

# Accuracy Limitations of Chronaxie Values

Leslie A. Geddes, *Life Fellow, IEEE*

**Abstract**—The strength-duration curve is a plot of the threshold current ( $I$ ) versus pulse duration ( $d$ ) required to stimulate excitable tissue. On this curve are two points: 1) rheobase ( $b$ ) and 2) chronaxie ( $c$ ). Rheobase is the threshold current for an infinitely long-duration stimulus. Chronaxie, the excitability constant, is the duration of a pulse of current of twice rheobasic strength. The mathematical expression for the strength-duration curve is  $I = b(1 + c/d)$ . Although there are many published values for chronaxie for various excitable tissues, the range of variability for a given tissue type is quite large. This paper identifies five factors that can affect the accuracy of chronaxie measurement and shows that the most reliable values can be obtained with a rectangular pulse delivered from a constant-current source.

**Index Terms**—Chronaxie, rheobase, stimulation, tissue excitability.

## I. INTRODUCTION

**C**HRONAXIE is the tissue-excitability parameter that permits choice of the optimum stimulus pulse duration for stimulation of any excitable tissue. Chronaxie ( $c$ ) is the Lapique descriptor of the stimulus pulse duration for a current of twice rheobasic ( $b$ ) strength, which is the threshold current for an infinitely long-duration stimulus pulse. Lapique showed that these two quantities ( $c, b$ ) define the strength-duration curve for current:  $I = b(1 + c/d)$ , where  $d$  is the pulse duration. However, there are two other electrical parameters used to describe a stimulus: energy and charge. The minimum energy occurs with a pulse duration equal to chronaxie. Minimum charge ( $bc$ ) occurs with an infinitely short-duration pulse. Choice of a pulse duration equal to  $10c$  requires a current of only 10% above rheobase ( $b$ ). Choice of a pulse duration of  $0.1c$  requires a charge of 10% above the minimum charge ( $bc$ ).

The published range of values for chronaxie have largely ignored five factors that can affect the value for chronaxie: 1) stimulus current waveform; 2) electrode properties; 3) stimulator output impedance; 4) tissue inhomogeneity; and 5) temperature. Examples are given to identify the effect of each factor. It is concluded that one or more of these factors are likely the reason for the variability of chronaxie values in the published literature.

## II. BACKGROUND

From time to time, statements are made that chronaxie, the excitability constant of tissue, is affected by electrode parameters (size, shape, material) and stimulus waveform [13].

Chronaxie ( $c$ ) is the duration for a rapidly rising current pulse of twice rheobasic intensity. The rheobase ( $b$ ) is the threshold

