In this review:

- 25 by 25 – is it realistic?
- Mitral regurgitation – when to intervene
- Oral anticoagulation in NVAF
- Public policy needed for CV disease
- Heart rhythm disturbances in athletes
- Severe chronic heart failure
- A Core to the Cor brings Accord
- PCSK9 could do with some inhibition
- Undetectable hs-TnT in the ED is a good thing
- The benefits of synchronised squeezing
- De-innovation of renal denervation
- INSTABILITY continues

Welcome to our review of the World Congress of Cardiology 2014 (WCC 2014) held recently in Melbourne, and the American College of Cardiology Annual Scientific Session (ACC.14) held in Washington in March.

Cardiologist Dr Ian Termouth attended WCC 2014 and identified a number of oral presentations as being of particular interest to the cardiology community. The scientific programme for WCC 2014 can be viewed online at its website http://www.world-heart-federation.org/congress-and-events/world-congress-of-cardiology-scientific-sessions-2014/.

Associate Professor Stewart Mann did not visit the ACC.14 meeting in person, but he considered himself a "virtual" attendee after being bombarded with website updates and papers published online or in print. While these may not now be breaking news, it does allow reflection of some of the discussion around newly presented studies that has taken place in local corridors as well as website forums. The studies highlighted by Stewart can be found online at the links provided.

We hope you enjoy these conference reviews and find them useful in your current practice.

Kind regards

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25 by 25 – is it realistic and can we achieve it?

The importance of policies for prevention

Speaker: K. Srinath Reddy, India

Summary and comment (IT): Dr Reddy spoke on the 25 by 25 plan. This is a planned road map to reach 2025 targets, that was set out in September 2011. Cardiovascular disease is an increasing cause of death and morbidity. Unfortunately I did find the presentation a bit frothy with lots of laudable aims, but a bit vague on some hard choices that will need to be looked at. Action on 6 major risk factors will avert 37 million deaths by 2025. A road map clearly will be needed.

The importance of governments

Speaker: Robert Beaglehole, NZ

Summary and comment (IT): Dr Beaglehole emphasised that interventions are key and need to be cost effective and easy. Tobacco control is also key, and achievable. Political leadership accountability is essential, and civil society plays a critical role. Tax is a major weapon e.g. on sugar, tobacco and alcohol. Aiming for 25% mortality reduction. A 30% reduction in tobacco usage is the aim. If we get 50%, many millions of lives could be saved. Some Pacific states have done quite a lot but there are some terrible statistics e.g. Samoa and Fiji. Some good options are available. There is potential to achieve immediate and powerful effects in our neighbours.

Reference: WCC 2014 presidential symposium: 25 by 25 – is it realistic and can we achieve it?

Mitral regurgitation – when to intervene

Speaker: Patrick O’Gara, USA

Summary and comment (IT): There are two major causes of mitral regurgitation: primary (ischaematosus, rheumatic, calcific, endocarditis) and secondary (ischaemic, dilated cardiomyopathy, hypertrophic obstructive cardiomyopathy). Thresholds for surgical intervention have been lowered by serial improvements in techniques and outcomes, but have outpaced good trial data. Sending an asymptomatic patient for a major procedure is always tricky. Dr O’Gara showed data strongly supporting intervening early in flail leaflets – as long as the surgeon can give a high degree of assurance of a repair not replacement. Vasodilator therapy is not recommended for normotensive asymptomatic patients with normal LV systolic function (which is a bit counter-intuitive as one would expect it to reduce LV size and MR fraction/delay dilatation). Surgery is recommended in chronic severe MR (EF 30–60%, LVESD >40), and in asymptomatic patients if done in a centre of excellence with good results. Neo chordae with PTFE seems to be a major surgical advance too. Thresholds for surgical intervention have been lowered by serial improvements in techniques and outcomes, but have outpaced good trial data. Sending an asymptomatic patient for a major procedure is always tricky. Dr O’Gara showed data strongly supporting intervening early in flail leaflets – as long as the surgeon can give a high degree of assurance of a repair not replacement. Vasodilator therapy is not recommended for normotensive asymptomatic patients with normal LV systolic function (which is a bit counter-intuitive as one would expect it to reduce LV size and MR fraction/delay dilatation). Surgery is recommended in chronic severe MR (EF 30–60%, LVESD >40), and in asymptomatic patients if done in a centre of excellence with good results. Data were presented for vastly increased survival out to 7 years for this aggressive approach. He also presented good data on ischaemic MR showing improved outcomes for repair versus replacement. Neo chordae with PTFE seems to be a major surgical advance too.

Reference: WCC 2014 symposium: Mitral valve disease repair

WCC & ACC 2014 Conference Review

Independent commentary by Stewart Mann, Associate Professor of Cardiovascular Medicine at the University of Otago, Wellington.

