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Artículo de revisión

Invasive hemodynamic monitoring by Swan-Ganz pulmonary artery catheter: concepts and utility

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ABSTRACT

Since its beginnings in the last century, pulmonary artery catheterization (PAC) has evolved into an invasive hemodynamic evaluation technique that can be performed at the patient's bedside through a Swan-Ganz catheter; this procedure has maintained an intermittent course in terms of its use; however, it has currently demonstrated relevance in specific scenarios. The PAC allows access to the central venous circulation, the right heart and the pulmonary artery; it performs the calculation of hemodynamic variables directly or indirectly by means of established formulas and methods. This in turn provides proper hemodynamic evaluation and classification, additionally, PAC makes possible specific tests (e.g. vasoreactivity test), which help to define the diagnosis, treatment, monitor the response to treatment, evaluation prior to advanced therapies (e.g. cardiac transplantation or mechanical circulatory assistance devices), and prognosis in our patients. In this article we discuss the concepts and usefulness of pulmonary artery catheterization.

Keywords: Hypertension, pulmonary; Hemodynamic monitoring; Cardiogenic shock; Heart failure (source: MeSH NLM).

Introduction

The first cardiac catheterization was performed in 1920 by Dr. Werner Forssmann by introducing a urological catheter in the antecubital vein up to the right atrium [1]. Subsequently, catheterizations were routinely conducted with semi-rigid devices, and in 1953 Lategola and Rahn innovated, by animal experimentation, a balloon catheter system to facilitate the use of right-sided and pulmonary catheterization without assisted fluoroscopy [2]. In 1970, as a result of serendipity and based on the described antecedents, Harold James Swan and William Ganz created the flexible catheter system with inflatable distal balloon for humans. They were motivated to develop a technique for the care and study of acute cardiac patients in whom fluoroscopy was not available or for those who were immobilized due to hemodynamic instability or other causes [3,4].

Pulmonary artery catheterization (PAC) by Swan-Ganz catheter (SGC) was considered a revolutionary method at the time; however, in subsequent years, with the advance of diagnostic and therapeutic techniques, its use declined. In the past decade its use has regained prominence regarding invasive hemodynamic monitoring in patients with heart failure, associated with a decrease in mortality and an increase in hospital stay [5-7]. Likewise, its use has become relevant in patients with cardiogenic shock (CS) hospitalized in intensive care units [8].

The aim of this article is to review the current literature on the concept, use, indications and usefulness of PAC by SGC, as well as the interpretation of cardiopulmonary hemodynamic parameters.

Concept

The pulmonary artery catheter is a balloon-tipped flow-directed catheter that allows rapid access to the central venous circulation, the right heart and the pulmonary artery (PA) [1,9]. Its length is approximately 110 cm, with a standard external diameter of 7 or 7.5 French. The balloon at the tip, when inflated, guides the catheter from the major intrathoracic veins through the right atrium (RA) and ventricular chambers into the PA [9,10]. Most have four separate lumens, each of which has individual functions [9,10]:

- The proximal lumen (blue) is located in the RA and measures intra-atrial pressure. It can also be used to administer medications.
- The distal lumen (yellow) is located at the distal end and resides in the PA. It is used to monitor pressures and to obtain a mixed venous sample.
- The red port is for inflating and deflating the balloon. Each catheter is accompanied by a 1.5 mL syringe that is used to inflate the balloon.
- The temperature sensor (thermistor) is used to measure the core temperature in the PA.

The objective of PAC is hemodynamic monitoring and its physiological parameters derived from the evaluation of left and right ventricular function [1,8,9]. The thermodilution technique that calculates cardiac output (CO) measures the blood temperature variability of the PA and that of the saline solution injected by the RA, which produces a change in resistance and voltage, generating a time-temperature curve from which the CO is estimated by means of the Stewart-Hamilton equation [10]. If the area under the curve is small, the temperature equilibrates rapidly with the ambient body temperature resulting in a high CO, and if the area under the curve is large it implies a low CO [11,12].

Placement

Prior to the procedure, the indication for SGC placement must be clear, any possible contraindication must be ruled out and the risk of complications must be assessed **(Table 1)** [9,11]. The SCG introducer should be placed percutaneously via the inferior vena cava through the femoral veins or via the superior vena cava through the subclavian or internal jugular vein (interfascicular access); the latter accesses are of choice for bedside management. The right interfascicular approach via internal jugular vein is the preferred approach due to the easy and quick access to the RA. Puncture should be performed with the patient in the supine or Trendelenburg position with ultrasound guidance, confirming adequate venous return, and dilating the access area through a dilator to facilitate entry of the venous introducer [13-15].

