

THE RHYTHM STRIP

The newsletter for Professionals in Cardiac Sciences Australia

Winter 2026

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Our Mission To support, represent, and advance the Cardiac Physiology profession.

Our Vision A skilled, recognised, and regulated Cardiac Physiology workforce meeting Australia's healthcare needs now and into the future.

Our Values

Clinical Excellence We lead the Cardiac Physiology profession to achieve the highest standards of patient care through evidence-based practice, ongoing education, and adherence to professional and ethical guidelines.

Visibility We advocate for the recognition of Cardiac Physiologists as a vital, distinct profession among policymakers, the healthcare community, and the public.

Empowerment We equip Cardiac Physiologists with the tools, resources and support to advance their roles and rights within healthcare. We actively influence systems and policies to enable our members to achieve their full potential.

Accountability We act with integrity, are open and transparent to our members, make evidence-informed decisions, encourage ethical behaviour at all levels across the profession, and accept responsibility for strategic actions and outcomes.

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From the Chair

Sam Burgoyne (WA)



A message from Professionals in Cardiac Sciences Australia (www.picsa.org.au)

PiCSA has had a lot of things going on over the last few months! One of our main efforts that was very exciting was involvement at the CSANZ Annual Scientific Meeting 2026. There was a lot of work and planning behind the scenes in the lead-up to this meeting, and we were particularly keen to have as many of the PICSA Board as possible together in person throughout the conference. Our main goal was to represent our profession – be that at the booth, doing presentations, informal conversations with key stakeholders, even just wearing our badges – it was a multi-pronged approach!

A sincere thank you to everyone who came and spoke with us at the PICSA booth, caught us throughout the sessions, or simply stopped to say hello. It was wonderful to meet some of our members and supporters in person, and to have the opportunity for meaningful conversations about our profession and where we are heading.

Having the Board together provided an important opportunity to reflect on PICSA's strengths, as well as where we can improve and where we need additional support. We had some really valuable and constructive conversations, both within the Board and with members and non-members throughout the conference.

The feedback was overwhelmingly positive, but importantly, it was also honest and constructive. We heard ideas about how PICSA can become more effective, more visible and ultimately more impactful for the profession. These conversations have helped shape some important discussions about where PICSA is heading and what our priorities should be moving forward.

A key area of focus is advocacy and the future of cardiac physiology. We are continuing to see changes in scope of practice, technology and the roles of other professions within areas that have traditionally been part of cardiac physiology. While collaboration across professions is important and something we strongly support, it also highlights the need for appropriate professional governance, clear scope of practice and a strong voice advocating for the role and value of cardiac physiologists.

We need to be thinking proactively about where our profession is going — not simply responding to change once it happens.

We are also continuing to strengthen PICSA's internal operations. Our Board is made up of passionate and dedicated volunteers, and we want to make sure their time and expertise are being used where they can have the greatest impact. We are therefore looking at ways to reduce some of the administrative workload and develop stronger operational support, allowing the Board to focus more on resource development, professional advocacy, education and initiatives that directly benefit our members.

As part of this, we are also looking for new Board members with fresh perspectives, ideas and insight. If you are passionate about cardiac physiology and would like to contribute to shaping the future of our profession, we would love to hear from you.

Education and professional development also remain a major focus. We have been working behind the scenes on a range of educational initiatives, including webinars such as our recent workplace update session. The challenges facing cardiac physiologists vary significantly between states, and the recent discussions have highlighted just how complex some of these issues are. Thank you to all of the presenters who contributed to this highly valuable and insightful session. Also, the CIED Scope of Practice currently being developed by PiCSA will be an important component in addressing some of the challenges discussed. A sincere thank you to everyone who has contributed their time, expertise and effort to this work. It is coming together extremely well, and we look forward to continuing to progress this important initiative.

We recognise that PICSA does not have all the answers. However, we are committed to listening, learning, connecting people and exploring practical ways we can support our members. Sometimes that starts simply by creating a space for people to share their experiences and realise that they are not facing these challenges alone.

We are also continuing to build positive relationships with other organisations and explore opportunities for collaboration. We are particularly excited about the possibility of working with CSANZ on an imaging quiz in WA — so watch this space!

There is a lot happening behind the scenes at PICSA, and we are excited about the direction we are heading. Most importantly, we want PICSA to be an organisation that listens to its members, represents the profession thoughtfully and constructively, and actively contributes to a strong and sustainable future for cardiac physiology.

Thank you to everyone who continues to contribute, challenge us, support us and share your ideas. We are building PICSA together, and your voice matters.

Sam Burgoyne

Join the ACCP to Elevate Your Career & Legitimise Your Profession

Accreditation and registration are essential for all Cardiac Physiologists, not just those specialising in Echo. If you work in Echo, your accreditation registry is the ASAR. However, if your expertise lies in ECG (non-invasive), Cath Lab, Cardiac Devices, and/or Electrophysiology, the Australian Council for Clinical Physiologists (ACCP) offers you similar recognition.

The ACCP is a mark of professionalism and helps establish national standards. By registering with the ACCP, you enhance your professional status and gain recognition from employers, patients, and professional bodies. Additionally, you can add the post-nominal “ACP – Cardiac” to your credentials.

The ACCP membership year aligns with the financial year. Visit the Australian Council for Clinical Physiologists website at <https://theaccp.org.au/> to learn more.

Supporting ACCP registration helps to improve patient safety and advances our profession as a whole. Together, let's elevate Cardiac Physiology to new heights.



Vice Chair's Update

Stronger Together: Why Now Is the Time to Unite

Alicia Donovan (WA)



Stronger Together: Why Now Is the Time to Unite

Australia's healthcare system is changing rapidly.

An ageing population, increasing patient complexity, workforce shortages and advances in technology are transforming the way healthcare is delivered. Allied health and health science professionals are increasingly at the forefront of this change, delivering highly specialised care that improves outcomes for patients every day.

Yet while our roles continue to expand, many of our professions face the same challenges—recognition, workforce shortages, inconsistent career pathways, limited visibility, and a lack of coordinated workforce planning.

Perhaps our greatest challenge, however, is that we continue to advocate as individual professions rather than as a collective.

Imagine what we could achieve if allied health and health sciences spoke with one voice.

Professional leadership, governance and meaningful consultation have never been more important. Across Australia, discussions around organisational structures and allied health leadership remind us that decisions affecting our professions must involve those who understand them. Without strong professional representation, there is a real risk that workforce capability, education and service development will be shaped without the voices of those delivering care.

But representation alone is not enough.

We must also invest in developing our professions from the ground up.

That starts with creating greater awareness of allied health and health science careers at university entry level, strengthening profession-specific education pathways, supporting clinical placements and mentorship, and developing clear pathways into advanced practice and leadership. Professions are not built by chance—they are built through deliberate investment.

At the same time, we cannot ignore the expertise we are losing. Across Australia, experienced clinicians are leaving our professions because they cannot see sustainable career pathways or opportunities for progression. Every time this happens, we lose years of knowledge, mentorship and clinical capability that cannot easily be replaced.

Retention is just as important as recruitment.

For emerging professions such as cardiac physiology, a unified professional identity is equally important. Consistent job titles, recognised scopes of practice and national education and competency frameworks are not simply administrative issues—they are the foundations of professional recognition, workforce planning and future growth.

Finally, we must move beyond traditional hierarchies and embrace genuine multidisciplinary collaboration. Every profession brings specialised expertise developed through rigorous education, training and experience. The best healthcare outcomes are achieved not when one profession leads, but when each profession is recognised as the expert within its own scope and works collaboratively towards a common goal.

PiCSA has always believed that stronger professions are built through education, collaboration, advocacy and professional leadership. As we continue to advance cardiac physiology, we also have an opportunity to contribute to a broader conversation about the future of allied health and health sciences in Australia.

The future of allied health and health sciences will not be defined by chance. It will be defined by the decisions we make today—how we educate, how we collaborate, how we advocate and how we support one another.

If we want stronger professions tomorrow, we must invest in them today. We must develop them together, champion them together and lead them together.

Because the future of Australian healthcare will not be built by individual professions working in isolation. It will be built by allied health and health sciences standing together—with one voice, one purpose and a shared commitment to delivering the very best care for our communities.

**Stronger together. One collective voice.
A stronger future for allied health and health sciences.**



PiCSA CASE FILES

Do you have an interesting case? Share it with us!

PiCSA Case Files is our new series showcasing interesting and educational cases from across all areas of Cardiac Physiology.

Designed to challenge clinical thinking, spark discussion and share valuable learning across our community.

Have you seen our first Case File?

Check it out on PiCSA's Facebook, LinkedIn, Instagram or X and see if you can pick the diagnosis!

YOUR CASE COULD BE NEXT!

Have an unusual, challenging or educational case worth sharing? We'd love to hear from you.

✉ Submit your case: info@picsa.org.au

Share a case. Start a conversation. Support learning.



Professionals in Cardiac Sciences Australia (PiCSA)



Professional in Cardiac Sciences Australia (PiCSA)



@picsa_au



@PiCSA_au





Membership Report + Announcements

Alicia Donovan (WA)

Professional Members 289
Early Career 44
Affiliate 30
Student 65
Life 4
Total 432

PiCSA Membership

Membership Update - Heading Towards 500 Members

PiCSA is heading rapidly towards 450 members, and we're not stopping there. Our next goal? **500 members**.

The continued growth of PiCSA reflects the strength of our profession and the value of having a national community connecting cardiac scientists across Australia.

Making membership easier

As our membership grows, we're continuing to improve the member experience.

We've been working behind the scenes to streamline the PiCSA website and membership area, making your membership account details more visible and easier to access. Updating your personal and professional details is now simpler too.

We encourage members to check their profile and make sure their details are up to date so you don't miss important PiCSA communications, education opportunities and member updates.

Share your expertise

Our members are our greatest resource, and we want to showcase the knowledge and experience within our growing community.

Have you

- published a paper or poster
- presented at a conference
- developed CPD content
- completed an interesting project or
- got an area of expertise you'd like to share?

We'd love to hear from you.

Members can contribute to the PiCSA education program by presenting a webinar, sharing research or professional work, contributing educational content, or suggesting topics and speakers for future sessions.

Help us reach 500

Do you have colleagues who aren't yet members? Encourage them to join PiCSA and be part of our growing professional community.

450 is within reach. 500 is the goal. Let's get there together.



Education Meetings – Save the Date



PiCSA EDUCATION MEETING CATH LAB



Presenters:

Jordan Jago:
Coronaries for Cardiac
Physiologists



Alexis Arriagada:
TBC

Tuesday 22nd September

- 7:00pm AEST
- 6:30pm ACST
- 5:00pm AWST

Online via Zoom

Register Now

www.picsa.org.au/events

PiCSA in Action: Education & Event Highlights

Connecting at CSANZ 2026

PiCSA was pleased to once again be part of the CSANZ Annual Scientific Meeting, held in Sydney in August. As one of the major events on the cardiology calendar, the meeting provided a valuable opportunity to connect with the wider cardiac community and represent Cardiac Physiologists from across Australia.

Throughout the meeting, the PiCSA booth was a great meeting point for members, Cardiac Physiologists, Doctors, students and other professionals working across cardiology. We enjoyed catching up with familiar faces, meeting many new ones and sharing more about PiCSA, our current activities and the work we are doing to support, advocate for and advance the Cardiac Physiology profession.



One of the most valuable parts of attending CSANZ was having the opportunity to speak directly with our members. We heard some wonderful positive feedback about PiCSA's education, resources and advocacy, while also having important conversations about what matters to our members, where we can continue to improve and what they would like to see from PiCSA in the future. These conversations are incredibly valuable in helping guide the work of the Association.



PiCSA in Action: Education & Event Highlights

CSANZ was also a particularly special meeting for the PiCSA Board, with all Board members able to come together face-to-face. While we work closely throughout the year through regular online meetings, emails and projects, for some members of the Board this was actually the first time they had met in person, despite having worked together for several years!

Having time together away from the Zoom screen provided an opportunity to strengthen relationships, share ideas and discuss PiCSA's priorities and future direction. As a nationally based Board, opportunities to be together in the same place are relatively rare, making the time in Sydney particularly valuable.

Of course, the Annual Scientific Meeting itself also provided an excellent opportunity to hear about the latest developments, research and innovation across cardiology and to connect with colleagues from across Australia and beyond.

A big thank you to everyone who visited the PiCSA booth, stopped us in the conference halls or took the time to have a conversation with one of our Board members.

Connecting with our members and the wider Cardiac Physiology community is an important part of what PiCSA is all about, and we look forward to continuing those conversations.



The PiCSA Board: Alicia Donovan, Ciara Devoy, Luke Shanahan, Sam Burgoyne, Justin Gordon, Bianca Coelho and Jordan Jago.



