Diastolic (Dys)Function in Sepsis

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Introduction

Sepsis is a clinical syndrome that results from the systemic response of the body to infection [1]. It is a serious clinical problem, accounting for substantial morbidity and mortality. The majority of these patients die of refractory hypotension and of cardiovascular collapse [2].

The hemodynamic consequences of sepsis are complex and wide ranging. These consequences can result from absolute or relative decrease in central blood volume [3], altered left ventricular (LV) [4, 5] and right ventricular (RV) function [6], and severe peripheral vasodilation [7]. The etiology of these cardiovascular abnormalities is complex but appears to be mediated by a circulating factor(s) [8].

Research regarding the cardiovascular manifestations of sepsis has tended to focus upon the evaluation of systolic performance. However, diastolic dysfunction is increasingly appreciated as a contributor to morbidity and mortality in other clinical settings [9]. Diastolic dysfunction can impact adversely on ventricular filling. However, the impact of sepsis upon diastolic function is incompletely understood.

The principal aim of this chapter is to review current methods of assessing diastolic function in the critically ill patient and examine the evidence regarding the impact of severe sepsis and septic shock upon ventricular diastolic function.

Definition of Diastole

The challenge of conceptually dividing diastolic from systolic ventricular function is highlighted by the number of definitions in the cardiac literature. The traditional definition of diastole refers to the period of the cardiac cycle from the end of ventricular ejection until the onset of ventricular tension development during the subsequent beat [10]. An alternative defines systole by the myocyte contraction-relaxation cycle and diastole refers to the remainder of the cardiac cycle [11]. However, since the traditional definition is more widely used clinically, it will be accepted here. Thus, diastole normally consists of isovolumetric relaxation, early diastolic rapid filling, diastasis (slow filling), atrial contraction (see Figure 1).
Evaluation of Diastolic Function

No single index reliably differentiates normal from abnormal diastolic function. Therefore, comprehensive evaluation of diastolic function relies upon measurement of a number of indices (Table 1). Although diastolic function is a complex interplay of numerous components, the most clinically relevant determinants of ventricular filling include ventricular relaxation, stiffness, and filling pressures. These determinants may be assessed either at cardiac catheterization or by echocardiography.

Ventricular relaxation is the result of a series of energy-consuming steps that result in a decline in myocardial tension [12]. It consists of the isovolumetric relaxation and early diastolic filling periods [13]. Classically, relaxation has been described by invasive measures such as the maximum rate of pressure decline (-dP/dt) and the time constant of relaxation (tau or \(\tau\)) [11].

Non-invasive measures, such as those performed during echocardiography, are more readily performed in the intensive care unit (ICU). These include Doppler evaluation of mitral valve inflow such as isovolumetric relaxation time (IVRT), peak E wave velocity, E/A ratio, E/time velocity integral (VTI), and the E-wave deceler-