The SGC should be introduced up to approximately 15 cm to obtain RA pressure waves, after which the distal balloon will be inflated and the catheter will continue to enter rapidly through the right ventricle (RV), to avoid ventricular ectopy, until obtaining PA pressure waves. Then, it should slowly progress until a pulmonary capillary pressure (PCP) curve is obtained, this happens 50 to 55 cm after catheter entry **(Figure 1)** [15].

Zeroing the pressure system is important to obtain an adequate PCP value and is carried out by positioning the transducer at the level of the RA (mid-axillary line, 4th intercostal space). The SGC should be fixed with the protective cap to mitigate the risk of infections and the adequate position should

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Table 1. Indications, contraindications and complications of the Swan-Ganz catheter.

INDICATIONS	RL	SOURCE
Patients with respiratory distress or altered systemic perfusion in the context of inadequate clinical management.	I	AHA 2013
Heart failure (reduced or preserved ejection fraction).	IIA	AHA 2013
- Acute heart failure with persistent symptomatology not responding to empirical management and/or uncertain hemodynamic status, acute renal failure and hypotension plus use of vasopressors.		AHA 2017
- Heart failure with organ dysfunction and absence of myocardial recovery, candidates for circulatory support and cardiac transplantation.		SCAI 2017
Cardiogenic shock	IIA	Ref.39,40
Cardiac transplant		SCAI 2017
Mechanical circulatory support		
- Initial acute management of patients on mechanical circulatory support.	IIA	SCAI 2017
- Persistent or recurrent heart failure after initiation of circulatory support.	IB	ISHLT 2013
- Right Ventricular Failure in Patients on Circulatory Support.	IB	ISHLT 2013
- Evaluation of circulatory support device dysfunction.	IB	ISHLT 2013
- Confirmation myocardial recovery associated with stepwise decrease in circulatory support pump speed and weaning of the circulatory support pump.	IIA	ISHLT 2013
- Monitoring during long-term circulatory support implantation	IIA	EACTS 2019
- Support in the management of fluid resuscitation and diagnosis of complications in patients on long-term circulatory support.	IIA	EACTS 2019
Other scenarios:		
- Cardiovascular postoperative monitoring.		
- Cardiac tamponade, restrictive and constrictive heart disease.		
- Pulmonary arterial hypertension.		
- Intracardiac shunts (congenital heart disease).		
- Acute pulmonary edema.		
- Mechanical complication after myocardial infarction.		
- Acute pulmonary thromboembolism.		
- Etiological evaluation of severe hypotensive states (hypovolemic, septic, cardiogenic shock).		
- Assessment of volume status in renal or hepatic failure.		
CONTRAINDICATIONS		
Absolute: Tricuspid or pulmonary mechanical valve prosthesis, intracavitary mass in right ventricle, right-sided endocarditis.		
Relative: Presence of endocardial pacing, tricuspid or pulmonary valvular biological prosthesis, respiratory distress syndrome due to pulmonary sepsis, complete left bundle branch block, significant arrhythmias, infection of the puncture site..		
COMPLICATIONS		
Hemo/pneumothorax, arrhythmias, conduction disturbance, hematoma, hypotension, vasovagal event, lower respiratory tract bleeding, catheter insertion site infection.		

AHA: American heart association. SCAI: Society of Cardiovascular Angiography and Interventions. EACTS: Expert consensus on long-term mechanical circulatory support. ISHLT: International Society for Heart and Lung Transplantation. RL: Recommendation level.

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be confirmed by radiologically locating the distal catheter in West's pulmonary zone III [9,14,15].

CO by thermodilution is carried out by infusing 10 mL of saline solution through the proximal lumen with a syringe in less than 4 seconds. The modification of the CO calculation constant depends on the infusion temperature, being 0.532 for 0 °C and 0.586 for 24 °C (this may vary according to the brand and type of monitor). Hemodynamic calculations will be carried out with the obtained CO. It should be taken into account that the presence of tricuspid insufficiency, low output states or intracardiac shunts may alter the accuracy of the results [9,11,15].

Hemodynamic monitoring

Right atrium

The RA pressure tracing shows different pressure curves (A-X-V-Y sequence). The A-wave represents atrial contraction, the X-descent represents the pressure drop during early ventricular systole and atrial relaxation, the V-wave represents atrial filling during ventricular systole and the Y-descent represents early diastole with rapid emptying of the RA. Thus, the X-descent and V-wave are systolic events, whereas the Y-descent, A-wave and the V-wave peak are diastolic events.

The central venous pressure (CVP) reflects the right intra-atrial pressure, it is preferably measured at the level of the Z-point and is represented by the correlation between the beginning of the QRS segment in the ECG and the intersection between the A and C wave. The CVP expresses the patient's volume status and its direct relationship with the RV, it can be interpreted as the filling pressures of the right side of the heart **(Table 2)** [16].

