TREATMENT OF THE CRITICALLY ILL PATIENT

Patients admitted to the ICU typically have multisystem disease, traumatic injuries, or are under intensive treatment regimens to avoid or manage end-organ dysfunctions. Despite vast advancements in knowledge, the interactions between dysfunctional organ systems remain complicated and are often overwhelming to the junior house officer. Physicians must systematically focus on each organ system and evaluate its real-time functional status as well as its interactions within the host. A thorough approach allows the physician to integrate organ system therapies into a treatment strategy for the patient as a whole. This chapter describes a system-by-system approach to evaluating, analyzing, integrating, and treating critically ill patients as well as some commonly encountered critical care complications.

One of the most important aspects of caring for the critically ill patient is performing a thorough physical exam on a daily basis. A methodical, detail-oriented examination of the ICU patient often identifies problems that, if acted on early, will markedly alter the patients’ recovery. In other words, being PROACTIVE, not REACTIVE, may prove to be the difference between the critically ill patient surviving their illness or not.

ICU PROGRESS NOTE

The ICU progress note is a concise, systematic, well-organized means of documenting the patient’s major problems, the events over the past 24 hours, the physical exam, pertinent laboratory data, and the treatment plan (see Sample ICU Progress Note). Although the
information may be found elsewhere in the chart, the physician’s interpretation of data and events, assessment and treatment plan communicates the medical decision-making process those who read the chart. A simple, organized approach to the daily progress note includes:

A. Outline of the patient’s problem list and / or injury summary
   -- Active problems, major inactive problems and significant allergies
   -- Significant past medical or surgical history relevant to the present illness
   -- List hospital day, post-trauma day, post-operative day, etc.

B. Outline of events and procedures over the past 24 hours

C. List of current medications including analgesic and sedation regimens

D. Sequential, system-specific physical exam and pertinent flow sheet data
   -- **CNS:** Central Nervous System functioning or other neurologic assessment and sedation level (Modified Ramsay Sedation scale, Richmond Agitation Scale, etc)
   -- **CV:** Cardiovascular function, including indicators of systemic perfusion, measured blood pressures, and calculated pulmonary artery catheter data
   -- **Pulm:** Pulmonary function, including mechanical ventilator settings, and arterial blood gas values
   -- **GI / Nut:** Gastrointestinal function and nutritional status
   -- **F / E / R:** Fluids, electrolytes and renal function
   -- **Heme / ID:** Hematologic function, including complete blood count, coagulation values; infectious disease status (recent culture data, antibiotic regimen, treatment duration)
   -- **Prophylaxis:** Therapies to prevent complications, such as deep venous thrombosis, ethanol withdrawal, stress gastritis, etc.
E. Other relevant laboratory or radiographic data

F. Assessment & Plan for the next 24 hours

Sample ICU Progress Note (written for a trauma patient)

PROBLEM LIST:

• s/p MVC (PTD # 3) • L pulmonary contusion • L hemopneumothorax s/p L chest tube •

Grade 4 splenic injury s/p splenectomy (POD #2) • Acute renal failure • ARDS • PMHx:

Hypertension / Gout • Allergies: Morphine

EVENTS OF PAST 24 HOURS:

• Increasing FiO₂ and PEEP • Renal Consult • L subclavian vein cordis / Swan-Ganz catheter placement • nasojejunal feeding tube placed

MEDICATIONS:

Dopamine gtt @ 5 • Fentanyl gtt • Ativan gtt • vaccines given (POD # 1)

EXAM AND FLOW SHEET DATA:

CNS: Intubated, sedated (RAS -2), moves all 4 extremities • Atraumatic head • pupils equal and reactive • EOM intact • Neck immobilized

CV: RRR w/o murmurs • no JVD • 2+ capillary refill • toes warm • minimal peripheral edema

• P 150 (sinus tach), BP 110/65, PAP 45/20, PCWP 14, CO 3.7, CI 2.5, EDVI 89

Pulm: Coarse BS bilaterally • L chest tube in place • Ventilator Setting: SIMV rate 16/4 -- FIO₂ 75% -- PEEP 12 -- PS 10 • ABG: 7.38 / 42 / 78 / 19 / -3

GI/Nut: Midline incision healing well • soft, non-tender, non-distended • ± bowel sounds •
nasojejunal feeding tube in stomach • tube feedings started (20 cc/hr)

\[F/E/R:\] LR @ 125 cc/hr • Electrolytes wnl • BUN 48 • Cr 1.9 • total I/O: 3400 / 2210 • UO 45 cc/hr

\[Heme/ID:\] WBC 17.5 • Hgb 9.4 • Hct 30 • coags wnl • blood cultures (-) x 2 • Vancomycin (D # 2 / 7)

\[Prophylaxis:\] SCD’s on LE • Heparin 5,000 U SQ Q12h • famotidine 20 mg iv Q12h

**ASSESSMENT AND PLAN:**

- **CNS:** Stable neuro exam; sedation level adequate; continue fentanyl and ativan gtt while on ventilator; wean narcotics as ventilator-dependence improves

- **CV:** Hemodynamically stable however, continues to require intermittent fluid challenge to maintain BP; this may be the cause of acute renal failure; will continue iv fluids and wait for capillary leak to cease; will wean dopamine gtt as tolerated

- **Pulm:** Worsening FiO\(_2\) and PEEP requirements overnight; likely ARDS complicated by pulmonary contusion; will obtain CXR this am and wean FiO\(_2\) / increase PEEP as tolerated by BP and CO; continue L chest tube to suction; will d/c when ventilatory status improves

- **GI/Nut:** s/p splenectomy; ileus continues; will continue trophic tube feedings

- **F/E/R:** Acute renal failure continues; will proceed with renal ultrasound to r/o postrenal cause; no significant renal toxic drugs currently given

- **Heme/ID:** s/p splenectomy; Hct stable; post-splenectomy vaccines given; will expect mild leukocytosis and thrombocytosis

- **Prophylaxis:** continue as prescribed
ROUTINE MONITORING

Critically ill patients require **continuous monitoring of basic physiologic parameters** to identify perturbations. Such **perturbations should then prompt a timely clinical adjustment** in order to **improve the patient’s outcome**:

1. **Continuous ECG**: allows use of computerized arrhythmia detection systems; rapid, continuous access enables prompt assessment of cardiac rhythm to ensure proper treatment; increases the likelihood of successful resuscitation.

2. **Blood pressure**: intermittent (sphygmomanometer) or continuous (intra-vascular) assessment of blood pressures (i.e., systolic / diastolic / mean arterial / central venous pressures, etc.); allows tracking of treatments and minute-to-minute titration of vasoactive drugs; continuous, intravascular methods are warranted in patients with significant hemodynamic instability.

3. **Pulse Oximetry**: continuous, quantitative arterial O₂ saturation (SaO₂); ensures adequate oxygenation of systemic arterial blood for tissue delivery and utilization.

4. **Temperature**: critically ill patients are at high-risk for thermoregulatory disorders due to their pathophysiology (i.e., fluid resuscitation, burns, sepsis, etc.); continuous measurements in the esophagus (esophageal probe) or central venous blood compartment (pulmonary artery catheter) are accurate methods to monitor core body temperature; changes in temperature should prompt investigation.

5. **Capnography**: allows continuous measurement of expired CO₂; changes warrant further investigation as they imply a change in clinical status (i.e., hypoventilation, overfeeding, fever, sepsis, etc.).
TRANSPORTING CRITICALLY ILL PATIENTS

Murphy’s Law dictates “Whatever may go wrong, will go wrong”; this is especially true during the transport of critically ill patients. Adherence to “common sense” guidelines will help to minimize potential adverse events:

1. **ALWAYS REMEMBER: MAINTAIN THE PATIENTS AIRWAY**; if airway concerns exist, intubate the patient prior to transport. **PERIOD.**

2. Only transport patients that are stable (unless the role for transport is to provide a life-saving intervention).

3. Pay attention to intravenous catheters / pumps and their attachments to each other; loss of intravenous access in a critically ill patient is dangerous.

4. Ensure the patient has sufficient transportable O₂, intravenous fluids, medications, etc.

5. Be certain enough assistance is available to safely transport the patient and the many machines; make sure the destination is prepared for the patient’s arrival and an expeditious interaction.

6. *Expect the unexpected*; have personnel, equipment, and supplies available that could make the difference in a crisis situation.

CENTRAL NERVOUS SYSTEM

Severe acute illness often results in altered mental status (AMS). **AMS may manifest a spectrum of disability from simple, mild delirium to complex, life-
threatening coma. The precise CNS pathophysiology of AMS states remains unknown; however, false neurotransmitters, excess catecholamines, alterations in ion fluxes, and deranged cerebral blood flow all appear to play a role.

Critically ill patients are often times intubated. In order to provide a humane, safe environment for the intubated patient, medications must be administered to provide sedation and analgesia. Inadequate sedation and pain control have well known adverse squealae (i.e., increased catabolism, tachycardia and higher myocardial oxygen consumption, immunosuppression, hypercoagulability, severe anxiety, etc.), so great care must be taken in finding the proper balance of medications.

When acute agitation occurs, life-threatening pathology (i.e., inadequate blood flow or nutrient availability to the brain) should be ruled out first. Assessment of vital signs, blood glucose concentration, and oxygenation / ventilation status must be performed prior to administering CNS-altering medications.

Benzodiazepines are potent inducers of sedation, amnesia, muscle relaxation, and anxiolysis. These properties make this class of drug ideal for short- to intermediate-term use in this patient population. Great care should be taken to choose a drug that will not accumulate in the patients system if end-organ dysfunction is present:

- **Lorazepam:** Good intermediate-duration benzodiazepine; metabolized by the liver with inactive metabolite excreted in the urine; very potent but has a long time to peak-effect (i.e., ideal agent for longer-term sedation)

- **Midazolam:** Shorter-onset, shorter-acting benzodiazepine; metabolism altered by calcium-channel blockers, erythromycin, and triazole antifungals
Either agent should be used and **titrated to achieve a sedation level** according to published scales (i.e., Modified Ramsay Scale, Richmond Agitation Scale, etc.). If over medication occurs (i.e., inadvertent over administration, accumulation of metabolites), cessation of the medication, preparation to institute cardiopulmonary support, and the use of flumazenil will assist to reverse the overly sedated state.

- **Propofol**: Non-benzodiazepine, lipid-based sedative-hypnotic; little analgesic properties; extremely short onset and half-life make accumulation unlikely (i.e., ultra-short-term drug); expensive; longer-term use has adverse financial and infectious consequences

- **Haloperidol**: Short-term treatment of agitation especially with components of delirium; however, use is not without potential adverse electrocardiographic consequences (i.e., prolongation of QT interval); use should be discontinued if the QT interval increases by > 50% of baseline or exceeds 450 msec

Critically ill patients may also have acute pain requirements due to recent surgeries or pre-hospital trauma. Opioid narcotic agents are best suited for acute pain control.

- **Morphine**: Intravenous opioid narcotic; commonly used (low cost, ease of use)

- **Fentanyl**: Newer, synthetic opioid; more potent and shorter acting than morphine; less histamine release than morphine (i.e., less potential for drug-induced hypotension)

These opioids may be administered as continuous infusions, intermittent boluses, or as part of **patient-controlled analgesia** (PCA) regimen. Since narcotics may produce respiratory depression, careful titration is necessary, especially when narcotics are combined with benzodiazepines. **Epidural anesthesia** provides good local analgesic properties with less need for intravenous narcotics, which should yield less respiratory compromise.
Many critically ill patients will require **long-term sedation and analgesia**. Mid- to long-acting medications provide the best means of achieving this goal. Current data suggests, however, that **interruption of on-going sedation** to allow the patient to awaken on a daily basis **leads to decreased mechanical ventilation days and ICU length of stay**.

**Neuromuscular paralysis** is rarely necessary, but may be used in patients with severe respiratory failure and the inability to properly oxygenate or ventilate. Eliminating the muscular elastic recoil of the chest wall and ventilator dyssynchrony may improve pulmonary compliance and ventilation / oxygenation ability.

**CARDIOVASCULAR SYSTEM**

Cardiovascular instability remains one of the most common problems encountered in ICU patients. Understanding the approach to evaluating the cardiovascular system is essential. As before, a **thorough physical exam must begin your assessment** of the patient.

**Inspection: Jugular Venous Distention (JVD)**

- **Neck vein visualization** (with the patient sitting at a 45-degree angle) implies a central venous pressure (CVP) of > 12–15 mmHg

- **JVD PLUS systemic hypotension** suggests **life-threatening pathology**:
  1. Tension pneumothorax
  2. Pericardial tamponade
  3. Severe cardiac dysfunction
Inspection: *Precordial Contusion (i.e., Bruising)*

- Associated with blunt trauma from a steering wheel; injury pattern implies possible **myocardial contusion.** *Treatment:* continuous ECG monitoring; correction of arrhythmias (most common: *sinus tachycardia*). *Transthoracic echocardiography should be performed if arrhythmias occur* to identify anatomic heart injury and / or pericardial effusion

Inspection: *Extremity Perfusion*

- Check extremities for **perfusion** (i.e., pulse, color, temperature, and capillary refill)
- **Note:** Pay special attention to sites distal to:
  - long bone fractures
  - joint dislocations
  - indwelling arterial catheters

**Blood Pressure (BP)**

Over the short term, blood pressure is considered **adequate if renal perfusion is maintained** (usually mean arterial pressure (*MAP*) > 70 mmHg in young, previously healthy individuals). **Pre-morbid medical problems** and **aging**, however, may alter this somewhat.

**NOTE:** if the **cuff is too small** for the arm (i.e., the patient is obese), the measured systolic BP will be **10–15 mmHg higher** than the actual pressure.

**Systolic Hypertension:** SBP >140 mmHg with normal DBP. In the acute setting, due to:
- Increased cardiac output
- Thyrotoxicosis
- Generalized response to stress
- Anemia
Pain and/or Anxiety

**Diastolic Hypertension:** DPB > 90 mmHg. Isolated DBP hypertension associated with:

- Intrinsic renal disease
- Endocrine disorders
- Renovascular hypertension
- Neurologic disorders

*Treatment:* Hypertension is concerning following an acute myocardial infarction (AMI), subarachnoid hemorrhage, or vascular anastomosis (esp. carotid artery surgery).

Ideally, the **SBP is maintained between 130-160 mmHg** in critically ill patients. A **SBP >180 mmHg usually requires immediate treatment.** Several drugs are commonly used to treat acute hypertension in the ICU setting; nitroprusside, hydralazine, labetalol, esmolol, or nitroglycerin. Rapid, easily reversible β-blockade (i.e., esmolol) should be used with nitroprusside in treating hypertension associated with a ruptured aortic aneurysm or blunt traumatic aortic injury. The emergency management of hypertension is discussed in Chapter 21 and the specific antihypertensive agents are discussed more fully in Chapter 22.

