Central Venous Catheterisation

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Abstract

Central venous pressure (CVP) monitoring is an essential tool in the management of critically ill patients. It is usually considered as a measure of intravascular volume status, though it may not always be true. If used appropriately, various pathophysiological conditions can be diagnosed/confirmed from the morphology of CVP trace.

Introduction

The role of vascular access cannot be underestimated during the treatment of patients. Entrance into the central venous compartment is possible using different types of central venous access devices (CVADs). The placement of central venous catheter is needed for optimal management of sicker patients. Central venous pressure (CVP) is one form of invasive haemodynamic monitoring widely used all over world. Annually several million central venous catheters are inserted in critically ill patients.

Historical background

Forssmann in 1929 described central venous compartment cannulation for the first time by inserting plastic tubing through vein of his own forearm up to the heart[1]. In 1950s, Aubaniac was successful in cannulating subclavian vein using central venous catheter[2]. Sven-Ivar Seldinger in 1952 developed an innovative technique of percutaneous insertion of large bore catheters[3]. Hughes and Magovern (1959) first described the use of right atrial pressure monitoring in post thoracotomy patients and correlated fall in CVP after blood loss and CVP improvement after blood transfusion[4].

Conventionally central venous access devises are inserted by palpation or inspection of the needle insertion sites. Central venous catheterization can also be accomplished using ultrasonographic (USG) guidance (Fig.1) with resultant lesser complication rates in experienced hands. Because of direct visualisation of the puncture needle and the blood vessel to be entered, USG guided technique is better for coagulopathic patients[5].

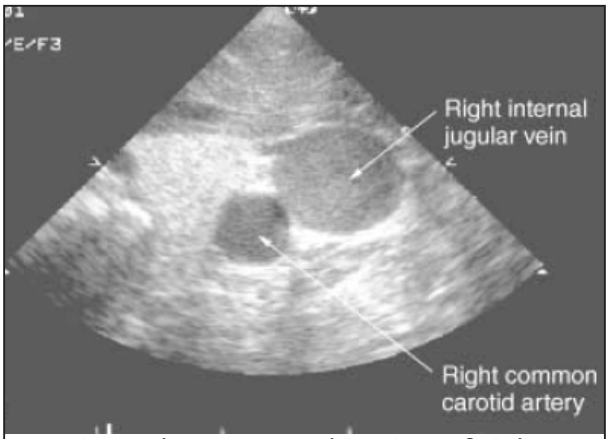

Fig1. Ultrasonographic view of right internal jugular vein and common carotid artery

Types of Central Venous Access Devices:

Peripheral Intravenous Central Catheter (PICC): (Fig.2)

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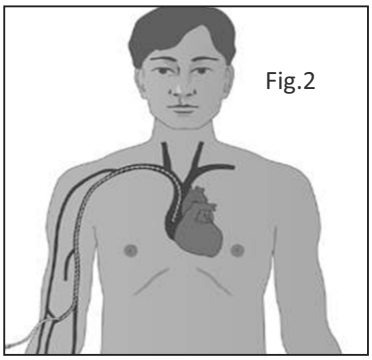


Fig.2

A PICC line is a type of long flexible tubing that is placed peripherally, usually in the antecubital or superficial saphenous vein and then threaded into a larger vein in the central part of the body. The end of the catheter that sticks out of the skin has a special cap. They are considered a mainstay of vascular access in neonatal intensive care units (NICUs). They are indicated in children who require intermediate-term IV access for prolonged home or hospital therapy.

Central Venous Catheter (CVC): (Fig.3)

Central Venous Catheter is inserted through internal jugular vein, subclavian "vein or less commonly femoral vein". CVCs are of different types. They are classified according to the number of lumens (1 to 4), type of catheter material (Teflon® or polyurethane, polyvinyl chloride or polyethylene), presence of in line filters, drug impregnation (Chlorhexidine/Silver sulfadiazine, Minocycline/Rifampin, Platinum/ Silver, heparin - bonded with benzalkonium chloride) etc. Teflon® or polyurethane and drug coated CVC have lesser incidence of infectious complications[6].

