

Original Research Article

CARDIAC WALL MOTION ABNORMALITIES IN COR PULMONALE

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ABSTRACT

Background: Aim: The aim is to analyze patterns, relevance, and outcomes of cardiac wall motion abnormalities in patients with cor pulmonale, using clinical and echocardiographic evidence to identify diagnostic and prognostic implications.^[1,2,3] **Materials and Methods:** This section should summarize the type and number of patients studied (retrospective/prospective), modalities used for diagnosis (primarily echocardiography, Doppler studies), inclusion/exclusion criteria, and statistical methods used for analysis, such as prevalence, correlation with disease severity, and associated complications.^[4,5] **Result:** The results must detail findings like the incidence of right ventricular (RV) dilatation, wall motion abnormalities, RV hypertrophy, paradoxical interventricular septal motion, and their statistical associations with severity of underlying cardiopulmonary process.^[5,6,4] **Conclusion:** Cardiac wall motion abnormalities, especially RV dysfunction and paradoxical septal motion, are significant diagnostic and prognostic markers in cor pulmonale and require systematic echocardiographic surveillance in high-risk groups.^[3,1,4]

INTRODUCTION

Cor pulmonale represents a pathophysiological condition in which the structure and function of the right ventricle are altered because of pulmonary hypertension secondary to disorders of the lung, pulmonary vasculature, or chest wall. It remains a major contributor to morbidity and mortality in chronic obstructive pulmonary disease (COPD), pulmonary embolism, and various interstitial lung disorders. The defining feature of cor pulmonale lies in the adaptation of the right ventricle (RV) to sustained pressure overload, which manifests echocardiographically as wall motion abnormalities. According to Mandoli and D'Andrea (2020), comprehensive echocardiographic evaluation serves as an indispensable tool in detecting subtle right ventricular strain patterns even before overt dilation and failure occur.

Initially, adaptive mechanisms such as enhanced RV wall tension maintain stroke volume, but prolonged strain leads to myocardial ischemia and localized hypokinesis. Vieillard-Baron et al. (2002) demonstrated that acute cor pulmonale, particularly due to massive pulmonary embolism, can result in abrupt RV dilation with paradoxical septal motion, a hallmark of pressure overload. Chronic adaptations, as noted by Weitzenblum et al. (2002), involve diffuse wall thickening transitioning to global

hypokinesis. Cor pulmonale represents structurally and functionally altered right ventricular myocardium secondary to chronic or acute pulmonary arterial hypertension, most commonly due to chronic obstructive pulmonary disease (COPD) or pulmonary embolism. Chronic pressure overload causes progressive changes in right heart morphology, with wall motion abnormalities playing a central role in diagnosis and prognosis.^[2,7,8,1,3]

MATERIALS AND METHODS

Study Design: Prospective/retrospective observational study of patients diagnosed with cor pulmonale at a tertiary care hospital.

Inclusion Criteria: Patients aged >18 years, diagnosed with cor pulmonale by clinical and echocardiographic criteria.

Exclusion Criteria: Congenital heart disease, left-sided heart failure.

Assessments: Comprehensive 2D and Doppler echocardiographic studies including measurements of RV wall motion, dimensions, tricuspid regurgitation, and pulmonary pressures.^[4,5] Conventional echocardiography remains the cornerstone for identifying wall motion abnormalities linked to cor pulmonale. M-mode and two-dimensional imaging highlight RV dilation, interventricular septal flattening, and reductions in

fractional area change. According to Garrison et al. (2023), wall motion scoring indices adapted from left ventricular models are suboptimally sensitive for RV analysis, necessitating specific segmental evaluation—particularly of the infundibular and free wall segments. Gautam et al. (2018) observed a robust correlation between COPD severity and echocardiographic indicators such as tricuspid annular plane systolic excursion (TAPSE) and RV fractional shortening, both reflective of wall motion integrity.

Advanced Echocardiographic Techniques Recent advancements have expanded assessment to include tissue Doppler imaging (TDI) and strain echocardiography, which quantify subtle wall deformation patterns. Mandoli et al. (2020) emphasize that speckle-tracking echocardiography identifies early myocardial involvement, offering a sensitive index of contractile dysfunction before morphological changes appear. Vitarelli et al. (2016) similarly demonstrated global and regional reductions in RV longitudinal strain in patients with pulmonary hypertension. Cleski et al. (2024) elaborated that these advanced modalities enable differentiation between adaptive hypertrophy and pathological contractile failure, ultimately refining prognosis and therapeutic strategies.

RESULTS

Present findings as:

- Incidence rates: RV dilation (48-55%), RV hypertrophy (25-28%), paradoxical septal motion (7.5-18%), regional wall motion abnormalities (up to 36%), pulmonary hypertension (56-73%).^[5,6,4]
- Statistically significant correlation between wall motion abnormalities and cor pulmonale clinical outcomes ($p < 0.05$ for RV dilation, RV failure, septal abnormality, and mortality).^[4,5]

Statistical Analysis

- Use chi-square/Fisher's exact test to analyze categorical variables.
- Correlate the extent of RV wall motion abnormality with severity of cor pulmonale, and clinical outcomes.
- Present confidence intervals, odds ratios for severe complications, Kaplan-Meier survival analysis for prognostic validation.^[4]
- Discuss any limitations in statistical power or confounders.

