Chapter 4
Cardiovascular Assessment

Irene Comisso and Alberto Lucchini

4.1 Introduction

In healthy people, cardiovascular system allows blood to reach the organ and tissues, providing oxygen and nutrients, and blood flow from peripheral tissues removes toxins and carbon dioxide (CO_2).

In intensive care unit (ICU) patients, cardiovascular function often results strongly compromised, thus determining the need for advanced monitoring and support. Instrumental monitoring is one of the most important components of cardiovascular function assessment, together with scores (such as the APACHE II or SOFA) and clinical observation. Since clinical scores and direct observation are not reliable enough to assess adequately the changes of patients' status during time, instrumental monitoring systems have found a rapid development in clinical practice whose main application, in the beginning, has been in anesthesia practices, where basic monitoring has been assumed as standard by several societies [1]. During the last three decades, more and more sophisticated devices to assess cardiovascular parameters have been tuned fine, allowing clinicians to

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obtain (even with relatively easy training) complementary information, that all together outline the general situation of the patient. Nonetheless, it has to be considered that cardiovascular monitoring (CM) is often invasive and expensive and requires sufficient expertise in device insertion and data interpretation. Similarly, not all monitoring devices are appropriated in different clinical situations. On these bases, a progressive implementation model [2] for CM monitoring in ICU has been proposed, defining three levels of complexity for CM, that should be adopted on a continuum according to the patient condition.

4.2 General Considerations

From a general point of view, you can consider the characteristics of a CM according to its continuity and invasiveness. According to the system used, the same parameter can be evaluated continuously or intermittently (central venous pressure obtained via a pressure transducer or a water manometer). Invasiveness refers to the extent of a barrier violation. Electrocardiogram is a noninvasive monitoring, while blood pressure obtained through a transducer is considered invasive (or minimally invasive), and pulmonary artery catheter represents the maximal invasivity. Precision and accuracy are also important variables to be considered. Precision indicates how a measurement produces the same result each time it is repeated under the same conditions [3]. Accuracy reflects how close is the actual measurement to the real value [3].

Basic monitoring includes those parameters recorded in all critically ill patients, while advanced monitoring comprises those who are introduced in specific critical conditions (Table 4.1). As it can be easily understood, cardiovascular parameters should always be evaluated with the respiratory ones, since there is a strict interaction between the two systems

Table 4.1 Characteristics of main parameters monitorized in ICU patients

	Basic	Advanced
ECG	3–5 leads continuous ECG	12-lead ECG
Pump function	Invasive or noninvasive blood pressure	Intermittent or continuous cardiac output
Oxygen	SpO_2, $ScvO_2$	SvO_2
Volemia/filling pressures	PVC	PAOP (wedge pressure) Stroke volume, intrathoracic blood volume/global end-diastolic volume

that is enhanced in critically ill patients undergoing to mechanical ventilation (MV).

Assessment of cardiovascular function in ICU patients consists of four evaluation points:

- Electrical activity
- Pump function effectiveness
- Oxygen transportation and consumption
- Volemia

4.3 Electrical Activity

Continuous ECG monitoring allows nurses and clinicians to quickly identify arrhythmia and promptly respond to such events.

The development of electrocardiographic (ECG) monitoring began during the first three decades of the twentieth century, with 3-lead ECG recording [4]. Further developments allowed the diagnosis of bundle branch block and cardiac ischemia, and in 1954 the standardization of 12-lead electrode positioning was

released [4]. Current standard for cardiac monitoring within coronary care units include heart rate and rhythm, ST-segment analysis, and QT-interval measurement [4].

ECG leads are classified as unipolar, with one registering and one indifferent electrode (aVR, aVF, aVL, and the six precordial leads V1–V6), and bipolar, with a positive and a negative electrode (the original Einthoven leads, I, II, III) [5].

Heart contraction is made possible by polarization and depolarization of muscle fibers. Evaluation of electrical activity through a 3- or 5-lead electrocardiogram (ECG) provides easy and immediate information about stimulus conduction through the heart, although a more accurate evaluation is only possible by 12-lead ECG. Cardiac cycle begins with spontaneous depolarization of sinus node cells, whose depolarization wave diffuses through the whole myocardial muscle, followed by atrial contraction (P wave). Atrial contraction allows ventricular filling. Electrical impulse reaches then the atrioventricular node (P-R interval) and is then diffused to ventricular cells, who undergo a depolarization process (QRS interval), with subsequent ventricular contraction. Finally, atrial and ventricular depolarization occur (the first is masked by the second because of its highest electrical potential) (T wave) (Fig. 4.1) [5].