**Mean Arterial Pressure (MAP):** calculated as \[
\text{MAP} = \text{DBP} + \left\{\frac{(\text{SBP} - \text{DBP})}{3}\right\}
\]

**Pulse Pressure (SBP – DBP)**

*Wide Pulse Pressure* (> 40 mmHg) associated with:

- Thyrotoxicosis
- Arterial venous fistula
- Aortic insufficiency

*Narrow Pulse Pressure* (< 25 mmHg) associated with:

- Significant tachycardia
- Early hypovolemic shock
- Pericarditis
- Pericardial effusion or tamponade
- Ascites
- Aortic stenosis

**Paradoxical Pulse:** SBP changes during the respiratory cycle as a function of changes in
intrathoracic pressures (see Chapter 13 for the technique to measure the paradoxical pulse). Normally, **SBP falls 6 - 10 mmHg with inspiration.** If this variation *occurs over a wider negative inspiratory range (>10 mmHg)*, the patient is said to have a **paradoxical pulse**.

![Diagram of the paradoxical pulse](image)

**Figure 20–1.** The paradoxical pulse.

(Figure 20–1). Associated conditions include:

- Pericardial tamponade
- Asthma and COPD
- Ruptured diaphragm
- Pneumothorax
Auscultation: *Heart Murmurs*

The presence of a premorbid cardiac murmur and, more importantly, the **interval development of a new cardiac murmur** are important in the care of the critically ill patient. **All new murmurs should be characterized** by their **intensity, location, and variation with position and respiration** as well as being **systolic or diastolic** in nature. In general, **diastolic murmurs are usually pathologic** (see Chapter 1 for more information on heart murmurs).

**Systolic Murmurs:** Abrupt onset of a new systolic heart murmur may be caused by:

1. **Papillary muscle dysfunction / injury:** usually occurs following AMI; characterized by low-grade (II/V) **apical pansystolic murmur**; diagnosis is made by either cardiac catheterization or by echocardiography.

2. **Intraventricular septal rupture:** indicated by abrupt appearance of loud systolic murmur usually following an AMI; usually accompanied by massive pulmonary edema; may require emergency cardiac catheterization and operative repair.

**Diastolic Murmurs:** The major concern of the sudden appearance of a diastolic murmur in the acutely ill or injured patient is **bacterial endocarditis**; more common long-term ICU patients. **Foreign bodies** (i.e., central venous and hyperalimentation lines, pulmonary artery catheters, etc.) contribute to the increasing incidence of bacterial endocarditis.

1. **Gallop:** Defined as three sequential heart sounds in which the first two beats of the triplet are closer together than the third (resulting in a sound that resembles the gallop of a horse). A newly occurring gallop may herald the onset of one or more of the following:

   - AMI
   - Anemia
   - Severe CHF
   - Mitral regurgitation secondary to injury of the papillary muscle
2. **Pericardial friction rub**: Described as the sound of two pieces of leather rubbing together; frequently high pitched and intermittent. Common following open heart surgery and in this setting does not necessarily indicate pathologic changes. Development of a pericardial friction rub should lead to the suspicion of:

- Pericarditis
- Pericardial effusion
- MI near the surface of the pericardium

**CARDIOVASCULAR PHYSIOLOGY**

**Definitions**

**Cardiac Output (CO):** Volume of blood pumped by the heart each minute (ml/min); calculated as heart rate (HR; beats/min) * stroke volume (SV; ml/beat); approximately 3.5 – 5.5 L/min (adult). HR is measured directly; SV depends on the following:

**Preload:** Initial length of myocardial muscle fibers is proportional to the left ventricular end-diastolic volume (LVEDV) which is governed by the volume of blood remaining in the left ventricle after each beat; as LVEDV increases, the stretch on myocardial muscle fibers increases (Panel 1, Figure 20-2); as the LVEDV (i.e., stretch) increases further, the energy of contraction increases proportionally until an optimal tension develops (Starling’s Law; Panel 2, Figure 20-2); when the myocardial muscle fiber is over-stretched, the contractile strength.
Figure 20-2. Representation of Starling's law. PCWP = pulmonary capillary wedge pressure.
**Afterload:** Resistance to ventricular ejection; measured clinically by aortic BP and calculation of systemic vascular resistance (SVR).

**Contractility:** Ability of heart to alter its contractile force and velocity independent of fiber length (i.e., the intrinsic strength of the individual muscle fiber cells). Contractility may be increased by stimulation of β-receptors in the heart (see below).

**Cardiac Index (CI):** Used to standardize CO based on body size; calculated as CO / (patient’s body surface area); normal CI ~ 2.8 – 3.2 L/min/m²; CI < 2.5 L/min/m² may require pharmacologic interventions if it is insufficient for the patients circulatory needs.

### Review of the Sympathetic Nervous System influence on the Cardiovascular System

CO and its determinants (i.e., preload, afterload, and contractility) are all influenced by the sympathetic nervous system (SNS). The SNS releases catecholamines (predominantly epinephrine and norepinephrine) which bind to end-organ receptors and exert a physiological response. Adrenergic receptors are divided into two major classes; alpha (α) and beta (β). End-organ function after receptor activation is summarized in Table 20–1.
Table 20–1. Adrenergic receptors and their actions on the cardiovascular system.

<table>
<thead>
<tr>
<th>Receptor</th>
<th>Location</th>
<th>Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alpha ($\alpha_1$)</td>
<td>Peripheral arterioles</td>
<td>Vasoconstriction (increased SVR)</td>
</tr>
<tr>
<td>Beta ($\beta_1$)</td>
<td>Myocardium</td>
<td>Increased contractility</td>
</tr>
<tr>
<td></td>
<td>SA node</td>
<td>Increased heart rate</td>
</tr>
<tr>
<td>Beta ($\beta_2$)</td>
<td>Peripheral arterioles</td>
<td>Vasodilatation (decreased SVR)</td>
</tr>
<tr>
<td></td>
<td>Bronchiolar smooth muscle</td>
<td>Bronchodilatation</td>
</tr>
</tbody>
</table>

Adrenergic receptors are important because many of the cardiovascular drugs used in the ICU act through their sympathomimetic properties. Such drugs have a specific receptor affinity (i.e., $\alpha$ versus $\beta$) and consequently differ in their end-organ effects. For example, drugs that act on the $\alpha_1$ receptors are called “vasopressors” because they cause non-specific systemic vasoconstriction. Conversely, drugs that act on $\beta_1$ receptor are called “inotropes” because they provide an increase in myocardial contractility and heart rate.

Since each drug exerts receptor-specific effects, their use provides differential activation of receptors and ultimately end-organ effects. Through tailoring pharmacologic support, the physician is able to provide the necessary cardiovascular assistance to critically ill patients. Commonly used sympathomimetics and their relative receptor affinities are listed in Table 20–2. A guide to administration of these agents appears in Table 20–11.
### Table 20–2. Relative actions of sympathomimetic drugs on adrenergic receptors.

<table>
<thead>
<tr>
<th>Drug</th>
<th>α</th>
<th>β₁</th>
<th>β₂</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phenylephrine</td>
<td>++++</td>
<td>0</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Norepinephrine</td>
<td>++++</td>
<td>++</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Epinephrine</td>
<td>++++</td>
<td>++++</td>
<td>++</td>
<td></td>
</tr>
<tr>
<td>Dobutamine</td>
<td>+</td>
<td>++++</td>
<td>++</td>
<td></td>
</tr>
<tr>
<td>Isoproterenol</td>
<td>0</td>
<td>++++</td>
<td>+++</td>
<td></td>
</tr>
<tr>
<td>Dopamine (µg/kg/min)</td>
<td>10 - 20</td>
<td>5 -10</td>
<td>1 - 5</td>
<td></td>
</tr>
</tbody>
</table>

Key: + = Relative effect; 0 = No clinically significant effect; D = Dopaminergic receptors

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**CENTRAL VENOUS PRESSURE (CVP)**

The CVP catheter is one of two major devices used for cardiovascular instrumentation. The other, the pulmonary artery catheter (a.k.a., PA catheter, Swan–Ganz catheter, or right-heart catheter), is considered in the next section. For CVP monitoring, a 14-gauge intravenous catheter is inserted into the central venous circulation via the internal jugular or subclavian vein (see Chapter 13). A pressure transducer and monitor connected to the catheter provide the measurements. A chest x-ray is required to confirm the position of...
the catheter in the superior vena cava. The zero point for the transducer is usually 5-cm below the sternal notch in the midaxillary line.

The CVP reading reflects right atrial pressures, and by association, right ventricular filling pressure. This filling pressure, or preload, is one determinant in the ability of the heart to pump blood (see Preload above). More importantly are the relative changes that take place in patient’s CVP as the fluid or cardiac status changes. Therefore, serial readings are recorded and compared over time with other physiologic measurements. The general implications of CVP readings are listed in Table 20–3.

<table>
<thead>
<tr>
<th>Reading (mmHg)</th>
<th>General</th>
<th>Clinical Description</th>
<th>Implications</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 3</td>
<td>Low</td>
<td>Intravenous fluids may be administered</td>
<td></td>
</tr>
<tr>
<td>3–10</td>
<td>Midrange</td>
<td>Probable clinical euvolemma</td>
<td></td>
</tr>
<tr>
<td>&gt;10</td>
<td>High</td>
<td>Suspect fluid overload, CHF, CP, COPD, tension PTX</td>
<td></td>
</tr>
</tbody>
</table>

CVP = central venous pressure, CHF = congestive heart failure, CP = cor pulmonale, COPD = chronic obstructive pulmonary disease, PTX = pneumothorax
**CVP Limitations**

- CVP does not entirely reflect total blood volume or left ventricular function
- CVP will be altered by:
  - changes in pulmonary artery resistance
  - changes in compliance of the right ventricle
  - intrathoracic pressures (i.e., mechanical ventilation)
- An accurate clinical picture may be limited by conditions that radically change intrathoracic pressure:
  - positive pressure ventilation, especially when high PEEP is used
  - pneumothorax, hemothorax, hydrothorax, or tension pneumothorax
  - the presence of intra-thoracic tumors
- CVP may be normal in the face of sepsis or hypovolemia when accompanied by compromised myocardial function
  - Occult left ventricular failure may occur in the presence of normal CVP
  - Patients with COPD may require an elevated CVP to optimize their cardiac output
- PA catheter readings are more accurate than CVP with regard to a patient’s fluid and cardiac status

**Technical Tips Regarding CVP Measurements**

- CVP readings are inaccurate if they do not fluctuate with respiration
- If appropriate, remove the patient from the ventilator when taking a CVP reading
- To ensure comparable readings, have the patient positioned in the same manner
for each measurement

  o Flatten the bed and use the same zero point for the transducer (5-cm below the sternal notch in the midaxillary line)
Figure 20–3. Relative positioning of the pulmonary artery catheter.
PULMONARY ARTERY CATHETERS

The pulmonary artery (a.k.a., PA, Swan–Ganz, or right-heart) catheter is a device that allows the direct measurement of central cardiovascular pressures which, after the appropriate calculations are performed, then yield important circulatory parameters useful in the treatment of acutely ill patients. The catheter is placed in a central vein (usually the subclavian or internal jugular) and then actually passes into the right atrium, across the tricuspid valve, into the right ventricle, through pulmonic valve with the distal end subsequently “floated” into the pulmonary artery (Figure 20–3). The PA catheter then allows the measurement of the pulmonary artery pressure (PAP), the pulmonary artery occlusion pressure (PAOP, also known as the pulmonary capillary wedge pressure, PCWP), and the CVP. Intravascular volume status, vascular tone (both pulmonary and systemic), and the heart’s pumping ability (cardiac output) are all then calculated. Newer technology also allows for the continuous monitoring of mixed venous oxygen saturation (SvO₂), measurement of the right ventricular ejection fraction (REF), and the right ventricular end diastolic volume index (RVEDVI).

Key point: The data obtained with a PA catheter is only as good as the initial setup and the actual measurements obtained (i.e., pressures). If the pressure measurements are in error or if patient data (height, weight, etc.) are incorrectly entered into the system, the subsequent calculations will be incorrect.

Indications
Common clinical conditions requiring PA catheter monitoring include:

- Acute heart failure  
- Shock states  
- Complex circulatory and fluid conditions (massive resuscitation)  
- Diagnosis of pericardial tamponade  
- Complicated MI  
- Intraoperative management in high-risk cardiac patients (i.e., aneurysm repair, elderly patient undergoing major surgery)
Catheter Description

The PA catheter generally consists of three (or four) lumens and a thermistor at the tip (Figure 20–4); markings are typically in 10-cm increments; the catheter is radiopaque

**Lumens:**

- **Balloon port:** usually a square white port; route to inflate the balloon at the tip of
the catheter; inflation of the balloon requires between 1.0 – 1.5 ml of air

- **Proximal port:** approximately 30-cm proximal to the tip; lies in the superior vena cava; may be used for fluid administration when not used for determinations of CVP and CO

- **Distal port:** lies in the PA beyond the balloon; this port is attached to a pressure transducer for continuous PAP tracings and intermittent PAOP measurement

**Thermistor:** temperature sensor that provides **continuous core temperature measurement** as well as measurements used in the thermal dilution CO techniques (see below)

**Modifications of Pulmonary Artery Catheter:** Several modifications of the original PA catheter allows for additional functions and measurement capabilities:

- **Pacing PA catheters:** extra ports (approximately 19 cm from the tip) through which pacing wires are passed into the right ventricle; other models contain electrodes along the surface of the catheter; capable of pacing both the right atrium and ventricle

- **Oximetric PA catheter:** standard PA catheter ports with fiberoptic components; emit light impulses to and from distal end of catheter; light impulses are then reflected back by hemoglobin and measured; allows continuous O₂ saturation monitoring (Figure 20–4)

- **Right ventricular ejection catheter:** determines right ventricular ejection fraction (REF) which is then used to calculate the RVEDVI (best indicator of preload)

**Contraindications for PA Catheter Use:** There are NO absolute contraindications if a PA catheter is needed to treat a patient in a critical care setting. Patients with **LBBB** may experience **complete heart block** (may require temporary pacemaker placement); **frequent manipulation** may increase the **risk of infection** (similarly to other iv catheters).
Materials

There are many versions of the flow-directed, balloon-tipped PA catheter (see generic representation in Figure 20–4). A PA catheter introducer insertion kit provides the introducer sheath (cordis catheter), flexible J-tip guidewire, vessel dilator, catheter contamination shield, and various other items needed to insert the catheter (Figure 20–5). The monitoring system (i.e., transducers, tubing, stopcocks) and pressurized flush system are usually set up by the nursing staff and should be operational prior to catheter insertion.

