2 Thompson PD, Clarkson P, Karas RH. Statin-associated myopathy. *J Am Med Assoc* 2003; **289**: 1681–90
3 Naka T, Jones D, Baldwin I, *et al.* Myoglobin clearance by super high-flux hemofiltration in a case of severe rhabdomyolysis: a case report. *Crit Care* 2005; **9**: R90–5
4 Sorrentino SA, Kielstein JT, Lukasz A, *et al.* High permeability dialysis membrane allows effective removal of myoglobin in acute kidney injury resulting from rhabdomyolysis. *Crit Care Med* 2011; **39**: 184–6

doi:10.1093/bja/aes086

## Efficacy of rocuronium and sugammadex in a patient with dermatomyositis

Editor—We report a patient with dermatomyositis who showed a slow onset of action of rocuronium-induced neuromuscular block and a slow recovery of neuromuscular function with sugammadex. A 75-yr-old male patient with dermatomyositis was undergoing open reduction of an elbow fracture under general anaesthesia. Preoperative muscle testing showed mild-to-moderate weakness in his extremities. Anaesthesia was induced with fentanyl and propofol and after loss of consciousness, the left ulnar nerve was stimulated at the wrist by a 5 s, 50 Hz tetanic stimulus, followed by supramaximal and square-wave stimuli of 0.2 ms duration, delivered in a train-of-four (TOF) mode at 2 Hz every 15 s. Contraction of the ipsilateral adductor pollicis muscle was measured using an acceleromyograph (TOF-Watch SX; Organon, Dublin, Ireland). Immediately after obtaining baseline TOF responses, the patient received 0.6 mg $kg^{-1}$ rocuronium. Complete neuromuscular block was obtained in 315 s and the patient's trachea was intubated thereafter without any difficulty. Anaesthesia was maintained with 1% end-tidal sevoflurane, 0.2 $\mu$g $kg^{-1}$ $min^{-1}$ remifentanil, and fentanyl as required. Twenty-five minutes after rocuronium administration, the first twitch (T1) of the TOF spontaneously recovered to 10% of the control. At that time, 2 mg $kg^{-1}$ sugammadex was administered to antagonize rocuronium-induced neuromuscular block. Lag time to the beginning of abrupt increase in T1 was 120 s, while the time to reach a TOF ratio of 0.9 was 345 s after sugammadex administration. Recurarization and respiratory complications were not seen after operation.

Our patient seemed to show normal sensitivity to rocuronium-induced neuromuscular block. It is not likely that there were abnormalities in the properties and number of nicotinic acetylcholine receptors on the motor endplate. However, our patient showed a slower onset of action of rocuronium. Maximal block after 0.6 mg $kg^{-1}$ rocuronium is normally obtained around 100 s.[1] Considering the normal sensitivity to rocuronium, the slow onset of action of rocuronium was probably due to slow diffusion of rocuronium from the plasma to the neuromuscular junction. In dermatomyositis, there is perivascular inflammation and intramuscular blood vessels show endothelial hyperplasia, fibrin thrombi, and obliteration of capillaries, resulting in a reduction in capillary blood flow to the muscles.[2] Therefore, the slower delivery of rocuronium to the neuromuscular junction may result in slower onset of action of rocuronium in patients with dermatomyositis. This could also explain the longer reversal time. The time to recovery from rocuronium-induced neuromuscular block to a TOF ratio of 0.9 after administration of a sufficient dose of sugammadex is generally 1.1–1.3 min, regardless of the depth of neuromuscular block.[3 4] Sugammadex may be restricted to the intravascular space due to its low volume of distribution and act mainly in the plasma, resulting in a rapid decrease in plasma concentrations of free rocuronium, which induces rocuronium molecules to extensively diffuse from the neuromuscular junction into plasma, along the concentration gradient. This probably leads to a rapid dissociation of rocuronium from the nicotinic acetylcholine receptors and restoration of normal neuromuscular transmission. Therefore, the delivery rate of sugammadex to the peripheral muscles has profound effects on recovery of the TOF ratio to 0.9.[5] An adequate dose of sugammadex can completely restore neuromuscular function even in dermatomyositis patients. However, it is still recommended that these patients should be observed for much longer than is usual practice in patients with normal neuromuscular function.

## Declaration of interest

T.S. has received speaker fees from MSD Inc., Japan.

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1 Kitajima O, Suzuki T, Fukano N, Saeki S, Ogawa S, Noda Y. Onset of rocuronium-induced neuromuscular block evaluated subjectively and accerelomyographically at the masseter muscle. *J Anesth* 2011; **25**: 376–9
2 Dalakas MC, Hohlfeld R. Polymyositis and dermatomyositis. *Lancet* 2003; **362**: 971–82
3 Suy K, Morias K, Cammu G, *et al.* Effective reversal of moderate rocuronium- or vecuronium-induced neuromuscular block with sugammadex, a selective relaxant binding agent. *Anesthesiology* 2007; **106**: 283–8
4 Suzuki T, Kitajima O, Ueda K, Kondo Y, Kato J, Ogawa S. Reversibility of rocuronium-induced profound neuromuscular block with sugammadex in younger and older patients. *Br J Anaesth* 2011; **106**: 823–6
5 Yoshida F, Suzuki T, Kashiwai A, Furuya T, Konishi J, Ogawa S. Correlation between cardiac output and reversibility of rocuronium-induced moderate neuromuscular block with sugammadex. *Acta Anaesthesiol Scand* 2012; **56**: 83–7

doi:10.1093/bja/aes087