MULTIPARAMETRIC MRI
OF THE PROSTATE

Dr Tom Dean

DR TOM DEAN MBBS (UNSW), FRCS (EDIN), FRACS (UROL)

Dr Dean is a consultant urological surgeon at the Sydney Adventist and Hornsby Hospitals. He has a particular interest in treatment for benign prostate hyperplasia including Green Light Laser prostatectomy, and the early diagnosis of prostate cancer including the role of prostate MRI. Other special interests include stone disease and incontinence.
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INTRODUCTION

Patients and doctors alike are confused about the need for early detection of prostate cancer which results in the death of some 3,000 men in Australia every year. Countless others suffer from the effects of metastatic cancer and its treatment even though they may eventually die from other causes. In the pre PSA era 80% of men had metastatic cancer at diagnosis. Yet early diagnosis with PSA testing is highly controversial because of concern about the risks of biopsy and curative treatment, and concern that treatment in many cases is not necessary. We clearly need to improve how to determine which patients need curative therapy.

CURRENT PRACTICE

Multiple factors determining biopsy include:
- Family and clinical history
- Digital rectal examination
- The PSA level and PSA change over time
- Prostate size.

Biopsies are currently performed under transrectal ultrasound guidance by the transrectal or transperineal route. Cancers are not usually easily visible on ultrasound so multiple random samples are required (especially when adequately sampling a large prostate) and important cancers can remain undiagnosed.

A MULTIPARAMETRIC PROSTATE MRI STUDY WILL INCLUDE:
1. T2 weighted imaging showing anatomical detail.
2. Diffusion weighted imaging (DWI) which detects movement of water molecules.
3. Dynamic contrast enhancement (DCE) with an agent such as Gadolinium.

HOW MRI CAN HELP:
1. Diagnosis:
   - help to improve staging accuracy e.g. by detecting seminal vesicle invasion
   - more accurate tumour grading
   - more accurate tumour localisation.

   Mp-MRI should be performed by an appropriately trained radiologist as part of a comprehensive urological assessment.

   The MRI must be performed correctly and reported accurately. Otherwise the study which is already expensive (as there is no rebate from Medicare) may need to be repeated at an even greater cost. In addition misleading results may compromise patient care.

   High quality studies performed by a radiologist with the appropriate experience are not widely available.

   Sydney Adventist Hospital radiologist Dr David Smit has a special interest in prostate MRI and has been trained at UMC St Radboud, Nijmegen, Netherlands under world leader Dr Jelle Barentsz in the performance and reporting of Mp-MRI of the prostate.

   In line with numerous overseas studies, our early experience at SAH has confirmed that Mp-MRI of the prostate can accurately predict the location and grade of cancers in 85-90% of cases. However it should not be considered an alternative to biopsy.

   References available on request.
THE CHANGING PARADIGM OF VASCULAR SURGERY

A/PROF Irwin V Mohan

INTRODUCTION
Atherosclerosis is a disease process affecting the large and medium-sized arteries; it is characterised by endothelial dysfunction, inflammation, and accumulation of lipids, cholesterol, calcium, and extracellular matrix within the intima of the vessel wall, and results in plaque formation, arterial obstruction, and decreased oxygen supply to end organs and muscles. Atherosclerosis is the leading cause of death in the western world. In Australia today there is an increasing elderly population with significant manifestations of atherosclerotic occlusive and aneurysmal disease.

The rapid pace of innovation and technological advancement, and the evolution of vascular surgery towards endovascular catheter-based revascularisation for all arterial disease, has fuelled the growth of endovascular procedures by further improving the safety, durability, and predictability of percutaneous intervention and revascularisation. Cost-effectiveness and quality-of-life outcomes favor the performance and safety of percutaneous therapy whenever feasible as a more effective treatment.

PERIPHERAL ARTERIAL DISEASE
Peripheral arterial disease (PAD) occurs in approximately 12% of the adult population over 50 years, and this incidence increases as one gets older. PAD is also a marker of systemic atherosclerosis, with increased cardiovascular morbidity and mortality. The most common presentation of mild to moderate lower limb PAD is intermittent claudication, or limb-threatening ischaemia. Patients with claudication have a severely impaired functional status and lower quality of life score; and peak exercise performance is about 50% that of age-matched controls, equivalent to moderate to severe heart failure using New York Heart Association criteria.

