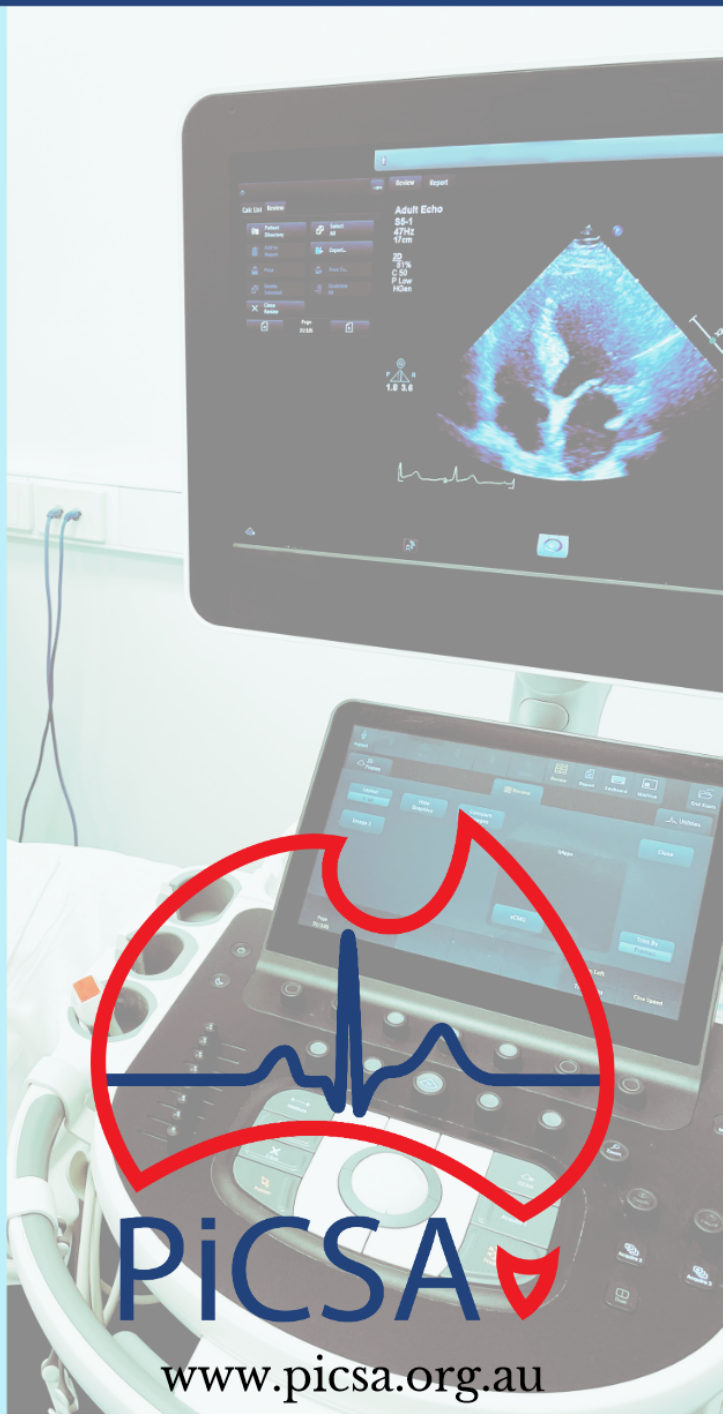
THE RHYTHM STRIP

The newsletter for Professionals in Cardiac Sciences Australia

Winter 2021



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

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From the Chair

Dean Metwally (NSW)

Hoping that your state/territory are staying safe during these trying times with the delta variant continuing to impact across the country. Please be extra vigilant with your exposure to patients – make sure your employer supplies you with appropriate personal protective equipment (PPE) and be mask fit-tested where possible.

I want to take the opportunity to thank all of the presenters and facilitators who enable us to provide regular education to our members. Structural Heart Disease Australia (SHDA) have been great partners with PiCSA, providing discounted access to their content. The Cabrini Institute, Macquarie University, Western Australia and Janssen have been very generous with their continued efforts in improving professional standards. Further, the Royal North Shore Hospital (RNSH) team have been sharing their high quality EP education, which has been well attended.

Reflecting on the National Cardiac Physiologist Day in May, Trivia night was unfortunately poorly attended with only 5 teams participating. Those that did attend had a great time and it became quite competitive. Congratulations to RNS on winning for the second consecutive year and I hope you enjoy hearing from a couple of the team members in this newsletter. If you or your colleagues have an alternate suggestion for celebrating our National day, please email secretary@picsa.org.au.

PiCSA continue to work with the Australian Council of Clinical Physiologists (ACCP) on the Accreditation Framework including competencies for the five Cardiac Physiology (ECG, Cath, Echo, Devices and EP) including CPD requirements. Tentatively, the ACCP is hoping to have the framework finalised ready for implementation by the end of 2021. It cannot be underestimated the volume of work that the ACCP have and continue to put into this. It is an exciting time to be a Clinical Physiologist as this is going to further validate our profession.

Has the formation of the ACCP had any impact within your workplace? Are you and your colleagues ACCP members? For managers, have you reviewed your practices position descriptions to include "eligible for ACCP registration"?

A reminder, that any Cardiac Physiologist who is registered with the Australian Sonographer Accreditation Registry (ASAR) but also performs other CP modalities (ECGs, cath lab duties, cardiac devices and EP), is strongly encouraged to also register with the ACCP. This provides safety to the public that there are standards in place, as there already are in Echo, for other modalities that CPs practice in.

As of 18 July 2021 there are 53 Cardiac Physiogoists who are registered as Accredited Cliniccal Physiologists through the ACCP. PiCSA hopes that this number will increase dramatically as employers increasingly recognise the importance of having a registered workforce.

The ACCP recommends "all accredited physiologists to include the post-nominal: 'ACP' in your clinical and professional communication. Your post-nominal accredited status, and the commitment to maintaining it is also a mark of the importance you signify in relation to governance and professional standards in your clinical work. Your accredited status is also a differentiating mark of excellence to peak professional bodies, employers, and your patients."

Take care

Take care,
Dean

PiCSA Chair

Thank you to Susan Boucaut former Clinical Advisor to PiCSA

Sue has been associated with cardiac physiology for all of her working life. It all started at The Prince Charles Hospital Queensland in April 1970. Sue retired as Director of Cardiac Sciences at the Prince Charles, Redcliffe and Caboolture Hospitals in September 2013.

