

Cardiology Symposium

Thursday 31 October 2019

Chest pain – Dr Peter Currie

Q. Does the lack of epicardium coronary artery disease truly exclude the diagnosis of angina?

A. No, absolutely not. I think the diagnosis of microvascular angina is real and if the history is highly suggestive of angina then treatment should be offered. The finding of no CAD however, lets us give these patients extra reassurance as regards their outlook. Possibly avoid beta-blockers and use Dihydropyridine CCBs.

Q. SCOT HEART suggest a lot of Scots have CAD - many with non-cardiac pain who benefit from secondary prevention. Should we even bother with history / examination?

A. I believe that history is the key to everything. If they have angina they should get secondary prevention. If they have non-anginal pain then the decision would be for primary prevention or not which is a different question.

Q. Can we treat the chronic Diabetic patient the same way as they have high risk of silent ACS?

A. I believe that there are actually very few "silent infarctions". If you are able to take a really detailed history, you can usually identify a time when things were not right in some way. That is not to say that I don't believe that diabetic can present with odd symptoms. This why NICE tells us to consider risk factor profiles in our assessment and that does improve the sensitivity / specificity of our history

Q. Is there a danger of aspirin over prescription. Is a more accurate way of determining need for secondary prevention CTCA from primary care?

A. I think what I was trying to get over is how strong the history and examination is in predicting both coronary artery disease and making a confident diagnosis of angina or non anginal pain. If your history is highly suggestive of non anginal pain the real likelihood is that you patient does not have either angina or CAD. Thus should not be prescribed aspirin. CTCA was useful in getting patients treated earlier with appropriate medication but I would ask us to consider good history taking as a very powerful tool to identify these patients also.

Palpitation – Dr Chris Skene

Q. What's the evidence of anti-coagulation of AF in dialysis patients?

A. There is no randomised data but registry data suggests warfarin is better than no anticoagulation and apixaban is better than warfarin.

Q. How manage 'benign VEs' i.e. no structural disease. Would you echo everyone referred with what sound like ectopics? How many PVCs per ECG strip is associated with untoward effects? How do we manage patients who have presented with palpitations, ECG showing PVCs?

A. We perform at least a 24hr ECG and echocardiogram.

Then treat according to:

Symptoms

- beta-blocker or CCB
- ablation if an easy source (e.g. RVOT – LBBB, positive QRS in inferior leads)

Burden

if >10000 ectopics per day or 10%, perform cardiac MRI to exclude cardiomyopathy – looking for contrast enhancement in ventricular wall or evidence of ARVC

Q. What duration of device detected AF do you consider anticoagulation indication? When would you recommend anticoagulation for stroke prevention in patients with AF? Only if paroxysms last more than 24 hours? In a patient with an elevated CHA2DS2VASc score - how long would AF have to last on an ambulatory monitor for you to consider it sufficient to prompt anticoag?

A. We are generally using the ESC Guideline for AF 2016

Qualifying AF episodes for consideration for anticoagulation:

- 12-lead ECG – full duration of ECG (only 10 seconds, I know)
- Ambulatory monitor – 30 seconds
- Device-detected (loop recorder, pacemaker, ICD etc) – 6 minutes

Then assess according to CHA2DS2Vasc – all post stroke patients would score at least 2 and warrant consideration for anticoagulation if they had a qualifying AF event.

Q. is AF ablation safe? A study in JACC last week suggests mortality rate of almost 1 in 200 at 30 days. Do we monitor 30 day outcomes in the UK?

A. The JACC paper is interesting and frightening. This is a very high mortality risk compared to previously studied trials.

This can in part be explained by a changing trend for AF ablation.

The original cohort of patients we offered ablation were relatively young and free of comorbidities, with paroxysmal atrial fibrillation.

- This group had a high chance of successful ablation and a low risk of complications. Part of this was driven by the lack of data on rhythm control being a means of preventing death.

Q. Is the football player screening occurring for all athletes above 16? (Both male and female)

I believe all 16 year old players who are being offered a professional contract under the jurisdiction of the Football Association are screened – male and female.

