Jurnalul Român de Anestezie Terapie intensivă 2009 Vol.16 Nr.1, 55-62

Non-cardiac surgery and obstetric analgesia in the patient with valvular disease: anesthetic considerations

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Summary

As patients are reaching higher ages now but also due to medical progress, progressively more patients present nowadays with valvular lesions for non-cardiac surgical procedures. Some patients may benefit from a regional anesthetic technique while others will be best managed with stress-free general anesthesia. The aim should not be to normalize the hemodynamics but to keep the patient as stable as possible as compared to the preoperative condition. Although many patients will have combined defects, this overview will focus on the most common individual defects, their etiology, symptoms, physiopathology and anesthetic management. Special attention will be paid to the obstetric population which is getting older as well. This is a challenge for obstetricians and anesthesiologists because of the physiological changes during and after pregnancy and the hemodynamic impact of labor, oxytocics, tocolytics, pain and anesthesia.

Key words: cardiac, valvular, obstetric, anesthesia, regional

There is not much data on perioperative risk analysis in patients with valvular disease with or without pulmonary hypertension. Ventricular dysfunction, arrhythmia, pulmonary hypertension and ischemia will all increase the cardiac risk.

Even with no major experience in cardiac surgery and cardiopulmonary bypass, care providers should understand the valvular diseases involved the effect of the disease upon anesthesia and vice-versa. Some physiological changes and perturbations such as pregnancy and aging may significantly influence the outcome, severity and course of the disease. Especially pregnancy seems to signify a considerable challenge for the anesthesiologist. In the first place there are the physiological changes during pregnancy, pain, delivery and the first days after delivery. The additional effects of oxytocics or tocolytics and the different types of anesthesia should not be underestimated. Based on the extent of literature on valvular lesions and pregnancy the impression may be raised that if pregnancy, delivery and anesthesia for this may pass uneventfully, any other patient can be managed. This does surely not account for the elderly.

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Preoperative care and planning

This consists of careful anamnesis, clinical examination, technical investigation such as ECG, chest X-ray, echocardiography, catheterisation, advice of the cardiologist, premedication, an anaesthetic conduction plan including monitoring techniques and postoperative care. The anaesthetist also needs to anticipate the effects of blood loss, stress, pneumoperitoneum, laryngoscopy, positional changes, tourniquets, cross clamping and release of them on large vessels. With respect to monitoring, the threshold for using direct arterial blood pressure monitoring should be low. Central venous pressure and pulmonary artery catheter can be useful to monitor fluid therapy but may induce rhythm problems, sometime difficult to treat in hypertrophic ventricles. Transesophageal echocardiography, when available, may also guide volume replacement and monitor ventricular function.

Basic principles

Stenotic lesions will cause pressure overload while leaking valves will rather cause volume overload. All valvular lesions may cause perioperative congestive heart failure. *Cardiac output* depends on four major determinants: preload, afterload, contractility and heart rate. In case of a valvular lesion the *transvalvular flow* will depend on the area of the defect causing obstruction or regurgitation, the duration of either the systole or diastole and the *transvalvular gradient* according to the following equation of Gorklin. The higher the value of the gradient or valvular area, the higher the transvalvular flow. For a given valvular area a doubling of the flow rate will increase the gradient four times.

$$\text{Gradient} = (\text{flow rate} / k \times \text{cm}^2)^2$$

In case of *operated defects* patients may be cured for 100% although some physiological alterations may remain such as dysrhythmia, heart failure, pulmonary hypertension (PHT) and ventricular hypertrophy. This wall thickness will determine the *wall tension* by the following equation:

$$\text{Wall tension} = \frac{\text{Pressure} \times \text{radius}}{2 \times \text{wall thickness}}$$

When the pressure within the ventricle will increase, then the duplication of muscle fibres will compensate this to maintain the wall tension. A slight decrease in radius may also partly decrease wall tension.

The *valvular* area in stenotic lesions is fixed whereas in valves causing regurgitation this may be dynamic and variable depending on pre- and afterload.

The *therapeutic goal* of both lesions is also quite opposite as in stenotic valves the purpose is to increase the flow while with regurgitating this should be reduced.

