EXPERIMENTAL STUDIES

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Correlation of compound action potential and electromyography with facial muscle tension

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Functional electric stimulation is a new method for dynamic rehabilitation of paralyzed muscles. The output of such prosthetic devices needs to be modulated by some index of the muscle movement. In facial paralysis a measure of the muscle contractions of the normal contralateral side seems to be an appropriate input. In the rabbit, we simultaneously measured the compound action potential of the buccal branch of the facial nerve, the electromyogram of the zygomaticus major muscle, and the muscle twitch tension through strain gauge. The compound action potential, electromyogram, and strain gauge each had a sigmoidal relationship to stimulus intensity. The compound action potential peak-to-peak amplitude was found to have a linear correlation to the peak twitch tension of the corresponding facial muscle. The electromyogram response, although more variable, also had a linear correlation with muscle contraction. The possibility of predicting the contraction of facial muscles before they actually occur is discussed in the context of available and future functional electric rehabilitation models. (OTOLARYNGOL HEAD NECK SURG 1995;112:279-90.)

Intact facial neuromuscular units are responsible for facial tone, subserving symmetric facial expression at rest, rhythmic blinking for eye protection, a competent oral sphincter necessary for normal chewing and swallowing, and facial movements during emotional expressions. The paralysis of this system is devastating for the patient and creates a serious challenge for the surgeon.

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An entirely satisfactory solution to dynamic rehabilitation of the paralyzed face has yet to be found. Reanimation is best achieved when directed by the seventh nerve nucleus. If the proximal segment of the seventh nerve is available, direct neurorrhaphy or cable grafts provide the best results. Because of the lack of spatial segregation of facial motoneurons[1,2] and the absence of neutropism of regenerating axons,[3,4] the outcome is plagued by frequent synkinesis. Surgical procedures [see Reference 5 for recent reviews] have been proposed when the proximal facial nerve is unavailable. The prevailing opinion is that their number attests to the lack of satisfactory results.[5]

The possibility of reanimating paralyzed muscles with galvanic electric stimulation has been investigated during the last decade. Implantable systems have been developed for lower[6] and upper[7] limb paralysis. One of the main problems with these devices has been the input that controls the system. Usually movements in other, unparalyzed body aparts have been used. These movements are unfortunately much cruder than the movements that are to be generated (for example, shoulder movements used to control delicate hand muscles[7]). Con-

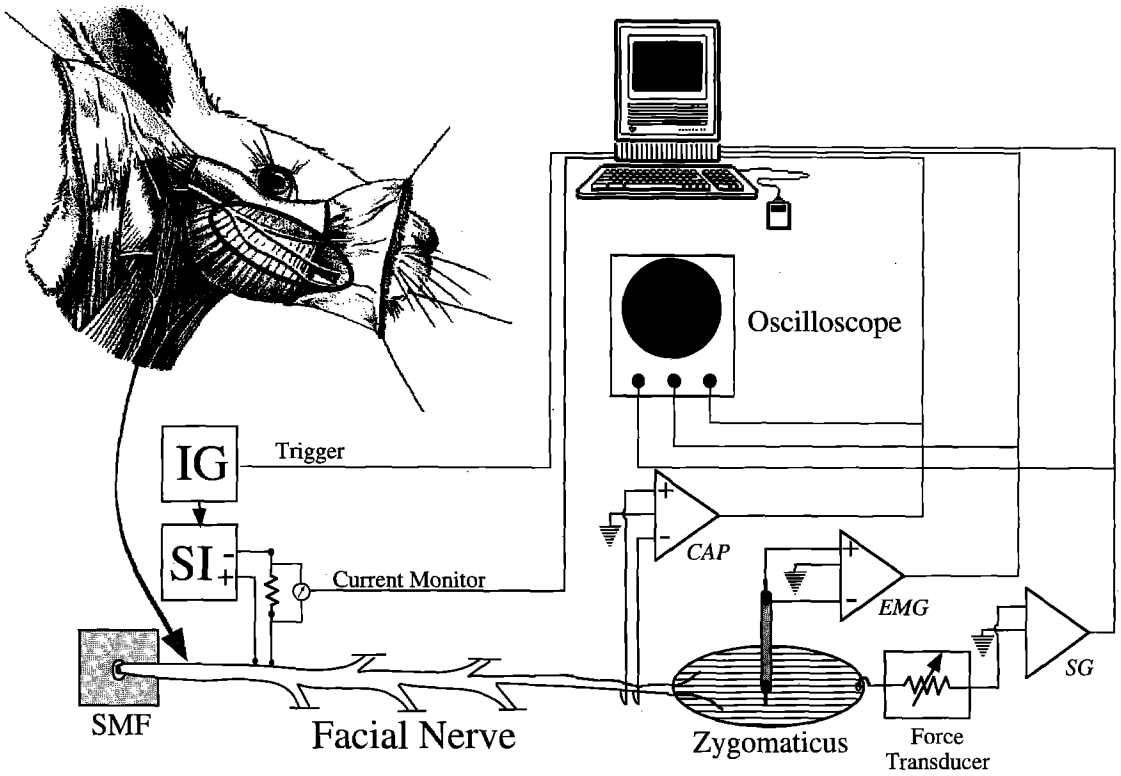

Fig. 1. Schema of experimental setup. After superficial parotidectomy all branches of the facial nerve except for the buccal branch to the zygomaticus muscle were transected. Bipolar stimulating electrodes were placed around the facial nerve at its exit from the stylomastoid foramen *(SMF)*. Bipolar recording electrodes suspended the facial nerve at a distance 4 to 4.5 cm distal to the stimulating electrodes and 2 to 3 cm proximal to the zygomaticus muscle. A bipolar concentric EMG needle was inserted into this muscle. A suture tied to the lateral upper lip was attached to an SG in the direction of the natural line of pull of this muscle. The interval generator *(IG)* triggered the computer and, after a 1-millisecond delay, triggered the nerve stimulator/stimulus isolator *(SI)* to produce a rectangular pulse of 100 microseconds' duration, with the cathode (negative lead) on the side of the recording electrodes. Stimulus intensity (i.e., the signal generated by the nerve stimulator/stimulus isolator hardware) refers to the current (in milliamps) recorded by measuring the voltage drop across a 1-kΩ resistor placed in series with this circuit. After amplification and filtering, the stimulus intensity, CAP, EMG, and SG signals were passed through an analog to digital board into the computer.

trary to limb muscles, in which the movements of opposite sides are quite different, axial muscles often act in symmetry. This prompted Zealar and Dedo[8] to propose a rehabilitation system of laryngeal paralysis based on contralaterally occurring movements. They used a strain gauge (SG) transducer to monitor the contralateral movements and provide an input to an electric stimulator on the normal side. This idea was later applied to facial paralysis reanimation.[9]

The facial musculature is a unique system in the mammalian body. Facial movements are mostly symmetric and do not involve joint movements. Thus the interplay of agonist and antagonist muscles is limited. Furthermore no proprioceptive receptors (such as Golgi tendon organs and muscle spindles) are present within facial muscles,[10] and no response to facial muscle contraction or stretching could be recorded from the facial nerve.[11] Thus the facial neuromuscular system seems in many ways simpler than others.