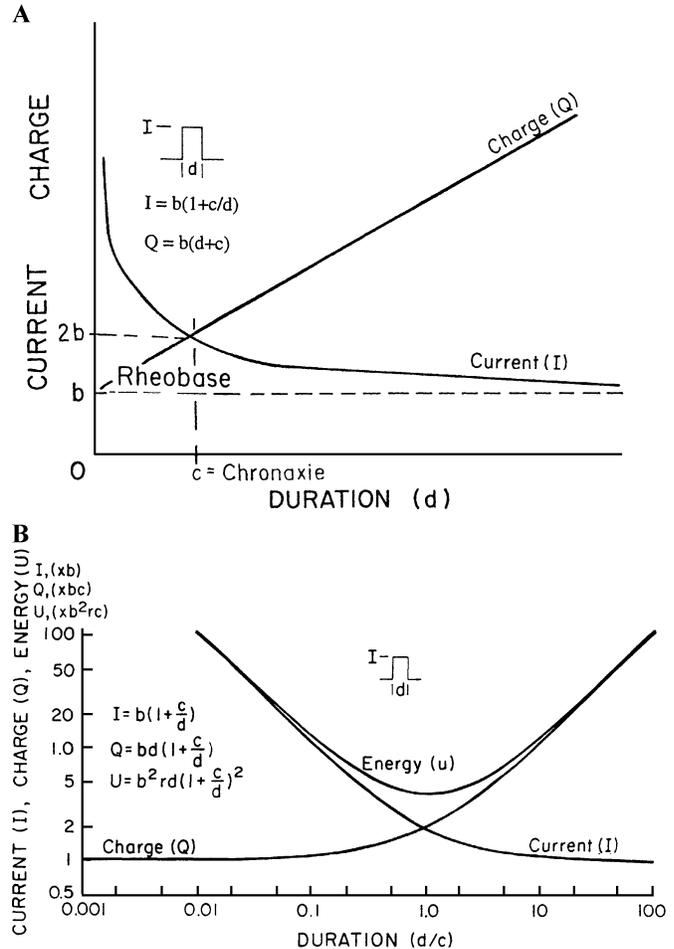


Fig. 1. (a) Lapique hyperbolic strength-duration ( $d$ ) curve for current ( $I$ ) and the Weiss linear strength-duration relationship for charge  $Q = Id$ . In (b) are shown universal strength-duration curves, plotted logarithmically, for current energy and charge, with the duration axis divided by  $c$ , the chronaxie.

current ( $I$ ) for stimulation with an infinitely long-duration pulse. Lapique [16], [17] showed that these two quantities describe completely the current strength ( $I$ )-duration ( $d$ ) curve, namely  $I = b(1 + c/d)$ . Fig. 1(a) represents the Lapique strength-duration curve for current for a rectangular pulse. Lapique used constant-current, capacitor-discharge pulses to obtain chronaxie values for a wide variety of excitable tissues. The duration ( $d$ ) was the time constant of the electrode-subject circuit ( $RC$ ), where  $R$  was a series resistor, (much higher than that of the electrode-subject impedance) and  $C$ , the capacitance, was chosen to obtain the desired pulse-duration range.

Earlier, Weiss [23] used rectangular, constant-current pulses for the same purpose and found that the threshold charge ( $Q = Id$ ) required for stimulation increased linearly with pulse duration. Fig. 1(a) shows the Weiss charge-duration relationship.

Manuscript received June 10, 2002; revised December 11, 2002.

The author is with the Department of Biomedical Engineering, Purdue University, West Lafayette, IN 47907-1296 USA (e-mail: geddes@ecn.purdue.edu).

Digital Object Identifier 10.1109/TBME.2003.820340

It is noteworthy that the charge for the Lapique expression ( $Q = bd + bc$ ) is linear with pulse duration ( $d$ ). The slope is  $b$ , and the intercept is  $bc$ , which is the minimum charge. Therefore, the Weiss and Lapique results carry the same message.

It is of value to create a universal strength-duration curve by dividing the duration ( $d$ ) axis by chronaxie ( $c$ ) and plotting the current, charge and energy versus duration logarithmically, as shown in Fig. 1(b). It is easily shown that the minimum energy in a pulse occurs with a pulse duration equal to chronaxie.

### III. FACTORS AFFECTING CHRONAXIE

Five factors affect the practical measurement of values for chronaxie: 1) stimulus waveform; 2) electrode characteristics; 3) stimulator output impedance; 4) tissue inhomogeneity; and 5) temperature. Examples will be given to identify the nature of each factor.

### IV. STIMULATION OF EXCITABLE TISSUE

To understand the influence of the five factors identified above, it is first necessary to establish the principles underlying excitation. Excitable cells are enveloped by a semipermeable membrane. Inside the cell is a high concentration of potassium ions ( $K^+$ ). Outside the cell is a high concentration of sodium ions ( $Na^+$ ). This ionic gradient is maintained by metabolism, the result being a resting transmembrane potential (RMP), the magnitude of which depends on the cell type. Typically the resting membrane potential is about 80 mV, with the outside positive with respect to the inside. The membrane permeability is a function of the transmembrane potential.

Fig. 2(a) illustrates the transmembrane charge distribution for a long cylindrical excitable cell, to which a rectangular wave cathodal current pulse of intensity  $I$  and duration  $d$  is applied. This cathodal current pulse removes positive charge and reduces the transmembrane potential by  $\Delta V$  to reach the threshold potential (TP), which produces a propagated action potential, during which a small number of  $Na^+$  ions enter the cell and  $K^+$  ions exit the cell. Fig. 2(b) illustrates the resulting action potential. Note that the transmembrane potential becomes slightly higher than zero at the peak of the action potential.

Fig. 2(a) depicts monopolar stimulation in which a small-area cathodal electrode is applied to the excitable tissue. The return path for the current is via a distant, large-area indifferent (reference or dispersive) electrode.

To reduce the transmembrane potential, positive charge is removed by the cathodal stimulus. For a rectangular pulse, charge ( $Q$ ) is the product of current ( $I$ ) and duration ( $d$ );  $Q = Id$ . Therefore, it would be expected that there should be a reciprocal relationship between current and duration over a range of durations; Fig. 1(a) illustrates this point.

