AFFIDAVIT OF JAMES J. RAMSEY

County of Davidson)
)
State of Tennessee)

I. My name is James J. Ramsey. I presently reside at 466 Rodney Street, Gallatin, Tennessee 37066.

II. I am a Licensed Clinical Perfusionist (LCP). Perfusionists perform and monitor the initiation, maintenance, and discontinuation of cardiopulmonary bypass (CPB) and other circulatory support technologies, commonly referred to as 'heart-lung' bypass procedures. I have been so certified (now licensed in the State of Tennessee pursuant to T.C.A. Section 63-28-101 et seq.) and practicing Perfusion Care since my graduation from Vanderbilt's Program in 1984, and to date have participated in approximately five thousand (5000) clinical applications of heart-lung machine and other circulatory support technologies.

III. I am currently employed by the Department of Cardiac and Thoracic Surgery, School of Medicine, Vanderbilt University Medical Center, Nashville, Tennessee. My position is that of Program Director for the post-graduate Allied Health Program in Cardiovascular Perfusion Technology. I have held that position for the past nine (9) years, and previously held that position from 1986 - 1989.

IV. I am an attorney, licensed to practice and currently practicing in the State of Tennessee. My BPR # is 16263.

V. Regarding my clinical practice during CPB, one of the clinical functions (and statutory duties) of the LCP is that of '....myocardial and (other) organ preservation', which in simple terms as related to CPB involves the application of appliance technologies

directed at the arrest of (stopping the beating of) the human and animal heart, preserving its function during the arrest period so as to enable it to function adequately following the arrest interval, modifying the re-perfusion of the arrested heart, and re-establishment of the function of the heart to a beating, working state. As a licensed perfusionist, I am also certified to undertake physiologic monitoring and analysis, as well as blood gas and chemistry monitoring and analysis. For nearly all of the clinical cases I have performed, it has been my duty to arrest the electromechanical function of the heart through a variety of means. For purposes of this affidavit, I will address only the actual electrical arrest of the beating human or animal heart.

VI. Attorneys for Philip Workman have asked me to review the New Lethal Injection Protocol adopted by the State of Tennessee on April 30, 2007. I have previously reviewed the older versions of the Tennessee Lethal Injection Protocols as well as documents which the State of Tennessee has provided in discovery. I have reviewed the new April 30, 2007 protocol and reach the following conclusions.

VII. As a preface, it is my professional opinion, based upon my knowledge, training, and experience in the field of Perfusion Care and especially in the area of myocardial arrest, and my review of the Tennessee execution protocol, that the potassium component of the lethal injection protocols currently used by the State of Tennessee (100 mg/mL of a 2mEq/ml concentrate) (sic)[1] is wholly ineffective in causing electrical arrest of the human heart. Furthermore it is a pathophysiological impossibility, based upon well-established and accepted mathematical equations, for the heart to succumb to electrical

[1]Potassium concentrations are scientifically measured in units of mEq/L.

arrest due to the potassium component of the lethal injection protocol. The loss of function of the heart, if and when it does arrest during lethal injection, is entirely due to suffocation and lack of oxygen delivery, and not electrical arrest due to potassium injection. I hold this opinion within a reasonable degree of scientific certainty.

VIII. The chemical compositions of extracellular and intracellular fluids are described in Exhibit #1. Based upon the relative concentrations of especially sodium (Na+), calcium (Ca++), and potassium (K+) ions inside and outside the cell, the 'resting membrane potential' of any living cell (and especially cells of 'excitable' tissue - nerves and muscles) is described by the Nernst Equation (see Exhibit #2).

IX. This electrical voltage of an excitable cell is commonly referred to as the 'action potential' for excitable cells, and is based upon the fact that at some point (generally due to some kind of electrical or other stimulus) the action potential of excitable tissues and cells can change, resulting in an alteration of the electrical potential. This alteration causes an action (in nerves, conduction of an electrical impulse, or in muscles, a contraction of the muscle fibers).

X. Application of the Nernst Equation may result in calculations for the various action potentials attributable to sodium, potassium, and calcium (see Exhibit #3 and #4). However, the Nernst Equation does not take into account the fact that the cell membranes of excitable tissue exhibit properties of varying states of permeability, relative to their structure and function. Permeability to ions results in a dynamic component regarding the action potential and its calculation.

XI. Further inquiry must include calculations that take into account the resting ionic

permeability for sodium, potassium, and calcium regarding excitable cells, in order to accurately calculate the action potential of excitable cells. The Goldman-Hodgkin-Katz Equation describes the calculation of action potential for excitable cells, inclusive of the factor referred to above as resting ionic permeability (see Exhibit #5).

XII. A calculated action potential of -86 mV is derived from that formula (see Exhibit #6 and #7).

XIII. However, the inquiry is not yet complete, because there is a phenomenon referred to as the 'sodium-potassium pump' that is present in the cell membranes of excitable cells (see Exhibit #8). The sodium-potassium pump is an active-transport mechanism (not a passive mechanism) that causes the continuous pumping of sodium and potassium in and out of the cell in order to establish and maintain the action potential. The net effect of the sodium-potassium pump, from an electrical perspective, results in a loss of positive charges from inside the cell, creating an additional negativity (-4 mV) across the cell membrane.

