

## ACKNOWLEDGEMENTS

First of all thank you to Allah the Almighty God for the blessing, generosity, courage and strength to complete this manuscript.

Secondly, thank you to all those involved in helping me make this dissertation a success.

I would like to thank my supervisors, Dr Nik Munirah bt. Nik Mahdi, Dr Mohd Shafie b. Abdullah, Lecturers and Consultants Radiologist of Department of Radiology, Universiti Sains Malaysia, Dr Mohd Ariff b. Abas, Head of Radiology Department, Hospital Raja Perempuan Zainab II as well as my co-supervisor Dr Mahamarowi b. Omar, Consultant Intensivist Department of Anaesthesiology and Intensive Care together with Dr Kamarul Imran b. Musa, Department of Community Medicine Universiti Sains Malaysia for their valuable guidance and continuous supervision in the conduct of this study.

My sincere appreciation also goes to all lecturers in the Department of Radiology Universiti Sains Malaysia and all staffs of the Department of Radiology, Hospital Universiti Sains Malaysia for their assistance rendered for the smooth running of this study.

My parents Ahmad Maulana b. Abdul Ghani, Halijah b. Din, my parents in law Zakaria b.Md. Nor and Nahariah Shaadan, for their endless encouragement and support throughout my life. My dearest husband who is also the co-researcher for this study, Dr Muhammad Habibullah and my lovely daughters Anis Maisarah and Farah Najihah, for their continuous support, patience and sacrifice, especially during the preparation of this manuscript.

## TABLE OF CONTENTS

<b>TITLE</b>	<b>PAGE NO.</b>
Acknowledgement	ii
Table of contents	iii
List of tables	viii
List of figures	ix
Abbreviations	x
Abstrak	xi
Abstract	xiv
 CHAPTER 1 : INTRODUCTION	 1
 CHAPTER 2 : LITERATURE REVIEW	 4
 2.1 Bedside Chest Radiograph	 4
2.1.1 Image quality in computed radiography versus conventional radiography	4
2.1.2 Role of daily chest radiographs in pulmonary oedema	6
 2.2 Vascular Pedicle Width (VPW)	 7
2.2.1 Introduction	7
2.2.2 Anatomy of vascular pedicle	7

2.2.3 Measuring the Vascular Pedicle	9
2.2.4 Variation of VPW in pulmonary oedema	11
2.2.5 Potential Modifying Factors in Determining VPW	13
2.2.5.1 Physiological factors affecting VPW	13
2.2.5.1(a) Respiration	13
2.2.5.1(b) Gravity	14
2.2.5.2 Technical factors affecting VPW	14
2.2.5.2 (a) PA versus AP position	14
2.2.5.2 (b) Tube to film distance	15
2.2.5.2 (c) Kilovoltage	15
2.2.5.2 (d) Rotation	15
2.3.4 Usefulness of Serial Measurements of VPW	17
2.3 Cardiothoracic Ratio (CTR)	19
2.3.1 Definition	19
2.3.2 Usefulness of Cardiothoracic Ratio in pulmonary oedema	22
2.4 Pulmonary oedema	23
2.4.1 Introduction	23
2.4.2 Type of Pulmonary Oedema	23
2.4.3 Radiological Detection and Differentiation of Oedema	24
2.4.4 Radiological Factors to Determine Type of Oedema	25

2.4.4.1 Distribution of Pulmonary Blood Flow	25
2.4.4.2 Distribution of Pulmonary Oedema	27
2.4.4.3 Lung Volumes	28
2.4.4.4 Septal lines, Peribronchial Cuffing, Air Bronchograms, and Pleural Effusions	29
2.4.4.5 Heart size	29
2.4.5 Value of Pulmonary Artery Catheterization in pulmonary oedema and VPW	30
2.4.6 Treatment of Pulmonary Oedema	31
2.4.6.1 Medical treatment	32
2.4.6.1 (a) Frusemide	32
2.4.6.1 (b) Morphine	32
2.4.6.1 (c) Nitrates	33
2.4.6.1 (d) Other drugs for pulmonary oedema	34
2.4.6.2 Noninvasive ventilation	34
 CHAPTER 3 : OBJECTIVES	 35
3.1 General Objective	35
3.2 Specific Objectives	35
3.3 Research Hypothesis	36
 CHAPTER 4 : METHODOLOGY	 37
4.1 Study Design	37
4.2 Sample size calculation	37

4.3 Study technique	38
4.3.1 Inclusion criteria	38
4.3.2 Exclusion criteria	38
4.4 Validation Study	40
4.5 Chest radiograph	41
4.6 VPW and CTR measurement	42
4.7 Net fluid balance	44
4.8 Treatment	44
4.9 Statistics	45
 CHAPTER 5 : RESULTS	 46
5.1 Socio-Demographic Characteristics of Participants	46
5.2 Participants treatment	46
5.3 Analysis Correlation VPW, CTR and Net Fluid Balance	51
5.4 Analysis Mean Changes of VPW, CTR and Net Fluid Balance	53
 CHAPTER 6 : DISCUSSION	 59
6.1 Overview and Demographic characteristics	59
6.2 Correlation between VPW, CTR and Net Fluid Balance	61
6.3 Mean Changes of VPW and Net Fluid Balance between Day 1 to Day 2 and Day 2 to Day 3	66
6.4 Limitation of Study	67
6.5 Recommendation	69

CHAPTER 7 : CONCLUSION	71
REFERENCES	72
APPENDICES	78
Appendix A: Data collection sheet	
Appendix B: Borang maklumat dan keizinan pesakit	

## LIST OF TABLES

TABLE	TITLE	PAGE NO.
Table 5.1	Demographic Characteristics	47
Table 5.2	Pearson Correlation Analysis of Vascular Pedicle Width (VPW) and Independent Variables (CTR and Net Fluid Balance) in 51 patients.	52
Table 5.3	Mean VPW, CTR, and Net Fluid Balance in day 1, day2 and day 3.	54
Table 5.4	Paired t- test analysis mean change of VPW, CTR, and net fluid balanced on between day 1 to day 2 and between day 2 to day 3	55

