

Echocardiography in Pulmonary Hypertension: The Fundamentals to Know by the Pulmonologist

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Abstract

While enormous advances have been made in the field of arterial hypertension, the definition remains unchanged, since it is a rise in pulmonary arterial pressure, which may be pre or post capillary, but that It will be necessary to confirm by a right arterial catheterization. Echocardiography remains the first time to reinforce or refute our clinical suspicion of arterial hypertension, thanks to the information that it gives us, the speed of tricuspid insufficiency, the speed of pulmonary insufficiency, cardiac output, pulmonary resistance, as well as etiological research such as left heart disease, and congenital heart disease. In this article, we will detail the pathophysiology of pulmonary arterial hypertension, as well as the methodology to calculate these pressures and these speeds, this will clarify to young pneumologists who have forgotten their notions of cardiology to fully understand what read in a report prepared by a cardiologist and why not in the near future to practice themselves echocardiography in this area.

Keywords: *Pulmonary Arterial Hypertension; Cardiac Preoperative; Pressure Measurement and Tricuspid and Pulmonary Failure Rates; Pulmonary Arterial Pressure Calculation; Pulmonary Resistance*

Abbreviations

AP: Pulmonary Artery; Copd: Chronic Obstructive; Dc: Continuous Doppler; DP: Doppler Pulsed; ECG: Electrocardiogramme; EFR: Respiratory Functional Exploration; ETT: Transthoracic Cardiac Ultrasound; ETO: Trans-esophageal Echocardiography; Nardy the Ejection Fraction of the VD; Lvef: Ejection Fraction of the VG; Pah: Primary Pulmonary Hypertension; HTP: Pulmonary Hypertension; IM: Mitral Regurgitation; IP: The Velocity of Lung Leakage Pulmonary Insufficiency; IT: Tricuspid Deficiency; ITV: Full Time Speed; Mode TM: Mode Time Movement; OD: Right Atrium; OG: Left Atrium; MPAP: Average Arterial Pressure; Pcap: PCP/Pulmonary Capillary Pressures; PaO₂: Partial Oxygen Blood Pressure; Pdep: Diastolic Pulmonary Arterial Remission; PAPS: Systolic Pulmonary Arterial; PAPO: Pulmonary Arterial Pressure Occlusive; POD: Pressure; PSGA: Arasternale Large Axis (Cut Ultrasound Para Sternal Left); PSPA: Arasternale Small axis (Cut Ultrasound Para Sternal Left); Right Atrial; Pvr: Pulmonary Vascular Resistances; RP: Pulmonary Shrinkage; SIV: Inter-ventricular Septum; TAP: Lung Acceleration Time; Taps: Amplitude of Systolic Displacement of the Tricuspid Ring; VG: Left Ventricle; VD: Right Ventricle; VCS: Superior Vena Cava; VCI: Inferior Vena Cava; VmaxIT (From IT Stream): Maximum Speed of Tricuspid Deficiency Flow; VSH: Hepatic Veins

Introduction

Pulmonary Hypertension (PHT) is a hemodynamic and physiopathologic state characterized by elevated mean arterial pressures (MPAP) > 25 mmHg, measured by catheterization heart rate, with concomitant assessment of pulmonary capillary pressures (Pcap). We will thus distinguish the pre-capillary HTP (Pcap < 15 mmHg) postcapillary HTP (Pcap > 15 mmHg). The improvement in performance of echocardiography in recent years has clearly refine the management of pulmonary hypertension [1-14].

This non-invasive examination aims to:

- Screening
- The positive diagnosis when there is a high clinical probability.
- Etiological diagnosis.
- Assessment of the impact and signs of poor prognosis.
- Monitoring.

We will detail in this article what we search in the echocardiogram to calculate pulmonary arterial pressure.

Reminder:

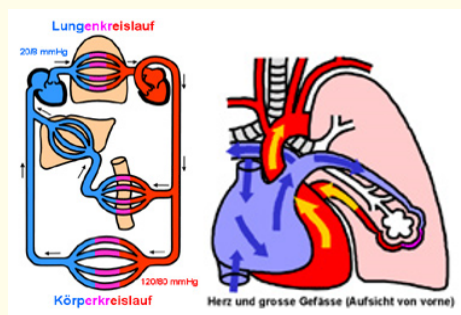
Reminder on the pulmonary circulation

The heart is a tissue organ located between the two lungs in the mediastinum. 1/3 is represented by the right heart and 2/3 by the left heart. Similarly, the ventricular walls are much thicker than the atria, as far as where the expulsion of the blood volume requires a force sufficient to fully irrigate the body through the great circulation (circulation at high pressure). the right heart and the left heart do not communicate with each other except in case of malformations. The left heart composed of the left ventricle and the left atrium (LA). At the posterior surface of the LA arrive the pulmonary veins (the only veins of the body which contain oxygenated blood), this oxygenated blood a Once in the LA, will go into the LV at the time of atrial systole, then from LV to the aorta at the time of ventricular systole, then will serve to irrigate all tissues of the body.

The right heart in turn, includes the right atrium (RA) and the right ventricle (RV). These two cavities communicate through an atrio-ventricular valve: the tricuspid valve, whose role is once closed to prevent the return from the blood of the RV towards the RA.

At the level of the RA in its upper part arrives the superior vena cava (SVC), in its posterior part, below the SVC, arrives the inferior vena cava (IVC). At the level of the atrioventricular orifice of the RV the trunk of the pulmonary artery (PA) and its two branches, the only arteries of the body that contain deoxygenated blood, this blood will be transported to the lung (small circulation), once the gas exchange made oxygenated blood returns to the LA level via the pulmonary veins and the cycle begins again. The pulmonary circulation is the functional circulation of the lung it comprises the capillary circulation, and the venous circulation which flows into the LA, the bronchial circulation being connected in series with the systemic circulation, the flow of the right ventricle adapts to that of the left ventricle and is therefore virtually equal to systemic flow.

