The flow velocities obtained with one such mixer were measured by orienting the axis of flow almost vertically and measuring the maximum height attained by the fluid under a stated driving pressure. The vertical exit velocity (V) was calculated at $V = (2gh)^{\frac{1}{2}}$, where g is gravity and h is maximum altitude. The velocity estimates are probably low because no corrections were made for air-friction effects or nozzle coefficient. Flow rates were measured by collecting the fluid passed by the mixers within a measured period.

Measurements on one model showed that, with a driving pressure of 6.8×10^4 newton/m^2 (gauge; 0.7 atm), the intracapillary flow velocity was 4.2 m/sec; the extracapillary, 2.55 m/sec. With the driving pressure increased to 13.6×10^4 newton/m^2, the velocities increased to 7.7 and 4.7 m/sec.

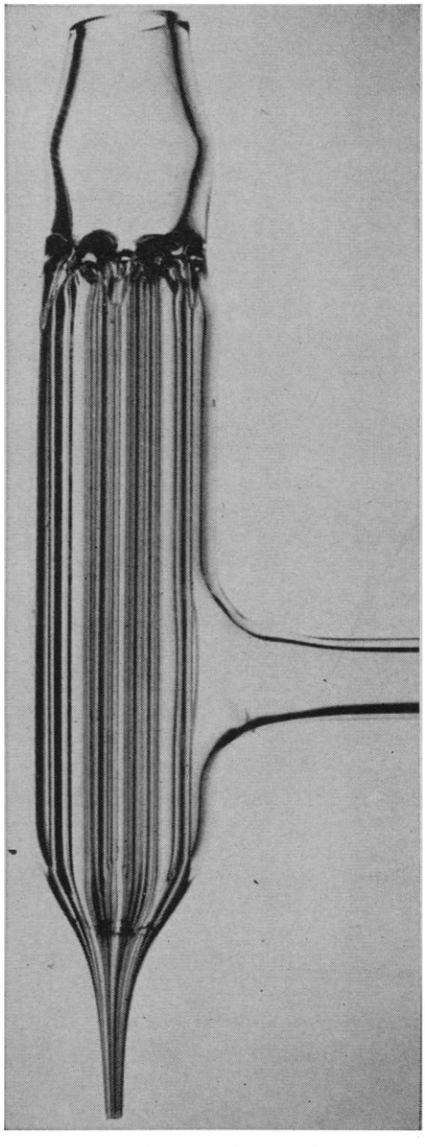

Fig. 1. Multicapillary mixer.

We have used two techniques to observe and analyze the mixing capabilities of the microcapillary system:

1) The production of a fluorescent compound by mixing two nonfluorescent solutions—quinine becomes fluorescent in an acid solution. A relatively nonfluorescent solution of quinine hydrochloride was passed through the intracapillary space, and acetic acid was forced through the extracapillary spaces, the mixture being a fluorescent acid solution.

2) The intracapillary solution was an alkaline 100-mg/liter solution of luminol (5-amino-2,3-dihydro-1,4-phthalazinedione). The other solution contained potassium ferricyanide (1 g/liter), with methylene blue or hemoglobin at several milligrams per liter. Production of visible light by combination of the two reactants was the indication of mixing.

The characteristics of the capillary mixer were determined by observation through a microscope or by scanning the mixing area with a photomultiplier using a 0.4-mm slit to define the measured region. At flow velocities of less than 0.5 m/sec the individual solutions remain unmixed for about 5 cm beyond the jet orifices, but, as the flow velocities increase, the pattern changes and the region of mixing moves closer to the orifices of the jets. While the fluid flow remains laminar, mixing at the boundaries results from diffusion of the reactants across the boundary; this mixing is ineffective, but, with increasing flow velocities, the point of turbulence approaches closer to the jet orifices and a sharply defined area of complete mixing is observed. With driving pressures of 3.4, 6.8, 10.2, and 13.6 newton/m^2 on the reservoirs of quinine and acid, one mixer gave flows of 0.51, 0.85, 1.10, and 1.33 ml/sec and velocities of 2.7, 4.2, 5.8, and 7.2 m/sec, respectively; under these conditions the point of maximum fluorescence was 2, 1, 0.25, and 0.20 mm away from the tip, which means that the mixing times were 0.74, 0.24, 0.044, 0.028 msec, respectively. The photomultiplier showed no observable change in light output beyond 0.75 mm away from the jet orifices when the driving pressure was 6.8×10^4 newton/m^2 or more. In order to ensure mixing in a sharp plane adjacent to the orifices without increasing the flow velocities, an electron-microscope grid (*2*) was placed within 0.1 mm of the orifice; although the grid added resistance to the flow, the light output indicated that solutions were completely mixed on emergence from the grid.