Fig. 2 depicts the case in which the stimulating current ( $I$ ) was at threshold value. Hodgkin [12] investigated the temporal transmembrane potential changes for subthreshold and threshold values for cathodal and anodal stimuli. Fig. 3 illustrates that with cathodal pulses of current of incrementing intensity (below threshold), the transmembrane potential decreased; then it returned to the resting membrane potential

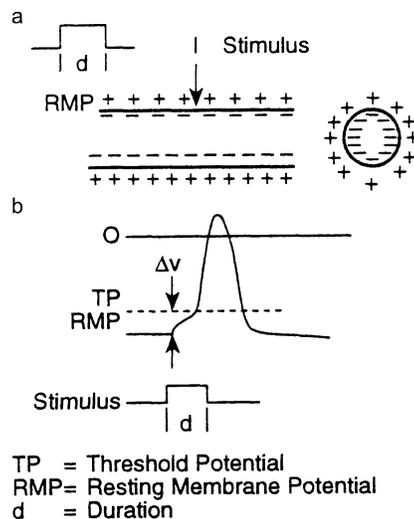


Fig. 2. The charge distribution along a cylindrical excitable cell with a cathodal stimulating electrode delivering a rectangular current pulse of intensity  $I$  and duration  $d$ . The mechanism of stimulation in which the current pulse reduces the RMP to the TP to produce a propagated action potential is shown in B.

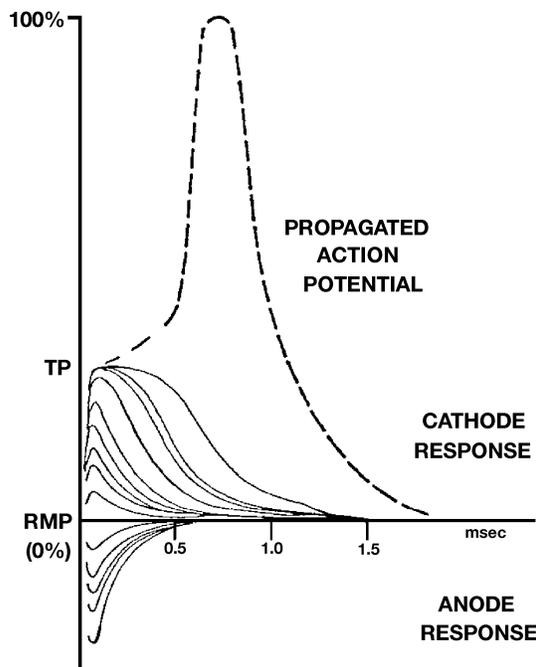


Fig. 3. Local potential changes under the cathode and anode with increasing stimulus intensity. Note that under the cathode, when the stimulus intensity reduced the membrane potential from the RMP to the TP, excitation occurred. Excitation did not occur under the anode with increasing stimulus intensity. (Redrawn from [12]).

with a time constant. With a sufficiently strong cathodal pulse, the TP was reached and a propagated action potential ensued.

With anodal pulses, the transmembrane potential increased, and then returned to the resting membrane potential with a time constant. Increasing the anodal pulse intensity incrementally increased the transmembrane potential transiently. However, no propagated action potential occurred with increasing anodal stimulus strength.

## V. STIMULUS WAVEFORM

From Fig. 2(b), it is clear that a rapidly rising, rectangular cathodal current pulse delivers the charge necessary for stimulation effectively. However, the temporal aspect of charge delivery is important because the cell tries to maintain its membrane potential. Prolongation of the rising phase of a stimulus allows time for the cell to resist the imposed change. With continued rise of the applied current, there comes a time when the cell can no longer resist this imposed change and excitation occurs at a higher stimulus intensity. This phenomenon is known as accommodation and was well described by Hill [11].

The temporal aspects of charge delivery by a current pulse were investigated by Wessale *et al.* [24] who compared stimulation threshold with a rectangular pulse and a capacitor-discharge pulse, both delivered by the same constant-current stimulator. In human subjects they found that the chronaxie values for sensation with skin-surface electrodes were different. The charge delivered for the rectangular wave is  $I_d$ , that delivered by the capacitor discharge is  $I\tau$ , where  $I$  is the peak current and  $\tau$  is the time constant, the time required for the current to fall to 37% of its peak value. The chronaxie obtained from the capacitor-discharge strength-duration curve was about twice that for the rectangular wave pulse. In addition, the ratio of the threshold peak capacitor-discharge to the rectangular-wave current pulse increased with decreasing pulse duration, indicating that the temporal delivery of charge is an important factor.

