

Finding problems fast: focused assessment to prevent complications

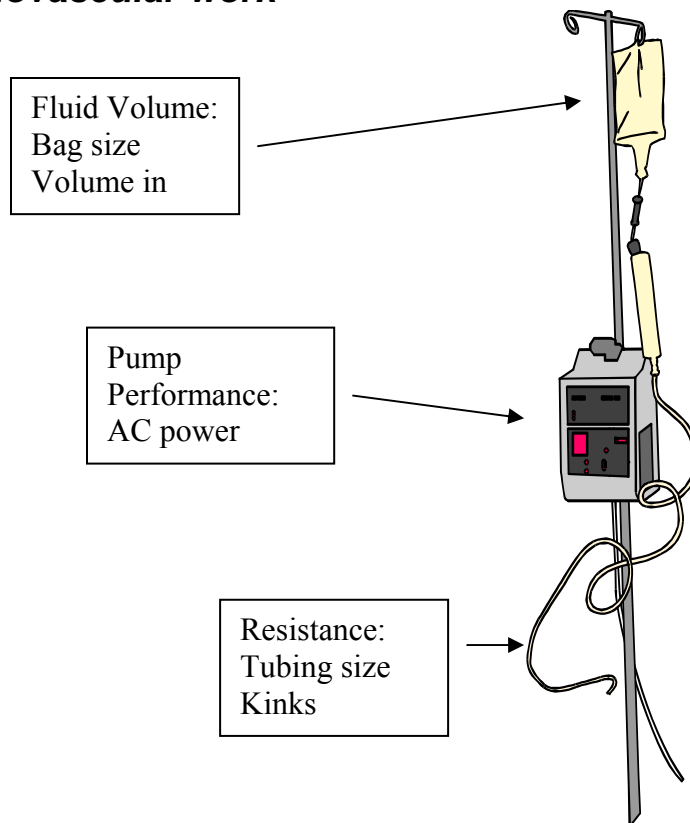
Cardiovascular Assessment

Nearly one million people die of cardiovascular disease each year in the US, and many more people have cardiovascular disease in some form or another. It is likely that most of your patients have some form of disease that affects their cardiovascular system. Therefore, thorough cardiovascular assessment could lead to early detection and treatment of potential problems. The first step in the process is to determine your patient's risk. A thorough assessment of her history is essential.

History

1. Personal
 - a. Chronic disease
 - b. Medications and herbal remedies
 - c. Social history
2. Family
 - a. Atherosclerotic disease
3. Present Illness
 - a. Chest pain
 - b. Shortness of breath
 - c. Nausea / vomiting

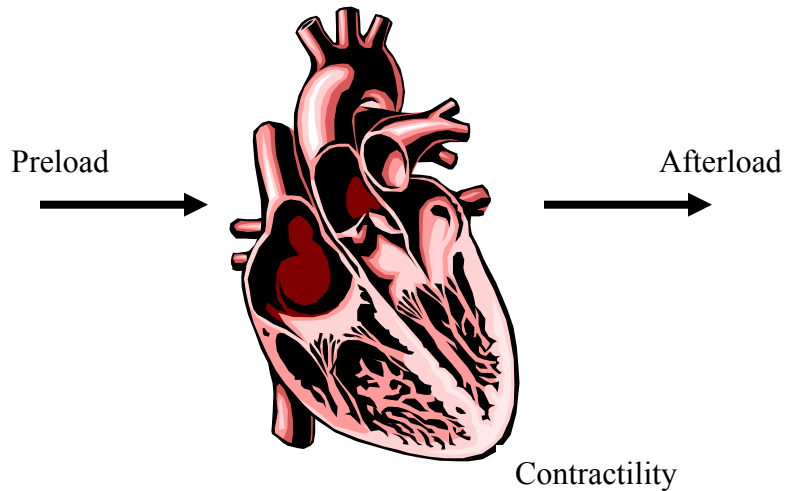
Factors affecting cardiovascular work



Hemodynamics

To understand cardiovascular function, it is essential to frame your assessment in hemodynamic terms. Physicians talk in hemodynamics. In order to communicate effectively with physicians, you must understand your patient's condition and be able to express it in hemodynamic terms.

By applying the principles of the IV pump it is easy to understand and apply hemodynamics to your patient's condition. Think of the IV pump and apply the principles to the diagram below.

