Summer 2023

GP Connect

Supporting best practice in cardio-metabolic health

Sydney Cardiology

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 summer edition of GP Connect 2023. This issue provides the latest on managing cardio-metabolic health for General Practitioners.

In this issue, Dr James Wong discusses the latest on the role of lipid management in reducing cardiovascular risk. In the current healthcare landscape, GPs will soon be able to prescribe select PSCK9s in consultation with a cardiologist. This means patients will face fewer barriers to reaching their treatment targets.

Do you have patients with signs and symptoms of ischemia, but non-obstructive coronary artery disease on CTCA/ coronary angiogram? Interventional cardiologist Dr Fiona Foo assists with her article on identifying patients with ischemia with non-obstructive coronary arteries (INOCA).

In exciting news, our Sydney Cardiology CBD clinic location is moving a few doors down. We are still providing the same cardiac expertise, conveniently located near Wynyard station. The room can still be contacted by the current CBD clinic number and will be operational in January 2023.

Towards the end of the issue, Dr Bill Petrellis provides a valuable article on his area of expertise, electrophysiology. The case study provides insight regarding the risk of sudden cardiac arrest of individuals with Wolff Parkinson White (WPW) pattern. Specifically, how the electrocardiogram (ECG) can provide valuable information about the likelihood of rapidly conducted atrial arrhythmias and the development of ventricular fibrillation.

I hope you not only enjoy this edition of GP Connect but also the unrestricted holiday season with family and friends. We remain open over the Christmas and New Year break to provide continued care to you and your patients in any way possible. I hope you and your staff enjoy a well-earned break, and we wish you good luck in the new year.

Thank you for your continued support,

Dr Gunjan Aggarwal

In this issue

Access past issues

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What you missed!

AusDoc How to treat: Exercise and cardiology | by Dr Fiona Foo

Click here to access the article*

*GPs must register/log in to access this article

*Wolff Parkinson White

Lipids: reducing cardiovascular risk

Dr James Wong

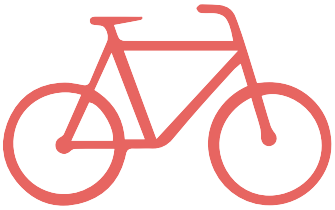
Specialising in general cardiology, prevention of coronary artery disease and hypertension.

Lifestyle cannot be ignored, and lifestyle therapy is the primary intervention for metabolic syndrome. LDL cholesterol is the dominant form of atherogenic cholesterol. The causal role of LDL-cholesterol in the development of atherosclerotic cardiovascular disease (ASCVD) is demonstrated beyond any doubt by genetic, observational, and interventional studies. The lower the LDL cholesterol the better the prognostic message continues to be reinforced by current evidence. Meta-analysis of clinical trials has indicated that the relative reduction in CVD risk is proportional to the absolute reduction of LDL cholesterol, irrespective of the drug(s) used to achieve such change.

'In all individuals, emphasise a heart-healthy lifestyle across the life course'. This was the number one message to reduce cardiovascular risk from the 2018 US Cholesterol Clinical Practice Guidelines.

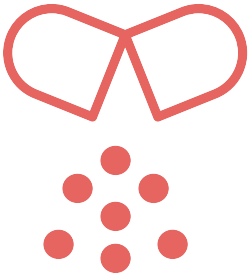
Diet

In terms of diet, it is recommended that patients should consume a dietary pattern that emphasises intake of vegetables, fruits, whole grains, legumes, healthy protein sources (low-fat dairy products, low-fat poultry (without the skin), fish/seafood, and nuts), and non-tropical vegetable oils; and limits intake of sweets, sugar-sweetened beverages, and red meats. This dietary pattern should be adjusted to appropriate calorie requirements, personal and cultural food preferences, and nutritional therapy for other medical conditions including diabetes. Caloric intake should be adjusted to avoid weight gain, or in overweight/obese patients, to promote weight loss.'



Exercise

'In general, adults should be advised to engage in aerobic physical activity 3-4 sessions per week, lasting on average 40 minutes per session and involving moderate-to vigorous-intensity physical activity.'



