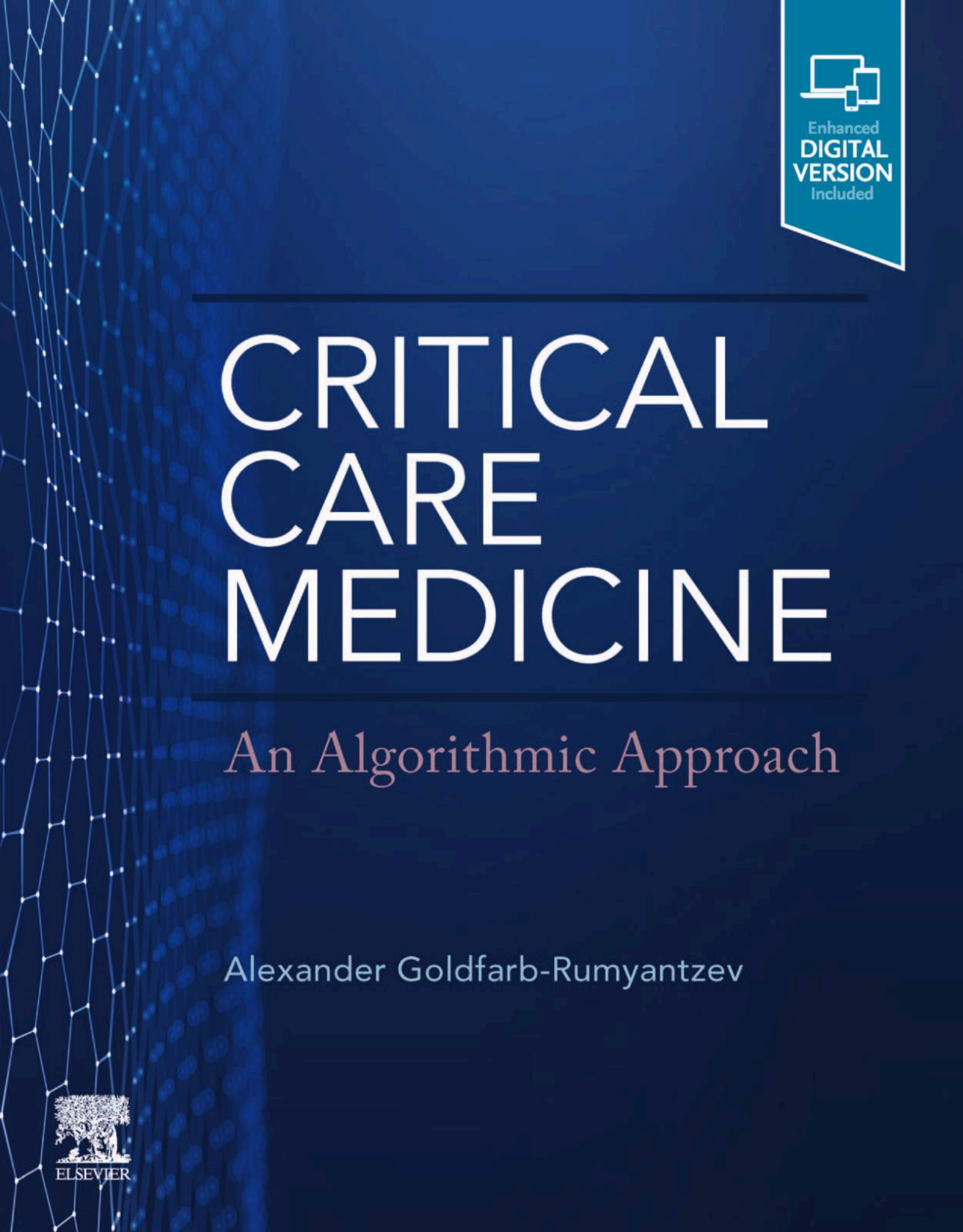
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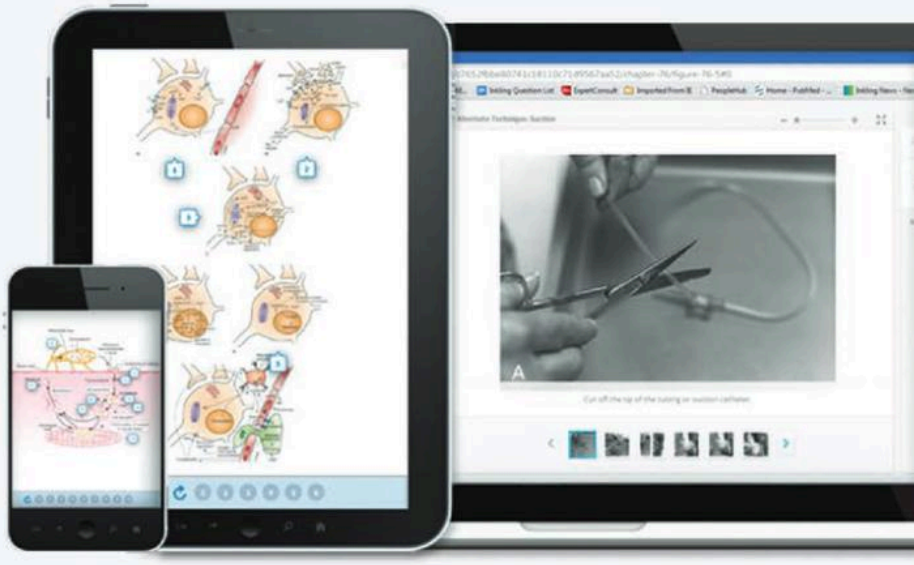

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CRITICAL CARE MEDICINE

An Algorithmic Approach

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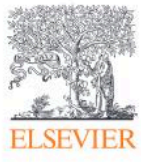

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Dedication

To my family, and specifically to my mom Tatiana, my late uncle Veniamin, and my children Levi and Ben – my constant source of inspiration.

Alexander Goldfarb-Rumyantzev

To my wife Judy, son Bobby, and daughter Debbie who have always supported my hours at work and to the late Frank Epstein – mentor, colleague, and friend for 40 years.

Robert S. Brown

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Dedicated in memory of Alexander Goldfarb-Rumyantzev, MD, PhD, PhD,
a colleague, co-author and friend,

This book is all Alex' doing – its conception, structure and writing. His unexpected passing on January 18th, 2021 was a loss to us all – his innovative thinking, logic, spirit, wit and humor will not be forgotten.

Robert S. Brown, MD

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Preface

Critical Care Medicine is a broad subject that covers many areas and almost all subspecialties of internal medicine. As one might remember from one's years in residency, the ICU rotation is exciting and the favorite of many. In this book we discuss practical issues of critical care medicine divided into chapters by subspecialty. Specifically, we separated the following aspects of critical care medicine: respiratory, cardiac and circulation, infectious disease, water and electrolytes and acid-base disorders, acute kidney injury and dialysis, gastroenterology, rheumatology, endocrinology, neurology, and COVID-19. Arguably, many aspects of critical care medicine are also relevant to general internal medicine. In effect, critical care is an internal medicine subspecialty focused on very sick people (plus invasive procedures). As such, the chapters in this book are applicable to the practice of general medicine as well. Therefore, the intended audience for this book includes critical care practitioners, as well as internal medicine physicians, and fellows and residents in critical care, internal medicine, and its subspecialties.

Let me point out what this book is NOT. First of all, it is important to note that the goal of this book is not to give comprehensive coverage of the topics, nor to provide a fundamental understanding of the physiology of the discussed conditions. Rather, we address the need for quick decision making in situations where timing is of essence. This book is intended to be a source of quick reference to provide help in approaching conditions frequently encountered in the intensive care unit, in formulating the plan of care, and in making a decision regarding the next step in management of a critical patient. In essence, this book allows the provider to alleviate the most urgent clinical matter and buy some time to regroup, think, call consults, and obtain more detailed and comprehensive information. By no means does it eliminates the need for a physician to read further and have a deeper understanding of the subject—of special importance is the understanding of the physiology of critical illnesses. Medicine is practiced in a rapidly changing environment and new information is coming daily. This book does not substitute the need to be on the top of contemporary literature. Understanding of the underlying disease process is very important, so, once the initial strategy is established and next steps are clear in general terms, the provider should probably step back and get additional information from more detailed sources. Along the same lines, this book cannot cover all topics, and the authors had to be selective. Because the purpose of the book is to be a source of quick reference, we selected topics representing common issues in critical care medicine, those that practitioners are dealing with on almost a daily basis, and those that require decisive steps.

The format of this book is different from most textbooks in that it is based mostly on graphical representation of information: diagrams, tables, algorithms. We believe that this format will be helpful to practitioners looking for concise data and references in an environment where decisions need to be made quickly.

Most of the references used for this book are open access sources. We specifically made an effort to select appropriate sources that would be easily available to readers, unless these sources were insufficient.

Four chapters in this book (Water and electrolyte disorders, Acid-base disorders, Acute kidney injury and dialysis, and COVID-19) were co-authored by Dr. Robert S. Brown.

We feel sure that you will find this book helpful in your daily practice and we are very much open to suggestions how to make the next edition better.

Alexander S. Goldfarb-Rumyantzev, MD, PhD, PhD

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Contents

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CHAPTER 1

Respiratory Failure

Alexander Goldfarb-Rumyantzev

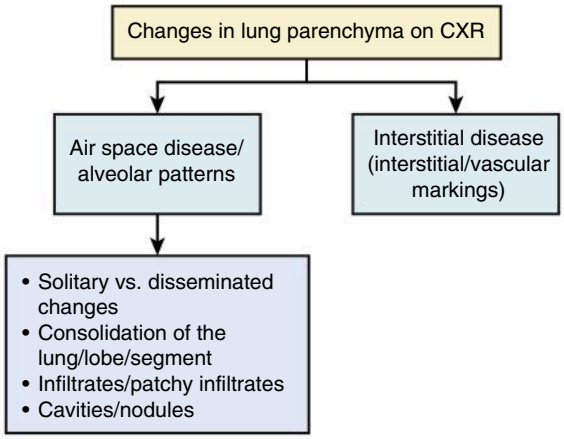
Pulmonary

The chapter addresses two large areas of critical care medicine, specifically, acute respiratory failure and means of artificial gas exchange, such as mechanical ventilation and extracorporeal membrane gas exchange.

