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Short communications

Propofol suppressed electromyographic fibrillation potentials in a patient with amyotrophic lateral sclerosis

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Amyotrophic lateral sclerosis (ALS) is a progressive degenerative disease of motor neurons with an incidence of approximately 1 to 2 per 100000 population [1]. Typically, the onset of the disease is heralded by both upper and lower motor neuron signs and symptoms, including distal limb weakness and spasticity of the legs, night cramps, and dysarthria or dysphagia [2]. Anesthesiologists should use sedation with caution for patients with ALS because of the risk of pulmonary aspiration [3]. Moreover, patients with ALS are at risk for succinylcholine-induced hyperkalemia and increased sensitivity to nondepolarizing muscle relaxants [4]. We report a case in which propofol silenced electromyographic fibrillation potentials in a patient with ALS who underwent widening of the tracheostomy orifice.

A 65-year-old, 60-kg woman with ALS was scheduled for widening of the tracheostomy orifice because of difficulty in inserting a tube for suction of sputum. She was diagnosed with ALS about 10 years ago and had been followed up in our hospital. Tracheostomy was carried out 6 years ago for the purpose of respiratory care because of dysfunction of the respiratory muscles. In a preoperative neurological assessment, motor nerve conduction studies and electromyography (EMG) of the lower extremities confirmed the advanced stage of ALS (Fig. 1). The only lesion in which significant waves could be detected by EMG was the sublingual muscles, and spontaneous potentials were demonstrated reproductively. She was not able to move her legs or arms by herself and had needed respiratory care for 6 years.

The patient and her family had been informed of the aim of the study, and their consents were obtained before the operation. She was not premedicated for the surgery, and controlled mechanical ventilation was started in the operating room. After placement of a peripheral intravenous catheter, electrocardiography (ECG), pulse oximetry, capnometry, and noninvasive blood pressure monitoring were performed. A coaxial needle electrode continuous electromyographic for monitoring was inserted percutaneously into the sublingual muscles deeply enough that motor unit potentials were displayed on the oscilloscope. Before induction of anesthetia, spontaneous potentials, referred to as "fibrillation potentials," were detected on the oscilloscope (Fig. 2a). Two hundred micrograms of fentanyl was injected intravenously, and then 50 mg of propofol was administered, followed by intravenous infusion of propofol 2 $mg \cdot kg^{-1} \cdot h^{-1}$. After the intravenous injection of 200 μg of fentanyl, the amplitude of the fibrillation potentials decreased by half (Fig. 2b) as preinduction but did not vanish completely. On the other hand, fibrillation potentials on the oscilloscope disappeared completely following administration of 50 mg of propofol (Fig. 2c). All operative procedures were accomplished uneventfully, and emergence from anesthesia was prompt after the infusion of propofol ceased.

In this patient with ALS, we were able to demonstrate that fibrillation potentials in the EMG recorded from sublingual muscles were suppressed completely after administration of propofol, but

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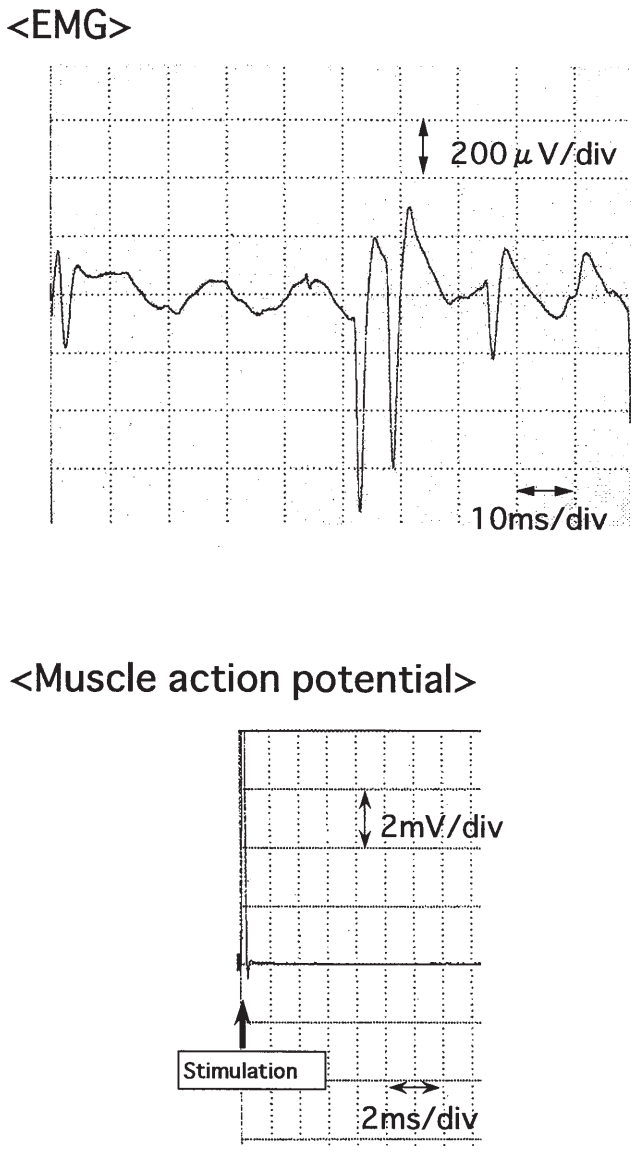

