The Effects of Ketamine on Dexmedetomidine-Induced Electrophysiologic Changes in Children

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Background

• Dexmedetomidine (DEX) is an alpha2-adrenergic agonist that is used for sedation and anesthesia in children.

• Adverse hemodynamic effects of DEX include bradycardia, hypertension and hypotension.

• We previously showed that DEX causes a reduction in heart rate and increase in blood pressure and significantly depresses sinus and AV nodal function in pediatric patients.1 We hypothesized that these sympatholytic EP effects of DEX might be antagonized by co-administration of ketamine (KET), which has sympathomimetic properties.2

• We therefore conducted this study in order to characterize the cardiac electrophysiologic (EP) effects of this clinically useful combination of anesthetic agents

Hypotheses

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Results

A significant increase in mean arterial pressure (MAP) was seen compared to baseline after loading of DEX (p < 0.001). This returned to baseline 5 minutes after co-administration of KET (p = 0.0043). The increase in MAP was accompanied by a significant decrease in heart rate after DEX (p < 0.001) followed by a return towards baseline after co-administration of KET (p= 0.0045).

Results (cont’d)

OT was prolonged after DEX and returned towards baseline after KET (p = 0.0043). AV nodal function showed a trend towards return to baseline function after KET, as measured by AV node effective refractory period (p = 0.069).

Methods

• Twenty-two children ages of 5 and 17 years undergoing EP study and ablation of supraventricular accessory pathways were enrolled.

• Propofol was titrated to maintain sedation and were kept constant beginning 15 minutes prior to study measurements.

• Hemodynamic and EP parameters were measured before and after a loading dose administration of DEX (1 mcg/kg IV over 10 minutes).

• At the completion of the loading dose of DEX (t=10 min) a continuous infusion of DEX 0.7 mcg/kg/hr was begun and KET 1mg/kg was given over 1 minute, followed by a continuous infusion of KET 1mg/kg/hr.

• After KET (t=15 min) hemodynamic and EP parameters were measured again (projected peak tissue concentration of both drugs.)

Conclusions

• We confirmed results from our prior study, i.e. that DEX increased MAP, reduced heart rate, and significantly depressed sinus and AV nodal function in pediatric patients.

• These changes were reversed or mitigated by the co-administration of KET.

Implications

The concurrent use of KET may reduce the undesirable hemodynamic and EP effects of DEX in children.

References


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