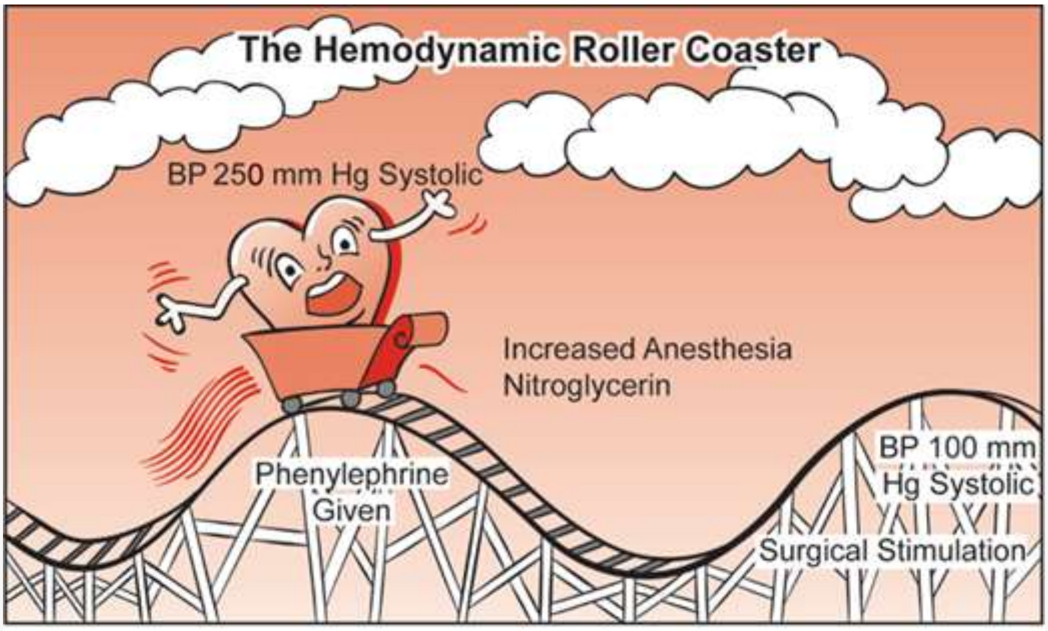
Chapter 2: Hemodynamics and Cardiac Anesthesia

INTRODUCTION

Understanding hemodynamic principles helps cardiac anesthesiologists determine the mechanisms underlying hemodynamic instability and guides treatment. Fortunately, the same basic physiologic principles apply in both the healthy patient undergoing laparoscopic cholecystectomy as well as in the patient with low ejection fraction undergoing multiple valve replacements. Unfortunately, the patient undergoing cardiac surgery is more likely to decompensate severely when faced with the hemodynamic roller coaster sometimes associated with general anesthesia induction (Figure 2–1).

FIGURE 2–1.

Anesthesia manipulations can often stress the heart and the patient. Although many healthy patients can tolerate these swings in blood pressure, the cardiac surgery patient may be unable to do so without developing myocardial ischemia and ventricular dysfunction. (Reproduced with permission from Wasnick JD: *Handbook of Cardiac Anesthesia and Perioperative Care*. Boston, MA: Butterworth Heinemann; 1998.)



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HEMODYNAMIC CALCULATIONS AND INVASIVE MONITORS: WHY ARE THEY IMPORTANT AND HOW DOES ONE DETERMINE THEM?

Blood Pressure

Although the absolute definition of hypotension is somewhat clouded in the literature,[1] a patient is considered hypotensive when the systolic blood pressure is reduced by 20% or more from the patient's baseline blood pressure. Different authors set different cutoffs as to what constitutes a hypotensive patient. Although in the past a systolic pressure of less than 90 mm Hg was thought hypotensive, this value has recently been suggested as being too low. For example, the new cutoff value for hypotension has been reset to 110 mm Hg systolic in trauma patients.[2] A mean arterial blood pressure less than 65 mm Hg has been associated with adverse outcomes perioperatively and should be corrected. However, at times during cardiac procedures surgeons may request relative hypotension to facilitate aortic cannulation or to control bleeding. At other times physical manipulation of the heart by the surgeons will transiently reduce blood pressure. Close communication between the surgeon and anesthesiologist is critical for effective hemodynamic management.