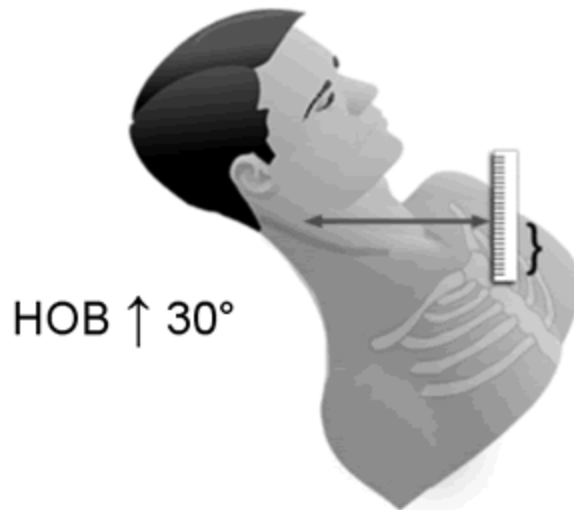


Component	Factors affecting	Assessment
Preload	Fluid volume	I & O Weight Jugular veins
Contractility	Fluid volume Oxygenation Resistance	Heart rate Systolic blood pressure Organ perfusion: -Brain -Kidneys Peripheral perfusion -Capillary refill -Pulses
Afterload	Arterial vascular resistance	Diastolic blood pressure Skin temperature and color

Assessing Jugular Veins

Assessment of the jugular veins can accurately assess your patient's fluid volume. Add JVD assessment to your assessment of I&O, patient weight and the presence of an S3 heart sound and you have a powerful assessment of your patient's preload.

1. JVD accurately predicts fluid overload >80% of the time*
2. Presence of JVD & S3 predicts fluid overload >86% of the time*
3. External jugular veins
 - a. HOB \uparrow 30 °
 - b. Normal crest: just above clavicle
 - c. Measurement of CVP
 - i. Second intercostal space
 - ii. Measure up to jugular crest
 - iii. Add 5 cm to obtain CVP
 - d. Non-visibility in supine position suggests hypovolemia



4. Right internal jugular
 - a. Visible, not palpable
 - b. Look for pulsations that:
 - iv. Increase on inspiration
 - v. Have a sharp descend
 - vi. Disappear with pressure

Presence of right internal jugular pulsation
indicates fluid volume overload.

*Butman, SM, et al. (1993). Bedside cardiovascular examination in patients with severe chronic heart failure: Importance of rest or inducible jugular venous distension. J Am Coll Cardiol, 22(4): 968-74.

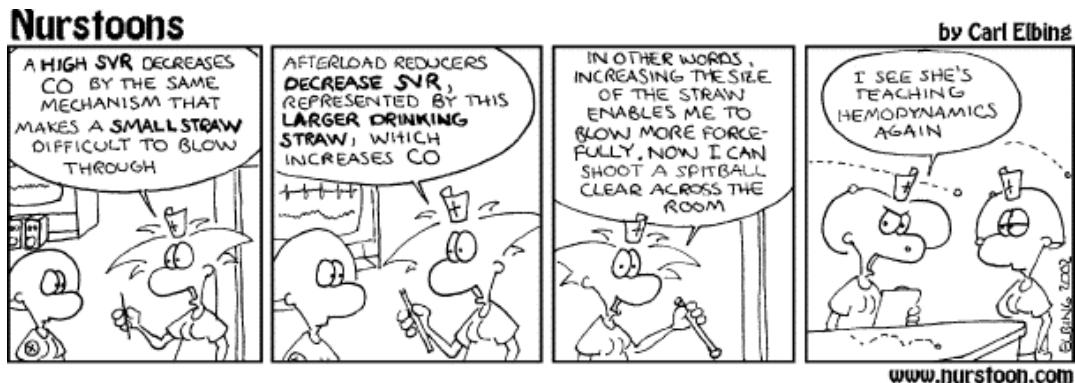
Assessing Pulses

By assessing the peripheral pulses, you will be able to assess cardiac function and peripheral vascular function.

1. Rate and rhythm
 - a. Rate: SNS stimulation from low cardiac output
 - b. Rhythm: detect abnormal conduction and arrhythmias
2. Character and quality
3. Peripheral pulses
 - a. 0 Absent
 - b. +1 Diminished, weak
 - c. +2 Brisk, expected
 - d. +3 Increased
 - e. +4 Bounding





Abnormal Pulses	Cause
Weak, thready pulse	Shock
Bounding pulse	Hyperdynamic phase of sepsis Hypertension
Pulsus alternans (strong, weak)	Severe cardiac dysfunction
Pulsus paradoxus	Mechanical ventilation Air trapping (asthma, COPD) Cardiac tamponade

Bounding pulse with decreased diastolic blood pressure may signal early sepsis.



