SCT Newsletter

May/June 2015

Hello and welcome to the May/June edition of the SCT newsletter. Most of the content in this newsletter is member supplied which is great to see. Massive thanks to everyone who submitted something and I look forward to seeing more submissions for the next newsletter.

Education Committee Update from Christine Shanahan

We had a meeting following the CCP examination on the 18th June in Auckland, and as per usual, there was all day discussion which included exam results and how best to improve training for our students around the country (CCP, CPM and MTEX).

Present at this meeting was: Christine Shanahan, Miriama Gideona, Angela Morgan, Ellen Woodcock and Graham Orsbourn (MTEX representative). Ian was excused as he had only just started back at work that week after taking parental leave, so we forgave him for that one! We have unfortunately had to say goodbye to Sacha Levings, as she has taken a role with Biotronik. I wish to thank her for her contribution to the education committee and wish her well in her new venture.

The Education committee ran the first online examination for CCP in June 2015, which proved to be very successful. There was overwhelming positive feedback from the students on its ease of use and there were no computer glitches or security issues to deal with! I wish to send a huge thank you to Ian McLeod from Tauranga, for all his volunteered hard work and hours in setting this up for the SCT students.

The next big task the committee will be working on is reviewing and revamping the CPM course this year. This should be ready for the 2016 student intake. As part of this work we will be reviewing the examination content and its format, with the goal of bringing that exam online for November 2016.

A huge congratulations must be sent to all those successful CCP students at the June exam sitting. Those students were:

- Mustafa Al Janabi (Middlemore Hospital)
- Simon Mummery (Auckland)
- Noah Pudumai (Auckland)
- Justine Paddison (North Shore Hospital)
- Delia Calain (Christchurch)
- Nick Hammington (Hutt hospital)

The next scheduled CCP and CPM examination is Wednesday 11th November. All practical assessments must be submitted no later than Friday 23rd October. All alternative assignments are due no later than 31st August for the November exam. The next education committee meeting is scheduled for Thursday 19th November. Exam results will be out on Friday 20th November. Any questions/suggestions regarding the CCP or CPM courses, please email Christine Shanahan on cshanahan@adhb.govt.nz

Amplatzer Devices by Sue Perkins, Cardiac sonographer, ACH.

Percutaneous closure of congenital heart defects is becoming preferred if possible to surgical repair. Clinicians are using these devices in the treatment of paravalvar leaks and to plug the LAA.

Amplatzer devices are used to non - surgically close these congenital defects:-

- A patent foramen ovale (PFO)
- An atrial septal defect (ASD)
- A patent ductus arteriosus (PDA).

PFOs are found in ~ 25% of the population, so why would it be necessary to close one?

- In patients who have stroke of unknown cause (cryptogenic stroke), the prevalence of PFO increases to about 40 percent.
- People with PFO do not need any treatment if there are no associated problems i.e. history of stroke or TIAs.

A PFO can be diagnosed by echocardiography.
A right to left shunt can be demonstrated during a Mueller or Valsalva maneuver.
Blood bypasses the lungs which act as a filter. Paradoxical embolisms are able to enter the systemic circulation and travel to an artery to the brain, causing a stroke or TIA.

Why close an ASD?

Depending on the size of the ASD, people with an ASD are at risk of developing:

- Arrhythmias - particularly atrial fibrillation
- Heart failure
- Heart infections (endocarditis)
- Pulmonary hypertension
- Stroke
- Sizeable ASDs with right heart dilation are associated with important age-related morbidity and mortality.

An atrial septal defect can be diagnosed with an echocardiogram.
Other tests may include cardiac catheterization, coronary angiogram (for older patients), ECG, MRI and transoesophageal echocardiography

Why close a PDA?

- If left untreated a PDA can lead to heart failure and pulmonary hypertension.
- It can also lead to the development of an Eisenmenger syndrome just as a large untreated ASD and VSD can
- An intracardiac communication allows high pulmonary artery pressures to develop and produces right-to-left intracardiac blood flow. Originally described in association with a large VSD, Eisenmenger syndrome can also manifest with a PDA.

Percutaneous left atrial appendage (LAA) closure is becoming a frequently performed procedure for patients with atrial fibrillation and high haemorrhagic risk. The Amplatzer™ Cardiac Plug (ACP) is one of the most commonly used devices for this purpose.

How long does it take for the ASO device to be endothelialized in the human body?