This system permits the simultaneous mixture of three or more reactants by addition of another set of capillaries. Use by the unit of very small quantities of reactant solutions makes it useful for studying biologic reactions for which materials are limited.

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Action of Anionic and Cationic Nerve-Blocking Agents: Experiment and Interpretation

Abstract. *Barbiturates and anesthetics similar to procaine bind to phospholipids in vitro. The former increase the binding of calcium to the phospholipids; the latter decrease it. The data can be correlated with the effects of these drugs on peripheral nerve. The nonpolar portion of the narcotic agents may lie between the lipid chains of the membrane, with the charged region in close approximation to the polar heads of the phospholipids.*

Most current views on the mechanism of anesthesia appear to fall into three categories: (i) biochemical theories which relate to oxygen consumption and oxidative metabolism (*1*); (ii) theories pertaining to the formation of hydrates (*2*); and (iii) theories relating directly to cell membrane phenomena, that is, lipid solubility and "surface effects." Limitations of the first two theories, which are concerned primarily with the central nervous system and mechanisms of general anesthesia, have been noted (*1, 3*). Evidence for the third category, which includes much of the data on the mechanism of local anesthetic action, includes correlations between anesthetic potency and solubility in media with low dielectric constants, polarizability, and penetration of lipid monolayers (*4*). Despite the wealth of chemical data, there has been little correlation of it with recent physiolog-

Table 1. Effect of procaine (2.0 m*M*) on the binding of calcium by phosphatidyl-L-serine (PLS) and synthetic L-α-cephalin (L-α-C).

Lipid	Bound Ca (μmole)	Inhibition (%)
	Control	
None	0.000	
PLS	.842	
L-α-C	.056	
	2.0 mM procaine	
PLS	0.698	17
L-α-C	.028	50

ical work (especially with voltage clamp experiments). Our studies were done in order to elucidate the mechanisms underlying the blocking action of certain anesthetics, especially anionic and cationic drugs, on peripheral nerve.

Anesthetics similar to procaine inhibit the increases in sodium and potassium conductances associated with excitation of the axon membrane (*5, 6*). That procaine is effective in the perfused, isolated axon provides strong evidence that local anesthesia is a membrane phenomenon (*7*). Feinstein (*8*) has shown that the binding of calcium to phospholipids is antagonized by anesthetics similar to procaine. We have confirmed and extended this observation and have also tested barbiturates and other anionic compounds.

Our methods were essentially those used by Feinstein for the measurement of calcium binding by phospholipids (*8*). One milliliter of a solution containing 116 mmole of NaCl, 2.5 mmole of KCl, 1.0 mmole $CaCl_2$ (omitted in some of the drug studies as noted in the tables below), and 2.0 mmole tris buffer (*p*H 7.4) per liter was combined with 2.0 ml of a mixture of two volumes of chloroform to one of methanol; the chloroform-methanol mixture contained 1.0 mg of phospholipid per milliliter. The aqueous phase contained 1 μc of Ca^{45} or 1 μc of a drug labeled with C^{14}. No lipid was added to the mixture of chloroform and methanol in the controls. The mixture (3 ml) was shaken for 10 minutes and then centrifuged for 5 minutes to separate the chloroform and aqueous phases. Both were assayed for radioactivity with a gas-flow or liquid scintillation counter. The lipids tested were synthetic L-α-cephalin (β, γ-dipalmitoyl-L-α-glyceryl phosphoryl-ethanolamine) and phosphatidyl-L-serine (*9*). Studies with C^{14}-labeled phosphatidyl ethanolamine demonstrate that under the conditions of these experiments more than 98 percent of the lipid remains in the chloroform phase.

