



Case Report

Septal Deviation Correction (Rhinoplasty) Under General Anaesthesia in a Patient Taking High-Dose Creatine

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Abstract

We present the case of a man taking high-dose creatine who underwent correction of a deviated nasal septum (rhinoplasty) under general anaesthesia and was discharged home the following day.

Knowledge

Mechanism of Action of Creatine in Skeletal Muscle

Creatine supports rapid ATP regeneration during high-intensity muscle activity.

1. Uptake and storage

- Creatine enters skeletal muscle through the creatine transporter.
- It is phosphorylated by creatine kinase to form phosphocreatine.

2. ATP buffering

- During contraction, ATP is converted to ADP.
- Phosphocreatine donates its phosphate group to ADP:



- This reaction rapidly restores ATP, particularly during the first seconds of intense activity.

3. Energy shuttle

- The creatine–phosphocreatine system transports high-energy phosphate between mitochondria, where ATP is produced, and the myofibrils, where ATP is consumed.

4. Additional effects

- Increased intracellular creatine may promote water retention within muscle cells and support training-related muscle protein synthesis and adaptation.
- Creatine does not directly activate or block acetylcholine receptors and has no established direct mechanism that alters neuromuscular blocker action.

The final additional effect may not be entirely accurate. This case report suggests that, in a patient taking a high dose of creatine and undergoing general anaesthesia, the supplement may shorten the duration of neuromuscular blockade.

Case Report

A 29-year-old man patient (179 cm, 82 kg, BMI: 25.06) was diagnosed with nasal septal deviation. At the preoperative examination, the patient was alert and oriented. ECG showed sinus rhythm at 65 beats per minute, with a normal blood pressure of 125/65 mmHg. He was taking 6 gr creatine.

Laboratory Tests

Hct: 39.3%; Hb: 13 g/dL; PLT: 198,000/ μ L; urea: 42 mg/dL; creatinine: 1.1 mg/dL; K+: 4.5 mEq/L; Na+: 139 mEq/L; CPK: 425 U/L [1].

General Anaesthesia

At the outset, 18-G and 20-G intravenous cannulas were placed in the left arm. Monitoring included blood pressure, SpO₂, BIS, ECG, neuromuscular transmission using train-of-four stimulation, and urine output.

Anaesthesia was Induced with the Following Drugs

Dexamethasone 8 mg, fentanyl 50 μ g, propofol (2-6 Isopropyl phenol) 1% 200 mg, rocuronium 40 mg I.V. Intubation of trachea with a 8.0 size endotracheal tube was facilitated with Mc Coy 4. Cormack – Lehane 2A.

Maintenance of anesthesia with Desflurane ET 6.0 MAC 0.9, Gas flow 7lt/min, O₂/air. FiO₂ 0.35 CMV: Vt :500ml, RR: 14/min PEEP 5 I: E ½. SPO₂: 99% and Remifentanyl 0.05-0.1 μ g/kg/min. Ondansetron 4 mg, fentanyl 250 μ g, paracetamol 1gr, morphine 4mg, omeprazole 40 mg and mefoxil 1gr given IV. We used warm Intravenous fluids R. Lactate to maintain normothermia. NMT monitoring used on orbicularis oculi muscle.

- 25 min post-intubation: Orbicularis oculi NMT showed TOF 4/4 (90%). Administered rocuronium 15 mg IV.
- 35 min post-intubation: NMT unchanged at TOF 4/4 (90%). Administered second dose of rocuronium 15 mg IV. Sugammadex 200 mg IV was given at the end of procedure and train of four 4/4 (90%).

Conclusion

This case report serves as an excellent catalyst for the scientific community. Reexamining creatine at the molecular level-especially with regard to the neuromuscular junction and its potential role in neuromuscular blockade—may open up new avenues in neurology and anesthesiology.

We can structure our argument around the following key points: Molecular Mechanisms to Be Examined: Synaptic energy load: Study of how phosphocreatine affects the reuptake and release of acetylcholine (ACh) in presynaptic vesicles.

Ion Channels: Investigation of any interaction between creatine and nicotinic acetylcholine receptors (nAChRs) or voltage-gated calcium channels.

Synaptic fatigue: Analysis of whether depletion of creatine stores mimics or enhances the effects of neuromuscular blockade.

References

1. NYSORA anaesthesia.