Hemodynamic Management

This pocket reference includes information for nurses on:

- Hemodynamic response
- Factors affecting:
  - HR
  - Preload
  - Afterload
  - Contractility
- Oxygenation
- Functional Hemodynamic Measurements

NOTE: This pocket card is for quick reference only. Please review and follow your institutional policies and procedures before clinical use.

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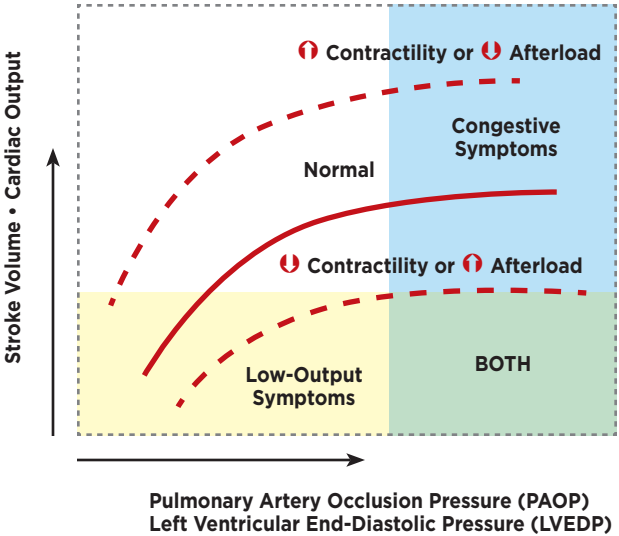
Hemodynamic Response

Normal Values:	Cardiac Output	4-8 L/min
	Cardiac Index	2.5-4 L/min/m^2
Calculation:	Cardiac Index	CO/BSA



Hemodynamic Response

Stroke Volume (SV): 50-100 mL/beat
Stroke Volume Index (SVI): 30-60 mL/beat/m^2
Ejection Fraction (EF): > 60%

Heart Rate

Normal Values:

• Adult/Adolescent	60-100 beats/min	• Toddler (1-2 y)	98-140 beats/min
• School Age (6-12 y)	70-118 beats/min	• Infant (1-12 mo)	100-180 beats/min
• Preschool (3-5 y)	80-120 beats/min	• Neonate (0-1 mo)	100-205 beats/min

Increased

Mechanism/Effect

- Sympathetic nervous system stimulation in response to stressful stimuli
- Compensatory mechanism for decreased CO and/or decreased SV
- Cardiac rhythm disorder from increased excitability of cardiac pacemaker cells

Causes

- Sympathetic stimulation (fear, pain, anxiety, stress)
- Exercise (physical activity)
- Decreased blood volume (preload)
- Increased vascular tone (afterload)
- Hypermetabolic states (fever, hyperthyroidism)
- Hypoxia and hypercarbia
- Conditions causing myocardial excitability, increased conduction (medications, cardiac cellular damage/edema)
- Medications and stimulants (caffeine, nicotine, cocaine)
- Accessory cardiac conduction pathways (re-entry phenomenon)

Treatment

- Correct sympathetic stimulus (control pain/anxiety, decrease physiologic stressors)
- Optimize preload, afterload, and contractility
- Optimize oxygenation/ventilation
- Decrease metabolic/oxygen demands (sedation, paralysis, fever management)
- Medications that slow conduction, decrease irritability (digoxin, calcium-channel blockers, beta-blockers, amiodarone, lidocaine, procainamide, adenosine, magnesium sulfate)

Decreased

Mechanism/Effect

- Parasympathetic nervous system stimulation (vagal stimulation)
- Compensatory mechanism for increased blood pressure
- Cardiac rhythm disorder from decreased excitability of cardiac pacemaker cells

Causes

- Cardiac conduction defect
- Vagal stimulation (intubation, suctioning, nasogastric tube placement, vomiting, Valsalva maneuver, diver's reflex, carotid pressure)
- Relaxed state (sleep, sedation)
- Severe hypoxia
- Conditions that cause decreased myocardial excitability
- Neurogenic (Cushing's triad, increased intracranial pressure, anoxic injury)
- Myocardial tissue damage
- Normal variation (physical fitness)
- Medications that cause bradycardia (tricyclic antidepressant overdose, digoxin)

Treatment

- Remove vagal stimulus
- Administer positive chronotropic medications (epinephrine, dopamine, atropine)
- Pacemaker (transcutaneous, transvenous, permanent)
- Optimize oxygenation and ventilation

Preload

Normal Values:

- CVP 2-8 mm Hg
- PAOP 6-12 mm Hg

Increased

Mechanism/Effect

- Increased myocardial muscle fiber stretch leads to increased ventricular blood volume, increased SV, increased CO, increased ventricular work
- Overstretched myocardial muscle fibers lead to decreased force of contraction, decreased SV, heart failure (Frank-Starling phenomenon)

