

Membrane parameters at the respective resting potentials in low, normal, and high extracellular potassium solutions were determined in intercostal muscle fibers from 15 patients with no known neuromuscular disease. In synthetic interstitial fluid (normal potassium concentration 3.5 mmol/liter), we found the following mean values: resting membrane potential  $RP = -83.3$  mV, space constant  $\lambda = 2364$   $\mu\text{m}$ , fiber diameter  $d = 49.3$   $\mu\text{m}$ , fiber input resistance  $R_{in} = 795$  k $\Omega$ , specific membrane capacitance  $C_m = 4.7$   $\mu\text{F}/\text{cm}^2$ , and specific membrane resistance  $R_m = 5970$   $\Omega\cdot\text{cm}^2$ . The specific membrane conductance was  $g_m = 168$   $\mu\text{S}/\text{cm}^2$ , 76% of it being chloride conductance, 24% being potassium conductance. The dependence of the membrane parameters on the extracellular potassium concentration followed the predictions by the constant field theory. There was no indication of active chloride transport. The resting membrane conductance decreased with temperature with a  $Q_{10}$  of 1.3. Excitability parameters were nearly independent of temperature between 37 and 27°C.

MUSCLE & NERVE 7:60-65 1984

## THE RESTING MEMBRANE PARAMETERS OF HUMAN INTERCOSTAL MUSCLE AT LOW, NORMAL, AND HIGH EXTRACELLULAR POTASSIUM

HUBERT KWIECIŃSKI, MD,  
FRANK LEHMANN-HORN, MD,  
and REINHARDT RÜDEL, PhD

In pioneer studies by Dillon et al.<sup>7</sup> and Creese et al.<sup>5</sup> the isolated human external intercostal muscle was found to be an excellent preparation for electrophysiological studies *in vitro*. Excised intercostal muscle is the only human muscle preparation that can be obtained without too much inconvenience to the donor and yet provides intact fibers for the experimenter. Over the years, there have been a number of investigations showing the partic-

ular usefulness of the intercostal muscle preparation in the study of human neuromuscular diseases.<sup>10,14,16-19,22</sup>

The electrical properties of human intercostal fibers have been studied in the past with the conventional 2-microelectrode technique. Recently, the 3-microelectrode voltage-clamp technique developed for mammalian muscle by Adrian and Marshall<sup>1</sup> was applied to human intercostal muscle to study the pathophysiology of muscle diseases characterized by variations of extracellular potassium.<sup>15,24</sup> The experiments reported here were carried out to obtain comprehensive information on the resting electrical membrane parameters of healthy intercostal fibers as control values for such studies on diseased muscle. In addition to the resting membrane parameters, we also investigated the fiber excitability. Studies of the voltage-dependence of the passive membrane conductance are described in a following paper.<sup>13</sup>

### MATERIALS AND METHODS

The study was carried out with the permission of the ethical commission of the Technical University of Munich, and abided by the Helsinki convention. During thoracotomy on 15 patients of either sex and with no known neuromuscular disease, speci-

From the Neurologische Klinik und Poliklinik der Technischen Universität München, West Germany (Drs. Kwieciński and Lehmann-Horn), and the Abteilung für Allgemeine Physiologie der Universität Ulm, West Germany (Dr. Rüdel).

Acknowledgments: The work was performed while Dr. Kwieciński was absent on leave from the Department of Neurology, Medical School, Warsaw, Poland, as a visiting fellow of the Alexander von Humboldt-Stiftung. Special appreciation is extended to Professor J. Dudel, Institute of Physiology of the Technical University of Munich, in whose department the experiments were carried out. The authors acknowledge with thanks the generous support of Professors I. Hausmanowa-Petrusewicz and A. Struppler. We are grateful to Drs. Mack and Präuer for providing the excellent muscle specimens, and to Ms. E. Höhne for technical assistance.

Address reprint requests to Dr. Kwieciński at the Department of Neurology, Medical School, Warsaw, Poland.

Received for publication March 18, 1983; revised manuscript accepted for publication July 18, 1983.

