Hemodynamic effects of dexmedetomidine during intra-operative electrocorticography for epilepsy surgery

The dominant challenge in epilepsy surgery is precise delineation of the epileptogenic zone in the brain. Noninvasive techniques such as video electroencephalography (EEG), single photon emission computerized tomography (SPECT), positron emission tomography (PET), magnetoencephalography (MEG), and high-resolution MRI all lack sufficient spatial resolution and sensitivity in many patients. Extraoperative seizure mapping with subdural and/or depth electrodes can be appropriate in many cases, but is not without significant drawbacks. Disadvantages include the requirement for more than one major intracranial operation, need for the patient to be reasonably compliant, insufficient seizure capture, and potential surgical complications including hemorrhage and infection. In addition, electrode coverage may not be adequate, and areas such as insular and frontobasal cortex can be technically challenging to access. Intraoperative electrocorticography (ECoG) avoids many of these disadvantages (notably aside from inadequate coverage), but at the expense of capturing only interictal EEG data. The quality of intraoperative EEG data, therefore, is of paramount importance during one-stage epilepsy surgeries in which ECoG is employed.

Dexmedetomidine is an intravenous alpha 2-adrenergic receptor agonist whose use in neurological surgery has blossomed over the past several years due to its sedation properties and desirable neurophysiological profile. This medication has been extensively studied in the context of various neurosurgical pathologies and treatment modalities including awake brain tumor surgery, functional MRI scanning, and endovascular aneurysm treatment. Its use has also been studied with electroconvulsive therapy, where postprocedural agitation was found to be improved but induced seizure duration not apparently altered.

The primary concerns regarding dexmedetomidine use in epilepsy surgery revolve around two issues, alteration of the EEG profile and hemodynamic effects, particularly bradycardia and hypotension. Dexmedetomidine does not seem to be a significant EEG confounder. Its intraoperative hemodynamic profile also seems to be safe, although this particular aspect has not previously been specifically addressed within the context of epilepsy surgery. The present study addresses this concern directly with a series of patients undergoing ECoG-guided anterior temporal lobectomy and amygdalohippocampectomy, the most common resective surgery for epilepsy in the adult population. The findings are that dexmedetomidine anesthesia does generate a measurable but clinically insignificant hemodynamic effect when used in this context, and that it does not otherwise adversely impact surgery. This study is a useful contribution to the body of data on which the practice of epilepsy surgery is based. Further studies such as this will allow epilepsy surgery to continue to become more safe and more efficacious.

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