The bronchial circulation is the nourishing circulation of the lung it comes when it from the aorta It is drained in the pulmonary veins, resulting in a physiological shunt by contamination of the arterial blood by bronchial blood. This explains in part (with the effect physiological shunt), that the oxygen partial pressure of arterial blood (PaO₂) is slightly lower than the alveolar oxygen pressure (PAPO).



Figure

Reminder on pulmonary hypertension

What we know

Normal pulmonary circulation is the “functional lung circulation.” It is a high-compliance, low-pressure system (mean pulmonary arterial pressure (PAPm) 14 ± 3 mmHg) with low resistance, homogeneous distribution of blood in the pulmonary capillaries and thus gas exchange, while limiting the work of the right ventricle. Pulmonary hypertension (PH) is defined as mean arterial pulmonary pressure (mPAP) greater than or equal to 25 mmHg measured at rest during right cardiac catheterization (Table 1). This vascular disease is characterized by progressive elevation of pulmonary vascular resistance leading to right heart failure and death.

Pulmonary Arterial Hypertension (PAH): PAPm ≥ 25 mmHg, Pulmonary Capillary Pressure (PCP) ≤ 15 mmHg and Pulmonary Vascular Resistance (PVR) > 3 Wood Units.

Definition	Hemodynamic criteria	Classification groups
HTP-Precapillary	Pulmonary Arterial Pressure: PAPm ≥ 25 mmHg PCP ≤ 15 mmHg Normal or decreased heart rate	Group 1: PAH Group 3: HTP associated with respiratory diseases Group 4: Chronic thromboembolic HT Group 5: Multifactorial HTP
HTP-postcapillary	PAPm ≥ 25 mmHg PCP > 15 mmHg Normal or decreased heart rate	Group 2: HTP of left heart disease

Table 1: Hemodynamic Definitions of Pulmonary Hypertension (HTP).

Clinical classification of pulmonary hypertensions

The international clinical classification of Pulmonary Hypertension (Table 2) distinguishes 5 groups of HTP, in which are grouped different pathologies sharing similarities in their clinical presentation, physiopathology, hemodynamic characteristics and therapeutic strategy.

<ol style="list-style-type: none"> 1. Pulmonary Hypertension (PAH) <ol style="list-style-type: none"> 1. Idiopathic 2. Inheritable 3. Induced by drugs or toxic 4. Associated with various pathologies. 5. Persistent pulmonary hypertension of the newborn <ol style="list-style-type: none"> 1. Veno-occlusive disease and/or pulmonary hemangiomatosis 2. Persistent pulmonary arterial hypertension of the newborn 2. Pulmonary Hypertension of Left Heart Diseases <ol style="list-style-type: none"> 1. Left ventricular systolic dysfunction 2. Left ventricular diastolic dysfunction 3. Valvulopathies 4. Congenital or acquired cardiomyopathies. 5. Congenital or acquired stenoses of the pulmonary veins <ul style="list-style-type: none"> • Pulmonary Hypertension of Chronic Respiratory Diseases. • COPD

Table 2: Clinical Classification of Pulmonary Hypertension (ESC/ERS guidelines 2015).

(This classification is given as an indication and only the data identified in bold are known in the 2nd cycle).

Physiopathology: the right heart and pulmonary hypertension

The functioning of RV is at the center of the clinical pathophysiology of HTP. In a patient with PH, one observes successively:

Myocardial hypertrophy, increased resistance of pulmonary veins (PVR): Pulmonary arteries lose their elasticity. Consequently, VD, unable to provide sufficient cardiac output, is hypertrophied in response to the chronic increase in afterload.

Systolic dysfunction of slowly progressive VD: Possible mechanisms involved in the progression of VD dysfunction are: ischemia, changes in the expression of genes encoding sarcomere proteins, and activation of the renin-angiotensin system. However, abnormal post-load elevation remains the main determinant of VD dysfunction in patients with HTP.

An expansion of the VD: To compensate for the alteration of the RV ejection fraction (FeRV) and the rise of the afterload. - and diastolic dysfunction; VD can no longer ameliorate venous return changes by maintaining constant pulmonary flow. Hypovolemia leads to decreased pulmonary flow and hypoxemia. The decrease in cardiac output is also due to the appearance of a tricuspid insufficiency (TI) caused by the dilation of the RV and thus of the tricuspid ring.

Diastolic LV dysfunction: With hypertrophy and dilatation, the RV gradually becomes more spherical, widens (especially in the transverse direction), and the interventricular septum flattens to become paradoxical, which favors the diastolic dysfunction of LV. On the other hand, an alteration of the LV ejection fraction (FeLV) is, although possible, relatively rare.

The increased resistance of the pulmonary vasculature is caused by obliteration of pulmonary vascularization and/or pathological vasoconstriction, proliferation of endothelial cells and smooth muscle, hypertrophy and chronic inflammation, leading to remodeling of the wall vascular. It is believed that vasoconstriction is due in part to the increased activity of thromboxane and endothelin-1 (two vasoconstrictors) and reduced prostacyclin and nitric oxide activity (two vasodilators). The increase in pressure in the pulmonary vasculature that results from the vascular obstruction thus created, damages the endothelium. The lesion then activates coagulation at the level of the intimal surface, which can aggravate pulmonary artery hypertension. This results in local coagulopathy at the endothelial surface that should not be confused with chronic pulmonary hypertension thrombo-embolic.

The diagnostic strategy of pulmonary hypertension

The diagnosis of HTP is based on a rigorous clinical procedure comprising 3 stages including the data of the interrogation, the clinical examination and the results of complementary explorations.

