

# Precapillary pulmonary hypertension causes increased biventricular hemodynamic forces

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Jones D, Johnson E. Precapillary pulmonary hypertension causes increased biventricular hemodynamic forces. Clin Cardiol J 2023;7(1):1-2.

## ABSTRACT

Elevated pulmonary vascular pressure and resistance characterise precapillary Pulmonary Hypertension (PHprecap). Since the prognosis of patients is poor, it is essential to comprehend the underlying pathophysiological mechanisms in order to direct and enhance treatment. Ventricular Hemo Dynamic Forces (HDF) is a possible early indicator of heart dysfunction that may help with therapy effectiveness assessment. Therefore, our goal was to find out whether HDF varied between patients with PHprecap and healthy controls. Patients who had PHprecap received cardiac magnetic resonance imaging with 4D flow, as well as age and sex matched healthy controls. Using the Navier-Stokes equations, biventricular

HDF were calculated in three spatial directions over the course of the cardiac cycle. In all three directions, biventricular HDF (N) indexed to stroke volume (l) were bigger in patients than controls. Data are shown as median N/l for patients and controls, respectively. In precapillary pulmonary hypertension, hemodynamic force analysis provides information on pathological heart pumping processes in addition to more well established volumetric and functional data. Left ventricular hemodynamic abnormalities are mostly caused by under filling rather than intrinsic ventricular dysfunction, and the right ventricle partially makes up for the increased afterload by increasing transverse forces.

**Keywords:** Hemodynamic; Magnetic resonance; Tricuspid valve; Stroke volume; pulmonary hypertension

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## INTRODUCTION

The term "precapillary Pulmonary Hypertension" (PHprecap) refers to a group of disorders with negative outcomes. Without underlying pulmonary or left ventricular cardiac disease, Pulmonary Arterial Hypertension (PAH) and Chronic Thromboembolic Pulmonary Hypertension (CTEPH) are subgroups of PHprecap that share the characteristics of high pulmonary arterial pressure and vascular resistance. The increased pulmonary vascular resistance causes an increase in pressure, affects cardiac shape, and impairs the left side of the heart's ability to fill properly, all of which contribute to early death. With maintained ventricular sizes, changes to the pumping processes may take place, and present techniques for assessing treatment effect and deterioration are inaccurate. The possibility to employ Hemodynamic Forces (HDF) estimated from Cardiac Magnetic Resonance imaging (CMR) as a marker of cardiac dysfunction may help doctors better assess the effectiveness of treatment in people with PHprecap. Ventricular contraction and relaxation produce pressure gradients within the ventricle, which leads to hemodynamic forces that cause the blood to accelerate. A reference standard CMR approach with 4D flow data, where blood flow is monitored in all three spatial directions and across time, can be used to calculate intracardiac HDF [1].

As force equals mass times acceleration, variations in the right (RV) and left (LV) ventricle sizes in patients may have an effect on HDF. As HDF is a measurement of the three dimensional acceleration of blood, ventricular pumping and filling mechanisms may affect HDF even with preserved end diastolic volume and ejection percent. The impulses that cause blood to flow, which may vary between ventricles with the same volumes, can thus be quantified by measuring HDF over time. Patients with PHprecap frequently have altered right ventricular morphological shape, altered LV regional contribution to Stroke Volume (SV), and decreased LV volumes, although their LV ejection fraction is usually unaffected [2]. Patients with ventricles that are volume overloaded have changed HDF, according to earlier studies. Therefore, in this patient population, ventricular HDF may have the

potential to be a more sensitive marker of abnormal cardiac pumping processes than standard volumetric and functional metrics, which may enhance assessment of treatment outcome. The purpose of this study was to determine whether individuals with precapillary pulmonary hypertension have different hemodynamic force patterns from healthy controls.

