Action potential of left ventricular muscle in normal and dystrophic mice

	RP(mV)	OS(mV)	AP(mV)	dv/dt(v/sec)	APD_{50}(msec)	APD_{80}(msec)
Normal (n = 197)	81 ± 5	26 ± 4	106 ± 6	330 ± 35	15 ± 2	36 ± 5
Dystrophy (n = 198)	77 ± 4	24 ± 4	101 ± 6	303 ± 31	19 ± 3	47 ± 8
p	< 0.001	< 0.01	< 0.001	< 0.001	< 0.001	< 0.001

Values in this table are expressed as mean ± SD. OS, overshoot; RP, resting potential; AP, action potential amplitude; APD_{50}, 50% duration of action potential; APD_{80}, 80% duration of action potential; dv/dt, maximal rate of depolarization.

RP, AP and dV/dt were significantly decreased in dystrophic mice.

The APD50 and APD80 were significantly longer in dystrophic mice than in normal mice. The representative recordings of action potentials are shown in the figure.

The basis for the RP of the myocardial cell can be understood in terms of the electrochemical gradient for potassium that exists across the sarcolemma. We reported that[4] both the potassium content and the RP of skeletal muscles in dystrophic mice were significantly lower than those in normal mice at the same age of 8 weeks. While there was no difference between the myocardial potassium contents in dystrophic and normal mice, the RP of LV in dystrophic mice was significantly lower than that in normal mice. This result may be explained by either an increased inward background current or a decreased outward current. It is well known that membrane conductance to potassium is significantly and rapidly altered by an alteration in the intracellular calcium ion concentration[5,6], and the plateau phase of the action potential is determined by the balance between the outward current and slow inward current[7].

The plateau of the action potential in dystrophic mice was more prolonged than that in normal mice. This finding could be explained by either an increased slow inward current or a decreased late outward current. In conclusion, we demonstrate in this study that myocardial cells of dystrophic mice have abnormal membrane permeability at the stage when changes in contents of potassium, water and fat are not developed.

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The size of motor units in laryngeal muscles of the rat

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Summary. The size of motor units in rat laryngeal muscles was determined by correlating the number of neurons labeled by i.m. injections of horseradish peroxidase with the number of motor end plates stained for acetylcholinesterase. The cricothyroid has a motor unit size of 8 muscle fibers per motor neuron and the posterior cricoarytenoid 4–5 muscle fibers per motor neuron.

Derived from the primitive sphincteric musculature of the pharynx, the laryngeal muscles in mammals have taken on such diverse functions as the control of air distribution in the lungs during respiration[1], protection of the pulmonary apparatus[2], and phonation or the production of sound[3]. It might be anticipated in such highly skilled musculature, that the size of motor units would be small. However, morphological support for this premise is scant and conflicting. Rüedi[4] states that there are 2–3 muscle fibers per motor unit in human laryngeal muscles but gives no details of how this figure was obtained. English and Blevins[5] estimate that there are 30 muscle fibers per motoneuron in the cricothyroid (CT) of human and 50–60 muscle fibers per motor neuron in CT of cat. They related the number of fibers (stained with silver) in muscle nerves to the number of muscle fibers (the innervation ratio) and assumed the same ratio of sensory to motor fibers as exists in spinal nerves (an untenable assumption[6]). Faaberg-Anderson[7] in a study of a newborn infant has determined that the average motor unit contains 166 fibers in CT and 116 in PCA.

Difficulties in accurately determining the size of laryngeal motor units can be related to the demonstration of multiple innervation of laryngeal muscles in some species[8–10], the great variability in the number of fibers in nerves supplying the larynx[11], variation in branching of laryngeal nerves[12] and the recent demonstration that, in the kitten, at least 2 nuclei in the brainstem provide motor innervation to laryngeal muscles[13].

We have determined the size of laryngeal motor units in the rat by correlating the number of cells labelled in the brainstem by retrograde transport of horseradish peroxidase (HRP, Boehringer) following i.m. injection of a 50% solution in 2% dimethylsulphoxide[14,15], with the number of motor end plates counted from serial longitudinal sections of muscle stained for acetylcholinesterase[16].