0148-639X/0701/0060 \$4.00/0  
© 1984 John Wiley & Sons, Inc.

mens of external intercostal muscle were removed. The patients were 26–68 years of age and were undergoing thoracotomy for lung tumor, spontaneous pneumothorax, bronchiectasis, pericardial cyst, hemothorax, and mediastinal tumor. None of the tumor patients had clinical or electromyographic signs of neuromuscular disease. In vitro, their intercostal muscle fibers gave results that were indistinguishable from those of the other patients. Immediately after removal, the specimens were placed in a continuously gassed (95% O<sub>2</sub>, 5% CO<sub>2</sub>) synthetic interstitial fluid (Bretag's solution)<sup>3</sup> having the following composition (in mM): NaCl 107.7, KCl 3.48, CaCl<sub>2</sub> 1.53, MgSO<sub>4</sub> 0.69, NaHCO<sub>3</sub> 26.2, NaH<sub>2</sub>PO<sub>4</sub> 1.67, Na gluconate 9.64, glucose 5.5, sucrose 7.6 (pH 7.4) and taken to the laboratory for dissection at room temperature. The isolated bundles of intact fibers were mounted in an experimental chamber of about 6 ml content which was continuously perfused at 6 ml/min with gassed Bretag's solution. The bath temperature was usually set at 37°C; it would be quickly reduced to 27°C. In some experiments a chloride-free solution was used in which NaCl and KCl were replaced by the respective methane sulfonate salts and CaCl<sub>2</sub> by Ca gluconate. To obtain control values for experiments with muscles from patients with hyperkalemic or hypokalemic periodic paralysis, we carried out experiments with a modified Bretag's solution in which the potassium concentration was changed to 7 or 1 mM, respectively, without correction of the resulting osmotic changes. Tetrodotoxin (TTX, Roth, Karlsruhe, West Germany), when added to any of these solutions, was at 0.3 mg/liter.

Standard glass microelectrodes were used for potential measurement and current injection. The current electrodes were filled with 2 M potassium citrate and had tip resistances of 5 to 8 MΩ. The voltage electrodes were filled with 3 M KCl and had resistances of 10 to 20 MΩ. We employed the 3-microelectrode voltage-clamp method developed for muscle fibers by Adrian and Marshall.<sup>1</sup> The procedure was identical to that used by Lehmann-Horn et al.<sup>14</sup> Three microelectrodes were inserted into the midregion of a fiber. The left and center electrodes (V<sub>2</sub> and V<sub>1</sub>, respectively) recorded the membrane potential while the third electrode was used to inject current into the fiber. A desk computer was programmed with the algorithm of Adrian and Marshall<sup>1</sup> to calculate from the voltage-clamp results the length constant and the longitudinal fiber resistance. Using a myoplasmic resistivity of 125 Ω·cm at 37°C<sup>17</sup> with a Q<sub>10</sub> of 1/

1.37,<sup>12</sup> the computer calculated the fiber diameter and the specific membrane conductance (resistance<sup>-1</sup>) at the resting potential. The specific membrane capacitance was calculated from the time for V<sub>1</sub> to reach 84% of its final value in an unclamped square current pulse.<sup>2</sup>

Excitability characteristics were determined using two microelectrodes, one for passing current and the other for recording the membrane potential. Current and voltage electrodes were inserted into the same fiber at a distance between 50 and 100 μm. Depolarizing constant-current pulses of 120 msec duration were passed into a fiber and the current and voltage traces were photographed from the oscilloscope screen. Starting with zero intensity, the pulse amplitude was gradually increased until the depolarization was just sufficient to elicit a single action potential. The following characteristics were obtained from the photographic records: the rheobasic current (I), i.e., the minimum current intensity that elicited a single action potential; the critical membrane potential (CMP), i.e., the potential at which a rheobasic pulse elicited a single action potential; the latency (L), i.e., the duration from the beginning of a rheobasic pulse to the beginning of the action potential; and the overshoot of the action potential (OS). The excitability characteristics were determined at 37 and 27°C. Values were expressed as mean ± SD.

## RESULTS

**Resting Membrane Parameters.** Table 1 summarizes the passive membrane parameters of intercostal fibers determined in three solutions with different potassium concentrations, [K]<sub>e</sub>. For each [K]<sub>e</sub>, the membrane parameters were determined in TTX-containing and in TTX-free solution. A significant difference between these results that could be attributed to the presence of TTX was never found. The data from 212 intercostal fibers in Bretag's solution with and without TTX were, therefore, combined to give the mean standard values listed in the uppermost line of Table 1. The mean specific membrane capacitance determined from these fibers was 4.7 ± 1.1 μF/cm<sup>2</sup>.

