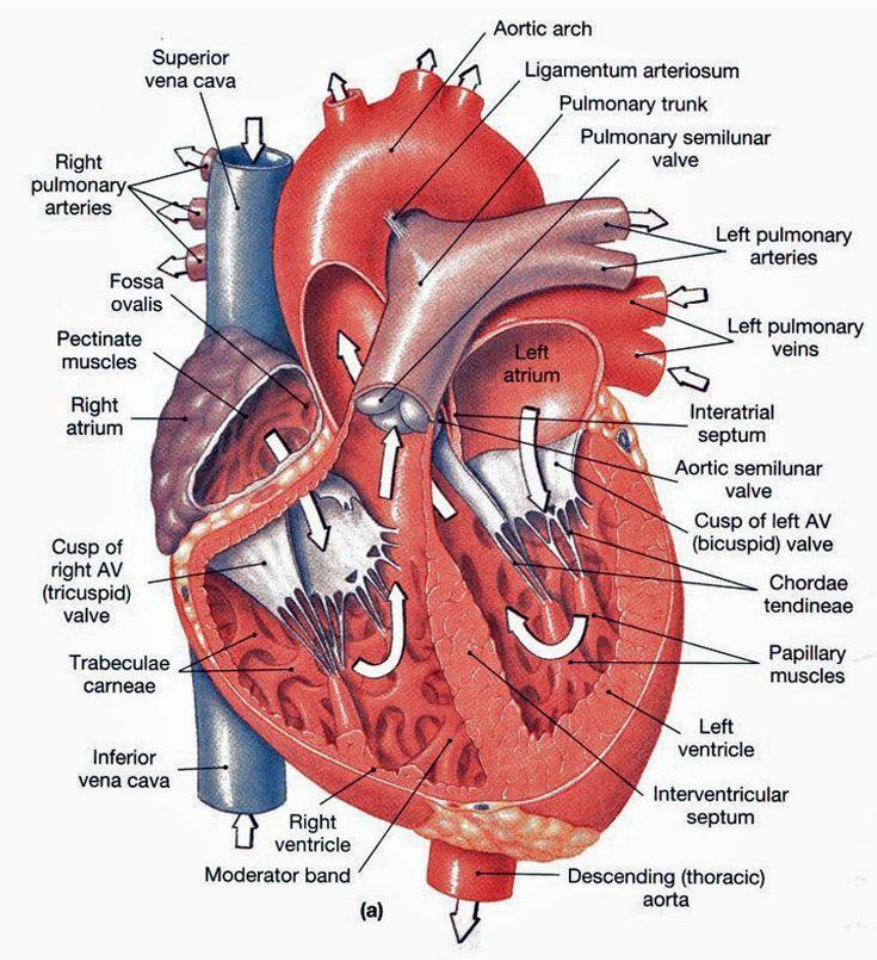
CHAPTER 2

THEORETICAL BACKGROUND

2.1 Basic Concept of Heart Failure



2.1.1 Anatomy and Physiology of Heart

Image 1. Anatomy of Heart

The heart are muscular, cone-shaped, hollow, upper-based organ, and its peak is below. Apex (top) tilted to the left. Heart weight is approximately 300 grams (Evelyn C. Pearce, 2009). The heart is a hollow muscular organ, resembling a pyramid or banana heart that is

the center of the blood circulation throughout the body, located in the thoracic cavity in the mediastinum section. The tip of the heart leads downward, to the front of the left. The base of the heart points upwards, backward, and slightly to the right. On the base of the heart there is aorta, pulmonary artery, upper and lower vein and lung vein (Syaifuddin, 2012).

The heart is an organ that functions to pump blood to meet the needs of oxygen supply for the entire network (Udjianti, 2010). The heart is a large pump organ that maintains circulation through the whole body (Evelyn C. Pearce, 2009). The cardiovascular system is a closed transport system consisting of: the heart as the pumper, the blood component as the carrier of oxygen and nutrients, the blood vessels as the medium that drains the blood component (Muttaqin, 2012).

2.1.1.1 The location of the heart

The heart is in the thorax, between the two lungs and behind the sternum, and more facing left rather than to the right.His precise position can be drawn on the skin of our breasts (Evelyn C. Pearce, 2009).

A pulled line pulls out of the third right ribcage cartilage, 2 centimeters from the sternum, up to the second left rib cage, 1 centimeter from the sterneum, pointing at the base of the heart, where the blood vessels enter and exit. The point to the left between the fifth and sixth ribs, or in the left five intercostal space, 4 centimeters from the medial line, indicates the position of the cardiac apex, which is the sharp end of the ventricle (Evelyn C. Pearce, 2009)

2.1.1.2 Cardiac Structure

The heart is an organ consisting of muscles. Cardiac muscle is a special tissue because when viewed from the shape and the structure is the same as the latitude, but the way it works resembles smooth muscle that is beyond our will (influenced by the autonomic nervous system) (Syaifuddin, 2012).

The heart consists of four chambers, two thin-walled chambers called the atrium (porch) and two thick-walled chambers called the ventricles (Muttaqin, 2012). It is divided by a septum (bulkhead so that it becomes two halves, left and right). Each ventricle has one valve in and out of the valve. The tricuspid valve opens from the right atrium into the ventricle, and the pulmonary valve opens from the right ventricle into the pulmonary artery. The mitral valve opens from the left atrium into the left ventricle, and the aortic valve opens from the left ventricle into the aorta (Kasron, 2016).

The heart consists of three major types of cardiac muscle, namely atrial muscle, ventricular muscle, and excitatory muscle fibers and special conduction. Atrial and ventricular muscle types contract in the same way as skeletal muscle, only the duration of contraction of these muscles is longer. In contrast, excitatoric and conductive specialty fibers contract very weakly because these fibers contain only a small amount they exhibit automatic rhythmic electrical discharges in the form of action potential or potential action conduction through the heart, acting as an excitatory system that regulates rhythmic heartbeats (Guyton & Hall, 2012).

The heart is composed of muscles that are special and wrapped in a membrane called the pericardium. This membrane consists of two layers, the visceral pericardium is a tight membrane attached to the heart and the parietal pericardium is a layer of fibrus folded out of the base of the heart and encloses the heart as a loose bag. Because of this arrangement, the heart is in two layers of pericardium pouch, and oiled from the liquid, the heart can move freely (Evelyn C. Pearce, 2009).

Inside the heart is covered in endothelium. This layer is called endocardium. The valves are only a thicker part of this membrane. The thickness of the heart wall is described as being composed of three layers; Pericardium, or outer wrap; Myocardium, the middle muscle layer; Endocardium, deep boundary (Evelyn C. Pearce, 2009).

The heart muscle wall is not the same thickness. The thickest ventricular wall and the left wall are thicker and the left ventricle wall is thicker than the right ventricle wall, because the left ventricular contraction force is much larger than the right. The atrium wall is composed of thinner muscles (Evelyn C. Pearce, 2009).

The physiological anatomy of the heart muscle represents a typical histological feature of the cardiac muscle, which shows its separate fibers, rejoins, and spreads again. The heart muscle is striated in a pattern similar to the pattern found in a typical skeletal muscle. Furthermore, the heart muscle has certain myofibrils that contain actin and myosin filaments, which are almost identical to the filaments found in skeletal muscle; during the contraction of these filaments lie side by side and insert each

other against one another as happens in skeletal muscle (Guyton & Hall, 2012).

Cardiac muscle as a syncytium, visible dark areas that cross the heart muscle fibers called interclatus disks, but the interclatus disc is actually a cell membrane that separates each of the heart muscle cells from each other. Thus, the heart muscle fibers consist of many interconnected cardiac muscle cells and lie side by side in a series (Guyton & Hall, 2012).

2.1.1.3 Circulatory and cardiac nerves

The right coronary artery comes from the anterior sinus of the aorta running forward between the pulmonary trunk and the right auricle giving branches to the right atrium and the right ventricle. On the inferior edge of the heart to the antrioventricular sulcus for anastomosis with the left coronary artery ventricating the right ventricle. The left coronary artery is larger than the right coronary artery of the posterior sinuses of the left aorta sinistra running forward between the pulmonary trunk and the left auricle into the atrioventricular sulcus leading to the apex of the heart providing blood for the right ventricle and the interventricular septum (Syaifuddin, 2012).