Pharmacotherapy

A new era in lipid management in primary care is on the horizon.

Statins remain the cornerstone of therapy among lipid-lowering drugs and are remarkably safe. There is no concern regarding safety with very low LDL cholesterol although longer-term monitoring is required. There has been an evolution of lower LDL cholesterol targets with justification for lowering LDL cholesterol coming from randomised controlled trials, imaging studies (REVERSAL, GLAGOV) and PCSK9 inhibitor clinical outcome studies (FOURIER, ODYSSEY) for patients at the highest risks, European Society of Cardiology has recommended LDL cholesterol of < 1.4mmol/L. There does not appear to be a threshold of LDL cholesterol below which no further benefit can be achieved, nor safety is compromised.

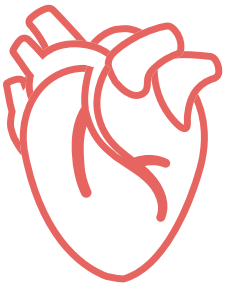
There is substantial residual CVD risk despite intensive statin therapy and there are newer non-statin agents (apart from ezetimibe) such as the PCSK9 inhibitors (evolocumab, alirocumab) which will improve prognosis. The PCSK9 inhibitors are highly effective in lowering LDL cholesterol (60-70% on top of statin therapy) and have been shown to reduce atheroma volume and significantly reduce cardiovascular events. The high cost has been a constraint but now patients with familial hypercholesterolaemia,

Lipids: reducing cardiovascular risk

GPs will soon be able to prescribe select PSCK9s in consultation with a cardiologist, meaning they have more options and face fewer barriers to helping patients achieve their treatment targets.

or non-familial hypercholesterolaemia with high LDL cholesterol despite statin therapy and ezetimibe, may qualify for PCSK9 inhibitor therapy on the PBS.

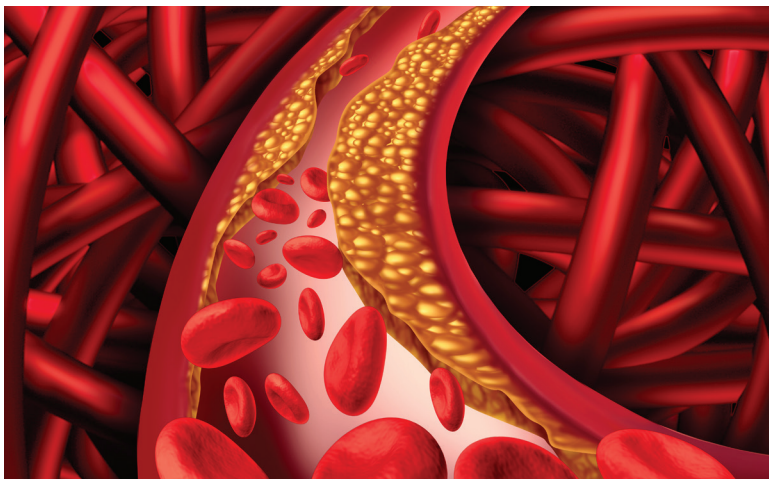
And there are more treatments on the horizon. Inclisiran, bempedoic acid, evinacumab, and Lp (a) lowering agents (pelacarsen, olpasiran) are newer agents that you will hear about. Icosapent ethyl is a pure fish oil which was shown in the Reduce IT study (4g/day) to reduce CVD events by a surprising 20-25% compared to placebo. This was a non-LDL cholesterol targeted trial to show cardiovascular benefits and future guidelines are awaited for this agent which is not yet available in Australia.

