Chapter 9
Thorax: Physiological Monitoring and Modeling for Diagnosis of Pulmonary Edema

Shabana Urooj
Gautam Buddha University, India

M. Khan
Jamia Millia Islamia, India

A. Q. Ansari
Jamia Millia Islamia, India

ABSTRACT

In this paper, the authors prove that variations in thoracic volumes are greatly responsive to the act of breathing (i.e., inspiration and expiration). These variations may be adopted for diagnosing various respiration related diseases and pulmonary edema. In this study, the authors present a method to estimate the thoracic volume non-invasively using anthropometric dimensions. The change in the geometry of thorax with the act of breathe is recorded by measuring the anthropometric parameters for nine healthy human subjects. The model based approach shows the extent of its sensitivity in terms of volumetric variations with the state of inspiration and expiration. Many deaths occur due to unavailability of health care and monitoring systems in rural areas and developing countries. The technique presented in this paper takes care of these situations and the volumetric estimation of thorax is independent of any instrumentation, expensive equipment, and clinical environment.

INTRODUCTION

A number of methods existing for the diagnosis in the area of diagnosis of Pulmonary Edema with and without the involvement (Urooj & Khan, 2010) of transthoracic electrical impedance measurement. The available literature leads to the systems which are capable to workout strictly in a clinical environment and dependent on a lot of instrumental set-ups. These set-ups may include electrode configurations, bio-impedance analyzers based on bulky and expensive electronic circuitry. Above all these set-ups are available only in urban and developed area. Model based approaches are also
encountered in the survey of literature but most of them are based on involvement of electrical impedance plethysmography and deep study of other input quantities. A simple cylindrical model is also discussed in subsequent section which is also based on a lot of physiological aspects of cardiac cycles. Keeping in view, all these points a non-invasive, simple and fairly adoptable model based technique is presented in this paper for diagnosis of various diseases. The considered methodology is sensitive to the act of breathe and hence able to estimate volume of thorax in the state of inspiration and expiration.

Pulmonary edema is fluid accumulation in the lungs, which collects in air sacs. This fluid collects in air sacs in the lungs, making it difficult to breathe. It leads to impaired gas exchange and may cause respiratory failure. According to medical dictionary it is defined as the edema of lungs usually resulting from mitral stenosis or left ventricular failure. Pulmonary Edema occurs in the same way that edema occurs elsewhere in the body. When the lungs expand and contract during normal breathing, they slide back and forth within the pleural cavity. To facilitate this, a thin layer of mucoid fluid lies between the parietal and visceral pleurae. The pleural membrane is a porous, mesenchymal, serous membrane through which small amounts of interstitial fluid carry with them tissue proteins, giving the pleural fluid a mucoid characteristic, which is what allows extremely easy slippage of the moving lungs. The total amount of fluid in each pleural cavity is normally slight, only a few milliliters. Whenever the quantity becomes more than barely enough to begin flowing in the pleural cavity, the excess fluid is pumped away by lymphatic vessels opening directly from the pleural cavity. Thus by analyzing the work of breathe the amount of fluid can be predicted in terms of thoracic volume variations.

BACKGROUND

In Kinnen, Kubicek, Hill, and Turton (1964) a cylindrical thorax model is constructed to investigate the origin of the impedance signal. The inner cylinder represents the blood volume of the heart and the primary arteriovenous system of the thorax. The medium inside the inner cylinder represented the lungs. In their model, the computed resistance for the inner cylinder was 495 \( \Omega \) and for the interspace 32 \( \Omega \). These values indicated that most of the current flux would tend to travel through the model’s lungs so that the origin of the impedance signal should be based primarily on the right ventricle. This is consistent with observations in patients with septal defects (Lababidi, Ehmke, Durnin, Leaverton, & Lauer, 1971). In these patients the cardiac output, measured by impedance plethysmography, correlates well with the blood flow in the pulmonary circulation. Sakamoto, Muto, Kanai, and Iizuka (1979) constructed an anatomically more realistic model, in which changes in vena cava, heart, lungs, aorta, and torso shape were investigated. The model permits an examination of the effect of conductivity changes of component structures on the measured impedance. The weakness in this work is that one does not know what quantitative changes in conductivity are brought about as a result of real or simulated blood circulation. Sakamoto, Muto, Kanai, and Iizuka (1979) also did studies with dogs and humans where they measured the isopotential lines on the surface of the thorax. Compared to the model studies, some practical experiments performed on animals gave different results concerning the origin of the signal. In Baker, Hill, and Pale (1974) an experiment performed on a calf in which the natural heart was replaced by an implanted prosthesis containing artificial right and left ventricles. Penney (1986)