The usual method for determining the component conductances, g<sub>K</sub> and g<sub>Cl</sub>, is to measure membrane conductances in chloride-containing and in chloride-free solutions. If g<sub>Na</sub> is negligible, and g<sub>K</sub> is not influenced by the absence of extracellular chloride, the membrane conductance in chloride-free solution is identical with g<sub>K</sub>, and the difference of the membrane conductances in chloride-

**Table 1.** Resting potentials (RP), calculated fiber diameters (d), length constants ( $\lambda$ ), specific membrane resistances ( $R_m$ ), specific membrane conductances ( $g_m$ ), and input resistances ( $R_{in}$ ) of human intercostal fibers at 37°C.\*

	Fibers (patients)	RP (mV)	d ( $\mu\text{m}$ )	$\lambda$ ( $\mu\text{m}$ )	$R_m$ ( $\Omega \cdot \text{cm}^2$ )	$g_m$ ( $\mu\text{S}/\text{cm}^2$ )	$R_{in}$ ( $\text{k}\Omega$ )
Bretag's solution†	212(15)	-83.3	49.3	2364	5970	167	795
3.5 mM K <sup>+</sup>	112 (15)	-83.7 ± 3.9	49.8 ± 4.1	2336 ± 203	5950 ± 1049	168 ± 25	781 ± 128
3.5 mM K <sup>+</sup> , TTX	100 (15)	-82.8 ± 3.2	48.7 ± 4.4	2391 ± 212	5992 ± 1115	167 ± 26	810 ± 122
1 mM K <sup>+</sup>	38 (7)	-99.6 ± 3.6	47.5 ± 5.0	3100 ± 476	10030 ± 2141	100 ± 17	1124 ± 229
1 mM K <sup>+</sup> , TTX	56 (8)	-99.7 ± 3.2	47.6 ± 4.3	3123 ± 478	10315 ± 2084	97 ± 16	1181 ± 186
7 mM K <sup>+</sup>	46 (8)	-69.0 ± 4.8	47.6 ± 5.2	1789 ± 250	3404 ± 778	294 ± 55	672 ± 137
7 mM K <sup>+</sup> , TTX	51 (8)	-70.5 ± 3.8	46.4 ± 4.8	1715 ± 288	3365 ± 681	297 ± 50	688 ± 111
3.5 mM K <sup>+</sup> , TTX 37°C	64 (8)	-82.3 ± 3.2	48.9 ± 3.8	2367 ± 205	5721 ± 1021	175 ± 31	797 ± 113
3.5 mM K <sup>+</sup> , TTX Cl-free, 37°C	57 (8)	-82.6 ± 3.2	54.2 ± 4.4	4798 ± 625	23762 ± 4782	42 ± 8	1498 ± 212
3.5 mM K <sup>+</sup> , TTX 27°C	27 (4)	-74.9 ± 2.9	50.2 ± 4.2	2429 ± 260	7381 ± 825	136 ± 15	909 ± 129
3.5 mM K <sup>+</sup> , TTX Cl-free, 27°C	23 (4)	-74.4 ± 2.3	55.0 ± 4.0	4658 ± 502	24210 ± 3481	41 ± 6	1660 ± 165

\*The last four lines provide information on temperature effects.

†The results obtained in Bretag's solution with and without TTX were pooled to give the standard values listed in the first line.

**Table 2.** Specific membrane conductances ( $g_m$ ) and component conductances ( $g_{Cl}$  and  $g_K$ ) of human intercostal muscle fibers at 37°C and 27°C.

Temperature (°C)	Fibers (patients)	$g_m$ ( $\mu\text{S}/\text{cm}^2$ )	$g_{Cl}$ ( $\mu\text{S}/\text{cm}^2$ )	$g_K$ ( $\mu\text{S}/\text{cm}^2$ )	$g_{Cl}^*$ (%)	$g_K^*$ (%)
37	121 (8)	175 ± 31	133 ± 25	42 ± 8	76	24
27	50 (4)	136 ± 15	95 ± 12	41 ± 6	70	30

\*Expressed as fraction of  $g_m$ .

