Discuss the etiology of Volkmann’s ischemic contracture

Ischemic contracture is a manifestation of ischemia induced from compartment syndrome. Compartment syndrome usually results from crush injuries, which in turn elevate the compartment pressure above 40 mm Hg – higher than the closing pressure of supplying blood vessels. Approximately 75% of cases of compartment syndrome are associated with fractures: tibial fracture has the highest association; supracondylar humeral, humeral shaft, and forearm fractures can also lead to compartment syndrome. Once perfusion to a compartment is impaired, resultant disruption of skeletal myocytes occurs: membrane integrity is compromised, leading to cytolysis with the release of active enzymes and proteins into the interstitial space. This increase in oncotic pressure further draws plasma from the vasculature, thus increasing pressure and resisting perfusion into the myofascial compartment. End manifestations of compartment syndrome are myoglobinuria, hyperkalemia, renal failure, irreversible muscle injury, and death.

The two most common locations of ischemic muscle contracture are the intrinsic muscles of the hand and the long digital flexors of the anterior forearm. Examination of the intrinsic tightness of these muscle groups can be assessed by extending the MCP joints and flexing the PIP joints. In particular, a Volkmann’s Contracture is a flexion contracture of the forearm flexors; it is best assessed by passively extending the digits with the wrist in full extension.

Treatment of Volkmann’s Contracture is as follows:

1. **Acute Stage:** Urgent Fasciotomy
2. **Mild Chronic Cases localized to the FDP muscle group:** Dynamic splinting, casting, and physical therapy.
3. **Long-standing cases:** Surgical release of flexor muscles from their origin.

Discuss the diagnosis of alveolar hypoventilation.

**Definitions**

- **ALVEOLAR VENTILATION**—the amount of fresh inspired air (non-dead space gas) that enters the alveoli per minute and is therefore available for gas exchange.
- **DEAD SPACE**—anatomic dead space is the volume of the conducting airways which is approximately $150 \text{ ml}$; physiologic dead space is defined as the volume of the lungs that does not participate in gas exchange; physiologic dead space is equal to anatomic dead space in normal lungs; physiologic dead space is greater than the anatomic dead space in diseased lungs with ventilation-perfusion inequalities.
- **MINUTE VENTILATION** = tidal volume $\times$ breaths/minute
- **ALVEOLAR VENTILATION** = (tidal volume $-$ dead space) $\times$ breaths/minute
- **HYPOVENTILATION**—conditions in which alveolar ventilation is abnormally low in relation to oxygen uptake or carbon dioxide output; causes arterial hypoxemia and hypercarbia; a feature of alveolar hypoventilation is that hypoxemia can often be eliminated by providing the patient with supplemental oxygen.

**Causes of Hypoventilation**

- Depression of respiratory center by drugs—i.e. morphine, barbiturates, benzodiazepines
- Diseases of the brain stem—i.e. encephalitis, stroke, intracranial bleed
- Diseases of nerves to respiratory muscles—i.e. Guillain-Barre, phrenic nerve injury in cardiac surgery
- Diseases of the myoneural junction—i.e. myasthenia gravis
- Diseases of the respiratory muscles—i.e. progressive muscular dystrophy
- Thoracic cage abnormalities
- Upper airway obstruction—i.e. thymoma, substernal goiter
- Hypoventilation associated with extreme obesity—Pickwickian syndrome
- Primary alveolar hypoventilation—hypoventilation without apparent cause in a lean patient (rare)

In ALL of these conditions, the lungs are NORMAL, which clearly distinguishes alveolar hypoventilation from those diseases in which the carbon dioxide retention is associated with chronic lung disease which ultimately causes ventilation-perfusion inequality. To differentiate between disorders of hypoventilation and disorders of ventilation-perfusion mismatch you must calculate the **A-a gradient** (the difference in $pO_2$ between Alveolar gas and arterial blood). An A-a gradient of less than 20 mm Hg is considered normal.

Alveolar gas equation:

$$ pO_2 \text{ (in inspired gas)} = pO_2 \text{ (in arterial blood)} \times RQ $$

The $pO_2$ in inspired gas ($pIO_2$) = fraction of inspired oxygen $\times$ (barometric pressure $-$ partial pressure of water vapor) = $FIO_2 \times 713$

**Example:**

Fraction of inspired oxygen = 21%  
$Alveolar \ pO_2 = 0.21 \ (713) - 40/0.8 = 100 \ mm \ Hg$

$pCO_2$ on ABG = 40 mm Hg  
$pO_2$ on ABG = 90 mm Hg  
$Alveolar$-arterial gradient = 100 mm Hg-90 mm Hg = 10 mm Hg

Respiratory Quotient $=$ 0.8

- With normal gas exchange the alveolar $pO_2$ is the major determinant of the arterial $pO_2$ and the A-a gradient will be normal; therefore a normal A-a gradient indicates a generalized hypoventilation disorder, i.e. central hypoventilation or neuromuscular disorder
- An abnormal or elevated A-a gradient indicates a ventilation perfusion mismatch (cardiopulmonary disorder) or a systemic imbalance between oxygen delivery and oxygen demand (identified by decreased mixed venous $pO_2$)

**References:**

Describe the treatment of non-healing infected toe

Wound-care patients are often best managed by a team approach, with critical input from a variety of medical and surgical subspecialties and a dedicated wound-care nursing staff. Once the cause of a patient's wound has been correctly identified and the other medical or surgical aspects of disease treatment have been initiated, wound care can be focused on healing.

Management of wound drainage is generally treated with daily dressing changes until limb edema is controlled and then by choice of a hydrocolloid dressing if there is minimal drainage, a polyurethane hydrofoam dressing if drainage is moderate, and calcium alginate dressings if drainage remains copious. Dry wounds or those with significant eschars may require hydration; the choice then is an amorphous hydrogel covered with a simple gauze dressing. These basic wound-dressing classes can be combined or supplemented with absorbent gauze products as needed.

