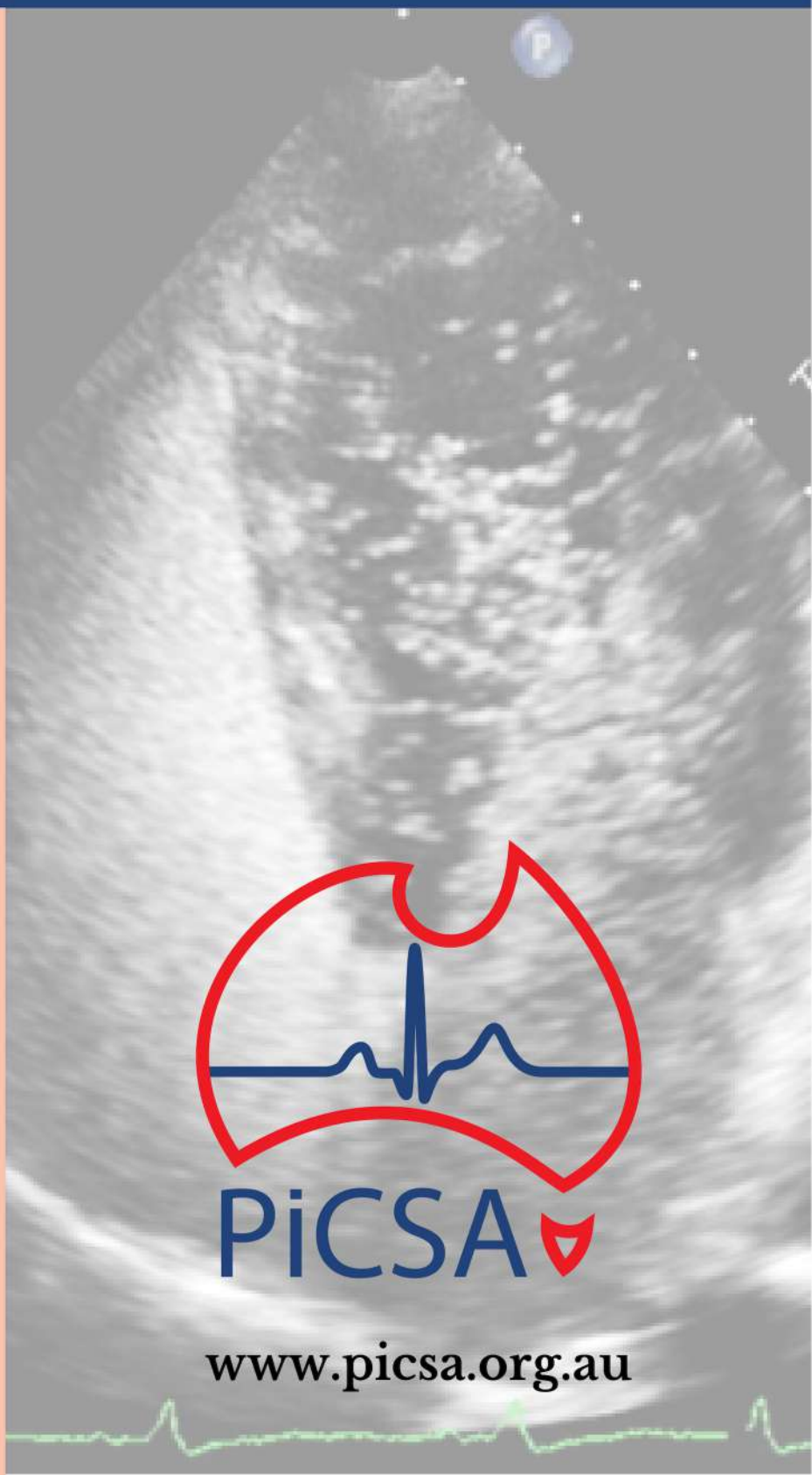
THE RHYTHM STRIP

The newsletter for Professionals in Cardiac Sciences Australia

Now welcoming readers from The Society of Cardiopulmonary Technology New Zealand

Summer 2024



Our Mission

To promote a commitment to excellence in standards of clinical best practice, provide the resources for ongoing continuing education of our members as well as promote and raise the profile of cardiac sciences both within & outside the profession.

Our Vision

To perform well as the peak professional resource and representative body for all Cardiac Physiologists in Australia through collaboration and cooperation with its members and other peak professional organisations.

Our Values

Adherence to the highest standards of professional and ethical behaviour.

Provision of safe, evidence based best practice to patents under our care.

Be accountable for our actions as health care practitioners.

PiCSA's Current Board Members

Miriam Norman (TAS)
Chair - Policy and Governance
PiCSA's representative on the CSANZ Allied Health, Science and Technology council

Jenny Fong (VIC)
Secretary

Leah Giles (VIC)
Business & Finance
PiCSA's representative to the ACCP

Tina Hetherington (QLD)
Membership and Website Services
Education Activities

Samantha Burgoyne (WA)
Professional Development

Luke Shanahan (QLD)
Relations and Strategy

PiCSA Professional Standards Committee Members

Bianca Coelho

PiCSA's Clinical Advisors

Malcolm Dennis
Tony Forshaw
Sharon Kay
Jason Riley

From the Chair

Miriam Norman (TAS)

I hope your year has started out strong. We are a little late getting out our "summer" newsletter, but we are excited to bring you important news on 4 fronts:

Position statement: We are thrilled to announce the newly revised position statement from PiCSA: "**Australian Guidelines for Entry and Practice in the Field of Cardiac Physiology**."

The Cardiac Physiology profession plays a critical role in diagnosing and treating heart conditions, yet standardised recommendations for qualification, certification, registration, accreditation, and remuneration have been lacking. Our position statement aims to address these gaps by defining governance structures, career pathways, and recommendations for stakeholders within our profession.
Building on the foundation of our earlier version, we have created a pivotal document to help shape the future of our profession. It contains recommendations relevant to Government agencies, employers, unions, and Cardiac Physiologists at all stages of their career. You can read the full document here (http://picsa.org.au/about/#core-documents). Our Summer newsletter contains a commentary on the associated costs of becoming a Cardiac Physiologist, which makes an interesting and valuable companion document to the position statement.

CIED Course: BIG news coming for Cardiac Device Physiologists

As stated in the 2022 Cardiac Society of Australia and New Zealand (CSANZ) Position Statement on the Follow-Up of Cardiovascular Implantable Electronic Devices, **Australia currently lacks an affordable, locally accessible, and nationally recognised postgraduate training program for Cardiac Device Physiologists**. There are many highly accomplished Cardiac Device Physiologists who have achieved excellency through self-directed learning, workplace-based training and international examinations, but there is a recognised need for a standardised, supported curriculum for the future workforce.

PiCSA is working towards a solution, but unfortunately, we can't make an announcement just yet.

Employer and student support of future training programs will be pivotal.
– if you want to express your interest or be involved in the discussion, please reach out to me at chair@picsa.org.au

NSW Echo: Cardiac Echo in the public sector wants better recognition and pay

The Health Services Union is actively addressing an ongoing pay disparity between sonographers with and without radiography qualifications, a move that benefits the broader Sonography profession, including Echo Physiology/Cardiac Sonography (where a tertiary foundation in physiology is common).

While this initiative may not immediately impact Cardiac Physiologists in other practice modalities, it signifies a positive step forward. PiCSA remains proactive in engaging with governmental bodies and unions to advocate for the interests of Cardiac Physiologists across all specialties. Our updated position statement, "Australian Guidelines for Entry and Practice in the Field of Cardiac Physiology," serves as a crucial framework for future policy discussions and potential industrial actions.

Representation within ASAR

Huge congratulations to both Bianca Coelho and Ashley Mattson on their appointment to the Australian Sonographer Accreditation Registry (ASAR) pool of course assessors! Now Cardiac Physiology will be better represented in determining the standards of sonographer education. You can read more about Bianca and Ashley in the current newsletter.

The PiCSA board has also been busy planning our education activities for 2024. Thank you to Luke Shanahan, Prof Kuljit Singh and the Gold Coast University Hospital for kicking off our first session for the year. If you want to volunteer to present, or to write for PiCSA in 2024, please reach out to secretary@picsa.org.au.

Thank you for your ongoing support and commitment to elevating the Cardiac Physiology profession. Together, we can drive positive change and ensure excellence in patient care.

Warm regards,
Miriam

Membership Report + Announcements

Tina Hetherington (QLD)

Professional Members 166
Associate 21
Affiliate 4
Student 43
Life 2

PiCSA Website

The new PiCSA website has now been live for 12 months.We have had fantastic feedback on our new site and our members are finding our Education Portal a great resource for continuing professional development.

The Education Portal continues to grow with new content added most months. Log in to your account at picsa.org.au and find the Education Portal under The Node menu.

PiCSA membership renewals

Many of our members annual PiCSA membership fees are now due. A large proportion of our members opted to auto-renew their PiCSA membership each year. If you chose this option, your membership payment will be automatically processed, and you will receive a 20% discount for opting to auto-renew!

If you need to update your payment details go to picsa.org.au and log-in to your account. Under The Node menu go to Membership Billing to update your payment details.

If you require assistance with renewing your PiCSA membership, please email membership@picsa.org.au

Many Great Benefits of PiCSA membership

- Access to Members Only articles, videos, education presentations and resources
- Exclusive discounts to industry affiliate partner events and courses
- Free advertising of employment opportunities on the PiCSA website
- Invitation to all in-person and online PiCSA educational meetings
- CPD point certificates for attendance at education meetings
- Quarterly newsletter update from the Board, and more!

PiCSA membership categories

Professional Membership ($110)*: This is for individuals who (1) hold a relevant undergraduate degree qualification and (2) have worked in the field of Cardiac Physiology for **over** two years. May be employed in a public, private, or industry setting.
* Non degree qualified physiologists with vast experience may also be considered for this category

Associate Member ($90): This membership category is for individuals who (1) hold a relevant undergraduate degree qualification and (2) have been working in the field of Cardiac Physiology for **less than** two years. May be employed in a public, private, or industry setting.

Student Membership (Free): This category is for individuals who are currently enrolled in an **undergraduate** degree program. The membership is valid for the duration of the degree program. Students must provide their enrolment details and expected graduation date to be eligible.

Industry Member ($110): This membership category is for Individuals with an entirely non-clinical role who work for a relevant supplier. This category does NOT apply to industry employed cardiac physiologists who assist with patient care (e.g., procedural support or interpretation of clinical data).

Affiliate Member ($90): Individuals with an interest in cardiac physiology who do not meet requirements for any of the other membership categories.

February Education Meeting

PiCSA's 2024 education calendar kicked off with an in-person event (streamed online) hosted by Luke Shanahan at the Gold Coast University Hospital. Thank you Luke Shanahan and Dr Kuljit Singh for your fantastic and engaging presentations. Your time, knowledge and skills are greatly appreciated. PiCSA hopes to deliver multiple education sessions in 2024.

If you or a colleague is interested in presenting, please email secretary@picsa.org.au.

Introducing the Latest Position Statement from PiCSA: Advancing the Cardiac Physiology Profession

We are thrilled to unveil the latest milestone in our ongoing efforts to advance the Cardiac Physiology profession: the newly revised position statement from PiCSA:

"Australian Guidelines for Entry and Practice in the Field of Cardiac Physiology"

This document represents a comprehensive guide to shaping the future of our profession - please head over to the PiCSA website to view this document (http://picsa.org.au/about/#core-documents).

Document Contributors and Revision History

The collective expertise of PiCSA members, alongside input from industry leaders and healthcare professionals, has shaped this document into a valuable resource. With its latest revision in March 2024, we have enhanced the layout, updated content for clarity, and included vital information on qualifications, career pathways and registration.

Background and Objectives

The Cardiac Physiology profession plays a critical role in diagnosing and treating heart conditions, yet standardised recommendations for qualification, certification, registration, accreditation, and remuneration have been lacking. This position statement aims to address these gaps by defining governance structures, career pathways, and recommendations for stakeholders within our profession.

Key Highlights

- **Professional Title and Modalities of Practice:** We advocate for a standardised national title, 'Cardiac Physiologist,' to streamline recognition across jurisdictions and emphasise the diverse modalities within our field.
- **Remuneration and Career Progression:** Recommendations are made to classify Cardiac Physiologists as health professional employees and establish equivalency in regulation and remuneration for advanced modalities of practice.
- **Education and Training:** The document outlines pre-entry qualifications, formal training pathways, and ongoing professional development requirements for Cardiac Physiologists.
- **Regulation and Accreditation:** We advocate for accreditation and registration through the ASAR and ACCP, with the goal of eventual federal mandate for registration across all modalities.
- **Continuing Professional Development (CPD):** CPD requirements are detailed, emphasising the importance of ongoing learning and certification maintenance.

Conclusion and Distribution

This position statement marks a significant step forward in advancing the Cardiac Physiology profession. It is a testament to the dedication and collaboration of PiCSA members and stakeholders. We urge widespread distribution of this document to ensure its impact reaches all relevant parties, from health workers' unions universities, students, employers, government health departments, and other relevant professional associations.

Thank you for your ongoing support and commitment to elevating the Cardiac Physiology profession. Together, we can drive positive change and ensure excellence in patient care.

New Cardiac Course Assessors for ASAR

We are thrilled to share some exciting news within our community! Two very talented and dedicated Cardiac Physiologists have been appointed to the pool of course assessors at the Australian Sonographer Accreditation Registry (ASAR).

Bianca Coelho is PiCSA's Professional Standards Committee chair and has been a regular contributor of written articles and live presentations. Bianca specialises in the modalities of echo physiology/cardiac sonography and cardiac devices.

