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EFFECT OF HEART RATE ON HEMODYNAMICS IN MITRAL STENOSIS

Mohammad Hafizullah[1], Mohammad Irfan[2], Jabar Ali[3], Adnan Mehmood Gul[4], Hikmatullah Jan[5]

[1-5] Department of Cardiology, Lady Reading Hospital & Khyber Medical University, Peshawar - Pakistan

Address for Correspondence:
Dr. Mohammad Hafizullah,
Professor,
Department of Cardiology,
Lady Reading Hospital & Khyber Medical University, Peshawar - Pakistan

E-mail: mhu5555@gmail.com

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All the authors contributed significantly to the research that resulted in the submitted manuscript.

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ABSTRACT

Objective: To assess the effect of heart rate (HR) on haemodynamic parameters in patients with Mitral Stenosis (MS).

Methodology: The study was conducted at Cardiology department, Lady Reading Hospital, Peshawar from November 2010 to April 2011. Patients with MS, regardless of severity, were included. Patients with severe heart failure, other valvular or structural heart disease were excluded. Echocardiographic parameters were recorded at slow and fast HR. Patients with tachycardia were given β-blockers and patients with bradycardia were given parenteral Atropine.

Results: A total of 60 patients were included, females were 57 (78%). Mean age was 31± 9 years. Mean slow and fast HR was 77±12bpm and110±13 bpm, respectively. Peak mitral valve gradient (PMVG) slow vs. fast HR was 12.8±4.80 and 14.93±7.18 mm Hg (p=0.000). Mean mitral valve gradient (MMVG) at slow vs. fast HR was 6.62±3.29 and 8.15±4.88 mm of Hg (p=0.000). E pulse Doppler (E) at slow vs. fast HR was 168±35 and 181±40 cm/s (p=0.013), while E tissue Doppler ($E^{/}$) velocity was 10.47±2.81 and 10.97±2.38 cm/s (P=0.098), respectively. $E/E^{/}$ ratio for slow and fast HR was 17±5.63 vs. 17±5.41 (P=0.792). Right ventricular systolic pressure (RVSP) at slow vs. fast HR was44±16 vs.49±17.05mm of Hg (P=0.001). The above parameters had insignificant change with the HR when there was accompanying more than mild MR.

Conclusion: Slowing HR in patients with MS significantly decreased PMVG, MMVG and RVSP. LV function did not change significantly with HR. Rate control drugs may be used in preference to improve symptoms in moderate and severe MS.

Key Words: Mitral Stenosis, Doppler echocardiography, RVSP, $E/E^{/}$ ratio.

INTRODUCTION

Rheumatic heart disease remains unconquered in developing countries and still presents as a formidable challenge.[1] Mitral Stenosis (MS) is the most commonly encountered presentation of rheumatic heart disease in developing countries like ours.[2] Patient with Mitral Stenosis become symptomatic with increase in left atrial size and elevation of pulmonary hypertension when mitral valve area reduces below 1.5 cm^2 and this warrants intervention.[3] Patients are more symptomatic when the heart rate is elevated, as during exercise.[4] Conversely, in asymptomatic patients with severe MS (mean gradient >10 mm Hg and mitral valve area [MVA] <1.0 cm^2), or symptomatic patients with moderate MS (mean gradient of 5 to 10 mm Hg and MVA of 1.0 to 1.5 cm^2), the measurement of pulmonary artery pressures (measured from the tricuspid regurgitant velocity) during exercise or dobutamine stress echocardiography can help distinguish those who could benefit from valvuloplasty or valve replacement.[4-6] Moreover, for a given MVA, patients with reduced atrioventricular compliance show a more pronounced increase in pulmonary arterial pressure during exercise or dobutamine than those with normal compliance. Hence, in some patients determined to have only moderate MS at rest, the physiologic effects of heart rate sensitivity and atrioventricular compliance can produce exercise-induced pulmonary hyper-tension and exertional dyspnea.[3,5] The current ACC/AHA guidelines have given a Class I recommendation (Level of Evidence: C) for stress echocardiography in patients with MS and discordance between symptoms and stenosis severity.[2] The threshold values proposed by the ACC/AHA guidelines for consideration for intervention are a mean transmitral pressure gradient >15 mm Hg during exercise or a peak pulmonary artery systolic pressure > 60 mm Hg during exercise.[2] In patients with pulmonary artery pressures or valve gradients above these values, percutaneous balloon valvotomy or surgical intervention is recommended, even for patients with apparently moderate MS at rest.[7-9]