Normally, ECG analysis is based on six points (Table 4.2):

- Presence/absence of electrical activity. This point may reflect a simple artifact (due, e.g., to electrodes disconnection), or highlight the presence of asistolia, or other condi-

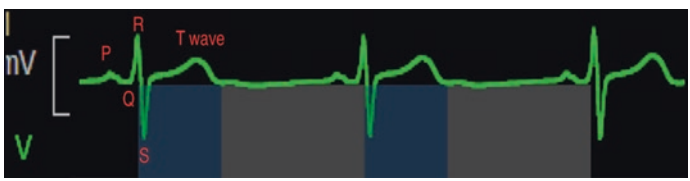

Fig. 4.1 Normal ECG visualization

Table 4.2 Steps for ECG interpretation and features

	Normal ECG findings	Arrhythmic features
Electrical activity and heart rate	Normal ECG features (see Fig. 4.1) Normal HR ranges between 60 and 100 beats per minute (BPM)	Asystole: no electrical activity is visible, and ECG lead reconnection has been excluded Pulseless electrical activity: when electrical activity is not followed by mechanical contraction Bradycardia describes a HR < 60 BPM Tachycardia describes a HR > 60 BPM
R-R interval	Regular interval, its duration depending from HR	In atrial fibrillation (AF), the most frequent arrhythmia, electrical atrial activity is disorganized, and only a few electrical impulses reach the ventricles
P wave	P wave present, sinus rhythm	In AF and in ventricular tachycardia, P waves are not identifiable
Relationship between P wave and QRS complex	Normally, the interval between P wave and QRS complex is <0.2 s; a P wave is always followed by a QRS complex	The relationship between P wave and QRS complex is inconstant in second third degree atrioventricular block
ST-segment elevation	ST segment aligned with the isoelectric line	ST segment alterations may be related to ischemic conditions
P-R interval	0.12–0.2 s	P-R interval is prolonged in first and second grade atrioventricular block and is absent in third grade atrioventricular block

tions in which electrical activity is absent (such as pulseless electrical activity, i.e., an electrical activity not followed by cardiac muscle contraction). Electrical activity results in heart rate (HR), expressed as number of QRS complexes in a minute
- Cardiac rhythm, highlighted by the R-R interval
- Presence of P wave, defining the presence or absence of atrial activity
- Relationship between P wave and QRS complex
- QRS width
- ST-segment elevation [6].

Avoidance of artifacts during ECG monitoring includes checking the correct positioning of the leads, since reversal between left and right arms or arms and legs can occur, thus leading to polarity inversion [7]. Artifacts can also be induced by patient's tremors [7].

4.4 Pump Function Effectiveness

Heart works as a pump in the circulatory system, being responsible, together with aortic compliance (what is called "Windkessel effect"—see arterial pressure monitoring paragraph) of continuous blood flow through vessels.

4.4.1 Cardiac Output

Cardiac output defines the amount of blood flowing through heart's chambers during 1 min and is expressed by the equation:

$$CO = HR \times SV$$

In healthy individuals, cardiac output ranges around 5 L/min. To easiest compare cardiac output in differently sized people, values are indexed over body surface area, thus determining the cardiac index (CI). CO is one of the most important hemodynamic parameters using in ICU patients, since blood flow through arteries is one of the determinants of oxygen delivery toward cells. It is possible to determine CO through Fick's principle or by dye dilution.

Fick's principle states that blood flow through an organ (or the whole body) can be measured from three variables:

- Amount of marker substance taken up by the organ per unit time
- Concentration of marker substance in arterial blood supplying the organ
- Concentration of marker substance in venous blood leaving the organ

Therefore, determining oxygen consumption (VO_2) per unit time, and dividing it by arteriovenous oxygen content difference, provides cardiac output measurements, as expressed in the formula:

$$VO_2 = \left(CO \times C_a\right) - \left(CO \times C_v\right)$$

Dye dilution has a wider bedside applicability, if compared with Fick's principle, and it is based on the concept that an indicator injected through a vessel at known volume and concentration can be detected downstream. Its concentration at detection site depends on blood flow per unit time. Further application of this principle consists in using cold normal saline solution and recording blood temperature variations using a thermistor. This is one of the applications of Swan-Ganz catheter (also called pulmonary artery catheter—PAC), originally conceived to determine pulmonary artery and

wedge pressure. PAC was introduced in clinical practice in 1970 [8] and, up to the mid-1980s, remained the most advanced cardiovascular monitoring system in ICU. It is a 110 cm catheter, provided with different lumens (Fig. 4.2):

- Distal lumen opens out in pulmonary artery and provides continuous pulmonary artery pressure (PAP) reading.
- Proximal lumen opens out in right atrium and provides continuous central venous pressure (CVP) reading.
- Thermistor lumen provides continuous blood temperature reading.
- Balloon lumen inflates a balloon close to the distal lumen; its occlusion stops blood flow through the pulmonary artery and provides a balloon downstream pressure reading (from the PAP lumen). This pressure reflects on left atrium pressure and is called wedge pressure (WP).

