Anesthetic Management of Patients with Heart Disease Undergoing Noncardiac Surgery

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In the first 30 years of this century, the number of Americans greater than 65 years of age will double, F1 resulting in over 70 million older individuals (Fig. 1). Other industrialized nations are experiencing a similar graying, and individuals over the age of 60 years have twice the incidence of symptomatic cardiac disease compared with younger patients.[1] In addition, the number of adults with congenital heart disease has increased dramatically in the past few decades due to advances in surgical treatment and medical management.[2] Currently, they number over a million. Thus, anesthesiologists will care for an increasing number of patients with heart disease, including patients having procedures in locations formerly restricted to exclusively healthy individuals, such as ambulatory surgery centers, radiology, and endoscopy suites.

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 the physiological compensation, determination of hemodynamic goals, and anticipation of hemody- T1 namic emergencies and their treatment (Table 1). In these lecture notes, I will outline these steps and provide three 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. If possible in advance of my lecture, please review the cases and outline how you would manage these patients in your own practice. They are real case examples.

DEFINITION OF CARDIOVASCULAR PATHOLOGY

Hemodynamically significant abnormalities of blood flow through the heart and great vessels must be understood in order to begin a rational approach to planning anesthetic management. Most 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 (greater than 6 metabolic equivalents) usually rules out the possibility of severe obstructions to flow. I usually ask whether a patient can climb a flight of stairs carrying groceries (about 5–6 metabolic equivalents). If at all possible, try to corroborate this history with family members or others who know the patient well. In my experience, some patients, especially men, tend to over estimate their exercise ability. In the absence of a convincing history of moderate to good exercise tolerance, additional testing should be considered if you anticipate that the results of the testing will affect the care of the patient. In 2007, the American Heart Association and the American College of Cardiology updated their guidelines on perioperative cardiovascular evaluation and care.[3] They recommend proceeding with surgery without further testing if the patient has good exercise tolerance ($\geq$ 4 metabolic equivalents) unless the patients has any of the following: unstable angina, recent myocardial infarction, decompensated heart failure, significant arrhymthmias, or severe valvular heart disease.

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., atrial septal defects), but some may be variable (e.g., 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

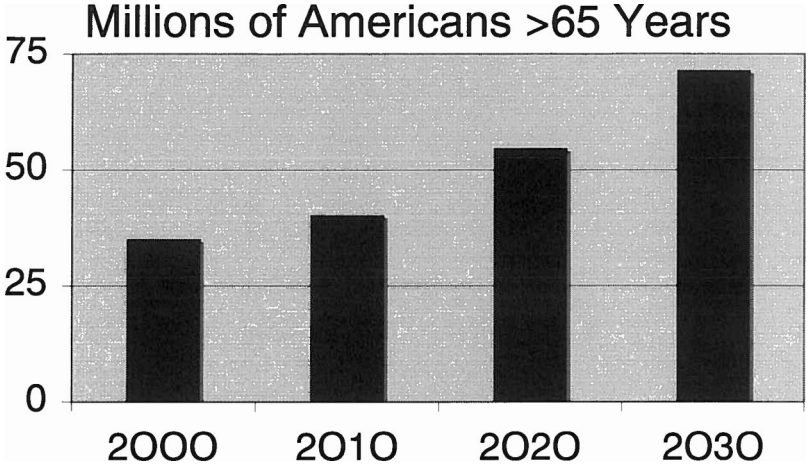

Figure 1. The number of individuals living in the United States who are over 65 years of age in the year 2000 and projected numbers for the years 2010–30. Data were taken from the United States Census Bureau tables.*

Table 1. Management Steps in Patients with Heart Disease

1. Define the cardiovascular pathology
2. Predict the physiological compensations
3. Determine the hemodynamic goals
4. Anticipate the cardiac emergencies and their treatment

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 PHYSIOLOGICAL COMPENSATIONS

Cardiovascular pathology produces changes in cardiac filling, ejection, and cardiopulmonary perfusion. The second step in our systematic approach is to predict the physiological compensations 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., systemic hypertension, aortic stenosis, or tetralogy of Fallot). As a result of hypertrophy, adequate ventricular filling requires higher atrial pressure and is more dependent on atrial contraction than in the absence of hypertrophy. These abnormalities of ventricular filling are called "diastolic dysfunction," and they are very common in the elderly. Bradycardia is tolerated poorly in patients with diastolic dysfunction, because the ventricle does not dilate adequately to accommodate all the venous return causing a decrease in cardiac output.

