Question answer

Health & Medicine, Nursing



1. Causes of Chronic congestive heart failure. Anything that weakens or damages the heart and stops it from pumping normally can cause chronic heart failure. In most cases, people who develop this condition have another medical problem that affects the heart or blood vessels (Figueroa & Peters, 2006). These medical problems include heart defects at birth, high blood pressure, heart attack, diseased heart muscle, leaking or narrowing heart valves, and abnormal heart rhythms (Figueroa & Peters, 2006). Diabetes, lung disease, anemia, underactive thyroid gland (hypothyroidism), alcohol abuse, and some medical treatments could also be causes of chronic congestive heart failure (Figueroa & Peters, 2006). These medical conditions and behaviors weaken the heart muscle, hence causing heart failure. One can be at the risk of having chronic congestive heart failure by smoking, being overweight or obese, being physically inactive, and having high blood cholesterol levels (Jeffrey & Ryan, 2007). All of these risk factors can be prevented or reduced to some certain degree. However, age is a risk factor for chronic congestive heart failure that cannot be prevented. When one approaches 75 years, the heart muscles become stiffer and less efficient: a natural consequence of aging (Jeffrey & Ryan, 2007). Causes of chronic heart failure such as high blood pressure and heart attacks become prominent from midlife onwards. Therefore, people age, they become more at risk of having chronic congestive heart failure (Jeffrey & Ryan, 2007).

2. Symptoms.

a. Fatigue.

This happens because the body requires oxygen and nutrients during a physical exercise. However, the failing heart cannot pump the required oxygen to the rest of the body (Trelogan, 2011).

b. Shortness of breath.

Shortness of breath happens because of fluids that go back into the lungs. This interferes with oxygen getting into the blood hence causing shortness of breath (Trelogan, 2011).

c. Difficulty breathing at night.

Lying down encourages fluid going back into the lungs and preventing breathing. It is said that sitting up straight in the middle of the night enables the victim to catch breathe (Trelogan, 2011).

d. Unintended or sudden loss of weight.

e. Asthma-like wheezing or a dry hacking cough that worsens with lying down, but improves with sitting up or standing.

Fluid that goes back into the lungs causes the victim to cough. This backing up of fluid is encouraged when lying down. When sitting up straight, less fluid backs up into the lung, improving the condition (Trelogan, 2011).

f. Accumulation of fluid in the feet, ankles, legs and abdomen.

Liver and kidney failure can also be associated with this symptom.

Accumulation of fluid in the feet happens when there has been prolonged

standing. This happens because of fluid retention (Trelogan, 2011).

g. Enlargement of liver.

h. Weight gain. – Muscle and appetite may have reduced, but this happens because salt and water are retained in the body. (Trelogan, 2011).

3. Tests.

1st test to order

Test

Result/ Abnormalities

Tranthoracic echocardiogram

Systolic heart failure: depressed and dilated left and/or right ventricle with low ejection fraction; diastolic heart failure: left ventricular ejection fraction (LVEF) normal but left ventricular hypertrophy (LVH) and abnormal diastolic filling patterns (Jeffrey & Ryan, 2007).

ECG

Evidence of underlying CAD, LVH, or atrial enlargement; may be conduction abnormalities and abnormal QRS duration (Jeffrey & Ryan, 2007).

CXR

Abnormal (Jeffrey & Ryan, 2007).

FBC

Laboratory testing may reveal important heart failure aetiologies, the presence of disorders or conditions that can lead to or exacerbate heart failure; laboratory testing could also reveal important modulators of therapy (Jeffrey & Ryan, 2007).

Serum electrolytes (including calcium and magnesium)

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Serum creatinine, blood urea nitrogen

Normal to elevated (Jeffrey & Ryan, 2007).

Blood glucose

Normal to elevated (Jeffrey & Ryan, 2007).

LFT

Normal to elevated (Jeffrey & Ryan, 2007).

TFTs (especially TSH)

Hypothyroidism: elevated TSH, decreased FT3, decreased FT4;

hyperthyroidism: decreased TSH, elevated FT3, elevated FT4 (Jeffrey & Ryan,

2007).

