Marwood
NURSING AND REHAB

A SUBSIDIARY OF
McLaren
PORT HURON

BEST PRACTICE: DISEASE MANAGEMENT

HEART FAILURE

For Nurses

Best practice disease management package was designed to support the nursing staff at Marwood Nursing and Rehab and reduce avoidable hospitalizations.

The Best Practice package includes education on disease management to enhance a nurse's knowledge of strategies and interventions for the management of Congestive Heart Failure.

OBJECTIVES

After reviewing the Best Practice package, nurses should have a renewed understanding of the many components necessary to manage patients with heart failure.

CONTENTS

Cardiac Assessment ..... 01 - 05

Pathophysiology of Heart Failure ..... 06 - 11

Understanding Ejection Fraction ..... 12 - 13

Cardiac Medications ..... 14 - 19

Labs and Diagnostic Tests ..... 20 - 23

E-Interact Pathway for Change-in-Condition ..... ???

Nursing Interventions and Rationales ..... 24 - 27

Notifying the Physician / Notes ..... 28 - 29

Key Components of the Cardiac Assessment

GENERAL APPEARANCE

Start a physical examination of a resident's cardiac status by noting the resident's general appearance:

- Level of consciousness: awake, alert, lethargic, stuporous, or comatose
- General build: thin, emaciated, obese, average
- Breathing: short of breath, unlabored
- Evidence of acute distress: acute pain, labored breathing

VITAL SIGNS

Start a physical examination of a resident's cardiac status by noting the resident's general appearance…

- Obtain temperature and note route
- Heart rate and rhythm
  - Pulse rate: time for 1 full minute
  - Compare apical and radial pulses
  - Note rhythm: regular, regularly irregular, or irregularly irregular

- Blood pressure
  - Take pressure in both arms, if possible
  - Determine pulse pressure by subtracting the diastolic pressure from the systolic pressure (normal pulse pressure is 30-40 mmHg)
- Assess for orthostatic hypotension (occurs when BP drops 15-20 mmHg or more)

HEAD, NECK, AND SKIN

- Facial expressions and color – grimacing, flushing, cyanotic lips, jaundiced
- Examine neck for jugular vein distention
- Examine skin for temperature (normal is warm and dry), diaphoresis, cyanosis, pallor, or jaundice

EXTREMITIES

Acute and chronic heart disease may present with symptoms associated with the extremities. Assess for the following:

- Nailbeds
  - Color: pale, cyanotic
  - Clubbing: swollen nail base and loss of normal angle
  - Capillary refill: estimates the rate of peripheral blood flow (normal return is within 2 seconds)
- Edema: describe in terms of depth of pitting that occurs with slight pressure
  - 1+ or mild: 0 to ¼ inch
  - 2+ or moderate: ½ inch
  - 3-4+ or severe: ¾ to 1 inch
- Pulses: apical, radial, dorsalis pedis most commonly assessed
  - Normal rate: 60-100
  - Rhythm: regular vs. irregular
  - Quality: absent, weak/thready, diminished, normal, bounding

CHEST PAIN

If chest pain is present, assess further:

- Characteristics of the pain
  - Location: substernal; epigastric; jaw, neck, arms, hands, shoulders
  - Description: sharp, dull, stabbing, aching, pressure, squeezing, burning, heavy
  - Intensity: using pain scale (0-10)
  - Duration: constant, intermittent
  - Alleviating factors: what makes the pain better
- Associated factors
  - Activity: at time of occurrence
  - Sleep/Rest: are they getting enough
  - Cough: frequency, severity, sputum
  - Labored or difficulty breathing
- History: has it occurred before, or is this new
- Troublesome side effects such as dizziness, constipation, GI symptoms, or fatigue. Bleeding.

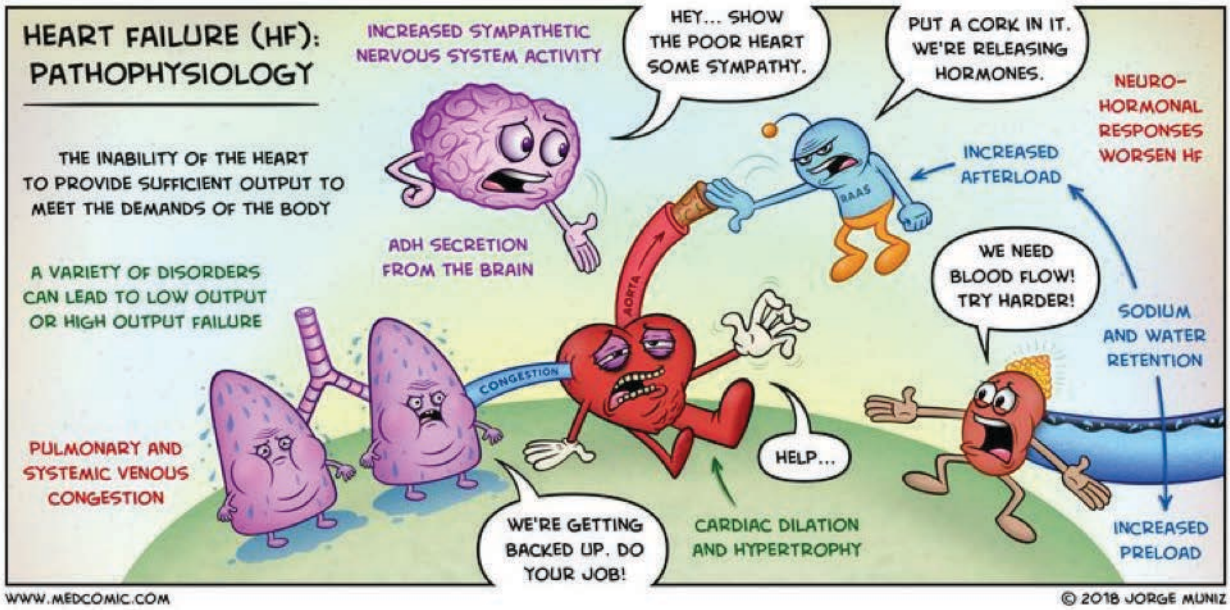
Nursing Interventions	Rationale
Nursing Assessment	
Auscultate apical pulse, assess heart rate, rhythm. Document dysrhythmia if telemetry is available.	Tachycardia is usually present (even at rest) to compensate for decreased ventricular contractility. Premature atrial contractions (PACs), paroxysmal atrial tachycardia (PAT), PVCs, multifocal atrial tachycardia (MAT), and atrial fibrillation (AF) are common dysrhythmias associated with HF, although others may also occur.
Note heart sounds.	S_1 and S_2 may be weak because of diminished pumping action. Gallop rhythms are common ($S_3$and S_4), produced as blood flows into noncompliant chambers. Murmurs may reflect valvular incompetence.
Palpate peripheral pulses.	Decreased cardiac output may be reflected in diminished radial, popliteal, dorsalis pedis, and post tibial pulses. Pulses may be fleeting or irregular to palpation, and pulsus alternans (strong beat alternating with weak beat) may be present.
Monitor BP.	In early, moderate, or chronic HF, BP may be elevated because of increased SVR. In advanced HF, the body may no longer be able to compensate, and profound hypotension may occur.
Inspect skin for pallor, cyanosis.	Pallor is indicative of diminished peripheral perfusion secondary to inadequate cardiac output, vasoconstriction, and anemia. Cyanosis may develop in refractory HF. Dependent areas are often blue or mottled as venous congestion increases.