Tissue necrosis requires debridement to facilitate healing because necrotic debris creates localized hypoxia and acidosis, inhibiting the development of granulation tissue and epithelialization. Necrotic tissue also harbors high levels of bacteria that can inhibit healing, even if not causing frank cellulitis.

Once the cause of the wound is known and confounding factors have been treated (e.g., infection resolved, limb revascularized, edema controlled, necrotic tissue debrided, and so forth), reasonable outcomes can be expected from wound-care interventions. Recently, there have been a series of well-controlled, prospective, randomized clinical trials of new agents for venous insufficiency ulcers and diabetic foot ulcers. These trials have allowed wound care outcomes to be studied, and surprisingly consistent results have been reported. In all the trials reviewed, for either diabetic foot ulcers or venous insufficiency ulcers, a critical cutoff of more than 0.1 cm of healing/week was associated with healing, regardless of the therapeutic modality used. Likewise, less than 0.05 cm of healing/week was predictive of nonhealing, without respect to the therapy employed. Thus, the author emphasizes the importance of regular measurements of wounds so that appropriate therapeutic decisions can be made. If the rate of healing is greater than 0.1 cm/week by week 2 to 4 after a therapy is instituted, the same regimen can be continued. If the rate of healing is less than 0.05 cm/week by week 2 to 4 after a therapy is instituted, reassessment is needed, and a change in approach should be made.
Management of a solitary pulmonary nodule

A solitary pulmonary nodule (SPN) is a well circumscribed mass in the lung parenchyma, less than 3 cm in diameter in an asymptomatic patient. 33% of these nodules are cancer- the percentage increases to 50% in patients over 50 years old. A definitive diagnosis must be made i.e. biopsy unless:

1) The mass is unchanged for at least 2 years (must have serial chest x-rays), the lesion has a benign pattern of calcifications (concentric or heavy calcifications, suggesting hamartoma), 3) the mass is clearly caused by TB, 4) the operative risk is prohibitively high, and 5) those patients in who small cell carcinoma is suspected.

Diagnosis:
Fine-needle aspiration is recommended only if the surgeon is looking for a reason not to operate (high-risk patient, or suspicion of small cell carcinoma), since a positive FNA requires definitive therapy and a non-diagnostic FNA requires a tissue biopsy. Sensitivity approaches 100%

Wedge resection or lobectomy will give a tissue diagnosis. If the frozen section reveals cancer, a lobectomy or pneumonectomy and mediastinal lymph node dissection is required for definitive therapy.

Differential diagnosis
1. primary lung carcinoma
2. metastasis to the lung
3. granulomatous disease (histoplasmosis or tuberculosis)
4. benign lung tumor (hamartoma)
5. AV fistula

Chest x-ray features that signify malignancy
1. large size (> 4cm)
2. indistinct or lobulated margins
3. growth in size since last CXR
4. doubling time between 35 and 280 days

Chest x-ray features that suggest a benign process
1. small size (<1 cm)
2. distinct margins
3. calcifications (especially if concentric or heavily calcified)
4. satellite lesions
5. no growth in > 1 year (doubling time >465 days), or very rapid growth (doubling time <35 days)
Treatment of acute fem-pop graft occlusions

Treatment of the patient’s lower-extremity symptoms should be chosen on the basis of the severity of the symptoms. Invasive intervention for asymptomatic disease is not appropriate.

Early heparin anticoagulation may limit the propagation of thrombus and prevent clinical deterioration, although there is little objective data on which to base this practice. Retrospective studies suggest that heparin decreases the risk of recurrent embolization in patients with embolic occlusions. Immediate surgical revascularization is indicated in the profoundly ischemic limb. Catheter embolectomy is also usually preferred for emboli to a non-atherosclerotic limb. While open surgical procedures remain the gold standard in the treatment of peripheral arterial occlusion, thrombolytic agents have been employed as an alternative to primary surgical revascularization in patients with acute limb ischemia. Systemic administration of thrombolytic agents, while effective for small coronary artery clots, fails to achieve dissolution of the large peripheral arterial thrombi. Catheter-directed administration of the agents directly into the occlusive thrombus is the only means of effecting early recanalization. Prior to 1999, urokinase was the sole agent used in North America for peripheral arterial indications, but the loss of the agent from the marketplace forced clinicians to turn to alternate agents, specifically alteplase and reteplase. Interest in the use of platelet glycoprotein inhibitors and mechanical thrombectomy devices also rose, coincident with the loss of urokinase from the marketplace. A number of reports from individual centers and three large prospective studies, which compared intra-arterial thrombolysis to surgical intervention, suggest that thrombolytic therapy may be an appropriate initial treatment of ALI, provided that the limb is not immediately or irreversibly threatened. Using this approach, the underlying lesions can be further defined by angiography, and the percutaneous or surgical revascularization


Ouriel. Comparison of surgical and thrombolytic treatment of peripheral arterial disease, Reviews in cardiovascular medicine vol 3, suppl. 2 2002

The effect of dopamine in hypertrophic subaortic stenosis

Idiopathic hypertrophic subaortic stenosis (IHSS) is a form of hypertrophic cardiomyopathy characterized by asymmetric left ventricular hypertrophy [usually preferential hypertrophy of the interventricular septum, or asymmetric septal hypertrophyASH)] and a dynamic left ventricular outflow tract pressure gradient related to a narrowing of the subaortic area from mid-systolic apposition of the anterior mitral valve leaflet against the hypertrophied interventricular septum systolic anterior motion (SAM) of the mitral valve. Nevertheless, the main pathophysiologic derangement in this disease is diastolic cardiac dysfunction (reduced filling) as a result of the stiff, non-compliant hypertrophied left ventricle. Only about one quarter of patients with this disease have a true left ventricular outflow tract pressure gradient. Such a gradient can be induced or exacerbated by physiologic maneuvers that either (1) increase myocardial contractility (exercise; inotropes such as dopamine, dobutamine, digitalis glycosides, or isoproterenol) or (2) decrease the volume of the left ventricle and promote left ventricular outflow tract obstruction (Valsalva, sudden standing, nitroglycerin, amyl nitrite, tachycardia). Conversely, maneuvers which increase the size and volume of the left ventricle by (1) improving venous return to the heart (squatting, volume expansion, passive leg raising) or by (2) elevating arterial pressure and left ventricular afterload (phenylephrine, sustained isometric handgrip) decrease left ventricular outflow tract obstruction and improve hemodynamics. Therefore, inotropes such as dopamine are contraindicated in patients with IHSS, as they decrease cardiac filling and promote SAM and left ventricular outflow tract obstruction in these patients.