Bianca's appointment to ASAR is a testament to her dedication and expertise in the field of Cardiac Physiology. With over six years of experience as the lead Cardiac Physiologist at Macquarie University Hospital, Bianca has consistently demonstrated a commitment to delivering high-quality cardiac services, including overseeing and assessing many students during clinical placement. Her contributions extend beyond clinical practice, as she has also made significant strides in research, education, and advocacy.

Ashley Mattson is a Cardiac Physiologist (specialty in cardiac sonography) and educator from Vancouver Island (Canada) living in Hobart, Tasmania (Australia). She has previously developed curriculum for the British Columbia Institute of Technology, has run a bedside echocardiography course for the College of Intensive Care Medicine of Australian and New Zealand, and has worked extensively with cardiology registrars and student sonographers.

Ashley has also recently designed and is now delivering New Zealand's first ever Postgraduate Diploma in Health Science (Cardiac Ultrasound) through the University of Auckland. This year she welcomed the first cohort of 16 students from across Aotearoa on their journey to becoming the first fully trained NZ cardiac sonographers. Ashley's experience in course creation makes her perfect for the ASAR course assessor role.

Ashley states: "It is nice to have someone who can represent ultrasound from a cardiac perspective. I feel like we are often forgotten since we are not as numerous.
After working with University and going through the accreditation process myself, I felt I had a good understanding of how accreditation works, and that perhaps I had something to contribute."

Bianca and Ashley's appointment as course assessors for the ASAR signifies not only their personal achievements but also a significant milestone for our profession. In this capacity, they emerge as the next generation of guardians, shaping standards for sonographer tertiary education and ensuring that Cardiac Physiology is appropriately represented.

PiCSA extends our warmest congratulations to Bianca and Ashley.

The Cost to Become (and Remain) a Cardiac Physiologist – An opinion piece

By Sam Burgoyne, WA

Cardiac physiologists play a crucial role in diagnosing and treating heart conditions. We undergo rigorous education and training, coupled with ongoing registration requirements, to ensure that we can provide high-quality care to patients. Many of us that have been in the industry for a while have probably been approached by people interested in joining our profession. It can be difficult to direct them, especially if you qualified a while ago and are not abreast of the current pathways for those wanting to become a cardiac physiologist. There are various courses and qualifications available within Australia, however, this article aims to offer a brief overview of the financial costs associated with entering the profession and maintaining various regurgitations and ongoing professional development.

Education - Graduate Studies

The typical starting point to becoming a cardiac physiologist in Australia involves completing a bachelor's degree in science. This cost varies depending on the university, whether you are a local or international student, and whether the course is undertaken as full-time versus part-time. As an approximate, this will cost $6,000 to $11,000 per year for an Australian citizen, and $20,000 to $45,000 per year for an international student. For an Australian citizen, you'd be looking at about $30,000 for a science degree, and as an international student, you might be looking at about $100,000. Other costs incurred as a student could include textbooks, accommodation, living expenses and other bills, which all add up to the total cost of obtaining a degree. Scholarships and financial aid are avenues that can help ease these financial obligations.

Education - Postgraduate Studies

The next step would then likely be a post-graduate qualification, such as a Master's degree or Diploma. Again, this cost varies depending on the university, international versus citizen, full-time versus part-time. For echocardiography related post-graduate courses, this ranges from approximately $8,000 to $40,000 a year for Australian citizens. For electrocardiography and electrophysiology related post-graduate courses, e.g. cardiac device training (IBHRE etc) & EP course (CEPIA), this ranges from approximately $2,500 to $10,000 per year. For all the post graduate diplomas in cardiac ultrasound, it is a mandatory requirement that you obtain at least 20 hrs of clinical placement prior to being accepted into the course. Depending on what the requirements are, you may need to have secured a practical placement. This practical placement is usually unpaid, and full-time, making it difficult to undertake paid work at the same time. At the time of publishing this article, government assistance or financial aid is not usually available for students during these practicum blocks.

Professional Registration

Post graduation, if you are practicing as an echo physiologist/cardiac sonographer, you need to obtain compulsory registration with the Australian Sonographers Accreditation Registry (ASAR) at the cost of $110 per year. If you are practicing in other specialties, such as electrophysiology, it is highly recommended (although not compulsory at the moment), to obtain registration with the Australian Council for Clinical Physiologists (ACCP). This usually costs $150 per year. Some workplaces will cover this cost for employees.

Ongoing Expenses - Continuing Education

Cardiac physiologists are required to participate in continuing education activities to maintain their certification and stay up to date of advancements in the field. These activities may include attending conferences, workshops, or completing online courses, with costs varying depending on the provider and format.

Ongoing Expenses - Professional Memberships

There are a multitude of professional organizations in our industry. A lot of these are listed on the PiCSA website. While not mandatory, they serve as a vital support system to represent the requirements of cardiac physiologists across Australia, such as CPD offerings like webinars, networking events and conferences. Some organisations provide professional indemnity insurance for additional payment, although many workplaces will provide this. Membership fees typically range $100 to $800 per year. CPD offerings range from $20 - $80, with some discounts for members. ASAR require 60 points over 3 years, and ACCP require 20 points per year, so you're likely looking at approximately 20 hours of CPD needed every year.

Financial costs are only one aspect to consider when entering the profession, albeit important. Becoming and remaining a cardiac physiologist is rewarding and fulfilling, making it a worthwhile profession that directly impacts the cardiovascular health of our community. There are multiple courses and pathways available within Australia for our industry, with fantastic CPD options including face to face conferences and online webinars. While some cost more than others, it's worth doing your research, considering what you hope to achieve for yourself in your chosen profession.

**While the information presented in this article is gathered primarily from our industry network and is current to the best of our understanding, readers should exercise judgment and consider additional sources where necessary*

CEPIA EGM series #7

Seek and You Shall Find

By Harley Cross and Jason Riley, CEPIA

The following is from a redo SVT procedure. For the purpose of this article some of the EGMs will be from the initial procedure, while others will be from the redo procedure to help 'tell the story'.

The patient was initially brought to the EP lab for investigation of increasing palpitations, now occurring 2-3 times a week.

Baseline retrograde study revealed eccentric atrial activation with CS 5-6 earliest and only minimal decremental conduction observed prior to VA block. The CS catheter was correctly placed with CS 9-10 at the ostium. During antegrade pacing a tachycardia of unknown mechanism was easily induced using PES x 1 and burst pacing without the use of Isoprenaline. An example of the initiation is seen in Figure 1.

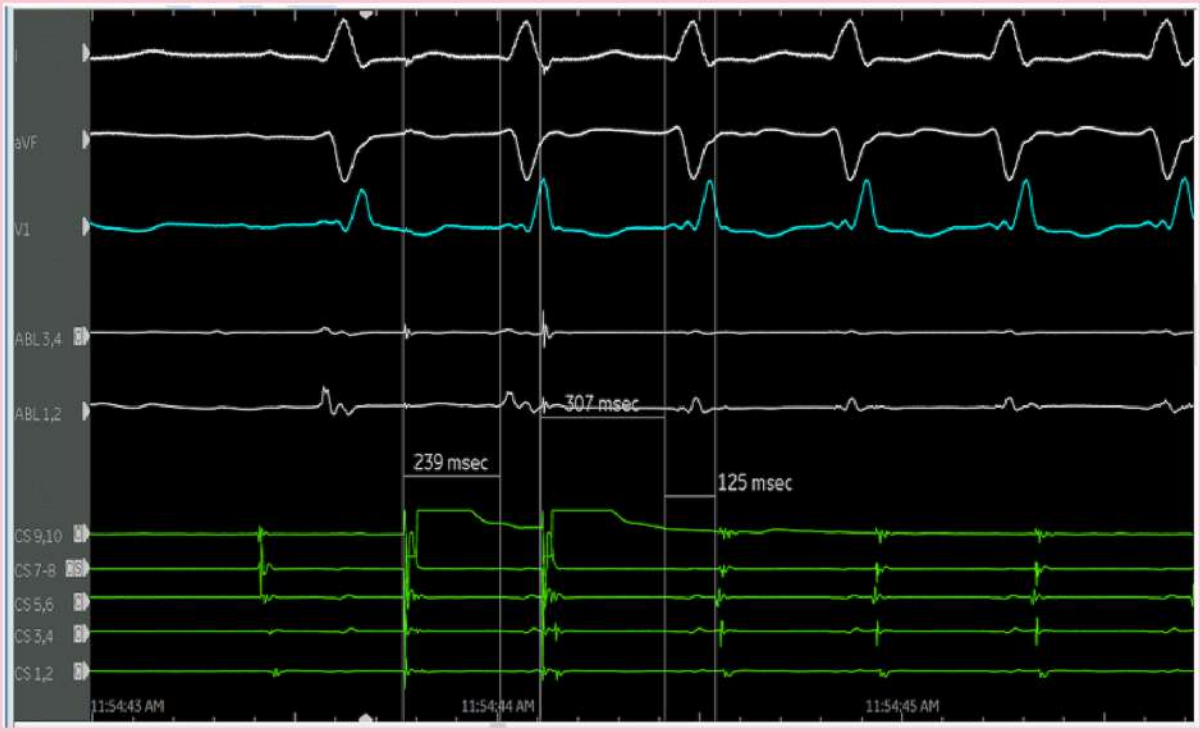


Figure 1: Induction of 1:1 SVT using a short burst of CS pacing. Eccentric atrial activation & VA 125ms. EGM was obtained from the redo procedure.

The tachycardia was sustained and demonstrated 1:1 conduction with RBBB aberrancy. See figure 2.

Based on these findings and the EGM what are the possible mechanisms of tachycardia?

CEPIA CARDIAC ELECTROPHYSIOLOGY INSTITUTE OF AUSTRALASIA

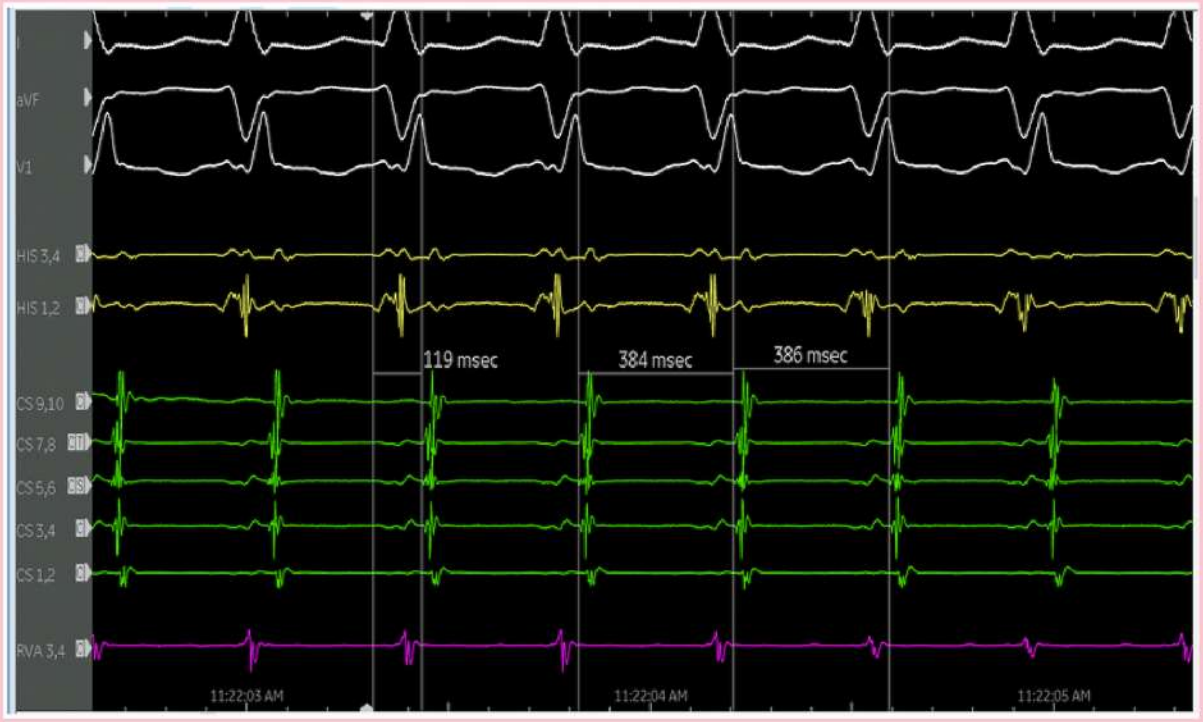


Figure 2: 1:1 SVT, RBBB aberrancy, VA 119ms, earliest A at CS 5-6. EGM is taken from initial procedure.

Based on the conduction intervals and appearance of this tachycardia the possible mechanisms include atypical AVNRT (slow/slow), orthodromic AVRT and atrial tachycardia with a long PR interval. Diagnostic pacing manoeuvres are required to confirm the exact mechanism.

Which manoeuvre should you perform? In our experience VODP is a 'go to'. With this one manoeuvre numerous responses are elicited to diagnose the mechanism. Attempted entrainment from the RV apex was performed and the response seen in Figure 3.

The response to RVA entrainment shows acceleration of the atria to the pacing rate of 360ms with the same atrial activation. This suggests a re-entrant mechanism. The PPI – TCL was 139ms with a V-A-V response. These three findings are highly suggestive that the mechanism is an atypical form (slow/slow) of AVNRT.

The target ablation site regardless of the AVNRT subtype is the AV node slow pathway. The slow pathway is commonly located in the posteroseptal region of the Triangle of Koch (TOK) in the right atrium. Slow pathways located in this region are referred to as the 'right inferior extension'.