Pulmonary Artery Catheterization Procedure

1. Informed consent is usually required since these catheters are usually placed in very ill
patients. Subsequently, the patient’s medical decision maker should grant consent.

2. The patient should be in the ICU with continuous ECG and hemodynamic monitoring. Emergency resuscitation medications must be on hand in the event of a refractory arrhythmia.

3. Choose a site (usually dictated by patient variables and operator experience). In a patient who may receive thrombolytic therapy or who has a coagulopathy, femoral and internal jugular veins are better routes due to their compressibility if a complication occurs. The easiest sites to “float” the PA catheter are the right internal jugular and the left subclavian vein. Rationale: the PA catheter is packaged in a coiled position; these sites lend to the natural curve of the catheter as it assists in placement.

4. The insertion site should be widely prepped with a topical anti-infective agent. Povidone iodine or chlorhexidine solutions are the most commonly used anti-infectives. IMPORTANT: Anti-infective agents must fully dry on the skin to be efficacious.

5. Full draping of the patient (not just the immediate site) is needed because of the length of the tubing and guidewire. Use a strict sterile approach with gown, gloves, and mask. Strict attention to sterile technique will decrease the rate of line infections greater than 6-fold.

6. With the patient in Trendelenburg position, cannulate the central vein (see Central Venous Catheterization, Chapter 13). Pass the flexible end of the J-wire (standard size: 45 cm long) into the vein through the needle. In general, never push a guidewire where it does not want to go and always keep one hand on the guidewire while it is in the patient (make sure the flexible tip end is passed since the stiff end may perforate the blood vessel).

7. Mount the introducer sheath on the vessel dilator. Pass the dilator / sheath unit over the wire. Make a full-thickness skin nick at the wire entry site (# 11 blade provided in the set).
8. **Pass the vessel dilator / sheath unit into the vessel using strict Seldinger technique** (Figure 20-6). A gentle, slight twisting motion may be necessary. Slowly remove the guidewire and the vessel dilator. Catheter sheaths have a hemostatic valve mechanism to prevent air from entering the central system and blood from escaping (place a finger over the end of the sheath if no valve is present); however, the side port does not so this should be capped or clamped. Mount a syringe on the side port and aspirate blood to confirm intravascular positioning of the sheath; flush with sterile saline after confirmation.

9. **Prepare the PA catheter** (attach to the monitor, flush lumens with sterile saline). Set the level of the pressure transducer to the middle of the patient’s chest (approximately the level of the left atrium), then zero the monitor. **Check balloon function** and **gently wave the**
catheter to ensure that an appropriate waveform is present on the monitor. Note:

Never fill the balloon with fluid; use only air. The volume is typically 1.0 – 1.5 mL (dependent on the size of the PA catheter). Next, place the catheter through the contamination shield.

10. The prepared catheter (fluid-filled, fully monitored with contamination sheath in place) may now be inserted into the sheath (Figure 20–7). Once you have advanced approximately 15 – 20 cm, gently inflate the balloon with 1.0 – 1.5 mL of air using the volume-limiting syringe provided with the set. If you encounter resistance to full inflation, the balloon may not have yet cleared the sheath (~ 20 cm) or that it may be extravascular.
11. Once the balloon is inflated, advance the catheter to the level of the right atrium under the guidance of the pressure waveform and the ECG. *Monitor the waveform and ECG at all times while advancing the balloon catheter.* Figure 20–8 displays the normal pressures that are encountered as the catheter is advanced. **IMPORTANT:** *ALWAYS advance the catheter with the balloon inflated.* *NEVER advance the catheter with the balloon deflated.* Conversely, *ALWAYS withdraw the PA catheter with the balloon deflated.*
12. Positioning of the PA catheter in the right atrium is probably best determined by watching for the characteristic waveform on the monitor (Figure 20-8). The right atrium is generally located approximately 30 cm from the right internal jugular or subclavian vein.
insertion site and approximately 35–40 cm from the left subclavian vein insertion site.

13. An abrupt change in the pressure tracing occurs as the catheter enters the right ventricle (Figure 20-8). There is generally little ectopy on entry into the right ventricle; however, as the catheter advances into the right ventricular outflow tract, PVC’s may occur.

14. Steadily advance the catheter until the ectopy disappears and the pulmonary artery tracing is obtained (Figure 20-8). If this does not occur (by the time 60 cm is reached), deflate the balloon, withdraw the catheter to 20 cm, and make another attempt with the balloon inflated after slightly rotating the catheter.

15. Once in the pulmonary artery, obtain the PCWP after advancing the catheter another 10–15 cm. The catheter’s final position should be such that the PCWP is obtained with full balloon inflation and the PAP tracing is present with the balloon deflated. In the “ideal position,” transition from PAP to PCWP (and vice versa) occurs within three or fewer heart beats. In an adult, the typical length to the pulmonary artery position is 45–60 cm. Table 20–4 shows normal PA catheter measurements important for patient evaluation and management.

16. Once the position is acceptable, lock the contamination shield onto the sheath. This allows readjustment of the catheter should this be necessary after the sterile field is taken down. Suture the sheath to the patient (using 3-0 nylon or 2-0 silk on a cutting needle), secure the catheter in place and dress the surgical site according to your institution’s practice. Connect catheters to the ports on the sheath. Inflow port on the sheath may be used for IV fluid and medication administration.

17. Obtain a chest x-ray to document the catheter’s present position as well as to rule
out a pneumothorax or other complication from central venous catheterization. A properly positioned catheter should lie just beyond the vertebral bodies in the non-wedged position.

18. **Common problems:** Catheter placement is more difficult if **severe PA hypertension** is present. If there is significant **cardiac enlargement**, particularly dilation of the right heart structures, the catheter may coil in its path to the RV outflow tract (fluoroscopy may be required for correct positioning). Further, under these conditions, the PA catheter may have **difficulty holding its proper position**. Placement of the catheter in the pulmonary artery may also be difficult in the setting of a **low cardiac output state** because the balloon-tipped catheter depends on blood flow to carry it through the right heart chambers.

19. **Cardiac output** may be measured by **thermal dilution** (i.e., Fick’s equation). Connect the thermistor to the cardiac output computer and then rapidly inject fluid (usually 10 mL of ice-cooled NS) through the right atrial port. The computer displays a curve and the CO is calculated from the **area under the thermal dilution curve**. Repeat two more times. If all of these values are approximately the same, then average the readings and record. Newer continuous cardiac output monitoring PA catheters are available in some units. Normal values for cardiac output and cardiac index are listed in Table 20–4.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Right atrial pressure</td>
<td>1 – 7 mm Hg</td>
</tr>
<tr>
<td>Right ventricular pressure</td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>15 – 25 mm Hg</td>
</tr>
<tr>
<td>Diastolic</td>
<td>0 – 8 mm Hg</td>
</tr>
<tr>
<td>PAP</td>
<td></td>
</tr>
<tr>
<td>Systolic</td>
<td>15 – 25 mm Hg</td>
</tr>
<tr>
<td>Measurement</td>
<td>Value</td>
</tr>
<tr>
<td>----------------------</td>
<td>----------------</td>
</tr>
<tr>
<td>Diastolic</td>
<td>8 – 15 mm Hg</td>
</tr>
<tr>
<td>Mean</td>
<td>10 – 20 mm Hg</td>
</tr>
<tr>
<td>PAOP (“wedge pressure”)</td>
<td>6 – 12 mm Hg</td>
</tr>
<tr>
<td>Cardiac output</td>
<td>3.5 – 5.5 L/min</td>
</tr>
<tr>
<td>Cardiac index</td>
<td>2.8 – 3.2 L/min/m2</td>
</tr>
<tr>
<td>Mixed venous O₂ saturation</td>
<td>65 – 85 %</td>
</tr>
</tbody>
</table>

PAP = pulmonary artery pressure, PAOP = pulmonary artery occlusion pressure

Complications of PA Catheters

1. **Most complications** occur in the course of PA catheterization and are related to central vein cannulation: arterial puncture with subsequent bleeding problems, placement of the wire or catheter in the extravascular space, and pneumothorax / hemothorax.

2. **Arrhythmias** are another common complication with transient PVC’s being the most frequent. PVC’s occur when the catheter is advanced into the right ventricular outflow tract. If a patient with a PA catheter suddenly develops frequent PVC’s, deflate the balloon and pull back the catheter to prepare for another attempt.

3. **VT and VF are rare occurrences.** If they continue after the catheter has been withdrawn, institute standard ACLS protocols and treat the patient according to published algorhythms.

4. **Transient RBBB occurs occasionally** as the catheter passes through the right ventricular outflow tract. *In a patient with preexisting LBBB, this may result in complete heart block.* In this setting, some form of backup pacing should be readily available. Complete heart block has been reported but occurs rarely.
5. Pulmonary infarcts and PA rupture are serious but infrequent complications of PA catheters and are usually secondary to an “over-wedge” or peripheral placement of the catheter. The patient should be placed affected-lung-down, intubated (if not already done), and the ET tube placed into the unaffected mainstem bronchi to protect the airway. Emergent thoracic surgery consultation is then warranted.

6. Most complications and problems tend to increase with the time the catheter is in place. The risk of bacteremia and spontaneous bacterial endocarditis (SBE) are significant in severely ill patients receiving chronic instrumentation. In the setting of unexplained fever, the PA catheter and sheath should always be removed and cultured. The catheter and sheath should be replaced at a different site if a pulmonary catheter is still indicated.

**PA Catheter Measurements**

**PA Pressure:** Measured when the PA catheter is in its resting position (balloon deflated). Measurements include pulmonary systolic arterial pressure (PAS), mean arterial pressure, and diastolic (PAD) arterial pressure.

**Pulmonary Artery Occlusion Pressure (PAOP):** (a.k.a., the “pulmonary capillary wedge pressure,” “PCWP”, or “wedge pressure”). This measured parameter is a reflection of the left atrial pressure and is measured when the balloon at the tip of the PA catheter is slowly inflated with air to occlude a branch of the PA.

**IMPORTANT:** The balloon must be fully deflated when not in active use to avoid pulmonary infarction.

In the absence of mitral valvular disease, PAOP correlates closely with the left atrial
pressure (LAP) and with the left ventricular end-diastolic pressure (LVEDP). This correlation exists because of the unobstructed continuity between the pulmonary artery and the left side of the heart. As a result of this continuity, the PAOP may never be greater than the PAD. If the LVEDP increases, this should be reflected by an increase in PAOP, which, in turn, increases the PAD. Therefore, if a PA catheter monitor shows a wedge pressure higher than the PAD pressure, a technical error must exist.

**Left Ventricular End-Diastolic Pressure:** LVEDP is a measure of preload and is used to guide fluid resuscitation to optimize cardiac output. Recall that to optimize stroke volume on the Starling curve, the preload must be adequate to stretch the wall of the left ventricle (Figure 20–2). Hypovolemia results in too little tension on the muscle fibers and therefore a decreased SV and CO. Conversely, too much preload stretches beyond the point of maximum tension and causes a decrease in CO. Clinically, the LVEDP and PAOP are used to keep preload in an optimum range. The normal PAOP varies between 6 and 12 mmHg, but may be higher for different disease states and for pre-existing cardiac disease leading to decreased chamber compliance.

**Right Ventricular Ejection Fraction (REF) / Right Ventricular End-Diastolic Volume Index (RVEDVI):** A rapid-response thermistor and the CO computer are used to calculate the REF. Once REF and CO are known, the RVEDVI may be calculated. The RVEDVI is another measure of preload, and it allows a more accurate assessment of volume status regardless of pulmonary disease. For example, a patient with severe ARDS may have markedly elevated peak inspiratory pressures. Although the CVP and PAOP may be falsely elevated, the RVEDVI is calculating a volume, not a pressure, thus allowing a determination
of volume status across a wide variety of clinical situations. The normal range for EDVI is 80–120 mL, but as with any value based on calculations, must be viewed with caution if the proper conditions are not met (i.e., bad data in \(\rightarrow\) bad data out).

**Differential Diagnosis of PA Catheter Abnormalities**

Table 20–4 shows normal PA pressures and cardiovascular performance measurements (see also Figure 20–8). Perturbations of these values indicate a disease process. The broad differential diagnoses based on these alterations are shown in Table 20–5.

**Clinical Applications**

The PA catheter allows the clinician to approximate the patient’s volume status and myocardial performance. As stated earlier, myocardial performance or CO depends on HR and SV. SV is, in turn, dependent on preload, afterload, and contractility.

**Heart Rate:** HR, in addition to SV, determines the CO (i.e., \(\text{CO} = \text{HR} \times \text{SV}\)). The body increases HR to increase CO in the face of inadequate tissue perfusion. Hence, tachycardia is an additional indicator of \(\text{O}_2\) debt (i.e., delivery / demand deficit). Tachycardia >120 bpm increases myocardial \(\text{O}_2\) demand significantly and should be promptly treated. The PA catheter allows the establishment of adequate myocardial filling pressures such that the HR may be clinically manipulated to maximize CO. In a patient with adequate filling pressures, slow HR (<80 bpm), and a low CO, drugs that speed up the heart (called “chronotropes”) may be used to increase CO. Alternatively, tachycardia >120 bpm with an adequate PAOP may be pharmacologically slowed to decrease the strain on the heart.
Preload (Stroke Volume): Indicated by the PAOP or EDVI, a reflection of left ventricular end-diastolic volume. In simple terms, preload is the amount of blood in the heart prior to contraction. Consequently, preload represents the stretch placed on the individual myocardial cell. When the PAOP is optimized, myocardial performance is optimized according to the Starling curve.

1. Clinical implications in a healthy heart. A low PAOP or EDVI means suboptimal myocardial muscle stretch. CO may be increased first by the administration of fluids. The result is an increase in LVEDV, an increase in myocardial muscle tension, and improved myocardial performance.

2. Clinical implications in a failing heart. Long-standing myocardial disease may shift the Starling curve to the right. Consequently, a significantly elevated PAOP may be required to optimize myocardial performance. It is common for patients who have just undergone heart valve replacement to require a PAOP of 20–25 mmHg to optimize cardiac output (due to decreased compliance of the post-operative heart muscle). Patients with a recent MI may similarly require a PAOP of 16–18 mmHg to optimize output.

Afterload: This is defined as the resistance to ventricular ejection and is measured clinically by the calculation of systemic vascular resistance (SVR). A normal SVR = 900 - 1200 dynes/sec/cm³.

1. Indications for afterload reduction.
   • Significant mitral regurgitation
• An increased PAOP coincident with elevated SVR / decreased cardiac index


**Contractility:** The ability of the heart to alter its contractile force and velocity independent of fiber length. This aspect is difficult to directly measure clinically, but may be estimated through surrogate markers. Correctable metabolic causes for depressed contractility include:

- Hypoxia
- Acidosis (pH < 7.3)
- Hypophosphatemia
- Adrenal insufficiency
- Hypothermia

Improving contractility may be achieved by adding adrenergic agonists. Digoxin may also improve myocardial contractility; however, ensure normal levels of serum potassium prior to the administration of the drug.