Causes

- Decreased fluid excretion (kidney failure)
- Excess fluid administration (fluid overload)
- Ineffective cardiac pumping (right and left heart failure)
- Aortic insufficiency
- Vasoconstriction (alpha stimulation)
- Inotropic medication
- Pregnancy

Treatment

- Correct condition causing fluid volume retention/overload)
- Vasodilatation (see increased afterload)
- **Diuretics**
  - Furosemide
  - Bumetanide
  - Mannitol
  - Spironolactone
  - Chlorothiazide
  - Hydrochlorothiazide
  - Metolazone
- Hemodialysis
- Continuous renal replacement therapy for fluid overload with kidney failure

Decreased

Mechanism/Effect

- Decreased myocardial muscle fiber stretch from decreased circulating blood volume, decreased SV, decreased CO, vascular volume depletion, hypotension, cardiovascular collapse
- Increased volume corresponds with increased CO to a point of optimal stretch of myocardial muscle fibers

Causes

- Vascular volume loss (hemorrhage, diarrhea, vomiting, burns, edema, decreased intake, diuretics, third spacing)
- Vasodilatation (medications, septic shock)
- Pathological conditions (diastolic ventricular failure, mitral or tricuspid stenosis)
- Loss of atrial kick

Treatment

- Correct condition causing fluid volume loss
- Volume expansion
  - **Crystalloid**
    - Saline
    - Lactated Ringer's
  - **Colloid**
    - Albumin
    - Plasma
    - RBCs
- **Vasoconstrictors** (norepinephrine, dopamine, phenylephrine, epinephrine) may be used to correct severe hypotension when given in conjunction with fluid resuscitation

Afterload

Normal Values:
- SVR 800-1,200 dynes sec/cm^5
- PVR 150-250 dynes sec/cm^5

Calculations:
- SVR = [(MAP– CVP) / CO] x 80
- PVR = [(MPAP – PAOP) / CO] x 80

Increased

Mechanism/Effect
- Increased resistance to forward flow is related to increased atrial resistance, increased PVR and SVR, increased end-diastolic pressure, increased aortic impedance, increased blood viscosity, and increased aortic blood volume
- Increased resistance to forward flow leads to increased left ventricular stroke work, which leads to increased myocardial oxygen demands, which lead to increased left ventricular failure

Causes
- Hypertension
- Sympathetic stimulation
- Peripheral vasoconstriction
- Vasopressors
- Aortic stenosis, pulmonic stenosis
- Polycythemia
- Medications/substances causing hypertensive effects (cocaine, amphetamines)
- Hypothermia
- High positive end-expiratory pressure
- Increased ventricular-wall tension (dilation)

Treatment
- Reduce sympathetic stimulation
- Normalize body temperature
- **Vasodilators**
  - Nitroglycerin
  - Nitroprusside
  - Labetalol
  - Amrinone
  - Milrinone
  - Hydralazine
  - Enalapril, captopril, lisinopril
  - Isosorbide
- **IABP increases pumping ratio** (1:1)
- **Diuretics** (see increased preload)

Decreased

Mechanism/Effect
- Decreased resistance to forward flow is related to decreased atrial resistance, decreased PVR and SVR, decreased end-diastolic pressure, decreased aortic impedance, decreased blood viscosity, and decreased aortic blood volume

Causes
- Vasogenic shock
- Septic shock (early), endotoxin release
- Anaphylactic shock
- Neurogenic shock
- Vasodilators
- Medications causing hypotension (narcotics, antidepressants)

Treatment
- **Vasopressors**
  - Epinephrine
  - Dopamine
  - Norepinephrine
  - Phenylephrine
  - Metaraminol
- **IABP decreases pumping ratio** 1:2 or 1:4
- **Volume expansion** (see decreased preload)
  - Crystalloids
  - Colloids

Contractility

Normal Values:
- SVI = 30-60 mL/beat/m^2
- RVSWI = 7-12 g/m^2/beat
- LVSWI = 35-85 g/m^2/beat

Calculations:
- SVI = CI/HR x 1000
- RVSWI = (MPAP – CVP) x SVI x 1000 x 0.0136
- LVSWI = (MAP – PAOP) x SVI x 1000 x 0.0136

Increased

Mechanism/Effect
- Increased contractile state of myocardium leads to increased force of contraction, which leads to increased SV, which leads to increased CO and myocardial oxygen demand

Causes
- Positive inotropic medications
- Increased HR (Bowditch's law)
- Sympathetic stimulation (beta$_1$ receptor)
- Electrolyte imbalance (hypercalcemia)
- Toxic ingestion (digoxin)
- Systemic inflammation

Treatment

When positive inotropic effects are undesirable because of increased myocardial oxygen demand
- **Correct positive inotropic effects** (correct calcium level, decrease sympathetic stimuli, decrease HR)
- **Negative inotropic medications**
  - Beta-blockers
  - Calcium-channel blockers
  - Barbiturates
- **Correct toxic effect**

Decreased

Mechanism/Effect
- Decreased contractile state of myocardium leads to decreased force of contraction, which leads to decreased SV, which leads to decreased CO