Patients with PAD are at a significantly higher risk of death, myocardial infarction and stroke, and a four-to-six-fold increased mortality, usually from cardiovascular or cerebrovascular disease. Despite the high prevalence of PAD and its strong association with cardiovascular morbidity and mortality, PAD receives much less attention than patients with coronary artery disease (CAD), and these patients are less likely to receive appropriate treatment to address their atherosclerotic risk factors than are those with CAD.

In Australia, diabetes is the fastest growing chronic disease. It is estimated that up to 280 Australians develop diabetes each day; there are almost 1 million Australians currently diagnosed with diabetes, and the total number of Australians with diabetes and pre-diabetes is around 3.2 million.

Research has demonstrated that a 1% decrease in measure of glycaemic control (the HbA1c), was associated with a 43% decrease in the risk of amputation or death associated with PAD.

The aetiology of leg ulcers is complex. PAD is present in 50% of patients with foot ulcers, and is an independent risk factor for ulceration and limb loss in diabetes. The lifetime risk of a foot ulcer in diabetic patients is between 15% to 25%, and the risk of major amputation in diabetes is 23 times that of a non-diabetic. Diabetic patients with foot ulcers and PAD are less likely to heal their ulcers.

WHEN TO REFER TO A VASCULAR SURGEON
Despite its prevalence, up to 50% of patients with PAD are either asymptomatic, or may present with hardly noticeable, but slowly progressive limitation of activity, especially if there are intercurrent disease (e.g. musculoskeletal, cardiac, or pulmonary disease). Management of PAD and all atherosclerotic disease, should address the condition itself, prevention of disease progression, and risk factor modification.

Specific symptoms of PAD are related to the area of the vasculature affected. Atherosclerosis occurs in specific areas of the lower extremity vascular bed, and below-knee vessel disease is most common in the diabetic population and may be asymptomatic because of associated neuropathy.

From the vascular surgical point of view, patients should be referred on the basis of symptomatic claudication with ankle brachial pressure less than 0.9, lifestyle dependent compromise including social and economic factors; and diabetic patients with absent pulses and especially those with neuropathy, and of course those with limb threatening ischaemia.

THERAPY FOR PAD
Traditionally, interventional management is reserved for patients with limiting claudication, or those with ulcers or limb threatening ischaemia, and consisted of endarterectomy or bypass surgery. Endovascular therapy can be performed using local anaesthetic techniques, and avoiding the need and risks of general anaesthesia enables the treatment of high-risk and elderly patients. (Figures 1A and 1B.) Endovascular therapy is equivalent or superior to bypass for patients with diabetes and ischaemic foot ulcers. (Figures 1Cand 1D)
The morbidity and mortality from catheter-based therapy is extremely low, compared with open surgical revascularisation, and there are continuous improvements in this evolving technology. Patients are able to eat and walk on the day of treatment, they can often return to normal activity within 24 hours of an uncomplicated procedure, and many cases are performed on a day-case basis, unlike open vascular surgery. Endovascular interventions for a target lesion generally do not preclude or alter subsequent surgery and may be repeated if necessary.

CAROTID ARTERY DISEASE

Up to 9% of the population over the age of 75 years will have a carotid artery stenosis of 50% or greater and carotid disease, like PAD, is considered a surrogate marker for risk of systemic atherosclerotic occlusive disease. The triggers that activate an asymptomatic carotid plaque to become symptomatic remains unknown, but the relationship between the severity of stenosis and the risk of stroke is well established. The Carotid Endarterectomy Trialists have convincingly demonstrated that symptomatic patients with a carotid artery stenosis of greater than or equal to 70%, benefit from carotid endarterectomy (CEA). (Figure 2A.) The benefits of intervention from surgery for asymptomatic carotid stenosis is seen in men more than women, and also in patients less than 75 years of age, and with a life expectancy of more than 5 years.

WHEN TO REFER TO A VASCULAR SURGEON

All patients with high grade asymptomatic carotid stenosis and those with focal neurological deficits referable to the carotid or vertebral circulation, including visual deficits, transient ischaemic attacks, and mini-strokes or strokes with good recovery, should be referred for vascular assessment.