Nursing Interventions	Rationale
Monitor urine output, noting decreasing output and concentrated urine.	Kidneys respond to reduced cardiac output by retaining water and sodium. Urine output is usually decreased during the day because of fluid shifts into tissues but may be increased at night because fluid returns to circulation when patient is recumbent.
Note changes in sensorium: lethargy, confusion, disorientation, anxiety, and depression.	May indicate inadequate cerebral perfusion secondary to decreased cardiac output.
Assess for abnormal heart and lung sounds.	Allows detection of left-sided heart failure that may occur with chronic renal failure patients due to fluid volume excess as the diseased kidneys are unable to excrete water.
Monitor blood pressure and pulse.	Patients with renal failure are most often hypertensive, which is attributable to excess fluid and the initiation of the renin-angiotensin mechanism.
Assess mental status and level of consciousness.	The accumulation of waste products in the bloodstream impairs oxygen transport and intake by cerebral tissues, which may manifest itself as confusion, lethargy, and altered consciousness.
Assess patient's skin temperature and peripheral pulses.	Decreased perfusion and oxygenation of tissues secondary to anemia and pump ineffectiveness may lead to decreased in temperature and peripheral pulses that are diminished and difficult to palpate.
Monitor results of laboratory and diagnostic tests.	Results of the test provide clues to the status of the disease and response to treatments.
Monitor oxygen saturation and ABGs.	Provides information regarding the heart's ability to perfuse distal tissues with oxygenated blood

I. PATHOPHYSIOLOGY OF IMPAIRED CARDIAC OUTPUT

How does the body compensate for impaired cardiac output?

- When cardiac output is compromised it results in specific responses as the body intrinsically attempts to maintain perfusion; blood is shunted to the vital organs in our body to maintain life.
- Sympathetic nervous system activation which triggers an increase in heart rate for an immediate bump in cardiac output.
- The body shunts oxygen rich blood to vital organs.
  - o Blood first shunted from the skin and gut causing a pale looking color to the patient's skin and the skin may feel cool. The patient will also have decreased gut motility and decreased bowel sounds.
  - o Next, blood is shunted from the kidneys and lungs. You will see decreased urine production and decreased respiratory effort. The patient may even go into respiratory failure
  - o Lastly the blood is shunted from the brain and heart.
- The Renin-Angiotensin-Aldosterone System (RAAS) is activated when cardiac output is impaired.



II. THE RENIN-ANGIOTENSIN-ALDOSTERONE SYSTEM (RAAS)

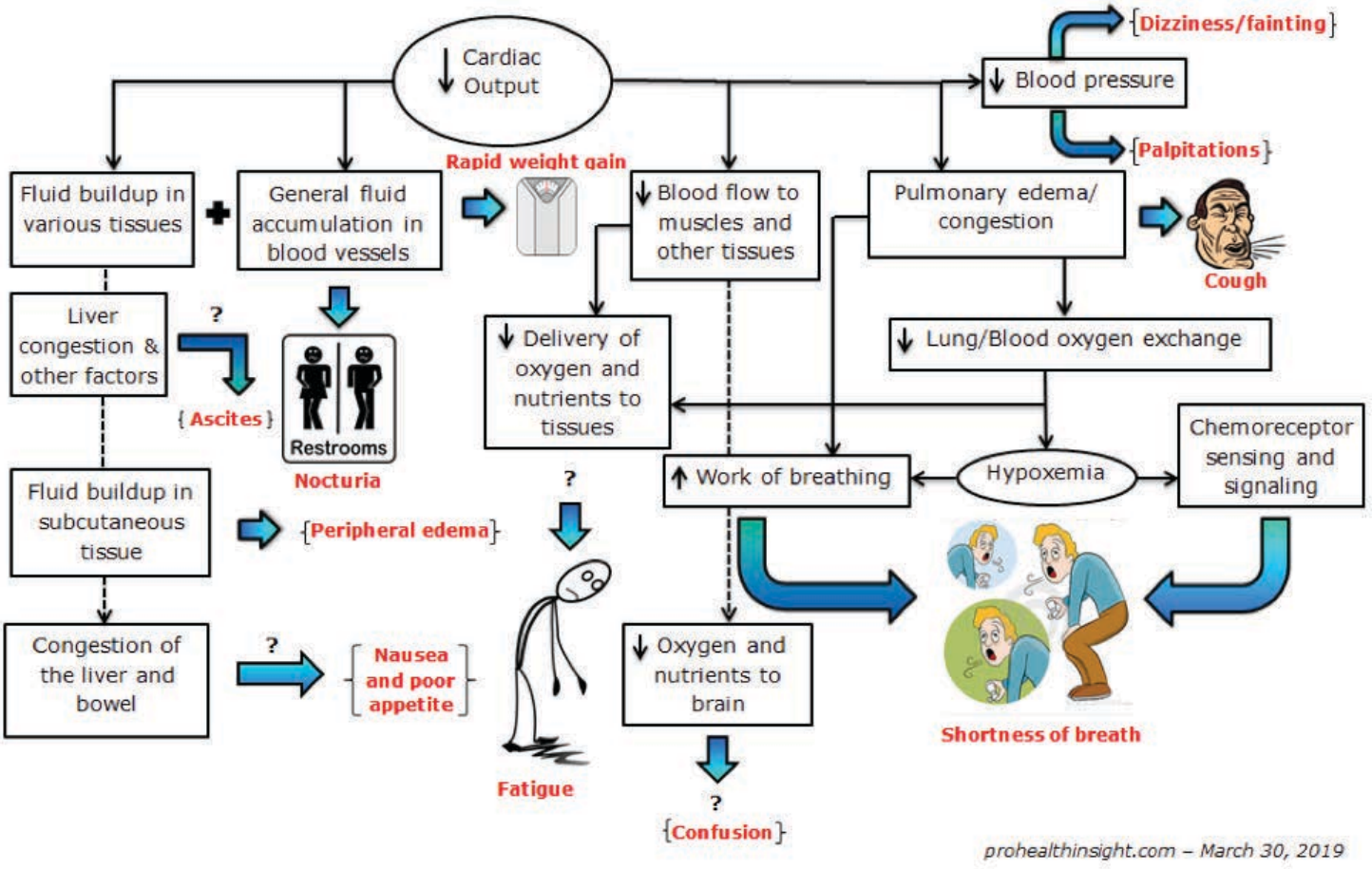
What is it?

- It is a hormonal system that regulates blood pressure, fluid and electrolyte balance, as well as systemic vascular resistance.
- The goal of the Renin-Angiotensin-Aldosterone System is to maintain normal blood pressure.