XIV. By adding the -4mV attributable to the sodium-potassium pump to the -86 mV action potential as calculated by the Goldman-Hodgkin-Katz Equation, a net action potential of -90mV is determined. This is in fact the physiological action potential for excitable cells (see Exhibits #9 and #10).

XV. Excitable cells in that resting state of -90 mV action potential are said to be 'polarized', that is, they are not in an electrically neutral state. In fact, the reason for the polarized condition is as previously stated: the cells being excitable, they are subject to and capable of causing an action of some kind (nerve impulse or muscle contraction).

XVI. Polarized cells are said to be in a resting state; that is, there is no action occurring as long as the action potential remains at -90 mV.

XVII. Excitable cells that undergo a change in action potential may become depolarized; that is, the action (resting) potential of the cell changes due to a stimulus, with the net electrical effect being that the -90mV potential is increased (becomes less negative). At the point that that the potential reaches the level of -65mV, the cell becomes depolarized. At this point, the characteristics of the cell membrane undergo radical changes of permeability, with sodium rushing into the cell through what are called sodium channels, and later potassium leaving the cell through a similar mechanism.

XVIII. The important point here is that these changes result in dramatic changes in electrical status, and as demonstrated in Exhibit #11 and #12, an electrical action occurs (change in cell polarization from negative to positive). Soon afterwards (milliseconds in time) the cell membrane potential is caused to become negative again, based upon among other things the active transport mechanisms associated with the cell membrane, and following the 'action' of the cell, it again reaches a resting state, with its resting potential now found to be -90mV (just as before its action or stimulus).

XIX. The same mechanism occurs in myocardial cells, as described in Exhibit #12, with several important differences. In the case of myocardial cells ('Cardiac Muscle'), the depolarization of the cells results in a muscle contraction rather than an electrical impulse (nerves). Secondly, calcium ions play a major role in the process, as calcium is an important contributor to the actual 'shortening' of the muscle fibers, characteristic of muscle contraction. Following the depolarization of myocardial cells, a re-polarization

occurs, with the cell again returning to its resting state.

XX. The question becomes -if we are to stop this process from occurring (arresting the heart so that the cells do not depolarize and therefore the myocardial fibers do not contract), how are we to accomplish this?

XXI. Based upon the science as previously described, there are two means of accomplishing this: (1) remove sodium from the extracellular space so that it cannot rush into the cell and depolarize it; or (2) alter the membrane potential to prevent the opening of the sodium channels, thereby preventing sodium from rushing into the cell causing depolarization.

XXII. Based upon the very high extracellular concentration of sodium (see Exhibit #1), it would be impractical to remove enough sodium so as to prevent depolarization as described in method #1 above.

XXIII. It is possible, however, to alter the membrane potential of excitable (myocardial)cells so as to prevent the opening of the sodium channels, thereby preventing sodium from rushing into the cells causing depolarization (in effect, stopping the beating and squeezing of the heart).

XXIV. In order to do that, it is possible to add enough potassium to the extracellular fluid space so that the action potential of -65mV as previously described is no longer a factor, and that the resting potential of the cell (previously -90mV) is raised (made more positive) such that the sodium channels never open, and a state of depolarization never occurs.

XXV. By applying the Goldman-Hodgkin-Katz Equation and solving for a potassium solution that would raise the resting potential of the myocardial cell from -90mV to -56 mV

(taking into account the target of -60 mV and accounting for the -4mV attributable to the sodium-potassium pump), we find that the minimum effective extracellular potassium concentration necessary to prevent the opening of sodium channels in the cell membrane as previously described is 16.4 mEq/L. (See Exhibit #13).

XXVI. It should be noted, per Exhibit #1, that the normal extracellular potassium concentration is in the range of 4 mEq/L.

XXVII. When the extracellular potassium concentration is 16.4 mEq/L or greater, the sodium channels will not open, there will be no net effective change in membrane potential, and no action potential or change of electrical status as previously described will occur. The net result: the myocardial muscle fibers will not shorten, and the heart will not beat.

XXVIII. In the operating room, the common method for infusing potassium-rich solutions into the heart so as to cause the heart to cease function is as follows:

a. High potassium solutions (20 mEq/L) are administered directly into the coronary arteries, both in a forward direction (antegrade) and in a backward direction (retrograde). This methodology insures that all segments of myocardial tissue (regardless of native supply being disrupted due to atherosclerotic disease) are exposed to the high potassium solutions.

b. High potassium solutions are delivered in both directions as well because often times patients have insufficiency of the aortic valve, which results in the inability of the antegrade delivery method to adequately perfuse myocardial tissues through the coronaries (the flow that would otherwise go through the coronaries is redirected into the left ventricle in that case).

c. High potassium solution is delivered in a profoundly hypothermic state; that is, at approximately 5-7 degrees Celsius (or nearly freezing, as opposed to normal body temperature of 37 degrees Celsius). The net effect of hypothermic delivery is an enhanced state of cellular arrest (see the temperature component as stated in the previous formulae).

d. High potassium solution is delivered at precise pressures, as measured both at the site of delivery, and at the appliance delivering the device ('back-pressure'), to insure adequate and effective delivery, and in the case of the operating room, adequate electrical arrest of the heart in order to preserve its function.