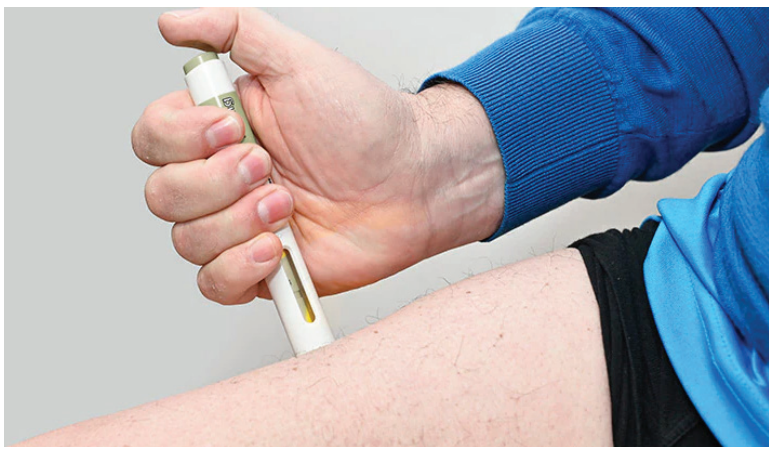


Measuring risk

For low-risk and intermediate-risk individuals, consider obtaining and using a coronary artery calcium score (CACS) for further risk stratification and decision-making. If CACS is zero, the person probably does not need statin therapy. However, this does not apply to all comers and statin therapy should still be considered in individuals with a very strong family history of early coronary events, those with high LDL cholesterol (>5.0 mmol/L) and those with diabetes. Cigarette smokers and those with chronic inflammatory illness may still be associated with substantial risk.

References. Grundy SM, et al 2018 Cholesterol Practice Guidelines, VuMedi 13th Orange Symposium for CVS disease prevention, AJGP vol49Aug2020, ESC 2021CVS prevention guidelines.





Sydney Cardiology

Our City Location Is Moving!

- Experience the same cardiac expertise
- Located near Wynyard Station
- Easily contacted on the same number 02 9422 6080

COMING JANUARY 2023
TO 68 PITT STREET

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:

Cardiology

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 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 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.

Endocrinology

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

Respiratory Medicine

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

Sydney Cardiology

Season's Greetings

During the festive season, Sydney Cardiology will be consulting at the following locations.

Patient appointments can be made through our website or by calling our rooms. In addition, our on call Cardiologist can be contacted on **9966 7700.**

MON	TUES	WED	THU	FRI
19 Dec All locations	**20 Dec** All locations	**21 Dec** All locations	**22 Dec** All locations	**23 Dec** Bella Vista Blacktown Chatswood Parramatta
26 Dec **PUBLIC HOLIDAY** Closed	**27 Dec** **PUBLIC HOLIDAY** Closed	**28 Dec** Bella Vista 8am-12pm	**29 Dec** Bella Vista 8am-12pm	**30 Dec** Bella Vista 8am-12pm
2 Jan **PUBLIC HOLIDAY** Closed	**3 Jan** All locations open*			

*The new CBD location opens on the 23rd of Jan 2023

sydneycardiology.com.au

Bella Vista	Blacktown	Chatswood	Parramatta	Sydney City Cardiology
02 9422 6000	**02 9422 6050**	**02 9422 6040**	**02 9422 6060**	**02 9422 6080**

Unexplained chest pain

Dr Fiona Foo

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

When to consider ischemia with non-obstructive coronary arteries (INOCA)

Have you ever had a patient with recurrent chest pain but normal or non-obstructive coronary arteries on a coronary angiogram/CT coronary angiogram? Is their chest pain psychological? Is it musculoskeletal? Is it gastroesophageal? Is it due to other cardiac causes e.g. pericarditis?

Consider this patient

A 55yo, thin southeast Asian female with no cardiac risk factors, and no significant past medical history; who has chest pain walking uphill. Her stress test was normal, and the subsequent CT coronary angiogram (CTCA) was normal. She had no improvement with long-acting nitrates. She has had symptoms for >1 year.

What do you do next?

*Not the patient's real image

What is INOCA?

