REVIEW ARTICLE

DIAGNOSTIC AND PREDICTIVE VALUE OF RIGHT HEART CATHETERIZATION-DERIVED MEASUREMENTS IN PULMONARY HYPERTENSION

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ABSTRACT

Right heart catheterization is a unique tool not only in the diagnosis but also in the management of patients with a wide range of cardiovascular diseases. The technique dates back to the 18th century, but the biggest advances were made in the 20th century. This review focuses on pulmonary hypertension for which right heart catheterization remains the diagnostic gold standard. Right heart catheterization-derived parameters help classify pulmonary hypertension into several subgroups, assess risk of adverse events or mortality and make therapeutic decisions. According to the European Society of Cardiology guidelines pulmonary hypertension (PH) is defined as an increase in mean pulmonary artery pressure (PAPm) > 25 mmHg, whereas a distinction between pre- and post-capillary PH is made based on levels of pulmonary artery wedge pressure (PAWP). Moreover, right atrial pressure (RAP), cardiac index (CI) and mixed venous oxygen saturation (SvO_2) are the only parameters recommended to assess prognosis and only in patients with pulmonary arterial hypertension (PAH). Patients with RAP > 14 mmHg, CI < 2.0 l/min/m^2 and SvO_2 < 60% are at high (> 10%) risk of death within the next year.

The purpose of this paper is to show that RHC-derived parameters can be used on a considerably larger scale than currently recommended. Several prognostic parameters, with specific thresholds have been identified for each subtype of pulmonary hypertension and can be helpful in everyday practice for treatment of PH.

KEY WORDS: pulmonary hypertension, right heart catheterization, pulmonary artery hypertension

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INTRODUCTION

Right heart catheterization (RHC) is a medical procedure that has been used for decades to evaluate heart function and to diagnose a wide spectrum of cardiovascular diseases. The history of catheterization began in 1711 when Stephen Hale performed the first catheterization of equine vessels using brass pipes to measure blood pressure and cardiac output. In 1844, physiologist Claude Bernard was the first to catheterize right and left ventricle in a horse using glass tubes. The 20th century witnessed rapid developments in cardiac catheterization beginning with self-cannulation using a urethral catheter by dr. Forssmann in 1929, through advances in the procedure made by dr. André Cournand and dr. Dickinson Richards in 1940, followed by the Nobel Prize in 1956 for dr. Cournand, dr. Richards and dr. Werner, and finally the development of a 'Swan–Ganz' catheter in 1970 by Jeremy Swan and William Ganz, from Cedars-Sinai Medical Center [1].

For decades RHC has been used in many clinical situations, to monitor patients in the Intensive Care Units and assess cardiac output, to perform pulmonary angiography, to detect and evaluate shunts between the systemic and pulmonary circulation, to diagnose pulmonary hypertension and examine patients before cardiac transplantation, to assess the heart function in valvular or congenital diseases, to distinguish between restrictive cardiomyopathy and constrictive pericarditis or to provide an access for endomyocardial biopsy heart defect closure (since 1976) or balloon pulmonary angioplasty [2]. RHC is part of a mandatory and comprehensive evaluation of PH patients. The RHC findings are used not only to confirm the diagnosis but also to predict survival or mortality in patients with specific PH subtypes.

THE AIM

The aim of this paper is to establish the cut-off values of several pulmonary hemodynamic parameters which can be useful in everyday practice. According to the current guidelines only some RHC parameters (RAP, CI and SvO_2) are relevant for estimating prognosis and only in PAH patients. PAH is a relatively rare disease, according to the Polish Registry of PH the average prevalence is 30.8 per million adults [3]. This entails difficulties in selecting a sufficiently large sample size to perform a reliable statistical analysis, nevertheless numerous studies have demonstrated the prognostic value of various RHC-derived parameters, not only in PAH, but also in other PH subgroups. Patients with