During her career she strived to represent Cardiac Physiology to enhance our standing and recognition within Queensland Health, and on a wider national basis. Sue has served on a number of local and statewide committees including the position of Queensland representative for the CSANZ Affiliates Group. Her commitment to tertiary student education was recognised when she was awarded an academic appointment as Senior Lecturer at the University of Queensland in 2008, after being awarded an academic title as Associate Lecturer in 2005. In November 2013, she was delighted to be granted life time membership from Professionals in Cardiac Sciences Australia (PiCSA).

Sue has encountered many challenges during her career; the biggest of these from her perspective has been the recognition of our profession as a legitimate entity within health services.

She has been a trailblazer for Cardiac Physiologists in her own state, and beyond. She spearheaded Queensland's fight for appropriate award conditions and has always promoted the concept that governance and educational standards for Cardiac Physiologists are best managed and implemented by those who have the skills, experience, knowledge and respect of their peers.

PiCSA would like to thank Sue for her time on PiCSA's Council of Clinical Advisors. On behalf of the Cardiac Physiology profession, we are thankful for her dedication and support in building and maintaining the commitment to ethical and responsible service delivery by qualified and well supported practitioners.

If you are interested in, or could recommend a colleague for, a Clinical Advisor position

Membership Report

Tina Hetherington (QLD)

Memberships

- Membership for undergraduate students is now free!
- PiCSA membership fees are now reduced by 50% as we are halfway through the membership year.

Professional Membership $55
Associate Membership $45
Industry Membership $55
Affiliate Membership $45
Student Membership FREE

1 Life member
1 Affiliate member
10 Associate members
3 Industry members
2 Student members
128 Professional members

Upcoming education meetings

September (see PiCSA announcements) RNSH Sydney EP education series.

September (date TBC) - via Zoom (PAH Brisbane)

- Breony Heanue: Mental Health of Cardiac Patients
- Supporting Young Hearts Program
- Australian Centre for Heart Health

14th Oct - via Zoom (Maquarie Uni Hosp Sydney)

- Assoc Professor Jodie Ingles
- Kenny Ng
- Miriam Norman

Postponement of the 2021 Annual General Meeting (AGM)

The PiCSA board are currently working with our legal team on a much needed update to our constitution. We would like this to be ready for voting at our AGM this year!

The previously announced AGM date (11th of September) will be moved to the end of November.

Please keep an eye on our socials for speaker announcements and updates closer to the date.

THANK YOU

A Special Thankyou to PiCSA member Sam Burgoyne

PiCSA are grateful to all our supporters who assist in organising our educational activites. The PiCSA Board would like to thank Sam Burgoyne who has organised several educational meetings base in Western Australia over the last 3 years.

Sam is an experienced Cardiac Physiologist who currently works for Genesiscare Bunbury. She is a senior cardiac sonographer and also undertakes work as a cardiac point-of-care educator. Sam has worked throughout Australia and Canada and has previously worked as a Clinical Applications Specialist and Secondary School Teacher!

Sam believes in evidence-based practice and the importance of striving for excellence so as to ultimately improve healthcare. She also chairs the ASA Cardiac Special Interest Group.

Thank you Sam for your ongoing support.

Echocardiography in Simple Shunts - ASDs

Justin Gordon (QLD), Queensland Children's Hospital

Communications between the atrial chambers are a common form of congenital heart disease presenting in adulthood. There are four types of defects which allow shunting at atrial level:

- ostium secundum defect
- sinus venosus defect
- ostium primum defect
- coronary sinus defect (rare and not discussed in this article)

Universally grouped together as atrial septal defects (ASDs), it should be noted that the only common feature between these defects is mixing of the systemic and pulmonary blood flow at the atrial level with resultant volume overload and dilatation of the right atrium (RA) and right ventricle (RV).

The true atrial septum, defined as the small amount of tissue which directly separates the atrial cavities and which can be removed without exiting the heart, is limited to the thin oval fossa valvular tissue and its immediate muscular rims (Figure 1). Inter-atrial communications outside of this region will involve structures other than the atrial septum making the evaluation of these shunts more complex.

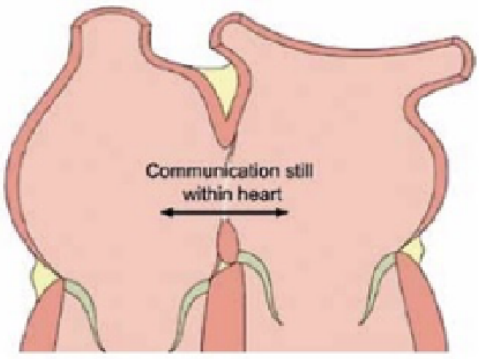


Figure 1. Atrial communication shown within the true atrial septum. Superiorly the atrial septum is formed by an infolding of the right and left atrial walls containing extracardiac tissue. Image courtesy of Gemma Price, 2009

Ostium Secundum Defect

Ostium secundum ASDs are deficiencies within the confines of the muscular borders of the oval fossa, the true atrial septum. These defects can be readily appreciated in most patients by transthoracic echocardiography in routine subcostal views (Figure 2). Larger defects may also be apparent from the apical 4 chamber view though care must be taken not to overestimate the size of the secundum ASD from this view due to artifactual drop out in the thin oval fossa region.

As transcatheter device closure of secundum ASDs has become a widely accepted and practiced procedure, the proper evaluation of these ASDs should routinely involve a measurement of the size of the ASD and also the relationship of the defect to its surrounding structures such as the mitral and tricuspid valves, superior and inferior caval veins and pulmonary veins.

The right parasternal view (described later) should be performed when assessing ostium secundum defects to evaluate suitability for transcatheter device occlusion. If the defect is too large or if there is a lack of suitable rims around the defect to provide anchorage for a device closure, then surgical closure is required.

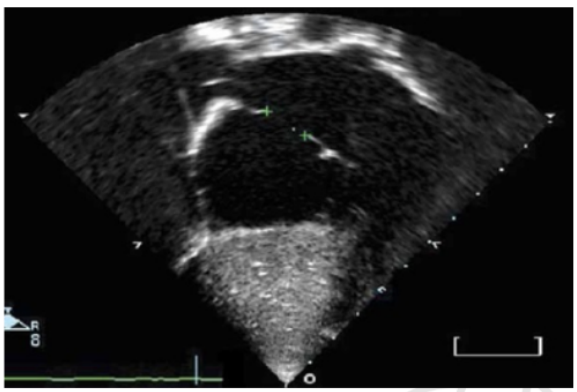


Figure 2. Subcostal measurement of a secundum ASD

Sinus Venosus Defect

Sinus venosus defects (SVASD) are relatively uncommon forms of inter-atrial communications. These defects are located separate from the oval fossa and true atrial septum. Al Zaghal et al propose that the anatomical prerequisite for a SVASD is overriding of the defect by the mouth of the superior, or rarely, the inferior caval vein (IVC). Along with the bi-atrial attachment of the caval vein (IVC or SVC) there is an association with partial anomalous pulmonary venous drainage (PAPVD) in up to 90% of cases.