- Therefore we could only justify the treatment if it was highly effective and unlikely to cause major complications. This population were studied in the CABANA trial where 2000 patients were split evenly between planned AF ablation and drug therapy by randomization. There were no early deaths and the observed mortality rates were around 5.5% in the drug therapy group and 4.5% in the ablation group after 4 years of follow up.

The new cohort of patients are older, have more comorbidities and particularly, heart failure.

- The CASTLE-AF trial looked at around 360 patients with heart failure and ICDs or CRT-Ds and atrial fibrillation. They were randomised to drug therapy or ablation. This trial was published in the NEJM and has shown a reduction in all the important outcomes, including death at 3 years (13% in the ablation group vs 25% in the drug therapy group).

Therefore, there has been a shift in the patients being offered ablation. Understandably, there will be an observed increase in the risk of death and of recurrence rates in the new cohort.

There is also a low operator volume effect and finally, there is probably a greater use of general anaesthesia in the USA compared to other countries around the world and this is known to enhance some of the worst risks of AF ablation, although it can give a higher success rate.

Therefore the true rate of ablation mortality is probably going to be higher in the group most likely to benefit.

Clinical cardiology: ask the experts? – Professor Stephen Leslie

Q. Regarding exercise, is the short term risk of MI increased in someone who takes little exercise currently? What exercise plan with regards intensity/frequency

A. I don't know if this is known - we do know that chronic exercise is associated with lower risk of MI, even 20 mins x 5 of walking per week. It seems that lack of sedentary behaviour is more important than absolute amount of physical activity so keep moving throughout the day seems important.

Q. Is there any evidence for aspirin in primary prevention?

A. Yes reduces MI and CV events but at the risk of bleeding so no overall net benefit in terms of mortality

Q. Why is the standard advice to give statins and not alter diet? Evidence shows that moving to a more vegetarian/plant based diet improves long-term outcomes

A. There are no good outcome trials for dietary intervention, but lots for statins.

Q. What if the patient disagrees and still wants to proceed with hip surgery because of quality of life?

A. not for cardiologist to say 'no' just to advise but doubt the surgeon and anaesthetist would go ahead - if they did the avoid spinal approach, watch fluid balance and cross fingers!

Q. What are your thoughts on a vegan diet for reducing cardiovascular risk? The game changer (Netflix) has advertised that it can reverse known CAD.

A. no good dietary intervention studies on CV outcomes

Q. What is evidence for PCI pre op to reduce risk of type 2 MI. Similarly, is there evidence that patients with type 2 MI have an angio for likely stenotic CAD

A. As far as I am aware there are no good trials that show reduced pre-op risk with PCI - indeed it will increase risk. If revasc is needed and surgery can be postponed then it is appropriate to do this. If surgery is urgent e.g. cancer and the patient does not have critical ischaemia e.g. rest pain or recent MI then I would have surgery first (preop decision making sometime helped by perfusion scanning) but generally base this on clinical need for surgery - if it can't wait then proceed but with caution - obviously avoid major surgery in immediate MI period or if pulmonary oedema or chest pain at rest

Q. Because of the possibility of outcome 2, would it have been better to have left the lesion alone?

A. I agree - I would not have stented this lesion.

Clinical cardiology: ask the experts? – Dr Ingibjorg Gudmundsdottir

Q. Regarding exercise, is the short term risk of MI increased in someone who takes little exercise currently? What exercise plan with regards intensity/frequency

A. Exercise is both an excellent marker of exercise tolerance and symptoms, in particular if there is a fairly sudden change. Exercise also increases overall cardiovascular mortality in the long run.

Q. Why is the standard advice to give statins and not alter diet? Evidence shows that moving to a more vegetarian/plant based diet improves long-term outcomes

A. It is a very good suggestion to advise a healthy diet, including vegetables, pulses etc. Refined carbohydrates are probably less beneficial.

Q. What if the patient disagrees and still wants to proceed with hip surgery because of quality of life?

A. A patient can not demand surgery that the surgeon/anaesthetist are not willing to provide because of risk. If the operating team is willing to offer surgery and the patient is informed and accepts the risk then that is fine.