PiCSA in Action: Education & Event Highlights



Ciara Devoy, PiCSA Secretary:

Thoroughly enjoyed CSANZ 2026 — always a great event to collaborate, share methods and ideas, and problem-solve across different states and territories. The highlight was definitely meeting the PiCSA team in person for the first time — and not on Zoom!

Justin Gordon, PiCSA Board Member:

Attending CSANZ was a fantastic opportunity to reconnect with past colleagues and meet cardiac physiologists from across Australia and New Zealand. As a PiCSA Board member, I was able to engage with both current and prospective members on topics ranging from professional representation to educational opportunities. A highlight of the conference was the inaugural State of Origin Imaging Quiz, won by the all-conquering Queensland team!



Sam Burgoyne, PiCSA Chair:

CSANZ gave me the opportunity to hear multidisciplinary discussions around other professions expanding their scope of practice. It certainly got the neurons firing, how can we create meaningful advanced pathways and new opportunities within our own profession?

Alicia Donovan, PiCSA Vice Chair:

A great few days at CSANZ! It was a great opportunity to catch up with colleagues, hear what other centres are getting up to, share ideas, and meet some new faces (and potential PiCSA members!). The PiCSA Board get-together was definitely a highlight too. Queensland team!



CSANZ Competition Winner – Congratulations Casey Sear

Congratulations to Casey Sear, winner of the PiCSA competition held at the recent CSANZ Annual Scientific Meeting.

Casey received a one-year PiCSA membership and a bottle of wine as his prize.

Thank you to everyone who entered the competition and stopped by the PiCSA booth to connect with our Board and representatives. It was a great opportunity to discuss Cardiac Physiology, the work of PiCSA, and the future of our profession with colleagues from across the cardiac physiology community.

Congratulations again to Casey, and thank you to everyone who visited us at CSANZ.



CSANZ Competition Winner: Casey Sear

National Workforce Meeting: Shared Challenges and Opportunities

At PiCSA's recent **National Workforce Update Meeting**, representatives from across Australia provided an overview of the Cardiac Physiology workforce in their states and territories. Despite differences in how services are structured, a number of clear themes emerged.

Recruitment and retention remain significant challenges, particularly for experienced Cardiac Physiologists working in echocardiography, electrophysiology and cardiac devices. Increasing patient volumes and service complexity are placing further pressure on already stretched teams, while competition from private practice and interstate opportunities continues to affect retention.

Another recurring issue was **career progression and professional recognition**. Differences in classifications, remuneration, role definitions and opportunities for advancement remain evident across Australia. Several presenters also highlighted concerns around role substitution and the need to ensure the expertise and scope of Cardiac Physiologists are appropriately recognised.

Rural and regional workforce shortages remain particularly challenging, contributing to unequal access to specialised cardiac services. However, outreach clinics and remote monitoring are helping services extend care beyond metropolitan centres.

There were also plenty of **positive developments**. Across the country, services are investing in graduate training, expanding advanced and physiologist-led roles, developing remote monitoring programs, improving classifications and creating new workforce positions.

Overall, the presentations highlighted a profession that is continuing to grow in **scope, complexity and responsibility**. They also reinforced the importance of a coordinated national approach to workforce development, career pathways, professional recognition and standards of practice as Cardiac Physiology continues to evolve.

We would like to thank all of our presenters for sharing their time, insights and experiences, and for contributing to such a valuable discussion about the future of Cardiac Physiology in Australia.

Education Meetings – Registrations Opening Soon



PICSA EDUCATION MEETING

Paediatrics

WEDNESDAY
28 OCTOBER 2026

7:00PM AEDT
6:30 PM ACDT
6:00 PM AEST
5:30 PM ACST
7:00 PM AEST

www.picsa.org.au/events
Online via ZOOM

Atrioventricular Septal Defects

Andrew Osborne Echocardiographic assessment of AVSD	Denise Bridges Electrical Anatomy of AVSD	Justin O'Leary Echocardiographic assessment of AVSD repair
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Nominations Open: the limbic Cardiology Award 2026

Nominations are now open for the **limbic Cardiology Award 2026**, recognising health professionals who go above and beyond for their patients and the cardiology profession.

Importantly, the award is open to **all health professionals working in cardiology**, including **Cardiac Physiologists**.

PiCSA has advocated for Cardiac Physiologists to be recognised as eligible for this award, providing an opportunity for our profession to be nominated and celebrated alongside our cardiology colleagues.

Who could you nominate?

Think of a colleague, mentor or Cardiac Physiologist who has made a difference through patient care, education, innovation, research, advocacy or simply going above and beyond in their role.

For nominations: Cardiac Physiologists can still nominate. The form asks whether the nominee is AHPRA-registered, members should select “yes” where the person being nominated is ACCP or ASAR accredited.

Nominations close 28 September 2026 and take approximately five minutes to complete.

Submit a nomination here: <https://thelimbic.com/cardiology/nominations-open-for-the-limbic-cardiology-award-2026/>

This is a great opportunity to shine a light on the Cardiac Physiologists who make a difference every day.

the**limbic***

Cardiology Award 2026

Recognising those who go above and
beyond for their patients and specialty.

Nominations open



Views that Inspire: Celebrating Clinical Curiosity

PiCSA is pleased to share more of the fantastic entries we received from our “Views That Inspire” competition earlier this year.

The creativity and variety across the submissions were outstanding, and we’re excited to continue showcasing some of the inspiring views shared by our community.

Lucy Blake

Deputy Chief Cardiac Sonographer
and Quality Management
Victorian Heart Hospital, VIC.

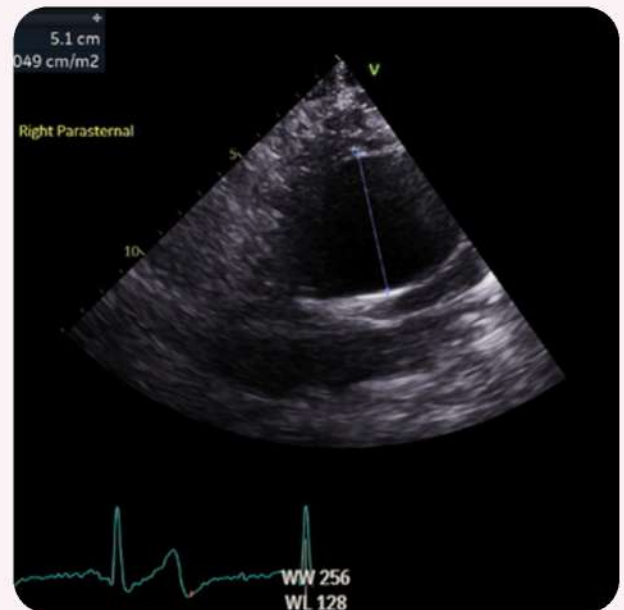
This patient was referred for dilated ascending aorta noted on CT after recent severe pneumonia. No previous TTE. The patient had very difficult parasternal imaging (ex smoker, recent pneumonia).

The aorta was not visible. Other windows also very challenging. At near completion still not having seen the ascending aorta the patient was getting uncomfortable and also shared she suffered from polymyalgia, reluctantly I asked if she could roll on her right side for one last image I promised.

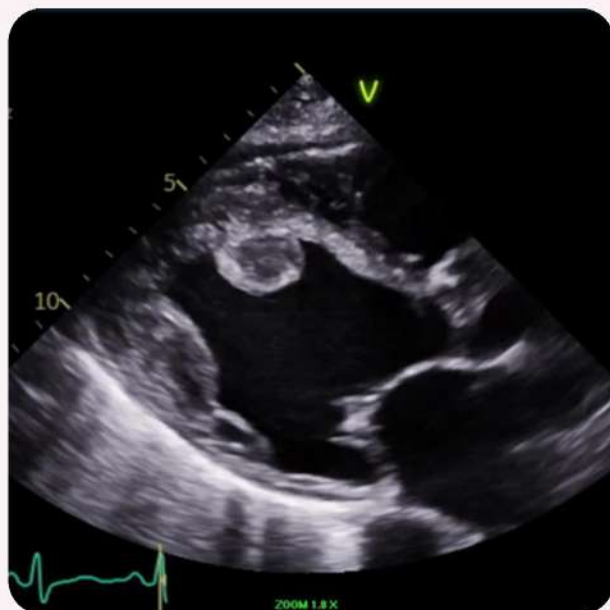
I instantly saw the ascending aorta BEAUTIFULLY right parasternally. Gratification!

It measured 5.1cm - reported as moderate to severely dilated.

“Gratification!”



“Beyond the Images”



Olivia Magnay
Cardiac Physiologist
Royal Hobart Hospital, TAS

This case inspired me because it reminded me that my role as a cardiac physiologist extends far beyond acquiring images and measurements.

Without discussing results, I took the time to ask the right question and build a rapport with the patient during this echo that revealed his medical history. By the time I had completed the scan and alerted the medical team, I had a clear clinical picture that the patient was taking pro-thrombotic medicine which deviated from conventional therapy.

Moments like this reinforce that meaningful patient care often starts with curiosity, conversation and connection, not just taking pictures.

Health literacy and its role within cardiovascular disease: A primer from a cardiology perspective

Crystal Williamson, Yingyan Chen,
Tuppence Richman & Prof Kim Greaves

ABSTRACT

Introduction:

Cardiovascular disease is the leading cause of mortality and morbidity worldwide. Cardiovascular disease is preventable by managing modifiable risk factors, but this requires support to improve patients' health literacy. Patients with cardiovascular disease have significant challenges understanding and engaging with healthcare, which can compromise their health outcomes.

Aims:

This commentary reviews relationships between health literacy and cardiovascular disease and proposes recommendations for improving cardiovascular management in healthcare settings.

Results:

Improving health literacy is a significant issue in cardiovascular disease prevention. There are factors that are associated with health literacy at both individual and system levels which may influence a patient's health literacy. Health professionals can support heart health literacy through checking their patients' health literacy needs, being aware of their own misconceptions and biases, using written, verbal, and visual communication strategies that are easy to understand, and avoiding unexplained medical jargon. Organisations can support this through training multidisciplinary teams and providing clear discharge information that patients can understand and act on.

Conclusion:

Supporting health literacy among cardiovascular disease patients is paramount to 1) enable patients to regain control over their health, 2) decrease cardiovascular disease hospital admissions and 3) reduce costs to the public health sector. Both individual and system level interventions are needed to support heart health literacy and improve patient outcomes in healthcare settings.

Heart disease is a very common condition. Most cases are linked to risk factors that are preventable. Helping patients with heart disease understand their condition is important for managing the disease. This can lead to better health for patients. Patients with heart disease are expected to understand a lot of new medical terms.

They may need to make lifechanging decisions while feeling stressed or unwell. Health professionals and services can do more to help patients understand their heart health and reduce their risk of poor outcomes.

Read the full article here:

<https://www.tandfonline.com/doi/full/10.1080/28355245.2025.2590786>



The Mysterious Case of the Methamphetamine Associated Cardiomyopathy and the Carcinoid-like Tricuspid Valve

Bethany Cooper, Echocardiographer,
Western Cardiology

Introduction

Presentations of methamphetamine-associated cardiomyopathy (MCMP) are becoming increasingly prevalent with the rise of methamphetamine abuse. The pathophysiology of MCMP is multifactorial, resulting from both catecholamine-induced cardiotoxicity (calcium overload, coronary vasoconstriction/vasospasm and oxidative stress) and chronic excessive sympathetic stimulation (increased heart rate, blood pressure and myocardial contractility).

Common echocardiographic findings of MCMP include ventricular dilation, impaired systolic function and valvular disease, all contributing to the clinical diagnosis of dilated cardiomyopathy and heart failure (1,2). Whilst right-sided heart disease is also well documented in cases of MCMP, it is usually in the context of biventricular failure and functional valve disease secondary to ventricular remodelling and pulmonary arterial hypertension. Less well studied is whether serotonergic activity, specifically associated with frequent methamphetamine use, may contribute to valvular degeneration (3).

I present a patient with MCMP who exhibits tricuspid valvulopathy similar to that seen in carcinoid heart disease (CaHD), for which there is a well-established association with serotonin.

Case Presentation

This 46 year old man initially presented in May 2020 with 1 week of shortness of breath and dry cough, and 2 weeks of fevers and visual hallucinations, on a background of active intravenous methamphetamine use, moderate alcohol and marijuana consumption, and multiple comorbidities including hepatitis C and emphysema. On examination, he had a heart rate of 110 bpm, blood pressure of 100/90 mmHg and was afebrile. A loud pansystolic murmur was noted, though he had no ankle oedema and JVP was not elevated. Additional testing however showed a troponin of 20, BNP of 2246 and cardiomegaly on chest x-ray. He was referred for an echocardiogram to exclude infective endocarditis and assess general cardiac structure and function due to his history of IVDU, murmur and tachycardia. This echocardiogram showed severe dilated left ventricular failure with a left ventricular end diastolic dimension (LVEDd) of 7.2 cm, left ventricular ejection fraction (LVEF) of 22% and severe mitral regurgitation. The right ventricle was mildly dilated at 4.7 cm with preserved systolic function. The tricuspid valve leaflets were moderately thickened and there was moderate-severe tricuspid regurgitation (Figures 1 and 2). After infective endocarditis was excluded, he was discharged with a diagnosis of methamphetamine-induced dilated cardiomyopathy and commenced on low dose ramipril and frusemide.