INOCA is ischaemia with non-obstructive coronary arteries. These patients have symptoms/signs of ischaemic but non-obstructive coronary artery disease (<50% stenosis) on CTCA/coronary angiogram.[1]

Prevalence:

It is highly prevalent: >50% of women and >30% of men with non-obstructive coronary artery disease on a Coronary Angiogram and evidence of angina.[2] Up to 50% of patients with known or suspected angina have INOCA.[3] Of these, 80% are found to have microvascular and vasospastic angina when studied using specific tests.[3,4]

Prognosis:

INOCA have reported an increased risk of major adverse cardiovascular events (MACE), a threefold to fourfold increased risk of hospital admission.[5,6] A meta-analysis reported an overall estimated incidence of MACE (all-cause mortality and myocardial infarction) of 0.98 per 100 person-years, compared with 0.2 per 100 person-years reported in a similarly aged general population in North America.[3] An international registry of microvascular angina reported an annual MACE incidence of 7.7% per patient-year.[7]

These conditions are associated with impaired quality of life, as well as greater morbidity and health resource utilisation.[8,9,10] An estimated 50% of patients with stable chest pain and normal angiography will experience recurrent episodes of chest pain, similar to those with obstructive coronary artery disease.[11,12] In addition, about 15-25% of these patients are readmitted with chest pain or undergo repeat angiography (or both).[4] Patients with chest pain and nonobstructive coronary arteries have a reduced quality of life at 12 months, compared with healthy controls.[7]

Identifying INOCA patients:[13]

1. **Exclude non-cardiac causes:** eg. psychological, musculoskeletal, gastro-oesophageal, and biliary.
2. **Exclude non-ischaemic cardiac causes:** e.g., pericarditis, myocarditis, valvular heart disease.
3. **Look for ischaemia:** typical chest pain with exertion/stress; ischaemic ECG changes; abnormal myocardial perfusion on stress imaging; stress-induced regional wall motion abnormalities.

4. **Do structural coronary angiography:** invasive coronary angiogram or CT coronary angiogram. Coronary artery stenosis <50%.
5. **Do functional coronary angiography/coronary physiology testing:**

These patients may have disorders of vasomotion. When we do coronary angiograms, we look at the epicardial coronary arteries. However, 90% of the coronary circulation is supplied by microvascular circulation. We can do "functional" coronary angiography/coronary physiology tests in the cardiac catheter theatre to identify patients with INOCA and look at the microvascular circulation. In some institutions, such as Macquarie University Hospital we use Abbott's PressureWire™ X Guidewire and Coroventis CoroFlow Cardiovascular System.

We exclude obstructive coronary artery stenosis with a fractional flow reserve wire. Then we can study the microvascular system and diagnose microvascular dysfunction; and perform vasoreactivity testing (using acetylcholine), for epicardial or microvascular vasospasm. Thus, we can diagnose INOCA and identify whether it is a coronary macrovascular or microvascular disorder - see table below.

Functional coronary angiography helps to define the underlying pathophysiologic mechanism and the INOCA endotype:[14]

- **Microvascular angina:** symptoms of myocardial ischaemia; absence of obstructive coronary artery disease (<50% stenosis or FFR>0.80); objective evidence of myocardial ischaemia, evidence of impaired coronary microvascular function (on coronary physiology testing).
- **Vasospastic angina:** >90% epicardial vasoconstriction (during vasoreactive testing), reproduction of usual anginal symptoms, and ischaemic ECG changes.
- **Mixed (microvascular and vasospastic):** overlap condition meeting criteria for both microvascular angina and vasospastic angina.
- **Non-cardiac symptoms:** unobstructed coronary arteries with normal coronary function test results.

Diagnosing microvascular dysfunction and/or vasospastic angina can help guide our management, which is associated with less angina and better quality of life.

Table 1 | Clinical attributes of INOCA coronary vasomotor disorders.[13]

	Diagnostic investigation	Putative mechanism	Clinical presentation	Nitrate response
Coronary macrovascular disorders				
Vasospastic angina	Spontaneous or inducible coronary artery spasm	Coronary artery spasm	Rest or unstable angina; smoking is a risk factor	Prompt
Coronary microvascular disorders				
Cardiac syndrome X	Positive stress ECG	Impaired microvascular vasodilation	Stable exertional angina; high prevalence in female patients	Limited
Microvascular angina	Impaired coronary flow reserve	Impaired microvascular vasodilation	Often rest angina; often female patients	Limited
Coronary slow flow phenomenon	Delayed vessel opacification on angiography	Increased microvascular resistance	Unstable angina; smoking is a risk factor	Variable
Microvascular spasm	Inducible pain and ischaemic ECG changes with acetylcholine, but no coronary artery spasm	Microvascular spasm	Unstable angina; often female patients	Variable