XXVIX. In spite of these extensive and precise efforts, it is in my experience still difficult to achieve adequate arrest of the heart for surgical purposes in some cases.

XXX. The lethal injection process as described in the discovery documents reviewed does not describe a precise or extensive effort to deliver the potassium solution to the heart - rather, they describe a crude and imprecise method of delivery through IV injection, with the profoundly inaccurate expectation that potassium solution in high concentrations would reach the coronary arteries and effect an arrest. One of the main contributing factors to low potassium concentration solutions reaching the heart would be that, given an intravenous injection, the solution would necessarily have to pass through the lungs (which have the surface area of approximately that of a tennis court), during which potassium concentrations would fall dramatically.

XXXI. Additionally, discovery documents (from State of Tennessee) describe that the amount and concentration of potassium delivered cannot result in the minimum potassium

concentration of 16.4 mEq/L being achieved that is required to arrest the electromechanical function of the heart. The resultant potassium concentrations as described in the Vitreous Electrolyte Panel and Profile Results for inmate Robert Glen Coe, for example, following lethal injection indicate an extracellular potassium concentration of 9 mEq/L, far short of the required minimum concentration of 16.4 mEq/L to cause electromechanical arrest of the heart.

XXXII. It should be noted that given the very high intracellular potassium concentration (per Exhibit #1), and the fact that postmortem cells would, if anything, leak potassium into the extracellular space, the postmortem potassium concentration is in all likelihood higher than it was a the time following injection.

XXXIII. In my professional opinion, and based upon my knowledge, training, and experience in the field of Perfusion Care and arrest of the heart's function, and following review of the disclosures by the State of Tennessee previously described, it is a pathophysiological impossibility, based upon well-established and accepted mathematical equasions, for the heart to succumb to electromechanical arrest due to the potassium component of the lethal injection protocol. The function of the heart, if and when it does arrest during lethal injection, is entirely due to suffocation and lack of oxygen delivery, and not electromechanical arrest due to potassium injection. I hold this opinion within a reasonable degree of scientific certainty.

I affirm or swear under the penalty of perjury that the foregoing is true and correct to the best of my knowledge

James J. Ramsey

Subscribed and sworn before me this 3rd day of May, 2007.

Notary Public, State of Tennessee

My Commission Expires:
9/25/10

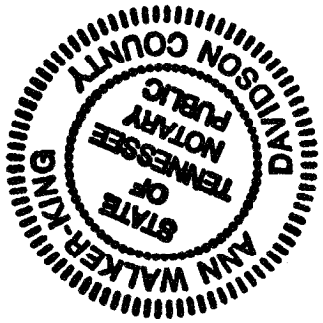


Exhibit 1

Chemical Compositions

	Extracellular*	Intracellular
Na^+	142	14
K^+	4	140
Ca^{++}	2.4	0.0001
Mg^{++}	1.2	58
Cl^-	103	4
HCO_3^-	28	10
Phosphates	4	75
SO_4^{--}	1	2

* All concentrations in mEq/L

Exhibit 2

Nernst Equation

$$E_{ion} = V_{in} - V_{out} = 2.303 \frac{RT}{zF} \log \frac{[C_{out}]}{[C_{in}]}$$

where

T = absolute temperature in °K = °C + 273.16,

R = ideal gas constant = 8.31451 J/(mol - °K),

F = Faraday's number = 96485.3 Coulombs/mol, &

z = valence of the ion.

Exhibit 3

Nernst Equation

At 37 °C & for an univalent ion:

$$E_{ion}\ (mV) = 61.6 \log \frac{[C_{out}]}{[C_{in}]}$$

For K^+:

$$E_{K^+} = 61.6 \log \frac{4}{140} = -95\ mV$$

Exhibit 4

Membrane Potential - Under Construction

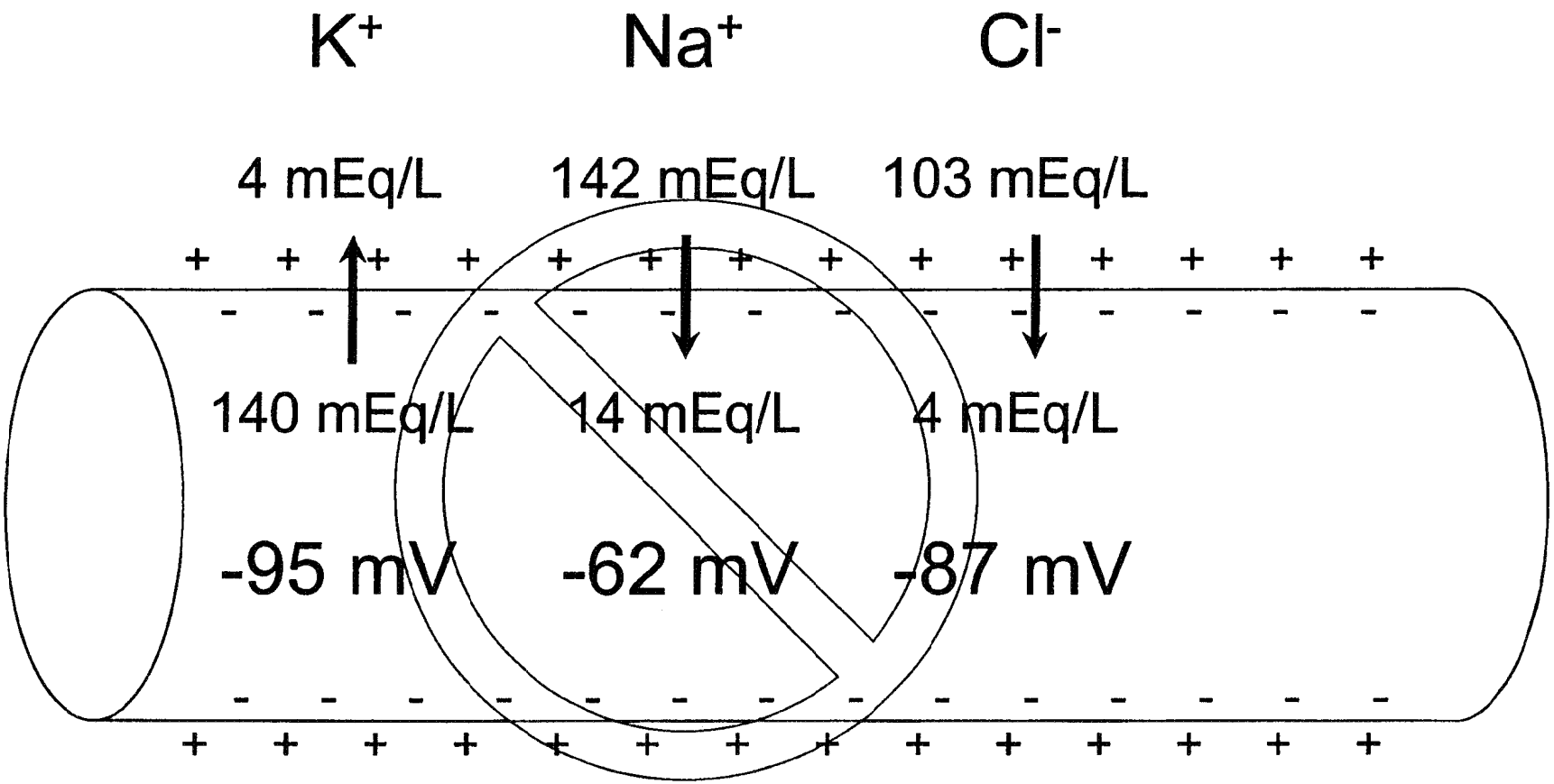


Exhibit 5

Goldman-Hodgkin-Katz Equation

$$E\ (mV) = 2.303\ \frac{RT}{F}\ \log \frac{[C_{K^+o}](P_{K^+}) + [C_{Na^+o}](P_{Na^+}) + [C_{Cl^-i}](P_{Cl^-})}{[C_{K^+i}](P_{K^+}) + [C_{Na^+i}](P_{Na^+}) + [C_{Cl^-o}](P_{Cl^-})}$$

where

T = absolute temperature in °K = °C + 273.16,

R = ideal gas constant = 8.31451 J/(mol - °K),

F = Faraday's number = 96485.3 Coulombs/mol, &

P = ionic permeability.

Exhibit 6

Goldman-Hodgkin-Katz Equation

At 37 °C & with resting ionic permeabilities of 1 for K^+, 0.0115 for Na^+, & 0.1 for Cl^-:

$$E_{REST}\ (mV) = 61.6 \log \frac{[4](1) + [142](0.0115) + [4](0.1)}{[140](1) + [14](0.0115) + [103](0.1}$$