Detection and confirmation of HTP (positive diagnosis): in the at-risk population who is:

- First-degree relative of a case of idiopathic PAH.
- Diffuse or limited systemic scleroderma or other connective tissue diseases.
- Sickle cell disease.
- Congenital heart disease.
- Pre-liver transplant checkup.
- HIV infection

The clinical signs are not specific: exercise dyspnea, chest pain, lipothymia and syncope of effort, asthenia, palpitations, hemoptysis.

Physical Signs

Signs of HTP: Holosystolic murmur of tricuspid insufficiency rising to deep inspiration (Carvallo's sign), B2 glow in the pulmonary focus, diastolic breath of pulmonary insufficiency.

Signs of right heart failure complicating the HTP: Tachycardia, galloping, jugular turgor, hepato-jugular reflux, hepatomegaly, edema of the lower limbs, anasarca.

Usual paraclinical examinations requested during a clinical suspicion of a PH

Even if they are not very sensitive, some elements found on the usual paraclinical examinations carried out as part of the dyspnea assessment can lead to a PH.

- Thoracic imaging: dilation of the pulmonary arteries, enlargement of the right heart. Normal radio does not invalidate the diagnosis.
- ECG: signs of right ventricular hypertrophy (Right axial deviation $> 110^\circ$, R/S in V1 > 1 where R in V1 > 7 mm, S in V1 < 2 mm, R in V1 + S in V6 > 10 mm, ST/t current change the derivations RV or rSR' en V1 with $R' \geq 10$ mm). Troubles of rhythm. A normal ECG does not exclude the diagnosis.
- VA / Q lung scintigraphy, EFR, thoracic angioscan.
- Biological tests: nonspecific.
- Doppler-coupled transthoracic cardiac echography (ETT): This is the non-invasive reference examination for any suspicion of HTP. The new ESC-ERS 2018 recommendations specify its role, mainly in screening by the speed of tricuspid insufficiency at rest, the etiological diagnosis of HTP and classification according to whether or not the presence of an associated pathology as well as the assessment of severity (diagnosis of severity).
- Right heart catheterization which represents the essential examination to confirm the diagnosis of pulmonary hypertension.

Echocardiography: a key non-invasive examination:

Doppler echocardiography is an imaging modality that allows the study of hemodynamic parameters in a non-invasive way. It is the non-invasive examination of reference to any suspicion of HTP. The reliability of the measurements nevertheless depends on a meticulous approach and a good understanding of the principles of Doppler and the dynamics of the fluids.

General principles

The Doppler principle establishes that the frequency of a reflected ultra-sound is altered by a moving target, such as a red blood cell. The amplitude of this Doppler shift is related to the speed of the blood cells, while its polarity reflects the direction of the blood flow, to (positive) or the opposite (negative) of the transducer. Currently, Doppler echocardiography comprises 3 modalities: pulsed Doppler (DP), continuous Doppler (DC) and color Doppler. The DP measures the flow velocities in a specific area. The DC can record very high flux rates, but it does not locate the point of origin of the velocities along the ultrasonic beam. Color Doppler uses DP technology over several regions of interest in the area covered by the ultrasound beam. In each of these regions, an estimation of the flow velocities is superimposed on a two-dimensional (2D) image using a color scale representing the direction of the flow, the average velocity and sometimes the variance of the velocity.

What is the point of measuring these flows and speeds?

The heart and the big vessels are likened to a pump with pipes, if we imagine that every two spaces in the heart and the vessels (separated by the valves) are a garden hose, then the flow velocity in this pipe through its orifice, will increase each time the diameter of the orifice decreases or there is a resistance downstream.

When the color Doppler shows a zone of aliasing that predicts the existence of a valve leap, by coupling it with the continuous Doppler, it will specify us the line where the velocity is maximum, without predicting the exact point.

As we have seen in the pathophysiology of pulmonary hypertension that there is a change in the structure of the pulmonary artery wall that results in an increase in resistance to blood flow from RV to AP through pulmonary valves, therefore an increase in the post load, the RV will react by hypertrophying, then after exhaustion dilating. RV dilatation will consequently result in a tricuspid insufficiency, then a hypertrophy of the RA which will try to compensate for this overload RV.

Doppler echocardiography therefore evaluates the speed of blood flow by relying on the displacement of red blood cells.

- Allows to estimate systolic pulmonary arterial pressure (PAPS) thanks to the evaluation of VMaxIT (of the IT flow) and the estimate of the right atrial pressure (Pr RA).
- Makes it possible to evaluate other indirect parameters suggestive of HTP such as the velocity of the pulmonary leak (IP), the impact on the right cavities (right ventricular hypertrophy (RV), RV dilatation, paradoxical septum, displacement amplitude systolic tricuspid ring (TAPSE), impaired right ventricular systolic function, dilation of the RA.
- Participates in the etiological assessment by looking for an alteration of systolic or diastolic left ventricular function, valvular pathology or congenital heart disease (inter-auricular communication, interventricular communication...).

Echocardiography in pulmonary hypertension from screening to prognosis

Screening and diagnosis of pulmonary hypertension

Measurement of PAPS from tricuspid insufficiency flow (TI=IT)

It should be known that 65% of normal subjects have a pulmonary leak (IP). 65% have a tricuspid leak (IT) physiological, 85% have IP or IT and IT is present in 95% in case of HTP. The speed of the leakage of the IT is all the more important as the pressure gradient between the VD and the OD is important: $\Delta P = PR_{VD} - PR_{OD}$. In the absence of pulmonary stenosis: RV pressure in systole = Systolic pulmonary artery pressure. we then have:

$PAPS = Pr_{VD} \text{ sys} = \Delta P + Pr_{OD} = 4 \times (V_{max} \text{ IT})^2 + Pr_{OD}$ According to the continuity equation of Bernouilli Where $PapS =$ systolic arterial blood of the pulmonary artery, $\Delta P = pr_{VD} - Pr_{OD} = V_{max}^2$: Maximum IT speed

The calculation of the PAPS is then done on an apical cut 4 cavities cardiac probe at the level of the peak shock figure 1, or left parasternal section (second intercostal space), in continuous doppler mode on the tricuspid valve to measure the maximum speed of In practice, we add a fixed value of 10 mmHg in normal adult subjects, 15 or 20 mmHg in case of right heart failure.