Cardiac output is determined with the thermodilution technique, according to the Stewart-Hamilton equation:

$$Q = \frac{V\left(T_{\mathrm{b}} - T_{\mathrm{I}}\right)K_1K_2}{T_{\mathrm{b}}\left(t\right)^{\mathrm{d}t}}$$

where

- V_{I} = injected volume.
- T_{b} = blood temperature (at pulmonary artery).
- T_{I} = injected dye temperature.
- K_1 = density factor.
- K_2 = computation constant.

To obtain a reliable curve, some issues have to be considered: the indicator mixing has to be rapid (bolus injection) and complete; blood flow and baseline temperature have to be constant; bolus volume should produce an adequate temperature variation [9].

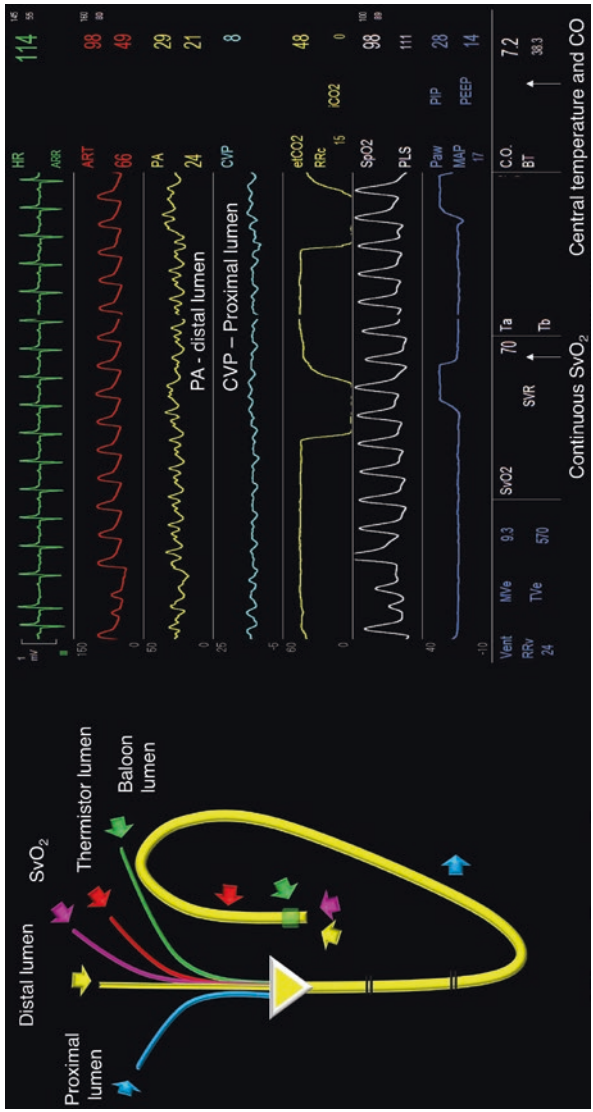

Fig. 4.2 Swan-Ganz catheter and wave visualization

Valvular insufficiency (tricuspid and pulmonary) may lead to CO underestimation, as backward flow might result in dye recirculation. Furthermore, flow variations are physiologically observed during different respiratory cycle phases (both in spontaneous breathing and mechanical ventilation), thus requiring 3–5 bolus injections.

As other invasive devices, PAC requires maximum sterile barrier precautions during its insertion. Its use in ICU patients is limited to situations in which CO monitoring is necessary together with PAP. Several studies [10–13] evaluated the relationship between PAC application and patients' survival, demonstrating no substantial benefit, but a high risk of complications (arrhythmia, endocarditis, valve damage, pulmonary artery embolus) related with PAC positioning. PAC positioning also seemed to be related to higher mortality in ICU patients, probably depending on strict indications to its positioning that refer to more severe clinical conditions.

Technologies' developments allowed introduction of new devices dedicated to cardiac output monitoring. A modified PAC was introduced in the early 1990s. This device is provided with a thermal filament which is warmed at 8 min intervals. Filament's warming increases blood temperature, whose variation is detected downstream by catheter's thermistor: practically, the catheter works with an inverse thermodilution curve. This device has the same invasiveness of a traditional PAC but with some advantages: particularly, inverse thermodilution is a semicontinuous measurement and allows clinicians to an easier and more prompt identification of clinical conditions changes [14].

Other technologies developed during recent years to determine cardiac output found wider application in ICU and high-risk surgical patients. These methods are classified as less invasive (requiring a femoral or radial artery catheter and a central venous catheter) [15] or minimally invasive (requiring a radial artery catheter) and are based on pulse contour analysis

algorithms. Some of them require calibration, while others do not. The most important advantage of these methods lays in continuous cardiac output measurement (which is determined beat by beat), immediately reflecting changes in hemodynamic condition. Furthermore, these technologies provide adjunctive parameters (related to volemia and fluid responsiveness). Likewise, some limitations for their applications have to be considered, since minimally invasive methods' reliability seems to be affected by hyperdynamic conditions and atrial fibrillation [16].

