressure management during pregnancy**

There is insufficient trial evidence to know what the best blood pressure levels are for patients with diabetes who fall pregnant and whether this changes with the degree of kidney involvement. Additionally, reductions in blood pressure may be beneficial to the mother, but detrimental to the developing foetus. Thus a balance may well be needed to ensure the wellbeing of both patients. Most authors would advise maintaining blood pressures somewhere between 120-140/80-90 mmHg.

Unfortunately, the agents used for blood pressure control in non pregnant women with diabetes, namely ACE inhibitors and angiotensin receptor blockers, are absolutely contraindicated in pregnancy. For this reason, when considering a pregnancy, timeous stopping and replacing these agents is mandated. A recent study also found that foetuses exposed to ACE inhibitors in the first trimester of pregnancy had a higher incidence of congenital malformations, typically cardiovascular, renal and neurological in nature.

This risk does not seem to be associated with other antihypertensive agents.

The ‘ideal’ choice of antihypertensive has yet to be proven in trials. It is often a case of trial and error, using a combination of methyldopa, calcium channel blockers and diuretics.

**Conclusion**

In developed countries, as many as 4% of pregnant women have either pre-existing or gestational diabetes. As discussed, many of these either have, or will develop kidney pathology. Trials on the treatment of this group are scarce and we must therefore be guided by expert opinion until further evidence is available.

It is hoped that ongoing studies into the increased risk of pre-eclampsia in women with diabetes reveal insights into the pathogenesis of this disease in all patients.
The ocular symptoms experienced during pregnancy are typically temporary and resolve during the postpartum period. These symptoms can either be associated with the incidence of new conditions or the aggravation of pre-existing conditions. The ocular manifestations of pregnancy can be categorised into the following three groups:

1. Physiologic changes
2. Pathologic (pregnancy-specific) conditions and
3. Modifications of pre-existing conditions.

**Physiologic Changes**
Physiologic changes which affect the cornea during pregnancy result in decreased corneal sensitivity as well as an increase in corneal thickness and curvature. These changes often cause variations in refraction and produce symptoms of fluctuating vision later in pregnancy. This refractive instability, along with pregnancy-induced dry eye, renders pregnancy a contraindication to refractive surgery and often causes contact lens intolerance.

Intraocular pressure in healthy eyes generally decreases during the third trimester and returns to normal postpartum. In ocular hypertensive subjects, pregnancy can cause an even greater decrease in intraocular pressure but this normally returns to pre-pregnancy levels postpartum.

**Pregnancy-Specific Eye Disease**

**Preeclampsia**
Symptoms of retinal vascular changes, which occur during preeclampsia, include decreased or dimming vision, photophobia (light sensitivity) or flashes of light, visual field defects and diplopia (double vision). If a pregnant woman experiences blurred vision accompanied by symptoms of abdominal pain and/or headache/s it is vital to rule out preeclampsia.

The ocular findings associated with preeclampsia are similar to those seen in hypertensive retinopathy. These changes include diffuse retinal oedema, haemorrhages, exudates and cotton wool spots.

**Central Serous Chorioretinopathy (CSCR)**
A circumscribed neurosensory retinal detachment can occur in the macula as a result of accumulation of sub-retinal fluid. CSCR occurs predominantly in men but does
have a strong association with late pregnancy in women. The presenting complaint is primarily moderately reduced vision and metamorphopsia (distortion of the image). Elevated levels of endogenous cortisol, which lead to increased permeability through the blood-retinal barrier, tend to reduce within a few months after delivery. Although visual acuity returns to normal, metamorphopsia and central visual field changes may persist.

**Vascular Occlusive Disorders**

Vascular occlusive disorders are associated with preeclampsia, pancreatitis, amniotic fluid emboli and hypercoagulability. The visual prognosis in these cases is usually guarded.

**Pre-existing Eye Disease**

**Diabetic Retinopathy**

Studies have shown that pregnancy is indeed a risk factor for worsening existing diabetic retinopathy. However, women who acquire gestational diabetes, and who have not had pre-existing diabetes, are not at risk for acquiring diabetic retinopathy.

**The Retina**

The retina is comprised of 9 layers. These layers are grouped to form the inner retina and the outer retina.

**Inner Retina:**

- **Internal Limiting Membrane (ILM).** This layer gives the retina its lustrous appearance and acts as the boundary between the retina and the vitreous body.
- **Nerve Fibre Layer (NFL).** This contains large blood vessels.
- **Ganglion Cell Layer (GCL).**
- **Inner Plexiform Layer (IPL).** This contains synapses between ganglion cells and bipolar cells.
- **Inner Nuclear Layer (INL).** This is where the capillary bed is found. In people with diabetes, microvascular changes originate in the capillary bed.

**Outer Retina:**

The outer retina does not contain any blood vessels and therefore receives its nutritional support from the choroidal vasculature.

- **Outer Plexiform Layer (OPL).** The cells in this layer are loosely packed. This allows for the formation of cystic spaces where fluid can easily accumulate.
- **Outer Nuclear Layer (ONL).** This contains the cell bodies of the retinal rods and cones

The retinal vasculature is found in the inner retina. The vascular endothelium has tight junctions, which maintain the blood-retinal barrier. As with the blood vessels in the rest of the body, chronically raised blood glucose causes the junctions of the retinal blood vessels to weaken.

The Retinal Pigment Epithelium (RPE), as the name suggests, is the pigmented layer of the retina. It is a mono-cellular layer, which is incapable of regeneration. The RPE is responsible for light absorption, epithelial transport, spatial ion buffering, the visual cycle, phagocytosis, secretion and immune modulation. The junctions between the RPE cells prevent leakage of extracellular fluid from the choroidal choriocapillaries into the retina. This forms the second blood-retinal barrier. The RPE is situated between the choroidal blood supply and the outer segment of the photoreceptors in the retina. It is tightly bound to the Bruch’s membrane, the innermost layer of the choroid.
The choroidal choriocapillaries are most densely concentrated directly under the macula. The walls of the blood vessels are fenestrated, which allows intravascular fluid to leak out into extravascular areas. This is the reason for the ‘damp’ environment of the choroid and the choroidal flush observed on fluorescein angiography.

In the macula area, the nerve fibre layer thins out and the inner retina disappears. There is a high concentration of tightly packed cones, which are responsible for central high definition vision. The macula appears pigmented on fundus photography due to an increase in the concentration of melanin. Hypofluorescence is observed on fluorescein angiography as the pigment blocks the underlying choroidal flush. The foveal avascular zone is highly dependent on the choroidal vasculature for its blood supply.

**Ocular Findings of Diabetic Retinopathy**

Vascular abnormalities relating to diabetes are best observed on fluorescein angiography.

Microaneurysms are the first clinical sign of diabetic retinopathy (Figure 3).

This abnormality occurs in the INL. Microaneurysms occur as a result of the loss of intramural pericytes, which are responsible for maintaining the rigidity and shape of the blood vessels. The firm structure of the capillaries maintains proper blood flow. The loss of pericytes results in weakening, loss of structure and subsequently ‘out-pocketing’ of the capillary walls. On fundus photography, one will observe tiny red dots in the superficial retinal layer. Microaneurysms are very small in size, are usually perfectly round and have distinct borders. Because the vessel walls are intact, hyperfluorescence is observed on fluorescein angiography. Microaneurysms can remain as long as the capillary walls remain intact. They will ‘disappear’ if the walls rupture and take the form of intra-retinal haemorrhages.

Intra-retinal haemorrhages (Dot-and-blot haemorrhages) ensue when the microaneurysms haemorrhage subsequent to a breakdown of the blood-retinal barrier. Because the cells in the OPL are loosely packed, the leaking plasma constituents will deposit in this layer simply due to the fact that there is sufficient space. Intra-retinal haemorrhages have irregular borders. On fundus photography, they will be seen under large blood vessels as they are found in the deep capillary beds. Hypofluorescence is observed on fluorescein angiography, since the leaked blood blocks the view of the underlying choroidal flush. These haemorrhages can take months to be resorbed.

When microaneurysms leak fluid constituents, the consequence is the formation of ‘hard exudates’ in the OPL. These exudates are waxy, yellow lesions with relatively distinct borders. They are highly refractile and tend to form circinate rings around leaking microaneurysms. Hypofluorescence is shown on fluorescein angiography as these lesions block background choroidal fluorescence.
The early detection of Intraretinal Microvascular Abnormalities (IRMA) is vital as they are a pre-cursor to retinal neovascularisation. IRMA is associated with retinal ischemia and is found deep in the INL. They are characteristically flat, fine, irregular and tortuous and are usually located between a venule and an arteriole.