$$= 61.6 \log \frac{6.033}{150.461} = -86\ mV$$

Exhibit 7

Membrane Potential - Construction Proceeding

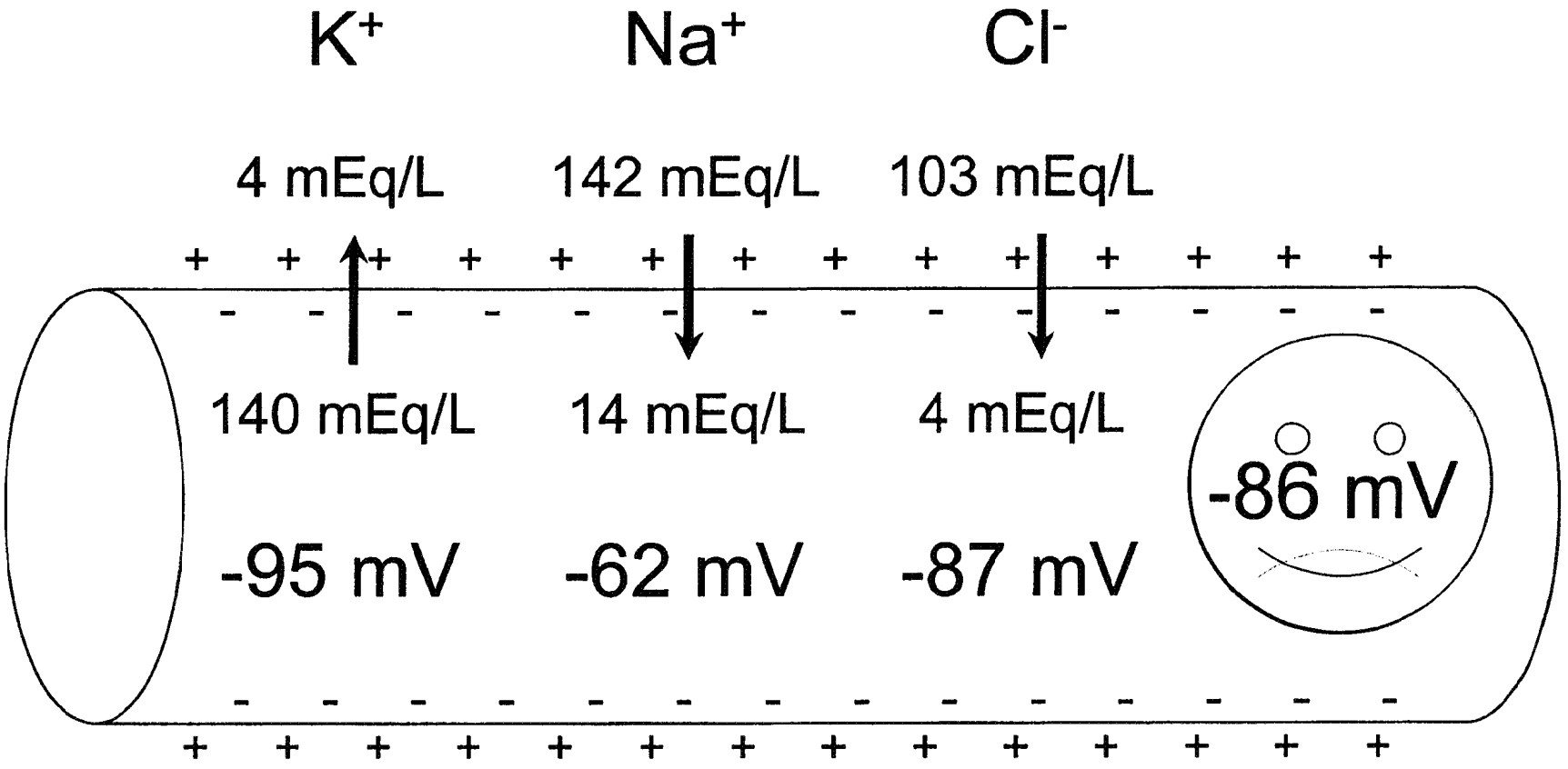


Exhibit 8

Sodium / Potassium Pump

- Cell membrane more permeable to K^+ than Na^+ so K^+ leaks out
- Continuous pumping of 3 Na^+ out of cell for every 2 K^+ into cell
- More positive ions being pumped out than in
- Results in loss of positive charges from inside cell
- Creates an additional negativity (-4 mV) across cell membrane

