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Original Article

# Determination of intravascular volume status in critically ill patients using portable chest X-rays: Measurement of the vascular pedicle width

**Nawal Salahuddin, M. Aslam, Ishtiaq Chishti[1], Shehla Siddiqui[2]**

**Abstract**

**Background and Aims:** Traditionally, invasive hemodynamic pressure measurements have been used to assess the volume status in critically ill patients. The vascular pedicle, as seen on chest radiographs, is the mediastinal silhouette of the great vessels. It is measured by drawing a perpendicular line from the point at which the left subclavian artery emerges from the aortic arch and measured across to the point at which the superior vena cava crosses the right main stem bronchus. We carried out this study to establish a correlation between the width of the vascular pedicle as seen on chest radiographs and daily fluid balance or pulmonary capillary occlusion pressure in mechanically ventilated, critically ill patients. **Materials and Methods:** In this prospective clinical study, 50 consecutive adult patients in a tertiary care, multidisciplinary intensive care unit underwent simultaneous measurements of the width of the vascular pedicle as seen on standardized, portable chest radiographs in the supine position and pulmonary capillary occlusion pressure measurements. Twenty-four hour fluid balance calculations were also recorded for each patient. **Results:** Vascular pedicle width measurements correlated closely with positive fluid balance, r = + 0.88, *P* 0.000. A Receiver Operating Characteristic curve demonstrated that a vascular pedicle width of 86.5 mm had a 100% sensitivity and an 80% specificity (area under the curve 0.823, 95% confidence intervals 0.714-0.932) for predicting fluid overload equal to and greater than 1200 ml. The correlation between pulmonary capillary occlusion pressures and width of the vascular pedicle was poor, r = 0.41, *P* 0.02. **Conclusions:** Our findings suggest that the vascular pedicle width on daily chest radiographs can be used to assess hypervolemia in the intensive care unit. Serial changes in the pedicle width could possibly be used to increase the accuracy of predictions.

**Key words:** Intravascular volume, critically ill patients, chest X-rays, vascular pedicle

## Introduction

Fluid overload in the critically ill patient has become recognized as an important predictor of increased mortality.[1] Due to overcrowding of intensive care units (ICU) and increased intensity of illness in patients presenting to the emergency departments,[2] a large number of critically ill patients are managed in acute care areas outside the ICU. Recent publications have shown the benefit of optimizing hemodynamics in the ER on mortality.[3]

Non-invasive measures are attractive; not only because of the obvious risk and costs reduction, but also because they can be utilized in areas outside the ICU. One such

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measure may be assessment of volume using the chest radiograph. The vascular pedicle, as seen on chest radiograph, is the mediastinal silhouette of the great vessels, i.e.; superior vena cava, azygous vein, thoracic aorta and the left subclavian artery. Measurement of this pedicle width was first described by Milne and colleagues in 1984.[4] As described, a perpendicular line is dropped from the point at which the left subclavian artery exits the aortic arch and measured across to the point at which the superior vena cava crosses the right main stem bronchus. This measurement is called the Vascular Pedicle Width (VPW). Milne *et al*, reported a ‘normal’ value of 48 ± 5 mm in healthy, upright volunteers undergoing PA chest X-rays.[4] Subsequent investigators have reported a significant correlation between vascular pedicle widths > 68 mm and the presence of hydrostatic pulmonary edema.[5-7]

The objectives of this study were to determine a correlation between the VPW and standard measures of fluid overload in the ICU such as 24h fluid balance and pulmonary capillary occlusion pressure (PCOP) as measured by the pulmonary artery catheter.

## Materials and Methods

### *Study design and setting*

This was a prospective observational study performed in the multidisciplinary ICU of a tertiary care, university hospital. The participating radiologist and critical care physician recording the primary variables (VPW and PCOP / daily fluid balance respectively) were kept blinded to each other’s measurements. The hospital Ethics Committee approved the study. No attempt was made to change routine ICU practices during the course of this study.