## VI. ELECTRODE CHARACTERISTICS

Electrodes separate the excitable tissue from the stimulator. Excitation occurs under the cathode, and because it is current density ( $\text{mA}/\text{cm}^2$ ) that stimulates, the cathode electrode is made smaller than the reference or indifferent electrode. If the cathodal electrode is distant from the excitable tissue, the rheobase will be higher. Therefore the current strength-duration curve will have the same shape, but it will lie above that obtained with an electrode that is closer to the excitable tissue.

The equivalent circuit for an electrode-tissue interface has evolved over many years [10]. Briefly, it consists of three components, a half-cell potential, resistance, and capacitance. For stimulation, the half-cell potential can be neglected. The simplest equivalent circuit is shown in Fig. 4;  $R_f$  is the Faradic resistance, which provides the nonlinear direct-current path [8] with the subject.  $R_w$  and  $C_w$  are the Warburg components that result when a metal electrode comes into contact with an electrolyte.  $R_f$ ,  $R_w$ , and  $C_w$  are all nonlinear circuit elements.  $R_f$  and  $R_w$  decrease with increasing electrode area and  $C_w$  increases with increasing electrode area. Because  $R_f$ ,  $R_w$ , and  $C_w$  are nonlinear, the electrode-subject impedance is a function of the current. The tissue is represented by resistances ( $R_t$ ,  $R_t^1$ ) and capacitance ( $C_t$ ), the latter being due to the cell membranes.

The best way to minimize stimulus waveform distortion that the electrode components can produce is to use a stimulator with a high output impedance, i.e., a constant-current stimulator. In this way, the impedance of the electrode-subject circuit will be negligible with respect to the output impedance of the stimulator. This subject is discussed in the section dealing with stimulator output characteristics.

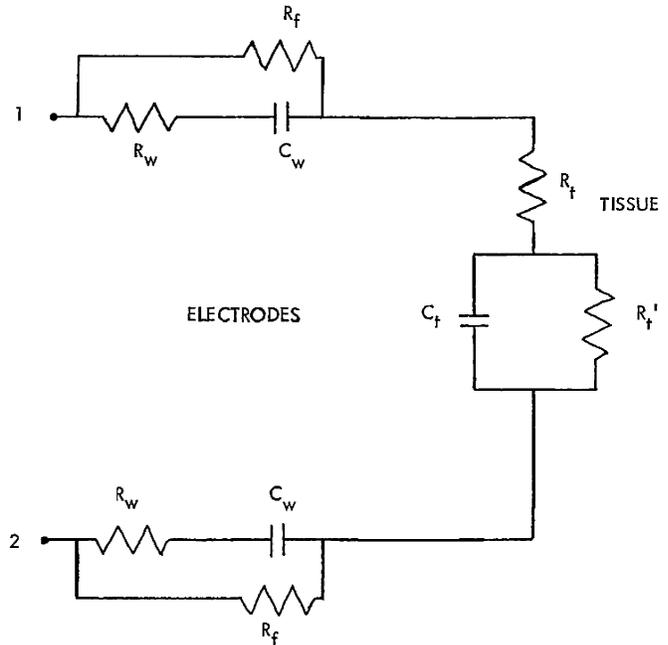


Fig. 4. Representation of a pair of electrodes on a subject;  $R_w$  and  $C_w$  represent the Warburg resistance and capacitance and  $R_f$  represents the Faradic resistance, all result from the contact between a metal electrode and an electrolyte. The subject's tissues are represented by ( $R_t$ ), ( $R_t^1$ ), and ( $C_t$ ). The resistances are due to the tissue fluids and the capacitance is due to the cell membranes.

The subject of electrode area became of considerable practical importance in the early days of implanted cardiac pacemakers. Because it is current density ( $\text{mA}/\text{cm}^2$ ) that stimulates, Furman *et al.* [2], [3] found that decreasing the pacing electrode area achieved pacing with less current, thereby increasing the longevity of the battery in an implanted pacemaker. The same fact applies for other types of implanted stimulators.

Another important aspect of a stimulating electrode is its shape. For all electrodes other than spherical, the current density across the electrode surface is not uniform, that under the perimeter being much higher than under the center of the electrode [1], [20]. Therefore, stimulation will occur somewhere under the electrode perimeter where the cathodal current density is highest.