Blood lipids

Elevated in dyslipidaemia, decreased in end-stage heart failure, especially in the presence of cardiac cachexia (Jeffrey & Ryan, 2007).

B-type natriuretic peptide (BNP)/N-terminal pro-brain natriuretic peptide (NTpro-BNP) levels

pro-bive) levels

Elevated (Jeffrey & Ryan, 2007).

Test to consider

Test

Result/ Abnormalities.

Standard exercise stress testing (bicycle or treadmill)

Reduced exercise capacity in idiopathic dilated cardiomyopathy; reduced

exercise capacity and signs of impaired myocardial perfusion in ischaemic

cardiomyopathy (Jeffrey & Ryan, 2007).

Cardiopulmonary exercise testing (CPX) with VO2 max

Reduced VO2max (Jeffrey & Ryan, 2007).

6-minute walking test exercise

As an alternative to CPX it may provide an objective assessment of the

patients functional status (Jeffrey & Ryan, 2007).

Right heart catheterisation

Provides objective haemodynamic assessment of left ventricular filling pressure and direct measures of cardiac output and pulmonary and systemic resistance (Jeffrey & Ryan, 2007).

Endomyocardial biopsy

Provides objective haemodynamic assessment of left ventricular filling

pressure and direct measures of cardiac output and pulmonary and systemic resistance (Jeffrey & Ryan, 2007).

Serum HIV Elisa

Positive or negative (Jeffrey & Ryan, 2007).

Emerging tests

Test

Result/ Abnormalities

iron levels and fasting transferring saturation

Usually decreased (Jeffrey & Ryan, 2007).

4. a). The cardiac glycosides (Cardenolides) – the digitalis preparation This class of drugs used to treat congestive heart failure has three drug members: digitoxin, digoxin, deslanoside (medifocus. com).

Physiology

The Cardiac Glycosides is mainly used to speed up the force of cardiac contraction.

1. An increase in the concentration of intracellular sodium. An enzyme called Na+- K+ ATPase cleaves ATP to ADP and Pi (medifocus. com). According to the article, energy is released from the hydrolysis of ATP drives the Na+-K+ pump which normally propels Na+ out of the cell and K+ into the cell. However, if this propulsion is disabled by the inhibition of this enzyme, the

net effect is the malfunction of the pump and an increase of sodium inside the cell with a loss of intracellular potassium to the extracellular space (medifocus. com). The entry of Na+ is partly due to the inactive re-entry of sodium inside the cell while the efflux of K+ is passive to the outside of the cell (medifocus. com).

2. An increase in the concentration of intracellular calcium. The heart has a second pump called the Na+-Ca2+ pump (medifocus. com). This pump takes 1 intracellular Ca2+ ion out of the myocyte in exchange for 4 extracellular Na+ ions brought into the myocyte (medifocus. com). This pump is turned on by a diffusion gradient difference in extracellular to intracellular sodium when the extracellular sodium concentration is higher than the intracellular sodium concentration (medifocus. com). The [Na+] outside drops because the [Na+] inside rises, and the pump becomes unbalanced and stops pumping Ca2+ out of the cell (medifocus. com). When the Na+ - K+ pump is immobilized, there is a rise of [Na+] inside the cell as well as a rise in the [Ca2+] inside (medifocus. com). The [Ca2+] inside will rise because of passive diffusion back into the cell coupled with the fact that the cell is not pumping any Ca2+ ions out (medifocus. com).

The main purpose of cardiac glycoside drugs is to increase the levels of [Ca2+] inside the cardiac cell, which in turn increases the force and velocity of contraction (medifocus. com).

b). Angiotensin Converting Enzyme Inhibitors (ACE Inhibitors).

The drug members for this are Coptopril (Capoten), Enalapril (Vasotec), and Lisinopril (Prinivil).