ABDOMINAL AORTIC ANEURYSM

Abdominal Aortic Aneurysms (AAA) exists when the aorta exceeds 3.0cm in maximum diameter (or dilatation greater than 50% the diameter of the native vessel). The prevalence of AAA is approximately 5% to 7% in men and 1% to 2% in women, and 25% of these are aorto-iliac. Thoracic aneurysms are five-times less common than aneurysms in the abdominal aorta, and other peripheral aneurysms may co-exist. Currently there are no national AAA screening guidelines in Australia. AAA expands at an average rate of 3-5mm each year, although larger aneurysms expand more rapidly. Symptomatic patients with abdominal or back pain, rapidly expanding aneurysms, aneurysms over 5.0 to 5.5cm in men, and perhaps at smaller sizes in women, are at significant risk of rupture and should be considered for surgery. (Figure 3A.)

Figure 2A. High grade symptomatic carotid stenosis

The single most important issue for symptomatic carotid interventions is rapid treatment and there is now a concerted drive towards treating symptomatic patients as soon as possible after onset of symptoms, as a greater proportion of patients will die or suffer permanent disability through delayed interventions. Urgent CEA for evolving symptoms has a higher risk of procedural stroke than CEA for stable symptomatic patients, but prevent more strokes. (Figure 2B.) Early intervention for symptomatic carotid stenosis remains a challenge for Carotid Artery Stenting (CAS), but CAS outcome may be equivalent to CEA for younger patients. CEA may be a more suitable option for older patients.

Figure 2B. Opened Carotid Artery with Haemorrhagic plaque

WHEN TO REFER TO A VASCULAR SURGEON

Patients found to have an AAA should be referred for vascular assessment at initial diagnosis, for general assessment and advice, and to arrange vascular surveillance, and general treatment. Patients should also be referred for assessment when the aneurysm is at the threshold size, and those with a rapid rate of growth, of greater than 0.5cm in 6 months or 1cm per year. Patients who are symptomatic, with abdominal or back pain and any history of syncope or collapse should be referred urgently.

Figure 2B. Opened Carotid Artery with Haemorrhagic plaque

Figure 3B. Open repair of abdominal aortic aneurysm with tube graft

THERAPY FOR AAA

Conventional open AAA repair consists of a laparotomy, aorto-iliac dissection, aortic cross clamping and replacement of the diseased aortic wall with a prosthetic graft. Endovascular intervention for AAA, provides an alternative to open repair, and involves placement of an endovascular stent to exclude the diseased aortic wall from the circulation. Advances in endovascular technology now mean that EVAR is applicable in up to 90% of all aneurysms, and is associated with significantly lower operative mortality compared to open surgery. Endovascular therapy is limited by the use of nephrotoxic contrast, the use of ionising radiation and some anatomical considerations. Both open and endovascular repair for aneurysms need long-term follow up and may be associated with a re-intervention rate. (Figure 3B and Figure 3C.)

Figure 3C. CT reconstruction - post endoluminal aortic aneurysm repair

References available on request.
ENDOSCOPIC SURGERY FOR CARPAL TUNNEL SYNDROME

Dr Stuart Kirkham

DR. STUART KIRKHAM MBBS, FRACS, FAORTHA
Dr Kirkham is an orthopaedic surgeon. After FRACS, he completed a further 12-months sub-specialisation in hand and upper limbs at Baylor College, Houston Texas. He has been in practice in Sydney since 2000. He is involved in a number of research projects and also teaches registrars for the AOA. His areas of practice are hand, wrist, elbow and shoulder. His interests include endoscopic carpal tunnel, Dupuytren’s, scaphoid, tennis elbow, rotator cuff, and arthritis.
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DEFINITION
Carpus = wrist, Latin.
Carpal tunnel syndrome (CTS) is the entrapment neuropathy of the median nerve at the wrist.

EPIDEMIOLOGY
Carpal tunnel syndrome is the most common neuropathy of the upper limb. (Cervical neuropathy is 2nd and ulnar neuropathy at elbow is 3rd in frequency).

It has been estimated to affect 3–6% of the adult population.