Causes
- Negative inotropic medications
- Parasympathetic stimulation (vagus nerve)
- Hypoxia
- Hypercapnia
- Metabolic acidosis
- Electrolyte imbalance
- Cardiotoxic medications/substances (chemotherapy, salicylate overdose)
- Toxin release (septic shock)

Treatment
- **Correct negative inotropic effects** (oxygen, ventilation, acid/base balance, electrolyte balance, remove vagal stimulus)
- **Positive inotropic medications**
  - Dobutamine
  - Dopamine
  - Amrinone
  - Milrinone
  - Digoxin
  - Epinephrine
  - Calcium infusion
- **Optimize preload**
  - Correct volume deficit
  - Correct volume excess

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Oxygenation

Normal Ranges:

• SvO_2	60%-75%
• $ScvO_2$	70%-85%

SvO_2 and $ScvO_2$ are measurements of the relationship between oxygen consumption and oxygen delivery in the body. They reflect the oxygen saturation returning from the body through the venous system. The 3 influencers of SvO_2 and $ScvO_2$ measurements are:

- Hemoglobin
- CO
- Metabolic demand/oxygen consumption

Conditions that decrease SvO_2 (increase oxygen demand)	**Medications that increase or decrease SvO_2*** (increase oxygen demand)	**Procedures that decrease SvO_2** (increase oxygen demand)	**Factors that increase SvO_2** (decrease oxygen consumption)
• Minor surgery • Fever • Bone fracture • Agitation • Increased work of breathing • Chest trauma • Multiple organ failure • Shivering • Burns • Sepsis • Head injury	• Norepinephrine • Dopamine • Dobutamine • Epinephrine *Although these medications all increase oxygen demand, SvO_2 may increase as CO improves.	• Dressing change • Nursing assessment • 12-lead ECG • Visitors • Bath • Chest x-ray • Endotracheal suctioning • Turning • Getting out of bed • Nasal intubation	• Hypothermia • Sedation/ analgesia • Anesthesia • Assist/control ventilation • Propranolol in head injury • Neuromuscular blockade

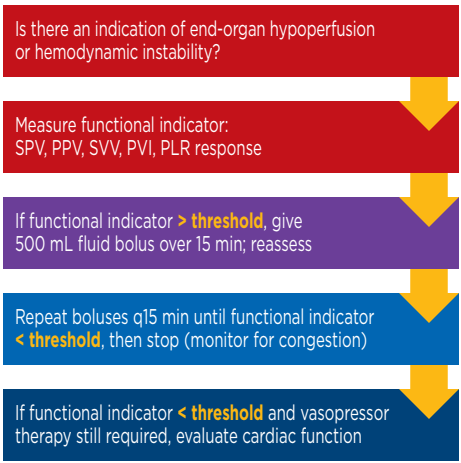
Hemodynamic Alterations in Shock			
Parameter	**Hypovolemic**	**Cardiogenic**	**Distributive**
BP	↓	↓	↓
CI	↓	↓	↑↔
CVP	↓	↑↔	↓
PAOP	↓↓	↑↑	↓
SVR	↑	↑	↓↓
SvO_2	↓	↓	↓

Functional Hemodynamic Measurements

For patients receiving ventilatory support with a Vt > 6 mL/kg, using arterial pressure data from a bedside monitor during respiratory cycle, systolic pressure variation (SPV), pulse pressure variation (PPV), and stroke volume variation (SVV) can be calculated using the following equations:

Formulas		
Variable	**Equation**	**Threshold for Responders**
SPV	SBPmax - SBPmin	> 10 mm Hg
SPV %	[(SBPmax - SBPmin)/(SBPmax + SBPmin/2)] x 100	> 10%
PPV %	[(PPmax - PPmin)/(PPmax + PPmin/2)] x 100	> 12.5%
SVV %	[(SVmax - SVmin)/(SVmax + SVmin/2)] x 100	≥ 12%
Pleth Variability Index (PVI)	Derived from oximeter perfusion index	12%-16%

Simplified treatment algorithm based on functional hemodynamic parameters



Legend: **BP**, blood pressure; **BSA**, body surface area; **CI**, cardiac index; **CO**, cardiac output; **CR**, classic reference; **CVP**, central venous pressure; **HR**, heart rate; **IABP**, intra-aortic balloon pump; **LVSWI**, left ventricular stroke work index; **MAP**, mean arterial pressure; **MPAP**, mean pulmonary artery pressure; **PAOP**, pulmonary artery occlusion pressure; **PLR**, passive leg raising; **PVR**, pulmonary vascular resistance; **RVSWI**, right ventricular stroke work index; **SBP**, systolic blood pressure; **SV**, stroke volume; **$ScvO_2$**, central venous oxygen saturation; **SVI**, stroke volume index; **SvO_2**, mixed venous oxygen saturation **SVR**, systemic vascular resistance; **Vt**, tidal volume