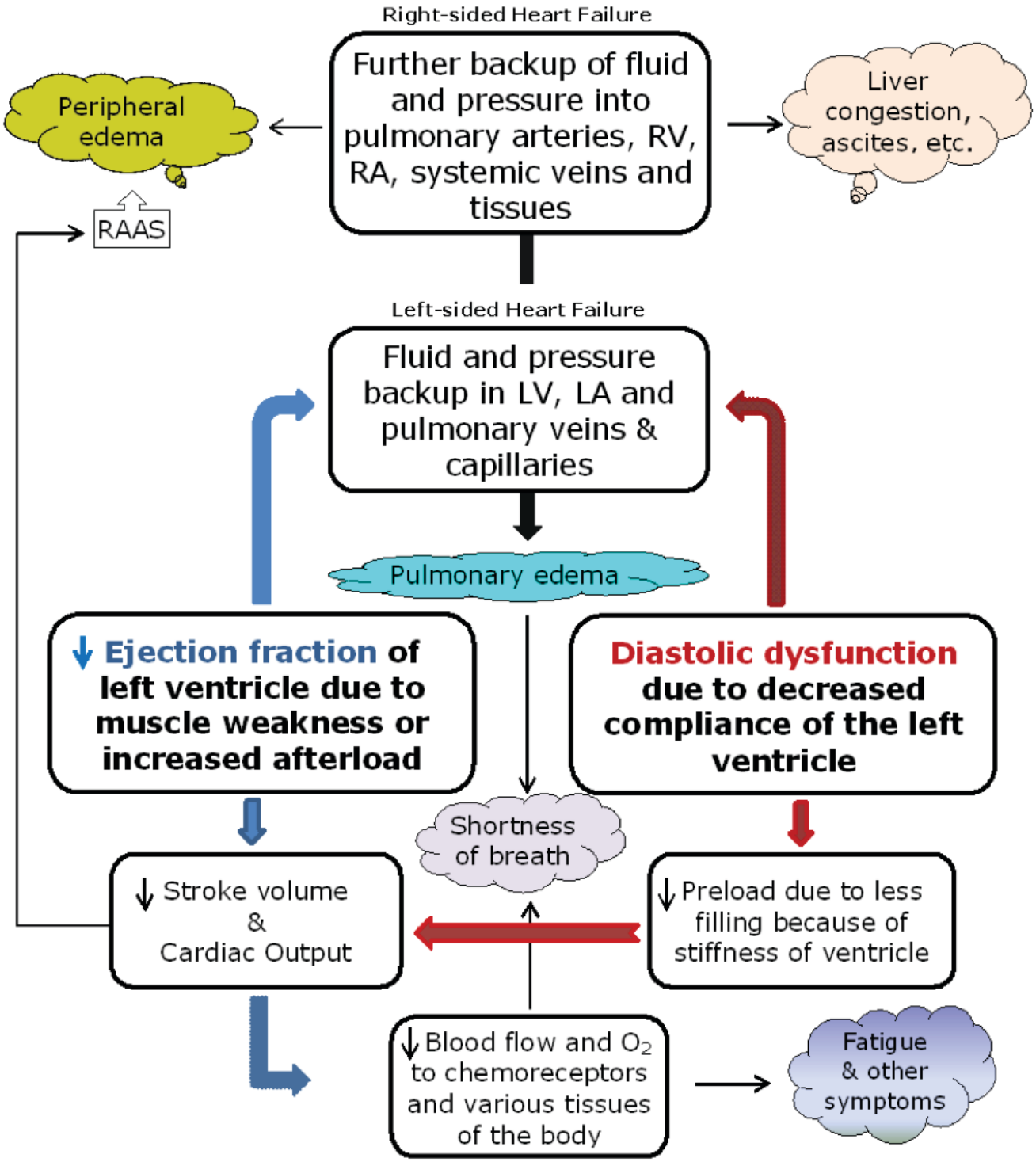
How it Works:

- When blood volume or sodium levels in the body are low, or blood potassium is high, cells in the kidney release the enzyme renin.
- Renin converts angiotensinogen, which is produced in the liver, to the hormone angiotensin I.
- An enzyme known as the angiotensin converting enzyme (ACE), found in the lungs, metabolizes angiotensin I into angiotensin II.
- Angiotensin II causes blood vessels to constrict and blood pressure to increase.
- Angiotensin II stimulates the release of the hormone aldosterone in the adrenal glands which causes the renal tubules to retain sodium and water and excrete potassium.
- Together, angiotensin II and aldosterone work to raise blood volume, blood pressure, and sodium levels in the blood to restore the balance of sodium, potassium, and fluids.
- The renin-angiotensin system becoming overactive results in consistent high pressure.
- Medications such as ACE inhibitors block aldosterone from retaining water and blocks vasoconstriction which lowers blood pressure.

CONGESTIVE HEART FAILURE SYMPTOMS AND PATHOPHYSIOLOGY



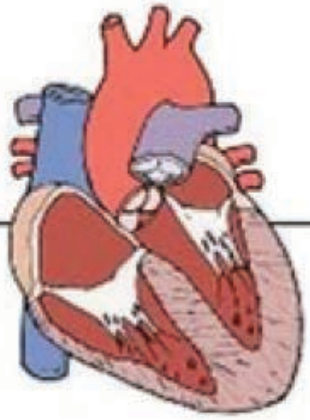
CONGESTIVE HEART FAILURE
BASIC PATHOPHYSIOLOGY



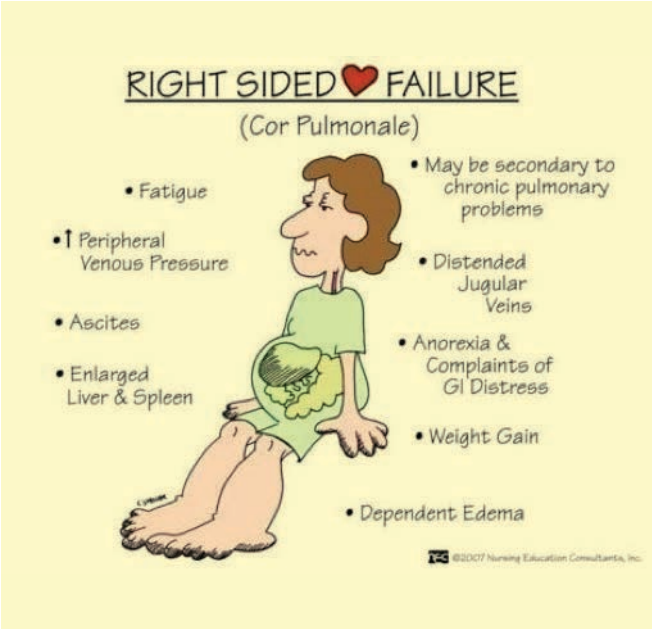
LA = left atrium; **LV** = left ventricle; **RA** = right atrium; **RV** = right ventricle; RAAS = renin-angiotensin-aldosterone system