Chest Assessment

1. Functional Area Location: named for the valve they assess.

Area	Location		Abnormality
Aortic	2 nd ICS R sternal border		Aortic Stenosis S2 is loudest here
Pulmonic	2 nd ICS L sternal border		Pulmonary stenosis or regurgitation
Tricuspid	L lower sternal border		Tricuspid stenosis
Mitral	5 th ICS		Mitral stenosis or regurgitation S1 is loudest here

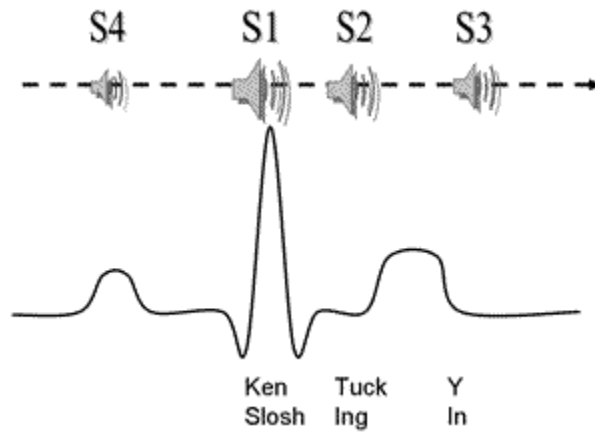
2. Palpation
 - a. Point of maximal impulse (PMI)
 - b. Mitral area, normal +2 pulse
 - c. Displaced to left = left ventricular enlargement
3. Heart Sounds

Normal heart sounds are produced by closure of the valves of the heart. Flow through the valves will affect the sound the valve makes. Thus, in situations of increased flow (exercise for example) the intensity of the heart sounds will be increased. In situations of low flow (shock for example) the intensity of the heart sounds will be decreased.

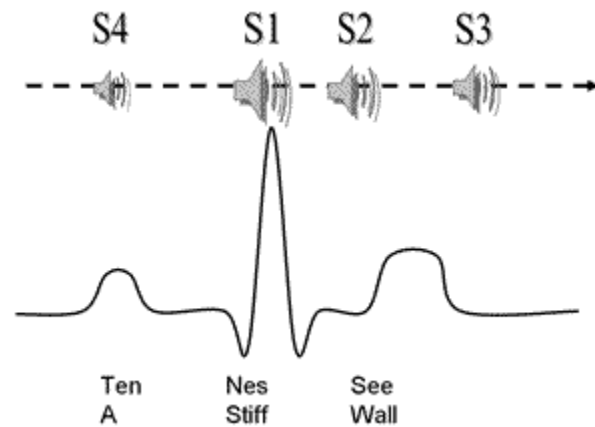
S1: The S1 sound is normally the first heart sound heard. See the diagram below for the location of S1 during the cardiac cycle. The S1 is best heard in the *mitral area*, and corresponds to closure of the mitral and tricuspid (AV) valves. A normal S1 is low-pitched and of longer duration than S2.

S2: The S2 sound is normally the second sound heard. The S2 is best heard over the *aortic area*, and corresponds to closure of the pulmonic and aortic valves. A normal S2 is higher-pitched and of shorter duration than S1.

- a. S1:
 - i. Mitral and tricuspid valve closure
 - ii. Best heard with diaphragm
- b. S2
 - i. Aortic and pulmonic valve closure
 - ii. Best heard with diaphragm
- c. S3
 - i. Indicates CHF
 - ii. Low pitched and soft
 - iii. Best heard with the bell



- d. S4
 - i. Indicates MI
 - ii. Low pitched and soft
 - iii. Best heard with the bell



4. Correlation with clinical condition
 - a. Age
 - i. Age where risk is increased?
 - b. Sex
 - i. Male vs. female?
 - c. Risk factors
 - i. History of atherosclerosis?
 - d. Clinical presentation
 - i. Chest pain

Problem	Pain characteristics
Acute coronary syndrome	Crushing, heavy
Pulmonary embolism	Pleuretic
Aneurysm	Boring through to back
Pericarditis	Sharp, worse in recumbent position
Pneumothorax	Pleuretic
Pneumonia	Pleuretic, localized

- ii. Shortness of breath
- iii. Nausea / vomiting
- iv. Palpitations
- v. Syncope

Epigastric pain could be GERD or AMI. Differential testing with nitroglycerine and a GI cocktail are not selective and specific.

Pulmonary Assessment

In order to understand how frequently your patients have pulmonary complications, just think about how many of them have diminished breath sounds in the bases of their lungs. On most floors that would be just about everyone. Contrary to popular belief, diminished breath sounds in the bases of the lungs are not normal findings. They indicate that your patient has developed atelectasis, and may suffer from additional complications as a result.

Atelectasis is the collapsing of dependent alveoli and can lead to pneumonia, sepsis, and acute respiratory distress syndrome. And although it is a natural phenomena from having our patients lying in bed, atelectasis is not a normal finding and deserves attention to pulmonary hygiene. Make sure that you are addressing this in your care and documentation.