Management

Depends on the underlying pathophysiologic mechanism/the clinical "endotype":

Guideline-recommended treatments based on clinical endotype:[15]

Diagnosis	Treatment	
Microvascular angina	Beta-blockers (nebivolol 2.5-10mg od); calcium channel blockers (amlodipine 10mg od) Nicorandil (10-20mg bd)	ACE inhibitors (ramipril 2.5-10mg od) A2RB • Stop nitrates
Vasospastic angina	Calcium channel blockers (verapamil 20mg od/ diltiazem 120-360mg od) Nitrates (ISMN 20-120mg od)	Nicorandil (10-20mg bd)
Mixed microvascular and vasospastic angina	Calcium channel blockers (amlodipine, verapamil, diltiazem) Nicorandil	ACE inhibitors
Non-Cardiac symptoms	Discontinue antianginal medication, and consider continuing cardiovascular risk reduction medication (eg. Statin) if coronary artery disease present	
Cardiovascular risk reduction	Statins Antihypertensives	Lifestyle: smoking cessation, exercise, cardiac rehabilitation, mediterranean diet, cognitive behaviour therapy, weight reduction if overweight.

Getting back to the initial case – we performed a coronary physiology study, and the diagnosis of coronary microvascular dysfunction/microvascular angina was made. We changed her over to a beta-blocker (as long-acting nitrates are not of benefit) to help with her symptoms and added a statin (event prevention).

References: 1. Bairey Merz CN, Pepine CJ, Walsh MN, Fleg JL. Ischemia and No Obstructive Coronary Artery Disease (INOCA): Developing evidence-based therapies and research agenda for the next decade. Circulation 2017;135:1075-92. doi:10.1161/CIRCULATIONAHA.116.024534. 2. Patel MR, Peterson ED, Dai D, et al. Low diagnostic yield of elective coronary angiography. N Engl J Med 2010;362:886-95. doi:10.1056/NEJMoa0907272. 3. Kunadian V, Chieffo A, Camici PG, et al. An EAPCI expert consensus document on ischaemia with non-obstructive coronary arteries in collaboration with European Society of Cardiology working group on coronary pathophysiology & microcirculation endorsed by Coronary Vasomotor Disorders International. Eur Heart J 2020;41:3504–20. 4. Ford TJ, Stanley B, Good R, et al. Stratified medical therapy using invasive coronary function testing in angina: the CorMicA trial. J Am Coll Cardiol 2018;72:2841–55. 5. Radico F, Zimarino M, Fulgenzi F, et al. Determinants of long-term clinical outcomes in patients with angina but without obstructive coronary artery disease: a systematic review and meta-analysis. Eur Heart J 2018;39:2135-46. doi:10.1093/eurheartj/ehy185. 6. Jespersen L, Abildstrom SZ, Hvelplund A, et al. Burden of hospital admission and repeat angiography in angina pectoris patients with and without coronary artery disease: a registry-based cohort study. PLoS One 2014;9:e93170. doi:10.1371/journal.pone.0093170. 7. Shimokawa H, Suda A, Takahashi J, et al. Clinical characteristics and prognosis of patients with microvascular angina: an international and prospective cohort study by the Coronary Vasomotor Disorders International Study (COVADIS) Group. Eur Heart J 2021;ehab282. doi:10.1093/eurheartj/ehab282. 8. Tavella R, Cutri N, Tucker G, et al. Natural history of patients with insignificant coronary artery disease. Eur Hear J Qual Care Clin Outcomes 2016;2:117–24. 9. Maddox TM, Stanislawski MA, Grunwald GK, et al. Nonobstructive coronary artery disease and risk of myocardial infarction. JAMA 2014;312:1754–63. 10. Jespersen L, Hvelplund A, Abildstrøm SZ, et al. Stable angina pectoris with no obstructive coronary artery disease is associated with increased risks of major adverse cardiovascular events. Eur Heart J 2012;33:734–44. 11. Di Fiore DP, Beltrame JF. Chest pain in patients with 'normal angiography': could it be cardiac? Int J Evid Based Healthc 2013;11:56-68. doi:10.1111/1744-1609.12002. 12. Tavella R, Cutri N, Tucker G, Adams R, Spertus J, Beltrame JF. Natural history of patients with insignificant coronary artery disease. Eur Heart J Qual Care Clin Outcomes 2016;2:117-24. doi:10.1093/ehjqcco/qcv034. 13. Beltrame, Tavella, R., Jones, D., & Zeitz, C. (2021). Management of ischaemia with non-obstructive coronary arteries (INOCA). BMJ (Online), 375, e060602–e060602. https://doi.org/10.1136/bmj-2021-060602. 14. Daniel Tze Yee Ang, Colin Berry. Learn about What an Interventionalist Needs to Know About INOCA with ICR Journal. What an Interventionalist Needs to Know About INOCA. Citation: Interventional Cardiology 2021;16:e32. DOI: https://doi.org/10.15420/icr.2021.16. 15. Guideline-recommended Treatments Based on Clinical Endotype. 16. Daniel Tze Yee Ang, Colin Berry. Learn about What an Interventionalist Needs to Know About INOCA with ICR Journal. What an Interventionalist Needs to Know About INOCA. Citation: Interventional Cardiology 2021;16:e32. DOI: https://doi.org/10.15420/icr.2021.16