References: Fauci et al. Harrison’s Principles of Internal Medicine, 14th Ed. pp 1330-1333.
Describe the findings on a PA catheter in a patient during an acute MI

Pulmonary artery (Swan-Ganz) catheters can be used to transduce central venous pressure and pulmonary artery pressures. In the setting of myocardial infarction, Swan-Ganz catheters can reveal 1) myocardial irritability and 2) heart failure.

Myocardial irritability may become evident in the form of a ventricular arrhythmia, which can be triggered by physical stimulation of the ischemic heart by the catheter. The pulmonary artery catheter can induce a right bundle branch block. Thus, in patients with left bundle branch block, care should be taken to use a Swan-Ganz catheter with a pacing device as the combination of right and left bundle branch blocks can lead to complete AV block.

Heart failure can be divided into left-sided and right-sided failure. Each is associated with characteristic central venous pressure (CVP) and pulmonary capillary wedge pressure (PCWP) findings. Left-sided failure is associated with pulmonary hypertension and progressive-right-sided heart failure. In right-sided failure, the central venous pressure is elevated and the pulmonary capillary wedge pressure can be normal.

Right-sided heart failure: High CVP

Left-sided heart failure: High PCWP

Marino PL, The ICU Book p.164
Describe the effect of PEEP during mechanical ventilation

PEEP—elevation of transpulmonary pressures at the end of expiration—has two components: *applied* PEEP and *intrinsic (auto, occult)* PEEP.

Applied PEEP prevents alveoli from derecruiting. This improves the V/Q matching and gas exchange, protects alveoli from the risk of shear stress that is an effect of repeated opening and closing, and prevents surfactant breakdown in collapsing alveoli and thus improves lung compliance. Detrimental effects of PEEP include increasing the risk of stretch injury to the lung (optimal PEEP includes a balance of recruiting alveoli in diseased lung regions without overdistending already recruited alveoli in healthy regions), and it also increases intrathoracic pressure which, in turn, can compromise the cardiac filling pressure in susceptible patients. Application of PEEP is optimal when the pulmonary pathologic process is generalized—stiff lungs, as in ARDS—rather when the disease is localized—as in pneumonia. In localized processes, PEEP can overdistend alveoli in normal lung regions and redirect blood back to diseased areas. It should be noted that the tendency for PEEP ventilation to reduce cardiac output is not a function of the PEEP level, but is rather a function of the PEEP-induced increase in mean intrathoracic pressure. Appropriate PEEP is adjusted via gas exchange goals: PEEP is applied to attain a minimally acceptable arterial Po2 at the lowest FiO2. Oxygen delivery is to be maximized; an increase in arterial oxygenation is to be augmented to a greater degree than cardiac output depressed. Other clinical misuses of PEEP include cases where reducing lung edema is desired (PEEP can promote water accumulation in lungs via impeding lymphatic drainage); and when control of mediastinal bleeding is the goal (PEEP is transmitted across the walls of blood vessels and thus does not reduce transmural pressure).

Intrinsic PEEP occurs when alveolar emptying is incomplete during expiration. Here, airflow is present at the end of expiration and thus creates a pressure drop from the alveoli to the proximal airways at end-expiration. It is dependent on minute ventilation and expiratory time fraction. As minute ventilation increases (increased i/e ratio), expiratory time fraction decreases and the potential for the development of intrinsic PEEP increases. In addition obstructive airway disease promotes auto-PEEP; in patients with COPD and asthma, occult PEEP is probably universal during volume-cycled mechanical ventilation. Auto-PEEP has similar effects on cardiac output as extrinsic-PEEP; there is an increased risk of alveolar rupture with auto-PEEP; and occult-PEEP can result in an increased work of breathing—it places the ventilatory cycle on a flatter, less compliant portion of the pressure-volume curve.

When setting the level of extrinsic PEEP, it should be enough to counterbalance the force causing small airways to collapse but should not exceed the level of intrinsic PEEP. This is accomplished by increasing the level of extrinsic PEEP to the point where it just begins to cause elevations in peak inspiratory pressure.

Describe the proper placement and confirmation of an ET tube

Preparing for endotracheal intubation: All intubation equipment should be prechecked and within reach before attempting to intubate the trachea, including laryngoscope with working light, endotracheal tube (generally females 7.0 mm and males 8.0 mm with stylet and cuff), and a functioning suction unit with rigid tip. These should be pre-checked and within reach before attempting to intubate the trachea. A stylet may or may not be necessary.

Endotracheal intubation: Proper positioning is essential. In-line manual cervical immobilization should be used in the cervical trauma patient. Otherwise, the head should be extended and the neck flexed. Several layers of folded towels may be placed under the occiput to achieve this position. The mouth is opened with the right hand. The left hand inserts the laryngoscope on the right side of the tongue, then sweeps the tongue up and to the left. The tip of the curved blade fits into the vallecula, while the straight blade fits under the epiglottis. Upward traction on the laryngoscope exposes the vocal cords and the endotracheal tube is inserted through the cords under direct vision. If available, an assistant may provide digital pressure over the cricoid cartilage to prevent aspiration of gastric contents during laryngoscopy. --Limit intubation attempts to 30 seconds and ventilate adequately between attempts. Optimize head position, blade type or size, and tube size before the next attempt at intubation.

