SPRING 2019

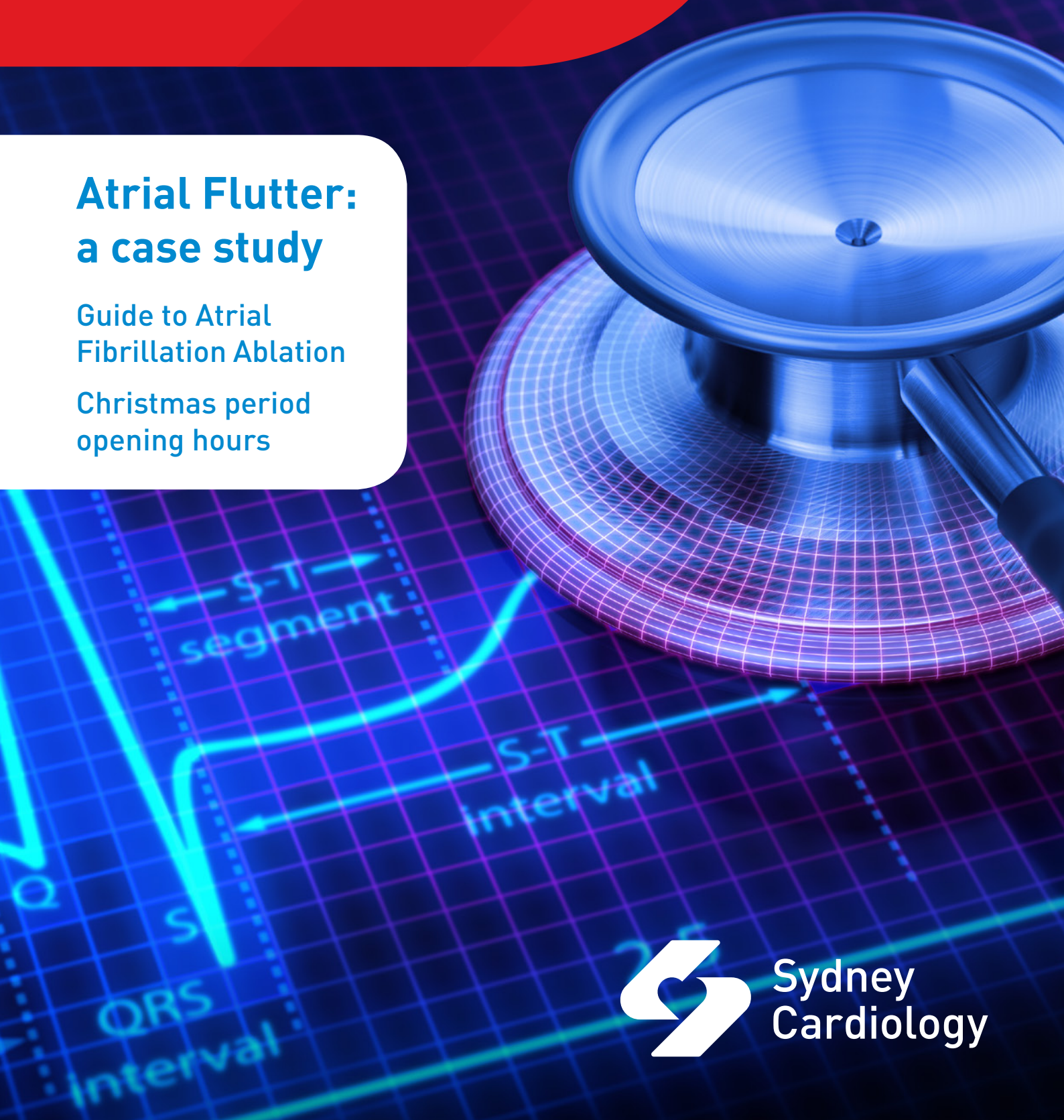
GP Connect

CARDIOVASCULAR CLINICAL UPDATE

Atrial Flutter: a case study

Guide to Atrial Fibrillation Ablation

Christmas period opening hours



Sydney Cardiology

WELCOME

From the editor – Dr Andrew Terluk

Thank you for reading our Spring edition of GP Connect. In this edition we get 'back to the basics' with an overview of atrial fibrillation and atrial flutter – hopefully the articles help add clarity in your clinical practice when managing these common conditions.

MEET OUR TEAM

We have experienced cardiologists in all major sub specialities to provide the highest quality of patient care. Our Sydney Cardiology team includes:

Dr James Wong

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

Dr Bill Petrellis

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

Dr Fiona Foo

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

Dr Gunjan Aggarwal

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

Dr Abhinav Luhach

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

A/Prof Martin Brown

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

Dr Ru-Dee Ting

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

Dr Andrew Terluk

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

OUR CARDIOLOGISTS PROVIDE ON-CALL SUPPORT 24/7 FOR GPs
PLEASE CALL 02 9966 7700

SAME DAY URGENT APPOINTMENTS

ATRIAL FLUTTER – A CASE STUDY

Dr Abhinav Luhach

A 67 year old male presents to you with a two month history of increasing lethargy and shortness of breath on exertion. He has felt a bit light headed at times during this period but has not had any palpitations or chest pain. He does not report cough, fevers or haemoptysis. He is a non-smoker and not had any recent overseas travel. He is overweight (BMI 34) and is on Irbesartan for hypertension. He drinks 1-2 standard drinks of alcohol 3 times a week.