Fig. 1. EMG recording from the sublingual muscle (*above*) and muscle action potential recorded from the left abductor hallucis muscle (*below*) on electrical stimulation (50 mA) to the left tibial nerve. Multiphasic motor unit potentials (neuropathic unit) were detected on EMG from the sublingual muscle (*above*). No muscle action potential could be detected, indicating that the motor nerve to the muscle did not exist

not with a small dose of fentanyl. This effect was manifested within 30 s. EMG monitoring is currently the only method available in clinical practice for assessing muscular activity. The sites chosen were considered only representative lesions of muscular activity in the body in general.

Fibrillation potentials on EMG can be seen at an advanced stage in motor neuron loss, which is the primary pathologic feature of ALS [5]. A study using in vitro autoradiography with ^{3}H-α-bungarotoxin in biceps brachii muscle from a patient with ALS, demonstrated that acetylcholine receptors existed over the entire muscle, whereas in the normal case the binding was restricted to the motor end-plate region [6]. These data suggested that the extrajunctional acetylcholine receptors might increase on the skeletal muscles of patients with ALS. Brumback et al. [7] reported that fibrillation potentials in denervated rat muscle were activated by succinylcholine and the fibrillation was silenced by tetrodotoxin and procaine hydrochloride. They suggested that increased acetylcholine receptors might play a role in the development of spontaneous fibrillation.

Some recent studies have reported the effects of various anesthetic agents on nicotinic acetylcholine receptors. Dilger et al. [8,9], using single-channel recording techniques to study the effects of general anesthetics on nicotinic acetylcholine receptor channels, reported that propofol, as well as isoflurane, binds to a site on the receptor protein and interrupts the flow of ions through the pore of the channel. Although fentanyl also appears to decrease the opening time of nicotinic acetylcholine ion channels activated by acetylcholine, fentanyl can reduce the channel opening time only at concentrations 400 times greater than clinical plasma levels [10]. It is suggested that a different mechanism may underlie the effect of propofol and fentanyl on the kinetics of nicotinic acetylcholine ion channels [10]. Thus, the suppression of fibrillation potentials may be attributed to interaction between propofol and fentanyl on the nicotinic acetylcholine receptors.

There is no evidence that a specific anesthetic agent or combination of drugs is the best for administration to a patient with ALS. Administration of succinylcholine for muscle relaxation to a patient with ALS should be avoided because of the likelihood of causing hyperkalemia [4]. Volatile anesthetics are believed to be more useful for neuromuscular disorders [2] than intravenous anesthetic agents, because volatile agents are significant skeletal muscle relaxants and will potentiate the effects of neuromuscular blocking agents, apparently by a postsynaptic action. Although propofol is not known to have any effect on the neuromuscular junction nor to interact with neuromuscular blocking agents, the present case suggests that propofol might cause some muscle relaxation by blocking effects on the acetylcholine receptor of skeletal muscles in some neuromuscular disorders.

We speculate that propofol might suppress spontaneous fibrillation mainly by blocking the function of nicotinic acetylcholine receptors on the skeletal muscles in ALS.