Patients may have several *combined defects*. A stenotic valve, such as the mitral valve may also be insufficient while it may cause regurgitation of another adjacent valve such as the combination of aortic stenosis and mitral valve regurgitation (bot having opposite hemodynamic goals) or mitral stenosis and tricuspid regurgitation. Treatment should be directed by the most important symptoms or predominant lesion. Dyspnea, pulmonary edema may indicate mitral valve disease symptoms of pulmonary congestion while angina and syncope may rather reflect aortic stenosis. However, others claim that if aortic stenosis is present, patients should receive treatment and management mainly focusing on this lesion of high priority considered as a *NYHA class IV* disease (symptoms at rest). NYHA classification mainly depends on LV dysfunction. Class IV disease is often considered to require repair before any other type of surgery.

Aortic stenosis (AS)

Etiology. Isolated aortic stenosis is mostly an acquired disease caused by senile degeneration and/or calcification of a previously normal valve (except for a congenital bicuspid valve). Rheumatic heart disease is also possible, although isolated AS caused by rheumatic disease is not common (in this case also mitral stenosis is possible). It seems to be more frequent in males, smokers, patients with hypertension, diabetes or hypercholesterolemia. Bicuspid valves, being present in up to 50% of the congenital forms, may equally cause stenosis.

Symptoms. The most suggestive symptoms are angina (30% often without evidence of coronary disease), syncope, congestive heart failure (dyspnea) and sudden death which may occur in 15-20% of cases, when heart massage may be inefficient. The latter is more common with valve areas less than 1cm^2 (nl area 2.5-3.5 cm^2). An area between 1 and 1.5 cm^2 is considered as moderate aortic stenosis. Symptoms may occur late in the course of disease and take 50-60 years if the lesion is acquired but it is commonly accepted that mortality will be 50% 5 years after the onset of angina, even sooner in case of syncope or congestive heart failure. Congenital lesions may take only 20-30 years to become obvious, sometimes on the occasion of pregnancy and delivery.

What happens? The pressure within the left ventricle will increase (pressure overload) which will be compensated by a concentric hypertrophy. This only partially solves the problem as it decreases diastolic compliance while higher end diastolic pressures are required to fill the stiff LV. Oxygen consumption will increase, oxygen delivery will decrease due to less diastolic relaxation and lower cardiac output. In addition the hypertrophic muscles will not receive as much collateral circulation, with less vasodilatation reserve than in normal tissue, thus causing subendocardial ischemia. Due to decreased diastolic relaxation of the left ventricle, the contraction of the left atrium will become more important and will contribute up to 40% of the left ventricle filling volume. It is therefore important to preserve the sinus rhythm with medication including statins which have shown to have a beneficial effect on heart rhythm and coronary perfusion. Sooner or later heart failure may develop due to progressive ischemia and fibrosis enhancing left ventricular stiffness. End stage AS will fail to sustain the cardiac output because of the excessive afterload rather than contractility failure.

Technical. Echocardiography and catheterisation will enable to determine the gradient, the valvular area, the degree of stenosis (and eventual presence of regurgitation), left ventricular end diastolic pressure/volume (LVEDP, LVEDV) and left ventricular ejection fraction (LVEF, < 50% enhances the mortality risk). A transvalvular flow velocity > 4.5 m/sec is also a factor of high risk. The EDV should be elevated while a normal value signifies relative hypovolemia. When the stenotic lesion becomes more severe, the peak values for gradient and flow velocity will occur later in the systolic phase. Catheterisation will also highlight the peak-to-peak pressure gradient ($PG = P_{LV}-P_{ao}$) and the condition of the coronary blood supply, as up to half of the patients, especially when older than 50, may have coronary heart disease. A pressure gradient of > 50 mmHg is considered as critical AS. However, this is true for patients with normal cardiac output, as patients with LV failure may have 'misleading' lower gradients.

To improve the condition of the patient, balloon valvuloplasty is possible in case of urgent surgery. This may decrease the gradient from 60 to 30 mmHg but will cause a mortality rate of 3-7% and a 50% re-stenosis within 6 months. NYHA class 4 patients will require replacement before any other elective surgery. Much advance may be offered by core valve placement in the formerly inoperable elderly patient (aging is an important risk factor).