Heart vein flow: Part of the blood from the heart wall flowing into the right atrium through the coronary sinus located at the back of the atrioventricular sulcus is a continuation of the magna cardiac vein that empties into the right atrium to the left of the inferior vena cava. cardiac vein minimae and media are coronary sinus branches, the rest return to the right atrium through the anterior cardiac vein, through the small vein directly into the heart chambers (Syaifuddin, 2012).

Superior and inferior cava veins pour their blood into the right atrium. The inferior vena cava vein is guarded by Eustachius seminular valve. The pulmonary artery carries blood out of the right ventricle. Four pulmonary veins carry blood from the lungs to the left atrium. The aorta carries blood out of the left ventricle (Evelyn C. Pearce, 2009).

The aortic opening and pulmonary artery are kept seminular valves. The valve between the left ventricle and the aorta is called the aortic valve, which prevents blood from flowing back from the aorta to the left ventricle. The valve between the right ventricle and the pulmonary artery that avoids blood flows back into the right ventricle (Evelyn C. Pearce, 2009).

After blood has passed through capillary networks in the myocardium, it enters a series of cardiac veins before draining into the right atrium through a common venous channel called the coronary sinus. Several veins that collect blood from a small area of the right ventricle do not end in the coronary sinus but instead drain directly into the right atrium. As a rule, the cardiac veins follow a course the closely parallels that of the coronary arteries (Patton & Thibodeau, 2010).

The right and left coronary arteries first leave the aorta and then branch off into smaller arteries. These small arteries surround the heart and deliver blood to all parts of this organ. Blood returning from the heart is primarily collected coronary sinus and straight back into the right atrium (Evelyn C. Pearce, 2009).

The heart is fed by the sympathetic nerve fibers, the parasympathetic, and the autonomic nervous system through the cardiac flexus. Nerve sympathetic derived from the cervical duct of the cervical, thoracic, but upper part of the sympathetic nerves from the vagus nerve. The afferent post ganglion fibers travel to the atrial sinus node and the atrioventricular node that is spread to other parts of the heart. Afferent fibers run with the vagus nerve and act as cardiovascular reflex that runs along with the sympathetic nerves (Syaifuddin, 2012).

2.1.1.4 Cardiac Cycle

Events that occur in the heart begin from the beginning of a heartbeat until the onset of the next heartbeat is called the heart cycle. Each cycle begins with the formation of spontaneous action potentials in the sinus node. This node is located on the superior lateral wall of the right atrium near the incoming superior vena cava, and the action potential propagates from here at high speed through both atria and then through the A-V bundle to the ventricle (Guyton & Hall, 2012).

Because there is a special arrangement in the conduction system from the atria to the ventricle, a delay is found for more than 0.1 second when the cardiac impulse is delivered from the atrium to the ventricle. This condition causes the atria to contract preceding ventricular contraction, thus pumping blood into the ventricle prior to strong ventricular contraction. Thus, the atrium acts as a precursor pump for the ventricle, and the ventricle will then provide the main source of power to pump blood into the vascular system (Guyton & Hall, 2012).

This wave of contraction moves through the file His then the ventricle contracts. Heart movement consists of two types, namely contraction or systole, and relaxation or diastole. The contraction of the two atria occurs simultaneously and is called the atrial systole, the laxity being the atrial diastole. Similarly, ventricular contraction and relaxation are also called systole and ventricular diastole. The duration of ventricular contraction was 0.3 s and the relaxation stage was 0.5 s. In this way the heart beats constantly, day and night, during his life. And the heart muscle gets rested during ventricular diastole (Evelyn C. Pearce, 2009).

The contractions of the two atria are short, while the ventricular contractions are longer and stronger. And that of the left ventricle is the strongest because it has to push blood throughout the body to maintain blood pressure throughout the body to maintain systemic arterial blood pressure. Although the right ventricle also pumps the same blood volume, but its task only sends it around the lungs where its pressure is much lower (Evelyn C. Pearce, 2009).

According Udjianti (2010) the heart cycle has two phases of diastolic phase and systolic phase. During the diastolic phase the right ventricle is filled with blood from the right antrium while the left ventricle is filled with blood from the pulmonary vein. In the systolic phase the blood in the right ventricle is pumped into the pulmonary artery toward the pulmonary capillaries for the process of oxygenation. Meanwhile blood from the left ventricle is pumped and distributed throughout the body to help tissue metabolism.

2.1.1.5 Blood Circulation

The function of the circulation is to satisfy the body's tissue needs for transporting nutrients to body tissues to transport useless products to deliver hormones from one part of the body to another, and in general, to maintain an appropriate environment within the entire tissue fluid the body in order for cells to survive and function optimally (Guyton & Hall, 2012).

The velocity of blood flow through most tissues is controlled by the response of the tissue requirements to the nutrients. The heart and circulation are then controlled to meet the cardiac output and the corresponding arterial pressure in order for the flow of blood flowing in the tissues to correspond to the required amount (Guyton & Hall, 2012).

Evelyn C. Pearce (2009) The heart is the main organ of blood circulation. The flow of blood from the left ventricle through the arteries, arterioles, and capillaries back to the right atrium through the vein is called large circulatory or systemic circulation. The flow from the right ventricle, through the lungs to the left atrium is a small blood circulation or pulmonary circulation.

a. Big Blood Circulation

   Blood leaves the left ventricle of the heart through this aorta branching into smaller arteries that deliver blood to various parts of the body. These arteries branch off and branch up even smaller until arterioles. These arteries have very muscular walls that narrow the tract and block the blood flow. Its function is to maintain arterial blood pressure and

by altering the size of the channel regulates blood flow in the capillaries (Evelyn C. Pearce, 2009).

Capillary walls are so thin that interchange of plasma and interstitial tissues can take place. Then these capillaries join and form larger vessels called venules, which then also unite into veins, to deliver blood back to the heart. All veins come together and unite again to form two venous stems, the inferior vena cava that collects blood from the body and the lower limbs, and the superior vena cava that collects blood from the head and upper limbs. Both of these blood vessels pour their contents into the right atrium of the heart (Evelyn C. Pearce, 2009).

b. Small Blood Circulation (Pulmonary Circulation)

The blood from the vein then enters the right ventricle that contracts and pumps it into the pulmonary artery. This artery is forked in two to deliver its blood to the right and left lungs. Blood is not difficult to enter the blood vessels that run through the lungs. Inside the lungs each artery divides into an arteriole and eventually becomes a pulmonary capillary that circles the alveoli within the lung tissue to pick up oxygen and release carbon dioxide (Evelyn C. Pearce 2009).

Then the pulmonary capillaries merge into the veins and the blood is returned to the heart by four pulmonary veins. And his blood was poured into the left atrium. This blood flows into the left ventricle. This ventricle contracts and blood is pumped into the aorta. So now begins again the circulation of blood (Evelyn C. Pearce, 2009).

Pulmonary Udema accompanies left-sided heart failure. The tissue fluid collects in the lungs and the lungs become functionally weak. Pulmonary Udema can also occur in overhydrated clients (getting too much fluid), the lungs become full of water and there is the possibility that it is "drowned" in its own lung udema (Evelyn C. Pearce, 2009).

Portal circulation, blood from the stomach, intestines, pancreas, and spleen are collected in the portal vein (gate vessels). Inside the heart the vein divides into the capillary system and then unites with the hepatic artery capillaries. This artery transports blood from the aorta of the heart and explores the whole organ. This double blood supply collected a unified vein system to form the hepatica vein. This vein delivers its blood to the inferior vena cava then to the heart (Evelyn C. Pearce, 2009).

Portal (obstruction) dams may occur when one or more branches of the portal vein are blocked, for example because there is severe injury to the liver or in some circumstances in liver inflammation. If this obstruction is severe, ascites complications may follow, ie excessive fluid retention in the peritoneal cavity. Coronary circulation (the circulation in the heart) provides blood for the heart itself (Evelyn C. Pearce, 2009).