The ideal facial reanimation technique should provide for movements of the paralyzed face in synchrony and symmetry with the normal side. A functional electrical stimulator device could achieve these goals, if provided with an index of the contralaterally occurring movements. To achieve synchronous movements, muscle contractions of the normal side have to be predicted before they occur. Therefore a measurement of muscle contraction

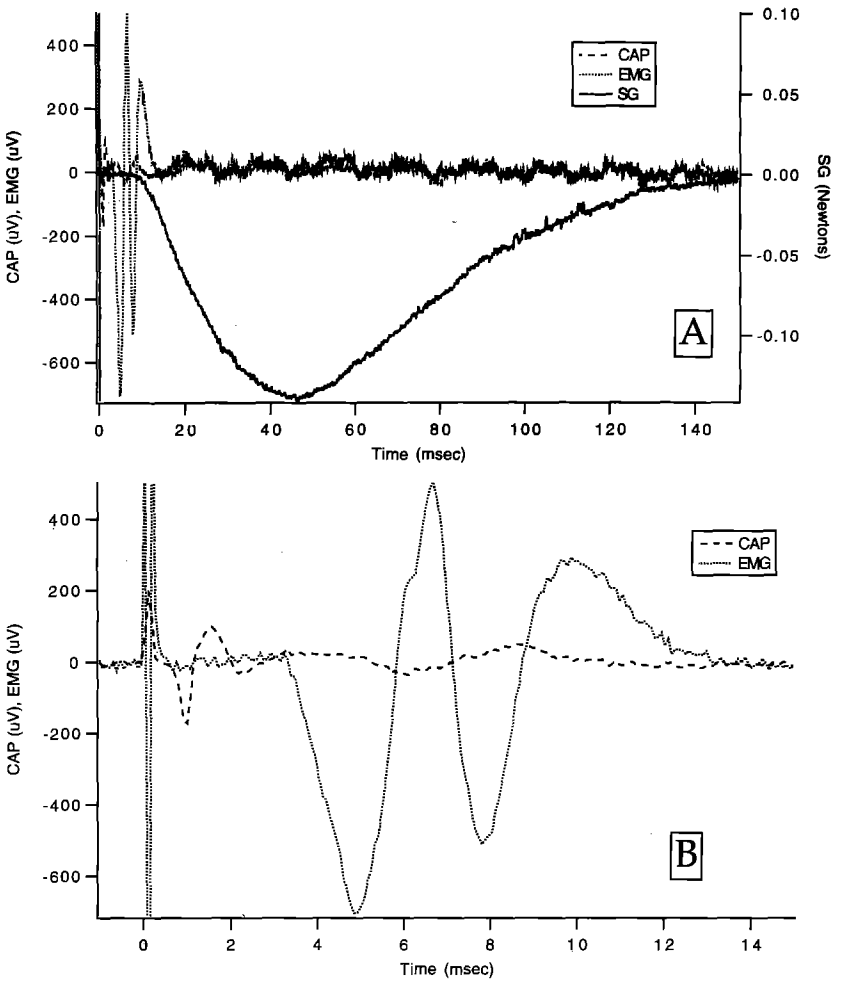

Fig. 2. Typical profile of CAP, EMG, and muscle twitch tension *(SG)* as a function of time. Traces are the result of a single electric pulse applied to the facial nerve trunk. Note different time scales in **A** and **B**.

needs to be derived. This event needs to occur early to provide time for information processing and routing to the contralateral muscles.

We raised the question as to what electrophysiologic signals can be used to predict muscle behavior. In limb muscles the electrical activity generated by the muscles during contraction (i.e., electromyogram [EMG]) has been found to be proportional to the actual muscle tension developed.[12-15] To our knowledge the relationship between facial EMG and corresponding facial muscle behavior has not yet been investigated. Rothstein and Berlinger[16] published strength-duration curves of the orbicularis oculi and zygomaticus minor muscle of the rabbit before and after denervation in response to electrical stimuli directly applied to the appropriate muscles. They stated that "there seemed to be a direct relationship between the strength of contraction of these muscle groups and the respective amplitude of the generated muscle potential."

We have investigated the relationship between compound action potential (CAP), EMG, and muscle tension as measured by SG in the frog sciatic nerve–gastrocnemius muscle system.[17] The CAP and EMG were found to have a linear correlation with muscle tension. In this study we assess whether facial nerve CAP and facial muscle EMG are related to facial muscle twitch tension. Electric stimuli were used to optimize the ability to ascertain fundamental relations between CAP, EMG, and SG.

METHODS

All animal protocols were approved by the University of California–Los Angeles animal care and use committee. Animals were cared for in compliance with all federal, state, and local regulations,

