CLINICAL STUDY

Pulmonary function alterations after correction of mitral stenosis

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Abstract

The aims of the study is to demonstrate the degree of pulmonary function reversibility after successful corretion of mitral stenosis, transvenous as well as surgical. Before and in the short- and long-term follow-up after mitral stenosis correction (mitral valve replacement and percutaneous balloon mitral valvuloplasty) echocardiographic and pulmonary function studies were performed in 125 patients with pure or dominant mitral stenosis in order to investigate the influence of hemodynamic changes on pulmonary function. Immediately after procedure moderation of bronchial obstruction could be detected although only in balloon valvuloplasty group. In pts after surgery dramatic decline of pulmonary function due to thoracotomy was found. In the long-term follow-up substantial improvement of all ventilatory parameters in both groups was revealed, diffusing capacity remained unchanged. The favourable evolution was comparable in both groups. The explanation are time consuming benefitial peripheral metabolic, circulatory and organic pulmonary changes, what implies also irrelevant changes of PF in the early phase and unalterable diffusing capacity. ***(Tab. 1, Fig. 4, Ref. 15.)***
Key words: pulmonary function, mitral valve replacement, mitral balloon valvuloplasty.

Mitral valve disease, especially mitral stenosis (MS) represents an important cause of pulmonary hypertension. While the pulmonary hypertension is initially a result of backward transmission of the elevated left atrial pressure, many patients (pts) subsequently exhibit marked pulmonary vasoconstriction, so that pulmonary hypertension is reactive as well as passive. Patients produce well recognized abnormalities of pulmonary function (PF) and develop principal symptoms of this valvular lesion: poor exercise tolerance and exertional dyspnea. Several factors causing these dominating clinical signs have been suggested. They include:

— low cardiac output due to obstruction of left ventricle inflow evoking early anaerobic metabolism and excessive production of lactic acid from inadequately perfused muscles,
— abnormal drive to breathe resulting from stimulation by pulmonary congestion and lactic acid,
— reduced lung compliance due to increased pulmonary capillary pressures,
— increased use of anatomic space due to rapid shallow breathing,
— increased dead space due to ventilation-perfusion mismatch (Tanabe et al., 1996).

These effects will be compounded by any inefficiency of PF resulting from reduced compliance, increased airways resistance and hindered gase exchange. The most fundamental mechanisms of pulmonary function disturbances are pulmonary congestion and pulmonary vascular disease.

With advancing stenosis of mitral valve persistent elevation of the pulmonary arterial pressure directs a brunt of pressure load on the right ventricle, thereby evoking tricuspid incompetence. At this point of natural history of MS the symptoms, especially respiratory, can modify and right ventricle failure sings dominates.

The severity of pulmonary pathology varies from patient to patient and the reversibility of lung damage is likely depending on the natural history of the individual. In many cases there is only slight impairment of PF and due to wide normal range of PF tests the dysfunction can be overlooked. The only possibility to detect the severity of PF disturbance is to evaluate PF in the follow-up after MS correction, i.e. after improvement of pulmonary hemodynamics.

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Mitral valve commisurotomy or replacement (MVR) are standard approaches in the treatment of pts with MS. In the 80th the advances in transcatheter techniques has allowed the stenotic valve to be opened by inflating a balloon catheter at the valve. Nowadays percutaneous balloon mitral valvuloplasty (PBMV) using Inoue balloon catheter has been proved as an effective treatment method, avoiding sternotomy and other negatives of surgery in extracorporeal circulation. Investigation of pts undergoing PBMV allows to assess the acute and direct effects of mitral obstruction relief on pulmonary hemodynamics and function.

Follow-up studies confirmed persistence of hemodynamic benefit after MS correction (Turcu et al., 1991). However complex evaluation of pulmonary dysfunction after both aproaches — successful MVR and PBMV in the long-term follow-up have received little attention. Therefore, the objectives of this study performed in a prospective manner were as follows: (1) to evaluate MVR and PBMV results in the long-term follow-up, (2) to demonstrate the degree of PF disturbance before correction of valve lesion, (3) to elucidate the degree of recovery of pulmonary function early after MVR and PBMV and in the long-term follow-up and (4) to compare the influence of both methods on the degree of PF reversibility.

Material and methods

The study population consisted of 125 symptomatic pts with pure or dominant MS. In *group I*, i.e. 57 (M/F=14/43) pts with mean age 55±6 years, stenotic mitral valve was replaced by mechanical prosthesis. *Group II*, i.e. 68 (M/F=16/52) pts with mean age 53±7 years, underwent successful PBMV by Inoue balloon technique. None of the pts had history of any particular disease of respiratory system. None significant difference between input data of selected two groups was found (Tab. 1).