## VII. STIMULATOR OUTPUT IMPEDANCE

Because the electrical circuit formed by the electrode-tissue interface (Fig. 4) resembles a nonlinear leaky capacitor, the output impedance of the stimulator will have an effect on the stimulus waveform. Briefly there are two types of output circuit: 1) constant voltage and 2) constant current. With the former type, the output voltage will be the same for all loads connected to it, i.e., the output impedance is low. With the constant-current stimulator, the current will be the same for all loads connected to it, i.e., the output impedance is high. However, with the constant-voltage stimulator, the current waveform will not be the same as the voltage waveform, owing to the complex electrode-subject impedance, which contains nonlinear resistive and capacitive elements.

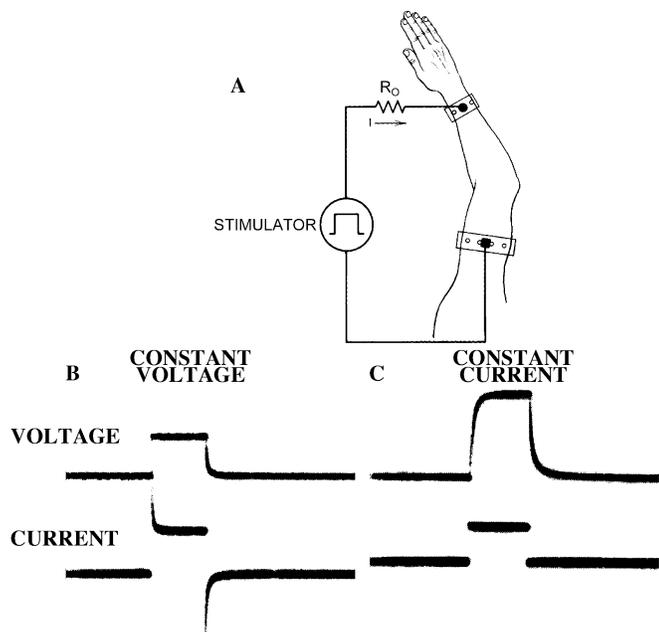


Fig. 5. In A is shown a rectangular-wave stimulator with an output impedance of  $R_o$  connected to electrodes on the arm. In B are shown the voltage and current waveforms obtained with a constant-voltage stimulator ( $R_o$  small). Note the peak on the current waveform. In C are shown the voltage and current waveforms when using a constant-current stimulator ( $R_o$  large) delivering a rectangular current waveform.

With the constant-current stimulator, the voltage waveform will not be the same as the current waveform. Because it is current that removes positive charge from a cell membrane to achieve excitation, it is necessary to focus on the current waveform.

An easy way of illustrating the importance of stimulator output impedance is to stimulate a tissue with a small-area (active cathodal) and large-area (indifferent anodal) electrode using a rectangular wave delivered from a constant-voltage and then a constant-current stimulator connected to the same electrodes. Fig. 5(a) illustrates these connections to a constant-voltage stimulator ( $R_o$  low) and then to a constant-current stimulator ( $R_o$  high). Fig. 5(b) shows the voltage and current waveforms for the constant-voltage stimulator and Fig. 5(c) shows the voltage and current waveforms for a constant-current stimulator. Note the high peak on the current waveform when a constant-voltage, rectangular-wave stimulator was used. Because the stimulator was a constant-voltage type, the voltage waveform is not distorted. Distortion of the current waveform is due to the capacitive nature of the electrode-electrolyte tissue interface. The spiked current makes it difficult to quantitate the current that stimulated.

With the constant-current stimulator shown in Fig. 5(c), note the undistorted current waveform. The constant-current stimulator is insensitive to the nature of the electrode-electrolyte tissue interface impedance. The capacitive nature of this interface reveals itself by producing an exponential rise and fall on the voltage waveform.