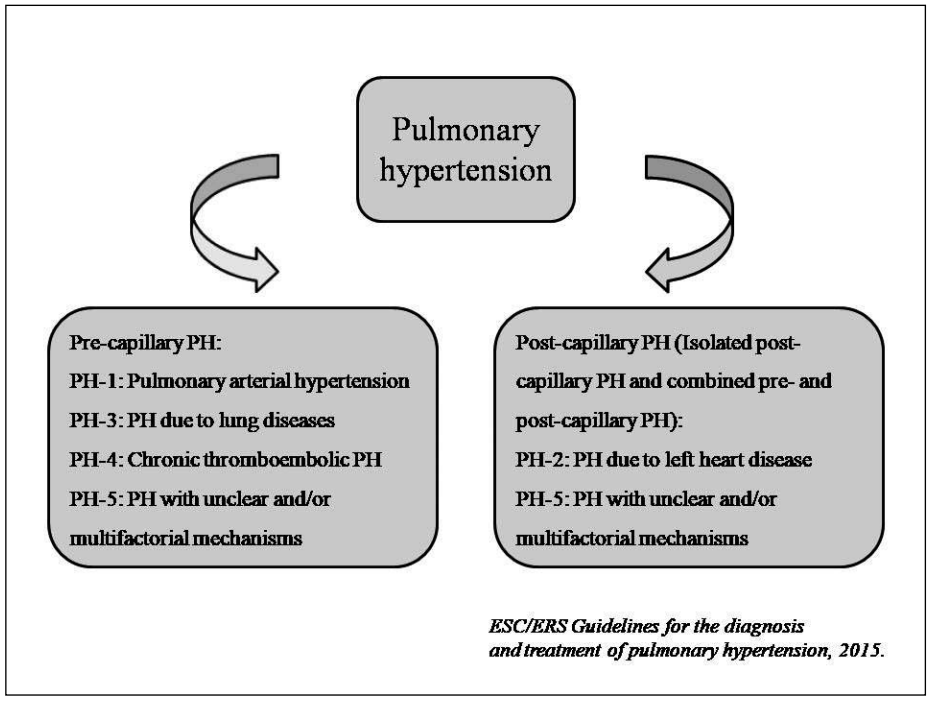

Fig. 1. Haemodynamic classification of pulmonary hypertension.

PH are a heterogeneous group of individuals, and although the current guidelines are quite narrowly applicable to RHC for prognosis, in our opinion the data presented below will show that RHC is essential for therapeutic decision-making and provides important prognostic information for each patient with PH.

REVIEW AND DISCUSSION

RHC remains the gold standard in the diagnosis of pulmonary hypertension (PH), which is defined as an increase in mean pulmonary artery pressure (PAPm) ≥ 25 mmHg, and further classified into pre-capillary and post-capillary PH (with two subgroups: isolated post-capillary PH and combined post-capillary and pre-capillary PH) (Fig. 1, Table 1) [4].

RHC is usually performed through puncture of the subclavian or jugular vein under local anaesthetic. Optimally, a balloon-tipped catheter is inserted under fluoroscopic guidance through a central vein, right atrium and right ventricle to the pulmonary artery.

It is recommended to measure several hemodynamic parameters during RHC i.e. right atrial pressure (RAP), right ventricular pressure (RVP), pulmonary artery pressure (PAP), pulmonary artery wedge pressure (PAWP) and cardiac output (CO). The measurements are then used to calculate cardiac index (CI), diastolic pressure gradient (DPG), transpulmonary pressure gradient (TPG) or pulmonary vascular resistance (PVR) (Table 2). In addition, blood is drawn from intracardiac chambers, great vessels and pulmonary artery to detect and quantify shunts between the systemic and pulmonary circulation, to measure mixed venous oxygen saturation (SvO_2) and to calculate cardiac output using the Fick method [5].