Table 2. Effect of divalent cation (at 1.0 m*M* concentrations) on binding of procaine-C^{14} to phosphatidyl-L-serine. The initial concentration of procaine in the aqueous solution was 2.0 m*M*. $CaCl_2$ was omitted from the standard aqueous solution. The ratio of the concentration of procaine in the organic phase to that of procaine in the aqueous phase was calculated. In the absence of lipid, this ratio (O/A of text) was negligibly affected by the addition of these divalent cations.

Ion	Distribution ratio	Procaine bound (μmole)	Inhibition (%)
	Control		
None	0.160	0.000	
	Phosphatidyl-L-serine		
None	0.534	0.469	
Mg	.411	.338	28
Ca	.327	.238	49
Ba	.310	.217	54
Ni	.216	.082	83

In the absence of lipid, there is no calcium taken into the organic phase (Table 1). The amount of calcium taken into the organic phase in the presence of phospholipid can be used as a measure of the amount bound to the phospholipids; about 85 percent of the calcium is bound by the phosphatidyl serine and only about 6 percent by phosphatidyl ethanolomine (Table 1). Procaine (2.0 m*M*) decreases calcium binding to phosphatidyl serine by 17 percent and to cephalin by 50 percent.

The binding of C^{14}-labeled procaine to phospholipid was studied. The distribution of procaine between organic and aqueous phases was calculated both in the absence (O/A) and in the presence (O'/A') of phospholipid. The increased amount of drug in the lipid phase in the presence of phospholipid $[O' - (A'O/A)]$ was taken as the amount of drug bound to the phospholipid. There was significant binding of procaine to phosphatidyl-L-serine. The binding of procaine to the phospholipid was antagonized by divalent ions, in the order Ni>Ba>Ca>Mg (Table 2). Similar results were obtained with L-α-cephalin. It is interesting that in experiments on peripheral nerve, when the calcium of the extracellular fluid is replaced, mole for mole, by nickel, the effects are the same as when the concentration of calcium was increased (*10*). Barium is also a good substitute for calcium, while magnesium is a rather poor one (*10*). Furthermore, nickel antagonizes the reduction by procaine of the amplitude of the action potential (*11*) as do increased calcium concentrations (*6, 12*).

Table 3 shows the correlation between the antagonistic effects of a number of local anesthetics on the binding of calcium to phosphatidyl serine and their anesthetic potency relative to that of procaine. Although relative anesthetic potency in general depends upon the method of testing (*13*), it is obvious that the order of inhibition of calcium binding is similar to that of the relative anesthetic potency. We have also tested a series of tropine esters and found that only those that are effective nerve-blocking agents antagonize the binding of calcium to phospholipid (*14*).

Table 3. Effect of cationic drugs on calcium binding by phosphatidyl-L-serine. The relative inhibition of calcium binding was determined from the reciprocal of the concentration of drug necessary to inhibit calcium binding to phosphatidyl-L-serine by 20 percent. The relative anesthetic potency was determined on frog sciatic nerve trunk at *p*H 7.18 to 7.20 (*12*).

Drug	Relative inhibition of Ca binding	Relative anesthetic potency
Procaine	1.0	1.0
Mepivacaine	1.6	2.1
Lidocaine	3.5	3.8
Hexylcaine	4.4	2.7
Piperocaine	4.7	3.5
Tetracaine	8.7	36.5
Dibucaine	10.1	53.8

Table 4 shows the effect of thiopental on calcium binding by phospholipids at *p*H 8.0. The increase in binding caused by the drug is dependent on its concentration and is less marked at *p*H 7.4 and *p*H 7.0. Similar results have been obtained with pentobarbital and phenobarbital. Pentobarbital labeled with C^{14} binds to phospholipid,

Table 4. Effect of anionic drugs on calcium binding to phospholipids at *p*H 8.0. The concentration of thiopental was 2.0 m*M* (in suspension), that of sodium lauryl sulfate, 1.0 m*M*. In the calculation of the percentage increase a correction was made for calcium taken into the organic phase in the absence of phospholipid.