Within a week before PBMV or surgery and in the early (7±4 days) and late (15±6 months) follow-up echocardiographic (ECHO) and PF studies were performed.

Based on transthoracic and transoesophageal ECHO findings (2-D, Doppler studies) (Hewlett Packard Sonos 1000,2000 echocardiograph) mitral valve area (MVA) and mean mitral gradient (mGrad) were measured, tricuspid regurgitation and pulmonary hypertension (PH) were evaluated, valve morphology was scored (Wilkins et al., 1988). According to the latest only pts with favourable valve morphology (ECHO score ≤10) undergoing PBMV were selected. Pts with critical values of pulmonary artery pressure, with hemodynamic relevant mitral regurgitation before and after procedure, with relevant restenosis or prosthetic valve dysfunction in the long-term follow-up were excluded.

PF testing using body pletyzmograph (Masterlab Jäger) (FVC, FEV1, TLC) and single-breath method (Diffusiontest Jäger) (DlCO) was performed. PF parameters were expressed in percent of predictive value according Krištúfek.

Results were analyzed by the Student∞s paired *t*-test and differences were considered significant when *p* values were less than 0.05.

Results

ECHO data (Tab. 1, Fig. 1) demonstrate tighter MS and advanced pulmonary hypertension in *group I* in comparison to *group II* although without significance. ECHO parameters immediately after PBMV and approximately a week after MVR as well as in the follow-up (Fig. 1) showed substantial increase of MVA, decrease of mGrad and reduction of PH. In *group I* persisted higher value of pulmonary artery pressure, but didn't achieve significance.

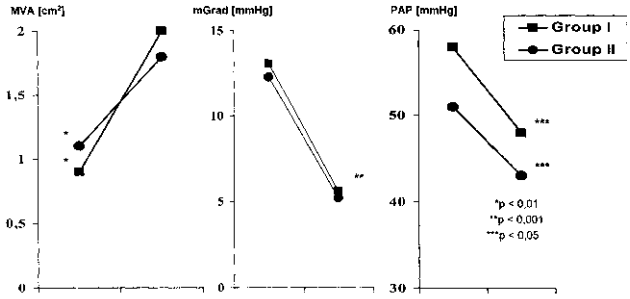

Fig. 1. Evolution of MVA, mGrad, PAP in the follow-up.

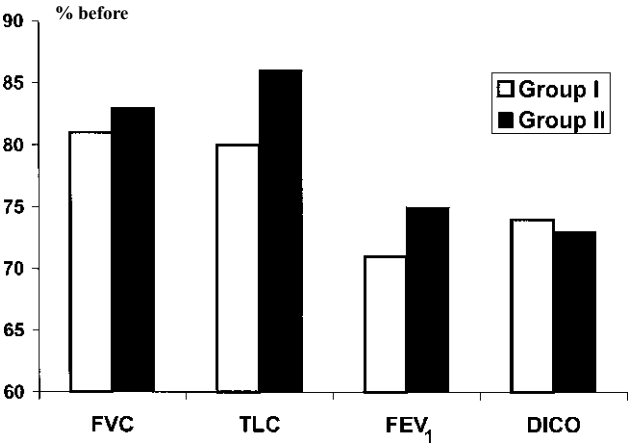

Fig. 2. Preprocedural PF.

PF testing before MVR and PBMV (Fig. 2) revealed borderline FVC, TLC, DlCO in both groups, in *group I* reduced FEV_1 (71 % pred vs 75, NS). Evaluation of PF in *group I early after MVR* (Fig. 3) [Δ = post-preprocedural values]) detected substantial impairment of all followed parameters except DlCO with high significance. *Early postPBMV* (Fig. 3) FEV_1 increased in *group II* significantly (Δ = 7 % pred, $p<0.05$). In follow-up *late* assessment (Fig. 4) of PF showed restitution of postoperative pulmonary dysfunction in pts after *MVR*. In comparison with preprocedural values all parameters improved, although significantly ($p<0.05$) only FVC in *both groups* and FEV_1 in *PBMV group*.

Tab. 1. Baseline characteristics of study population.