Diagnostic Tests

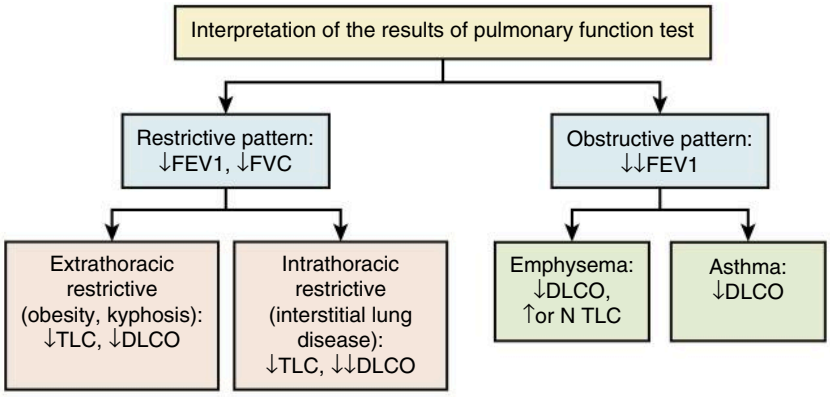
Chest X-Ray Assessment Algorithm

- Technical issues:
  - view (anterior-posterior/lateral), position/rotation
  - quality and penetration
  - inspiratory effort (number of ribs)
- Evaluate soft tissue
- Evaluate bones: ribs, vertebrae
- Heart, mediastinum, trachea
- Lungs contour: costo-diaphragmal angles, diaphragm, presence of pleural effusion/pneumothorax
- Lungs parenchyma:
  - dilated hila (dilated veins in congestive heart failure [CHF], dilated arteries in congenital defects, lymph nodes, tumor masses)
  - changes in lung parenchyma (e.g., infiltrate, pulmonary edema)



Pulmonary Function Test Interpretation

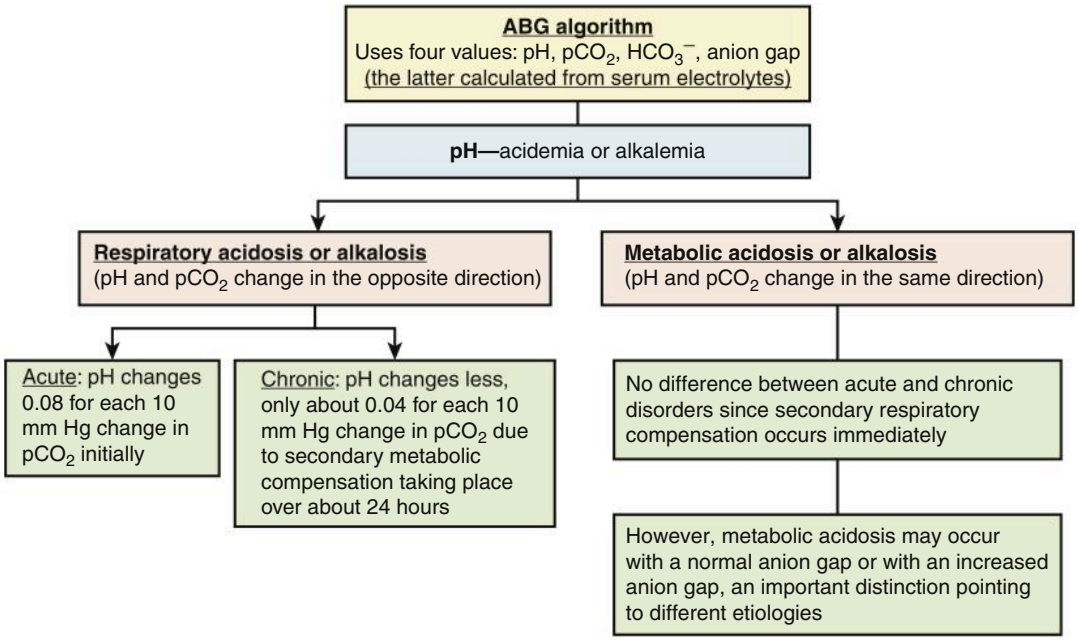
The pulmonary function test is used to diagnose and stage restrictive (caused by extrathoracic or intrathoracic problem) or obstructive lung disease. Restrictive lung diseases cause problems that impair lung expansion, which lead to decreased lung volume (e.g., obesity, interstitial lung disease). On the other hand, in obstructive lung disease, lung volume is usually preserved, but there is an impairment to air flow, potentially caused by bronchospasm or other airway obstruction.



Arterial Blood Gas Analysis

Acid-base disorder diagnostic algorithm

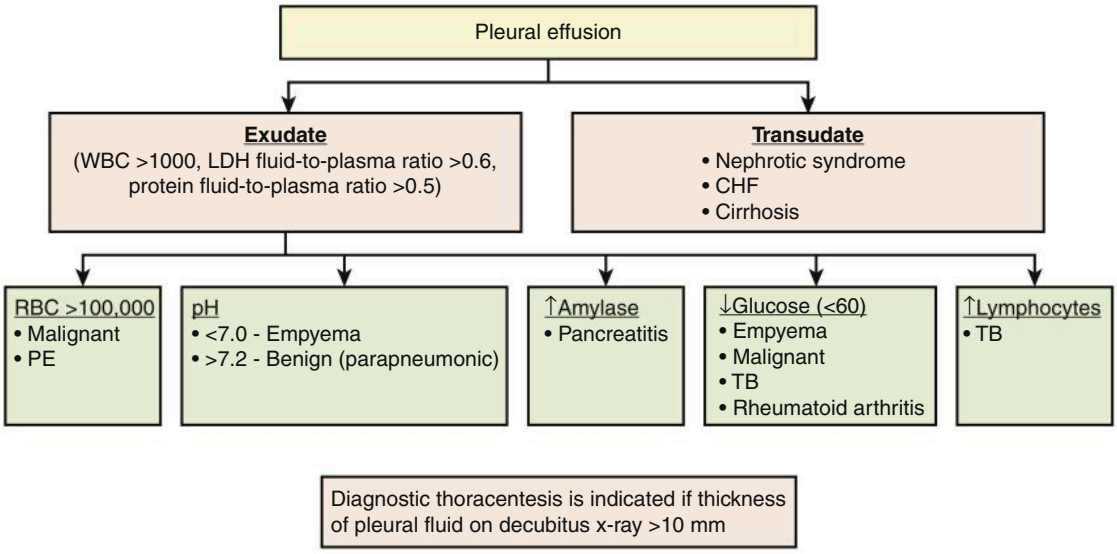
The following diagram provides the algorithm of interpretation of arterial blood gases (ABGs) used in conjunction with plasma chemistry. To use this algorithm, first examine the pH and identify acidemia or alkalemia, then using the bicarbonate concentration from the serum electrolytes and pCO_2, identify whether the primary cause of the disorder is metabolic or respiratory. Finally, perform a calculation to examine if secondary metabolic compensation for a primary respiratory disorder or respiratory compensation for a primary metabolic disorder is appropriate. If not, there is a second primary disorder, considered to be a "complex" (meaning more than one) acid-base disorder, rather than a "simple" (meaning single) acid-base disorder underlying the observed changes.



Please note that more extensive discussion on acid-base disorders is available in the Chapter 5 of this book.

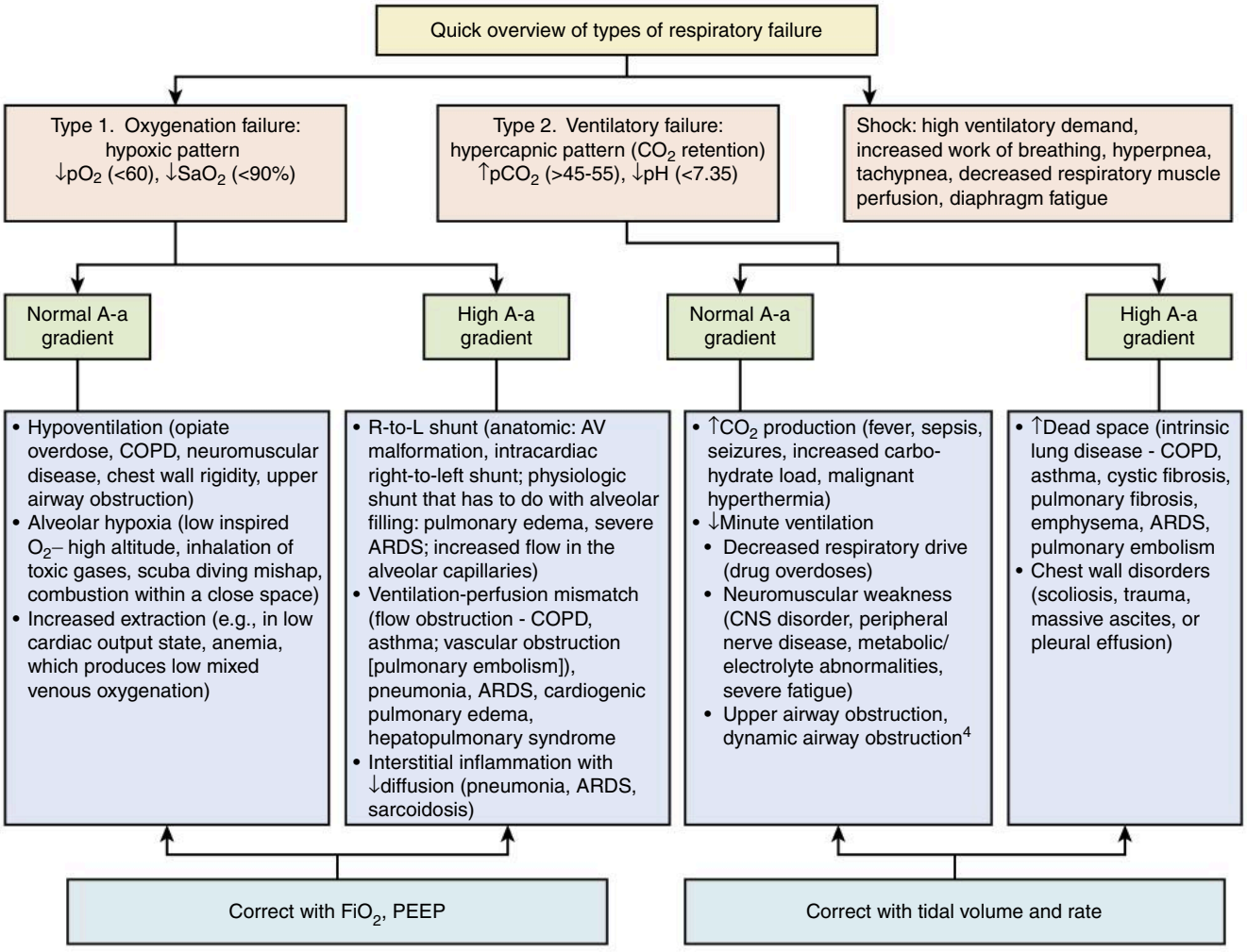
Pleural effusion

The normal amount of pleural fluid is about 10 mL. Pleural effusion might be formed due to several potential causes: increased fluid formation (increased amount of interstitial fluid in the lungs, increased intravascular pressure in the pleura, decreased pleural pressure, increased permeability of the pleura, increased pleural protein level, increased amount of peritoneal fluid disruption of blood vessels or lymphatics in the thorax) or decreased fluid absorption (obstruction of the lymphatics draining pleural fluid, disruption of the aquaporin system in the pleura, elevated systemic vascular pressure). The first diagnostic question of pleural fluid analysis is if it represents a transudate or an exudate.[2]



