

Winter 2022

# GP Connect

Supporting best practice in cardio-metabolic health



## From the editor – Dr Gunjan Aggarwal

Specialising in general adult cardiology and non-invasive cardiac imaging, particularly echocardiography and cardiac computed tomography (CT).

Welcome to the winter edition of GP Connect 2022. This issue provides updates in cardiac electrophysiology by focusing on a novel pacing technique.

Left bundle branch area pacing (LBBAP) has gained attention worldwide as a pacing strategy in patients with AV block to reduce the risk of pacing-induced heart failure. It is a new technique that is feasible and safe in a high percentage of patients. It serves as an alternative to His bundle pacing (HBP) for preventing intraventricular dyssynchrony associated with traditional right ventricular pacing (RVP).

Dr Bill Petrellis provides a valuable introduction to this novel technique including a clinical vignette highlighting its potential use. There is no doubt that LBBAP has great potential in the field of cardiac pacing and as more randomized trial evidence emerges its use will no doubt increase in the future.

There are also additional articles on the benefits of salt restriction and the impact of statin therapy on atherosclerotic plaque progression.

I hope you enjoy this edition of GP Connect. We are always available to assist you in any way that we can as you navigate the COVID pandemic which hopefully is coming to an end. Stay safe.

Yours Sincerely

Dr Gunjan Aggarwal.

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## Access past issues

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### CVD Risk in Women

As seen in Australian Doctor How to Treat - Written by Dr Fiona Foo

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\*GPs must register/log in to access this article



### Procedures with A/Prof Martin Brown

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# Effects of using salt substitute instead of regular salt



## Dr Gunjan Aggarwal

Specialising in general adult cardiology and non-invasive cardiac imaging, particularly echocardiography and cardiac computed tomography (CT).

The Salt Substitute and Stroke Study (SSaSS) results were recently presented at the European Society of Cardiology meeting. It was funded by the NHMRC and the George institute for Global Health in Sydney.

The study enrolled 20,000 patients over the age of 60 with uncontrolled hypertension or previous stroke from 600 rural villages in China and followed them over a period of 4.7 years. 72% had a history of stroke and 88% had known hypertension. The villages were randomly assigned to either regular salt or a salt substitute (containing 75% regular salt and 25% potassium chloride). This salt substitute looked and tasted identical to regular salt. It was a simple way of reducing sodium intake and increasing potassium content in the diets of patients with hypertension. Patients with significant kidney disease were excluded due to the elevated risk of hyperkalemia.

There was an overall 12% reduction in death, 14% reduction in stroke and 13% reduction in major cardiovascular events, all highly significant<sup>1</sup>. The main safety outcome was the rate of adverse events related to hyperkalemia which was not significantly different between the 2 groups. The degree of BP lowering was modest at around 3mm Hg for the salt substitute group.

There is some controversy about the effects of salt intake with previous observational studies such as the PURE study<sup>2</sup> showing a J curve where there may be elevated risk of cardiovascular events and mortality with low salt consumption (or mean intake around 4 grams/day). However other studies have cited that estimation of salt intake using a single spot urine sodium measurement derived formula may lead to error<sup>3</sup> and this association changes to a linear relationship when more accurate measures such as a 24 hour urine sodium collection is used instead<sup>4</sup>. Using a sodium to creatinine ratio also relies on the creatinine which may reflect muscle mass and a malnourished state that may have an independent impact on overall health and measures of mortality.



The study pertains to a population of rural Chinese patients and it is not clear if these results are generalisable to Western populations. Nevertheless the authors concluded that a simple intervention such as using substitute salt in the study population provided significant health benefits. Hopefully this study will serve as an impetus for the food industry and governments to encourage use of salt substitutes that contain potassium as a low cost and effective public health intervention.