In this program we will explore pulmonary assessment with the intention of finding pulmonary problems in their early stages and implementing interventions that prevent complications. Early identification of pulmonary problems and prompt initiation of interventions can decrease complications and improve mortality for your patients.

So, let's get started...

History

4. Personal
 - a. Chronic disease
 - b. Medications and herbal remedies
 - c. Social history
5. Family
 - a. Chronic or genetic disease
6. Present Illness
 - a. Dyspnea
 - b. Chest pain or tightness
 - c. Nocturnal dyspnea
 - d. Use of pulmonary medications at night
 - e. Morning symptoms

Factors affecting oxygenation

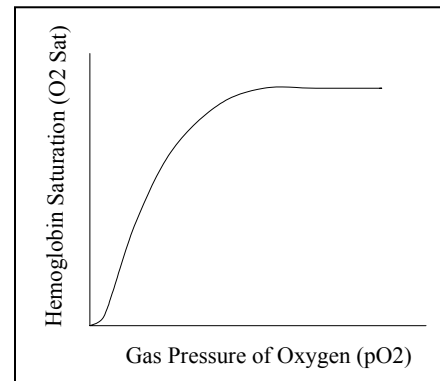
1. Three step process
 - a. Alveoli (FiO₂)
 - i. Assess breath sounds
 - ii. Work of breathing
 - iii. Maximize airflow delivery
 - iv. Prevent atelectasis
 - b. Interstitial space
 - i. pO₂:FiO₂
 - ii. Signs of dysfunction:
 1. Subjective ↑ in WOB
 2. ↑ RR
 - c. Capillary network
 - i. pO₂



Perfusion:

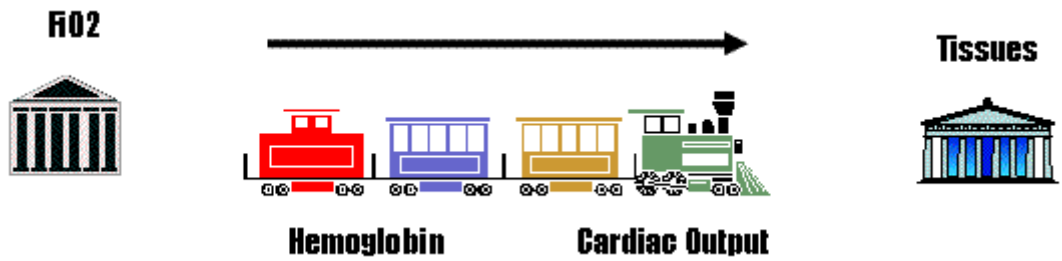
1. Oxygen binding
 - a. pO₂
 - b. Oxyhemoglobin
 - c. Normalize pH and body temp. to maximize O₂ saturation

2. Hemoglobin
 - a. Maximize available Hb
 - i. Minimize loss
 - ii. Maximize production
 - iii. Replacement issues
 - b. Oxygen-carrying chemicals



3. Cardiovascular system (hemodynamics)
 - a. Preload
 - b. Cardiac output
 - c. Afterload
4. Capillary Perfusion (delivery to tissues)
 - a. Global flow (MAP)
 - i. Maintain acceptable MAP that maintains tissue perfusion
 - ii. Pressors will decrease tissue perfusion and ↑ O₂ consumption
 - b. Shunting during low perfusion
 - i. Blood shunts past capillary network
 - ii. Sluggish flow contributes to microvascular clotting

5. Oxygen consumption
 - a. Factors that ↑ consumption
 - i. Activity
 - ii. Body temperature
 - iii. Vasopressors
 - iv. Sepsis
 - b. Interventions to ↓ consumption
 - i. Manage activity and body temp.
 - ii. Balance V:P train to maximize delivery



Oxygen delivery and oxygen consumption

Use the Ventilation-Perfusion Train to balance oxygen delivery and oxygen consumption to avoid complications from traditional resuscitation interventions.

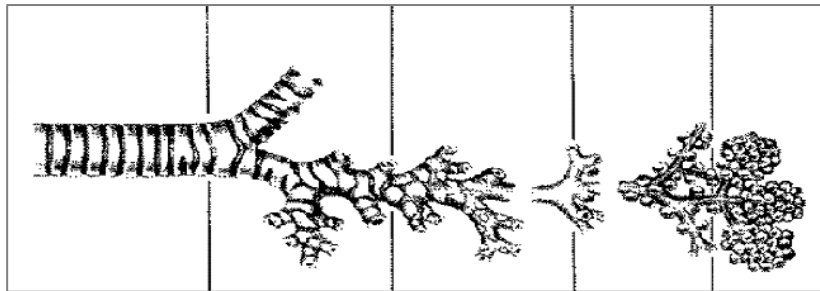
Component	Factors affecting	Improvement
Lungs	Fluids Secretions Atelectasis	Pulmonary hygiene Balance fluids
Hemoglobin	Loss Production	Decrease loss 'Tank up' Prevent dilution Replace
Cardiac Output	Preload, contractility, afterload	Consider pre-existing disease Improve hemodynamics: -Preload -Contractility -Afterload
Consumption	Fever Activity	Decrease fever Decrease activity Sedation