Figure 1: Apical 4-chamber of initial echo (May 2020).

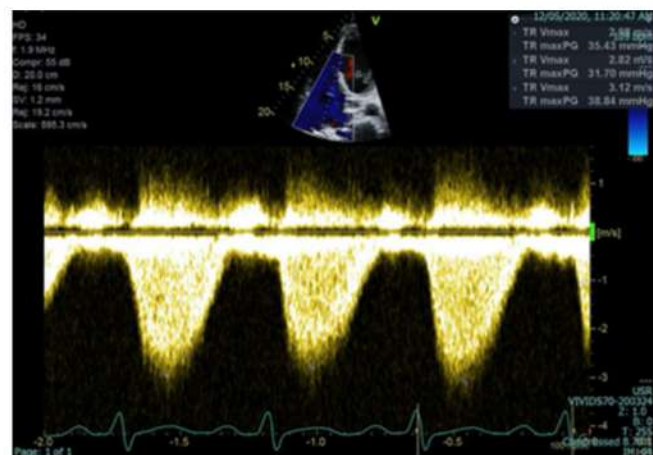


Figure 1: Continuous wave Doppler of moderate-severe tricuspid regurgitation (May 2020).

In December 2020, he presented to hospital again, this time showing immediate signs of congestive cardiac failure exacerbation, with pitting oedema to the knees and elevated JVP. He was commenced on entresto and ivabradine, however was found to be non-compliant with medication and fluid restrictions. He was referred for a follow up echocardiogram to assess progression of his dilated cardiomyopathy, which showed a relatively stable left ventricle with LVEDd of 7.0 cm and LVEF of 19%. The right ventricle however was now severely dilated at 5.9 cm with severe systolic impairment. The tricuspid valve leaflets were largely immobile and retracted, with complete failure to coapt, and there was torrential tricuspid regurgitation (figure 3 and 4). The RA volume had increased from 50 ml/m² to 81 ml/m². Due to the appearance of the tricuspid valve the treating team was encouraged to exclude carcinoid heart disease, however they were made aware of previous literary reports of methamphetamine-induced valvulopathy resembling carcinoid disease. As further testing uncovered no evidence of neuroendocrine tumour or carcinoid syndrome, a methamphetamine and serotonergic-associated process was presumed to be a contributor in the right heart valvulopathy.



Figure 3: Apical 4-chamber of second echo (Dec 2020) showing significant right heart dilatation.

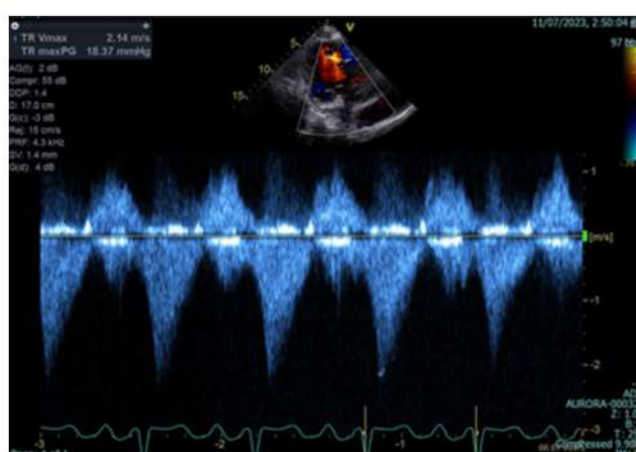


Figure 4: Continuous wave Doppler of torrential tricuspid regurgitation (Dec 2020).

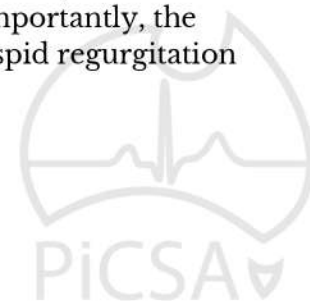
Over the next two echocardiograms, his right heart continued to dilate (2021: RV 5.9 cm, RA vol 106 ml/m², 2023: RV 6.7 cm, RA vol 117 ml/m²) and his tricuspid leaflets became increasingly retracted with persistent torrential tricuspid regurgitation. The patient unfortunately was not a candidate for transplant due to his history of drug use. Figure 5 shows a 3D tracing of the tricuspid coaptation defect measuring 3.8 cm² at his last scan with us in 2023.



Figure 5: 3D tracing of tricuspid coaptation defect (2023).

Discussion

Multiple echocardiographic features in this case bore a striking resemblance to classical appearances of carcinoid heart disease, primarily the progressive thickening and retraction of the tricuspid valve leaflets, but also failure of leaflet coaptation and resultant severe tricuspid regurgitation, as well as progressive right ventricular and right atrial dilatation. Importantly, the specific tricuspid valvulopathy of this case differs from the usual functional tricuspid regurgitation seen in methamphetamine-associated right ventricular dilation.



Although further studies are required on the development of CaHD, there is strong evidence supporting the role of serotonin in fibroblast growth and fibrogenesis, the resulting fibrous tissue deposition on valve leaflets and subvalvular apparatus, and the subsequent stiffening and retraction of valve leaflets (4,5). As the lungs metabolise the majority of circulating serotonin, this disease rarely affects the left heart, unless there is a pathway available for serotonin to bypass the lungs, such as a right-to-left shunt (5). In this case, the left-sided valve disease (severe mitral regurgitation) is still presumed to be secondary to methamphetamine-induced dilated cardiomyopathy, as it was well documented before the development of the right-sided valvulopathy.

Although the patient had no evidence of a neuroendocrine tumour, his history of chronic methamphetamine use provides a rare, but possible alternative explanation for the distinctive appearance of the tricuspid valve. The relationship between methamphetamine use, serotonin and right-sided valvular dysfunction has been suggested before in the setting of methamphetamine-induced pulmonary arterial hypertension, where tricuspid and pulmonary valve disease resembling CaHD were described (3). Similar carcinoid-like valvular disease has also been reported in patients using other drugs associated with serotonergic activity (6).

Conclusion

This case highlights the importance of using multimodality imaging and considering a patient's history when assessing echocardiographic findings. Despite several echocardiographic findings typical of carcinoid heart disease, in particular the progressive thickening and retraction of the tricuspid valve leaflets, there was no evidence of a neuroendocrine tumour or carcinoid syndrome to explain this appearance. It is thus recommended that sonographers and clinicians be wary of unusual right heart valvulopathies in methamphetamine-induced cardiomyopathy, and if carcinoid syndrome is excluded, consider an alternative serotonergic process as the culprit.

References

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Remote Monitoring: Is Our Governance Keeping Pace?

Alicia Donovan, Fiona Stanley Hospital, WA

Remote monitoring is growing faster than the systems supporting it.

Remote monitoring has transformed the care of patients with cardiac implantable electronic devices (CIEDs), enabling earlier detection of arrhythmias, heart failure deterioration, lead issues and device-related concerns. Its role in modern CIED care continues to expand.

However, as technology advances, are our governance, workforce and clinical systems keeping pace?

Recent Australian and New Zealand data highlight the scale of this challenge. Between 2019 and 2023, CIED implantations increased by 16.5%, while remote monitoring use increased substantially. Clinics reported that, on average, 30.2% of their workload was dedicated to remote monitoring, with 90.5% identifying Cardiac Physiologists as primarily responsible for monitoring remote alerts. These findings reinforce that remote monitoring is not simply a technology-enabled service - it is a specialist clinical service requiring expert interpretation, clinical judgement and timely decision-making.

Technology has limitations

A common misconception is that remote monitoring provides continuous, real-time surveillance. It does not. CIED remote monitoring remains dependent on patient connectivity, telecommunications infrastructure, manufacturer platforms and local clinical review processes.

This is particularly relevant across Australia and, for example, in Western Australia, where FIFO workers and patients living or working in remote communities may experience unreliable network coverage.

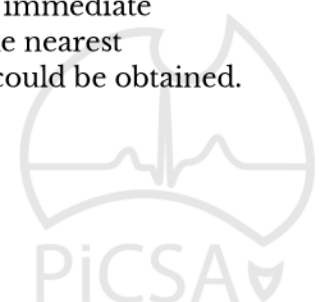
A recent case highlighted this risk. A patient experienced a pre-syncopal episode while working at a remote mining site. They attended the onsite medical centre, were assessed and considered fit to return to work. As an additional precaution, the patient completed a remote transmission, believing that if there were any concerns, the monitoring clinic would contact them.

The transmission subsequently identified rapid atrial fibrillation meeting programmed detection criteria, resulting in ICD therapy. However, because of limited network connectivity at the remote site, the transmission was not received by the monitoring clinic for almost one month.

The patient had done everything correctly. They sought medical assessment, completed the transmission and reasonably believed that no contact from the clinic meant there were no concerns.

This highlights a critical message: remote monitoring is not an emergency service.

Following this event, the patient's risk was formally reviewed, and an individualised action plan was implemented. The patient was specifically instructed that if they experienced another ICD shock, pre-syncope, syncope or other concerning symptoms, they were not to rely on the remote monitoring system or wait for the transmission to be received. They were to seek immediate medical assessment and take their remote monitoring equipment with them to the nearest appropriate regional hospital or healthcare facility where a reliable transmission could be obtained.



When the patient subsequently experienced another event, the action plan worked as intended. The patient presented to the regional hospital with their remote monitoring equipment, a transmission was successfully obtained, and the monitoring team was able to promptly review the event and coordinate appropriate management.

This demonstrates the value of proactive risk assessment: the risk was identified, a specific mitigation strategy was implemented, and when the risk materialised again, the system had a plan in place to manage it.

For patients who live or work in remote locations, individualised action plans should consider connectivity, access to healthcare and the practical limitations of remote monitoring. These plans should be clearly documented, communicated to the patient and reviewed when circumstances change.

Workforce is part of the risk

Every transmission requires review, prioritisation, interpretation, documentation and, where appropriate, clinical action. As device complexity and monitoring volumes increase, so does the cognitive workload placed on Cardiac Physiologists.

Data overload, fatigue and competing clinical priorities are therefore not simply workforce concerns - they are patient safety risks.

Technology does not replace clinical expertise - it increases the need for it.

Remote monitoring workload should be appropriately recognised within position descriptions, workforce models and service planning. Resourcing must reflect patient numbers, transmission and alert volumes, device complexity and the cognitive workload associated with clinical decision-making.

Without adequate resources, Cardiac Physiologists can become vulnerable to system-level failures that are beyond individual control. Formal risk assessments provide an important mechanism to identify these vulnerabilities, establish mitigation strategies and provide evidence for appropriate workforce and resource allocation.

Where to from here?

Artificial intelligence, predictive analytics and wearable technologies will continue to shape the future of cardiovascular care. However, safe remote monitoring will always depend on more than technology alone.

As Cardiac Physiologists, we have an opportunity to lead this evolution by advocating for robust governance, sustainable workforce models, clear escalation pathways and patient-specific action plans.

The future of remote monitoring is not simply about receiving more data. It is about ensuring we have the systems, resources and clinical expertise to safely interpret and act on that information.

Technology will continue to advance. Our governance, workforce planning and patient safety strategies must advance with it.

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Scanning for Dextrocardia

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Dextrocardia often carries a reputation as one of the most intimidating findings in echocardiography. Many challenges come from the classification of a patient with the term “dextrocardia” but what does this term mean to the echocardiographer?

True dextrocardia is a congenital condition in which the heart is positioned on the right side of the chest with a change in the cardiac orientation so that the apex is directed to the patients right (figure 1).

The heart is usually a mirror image of the left sided heart and is accompanied by mirror image arrangement of all thoracic and abdominal organs. True dextrocardia hearts have the lowest incidence of associated congenital heart disease and are more often followed up within adult cardiology services for acquired or adult-onset heart disease.

The term “dextrocardia” has also been applied to the heart that is displaced into the right chest by extracardiac factors such as diaphragmatic hernia or lung hypoplasia. In these cases, the heart axis orientation remains normal with the cardiac apex pointing to the left. These hearts should be more accurately described as having “dextroposition”.

This article will focus on the echocardiography approach to the patient with true dextrocardia and provide a standardised echocardiographic approach to the transducer position and orientation, creating a simplified understanding to these seemingly complex hearts.