1. Neal et al. Effect of Salt Substitution on Cardiovascular Events and Death. *New England Journal of Medicine*. August 29, 2021. 2. Mente et al. Urinary sodium excretion, blood pressure, cardiovascular disease, and mortality: a community-level prospective epidemiological cohort study. *Lancet*. 2018 Aug 11;392(10146):496-506. 3. Tan et al. Salt and cardiovascular disease in PURE: A large sample size cannot make up for erroneous estimations. *J Renin Angiotensin Aldosterone Syst*. 2018 Oct-Dec; 19(4): 1470320318810015. 4. He F et al. Formulas to Estimate Dietary Sodium Intake From Spot Urine Alter Sodium-Mortality Relationship. *Hypertension*. 2019;74:572-580.

## Our team

We have experienced cardiologists in all major sub-specialties to provide the highest quality of patient care. We also have specialists in related fields including endocrinology and respiratory medicine.

Our Sydney Cardiology team includes:



### Dr James Wong

Specialising in general cardiology, prevention of coronary artery disease and hypertension.



### Dr Abhinav Luhach

Specialising in general adult cardiology, cardiac CT, and preventive cardiology.



### Dr Suja Padmanabhan

Specialising in diabetes and general endocrinology with a special interest in diabetes in pregnancy and women's health.



### Dr Gunjan Aggarwal

Specialising in general adult cardiology and non-invasive cardiac imaging, particularly echocardiography and cardiac CT.



### Dr Andrew Terluk

Specialising in general cardiology with an interest in cardiomyopathy in the setting of cancer.



### Dr Tracy Smith

Respiratory and sleep physician specialising in respiratory disease with a special interest in respiratory failure due to lung or heart disease.



### Dr Ru-Dee Ting

Specialising in general and interventional cardiology, including cardiac haemodynamic studies and complex coronary intervention.



### Dr Fiona Foo

Specialising in general and interventional cardiology with an interest in heart disease affecting women and sports cardiology.



### Dr Bill Petrellis

Specialising in general adult cardiology and electrophysiology, including atrial fibrillation and device implantation.



### A/Prof Martin Brown

Specialising in advanced heart failure, pulmonary hypertension, and transplant cardiology.

# What is Conduction System Pacing?



## Dr Bill Petrellis

Specialising in general adult cardiology and electrophysiology, including atrial fibrillation and device implantation

## Progressive Evolution of Pacing Technology

Admittedly, there has been little to be excited about regarding cardiac pacing over the years. Pacemakers are commonplace, lifesaving implantable devices that have been available for decades allowing us to manage patients with symptomatic bradycardia, whether due to slow atrial fibrillation, sinus node dysfunction or heart block.

There has been a progressive evolution of pacing technology over the years. Devices are smaller, battery longevity has increased and pacing algorithms have been developed to manage atrial arrhythmias and to reduce the burden of unnecessary ventricular pacing.

Leadless pacemakers have recently been developed, which pace the heart via an all-in-one battery and electrode "capsule" delivered to and anchored within the right ventricular chamber. Future improvements will include "far field" atrial sensing of a single chamber device and the use of two "capsules", one in the atrium and another in the ventricle, connected wirelessly to permit AV synchrony, an important factor which helps preserve cardiac efficiency.

Traditional pacing techniques involve placing a lead within the RV, usually at the apex and more recently, the mid RV septum. Pacing the right ventricle alone induces electrical dyssynchrony, that is, the right ventricle contracts first and left ventricle is activated much later as the action potential propagates by slow conduction from the right ventricle via the myocardium, across the interventricular septum, and then on to activate the left ventricle. The electrical representation of this phenomenon is the broad, abnormal appearance of the paced ventricular QRS complex, which is effectively, pacing induced left bundle branch block (LBBB). The mechanical consequence of ventricular dyssynchrony is its potential to induce heart failure, a condition commonly referred to as pacemaker mediated cardiomyopathy. Other detrimental effects include an increased risk of atrial fibrillation and increased mortality. As such, there has been much effort in the study of alternate site pacing.