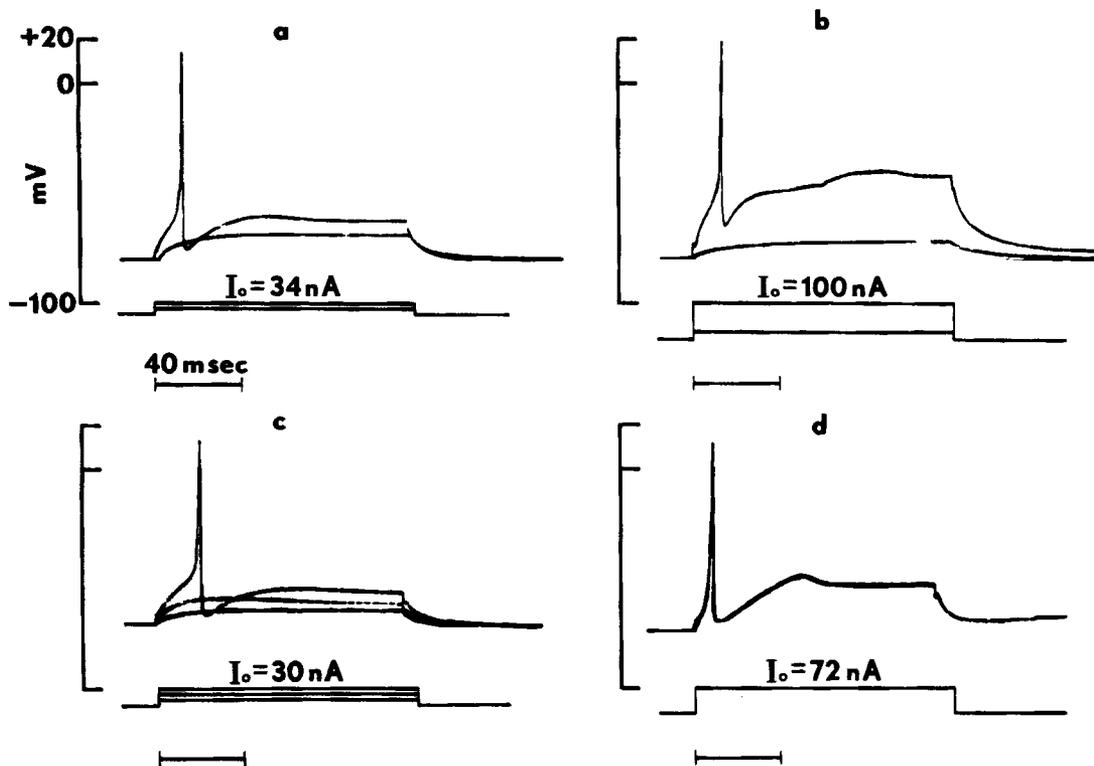
containing and chloride-free solution is identical with  $g_{Cl}$ . Because of the increased excitability of fibers in chloride-free solution, such experiments have to be carried out in the presence of TTX. The membrane parameters obtained in an experimental series carried out with fibers from eight different biopsies are compiled in the lower part of Table 1. The component conductances calculated from these data are listed in Table 2. According to these calculations, 76% of the resting membrane conductance is attributable to  $g_{Cl}$ . We also determined membrane conductances at 27°C to evaluate their temperature dependence ( $Q_{10} = g_{37^\circ\text{C}}/g_{27^\circ\text{C}}$ ). The specific membrane conductance was found to have a  $Q_{10}$  of 1.3,  $g_{Cl}$  declined with a  $Q_{10}$  of 1.4, and  $g_K$  demonstrated little change with temperature ( $Q_{10}$  about unity). Thus, the temperature dependence of  $g_m$  can be largely attributed to a temperature-dependent  $g_{Cl}$ .

**Excitability Parameters.** These are summarized in Table 3. Representative recordings from which excitability data were obtained are shown in Fig. 1a. The fibers mostly responded with a single action

potential even if the long-lasting depolarization was well above threshold (Fig. 1b). In a few fibers, a second action potential having a reduced amplitude was elicited. We also evaluated the effects of cooling on the fiber excitability. At 27°C (Fig. 1c), the rheobasic current was smaller than, and the critical membrane potential was slightly positive to the respective values at 37°C, probably owing to a slight decrease of the resting potential. Raising the current amplitude to 2–3 times rheobase resulted in responses similar to those at 37°C (Fig. 1d).