### *Patient selection*

Consecutive adult, critically ill patients on mechanical ventilation admitted to the intensive care unit and with an indwelling pulmonary artery catheter were enrolled. The period of study was from December 2005 to December 2006. Patients on a PEEP greater than 10 mmHg were excluded.

### *Measurements and radiographic technique*

Each enrolled patient underwent a chest radiograph, hemodynamic measurements and calculation of fluid balance over the past 24h, on day three of admission to the ICU. Central venous pressure and PCOP measurements were made independently by a single, board certified critical care physician according to the method described by Ely.[8] Data on daily fluid balance was collected from ICU nursing flow sheets. Conventional 14 x 17 inch portable, supine, antero-posterior chest radiographs were obtained on each patient within an hour of the hemodynamic measurements. The radiographic technique involved a 40-inch focal film distance, 60-70 kilovoltage peaks and a typical 3-6 mA exposure adjusted to patient body habitus. Each radiograph was processed in a standard rapid processor with a processor time of 45 seconds. We attempted to adjust for confounding due to variation in chest radiograph technique by limiting the inclusion to very strictly standardized films as regards to patient positioning, angle, ventilatory parameters, exposure, film distance and the quality of film. A radiologist recorded the vascular pedicle width according to the method described by Ely *et al*,[8] using the linear distance between two points; one at the origin of the left subclavian artery from the aortic arch and the second, the point of crossing of the superior vena cava over the right mainstem bronchus.

### *Statistical analysis*

Continuous measurements are expressed as Mean ±SD. Qualitative variables are expressed as percentages. Pearson’s correlation coefficient (r) was used to assess the linear relationship of the VPW with daily Fluid balance, PCOP and CVP. A ROC curve was used to estimate the discriminative value of a VPW measurement to correctly predict a positive fluid balance of ≥ 1200 ml. A $p$ value $< 0.05$ was considered as statistically significant. All $P$ values were two-sided. The Statistical package for social science (SPSS) version 11.5 was used for data analysis.

## Results

70 patients were enrolled from December 2005 to 2006. Only 50 patients were included in the final analysis due to technically unacceptable chest radiographs.

### *Clinical characteristics*

Mean age of enrolled patients was 54.2 ±13 years, 61% were males. At admission to the intensive care unit 20 (40%) were in shock and 25 (74%) had ARDS. Sepsis was the admitting diagnosis in 82.4% (28) and 11% (4) had head injuries. Acute renal failure developed in 46% (23) patients. The ICU Mortality rate was 36.7% (18).

### Measurements

Average fluid balance was + 1719 ml (± 1936.5). Forty patients (81.6%) were in positive fluid balance. Mean CVP, PCOP and VPW were 12 mmHg (± 4), 15.6 mmHg (± 5) and 77.6 mm (± 12.6) mmHg.

### Correlation analysis and ROC curve

Pearson correlation was performed between a) VPW and 24h Fluid balance, b) VPW and PCOP and c) VPW and CVP. The highest degree of correlation was found for VPW and 24h Fluid balance (Pearson Correlation coefficient r = + 0.887, significance level *P* 0.000, [Figure 1]).

Poor correlation was observed between VPW and PCOP (r = + 0.426, *P* 0.002, [Figure 2]) and CVP and VPW (r = + 0.32, *P* 0.025).

The ROC curve for the VPW measurements and fluid overload showed a quick rise and allowed for an optimal distinction between or < 1200 ml fluid overload at a VPW measurement of 86.5 mm, Sensitivity 100%, Specificity 80%, area under the curve 0.823, 95% CI 0.714 - 0.932 [Figure 3].