<table>
<thead>
<tr>
<th>TABLE 20–5. Differential diagnosis by category based on perturbations in hemodynamic parameters.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systemic Diagnosis</td>
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<tr>
<td></td>
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<tr>
<td></td>
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<tr>
<td>Cardiogenic shock</td>
</tr>
<tr>
<td>Cardiac tamponade</td>
</tr>
<tr>
<td>Pulmonary embolism</td>
</tr>
<tr>
<td>Hypovolemic shock</td>
</tr>
<tr>
<td>Neurogenic shock</td>
</tr>
<tr>
<td>Septic shock</td>
</tr>
</tbody>
</table>
These are the trends that are usually seen with the conditions noted. Clinical variables (medications, secondary conditions, etc.) may vary these trends somewhat. **Highlighted areas** denote major differences between subgroups.

CVP = central venous pressure; CO = cardiac output; PAOP = pulmonary artery occlusion pressure; LVEDP = left ventricular end-diastolic pressure; PAP = pulmonary artery pressure; PVR = peripheral vascular resistance; SVR = systemic vascular resistance.

⇑ = usually increased; ⇓ = usually decreased; — = usually unchanged.

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**DETERMINATIONS OF CARDIAC OUTPUT**

Methods available to determine CO include thermal dilution, A–V O₂ difference calculation, and continuous cardiac output measurements.

**Thermal Dilution Technique**

This technique requires the use of a PA catheter. A measured amount of saline (usually 10 mL) at a known temperature is injected into the proximal port of the PA catheter, and a temperature-sensitive thermistor located at the distal end of the pulmonary artery senses the temperature change in the surrounding blood. The cardiac output computer then creates a curve over time integrating the magnitude and rate of change in temperature. Utilizing the Fick equation, CO is then calculated as the area under the curve.
**Arteriovenous Oxygen (A–VO$_2$) Difference**

A reasonable estimate of cardiac output may be made on the basis of A–VO$_2$ difference. A–VO$_2$ difference is calculated as the oxygen content of arterial blood drawn from a peripheral artery minus the oxygen content of mixed venous blood drawn from the distal lumen of a PA catheter (Table 20–6).

<table>
<thead>
<tr>
<th>A-VO$_2$ Difference (vol %)</th>
<th>Cardiac Index (L/min/m$^2$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 6</td>
<td>&lt; 2</td>
</tr>
<tr>
<td>4–5</td>
<td>3 – 4</td>
</tr>
<tr>
<td>&lt; 3</td>
<td>&gt; 5</td>
</tr>
</tbody>
</table>

A–VO$_2$ difference = Arterial O$_2$ content - Mixed venous O$_2$ content; Cardiac index = Cardiac output / Body surface area.

**Concept:** The A–VO$_2$ difference measures the extraction of oxygen by the tissues during a single transit time through the circulation. Thus, the A–VO$_2$ difference is a function of (1) PaO$_2$, (2) Hemoglobin concentration, (3) CO, and (4) tissue O$_2$ consumption.

- **If cardiac output is low:** Transit time is long and the tissues extract large amounts of oxygen during a single circulation time. Thus, the oxygen content of mixed venous blood is low and the A–VO$_2$ difference is large

- **If cardiac output is high:** Conversely, circulation time is shorter and the amount of oxygen extracted is lower. As a result, the A–VO$_2$ difference is low.

**Calculations:** Since the A–VO$_2$ difference is inversely proportional to CO, the following approximations may be made:
1. **Determining Oxygen Content.** To calculate the A–VO\(_2\) difference, the oxygen content of both arterial and mixed venous blood must be determined. Oxygen content *per se* describes the amount of \(O_2\) the blood is able to carry, thus:

Oxygen content (mL \(O_2/dL\) blood) = Oxygen bound to Hgb + Oxygen dissolved in plasma

so:

Arterial \(O_2\) content (CaO\(_2\)) = \(\text{SaO}_2 \times [\text{Hgb}] - 1.39\) + \((0.0031 \times \text{PaO}_2)\)

where:

\(\text{SaO}_2\) = arterial \(O_2\) saturation

\([\text{Hgb}]\) = hemoglobin content (g/dL)

\(\text{PaO}_2\) = arterial partial pressure of \(O_2\) (mm Hg)

The normal \(O_2\) content of arterial blood is 16–20 mL of \(O_2/100\) mL of blood. The constant 1.39 is the \(O_2\)-binding capacity of Hgb (mL of \(O_2/g\) of Hgb), and 0.0031 is the mL of \(O_2\) dissolved in 100 mL of plasma per mmHg of PaO\(_2\). This clearly demonstrates that only a small percentage of \(O_2\) is dissolved in plasma and that the vast majority is carried by hemoglobin. This is very important when evaluating the \(O_2\) carrying capacity of blood.

Similarly:

Venous \(O_2\) content (CvO\(_2\)) = \(\text{SvO}_2 \times [\text{Hgb}] - 1.39\) + \((0.0031 \times \text{PvO}_2)\)

where:

\(\text{SvO}_2\) = mixed venous \(O_2\) saturation

\(\text{PvO}_2\) = peripheral venous partial pressure of \(O_2\) (mm Hg)

Assuming that the amount of \(O_2\) dissolved blood in plasma is small, then:

\[\text{A–VO}_2\text{ difference} = \text{CaO}_2 - \text{CvO}_2 = 1.39 \times [\text{Hgb}] \times (\text{SaO}_2 - \text{SvO}_2)\]

2. **Calculation of A–VO\(_2\) Difference.**

- Obtain hemoglobin concentration through standard laboratory means
• Determine the peripheral SaO\textsubscript{2} from heparinized arterial blood or from a reliable pulse oximeter

• Determine the SvO\textsubscript{2} from a heparinized mixed venous blood sample from the distal lumen of a PA catheter or from an oximetric SvO\textsubscript{2} monitor (see the following discussion)

• Calculate the A–VO\textsubscript{2} difference according to the preceding formula

• Determine the CI based on the data contained in Table 20–6

**Continuous SvO\textsubscript{2} Monitoring**

Oximetric PA catheters house fiberoptic channels that allow direct measurement of mixed venous Hgb saturation (SvO\textsubscript{2}). These fiberoptics carry light impulses that are reflected by Hgb according to its O\textsubscript{2} saturation. An optical microprocessor then displays a continuous graph of SvO\textsubscript{2} measurements. Calibration is periodically confirmed by ABG’s measured from heparinized blood drawn from the oximetric catheter’s **distal port**.

**Clinical Application**

• Follow trends in the O\textsubscript{2} supply / demand balance

• **A decrease in SvO\textsubscript{2} is THE BEST indicator of decreased peripheral O\textsubscript{2} delivery.** This is an early sign of organ dysfunction and should allow correction of the problem before hemodynamic compromise occurs

• **Fix the underlying cause.** Treatment interventions (i.e., transfusions, fluid administration, inotropic drug use, etc.) may be assessed by following SvO\textsubscript{2} changes long
before other hemodynamic parameters are adversely affected

- Clinically, \( \text{SvO}_2 \) values between 65% and 85% represent adequate tissue \( O_2 \) delivery and extraction. This generally implies appropriate perfusion of peripheral tissues.

- \( \text{SvO}_2 \) of < 60% should prompt an immediate assessment of \( O_2 \) delivery. As \( O_2 \) delivery falls, \( \text{SvO}_2 \) falls proportionally because there is less \( O_2 \) for the tissues to extract.

- Conversely, if \( \text{SvO}_2 \) is < 60% and \( O_2 \) delivery is unchanged, unrecognized conditions causing increased \( O_2 \) demand should be identified.

In summary, a decline of \( \text{SvO}_2 \) must prompt a review of the parameters describing \( O_2 \) delivery (i.e., CO, [Hgb], \( \text{SaO}_2 \)) and consumption (\( \text{SaO}_2 - \text{SvO}_2 \)). The specific remedy for these declines of \( \text{SvO}_2 \) includes:

- Supplemental \( O_2 \) and / or ventilatory support for \( \text{SaO}_2 < 90\% \)

- Optimization of myocardial performance for decreased CO

- RBC transfusion for low Hgb states

- Identification and treatment of conditions leading to increased metabolic demands (i.e., unrecognized seizures, shivering, excessive mobilization, and large tissue defects) as these produce a significant increase in \( O_2 \) demand (Figure 20–9)

- Inaccurate readings of \( \text{SvO}_2 \) may occur as a result of fibrin buildup on the tip of the catheter, fiberoptic fracture (rare), or impingement of the tip of the catheter on the vessel wall. Overall, however, these catheters are extremely accurate and sensitive (with daily calibration and catheter maintenance).
Continuous Cardiac Output Measurement

New technology allows measurement of the CO on a continuous basis. The specially designed PA catheter emits small pulses of energy that heat the surrounding blood. The cardiac output computer then calculates the CO based on the magnitude and the rate of temperature change (with the Fick equation). This continuous measurement, as well as its many calculated derivatives, is intermittently updated and displayed on the device.

Continuous SaO₂ Monitoring (Pulse Oximeter)

The same fiberoptic technology used to measure mixed venous O₂ saturation is also used to measure arterial O₂ saturation (SaO₂). A light-emitting external probe is placed
around a well-perfused appendage such as a digit, earlobe, lip, or bridge of the nose. The light is transmitted through the appendage to be reflected by hemoglobin according to its O\textsubscript{2} saturation (recall that the hemoglobin molecule absorbs different wavelengths of light at different O\textsubscript{2} saturations). The oximeter, in addition to calculating Hgb O\textsubscript{2} saturation, may also determine the pulse rate and is referred to as the “pulse oximeter.” A SaO\textsubscript{2} of < 90 % implies inadequate oxygenation and under most circumstances requires immediate intervention. One exception would be a patient with severe COPD who may have a normal O\textsubscript{2} saturation in the upper 80% range. Conversely, a SaO\textsubscript{2} > 90% does not necessarily imply adequate O\textsubscript{2} delivery (see following section). Pulse oximeter is not useful in the setting of smoke inhalation and CO poisoning as the Hgb molecule has a higher affinity for CO than for O\textsubscript{2}.

![Figure 20–10. Ventilation and oxygenation in typical alveoli.](image-url)
CLINICAL PULMONARY PHYSIOLOGY

The goal of treating any critically ill patient is to optimize oxygenation, ventilation, and tissue perfusion. Pulmonary and cardiovascular physiologies are intimately interwoven to achieve this goal. Optimizing cardiovascular function carries little benefit if there is no $O_2$ for the Hgb to transport (i.e., low $SaO_2$). Basic pulmonary physiology concepts include (Figure 20–10):

*Ventilation*: mechanical movement of air into and out of the respiratory system; primarily results in excretion of $CO_2$

*Oxygenation*: diffusion of $O_2$ from the alveoli into the pulmonary capillary blood for systemic distribution

**Ventilation**

Several parameters, such as volumes and capacities, are important in assessing the adequacy of ventilation. Spirometry provides both dynamic information (i.e., ability to move air into and out of the lungs) as well as static volume measurements. The lung volume subdivisions and capacities are shown on a spirometric graph (Figure 20–11).
Lung Volumes: Total Lung Capacity (TLC), or the amount of gas in the lung at full inspiration, is comprised of four basic lung volumes:

1. **Inspiratory Reserve Volume (IRV):** The volume of gas that may be maximally inspired beyond the standard, resting tidal volume breath

2. **Tidal Volume (TV):** The volume of inspired gas during a normal breath; approximately 6 – 8 mL/kg in resting, healthy adults

3. **Expiratory Reserve Volume (ERV):** The volume of gas that may be maximally expired beyond the amount expired at the end of a normal tidal volume breath

4. **Residual Volume (RV):** The volume of gas that remains in the lung after a maximal expiratory effort
**Lung Capacity:** The sum of two or more of these lung volumes make up four divisions called lung capacities (See Figure 20–11).

1. **Vital Capacity (VC):** The *volume of gas expired after a maximal inspiration followed by maximal expiration* \( \text{VC} = \text{ERV} + \text{TV} + \text{IRV} \). VC is frequently used in determining whether a patient may successfully be weaned from the ventilator (normal VC ~ 65 – 75 mL/kg; \( \text{VC} < 15 \text{ mL/kg} \) is an indication for continued ventilatory support)

2. **Inspiratory Capacity (IC):** The *volume of gas expired from maximal inspiration to the end of a normal, resting TV* \( \text{IC} = \text{TV} + \text{IRV} \)

3. **Functional Residual Capacity (FRC):** The *amount of gas remaining in the lung following a normal TV expiration* \( \text{FRC} = \text{ERV} + \text{RV} \); acts as a buffer against extreme changes in alveolar PO\(_2\) and consequent dramatic changes in arterial PO\(_2\) with each breath
Clinical Implications

These volumes and capacities are important factors in assessing ventilation because they may change under different conditions (i.e., atelectasis, obstruction, consolidation, small airway collapse, etc.). For example, as the ERV decreases with small airway collapse the FRC is similarly decreased (Figure 20–12). These alterations in lung volumes consequently affect respiratory reserve and the patient’s ability to ventilate and oxygenate. The contributing factors and the point at which they influence such volume changes must be understood to optimize support.

Critical Closing Volume (CCV): the minimum volume and pressure of gas necessary to prevent small airways from collapsing during expiration. When collapse occurs, blood is shunted around non-ventilated alveoli. This decreases the available surface area for gas exchange. The CCV is greatly affected by lung compliance. Therefore, different minimum volumes and pressures may be required to prevent collapse under varying lung conditions as compliance changes. If the CCV is greater than the FRC (air in the lung after tidal expiration), collapse tends to occur at a higher proportion of airways (Figure 20–12).

One method to overcome the CCV is to increase the amount of Positive End-Expiratory Pressure (PEEP) in the lung (see below for the discussion on PEEP). The effect of PEEP is to increase FRC by minimizing small airway collapse at the end of expiration. This improves alveolar ventilation, decreases shunting, and ultimately improves oxygenation (Figure 20–13).
Lung Compliance: expresses the change in lung volume and the change in pressure required to produce such a volume change (Figure 20–14). May be measured at the bedside and is a reflection of FRC and CCV.

Lung Compliance = ΔV / ΔP

**Dynamic Compliance:** determined by measuring the tidal volume and dividing it by the peak inspiratory pressure.

\[
\text{Dynamic Compliance} = \frac{TV}{\text{PIP} - \text{PEEP}} = \sim 80–100 \text{ mL/cm H}_2\text{O}
\]

where: PIP = Peak Inspiratory Pressure

**Static Compliance:** similar to Dynamic Compliance, except Static PIP is substituted for PIP. Static peak pressure (a.k.a., Plateau Pressure) is measured by occluding the exhalation port at the beginning of exhalation (no flow = static pressure).

