

Original Article: Anatomy and Physiology of Coronary Arteries


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ABSTRACT

When a coronary artery narrows or closes, the area of the heart through which the artery is lubricated becomes ischemic and damaged, and a heart attack may occur. Oxygen is directly related to heart activity. The more active the heart, the greater the need for oxygen, and the coronary artery blood flow is adjusted according to the heart muscle's need for oxygen. The heart muscle consumes about 65% of the oxygen in the coronary artery, while other tissues in the body consume a maximum of 25% of the oxygen in the blood of the coronary artery. Also, unlike other tissues in the body, 75% of the heart muscle blood is supplied at rest by diastole of the heart. The coronary arteries are responsible for supplying blood to the heart muscle. The right and left coronary arteries branch from the aorta just above the aortic valve, then enter the heart and supply blood to the capillaries of the heart muscle. The two grooves meet at the posterior region of the heart in a place called the CRUX, where the AV group is located. If the RCA supplies blood to the cortex, these people are called the dominant right. The descending RCA branch intoxicates the left posterior muscle. Approximately 18% of people with CCA and RCA donate blood to the heart crocus, in which case it is called a balanced arterial pattern.

Introduction

It originates from the right aortic sinus and moves in the right posterior ventricular atrial groove [1-3]. Its branches drink into the ventricles and in more than 50% of people SA and in 67% of people it supplies blood to the heart cortex. (The cortex consists of the junction of two ventricular atrial grooves and the interventricular groove) [5-7].

Left coronary artery (LCA)

The left coronary artery, after branching from the aorta, forms a short part called the left main stem and in the distal part is divided into two descending anterior left branches and the circumflex artery [8-10]. The left ventricular myocardial (LAD) branch also supplies blood to parts of the right ventricle, the anterior apex, and part of the posterior apex. CA and its

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branches supply blood to most of the left atrium, the lateral wall of the left ventricle, and part of the posterior wall of the left ventricle [11-13]. The AV node in 10 to 15% of people receives blood through the LCX. These people are called the dominant left [14-16].

Lateral blood flow

Lateral arteries are arteries between arteries that can connect two branches of the coronary artery [17-19]. These arteries are present from birth, but not until the heart muscle is deprived of oxygen or anemia. It does not play a significant role in the blood supply to the heart muscle [20-22]. The lateral arteries are very thin and narrow because they are devoid of smooth muscle and are unable to respond to vasoactive or metabolic factors. The three main factors for stimulation and growth of lateral arteries are: coronary artery disease [23-25], chronic myocardial hypoxia, and myocardial hypertrophy. Once the lateral arteries are stimulated, their diameter increases and a layer of smooth muscle forms in them. Eventually they become ontologically similar to arteries. If they are fully developed, they will expand under the administration of nitrates [26-28]. The time required for the lateral arteries to develop in anemic areas of the heart muscle is less than 9 days [29].

Note: With increasing age and progression of sclerotic lesions, these lateral arteries dilate, and therefore myocardial infarction is associated with lower mortality in the elderly than in the young [30].

The constituent layers of the coronary artery are

A) The intima layer: The intima layer is a single continuous layer of epithelial cells that covers the inner surface of the duct of all arteries. Endothelial cells naturally form a barrier that controls the entry of substances from the blood

into the arterial wall [31-33]. These materials usually enter the cell through dedicated transmission systems. Endothelial cells secrete substances that affect blood clotting and contraction and relaxation of the vascular smooth muscle adjacent to them [34].

Naturally, there are no other cells in the intima of most arteries. It consists of only one type of smooth muscle cell, which is located in one layer or multiple layers [35-37]. In addition to smooth muscle cells, the most important cells that make up connective tissue are the arterial wall, which produces elastic collagen fibers and peregoglycans [38-40]. The media is bounded on the inside by the elastic layer and on the outside by the external elastic [41].

B) Media layer: Media is an elastic and muscular covering that has more elastic tissue in the aorta and large arteries, but has more muscle tissue in the small arteries and arteries [42-44]. In the medial part of most arteries there is a feeding area, the outer part is fed by small blood vessels located in the adventitia. The inner layers receive nutrients from the vein [45].