Pulmonary Assessment

1. Inspection
 - a. Thoracic size & shape
 - b. Work of breathing
2. Palpation
 - a. Excursion
 - i. Symmetry
 - ii. Volume
 - b. Sensation
3. Percussion
 - a. Resonance
 - b. Diaphragm excursion
4. Auscultation
 - a. Normal sounds
 - b. Adventitious sounds
 - i. Wheezing: musical, whistling sound
 1. Expiration > inspiration
 2. From narrowed airways
 - a. Bronchoconstriction
 - b. Secretions

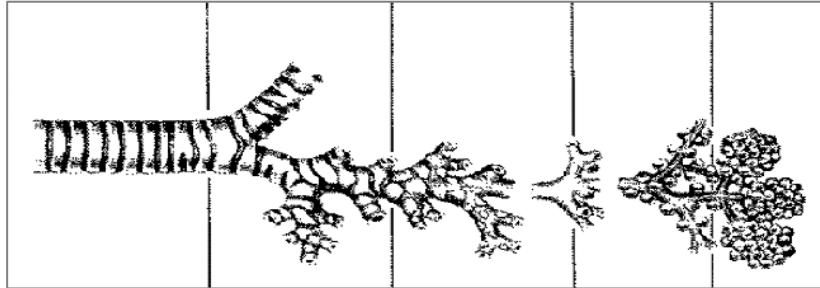
Pulmonary Hygiene:

- Deep breathing
- Incentive spirometry
- Coughing
- Forced expiration
- Turning and positioning
- Ambulation
- Hydration
- Bronchodilators



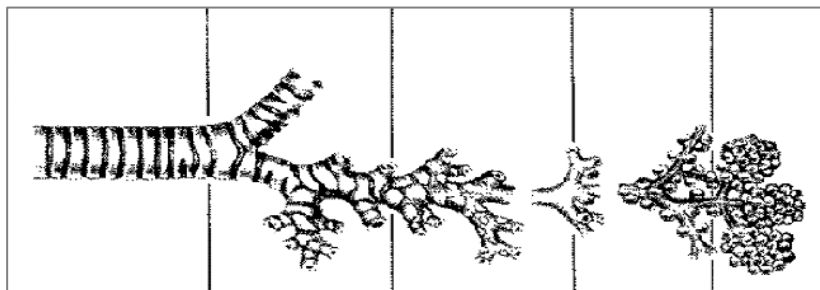
3. Interventions:
 - a. Bronchodilation
 - b. Hydration
 - c. Coughing

- ii. Rales: crackling sound
 1. End inspiration
 2. From collapsed or waterlogged alveoli
 3. Fine: beginning of fluid buildup
 4. Coarse: greater volume of fluid buildup



- 5. Interventions:
 - a. Manage fluids
 - i. Budget volume resuscitation
 - ii. Diuretics
 - b. Expectorate
 - i. Turn & position
 - ii. Deep breathing
 - iii. Forced expiration
 - iv. Vibration & percussion

- iii. Rhonchi: bubbling
 1. Inspiration > expiration
 2. Results from air bubbling past secretions in the airways



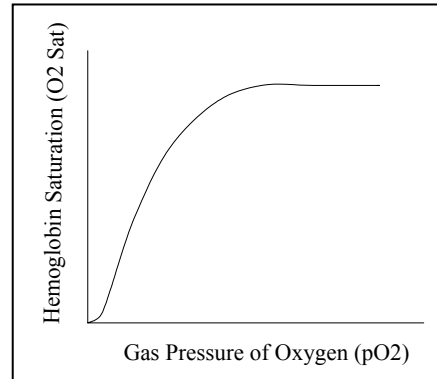
- a. Deep breathing
- b. Coughing
- c. Hydration
- d. Mobilize

- iv. Friction rub: creaking, leathery sound
 1. End of inspiration and beginning of expiration
 2. From rubbing of inflamed pleural surfaces

