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Muscle weakness after administration of neuromuscular blocking agents: Do not immobilize the diaphragm unnecessarily

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To the Editor

Dr. Testelmans and colleagues (1) are commended on rekindling interest in muscle weakness following neuromuscular blocking agents (NBAs). Prior observations in patients given NBAs have revealed that muscle weakness after neuromuscular blockade can persist for >72 hrs (2). Importantly, even a minimal degree of NBA-evoked muscle weakness impairs upper airway and pulmonary function in a clinically relevant fashion (3).

Dr. Testelmans and colleagues (1) reported that diaphragmatic muscle force is significantly lower in mechanically ventilated rats after 24 hrs of rocuronium infusion than in animals that were not given NBAs. We believe that the differences between groups may be due to immobilization-induced atrophy of the diaphragm, which may occur in the rat within 24 hrs (4). Dr. Testelmans and colleagues did not find electromyographic activity in the control group of mechanically ventilated rats, which suggests that diaphragmatic activity was low in these animals. However, some diaphragmatic activity was likely present in these rats. Phasic respiratory activity of the phrenic nerve persists even in decerebrated and controlled ventilated animals (5), and intermittent diaphragmatic contractions during controlled ventilation have also been shown in human studies. Since a few muscle contractions a day may be sufficient to prevent a decrease in muscle force and cross-sectional area in denervated skeletal muscle in rats (6), it is not a surprise that the degree of immobilization-induced changes (decrease in muscle force and an up-regulation of the ubiquitin proteasome pathway) were more marked in rats having blocked diaphragmatic contraction by infusion of NBAs.

Recent data from animal studies suggest that controlled mechanical ventilation can cause ventilator-induced diaphragmatic dysfunction, which is attenuated during assisted modes of

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ventilation. The clinical relevance of ventilator-induced diaphragm dysfunction has been questioned, since patients often fail to wean for other reasons (e.g., ongoing parenchymal disease).

We believe that spontaneous breathing may have benefits in patients with acute respiratory failure, but these must be counterbalanced with the potential risk of high transpulmonary pressures that patients may generate. The data of Dr. Testelmans and colleagues support the view that every effort should be made to not immobilize the diaphragm unnecessarily during mechanical ventilation.

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