## LITERATURE REVIEW

In this observational study, healthy individuals served as the reference group and patients being evaluated for possible precapillary Pulmonary Hypertension (PHprecap) at our tertiary PH facility, where CMR was performed as part of the standard examination. Between 2016 and 2021, recruitment took place, and data was collected. The patient inclusion criteria were a verified diagnosis of PHprecap caused by Chronic Thromboembolic Pulmonary Hypertension (CTPH) or Pulmonary Arterial Hypertension (PAH) (CTEPH). After having previously taken part in a baseline test, ten healthy subjects from the ongoing population-based study SCAPIS (Swedish Cardio Pulmonary bio Image Study) were added as a control group and received an extra examination with CMR and 4D flow. From earlier trials, our team added two healthy controls. With the aid of a group level comparison for age and sex, the controls were matched to the patient cohort. Spirometry, carotid artery ultrasonography, coronary computed tomography, clinical labs, and the subject's medical history were all included in the SCAPIS baseline examination. Any previously diagnosed systemic or cardiovascular disease, any pathology found during imaging examinations, use of cardiovascular medications, smoking, systemic blood pressure greater than 140/90 mmHg, and any pathology found during electrocardiography or CMR were all exclusion criteria for the healthy subjects [3,4].

## DISCUSSION

In this investigation, precapillary pulmonary hypertension patients and healthy controls were both subjected to biventricular hemodynamic forces. Patients displayed greater biventricular hemodynamic forces that were indexed to SV in all three spatial directions, demonstrating biventricular disease in the mechanisms of both pumping and filling that cannot be

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**Received:** 22-Nov-2022, Manuscript No. PULCJ-22-5710; **Editor assigned:** 24-Nov-2022, PreQC No. PULCJ-22-5710 (PQ); **Reviewed:** 08-Dec-2023, QC No. PULCJ-22-5710; **Revised:** 16-Jan-2023, Manuscript No. PULCJ-22-5710 (R); **Published:** 24-Jan-2023, DOI: 10.37532/2753-1201.23.7(1).1-2.

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adequately explained by variations in blood volume. Biventricular hemodynamic force analysis has the potential to improve treatment effect guidance in this patient cohort and may be a more sensitive indicator of pathological cardiac pumping mechanisms than typical volumetric and functional parameters in patients with PHprecap [5].

## Cardiac pumping physiology

**Pulmonary arterial hypertension and chronic thromboembolic pulmonary hypertension:** While persistent thromboembolic pulmonary hypertension is predominantly linked to pulmonary artery blockages, pulmonary arterial hypertension is thought to be principally induced by vascular remodelling of the pulmonary arteries. The pathophysiological effects on the heart pumping mechanisms are comparable despite the fact that the etiologies of the two groups are different. In our patient sample, septal flattening typical of PHprecap patients was seen, which may have an impact on the hemodynamic pressures in the RV and LV.

**RV systolic HDF:** In comparison to controls, patients' RV systolic HDF indexed to SV was greater in the two transverse directions, showing that more force per ml of blood volume is needed to attain RV SV in these directions. Reduced RV Atrioventricular Plane Displacement (AVPD) and RV longitudinal strain in patients may suggest that an increased RV contractile drive is mostly obtained in the transverse directions in order to resist the higher pulmonary arterial pressure and preserve RV SV. The myocardial fibers are arranged in counter wound helices in a healthy ventricle, allowing for a twisting motion during systole. The myocardial structure may change as a result of ventricular hypertrophy or dilatation, altering ventricular motion and consequently the intraventricular blood flow. Between patients and controls, there was no difference in systolic HDF indexed to SV in the apex base direction. The main cause of SV in the hearts of healthy persons is AVPD. Despite decreased tricuspid valve motion, patients with PHprecap and RV dilatation as a result of volume overload often have retained longitudinal contribution to SV through increased short axis area. Patients with PHprecap have preserved RV SV and cardiac output in the compensated stage, and our data with preserved RV SV in patients could explain why patients and controls had similar systolic force patterns in the RV apex base direction despite patients having decreased RV EF.

