ß-Blocker Use in Patients with CAD

1. Is there a role for using ß-blocker therapy in patients with coronary artery disease who have concomitant chronic, obstructive airway disease requiring ß-agonist inhaler therapy?

Question submitted by: Dr. Carla Milo, Atikokan, Ontario

The combination of heart failure and chronic obstructive pulmonary disease places the clinician between the proverbial rock and hard place, and it presents many diagnostic and therapeutic challenges. While robust clinical trial data is lacking, low-dose initiation and gradual up-titration of a cardioselective ß-blocker, such as bisoprolol, is currently recommended in patients suffering from both disease processes.[1] Although ß-agonist therapy can be safely used in select cardiac patients, clinicians should carefully consider the etiology of dyspnea and obtain objective evidence of airflow obstruction with pulmonary function testing before prescribing ß-agonists to patients with left ventricular dysfunction.

Reference
1. Hawkins NM, Petri MC, Macdonald MR, *et al*: Heart Failure and Chronic Obstructive Pulmonary Disease the Quandary of Beta-blockers and Beta-agonists. JACC 2011; 57(21):2127–2138.

Answered by:
Dr. Theodore K. Fenske

ACEi Induced Cough: A "Class Effect?"

2. Is an ACEi induced cough considered to be a "class effect" or Is it reasonable to consider alternative ACEi's?

Question submitted by: Anonymous

Approximately 20% of patients who receive angiotensin converting enzyme inhibitors (ACEi) develop a dry cough. The cough appears to be a class effect, so it is highly unlikely that switching to a different ACEi would result in resolution of the cough. Although resolution of the cough after ACEi withdrawal usually occurs within days, it may take weeks. ACE inhibitor cough may be associated with upper respiratory symptoms of rhinitis. Women, individuals with ACE genotype II, and those of black or Asian ethnicity have been reported to be at increased risk for ACEi cough.

Although the mechanism of ACEi cough was originally attributed to an elevation in bradykinin levels, there is some evidence that increased production of arachidonic acid metabolites and nitric oxide may also play a role.

If the cough is severe enough to require discontinuation of the ACEi, switching to an angiotensin receptor blocker is supported by current guidelines. However, a small percentage of patients also experience cough from ARB's.

Answered by:
Dr. Brett Heilbron

COX-2 Selective Inhibitor Therapy

3. What are the problems with using COX-2 selective inhibitor therapy on cardiovascular function?

Question submitted by: Anonymous

Concerns about the possibility of cardiovascular side effects in users of nonsteroidal anti-inflammatory drugs (NSAIDs) with cyclooxygenase-2 (COX-2) selectivity, like Celecoxib, have been raised since the withdrawal of rofecoxib in 2004 due to a documented increase in heart attack and stroke rates associated with long-term, high-dosage use. Through mechanisms related to prostaglandin inhibition, including sodium retention and vasoconstriction, all the NSAIDs, specific or not, have been shown to impair endothelial function, increase blood pressure, and reduce the efficacy of most antihypertensive medications.[1] This is of particular concern since NSAIDs are some of the most commonly used medications with a high prevalence of use among groups that are at risk for significant adverse drug-related events. In a recent survey, over half of people reporting NSAID use also had documented hypertension, and one in five people with CVD reported regular use of NSAID therapy.[2] While the risks of NSAIDs for CVD, hypertension, or renal disease may be regarded as acceptable by some clinicians or ignored by others, a dose of caution is recommended, particularly in those with established vascular disease. Selective COX-2 inhibitors in all dosages and nonselective NSAIDs in high-dosages have been shown to increase mortality in patients with previous MI.[3] Hence, if possible, patients who have documented hypertension and/or coronary artery disease should avoid the chronic intake of NSAIDs in general, and COX-2 inhibitors, in particular.