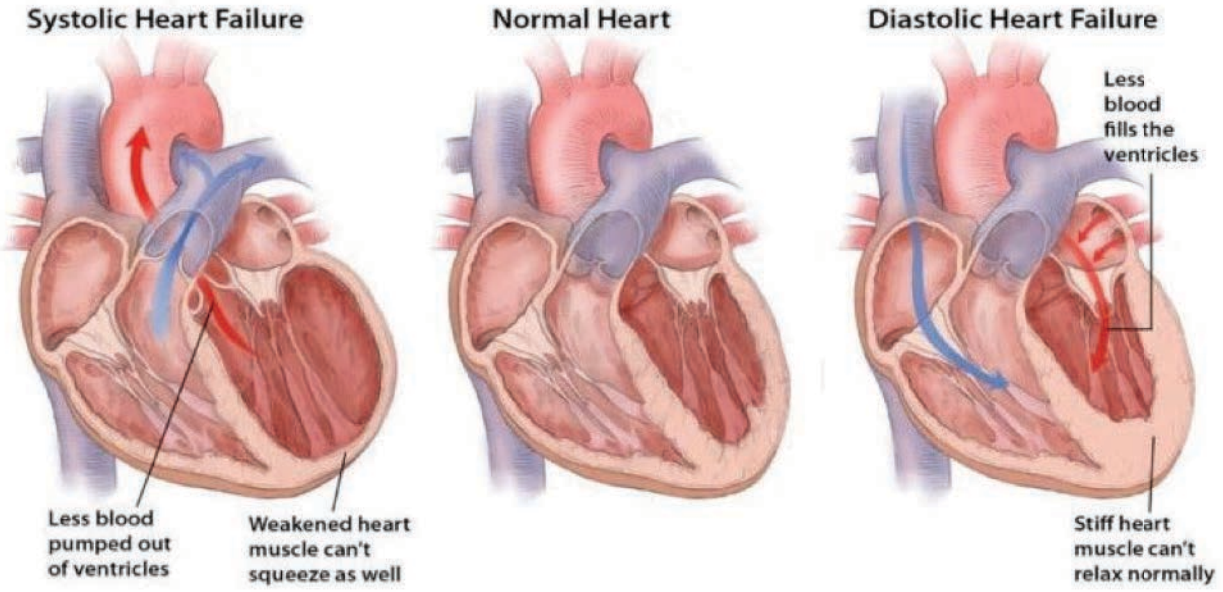
Prohealthinsight.com *- Feb. 22, 2019*

RIGHT VS. LEFT HEART FAILURE

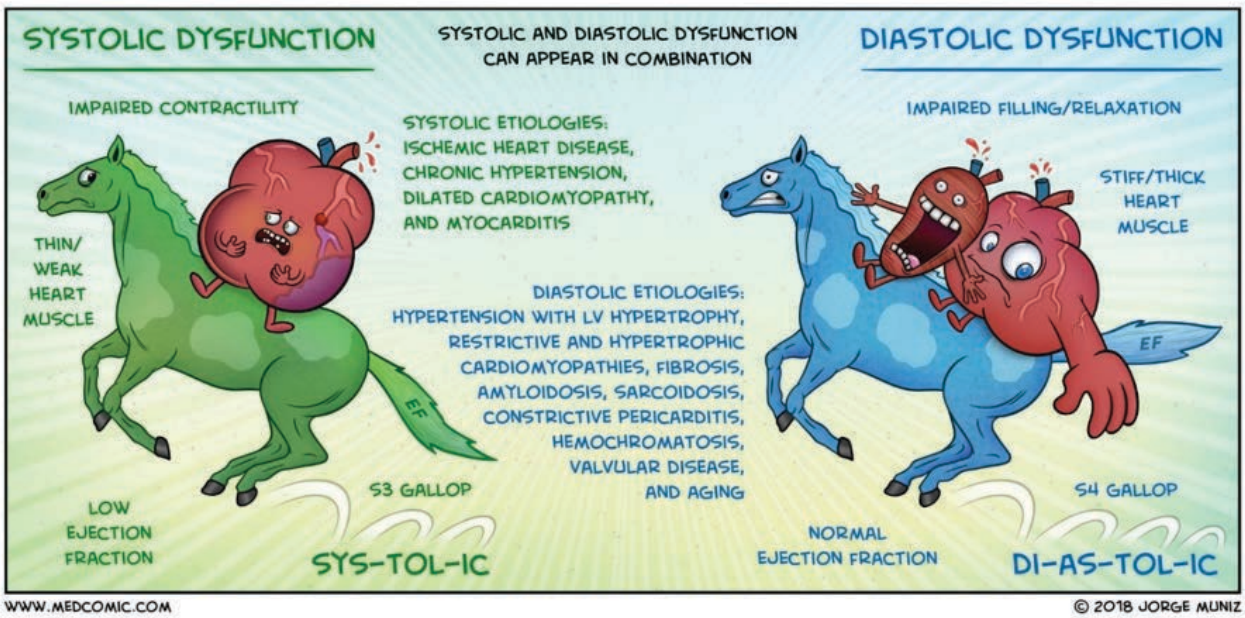


- Right heart failure
  - Congestion of peripheral tissues
    - Dependent edema and ascites
    - Liver congestion
      - Signs related to impaired liver function
    - GI tract congestion
      - Anorexia, GI distress, weight loss
- Left heart failure
  - Decreased cardiac output
    - Activity intolerance and signs of decreased tissue perfusion
  - Pulmonary congestion
    - Impaired gas exchange
      - Cyanosis and signs of hypoxia
    - Pulmonary edema
      - Cough with frothy sputum
      - Orthopnea
      - Paroxysmal nocturnal dyspnea





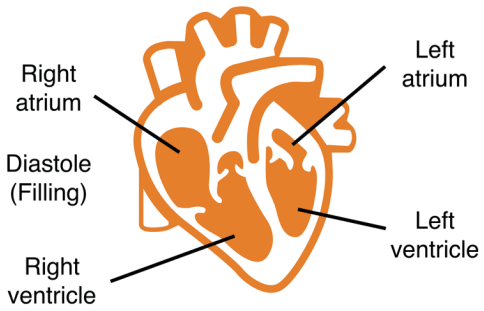
- **systolic heart failure** (pumping problem)
  - the inability of the heart to contract enough to provide blood flow forward
  - causes problems with contraction and ejection of blood
- **diastolic heart failure** (filling problem)
  - the inability of the left ventricle to relax normally, resulting in fluid backing up into the lungs
  - Diastolic failure leads to problems with heart relaxation and filling with blood



UNDERSTANDING EJECTION FRACTION IN HEART FAILURE

Ejection fraction (or EF) is a medical measure of the proportion of blood being pumped out of the heart each time it contracts.

EF values are always **percentages** so, for example, an ejection fraction of 50 means that 50% of blood is being pumped from the heart with each beat.[1]



The ventricles fill normally with blood

In a **healthy heart**, the walls of the ventricles are **flexible**, allowing blood to fill the ventricles properly before being pumped out. The chamber walls are also **strong**, so the right amount of blood can be pumped around the body.

A normal EF is generally considered to be between **50 and 70%**.

The enlarged ventricles fill with blood

A rapid or recent **reduction in EF**, or a lower percentage of blood being pumped out, can be an indicator of new or worsening heart failure. This is also known as Heart Failure with Reduced Ejection Fraction (HFrEF).

In heart failure with **reduced ejection fraction** the ventricles are unable to contract normally because they are **enlarged and 'flabby'** – this means the heart struggles to pump enough blood around the body. Patients with this form of heart failure will have a **lower-than-normal EF**.

This is generally indicated by an EF of **less than 40%**.

The stiff ventricles fill with less blood than normal

It is however possible to have a normal EF value and still receive a diagnosis of heart failure. This is also called heart failure with **normal ejection fraction** or Heart Failure with Preserved Ejection Fraction (HFpEF).

In heart failure with **preserved ejection fraction**, the heart contracts normally. However, the ventricles become **stiff** and are unable to fill properly.[1] People with this type of heart failure will have a **normal** (or preserved) **EF**, but less blood is pumped out because not enough blood can enter the heart chambers first.

American Heart Association.
Rise Above Heart Failure®

HF and Your Ejection Fraction Explained

The Ejection Fraction compares the **amount of blood in the heart** to the **amount of blood pumped out.** The fraction or percentage helps describe how well the heart is pumping blood to the body.

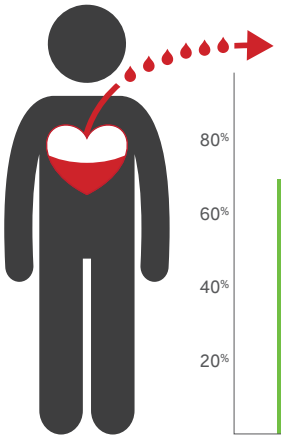
EJECTION FRACTION =

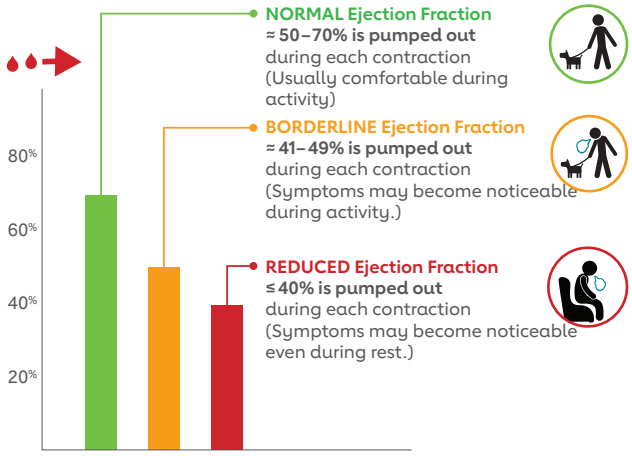
amount of blood **pumped out**

amount of blood **in chamber**

Source: 2013 ACCF/AHA Guidelines for the Management of Heart Failure
Source: http://www.ncbi.nlm.nih.gov/pubmed/22172436

How much blood is pumped out?





NORMAL Ejection Fraction
≈ 50–70% is pumped out during each contraction (Usually comfortable during activity)

BORDERLINE Ejection Fraction
≈ 41–49% is pumped out during each contraction (Symptoms may become noticeable during activity.)

REDUCED Ejection Fraction
≤ 40% is pumped out during each contraction (Symptoms may become noticeable even during rest.)

It is also possible to have a diagnosis of heart failure with a seemingly normal (or preserved) ejection fraction of greater than or equal to 50%.

With the proper care and treatment, many patients are able to improve their ejection fraction and live a longer and healthier life. Talk with your healthcare provider about your options.

