Cardiovascular diseases are an equally common cause of death, and a more common cause of long-term disability, in women compared with men. This has been poorly recognised and unfortunately very few women were included in the landmark cardiovascular treatment trials of the late 20th century and the early 21st century. Until now there has been a paucity of reliable information about potentially important sex differences in cardiovascular diseases. It is now understood that the commonly used risk calculators, based on Framingham risk scores, significantly underestimate cardiovascular risk in individual women, especially those with multiple risk factors.1

Some important information about coronary disease in women has arisen recently from the WISE Study.2 Firstly, using intracoronary ultrasound, it has been demonstrated that many women have more extensive coronary atherosclerosis than demonstrated using conventional coronary angiography. This seems to be due to a more diffuse distribution of soft non-calcified plaque in women which can give the appearance of “normal” (though perhaps small diameter) coronary arteries.3 In women there seems to be an increased predilection to superficial erosion of these plaques (rather than the localised acute plaque rupture typically seen in the setting of ST elevation myocardial infarction) and this may lead to distal microembolisation and contribute to impaired microvascular coronary flow.4,5 Sex related differences in thrombogenicity and propensity to inflammation may play a role in this process.

Continued on page 3

Model of Microvascular Angina in Women1

### DR MELISSA DOOHAN MBBS (HONS) PHD FRACP DDU

Dr. Melissa Doohan is an experienced cardiologist who is interested in the full spectrum of diagnostic and clinical cardiology. She completed her combined medical degree at University of Sydney and then undertook basic and advanced physician’s training at Royal North Shore Hospital. She was subsequently awarded a PhD in the field of cardiac cellular physiology. Dr Doohan currently consults in the San Clinic in Wahroonga and in North Shore Cardiac Centre in St Leonards.
I am often asked how my background in portraiture changes my approach to rhinoplasty. Essentially artists look at the reflection of light on form, as well as the object itself. As rhinoplasty surgeons, we are taught angles and measurements but far more important in achieving a natural outcome is to be able to create a normal light reflex. This light reflex is seen along the brow tip aesthetic line, i.e. from eyebrow, down the lateral edge of the bridge of the nose and into the tip region. Portrait artists over the centuries have emphasised this reflection of light as it outlines the change in plane, e.g. from the flat area over the roof of the bridge of the nose to where it joins the slanted side walls of the nose. This change should be gradual yet definite.

Until the early 1990s, rhinoplasty was largely performed via the nostrils. After removing the nasal hump, the side walls of the nose lost support, collapsing inwards over time (the inverted “V” deformity). This disrupted the brow tip aesthetic line/light reflex making the nose eventually look operated on and unnatural.

Since the early 1990s, the open technique (surgical entry via an incision between the nostrils) has gained increasing popularity as it allows better access to insert spreader grafts to support the side wall of the nose (see Fig. 1 and 2a – 4b).
The three quarter view of the patient allows for the best demonstration of the light reflex. Most patients focus on the frontal or profile view but in fact we are rarely addressed at this angle. Most people will hold us in conversation or photograph us from the three quarter view. Therefore, this is the view we need to focus on as rhinoplasty surgeons. Get the light reflex right, and you will give the nose grace. Not surprisingly most portraits are painted from this angle as well. It is the most aesthetic.

The brow tip aesthetic line/light reflex can be disrupted by trauma including previous surgery (Fig. 5a – 5b). As stated above, spreader grafts are our most valuable tool to prevent collapse. They will also improve the airway. These and other camouflage grafts are harvested from the nasal septum or occasionally from the ear, as it has similar cartilage. These grafts can transform a twisted nose into one with a normal light reflex, restoring a natural appearance.

Age related changes can lengthen the brow tip aesthetic line. Years can be removed from the facial appearance by restoring the natural tip rotation i.e. shortening the brow tip aesthetic line (Fig. 6a – 6b).

Most patients presenting for rhinoplasty simply want a nose which functions well and looks natural. In fact, one that nobody notices. This is best achieved by focusing not on angles but on light reflexes. If the light reflex is normal, it will photograph well and in this digital age that equals success.