### VIII. TISSUE INHOMOGENEITY

When an electrode is applied to excitable tissue to determine its chronaxie, it is assumed that the tissue is homogeneous. However, a specimen of excitable tissue may contain several types of excitable tissue; therefore, the location of the stimulating electrode becomes an important consideration. For example, if an electrode is inserted into skeletal muscle and muscle twitch is used as the response to the stimulus, the chronaxie so obtained will be that of the motor nerves which spread through the muscle. To obtain the chronaxie of skeletal muscle fibers alone, a myoneural blocking agent must be used. In fact, there are three excitable tissues in skeletal muscle, each with its own chronaxie: 1) the motor nerve; 2) the myoneural junction; and 3) the skeletal muscle fibers. Interestingly, before clinical electromyography was introduced by Jasper *et al.* [14], the diagnosis of motor-nerve injury and reinnervation was made by measuring chronaxie over several months. After nerve injury in man, the chronaxie becomes long, being that of the denervated skeletal muscle. During reinnervation, the chronaxie becomes shorter, ultimately becoming that of the motor nerve when innervation is complete [21].

When the chronaxie of nerve is measured, it is important to recognize that most nerve trunks contain bundles of fibers having different diameters and, hence, different propagation velocities [4] and [5], with each fiber group having its own chronaxie. The indicator of excitation in nerve is the evoked action potential and an adequate length of nerve is needed so that the action potentials of all of the differently propagating fiber groups will be displayed separated by an adequate time interval. When this is achieved, the chronaxie for each fiber group can be obtained by choosing a pulse duration and increasing the pulse intensity until the action potential for a particular fiber group appears. The intensity for the appearance of each fiber group is identified as threshold and the procedure is repeated for other pulse durations. In this way a strength-duration curve can be plotted for each fiber group, from which the chronaxies can be determined. This technique was described by Smith *et al.* [22] to determine the chronaxie values for vagal nerve fibers. Failure to have a long enough nerve segment may only allow identification of the chronaxie for the fastest propagating fibers.

Another example of complex excitable tissue is heart muscle, namely the atria and ventricles. Within the atria is the sinoatrial node with its own chronaxie. The atrial muscle also contains propagating fibers that are a modified form of atrial muscle. The atria also contain autonomic nerve fibers. Likewise, the ventricles contain the A-V node, bundle of His, Purkinje fibers and sympathetic nerves, all embedded at different places in ventricular muscle, each with its own chronaxie. Therefore, electrode location and specimen choice are important considerations when measuring chronaxie.

The foregoing discussion identifies the importance of electrode location when determining the chronaxies of the components of an inhomogeneous excitable tissue. The statement made by Irnich [13] concerning electrode size as a factor in determining chronaxie, may be more due to electrode location rather than size. In fact Lucas [19], using frog and toad skeletal muscle, pointed out the importance of electrode location in distinguishing the chronaxie of nerve and muscle.

## IX. TEMPERATURE

All metabolic processes are influenced by temperature and it is not surprising that chronaxie is temperature dependent. Lapique [15], [18] was well aware of the effect of temperature on chronaxie. Geddes and Bourland [6] and [7], in an intact dog study in which body temperature was lowered to 23 °C, ventricular muscle time constant (analog of chronaxie) increased 5.9% per degree reduction in body temperature. Therefore, when reporting a value for chronaxie, temperature must be specified.

## X. DISCUSSION

In a recent paper, the author [9] listed the values for chronaxie from the published literature. In view of the five factors that can affect the accuracy of chronaxie, it is useful to examine the range of chronaxie values for the same tissue.

In the aforementioned paper [9], the chronaxie values for mammalian ventricles at body temperature range from 0.5 ms (human) to 2.0 to 4.1 ms (dog); this is a 8.2/1 ratio. The measurements were taken with different types of electrodes and with stimulators having unknown output impedances.

The chronaxie values for human arm sensory nerves range from 0.35 to 1.17 ms, a ratio of  $1.17/0.35 = 3.3$ . Again, the values were obtained with insufficient information to establish the cause of variability.

The chronaxie values for human denervated skeletal muscle ranges from 9.5 to 30 ms at body temperature, representing a ratio of 3.16. As stated earlier, a reduction in chronaxie occurs during reinnervation, as was elegantly shown by Ritchie [21]. A chronaxie study of mammalian skeletal muscle to which a myoneural blocking agent is applied would shed light on this situation.

Therefore, it is clear that the published values for chronaxie have a wide range. If chronaxie is the best descriptor of tissue excitability in a homogeneous tissue specimen, at a known temperature, it should be determined with a constant-current stimulator providing a rectangular cathodal stimulus waveform.

Because the published chronaxie values exhibit variability, it is logical to ask the question: Of what value is chronaxie? The answer is simple; chronaxie is derived from the strength-duration curve for current and it shows that if the stimulus duration is shorter than chronaxie, more current is required to stimulate, with any type or location of electrodes with a stimulator of any known or unknown output impedance.