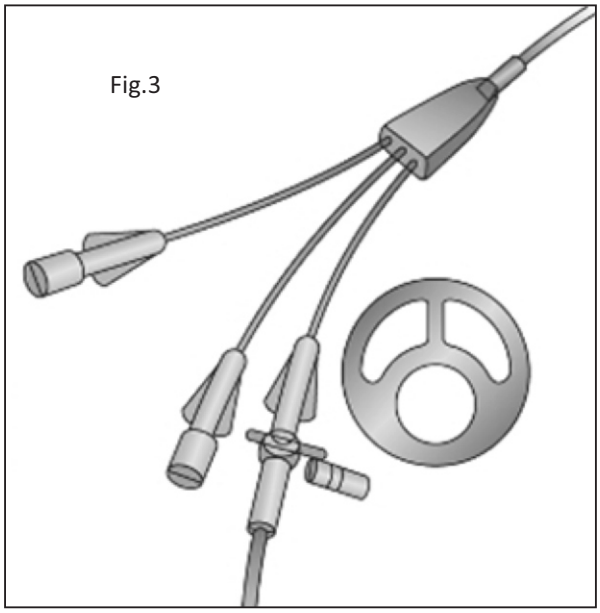
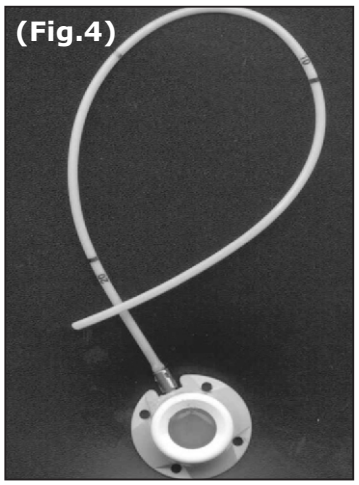


Fig.3

Implantable access ports (IAPs): (Fig.4)

Implantable access ports (Mediport and Port-A-Cath) are surgically inserted in operation theatre and are used for prolonged vascular access. Subclavian venous route is mainly favoured. Risk of infection and frequent dressing changes are minimised. Huber needle must be used to access port.


(Fig.4)

Haemodialysis Catheters:

These are large calibre two lumens catheters with or without additional small lumen. Internal jugular or femoral veins are commonly used. They allow large flows of blood to be withdrawn from one lumen, to enter the dialysis circuit, and to be returned via the other lumen. Non tunnelled catheters can be used up to 10 days. Catheters tunnelled subcutaneously may be used for few months till arterio-venous fistula or graft is in place.

Umbilical artery catheters and umbilical vein catheters:

Umbilical vascular systems are used in first few days of life. They can be used for arterial / central venous pressure monitoring, for medication administration or frequent sampling of blood in neonatal emergencies.

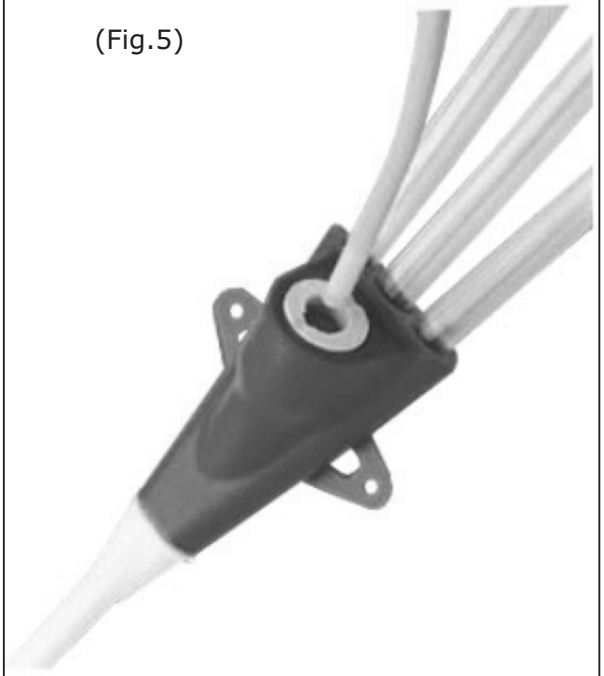
Introducer sheaths:

These are mainly used for insertion of pulmonary artery catheters. They can also be

used for rapid administration of fluids when needed. Internal jugular vein is commonest site for sheath insertion.

Advanced Venous Access Catheter (AVA): (Fig.5)

(Fig.5)



AVA consists of three large bore lumens and one introducer sheath. The lumens can be used for rapid fluid infusions or for haemodialysis. Sheath is used for insertion of pulmonary artery catheter.