On examination blood pressure is 134/82mmHg and heart rate 146bpm. There are no murmurs. Chest clear, jugular venous pressure is not raised and there is no lower limb oedema.

An ECG was performed. What does this show?

A. Atrial fibrillation

B. Atrial flutter

C. Sinus tachycardia

D. Left bundle branch block

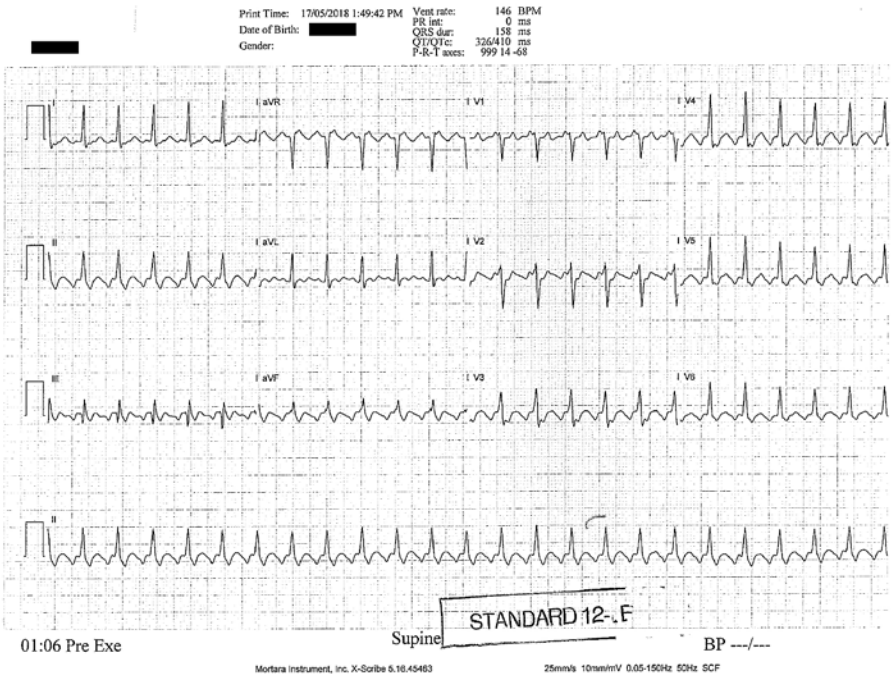
E. Ventricular tachycardia

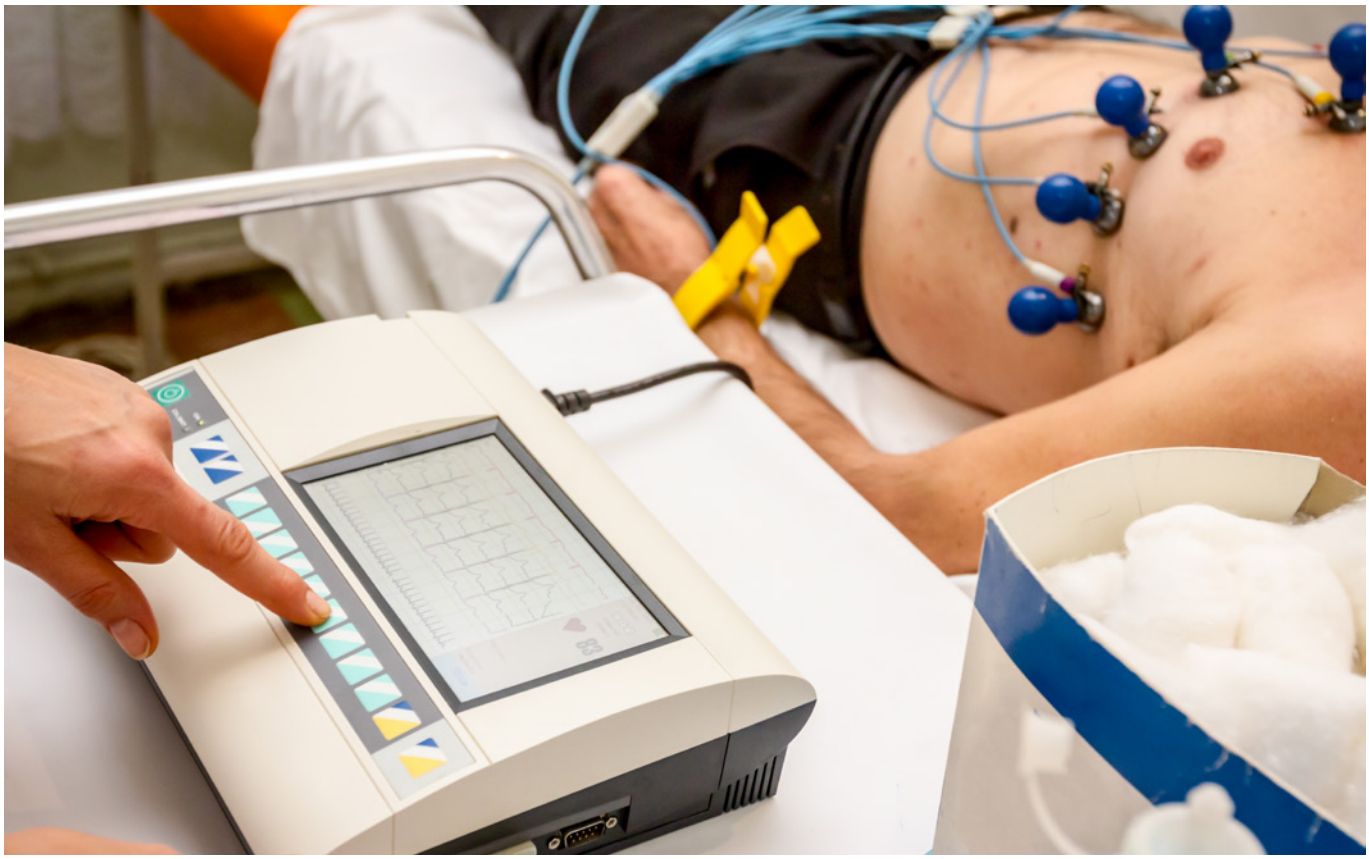
In atrial fibrillation there is chaotic atrial activity characterised by the absence of discrete P waves and an irregularly irregularly ventricular response. There are small, irregular baseline undulations of varying amplitude and morphology representing atrial electrical activity.

With atrial flutter there are sawtooth flutter waves with continuos electrical activity (ie. no isoelectric line between the flutter waves). The flutter waves are all of same morphology. They are easiest to see in leads II, III, aVF and V1. With typical flutter (the most common pattern) the flutter waves are negative (inverted) in these leads. The atrial flutter rate is ~300/min and because of the effect of the AV node the ventricular rate is slower, classically 150/min (ie. 2:1 flutter) as is the case here, but can be 4:1 or even have variable block (in which case the ventricular rhythm is actually irregular) A higher grade of block may signify conduction disease of the AV node or the effect of rate lowering medication. Hence the answer is B.

In sinus tachycardia the heart rate is >100/min. P waves are present before each QRS complex and the PR interval is constant. The contour of the P wave is normal, although amplitude may be greater.

LBBB has a characteristic QRS morphology which also requires it to be widened (>120msec) which is not the case here. VT requires at least 3 consecutive, abnormal and widened QRS complexes which are dissociated from atrial activity.





WHAT TREATMENT WOULD YOU INSTITUTE?

Whilst symptomatic this patient is not compromised by his arrhythmia. Blood pressure is satisfactory and examination does not reveal signs of heart failure. Therefore initial aims of management are twofold- 1) rate or rhythm control and 2) stroke prevention. It would also be important to identify and treat any potential triggers for arrhythmia (see below) such as infection or electrolyte imbalance. Whilst there is some variation in practice between clinicians as to the medications used the principles outlined above do need to be followed.

For rate control I would start by using a beta blocker (such as metoprolol) provided there are no contraindications. Alternative classes of medications that can be used are non-dihydropyridine calcium channel blockers (diltiazem or verapamil) or digoxin (generally reserved for the elderly or as add-on therapy, but also useful in heart failure).