## Discussion

The main results of this study are that the VPW obtained from standard, portable chest radiographs in the supine position has an almost linear relation with fluid overload as measured by fluid balance. Also that the Pulmonary Capillary Occlusion Pressure (PCOP) may not be a reliable predictor of fluid overload a finding that has been corroborated by the results of other investigators.[9-12]

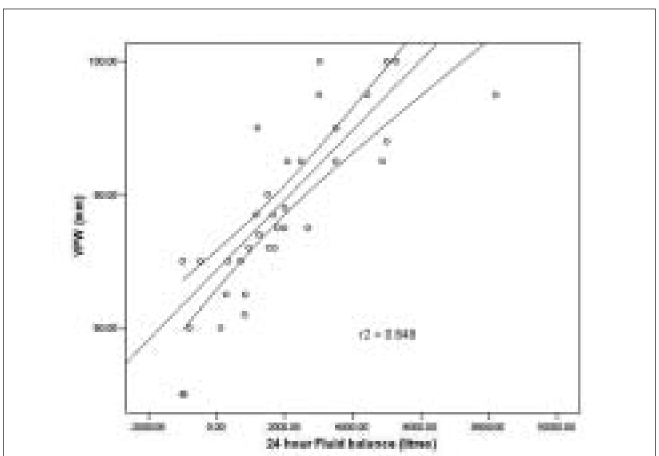


**Figure 1:** Relationship between vascular pedicle width on chest radiograph and net fluid balance in past 24 hours

The Azygous vein, Superior vena cava, Subclavian artery and Thoracic aorta together form the 'Vascular Pedicle'. In a study on 83 normal, healthy individuals with chest X-rays obtained in the upright position, a mean 'normal' vascular pedicle width of 48 ± 5 mm was reported.[4] Vascular Pedicle Widths appear to be unaffected by the respiratory cycle or by spontaneous or mechanical breaths.[4,8]

Milne *et al.*[4] and Pistolesi *et al*,[13] described widening of the mediastinal vascular pedicle in relation to changes in blood volume. These investigators suggested that widening of the pedicle occurs due to engorgement of the distensible venous structures, namely; superior vena cava and azygous vein. In a study on eight normal, male volunteers, Luft and colleagues reported on an enlargement of the central, mediastinal veins after sodium loading.[14] Haponik *et al*,[5] found that in patients with

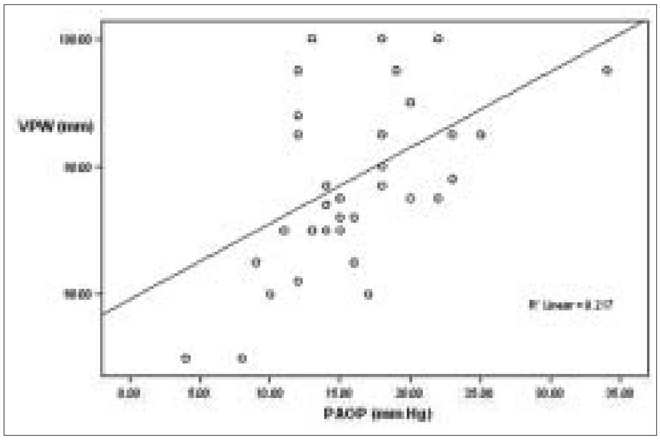


**Figure 2:** Relationship between vascular pedicle width and pulmonary artery occlusion pressure

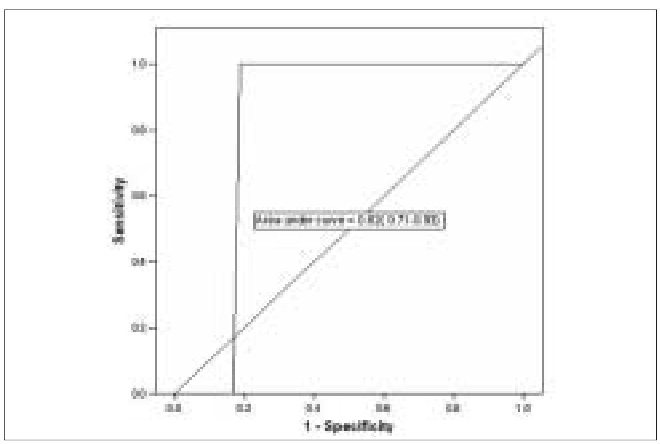


**Figure 3:** Receiver operating characteristics curve indicating the sensitivity and 1 - specificity of vascular pedicle width measurements in predicting a fluid overload of >1200 ml

burns, an increase in the vascular pedicle was seen after intravascular volume resuscitation.