Using conventional echocardiographic views, demonstration of a SVASD can often be difficult, with defects in this area frequently missed. From a standard subcostal 4 chamber view there may be no defect noticeable at the level of the oval fossa. The atrial septum may be thought to be intact and the patient referred for more invasive imaging techniques such as intracardiac or transoesphageal echocardiography to obtain a diagnosis.

The modified sagittal subcostal view, routinely performed in a paediatric echocardiographic laboratory, and the right parasternal view, conventionally used to assess the aortic valve and the ascending aorta, have been reported to adequately identify both SVASD and PAPVD in the majority of patients and should be performed when an ASD is suspected.

Subcostal Sagittal View

The subcostal sagittal view is used in adult echocardiography laboratories to demonstrate collapsibility of the IVC to estimate right atrial pressures. In paediatric echocardiography this view is routinely used during the sequential segmental analysis scan to assess the atrial septum. With superior and rightward angulation from the standard IVC/RA junction view, the SVC and IVC can be imaged along their long axis with visualisation of the bi-caval junctions with the RA and appreciation of any defects within this region (Figure 3).

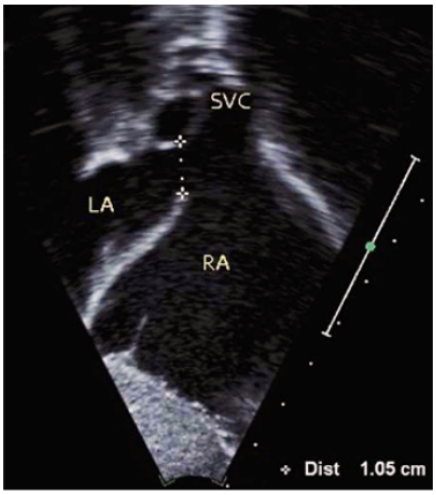


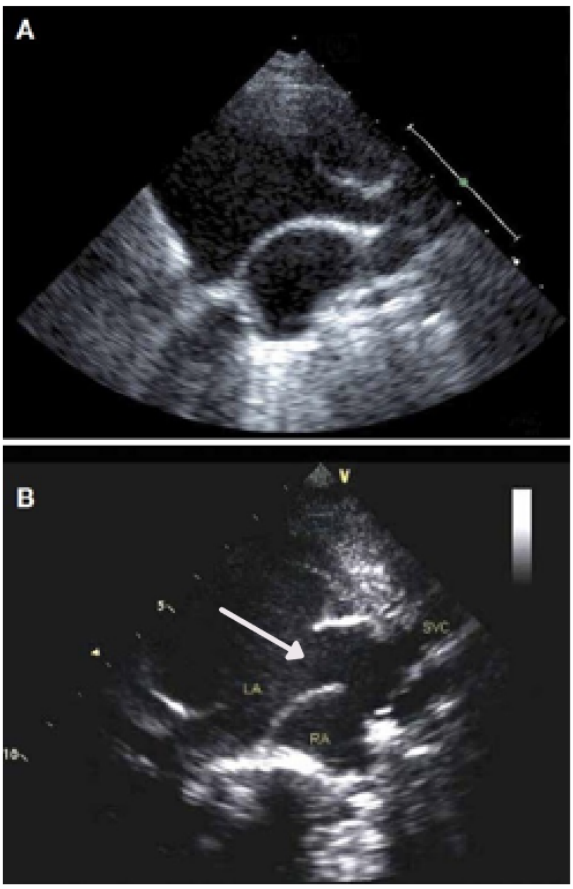
Figure 3. Sinus venosus ASD viewed from the subcostal sagittal view. The image marker is directed towards 12 o'clock with slight superior and rightward angulation

Right Parasternal View

The subcostal sagittal view may be limited in obese patients in whom a subcostal approach may provide poor quality images. For the assessment of ASDs, a right parasternal view may provide adequate diagnostic images. The right parasternal view can be obtained by applying the probe on the right side of the patients' sternum while they lie in a right lateral decubitus position. The position of the probe may vary between patients and range from the sub-clavicular area to the lower chest, but the image index marker should be orientated towards the patient's head, rotated to approximately 1pm. When the aortic valve and ascending aorta are visualised, slight rightward and inferior angulation should allow visualisation of the SVC as it enters the RA and the inter-atrial septum. From this view, visualisation of the superior posterior portion of the atrial septum is greatly enhanced and defects within this portion readily appreciated (Figure 4).

Sinus venosus defects do not close spontaneously, and surgery is required to close the defect leaving unobstructed drainage of the caval veins to the RA and redirection of the pulmonary veins to the left atrium (LA).

within this region (Figure 6).



Figures 4a and 4b. Right parasternal bicaval view showing (A) an intact atrial septum and (B) a sinus venous ASD with the bi-atrial SVC connection

Ostium Primum Defects

In the ostium primum variant of an ASD, it is the atrioventricular septal structures rather than the atrial septum which are deficient. It is for this reason that the ostium primum defect is not a 'simple' ASD, rather a partial form of the congenital cardiac malformation known as atrioventricular septal defect (AVSD).

Whether a partial AVSD with shunting only at the atrial level, or a complete AVSD with shunting at atria and ventricular level, the characteristic feature of this defect is the presence of a common atrioventricular junction which bears no resemblance to the junctional structures of the mitral and tricuspid valves seen within the normal heart. The common atrioventricular junction is guarded by a common, five leaflet atrioventricular valve, which in the partial

The ostium primum ASD is best appreciated from the apical 4 chamber view with the defect noted at the crux of the heart. This view will also show the distinguishable lack of atrioventricular valve offsetting found between the normal mitral and tricuspid valves (Figure 6).