Drug	Lipid	Bound Ca (μmole)	Increase (%)
None	None	0.002	
Thiopental	None	.012	
None	PLS	.834	
Thiopental	PLS	.956	15
None	L-α-C	.062	
Thiopental	L-α-C	.139	124
None	None	.001	
Sodium lauryl sulfate	None	.026	
None	PLS	.068	
Sodium lauryl sulfate	PLS	.930	12
None	L-α-C	.085	
Sodium lauryl sulfate	L-α-C	.286	206

and the amount bound is slightly increased in the presence of calcium (Table 5). Similar effects are obtained with C^{14}-phenobarbital.

The anionic detergent, sodium lauryl sulfate, also increases the binding of calcium to phosphatidyl serine (Table 4). Although this agent is structurally unrelated to the barbiturates, it has, as do the barbiturates, an acidic group attached to a lipid-soluble moiety. Like the barbiturates (*15–17*), it decreases the amplitude of the action potential in peripheral nerve (*18*). Sodium lauryl sulfate also affects the position on the voltage axis of the curves of membrane conductance of the voltage clamped squid axon in much the same way as does raising the external calcium concentration (*18*). Data from voltage clamp studies of the lobster axon (*19*) suggest that treatment with pentobarbital or thiopental causes a reduction in the magnitude of the membrane sodium and potassium conductance increases, as well as in the rate of increase of sodium conductance. Both procaine (*5, 6, 20*) and calcium (*20, 21*) are known to affect these same membrane parameters.

On the basis of the effects of calcium on the electrical properties of nerve fibers, it has been proposed (*21*) that the first step in the depolarization of a nerve may be the removal of calcium from some site on the membrane to which it is bound. The data suggest that the polar heads of phospholipids present in nerve membranes (*22*) may be these binding sites. The action of the anionic and cationic anesthetics could then be accounted for by the molecular models shown in Fig. 1. We would expect the organic, nonpolar portions of the drug molecules to lie between the fatty acid chains of the lipid molecules, with the polar heads of the drug molecules in close proximity to the polar heads of the phospholipids. If the drug were a cation, such as procaine, its polar head would tend to neutralize the negatively charged site on the phospholipid so that fewer sites would be available for the binding of divalent cations. Conversely, if the drug were an anion, such as pentobarbital, its negatively charged head would tend to neutralize the cationic nitrogen of the phospholipid, so that there would be an increased tendency for divalent ions to bind to the phospholipids. That both phenobarbital (*15*) and procaine (*23*) are able to counteract some of the effects of low calcium on nerve may be related to

Table 5. Binding of pentobarbital-C^{14} to phospholipids, at *p*H 8.0. All ions were tested at 1.0-m*M* concentrations. The distribution ratio is the ratio of the concentration of pentobarbital in the organic phase to pentobarbital in the aqueous phase. The initial concentration of pentobarbital in the aqueous solution was 2.0 m*M*. $CaCl_2$ was omitted from the standard aqueous solution for these studies.

Ion	Ratio	Pentobarbital bound (μmole)	Increase (%)
		No lipid	
None	7.3	0.000	
Ca	7.3	.000	
Ni	7.8	.000	
		Phosphatidyl-L-serine	
None	8.0	0.190	
Ca	8.1	.194	2
Ni	8.9	.229	21
		L-α-*cephalin*	
None	7.6	0.070	
Ca	7.7	.079	13
Ni	8.5	.140	100

polar group interactions such as these.

It has been postulated (*24*) that the polar heads of the phospholipids are concerned with the mechanism responsible for the changes in the conductance of sodium and potassium during excitation. Binding of the polar heads of the phospholipid by the anesthetic drugs would result in a decrease in their ability to change configuration and ion specificity under the influence of a change in the electric field. This should reduce the magnitude and rate

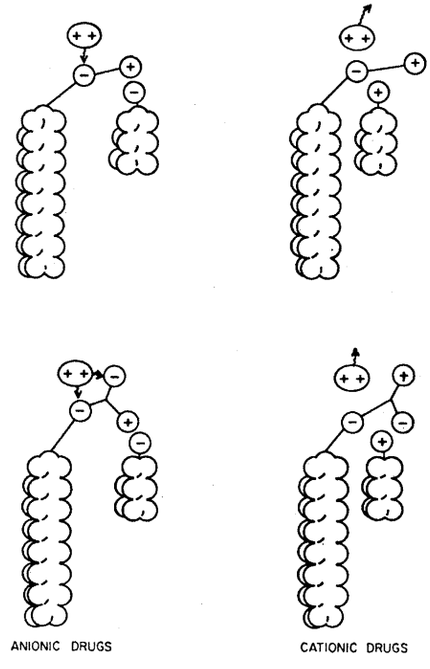

Fig. 1. Molecular models of interactions between divalent cations, anionic and cationic drugs, and phospholipid polar heads. (Top) Phosphatidyl ethanolamine; (bottom) phosphatidyl serine.