Group	I	II
N	57	68
Age (yrs)	55±6	53±7
MVA ($m^{2)}$	0.9±0.3	1.1±0.2
mGrad (mmHg)	13.1±2.2	12.3±3.6
PAP (mmHg)	58±8	51±11

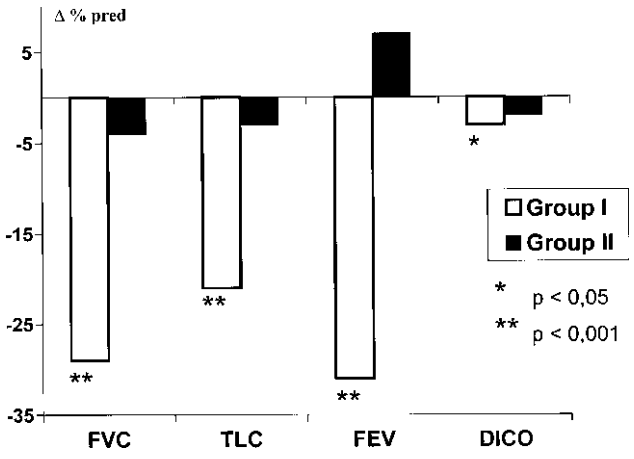

Fig. 3. Post-preprocedural differences in PF in early follow-up.

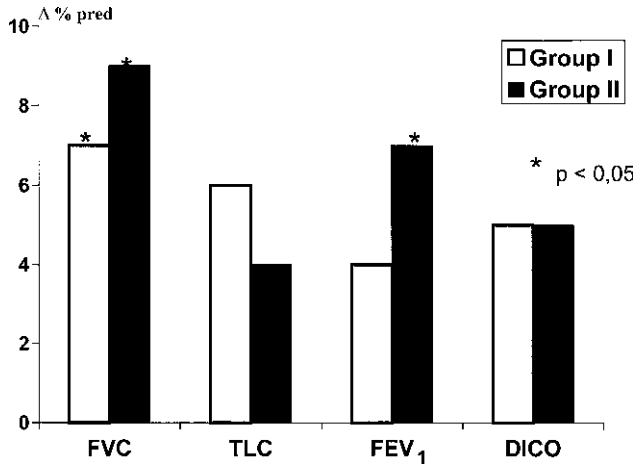

Fig. 4. Post-preprocedural differences in PF in late follow-up.

Discussion

The effects of mitral valve disease on pulmonary function are well known and include (1) reduction in static and dynamic volumes, (2) peripheral airway obstruction, (3) reduction in pulmonary diffusing capacity. It is not resolved, if the crucial factor of PF disturbances are organic changes of lung interstitium or functional hemodynamic changes (Grossman and Braunwald, 2000). The determinating role probably play (1) the interindividual variability of pulmonary vasculature reaction on pulmonary hypertension, (2) duration and (3) severity of valvular lesion. Important seems to be the presence of bronchial hyperreactivity, occuring due to pulmonary congestion (Nishimura et al., 1994). The pathological mechanisms are multiple, yet not clear.

In *preprocedural PF testing* detected borderline values of spirometric volumes in both groups indicate due to extreme wide range of normal values *the trend* to restrictive as well as obstructive ventilatory disturbance. Definite airway obstruction however we could find in the *group I* with significant difference in comparison to *group II*. This fact could be explained by slight more advanced MS and pulmonary hypertension in *group I*. Comparable results could be detected in pts with coequal MVA (Ohno et al., 1987, Yoshioka et al., 1990). About DlCO very controversary opinions are reported (Yoshioka et al., 1990; Ray et al., 1994; Messner-Pellenc et al., 1996). If we summarise, DlCO at rest doesn't differ from normal subjects significantly, more over may be increased possibly due to an enlargement in pulmonary capillary blood volume (Ota et al., 1994). Substantial decrease in DlCO has been reported particularly in pts with a longer disease duration. This reduction can be explained by anatomical alterations of alveolar capillary membrane (thickening of capillar and alveolar walls and/or obliteration of proportion of the capillary bed) leading to abnormally low diffusing capacity. In our study groups DlCO was slightly decreased comparable with data in pts with pronounced pulmonary hypertension (Ohno et al., 1987; Yoshioka et al., 1990; Ray et al., 1994).

Since corrective surgery on mitral valve has become standard practice much informations has accumulated on positive hemodynamic changes after MVR. The question about reversibility of pulmonary dysfunction remained open. It has generally been believed, that MVR prevents further deterioration of, but does not improve already impaired PF. However failure of PF improvement in many cases could be explained by heart surgery itself, by short-term follow-up, by evaluation without respecting the disease severity, respiratory diseases association, smoking history (Yoshioka et al., 1990; Šimková et al., 1997). The crutial moment seems to be the early timing of PF evaluation after surgery (0—6 months) (Rhodes et al., 1985; Mustafa et al., 1984), because objective assessment of PF is possible after 6 months (Šimková et al., 1997). Respecting above mentioned and in order to avoid these limitations we excluded smokers, pts with COPD history and the variables (severity of MS) in our study groups were comparable. The control assessments were performed 15±6 months after correction of MS.