Anesthetic management. This should aim at maintaining a sinus rhythm, avoiding tachycardia (reducing the systolic duration and increasing oxygen requirements) and maintaining an adequate circulatory volume and filling pressures (preload, 'normovolemic expansion'). The systemic vascular resistance (afterload) should be maintained, according to others, even elevated, to keep the aortic diastolic pressure high enough. In case of chest pain the patient should receive oxygen rather than nitrates which will induce arterial and venous dilatation. Phenylephrine may be a better choice to increase coronary perfusion in case the decrease in diastolic aortic pressure would be the cause of ischemia, because il will also decrease the heart rate. Although heart rate increase should be avoided, these patients will not support extreme bradycardia either. A good premedication will decrease stress and induce some beneficial bradycardia (60-70 bpm). It should not be forgotten that due to lower cardiac output in stenotic lesions benzodiazepines should be administered cautiously (e.g. midazolam 0.5 mg increments) while enough time should elapse to wait for the full effect of the previous dose. Contractility is already increased so that this may not be improved by drug therapy. Vasodilating and myocardial depressant induction agents should be used cautiously. Muscular blocking agents causing tachycardia (pancuronium) or at risk for histamine release should be avoided. Other anesthetics which should be avoided are ketamine, atropine and isoflurane because of the increase in heart rate. Midazolam and etomidate may be a better choice for induction than propofol or thiopenthal. Beta-blockers may prolong filling time but may decrease contractility.

Obstetric analgesia and anesthesia. There is a growing trend to allow vaginal delivery (but no pushing allowed) rather than a routine surgical intervention. Low dose epidural started as early as possible will offer benefits in practically all conditions as none of them will well tolerate the hemodynamic changes related to stress, pain, bearing down or contractions. Care should only be taken in cases of anticoagulation. To preserve the physiological venous return, left lateral tilt should be applied at all times, which is better than the administration of additional fluids or vasopressor drugs. The use of opioids alone, by CSE or CSA, is to be considered. Many successful cases of regional analgesia for aortic stenosis have been reported up to date. For C-section, general anesthesia is used 6 times more frequently than in the non-cardiac population. Agents such as etomidate and succinylcholine are recommended although hemodynamic consequences during intubation are probably more dangerous than the risk of aspiration or moderate hypotension. If LRA is the option, it is still commonly thought that an epidural anesthesia may be a better idea than a spinal one, because of its higher predictability and less hemodynamic perturbations. It may be wise to maintain the epidural for the first 48 hrs when the risk of fluid overload is the highest. Furosemide may be practical sometimes. Several cases have been reported with carefully titrated techniques such as epidural, CSE, CSA or EVE while single dose spinal is commonly accepted as the technique to avoid.

Mitral stenosis (MS)

Etiology. The cause will be mostly the rheumatic heart disease and it may also induce a lesion of the myocardial fibres. However, only 50% will have a history of rheumatic fever. Other causes may consist of rheumatoid arthritis, congenital MS, lupus erythematosus or carcinoid syndrome. It will be isolated in only 25% of cases, while in 40% of the cases there will be insufficiency as well. In females the defect is four times more frequent.

Symptoms. These may last up to 20 years to develop. Due to pulmonary congestion dyspnea may be the most predominant symptom. Atrial fibrillation may also be a predominant symptom.

What happens? Hypertension in the left atrium (> 25 mmHg necessary to maintain normal cardiac output

in severe MS) will cause hypertrophy and distention, keeping atrial pressure constant, on his turn inducing atrial fibrillation, further reducing ventricular filling. Atrial fibrillation with fast response, tachycardia but also acute fluid load, head down position, exercise, pregnancy/labor, anemia, hypervolemia, hyperthyroidism and infection may increase the transvalvular flow and hence the gradient (x4 for a doubling of the flow) and increase the risk of pulmonary edema, PHT, right heart failure by pressure overload (the thin-walled right ventricle is not used to manage afterload increase) and tricuspid regurgitation. Loss of RV compliance and excessive distention will result in leftward displacement of the interventricular septum. Overload of the right ventricle and pulmonary circulation will induce a redistribution of the flow from the basal to the apical compartments thus enhancing the ventilation/perfusion mismatch. RV failure is a poor prognostic sign.

Technical. Echocardiography may show left atrium enlargement and overload, mitral fibrosis and calcification, a high gradient between left atrium and ventricle (< 5 mmHg, mild; 5-12 mmHg, moderate; > 12 mmHg, severe), left ventricular dysfunction and underfilling (underloading) and decreased wall thickness and contractility (disuse atrophy). The normal mitral valve area is between 4 and 6 cm^2, mild stenosis is considered with areas < 2 cm^2 and severe if < 1 cm^2.