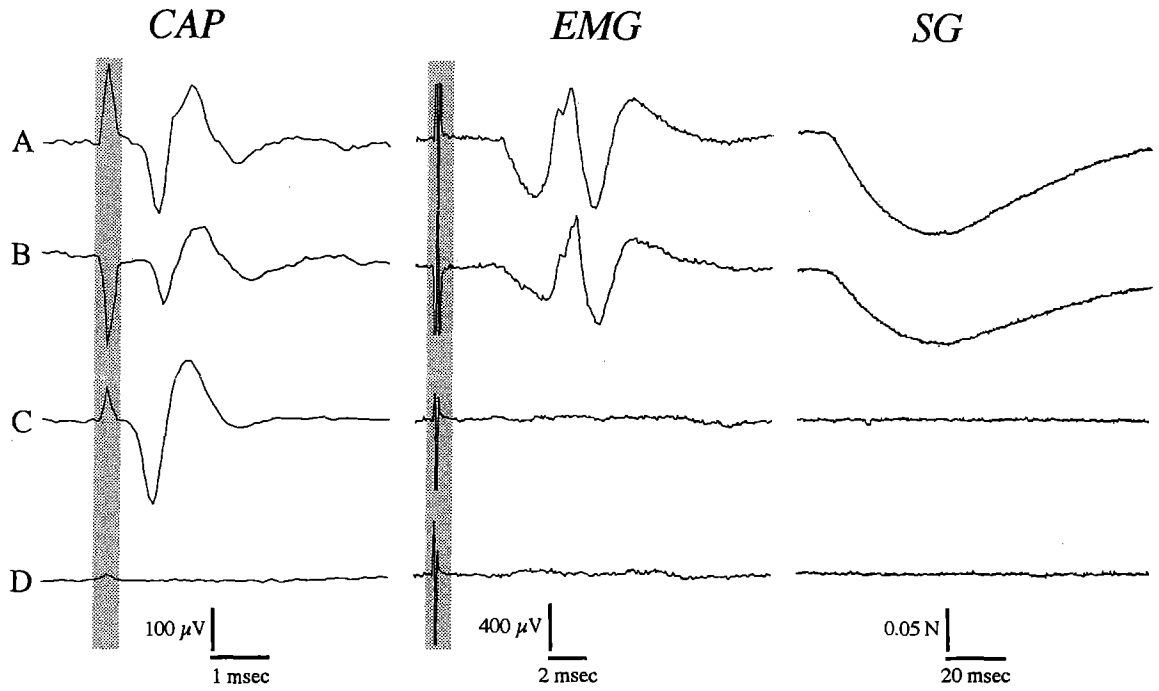

Fig. 3. Representative CAP, EMG, and SG waveforms after manipulations of the experimental preparation to verify their physiologic nature. Note that each set of waveforms (i.e., CAP, EMG, and SG) have correspondingly different amplitude and time scaling as indicated by the bars at the bottom of each wave set (SG amplitude scale units are in newtons). The shaded area highlights stimulus artifact. These representative waveforms were recorded from one animal. The traces in each row reflect the corresponding CAP, EMG, and SG waveforms at each of the following conditions: **A,** normal; **B,** inverted stimulus polarity; **C,** nerve transection between CAP recording electrodes and zygomaticus muscle; and **D,** nerve transection between stimulating electrodes and CAP recording electrodes.

and in accordance with the *Guide for the Care and Use of Laboratory Animals* (National Institutes of Health publication no. 80-23, rev. 1978). New Zealand white rabbits (4 to 5 kg each) were anesthetized with intramuscular ketamine hydrochloride (35 mg/kg) and xylazine hydrochloride (5 mg/kg). One side of the face was shaved. A superficial parotidectomy was performed, exposing the majority of the facial nerve from the stylomastoid foramen distally. A 1.5- to 2.5-cm distal segment of the buccal branch that directly innervated the zygomaticus muscle was then elevated from its underlying attachments, leaving the epineruium intact. All other branches of the facial nerve were then transected.

The experimental setup is illustrated in Fig. 1. A bipolar stimulating electrode was placed around the facial nerve at its exit from the stylomastoid foramen. The electrode closest to the recording electrode was the negative lead (cathode). The CAP was recorded with a custom-made bipolar silver electrode system with an interelectrode distance that varied from 0.5 to 1.5 cm. The distance between stimulating electrodes and CAP recording electrodes averaged 4 cm. The CAP electrode was positioned to delicately lift the isolated buccal branch of the facial nerve to maximize electrode-nerve contact and minimize coupling of stimulus artifact from surrounding tissues. A bipolar concentric EMG electrode (Teca Corp., Pleasantville, N.Y.) was inserted into the zygomaticus major muscle to record the stimulus-evoked EMG. One end of a silk suture was passed through the lateral upper lip at the point of insertion of the zygomaticus major muscle fibers. The other end of the suture was attached to an SG force transducer aligned in the natural line of pull of the muscle. No specific muscle length adjustments[18] were made because the normal insertions of the studied muscle were left undisturbed.

The location of the electrodes and the position and tension of the SG were kept constant through the experiment. The signals from CAP and EMG electrodes were each differentially recorded and amplified (10,000 to 50,000 gain; bandpass, 30 Hz to 10 K Hz). The signal from the SG was amplified

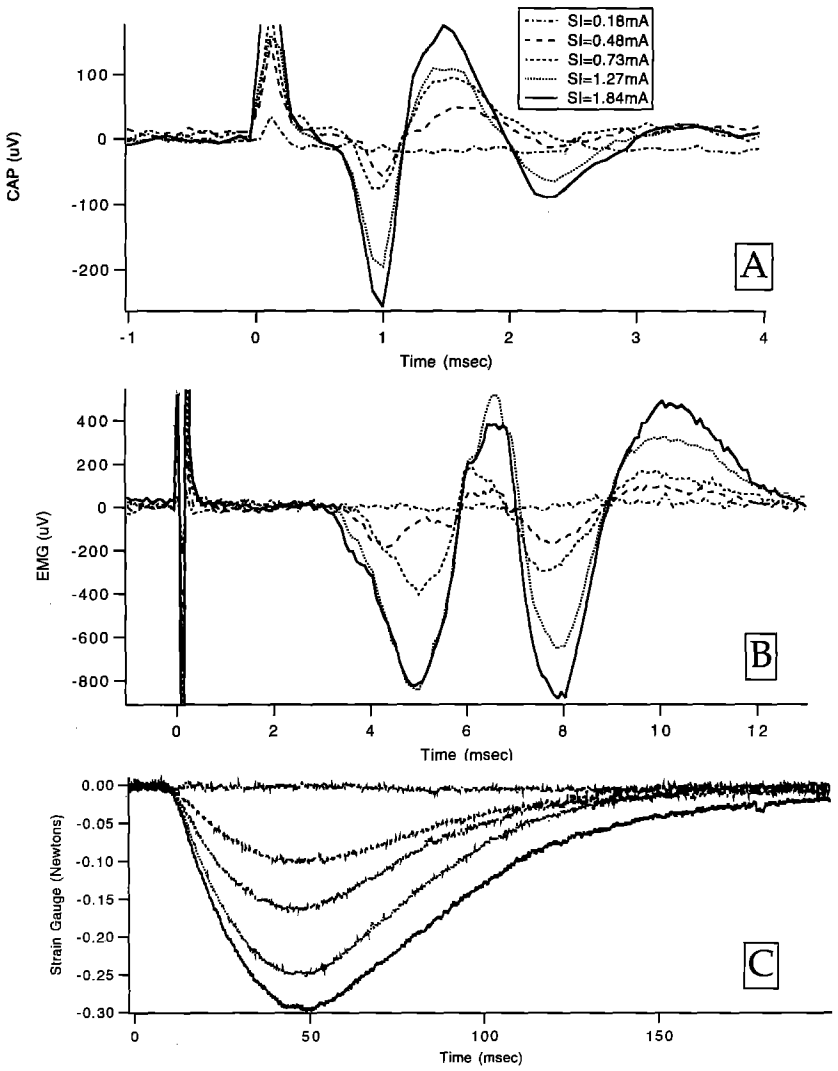

Fig. 4. Superimposed representative traces of CAP, EMG, and SG vs. time for varing stimulus intensities *(SI)* from subthreshold to maximal response in one animal.

