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A SIMPLE SIMULATOR OF MEMBRANE POTENTIALS

by C. Leon Harris
Department of Biological Sciences
State University College
Plattsburgh, New York 12901

Among the most difficult topics for biology students is the electrical behavior of excitable membranes. Few biology students are prepared to understand concepts such as equilibrium potential, conductance and membrane capacitance. Consequently a large proportion of students fail to appreciate the elegance of mechanisms for the production of resting, action, and postsynaptic potentials. One approach to this difficulty is the use of simulation whereby the student can recreate these electrical events with an artificial device. A number of electronic simulators have been described (1, 3, 4) but these are generally complex and not intended for direct manipulation by students. More useful would be a simple device which allows the student to see the consequences of changing equilibrium potentials and conductances.

Hodgkin-Huxley Model of Membrane

The simulator described here is based on the Hodgkin-Huxley electrical model of the membrane of the squid giant axon (2) (Fig. 1). This model is frequently described in physiology texts and is generally applicable to the membranes of other excitable tissues. The two horizontal lines represent the inner (cytoplasmic) and outer (ECF) conductive fluids adjacent to a patch of membrane. These conductors are joined by the capacitor C_M and three other branches. (The capacitance results from the separation of the two conductors by the insulating lipid bilayer.) The other three branches represent channels for the movement of Na^+, K^+, and Cl^-. (Other highly permeable ions are usually lumped with Cl^- and termed leakage.) It is reasonable to represent ion movements by electrical currents since current is, by definition, moving charge. The permeability of the membrane for an ion can be represented by conductance, which is the reciprocal of electrical resistance. Thus the symbol for an electrical resistor in the three branches represents the conductance g for a particular ion. By Ohm's Law the current through the conductance (representing the rate of movement of an ion through its channels) equals Eg, where E is the voltage across the conductance g. In membranes E is the driving force tending to push ions through channels and is proportional to the disparity between the equilibrium potential (determined by the Nernst equation) for the ion and the actual membrane potential. If the equilibrium potential for a particular ion equals the membrane potential there is no net movement of that ion through its channels. (This occurs when the ratio of concentrations of the ion on each side of the membrane is such that the membrane potential is just balanced by the diffusional potential of the ions.) The greater the difference between the actual membrane potential and the equilibrium potential for an ion the greater will be the effective force pushing ions through the channels. If we let the membrane potential be E_M and the equilibrium potential for Na^+ be E_{Na}, we see that the current due to Na^+, I_{Na}, equals g_{Na} $(E_M - E_{Na})$. Potassium and chloride currents are similarly derived. In the electrical model

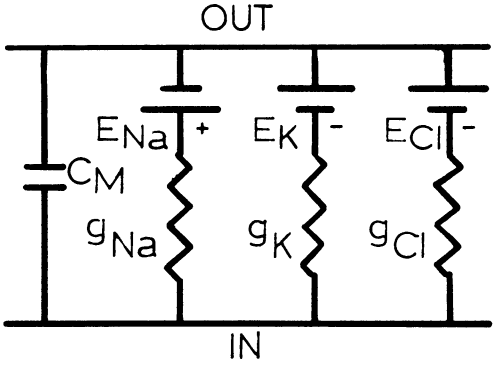

Fig. 1. The electrical model of squid giant-axon membrane. C_M is membrane capacitance; E_{Na}, E_K and E_{Cl} are equilibrium potentials; g_{Na}, g_K and g_{Cl} are conductances. Adapted from Hodgkin and Huxley, 1952.

this situation is simulated by placing each conductance in series with a battery which represents the equilibrium potential for the appropriate ion. In most cells a relatively large concentration of intracellular proteins attracts K^+ into the cell and repels $C1^-$ out of the cell. This creates ratios of ion

concentrations which make E_K and E_{Cl} both negative inside, in accordance with the Nernst equation. Although sodium pumps contribute little to membrane potentials and are omitted from the model, they do create a high extracellular Na^+ concentration which makes E_{Na} positive inside.

It can be shown by simple electronic theory that this model will produce a membrane potential which is the weighted average of the equilibrium potentials. That is,

$$E_M = \frac{g_{Na}E_{Na} + g_K E_K + g_{Cl}E_{Cl}}{g_{Na} + g_K + g_{Cl}}$$

(This equation is not consistent with the Goldman constant field equation, which is based on a different model of membrane.) It follows, therefore, that increasing g_{Na} will bring E_M closer to E_{Na}, and increasing the other conductances will likewise make the membrane potential change in the direction of the corresponding equilibrium potential. Changes in E_M will not be instantaneous, however. The capacitance C_M represents a large reservoir of charge which must be redistributed whenever E_M changes. This redistribution takes place exponentially with a time constant of $C_M/(g_{Na} + g_K + g_{Cl})$. Normally this time constant equals approximately 1 msec, but changes in E_M can be made faster if the time constant is reduced by increasing any of the conductances.