Checking for tube placement: Once the endotracheal tube has been inserted between the vocal cords, inflate the cuff, remove the styles, attach positive pressure ventilation source to the endotracheal tube and check for bilateral breath sounds. Check for esophageal intubation by listening for gurgling over the epigastrium. If breath sounds are heard on one side, slowly withdraw the tube until bilateral breath sounds are present. Proper positioning is usually achieved with an endotracheal tube taped at the 21 cm mark at the teeth in females and 23 cm at the teeth in males. Don't forget to obtain a CXR to check for tube placement.

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<table>
<thead>
<tr>
<th>Table 6.2. Technique for Orotracheal Intubation</th>
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<tbody>
<tr>
<td>Place patient on 100% oxygen</td>
</tr>
<tr>
<td>Assemble drugs and equipment</td>
</tr>
<tr>
<td>Clean and suction mouth</td>
</tr>
<tr>
<td>Place patient in proper position</td>
</tr>
<tr>
<td>Check endotracheal tube size and cuff</td>
</tr>
<tr>
<td>Insert stylet and lubricate tip of tube</td>
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<tr>
<td>Administer drugs (see Table 6-3)</td>
</tr>
<tr>
<td>Displace tongue</td>
</tr>
<tr>
<td>Lift jaw and tongue away at 45-degree angle</td>
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<tr>
<td>to visualize cords</td>
</tr>
<tr>
<td>Use free hand to guide tube through cords</td>
</tr>
<tr>
<td>Remove stylet</td>
</tr>
<tr>
<td>Inflate cuff</td>
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<tr>
<td>Listen for breath sounds</td>
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<tr>
<td>Secure tube</td>
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<tr>
<td>Connect patient to ventilator</td>
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<tr>
<td>Mnemonic for intubations: SOAP ME</td>
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<tr>
<td>Suction</td>
</tr>
<tr>
<td>Oxygen</td>
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<tr>
<td>Airway equipment</td>
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<tr>
<td>Pharmacology (have drugs ready)</td>
</tr>
<tr>
<td>Monitoring</td>
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<tr>
<td>Equipment</td>
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<tr>
<th>Table 6-3 Suggested Pharmacologic Agents for Intubation</th>
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<tbody>
<tr>
<td>Oxygen</td>
</tr>
<tr>
<td>Thiopental (Pentothal), 1.5 mg/kg in an adult or</td>
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<tr>
<td>Methohexital (Brevisal), 1 mg/kg</td>
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<tr>
<td>Midazolam (Versed), 10 mg total to adult</td>
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<tr>
<td>Succinylcholine: 1.5 mg/kg in adult</td>
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<tr>
<td>2 mg/kg in child &lt;10 yr old</td>
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<tr>
<td>3 mg/kg in newborn</td>
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<table>
<thead>
<tr>
<th>Table 6-4 Choice of Endotracheal Tube According to Patient Size</th>
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<tbody>
<tr>
<td>Age</td>
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<tr>
<td>-------</td>
</tr>
<tr>
<td>Premature</td>
</tr>
<tr>
<td>Newborn</td>
</tr>
<tr>
<td>6 mo</td>
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<tr>
<td>18 mo</td>
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<tr>
<td>3 yr</td>
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<tr>
<td>5 yr</td>
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<tr>
<td>6 yr</td>
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<tr>
<td>8 yr</td>
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<tr>
<td>12 yr</td>
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<tr>
<td>16 yr (small adult)</td>
</tr>
<tr>
<td>Most women</td>
</tr>
<tr>
<td>Most men</td>
</tr>
<tr>
<td>Large adult</td>
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</tbody>
</table>

*Size measured in internal diameter.

*Size in French No. R. 1.14 times outside diameter.
Confirmation of ET tube placement:
- During intubation, direct visualization of the endotracheal tube passing through the vocal cords into the trachea constitutes firm evidence of correct tube placement, but should be verified with additional techniques.
- Verification of endotracheal tube placement should be completed in all intubated patients, and reconfirmation of endotracheal tube position should be done in all patients when their clinical status changes, or when there is any concern about proper tube placement.
- Standard physical examination methods, such as auscultation of lungs and epigastrium, visualization of chest movement, and fogging in the tube, are not sufficiently reliable to exclude esophageal intubation in all situations.
- Verification techniques include capnometry, esophageal detection devices, and revisualization with direct laryngoscopy.

**End-tidal CO\textsubscript{2} detection**, either qualitative, quantitative, or continuous, is the most accurate and easily available method to monitor correct endotracheal tube position in patients who have adequate tissue perfusion.
- Pulse oximetry and esophageal detector devices are not as reliable as end-tidal CO\textsubscript{2} determinations in patients who have adequate tissue perfusion.
- For patients in cardiac arrest, and for those with markedly decreased perfusion, when end-tidal CO\textsubscript{2} does not confirm tracheal intubation, other methods of confirmation, such as direct visualization, should be done.
- X-ray confirmation when possible

References:
1) Mastery of Surgery. Nyhus, 3\textsuperscript{rd} edition; Chapter 6.
2) American College of Emergency Physicians. [http://www.acep.org/1,4923,0.html](http://www.acep.org/1,4923,0.html)
4) Virtual Naval Hospital. [http://www.vnh.org/GMO/ClinicalSection/89AirwayManagement1.html](http://www.vnh.org/GMO/ClinicalSection/89AirwayManagement1.html)
**Anatomy of Subclavian Artery**

Artery supplying the upper extremity continues as single trunk from its commencement down to the elbow. However, different portions of it have received different names according to the regions through which they pass. The part of the vessel extending from origin to the outer border of the first rib is called subclavian. Beyond this point to the lower border of the axilla is called axillary. From the lower margin of the axillary space to the bend of the elbow is called brachial.

Right subclavian artery arises from innominate artery behind the right sternoclavicular articulation. Left subclavian artery arises from the arch of the aorta.

Each subclavian artery is divided into three parts. First portion extends from origin of the vessel to the medial border of the scalene muscle. Second portion lies behind the muscle. And third portion extends from the lateral margin of the muscle to the outer border of the first rib, where it becomes the axillary artery.

