CHAPTER 2
THEORETICAL BACKGROUND

2.1. Medical Theoretical Review

2.1.1. Anatomy and Physiology of Heart

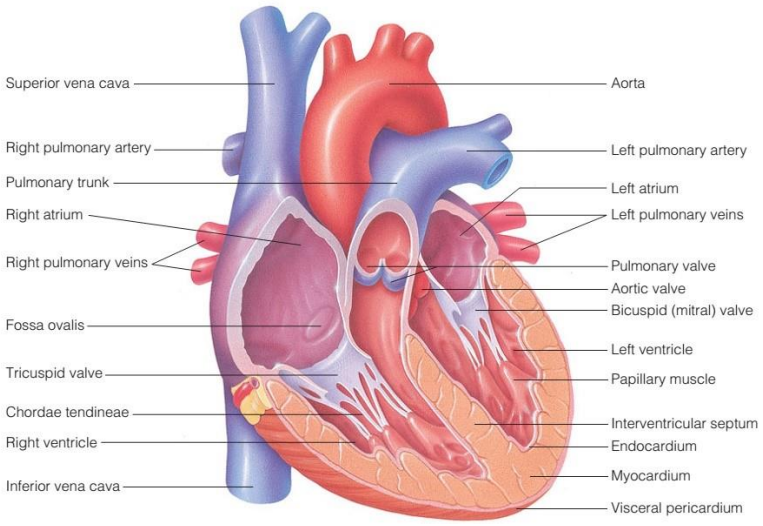


Figure 2.1.1 Heart Anatomy
(LeMone *et all*, 2011)

Human heart is four-chambered muscular organ, shaped and sized roughly like a person's close fist. It lies in the mediastinum, or middle region of thorax, just behind the body of the sternum between the points of attachment of the second through the sixth rib. Approximately two thirds of the heart's mass are to the left of the midline of the body (Patton & Thibodeau, 2013).

Heart has its own special covering, a loose fitting inextensible sac call pericardium. The pericardium consists of two parts: a fibrous portion and a serous portion. The fibrous pericardial sac with its smooth, well-lubricated lining provides protection against friction (Patton & Thibodeau, 2013).

Three distinct layers of tissue make up the heart wall in both the atrial and the ventricle: epicardium, myocardium, and endocardium. Epicardium is the outer layer of the heart, myocardium is middle layer of specially constructed and arranged cardiac muscle cell. Endocardium is the lining of the interior of the myocardial wall (Applegate, 2011).

The interior of the heart is divided into four chamber, the two upper chamber called atrium and the lower chamber are called ventricle. The two chambers of the heart are separated into left and right chamber by the interatrial septum. Atrial receive blood from vessel. The ventricle is the two-layer chambers of the heart. The ventricle is separated into left and right chamber by the interventricular septum. Because the ventricle receiving blood from the atria and pump blood out of the heart into arteries, the ventricle considered to be the primary pumping of the heart. Two of the valves are called atrioventricular valve and semilunar valve. The right atrioventricular valve is called tricuspid valve and left atrioventricular valve is called bicuspid valve. The semilunar valve at the entrance of the pulmonary trunk is called the pulmonary valve and the semilunar valve at entrance of the aorta is called aortic valve (Patton & Thibodeau, 2013).

The heart is often described as a double pump, because every side of the heart receives and secretes blood. Blood enters the right atrium and moves to the pulmonary bed at almost the exact same time that blood is entering the left atrium. The circulatory system has two parts: the systemic circulation (a high-pressure system), which supplies blood to all other body tissues, and the pulmonary circulation (a low-pressure system). The systemic circulation consists of the left side of the heart, the aorta and its branches, the capillaries that supply the brain and peripheral tissues, the systemic venous system, and the vena

cava. The pulmonary circulation consists of the right side of the heart, the pulmonary artery, the pulmonary capillaries, and the pulmonary vein. Pulmonary circulation begins with the right side of the heart. Deoxygenated blood from the venous system enters the right atrium through two large veins, the superior and inferior venae cave, and is transported to the lungs via the pulmonary artery and its branches. After oxygen and carbon dioxide are exchanged in the pulmonary capillaries, oxygen rich blood returns to the left atrium through several pulmonary veins. Blood is then pumped out of the left ventricle through the aorta and its major branches to supply all body tissues by the systemic circulation (LeMone et all, 2011).

2.1.2. Definition of Congestive Heart Failure

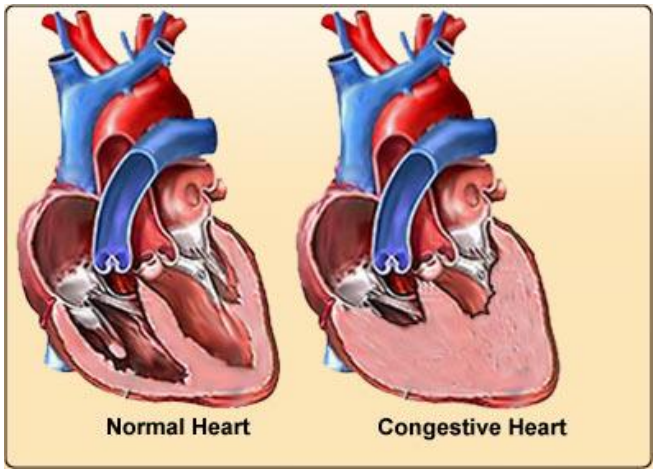


Figure 2.1.2 Normal and congestive heart

(Roland, 2017)

Heart failure is a condition when the heart is unable to maintain sufficient circulation for the body's needs, although the venous filling pressure is normal. but another definition says that heart failure is not a disease that is limited to an organ system, but rather a clinical syndrome due to cardiac abnormalities characterized by a form of hemodynamic, renal, neutral and hormonal response, as well as a pathophysiological

state in which cardiac abnormalities cause heart failure to pump blood to meet tissue requirements or can only fill it by increasing the filling pressure (Muttaqin, 2012).