TABLE I. *Electrical properties of the squid giant axon.*

Resting potential	-65 mV
E_{Na}	+55 mV
E_K	-75 mV
E_{Cl}	-67 mV
Magnitude of action pot	90 mV
g_{Na} (resting)	0.0033 mmhos/cm^2
g_K (resting)	0.24 mmhos/cm^2
g_{Cl}	0.30 mmhos/cm^2
C_M	1.1 μF/cm^2
Time constant (resting)	0.7 msec

Compiled from Hodgkin & Huxley (1952) and Wilson (1974, Tables 7-3 and 7-5).

Normal resting values of the squid giant axon are presented in Table 1. Since g_{Na} is so small E_M is determined primarily by E_K and E_{Cl} at rest. Hodgkin and Huxley found that the action potential results from changes in g_{Na} and g_K, which in turn result from changes in membrane potential (Fig. 2). When the membrane is depolarized there is a rapid increase in g_{Na} (sodium activation) and a slower increase in g_K (potassium activation). Beyond a certain level of depolarization, called threshold, increasing g_{Na} results in more depolarization, which increases g_{Na} further, and so on. (This is an example of positive feedback.) The result is a rapid change in E_M in the direction of E_{Na}, which is the rising phase of the action potential. This depolarization turns itself off, however, by increasing g_K and by the process of sodium inactivation, which reduces g_{Na}. The falling phase of the action potential is due primarily to the return of g_{Na} to a low value. Potassium activation speeds up the recovery from the action potential by decreasing the time constant, and it also causes the positive hyperpolarizing afterpotential. Hodgkin and Huxley succeeded in determining the voltage- and time-dependence of g_{Na} and g_K and in fitting these into an equation which describes the action potential of the squid giant axon. With some modification the Hodgkin-Huxley equation and the electrical model on which it is based can also describe action potentials in other nerve and muscle membranes. Moreover, the electrical model can be used to explain depolarizing potentials in receptors and synapses in terms of increasing g_{Na}, and hyperpolarizing potentials as increasing g_K and/or g_{Cl}.

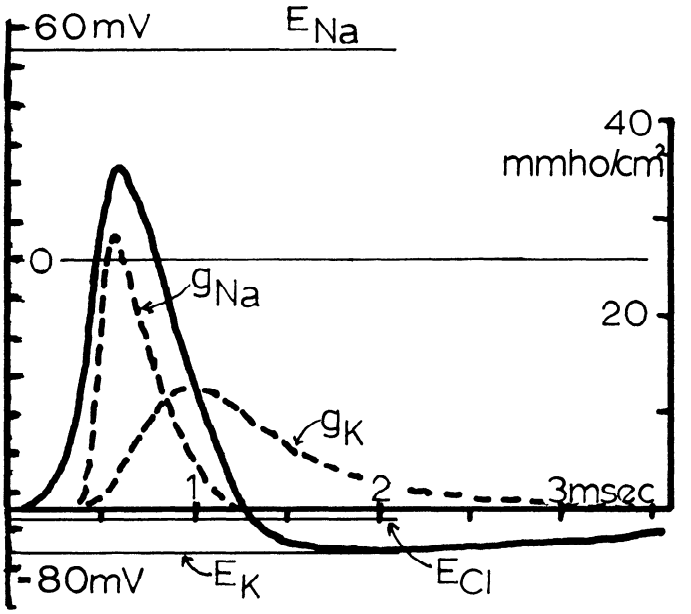

Fig. 2. The action potential of a squid giant axon. Conductance changes are superimposed, and equilibrium potentials are indicated. Adapted from Hodgkin & Huxley, 1952.

The Voltage Clamp

The voltage clamp is an indispensable tool for the study of ionic currents in membrane. It is a device which automatically maintains the voltage across a membrane at a constant, preset level. It is easier to study conductance changes with the voltage clamp because they will be directly proportional to the membrane currents (by Ohm's Law), and they will not be influenced by a changing membrane potential. Figure 3 shows the currents measured across the squid giant axon membrane clamped above the threshold for an action potential. In the normal axon there is an initial transient inward current which is due to sodium. This is followed by an outward potassium current which is maintained as long as the membrane is clamped. Thus there is an initial influx of Na^+ due to sodium activation, then this is terminated by sodium inactivation, and finally there is the efflux due to potassium activation. These sodium and potassium currents can be separated by the drugs tetrodotoxin (TTX) and tetraethylammonium (TEA), which block sodium and potassium activation, respectively. Electrically the voltage clamp amounts to a constant voltage imposed

across the membrane. This can be added to the electrical model by connecting a battery across the inside and outside conductors.

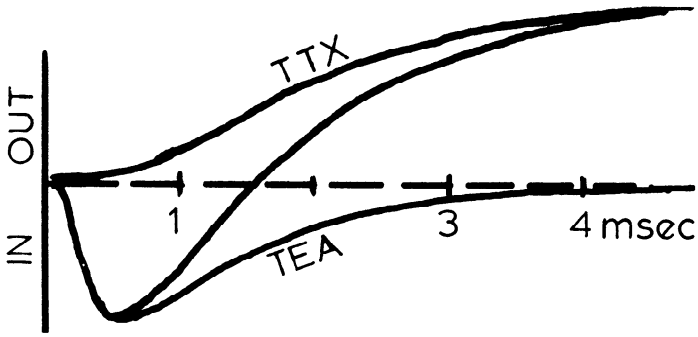

Fig. 3. Current through a giant axon voltage clamped above threshold for an action potential. The effects of tetrodotoxin (TTX) and tetraethylammonium (TEA) are indicated.

