Pulmonary function derangements in isolated or predominant mitral stenosis – Preoperative evaluation with clinico-hemodynamic correlation

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Abstract: Introduction: It is well known that mitral stenosis (MS) is complicated by pulmonary hypertension (PH) of varying degrees. The hemodynamic derangement is associated with structural changes in the pulmonary vessels and parenchyma and also functional derangements. This article analyzes the pulmonary function derangements in 25 patients with isolated/predominant mitral stenosis of varying severity. Aims: The aim of the study was to correlate the pulmonary function test (PFT) derangements (done by simple methods) with: a) patient demographics and clinical profile, b) severity of the mitral stenosis, and c) severity of pulmonary artery hypertension (PAH) and d) to evaluate its significance in preoperative assessment. Subjects and Methods: This cross-sectional study was conducted in 25 patients with mitral stenosis who were selected for mitral valve (MV) surgery. The patients were evaluated for clinical class, echocardiographic severity of mitral stenosis and pulmonary hypertension, and with simple methods of assessment of pulmonary function with spirometry and blood gas analysis. The diagnosis and classification were made on standardized criteria. The associations and correlations of parameters, and the difference in groups of severity were analyzed statistically with Statistical Package for Social Sciences (SPSS), using nonparametric measures. Results: The spirometric parameters showed significant correlation with increasing New York Heart Association (NYHA) functional class (FC): forced vital capacity (FVC, \( r = -0.4^* \), \( p = 0.04 \)), forced expiratory volume in one second (FEV1, \( r = -0.5^* \), \( p = 0.01 \)), FEV1/FVC (\( r = -0.44^* \), \( p = 0.02 \)), and with pulmonary venous congestion (PVC): FVC (\( r = -0.41^* \), \( p = 0.04 \)) and FEV1 (\( r = -0.41^* \), \( p = 0.04 \)). Cardiothoracic ratio (CTR) correlated only with FEV1 (\( r = -0.461^* \), \( p = 0.02 \)) and peripheral saturation of oxygen (SPO2, \( r = -0.401^* \), \( p = 0.04 \)). There was no linear correlation to duration of symptoms, mitral valve orifice area, or pulmonary hypertension, except for MV gradient with PCO2 (\( r = 0.594^* \), \( p = 0.002 \)). The decreased oxygenation status correlated significantly with FC, CTR, PVC, and with deranged spirometry (\( r = 0.495^* \), \( p = 0.02 \)). Conclusions: PFT derangements are seen in all grades of severity of MS and correlate well with the functional class, though no significant linear correlation with grades of severity of stenosis or pulmonary hypertension. Even the early or mild derangements in pulmonary function such as small airway obstruction in the less severe cases of normal or mild PH can be detected by simple and inexpensive methods when the conventional parameters are normal. The supplementary data from baseline arterial blood gas analysis is informative and relevant. This reclassified pulmonary function status might be prognostically predictive.

Keywords: mitral stenosis, pulmonary hypertension, pulmonary function test

Introduction

The associated derangements in pulmonary hemodynamics and pulmonary function in mitral stenosis are known in the literature [1–3]. The increased left atrial pressure leading on to increased pulmonary venous, capillary, and then pulmonary arterial pressure (PAP) forms the basis for the functional or reversible and the fixed structural changes in the lungs, which results in various functional derangements ranging from alterations in pulmonary volumes, compliance, resistance, distribution, and diffusion [1, 2]. However, the assessment of their severity in relation to clinical and hemodynamic grade has given varied results [1–4]. Evaluating the degree of lung function abnormality that can result from mitral disease with simple tests, and assessing their severity, and detection of early changes in mitral stenosis patients should be interesting and is considered a necessity in these patients coming for surgical treatment. The purpose of the study was to make such
an assessment and evaluate the correlation of each of these indices with cardiac indices, thus, find out whether such simple evaluation could correlate with quantitative changes of mitral disease (measured noninvasively) and, thus, to what degree reflect the hemodynamic severity of the disease, and thereby predict prognosis in this surgical subset.

**Methods**

**Study design**

This was a cross-sectional study to assess the pulmonary function before surgery in isolated mitral stenosis.