Currently there is no role for transcatheter device occlusion of the ostium primum ASD with surgical repair required for all cases. The objectives of the repair are to close the atrial defect and repair the often regurgitant atrioventricular valve. The zone of apposition between the inferior and superior bridging

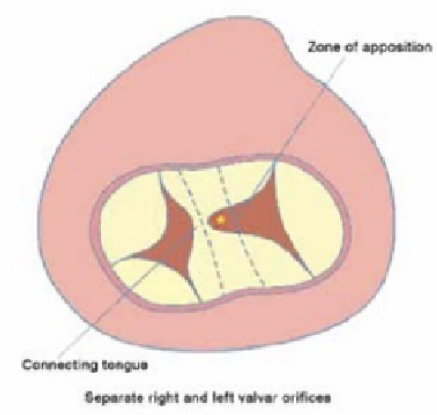


Fig 5. Diagram of the common atrioventricular junction of the partial AVSD (ostium primum ASD). The superior and inferior bridging leaflets are joined by a connecting tongue of tissue tethered to the ventricular septal crest creating separate left and right valvar orifices. Image courtesy of Gemma Price, 2009

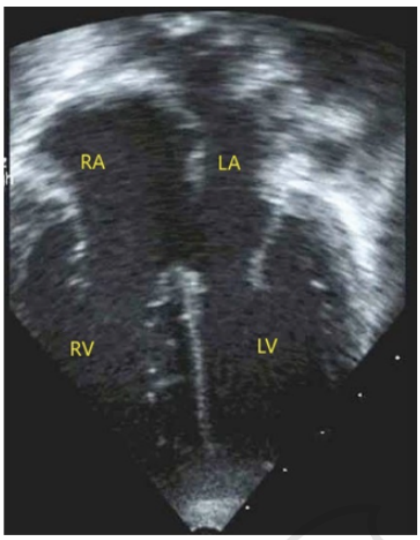


Fig 6. Apical 4-chamber view of the ostium primum

AVSD variant is tethered to the ventricular crest creating separate left and right valvular orifices, allowing shunting to occur at only

ASD. Note the separate left and right valvar orifices and lack of atrioventricular valve offsetting

leaflets is often closed to restore a bifoliate left atrioventricular valve (LAVV), if this can be done without producing LAVV stenosis. Ongoing echocardiographic follow-up is required in this condition to assess the integrity of the atrioventricular valves and patency of the left ventricular outflow tract (LVOT) which, due to its tunnel like anatomy, has the potential to become obstructed both pre and post operatively.

Conclusion

Atrial septal defects are a common incidental finding in an adult echocardiography lab. These so called "simple" shunts are usually evaluated by standard echocardiographic views with the emphasis in many practices placed on the calculation of a Doppler derived estimation of the intracardiac shunt. Commonly, patients with suspected ASDs are referred for more invasive diagnostic procedures such as transoesophageal echocardiography (TOE). With the routine use of nonstandard imaging planes (subcostal sagittal view, right parasternal view) and understanding the atrial septal defect anatomy and associated anomalies, the necessary information may be obtained through transthoracic echocardiography alone.

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Cardiac Antiarrhythmic Drugs

Amanda Marie Salma & Isabelle Saad (NSW), Macquarie University Hospital

The Cardiac Action Potential

The cardiac action potential (AP) is an electrochemical process which results in the contraction of cardiac myocytes. This process involves four phases that involve movement of Ions across the cardiac membrane otherwise known as the sarcolemma (1).

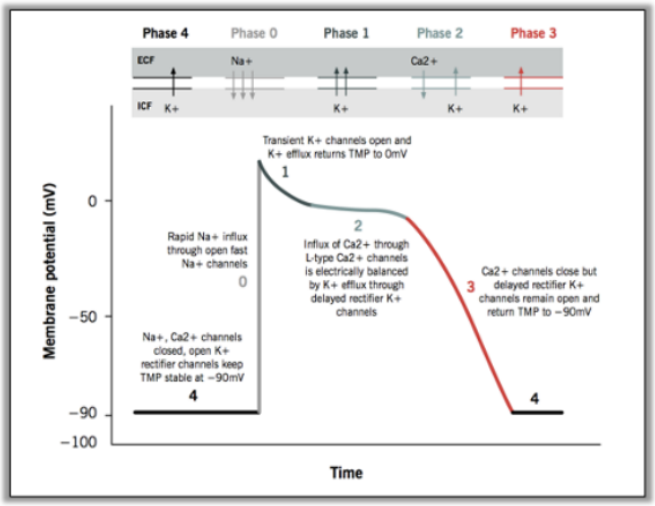

Figure 1. Phases of the cardiac resting membrane potential (1).

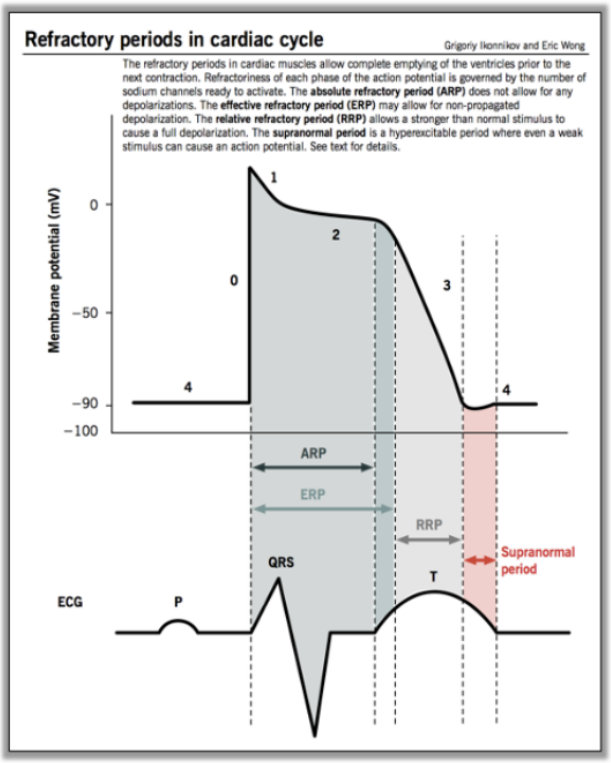

Figure 2. Relationship between the cardiac cycle and the membrane potential (1)..

Phase 4:

The first and last phase of the cardiac AP. The resting transmembrane potential (TMP) is negative at -90mV as potassium (K+) channels slowly but constantly release K+ out of the cell (1).

Phase 0:

Depolarisation of the cell occurs during this phase due to rapid but brief movement of Sodium (Na+) into the cell, when the TMP has increased to -40mV, Na+ channels close and Calcium channels open to allow a small influx of calcium (Ca2+) into the cell (1).