The Simulator

If the electrical model of membrane is valid it should be possible to produce an artificial analog. The simulator described here is an attempt to do so. Instead of using resistors and voltages equal to those found in a membrane it is more practical to use components with values merely proportional to the actual values. It is also convenient to select a capacitor with a large value so that the time scale can be expanded for easier control and recording on a polygraph. The time constant chosen is 7 sec, compared to the actual value of approximately 0.7 msec, so that 1 sec of real time simulates 0.1 msec. Figure 4 shows the circuit diagram of the simulator. The values and purposes of each component are explained in the legend. The circuit essentially duplicates Figure 1 except that potentiometers are added to allow control of conductances and equilibrium potentials. Also voltage clamping is simulated by connecting E'_M across the circuit through R11 when the appropriate switch is closed. In the voltage clamp mode the output represents current through the membrane rather than voltage across the membrane.

The appearance of the simulator is shown in Figure 5. All components are widely available except for the slide potentiometers. The total cost of parts is approximately $20. A diagram of the electrical model can be placed on the simulator to remind students of the function of each control. Complete construction details are available from the author.

Prior to use, the simulator must be calibrated with the polygraph to be used. The pen of the polygraph is first positioned on the zerovolt baseline with the simulator turned off. Then the simulator is turned on (voltage clamp off), and $|E_K|$, $|E_{Cl}|$, g_K, and g_{Cl} are set at maximum while E_{Na} and

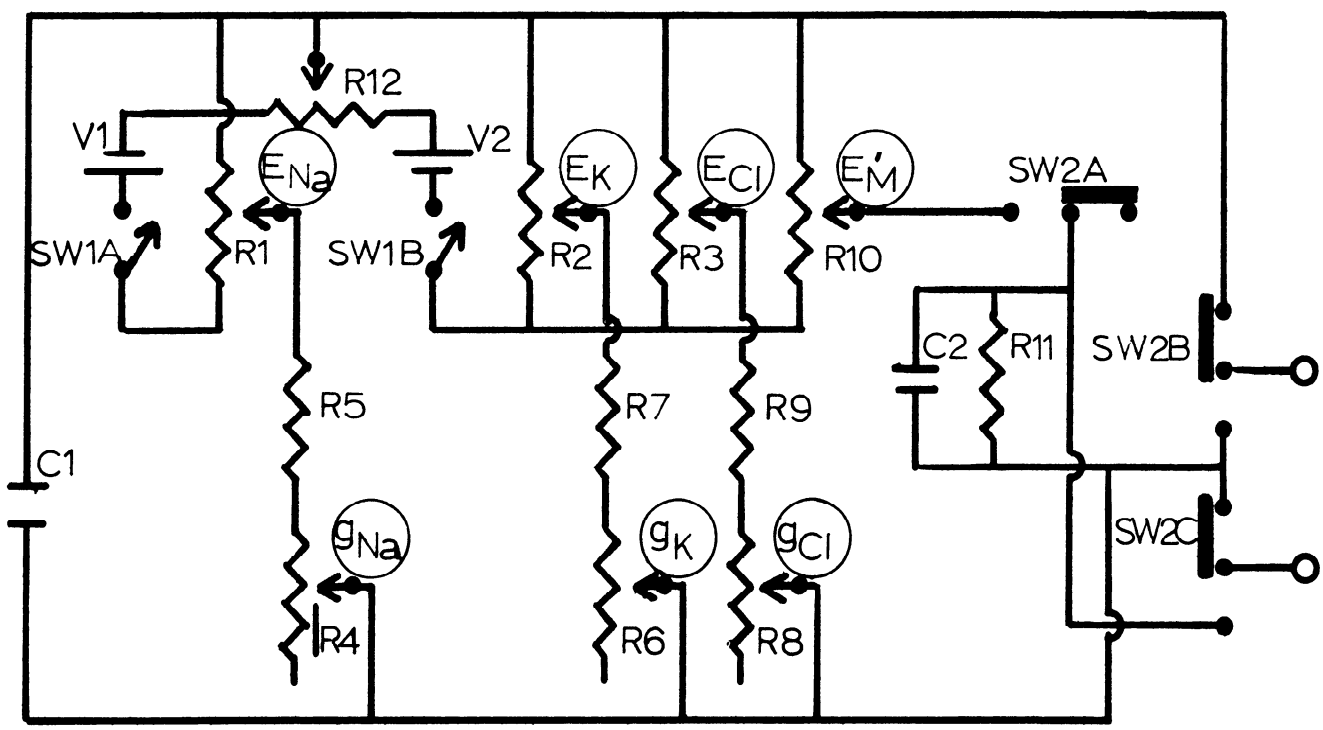

Fig. 4. Schematic diagram of the simulator circuit.

V1, V2 . . . . 1.5 volt battery. Voltage source for equilibrium and clamping potentials.

C1 . . . . 20 μF electrolytic capacitor. Simulates C_M, giving an actual time constant of 7 sec at rest.

C2 . . . . 200 μF electrolytic capacitor. Maintains the same time constant with voltage clamp on.