Women with angina-type symptoms and an absence of haemodynamically significant coronary artery disease (sometimes referred to as having “Syndrome X”) often have evidence of subendocardial ischaemia on modern myocardial imaging e.g. MRI or PET scanning. Although we know that women can experience intermittent epicardial coronary artery spasm which may sometimes account for the ischaemia, it is becoming increasingly clear that it often relates to microvascular dysfunction. The exact relationship between epicardial coronary atherosclerosis and microvascular dysfunction remains unclear, but microvascular dysfunction can result from an imbalance between endothelially derived vasodilator substances (such as nitric oxide) and an increased propensity to vasoconstriction in response to certain stimuli (such as increased heart rate). Invasive coronary reactivity testing can be used to diagnose these abnormalities.

Unfortunately, this type of testing is not without risk and optimal methods and appropriate diagnostic criteria have not yet been defined. Whilst studies have previously suggested that women with “Syndrome X” do not have increased cardiovascular mortality due to myocardial infarction, the WISE investigators have demonstrated that they have higher rates of composite cardiac events over 5 years including hospitalisations for heart failure and strokes. Those women with “Syndrome X” who also had demonstrable myocardial ischaemia, presumably from microvascular dysfunction, had a greater frequency of adverse cardiovascular events, even after accounting for conventional coronary risk factors.

At present, apart from managing women with macrovascular coronary artery disease along conventional lines, results from the WISE Study suggest we should also consider aggressive management of risk factors in symptomatic women without haemodynamically significant coronary disease, especially in those with multiple risk factors, demonstrable myocardial ischaemia or poor functional capacity on stress testing. Ongoing and future studies may guide us further in preventing cardiovascular diseases in the female population and define a place for newer more targeted therapies in affected women.

References available on request

CARDIOVASCULAR DISEASE: IMPORTANT GENDER DIFFERENCES Continued from page 1
PREGNANCY IN OBESE WOMEN AND AFTER GASTRIC BANDING

Dr Kris Urbaniak

The most recent official statistics put the number of Australians with excessive weight at an alarming 63%, and 50% applies to pregnant women: 30% are overweight and 20% obese. This is a rather sober world record, and doctors providing antenatal care need to be prepared to deal with a problem of this magnitude, as those women require complex care and often have numerous co-morbidities.

Various pregnancy complications are increased in overweight women: miscarriage, pre-term labour, pre-eclampsia, gestational diabetes, prolonged labour, instrumental and caesarean delivery, postpartum haemorrhage, infection, wound breakdown and DVT. Their babies have more macrosomia but also IUGR, birth trauma, and admission to SCBU. The fetal demise rate is doubled. To close the circle, children of overweight mothers are more likely to be overweight in childhood and as adults, with accompanying type II diabetes, hypertension, and cardiovascular disease.

Maternal weight normalisation before pregnancy can reduce most of those risks and should be addressed at the preconception visit. In addition to high-intensity counselling, nutritional education, exercise and behavioural strategies, it is worth making the point that there is dose-response between degree of obesity and adverse outcomes of pregnancy, and even a modest weight reduction is beneficial.

Weight-loss modifying medication can be added at BMI>30 or BMI>25 with co-morbidities. Bariatric surgery can be considered at BMI>40 or BMI>35 with co-morbidities, which should be screened for: diabetes, hypertension, cardiovascular disease, dyslipidaemia, thyroid disease, musculoskeletal problems, biliary tract disease, sleep apnoea and depression. Subfertility affects 18% of obese multiparous and 36% of nulligravid women, and weight loss should precede fertility treatment.

Whether following an aggressive weight reduction program, or bariatric surgery, pregnancy should be avoided during a rapid weight loss phase because of the expected state of caloric and nutritional deficits. With laparoscopic adjustable gastric banding (LAGB), the procedure most popular in Australia, this phase usually lasts 12 months. Liaison with a bariatric surgeon is paramount.

The first trimester pregnancy consultation is extremely important. Again, thorough examination and tests are performed to identify obesity related complications. The whole course of pregnancy care is then carefully planned and appropriate consultations arranged.