During RHC, when the diagnosis of pulmonary hypertension is confirmed, pulmonary vasoreactivity (vasodilator) testing should be performed for Group 1 PH and Group 2 PH, to identify patients with a reversible form of PH. Current guidelines recognize the use of four medications for assessment of pulmonary vasoreactivity, i.e. inhaled nitric oxide, inhaled iloprost or epoprostenol and intravenous adenosine (Table 3). In patients with pulmonary arterial hypertension (PAH, PH-1), a positive acute response (responders) is defined as a reduction of the PAPm ≥10 mmHg to reach an absolute value of PAPm ≤ 40 mmHg with an increased or unchanged CO [4]. A decrease of TPG below 15 mmHg, during vasoreactivity testing with sodium nitroprusside or nitric oxide, is mandatory for PH-2 patients with severe heart failure to be eligible for a heart transplant [6].

The current cut-off value of PAPm for a diagnosis of PH was established in 1973, but available data suggests that normal resting PAPm is 14.0 ± 3.3 mmHg, therefore, new definitions of PH and PH in left heart disease (LHD) were proposed during the 6th World Symposium on Pulmonary Hypertension in Nice in 2018 (Table 4) [7]

Regarding the new PH definition evidence supporting its usefulness has been available in the literature for years. Elevated PAPm ≥ 20 mmHg is associated with a significantly worse prognosis in patients with connective tissue disease (log-rank test p = 0.005) [8] and idiopathic pulmonary fibrosis (IPF) (log-rank test p = 0.001), compared with PAPm < 20 mmHg [9]. Patients with PAPm = 19–24 mmHg show a 23% (95% CI [12–36%], P < 0.0001) and a 7% (95% CI [1–12%], P = 0.0149) increase of the adjusted hazard for mortality and hospitalization, respectively, compared to patients with PAPm ≤ 18 mmHg [10].

PULMONARY ARTERIAL HYPERTENSION (PH-1)

According to the current PH guidelines the RHC-derived measurements are one of the variables used to estimate 1-year mortality in patients with PAH. RAP, CI and SvO_2

$$PCa = \frac{SV}{PP}$$

PCa (PAC) = pulmonary vascular compliance (pulmonary arterial capacitance)
SV = stroke volume
PP = pulse pressure

Fig. 2. Formula of the pulmonary vascular compliance.

$$Rup = \frac{100 \times (mPpa - Poccl)}{(mPpa - Ppao)}$$

Ppao = pulmonary artery occluded pressure,
mPpa = mean pulmonary artery pressure,
Poccl = occlusion pressure

Fig. 3. Formula of the upstream resistance.

are used to classify patients into one of the three groups according to mortality risk: low risk < 5% (RAP < 8 mmHg; CI ≥ 2.5 l/min/m^2; SvO_2 > 65%), intermediate risk 5 – 10% (RAP 8 –14 mmHg; CI 2.0 – 2.4 l/min/m^2; SvO_2 60 – 65%) and high risk > 10% (RAP > 14 mmHg; CI < 2.0 l/min/m^2; SvO_2 < 60%) [4]. The French PAH registry shows that the initially measured hemodynamic parameters have no prognostic value, but RAP and SVI measured in patients receiving treatment (PDE-5 I, ERB, prostanoids) are predictive of poor prognosis. The risk of death or lung transplantation increases by 5% per each 1 mmHg of RAP increase (HR = 1.05; 95% confidence interval: 1.02 – 1.09; p <0.001) and by 28% per each 10 mml/m^2 of SVI decrease (HR = 1.28; 95% confidence interval: 1.11 – 1.49; p <0.001). The optimal cut-off value is 9 mm Hg for RAP (AUC = 0.62; 95% confidence interval: 0.57 – 0.67; p<0.01) and 38 ml/m^2 for SVI (AUC = 0.68; 95% confidence interval: 0.64–0.72; p< 0.01). It is noteworthy that this negative impact of low SVI is seen even among those considered to be at lower risk (e.g. low NYHA class or CI ≥2.5 l/min/m^2) [11]. In 2007, a study carried out in a small group of patients (64 vs. 981 patients) demonstrated that SVI ≤ 25 ml/m^2 was a negative prognostic factor (log-rank test, p=0.010) [12]. Another parameter which is significantly associated with poor outcomes is increasing DPG (Hazard ratio [HR] 1.29 per 10 mmHg increase) [13].