Congestive heart failure is the inability of the heart to pump adequate blood to meet the tissue needs for oxygen and nutrients. Heart failure is also a pathophysiological condition of cardiac abnormalities resulting in the heart failing to pump blood to meet the needs of tissue metabolism and / or its ability only if accompanied by elevation of left ventricular filling pressure (Kasron, 2016). In line Udjianti (2010) also adds that. This results in stretching of the heart space (dilatation) to hold more blood to pump through the body or cause the heart muscle to become stiff and thickened. The heart is only able to pump blood for a short time and the weakened heart muscle wall is unable to pump strongly. As a result, the kidneys often respond by holding water and salt. This will cause fluid dams in some body organs such as hands, feet, lungs, or other organs so that the client's body becomes swollen (congestive).

The conclusion of the about definitions is that congestive heart failure is an abnormal state in which there are abnormalities of heart function that cause the heart is unable to pump out blood into the tissues to meet the needs of metabolism. the heart muscle becomes thick and stiff and makes the heart unable to pump blood throughout the body, one of the kidneys. causing body fluids cannot be processed by the kidneys and unstoppable in the organs of the client's body thus making the client become swollen in certain parts (congestive).

2.1.3. Etiology of Congestive Heart Failure

2.1.3.1. According Kasron (2016) and Oktavianus & Sari (2014) there are several etiologies or causes of congestive heart failure, there are:

a. Heart muscle abnormalities.

Heart failure is common in people with heart muscle abnormalities due to decreased cardiac contractility. the underlying conditions for the abnormality of heart function include coronary atherosclerosis, arterial hypertension and degenerative or inflammatory disease.

b. Coronary atherosclerosis

Coronary atherosclerosis results in myocardial dysfunction due to impaired blood flow to the heart muscle. hypoxia and acidosis (due to accumulation of lactic acid). Myocardial infarction or cardiac cell death usually precede the occurrence of heart failure. inflammation and degenerative myocardial disease, are associated with heart failure due to conditions that directly damage the heart fibers, causing contractility to decrease.

c. Systemic or pulmonary hypertension.

Increase the workload of the heart and in turn lead to cardiac muscle fibers.

d. Inflammation and degenerative myocardial disease.

Strongly associated with heart failure because this condition directly damages the heart fibers, causing contractility to decrease. Other heart disease. Heart failure can be as a result of true heart disease, which directly affects the heart. The mechanisms usually involved include impaired blood flow into the heart (semilunar valve stenosis), the inability of the heart to fill the blood (tamponade, pericardium, pericarditis, constrictive or AV stenosis), sudden increase afterload.

e. Systemic factors.

There are a number of factors that play a role in the development and severity of renal failure. Increased rates of metabolism, hypoxia and anemia require increased cardiac output to meet systemic oxygen demand. Hypoxia and anemia can also decrease oxygen supply to the heart. Respiratory or metabolic acidosis and electronic abnormality can decrease cardiac contractility.

2.1.3.2. According to (Wijaya & Putri, 2013), In general, heart failure can be caused by things that can be grouped into:

a. Myocardial dysfunction
   1) Myocardial ischaemia
   2) Myocardial infarction
   3) Myocarditis
   4) Cardiomyopathy

b. Excessive pressure load systolic (Systolic overload)
   1) Aortic stenosis
   2) Hypertension
   3) Aortic coartase

c. Overloaded diastolic pressure load (diastolic overload)
   1) Mitral valve insufficiency and tricuspidalis
   2) Excessive transfusion

d. Increased metabolic demand (demand overload)
   1) Anemia
   2) Thyrotoxicosis

e. Ventricular filling disorder
   1) Primary (failure of systolic distension)
      a) Testicular pericarditis
      b) Heart tamponade

2) Secondary

a) Mitral or tricuspidal stenosis

2.1.4. Pathophysiology of Congestive Heart Failure

2.1.4.1. According to Kasron (2016) the mechanisms underlying Congestive Heart Failure include cardiac contractility disorders that cause cardiac output to be lower than normal cardiac output. When cardiac output is reduced, the sympathetic nervous system will speed up the heart's frequency to maintain cardiac output. If this mechanism fails then the stroke volume must be adjusted. Stroke volume is the amount of blood pumped in each contraction, which is influenced by three factors that if one component is disturbed then the cardiac output will decrease. the three factors are:

a. Preload (initial load) is the amount of blood that fills the heart is directly proportional to the pressure caused by the length of the heart muscle strain.
b. Contractility. Changes in the strength of contractions relate to the length of the heart muscle tension.
c. Afterload (final load). The amount of ventricular pressure that must be generated to pump blood against the pressure required by arterial pressure (Oktavianus & Sari, 2014)

The condition that causes increased preload is aortic regurgitation, ventricular septal defect. The cause of increased afterload is in the state of aortic stenosis and systemic hypertension. Myocardial contractility may decrease in myocardial infarction and cardiac muscle abnormalities (Kasron, 2016).

2.1.4.2. Kasron (2016) said that the mechanisms underlying Congestive Heart Failure include decreased ability of cardiac contractility, so that the pumped blood in each contribution decreases causing a decrease in blood throughout the body. If the blood supply is less to the kidney will affect the mechanism of release of renin angiotensin and eventually formed angiotensin II resulted in the stimulation of aldosterone secretion and cause sodium and water retention. These changes increase the extravascular fluid resulting in an imbalance of fluid volume and subsequent pressure is edema. Peripheral edema results from accumulation of intravenous indoor fluid. This process arises problems such as nocturia where decreased vasoconstriction of the kidney at rest and also fluid redistribution and absorption at lying down. Continued cardiac failure may lead to ascites where ascites may produce symptoms of gastrointestinal symptoms such as nausea, vomiting, anorexia.

If the blood supply is not smooth in the lungs (blood does not enter the heart) causes fluid accumulation in the lungs that can decrease the exchange of O2 and CO2 between air and blood in the lungs. So that arterial oxygenation is reduced and there is an increase in CO2 that will form the acid in the body. This situation will provide a symptom of shortness of breath (dyspnea), orthopnea (dyspnea when lying down) occurs when the blood flow from the extremities increases venous return of venous to the heart and lungs (Kasron, 2016).