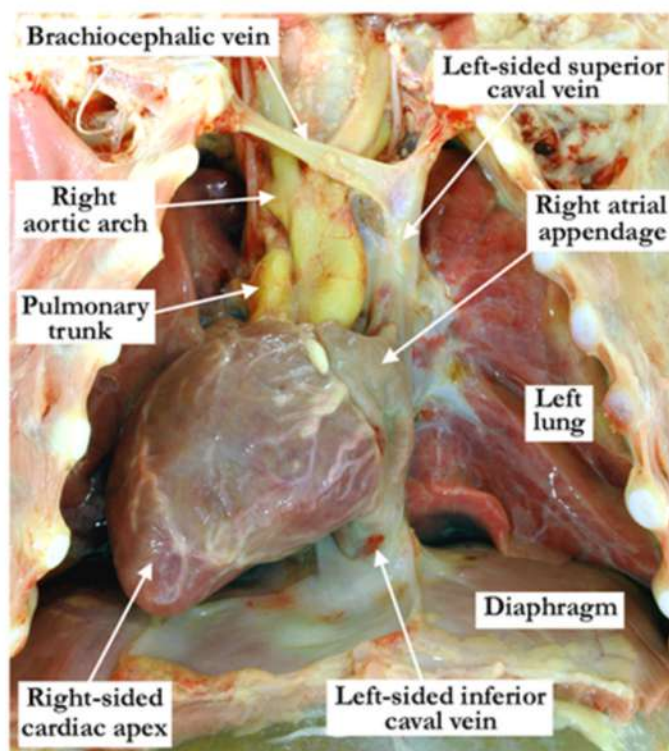


Figure 1: Anatomical Dextrocardia Heart

Scanning Protocol for True Dextrocardia

It is essential to understand and visualise the position and orientation of the heart within the chest when scanning these patients. This helps maintain correct transducer image marker position and appropriate angulation during left ventricle (LV) sweeps to obtain standard echocardiographic projections. For note, the main difference in image acquisition in dextrocardia is orientating the image marker rotated 90° clockwise from the standard parasternal long and short axis positions to align with the long- and short-axis planes of the dextrocardiac heart.



1: Abdominal Situs View

Place the patient in a supine position.

The transducer is placed in the subxiphoid position with the index marker positioned to 3 o'clock.

This image is diagnostic for true dextrocardia with the abdominal aorta and IVC viewed as mirror image to the normal transverse abdominal situs plane (figure 2).

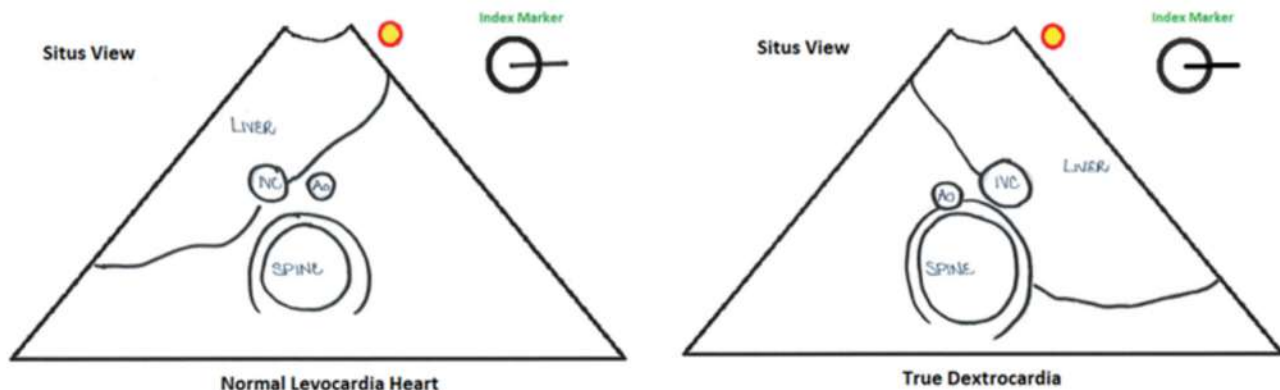


Figure 2: Normal Levocardia Situs Solitus vs Dextrocardia Mirror-Image Situs Inversus echocardiographic image display.

2: Subcostal Four-Chamber (S4C) View

Keep the index marker positioned to 3 o'clock.

Tilt the transducer anteriorly to obtain the subcostal four chamber view.

The cardiac apex will appear pointing to the patient's right side, projected onto the left side of the screen (figure 3). This view is diagnostic of dextrocardia with the orientation of the cardiac apex directed to the patient's right and is the indication to continue with the apical view from the patient's right side.

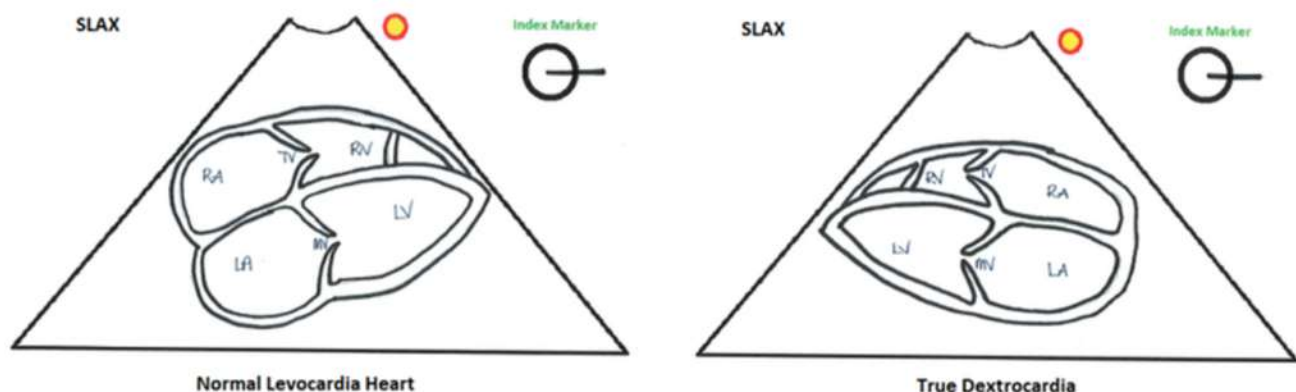


Figure 3: Normal Levocardia Subcostal 4-chamber view vs true Dextrocardia Subcostal 4-chamber echocardiographic image display.

3: Apical Four-Chamber (A4C) View

Place the patient in a partial right lateral decubitus position with the right arm raised.

Position the transducer at the 4th-5th intercostal space. The apical view may need to be acquired from a more medial position than a true left sided apex.

The index marker must be positioned at 3 o'clock as it would in a normal levocardia scan. The left sided structures will now appear on the right side of the screen, and the right sided structures will appear on the left side of the screen (figure 4).

Rotation of the image marker to "correct" the image projection on the screen must **NOT** be performed. The dextrocardiac heart should always be displayed in the anatomically correct position.

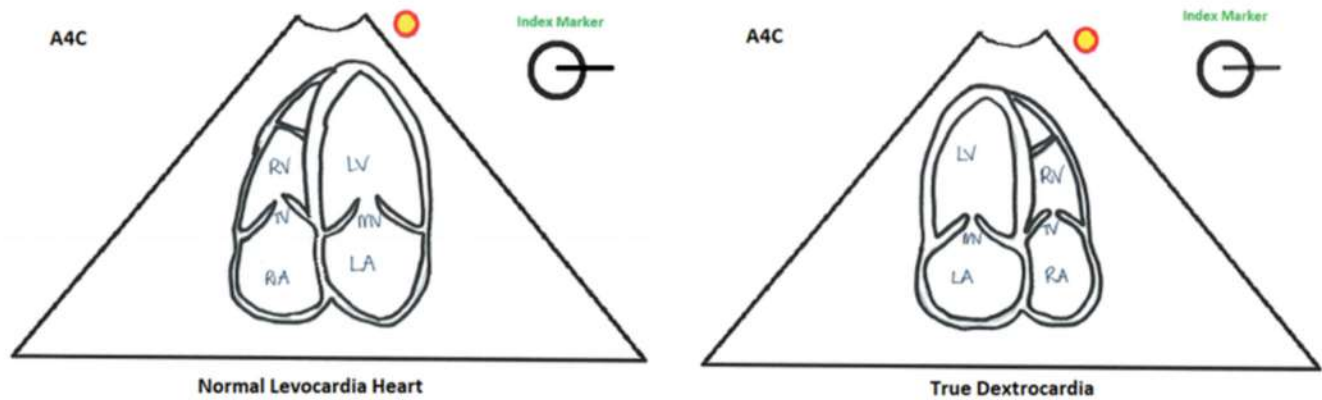


Figure 4: Normal Levocardia Apical 4-chamber view vs true Dextrocardia Apical 4-chamber echocardiographic image display.

4: Apical five chamber (A5C) view

Keep the patient on their right side.

Keep the transducer in the same position on the chest as it was for the A4C.

The index marker should remain at 3 o'clock.

Tilt the transducer anteriorly to bring the aortic valve and aorta into view. The aorta will appear to come off the LV in the opposite direction to a normal levocardia heart (figure 5).

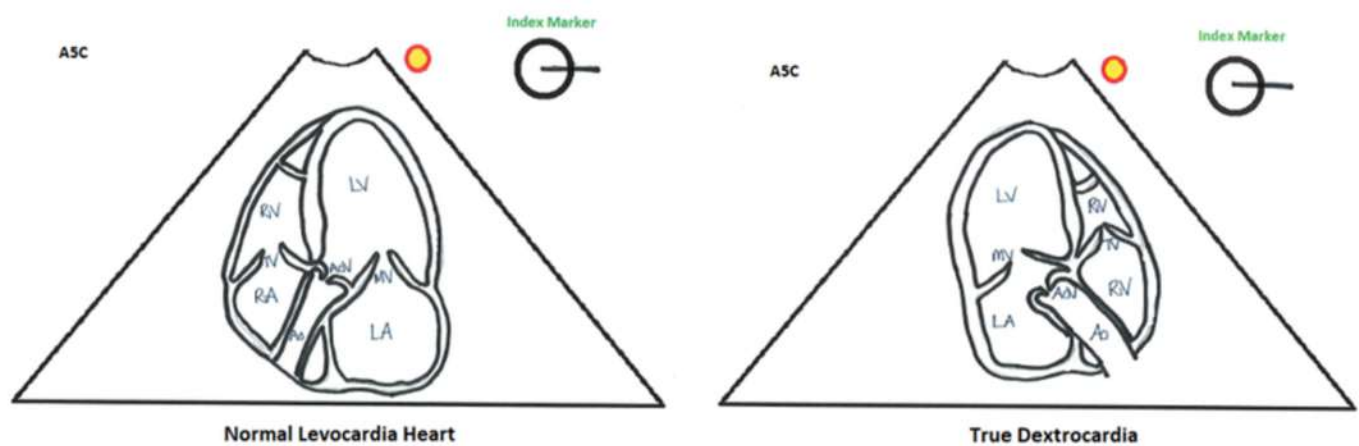


Figure 5: Normal Levocardia Anteriorly directed 4-chamber view vs true Dextrocardia Levocardia Anteriorly directed Apical 4-chamber echocardiographic image display.

5: Parasternal Long Axis (PLAX) View

Keep the patient on their right side as per the apical imaging.

Place the transducer on the upper to mid right sternal border at the 3rd or 4th intercostal space.

To correctly orientate the transducer with the long axis of the LV in a dextrocardia heart, the index image marker is rotated to ~ 1 o'clock so that it is directed towards the left clavicle. The ultrasound beam is now orientated to the long axis of the LV, with the scan plane running parallel to an imaginary line joining the left shoulder and the right hip. Following this convention, the index marker is now 90° clockwise to your standard PLAX position and the image will display in a similar view of a normal levocardia heart PLAX echocardiography image (figure 6). Parasternal RV inflow and RV outflow views are performed as per normal.