**Study population**

Twenty-five patients with mitral stenosis of varying New York Heart Association–functional class (NYHA–FC) and severity who were scheduled for mitral valve (MV) surgery in our center were considered for the study group. All patients had symptomatic mitral stenosis with good ventricular function, with no other significant valvar or myocardial disease. Only nonsmokers and those with no respiratory disease (as per symptoms and on chest X-ray [CXR]) were included. The diagnosis of the valvular lesion and its severity was made by clinical examination, chest X-ray, and echocardiography. The severity of mitral valvar lesion was graded (according to the mitral valve orifice area [MVOA] as mild [1.5–1 cm²], moderate [1–1.5], severe [<1.0 cm²], and critical [<0.8 cm²]). The patients were classified into groups based on the severity of pulmonary hypertension as normal (≤30 mmHg systolic pulmonary artery pressure [SPAP]), mild (31–45), moderate (46–65), and severe (>65).

**Pulmonary function**

Pulmonary function test (PFT) measurements were done by simple, day to day, and inexpensive method – spirometry (with computerized pulmonary function system) for measuring the forced vital capacity (FVC), forced expiratory volume in one second (FEV1), peak expiratory flow rate (PEFR), and forced expiratory flow at 50% of FVC (FEF-50). The observed or measured values were expressed as a percentage of predictive values which were computed according to age, sex, weight, and height and were considered normal if they were >80% of predictive values. Oxygen saturation by pulse oximetry and room air blood gas analysis (before surgery) was also considered.

The PFT derangements were classified, according to Miller’s [5] prediction quadrant as: restrictive – FVC <80% predicted, but FEV1/FVC normal or >70%; obstructive – FVC normal, but FEV1/FVC <70% predicted; and mixed – FVC <80% predicted, FEV1/FVC <70% [5]. The patients were classified further based on the severity of ventilatory impairment according to criteria of Conrad [6]: mild, the spirometry values (FEV1 and FVC) being 60–79%; moderate, 41–59%; and severe, <40%. In this mild group, we included those with normal FEV1 and FVC but with early sign of airway obstruction (ESAO) as shown by FEF-50 less than predicted.

With noninvasive assessment of the valvular disease and its severity, and simple methods of assessment of the pulmonary function derangement, the parameters were correlated. The pulmonary function test results were analyzed in relation to patient demographics, clinical grade, severity of valvar lesion, and severity of pulmonary hypertension. Informed consent was obtained from all patients and their families. The study was approved by our research and ethical committee.

**Statistical analysis**

Analysis of the data was done using SPSS 18 (PASW). The data consisted of many continuous variables, and few were categorical as to functional class, pulmonary venous congestion on CXR, the type, and severity of PFT derangement (which were then graded as 0, 1, 2, and 3; thus converting them into nominal variables). The continuous variables were expressed as mean ± SD. Median and interquartile range (IQR) were also tabulated for the hemodynamic and functional parameters. The few categorical variables were expressed in numbers and percentages. Spearman rank correlation \( r \) and two-tailed \( p \) were obtained. A \( p \) value of <0.05 was considered significant. Comparison of mean ranking of subgroups was done by Kruskal–Wallis test.

**Results**

In our study on 25 patients with mitral stenosis, with a female preponderance (see Table I for characteristics), the main symptom was exertional dyspnoea of class II–IV, of varying duration from 3 months to 23 years. Six patients were in class IV with intermittent congestive failure which was controlled with diuretics. Two patients presented with acute pulmonary edema, which was treated conservatively.

Chest X-ray showed cardiomegaly in majority of patients (20 pts. [80%]), which varied from mild to moderate in 12 and severe in 8 cases. Pulmonary venous congestion of varying degree was noted in 21 (84%): mild – 11, moderate – 8, and severe – 2. Abnormal pulmonary vascular pattern with cephalization and peripheral cutoff was seen in 12 cases mainly of moderate to severe pul-
Pulmonary function in mitral stenosis

Our group showed a predominant restrictive pattern in 12, followed by mixed in 8, and obstructive in 1. Three patients with normal values of conventional spirometry had only ESAO (shown by decreased FEF-50), and in one, both were normal. The severity grading was as follows: mild – 10, moderate – 10, severe – 4, and normal – 1. Of the 4 patients with severe dysfunction, the FEV1 was <1 L in 3 and 1.1 L in 1.

The FVC was reduced in 20 patients (80%), while FEV1 was reduced in 16 (64%), PEFR in 19 (76%), and FEF-50 in 19 (76%).