(10× gain, low-pass filter at 3 kHz). After this preprocessing these signals were then brought to a Macintosh II FX (Apple Computer, Inc., Cupertino, Calif.) computer through an A/D board (MIO-16H; National Instruments, Austin, Tex.) with a resolution of 5 mV. Data acquisition was triggered by the output of a pulse timing unit (WPI-interval generator 1830 and WPI-pulse train module 1831), which also provided the timing, after a delay of 1 milliseconds, to a constant current stimulating unit.

The standard experimental procedure consisted of recording the CAP, EMG, and SG signals subsequent to electrical stimulation of the facial nerve trunk. Positive or negative electric pulses of variable intensities and a fixed duration of 100 microseconds were applied to the facial nerve at its exit from the stylomastoid foramen. The electric current was calculated from the voltage drop across a 1 kΩ resistor placed in series in the stimulating circuit. Several experimental manipulations were used to ascertain the physiologic nature of the recorded responses (see "Results").

Data collection and analysis were carried out with a custom module written within the Lab View software package (National Instruments). The largest peak was most often negative, and therefore the largest negative peak was chosen as the index peak. Peak amplitude (p), peak-to-peak amplitudes (p-p), and areas under the curves were computed. The p-p was calculated between the largest negative peak and the following positive one. To compare results across animals, electric stimuli and measures of

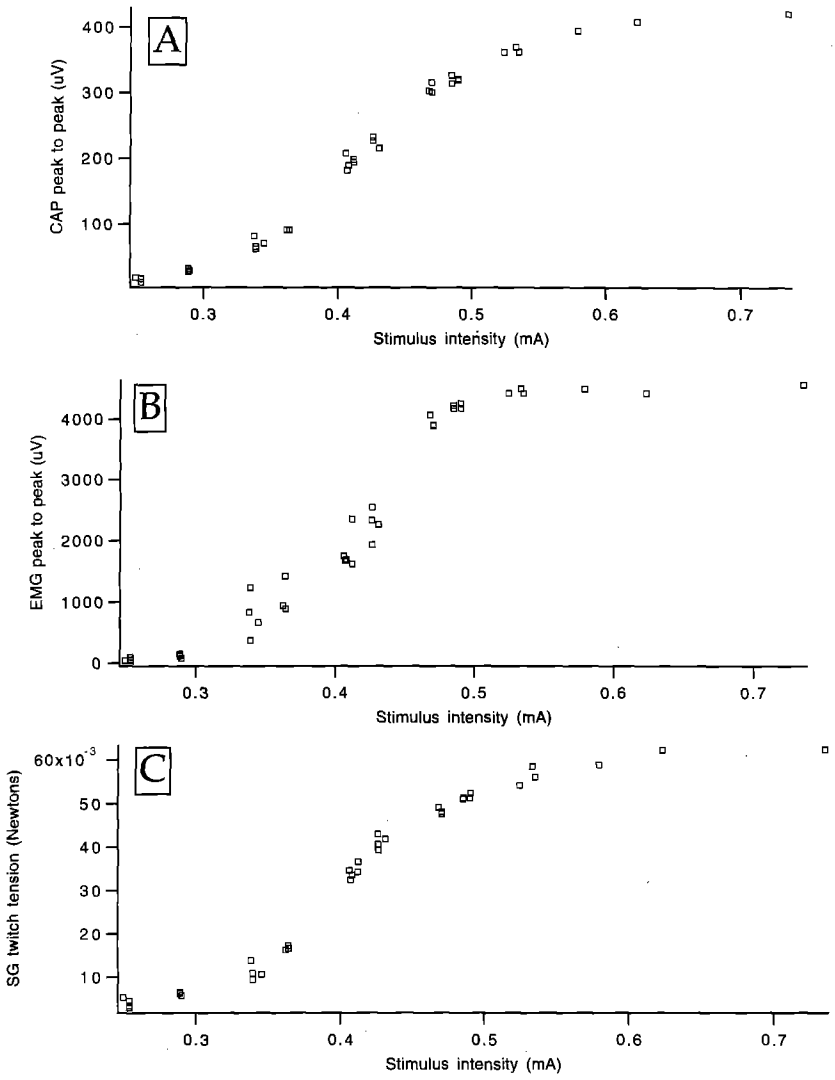

Fig. 5. A, $CAP_{p\text{-}p}$; B, $EMG_{p\text{-}p}$; and C, SG as functions of stimulus intensity in a single animal. At each intensity the electric stimulus was repeated 3 to 5 times to demonstrate the variability in the recorded responses.

CAP, EMG, and SG were normalized to percentage SG values according to those stimulus intensities that produced a given magnitude of SG response. In other words, percentage of SG values were established by setting the minimum as 0% and maximum as 100% of the SG response. Scaling of intermediate SG response value was accomplished according to the following formula:

$$SG(\%) = [(SG - SG_{min})/(SG_{max} - SG_{min})] \times 100$$

Stimulus intensity values were likewise normalized to those values producing a given SG(%), the normalized SG response. CAP_{max} and EMG_{max} were defined as those responses produced by the stimulus intensity producing SG_{max}.

Data plotting and curve fitting were done with the software program IGOR (Wave Metrics Inc., Lake Oswego, Ore.). The data were fitted with the best fit curve, and significance was tested using chi-squared statistics.

RESULTS

Figure 2 illustrates the temporal relationship of a typical CAP, EMG, and SG recording. To ascertain a clearer view of CAP and EMG, the first 14 milliseconds of the records in Fig. 2*A* are expanded in Fig. 2*B*. The overall shape of each trace is similar to previously published CAP and EMG recordings.[12,19,20] The first peak in both traces represents the stimulus artifact. The first negative peak in the CAP

