CARDIOLOGY Rounds®

As presented in the rounds of the Division of Cardiology, St. Michael's Hospital, University of Toronto

Valvular heart disease in pregnancy

By AFSANEH POURDOWLAT, MD

Management of pregnancy in patients with valvular heart disease (VHD) continues to pose a challenge to clinicians. VHD may have a significant impact on fetal and maternal health during pregnancy, labour, and delivery. Its presence may be recognized for the first time during pregnancy when hemodynamic changes associated with the gravid state cause symptoms in a previously asymptomatic patient or accentuate a preexisting condition. The most common cause of VHD in pregnant women has shifted in recent years from rheumatic to congenital valve disease. However, among many immigrant women, rheumatic heart disease remains the prevalent cause.

Hemodynamic changes in pregnancy

In brief, cardiovascular changes during pregnancy include:

- an increase in cardiac output
- an increase in blood volume that begins as early as the 6th week of pregnancy and continues progressively until it is 150% of its original level
- increases in plasma volume that are out of proportion to red cell volume leading to physiologic anemia
- an increase in cardiac stroke volume from the 6th to the 28th week of pregnancy
- an increase in cardiac output during the 3rd trimester due to an increase in heart rate
- a fall in pulmonary pressure with a decrease in pulmonary vascular resistance
- decrease in venous return because of progressive inferior vena cava (IVC) compression in the 3rd trimester

In addition to all of the above, uterine contractions during labour are associated with increases in blood pressure (BP), heart rate, cardiac output, and oxygen consumption. These changes are summarized in Figure 1.[1]

Signs and symptoms associated with VHD in pregnancy

Symptoms that are commonly used for assessing cardiac disease (eg, fatigue, dyspnea, orthopnea, presyncope, syncope, and pedal edema) are frequently present in a normal pregnancy. Table 1 summarizes the clinical signs and changes specifically related to VHD during pregnancy.

Effects of pregnancy on preexisting VHD

There are some general observations regarding the effects of pregnancy on preexisting VHD.

- Regurgitant lesions are better tolerated than stenotic lesions during pregnancy.
- Patients who are asymptomatic or minimally symptomatic before pregnancy usually tolerate pregnancy well.[2]
- Ideally, symptomatic VHD should be treated before pregnancy.

Editor's note: This March issue, as well as the upcoming April and May issues of *Cardiology Rounds,* are late in reaching you. This delay was caused by the SARS emergency measures imposed in most of the hospitals in Toronto during March and April. During that time, almost all academic rounds were cancelled. We apologize for the delay in the series.

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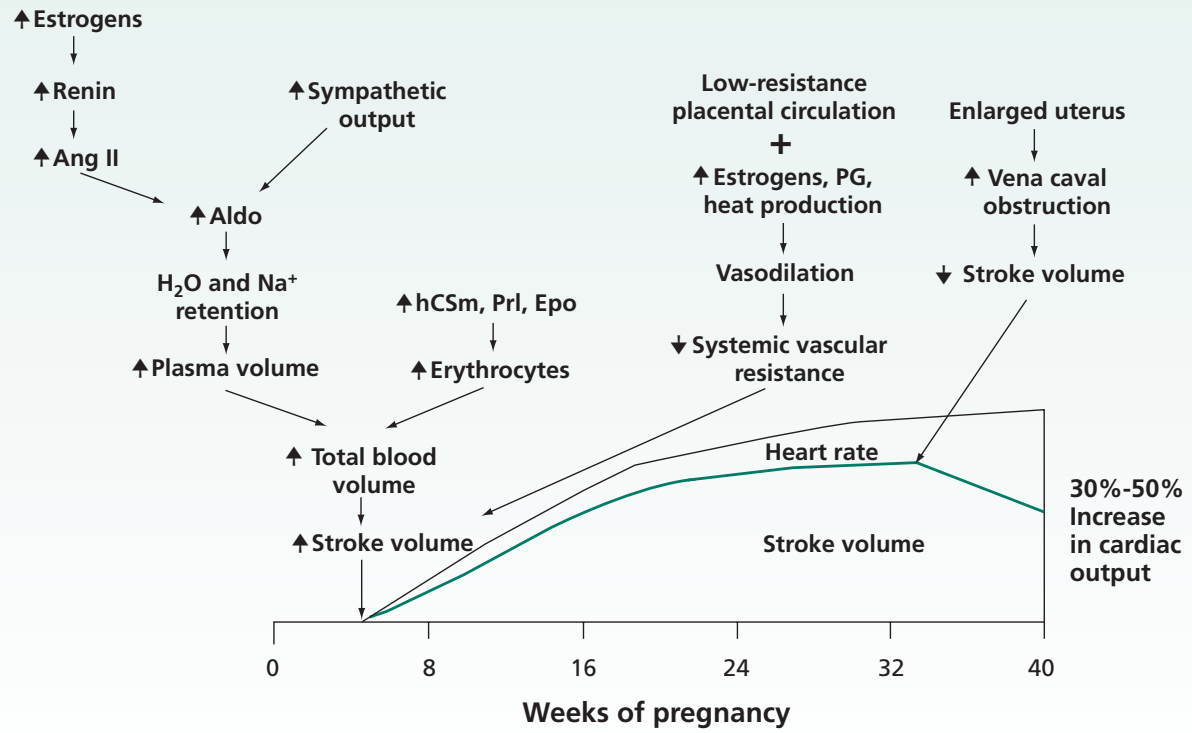
Figure 1: Hemodynamic changes during pregnancy[1]