2.1.2 Definition of Heart Failure

Heart failure is a complex clinical syndrome characterized by impaired myocardial performance and progressive maladaptive neurohormonal activation of the cardiovascular and renal systems leading to circulatory insufficiency and congestion. (Samara & Tang, 2012)

Heart failure, sometimes called congestive heart failure is the inability of the heart to pump enough blood to meet the oxygen and tissue nutrients. Heart failure is a clinical syndrome characterized by fluid overload and poor tissue perfusion. (Brunner & Suddarth, 2013)

HF is a complex clinical syndrome that results from any structural or functional impairment of ventricular filling and ejection of blood. (Yancy *et al*, 2013)

Heart (or cardiac or myocardial) failure is said to exist whenever ventricular function is depressed through myocardial damage, insufficient coronary flow, or any other condition that directly impairs the mechanical performance of the heart muscle. (Mohrman & Heller, 2014)

Heart failure is a chronic, progressive condition in which the heart muscle is unable to pump enough blood through to meet the body's needs for blood and oxygen. Basically, the heart can't keep up with its workload. (American Heart Association,2018)

According author heart failure is the inability and ineffectiveness of heart performance so that the disruption of blood and oxygen supply in the body.

2.1.3 Etiology of Heart Failure

According to Brunner & Suddarth (2010), The etiology of heart failure include:

2.1.3.1 Myocardial dysfunction is most often caused by coronary artery disease, cardiomyopathy, hypertension, or valvular disorders. Atherosclerosis of the coronary arteries is the primary cause of heart failure. Ischemia causes myocardial dysfunction because of resulting hypoxia and acidosis from the accumulation of lactic acid. Myocardial infarction causes focal heart muscle necrosis, the death of heart muscle cells, and a loss of contractility; the extent of the infarction correlates with the severity of heart failure. Revascularization of the coronary artery by a percutaneous coronary intervention or by coronary artery bypass surgery may correct the underlying cause so that hearth failure is resolved.

2.1.3.2 Cardiomyopathy is a disease of the myocardium. There are three types: dilated, hypertrophic. Dilated cardiomyopathy, the most common type of cardiomyopathy, causes diffuse cellular necrosis, leading to decreased contractility (systolic failure). Dilated cardiomyopathy can be idiopathic (unknown cause), or it can result from an inflammatory process, such as myocarditis, from pregnancy, or from a cytotoxic agent, such as alcohol or adriamycin. Hypertrophic cardiomyopathy and restrictive cardiomyopathy lead to decreased distensibility and ventricular filling (diastolic failure). Usually, hearth failure due to cardiomyopathy becomes chronic.

However, cardiomyopathy and hearth failure may resolve after the end of pregnancy or with the cessation of alcohol ingestion.

2.1.3.3 Systemic or pulmonary hypertension increases afterload (resistance to ejection), which increases the workload of the heart and leads to hypertrophy of myocardial muscle fibers; this can be considered a compensatory mechanism because it increases contractility. However, the hypertrophy may impair the heart's ability to fill properly during diastole.

2.1.3.4 Valve heart disease is also a cause of heart failure. The valves ensure that blood flows in one direction. With valve dysfunction, blood has increasing difficulty moving forward, increasing pressure within the heart and increasing cardiac workload, leading to diastolic hearth failure.

2.1.3.5 Several systemic conditions contribute to the development and severity of heart failure, including increased metabolic rate (eg, fever, thyrotoxicosis), iron overload (eg, from hemochromatosis), hypoxia, and anemia (serum hematocrit less than 25%). All of these conditions require an increase in cardiac output to satisfy the systemic oxygen demand. Hypoxia or anemia also may decrease the supply of oxygen to the myocardium.

Cardiac dysrhythmias may cause heart failure, or they may be a result of heart failure; either way, the altered electrical stimulation impairs the myocardial contraction and decreases the overall efficiency of myocardial function. Other factors, such as acidosis (respiratory or metabolic), electrolyte abnormalities, and antiarrhythmic medications, can worsen the myocardial dysfunction.

2.1.4 Pathophysiology of Heart Failure

According Muttaqin (2012), When the heart reservation (Cardiac protected) is normal to respond to inadequate stress to meet the metabolic needs of the body, the heart fails to perform its duties as a pump, and consequently heart failure occurs. If a normal cardiac reservation is called tiredness and failure, the physiological response. All of these responses show the body's ability to keep the perfusion of vital organs nonetheless. There are four primary rspons attitudes to heart failure:

2.1.4.1 Increased sympathetic adrenergic activity;

2.1.4.2 Increased initial load due to activation of neurohormin;

2.1.4.3 Ventricular hypertrophy

2.1.4.4 Overload Volumer

These four responses are an attempt to maintain cardiac output. These tactics may be sufficient to maintain normal conditions or impede normal on early breaks and breaks. However, abnormalities in work and decline can occur during the move. By continuing to fail, the compensation will be more effective.

a. Increased sympathetic adrenergic activity

   A decrease in stroke volume in heart failure will evoke a com-monational sympathetic response. Increased sympathetic adrenergic activity stimulates catecholamine and adrenergic heart and adrenal medulla nerves. Heart rate and contraction strength will increase to increase cardiac output. The peripheral artery also performs vasoconstriction to stabilize arterial pressure and redistribution of blood volume by reducing blood flow to low-metabolic organs such as skin and kidneys. It is intended that perfusion to the heart and brain can be maintained. Venoconstriction increases the venous return to the right side of

the heart, further increasing the contraction force in accordance with Starling's law (Mutaqqin, 2012).

In the state of heart failure, baroresepto in activity leads to increased sympathetic activity in the heart, kidneys, and peripheral blood vessels. Angiotensi II can increase the sympathetic activity. The excessive activity of the sympathetic nervous system leads to an increase in noradrenaline, plasma, which further leads to vasoconstriction, tachycardia, and salt and water retention. Excessive sympathetic activity can also cause necrosis of heart muscle cells. This change can be attributed to the observation that the storage of norepinephrine in the myocardium is reduced in chronic heart failure. (Mutaqqin, 2012).

b. Early Load Upgrade through the Renin-Angiotensin-Aldosteron System

Activation of the renin-angiotensi-aldosterone (RAA) system causes sodium and water retention by the kidneys, increased ventricular volume, and fiber strain. This increased initial load will increase the contractility of the myocardium in accordance with the straling law. The exact mechanism that leads to activation of the RAA system in heart failure remains unclear. The RAA system aims to maintain adequate fluid and electrolyte balance and maintain blood pressure (Mutaqqin, 2012).

Renin is an enzyme secreted by juxtaglomerular cells, which lie adjacent to the afferent renal arteriole and is associated with the macula densa of the distal tubule. Renun is an enzyme that converts angiotensin (mostly from the liver) to angiotensin I.

Angiotensin converting enzyme (ACE) bound to the plasma membrane of endothelial cells will break down two amino acids and angiotensin I to form angiotensin II. Angiotensin II has several important functions for maintaining circulating homeostasis, which stimulates arteriolar constriction in the kidneys and systemic circulation, and reabsorbs sodium in the proximal portion of the nephron (Mutaqqin, 2012).

Angiostensin II also stimulates the adrenal cortex to secrete aldosterone, which will stimulate reabsorption of sodium (in exchange with potassium) in the distal portion of the nephron, as well as in the colon, salivary glands, and sweat glands. Renin is secreted in conditions of decreased blood pressure, deficiency of sodium, and increased renal sympathetic activity (Mutaqqin, 2012).

Angiostensin I is mostly converted in the lungs to angiotensin II, a potent presor substance, by angiotensin converting enzyme (ACE). ACE can also break down bradykinin and work on a number of other peptides. Angiostensi II is ruptured rapidly by a non-specific enzyme called angiotensinase. Angiotensi II memes a major role in the RAA system because it attaches blood pressure in several ways such as vasoconstriction, salt retention and caries, and tachycardia.