Effective ablation of the slow pathway in sinus rhythm resulted in good junctional stimulation.However, the same tachycardia was still inducible post ablation. The procedure was deemed unsuccessful, with the plan to bring the patient back for an attempted redo slow pathway ablation in two months.

Figure 3 RVA entrainment demonstrating a long PPI -TCL >115ms and VAV response. Fittings most consistent with AVNRT

What could be done differently the next time?

At this stage it's important to remember that not all slow pathways sites are found in the classic 'right inferior extension region' in the right atria and ablation here can be unsuccessful regardless of the lesion quality.

In a small percentage of AVNRT patients, the slow pathway may exit in the roof of the coronary sinus (~2-4cm from the ostium). When this happens, the slow pathway is known as a 'left inferior extension'. In even rarer cases the slow pathway may be located at the inferior basal left atria near the mitral annulus, these sites are referred to as the 'inferolateral left atria slow pathway'.

Given the appearance of the coronary sinus electrograms during tachycardia it seems safe to assume that the slow pathway may be exiting at either the left inferior extension or the inferolateral left atrial site.

Mapping of the atypical slow pathway sites can be performed in a similar way to mapping a concealed accessory pathway. That is, they can be mapped with RVA pacing or during tachycardia with the aim to identify and target the earliest atrial signal. If mapping retrograde you must ensure that retrograde conduction is occurring via the slow pathway and not the fast pathway.

In this case mapping was performed during AVNRT. Figure 4 shows two mapping sites, the first site (left image) was on the roof of the coronary sinus with the earliest signals being 18ms ahead of CS 5-6 (our earliest atrial electrogram pair). The second site (right image) was at the inferolateral MA region (~5-4 o'clock) and was 24ms ahead of CS 5-6.

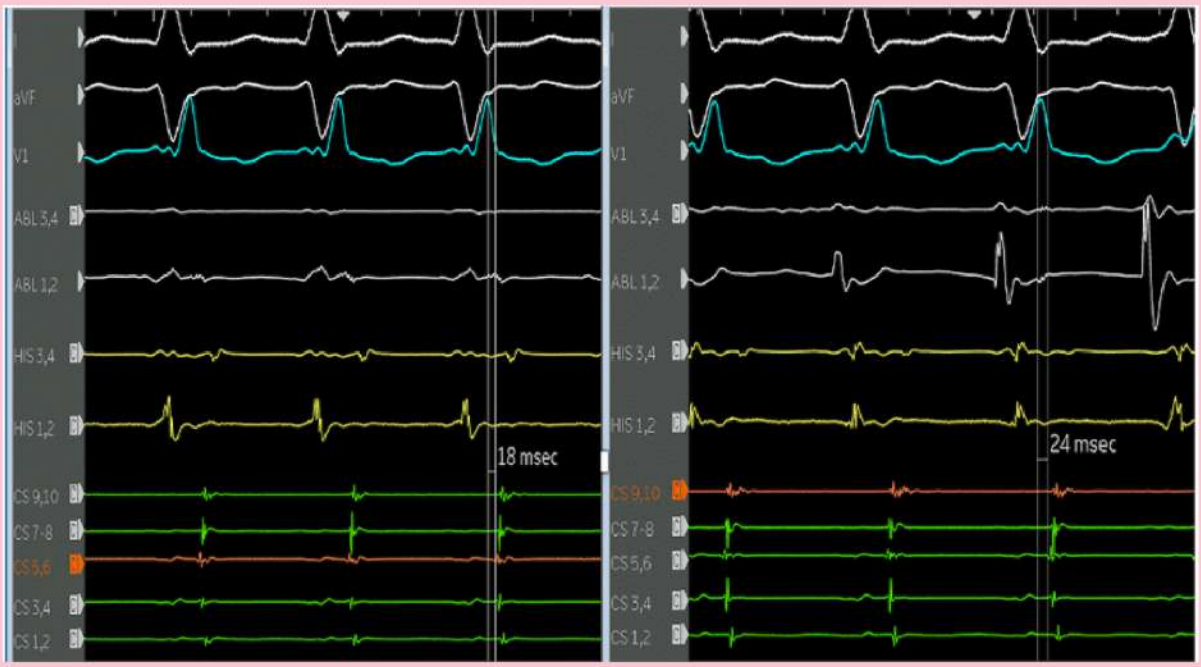


Figure 4: Left image shows the earliest sites on the roof of the CS. Right image shows the earliest site at the inferolateral MA region.

Based on these maps our patient's slow pathway is likely located at the rare inferolateral left atrial site. Ablation was performed at this site during tachycardia and resulted in termination. After several consolidation ablations no further AVNRT was inducible.

This case highlights the importance of remembering that not all slow pathways can be ablated from the right inferior extension in the TOK. In rare cases the slow pathway may exit from within the coronary sinus, in the left atrium and in very rare cases the anterior superior right atrial septum near the fast pathway.

CEPIA CARDIAC ELECTROPHYSIOLOGY INSTITUTE OF AUSTRALASIA

PICSA

Wolff-Parkinson-White: an incidental finding

By Edward Westcott

Introduction:

Wolff-Parkinson-White (WPW) is a congenital abnormality of the heart's electrical conduction system where there is a presence of an abnormal electrical pathway between the atria and ventricles. There are several anatomical forms of the abnormal atrio-ventricular conduction, which bypass the usual pathway of electrical activity from the sinoatrial (SA) node to the atrioventricular (AV) node.

This abnormality was first described in 1930 by Louis Wolff, John Parkinson, and Paul Dudley White and was referred to as an accessory pathway (Sapra, et al., 2020). The accessory pathway can have a singular anatomical pathway or multiple pathways, in which one of the pathways is predominant, and the other pathway may lay dormant (also known as latent).

The aetiology of WPW pattern arises from the fusion of ventricular pre-excitation and is thought to arise from chamber myocardium during improper early atrial and ventricular folding in the cardiac embryogenesis stage. WPW can be described as either a pattern, or a syndrome. WPW pattern is the presence of electrocardiographic (ECG) features without the presence of symptoms, whereas WPW syndrome is the presence of the ECG features and the presence of, or history of symptoms.These symptoms include palpitations due to supraventricular tachyarrhythmias; dizziness and presyncope due to the rate of the tachyarrhythmias. The inappropriate conduction poses a large risk due to inducible tachyarrhythmias, which can lead to sudden cardiac death (Wilson, 2023).

The prevalence of the disease is 1 to 3 persons per 1000 (0.1%-0.3%). Most patients with WPW do not show symptoms until an arrhythmia/tachyarrhythmia is induced, which changes the presentation from pattern to syndrome. If the tachyarrhythmia is not treated, it can lead to sudden cardiac death (Han, et al., 2022).

Case Presentation

A 55-year-old male was referred to a tertiary teaching hospital in Queensland for a sleep study in 2021 for assessment of Obstructive Sleep Apnoea (OSA). The patient was the same weight and height now that they were in 2021 at a height of 184cm and a weight of 86kg (BMI 25.4). The patient reported no previous cardiac history. During the overnight sleep study a possible arrhythmia was recorded on the 3-lead electrocardiogram (ECG).

To further investigate the arrhythmia, a 12-lead electrocardiogram (ECG) was performed. The ECG confirmed the presence of a Wolff-Parkinson-White pattern. The patient was then referred for an Exercise Stress Test (EST) which was negative, proving no inducible arrhythmia. The patient was subsequently monitored, and the patient reported brief episodes of fluttering heart rate and palpitations, which occurred rarely and were sudden in onset and offset. In 2023, the patient was referred to the cardiology department for an investigation and possible ablation of the accessory pathway associated with WPW pattern.

This education article was kindly provided by:

Griffith UNIVERSITY

ECG Analysis and Interpretation

The patient was referred to the Electrophysiology (EP) department for mapping of and potential ablation of the accessory pathway associated with his WPW syndrome. As part of the routine preparation for the EP investigation, a 12-lead ECG was performed to identify specific features of the WPW pattern. Characteristic diagnostic criteria need to be met on the ECG to confirm the WPW pattern and the possible location of the accessory pathway. Figure 1 shows the patient's pre-EP study 12-lead ECG.

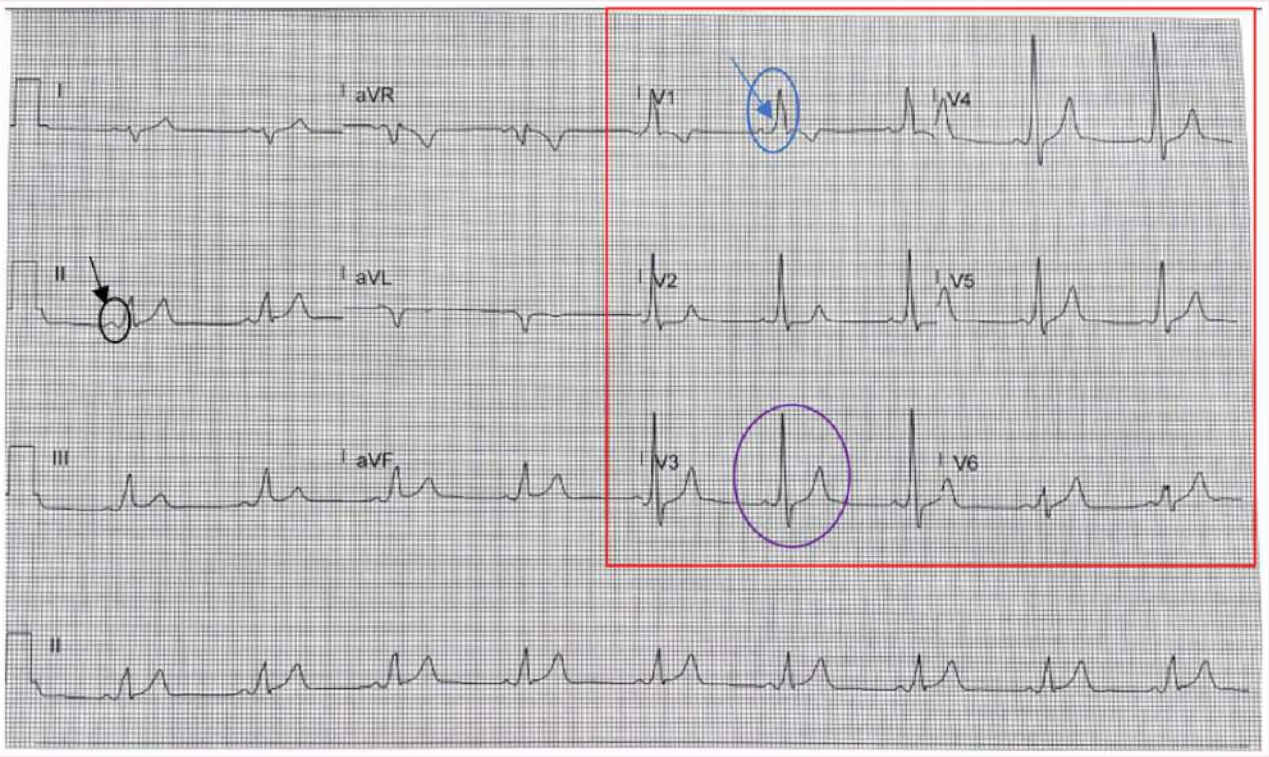


Figure 1 This ECG shows the critical features of WPW. Short PR interval is shown in lead II (circled and arrowed in black). A present delta-wave in each QRS complex, best shown in V1 (circled and arrowed in blue). The delta wave (or depolarisation of the pre-excitation pathway) is shown as a 'slur' in the R-wave of the QRS complex circle. This pre-excitation leads to the QRS broadening (>120msec) and is represented by the purple circle. This WPW ventricular pre-excitation demonstrates a left-sided pathway due to positive concordance in all pre-cordial leads (V1-V6).

Normal ECG reference ranges	Patients' ECG features
Rhythm regular	Rhythm regular
Rate 60-100bpm	Rate 55bpm (Bradycardic)
PR interval 120-200 msec	PR = 110msec
Q-wave absent or septal	Delta wave present and positive pre-cordially
QRS segment 80-120 msec	QRS = 130msec
R-wave progresses normally from V1-V6 (negative V1 – positive V6)	R-wave positive in all precordial leads with R/S > 1 in V1. Left sided pre-excitation

Table 1: Analysis of 12 lead ECG prior to AP ablation

This education article was kindly provided by:

Griffith UNIVERSITY

Discussion

Wolff-Parkinson-White is a relatively rare disease affecting around 0.1-0.3% of the world's population. However, because most patients are asymptomatic and may dismiss cardiac related symptoms such as racing heart and palpitations as they only occur rarely, the prevalence can be underestimated. The patient in this instance was asymptomatic and was referred to a tertiary teaching hospital for investigation of non-cardiac related medical symptoms. Wolff-Parkinson-White is a congenital accessory pathway in the hearts' electrical conduction system, by which the accessory pathway bypasses the hearts normal conduction pathway (Sapra, et al., 2020). A schematic representation of the accessory pathway is demonstrated in Figure 2.