Changes in venous calibre indicate areas of retinal hypoxia or ischaemia. These changes can present as venous dilation, beading or loop formation. Detection of these venous abnormalities is vital as they are an important predictor for neovascularisation.

Retinal neovascularisation is classified as ‘neovascularisation at the disc’ if it is found on or within 1 disc diameter of the optic nerve head. ‘Neovascularisation elsewhere’ occurs outside of 1 disc diameter from the optic nerve head, usually along the course of the major blood vessels in the nerve fibre layer (Figure 5).

These new blood vessels are weak, have thin walls which do not contain pericytes and can therefore haemorrhage relatively easily. These leak fluorescein in fluorescein angiography (Figure 6).

Microaneurysms should be very carefully watched if they are close to the macula as they may leak fluid and result in thickening of the macula known as macular oedema. Pre-retinal haemorrhages occur between the posterior vitreous face and the ILM of the retina or under the ILM. They are relatively large boat-shaped areas of bleeding, which obscure the underlying retina. Fluorescein angiography displays hypofluorescence.

Nerve fibre layer haemorrhages are relatively small and follow the contours of the nerve fibre layer causing them to appear flame-shaped. They are usually in the same plane as the large retinal blood vessels.

‘Cotton wool spots’ occur as a consequence of ischaemia to the NFL. They are generally white superficial lesions which have feathery borders and follow the contours of the NFL.

Microaneurysms should be very carefully watched if they are close to the macula as they may leak fluid and result in thickening of the macula known as macular oedema. The accumulation of fluid alters the architecture of the photoreceptors, preventing the macula from functioning adequately. Patients subsequently experience symptoms of decreased central vision, metamorphopsia and impaired colour vision. Macular oedema can occur at any stage of diabetic retinopathy, although it is more likely to occur as the disease progresses.
Hypertension, preeclampsia, increased severity and duration of diabetes prior to pregnancy and poor pre-pregnancy glycaemic control all accelerate the worsening of diabetic retinopathy during pregnancy.

Macular oedema may worsen and often coexists with nephropathy, hypertension and proteinuria. Although this often regresses postpartum, it may persist and result in long-term visual degradation.

**Screening Protocol for Pregnancy and Diabetes**

Women who have diabetes and plan to become pregnant should have a pre-pregnancy ophthalmological assessment. Then during the first trimester of pregnancy, a follow-up should be scheduled. For those women with diabetes in whom no retinopathy or moderate non-proliferative diabetic retinopathy is discovered, a one-year follow-up should be scheduled.

Patients who have severe, non-proliferative diabetic retinopathy, or worse, should be followed closely (monthly).

Patients who have gestational diabetes only, do not require retinopathy screening.

**Treatment of Diabetic Retinopathy in Pregnancy**

The standard treatment of proliferative diabetic retinopathy in pregnancy is pan-retinal photocoagulation (PRP) laser and the standard treatment of clinically significant macular oedema is focal or grid photocoagulation laser (depending on whether the macular leakage is focal or diffuse).

The risk of anti-VEGF agents such as bevacizumab (Avastin) in pregnancy cannot be ruled out.

**Conclusion**

By performing a full ocular assessment, we are able to detect the effects of a wide variety of systemic diseases. In many of these conditions, the ocular signs may be the first presentation of the disease. The importance of a thorough ocular examination cannot be over emphasized in detecting, diagnosing and monitoring diabetes-related eye disease.

Optical Coherence Tomography (OCT) scanning has opened up a new domain for clinically correlating a flat two-dimensional image obtained with conventional fundus photography with that of the three-dimensional imaging of ocular pathology. The accurate measurement capability of the OCT allows very precise monitoring of progression or improvement of retinal pathology.

It is important for all healthcare professionals to work together in a coordinated system, to ensure that patients do receive appropriate screening and correct management.
Non Alcoholic Fatty Liver Disease

Introduction
Non-alcoholic fatty liver disease (NAFLD) refers to hepatic fat accumulation in the absence of a readily identifiable secondary cause e.g. excess alcohol. Awareness of NAFLD has increased rapidly over the last two decades, in keeping with the increase in prevalence of obesity and type 2 diabetes mellitus (T2DM). It is now the most common liver disorder in Western developed countries, strongly associated with the metabolic syndrome and its major pillars. Most patients are asymptomatic and NAFLD is suspected based on incidental elevation of aminotransferases or hepatic fatty infiltration on abdominal imaging. NAFLD is a risk factor for future liver cirrhosis and may be a causative factor in the entity of ‘idiopathic’ (cryptogenic) liver cirrhosis.

The term NAFLD encompasses a broad spectrum of hepatic fat accumulation and the consequences thereof (Figure 1). It can be broadly divided into non-alcoholic fatty liver (NAFL) and non-alcoholic steatohepatitis (NASH). NAFL refers to ‘benign’ hepatic steatosis without evidence of a significant inflammatory response, whereas NASH is associated with significant hepatic inflammation, which may progress to fibrosis and even cirrhosis.

In the pre-fibrotic stages, NASH may be indistinguishable from alcoholic steatohepatitis on histology. At the fibrotic stage, the distribution pattern of fibrosis may enable differentiation between the two. Centrilobular fibrosis is more common in NASH whereas peri-hepatic vein and peri-portal fibrosis indicates alcoholic liver disease. For the diagnosis of NAFLD, daily alcohol intake must be less than 20 g per day for females and less than 30 g per day for males, i.e. 2 standard alcoholic drinks per day for men and 1.5 for women. The steatotic liver may be more vulnerable to injury and progression when challenged with additional insults (especially alcohol).

Obesity, type 2 diabetes and hyperlipidaemia, specifically hypertriglyceridaemia are frequently associated with NAFLD. Central visceral obesity appears to be an important risk factor, even in patients with a normal body

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Figure 1: The spectrum of NAFLD
mass index (BMI) (Table 1). The exact prevalence of NAFLD is unknown, but it is estimated to occur in more than 60% of the obese population (regardless of the diabetes status), as well as in more than 50% of patients with type 2 diabetes. However, only a small proportion of these will have NASH or worse.

Table 1: Risk factors for NAFLD

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<th>Risk Factor</th>
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<tr>
<td>Visceral adiposity</td>
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<tr>
<td>Hypercholesterolemia</td>
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<tr>
<td>Hypertriglyceridaemia</td>
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<tr>
<td>Diabetes / Insulin resistance</td>
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Pathogenesis
The pathogenesis remains poorly understood, but insulin resistance (IR) is strongly associated with hepatic fat accumulation. Hepatic IR contributes to fat accumulation in hepatocytes by disrupting the balance between hepatic fat import and export. Simplistically, increased intrahepatic levels of free fatty acids (due to IR) provide a source for oxidative stress, which leads to inflammation and ultimately fibrosis. It is not fully understood why plain steatosis develops in some and steatohepatitis in others.

Clinical features and Laboratory abnormalities
Most patients have no signs or symptoms of liver disease, but have incidentally discovered abnormalities on liver function testing. Hepatomegaly is the only physical finding in most patients, but need not be present. If liver cirrhosis is present, one may find clinical features of portal hypertension and haematological features of hypersplenism. Medications contributing to or causing NAFLD (e.g. glucocorticoids, oestrogens, tamoxifen, amiodarone and certain classes of anti-retroviral medication) need to be considered in the aetiology and discontinued if possible.

Liver biopsy remains the gold standard for diagnosis. It is useful for grading the severity of fat accumulation, as well as screening for the presence of inflammation or fibrosis, provided that a representative sample is obtained.

Liver enzymes are not a sensitive marker of NAFLD. Patients with liver function abnormalities in NAFLD usually have mild to moderate elevations in liver enzymes but normal levels do not exclude this entity. When abnormal, the aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are usually not more than two to five times elevated. The AST/ALT should be < 1 in NAFLD. [A ratio of > 1 is seen in early alcoholic fatty liver disease]. Serum alkaline phosphatase (ALP) and gamma-glutamyltransferase (GGT) may be elevated but usually to a less significant degree. It is advocated that other chronic liver diseases such as auto-immune hepatitis, the viral hepatitides (A, B, C) and haemochromatosis be excluded. The decision on how extensive the serologic evaluation needs to be should be tailored to the individual patient. Elevated ferritin and other parameters of excess iron are often found in NAFLD, but not to the levels found in haemochromatosis.

The diagnosis of NAFLD is based on the exclusion of other causes of hepatic steatosis and a liver biopsy may be needed if the diagnosis is uncertain.