## DISCUSSION

The results of the present study can be compared with several earlier determinations of membrane parameters at the resting potential (see Table 4). Such comparisons are inevitably vague because of the different solutions used by different experimenters. We prefer the potassium concentration of 3.5 mM in our standard solution because, according to Bretag,<sup>3</sup> this is the true potassium concentration in interstitial fluid, while the higher values of 4.5 to 5 mM used by others correspond to



**FIGURE 1.** Intracellularly recorded responses to 120-msec constant-current pulses at 37°C (a,b) and at 27°C (c,d). Upper traces: membrane potential. Lower traces: depolarizing current. In each panel, the current intensity associated with triggering of action potentials is designated as  $I_0$ . Bar = 40 msec.

the serum potassium concentration. The earliest data reported are those of Elmqvist et al.,<sup>9</sup> who used Liley's solution containing 5 mM potassium. When our data obtained with 3 and 7 mM potassium are interpolated, there is agreement between our results and theirs. Lipicky et al.<sup>16,17</sup> used a solution containing 4.5 mM potassium. For unknown reasons, their membrane potential and resistance values are much lower than Elmqvist's and ours. A possible explanation is that membrane depolarization caused by the slight injury produced during repeated impalements resulted in depolarization and too small resistance values. Such depolarization is prevented by the voltage clamp. Because of this and the use of the synthetic interstitial fluid, we believe that our rather high value of about  $6000 \Omega \cdot \text{cm}^2$  is representative for the specific membrane resistance of intact intercostal muscle in situ.

Our calculated mean fiber diameter ( $d = 49.3 \mu\text{m}$ ) is halfway between the extreme values of 45.5 and  $53.6 \mu\text{m}$  obtained by direct measurements from histologic transverse sections.<sup>26</sup> This congru-

ence of data is a positive test of the validity of the method and of our choice of  $R_i$ . Our mean specific membrane capacity ( $C_m = 4.7 \mu\text{m}/\text{cm}^2$ ) is almost identical with the value reported by Elmqvist et al.<sup>9</sup>

At 37°C, the specific membrane resistance in chloride-free solution was about 4 times the value in chloride-containing solution. This indicated that normally the chloride conductance is the major component of  $g_m$ . The usual calculation  $g_{\text{Cl}} = g_m - g_{\text{K}}$  shows that  $g_{\text{Cl}}$  accounts for 76% of the total membrane conductance, which is in close agreement with the value of 81.5% reported by Lipicky.<sup>16</sup> Our experiments with 1 mM potassium show that some of the assumptions made in the determination of component conductances may not be justified. For example, the membrane conductance in TTX-containing 1 mM potassium solution was found to be  $96.9 \mu\text{S}/\text{cm}^2$ . If we calculate  $g_{\text{K}}$  in this low potassium solution assuming  $g_{\text{K}} = g_m - g_{\text{Cl}}$  with  $g_{\text{Cl}} = 132.7 \mu\text{S}/\text{cm}^2$  (as determined in chloride-free 3.5 mM potassium solution), we obtain a negative value for  $g_{\text{K}}$ , which is obvious nonsense. This result suggests that  $g_{\text{K}}$  is not, or at least

**Table 3.** Resting potentials (RP), electrical threshold potentials (CMP), action potential overshoots (OS), rheobasic currents (I), and latencies (L) of human intercostal muscle fibers at 37°C and 27°C.

Temperature (°C)	Fibers (patients)	RP (mV)	CMP (mV)	OS (mV)	I (nA)	L (ms)
37	34 (5)	-82.3 ± 3.2	-58.4 ± 3.4	16.8 ± 4.3	40.7 ± 6.7	11.2 ± 3.0
27	24 (4)	-74.9 ± 2.9	-52.4 ± 1.9	15.5 ± 3.9	34.2 ± 4.6	12.6 ± 3.3

**Table 4.** Listing of determinations of membrane parameters of human intercostal muscle fibers.\*

Authors	Year	[K] <sub>e</sub> (mM)	Fibers (patients)	RP (mV)	R <sub>m</sub> (Ω·cm <sup>2</sup> )	C <sub>m</sub> (μF/cm <sup>2</sup> )	g <sub>m</sub> (μS/cm <sup>2</sup> )	g <sub>Cl</sub> (μS/cm <sup>2</sup> )	g <sub>K</sub> (μS/cm <sup>2</sup> )	g <sub>Cl</sub> (%)
Elmqvist et al. <sup>9</sup>	1960	5	7 (2)	-86.0	4070	4.8	246	—	—	—
Lipicky et al. <sup>17</sup>	1971	4.5	74 (11)	-75.6	2619	5.2	381	439	117	79
Lipicky <sup>16</sup>	1979	4.5	223 (21)	—	1876	—	533	500	106	81.5
Lehmann-Horn et al. <sup>16</sup>	1981	3.5	46 (3)	-82.8	6900	3.6	145	103	42	71
Present study	1983	3.5	221 (15)	-83.3	5970	4.7	167	133	42	76

\*For abbreviations see legend to Table 1.

not always, independent of [Cl]<sub>e</sub>, and/or that g<sub>Cl</sub> is not independent of [K]<sub>e</sub>. At any rate, the results of such calculations should be considered with care.