Right ventricle

RV tracings show a rapid pressure increase during ventricular contraction and a rapid pressure decrease during relaxation, with a diastolic phase characterized by an initially low pressure that gradually increases. The RA pressure should be fairly close to the RV end-diastolic pressure, unless tricuspid stenosis is present. With atrial contraction, an A-wave may appear at the end of ventricular diastole, which is an abnormal finding and usually indicates decreased distensibility, as in patients with pulmonary hypertension (PHT), RV hypertrophy or volume overload.

Pulmonary artery distensibility (PAD), determined by the resistive and pulsatile components of RV load, represents the relationship between stroke volume and pulmonary pulse pressure (PPP). It has been proven to be a strong prognostic indicator of mortality and RV dysfunction in type II PHT and heart failure with reduced ejection fraction (HFrEF); a value lower than 2.15 is associated with lower survival, even in patients with normal pulmonary vascular resistance (PVR) [17,18].

On the other hand, effective pulmonary artery elastance (PAE), a measure that relates PA systolic pressure (PAPs) to LV ejection volume, represents, like DAP, the RV afterload and function and, therefore, is a more specific predictor of mortality and RV dysfunction (independently of PVR and diastolic pulmonary gradient) in patients with type II PHT and heart failure

Table 2. Direct measurement parameters in hemodynamic monitoring by Swan-Ganz catheter.

Name	Normal range	Description
Central venous pressure (CVP)	2 – 6 mmHg	Represents right atrial pressure, and interprets right-sided filling pressures of the heart. CVP > 15 mmHg indicates overloaded right-sided pressures.
RV pressure (RVP)	RVSP: 15 – 30 mmHg RVDP: 2 – 8 mmHg	Has a rapidly ascending and descending sinusoidal wave. The presence of an A-wave at the end of diastole usually indicates decreased distensibility.
PA pressure (PAP)	SBP: 15 – 30 mmHg DAP: 8 – 15 mmHg MAP: 14 – 16 mmHg	It shows a rapid ascent and slow descent with a dicrotic notch, representing pulmonary valve closure. High PAPs associated with an elevated heart rate is indicative of right ventricular dysfunction and high incidence of cardiac events.
Capillary wedge pressure (CWP)	6 – 12 mmHg	The waveform is similar in appearance to the right atrial pressure wave with some differences (greater variability with the ventilatory cycle and the magnitude of the v-wave exceeds the a-wave in the tracing). CWP > 15 mmHg indicates hydrostatic edema, CWP should be corrected if PEEP is high and >10mmHg.
Cardiac output (CO) Cardiac index (CI)	4 – 8 L/min 2.5 – 4 L/min/m^2	Calculable by thermodilution using the Stewart-Hamilton equation and by the Fick principle; calculation by thermodilution is preferable except in the presence of left-to-right intracardiac shunt, where the use of Fick is recommended. CO < 4 L/min indicates low cardiac output. CI < 2.2 is a diagnostic criterion for cardiogenic shock.
Left atrium (LA)	5 – 10 mmHg	Inversely related to left ventricular distensibility. CWP is directly associated with LA pressures and these in turn with LV end-diastolic pressure.
Mixed venous saturation (SvO_2)	60 – 80 %	Oximetry analysis of a blood sample taken from the pulmonary artery (distal lumen). Central venous saturation < 60 % in myocardial infarction is indicative of low output state and cardiogenic shock. Mixed venous saturation < 60 % is an indicator of hypoperfusion, lactic acidosis and poor prognosis.

PHT: pulmonary arterial hypertension. **SBP:** systolic blood pressure **DAP:** diastolic blood pressure. **MAP:** mean arterial pressure. **PEEP:** positive end-expiratory pressure. **RV:** right ventricle. **RVDP:** right ventricular diastolic pressure. **RVSP:** right ventricular systolic pressure. **LV:** left ventricle.

