Paracetamol

Paracetamol (acetaminophen) is generally not considered an NSAID because it has only little anti-inflammatory activity. It treats pain mainly by blocking COX-2 mostly in the central nervous system, but not much in the rest of the body.

- Quadroiliac Plane Block for Analgesia in Pediatric Inguinal Hernia: A Case Report
- Perspectives on Analgesia for Craniotomy: A Survey of Anesthetic Practices
- Sacral Erector Spinae Plane Block for Hypospadias Surgery in a Patient With Denys-Drash Syndrome: A Case Report
- Liposomal Bupivacaine in Single-Injection Quadratus Lumborum Block for Pediatric Kidney Transplant: Case Report of a Novel Application
- Regional anesthesia in colorectal laparoscopy: a retrospective comparison of quadratus lumborum and TAP blocks
- Effect of Dinalbuphine sebacate on postoperative multimodal analgesic strategy in video-assisted thoracoscopic surgery: a double-blind randomized controlled trial
- Optimal Analgesic Volume for Popliteal Plexus Block After Total Knee Arthroplasty: A Blinded RCT Protocol
- Does Ultrasound-Guided Quadratus Lumborum Block Improve Pain after Hysterectomy

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Analgesia data from 173 patients in French, Canadian, American, and Australian and New Zealand ICUs suggest that acetaminophen/paracetamol is the most common first-line analgesic (49.1% of patients) [1].

Paracetamol, an antipyretic frequently employed in patients with cerebral damage, may cause hypotension.

Picetti et al., evaluated the cerebral and hemodynamic effects of intravenous (IV) paracetamol for the control of fever in Neuro-Intensive Care Unit (NICU) patients.

This prospective observational study in which they enrolled 32 NICU patients: Subarachnoid Hemorrhage (SAH, n = 18), Traumatic Brain Injury (TBI, n = 10), Intracerebral Hemorrhage (ICH, n = 2) and Acute Ischemic Stroke (AIS, n = 2).

The administration of paracetamol resulted in a decrease of core body temperature (Tc) (p = 0,0001), mean arterial pressure (MAP) (p = 0,0006), cerebral perfusion pressure (CPP) (p = 0,0033), and jugular venous oxygen saturation (SjVO2) (p = 0.0193), and in an increase of arteriojugular venous difference of oxygen (AVDO2) (p = 0.0012). The proportion of patients who had an infusion of norepinephrine increased from 47 % to 75 % (p = 0.0039 McNemar Test). When intracranial pressure (ICP) at the start of paracetamol infusion (t-0) was compared with the measurement of ICP after 2 h, a significant correlation was observed (r = 0.669, p = 0.0002). This marked and significant correlation can be explained by the fact that for the higher levels of ICP assessed at t-0 (greater than 15 mmHg), they observed a marked reduction of ICP concomitant with the decrease of Tc. No problems related to norepinephrine administration and/or increase in dosage were observed.

Paracetamol administration is effective but exposes patients to hypotensive episodes that must be recognized and treated expeditiously to prevent further damage to the injured brain [2].

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Zeiler FA, AlSubaie F, Zeiler K, Teitelbaum J, Bernard F, Skrobik Y. Analgesia in Neurocritical Care: An International Survey and Practice Audit. Crit Care Med. 2016 Mar 15. [Epub ahead of print] PubMed PMID: 26983164.
2)
Picetti E, De Angelis A, Villani F, Antonini MV, Rossi I, Servadei F, Caspani ML. Intravenous paracetamol for fever control in acute brain injury patients: cerebral and hemodynamic effects. Acta Neurochir (Wien). 2014 Oct;156(10):1953-9. doi: 10.1007/s00701-014-2129-2. Epub 2014 May 17. PubMed PMID: 24838770.