INTRODUCTION

The successful transition from full circulatory support to the normal circulatory physiology requires careful attention to a number of parameters that are often perturbed while on cardiopulmonary bypass (CPB). Many of these parameters, such as temperature, extracellular glucose and potassium concentrations, hematocrit, and acid-base status, are relatively easy to restore. However, the re-establishment of many aspects of normal cardiovascular function, such as peripheral vascular resistance, blood pressure, and cardiac function, are not as straightforward, due in part to the unusual physiologic conditions imposed. The purpose of this chapter is to elaborate the cardiovascular physiology relevant to the transition from full circulatory assist, and to describe a model that will allow for a better understanding of the unique consequences that occur.
THE DETERMINANTS OF VENTRICULAR FUNCTION

The fundamental determinants of ventricular function are classically separated into heart rate, preload, afterload, and contractility. It is particularly important to review these concepts in the context of external circulatory support, as the conditions under which the heart operates are far from classical. For example, consider that while on CPB the heart is performing no external work, despite the fact that it may have normal electrical activity, normal excitation—contraction coupling, and normal myocyte function. In other words, despite the fact that the heart may have normal function on the cellular level, the heart is not participating in generating pressure or flow in the vasculature. Although this point may seems obvious, it is important to understand these conditions to appreciate the types of physiologic changes that occur during the transition to the normal circulatory state.

Preload

Preload represents the stretch on the muscle just prior to its contraction. The Frank-Starling relationship, arguably the most important observation in cardiovascular physiology, describes the increased strength of contraction associated with increased preload (Fig. 1). While preload is most simply represented by a weight hanging from a muscle strip just prior to contraction, preload in the intact heart is quite a bit more complicated and is influenced by a number of factors that impact on ventricular filling (and hence end-diastolic stretch). These factors include blood volume, venous capacitance, ventricular compliance, atrial function, valvular function, and heart rate. As intravascular volume increases, so too increases venous pressure, and hence, the driving force for ventricular filling. Constriction or dilation of the venous capacitance vessels will alter the venous pressure associated with a given intravascular volume; sudden changes in venous capacitance may be associated with considerable hemodynamic instability, which are predominantly caused by changes in the driving force for ventricular filling. Although the filling pressure provides the energy for ventricular distention, the ventricular volume that results is also determined by the compliance of the ventricle. Ventricular compliance may be thought of as the passive mechanical properties of the chamber; a noncompliant ventricle will require higher filling pressures to achieve a given ventricular volume. The majority of ventricular filling occurs early in diastole, and it is manifest on the transmitral flow tracing as the “e” wave (Fig. 2). The pressure gradient between the atrium and ventricle is rapidly dissipated by the rapid movement of blood out of