## LIST OF FIGURES

FIGURE	TITLE	PAGE NO.
Figure 2.1	Diagram of vascular pedicle	8
Figure 2.2	Measuring of Vascular pedicle width	10
Figure 2.3	Variation of vascular pedicle width in lung oedema	12
Figure 2.4	Measurement of cardiothoracic ratio	21
Figure 2.5	Distribution of pulmonary blood flow	26
Figure 2.6	Regional distribution of lung oedema.	
27Figure 2.7	Pulmonary blood volume	28
Figure 4.1	Measurements of VPW and CTR	43
Figure 5.1	Pie chart shows gender distribution of patients involved in this study	48
Figure 5.2	Pie chart shows race distributions of patients involved in this study	49
Figure 5.3	Bar chart shows type of treatment given to patient in the study	50
Figure 5.4	Error bar shows significant reduction of net fluid balance in pulmonary oedema patient in consecutive days with treatment.	56
Figure 5.5	Error bar shows significant reduction of mean VPW in pulmonary oedema patient in consecutive days with treatment	57
Figure 5.6	Error bar shows no significant changes of mean CTR in pulmonary oedema patient in consecutive days with treatment. and combination treatment (IV frusemide and dialysis) from day 1 to day 3.	58



## **ABBREVIATIONS**

VPW	Vascular pedicle width
CTR	Cardiothoracic ratio
ICU	Intensive Care Unit
CXR	Chest radiograph
CPAP	Continuous positive pressure ventilation
BIPAP	Bi level positive pressure ventilation
PA	Posteroanterior
ARDS	Acute Respiratory Distress Syndrome
PAOP	Pulmonary artery occlusion pressure
ALI	Acute Lung Injury
PAC	Pulmonary artery catheter
ISDN	Isosorbide dinitrate
SD	Standard deviation

**KAJIAN PROSPEKTIF BERKENAAN HUBUNGKAIT DI ANTARA ‘VASCULAR PEDICLE WIDTH’ DAN NISBAH KARDIOTORASIK DI KALANGAN PESAKIT DEWASA YANG MENGHIDAP PARU-PARU BERAIR (PULMONARY EDEMA) YANG MENERIMA RAWATAN DI UNIT RAWATAN RAPI**

Dr Siti Aishah Ahmad Maulana

Sarjana Perubatan Radiologi

Jabatan Radiologi

Pusat Sains Perubatan, Universiti Sains Malaysia

Kampus Sihat, 16150 Kelantan, Malaysia

**Pengenalan:** Mengenali status aras kandungan cecair di dalam badan di kalangan pesakit-pesakit kritikal adalah amat penting. Mengikut tradisi, penggunaan pemantauan tekanan hemodinamik yang invasif seperti ‘pulmonary artery catheter’ telah digunakan untuk menilai status aras kandungan cecair di dalam badan di kalangan kumpulan pesakit kritikal ini. Namun maklumat yang boleh diperolehi daripada ‘vascular pedicle width’ (VPW) pada radiografi dada yang diambil dalam posisi baring di unit rawatan rapi adalah sangat menjimatkan dan kurang invasif untuk mengenal pasti status kandungan cecair di dalam badan pesakit-pesakit yang kritikal. Penggunaan VPW dalam menunjukkan kandungan cecair di dalam badan dilihat dalam beberapa kajian VPW daripada radiografi dada terutamanya dalam memantau pesakit yang menghadapi kelebihan kandungan cecair di dalam badan dan menerima rawatan.

**Objektive:** Untuk menentukan hubungkait di antara ‘vascular pedicle width’(VPW), nisbah kardiotorasik dan jumlah baki kandungan cecair badan dengan menggunakan radiografi dada bersiri di kalangan pesakit-pesakit dewasa yang disahkan menghidap paru-paru berair yang menerima rawatan di unit rawatan rapi di Hospital Universiti Sains Malaysia.

**Metodologi:** Kajian ini adalah kajian prospektif yang dilaksanakan daripada Jun 2008 sehingga Jun 2009. Ia melibatkan 51 orang pesakit yang disahkan menghidap penyakit paru-paru berair (pulmonary oedema) di unit rawatan rapi Hospital Universiti Sains Malaysia. Radiografi dada bersiri dalam posisi menelentang diambil dari hari pertama bermulanya penyakit paru-paru berair di unit rawatan rapi sehingga hari ketiga. Radiografi dada yang pertama diambil sebelum rawatan diberi. Radiografi dada yang berikutnya diambil setiap 24 jam sehingga hari ketiga. Data 24 jam baki kandungan cecair badan untuk tiga hari berturut-turut bagi setiap pesakit diambil daripada carta pemantauan pesakit mengikut hari radiografi dada diambil. VPW dan nisbah kardiotorasik dikira oleh penyelidik pada waktu berbeza tanpa makluman tentang data klinikal pesakit yang berkaitan.

**Keputusan:** Lima puluh satu pesakit terlibat di dalam kajian ini. Di mana nilai purata umur adalah 54.43. Seramai 39 pesakit (76.5%) menerima rawatan intravena Frusemide sahaja, manakala 12 pesakit (23.5%) menerima gabungan rawatan intravena Frusemide dengan dialisis sebagai rawatan paru-paru berair. Didapati terdapat hubungkait yang lemah tetapi tidak signifikan di antara VPW dan nisbah kardiotorasik pada hari pertama ( $r_1 = 0.10$ ,  $p = 0.34$ ), hari kedua ( $r_2 = -0.01$ ,  $p = 0.92$ ) dan hari ketiga ( $r_3 = 0.02$ ,

$p=0.91$ ). Keputusan yang sama dilihat di antara VPW dan jumlah baki cecair badan iaitu hubungan yang lemah tetapi tidak signifikan pada hari pertama ( $r_1=0.10$ ,  $p=0.47$ ), hari kedua ( $r_2= -0.05$ ,  $p= 0.73$ ) dan hari ketiga ( $r_3= -0.05$ ,  $p= 0.74$ ). Namun dengan menggunakan ujian t-berpasangan (paired t-test) menunjukkan perubahan yang signifikan terhadap purata perubahan VPW di antara hari pertama dengan hari kedua dan di antara hari kedua dengan hari ketiga ( $p < 0.001$ ). Perubahan yang signifikan juga dilihat pada purata perubahan jumlah baki cecair badan di antara hari pertama dengan hari kedua dan di antara hari kedua dengan hari ketiga ( $p < 0.001$ ). Tetapi tiada hubungan yang signifikan dilihat pada purata perubahan nisbah kardiotorasik di antara hari pertama dengan hari kedua dan di antara hari kedua dengan hari ketiga ( $p=0.58$ ). Tambahan pula, terdapat penurunan setiap hari purata VPW dan purata jumlah baki cecair badan dalam tempoh 3 hari dengan intravena Frusemide dan juga rawatan kombinasi.

**Kesimpulan:** Purata perubahan yang signifikan dilihat pada VPW dan jumlah baki cecair badan di antara hari pertama ke hari kedua dan di antara hari kedua ke hari ketiga sebaliknya tiada purata perubahan yang signifikan dilihat pada nisbah kardiotorasik di antara hari pertama ke hari kedua dan di antara hari kedua ke hari ketiga.