## Cardiac Resynchronisation Therapy (CRT)

The advent of CRT at the turn of the century, was a major advancement in the management of heart failure patients with intrinsic LBBB. This technique involves positioning one lead within the right ventricle (RV) and another to pace the left ventricle (LV) via a lead advanced into a suitable coronary sinus tributary overlying the lateral left ventricular myocardium. The

timing of left and right ventricular pacing can be adjusted to reduce dyssynchrony caused by the pre-existing LBBB. Most commonly, the LV lead is activated before the RV lead by a mere 20 to 40 milliseconds resulting in a narrower paced QRS pattern. The seemingly insignificant advancement of left ventricular activation results in a marked improvement of LV systolic contraction and reduced functional mitral regurgitation in up to 70% of patients. Occasionally, cardiac resynchronisation results in normalisation of the ejection fraction, and these individuals are known as super responders. However, CRT is limited by the coronary sinus anatomy, the presence of LV scar and the inherent potential for phrenic nerve stimulation, a structure which overlies the lateral left ventricle. Placing an LV lead is often technically challenging, not always successful, and furthermore, 30% of recipients are non-responders despite successful LV lead placement.

I recall during the second year of my advanced cardiology training at the Austin Hospital in 2002, a newly minted consultant cardiologist, who had just returned from the United States, requested CRT for an inpatient with heart failure. The experienced senior staff electrophysiologist accepted the challenge to implant his first CRT device. I recall his frustration while we spent several hours at the private hospital on a Saturday morning, trying to position a simple bipolar lead in the coronary sinus using rudimentary sheaths. Today, we have an almost endless catalogue of sheaths, additional tools and quadripolar leads, which have significantly increased the success rate of lead placement and improved RV/LV resynchronisation. Multipolar pacing and AV synchrony algorithms have further added to cardiac mechanical optimisation and perhaps CRT response.

Conduction system pacing is a technique that recruits the native His-Purkinje system producing physiological pacing which results in a narrow paced ventricular QRS complex. It therefore avoids the aforementioned phenomenon of electrical dyssynchrony induced by standard pacing techniques, including CRT.

Fast forward to 2004 during the second year of my Electrophysiology and Pacing fellowship at the University of Western Ontario in London, Canada. I quickly became a proficient implanter, spending a day or two each week grinding through my list of 5-7 new implants, upgrades, and generator replacements. But there was one thing I really didn't look forward to - my scheduled afternoon in the pacing research lab.

One of my supervising professors in London was researching the viability of His bundle pacing and comparing its haemodynamics (including cardiac output) to conventional RV apical pacing. The His bundle is a collection of heart muscle cells specialised for electrical conduction. It transmits electrical impulses from the atrioventricular node (AVN) to the bundle branches, causing the ventricles to contract. Hours were spent testing His bundle pacing on consenting pacemaker recipients, positioning, and fixing the lead, pacing at different rates and measuring haemodynamics, only to remove it and leave the final permanent lead at the RV apex.

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At the time, I did not appreciate the significance of his research, nor did I find it remotely interesting, for shame.... then there was radio silence for over a decade.

## Conduction System Pacing

An enthusiastic explosion for conduction system pacing occurred around 2015, which was building over the preceding 5 years. #don't-dis-the-His (<https://twitter.com/hashtag/dontdisthehis?lang=en>) was its catch cry. Imagine, cardiac pacing which produces a normal paced QRS complex and the ability to correct intrinsic LBBB. Yes, you read correctly, pacing the His bundle above the site of LBBB often corrected it to produce a narrow paced QRS. Fantastic!

I was invited to attend a His bundle pacing course at Geelong in July 2017. We observed several cases and attended lectures outlining the technique. I was excited to return to Sydney and offer it to my patients, but my initial enthusiasm quickly waned. I was discouraged that the "landing site" for the His bundle lead was rather small making it a tricky implant. I also noted that the lead dislodgement rate was much higher than conventional pacing, requiring lead repositioning with its inherent high risk of infection, not to mention patient inconvenience. More importantly was the observation of increasing His capture threshold over time and the potential for non-capture, an unacceptable outcome for pace dependant patients with complete heart block. Another frequent observation was that loss of His bundle capture resulted in capture of the septal myocardium, so the benefit of physiological pacing was lost, despite all efforts.