PATHOLOGY
Macro: The nerve is compressed, deep to flexor retinaculum (aka transverse carpal ligament). Anything which either (a) increases size of contents, or (b) decreases the size of tunnel will cause crowding of Median nerve. The nerve may appear flattened and adjacent areas may appear expanded, “hourglass shape”. There may be pale hypo-aemia. There can be hyperaemia and micro-vascular congestion in adjacent areas of the nerve.

Micro: Firstly the nerve undergoes demyelination. At this stage normal “saltatory” (jumping) conduction cannot occur between nodes of Ranvier. Later, there may be axonal loss and even axonal fibrosis, which is irreversible.

AETIOLOGY
1. “Idiopathic” group which represents 98% of all cases,
2. Demonstrable causes. 2%:
   a. Wrist injury – fractures, local swellings.
   b. Fluid retention disorders – pregnancy, lactation, lymphoedema, eg mastectomy, CCF, CRF, hypothyroidism.
   c. Space occupying lesions – ganglion, flexor synovitis, eg Rheumatoid.
   d. Conditions that mimic carpal tunnel syndrome – peripheral neuropathy (diabetes, chemotherapy, vitamin deficiency etc), vibration arm syndrome (motorbikes, jackhammers etc).

CLASSIFICATION
Mild NCS ~ 45 m/s sx < 3 months
Moderate NCS 35-45 m/s sx 3-12 months
Severe NCS < 35 m/s sx > 1 year

SYMPTOMS
Sensory - waking up at night, tingling, pins and needles, numbness, clumsiness, dropping things, especially in kitchen or work when holding a steering wheel. The classical presentation is a sensory disturbance in the median 3½ fingers distribution, but this varies significantly amongst individuals. It can be all 5 digits.

Motor Thenar muscle wasting is only seen in the worst cases and is a sign of very late disease with a poor prognosis.

SIGNS
• Tinel’s – described by Jules Tinel, French neurologist.
• Phalen’s – described 1950 by George Phalen.
• Derkan’s – the most sensitive and specific.

Interestingly, these signs start to become negative in advanced disease. My opinion is that this heralds a worse prognosis. Once the nerve is no longer physically irritable to examination it is less healthy. This is a current research topic of mine.

INVESTIGATIONS
Nerve conduction studies (NCS)– is not only diagnostic beyond doubt; but also quantifies the severity of the disease in a way that can be reproduced post-treatment if necessary.

I tend to only use it in 2 circumstances: (a) if the diagnosis between wrist and neck needs localisation or (b) if the patient seems to be a trouble maker – it can be a valuable evidence in the case of malingerers.

DIAGNOSIS
Mostly clinical (history and exam); NCS is definitive – see above.

NATURAL HISTORY
Unfortunately, most patients get steadily worse over time. There is published evidence to show that waiting or postponing treatment is not helpful.

OUTCOMES
99% of patients go home within 1–2 hours. 90% or more take nil post-operative analgesics – including nil Panadol. 95% will drive a car the next day. Office workers and self-employed typically resume typing and working within a few days. Manual labourers resume at 3–4 weeks. Golf and tennis usually takes 4–5 weeks.

Sensory relief – most patients report that the nocturnal symptoms resolve or improve within 1–2 days. The recovery of median nerve sensibility varies depending upon severity and duration of disease, and medical comorbidities. Maximal nerve regeneration may take 1–2 years.

Pillar pain – occurs with activities in 5% of cases and typically lasts 4–6 weeks.

ENDOSCOPIC CTR
Day surgery; Takes 5 minutes. Usually a light GA is preferred. A camera is introduced into the CT & the TCL is divided, making space for the nerve. The skin incision is 1cm and is hidden in the transverse wrist crease in non-glabrous skin (less keratinised, less eccrine glands, hair follicles). Soft dressings; no plaster slabs. A relative takes patient home at 1–2 hours post-operative.

References available on request.

Figure 1. CTR wound at 2 weeks

Figure 2. ECTR wound at 6 months
Chronic heart failure (CHF) is a growing pandemic in Australia. CHF affects 2.5% of Australians aged 55–64 years, and 8.2% of those aged older than 75 years. It is recognised as the 8th most common cause of death in Australia. For patients with end stage heart failure deteriorating on maximal medical therapy the options are limited – some will qualify to wait for a heart transplant whilst the others face ongoing poor quality of life and recurrent hospitalisation.