Abnormalities in cardiac ejection are also predictable. The most common example is reduced ejection due to 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 (due to 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, while the converse relieve it.

Similarly, shunt physiology is 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, increasing systemic vascular resistance relieves the cyanosis by decreasing the right-to-left intraventricular shunting. 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. Fn1

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 physiological compensations we predicted in the prior steps of this process. For instance, when ventricular filling is impaired because of hypertrophy, our goal is to maintain preload (high filling pressures), generous volume administration, and avoidance of factors that decrease venous return. Please note that the hemodynamic goals during anesthesia may be quite different than

*www.census.gov/ipc/www/usinterimproj/natprojtab02a.pdf.

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 it. 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 diastolic dysfunction 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 bpm) and high systemic vascular resistance (greater than 1500 dynes/sec/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 prior 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 following 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-year-old male is scheduled for emergency laparatomy to relieve a small bowel obstruction. He has known of his heart murmur for more than 10 years and recently has noted dizziness during bowel movements. He is not physically active due to arthritis. He takes 5–10 aspirin a day. Examination reveals a 60 kg male in abdominal discomfort with BP 110/90, HR 90, and RR 20. 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-year-old female 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 months. She ate a full meal 1 hour prior to admission (including the penny). Examination reveals a 15-kg frightened female with BP 100/60, HR 100, RR 30, and sat 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-year-old female 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 BP 90/70, HR 95, RR 20, and oxygen saturation of 92%. What are your hemodynamic goals and anesthetic plan?

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1. Rosamond W, Flegal K, Furie K. Heart disease and stroke statistics—2008 update: a report from the Am Heart Association Statistics Committee and Stroke Statistics Subcommittee. Circulation 2008;117:e25–146
2. Russell IA, Rouine-Rapp K, Stratmann G, Miller-Hance WC. Congenital heart disease in the adult: a review with internet-accessible transesophageal echocardiographic images. Anesth Analg 2006;102:694–723
3. Fleisher LA, Beckman JA, Brown KA. ACC/AHA 2007 guidelines on perioperative cardiovascular evaluation and care for noncardiac surgery: a report of the Am College of Cardiology/Am Heart Association Task Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines on Perioperative Cardiovascular Evaluation for Noncardiac Surgery) developed in collaboration with the Am Society of Echocardiography, Am Society of Nuclear Cardiology, Heart Rhythm Society, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society for Vascular Med and Biology, and Society for Vascular Surgery. J Am Coll Cardiol 2007;50:e159–241

2007 Algorithm

Step 1: Emergency ⇒ OR, if not

Step 2: Active CC = STOP, if not

Step 3: Low risk surgery ⇒ OR, if not

Step 4: MET ≥ 4 ⇒ OR, if not

Step 5: 2-3 RF + hi risk ⇒ ? E&M

AHA/ACC Guidelines

- Periop CV evaluation noncardiac pts
- Fleisher et al. Oct 2007 JACC & Circ
- Tailored evaluation based on risk
- Urgent & emergent surgery proceed
- Consultation: 3.4% + data, 42% ∅

If CRV ≠ option - no stress testing

Active Cardiac Conditions

- ✓ Unstable/severe angina or recent MI*
- ✓ Decompensated or new heart failure
- ✓ Significant arrhythmias:
  - 2-3° AVB and symptomatic sinus bradycardia
  - symptomatic vent arrythmias & new VT
  - SVT with poorly controlled rate (HR >100)
- ✓ Severe valvular disease

Delay or cancel, evaluate & Rx

Clinical Risk Factors

- History of ischemic heart disease
- History of compensated or prior HF
- History of cerebrovascular disease
- Diabetes mellitus
- Renal insufficiency