Acute Respiratory Failure

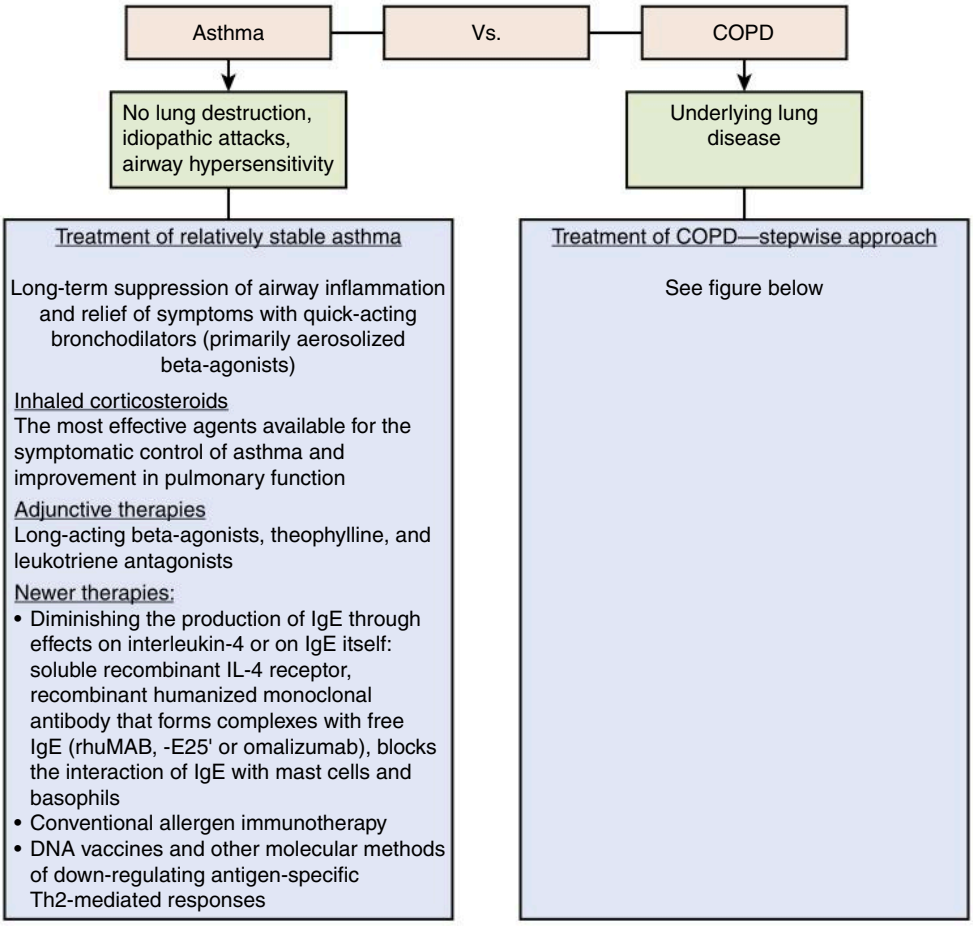
Acute respiratory failure is one of the most common conditions that requires patient to be treated in Intensive Care Unit (ICU). Unlike many other life-threatening conditions requiring ICU admission, respiratory failure presents immediate risk and needs to be addressed promptly. In a simplified format, respiration entails gas exchange with O_2 being absorbed and CO_2 excreted by the lungs. As a result, respiratory failure could be viewed either as a deficiency in oxygenation or as a failure to excrete CO_2. Some look at respiratory failure in sepsis as a separate entity, whereas others classify it within either hypoxemic or ventilatory failure. The next chart is a general algorithm describing types of respiratory failure and their mechanisms.[3] We provide more details about specific conditions below.