Branches of the subclavian artery are vertebral, thyrocervical, internal mammary and costocervical. On the right side, costocervical trunk arises from the second portion of the vessel, while on the left side, all four branches arise from the first portion of the vessel. In general, there is a free interval of 1.25-2.5cm between commencement of the artery and the origin of the nearest branch.
Discuss the contraindications to pneumonectomy

Lung cancer continues to be the leading cause of cancer deaths in the United States. In patients with resectable non-small cell lung cancer, surgical resection is the treatment of choice. An accurate preoperative general and pulmonary-specific evaluation is essential as postoperative complications and morbidity can be significant and may direct surgical planning. Resectability is determined by anatomic features and the ability to withstand the surgery and the loss of the resected lung.

The degree of pulmonary dysfunction after operation is related directly to the type of procedure and the preoperative pulmonary function. Thoracic procedures, particularly lung resections and pneumonectomy, cause severe depression of respiratory function (TLC, FRC, and the surface area for gas exchange is reduced). Increased age, smoking, obesity and obstructive pulmonary disease all diminish the FRC (functional reserve capacity) and ERV (expiratory reserve volume). The additional effect of a post-operative reduction in FRC and ERV results in airway closure and atelectasis. Therefore, it is essential to perform objective pulmonary function tests on patients with age over 45 years old, dyspnea with moderate exertion, change in exercise tolerance, or smoking history greater than 10 pack years.

In restrictive disease, the volume capacity of the lungs and/or chest cage is limited. With restrictive disease, the patient has limited capacity to expand the lungs but no difficulty emptying the lungs. In obstructive disease, the lungs can expand but emptying is impeded by narrowing of the airways.

Obstructive disease is the more common and difficult problem in thoracic surgery. Patients with normal lungs do not require muscular effort during inspiration, except with extreme exercise. In patients with obstructive lung disease, active effort to push air out of the lungs, results in airway collapse and air-trapping. In these patients, the postoperative decrease in lung volume results in closure of a significant portion of the airways in the dependent portion of the lungs, resulting in micro- and gross atelectasis.

*Obstructive pulmonary disease is the most important risk factor* in surgical patients and the degree of expiratory obstruction is related directly to the risk of postoperative complications. Restrictive pulmonary disease is usually tolerated more easily.

The measurement of spirometric indexes (FEV\(_1\)) and diffusing capacity of the lung for carbon monoxide (DLCO) should be performed first. If FEV\(_1\) and DLCO are > 60% of predicted, patients are at low risk for complications and can undergo pulmonary resection, including pneumonectomy, without further testing. However, if FEV\(_1\) and DLCO are < 60% of predicted, further evaluation by means of a quantitative lung scan is required. In patients with abnormalities in FEV\(_1\) or DLCO identified preoperatively, it is essential to estimate the likely post resection pulmonary reserve. The amount of lung function lost in lung cancer resection can be estimated by using either a nuclear perfusion scan or the number of segments removed. If lung scan reveals a predicted postoperative (ppo) values for FEV\(_1\) and DLCO of > 40%, the patient can undergo lung resection. A *ppo FEV\(_1\)* or DLCO < 40% indicates an increased risk for perioperative complications, including death, from lung cancer resection. If the ppo FEV\(_1\) and ppo DLCO are < 40%, exercise testing is necessary. Formal cardiopulmonary exercise testing is a sophisticated physiologic testing technique that includes recording the exercise ECG, heart rate response to exercise, minute ventilation, and oxygen uptake per minute, and allows calculation of maximal oxygen consumption (VO\(_2\)max). Risk for perioperative complications can generally be stratified by VO\(_2\)max. Patients with preoperative VO\(_2\)max > 20 mL/kg/min are not at increased risk of complications or death; VO\(_2\)max< 15 mL/kg/min indicates an increased risk of perioperative complications; and patients with VO\(_2\)max < 10 mL/kg/min have a very high risk for postoperative complications. If this reveals a maximal oxygen uptake (VO\(_2\)max) of > 15 mL/kg, surgery can be undertaken. If the VO\(_2\)max < 15 mL/kg, pneumonectomy is not an option.


Beckles MA, Spiro SG: The physiologic evaluation of patients with lung cancer being considered for resectional surgery, *Chest*. 2003 Jan; 123(1s):105s-114s
Describe the anatomy of the IMA

The inferior mesenteric artery supplies blood to the hindgut, from the splenic flexure to the superior portion of the rectum. It originates from the abdominal aorta and courses retroperitoneally to the left. It normally gives off several branches: the left colic artery, sigmoid arteries (usually 3-4), and the superior rectal artery. The colic and sigmoid arteries anastomose distally to form part of the marginal artery. There are certain variations that are seen. Occasionally, the left colic can provide blood more proximally to the transverse colon. In some individuals a direct connection between the middle colic artery and left colic artery is present—this anastomosis is known as the Arc of Riolan or the meandering artery of Gonzalez.

Sources

Describe the mechanism of NO smooth muscle relaxation

Endothelium derived Nitric Oxide (NO) is released in response to acetylcholine stimulation, hypoxia, endotoxin, cellular injury or mechanic shear stress from circulating blood. Induction of vascular smooth muscle relaxation by NO requires the activation of soluble guanylate cyclase and an increase in cytosolic cyclic guanosine monophosphate (cGMP) within myocytes. Methylene blue inhibits guanylate cyclase, prevents cGMP production and inhibits vascular relaxation. cGMP is also present in platelets and can be activated by NO. Increased cGMP within platelets is associated with reduced adhesion and aggregation. Therefore, NO induces vasodilation as well as platelet deactivation.

\[
\text{NO} \not\rightarrow \text{cGMP} \not\rightarrow \text{relaxation.}
\]