In animal models, most ASO devices are reported to be endothelialized in 3 months. Antiplatelet therapy and prophylaxis for endocarditis are recommended for 6 months following device implantation.

Amplatzer Inventor

Kurt Amplatz was born 1925 in Austria and came to live in United States in 1952.
He was a world-renowned University of Minnesota Radiology professor and researcher. He was a medical device pioneer.
His research led to the invention of the line of AMPLATZER occlusion devices.
AGA Medical was founded in 1995 by Dr. Kurt Amplatz and his son, Curtis.
On November 18, 2010, St. Jude Medical welcomed AGA Medical Corporation and the AMPLATZER® line of products to its Cardiovascular Division.
Before the Amplatzer device became available patients with congenital heart defects required open heart surgery.
“Now you can do the whole thing with a little stick in the groin. The patient goes home the next day without a scar on the chest” Amplatz says. The father and son worked out a way to braid threads of a super elastic (pseudo elasticity) alloy called nitinol into a mesh that formed into occluders that expanded into a previously memorized shape after passing through a catheter. Nitinol is an alloy of nickel and titanium discovered by the American

Navy in the 1959. The name is derived from its composition and its place of discovery- nickel Titanium – Naval Ordnance Laboratory
It has 2 unique properties:

1. Shape memory (hysteresis)
2. Super elasticity (pseudo elasticity) – like a super spring.

Biocompatibility, Corrosion Resistance
Nitinol has excellent biocompatibility, very high corrosion resistance, and excellent cytocompatibility.
The Nickel in Nitinol is chemically joined to the Titanium in a strong intermetallic bond, so the risk of reaction, even in patients with Nickel-sensitivity, is extremely low.

Adverse effects of Amplatzer devices.

The major adverse events associated with use of the Amplatzer devices include:

- Device embolization
- Device perforation or erosion
- Thromboembolism
- Incomplete septal defect closure.

Amplatz devices are being used to treat paravalvar leaks seen with mechanical prosthetic valves. The Amplatz Vascular Plug II and muscular ventricular septal defect (VSD) occluder are used depending on the size of the defect.
Dislodgement or device embolisation is the most serious complication. There is careful measurement of the ASD defect to be closed to determine if there are sufficient rims for the device to sit safely. Dislodgement occurs in 1 in 100 cases. If the device is small, embolisation to the abdominal aorta is usual and this can usually be retrieved via catheter. Larger devices often need to be removed surgically and usually need immediate attention. There is a potential for eroding and finally penetrating cardiac structures if the rim of one of its disks is rubbing against the atrial or aortic wall.
The “culprit lesion” is usually created by the anterior–superior border of the device leading to right or left atrial perforation or aortic wall erosion – in cases where the device is splayed over the aortic root. It is obvious that patients with a deficient superior atrial septal rim or deficient aortic rim are at higher risk for cardiac perforation.

CSANZ Conference report from Gay Noyer

CSANZ15 was a well-run scientific programme with highlights from regional and international developments in cardiology and presentations of innovative local research.

Genetics featured quite frequently during the conference at various stages, as there have been huge advances in this field during the last decade.

Genome-wide association studies (GWAS) examine many common genetic variants in different individuals to see if any variant is associated with a trait, especially major diseases.To track these disease carrying mutations, the importance of taking a family

history is stressed, followed by the drawing up a pedigree going back at least 3 generations, if possible, especially in patients one of the cardiomyopathies or channelopathies. There is variable penetrance within families and also age variability. Genetic variance and environmental factors affect when conditions may become apparent. Previously, many syncopal episodes and seizures have been put down to epilepsy, but now the majority of autopsy negative deaths are recorded as due to arrhythmias, often due to cardiac inherited diseases.

Genetic variants of a particular chromosome have recently been associated with a markedly increased risk of AF and led to the identifying of individuals at higher risk of developing the condition. As with other familial linked diseases, genetics can predict the efficacy of anti-arrhythmic drugs, the risk of AF recurrence after DC cardioversion and the efficacy of AF ablation. The aim is to collect data and create a bio-bank, with AF genomic testing now available cheaply on Auckland's North Shore, as a predictive, not diagnostic test.