The patients with restrictive pattern had an equal distribution of mild to moderate grade (6 each). Four of these also showed ESAO. In these, 6 had severe pulmonary artery hypertension (PAH), moderate, and 2 mild. The mitral stenosis was critical in 2, severe in 4, and moderate in 6.

The patients with mixed pattern had an equal distribution of moderate and severe grades (4 each). Four also

### Table I

| Parameters                          | N (%) | Range  | Mean ± SD  | Median | IQR |
|------------------------------------|-------|--------|------------|--------|-----|
| Age                                | 20–57 years | 37.28 ± 9.14 | 37 | 12.5 |
| Sex (M:F)                          | 3:22 (12%:88%) |
| NYHA–FC                            | II – 5 (20%) | III – 14 (56%) | IV – 6 (24%) |
| Duration of symptoms               | 3 months – 23 years | 6.75 ± 6.67 | 5 | 7 |
| Cardiothoracic ratio (CTR)         | 37–80% | 56.51 ± 11.51 | 55 | 14.55 |
| MVOA cm²                           | 0.68–1.5 | 0.96 ± 0.21 | 0.9 | 0.175 |
| MV gradient (mmHg)                 | 8–28 | 17.03 ± 6.62 | 14.3 | 10.5 |
| SPAP (mmHg)                        | 31–140 | 64.44 ± 26.85 | 58 | 33.5 |
| LA size (cm)-echo                  | 3.2–8.3 | 5.91 ± 1.19 | 6.1 | 1.60 |
| PA size (cm)-echo                  | 18–41 | 29.92 ± 5.84 | 30 | 9.5 |
| FVC                                | 0.74–2.73 | 1.64 ± 0.55 | 1.74 | 0.81 |
| FVC% predicted                     | 37–114.90 | 66.54 ± 19.18 | 68 | 26.52 |
| FEV1                               | 0.15–2.72 | 1.46 ± 0.57 | 1.39 | 0.69 |
| FEV1 % predicted                   | 10.1–123.50 | 67.16 ± 24.50 | 66 | 37.63 |
| FEV1/FVC                           | 18.19–117 | 83.37 ± 21.17 | 91.57 | 20.77 |
| FEV1/FVC % predicted               | 22.0–132 | 102.06 ± 23.39 | 109 | 18.85 |
| PEFR                               | 0.77–6.23 | 3.31 ± 1.40 | 3.1 | 2.00 |
| PEFR% predicted                    | 13–102.60 | 59.63 ± 24.96 | 63 | 44.59 |
| FEF-50                             | 0.31–6.16 | 2.38 ± 1.41 | 2.29 | 0.89 |
| FEF-50% predicted                  | 5–137.33 | 69.07 ± 35.83 | 68 | 34.63 |
| Preoperative SPO2                  | 94–99% | 97.57 ± 12.25 | 97.95 | 2.40 |
| Preoperative PaO2 mmHg             | 77.60–128.00 | 100.33 ± 12.73 | 100 | 12.05 |
| Preoperative PaCO2 mmHg            | 31–38 | 34.74 ± 1.81 | 35 | 3.15 |

*Abbreviations:* NYHA = New York Heart Association, FC = functional class, CTR = cardiothoracic ratio, SPAP = systolic pulmonary artery pressure, LA = left atrium, PA = pulmonary artery, MVOA = mitral valve orifice area, SD = standard deviation, IQR = interquartile range, LA = left atrium, PA = pulmonary artery, FVC = forced vital capacity, FEV1 = forced expiratory volume in one second, PEFR = peak expiratory flow rate, FEF-50 = forced expiratory flow at 50% FVC, SPO2 = peripheral saturation of oxygen, PaO2 = partial pressure of oxygen in arterial blood, PaCO2 = partial pressure of carbon dioxide in arterial blood.
stenosis of moderate (1) to severe (2) grades. Mild respiratory impairment, and moderate PAH, with critical in 1, and moderate in 4. Severe stenosis was a feature in 3, had ESAO. The PAH grades were mild in 2, and moderate and severe in 3. Severe stenosis was a feature in 3, critical in 1, and moderate in 4.

With a normal spirometry, 3 had ESAO with only mild respiratory impairment, and moderate PAH, with stenosis of moderate (1) to severe (2) grades.

The individual correlations and significance of parameters are as in Table II. Thus, significant correlation of spirometric parameters and oxygen status was seen with NYHA–FC and with pulmonary venous congestion on chest X-ray. Other relevant associations were oxygenation status with cardiothoracic ratio (CTR) and spirometric indices (Fig. 1a, b); PCO2 with MV gradient, and PAH, and PFT grade.

