What we need to know about cardiac stress testing?

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Introduction

The left main coronary artery (dividing into left anterior descending and circumflex branches), and the right main coronary artery constitute the major vessels of the coronary circulation. The left and right coronary arteries originate at the base of the aorta from cavities called the coronary ostia. The coronary arteries, when functioning normally, ensure adequate oxygenation of the myocardium at all levels of cardiac activity. Constriction and dilation of the coronary arteries controls the amount of blood flow to the myocardium. This process can be disturbed by atherosclerotic processes causing an abnormal deposition of lipids, plaque formation and thickening of the vessel wall. These changes lead to a narrowing of the lumen which restricts blood flow and is termed coronary artery disease (CAD). Coronary artery disease causes changes in both structure and function of the blood vessels. This typically will result in patients experiencing angina (chest pain) caused by a difference between oxygen supply (decreased coronary blood flow) and oxygen demand (increased myocardial oxygen consumption). The decreased flow can result from chronic vessel narrowing (stenotic lesion), or from a blood clot (thrombus) Some of these changes can be determined from an electrocardiogram (ECG) that can detect changes to the rhythm and electrical activity of the heart**.** The ECG leads each record the electrical activity of the heart from a different perspective. This correlates to different anatomical areas of the heart thus helping identify areas of ischemia or myocardial infarction. [1]

Basic Principles of ECG

Contractions of the heart muscle during the cardiac cycle are triggered by an electrical impulse. This originates in a region of the right atrium near to the point of entry of the superior vena cave and is called the sino-atrial node, (SA node). This impulse spreads through the walls of both atria causing them to contract simultaneously, thus pushing blood into the ventricles. The impulse is then picked up by a second node, the atrio-ventricular node (AV node), situated in the right atrium near the intra atrial septum. [1]

From the AV node the impulse is conducted along a short bundle of conductive fibres known as the Bundle of His. This bundle quickly separates into two branches the Left and Right Bundle Branches. The impulse is conducted to the apex of the heart where the bundle branches turn upwards along the walls of the ventricles. They then pass through the ventricular myocardium along a finely branched network of Purkinje fibres, causing the ventricles to contract pushing blood out of the heart into the aorta and pulmonary artery. [1]

Electrical recovery of the myocardium from the impulse then follows along the same pathway, before the next impulse can be generated. This heart cycle is known as sinus rhythm. This is represented by the cardiac cycle consisting of the P wave, a QRS complex and a T wave, Diagram 1.

Diagram 1 represents the cardiac cycle

P wave corresponds to atrial depolarisation.

QRS complex corresponds to ventricular depolarisation.

T wave corresponds to repolarisation of the ventricles.

Overall changes to the sinus rhythm known as arrhythmias are commonly caused by heart disorders such as coronary artery disease, heart valve disorders and heart failure. Testing the heart during exercise can help identify coronary artery disease. In coronary artery disease, blood flow through the coronary arteries could be either partially or completely blocked. If the coronary arteries are only partially blocked, the heart may have an adequate blood supply when the person is resting but not when the person exercises, known as ischemic heart disease.

Stress testing

The patient will either walk on a treadmill (occasionally ride a fixed cycle) or will be given an intravenous (IV) pharmaceutical that simulates exercise while connected to an electrocardiogram (ECG) machine, usually with the standard 10 connections used to record a 12-lead ECG.

An intravenous cannula must be inserted and patency checked before starting the stress test,

irrespective of the protocol used.

From numerous clinical articles and guidelines, including the procedure guidelines adopted by the British nuclear medicine groups [2], stress testing with myocardial perfusion imaging is useful in a number of clinical situations, these include see table 1.

Table 1, adapted from indications for radionuclide myocardial perfusion imaging [2]

To assess the presence and degree of coronary obstruction.

To aid the management of patients with known coronary disease.

To determine the likelihood of future coronary events.

To guide strategies of myocardial revascularisation.

