Physiological Interactions

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CONTENTS

8.1 Introduction 109
8.2 Physiological Basis of Heart-Lung Interactions 109
8.3 Influence of the Moving Heart on Thoracic Organs 110
  8.3.1 Aorta 110
  8.3.2 Lung 112
  8.3.3 Coronary Arteries 114
  8.3.3.1 Optimal Reconstruction Window for Coronary Angio-Computed Tomography 116
8.4 Influence the Bolus of Contrast Media by Heart-Lung Interactions 117
  8.4.1 Effect of the Systemic Venous Return 117
  8.4.2 Effect of the Pulmonary Resistance 118
  8.4.3 Effect of Cardiac Output 118
  8.4.4 Effect of Circulatory Blood Volume 120
  8.4.5 Consequences in Designing Protocols 120
  8.4.6 Effect of Physiological Parameters on Pulmonary Perfusion 121
8.5 Interaction among Pulmonary, Systemic, and Lymphatic Circulations 122
8.6 Heart-Lung Interactions in Near-Normal Anatomical Conditions 124
References 127

8.1 Introduction

Improvement in both temporal and spatial resolution of MDCT has brought the ability to explore both the heart and lung within a single examination and opened the field of functional evaluation. However, interactions between heart and lung have long been identified as important physiological phenomena in pathology (Pinsky 2005). For the radiologist, they are involved in difficulties of interpretation in a MDCT thorax examination and in the understanding of observations in physiological normal or near-normal clinical situations. In this chapter, after a brief overview of physiological heart-lung interactions, we will overview the consequences in imaging the heart and lung with the effects of the beating heart on thoracic organs, those of respiratory maneuvers, and of cardiac functional parameters on vascular enhancement. Then, interactions among the pulmonary, systemic circulation, and lymphatic circulations in normal subjects will be considered. Finally, near-normal conditions, such as clinically silent right-left shunts by patent foramen ovale, deformities, or postoperative conditions, will be described in this physiologic perspective.

8.2 Physiological Basis of Heart-Lung Interactions

The basic physiological heart-lung interactions can be understood as the effects of lung volume variations on both the cardiac rhythm and function and are mainly explained by variations of pressure. Lung volume varies in a tidal fashion during spontaneous respiration. During inspiration, intrathoracic...
pressure decreases owing to the contraction of the respiratory muscles. Inflation induces immediate changes in autonomic output, causing cardiac acceleration (Glick et al. 1969). This is otherwise known as respiratory sinus arrhythmia, a normal responsiveness that is lost in diabetic peripheral neuropathy. Lung inflation to larger tidal volume (> 15 ml/g) increases heart rate by sympathetic withdrawal and reflex arterial vasodilatation (Pinsky 2005). Thus, the heart has the intrinsic ability to vary its rate in synchrony with ventilation. Changes, or the rate of changes, in myocardial wall stretch might alter intrinsic heart rate independently of autonomic tone (Bernardi et al. 1989).

Interactions between respiration and cardiac function can be understood based on the effects of changes in intrathoracic pressure and lung volume on both venous return and left ejection fraction. During spontaneous ventilation, venous return increases when intrathoracic pressure decreases, that is, during inspiration (Pinsky 1984a). When filling increases on the right side, less filling occurs on the left side. In addition, the pooling of blood in the pulmonary circulation decreases filling pressure on the left side of the heart. The reverse occurs during expiration, and an increase in intrathoracic pressure augments the left ventricular afterload (Pinsky 1984a). This phenomenon, called interventricular dependence, is anatomically in relation to the presence of the constraining pericardium and the fact that ventricles share the interventricular and interatrial septum. Interventricular dependence works both ways even if the right side of the heart is more vulnerable to compressive forces. Interventricular dependence is increased when ventricular volumes are increased, i.e., in dilated cardiomyopathies or when the pericardium is relatively resistant to stretch, such as the stiffer pericardium of constrictive pericarditis or increased intrapericardial pressure of tamponade. In this setting, increasing right ventricular volume shifts the intraventricular septum into the left ventricle and simultaneously decreases left ventricular diastolic compliance and end-diastolic left volume.

Sustained increase in intrathoracic pressure, as seen with the Valsalva maneuver, will eventually decrease aortic blood pressure and arterial pressure because venous return decreases. Hyperinflation compresses the heart between the expanding lungs (Butler 1983) and increases juxtacardiac intrathoracic pressure more than the lateral chest wall intrathoracic pressure.

Ventilation alters pulmonary vascular resistance by a process known as hypoxemic pulmonary vasconstriction. If regional alveolar PO2 decreases below 60 mmHg, local pulmonary vasomotor tone increases, then reducing blood flow. Decrease in end-expiratory volume promotes alveolar collapse, stimulating hypoxemic pulmonary vasconstriction. However, changes in intrathoracic pressure that occur without changes in lung volume, as may occur with obstructive inspiratory effort or Valsalva maneuver, will not alter pulmonary vascular resistance (Pinsky 2005, 1984b).

8.3 Influence of the Moving Heart on Thoracic Organs

The transmitted motion of the beating heart is the origin of many pitfalls in imaging the aorta, the coronary arteries, and the pulmonary vessels. This knowledge is essential for the radiologist since for obvious reasons of radiation dose, non-ECG-gated scans will continue to be performed. In addition, their occurrence is not entirely suppressed by using ECG gating.

8.3.1 Aorta

Pendular and circular aortic motion that can lead to false-negative or false-positive diagnoses of aortic dissection has long been identified (Batra et al. 2000). These artifacts are viewed as more or less prominent double contours of the vessels (Fig. 8.1) or as a hypodense curvilinear interface or a crescent-like thin flap along the wall of the aortic root or sometimes along the pulmonary artery trunk (Fig. 8.2). They mainly affect the aortic root and ascending aorta and the left anterior and right posterior quadrants and are of variable prominence (Batra et al. 2000; Ko et al. 2005). Intricate interferences among heart rate, aortic motion, and simultaneous rapid helical data volume acquisition by multiple-detector rows are involved in its origin. The occurrence of aortic motion artifacts is diminished in elderly patients by the age-related reduced distensibility of the aorta, after a previous surgery, and in pathologic situations, such as mediastinal masses and aortic aneurysms (Set et al. 1993).