Pacemaker follow up is also time consuming as there is a necessity to confirm His bundle capture using a 12 lead ECG, not to mention issues arising from lead over- and under sensing. My conclusion was that although His bundle pacing is the most elegant solution in theory, it is not so in practice.

In November 2019, I had the opportunity to visit Fuwai Hospital, National Cardiovascular Centre, in Beijing China. This impressive true "centre of excellence" is focussed solely on the management of cardiovascular disease. The facility boasts 700 beds, 12 operating rooms, 2 hybrid operating rooms and 10 interventional catheterization rooms. On offer was an excellent training program for left bundle area pacing (LBAP), an alternative method for conduction system pacing.

LBAP involves advancing a lead just beyond the tricuspid valve, positioned perpendicular to the basal right interventricular septum and then vigorously advanced into the septum aiming to penetrate the myocardium to reach the left bundle branch (see figures A and B). The landing site is larger than the His region allowing for a simpler procedure. The resultant lead position is stable and lead parameters exhibit excellent long term sensing and low capture threshold. Furthermore, follow up is no different to a standard pacemaker and physiological pacing is effectively achieved. I excitedly returned home only to suffer the worst "plane flu" I had ever experienced which almost knocked me off my feet for the next 2 weeks. The world has since changed, but at the time, I took some OTC meds, avoided intubation and "soldiered on".



(continued)

Figure A. The His bundle is a small and discrete area. Positioning a lead at this location can be difficult due to instability, risk of dislodgement and increasing long term capture threshold. As it lies in a position between the atrium and the ventricle, there are often issues with over- or under sensing which may compromise device function. The LBB landing area is larger, as the left bundle branch fans out after its bifurcation beyond the distal His bundle deep within the septum. Consequently, it is easier to locate and lead stability and long-term parameters are superior to His bundle pacing.

Figure A.

### Left Bundle Branch (LBB) Pacing

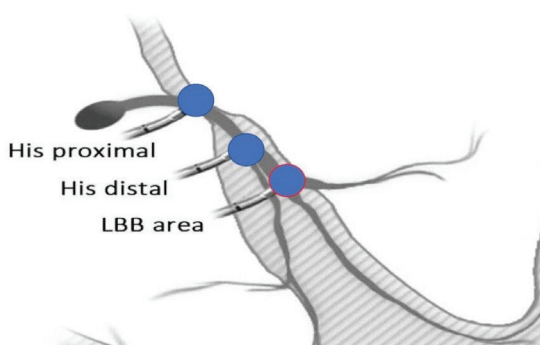
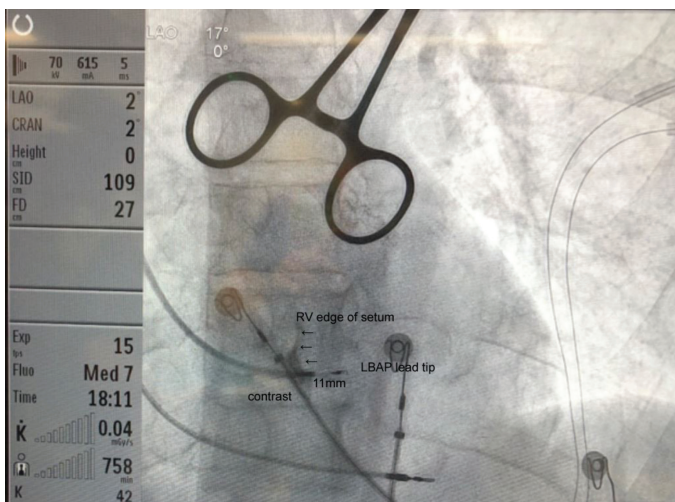


Figure B

This LAO image shows the final tip position of the LBAP lead within the interventricular septum. Note that contrast is being injected into the long sheath which has delivered the lead to the septum. The contrast delineates the right side of the interventricular septum and clearly demonstrates that the lead has penetrated the septum towards the left bundle branch. The distance between the lead helix tip to the proximal electrode is 11mm. The lead shown at the RV apex was later repositioned to the right atrial appendage and acts as back up pacing during the procedure.