Phase 1:

Early repolarization is achieved when K+ channels are momentarily opened to reach a TMP of approximately 0mV through a small efflux of K+(1).

Phase 2:

Calcium channels and potassium channels both remain open creating a transient period of equilibrium, maintaining the TMP at approximately 0mV.

Accumulation of Ca2+ contributes to calcium induced calcium release (CICR) via T-tubules in the sarcolemma that stimulate calcium release from ryanodine receptors in the sarcoplasmic reticulum, generating a myocyte contraction

Phase 3:

In this phase calcium channels slowly close and potassium continues to leave the cell, therefore restoring the TMP to -90mV and preparing the cell for another cycle.sodium/calcium exchange, calcium ATPase and Na+/K+ ATPase pumps allow for the outward movement of ions into the extracellular fluid and allow for the transport of potassium inside the cell, achieving normal gradients (1).

(1).

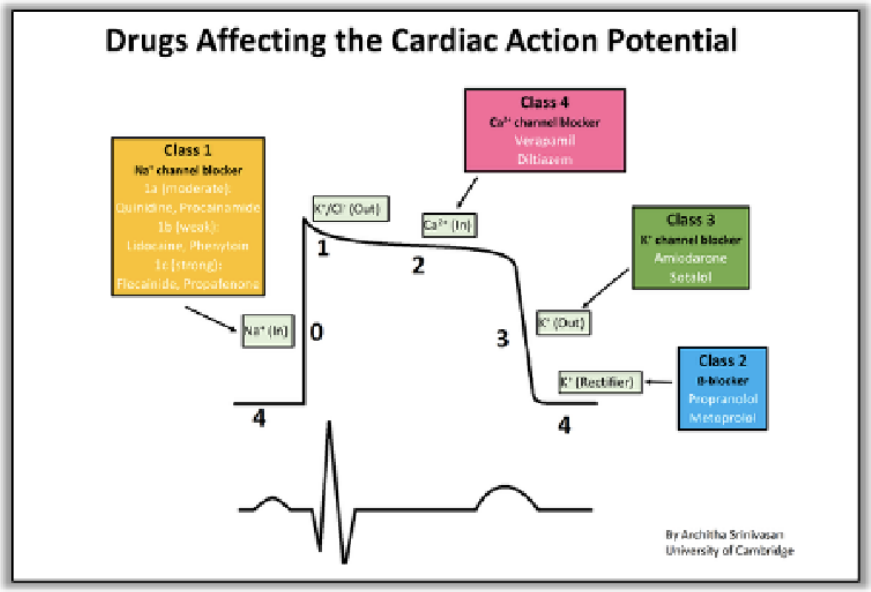

Figure 3. Drug classes and their effects on the cardiac action potential (2).

Class I

Class I drugs, known as membrane stabilizing drugs, are used to suppress abnormal rhythms of the heart such as: Atrial flutter (AFL), atrial fibrillation (AF), ventricular flutter (VF) and ventricular fibrillation (VFib). Class I drugs are sodium (Na+) channel blockers which directly bind to Na+ channel receptors and prevent rapid depolarization of non-nodal cardiac cells (3). This occurs in phase 0 resulting in a decrease in amplitude of the AP. The blockage of sodium channels reduces myocyte conduction velocity resulting in suppression of abnormal heart conduction causing tachycardias (3). There are three subgroups of Class I drugs. These are:

Class Ia:

Class Ia is an intermediate acting Na+ channel blocker and has a negative dromotropic effect on cardiac myocytes. Meaning that this class prolongs the duration (APD) of electrical stimulation on cells and as a result prolongs the Effective Refractory Period (ERP) between impulses making it effective in suppressing reentrant tachyarrhythmias due to conduction abnormalities. Class Ia drugs can also have a secondary effect on K+ (potassium) channels resulting in a prolonged ERP by regulating the slow efflux of K+ ions out of the cell (phase 4). This effect can be seen in an Electrocardiograph (ECG) as a prolonged Q-T interval (3). Class Ia drugs are prescribed for atrial, supraventricular (SVT) & ventricular tachyarrhythmias. **Quinidine** is an example of a class Ia anticholinergic drug, it acts by delaying repolarization and can have side effects such as cinchonism, nausea & cramping and enhanced digitalis toxicity (3).

Class Ib:

Class Ib agents such as **Lidocaine** are weak Na+ channel blockers that rapidly dissociate from their substrates. Therefore, due to class Ib's indirect blockade K+ channels and fast dissociation it does not prolong the APD and ERP as seen in Class Ia, although Class Ib drugs can also decrease AP amplitude (phase 0) like their class Ia. These agents target ventricular tachyarrhythmias (4).

Class Ic:

Class Ic drugs such as are slow dissociating Na+ channel blocker making them more potent than Class Ia and Ib in their ability to prolong repolarization of non-nodal cardiac muscle cells. This class does not directly block potassium channels and therefore has little to no effect on the APD. Class Ic agents are used for the suppression of SVT and VT. A common Class Ic drug used is **Flecainide** for the treatment of the aforementioned dysrhythmias however, due to the negative inotropic effects of this drug, it is contraindicated in patients with sustained ventricular arrhythmias as there is risk of sudden cardiac death (1, 4).

Class II:
Class II antiarrhythmic drugs are commonly known as **beta-blockers**. Beta-blockers mostly affect the sinoatrial (SA) and atrioventricular nodes (AV). The role of the Beta-blockers is to slow down heart rate through decreasing automaticity in the SA node, prolonging the refractory period and thus slowing conduction velocity. As automaticity in the SA decreases, the P-R interval is prolonged on the ECG. **Propranolol** is an example of a class II non-selective beta-blocker. This drug has a direct membrane effect blocking the Na+ channels and prolonging the duration of the action potential. **Propranolol** is used for the treatment of supraventricular arrhythmias. Side effects can occur if taking this class II drug. These include: Bradycardia, hypertension and AV blockade (5). However, it is not common for Beta-blockers to have a proarrhythmic effect (4).

Class III:
Class III antiarrhythmics such as **amiodarone**, **sotalol** and **bretylium** are potassium rectifier channel antagonists. These drugs prevent their substrates from activating and causing repolarization, as observed in phase 3 of the cardiac action potential. Refer to figure 1. This then results in prolonged depolarisation due to an increase in phase 1 and 2 of the cardiac action potential which correlates to the effective refractory period duration, delaying myocyte repolarization (3). Refer to figure 2 (1). To reiterate, this effect corresponds to the QT interval on an ECG. This class of drug may have proarrhythmic effects and is contraindicated in patients with a long QT interval as stated earlier as class III drugs extend the ERP and as a result of this, arrhythmias such as torsades de pointes or ventricular tachycardia may be triggered. **Amiodarone** has an array of adverse effects such as lung, liver, and thyroid toxicity. Its therapeutic use should be administered with careful consideration (3).