The spirometric parameters showed a steady decline in values with increasing functional class from II to IV, with very low values of FEV1 <1 L/s in class IV, while the small airway parameters of PEFR and FEF did not have a direct relation with the class, though the values showed a decrease in higher class (Fig. 2a). The mean oxygen saturation (SPO2) was as follows: 98.38, 97.65, 96.7% (K–W asymp. sig., 0.161); PaCO2: 33.82, 34.70, 35.58 mmHg (K–W asymp. sig., 0.140); and PaO2: 91.86 mmHg (K–W asymp. sig., 0.135); PaO2: 104.18, 102.58, 101.45, 98.38% (K–W asymp. sig., 0.135); PaO2: 96.7% (K–W asymp. sig., 0.135); PaO2: 104.18, 102.58, 101.45, 98.38% (K–W asymp. sig., 0.135).

| Parameters | Demographics and clinical status | CTR | PVC | MVOA | MV gradient | SPAP |
|------------|----------------------------------|-----|-----|------|-------------|------|
| Duration (of symptom) | NYHA–FC |
| FVC | −0.05 | −0.40* | −0.39 | −0.41* | −0.11 | 0.24 | −0.15 |
| (0.81) | (0.04) | (0.05) | (0.04) | (0.60) | (0.24) | (0.48) |
| FVC% | −0.04 | −0.22 | −0.30 | −0.39* | −0.09 | 0.22 | −0.11 |
| (0.83) | (0.27) | (0.14) | (0.04) | (0.65) | (0.28) | (0.59) |
| FEV1 | −0.13 | −0.5* | −0.46* | −0.41* | −0.06 | 0.10 | −0.18 |
| (0.51) | (0.01) | (0.02) | (0.04) | (0.74) | (0.60) | (0.37) |
| FEV1% | 0.04 | −0.39 | −0.39 | −0.33 | 0.10 | 0.07 | −0.08 |
| (0.84) | (0.05) | (0.05) | (0.10) | (0.610) | (0.73) | (0.67) |
| FEV1/FVC | 0.02 | −0.44* | −0.26 | −0.09 | 0.19 | 0.18 | −0.09 |
| (0.93) | (0.02) | (0.19) | (0.66) | (0.35) | (0.37) | (0.66) |
| FEV1/FVC% | 0.005 | −0.39* | −0.35 | 0.03 | 0.173 | 0.214 | −0.07 |
| (0.98) | (0.04) | (0.08) | (0.87) | (0.41) | (0.30) | (0.73) |
| PEFR | −0.05 | −0.33 | −0.27 | 0.23 | −0.29 | 0.37 | −0.11 |
| (0.81) | (0.11) | (0.19) | (0.27) | (0.151) | (0.07) | (0.58) |
| PEFR% | −0.004 | −0.07 | 0.09 | 0.03 | −0.14 | 0.26 | 0.02 |
| (0.98) | (0.72) | (0.65) | (0.88) | (0.51) | (0.20) | (0.91) |
| FEF-50 | −0.17 | −0.40 | −0.08 | 0.63 | −0.29 | 0.36 | −0.02 |
| (0.40) | (0.05) | (0.09) | (0.76) | (0.16) | (0.08) | (0.91) |
| FEF-50% | −0.11 | −0.17 | −0.08 | 0.11 | −0.24 | 0.30 | 0.09 |
| (0.59) | (0.42) | (0.69) | (0.61) | (0.26) | (0.15) | (0.64) |
| Preoperative SPO2 | −0.14 | −0.41* | −0.40* | −0.40* | −0.114 | 0.025 | −0.34 |
| (0.51) | (0.04) | (0.04) | (0.04) | (0.58) | (0.91) | (0.09) |
| Preoperative PaO2 | 0.01 | −0.38 | −0.51** | −0.56** | 0.24 | −0.19 | −0.34 |
| (0.94) | (0.06) | (0.01) | (0.004) | (0.25) | (0.36) | (0.08) |
| Preoperative PaCO2 | 0.18 | 0.38 | 0.10 | 0.06 | −0.22 | 0.59** | 0.24 |
| (0.37) | (0.05) | (0.63) | (0.76) | (0.28) | (0.002) | (0.24) |

Spearman correlation coefficient (r) in the upper row and two-tailed significance p in the lower row.