hCSm = human choriontrophic somatomammotropin; Prl = prolactin; Epo = erythropoietin; PG = prostaglandin

A study performed in California and published in the *Journal of the American College of Cardiology*[3] followed 66 pregnancies in 64 women with VHD and matched them in age, ethnicity, obstetric/ medical history etc. with control individuals, demonstrated the following results. There was a significantly higher incidence of congestive heart failure (CHF), arrhythmias, and hospitalizations in women with VHD. Patients with VHD needed cardiac medications; in order of frequency, these included diuretics, beta-blockers, calcium channel blockers (CCBs), digoxin, heparin, and hydralazine. The mode of delivery was vaginal in 92% of women with VHD compared to 87% of controls. Post-delivery pulmonary edema occurred in 3 patients with moderate to severe mitral stenosis (MS) and in 2 patients with severe aortic stenosis (AS). Fetal outcome was associated with a marked increase in the rate of intrauterine growth retardation (IUGR) and lower birth weight. Although there was an increase in the incidence of pre-term birth, it did not reach clinical significance.

Figures 2 and 3 show the changes in NYHA class between the first visit and the follow-up visit during pregnancy in women with mitral valve disease and aortic valve disease.

Since mitral stenosis and aortic stenosis cause complications and are frequently more prominent in pregnancy, these two conditions are reviewed below as subgroups of VHD in pregnancy.

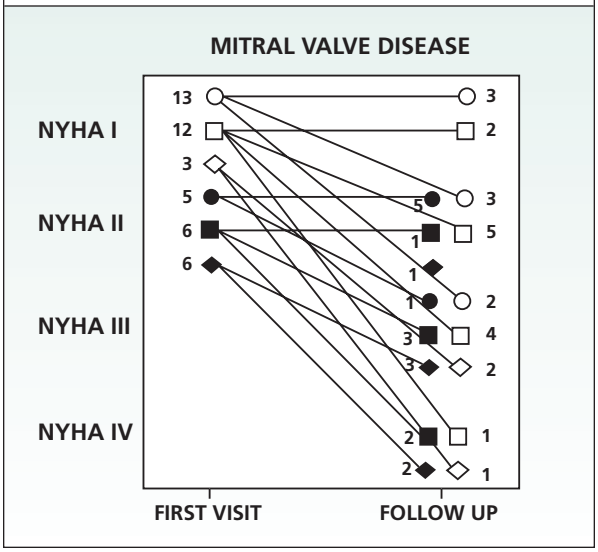
Mitral stenosis

A survey of deliveries from 1970 through 1983 in the USA demonstrated a rate of maternal heart disease in 1.3% of deliveries, of which 60% were rheumatic in nature. Mitral stenosis, which may have a natural history of 20-25 asymptomatic years, is by far the most common rheumatic valvular disease in pregnancy.[1]

The pressure gradient across the narrowed orifice between the left atrium (LA) and the left ventricle (LV) is a function of both the cross-sectional area of the valve and the square of the flow velocity through it. A 50% increase in cardiac output during pregnancy may cause a marked increase in the pressure gradient across the valve. Patients with moderate to severe MS (mitral valve area [MVA] < 1.5 cm) are at increased risk of dramatic and, at times, fatal pulmonary edema. Combined with the rapid heart rate during a physiological response, cardiovascular hemodynamics are significantly affected by a decreased diastolic filling time, and the increased LA pressure. There is a documented increased incidence of arrhythmias in normal pregnancy that, together with increased LA pressure, can cause a significant increase in the rate of atrial arrhythmias, a loss of atrial contractivity, and a rapid ventricular response.[4]