Dr Nik Munirah Nik Mahdi: Supervisor

Dr Mohd Shafie Abdullah: Supervisor

Dr Mohd Ariff Abas: Co-Supervisor

Dr Mahamarowi Omar: Co-Supervisor

# **A PROSPECTIVE STUDY OF RELATIONSHIP OF VASCULAR PEDICLE WIDTH AND CARDIOTHORACIC RATIO IN ADULT PULMONARY OEDEMA PATIENTS DURING TREATMENT IN ICU**

Dr Siti Aishah Ahmad Maulana

MMed Radiology

Department of Radiology

School of Medical Sciences, Universiti Sains Malaysia

Health Campus, 16150 Kelantan, Malaysia

**Introduction:** Determination of intravascular volume status in critically ill patient is very important. Traditionally, invasive haemodynamic pressure measurements have been used to assess the volume status in this group of patients. However, the information available from vascular pedicle width (VPW) in portable supine chest radiographs are least costly and least invasive of determining volume status in critically ill patient. The true utility of VPW reflecting intravascular volume status seen in few studies from portable supine chest radiographs especially in monitoring patients who have volume overload and are receiving treatment.

**Objectives:** To determine the relationship of vascular pedicle width (VPW), cardiothoracic ratio (CTR) and net fluid balance by using serial portable supine chest

radiograph in adult pulmonary oedema patients during treatment in ICU in Hospital Universiti Sains Malaysia.

**Patients and Methods:** A prospective study was done from Jun 2008 until Jun 2009 involving 51 patients who had been diagnosed to have pulmonary oedema in Intensive Care Unit (ICU) and Neuroscience ICU, Hospital Universiti Sains Malaysia. Serial supine portable chest radiographs were taken from day 1 of the onset of pulmonary oedema in ICU until day 3 consecutively. First chest radiograph was taken before starting treatment. Subsequent chest radiographs were taken about 24 hours apart. Three consecutive 24 hours net fluid balance data were taken from ICU monitoring chart according to the day of CXRs. Computed chest radiograph is used for evaluation of the VPW and CTR. The VPW and CTR were measured by researcher in separated occasion without clinical data related to patient available.

**Results:** Fifty one patient involved in this study with the mean age of 54.43. Total of 39 patients (76.5%) received intravenous (IV) Frusemide and 12 patients (23.5%) received IV Frusemide in combination with dialysis as treatment of pulmonary oedema. There was a weak but not significant correlation between VPW and CTR in each day from day 1 ( $r_1=0.10$ ,  $p=0.34$ ), day 2 ( $r_2=-0.01$ ,  $p=0.92$ ) and day 3 ( $r_3=0.02$ ,  $p=0.91$ ). Similar findings of a weak but not significant correlation was also seen between VPW and net fluid balance on day 1 ( $r_1=0.10$ ,  $p=0.47$ ), on day 2 ( $r_2=-0.05$ ,  $p=0.73$ ) and on day 3 ( $r_3=-0.05$ ,  $p=0.74$ ). However by using paired t-test significant mean changes of VPW between day 1 to day 2 and between day 2 to day 3 ( $p<0.001$ ). Significant mean changes of net fluid balance were also seen between day 1 to day 2 and between day 2 to day 3 ( $p<0.001$ ). No significant mean changes of CTR seen between day 1 to day 2 and between day 2 to day 3.

( $p=0.58$ ). In addition, there were daily reduction of the mean of VPW and net fluid balance in 3 days duration with IV Frusemide and combination treatment. However, no significant difference between both treatments with the mean of VPW ( $p=0.099$ ) and net fluid balance ( $p=0.162$ ) in 3 days period.

**Conclusion:** This study showed that there was strong significant mean changes of VPW and net fluid balance between day 1 to day 2 as well as day 2 to day 3. However, no significant mean changes of CTR between day 1 to day 2 and day 2 to day 3.

Dr Nik Munirah Nik Mahdi: Supervisor

Dr Mohd Shafie Abdullah: Supervisor

Dr Mohd Ariff Abas: Co-Supervisor

Dr Mahamarowi Omar: Co-Supervisor

## CHAPTER 1 : INTRODUCTION

Assessment the intravascular volume status of clinically ill patients can be difficult, yet efforts to manipulate fluid balance occur daily in the intensive care unit (ICU). Patients often require invasive hemodynamic monitoring such as Swan-Ganz catheter or central venous catheter, which predisposed to misinterpretation and potential to produce harm. Therefore the ability to discriminate changes in fluid balance noninvasively would be of the highest clinical utility to the practicing intensivist.

Portable, supine chest radiographs (CXRs) remains one of the simplest examinations used at the bedside to evaluate the cardiopulmonary status of patients in the intensive care unit. It has been employed for more than a century and the most commonly used noninvasive tool for identifying and quantifying the severity of pulmonary oedema. It is not only because of the least risk and costs reduction, but also because they can be utilized in areas outside the ICU.

Vascular pedicle width (VPW) is a measurement of mediastinal silhouette of the great vessels from upright chest radiograph was first described by Milne and colleagues in 1984. Other previous studies did prove that VPW measurement is important and reliable in assessing the intravascular volume status especially in critically ill patients. Hiponik *et al*, 1986 reported a significant correlation between vascular pedicle widths of more than 68mm and the presence of hydrostatic pulmonary oedema. In our study, the concern is to find the relationship in serial measurement of VPW with cardiothoracic ratio and net fluid balance. Net fluid balance has been chosen because of it is intensively charted by the ICU nurses for 24 hours in which it reflects 24



hours fluid balance in a patient during treatment. A study by Pistolesi et al in 1984 of upright non mechanically ventilated patients undergoing cardiac catheterization demonstrated that VPW and total blood volume were significantly correlated. Another study by Haponik et al in 1986 in burn patients who received intravascular volume expansion, presence of widening of VPW on serial films preceded development of pulmonary oedema. However previous study by Martin et al in 2002 on serial measurement of VPW and CTR in 5 days duration showed that no correlation between these two variables.