Indications for Central Venous catheter insertion:

1) Central Venous Pressure monitoring
2) Pulmonary artery catheter insertion and pressure monitoring
3) Transvenous cardiac pacing
4) Intravascular fluid assessment when urine output is inadequate
5) Temporary haemodialysis
6) Vasopressors and other vasoactive drug administration
7) Total parenteral nutrition
8) Chemotherapy and administration of peripheral vein irritants
9) Rapid administration of large volumes
10) Aspiration of air emboli
11) Frequent blood sampling

Contraindications:

Absolute:

1) Infection/fresh burns at the site of insertion
2) Thoracic inlet / superior vena cava syndrome

Relative:

1) Coagulopathy
2) Newly inserted Pacemaker wires
3) Contra lateral diaphragmatic dysfunction*
4) Large goitre*
5) Previous neck surgery*

(*internal jugular vein cannulation avoided)

Complications of Central venous Catheterisation:

1) Mechanical

Vascular Injury

Arterial puncture (most common)

Venous laceration Cardiac chamber puncture (Haemopericardium, cardiac tamponade)

Haemomediastinum

Aortic puncture (rare)

Arterio venous fistula (rare)

Pseudoaneurysm formation (rare)

Respiratory problem

Pneumothorax

Subcutaneous and mediastinal emphysema

Haemothorax

Compression of trachea due to haematoma

Chylothorax (left IJV cannulation)

Tracheal perforation

Perforation of endotracheal tube cuff

Nerve Injury

Horner's syndrome

Brachial plexus injury

Phrenic nerve injury

Chronic pain syndrome Arrhythmias

2) Thromboembolic

Venous thrombosis

Pulmonary embolism

Arterial thrombosis and embolism

Guide-wire and catheter embolism

Superior vena cava syndrome

Air embolism

3) Infectious

Insertion site infection

Catheter infection

Bloodstream infection

Endocarditis

4) Misinterpretation of data

Data may be wrongly interpreted by the clinician or nursing staff.

Assembly for CVP measurement:

For CVP measurement some steps are to be followed.

1) Transducer preparation
2) Transducer zeroing
3) Transducer leveling
4) CVP measurement

Transducer preparation:

A sterile, disposable transducer is connected to electronic monitor. One pressurised normal saline is attached to proximal end of transducer through Leur lock system. A three-way bivalve is connected to the distal end of transducer. One pressure monitoring line is attached to one free end of bivalve. This whole assembly is flushed with normal saline from pressurised bag to remove air.

Transducer Zeroing:

The CVP measurement is done with reference to ambient pressure. Ambient pressure is equalled to transducer pressure. This is called as zeroing of the transducer. The transducer is opened to ambient air through remaining free end of the bivalve. Zeroing method described for the electronic monitor is followed. The monitor will show the transducer pressure as 'zero'.

Transducer levelling:

The level of the transducer should be at heart level. In supine position, mid axillary level corresponds to centre of right atrium. In semi-recumbent and sitting positions, sternal angle corresponds to level 5 cm above midpoint of right atrium. So the transducer should be levelled 5 cm below sternal angle.

CVP measurement:

The transducer is connected to proximal end of the distal lumen of the central venous catheter through pressure monitoring line. The system is deaired. CVP trace starts appearing on the monitor.

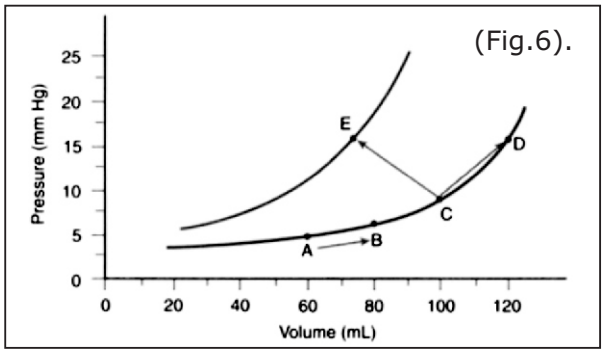
Central venous pressure (CVP) is the pressure within superior vena cava or right atrium and is considered as one of the filling pressures in heart along with pulmonary capillary wedge pressure (PCWP). It reflects the driving force for filling the right atrium and ventricle and is usually described as preload of heart.

CVP can be obtained by measuring distension of jugular vein on physical examination, with simple water manometer or by using sophisticated transducers and electronic monitors. Measurement of CVP is commonly used to serve as a guide to fluid balance though poor relationship with fluid responsiveness had also been described[7]. It is also used to estimate circulating blood

volume and to help in the management of circulatory failure.