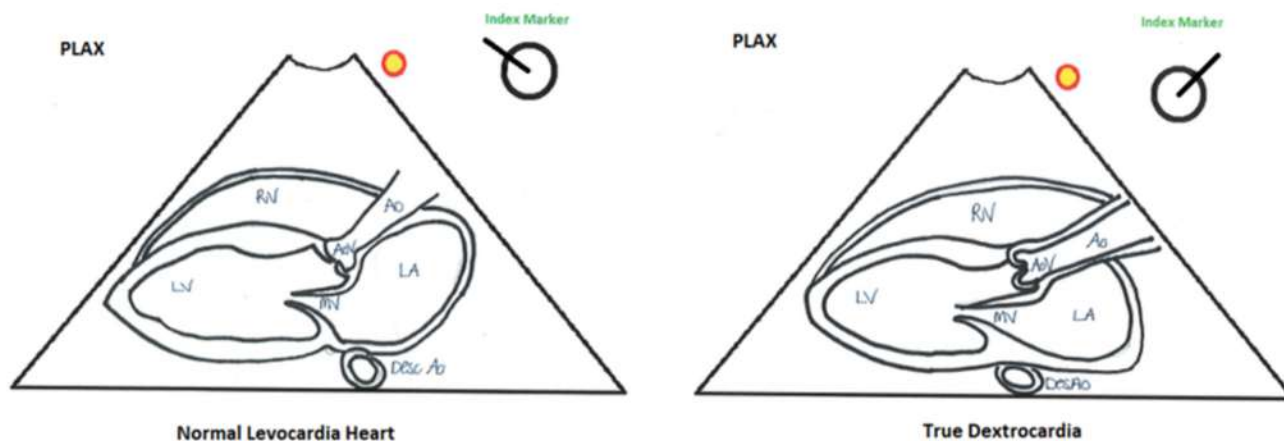


Figure 6: Normal Levocardia Parasternal Long Axis View vs true Dextrocardia Parasternal Long Axis View echocardiographic image display.

6: Parasternal Short Axis (PSAX) View

Maintain the same transducer position on the chest as the PLAX view.

Rotate the transducer 90° in a clockwise direction from the PLAX position.

The index marker should be positioned ~4-5 o'clock.

Short axis sweeps of the dextrocardia heart should be performed with the transducer tilted from the cardiac base to the apex so that the ultrasound beam is directed in the same orientation as the heart, towards the right hip.

Angling to the level of the aortic valve first the structures on display will show the TV on the left and the PV on the right (figure 7).

Angling to the level of the papillary muscle the RV will appear on the left of the screen (figure 8). Perform all sweeps, Doppler and CFI as per normal scanning protocol.

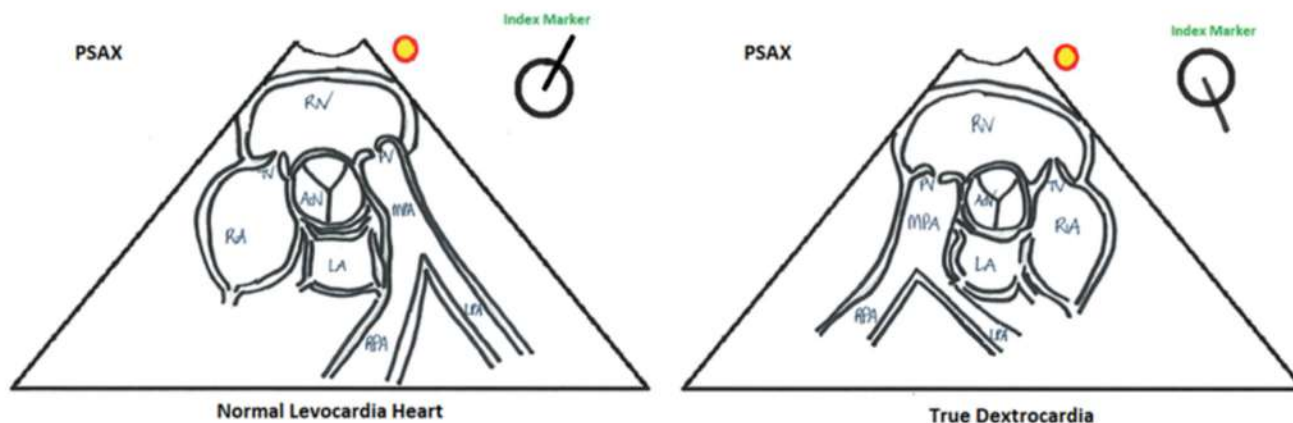


Figure 7: Normal Levocardia Parasternal Short Axis View at level of Aortic Valve vs true Dextrocardia Parasternal Short Axis View at level of Aortic Valve echocardiographic image display.

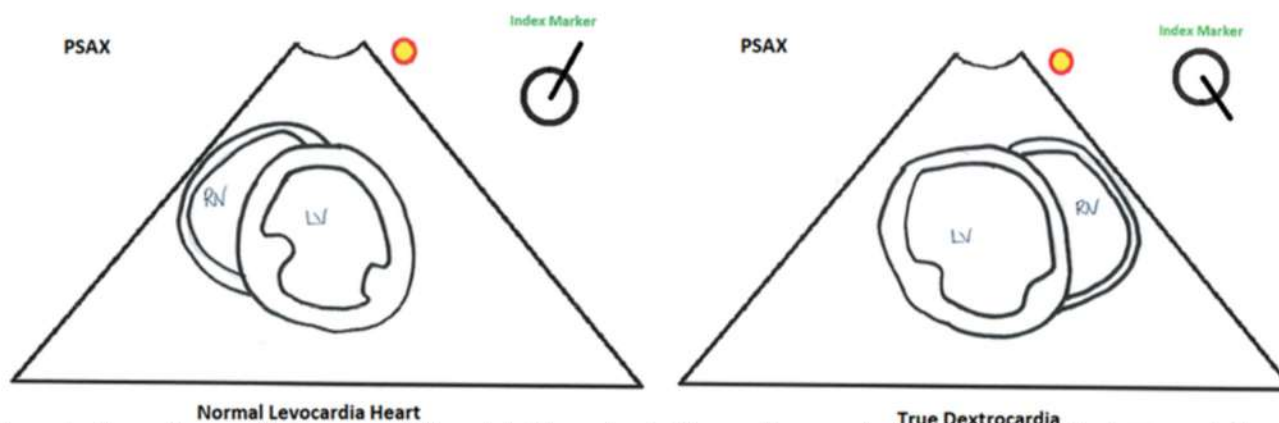


Figure 8: Normal Levocardia Parasternal Short Axis View at level of the papillary muscles vs true Dextrocardia Parasternal Short Axis View at level of the papillary muscles echocardiographic image display.

7: Suprasternal Notch Long Axis View

Position the patient in the supine position and arch the neck.

Place the transducer in the suprasternal notch.

The index marker should be placed at ~12 o'clock.

Perform all sweeps, Doppler and CFI as per normal scanning protocol.

The image will appear as per the normal standard normal levocardia heart.

8: Suprasternal Notch Short Axis View

Position the patient in the supine position and arch the neck.

Place the transducer in the suprasternal notch.

The index marker should be placed at ~3 o'clock.

Perform all sweeps, Doppler and CFI as per normal scanning protocol.

Ultimately, confidence in scanning dextrocardia comes from consistency. By maintaining a structured, segmental approach and adapting probe orientation appropriately, what initially feels complex becomes both logical and manageable.

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Interrupted Aortic Arch: A rare but critical congenital heart defect

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Case Study

- 1 day old newborn referred for a murmur
- Found to have absent femoral pulses
- Transthoracic echo showed:
 - An interrupted aortic arch
 - The interruption occurred distal to the left subclavian artery (Type A).
 - Moderate size patent ductus arteriosus with bidirectional blood flow (supplying the descending aorta)

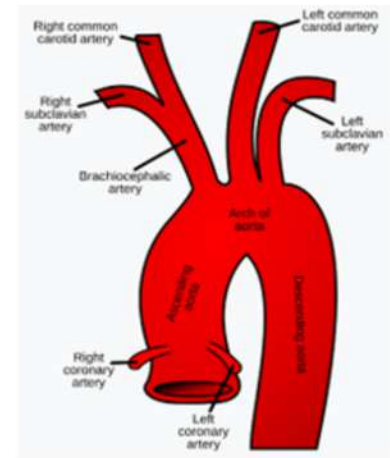


Figure 1: Normal aortic arch anatomy.

Echo Findings

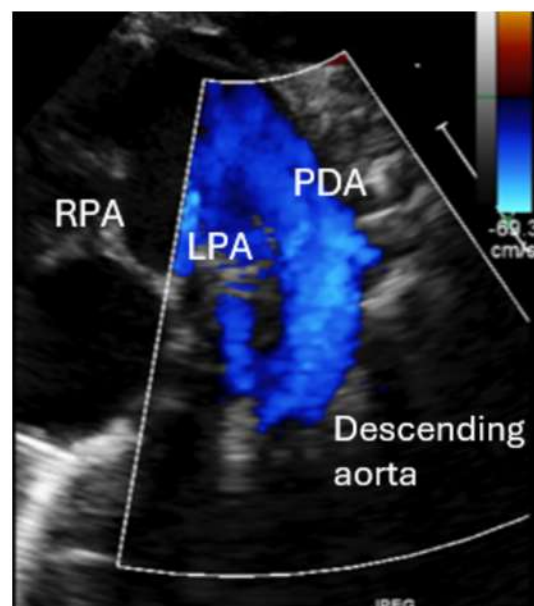
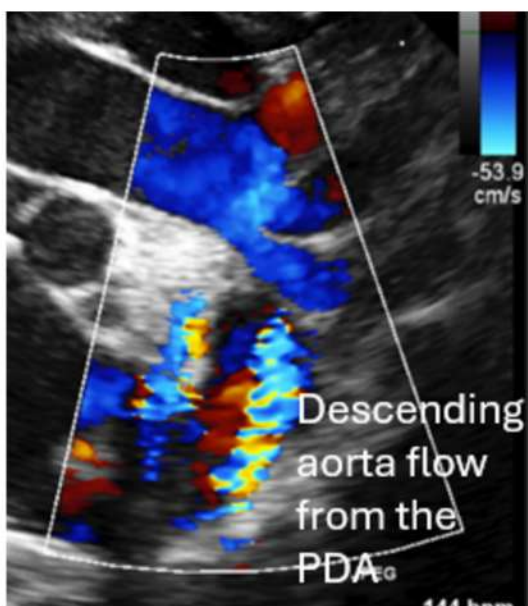
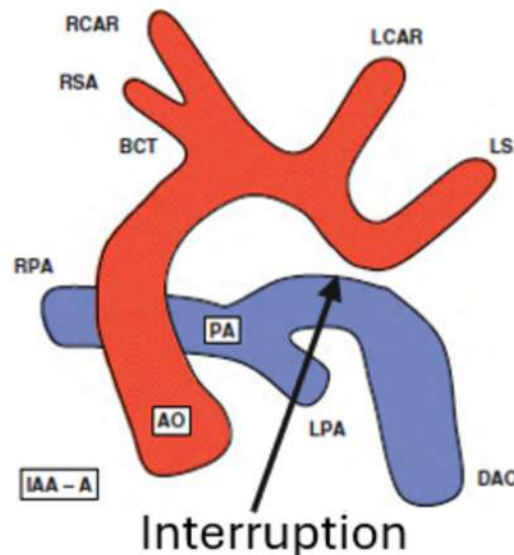
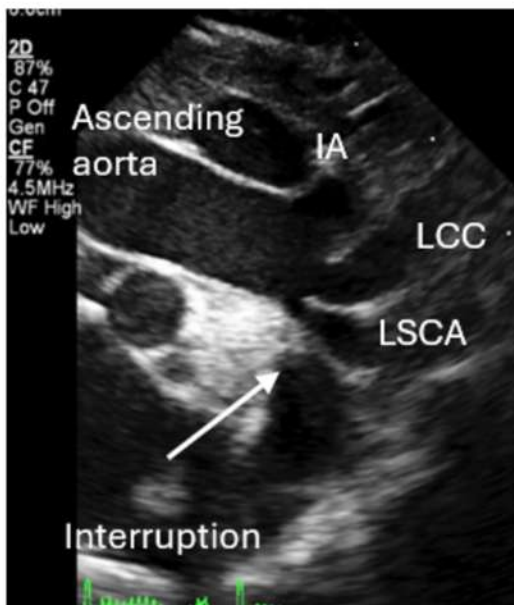


Figure 2: Echo findings. IA- Innominate artery (Brachiocephalic), LCC-Left common carotid, LSCA- Left subclavian artery, DA- Descending aorta, LPA/RPA- left/right pulmonary artery, PDA- patent ductus arteriosus

Definition Interrupted aortic arch (IAA)

- An anatomical disruption between the ascending aorta and descending aorta.
- Three main types; classified according to the site of interruption
- Patient is dependent on PDA flow to the descending aorta
- Affects approximately 1.3% of CHD patients.
- Association with DiGeorge syndrome

Typical presentation and symptoms

- Lethargy
- Poor feeding
- Weak or absent femoral pulse
- Cyanosis
- Usually presents within days of life

Management

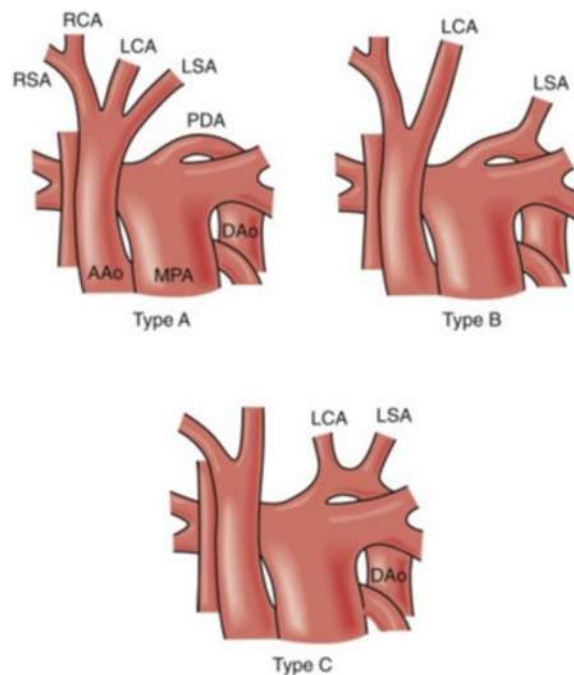
- Urgent medical intervention using Prostaglandin to keep the PDA open
- Surgical repair

Three Classifications

Type A: Interruption occurs distal to the left subclavian artery (up to 40% of cases).