C) Adventitis: Adventitis is the outermost layer of the artery. This coating contains an intricate network of weak collagen bundles, elastic fibers, smooth muscle cells, and fibroblasts. There are small blood vessels in this layer of nerves and vasovasum [46].

Pulmonary artery catheter (Ganz catheter)

It has four ducts. They enter the superior or inferior vena cava through a small incision. From there, they lead to the right atrium and through the tricuspid valve into the right ventricle, and from there [47-49], at the same time as the pulmonary valve opens, they lead to the pulmonary artery. The catheter is attached to the monitor from the outside and draws pressure based on the location of the catheter (capillary bed [50-52], pulmonary artery, or

right ventricle). The recorded pressure can be related to systolic pressure of the pulmonary artery, diastolic pulmonary artery, middle pulmonary artery [53-55], while the balloon of the catheter head is inflated to cause the catheter head to get stuck in the end branch of the pulmonary artery, the mentioned pressure is measured [56-58]. The balloon should not be inflated for more than a few seconds, as this will damage the pulmonary circulation. After the balloon is deflated, the catheter is sent forward. In this case, the catheter enters the pulmonary capillaries and inflates again, measuring the pressure at the pulmonary capillaries [59].

Complications of pulmonary pressure control

Cardiac arrhythmias, venous spasm, incision infection, perforation of the heart and rarely cardiac arrest, pulmonary infarction embolism, arterial rupture, pulmonary thromboembolism.

Note: Usually, critically ill patients need more ventricular filling pressure to reach the desired cardiac output. These patients may need to maintain a wedge pressure of 1-2 mmHg [60].

Nursing care of the patient with Swan Ganz catheter

After placing the catheter, the skin surface is sutured, and a chest radiograph is taken to ensure replacement. To control the incidence of pneumothorax and catheter contraction in a cavity, radiographic imaging of the chest is essential [61-63]. The dressing on the catheter entry site should be changed daily. Control the dressing site for redness, inflammation and drainage. Use betadine solution or alcohol in the dressing. Keep the dressing dry [64-66]. If the patient has ventricular fibrillation and needs a defibrillator, the transducer must first be removed from the catheter, as the electric shock will damage the transducer [67].

Catheter removal should be done slowly to prevent right ventricular stimulation and

ventricular tachycardia. After removal, the catheter head should be sent to the laboratory for culture and the incision site should be bandaged with betadine and dry gas [68].

Measurement of cardiac output

Using the Swan Ganz catheter head, the cardiac output power was also examined. Normal saline solution or 5% sugar serum at room temperature is injected into the right atrium. This solution mixes with the patient's blood and due to its cold relative to the patient's blood temperature [69-71], the patient's blood temperature decreases at any time. Blood temperature changes are measured by a transducer at the tip of the catheter in the pulmonary artery [72-74]. Therefore, based on this, the blood temperature will change and show a different temperature than the heart measurements. The amount of output is measured by the degree and speed of blood changes [75].

Direct measurement of arterial blood pressure

This method is used for direct measurement of blood pressure in critically ill patients with severe hemodynamic disorders. In this procedure, a catheter is inserted into the patient's artery [76-78]. Lateral blood sampling should be performed before catheter placement with an Allen test or Doppler test. If the lateral circulation is not sufficient, the artery becomes blocked by passing a catheter, the end perfusion is defective, and there is a risk of ischemia and myocardial infarction [79-81]. The catheter is connected from the outside to a three-way from there to the transducer. To prevent the risk of clot formation, heparin serum is attached to the catheter [82-84]. Be sure to mention on the arterial catheter that the catheter is inserted into the artery and to prevent any injection into it. The transducer is sensitive to the blood pressure in front of it and displays the measured pressure in millimeters of mercury on the monitor screen.

Waves from normal arterial pressure include an ascending slope along the systole followed by a negative fall along the diastole. Intra-arterial blood pressure monitoring is to continuously evaluate the perfusion of arterial blood to vital organs of the body [85-87].

Note: The mean (mean) arterial pressure of MAP is a clinically important parameter for tissue perfusion [88].