For the past 20 years right heart catheterization with the pulmonary artery catheter, has been accepted as the traditional modality for hemodynamic assessment and management of the critically ill. The pulmonary capillary occlusion or wedge pressure is measured and interpreted as left ventricular filling pressure. In recent years, reports have challenged the benefits of the catheter.[15,16] In 1990, Iberti and colleagues presented large gaps in the understanding of physicians and nurses of how to correctly measure and utilize the information obtained by a pulmonary artery catheter.[17] An important study was by the FACTT study investigators in 1996. They reported the results of a randomized trial comparing haemodynamic management guided by a pulmonary artery catheter versus a central venous catheter in 1000 patients with established acute lung injury.[16] No difference in 60-day mortality was found between the PAC group and the CVC group.

Yet, recognizing and acting on fluid overload is extremely important. Increasingly, literature is available that discriminates between fluid-overloaded and 'dry' states, as predictors of survival in patients with acute lung injury /ARDS.[18] The FACTT trial[16] that randomized patients to either 'fluid-restriction' or conventional therapy reported that the conservative strategy of fluid management improved lung function and shortened the duration of mechanical ventilation and intensive care without increasing nonpulmonary-organ failures. Shoemaker and Kern in a meta-analysis reviewed 21 randomized, controlled trials and concluded a significant improvement in mortality when hemodynamics were optimized early on in the presentation of critical illness.[19]

So clearly the development of other less invasive technologies is warranted. In recent years preload assessment by intrathoracic blood volume by transpulmonary artery thermodilution, pulsed wave Doppler echocardiography, inferior vena cave collapsibility on inspiration, superior vena cava variation and respiratory variation in the aortic or pulmonary outflow Doppler signals.[20-26]

An alternate and cheaper direction is radiographic assessment. Chest radiographs are obtained almost every day in the ICU to confirm position of lines and tubes, assess for pulmonary edema, barotrauma, atelectasis or infection. Over 60% of chest X-rays will result in an institution of diuretic therapy[27] which suggests that clinicians already incorporate data from X-rays into a decision-making process.

Other investigators have also explored the VPW as a noninvasive predictor of hypervolemia. In 1998, Thomason and colleagues[6] published the results of a prospective study where they studied the utility of supine chest X-rays in distinguishing between hydrostatic pulmonary edema and permeability pulmonary edema. In 33, ventilated patients with various critical illnesses, they reported a significant correlation between the VPW and the pulmonary capillary occlusion pressure ($r = 0.45$, $P$ value 0.0076) with an ROC curve indicating a greater discrimination for VPWs between 63-70 mm (AUC 0.706). We observed similar results in our study. The correlation coefficient (r) between VPE and PCOP in our sample was 0.42, $P$ value 0.002 and we also observed a similar ROC curve with an optimal discriminative power of the VPW for hypervolemia at a measurement of 74.5 mm (AUC 0.724). Ely *et al.*,[8] in 2001 reported on a study of 100 mixed, critically ill, ventilated and non-ventilated patients, they reported that by combining the VPW (at a cutoff value of >70 mm) and a cardiothoracic ratio of >0.55, the likelihood ratio of the CXR in determining volume status increased to 3.1 (95% CI, 1.9-6). Martin[7] studied 133 portable, supine chest radiographs in 36 ventilated patients with acute lung injury/ ARDS. They noted that a clinically detectable reduction ($P = 0.02$) in the VPW associated with a 3.3L diuresis and mean weight loss of 10 kg over a five day period.