Discuss systemic vascular resistance during hypovolemic shock

Hypovolemic shock can result from many causes including hemorrhage, protracted vomiting, diarrhea, fluid sequestration, inappropriate diuresis, dehydration, and anaphylaxis. Beyond a 10% blood loss neuroendocrine response to hypovolemia is initiated. Blood flow to the adrenal medulla is increased resulting in a quick delivery of catecholamines to the systemic circulation. Thus, by adrenergic receptors in the periphery, systemic vascular resistance and therefore blood pressure increases. Splanchnic circulation is reduced resulting in a redistribution of available blood volume. At a blood loss of 30-50% redistribution fails and adrenergic effects cannot overcome the hypovolemia resulting in a drop in blood pressure. Once hypotension occurs, blood is redistributed to favor the brain over the heart and kidneys. At greater than 50% volume loss all compensatory mechanisms are overcome.
Describe the signs of cardiac tamponade on transthoracic echo

Accumulation of fluid in the pericardium with a resultant increase in pericardial pressure and impairment of ventricular filling results in cardiac tamponade. The hallmarks of cardiac tamponade are increased intracardiac pressure and the resulting impaired ventricular filling and depressed cardiac output. In tamponade, ventricular filling is impaired throughout diastole; by comparison, early diastolic filling is relatively normal with pericardial constriction. Invasive hemodynamic assessment will reveal equalization of right and left atrial as well as right and left ventricular diastolic pressures. Echocardiographic evidence of tamponade physiology includes a compressed/small right ventricular chamber with late-diastolic invagination of the right atrial and right ventricular free wall on two-dimensional imaging (see Chapter 43) . Because of the frequent coexistence of tachycardia, the latter is generally best appreciated with high-temporal resolution M-mode echocardiography. Localized right atrial, left atrial, and left ventricular diastolic collapse may also be seen and are particularly relevant for loculated effusions such as those following trauma and cardiac surgery. Pseudoprolapse of the mitral valve may be seen because of the compressed left ventricular cavity In addition to diastolic invagination, M-mode echocardiography may demonstrate exaggerated inspiratory septal motion and variation in the duration of aortic valve opening. Finally, Doppler echocardiography may be used to assess transtricuspid and transmitral flow profiles and demonstrate the exaggerated peak E wave response seen in tamponade. It is important to note that many of these typical echocardiographic findings may be absent in patients with significant pulmonary artery hypertension

Source: Goldman: Cecil Textbook of Medicine, 21st ed., Copyright © 2000 W. B. Saunders Company pgs349-350

Cardiac tamponade occurs when pericardial effusion of sufficient magnitude has accumulated to result in equilibration of intrapericardial and passively determined intracardiac pressures. Immediately after mechanical systole the ventricle begins to relax, with a greater degree of active relaxation attributed to the left ventricle compared with the right. This results in disproportionate or favored filling of the left ventricle with a transient elevation in intrapericardial pressure. This results in early diastolic collapse of the highly compliant right ventricular outflow tract. The atrial corollary of this phenomenon is exaggerated atrial emptying. Because of elevated intrapericardial pressures, filling of the right ventricle is impeded in early diastole (a manifestation of which is early diastolic right ventricular collapse) and occurs to an exaggerated degree with atrial systole. This results in a delayed and exaggerated contraction of the right atrium with actual invagination of the atrial wall in late diastole. Echocardiographically exaggerated respiratory variation can be documented by examining the left ventricular and right ventricular outflow tract flows in systole and noting exaggerated phasic variation in velocities and time velocity ventricles. Similarly, in a hemodynamically significant effusion, ventricular filling is impeded.

Source: Braunwald: Heart Disease: A Textbook of Cardiovascular Medicine, 6th ed., Copyright © 2001 W. B. Saunders Company pgs.203-204

Cardiac tamponade--changes of effusion plus:

1. RV compression
   a. RV diameters decreased, especially outflow tract (7 mm)
   b. Early diastolic collapse of right ventricle
2. RA free wall indentation (collapse) during late diastole and/or isovolumic contraction lasting at least one third of the cardiac cycle
3. LA free wall indentation (cases with fluid behind left atrium)
4. LV free wall paradoxic motion
5. SVC and IVC congestion (unless volume depletion); IVC >2.2 cm with <50% inspiratory collapse.
6. Exaggerated inspiratory effects (especially with pulsus paradoxus with reciprocal right-heart/left-heart effects during inspiration and expiration)
   a. Right ventricle expands
   b. Interventricular septum shifts to left
   c. Left ventricle compressed
d. Mitral
   (1) d-e amplitude decreased
   (2) e-f slope decreased or rounded
   (3) Open time * decreased; delayed mitral opening

e. Aortic valve: opening decreased*; premature closure *
f. Echographic stroke volume decreased

7. Notch in RV epicardium during isovolumic contraction
8. Course oscillations of LV posterior wall
9. Pseudohypertrophy: apparent wall thickening due to compression

C. Doppler studies: with any degree of tamponade
   1. Major changes on first beats during inspiration and expiration
   2. Generally reduced flows/stroke volumes
   3. Exaggerated inspiratory augmentation of right-sided and decrease of left-sided flows
   4. Respiratory variation in superior and inferior vena caval flow velocities marked in tamponade, less increased with effusion; double-peaked superior vena cava systolic wave. Decreased expiratory diastolic SVC flow.
   5. Hepatic vein expiratory effect:
      a. Marked atrial reversal (AR wave)
      b. Marked decrease or reversal of diastolic forward flow
      c. (Occasional) systolic flow reversal
   6. (Transesophageal echocardiograms): expiratory increase in pulmonary vein diastolic forward flow
   7. Marked inspiratory decrease in LV ejection time; increased RV ejection time
   8. Marked inspiratory increase in LV isovolumic relaxation time; decreased RV isovolumic relaxation time
   9. Hepatic vein velocity difference between systole and atrial reversal <0 cm/sec

SVC and IVC=superior and inferior venae cavae; LA=left atrial; LV=left ventricular; RA=right atrial; RV=right ventricular.