Imaging
The hallmark of NAFLD on radiological imaging is an increased fat content in the liver. No imaging modality can reliably distinguish between NAFL and NASH. Due to its low cost, availability and safety profile, ultrasound (US) is currently the imaging modality of choice. US has a sensitivity of 60-94% and a specificity of 84-95% to detect fatty liver. The sensitivity decreases with a lower liver fat content and therefore one may miss the diagnosis in a very fibrosed liver. Liver stiffness, as measured by US techniques such as FibroScan®, has been used to estimate the presence and severity of liver fibrosis. It appears to be a suitable tool to screen high-risk populations, but lacks specificity.

Histology
Significant steatosis is defined as fat accumulation in more than 5% of hepatocytes. Liver biopsy remains the gold standard for diagnosis. It is useful for grading the severity of fat accumulation, as well as screening for the presence of inflammation or fibrosis, provided that a representative sample is obtained. Only histology can adequately differentiate between NAFL and NASH. Nevertheless, liver biopsy remains an invasive procedure; although the risk for significant complications is small, it is not negligible. In view of the high prevalence of NAFLD, routine liver biopsy could add markedly to the health expenditure in this patient group. In most cases, a liver biopsy is reserved for patients with an unclear diagnosis of NAFLD, or with stigmata of possible liver cirrhosis.

NAFLD and Diabetes
A clear association exists between diabetes and NAFLD. Even in patients with ‘pre-diabetes’ the risk of NAFLD is higher than in the background population. NAFLD predicts the development of diabetes and vice versa. It is advised that all patients with NAFLD should be screened for diabetes. Having
both conditions in the same person increases the morbidity associated with having either alone. Furthermore, NAFLD has been associated with an increased risk of micro and macrovascular complications in patients with diabetes. Diabetes is particularly associated with disease progression in patients with NASH. Patients with NAFLD are also at increased risk of hepatocellular carcinoma (HCC), independent of the presence of viral hepatitis. The majority of a recently studied cohort of patients with NAFLD and HCC also had diabetes (59%) - the question remains if this merely reflects the overall increased cancer risk in patients with diabetes, independent of NAFLD.

**NAFLD progression**

Hepatic steatosis alone is regarded as a benign form of NAFLD, but the presence of NASH is associated with an increased risk of liver fibrosis and subsequent end stage liver disease. The only conclusive way to differentiate between the two is by biopsy. It is therefore important to define risk factors for progression to NASH and its complications. Insulin resistance, hypertension and elevated ALT amongst others (Table 2) have all been shown to be independently associated with NASH, while T2DM and smoking have been associated with progression to more advanced liver fibrosis in patients with NASH. However, in general, most people with NASH will not progress and go on to cirrhosis.

Nevertheless, it is recommended to avoid all hepatotoxins in anyone with any evidence of, or, indeed, anyone at significant risk of NAFLD.

<table>
<thead>
<tr>
<th>Table 2: Markers for progression of NAFLD</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Older age</strong></td>
</tr>
<tr>
<td><strong>Diabetes Mellitus</strong></td>
</tr>
<tr>
<td><strong>BMI &gt; 28 kg/m²</strong></td>
</tr>
<tr>
<td><strong>Visceral adiposity</strong></td>
</tr>
<tr>
<td><strong>Aminotransferases &gt; 2 x elevated</strong></td>
</tr>
</tbody>
</table>

**Management**

The main goal of therapy should be to prevent progression from NASH to liver fibrosis. As with type 2 diabetes, the mainstay of management in overweight patients is directed at weight loss. The degree of hepatic fatty infiltration has been shown to decrease with gradual, sustained, weight loss of 5-7%. As with all lifestyle interventions, this is easier said than done. Good glycaemic control is mandated in people with diabetes, but is not always effective in reversing NAFLD.

There are good arguments in favour of bariatric surgery, especially in the obese patient with T2DM and NAFLD. However, no randomized controlled trials have examined bariatric surgery as a treatment option for NASH.

Patients with NAFLD are at increased risk for cardiovascular disease, *per se*. They require optimal management of all the other classical macrovascular risk factors. Statin therapy has been shown to be safe in patients with NAFLD. It acts indirectly by lowering lipid levels.

No specific pharmacological intervention has been shown to be effective in the treatment of NASH/NAFLD. Various drugs have been studied, but none have shown proven benefit in liver damage, independent of weight loss. Metformin has been widely studied, but results are disappointing. Peroxisome proliferator activated receptor gamma (PPARγ) is a nuclear receptor expressed mainly in adipose tissue and the liver. It regulates the uptake of fatty acids and glucose. Early use of pioglitazone, a PPARγ agonist, has shown some improvement in progression to liver fibrosis but this needs further study.

Incretins inhibit glucagon secretion and decrease liver gluconeogenesis and glycogenolysis. This results in improved glycaemic control, weight loss and increased insulin sensitivity, which could be beneficial in NAFLD. Preliminary data with exenatide indicates a decrease in fat accumulation in the liver, while lowering liver enzymes. However, histologic evidence of benefit is unavailable and, at present, incretins are not indicated for the treatment of NAFLD.

Based on its antioxidant properties, vitamin E has been tested in NASH. In the PIVENS trial (Pioglitazone versus Vitamin E versus Placebo for the Treatment of Nondiabetic Patients with Nonalcoholic Steatohepatitis), vitamin E therapy demonstrated a significant improvement in steatosis and inflammation in adult patients without diabetes or cirrhosis. Evidence of its benefit remains inconclusive in patients with diabetes. The recommended dose in patients without diabetes, but with active NASH, is 800 IU/day.

Ursodeoxycholic acid (UDCA) has theoretical benefit by decreasing cytokines implicated in the pathophysiology of NASH, but results of clinical trials were disappointing.

On a final clinical note, patients with chronic liver disease, including NAFLD and NASH require vaccinations for viral hepatitis if they are not already immune.

**Summary**

- NAFLD is currently the leading cause of liver disease in the developed world.
- Insulin resistance plays a critical role in the pathogenesis.
- NAFLD is a diagnosis of exclusion.
- NASH is ‘benign’, but NASH can progress to cirrhosis.
- Liver biopsy is reserved for patients who have risk factors for NASH and/or in whom other liver diseases need to be excluded.
- No effective pharmacological therapy is currently available but sustained weight reduction may improve the outcome.
- Patients with NAFLD are at increased risk for cardiovascular disease and thus concomitant traditional risk factors require optimisation of therapy.
Globally, the number of women with diabetes in pregnancy is increasing in line with the incidence of obesity amongst women of reproductive age. The current ‘obesity epidemic’ is also contributing to the increasing number of women developing impaired glucose tolerance during pregnancy.

In general, women of childbearing age should maintain good nutritional status through a lifestyle that optimizes maternal health and reduces the risk of birth defects and suboptimal foetal growth and development. This includes appropriate weight gain, physical activity, consumption of a healthy, balanced intake of a variety of foods, appropriate and timely vitamin and mineral supplementation and avoidance of harmful substances.

“For the little one, the very first menu has a lifelong effect!”

This quote from the University of Stellenbosch Nutrition Information Centre highlights the importance of nutrition during pregnancy.

**Pre-Pregnancy care**

Diabetes and/or being overweight may have a negative impact on maternal and foetal pregnancy outcomes. Planning and preparation is essential. This includes optimizing glycaemic control, folic acid supplementation and stopping all unsafe medication prior to conception.

This remains a major clinical challenge as the unintended pregnancy statistics show that health care professionals are not achieving this goal.

According to data published in 2011 for the United States, 49 % of pregnancies were unintended. There appears to be a trend amongst women aged 19 years and younger, where more than 4 out of 5 pregnancies were unintended.

Large increases in unintended pregnancy rates were found among women with lower education, low income, and women cohabiting with their partners.

This is concerning - much more emphasis should be placed on pre-pregnancy education.

1. **Weight**

Pre-pregnancy body mass index (BMI) is an independent predictor of many adverse outcomes in pregnancy (Table 1).