In addition, the chronaxie value, however determined, identifies the pulse duration for minimum energy. In addition, the charge delivered at chronaxie, however determined, is  $2bc$ , twice the minimum charge. Therefore, if minimum charge delivery is sought to prolong the life of a battery in an implanted stimulator, a pulse duration of less than the measured chronaxie should be selected; a duration of one-tenth chronaxie provides a charge that is only 10% above the minimum charge.

## XI. CONCLUSION

Although the published literature fails to provide a narrow range of chronaxie values for a specific excitable tissue at a

known temperature, the concept of chronaxie and rheobase provide a basis for predicting the change in current, energy, and charge of the pulse when duration is changed.

## ACKNOWLEDGMENT

The author would like to thank R. Roeder Ph.D. for her assistance.

## REFERENCES

- [1] P. N. Caruso, J. A. Pearce, and D. P. DeWitt, "Temperature and current density distributions at electrosurgical dispersive electrode sites," in *Proc. N. Eng. Bioeng. Conf.*, vol. 7, 1979, pp. 373–376.
- [2] S. Furman, J. Garvey, and P. Hurler, "Pulse duration variation and electrode size as factors in pacemaker longevity," *J. Thoracic Cardiovasc. Surg.*, vol. 69, no. 3, pp. 382–389, 1975.
- [3] S. Furman, B. Parker, and D. Eicher, "Decreasing electrode size and increasing the efficiency of cardiac stimulation," *J. Surg. Res.*, vol. 11, pp. 105–110, 1971.
- [4] H. S. Gasser and J. Erlanger, "The role played by the sizes of the constituent fibers of a nerve trunk in determining the form of its action potential wave," *Amer. J. Physiol.*, vol. 80, pp. 522–547, 1927.
- [5] —, "A study of the action currents of nerve with the cathode ray oscilloscope," *Amer. J. Physiol.*, vol. 62, pp. 496–524, 1922.
- [6] L. A. Geddes and J. D. Bourland, "The strength-duration curve," *IEEE Trans. Biomed. Eng.*, vol. BME-32, pp. 458–459, June 1985.
- [7] —, "Tissue stimulation: theoretical considerations and practical applications," *Med. Biol. Eng.*, vol. 23, no. 2, pp. 131–137, 1985.
- [8] L. A. Geddes and R. Roeder, "Measurement of the direct-current (Faradic) resistance of the electrode-electrolyte interface," *Ann. Biomed. Eng.*, vol. 29, pp. 181–186, 2001.
- [9] L. A. Geddes, "Chronaxie," *Austral. Phys. Eng. Sci. Med. Biol.*, vol. 22, pp. 13–17, 1999.
- [10] —, "Evolution of circuit models for the electrode-electrolyte interface," *Ann. Biomed. Eng.*, vol. 25, pp. 1–14, 1997.
- [11] A. V. Hill, "Excitation and accommodation in nerve," *Proc. Roy. Soc.*, vol. B119, pp. 305–354, 1935.
- [12] A. L. Hodgkin, "The subthreshold potentials in a crustacean nerve fiber," *J. Physiol.*, vol. 126, pp. 87–121, 1939.
- [13] W. Irnich, "The chronaxie time and its practical importance," *PACE*, vol. 3, pp. 292–301, 1980.
- [14] H. H. Jasper, R. H. Johnson, and L. A. Geddes, "The RCAMC electromyograph," Research Council of Canada, Canadian Army Medical Rep. C6174, Apr. 1945.
- [15] L. Lapique, "Influence d'une variation locale de température sur l'excitabilité du nerf moteur," *Comptes Rendus Soc. de Biol.*, vol. 62, pp. 35–37, 1907.
- [16] —, "Definition expérimentale de l'excitation," *Comptes Rendus Acad. Sci. (Paris)*, vol. 67, no. 2, pp. 280–283, 1909.
- [17] —, *L'Excitabilité en Fonction du Temps*. Paris: Presses Universitaires de France, 1926.
- [18] —, "Consideration préalables sur la nature du phénomène par lequel l'électricité excite les nerfs," *J. Physiol. Pathol. Génér.*, vol. 9, pp. 565–578, 1907.
- [19] K. Lucas, "The analysis of complex excitable tissues by their response to electric currents of short duration," *J. Physiol.*, vol. 35, pp. 310–331, 1906–7.
- [20] K. M. Overmeyer, J. A. Pearce, and D. P. DeWitt, "Measurement of temperature distribution at electrosurgical dispersive electrode sites," *Trans. ASME, J. Biomech. Eng.*, vol. 10, pp. 66–72, 1979.
- [21] A. Ritchie and C. R. Smith, "The electrical diagnosis of peripheral nerve injury," *Brain*, vol. 67, pp. 314–330, 1944.
- [22] C. R. Smith, L. A. Geddes, and J. D. Bourland *et al.*, "The chronaxie and propagation velocity of canine cervical vagus nerve fibers in vivo," *J. Cardiovasc. Eng.*, vol. 1, no. 2, pp. 77–84, 2001.
- [23] G. Weiss, "Sur la possibilité de rendre comparable entre eux les appareils servant à l'excitation électrique," *Arch. Ital. Biol.*, vol. 35, pp. 413–446, 1901.
- [24] J. L. Wessale, L. A. Geddes, G. M. Ayers, and K. S. Foster, "Comparison of rectangular and exponential pulses for evoking sensation," *Ann. Biomed. Eng.*, vol. 20, pp. 237–244, 1992.