Table 1: Demographics of COR Pulmonale

Characteristic	Study A	Study B	Study C
Mean Age (years)	55.6	54.9	56.0
Age Group (most common)	50–59 ^[3]	40–50 & >60 ^[6]	50–59 ^[6]
Male (%)	68 ^[3]	80.8 ^[6]	68 ^[1]
Female (%)	32 ^[3]	19.2 ^[6]	32 ^[1]
Major Etiology	COPD, Chronic Bronchitis, Emphysema, Smoking ^[6]	Smoking, COPD ^{[3][2]}	Lung diseases (COPD, CF in children) ^[1]

Table 2: Echocardiographic Findings

Finding	Prevalence (%)	Statistical Notes
RV Dilation	48 ^[5]	About half of moderate-severe cases
RV Hypokinesia	77 ^[10]	Highly sensitive in diagnosis
Paradoxical Septal Movement	18 ^[5]	Sign of systolic overload
Tricuspid Regurgitation	45 ^[13]	Common, increases with severity
Pulmonary Hypertension	24–56 ^{[5][9]}	Severity-dependent, up to 73% in severe COPD
LV Systolic Dysfunction	13 ^[5]	Present only in severe group

Table 3: Wall Motion Abnormalities by Disease Severity

Severity	% With Wall Motion Abnormalities	% With Cor Pulmonale	% With RV Failure	% With Pulmonary Hypertension
Mild	0 ^[12]	0 ^[12]	0 ^[5]	50 ^[5]
Moderate	11.1–27.8 ^{[5][12]}	22–27 ^[12]	11 ^[5]	27.8 ^[5]
Severe	33–68.2 ^{[5][12]}	76.7 ^[5]	23.3 ^[5]	73.3 ^[5]
Very severe	86.7 ^[12]	88.2 ^[13]	~23 ^[5]	66.7 ^[4]

Table 4: Correlation between wall motion abnormality and clinical outcomes

Echo Abnormality	Admission Rate	Mortality Rate	Other Outcome Associations
RV Dysfunction/Wall Motion Abnormality	High ^[11]	Higher ^[7]	Poor functional/exercise capacity ^[11]
Paradoxical Septal Movement	Increased	Elevated	Reduced survival, increased rehospitalisation ^{[8][11]}
Pulmonary Hypertension	Associated	Inversely correlated with survival ^[7]	Lower 6MWT ^[11]
LVDD	Variable	Not significant	More frequent in severe disease ^[9]

DISCUSSION

Chronic Cor Pulmonale and Remodeling in chronic cor pulmonale, persistent pulmonary hypertension provokes progressive remodeling of myocardial architecture. Weitzenblum et al. (2002) described an evolution from concentric hypertrophy to eccentric dilation, accompanied by fibrosis and myofibrillar disarray. Histopathological studies correlate regional hypokinesis with fibrotic replacement, particularly along the RV free wall and interventricular septum. This chronic remodeling reduces contractile synchrony, producing global wall motion abnormalities and interventricular dependence. Garrison et al. (2023) further noted that chronic hypoxemia diminishes coronary perfusion reserve, thereby exacerbating ischemic wall impairment.

Acute Cor Pulmonale: Dynamics of Dysfunction
Acute cor pulmonale (ACP) develops abruptly due to rapid elevation in pulmonary vascular resistance, most commonly secondary to pulmonary embolism, hypoxic vasoconstriction, or mechanical ventilation with high airway pressures. Vieillard-Baron et al. (2002) illustrated that echocardiographic evaluation at the bedside can reveal acute RV dilation, loss of longitudinal shortening, and paradoxical septal motion. These findings, confirmed by ATS Journals (2002), stem from an abrupt shift of the interventricular septum toward the left ventricle during systole, reducing left ventricular filling and cardiac output. The resulting wall motion pattern differs markedly from chronic forms, emphasizing the utility of serial echocardiographic monitoring.

Interventricular Dependence and Septal Motion
The interventricular septum plays a pivotal role in cardiac mechanics, and its motion abnormalities serve as indirect indicators of RV overload. In cor pulmonale, septal flattening or paradoxical movement during systole reflects pressure overload and altered inter-chamber interactions. Mandoli et al. (2020) described this feature as the “D-shaped” left ventricle seen in short-axis echocardiographic views. Celeski et al. (2024) noted that septal motion distortion not only impairs systolic performance but also contributes to diastolic dysfunction by limiting LV filling. Therefore, assessment of septal motion provides valuable diagnostic and prognostic insights in cor pulmonale.

Segmental Wall Motion in COPD-Related Cor Pulmonale
Cor pulmonale related to COPD presents gradually, driven by chronic hypoxemia and pulmonary vascular remodeling. Gautam et al. (2018)

found that the basal and mid-free wall segments of the RV exhibit early hypokinesis even before significant elevation of pulmonary pressures. Danchin et al. (1987) confirmed that segmental dysfunction correlates with disease severity and arterial hypoxemia levels. The anterior free wall and apical segments typically remain preserved until advanced stages. Such segmental variations emphasize the importance of detailed mapping in echocardiographic analysis rather than reliance on global indices alone.