Comparing dynamic with static compliance may indicate the type of processes
causing changes in the elasticity of the lung. *Dynamic compliance is affected by both elasticity and airway resistance*. *Static compliance*, in contrast, is not affected by airway resistance because there is no flow.

1. *Reduction in dynamic compliance without a change in static compliance* indicates an *airway resistance problem* (i.e., obstruction, bronchospasm, or collapse of the small airways)

2. *Reduction in both static and dynamic compliance* indicates a *decrease in lung elasticity* (i.e., pulmonary edema, atelectasis, or excessive PEEP)
**Oxygenation**

Oxygenation is the process of transporting oxygen from the alveolus across the capillary membrane into the pulmonary circulation and subsequently distributing that oxygen to the body’s tissues. To assess the patient’s ability to properly oxygenate, the following should be considered:

- **Arterial Hgb content (gm/dL)** – obtained by standard laboratory measurements
- Measurement of **systemic arterial O$_2$ saturation (SaO$_2$)** – see above for details
- Calculation of **oxygen carrying capacity (CaO$_2$)** and **oxygen delivery (DO$_2$)**
- Calculation of **Alveolar-to-arterial (A-a) gradient** and **right-to-left shunt fraction (Qs/Qt)**

**Oxygen Carrying Capacity:** The ability of the blood to carry O$_2$ to the periphery is dependent on the O$_2$ content (CaO$_2$). The CaO$_2$ is directly influenced by Hgb concentration and the saturation of Hgb with O$_2$ (SaO$_2$) (i.e., $\text{CaO}_2 = \text{SaO}_2 \times \{1.39 \text{ [Hgb]}\}$)

**Oxygen Delivery:** Delivery of O$_2$ to the tissue depends on the CaO$_2$ and the CO.

\[
\text{O}_2 \text{ delivery (DO}_2; \text{ mL O}_2/\text{min}) = \text{CaO}_2 \times \text{CO} = \text{SaO}_2 \times \{1.39 \text{ [Hgb]}\} \times \text{CO} (\text{L/min})
\]

These variables are measured with a PA catheter, pulse oximeter, and measured Hgb concentration. **Normal DO$_2$ is around 800 mL of O$_2$/min,** with an average normal O$_2$ uptake of 250 mL of O$_2$/min.

**Note:** this equation simplifies O$_2$ delivery to three parameters: CO, SaO$_2$, and [Hgb]. PaO$_2$ has been omitted due to the *vanishingly small role* it plays with regard to
CaO\textsubscript{2} of blood (remember it contribution is 0.0031*PaO\textsubscript{2}!!).

**Alveolar-to-arterial (A-a) gradient:** This calculation is performed to assess the ability of the patient’s lungs to adequately oxygenate. Many factors influence this, and the calculation is used as a tool to determine the etiology of hypoxemia. To calculate:

1. Place the patient on 100% O\textsubscript{2} (FiO\textsubscript{2} = 1.0) for 20 minutes and obtain a peripheral ABG measurement to determine the Partial Pressure of O\textsubscript{2} (PaO\textsubscript{2}).

2. Calculate the Alveolar Partial Pressure of O\textsubscript{2} (PAO\textsubscript{2}). After breathing 100% oxygen for 20 minutes, the only gases remaining within the alveoli (other than O\textsubscript{2}) oxygen are H\textsubscript{2}O and excreted CO\textsubscript{2} from tissue metabolism (i.e., the N\textsubscript{2} has been “washed out”). Thus, the PAO\textsubscript{2} within the alveoli is calculated as:

\[
[\text{Barometric pressure} – \text{H}_2\text{O partial pressure} – \text{CO}_2 \text{ partial pressure}] * \text{FiO}_2 = \\
[\text{PB} – \text{PH}_2\text{O} – \text{PCO}_2] * \text{FiO}_2 = [760 \text{ torr} – 47 \text{ torr} – 40 \text{ torr}] * \text{FiO}_2 = 673 \text{ torr} * \text{FiO}_2
\]

Since the patient is breathing 100% O\textsubscript{2}, (FiO\textsubscript{2} = 1.0), the equation simplifies to:

\[
\text{PAO}_2 = 673 \text{ torr} * \text{FiO}_2 = 673 \text{ torr} * 1.0 = 673 \text{ torr}
\]

3. Calculate: \textit{A – a gradient} = \text{PAO}_2 – \text{PaO}_2 = 673 \text{ torr} - \frac{\text{PaO}_2}{0.8}

where: 0.8 \sim \text{respiratory quotient (constant)}

**Rule:** the larger the A – a gradient, the more serious the degree of oxygenation compromise; A–a gradient > 400 torr indicates severe respiratory distress resulting from a process interfering with oxygen diffusion capacity; normal A – a gradient \sim 20 – 65 torr.
**Shunt Fraction:** The shunt fraction (normal < 5 %) reflects the portion of CO that traverses the heart from the right to the left without increasing O₂ content (i.e., ~ 5% of pulmonary capillary blood leaves the lung without being oxygenated). In an ideal state, the volume of lung ventilation equals the volume of pulmonary capillary blood flow (Figure 20–15). Alterations in these ventilation–perfusion relationships result from two causes:

- Relative obstruction of alveolar ventilation
- Relative obstruction of pulmonary blood flow

1. **Perfusion greater than ventilation:** A common scenario is that of pulmonary consolidation due to infection or secretions (Figure 20–16). Alveolus (A) receives no ventilation because of bronchiolar obstruction, yet normal pulmonary capillary perfusion continues (i.e., a complete pulmonary A–V shunt exists with respect to that alveolus).
2. **Ventilation greater than perfusion:** Impairment of pulmonary blood flow to the alveolar level occurs in the case of the post-operative lung surgery patient or one who has had a pulmonary embolism (Figure 20–17). Uniform ventilation continues to alveoli (A) and (B), but no blood flow passes alveolus (A). This situation *increases the ventilated physiologic dead space and increases the shunt equation.*
3. **Compensation mechanism:** Figure 20–18 represents the compensatory changes that occur when an alveolus is partially occluded. Blood flow is preferentially shunted to more
efficiently ventilated alveolar units.

**Principle:** Recognize that *at any given time, gradations of each of these situations will exist simultaneously within the lung* (remember that the normal shunt fraction is ~ 5%). Therefore, alterations in either ventilation or perfusion may seriously affect oxygenation.

1. **Decreased lung-to-blood transfer.** Associated factors include:
   - Pulmonary edema
   - ARDS
   - Bronchial secretions
   - Atelectasis
   - Pneumonia
   - Pneumonitis

2. **Decreased perfusion.** Associated factors include:
   - Massive PE
   - Continued micro-pulmonary embolization
   - Post-operative changes

**Calculation of the Shunt Fraction:** In mathematical terms, \( Q = \text{flow} \). \( Qt \) = the total CO in a system, while \( Qs \) = amount of flow through the pulmonary shunt. By definition, \( Qs \) (Figure 20–19) represents that portion of the total CO (\( Qt \)) that does not participate in gas exchange (i.e., the volume of blood shunted past non-ventilated alveoli). Thus:

\[
\text{Shunt Fraction (%) } = \frac{Qs}{Qt} = \frac{C_{CO_2} - C_{aO_2}}{C_{CO_2} - C_{vO_2}} \times 100
\]

where:
- \( C_{CO_2} \) = alveolar capillary \( O_2 \) content (mL/100 mL)
- \( C_{aO_2} \) = arterial \( O_2 \) content (mL/100mL), and
- \( C_{vO_2} \) = pulmonary mixed venous \( O_2 \) content (mL/100mL)

With an \( FiO_2 = 100\% \), the \( O_2 \) oxygen content of capillary blood (\( C_{CO_2} \)), systemic arterial blood (\( C_{aO_2} \)), and pulmonary mixed venous blood (\( C_{vO_2} \)) is calculated as follows:
using the alveolar PO₂ (PAO₂) from the alveolar–arterial gradient calculation

\[ \text{CCO}_2 = [\text{Hgb}] \times 1.39 \times 1 + \text{PAO}_2 \times 0.0031 \]

using the SaO₂ and PaO₂ from arterial blood gas analysis

\[ \text{CaO}_2 = [\text{Hgb}] \times 1.39 \times \text{SaO}_2 + \text{PaO}_2 \times 0.0031 \]

and finally, using the SvO₂ and PvO₂ from a mixed venous blood sample

\[ \text{CVO}_2 = [\text{Hgb}] \times 1.39 \times \text{SvO}_2 + \text{PvO}_2 \times 0.0031 \]

**INDICATIONS FOR INTUBATION**

The decision to intubate a patient to provide mechanical ventilatory support is often a difficult and stress provoking process for clinicians. The primary objective of mechanical ventilation is to decrease the patients’ work of breathing and reverse life-threatening hypoxia.
and hypercapnia. A recent point-prevalence study demonstrated the most common indications for intubation and mechanical ventilation were respiratory failure (66%), coma (15%), acute exacerbation of COPD (13%), and neuromuscular disorders (5%). The following basic checklist may be used to determine the need for respiratory support:

- **Inability to adequately ventilate** (i.e., airway obstruction, severe chest trauma, excessive sedation, neuromuscular disease, paralyzed or fatigued respiratory muscles, etc.)

- **Inability to adequately oxygenate** (i.e., pneumonia, asthma / chronic obstructive pulmonary disease (COPD), pulmonary embolism (PE), pulmonary edema, acute respiratory distress syndrome (ARDS), etc.)

- **Excessive work of breathing** (i.e. severe bronchospasm, airway obstruction, etc.)

- **Airway protection** (i.e., unconsciousness, altered mental status, massive resuscitation, facial or head trauma, etc.)

These basic indications should be used in conjunction with clinical judgment in the final decision regarding mechanical ventilation. The decision to intubate a patient who is clinically decompensating, if made in a timely fashion, may turn an otherwise chaotic intubation into a controlled, elective procedure. Diagnostic factors important in determining impending respiratory collapse of the adult patient are listed in Table 20–7.

| TABLE 20–7. INDICATORS OF IMPENDING RESPIRATORY FAILURE NECESSITATING INTUBATION AND MECHANICAL VENTILATION. |
|-------------------------------------------------|-------------------------------------------------|
| Condition                                      | Normal Range (adults)                          |
| Respiratory impairment                         |                                                 |
| - Tachypnea > 30 breaths / min                 | 10 – 20 breaths / min                          |
| - Dyspnea                                      |                                                 |
**Neurologic impairment**
- Loss of gag reflex
- Altered mental status (i.e., patient is unable to protect airway against aspiration)

**Gas exchange impairment**
- $\text{Paco}_2 > 60 \text{ mmHg}$
- $35 – 45 \text{ mmHg}$
- $\text{PaO}_2 < 70 \text{ mmHg (on 50% mask)}$
- $80 – 100 \text{ mmHg (on room air)}$
- $\text{SaO}_2 < 90$

**SECURING THE AIRWAY**

An essential treatment component of respiratory failure is securing and maintaining a patent airway (See Chapter ____). Briefly, the airway may be kept open by the **chin-lift or jaw-thrust maneuver**; however, these must be done with great care in the trauma patient if there is a suspicion of a cervical spine injury. **Nasopharyngeal or oropharyngeal airways** also assist in keeping the tongue from obstructing the oropharynx. Definitive airway management includes **oral or nasal endotracheal intubation**. Longer-term options include:

- **Tracheostomy** should be considered in patients whom long-term intubation is anticipated and in those patients with severe maxillofacial injuries. This is an **elective procedure** (as opposed to cricothyroidotomy). Improved patient comfort and oral hygiene, ease of secretion removal, and a more secure airway prove tracheostomy is a worthwhile procedure.
• **Crichothyroidotomy** is an emergency procedure when other attempts as securing the airway have failed. Extend the neck (if possible); midline incision with #11 blade; puncture crichothyroid membrane with knife and rotate 90 degrees; keep finger in the crichothyroidotomy site; place 6 cm ETT or cricoid tube into crichothyroidotomy site; confirm placement. Revision to a definitive airway should be performed when possible

• **Complications:** esophageal intubation; pneumothorax; pneumomediastinum; recurrent laryngeal nerve injury; hemorrhage; tracheal stenosis (may be avoided by keeping cuff pressures < 25 mmHg); ET tube dislodgement / self-extubation (life-threatening problem; use restraints liberally); unexplained tidal volume loss (check circuit; check ET tube cuff)

---

**MECHANICAL VENTILATORS**

**Ventilator Classifications**

Although newer ventilator modes combine many of the qualities of these classes, it is conceptually advantageous to discuss the types separately:

**Volume Limited:** A preset volume of air is delivered regardless of the opposing airway pressures; most common class of ventilator used (Note: a pressure limit setting usually allows the modulation of excessive pressure to prevent “volutrauma”).

**Pressure Limited:** These ventilators deliver a volume of air until a preset pressure is
reached; mostly used in neonatal units (Note: not generally used to ventilate adult patients
because changes in airway pressure brought about by changes in lung and chest wall
compliance may result in wide ranges of minute ventilation). This technique is reserved for
patients who fail to respond to traditional volume modes of ventilation.

**High-Frequency Ventilation:** Rapid oscillations of breath (60–1200 cycles / min) used
with or without the bulk delivery of gases to the lung. Several forms of this type of
ventilation exist, including *high-frequency jet ventilation, high-frequency positive pressure
ventilation, high-frequency oscillation,* and *high-frequency percussive ventilation.*

To understand the components of mechanical ventilation that may be manipulated to
bring about physiologic and anatomic changes, the following properties of ventilator
mechanics must be understood:

**Pressure Support (PS):** The ventilator provides a **preset level of positive pressure**
only during the inspiratory phase to augment spontaneous respirations (the positive
pressure is turned off during expiration). This enables the patient to use their respiratory
muscles and decreases the potential for atrophy.

**Positive End-Expiratory Pressure (PEEP):** The ventilator **maintains positive airway
pressure at the end of expiration even though net airflow is zero.** PEEP increases
alveolar ventilation by preventing small airway collapse, thereby improving lung
compliance and maintaining / increasing FRC. PEEP also is often used prophylactically
against postoperative atelectasis and has become a standard maneuver to treat pulmonary
edema. Increasing levels of PEEP are typically used to decrease the FiO2 in an attempt to
limit oxygen toxicity. One disadvantage of PEEP, however, is that it may decrease cardiac
output (CO) by decreasing left ventricular end-diastolic volume (LVEDV) and should be used cautiously in patients at risk for myocardial ischemia.
Controlled Ventilation (CV)
- Mechanical Ventilation
- Rate is fixed by ventilator
- Patient is not allowed to breathe spontaneously in between mechanical breaths.