Table 2 contains the US Institute of Medicine (IOM) recommendations for total weight gain and rate of weight gain during pregnancy. This has been the standard for more than 30 years and many studies continue to find that the recommended IOM range is generally associated with better
Table 1: The possible adverse outcomes that may occur with increased BMI during pregnancy

<table>
<thead>
<tr>
<th>Maternal complications</th>
<th>Birth outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Pre-eclampsia</td>
<td>• Neural tube defects</td>
</tr>
<tr>
<td>• Gestational diabetes mellitus</td>
<td>• Congenital anomalies</td>
</tr>
<tr>
<td>• Infections such as urinary tract infection</td>
<td>• Intrauterine foetal death and stillbirth</td>
</tr>
<tr>
<td>• Complicated delivery</td>
<td>• Macrosomia</td>
</tr>
<tr>
<td>• Caesarean section wound-related infections</td>
<td></td>
</tr>
</tbody>
</table>

Table 2: Institute of Medicine (IOM) recommendations for total weight gain and rate of weight gain during pregnancy

<table>
<thead>
<tr>
<th>Pre Pregnancy BMI (kg/m²)</th>
<th>Total weight gain (kg)</th>
<th>Rates of weight gain in 2nd and 3rd trimester (Mean kg/week*)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight (&lt;18)</td>
<td>12.5-18</td>
<td>0.51 (0.44-0.58)</td>
</tr>
<tr>
<td>Normal weight (18.5-24.9)</td>
<td>11.5-16</td>
<td>0.42 (0.35-0.50)</td>
</tr>
<tr>
<td>Overweight (25.0-29.9)</td>
<td>7-11.5</td>
<td>0.28 (0.23-0.33)</td>
</tr>
<tr>
<td>Obese (≥30.0)</td>
<td>5-9</td>
<td>0.22 (0.17-0.27)</td>
</tr>
</tbody>
</table>

*Calculations assume a 0.5 to 2 kg weight gain in the first trimester.

Table 4: Folate is found in small amounts in many foods. Good sources include:

<table>
<thead>
<tr>
<th>Good sources</th>
<th>Good sources</th>
</tr>
</thead>
<tbody>
<tr>
<td>liver</td>
<td>spinach</td>
</tr>
<tr>
<td>asparagus</td>
<td>peas</td>
</tr>
<tr>
<td>chickpeas</td>
<td>fortified breakfast cereals</td>
</tr>
<tr>
<td>asparagus</td>
<td>broccoli</td>
</tr>
</tbody>
</table>

*Folate, known as folate in its natural form, is one of the B-group vitamins. Folic acid in synthetic form (found in fortified food and supplements) is absorbed twice as well as dietary folate. 1 µg of folate acid = 1.7 µg of dietary folate.

As experts in food and nutrition, registered dietitians are in a unique position to provide counselling to assist women pre-conception to achieve a body weight as close as possible to that required for health.

2. Folic acid supplementation

A folate-rich diet, in addition to 400 µg per day folic acid supplementation, is recommended for all women capable of becoming pregnant, including adolescents. A daily dose of 5 mg is recommended for women with diabetes from 3 months prior to conception because the risk of neural tube defects (NTD) in this population is higher than in the background population. The Endocrine Society recommends that after 12 weeks of gestation, the dose of folic acid be reduced to 0.4 to 1.0 mg/day which should be continued until the completion of breastfeeding.

Figure 1 illustrates the effect of folic acid fortification in wheat and maize flour. Although the amounts are minimal, it has resulted in a significant reduction in NTD’s since 2001. Early folic acid supplementation is still fundamental.
3. Glycaemic Control
It is the responsibility of health care providers to counsel women with diabetes to plan their pregnancies. This is to ensure they have good glycaemic control prior to conception, which results in the best chance of a healthy pregnancy outcome.

Studies have shown that maternal glycaemic control in women with diabetes influences foetal growth as early as the first trimester. An elevated first-trimester HbA1c level is the strongest predictor of macrosomia*. Maternal hyperglycaemia causes foetal hyperinsulinaemia, which results in accelerated foetal growth. Cardiac and central nervous system (CNS) congenital abnormalities are the most common diabetes related anomalies.

*Macrosomia - Approximately half of all neonates born to women with diabetes are large for gestational age meaning a birth weight above the 90th percentile for that gestational age.

4. Other factors to consider
- Smoking
- Caffeine - this will be discussed in the section on pregnancy below
- Alcohol
- Drugs and other medication
- Screening for diabetes-related complications

Pregnancy
Nutritional requirements during pregnancy
During pregnancy, women should be assessed and monitored by a registered dietitian to ensure that nutrition therapy promotes euglycaemia as far as possible. Appropriate weight gain and adequate nutritional intake is essential.

Energy
Caloric excess does not guarantee adequate intake or nutrient status critical to healthy pregnancy outcomes. Equally, hypocaloric diets are not recommended as they may result in weight loss and substantial ketosis. They are also likely to be deficient in necessary nutrients.

| Table 5: Recommendations by the American Diabetes Association (ADA) and the United Kingdom National Institute for Health and Care Excellence (NICE) for pre pregnancy HbA1c |
| <7 % | <6.5 % |
*these targets need to achieved without causing problematic hypoglycaemia

| Table 6: Estimated Energy Requirement (EER) for Pregnancy (IOM, 2006) |
| EER (kcal/kJ per day) = Non pregnant EER + Pregnancy Energy |
| 1st trimester | 0 extra kcal / kJ |
| 2nd trimester | 340 extra kcal / 1428 kJ |
| 3rd trimester | 452 extra kcal / 1898 kJ |

The ADA (2004) proposed that for a mother who has an ideal BMI, the usual energy requirements are approximately 30 kcal / 126 kJ/kg body weight per day. For a mother with a BMI> 25 kg/m², a 20 % energy restriction to approximately 24 kcal / 101 kJ/kg body weight is needed per day.

It is suggested that obese women with overt or gestational diabetes reduce their calorie intake by one third (compared with their usual intake before pregnancy) while maintaining a minimum intake of 1600-1800 kcal/day. This improves mean glycaemia without inhibiting foetal growth or attainment of a healthy birth weight, or inducing ketosis. Some studies in women with type 1 diabetes have revealed that an energy intake of less than 1500 kcal / 6300 kJ per day may increase ketosis risk and this may be linked to impaired foetal brain development.

Carbohydrate (CHO)
It has been suggested that CHO is limited to 35-45 % of total energy. It is preferable for this to be distributed in 3 small to moderate sized meals through the day and snacks if necessary. For patients on basal bolus regimens (BBR), it needs to be remembered that these snacks also need to be covered with rapid-acting insulin. There is no conclusive evidence for the ideal percentage of carbohydrate in the diet of women with overt or gestational diabetes. However there are proposed values such as 40-45 % of total energy, and even as high as 60 % provided it comes from complex sources. The IOM has suggested a minimum of 175 g / day which is higher than the general amount of 130 g/day. This is to account for foetal brain glucose utilisation.

However, irrespective of which guideline is used, there needs to be an overall restriction and reduction of carbohydrate which should be guided by the patients’ blood glucose levels. This highlights the necessity of frequent blood glucose monitoring.

Manipulating the type of carbohydrates selected may be useful, and although no interventional studies have been conducted using the glycaemic index in the context of pregnancy and diabetes, it is prudent to suggest lower glycaemic index choices.
The recently published ROLO study, a randomised controlled study, looked at the role of a low glycaemic index (GI) diet in pregnant women, without diabetes, to prevent macrosomia. This study included 800 women, all in their second pregnancy, who had previously delivered an infant with a birth weight more than 4 kg. The chief outcome measure was difference in birth weight. The secondary outcome measure was difference in gestational weight gain. Subjects were randomised to receiving no dietary intervention or being started on a low GI diet early in pregnancy. It was concluded that a low GI diet in pregnancy did not reduce the incidence of large for gestational age (LGA) infants in a group at risk of foetal macrosomia. However there was a significant positive effect on gestational weight gain and maternal glucose intolerance. Thus a low GI diet may blunt the mid and late pregnancy increase in insulin resistance and may influence normal infant birth weight and normal maternal weight gain.

**Micronutrients**

**Folic acid**

Pregnant women in the general population are advised to increase their intake to 600 µg folic acid or dietary folate equivalent. N.B this amount is 5 mg in women with diabetes.

**Iron**

Iron deficiency with ensuing anaemia is the most widespread micronutrient deficiency affecting mostly pregnant or lactating women. Maternal and foetal demand for iron increases during pregnancy, and iron deficiency anaemia during the first two trimesters of pregnancy can increase the risk for preterm labour, low birth weight and infant mortality. This stresses the need for encouragement of iron-rich foods and iron supplementation. A low-dose iron supplement such as 30 mg/day is recommended.

**Vitamin D**

The need for and safety and effectiveness of Vitamin D supplementation during pregnancy continues to be controversial. The IOM recommends 600 IU per day for adults, which includes pregnant women, however 1000-2000 IU/day supplementation is considered safe.

**Calcium**

The Dietary Reference Intake (DRI) for calcium in pregnancy is similar to that for non-pregnant women of the same age, due to increased efficiency in calcium absorption during pregnancy and maternal bone calcium mobilisation. Women with suboptimal intakes (< 500 mg/day) may need additional amounts to meet both maternal and foetal bone requirements. The IOM suggest an intake of 1000 mg/day with an upper limit of 2500 mg.

