

to 60 years of age, ASA I and II, undergoing anterior cervical spine surgery were selected. Patients with comorbidities, difficult airway, and previous hoarseness were excluded. Study conducted over 1 year primarily to observe changes in ETT cuff pressure and secondarily to observe peak/mean airway pressures changes and postoperative hoarseness or sore throat. Sample size was calculated using effect sizes from previous studies and power/sample size calculation software with 80.0% statistical power type II error = 0.20 and 5% type I error probability $\alpha = 0.05$, Alpha error = 0.05. After anesthesia induction, patients were intubated with flexometallic ETT and cuff was inflated to 20 cmH₂O. Intraoperative cuff pressure changes were monitored every 10 minutes until extubation, including application of manual and self-retaining retractors. Peak and mean airway pressures were also recorded. Postoperatively, presence and severity of sore throat and hoarseness were recorded. Statistical analysis performed using ANOVA for intragroup data and Wilcoxon's signed rank test for hoarseness and sorethroat with SPSS 11.5.

Results: Cuff pressure measured at various time points after application of retractor was significantly higher compared with cuff pressure at induction ($p < 0.05$). No significant rise was recorded in airway pressures. Hoarseness and sorethroat at 1, 4, and 24 hours improved significantly ($p < 0.001$).

Conclusion: Application of retractors during anterior cervical spine surgeries causes significant rise in ETT cuff pressure leading to hoarseness and sorethroat immediately postoperatively.

Keywords: ETT, cuff pressure, hoarseness

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A039 Enzymatic Evaluation of General Anesthesia-Induced Neurotoxicity in Aneurysmal Subarachnoid Hemorrhage Patients

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Introduction: Animal and observational human studies suggest that general anesthetics cause neurotoxic changes in developing brain. No direct evidence of neurotoxicity in adults exposed to general anesthetics is available. Proposed mechanism of neurotoxicity is neuroapoptosis. Caspase-3 is the biomarker.

Aim: Estimation of changes in cerebrospinal fluid (CSF) and serum caspase-3 in subarachnoid hemorrhage (SAH) patients exposed to general anesthetics.

Methodology/Description: Prospective randomized double-blind study—from January 2016 to May 2017. Thirty-two patients with good grade aneurysm—clipping and endovascular coiling were recruited and randomized to four groups for the maintenance of anesthesia—propofol, isoflurane, sevoflurane, and desflurane. Lumbar drain preoperatively was inserted for CSF sampling. Standard monitors and induction techniques were used. Concentrations of anesthetic agents were compared using state entropy—maintained between 40 and 60. CSF and serum samples collected at baseline, 1 hour after exposure to anesthesia, after the cessation of anesthesia. Samples were centrifuged, stored and later analyzed using ELISA.

Results: CSF caspase-3 levels significantly decreased from baseline to 1 hour after exposure to anesthetic agents, which then reached to baseline levels after the cessation of anesthesia. These changes were statistically significant and comparable between all the groups. Serum caspase-3 levels significantly increased from baseline to 1 hour after exposure to anesthetic agents, which then reached to baseline levels after the cessation of anesthesia. These changes were statistically significant and comparable between all the groups.

Conclusion: Intravenous and inhalational anesthetic agents have same effect on the serum and CSF caspase-3 levels. The reduction in CSF caspase-3 levels suggests the role of anesthesia in reduction of apoptotic mechanism. Increase in serum caspase-3 levels after exposure may be due to caspase-dependent apoptosis in peripheral mononuclear cells secondary to inflammatory stress response.

Keywords: CSF, SAH, caspase-3

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A040 Effect of Anesthetic Agents on Cognitive Function and Peripheral Inflammatory Biomarkers in Young Patients Undergoing Surgery for Spine Disorders

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Introduction: Exposure to anesthesia has been postulated to affect the cognitive function by inciting central nervous system (CNS) inflammation. So, we planned to compare the pharmacological effect of propofol, desflurane, and sevoflurane on postoperative cognitive function and measure the change in concentration of serum S-100 β , interleukin 6 (IL-6), and tumor necrosis factor alpha (TNF- α) to look for the contribution of systemic inflammation.

Methodology/Description: A prospective, double-blind, randomized trial. Institutional ethics committee approval and consent from patient obtained. We enrolled 66