Type B: Interruption occurs between the left common carotid artery and the left subclavian artery (most common, ~50–70% of cases). It is strongly associated with DiGeorge syndrome.

Type C: Interruption occurs between the right and left common carotid arteries (~ 1% of cases)



Concealed Conduction

By Harley Cross (BAppSc (HMS), Grad Dip Cardiac EP, CEPIA)

Mechanism and Clinical Recognition

Concealed conduction refers to depolarisation of cardiac tissue by an impulse that produces no recognisable deflection on the surface ECG but still alters the refractory state of that tissue and therefore the conduction of the impulses that follow. It is not an arrhythmia in its own right and requires no specific treatment. It is, however, the explanation for a substantial proportion of ECG findings that otherwise appear inconsistent: unexpected atrioventricular block, bundle branch block that persists well beyond the point at which a purely rate-related mechanism should have resolved, alternation between two different QRS morphologies despite an identical coupling interval, and variable atrioventricular relationships during PVCs and ventricular tachycardia.

This review sets out the physiological basis of concealed conduction and then applies it, step by step, to four ECG examples in this article

1. A sinus rhythm with ventricular bigeminy causing post-extrasystolic PR prolongation.
2. A sinus rhythm with atrial bigeminy and alternating bundle branch block.
3. An SVT with aberrancy that persists longer than expected.
4. A wide complex tachycardia demonstrating fusion at onset and concealed conduction affecting the return PR interval after termination.

The term concealed conduction is attributed to Karel Frederik Wenckebach, who described the general principle that an impulse could partially penetrate cardiac tissue and alter its subsequent behaviour without itself producing a visible deflection, during his work in Theodor Engelmann's laboratory around the turn of the twentieth century.

The concept was formalised in its modern form by Richard Langendorf and Alfred Pick, whose 1956 paper in *Circulation* established the principal framework still in use.¹ Working from surface ECGs alone, they showed that atrioventricular conduction disturbances that otherwise appeared anomalous were consistent once an unseen, incompletely conducted impulse was assumed to have entered and partially discharged the AV node. Langendorf, Pick and Katz subsequently demonstrated that the ventricular response during atrial fibrillation is substantially governed by concealed conduction within the AV junction, rather than by simple filtering of atrial impulses at a fixed ratio.² Lehmann and colleagues later used His bundle recordings to localise where within the node this concealed penetration occurs and to characterise its effect on subsequent conduction.³

Physiological Basics

An impulse that enters excitable tissue, partially or fully depolarises it, and then fails to produce a recognisable QRS or P wave nonetheless resets that tissue's refractory period from the time of the unseen depolarisation, not from the time of the last beat visible on the ECG. This has two consequences that account for most of the clinical findings attributed to concealed conduction.

First, it can prolong refractoriness and cause unexpected block. A sinus P wave that would ordinarily conduct normally instead encounters an AV node still recovering from an earlier, invisible incursion, typically a blocked premature atrial or ventricular complex and is delayed or blocked outright. This underlies pseudo-Mobitz patterns, unexpectedly long PR intervals following a PVC, and P waves that appear to be dropped for no evident reason.

Second, it can perpetuate functional block by repeatedly re-refracting tissue before it has the opportunity to recover. This is the mechanism relevant to two of the four cases below. The specific variant responsible for bundle branch aberrancy is concealed transseptal conduction: an impulse conducting down one bundle branch also crosses the septum and activates the other bundle branch retrogradely. This retrograde activation produces no separate deflection on the surface ECG, but it repeatedly discharges the blocked bundle just before the next antegrade impulse arrives, so that bundle does not get the opportunity to recover. As a result, aberrant QRS morphology can persist well beyond the point at which a purely rate-related cause (Ashman phenomenon and its phase 3 and phase 4 variants) should have resolved.^{4,5} A 2021 case-based review in *Circulation* sets out tachycardia-dependent block, deceleration-dependent block and concealed transseptal conduction as the three principal mechanisms of aberrant conduction, noting that concealed transseptal conduction is the most likely of the three to produce sustained, rather than transient, aberrancy.⁵

Case 1: Sinus Rhythm with Ventricular Bigeminy



Figure 1: Limb lead ECG showing sinus rhythm with ventricular bigeminy. The PR interval of the sinus beat following each PVC is longer than the PR interval of the sinus beat preceding it.

Of the four examples in this review, this is the most direct demonstration of concealed conduction, because it requires comparing only two PR intervals either side of a single ectopic beat rather than tracking a mechanism across several cycles. Each PVC in this bigeminal rhythm is followed by a sinus beat whose PR interval is measurably longer than the PR interval of the sinus beat that preceded the PVC.

1. Each ventricular ectopic beat generates a wavefront that, in addition to activating the ventricles, conducts retrogradely toward the AV node and His bundle.
2. This retrograde wavefront frequently does not proceed all the way to the atrium as a visible retrograde P wave, but it still partially depolarises the AV node and para-Hisian tissue. This is concealed conduction in its most literal form: the event responsible for the downstream finding produces no deflection of its own.
3. This partial, invisible depolarisation leaves the AV node relatively refractory at the moment the next sinus P wave arrives. The sinus impulse still conducts, but more slowly than it would have in the absence of the preceding concealed incursion.

4. The result is directly measurable on the surface ECG: the PR interval of the sinus beat immediately following the PVC is longer than the PR interval of the sinus beat immediately preceding it, despite both being ordinary conducted sinus beats with no structural difference in the AV node between them. Once the concealed effect of the PVC has dissipated, the following PR interval returns to baseline — until the next PVC in the bigeminal pattern repeats the process.

This is essentially the same phenomenon Langendorf and Pick described in their original account of concealed conduction, and the mechanism localised by Lehmann and colleagues to a specific level of retrograde penetration within the AV node.^{1,8} It provides a useful bedside checkpoint: when a PVC bigeminal rhythm is present, comparing the pre- and post-extrasystolic PR intervals will usually confirm that some degree of concealed retrograde conduction has occurred, even without direct evidence of a retrograde P wave. This phenomenon more commonly occurs when the PVCs are interpolated.

Case 2: Sinus Rhythm with Atrial Bigeminy and Alternating Bundle Branch Block

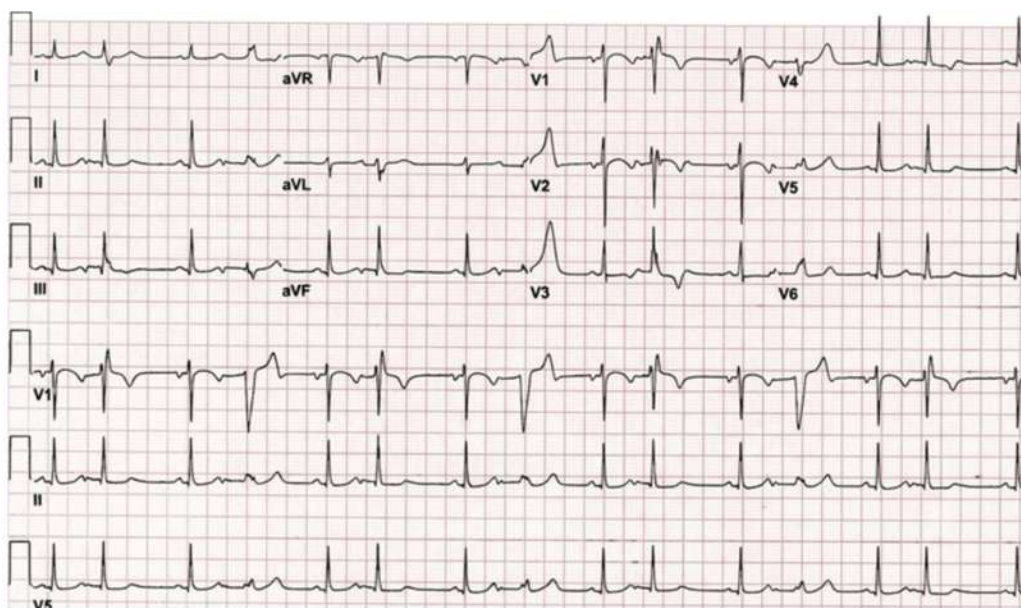


Figure 2: Twelve-lead ECG showing sinus rhythm with atrial bigeminy and beat-to-beat alternation between right and left bundle branch block morphology.

This tracing was taken from Saini and colleagues 2018 Circulation ECG challenge.⁶ Every second beat is a premature atrial complex, confirmed by an early, differently shaped P wave, and each premature beat has the same coupling interval to the preceding sinus beat. Despite this, consecutive premature beats alternate between a right bundle branch block pattern and a left bundle branch block pattern. The explanation comes down to timing: what each premature beat's retrograde concealed conduction does to the bundle branch it did not use.

1. The first premature beat conducts antegradely down the left bundle, producing a right bundle branch block pattern QRS (the right bundle is the one that blocks). The wavefront then crosses the septum and activates the right bundle retrogradely. This retrograde activation produces no separate deflection on the ECG, but it resets the right bundle's refractory period to a later point than antegrade activation alone would have produced.
2. The next beat is a normally conducted sinus beat, activating both bundles antegradely without aberrancy. Because the left bundle was activated early (by the preceding premature beat) and the right bundle was activated late (by concealed retrograde conduction), this sinus beat represents a longer preceding cycle for the left bundle than for the right bundle. In other words the left bundle 'thinks' it has a much longer time to recover before the next impulse arrives.

- When the next premature atrial complex arrives at the same coupling interval as the first, the left bundle is now the one facing a relatively short cycle after a longer preceding cycle, the same long-short relationship that underlies the Ashman phenomenon. The impulse therefore blocks in the left bundle and conducts down the right bundle instead, producing a left bundle branch block pattern. This beat then conceals retrogradely into the left bundle at a longer coupling interval, and the asymmetry reverses in preparation for the next premature beat that results in right bundle branch block.

Neither bundle branch is structurally diseased in this mechanism; both are capable of normal conduction. The alternation reflects a self-perpetuating asymmetry in refractory timing between the two bundles, driven entirely by concealed retrograde conduction that leaves no direct trace on the ECG. Recognising this avoids an incorrect diagnosis of fixed bilateral bundle branch disease. Figure 3 below shows the bundle branches alternating refractory periods based on the preceding concealed conduction.

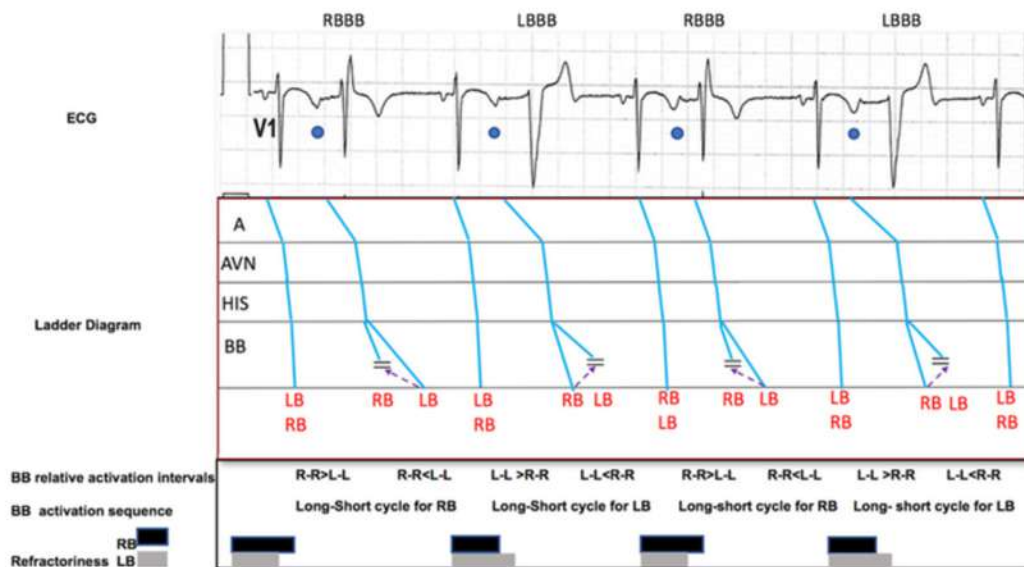


Figure 3: Ladder diagram with associated refractory period duration of each bundle branch following concealed conduction. Note: the diagram can be misleading, as it suggests that the conducted bundle is activated later than the blocked bundle. This is not the case — the bundle with the intact conduction is activated first, which gives rise to a longer coupling interval to the next sinus beat with normal conduction. This in turn sets up the next anticipated longer refractory period on the following PAC.