Mechanical circulatory assist devices have developed to “bridge” patients to transplant, and possibly offer others a “destination” therapy with improved quality of life and survival.

Mechanical circulatory support device therapy is approaching its 50th anniversary, since the implantation of the first ventricular assist device (VAD) by DeBakey in 1966. First generation pumps were bulky pulsatile devices with limited durability, high failure rates and device related complications such as stroke and infection. Despite these failings the devices proved superior to optimal medical therapy, evident in the 2001 REMATCH trial (Randomised Evaluation of Mechanical Assistance for the Treatment of Congestive Heart Failure). VAD technology has evolved to continuous flow “3rd generation” devices. Patients now have access to smaller devices with improved durability, low device complications and resulting better patient outcomes. Among all continuous-flow pumps, actuarial survival is 80% at 1 year and 70% at 2 years. Freedom from neurological events, device failure and pump infection at 12 months is 89%, 96% and 98% respectively.

The Heart Lung Transplant Unit at St Vincent’s Hospital utilises the HeartWare® HVAD®, an intra-pericardially placed pump weighing 140g (Figure 1). It has a single moving part, an impeller that spins to potentially generate up to 10L/min of flow. The impeller is suspended by a combination of passive magnetic and hydrodynamic forces. The simple implant procedure involves attaching a sewing ring to the apex of the heart, enabling a short inflow cannula to be placed into the left ventricle. The device sits within the pericardium and a 10mm outflow graft connects the HVAD® to the ascending aorta (Figure 2). A 4.2mm driveline exits the skin below the right costal margin and connects to an external controller. The controller has 2 batteries, providing 6 hours of power or connection to wall or car electricity outlet. The pump parameters and alarms can be read and adjusted from the controller.

Since 2008, 77 HVAD® pumps have been implanted at St Vincent’s Hospital as a bridge to transplant. There was a 91% and 86%, 6 and 12 month survival (alive or transplanted). There is limited experience in the destination therapy cohort (10 patients since 2006), however 6 and 12 month survival is 100% and 87.5% respectively.

VAD technology is continually evolving and the development of even smaller devices allow for implantation using minimally invasive surgical techniques via a mini-thoracotomy. Excellent results in the bridge to transplant population will ensure the continuing use of VAD’s in this patient group. The combination of further miniaturisation and improved mid-term patient outcomes may allow for a broadening of the indications for VAD therapy, and enable more end stage heart failure patients to use these devices for destination therapy.

References:
INTRODUCTION
The term ‘rhinosinusitis’ describes a constellation of disease entities with a common feature - inflammation of the mucosa lining the nasal cavity and the paranasal sinuses. Rhinosinusitis is classified according to the duration of symptoms (Table 1).

ACUTE RHINOSINUSITIS
Acute rhinosinusitis (ARS) is a common disorder affecting approximately 31 million people in the US annually with direct costs estimated at $US3 billion. It is frequently caused by an acute viral infection associated with the common cold, but bacteria have also been implicated primarily, or resulting in a secondary infection. The principal bacterial pathogens in ARS are Streptococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis and Streptococcus pyogenes. Key diagnostic criteria include symptoms following upper respiratory tract infection, purulent nasal discharge (Figure 1), unilateral maxillary sinus tenderness, maxillary tooth or facial pain (especially unilateral), and a history of initial improvement followed by a worsening of symptoms. Other non-specific symptoms include malaise, halitosis, nasal congestion, hyposmia/anosmia, fever and cough. Complications are uncommon, occurring in an estimated 1 in 1,000 cases, with the majority settling with empirical antibiotic therapy.

CHRONIC RHINOSINUSITIS (CRS)
Whilst many of the symptoms are similar, understanding the distinction between the acute forms of rhinosinusitis and CRS has both clinical and scientific importance. In CRS (Figures 2 and 3) the duration is greater than 12 weeks, and whilst disease fluctuations occur, the signs and symptoms of CRS never completely resolve between these, setting CRS apart from ARS, subacute rhinosinusitis, and recurrent ARS.

