South Island Division of Family Practice /Victoria Division of Family Practice
CARDIOLOGY WORLD CAFÉ
THURSDAY, JANUARY 22, 2015
THE PARKSIDE HOTEL/URBAN BALLROOM
6:00pm – 9:00pm

SPEAKER SUMMARIES/DISCUSSION POINTS:

Please click on the Following Links for Each Speaker's Summary

1. Dr Karan Shetty: Approach to Dyspnea
2. Dr. Laurence Sterns: Anticoagulation in Atrial Fibrillation Patients
3. Dr. Jennifer Rajala: Approach to the post MI Patient
4. Dr. Chris Lane: Syncope
5. Dr. Richard Leather: Holter Monitors: What's significant?
6. Dr. Imad Nadra: Approach to the Eldery with Aortic Stenosis
7. Dr. David Massel: Chest Pain
8. Dr. Elizabeth Swiggum: Approach to Heart Failure

1) Dr. Karan Shetty, M.D., F.R.C.P.C.

TOPIC: Approach to Dyspnea

Dyspnea is complex as any form of heart disease; electrical, muscles, valves arteries, can produce dyspnea. Major diagnoses are pulmonary disease, anemia, deconditioning. Cardiac dyspnea is distinguished by associated angina, orthopnea, and edema. It may be overlooked when there has been no antecedent cardiac risk factors or pre-existing disease. Common cardiac diagnoses causative are ischemic heart disease (sometimes silent angina), aortic stenosis, mitral regurgitation, congestive heart failure, cardiomyopathy, bradycardia and rapid atrial fibrillation. Symptoms at low level exertion often portend severe disease and limited survival. Treatments beyond correction of the underlying conditions involve anti-failure therapies utilizing a chronic disease management model.

2) Dr. Laurence Sterns, M.D., F.R.C.P.C.

TOPIC: Anticoagulation in Atrial Fibrillation Patients

Two main topics

- New Canadian Guidelines
- NOAC vs. warfarin

Canadian Guidelines

The new Canadian guidelines for anticoagulation in AF patients extend the number of patients recommended for anticoagulation. The main reason for this change is the safety of the new agents compared to the new and historic data on warfarin. This allows for a greater risk/benefit ratio in patients with lower risks of stroke. The recommendations are a modification of the CHADS2 risk calculator, with the age used as 65 rather than 75, and anticoagulation recommended for any patient with a score of 1 or more (thus any single risk factor).
The risk factors are:

- Congestive Heart Failure (low EF or preserved EF with CHF hospitalization
- Hypertension (history of, or treated)
- Age over 65
- Diabetes
- Stroke, TIA, or previous systemic embolization

NOAC vs. Warfarin

Despite the reticence of paying agencies to accept overwhelming data, all of the Novel Oral Anti-Coagulants (NOAC) have shown to be at least equivalent or superior to warfarin when looking at both safety and efficacy data. From a safety standpoint, all NOACs have less than half of the risk of intracranial bleeding of warfarin, have overall bleeding risks similar to or lower than warfarin, and mortality trends in the range of 10% less than warfarin treated patients. The stroke prevention data shows equivalent protection with rivaroxaban and low dose dabigatrin, with superior prevention with high dose dabigatrin and apixaban.

The Canadian Cardiovascular Society guidelines recommend use of the NOACs in preference to warfarin in most patients. Exceptions to this recommendation are in patients with valvular AF or prosthetic cardiac valves, in patients with severe kidney disease (GFR<30), or likely those with a history of severe GI bleeds. There is also probably little benefit to changing patients who have been stable on warfarin therapy to the new agents, as patients with stable INRs are less likely to have bleeding issues than those with unstable INRs. However, the suggestion of paying agencies to trial warfarin before moving to a NOAC is not as safe as initiating therapy with a NOAC because the risk of bleeding or stroke on warfarin is highest in the first several months of therapy. There is

also no clear consensus on patients who also require anti-platelet therapy for concurrent coronary artery disease. Numerous studies are underway on this patient group, with present recommendations being to consult with the specialists involved in the patient to make individual decisions.

Learning Objectives
To better understand

1) Atrial fibrillation 2015- who does not need anticoagulants (NOACs)
2) New anticoagulants preference over Coumadin
3) When can NOACs be harmful

3) Dr. Jennifer Rajala, M.D., F.R.C.P.C.

TOPIC: Approach to the post MI Patient

Atypical chest pain is common post MI.
Call the cardiologist if there is a potential need to stop antiplatelet medications early.
ACE Inhibitors provide mortality benefit. Beta blockers may not be of as much benefit in patients with normal LVEF >1 year post MI.

Learning Objectives:
To better understand

1) What type of chest pain post MI is worrisome and what is innocent?
2) When is ok to stop antiplatelet therapies?
3) What benefits do ACEI have?
4) In whom and when can beta blockers be stopped?