Class IV:
Class IV antidysrhythmic drugs such as **Verapamil** and **Diltiazem** are Ca++ channel blockers. This drug class has a negative inotropic effect on cardiac myocytes by delaying the influx of calcium into the cell needed for depolarisation. This occurs during phase 4 -0 of the cardiac AP. refer to figure 1. It is used in the treatment of atrial dysrhythmias and is contraindicated in patients with preexisting bradycardia, AV block, and heart failure (6).

Class V:
Adenosine binds to A1 receptors in cardiac non-nodal cells which are coupled with G-proteins which stimulate the opening of K+ channels resulting in hyperpolarisation of the cell (phase 4), this is particularly seen in vascular smooth muscle. Due to the potassium efflux, calcium is inhibited from entering the cell and therefore cannot regulate smooth muscle contraction further causing muscle relaxation. Furthermore, in pacemaker cells, when adenosine binds to A1 receptors it can have a negative chronotropic and dromotropic effect, however it is not uncommon to observe patients experiencing baroreceptor reflexes which results in an increased heart rate due to a sudden onset of systemic hypotension caused by vasodilation. **Adenosine** is very useful in the treatment of SVT; however, it is known to cause headaches and flushing, due to its negative chronotropic and dromotropic effects it is strongly contraindicated in patients with 3rd degree AV block (3). Atropine is a muscarinic receptor antagonist. This drug affects vagal activity in the parasympathetic nervous system. **Atropine** binds to muscarinic receptors which results in the activation of potassium/acetylcholine causing an increase in K+ out of the cell and subsequent hyperpolarization (phase 4), as seen with adenosine. Pacemaker cells are particularly affected by **atropine** and can be used in the treatment of bradycardia or AV block and is contraindicated in glaucoma patients (3).

In Summary there are many available drugs used in the treatment and management of cardiac dysrhythmias. It is recommended that careful consideration be taken of a drugs mechanism,

therapeutic use, adverse effects and contraindications be taken before administration to patients.

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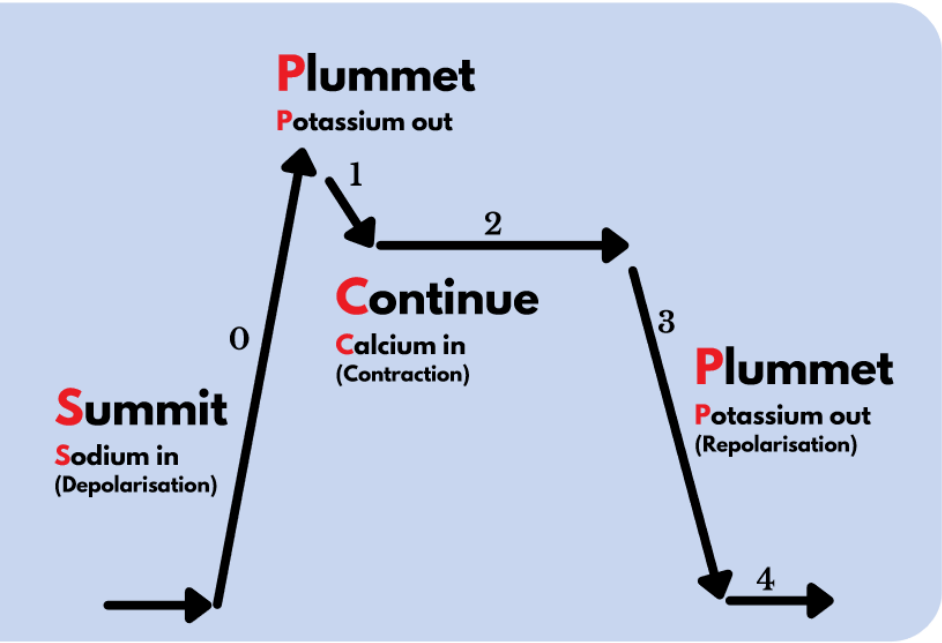
Tricks to remembering the Action Potential and Antiarrhythmic Drugs

Miriam Norman (TAS), Royal Hobart Hospital

The previous article from Amanda Salama and Isabelle Saad gives a lot of very excellent detail, but these details can be hard to recall in an exam!

Here is a simple catchphrase that will help you remember the main ions involved with the phases of the cardiac action potential:

Summit, Plummet, Continue, Plummet

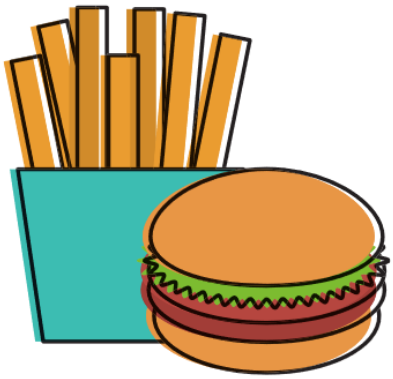


It also isn't easy to remember the different classes of cardiac drugs. A mnemonic to help you remember the 4 broad categories is:

Some Block Potassium Channels

Some	Sodium channel blockers	Class I
Block	Beta blockers	Class II
Potassium	Potassium channel blockers	Class III
Channels	Calcium channel blockers	Class IV

Class I agents (Na+ channel blockers)

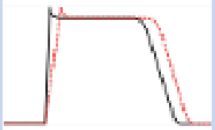
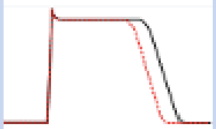
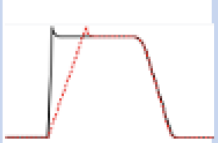


These agents all interfere with the sodium (Na+) channel, but they are sub-grouped into different classes.

There is a popular mnemonic to help remember the class 1 agents. It's like ordering a burger!

Double Quarter Pounder, Lettuce Mayo Tomato, More Fries Please.