A *pulmonary artery catheter* may be indicated in patients with congestive heart failure to get a better idea of filling volumes and pressure. However cases have been described with traumatic pulmonary artery rupture especially since they tend to be introduced too far in case of PHT. The wedge pressure trace may not be attainable. It is recommended not to search for the most distal catheter tip position. They are mainly recommended in cases of dyspnea and respiratory distress, severe stenosis and when large fluid shifts are expected. An additional risk of placing central catheters may be the sudden development of arrhythmia. It is still a subject of debate which patients should receive placement before anesthesia.

Anesthetic management. This should aim at avoiding tachycardia (reducing diastolic time more than systolic) in the first place, maintaining sinus rhythm (although a sinus rhythm will contribute little here in ventricular filling), afterload (surely avoiding decreases), adequate volume and fairly high filling pressures (with careful volume expansion) while avoiding PHT. Balloon valvuloplasty and cardioversion before surgery may be beneficial. Drug therapy may consist of digoxin, diuretics, beta-blocking agents (will further reduce RV contractility but most probably the beneficial effect on heart rate is more important), anticoagulants, dobutamine and epinephrine (but inducing tachycardia). Hypotension should be treated with digoxin (heart rate slowing as well) or alpha-agonists although the latter may also increase pulmonary vascular resistance.

If secondary *pulmonary hypertension* (PA systolic pressure > 30 mmHg) and RVF exist anesthetic management will be quite challenging. Any valvular defect or LV disease may cause secondary PHT. An increase in CVP may be caused by pulmonic and tricuspid regurgitation. If both CVP and PAP are increased this may indicate RVF. The use of alpha-agonists may further increase PHT while vasodilators (nitroglycerin thought useful) will also causes systemic hypotension. The best treatment may consist of inotropics (to support the failing RV) and substances which reduce PVR and PA pressure. Mostly used are dobutamine (improve RV hemodynamics and reduce PA pressure), epinephrine, phosphodiesterase-III inhibitors (milrinone, amnirone, reducing PVR and enhancing RV inotropism) and phosphodiesterase-V inhibitors (sildenafil). A pulmonary vasodilator (NO, or prostacyclin also affecting platelet aggregation and inflammation) may be combined with a systemic vasoconstrictor. Carbondioxide retention, hypoxia (oxygen supplementation), hypothermia, stress and pain may further increase pulmonary vascular resistance (PVR) or better vascular impedance, a dynamic parameter of the flow/pressure relationship, including viscosity, compliance, reflected waves and pulsatile flow.

Obstetric analgesia and anesthesia. In fact the same principles apply as for aortic stenosis unless congestive failure is more likely to occur with incautious volume administration. Due to the demand for regional anesthesia the coagulation status will receive close attention. Special care should be given during the first hours after delivery and the offset of anesthesia as patients should be able to manage fluid excess by the amounts given earlier and the autotransfusion following uterine contraction. By effective analgesia such as a low dose (CS) epidural, pain and stress, further increasing PVR may be avoided.

In case of PHT, severe bradycardia (90-120 bpm optimal?), myocardial depression, hypervolemia, after- and preload (venous return) reduction are not desirable either. A C-section will cause a poorer outcome. General anesthesia is the rule if PHT is present. Ketamine, nitrous oxide, prostaglandin E2 and F2a, ergometrine and oxytocin should be withheld.

Pulmonic stenosis (PS)

Etiology. With only 10% of congenital heart disease (CHD), PS may result from rheumatic disease or carcinoid. 90% will be pure valvular while also sub- and supravalvular stenosis is possible. In Fallot's

tetralogy 90% of the patients may have a bicuspid stenotic pulmonic valve.

Symptoms. Mild to moderate forms may be symptomless but in severe stenosis, fatigue and dyspnea during exercise will appear. Similar to AS, angina and syncope are equally possible, indicating insufficient stroke volume. Peripheral edema is suggestive for RHF. Exercise, especially in pregnant patients may provoke symptoms.

What happens? As the normally thinwalled RV with low intraventricular pressure will show compensatory thickening, subendocardial ischemia may follow. With co-existing ASD, VSD or oval foramen cyanosis may occur due to right to left shunting. The tricuspid valve may regurgitate in severe forms. Balloon valvuloplasty may be done when the gradient exceeds 50 mmHg.