Data from a recent Australian national health survey has shown that 9.0% of Australians suffer from CRS symptoms. CRS has significant socioeconomic implications, with an estimated annual direct health care cost in the US of $US5.8 billion. Patients with CRS visit primary care clinicians twice as often as those without the disorder, and or have five times as many prescriptions filled. CRS is the second most prevalent chronic health condition in the US population. It is extremely detrimental to the quality of life of those suffering from it, and quality of life measures are similar or worse than chronic obstructive pulmonary disease (COPD), chronic back pain, and congestive cardiac failure.

MANAGEMENT
Treatment is medical in the first instance. Empirical antibiotics, systemic and intranasal topical corticosteroids and saline douches are used as the first line treatment and approximately 30% of patients respond to this treatment alone. Endoscopic sinus surgery has been accepted as the treatment of choice for chronic sinusitis refractory to medical treatment. The aim of sinus surgery is to reventilate the sinuses, remove inflammatory mediators and facilitate topical therapy.
Successful surgical intervention in diseased sinuses is dependent on total removal of all inflammatory mediators - “inflammatory load hypothesis”. There are a number of factors affecting the inflammatory load (Figure 4) including biofilm formation, eosinophilic mucin and fungal antigens. Leaving inflammatory mediators behind by not addressing a sinus when it is diseased exposes the patient to persistence of symptoms and potentially revision surgery. A number of new techniques have been devised to achieve total removal of all inflammatory mediators. In particular the Endoscopic Modified Lothrop Procedure (Figure 5) has been increasingly used to treat the chronically diseased frontal sinus. The EMLP\textsuperscript{11,12} unites the left and right frontal sinuses through a common opening by removing the floor of the frontal sinus. Its success in treating recalcitrant disease is thought to be because it facilitates complete removal of inflammatory mediators within the frontal sinus intraoperatively and allows topical treatment postoperatively.

**SUMMARY**

Rhinosinusitis is a common disorder with a significant impact on health and quality of life. The diagnosis is made using a combination of symptoms, signs and radiological findings. Medical treatment alone is often sufficient to resolve symptoms. Surgical treatment is reserved for cases that fail medical treatment or with acute complications. Endoscopic Sinus Surgery has evolved dramatically over the past 5 years with a focus now on removing all inflammatory mediators within affected sinuses for optimum results.

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**Table 2. EPOS Criteria for the diagnosis of CRS**

<table>
<thead>
<tr>
<th>Symptoms two or more for &gt; 12 weeks</th>
<th>Special Assessment</th>
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<tbody>
<tr>
<td>Nasal blockage / obstruction / congestion*</td>
<td>Endoscopic assessment? nasal polyposis\†</td>
</tr>
<tr>
<td>Nasal discharge (anterior / posterior nasal drip)*</td>
<td>CT Sinuses\†</td>
</tr>
<tr>
<td>Facial pain / pressure</td>
<td>Allergy Testing if history suggestive</td>
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<tr>
<td>Reduction or loss of smell</td>
<td>*Must include at least one of these symptoms</td>
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<td></td>
<td>\†Must include at least one endoscopic or radiological finding</td>
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</tbody>
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**References:**

NEWLY ACCREDITED SPECIALISTS

Dr Katrina Adorini  
MBBS, B.Med.Sci, FRACP  
Cardiology, Angiography, Echocardiology  

Dr Adorini graduated from Medicine at The University of Sydney. She trained at Concord and St Vincent’s Hospitals in Cardiology with a strong focus on heart failure, transplant medicine and pulmonary arterial hypertension. She now practices at Sydney Adventist Hospital and has joined Specialist Cardiology at the San Clinic. P: 9473 8633

Dr Ian Bilmon  
BSc (Mathematics), BMBS (Hons), FRACP, FRCPA  
Clinical Haematology  

After completing an undergraduate degree in Pure Mathematics, graduating in Medicine and pursuing joint Fellowships in Clinical and Laboratory Haematology, Dr Bilmon undertook a research Fellowship at the Westmead Millennium Institute (University of Sydney) and a Fellowship in allogeneic Haematopoietic transplantation through St Vincent’s Hospital, Darlinghurst. His subspecialty areas of interest include stem cell transplantation and Haematologic malignancies, including lymphoma, myeloma, and marrow disorders of the elderly. P: 9473 8833