Beta blocker therapy for mitral stenosis is controversial. Although pulmonary capillary wedge pressures (PCWP) were lower during exercise in the β-blocker group (22±4 vs. 31 ± 9 mmHg; P>0.05), exercise performance was not enhanced and cardiac output response during exercise was reduced (control = 41% increase vs. 12% for β-blockade).[10] In another study β blocker Atenolol (50 to 100 mg/day) were assessed in 15 patients with mitral stenosis at rest and during upright bicycle ergometry. Beta blocking therapy did not improve exercise time, external work, maximal oxygen consumption rate, or anaerobic threshold. However Atenolol decreased heart rate by 20 % and prolonged diastolic filling period by 40%.[11] Both continuous-wave and pulsed-wave Doppler echocardiography can provide accurate quantification of the transvalvar gradients.[12,3] Mitral stenosis critically limits the mitral flow during exercise and can provoke hemodynamic deterioration.[13]

The objective of this study was to assess the effect of heart rate on haemodynamics measured by different echocardiographic parameters in patients with Mitral Stenosis.

METHODOLOGY

The study was conducted by Cardiology department, Postgraduate Medical Institute, Lady Reading Hospital Peshawar from November 2010 to April 2011. Patients with Mitral Stenosis, regardless of the severity, presenting to Cardiology department in outpatient department or echocardiography suite were included in the study. Informed consent was taken from every patient for study participation. Patients with severe heart failure, other valvular disease or other structural heart disease were excluded from the study. Echocardiographic parameters were recorded at slow and fast heart rate according to established protocol. Patients with tachycardia (heart rate ⩾100/minute) were given beta blockers to reduce heart rate and patients with bradycardia (heart rate ⩽60/minute) were given intravenous Atropine in multiple of 0.5 mg to increase heart rate. Every patient served as his own control. Data was recorded on a study proforma. SPSS version 17 was used to analyze the data. Paired T test was applied to compare the parameters at low and fast heart rate.

RESULTS

A total of 60 patients were included in the study, females were 57 (78%). Mean age was 31± 9 years. Mean slow heart rate (HR) was 77±12 bpm while mean fast heart rate was 110±13 bpm. Ten patients (17%) had more than mild mitral regurgitation. Peak mitral valve gradient (PMVG) at slow heart rate was 12.8±4.80 mm of Hg which increased to 14.93±7.18 mm (P=0.000) when heart rate increased translating into an increase of 0.0645 mmHg per beat (Figure 1).

Mean mitral valve gradient (MMVG) at slow heart rate was 6.62±3.29 mm of Hg which increased to 8.15±4.88 mm of Hg on increasing heart rate from 77 to 110 beats per minute meaning increase of 0.046 mmHg per beat or 0.46 per 10 beats (p=0.000) (Figure 2).

E pulse Doppler (E) was 168±35 cms/s at slow heart rate where as it was 181±40 cms/s (p=0.013) at fast heart rate. E tissue Doppler (E′) velocity at slow and fast heart rate was 10.47±2.81 cms/s vs. 10.97±2.38 cms/s (P=0.098) respectively. Left ventricular function assessed by E/E′ ratio for slow versus fast heart rate was 17±5.63 vs. 17±5.41 (p=0.792) respectively. (Figure 3) Right ventricular systolic pressure (RVSP) at slow heart rate was 44±16 mm of Hg