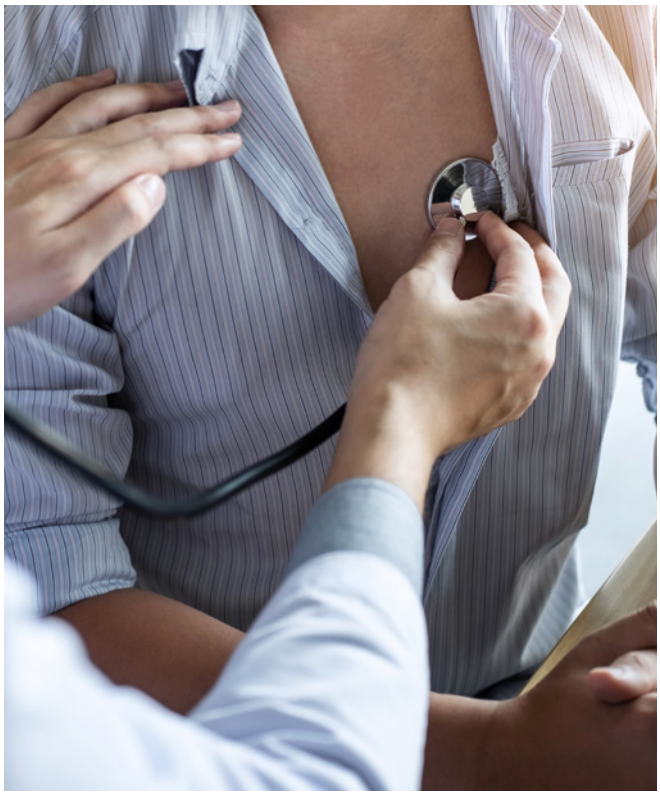
Anti-arrythmics (such as amiodarone or flecainide) can be used with the aim of restoring sinus rhythm but clinicians need to be familiar with potential adverse effects and contra-indications of these medications. In this situation of new onset arrhythmia a common approach would be for initial rate control whilst planning for an elective electrical cardioversion. This strategy has higher rates of achieving sinus rhythm. Anti-arrythmics (eg. sotalol) can also be used to maintain sinus rhythm once it has been achieved following electrical cardioversion.

In all cases of atrial fibrillation or flutter it is important to calculate the stroke risk. This is preferably done via the CHA_2DS_2-VA score (calculators are available online or as a smartphone app). A score of greater than 1 is an indication to anticoagulate although physicians should considering anticoagulating patients with a score of 1. Anticoagulation is done via a NOAC or less commonly these days with warfarin. When commencing anticoagulation is important to consider the patient's bleeding risk and renal function (use creatinine clearance). NOACs should not be used for valvular AF

WHAT INVESTIGATIONS WOULD YOU PERFORM?

These are generally aimed at identifying underlying conditions that might precipitate arrhythmia. General baseline investigations would include renal function/electrolytes, FBC, liver function tests and a TSH level. Given this patient's presentation a chest x-ray would be appropriate.

An echocardiogram is also warranted to check for any underlying structural heart disease (such as heart failure/cardiomyopathy or mitral valve disease). Left atrial size is also relevant in atrial arrhythmias.



Further testing depends on the clinical scenario. Here, a sleep study may be useful given the association between obesity, hypertension and atrial arrhythmias. In other settings it might be appropriate to investigate for pulmonary embolus depending on the presence of risks factors and the clinical suspicion.

HOW DOES ATRIAL FLUTTER DIFFER FROM ATRIAL FIBRILLATION?

From a practical standpoint there is not much difference in initial management of the two conditions. The broad principles of management (rate/rhythm control and anticoagulation) and the drugs used to achieve these are the same.

However there are significant electrophysiological differences. In the majority of cases, atrial flutter is due to a reentry circuit around the tricuspid annulus in the right atrium. The circuit runs counterclockwise (resulting in "typical" flutter) or less commonly in a clockwise direction. In contrast, atrial fibrillation is a continuous and disorganised electrical rhythm.

Atrial flutter can be more difficult to rate control with medications than atrial fibrillation. On the other hand, it is generally easier to electrically cardiovert atrial flutter back to sinus rhythm compared to atrial fibrillation although there other variables that determine the success rate of cardioversion. The major difference in management is because of the well defined, organised reentry circuit in atrial flutter, catheter ablation has an excellent long term success rate. With atrial fibrillation, ablation is done with the aim of electrically isolating the pulmonary veins. This procedure is more prolonged and has lower success rates than flutter ablation. Sometimes another repeat ablation procedure may be required for fibrillation. Finally it is not uncommon for patients to have both atrial arrhythmias.

CHRISTMAS PERIOD OPENING HOURS

Over the Christmas/New year period, Sydney Cardiology rooms are open at the following locations.

Please call our rooms to make an appointment.

For the on call cardiologist, please call our pager service on 9966 7700.

MON	TUES	WED	THU	FRI
16 Dec All Locations	**17 Dec** All Locations	**18 Dec** All Locations	**19 Dec** All Locations	**20 Dec** All Locations
23 Dec Chatswood Bella Vista	**24 Dec** Chatswood Bella Vista	**25 Dec** Christmas Day **PUBLIC HOLIDAY** Closed	**26 Dec** Boxing Day **PUBLIC HOLIDAY** Closed	**27 Dec** Chatswood Bella Vista
30 Dec Chatswood Bella Vista	**31 Jan** Chatswood Bella Vista	**1 Jan** New Year's Day **PUBLIC HOLIDAY** Closed	**2 Jan** Chatswood Bella Vista	**3 Jan** Chatswood Bella Vista
6 Jan All Locations	**7 Jan** All Locations	**8 Jan** All Locations	**9 Jan** All Locations	**10 Jan** All Locations

