Anesthetic Management of Patients with Heart Disease

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Anesthesiologists will need to use their knowledge and skills in cardiovascular medicine at a progressively increasing rate, because:

1. The incidence of acquired heart disease is growing as our society ages,
2. The incidence of congenital heart disease is growing as surgical therapy improves, and
3. Even ambulatory centers and office operatories accept patients with severe heart disease.

In fact, the most rapidly growing segment of our population is the elderly, of whom the majority has coronary artery disease and over 2% have significant aortic stenosis. Currently, the adult population in the US includes a million individuals with congenital heart disease, and this number is growing at a rate of 5% each year. Thus, anesthesiologists will care for an increasing number of patients with heart disease.

This lecture will describe a physiologic approach to planning anesthetic management for patients with heart disease. It is not a "cookbook" approach. Indeed, the underlying assumption of this approach is that the choice of anesthetic technique or dose is quite secondary provided appropriate hemodynamic goals and surgical requirements are attained. During my lecture, I will illustrate the use of this approach with a few case examples.

Overview

In patients with heart disease, I recommend a four-step approach to planning anesthetic management: definition of cardiovascular pathology, prediction of cardiovascular pathophysiology, determination of hemodynamic goals, and anticipation of hemodynamic emergencies. In these lecture notes, I will outline these steps and provide four case examples for your consideration. In my lecture, I will review the four steps and use them to determine anesthetic management plans for the case examples as time and questions permit.

Definition of Cardiovascular Pathology

Hemodynamically significant abnormalities of blood flow through the heart and great vessels must be understood to begin a rational approach to planning anesthetic management. Cardiovascular pathology falls into three general categories: obstruction to blood flow, regurgitation of blood flow, and shunting of blood flow. For instance, obstructions include coronary artery or valvular stenosis. Most obstructions are fixed (e.g., aortic stenosis), but some can vary (e.g., right ventricular obstruction in tetralogy of Fallot and left ventricular obstruction in hypertrophic cardiomyopathy). The severity of the obstructions should be defined, if possible. Fortunately, a history of good exercise tolerance usually rules out the possibility of severe obstructions to flow, but in the absence of such a history, additional testing should be considered. However, as a general rule, do not request cardiovascular testing unless you believe that the results of the testing will alter your management of the patient.

Regurgitation of flow is a valvular problem usually identified by auscultation. Again, the degree of exercise tolerance gives a guide to severity, but the chest radiograph will often be of additional help. For instance, severe aortic and mitral regurgitation lead to marked pulmonary vascular changes and cardiomegaly.

Shunting of flow can occur at four anatomic levels: atrial, ventricular, great vessel, or peripheral. Shunting abnormalities include atrial septal defects, ventricular septal defects, patent ductus arteriosus, and arteriovenous malformations. The size of most shunts is fixed (e.g., in cases of atrial septal defects), but some may be variable (e.g., in the ductus arteriosus in newborns). Usually the patient or his past medical record will reveal the results of prior cardiac evaluation with definition of shunt level and size. The most feared complication of shunts is severe pulmonary hypertension. This complication is always accompanied by markedly reduced exercise tolerance and usually frank cyanosis.

Prediction of Pathophysiology

Cardiovascular pathology produces changes in cardiac filling, ejection, and cardiopulmonary perfusion. The second step in our systematic approach is to predict the pathophysiology resulting from the abnormalities defined by the history, physical examination, and

other evaluations. For instance, concentric ventricular hypertrophy is the normal compensatory response when ventricular ejection occurs against increased impedance (e.g., aortic stenosis or tetralogy of Fallot). As a result of hypertrophy, adequate ventricular filling will require higher pressure and be more dependent on atrial contraction than in the absence of hypertrophy. Moreover, at slow heart rates, the hypertrophied ventricle will not dilate adequately to accommodate all the venous return and, as a result, cardiac output falls.

Abnormalities in cardiac ejection are also predictable. The most common example is reduced ejection resulting from ischemia or infarction. However, other predictions require more thought. For instance, in severe aortic stenosis, left ventricular ejection is reduced but requires increased work and myocardial oxygen supply. When systemic vascular resistance falls, the work of this ventricle is maintained (because of the stenotic aortic valve), but its oxygen supply is decreased (as a result of lower aortic diastolic pressure). In contrast, in tetralogy of Fallot and idiopathic hypertrophic subaortic stenosis, the degree of ventricular outflow obstruction depends on the degree of ventricular filling and the vigor of contraction. Low preload and high contractility pronounce obstruction; the converse relieve it.

Shunt physiology is similarly predictable: the degree of shunting at the ventricular or great vessel level will depend on the size of the shunt and the relative resistances to flow in the pulmonary and systemic vasculature. For instance, an infant with hypoplastic left heart syndrome (large shunt at the great vessel level) will become hypotensive and acidotic if given too much supplemental oxygen (the oxygen lowers pulmonary vascular resistance and results in a "steal" of blood from the systemic circulation). In contrast, when an infant with a large ventricular septal defect (e.g., tetralogy of Fallot) becomes cyanotic, the cyanosis can be relieved by increasing systemic vascular resistance (and raising pulmonary blood flow). Thus, when large shunts are present at the ventricular or great vessel level, the balance of pulmonary and systematic flows can be adjusted by changing the vascular resistances.