Independent commentary by Ian Termouth, Cardiologist, Taranaki District Health Board.
Overcoming thrombosis: one oral anticoagulant for five indications

Speaker: Greg Lip, UK

Summary and comment (IT): In Europe many patients with high CHADS-VASC scores receive no anticoagulants, and (not surprisingly) they get lots of strokes. Dr Lip compared the different NOACs and was in favour of dabigatran (sponsor’s product), but did allude to its possible slightly increased risk of MI. He discounted this but was pretty fair about the other agents. Rivaroxaban and apixaban also seem pretty good; my reading is that if all were available apixaban might have the edge (rumours are that Pharmac is looking at it but those could be just that). Rivaroxaban is available in NZ on a discretionary basis – I use quite a lot of it. In the real world there is less bleeding with NOACs than with warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot.

Reference: WCC 2014 sponsored symposium: Use of oral anticoagulation in NVAF...where are we now?

Heart rhythm disturbances in athletes

Concerning symptoms in an athlete

Speaker: André de Gerche, Australia

Summary and comment (IT): Symptoms of concern in athletes include syncope (vasovagal, collapse), chest pain (rare and common types), impaired performance, and palpitations. Dr de Gerche presented some Italian data (they screen more than most): 5.6% of athletes have had syncope, but no sudden cardiac death in 7568 athletes after 7 years of follow-up. Syncope is a much older and more conservative definition than what many modern cardiologists would accept. Dr de Gerche also provided an overview of malignant ventricular arrhythmias, including some new data from athletes. He also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot. Dr Lip also discussed the conundrum of a STEMI or NSTEMI with an ACS; warfarin. Apixaban and rivaroxaban can be blister packed which is a bonus – dabigatran cannot.

Reference: WCC 2014 symposium: Heart rhythm disturbances in athletes

Severe chronic heart failure

Heart failure epidemics: already at the door?

Speaker: Mariell Jessup, USA

Summary and comment (IT): Dr Jessup spoke on the incoming tsunami of heart failure. Basically as sleek, what I term McDonaldisation’s toxicity, hypertension etc. goes up heart failure is sure to follow. Dr Jessup showed depressing statistics for glucose, obesity, metabolic syndrome, diabetes, insulin resistance and risk of CHF. He also spoke on SCD in athletes – drugs are often underlyingly. Biggest advance is having KEDs at sports venues (I can proudly say that every secondary school in Taranaki has one now thanks to my staff!). Drug use is very common “You don’t win the Tour de France on mineral water”. Lots of agents: steroids, cocaine, and some exotic sounding stuff from China that is not well characterised.

Reference: WCC 2014 symposium: Heart rhythm disturbances in athletes

Pharmacological management of heart failure: where do we stand?

Speaker: Michel Komajda, France

Summary and comment (IT): Dr Komajda discussed the pharmacological treatment of heart failure. He talked about ACE inhibitors – good (no news there), ARBS – good (albeit). He showed the annual rates of death in the placebo groups were 9–16% in trials (where we know outcomes are always better). Eplerenone good, spironolactone good. Then the newer interesting bits – irbesartan – 16% reduction in death vs placebo in patients on good standard treatments, and reductions in all subgroups – although not all significantly. Not yet funded but maybe a major advance if/when it is. Alikiren – renin inhibitor, ATMOSPHERE trial ongoing … watch this space. Refractory syncope (vasovagal) may need fludrocortisone or midolrine, but there is no role for pacing. Heat stroke is more common in intermediate distances like 5–10k than marathons. Treatment is ice tub; this out performs all other treatments. Avoid NSAIDS pre-race. Prognosis depends on duration of unconsciousness. When testing athletes rely on their histories, not use standard tests, and try to replicate their training environment.

Reference: WCC 2014 WHF-WHO Joint session

Atrial fibrillation in athletes

Speaker: John Camm, UK

Summary and comment (IT): This was a pretty depressing talk for those of us trying to postpone obsolescence/our own angiorry by exercise. There is a U-shaped relationship between atrial fibrillation (AF) and exercise. Athletes get more AF – and the definition of an athlete was pretty kind – even I could make it! Doubling of AF in men > 50y who have always done a fair bit (about 150 minutes a week). Endurance athletes have a 5-fold greater rate of AF than age- and sex-matched sedentary individuals. This may be due to bigger atria. BUT the AF in an athlete seems to be a more benign beast than the common or garden AF. He also spoke on SCD in athletes – drugs are often underlyingly. Biggest advance is having KEDs at sports venues (I can proudly say that every secondary school in Taranaki has one now thanks to my staff!). Drug use is very common “You don’t win the Tour de France on mineral water”. Lots of agents: steroids, cocaine, and some exotic sounding stuff from China that is not well characterised.