We were able to show similar results in our cohort of a mixed population of surgical and medical, critically ill, ventilated patients. Our results show that a cutoff value of 86.5 mm of the VPW has a high discriminative ability to predict high filling pressures as measured by a pulmonary artery catheter.

The VPW can be useful in predicting response to therapy. Haponik[5] described an increase in the vascular pedicle after volume resuscitation in patients with burns. Among a sample of 42 patients, those that had an increased VPW in the first 24h of resuscitation were more likely to develop pulmonary edema in the first 3.3 days after the injury. In

1990, a study on 36 pairs of CXRs taken on 22 stable, patients before and after hemodialysis demonstrated that the VPW decreased significantly with dialysis. They concluded that the VPW and transverse diameter of the heart may be useful in estimating dry weights in patients in whom clinical assessment is difficult.[28]

There are however various limitations in VPW for volume ascertainment that need to be kept in mind while making an assessment. Cardiac surgery, prior mediastinal irradiation and obesity can erroneously give a widened VPW, making it impossible to use the pedicle width for volume interpretation in these patients. The vascular pedicle width is susceptible to changes in posture, compression by mediastinal structures or positive pressure ventilation. Therefore, absolute numbers may be less reliable compared to daily changes in width that may correlate better with increase or decrease in intravascular volume. A limitation of our study is that we did not use serial chest radiographs and compared serial changes in the VPW with daily changes in weight or fluid balance.

## Conclusions

This prospective study of 50 mechanically ventilated patients demonstrates the high correlation between the VPW as measured on a standard portable chest radiograph and a usual predictor of hypervolemia such as fluid balance over the past 24h. A poor correlation was observed between the VPW and the PCOP, providing further support that pressure measured in the pulmonary capillary does not reliably predict volume responsiveness. We consider that our results are useful to most physicians attempting to decide on volume status in ventilated patients, since they illustrate the powerful predictive ability of a bedside chest radiograph.

## References


1. Alsous F, Khamiees M, DeGirolamo A, Amoateng-Adjepong Y, Manthous CA. Negative fluid balance predicts survival in patients with septic shock: A retrospective pilot study. Chest 2000;117:1749-54.
2. Sinuff T, Kahnamoui K, Cook DJ, Luce John M, Levy MM; Values Ethics and Rationing in Critical Care (VERICC) Task Force. Rationing critical care beds: A systematic review. Crit Care Med 2004;32:1588-97.
3. Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, *et al*. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med 2001;345:1368-77.
4. Milne EN, Pistolesi M, Miniati M, Giuntini C. The vascular pedicle of the heart and the vena azygos, Part 1: The normal subject. Radiology 1984;152:1-8.
5. Haponik EF, Adelman AM, Munster AM, Bleecker ER. Increased vascular pedicle width preceding burn-related pulmonary edema. Chest 1986;90:649-55.
6. Thomason JW, Ely EW, Chiles C, Ferretti G, Friemanis RI, Haponik EF. Appraising pulmonary edema using supine chest roentgenograms in ventilated patients. Am J Respir Crit Care Med 1998;157:1600-8.
7. Martin GS, Ely EW, Carroll FE, Bernard GR. Findings on the portable chest radiograph correlate with fluid balance in critically ill patients. Chest 2002;122:2087-95.
8. Ely EW, Smith AC, Chiles C, Aquino SL, Harle TS, Evans GW, *et al*. Radiologic determination of intravascular volume status using portable, digital chest radiography: A prospective investigation in 100 patients. Crit Care Med 2001;29:1502-12.
9. Michard F, Boussat S, Chemla D, Anguel N, Mercat A, Lecarpentier Y, *et al*. Relation between respiratory changes in arterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. Am J Respir Crit Care Med 2000;162:134-8.
10. Tavernier B, Makhotine O, Lebuffe G, Dupont J, Scherpereel P. Systolic pressure variation as a guide to fluid therapy in patients with sepsis-induced hypotension. Anesthesiology 1998;89:1313-21.
11. Tousignant CP, Walsh F, Mazer CD. The use of transesophageal echocardiography for preload assessment in critically ill patients. Anesth Analg 2000;90:351-5.
12. Kumar A, Anel R, Bunnell E, Habet K, Zanotti S, Marshall S, *et al*. Pulmonary artery occlusion pressure and central venous pressure fail to predict ventricular filling volume, cardiac performance or the response to volume infusion in normal subjects. Crit Care Med 2004;32:691-9.
13. Pistolesi M, Milne EN, Miniati M, Giuntini C**.** The vascular pedicle of the heart and the vena azygos: Part 2; aquired heart disease. Radiology 1984;152:9-17.
14. Luft FC, Klatte EC, Weyman AE, Bloch R, Rankin LI, Fineberg NS, *et al*. Cardiopulmonary effects of volume expansion in man: Radiographic manifestations. AJR Am J Roentgenol 1985;144:289-93.
15. Blumberg MS, Binns GS. Swan-Ganz catheter use and mortality of myocardial infarction patients. Health Care Financ Rev 1994;15:91-103.
16. National Heart, Lung and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network, Wiedemann HP, Wheeler AP, Bernard GR, Thompson BT, Hayden D, *et al*. Comparison of two fluid-management strategies in acute lung