Q. What is evidence for PCI pre op to reduce risk of type 2 MI. Similarly, is there evidence that patients with type 2 MI have an angio for likely stenotic CAD

A. This advice is based on recommendations regarding patients having lung transplant workup. Lungs for transplant are a limited resource and it is important that the patient is in as good a condition as possible, will survive the surgery and be haemodynamically stable in the process.

Q. Because of the possibility of outcome 2, would it have been better to have left the lesion alone?

A. Not really, the patient probably became too unwell to have the lung transplant.

The echocardiogram or the magnetic resonance imaging – Dr Nicola Johnston

Q. From a perspective of working up ischaemic stroke aetiology, does cardiac MRI add anything compared to TTE and, where relevant, TOE?

A. No, CMR is not useful for reliable identification of potential cardiac source of embolus. CMR is very good at identifying ventricular thrombus but less reliable and not routinely used for identification of atrial thrombus. It is also not the most sensitive test for identification of PFO and a combination of bubble TTE and TOE remains the best combination for this.

ANDREW RAE GILCHRIST LECTURE- Speaking to patients: ‘Words that maim, words that heal’ – Dr John Mandrola

Q. Should we give false reassurance to palliative patients that they will be ok to make them feel better “subjectively”?

A. No, we should not give false reassurance to palliative patients. Tho I object a bit to the modifier palliative for patients. Since we cure so rarely in medicine, nearly all our patients are palliative patients. That said, I assume the reader means hospice-like patients. In this case, I love the approach of Lakin and Jacebsen in JAMA

IM <https://jamanetwork.com/journals/jamainternalmedicine/article-abstract/2715161>

They write that a good way to approach these tough conversations is such: “I am hoping that you have a long time to live with your heart disease and I am also worried that the time may be short, as short as a few [years.]”

I often ask patients who are at end of life what they think is going on. Then. I ask if it’s ok for me to tell them what I think.

Q. How do we reconcile avoiding over diagnosis with the complaints culture?

A. This is a tough one. I am not familiar with the complaints culture term but I think I know what it means. Since I work in the real world of clinical care I am a pragmatist. Sometimes we have to do a scan, or a procedure. But I think it's less than we think. Doing a test or a procedure often placates people b/c of its caring signal: To wit, this doctor cares enough to order a scan or a monitor. But we can also put out huge caring signals by listening, making connection and promises to follow-up. I often counter the urge to over test and over treat with studies showing the harm of testing. For instance, 40% of scans of done for coronary calcium find incidental findings. I tell the story of a man who had a stroke from a Cath that was done to work up asymptomatic premature beats. There exists troves of data on the iatrogenic effects of medicine. People don't know about them—they often assume more is better. That can be countered with evidence, Sometimes tho, we all have to submit to pragmatism.

Q. How do we get around the nocebo effect when patients ask about potential drug side effects?

A. I have a slide on how to get around nocebo effects: I did not show it b./c of time constraints: Here it is:

Strategies to Reduce the Ethical Dilemma of Avoiding the Nocebo Effect

1. Focus on tolerability
 - o Use positive framing
 - o Positive frame: "the great majority of patients tolerate this treatment very well"
 - o Negative frame: "5% of patients report..."
2. Permitted Non-information
 - o Ask whether agrees to receive no info on mild or transient AE
 - o Of course severe AE must be shared
3. Patient Education:

Educational Interventions by Pharmacists to Patients With Chronic Pain
Systematic Review and Meta-analysis
Michael L. Bennett, MBChB, MD, FRCP,* Anne-Marie Bagnall, PhD,† Gary Raine, BSc,‡ S. José Class, PhD,‡ Alison Blenkinsopp, PhD,§ Andrew Dickman, BSc,|| and John Ellershaw, MD||
Bennett Clin J Pain 2011

Pharmacist-delivered Education
Meta-analysis of 4 RCTs

- Reduced pain
- Fewer reports of AE

● Hauser et al Dtsch Arztebl Int. 2012
● Colloca PsychSom Med 2011

<https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3401955/?report=classic> ●