Anesthesia. Anesthetic procedures seem to be better tolerated than with any other stenotic disease. Despite clear goals for PS, recommendations may be similar as for other stenotic defects. As long as there is no RVF enough flow may reach the left part. In severe cases with insufficient stroke volume angina may occur similar as with AS and MS. Hypotension and tachycardia should be avoided. Endocarditis is at the highest risk in CHD such as Fallot's tetralogy. Unrepaired (mostly) PS offers a low risk of endocarditis (see below). A pulmonary artery catheter may be difficult to pass. A simple CVP line may be sufficient to follow RV filling and pressures.

Aortic insufficiency / incompetence (AI)

Etiology. Most important primary causes are rheumatic heart disease and infective endocarditis. Their evolution is chronic allowing the heart to slowly adapt. Endocarditis and secondary AI following trauma and dissection of the aorta, connective tissue disease (syphilis, Marfan's disease) may cause acute symptoms. Aging and chronic hypertension may also contribute to it development. A congenital form (bicuspid valve) is rather exceptional.

Symptoms. The most important, early and less specific symptoms are fatigue and dyspnea. With more severe regurgitation, acute pulmonary edema and congestive heart failure may develop, due to increasing left ventricular end-systolic diameter (> 55 mm) and ejection fraction becoming less than 50%. Acute AI is poorly tolerated and patients rapidly develop heart failure, as the mitral valve will be unable to contain the regurgitated volume.

What happens? The extent of regurgitation will depend on the valve diameter, the duration of the diastole and the diastolic pressure gradient between aorta and left ventricle. A volume of regurgitating blood < 3L is considered mild, more than 6L is severe AI. As long as the protective mitral valve is OK, the problem of increased preload is limited to the left ventricle only. Higher than normal left atrial pressure and volume may follow the premature mitral valve closure. As with aortic valve stenosis, also this lesion may cause mitral valve regurgitation in later stages of the disease due to ventricular dilatation and overload. Similar to aortic stenosis there will be a ventricular hypertrophic response but in this case it will be eccentric due to serial, rather than parallel multiplication of muscle fibres. This will increase the left ventricular compliance. Cardiac ischemia and loss of compliance and contractility in a severely hypertrophic LV will occur only late in the disease. When afterload reduction may not be further compensated by higher ventricular output angina during exercise may appear.

Technical. On the arterial pressure recording the dicrotic notch may be lacking due to the lower diastolic arterial pressure. The P-V loops for valvular insufficiency are shifted to the right and more with chronic defects than with acute insults. There will be no isovolumetric dilatation (early volume loading of the LV) and also the isovolumetric contraction will be very short. Stroke volume can be twice as large. (Colour Flow Doppler) Echocardiography may allow estimation the size and volume of regurgitation, the cause of MVR and the dimensions of the LA.

Anesthetic management. A reduction of the afterload is only effective if also preload will be carefully optimised. A decreased afterload may enable forward ventricular ejection. Due to this stroke, volume and EF may be sufficient during the early stages of the disease. The decrease in diastolic aortic pressure will be better tolerated and will cause less ischemia than in aortic stenosis. Tachycardia i.e. heart rate of 90 bpm or even more, reducing the ventricular filling time and hence the time for regurgitation, is recommended although the total volume of regurgitation per minute will remain the same, but its most beneficial effect will be a decrease in ventricular expansion and increase in diastolic arterial pressure improving coronary perfusion. Contractility may be increased with either dobutamine, amrinone or milrinone (all decreasing afterload and pulmonary artery pressure as well). Levosimendan may have inotropic and lusitropic effects but will also decrease afterload and the risk of atrial fibrillation. With respect to general anesthesia the choice of induction agents (mostly vasodilating) may be freer than for stenotic lesions. Volatile anesthetics may be beneficial because they have less effect on contractility (except halothane) while decreasing afterload. Regional anesthesia may be beneficial because it reduces afterload but care

should be taken to maintain the preload and hence the stroke volume. An upper sensory level should be restricted to an area below T4 to avoid the risk of bradycardia.