To assess the adequacy of percutaneous and surgical revascularisation.

To assess myocardial viability and hibernation.

Some patients with abnormal resting ECG, or those who are unable to walk safely on a treadmill or cycle, can be exercised pharmacologically. The patient will typically receive a pharmaceutical agent such as a vasodilator (dipyridamole or adenosine). Alternatively, the inotropic drug dobutamine can be used to simulate an exercise test.

For any stress procedure used, it is important that in the event of a cardiac or respiratory arrest or other complication requiring immediate medical assistance, the supervising stressor must initiate basic life support as appropriate and should call the emergency cardiac team.

Exercise Stress Testing

Unless medically contraindicated, prior to an exercise test patients should be withdrawn from medications that interfere with physiological exercise such as beta-adrenoceptor antagonists and calcium channel antagonists for 24-48 hours.

It is desirable that patients are also withdrawn from caffeine products prior to the test as this will allow the use of vasodilators (dipyridamole or adenosine) in case it is not possible to perform the exercise test.

Some of the contra-indications to low-risk exercise tolerance testing are listed in table 2. In general these contraindications also apply to pharmacological stress testing.

Table 2, adapted from contra indications [3]

Severe angina or worsening rest angina

Angina which is less than 1 month post MI, or post revascularisation

Known left main stem stenosis

Uncontrolled raised BP (Systolic BP >180mmHg and/or Diastolic BP>100mm Hg)

Hypotension (Systolic BP< 90mmHg)

History of sustained ventricular arrhythmias

Repolarisation abnormalities that prevent ST analysis, e.g.: left bundle branch block

Significant aortic stenosis

Hypertrophic obstructive cardiomyopathy

Treadmill testing is performed with either standard or modified Bruce protocol with 12 lead ECG monitoring during each minute of exercise and continuous monitoring of specific leads. Although with modern electronic equipment each lead can be cycled through for viewing independently and recording for future reference.

The level of exercise is generally increased in 3-minute stages of progressively increasing grade (% incline) and speed (mph, km/h). Throughout the exercise test the patients' clinical symptoms should be monitored continuously and blood pressure response measured every two minutes. Exercise end points are typically; physical exhaustion, development of severe angina, sustained ventricular arrhythmias or external hypotension, a specific list is included in table 3.

Table 3, modified from end points [3]

Technical difficulties monitoring the ECG or systolic blood pressure.

Ataxia, dizziness, or near syncope.

Signs of poor perfusion (cyanosis or pallor).

Exercise induced arrhythmias.

Rapid ST elevation with or without pain

Systolic Blood Pressure failing to increase >20mmHg.

>3mm ST depression without symptoms.

>2mm ST elevation with symptoms.

Severe chest pain or dyspnoea SBP rise >230mm Hg.

Heart rate falling >20% of starting rate.

Exercise should be symptom-limited with patients achieving at least 85% of the age and gender maximum predicted heart rate (MPHR). The radiopharmaceutical should be injected close to peak exercise and the patient should continue exercising if possible for one minute after injection of perfusion tracer. [2]

Perfusion defects can be missed or underestimated in patients with sub maximal exercise stress. Pharmacological stress testing should be used for patients who have been shown to be unable to achieve ≥ 85% MPHR from a previous exercise study. Similarly patients suspected of being unable to achieve ≥ 85% MPHR i.e. those who cannot exercise adequately for diagnostic purposes typically arthritic disease, lung disease, or are unable to withdraw from medication. Thus any physical limitation that prevents a patient from exercising maximally is an indication for vasodilator stress.

Pharmacological Stress Testing

Patients with significant hypotension (systolic blood pressure less than 90 mm Hg) should not be stressed with vasodilators. Caution is also required in selecting patients with reversible airways disease who may safely discontinue theophylline containing medication. Patients who have asthma and or inhalers are not generally contraindicated, but they may be at a higher risk of developing bronchospasm.