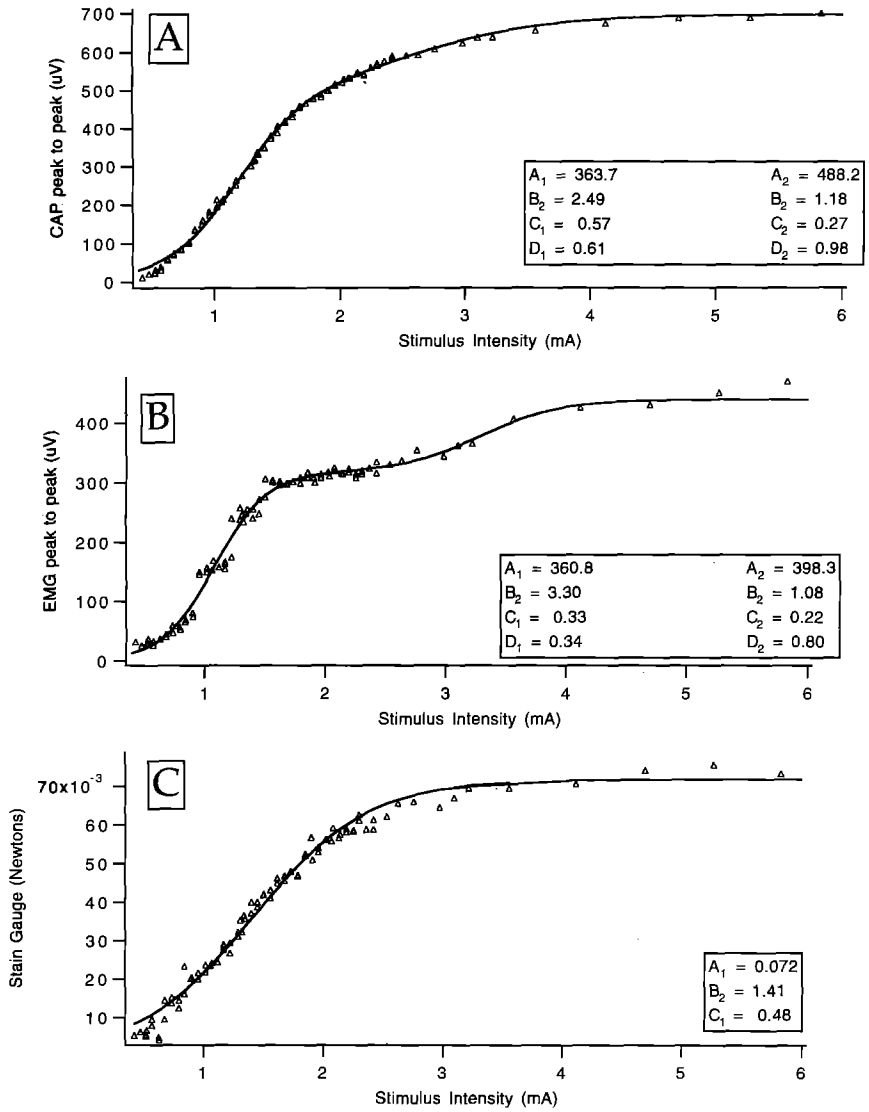

Fig. 6. CAP, EMG, and SG as a function of stimulus intensity in a representative animal (rabbit 17). The data were best fitted with a double sigmoid with the coefficients depicted on each graph. See text for formula.

recording occurred at 1.0 millisecond. Given an interelectrode distance of 4 cm, this implies an action potential speed of 40 m/second, which is within the known range of mammalian facial nerve fibers.[21,22] In the EMG recordings the largest negative peak occurred at a later time, with a delay of 4.8 milliseconds. The amplitude of the EMG signal is larger than the CAP signal by a factor of 3 to 4. The SG output peaked at a later time at about 50 milliseconds and had slower rise and fall times.

A variety of experimental manipulations illustrated in Fig. 3 were applied to a typical preparation to verify the physiologic nature of the recorded signals. Inverting the polarity of the electrical stimulus *(row B)* resulted in an inverted artifact (best seen in the CAP traces) and in smaller and delayed responses for all three measures. This difference in response was present at all stimulus intensities and resulted in different thresholds for normal and inverted electric stimuli. This pattern is consistent with the well-known anode block of nerve excitation.[23] Cutting the facial nerve branch between the CAP electrode and the muscle *(row C)* abolished the EMG and SG signals but preserved the CAP response. Only when the nerve was cut proximal to the CAP electrode *(row D)* was the CAP response eliminated. These findings are what one would expect physiologically because the EMG and SG are de-

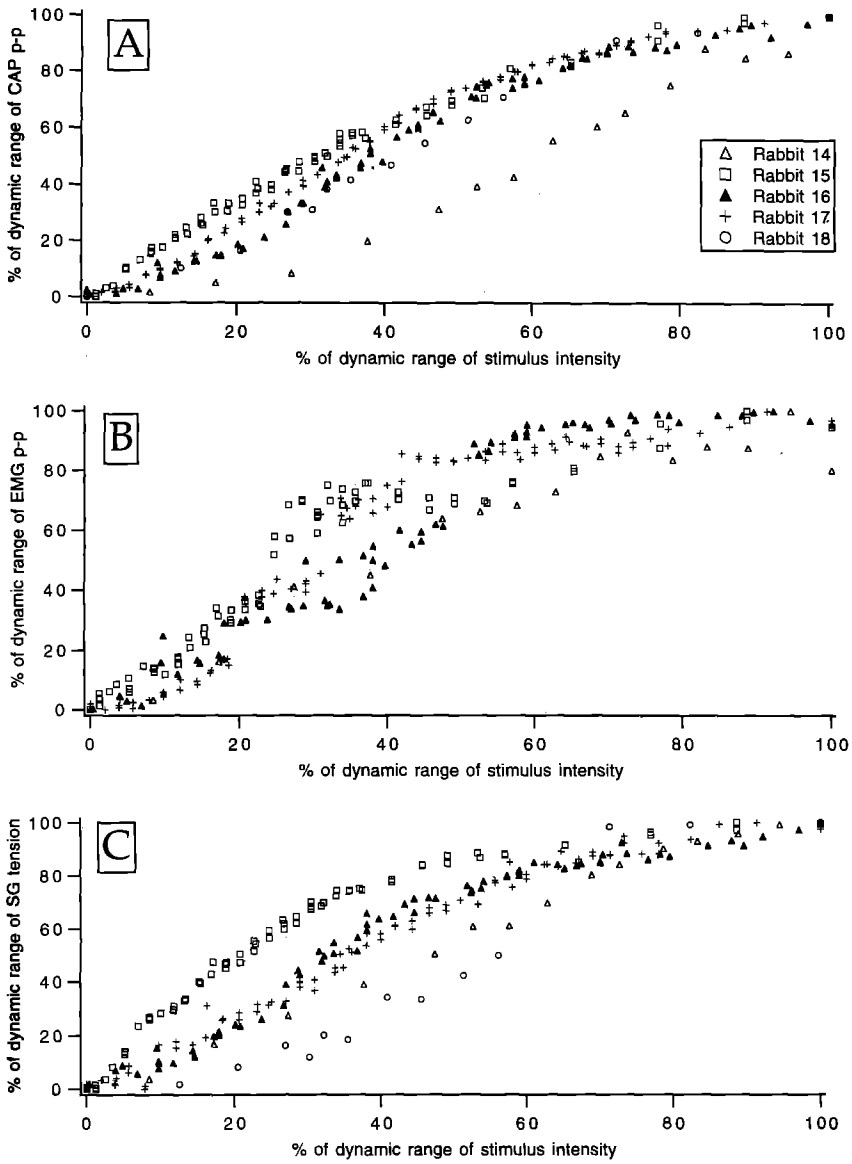

Fig. 7. Normalized CAP, EMG, and SG as a function of stimulus intensity in all rabbits studied.

pendent on the nerve conducting into the muscle through the neuromuscular junction.