In the REVEAL registry PVR was included as one of the risk factors in patients with PAH. PVR < 5 Wood units (WU) was associated with better survival outcomes. Previously diagnosed patients with a PVR < 5 WU had a 5-year survival of 73.9% ± 2.3%, compared with 66.0% ± 1.8% in patients with a PVR of 5 to 10 WU. Newly diagnosed patients with a PVR < 5 WU had a 5-year survival of 70.1% ± 4.9%, compared with 57.3% ± 3.2% in patients with a PVR of 5 to 10 WU [14]. The REVEAL Registry Risk Score Calculator has been developed to predict 1-year survival in patients newly diagnosed with PAH. The risk calculator is based on 15 variables, including echocardiographic findings, blood test results, clinical characteristics and classification, as well as such RHC-derived parameters as RAPm > 20 mmHg and PVR > 32 WU [15].

In patients with systemic sclerosis-related pulmonary arterial hypertension (SSc-PAH) several hemodynamic measurements are also predictors of increased mortality. A study by Mukherjee et al. demonstrated that raised RAPm, raised PAPm and low CI were related to survival. The Cox multivariate regression analysis revealed that RAPm was the strongest independent factor for a poor outcome, with hazard ratio 20.7 and a p value of 0.0001. As there was a relative correlation between RAPm, PAPm and CI, the latter two values could not be considered as independent predictors [16]. In addition, data from the REVEAL study showed that RAPm > 20 mm Hg and PVR > 32 WU were predictors of mortality in the SSc-PAH group [17]. Campo et al. proved that PVR > 7.2 WU (HR = 3.13, 95% confidence interval: 1.50 – 6.52; p < 0.01), SVI < 30 ml (HR = 2.34, 95% confidence interval: 1.11 – 4.96; p = 0.03) and PCa < 1.25 ml/mm Hg (HR = 3.06, 95% confidence interval: 1.41 – 6.65; p < 0.01) were strong predictors of mortality [18].

Patient selection for liver transplantation due to portopulmonary hypertension (PP-PH) requires careful hemodynamic assessment. Studies in patients with PP-PH at Mayo Clinic provided the following results: PAPm > 50 mmHg was associated with 100% cardiopulmonary mortality, whereas values of PAPm 35-50 mmHg and PVR > 250 dynes·s·cm^2 were linked to the mortality rate around 50%. No cardiopulmonary mortality was reported in patients with PAPm <35 mmHg or TPG <15 mmHg [19].

Investigators are still searching for new RHC-derived parameters. One of them is pulmonary vascular compliance (PCa), defined as SV divided by pulse pressure – the difference between systolic pulmonary artery pressure and diastolic pulmonary artery pressure (PAPs – PAPd), which describes elastic properties of the pulmonary arterial system (Fig. 2). In adult patients with PAH-related congenital heart disease, the Kaplan-Meier survival curves show that PCa < 1.04 ml/mmHg is a risk factor for mortality (Log rank: P < 0.001). ROC curve analysis for PCa = 1.04 ml/mmHg shows 87% sensitivity and 64% specificity (AUC = 0.746, 95% confidence interval: 0.657 – 0.836, P < 0.001) [20]. Also in patients with idiopathic PAH (IPAH), PCa has been recognized as an indicator of mortality in univariate analysis (HR=17.0 per ml/mmHg decrease; 95% confidence interval: 13.0 – 22.0; p < 0.0001). Patients with PCa = 0.40-0.81 ml/mm Hg have a 61% 4-year mortality [21].