Physiology

Question answer – Paper Example

Renin is freed into the blood from the kidneys when there is low blood pressure (medifocus. com). Renin modifies angiotensinogen in the blood to Angiotensin I which then, in the presence of Angiotensin converting enzyme, is changed into Angiotensin II (medifocus. com). According to the book, Angiotensin II is a potent vasoconstrictor. An improved peripheral opposition generates a lot of afterload on the left ventricle. Untreated and persistent hypertension eventually creates so much work for the left ventricle that it will fail (medifocus. com). Angiotensin-Converting Enzyme Inhibitors (ACE Inhibitors) prevent the conversion of Angiotensin I to Angiotensin II (Diane & Samantha, 2008). These medications do this by hindering the enzymatic activity of Converting Enzyme - the enzyme that converts Angiotensin I to Angiotensin II (Diane & Samantha, 2008). Once Converting Enzyme is hindered, the systemic blood pressure falls and with the lower blood pressure there is an improvement in cardiac function (medifocus. com). 5. When hospitalized, the patient should be continuously monitored by electrocardiography and the diagnosis of acute MI confirmed by serial ECGs (Ryan, et al 2010). The patient should be monitored closely for adverse electrical or mechanical events because reinfarction and death commonly occur within the first 24 hours (Ryan, et al 2010). Physical activities of the patient should be restricted for at least 12 hours, and pain and/or anxiety should be reduced with appropriate painkillers.

Patients who endure a large anterior MI or who have a LV mural thrombus seen on echocardiography are at high risk of having an embolic stroke (Ryan, et al 2010). This risk can be reduced by administering intravenous heparin early. For the patient with a small anterior MI or LV mural thrombus

Question answer – Paper Example

who did not receive reperfusion therapy, heparin beyond that of aspirin, ßadrenoceptor blocking agents, nitrates, and Angiotensin converting enzyme (ACE) inhibitors should be administered (Ryan, et al 2010). For the patient given thrombolytic therapy, the recommendation for subsequent heparin administration are based more on current practice than on evidence and depend on the specific thrombolytic agent (Ryan, et al 2010). There is partial evidence that heparin beneficial in the patient who receives a nonspecific fibrinolytic agent such as streptokinase, anisoylated plasminogen streptokinase activator complex (APSAC), or urokinase (Ryan, et al 2010). After administering TPA, intravenous heparin increases the likelihood of patency in the infarct-related artery but this may not necessarily lead to improved clinical outcome (Ryan, et al 2010). After performing primary PTCA, high-dose intravenous heparin is recommended (Ryan, et al 2010). Aspirin, 160 to 325 mg daily, initially given in the ED, should be continued indefinitely (Ryan, et al 2010).

It is important to give the patient intravenous nitroglycerin for 24 to 48 hours after hospitalization despite the absence of definitive outcome data (Ryan, et al 2010). There is concern about oral nitrate preparations in the patient (Ryan, et al 2010). This is because of inability to titrate the dose to effect in an acutely evolving hemodynamic situation (Ryan, et al 2010). However, intravenous mixture of nitroglycerin can be titrated successfully with frequent measurement of heart rate and cuff blood pressure (Ryan, et al 2010). Nitroglycerin should not be used as an alternative for narcotic painkillers that are often required in the patient (Ryan, et al 2010). The patient should also receive early intravenous ß-adrenergic blocker

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therapy, followed by oral therapy (Ryan, et al 2010). According to Ryan, calcium channel blockers do not reduce mortality in patients, but in certain persons with cardiovascular disease they are harmful. In a patient without ST-segment elevation or LBBB in whom pulmonary congestion is missing, diltiazem reduces the incidence of recurrent ischemic events (Ryan, et al 2010). However, its benefit beyond that of ß-adrenoceptor blockers and aspirin is unclear (Ryan, et al 2010).

An ACE inhibitor should be given within hours of hospitalization, provided that the patient does not have hypotension or a contraindication (Ryan, et al 2010). Consequently, the ACE inhibitor should be continued indefinitely (Ryan, et al 2010). ACE inhibitors can be stopped in patients who have no complications and no signs of symptomatic or asymptomatic LV dysfunction by 6 weeks (Ryan, et al 2010). When the patient is admitted at the hospital, a profile of the lipid and serum electrolyte concentration should be measured in all patients (Ryan, et al 2010).

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