MAP represents the average perfusion pressure throughout the cardiac cycle. Since one-third of the cardiac cycle is during systole and two-thirds is during diastole, the formula for Calculating

MAP is as follows: (systolic pressure) + (Diastole × 2)/3.

MAP maintenance greater than 60 mm Hg is essential for adequate tissue perfusion of the coronary arteries, brain, and kidneys. MAP between 70 and 90 mmHg is ideal. During cardiac output, the body maintains blood pressure by contracting peripheral arteries. Here the MAP drop may remain constant, but the pulse pressure (difference between systole and diastole) is low [89].

Complications

Local obstruction is accompanied by ischemia of the post-stenosis area. Cardiac catheterization and angiography are still the definitive and standard methods of examining the anatomy and physiology of the cardiovascular system. Cardiac catheterization and angiography are performed to diagnose therapeutic intervention or both. Cardiac catheterization is recommended when evidence of a suspected clinical condition, severity of anatomy, or physiology is required. Especially when the patient has increasing symptoms of heart failure or other tests such as exercise testing or echocardiography have shown that the client may rapidly worsen, myocardial infarction or

other complications are imminent [90]. It is the only method that accurately determines the anatomy of the coronary arteries and the decision to perform coronary artery surgery or balloon angioplasty. In the case of cardiomyopathy or valvular disease, hemodynamic studies can be performed to begin appropriate treatment. Relative contraindications for catheterization and angiography of the heart include uncontrolled ventricular irritability, hyperkalemia, digital poisoning, hypertension, concomitant infectious and febrile illnesses, irreversible heart failure, increased PT time of more than 18 seconds, severe contrast allergy, and severe renal failure or anuria.

Right heart catheterization

Right heart catheterization is usually performed before left heart catheterization. This operation is performed under fluoroscopy using a balloon catheter. A catheter is inserted through a vein (thigh, arm, clavicle, or internal jugular vein) into the superior vena cava, which can be used to measure blood pressure in the arteries and blood vessels, and also to take a blood sample to measure the percentage of oxygen saturation. With this method, blood pressure and oxygen saturation can be measured in the right atrium, right ventricle, pulmonary artery, capillary network of the lungs.

Possible side effects of right heart catheterization

Dysrhythmia, venous spasm, catheter entry infection, perforation of the heart, and rarely cardiac arrest are some of the complications of this procedure. Fatal arrhythmias may occur when catheters enter the heart [91].

Left heart catheterization

In order to evaluate the capacity of coronary arteries, function is performed in the left ventricle and mitral and aortic valves.

Possible complications of left heart catheterization

Dysrhythmia, myocardial infarction, perforation of the heart wall or large arteries, and systemic arterial embolism. In this procedure, the doctor usually inserts a catheter into the right arm or femoral artery and directs it to the ascending aorta and left ventricle. Left heart catheterization is performed retrograde, meaning that the catheter is inserted into the aorta and pushed from the aorta into the left ventricle.

Increased levels of cortisol and catecholamines

Involved in the development of HTN and atherosclerosis.

Catecholamine: It has known effects on platelet aggregation and fat metabolism. Cortisol increases the sensitivity of arterioles to catecholamines.

Other contributing factors:

- ✓ Avoid coffee and sucrose.
- ✓ Deficiency of vitamins C, E, calcium, magnesium, chromium, manganese, lithium, fluorine.
- ✓ Vitamin deficiency.
- ✓ Urbanization.
- ✓ High hematocrit and increased sodium ESR.
- ✓ Abnormal metabolism of the amino acid cysteine.

Note: If there is a risk factor in a person, the person is not twice as likely to be at risk, but is twice as likely to be affected by the algebraic sum. For example, smoking increases the chance of developing CAD by 3 to 5 times, and high cholesterol increases the risk of CAD by 3 to 5 times. If these two factors are combined, the chances of getting it are quadrupled.

Calcium channel blockers

Note: Calcium channel blockers are used to prevent and treat vascular spasms caused by invasive venous procedures.

Amylodipine and Fludipine: Amylodipine and fludipine are calcium channel blockers that can also be used in congestive heart failure.