**Omega 3 fatty acids**

These are especially important during pregnancy for foetal growth and development. Intake of n-3 fatty acids (200 µg), particularly docosahexaenoic acid (DHA) is associated with improved infant visual and cognitive development. This is found in approximately 240 g of fatty fish eaten per week during pregnancy. It is also found in flaxseed, canola oil and DHA enriched eggs. High methyl mercury fish sources should be limited e.g. shark, swordfish, king mackerel and white tuna.

**Safe food handling:** pregnant women and their unborn children are more susceptible to food poisoning. Patients must ensure that foods are cooked well and reach the safe minimum internal temperature appropriate for each food. Unpasteurized milk, soft cheeses and juice should be avoided. There should also be caution with certain contaminants such as listeria monogenes and polychlorinated biphenyls (PCB’s).

**Artificial sweeteners**

There is limited research on the safety of their use. They currently have GRAS status i.e. Generally Regarded As Safe.

**Caffeine**

The Foods Standards Agency (FSA) in the UK has issued new advice to pregnant women about the amount of daily caffeine consumption that is safe for pregnancy. In light of new research, they have reduced the recommendation from 300 mg/day to 200 mg/day (approximately 1-2 cups of coffee).

**Gestational Diabetes and Glucose Monitoring During Pregnancy**

Risk factors for developing gestational diabetes are:

- A BMI above 30 kg/m²
- Previous macrosomic baby weighing > 4.5 kg
- Previous gestational diabetes
- Family history of diabetes - first degree relative with diabetes
- Family origin with high prevalence of diabetes

**Diagnostic Criteria**

Most Associations recommend the 2-hour 75 g oral glucose tolerance test (OGTT) at 24-28 weeks of gestation:

The International Association of Diabetes and Pregnancy Study Group (IADPSG) diagnostic criteria are:

- Fasting plasma glucose: ≥ 5.1 mmol/L
- One-hour post OGTT plasma glucose: ≥ 10.0 mmol/L
- Two-hour post OGTT plasma glucose: ≥ 8.5 mmol/L
The diagnostic criteria from the NICE 2015 Guideline on Diabetes in pregnancy are:

- Fasting plasma glucose level: ≥ 5.6 mmol/L or
- Two-hour post OGTT plasma glucose level: ≥ 7.8 mmol/L.

Treatment for GDM must begin immediately after diagnosis. This should include dietary intervention, blood glucose monitoring, possible insulin therapy and frequent visits to health care providers to ensure behaviour and self care modifications.

As nutrition plays a critical role in the management of GDM, it is recommended that women diagnosed with GDM be referred to a registered dietitian for individual nutrition management.

Please note that different doctors apply different monitoring targets to their patients. It is important that these are consistent throughout the healthcare team, and that they are clearly communicated to the registered dietitian involved as he/she will most often be first line in offering advice.

**Monitoring Targets to Achieve**

**HbA1c**
ADA: <6% achieved without significant hypoglycaemia

**Blood Glucose Monitoring**
ADA 2015 and Endocrine Society 2013 recommendations for management of GDM:
- Pre-prandial blood glucose: ≤ 5.3 mmol/L
- One-hour post meal blood glucose: ≤ 7.8 mmol/L
- Two-hour post meal blood glucose: ≤ 6.7 mmol/L

**NICE 2015**
- Fasting blood glucose: 5.3 mmol/L
- One-hour postprandial blood glucose: 7.8 mmol/L
- Two-hour postprandial blood glucose: 6.4 mmol/L

Breastfeeding in patients with type 1 diabetes may lead to hypoglycaemia and insulin doses may need to be adjusted.

Patients with overt or gestational diabetes should have weekly contact with their healthcare team.

**Postpartum care**

**Breastfeeding**
All women should be encouraged to attempt to breastfeed their babies by informing them of the immediate nutritional and immunological benefits of breastfeeding for the baby.

Breastfeeding can aid weight loss as it uses substantial amounts of energy. A healthy breastfeeding mother can lose as much as 0.45 kg per week and still supply adequate milk to maintain the growth of her infant.

Breastfeeding in patients with type 1 diabetes may lead to hypoglycaemia and insulin doses may need to be adjusted. If this occurs, it is advised that a meal or snack may be needed before or during feeding.

**Weight**
Postpartum weight gain after GDM is linked to an earlier progression to type 2 diabetes.

Patients with GDM and type 2 diabetes should be motivated to continue with their healthy way of living and eating that they had adopted during pregnancy, to support the weight loss recommended in the postpartum period.

**Screening** for diabetes after GDM, using non pregnancy criteria, should be done 6-12 weeks postpartum, and every one to three years thereafter.

**Conclusion**
This article highlights the importance of nutrition before, during and after pregnancy. All these measures may assist in reducing diabetes associated pregnancy complications, and result in a healthy pregnancy outcome.
The Romantic Poet Shelley penned these famous words in 1819 in the poem Ode to the West Wind. He surely was not referring to contracting influenza, but was rather spurring reform in England, his troubled homeland. I use it here today to start the discussion on how to guide your patients in making an informed decision on whether or not to have an annual influenza vaccination.

The start of the ‘flu season’ often serves as a herald of autumn. Viral influenza, a common and sometimes troublesome infection is seen more often at this time of the year and onwards into the winter months. Furthermore, bacterial infections such as those caused by the pneumococcal bacteria also tend to present more commonly in wintertime. Infections resulting from these viruses and bacteria can result in the development of bronchitis, pneumonia and otitis media.

Influenza typically is highly contagious, and tends to have a sudden onset of symptoms, which usually include fever, chills, coughing and aching muscles and joints. Most people will make a complete and uneventful recovery usually within two weeks. However, in some people, complications may arise. Rarely, influenza can be fatal. People with diabetes are considered to be at greater risk of complications arising from influenza. This is especially true for those people who might have additional healthcare problems such as heart and kidney disease. In previous years, where the Swine Flu strain has been troublesome, pregnant women were also regarded as a high-risk group.

Much like the routine practice of assessing people with diabetes for potential complications arising from sub-optimal glucose control, immunization against influenza forms part of a preventative strategy endorsed by numerous healthcare bodies globally. It has been said that vaccinating people considered to be at high risk before the ‘flu season is the most effective measure for reducing the negative impact of influenza. Having a ‘flu shot is one additional way our patients can remain in control of their diabetes.

In order to attain the maximum benefit from influenza vaccination, it should be administered well in advance of winter. In South Africa, vaccine supplies are typically made available in early to late March of each year. Immunity to the virus takes at least two weeks to develop after receiving the vaccine. All ‘flu vaccinations are given by means of a single injection into the upper portion of either the right or left arm. Children aged six and above as well as those who are pregnant are also eligible for vaccination.

Although the vaccines are cultured in eggs, generally, egg-allergic patients can safely receive their vaccination. However, individuals with a history of severe (life threatening) allergy to eating eggs should consult with an allergy specialist prior to receiving it. Only recently has a vaccine cultured in
an egg-free medium become available in the United States. For the time being, this novel development is not yet available in South Africa.

The treatment people with diabetes are currently taking to control their blood glucose does not influence the effectiveness of the influenza vaccine. Because the vaccine is not manufactured from a live virus, receiving it cannot cause the development of influenza. Some people however might develop a low-grade fever in the first few days after their vaccination. This reflects their immune response to the vaccine and is typically short-lived. Those who have a febrile illness at the time of the scheduled vaccination should defer their shot until this resolves.

In spite of receiving the flu vaccination, people with diabetes may still develop influenza. The good news is that their illness will be of shorter duration, less severe and they will be at lower risk of developing any complications such as pneumonia.

For some people with diabetes, contracting the ‘flu can result in several complications. These can include an abrupt deterioration in blood glucose control and the development of ketones and possibly diabetic ketoacidosis in those with type 1 diabetes. It is vital to remind our patients at this time of year of this possibility and it is an appropriate time to review their sick-day management protocols.

Because the viruses causing influenza can vary from year to year, it is important that patients are vaccinated before winter each year. I also make a point of reminding anybody who receives a ‘flu shot that the influenza vaccination does not prevent them contracting the ‘common cold’. The World Health Organisation has recommended coverage of three strains for the 2015 Southern Hemisphere influenza season:
- an A/California/7/2009 (H1N1)pdm09-like virus;
- an A/Switzerland/9715293/2013 (H3N2)-like virus;
- a B/Phuket/3073/2013-like virus.