The weight gain targets are established primarily to prevent excessive weight gain. The following modified WHO guidelines should be helpful:

<table>
<thead>
<tr>
<th>BMI</th>
<th>CLASSIFICATION</th>
<th>RECOMMENDED WEIGHT GAIN</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;18.5</td>
<td>Underweight</td>
<td>13 – 16kg</td>
</tr>
<tr>
<td>18.5 – 24.9</td>
<td>Normal range</td>
<td>11.5 – 16kg</td>
</tr>
<tr>
<td>25 – 29.9</td>
<td>Overweight</td>
<td>7 – 11kg</td>
</tr>
<tr>
<td>30 – 34.9</td>
<td>Obese class 1</td>
<td>5 – 9kg</td>
</tr>
<tr>
<td>35 – 39.9</td>
<td>Obese class 2</td>
<td>0 – 4kg</td>
</tr>
<tr>
<td>40 and above</td>
<td>Obese class 3</td>
<td>0% lose up to 4kg</td>
</tr>
</tbody>
</table>

The available data suggest that over 30% of overweight/obese pregnant women receive no weight gain information at all! Another area not widely addressed is exercise, yet obese women are likely to become even less active in pregnancy and need ongoing advice and support. One should reinforce both lifestyle physical activity changes (like stairs not lift etc.), and structured exercise. The aim is not to achieve peak fitness but to maintain some fitness, prevent excessive weight gain and help to reduce insulin resistance. Strenuous programs are avoided, the best is to engage in low impact aerobic exercises involving large muscle groups, 30 mins daily. Walking, swimming, aqua aerobics, and stationary cycling with a target HR of 110-131 are recommended, while exercises in supine position or involving Valsalva maneuver may be dangerous. Weight loss should be avoided as it causes fetal growth restriction and ketonemia. Data from obese diabetic women show that infants exposed to ketonemia in utero have a lower IQ. If the woman had the LAGB procedure, the band may need adjustment to allow for some limited weight gain.

Obesity interferes with ultrasound imaging which is best performed in specialist clinics with ample expertise. Establishing EDD by early U/S is paramount to management of advanced pregnancy; expert morphology scan is necessary as there is higher incidence of congenital abnormalities, especially NTDs (OR 1.9). There is need for ongoing ultrasound monitoring of fetal growth and wellbeing for the length of those high risk pregnancies. Moreover, physical examination is often difficult, even checking fetal position and FHR may be problematic. While official guidelines are lacking, prudence dictates increased fetal supervision in 3rd trimester as fetal demise is more common, particularly after 40 weeks (OR 4.6). All efforts should be made to aim at vaginal birth but C/S may be recommended for EFW of 5000g, or 4500g when the baby is the last weeks of pregnancy and labour, including any intervention should be individualised and facilitated by a specialist with relevant clinical interest.

The incidence of all postpartum complications is higher in obese women and unfortunately includes lactation problems in dose-response relation with BMI. Women with BMI>40 are least likely to breastfeed yet they and their infants are the ones who would most benefit from it. Management of lactation issues should include all members of the perinatal care team. One of main causes of female obesity is retention of pregnancy weight gain and successful lactation helps with weight loss. Another barrier to return to pre-pregnancy weight is further reduction in mobility postpartum; this is a complex issue in obese woman and can be permanent. Our efforts should be directed at reinforcing daily physical activities and in fact increasing structured exercise.

Finally, contraceptive advice: there is no evidence of increased failure rate of COC in obese women or that it causes BMI increase. However, there is increased risk of DVT, lowest with COC containing levonorgestrel so those should be preferred if the pill is chosen, vaginal rings release 15mcg ethinylestradiol daily, less than lowest oral COC so they are reasonable choice. IUCDs, medicated or copper, are recommended. If a permanent method is considered a definite consideration should be given to vasectomy.

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Dr Kris Urbaniak is a senior Obstetrician and Gynaecology consultant practicing at SAH and Hornsby Hospitals. His interests include high risk pregnancy, prolapse surgery, ultrasound in O&G and menstrual disorders.

His interests include high risk pregnancy, prolapse surgery, ultrasound in O&G and menstrual disorders.
Alcohol is one of the most widely used social drugs in the world. The average Australian adult consumes 10.3L of ethanol annually equivalent to 783 beers (Australian Bureau of Statistics, 2011, WHO, 2011). While most Australians use alcohol responsibly the growing problem of alcohol abuse costs the Australian government over $25 billion annually and claims 2.5 million lives worldwide each year (WHO, 2011).