SUBSTRATE OF NITRIC OXIDE SYNTHASE

A chance observation 2 decades ago led to the discovery that the endothelium plays a key role in vasodilation. Many different stimuli act on the endothelial cells to produce endothelium-derived relaxing factor (EDRF), a substance now known to be nitric oxide. Since the discovery of nitric oxide, it has become increasingly recognized that not only is nitric oxide important as a mediator of cardiovascular physiology, but it has been shown to play an important role in the pulmonary, gastrointestinal, immunologic, and central nervous systems.

Nitric Oxide Synthesis: Nitric Oxide is synthesized in endothelial cells from the amino acid L-arginine in a reaction catalyzed by nitric oxide synthase (NOS). Three isoforms of NOS have been identified: NOS 1 is found in the central nervous system; NOS 2 is found in the macrophages and other immune cells; and NOS 3 is found in endothelial cells.

NOS 1 and NOS 3 are constitutive forms present in the brain and vascular system and are activated by agents that increase intracellular Ca$^{2+}$ concentration, including the vasodilators acetylcholine and bradykinin. The NOS in immune cells (macrophages) is an inducible enzyme not constitutively present, and this form is not induced or activated by Ca$^{2+}$ but is induced by cytokines.

IN THE CARDIOVASCULAR SYSTEM:
- The NO that is formed in the endothelium diffuses to smooth muscle cells where it activates soluble guanylyl cyclase, producing cGMP, which in turn mediates the relaxation of vascular smooth muscle.
- Nitric oxide production by NOS 3 by the vascular endothelium is responsible for the vasodilator tone that is essential for the regulation of blood pressure.
- Nitric oxide is also involved in vascular remodeling and angiogenesis, and may be involved in the pathogenesis of atherosclerosis.
- Utility of nitroglycerin and nitroprusside and other known vasodilators that have been in clinical use as antihypertensives and anti-anginal drugs relates to their conversion to nitric oxide in vivo.
- Nitric oxide also contributes to the control of platelet aggregation and the regulation of cardiac contractility.
- NO is involved in the mediation of hypotension in septic shock.

IN THE PULMONARY SYSTEM:
- NO may be useful in the treatment of pulmonary hypertension.

IN THE NERVOUS SYSTEM:
- NO is a neurotransmitter that underpins several functions, including the formation of memory.
- In the periphery there is a widespread network of nerves, previously recognized as nonadrenergic and noncholinergic, that operate through a NO-dependent mechanism to mediate some forms of neurogenic vasodilatation.

IN THE IMMUNE SYSTEM:
- NO is produced in large quantities during host defense and immunologic reactions. Because it has cytotoxic properties and is generated by activated macrophages, it is likely to have a role in nonspecific immunity.


MECHANISM OF NITRIC OXIDE TREATMENT IN ARDS

Nitric oxide is an endogenous endothelium-derived relaxing factor (ERDF) and a potent endogenous vasodilator that has been recently administered for the treatment of ARDS. In the ventilated patient, it is delivered in parts per million (ppm) during the inspiratory phase blended with oxygen and delivered as a gas by ventilator. When inhaled, NO produces local vasodilatation in ventilated regions of the lungs and reverses pulmonary vasoconstriction due to hypoxemia. It was first used in experimental models and reversed pulmonary vasoconstriction caused by severe hypoxia. In healthy volunteers it had similar effects without causing systemic vasodilatation or other side effects.

Nitric oxide has pharmacodynamic advantages over an intravenous vasodilator such as prostacyclin. It is delivered as a gas and therefore reaches only ventilated areas. Conversely, prostacyclin has a nonselective effect on the pulmonary vasculature that can lead to ventilation-perfusion mismatching. That is, vasodilatation occurs in perfused as well as nonperfused areas of the lungs, which can result in suboptimal gas exchange. Improvement in arterial oxygenation with NO in patients with ARDS is thought to be caused by redistribution of pulmonary perfusion toward ventilated areas only. Another advantage of NO results from its rapid inactivation by hemoglobin to form methemoglobin. This process localizes the effects of NO to vascular smooth muscle adjacent to the alveolar unit, preventing substantial systemic vasodilatation. In addition, unlike intravenous prostacyclin, NO does not precipitate myocardial ischemia or heart failure and may be safe in patients with these conditions.

Potential concerns with NO are direct toxicity, toxicity of its oxidative product nitrogen dioxide (NO₂), formation of methemoglobin, inhibition of platelet aggregation, and possible negative inotropic effect. Blood methemoglobin, which is formed when NO reacts with hemoglobin, is regularly measured in patients receiving NO. The level of methemoglobin during NO therapy depends on the amount formed from oxidation and the amount eliminated by reduction within red blood cells. Hence the concentration depends on patients' methemoglobin reductase system capacity, NO dose, and possibly length of treatment. It is recommended to keep the concentration below 2%, although patients rarely show signs of cyanosis at concentrations under 25%. Trials of NO in ARDS generally recorded very low levels of methemoglobin (< 1.5%) and no signs of NO-associated toxicity.
Discuss the prevalent risk factors for ARDS

Acute Respiratory Distress Syndrome is a condition characterized by acute hypoxemic respiratory failure due to pulmonary edema caused by increased permeability of the alveolar-capillary barrier. ARDS represents the most serious manifestation of a spectrum of responses to acute lung injury; these responses occur as complications of a more widespread systemic response to acute inflammation or injury. The acute lung injury may occur in the setting of severe hypoxemia of acute onset.

ARDS is defined by the severity of hypoxemia (arterial pO2 to inspired oxygen fraction ratio [PaO₂/FIO₂] = 200 mmHg), along with bilateral diffuse opacities on frontal CXR when left atrial hypertension has been excluded. ARDS should be viewed as an early and recognizable manifestation of a systemic infectious or inflammatory disorder. The lung figures prominently in systemic injury because it receives the entire cardiac output and because pulmonary impairment is readily apparent clinically.

Acute lung injury develops rapidly after a predisposing condition triggers a systemic inflammatory response. ARDS is most strongly associated with conditions that produce direct alveolar injury or indirect injury via the pulmonary capillary bed.