The natriuretic atrial peptide (PNA) secreted by the heart then enters the circulation. The secretion is primarily affected by increased pressure on the atrial or ventricular wall, usually due to increased atrial or ventricular filling pressure. PNA causes dilatation of arteries constricted by other neurohormones and increases salt and water excretion (Mutaqqin, 2012)

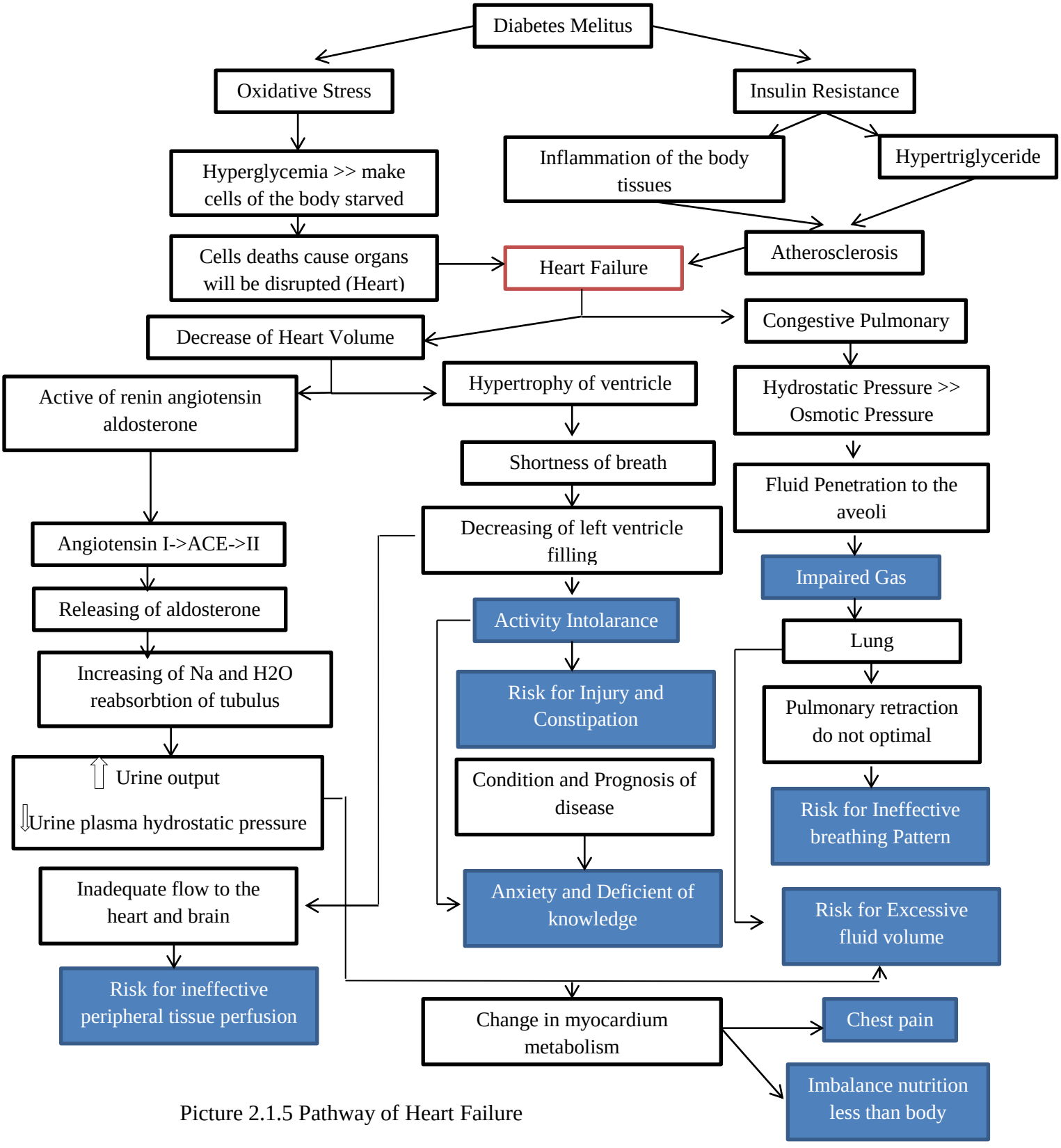
c. Ventricular Hypertrophy

The response to other heart failure is ventricular hypertrophy or increased ventricular wall thickness. Hypertrophy increases the number of sarcomomes in myocardial cells; depending on the hemodynamic load jenus that causes sarcoma heart failure can increase in parallel or serial. For example, a pressure load generated by aortic stenosis, will be accompanied by the addition of wall thickness without the addition of the size of space in it. Myocardial responses to volume loads such as aortic regurgitation, are characterized by dilatation and an increase in the number of serialized sarcomeres. Both patterns of hypertrophy are known as concentric hypertrophy and eccentric hypertrophy (Mutaqqin, 2012).

d. Overload Volume

Cardiac remodeling occurs in order to produce a large stroke volume, because each sarcoma has a limited peak shortening distance, the increase in stroke volume is achieved by increasing the number of series sarcomas, which will lead to an increase in ventricular volume. This widening requires greater wall tension to create the same intraventricular pressure requiring an increase in the number of parallel myofibrils. As a result, there is an increase in the thickness of the left ventricle wall. Thus, excess fluid volume causes widening space and eccentric hypertrophy (Mutaqqin, 2012).

2.1.5 Pathway of Heart Failure



Picture 2.1.5 Pathway of Heart Failure

2.1.6 Clinical Manifestation

According to Brunner & Suddart (2013), Signs and symptoms of heart failure can be associated with impaired ventricle. Left heart failure has a different manifestation of right heart failure. In chronic heart failure, the bias patient shows signs and symptoms of both types of heart failure

2.1.6.1 Left Heart Failure:

a. Pulmonary congestion: dyspnea, cough, lung krekels, low oxygen saturation levels, presence of additional heart sound S3 sounds or "ventricular gallop" can be detected by auscultation.
b. Dyspnea during activity (DOE), orthopnea, paroximal nocturnal dispnea (PND).
c. Dry cough and no phlegm at the beginning, over time can turn into cough with phlegm.
d. Sputum foaming, plentiful, and pink (bloody)
e. Krekels on both basal lung and can develop into krekels throughout the pulmonary area.
f. Inadequate tissue perfusion
g. Oliguria and nocturia
h. With the development of heart failure will arise symptoms such as: indigestion, dizziness, headache, confusion, anxiety, anxiety; skin pale or cold and moist.
i. Tachycardia, weakness, weak pulsation; fatigue.

2.1.6.2 Right heart failure :

a. Congestion on visceral and peripheral tissues
b. Lower extremity edema (dependent edema), hepatomegaly, ascites (accumulation of fluid in the

peritoneal cavity), loss of appetite, nausea, weakness, and weight gain due to fluid accumulation.

2.1.7 Classification of Heart Failure

Samara & Tang (2012) referred the classification of heart failure to The American College of Cardiology and the American Heart Association, and The New York Heart Association (NYHA).

2.1.7.1 The American College of Cardiology and the American Heart Association (ACC/AHA) guidelines currently classify heart failure on the basis of the evolution of the disease across a continuum:

a. Stage A: clients at high risk for developing heart failure without structural heart disease or symptomatic heart failure.
b. Stage B: clients with structural heart disease who have not yet developed symptoms of heart failure.
c. Stage C: clients with structural heart disease with prior or current symptoms of heart failure.
d. Stage D: clients with refractory end-stage heart failure who require specialized advanced treatment.

2.1.7.2 The New York Heart Association (NYHA) functional classification, although subjective and vague, remains the most commonly used standard by which the severity of functional impairment is graded.

a. Class I

   Clients have cardiac disease but without resulting limitations of physical activity. Ordinary physical activity

does not cause undue fatigue, palpitations, dyspnea, or anginal pain.

b. Class II

Clients have cardiac disease resulting in slight limitation of physical activity. They are comfortable at rest. Ordinary physical activity results in fatigue, palpitations, dyspnea, or anginal pain.

c. Class III

Clients have cardiac disease resulting in marked limitation of physical activity. They are comfortable at rest. Less than ordinary physical activity causes fatigue, palpitations, dyspnea, or anginal pain.

d. Class IV

Clients have cardiac disease resulting in an inability to carry on any physical activity without discomfort. Symptoms of cardiac insufficiency or of the anginal syndrome may be present even at rest. If any physical activity is undertaken, discomfort is increased.

2.1.8 Complications

The compensatory mechanisms initiated in heart failure can lead to complications in other body systems. Congestive hepatomegaly and splenomegaly caused by engorgement of the portal venous system results in increased abdominal pressure, ascites, and gastrointestinal problems. With prolonged right-sided heart failure, liver function may be impaired. Myocardial distention can precipitate dysrhythmias, further impairing cardiac output. Pleural effusions and other pulmonary problems may develop. Major complications of severe

heart failure are cardiogenic shock and acute pulmonary edema. (LeMone *et al*., 2011)

2.1.9 Prognosis of Heart Failure

Heart failure is associated with high rates of morbidity and mortality. In the Framingham Heart study, clients with heart failure had mortality rates four to eight times those of age-matched controls. A client with NYHA class IV heart failure has a 1-year survival between 30% and 50%—a mortality rate comparable to that of advanced malignancies. Several risk scores have been developed to characterize the risk of heart failure hospitalization and mortality. The Seattle Heart Failure Model is perhaps the most widely used of these and incorporates demographic, clinical, pharmacologic, and laboratory data to provide accurate 1-, 2-, and 3-year survival estimates. (Samara & Tang, 2012)

2.1.10 Diagnostic Examination of Heart Failure

2.1.10.1 Echocardiogram

Two-dimensional echocardiogram with doppler flow is the gold standard of diagnostic test. It can determine ventricular wall size and motion, and can differentiate systolic from diastolic dysfunction. Findings in systolic heart failure include dilated left ventricle. An echocardiogram can evaluate valvular status as a mechanical cause of heart failure (Schumacher & Chernecky, 2010).

