Editorial

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Dexmedetomidine in anaesthesia practice: A wonder drug?

Dexmedetomidine has become of the frequently used drugs in anaesthetic armamentarium, along with routine anaesthetic drugs, due to its haemodynamic, sedative, anxiolytic, analgesic, neuroprotective and anaesthetic sparing effects. Other claimed advantages include minimal respiratory depression with cardioprotection, neuroprotection and renoprotection, thus making it useful at various situations including offsite procedures.[1] α-1 to α-2 ratio of 1:1600 makes it a highly selective α-2 agonist compared to clonidine, thus reducing the unwanted side effects involving α-1 receptors.

High selectivity of dexmedetomidine to α-2A receptors (which mediate analgesia and sedation) has been exploited by various authors in regional anaesthesia practice.

Due to its central sympatholytic effect, dexmedetomidine is useful in blunting haemodynamic responses in perioperative period. It is successfully used in intravenous doses varying from 0.25 to 1 mcg/kg for attenuating intubation response.[2-5] Optimal dose for attenuating pressor response seems to be 1 mcg/kg with lesser doses not being effective.[5] Infusion continued into the postoperative period has been associated with reduced haemodynamic fluctuations and decrease in plasma catecholamines.[3] Doses in the range of 0.5 mcg/kg not only blunted the extubation response but also reduced the emergence reaction and analgesic requirement to extubation following rhinoplasty and neurosurgery. There was no delay in recovery or prolonged sedation when boluses were administered before induction or before extubation. Similar was the observation when duration of infusion was within 2 hrs.[6,7] Bradycardia and hypotension are the major side effects observed following dexmedetomidine infusion. Bradycardia is attributed to reflex response for transient hypertension during initial part of infusion. Subsequent decrease in heart rate is due to decrease in central sympathetic outflow. Hypotension is attributed to decreased central sympathetic outflow. Transient hypertensive response has been observed with higher doses (1–4 mcg/kg). This is attributed to initial stimulation of α-2B receptors present in vascular smooth muscles. This hypertensive episode settles once there is decrease in central sympathetic outflow. Mason *et al.* observed increased incidence of hypertension in children less than 1 year, undergoing magnetic resonance imaging (MRI) under dexmedetomidine sedation, and observed that younger children and multiple bolus therapies are highly significant predictors of the occurrence of hypertension.[8]

The highly selective effect of dexmedetomidine promotes its use for intensive care unit (ICU) sedation. Reduced ICU stay, decreased duration of ventilation, haemodynamic stability and reduced agitation are claimed advantages. However, a meta-analysis by Tan[9] did not find any significant advantage with regards to duration of mechanical ventilation. It was further suggested that the risk of bradycardia was significantly higher when both a loading dose and high maintenance doses (more than 0.7 mcg/kg/hr) were used. Jones *et al.* retrospectively analysed different doses of dexmedetomidine for ICU sedation and noted that doses greater than 0.7 mcg/kg/hr did not enhance sedation or incidence of side effects.[10] This is further endorsed by the meta-analysis by Tan where it was observed that incidence of bradycardia requiring intervention increased in studies that used both a loading dose and maintenance doses of dexmedetomidine in excess of 0.7 mcg/kg/hr.[9]

By virtue of its effect on spinal α-2 receptors, dexmedetomidine mediates its analgesic effects. Dexmedetomidine has been found to prolong analgesia

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when used as an adjuvant to local anaesthetics for subarachnoid block, epidural and caudal epidural blocks. However, there is no proper consensus regarding the dose of drug to be used for neuraxial blocks. Doses varying from 3 to 15 mcg have been used as adjuvant to bupivacaine for spinal anaesthesia. There has been dose-dependant prolongation of analgesia. However, the incidence of side effects due to dexmedetomidine alone is difficult to assess as different doses of bupivacaine were used in different studies.[11-13] Addition of dexmedetomidine 2 mcg/kg to caudal bupivacaine 0.25% at 1 ml/kg significantly promoted analgesia after anaesthetic recovery in children aged 6 months to 6 years, without increasing the incidence of side effects.[14]

Animal studies have shown dose-dependant reduction in minimum alveolar concentration (MAC) of isoflurane following epidural administration of dexmedetomidine in dogs at 2 hrs and 4.5 hrs.[15] However, Konacki *et al.*, in their study on rabbits, noted no sensory or motor effects of epidural dexmedetomidine when administered without local anaesthetic, but for its potential to neurotoxicity. Following epidural anaesthesia in rabbits, they found evidence of demyelinisation of the oligodendrocytes in the white matter in dexmedetomidine group which was significantly higher than when only lignocaine was used.[16] In a recent study conducted on patients undergoing thoracic surgery under combined epidural and general anaesthesia, dexmedetomidine administered via epidural route provided good postoperative analgesia with reduction in anaesthetic requirements.[17] Dexmedetomidine has been successfully used in children as adjuvant in caudal epidural. 1–2 mcg/kg dexmedetomidine used along with bupivacaine provided prolonged analgesia without significant side effects.[18,19] However, its superiority against clonidine is yet to be fully established.[19]

Dexmedetomidine is finding its way into every segment of anaesthesia practice, and barring few animal study reports, no significant side effects so far being described, this drug may stay put firmly in anaesthetist's armamentarium.

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