Indeed, wider importance and application has been reached during recent years by noninvasive measurements, including transthoracic echocardiography [17, 18].

Cardiac output measurements' methods have been validated toward PAC (which is considered the gold standard).

Currently, CO measurement is mainly indicated in high-risk surgical patients (such as cardiac surgery or liver transplant), in patients with septic shock and acute respiratory distress syndrome [16].

4.4.2 Arterial Pressure Monitoring

Arterial blood pressure (ABP) represents the force exerted from blood on arterial walls and derives from interaction between three factors: hydrostatic pressure (which, in turn, is related to the height of blood column and its density), hemodynamic pressure (coming from the strength of heart contraction), and kinetic energy (related to blood progression within cardiovascular system) [19]. In ICU patients, arterial blood pressure is usually measured using invasive catheters, which are generally inserted in large vessels (such as radial or femoral artery). The catheter is connected to an electronic pressure transducer using a tubing system filled with normal saline solution. The electronic transducer allows conversion of mechanical pressure wave into an

electric one. Intra-arterial catheters provide more reliable data, compared with oscillometric systems. Furthermore, values obtained using these devices are continuous, providing clinicians immediate information concerning clinical stability variations and responses to treatments. Arterial catheters also allow collection of arterial blood without the need for peripheral puncture. Finally, analysis of the arterial waveform might highlight adjunctive information regarding patient's volemia and predict fluid responsiveness.

When measuring arterial blood pressure, three values are considered and displayed on the monitor: systolic (SBP), diastolic (DBP), and mean (MBP) pressure. SBP is the peak pressure reached during the cardiac cycle, resulting from interaction of several factors (EDV, SV, heart contractility force, blood density, arterial walls compliance); DBP is the trough during cardiac cycle and is mainly determined by arterial walls compliance [19, 20]. MAP is defined as mean pressure (usually equated as MAP = (SBP + 2DBP)/3) during cardiac cycle, and it is considered a hemodynamic target during resuscitation maneuvers [20]. The difference between SBP and DBP is called pulse pressure (PP), and it determines the peripheral palpability of arterial pressure wave (e.g., at radial, pedidial, or femoral site) [20].

When visualizing an arterial pressure waveform, several components can be identified [19] (Fig. 4.3):

- Anacrotic limb, corresponding to pressure increase due to left ventricle contraction; it ends with the top rounded, also called anacrotic shoulder.
- Dicrotic limb, corresponding to a decrease in pressure; it ends with the dicrotic notch, which reflects the closure of aortic valve.
- After closure of aortic valve, ABP still decreases until it reaches diastolic value; time and slope of this curve portion depend on heart rate and arterial compliance.

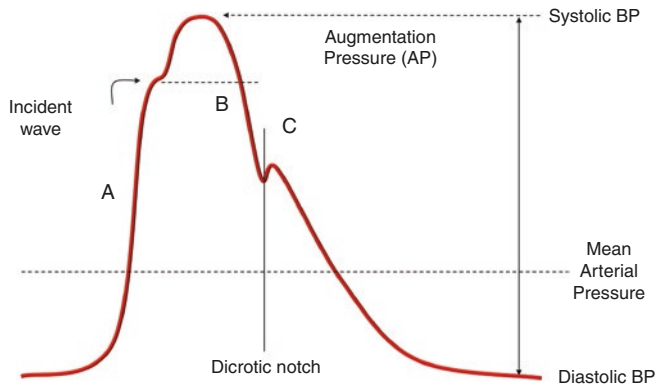

Fig. 4.3 Arterial waveform. *A* = anacrotic limb; *B* = dicrotic limb; *C* = dicrotic notch

It is important to consider that ABP results not only from ventricular ejection force but also from the reflection waves directed toward the heart. Moreover, arterial wall's structure works as a reservoir, which is filled during systole, and releases blood during diastole, thus allowing continuous blood flow over the whole cardiac cycle (Windkessel effect). Arterial waveforms significantly differ according to the measurement site, since the reflection wave effect becomes more evident as more distant from aortic root the measurement is performed. Furthermore, reduction in aortic elasticity can result in increased and earlier reflection wave. Patient's position during measurement can also affect measured values, due to the effects of the hydrostatic column (therefore, in a standing position, arterial pressure measured at foot level will be higher than the one at neck level).

Analysis of arterial waveform found important implications during the 1990s, when algorithms considering pulse contour analysis allowed continuous measurement of cardiac output. Adjunctive considerations were conducted on pressure and

stroke volume variation during respiratory cycle. The underlying consideration is that nearly 50% of patients (defined as preload nonresponders) don't show a positive response to fluid challenge during shock resuscitation [21]. Considering that fluid overload may lead to pulmonary and cerebral edema, it's easy to understand the need to develop criteria and parameters to guide fluid bolus administration and early identify patients potentially nonresponding to these treatments [22].