Cardiac physiology and CVP monitoring:

According to Frank- Starling's principle, force of myocardial contractility is determined by end-diastolic fibre length of myocardium at any level of inotropy within physiological limit. End-diastolic myocardial fibre length depends on the filling volume (preload) of the heart chamber. Therefore filling volumes of ventricles determine their stroke volumes. Because cardiac filling volumes measurement is difficult in clinical practise, cardiac filling pressures are measured to monitor cardiac filling volumes. But caution should be exercised as their relationship is not always in same direction. In fact, the diastolic pressure-volume relationship in cardiac muscle is curvilinear, with a progressively steeper slope at higher volumes (Fig.6).



(Fig.6).

Above figure demonstrates the curvilinear relationship between ventricular diastolic pressure and volume. Increase in 20 ml volume from 60 to 80 (A to B) ml does not change the pressure in lesser steep portion of the curve whereas increased volume from 100 to 120 (C to D) ml causes rise of pressure from 8 to 15 mm of Hg in greater steep portion of the curve. Similarly, increase in pressure from 8 to 15 mm of Hg can cause increase (D) or decrease (E) in volume which in turn depends on the stiffness/compliance of ventricular myocardium. By definition, diastolic dysfunction is present when ventricular pressure is abnormally elevated for any given ventricular volume.

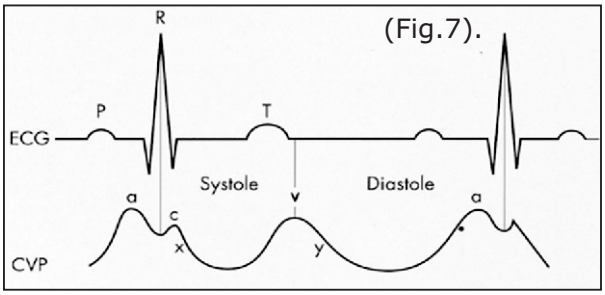
CVP also reflects functional capacity of right ventricle. Based on the Frank-Starling mechanism, higher right heart filling pressures are required to maintain ventricular stroke output when right ventricular contractility is impaired.

The transmural pressure (a difference between intracardiac and intrathoracic - extracardiac pressure) is responsible for the filling volumes/preload of ventricles. Anything that increases intrathoracic or intrapericardial pressure will decrease the venous return although CVP value may be higher.

Central venous pressure is a result of interaction between intravascular volume, ventricular compliance and transmural pressure.

Normal CVP waveform:

Normal mechanical events of the cardiac cycle are responsible for the sequence of waves seen in a typical CVP trace. The CVP waveform consists of five phasic events, three peaks (a, c, v) and two descents (x, y) (Fig.7).



(Fig.7).

The CVP waveform consists of three positive and two negative deflections (Table 1). The first most prominent wave is 'a' wave. It occurs in late diastole after P wave of ECG and is caused by atrial kick. The second positive deflection, 'c' wave is seen in early systole after R wave in ECG. It is generated during

Waveform Component	Phase of Cardiac Cycle	Mechanical Event
a wave	End-diastole	Atrial contraction
c wave	Early systole	Isovolumic ventricular contraction, tricuspid motion toward the right atrium
v wave	Late systole	Systolic filling of the atrium
h wave	Mid to late diastole	Diastolic plateau
x descent	Midsystole	Atrial relaxation, descent of the base, systolic collapse
y descent	Early diastole	Early ventricular filling, diastolic collapse

isovolumic contraction of ventricle because of increase in atrial pressure due to ballooning of tricuspid valve into right atrium. Atrial pressure falls further during ventricular systole because of continuous atrial relaxation producing 'x' descent. Last positive deflection is 'v' wave during late ventricular systole. It is produced by continuous atrial filling when tricuspid valve remains closed resulting in increase in atrial pressure. During early diastole, tricuspid valve opens and blood flows from atrium to ventricles leading to rapid fall in atrial pressure (diastolic collapse). This produces 'y' descent. In slower heart rates with elevated venous pressures, pressure plateaus during mid to late diastole occasionally producing 'h' wave.

Interpretation of Central Venous Pressure:

Normal CVP in an awake, spontaneously breathing patient ranges between 1 and 10 mm Hg (mean - 5 ± 3 mm Hg). Traditionally low CVP value is considered as intravascular fluid deficit and vice versa. However trend of the CVP values is of more clinical significance than a single value.