Relatives of patients with LQTS, who have a normal or borderline QTc can undergo an exercise stress test. QTc prolongation at 4 minutes of recovery after an ETT can be used for the diagnosis of LQTS. A QTc ≥445 msec during the fourth minute of recovery is highly suggestive of LQTS. At 100 bpm a QT ≥ 370 msec is also highly suggestive of LQTS. Treadmill stress testing can also unmask patients with concealed LQTS.
A postural ECG done with a patient supine for several minutes then standing, should show the heart rate going up with a corresponding shortening of the QT interval. Due to the fact that heart rate goes up more than the QT comes down, the QTc actually goes up slightly. In LQTS sufferers the QTc often goes up substantially, e.g. 90-100 msec compared to 10-15 msec in healthy people.

High Performance Sport NZ spoke on screening young athletes (aged from puberty to 35 years). Although some countries and ethnicities decreased the rate of sudden cardiac death in young athletes with routine screening, in New Zealand it is felt that young athletes are not placed at a higher risk by participating in sports, as the incidence of SCD for those participating in sports is lower than that in the general population of the same age range and gender.
With elite athletes, HPSNZ generally investigates family history, plus a physical examination, with a routine12 lead ECG and biennial follow-ups.
It was noted that regular athletic training produces common ECG alterations that can be mistaken as abnormal and that reference be made to the "Seattle Criteria" – electrocardiographic interpretation in athletes.
Increased numbers of automatic external defibrillators in public places and at sports facilities were thought to be of greater benefit for athletes as well as spectators.
Physical inactivity is viewed as the greater problem!

As the proportion of adults with adult congenital diseases increases there are a large number of patients with congenital defects that require conventional pacemaker therapy post surgery, however, it is well known that RV apical pacing has detrimental effects on LV function. This, plus possible conduction disease, may further compromise cardiac performance. Cardiac resynchronization may have the potential to benefit a large number of ACHD patients with varying lesions. However, these patients pose additional challenges to CRT and associated lead placement because of their anatomy and physiology, plus previous sternotomies, scarring and especially abnormal coronary sinus anatomy.

Extensive heart failure, electrophysiology and device sessions covered a wide range of sub-topics. Susan Sinclair is working on a draft document outlining recommended practices for dealing with terminally ill patients with pacemakers or ICDs. This document will hopefully be available for hospices and the general public.
Terminally ill patients are at high risk of painful shocks at the end of life due to tachyarrhythmias and arrhythmia storms. ICD patients and their families need to be given information on what the device is and how it works, along with discussions at the original implant. A management plan for the dying patient should be in place in their file
She also went over the clinical evaluation required at the time of generator changes. This includes a full site review covering the possibility of resiting, patient discomfort, low grade infections and venous occlusions. Assessment of valvular and ventricular function from echo, LV pacing burden and the need to upgrade to CRT. The appropriateness of the current system must be reevaluated with regard to the reason for the original implant, pauses in AF, the need for a ventricular lead due to AV block and also rate sensor inclusion. Lead function is examined to ascertain any abnormalities, compatibility, placement and any alerts.

A high rate of biventricular pacing is required to achieve maximum benefit from CRT. With patients in AF, the use of CRT biventricular pacing rates reported by the CRT device may be misleading, since these rates include fusion and pseudo-fusion between the device pacing and the patient's intrinsic conduction.
AF increases the risk of inappropriate shocks. AV node ablation is effective in improving response by ensuring ventricular capture and reducing ICD shock burden. Without AV node ablation a high percentage of RV pacing is only achieved with a higher pacing rate.

Atrial reverse remodelling may occur in CRT responders. Reduced left atrial size and better atrial hemodynamics may result from improved LV haemodynamics. Some patients with persistent or permanent AF may convert to sinus rhythm after several months of CRT. Therefore, it may be prudent to wait for a period of time after CRT placement before performing AVN ablation, The disadvantages of CRT and AVN ablation with AF are continued AF, risk of thrombosis, loss of AV synchrony, it is an irreversible procedure, pacemaker dependency, complications of device or lead failure, constant pacing leads to rapid battery drain and therefore more generator replacements and consequently the probability of more complications.
Congratulations to Helen Logan for winning the MTEX case presentations at a recent block course. She produced a very well researched case study on alcohol septal ablation at CSANZ.

Thank you to the SCT Council for the Fellowship funding to cover my registration to attend this year's conference in Auckland.

SCT council Nominations

Ask not what the SCT can do for you, ask what you can do for the SCT!

We are still accepting nominations for the council for 2015-2016 but time is running out so if you would like to have the opportunity to be on the SCT council then please fill in the attached form and return it by Friday the 17th of July.