After preparing the required supplies (pressure bag, normal saline bag, monitoring kit), setting an arterial transducer is detailed in Table 4.3.

During preparation of an arterial line, some important principles have to be considered:

- Tube length should not exceed 120 cm; tubes should be stiffer than the ones used to administer fluids, in order to reduce pressure wave dispersion through the tube walls.
- Avoid air bubbles within tubing system: small ones can lead to reduced signal resonance (with falsely high SBP readings), while large ones will reduce signal amplification (with falsely

Table 4.3 Steps to set the pressure transducer

Action	Rationale
Insert aseptically the spike into the bag, and fill almost half of the drip chamber	Avoid fluid contamination and air bubbles into the tube
Turn the stopcock off to the patient, and pull the fast-flush device	Priming the tube portion to the transducer system
Turn the stopcock off to the transducer and pull the fast-flush device	Priming the tube portion to the patient
Remote any remaining air bubble keeping the fast-flush device open	Avoid air in the system
Place normal saline bag inside a pressure bag, and inflate it to 250–300 mmHg	Keep a small continuous flush and avoid blood reflux

low DBP) [19, 21]. Similarly, clots should be prevented by continuous tube-flushing (obtained through a 300 mmHg pressurized normal saline bag), and catheter kinking avoided through adequate dressing.

Accuracy of the measurement requires to apply some principles summarized in Table 4.4 [19, 23].

A simple evaluation of dynamic response can be obtained by performing a square wave test (Table 4.5) and by observing the resultant oscillations (Fig. 4.4). In order to perform this assess-

Table 4.4 Principles to obtain accurate invasive pressure values

Zeroing	Refers to attributing a "zero point" to the measurement, above which an invasive pressure is measured; the "zero point" normally refers to atmospheric pressure; after zeroing the transducer system, it will be possible to associate numeric values to the pressure wave
Leveling	Refers to positioning of the transducer system: when measuring cardiovascular pressures, the transducer level should be at fifth intercostal space on the midaxillary line or the sternal angle (where the sternum and second rib attach); in first case, the patient is required to be supine, and in the second, measurements can be obtained even at 60° elevation When pulmonary artery pressure is measured, the phlebostatic axis is defined by the midpoint between the anterior and posterior surfaces of the chest at the fourth intercostal space when the patient is supine When the transducer is under the phlebostatic axis, the measured value will be higher than the real pressure; conversely, when it is over the phlebostatic axis, the measured value will be lower than the real pressure
Damping	Refers to the dynamic response of the system to a sudden, high pressure (obtained releasing the transducer's fast-flush valve). Underdamped systems overestimate systolic pressure and underestimate diastolic pressures. Conversely, overdamped systems will underestimate systolic pressures and overestimate diastolic pressures (Fig. 4.4)

Table 4.5 Square wave testing

Step	Action
1	Activate snap or pull tab on flush device
2	Observe square wave generated on bedside monitor
3	Count oscillations after square wave
4	Observe distance between the oscillations

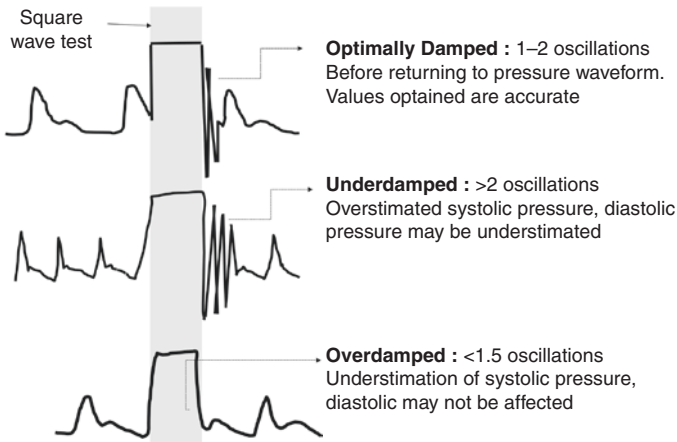

Fig. 4.4 Square wave test with optimally damped signal, underdamped and overdamped signal, during arterial invasive monitoring

ment accurately, a flush device that can be activated rapidly and then released is required. A flush device that does not close rapidly after activation (squeeze or press type) may not close the restrictor quickly and may produce erroneous results.

The same consideration can be applied for other blood pressure measured using a transducer (pulmonary artery pressure and central venous pressure).