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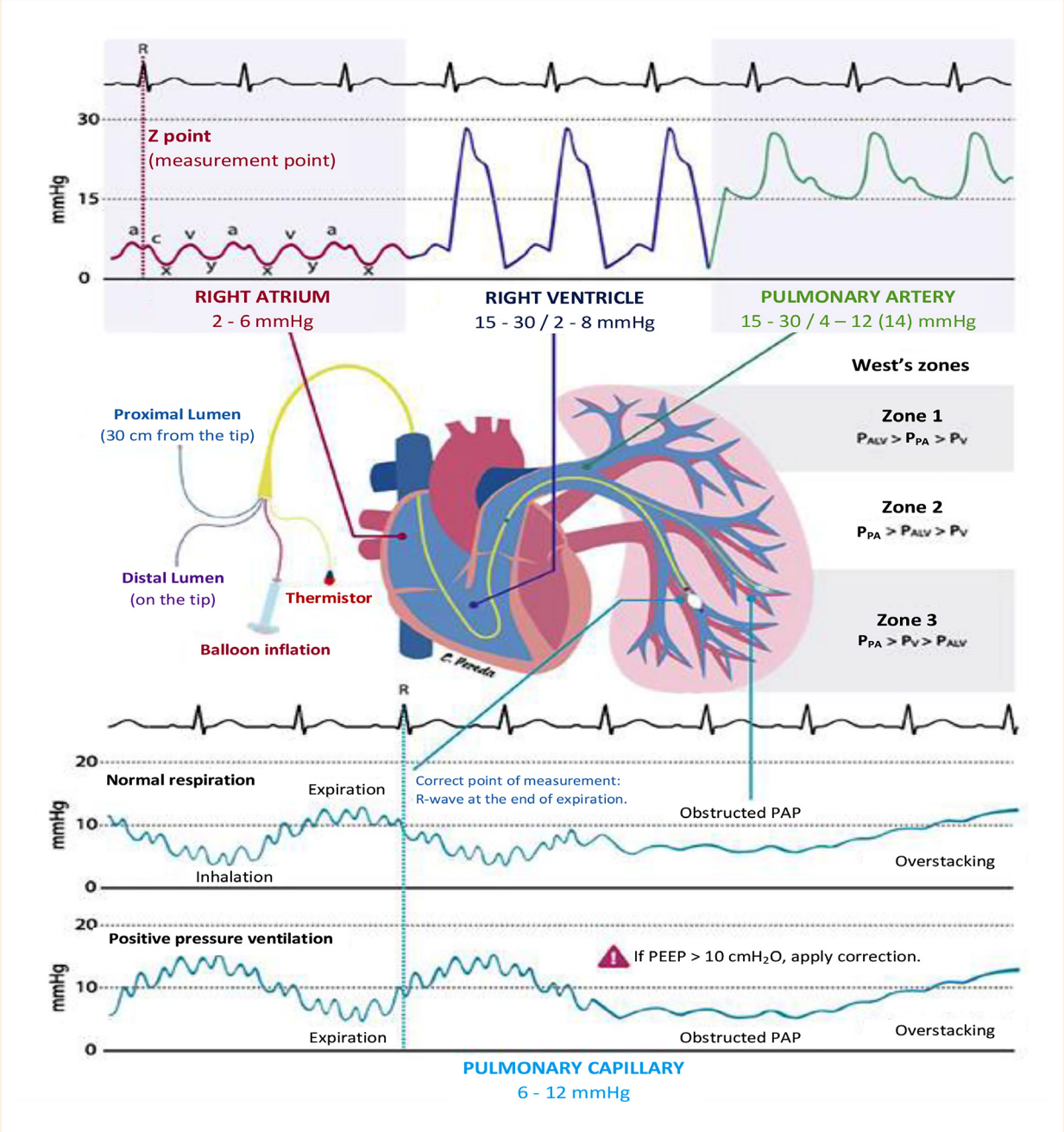

Figure 1. Hemodynamic monitoring by Swan-Ganz pulmonary artery catheter.

PAP: Pulmonary artery pressure. **PEEP:** Positive end-expiratory pressure.

(HF) even with HFrEF. Values greater than 1 support the need for therapy aimed at improving total RV load rather than the precapillary component, and its use is recommended in patients with decompensated HF and PHT II, as well as RV failure [17,18].

The pulmonary arterial pulsatility index (PAPi) determined by the relation between PPP and the RA, is a parameter that predicts severe RV dysfunction in the context of inferior myocardial infarction and/or LV mechanical circulatory support (MCS), developed with the aim of identifying patients requiring right mechanical assistance; it also tends to be more predictive in patients with inotropic support and is useful as a prognostic indicator of survival of PHT when values are < 0.95 [19,20].

Tabla 3. Interpretation of the hemodynamic monitoring according to scenarios.