Dr Jonothan Brock  
BAppSci(OT), BMed, FANZCA  
Anaesthetist  

After completing his medical training at the University of Newcastle, Dr Brock underwent his Anaesthetic training and Provisional Fellowship year at Royal North Shore Hospital. Dr Brock maintains his interest in teaching, and is currently an instructor on EMAC courses for Anaesthetic staff, and is an anatomy lecturer for senior Anaesthetic trainees. Dr Brock’s interests include Anaesthesia for orthopaedic, vascular, and neurosurgical interventions, as well as sedation and regional Anaesthesia for surgical procedures. P: 9550 4866

DIARY DATES

SAH GRAND ROUNDS (ALL GP’S INVITED)  
October 15  Professor Sharon Killbreath – Breast cancer and exercise, lymphoedema management and prevention of shoulder impairments post mastectomy.  
November 11  Dr Sultan Baskar – Multi-resistant organisms and the impact for the house care provider.  
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).

GP CONFERENCES (CPD points available with proof of attendance)  
October 17  Oncology

FREE PUBLIC FORUM (everyone welcome)  
November 6  Pre-Conception Care/Maternity  
Dates and topics are subject to change. Contact 9487 9871 to register or visit www.sah.org.au for further details.

EVENTS  
27 October  San Foundation’s Annual Memorial Service at Wahroonga Seventh-Day Adventist Church, 2pm. All welcome to the ecumenical service of remembrance and thanksgiving. Contact 9487 9405.

November 1  SAH Clinical Education Centre official opening.

December 8  San Carols by Candlelight

NEWS FROM THE SAN

• The SAH Clinical Education Centre will be officially opened by the NSW Governor and Federal and State Government representatives on 1st November. Providing education and simulated learning facilities for multidisciplinary clinical training of medical, nursing and allied health professionals, the purpose-built centre will be home to the Sydney Adventist Hospital Clinical School of The University of Sydney (SAHCS) and Avondale College of Higher Education, Faculty of Nursing and Health. Dedications of an auditorium seat or learning space in someone’s honour are welcomed and can be discussed with Assistant Director of Medical Services Catherine Murphy on 9487 9400. See www.sah.org.au for more details.

• The $181 million Redevelopment currently underway on site providing for up to 200 new beds, up to 12 additional operating theatres, a new Maternity and Women’s Health Unit, a new entry and arrivals building and the new Integrated Cancer Centre, will be completed as from mid next year.

• Urologist A/Prof Henry Woo will lead a prostate cancer data collection pilot project at SAH funded by the Cancer Council. The project aims to create a nationally recommended standard for clinical prostate cancer data collection and improve patient outcomes.

• The National Blood Authority ranked San Pathology number one for their low red blood cell discard rates. San Pathology’s 0.7% wastage is significantly lower than the national average of 6%.

• SAH’s Australasian Research Institute and Gordon Rugby players have teamed up in a project to determine whether there is a correlation between dietary intake of antioxidants and their concentrations in the blood and skin.

• On 25 October SAH is hosting a conference of wound experts including the internationally acclaimed Leg Club founder, Ellie Lindsay. The Leg Club is renowned across the UK for its results in healing leg ulcers and restoring quality of life to sufferers. See www.sah.org.au/conferences for bookings.

• SAH’s HealthCare Outreach program has been renamed ‘Open Heart International’ in recognition of the open hearted nature of its Australia-wide volunteers.

• Open Heart International volunteers from the San’s Coronary Care Unit are organising a 20km coastal trek to raise funds for a trip to Cambodia in October. Volunteers have been travelling to Cambodia since 2007 performing free cardiac surgery on children who otherwise would not survive. Support trekkers and donate online at www.ohi.org.au.

• SAH is the first private hospital in NSW offering a re-entry program for nurses returning to the workforce. The next course starts February 2014. Contact Fiona Blades on 9487 9206.

• Petrea King’s acclaimed educational and life-changing “Living Well with Cancer” program for cancer sufferers and their carers commences 16 October at San Cancer Support Centre. See www.sah.org.au for more details.