Fig. 4.5 Pulmonary artery pressure waveform

4.4.3 Pulmonary Artery Pressure

PAP values are detected through the distal lumen of a PAC. PAP wave is in some way similar to the systemic arterial pressure one, but values are lower, ranging between 20 and 30 mmHg for systolic pulmonary pressure and 5 and 10 mmHg for diastolic. PAP monitoring aims to identify and manage pulmonary hypertension (PH), a threatening condition that may lead to increased cardiac workload [23, 24] (Fig. 4.5).

Usually, PH is defined as a mean arterial pressure ≥25 mmHg at rest, measured by right heart catheterization [25]. Precapillary pulmonary artery hypertension (PAH) requires the measurement of wedge pressure and can be induced from lung diseases. The diagnostic criteria pointed out during the fourth World Symposium on Pulmonary Hypertension keep the pulmonary artery wedge pressure cutoff for the definition of precapillary PAH at ≤15 mmHg [25]. Several conditions (both congenital and disease related) have been associated with PAH [26].

PAH pathogenesis derives from an imbalance between vasodilators and vasoconstrictors molecules and can be enhanced by the reaction with some drugs.

4.5 Oxygen Transportation and Consumption

Oxygen is used by cells during metabolic processes, being transported by blood hemoglobin to peripheral tissues. Blood oxygen content is expressed by the equation:

$$CaO_2 = (1.34 \times Hb \times SaO_2) + (0.003 \times PaO_2)$$

It is therefore easy to understand how alteration of a single or multiple factor may affect oxygen availability. Anemia correction, oxygen fraction increasing, and cardiac function improvement are all interventions aiming to increase the amount of available blood oxygen. Oxygen extraction from cells depends on several factors, such as cells perfusion and metabolic activity. In ICU patients, some factors (fever, burns, shivering, and infectious and inflammatory reactions) may increase oxygen extraction, while other conditions (neuromuscular blockade, deep sedation, microvascular thrombosis, shunt) might decrease it.

Venous oxygen saturation is defined as the percentage of venous hemoglobin saturated by oxygen; venous oxygen saturation values normally range between 60 and 80% and vary according to measurements' districts. It can be measured collecting a blood sample from distal lumen of a central venous catheter (which is called central venous oxygen saturation—$ScvO_2$) in jugular or subclavian vein or Swan-Ganz catheter (which is called mixed venous oxygen saturation—SvO_2) [27]. Accurate $ScvO_2$ measurement might depend from distant positioning of catheter's tip from right atrium.

SvO_2 is considered as most accurate, since it reflects oxygen consumption at whole organs, including coronary and pulmonary circulation, while $ScvO_2$ provides an index of oxygen consumption at higher portions of the body. Studies have shown a good correlation between $ScvO_2$ and SvO_2, the first generally overestimating the second by 3–8% [28], but in patients with septic shock, the bias between the two measurements might be significantly higher, leading to misinterpretation of falsely high oxygen availability [29, 30] and suggesting that trends are more helpful than single values in estimating patients response to treatments. Venous oxygen saturation has been evaluated as consistent endpoint in studies [31] evaluating fluid challenge resuscitation in severe sepsis and septic shock, showing a consistent mortality reduction, particularly when treatment was initiated prior that severe organ damages emerge.

Decreased venous oxygen saturation (<60.8% [32]) may depend from insufficient oxygen delivery or increased oxygen extraction at cellular level [33]. Increased venous oxygen saturation (>77.4% [32]) usually reflects a decreased consumption (e.g., during general anesthesia or in severe hypothermic conditions) or a delivery exceeding cells requirements [33].

4.6 Volemia

Determination of patient's volemia might be crucial to manage a cardiovascular dysfunction condition and may help in differentiating the most appropriate therapeutic choice, particularly targeting the administration of fluids and inotropes.

4.6.1 Filling Pressures: Central Venous Pressure and Pulmonary Artery Occlusion Pressure

Central venous pressure (CVP) is defined as the pressure measured through a venous catheter whose tip is positioned close to the right atrium. CVP can be defined as the pressure resulting from the interaction between venous return and cardiac function. CVP has been widely used as a surrogate indicator of the volemic status of patients, according to the principle that a larger volume reflects on a higher pressure inside atrium. This principle is normally true in healthy subjects, nonetheless, it cannot be always considered true in ICU patients, in which many factors interact, determining alterations in CVP measurements. For example, several conditions common for ICU patients (such as pneumothorax, pericardial tamponade, heart failure) can result in high CVP readings, who often do not really reflect a normovolemic status.

CVP can both be measured using a transducer or a water manometer (in this case the observed value won't be continuous). As for any invasive pressure, leveling and zeroing procedures are required (Table 4.4). The transducer should be positioned at right atrium level (with patient supine on a flat position, or with head of bed elevated by 30°, 45°, or 60°, since the right atrium is anterior and round, and its midpoint remains at the same vertical distance below the sternal angle). The atrium position on the chest is normally identified by intersection of midaxillary line and fourth intercostal space (more easier, 5 cm below the sternal angle) [20].