Sydney Cardiology

WISHING YOU A JOYFUL FESTIVE SEASON

LISTEN TO YOUR HEART

Sydney Cardiology

WEDNESDAY NOVEMBER 20

Hilton Hotel Sydney

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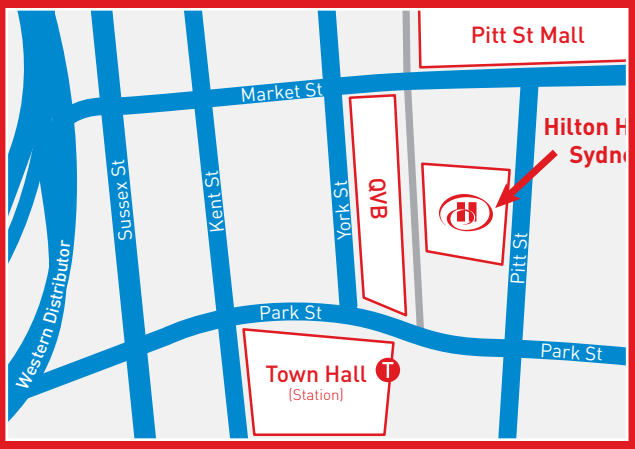


As part of our ongoing commitment to providing quality education, Sydney Cardiology Group is pleased to invite you for canapés and drinks at our next GP Education event.

WHEN **Wednesday 20th November –** 5:30pm-7:30pm

WHERE **Hilton Hotel Sydney –** 488 George Street, Sydney NSW 2000

Speakers

Dr Fiona Foo

Females at Risk; Non Traditional Risk Factors for Heart Disease in Women

Dr Andrew Terluk

How to perform a Cardiac Check Up

Dr Abhinav Luhach

Coronary Artery Calcium Scoring & CT Coronary Angiograms

Registration

Please email **events@sydneycardiology.com.au** or phone **9422 6080** by Wednesday 30th October 2019.

GUIDE TO ATRIAL FIBRILLATION ABLATION

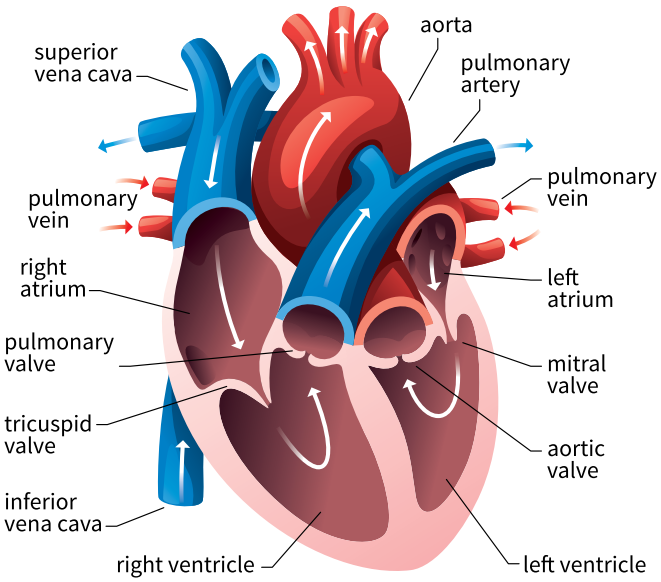
Dr Bill Petrellis

STRUCTURE OF THE HEART

The heart has 4 chambers. The right and left atria (upper chambers) collect blood to fill the right and left ventricles (lower chambers) which in turn pump blood to the body and lungs.

The lungs provide oxygen to the blood with breathing. This "red" blood returns to the left atrium by 4 pulmonary veins (2 from each lung). Blood passes from the left atrium to the left ventricle which pushes blood from the heart to the body including brain, vital organs and muscles. The organs use up the oxygen and this "blue" blood returns to right heart. The right atrium collects the blood, passes it to the right ventricle and then on to the lungs again for oxygen.

The heart beats because of electrical signals it receives. Normally, the sinus (sinoatrial) node within the right atrium, regulates the heart rhythm by sending an impulse through the upper chambers causing them to squeeze ("lub"). This impulse then travels to the lower chambers via the AV node, causing the lower chambers to squeeze ("dub"), usually 60 to 80 times every minute. This upper and lower sequence ("lub-dub") represents one heart beat.



WHAT IS ATRIAL FIBRILLATION?

Atrial fibrillation is a rhythm disturbance of the upper chambers and occurs when the left and right atrium no longer beat in an organised manner. During atrial fibrillation, the sinus node is suppressed by rapid erratic electrical activity (fibrillation) causing the upper chambers to beat between 300-600 times every minute. Fortunately, the AV node functions as an "electrical filter" and does not allow all of these beats to get to the ventricles. In atrial fibrillation, the ventricles often beat between 80 to 180 beats per minute. Atrial fibrillation may cause palpitations, light headedness, fatigue, shortness of breath or reduced ability to exercise or perform daily activities. Symptoms are due to heart rhythm irregularity and increased heart rate.

HOW IS ATRIAL FIBRILLATION TREATED?