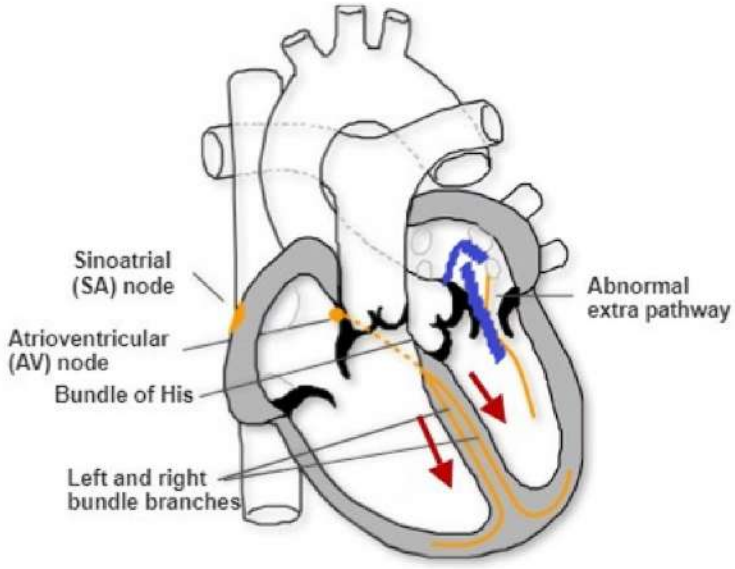


Figure 2 This schematic diagram demonstrates the normal direction of cardiac electrical activity from the SA node to AV node and down the Bundle branches as shown by the down pointing red arrows. The accessory pathway present in WPW, results in an abnormal direction of electrical activity (shown by the purple arrow) in which the electrical activity passes in a retrograde direction from the ventricles to the atria. Image adapted from the LifeART collection images 1989-2001 by Lippincott Williams & Wilkins, Baltimore, MD

This congenital malformation of the accessory pathway predisposes the individual to life-threatening arrythmias and possible sudden cardiac death due to changing refractory periods and re-entrant circuits. The anatomical positions of the pathways differ greatly for each individual. They can present as a signal anatomical pathway connecting the atria and ventricles or it can present as multiple pathways connecting the ventricles; either close to each other or completely independent of one another. If multiple pathways are present, one pathway will be dominant, and the other/s will be latent.

The WPW syndrome is commonly associated with a mutation in the PRKAG2 gene, which is the gamma-2 subunit of the AMP-activated protein kinase (AMPK). The conduction system abnormalities seen in this PRKAG2 gene originate from glycogen-filled cardiomyocytes causing the development of pre-excitation muscular bypass pathways.

The pathways are independent of the normal conduction system (Dyck & Lopaschuk, 2006). The accessory pathways attach the higher atrial chambers to the lower ventricular chambers, mainly attaching to the ventricular septum, which bypasses the normal conduction pathway which is through the atrioventricular (AV) node. This means that there is no slowing of the signal from the sinoatrial (SA) node in the atrium to the AV node and the ventricles. On an ECG, the slowing of the signal in sinus rhythm through the AV node is the represented by the PR interval (Znojkiewicz & Spector, 2013).

This education article was kindly provided by:

Griffith UNIVERSITY

PiCSA

In WPW patients, such as in the case of this patient, the PR is shortened (<120msec), as the AV has been bypassed and the ventricles have started to depolarise prior to normal conduction. The bypass accessory pathway is typically the origin for the re-entrant supraventricular tachyarrhythmias that can be induced and are generally associated with WPW syndrome. The accessory pathway is not composed of the fast firing, pacemaker Purkinje fibres, but comprised of electrically active muscular cardiomyocytes.

The bypass tracts conduct the action potentials directly from the atrial chambers to the ventricular septum, and this starts to depolarise the muscular septum prior to the normal conduction pathway. As a result, the depolarisation is more spread out over time, as muscle cells must depolarise from cell-to-cell, generating a broader than normal QRS complex. Since this is abnormal depolarisation of the septum and it is occurring before the normal conduction, it changes the axis of the Q-wave, and a delta wave is present. This delta wave is shown as a 'slur' to the start of the R-wave, which is evident in this patient's ECG (Znojkiewicz & Spector, 2013). The reason why the slurring R-wave does not continue is because the normal conduction signals fuse with the preexcitation so there is sharp point to the R-wave and regular S-wave. Due to the abnormal depolarisation of the ventricles, there will be abnormal repolarisation which is represented by a change in the morphology of the T-waves in some or all leads, in the case of this patient, it is just V1 where the T-wave are inverted. Figure 3 demonstrates the characteristic triad of ECG features associated with WPW pattern.

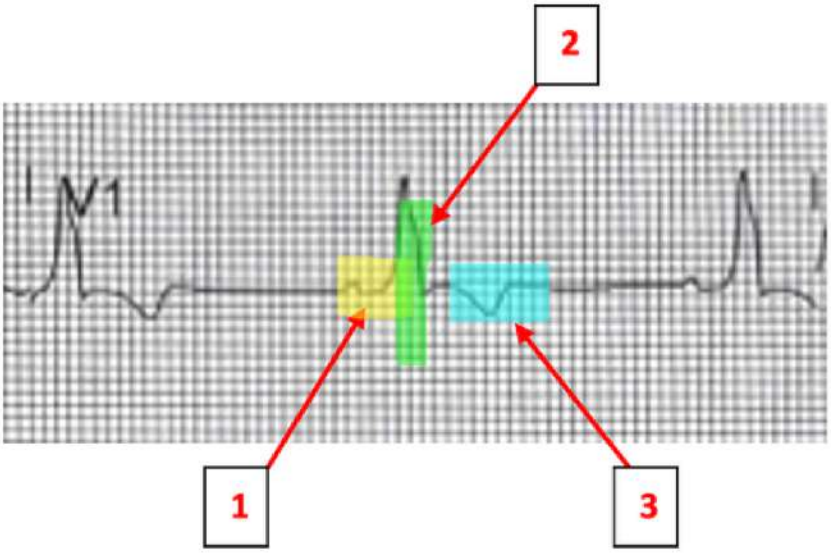


Figure 3. A zoomed in view of V1 from the 12-lead ECG shown in Figure 1. This image shows the characteristic triad of ECG features noted in WPW pattern: 1. Shortened PR interval of <120msec, 2. Wide QRS complex >120msec, 3. Slurred upstroke of the QRS complex (Delta wave). This particular ECG is consistent with a left sided bypass pathway because there is an inverted T wave following a positive delta wave.

Since there is a pre-excitation pathway present in the conduction system, and the normal conduction does not cease, the action potentials around the heart are in different periods of relative and absolute refraction. The refractory periods are the prime instigator of arrythmias and re-entrant circuits which induce supraventricular tachyarrhythmias (Hoyt & Snyder, 2013). Re-entrant circuits are established when separate pathways are in refractory periods and a retrograde signal, or another signal is transmitted during a period of refractory, and the signal arrives after the initial refractory period, allowing the timing of the signals to re-enter into the pathway and cause tachyarrhythmias (Hassett, Cho & Suarez, 2021).

The most common are tachyarrhythmias in WPW patients are paroxysmal supraventricular tachyarrhythmias (PSVT), such as atrial fibrillation, atrial flutter, and atrioventricular re-entry tachycardia (AVRT) (Boron & Boulpaep, 2017).

This education article was kindly provided by:

Griffith UNIVERSITY

PICSA

As shown in figure 4, re-entrant excitation starts from when an impulse is sent through while a unidirectional block prevents for a transmission of the signal. In normal conduction, the action potentials collide and cannot pass through each other due to the absolute refractory period of the pathway. In the abnormal situation, like in WPW, the signal travels through the conduction system and branches, the wave of excitation can travel in the reverse direction as one pathway of the system is in refractory, and doesn't have a signal passing through it (Hassett, Cho & Suarez, 2021). The unidirectional block allows for the retrograde transmission of the signal and the system will then behave as an independent (ectopic) pacemaker. This ectopic rate is much higher than the sinus rate, roughly 150bpm and greater. This pathophysiological re-entry is the basis for lots of the re-entrant tachycardias. In terms of AVRT, which is the most common in WPW, has two forms, Orthodromic AVRT and Antidromic AVRT (Boron & Boulpaep, 2017).

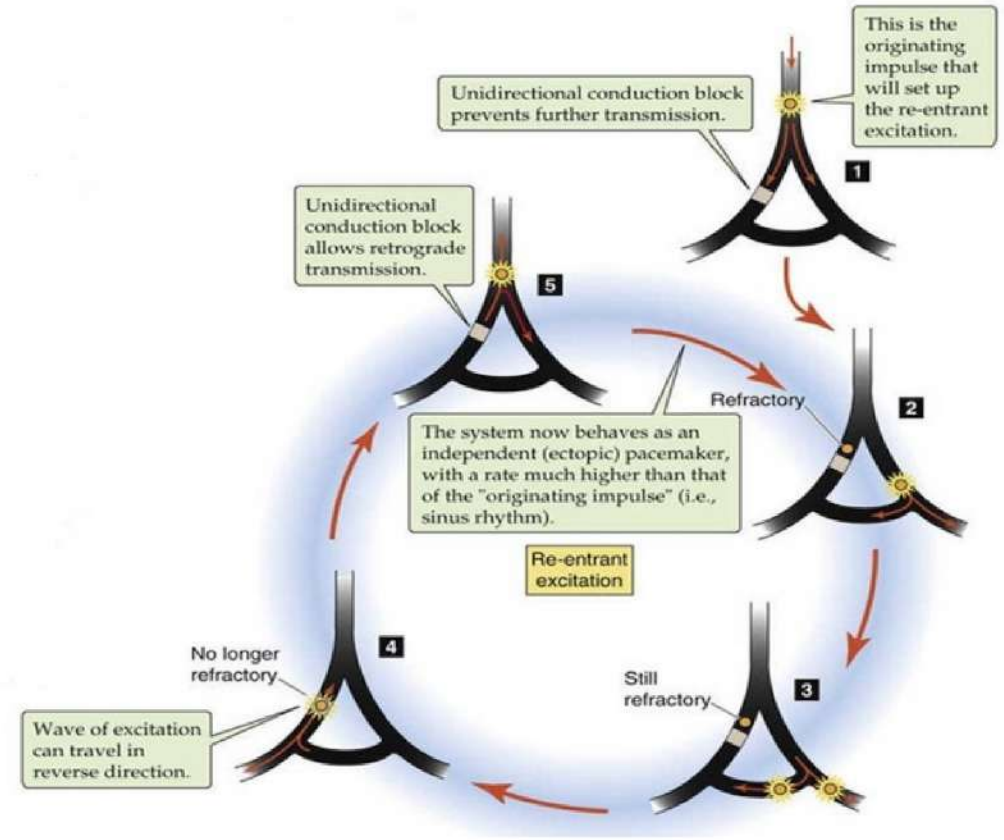


Figure 4: Demonstrates the re-entrant excitation pathway (image is Figure 21-15d, Boron, W. F., & Boulpaep, E. L. (Eds.). (2017). Medical physiology (Third (3). Elsevier

Orthodromic is the most common, accounting for 90-95% of AVRTs, and Antidromic AVRTs account for 5-10%. They both can originate due to a premature contraction of the atrium or ventricles. In orthodromic, if the atrial premature contraction initiated the AVRT, it would block in the accessory pathway and conduct antegrade through the normal conduction pathway, with the impulse travelling back to the atria through the accessory pathway. If the ventricular premature contraction initiates the AVRT, the normal conduction pathway will be blocked, and a retrograde signal will be sent back to atria via the accessory pathway, meaning Orthodromic AVRT is an anterograde conduction through the AV node and His-Purkinje system with a retrograde signal

This education article was kindly provided by:

Griffith UNIVERSITY

back through the accessory pathway (Dowd, 2014). The opposite is true for Antidromic AVRT, with the retrograde conduction through the AV and His-Purkinje system with an anterograde conduction through the accessory pathway, with the opposite initiations for atrial and ventricular premature contractions. These arrhythmias can resolve themselves via the same mechanisms that induced them, however, sustained periods of these arrythmias are not compatible with life due to greatly reduced cardiac output and decrease oxygen supply to cardiac cells and other major organs. This is why it is important to prevent the initiation of the arrhythmias, and this can be done by ablating the accessory pathways (Page et al., 2016).

Management and Treatment

The management and treatment for this patient was to perform an Electrophysiology (EP) mapping study and perform an ablation of the accessory pathway to prevent a supraventricular tachyarrhythmia and other life-threatening arrhythmias which would lead to sudden cardiac death. This is the most common form of treatment for WPW as if successful, it cures the abnormality. The EP mapping of the heart accurately maps and tests where the accessory pathway is located. This is an important process as ablation must be precise. Ablation burns the accessory pathway, killing the electrically active myocytes and preventing the conduction of a signal down this bypass pathway which can induce dangerous arrhythmias.

Conclusion

In conclusion, the patient was asymptomatic and the finding of Wolff-Parkinson-White was incidental finding when the patient presented for assessment of OSA. However, WPW pattern was detected, and the patient underwent further testing to diagnose the severity and risk associated with developing a dangerous arrhythmia and sudden cardiac death. This was important, as the patient subsequently become symptomatic, and thus WPW syndrome was established. The clinical decision by the treating team to ablate the accessory pathway and avoid the risk of any life-threatening arrhythmia being induced due to the bypass pathway was a crucial risk aversion strategy in this patient.

Acknowledgements
I would like to acknowledge Mrs Alison White, Senior Lecturer Griffith University for her support and guidance in the editing of this case study.

This education article was kindly provided by:

Griffith UNIVERSITY

References:

Boron, W. F. & Boulpaep, E. L., 2017. Cardiac Electrophysiology and the Electrocardiogram. In: W. F. Boron & E. L. Boulpaep, eds. Medical Physiology. Amsterdam: Elsevier, p. 504.

Dowd, F., 2014. Wolff Parkinson White Syndrome. In: F. Dowd, ed. Biomedical Sciences. Amsterdam: Elsevier, pp. 1-5.