Assist-Controlled Ventilation (AC)
- Each inspiratory attempt triggers a mechanical breath.

Synchronous Intermittent Mandatory Ventilation (SIMV)
- Patient is allowed to breathe spontaneously in between synchronized mechanical breaths.

Pressure Support Ventilation (PSV) + SIMV
- Patient triggers positive pressure support during inspiration of spontaneous breath in between SIMV mechanical breaths.

Time

Figure 20–20. Representation of different ventilator modes.
Ventilator Modes (Figure 20–20)

**Controlled Ventilation (CV):** The patient receives only ventilator-delivered breaths at a set rate (i.e., the patient cannot initiate a breath on their own). This mode was used in the past on patients who were intentionally paralyzed by drugs due to extreme illness.

**Assist-Controlled Ventilation (AC):** The patient gets a full mechanical tidal volume each time they attempt an inspiratory effort. The respiratory frequency is determined by the patient, although a backup rate is set to ensure minimum minute ventilation.

- **Advantages:** Patients may easily increase their minute ventilation even if they are weak and have a poor inspiratory effort
- **Disadvantage:** Predisposition to hyperventilation if the patient becomes agitated or has an altered respiratory drive because of neurologic injury. Agitation may also lead to “breath stacking”, in which the ventilator delivers a second tidal volume before completing the expiratory phase of the first breath. Fortunately, this is rarely a clinical problem because the patient often feels more comfortable and consequently less agitated because of the decreased work of breathing on AC.

**Synchronous Intermittent Mandatory Ventilation (SIMV):** The ventilator delivers a set number of breaths each minute and allows the patient to supplement ventilation with their own inspiratory efforts between machine breaths. This allows the patient to use their respiratory muscles and prevent atrophy. As the ventilator rate progressively is decreased, the patient assumes more and more the work of breathing. The ventilator
also senses when the patient is taking a spontaneous breath and will not deliver the mandatory tidal volume until after the patient’s own cycle is completed. This was developed to prevent the patient’s working against the ventilator or receiving a double tidal volume (i.e., a mechanical tidal volume superimposed on top of a spontaneous breath). When combined with PS and PEEP, this mode is the most commonly used type of ventilatory support.

*Pressure Support Ventilation (PSV) plus SIMV:* A preset level of positive pressure is turned on only during the inspiratory phase and is turned off during expiration. The patient controls the rate and inspiratory time while augmenting tidal volume and inspiratory flow. The higher the pressure support, the less work the patient expends to take a breath. Thus, PSV is comfortable because the patient has more control of his or her ventilation. PSV serves as an ideal weaning mode because the pressure may be turned down slowly, with changes as small as 1 cm H₂O. This allows the patient to assume the workload of breathing in small increments. PSV is often integrated with SIMV as a backup to ensure minimum minute ventilation.

*Pressure Regulated Volume Control (PRVC):* This mode of ventilation is used in the setting of increased airway pressures. A microprocessor in the ventilator adjusts the pressure as needed to achieve the proper tidal volume. Since the pressure is constantly changing, the net peak airway pressures are less. As the patient’s condition improves, less pressure is necessary to achieve a preset tidal volume. Conversely, as the patient’s
condition worsens, more pressure is delivered to ensure the set tidal volume. This method is one of the preferred modes in critically ill patients who manifest acute lung injury (ALI) or acute respiratory distress syndrome (ARDS) and peak inspiratory pressures of greater than 35 mmHg.

**Continuous Positive Airway Pressure (CPAP):** Positive pressure is maintained throughout inspiration and expiration without mechanical assistance during ventilation. This is equivalent to PS plus PEEP at a constant pressure level. The patient does all the breathing on their own. This mode is often used as a last step before extubation. A CPAP trial may be performed at room air or at FiO₂ of 40%.

**High-Frequency Positive Pressure Ventilation (HFPPV):** The physiologic explanation of HFPPV remains complex; suffice to say this method combines low-volume (sub-anatomic dead space) and rapid-sequence (> 60 breaths / min) ventilation to increase FRC and gas exchange ability. Despite the marked reduction in bulk gas flow rates, oxygenation and CO₂ exchange are still maintained. HFPPV may be ideally suited to treat such conditions as bronchopleural fistulas, to provide ventilation during operations requiring a minimum of lung movement, to have less adverse impact on circulatory parameters, or to allow similar or improved gas exchange at lower peak airway pressures.

**Volumetric Diffusive Respirator (VDR):** This ventilator combines the attributes of pressure control ventilation (set rate with tidal volume dependent on set peak inspiratory pressure
and lung compliance) with HFPPV (to optimize oxygenation). This particular method, also
known as **High-Frequency Percussive Ventilation (HFPV)**, has been used mostly in the
burn population due to its **incredible ability to mobilize pulmonary secretions**
**secondary to inhalational injury**. As with HFPPV, the technical aspects of the VDR are
too detailed to be described here; look for this method to gain more exposure as its utility in
other patient populations is assessed.

**Ventilator Management**

**Ventilator Orders**

Once the decision has been made to place a patient on a ventilator, the patient must be
intubated with an appropriate endotracheal tube (see Chapter 13). The following is a sample
of typical initial ventilator settings for an adult:

- **Mode** (i.e., AC, SIMV)  • **FiO₂** 30 – 100%
- **Rate** 8 – 12 / min  • **Tidal volume** 6–8 mL/kg
- **Pressure support** (level depends on the clinical situation)
- **PEEP** (5 cm H₂O or higher, if needed)

**Ventilator Setting Changes**

Five basic respiratory parameters (**FiO₂** • minute ventilation • **PS** • **PEEP** • I/E ratio)
may be changed to improve ventilation, oxygenation, and / or compliance. Further, attention
to the proper use of these factors will help to prevent ventilator induced lung injury.

1. **FiO₂**: Initially, choose an **FiO₂** that ensures **adequate arterial O₂**
saturation ($\text{SaO}_2 > 90\%$). Increasing the level of PEEP is often a helpful means of decreasing the FiO$_2$ requirement while maintaining adequate oxygenation. Once adequate oxygenation is established, the FiO$_2$ is decreased to avoid oxygen toxicity (avoid FiO$_2$ > 60%)

a. Oxygen toxicity: damage to lungs occurs if the intra-alveolar O$_2$ concentration is > 60% (injury actually occurs after a few hours if FiO$_2 = 1.0$). The mechanism probably involves generation of reactive O$_2$ species which oxidizes the cell membranes. Oxygen toxicity has not been documented if FiO$_2$ is maintained < 60% 

b. Nitric Oxide (NO): NO has been used for its potent pulmonary vascular relaxation properties. Although it will dilate pulmonary blood vessels and improve ventilation / perfusion mismatches, the use of NO has never been shown to improve mortality or shorten mechanical ventilator time in adult patients with severe ARDS

1. Minute volume: Adjust to maintain PCO$_2$ within a normal range (35–45 mmHg). Usually done by increasing tidal volume and / or respiratory rate. Once a tidal volume is chosen, set the respiratory rate (~ 8 – 16 breaths/min) to allow adequate minute ventilation

2. Pressure support: After the patient’s respiratory pattern is established on SIMV, PS may be added initially at a level of 5 – 8 cm H$_2$O. PS may then be turned up to a level that allows the patient to breathe at a comfortable respiratory rate (i.e., < 30 breaths / min). Depending on the
overall stability and mental status of the patient, the number of SIMV backup breaths may be turned down to allow the patient to assume more control of their ventilation. PS rarely needs to be > 35 cm H₂O

3. **PEEP:** “Extrinsic PEEP” supplied by the ventilator is added to decrease FiO₂ while maintaining PaO₂. PEEP ~ 5 cm H₂O is considered physiologic. If the patient continues to deteriorate, PEEP is added in 2-3 cm increments until oxygenation is improved. In acute lung injury, the PEEP where lung compliance is optimized ranges from 12–16 cm H₂O. Serial measurements of compliance are necessary to confirm improvement in pulmonary mechanics.

   • **High-Dose PEEP:** If additional PEEP is required, a PA catheter is essential to monitor CO, SvO₂, PA pressures, and shunt fraction. Static pulmonary compliance and SaO₂ are also followed. At high levels of PEEP, intrathoracic pressure increases to a point that venous return is impaired. Thus, LVEDV decreases which leads to a drop in CO (this point defines the maximum level of PEEP; this level may vary considerably from patient to patient or for the same patient over time.

4. **Inspiratory-expiratory (I/E) ratio:** The normal I/E ratio is 1:2 or 1:3. In patients with obstructive respiratory diseases, longer expiratory times allow full exhalation and guard against “breath stacking” (auto PEEP) or “intrinsic PEEP”. By reversing the I/E ratio (i.e., 2:1 or 3:1), a.k.a., “Inverse Ratio Ventilation”, distension and progressive
recruitment of alveoli occurs which leads to improved oxygenation.

There may also be a favorable redistribution of pulmonary tissue edema fluid. This technique is used in patients with severe consolidating lung diseases in an attempt to improve oxygenation by increasing mean airway pressure. This beneficial effect on oxygenation is lost if “breath stacking” occurs (i.e. breath stacking or intrinsic PEEP is detrimental to gas exchange)

**PEEP Side Effects**

- Falsely elevated PAOP (PCWP)
- Decreased cardiac output
- Barotrauma / Volutrauma (leading to pneumothorax, alveolar rupture, etc.)
  - high peak airway pressures (> 45 cm H₂O)

**Ventilator Weaning**

Prior to weaning the patient from the ventilator, the patient’s pulmonary mechanics and oxygenation should be assessed (Table 20–8). Additionally, the major problem that required the patient be placed on mechanical ventilation must have been corrected.

**Pulmonary Mechanics:** These data provide useful information regarding a patient’s ability to perform the work of respiration. Routine pulmonary mechanics consist of:

- Vital capacity
- Tidal volume
- Spontaneous respiratory rate
- Lung compliance
- Inspiratory force: the maximum negative pressure that may be exerted against a
completely closed airway (i.e., a function of respiratory muscle strength). An inspiratory force **between 0 and –25 cm H\(_2\)O** would indicate that the patient is **incapable of generating adequate inspiratory effort** for successful extubation.

**Weaning Modes:** Modern respirators are designed to facilitate weaning. Once the preceding criteria have been met, a ventilator mode appropriate to the clinical situation may be selected. SIMV and PSV are considered weaning modes because the patient is allowed to assume more of the workload of breathing as mechanical support is reduced.

**Order of Weaning:** The following steps are taken routinely to wean the patient from the ventilator:

1. Sequentially **reduce FiO\(_2\)** by 10% until 50% is reached; use **pulse oximetry** (SaO\(_2\)) to assist in weaning because it reduces the number of ABGs needed. FiO\(_2\) may be **steadily decreased** as long as SaO\(_2\) > 90 – 92% or PaO\(_2\) > 70 mmHg.

2. Sequentially **reduce the IMV rate to a level of 4-8 breaths per minute**. Add **PS to maintain adequate minute volume**; ABGs as well as capnography are used to monitor for hypercarbia.

3. Sequentially **reduce PEEP** in 2 to 3 cm H\(_2\)O increments while maintaining SaO\(_2\) > 90 % until a level of 5 cm H\(_2\)O is achieved.

4. Sequentially **reduce PS** by 2 to 3 cm H\(_2\)O increments while maintaining minute ventilation (goal: 5 - 10 cm H\(_2\)O); monitor respiratory rate, work of breathing, and PCO\(_2\).

**Essential Tips in Ventilator Management**

- Avoid changing more than one ventilator parameter at a time
• \( PO_2 < 60 \text{ mmHg} \) or \( SaO_2 < 90\% \) = **return to previous levels** of respiratory support

• \( PO_2 60-70 \text{ mmHg} \) or \( SaO_2 \sim 90 - 92 \% \) = **hold at the current level** of respiratory support

• \( PO_2 > 70 \text{ mmHg} \) or \( SaO_2 > 93 \% \) = **continue systematic weaning**

---

**TABLE 20–8. CRITERIA FOR EXTUBATION FROM MECHANICAL VENTILATION.**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pulmonary mechanics</strong></td>
<td></td>
</tr>
<tr>
<td>Vital capacity</td>
<td>&gt; 10–15 mL/kg</td>
</tr>
<tr>
<td>Resting minute ventilation</td>
<td>&gt; 10 L/min</td>
</tr>
<tr>
<td>(tidal volume * rate)</td>
<td></td>
</tr>
<tr>
<td>Spontaneous respiratory rate</td>
<td>&lt; 33 breaths/min</td>
</tr>
<tr>
<td>Lung compliance</td>
<td>&gt; 100 mL/cm H(_2)O</td>
</tr>
<tr>
<td>(Negative) Inspiratory force (NIF)</td>
<td>&gt; -25 cm H(_2)O</td>
</tr>
<tr>
<td><strong>Oxygenation</strong></td>
<td></td>
</tr>
<tr>
<td>A-a gradient</td>
<td>&lt; 300–500 mm Hg</td>
</tr>
<tr>
<td>Shunt fraction</td>
<td>&lt; 15%</td>
</tr>
<tr>
<td>( PO_2 ) (on 40% F(_i)O(_2))</td>
<td>&gt; 70 mm Hg</td>
</tr>
<tr>
<td>( PCO_2 )</td>
<td>&lt; 45 mm Hg</td>
</tr>
</tbody>
</table>

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**Checklist for Extubation**

• **Correction of primary problem** that triggered intubation and mechanical ventilation (i.e., successfully treated pneumonia, returned hemodynamic stability, etc.)

• **Level of consciousness stable or improved**

• **Stable vital signs**

• Pulmonary mechanics and oxygenation meet acceptable criteria (Table 20 – 8)

**Extubation Trials**
Once weaning has achieved minimal ventilatory settings, various trials of mechanical support may be attempted while the patient is still intubated:

1. **CPAP trials** (with 5 cm H₂O positive pressure) remain the most commonly used method; CPAP trial with an FiO₂ at 21% (room air) or 40% should result in a PaO₂ of > 50 mmHg or 70 mmHg, respectively.

2. **T-piece trials** which provide only humidified air with no pressure, are also occasionally used, but may be unnecessarily stressful to the patient due to the lack of pressure support or CPAP.

**CPAP trials** are thought to be more physiologic because positive pressure partially counterbalances the added resistance encountered by breathing through a long, narrow endotracheal (ET) tube. These trials may vary in duration from 30 min to several hours and are used primarily as the last test prior to extubation. Patients without COPD usually are tested with an extubation trial with: IMV rate 4 / FiO₂ 30% / PEEP 5 cm H₂O. The ventilator remains at the bedside in case respiratory support needs to be restarted.

**Extubation**

A patient who is able to maintain a PO₂ > 70 mmHg, a PCO₂ < 45 mmHg, and a respiratory rate < 25 breaths / min for 1–2 h on a T-piece or CPAP trial is ready for extubation.