A further point worthy of mention is that the annual American Diabetes Association Clinical Guidelines continue to endorse the additional provision of Pneumococcal immunization. Here again, people with diabetes are at greater risk for morbidity and mortality arising from Pneumococcal bacteraemia. This is especially true for those above 65 years of age and those with existing chronic pulmonary, renal or cardiac diseases. The value of immunization in respect of Pneumococcal infections is to reduce the invasive disease complications. As such, the target groups for vaccination here are the same for influenza. Typically, the Pneumococcal vaccine is given as a one-off. Repeat vaccination is advised for those older than 64 years of age who were immunized when they were below 65 years of age and the vaccine was administered more than 5 years previously. Additional recipients of repeat vaccination should include those with chronic kidney disease. The Pneumococcal vaccination may be given simultaneously with the influenza vaccination, but as a separate injection at a separate site. The provision of both vaccines does not increase side effects or reduce either vaccine’s efficacy.

Ultimately the best way to reduce the risk of ‘catching the ‘flu’ is by preventing the spread of germs. We should all continue to encourage people to cover their coughs and sneezes, quickly dispose of any tissues after one use and to wash their hands regularly (especially before and after shaking hands in greeting).

Finally, provision of both the aforementioned vaccinations can be considered appropriate to those members of the healthcare team who come into direct contact with patients. If vaccination is performed in the medical office or healthcare centre, it is incumbent that appropriate standard operational procedures are followed. This should include immediately available resuscitation equipment.

It thus seems wise that we should initiate discussion with our patients about the benefits of being vaccinated.

**If vaccination is performed in the medical office or healthcare centre, it is incumbent that appropriate standard operational procedures are followed**

Shelley ends his poem with a hopeful message. My wish is that you and your patients have a healthy and uneventful winter.

“O Wind, If Winter comes, can Spring be far behind?”

South African Journal of Diabetes  ▪ May/June 2015  26
Mood disorders during and after pregnancy

Introduction
Many people consider pregnancy a time of happiness and well being, but it is common for women to experience changes in their feelings and mood during pregnancy. This may include being more tired, irritable or worried. If these mood symptoms become severe enough, they may need medical intervention. About 10-20 % of moms-to-be have increased vulnerability to psychiatric conditions such as depression, anxiety disorders, eating disorders and psychoses.

Psychiatric conditions associated with Pregnancy
- **Depression**: is the most common disorder associated with pregnancy.
- **Psychosis**: While it is rare for women to experience first-onset psychoses during pregnancy, relapse rates are high for women previously diagnosed with some form of psychosis.
- **Bipolar disorder and Schizophrenia**: It appears that some women with bipolar disorder and schizophrenia experience a relief from symptoms during pregnancy, but the risk of relapse in the postpartum period is high.
- **Panic disorder**: Women who have pre-existing panic disorder will most likely continue to have symptoms during pregnancy.
- **Obsessive Compulsive Disorder (OCD)**: Several reports suggest that women may have an increased risk of the onset of OCD during pregnancy and the postpartum period. In one study of 109 women with diagnosed OCD, 39 % of the participants reported that their OCD began during pregnancy.
- **Eating Disorders**: The prevalence of eating disorders in pregnant women is approximately 4.9 %. Obesity and starvation (anorexia nervosa) have negative consequences for the mother and the baby.

These psychiatric conditions are often underdiagnosed because they are thought of as ‘mere hormonal changes’. They are also undertreated because of potential harmful effects of medication on the growing foetus.

Depression during Pregnancy
Is there any difference in the incidence of depression for those mothers who have diabetes? Research done is inconclusive. Kozhimannil (2009) from Harvard Medical School noted that pregnant women and new mothers had

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nearly twice the incidence of depression as non-diabetic women who were pregnant or had just given birth. She noted that more research is needed to determine if diabetes plays a direct role in depression during and immediately after pregnancy. Considering that depression is twice as likely in all people with diabetes, the incidence of depression during pregnancy is no different.

Katon et al (2011) found that neither pre-existing diabetes nor gestational diabetes was associated with increased risk of antenatal depression. Women with the highest risk of having peri-natal depression are those who have a previous history of depression, who discontinue their anti-depressant medication(s), who have a previous history of postpartum depression and a family history of depression.

Several psychosocial correlates may also contribute to depression during pregnancy:

1. A lack of support from family and friends and feeling isolated – Joan Webster et al (2001) found that women with low social support in pregnancy were more likely to report poorer health during pregnancy and postpartum, to seek medical help more frequently and to be more depressed postpartum.
2. A young age at the time of pregnancy - Dr. Stefanie Mollborn (2010) of the University of Colorado, showed that teenage mothers had higher levels of depression than other teenagers or adult mothers, especially if they had been depressed before the pregnancy.
3. Anxiety or negative feelings about the pregnancy make depression more likely.
5. Previous infertility - a woman who had been infertile and finally becomes pregnant, has fluctuations in hormone levels that occur while attempting to get pregnant, followed by the hormonal changes in pregnancy which can increase the risk of depression.
6. A difficult pregnancy and/or delivery with the threat of losing the baby, as may happen with women with diabetes.
7. A previous child born with long- or short-term problems creates great anxiety about the current pregnancy and can give rise to depression.
8. Marital problems - a partner who is unhappy about the pregnancy or an environment of domestic violence can make pregnancy more difficult for the mother and lead to depression.
9. Other stressful life events at the time of pregnancy.
10. Substance abuse.

**Postpartum Depression**

Women who are depressed during pregnancy have a greater risk of being depressed after giving birth. Levels of oestrogen and progesterone increase greatly during pregnancy. In the first 24 hours after childbirth, hormone levels quickly return to normal. This big change can give rise to mood changes. Levels of thyroid hormones may also drop after giving birth, leading to hypothyroidism. Symptoms would include tiredness, sluggishness and depression. A TSH test should indicate if thyroid levels are normal.

50-80% of women develop ‘baby blues’ for 1-3 weeks after delivery. They have mood swings, feel anxious or overwhelmed, cry more often, lose their appetite and have trouble sleeping. Symptoms that last longer than 3 weeks, and are more severe, should be regarded as postpartum depression. Postpartum depression occurs in about 10% of births.

Symptoms can begin anytime within the first year after childbirth. They include the usual symptoms of depression such as tearfulness, loss of interest in usual activities, feelings of guilt, worthlessness or incompetence, irritability, sleep and appetite disturbances, and poor memory and concentration. Symptoms more specific to the postpartum period are very negative thoughts about the baby, thoughts of hurting the baby, thoughts of hurting themselves, not having any interest in the baby and thoughts of giving the baby away.

Psychosocial factors that may play a role in postpartum depression include:

- tiredness after delivery and a lack of sleep or broken sleep
- feeling overwhelmed by the new baby
- self doubt about being a good mother
- stress from the changes in work and home routines
- a sense of loss of the life led before the baby

Some women don’t tell anyone about their symptoms. They feel embarrassed, ashamed, or guilty about feeling depressed when they are supposed to be happy. They worry they will be viewed as an unfit parent.

Without treatment, postpartum depression can interfere with a mother’s ability to care for herself or her baby. She lacks the energy to focus or apply herself to meeting the needs of her baby. She is less able to follow medical or baby-care instructions. She may miss follow-up visits or she may try to self-medicate with alcohol, or over-the-counter or illegal drugs. She is less likely to manage her diabetes effectively. Initially the mother may feel guilty and lose confidence in her ability as a mother, which makes her even more depressed. If major depression develops, she loses touch with all around her and all feelings. Even the guilt is repressed. She becomes emotionally flat.

Researchers believe postpartum depression in a mother can affect her baby in the present and in the future. Babies born to mothers who are depressed may be premature.
have a low birth weight, be less active, show less attention and are more irritable and agitated than babies born to moms who are not depressed.

Depressed mothers have difficulty bonding with their infant, which makes it difficult for the baby to form an attachment to her. Research has shown that lack of attachment causes the baby to have delays in language development, express behaviour problems and have higher levels of generalized anxiety from childhood onwards. Thus it is very important that another caregiver helps to meet the baby’s needs until the mother is emotionally healthier.

The Edinburgh Postnatal Depression Scale (EPDS) is used to detect Postnatal Depression.

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**Edinburgh Postnatal Depression Scale (EPDS)**

<table>
<thead>
<tr>
<th>Name: _____________________________</th>
<th>Address: ____________________________</th>
</tr>
</thead>
<tbody>
<tr>
<td>Your Date of Birth: ____________________________</td>
<td>Phone: _____________________________</td>
</tr>
<tr>
<td>Baby’s Date of Birth: ____________________________</td>
<td>Date: ________________________________</td>
</tr>
</tbody>
</table>

As you are pregnant or have recently had a baby, we would like to know how you are feeling. Please check the answer that comes closest to how you have felt **IN THE PAST 7 DAYS**, not just how you feel today.