Scenario	BP	HR	RA	PCP	CO	SVR	Actions
	–	–/↑	–/↓	–/↓	–/↓	–	Stop acute decongestion.
Unclear volume, perfusion and vascular resistance status.	–/↑	–/↑	↑↑	↑↑	↓	↑↑	Diuretic + vasodilator. Maintain CVP < 8mmHg, PCP < 15mmHg, SVR 1000 to 1200 dynas/s/cm^{-5} and MAP> 65mmHg.
Deterioration of renal function during decongestion.	↓	↑	↑↑	↑↑	↑	–	Inotropics (MAP > 65mmHg and CI > 2 L/min/m^2)
Dyspnea and unclear volume status.	–	–	↑↑	–/↓	–/↓	–\↑	Diagnostic suspicion and management of RV infarction, PHT, pericarditis, PTE.
	–	–	↑	↑↑	–	–	Diuretics
Hypotension of undefined etiology..	↓↓	↑	–/↓	–/↓	↓	↑↑	IV Fluid (Probable Hypovolemic Shock)
	↓↓	↑	–	–	–\↑	↓↓	IV fluid, vasopressor, etiologic management. (Probable distributive shock)
	↓↓	↑	↑↑	↑↑	↓↓	↓\↑	Inotropic and vasopressor. Possibility of MCS.

RA: right atrium. **HR:** heart rate. **CO:** cardiac output. **PHT:** pulmonary arterial hypertension. **CI:** cardiac index. **IV:** intravenous. **BP:** blood pressure. **MAP:** mean arterial pressure. **CVP:** central venous pressure. **PCP:** pulmonary capillary pressure. **SVR:** systemic vascular resistance. **PTE:** pulmonary thromboembolism. Adapted from: Hsu S, Fang JC, Borlaug BA. Hemodynamics for the Heart Failure Clinician: A State-of-the-Art Review [published online ahead of print, 2021 Aug 8]. J Card Fail. 2021;S1071-9164(21)00306-7. doi:10.1016/j.cardfail.2021.07.012

Pulmonary artery

The PA pressure tracing shows a rapid rise in pressure, a systolic peak, a decrease in pressure after peak ejection, and a well-defined dicrotic notch from pulmonary valve closure during the pressure decrease. There should be no systolic pressure difference between the right ventricle and the PA unless stenosis of the pulmonary artery or pulmonary valve exists.

The shape of the PA wave, like other right heart pressure wave shapes, is subject to respiratory changes, thus patients on mechanical ventilation, with severe lung disease, morbid obesity or respiratory distress, can create substantial changes in intrathoracic pressure with noticeable differences in PA pressures during respiratory phases. Most experts consider that the end of expiration is the appropriate point to assess pulmonary artery (and other cardiac chamber) pressures because it is in this phase that intrathoracic pressure is closest to zero [21].

The PAPs is produced at the same time as the T-wave in the ECG and has been shown to be a parameter related to major cardiac events. A PAPs associated with a high heart rate determines a greater consumption of oxygen by the RV in type I PHT and, therefore, a greater risk of RV dysfunction [22].

The measurement of mean pulmonary arterial pressure (mPAP) is relevant for the diagnosis and management of pulmonary hypertension. According to the latest consensus of the Sixth World Symposium on Pulmonary Hypertension (WSPH), a value > 20 mmHg represents a cut-off point for the diagnosis of PHT, since adequate medical management from this value upwards shows benefits in survival [23].

Pulmonary capillary pressure

The PCP is measured directly in the absence of antegrade flow from the PA, so that it is transmitted from the left atrium, through the pulmonary veins and pulmonary capillary bed. It is usually a few millimeters of mercury below left atrial pressure (0 to 5 mmHg) [24,25]. Characteristics of a good wave include the presence of A- and V-waves, fluoroscopic confirmation of the location of the catheter tip in distal PA with the balloon inflated, observation of a mPAP curve when the balloon is deflated or the

catheter is removed from position, and an oxygen saturation with the balloon inflated > 90%, the latter being the most specific **(Table 2)**. If the catheter tip is poorly positioned in a peripheral branch of the PA with the balloon over distended, an "overstacking" phenomenon occurs and a false PCP curve with an oscillating line without A- and V-waves is obtained, which can lead to abrupt rupture of the PA [25].

In patients with mechanical ventilation with positive end-expiratory pressure (PEEP) >10 cmH_2O, there is a significant increase in alveolar pressure, which reduces the proportion of WEST's pulmonary zone 3, directly affecting the pressures of the right side, causing an overestimation of PCP [25]. Direct correction of PCP due to elevated PEEP is carried out by subtracting the esophageal pressure measured with an intraesophageal balloon from the PCP; however, there are other practical methods such as subtracting 2 to 3 mmHg of PCP for each 5 cmH_2O increase in PEEP or arguing that the corrected PCP is equal to the measured PCP minus half the quotient of PEEP divided by 1.36 [16,25,26].

If the PCP is high, the increase in the ratio between RA and PCP serves as an indicator of RV dysfunction and increased complications in advanced HF; its value is associated with increased pulmonary resistance and in-hospital mortality [26].