There are two approaches for the treatment of symptoms of atrial fibrillation. One approach is to restore and maintain sinus rhythm and the other is to simply control the rate of the ventricles during atrial fibrillation.

The first approach (rhythm control) is achieved by using heart rhythm medications (anti-arrhythmic drugs) that are between 40-65% effective at maintaining normal rhythm. These medications work at the atrium to suppress atrial fibrillation and restore normal sinus node function. Often, the heart must be electrically "reset" by an external shock known as cardioversion if medication does not work in the first instance. Cardioversion is performed in hospital under brief general anaesthesia. Anti-arrhythmic medication is usually continued in order to maintain sinus rhythm. Examples of such medications include sotalol (Sotacor), flecainide (Tambocor, Flecatabs) and amiodarone (Cordarone, Aratac).

The second approach (rate control) is achieved by medications such as beta- blockers, calcium channel blockers or digoxin which prevent the heart from going too fast. Such medications include metoprolol (Tenormin), bisoprolol (Bicor), atenolol (Noten), diltiazem (Cardizem), verapamil (Isoptin) and digoxin (Lanoxin). These medications work at the AV node to allow fewer impulses to travel from the atrium to the ventricle. The heart rate is slowed but remains irregular as atrial fibrillation continues.

Although it seems intuitive that the first approach should be more effective, this has not been demonstrated in large research studies. The appropriate treatment of atrial fibrillation is therefore individualised.

Atrial fibrillation itself does not put one at risk of dying suddenly or having a heart attack. It does, however, carry an increased risk of stroke. Individuals who are considered to be at moderate or high risk of stroke should receive an anticoagulant (blood thinner) such as warfarin (Coumadin, Marevan), dabigatran (Pradaxa), apixaban (Eliquis) or rivaroxaban (Xarelto) to reduce this risk. Individuals who are considered to be at low risk of stroke do not require an anticoagulant or aspirin.

ATRIAL FIBRILLATION ABLATION

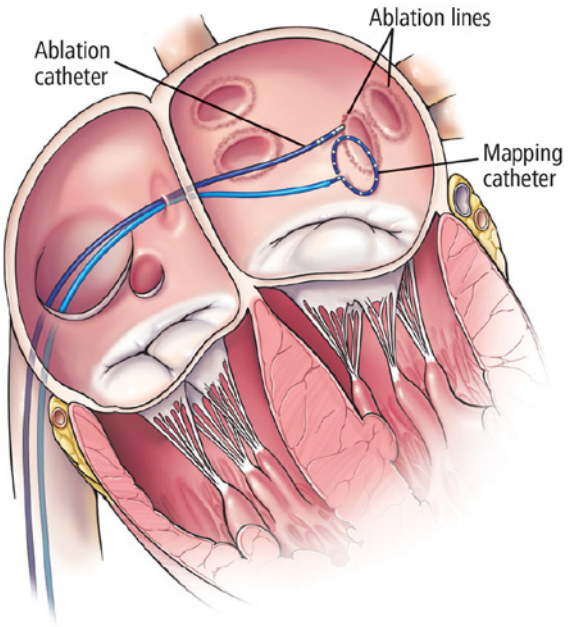
Treatment with drugs is the first line of therapy for atrial fibrillation. However, many patients are not happy with drug treatment. Drugs may not control symptoms or may cause side effects. Other patients wish to avoid medications for as long as possible.

Catheter ablation is an alternative to medication for arrhythmia management and has been widely used since 1990 for a variety of arrhythmias. Catheter ablation for atrial fibrillation has been used since 2000 and is ever-evolving with new technologies and techniques.

Ablation of atrial fibrillation involves the creation of scars within the left atrium to stabilise electrical short circuits. These scars are created by catheters (wires or balloons) that are introduced into the heart via the leg veins at the groin. Usually, 3 to 4 catheters are located within the heart recording electrical activity, one of which has the ability to create scars in the heart using either radiofrequency (RF) ablation (heat / cautery) or cryoablation (freezing).

Currently, it is thought that most atrial fibrillation is initiated by abnormal rapid electrical firing within the pulmonary veins that return "red" blood from the lungs to the left atrium. The ablation procedure involves burning or freezing around the 4 pulmonary veins to prevent this abnormal electrical activity entering the heart, thereby preventing atrial fibrillation. Some patients require the creation of additional scars at other areas to prevent atrial fibrillation.

The ablation procedure is performed in a laboratory with several staff including a specialist in cardiac rhythm disturbance (electrophysiologist), nursing and technical staff. It usually takes between 2 and 4 hours and is performed under general anaesthesia. Local anaesthetic agent is used at the groin allowing painless insertion of the catheters which are then guided to the right atrium under x-ray vision. The ablation catheter is passed from the right to the left atrium by creating a small hole in the wall that separates them (atrial septum) thereby allowing access to the pulmonary veins. This small hole usually heals spontaneously within a few weeks without consequence.