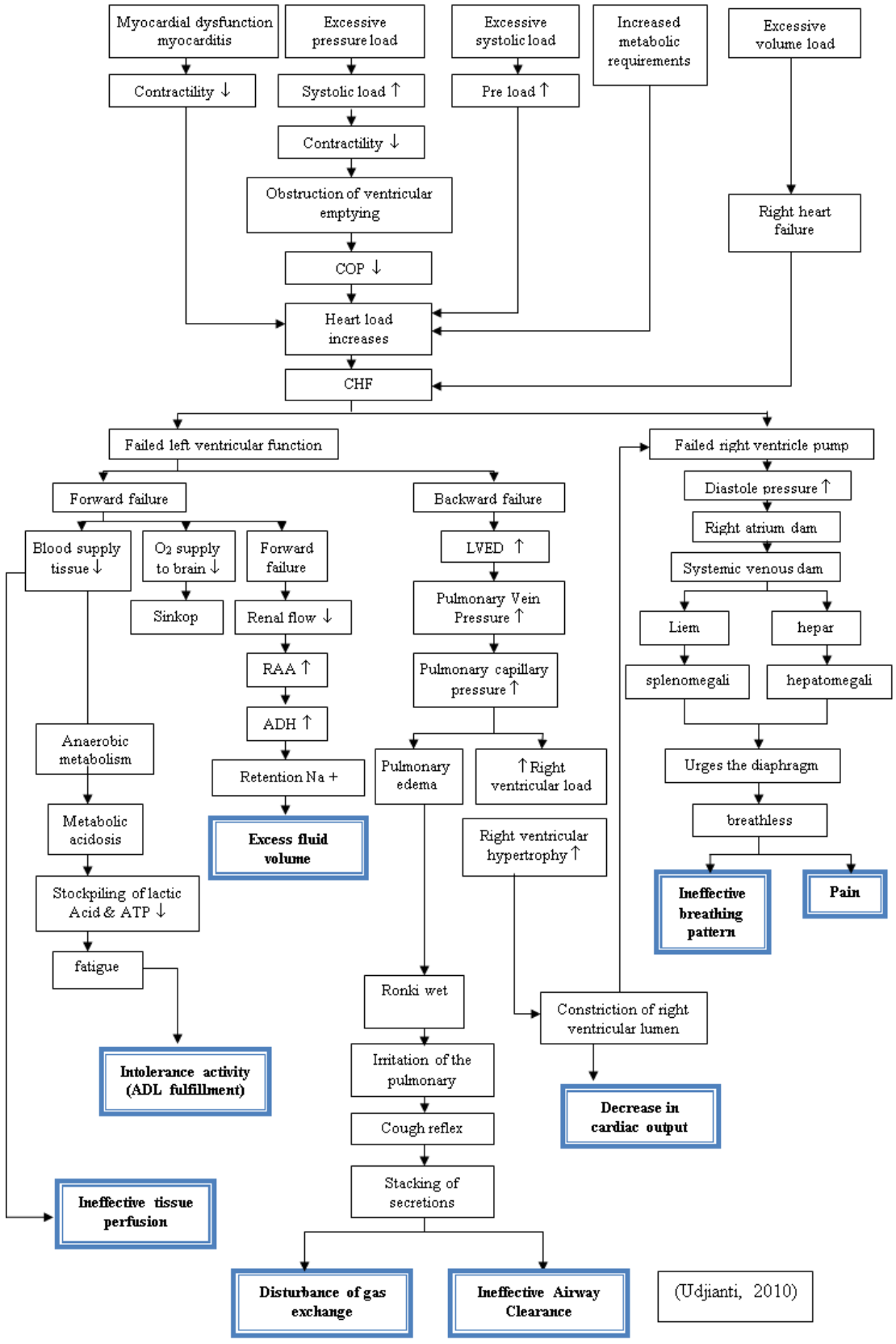
Kasron (2016) also adds that if there is enlargement of the veins in the liver resulted hepatomegaly and tenderness in the right quadrant the reduced blood supply in the muscle and skin

areas, causing the skin to become pale and cold and arising symptoms of fatigue, weakness, lethargy.

2.1.4.3. According to (Wijaya & Putri, 2013), there is a compensatory response such as:

a. Increases initial loads due to activity of renin angiotensin aldosterone (RAA), RAA activity causes Na and water retention by the kidneys, increasing the volume of the ventricle. This initial increase in load will increase the contractual contract of the myocardium.
b. Ventricular atrophy, the final compensatory response to heart failure is that myocardial hydrochropy increases the thickness of the wall.

2.1.5. Pathway of Congestive Heart Failure



2.1.6. Classification of Congestive Heart Failure

According to Kasron (2016) classifications of Congestive Heart Failure are:

2.1.6.1. Acute-chronic heart failure

a. Acute heart failure occurs suddenly, marked by decreased cardiac output and inadequate tissue perfusion. This can lead to pulmonary edema and collapse of blood vessels.
b. Chronic heart failure the occurrence is slowly characterized by ischemic heart disease, chronic lung disease. In chronic heart failure there is water retention and sodium in the ventricle causing hypervolemia, resulting in dilated ventricle and hypertrophy.

2.1.6.2. Right-left heart failure

a. Left heart failure occurs because the ventricle fails to pump blood adequately, causing pulmonary congestion, hypertension and abnormalities in the aortic / mitral valve.
b. Right heart failure is due to an increase in pulmonary pressure due to left heart failure that lasts long enough so that the dammed fluid will accumulate systemically in the feet, ascites, hepatomegaly, pleural effusions etc.

2.1.6.3. Systolic-diastolic heart failure

a. Systolic occurs due to decreased contractility of the left ventricle so that the left ventricle is unable to pump blood as a consequence of decreased cardiac output and hypertrophy ventricle.
b. Diastolic because of the inability of the ventricle in the blood fission resulting stroke volume of the cardiac output drops.

2.1.7. Sign and Symptoms of Congestive Heart Failuree

2.1.7.1. According to Oktavianus & Sari (2014). Common signs and symptoms are:

a. Orthopnea is breathless when lying down.
b. Dispone on effort (DOE) is breathless when do activities.
c. Proximal Nocturnal Dyspnea (PND) is breathless suddenly at night accompanied by cough.
d. Heart palpitations.
e. Quickly tired
f. Coughs

2.1.7.2. According to Schumacher & Chernecky (2010) and Oktavianus & Sari (2014). The dominant sign of heart failure is increased intravascular volume. Congestion tissue occurs due to increased arterial and venous pressure due to decreased cardiac output in heart failure.