2.1.10.2 ECG

12- lead ECG may show a prior MI, ventricular enlargement, or the presence of dysrhythmias (Schumacher & Chernecky, 2010).

2.1.10.3 Chest X-ray

Photo of posterior-anterior chest X-ray can show the presence of venous hypertension, pulmonary edema, or cardiomegaly. Evidence of increased pulmonary venous pressure is the diversion of blood flow to upper region and increase the size of blood vessels (Schumacher & Chernecky, 2010).

2.1.10.4 Radionuclide Ventriculography

Radionuclide Ventriculography gives an accurate measurement of global and regional function by measuring the amount of blood pumped out but is unable to assess valvular abnormalities or cardiac hypertrophy directly (Schumacher & Chernecky, 2010).

2.1.10.5 Brain Natriuretic Peptide measurement

BNP is a hormone that is released into the bloodstream by the failing ventricle in an attempt to help out by its natural vasodilatory and diuretic responses. The BNP blood level can help differentiate whether shortness of breath is due to hearth failure or a primary pulmonary problem. An elevated level is an indication of heart failure (Schumacher & Chernecky, 2010).

2.1.10.6 Treadmill Stress test and cardiac catheterization

A treadmill stress test or cardiac catheterization (or both) may be performed as treatable of heart failure (Schumacher & Chernecky, 2010).

2.1.11 Management of Heart Failure

According to Schumacher & Chernecky (2010), Heart failure Management are:

2.1.11.1 Pharmacologic therapy

a. Oxygen Therapy

Oxygen administration primarily aimed for patient with heart failure accompanied by pulmonary edema. Fulfillment of myocardial oxygen will reduce the oxygen needs and help fill the body's oxygen needs.

b. Diuretic

Decreasing venous return (*preload*) reduces the amount of volume returned to the left ventricle during diastole.

c. Vasodilator

IV nitroglycerin is a vasodilator that reduces circulating volume by decreasing preload and also increases coronary artery circulation by dilating the coronary arteries.

d. Morphine

Morphine sulfate reduce preload and afterload and is frequently used in the treatment of pulmonary edema.

e. Positive Inotropes

Inotropic therapy increases myocardial contractility. Digitalis is a positive inotrope that improve ventricular function.

f. ACE Inhibitor

ACE inhibitor help decrease preload by preventing sodium and water reabsorption

2.1.11.2 Non pharmacologic Therapy

a. Limit sodium intake. Excess sodium contributes to water retention. That make heart work harder.
b. Get plenty of rest, at least 8 hours daily.
c. Improve Physical conditioning with moderate exercise to prevent deconditioning (Kowalak, 2011).

2.1.11.3 Medical Treatment

a. Cardiac Resynchronization Therapy

Also called biventricular pacemaker, pacer leads are inserted into the left and right ventricles and the coronary sinus to resynchronize the contractility of the ventricle (Kowalak, 2011).

b. Heart Transplantations

Heart transplantation is a drastic step, heart failure must be end-stage with refractory cardiogenic shock or dependence on IV inotropic support to maintain adequate organ perfusion (Kowalak, 2011).

c. Left Ventricular Assist Device (LVAD) or Mechanical Heart pump

LVAD is mechanical device that is implanted into the abdomen and attached to the weakened heart to help it pump (Kowalak, 2011).

2.2 Basic Concept of Nursing Care Plan

2.2.1 Assessment

According Kasron (2016), often patients come with complaints of shortness of breath, chest pain, palpitations and dizziness or syncope, which needs to be done when the history is hammering the characteristics of the main symptoms, such as onset, progressiveness, and degrees

2.2.1.1 Main Complaint

Asking about the client's health history by asking for major complaints such as: Fatigue, fluid retention, irregular pulse, dyspnea, chest pain, headache, fatigue, tenderness in calfof leg and others (Manurung, 2016).

2.2.1.2 History of Previous Disease

Inquire about those who deal directly with the cardiovascular system. Ask the patient for a history of chest pain, shortness of breath, alcoholics, rheumatic fever anemia. sore throat caused by streptococcus, congenital heart disease, stroke, fainting of hypertension, thromboplebitis, pain loss, varicose veins and edema.(Manurung, 2016).

2.2.1.3 History of Treatment

Ask the patient about any treatment that a patient has had before such as taking aspirin. Treatment assessment should be written down the name of the drug and the patient understands its usefulness and side effects. The drugs that can affect the cardiovascular system such as Anticonvulsants, antide pressant, antipsychotics, cerebral stimulants, cholinergics estrogens, nonnarcotic analgesics and antipyretics, oral

contraceptives, sedatives and hypnotics, spasmolytics (Manurung, 2016).

2.2.1.4 History of Surgery or Other Treatment

Patients should also be asked specifically about the surgical treatments that have been undertaken, Hospitals care related to cardiovascular The results of diegnostic data ever performed during the perwatan should be more in the review. It should be noted that ECG and X-rays can be used as basic data (Manurung, 2016)

2.2.1.5 Healthy Lifestyle

According to Manurung (2016), The strong relationship between the components of the patient's lifestyle and the health of the vascular cardio is very influential, the patterns are yet another:

a. The pattern of healthy perception and healthy management. The nurse should ask for a major risk factor. Major cardiovascular risk factors: Increased serum lipid smoking, lack of activity and obesity. Patterns of life stress and DM should be asked as well. If the patient smokes asked about the type of cigarette, the number of cigarettes per day and the patient attempt to quit smoking. Alcohol use should also be noted (Genis, number, reaction change, and frequency).

   The habit of taking medicines includes recresional medicines. Ask about a history of allergies, the nurse asks how the drug and allergic reactions have been experienced. Confirmation of blood diseases associated with heredity and family history that tends to coronary artery disease,

vascular disease such as intermittent claudication, varicosities. Ask family health history on non cardiac conditions such as asthma, kidney disease and obesity should be assessed because it can result in the cardiovascular system.

b. Metabolic nutrition patterns. Being overweight and underweight can identify as a cardiovascular problem. Daily type of diarrhea is reviewed to determine the patient's lifestyle. The amount of salt and fat intake also needs to be assessed

c. Pattern Elimination : Skin color, temperature, wholeness / in- tegrity and turgor may be able to inform the circulatory problem. Arterisklerosis causing cold and cyanotic extremities can identify heart failure. Ureter Patients may be reported to have an increase in elimination Problems with constipation should be noted in the Mening or valsava manufer should be on the patient's hin with cardiovascular problems

d. Patterns of exercise-activity : The benefits of exercise on cardiovascular health can not be denied. Proper aerobic exercise becomes very tedious and Nurses must be careful in determining exercise, length of exercise, frequency and effects that are not on. want that will arise during the exercise. The length of time for exercise should be noted, other symptoms that identify from cardiovascular problems such as headache, chest pain, shortness of breath during lati should be recorded. Patients should also be asked for the ability to perform daily activities.

e. Pattern of rest-sleep : Cardiovascular problems often interfere with sleep, PND associates advanced heart failure. Many patients with heart failure require sleep with their heads elevated with pillows and the nurse notes the number of cushions required for comfort. Nocturia is often found in patients with carcodovascular problems, which interfere with normal sleep patterns.

f. Cognitive-perspectives pattern : The nurse asks the patient about the problem of cognitive perception. Pain associated with cardiovascular such as chest pain and intermittent claudica- tion should be asked or reported. Cardiovascular problems such as arrhythmias, hypertension and stroke may cause problems of vertigo, language and memory

g. The pattern of self-perceptions : If there are acute cardiovascular events, usually the patient's self-perception is often affected. Invasive diagnostics and palliative procedures often play an important role. Patients with chronic carcodascular problems are usually unable to identify the cause.

h. Pattern of role relationships : The sex, race and age of the patient are linked to carcovascular health Discuss with the patient the status of agriculture, the role in the household, the number of children and their age, the living environment and other assessments that are important in identifying strengths and suf fort sistern in life patient. The nurse should also assess the level of comfort or discomfort in performing the role function that has the potential to be a stress or conflict.

i. Sexuality and reproduction patterns :Patients with cardiovascular problems usually have an effect on sex patterns and satisfaction. Patients have a fear of sudden death during sexual intercourse and cause major changes in Fatique sex or shortness of breath can also limit sex activity. Impotence can be a sign of peripheral cardiovascular disease disorder, this is a side effect of several treatments used to treat cardiovascular problems such as beta-blockers, diuretics. Patient and partner counseling may be recommended.

j. Pattern of stress coping tolerance : Patients should be asked to identify stress or anxiety. The usual cop method should be studied. explosive, angry and hostile behaviors can be linked to the risk of heart disease. Information about suffort family systems, friends, psychologists or religious leaders can provide the best source for developing a treatment plan.

k. Patterns of values and beliefs : Individual values and beliefs are influenced by cultures and cultures that play an important role in the complicated level facing the patient when faced with cardiovascular disease.