To assess whether the increase in PA pressure is due to an isolated elevation of pulmonary wedge pressure, the transpulmonary arterial pressure gradient was traditionally used, the value of which varies according to mPAP flow and LV filling pressures; currently, it is notably less relevant. However, the diastolic pulmonary arterial pressure gradient is a more accurate marker that allows adequate classification of PHT according to its pre- and post-capillary component; in addition, a high value is considered a predictor of mortality, poor prognosis and hospitalization for heart failure [24,27].

Finally, pulmonary vascular resistance (PVR) is a marker that has gained prominence since the sixth WSPH; and determines the presence of pulmonary vascular disease by being a better indicator of precapillary PHT, in contrast to the diastolic pulmonary gradient (DPG) [3]. It is also used as a therapeutic parameter in patients with congenital heart disease and in patients awaiting cardiac transplantation **(Figure 1)** [24,27,28].

Left ventricle (LV)

LV pressure has a rapid acceleration during initial systole, followed by a rapid decline. The initial diastolic pressure is low, while the final diastolic pressure increases slowly until the left atrium contracts, which represents the true left ventricular preload [29]. With the help of PAC, CO and thus cardiac index (CI) can be calculated by thermodilution and by Fick's principle [10] **(Figure 2).** The presence of a decreased CI is the main hemodynamic indicator for the diagnosis of CS. It can help to recognize a malfunction of MCS devices and to assess myocardial recovery aimed at weaning. Together with systemic vascular resistance (SVR), the CI allows differentiation of the various types of CS phenotypes [6]. With the values of CO, cardiac power (CP) can be calculated, which is the strongest independent hemodynamic marker of in-hospital mortality; the Shock trial reported CP within the risk stratification of patients with myocardial infarction, a value < 0.6 Watts is indicative of severe LV dysfunction [10,29].

On the other hand, the LV transmural filling pressure represents LV preload, as well as the difference between pulmonary capillary pressure (PCP) and pericardial pressure (PP), using CVP instead of PP for calculation. PCP varies with changes in LV transmural pressure and PP, so in a patient with HF it shows the pericardial restrictive effect on the LV preload [11,30].

There is also a biventricular assessment measure called systolic work index (SWI). This parameter evaluates ventricular work (energy) and has shown that its decrease in the LV is a predictor of poor prognosis and the need for greater hemodynamic support; in addition, the SWI of the RV is a predictor of RV failure in MCS and mortality in post-lung transplant patients [31] **(Figure 2).**

Intracardiac shunt (IS)

PA oxygen saturation greater than 75% may indicate the presence of a left-to-right IS, so it is recommended to measure the superior and inferior vena cava, RA (middle, high and low), RV and PA. Increased oxygen saturation ≥ 7% may be indicative of a left-to-right atrial shunt, whereas ≥ 5% may indicate a shunt at the level of the RV or the PA. The direct Fick method is the preferred means of CO measurement when a left-to-right IS is suspected [32].

Indications and usefulness

Cardiogenic Shock

Current studies suggest that the use of PAC in CS is associated with a benefit in patient morbidity and mortality; the American Heart Association (AHA) recommends the early use of PAC in the management of CS, especially in cases of refractoriness to treatment and/or therapeutic uncertainty [1,8,33,34]. A large retrospective study found that the use of PAC in CS was associated

RIGHT HEMODYNAMIC CALCULATIONS

RA/PCP RELATION	**NV <0.3**
>0.5 is associated to RV dysfunction and greater in-hospital mortality.	
PAPi = (sPAP – dPAP)/ CVP	**NV ≥ 2**
< 0.9 in inferior MI predicts RV failure and greater in-hospital mortality. <1.85 is a marker for poor prognosis and RV failure in heart failure under MCS.	
RVSWI = [((mPAP - CVP) x CI) x 13.6]/ HR	**NV = 5-10 g/m^2/beat**
<7.25 is a risk factor for decompensation and RV failure in heart failure with reduced LVEF. < 5 greater mortality in post MCS.	
PAD = SEV/(sPAP – dPAP)	**NV ≥ 2 ml/mmHg**
< 2.15 is associated with greater mortality in heart failure plus PHT II.	
PEAE = sPAP/SEV	**NV =0.7 – 1 mmHg/ml**
>1 greater mortality in PHT II and heart failure associated to the need for right ventricular load improvement.	
TGP = mPAP – PCP	**NV ≤ 12mmHg**
>12 mmHg mixed PHT II (pre – post capillary).	
PDG = dPAP – PC	**NV ≤ 5mmHg**
> 7 mmHg mixed PHT II, predictor of mortality and hospitalization due to heart failure.	

