Pulmonary artery hypertension in mitral stenosis: Role of right ventricular stroke volume, atrio-ventricular compliance, and pulmonary venous compliance

Sir,

The normal mitral valve area (MVA) is approximately 4–6 cm^2. At a MVA below 1 cm^2 (severe mitral stenosis [MS]) patients usually become symptomatic even at rest.[1] In patients of severe MS, a substantial increase in left atrial pressure (LAP) occurs and a gradient develops across the MV to accomplish left ventricular (LV) filling. The increased LAP passively elevates pulmonary venous and pulmonary capillary pressures and cause symptoms of pulmonary congestion.[2] When the MVA is reduced to 1 cm^2, a mean gradient of 20 mmHg across the stenosed MV is required to maintain normal cardiac output at rest.[3] The persistently raised LAP results in left atrial (LA) dilatation, atrial fibrillation, pulmonary venous hypertension, reflex pulmonary arteriolar constriction, obliterative changes in pulmonary vascular bed, pulmonary artery hypertension (PAH), right ventricular (RV) hypertrophy, its dilatation, tricuspid valve dysfunction, systemic venous congestion, compromised LV filling, and a state of subnormal cardiac output.[4] The pulmonary arterioles may react with vasoconstriction, intimal hyperplasia, and medial hypertrophy, which further increases PAH.[5]

We discuss role of RV stroke volume, atrio-ventricular compliance, and pulmonary venous compliance in the development of PAH in MS. A close scrutiny of causes of increased LAP is necessary to understand genesis of PAH. The pressure in a relatively thin-walled chamber like left atrium depends on several factors such as its stiffness, mechanical effects of its rhythmic contraction and relaxation, diastolic period, the quantity of blood entering and exiting it, and the net atrio-ventricular compliance which include compliance of LA and LV, and compliance of the pulmonary venous system. For the increase in LAP to be substantial that develops in severe MS, the compliance of LA and pulmonary venous system should be low else the effect of damming of blood will dissipate in left atrium and pulmonary vascular bed and a substantial increase in LAP will not occur. In a study of 20 patients, a subgroup of patients was found who responded to exercise by a significant increase in PA pressure, in that subgroup the net atrio-ventricular compliance was found significantly low. [6] Apparently, in patients of MS a wide spectrum of atrio-ventricular compliance exist - patients with low compliance and patients with normal compliance. Patients with low compliance develop significant PAH, severe increase in LAP and symptoms of MS on exercise or in situations of increased cardiac output, whereas patients with normal compliance remain asymptomatic in situations of increased cardiac output as the increased RV stroke volume is accommodated in the compliant pulmonary venous bed [Figure 1].

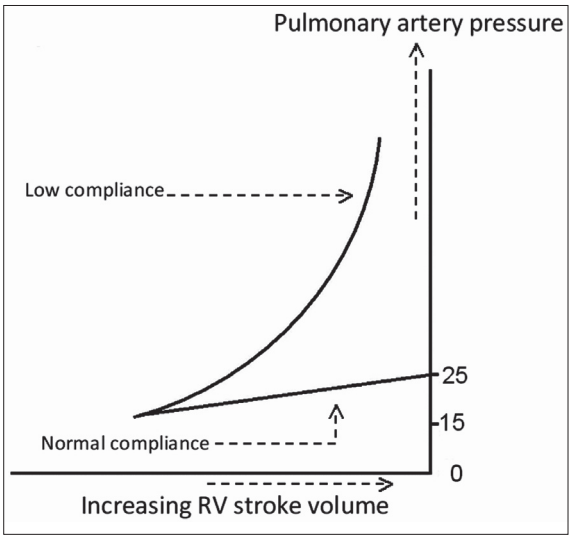

Figure 1: Schematic diagram to show effect of increasing right ventricular (RV) stroke volume on pulmonary artery pressure in presence of low pulmonary venous and/or low atrio-ventricular compliance and normal pulmonary venous and normal atrio-ventricular compliance

Atleast two more factors should be considered in the genesis of raised LAP, the effect of LV relaxation on transmitral flow and the role of RV stroke volume. The flow across MV during early diastole is described passive; however, it is well established that the flow across the MV is augmented by suction effect generated by LV relaxation during early diastole and by LA systole during late diastole.[7] Conceivably, in presence of MS, the suction augmentation of early diastolic flow will be reduced and/or ineffective. Arguably, to generate flow across the stenosed MV, the LA has to be pressurised, it is intuitive that RV stroke volume generated during systole pressurises LA and pulmonary vascular system which raises

the mean pulmonary artery (PA) pressure and LAP, and serve as potential energy to achieve transmitral flow during diastole. The RV stroke volume raises the mean pulmonary vascular system filling pressure and drives the flow across the mitral valve during diastole; the concept is similar to mean systemic filling pressure.[8] However, over a period, reflex protective pulmonary vasoconstriction and pulmonary arteriolar obliteration sets in, which further increases PA pressure and result in remodelling of the RV to overcome raised pulmonary vascular resistance. Apparently, the PAH in MS is benign and mechanical in the beginning and develops as a compensatory mechanism to overcome resistance offered by the stenosed mitral valve; later, it progresses secondary to pulmonary vasoconstriction and pulmonary arteriolar obliteration. However, in a given patient it is not possible to know the contribution of each of these factors in the genesis of PAH. Apparently, the PAH in MS is not only due to raised LAP and raised pulmonary vascular resistance secondary to pulmonary vasoconstriction and obliterative changes in pulmonary vascular bed, but it also depends on contribution of net atrio-ventricular compliance, pulmonary venous compliance, and RV stroke volume; an increased RV stroke volume and low net atrio-ventricular compliance and pulmonary venous compliance results in severe increase in PA pressure and symptoms of pulmonary congestion.

It is intuitive from the above discussion that in patients of MS sustained RV performance and persistently maintained PA pressure is essential for achieving transmitral flow, LV filling, and LV stroke output. The clinical situations that decrease RV performance can result in low cardiac output, whereas situations where RV performance remains unaffected or increases in response to pathophysiological stimulation might result in increased LAP, pulmonary congestion, edema, but sustained cardiac output. Apparently, in patients of MS, RV performance in response to varying clinical demands and basal atrio-ventricular and pulmonary venous compliance decides the clinical features. This could be the mechanism that explains varying clinical presentation of similar degree of tachycardia in patients having similar degree of MS. Apparently, patients who can mount RV performance and increase or maintain PA pressure in response to pathophysiologic challenges maintain cardiac output but are likely to develop pulmonary congestion and edema, whereas those who develop RV decompensation and cannot sustain PA pressure develop low cardiac output and possibility of acute cardio vascular collapse. Arguably, while anesthetising these patients, it is important to sustain RV function else the patient may develop cardiovascular decompensation.

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Quick Response Code:	Website: www.joacp.org
	DOI: 10.4103/0970-9185.94916