INTRODUCTION
Tetanus is a rare condition. However, it still remains a fatal disease caused by an infection with gram positive obligate anaerobic bacterium Clostridium tetani. In the US, 233 cases and 26 deaths were reported from 2001-2008. This may lead to unfamiliarity in anesthetic management for emergencies and is compounded by little evidence for anesthetic protocols. We are describing a child with generalized tetanus needing urgent intubation for respiratory compromise.

CASE REPORT
A 6 y/o, 22 kg, unimmunized boy presented with intermittent spasms. Family reported trouble opening his mouth, ‘funny’ facial movements and whole body arching. Upon arrival, clinical diagnosis of tetanus was made with neck stiffness, lockjaw (trismus), arching of trunk and head with spasms (opisthotonus) and grimacing (risus sardonicus). Therapy was initiated with intramuscular (IM) immunoglobulin and tetanus/diphtheria vaccine, intravenous Metronidazole and intravenous lorazepam. No obvious site of injury found upon exam.

Due to desaturation with spasms, patient was transferred to the PICU to escalate sedation. Patient’s spasms continued to increase in frequency despite therapy compromising airway sufficiency. The anesthesiology team was consulted to help with an urgent intubation.

PREINDUCTION CONSIDERATIONS
• Lock Jaw can be triggered with any stimulation
• Aspiration risk due to pharyngeal spasm and increased intra-abdominal pressure
• Hypoxemia due to chest wall rigidity causing a restrictive defect
• Hyperkalemic arrest with Succinylcholine possible due to muscle breakdown with spasms
• Uncertainty about drug safety due to lack of studies
• Autonomic dysfunction with advanced disease

INDUCTION/INTUBATION
Patient was taken to the operating room. ENT surgery was on stand by. An iv-induction was performed with fentanyl, propofol and rocuronium. Easy bag-mask ventilation was maintained. Direct laryngoscopy showed grade 1 view, 5.0 cuffed ETT passed easily and maintained on sevoflurane/oxygen/air.

PICU COURSE
In the PICU, management continued with midazolam and fentanyl drip. Break through spasms were treated with boluses of benzodiazepines and metronidazole and IM immunoglobulin continued. Daily lightening from sedation showed continued spasms. Tracheostomy placed uneventfully for long-term care. A source for infection was never found.

DISCUSSION
Tetanus is an infectious disease caused by tetanospasmin and tetanolysin produced by Clostridium tetani. Tetanolysin locally damages tissue and creates an anaerobic environment required for bacterial growth. Tetanospasmin may spread by intra-axonal and retrograde transport to the cell body of motor, sensory and autonomic neurons. This will lead to irreversible binding of pre-synaptic membrane proteins and inhibit the release of glycine and gamma-aminobutyric acid (GABA), both essential for inhibitory control of motor neurons leading to muscle rigidity and spasms. Benzodiazepines enhance GABA agonism in the postsynaptic membranes and hence are the first line treatment for spasms. Propofol works by potentiation of the post-synaptic GABA receptor. The response to non-depolarizing paralytics seems to be normal in tetanus. Even though depolarizing paralytics are considered safe, the concern for hyperkalemic arrest in advanced tetanus still remains. Sevoflurane is thought to enhance the activity of postsynaptic inhibitory receptors while inhibiting excitatory sympathetic channel activity. Even though it seems safe, studies are lacking. Trismus is manageable with paralytics. Risk for aspiration should still be considered given pharyngeal spasms.

1 Center for Disease Control and Prevention, 2014