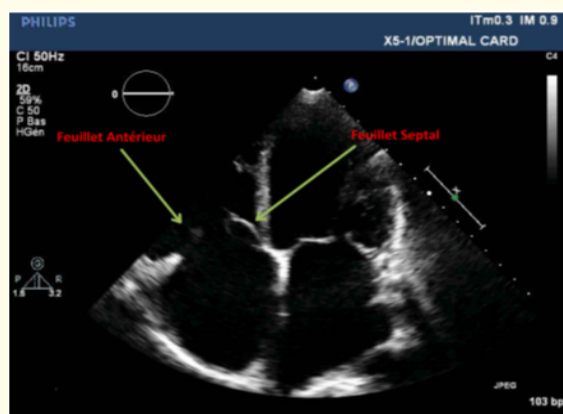


Figure 1: Apical cut 4 cavities (we see the two sheets of the tricuspid valve).

The pressure RA (PRA) is also calculated from the diameter of the VCI in the costal mode (TM mode) in the costal section at 3 - 4 cm from the end of the RA and we look at the respiratory movements of the IVC, normally the diameter of the inferior vena cava varies between 17 - 19 mm is reduced by 50% in inspiration and is maximal in expiration.

- If the diameter of the VCI < 17 mm and collapse ≥ 50%, the PrOD = 05 mmhg.
- If the diameter of the VCI > 17 mm and collapse ≥ 50%, the PrOD = 10 mmhg.
- If the diameter of the VCI > 20 mm and collapse ≥ 50%, the PrOD= 15 mmhg. This estimate is valid only in spontaneous ventilation. It is unusable in patients with assisted ventilation. We can more precisely use the formula PRA = 1.7 × E/Ea + 0.8): but this method is rather reserved for cardiologists.

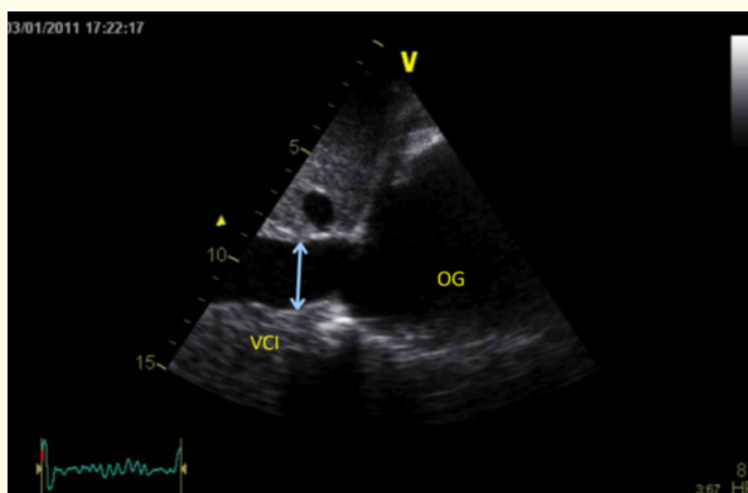


Figure 2: Measurement perpendicular to the large VCI at the end of exhalation, near the junction of the hepatic veins at about 0.5 - 3 cm from the entrance of the OD.

VIT (m/s) PTRV	Estimated PAPs* (mmHg)	Other HTP** Signs HTP	Probability	Recommendation Level/ level of evidence
≤ 2.8	≤ 36	No	Unlikely	I-B
≤ 2.8	≤ 36	Yes	Possible	Ila-C
2.9 - 3.4	37 - 50	Yes /No	Possible	Ila-C
> 3.4	> 50	Yes /No	Likely	I-B

Table 3: Suspicion of pulmonary hypertension at the cardiac echo-Doppler.

Other evocative echocardiographic signs of HTP: increased speed of PI, short APT and later signs: dilatation of right cavities, abnormal shape and paradoxical movement of SIV, parietal hypertrophy of VD, and dilation of AP.

Right heart catheterization is practiced as soon as there is a probability of HTP.

In summary:

$$\Delta P \text{ s RV/ RA} = 4 V_{\text{max}}^2 \text{ IT}^2$$

$$\text{PVDs} = \Delta P \text{ s RV/RA} + \text{POD}$$

PAPs = PRVs (in the absence of pulmonary stenosis)

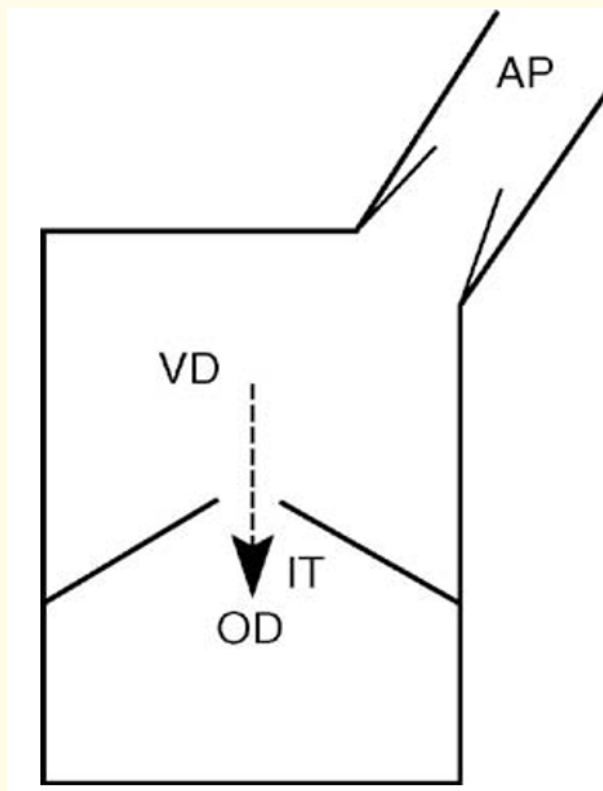


Figure 3

$$PAPs = 4 V_{max} IT^2 + POD$$

PAH = PAP > 35mmHg at rest and low LV filling pressures. Low LV filling pressures favor the pre-capillary origin but sometimes the filling deficit can unmask post-capillary HTP.