LEFT HEMODYNAMIC CALCULATIONS

CO = (SEV x HR)/1000	**NV = 4 – 6 L/min**
CI = CO/BSA	**NV = 2.5 – 4 L/min/m^2**
CI <2.2 L/min/m2 major diagnostic parameter of cardiogenic shock (SCAI C). CO < 4 L/min hypoperfusion state indicative of increased inotropic support.	
LVSWI = [((mSBP - PCP) x CI) x 13.6] /HR	**NV = 45 – 80 g/m^2/beat**
<18 g/m2/latido is associated to greater mortality and sub - optimal medical therapy.	
SEV = CO/(HR X 1000)	**NV = 60 - 100 ml**
SEVI =SEV/BSA	**NV = 30 - 65 ml**
Useful for postsurgical management and critical scenarios of increased cardiac demand and intravascular volume.	
SVR = [(mSBP - CVP)/CO]*80	**NV = 800 – 1500 dynas x seg x cm^{-5}**
Response of the systemic microvasculature to changes in the cardiac index. Useful in the differential diagnosis and classification of cardiogenic shock.	
CP = (mSBP x CO)/451	**NV > 1 Watt**
CPI = CP/BSA	**NV >0.5 Watts/min/m^2**
CP <0.6 Independent predictor of in-hospital mortality in cardiogenic shock. CPI <0.3 High mortality in cardiogenic shock and decompensation in advanced heart failure.	

mPAP> 20mmHG	**PVR < 3 UW**	**PVR ≥ 3 UW**
	PVR = (mPAP – PCP)/CO	
PCP ≥ 15 mmHg	Isolated PHT II	Mixed PHT II
PCP < 15 mmHg	High flow	Isolated pre – capillary PHT

Figure 2. Hemodynamic calculations by Swan-Ganz pulmonary arterial catheter.

RA: right atrium. **CS:** cardiogenic shock. **PAD:** pulmonary artery distensibility. **PEAE:** Pulmonary effective arterial elastance. **HR:** heart rate. **CO**, cardiac output. **PDG:** pulmonary diastolic gradient. **TPG:** transpulmonary gradient. **CI:** cardiac index. **MI:** myocardial infarction. **RVSWI:** Right ventricular systolic work index. LVSWI: Left ventricular systolic work index. **CPi:** Cardiac power index. **SEVi:** Systolic ejection volume index. **SEV:** Systolic ejection volume. PAP: Pulmonary artery pressure. **dPAP:** Diastolic pulmonary artery pressure. PAPi: Pulmonary artery pulsatility index. **mPAP:** Mean pulmonary artery pressure. **sPAP:** Systolic pulmonary artery pressure. **mSBP:** Mean systemic blood pressure. **CP:** cardiac power. **PCP:** Pulmonary capillary pressure. **PEEP:** Positive end-expiratory pressure. **CVP:** central venous pressure. **SVR:** Systemic vascular resistance. **PVR:** Pulmonary vascular resistance. **BSA:** Body surface area. **NV:** Normal values.

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with lower mortality and lower incidence of in-hospital cardiac arrest compared to those under conventional management [35]. Another multicenter study of similar characteristics demonstrated that PAC-guided management offers greater in-hospital survival in the various CS scenarios before the onset of MCS, because it allows recognition of the type of scenario, the need for drug titration, and the time of onset of MCS [8,36].

The recognition of the scenario and hemodynamic phenotype of the CS by PAC based on the assessment of CP, PAPi and LVEFP, has an important role in guiding the management strategy until patient recovery or the use of MCS; in addition, it helps to prevent the hemometabolic consequences of the hypoperfusion status and organ failure produced by CS **(Table 3)** [1,8,34].

Within these presentation phenotypes, CS due to RV failure is reported to account for 30% of all clinical presentations. In contexts such as this, the European Society of Cardiology (ESC) recommends the use of PAC in patients who do not respond to initial therapies (IIb C) or in case of diagnostic or therapeutic doubt. The usefulness of the PAC is essential for correct decision making in the preservation of an optimal euvolemic state and the decision of inotropic management and/or the use of MCS [36,37].

On the other hand, a substudy of the Shock trial reported that in patients with CS, SIRS is an important predictor of death and is associated with a non-significant increase in deaths with low SVR values, so PAC can be important for identifying this type of scenario, allowing early and appropriate management and follow-up [38].

There is benefit in measuring hemodynamic parameters by PAC in CS because they are useful predictors of mortality, as reported by the CardShock study, a prospective, multicenter registry that showed that HF, CPi, and indexed SV are strong predictors of 30-day mortality; likewise, PAC-guided management of CS is associated with more aggressive treatment without compromising 30-day survival [25]. Finally, a contemporary retrospective study reported a lower incidence of mortality, stroke, and hospital readmission associated with greater use of MCS and cardiac transplantation. Therefore, it is concluded that PAC in CS is associated with greater clinical benefit and greater use of advanced therapies [39,40].

