RAPID SEQUENCE INTUBATION

I have NO Financial Disclosures

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THE DISCUSSIONS FOR TODAY

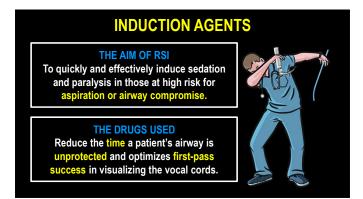
Ketamine / Ketofol Special Case Interesting Idea SUX v ROC!!! SCCM Guidelines

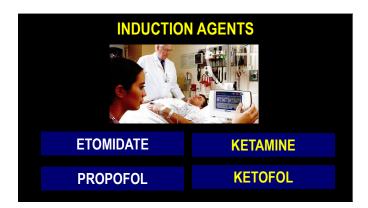


Delayed Sequence Oxygenation

"Expertise in Procedural Sedation and Analgesia is a Core Competency In Emergency Medicine Practice."

- ACEP Clinical Policy Guidelines

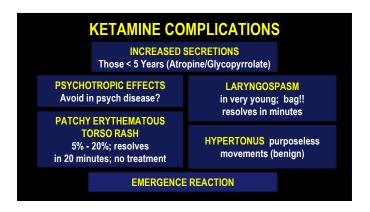






KETAMINE	
"SAFEST ANETHETIC AGENT IN THE WORLD" Used for Pediatric Procedural Sedation for Decades Used in the Most Rural Clinics	
SMART USE OF KETAMINE Dissociative Anesthetic Procedures with Greater Post - Procedural Pain Patients on Chronic Narcotics	

KETAMINE "Low Cost + Large Therapeutic Window"
MAINTAINS ALL VITALS & REFLEXES No Histamine Release - Best CV Stability of all drugs Allows Placement - into unobservable positions
ADDITIONAL CLINICAL EFFECTS May Raise BP & Pulse (weakly releases norepinephrine), Potent Bronchodilator, Intense Co-Analgesic, Sedative and Amnestic most effective with a low-dose opioid.



KET	TAMINE
EMERGENCE REACTION "the feared complication"	
PRESENTATION patients awaken feeling disconnected, fearful or anxious, and may report nightmares / hallucinations	
INCIDENCE 50% (adults) 10% (children)	

KETAMINE
EMERGENCE REACTION > 10 years, female gender, personality disorders, rapid administration, and recovery stimulation
PREDOMINANT SYMPTOMS 25% Recall Dreams, < 1/3 Found It Unpleasant
PREVENTION/TREATMENT Administer Ketamine: very slowly Recover Patient: dark room with minimal stimulation Benzodiazepines: give if symptoms develop





KETOFOL
KETAMINE Dissociative Sedative, Analgesic and Amnestic
PROPOFOL Sedative, Hypnotic, Anti-Emetic and Amnestic
SMART COMBINATION Opposing Hemodynamic & Respiratory Effects

KETOFOL				
TREAT PAIN FIRST Every Single Time	RECOVERY 15 min; 96% in 30 min			
DOSING 1:1 Ketamine:Propofol	VITAL SIGNS modest raise in BP & HR			
ALIQUOTS 1 – 3 mL Boluses	SUCCESS RATE > 96%			
Latest ACEP Clinical Policy Level B Recommendation Ketofol is safely given to CHILDREN & ADULTS.				

KETOFOL
BENEFITS Low Incidences of Hypotension, Vomiting, Aspiration, Bradycardia, or other Adverse Outcomes or Sequelae
COMPLICATIONS Mild Emergence - Treat with Midazolam Hypoxemia - Airway Alignment or Bagging Muscular Rigidity - No Intervention Required
THE FUTURE Ketamine:Propofol @ 1:2, 1:3 or 1:4 Increases Cardiorespiratory Stability and Relaxation Decreases Emergence Reaction

NOW, A SURPRISE

INTERESTING CASE A 28 y/o man w/o PMHx is BIB EMS. He was found, highly agitated and pulled a steak knife on responding officers. He is in 4-point restraints but is not resisting any longer. Vital Signs are BP 78/56 (MAP = 63), HR 82, RR 22, T 98.8 °F. The patient appears exhausted, dehydrated, and altered. EMS relates that police requested sedation upon their arrival. His BP does not respond to IVFs or Narcan. GCS is 10. Why is the patient not responding to your interventions?

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KETAN		N THE		

Be aware of catecholamine depletion in the critically ill.

Ketamine has a direct biphasic myocardial effect.

The first response is direct myocardial depression.

The second response is direct myocardial stimulation.

It is thought that direct sympathetic stimulation makes ketamine an attractive option for unstable patients

KETAMINE USE IN THE CRITICALLY ILL

However, there may not be a secondary response that will increase in myocardial stimulation and blood pressure.

Ketamine's intrinsic myocardial depression may predominate in those with massive stress-induced catecholamine depletion.

Ketamine's secondary effect of myocardial stimulation is dependent upon intrinsic beta-adrenergic tone.

Comparative Study > Acta Anaesthesiol Scand. 1996 Mar;40(3):338-41. doi: 10.1111/j.1399-6576.1996.tb04442.x.