Each practitioner must determine for each individual patient what systemic pressures, high and low, warrant treatment. In the adult cardiac surgery patient, it is likely that any patient with a systolic blood pressure much less than 90 mm Hg would be considered in need of hemodynamic intervention of some kind depending upon the etiology of the hypotensive episode. Hypertension is also aggressively treated in the cardiac surgery patient. This chapter will examine how to approach the hypotensive cardiac patient and to apply appropriate therapy. Some of the causes of perioperative hypotension are presented in Table 2–1.

Chapter 3: Perioperative Rhythm Abnormalities

INTRODUCTION

Alterations in heart rhythm and rate can have sweeping and at times ultimately fatal hemodynamic consequences perioperatively. Thus, the rapid interpretation of abnormal rhythms and their correction is critical in cardiac anesthesia practice.

THE ELECTROCARDIOGRAM

The electrocardiogram (ECG) remains one of the main monitors used by anesthesiologists. It is primarily employed in anesthesia practice to detect heart rate and rhythm changes and perioperative myocardial ischemia. The ECG detects electrical currents generated by the electrical activity of the heart. ECG leads are placed in different positions and provide various perspectives (depending upon where the lead is placed) of the electrical activity of the heart as electrical vectors point toward or away from the examining leads. Examining the ECG in multiple leads provides the anesthesiologist the ability to discern if perceived changes in the ECG pattern are widespread (found in multiple leads) or are perhaps less significant (motion artifact). At the end of diastole, atrial depolarization generates the “P” wave and is followed by atrial contraction. Following atrial contraction, the ventricle is loaded awaiting systole. Systole commences at the QRS beginning with isovolumetric contraction following a 120- to 200-ms conduction delay at the AV node. Subsequently, intracavitary pressure builds, the atrioventricular valves (e.g., mitral or tricuspid) close, and the arterioventricular valves (e.g., aortic, pulmonic) open resulting in ventricular ejection of the stroke volume (SV). The QRS represents the electrical activity generated by the depolarization of the left and the right ventricles. Depolarization proceeds from the AV node through the interventricular septum via the His-Purkinje fibers. The QRS segment lasts approximately 120 milliseconds. Repolarization of the ventricles produces the ST segment and the T wave. Electrolyte abnormalities (e.g., hypocalcemia) and drug effects (e.g., droperidol) can delay repolarization leading to a prolonged QT interval. This can result in potentially life-threatening ventricular arrhythmias (see Figures 3–1 and 3–2).

FIGURE 3–1.

Conducting system of the heart. Left: Anatomic depiction of the human heart with additional focus on areas of the conduction system. Right: Typical transmembrane action potentials for the SA and AV nodes, other parts of the conduction system, and the atrial and ventricular muscles are shown along with the correlation to the extracellularly recorded electrical activity, that is, the electrocardiogram (ECG). The action potentials and ECG are plotted on the same time axis but with different zero points on the vertical scale for comparison. AV, atrioventricular; LAF, left anterior fascicle; SA, sinoatrial. (Data from Donahue JG, Choo PW, Manson JE, et al. The incidence of herpes zoster. *Arch Intern Med*. 155:1605–1609, 1995; Choo PW, Galil K, Donahue JG, et al. Risk factors for postherpetic neuralgia. *Arch Intern Med*. 1997;157:1217–1224.)

