INTRODUCTION: Traumatic brain injury (TBI) is caused by a blow to the head or a penetrating injury that disrupts the normal function of the brain (Cole, 2004). TBI patients demonstrate a number of complications such as memory loss, anxiety and depression (Salmond and Sahakian, 2005). Guanosine (GUO) has been implicated in neuroprotection through the modulation of glutamatergic system. The objective was to evaluate whether treatment with GUO was able to avoid the behavioral alteration caused by TBI. MATERIAL AND METHODS: The animals were anesthetized, the cannula was placed over the craniotomy with dental cement and the TBI were realized (D'Ambrosio et al. 2004). After 2h GUO treatment (7.5mg/Kg intraperitoneal) was started and continued daily until 20 days. Locomotor activity was measured in the open field test starting on day 6 after the TBI until the day 9 (Walsh and Cummins, 1976), in day 14 anxiety was evaluated (Pellow et al., 1985), in days 15 and 16 the step-down passive avoidance task was used to study nonspatial short and long term memory (Sakaguchi et al., 2006) and in days 19 and 20 the novel object recognition test was applied (De Lima et al., 2005). RESULTS: The locomotor activity was not altered in any of the groups. TBI group demonstrated a significant increase in anxiety behavior and a significant decrease of nonspatial short and long term memory. However, the GUO treatment shows an improvement in behavioral outcomes. CONCLUSION: GUO was able to blunt the alterations indicating that TBI affects the animal’s behavior.

Keywords: Guanosine, trauma brain injury, neuroprotection.