Determination of Hemodynamic Goals

This is the third and most crucial step in our systematic process. In this step, we determine goals for the major hemodynamic variables affected by anesthesia: preload, heart rate, systemic vascular resistance, pulmonary vascular resistance, contractility, and rhythm. The goals for these variables depend on the cardiovascular pathology of the patient and are the direct consequences of the pathophysiology we predicted in the previous step of this process. For instance, when ventricular filling is impaired because of hypertrophy, our goal for preload is high filling pressures, adequate volume administration, and avoidance of factors that decrease venous return. Please note that the hemodynamic goals during anesthesia may be quite different than the goals during chronic care of the patient. In the same patient with ventricular hypertrophy, a primary care physician may have spent months reducing preload ("pruning") to relieve symptoms of pulmonary congestion. However, this state of relative dehydration may be disastrous in some patients during induction of anesthesia.

Similarly, heart rate must be maintained at low normal levels in patients with dynamic ventricular outflow obstructions (tetralogy of Fallot and hypertrophic obstructive cardiomyopathy) to allow for adequate ventricular filling and ejection. Systemic vascular resistance should be reduced in patients with mitral regurgitation to promote forward ejection of blood, and maintained, or even augmented, in patients with aortic stenosis to provide adequate coronary artery blood flow. Pulmonary vascular resistance is difficult to lower in most patients without producing systemic hypotension. However, in patients with reactive pulmonary vasculature, it is not difficult to raise. Hypercarbia, metabolic acidosis, hypoxia, and light anesthesia can result in dramatic increases in pulmonary vascular resistance. In the rare infant with too much pulmonary blood flow, one or more of these "therapies" may reverse hypotension and improve the systemic circulation.

In anesthesia dogma, too much emphasis has been placed on maintaining cardiac contractility. Indeed, the most successful cardiac drugs of our time have been myocardial depressants. Most patients tolerate modest decreases in contractility and some benefit: patients with coronary artery disease, hypertrophic obstructive cardiomyopathies, and tetralogy of Fallot. In contrast, no patient is improved by the loss of sinus rhythm, and some tolerate it quite poorly: patients with impaired ventricular filling who need their atrial "kick" to maintain an adequate stroke volume (i.e., aortic stenosis).

In each patient, some goals are more important than others, and this fact allows the clinician to prioritize management and interventions. For instance, a relatively slow heart rate (<80/min) and high systemic vascular resistance (greater than 1500 dyne $\cdot$ s $\cdot$ cm^{-5}) are most important in patients with severe mitral stenosis. If such a patient is tachycardic, hypotensive, and overtly in congestive heart failure, the correct interventions may include a β-blocker and a vasoconstrictor. Clearly these interventions would be grossly inappropriate in many patients with congestive heart failure, but they will be effective in this patient because they will restore the appropriate hemodynamic goals.

Anticipating Emergency Treatments

This last step is really an extension of the previous one, but I list it separately to emphasize its importance. A few life-threatening hemodynamic changes recur often enough in patients with cardiovascular disease that they should be anticipated, and the treatment ready to execute. Examples would include severe hypotension after induction of anesthesia in patients with aortic stenosis, and severe cyanosis in patients with tetralogy of Fallot. Indeed, phenylephrine may be the drug of choice in both these emergencies, because the goal is the same—increased systemic vascular resistance. In a true crisis, the difference between effective management and chaos is anticipation and planning.

Case Examples. Please consider the following case scenarios. In my lecture, I will outline my hemodynamic goals and anesthetic plan for these patients.

1. A 75-yr-old man is scheduled for emergency laparotomy to relieve a small bowel obstruction. He has known of his heart murmur for more than 10 yr and recently has noted dizziness during bowel movements. He is not physically active because of arthritis. He takes 5–10 aspirin a day. Examination reveals a 60-kg man in mild abdominal discomfort with blood pressure 110/90, heart rate 90 bpm, and respiratory rate 20 breaths/min. He has severely reduced neck extension, diminished carotid pulsations, small mouth, systolic ejection murmur radiating to the neck, and a moderately distended abdomen. What are your hemodynamic goals and anesthetic plan?
2. A 3-yr-old female patient is scheduled for emergency esophagoscopy to remove a penny from her upper esophagus. She has tetralogy of Fallot palliated with a right Blalock-Taussig shunt (subclavian to pulmonary artery). She becomes cyanotic when she cries, and is scheduled to undergo complete correction of her cardiac defect in 3 mo. She ate a full meal 1 h before admission (including the penny). Examination reveals a 15-kg frightened female with blood pressure of 100/60, heart rate 100 bpm, respiratory rate 30 breaths/min, and oxygen saturation of 92%. She has a systolic ejection murmur heard throughout the precordium, and her fingers are mildly cyanotic and clubbed. What are your hemodynamic goals and anesthetic plan?
3. A 20-yr-old woman is scheduled for elective laparoscopic cholecystectomy. She has tricuspid atresia palliated by a series of operations culminating in a Fontan procedure at age 10. Her physical activity is limited to shopping and housework. Her medications include digoxin, diuretic, and ACE inhibitor. Examination reveals a 50-kg female in no distress with blood pressure of 90/70, heart rate 95 bpm, respiratory rate 20 breaths/min, and oxygen saturation of 92%. What are your hemodynamic goals and anesthetic plan?