WHAT ARE THE SUCCESS RATES?

It is important to understand that there is no "forever" cure for atrial fibrillation and that ablation is not effective for all people. The goal of treatment is to achieve a lower symptom burden and an improved quality of life. The success rate of ablation is approximately 70% and the definition of "success" ranges from feeling better but still needing medication, to being free of atrial fibrillation and off medication. About 30% of patients require more than one procedure which adds an additional 10% benefit. Importantly, the majority of patients undergoing ablation still require a lifelong blood thinner as the risk for stroke is not reduced by ablation, despite apparent success.

WHAT ARE THE DOWN SIDES?

Although ablation procedures are generally safe, ablation for atrial fibrillation is associated with serious and potentially life threatening risks. Candidates therefore require careful selection and must be fully informed of the risks and outcomes before agreeing to the procedure.

- Bleeding or bruising from the site where tubes are inserted at the groin 3-5% (includes haematoma, inadvertent artery injury (laceration / dissection / false aneurysm), deep vein thrombosis
- Heart perforation with cardiac tamponade (bleeding around the heart) 1%
- Blood clot formation that could lead to stroke or lung embolus 0.5 - 1%
- Pulmonary vein stenosis (narrowing damage) 1%
- Damage to the "AV node" and requiring a pacemaker 1%
- Atrio-oesophageal fistula (traumatic hole from the heart to oesophagus) 1 in 5000 (radiofrequency ablation) which is often fatal
- Phrenic nerve paralysis (injury to nerve of diaphragm) 2 - 6% (cryoablation)
- Gastic dysmotility (injury to nerves of the stomach) 1%
- Worsening of arrhythmia (esp. in first 3 months) e.g. left atrial flutter - 5%
- Failure of procedure and need for re-do ablation 30%
- Other: infection, myocardial infarction (heart attack)
- Rarely, complications that lead to emergent surgery or death

It is emphasized that the procedure is aimed at symptomatic improvement, is elective, non-lifesaving and with infrequent but potentially life threatening risks.

WHO IS A CANDIDATE?

Ablation for atrial fibrillation is generally reserved for patients who have symptoms that significantly impact their lives despite drug therapy. Other factors such as age, duration of atrial fibrillation, size of the heart chambers, previous stroke or other medical conditions may play a role in deciding whether you are a suitable candidate.

WHAT PREPARATION IS REQUIRED BY THE PATIENT?

Anticoagulation is required for at least one month prior to the ablation procedure and will be discontinued beforehand. Warfarin is usually stopped 5 days in advance and is replaced by a short acting injectable anticoagulant called Clexane 2 days prior. Newer anticoagulants may continue until two days prior without the need for Clexane.

A transoesophageal echocardiogram (TOE) is routinely performed under anaesthesia immediately before the procedure is commenced. This internal cardiac ultrasound is performed to exclude a blood clot within the heart which might otherwise be disturbed by the catheters during the procedure causing a stroke.

A cardiac CT scan is required to create a 3-dimensional model of the left atrium and pulmonary veins. It provides critical information that is needed for the selection of catheters and technique to ensure a successful and safe procedure. It should be performed 2 weeks in advance and a specific referral will provided.

PATIENT AFTERCARE?

After ablation, the catheters are removed and a suture is placed at the groin for 2 hours to reduce the likelihood of bleeding. You will be transferred to the coronary care unit where you must lie flat for several hours to allow the puncture sites to seal.

Nursing staff will administer an injection of Clexane overnight. Newer anticoagulants such as Pradaxa, Eliquis, Xarelto are started the following day. Warfarin is recommenced on the evening of the procedure and self administered Clexane injections are required for at least 3 days while the warfarin level (INR) is restored.

An overnight hospital stay is required and most patients are discharged the following day. You may return to your usual activities by day 3 to 5.

Blood thinners will continue for a minimum period of 8 weeks after ablation and will continue indefinitely in most patients. Antiarrhythmic medications used to control your heart rhythm may be withdrawn at 2 or 3 months following ablation but this will be determined during subsequent follow up visits.

A/Prof Martin Brown is now consulting at Chatswood.

A/Prof Martin Brown has moved his patient consulting from Bella Vista to Chatswood.