Obstetric analgesia and anesthesia. Pregnancy itself may have beneficial effects on the regurgitating valve because of a rather fast heart rate (reducing regurgitation time) and the decrease of afterload. The intravascular volume expansion of pregnancy or following prehydration may offer an adequate preload but may increase regurgitation. Patients will not tolerate myocardial depression, bradycardia and increases in systemic vascular resistance. LRA is surely indicated for C-section as it decreases the afterload.

Mitral valve regurgitation (MVR)

Etiology. Mitral valve regurgitation (MVR) is the most frequent valvular problem. A distinction should be made between primary and secondary, acute and chronic MVR. Any of the components of the valve may be involved such as the chordae, papillary muscles, leaflets and annulus. Primary MVR finds it's most important cause in rheumatic fever, bacterial endocarditis, connective tissue disease, degenerative disease of the annulus (mostly females) and finally a congenital form. MVR may be secondary to ischemia, infarction (posterior papillary muscle dysfunction or chordae rupture, less closing force due to less LV contractility in LVF), dilated cardiomyopathy (annular dilatation following LV dilatation such as in AS or AI).

Symptoms. Patients may be asymptomatic for years. Left ventricular enlargement and hypertrophy will occur later than with AI. Acute symptoms may consist of pulmonary edema and congestive heart failure and mostly follow infective endocarditis or ischemic disease with papillary muscle or chordal dysfunction. It required urgent surgical intervention. Chronic disease will become evident by atrial fibrillation (present in 30% of the cases due to left atrial enlargement), nocturnal dyspnea and dyspnea during exercise. Pulmonary hypertension may exist in 20%.

What happens? During the systole the left ventricle leaks into the left atrium. Up to 50% of the end-diastolic volume may pass the mitral valve before the aortic valve opens. The transvalvular flow depends on the valvular area (variable), the duration of the systole and the pressure gradient between left ventricle and left atrium. The left ventricle will enlarge and become hypertrophic but later than with AI. The left atrium will dilate (detectable by ECG and chest X-ray) as well and will compensate for the volume overload while it will reduce overload of the pulmonary circulation. While in chronic MVR this compensation may progress slowly, acute MVR such as after myocardial infarction with papillary muscle rupture, may cause pulmonary edema as there is not enough time for compensation. The ejection fraction may be as high as 80% whereas 50-60%, as measured in normal patients, would signify depressed contractility. However, this value may be overestimated due to the fact that both forward and regurgitating volumes will be measured.

Anesthetic management. Vasoconstriction and afterload increases should be avoided. Vasodilatation (afterload reduction) will be less efficient than in aortic valve regurgitation. Also incautious volume expansion will not be tolerated well and will cause more blood to regurgitate. Nevertheless a mild preload maintaining relatively high filling pressure is recommended especially in combination with a regional anesthetic technique (which is beneficial because of afterload reduction and avoidance of intubation stress). Excessive filling is bad. Contractility should not decrease. Therefore older volatile anesthetics should be avoided. Bradycardia should be avoided as regurgitation time may equally be prolonged. Heart rates of 90 bpm or even more are recommended. As with aortic regurgitation dobutamine and milrinone may be helpful because they decrease afterload and pulmonary artery pressure and increase contractility. A central line may allow to evaluate volume shifts and pacing possibilities in case the heart rate would be too low (same for AI). On the wedge trace a 'v' wave may be seen.

Obstetric analgesia and anesthesia. The same principles may exist as for aortic insufficiency. Afterload reduction may be less tolerated. Hypotension should be treated with ephedrine rather than phenylephrine. Atrial fibrillation should be avoided although this will be less of a problem in the younger obstetric population. In case of chronic atrial fibrillation, mainly the coagulation status and the degree of emergency will determine the choice of the anesthetic technique.

OTHER MITRAL VALVE REGURGITATING DISEASE

Two other regurgitating conditions may involve the mitral valve but require different treatment than simple mitral valve regurgitation as described above.

Mitral valve prolaps (MVP)

Etiology. This disease may occur in as much as 3% of the population. It is mostly caused by connective tissue defects.

Symptoms. The rather atypical symptoms consist of palpitations / arrhythmias, fatigue, orthostatic hypotension, chest pain, dyspnea, syncope and sudden death. Most prescribed medication include antiplatelet drugs or anticoagulants. On auscultation a midsystolic

click will be noticed at the apex (the location of most mitral sounds) followed by a murmur.