Reference: WCC 2014 symposium: Heart rhythm disturbances in athletes

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Password: healthy

Reference: www.researchreview.co.nz

A clear cut woohoo moment. Lots of new agents in the pipeline, and as always no doubt analysis/data dredging does seem to show some benefit in some groups, but not any false dawn. HFPEF – basically nothing proven to work yet. TOPCAT negative – although it could be wrong. LCZ696 – new in class Angiotensin Receptor Neprilysin renin inhibitor – PARADIGM-HF stopped early for benefit vs ACE inhibitor. Hopefully not another false dawn. HFPEF but I could be wrong. LCZ696 – new in class Angiotensin Receptor Neprilysin renin inhibitor – PARADIGM-HF stopped early for benefit vs ACE inhibitor. Hopefully not another false dawn. HFPEF but I could be wrong.

Reference: WCC 2014 symposium: Severe chronic heart failure
A Core to the Cor brings Accord

Authors: Adams D et al., for the U.S. CoreValve Clinical Investigators

Summary: This study compared transcatheter aortic-valve replacement (TAVR), using a self-expanding transcatheter aortic-valve bioprosthesis, with surgical aortic-valve replacement (AVR) in patients with severe aortic stenosis and an increased risk of death during surgery. 795 patients were randomised 1:1 to either TAVR or surgical AVR. At 1 year, TAVR was superior to surgical AVR for the primary endpoint of all-cause mortality (14.2% vs 19.1%; p=0.04).

Comment (SM): The key to this study was the selection of patients at documented high risk for open aortic valve surgery randomised to this or to TAVR with the Medtronic CoreValve self-expanding prosthesis. The first 3 patients in each centre were not counted, being deemed to be “roll-in” subjects where operators were getting over their initial learning curve – one could argue how many “roll-ins” you need and wonder what happened to them. The endpoint of 1-year mortality appears to be pretty realistic and TAVR proved superior at 1 year. Interestingly, the difference was not due to early postoperative mortality in the surgical group as curves did not separate until after about 2 months. The benefits of the CoreValve might outperform those of balloon-expandable devices although a head-to-head comparison would be needed to be sure.


PCS9 is a pesky enzyme that could do with some inhibition

Authors: Blom D et al., for the DESCARTES Investigators

Summary: This 52-week study investigated the safety and efficacy of evolocumab in patients with hyperlipidaemia. Patients were stratified according to risk before being started on background therapy with diet alone, diet plus atorvastatin 10 mg/day, atorvastatin 80 mg/day, or atorvastatin 80 mg/day plus ezetimibe 10 mg/day for 4–12 weeks. Patients with LDL cholesterol >1.9 mmol/L at the end of the run-in period were then randomised 2:1 to receive either evolocumab 420mg or placebo every 4 weeks. 901 patients were included in the primary analysis. After 52 weeks’ treatment with evolocumab, LDL cholesterol decreased by 55.7% from baseline in patients who underwent background therapy with diet alone, by 61.6% in those who received atorvastatin 10 mg/day, by 56.8% in those who received atorvastatin 80 mg/day, and by 48.5% in those who received atorvastatin 80 mg/day and ezetimibe (all p<0.001).

Comment (SM): PCSK9 is an enzyme synthesised in the body as a proprotein (PCSK9-ogen?) and stripped of a few peptides by a convertase for which there are now a number of inhibitors. Use of these inhibitors reduces active PCSK9 and consequently LDL cholesterol. If the LDL hypothesis holds good for non-statin drugs (yet to be convincingly shown) then use of such inhibitors may well lead to potentially improved clinical outcomes although drugs manufactured as monoclonal antibodies (such as evolocumab, alirocumab, bococizumab and others) might have a hard time competing financially with generic statins shown) then use of such inhibitors may well lead to potentially improved clinical outcomes although drugs manufactured as monoclonal antibodies (such as evolocumab, alirocumab, bococizumab and others) might have a hard time competing financially with generic statins.


Undetectable high-sensitivity troponin at presentation puts you on your bike

Authors: Bandstein N et al.

Summary: This Swedish study investigated whether an undetectable (<5 ng/L) high-sensitivity cardiac troponin T (hs-cTnT) level and an ECG without signs of ischaemia can rule out MI in the emergency department. 14,636 patients who sought medical attention for chest pain over a 2-year period were evaluated. 61% of patients had an initial hs-cTnT of <5 ng/L, 21% had 5–14 ng/L, and 18% had >14 ng/L. During 30 days of follow-up, only 33 (0.44%) of patients with undetectable hs-cTnT had an MI, of which 15 had no ischaemic ECG changes.