I've highlighted the more common ones in red (these are the most important ones to remember for everyday practice)

Class Ia	**Double** **Quarter** **Pounder**	**Disopyramide,** **Quinidine,** **Procainamide**	 Ia **lengthens** the action potential **(right shift)**	**Major role** **=** **Rhythm control**
Class Ib	**Lettuce** **Mayo** **Tomato**	**Lidocaine,** **Mexiletine,** **Tocainide**	 Ib **shortens** the action potential **(left shift)**	
Class Ic (can ↑ pacing threshold)	**More** **Fries** **Please**	**Moricizine,** **Flecainide,** **Propafenone**	 Ic does not significantly change the duration of the action potential **(no shift)**	

Class II agents (Reduce Ca++ influx in phase 2)

These are conventional beta blockers, and **end in "ol"**
e.g. atenolol, metoprolol, propranolol, sotalol

They decrease sympathetic activity on the heart and **slow AV node conduction.**

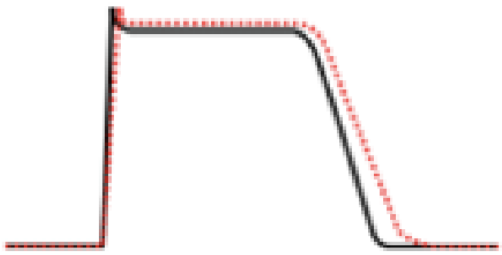
Major role = Rate Control

Often used to treat SVT

Note that if the last 4 letters aren't **olol**, then there is additional capability.

For example, "Sot**alol**" is also a class III agent, and "Carved**ilol**" has both beta and alpha blocking ability.

Class III agents (Block K+)



These agents affect potassium (K+) efflux and prolong the repolarisation period.

e.g. **Amiodarone** and **Sotalol**

Often used to treat re-entrant arrhythmias such as WPW syndrome, and to prevent (and treat) AF and VT.

Amiodarone can also increase pacing thresholds.

Memory tip: Think "Amy turned 3 and got a potato"
(*Ami has 3 letters – class III - potato for potassium!*)

Class IV agents (target Ca++ channels)

These agents are slow-channel calcium blockers and decrease conduction through the AV node. They also shorten phase II (the plateau) of the cardiac action potential.

e.g. **Verapamil** and **Diltiazem**

Prevent recurrence of paroxysmal SVT, and reduce V rate during AF.

Memory tip: The mnemonic at the top of this article "Some Block Potassium

Channels" will help you remember that class 4 involves Calcium.

Class V agents (Other/Unknown Mechanism)

These agents don't neatly fit into categories I through IV.

DAMS

- Digoxin: reduces AV node conduction and acts on the central nervous system to increase vagal activity
- Adenosine: Can terminate SVTs
- Magnesium Sulphate: Can treat Torsades de Pointes

Extras

Antidotes to remember (these are not cardiac drugs, but may be used in cardiac procedures and can appear in the IBHRE exams)

The antidote to Fentanyl is Naloxone
Memory tip: The Ox ate Fennel

The antidote to Midazolam is Flumazenil
Memory tip: King Midas got the Flu

Development of Chronotropic Incompetence in a Patient with a Secondary Prevention ICD

Jenny Fong and Janet Chen (VIC), Austin Hospital

A 73yo male presented to his GP with increasing exertional dyspnoea and lethargy. He did not have chest pain. His ECG showed sinus bradycardia with a heart rate (HR) of 45 beats per minute, PR interval 220ms and QRS duration 150ms. He was subsequently referred with a request "please turn heart rate up to 50".

This patient is well known to our cardiac device clinic - he has a single chamber implantable cardioverter defibrillator (ICD) with a high voltage lead placed in the right ventricular (RV) apex. He has a history of coronary heart disease with a myocardial infarct in 1987. A secondary prevention ICD was implanted in 2000 after presentation with resuscitated cardiac arrest, without an indication for pacemaker support. His current device (3rd generator) has been in place since 2011.

Device interrogation showed the following:

- Battery longevity 2.4 years (minimum)
- Normal device and lead function
- 17% ventricular pacing
- Parameters: VVI 50 bpm, hysteresis rate 40 bpm (Figure 1)
- Rate response (RR) was programmed *OFF*
- HR histogram profile was typical for chronotropic incompetence (Figure 2)

Basic Operation	
Mode	VVI
Magnet Response	Normal
V. Noise Reversion Mode	Pacing Off
Sensor	Passive
Threshold (Measured Avg.)	Auto (-0.5) (2.0)
Slope	10
Max Sensor Rate	100 min-1
Reaction Time	Fast
Recovery Time	Medium
Rates	
Base Rate	50 min-1
Rest Rate	Off
Max Sensor Rate	100 min-1
Hysteresis Rate	40 min-1
Search Interval	Off
Cycle Count	1 cycles

Figure 1 Bradycardia parameters

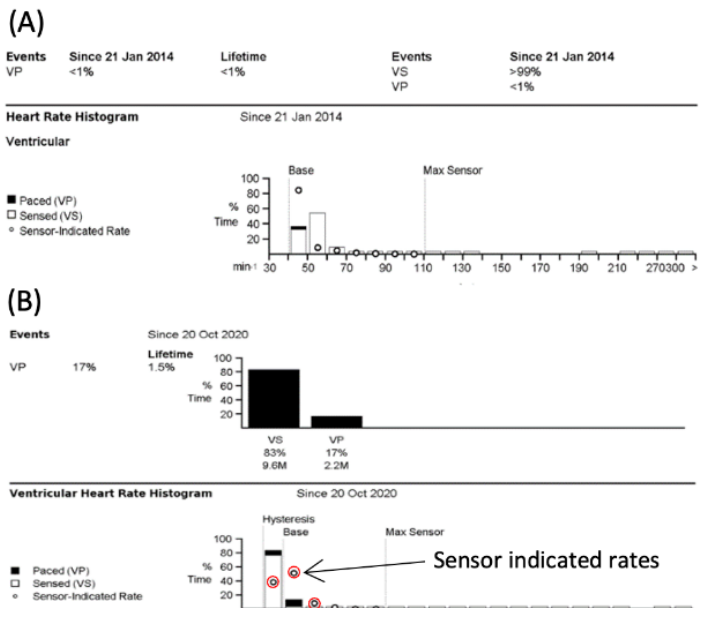
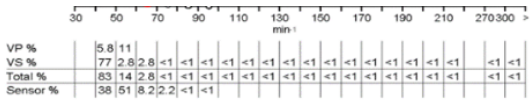


Figure 2 A, January 2014 histogram showing a normal HR spread with minimal ventricular pacing. Passive activity sensor data (circles) indicates that the patient's own chronotropic response was superior to that suggested by the device's RR algorithm.