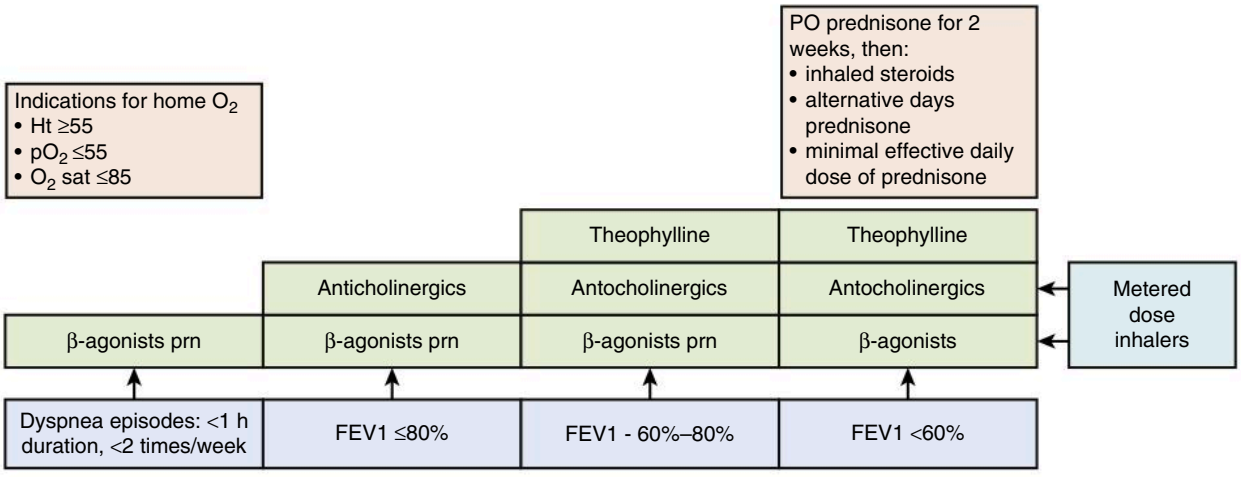


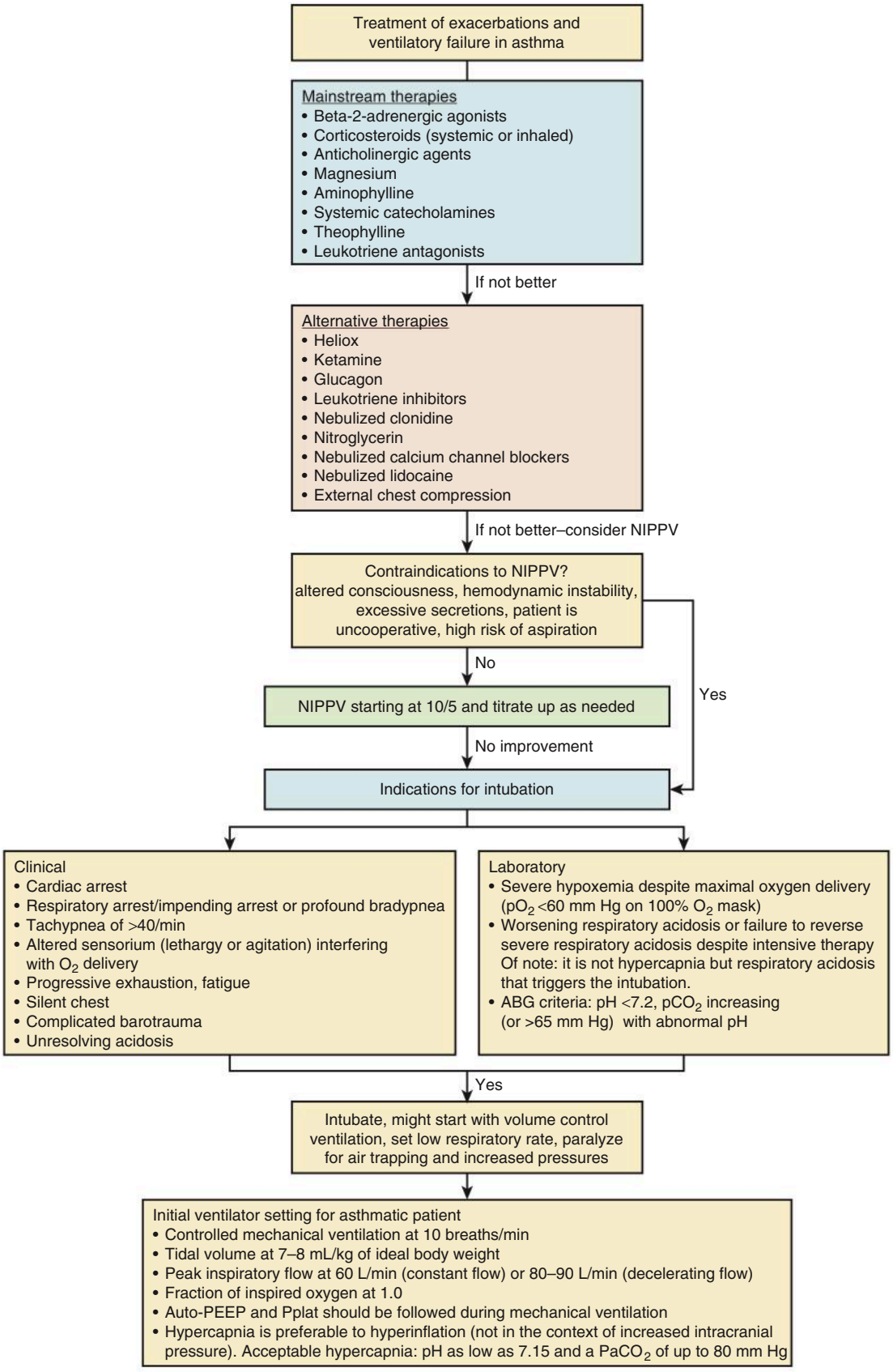
Ventilatory Failure

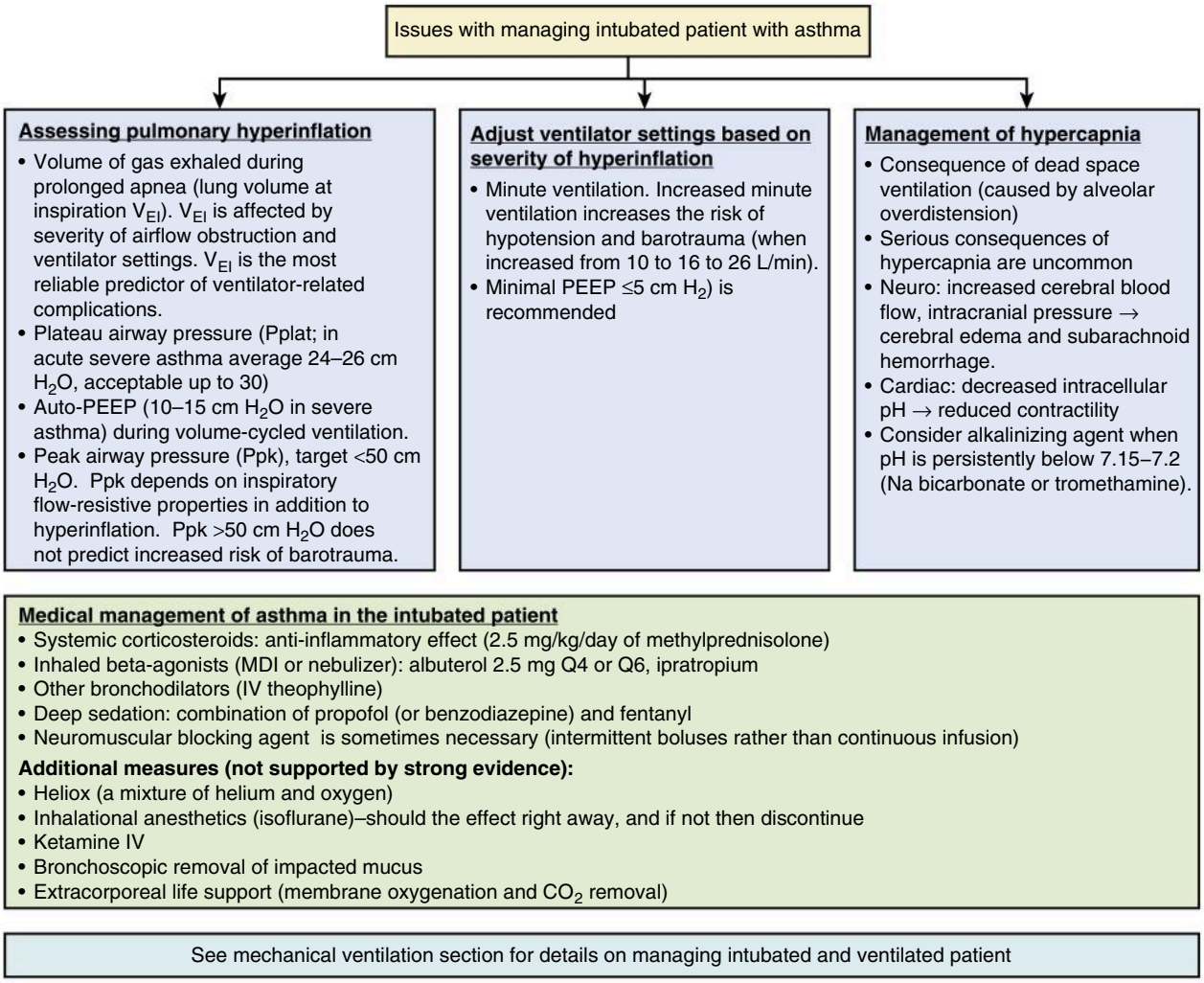
Asthma and Chronic Obstructive Pulmonary Disease

Although pathophysiologies of asthma and chronic obstructive pulmonary disease (COPD) are different, the end result leading to ventilator failure is similar and is based on hypoventilation. Therefore whereas approaches to treatment of noncritical stable asthma and COPD might be different, once it reaches the stage of respiratory failure, the focus in both conditions is to relieve bronchospasm and provide adequate ventilation. However, one has to be cautious about gas trapping which can precipitate hemodynamic instability and barotrauma.[5,6]






Treatment of exacerbations and ventilatory failure in asthma
Mainstream therapies
• Beta-2-adrenergic agonists
• Corticosteroids (systemic or inhaled)
• Anticholinergic agents
• Magnesium
• Aminophylline
• Systemic catecholamines
• Theophylline
• Leukotriene antagonists
If not better
Alternative therapies
• Heliox
• Ketamine
• Glucagon
• Leukotriene inhibitors
• Nebulized clonidine
• Nitroglycerin
• Nebulized calcium channel blockers
• Nebulized lidocaine
• External chest compression
If not better–consider NIPPV
Contraindications to NIPPV? altered consciousness, hemodynamic instability, excessive secretions, patient is uncooperative, high risk of aspiration
No
Yes
NIPPV starting at 10/5 and titrate up as needed
No improvement
Indications for intubation
Clinical
• Cardiac arrest
• Respiratory arrest/impending arrest or profound bradypnea
• Tachypnea of >40/min
• Altered sensorium (lethargy or agitation) interfering with O_2 delivery
• Progressive exhaustion, fatigue
• Silent chest
• Complicated barotrauma
• Unresolving acidosis
Laboratory
• Severe hypoxemia despite maximal oxygen delivery (pO_2 <60 mm Hg on 100% O_2 mask)
• Worsening respiratory acidosis or failure to reverse severe respiratory acidosis despite intensive therapy Of note: it is not hypercapnia but respiratory acidosis that triggers the intubation.
• ABG criteria: pH <7.2, pCO_2 increasing (or >65 mm Hg) with abnormal pH
Yes
Intubate, might start with volume control ventilation, set low respiratory rate, paralyze for air trapping and increased pressures
Initial ventilator setting for asthmatic patient
• Controlled mechanical ventilation at 10 breaths/min
• Tidal volume at 7–8 mL/kg of ideal body weight
• Peak inspiratory flow at 60 L/min (constant flow) or 80–90 L/min (decelerating flow)
• Fraction of inspired oxygen at 1.0
• Auto-PEEP and Pplat should be followed during mechanical ventilation
• Hypercapnia is preferable to hyperinflation (not in the context of increased intracranial pressure). Acceptable hypercapnia: pH as low as 7.15 and a $PaCO_2$ of up to 80 mm Hg



Medical Management of Asthma in the Intubated Patient

- Systemic corticosteroids: antiinflammatory effect (2.5 mg/kg per day of methylprednisolone)
- Inhaled beta-agonists (MDI or nebulizer): albuterol 2.5 mg Q4 or Q6, ipratropium
- Other bronchodilators (IV theophylline)
- Deep sedation: combination of propofol (or benzodiazepine) and fentanyl
- Neuromuscular blocking agent is sometimes necessary (intermittent boluses rather than continuous infusion)

Additional Measures (not Supported by Strong Evidence)

- Heliox (a mixture of helium and oxygen)
- Inhalational anesthetics (isoflurane)—the effect should be right away; if not, then discontinue
- Ketamine IV
- Bronchoscopic removal of impacted mucus
- Extracorporeal life support (membrane oxygenation and CO_2 removal)

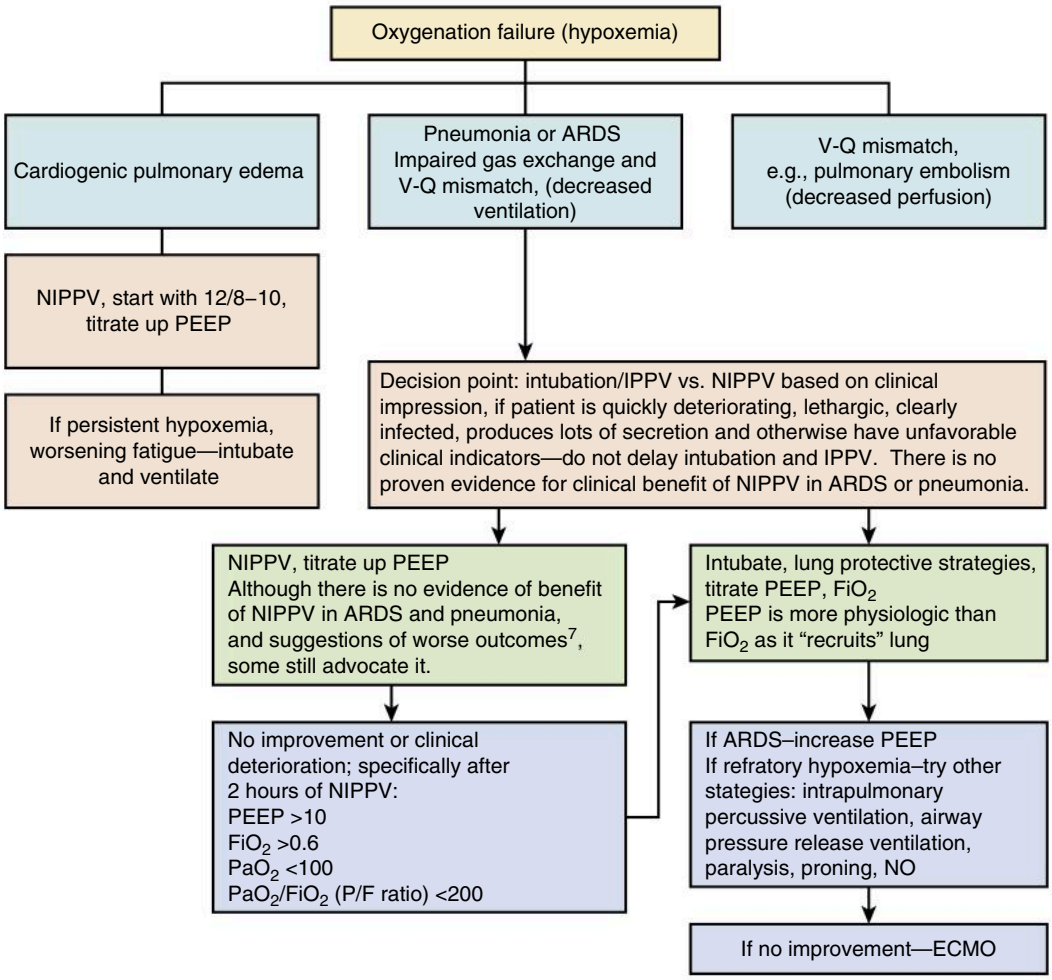
MDI, Metered dose inhaler.

See Mechanical Ventilation section for details on managing intubated and ventilated patient

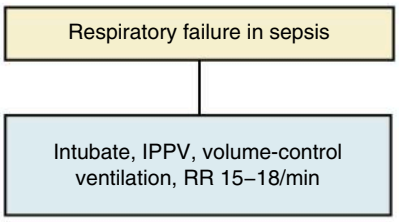
Hypoxemic Respiratory Failure

A number of mechanisms can lead to hypoxemic respiratory failure, resulting either from oxygen delivery problems (acute respiratory distress syndrome [ARDS], pneumonia, pulmonary edema, high altitude) or lung perfusion problems (pulmonary embolism, shunting).

Below is the general approach to treatment of hypoxemic respiratory failure; we also discuss special cases (ARDS, pulmonary embolism) in more detail.



Other than ventilator failure and hypoxemic respiratory failure, some separate respiratory failure in sepsis into a separate entity, whereas in fact it is for the most part a multifactorial combination. Intubation and invasive positive pressure ventilation (IPPV) is the treatment of choice for the respiratory failure in sepsis.