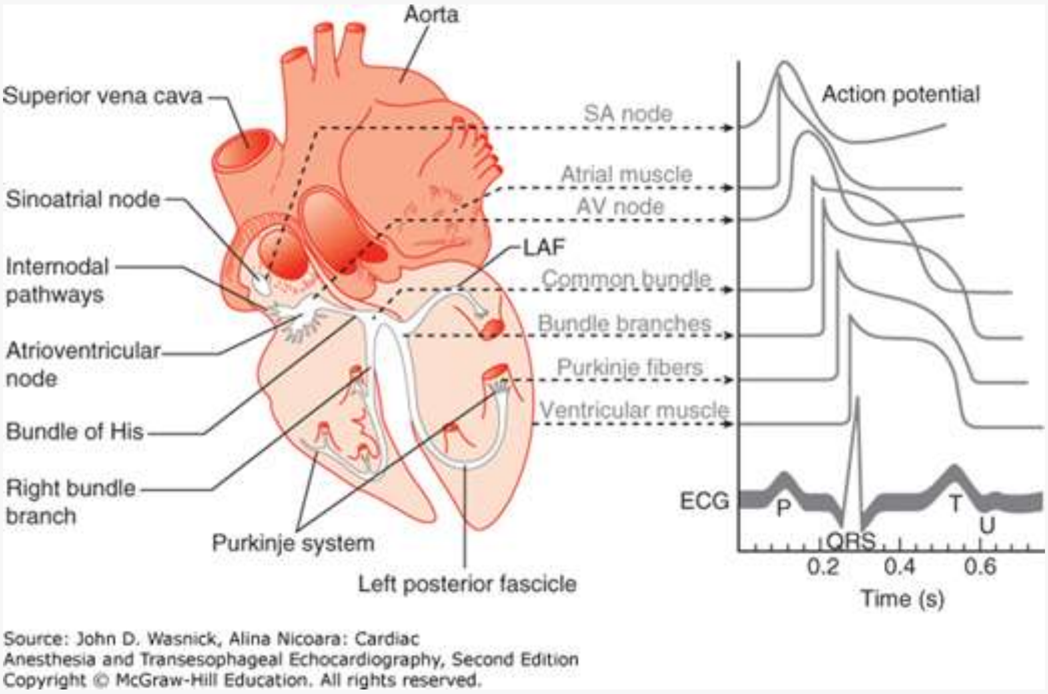


FIGURE 3–2A,B.

The progression of cardiac conduction in the heart during a cardiac cycle. (Adapted with permission from Rushmer RF: *Cardiovascular Dynamics*, 2nd ed. Philadelphia, PA: Saunders; 1961.)

Chapter 5: The Complicated Patient for Cardiac Anesthesia and Surgery

INTRODUCTION

The elective patient for cardiac anesthesia and surgery free of other disease processes is increasingly a *rara avis*. Prior to advances in percutaneous interventions, the routine cardiac surgery patient was an otherwise healthy middle-aged biological male in need of a one to two vessel coronary artery bypass—How times have changed. Today's cardiac surgery patient is likely to be quite elderly with multiple medical problems presenting for combined revascularization and valvular replacement surgery. Moreover, many patients will have had over the course of their lives other cardiac procedures including previous operations and/or percutaneous interventions. Further complicating matters, many of these patients suffer from both systolic and diastolic dysfunction.

THE PATIENT WITH IMPAIRED SYSTOLIC AND DIASTOLIC VENTRICULAR FUNCTION

Systolic Dysfunction

In past decades, patients presented for cardiac surgery in need of one to two vessel bypass grafts. Usually, their chief complaint was angina and they had no or minimal myocardial damage. Patients with ejection fractions (EFs) of greater than 60% were the rule and not the exception.