*Correlation significant at the 0.05 level; **Correlation significant at the 0.01 level.

Abbreviations: NYHA = New York Heart Association, FC = functional class, CTR = cardiothoracic ratio, PVC = pulmonary venous congestion, MVOA = mitral valve orifice area, SPAP = systolic pulmonary artery pressure, FVC = forced expiratory volume, FEV1 = forced expiratory volume in one second, PEFR = peak expiratory flow rate, FEF-50 = forced expiratory flow at 50% FVC, SPO2 = peripheral saturation of oxygen, PaO2 = partial pressure of oxygen in arterial blood, PaCO2 = partial pressure of carbon dioxide in arterial blood.
and PEFR\% (r = 0.43*, p = 0.03), while the PCO2 values showed negative correlation with only FEV1\% (r = −0.52*, p = 0.007), FEV1/FVC (r = −0.49*, p = 0.01), and SPO2 (r = −0.41*, p = 0.04).

There was no direct or linear correlation of the type or severity of PFT derangements with the degree or severity of PHT. The one with normal PAP had fairly normal spirometry but diminished flow rates. Even some with mild PAH showed significant lung function derangements (Fig. 2b). However, the mixed types and those of severe grades showed a trend towards higher FC, lower oxygen status, and higher PCO2 ([r = 0.431*, p = 0.031]; [r = −0.533**, p = 0.006, r = −0.558**, p = 0.004]; [r = 0.463*, p = 0.02]).
Pulmonary function spirometric parameters did not have any direct or linear correlation to duration of disease or the severity of MS (based on MVOA). The FEV1/FVC% showed a decline with decreasing valve orifice. Parameters of peripheral airway flow disturbances were considerably low in patients with moderate stenosis compared to higher grade (Fig. 2c).

Other cardiac investigations in left atrial (LA) and pulmonary artery (PA) size, did not bear any correlation to PFT.

The mean ranking (Kruskal–Wallis) did not reveal any statistically significant difference between the subgroups in each of the groups (based on each FC, PAH grade, and grade of valvar stenosis), except for the mild dif-
ference with oxygenation status in the functional class mentioned above.

**Discussion**

The most common or dominating symptom of mitral stenosis is dyspnoea and impaired exercise tolerance. The increased left atrial pressure in mitral stenosis leads to reversible and or fixed changes in the lung. The increased lung water or pulmonary congestion and later pulmonary vascular disease with the structural changes form pathophysiologic basis of this, which is then expected to be evaluated by pulmonary function tests [2, 7]. The disturbances reported being reduction in static and dynamic lung volumes, unevenness in ventilation–perfusion ratio, peripheral airway obstruction, and reduction in diffusing capacity in severe stenosis [1, 2].

Studies attempting to correlate the PFT derangements with degree of functional incapacity and hemodynamics in mitral valve disease, with conflicting or varying results, have been published [1, 3, 4, 12]. Lung function studies have noted reduction in many of the parameters [1, 3, 8]. While some early studies as that of Palmer et al. [1], and then of Carmo et al., Strzyzakowska et al., and Saxena et al. [3, 9, 10], could not demonstrate good correlation between the severity of symptoms, clinical grade of the lesion, and the decreased pulmonary function, other studies have reported on correlations between the clinical and hemodynamic severity of the lesion and the pulmonary function derangements [2, 7, 11, 12]. Few have noted the negative correlation between vital capacity and PAP [12]. Carmo et al. noted a restrictive pattern in their series, due to pulmonary congestion, which did not have significant correlations to clinical, hemodynamic, and echocardiographic grades [3]. These variations were probably due to improper selection of cases like inclusion of smokers and those with respiratory disease, as stated by Rhodes et al. [2], also inclusion of combined mitral and mixed valvular lesions, and also because of the wide normal range of pulmonary function tests which really needs a serial assessment, over the course of the disease.

Rhodes et al. found direct correlation of these derangements to severity of the valve disease [2]. Later, studies by Nour et al. confirmed this by adding the point of correlation to severity and respiratory symptoms [8] and Doo et al. correlated derangements with mitral valve orifice area [12]. Studies in Indian patients by Chatterji et al. found that FVC values were reduced in direct proportion to PAP, left atrial pressure, mitral valve area, and gradient in 3/4th of their patients [12]. One study by Ohno et al. is interesting, where it appears to have definite correlation to the degrees of pulmonary hypertension, where he found the PFT to be normal or near normal in patients with normal PAP and mild changes returning to normal after surgery, and with increasing severity, the derangements were more and the degree of reversion towards normal to be less and or prolonged [14]. Though Seboldt et al. could correlate with the grading and severity, he found that all changes were irreversible in spite of hemodynamic improvements [11].

