

26098 - THIOPENTONE BARBITURATE COMA: A REVIEW OF OUTCOMES AND COMPLICATIONS.

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INTRODUCTION: Barbiturate coma effectively lowers intracranial pressure (ICP) in head trauma or intracranial hemorrhage [1]. It remains second-line therapy in refractory intracranial hypertension [2] because of associated adverse effects [3]. There is relatively less published data on thiopentone coma compared to pentobarbital. We therefore investigated the clinical outcomes and adverse effects associated with thiopentone coma in patients with refractory intracranial hypertension.

METHODS: We conducted a retrospective cohort study of all patients receiving thiopentone for control of refractory intracranial hypertension in a neurosurgical intensive care unit (ICU) from January – December 2004. Outcome measures studied included ICP reduction, survival to ICU and hospital discharge, Glasgow Coma Scale (GCS) at ICU and hospital discharge, Glasgow Outcome Scale (GOS) 6 months post-injury, and associated adverse effects (hypotension, ileus, infection, renal dysfunction and electrolyte abnormalities).

RESULTS: 28 patients were studied. All values are expressed as median (IQR) or mean \pm SD. Post-resuscitation GCS at hospital admission was 9 (4-11) and the APACHE II score at ICU admission was 24 ± 4 . ICP prior to initiating barbiturate coma was 41 ± 16 mmHg. Duration of thiopentone therapy was 51 ± 34 hours, and maximum infusion rate was 294 ± 120 mg/h. Control of ICP to <25 mmHg was achieved within 36 hours in 22 patients (79%). All but 1 of the non-responders died in ICU. Sixteen (57%) patients survived to ICU discharge and 15 (54%) survived to hospital discharge. GCS at ICU and hospital discharge was 10 (9-11) and 11 (10.5-15) respectively. GOS at 6 months amongst survivors was 4 (3-5). Twenty-seven (96%) received a norepinephrine infusion (maximum dose 0.19 ± 0.23 mcg/kg/min). Pneumonia occurred in 11 patients (39%) following institution of thiopental coma. Hypokalemia (2.1 ± 0.6 mmol/L) occurred in 20 patients (71%), and was associated with ventricular bigeminy in 1 patient. Ten (36%) patients had rebound hyperkalemia (5.7 ± 0.8) on weaning of thiopentone infusion; non-fatal ventricular tachycardia occurred in 1 patient. Renal dysfunction (none requiring dialysis) occurred in 4 patients. Of the 14 patients who received enteral feeding during barbiturate coma, intolerance was observed in 3 (21%).

DISCUSSION: Survival in our series compares favorably to earlier reports [2,5], with relatively good outcome amongst survivors. Associated pneumonia has traditionally been a concern; however our observed rate was comparable to that seen in head-injured patients [4]. Our results suggest that judicious application of thiopentone coma in refractory intracranial hypertension is useful and may not necessarily be associated with a high incidence of adverse effects.

REFERENCES:

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