ARDS

ARDS is characterized by increased permeability of the alveolar capillary membrane, diffuse alveolar damage, and accumulation of proteinaceous alveolar edema. Mortality remains very high (>40%) and does not seem to decrease between 1994 and 2006.[8] That, in addition to high incidence and relatively limited therapeutic options, makes ARDS a serious and mostly unresolved issue in critical care.[1,3,9–12]

Establish the diagnosis: ARDS definition

1. Acute onset, presence of inciting event
2. PaO_2/FiO_2 ≤200 (regardless of PEEP level)
3. Bilateral infiltrates seen on frontal chest radiograph
4. PCWP ≤18 mm Hg or no clinical evidence of left atrial hypertension

Classification by PaO_2/FiO_2 ratio
- Acute lung injury (ALI) or mild ARDS: <300
- Moderate >100–200
- Severe ≤100

Differential diagnosis and diagnostic steps

- Rule out other similar presentations: interstitial lung disease, malignancy presenting similar to ARDS, acute eosinophilic pneumonia, diffuse alveolar hemorrhage, hypersensitivity pneumonitis, and pulmonary alveolar proteinosis
- Consider BAL: identify infectious causes (e.g., bacterial or viral)
- Consider lung biopsy if
  - o high clinical suspicion for a "contributive result" (results leading to additional therapy)[13]
  - o the risk of empirical therapy is too high
  - o when empirical therapy has been unsuccessful[14]
  - o note that lung biopsy presents substantial risk in ventilated patient on high PEEP and benefits must clearly outweigh risks

Therapies

Effective therapies:
- Lung protective ventilation (lower tidal volume and airway pressures) (see mechanical ventilation box below)
- Neuromuscular blockade[15,16] cisatracurium should be considered for short-term use (<48 h) in patients with severe ARDS (defined as PaO_2/FiO_2 <120 mm Hg) until further studies are available[17]
- Esophageal pressure to adjust PEEP (improves oxygenation)
- Fluid conservative vs fluid-liberal therapy (see fluid management box below)
- Extracorporeal membrane oxygenaton (see ECMO section for details)

No proven benefit:
- High PEEP
- High frequency ventilation
- Early prone positioning
- Continuous administration of surfactant has no effect on 30-day survival, duration of mechanical ventilation, or physiologic function
- Activated protein C (APC)
- GM-CSF
- Pulmonary artery catheter
- Methylprednisolone/steroids (questionable benefit). Some recommend 7–14-day trial of 2–4 mg/kg prednisone in patients with severe ARDS who show no clinical signs of improvement. Rule out or treat systemic infections.
- Omega-3 fatty acid (may be harmful)
- Beta-2 agonists
- Antioxidants
- Vasodilator therapy (liposomal prostoglandin E1, nitric oxide). Liposomal PGE^1 blocks platelet aggregation, downregulates neutrophil-mediated inflammation, produce vasodilatation.
- Ketoconazole inhibits tromboxane synthesis and biosynthesis of leukotriens
- N-acetylcysteine

Details of some specific therapeutic approaches

Details of fluid management

- Excessive fluid administration may lead to an increased amount of pulmonary edema and a worsening of oxygenation. On the other hand, inadequate intravascular volume may create decreased cardiac output and inadequate perfusion of the organs. Monitor biochemical markers of organ dysfunction and lactate.
- If the patient is anemic (Hct <21), transfusion of packed RBC can be used as a volume expander.
- Otherwise crystalloids or colloids should be utilized.
- Volume depletion may be accomplished with diuretics.

Details of mechanical ventilation

- Optimize sedation and analgesia.
- Lower initial tidal volume (6–8 mL/kg), do not produce a transpulmonary pressure that exceeds 30–35 cm H_2O.
- Titrate PEEP to maintain an arterial oxygen saturation of >90% at an FiO_2 <60% (usually PEEP 5–12 cm H_2O).
- Low volume-low pressure ventilation (plateau pressure <30 cm H_2O) to reduce mechanical stretch improves outcome in ARDS, but might lead to hypoxemia, poor lung compliance, severe respiratory acidosis.[11]
- Avoid oxygen toxicity if possible (FiO_2 <0.6).
- Inverse ratio ventilation (strategy, when the amount of time the lungs are in inhalation is greater than the amount of time they are in exhalation) may enhance recruitment at the expense of ventilation. The resultant effect on $PaCO_2$ is "permissive hypercapnia" (keep static peak airway pressure <40 cm H_2O, maintain O_2 saturation >90%, while tolerating pH as low as 7.15 before initiating IV buffering agents bicarbonate or THAM. Higher doses of propofol are required to sedate patients managed with permissive hypercapnia).
- Consider mechanical ventilation in the prone position.[1]
- Inhaled nitric oxide at 10 parts per million to improve VQ matching and oxygenation but has not been shown to alter outcome
- High-frequency oscillatory ventilation (see[10] for specific severity criteria)
- Airway pressure (release) ventilation (alternative approach to open lung)
- ECMO

Outcome: resolution might be slow and mortality remains very high

Resolution mechanism

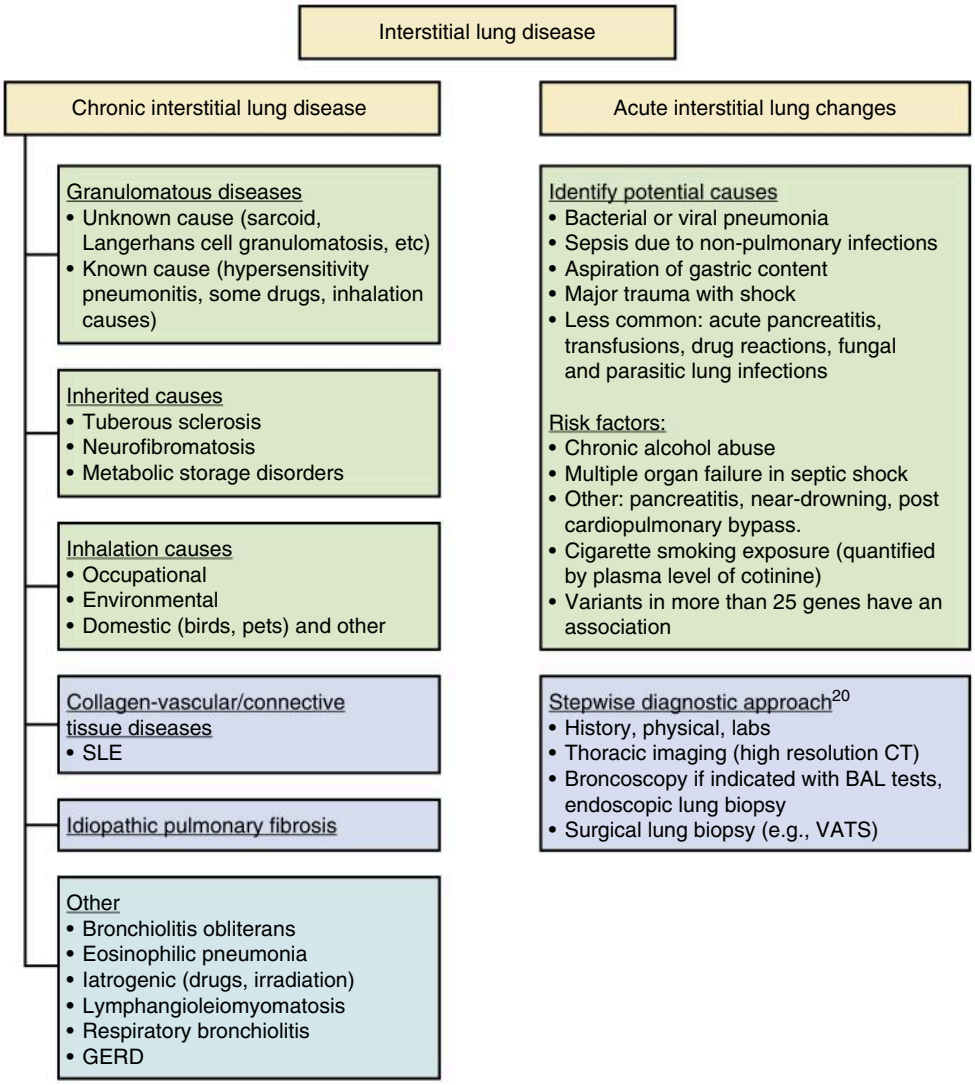
- reabsorbtion of alveolar edema
- repair of epithelial and endothelial barriers
- removal of inflammatory cells and exudate from distal airspaces

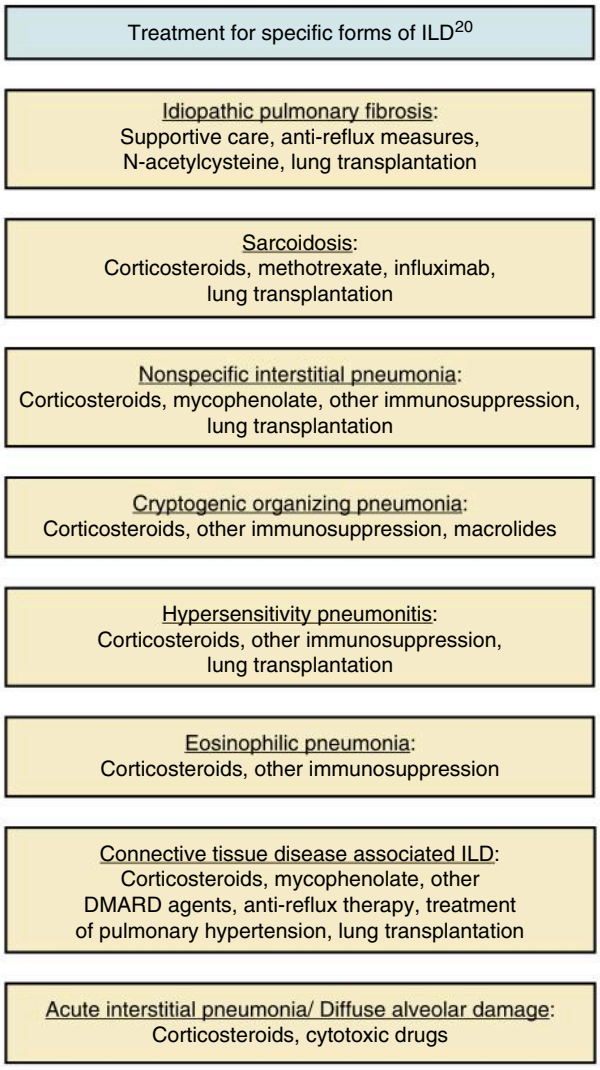
Causes of death[15]

- underlying illness or injury
- sepsis
- irreversible respiratory failure
- associated multi system organ failure due to unremitting hypoxia