Strain Pattern Analysis
Strain echocardiography has become the preferred method for quantitative analysis of RV wall motion abnormalities. Vitarelli et al. (2016) and Mandoli et al. (2020) documented that impaired longitudinal strain in the basal and mid-lateral walls closely corresponds to hemodynamic markers of pulmonary hypertension. This technique detects early contractile failure even in the presence of preserved TAPSE values. Oracles (2025) proposed diagnostic cut-offs using global longitudinal strain and strain rate indices to define subclinical cor pulmonale, allowing for early intervention before symptomatic progression.

Diagnostic Criteria and Integration with Clinical Findings
Accurate diagnosis of cor pulmonale requires correlation between echocardiographic, clinical, and laboratory findings. According to Oracles (2025), diagnostic criteria include evidence of pulmonary hypertension, RV hypertrophy or dilation, and characteristic wall motion abnormalities without primary left heart disease. HealthCare Bulletin (2024) highlighted that integrated echocardiographic profiles combining wall motion, valvular dynamics, and hemodynamic parameters improve specificity. IJIRM (2025) also noted that radiological findings, such as pulmonary artery enlargement, should complement echocardiographic assessment to confirm severity and chronicity.

Functional Implications and Hemodynamic Correlates
Wall motion abnormalities directly impair right ventricular output, influencing systemic venous and left ventricular filling pressures. Konstam et al. (2018) emphasized that RV mechanical inefficiency causes reduced stroke volume and venous congestion, culminating in right-sided heart failure. Mohanan et al. (2014) elaborated that in acute settings, severe wall motion impairment can result in cardiogenic shock if not rapidly corrected. Quantitative Doppler measures complement visual assessment by estimating RV systolic pressure, tricuspid regurgitant velocity, and pulmonary vascular resistance.

Comparison Between Acute and Chronic Forms

Feature	Acute Cor Pulmonale	Chronic Cor Pulmonale
Onset	Sudden (minutes to hours)	Gradual (months to years)
Wall motion	Regional akinesis and paradoxical septal motion	Global hypokinesis and dilation
RV remodeling	Minimal structural adaptation	Marked hypertrophy and fibrosis
Echocardiographic findings	RV dilation, D-shaped LV	RV hypertrophy, septal flattening
Outcome	Potentially reversible	Often progressive and chronic

Prognostic Value of Wall Motion Abnormalities
Wall motion abnormalities serve not only as diagnostic markers but also as predictors of prognosis. Medscape (2024) noted that the degree of RV wall dysfunction correlates with survival and exercise tolerance in patients with pulmonary hypertension. Longitudinal strain values below -15% have been associated with adverse outcomes. Mandoli et al. (2020) reaffirmed that normalization of deformation indices following optimized management reflects improved RV-pulmonary artery coupling and better clinical recovery.

Therapeutic Implications and Monitoring
Echocardiographic monitoring of wall motion abnormalities aids in guiding therapeutic interventions. Garrison et al. (2023) and Konstam et al. (2018) highlighted the role of serial echocardiography in evaluating response to oxygen therapy, pulmonary vasodilators, and mechanical ventilation adjustments. Improvements in RV contractility and normalization of septal motion often parallel hemodynamic recovery. Celeski et al. (2024) further suggested that three-dimensional echocardiography offers superior reproducibility in tracking structural reversal following effective management.

Integration with Advanced Imaging and Future Prospects
Advanced modalities such as cardiac magnetic resonance imaging (MRI) and three-dimensional echocardiography have enhanced visualization of RV morphology and wall motion. Mandoli, Bombardini, and D'Andrea (2020) proposed hybrid imaging approaches integrating hemodynamic strain data with cardiac MRI-derived fibrosis mapping for comprehensive assessment. Future investigations aim to quantify wall motion abnormalities through artificial intelligence-assisted imaging, as referenced by PubMed (2022) and Sciencedirect (2024). These tools promise greater diagnostic accuracy, early detection, and improved patient outcomes. Critique methodology, point out possible sources of error such as interobserver variability in echocardiography, and underline the importance of early detection for targeted management strategies.

CONCLUSION

Cardiac wall motion abnormalities in cor pulmonale reflect the continuum between adaptation and decompensation of the right ventricle under

pulmonary pressure overload. Modern echocardiography—both traditional and advanced—has revolutionized diagnosis, allowing for quantitative and qualitative assessment of regional and global dysfunction. From acute to chronic forms, wall motion patterns encapsulate the hemodynamic burden and functional prognosis of affected patients. Integration of strain imaging, Doppler metrics, and advanced imaging provides a comprehensive framework for early detection, monitoring, and management of cor pulmonale. Ultimately, understanding and identifying these wall motion characteristics enable clinicians to optimize care strategies and improve long-term outcomes for individuals with pulmonary-induced cardiac dysfunction.

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