What happens? It concerns one of the mitral valve leaflets becoming hypertrophic i.e. thickening > 2 mm. It will be more evident during the systole mainly mid and late phase. Related to the anatomy of the leaflets there may be minimal or moderate regurgitation. The degree of regurgitation will also depend on left ventricular filling and size. Endocarditis is a substantial risk although questioned recently (see below).

Anesthetic management. Except for endocarditis prophylaxis there are only limited defined anesthetic goals. Atrial and ventricular rhythm abnormalities may occur frequently and should be treated. Anesthetic management despite valve regurgitation seems to correspond with the goals for a stenotic lesion such as avoidance of tachycardia, vasodilatation, enhancement of contractility and preload reduction. Adequate intravascular volume (preloading) is mandatory because a decrease will cause more valvular displacement into the left atrium during the systole. b-blocking agents may be effective for heart rate limitation and decreasing contractility.

Hypertrophic Obstructive Cardiomyopathy (HOCM) + MVR

Etiology. In fact this is still unknown but most probably congenital elements may be involved. Some other disease may resemble the physiopathology of hypertrophic obstructive cardiomyopathy such as Pompe's disease and Hoonan's syndrome. Synonyms of this defect are idiopathic hypertrophic subaortic stenosis (IHSS) or asymmetric septal hypertrophy (ASH).

Symptoms. HOCM symptoms reflect left ventricular dysfunction (hypotension, ischemia, etc.). When mitral regurgitation is present, symptoms of pulmonary volume overload may predominate. Paroxysmal ventricular arrhythmia is also possible.

What happens? Septal hyperthophy will cause obstruction of the ventricular outflow and anterior displacement of the mitral valve during the systole. Late in the disease regurgitation will occur frequently during the early phase of the systole. Diastolic dysfunction will result in decreased LV relaxation. Despite the stenotic nature, HOCM is a dynamic defect depending on preload and contractility.

Anesthetic management. Any effort to reduce left ventricular volume will make obstruction become worse. As a consequence hypovolemia and tachycardia should be avoided. Normovolemia should be aimed at, although some authors recommend hypervolemia. Inotropic drugs, increasing left ventricular contractility may worsen the situation as well. The use of b-blocking agents may improve the clinical condition. Vasodilators, although facilitating ventricular emptying, will after all be less beneficial because they will also reduce left ventricular end systolic volume. Afterload should be maintained or slightly increased. A slow sinus heart rate should be aimed at because the atrial contraction will contribute for 30-40% in left ventricular filling. Especially in the fasted patient the intravascular volume may require expansion. Hypotension should be treated drastically with volume expansion and vasoconstrictors, preferably phenylephrine.

Obstetric analgesia and anesthesia. In most cases, therapy with beta-blocking agents has been stopped during pregnancy to avoid fetal bradycardia and uterine stimulation. There is no clear reason why vaginal delivery would be less safe than a Cesarean section. Low dose labor analgesia has been reported without major problems. For Cesarean section mostly general anesthesia is recommended because its major afterload and preload decreases during spinal anesthesia will not be tolerated.

Endocarditis prophylaxis

During the last years recommendations for endocarditis prophylaxis have changed significantly. As the benefit is not commonly demonstrated prophylactic measures have been reduced. Major determinants are the type of valvular disease, the type and location of surgery and the severity of the intervention. Two microbes are important in the development of endocarditis. Streptococcus Viridans will signify a substantial risk in dental, oral and respiratory procedures while Enterococcus Fecalis will be involved in genito-urinary and gastro-intestinal surgery. Previously i.e. before 2008 congenital defects, acquired valvular disease, hypertrophic cardiomyopathy and mitral valve prolaps are considered to signify a moderate risk for which prophylaxis was 'recommended' (Class I). High risk patients were those with prosthetic valves. Actually prophylaxis is considered to be 'reasonable' (Class IIa) for patients undergoing oral or dental procedures and with prosthetic valves (or prosthetic material used for repair), previous infectious endocarditis, CHD (un-repaired cyanotic, repaired with residual effects, repaired with prosthetic material or device during 6 months after procedure) and finally cardiac transplant with valve regurgitation. Prophylaxis does not seem to be recommended anymore for non-dental procedures in the absence of active infection. For urological and gastro-intestinal reasons, including esophagoscopy, gastro-duodenoscopy and colonoscopy, antibiotics are only indicated in case of active infection

to prevent sepsis or wound infection. Before 2008 it was already agreed that prophylaxis was not recommended in isolated atrial septal defects, patients with repaired atrial, ventricular defects or ductus arteriosus for more than 6 months after the intervention, MVP without regurgitation or thickened leaflets, patients with innocent, physiological or functional murmurs, physiologic MVR without murmur or abnormal valve.