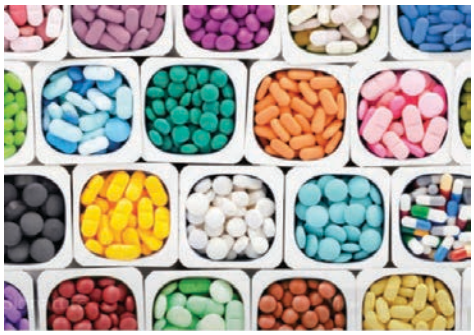
www.RiseAboveHF.org

Supported by NOVARTIS

PHARMACOLOGY MANAGEMENT

Cardiovascular diseases may be treated with one or more of the following classes of medications:

- Cardiac glycosides
- Nitrates
- Beta blockers
- Calcium channel blockers
- Diuretics
- ACE inhibitors
- Anticoagulants/Antiplatelets
- Antilipemics



CARDIAC GLYCOSIDES

Cardiac glycosides, also called digitalis glycosides, are a group of drugs that inhibit the sodium-potassium pump, causing cardiac muscle fibers to contract more efficiently.

- Mode of Action
  - Increases force of cardiac contraction, cardiac output, and tissue perfusion
  - Decreases ventricular rate
- Indications
  - CHF
  - Atrial tachycardia, flutter, or fibrillation
- Care Considerations
  - Check apical pulse before administration. Hold according to physician-ordered parameter.
  - Can lead to bradycardia and toxicity (anorexia, diarrhea, nausea & vomiting, confusion) – obtain labs for drug levels as ordered.
- Examples: Digoxin (Lanoxin), Digitoxin, Ouabain

NITRATES

Nitrates are a medications that affect the blood vessels in the venous circulation and coronary arteries.

- Mode of action
  - Act directly on the smooth muscle of blood vessels, causing relaxation and dilation
  - Decrease myocardial demand for oxygen
- Indication
  - Angina pectoris (anginal pain)
- Care Considerations
  - Can cause hypotension
  - Sublingual onset is 1-3 minutes, peaks in 4 minutes
- Examples: Nitrostat, Transderm-Nitro patch, Isordil, Imdur

BETA BLOCKERS

Beta blockers decrease the effects of the sympathetic nervous system.

- Mode of action
  - Block beta1 (cardiac) and beta2 (pulmonary) adrenergic receptor sites
  - Decrease heart rate, reduce force of contractions
- Indications
  - Cardiac dysrhythmias
  - Hypertension
  - Angina pectoris
  - Myocardial infarction
- Care Considerations
  - Can cause bradycardia, confusion, drowsiness, fatigue, vertigo, dry mouth
- Examples: Propranolol (Inderal), Lopressor, Atenolol

CALCIUM CHANNEL BLOCKERS

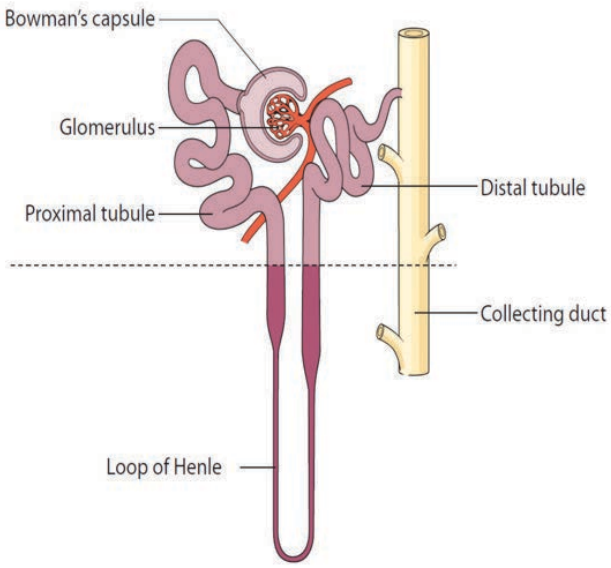
Calcium channel blockers prevent calcium from entering cells of the heart and blood vessel walls.

- Mode of Action
  - Decrease cardiac contractility and the workload of the heart
  - Relax coronary arteries, decreasing oxygen demand
- Indications
  - Angina pectoris
  - Hypertension
  - Cardiac dysrhythmias
- Care Considerations
  - Can cause bradycardia and hypotension
- Examples: Norvasc, Cardizem, Procardia, verapamil HCl

DIURETICS

Diuretics produce increased urine flow by inhibiting sodium and water reabsorption from the kidney tubules, resulting in decreased fluid volume and subsequent decrease in BP. They are categorized according to the segment of the tubules on which they act.

- Mode of Action
  - Thiazide and thiazide-like: act on the distal convoluted renal tubule to promote sodium, chloride, and water excretion, decreasing preload and cardiac output
  - Loop: inhibit sodium and water reabsorption from the loop of Henle, causing rapid diuresis and decreasing vascular fluid volume, cardiac output, and blood pressure
  - Potassium sparing: act on the collecting tubules to promote sodium and water excretion and potassium retention, increasing urine output and decreasing fluid overload
- Indications
  - Hypertension
  - Peripheral edema
  - Edema associated with CHF
  - Renal disease
  - Pulmonary edema
- Care Considerations
  - Require supplementation with potassium, unless potassium sparing
  - May cause dehydration, hypotension, and dizziness
  - Offer once daily dosing in the morning to avoid sleep disturbance resulting from nocturia
- Examples
  - Thiazide and thiazide-like: hydrochlorothiazide, Zaroxolyn, HydroDiuril
  - Loop: bumetanide, furosemide, torsemide
  - Potassium sparing: spironolactone, Dyazide, triamterene



ACE INHIBITORS

ACE inhibitors inhibit angiotensin-converting enzyme (ACE), which in turn inhibits the formation of angiotensin II (vasoconstrictor) and blocks the release of aldosterone.

- Mode of Action
  - Inhibits vasoconstriction, resulting in vascular relaxation.
  - Ultimately blocks aldosterone, causing sodium excretion and retention of potassium.
- Indications
  - Hypertension
  - Heart failure
- Care Considerations
  - Can cause hypotension, particularly in those also taking diuretics.
  - Can cause dizziness, nocturia, and hyperkalemia
- Examples: captopril, Lotensin, Vasotec, lisinopril

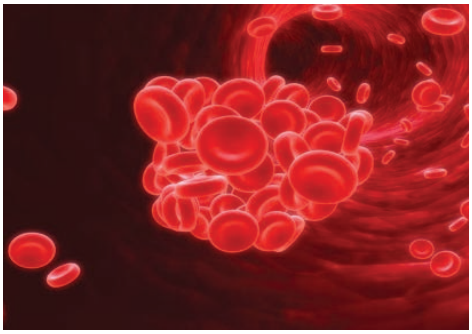
ANTICOAGULANTS / ANTIPLATELETS

Anticoagulant and antiplatelet drugs suppress thrombosis.

- Mode of Action
  - Anticoagulants: prevent formation of clots that inhibit circulation, either by inhibiting thrombin or depressing the synthesis of vitamin K clotting factors
    - Examples: heparin, low molecular weight heparin (Lovenox), warfarin
  - Antiplatelets: prevent arterial thrombosis by preventing the clumping together of platelets to form a clot
    - Examples: aspirin, clopidogrel, Aggrenox, Ticlid
- Indications
  - Thromboembolism
  - Postoperative DVT prevention
  - Prevention of thrombosis after CVA or MI
  - Prevent/reduce risk of MI or stroke

Anticoagulant and antiplatelet medications are associated with a high risk for bleeding.

- Care Considerations
  - Monitor for bleeding (i.e. bleeding from the mouth, nose (epistaxis), urine (hematuria), stool, and skin (petechiae, ecchymosis purpura).
  - Obtain labs as ordered (i.e. PT/INR, PTT, CDC, platelets).
  - Report lab results timely to physician, especially critical values.
  - Avoid large amounts of green leafy vegetables, which are rich in vitamin K (antagonist to anticoagulants
  - Use soft toothbrush. Shave with electric shaver
  - Increased risk of bleeding when also taking these types of medications:
    - NSAIDS
    - Sulfonamides
    - Phenytoin
    - Tagamet
    - Allopurinol
    - Hypoglycemic drugs



ANTILIPEMICS

Antilipemics, also called hypolipidemics or antihyperlipemics, decrease blood lipid concentrations. Lipids composed of cholesterol, triglycerides, and phospholipids contribute to atherosclerotic plaque in blood vessels.

