Chapter 4
Clinical Examination and Assessment

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4.1 Importance of Clinical Examination

Clinical examination is the first step in both establishing a diagnosis and immediate institution of therapies. The first step in establishing the diagnosis of respiratory disease is appropriate interpretation of clinical findings. Respiratory symptoms and signs may occur not only due to respiratory diseases but also other systems that impact on the respiratory system.

Within the first few minutes of an encounter, the patient can be classified into the following categories:

(1) The clinical examination shows that the patient is in extremis and in imminent danger of dying and requires immediate intervention
(2) The clinical examination shows a serious situation that may require some treatments to be administered before a definitive diagnosis can be made
(3) The clinical examination shows a sick child but one has enough time to perform a detailed clinical exam as well as diagnostic assessment before any preliminary treatment is started

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(4) The clinical examination shows a clinically stable patient but with a problem that needs to be investigated without needing any preliminary treatment.

4.2 Respiratory Distress and Respiratory Failure

The term respiratory distress is used to indicate signs and symptoms of abnormal respiratory pattern with an increased work of breathing and discomfort. A child with nasal flaring, increased rate (tachypnea), increased (hyperpnea) or decreased (hypopnea) depth of respiration, chest wall retractions, stridor, grunting, dyspnea, or wheezing has respiratory distress. Taken together, the magnitude of these findings is used to judge clinical severity.

Nasal flaring is a nonspecific, but a relatively sensitive sign of respiratory distress especially in infants and newborns. Although teleologically intended to decrease airway resistance, flaring of ala nasi is relatively ineffective for that purpose. It is however, a very important sign to identify an infant in some form of distress.

Rate and depth of respiration Prominent tachypnea is a hallmark of children with decrease lung compliance observed in alveolar-interstitial disease. Elastic work of breathing (W_{Elast}), determined mainly by V_T is increased disproportionately compared to resistive work of breathing (W_{Resist}) which is determined mainly by the respiratory rate. Thus, rapid and shallow respirations minimize work of breathing in such situations. Diseases of resistance are associated with increased depth of respiration at relatively slower rates. Young infants, because of their softer chest wall, have greater W_{Elast} and therefore they tend to breathe rapidly in all diseases affecting the respiratory system. Tachypnea is also prominent in situations with stimulation as J receptors which are located in the alveolar walls close to pulmonary capillaries. They are activated by distention of pulmonary capillaries, and accumulation of interstitial fluid. Increased in depth of breathing is seen much more commonly in non-respiratory diseases such as a response to metabolic acidosis, anxiety, and abnormal CNS impulses. For reliable interpretation of tachypnea, the child should be observed in a relative position of comfort and an environment that is least anxiety provoking.

Stridor is a high-pitched, coarse sound, characteristically during inspiration. It is indicative of extra thoracic airway obstruction (laryngotracheitis, vocal cord paralysis etc.) which gets worse during inspiration. In such a situation, the high negative intrathoracic pressure created to overcome the obstruction causes the extra thoracic airway to collapse below it. Inspiration is prolonged when this occurs.

Wheezing is a high-pitched, musical sound mostly heard during exhalation (but may also be during inspiration) in intrathoracic airway obstruction. The equal pressure point (EPP) is moved distally causing widespread intrathoracic airway collapse and prolongation of exhalation.

Rhonchi are low-pitched wheezes that are a result of pulmonary secretions in the larger bronchial airways. They are often biphasic in nature. Air flowing past the loose secretions creates a sound of "gurgling or rattling" throughout the lung fields.

Crackles or Rales are discontinuous "faint popping" sounds heard during inspiration. The sound auscultated is a result of opening of air-fluid menisci in the late generation bronchi. When crackles are heard earlier in the inspiration phase it is indicative of fluid in the larger airways (congestive heart failure), whereas crackles later in the inspiratory phase that are fine or high-pitched are associated with fluid in the smaller airways (pneumonia). Pathologies associated with crackles/rales often have poor lung compliance secondary to increased lung water, decrease alveoli surface area, and surfactant dysfunction.

Chest wall retractions are a manifestation of increased negative intrathoracic pressure during inspiration. They can also be observed, even in normal conditions, in children with weak chest walls. Excessively high negative intrathoracic pressures are generated in extra thoracic airway obstruction which worsens during inspiration and diseases of poor compliance where higher pressures are needed to maintain V_T. In newborns, especially those born prematurely, a pattern of paradoxical or see-saw respiration is observed where the chest caves in and abdomen bulges out during inspiration and the opposite occurs during exhalation.