Exhibit 9

Membrane Potential - Construction Complete

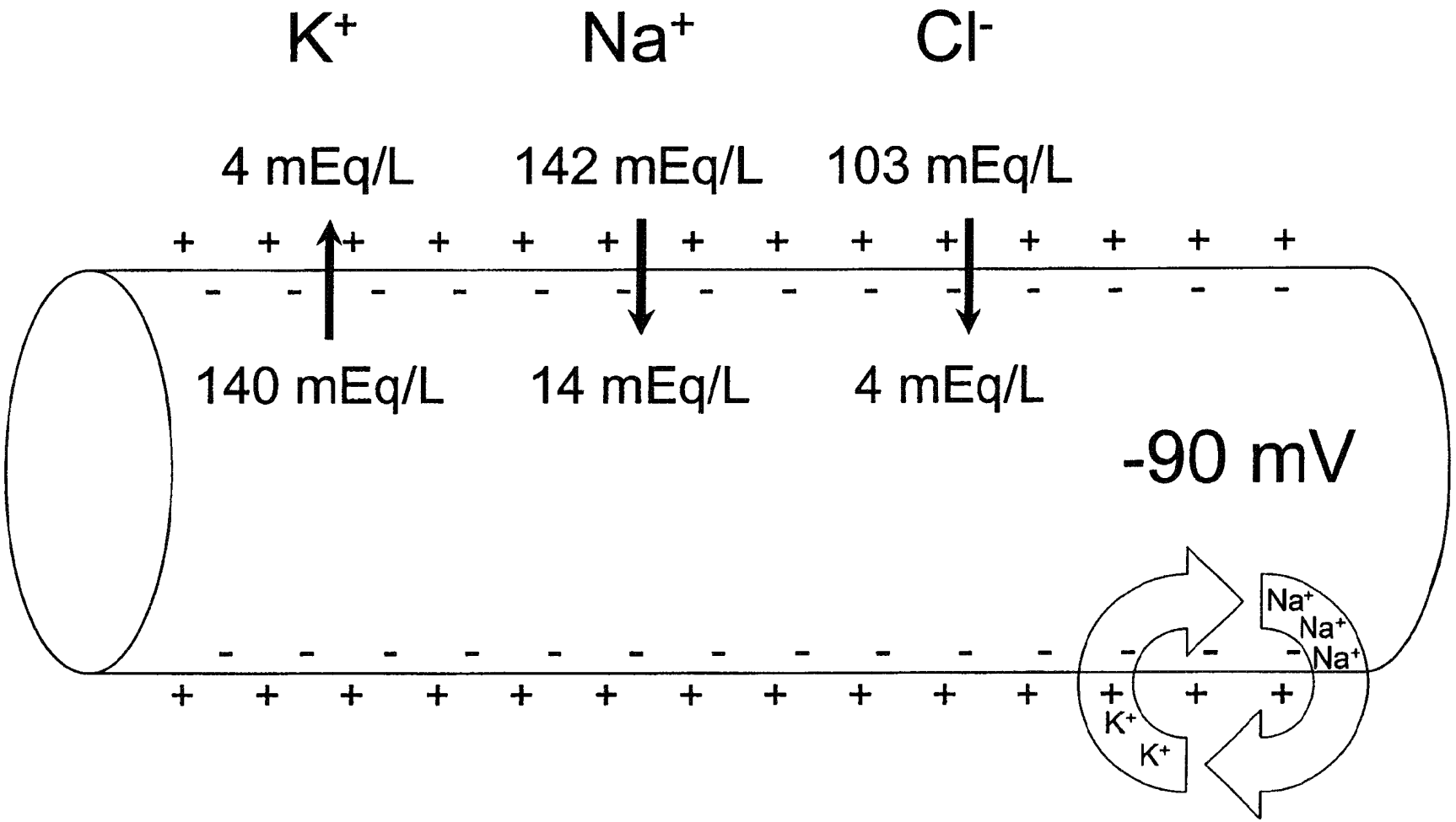


Exhibit 10

Resting Membrane Potential

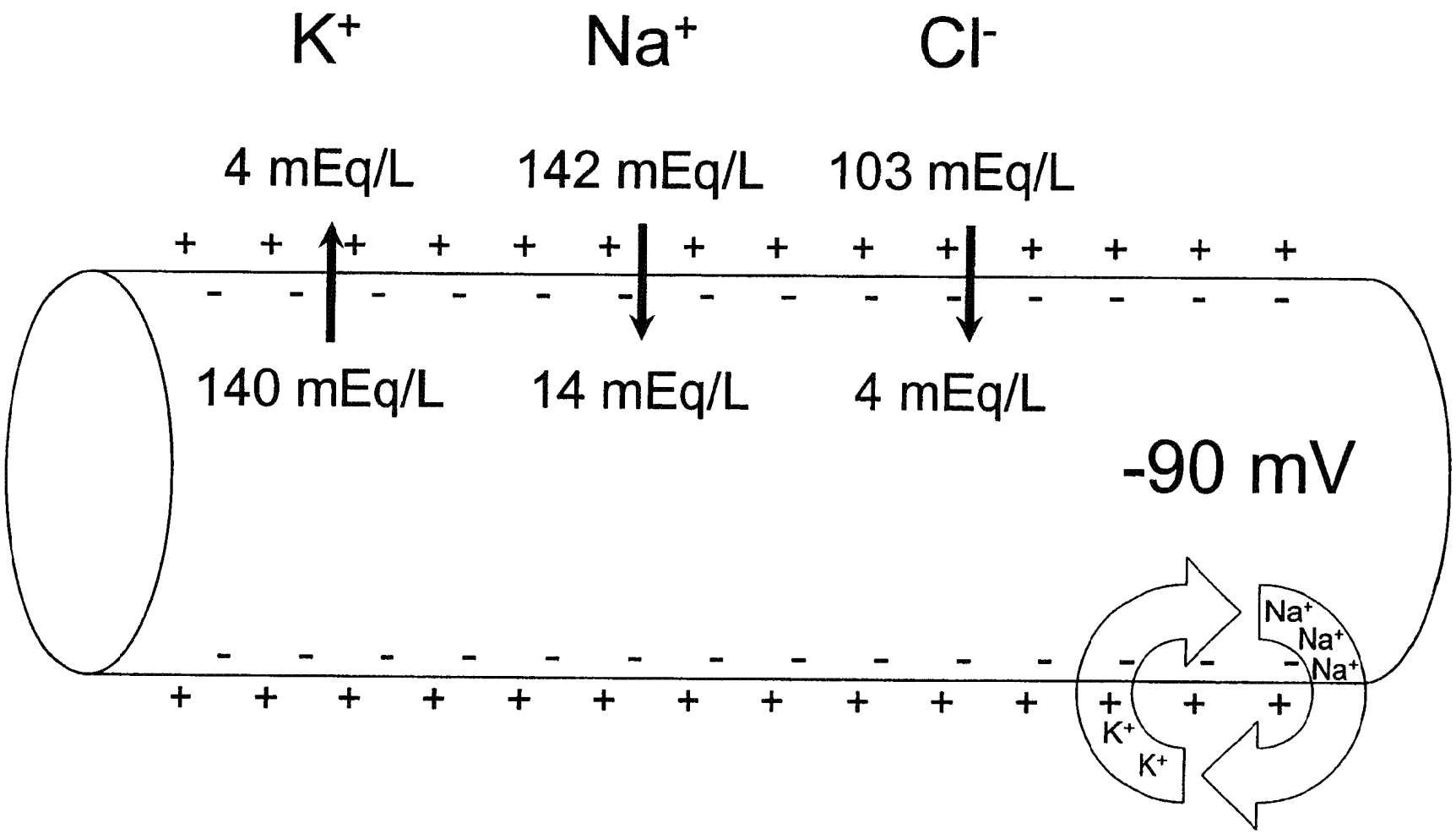
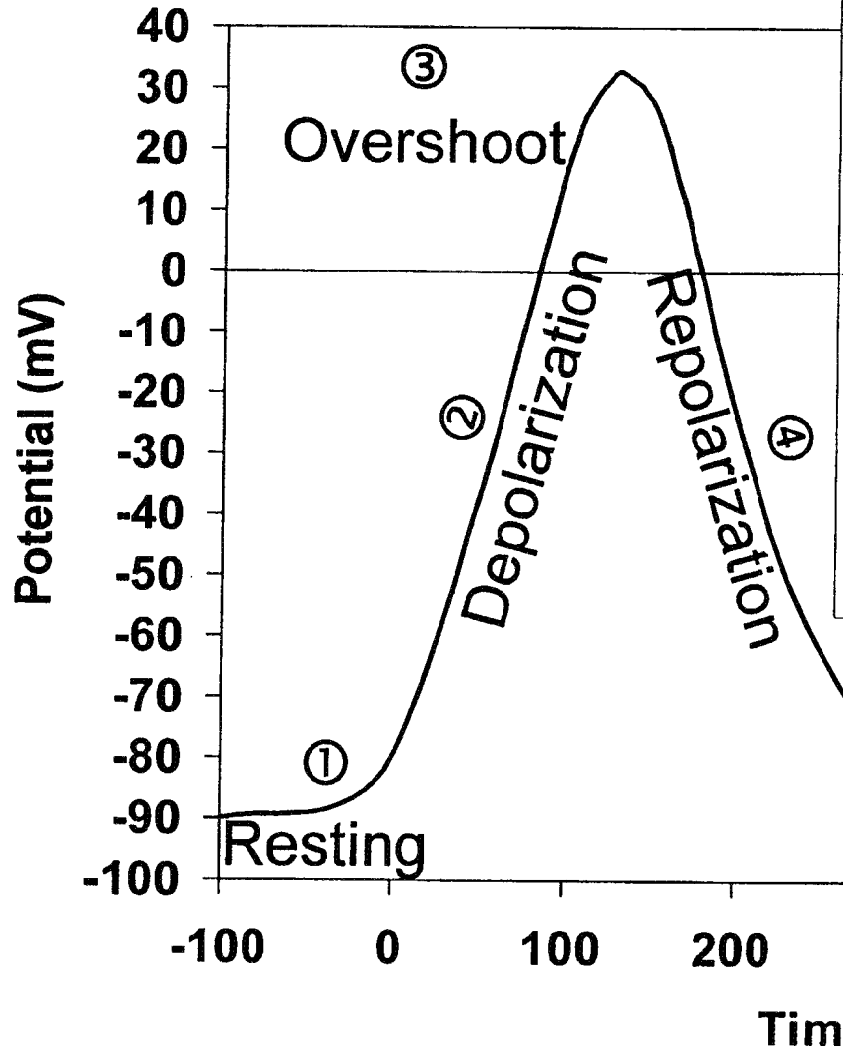


Exhibit 11

Nerve Action Potential



① Membrane polarized @ -90 mV

② Trigger initiated; threshold reached; membrane suddenly permeable to Na^+ by opening Na+ channels; Na^+ rushes into cell

③ Magnitude of positive potential dictated by type & size of nerve cell

④ Na^+ channels close; K^+ channels more permeable; K^+ leaves cell; resting potential of -90 mV returned; Na^+ / K^+ pump returns concentration gradients