Our experiments at low temperature show that the Q<sub>10</sub> of the membrane conductance is 1.3, a value which is compatible with the assumption of passive rather than active processes. The variation of g<sub>m</sub> with temperature was found to be caused mainly by the temperature dependence of g<sub>Cl</sub>. Our Q<sub>10</sub> of 1.4 is in favor of passive chloride movements rather than a carrier mechanism in accordance with Palade and Barchi<sup>23</sup> who considered the pathway for chloride through the membrane as being an aqueous pore. In an earlier study, Lehmann-Horn et al.<sup>14</sup> found Q<sub>10</sub> values of 1.25 and 0.80 in muscles of two control persons. Abnormally low Q<sub>10</sub> values of 0.15 and 0.25 were found for g<sub>Cl</sub> in intercostal muscles of patients with paramyotonia congenita.<sup>14</sup> In the present and in the earlier<sup>14</sup> study, g<sub>K</sub> showed virtually no temperature dependence. This result is unexpected; it must be taken with care for the reasons discussed previously.

Our data provide the first systematic investigation of the dependence of the specific membrane conductance on the extracellular potassium concentration in human skeletal muscle. The results show that g<sub>m</sub> decreases and increases considerably with lowering and raising [K]<sub>e</sub>, respectively, as expected from the constant field theory.<sup>11</sup> Also, the dependence of the resting potential on [K]<sub>e</sub> followed fairly well the predictions of the constant field theory. If we assume intra- and extracellular potassium activity coefficients<sup>4</sup> of 0.59 and 0.75,

respectively, an intracellular potassium concentration of 143.9 mM, and a resting sodium-to-potassium permeability ratio of 0.01, the Goldman-Hodgkin-Katz equation<sup>11</sup> predicts 103.3, 83.7, and 69.3 mV, respectively, for the resting potential in 1, 3.5, and 7 mM potassium solution (Table 1). Our results are consistent with the assumption that chloride distributes itself passively between the intra- and extracellular spaces.<sup>20</sup> This is not a trivial result because in other mammalian skeletal muscles a dependence of the resting potential on [Cl]<sub>e</sub> was found and the existence of active chloride transport in these muscles was postulated.<sup>8</sup> However, [Cl]<sub>e</sub> did not affect the resting potential in rat and goat muscles.<sup>6</sup> Active chloride transport is also improbable because, if existent, it would be extremely uneconomical in cells to have a g<sub>Cl</sub> as high as that of an intercostal muscle.

Tetrodotoxin did not change the resting potential at any [K]<sub>e</sub> investigated. Seabrooke and White<sup>25</sup> also found no influence of TTX on the resting potential at varied [K]<sub>e</sub> in nondenervated mammalian muscle, while in denervated muscle depolarization was reduced in the presence of TTX.

The excitability parameters of intercostal muscle were not much changed by lowering the temperature by 10°C. The observed shift of the critical membrane potential to a less negative value is explained by the lowered resting potential. Similar findings were reported by Thesleff and Ward<sup>27</sup> and Marshall and Ward.<sup>21</sup> The decrease of rheobase with decreasing temperature can be explained by the concomitant increase of the specific membrane resistance.