The ability of VPW to distinguish various form of pulmonary oedema among supine critically ill patient had also been suggested by Thomason et al in 1998. They found the role of portable, supine, anteroposterior chest radiographs in differentiating acute respiratory distress syndrome (ARDS) from pulmonary oedema due to volume overload can improve by over 15%-30% by incorporating VPW and cardiothoracic ratio (CTR) into standard practice (Thomason *et al*, 1998). However, our study interest is among pulmonary oedema patients with volume overload state such as cardiac and renal failure patients rather than pulmonary oedema due to permeability oedema such as trauma or ARDS. A study by Milne *et al* in 1985 in 216 upright posteroanterior films , they concluded a widening of VPW (>53mm) was predictive of volume-overload states such as cardiac or renal failure whereas the VPW below 43mm was the most predictive of patients with an injury pattern of oedema,

Furosemide was chosen as main treatment in our study because it is a major and important treatment in pulmonary oedema patients in ICU setting and even in wards and Emergency Unit. This is preferred treatment for pulmonary oedema due to massive

diuresis (which able to assess from input/output fluid from the nurses sheet). Cotter *et al* in 2001 commented that intravenous loop diuretics have formed the mainstay of treatment for acute cardiogenic oedema since 1960s. Besides, a study by Martin *et al* in 2002 showed that significant reduction of VPW in treated patient with Frusemide but no significant changes of CTR from baseline during 5 days treatment period.

We hope that our study is a pioneer for the implementation of serial measurement of VPW in clinical setting which assessable from chest radiographs. A study by Ely *et al* in 2001 stated that the accuracy of radiologists impressions for differentiating pulmonary artery occlusion pressure ( $PAOP \geq 18\text{mmHg}$  from  $PAOP < 18\text{mmHg}$ ) could be increased to 70% by using Receiver Operating Characteristic (ROC) curve with cut offs of 70mm for VPW and 0.55 for CTR were derived.

We believed that appraisal of VPW and other radiographic signs should be incorporated into newly implemented, comparative studies of ICU costlier and /or more invasive technologies.

We did agree that if the intravascular volume status remains unclear after radiographic assessment using VPW, other invasive studies such as pulmonary catheter placement, echocardiogram with or without esophageal Doppler monitoring according to the patient's clinical status and risk or benefit considerations must be pursued.

## **CHAPTER 2: LITERATURE REVIEW**

### **2.1 BEDSIDE CHEST RADIOGRAPH**

Bedside chest radiograph plays a major role in patient care. Studies show that most request (76 - 94%) for bedside chest radiographs are valid (Wandtke JC, 1994).

Daily chest radiograph (CXR) are obtained in most ICU patients. In many hospital up to one third of all inpatient chest radiographs taken in ICU particularly in patients undergoing mechanical ventilation and patient who are hemodynamically unstable or who have had invasive procedures (Henschke et al, 1997). CXRs are the most commonly used non invasive studies to identify the presence, severity or change in pulmonary oedema in the ICU (Ely et al, 2001). Moreover Milne et al in 1985 had previously elucidated the relationship of VPW and CTR to the etiology of pulmonary oedema by using upright, posteroanterior CXR, but CXR obtained in critically ill patients usually taken in the supine and anteroposterior position like in ICU.

#### **2.1.1 IMAGE QUALITY IN COMPUTED RADIOGRAPHY VERSUS CONVENTIONAL RADIOGRAPHY**

Computed radiography (CR) systems are the common radiographic systems nowadays in medical application because of their reliability and flexibility to address a variety of clinical application and lower costs. It often requires fewer retakes due to inadequate exposure and the image acquisition is also faster. Besides ability to adjust image brightness and contrast in wide range of thickness in one exposure, unlike

conventional radiography which may require a different exposure and multiple films speeds in one exposure to cover wide thickness range in a component so it causes impairment in diagnostic accuracy. Furthermore, CR images can be enhanced digitally to aid in interpretation. CR system is also use to transfer the images to workstation by using picture archiving and communications systems (PACS).

The ability of CR to adjust the contrast with adequate resolution is very helpful in measuring vascular pedicle width which involved major vascular structures in the mediastinal region. Ishigaki et al in 1996 noted the improvement of contrast in CR help to visualize low contrast region including the mediastinum. In our study, portable CR of the chest was used and every radiograph was displayed for interpretation on workstation with computerized caliper used to achieve acceptable radiographic measurement of VPW and CTR.

### **2.1.2. ROLE OF DAILY CHEST RADIOGRAPHS IN PULMONARY OEDEMA**

There are some arguments for daily chest radiograph in critically ill patients especially in ICU. Miller *et al* in 2006 suggested daily chest radiographs are warranted in patients with unknown or presumably changing intravascular volume. Besides, these patients whose intravascular volume status is in question would potentially benefit with minimal risk (Miller *et al*, 2006). Over 60% of chest radiographs will result in an institution of diuretic therapy in pulmonary oedema patients (Marik *et al*, 1997).

Hydrostatic pulmonary oedema is associated with a larger CTR and increased in VPW as described by Thomason *et al* in 1997 that hydrostatic pulmonary oedema was found to correlate best using a VPW >63mm coupled to CTR >0.52. A study by Martin *et al* in 2002 showed significant reduction of VPW ( $p=0.04$ ) but no reduction in CTR in 5 days period. Thus, in our study concern was to monitor the measurement of VPW and CTR in pulmonary oedema patients serially for 3 consecutive days. In addition, Miller *et al* in 2006 concluded the utility of serial measurement of VPW depends upon standardized performance of routine, serial chest radiographs.