1. **Disconnect** the ET tube from the ventilator or T piece

2. **Suction** the ET tube and oral pharynx

3. Have the patient take a deep breath while you deflate the ET tube balloon

4. As the patient expires forcefully, remove the tube and suction any secretions
5. Apply a nasal cannula with O₂ flow at 2–4 L/min

6. Check post-extubation ABG to ensure adequate ventilation and oxygenation

Nutrition

The nutritional support of the critically ill patient is crucial to patient survival.

Restoring the patient to an anabolic state will hasten recovery and avoid complications. Protocols for nutritional support are covered in Chapters 11 and 12.

Remember the following two rules:

1. The “2-day” rule applies to most patients. If you do not think the critically ill patient will take nutrition for 2 days because of postoperative ileus, intubation, etc., be sure to make arrangements for nutritional support.

2. “If the gut works, use it.” That is, do not use parenteral nutrition if the GI tract is functioning properly. Enteral nutrition (i.e., oral, NG tube, jejunostomy tube) should be used in all patients with a functioning intestinal tract. Enteral feeding is reviewed in Chapter 11.

COMPLICATIONS IN CRITICAL CARE

Acute Respiratory Distress Syndrome (ARDS)

ARDS, also called “wet lung” or “shock lung,” is defined as respiratory failure associated with acute pulmonary injury and is manifested by marked respiratory distress
and hypoxia. Pulmonary capillaries become more permeable, resulting in pulmonary edema in the setting of low to normal PA pressures. Clinical Criteria ARDS include:

- PaO₂: FiO₂ ratio of < 200
- Diffuse bilateral infiltrates on chest x-ray
- Pulmonary Artery Occlusion Pressure (i.e., PAOP or “wedge pressure”) < 18 mmHg
- Lack of an alternative clinical explanation for pulmonary findings

Etiology

The causes of ARDS are multifactorial and include anything that could activate the Systemic Inflammatory Response Syndrome (SIRS). These include, but are not limited to:

- Severe head injury
- Severe trauma with prolonged hypotension
- Massive fluid resuscitation
- Sepsis
- Severe Pancreatitis
- Severe Burns
- Severe chest trauma / pulmonary contusion
- Aspiration, Chemical Pneumonitis, or Inhalational Injury

Three primary mechanisms of the final common pathway of SIRS-induced lung injury include:

1. Increased pulmonary vascular resistance: Pulmonary edema is caused by a dramatic increase in pulmonary capillary hydrostatic pressure. This increase forces fluid across the capillary membrane and results first in interstitial and then alveolar edema.

2. Permeability edema: Circulating toxic substances (interleukin – 1, TNF - α, etc.) within the bloodstream may cause the pulmonary capillary membrane to become leaky
and allow extravasation of protein into the interstitial space. This extravasation increases the interstitial hydrostatic pressure and eventually results in injury to the alveolar membrane. At this point, fluid and protein migrate into the alveolar space and directly impede oxygen exchange. Several factors have been implicated as mediators to this increased capillary–alveolar permeability, including prostaglandins and oxygen radicals.

3. Injury to the alveolar membrane: Conditions promoting direct toxicity to the alveolar membrane include: • Smoke inhalation • High doses of oxygen (> 60% FiO₂) • Aspiration

Treatment

Primary efforts are directed at treating the underlying condition while providing sufficient pulmonary support. Currently, no specific therapy is available for ARDS except prevention.

1. Aggressive ventilatory support: Use PEEP to maintain the FiO₂ < 0.6 while maintaining a SaO₂ > 93%. Use the SaO₂, volume status, and level of PEEP to guide ventilatory management. Although some may advocate increased levels of PEEP to minimize intrapulmonary shunting (Qs/Qt), doing so may necessitate increased intravascular volume and inotropic support of the heart. Most clinicians use SaO₂ as a guide to altering PEEP levels, rather than following the specific shunt fraction.

2. Aggressive fluid administration: Maintain CO and peripheral perfusion; use of colloid versus crystalloid remains controversial. Many clinicians recommend the use of crystalloid (NS, lactated Ringer’s) and packed red blood cells (PRBCs) to maintain the Hct > 24% in most patients (Hct > 30% in patients with known ischemic cardiac disease).
3. **Aggressive monitoring:** Use a PA catheter to guide fluid administration (by following filling pressures), and observe the effect of added PEEP on CO. Inotropic agents may be indicated if CO remains low despite adequate filling pressures. Use an arterial line to obtain arterial blood for frequent ABG determinations.

4. **Pulmonary toilet:** To manage secretions

5. **Chest x-rays:** To monitor lung improvement

6. **Watch for associated DIC, other complications related to SIRS activation**

7. **Steroids are not indicated** in the treatment of ARDS.

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**Upper Gastrointestinal Hemorrhage**

Critically ill patients are at increased risk for gastrointestinal hemorrhage secondary to stress-induced mucosal ulcerations. The development of such ulcerations is a serious, yet preventable, complication. The pathophysiology is related to **diminished blood flow to the viscera** during stress situations leading to **alterations in the mucosal barrier** to the effects of gastric acid. **Head injury** (Cushing’s ulcers), **mechanical ventilation**, **non-steroidal anti-inflammatory (NSAID)** use, **shock / trauma / burns** (Curling’s ulcers), **coagulopathy**, or a **history of peptic ulcer disease** or **portal hypertension** are a few of the risk factors.

**Prophylaxis**

- **Enteral feedings** (when tolerated): **method of choice** to protect the gastric lining
- **Routine cardiovascular support** of visceral perfusion
- **Routine use of acid suppression methods.** Meta-analysis has demonstrated that...
prophylaxis using \textbf{H}_2\text{-blockers} (i.e., ranitidine, famotidine, etc.) \textbf{significantly reduces} stress-induced GI ulcerations and clinically significant hemorrhage. Proton-pump inhibitors (i.e., lansoprazole, omeprazole, etc.) may be used for \textbf{refractory bleeding} or in patients that have \textbf{side-effects to histamine blockade} (i.e., thrombocytopenia)

- \textbf{Antacid administration} (i.e., Maalox, Mylanta, sucralfate, etc.); in patients with renal failure, use aluminum hydroxide to avoid magnesium-containing antacids and resultant aluminum toxicity

\textit{Treatment of Ulceration}

\textbf{Early endoscopy} is indicated in upper GI bleeding

1. \textbf{A visible (bleeding) vessel} warrants \textbf{endoscopic or operative intervention}

2. \textbf{Diffuse gastritis} is best treated initially with \textbf{aggressive acid suppression} and \textbf{empiric treatment for} \textit{H. pylori}

3. \textbf{Persistent bleeding} from gastritis may warrant \textbf{total gastrectomy}

\textbf{Shock}

Shock is defined as \textbf{hypotension leading to the inadequate delivery or utilization of} \textit{O}_2 \textbf{and nutrients at the tissue level to meet metabolic needs}. If uncorrected, shock leads to \textbf{cellular dysfunction} that then produces \textbf{organ failure} and ultimately death to the organism. The causes of shock are multifactorial; however, \textbf{treatment of shock is always directed at correcting the underlying problem}. Endogenous compensatory mechanisms directed at reversing hypotension and shock include the \textbf{release of catecholamines},
cortisol, and activation of the renin / angiotensin / aldosterone axis.

The morbidity and mortality of shock are related to the etiology but probably more to the degree and time of circulatory compromise. With the causes identified and corrected, the organism is resuscitated to restore tissue perfusion and reverse the sequelae of shock. The most common etiologies of shock include:

1. **Hypovolemic Shock:** Caused by inadequate circulating blood volume (at least 20% loss) caused by severe fluid depletion (dehydration) or acute hemorrhage.

   Compensation includes low CO, low PAOP, and elevated PVR as a result of reflex vasoconstriction (Table 20 – 9 lists physiologic changes associated with hypovolemic shock).

   **Treatment:**  
   1. **Control the source** of intravascular volume loss
      2. **Rapidly replete intravascular volume** with:
         a. PRBCs
         b. isotonic crystalloid fluids (normal saline, lactated Ringer’s): due to equilibration with the interstitial space, the 3:1 (crystalloid:volume loss) rule must be considered
         c. colloid solutions (albumin, hetastarch, etc.)

2. **Cardiogenic Shock:** Caused by primary heart failure either from intrinsic cardiac abnormalities (i.e., severe valvular disease, acute myocardial infarction, coronary ischemia, arrhythmias, etc.) or extrinsic processes (i.e., tension pneumothorax, pericardial tamponade, PE, etc.). Compensation includes low CO, high PAOP, and elevated PVR.
**Treatment:** Directed at improving cardiac performance

1. **Optimize preload** (filling pressures)
2. **Decrease afterload** (vasodilation with nitroglycerin, nitroprusside, etc.)
3. **Improve cardiac contractility** (dobutamine)
4. **Resolve extrinsic processes** (if necessary)
5. **Consider mechanical support** (intra-aortic balloon counterpulsation) if other measures fail

3. **Septic Shock:** Caused by intravascular fluid loss (i.e., extravascular fluid sequestration – “third spacing”) due to systemic infection or some other initiator of the SIRS response. Compensation includes hyperdynamic CO (until late stages), low PAOP, and low PVR.

**Treatment:**

1. **Treat the cause of sepsis** (“source control – parenteral antibiotics, abscess drainage, etc.) or SIRS activation
2. **Administer fluids to replace intravascular losses** (usually 1 – 2 times the circulating blood volume)
3. **Increase PVR** (pressors such as norepinephrine, phenylephrine, ect.)
4. **Provide inotropic support to the heart** (as needed); may need a PA catheter to guide fluids and pressor administration

4. **Neurogenic Shock:** Caused by loss of sympathetic vascular tone (i.e., due to a high
thoracic or cervical cord injury) producing an increase in vascular capacitance.

Compensation includes low CO, low PAOP, and low PVR.

_Treatment:_ 1. **Optimize filling pressures** by intravenous fluid administration

2. **Increase PVR** (pressors such as norepinephrine, phenylephrine, etc.)

3. **Keep fluids and ambient room temperature warm** because these patients lose the ability to thermoregulate

All forms of shock require therapies to improve the delivery of O₂ to the tissue level. Optimization of oxygen carrying capacity of blood (SaO₂, [Hgb]) and providing adequate CO are the mainstays of treatment. Recent studies suggest [Hgb] ~ 7 – 9 gm/dL are satisfactory and were associated with decreased mortality in critically ill patients unless they had known ischemic heart disease (where [Hgb] ~ 10 – 12 gm/dL was optimal). In this cooperative study, previous transfusion practices of maintaining all patients at a goal [Hgb] ~ 9 – 10 gm/dL were associated with higher 30-day mortality unless the patient had a concurrent acute myocardial infarction or unstable angina pectoris.

**Acute Renal Failure (ARF)**

ARF is the sudden development of renal insufficiency that results in retention of nitrogenous wastes (BUN, creatinine) and variable effects on fluid balance. Diverse opinion persists as to the magnitude of BUN or creatinine elevations required to diagnose ARF (see Chapter 6). In general, oliguria / anuria and progressive azotemia are the final
common pathway of a number of pathologic processes that constitute ARF which occurs in approximately 5% of ICU admissions. Although multiple etiologies exist, ARF is usually divided into prerenal, renal, and postrenal causes (see Chapter 6). Despite the etiology, once ARF is recognized, the primary goal is to treat the underlying cause.

Critically ill patients often require radiographic imaging with intravenous contrast materials. A recent study in well-hydrated patients with baseline creatinine > 2-times normal suggests that the use of N-acetylcysteine 24 hrs before and after the administration of contrast will likely prevent contrast-induced nephropathy. With this new therapy, the incidence of contrast-induced nephropathy should decline over time.

Many critically ill patients are unable to receive oral nutrition or fluid and are therefore particularly susceptible to fluid and electrolyte derangements. Further, sepsis and other initiators of the SIRS response lead to massive fluid shifts that may result in severe hypovolemia. The following data provide information regarding fluid status:

- **Urine output:** (best and simplest) minimum of 0.5 cc/kg/hr for adults; 1.0 cc/kg/hr for children; 2.0 cc/kg/hr for infants

- **Daily weight:** Changes result from the net loss or gain of fluid

- **PA catheter data**

Acute tubular necrosis (ATN) from multiple etiologies (i.e., nephrotoxic medications, ischemia, hypotension, etc.), intravascular volume depletion, and congestive heart failure are the most common causes of renal failure in the ICU patient. The following describes the general approach to ARF in an ICU patient:
Physical Examination

1. Vital signs: Hypotension with or without associated tachycardia and orthostatic blood pressure changes may be a sign of hypovolemia (prerenal)

2. Mucous membranes: Dry mucous membranes indicate overall fluid depletion

3. Lungs: Fluid overload often manifests auscultated crackles (pulmonary edema)

4. Abdomen: Low urine output may result from post-renal obstruction (palpable bladder); distended abdomen may indicate an ileus with associated bowel fluid sequestration

5. Extremities: Fluid overload may be evident (i.e., peripheral, dependent edema)

Diagnostic Studies

1. Laboratory results (serum and urine electrolytes / urine eosinophils and myoglobin)

2. Bladder catheterization: If a catheter is in place, irrigate gently to confirm drainage

3. Radiographic studies: Renal ultrasonography helps evaluate for possible postrenal obstruction. Avoid intravenous contrast studies if possible (i.e., contrast nephropathy)

Therapeutic trials: Certain therapies may be used to differentiate pre-renal from renal or post-renal azotemia (see Chapter 6). After obstruction has been ruled out, failure to respond to these measures likely indicates an intrinsic renal cause of ARF:

- Fluid challenge (rapid 1000 mL of IV 0.9 % saline infusion)
- Furosemide 80 mg IV push (furosemide has little effect in ATN)
- Intravenous inotropic support

Management: As a general approach, daily fluid intake and output as well as body weight should be closely reviewed. Follow serum electrolytes, particularly potassium,
closely (remove potassium from the IV fluids immediately) in cases of ARF to prevent accumulation of deadly potassium levels). Otherwise, management involves correction of the underlying abnormality and supportive care.