Interstitial Lung Disease and Pulmonary Fibrosis

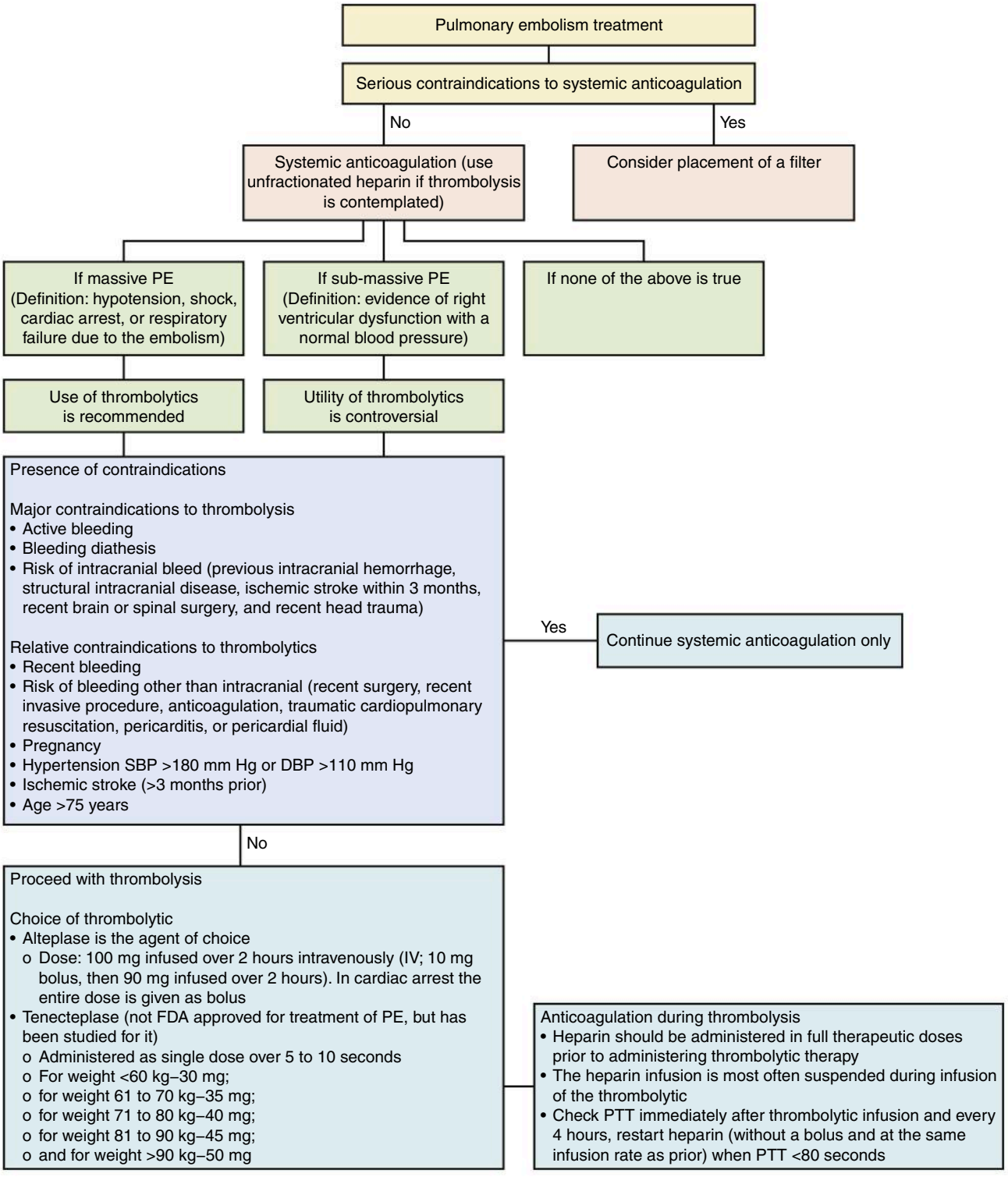
Underlying interstitial lung disease (ILD) might be a cause of hypoxemic respiratory failure. ILD refers to lung diseases affecting the interstitium of the lungs (alveolar epithelium, pulmonary capillary endothelium, basement membrane, and perivascular and perilymphatic tissues).[19] Detailed discussion of ILD management is outside the scope of this chapter; however, we briefly discuss the causes and diagnostic approach to ILD below.





Pulmonary Embolism

After establishing the diagnosis of pulmonary embolism, therapeutic options are (1) anticoagulation, (2) thrombolysis/thrombectomy, or (3) if anticoagulation is contraindicated—placement of intravenous filter. Treatment of pulmonary embolism is discussed in the diagram below.[21]


Pulmonary embolism treatment
Serious contraindications to systemic anticoagulation
No
Yes
Systemic anticoagulation (use unfractionated heparin if thrombolysis is contemplated)
Consider placement of a filter
If massive PE (Definition: hypotension, shock, cardiac arrest, or respiratory failure due to the embolism)
If sub-massive PE (Definition: evidence of right ventricular dysfunction with a normal blood pressure)
If none of the above is true
Use of thrombolytics is recommended
Utility of thrombolytics is controversial
Presence of contraindications
Major contraindications to thrombolysis
• Active bleeding
• Bleeding diathesis
• Risk of intracranial bleed (previous intracranial hemorrhage, structural intracranial disease, ischemic stroke within 3 months, recent brain or spinal surgery, and recent head trauma)
Relative contraindications to thrombolytics
• Recent bleeding
• Risk of bleeding other than intracranial (recent surgery, recent invasive procedure, anticoagulation, traumatic cardiopulmonary resuscitation, pericarditis, or pericardial fluid)
• Pregnancy
• Hypertension SBP >180 mm Hg or DBP >110 mm Hg
• Ischemic stroke (>3 months prior)
• Age >75 years
Yes
Continue systemic anticoagulation only
No
Proceed with thrombolysis
Choice of thrombolytic
• Alteplase is the agent of choice
o Dose: 100 mg infused over 2 hours intravenously (IV; 10 mg bolus, then 90 mg infused over 2 hours). In cardiac arrest the entire dose is given as bolus
• Tenecteplase (not FDA approved for treatment of PE, but has been studied for it)
o Administered as single dose over 5 to 10 seconds
o For weight <60 kg–30 mg;
o for weight 61 to 70 kg–35 mg;
o for weight 71 to 80 kg–40 mg;
o for weight 81 to 90 kg–45 mg;
o and for weight >90 kg–50 mg
Anticoagulation during thrombolysis
• Heparin should be administered in full therapeutic doses prior to administering thrombolytic therapy
• The heparin infusion is most often suspended during infusion of the thrombolytic
• Check PTT immediately after thrombolytic infusion and every 4 hours, restart heparin (without a bolus and at the same infusion rate as prior) when PTT <80 seconds

Mechanical Ventilation

Noninvasive Positive Pressure Ventilation

Noninvasive positive pressure ventilation (NIPPV) is an alternative to intubation and invasive ventilation that can be used for both ventilatory (COPD, asthma) and hypoxemic (cardiogenic pulmonary edema) failure.[22] It is a cost-effective and less invasive modality, but current evidence only supports its use in obstructive pulmonary disease and in cardiogenic pulmonary edema.

Benefits compared to invasive ventilation

- Lower risk of nosocomial infections (pneumonia, UTI, and catheter related)
- Less antibiotic use
- Shorter length of stay in the ICU
- Lower mortality
- No need for sedation/analgesia
- More comfortable to patient
- More rapid weaning

Indications

- Clinical: progressive dyspnea, tachypnea, increased work of breathing
- Respiratory acidosis and hypercapnea (pCO_2 >45 mm Hg, pH <7.35)
- Hypoxemia (PaO_2/FiO_2 <200)
- The use of NIPPV is well supported in the treatment of chronic obstructive pulmonary disease exacerbations, decompensated congestive heart failure, and in immunocompromised patients; but there is lack of evidence to support it in respiratory failure NOT caused by COPD or CHF
- Other acute respiratory failure scenarios where it can be used: facilitation of weaning/extubation and extubation failure, immunosuppressed patient, DNI status, palliative in end-stage patients, community acquired pneumonia (with or without COPD), asthma, obesity hypoventilation, post-op respiratory failure, and more
- Current data about the use of NIPPV in acute respiratory distress syndrome and pneumonia (hypoxemic respiratory failure) demonstrate poor outcomes

Contraindications

- Respiratory arrest
- Aspiration risk: inability to protect airway, swallowing impairment, excessive secretion
- Recent upper airway or GI surgery
- Technical issues: inability to fit the mask, uncooperative patient
- Medically unstable (shock, GIB, uncontrolled ischemia/arrhythmia) or multiorgan failure

Initial setting

- Set pressures at low levels initially (PEEP 4–5 cm H_2O, pressure support ~8 cm H_2O)
- Set FiO_2 aiming at SaO_2 >90%
- Reset pressures (increase PEEP to get SaO_2 >90%, pressure support to get expired tidal volume ≥6 mL/kg
- ABG at baseline and 1 hour after start
- Humidification if applied for >6 hours

Devices for NIPPV

- Helmet
- Total face mask
- Full face mask
- Nasal mask
- Mouthpiece
- Nasal pillows

Ventilation modalities

Bilevel ventilation (combination of pressure support and PEEP)

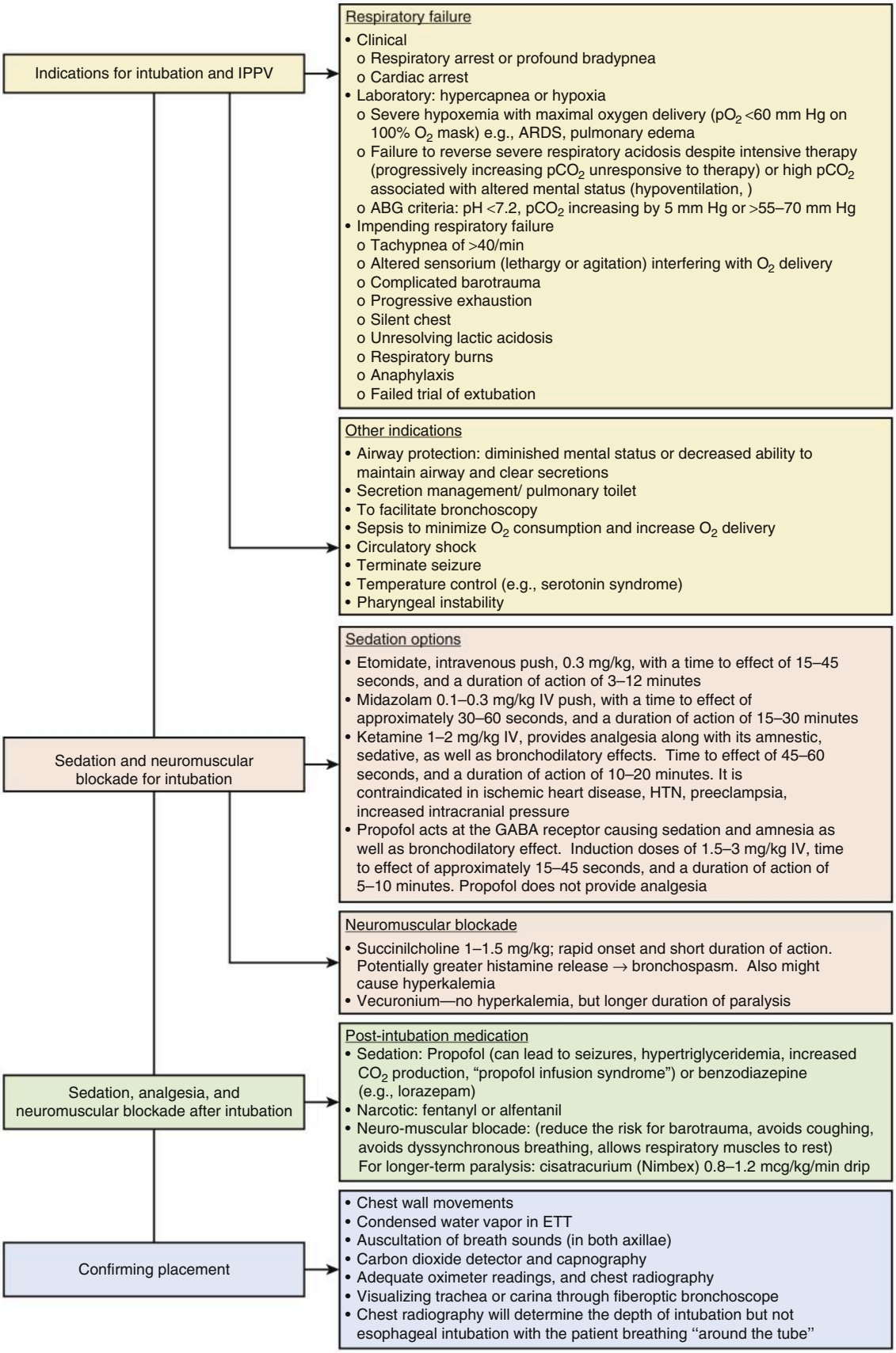
- Pressure-control: fluctuates between high inspiratory and low expiratory pressures; ventilator switch to expiration only after preset inspiratory time is reached (patient is unable to control the duration of inspiration)
- Proportional-assist ventilation: targets spontaneous inspiratory flow rate as a surrogate of patient's effort