## **2.2 VASCULAR PEDICLE WIDTH (VPW)**

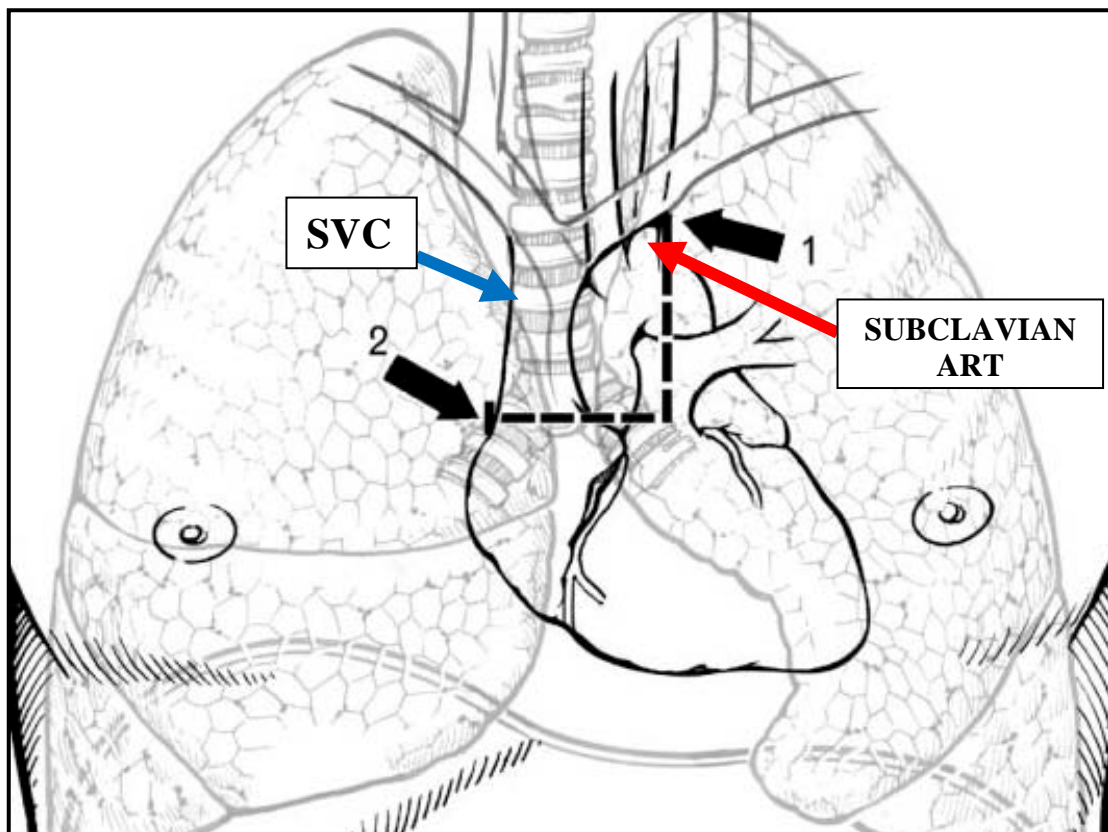
### **2.2.1 INTRODUCTION**

The technique of analysis of cardiac and pulmonary circulatory abnormalities on the chest radiograph has been described extensively. The fact that systemic vessels are readily seen on chest radiograph make the physician able to correlate changes in these vessels with alteration in the systemic circulation. Previous study by Milne et al in 1984 about the relationship between the size of the azygos vein, right atrial pressure and circulating blood volume showed that increased in circulating blood volume caused an increase in width of the azygos vein and the vascular pedicle.

### **2.2.2 ANATOMY OF VASCULAR PEDICLE**

On the frontal chest radiograph, the vascular pedicle extends from the thoracic inlet to the top of the heart. Its right-hand boundary is formed by the right brachiocephalic vein above and by the superior vena cava below. The left-hand border of the pedicle is formed by the left subclavian artery above the aorta. The right-hand border of the pedicle is entirely venous and lies quite far anteriorly. Whereas the left-hand border of the pedicle is usually arterial and lies quite posteriorly (Figure 2.1). The difference in level of the left and right boundaries is of considerable significance in determining how extravascular fluid collection within the pedicle will be presented radiologically (Milne *et al*, 1984).

A further point of diagnostic importance is that the veins are much more compliant than the arteries, and alterations in intravascular fluid volume will therefore be reflected by a much greater change in size of the right side of the pedicle compared to the left (Milne *et al*,1984).



**Figure 2.1: Diagram of Vascular Pedicle Width:**

The right border of the vascular pedicle is all venous component while the left border is all arterial component.

1: The point of subclavian artery origin which exit from aorta arch.

2: The point at which the SVC crosses the right main bronchus.

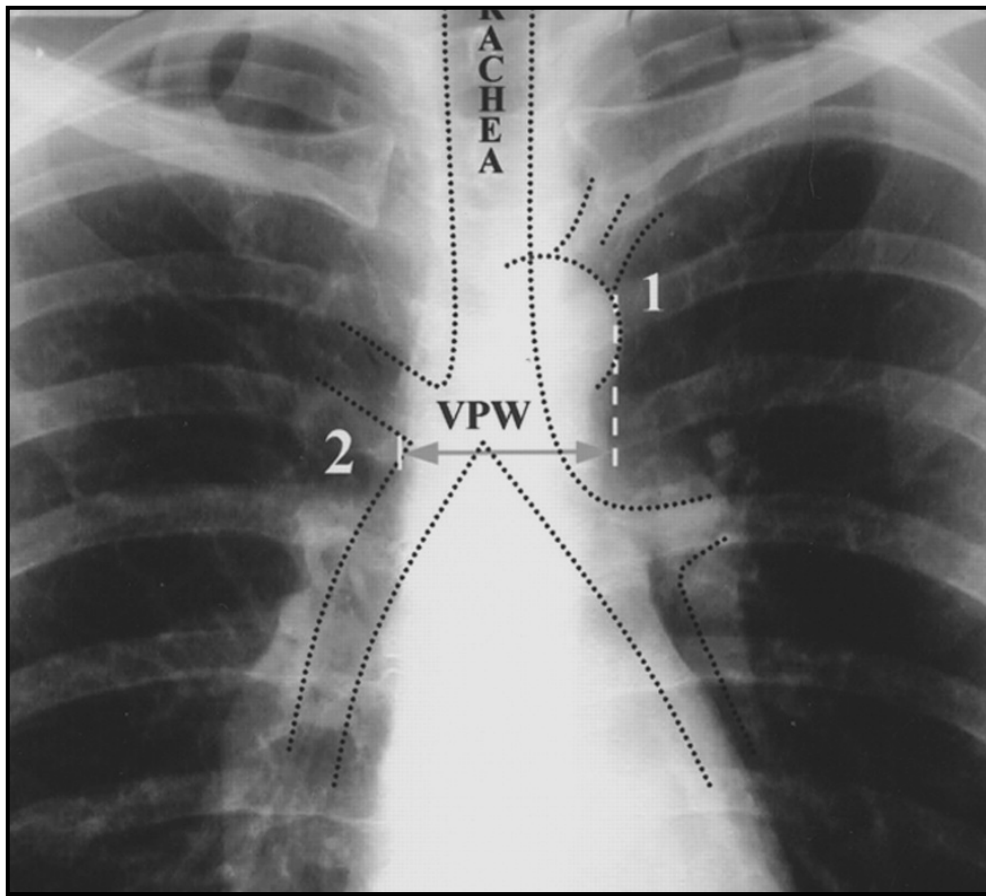
**Adapted from Milne et al, 1984.**

### 2.2.3 MEASURING THE VASCULAR PEDICLE

According to Milne *et al* in 1984 and 1985, as well as in recent publications by Ely *et al*, 2001, VPW is measured by dropping a perpendicular line from the point at which the left subclavian artery exits the aortic arch and measuring across to the point at which the superior vena cava crosses the right main stem bronchus (Figure 2.1). When the right border of the pedicle is indistinct, the vertical lateral border of the superior vena cava or right brachiocephalic vein have been used in measuring VPW (Ely *et al*, 2001).