Figure 1: Peak MVG and Heart Rate

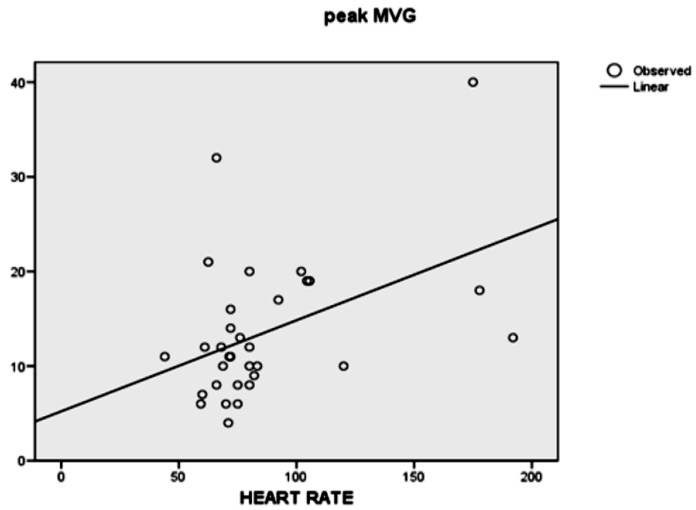


Figure 2: Mean MVG and Heart Rate

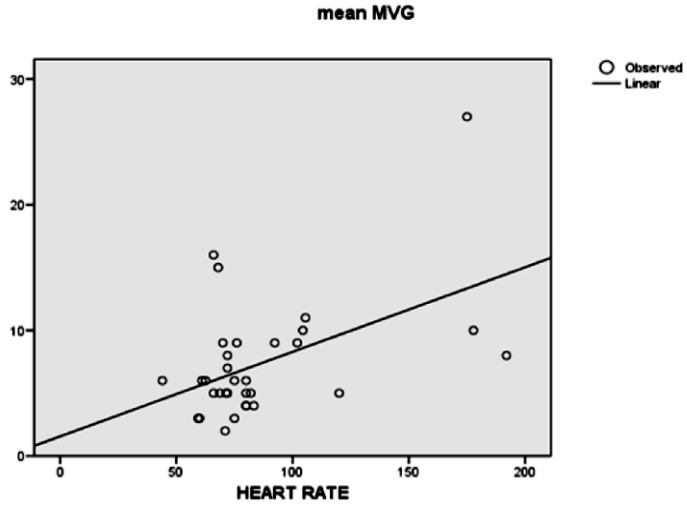


Figure 3: E/E′ and Heart Rate

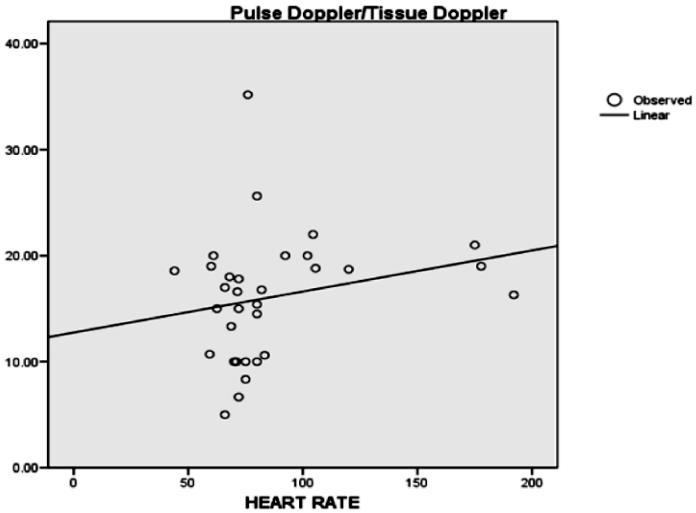
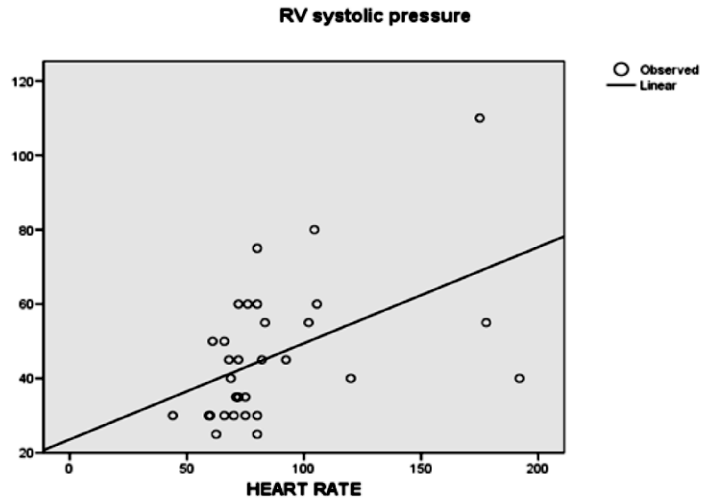


Figure 4: RVSP and Heart Rate



which increased to 49±17.05mm of Hg (p=0.001) at fast heart rate. (Figure 4). The above parameters had insignificant change with the heart rate when there was accompanying more than mild mitral regurgitation.