2.2.1.6 Physical Examination of Heart

According to Kasron (2016), before starting to perform a physical examination of the heart, the examiner can already predict the prognosis of the heart position to the front thoracic wall. Most of the heart (23 parts) is located on the left side of the stermum, and only 1/3 lies to the right of the sternum. Some of the front (anterior) surfaces of the heart consist of the

right ventricle and the adjacent pulmonary artery directly to the front thoracic wall.

While the left ventricle is located on the left and right ventricular rear only occupies a small portion of the anterior heart surface, but this part is very important, because the front of the left ventricle is causing apical impulse, is a short systolic pulse, which is located between the fifth little medial costae from the left midclavicular line. or about 7-9 cm from the midsternal line

The right side of the heart comes from the right atrium, whereas the left atrium is positioned posteriorly, and can not be detected directly. The top of the heart consists of several large blood vessels of the aorta and the pulmonary artery. When going to a physical examination of the heart, the examiner can also imagine the flow of blood in the four heart cavities, when opening and closing the heart valves.

The patient should lie on his back, with the examiner standing on the right side of the bed. The head of the bed can be slightly elevated if the patient feels more comfortable with this position. Physical examination of heart includes :

a. General Condition

   The patient's general inspection often provides precious clues to the diagnosis of heart disease. Is the patient in acute distress? What is the patient's breathing? Does he breathe hard? Does wearing additional breathing muscles?.

b. Skin

Skin Inspection can reveal many changes related to heart disease. Is there cyanosis? If yes, is it central or peripheral? Peripheral cyanosis. Blue color, deoksihemoglobin 5gdL For example occurs in vasoconstriction, shock, heart failure and not seen in anemia. In addition, the cold can also cause peripheral cyanosis. Also when there is venous or arterial obstruction. Causes of central cyanosis also cause peripheral cyanosis Temperature may reflect heart disease. Anemia and thyrotoxicosis tend to make skin warmer, intermittent claudication associated with coldness of the inferior extremity when compared with superior extremity.

Nail Inspection Often, the hemorrhage spinter can be seen as a reddish brown line at the base of the nail. This bleeding runs from the proximal free edge and is classically associated with subacute bacterial endocarditis. But this discovery is not specific because it is found in many other conditions, even including local trauma to the nail. Finger with splinter hemorrhage in patients with endocarditis, trauma, infective.

Capillary nail pulsation (Quincke's sign), Skin blistering occurs at the nail root when emphasis is applied on the tip of the nail. occurs in aortic regurgitation and thyrotoxicosis. Body finger, clubber, body finger can be caused karen bronchial carcinoma, lung infection / fibrosis, cyanocic heart disease

c. Sight and Eyes

Eye Inspection The presence of a yellowish plaque on the eyelid, called xanthelasma, should awaken the kecurcostaean to hi-perlipoproteinema, although this lesion is less specific than xanthoma.The eyelids can be checked to see if there is xanthelasma in the form of a yellow plaque that can mean hyperlipidemia.

Retina checked for hypertensive damage Movement of the lens of the eye is common in patients with Marfan syndrome, an important cause of aortic regurgitation. Conjunctival haemorrhages are common in inffective endocarditis. Hypertelorism, or wide-range eyes, is associated with congenital heart disease, particularly pulmonary stemosis and supravalvular aortic stenosis. Reina examination can provide useful information about diabetes, hypertension and atherosclerosis.

d. Mouth

Inspection of the Mouth Ask the patient to open his mouth wide wide a. Is the arched palate inspecting the palate. high? The high-arched palate may be associated with congenital heart disorders such as mitral knee prolapse. Is there a petekiae on the palate? Subacute bacterial endocarditis is often accompanied by petechia in the palate.

e. Neck

Neck Injections Neck examination can show webbing. Webbing is present in people with Turner syndrome, who may have coarctation of the aorta, or Noonan syndrome.

Pulmonary stenosis is a cardiac abnormality that accompanies this condition.

f. Inspection and Palpation of Heart

According Manurung (2016), The overall inspection of the structure of the sternum, including the sternoclavicular junction, the manubrium and the top of the sternum is an early stage in examination. The obtained DNA, Angle of Louis, is digambarked where the manubrium and sternum body b unite at the center of the sternum. The angle of louis lies not in the second rib cage and can then be used to calculate the ICS and the location of the specific auscultation area.

Area of the heart (prekordial) in inspection and palpation simultaneously to determine the existence of abnormal pulsation or encouragement. Palpation is done systematically Following the anatomical structure of the heart starting from the aorta area, pulmonary area, tricuspidal area, apical area and epigastric area In the following way:

1) Help the patient regulate the supin position and the examiner nurse stands on the right side of the patient.
2) Determine the location of the angle of louis (Angle of Louis).
3) Move the fingers down toward each side of the corner so that it will feel the 2nd intercostal space. The aortic area is located in the 2nd right intercostal space and the pulmonal terle area is not in the 2nd left intercostal space.

4) Inspect and then palpate the aortic area and pulmonary area to determine the presence or absence of pulsation.
5) From the pulmonary area move your fingers down along the three left intercostal spaces. The ventricle or tricuspid area is located in the left intercostal space facing the sternum. Observe whether or not there is a pulsation. From the tricuspid area, move your arms laterally to the left midclavicular arena where an apical or PMI (Point of Maximal Impulse) will be found.
6) Inspection and palpation of the pulsation in the apical area. About 50% of adults will show apical pulsation. Size the heart can be known by observing the apical location In adults is normally located in the space between the left ribs 2-3 cm from the midclavicular line. While the child normally located in the space between the left fourth ribs. When the heart is enlarged then this pulsation will shift laterally to the midclavicular line.
7) To determine aortic pulsation, inspect and palpate the epigastric area on the basis of the sternum. At the time of palpation will be found in the presence of thrill (thrill) in patients with valve disorders. Patients with pulmonary stenosis will find a thrill between the 2nd left of the sternum.

   Patients with septal septal defect, thrill are found in the left 4th rib of the sternum. Patients with aortic stenosis, thrill are found in the second right of the sternum (base). And for patients with mitral insufficiency, thrill can be found in the apex. The vibration is more palpable if the sufferer bends forward, with breath held

expiratory time, unless the mitral stenosis is more palpable if the patient is lying to the left.

g. Percussion Of Heart

According Kasron (2016), Boundaries of the heart The position of the heart lies between the two lungs and is in the middle of the chest, resting approximately 5 cm above the xiphoideus processus. on the thoracic diaphragm and borders of the heart when interpreted to the chest wall :

1) On the right edge of the cranial (cranial dextra) is on the cranial edge of the cartilaginis pars cartilaginis costa dextra, 1 cm from the lateral edge of the sternum.
2) On the right edge of caudal (caudal dextra) is at the edge of the cranialis pars cartilaginis costa VI dextra, 1 cm from the lateral edge of the sternum.
3) On the left edge cranial (cranial sinistra) janturg bene on the edge of caudal pars cortilbginis costa II sinistra in sterile leteral sternum
4) On the left edge of the caudal (caudal sinistra) is a carefree intercostal 5, approximately 9 cm to the left of the midsternal or d linea medioclavicularis sinistra.

Normal left heart or peripheral border or left edge of intercostal space II / IV on the left parasternal line of relative heart revelation and absolute heartbeat needs to be sought to determine the magnitude of the heart.