Monitoring

1. Respiratory rate & rhythm
 - a. Work of breathing
 - b. Neurochemical control

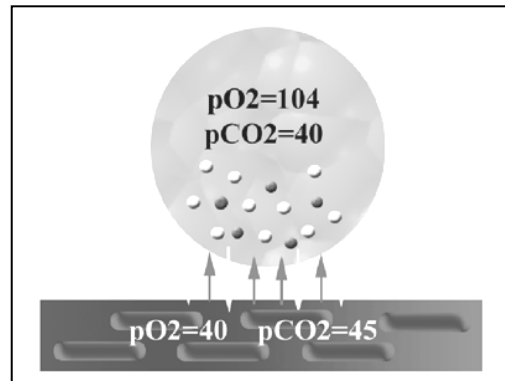
2. Pulse oximetry: what does it really tell us?
 - a. Therapeutic effectiveness
 - b. Changes in condition
 - c. Factors that ↓ accuracy
 - i. ↓ perfusion
 - ii. Placement
 - iii. Other compounds bound to hemoglobin
 - iv. Artifact



3. Arterial blood gases
 - a. Acid / base balance
 - b. Ventilation
 - c. Oxygenation

4. pO₂:FiO₂
 - a. Divide the pO₂ by the FiO₂
 - b. Assesses efficiency of the A/C membrane
 - c. May be ↓ in situations with O₂ sat. and pO₂ within normal range

5. Laboratory data
 - a. H&H
 - b. Electrolytes
 - c. Albumin
 - d. Lactic acid



FiO₂ conversions

1L NC = 24%
2L NC = 28%
3L NC = 32%
4L NC = 36%

Multisystem Assessment

Interactions between failing organs

When one major system begins to fail, there are consequences for all other systems. A patient admitted with a myocardial infarction (MI) will have decreased cardiac output and perfusion to other body systems will also decrease. Though the focus is on the heart, renal function, pulmonary function, and even brain function can be affected. Our job is to detect these changes before they become complications and the patient starts decompensating.

There is a commonly held belief that your patient needs a mean-arterial blood pressure (MAP) of 60 mmHg in order to adequately perfuse the brain and other organs. Although this may be true for some individuals, many patients will perfuse adequately with a MAP of 50 mmHg, while others will require a MAP of 70 mmHg or greater.

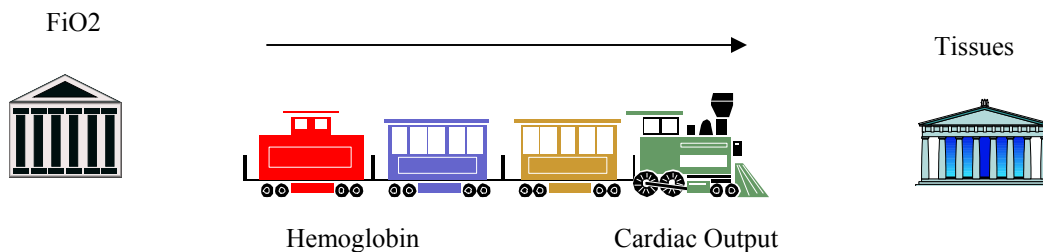
Identification of adequate perfusion should be individualized. If your patient is young and healthy, low perfusion pressures may be fine, but your elderly patient with multiple medical problems will need a higher perfusion pressure.

You can assess your patient's perfusion through evaluation of her blood pressure, testing her capillary refill, and assessing her brain and kidney function.

1. Perfusion
 - a. Mean Arterial Pressure (MAP)
 - i. $(\text{diastolic} \times 2 + \text{systolic}) / 3$
 - ii. MAP should be 60-90 mmHg
 - b. Capillary refill
 - i. Peripheral
 - ii. <3 seconds
 - c. Organ function (look at together)
 - i. Brain (requires a CPP of 70 mmHg)
 1. Level of consciousness
 2. Behavior
 - ii. Kidneys (requires an internal MAP of 80 mmHg)
 1. Urine output

Your patient also needs adequate oxygenation. The goal is to increase oxygen delivery, while decreasing oxygen consumption so that a balance is achieved. Simply applying oxygen is not enough and can be harmful!

1. Oxygen delivery
 - a. Pulmonary
 - b. Hemoglobin
 - c. Cardiac output
2. Oxygen consumption
 - a. Decrease fever
 - b. Decrease activity

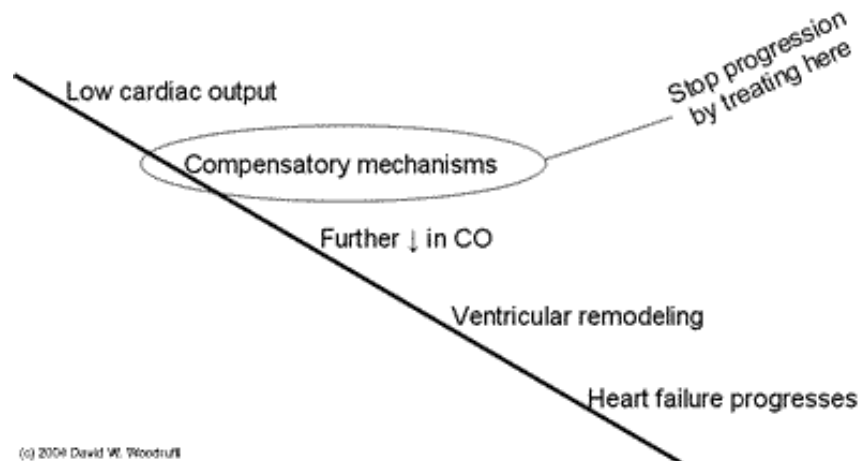


Applying oxygen to increase oxygen saturation, and giving fluids and vasopressors to increase blood pressure can be harmful. Balancing the ventilation-perfusion train is the key to maintaining oxygenation.