Positive pressure

- CPAP: constant positive pressure is applied (to raise functional residual capacity and open flooded alveoli)
- Pressure-support ventilation: preset positive pressure boost triggered by the patient; pressure applied until inspiratory flow falls below a target pressure (patient controls breathing rate, duration of inspiration and expiration)

Endotracheal Intubation

Intubation is one of the most frequent procedures in critically ill patients. Whereas some indications are very clear, certain situations represent a gray area, where the decision might not be straightforward, mostly based on uncertainty whether the patient is going to deteriorate. As any invasive procedure, it carries a burden of complications and entails a commitment to mechanical ventilation, sedation, and sometimes paralysis, which should also be considered in the risk-benefit analysis.[5]



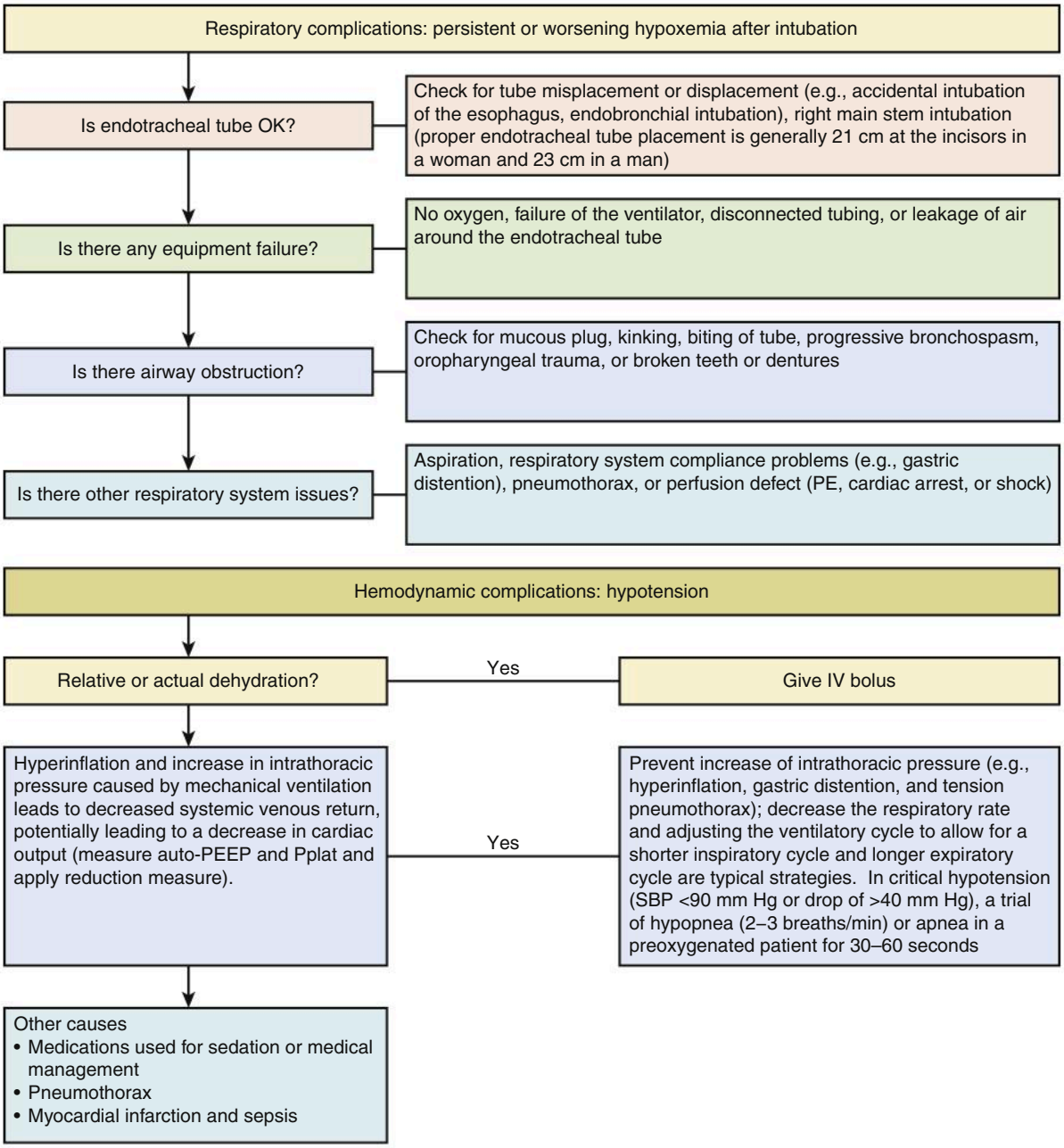
Complications of Intubation and Mechanical Ventilation

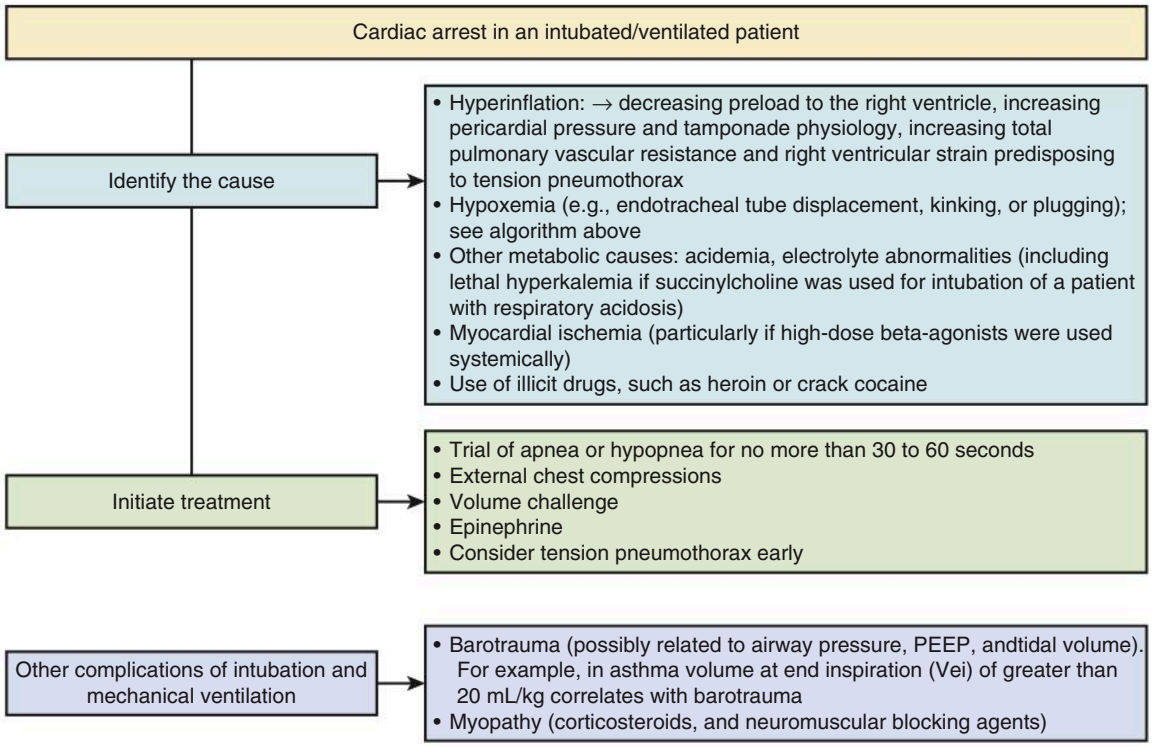
Complications of IPPV

- Ventilator-associated pneumonia
- Sepsis
- Venous thromboembolism
- Barotrauma
- Hypotension (by decreasing venous return, increase in right ventricular afterload risk related to degree of hyperinflation): 30–60-second apnea trial is recommended, rapid infusion of fluid, then if not better consider pneumothorax or myocardial depression
- Central Nervous System (CNS) injury (cerebral anoxia due to cardiorespiratory arrest prior to intubation)
- Muscle weakness due to acute myopathy (possibly effect of glucocorticoids and neuromuscular paralysis or due to prolonged near-total muscle inactivity)
- Pneumothorax (chest tubes should be placed by blunt dissection to avoid piercing hyperinflated lung)

Some of the specific complications of intubation and mechanical ventilation are discussed in more detail below.

(With permission from Henderson JJ, Popat MT, Latto IP, Pearce AC, Difficult Airway Society 2004.)





Difficult Airway

Difficult airway refers to two different clinical scenarios: difficult mask ventilation and difficult endotracheal intubation.[7,23,24]

Difficult Tracheal Intubation

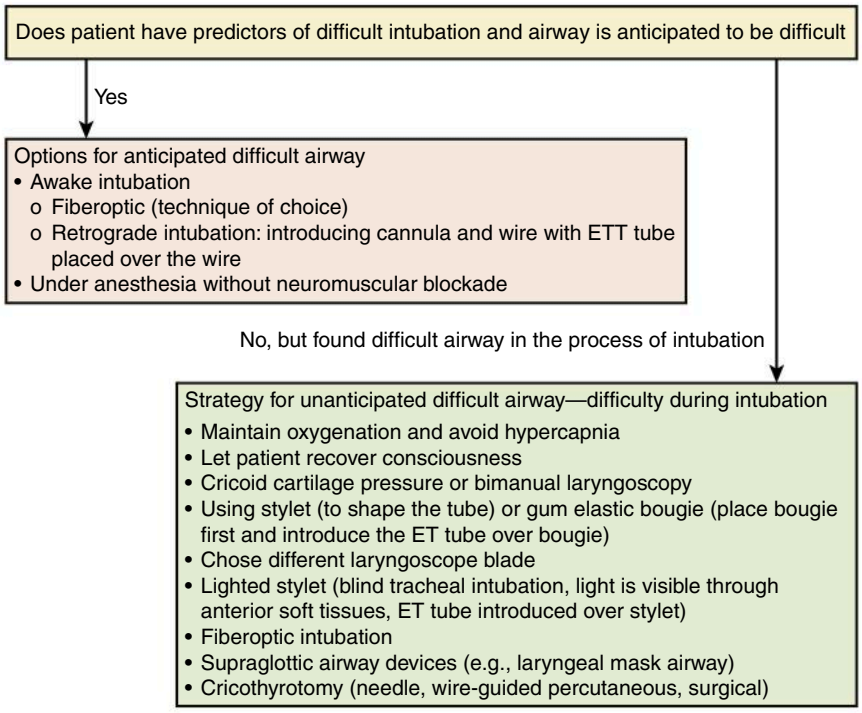
- Difficulty in visualization of the larynx (difficult direct laryngoscopy)
- Anatomic abnormalities (distortion or narrowing of larynx or trachea)

