INTRODUCTION

Bacterial meningitis still the cause of extensive brain injury despite the use of highly active antibiotics.

The mechanism of this injury is not completely understood however several factors are found to be responsible for brain injury including bacterial components, host factors such as cyteokines, arachidonic acid metabolities, platelet activating factors, complement, granulocytes and reactive oxygen intermediates, the proinflammatory cytokines, tumor necrosis factor and interleukin (iL)-IB ***(Ramilo et al, 1990 and Waage et al., 1989).***

Recently NO has attracted attension as a potensial neurotexic factor, NO is a short-lived free radical produced by a Varity of cell types and involved in physiologic processes, such as smooth muscle relaxation, neuronal signaling, inhibition of platelet aggregation and regulation of cell-mediated cytotoxicity ***(Anggard, 1994, Bredt et al., 1994, Lowenstein et al., 1994*** and ***Moncada et al., 1993).***

Including neurotransmission, regulation of cerebral vascular tone and cerebral blood flow, and mediation of ischemic and excitotoxic neuronal injury ***(Wang and Ferriereo et al., 1995).***

Berkowitz et al., (1993) reported that NO contributes to pial arteriolar dilation and impaired autoregulation of cerebral blood flow during experimental H.influenzae meningitis in rats Recently C.S.F levels of the degradation products of NO (nitrate and nitrite) were reported to be elivated in experimental animals and patients with bacterial meningitis ***(Visser et al., 1994 and Koedel and Buster et al., 1995).***

In 1998 ***Tsukahara et al.,*** reported that NO production was enhanced in the C.S.F of children with septic meningitis and was normal in those with aseptic meningitis.