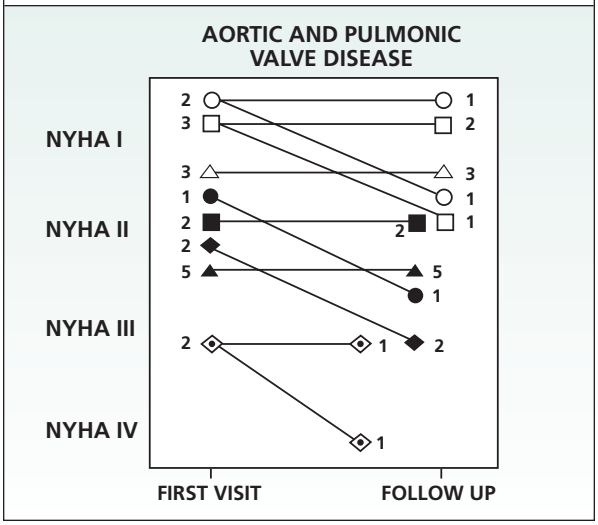
Rarely, patients with a severe MS may have critically elevated pulmonary vascular resistance and pulmonary hypertension, resulting in right ventriclar (RV) failure and

Figure 2: Change in NYHA functional class between the first and follow-up visit during pregnancy, in patients with predominant mitral valve disease[3]



Circles, mild mitral stenosis (MS); **squares,** moderate MS; **diamonds,** severe MS. **Open symbols,** NYHA functional I on presentation; **closed symbols,** NYHA class II on presentation.

Figure 3: Change in NYHA functional class between the first and follow-up visit during pregnancy, in patients with predominant aortic and pulmonic valve disease[3]



Circles, mild aortic stenosis (AS); **squares,** moderate AS; **diamonds,** severe AS; **triangles,** pulmonic stenosis. **Open symbols,** NYHA functional class I on presentation; **closed symbols,** NYHA class II on presentation; **dotted diamonds,** NYHA functional class III on presentation.

low cardiac output. In these cases, termination of the pregnancy should be considered.

Clinical evaluation

In mitral stenosis, symptoms are typically related to left-sided failure. Usual clinical findings are tachycardia, elevated jugular venous pressure (JVP), chest rales, RV lift with a palpable P2, evidence of ascites, peripheral edema, and auscultatory findings (Table 1).

Diagnostic tests

ECG can show signs of left atrial enlargement (LAE), right axis deviation (RAD), and occasionally, RV hypertrophy.

Chest x-ray shows a small heart, a large left atrium, and pulmonary congestion; however, an x-ray is not recommended as a part of routine work-up unless it is clinically indicated to exclude pneumonia or for the evaluation of hemoptysis.

Echocardiography is the major diagnostic tool; however, in order to prevent errors, the recommendation is to rely on transmitral gradient or to employ the continuity equation method rather than pressure half-time to assess severity.[5]

Management

Management of the pregnant woman with mitral stenosis includes:

- limitation of physical activity to prevent tachycardia
- judicious restriction of salt intake
- cautious use of diuretics
- slowing the heart rate with beta-blockers (digoxin is not a good choice since sympathetic stimuli such as exercise or emotional stress can overwhelm the weak vagotonic effects of digoxin)
- anticoagulation therapy in women with atrial fibrillation
- continuation of beta-hemolytic streptococcus antibiotic prophylaxis in rheumatic MS

General recommendations for bacterial endocarditis prophylaxis

Recommendations for bacterial endocarditis prophylaxis in pregnant women with VHD, according to AHA/ACC guidelines are summarized in Table 2.[6]

When symptoms persist despite medical management, interventional therapy may be necessary. Early studies demonstated that open commissurotomy, valvular repair, or replacement are associated with a fetal mortality rate between 20% to 33% because of decreased placenta perfusion and fetal hypoxia, with maternal mortality as high as 5%.[7]

The procedure of choice to improve hemodynamics in symptomatic MS is percutaneous balloon mitral valvuloplasty (PBMV).[8-11] Complications of PBMV include: cardiac tamponade, maternal arrhythmia associated with fetal distress, initiation of uterine contractions, and mitral insufficiency.[9,11,12] To decrease the amount of radiation exposure, the abdomen and pelvis should be shielded or the procedure could be performed with transesophageal echocardiography (TEE) guidance.[10]

A study published by de Souza et al compared PBMV results to open mitral valve commissurotomy (OMVC) from 1985 to 1995 in 45 pregnant patients with NYHA class III or IV, despite hospital admission and medical treatment.[11] Of these patients, 21 received PBMV and 24 underwent open heart surgery. Of the 24 patients who underwent open heart surgery, 8 who needed mitral valve replacement