*Conditions that may lead to Acute (formerly Adult) Respiratory Distress Syndrome:*

**Direct Alveolar Epithelial Injury:** Aspiration, Diffuse infection, Near-drowning, Toxic Inhalation, Airway Contusion.

**Indirect Alveolar Epithelial Injury:** Sepsis syndrome (43% risk of ARDS), Severe non-thoracic trauma, Hypertransfusion, Cardiopulmonary bypass, and Drug overdose (13% risk).

Reference: *Harrison's Principles of Internal Medicine.* Honig, Ingram, 14th edition; Chapter 265

*Cameron: Current Surgical Therapy.* 7th edition; pp1358-9
Describe the signs of a large AV fistula.

**Definition of an Arteriovenous Fistula**

?? An arteriovenous (AV) fistula is formed by an abnormal connection between arteries and veins. Higher-pressure arterial flow is directed into the lower-pressure veins which diverts blood supply to distal tissues and engorges distal veins.

**Etiology of Arteriovenous Fistulas**

?? Post-traumatic—i.e. penetrating trauma—GSW or stab wounds, especially in thigh
?? Iatrogenic—i.e. low groin puncture that perforates the superficial or deep femoral artery and an adjacent vein
?? Rupture of an arterial aneurysm into an adjacent vein
?? Inflammatory necrosis associated with neoplasms or infection
?? Surgical (hemodialysis access)
?? Congenital—hemangiomas, hereditary hemorrhagic telangiectasia (Osler-Weber-Rendu Syndrome)

**Physiology of Arteriovenous Fistulas**

?? Depend on the size of the proximal and distal arteries and veins, the collateral flow around the fistula, and the diameter of the fistula
?? Large functioning AV fistulas may cause a fall in both systolic and diastolic blood pressures, an increase in cardiac output, and increase in venous blood pressure both proximal and distal to the fistula, an increase in pulse rate, slight increase in the size of the heart, and an increase in blood volume

**Signs of a Large Arteriovenous Fistula**

?? Audible bruit (continuous machinery-like murmur)
?? Palpable thrill
?? Massive distension of distal superficial veins since arterial flow is directed into distensible veins
?? Warm temperature of the skin overlying the AV fistula, but distally the temperature is often decreased
?? In an affected extremity—larger size, warmer skin, presence of cellulitis, venostasis, edema, and dermatitis with pigmentation secondary to chronically elevated venous stasis pressure.
?? Widened pulse pressure
?? Mild tachycardia
?? High Output Heart Failure—3rd and 4th heart sounds, midsystolic murmur secondary to increased cardiac output, EKG changes of left ventricular hypertrophy
?? Branham’s sign—decrease in tachycardia when the artery leading to the fistula or the fistula itself is occluded; this maneuver also raises arterial and lowers venous pressure

**References:**

Hemodynamic effects of a tension pneumothorax

Increased pressure in the pleural space causes reduced systemic veinous return to the heart and reduces cardiac output. Neck veins will be distended unless the patient is also intravascularly depleted. Despite this, the most common cause of distended neck veins in thoracic trauma patient is tension pneumothorax. Lung tissue is compressed, and there is a decrease in pulmonary compliance, diffusing capacity, ventilatory capacity, and an increase in peak pressures.

Pneumothorax on Positive Pressure Ventilation

Pneumothorax results from air entering the potential space between the visceral and parietal pleura. Both penetrating and non-penetrating trauma may cause this injury. Lung laceration with air leakage is the most common cause of pneumothorax resulting from blunt trauma.

The thorax is normally and completely filled by the lung, held to the chest wall by surface tension between the pleural surfaces. Air in the pleural space collapses lung tissue. A ventilation/perfusion defect occurs because the blood perfusing the nonventilated area is not oxygenated.

A pneumothorax is best treated with a chest tube in the 4th or 5th intercostal space, anterior to midaxillary line. Observation and/or aspiration of any pneumothorax is risky. Once a chest tube is inserted and connected to an underwater seal apparatus with or without suction, a CXR is necessary to confirm re-expansion of the lung. General anesthesia or POSITIVE PRESSURE VENTILATION should never be administered in a patient who sustains a traumatic pneumothorax or who is at risk for unexpected intraoperative pneumothorax until a chest tube is inserted. A simple pneumothorax can readily be converted into a life-threatening tension pneumothorax, particularly if it is initially unrecognized and positive pressure ventilation is applied. The patient's chest should be decompressed before transporting the patient with a pneumothorax via air ambulance.

References
ATLS Manual
Discuss the most important determinant of O2 content

Oxygen content of the blood is determined predominantly by the ability of hemoglobin to bind to oxygen. Therefore, it is linearly related to hemoglobin concentration (Hgb) and percent oxygen saturation (SO₂). A minor contributor to oxygen content is the amount of oxygen dissolved in plasma, which is proportional to the partial pressure of oxygen in plasma (PO₂). Oxygen content of blood (CO₂) is calculated:

\[ CO₂ = (1.37 \times % \text{ SO}_2 \times \text{Hgb}) + (0.003 \times \text{PaO}_2) \]

In clinical situations, PO₂ and oxygen saturation are often used interchangeably. Although related, PO₂ and oxygen saturation have a complex relationship, as described by the hemoglobin-oxygen dissociation curve. At low levels of oxygen tension (point A to point B), increases in PO₂ translate into only small increases in the percentage of oxygen bound to hemoglobin. During mid-range oxygen tension (point B to point C), however, the relationship of PO₂ to oxygen-hemoglobin binding is nearly linear, with significant increases in oxygen saturation resulting from increases in PO₂. This relationship is not linear at higher oxygen tension (point C to point D), such that continued increases in PO₂ result in very little increase in oxygen saturation of hemoglobin.

**Figure:** Oxygen and hemoglobin (Hgb) dissociation curve. A sigmoid-shaped curve shows maximal oxygen loading in the lung and unloading of O₂ in the periphery occurring over a very narrow range of PaO₂.