Comment (SM): Most units in New Zealand are now using a new generation of high-sensitivity troponin test (e.g. hs-cTnT) for patients presenting with chest pain. Although for safety, many algorithms recommend a second test after 3–6 hours, this study suggests that if the initial test yields an undetectable level (hs-cTnT <5 ng/L) and the ECG is normal then the risk of MI within 30 days was negligible and no deaths were recorded. Prognostication in such (or any other) circumstances can never be perfect but, assuming follow-up was indeed complete and preferably if the study is replicated, this is as near as it gets for permitting a rapid discharge policy, as long as it is combined with that unmeasurable but essential factor – clinical common sense.

Reference: Undetectable high sensitivity cardiac troponin T level in the emergency department and risk of myocardial infarction. J Am Coll Cardiol 2014; published online Mar 30
http://content.onlinejacc.org/article.aspx?articleId=1854323

The benefits of synchronised squeezing are extended and clarified

Authors: Goldenberg J et al.

Summary: This study evaluated the effect of cardiac-resynchronisation therapy with a defibrillator (CRT-D) on long-term survival in patients with left bundle-branch block (LBBB) who had participated in the MADIT-CRT trial. Post-trial follow-up over a median period of 5.6 years was assessed in 1 691 patients who survived MADIT-CRT (phase 1) and then in 854 patients who were enrolled in post-trial registries (phase 2). Seven years after initial enrollment, the cumulative rate of death from any cause among patients with LBBB was 18% in patients randomised to CRT-D and 23% in those assigned to defibrillator therapy alone (p<0.001). The long-term survival benefit of CRT-D in patients with LBBB did not differ significantly according to sex, cause of cardiomyopathy, or QRS duration. CRT-D was not associated with any clinical benefit and possibly with harm in patients without LBBB.

Comment (SM): Although a notable sceptic with regard to the benefits and cost-benefit ratios of new cardiac technologies, even I have to concede that cardiac resynchronisation therapy has some hugely beneficial effects in otherwise severely handicapped patients with advanced heart failure. This study suggests we should consider the therapy for all patients with even mild heart failure and LBBB. Patients with any other ECG finding did not benefit so the decision could now be regarded as simple and may not require any sophisticated echo analysis to back it up.


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De-innovation of renal denervation

Authors: Bhatt D et al., for the SYMPLICITY HTN-3 Investigators

Summary: SYMPLICITY HTN-3 evaluated the safety and efficacy of renal artery denervation in patients with severe resistant hypertension. 535 patients were randomised 2:1 to renal denervation or a sham procedure. Patients in the treatment group underwent renal artery denervation with the use of radiofrequency energy delivered by the Symplicity renal-denervation catheter. Office SBP decreased in both arms but the primary efficacy endpoint of difference in office SBP at 6 months did not differ significantly between groups. In conclusion, renal arterial denervation with the Symplicity denervation system was not superior to a sham procedure and medical therapy in reducing BP at 6 months in patients with severe resistant hypertension.

Comment (SM): This is indeed confession time as my scepticism antennae were not alerted sufficiently to ask major questions over the extraordinary and revolutionary results of SYMPLICITY 1 and 2. Last year, I had read and reviewed papers highlighting flaws in the methodology of these studies and predicting that results of a trial with better selection of patients and objective measurements of blood pressure would show a much less dramatic reduction of pressure. Even these authors might not have expected the effects of renal denervation reported in SYMPLICITY 3 to be so miniscule which has led me to feel some guilt over my own earlier lack of critical appraisal. The only slight comfort is the illustrious company this failing has kept with many academic reputations and financial investments suddenly coming under question. Perhaps then unsurprisingly, much of the subsequent discussion has focused on possible flaws in SYMPLICITY 3 (the main one being concern over the multitude of operators with little experience of the technique) rather than clear methodological deficiencies in 1 and 2. The technique may not be buried yet but some temperance of the enthusiasm is definitely required until more careful studies are completed.


INSTABILITY continues

Authors: The STABILITY Investigators

Summary: The STABILITY trial investigated the safety and efficacy of darapladib in patients with stable coronary heart disease. 15,828 patients were randomised 1:1 to treatment with darapladib 160 mg/day or matching placebo. Over a median follow-up of 3.7 years, the primary outcome of cardiovascular mortality, nonfatal MI, or nonfatal stroke did not differ significantly between groups.

Comment (SM): Another disappointingly negative trial here, especially sad as there were many enthusiastic participants in the STABILITY trial in New Zealand. The results were also confirmed subsequently by announced negative results of a second trial – SOLID-TIMI 52 which is yet to be presented. Darapladib is an anti-inflammatory drug that inhibits lipoprotein-associated phospholipase A2 (Lp-PLA2) thought to play a major role in causing instability of atheromatous plaques and there were very high hopes for therapeutic use of an inhibitor which seem to have been largely dashed. Another beautiful idea confounded by hard data.


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