In cardiomegaly, the heart's heart limit widens left and right. Left ventricular dilatation causes the apical cord to

shift to the lower-lateral. The liver of the heart is the heartbeat boundary of RSI IIl on the left parasternal line. Cardiomegaly can be found in athletes, heart failure, hi-perception, coronary heart disease, acute myocardial infarction pericarditis, cardiomyopathy, myocarditis, tricuspid regurgitation, aortic insufficiency, moderate ventricular defect, thyro- thotoxic sis, left atrial hypertrophy, .

In right ventricular hypertrophy, the heart's peripheral boundaries extend to the right lateral and / or to the upper left. In absolute concentrated pericarditis the heart widened to the right and to the left. In pulmonary emphysema, a narrowing of the heart may even disappear in severe pulmonary emphysema, so the heart's boundary in such circumstances is difficult to determine.

Percussion is carried out between the costae to the costae, the fourth and fifths of the left anterior axillary line to the right anterior axillary line. Usually there is a change from the percussion from the sonor to the frost approximately 6 cm in the lateral left of the sternum. This dim is due to the heart.

h. Auscultation of Heart

According of Manurung (2016), Auscultation of heart is useful for finding sounds caused by abnormalities in the heart structure with changes in blood flow generated during the heart cycle. Stethoscope is a tool used in mengauskultasi heart. It consists of two parts: Bell (for measuring low-pitched sounds such as heart sound 3 and

heart sound 4, mid systolic mice / tricuspid) and Diaphragm (for listening to high-pitched sounds such as heart sound 1 and Bj 2, opening snap, ejection sound, pericardial friction rub, systolic noisiness and early diastolic).

How / auscultation method:

1) Prepare a quiet room / environment and not bericik.
2) The patient lies on his back with a slightly elevated head.
3) Always check the right side of the patient.
4) Listen with the diaphragm of the statescope in the 2nd right intercostal space near the sternum (for the aortic region).
5) Listen with the diaphragm of the statescope in the left 2nd cascal space near the sternum (for the pulmonary region) arkan with the diaphragm in the intercostal space
6) listen with the stethoscope diaphragm in the intercostal space 3.4, and 5 near the left sternum (for the tricuspid area).
7) Listen with the diaphragm of the central wing of the apek (PMI) of the 4th intercostal space of the midclavicular line (for the mitral area).
8) Then the patient is turned to the left side and with a stethoscope bell on the stale will clarify the presence of S3 and mitral noise.
9) Patients are asked to sit upright forward and tilt breath after exhale. Listen with the stethoscope diaphragm in the 3rd and 4th left intercostal spaces near the sternum and this position will clarify the presence of aortic noise.

10) Record the heart sounds 1,2,3,4 as well as the loudness and strength / sound intensity

i. Examination of Jugular Venous Pulse

According to Manurung (2016), Internal jugular vein reflects right atrial pressure Should be observed maximum JVP height and range of pulsation.

1) Position the patient with an angle of 45 and the neck is supported so that the neck muscles are relaxed and the neck needs to be rotated to lateral.
2) Observe the border of the sternocleidomastoideus muscle with the clavicle and note the jugular venous flow, see pulsation
3) Try palpation pulsation, if it can be felt means posible derived from the carotid artery. Because jugular venous pulsation is unlikely to be palpated. Jugular venous pulsations are usually complex with the incoming tide (inward), then the jugular artery usually simple form of waves out (outward) are dominant. JVP usually decreases during inspiration
4) Estimate the height of the particle of a human-angle internal pulsation. External jugular vein is usually easier to spot, because it is the muscle on lateral sternokleidomas- toideus and more superficial. JVP improvements usually indicate:
   a) Heart failure
   b) Superior vena cava obstruction.
   c) Increased blood volume (pregnancy, acute nephritis, excess fluid therapy).

j. Examination of Dependent Edema

According of Kasron (2016), Edema Examination When high peripheral venous pressure, such as in congestive heart failure, pressure within the vein is propagated retrograde to smaller vessels. A fluid transudation occurs, resulting in edema in the de- pendent region. This increase in tissue fluid causes edema that is "concave when pressed.

Test of Edema To check for pitting edema, the finger is emphasized to the dependent region, such as the pretibial region, for 2-3 seconds. If there is pitting edema, the fingers will sink into the tissue, and when the finger is lifted, the former finger pressure will remain. Pitting edema is usually classified from 1+ to 4+, depending on how long the basin lasts. The most obvious is 4+. In bedridden patients, dependent areas are usually sakruin and not pretibia. In such patients the examiner should check for possible edema in the sacrum.

2.2.2 Nursing Diagnosis and Intervention

According Nanda Nic –Noc (2015-2017), the diagnose and nursing intervention in patients with heart failure include :

2.2.2.1 Impaired Gas Exchange related to ventilation-perfussion

Goals : After given the nursing care of 1xshift, expected damage to gas exchange is resolved, with the result criteria:

1) The client is able to issue a secret
2) RR of normal client 16-20 x / min
3) A regular breathing rhythm
4) The depth of inspiration is normal
5) Patient oxygenation is adequate

Intervention :

1) Position the patient to maximize the air vents
2) Perform physical therapy chest, as needed
3) Remove the secret by performing an effective cough or by performing suctioning
4) Record and monitor slowly, deep breathing and coughing
5) Provide aerosol treatment, as needed
6) Give oxygen therapy, as needed
7) Regulation of fluid intake to achieve fluid balance
8) Monitor respiratory status and oxygenation

2.2.2.2 Ineffective Breathing Pattern related to fatigue muscle tiredness

Goals : After the nursing action for 1x shift, the patient showed the effectiveness of the breath pattern, with the result criteria:

1) Frequency, rhythm, depth of breathing within normal limits
2) Not using the auxiliary muscles of breathing
3) Signs Vital signs in the normal range (blood pressure, pulse, respiration) (Bp 120-90 / 90-60 mmHg, pulse 80-100 x / min, RR: 18-24 x / min, temperature 36.5 - 37.5 C)

Intervention :

1) Position semi-fowler patient
2) Auscultation of breath sounds, note the result of decreased ventilation area or the absence of an adventitious sound
3) Respiratory Monitor and appropriate oxygen status
4) Maintain a patent airway
5) Collaboration in oxygen delivery therapy
6) Monitor oxygen flow
7) Monitor the speed, rhythm, depth and effort of the patient while breathing

8) Record chest movements, symmetrical or not, using respiratory auxiliary muscles
9) Monitor breath sounds like snoring
10) Monitor the breath pattern: bradypnea, tachypnea, hyperventilation, respiratory kussmaul, respiration cheyne-stokes etc.

2.2.2.3 Excess fluid volume related to Excess fluid intake

Goals : After being given nursing care for 1 x shift expected excess fluid volume can be reduced with the results criteria:

1) Oxygen saturation in expected range (90-100%)
2) RR within the expected limit (20-30x / mnt)
3) There is no dyspnea at rest
4) Fatigue decreases.

Intervention :

1) Monitor Laboratory examination results relating to fluid balance.
2) Monitor the results of laboratory tests related to fluid retention.
3) Monitor signs and symptoms of fluid retention and electrolyte imbalances
4) Monitor Vital signs, if needed.
5) Monitor patient response in electrolyte-related medication administration.
6) Provide medication as indicated by the patient.
7) Give medication in accordance with applicable standard procedure (method 6 True).
8) Monitor for possible allergic or contraindications related to therapy.
9) Help the patient to take the medicine.

10) Give diuretic medication as indicated.
11) Give antihypertensive medications as indicated

2.2.2.4 Activity Intolerance related to decreased cardiac output

Goals : After intervention during 1x shift expected client condition stable during activity with results criteria :

1) O2 saturation when activity is within normal limits (95-100%)
2) The pulse during activity is within normal limits (60-100x / min)
3) RR when activity is within normal limits (12-20x / mnt)
4) Systole blood pressure when activity is within normal limits (100-120mmHg)
5) Diastolic blood pressure when activity is within normal limits (60-80mmHg)
6) The ECG results are within normal limits
7) Does not look tired
8) Not looking lethargic
9) There is no decrease in appetite
10) No headaches
11) Sleep quality and rest within normal limits

Intervention :

1) Collaborate with other health teams to plan, monitoring client activation programs.
2) Help clients choose activities that fit the conditions.
3) Help clients to do physical activity / exercise regularly.
4) Monitor the emotional, physical and social and spiritual status of the client towards practice / activity.
5) Monitor results of client ECG examination at rest and activity (if possible with exercise tolerance test).