Table 1. Effects of pregnancy on the findings associated with valvular lesions[1]

	Heart Sounds	Murmur	Other	Doppler echocardiography
Aortic stenosis (AS)	Diminished or single S2 — unchanged	Increased in intensity and duration	Systolic ejection click unchanged	Increase in Doppler gradient; AVA unchanged
Aortic insufficiency (AI)	Diminished S2 — unchanged	Decreased or unchanged	Wide pulse pressure increased or unchanged	LV dimensions may increase secondary to pregnancy not AI
Mitral stenosis (MS)	Loud S1 — increased; P2 — increased	Increased	S2 — OS interval — decreased or unchanged	Increase in Doppler gradient, decrease in pressure half-time and increase in calculated MVA
Mitral regurgitation (MR)	Diminished S1 — unchanged	Decreased or unchanged	S3 unchanged	LV dimensions may increase secondary to pregnancy not MR
Pulmonic stenosis (PS)	Diminished P2 — unchanged	Increase in intensity and duration	Systolic ejection click unchanged	Increase in Doppler gradient
Pulmonic insufficiency (PI)	Diminished P2 — unchanged	Decreased or unchanged	N/A	RV dimensions may increase secondary to pregnancy not PI
Tricuspid stenosis (TS)	N/A	Increased	N/A	N/A
Tricuspid regurgitation (TR)	N/A	Decreased or unchanged	N/A	RV dimensions may increase secondary to pregnancy not TR

were excluded from the study. PBMV was successful in 95% of patients. A complication of PBMV – new mitral insufficiency – developed in 4 patients and 1 patient advanced from a mild to a moderate degree of mitral insufficiency. There was 1 maternal death in the surgical group, as well as 6 fetal and 2 neonatal deaths. In the PBMV patients, one neonatal death occurred in a premature child with an esophageal malformation. Overall, the combined difference between fetal and neonatal mortality in the two groups was significant (p=0.025).

With mitral stenosis, the recommended mode of delivery is vaginal unless there is an obstetric indication for cesarean section. Prior to delivery, a careful fluid balance should be maintained, and during labour, the patient should be in a lateral recumbent position with the administration of oxygen.

Delivery and labour should proceed under epidural anesthesia. In cases of moderate to severe MS, hemodynamic monitoring to maintain LV filling pressure at approximately 14 mm Hg is recommended.

Aortic stenosis

This condition is less common in pregnancy and is primarily caused by congenital abnormalities of the aortic valve. The classic triad of symptoms related to aortic stenosis and its severity – dyspnea, chest pain, and syncope – are not very helpful since dyspnea and presyncope are common symptoms even in normal pregnancies. Symptoms of aortic stenosis usually appear during the 2nd and 3rd trimesters. The incidence of pulmonary edema is much lower than that in mitral stenosis.

The best diagnostic tool is echocardiography, which helps with estimations of the aortic valve area (AVA) and the association of LV hypertrophy (LVH) and aortic insufficiency. Patients with an ejection fraction (EF) <55% should be considered at risk for the development of significant heart failure.

Management and follow-up recommendations[1,12,13]

If the initial transvalvular gradient is > 50 mm Hg or the patient is suffering from symptoms during the first visit, a repeated echocardiography is needed in the second trimester. Since outflow obstruction with congenital aortal valve abnormalities have a pattern of inheritance, there is a 15% risk of fetal abnormalities in these patients. A fetal echocardiography is indicated as part of the follow-up work-up. Patients with a moderate to severe degree of stenosis are prone to have cardiac arrhythmias. To exclude the possibility of malignant asymptomatic arrhythmias, ambulatory electrocardiographic monitoring should be considered.

Cardiac catheterization is performed only in conjunction with percutaneous balloon valvuloplasty or when evaluation of the patient through symptoms, physical examination, and noninvasive data is inconclusive.