Surgical correction of congenital heart disease causing Eisenmenger syndrome is considered useful in patients with PVRi < 4 WU.m^2 and PVR < 2.3 WU. Surgery is not the

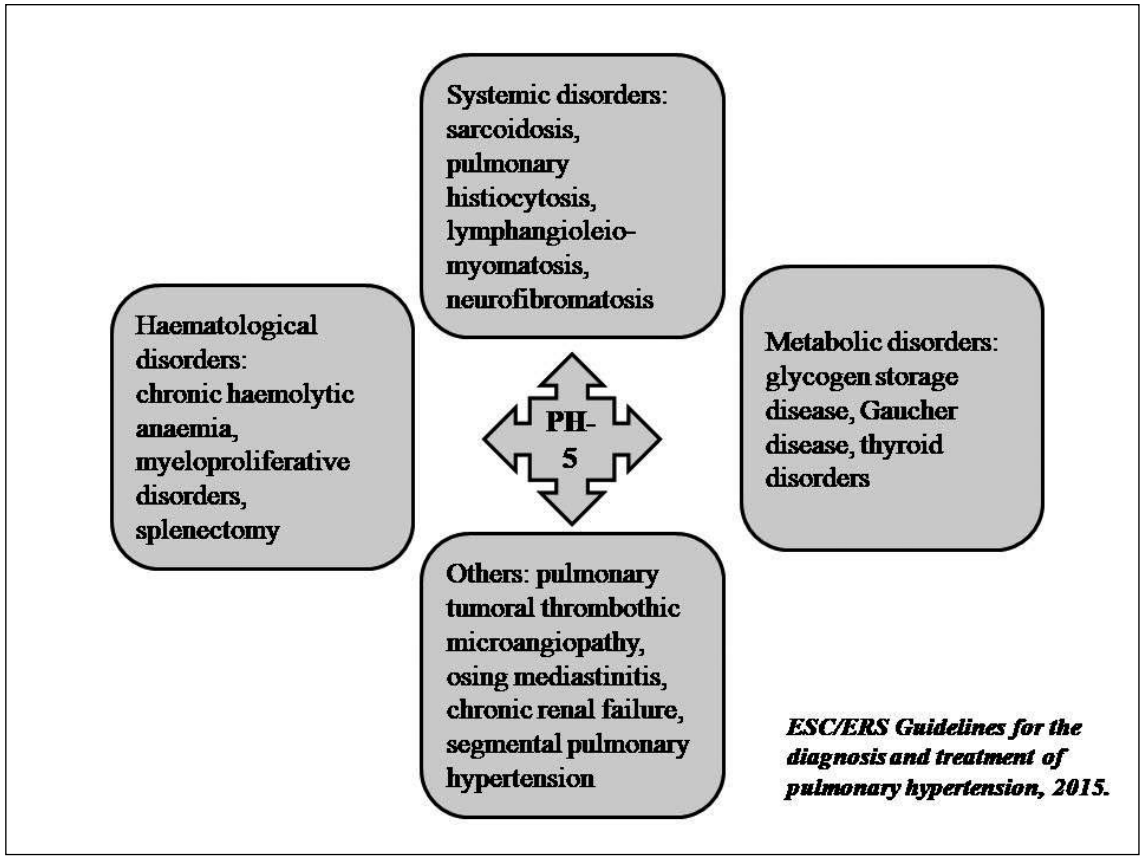

Fig. 4. Clinical classification of the group 5 pulmonary hypertension.

best option in subjects with PVRi > 8 WU.m^2 and PVR > 4.6 WU. Patients with an intermediate PVRi of 4-8 WU.m^2 and PVR of 2.3-4.6 WU require individualized approach [4]. In univariate analysis, SVi (HR = 1.058; 95% confidence interval: 1.02 – 1.10; p = 0.002), PVRi (HR = 1.024; 95% confidence interval: 1.00-1.05; p = 0.01) and ΔPVRi after vasoreactivity testing with intravenous epoprostenol (HR = 0.972; 95% confidence interval: 0.95-0.99; p = 0.02) were found to be significant predictors of poor outcomes in patients with PAH associated with congenital heart disease and Eisenmenger syndrome and receiving bosentan therapy. ΔPVRi was demonstrated as the only independent predictor of clinical worsening (HR = 0.973; 95% confidence interval: 0.95 – 0.99; p = 0.01), and ΔPVRi < 25%, with 56% sensitivity and 100%, specificity could predict clinical worsening. The area under the ROC curve was 0.773 (95% confidence interval: 0.608 – 0.892) [22].