In vitro myocardial depression by ketamine or thiopental is dependent on the underlying betaadrenergic tone

T A Thurston 1, B P Mathew

Affiliations + expand

PMID: 8721465 DOI: 10.1111/j.1399-6576.1996.tb04442.x

CATECHOLAMINE DEPLETION
Manifested by Anxiety, Somnolence or Psychotic states
SIMULANT USE
Methamphetamine and Cocaine
CONGESTIVE HEART FAILURE
Disturbed Cardiac Norepinephrine Homeostasis (Depletion)

CATECUOL AMINE DEDICTION

LEWY BODY DISEASES Dementia and Parkinsonism SEPSIS OR SHOCK

KETAMINE MECHANISM	OF AU	HUN

DISASSOCIATIVE ANESTHETIC

NMDA receptor complexes binding (neuro-inhibition) partial agonist opiate mu binding (sedation and comfort)

HEMODYNAMIC EFFECTS mediated through catecholamine release catecholamine-depleted patients may be unprotected from the unopposed direct myocardial depressant effects

CLINICAL KETAMINE IS RACEMIC!!
R-Ketamine is used in studies of depression. S-Ketamine increases cardiac output. The metabolite S-norketamine reduces cardiac excitation in a dose dependent manner.

BACK TO THE PATIENT

How would you treat this catecholamine-depleted patient with recalcitrant hypotension?

Use Phenylephrine (Neo synephrine®)

Hey!! What About Norepinephrine (Levophed®)

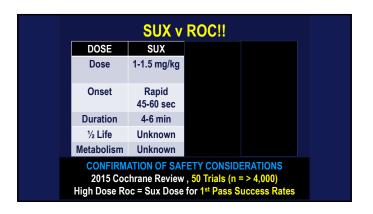
INTERESTING IDEA			
KETAMINE Provides a positive CV profile and has been suggested for hypotensive patients.	MIDAZOLAM Used with ketamine but may cause hypotension.		
Direct inhibitory CV effect and caution has been advised for hypotensive pts. Lower dose may improve CV profile during this critical period.	LIDOCAINE Enhances effect of thiopentone, propofol, midazolam and volatile agents w/o negative CV effects.		
HYPOTHESIS Use lidocaine with lower dose ketamine during induction in those with septic shock to provide a better hemodynamic profile.			

ketamine du endotrachea	February 2021 Double-Blinded of adding lidocain uring rapid sequen al intubation in pact c: A randomised co	nce atients with	HYPOTHESIS Septic shock pts may require intubation. Hypotension may seriously complicate anesthesia induction for patients in circulatory failure.
METHODS - Compares two RSI Protocols 1 mg/kg ketamine versus 0.5 mg/kg ketamine + 1 mg/kg lidocaine 0.05 mg/kg midazolam for each group (n=22)			PRIMARY OUTCOME = MAP OTHER OUTCOMES Post-induction Frequency of Hypotension, Heart Rate, and Cardiac Output

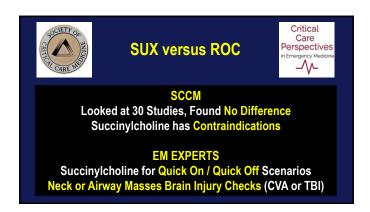
The benefit of adding lidocaine to ketamine during RSI endotracheal intubation in patients with septic shock: A randomized controlled trial. Ketamine / Ketamine P Values Lidocaine 73 +/-10.2 mm Hg 82.8 +/- mm Hg MAP @ 5 min < 0.001 Post-Intubation 17 patients (77%) < 0.001 1 patient (5%) Hypotension The ketamine-lidocaine group showed higher MAP in almost all readings after induction compared to the ketamine group. Cardiac output and heart rate were comparable between groups.

SUX versus ROC!!!					
	INTERNS IN THE ICU OUR PATIENT OF E PREFER (SCURLINGHUM) SUCCENTRUMENT (SCURLINGHUM) OUT OF THE ICU OUT OF				
	SUX) ROC! (WON! PUT ACTO) SLEP ALERANI!				

NEUROMUSCULAR BLOCKERS
SUCCINYLCHOLINE MECHANISM OF ACTION ACh analogue that stimulates ALL cholinergic receptors (parasympathetic and sympathetic), causing continuous stimulations, fasciculations, followed by muscular paralysis.
ROCURONIUM MECHANISM OF ACTION A nondepolarizing paralytic agent with competitive antagonism at the ACh receptor. It is a superior, less tachycardiac and histamine releasing alternative to pancuronium.



SUX vs ROC!!!				
	SUX	ROC		
Cost of Vial	\$40/200 mg	\$40/100 mg		
Storage	2 weeks	12 weeks		
Side Effects	Hyperkalemia Malignant Hyperthermia Fasciculations Bradycardia	Liver Toxicity		
Contraindications	Prior Stroke Baseline NM Disease Recent Burns CKD	Liver Disease		



SAFE APNEA TIME (SAT) SAT is the Time Required for a Patient to desaturate < 88% after paralysis ROCURONIUM SAFE APNEA TIME 40 seconds longer compared to succinylcholine PROPOSED MECHANISM Increased Muscle O₂ Consumption Fasciculations with Succinylcholine MEAN RECOVERY TIME POST-APNEIC HYPOXIA Rocuronium's time is significantly less

DELAYED SEQUENCE OXYGENATION Procedural Sedation for Pre-Oxygenation		
	TARGET PATIENTS Agitated, Delirious, Combative	
	SCCM Low Quality Conditional Recommendation that would Increase Safe Apnea Time	
	EM EXPERTS NRB first, dexmedetomidine (Precedex) or Ketamine and Prepare Your Team.	