The right and left ventricle can fail separately. Left ventricular failure most often precedes right ventricular failure. Failure of either ventricle may result in decreased tissue perfusion, but congestion manifestations may differ depending on which ventricular failure occurs.

a. Left Congestive Heart Failure

Pulmonary congestion is prominent in left ventricular failure because the left ventricle is unable to pump blood coming from the lungs. clinical manifestations that occur are:

1) Dyspnea: Occurs because of accumulation of fluid in the alveoli that disrupt gas exchange.
2) Paroxysmal nocturnal dyspnea
3) Orthopnea

4) Fatigue: occurs due to insufficient cardiac output that inhibits tissue from normal circulation and oxygen and decreases the discharge of cytotoxic sisal outcome. Also because of the increased energy used for breathing.
5) Dry cough worse at night: occurs due to increased pulmonary venous pressure (pulmonary edema).
6) Insomnia: occurs due to respiratory distress and cough.
7) Anxiety: occurs due to tissue oxygenation disorders, stress due to the pain of breathing and the knowledge that the heart is not functioning properly.
8) Cardiomegaly.
9) Tachypnea
10) Tachycardia
11) Third heart sound
12) Arrhythmia.
13) Cyanosis

b. Right Congestive Heart Failure

Congestion of peripheral and visceral tissue stands out. Because the right side of the heart is unable to empty the blood volume adequately so it can not accommodate all the blood that is normally returned from the venous circulation.

1) Peripheral pitting edema: Edema dependent/lower extremity.
2) Weight gain.
3) Ascites.
4) Jugular venous distention.
5) Liver engorgement or discomfort.
6) Hepatosplenomegaly and pain tenderness on upper right quadrant of abdomen occurs due enlargement vein in liver.

7) Anorexia and nausea: occurs because enlargement vein and vein static in abdomen cavity and abdominal bloating.
8) Nocturia: cardiac output improves so renal perfusion increases and diuresis develops.
9) Fatigue: occurs due to inadequate disposal of inadequate metabolic waste products.
10) Positive HJ reflux.
11) Elevated of jugular vein

2.1.7.3. New York Heart Association (NYHA) (2016) class of heart failure, devided into 4 fungtion disorders:

a. Class I: No limitation of physical activity. Ordinary physical activity does not cause fatigue, dyspnea, palpitation, or anginal pain or if the client can do strenuous activity without complaint.
b. Class II: Slight limitation of physical activity. No symptoms, at rest. Ordinary physical activity result in fatigue, dyspnea, palpitation, or anginal pain or If the client is unable to perform more severe activities than daily activities without complaints.
c. Class III: Marked limitation of physical activity but usually comfortable at rest. Ordinary physical activity causes fatigue, dyspnea, palpitations or anginal pain or If the client is unable to perform daily activities without complaint.
d. Class IV: Inability to carry on any physical activity without discomfort, symptoms of cardiac insufficiency or of angina may be present even at rest. If any physical activity is undertaken, discomfort is increased or If the client can not do any activity at all and must lay down

(Schumacher & Chernecky, 2010; Oktavianus & Sari, 2014).

2.1.8. Treatment of Congestive Heart Failure

2.1.8.1. Treatment according to class of New York Heart Association (NYHA) (2016) in Kasron (2016):

a. Class I: Non-Pharmacology are: Low-Salt diet, restricting fluids, reducing weight, avoid alcohol and smoke, physical activities, stress management.
b. Class II and III: Pharmacology are: diuretic, vasodilator, ace inhibitor, digitalis, dopamineroik, oksigen.
c. Class IV: diuretic combination, digitalis, ace inhibitor, life time.

2.1.8.2. Non-Pharmacology

a. Low-salt diet

   Sodium restriction to prevent, control, or eliminate edema.
b. Increases oxygenation by oxygen delivery and decreases oxygen consumption through rest and activity restriction
c. Restricting fluids

   Reduces heart load and avoids excess body fluid volume.
d. Reducing weight
e. Avoiding alcohol
f. Management stress

   Psychological responses can affect the increased work of the heart.
g. Reduces physical activity

   Excess physical activity leads to increased cardiac work that needs to be limited. In severe heart failure with activity restriction, but when stable clients are

recommended to increase activity regularly. Physical exercise can be 3-5 x/ week for 20-30 minutes or static bikes 5 x/ week for 20 minutes with a maximum 70-80% heart rate on mild or moderate heart failure.

h. Stopping aggravating drugs such as NSAIDs because the effects of prostaglandins on the kidneys lead to water and sodium retention.

1) Chronic Congestive Heart Failure
   a) Oxygenation
   b) Low-salt diet (<4 g / day)
   c) Activity limitation
   d) Avoiding NSAIDs Drugs
   e) Restriction of liquid (about 1200-1500 cc/ day)
   f) Regular exercise
2) Acute Congestive Heart Failure
   a) Oxygenation (mechanical ventilation)
   b) Limitations of fluids (<1.5 liters / day)

(Kasron, 2016; Oktavianus & Sari, 2014).

2.1.8.3. Pharmacology

Purpose: to reduce afterload and preload

a. First line drugs; diuretic

Aim: reduce afterload on systolic dysfunction and reduce pulmonary congestion in diastolic dysfunction.

The drugs are: thiazide diuretics for moderate CHF, loop diuretic, metolazone (combination of loop diuretic to increase fluid excretion), Potassium-Sparing diuretic.

b. Second Line drugs; ACE inhibitor

Aim; helps increase COP and decreases heart work. The remedy is:

1) Digoxin; increase contractility. It is not used for diastolic failure which requires ventricular development for relaxation
2) Hydralazine; decreases afterload on systolic dysfunction.
3) Isobar bide dinitrate; reduce preload and afterload for systolic dysfunction, avoid vasodilators in systolic dysfunction.
4) Calcium Channel Blocker; for diastolic failure, improving relaxation and filling and filling of the ventricles (not used in chronic CHF).
5) Beta Blocker; often contraindicated because it suppresses myocardial responses. Used in diastolic dysfunction to reduce HR, prevent myocardial ischemia, decrease BP, left ventricular hypertrophy (Kasron, 2016; Oktavianus & Sari, 2014).