On the whole, PFT is expected to correlate with the severity of the disease as estimated by the degree of pulmonary hypertension [12]. With early changes being functional and reversible, later organic changes like...
hyalinization of capillaries and arterioles occurs and gets progressive, thus, becoming irreversible [7, 11, 14].

Thus, even with many studies, a consistent correlation between pulmonary pressures, function, and the clinical grades is lacking.

Our study was mainly focused on patients with isolated or predominant mitral stenosis in nonsmokers and with no respiratory disease (in contrast to the mixed disease pattern in many previous reports [4, 7, 10, 15]). A predominant restrictive PFT pattern noted in this study stands in similarity with that reported in many previous studies of Chandra et al., Santos et al., and Garcia-Lazaro et al. [15–17], but contrasting to that of Gomez-Hospital et al. who had less number with restriction [18, 19], followed in addition by a mixed pattern with mild to moderate restriction and obstruction. In a recent study, Bitner and Nowak [4] has stressed that derangements result only in mild restriction in patients with valvular disease without left ventricular failure, but we have noted a wide range of severity of the lung functional impairment in these small selected group with good left ventricular function.

The steady decline in spirometric parameters with increasing NYHA–FC was noted as a consistent trend (though not statistically significant), while these did not show any significant correlation with the duration of symptoms, degree of stenosis (MVOA) or PAP, except for an increased PaCO2 in blood gas with higher valve gradient. Thus, the extent of functional status reflects the pulmonary function as stated by Ozyilmaz et al. [20].

In addition, one notable feature was that, in some patients with no significant abnormality of FEV1 and FVC, there was definite reduction in FEF-50 and in PEFR, indicating some degree of small airway obstruction. This can be explained by the fact that, in early stages, the measured pressure in the pulmonary artery is normal, while pulmonary venous congestion might have occurred which leads to submucous venous congestion, airway compression by dilated vessels due to venous congestion, and bronchial epithelial hyperplasia and bronchoconstriction causing (contributing to) some amount of airway obstruction, amounting to reduced FEF-50 and PEFR. This is what is called as early small airway obstruction (ESAO), and this may be a sensitive index of airway obstruction as first noted and reported by Rampulla et al. [21] and later by Kadam et al. [22]. The point mentioned by Rhodes et al. [2] based on their study in 1982 was that airflow abnormalities are unlikely in nonsmokers, but our findings of abnormal FEF in some patients are in agreement with that of Kadam et al. [22]. Studies have described no change in this airflow limitation after intervention [19]; hence, an early detection of this is justified/warranted.

In contrast to what has been found in previous reports that patient with functional class II and those with normal PAP had normal pulmonary function [7], in our study, even those with normal PAP were noted to have pulmonary function abnormalities as found by the simple method performed.

The observation made here that spirometric and other parameters are decreased even in mild PAH cases and do not go along with increasing PAP, indicates that part of the changes may be due to the congestive phenomenon itself, brought by the pulmonary venous congestion, interstitial and alveolar edema, and submucosal edema, and not related directly to PA pressure. PAH in mitral stenosis is often disproportionate with disease severity or left atrial pressure. Thus, the presence of PFT derangements in mild PAH cases reduces the degree of its direct correlation to PAP. Recent studies by Garcia-Lazaro et al. showed that the predominant pattern, though reflective of the lung damage, was not exactly predictive of PAH reversal or regression [17]. A correlation with the structural changes will be more informative and beneficial.

The lack of direct correlation to MVOA in our study is in contrast to that of Chatterji and Doo [12, 13] who have stressed on this aspect. The relation or association with mitral orifice area suggests that pulmonary venous congestion of long standing can contribute to this disorder, though not directly related to severity of the disease.

The correlation of vital capacity to CTR (negative correlation) has been mentioned in many studies [2, 7] where the decrease in vital capacity is caused by increased cardiac volume in addition to the pulmonary congestion, while in our case, only FEV1 and oxygeneation correlated with CTR. The lack of correlation to duration of symptoms can be explained by the subjective nature of the variable, and patient dependent, which cannot be avoided. Few of the absent linear correlation to cardiac investigations in this study resembles that of Carmo et al. and Strzyzakowska et al. [3, 9].