The absence of detectable IT does not mean no PAH (false reassurance).

The limits of this method are:

- The absence of tricuspid insufficiency
- An error in ΔP measurement or RA pressure.
- A major tricuspid leak because the speed of the jet regurgitation will be reduced which goes under the estimated PAPS.
- Stenosis of the pulmonary artery: pulmonary narrowing (PR).
- Increase in PAPS in elderly, hypertensive and obese patients In these cases, the method must be changed.

Estimation of the PAPS from the pulmonary insufficiency (IP) flow

If the PAPS could not be directly estimated from the IT flow, it can be estimated from the PAPm and PAPd values calculated from the IP flow by the empirical formula:

$PAPs = 3PAPm - 2PAPd$ The PAPS thus calculated is well correlated with that obtained from the IT Flux. The MPAP can be measured directly on the IP or on the IT ($MPAP = 0.61 PAPS + 2$) Is On IT and IP ($MPAP = (PAPS + 2 PDEP)/3$ Yes From the protodiastolic velocity of the IP recorded in continuous Doppler ($Vpd ip$):

$$PAPm = 4 Vpd IP^2 + POD$$

Age	N	PAPS repos mm Hg
< 40 yrs	8	24.5 ± 5.5
41 - 50 yrs	31	25.0 ± 5.5
51 - 60 yrs	30	28.0 ± 5.2
61 - 70 yrs	12	34.0 ± 4.1
71 - 80 yrs	18	33.00 ± 6.6

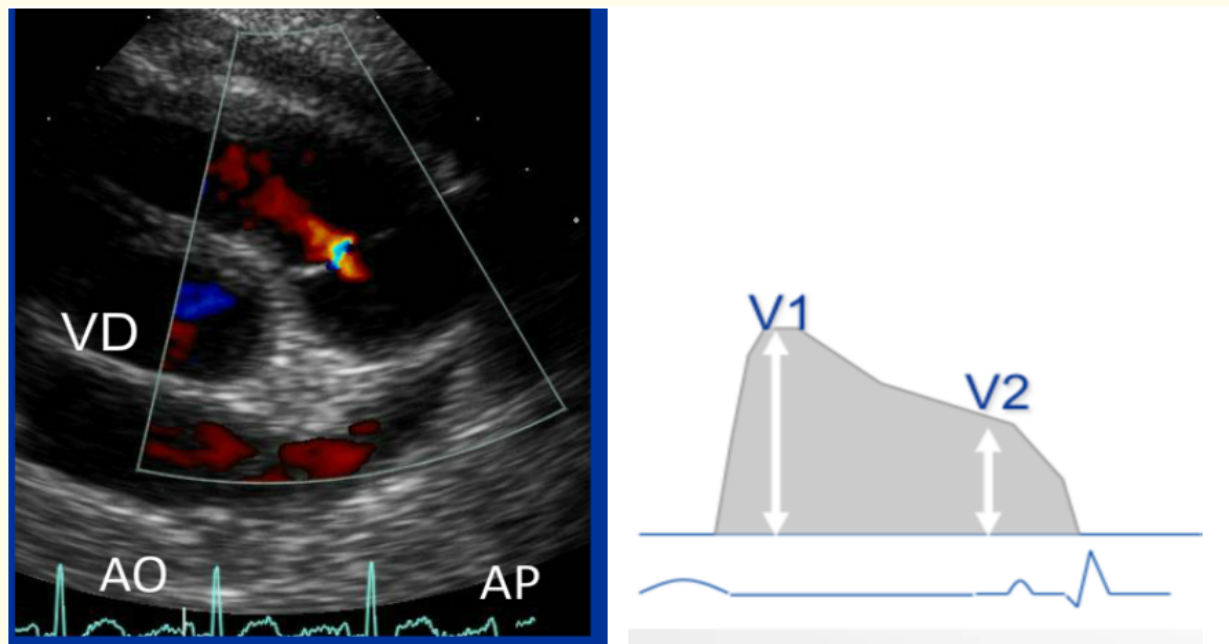


Figure 4

In summary:

$$\text{PAP Diastolic} = 4V_2^2 + \text{POD}$$

$$\text{PAP m} = 4V_1^2 + \text{POD}$$

$$\text{PAP systolic} = 3\text{PAPm} - 2\text{PAPd}$$

Pressure blood pulmonary diastolic	<ul style="list-style-type: none"> • PSPA incidence, modified PSGA continuous Doppler flow • pulmonary insufficiency (PI) 	Peak speed diastolic IP flow (V2)	$\text{PAPd} = 4 V_2^2 + \text{pres-sion OD}$	
Pressure blood pulmonary average	<ul style="list-style-type: none"> • PSPA incidence, modified PSGA continuous Doppler flow • pulmonary insufficiency (PI) 	Peak speed protodiastolic (V1)	$\text{PAPm} = 4 V_1^2 + \text{POD}$	< 25 mmHg

In the absence of IT and IP one calculates PAT: The pulmonary acceleration time it is the time between the beginning of the passage of the GR of the RV towards the PA and the time where it stops at the level pulmonary valves, at the time of closure of the valves, that is to say when the Pr of the PA becomes greater than that of the RV. And since we want to evaluate the speed of passage of the red blood cell to a specific point, we will use pulsed Doppler at the level of the entry of the pulmonary artery.