Dyck, J.R.B. and Lopaschuk, G.D. (2006). AMPK alterations in cardiac physiology and pathology: enemy or ally? The Journal of Physiology, 574(1), pp.95–112.

Han, B, Wang, Y, Zhao, J, Lan, Q, Zhang, J, Meng, X, Jin, J, Bai, M, Zhang, Z, 2022. Association of T-box gene polymorphisms with the risk of Wolff–Parkinson–White syndrome in a Han Chinese population. Medicine, 101(32), pp. 1-9.

Hassett, C.E., Cho, S.-M. and Suarez, J.I. (2021). Tachyarrhythmias and neurologic complications. Handbook of Clinical Neurology, 177, pp.151–162.

Hoyt, W. and Snyder, C.S. (2013). The asymptomatic Wolff–Parkinson–White syndrome. Progress in Pediatric Cardiology, 35(1), pp.17–24.

Page, R.L., Joglar, J.A., Caldwell, M.A., Calkins, H., Conti, J.B., Deal, B.J., Estes, N.A.M., Field, M.E., Goldberger, Z.D., Hammill, S.C., Indik, J.H., Lindsay, B.D., Olshansky, B., Russo, A.M., Shen, W.-K., Tracy, C.M. and Al-Khatib, S.M. (2016). 2015 ACC/AHA/HRS Guideline for the Management of Adult Patients With Supraventricular Tachycardia: Executive Summary. Circulation, 133(14)
Roa, A. L., Salerno, J. C., Asif, I. M. & Drezner, J. A., 2014. Evaluation and Management of Wolff-Parkinson-White in Athletes. Sports Health, 6(4), pp. 326-332.

Sapra, A, Albers, J, Bhandari, P, Davis, D, Ranjit, E, 2020. Wolff-Parkinson-White Syndrome: A Master of Disguise. Cureus, 12(6), pp. 1-5.

Wilson, J. H., 2023. Arrhytmias During Pregnancy. In: J. H. Wilson, ed. Maternal Cardiac Care. Amsterdam: Elsevier, pp. 85-89.

Znojkiewicz, P. and Spector, P. (2013). Dysrhythmias and Tachyarrhythmias.

This education article was kindly provided by:

Griffith UNIVERSITY

PICSA

Bubble studies:

When and how to perform agitated saline enhanced echocardiography

By Miriam Norman (TAS)

Agitated saline enhanced echocardiograms or "bubble studies" are a form of contrast study that can increase the diagnostic sensitivity of a standard trans-thoracic echocardiogram (TTE). Agitated saline is inexpensive and can improve the visualisation of blood flow patterns within the heart and major vessels. According to the ASE guidelines, all echo labs should be prepared to perform a bubble study "on demand" when standard imaging is insufficient. It is important to know **when** to add it to your standard study, and **how** to perform it correctly(1).

When should we do a Bubble Study?

The most common indications for a bubble study are:

1. Suspected **intracardiac** shunts – e.g., patent foramen ovale (PFO) or atrial septal defect.
2. Suspected **extracardiac** shunts – e.g., pulmonary arteriovenous malformation or hepatopulmonary syndrome.
3. Suspected **persistent left superior vena cava** (PLSVC) – the most common thoracic venous anomaly.

Less common indications include:

1. Intensifying tricuspid regurgitation (TR) signal for RV systolic pressure evaluation
2. Improving views of the right heart borders (e.g., when performing pericardiocentesis, evaluating RV wall thickness, regional hypokinesis or intracardiac masses)
3. Screening for thrombi in the right heart, pulmonary trunk and arteries (contrast filling defects suggest thrombi may be present).

Even if the TTE referral does not expressly ask for a bubble study, or list one of the above indications, it is within our scope of practice as Echo Physiologists to flag scenarios when a bubble study might be beneficial, and to appropriately facilitate it, ideally as an extension of a current study or as part of a subsequent scan.

Consider a bubble study in the following patients:

1. Unexplained right heart dilatation - possible atrial septal defect (ASD) with no clear flow across septum on colour Doppler.
2. Unexplained hypoxia – possible ASD or PFO.
3. Dilated coronary sinus - possible PLSVC which can significantly impact cardiac surgery.
4. Inadequate TR Doppler signal - where assessment of pulmonary artery pressure is critical.
5. Cardiac Source of Emboli – suspected paradoxical embolism across a PFO. This is the most common scenario we encounter that might warrant a bubble study, typically in (a) stroke patients and (b) divers with a history of decompression illness.

What about cryptogenic ischaemic stroke/transient ischaemic attack (TIA)?

In cases where traditional stroke risk factors, such as hypertension, valvular abnormalities, or atrial fibrillation, are not apparent, the absence of a PFO during a standard TTE does not definitively rule out the possibility of paradoxical emboli: PFOs are present in 20 - 30% of the population, but often remain undetected during a standard TTE. To reveal these hidden defects, the use of agitated saline and provocative manoeuvres may be necessary.

PFOs can be innocent incidental findings rather than the direct cause of stroke. Current research suggests that in patients under the age of 55, a PFO is more likely to be the culprit, so bubble studies prove to be most valuable in this specific age group.

The bubble study should only be performed if detection of a shunt is likely to result in a change of management (e.g., PFO closure). The ideal approach is to develop a local protocol to decide when to offer a bubble study as an addition to the standard TTE. An example of a local decision tree is provided in Figure 1.

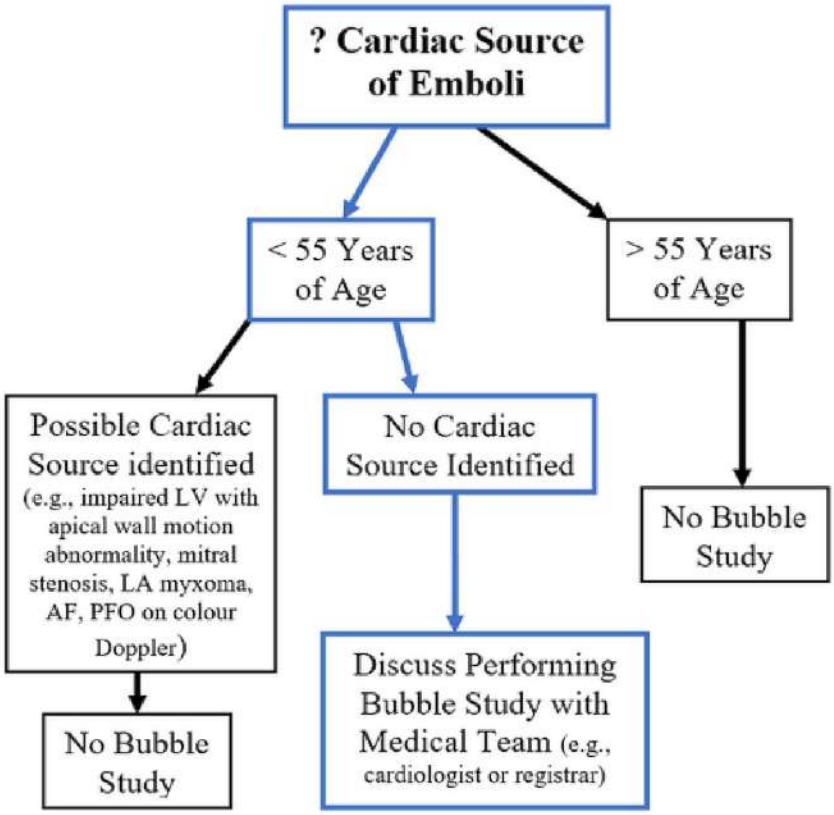


Figure 1: Example of a local decision tree re patient selection for bubble study following cryptogenic embolic stroke or TIA (Royal Hobart Hospital Echocardiography Department)

Decompression sickness/paradoxical gas embolism in divers

PFOs have garnered particular attention in the context of diving-related health concerns. Dissolved gases emerge from solution as bubbles inside the venous blood and other tissues during decompression and may pass through the PFO and into the arterial circulation(2). Studies have shown that divers with PFOs are 4x more likely to develop neurological decompression illness. Risks are greater if the PFO is large, and if the diver performs Valsalva-like manoeuvres after the dive. The peak time for bubble formation is 30 to 60 min post-dive, which unfortunately coincides with the time that divers are most likely to be climbing in and out of boats and straining to lift heavy equipment (2,3).

The current consensus is that PFO screening via TTE +/- bubble study does not need to be done routinely on all divers. Consideration should be given to testing for PFO only when there is a history of more than one episode of decompression sickness with cerebral, spinal, vestibulocochlear or cutaneous manifestations(2,3).

Contraindications

- **Known significant right to left shunt.** 2D colour Doppler examination should always be performed first. If you can already see it on colour Doppler, you don't need to do a bubble study to prove that a shunt is there.
- **Pregnancy:** In general, saline contrast should be avoided during pregnancy to minimise any possible embolic risk (although the theoretical risks are low).

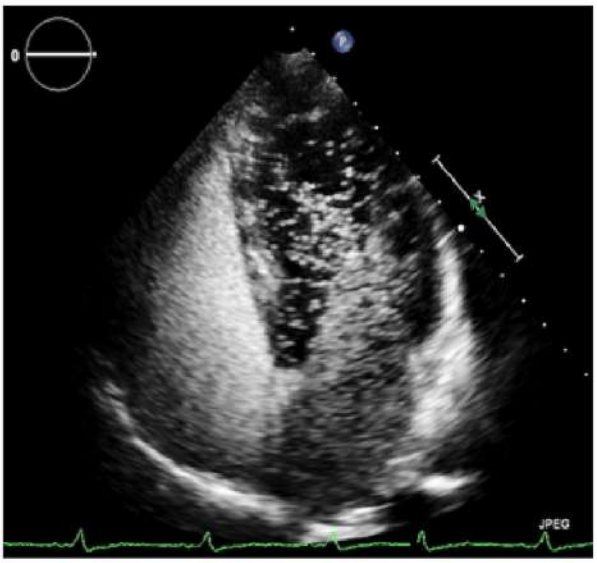


Figure 1: A positive bubble study. Image reference: https://smartcd.com.au/cardiac/agitated-saline-bubble-echocardiogram/

Possible Adverse effects:

Agitated saline bubble studies are considered a very safe addition to standard trans-thoracic echocardiography, but complications can occur:

- **From intravenous cannulation**:
  - A little discomfort from the area where the cannula was inserted is common.
  - Rare complications include cannulation failure, thrombosis, arterial puncture, and infection.
- **From injection of saline**:
  - Headache - uncommon
  - Stroke or transient ischaemic attach (TIA) has been reported but is very rare.
- **Blood exposure –** If blood has been added to the saline mixture, it is especially important for lockable syringes and stopcocks to be used, otherwise pressure from agitation or injection can cause disconnection of the apparatus, exposing people to a hazardous spray of the solution.

To minimise the number of bubbles crossing into the arterial circulation it is best to start gently with a conventional study. Provocative manoeuvres should only be performed if necessary.

Performing a Bubble Study

Obtain physician's order and patient consent: These are usually verbally obtained prior to the procedure.

Equipment:

- Sterile normal saline
- Two 10 mL lockable syringes
- 3-way lockable stopcock
- T extension
- IV catheter
- Tourniquet
- Alcohol swabs
- Tape
- Gauze
- Band-Aid/adhesive dressing
- Gloves

It may be useful to have a few pre-prepared trays, or to set up a draw with all the necessary equipment.

Intravenous access: Canulation of the forearm or hand vein is standard, but femoral vein injection is recommended when the standard approach fails to achieve full opacification of the right atrium (RA). Cannula insertion and management is subject to an Australian safety standard (see https://www.safetyandquality.gov.au/standards/clinical-care-standards/management-peripheral-intravenous-catheters-clinical-care-standard) and is typically performed by nursing or medical staff according to their usual procedural guidelines4. An Echo Physiologist may also perform cannulation and injection if local policies allow (e.g., with formal approval and appropriate training)5.

Bubble Preparation: Draw 8-10 mL saline and ⩽ 0.5 mL air into one syringe. Using the 3-way stopcock, agitate briskly between the two syringes to eliminate large bubbles and create tiny ones. A useful video of the agitation process can be found here:
https://www.youtube.com/watch?v=sYM1HYFpyXA

Communication and injection: Effective communication reduces the need to repeat the study. The injector must indicate that they are ready and then wait for Echo physiologist to say "go ahead and inject" before rapidly injecting, and they must also call out "injecting now" as the microbubbles are delivered. Care should be taken to deliver only the agitated solution, and no large bubbles of air. The onset of the video recording should correlate with the delivery of the injection.

Views: For PFO/ASD screening, you need to visualise the RA, LA and interatrial septum. The apical 4 chamber (A4C) view is usually preferred to avoid shadowing of the left heart by the highly reflective bubbles in the right heart. If the apical window is poor, a subcostal 4 chamber (S4C) view can be used as an alternative.
For PLSVC screening, the parasternal long axis (PLAX) view is very useful. If PLSVC is present, left arm injection will show the arrival of bubbles in the coronary sinus (CS) before the right sided chambers.

Settings: Tissue harmonic imaging is recommended for maximum detection of microbubbles.

Timing: At least 20 consecutive beats should be recorded. The video recording should start at the time of saline delivery, so that the first recorded beats are of a bubble-free RA.