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

WPW* pattern - pathway to sudden death?

Dr Bill Petrellis

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

WPW Pattern and the risk of Sudden Cardiac Arrest: case study

What is an accessory pathway?

In the normal heart, the atria and the ventricles are electrically isolated. Conduction of electrical impulses from the atria to the ventricles occurs via the AV node and His-Purkinje system.

Accessory pathways are due to congenital failure of resorption of the myocardial syncytium at the annulus fibrosis of the AV valves during foetal development. This residual myocardial tissue typically conducts electrical impulses more quickly than the AV node, resulting in a short PR interval seen on the surface ECG. **Preexcitation pattern** occurs because of the direct connection between the atria and ventricles via the accessory pathway, thereby allowing electrical signals to "bypass" the AV node, leading to earlier than usual activation of the ventricle generating a "delta wave" manifest as a slurred upstroke of the QRS complex.

Roughly 70% of accessory pathways are capable of bidirectional conduction (antegrade and retrograde) between the atrium and ventricle. Around 20% of accessory pathways are only capable of *retrograde conduction* (from the ventricle to atrium) and are known as "concealed pathways" as they do not generate a delta wave. These pathways are capable of supporting "orthodromic re-entrant tachycardia" (AVRT circuit: antegrade limb = AV node, retrograde limb = concealed pathway).

Less commonly ~ 10% of accessory pathways are capable of conduction only in an antegrade direction. These connections produce a delta wave and may give rise to "antidromic re-entrant tachycardia" (AVRT circuit: antegrade limb = manifest pathway, retrograde limb = AV node).

*Wolff Parkinson White

WPW "pattern" vs "syndrome". What is the difference?

"WPW pattern" refers to the presence of preexcitation on the ECG of an asymptomatic individual. "WPW syndrome" refers to the presence of preexcitation in an individual with symptomatic arrhythmias involving the accessory pathway.

Both are infrequent and occur in < 1% of the general population. WPW pattern is between 10 and 100 times more common than WPW syndrome.

What are the different types of supraventricular arrhythmias which occur in WPW syndrome?

- AV reentrant tachycardia (AVRT) up to 80%
- Atrial fibrillation (AF) 15-30%
- Atrial flutter < 5% percent

What are the symptoms of patients who develop an arrhythmia?