Heart failure

Routine hemodynamic monitoring by PAC is not recommended in patients with HFrEF because, according to the ESCAPE study, it has not shown benefit on mortality during its initial routine use in patients with acute decompensated HF [25]. In 2019, a large retrospective study found an association between the use of hemodynamic monitoring in patients with HF without CS and elevated mortality; however, the same study describes a significant decrease in the mortality rate over time associated with increased use of advanced therapies and MCS, where the use of SGC proves to have a greater benefit [37]. Its usefulness becomes important in patients with HF whose perfusion or volume status is uncertain, especially in cases of advanced HF or CS, since literature describes benefit in its use by improving survival, decreasing complications and encouraging the timely use of advanced support [1,36,41].

On the other hand, approximately half of patients with HF have preserved ejection fraction (HFpEF), its diagnosis is based on the use of non-invasive parameters; however, according to the new ESC HF 2021 guideline, the confirmatory diagnostic test consists of the invasive evaluation of PCP at rest (>15 mmHg) and at exercise (>25 mmHg), although its use is limited and does not have a routine indication; PAC offers a relevant benefit when noninvasive markers provide low diagnostic sensitivity; moreover, it plays an important role in the approach to differential diagnoses, as well as additional information on the possible entities causing HFpEF [42,43]. Hemodynamic monitoring on exertion is useful since it directly assesses pressure variability and provides predictive value by calculating the PCP/CO slope. In hospitalized patients, an increase in PCP with the leg raise test is a good indicator of HFrEF and non-cardiac dyspnea [1,42].

In cases of acute mechanical complications, frequently due to myocardial infarction, PAC is usually not required, except in the context of a patient with hemodynamic instability, post-infarction heart failure progression or the diagnostic suspicion of an IS due to rupture of the interventricular septum when obtaining oxygen saturation between cardiac chambers (RA, PA, RV); likewise, PAC can serve as a confirmatory diagnostic tool for IS by calculating the ratio of systemic and pulmonary flow [44].

Pulmonary hypertension

The PAC is useful in PHT because it directly determines PA pressures. It was shown that patients who were in an apparent gray zone (mPAP: 21-24 mmHg) presented an increase in mortality, which is why the 6th WSPH proposed a change in the cut-off points for the diagnosis of PHT (mPAP > 20 mmHg) [5,45].

According to the ESC, the PAC is the gold standard to confirm the diagnosis of PHT because it allows defining the hemodynamic profile, severity, classification, and the approach for differential diagnoses [43]. Likewise, it allows additional evaluations such as the vasoreactivity test or hemodynamic monitoring on exertion, parameters with great relevance

during the assessment of prognosis, therapeutic orientation, and response to therapy.

Furthermore, PAC is useful in the diagnosis of exercise-induced PHT and is necessary to distinguish it from HFpEF. Recent studies have shown that these patients have worse clinical prognosis; however, early diagnosis and early initiation of treatment may improve survival [1].

Left ventricular assist devices (LVADs) and cardiac transplantation (CT)

The role of the PAC is fundamental in both scenarios. In the pre-CT evaluation, the assessment of the hemodynamic status of patients influences the presence of complications such as acute HF or post-CT death; the presence of precapillary component (PVR > 3.5 UW) is a relative contraindication, and its evaluation through a reversibility test stratifies the risk profile [1,16].

During LVAD consideration, a comprehensive RV assessment with hemodynamic predictors using PAC is important to assess the risk of RV dysfunction (42% of cases), as it has important repercussions on morbidity and mortality. Additionaly, during post-implantation management, it allows faster optimization of the device compared to clinical-echocardiographic follow-up; likewise, it provides better diagnostic accuracy when device-associated complications occur [9,32,46].

Other pathologies

PAC can be useful in patients with congenital and/or valvular heart disease when noninvasive evaluations are inconclusive and for the differential diagnosis between constrictive pericarditis and restrictive cardiomyopathy [1,9].

Conclusions

The SGC has currently proven to be useful in the management of various scenarios for both patients with cardiogenic shock and advanced heart failure as well as for patients with PHT, HF and even HFpEF due to its diagnostic utility and therapeutic support.

SCG monitoring and the interpretation of hemodynamic parameters are a relevant tool in patient management, capable of supporting the decision to provide advanced care, MCS and CT, and therefore it is regaining prominence in the cardiovascular area.

Contribution of the authors

All authors participated in the preparation, writing and correction of the manuscript, as well as in the elaboration of figures and tables.

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