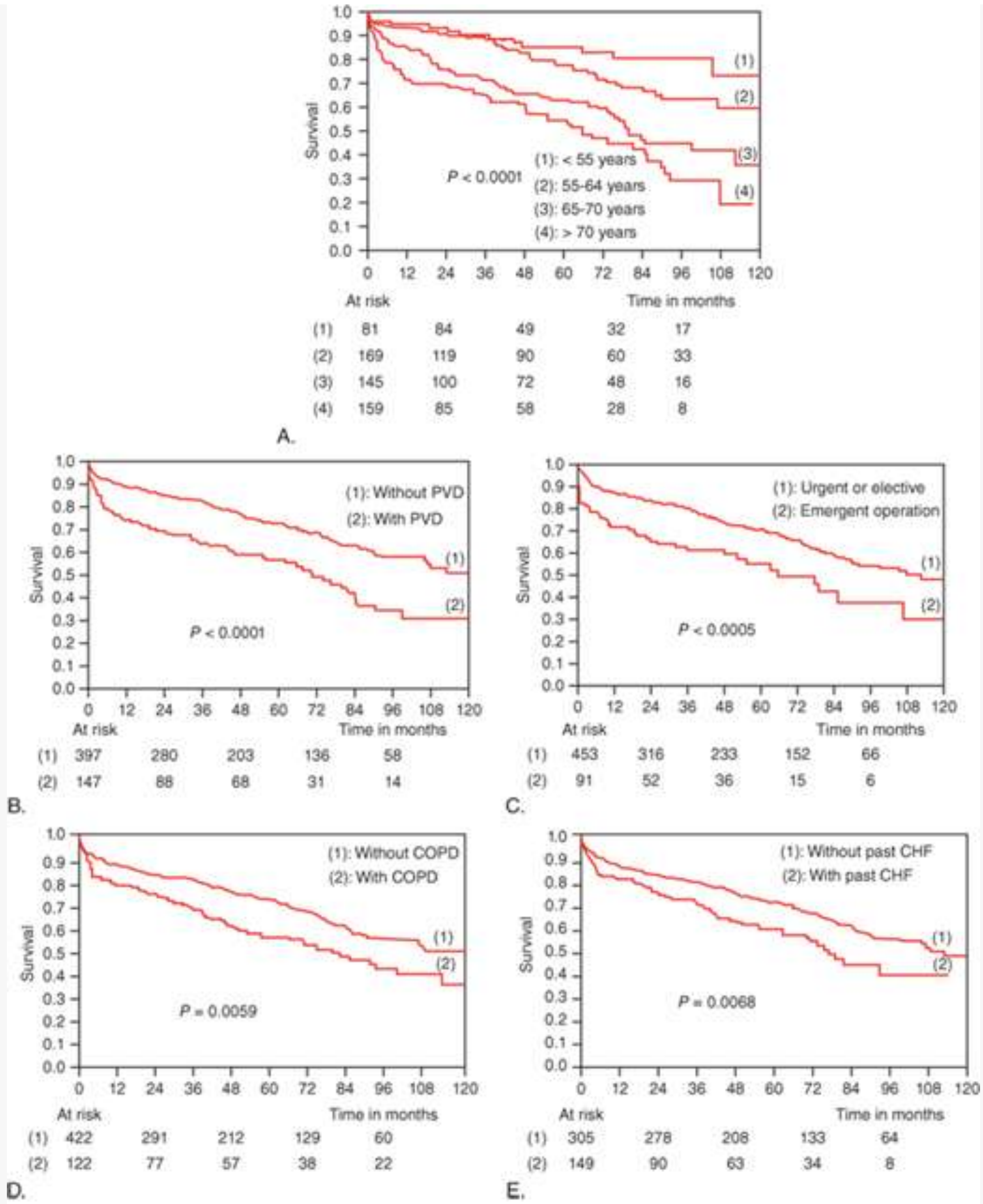
Such patients tended to tolerate the peri-induction period well and were readily separated from cardiopulmonary bypass (CPB) with little need for pharmacological or mechanical support. Ventricular function tended to be preserved throughout surgery and recovery, assuming acceptable myocardial preservation and surgical techniques.

Today's cardiac surgical patient is far more challenging. Patients are older with varying degrees of systolic and diastolic dysfunction frequently presenting in congestive heart failure.

Congestive heart failure affects more than 5 million Americans with coronary artery disease (CAD) as a primary etiology.[1] Cardiac surgical outcomes are worse in patients with previous episodes of congestive heart failure, chronic obstructive pulmonary disease, increased age, and peripheral vascular disease (Figure 5–1).

FIGURE 5–1.

In a retrospective analysis of 525 patients with ejection fractions less than 25%, long-term outcomes were identified in these Kaplan-Meier survival curves. Increasing age (A), presence of peripheral vascular disease (B), emergent nature of surgery (C), presence of chronic obstructive pulmonary disease (D), and previous episodes of pulmonary congestion (E) all predict a poorer long-term outcome following coronary artery bypass surgery. [Reproduced with permission from DeRose JJ Jr, Toumpoulis IK, Balaram SK, et al: Preoperative prediction of long-term survival after coronary artery bypass grafting in patients with low left ventricular ejection fraction, *J Thorac Cardiovasc Surg.* 2005 Feb;129(2):314-21.]



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However, preserved right ventricular function in the setting of a severely compromised left ventricle (EF < 25%) may improve perioperative outcomes[2] (Figure 5–2). Impaired LV diastolic function produces an increase in left ventricular end-diastolic pressure (LVEDP) that is transmitted to the pulmonary circulation. A patient whose right ventricle functions well and tolerates any perioperative worsening in LV diastolic dysfunction has a potentially better surgical outcome than a patient with biventricular failure.