A short PAT appearance (< 100 ms) with meso-systolic notch is very suggestive of precapillary HTP at high pulmonary pressures.

A short TAP at normal pressures is suggestive of an acute pulmonary embolism. A TAP> 100 ms has a good negative predictive value to eliminate a PH.

Note: It is always necessary to confirm its measurements by comparing 2 measures (in general: at the level of the IT and the IP).

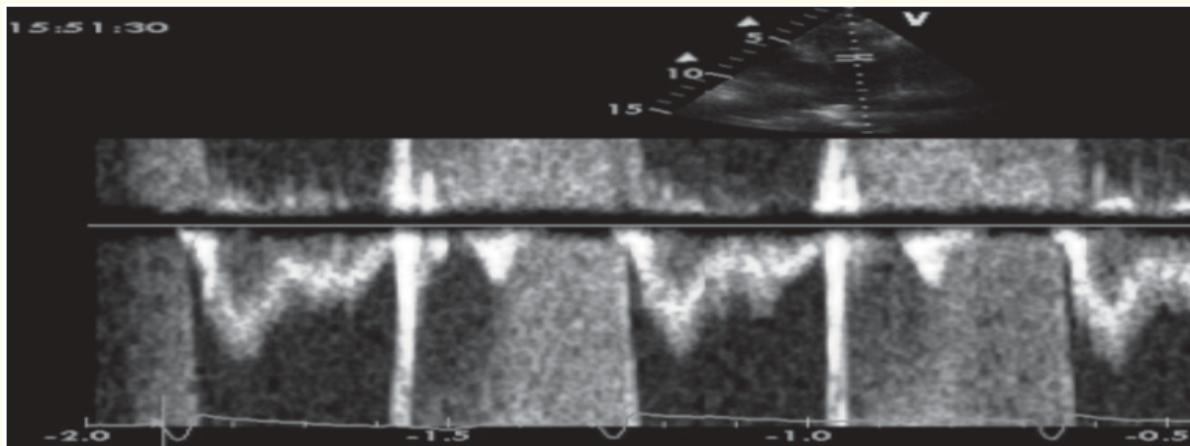


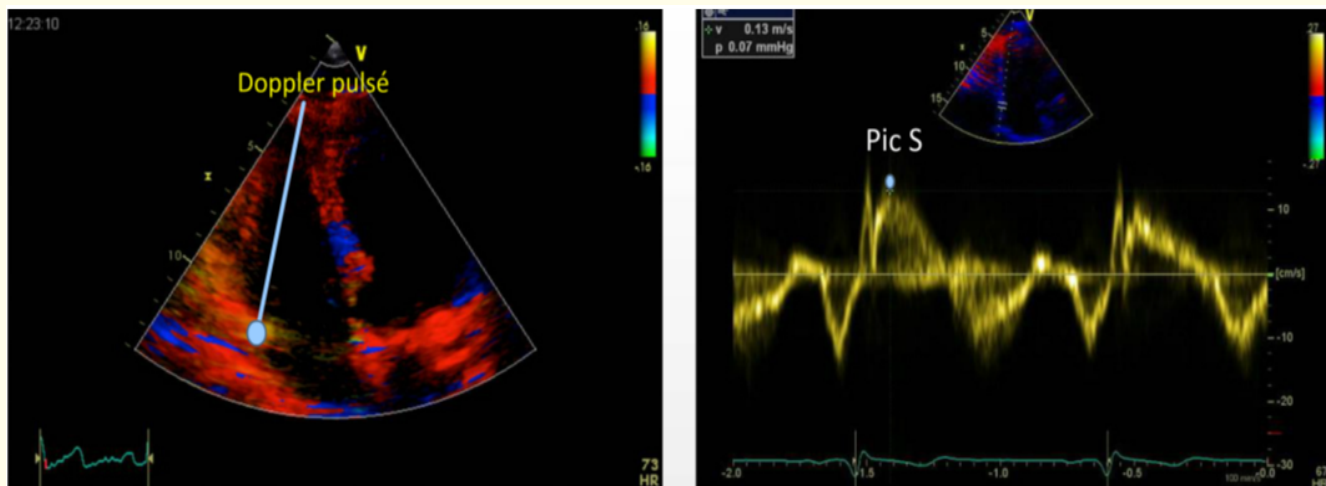
Figure 5: Measurement of pulmonary acceleration time.

Calculation of the TAPs: Tricuspid amplitude of the systolic excursion of the plane of the ring (TAPs). Measured in TM in the lateral portion of the tricuspid ring or tissue Doppler derived mode: tissue tracking, it reveals to be a useful index for evaluating the longitudinal function of DV and is a good reflection of its contractility (systolic function). This displacement is related to the contraction of the Wolf spur and exceeds 20 mm in the normal state. It is more important than that other walls that move only 6 mm. It is particularly interesting in clinical practice given the ease with which it is measured using the TM mode; the cursor is placed in such a way that it crosses the lateral tricuspid ring in an apical four-cavity cut. The TAPSE measures the movement of the tricuspid valve towards the apex of the RV during systole. this old index to a renewed interest, because of its prognostic value found recently in the PHT (threshold at 15 and 18 mm).

Methods	Tissue Doppler with tricuspid ring measures systolic S-wave peak
Advantages	Simple Reproducible (Except middle segment and apical)
Limits	Dependent angle Invalid VD segmental anomaly faces

Calculation of pulmonary vascular resistance PVR

In systole, the pulmonary vascular tree must absorb the entire systolic volume because the mitral valve is closed, which is why PCRs are ten times lower than SAR. When cardiac output increases with effort, pulmonary vascular resistance decreases in order to contain this excess volume, but the decrease in RAP cannot be significant since the pulmonary bed is already in active vasodilatation at rest. The PAP therefore amounts to effort. An excessive increase of PAP to exercise is an early sign of PAH. On the other hand, its increase at rest is a late sign since 50% of the traffic must be obstructed so that it remains permanently elevated.

