INTRODUCTION

The mortality and morbidity from acute bacterial meningitis are still high despite substantial progress in diagnosis and treatment (Feigin and Dodge, 1976).

The development and course of meningeal infection are governed by the complex interaction of many variables including microbial virulence factors as encapsulation, clearance of bacteria from the blood, antibacterial mechanisms in the subarachnoid space, anatomic and functional changes resulting from inflammation and antibiotic therapy (Scheld, 1981).

Because bacterial meningitis is in essence of a closed space infection, local defence mechanisms may be important factors that limit bacterial growth.

Normal CSF can be considered as a site of "local defect in host defence", because it contains no phagocytic cells but has a low protein concentration with a predominance of albumin, (Tibbling et al., 1977). Also it contains no IgM and has low levels of C_3 and C_4 (Petz, 1978). Moreover, the bactericidal and opsonic activities are not detectable in normal CSF (Simberkoff et al., 1980).

As a result, the CSF could be overcome by organisms and so multiplication takes place. Once established, bacterial meningitis initiates inflammaotory reactions in the CSF that results in the generation of chemotactic factors and various classes of immunoglobulins leading to improvement of the local host defence (Greenwood, 1978).

Our present study aims at exploring the improvement of the local defence mechanisms in the form of the change in immunoglobulin levels, and the difference between the levels in CSF and in serum.