DISCUSSION

This study is the first in Pakistan to study the effect of heart rate on cardiac hemodynamic measured by different echo parameter in patients with Mitral Stenosis of different severity. A total of 60 patients were included in this study and 57(78%) were females. For a long time it has been recognized that patients can have severe Mitral Stenosis and yet be asymptomatic, although this state does not usually last long. Conversely, a patient can have Mitral Stenosis that objectively ought to be mild, with valve area more than 1.5 cm^2 and be quite symptomatic. It is these patients where there is a need to evaluate beyond a history, physical examination, and a resting echo Doppler. Symptoms of dyspnea at rest or on exertion, orthopnea, and paroxysmal nocturnal dyspnea can be related to either medically treatable hemodynamic changes with Mitral Stenosis or to concomitant diseases, such as chronic obstructive pulmonary disease or coronary artery disease. To better evaluate such patients, measurement of the hemodynamic during exercise, in the past by catheterization and more recently by Doppler echocardiography, has been used.[4]

Our study documents that both peak and mean gradient decreased significantly by reduction in heart rate in patients with Mitral Stenosis measured by Doppler echo-cardiography. In one study Dobutamine Stress Echo (DSE) was performed in 53 patients with mean age of 37.4 ± 11.3 years. During follow-up (60.5 ± 11.0 months), 29 patients presented with clinical events; the best performance of DSE for prediction of clinical events was at a cutoff value of 18 mm Hg DSE-mean gradient which had a sensitivity of 90%, specificity of 87%, and accuracy of 90%.In this study the authors stated the addition of DSE to 'conventional cardiology work-up' would allow a 17% increment for detection of high-risk patients in the entire population and 40% increment in patients with presumed moderate disease.[4] Conventional measures of mitral inflow velocities (E and A) are limited during sinus tachycardia, because the E and A wave tend to become fused at heart rates higher than 90 beats per minute. However, the E/E/ ratio still provides useful information about filling pressures in the setting of sinus tachycardia.[15] Similarly, the presence of atrial fibrillation greatly reduces the amount of information that can be gleaned from traditional mitral in-flow measurements since there is no A wave. Once again, published data suggest that E/E/ still gives information about hemodynamic and carries prognostic significance in atrial fibrillation.[16] But in our study the E/E/ ratio for slow vs. fast heart remained the same thus statistically insignificant.

We also studied Right ventricular systolic pressure (RVSP) which decreased appreciably at slow rate as compared to fast heart rate. Tunick et al[17] using post-exercise Doppler recordings found that patients who were limited by dyspnea exhibit a significant higher increase in SPAP (systolic pulmonary artery pressure) during exercise, as well as shorter exercise capacity, than those who were limited by fatigue. Hecker et al[18] showed that dyspnea elicited by dobutamine or exercise was related with the increase in valve gradient.

Reduction in peak and mean mitral valve gradient with reduction in right ventricular systolic pressure can be used to clinical advantage in symptomatic patients with moderate and severe Mitral Stenosis. Slowing of heart rate does not affect left ventricular systolic function the favourable effects are not confined to sinus rhythm and patients in atrial fibrillation with fast ventricular response also benefit from

rate control. Employment of beta blockers and Calcium Channel blockers can effectively reduce heart rate and favourably effect symptoms in the setting of moderate to severe Mitral Stenosis. Such like drugs or new agents like Ivabridine which effectively reduce heart rate may be used as drugs of first choice to control symptoms before percutaneous intervention. As against patients treated with large dosage of diuretics resulting in dehydration and electrolyte abnormalities patients treated with heart rate controlling therapeutic agents may have more favourable effect on symptoms.

CONCLUSION

Slowing the heart rate significantly decreased the peak and mean mitral valve gradients and right ventricular systolic pressure. These changes were insignificant if there was accompanying more than mild mitral regurgitation. Tissue Doppler parameters were not significantly changed with heart rate. Effective control of heart rate by employing drugs may offer advantage in ameliorating symptoms in patients afflicted with moderate to severe Mitral Stenosis.