Figure B.

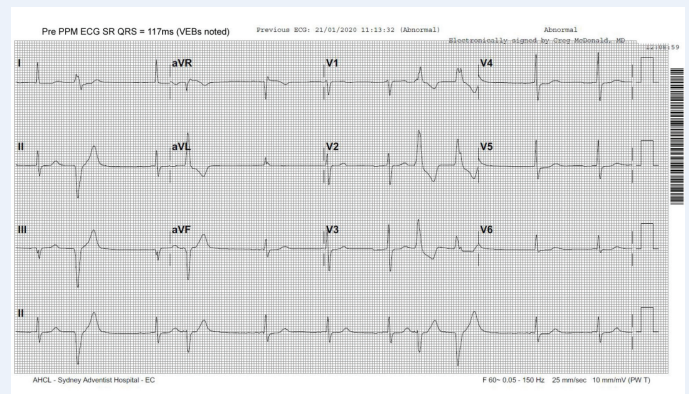


## Case Study:\*

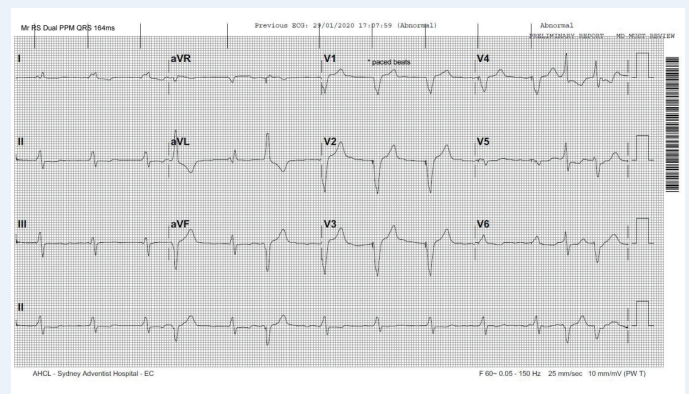
Mr RS is an 84yo man with idiopathic dilated cardiomyopathy, paroxysmal atrial fibrillation, and bradycardia.



His baseline ECG shows SR (with VEBs). Note the intrinsic narrow QRS width of 117ms



A Biotronik dual chamber pacemaker was implanted in January 2020 for symptomatic bradycardia. The post pacemaker ECG shows the RV paced beats have a QRS width of 164ms (the lead was positioned at the mid RV septum).



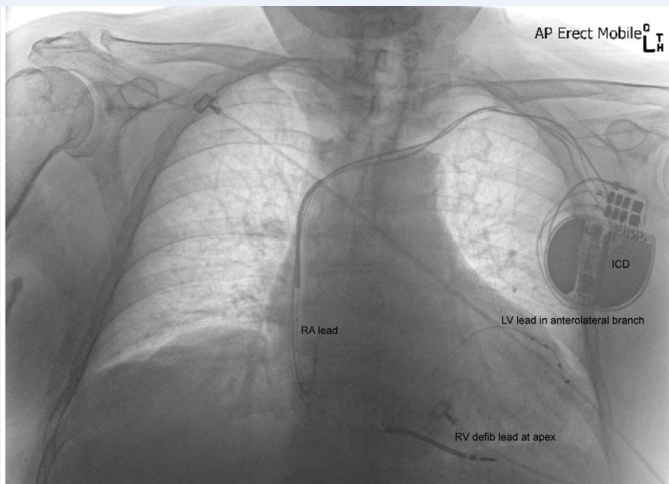
He subsequently developed heart failure with NYHA class III symptoms. Echocardiography revealed severe LV dysfunction with ejection fraction 32% and increased LV diastolic diameter of 6.2cm.