Changes in CAP, EMG, and SG signals as progressively larger electric stimuli were applied to the preparation are shown in Fig. 4. The delay of various peaks varied little with stimulus intensity. This is to be expected because the nerve is stimulated directly, and the first motoneurons to be activated are the largest one and therefore have the fastest conducting axons.[24] With increasing stimulus intensities, progressively larger CAP, EMG, and SG signals were obtained.

The variability in these recorded responses in one animal is illustrated in Fig. 5. The stimulus intensity was increased in relatively large increments and repeated 3 to 5 times at each stimulus intensity. The $CAP_{p\text{-}p}$ and SG_p signals demonstrated low variability ($\pm 5\%$), whereas the $EMG_{p\text{-}p}$ variability was higher ($\pm 30\%$).

In Fig. 6 the $CAP_{p\text{-}p}$, $EMG_{p\text{-}p}$, and SG_p are plotted as functions of stimulus intensity for a typical animal preparation. The data were fitted with several types of mathematical functions, including a straight line, a polynomial curve, an exponential curve, a single sigmoid curve, and the sum of two sigmoid curves (data not shown). The best fit was

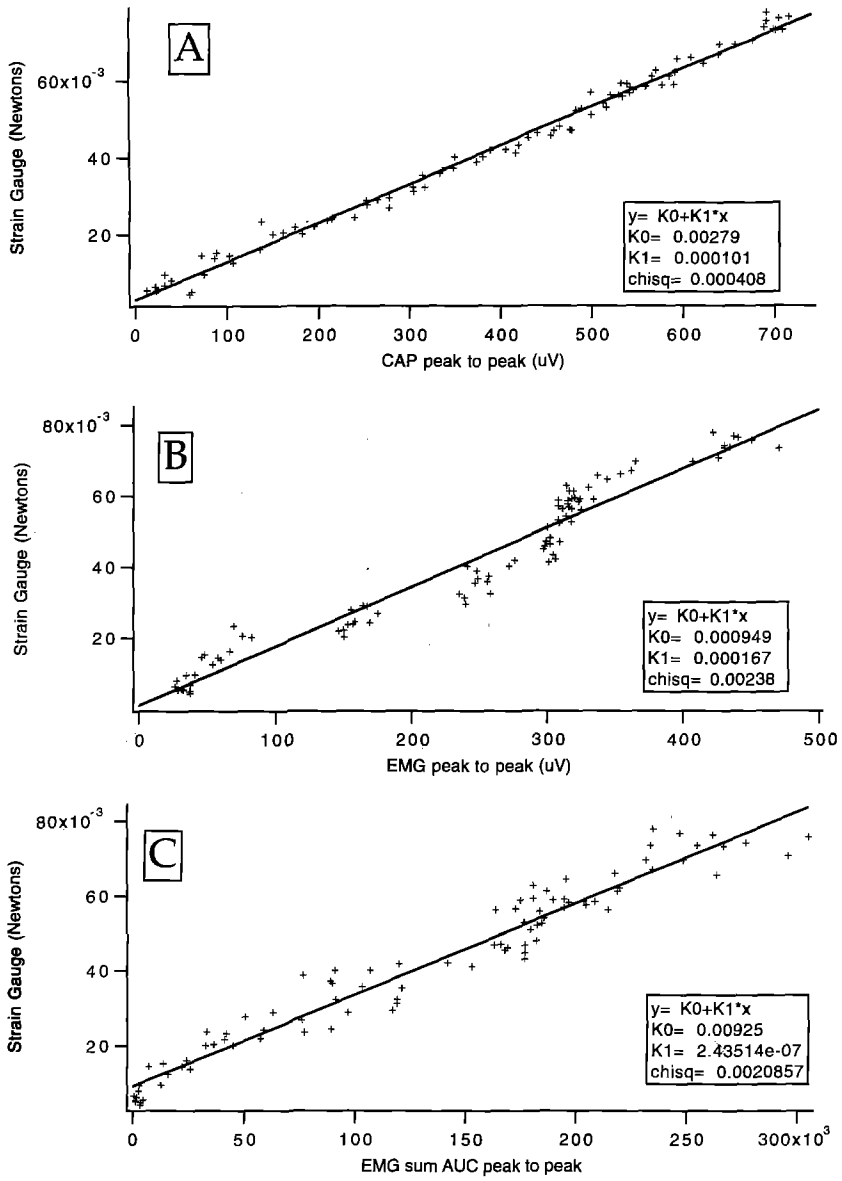

Fig. 8. SG peak twitch tension as a function of **A,** $CAP_{p\text{-}p}$; **B,** $EMG_{p\text{-}p}$; and **C,** EMG sum of the area under the curve *(AUC)* of the two largest peaks in a single animal (rabbit 17).

obtained with a double sigmoid curve for all three measures of the following form:

$$f(x) = \left[\left(\frac{A_1}{1 + e^{\left(\frac{-x + B_1}{C_1}\right)}} \cdot D_1\right) + \left(\frac{A_2}{1 + e^{\left(\frac{-x + B_2}{C_2}\right)}} \cdot D_2\right)\right]$$

The curve and its parameters are displayed on each graph. These results are similar to our findings in the frog sciatic nerve–gastrocnemius muscle preparation.[17]

Similar plots for five animals are shown in Fig. 7. The thresholds and dynamic range varied in individual animals, and the results were thus normalized to compare data across experiments. All measures from all animals exhibited sigmoid relations similar to the corresponding graphs shown in Fig. 6.