R1, R2, R3, R10 . . . . 5 K rotary potentiometers. Form dividers across V1 and V2 for the selection of equilibrium and clamping potentials.

R4, R6, R8 . . . . 1 Meg ohm slide potentiometers. R4 is modified by insulating the high resistance end of the slide with tape. Together with R5, R7 and R9 these select the conductances. (Source for these potentiometers: Masline Electronics, In., 511 Clinton Ave. S., Rochester, N. Y. 14620. Order #MSC16L.

R5, R7, R9 . . . . 8.2 K resistors. Determine the maximum conductance values.

R11 . . . . 33 K resistor. Simulated voltage clamp currents form a voltage drop across this resistor which produces the output of the simulator in the voltage clamp mode.

R12 . . . . 1 K trimmer potentiometer. Corrects differences in V1 and V2.

SW1 . . . . DPDT switch. Turns on simulator.

SW2 . . . . 4 PDT switch. Turns on voltage clamp. Three sections of the switch used.

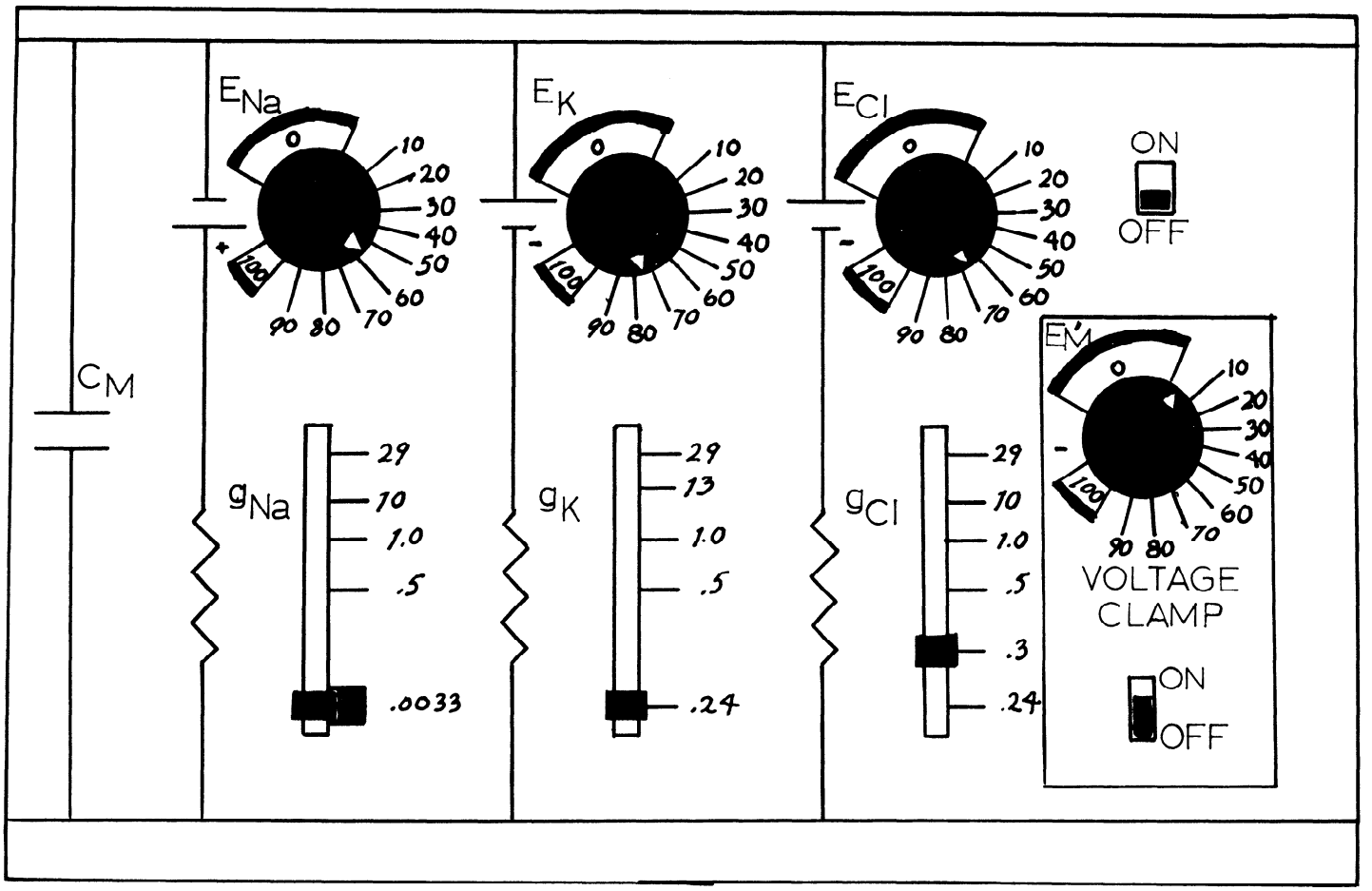

Fig. 5. Top view of the simulator. Overall size 7" x 11" x 2".