Demonstrating the initial entry of bubbles into the RA, as well as the subsequent complete opacification of the atrium, is important. Bubbles appearing in the left atrium (LA) may be early (intracardiac shunt) or late (extracardiac): Count the beats from the time of full RA opacification to the time of appearance in the LA/LV to determine the shunt location:

- **Intracardiac shunt:** bubbles in left sided chambers appear <6 beats after full RA opacification, and mostly disappear by 16 beats after full RA opacification.
- **Extracardiac shunt:** bubbles enter the LA via the pulmonary veins. Bubbles in left sided chambers appear >6 beats after full RA opacification. By beat 16 beats there may be more bubbles in the left sided chambers than the right.

Provocative manoeuvres:
From a clinical perspective, a PFO detected via a conventional study (with gentle breathing) is likely to be associated with a greater clinical risk than a PFO that is revealed only during a provocative manoeuvre. It is recommended to do a conventional study first, and only proceed to provocative manoeuvres if indicated as per the following tables:

Conventional bubble study[6]
with quiet, normal respiration throughout

	Intra-cardiac (PFO/ASD) **shunt**	**Extra-cardiac** (transpulmonary) **shunt**	**PLSVC** Left arm injection
	A4C view preferred Show IAS	**A4C view preferred** Show PVs if possible	**PLAX view** Show CS and RV (RA not visible in this view)
Inject & record	**Start video** recording **at time of injection** Initial few beats are recorded **before** contrast reaches the heart		
Beat 1	Begin counting beats when RA and RV are fully opacified		**CS** opacifies first, 1 or 2 beats **prior** to pacification of the RV (typically 6-9 beats post injection)
≤ Beat 6	**Early appearance** of left sided bubbles indicate **intra-cardiac** R-L shunt		
~Beat 9		**Late appearance** of left sided bubbles indicate **extra-cardiac** shunt. Bubbles enter LA via pulmonary vein(s)	
~Beat 16	**Early** clearing of bubbles is underway in LA and LV	LA and LV are still filled with bubbles (**late** clearing)	
≥Beat 20	**Stop video** recording after a minimum of 20 consecutive beats		
	Negative study if no bubbles seen in LA or LV **but** Intra-cardiac shunt **NOT** yet excluded **Repeat** if negative, using additional steps	Negative study if no bubbles seen in LA or LV extra-cardiac shunt is excluded, **but intra-cardiac shunt is not excluded** **Repeat** if negative using additional steps	PLSVC excluded if RV opacifies before CS. No need to repeat

A4C, apical 4 chamber; ASD, atrial septal defect; CS, coronary sinus; IAS, Interatrial septum; IVC, inferior vena cava; LA, left atrium; LV, left ventricle; PFO, patent foramen ovale; PLAX, parasternal long axis; RA, right atrium; RV, right ventricle; SVC, superior vena cava; TV, tricuspid valve.

Additional steps to try[6]

Specific interventions during agitated saline injection designed to increase right atrial contrast and improve the detection of a PFO

Manoeuvre(s)	Specific intervention/timing	Mechanism
Add blood	10% blood added to 10% air and 80% saline	Produces smaller, more concentrated microbubbles
Cough	Performed during full RA opacification	Transiently increases RA pressure, creating an RA > LA pressure gradient
Abdominal compression[7]		
Valsalva[7] (additional contraindications apply)	Instruct patient to **release** Valsalva during full RA opacification. This causes a sudden increase in venous return to the LA.	
Femoral vein injection	Performed instead of arm injection	Useful if the inflow stream does not allow full opacification of the RA. - IVC inflow is directed to the IAS; - SVC inflow is directed to the TV

IAS, Interatrial septum; IVC, inferior vena cava; LA, left atrium; PFO, patent foramen ovale; RA, right atrium; SVC, superior vena cava; TV, tricuspid valve.

An adequate Valsalva manoeuvre with agitated saline injection greatly increases the sensitivity of TTE for the presence of a PFO, however not all patients are able to perform a Valsalva, even with repeated coaching and practice runs. Abdominal compression (using 2 hands on the abdomen) is sometimes used as a substitute.

Disclaimer:
This review article should not be used as the sole basis to make procedural decisions. Responsibility for the referral, method, and interpretation of the agitated saline bubble study ultimately lies with the supervising physician.

References and additional reading:

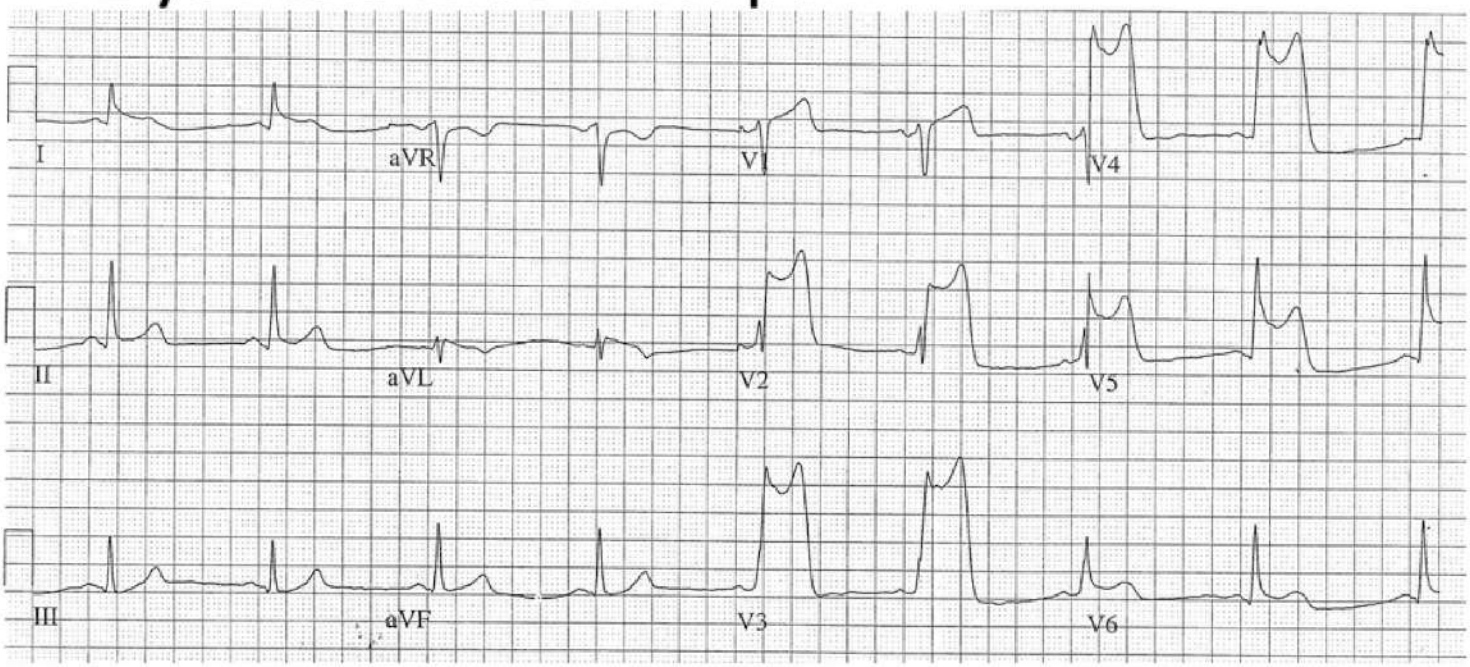
1. Maggiore P, Bellinge J, Chieng D, et al. Ischaemic stroke and the echocardiographic "bubble study": are we screening the right patients? Heart Lung Circ 2019;28:1183-9.
2. Sykes O, Clark JE. Patent foramen ovale and scuba diving: a practical guide for physicians on when to refer for screening. Extrem Physiol Med. 2013 Apr 1;2(1):10. doi: 10.1186/2046-7648-2-10. PMID: 23849539; PMCID: PMC3710076. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3710076/pdf/2046-7648-2-10.pdf
3. Tso JV, Powers JM, Kim JH. Cardiovascular considerations for scuba divers. Heart. 2022 Jun 24;108(14):1084-1089. doi: 10.1136/heartjnl-2021-319601. PMID: 34670825; PMCID: PMC9018859. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC9018859/pdf/nihms-1748394.pdf
4. Australian Commission on Safety and Quality in Health Care. Management of Peripheral Intravenous Catheters Clinical Care Standard. Sydney: ACSQHC; 2021. https://www.safetyandquality.gov.au/sites/default/files/2021-05/management_of_peripheral_intravenous_catheters_clinical_care_standard_-_accessible_pdf.pdf
5. Porter TR, Abdelmoneim S, Belcik JT, McCulloch ML, Mulvagh SL, Olson JJ, Porcelli C, Tsutsui JM, Wei K. Guidelines for the cardiac sonographer in the performance of contrast echocardiography: a focused update from the American Society of Echocardiography. J Am Soc Echocardiogr. 2014 Aug;27(8):797-810. doi: 10.1016/j.echo.2014.05.011. PMID: 25085408. https://www.asecho.org/wp-content/uploads/2014/07/2014_Contrast-Sonography.pdf
6. Mitchell C, Rahko PS, Blauwet LA, Canaday B, Finstuen JA, Foster MC, Horton K, Ogunyankin KO, Palma RA, Velazquez EJ. Guidelines for Performing a Comprehensive Transthoracic Echocardiographic Examination in Adults: Recommendations from the American Society of Echocardiography. J Am Soc Echocardiogr. 2019 Jan;32(1):1-64. doi: 10.1016/j.echo.2018.06.004. Epub 2018 Oct 1. PMID: 30282592. https://www.asecho.org/wp-content/uploads/2018/10/Guidelines-for-Performing-a-Comprehensive-Transthoracic-Echocardiographic-Examination-in-Adults.pdf
7. Ginghina C, Beladan CC, Iancu M, Calin A, Popescu BA. Respiratory maneuvers in echocardiography: a review of clinical applications. Cardiovasc Ultrasound. 2009 Aug 26;7:42. doi: 10.1186/1476-7120-7-42. PMID: 19709411; PMCID: PMC2745370. https://cardiovascularultrasound.biomedcentral.com/articles/10.1186/1476-7120-7-42

The "ECG of the Week" series has been kindly provided by Abbott Senior Technical Specialist and PiCSA Clinical Advisor **Malcolm Dennis**.

Malcolm has permitted this ECG content to be shared / sent on / printed as handouts, and used for any non-commercial teaching purposes.

ECG of the week # 22
71 yo male with chest pain

Abbott



What is the abnormality shown on the ECG?
For the extra marks this week:

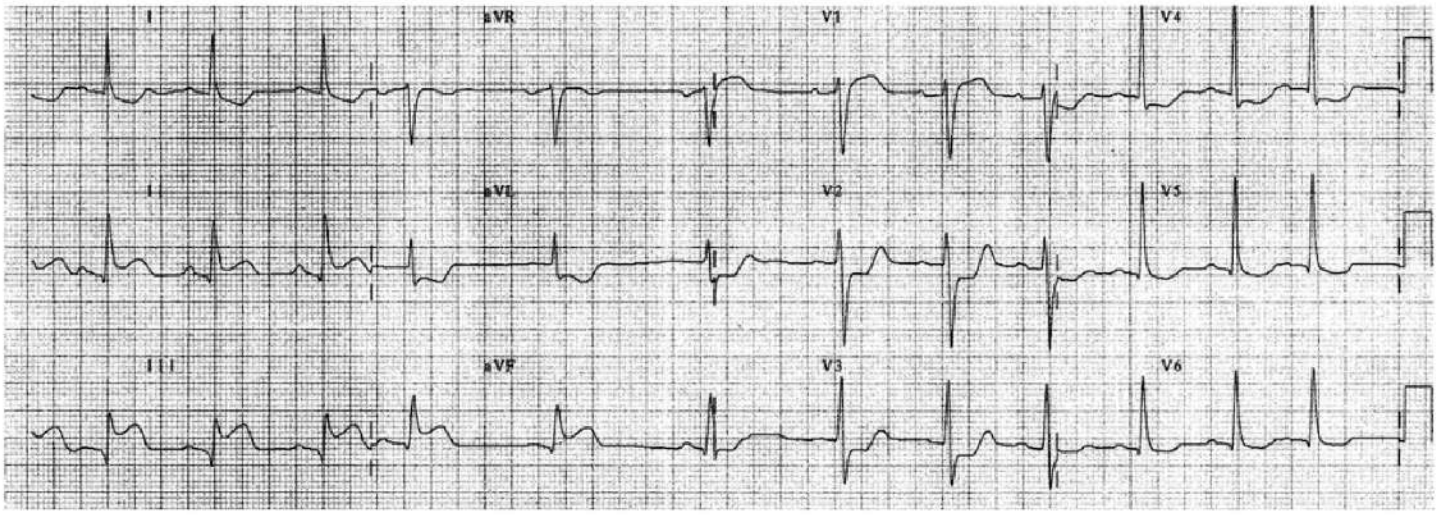
What is the old fashioned nickname for the ECG appearance?
What is the importance of leads aVR, I and aVL in considering this ECG?

ECG of the week is supplied by Malcolm Dennis, Abbott

See page 30 for the answer!

ECG of the week #23

70 yo with chest pain, elevated neck veins and BP 75/.



What is shown on the ECG above?