PULMONARY HYPERTENSION DUE TO LEFT HEART DISEASE (PH-2)

Prognostic factors have also been identified in pulmonary hypertension due to left heart disease (PH-LHD), including subsets of patients with an LVEF ≥ 50% (PH-HFpEF) and LVEF < 50% (PH-HFrEF). Despite significantly higher DPG values in the PH-HFpEF compared to the PH-HFrEF group, the variable was not a significant predictor of survival. On the other hand, both groups had similar PCa values, but PCa < 1.1 ml/mmHg in PH-HFpEF [23] and PCa < 2.15 ml/mmHg in PH-HFrEF [24] were established as significant predictors of survival.

The interpretation of DPG values appears more complex. An elevated DPG ≥ 7 mmHg is associated with increased mortality in patients with PH due to LHD, but the correlation seems to be weak, and other factors e.g. levels of *N*-terminal pro-brain natriuretic peptide (NT-proBNP) or NYHA class have better prognostic values [25]. On the other hand, available evidence suggests that DPG ≥ 7 mmHg does not have a significant effect on survival in PH-LHD, but elevated PVR > 3WU and TPG > 9 mmHg can be a predictor of death [26].

RHC is mandatory at all stages of the qualification process of heart failure patients for a heart transplant (HT). It should be performed not only prior to an adult patient being listed for cardiac transplantation but also repeated periodically until transplantation. The 2016 guidelines for HT recommend to adjust the assessment process to specific clinical circumstances [6]. The risk of death due to right heart failure after heart transplantation is significantly increased in some clinical situations: if PVR > 5 WU or PVRI > 6 WU.m^2 (children), or TPG exceeding 16-20 mmHg; if PAPs > 60 mmHg in combination with one of the former values or if PVR cannot be reduced to < 2.5 after a vasodilator challenge without a drop in SBP < 85 mmHg. Vasodilator testing should be performed in subjects with PAPs > 50 mmHg and either TPG > 15 mmHg

or pulmonary PVR > 3WU (with SBP > 85 mmHg). The diagnosis of irreversible pulmonary hypertension should be suspected in patients with unsuccessful acute vasodilator testing and medical treatment failure [27].

A left ventricular assist device (LVAD) can significantly reduce PVR in heart transplant candidates. Regardless of pre-implantation PVR: low (< 5 WU) or high (≥ 5 WU), a 3-year survival after transplantation is similar between groups (85.0% and 79.0%, respectively), however posttransplant in-hospital mortality remains significantly increased among patients with the initially high PVR (P<0.05) [28]. Even PVR ≥3 WU vs. PVR < 3 WU places patients with HF and LVAD at higher risk of death (HR 1.55; P = 0.026), whereas elevated DPG is only associated with the development of RV failure (HR: 3.30; P = 0.004 for DPG ≥ 7 versus DPG < 7) [29].

There are other parameters, not included in the current HT guidelines, which can help evaluate patients before and after HT. One of them is relative pulmonary hypertension calculated from mean artery pressure (MAP) and PAPm (MAP/PAPm). Preoperative value < 3 in transplant candidates > 60 years of age is associated with lower survival rates after transplantation (HR 5.39; 95% confidence interval: 1.64 – 17.74; p=0.006) [30]. Postoperative PAPm > 20 mmHg, compared to PAPm ≤ 20 mmHg, is linked to a significantly increased mortality rate within one year after transplantation (11.5 ± 0.7 vs. 15.6 ± 0.6 years, p <0.001) [31].