Metabolic Equivalents

1 MET: Resting or TV watching

2 METs: Dress without stopping

3 METs: Slow level walking

4 METs: Golf in a cart or bowling

5 METs: Climb stairs without resting

6 METs: Fast walking or stairs with load

Hemodynamic Goals

	HR	PrLd	SVR	PVR	Con	Rhy
CAD	⇓	⇓	nl	nl	⇓	S
MS	⇓	⇑	⇑	⇓	nl	S
AR	⇑	⇑	nl	nl	nl	S
MR	⇑	nl	⇓	nl	nl	S

The Four Step System

- Define CV pathology
- Predict CV pathophysiology
- Determine hemodynamic goals
- Anticipate CV emergencies

Aortic & Mitral Stenosis

	Aortic Valve	Mitral Valve
Normal	2-3	4-6
Mild	1.5-2	2-4
Moderate	1-1.5	1-1.5
Severe	<1	<1

Modified Bernoulli

$$p + 1/2\rho V^2 + \rho g h = k$$

Modified Bernoulli

$$\Delta P = 4V^2$$

4 m/s V = 64 mmHg gradient

Continuity Equation

$$A_1 * V_1 = A_2 * V_2$$

$$AV_{area} * AV_{velocity} = LVOT_{area} * LVOT_{velocity}$$

V_1 = 4 m/s AV velocity

V_2 = 1 m/s LVOT velocity

A_2 = 4 cm^2 LVOT area

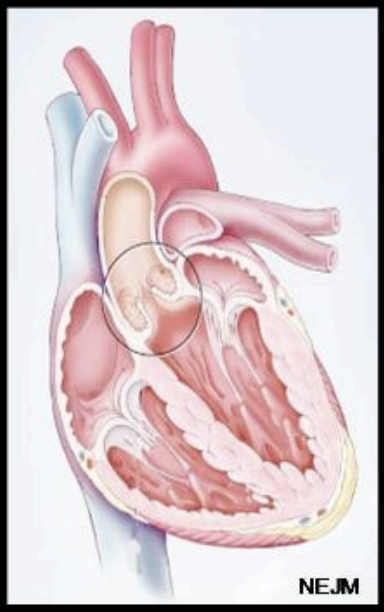
A_1 = 1 cm^2
AV area

$A_2 = (d/2)^2 * \pi$

Critical Aortic Stenosis

- Angina
- Syncope
- Heart failure

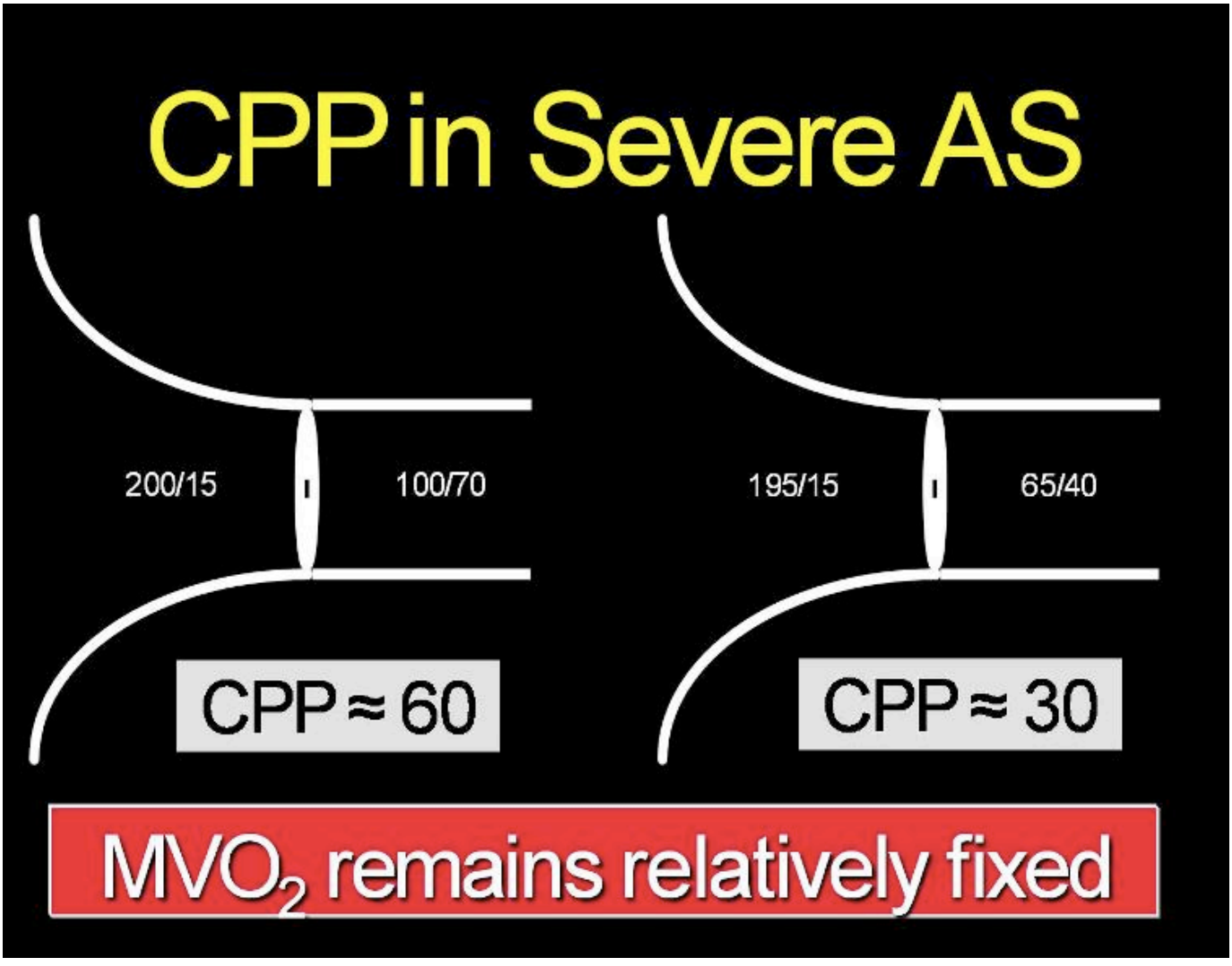
? Poor METs



Predicted periop mortality > 10%

Obstruction

- Valvular stenosis:
  - pressure overload & ⇑MVO_2
  - concentric ventricular hypertrophy
  - decreased ventricular compliance
  - AS: SVR & MVO_2 uncoupled


CPP in Severe AS
200/15
