Dr Julie Bradley

MBBS FRACP

Julie graduated from the University of Adelaide, MBBS with Distinction in 1978 and completed her physician training at Flinders Medical Centre and cardiology training at the Royal Adelaide Hospital. In 1985 Julie obtained her FRACP and became Adelaide's first female Cardiologist. From 1985- 1986 she studied Doppler echocardiography at the Brompton Hospital in London and in 2005 was awarded her FCSANZ.

Julie is a Senior Staff Specialist at the Royal Adelaide Hospital, an active member of the Structural Heart Disease program and Head of the Royal Adelaide Hospital Echocardiography Department.

She continues her interest in echocardiography, coronary angiography, women's heart health issues and cardiac failure, and is further developing her interest in Non-Invasive Cardiac Imaging, in particular Coronary CT Angiography and Cardiac MRI.

Julie joined Adelaide Cardiology in 2009 and consults at the St Andrew's Medical Centre.

Our Cardiologists

Adelaide Cardiology provides an extensive range of cardiac services and subspecialties ensuring that patients have access to the complete range of cardiac care within our Practice.

Dr Peter Steele
Interventional

Dr Joseph Montarello
Interventional

Dr Michael Brown
Interventional, Non-Invasive Cardiac Imaging (CT, MRI)

A/Prof Glenn Young
Electrophysiology

Dr Daniel Cehic
Electrophysiology

Dr Peter Sage
Interventional

Prof Stephen Worthley
Interventional, Non-Invasive Cardiac Imaging (CT, MRI)

Dr Patrick Disney
Echocardiography, Adult Congenital Heart Disease

Dr Karen Teo
Non-Invasive Cardiac Imaging (CT, MRI)

Dr Julie Bradley
Echocardiography

Dr Georgy Chacko
Interventional

Dr Maria Santos
Electrophysiology

Dr G (Srini) Srinivasan
Echocardiography, Non-Invasive Cardiac Imaging (MRI)

Dr Jamie Morton
Echocardiography, Non-Invasive Cardiac Imaging (CT)

Dr Luay Samaraie
Non-Invasive Cardiac Imaging (CT and MRI)

Dr Charles Tie
Interventional

Locations

City & Suburbs

St Andrew's Medical Centre:
Level 2, 321 South Terrace Adelaide

Leabrook Clinic:
286 Kensington Road Leabrook

Kurralta Park Clinic:
Tennyson Centre,
520 South Road Kurralta Park

Modbury Clinic:
71 Smart Road Modbury

Unley Road Clinic:
313 Unley Road Malvern

Regional

Angaston Hospital:
29 North Street Angaston

Bridge Clinic:
8 Standen Street Murray Bridge

Broken Hill Base Hospital:
Thomas Street Broken Hill

Clare Medical Centre:
Old North Road Clare

Gawler Health Services:
21 Hutchinson Road Gawler

Littlehampton Medical Centre:
89 North Terrace Littlehampton

Mannum Medical Centre:
Parker Street Mannum

Minlaton Medical Centre:
7 South Terrace Minlaton

Wallaroo Hospital:
Ernest Terrace Wallaroo

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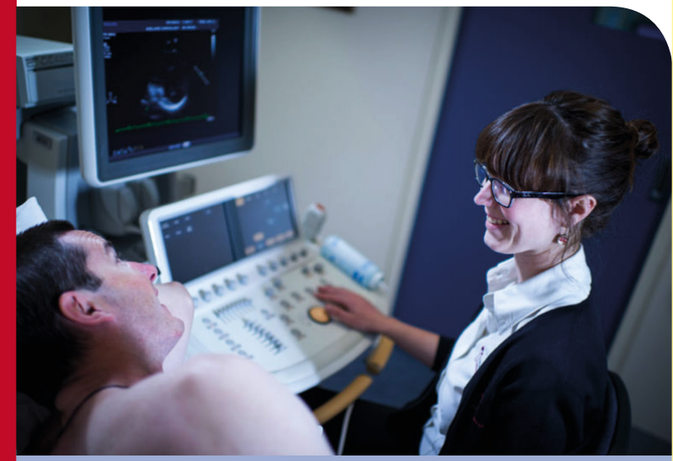
Adelaide Cardiology



the beat

summer 2015

GenesisCare

Adelaide Cardiology News

Adelaide Cardiology is pleased to welcome Dr Charles Tie to the practice.