2.1.9. Diagnostic Examination of Congestive Heart Failure

2.1.9.1. According to Kasron (2016) diagnostic examination of congestive Heart Failure are:

a. ECG.

Know the atrial or ventricular hypertrophy, infarction, axis storage, ischemia and pattern damage.

b. Blood laboratory test.

1) Enzyme liver: Increased in heart failure /congestion.
2) Electrolytes: Possibility of change due to fluid transfer, decreased kidney function.
3) Pulse oximetry: The oxygen situation is likely to be low.
4) BGA (blood gas analysis): Left ventricular failure characterized by mild respiratory alkalosis or hypoxemia with elevated PCO2.

5) Albumin: May decrease as a result of decreased protein input.

c. Radiologist.

1) Echocardiogram sonogram: May show enlarged chamber changes in the function of the valve structure decreased ventricular contractility.
2) Heart Scan: Actions of injecting fractions and estimating wall motion.
3) Chest x-ray: Indicates cardiac enlargement. Shadows reflect dilatation or chamber hypertrophy or changes in blood vessels or increased pulmonary pressure.

2.1.9.2. According to Oktavianus & Sari (2014):

a. Cardiac catheterization

Abnormal stresses indicate and help to differentiate left heart failure and kana, valve stenosis or insufficiency and assess the potential of coronary arteries.

b. Hematology and clinical chemistry: Hb, Ht, Leukocyte, Urea, Creatinine, SGOT, SGPT, Blood sugar, cholesterol and triglyceride.

Electrolytes (Na, K, Cl, Mg): may change due to fluid transfer, decreased kidney function, and diuretic diuretics.

c. Cardiac enzymes

Increased if cardiac tissue damage, eg myocardial infarction (creatinine phosphokinase / CPK, CPK isoenzymes, and lactate dehydrogenase / LDH, LDH isoenzymes).

2.1.10. Prognosis of Congestive Heart Failure

The prognosis of Congestive Heart Failure is dependent on the degree of myocardial dysfunction. For class I-III obtained mortality 1 and 5

years each of 25% and 52% while the 1-year mortality fourth grade is about 43% - 50% (Muttaqin, 2012).

2.1.11. Complications of Congestive Heart Failure

2.1.11.1. According to Wijaya & Putri (2013) and Kasron (2016) complications of Congestive heart failure are:

a. Cardiogenic shock.
b. Episodes of thromboembolism due to clot formation due to blood vessel stasis.
c. Effusion and pericardium tamponade.
d. Digitalis toxicity due to the use of digitalis drugs.
e. Pulmonal edema acut.

2.2. Theoretical Review of Nursing Care of Congestive Heart Failure

2.2.1. Nursing Care Plan

2.2.1.1. **Assessment**

Heart failure is a clinical syndrome characterized by a number of symptoms and signs, and is caused by various heart abnormalities, such as: cardiac rhythm disturbances, endocardial, pericardial, valvular, or myocardial disorders. Myocardial abnormalities may be systolic (associated with ventricular contraction and emptying), diastolic (associated with relaxation and ventricular filling), or a combination of both (Muttaqin, 2012).

Assessment of clients with heart failure is an important aspect of the treatment process. It is important to plan the next action. The nurse collects basic data on the client's current status information on the assessment of the cardiovascular system as a review priority. The patient's systematic review includes a careful history, especially with regard to symptoms. There is general physical weakness,

such as: chest pain, difficulty in shortness (dyspnea), palpitations, fainting (syncope), or cold sweat (diaphoresis). Each symptom should be evaluated time and duration as well as triggering and mitigating factors (Muttaqin, 2012).

a. History

In the history, the section studied is a major complaint, current disease history, and past medical history.

1) The main complaint

The most common complaints are the client's reasons for requesting health care, including: dyspnea, physical weakness, and systemic edema.

a) Dyspnea

Dyspnea or breathless complaints are secondary pulmonary congestion manifestations of left ventricular failure in contractility, this reducing stroke volume. With increasing LVDEP, there is also a rise in left atrial pressure (LAP), since the atria and ventricle are directly related during diastole. Increased LAP is passed backward into the vascular woven lungs, increasing capillary pressure, and pulmonary veins.

If the hydrostatic pressure of the lung capillary lung exceeds the vascular oncotic pressure, fluid transudation will occur into the interstitial. If the fluid transudation rate exceeds the rate of lymphatic drainage, there will be interstitial edema. A further increase in pressure may lead to fluid seeping into the alveoli and pulmonary edema occurs (Muttaqin, 2012).

b) Physical weakness

The main manifestation of decreased cardiac output is weakness and fatigue in conducting activities (Muttaqin, 2012).

c) Systemic edema

Pulmonary artery pressure may increase in response to a chronic increase in pulmonary venous pressure. Pulmonary hypertension increases resistance to right ventricular ejection. Mechanism of occurrence as it occurs in the left heart, also will occur in the right heart, where the end will occur systemic and systemic edema (Muttaqin, 2012).

2) Current Illness History

Assessment of the current illness history that supports the main complaint by conducting a series of questions about the main complaint chronology. Assessments obtained in the presence of vascular pulmonary congestion symptoms are dyspnea, orthopnea, proximal nocturnal dyspnea, cough, and acute pulmonary edema. In dyspnea studies (characterized by rapid, shallow breathing, and difficult sensations in obtaining adequate air and suppressing the client) does it interfere with other activities such as complaints about insomnia, anxiety, or weakness caused by dyspnea (Muttaqin, 2012).

3) Historical Illness

Assessment of historical illness is supported by assessing whether previous clients have suffered typical chest pain of myocardial infarction, hypertension, diabetes mellitus, and hyperlipidemia.