CVP waveform (Figs. 4.6 and 4.7) is composed of three prominent positive waves (a, c, and v) and two prominent negative waves (x and y descents). The "a" wave is generated by atrial contraction; the "c" wave is due to backward closure of the tricuspid valve (onset of systole), and "v" wave reflects atrial

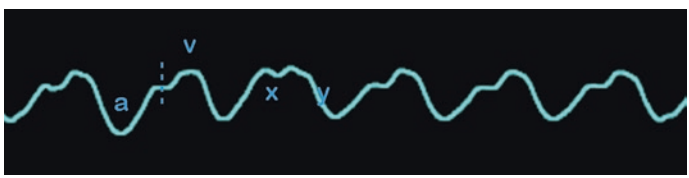

Fig. 4.6 Central venous pressure waveform. Dotted line shows the ideal site for measurement

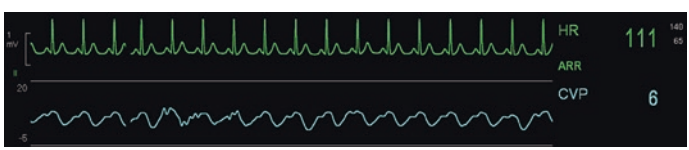

Fig. 4.7 Central venous pressure waveform pooled together with the ECG waveform. Observe waves a (right after the P wave and before the QRS complex on the ECG, expression of atrial contraction) and v (corresponding to the descent T on the ECG, expression of atrial filling)

filling during diastole; the "x" descent comes from the fall in atrial pressure during atrial relaxation, while the "y" descent comes from a fall in atrial pressure (onset of diastole, emptying of atrium in the ventricle) [34]. To obtain a more reliable measurement, CVP should be obtained at the end of expiration, in order to reduce the effects of transmural pressure. The preferred site for measurement is the leading edge of the "c" wave (generally approximated by the base of the "a" wave) [35].

Pulmonary artery occlusion pressure (PAOP), also known as pulmonary artery wedge pressure (PAWP), is obtained performing right heart catheterization using a Swan-Ganz catheter. During its positioning, the Swan-Ganz catheter balloon is inflated in the right atrium until it reaches the wedging position that means the occlusion of a pulmonary artery branch. Balloon's inflation should follow some simple principles, to avoid severe complications: the air volume used should range between 1 and 1.5 mL. Recent summarized recommendations report that repeated inflations and deflations of the balloon should be avoided, since they have been associated to pulmonary artery's rupture [25]. PAWP values normally range between 5 and 12 mmHg, with a slight increase (up to 15 mmHg) related with age. Its measurement should always be standardized to an ideal position, with patient lying supine and the transducer at mid-thoracic line, halfway between the anterior sternum and the bed surface (left atrium) [25].

PAWP has been considered for a long time a surrogate marker of left ventricular preload, according to the principle that under normal (and static) conditions it is equivalent to left atrial pressure, which, in turn, equates to left ventricular end-diastolic pressure (LVEDP) [36]. This assumption remains actually true only in the absence of particular conditions, such as mitral valve and left ventricular wall pathologies, and when the effect of intrathoracic pressures is minimized (that means at end of expiration). Such criteria strongly limit the effective applicability of this measurement as preload index; therefore the association with other measurements is required [36].

4.6.2 Volumetric Indicators

The need for assessing volemia in critically ill patients gave course to development of the so-called "volumetric indicators" that usefully guide clinicians in fluid replacement. Volemia is defined as the total blood flowing through the circulatory system. Hypovolemic conditions can be both absolute and relative. The first is characterized by an important circulating volume loss (hemorrhage or dehydration due, e.g., to fever, burns, renal failure, vomiting, or diarrhea). The second is attributable to redistribution of volume in third space (such as in capillary leak syndrome) or in body cavities (such as in pulmonary edema, ascites, pleural effusion). In relative hypovolemia, an imbalance of fluid homeostasis between capillary and interstitial space (which is normally controlled by electrolytes and protein concentration) is observed.

Recent developments in hemodynamic monitoring devices offer relatively easy-to-use solutions to assess blood volumes at the bedside. The principle on which these measurements are based refers to dilution of a thermal indicator (in the past, it was a colorimetric one) injected at a known temperature through a central vein and detected though a thermistor placed in arterial catheter. This technique is known as transpulmonary thermodilution. The analysis of the thermodilution curve provides the so-called "mean transit time" (MTt) that defines mean time needed for passage of every indicator's molecule. After injection, thermal indicator distributes to intrathoracic thermal volume (ITTV), clinically represented by global end-diastolic volume (GEDV), extravascular lung water (EVLW), and pulmonary blood volume (PBV) (Table 4.6).