Case 3: SVT with Persistent Aberrancy

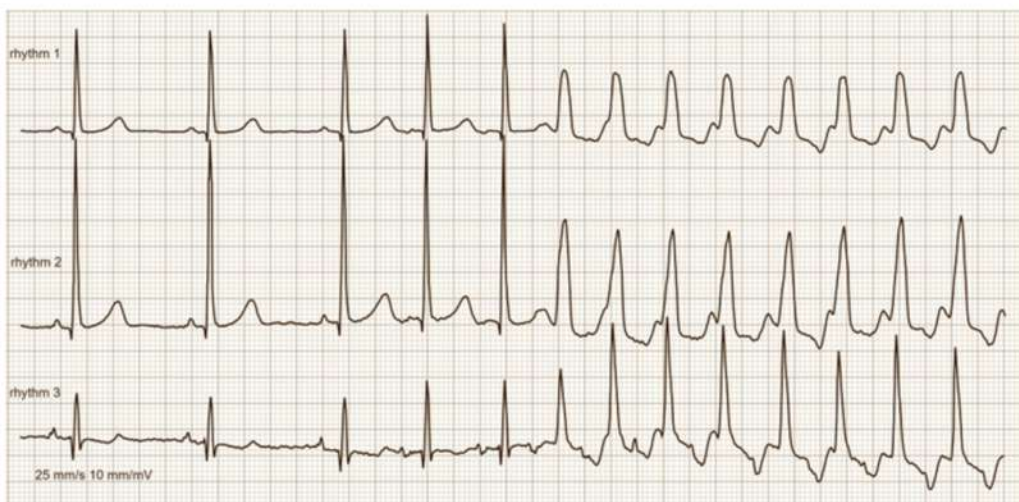


Figure 4: Three rhythm strips demonstrating abrupt onset of a supraventricular tachycardia with aberrant conduction persisting from the first beat.

In the above example, each strip shows the same pattern: several narrow-complex beats followed by an abrupt increase in rate accompanied by a wide QRS morphology that then persists for the remainder of the visible tachycardia.

The first question to ask is whether this is SVT with aberrancy or VT. In this example the mechanism is clearly atrial tachycardia, as an ectopic P wave is visible before each QRS.

The next question to ask is why the aberrancy continues beyond the first few beats.

Ordinarily the bundle branches adapt quickly to the new heart rate and the aberrancy resolves. The same concealed transseptal mechanism described in Case 2 explains why the aberrancy does not resolve after the first beat or two, despite the tachycardia continuing at a stable cycle length.

1. At tachycardia onset, the abrupt shortening of cycle length finds one bundle branch — typically the one with the longer baseline refractory period — still refractory from the preceding cycle. This first aberrant beat reflects ordinary phase 3, tachycardia-dependent block and is expected whenever cycle length shortens abruptly.
2. Because the impulse conducts down the unaffected bundle first, it also activates the blocked bundle retrogradely by crossing the septum. This retrograde activation is invisible on the ECG, but it restarts the blocked bundle's refractory period from a point later in the cycle than direct antegrade activation would have produced.
3. When the next tachycardia impulse arrives, the previously blocked bundle — having been reset late by concealed retrograde conduction rather than on schedule by antegrade activation — is still refractory. The impulse again conducts preferentially down the other bundle, and the same retrograde concealment recurs. This repeats on every cycle for as long as the tachycardia continues, which is why the aberrancy outlasts the initial rate change rather than resolving after a few beats. Where this case differs from the above is that the cycle length remains stable; it is not manipulated by the intervening sinus beats that cause the alterations in refractory periods between the bundle branches. In this example the same bundle is repeatedly activated later in the tachycardia cycle, so it never has time to fully recover.
4. The pattern is broken only by an event that alters the timing relationship between the two bundles, most reliably a premature ventricular contraction delivered during the tachycardia, which can activate the blocked bundle directly rather than via the delayed transseptal route, allowing it to recover in time for the next impulse and restoring a narrow QRS without terminating the tachycardia. This manoeuvre, sometimes described as peeling back refractoriness, is used diagnostically in the electrophysiology laboratory to confirm that persistent aberrancy reflects concealed conduction rather than fixed bundle branch disease.⁷

The practical point is that a change in QRS morphology during SVT reflects the refractory state of the His-Purkinje system and does not by itself indicate a change in the underlying arrhythmia mechanism.

Case 4: Confirming VT Using Concealed Conduction and Fusion

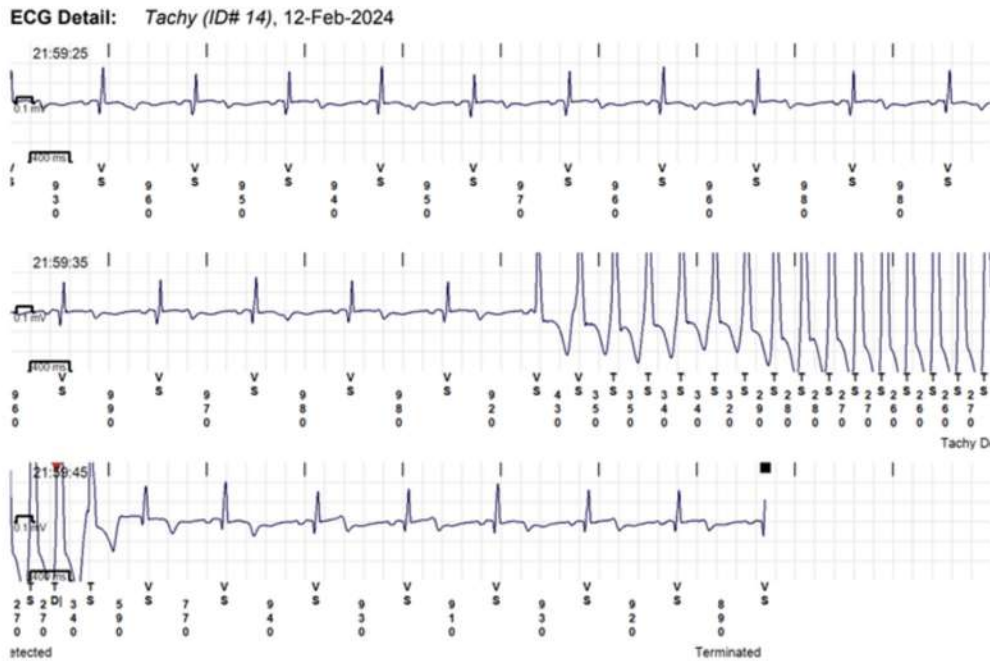


Figure 5: Onset of wide complex tachycardia in a patient with an Implantable Loop Recorder (ILR). Evidence of fusion at the onset and concealed conduction at termination.

This intracardiac tracing shows an ILR-detected episode of a regular wide complex tachycardia, with beats of intermediate QRS morphology consistent with fusion visible early in the episode. Fusion beats occur when a sinus impulse and the tachycardia wavefront both contribute to ventricular activation, and their presence is a reliable indicator of atrioventricular dissociation and therefore of a ventricular origin for the tachycardia.^{8,9} Another clue to diagnosing VT in this example lies in its termination and the effect on the first returning sinus beat.

1. During VT, each ventricular impulse has the opportunity to conduct retrogradely toward the atrium through the His-Purkinje system and AV node. This retrograde conduction is frequently incomplete: the impulse penetrates part of the way into the AV node or His bundle, partially discharges it, and fails to reach the atrium. This is concealed retrograde conduction, and it explains why 1:1 ventriculoatrial conduction is uncommon during VT even when no retrograde P wave is visible on the surface trace.⁷
2. A sinus impulse arriving at the AV node while it remains partially refractory from this concealed retrograde activity is delayed or blocked outright and cannot proceed to activate the ventricle. This is why sinus P waves marching through a VT frequently fail to conduct even when their timing, considered in isolation, appears favourable: the AV node's refractory state at that instant is determined by the preceding concealed ventricular-to-atrial conduction, not by the visible timing of the P wave alone.
3. Following termination of VT, there is a return of sinus rhythm. Attention should be drawn to the first PR interval upon return of sinus rhythm: it is considerably longer than the PR intervals both before the VT and immediately following this beat. While no clear P wave can be seen, its presence can be inferred from the narrow QRS complex — if the PR interval were the same as the surrounding beats, the P wave would be expected to be visible, so it must instead be hidden within the terminal T wave of the preceding VT complex. This long PR interval is generated via a mechanism similar to that described in Case 1. There was concealed penetration of the VT into the His–AV node, and once VT terminates and sinus rhythm resumes, the first conducted beat encounters partially refractory tissue and propagates more slowly than expected.

Take Home Message

- Concealed conduction can manifest in many different ways. An unexpectedly blocked or delayed P wave does not necessarily indicate AV nodal disease. Before proceeding to further investigation, look for a preceding PVC, blocked PAC, or other unseen impulse that could have left the AV node refractory. Concealed conduction is a recognised cause of transient, self-limiting pseudo-AV block.^{1,10}
- Bundle branch block that persists throughout a tachycardia does not necessarily indicate fixed conduction disease. If aberrancy outlasts the first few beats of a stable tachycardia, concealed transseptal conduction should be considered, and a well-timed premature ventricular contraction delivered in the EP laboratory can be diagnostic.
- Fusion and capture beats during a wide complex tachycardia remain reliable markers of a ventricular origin. Concealed retrograde conduction into the AV node is the reason they appear intermittently rather than at every opportunity, and their patchy occurrence should not be taken as evidence against the diagnosis.
- Ventricular rate control during atrial fibrillation is influenced substantially by concealed conduction within the AV junction, not solely by the number of atrial impulses reaching the node. This contributes to the variability in ventricular response seen both between patients and within the same patient over time.²

Concealed conduction is a reminder that the surface ECG is a partial record of conduction system activity, not a complete one. A substantial amount of activity within the AV node, bundle branches and accessory pathways produces no P wave, QRS complex, or intracardiac deflection, yet still determines the pattern observed on the tracing. Langendorf and Pick established this from surface ECG analysis alone, well before invasive electrophysiology was available; the cases above show that the same reasoning, applied systematically and beat by beat, continues to explain findings that would otherwise appear inconsistent.

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What Do You Think?

Associate Professor Harry Mond, CardioScan Medical Director



ECG interpretation

In 1970, I commenced work as a “novice” cardiologist at The Royal Melbourne Hospital. Training at that time was learning how to recognise the diastolic murmur of mitral stenosis and making sure the ECG and chest x-rays were the correct way up. As this was prior to coronary artery surgery, stenting and echocardiography, the ECG was a highly respected tool for the diagnosis of many conditions. I was fortunate to befriend a number of the gurus of ECG interpretation who taught me to look into the soul of an ECG. Today, even though the ECG is the first investigation performed by a cardiologist, the art of ECG interpretation has been lost because we depend on the new tools for cardiac diagnosis.

In 1972, I helped arrange a 2-day workshop at the hospital with the two top ECG educators at the time; Leo Schamroth and Barny Marriott. Leo showed an ECG which he had published a few years earlier on a previously unknown disorder. Here is the ECG and this was the quality of almost all ECGs at that time.



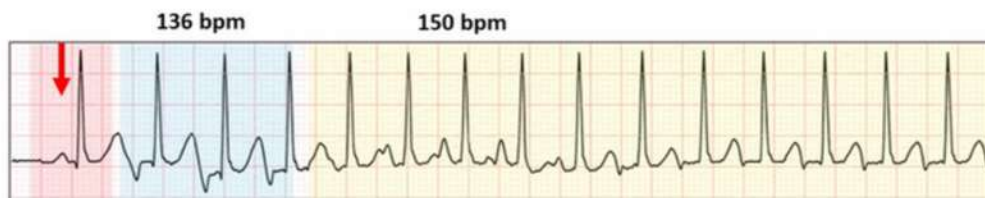
What do you think? Answer at end. Write to me if you got it right.

How does one “attack” an ECG to release the hidden gems in its soul?