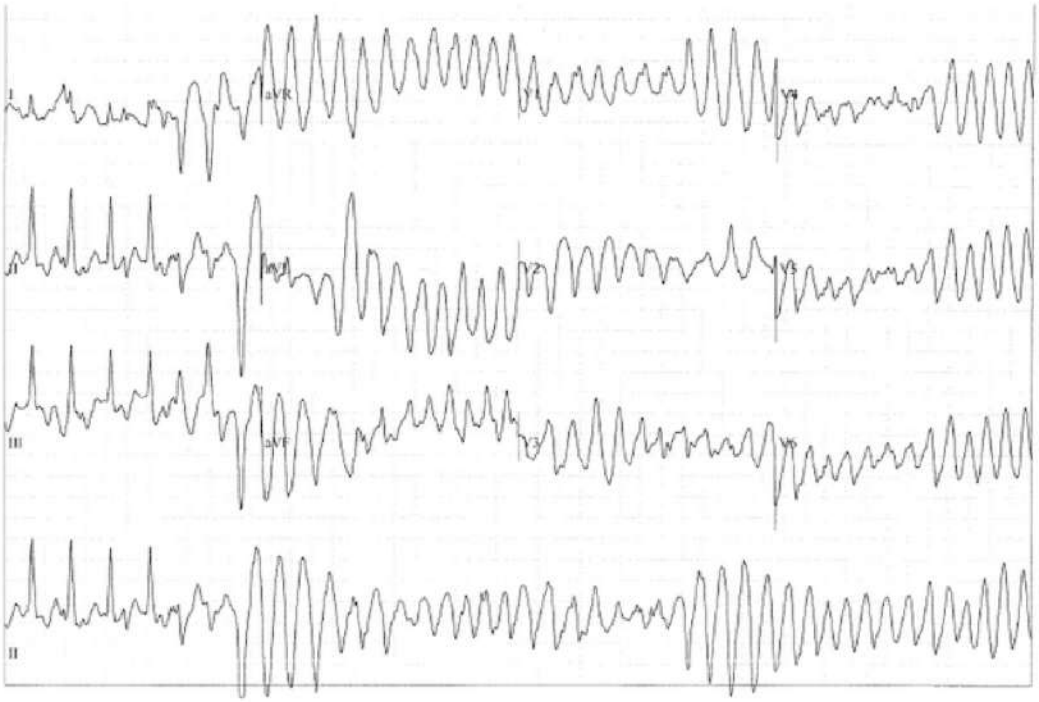
For the extra marks this week:
Two additional pieces of information can be crucial in assessing and managing this patient.
What are they

ECG of the week is supplied by Malcolm Dennis, Abbott

See page 31 for the answer!

ECG of the week #24

30 yo male with known HOCM (ECG during stress test)



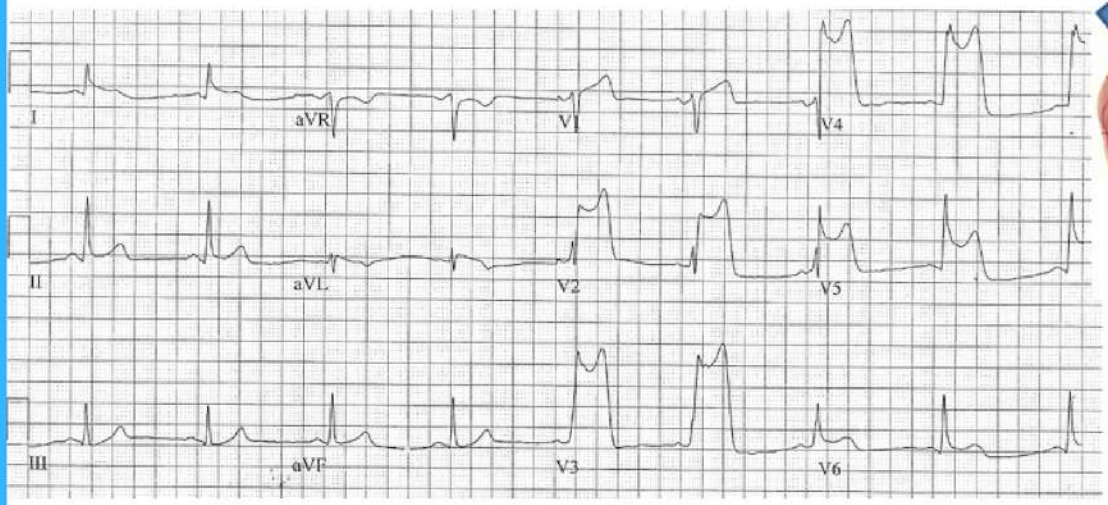
This young man's father (age 31) and two uncles died from sudden cardiac death.
Brother and sister also have HOCM.
What is the abnormality seen in the ECG?
For the extra marks this week:
What is the mechanism/cause of the ECG above, and what are the implications for stress testing?

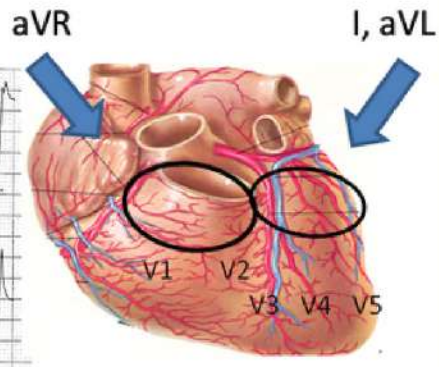
ECG of the week is supplied by Malcolm Dennis, Abbott

See page 32 for the answer!

Answers:

ECG of the week # 22
71 yo male with chest pain





Respective territories (roughly) covered by aVR (first septal branch (S1)) and leads I and aVL (first diagonal (D1) branch of LAD)

Sinus rhythm with extensive anterior / anterolateral AMI. ST elevation is striking in V1 to V5, reaching an impressive 19 mm in V3. This was once known as 'tombstone' ST elevation, presumably partly based on the shape, and partly because of the mortality once seen with the size of the infarct. It turns out that the degree of ST elevation is not a great way to judge infarct size. Instead, Hein Wellens, on one of his visits to Australia advised of the importance of looking to leads aVR, and I and aVL to judge the size of anterior infarcts in terms of how proximal the LAD lesion is and the extent of territory involved.

Leads I and aVL overlie the territory supplied by the first diagonal branch (D1), showing ST elevation when the LAD lesion is proximal to D1.
aVR overlies the territory supplied by the first septal branch (S1) and shows ST elevation when the lesion is proximal to S1.
Note that the first LAD branch may be either S1 or D1. Here aVR doesn't show ST elevation so the S1 branch must come first and is above the occlusion. I and aVL do show elevation so the lesion is proximal to S1.
If this pattern was reversed (elevation aVR but not I and aVL) then D1 is above, and S1 below the lesion
If they all have elevation then the lesion is proximal to both S1 and D1 (larger MI still)

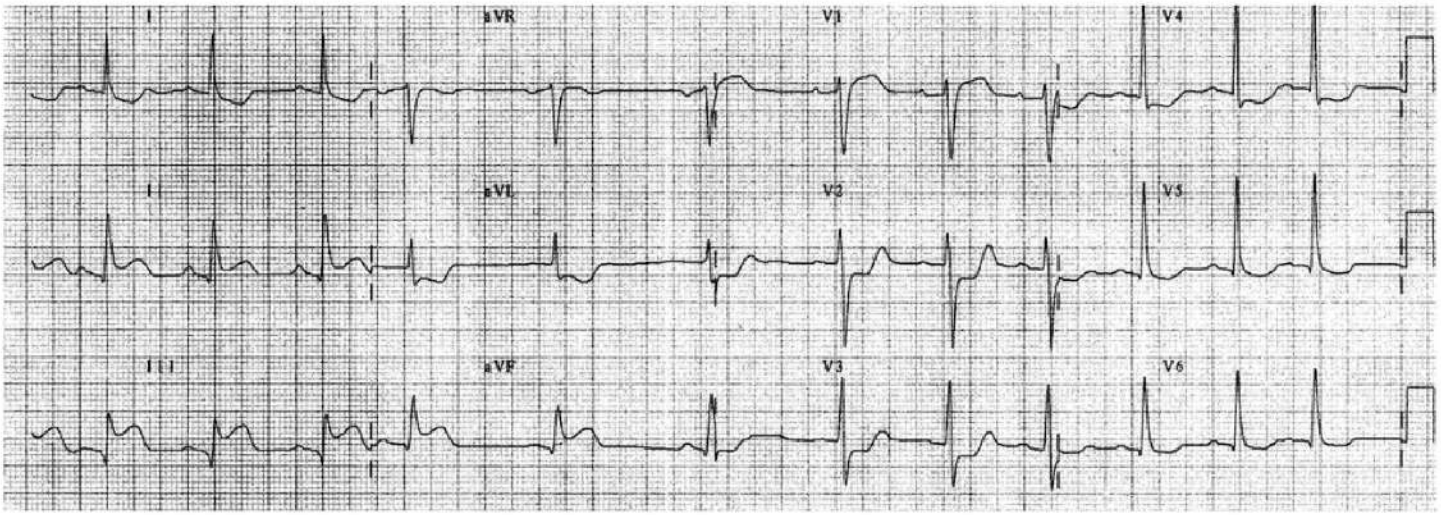
ECG of the week is supplied by Malcolm Dennis, Abbott

Answers:

ECG of the week #23

70 yo with chest pain, elevated neck veins and BP 75/.

Abbott



Acute inferior infarction is evident, with indicative ST elevation in II, III and aVF, along with reciprocal depression in I and aVL The ST depression in V2 and V3 is suspicious for posterior involvement, but posterior leads proved that not to be the case. Instead, the ST depression in V2,3 is reciprocal to right ventricular infarction which may accompany inferior MI. V1 does show ST elevation as did right sided chest leads. V2 and V3 are a fair way further to the left than V1, enabling them to show reciprocal depression.

Early identification of RV AMI is important to management which can be quite different to LV infarction. The most eye catching feature for suspicion of RV AMI is that the ST elevation in the right-sided inferior lead III is greater than in the left-sided inferior lead II. If you don't routinely record right sided leads in inferior infarction, you should certainly do them when this sign is present. And record them early as the defining ST elevation may be short lived.

Chest auscultation is also useful. There is hypotension and the JVP is elevated, but neither feature is specific to right ventricular failure. If the chest is clear on auscultation then the hypotension and venous congestion don't appear to be due to LV failure, but rather to pure right ventricular failure, in which the management is discretely different to LV failure.

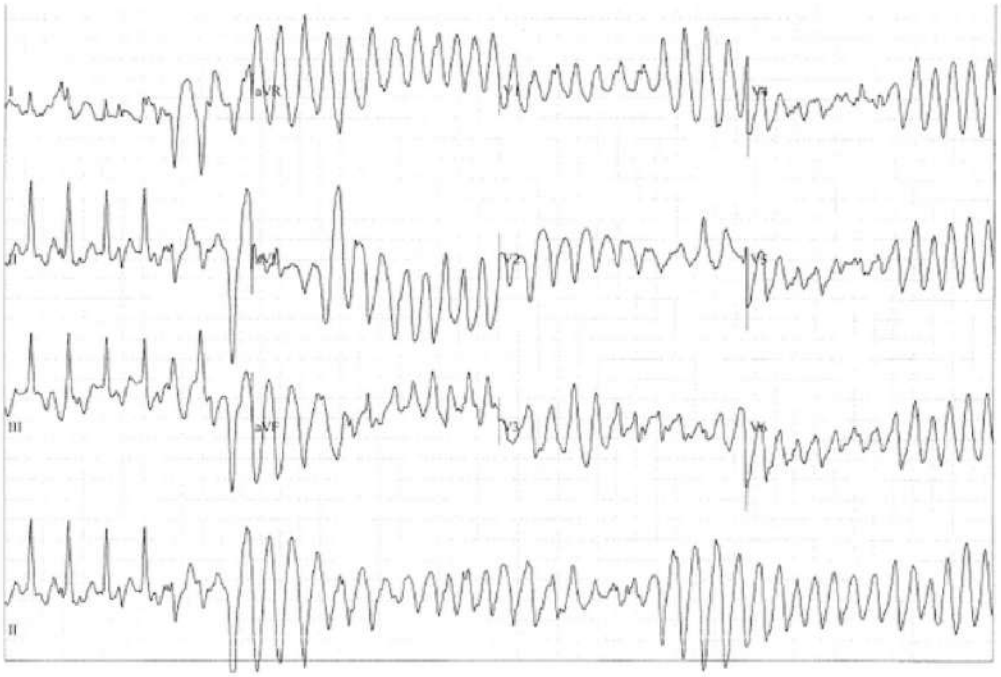
ECG of the week is supplied by Malcolm Dennis, Abbott

PiCSA

Answers:

ECG of the week #24

30 yo male with known HOCM (ECG during stress test)



Torsades de Pointes (TdP) polymorphic ventricular tachycardia, rate approx 300/min., intervenes after the initial sinus tachycardia at 150/min during stage 4 of the Bruce Protocol for exercise testing. Whilst there is some organization of the QRS (unlike VF) the polymorphism is obvious.
Hypertrophic Obstructive Cardiomyopathy (HOCM) remains a common cause of sudden cardiac death in young people.
In HOCM, the hypertrophied septum bulges into the outflow tract of the left ventricle. On exertion , greater muscle shortening exaggerates this bulging, and may critically obstruct LV outflow, resulting in collapse and VT/VF from myocardial ischaemia.
This patient (already implanted with an ICD) had requested a stress test to evaluate whether it was safe to take up exercise.
(NB: Stress testing is otherwise largely contraindicated in HOCM (as is strenuous exercise).
The patient is on Amiodarone which may account for the TdP form of VT rather than the more usual VF or monomorphic VT seen in HOCM.