**RV diastolic HDF:** In healthy hearts, the AV plane is mostly moved towards the atrium to prolong the ventricle and change where the tricuspid valve sits in respect to the blood, which fills the RV. It has been demonstrated that patients with PHprecap have lower filling rates, AVPD, and RV isovolumetric relaxation times than controls, with a greater contribution from atrial contraction. Increased End Diastolic Volume (EDV) and End Systolic Volume (ESV) in our patient cohort demonstrate pathological dilatation with an excessively stretched RV myocardial. An increased ventricular stiffness and decreased ventricular compliance, where a greater volume of intraventricular blood is accelerated compared to controls, could be indicated by increased diastolic forces and RV EDV but with intact RV SV.

**LV systolic HDF:** In the LV apex base and lateral wall septum axis, individuals with PHprecap had higher systolic HDF indexed to SV than controls. As the lateral wall septum direction is parallel to the LV outflow tract, these two directions together make up the primary direction of systolic blood flow in a healthy heart. A changed pumping mechanism to accomplish SV is indicated by our patient cohort's increased systolic longitudinal HDF and decreased AVPD. Despite having intact LV EF, patients with pulmonary hypertension have previously been observed to have impaired longitudinal LV function.

**LV diastolic HDF:** The LV volumes in PHprecap are less than in healthy controls because of increased pulmonary circulation resistance, which causes a reduction in the LV filling. In our study, patients decreased LV AVPD and LV EDV is clear signs of under filling. According to the information above, HDF is a measurement that takes into account the physiological mechanisms of the myocardium and how they affect

intraventricular blood flow patterns, rather than just serving as a substitute for cardiac volumes. Recent research employing echocardiography revealed decreased LV diastolic intraventricular pressure gradients in PHprecap, and this parameter was proposed as a sign of poor LV suction in this patient population [6].

**Indexing to SV:** Comparing the functional properties of hearts of various sizes is made easier by indexing HDF to SV. Given that force is inversely proportional to mass and acceleration, indexing HDF to SV reduces the significance of volumetric discrepancies while highlighting the acceleration component. Systolic forces in a healthy heart should rise proportionally to SV because, according to Newton's second law of motion, force equals mass times acceleration. A bigger blood acceleration is indicated by an increased HDF/SV, which is most likely the result of sympathetic upregulation and higher blood pressure to preserve cardiac output. The Frank-Starling law states that during diastole, the volume of the stroke should equal the fullness of the ventricle in a healthy heart. As previously demonstrated, HDF analysis cannot distinguish between heart failure patients with maintained EF and controls, however patients with impaired ventricular volumes were found to have impaired volume-normalized HDF, ventricular volumes play a significant role in determining HDF. When we indexed HDF to SV, we also noticed variations between patients with PHprecap and controls. In the LV, but not in the RV, there were differences in stroke volume between patients and controls. Differences between the groups were identified in one direction in the RV and none of the directions in the LV when quantifying RMS HDF in absolute values.

Although assessed with a cine feature tracking model, which has been found to be less sensitive than our method based on 4D flow, it has been shown that patients with heart failure and retained LV EF have lower LV hemodynamic longitudinal forces per blood volume compared to controls. However, our group of patients exhibits higher forces per blood volume in this direction than controls despite having intact LV EF. The LV SV, EDV, and ESV are typically smaller in PHprecap patients. Even if the LV function is compromised, sympathetic system activation to maintain pressure can help preserve EF and may lead to enhanced blood clotting. The possibility exists for hemodynamic force analysis to be a more sensitive indicator of heart degeneration than conventional volumetric.

## CONCLUSION

In precapillary pulmonary hypertension, hemodynamic force analysis provides information on pathological heart pumping processes in addition to more well established volumetric and functional data. Left ventricular hemodynamic anomalies are mostly caused by under filling rather than intrinsic ventricular dysfunction, and the right ventricle partially makes up for the higher afterload by increasing transverse forces.

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