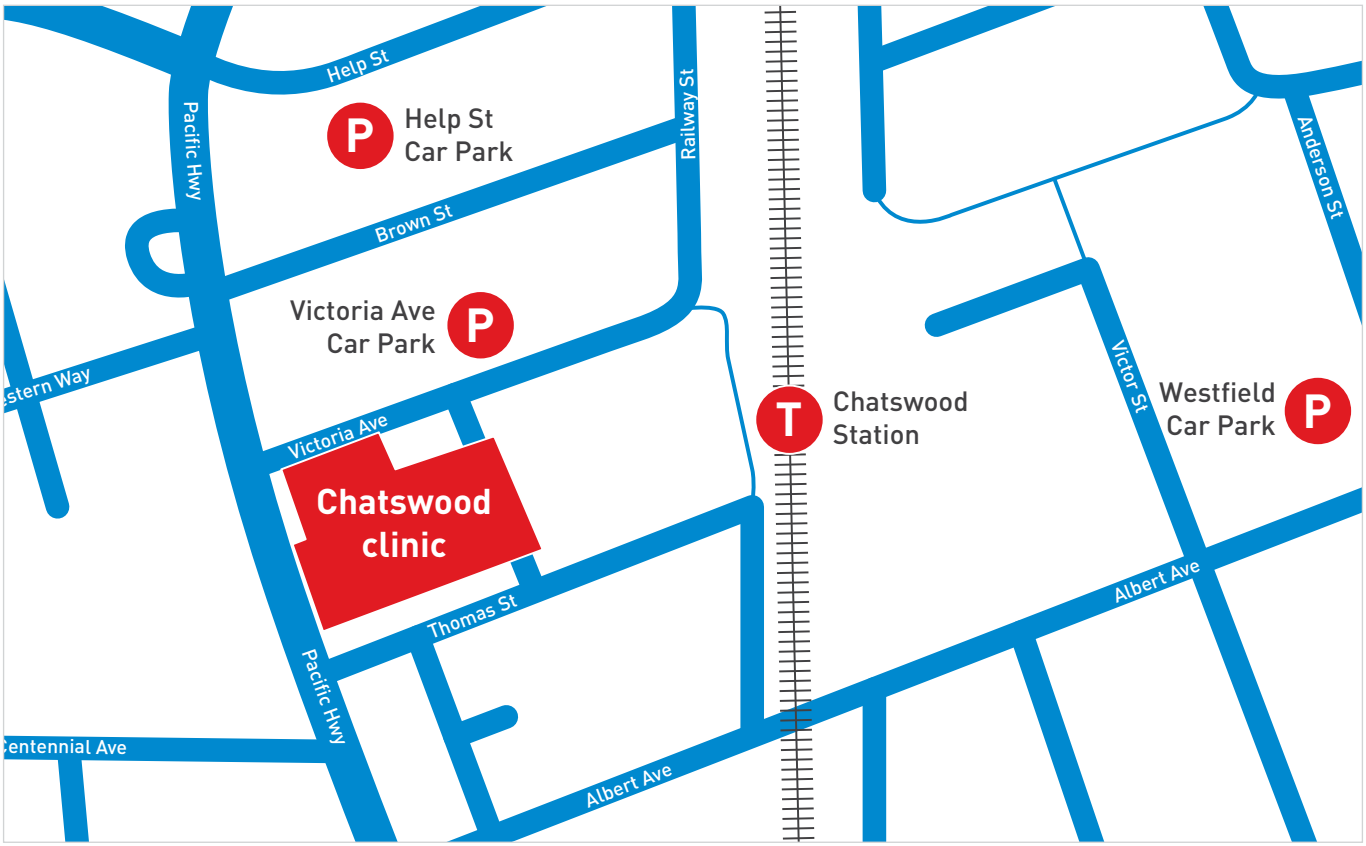
The new address is:

Suite 901
Level 9 Tower B (ACY Group)
799 Pacific Highway
Chatswood 2067

Chatswood contact details:

Phone (02) 9422 6040
Fax (02) 9411 1904

Referrers and patients can now consult with A/Prof Martin Brown at our Chatswood location. The new Sydney Metro Northwest means travelling to Chatswood is even easier. With trains arriving every 5 minutes a trip from Castle Hill Station to Chatswood Station will take less then 30 minutes.



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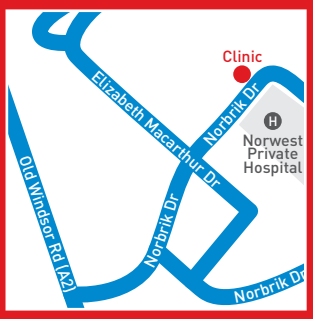
5 convenient locations across Sydney



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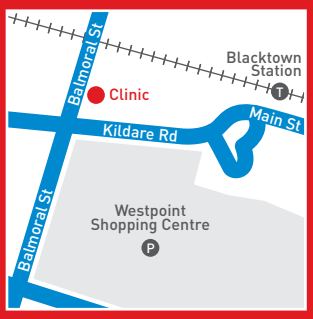

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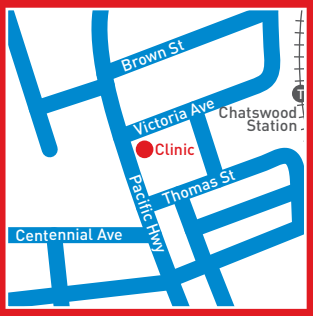

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Contact our rooms during office hours or use paging service after hours.

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Contact our after hours service on **02 9966 7700**
and ask for the Doctor on call.

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