Abnormal CVP waveforms:

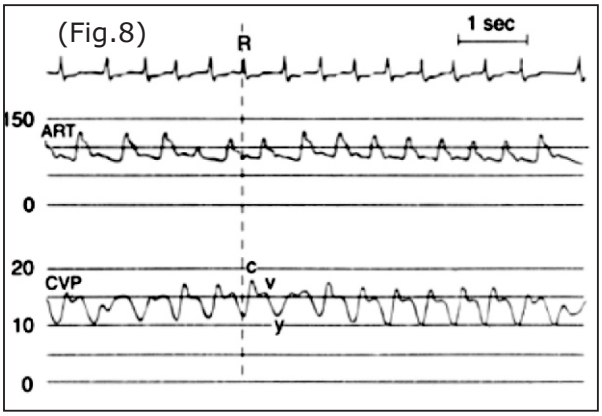
Morphology of the CVP waveform changes in different pathological conditions. Cardiac arrhythmias are most commonly diagnosed by quick examination of the CVP waveform morphology.

Atrial Fibrillation: (Fig.8)

- loss of 'a' wave

- prominent 'c' wave

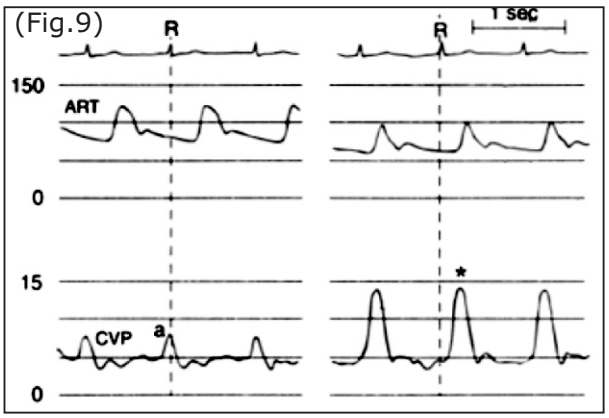
(Fig.8)



- because of larger volume of blood in atrium at end-diastole and early systole due to lack of Atrial kick

Atrioventricular dissociation or junctional rhythm: (Fig.9)

(Fig.9)



- cannon 'a' waves

- atrium contracts during ventricular systole when tricuspid valve is closed

Tricuspid regurgitation: (Fig.10)

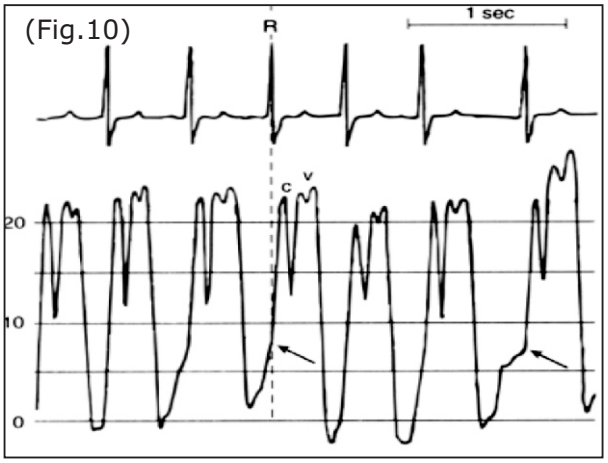
- tall systolic 'c-v' wave

- loss of 'x' descent

- abnormal filling of right atrium due to incompetent valve during ventricular systole.

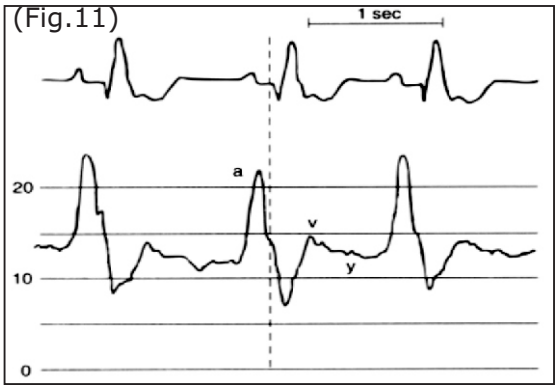
- resembles right ventricular pressure (Ventricularisation of CVP trace)

- right ventricular end-diastolic pressure overestimation, so should be calculated at 'R' wave of ECG before beginning of regurgitant



'c-v' wave.