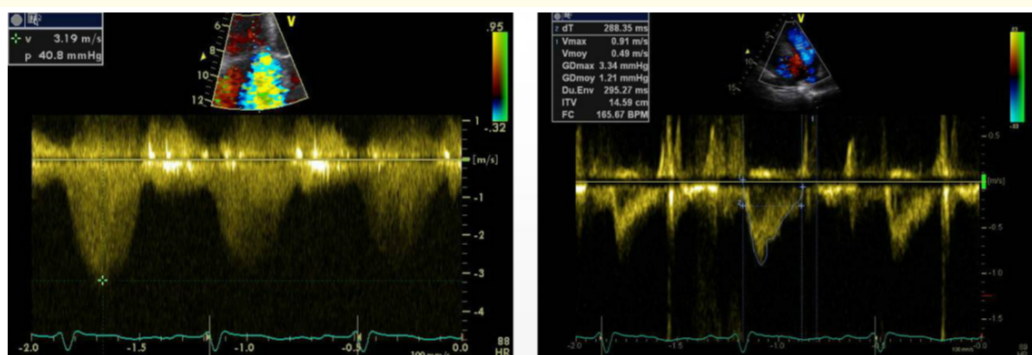


Dysfonction VD si pic onde S < 10 cm/s

Figure 6: Measurement of TAPSE

Peripheral vascular resistance (PVR) measurement in echocardiography:

- $PVR \text{ (united woods)} = 10 \times V_{\max} \text{ TI/ITS under pulmo} + 0.16$
- $V_{\max} \text{ TI/ITS under pulmo} \geq 0.2$: High PVR
- $V_{\max} \text{ TI/ITS under pulmo} < 0.2$: Normal PVR even if HTAPAt the end of this chapter, we have understood how useful it is, and how to detect pulmonary hypertension, which it will of course be necessary to confirm by right heart catheterization.



Resist pulmonary vascular	<ul style="list-style-type: none"> • PSPA incidence, doppler pulse under pulmonary (in pulmonary chamber of pulmonary rejection) • Incidence apicale 4 cavites, PSPA, PSGA modifiee, Doppler continu flux IT 	<ul style="list-style-type: none"> • ITV under pulmonary • Maximum speed of the IT 	$(V_{\max} \text{ IT/ITV sous pulm}) + 0,16$	$V_{\max} \text{ IT/ITV sous pulm} < 0,2$
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Figure 7: Measurement of resistance of pulmonary veins

Etiological diagnosis

Following the algorithm below, the first step when finding pulmonary hypertension is to look for an etiology of a post-capillary PHT that can be potentially reversible in order to treat it: the most common etiologies are left-sided heart disease, respiratory causes, hypertension secondary to thromboembolic diseases, then look for the etiologies of pre-filarial pulmonary hypertension in group 1 and 5.