Patients must withdraw from taking caffeinated medications, food and beverages for 12-24 hours prior to test. This is because the effect of the vasodilator is abolished in the presence of caffeine since they compete for the same binding sites, leading to false negative results. [4]

Dipyridamole Testing

Dipyridamole (persantine) is a potent vasodilator that increases coronary blood flow velocity four to five times above normal baseline levels. It has minor systemic effects that include 4-10% decrease in systolic blood pressure and 20-40% reflex increase in heart rate. It can be administered intravenously or orally, the standard IV dose of dipyridamole is 0.56 mg/kg infused over 4 minutes. Peak coronary blood flow occurs 4-5 minutes after intravenous injection. Perfusion tracer should be injected at 7-8 minutes from the start of the dipyridamole infusion during peak response. The actions of dipyridamole can rapidly be reversed by an intravenous injection of 100-200mg of aminophylline. The main incidence of adverse reactions are angina, headache, dizziness, ST changes extra systoles, hypotension and nausea. Up to 50% of patients will experience some of these side effects. Chest pain or bronchospasm that is severe or sustained occurs in less than 1% of patients. It should be treated with intravenous aminophylline. A standard ECG and monitoring the patients vital signs should be performed, as with exercise stress testing, until the haemodynamic effects of dipyridamole have resolved, typically the test will last 12-15 minutes. [5]

Adenosine Testing

Adenosine is produced at an intra-cellular level but it does not exert its effects until it leaves the cell and interacts with the cell membrane. In coronary arteries, this interaction leads to vasodilatation and is used to increase myocardial perfusion. Intravenously injected adenosine has a very short half-life (2-10 sec.) and therefore it requires continuous infusion. The agent produces a slight increase in heart rate and a slight decrease in systolic and diastolic blood pressure.

It is generally infused at one hundred and forty micrograms per kilogram of body weight per minute (140µg/kg/min) as a continuous infusion for six minutes. The radiopharmaceutical is injected around the third minute of the infusion or sooner if the patient symptoms are distressing. Side effects related to adenosine are frequent, but usually self-limiting and of short duration. Discontinuation of infusion may be necessary if the effects are unbearable. Common side effects include flushing, hypotension, AV block, ST segment depression, and arrhythmia. If first degree AV block occurs the patient should be observed as a quarter of patients will progress to a higher degree of heart block. The test must be stopped immediately and medical advice sought. Intra venous aminophylline or theophylline has been used to terminate persistent side effects (50-125 mg by slow intravenous injection). [6]

Dobutamine Testing

Dobutamine is a beta adrenoceptor agonist that increases heart rate (positive chronotropic effect) and myocardial contractility (positive inotropic effect). It increases coronary blood flow between 2 to 3 times baseline which is less than that observed with dipyridamole or adenosine. [7]

Dobutamine can be used in patients that are unable to exercise and have contraindications to vasodilator stress such as severe reactive airway disease, high grade A-V block, or methylxanthine medication. The general contra indications to exercise testing apply to dobutamine infusion. Calcium channel blockers and beta-blockers should be discontinued for 24-48 hours prior to the examination if possible.

Dobutamine is given intravenously by a stepped infusion beginning at 10 ug/kg/min. and increased by 10 ug every 3 minutes to a maximum infusion of 40 ug/kg/min. The radiopharmaceutical tracer is injected one minute after the final increase and the infusion is continued for an additional 2 minutes. If the patient reaches a satisfactory haemodynamic state or develops untoward side effects, the injection of the radiotracer may be made sooner. The infusion should be terminated if the patient develops a ventricular tachycardia or ST segment elevation. If the heart rate has not doubled with dobutamine by seven and a half minutes into the infusion, it is unlikely to reach 85% of the MPHR. Atropine (0.6 mg) may be injected intravenously to increase the heart rate above 85% of MPHR. Potential side effects include sinus tachycardia or atrial tachyarrhythmia. A beta blocker can be given IV to reverse the effects of atropine and dobutamine. [7]

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