Table 2: Recommendations of the AHA/ACC task force on antibiotic prophylaxis for selected valve lesions[6]

Valve lesion by endocaritis risk	Vaginal delivery	Cesarean section
High risk for endocarditis • Prosthetic cardiac valves (includes bioprosthetic and homograft valves) • Prior bacterial endocarditis • Complex cyanotic congenital heart disease* • Surgically constructed systemic pulmonary shunts or conduits	Optional	Not recommended
Moderate risk for endocarditis • Most other congenital cardiac malformations • Acquired valvar dysfunction (eg, rheumatic heart disease) • Hypertropic cardiomyopathy • Mitral valve prolapse with leaflet thickening and/or regurgitation	Not recommended	Not recommended
Negligible risk for endocarditis • Mitral valve prolapse without valvar regurgitation • Physiologic, functional, or innocent murmurs • Previous rheumatic fever without valvar dysfunction	Not recommended	Not recommended

* Includes single ventricle states, transposition of the great arteries, tetralogy of Fallot.
AHA/ACC = American Heart Association/American College of Cardiology

Management of these patients includes limiting physical activity. Patients with moderate stenosis (a peak gradient of 50 to 75 mm Hg or AVA of 1.0 to 1.5 cm^3) usually respond to medical management; however, severe aortic stenosis with peak gradient >75 mm Hg or AVA <1.0 should be considered for interventional management. The procedure of choice for patients with aortic stenosis, as in mitral stenosis, is percutaneous balloon valvuloplasty.

Conclusion

VHD in pregnancy is a rare, but challenging problem. Patients with VHD should be followed carefully and hospitalized for their delivery or other medical conditions in a tertiary centre where a cardiology consultation service is available. Patients who are asymptomatic prior to pregnancy usually tolerate pregnancy well. Also, regurgitant lesions are better tolerated than stenotic lesions. Patients with VHD who are planning to become pregnant should be carefully evaluated before becoming pregnant. Clinicians should keep in mind that VHD is a chronic progressive problem and that patients get used to and adapt to symptoms. If a patient has moderate symptoms before pregnancy, clinicians should be aware that these symptoms will significantly worsen during the pregnancy. Therefore, it is reasonable to think about balloon valvuloplasty ahead of time. If a patient presents during pregnancy with significant symptoms that are not responsive to medical treatment, TEE-guided percutaneous balloon valvuloplasty with abdominopelvic shielding is the procedure of choice. This condition does not warrant the consideration of changing the mode of delivery; however, many of these patients will need careful monitoring during labour and delivery.

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Abstract of Interest

Outcome of pregnancy in Women with Fallot's Tetralogy

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BACKGROUND: Pregnancy (pg) outcome in patients (pt) with Tetralogy of Fallot (TOF) is incompletely defined.

METHODS: The records of female pt >18 years, with TOF were reviewed for clinical and hemodynamic data at 1st assessment, last clinic visit, and before and after completed pg.

RESULTS: Seventy-two TOF pt responded to our questionnaire. Forty-three pt (mean age, 26 ± 5 years) had 112 pg, (range, 1-5 per pt); 82 were sucessful. Seventeen pt (41%) had a prior shunt, and all had eventual surgical repair. Eight pg (in 3 pt) occurred before repair. Sixteen pt had reoperation (6 before pg). At 1st assessment >18 years, RV pressure (RVSP), available in 37 pt, was 20-35 mm Hg in 13, 36-60 mm Hg in 15, and >60 mm Hg in 9; of those with raised RVSP, 6 had PA hypertension. Three pt had moderate RV systolic dysfunction and 13 had moderate or severe RV dilation due to PV regurgitation (PR). Sixteen pt had 29 (26%) spontaneous 1st trimester miscarriages, and 1 term stillbirth. Mean overall birth weight was 3.2 kg (range 2.1-4.2). Higher birth weight correlated with absence of PA abnormalities (r=0.425), lower RVSP (r=0.358), and older maternal age (r= 0.303). Of the 6 pt with PA hypertension, 2 had cardiovascular events and 1 had 4 first trimester miscarriages. Six of the 43 pt had cardiovascular complications: supraventricular tachycardia in 2 pt (1 with PA pressure 122 mm Hg, and the other with severe PR, severe RV dilation, moderate dysfunction); congestive heart failure in 2 pt (due to cardiomyopathy in 1 and pre-eclampsia in 1); pulmonary embolism in 1 pt with PA hypertension; and progressive RV dilation in 1 pt with severe antepartum PR. There were no peripartum deaths. Five (7%) infants had congenital anomalies; VSD in 1, hypoplastic left heart in 1, mitral valve prolapse in 1, one with clubbed feet, strabismus, cleft lip and palate, and 1 infant with pyloric stenosis.

CONCLUSIONS: Pt with TOF have increased fetal loss, and their offspring have more congenital anomalies than the general population. The absence of PA abnormalities, older maternal age and lower RVSP, is associated with higher infant birth weight. Adverse maternal events are rare and may be associated with pre-existance PA hypertension or RV volume overload.

J Am Coll Cardiology 2003;41(6) suppl A: 490 A. Abstract: 1192-159.

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This publication is made possible by an educational grant from

Novartis Pharmaceuticals Canada Inc.