REFERENCES

1. Gul AM, Hassan MU, Hafizullah M. Rheumatic heart disease in urban school children of Peshawar. J Postgrad Med Inst 2009;23:337-40.
2. Faheem M, Hafizullah M, Jan J, Khan AM. Pattern of valvular lesions in rheumatic heart disease. J Postgrad Med Inst 2007;21:99-103.
3. Hafizullah M. How severe is severe mitral stenosis? J Postgrad Med Inst 1998;12:17-22.
4. Bonow RO, Carabello BA, Chatterjee K, de Leon AC Jr, Faxon DP, Freed MD, et al. 2008 Focused update incorporated into the ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Valvular Heart Disease). Circulation 2008;118;e523-661.
5. Schwammenthal E, Vered Z, Agranat O, Kaplinsky E, Rabinowitz B, Feinberg MS. Impact of atrioventricular compliance on pulmonary artery pressure in mitral stenosis: an exercise echocardiographic study. Circulation 2000;102:2378-84.
6. Reis G, Motta MS, Barbosa MM, Esteves WA, Souza SF, Bocchi EA. Dobutamine stress echocardiography for noninvasive assessment and risk stratification of patients with rheumatic mitral stenosis. J Am Coll Cardiol 2004;43:393-401.
7. Sagar KB, Wann LS, Paulson WJ, Lewis S. Role of exercise Doppler echocardiography in isolated mitral stenosis. Chest 1987;92:27-30.
8. Sicari R, Niohyannopoulos P, Evangelista A, Kasprzak J, Lancellotti P, Poldermans D, et al. Stress echocardiography consensus statement of the European Association of Echocardiography. Eur J Echocardiogr 2008;9:415-37.
9. Thomas L, Joseph P, Steven R, Bernard J. Effects of chronic β-blockade therapy on rest and exercise hemodynamic in mitral stenosis. Cathet Cardiovasc Diagn 1995;35:110-5.
10. Stoll BC, Ashcom TL, John JP, Johnson JE, Rubal BJ. Effect of atenolol on rest and exercise hemodynamic in patients with mitral stenosis. Am J Cardiol 1995:75:482-4.
11. Nishimura RA, Rihal CS, Tajik AJ, Holmes DR. Accurate measurement of the transmitral gradient in patients with mitral stenosis: a simultaneous catheterization and Doppler echocardiographic study. J Am Coll Cardiol 1994;24:152-8.
12. Wang A, Ryan T, Kisslo KB, Bashore T, Harrison JK. Assessing the severity of mitral stenosis: variability between noninvasive and invasive measurements in patients with symptomatic mitral valve stenosis. Am Heart J 1999;138:777-84.
13. Leavitt JI, Coats MH, Falk RH. Effects of exercise on transmitral gradient and pulmonary artery pressure in patients with mitral stenosis or a prosthetic mitral valve: a Doppler echocardiographic study. J Am Coll Cardiol 1991;17:1520.
14. Dahan M, Paillole C, Martin D, Gourgon R. Determinants of stroke volume response to exercise in patients with mitral stenosis: a Doppler echocardiographic study. J Am Coll Cardiol 1993;21:384-9.
15. Jan H, Hassan MU, Hafizullah M. Long term echocardiographic follow up of percutaneous tranvenous mitral commissurotomy (PTMC). J Postgrad Med Inst 2004;18:529-34.
16. Cheitlin M. Stress echocardiography in mitral stenosis: when is it useful? J Am Coll Cardiol 2004;43:402-4.
17. Nagueh SF, Mikati I, Kopelen HA, Middleton KJ, Quinones MA, Zoghbi WA. Doppler estimation of left ventricular filling pres-sure in sinus tachycardia. A new application of tissue doppler imaging. Circulation 1998;98:1644-50.
18. Okura H, Takada Y, Kubo T, Iwata K, Mizoguchi S, Taguchi H, et al. Tissue Doppler-derived index of left ventricular filling pressure, E/E', predicts survival of patients with non-valvular atrial fibrillation. Heart 2006;92:1248-52.

19. Tunick PA, Freedberg RS, Gargiulo A, Kronzon I. Exercise Doppler echocardiography as an aid to clinical de cision making in mitral valve disease. J Am Soc Echocardiogr 1992;5:225-30.

20. Hecker SL, Zabalgoitia M, Ashline P, Oneschuk L, O'Rourke RA, Herrera CJ. Comparison of exercise and dobutamine stress echocardiography in assessing mitral stenosis. Am J Cardiol 1997;80:1374-7.