Conclusion

Due to increased aging of the patient and medical progress, progressively more patients are presenting nowadays with valvular problems. Choosing the optimal anesthetic technique should be done on an individual basis taking into account the disease involved, its severity, maternal and/or fetal prognosis, anxiety, medication (anticoagulation), need for specific monitoring, degree of emergency and the preference/experience of the anesthetist and the patient (not necessarily). Although all techniques have been described at least once for any kind of valvular disease, anesthetist should avoid heroism and only take into account the safety of the patient rather than personal glorification.

Table 1. Anesthetic goals

	Heart rate	PL	AL	Contractility	Anesthesia
AS / PS	↓	↑	↑?	---	GA > RA
MS	↓	- ↑	--	---	GA > RA
AI	↑	-- ↑	↓	↑	RA > GA
MVR	↑	-- ↑	↓	↑	RA > GA
MVP	↓	↑	---	---	RA > GA
HOCM+MVR	-- ↓	↑	-- ↑	-- ↓	GA > RA

To end, the table above will summarizes the most important anesthetic goals with respect to heart rate, preload (PL), afterload (AL), contractility and anesthetic technique (GA or RA) for the valvular defects cited above (AS, MS, AVR, MVR, MVP, HOCM + MVR). Another way to make such an overview might be to summarize changes to avoid. In that case the arrows are not just in the opposite direction. It's not that simple.

Recommended reading

Konstadt S. Anesthetic considerations for the patient with heart disease undergoing noncardiac surgery. ASA refresher course lectures (repeated annually).

Johnston WE. Anesthesia for the patients with valvular heart disease. ASA refresher course lectures (repeated annually).

Yarmush L. Noncardiac surgery in the patient with valvular heart disease. In: Anesthesiology Clinics of North America 1997; 15(1): 69-91

Carabello BA, Crawford FA. Valvular heart disease. N Engl J Med 1997; 337: 32-41

ACC/AHA guidelines for the management of patients with valvular heart disease. Task force on practice guidelines. J Am Coll Cardiol 1998; 32: 1486-1588

Harle C. Valvular Heart disease and pulmonary hypertension. In: Anaesthesia and critical disease, McConachie Ed. Cambridge Univ Press, 2006, pp 141-151

Hartman GS, Thomas SJ. Valvular heart disease. In: Aneshesiology. Yao & Artusio's Eds. 6*th Edition. Lippincott, Williams & Wilkins 2008 pp 198-228*

Nishimura RA et al. ACC/AHA 2008 Guideline update on valvular heart disease: focused update in infective endocarditis. JACC 2008; 52: 676-685

Chirurgia noncardiacă şi analgezia obstetricală la pacientul cu valvulopatie: consideraţii anestezice

Rezumat

Pe măsura creşterii duratei de viaţă şi a progreselor recente în medicină, mult mai mulţi pacienţi cu leziuni valvulare sunt supuşi în prezent intervenţiilor chirurgicale non-cradiace. Unii pot beneficia de tehnicile de anestezie regională, în timp ce alţii vor fi cel mai bine rezolvaţi printr-o anestezie generală lipsită de stres. Scopul trebuie să fie nu normalizarea hemodinamicii, ci menţinerea pacientului cât mai stabil posibil comparativ cu situaţia preoperatorie. Deşi mulţi pacienţi pot avea o combinaţie de leziuni, acest referat tratează defectele individuale cele mai frecvent întâlnite, etiologia acestora, simptomele, fiziopatologia şi conduita anestezica. O atenţie specială este acordată cazurilor obstetricale cu care se prezintă şi o populaţie mai vârstnică. Aceasta este o provocare pentru obstetrician şi anestezist datorita modificarilor fiziologice din timpul şi de după sarcină şi al impactului hemodinamic al naşterii, ocitocicelor, tocoliticelor, durerii şi anesteziei.

Cuvinte cheie: cardiac, valvular, obstetrică, anestezie, regional