Aortic stenosis is one of the most common valve diseases and its prevalence is expected to rise with an aging population. In some clinical situations RHC can help with patient evaluation and qualification for surgical treatment or transcatheter aortic valve implantation (TAVI). There is a significant association between PH and reduced survival after surgical aortic valve replacement (p=0.006) or TAVI (1-year mortality unadjusted: HR: 2.03; 95% confidence interval: 1.07 – 3.85; p = 0.030; after adjustment: HR: 1.95; 95% confidence interval: 1.01 – 3.76; p = 0.046) [32]. Of all PH subgroups, Cpc-PH remains the strongest predictor of death both for surgical (HR 4.39, 95% confidence interval: 2.40 – 8.03; p < 0.001) [33] and TAVI patients (adjusted HR: 3.28; 95% confidence interval: 1.43 – 7.53; p = 0.005 at 1 year) [32]. In addition, among patients with severe aortic stenosis (AVA < 1 cm^2) lack of postoperative reduction in PAPm ≥ 10 mmHg is an independent predictor of mortality (HR: 0.93; 95% confidence interval: 1.2 – 12.5; p = 0.048), whereas preoperatively elevated PAWP is a significant predictor of reduced PAPm (OR, 1.26; 95% confidence interval: 1.13 – 1.41; p < 0.0001) [34]. In aortic regurgitation, PH has been less studied.

The relationship between prognosis in mitral regurgitation and parameters related to pulmonary hypertension has been described in the literature, however, these are forecasts based mainly on PAPs values from echocardiographic measurements, which is beyond the scope of this study.

Rheumatic heart disease, especially mitral stenosis (MS) remains a major health problem in developing countries, and percutaneous balloon mitral valvotomy (PBMV) is a safe and effective procedure in symptomatic patients. Baseline PVR> 1.81 WU has been shown to be an independent predictor of persistent elevation of RVSP > 50 mmHg immediately after PBMV with 69% sensitivity and 86% specificity (95% confidence interval: 64–95; p = 0.002; AUC = 0.79) [35]. Furthermore, post-PBMV PAPm has been established as an independent predictor of all-cause mortality (per mmHg – HR: 1.045; 95% confidence interval: 1.015 – 1.077; p = 0.003) and mitral valve reintervention (per mmHg – HR: 1.055; 95% confidence interval: 1.024 – 1.087; p < 0.001) [36].

PULMONARY HYPERTENSION DUE TO LUNG DISEASES AND/OR HYPOXIA (PH-3)

Although RHC is not recommended in conventional evaluation of PH-3 patients, it may offer clues that help narrow the differential diagnosis and make therapeutic decisions regarding lung transplantation. PH was found to have a negative effect on survival after lung transplantation (LT) within 90 days of follow-up, compared to non-PH patients (p = 0.043 and p = 0.003, respectively), but at one year after LT only pre-capillary PH versus post-capillary PH remained a negative prognostic factor (p = 0.037 and p = 0.447, respectively) [37]. In patients with end-stage lung disease awaiting LT, PAPm ≥ 30 mmHg, PAPd ≥ 20 mmHg and PAPs ≥ 44 mmHg are associated with worse prognosis with sensitivity = 70%, 70%, 73%, specificity = 76%, 69%, 72% and AUC = 0.67, 0.68, 0.72, respectively [38]. Despite the correlation between mortality in patients awaiting LT and exacerbation of PH, PAH-approved drugs are not recommended due to inhibition of hypoxic pulmonary vasoconstriction [4].