Prediction: Conditions Associated With Difficult Airway

- Abnormal facial anatomy/development
  - Small mouth, large tongue, dental abnormality
  - Obesity, advanced pregnancy, acromegaly
- Inability to open mouth
- Cervical immobility/abnormality
  - Short neck/obesity
  - Poor cervical mobility
- Pharyngeal and laryngeal abnormality
  - High or anterior larynx
  - Deep vallecula
  - Tumor
  - Subglottic stenosis
  - Anatomical abnormality of epiglottis

Other predictors

- Past airway difficulty
- Age >55 years
- Body mass index >26 kg/m^2
- Presence of beard
- Lack of teeth
- History of snoring



Extubation

Early weaning seems to be beneficial. There is higher mortality, increased rate of pneumonia, and longer hospital stay observed in the group with delayed discontinuation of mechanical ventilation. Although there is still a possibility of selection bias, if the patient seems to be ready to be extubated and meets the criteria, the extubation and discontinuation of mechanical ventilation should not be delayed. While that is true, approximately 15% of all patients who have been extubated and in whom mechanical ventilation has been discontinued require reintubation within 48 hours. Below are the approaches to weaning and extubation.[25]

Strategies to Reduce the Duration of Mechanical Ventilation

- Low TV (6 mL/kg of ideal body weight) in patients with ARDS
- Sedation
  - o Wake up (interrupt sedation) patient daily and prior to spontaneous breathing trial
  - o No use of sedatives
- Early physical therapy
- Conservative fluid management
- Strategies to reduce ventilator-associated pneumonia

Typical Readiness Criteria

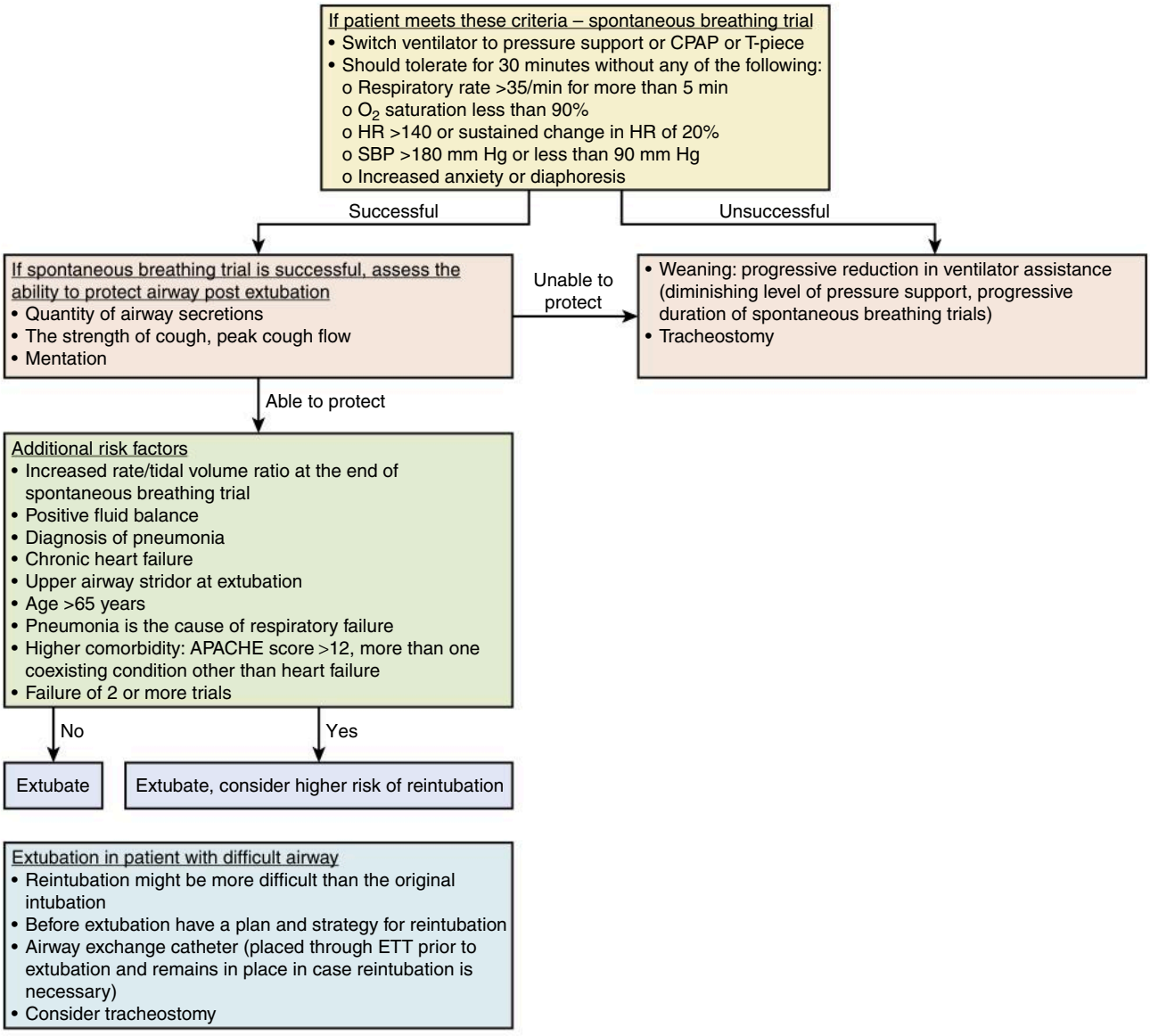
- Hemodynamic stability
- Ratio of PaO_2 (mm Hg) to FiO_2 >200 with PEEP of 5 or less
- Improvement in underlying condition causing respiratory failure

Other criteria

- Improved clinical status
- Adequate oxygenation
- pH 7.33–7.48 with acceptable $PaCO_2$
- Respiratory rate (RR) of 25 or less
- Vital capacity of 10 mL/kg or more
- Maxim inspiratory pressure force <–25 cm H_2O
- TV >5 mL/kg

Ratio of RR (breaths/min) to TV (liters) 105 or less during 1-minute trial with T-piece (also called rapid shallow breathing index or RSBI)

PEEP, Positive end-expiratory pressure.

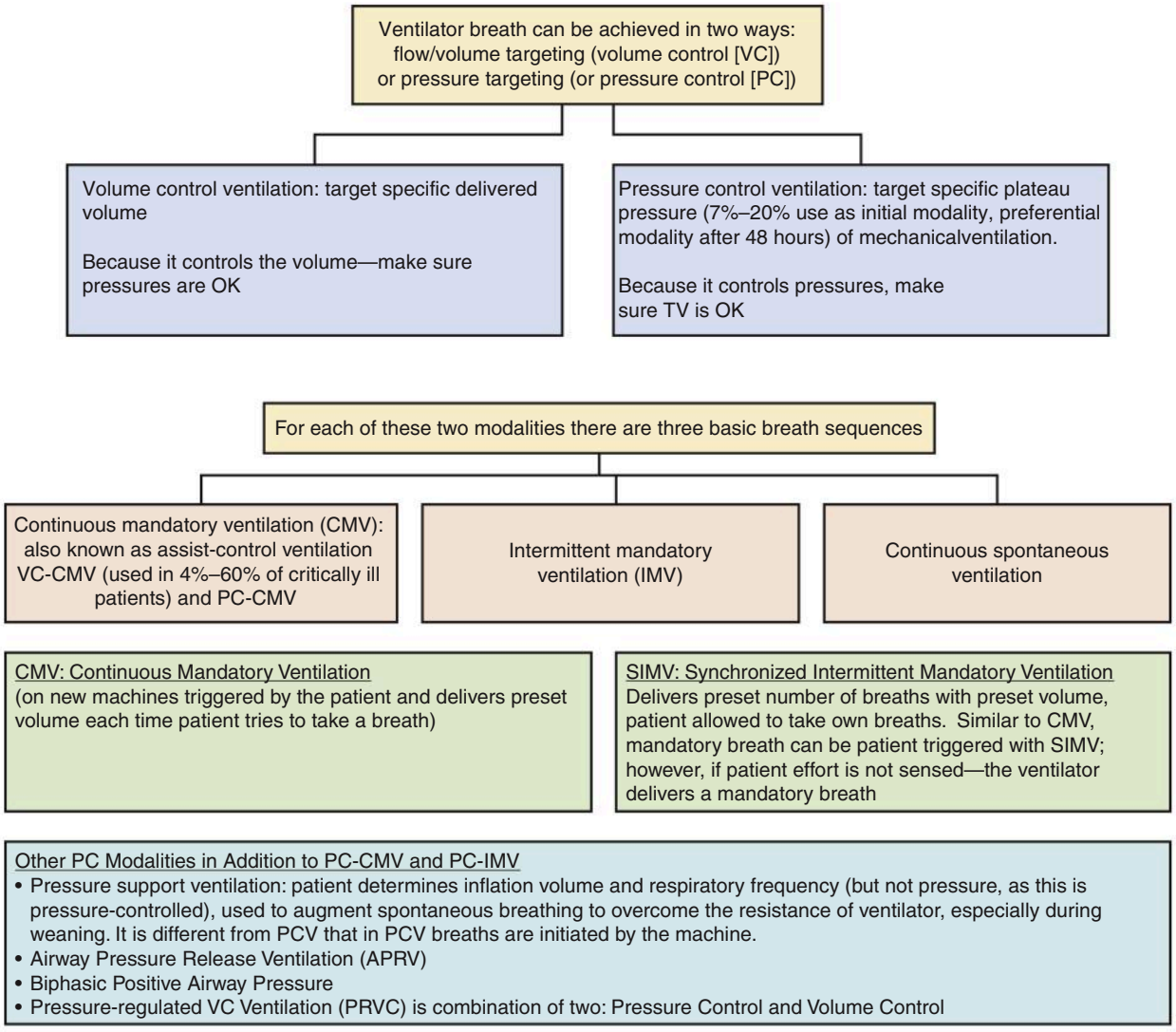


Details of Mechanical Ventilation

Ventilation Modalities

The ventilation modality is determined by three factors[26]:

- Trigger: spontaneous (describes the patient effort) or machine (machine or patient initiates the breath): assist control (AC) or intermittent mandatory ventilation (IMV). Variable flow shapes indicate IMV.
- Target: pressure control (PC) (pressure is the target) or volume control (VC). If various pressures are in different breaths, then it is volume controlled; if volume is different in different breaths, it is pressure controlled (pressure should be stable in each breath).
- Cycle: what turns the breath off: time, volume, or flow criteria/pressure.



Other PC Modalities in Addition to PC-CMV and PC-IMV

- Pressure support ventilation: patient determines inflation volume and respiratory frequency (but not pressure, as this is pressure controlled), used to augment spontaneous breathing to overcome the resistance of ventilator, especially during weaning, or to augment spontaneous breathing. It is different from PCV in that in PCV breaths are initiated by the machine.
- Airway pressure release ventilation (APRV)
- Biphasic positive airway pressure
- Pressure-regulated VC ventilation (PRVC) is combination of two: PC and VC

PC-CMV, Pressure control–continuous mandatory ventilation.