- Mode of Action
  - Inhibit cholesterol synthesis in the liver by inhibiting enzymes needed for cholesterol biosynthesis
- Indications
  - High cholesterol
- Care Considerations
  - Can cause constipation and peptic ulcer – administer with sufficient water or food to alleviate GI discomfort
  - Can cause an increase in liver enzyme levels – monitor lab tests for liver function, as ordered
- Examples: Questran, Colestid, Lipitor, simvastatin, Prevachol

CARDIAC BIOMARKERS (LABS)

What are they?

- Cardiac biomarkers show up in the blood after the heart has been under severe stress, such as a heart attack, because the heart isn't getting enough oxygen rich blood.
- Lab tests measure the levels of cardiac biomarkers in the blood. These marker's include enzymes, hormones, and proteins such as Troponin, CK, CK-MB, and Myoglobin.
- Levels can also be high for other reasons such as heart and respiratory failure

TROPONIN

What is it?

Troponin is a group of proteins found in the skeletal and heart muscle fibers that regulate muscular contractions.

- Normally, troponin is present in very small to undetectable quantities in the blood. When there is damage to the heart muscle cells, troponin is released into the blood. The more damage there is, the greater the concentration in the blood.
- A troponin lab test measures the amount of cardiac-specific troponins circulating in the blood stream following a cardiac event.
- Troponin in released with very small areas of myocardial damage as early as 1 to 3 hours after injury or event, and levels return to normal withing 3 to 10 days.
- Normal lab findings: Troponin < 0.04

BRAIN NATRIURETIC PEPTIDE (BNP)

What is it?

B-type Natriuretic Peptides (BNP) blood test measures the levels of the BNP hormone in the blood. A BNP test can help rule out heart failure.

BRAIN NATRIURETIC PEPTIDE (BNP) - CONT'D.

- They help detect, diagnose, and in some cases evaluate the severity of heart disease, including Congestive Heart Failure.
- BNP is continually produced in small quantities in the heart and released in large quantities when the heart senses that it needs to work harder. This supports fluid retention and volume expansion in the arteries and veins.
- BNP and another heart hormone, called atrial natriuretic peptide (ANP), work together to keep veins and arteries widened, or dilated. This allows blood to easily pass through and prevents clots from forming. BNP and ANP also help kidneys more easily remove fluid and salt from the body.
- When a patient has Congestive Heart Failure , the heart can't pump blood properly throughout the body because the walls of the heart chambers, known as the ventricles, become tense or too weak. This affects pressure and fluid levels in the heart and throughout the body. When this happens, heart cells produce extra BNP to help maintain the balance of fluids in your body cells and regulate your blood pressure.
- In general, the more serious your heart failure, the higher your levels of BNP will be. But test results vary by age, sex, and body mass index. Normal values tend to go up with age. They also tend to be higher in women and lower in men. Both men and women who are obese tend to have lower levels.

A normal level of NT-proBNP, based on Cleveland Clinic's Reference Range is:

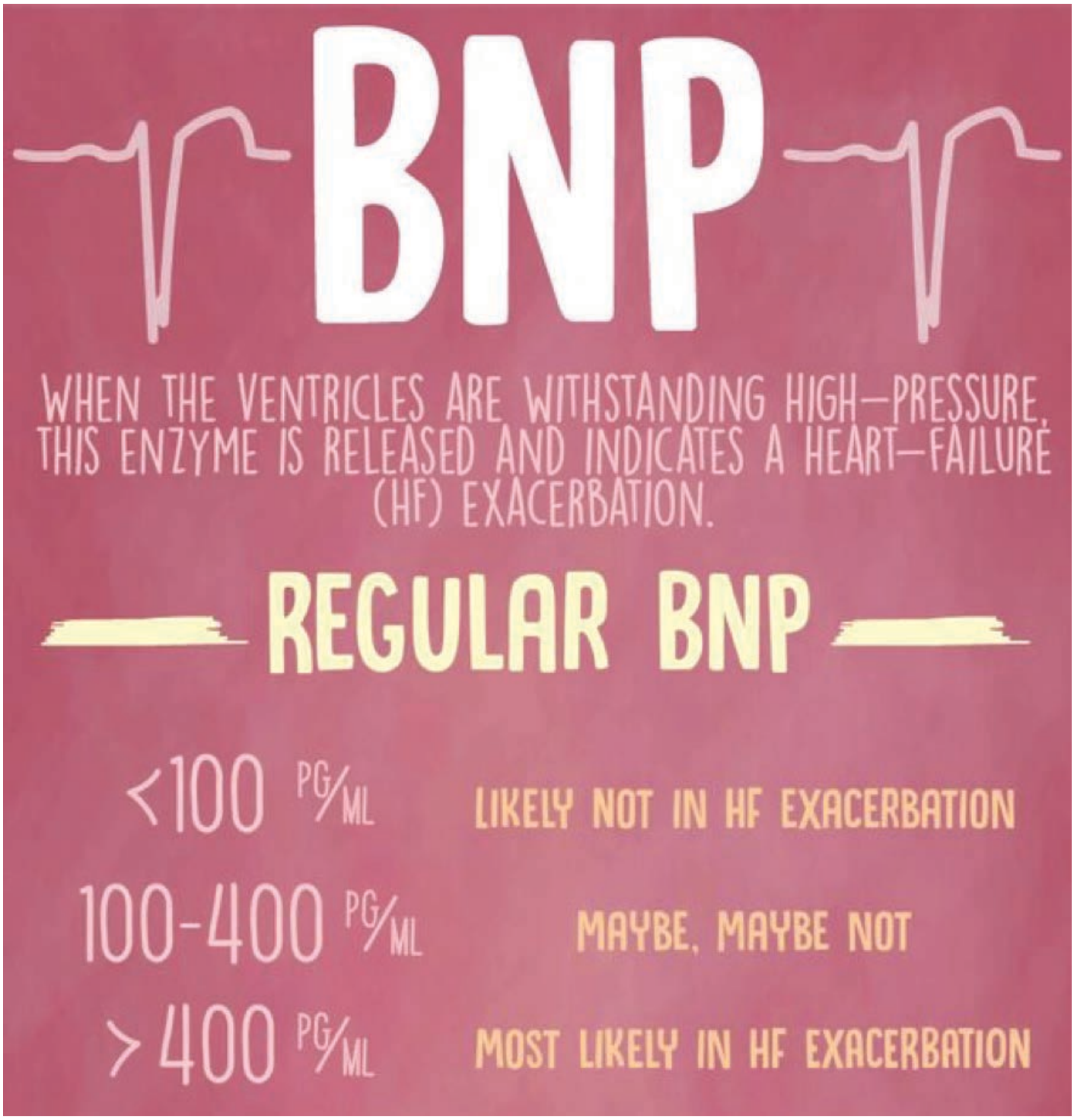
- Less than 125 pg/mL for patients aged 0-74 years
- Less than 450 pg/mL for patients aged 75-99 years