Alcohol (ethanol) is a central nervous system (CNS) depressant. While able to produce the desirable short term social effects of disinhibition and euphoria, exposure to ethanol is associated with increased risk of dementia and cancers such as mouth, pharynx, larynx, oesophagus, breast, bowel and liver (Australian Cancer Council 2012). In fact the Cancer Council of Australia stated that: “Any level of alcohol consumption increases the risk of developing alcohol-related cancer [and] the level of consumption increases the risk of developing other health problems such as weight gain, cirrhosis of the liver and stroke. How does ethanol cause this range of health problems? A growing body of evidence is showing that ethanol increases reactive oxygen species (ROS) or ‘free radical’ production resulting in a state of ‘oxidative stress’ causing significant damage to cellular components (Figure 1).

The rate at which damage is caused is determined by how fast the free radicals are generated and then detoxified by the body’s defense antioxidants. Alcohol (ethanol) it seems is able to dramatically increase free radical production not only in front line tissues like the liver but also in the cells of the brain. Research at the Australasian Research Institute (ARI) at Sydney Adventist Hospital has shown for the first time that free radical production increases significantly in the metabolic support cells of the brain, (i.e. astroglia), within 30 min; even when exposed to concentrations equivalent to a blood alcohol level of 0.005% (1mM). This is 10 fold lower than the legal limit (Figure 2)!

At this level of exposure oxidative stress increased by 145% resulting in decreased normal cell growth and increased DNA repair enzyme activity, indicating significant DNA damage. The ARI research team were also the first to show that exposure to blood alcohol levels of 0.05% (10mM) for 30 minutes markedly decreased (> 40%, see Figure 3) concentrations of the essential molecule NAD(H). NAD(H) is needed for energy production, DNA repair and normal activity of the ‘longevity’ enzymes called sirtuins. This decrease in NAD(H) therefore resulted in decreased activity of the ‘longevity’ enzyme sirt1. Importantly low levels of sirtuin activity have been linked to accelerated aging (Bonda et al 2011) While it is recognised that ethanol is largely consumed for its psychosocial effects, as it has no nutrient value it would seem wise to reduce as far as possible the amount of alcohol that is consumed, if optimal health is to be preserved, particularly in the brain.

**References**
Oysten L. Honours Thesis, School of Medical Sciences University of NSW, Sydney 2012
The Sleeve Gastrectomy is a relatively new option in the treatment of morbid obesity and related co-morbidities and is rapidly gaining popularity both in Australia and abroad. It is a simple and effective procedure, more reliable than the gastric band and with fewer side effects than the gastric bypass. Clinicians may wish to familiarise themselves with this increasingly common bariatric operation.

**HISTORY**
The Sleeve Gastrectomy was first performed as an independent bariatric procedure by Dr Michel Gagner in New York in 2002. Previously part of a bypass procedure known as the duodenal switch, it began being performed as a stand-alone procedure when a group of high risk patients underwent the Sleeve (as the first stage of an intended 2-stage procedure) and achieved surprisingly good results. Since then it has been refined and standardised, and has become widely accepted as an effective treatment for obesity.

**WHAT’S INVOLVED**
The Sleeve Gastrectomy is a laparoscopic procedure, and involves dividing the stomach parallel to the lesser curve to create a tubular neo-stomach approximately 25cm long and 2cm diameter. (Figure 1) It differs significantly from the old stomach stapling operations of the past, in that the unwanted stomach, approximately 80% of the original volume, is removed. The elegant aspect of this procedure is that the small bowel, and therefore absorption, is left unaffected. Further, unlike the Gastric Band, there are no device related problems.

The length of hospital stay averages 3 days, and 2 weeks time off work is sufficient.

**HOW DOES IT WORK?**
The Sleeve Gastrectomy broadly works in 3 ways:

1. Major gastric capacity reduction: Stomach capacity is reduced by at least 80%. Patients feel full and satisfied by the equivalent of just a cup of food.

2. Reduction in hunger hormones: The hunger hormone Ghrelin, which is secreted by the stomach, is reduced by 50-65%. Patients report definite and durable hunger reduction.

3. Dumping effects to liquids high in sugar and/or fat. As the small tubular stomach directs the food stream directly towards the duodenum, liquid or soft calories such as milkshakes, soft drinks, ice-cream, and chocolate overcome the pylorus and enter the duodenum soon after consumption. This leads to bloating, crampy discomfort, and light-headedness and quickly results in improved dietary choices.