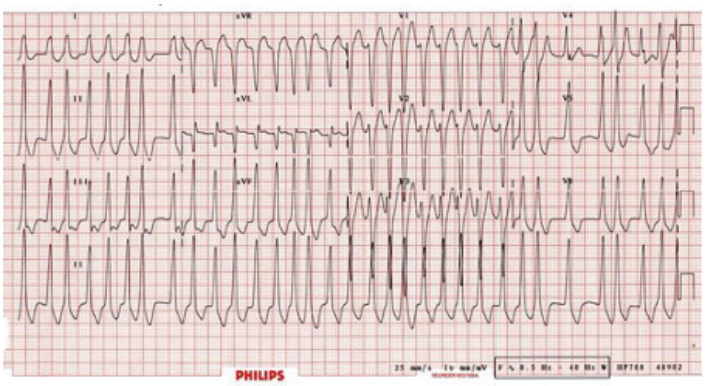
- Palpitations
- Light-headedness and/or dizziness
- Syncope or presyncope
- Chest pain
- Sudden cardiac arrest

Sudden cardiac arrest

Sudden death is due to ventricular fibrillation (VF) caused by rapid antegrade ventricular conduction by an accessory pathway during AF that persists and ultimately degenerates into VF. VF as the initial manifestation of WPW syndrome is rare ~1%.

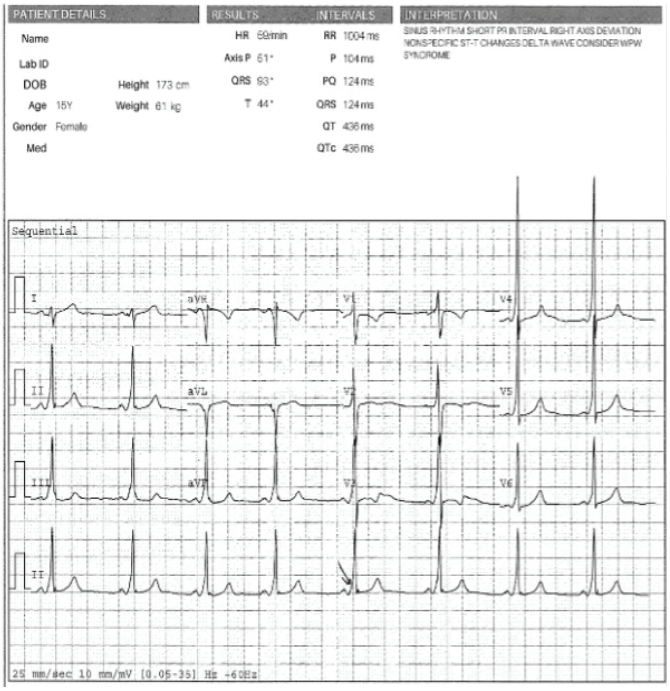
Patients with WPW pattern who are at increased risk for VF include those with a history of AVRT or AF, a very short antegrade refractory period (<250ms = 240bpm) of the accessory pathway noted during an EP study and short RR intervals (<250ms = 240bpm) during an induced or spontaneous episode of AF (see image).

WPW* pattern - pathway to sudden death?



In contrast, intermittent preexcitation (loss of delta wave) during atrial fibrillation suggests that the bypass tract has a long refractory period, making the development of VF unlikely.

AV node-blocking medications, (eg. verapamil, adenosine and digoxin) are associated with an increased risk of VF in patients with preexcitation and AF due to preferential conduction via the accessory pathway.



A subsequent holter showed preexcited sinus rhythm 65bpm (range 42-139bpm) with a single ventricular and only 3 atrial ectopic beats. Pre-excitation was observed at heart rates below 70bpm, but was absent at higher rates.

An interesting observation was made just before 10:00pm. The trace shows pre-excited sinus rhythm 56bpm followed by a late coupled atrial ectopic beat (the ectopic occurred 640ms after the preceding beat, i.e. at 93bpm) which was not preexcited.