Grunting is the child's attempt to maintain lung volumes without alveolar collapse. Grunting creates positive pressure during exhalation by partial closure of the glottis. This to a) maintain FRC in alveolar-interstitial disease to counter hypoxia and b) decrease transmural pressure and displacing the EPP more proximally to minimize airway collapse during exhalation in intrathoracic airway obstruction. Grunting can also be a manifestation of general distress as associated with pain and sepsis.

Other signs are useful in localizing the site of pathology as described below. Respiratory failure is defined conceptually as the inability to maintain adequate oxygenation or ventilation while breathing spontaneously in room air. Inability to adequately oxygenate results in hypoxemia, which is defined as an arterial oxygen tension (PaO_2) less than 60 mmHg or an arterial oxyhemoglobin saturation of < 90%. Hypoxemic respiratory failure refers to hypoxemia resulting from respiratory disease. Hypercarbia is defined as an increase in arterial carbon dioxide greater than 45 mmHg. Hypercarbic respiratory failure refers to hypercarbia and respiratory acidosis due to respiratory disease while breathing spontaneously. Therefore, whereas respiratory distress is a clinical assessment, the diagnosis of respiratory failure requires measurement of indices of gas exchange.

Diseases characterized by CNS excitation, such as encephalitis, and neuroexcitatory drugs are associated with central neurogenic hyperventilation. Similarly, diseases that produce metabolic acidosis, such as diabetic ketoacidosis, salicylism, and shock, result in hyperventilation and hyperpnea. Patients in either group could present clinically with respiratory distress; they are distinguished from patients with respiratory disease by their increased chest expansion per breath (i.e., tidal volume) as well as the respiratory rate. Patients with neuromuscular diseases, such as Guillain-Barré syndrome or myasthenia gravis, and those with an abnormal respiratory drive can develop severe respiratory failure but are not able to mount sufficient effort to appear in respiratory distress. In these patients, respirations are ineffective or can even appear normal in the presence of respiratory acidosis and hypoxemia.

Caveats:

1. Respiratory distress can occur in patients without respiratory disease
2. Respiratory failure can occur in patients without respiratory distress.
3. Hypoxemia can occur due to reasons other than respiratory disease (e.g., cyanotic heart disease)
4. Hypercarbia can occur without respiratory disease (e.g., diuretic use, excess bicarbonate due to acetate administration)

4.3 Characterizing Severity of Disease with Clinical Examination

Clinical examination of the respiratory system includes inspection, palpation, percussion and auscultation. Inspection of the respiratory system requires observation of respiratory rate, rhythm, pattern and effort of breathing. Auscultation of the lungs may reveal rales/crackles, stridor or wheeze. These can also be graded for severity based on the duration and whether they occur during one phase or both phases of breathing. There are scoring systems such as the croup score or the asthma score which offer a way to quantify the severity of the respiratory distress. The Table 4.1 shows a method to quantify the severity based on the respiratory signs and its effect on gas exchange and other systems.

4.4 Using Clinical Signs to Locate Site of Pathophysiology

While breathing rate, depth of respiration, presence of retractions, stridor, wheezing, and grunting are valuable in evaluating the severity of respiratory distress, they are also very useful in localizing the site of respiratory pathology (Table 4.2). Rapid

Table 4.1 Clinical signs with differing degrees of respiratory distress

Clinical sign	Mild	Moderate	Severe
Respiratory rate	+	++	+++
Retractions	Subcostal	Subcostal + Intercostal	Subcostal + intercostal + suprasternal
Asynchrony	None	Mild Asynchrony	Paradoxical Breathing
Stridor	None	Inspiratory	Inspiratory + Expiratory
Wheezing	None	Expiratory	Expiratory + Inspiratory
Gas exchange	SpO2 > 92% in RA	SpO2 > 92% with FiO2 < 0.3	SpO2 < 92% with FiO2 > 0.3 and < 0.5
Mental status	Awake	Restless	Decreased

Table 4.2 Interpreting the clinical signs of respiratory disease

Sign	Extra-thoracic airway obstruction	Intrathoracic-extrapulmonary airway obstruction	Intrapolmonary airway obstruction	Parenchymal pathology
Tachypnea	+	+	++	++++
Stridor	++++	++	–	–
Retractions	++++	++	++	+++
Wheezing	±	+++	++++	±
Grunting	±	±	++	++++