Here is an example, already completed.

I have felt happy:  
a. Yes, all the time  
\(\text{D}\) Yes, most of the time  
c. No, not very often  
d. No, not at all

This would mean: “I have felt happy most of the time” during the past week.

Please complete the other questions in the same way.

---

**In the past 7 days:**

1. I have been able to laugh and see the funny side of things  
a. As much as I always could  
b. Not quite so much as before  
c. Definitely not so much now  
d. Not at all as usual

2. I have looked forward with enjoyment to things  
a. As much as I ever did  
b. Rather less than I used to  
c. Definitely less than I used to  
d. Hardly at all

3. I have blamed myself unnecessarily when things went wrong  
a. Yes, most of the time  
b. Yes, some of the time  
c. Not very often  
d. No, never

4. I have been anxious or worried for no good reason  
a. No, not at all  
b. Hardly ever  
c. Yes, sometimes  
d. Yes, very often

5. I have felt scared or panicky for no very good reason  
a. Yes, quite a lot  
b. Yes, sometimes  
c. No, not much  
d. No, not at all

6. Things have been getting on top of me  
a. Yes, most of the time I haven’t been able to cope at all  
b. Yes, sometimes I haven’t been coping as well  
c. No, most of the time I have coped quite well  
d. No, I have been coping as well as ever

7. I have been so unhappy that I have had difficulty sleeping  
a. Yes, most of the time  
b. Yes, sometimes  
c. Not very often  
d. No, not at all

8. I have felt sad or miserable  
a. Yes, most of the time  
b. Yes, quite often  
c. Not very often  
d. No, not at all

9. I have been so unhappy that I have been crying  
a. Yes, most of the time  
b. Yes, quite often  
c. Only occasionally  
d. No, never

10. The thought of harming myself has occurred to me  
a. Yes, quite often  
b. Sometimes  
c. Hardly ever  
d. Never

---


Women who experience a mild form of depression can improve with changes to their lifestyle that increase their body’s ability to cope with the stresses. Lifestyle changes that will help women cope are the first line of defence:

- Maintain proper nutrition by eating healthily and drinking more water.
- Avoid all alcohol: alcohol is a depressant and is harmful to the developing baby.
- Get regular exercise; this produces endorphins (mood enhancers) that help combat fatigue, lethargy and sleep disturbances.
- Maintain a regular sleep-wake pattern as much as possible.
- Use stress management techniques such as yoga, relaxation exercises, time management skills, etc.
- Talk to a trusted friend or family member about things of concern. Webster et al (2008) found that social support of the mother during and after pregnancy reduces the risk of postpartum depression.

- Join a support group for moms (with or without depression) - talking with other mothers enables the woman to learn from their experiences and feel less overwhelmed.
- Avoid making any major life changes during pregnancy or right after giving birth. Major changes can cause unneeded stress. Sometimes big changes can’t be avoided. When big changes cannot be avoided, try to arrange support and help in the new situation ahead of time.

These strategies alone may not be enough to recover from depression. Women may need psychotherapy and/or medication. A combination of both is usually best.

### Detecting Postnatal Depression using the Edinburgh Postnatal Depression Scale (EPDS)

“The EPDS was found to have satisfactory sensitivity and specificity, and was also sensitive to change in the severity of depression over time. The scale can be completed in about 5 minutes and has a simple method of scoring”.1

The 10-question Edinburgh Postnatal Depression Scale (EPDS) is a valuable and efficient way of identifying patients at risk for perinatal depression. The EPDS is easy to administer and has proven to be an effective screening tool.

- Mothers who score above 13 are likely to be suffering from a depressive illness of varying severity.
- The EPDS score should not override clinical judgment. A careful clinical assessment should be carried out to confirm the diagnosis.
- The scale indicates how the mother has felt during the previous week.
- In doubtful cases, it may be useful to repeat the tool after 2 weeks.
- The scale will not detect mothers with anxiety neuroses, phobias or personality disorders.

### Instructions for using the Edinburgh Postnatal Depression Scale:

1. The mother is asked to check the response that comes closest to how she has been feeling in the previous 7 days.
2. All the items must be completed.
3. Care should be taken to avoid the possibility of the mother discussing her answers with others (Answers come from the mother or pregnant woman).
4. The mother should complete the scale herself, unless she has limited English or has difficulty with reading.

### SCORING

**QUESTIONS 1, 2, & 4 (Without an *)

Are scored 0, 1, 2 or 3 with ‘a.’ scored as 0 and the ‘d.’ scored as 3.

**QUESTIONS 3, 5 - 10 (Marked with an *)

Are reverse scored, with ‘a.’ scored as a 3 and ‘d.’ scored as 0.

Maximum score: 30

Possible Depression: 10 or greater

Always look at item 10 (suicidal thoughts)

Users may reproduce the scale without further permission, providing they respect copyright by quoting the names of the authors, the title, and the source of the paper in all reproduced copies.

- EPDS - Online version available from http://psychology-tools.com/epds/


Table 1: Effects of exposure to SSRIs, SNRIs, and other antidepressants during pregnancy

<table>
<thead>
<tr>
<th>Medication</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>fluoxetine</td>
<td>Exposure is not associated with increased teratogenic effects in humans, but perinatal effects of third-trimester exposure have been reported. Earlier studies indicated minor malformations in the neonates. However, a study of 55 preschool children exposed to fluoxetine in utero reported no long-term adverse effects with respect to IQ, language, or behaviour. A recent follow-up study of 40 children exposed in utero and followed for 15–71 months also reported no effect on cognition, language, or temperament.</td>
</tr>
<tr>
<td>(Prozac)</td>
<td></td>
</tr>
<tr>
<td>paroxetine</td>
<td>One case series involving 97 exposures to paroxetine in pregnancy did not indicate any increased risk of major malformations. One recent report indicates temporary neonatal respiratory distress associated with late third-trimester paroxetine use in 12 infants, which resolved with no residual effects.</td>
</tr>
<tr>
<td>(Paxil)</td>
<td></td>
</tr>
<tr>
<td>fluvoxamine</td>
<td>A case study of 26 infants exposed to fluvoxamine in utero reported that exposure was not associated with increased risk of malformations, lower birth weights, or younger gestational age.</td>
</tr>
<tr>
<td>(Luvox)</td>
<td></td>
</tr>
<tr>
<td>sertraline</td>
<td>A case study of 147 infants exposed to sertraline in utero reported that exposure was not associated with increased risk of malformations, lower birth weights, or younger gestational age.</td>
</tr>
<tr>
<td>(Zoloft)</td>
<td></td>
</tr>
<tr>
<td>citalopram</td>
<td>A review of 375 cases of citalopram exposure in early pregnancy found that the rate of congenital anomalies was no higher than that for other SSRI exposures or for the general population.</td>
</tr>
<tr>
<td>(Celexa)</td>
<td></td>
</tr>
<tr>
<td>venlafaxine</td>
<td>150 women exposed to venlafaxine during the first trimester were monitored. No adverse effects in infants were noted.</td>
</tr>
<tr>
<td>(Effexor)</td>
<td></td>
</tr>
<tr>
<td>mirtazapine</td>
<td>Two case studies of pregnancies with early exposure to mirtazapine reported no adverse effects. In another study, seven women exposed to mirtazapine in the first and second trimester delivered healthy babies.</td>
</tr>
<tr>
<td>(Remeron)</td>
<td></td>
</tr>
<tr>
<td>bupropion</td>
<td>There are no human data published, although a bupropion registry exists.</td>
</tr>
<tr>
<td>(Wellbutrin)</td>
<td></td>
</tr>
</tbody>
</table>

Table 2: Effects of exposure to SSRIs, SNRIs, and other antidepressants during lactation