Ask about the usual medicines taken by clients in the past that are still relevant. These medications include diuretic, nitrate, beta-blocking drugs, and antihypertensive medications. Note the side effects that occurred in the past. Also have to ask for a drug allergy and ask what allergic reactions arise. Often clients confuse some allergies with drug side effects (Muttaqin, 2012).

4) Family History

The nurse asks about the disease that family has experienced, and if any family member dies, then the cause of death is also asked. Ischemic heart disease in the elderly at the young age is a major risk factor for ischemic heart disease in offspring (Muttaqin, 2012).

5) History of Work and Habit

The nurse asks about the workplace situation and the environment. Social Habits: Asking about habits in lifestyle, such as drinking, alcohol, or certain medications. Smoking habits: asking about smoking habits, how long, how many sticks per day, and types of cigarettes. In addition to the above questions, the biographical data is also a data to know, namely; name, age, sex, residence, tribe, and religion shared by the client. In asking a question to the client, should be noted the condition of the client. When client in critical condition, the question raised is not an open question but a question whose answer is yes and no. Or questions that can be waived by gestures, is nod or shake your head just, so it does not require a great energy (Muttaqin, 2012).

6) Psychosocial

Anxiety and anxiety are due to tissue oxygenation, stress due to breathing pain, and knowledge that the heart is not functioning properly. A further reduction in cardiac output may be accompanied by insomnia or confusion.

There is a change in the integrity of the ego found by the client denying, the fear of dying, the doom is imminent, angry at the unnecessary illness, worrying with family, work, and finances. Signs: refusing, denying, anxiety, lack of eye contact, anxiety, anger behavior, focus on yourself. Social interaction: stress due to family, work, economic cost difficulties, coping difficulties with existing stressors (Muttaqin, 2012).

b. Physical Examination

According to Muttaqin (2012) physical examination consists of general circumstances and B1-B6 assessment.

1) General Condition

On examination of the general state of the heart failure client is usually obtained some good awareness or compos mentis and will change according to the level of interference that involves perfusion of the central nervous system.

a) B1 (Breathing)

Assessments obtained in the presence of pulmonary vascular congestion are dyspnea, orthopnea, proximal nocturnal dyspnea, cough, and acute pulmonary edema. Crackles or smooth wet Ronchi are generally heard on the posterior floor of the lung. It is controlled as evidence of left ventricular failure. Before crackles is considered a pump failure, the client must be

instructed to cough in order to open the basilar alveoli that may be compressed from under the diaphragm.

b) B2 (Bleeding)

The following will be explained about what assessments are performed on cardiac and vascular examinations.

1) Inspection

Inspect scarring of heart surgery. See the impact of decreased cardiac output. In addition to the resulting symptoms and pulmonary vascular congestion, the failure of the left ventricle is also associated with non-specific symptoms associated with decreased cardiac output. Clients can complain of weakness, fatigue, apathy, lethargy, difficulty concentrating, memory deficit, and decreased exercise tolerance. These symptoms may arise at a chronic low cardiac output level and are a major complaint of the client. Unfortunately, these symptoms are not specific and are often considered depression, neurosis, or functional complaints. Therefore, potentially this is an important indicator of the storage of pump functions that are often unrecognized, and the lien is also given improper beliefs or given tranquilizers (a preparation that improves mood-mood). Remember, the presence of non-specific symptoms of low cardiac output requires careful evaluation of the heart as well as psychic examination that will provide information to determine appropriate management.

(1) Jugular venous distention: if the right ventricle is unable to compensate, space dilatation, increased volume and pressure in the right ventricular end diastolic, resistance to ventricular fill, and a further increase in right atrial pressure. This increased pressure reverses the upper course of the vena cava and can be seen with an increase in jugular venous pressure. One can evaluate the best of these by looking at the veins in the neck and looking at the height of the blood column. In the client lying in bed with the head of the bed elevated between 30 ° and 60 °, the normal person's blood column in external jugular veins will be just a few millimeters above the upper limit of the clavicle, when this is seen at all.

(2) Edema. Edema is often considered as a sign of heart failure that can be trusted. Of course, often when the right ventricle has failed. At least this is a reliable sign of centric dysfunction. Many people, especially the elderly who spend their time to sit on a chair with their legs hanging. As a result of this position of the body, there is a decrease in subcutaneous tissue turgor associated with old age, and possibly primary venous disease such as varicosities. Ankle edema may occur that represent this factor rather than the failure of the ventricle.

Edema associated with failure in the right ventricle, depending on its location, when the

client stands or wakes up, watches his ankle and elevates the foot if failure worsens. When the client is lying in bed, the part rubbing against the bed becomes the sacrum area. Edema must be considered in that place. Visible clinical manifestations include edema of the lower extremity (dependent edema), which is usually pitting edema, weight gain, hepatomegaly/ hepatic hemorrhage), distention of the neck veins, ascites (accumulation of fluid in the peritoneal cavity), anorexia and nausea, nocturia, and weakness.

Edema begins in the legs and heels (dependent edema and gradually increases to the legs eventually to external genitalia and lower body. Calculary edema is often rare in long-lying clients, because the sacral region becomes a dependent region Pitting edema is which will remain sunken even after a slight emphasis with the fingertip.

2) Palpable

Because increased heart frequency is the heart's initial response to stress, sinus tachycardia may be suspected and often found in client examinations with cardiac pump failure. Other rhythms associated with pump failure include: premature atrial contraction, proximal atrial tachycardia, and premature ventricular pulse.

(1) Changes of pulse. Examination of arterial pulses during heart failure indicates rapid and

weak pulses. Rapid heartbeat or tachycardia, reflecting response to sympathetic nerve stimulation. A significant decrease in stroke volume and peripheral vasoconstriction reduce pulse pressure (the difference between systolic and diastolic pressure), resulting in a weak pulse or thread pulse. systolic hypotension is found in more severe heart failure.

In addition, in the left heart failure that can arise pulsus alternans (a change in the strength of the arterial pulse). Pulsus alternans exhibit severe mechanical dysfunction with recurrent pulse to stroke variation in stroke.

3) Auscultation

Blood pressure usually decreases due to decreased stroke content.