g_{Na} are set at minimum. The amplification is increased to give a convenient downward pen deflection. E_{Na} and g_{Na} are then set at their maximum values while the other controls are set at minimum. The trimmer resistor is then adjusted to give an upward deflection equal to the downward deflection previously produced. This must be repeated several times to balance out any differences in the two batteries. Finally the vertical scale is calibrated by setting the K and Cl controls at maximum, the Na controls at minimum, and the amplification of the polygraph for the desired maximum deflection to represent the maximum equilibrium potential (100 mV). The equilibrium potential dials are calibrated by marking the positions which give the correct deflection of the pen. For example, E_{Na} is calibrated by setting g_{Na} at maximum and the other g's at minimum and marking the voltages simulated by various positions of the E_{Na} control. Calibration of conductances is done with an ohmmeter across each slide resistor and fixed series resistor (8.2 K ohm). Since an ohmmeter reading of 8.2 K represents 29 mmhos/cm^2, a potentiometer setting which gives a reading of 23.8 K represents 10 mmhos/cm^2, 238 K represents 1 mmhos/cm^2, and so on.

Use of the Simulator

In my physiology course I have the simulator available for students to use at their convenience. Students are instructed to read relevant chapters in the text and a description similar to this report. They are required to conduct and report on the following experiments and are encouraged to design their own experiments.

1. Simulate the resting potential of the squid giant axon. Use the parameters in Table I (Fig. 6a).
2. Simulate the effect on the resting potential of increasing $|E_K|$ to -93 mV, which could be done by reducing the external K^+ concentration to one-half its normal value.
3. Simulate the effect of doubling the external K^+ concentration. Use the Nernst equation to determine the new value of E_K.
4. Show that changing the Na^+ concentration in a Ringer's solution would have virtually no effect on the resting membrane potential.
5. Show that increasing the conductance for any ion brings E_M closer to the equilibrium value for the ion.
6. Simulate an action potential in a squid giant axon. Begin with the normal resting values and simulate the conductance changes in Figure 2. For 3 sec smoothly increase g_{Na} to 29 mmhos/cm^2, then decrease g_{Na} during the next 7 sec while slowly increasing g_K. g_K should peak at 13 mmhos/cm^2 at the same time g_{Na} returns to 0. For the next 15 sec gradually return g_K to its resting level. Practice this several times. Measure the magnitudes of the action potential, overshoot and positive after-potential and compare them with the values in Figure 2. Note that 1 sec of real time represents 0.1 msec (Fig. 6b).

7. Demonstrate the importance of potassium activation by omitting it from the simulation of an action potential (Fig. 6c).
8. Simulate the effect on action potentials of a reduction in external Na+ concentration to one-half of normal (Fig. 6d).

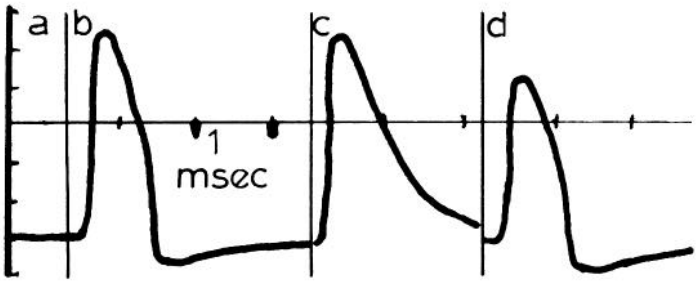

Fig. 6. Simulated resting and action potentials of squid giant axon. a. Resting potential (−60mV). b. Normal action potential. c. Action potential without potassium activation. d. Action potential with external Na+ concentration reduced to one-half of normal. (E_{Na} = 38 mV.)

9. Turn on the voltage clamp and simulate a normal action potential as before with E'_M set at 56 mV above the resting value of E_M. Keep g_K at 13 mmhos/cm^2 as it would be in a normal clamp experiment (Fig. 7a).
10. Repeat with different settings of E_M. How are the sodium and potassium currents affected by increasing $|E_M|$? Why?
11. Simulate the effects of TTX and TEA (Figs. 7b and 7c).

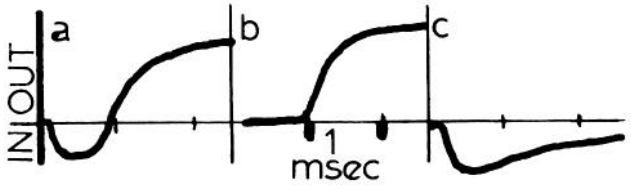

Fig. 7. Simulated currents in voltage-clamped axon. a. Normal axon with E_M= −4mV. b. Same with sodium activation blocked. c. Same with potassium activation completely blocked.

12. With the voltage clamp off simulate an EPSP by increasing both g_{Na} and g_K to 1 mmho/cm^2 for 1 sec (Fig. 8a).
13. Simulate an IPSP by increasing g_K and/or g_{Cl} to 29 mmhos/cm^2 for 1 sec (Fig. 8b).
14. Reduce $|E_K|$ so that $E_M < E_K$. Show that an increase in g_K which would normally produce a hyperpolarizing IPSP now produces a depolarization. Describe how this property could be used in a real experiment to determine whether an IPSP is due to a change in g_K.
15. Demonstrate synaptic inhibition by simulating a train of EPSPs being interrupted by IPSPs. Begin with a series of EPSPs at a rate of one every 10 sec (simulating a frequency of 1000/sec), then add IPSPs at the same rate (Fig. 8c).