**Leslie A. Geddes** (A'47-M'55-SM'58-F'77-LF'87) was born in Scotland and educated in Canada. He received the B.Sc. and M.Sc. degrees in electrical engineering from McGill University, Montreal, QC, Canada, and the Ph.D. degree in physiology from Baylor University College of Medicine, Houston, TX. He was awarded the D.Sc. Honoris Causa by McGill University in 1971.

He is the Showalter Distinguished Professor Emeritus of Bioengineering and founding Director of the Hillenbrand Biomedical Engineering Center at Purdue University (1974–1991), West Lafayette, IN. While at McGill, he was an Instructor in electrical engineering and in neurophysiology. While at Baylor, he was Assistant, Associate, and Full Professor of Physiology and Director of the Division of Biomedical Engineering. Also, while in Houston, he was Adjunct Professor of Physiology at the University of Texas Dental Branch and Adjunct Professor of Physiology at Texas A&M Veterinary College. He has conducted research in electromyography, cardiac output, cardiac pacing, ventricular defibrillation, and blood pressure. His physiological studies have been carried out on exotic animals. The properties of stimulating and recording electrodes have been a continuing interest since graduation from McGill. Several new techniques for teaching in medical education were developed by Dr. Geddes and his colleagues. He has written 20 books, holds 22 patents, and has published more than 700 scientific papers, receiving the Nightingale Prize for one of the papers and the Texas Medical Association award for a videotape on acute myocardial infarction.

Dr. Geddes was elected to Phi Epsilon Alpha, honors engineering society while at McGill University. At Purdue, he was elected to the Phi Zeta honors Veterinary Medicine society. He is a fellow of numerous scientific societies, including the AAAS, the American College of Cardiology, the Australian College of Physical Scientists in Medicine, the American Physiological Society, the Radio Club of America, the Royal Society of Medicine, and the International Academy of Medical and Biological Engineering. He is a registered professional engineer in Texas and a member of the National Society of Professional Engineers. He is board-certified diplomate in forensic engineering by the National Academy of Forensic Engineers (Fellow-494F) and is a certified clinical Engineer. He received the AEMB award for leadership in biomedical engineering in 1985 and was elected to the National Academy of Engineering in the same year. In 1985, he was the Rosenstadt Professor of Health Sciences at the University of Toronto. In 1986, he received the IEEE/EMBS Career Achievement Award and the AAMI Laufman-Greatbatch award in 1987. In 1989, he received the Outstanding Educator award from the American Society for Engineering Education. In 1994, he received the Edison Gold Medal from the IEEE. In 1997, he received the World of Difference award from the Indiana Health Industry Forum for contributions to the health industry. Also in 1997, he received the Henri Busignies award from the Radio Club of America for his major contributions to medical instrumentation for the benefit of mankind. In 2001, he received the Third Millennium Medal from the IEEE. In 2001, he received the Lee Deforest award for significant contributions to the advancement of radio communications. In 2002, he received the Devteq Patient Safety Award from the American Board of Clinical Engineers. He serves as a consulting editor to numerous scientific journals and is a consultant to NIH, FDA, and NSF. He is listed in *Who's Who, Leaders in the Southwest, American Men of Science* and the *Royal Blue Book*.