Physical signs associated with left ventricular failure can be identified easily in parts that include: third and fourth heart sounds (S3, S4) as well as lung cracks, S4 or atrial gallops, following atrial contractions and sounds best with bell stetoscope that are attached appropriately to the cardiac apex. The left lateral position may be required to get the sound. This is heard before the first heart sound (S1) and is not always a sure sign of congestive failure but may decrease myocardial compliance (increased stiffness). This may be an early indication of premonitory failure. S4 sounds is a common sound heard in clients with acute myocardial infarction and may not have significant prognosis but may indicate a recent failure.

S3 or ventricular gallop is an important sign of left ventricular failure and in adults is almost never present in the presence of significant heart disease. Most doctors will agree that action against congestive failure is indicated by this sign. S3 sounds at the beginning of diastolic after a second heart sound (S2) and is associated with a passive period of passive ventricular filling. It can also be heard best with a stethoscope bell placed on apex, with the client in the left lateral position and at the end of expiration. Additional heart sounds due to valve abnormality usually get if the cause of heart failure due to valve abnormalities.

4) Percussion

Limit heart there is a shift that indicates the presence of cardiac hypertrophy (cardiomegaly).

c) B3 (Brain)

Awareness is usually compos mentis, in obtaining peripheral cyanosis when severe tissue perfusion disorders. The client's objective assessment: the face of grimacing, weeping, moaning, moaning, and wriggling.

d) B4 (Bladder)

Measurement of the volume of urine output is related to fluid intake, so nurses need to monitor the presence of oliguria because it is the first sign of cardiogenic shock. The presence of limb edema indicates severe fluid retention.

e) B5 (Bowel)

Clients are usually found nausea and vomiting, decreased appetite due to enlargement of the veins

and venous stasis in the abdominal cavity, and weight loss.

Hepatomegaly and tenderness in the right upper quadrant of the abdomen occur due to enlargement of the veins in the liver is a manifestation of heart failure. When this process develops, the pressure in the portal vessels increases, so the fluid is pushed out of the abdominal cavity, a condition called ascites. Collection of fluid in the abdominal cavity can cause pressure on the diaphragm and respiratory distress.

f) B6 (Bone)

Things that usually happen and found in B6 assessment is as follows:

(1) Cold Skin, front fissure in the left ventricle gives rise to signs of diminished perfunctory organ function. Since blood is transferred from non-vital organs in order to maintain perfusion to the heart and brain, the earliest manifestation of future failure is reduced perfusion of s organs such as skin and skeletal muscles. Pale and cold skin is implicated by peripheral vasoconstriction, a further decrease in cardiac output and increased reduced hemoglobin levels results in cyanosis. Vasoconstriction of the skin inhibits the body's ability to release heat. Therefore, mild fever and excessive sweating can be found.

(2) Easy fatigue occurs due to poor cardiac output, thus inhibiting tissue from normal circulation and oxygen as well as decreasing disposal of catabolism. Also occurs due to increased energy used for breathing and insomnia that occur due to

respiratory distress and cough. Less perfusion of the skeletal muscles causes weakness and fatigue. These symptoms can be exacerbated by fluid and electrolyte imbalances or anorexia. Personal hygiene fulfillment changes.

2.3 Nursing Diagnosis and Intervention

2.3.1 Decreased cardiac output related to decreased left ventricular contractility, changes in the frequency, rhythm, and electrical conduction.
Intervention:

2.3.1.1 Assess and report the sign of decreased cardiac output.
Rational: The incidence of mortality and morbidity in connection with MI over the first 24 hours.

2.3.1.2 Auscultate the heart sounds.
Rational: s1 and s2 may be weak due to the decrease pump operation. General gallop rhythm (S3 and S4).

2.3.1.3 Monitor for urine output, record output and density or concentration of urine.
Rational: Kidneys respond to lower cardiac output with fluid retention and sodium.

2.3.1.4 Assess changes in sensory, example: lethargy, anxiety, and depression.
Rational: indicate inadequate cerebral perfusion secondary to decreased cardiac output.

2.3.1.5 Rest the clients with optimal bed rest.
Rational: The rest will reduce the work of the heart, improves cardiac reserve power, and lowers blood pressure.

2.3.1.6 Give the rest of psychology with quiet neighborhood.
Rational: stress-related emotions produce vasoconstriction, increased blood pressure and frequency or heart work.

2.3.1.7 Avoid dynamic maneuvers such as crouching when doing bowel and clenched hands.

Rational: increase frequency or workload of the heart.

2.3.1.8 Give an additional oxygen by nasal cannula or mask as indicated.

Rational: Increasing oxygen to the myocardium needs to counteract the effects of hypoxia or ischemia.

2.3.1.9 Collaboration to give medications as indicated, for example diuretics, vasodilators, digoxin, captopril, morphine sulfate, an anti-coagulant.

Rational: Helping nursing process quickly and accurately.

2.3.1.10 Monitor ECG and X-ray

Rational: Sinus tachycardia segment depression and flat of the wave T may occur due to increased oxygen demand. Photos of the chest may show an enlarged heart and pulmonary congestion changes (Muttaqin, 2012).

2.3.2 Acute pain related to biological injury agent secondary with of decreased blood supply to the myocardium, increased production of lactic acid.

Intervention:

2.3.2.1 Assess the characteristics of the pain, location, intensity, and duration (PQRST).

Rational: Variation due to appearance and behavior occur as pain assessment findings.

2.3.2.2 Instruct the patient to report pain immediately.

Rational: Cardiogenic shock results in severe pain that affects the sudden death.

2.3.2.3 Set physiological position.

Rational: Increasing the intake of oxygen to tissue ischemia.

2.3.2.4 Teach relaxation techniques or distraction during pain.
Rational: Increase oxygen intake and decrease the perception of pain.

2.3.2.5 Give antianginal medicine (nitroglycerin).
Rational: As prevent of the angina pain with coronary vasodilation.

2.3.2.6 Give analgesics, morphine 2-5 mg via intravenous.
Rational: Reducing severe pain (Muttaqin, 2012).

2.3.3 Disturbance gas exchange related to fluid permeation, secondary pulmonary congestion, alveolar capillary membrane changes and interstitial fluid retention.
Intervention:

2.3.3.1 Assess respiratory rate and depth.
Rational: Useful in evaluating the degree of respiratory distress and chronicity of the disease process.

