Stress ulcers in neurosurgery

The risk of developing stress ulcers (SU) AKA Cushing's ulcers is high in critically ill patients with CNS pathology. These lesions are AKA Cushing's ulcers due to Cushing's classic treatise [1].

17% of SUs produce clinically significant hemorrhage. CNS risk factors include intracranial pathology:

brain injury (especially Glasgow Coma scale score < 9), brain tumors, intracerebral hemorrhage, SIADH, CNS infection, ischemic stroke, as well as spinal cord injury. The odds are increased with the coexistence of extra-CNS risk factors including long-term use of steroids (usually>3 weeks), burns>25% of body surface area, hypotension, respiratory failure, coagulopathies, renal or hepatic failure, and sepsis.

The pathogenesis of SUs is incompletely understood but probably results from an imbalance of destructive factors (acid, pepsin & bile) relative to protective factors (mucosal blood flow, mucus-bicarbonate layer, endothelial cell replenishment & prostaglandins).

CNS pathology, especially that involving the diencephalon or brainstem, can lead to a reduction of vagal output which leads to hypersecretion of gastric acid and pepsin. There is a peak in acid and pepsin production 3–5 days after CNS injury [2].

1)
Cushing H. Peptic Ulcers and the Interbrain. Surg Gynecol Obstet. 1932; 55:1–34
2)
Lu WY, Rhoney DH, Boling WB, et al. A Review of Stress Ulcer Prophylaxis in the Neurosurgical Intensive Care Unit. Neurosurgery. 1997; 41:416– 426