Milne *et al* in 1984 reported that on the upright PA films, the normal VPW to be  $48 \pm 5$  mm, however in supine anteroposterior positioning VPW would increase to 58-64 mm or would increase by approximately 20%. A study by Thomason *et al* in 1998, the optimum cut off for VPW in distinguishing between permeability and hydrostatic pulmonary oedema was found to be 68 mm whereas a study by Salahuddin *et al* in 2007, prospectively measured the VPW among 50 adult patients in a tertiary center. VPW measurement correlated closely with positive fluid balance,  $r = +0.88$ ,  $P = 0.000$ . A Receiver Operating Characteristic curve demonstrated that a VPW of 86.5 mm had 100% sensitivity and 80% specificity for predicting fluid overload equal to greater than 1200ml. Another study by Ely *et al*, 2001, the best VPW cut-off for differentiating a high versus normal to low intravascular volume status on the supine, portable CXR was 70 mm regardless of the presence or absence of pulmonary oedema.





**Figure 2.2: Measuring Vascular Pedicle Width**

- 1) A perpendicular line from the point at which left subclavian artery exits arch of aorta.
- 2) A line across to the point at which superior vena cava crosses the right main stem bronchus.

VPW measures from point 1 to perpendicular dropped from point 2

**Adapted from Miller *et al*, 1984**

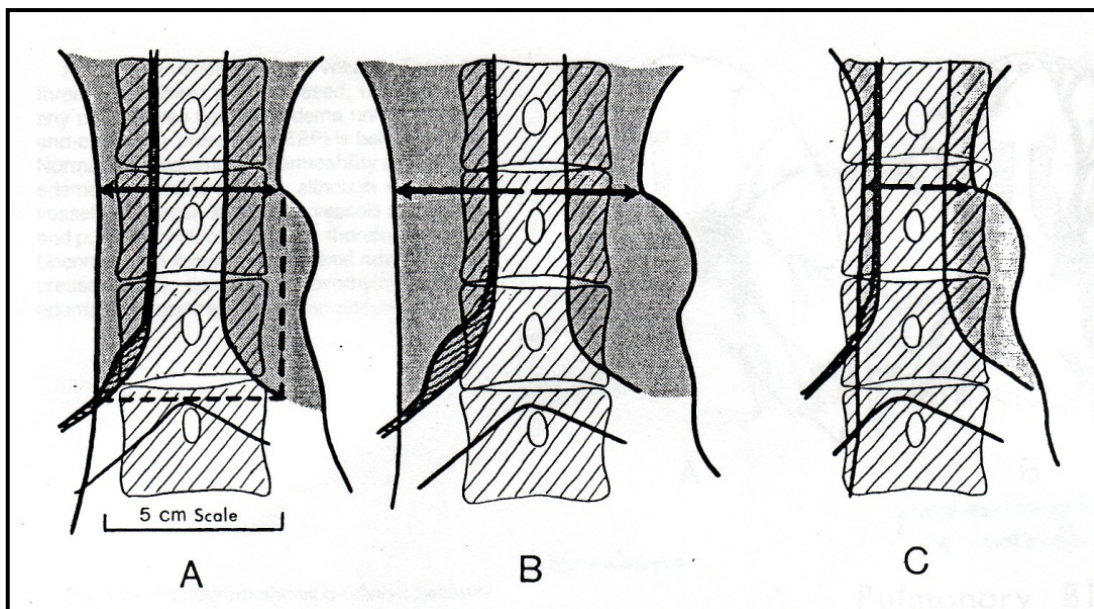
#### **2.2.4 VARIATION OF VPW IN PULMONARY OEDEMA**

A study by Milne et al in 1985, there are three variation of VPW in pulmonary oedema which diminished, normal and increased (for an erect 70 kg patient). Normal is defined as 43-53 mm commonest occurs commonly in capillary permeability and acute cardiac failure. Diminished or narrowed is less than 43mm, which occurs in capillary permeability oedema. Increased or widened is more than 53mm, this always seen in over hydration, renal failure and chronic cardiac failure. Another study by Figueras et al in 1978, VPW was greater than 53mm in 60% of cardiac failure patients, however 40% were in the normal range, this was due to acute cardiac failure or mitral valve disease cases had not involve in the increment of circulating blood volume, eventhough 60% of the patients were film in supine position, which increases VPW with an average of 20%. This correlate well with the changes of intravascular fluid volume in fluid overload condition such as in chronic cardiac failure and renal failure. There is blood congestion which reflect the greater change in the right side of pedicle compare to the left side (Milne et al,1984).

A study by Milne et al in 1980 showed 85% of widened vascular pedicles were in renal failure or overhydration patients. In their study also found that in capillary permeability oedema 35% of patients had a normal pedicle while 35% had a narrowed vascular pedicle. Moreover, the ability of VPW to distinguish various forms of pulmonary oedema among supine critically ill patients has been suggested by few groups. In 1998 Thomason *et al*, assessed the role of portable, supine, anteroposterior CXRs in differentiating acute respiratory distress syndrome (ARDS) from pulmonary

oedema due to volume overload. The accuracy of CXR interpretation could be improved by over 30% by incorporating the VPW and/or cardiothoracic ratio (CTR).

Another prospective study which rely upon volume status as measured by pulmonary artery catheters as compared with supine chest radiograph among ICU patients (by analyses of digital supine CXRs among 100 patients), VPW and CTR were confirmed by Ely *et al*, 2001.



**Figure 2.3: Variation of vascular pedicle width in lung oedema.**

- A. Normal- commonest in capillary permeability oedema.
- B. Widened- commonest in over hydration or renal failure.
- C. Narrowed- commonest in capillary permeability oedema.

**Adapted from Milne *et al*, 1984.**

### **2.2.5 POTENTIAL MODIFYING FACTORS IN DETERMINING VPW**

The vascular pedicle width is affected by various factors. They are divided to anatomical and physiological factors. In anatomical aspect VPW correlates well with body weight and surface area, but poorly correlates with height (Milne *et al*, 1984). The patient's build must be taken into account by radiologist in considering the width of vascular pedicle. A study by Milne *et al*, 1984, a 70kg subject of average build may have same VPW as a tall thin man or small fat one.

Another recent study was done by Azura *et al* in 2009 found that the mean weight of 140 ventilated ICU patients was 62.6 kg( $\pm$  18.1) with the mean of VPW of 64.4 mm ( $\pm$  10.6) which was smaller than other previous studies. They concluded that the possible cause was due to small body habitus of Asian population compared to Caucasian population. Moreover, there was statistically significant correlation was seen between the weight and VPW in their study ( $p < 0.001$ ) (Azura *et al*, 2009).

#### **2.2.5.1 Physiological factors affecting VPW**

Physiological factors affecting VPW are listed below, they include respiration and gravity.