MS is now commonly treated by ballon dilatation and this has provide opportunity to observe the effects of relieving mitral valve obstruction free from obscuring effects of open heart surgery including timing limitation. PBMV provides good long-term results in pts with MS with pliable, non-calcified valve with low ECHO score (Turku et al., 1991; Fridrich and Mizera, 1998). PBMV seems te be a very suitable treatment method for detection of immediate pulmonary hemodynamic and functional changes due to relief of MS like demonstrated in previous study (Yoshioka et al., 1990). The authors presented ventilatory function improvement in a week after PBMV, what means reversible hemodynamic alterations.

We demonstrated objective improvement of hemodynamics (increase of MVA, decrease of mGrad and PH) in the long-term follow up in both groups. More over we focused on evaluation of respiratory reserve during followed period.

Early postprocedural PF changes are evidently more favourable in *group II*. Increase of FVC,TLC, FEV1 can be explained by regression of pulmonary congestion and vasoconstriction after PBMV. Pts sensed relief of dyspnoe immediately after dilatation of the valve corresponding with hemodynamic changes. Controversary in *group I* we detected dramatic decline of all spirometric parameters, DlCO remained unchanged. This is the logical consequence of open heart surgery (Šimková et al., 1997; Šimková and Krištúfek, 1997). Therefore positive response in pulmonary he-

modynamics coudn't be followed by improvement of PF and thus we coudn't detect substantial improvement.

Immediate benefitial effects of PBMV on hemodynamics and PF create conditions for exercise performance enhancement and subjective improvement in short-term follow-up studies (Messner-Pellenc et al., 1996). In our pts significant decrease of NYHA class was detected early after correction of MS only in *group I*. The reconvalescence in *group II* lasted for more than 6 weeks.

Late postprocedural PF tests revealed significant improvement of FVC and TLC, i.e. disappearance of restrictive pattern. The enhancement of FVC was less expressed in *postMVR group*, might represent a residual effect of open heart surgery or perhaps advanced pulmonary changes in pts undergoing surgical correction. It has been reported, that postoperative improvement of vital capacity may be related not only to relief of pulmonary congestion, but to the reduction of cardiac size (Rhodes a spol., 1985). In our pts we didn't found any substantial differences in cardiac size (i.e. cardiothoracic ratio changed in *group I* from 58 % to 56 %). Abnormalities of FEV_1 preoperatively indicated airways obstruction. The values in acute as well as in chronic evaluation reached normal range in all patients, the enhancement was significant only in *group II.* Short-term follow-up after PBMV detected immediate improvement of obstruction in peripheral (Yoshioka a spol., 1990) and central airways (Messner-Pellenc a spol., 1996). Airway obstruction is determined not only by congestion, important is the presence of bronchial hyperreactivity as well as chronic organic changes like fibrosis of peripheral airways and alveoli. The particular reversibility in followed pts suggests more hemodynamic changes, although a partial regression of organic changes couldn't be excluded.

Preprocedural values of DlCO in our pts (both groups) were slightly reduced. To what extent this abnormality is reversible in the late follow-up after relief of MS is uncertain, because performed studies lasted only for 3—6 months (Yoshioka a spol., 1990; Ray et al., 1994; Messner-Pellenc et al., 1996). The failure of DlCO to increase suggests that the effects of MS on small pulmonary vessels and alveolar capillary membrane may have been no longer reversible, or need longer period to regress. In our study we couldn't find significant changes of DlCO in the whole followed period too, although the trend indicates slight improvement in both groups.

The issue of our results can be characterised as follows: (1) evolution of preprocedural impairment of PF showed in the early investigation improvement only in *pts after PBMV* in peripheral airway obstruction, a very sensitive indicator for pulmonary congestion and pulmonary hemodynamics changes, (2) controversary *pts after MVR* in the early period demonstrated dramatic decline of ventilatory parameters — a reflection to open heart surgery, (3) significant improvement of crutial PF parameters in the long-term evaluations suggest partial reversibility of pulmonary hemodynamic and structural abnormalities, (4) less expressed beterment in the *postMVR group* is due to perhaps more severe valvular lesion and postthoracomy consequencies, (5) the positive influence of MS correction on pulmonary dysfunction is comparable in the both approaches — surgical as well as transcatheter.

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