Charles graduated from University of Otago, New Zealand in 2001. His internship, general medical and cardiology training were completed at the Dunedin Hospital. He was awarded FRACP (dual training in Cardiology and General Medicine) in 2012 and went on to complete 18 months of Interventional Fellowship at the Christchurch Hospital. He has a special interest in the diagnosis and management of coronary artery disease as well as various aspects of interventional cardiology ,and will be performing diagnostic and interventional procedures at St Andrew's Hospital.. Charles will commence consulting at the St Andrew's Medical Centre and our Leabrook, Unley Road, Modbury and Wallaroo Clinics in February.

Adelaide Cardiology CPD Education Event: 10th March 2015

Dr's Peter Steele and Srini Srinivasan will host a dinner on the 10th of March (at 6.30pm at the Lenzerheide Restaurant, 146 Belair Road, Hawthorn) to review :

Chest Pain Assessment and Coronary Artery Disease in General Practice

To register for this event please email **jbreen@adelaidecardiology.com.au** or ph: **0428 287 952**

RACGP Category 2 Points will be awarded for this event.

Details of additional CPD events can be found at:
www.adelaidecardiology.com.au

Dr Julie Bradley Presents: A Guide to Cardiac Investigation of Chest Pain

Assessment of chest pain is challenging. It requires a stepwise approach, mindful of the clinical setting, pre-test probability of disease (Bayes Theorem), a knowledge of the predictive accuracy of the test and assessment of the relative risks and benefits of the test.

In the patient presenting with acute chest pain and in the absence of trauma, four potentially fatal conditions need to be considered and excluded:-

1. Acute coronary syndrome (ACS) – encompassing acute myocardial infarction (AMI) and unstable angina pectoris (UAP).
2. Pulmonary embolism.
3. Aortic dissection.
4. Spontaneous pneumothorax.

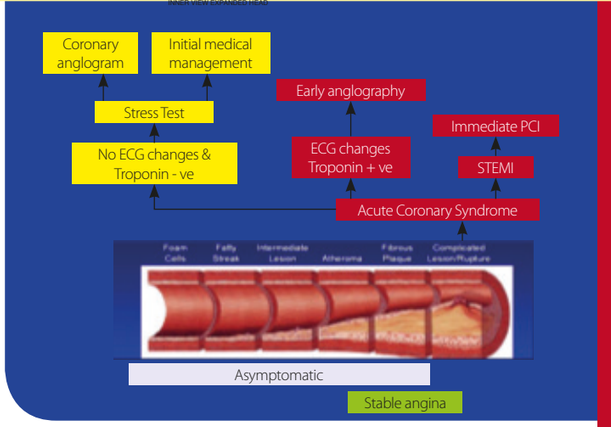
ACS is the most common and challenging to diagnose because it is not excluded with an acceptable level of accuracy on initial clinical evaluation or a single investigation.

The aim of chest pain assessment is to :-

1. (a) Identify ST elevation myocardial infarction (STEMI) quickly and accurately to enable rapid transfer to catheter laboratory for immediate percutaneous coronary intervention (PCI), which requires an ECG only and has a major impact on outcome.
   (b) Identify non-STEMI or UAP to permit appropriate medical therapy and early angiography (<24hours) if appropriate, with associated improved outcome.
2. Accurate exclusion of myocardial ischaemia with minimization of missed diagnoses and unnecessary testing.

In general practice, the aim should be to distinguish patients who require urgent hospital assessment for possible ACS from those with more stable symptoms who can be investigated as an outpatient. This is largely based on clinical judgement, as patients with suspected ACS and prolonged chest pain or ongoing symptoms, pain within 12 hours that has resolved with ECG changes or progressive unstable angina symptoms, require serial troponins and cardiac monitoring in hospital, even if initial ECG is normal.

