INTRODUCTION

Sepsis is a systemic, deleterious host response to infection may progress to severe sepsis (acute organ dysfunction secondary to documented or suspected infection and septic shock (severe sepsis plus hypotension not reversed with fluid resuscitation). Severe sepsis and septic shock are major healthcare problems, affecting millions of people around the world each year, killing one in four (and often more), and increasing in incidence . Similar to polytrauma, acute myocardial infarction or stroke, the speed and appropriateness of therapy administered in the initial hours after severe sepsis develops are likely to influence outcome **(1).**

In most cases, the assessment of infection response to antibiotics relies mostly on the evolution of the same criteria used for diagnosis **(2)** . Microbiological criteria are also of little help in the assessment of response, because of the time needed to obtain culture results, the interference of antibiotics on bacterial growth in vitro and possible difficulties in recollecting some microbiological samples. These areas of uncertainty in the clinical decision-making process led investigators to look at the inflammatory cascade for potential objective markers of infection .These biomarkers, among which C-reactive protein (**CRP**) is one of the most studied **(3),** could be used as surrogates of infection diagnosis.

It has been shown that a single CRP measurement helps in the diagnosis of infection with some controversy concerning prognosis **(4)** and that serial determinations are useful in the prediction of infection as well as in monitoring its response to treatment **(5).**

Thyroid hormones play an important role in the adaptation of metabolic function to stress and critical illness. In hospitalized patients, thyroid hormone alterations are very common, particularly in elderly patients or in those with critical illness **(6).** Low triiodothyronine (T3) is commonly observed in the latter group of patients, which can be attributed to increased deiodination of thyroxine (T4) to reverse T3 (rT3), rather than T3, and increased catabolism of T3 to 3,3-diiodothyronine (T2)**(7).**With increasing severity of illness, low total and free T4, and sometimes low TSH, can be observed . Decrease in plasma T4-binding globulin (TBG) or transthyretin as well as accumulation of substances that lower the plasma thyroid hormone-binding capacity appear also to be important for the above-mentioned alterations in thyroid hormone levels during critical illness **(8)** .

These alterations in thyroid hormone levels are referred to as "euthyroid sick syndrome" or "nonthyroidal illness syndrome" {NTIS}, which is characterized by low serum levels of free and total triiodothyronine (T3) and high levels of reverse T3 (rT3) accompanied by normal or low levels of thyroxin (T4) and thyroid-stimulating hormone (TSH).For the vast majority of patients, thyroid

function abnormalities observed during critical illness are transient and do not represent an underlying thyroid disease.

Thyroid dysfunction is associated with the mortality of patients admitted to the ICU **(9).** The magnitude of the thyroid function test result abnormalities seems to depend on the severity and duration of illness, rather than the type of illness **(10)** .

Researchers in some studies demonstrated that free triiodthyronine (FT3) levels in non survivors were significantly lower than those in survivors in acute respiratory distress syndrome and prolonged mechanical ventilation**(11)** ,whereas other researchers showed that there was no association between FT3 levels and ICU patient outcomes**(12).** Conflicting results also were reported in terms of other indicators, such as FT4 and TSH **(13).**

AIM OF THE WORK

The aim of this work is to investigate the relation between thyroid hormone and mortality in patients with sepsis, severe sepsis and septic shock in comparison with C reactive protein and interleukin-6 .