In November 2020, he was referred for CRT defibrillator upgrade of his existing pacemaker in an attempt to improve LV function and as primary prophylaxis for ventricular arrhythmias.

The new RV defibrillation lead was positioned at RV apex and the existing RV pacing lead was removed. Coronary sinus venography showed three potential targets, but two were not suitable (corkscrew origin and short vessel with distal tapering). A quadripolar LV lead was placed within the remaining anterolateral branch. However, phrenic nerve stimulation was noted when pacing poles 2, 3 and 4 but none when pacing pole 1 to RV coil. The threshold was excellent, and the lead position was accepted as there were no alternative targets.

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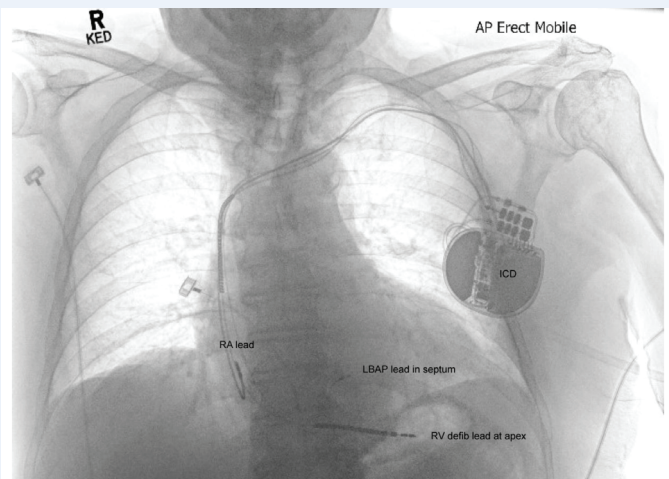
## CXR post CRT ICD upgrade



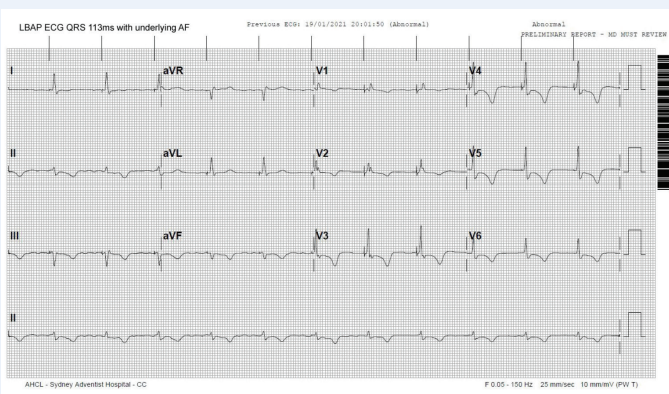
In December 2020 at the first ICD check, he reported recurrent intolerable phrenic nerve stimulation which could not be eliminated by programming. The LV lead was deactivated.

In January 2021 he proceeded to LBAP lead placement. The LV lead had not dislodged which may have otherwise caused phrenic stimulation and was subsequently extracted. The LBAP lead was positioned successfully.

## CXR post LBAP lead placement and removal of LV lead.



The ECG after LBAP upgrade shows underlying atrial fibrillation with narrow paced QRS complexes measuring 113ms. The morphology is similar to the intrinsic QRS before pacing (note the RS' pattern in V1).



At the February 2021 ICD check, LBAP lead parameters were stable and the patient reported subjectively improved symptoms.

At the October 2021 ICD check, LBAP threshold was 0.8mV. He felt subjectively much improved and echocardiography showed a significant objective improvement with LV ejection fraction 45% and a reduced LV diastolic diameter of 5.7cm.