Risk stratification of ACS patients into high, intermediate and low risk categories according to NHF/CSANZ guidelines is recommended to assist with appropriate use of resources.



1. ECG - essential in all patients. Emergency ambulance hospital transfer if STEMI or ST changes (even if initial troponin is negative).
2. Cardiac biomarkers - serial high sensitivity troponin has an essential role in diagnosis of AMI. It is likely to rule out AMI earlier than less sensitive assays and accelerate risk stratification models but at expense of reduced specificity.

Investigations for myocardial ischaemia and coronary artery disease.

There are 2 groups of patients, those with ACS where AMI has been excluded and those with stable chest pain for investigation for possible angina. In general, prognostically important coronary artery disease (CAD) is assessed non-invasively by stress imaging (ECG, ECHO, nuclear or MRI) whereas anatomical information has traditionally been obtained by invasive coronary angiography. The recent development of CT coronary angiography (CTCA) provides a non-invasive anatomical assessment of presence or absence of CAD.

Each investigation has its strengths and limitations and provides most value in patients at intermediate risk. In low risk patients with low pre-test probability of CAD, a negative test is likely to be reliable but there is increased likelihood of a false positive result. The converse occurs in the high risk patient with the additional risk of precipitating ischaemia or infarction with stress testing.

3. Exercise stress test – is inexpensive and readily available but is largely limited now to identifying patients who can be safely discharged from hospital ED. Limitations include inability to exercise, inadequate heart rate response, abnormal resting ECG and some drugs e.g. digoxin. It has a fairly low predictive accuracy for detecting reversible ischaemia (sensitivity 68%, specificity 77%) and has mostly been replaced by other stress imaging modalities, predominantly stress ECHO.
4. Stress ECHO – is the combination of 2D ECHO assessment of left ventricular (LV) function and either exercise or pharmacologic (dobutamine + atropine) stress, if unable to exercise. Resting and stress images are compared looking for development of regional wall motion abnormalities to indicate reversible ischaemia, identify a 'culprit' lesion and risk stratification (sensitivity 76%, specificity 88%). Dobutamine stress ECHO can also be used to assess viability and valvular disease severity. Limitations of image quality have significantly improved with ECHO contrast and inadequate heart rate response can be minimized by holding B-blockers for 3 days prior. Advantages of the technique include low cost, no radiation and wide availability. A negative stress ECHO is associated with a < 1%/year event rate (death, AMI, revascularization) over the next 5 years.
5. Radionuclide myocardial perfusion imaging (rMPI) – involves injection of a radioisotope(Tc -99, sestamibi, thallium) that is distributed throughout the myocardium in proportion to blood flow combined with exercise or pharmacologic stress usually with dipyridamole (vasodilator). Myocardial perfusion images are compared to identify regions of reversible ischaemia (sensitivity 87%, specificity 72%). Slightly higher sensitivity compared with stress ECHO is offset by lower specificity, cost and radiation. Dipyridamole should not be used in patients with severe asthma or a-v block. Caffeine should be held for 48hrs prior. A normal rMPI is associated with < 1%/ year event rate.
6. Stress MRI – has emerged as a safe and more accurate alternative to rMPR and stress ECHO with no radiation but is not widely available and can only be performed in selected patients. It is the gold standard for assessment of myocardial viability and scar.
7. Coronary angiography – is the gold standard for assessment of CAD but is invasive with 2% risk of death or other complications. The risks and benefits need to be weighed on an individual basis but is a necessary requirement for any invasive treatment strategy.
8. CT coronary angiography – is rapidly evolving , with a high negative predictive value of 99% for exclusion of CAD. It is likely to be used for rapid assessment and discharge from ED in 'low risk' chest pain population. Limitations include sinus rhythm, < 60bpm, calcification, renal function and radiation.

Local expertise, patient characteristics (e.g. poor ECHO images) and patient preference should also be considered in selection of the appropriate test.

References:

Med J Aust 2006; 184 (8Suppl):S1 – 30
Med J Aust 2013; 199(1):30-34
NEJM 2005;342:1187-1195.