Figure 2 B, October 2020 histogram showing chronotropic incompetence with the HR mostly sitting between 40-50bpm. The patient is not achieving the suggested sensor indicated rates (red circles) as determined by the device's passive RR algorithm.



Development of Chronotropic Incompetence in a Patient with a Secondary Prevention ICD- *continued*

Programming Changes:

In clinical practice, the Heart Rhythm Society recommends minimal ventricular pacing with preservation of AV synchrony(1).

Studies have demonstrated that long term RV apical pacing is correlated to increased death and hospitalisation for heart failure (2,3). The resulting atrio-ventricular and interventricular mechanical dysynchrony contributes to LV dysfunction and increases the incidence of atrial fibrillation (4,5,6).

Furthermore, some patients may show retrograde ventriculoatrial (VA) conduction during RV only pacing, resulting in atrial contraction against closed AV valves and a clinical diagnosis of pacemaker syndrome. Symptoms can include fatigue, dizziness and hypotension, and signs include cannon waves on the jugular vein and surface ECG. (7). For this reason, RV only pacing is generally contraindicated for sinus node disease.

Considering the evidence above, programming options were discussed with the pacing team:

Rate response was turned *ON* for the patient to receive pacing only when the sensor deems appropriate.

Programmed lower rates remain unchanged (VVI 50, hysteresis rate 40) so that the patient will not receive unnecessary pacing at rest.

Given the patient has intact AV conduction, consideration of adding an atrial lead would be appropriate at the time of generator replacement. The atrial lead will give the patient more physiological HR support (via atrial pacing) and help avoid ventricular pacing.

Patient is due back to clinic in 3 months. We will review his symptoms, pacing percentage and compare changes on his heart rate histogram.

Learning points:

- When there is no pacing indication, patients may receive a single chamber (VVI) defibrillator and be programmed to a low base rate to avoid unnecessary pacing.
- Hysteresis can help avoid unnecessary pacing.
- Heart rate histograms and sensor indicated rates can be used to assist with diagnosing chronotropic incompetence and to serve as a guide in device programming.
- If required, chronotropic support can be provided (through RR function) during times of activity. Please note that appropriate clinical review is required before programming RR ON, especially in patients with known ischaemic heart disease and/or chest pain. Patients must be advised to contact the device clinic if the new RR feature makes them feel worse instead of better.

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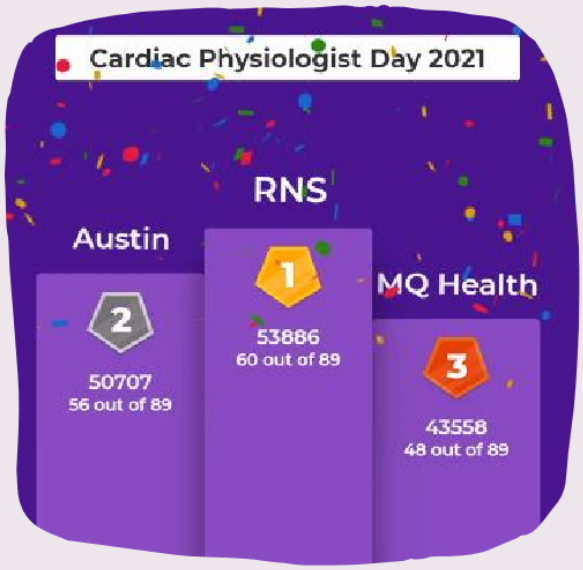
What do the winners enjoy most about being a Cardiac Physiologist?

My favourite thing about being a cardiac physiologist is the rewarding opportunity to be a part of a patient's diagnosis, treatment and follow up care. I am continuously learning as no two hearts are the same!

- Salwa Sandrussi

I enjoy the different variety of roles a career as a Cardiac Physiologist offers. Cardiology is an ever-expanding and developing field and there is always something to learn.

- Kenny Ng



PiCSA are excited to announce our affliation with the Australasian Sonographers Association (ASA)

The terms “Echocardiographer”, “Cardiac Sonographer” and “Echo Physiologist” are synonyms. This sub-group of Cardiac Physiologists (or sub-group of Sonographers if you will) have enjoyed the support of both PiCSA and the ASA for many years, and the two organisations have occasionally worked together behind the scenes.

PiCSA is now very pleased to announce that a formal affiliation has been created with the ASA so that we can further maximise support for Cardiac Physiologists who perform ultrasounds.

With over 7,000 members and a membership of more than 75% of Australasia’s sonographers, the ASA has a significant role in supporting, advising and advocating for the broader sonography group to provide the best possible outcomes for ultrasound patients. By working together and sharing our strengths we hope our organisations can do more and do better.

As a PiCSA member, look out for promotions and discounts on upcoming education events with the ASA.

Check our their website at *www.sonographers.org*

Conferences and Resources

PiCSA online educational content (members only)
https://picsa.org.au

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

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

Echo Supervisors Summit
October 2021 (tbc)
https://echosupervisor.com/

Echo Australia has been postponed until 2022

ASUM Conference 2021
19-21 November 2021
International Convention Centre, Sydney
https://conference.asum.com.au

Call for Volunteers!

There are many ways to volunteer within the PiCSA organisation. What skills can YOU offer?

- Leadership
- Finance/Accounting
- Graphics/Design
- Photography
- Social Media
- Website Design
- Speaking
- Teaching
- Recruiting
- Administration
- Secretarial/Organisational skills
- Policy Development
- Professional Expertise
- Communications
- Research
- Networking
- Strategic Thinking
- Risk Management
- Enthusiasm

Interested? Get in touch!
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.

Join us in-person for the ASA Brisbane Cardiac Student Seminar hosted at CQU

This seminar will showcase expert speakers who will switch between live-scanning workshops and lectures throughout the day. This event is specifically tailored to cardiac student sonographers.

Speakers include:

- **Peter Eades** – Student Practicum Engagement
- **Ruth Ramm** – Fetal Heart Development and Cardiac Anatomy
- **Nikki Rathbone** – Ergonomics/Strong to Scan Program and Exercise Stations
- **Kate Marriott** – Interactive Quiz

Registration closes Friday 27 August. Capacity is limited, book now!

Date: **Saturday 28 August 2021**
Time: 9.00 am – 4.00 pm
Venue: CQU Brisbane, Room TBC
Cost: Student member rate $25, non-member rate $150
Morning tea and lunch provided

Register here: www.sonographers.org/cpds/cardiac-student-seminar

Student non-members can **sign up to the**


ASA FREE, and then register for this event to take advantage of this amazing member rate.
Become an ASA member free here
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