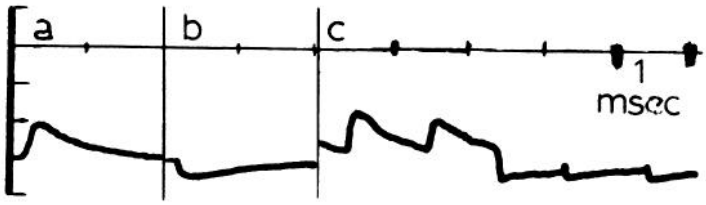

Fig. 8. Simulated synaptic potentials. a. An EPSP. b. An IPSP. c. Simulation of a train of EPSPs interrupted by IPSPs.

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INCONSISTENCY OF THE VAN'T HOFF-SCHOLANDER MECHANISM OF OSMOSIS

R. Howard[1] and H. Bradner
Scripps Institution of Oceanography
University of California, San Diego
La Jolla, CA 92093

With the recent appearance of a book by Hammel and Scholander (1), a mechanism of osmosis first suggested by van't Hoff in 1887 has been extended to become a theory popularly accepted by many physiologists. The ideal gas analog of van't Hoff is given in most textbooks on chemical thermodynamics and is commonly recommended (2) as an appropriate model for pedagogical use provided that its limitations are noted. The extensions proposed by Scholander and Hammel have been summarized in several articles (3) and have been sharply criticized by Andrews (4) and Plesset (5) among others.

Here it is simply shown that the asymmetry of the theory leads to a logical inconsistency. Nevertheless, for dilute, ideal solutions the van't Hoff mechanism as visualized by Scholander can provide a pictorial representation that may still be of some further pedagogical value.

Hildebrand (6) has nicely re-stated that if a solution is "ideal", in the sense of the solute following Henry's law and the solvent following Raoult's law, then its osmotic pressure can be expressed in terms of composition.

$$\pi \bar{v}_1 = RT \ln (N_1 + N_2)/N_1 \qquad (1)$$

where π is the osmotic pressure, N_1 the number of moles of solvent, N_2 that of the solute, and $\bar{v}_1$ is the partial molar volume of the solvent, i.e., the increase in volume of the solution when a mole of solvent is added to an infinitely large volume of solution.

With dilute solutions, the logarithm can be expanded, and neglecting higher powers we may write

$$\pi \bar{v}_1 = N_2/N_1 \, RT \qquad (2)$$

Setting $c_2 = N_2/N_1 \bar{v}_1$ we obtain van't Hoff's expression

$$\pi = c_2 RT \qquad (3)$$

which he recognized as analogous to the state equation for an ideal gas. With the expansion, it is readily apparent that Hammel and Scholander are incorrect when they claim that "the osmotic pressure of ideal solutions faithfully reflect[s] the kinetic state of gases." That the gas constant is involved in an equation for solutions should be no more surprising to Scholander than that it is involved in the law of Dulong and Petit for the specific heat of solids. Afterall, the gas constant is but Avogadro's number times Boltzmann's constant, which is merely a conversion factor between ergs and degrees. What is most disconcerting, however, is the lack of any further justification by Hammel and Scholander for their first proposition that "van't Hoff's law can be extended to apply to all molecules in the solution and in the pure solvent and not restricted to a dilute solution."

As a consequence of this proposition, Scholander introduces his new concept of mutually non-interacting, independent solute and solvent pressures. He maintains with van't Hoff that any osmotic pressure of a solution is due solely to the solute pressure. Since total measured pressure, implicitly defined by Scholander as the sum of solute and solvent pressures, must equal the ambient hydrostatic pressure, he concludes the solvent pressure must often be negative! Considering a molar solution, he writes: "The solute pressure is +24 atm [gage], the solvent pressure is −24 atm and the solution pressure is ambient." These pressures are to be thought of as always present in the solution. Towit, even though osmotic pressure can manifest itself only in the presence of an appropriate membrane, the solute pressure within a solution is always positive, and the solvent pressure always below ambient, whether or not the solution is exposed to any membrane.

Scholander's extension from van't Hoff's original suggestion is that the solute can induce this tension in the solvent through bombardment of a "free surface" - although the experimental justification he suggests for this has been adequately discounted (7). Scholander had considered solutions upon which an external force, in addition to a membrane, was able to distinguish between the solute and solvent particles, e.g., a magnetic field selectively affecting a paramagnetic solute. Yet his analysis of the field effects was very much incomplete. To investigate Scholander's proposed solute-"free surface" interaction, more simple experimental setups could be considered that do not involve such fields. Since osmotic transport can be appreciably more rapid than diffusive phenomena, the apparatuses shown in Figure 1 could demonstrate osmosis when fresh water is carefully layered upon sea water, and the significance of the membrane rather than the free surface demonstrated.

The apparent paradoxes Scholander is able to envisage lead to a situation akin to that caused by Dingle in his repeated attempts to discredit special relativity. The answer here, of course, lies in the fact that any partial molar quantity is really a property of the solution as a whole, not an independent property of the component, e.g., the "density of solvent within solution" is not intrinsic to the solvent.