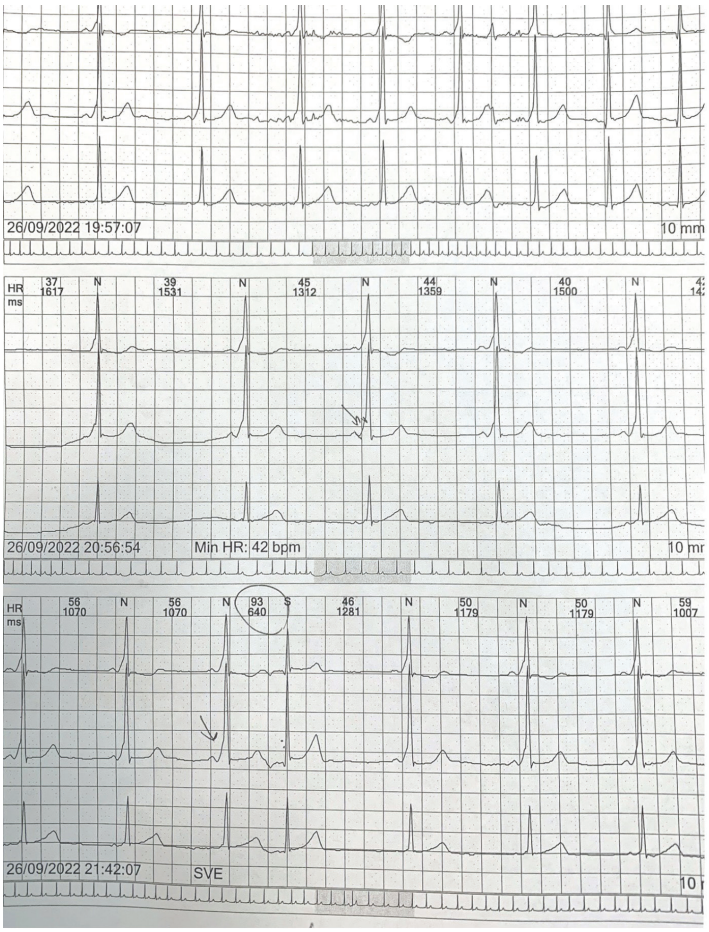
Case Study:

*Not the patient's real image

A 15yo endurance swimmer presented to her GP with a few days of fatigue. She had also experienced a brief 'fugue' while swimming, stating that she could not remember how she had reached the end of the pool during squads. There was no history of palpitations or symptoms to suggest SVT, presyncope or syncope.

Routine tests included a 12 lead ECG (see image) with referral for risk assessment of sudden cardiac arrest.

WPW* pattern - pathway to sudden death?



This finding confirms that the accessory pathway has a long refractory period (indicating weak conduction, that is, it blocks at 93bpm) and is therefore not likely to permit rapid conduction of atrial arrhythmias that can cause syncope or sudden death. This observation also likely explains why she has never experienced SVT. We can confidently conclude that this individual is not at risk of sudden cardiac death and does not need an EP study. Her accessory pathway is purely "cosmetic".

Discussion

The absence of palpitations, presyncope and syncope are significant negative features of the clinical history. However, this case highlights that a single and seemingly unremarkable holter trace, can provide all of the information needed to reassure an individual, her parents and the referring physician. Importantly, it provided an answer without the need to resort to an invasive test (EP study) in a young individual. It also allowed her to confidently pursue her aspirations in her chosen competitive sport.

I did not consider "fugue" a clinically relevant symptom. This young lady tested positive for COVID-19 within days of this symptom and has since made a full and uncomplicated recovery.

Save the date!

Cardiology in primary care: CPD activity

Wednesday, 15th February 2023

Raddison Blu Plaza Hotel Sydney

6pm start for canapés

Speakers:

Dr Fiona Foo

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

Dr Abhinav Luhach

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

Dr Gunjan Aggarwal

General adult cardiology and non-invasive cardiac imaging, particularly echocardiography and cardiac CT.

Click here for pre-registration

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

Blacktown

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

Tel: 02 9422 6050 | Fax: 02 9676 8900

Chatswood

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

Tel: 02 9422 6040 | Fax: 02 9411 1904

Parramatta

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

Tel: 02 9422 6060 | Fax: 02 9635 1247

Sydney City

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

Tel: 02 9422 6080 | Fax: 02 9422 6081

The new CBD location opens on the 23rd of Jan 2023

Sydney Cardiology offers a free after-hours consult service for GPs
Call (02) 9966 770 for specialist advice

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