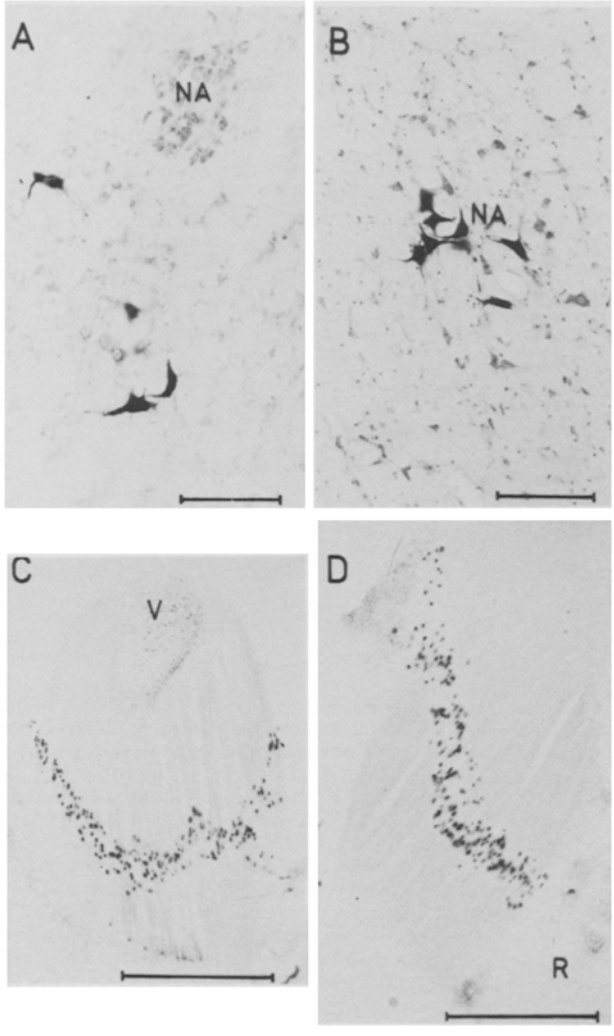
Under chloral hydrate anaesthesia (0.3 ml/100 g b.wt of a 12% aqueous solution) HRP was injected into the CT or PCA of 23 adult male hooded rats (Wistar strain) weighing 200–500 g, using a 1 µl Hamilton syringe or glass pipette attached to a hydraulic system.

After survival times of 24–48 h, rats were perfused and serial 50 µm coronal sections processed according to a modified histochemical method of Mesulam[17]. Spread beyond the injection site was controlled and assessed by ligating and cutting the remaining nerves to laryngeal muscles other than those injected, examining brainstem sections for signs of contralateral uptake in the nucleus ambiguus (NA) and observation of characteristic ipsilateral distribution of cells within the NA for individual muscles. No NA cells were labeled by sham operations where HRP was intentionally spilt over the exposed muscles. Motor end-plate counts were made from laryngeal muscles of rats of the same species and weight rather than from the same HRP injected animal as the fixative used in the acetylthiocholine technique[18] was not compatible with the HRP technique[16].

There was no evidence in the rat of dual innervation of laryngeal muscles from 2 separate brainstem nuclei as has been reported in the kitten[13]. Neurons labeled by HRP in the present study showed 2 peaks in estimates of cross section area of their profiles[19], but these cells were evenly distributed throughout the muscle representation within NA. The 2 neuron sizes are thought to innervate the different muscle types shown histochemically[20] in rat laryngeal muscles. The labelled cells appeared to be only *a* motor neurons since no muscle spindles have been observed in laryngeal muscle of the rat[21,22]. In other muscles it might be expected that *γ* motor neurons would also be labeled as, for example, labeling of cells of the trigeminal mesencephalic nucleus by injections of HRP into masticatory muscles[23] indicates that HRP can penetrate the capsule of muscle spindles.

Although there was some variation in number of HRP labeled cells[19] it was considered that the experiment in which the greatest number of cells were labeled following i.m. injection most closely indicated the case where all of the motor end plates were exposed to HRP. It should be noted that no cells in NA have been shown to contain endogenous peroxidase by the Mesulam method[24].

The maximum number of cells labeled in NA following injections into CT was 175 and the number of motor end plates 1362 ± 170 indicating a motor unit size of approximately 8 fibers per motor neuron. The maximum number of cells labeled in NA following injections into PCA was 166 and the number of motor end plates 764 ± 16 indicating a motor unit size of approximately 4–5 fibers per motor neuron. These figures correlate well with the extremely short duration of the action potential in rat CT and PCA[25]. It appears that the laryngeal muscles of the rat may possess a greater safety factor than those of the cat or human and are capable of fine control.



Cells labeled following i.m. injection of CT (A) and PCA (B) with HRP. Note that cells supplying CT are located beyond the compact nuclear group identified as NA on the basis of Nissl staining. Bar 100 µm. Motor end plates stained for acetylcholinesterase in longitudinal sections of CT (C) and PCA (D).
V, ventral; R, rostral; Bar 1 mm.

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