**HOW EFFECTIVE IS IT?**
Because the Sleeve cannot be as easily ‘cheated’ with liquid calories in the way the Band can, the weight loss results are much more consistent. The average patient will lose approximately 70-80% of their excess weight within 1 year, and importantly, almost all patients lose at least 50% excess weight. This translates into high rates of resolution of co-morbidities, especially for diabetes (90% improved, 50% resolved), sleep apnoea, (85% improved, 40% resolved), hypertriglyceridaemia (85% improved), and hypertension (75% improved, 40% resolved). Major improvements occur in quality of life, especially musculoskeletal pain, energy levels, and self-esteem.

**WHAT ARE THE RISKS?**
The Sleeve Gastrectomy is considered a safe procedure, with a risk of complications somewhere between those of the Band and Bypass procedures. The main specific complications of the Sleeve relate to the long staple line: either leak or bleeding may occur in 1-2% of patients. The risk of other general complications such as venous thromboembolism is similar to other abdominal procedures.

**ARE THERE ANY LONG TERM ISSUES?**
Over time the capacity of the neo-stomach is expected to gradually increase, however the extent, and possible effect this may have on weight regain remains unclear. It is vitally important that patients adopt improved dietary patterns, exercise and a healthier overall lifestyle in conjunction with any bariatric procedure in order to achieve long term weight and co-morbidity control. Regular and ongoing support from a multidisciplinary team are crucial for this.
Cervical disc replacement, also known as cervical disc arthroplasty (CDA), is now freely available to private patients in Australia. MBS funding was initially removed in 2006, meaning patients had to pay for the cost of the prosthesis themselves. Even though CDA had been approved by the FDA in July 2007, Medicare deemed there to be a lack of level 1 evidence at the time. In January 2013, the latest prostheses list was published and included several CDA models which have been in clinical use in Australia for over two years.

Cervical degenerative disc disease is a common pathological condition that may cause neck pain, shoulder and arm pain, sensory loss, motor weakness or headache. Most patients respond well to conservative treatment options, such as physiotherapy, traction and analgesic or anti-inflammatory medication. Surgical treatment can be considered when conservative treatment fails and can stabilise or improve in over 90% of patients. Primary goals of surgical intervention are to relieve pain and to prevent progression of neurological deficit. While surgery is accepted in cases of radiculopathy or myelopathy, controversy exists in cases of axial neck symptoms, but CdA may have the added advantage of preventing future degeneration. Currently, there is much debate about whether fusion is a contributing factor to ADD, or whether it is simply the result of natural progression of degeneration in an aging spine. However, the ever more widely held belief that increased stress at adjacent segments accelerates their degeneration has led to the development of a non-fusion operation for preserving motion and decreasing stresses.

Our vertebrae normally move forward, backwards and side-to-side, as well as rotate. These types of movements travel in either of two directions: translational or rotational. Translational planes have three types or “degrees” of movements (forward/backward, side-to-side, and up/down). The rotational axes have three degrees of movement as well (rotating, forward/backward bending, and side to side bending). When the spine is completely free to move in all these directions, it is said to have 6 degrees of freedom (3 degrees in translation and 3 degrees in rotation). The M6 disc (pictured) is an example of an unconstrained artificial disc allowing movement in all these planes, mimicking the function of a natural disc.

In the last 10 years CDA technology has been greatly improved. A number of devices are currently available, each varying in terms of materials and range of motion. In theory, CDA should reduce the likelihood of developing adjacent segment degeneration by maintaining normal disc kinematics. In biomechanical cadaveric studies, CDA has also been shown to maintain motion and mechanics within physiologic ranges at the index segment and decrease stresses on adjacent segments.

CDA and fusion appear to be equally effective in resolving pre-operative symptoms, but CDA may have the added advantage of preventing future symptoms by maintaining mobility. CDA may be more suitable in younger patients with generally less degenerative change in the spine. It is less suitable in certain ossifying conditions of the spine, trauma or rheumatoid arthritis.

References

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Mr Sergides is a Consultant Neurosurgeon on the North Shore of Sydney who treats conditions of the brain, spine and peripheral nerves. He is dually qualified and has Fellowships of the Royal Australasian College of Surgeons and of the Royal College of Surgeons England. Rooms telephone: 9496 2050 Web: sydneyspinesurgeon.com
**NEWLY ACCREDITED DOCTORS**

**Dr Kumud Dhital**  
BSc BM BCH FRCS (Eng) FRCS-CTh PhD FRACS  
Cardiothoracic Surgeon

Dr Dhital studied at Oxford University and the University of London. He has worked as Assistant Professor of Surgery and Director of Cardiopulmonary Transplantation & Mechanical Assistance for the University of Pittsburgh (USA) and as a consultant at Papworth Hospital Cambridge as Director of Lung Transplantation. He provides a comprehensive adult cardiothoracic service and has established a program for pulmonary endarterectomy inpatients with chronic thromboembolic pulmonary hypertension. He is a Senior Lecturer in the Faculty of Medicine at UNSW. Contact 8382 3069.