Evaluation of the blood gas showed mild changes with a mean SaO2 as 97.57%, with only one patient having saturation less than 95%. The correlations of these mild changes highlighted the decreasing oxygenation with pulmonary congestion and increased cardiac volume and with deranged spirometry, worsening the functional class. The borderline high PaCO2 of 35–40 mmHg seen in seven cases was mostly in those with moderate or severe pulmonary hypertension (r = 0.400*, p = 0.048) and also with higher mitral valve gradient, and with severe dysfunction types. These indicate that these alterations can be due to pulmonary function disturbances in addition to the cardiac factor. This is an interesting and important factor to be guarded as these patients stand a chance to have fluctuations in blood CO2 status with postoperative hemodynamic changes, with tendency for hypercarbia, needing ventilator adjustments [23].

The impact of pulmonary venous congestion on respiratory system is shown by its correlation with the spi-
rometric parameters and arterial oxygen. Also, one can think of the interstitial and alveolar edema in the early stages to cause or contribute to some restriction in the ventilatory parameters before the true restriction by fibrosis sets in. This early edema and restriction caused by it may be reversible. Of course, study of the changes in the diffusion capacity may add more information to this.

These studies, along with ours, raise the need for a correlation with histopathologic analysis of the structural changes in the pulmonary vessels and parenchyma.

In evaluating the significance of PFT in the preoperative assessment – our last goal, one can say that though PFTs cannot be implemented as the single decision making tool for intervention in severe mitral stenosis, it can be a supplementary or complementary investigation, that even in patients with mild/moderate stenosis or mild pulmonary hypertension and or those with mild symptoms, with PFT changes, the possibility of early structural change is to be considered, and early surgery is advisable without waiting for full symptomatic picture to manifest, as mentioned earlier by Gomez-Hospital in his articles [18, 19, 24]. In those with more severe stages, it can to a certain extent predict the degree of structural damage in the lungs and thereby guard us regarding the prognosis such as high postoperative morbidity, with the probability of ventilatory disturbances in the postoperative period, and also a functional limitation with the possibility of lack of normalization of pulmonary hemodynamics and lack of normalization of the exercise capacity, thus, affecting the overall outcome.

Though the study has its limitations of having small sample size and use of only noninvasive measures, it is particularly focused on nonsmokers, with isolated mitral stenosis and good ventricular function, and factors intrinsic to the disease. This article has demonstrated certain important aspects relevant to the clinician and surgeon, which changes definitely occur even in early stages of mitral stenosis regardless of its duration, and or severity, in contrast to many other reports. Also, it has represented the data showing changes in the oxygenation and ventilation status by blood gas analysis which can be supplementary information.

**Conclusion**

Simple and inexpensive pulmonary function tests indicate that, in mitral stenosis, pulmonary function derangements occur in varying degrees which then relates to the functional class of disability, though not directly correlating with the severity or chronicity of stenosis, or pulmonary pressures. However, these simple methods can detect early small airway disease in patients with mitral stenosis when pulmonary hypertension is not manifested and or conventional spirometric parameters are normal. This, with a baseline blood gas analysis, reclassifies the respiratory function status or respiratory reserve status which adds to their importance in the preoperative evaluation schedule of these patients, which might be useful to predict the postoperative prognosis to certain extent.

**Abbreviations**

PFT: pulmonary function test
CXR: chest X-ray
CTR: cardiothoracic ratio
NYHA: New York Heart Association
FC: functional class
MV: mitral valve
MVOA: mitral valve orifice area
MS: mitral stenosis
PH: pulmonary hypertension
PAH: pulmonary artery hypertension
LA: left atrium
PA: pulmonary artery
SPAP: systolic pulmonary artery pressure
PVC: pulmonary venous congestion
FVC: forced vital capacity
FEV1s: forced expiratory volume in one second
PEFR: peak expiratory flow rate
IFE: forced inspiratory flow rate
FVE: forced expiratory flow rate
PaO2: partial pressure of oxygen in arterial blood
PaCO2: partial pressure of carbon dioxide in arterial blood
K-W: Kruksal-Wallis test
SD: standard deviation
IQR: interquartile range
SPSS: Statistical Package for Social Sciences

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