## Discussion.

As exemplified in the case above, effective resynchronisation can be achieved using physiological pacing. The pacing system is inherently simpler as it requires a single ventricular lead positioned at the left bundle area rather than a separate LV lead in the coronary sinus. Ironically, the most frequently used lead today for His bundle and LBA pacing is the very same lead used by my supervising professor in London almost 20 years ago, so the technology has been tried and tested. LBAP offers no disadvantage to pacing follow up, the pacing systems are technically MRI compatible and the implant procedure is relatively straight forward.

## Conclusion.

Despite hundreds of studies aiming to improve the responder rate for CRT using complex and time consuming echocardiographic measurements in years gone by, none were proven to be significantly better in predicting CRT response compared to a narrow paced QRS after device implant. CRT rarely results in a paced QRS morphology as close to normal as does physiological pacing. Early emerging data suggests that physiological pacing is superior to CRT with respect to improved LV function and that LBAP is not inferior to His bundle pacing in that regard. By preventing electrical dyssynchrony, physiological pacing will also likely eliminate the occurrence of pacemaker induced cardiomyopathy.

Watch this space – I'm excited!



# Our services

Sydney Cardiology is a world class comprehensive cardiology service, delivered with expertise and experience. Using state of the art diagnostic equipment in all five clinic locations, Sydney Cardiology strives to provide exemplary outcomes for long term patient care.

## Urgent access

We provide same-day urgent appointments and 24/7 on-call support for GPs with a dedicated phone number, **02 9966 7700**.

## Non-invasive testing

Including stress-echocardiography, echocardiography, holter monitor studies, ambulatory blood pressure studies, coronary calcium score, dobutamine stress echo, electrocardiogram and event monitor recording.

## Echo, ABP, and holter monitor-only referral services

We provide echo-only, ABP-only, and holter monitor-only referral services, with a summary report on any adverse findings.

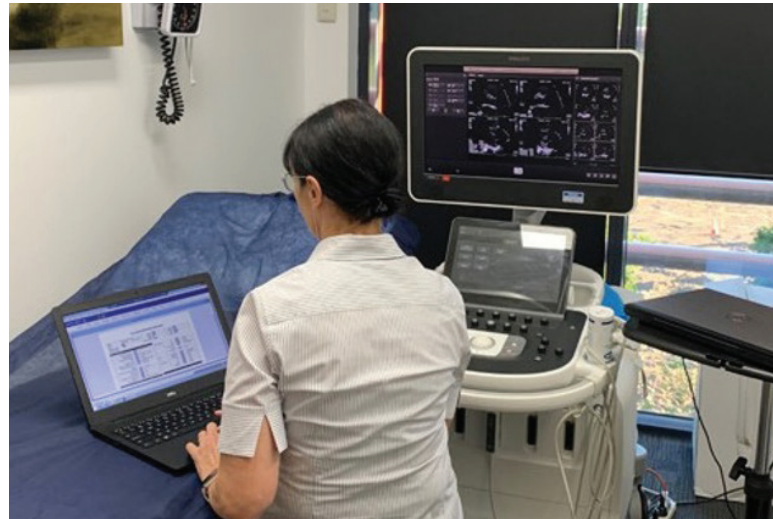
## Electrophysiology

Including diagnostic electrophysiology studies, ablation of cardiac arrhythmias, cardiac device implantation, pacemakers and defibrillators, and follow up of implanted cardiac devices.

## Cardiac procedures

Including coronary angiography, cardiac biopsies, right heart catheterisation, transesophageal echocardiogram and coronary angioplasty.

Including renal and lower limb angioplasty, ankle brachial index and SphygmoCorR central blood pressure testing.



## ECG fax service

For urgent advice, 12-lead ECGs can be faxed to our locations.

Bella Vista - Fax: 02 9672 6214

Blacktown - Fax: 02 9676 8900

Chatswood - Fax: 02 9411 1904

Parramatta - Fax: 02 9635 1247

Sydney City - Fax: 02 9422 6081

## Peripheral vascular services

Including renal and lower limb angioplasty, ankle brachial index and SphygmoCorR central blood pressure testing.