<table>
<thead>
<tr>
<th>Medication</th>
<th>Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>fluoxetine</td>
<td>Low levels of fluoxetine and its main metabolite, norofluoxetine, found in the sera of infants. Some short-term adverse effects have been reported, including colic, seizures, irritability, withdrawal symptoms, and cyanosis.</td>
</tr>
<tr>
<td>(Prozac)</td>
<td></td>
</tr>
<tr>
<td>paroxetine</td>
<td>Undetected or very low levels of paroxetine found in the sera of infants. No adverse effects have been reported.</td>
</tr>
<tr>
<td>(Paxil)</td>
<td></td>
</tr>
<tr>
<td>fluvoxamine</td>
<td>Low levels of fluvoxamine found in the sera of all infants exposed through breast milk. No adverse effects have been reported.</td>
</tr>
<tr>
<td>(Luvox)</td>
<td></td>
</tr>
<tr>
<td>sertraline</td>
<td>Low levels of both sertraline and its metabolite found in the sera of infants. One study reported that sertraline was significantly more likely to be detected in an infant if the maternal daily dose was 100 mg or higher. No adverse effects have been reported.</td>
</tr>
<tr>
<td>(Zoloft)</td>
<td></td>
</tr>
<tr>
<td>citalopram</td>
<td>Low levels of citalopram and its metabolite found in all studies. Only one adverse effect, uneasy sleep, has been noted in one study.</td>
</tr>
<tr>
<td>(Celexa)</td>
<td></td>
</tr>
<tr>
<td>venlafaxine</td>
<td>In 12 documented cases of exposure to venlafaxine, no adverse effects in infants have been reported.</td>
</tr>
<tr>
<td>(Effexor)</td>
<td></td>
</tr>
<tr>
<td>mirtazapine</td>
<td>No human data published.</td>
</tr>
<tr>
<td>(Remeron)</td>
<td></td>
</tr>
<tr>
<td>bupropion</td>
<td>Peak effects found 1-2 hours after dose in three case studies.</td>
</tr>
<tr>
<td>(Wellbutrin)</td>
<td></td>
</tr>
</tbody>
</table>
Medication during pregnancy

Many health care providers are hesitant to prescribe anti-depressant medication during pregnancy and pregnant mothers are reluctant to take them for fear of harmful effects on the baby. Research on the short-term effects of antidepressant medications, particularly the selective serotonin reuptake inhibitors (SSRIs) and serotonin norepinephrine reuptake inhibitors (SNRIs), suggests that they can be used by pregnant and breastfeeding women (Misri et al 2005). This group compiled a list of antidepressants and their known side-effects.

The group also give some guidelines for the management of medicines in the perinatal period:

1. Watch for recurrent depression leading to relapse during perinatal period.
   - If medications are discontinued upon conception and relapse occurs, reinstate.
   - Use a single medication rather than multiple medications.
   - Do not switch medications if current regimen is effective.
   - Make sure communication lines are open between psychiatrist and obstetrician.
   - Make sure all health care providers (primary care physician, midwife, nurses, etc.) are aware of the diagnosis and treatment plans.

2. Monitor breast milk and infant serum levels of maternal medications or metabolites thereof.


   - Follow patient for at least 1 year after remission (longer if breastfeeding, or mood cycling).
   - Discuss future pregnancies and risk of recurrence.
   - Ensure adequate follow-up in the community.

Psychotherapy is necessary for the issues underlying the depression, for instance the first-time mother who is feeling overwhelmed by the responsibility of having a baby, or who has lost a previous baby. Difficult home situations can also be worked through. Gaining insight into these issues enables the mother to develop support and gain inner strength to deal with her new baby when it arrives.

Conclusion

Depression in pregnancy is a condition not to be taken lightly! Mothers-to-be and new mothers need a good support system in place to reduce the risk of developing depression. But once the professional suspects a mother has depression, every effort should be made to diagnose the severity of the depression and to remedy the underlying reasons with psychotherapy. Where necessary, the appropriate medication should relieve the depression and allow the mother to cope better and the baby to thrive.

REFERENCES AVAILABLE ON REQUEST
Presents a Five-Day Advanced Course in Diabetes Care for Health Professionals 2015

DIABETES ~ THE BURDEN, THE RELIEF

Latest estimates place the prevalence of Diabetes Mellitus in South Africa at 8.4 % (International Diabetes Federation, 2014) and this is increasing. 50-85 % of persons with the condition are undiagnosed and at risk from disabling and life threatening complications. Diabetes, together with its associated cardiovascular risk factors is one of the leading causes of death, either directly or indirectly, in our population.

Over the past three decades, it has become evident that good control of diabetes, as well as the common co-morbidities of hypertension and the dyslipidaemias, is vital to prevent or delay the devastating long-term complications of diabetes. To achieve this, people with diabetes need to understand their largely silent condition and the correct principles of self-care.

Health professionals often do not have access to updated approaches to a chronic, mostly self-managed condition such as diabetes ~ vital opportunities for therapeutic interaction and patient education are lost. Additionally, insight is needed into the ever-widening range of available medications and treatment strategies as well as the relationships between cardiovascular and other risk factors and diabetes.

As health services evolve, there is a move towards Team Management of Chronic Conditions. This has resulted in the rest of the Health Care Team (Nurses, Pharmacists, Dieticians, Podiatrists, Biokineticists and others) playing an ever-increasing role in diabetes care.

WHO SHOULD ATTEND THE COURSE?

This is an Advanced Course, and is aimed at Health Care Professionals who have a basic knowledge and understanding of diabetes mellitus. It is designed to give an extensive overview of the core principles of modern team diabetes management, so enabling the participants to understand the condition in sufficient depth, to make a real difference in the lives of people with diabetes. Pre and Post Course multiple-choice knowledge evaluation tests are administered, to allow for evaluation of the learning experience.

Attendance is also part of the contractual requirements for Practitioners wanting to open CDE affiliated “Centre for Diabetes Excellence” Branches.

CPD ACCREDITED

The Course offers 34 contact hours. The Course is accredited to provide 30 CPD points for Medical Practitioners and other Healthcare Practitioners registered with the Health Professions Council of South Africa.

Pre-Course readings will be supplied by e-mail to all delegates and an electronic manual of all speaker notes will be provided on the first day of each Course.

Official completion certificates will be provided to delegates who achieve a mark of at least 60 % in the final Post-Course Knowledge Evaluation.

COMMENTS FROM DELEGATES TO PREVIOUS 5-DAY COURSES:

I realise that I had been blundering around in the dark in treating my patients with diabetes and now someone has turned on the light! This a life changing Course. You have reformed my medical practice forever - General Practitioner
It was a superb Course & should result in a marked improvement in the care of people with diabetes - Registered Nurse

I enjoyed the Course thoroughly. I will manage patients with diabetes with more self-confidence. The talks were excellent, well organized and well presented - Registered Dietician

The message that you convey is that you care. The variety of topics was great. The balance between active participation and listening was great. The great teaching skills in all lectures promote learning - Registered Nurse

All speakers were excellent and displayed an impressive knowledge of their subjects. Your commitment as professionals is highly commendable. I learned a lot from this superb Course. Consequently, I will be able to treat my patients better - Medical Specialist

ANSWERS TO FREQUENTLY ASKED QUESTIONS

Presented at: Glenhove Conference Centre, 52 Glenhove Road, Melrose Estate, Johannesburg (Please note change).

Dates & Fees: Available at www.cdecentre.co.za (Click on CDE Education). Early bookings are advised.

Course Hours: Five days of lectures, workshops and discussion (08h00 – 17h00).

Dress: Comfortable, smart-casual

Language Medium: English

Course Information: The Course Coordinator

Tel: +27 11 053-4400 / Fax: +27 (0)86 247-0674

E-mail: John@cdecentre.co.za

PROGRAMME SUMMARY

The Course is aligned with the latest evidence based treatment guidelines. Case studies and problem solving approaches are a vital part of the learning process.

TOPICS INCLUDE:

- Holistic Team Care Philosophy & Educational Approaches
- Diagnosis, Classification, Pathophysiology & Prevention of Type 1 & Type 2 Diabetes Mellitus
- Other types of diabetes including Gestational Diabetes
- Treatment of Type 1 & Type 2 Diabetes
- Psychological Adjustment to Diabetes
- Meal Planning & Nutrition in Diabetes
- The Importance of Exercise in Diabetes
- The Medical Management of Diabetic Ketoacidosis
- The Foot of the Person with Diabetes
- Acute Complications of Diabetes
- Diabetes as a Micro- & Macro-vascular Disease & Risk Factor Control
- Managed care in diabetes
- Interactive Team-facilitated Case Study Sessions
- Practical Workshop on Self Care Devices & Equipment

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Diabetes Specialist Nurse

Rosemary Flynn
MSc (Clinical Psychology)
Clinical Psychologist
Currently, we are witnessing an alarming increase in diabetes in South Africa, both in the young and in adults, regardless of background, ethnicity or age.

Many people with diabetes are not aware of the best approach for their diabetes care...

Neither are many of their healthcare providers...

The CDE provides advanced, continuing education, mentorship and accreditation in diabetes care to members of the wider diabetes team.

To find out more about our Healthcare provider training and support, please contact the CDE Provider Network Liaison Department on 011 712-6000 or e-mail Providers@cdecentre.co.za

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