##### **2.2.5.1(a) Respiration**

There is variation between inspiration and expiration. On inspiration the intrathoracic pressure becomes more negative, blood is pumped into the thorax and the

vascular pedicle become larger. On expiration, the intrathoracic pressure becomes more positive and blood is no longer pumped in, however VPW does not usually diminish because the diaphragm moves up, compacting the mediastenum and tending to widen the pedicle. The result of the two opposing factors is that the pedicle usually stay the same or change by only a few millimeters (Milne *et al*, 1984).

#### **2.2.5.1(b) Gravity**

Gravity plays a major effect in changing the width of VPW. Study by Milne *et al* in 1984 in 10 volunteers show VPW increased from 7 to 40% in supine position. While another study using 32 cardiac patients examined in erect position, VPW increased to  $21 \pm 7.3\%$ .

#### **2.2.5.2 Technical factors affecting VPW**

Few technical factors affecting VPW which are position, tube to film distance, kilo voltage and rotation. Assessment for the change of VPW size in serial radiographs can be a considerable clinical value (Pistolesi *et al*, 1984).

##### **2.2.5.2(a) PA versus AP position**

There is little appreciable change in width of the pedicle between the two positions whether PA or AP projection since one border of the pedicle lies anteriorly and other posteriorly (due to one or the other vascular border is always close to film).

Changing from the PA to AP position will cause a small geometric enlargement in VPW. (Milne *et al*, 1984).

#### **2.2.5.2(b) Tube to film distance**

On radiographs obtained at 40 inches; as opposed to the conventional 72 inches VPW would geometrically increase by approximately 5% (Milne *et al*, 1984). In other words, the closer the tube to the film, the larger the width of VPW.

#### **2.2.5.2(c) Kilovoltage**

The margin of vascular pedicle can usually be seen whether a radiograph is over penetrated or underpenetrated, however it is difficult to visualize the vein (specifically azygos vein) on an underpenetrated radiograph (Milne *et al*, 1984).

#### **2.2.5.2(d) Rotation**

The great vessels tend to be arranged partially around the circumference of a circle surrounding the trachea, rotation has a smaller effect on the apparent width of the pedicle. If the patient is turned to the left, the apparent VPW diminishes and if he is turned to his right the apparent VPW increases. On average turning the patient 15 degree to the left, decreases VPW by 6% and turning the same amount to the right increases VPW by 6% (Milne *et al*, 1984).

In addition to patient positioning and technical factors, some have raised concern that disease process or respiratory effort might affect the assessment of VPW. Indeed, when components of the silhouette have been altered by mediastinal disease, vascular engorgement, or the effects of prior trauma, radiation or thoracic surgery, the utility of VPW may be compromised (Miller *et al*, 2006).

Increased VPW may also occur in patients with extravascular bleeding. On the other hand, while elevated intravascular volume predominantly causes widening to the right of the midline, aortic injury causes widening predominantly to the left of midline, together with vanishing of the right paratracheal stripe and azygos vein.

### 2.2.6 USEFULNESS OF SERIAL MEASUREMENTS OF VPW

Serial measurement of VPW from portable CXRs has significant assessment of intravascular volume status. In a study by Pistolesi *et al*, 1984, of upright, non mechanically ventilated patients undergoing cardiac catheterization, the VPW and total blood volume were rightly correlated. A change in volume status correlated with the change in VPW (  $r = 0.93$ ,  $p < 0.001$ ). Furthermore, an increase in 1 L in total blood volume resulted in an increase of VPW by 5 mm when CXRs were serially obtained in upright position (Pistolesi *et al*, 1984).

A study by Pistolesi *et al* in 1984 among 42 patients with cutaneous burn, the patients preceded development of pulmonary oedema following fluid resuscitation as evidence of widening of vascular pedicle on serial films. They highlighted that development of pulmonary oedema occurred during first 3.3 days after injury was associated with widening of vascular pedicle by 69mm (Pistolesi *et al*, 1984) during initial 24 hours of fluid resuscitation. Patients who later developed pulmonary oedema had received more intravenous fluid (16.0L compared with 8.9L) during this period. Furthermore 12 of 13 patients who at least increased VPW by 1 cm later end up with pulmonary oedema.

A study by Martin *et al* in 2002, prospectively measuring changes in net fluid intake and in patient's weight and comparing these with serial VPW measurements. Among 133 portable supine chest radiographs in 36 mechanical ventilated patients with acute lung injury, they found that clinically detectable and significant reduction of VPW



over a 5 day period ( $p= 0.02$ ) was associated with a mean net diuresis of 3.3L and weight loss of 10kg.

Clinical application of these radiographic signs requires an awareness of potential confounding factors as mentioned before such as patient posture, radiographic technique and ventilator- patient interactions. Thus limitations imposed by these and other factors should encourage standardisation of interpreting portable chest radiograph (Meade *et al*, 2000). By this standardisation we can optimize pertinent, available information on our patients' films.

## **2.3 CARDIOTHORACIC RATIO (CTR)**

### **2.3.1 DEFINITION**

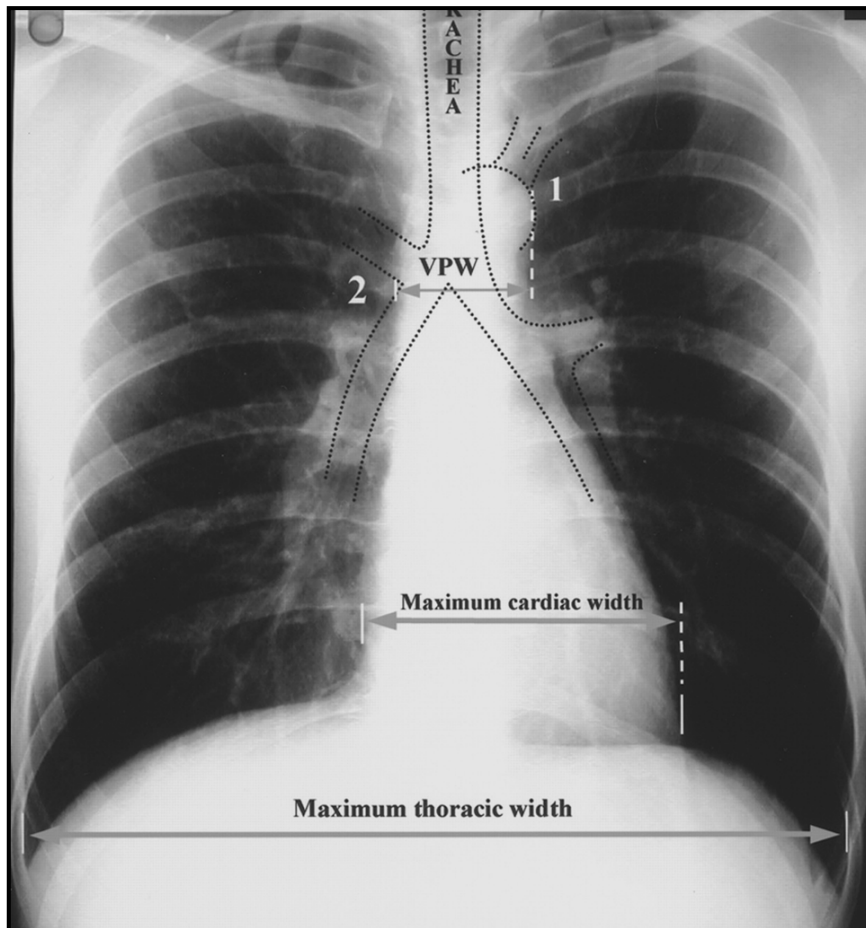
The cardiothoracic ratio was calculated as the ratio of the maximal transverse diameter of the cardiac silhouette ('heart diameter') to the distance between the internal margins of the ribs at the level of the right hemidiaphragm 'transverse thoracic diameter' (Figure 2.4) (Martin et al, 2002).

