INTRODUCTION

Three basic pathophysiologic conditions are responsible for the development of heart failure in man : pressure overload, volume overload, and primary myocardial disease (cardiomyopathy) ***(Meerson, 1983).***

In fact, it is now recognized that heart failure is a condition with varied clinical presentations that range from asymptomatic left ventricular dysfunction to overt congestive heart failure ***(The SOLVD Investigators, 1992).***

Despite the repeated observation that patients with heart failure have elevated levels of TNF-α in their plasma ***(Levine et al., 1990; McMurray et al., 1991; Dutka et al., 1993; Wiedermann et al., 1993; Katz et al., 1994; Matsumori et al., 1994)***, the clinical significance of this finding remains unknown.

Indeed, although the elaboration of TNF-α in patients with heart failure was proposed originally as a biochemical mechanism for the cachexia that occurs in this syndrome ***(Levine et al., 1990),*** it is also known that overexpression of this proinflammatory cytokine can produce left ventricular dysfunction, pulmonary edema, and cardiomyopathy in human subjects ***(Suffredini et al., 1989).***

This observation raises the possibility that TNF-α might actually contribute to the primary progression of heart failure ***(Packer, 1995).***

Although previous studies have consistently identified elevated levels of TNF-α in patients with heart failure, only 30% to 40% of the patients in these studies actually have elevated circulating levels of TNF-α ***(Levine et al., 1990; Matsumori et al., 1994).***

Thus, for reasons that are unclear, not all patients with heart failure appear to elaborate TNF-α. Furthermore, the actual elaboration of TNF-α in pediatric patients with heart failure has not yet been investigated.