6) Collaboration of antihypertensive drug delivery, digitalis medicine, diuretic and vasodilator.
7) Monitor the effects of client treatment.
8) Monitor adequate nutrition intake as a source of energy.
9) Encourage clients and families to recognize signs and symptoms of fatigue during activity.
10) Encourage clients to limit heavy enough activities such as walking away, running, lifting heavy loads, etc.
11) Monitor the client's oxygen therapy response.

2.2.2.5 Risk for Injury

Goals : After care done nursing for 1 x shift expected risk of injury can be minimized with result criteria:

1) Patients recognize signs and symptoms that indicate scale injury risk factors 5
2) Patients can identify potential health risks of scale 5
3) Level of awareness of the patient either scale 5
4) The patient's cognitive status is well scale 5
5) The patient's cognitive orientation is good at scale 4
6) Patients know about the risk of injury scale 5
7) Patients know the strategy to address the risk of injury scale 5
8) Patients are aware of and can use safeguards according to scale 5 procedures
9) Patients can demonstrate self-protection from the risk of injury to scale 5.

Intervention :

1) Create a safe environment for patientsIdentify the patient's security needs, based on the physical level, cognitive function and behavioral history

2) Eliminate environmental hazard
3) Keep harmful objects out of the environment
4) Keeping up with a siderail if needed
5) Provide a low bed if needed
6) Place furniture in the room with the best arrangement for patient and family disability accommodation
7) Keep away from unnecessary, horrible and hot exposure
8) Lighting manipulation for therapeutic benefits
9) Cognitive identification and physical deficiency of the patient may increase the potential for injury
10) Identify the habits and risk factors that affect for injury.
11) Find patient and family injury history information.
12) Identify environmental characteristics that could increase the potential for injury.
13) Monitor gait, balance, and fatigue levels that can allow patients to injury

2.2.2.6 Constipation related to inadequate toileting and fiber intake is not enough

Goals : After nursing care done during 1x shift, patient expected to do defekasi with Criteria Result:

1) Maintain soft fecal form every 1-3 days
2) Free from discomfort and constipation
3) Identify indicators to prevent constipation
4) Feces are soft and shaped

Intervention :

1) Monitor signs and symptoms of constipation
2) Monior bowel sounds
3) Monitor feces: frequency, consistency and volume

4) Consult with your doctor about decreasing and increasing bowel sounds
5) Monitor signs and symptoms of intestinal rupture / peritonitis
6) Explain the etiology and rationalization of action to the patient
7) Identify the causal factors and contribution of constipation
8) Support fluid intake
9) Collaborate on laxatives
10) Monitor for signs and symptoms of impaction
11) Monitor bowel movement, including consistency of frequency, shape, volume, and color
12) Monitor bowel sounds
13) Consult with your doctor about decreased / increased frequency of bowel sounds
14) Monitor for signs and symptoms of intestinal rupture and / or peritonitis
15) Explain the etiology of the problem and the thought to action for the patient
16) Evaluation of drug profiles for gastrointestinal side effects
17) Advise patient / family to record the color, volume, frequency, and consistency of the stool

2.2.2.7 Ineffective peripheral tissue perfusion related to Lack of knowledge about the weight factor (eg smoking, monotonous lifestyle, trauma, obesity, salt intake, immobility)

Goals : After the act of nursing care for 1x shift is expected network perfusion is not disturbed by Criteria Results:

1) Demonstrate the circulation status indicated by:
   a) Systole and diastole pressure in the expected range
   b) No orthostatic hypertension

c) No signs of increased intracranial pressure (no more than 15 mmHg)

2) Demonstrate, cognitive abilities characterized by:
   a) Communicate clearly and in accordance with ability
   b) Show attention, concentration and orientation
   c) Processing information
   d) Making decisions correctly
3) Indicates the sensory function of complete cranial motion: the level of consciousness improves no movement of the involuntary movement

Intervention :

1) Monitor the presence of certain areas that are only sensitive to heat / cold / sharp / dull
2) Monitor the existence of paretese
3) Instruct the family to observe the skin if there is any contents or laceration
4) Use gloves for protection
5) Restrict movement to the head, neck and back
6) Monitor defecation capabilities
7) Collaboration of analgesic administration
8) Monitor the presence of thrombophlebitis
9) Discuss the cause of the sensation change

2.2.2.8 Acute pain related to agents of biological injury

Goals : After the nursing care provided by nursing care for 1x shift, the pain perceived the client is reduced by the criteria of the results:

1) Clients report reduced pain
2) The client can recognize the duration (onset) of pain
3) The client can describe the underlying factors

4) Clients can use non-pharmacological techniques
5) Clients use appropriate analgesic instructions
6) Clients report reduced pain
7) The client does not seem to complain and cry
8) The facial expression of the client shows no pain
9) The client is not nervous

Intervention :

1) Assess comprehensively for pain including location, characteristics, duration, frequency, quality, pain intensity and precipitation factor
2) Observe the reaction of nonverbal discomfort
3) Use therapeutic communication strategies to express the pain experience and client acceptance of the pain response
4) Determine the effect of the experience of pain on quality of life (appetite, sleep, activity, mood, social relations)
5) Determine factors that can aggravate pain. Evaluate with clients and other health teams about the size of pain control that has been done
6) Provide information about the pain including the cause of the pain, how long the pain will be gone, the anticipation of the discomfort of the procedure
7) Control environment that can affect client inconvenience response (room temperature, light and sound)
8) Eliminate precipitation factors that can improve the client's pain experience (fear, lack of knowledge)
9) Teach how to use non-pharmacological therapy (distraction, guide imagery, relaxation)
10) Collaboration of analgesic administration

2.2.2.9 Imbalance nutrition less than body requirements related to decreased intake, nausea, and anorexia

Goals : After nursing care done for 1x shift is expected fulfillment of patient needs fulfilled with result criteria:

1) Sufficient nutrition intake.
2) Adequate food and fluid intake
3) Decreased intensity of nausea vomiting
4) Decreased frequency of nausea and vomiting.
5) Patients experience weight gain

Intervention :

1) Assess nutritional status of patients
2) Keep your oral hygiene, always recommend oral hygiene.
3) Delegative nutrition that matches the patient's needs: diet of diabetes mellitus patients.
4) Give appropriate information to the patient about appropriate and appropriate nutritional needs.
5) Instruct patients to consume foods high in iron such as green vegetables
6) Assess the frequency of nausea, duration, severity, frequency factor, precipitation that causes nausea.
7) Instruct the patient to eat little by little but often.
8) Instruct patient to eat while warm
9) Delegative treatment of antiemetic therapy
10) Discuss with family and patient the importance of nutritional intake and the things that cause weight loss.
11) Weigh the patient's weight if possible regularly.

2.2.2.10 Anxiety related to shortness of breath due to inadequate oxygen supply

Goals : After being given nursing care for 1x shift expected clients do not experience anxiety, with the result criteria:

1) Anxiety on clients is reduced from scale 3 to scale 4

Intervention :

1) Listen to the cause of the client's anxiety intently
2) Observe verbal and nonverbal signs of client anxiety
3) Encourage families to stay with clients
4) Reduce or eliminate the stimuli that cause anxiety on the client

2.2.2.11 Deficient knowledge related to limitations of cognitive and unfamiliar with information sources

Goals : After giving nursing care for 1 × shift is expected to increase the knowledge of patients and families with the outcome criteria:

1) Patients and families express an understanding of the disease, condition, prognosis and treatment program
2) Patients and families are able to carry out properly described procedures
3) Patients and families are able to explain again what the other health / safety team described.

Intervention :

1) Assess the patient's level of knowledge about the specific disease process
2) Describe the pathophysiology of the disease and how it relates to anatomy and physiology, in a proper way

3) Describe the usual signs and symptoms of the disease, in the right way
4) Describe the disease process, in the right way
5) Identify possible causes, in appropriate way.
6) Provide information to the patient about the condition, in a timely manner
7) Avoid blank guarantees
8) Provide for family or SO information about patient progress in a proper way Discuss lifestyle changes that may be needed to prevent future complications and / or disease control processe.
9) Discuss the therapeutic or treatment options
10) Support patient to explore or get second opinion in the right way or indicated
11) Refer patients to groups or agencies in local communities, in a timely manner
12) Instruct patients about signs and symptoms to report to health care providers, in a timely manner