Multisystem effects of cardiac dysfunction

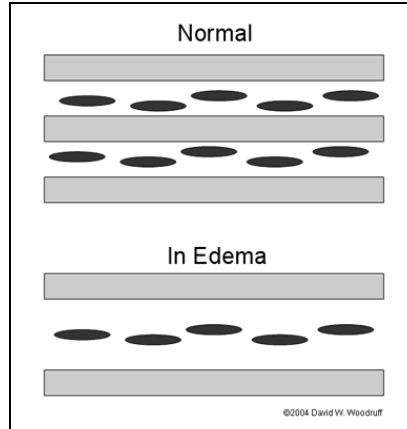
Cardiac dysfunction is the result of a progressive disease state where low cardiac output causes stimulation of the compensatory mechanisms of the sympathetic nervous system, renin-angiotensin system, and aldosterone system to be released. These compensatory mechanisms cause the progressive dysfunction of heart failure.

Downhill slide in heart failure



1. Decreased perfusion
 - a. Global oxygen debt
 - i. ↓ cardiac output
 - ii. ↓ oxygen delivery to the tissues
 - iii. Less efficient at oxygen extraction
 - b. Decreased cardiac reserves
 - i. ↓ oxygen delivery to the heart
 - ii. ↓ heart's response during stress
2. Decreased oxygenation
 - a. Pulmonary edema
 - i. Atelectasis
 - ii. Pneumonia
 - b. Decreased oxygen delivery
 - i. ↓ pulmonary perfusion
 - ii. ↓ cardiac output
3. Poor oxygen utilization
 - a. Due to chronic hypoxia, cells become less efficient at extraction
4. Volume overload
 - a. Interstitial edema
 - b. Widens perfusion area

Oxygenation with edema present



Assessment

1. Respiratory
 - a. Assess for pulmonary edema
 - b. Assess for atelectasis
 - c. Assess the bases in the back first!
2. Cardiac
 - a. Preload
 - i. Intake and output
 - ii. Weight
 - b. Contractility
 - i. Systolic blood pressure
 - ii. Organ perfusion
 - iii. Peripheral perfusion
 - c. Afterload
 - i. Skin color and temperature
 - ii. Diastolic blood pressure

Avoid complications

1. Good pulmonary hygiene
2. Manage fluid volume
3. Assess cardiac output
4. Do not cluster nursing activities

Note: It will take the CHF patient much longer to recover from cellular hypoxia than other patients.

Multisystem effects of respiratory dysfunction

The respiratory system must feed the rest of the body with oxygen. When the patient has respiratory failure, then global hypoxia results. Respiratory distress increases oxygen consumption of the respiratory system from 3% at rest to 60% during distress. As more oxygen is required by the respiratory system, less is available for the tissues resulting in a large delivery and consumption imbalance. It may take the patient as long as 24 hours to recover from this imbalance, therefore respiratory distress is to be avoided.

1. Increased oxygen consumption of the lungs
 - a. At rest = 3% of total
 - b. During distress = 60% of total
2. Poor ventilatory function
 - a. Air movement is decreased
 - b. Less turnover of oxygen at alveolar level
 - c. Less efficient, more energy needed
3. Poor perfusion
 - a. Hypoxia
 - b. Vasoconstriction
 - c. Further hypoxia
4. Decreased oxygen delivery
 - a. ↓ oxygenation of the heart
 - b. ↓ contractility (high-output failure)
 - c. ↓ delivery to tissues

Assessment

1. Lungs
 - a. Arterial blood gas (ABG)
 - b. $pO_2:FiO_2$
2. Heart
 - a. Systolic blood pressure
 - b. Look for tachycardia as a sign of SNS stimulation
3. Organs
 - a. Brain, kidneys
4. Peripheral
 - a. Pulse oximetry

Avoid complications

1. Good pulmonary hygiene
2. Avoid hypoxia and respiratory distress
3. Assess ABG, monitor SpO_2

Note: Pulse oximetry is a monitoring tool, not a diagnostic tool! An ABG must be done in all patients with respiratory distress. Don't rely on the pulse-ox reading!