## In-hospital care

All patients with appropriate private health coverage undergoing hospital procedures, do not incur any out-of-pocket costs. Sydney Cardiology has access to leading private hospitals, including:

### **Sydney Adventist Hospital**

Wahroonga

### **Norwest Private Hospital**

Bella Vista

### **Macquarie University Hospital**

North Ryde

### **Northern Beach Hospital**

Frenchs Forest

## Patient fees

Sydney Cardiology is a private clinic however there are no out of pocket costs for Pensioners and Department of Veterans Affairs patients.

## Referrals

To request a referral pad, click [here](#)

# Statin therapy and coronary artery plaque progression



## Dr Abhinav Luhach

Specialising in general adult cardiology, cardiac CT, and preventive cardiology

### What effect does statin therapy have on coronary artery plaque progression and composition?

Intuitively, cardiologists have long felt it is right to offer statin therapy to patients with raised CT coronary calcium scores. However, as I have written before there is not much evidence to support this widespread practice. This is one reason why I suspect it is only recently that CT calcium scoring has made its way into risk assessment guidelines (both international, and more recently local).

In this context, the study by Rosendaal and colleagues published in JAMA<sup>1</sup> is reassuring to cardiologists and patients alike. It builds on the work of a few prior similar studies and also fits in neatly with our understanding about the importance of coronary artery plaque composition and the difference between vulnerable plaque and stable plaque.

This was a multinational study involving approximately 850

patients who underwent serial CTCA, 2 or more years apart. The main outcome was progression of plaque composition of individual coronary plaques. The mean age of patients was 62 years and 64% received statin therapy. Plaques were characterised as being in 1 of 6 different categories based on their density, as determined by Hounsfield units (HU) by CT.

Plaques with the low density were labelled as being either low attenuation plaques (HU <75) or fibrofatty plaques (HU 76-130). Cardiologists are most worried about these so-called "soft plaques" that are non-calcified and prone to rupture. It is these vulnerable plaques most associated with acute coronary syndrome. On the other end of the spectrum, plaques were labelled as being high-density (HU 701-1000) or simply as 1K plaques (HU >1000), which are likely to be more stable.

The study showed that in patients not treated with statins, all types of coronary plaque increased with time. Those patients receiving statins had decreases in the volume of their low attenuation and fibrofatty plaques, but also greater progression of both high density and 1K plaques. The results suggest that statin therapy is associated with transformation of coronary artery plaque towards high density calcium.

This study also illustrates why it is not clinically useful to repeat CT CAC scores serially in patients, if the initial result is elevated. We should also remind patients of the important role statins have in influencing plaque progression and in plaque stabilisation.

1. Rosendaal AR et al. JAMA Cardiol 2021;6(11):1257-66.

## Clinic locations

All clinics have emergency appointment timeslots available for same-day referrals. Contact any of our clinics directly for more assistance.

### Bella Vista

Suite 213, Q Central,  
10 Norbrik Drive,  
Bella Vista NSW 2153

Tel: 02 9422 6000 | Fax: 02 9672 6214

### Chatswood

Suite 901, Level 9, Tower B,  
799 Pacific Highway,  
Chatswood NSW 2067

Tel: 02 9422 6040 | Fax: 02 9411 1904

### Sydney City

Suite 102, Level 1,  
37 Bligh Street,  
Sydney NSW 2000

Tel: 02 9422 6080 | Fax: 02 9422 6081

### Blacktown

Suite 4,  
15-17 Kildare Road,  
Blacktown NSW 2148

Tel: 02 9422 6050 | Fax: 02 9676 8900

### Parramatta

Level 5 Suite 501, B1 Tower,  
118 Church Street,  
Parramatta NSW 2150

Tel: 02 9422 6060 | Fax: 02 9635 1247

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Call (02) 9966 770 for  
specialist advice

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