DEXMEDETOMIDINE AND PROFOFOL ADMINISTRATION IN NEUROLOGICALLY IMPAIRED CHILDREN UNDERGOING EEG NEURODIAGNOSTIC STUDIES

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Introduction: Neurologically impaired children often require electroencephalograms (EEG) for diagnosis of epilepsy and monitoring drug therapy. Historically a subset of this patient population presents a major behavioral challenge due to their inability to communicate and cooperate with instructions. At our institution, these children may receive oral midazolam premedication (0.5 mg/kg), and are induced with sevoflurane and maintained on propofol for these studies. Agitation during induction of anesthesia is often encountered, sometimes even after midazolam. Propofol, benzodiazepines, barbiturates and many inhalation agents suppress EEG signals, rendering test results suspect. Dexmedetomidine (DEX) is a selective, alpha-2 adrenergic receptor agonist with sedative, antipruritic, antiemetic, analgesic and sympatholytic properties. It has been used as a treatment for opioid withdrawal, weaning patients from ventilatory support, as pre-anesthetic medication, and as a maintenance agent for “awake” craniotomies, cardiac surgery, radiological studies and neurophysiologic tests. We present a review of our experience with DEX and propofol in a group of neurologically impaired children undergoing EEG.

Methods: We retrospectively reviewed the records of patients who received both DEX and propofol in a sequential fashion during an EEG.

<table>
<thead>
<tr>
<th>Pt #</th>
<th>AGE (yr)</th>
<th>Wt (kg)</th>
<th>DIAGNOSIS</th>
<th>PREMED</th>
<th>INDUCTION</th>
<th>DEX (mcg/kg/hr)</th>
<th>PROPOFOL (mcg/kg/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>9</td>
<td>21.3</td>
<td>Autism</td>
<td>DEX 80 mcg PO</td>
<td>70% N2O</td>
<td>0.5-0.7</td>
<td>150-250</td>
</tr>
<tr>
<td>2</td>
<td>14</td>
<td>39</td>
<td>Cerebral Palsy</td>
<td>None</td>
<td>70% N2O/ Sevo</td>
<td>0.5-0.7</td>
<td>100-150</td>
</tr>
<tr>
<td>3</td>
<td>19</td>
<td>26.2</td>
<td>Convulsions/ Chondrodystrophy</td>
<td>None</td>
<td>30% N2O/ DEX 25mcg IV ~ 10 min</td>
<td>0.7</td>
<td>150</td>
</tr>
</tbody>
</table>

Results: The records of three patients who had received DEX and propofol sequentially were reviewed. All were neurologically impaired and would not cooperate for the EEG without sedation. One patient was premedicated with DEX, all received N2O for induction only, and one patient also received Sevoflurane for induction. DEX was the initial maintenance sedative for all patients. After a period of EEG recording, DEX was stopped and anesthesia was maintained with propofol. Vital signs were stable and within normal limits throughout the procedures. Figure 1 shows the EEG tracings for each patient comparing DEX to propofol. Epileptiform activity can be seen in each patient after stabilization on the DEX infusion. When the propofol was administered, all patients demonstrated an overall EEG slowing to 1-2 Hz, giving an appearance of an alpha coma. The epileptic activity disappeared in patients 1 and 3 when propofol was administered.

Conclusion: Tobias et al have shown DEX to be efficacious in the children undergoing EEG studies. Our review of the records from patients sequentially receiving DEX and then propofol illustrates significant propofol attenuation of EEG signals. The time, financial cost and anesthetic risk of these studies is not insignificant. The suppression of epileptiform activity by propofol may mislead clinicians and could alter therapy, thereby subjecting the child to untoward events and possible brain damage. It should be noted that DEX has been demonstrated to have proconvulsant properties when administered with pentylentetrazol or enflurane in an experimental rat model, and anticonvulsant effects in association with bupivacaine induced seizures. DEX may represent a sedative agent that produces minimal effects on epileptiform activity and therefore a better agent in this patient population. Further investigation of this drug is warranted.
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