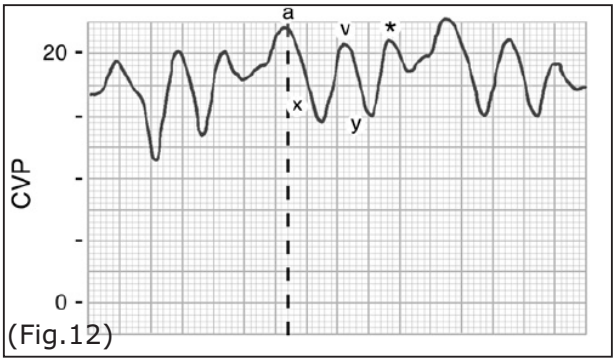
Tricuspid stenosis: (Fig.11)



- tall 'a' wave
- attenuation of 'y' descent
- pressure gradient across tricuspid valve throughout diastole
- mean CVP elevated
- slow and less diastolic emptying of blood from right atrium

Pericardial constriction: (Fig.12)

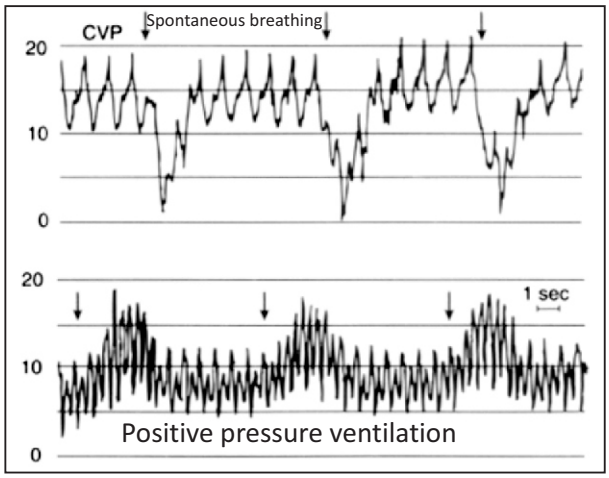
- tall 'a' and 'v' waves
- steep 'x' and 'y' descents
- M or W configuration
- mean CVP raised
- extramural compressive effect of the pericardium



Cardiac tamponade:

- dominant 'x' descent
- attenuated 'y' descent
- marked increase in CVP
- extramural compressive effect of effusion

Effects of respiration on CVP waveforms: (Fig.13)



During spontaneous breathing, intrathoracic pressure falls and it is transmitted in CVP waveform as fall in mean pressure while positive pressure ventilation causes rise in intrathoracic pressure with resultant rise in mean CVP. Therefore CVP should be recorded at end-expiration.

Summary:

The choice of central venous access devises, their insertion techniques and site of insertion depends on the indication. Ultrasonography guided catheter insertion is less dangerous in

experienced hands. Understanding technical details of invasive monitoring helps in better interpretation of the information. CVP measurements depend on intravascular volume, ventricular compliance and transmural pressure. Different pathophysiologic conditions can be diagnosed using CVP waveforms.

References:

1) Forssmann W. Die Sondierung rechten Herzens. Klin Wschr. 1929;8:2080
2) Aubaniac R. Subclavian intravenous injection; advantages and technic. Presse Med. Oct 25 1952;60(68):1456
3) Sven Evar Seldinger, Catheter replacement of the needle in percutaneous arteriography. Acta Radiologica (Stockholm) 39:368-376, May1953.
4) Hughes RE, Magovern GJ. The relationship between right atrial pressure and blood volume [abstract]. Arch Surg 1959;79:238
5) Frantz J. Gibbs and Michael C. Murphy, Ultrasound guidance for central venous catheter placement. Hospital Physician March 2006.
6) Frasca et al. Prevention of central venous catheter-related infection in the intensive care unit. Critical Care 2010, 14:212.
7) Paul E. Marik, Michael Baram and Bobbak Vahid, Does Central Venous Pressure Predict Fluid Responsiveness? A Systematic Review of the Literature and the Tale of Seven Mares. CHEST 2008; 134:172–178.
8) Ronald D. Miller, MD, Lars I. Eriksson, Lee A. Fleisher, MD, Jeanine P. Wiener-Kronish, MD and William L. Young, Miller's Anesthesia. Seventh edition, ELSEVIER.