4) Dr. Chris Lane, BSC., M.D., F.R.C.P.C.

TOPIC: Syncope

Syncope has etiologies in the cardiac or neurologic systems. Cardiac structural lesions of inflow or outflow must be considered as well as bradycardia and tachy arrhythmias. Most syncopes are vasodepressor and this can usually be obtained through obtaining a detailed history.

Learning Objectives:
To better understand

1) What features of syncope suggest it is benign and what features would raise concerns that further work up is required?
2) What investigations could be considered for syncope and for which patients?
3) What is the utility of tilt table testing and when should it be done?
4) What driving restrictions result for a patient who has syncope?

5) Dr. Richard Leather, BSc., M.D., F.R.C.P.C.

TOPIC: Holter Monitors: what is significant?

Holter monitoring is often performed for a variety of reasons. Many, if not most, Holter monitors reveal arrhythmias, and it can be challenging to know which ones require investigations or treatment.
Concerning Holter findings which warrant follow-up are:

- Non-sustained VT x greater than 3 beats, >1000 PVCs in 24 hours,
- Episodes of Atrial fibrillation,
- SVT>30 seconds,
- Pauses > 5 seconds,
- Symptomatic pauses> 3 seconds,
- Mobitz 2 or greater heart block,
- Significant ST depression.

Learning Objectives:
To better understand

1) what patients may benefit from a holter monitor
2) what abnormalities may indicate heart disease vs physiologic limits

6) Dr. Imad Nadra, M.D., F.R.C.P.C.

TOPIC: Approach to the Elderly with Aortic Stenosis.

Aortic stenosis is very common in elderly people and symptoms of chest pain. Shortness of breath and presyncope may be underappreciated due to sedentary life.
Other co-morbidities may mimic these symptoms. The murmur may not be loud in late stage Aortic Stenosis.
TAVI is a good alternative for patients who have low frailty and are ineligible for conventional aortic valve replacement.

Learning Objectives:
To better understand

1) How the symptoms and signs of AS may be different in the elderly vs younger patients.
2) The role and limitations of transcutaneous aortic valve implant and surgical AVR.

7) Dr. David Massel, M.D., F.R.C.P.C.

TOPIC: Chest Pain

Chest pain presents a diagnostic challenge in outpatient family medicine. Non-cardiac causes are common, but it is important not to overlook serious of life-threatening conditions. In patients with symptoms suggestive of stable ischemic heart disease the probability of having obstructive coronary artery disease (CAD) is primarily obtained using a thorough history. Classically, angina is described as a dull retrosternal discomfort/ache/heaviness that

might radiate to the jaw, neck, shoulders or arms, is provoked by exertion or emotional stress, and is relieved within 5 minutes of rest or nitroglycerine use. Non-classical symptoms are common, particularly among diabetic patients and women.

All risk factors should also be considered.

- Non-modifiable: Age, Sex, Family history of premature established CV disease, and Ethnic origin
- Modifiable: Tobacco use/smoking history, Dyslipidemia, Diabetes, Hypertension, Chronic kidney disease, Physical inactivity, Diet, Obesity or metabolic syndrome, and Depression.

The physical examination has low sensitivity for the detection of CAD but abnormalities such as gallops, bruits or absent pulses (evidence of vascular disease), or obvious chest wall problems can be of help. Routine laboratory tests should be obtained to determine the presence and severity of factors that might influence angina (increase myocardial oxygen demand or decrease supply), choice of tests, or implementation of therapy such as CBC, Lytes, Urea, Cr, FBS, Lipid Profile, and TSH. A normal ECG does not exclude the diagnosis, but an abnormal resting ECG increases the probability and might influence the choice of diagnostic tests. Many tests are available for diagnosis and prognosis and should be individualized.

Learning Objectives:
To better understand

1) What is the usual history of angina in 2014
2) How does a vascular risk assessment help diagnose heart disease
3) What tests can be done in the family doctor's office to assess urgency
4) What tests do cardiologists do and when to make a final disposing plan

8) Dr. Elizabeth Swiggum, M.D., F.R.C.P.C.

TOPIC: Approach to Heart Failure

Heart failure has many clinical faces in the outpatient setting and making a diagnosis is key. Biomarkers and echocardiography are the mainstay of the most commonly used diagnostic tests and there are nuances to correctly interpreting the findings in an individual patient depending on age, BMI and renal function. Specifically heart failure with preserved ejection fraction is a new therapeutic challenge. Neurohumoral modulator medications (beta blockers, ACE or ARB, diuretics and mineralocorticoid antagonists) are the prime treatments but hypotension, renal perfusion and orthostasis are often limiting side effects.

Learning Objectives:
To better understand

1. Heart Failure: to BNP or to NT BNP? What's the difference?
2. Heart failure with preserved EF – targets and limits
3. Heart Failure with reduced EF – common causes you don't want to miss
4. Up titration of cardio protective medication and low BP – How low can I go?