In healthy subjects, intrathoracic blood volume (ITBV) (resulting from summation of GEDV and PBV) represents approximately 26% of global blood volume. ITBV can be mathematically derived from GEDV [37] (Table 4.6).

Table 4.6 Volumetric measurements

Intrathoracic thermal volume (ITTV)	ITTV = MTt × CO	Total amount of blood and water inside chest
Total pulmonary volume (TPV)		Total amount of blood and water inside lungs
Global end-diastolic volume (GEDV)	ITTV-TPV	Sum of the volume of the four cardiac chambers
Intrathoracic blood volume (ITBV)	1.25 × GEDV [37]	Sum of the volume in cardiac chambers and pulmonary vascular bed
Extravascular lung water (EVLW)	ITTV-ITBV	Amount of water within the lung interstitial space

GEDV, whose normal values range between 600 and 800 mL/m^2, includes the volume of the four cardiac chambers and the blood volume between the injection site (via a CVC at the superior vena cava) and the thermistor (femoral artery) [38]. As for CO measurement, uneven dye mixing (aortic aneurism, intracardiac shunt, vascular pulmonary bed reduction) may lead to incorrect volumetric esteem.

ITBV and GEDV (indexed on body surface area, ITBVI, and GEDVI) are used as preload indexes in several ICU and anesthesia conditions (sepsis, solid organ transplant) and have shown a better performance in guiding fluid and inotropic therapy, compared to previously used CVP and PAWP.

During lung transplantation, ITBVI showed a good correlation with stroke volume index (SVI), while only poor correlation was found between ITBVI and PAOP [39]. Similar results were obtained in hyperdynamic patients undergoing liver transplantation [40]. ITBVI was also found to be a better preload indicator than cardiac filling pressures (CVP and PAOP) in

patients with sepsis or septic shock [41]. Extravascular lung water (EVLW) is a bedside measurement of the amount of lung water outside the vascular compartment. Practically, it is a measurement of the amount of pulmonary edema (defined as the difference between PTV and ITBV), previously assessed by radiologic imaging (such as chest X-ray, computerized tomography, and magnetic resonance imaging) [42].

The gold standard for EVLW measurement is the ex vivo gravimetry, obtained weighting lungs before and after their dry out [42]. Obviously, this method is inapplicable in alive patients. Dye dilution methods allowed bedside measurement of EVLW from the 1980s, and further thermal dilution had a wide diffusion [43]. Recently, estimation of EVLW has also been conducted by using the chest ultrasound, showing good performances in terms of sensitivity (81%) and specificity (90.9%) [44], although pulmonary edema detection may be limited from the lung region where it is performed [45].

The initially fixed cutoff value indexed on body surface area of 7 mL/kg body weight [46] has been recently increased to 10 mL/kg [47]. Recently, indexation of EVLW (EVLWI) to predicted body weight (rather than actual body weight) has been proposed, to avoid underestimation of EVLW in obese patients, and in those who develop positive fluid balance, it has been introduced, showing a better correlation with lung injury scores and oxygenation. Also, EVLW indexed on predicted body weight had a better correlation with patients' outcome [48]. Other authors also suggested EVLW indexation to patient's height, as it is considered the main determinant of lung volume [49]. EVLWI measurement using transpulmonary thermodilution might be overestimated by lung resection and underestimated by pulmonary embolism [45].

Currently, EVLW is not included in ARDS as defined by Berlin criterion, although diagnosis might be improved by using it [45].

EVLW guides clinicians when acting fluid, diuretic, and inotropic therapy, in addition to organ support treatments, such as mechanical ventilation and continuous renal replacement therapy. EVLW demonstrated prognostic power, because its mean value is higher in non-survivor critically ill patients [50]. Fluid therapy oriented on EVLW values seems to reduce ICU length of stay and mechanical ventilation duration [51]. Recently, EVLW has been investigated in patients with acute postoperative hypoxemic respiratory failure treated with noninvasive ventilation (NIV). Before starting NIV, EVLW was found to be significantly lower in patients who did not later require intubation (8.6 ± 1.08 vs. 11.8 ± 0.99, $P < .01$) [52]. Similarly, after 1 h from beginning NIV treatment, EVLW significantly decreased in patients who did not require intubation (8.6 ± 1.08 vs. 6.2 ± 0.96, $P < .01$) [52].

Take-Home Messages

1. Cardiovascular monitoring provides data to adequately frame hemodynamic condition, but it cannot itself change patient's outcome.
2. Proper treatment decisions need reliable data. Therefore, appropriate technique (particularly concerning transducer's leveling, zeroing, and signal's damping) has to be applied.
3. No single data should be used to implement clinical decisions: every measurement should be considered together with other available ones and with global patient's condition (including other vital functions' assessment). Similarly, trend values should be considered to assess patient's responses to treatments.
4. Monitoring devices should be chosen according to patient's condition and staff confidence with their use and interpretation.

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