## REFERENCES

1. Adrian RH, Marshall MW: Sodium currents in mammalian muscle. *J Physiol (Lond)* 268:223–250, 1977.
2. Boyd IA, Martin AR: Membrane constants of mammalian muscle fibres. *J Physiol (Lond)* 147:450–467, 1959.
3. Bretag AG: Synthetic interstitial fluid for isolated mammalian tissue. *Life Sci* 8:319–329, 1969.
4. Charlton MP, Silverman H, Atwood HL: Intracellular potassium activities in muscles of normal and dystrophic mice: An in vivo electrometric study. *Exp Neurol* 71:203–219, 1981.
5. Creese R, Dillon JB, Marshall J, Sabawala PB, Schneider DJ, Taylor DB, Zinn OE: The effect of neuromuscular blocking agents on isolated human intercostal muscles. *J Pharmacol Exp Ther* 119:485–494, 1957.
6. DeCoursey TE, Bryant SH, Owenburg KM: Dependence of membrane potential on extracellular ionic concentrations in myotonic goats and rats. *Am J Physiol* 240:C56–C63, 1981.
7. Dillon JB, Fields J, Gumas T, Jenden DJ, Taylor DB: An isolated human voluntary muscle preparation. *Proc Soc Exp Biol Med* 90:409, 1955.
8. Dulhunty AF: The dependence of membrane potential on extracellular chloride concentration in mammalian skeletal muscle fibres. *J Physiol (Lond)* 276:67–82, 1978.
9. Elmqvist D, Johns TR, Thesleff S: A study of some electrophysiological properties of human intercostal muscle. *J Physiol (Lond)* 154:602–607, 1960.
10. Gruener R, Stern L, Markovitz D, Gerdes C: Electrophysiologic properties of intercostal muscle fibers in human neuromuscular diseases. *Muscle Nerve* 2:165–172, 1979.
11. Hodgkin AL, Katz B: The effect of sodium ions on the electrical activity of the giant axon of the squid. *J Physiol (Lond)* 108:37–77, 1949.
12. Hodgkin AL, Nakajima S: The effect of diameter on the electrical constants of frog skeletal muscle fibres. *J Physiol (Lond)* 221:105–120, 1972.
13. Kwieciński H, Lehmann-Horn F, Rüdell R: Membrane currents in human intercostal muscle at varied extracellular potassium. *Muscle Nerve*, submitted for publication.
14. Lehmann-Horn F, Rüdell R, Dengler R, Lorković H, Haass A, Ricker K: Membrane defects in paramyotonia congenita with and without myotonia in a warm environment. *Muscle Nerve* 4:396–406, 1981.
15. Lehmann-Horn F, Rüdell R, Ricker K, Lorković H, Dengler R, Hopf HC: Two cases of adynamia episodica hereditaria: In vitro investigation of muscle cell membrane and contraction parameters. *Muscle Nerve* 6:113–121, 1983.
16. Lipicky RJ: Myotonic syndromes other than myotonic dystrophy, in Vinken PJ, Bruyn GW (eds): *Handbook of Clinical Neurology*, Vol 40. 1979, pp 533–571.
17. Lipicky RJ, Bryant SH, Salmon JH: Cable parameters, sodium, potassium, chloride, and water content, and potassium efflux in isolated external intercostal muscle of normal volunteers and patients with myotonia congenita. *J Clin Invest* 50:2091–2103, 1971.
18. Ludin HP: Microelectrode study of normal human skeletal muscle. *Eur Neurol* 2:340–347, 1969.
19. Ludin HP: Microelectrode study of dystrophic human skeletal muscle. *Eur Neurol* 3:116–121, 1970.
20. Macchia DD, Baumgarten CM: Is chloride passively distributed in skeletal muscle in vivo? *Pfluegers Arch* 382:193–195, 1979.
21. Marshall MW, Ward MR: Anode brake excitation in denervated rat skeletal muscle fibres. *J Physiol (Lond)* 236:413–420, 1974.
22. Norris FH: Micropipette recording from human striated muscle. *J Neurol* 213:1–15, 1976.
23. Palade PT, Barchi RL: Characteristics of the chloride conductance in muscle fibers of the rat diaphragm. *J Gen Physiol* 69:325–342, 1977.
24. Rüdell R, Lehmann-Horn F, Ricker K, Küther G: Hypokalemic periodic paralysis: In vitro investigation of muscle cell membrane and contraction parameters. *Muscle Nerve*, in press.
25. Seabrooke SR, White NK: The contribution of tetrodotoxin-sensitive sodium channels to the resting membrane potential of innervated and denervated mouse skeletal muscle fibres. *J Physiol (Lond)* 318:36P, 1981.
26. Stern LZ, Payne CM, Gruener R, Anderson RM, Hannapel LK: Intercostal muscle biopsy in human neuromuscular diseases. *J Neurol Neurosurg Psychiatry* 38:900–913, 1975.
27. Thesleff S, Ward MR: Study on the mechanism of fibrillation potentials in denervated muscle. *J Physiol (Lond)* 244:313–323, 1975.