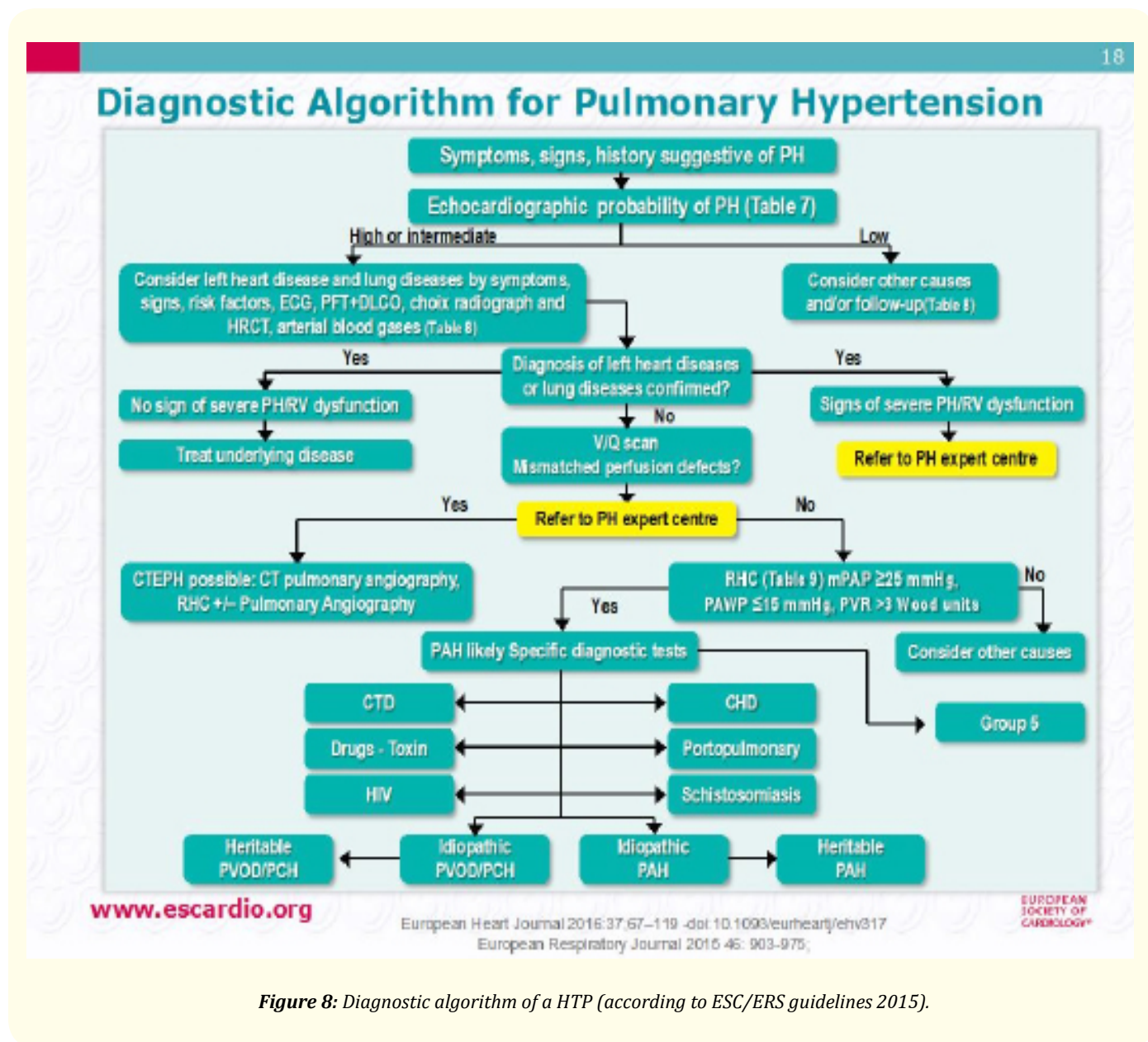


Figure 8: Diagnostic algorithm of a HTP (according to ESC/ERS guidelines 2015).

This research is done by evaluating the function of the LV (systolic function: LVEF: LV ejection fraction that must be ≥ 55%, eliminate left valvulopathy, mitral regurgitation (IM).

Search for a left heart disease
Left valvulopathies: mitral regurgitation, mitral stenosis, aortic stenosis and aortic insufficiency, which are seen in color doppler (alliazing).
Systolic dysfunction of LV (Calculation of LV ejection fraction which must be ≥55%.
Evaluation of left ventricular filling pressures (pulsed Doppler mitral flow analysis which is the cornerstone of assessment of LV diastolic function, pulmonary venous flow, left ventricular filling in TM color, tissue Doppler the mitral ring, and the surface of the left ear).

In the absence of left heart disease, search	
Post-acute pulmonary heart embolic	Absence of parietal hypertrophy of RV
	IT flow rate typically < 3.5 m/s
Chronic pulmonary heart post-embolic or respiratory	Presence of parietal hypertrophy of RV
	IT flow speed can be > 3.5 m/s
	Pulmonary stenosis to be eliminated (table VD pressure overload without PAH)
Shunt left-right	Visualization of the shunt in color doppler
	ETO interest to visualize the defect when the velocity of the shunt is low because of PAH, or that the shunt is difficult to visualize by flying transthoracic (sinus venosus)
Primitive HTAP	Diagnosis of exclusion (absence of heart disease left, shunt, pulmonary heart respiratory or post-embolic)

Awareness and prognosis

Criteria Prognostic (Mortality Estimated 1 year Old)	Risk Low < 5%	Risk Intermediate 5 - 10%	Risk Thieve > 10%
Signs of decompensation Heart Right	Absence	Absence	Presence
Progression of Symptoms	Not	Spring	Fast
Syncope	Not	Occasional	Repeats
Class Functional OMS	I, II	III	IV
Test of Walking of 6 minutes	> 440m	165 - 440m	< 165m
Event of effort Cardio Lung	Pic VO ₂ > 15ml/min/kg (> 65% Before.) VE/VCO ₂ < 36	Pic VO ₂ 11-15ml/min/kg (35-65% Before.) VE/VCO ₂ 36 - 44.9	Pic VO ₂ < 11 ml/min/kg (< 35% Before.) VE/VCO ₂ ≥ 45
Rate Plasma of NT- Probnp	BNP < 50 ng/I NT-proBNP < 300 ng/I	BNP 50-300 ng/I NT-proBNP 300-1400 ng/I	BNP > 300 ng/I NT-proBNP > 1400 ng/I
Imaging (Ultrasound MRI)	OD Area < 18 cm ² Not Effusion Pericardiac	Area OD 18-26 cm ² Not Yes Effusion Pericardiac Minimal	Area of > 26 cm ² Enhancement Pericardiac
Hemodynamic	POD < 8 MmHg Ic 2.5 l/min/m ² SvO ₂ > 65%	POD 8-14 mmHg IC 2,0 - 2,4l/min/m ² SvO ₂ 60-65%	POD > 14 MmHg IC < 2,0l/min/m ² SvO ₂ < 60%

Table 9: Clinical, Radiological and hemodynamic prognosis factors.

Pulmonary arterial hypertension. College of Respiratory Teachers - 2017. Item 222 the echocardiographic prognostic criteria of pulmonary hypertension are:

- The size of the right atrium (risk of death increased by 40% if the right atrium area ≥ 26 cm²).
- The presence of a pericardial effusion increases the risk of death by 40%).
- The index of diastolic eccentricity.
- Tei index (poor prognosis if ≥ 0.83).
- The amplitude of the systolic excursion of the plane of the tricuspid ring (survival <30 months if TAPSE < 18).

Conclusion

An essential examination for PAH screening, the Doppler ultrasound still suffers from its daily difficulty in asserting, formally, an increase in pulmonary pressures and its ability to discriminate pre- or post-capillary character. Hence its role ultimately limited to diagnosis in the new recommendations, based on the velocity of IT. However a systematic and standardized analysis of Doppler and ultrasound parameters bring a lot of information quantifying the adaptation of the geometry and the right and left function, approach directly or indirectly the pulmonary pressures, the ventriculoarterial coupling. A big work has been realized these last years, in France, under the impetus of the national reference center and expert centers, allowing a better knowledge

The role of cardiac echo-doppler in the positive diagnosis and not only in screening. Obviously, the need is for the realization of longitudinal studies, with centralized reading, with systematic and exhaustive analyzes of ultrasound parameters including the new parameters of quantification of the function VD in particular those derived from tissue Doppler. This work should focus on determining reliable, reproducible parameters for participating in patient follow-up and prognosis. It seems clear that from a more global point of view, the ultrasound items must integrate a gravity score combining clinical, biology, ultrasound and hemodynamics that should only be understood by the cardiologist.

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