To examine the relationship between CAP or EMG and muscle contraction, $CAP_{p\text{-}p}$ and $EMG_{p\text{-}p}$ recordings were plotted against muscle twitch tension in Figs. 8 and 9. Data from a typical animal are shown in Fig. 8, and normalized data from several animals are plotted in Fig. 9. All relationships were best fitted by a straight line confirming the presence of a linear relationship between EMG and SG of previous studies on limb muscles.[12,13,17] The linear

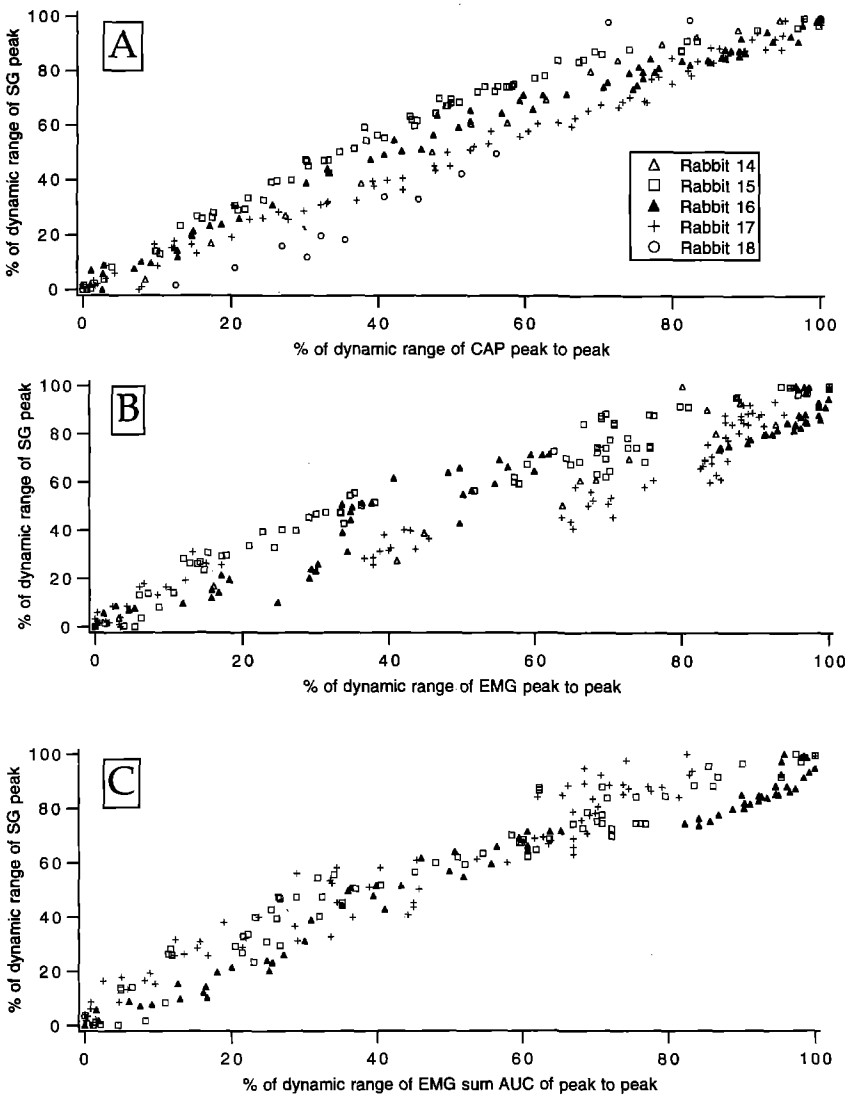

Fig. 9. Graphs of percentage of dynamic range of SG peak twitch tension vs. **A,** $CAP_{p\text{-}p}$; **B,** $EMG_{p\text{-}p}$; and **C,** EMG sum of the area under the curve *(AUC)* of the two largest peaks for all animals studied.

relationship of CAP and SG extends our findings in the frog to the facial neuromuscular system.

DISCUSSION

The ensemble of motoneurons innervating a given muscle is called a motoneuron pool. The recruitment pattern of motoneurons with a pool is organized according to Henneman's size principal[25]: the size of motoneurons correlates with their susceptibility to discharge. The motoneuron size has been correlated to the electrophysiologic threshold for discharge,[26] the velocity of its action potential,[24] the speed of contraction,[27] the tetanic tension of muscle fibers,[28] and even the discharge frequency of human motor units during voluntary contraction.[29] Small motoneurons are more numerous,[30] are more readily discharged physiologically, and fire more often than the large ones.[14,15,20,29] The force they generate is small,[28,30] and thus a fine gradation can be produced and precisely controlled by the recruitment of varying numbers of small motor units. With increasing demands, larger tensions are needed. At that point, larger excitatory inputs are present, and the larger units, which are physiologically less susceptible to discharge, can be fired. Their tension output is greater[20] and is produced more rapidly.[15,20,29]

Conversely, the recruitment during electrical stimulation is in a reverse order, because large-

diameter neurons have the lowest threshold to electrical excitation. Therefore, when CAP recordings and analysis are centered around the largest peak, they predominantly reflect the properties of large-diameter, fast fibers. Since the pioneering work of Gasser and Erlanger,[19] researchers have attempted to study and model the CAP waveform as the sum of individual action potentials to electrical [see References 31 for a review] and sensory stimulation.[32] Nevertheless, to our knowledge, a direct study of the relationship between CAP parameters and electrical stimulus intensity has not been conducted until recently.[17]

This study has demonstrated that the p-p of the electrically induced CAP of a facial nerve branch has a sigmoid-type relationship (Fig. 6*A*) with stimulus intensity. The fact that the data are best fitted with the sum of two sigmoid curves suggests that more than one population of facial motoneurons is present. These populations exhibit different electric stimulation thresholds and different action potential velocities. Previous single-fiber recordings from facial motoneurons in the cat support this point of view.[21,22]

The presence of a similar sigmoidal relationship of EMG (Figs. 6*B* and 7*B*) and SG (Figs. 6*C* and 7*C*) vs. stimulus intensity seems to imply that the facial neuromuscular system follows the general organization of other somatic motoneuron pools. At low levels of activity the recruited units will have a small fiber diameter and thus low physiologic and high electric thresholds. They will generate small EMGs and small contraction forces (small SG responses), as seen by the small changes in EMG and SG for large electric stimulation variations at high stimulation intensities. At progressively higher levels of activity, recruited units will have larger fiber diameters and thus higher physiologic and lower electrical stimulation thresholds. They will generate large EMGs and large contraction forces (large SG responses), as seen by the large changes in EMG and SG for small electrical stimulation variations at lower stimulation intensities (steep portion of the curves).

Because the relationships of CAP, EMG, and SG to stimulus intensity have similar shapes, it is not surprising to find a linear relationship between CAP or EMG and muscle tension. Similar linear relationships between EMG and SG have been found in limb muscles.[12,14,17,20] Also, this study confirms our previous findings of a linear relationship between CAP and SG in the frog sciatic nerve–gastrocnemius system. Activation of a motoneuron results in the contraction of all muscle fibers of the neuromuscular unit.[33] Our results suggest that the EMG produced and muscle force generated are proportional to the activation of the motoneuron.

The implications of these findings to future facial palsy electronic rehabilitation devices are significant. Recording the CAP or EMG will allow a direct prediction of the amount of muscle contraction some 40 milliseconds before it actually occurs (Fig. 2). If symmetric facial movements are the goal, a recording of CAP or EMG on the healthy side could provide a precise index of contralaterally occurring movements. This could be used as the input or afferent limb of a facial functional electric stimulator. Preliminary studies with contralateral EMG as input have given encouraging results.[16,34] Also, a delay of 40 milliseconds should be sufficient to handle data analysis and routing of signals to the paralyzed side, achieving therefore synchronous bilateral facial movements. Whether this delay is necessary or whether inputs based on the measure of facial contractions, as proposed by Broniatowski et al.,[9] are sufficient could not be addressed from our data.