Antiplatelet and anticoagulants: Antiplatelet drugs prevent platelet aggregation, which causes blood to flow. Even low-dose aspirin has anticoagulant properties and prevents platelet adhesion. In some cases where there is a risk of gastrointestinal bleeding, ticlopidine is preferred. Drugs such as Plavix are currently recommended to reduce the risk of platelet adhesions.

Oxygen therapy: If the patient develops angina pain and rapid breathing or low blood oxygen concentration, the nurse should begin oxygen therapy. Oxygen is usually administered through a nasal catheter at a rate of 2 liters per minute, even in cases where the patient has no respiratory problems.

Nursing diagnoses in patients with angina

Impaired myocardial infarction secondary to CAD in the form of chest pain.

Anxiety associated with fear of death.

Lack of knowledge about the nature of the underlying disease and methods of preventing complications.

Failure to follow the treatment plan due to not accepting the need for lifestyle changes.

Clinical manifestations

The first sign is usually pain in the front of the chest that spreads to the neck, left and right hands, back and lower jaw. This pain can occur suddenly or without activity during activity. It is so severe that patients often call it the worst

pain of their lifetime that does not go away with rest. It lasted much longer than unstable angina pain. If the pain persists for more than 15 minutes, the possibility of a heart attack should be suspected, and sometimes the pain starts only in the epigastric region, but its severity and duration are much greater than other areas of pain. Cold sweats are usually accompanied by pain on the forehead, limbs and palms, and the cause of this phenomenon is the secretion of catecholamines, which causes narrowing of blood vessels and secretion of sweat glands. Parasympathetic activates catecholamine secretion and induces vomiting.

Note: Any person over the age of 35, especially a man who presents with epigastric pain, cold sweat vomiting, and the first diagnosis should be considered for a heart attack. The patient is usually restless, punching his right hand and squeezing his left chest (Levin sign). They say something is exploding inside his chest. Painless infarction is seen in the elderly and diabetics, and some patients have transient hypertension at the beginning of MI and up to cardiac output with normal output. If MI is uncomplicated, stroke volume and output are normal. If hypotension and shock are seen during myocardial infarction, the volume of stroke, cardiac output, and systolic pressure decreases and left ventricular capacity decreases.

Note: Inferior infarction occurs due to vagal stimulation in 1.2 patients with bradycardia and hypotension. Anterior infarction is due to sympathetic stimulation and occurs in 1.4 patients with tachycardia and hypertension.

Conclusion

Cardiovascular and chest pain, palpitations, heart sounds in the form of weakening of the first sound, the presence of the third and four S sounds, and the doubling and reversal of the S sound and sometimes the intensification of the

S1 sound. Increased dilation of the jugular vein may be seen if MI causes heart failure. Hypertension due to sympathetic stimulation or hypotension due to decreased contractility, cardiogenic shock, or medications. Pulse abnormalities that may indicate atrial fibrillation and changes in the ST and ECG may indicate tachycardia and bradycardia. Respiratory Shortness of breath, shortness of breath, increased respiratory rate, real If MI causes pulmonary congestion and pulmonary edema may be present, and nausea and vomiting in the intestinal tract, decreased urinary output in genitourinary urine may indicate cardiogenic shock. Cold and damp skin, sweating, pale appearance due to sympathetic stimulation due to lack of contraction indicating cardiogenic shock. In the nervous system, anxiety, restlessness, and lightheadedness may indicate increased sympathetic stimulation and oxygen delivery to the brain. These symptoms also indicate cardiogenic shock.

Note: If the patient is receiving thrombolytic MI for treatment, has visual disturbances, changes in speech, motor function, and further changes in the level of consciousness, indicating cerebral hemorrhage.

The cycle of long-term effects of myocardial ischemia causes fever, and the fever occurs due to the release of the heart's intracellular material into the bloodstream and the reaction to it. The duration of the fever is important, the severity is not important. If fever occurs after the second week of a heart attack, it may be related to Dresler syndrome. Non-specific laboratory results of leukocytosis appear within a few hours after the onset of pain. The WBC has reached 15,000 and lasts for about 7-7 days. High sodium ESR is elevated within the first week after MI and remains high for several weeks (fever occurs on the second and third three days after myocardial infarction due to the release of cardiac intracellular substances into

the bloodstream and the reaction to these substances).

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