Abstract

Intramuscular dexmedetomidine 30 minutes before extubation to assess its effect on post-extubation haemodynamics: A double blind placebo controlled study

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Introduction: The effect of intramuscular dexmedetomidine given 30 minutes before extubation on post extubation haemodynamic is studied. Materials and Methods: 50 ASA grade 1 and 2 patients of either sex undergoing elective laminectomy were included in the study. the patients were divided into study group and control group. The study group received dexmedetomidine (2.5 mcg/kg i. m.) in 2.5 ml saline 30 min. before extubation and the control group received normal saline (placebo, i. m.) 2.5 ml. The HR, SBP, MAP, DBP were studied at 1 min., 5 min., 10 min., 15 min., 30 min. post extubation in the study and the control group. Result: The heart rate measured at 1 min., 5 min., 10 min., 15 min., 30 min. post extubation was significantly lower in the study group compared to the control group (P < 0.05) he SBP at 1 min., 15 min., 30 min. post extubation was significantly lower in the study group than in the control group (P < 0.05). The MAP at 1 min., 10 min., 15 min., 30 min. post extubation was significantly lower in the study group than in the control group (P < 0.05). The DBP at 15 min., 30 min. post extubation was significantly lower in the study group than in the control group (P < 0.05). Conclusion: Dexmedetomidine provides greater haemodynamic stability in the post extubation period as compared to the placebo.

Dynamic cerebral autoregulation following loading dose of Dexmedetomidine; A transcranial doppler study

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Background: Dexmedetomidine has been widely used in neuroanaesthesia and critical care settings. The effects of Dexmedetomidine on cerebral vascular autoregulation and haemodynamic has not been studies in our population. This study is aimed to address this issue. Materials and Methods: Fifteen patients undergoing spinal surgery intracranial pathology were included in this study. Transient hyperemic response testing was done bilaterally with transcranial color Doppler. Dynamic autoregulation was assessed with Transient hyperemic response ratio (THRR) and Strength of autoregulation (SA) at baseline and after infusion of Inj. Dexmedetomidine 1 mcg/Kg over 10 minutes. Results: In both hemispheres of the patients without intracranial pathology, significant reduction in THRR and SA values were observed (P < 0.001) after administration of loading dose of Dexmedetomidine. Conclusion: Our study shows that the loading dose of Dexmedetomidine impairs cerebral autoregulation. Caution should be exercised in using this drug in patients with impaired autoregulation.

Haemodynamic changes in cervical myelopathy patients undergoing surgery in prone position - A pilot study

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Background: Positioning a patient in prone position under anaesthesia significantly alters cardiovascular physiology. Cervical myelopathy patients are known to have autonomic dysfunction. Such patients when positioned in prone position under anaesthesia carry a higher risk of developing haemodynamic changes and this can compromise spinal cord perfusion. Materials and Methods: This prospective observational study was conducted on 15 patients with cervical myelopathy who underwent surgery in prone position. The haemodynamic parameters were recorded at baseline, post induction, post intubation, prior to prone position, post prone position, and every five minutes thereafter up to 20 mins. The haemodynamic parameters that were recorded using the NICOM monitor: HR - Heart rate (beats/min), NIBP - non invasive blood pressure (mmHg), MAP - mean arterial pressure (mmHg), CO - cardiac output (l/min), CI - cardiac index (l/min/m²), SV - Stroke volume (ml/beat), SVV -stroke volume variability (%), TPR - total peripheral resistance (dynes. sec/cm²). Results: We found that MAP, HR, SV, TPR significantly decreased from baseline values to 20 min post positioning. Changes in CO, CI and SVV were not statistically significant. Conclusions: Hypotension can occur commonly in cervical myelopathy patients after prone positioning. We conclude that the decrease in MAP is due to decrease in TPR, SV. But the cardiac output is maintained. We can use of vasopressors or inotropes to treat such hypotension rather than administering IV fluids.