**Dr Fiona Foo**  
MBBS (Hons) FRACP  
Interventional and General Cardiologist

After training at the University of Western Australia, Concord Hospital and Fremantle Hospital, Dr Foo completed Interventional Cardiology fellowships in The Mazankowski Alberta Heart Institute in Edmonton, Canada and the Golden Jubilee National Hospital in Glasgow, Scotland. She has interests in coronary interventions via the radial approach; use of fractional flow reserve, acute coronary syndromes, cardiac risks of non-cardiac surgery after coronary revascularisation, women and ischaemic heart disease, cardiology in the developing world, and sports cardiology. Contact 9422 6000.

**Dr Charlie Lin**  
BSc (Med) MBBS FRACS (Orth)  
Orthopaedic Surgeon

Dr Lin has been trained in Sydney, at the Rubin Institute of Advanced Orthopaedics in Baltimore, Maryland, and at the Harborview Medical Center in Seattle, Washington USA. His subspecialty interest is in hip and knee replacement, reconstruction and revision surgery as well as complex trauma surgery. Contact 8039 0188.

**Dr Geoffrey CS Smith**  
MBChB MRCS (Ed) FRACS (Orth)  
Orthopaedic Surgeon - Shoulder, Elbow, Wrist and Hand

Dr Smith obtained his medical degree at the University of Leeds and began surgical training in Edinburgh receiving membership of the Royal College of Surgeons of Edinburgh in 2001. He completed Advanced Orthopaedic Training in Queensland and was the Shoulder and Elbow Fellow at the Royal North Shore Hospital before returning to the UK for the Shoulder and Elbow Fellowship at the Avon Orthopaedic Centre and subsequently the Hand and Wrist Fellowship at the Bristol Royal Infirmary. Dr Smith has an active research interest including stemless shoulder arthroplasty, shoulder resurfacing, shoulder instability and elbow hemiarthroplasty. Contact 8039 0181.

**NEWS FROM THE SAN**

2013 is the 110th anniversary of when Sydney Adventist Hospital opened as a 70 bed home of health and healing - a ‘Sanitarium’ in 1903. There will be a range of activities to celebrate this.

Additional car parking is now available on the San Campus as the Redevelopment progresses. The new main multi-deck 894 spot car park will be open from June 2013. The additional 200 beds and extra operating theatres, new maternity unit, integrated cancer centre and entry and arrivals area are on target to open from mid-2014. The New Education Centre will open later this year and will house the Sydney Adventist Hospital Clinical School of The University of Sydney and the School of Nursing of Avondale College of Higher Education.

**GP’S ARE INVITED TO ATTEND GRAND ROUNDS AT THE HOSPITAL IN 2013**

8 April  
Dr Ross Grant  
Omega 3 Interventions study with non-stemi MI patients

6 May  
Steve Crago (Pharmacist)  
Antibiotic Stewardship

4 June  
A/Prof Peter Popantoniou – Orthopaedics

24 July  
A/Prof Henry Woo – Urology

19 August  
Dr Auriel Jameson – General Medicine

15 October  
Professor Sharon Kilbreath  
Breast cancer and exercise, lymphoedema management and prevention of shoulder impairments post mastectomy.

11 November  
Professor John Watson – Neurology

Grand Rounds are held in the Level 2 Conference room from 12.30 – 13.30pm. (Light refreshments available from 12.00pm. please register on arrival).

**DIARY DATES**

**FREE PUBLIC FORUMS** (everyone welcome)

APRIL 17  
Women’s Health

SEPT 10  
Men’s Health

**GP CONFERENCES** (CPD points available with proof of attendance)

MAY 22  
Women’s Health

JUNE 5  
Urology

JULY 24  
Cardiac

AUG 21  
Emergency Medicine

OCT 17  
Onkology

Dates and topics are subject to change. Contact 9487 9871 to register or visit www.sah.org.au for further details.