ECG of the week is supplied by Malcolm Dennis, Abbott

Height and Weight Tables

Feet / Inches	Centimetres
1 foot	**30**
1 inch	**2.5**
4'3"	130
4'4"	132
4'5"	135
4'6"	137
4'7"	140
4'8"	142
4'9"	145
4'10"	147
4'11"	150
5'0"	152
5'1"	155
5'2"	158
5'3"	160
5'4"	163
5'5"	165
5'6"	168
5'7"	170
5'8"	173
5'9"	175
5'10"	178
5'11"	180
6'0"	183
6'1"	185
6'2"	188
6'3"	191
6'4"	193

Feet / Inches	Centimetres
6'5"	196
6'6"	198
6'7"	201
6'8"	203
6'9"	206

Stones/Pounds	Kilograms
1 stone	**6.4**
1 pound (lb)	**0.5**
6 stone 4 lb	40
7 stone 1 lb	45
7 stone 12 lb	50
8 stone 9 lb	55
9 stone 6 lb	60
10 stone 3 lb	65
11 stone 0 lb	70
11 stone 11 lb	75
12 stone 8 lb	80
13 stone 5 lb	85
14 stone 2 lb	90
14 stone 13 lb	95
15 stone 10 lb	100
16 stone 7 lb	105
17 stone 4 lb	110
18 stone 1 lb	115
18 stone 12 lb	120
19 stone 10 lb	125
20 stone 7 lb	130

HR ⟷ Cycle Length Table

Heart Rate	Cycle length
20	**3000**
22	2727
24	2500
26	2308
28	2143
30	**2000**
32	1875
34	1765
36	1667
38	1579
40	**1500**
42	1429
44	1364
46	1304
48	1250
50	**1200**
52	1154
54	1111
56	1071
58	1034
60	**1000**
62	968
64	938
66	909
68	882
70	**857**
72	833
74	811
76	789
78	769
80	**750**
82	732
84	714
86	698
88	682
90	**667**
92	652
94	638
96	625
98	612
100	**600**
102	588
104	577
106	566
108	556
110	**545**
112	536
114	526
116	517
118	508
120	**500**

Heart Rate	Cycle length
122	492
124	484
126	476
128	469
130	**462**
132	455
134	448
136	441
138	435
140	**429**
142	423
144	417
146	411
148	405
150	**400**
152	395
154	390
156	385
158	380
160	**375**
162	370
164	366
166	361
168	357
170	**353**
172	349
174	345
176	341
178	337
180	**333**
182	330
184	326
186	323
188	319
190	**316**
192	313
194	309
196	306
198	303
200	**300**
202	297
204	294
206	291
208	288
210	**286**
212	283
214	280
216	278
218	275
220	**273**
222	270

Heart Rate	Cycle length
224	268
226	265
228	263
230	**261**
232	259
234	256
236	254
238	252
240	**250**
242	248
244	246
246	244
248	242
250	**240**
252	238
254	236
256	234
258	233
260	**231**
262	229
264	227
266	226
268	224
270	**222**
272	221
274	219
276	217
278	216
280	**214**
282	213
284	211
286	210
288	208
290	**207**
292	205
294	204
296	203
298	201
300	**200**
302	199
304	197
306	196
308	195
310	**194**
312	192
314	191
316	190
318	189
320	**188**
322	186
324	185

2024 Maximum Heart Rate Table

Birth Year	Age at Birthday	85% Max HR	Max HR 220-age
1920	**104**	**99**	**116**
1921	103	99	117
1922	102	100	118
1923	101	101	119
1924	100	102	120
1925	**99**	**103**	**121**
1926	98	104	122
1927	97	105	123
1928	96	105	124
1929	95	106	125
1930	**94**	**107**	**126**
1931	93	108	127
1932	92	109	128
1933	91	110	129
1934	90	111	130
1935	**89**	**111**	**131**
1936	88	112	132
1937	87	113	133
1938	86	114	134
1939	85	115	135
1940	**84**	**116**	**136**
1941	83	116	137
1942	82	117	138
1943	81	118	139
1944	80	119	140
1945	**79**	**120**	**141**
1946	78	121	142
1947	77	122	143
1948	76	122	144
1949	75	123	145
1950	**74**	**124**	**146**
1951	73	125	147
1952	72	126	148
1953	71	127	149
1954	70	128	150
1955	**69**	**128**	**151**
1956	68	129	152
1957	67	130	153
1958	66	131	154
1959	65	132	155
1960	**64**	**133**	**156**
1961	63	133	157
1962	62	134	158
1963	61	135	159
1964	60	136	160
1965	**59**	**137**	**161**
1966	58	138	162
1967	57	139	163
1968	56	139	164
1969	55	140	165
1970	**54**	**141**	**166**

Birth Year	Age at Birthday	85% Max HR	Max HR 220-age
1970	**54**	**141**	**166**
1971	53	142	167
1972	52	143	168
1973	51	144	169
1974	50	145	170
1975	**49**	**145**	**171**
1976	48	146	172
1977	47	147	173
1978	46	148	174
1979	45	149	175
1980	**44**	**150**	**176**
1981	43	150	177
1982	42	151	178
1983	41	152	179
1984	40	153	180
1985	**39**	**154**	**181**
1986	38	155	182
1987	37	156	183
1988	36	156	184
1989	35	157	185
1990	**34**	**158**	**186**
1991	33	159	187
1992	32	160	188
1993	31	161	189
1994	30	162	190
1995	**29**	**162**	**191**
1996	28	163	192
1997	27	164	193
1998	26	165	194
1999	25	166	195
2000	**24**	**167**	**196**
2001	23	167	197
2002	22	168	198
2003	21	169	199
2004	20	170	200
2005	**19**	**171**	**201**
2006	18	172	202
2007	17	173	203
2008	16	173	204
2009	15	174	205
2010	**14**	**175**	**206**
2011	13	176	207
2012	12	177	208
2013	11	178	209
2014	10	179	210
2015	**9**	**179**	**211**
2016	8	180	212
2017	7	181	213
2018	6	182	214
2019	5	183	215
2020	**4**	**184**	**216**

"What day of the week was that?"

A tool to help match CIED event history with patient symptoms

2023

January

Su	Mo	Tu	We	Th	Fr	Sa
1	2	3	4	5	6	7
8	9	10	11	12	13	14
15	16	17	18	19	20	21
22	23	24	25	26	27	28
29	30	31				

February

Su	Mo	Tu	We	Th	Fr	Sa
			1	2	3	4
5	6	7	8	9	10	11
12	13	14	15	16	17	18
19	20	21	22	23	24	25
26	27	28				

March

Su	Mo	Tu	We	Th	Fr	Sa
			1	2	3	4
5	6	7	8	9	10	11
12	13	14	15	16	17	18
19	20	21	22	23	24	25
26	27	28	29	30	31	

April

Su	Mo	Tu	We	Th	Fr	Sa
						1
2	3	4	5	6	7	8
9	10	11	12	13	14	15
16	17	18	19	20	21	22
23	24	25	26	27	28	29
30						

May

Su	Mo	Tu	We	Th	Fr	Sa
	1	2	3	4	5	6
7	8	9	10	11	12	13
14	15	16	17	18	19	20
21	22	23	24	25	26	27
28	29	30	31			

June

Su	Mo	Tu	We	Th	Fr	Sa
				1	2	3
4	5	6	7	8	9	10
11	12	13	14	15	16	17
18	19	20	21	22	23	24
25	26	27	28	29	30	

July

Su	Mo	Tu	We	Th	Fr	Sa
						1
2	3	4	5	6	7	8
9	10	11	12	13	14	15
16	17	18	19	20	21	22
23	24	25	26	27	28	29
30	31					

August

Su	Mo	Tu	We	Th	Fr	Sa
		1	2	3	4	5
6	7	8	9	10	11	12
13	14	15	16	17	18	19
20	21	22	23	24	25	26
27	28	29	30	31		

September

Su	Mo	Tu	We	Th	Fr	Sa
					1	2
3	4	5	6	7	8	9
10	11	12	13	14	15	16
17	18	19	20	21	22	23
24	25	26	27	28	29	30

October

Su	Mo	Tu	We	Th	Fr	Sa
1	2	3	4	5	6	7
8	9	10	11	12	13	14
15	16	17	18	19	20	21
22	23	24	25	26	27	28
29	30	31				

November

Su	Mo	Tu	We	Th	Fr	Sa
			1	2	3	4
5	6	7	8	9	10	11
12	13	14	15	16	17	18
19	20	21	22	23	24	25
26	27	28	29	30		

December

Su	Mo	Tu	We	Th	Fr	Sa
					1	2
3	4	5	6	7	8	9
10	11	12	13	14	15	16
17	18	19	20	21	22	23
24	25	26	27	28	29	30

2024

January

Su	Mo	Tu	We	Th	Fr	Sa
	1	2	3	4	5	6
7	8	9	10	11	12	13
14	15	16	17	18	19	20
21	22	23	24	25	26	27
28	29	30	31			

February

Su	Mo	Tu	We	Th	Fr	Sa
				1	2	3
4	5	6	7	8	9	10
11	12	13	14	15	16	17
18	19	20	21	22	23	24
25	26	27	28	29		

March

Su	Mo	Tu	We	Th	Fr	Sa
					1	2
3	4	5	6	7	8	9
10	11	12	13	14	15	16
17	18	19	20	21	22	23
24	25	26	27	28	29	30
31						

April

Su	Mo	Tu	We	Th	Fr	Sa
	1	2	3	4	5	6
7	8	9	10	11	12	13
14	15	16	17	18	19	20
21	22	23	24	25	26	27
28	29	30				

May

Su	Mo	Tu	We	Th	Fr	Sa
			1	2	3	4
5	6	7	8	9	10	11
12	13	14	15	16	17	18
19	20	21	22	23	24	25
26	27	28	29	30	31	

June

Su	Mo	Tu	We	Th	Fr	Sa
						1
2	3	4	5	6	7	8
9	10	11	12	13	14	15
16	17	18	19	20	21	22
23	24	25	26	27	28	29
30						

July

Su	Mo	Tu	We	Th	Fr	Sa
	1	2	3	4	5	6
7	8	9	10	11	12	13
14	15	16	17	18	19	20
21	22	23	24	25	26	27
28	29	30	31			

August

Su	Mo	Tu	We	Th	Fr	Sa
				1	2	3
4	5	6	7	8	9	10
11	12	13	14	15	16	17
18	19	20	21	22	23	24
25	26	27	28	29	30	31

September

Su	Mo	Tu	We	Th	Fr	Sa
1	2	3	4	5	6	7
8	9	10	11	12	13	14
15	16	17	18	19	20	21
22	23	24	25	26	27	28
29	30					

October

Su	Mo	Tu	We	Th	Fr	Sa
		1	2	3	4	5
6	7	8	9	10	11	12
13	14	15	16	17	18	19
20	21	22	23	24	25	26
27	28	29	30	31		

November

Su	Mo	Tu	We	Th	Fr	Sa
					1	2
3	4	5	6	7	8	9
10	11	12	13	14	15	16
17	18	19	20	21	22	23
24	25	26	27	28	29	30

December

Su	Mo	Tu	We	Th	Fr	Sa
1	2	3	4	5	6	7
8	9	10	11	12	13	14
15	16	17	18	19	20	21
22	23	24	25	26	27	28
29	30	31				

Newsletter Article Submission deadlines

Submissions are due on the 4th Sunday of the 2nd month of each season

Autumn 2024	**Sunday 28th of April 2024**
Winter 2024	**Sunday 28th of July 2024**
Spring 2024	**Sunday 27th of October 2024**

Authors can claim CPD points for contributing to this newsletter.
Submissions should be emailed to secretary@picsa.org.au

"Hello my name is ... I am a Cardiac Physiologist"

One very easy way to promote the visibility, recognition and regulation of the Cardiac Physiology profession is to simply introduce ourselves, and state what we are, using the recommended title "Cardiac Physiologist". We can all do this – regardless of whether or not our employer has updated our statement of duties in line with the national recommendation.
Simply introducing ourselves correctly will educate those around us about our professional identity. It seems like a simple small thing, but it is a very powerful way to connect with patients and to advance our profession.

Remember to always say **"Hello my name is ... I am a Cardiac Physiologist"**

More information can be found in on this page located on our website
https://picsa.org.au/about/#core-documents

Conferences and Resources

PiCSA online Education Portal (members only)
https://www.picsa.org.au

Cardiac Electrophysiology Institute of Australia (CEPIA)
Grad dip of Cardiac Electrophysiology - now delivered 100% online
https://www.cepia.com.au/

Cardiac Physiology in Practice
Online ECG course
https://cardiacphysinpractice.com/

Echo Ed online education
https://echoedonline.com.au

Echo Supervisors Summit
https://echosupervisor.com/

Australasian Sonographers Association (ASA)
www.sonography.org

Cardiac Physiology Australia (CPOZ)
20 April 2024
Indooroopilly Golf Club, Indooroopilly, QLD
www.cpoz.au

Port Douglas Heart Meeting and Expo
5-8 June 2024
Sheraton Grand Mirage Resort, Port Douglas

Call for Volunteers!

There are many ways to volunteer within the PiCSA organisation.

Interested? Get in touch!
secretary@picsa.org.au

Affliated Partners:

asa australasian sonographers association

AUSTRALIAN COUNCIL FOR CLINICAL PHYSIOLOGISTS

STRUCTURAL HEART DISEASE AUSTRALIA

CARDIAC PHYSIOLOGY IN PRACTICE

CEPIA CARDIAC ELECTROPHYSIOLOGY INSTITUTE OF AUSTRALASIA

For PiCSA affiliation enquiries, please email:
membership@picsa.org.au

There are a range of ways you can get information and find out what's happening with PiCSA, connect now!

PiCSA

Professionals in Cardiac Sciences Australia (PiCSA)

@picsa_au

@PiCardiacSA

www.picsa.org.au

Thank you for reading