CHRONIC THROMBOEMBOLIC PULMONARY HYPERTENSION (PH-4)

Not only direct RHC-derived measurements can be used to predict outcome in PH patients. Evidence shows that analysis of pulmonary artery occlusion pressure waveform might identify CTEPH patients with persistent PH after pulmonary thromboendarterectomy (PTE), which contributes to poor outcome. Pulmonary vascular resistance can be divided into large arterial (upstream, Rup) and small arterial plus venous (downstream) components (Fig 3). Lower Rup can be seen in patients with CTEPH with small vessel disease and embolic material so that it makes it impossible to perform an effective PTE. Patients with Rup <60% seem to be at the highest risk of persistent PH after PTE [39]. Different cut-off points may influence the sensitivity and specificity of the Rup in distinguishing operable from inoperable CTEPH. The cut-off point of 79.3% gives a 100% sensitivity and 57.1% specificity, whereas the cut-off value of 83.8% decreases sensitivity to 83.3% but specificity is increased to 71.4% [40].

The level of preoperative PVR, with a threshold > 800 dyn.sec.cm-5, was found to be a risk factor for increased mortality in patients after PTE, with sensitivity and specificity of 77% and 60%, respectively [41]. PAPm ≥ 38mmHg

and PVR ≥ 425 dyn.sec.cm-5 were predictors of poor outcome at 3-6 months post PTE [42].

Recent years have seen major advances in the CTEPH treatment. Balloon pulmonary angioplasty (BPA) has become a very useful alternative and complementary therapy to PTE, especially in inoperable patients with distal lesions. It is very important because at the moment of CTEPH diagnosis, almost 40% patients are considered inoperable [43]. In the biggest Japan BPA registry after a series of BPA procedures PAPm decreased from 43.2 ± 11.0 to 22.5 ± 5.4 mmHg [44], whereas in Polish patients with residual PH after PTE, a series of BAP reduced PAPm from 44.7 ± 6.4 to 30.8 ± 7.5 mm Hg (31% decline; $p < 0.001$) [45]. Substantial evidence shows that several RHC-derived measurements can be helpful in patient assessment before and after BPA. In multivariate analysis, PAPd at baseline was found to be an independent predictor of residual PH (PAPm > 30mmHg at follow-up) after BPA (OR: 2.04; 95% confidence interval: 1.06 – 5.76; $p = 0.029$) [46] and a relative increase in SvO2 > 125.4% over the baseline value significantly correlated with increased eGFR one year after BPA, with 100% specificity and 24.1% sensitivity [47].

It is essential to bear in mind that there are several potential risks related to the BPA procedure, with reperfusion pulmonary injury (RPI) being most important. Post-BPA pulmonary arterial pressure distal (Pd) to the site of stenosis is associated with RPI occurrence (OR: 1.139, 95% confidence interval: 1.053 – 1.231, $p = 0.001$) and post-BPA Pd > 19.5 mmHg can predict RPI with 79.6% sensitivity and 75.4% specificity, whereas the area under ROC curve is 0.814 [48].

PULMONARY HYPERTENSION WITH UNCLEAR AND/OR MULTIFACTORIAL MECHANISMS (PH-5)

PH-5 is a heterogeneous group of diseases with unclear or multifactorial mechanisms, requiring an individual approach to patients, for whom no universal treatment is available (Fig. 4) [4]. This category includes PH associated with sickle cell disease (SCD), where average life expectancy is 25.6 months, whereas a 119-month survival of 70% has been reported in patients with SCD without PH. RHC-derived measurements that affect outcome include PAPm, where each increase of 10 mmHg is associated with an approximately 2-fold increase in the rate of death [49], and TPG ≥12mmHg is deemed to be an independent predictor of increased mortality [50].

CONCLUSIONS

Despite the availability of a wide range of non-invasive diagnostic tests RHC remains the gold standard for assessing pulmonary artery pressure. RHC-derived parameters are useful not only for PH diagnosis but also for prognostic purposes in numerous PH subtypes, facilitating the decision-making process. Although they are not included in the current ESC guidelines, they may be extremely useful in everyday practice for evaluation of a heterogeneous group of patients with PH. Comprehensive PH patient assessment is the cornerstone of individualized care with a significantly better outcome.

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Conflict of interest

The Authors declare no conflict of interest.

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