100/70
CPP ≈ 60
195/15
65/40
CPP ≈ 30
MVO2 remains relatively fixed

Kertai: AJM 2004

- 108 pts with AS vs 216 controls
- Non-cardiac surgery 1991-2000
- Outcomes: death or MI (controls: 2%)
  - Severe AS: 31%
  - Moderate AS: 11%
- Adjusted odds ratio 5.2 (1.6-17)

Regurgitation

- Semiquantitative evaluation at best
- Marked dependence on loading
- MR may be intermittent with ischemia
- Heart size & symptoms correlate little
- Exercise tolerance is good guide

Shunting

- Three cardiac levels of shunting:
  atrial, ventricular, & great vessel
- Fixed or dynamic shunt
- Restrictive or unrestrictive flow
- Exercise tolerance good guide

Shunting Complications

- Systemic emboli and endocarditis
- Congestive heart failure
- Pulmonary hypertension:

  flow related only - normal resistance

  increased resistance

  severe PHTN: Eisenmenger's

Regurgitation

- Valvular regurgitation
  - volume overload & increased MVO_2
  - eccentric hypertrophy
  - venous congestion
  - resistance dependent output

Shunting

- Volume &/or pressure overload
- Eccentric hypertrophy
- PVR:SVR determine shunting when:
  - ventricles or great vessels involved
  - shunts are large or unrestrictive

Determine Goals

- Heart rate
- Preload
- Systemic vascular resistance
- Pulmonary vascular resistance
- Contractility & Rhythm

Fontan Palliation