in a prospective, randomized study. Induction with propofol, 2-2.5 mg/kg IV, and maintained with sevoflurane 1-2 % end-tidal minimum alveolar concentration (MAC), in a 1:1 oxygen:air mixture, in combination with a fentanyl 5 μg/kg. Intravenous rocuronium 1 mg /kg, followed by 0.07 mg/kg. Anesthesia was maintained by a sevoflurane volatile anesthetic together with fentanyl 0.1 to 0.2 μg/kg every 30 minutes. In Group N (Neostigmine)(n=15) patients received standard intravenous neostigmine 0.07 mg/kg and atropine 0.02 mg/kg doses before extubation. In Group N+S(Neostigmine+Sugammadex) (n=15) patients received an intravenous bolus dose of 1 mg/ kg of sugammadex five minutes after standard reversal dose. In both groups, reversal agents of blokade was provided if there is at least one twitch at TOF monitor. The time to recovery of the train-of-four (TOF) ratio to 0.9 was recorded.The primary efficacy variable was the time from the start of administration of neostigmine/atropine or sugammadex to recovery of the T4/T1 ratio to 0.7 and 0.9 Extubation times, recovery times, after extubation tidal volumes, respiratory rate, arterial oxygen saturation (SaO2) values were collected at 5, 10, and 30 min.

Results and discussion: Age, sex, gender, operation types and duration of operations were not different between groups (p>0.05).The time to achieve TOF ratios of 0.7 and 0.9 were significantly shorter in Group N+S in comparison toGroup N (p=0.0001). In Group N, the extubation time (13.8 ± 7.3 minute) was longer than Group N+S (4.8 ±3.3 min) (p=0.0001). In Group N, the recovery time (Aldrete score >9) after reversal of neuromuscular blokade (36.7 ± 11.2 minute) was longer than Group N+S (13.2 ±5.4 min) (p=0.0001). In Group N, tidal volumes after extubation was smaller (178.4 ± 33.8 ml) in comparison to Group 2 (212.4 ± 31.8 ml) (p=0.0001).

Conclusion(s): Sugammadex can be safely used after administration of neostigmine and atropine atropine for reversal of residual NMB without any significant adverse events.

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9AP5-1

The effect of different storage temperature on the pharmacodynamic dose-response of cisatracurium besylate

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Background and Goal of Study: To evaluate the pharmacodynamic dose-response of cisatracurium besylate stored under refrigeration or at room temperature when given as abolus under total IV anesthesia with propofol.

Materials and methods: 120 ASA I or II patients aged 18-65 yr undergoing elective procedures were randomly divided into three groups (n=40 each): group 1 cisatracurium stored at 4-8°C; group 2 cisatracurium stored at room temperature for 30 days and group 3 cisatracurium stored at room temperature for 60 days. Anesthesia was induced with TCI of propofol (Cp 3μg/ml) and remifentanil (Ce 3-5 ng/ml). A bolus of cisatracurium 0.2mg/kg was given IV over 5-10 s as soon as the patients lost consciousness. Neuromuscular block was monitored with TOF-Watch SX (Oaganon, the Netherlands). Single stimulation (0.1 Hz) was applied to the ulnar nerve at wrist. The maximal degree of neuromuscular block, onset time, clinical duration and recovery index were recorded. The patients were intubated and mechanically ventilated when neuromuscular block reached the maximal degree. The intubation condition was evaluated.

Results and discussion: The maximum block was 100% in all patients except one in group 3 was 95%. There was no significant difference among groups with regard to the frequency of excellent or good intubation condition. Compared with group 1 and 2, the onset time was significantly longer, clinical duration and 75% recovery time were significantly shorter in group 3, while recovery index varied no significance.

Conclusion(s): Compared with that stored under refrigeration, the onset time was significantly longer, clinical duration and 75% recovery time were significantly shorter when cisatracurium besylate was stored at room temperature for 60 days, recovery index was independent of storage temperature.

9AP5-2

Alanine minimises liver injury after ischemia reperfusion: influence of adenosine nucleotides

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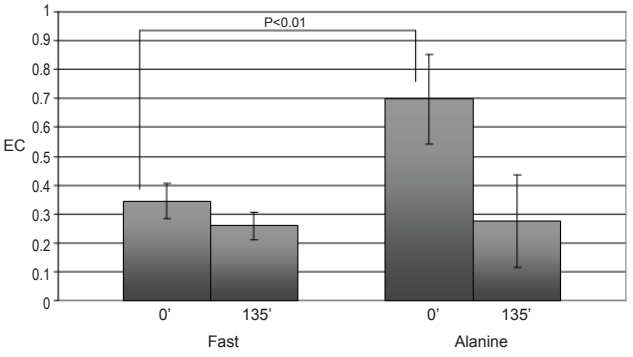
Background and Goal of Study: The pre-existing nutritional status of the liver, *i.e.* fasting, is known to contribute to the extent of tissue injury and primary non-function after different insults (1, 2). Different exogenous substrates have been evaluated to provide energy to livers. The aim of this study was to determine the role of alanine (Al), the most important amino acid precursor of glucose, on hepatic injury after ischemia-reperfusion (IR) in *ex vivo* perfused rat liver.

Materials and methods: After University Animal Care Committee approval, female Wistar rats were fasted for 18 hours with free access to tap water. Animals were anaesthetised**,** the portal vein cannulated, the liver removed and immediately perfused in a closed *ex vivo* system with HBSS supplemented with insulin, HEPES and O_2 at 37°C at a flow rate of 5 ml/min. The experiment consists of three phases: perfusion for 15 min, ischemia for 60 min, and reperfusion during 60 min. Animals were randomly divided into two groups: control in which rat livers were perfused with the HBSS enriched solution containing 1 g/l glucose and Al group in which 25 g/l Al (concentration based on previous results) was added to perfusate (without glucose) from the start of the experiment (n = 10 in each group). Glucose, lactate, potassium, and enzymes were analysed in perfusate samples at different time-points. The energy charge (EC=[ATP]+1:2[ADP]/[ATP]+[ADP]+[AMP]) in hepatocytes was determined in tissue biopsies at 0 and 135 min using HPLC technique as described elsewhere. Mean ± SD. Student's *t* test.

Results and discussion: Al minimises enzymes release after IR. EC in tissue was higher in Al treated-livers when compared to control group at start of experiment (Fig 1). It has been shown that Al improves the recovery of ATP through pyruvate and the Krebs cycle and the gluconeogenic capacity of livers from fasted animals (2, 3).



[Figure 1]

Conclusion(s): The infusion of Al might protect against liver IR injury in fasting patient by increasing ATP level.

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