injury. N Engl J Med 2006;354:2564-75.

17. Iberti TJ, Fischer EP, Leibowitz AB, Panacek EA, Silverstein JH, Albertson TE. A multicenter study of physicians' knowledge of the pulmonary artery catheter. JAMA 1990;264:2928-32.
18. Mitchell JP, Schuller D, Calandrino FS, Schuster DP. Improved outcome based on fluid management in critically ill patients requiring pulmonary artery catheterization. Am Rev Respir Dis 1992;145:990-8.
19. Kern JW, Shoemaker WC. Meta-analysis of haemodynamic optimization in high-risk patients. Crit Care Med 2002;30:1686-92.
20. Combes A, Arnoult F, Trouillet JL. Tissue Doppler imaging estimation of pulmonary artery occlusion pressure in ICU patients. Intensive Care Med 2004;30:75-81.
21. Kircher BJ, Himelman RB, Schiller NB. Noninvasive estimation of right atrial pressure from the inspiratory collapse of the inferior vena cava. Am J Cardiol 1990;66:493-6.
22. Viellard-Baron A, Prin S, Chergui K, Dubourg O, Jardin F. Haemodynamic instability in sepsis: Bedside assessment by Doppler echocardiography. Am J Respir Crit Care Med 2003;168:1270-6.
23. Fiessel M, Michard F, Mangin I, Ruyer O, Faller JP, Teboul JL. Respiratory changes in aortic blood flow velocity as an indicator of fluid responsiveness in ventilated patients with septic shock. Chest 2001;119:867-73.
24. Barbier C, Loubières Y, Schmit C, Hayon J, Ricôme JL, Jardin F, *et al*. Respiratory changes in IVC diameter are helpful in predicting fluid responsiveness in ventilated septic patients. Intensive Care Med 2004;30:1740-6.
25. Marx G, Cope T, McCrossan L, Swaraj S, Cowan C, Mostafa SM, *et al*. Assessing fluid responsiveness by stroke volume variation in mechanically ventilated patients with severe sepsis. Eur J Anaesthesiol 2004;21:132-8.
26. Godje O, Hoke K, Goetz AE, Felbinger TW, Reuter DA, Reichart B, *et al*. Reliability of a new algorithm for continuous cardiac output determination by pulse-contour analysis during haemodynamic instability. Crit Care Med 2002;30:52-8.
27. Marik PE, Janower ML. The impact of routine chest radiography on ICU management decisions: An observational study. Am J Crit Care 1997;6:95-8.
28. Don C, Burns KD, Levine DZ. Body fluid volume status in heamodialysis patients: The value of the chest radiograph. Can Assoc Radiol J 1990;41:123-6.

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