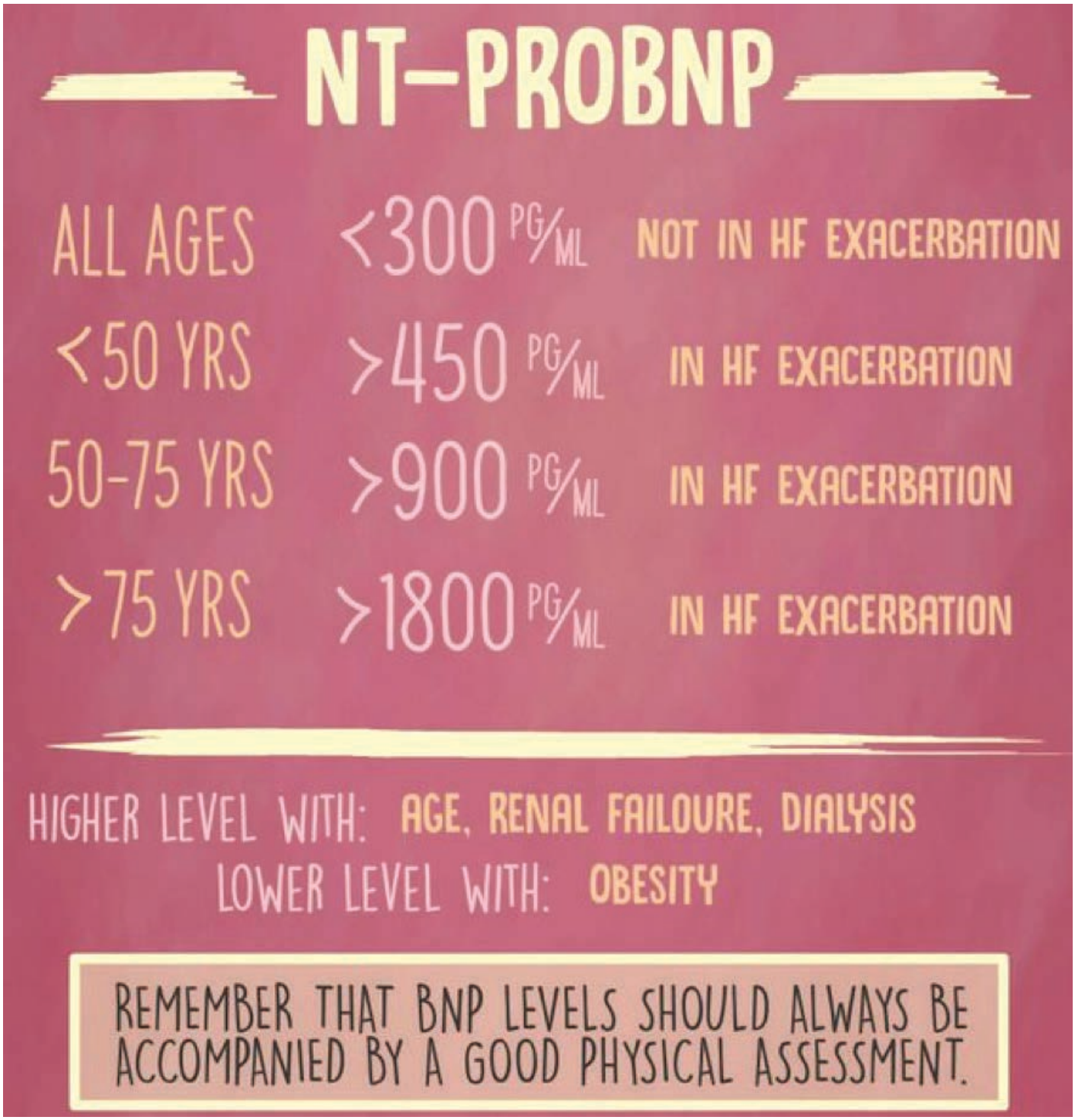
Other Cardiac Biomarkers: Creatine Kinase (CK), Creatine Kinase-MB (CK-MB) Myoglobin

To confirm a heart failure diagnosis, the physician may also recommend the following tests:

- CBC, CMP
- Chest X-ray
- Echocardiogram
- Electrocardiogram (EKG)
- Cardiac Catheterization

BRAIN NATRIURETIC PEPTIDE (BNP)




NT-PROBNP
ALL AGES <300 PG/ML NOT IN HF EXACERBATION
<50 YRS >450 PG/ML IN HF EXACERBATION
50-75 YRS >900 PG/ML IN HF EXACERBATION
>75 YRS >1800 PG/ML IN HF EXACERBATION
HIGHER LEVEL WITH: AGE, RENAL FAILOURE, DIALYSIS
LOWER LEVEL WITH: OBESITY
REMEMBER THAT BNP LEVELS SHOULD ALWAYS BE ACCOMPANIED BY A GOOD PHYSICAL ASSESSMENT.