To demonstrate a true paradox of Scholander's mechanism, it is simple to reconsider the original experiment displaying osmosis that was done by Abbe Nollet in 1748. He found that when alcohol and water were separated by an animal bladder membrane, the water passed through the membrane into the solution of alcoholic spirits. Yet if such a solution is instead separated from the alcohol by a rubber membrane, then it is the alcohol which passes through into the solution. Meyer (8)

[1] R. Howard is the recipient of a Fannie and John Hertz Foundation fellowship.

had pointed this out in 1890 to van't Hoff who quietly ignored the observation just as Scholander has neglected it. The paradox of Scholander's solute and solvent pressures is readily apparent in Figure 2.

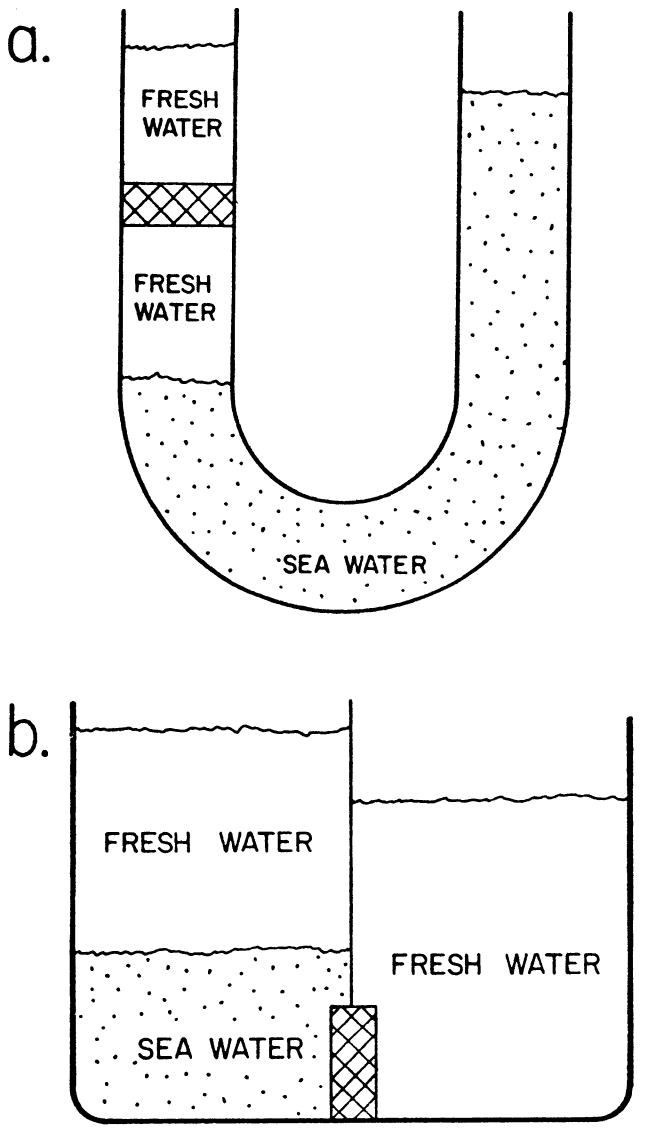

Fig. 1. With a membrane impermeable to salt but passing water, the setup shown in a. demonstrates osmotic flow without solute-"free surface" interaction and in b. (suggested by G. Wick) demonstrates no osmotic flow despite such interaction.

Given two substances, there is no *thermodynamic* reason that a semipermeable membrane could not exist that selectively passes either substance. This symmetry comes from the solution's blindness as to which is the solvent and which the solute, since only the membrane need differentiate between them. Since "water pressure" cannot be simultaneously negative, as in Scholander's solvents, and positive, as in Scholander's solutes, the logical inconsistency of the asymmetrical view is demonstrated.

Scholander's thinking seemingly originated from his careful experiments which showed close agreement between negative hydrostatic pressures in the xylem of trees, measured by compression within a bomb, and the osmotic pressures in the parenchyma cells as determined by the freezing point. However, no causal relationship between osmosis and tensility need be invoked. Water, due to its cohesiveness, can be drawn up a tube by stopping the top of a filled tube with a soaked sponge and dipping the bottom of the tube into a beaker of water.

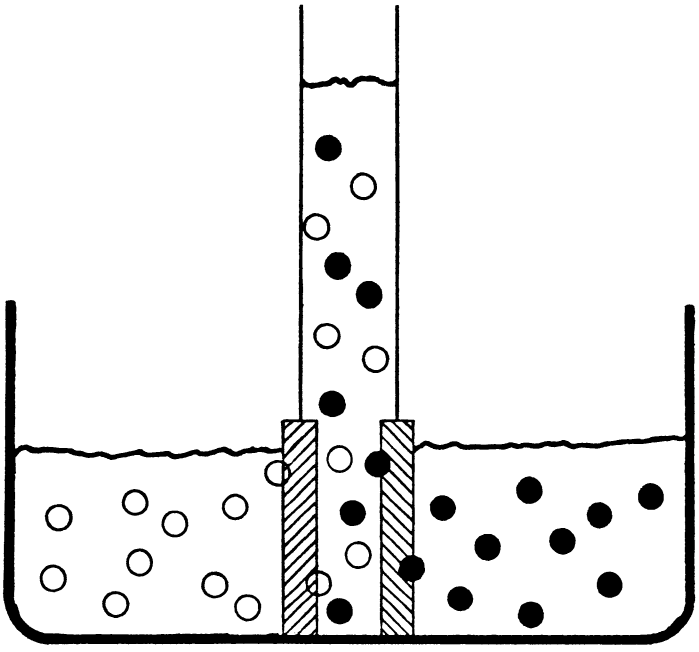

Fig. 2. Demonstration of the symmetry of solvent and solute. Two semipermeable membranes, each selectively permeable to only one component, stand between their permeating components and a solution. Neither component can be considered under "negative pressure".