2.3.3.2 Monitor level of consciousness and mental status. Investigate changes.
Rational: Restlessness and anxiety are common manifestations of hypoxia.

2.3.3.3 Monitor vital signs.
Rational: Changes in vital signs and development of dysrhythmias reflect effects of hypoxia on cardiovascular system.

2.3.3.4 Instruct client in effective coughing and deep breathing.
Rational: Clears airways and facilitates oxygen delivery.

2.3.3.5 Elevate head of bed and assist client to assume position to ease work of breathing.
Rational: Oxygen delivery may be improved by upright position and breathing exercises to decrease airway collapse, dyspnea, and work of breathing.

2.3.3.6 Auscultate heart tones and breath sounds.

Rational: S4 is commonly heard in severely hypertensive clients because of the presence of atrial hypertrophy. Development of S3 indicates ventricular hypertrophy and impaired cardiac functioning.

2.3.3.7 Monitor client's oxygen saturation.

Rational: Decrease of saturation can be developing complications requiring further evaluation and treatment.

2.3.3.8 Review serial chest x-rays and ECG result.

Rational: Changes on x-ray and ECG reflect progression or resolution of Complications.

2.3.3.9 Collaborate to administer oxygen therapy by appropriate.

Rational: The purpose of oxygen therapy is to maintain PaO2 above 60 mm Hg, or greater than 90% O2 saturation. Oxygen is administered by the method that provides appropriate delivery within the client's tolerance.

2.3.3.10 Collaborate to administer medications, as indicated.

Rational: Diuretics are considered first-line medications for uncomplicated hypertension and may be used alone or in association with other drugs, such as beta blockers, to reduce BP in client switch relatively normal renal function (Muttaqin, 2012).

2.3.4 Intolerance activity related to imbalance between oxygen supply to the tissues secondary to the needs of the decrease in cardiac output.

Intervention:

2.3.4.1 Record the frequency of the heart, rhythm and changes in blood pressure during and after the activity.

Rational: The client's response to activity can identify a decrease in myocardial oxygen.

2.3.4.2 Increase rest, limit activity and provide leisure activities that are not heavy.

Rational: Decrease myocardial work / oxygen consumption.

2.3.4.3 Encourage clients to avoid abdominal pressure, e.g. straining during defecation.

Rational: By straining will increase bradycardia, decrease cardiac output and tachycardia and increase blood pressure.

2.3.4.4 Keep the client on the bed rest.

Rational: To decrease the burden of the heart.

2.3.4.5 Maintain the addition of O_2 as needed.

Rational: To increase tissue oxygenation.

2.3.4.6 Give the diet as needed (water and Na restriction).

Rational: Prevent fluid retention and edema due to decreased cardiac contractility (Muttaqin, 2012).

2.3.5 Excess fluid volume related to systemic fluid overload as a secondary effect of decreased cardiac output.

Intervention:

2.3.5.1 Assess for edema of the extremities.

Rational: Suspected excess fluid volume.

2.3.5.2 Assess blood pressure.

Rational: Increased blood pressure can be interpreted the heart's workload increased due to an increased amount of fluid.

2.3.5.3 Measure the intake and output.

Rational: Decreased cardiac output resulting in impaired renal perfusion, sodium retention / water, and a decrease in urine output.

2.3.5.4 Weigh the body weight.

Rational: show impaired fluid balance of a sudden change of weight.

2.3.5.5 Encourage clients to drink enough water accordance with the indications.

Rational: Meet the needs of body fluids.

2.3.5.6 Give a diet without salt.

Rational: sodium increases fluid retention and increase plasma volume impact on improving cardiac workload and will make the need for increased myocardium.

2.3.5.7 Collaboration to give diuretics, example furosemide, spironolactone, hydro lactone.

Rational: Helping nursing quickly and precisely (Muttaqin, 2012).

2.3.6 Ineffective breathing pattern related to lung development is not optimal excess fluid in the lungs secondary to acute pulmonary edema.

Intervention:

2.3.6.1 Auscultation of breath sounds (crackles).

Rational: An indication pulmonary edema secondary to cardiac decompensating.

2.3.6.2 Assess for edema.

Rational: Suspected congestive failure or fluid volume overload.

2.3.6.3 Measure the intake and output

Rational: Renal perfusion resulting in disorders, retention of sodium or water, and a decrease in urine output.

2.3.6.4 Give a diet without salt.

Rational: sodium increases fluid retention and increase plasma volume impact on improving cardiac workload and will make the need for increased myocardium.

2.3.6.5 Give diuretics, example furosemide, spironolactone, hydro lactone.

Rational: helping nursing quickly and precisely. (Muttaqin, 2012).

2.3.7 Ineffective peripheral tissue perfusion related to decreased cardiac output.

Intervention:

2.3.7.1 Investigate sudden changes or continued alterations in mentation, such as anxiety, confusion, lethargy, and stupor.

Rational: Cerebral perfusion is directly related to cardiac output and is influenced by electrolyte and acid-base variations, hypoxia, and systemic emboli.

2.3.7.2 Inspect for pallor, cyanosis, mottling, and cool or clammy skin. Note strength of peripheral pulses.

Rational: Systemic vasoconstriction resulting from diminished cardiac output may be evidenced by decreased skin perfusion and diminished pulses.

2.3.7.3 Monitor respirations, noting work of breathing.

Rational: Cardiac pump failure and ischemic pain may precipitate respiratory distress; however, sudden or continued dyspnea may indicate thromboembolic pulmonary complications.

2.3.7.4 Monitor intake, noting changes in urine output, as indicated.

Rational: Decreased intake or persistent nausea may result in reduced circulating volume, which negatively affects perfusion and organ function.

2.3.7.5 Assess gastrointestinal function, no anorexia, decreased or absent bowel sounds, nausea and vomiting, abdominal distention, and constipation.

Rational: Reduced blood flow to mesentery can produce gastrointestinal dysfunction, such as loss of peristalsis (Muttaqin, 2012).