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and shallow respirations (tachypnea) are characteristic of parenchymal pathology. Chest wall, intercostal, and suprasternal retractions are most striking in extra-thoracic airway obstruction as well as diseases of decreased compliance. Inspiratory stridor is a hallmark of extra- thoracic airway obstruction. Expiratory wheezing is characteristic of intrathoracic airway obstruction, either extrapulmonary or intrapulmonary. Grunting is produced by expiration against a partially closed glottis in small airway obstruction (bronchiolitis) to maintain a higher positive pressure in the airway during expiration, decreasing the airway collapse. Grunting is most prominent in alveolar-interstitial disease to help maintain FRC.

4.5 Presentation Profiles of Respiratory Failure in Childhood

When mechanical dysfunction is present (by far, the most common circumstance), arterial hypoxemia and hypercapnia (and hence, pH) are sensed by peripheral (carotid bodies) and central (medullary) chemoreceptors. After being integrated with other sensory information from the lungs and chest wall, chemoreceptor activation triggers an increase in the neural output to the respiratory muscles, which results in the physical signs that characterize respiratory distress. When the problem resides with the respiratory muscles (or their innervation), the same increase in neural output occurs, but the respiratory muscles cannot increase their effort as demanded; therefore, the physical signs of distress are more subtle. Finally, when the control of breathing is itself affected by disease, the neural response to hypoxemia and hypercapnia is absent or blunted and the gas exchange abnormalities are not accompanied by respiratory distress (Fig. 4.1).

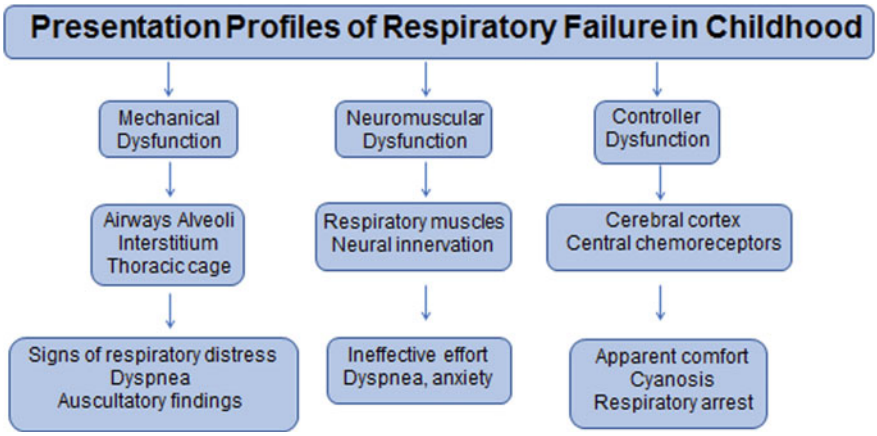

Fig. 4.1 Respiratory failure in children may present in different forms depending on the type of dysfunction

4.6 Respiratory Distress Without Respiratory Disease

Although respiratory distress most frequently results from diseases of lungs, airways, and chest wall, pathology in other organ systems can manifest as respiratory distress and lead to misdiagnosis and inappropriate management (Table 4.3).

Table 4.3 Non-pulmonary causes of respiratory distress

System	Example (s)	Mechanism (s)
Cariovascular	Left-to-right shunt Congestive heart failure Cardiogenic shock	↑Lung blood/water content Metabolic acidosis Baroreceptor stimulation
Central nervous system	Increased intracranial pressure Encephalitis Neurogenic pulmonary edema Toxic encephalopathy	Stimulation of brain stem respiratory centers
Metabolic	Diabetic Ketoacidosis Organic academia Hyperammonemia	Stimulation of central and peripheral chemoreceptors
Renal	Renal tubular acidosis Hypertension	Stimulation of central and peripheral chemoreceptors Left ventricular dysfunction Pulmonary edema
Sepsis	Toxic shock syndrome Meningococcemia	Cytokine stimulation of respiratory centers Baroreceptor stimulation from shock Metabolic acidosis

Respiratory distress resulting from heart failure or diabetic ketoacidosis may be misdiagnosed as asthma and improperly treated with albuterol, resulting in worsened hemodynamic state or ketoacidosis. Careful history and physical examination provide essential clues in avoiding misdiagnosis.

Suggested Reading

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