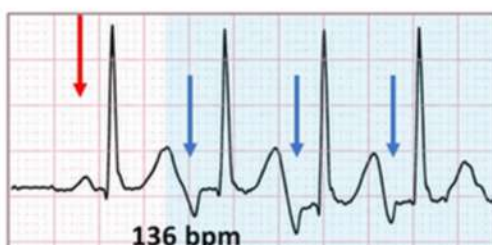
Let us analyse what appears to be a simple supraventricular tachyarrhythmia.



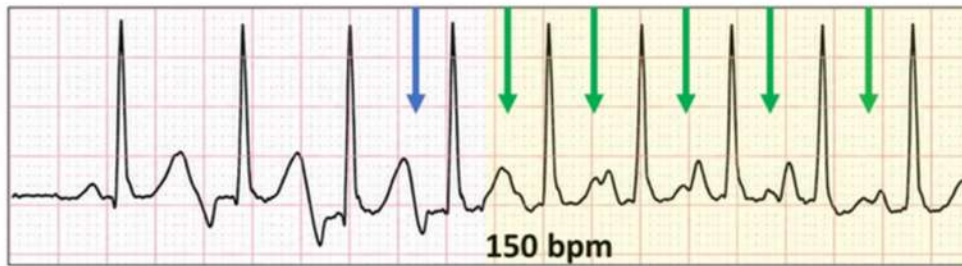
The first complex is sinus generated (red arrow and highlight). The tracing then deteriorates into a narrow complex tachycardia. Most physicians would call this a supraventricular tachycardia and leave it to the EP study to make a diagnosis. We need to look further into its soul.



There are two different tachycardia rates with different P wave configurations. Let us review the first one (blue highlight).

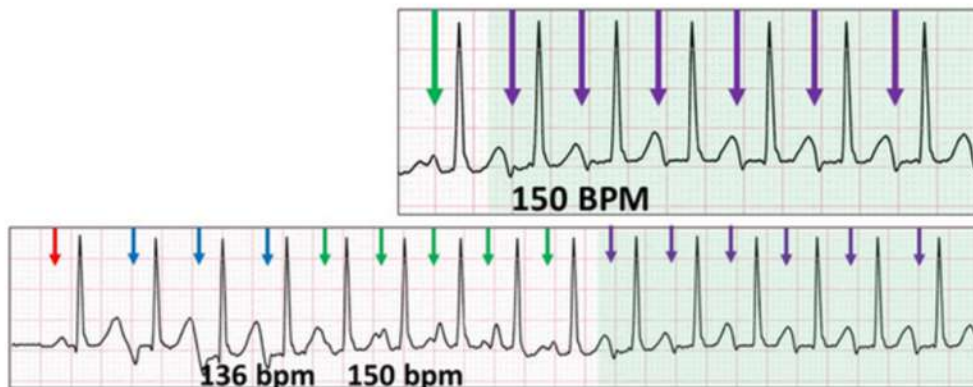


Inverted P waves (blue arrows). This is most likely a junctional tachycardia. What about the second (yellow highlight)?



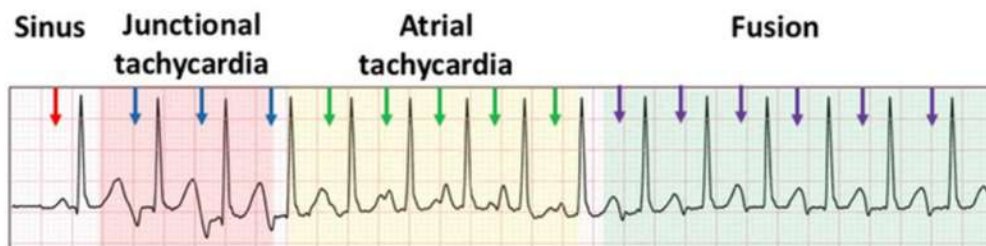
Transition to a tachycardia with P waves similar to the sinus P waves (green arrows). At 150 bpm, this is most likely an atrial tachycardia, but atrial flutter should be considered.

We have two supraventricular tachyarrhythmias competing with each other for ventricular depolarization. What happens when the rates approximate each other?



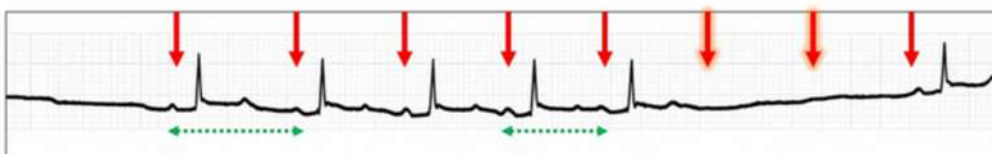
Once again, the P waves are inverted but smaller than earlier (green highlight). Both supraventricular ectopic foci depolarize the atria as fusion beats although probably only the junctional focus depolarizes the ventricle.

In summary:



This ECG highlights a poorly recognised and understood feature of junctional rhythm. If with junctional tachycardia, there is no retrograde conduction, then the atrium continues to depolarize separately. This can be sinus rhythm or an atrial tachyarrhythmia and even atrial fibrillation. With retrograde conduction, there is now competition between the supra-nodal and junctional foci as seen with our case study. If you got it correct, please write to me.

What about the Schamroth ECG? Firstly, wave the magic wand of AI.



This is the first description of sino-atrial Wenckebach block. Note the two dropped P waves (ghosted red arrows).

Harry Mond

PiCSA Member Spotlight

Jordan discovered Cardiac Physiology during a university placement at Monash Heart while studying Human Physiology and Chemistry. Fascinated by the profession, Jordan has continued to develop expertise across interventional cardiology and cardiac devices, with a particular interest in intravascular imaging, rhythm management and emerging technologies that can improve patient care. Plus, Jordan loves getting to play with the latest “toys”!



What made you decide to become a physiologist, specifically within the field of cardiology?

I studied human physiology and chemistry, which were two areas I was really passionate about. During my studies, I was given the opportunity to complete a placement at Monash Heart, and that was where I first discovered cardiac physiology. I found the role fascinating and immediately wanted to learn more about the profession and the different areas we could work in. Unfortunately, I still feel that cardiac physiology doesn't always receive the recognition it deserves! If it wasn't for that placement, I genuinely don't know what I would be doing today.

What do you specialise in?

I've completed a lot of training in intravascular imaging within the interventional cardiology space, which has been a major area of interest for me. I've also completed my IBHRE certification in cardiac devices. Cardiac rhythm management is something I find particularly interesting, and I'm currently halfway through my CEPIA postgraduate studies in electrophysiology. I really enjoy continuing to develop my skills across different areas of cardiac physiology and seeing how they all connect within patient care.

What are you passionate about?

One thing I'm particularly passionate about is the development of new technologies and learning how they can improve patient care. Cardiology is constantly evolving, and I really enjoy understanding how new equipment, techniques and technologies work, as well as learning how to use them in clinical practice. I'm always interested in what's coming next and how it might change the way we work. Plus, I love new toys!!!!

What do you do for fun/hobbies?

Outside of work, I enjoy spending time with my big little puppy, Winston, who is a Great Dane. He is the sweetest and most loyal boy, and I absolutely adore him. I also occasionally partake in a run... Although “occasionally” seems to be becoming more frequent! My next big goal is to complete a full marathon next year.

What do you find most valuable about PiCSA?

Having a professional body that supports cardiac physiologists, advocates for the profession and provides strong representation is incredibly important. I particularly value PiCSA's education and professional development opportunities, as well as its role in giving our profession a stronger, unified voice.



Advertising Your Job Vacancies with PiCSA

Did you know that PiCSA members can advertise job positions completely free of charge?

Simply send us your vacancy details, and we'll share the listing across all our key channels. Your role will be featured on the PiCSA website, included in our monthly Member Update email, and promoted through our social media platforms- Facebook, LinkedIn, X, and Instagram. It's an easy and effective way to reach qualified cardiac physiology professionals across Australia and across our wider professional community..

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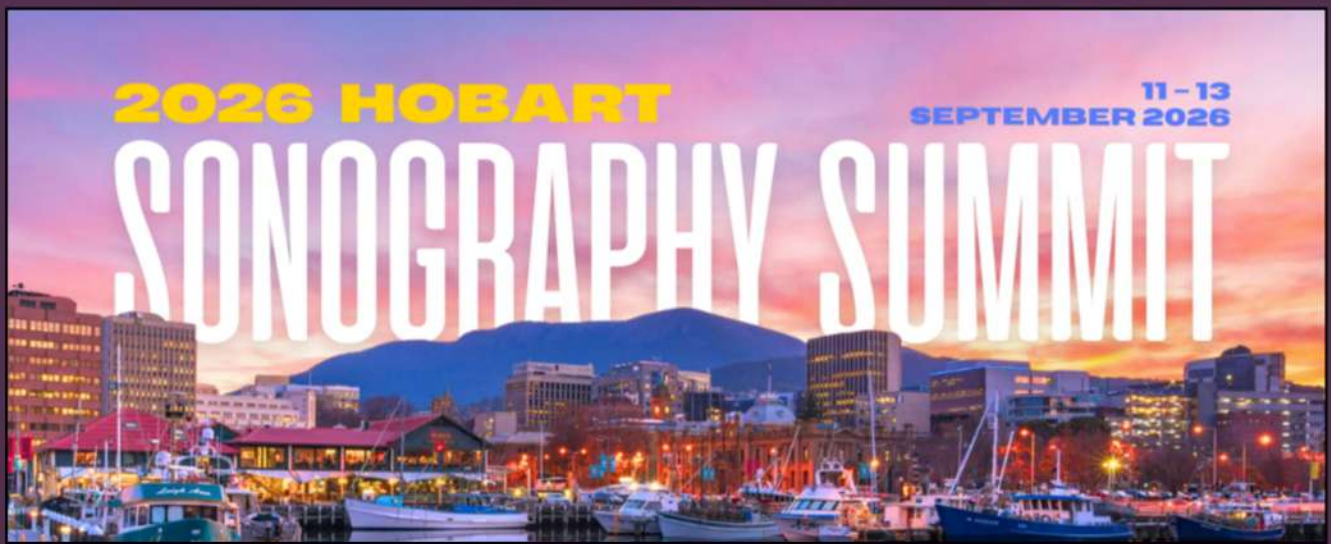
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2026 Practical ECHO Skills for CHD Patients

10.5 hours

12th September 2026



Newsletter Article Submission deadlines

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



Spring 2026	Sunday 25th October 2026
Summer 2027	Sunday 24th January 2027
Autumn 2027	Sunday 25th April 2027
Winter 2027	Sunday 25th July 2027

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



Boosting Visibility: Small Actions with Big Impact for all Cardiac Physiologists

One simple yet impactful way to boost the visibility, recognition, and regulation of the Cardiac Physiology profession is to proudly introduce ourselves using the recommended title: “**Cardiac Physiologist**.” Regardless of whether your employer has updated your award or statement of duties, this small action can have a big impact.

When we introduce ourselves correctly, we not only educate those around us about our professional identity but also build trust and understanding with our patients. It’s a simple phrase—“**Hello, my name is ..., and I am a Cardiac Physiologist**”—but it carries immense power to advance our profession.

Looking for other ways to increase visibility? Consider these ideas:

- **Display your title:** Add “Cardiac Physiologist” to your name badge, your email signature, and your business cards.
- **Be a role model:** Encourage colleagues to use the correct title, showing solidarity and professionalism. It’s ok to be more specific (e.g., “Cath Lab Physiologist”)
- **Engage on social media:** Post about your work using #CardiacPhysiologist to connect with peers and raise awareness. You can also use **both** #EchoPhysiologist and #CardiacSonographer if applicable.
- **Share your expertise:** Offer to speak at community events, webinars, or conferences to highlight our role in healthcare.
- **Collaborate across teams:** Use multidisciplinary meetings as an opportunity to clarify and promote the Cardiac Physiologist title.

*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)

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Echo Guru

<https://echoguru.com/>

Australasian Sonographers Association (ASA)

www.sonography.org



Proposed 2026 PiCSA Education Calendar

January	- ECG
February	- Echo
March	- Cath Lab
April	- Devices
May	- EP
	Trivia Night
June	- ECG
July	- Professional Issues
August	- Echo
September	- Cath Lab
October	- Paediatric
November	- Devices/EP

Call for Volunteers!

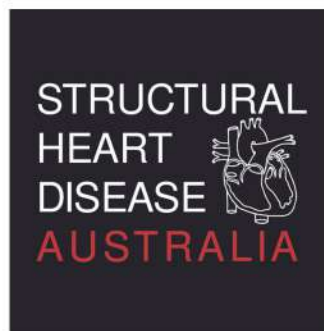
There are many ways to volunteer within the PiCSA organisation.

Interested? Get in touch!

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Affiliated Partners:



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



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Thank you for reading