Multisystem effects of renal failure

Renal failure causes the build-up of waste products, fluid & electrolyte imbalances, and acidosis. These changes will affect the function of all the organ systems of the body.

1. Decreased filtration
 - a. ↑ BUN
 - b. Acidosis
2. Volume overload
 - a. Fluid retention
 - b. Fluid shifts
3. Metabolic acidosis
 - a. Loss of renal acid/base control
 - b. Chronic acidosis, with respiratory compensation
4. Electrolyte abnormalities
 - a. ↑ K⁺
 - b. ↓ Na⁺

Comparison of Acute and Chronic Renal Failure

	Acute	Chronic
Onset	Hours Secondary condition	Months Primary condition
Symptoms	Severe	Mild
Mortality	40%	<1%

Assessment

1. Fluid volume
 - a. I & O
 - b. Weight
2. Electrolytes
3. BUN / Cr
4. Creatinine clearance

Estimated CCr (mL/min) =

$(140 - \text{age})(\text{weight in kg})$

72 X serum Cr in mg/dL

(multiply result by 0.85 in women)

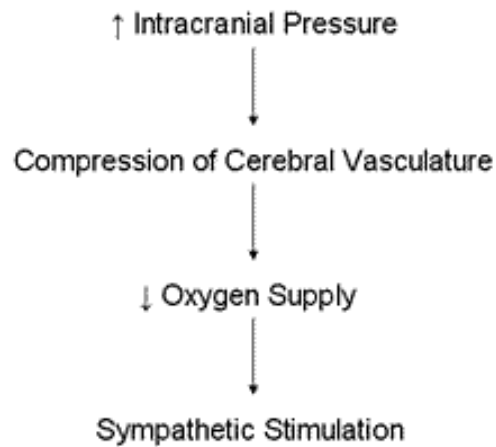
Avoid complications

1. Watch lungs for volume overload
2. Assess electrolytes

Note: It is more important how fast fluid and electrolyte changes occur, rather than how bad they are. Patients with acute renal failure will not tolerate fluid and electrolyte changes as well as patients with CRF.

Multisystem effects of neurological dysfunction

When your patient has a stroke or other neurological disease, inflammation and swelling cause intracranial pressure to rise. Because there is no extra room in the skull to accommodate the swelling, then compression of the vasculature results and hypoxia ensues.



1. Competing excitation of the autonomic nervous system
 - a. Sympathetic
 - i. Goal is to ↑ cerebral perfusion
 - ii. Causes:
 1. Cardiac arrhythmias
 2. GI bleeding
 - b. Parasympathetic
 - i. Responding to ↑ systolic blood pressure
 - ii. Causes:
 1. Bradycardia
 2. Respiratory depression
2. Compression and ischemia of vital function centers

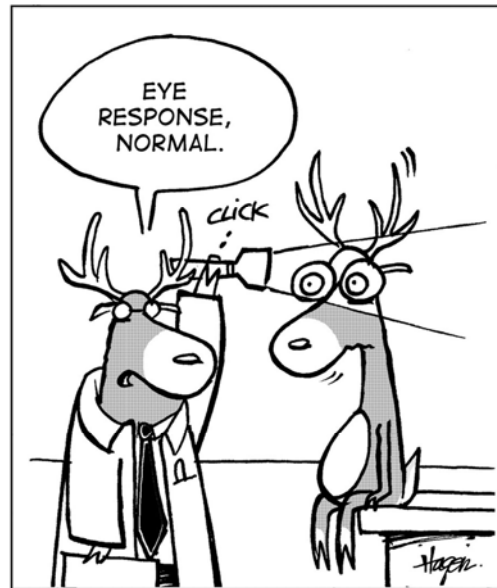
Assessment

1. Quick 5-point neuro check
 - a. Behavior
 - i. Affect
 - ii. Irritability
 - iii. Restlessness
 - b. Speech
 - i. Appropriateness
 - ii. Slurring
 - c. Content of arousal
 - i. Person
 - ii. Place
 - iii. Time
 - d. Arousability
 - i. Spontaneously
 - ii. To voice
 - iii. To tactile stimuli
 - e. Systolic blood pressure
 - i. \uparrow ICP stimulates the SNS
 - ii. Systolic B/P increases

Some factors that \uparrow ICP:

- Fluid volume
- Mechanical ventilation
- CPAP, BiPAP
- Coughing
- Abdominal pressure
- Sodium level
- Albumin level
- Glucose level
- Oxygenation
- Carbon dioxide level
- Positioning
- Nursing care

2. Cushing's triad
 - a. Increased SBP
 - b. Wide pulse pressure
 - c. Bradycardia



DEER PHYSICALS

Avoid complications

1. Decrease stimulation to decrease oxygen consumption
2. Increase oxygen delivery
3. Maintain perfusion pressure