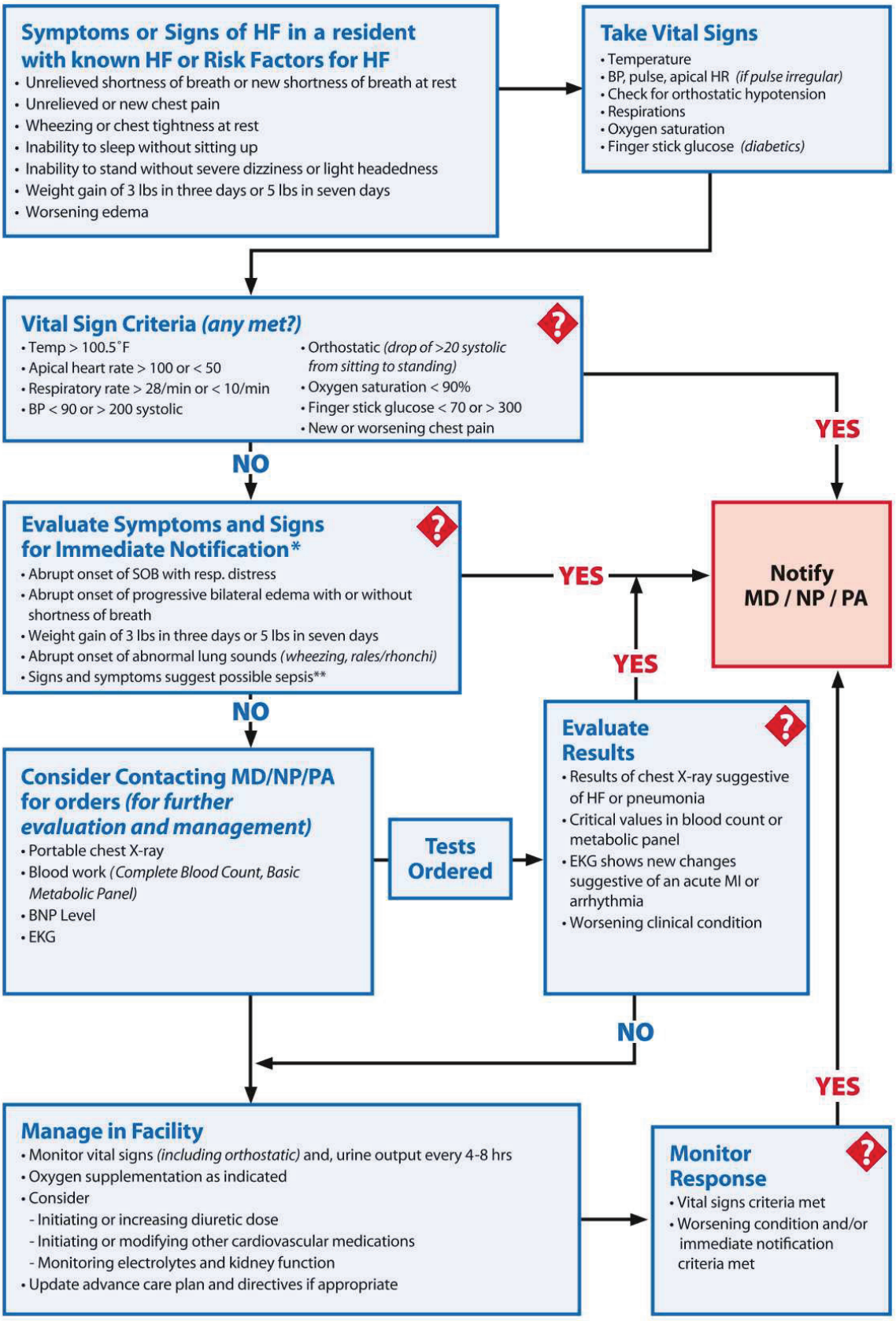
THERAPEUTIC INTERVENTIONS

Give oxygen as indicated by patient symptoms, oxygen saturation and ABGs.	Makes more oxygen available for gas exchange, assisting to alleviate signs of hypoxia and subsequent activity intolerance.
Implement strategies to treat fluid and electrolyte imbalances.	Decreases the risk for development of cardiac output due to imbalances.
Administer cardiac glycoside agents, as ordered, for signs of left sided failure, and monitor for toxicity.	Digitalis has a positive isotropic effect on the myocardium that strengthens contractility, thus improving cardiac output.
Encourage periods of rest and assist with all activities.	Reduces cardiac workload and minimizes myocardial oxygen consumption.
Assist the patient in assuming a high Fowler's position.	Allows for better chest expansion, thereby improving pulmonary capacity.
Reposition patient every 2 hours	To prevent occurrence of bed sores
Instruct patient to get adequate bed rest and sleep Encourage rest, semirecumbent in bed or chair. Assist with physical care as indicated.	To promote relaxation to the body Physical rest should be maintained during acute or refractory HF to improve efficiency of cardiac contraction and to decrease myocardial oxygen demand/ consumption and workload.
Provide quiet environment: explain therapeutic management, help patient avoid stressful situations, listen and respond to expressions of feelings.	Psychological rest helps reduce emotional stress, which can produce vasoconstriction, elevating BP and increasing heart rate.
Provide bedside commode. Have patient avoid activities eliciting a vasovagal response (straining during defecation, holding breath during position changes).	Commode use decreases work of getting to bathroom or struggling to use bedpan. Vasovagal maneuver causes vagal stimulation followed by rebound tachycardia, which further compromises cardiac function.

THERAPEUTIC INTERVENTIONS - CONT'D.

Elevate legs, avoiding pressure under knee. Encourage active and passive exercises. Increase activity as tolerated.	Decreases venous stasis, and may reduce incidence of thrombus or embolus formation.
Check for calf tenderness, diminished pedal pulses, swelling, local redness, or pallor of extremity.	Reduced cardiac output, venous pooling, and enforced bed rest increases risk of thrombophlebitis.
Withhold digitalis preparation as indicated, and notify physician if marked changes occur in cardiac rate or rhythm or signs of digitalis toxicity occur.	Incidence of toxicity is high (20%) because of narrow margin between therapeutic and toxic ranges. Digoxin may have to be discontinued in the presence of toxic drug levels, a slow heart rate, or low potassium level.
Administer supplemental oxygen as indicated.	Increases available oxygen for myocardial uptake to combat effects of hypoxia.

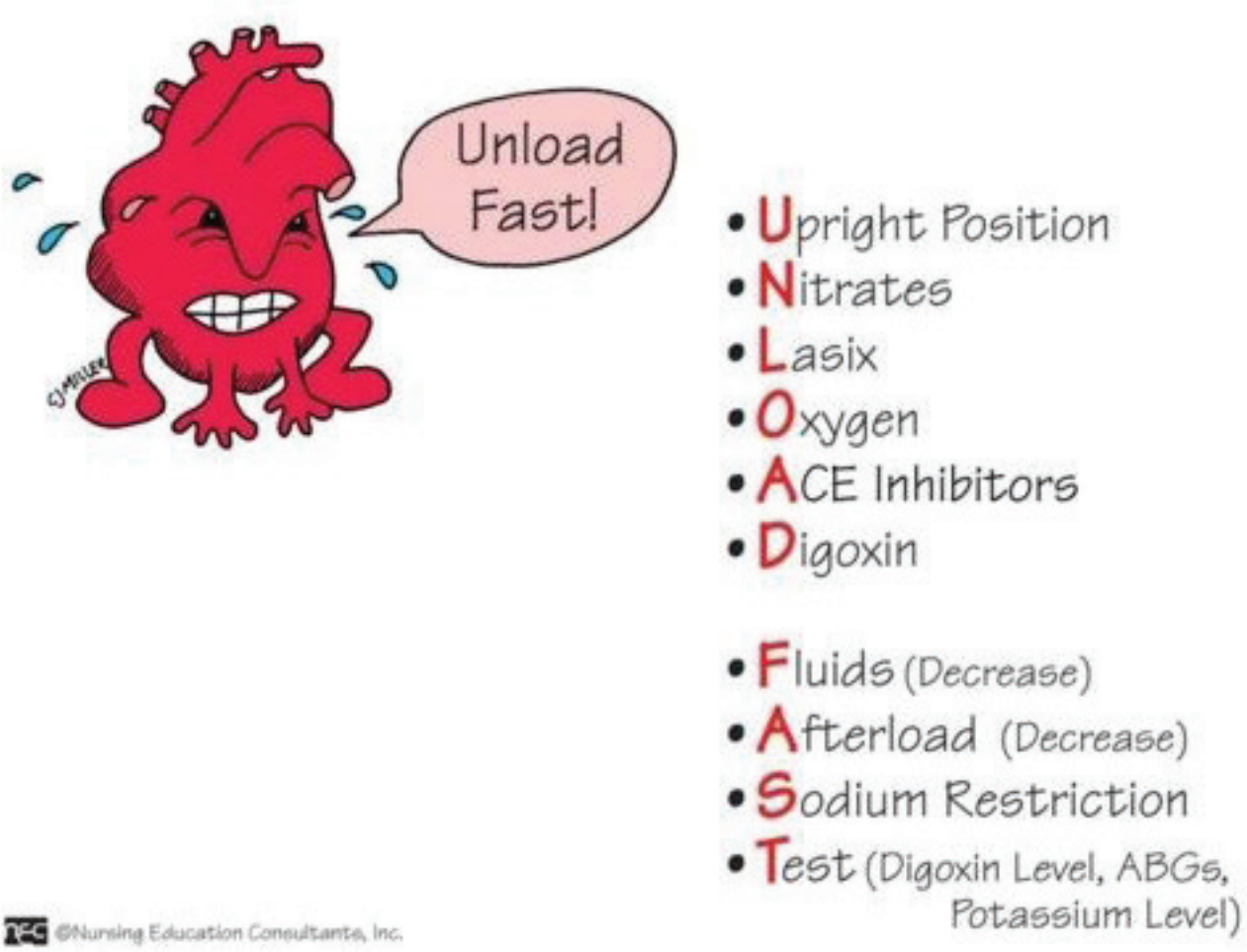
CARE PATH - SYMPTOMS OF HEART FAILURE (HF)



** Refer also to other INTERACT Care Paths as indicated by symptoms and signs*

*** If sepsis is been considered, refer to INTERACT Guidance on Possible Sepsis and INTERACT Guidance on Infections*

TREATING CONGESTIVE HEART FAILURE



REFERENCES

- https://www.thecompliancestore.net/
- https://prohealthinsight.com/
- https://medcomic.com/
- https://www.heart.org/en/health-topics/heart-failure
- https://pathway-interact.com/
- https://nurseslabs.com/heart-failure-nursing-care-plans/

NURSE-PHYSICIAN COMMUNICATION

Report the following abnormal findings to physician:

- Evidence of acute distress: chest pain, labored breathing, new/worsening SOB.
- Acute changes in level of consciousness or mental status: lethargy, confusion.
- New or worsening edema.
- Weight change of 3 or more pounds in one week or less in resident with heart failure.
- New onset bradycardia or tachycardia. Also report high or low heart rates in accordance with physician-ordered parameters.
- Blood pressure: systolic over 180 and/or diastolic over 120. Also report abnormal BP
- in accordance with physician-ordered parameters.
- Abnormal drug levels (i.e. Digoxin).
- Abnormal lab values (particularly PT/INR, potassium, liver function).

NOTES

1300 Beard Street
Port Huron, MI 48060
810-982-9500

PH-MRA-29 (12.19)