The rate at which water moves up the tube is directly related to its rate of evaporation from the sponge. The osmotic pull of the cells on the water must of course be at least equal to the water's tension if the cell is to obtain nourishment. That Scholander measured them to be nearly equal only demonstrates the plant's efficiency in not excessively concentrating its intracellular solutions.

The theoretical treatment by Hammel and Scholander is very incomplete, and can lead to inconsistencies. Their neglect of any virial expansion for the equation of state does not encourage application of their picture to any real solution. Statistical mechanical models (9) provide much more hope for understanding solutions and gels as, for example, the McMillan-Mayer theory (10) which is particularly suitable for an investigation of osmotic systems. Nevertheless, Scholander's picture of a quasi-gas solution "stretched" by solute particles may continue to be attractive for pedagogical use. The theory of van't Hoff may indeed hold a position analogous to that of the Bohr theory of the atom, applicable to a simple system where it matches experimental values, conceptually wrong, but perhaps pictorially useful.

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NEWS RELEASE:

A LABORATORY BIOLOGY TEACHING LIBRARY

A laboratory biology teaching library is being established at The University of Calgary in order to improve communication among persons actively involved in undergraduate biology laboratory instruction.

Jon Glase (Cornell University), Don Igelsrud (University of Calgary), Patricia Paulus (Texas Christian University), and Ruth Von Blum (University of California, Berkeley) have formed a committee to establish the library and are sending a letter requesting written materials to 3500 institutions in the U.S. and Canada. The committee will identify a group of individuals actively concerned with problems of teaching biology in the laboratory so an organization can be established.

The main function of the library will be to collect in one place descriptions of biological systems and supporting materials that the contributor believes are reliable and effective in biology laboratory instruction. The committee is most interested in acquiring information about reliable, possible unique, biological systems for teaching undergraduates in a laboratory setting.

Contributors are being asked to send their current laboratory manuals and preparatory information for undergraduate laboratory courses indicating the authors of each exercise and annotating those materials which are particularly reliable and effective. Authors may send only their best materials if they wish. The materials will be used in the following ways depending on the contributor's preference:

1. For identification to help establish an organization and then held in confidence.
2. Available for study by visitors to the library.
3. Released for copying with the author's written permission.
4. Available for copying without permission of the author.

Unless otherwise stated the library will assume the contributor wishes the materials be used only to the first extend. With good participation, the library expects to obtain sufficient outside funding to produce a detailed index so persons with specific interests can communicate with each other. An index would be sent to each contributor.

A conference on laboratory instruction is planned for one of the national meetings in 1978 and will be organized in conjunction with the formation of the new organization. By establishing communication among laboratory instructors the group hopes to begin to solve some of the common problems confronted in the laboratory: finding and maintaining reliable living materials, producing modern laboratory exercises involving living materials and investigative inquiry, training others involved in the teaching of and preparation for laboratory classes, developing fair and relevant methods of examination, and so on.

The library was suggested by Don Igelsrud to identify persons actively working in biology laboratory instruction so that communication could be facilitated. The biology departments of The University of Calgary and Cornell University will fund the initial costs of the library. It will be located in The University of Calgary Biology Department which has a strong commitment to laboratory teaching, offers laboratories with appropriate courses, and has eight full time laboratory instructors, marine and fresh-water aquarium facilities, greenhouses, and a large support staff. The facilities and faculty are available during the summer months (May through August) and Calgary's close proximity to Banff and the Canadian Rockies make it ideal for winter as well as summer visits.

The group recognizes a need for an organization that addresses itself to the problems of biology laboratory teachers. Most papers on biological education are philosophical rather than practical in nature. Commercial laboratory manuals are often based on traditional procedures and materials that are unreliable. The methods, materials and experience required to greatly improve laboratory instruction do exist, however. For example, many reliable living organisms are available but have not been widely used in teaching because of poor communication. An organization of biology laboratory teachers would encourage the discussion of common problems and facilitate the improvement of laboratory programs.

Biologists who do not see a copy of the letter but are actively involved in laboratory instruction and are interested in participating should write to: Laboratory Biology Teaching Library, Department of Biology, The University of Calgary, Calgary, Alberta, Canada, T2N 1N4 (403) 284-6127.

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Editor: Orr E. Reynolds, Education Office, APS
Associate Editors: Robert E. Fellows, Dept. of Physiology and Biophysics, University of Iowa
Michael J. Greenberg, Dept. of Biological Science, Florida State University

Executive Editor: Mary Dittbrenner, Education Office

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