**Prerenal**

1. Optimize hemodynamic status to maximize CO and renal perfusion
2. Replete intravenous fluids (use PRBCs in anemic patients, otherwise, use isotonic fluids or albumin)
3. With optimal fluid status, check urine output; if still suboptimal, use inotropic support (dopamine 2 – 5 mg/kg/min) to dilate renal blood vessels; a PA catheter is usually needed to further monitor the patient and adjust pharmacologic support

**Renal**

1. Optimize fluid status and cardiovascular function (see above)
2. Furosemide challenge (20–40 mg IV); if no response, try mannitol 12.5–25 g IV
3. Restrict fluids and salt (particularly potassium) if these become troublesome
4. Treat ARF-induced metabolic acidosis with sodium bicarbonate if pH < 7.25
5. Hemodialysis if necessary (see below)

**Postrenal**

1. Place and check bladder catheter for patency; replace immediately if questionable
2. Obtain a urologic consultation; prostatic obstruction may be easily corrected with catheterization; decompression of the upper urinary tracts may require stents or percutaneous drainage

Fortunately, most causes of ARF are reversible (see Chapter 6). Hemodialysis,
however, may be necessary for the following reasons:

- **Fluid overload** causing respiratory compromise
- Severe **metabolic acidosis**
- Severe **uremia**
- Severe **electrolyte disturbances**
- Severe **uremia**
- Toxic **accumulation of drugs**

**Abdominal Compartment Syndrome (ACS)**

ACS is a recently recognized entity caused by **massive intra-peritoneal bowel edema and fluid sequestration**, or from significant **retroperitoneal hemorrhage** causing a mass-effect. The **increased intra-abdominal pressure** directly **decreases visceral perfusion**, most notably to the kidneys, which ultimately leads to **organ dysfunction** and **respiratory compromise**.

**Diagnosis:** Assessment of **bladder pressures (> 30 mmHg)** via a foley catheter; a **tense, distended abdomen** with the appropriate **clinical history; increasing peak airway pressures**.

**Treatment:** **Early decompressive celiotomy** remains the treatment of choice (allows restoration of intra-abdominal pressures and hemodynamics). The abdominal fascia may be closed when the edema and organ dysfunction resolves.

**Acalculous Cholecystitis**

Cholecystitis in the absence of gall stones is not uncommon in the ICU patient. Although the precise etiology remains unknown, it is probably related to **diminished blood**
flow to the gall bladder and to bacterial overgrowth.

**Diagnosis:** Presenting signs are similar to those in healthy patients with cholecystitis and include right upper quadrant pain, fever, leukocytosis, and elevated liver chemistries (especially bilirubin or alkaline phosphatase). The work-up should also include a gall bladder ultrasound. A HIDA scan may be added if the ultrasound is non-diagnostic (nonvisualization of the gallbladder is highly suggestive of acalculous cholecystitis).

**Treatment:** Prevention of this potentially fatal condition is key. Maintain a high-index of suspicion for the development in the critically ill patient. Treatment is surgical (cholecystectomy), and should be done as early as possible. If the patient is too critically ill to tolerate an operation, a percutaneous cholecystostomy tube could be placed to drain the gall bladder and allow a “cooling off” period. Interval cholecystectomy should then be performed when possible.

**Acute Adrenal Insufficiency**

Acute adrenal insufficiency is an increasingly common, yet difficult to diagnose condition in critically ill patients. The symptoms range from subtle organ dysfunction to life-threatening, pressor-refractory circulatory shock. Categories include:

1. **Chronic primary adrenal insufficiency:** Due to loss of adrenal function from surgery (adrenalectomy) or from metabolic disturbances; patients will take chronic steroid replacement; home steroid medication should be continued without interruption; may need hydrocortisone 25 – 75 mg IV QD x 2 – 3 days for mild to moderate surgical stress; for severe stress, up to 300 mg IV QD may be necessary.
2. **Chronic secondary adrenal insufficiency:** Due to ACTH insufficiency likely secondary to exogenous glucocorticoid administration (steroid replacement therapy) or due to pituitary failure (post-surgical); patients will take chronic steroid replacement; home medication should be continued without interruption; may need hydrocortisone 25 – 75 mg IV QD x 2 – 3 days for mild to moderate surgical stress; for severe stress, up to 300 mg IV QD may be necessary.

3. **Acute adrenal crisis:** Due to acute direct (adrenal hemorrhage) or indirect (pituitary hemorrhage) adrenal loss; also due to chronic primary / secondary adrenal insufficiency patients who are acutely stressed (i.e., infection, trauma, burns, etc.)

    **Diagnosis:** Evidence continues to accumulate on the incidence of acute adrenal crisis in the ICU patient population, thus, the precise definition and treatment regimens remain controversial. Random serum cortisol levels of $<25\text{ µg/dL}$ or $>45\text{ µg/dL}$ have been suggested to predict poor outcome and increased mortality in some reports while in others the patients’ ability to respond to ACTH stimulation has been studied. Patients who fail to increase stimulated cortisol level $\sim 9\text{ µg/dL}$ above baseline ("non-responders") or patients who have a stimulated cortisol $<18\text{ µg/dL}$ appear to have a worse prognosis. As with all these patients, reasonable source control must have been achieved and other etiologies have been ruled out to account for the cardiovascular collapse. Empiric steroid replacement should be started on:

    1. Patients with positive random cortisol levels (see above)
    2. Patients who are “non-responders”
3. Patients with pressor-unresponsive shock and no known premorbid adrenal insufficiency (provided they do not have an infectious source):

Treatment:

1. Send blood for assessment of cortisol level
2. Start Hydrocortisone 50 mg IV Q8 hr
3. Continue the steroid replacement for approximately 2-3 days (with a rapid taper):
   a. If the random cortisol level is < 25 µg/dL, or
   b. If the random cortisol level is > 25 µg/dL and clinical improvement was demonstrated with treatment

1. Increase the glucocorticoid dose (300 – 400 µg QD) if the patient remains “pressor non-responsive”
2. Wean the steroids quickly as tolerated (once hemodynamic stability is achieved)

Disseminated Intravascular Coagulation (DIC)

DIC is a complex management problem that often presents in the critically ill patient. This clinical syndrome may accompany a number of disease states, including shock syndromes, sepsis, malignancy, and some obstetric conditions. As with many of the pathologic conditions that accompany major illness (i.e., ARDS, ARF, etc.), the successful treatment of DIC depends on treating the underlying condition.
**Diagnosis:** DIC is usually contemplated in critically ill patients who develop **coagulopathy** and **thrombocytopenia.** The following list details other laboratory findings that are caused by the effect of plasmin on fibrinogen (i.e., results in **increased levels of fibrin monomers** and **feedback stimulation of the fibrinolytic system):**

- **Low fibrinogen level**
- **Elevated Fibrin Split Products (FSP) level** (a.k.a. Fibrin Degradation Products)
- **Elevated PT / PTT**
- **Characteristic microangiopathic RBC morphologies**

**Treatment:** (controversial)

1. The most important element of therapy is to **identify and treat the underlying cause** (i.e., sepsis source control, adequate resuscitation of associated shock, etc.)

2. If **thrombosis occurs** (i.e., DVT, PE), begin **heparin therapy** (see Chapter 22)

3. Administer **FFP or cryoprecipitate** to **replenish fibrinogen stores**

4. If the patient is **bleeding severely,** despite replacement therapy with FFP/cryo and platelets, begin **antifibrinolytic therapy** with **epsilon–aminocaproic acid (Amicar).**

In general, if there is no improvement after 12 h, therapy should be stopped.

**Infections**

**Line Sepsis:** Indwelling catheters not only provide a convenient means of infusing fluids and medications, but also act as a portal of entry for bacteria. With the widespread use of indwelling intravenous catheters (i.e., central venous lines), the diagnosis of **infection**
from the catheter itself must be considered when evaluating a febrile patient in the ICU. As a general rule, fever in a person with a central line should be attributed to the line until proven otherwise. The most common mechanism of line sepsis is entry of skin flora along the catheter tract. The use of clear polyurethane dressings left in place for prolonged periods has been associated with increased risk of infection and should be avoided. Some institutions have a policy of routine line changes over a guidewire every 3 – 4 d. Little objective data support this practice; in fact, recent CDC guidelines suggest this practice is associated with an increased rate of complications. Prevention of line sepsis is best accomplished by meticulous aseptic technique during line placement (including fully gowing, gloving, and draping) and meticulous care of the line once in place.

**Diagnosis:** A presumed episode of line sepsis is treated by determining whether the line is actually responsible. The catheter may be changed over a guidewire with the intracutaneous segment and tip sent for culture. The site in question should be abandoned and a new site for IV access chosen if the catheter culture becomes positive.

Erythema at the entry site is highly suggestive of the catheter being the source. In this case, the catheter should be removed and a new site utilized for IV access. In the absence of florid sepsis, or if placement of a new line would jeopardize the ability to obtain vascular access, quantitative cultures of blood from a peripheral site and the line in question may be obtained and treatment be based on the results of these cultures.

**Treatment:** Short-term central venous catheters suspected of being infected are best treated by removing the line and sending it for culture to confirm the source. Empiric antimicrobial therapy may be started in the interim. If the colony count from the catheter is
> 15, the result is interpreted as probable catheter infection. Antibiotics should be continued (~ 5 days) if the patient is demonstrating signs of systemic infection.

**Ventilator-Associated Pneumonia (VAP):** Defined as a clinical pneumonia that develops after 48 h of mechanical ventilation, VAP occurs in approximately 25 % of intubated, ICU patients with an overall mortality from 20 – 50 %. The strongest risk factor for pneumonia in the ICU population is mechanical ventilation (6 – 15 fold increase); however, age > 70 years, chronic lung disease, naso-enteric tubes, altered mental status, chest trauma / surgery, and frequent transportation of the patient have all been shown to be associated with an increased incidence of VAP.

**Diagnosis:** Includes a positive airway culture (preferably a bronchoalveolar lavage (BAL) specimen with quantitative cultures showing > 10⁴ CFU/mL) plus 3 of the 4 following:

- New, persistent, or progressive pulmonary infiltrate (by chest x-ray)
- Purulent tracheobronchial secretions
- Fever
- Leukocytosis

**Treatment:** Empiric therapy should be instituted when VAP is suspected and customized according to the institutions antibiogram for the particular ICU. Directed therapy may then be formulated when bacterial culture and sensitivity data becomes available. In general, antibiotic therapy should be continued for 10 – 14 days. Repeat BAL with cultures should be performed if the patient fails standard antibiotic therapy.
Pulmonary Embolism (PE)

PE remains a **major cause of death** in the United States (~ 150,000 deaths annually).

**Deep venous thrombosis (DVT)** is known to be **responsible for a majority of PE** in hospitalized patients. It is estimated that about **90% of all PE originate in the femoral–iliac–pelvic veins**. DVT is caused by the classical causes of thromboses (Virchow’s triad): vessel injury, hypercoagulability, and blood stasis.

**Prevention of DVT:** Prevention is especially important in **“high-risk” patients** (those with malignancy, obesity, previous history, age >40 years, extensive abdominal/pelvic surgery, long bone and/or pelvic fractures, and prolonged immobilization). For patients undergoing surgery, **prevention should be initiated in the operating room. Intermittent compression stockings** and the selected use of **heparinoids** have greatly reduced the incidence of DVT in the post-operative patient. **NOTE:** prophylaxis against DVT is **effective only when started preoperatively** for those patients undergoing surgery.

**Physical Methods:** Includes **leg elevation, intermittent compression devices**, and **early postoperative ambulation (most important).**

**Pharmacologic Methods**

- **Heparin** 5000 U SQ q12h. Check the platelet count intermittently (~ every 3 days) because of risk of heparin-induced thrombocytopenia (HIT)

- **Coumadin** for chronic therapy

- **Low molecular weight heparins** (i.e. Enoxaparin, dalteparin, etc.) is now the drug of choice in our institution for high-risk patients, despite the high cost of therapy

*Diagnosis of Pulmonary Embolus*
• Maintain a **high index of suspicion** in high-risk patients

• **Signs and symptoms:** *None is diagnostic*, but may include dyspnea, tachypnea, tachycardia, chest pain (usually pleuritic), and $PO_2 < 80$ mmHg (compared with baseline)

• Routine **chest x-ray** may show localized volume loss or **Hampton’s hump** due to pulmonary infarction

• **Spiral CT scan:** This scan is helpful in identifying proximal pulmonary emboli and is **considered by our institution to be the test of choice**

• **Pulmonary angiogram:** The “gold standard” but it is quickly losing favor to spiral CT scan as the imaging technology improves

• **Nuclear V/Q scan:** Is insensitive and therefore often not helpful. A **normal scan** effectively rules out PE, and a **positive scan is sufficient evidence to treat the patient.**

An indeterminate scan in a symptomatic patient with a high index of suspicion necessitates angiography

**Treatment**

1. **Support oxygenation.** Monitor ABGs and support as necessary
   (intubation may be necessary)

2. Use **intravenous heparin;** prevents clot propagation, decreases inflammation, and allows intrinsic fibrinolysis to lyse the clot

   a. **Bolus with 80–100 U/kg** and start an **intravenous drip at 10–15 U/kg/h.**
      Adjust the drip to keep the PTT at 2–2.5 _control values._ The half-life of heparin is 1.5 h, so **check the PTT at 3–6 h after adjusting the rate** of heparin administration
b. **Monitor the platelet count** because some patients may manifest

“heparin-induced thrombocytopenia”

c. Start **oral warfarin** (Coumadin) by day 3 of heparin therapy, to maintain a therapeutic ratio (see Chapter 22)

1. In cases of **massive embolus, thrombolytic therapy** (TPA) may be used in the absence of contraindications

2. **Open embolectomy**, using cardiopulmonary bypass, has been effective in rare cases of massive PE

3. In patients who cannot undergo systemic anticoagulation (those with recent surgery, stroke, GI bleeding, etc.) or patients with recurrent emboli despite adequate therapy, vena caval interruption may be indicated using an percutaneous intracaval filter

<p>| TABLE 20 – 9. PHYSIOLOGIC CHANGES ASSOCIATED WITH DEGREE OF HEMORRHAGIC SHOCK. |
|-----------------------------|------------------|------------------|------------------|------------------|
|                            | <strong>CLASS I</strong>      | <strong>CLASS II</strong>     | <strong>CLASS III</strong>    | <strong>CLASS IV</strong>     |
| Blood loss (%)             | &lt; 15             | 15 – 30          | 30 – 40          | &gt; 40             |
| Blood loss (ml)            | &lt; 750            | 750 – 1500       | 1500 – 2000      | &gt; 2000           |
| Mental status              | --               | Anxiety          | Confusion        | Lethargy         |
| Heart rate                 | --               | Mild ↑           | Moderate ↑↑      | Severe ↑↑↑        |
| Blood pressure             | --               | --               | --               | --               |
|   systolic                 | --               | --               | $\downarrow$     | $\downarrow$     |
|   diastolic                | --               | ↑↑               | ↑↑               | ↑↑               |
| Respiratory rate           | --               | Mild ↑           | Moderate ↑↑      | Severe ↑↑↑        |
|  (breaths/min)             | --               | --               | --               | --               |</p>
<table>
<thead>
<tr>
<th>Urine output</th>
<th>--</th>
<th>Mild ↓</th>
<th>Oliguria</th>
<th>Anuria</th>
</tr>
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* = based on 70 kg adult; -- = No significant change; ↑ = increased; ↓ = decreased.