Future work is needed to study a more physiologic CAP and EMG response, such as the one evoked during a facial reflex, and its relationship to twitch tension. The determination of the feasibility of such recordings is necessary before a facial functional electrical stimulation prosthesis could be built. Also, how twitch tension translates into tetanic tension in facial muscles needs to be investigated. The effect of facilitation at high stimulus frequencies also needs to be further studied. Finally, changes of facial muscle properties with denervation and reinnervation need to be evaluated.

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REFERENCES

1. Sunderland S, Cossar RF. The structure of the facial nerve. Anat Rec 1953;116:147-65.
2. Thomander L, Aldskogius H, Grant G. Motor fiber organization in the intratemporal portion of the facial nerve in the rat. In: Graham MD, House WF, eds. Disorders of the facial nerve. New York: Raven press, 1982:75-82.
3. Summerbell D, Stirling RB. The innervation of dorsoventrally reversed chick wings: evidence that motor axons do not actively seek out their appropriate targets. Journal of Embryology and Experimental Morphology 1981;61:233-47.
4. Weiss P, Taylor AC. Further experimental evidence against "neurotropism" in nerve regeneration. J Exp Zool 1944;95: 233-57.

5. Rubin LR. The paralyzed face. St. Louis: Mosby–Year Book, Inc., 1991.
6. Kralj A, Bajd T, Turk R, Benko H. Posture switching for prolonging functional electrical stimulation standing in paraplegic patients. Paraplegia 1986;24:221-30.
7. Smith B, Peckham PH, Keith MW, Roscoe DD. An externally powered multichannel, implantable stimulator for versatile control of paralyzed muscle. IEEE Trans Biomed Eng 1987; BME-34:499-508.
8. Zealar DL, Dedo HH. Control of paralyzed axial muscles by electrical stimulation. Trans Am Acad Ophthalmol Otolaryngol 1977;84:ORL-310.
9. Broniatowski M, Grundfest-Broniatowski S, Davies R, et al. Dynamic rehabilitation of the paralyzed face. III. Balanced coupling of oral and ocular musculature from the intact side in the canine. Otolaryngol Head Neck Surg 1991;105:727-33.
10. Bruesch Sr. The distribution of myelinated afferent fibers in the branches of the cat's facial nerve. J Comp Neurol 1944; 81:169-91.
11. Nordin M, Hagbarth KE, Thomander L, Wallin U. Microelectrode recordings from the facial nerve in man. Acta Physiol Scand 1986;128:379-87.
12. Bigland B, Lippold OJ. The relation between force, velocity and integrated electrical activity in human muscles. J Physiol 1954;123:214-24.
13. Milner-Brown HS, Stein RB. The relation between the surface electromyogram and muscular force. J Physiol 1975;246: 549-69.
14. Tanji J, Kato M. Recruitment of motor units in voluntary contractions of a finger muscle in man. Exp Neurol 1973;40: 759-70.
15. Freund HJ, Budingen HJ, Dietz V. Activity of single motor units from human forearm muscles during voluntary isometric contractions. J Neurophysiol 1975;38:933-46.
16. Rothstein J, Berlinger NT. Electronic reanimation of facial paralysis—a feasibility study. Otolaryngol Head Neck Surg 1986;94:82-5.
17. Dulguerõv P, Goodnight JW, Berke GS, Hoffman LF. Can we predict muscle contractions? Lessons from the frog sciatic nerve and implications for functional electric stimulation of facial muscles. St. Petersburg Beach: ARO, 1993.
18. Buller AJ, Eccles JC, Eccles RM. Differentiation of fast and slow muscles in the cat hind limb. J Physiol 1960;150: 399-416.
19. Gasser HS, Erlanger J. The role played by the sizes of constituent fibers of a nerve trunk in determining the form of its action potential wave. Am J Physiol 1927;80:522-47.
20. Milner-Brown HS, Stein RB, Yemm R. Changes in firing rate of human motor units during linearly changing voluntary contraction. J Physiol 1973;230:371-90.
21. Kitai ST, Tanaka T, Tsukahara N, Yu H. The facial nucleus of cat: antidromic and synaptic activation and peripheral nerve representation. Exp Brain Res 1972;16:161-83.
22. Fanardjian VV, Manvelyan LR, Kasabyan SA. Mechanisms regulating the activity of facial nucleus motoneurons. 1. Antidromic activation. Neuroscience 1983;9:815-22.
23. Lorente de No R. A study of nerve physiology. New York: Rockefeller Institute for Medical Research, 1947.
24. Hursch JB. Conduction velocity and diameter of nerve fibers. Am J Physiol 1939;127:131-9.
25. Henneman E. Relation between size of neurons and their susceptibility to discharge. Science 1957;126:1345-7.
26. Luscher H-R, Ruezel P, Henneman E. How the size of motoneurons determines their susceptibility to discharge. Nature 1979;282:859-61.
27. Bessou P, Emonet-Denand F, Laporte Y. Relation entre la vitesse de conduction des fibres nerveuses motrices et le temps de contraction de leurs unites motrices. R Acad Sci (Paris) 1963;256:5625-7.
28. Jami L, Petit J. Correlation between axonal conduction velocity and tetanic tension of motor units in four muscles of the cat hind limb. Brain Res 1981;229:193-6.
29. Borg J, Grimby L, Hannerz J. Axonal conduction velocity and voluntary discharge properties of individual short toe extensors motor units in man. J Physiol 1978;277:143-52.
30. Henneman E, Olson CB. Relations between structure and function in the design of skeletal muscles. J Neurophysiol 1965;28:581-98.
31. Schoonhoven R, Stageman DF. Models and analysis of compound action potentials. Crit Rev Biomed Eng 1991;19:47-111.
32. Kiang NYS, Moxon EC, Kahn AR. The relationship of gross action potentials recorded from the cochlea to single unit activity in the auditory nerve. In: Ruben R, Elberling C, Salomon G, eds. Electrocochleography. Baltimore: University Park Press, 1976:95-115.
33. Burke RE. Motor units: anatomy, physiology, and functional organization. In: Brooks VB, ed. Handbook of physiology. Section 1: The nervous system. Vol. 2: Motor control. Bethesda, Md.: American Physiological Society, 1980:345-422.
34. Tobey DN, Sutton D. Contralaterally elicited electrical stimulation of paralyzed facial muscles. Otolaryngol Head Neck Surg 1978;87:812-8.