Exhibit 12

Cardiac Muscle Action Potential

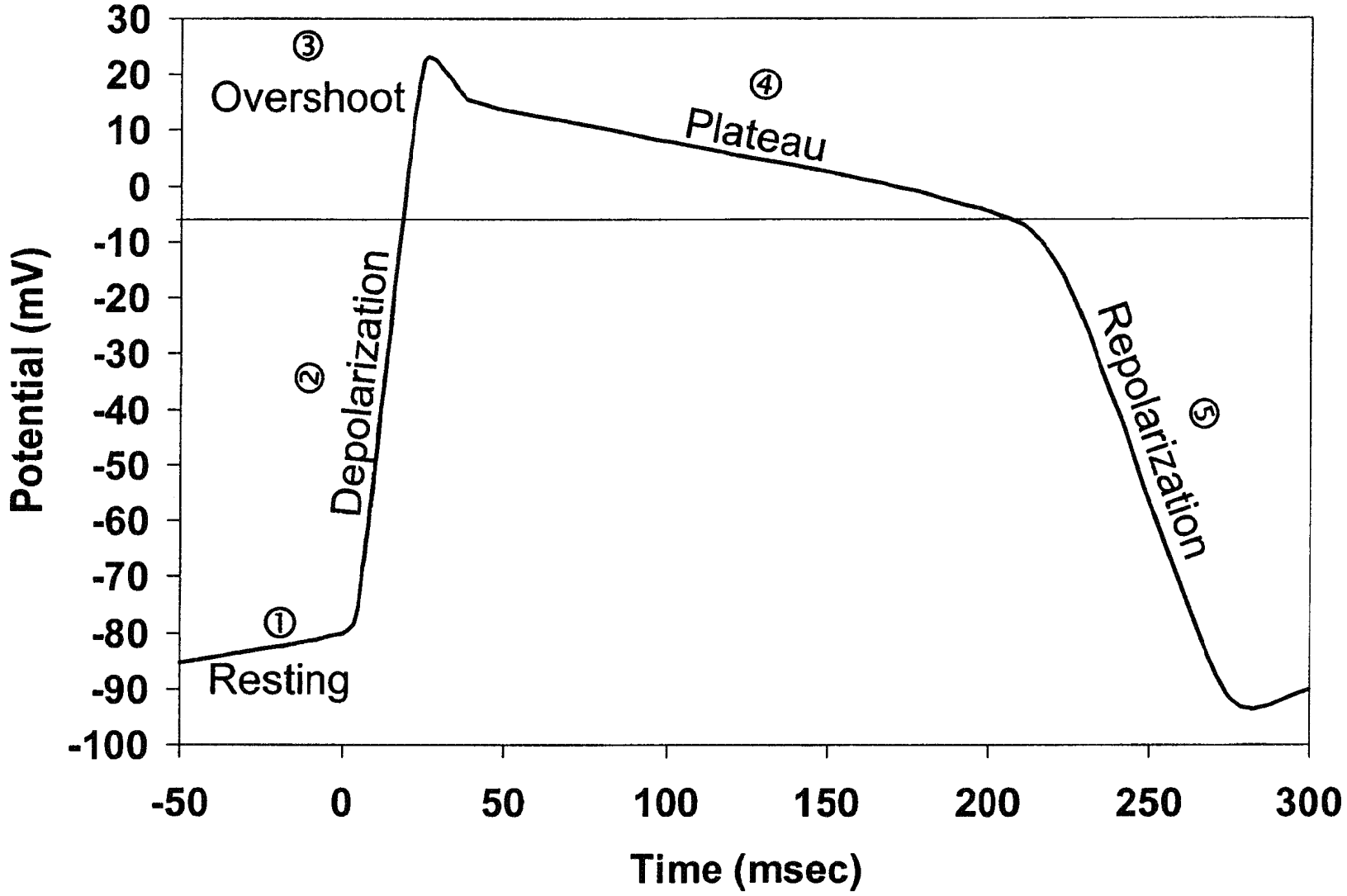


Exhibit 13

Hyperkalemic Cardioplegia
- Minimal Effective Concentration

Alter Resting Membrane Potential of -95 mV to be greater than Threshold Potential of -65 mV

- Set Target of -60 mV
- Account for -4 mV from Na^+ / K^+ Pump
- Now Target of -56 mV
- Use Goldman-Hodgkin-Katz Equation to determine extracellular [K^+

$$E\ (mV) = 2.303 \frac{RT}{F} \log \frac{[C_{K^+o}](P_{K^+}) + [C_{Na^+o}](P_{Na^+}) + [C_{Cl^-i}](P_{Cl^-})}{[C_{K^+i}](P_{K^+}) + [C_{Na^+i}](P_{Na^+}) + [C_{Cl^-o}](P_{Cl^-})}$$

$$-56\ mV = 61.6 \log \frac{[?](1) + [142](0.0115) + [4](0.1)}{[140](1) + [14](0.0115) + [103](0.1)} = 61.6 \log \frac{[?] + 2.033}{150.461}$$

$$[?] = 16.4\ mEq/L$$