The upper limit of normal CTR is generally held to be around 50%. The bigger ratio may occur in some population such as among Africans and Asians which can be up to 55% (Grainger, Allison, 2002).

The most commonly used radiographic definition of cardiomegaly on the standard, upright, posteroanterior chest radiograph is a CTR over 0.5 (Badgett *et al*, 1997). Milne *et al* in 1998 determined the portable anteroposterior chest radiographs with focal film distance of 40 inches, a correct factor of 12.5% for CTR can be used to approximate whether or not cardiomegaly exists.

CTR is increased by nonstandard radiographic techniques such as poor inspiration, radiographs exposed supine, prone, anteroposterior or with a short focus-film distance. It is also influenced by age and build of the patient (Grainger, Allison, 2002).

A recent meta-analysis of 29 studies determined that cardiomegaly on the CXR was the best correlation for predicting a reduced ejection fraction, with a sensitivity and specificity of 51% and 79%, respectively (Badgett *et al*,1996). It is important to recognize confounding factors that can lead to false-positive interpretations of cardiomegaly, such as an apical fat pad, a transversely positioned heart, an expiratory film, or a decrease in the thoracic width (Badgett *et al*,1996). Portable films which are taken in the anteroposterior and supine position enlarge the appearance of the cardiac silhouette.



**Figure 2.4: Measurement of cardiothoracic ratio:**

Percentage of the heart size with its internal thoracic diameter. It is derived from dividing the widest transverse diameter of the cardiac silhouette by the widest transverse diameter of the internal thoracic above the diaphragm

**Adapted from Martin *et al*, 2002**

### **2.3.2 USEFULNESS OF CARDIOTHORACIC RATIO IN PULMONARY OEDEMA**

A prospective study by Thomason *et al* in 1998 obtained and evaluated chest radiograph in 33 supine, mechanically ventilated patients with pulmonary artery catheters. They used portable, anteroposterior supine chest radiograph in distinguishing hydrostatic pulmonary oedema from permeability pulmonary oedema. Their study showed hydrostatic pulmonary oedema associated with larger CTR, subjective impression of cardiomegaly and increased VPW. Hydrostatic pulmonary oedema was found to correlate best using a VPW of more than 63mm with CTR of more than 0.52. With this approach, the radiologists may have been able to improve their diagnostic accuracy to 73%. A recent study was done by Azura *et al* in 2009 showed that significant strong correlation of VPW and CTR in non serial chest radiographs in 140 ventilated ICU patients.

Another prospective study by Ely *et al* in 2001 by using portable, digital chest radiographs to determine the intravascular volume status. The result proved that VPW and CTR were the most important individual radiographic predictor of volume status (analysis used logistic regression and receiver operating curve).

## **2.4 PULMONARY OEDEMA**

### **2.4.1 INTRODUCTION**

Increased extravascular lung water is a common and sometimes life threatening clinical problem particularly in critical-care unit patients. There are four principle mechanisms of oedema formation which are increased hydrostatic pressure gradient across the capillary wall, diminished osmotic pressure gradient across the wall, increased capillary permeability (damage to the endothelial cell junction, which permits both fluid and large molecules to leak out of the vessels) and ability of the lymphatics to remove excess extravascular lung water (Milne *et al*, 1984). Pulmonary oedema cannot be detected at an early stage or quantitated accurately by physical examination alone (Ingram *et al*, 1980). Several non invasive techniques have therefore been developed, which attempt to detect oedema by assessment by its effects on the physical properties of the lungs (Gray *et al*, 1979).

### **2.4.2 TYPES OF PULMONARY OEDEMA**

There are two general different types of pulmonary oedema which are cardiogenic pulmonary oedema which is the most common type (also termed hydrostatic oedema commonly resulting from myocardial or valvular heart disease) and noncardiogenic pulmonary oedema (this is due to increased permeability pulmonary oedema) which can be caused by a wide variety of pathologic, trauma and infective condition resulting in injury to the pulmonary microvasculature (Milne *et al*, 1984). Non

cardiogenic pulmonary oedema also includes over hydration usually caused by an excess of saline effusion or renal failure.

### **2.4.3 RADIOLOGICAL DETECTION AND DIFFERENTIATION OF OEDEMA**

The ability of the radiologist to quantitate oedema from the chest radiograph was first demonstrated objectively by Milne *et al*, (1985). Chest radiograph is the only practical method of detecting pulmonary oedema at an early stage and following its evolution accurately. The validity of the radiographic method is now widely accepted by clinicians and physiologist (Staub NC, 1983). A recent regional study by Azura *et al* in 2009 in 140 ventilated patients in ICU found that a patient will have 1.2 time risk of having pulmonary oedema when there is an increment of VPW measurement by 0.17mm.

The distinct mechanisms of cardiogenic and noncardiogenic pulmonary oedema result in some moderate distinguishing findings on a posteroanterior or portable anteroposterior chest radiograph. A measurement of the width of vascular pedicle may improve the diagnostic accuracy of the chest radiograph, but its utility in distinguishing cardiogenic from noncardiogenic oedema needs further evaluation (Ely *et al*, 2002). There are several explanations for the limited diagnostic accuracy of the chest radiograph. Oedema may not be visible until the amount of lung water increases by 30 percent (Pistolesi *et al*, 1978). Any radiolucent material that fills the airway spaces (such as alveolar haemorrhage, pus, bronchoalveolar carcinoma) will produce radiographic image similar to that of pulmonary oedema. In many instances, clinical history, symptoms and signs and clinical course of the patient's disease will be sufficient to