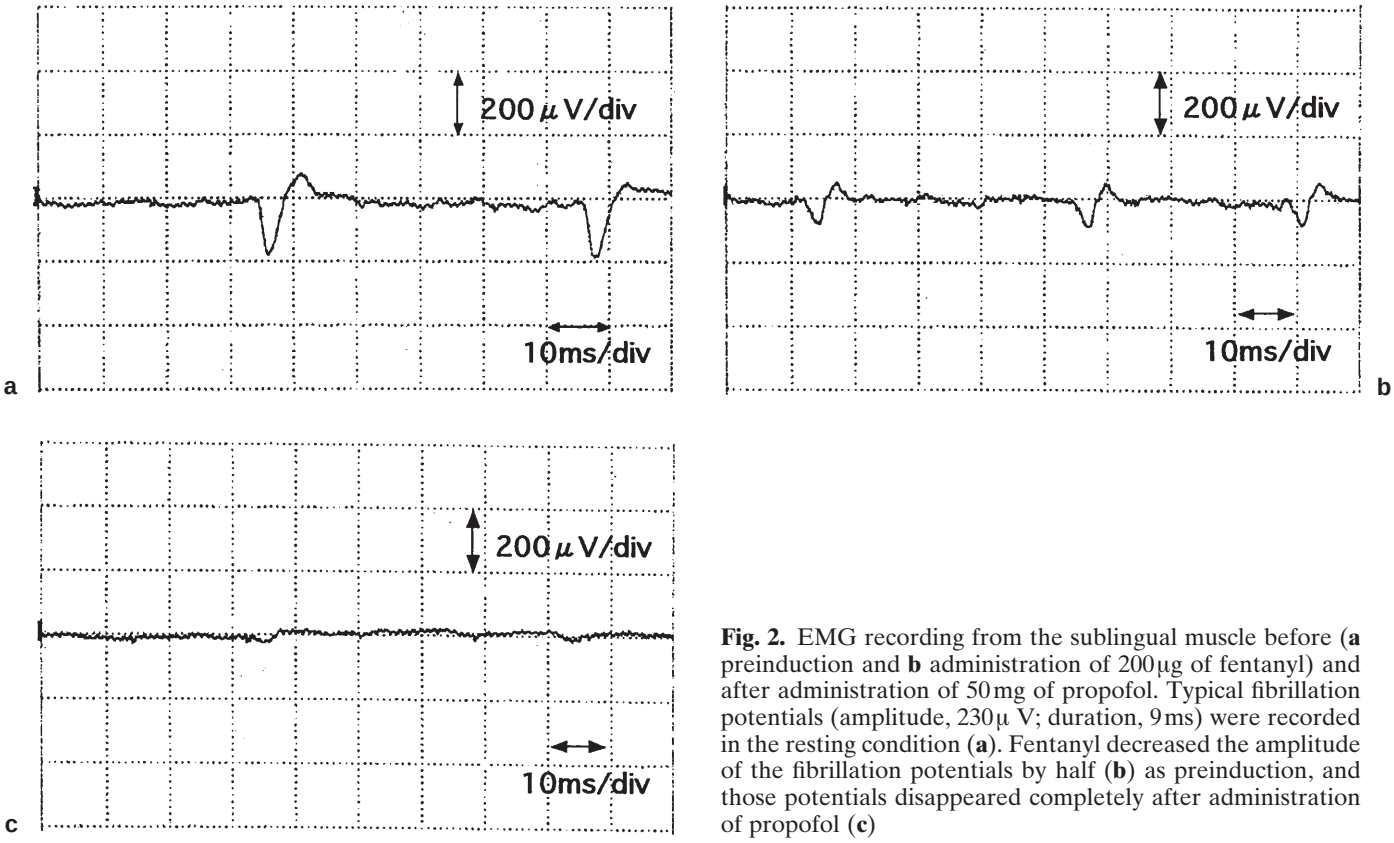

Fig. 2. EMG recording from the sublingual muscle before (**a** preinduction and **b** administration of 200μg of fentanyl) and after administration of 50mg of propofol. Typical fibrillation potentials (amplitude, 230μ V; duration, 9ms) were recorded in the resting condition (**a**). Fentanyl decreased the amplitude of the fibrillation potentials by half (**b**) as preinduction, and those potentials disappeared completely after administration of propofol (**c**)

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