References
1. Dabu-Bondoc S, Franco S: Risk-benefit Perspectives in COX-2 Blockade. Curr Drug Saf 2008; 3(1):14–23.
2. Adams RJ, Appleton SL, Gill TK, *et al*: Cause for Concern in the Use of Non-steroidal Anti-inflammatory Medications in the Community — A Population-based Study. BMC Fam Pract 2011; 12:70.
3. Gislason GH, Jacobsen S, Rasmussen JN, *et al*: Risk of Death or Reinfarction Associated with the Use of Selective Cyclooxygenase-2 Inhibitors and Nonselective Nonsteroidal Antiinflammatory Drugs After Acute Myocardial Infarction. Circulation 2006;113(25):2906–2913.

Answered by:
Dr. Theodore K. Fenske

Which Omega-3 Prep is Recommended?

4. Which Omega-3 prep should be recommended?

Question submitted by: Dr. Maureen Conly, Vancouver, British Columbia

Most over the counter fish oil preparations contain about 300 mg of polyunsaturated omega-3 fatty acids (PUFA) per capsule in the form of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). In doses of 1,500 to 1,800 mg q.d., these PUFAs have reduced death rates in patients who have survived a recent myocardial infarction.[1] On the other hand, doses of 3 g q.d. seemed to increase the death rate in men with chronic angina.[2] There is no evidence that omega-3 supplements reduce or increase heart disease in the general population. It seems prudent to recommend regular consumption of fatty fish and avoidance supplements, at least in high doses.

References
1. Burr ML, Fehily AM, Gilbert JF, *et al*: Effects of Changes in Fat, Fish, and Fibre Intakes on Death and Myocardial Reinfarction: Diet and Reinfarction Trial (DART). Lancet 1989; 2(8666):757–761.
2. Raitt MH, Connor WE, Morris C, *et al*: Fish Oil Supplementation and Risk of Ventricular Tachycardia and Ventricular Fibrillation in Patients with Implantable Defibrillators: A Randomized Controlled Trial. JAMA 2005; 293(23):2884–2891.

Answered by:
Dr. Thomas W. Wilson

Spiral CT for CAD

5. Is there any value in spiral CT for coronary artery disease? Is the sensitivity/specificity better than stress testing?

Question submitted by: Dr. B.L. Chandrarajan, Kingston, Ontario

Very specialized spiral CT scans (thin-slice multi-detector row spiral computed tomography [MDCT]) are increasingly used for the detection of coronary artery disease. In a recent study, when compared to invasive coronary angiography, MDCT correctly identified about 75% of coronary lesions. About 12% of the arteries were unevaluable by MDCT, and, when these were excluded from the calculations, the sensitivity and specificity of MDCT was approximately 90 to 93%. The MDCT can be augmented by the injection of contrast media, and a 3D reconstruction of the coronary anatomy has been shown to be very accurate in detecting the disease and increasingly accurate in detecting the severity of the disease. The major risk from this is radiation exposure, but this risk is mediated by not being exposed to the potential complications of invasive coronary angiography. The next part of your question, "is the sensitivity/specificity better than stress testing?" is more difficult to answer. The stress test depends upon ECG changes that may develop when part of the heart is ischemic with exercise. The sensitivity and specificity of an ECG stress test really depends upon the population that is being tested. For example, a positive test in a 55-year-old male smoker has a very high sensitivity and specificity for coronary artery disease, especially if symptoms are present. On the other hand, for a 45-year-old female, even with the symptoms present, the sensitivity and specificity is in the 55 to 65% range.

The CT angiogram is certainly more specific than the stress test in detecting the presence of coronary artery disease, but in most cases, it requires a stress test to determine the significance of the disease and/or the need to proceed to coronary angiography. In addition, in patients with known coronary disease, or with symptoms suggestive of coronary disease, the duration of the stress test has important prognostic information. Especially in the symptomatic patient, I believe these two tests are complementary rather than competing.

In the asymptomatic population, most physicians would agree that the CT scan to detect the presence of coronary artery disease should not be done routinely. This is especially true in the over 70 population, where coronary calcification is almost the rule rather than the exception. If the screening stress test is abnormal, either because of ECG abnormalities, symptoms, or decrease in blood pressure, a CT angiogram could be the next step. In the absence of an abnormal stress test or symptoms, I would not recommend a CT scan for the detection of coronary artery disease.

Answered by:
Dr. Wayne Warnica