of sodium and potassium conductance increases in the nerve membrane, with a consequent reduction in the amplitude of the action potential.

That the local anesthetics which are similar to procaine seem to be more active in their ionized form (*25*), and that some quaternary analogs of these molecules (*20, 26*) are effective as local anesthetics in isolated axons, further emphasizes the contribution of the polar groups to the production of anesthesia. The fact that many polyvalent ions such as Fe^{3+}, Al^{3+}, La^{3+}, Cr^{3+}, Co^{2+}, and Cd^{2+} reversibly block the action potential of the nerve (*10, 27*) provides further evidence that interaction between charged groups in the membrane and charged blocking agents is either partly (in the case of the barbiturates or of drugs similar to procaine) or entirely (in the case of the polyvalent ions) responsible for the production of the block.

The anesthetic potency of drugs has, in general, been rather well correlated with their solubility in media with low dielectric constants, their polarizability, or their ability to penetrate lipid monolayers (*4*). Voltage clamp studies (*28*) have demonstrated that the primary effect of alcohols is to decrease the sodium and potassium conductances of the membrane without significantly affecting the time parameters for the initiation of the sodium conductance. On the other hand, many of the inorganic polyvalent cations seem primarily to affect the rate parameters of the ionic conductances and only secondarily to decrease the maximum conductance values at large depolarization steps (*6, 10, 20, 21, 27*). We conclude that ionic conductance through the nerve membrane involves both the polar heads of the phospholipid and the lipid bilayer. Ions which can form a sufficiently strong bond with the polar heads of the phospholipids do so; ions which do not, cannot act as anesthetics unless their solubility in media of low dielectric constants is great enough to coax them into place in the membrane. Inhibition of electrically induced changes in the configuration of the polar head of the phospholipid, increase in the pressure within the lipid layer by penetration of lipid soluble material, or both, interfere with excitability phenomena; the former particularly by slowing the conductance increases, the latter by reducing the magnitude of these increases. Some overlap of these effects may occur.

Although this discussion has been

concerned primarily with the effects of several drugs on peripheral nerve, the fact that most of these drugs block synaptic transmission as well as axonal conduction suggests that a similar ion-binding process may also participate at the synapse (*17, 18*).

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Scaling of Musical Preferences by the Mentally Retarded

Abstract. *The ability of institutionalized retardates in scaling musical preferences was compared with that of normals. The retardates' scale values and scale forms obtained by two kinds of scaling procedures are very similar to those of normals. Deficits, however, are observed in their lower internal consistency and relative uncertainty and in higher response polarization and perseveration.*

The scaling of musical preferences, a matter of linking the number system to a preference schema, is possible only when a person is reasonably familiar with the properties of numerical series and has developed his preference schema for music. Moreover, there must exist a certain degree of isomorphism between these two personalized systems. We have tried to assess the ability of mentally retarded adults in scaling musical selections, to clarify the nature of scale forms and response patterns, and to infer the underlying numerical and preference structures, comparing them with those of normal adults.

The subjects were 106 institutionalized retardates (54 men and 52 women), 54 aides in a state hospital (24 men and 30 women), and 42 college students (23 men and 19 women). The mean chronological ages, mental ages, and Stanford-Binet IQ's for the retardates were, respectively, 35.9 years (σ, 6.9), 8.5 years (σ, 1.2), and 56.4 (σ, 7.3) for men; and 35.7 years (σ, 6.4), 8.9 years (σ, 1.4), and 59.4 (σ, 9.2) for women. Retardates whose mental age was less than 6 years were excluded. The causes of retardation were: familial, 38; uncertain, 65; and infection, 3. The mean age and education for the aides were 45 years (σ, 10.3) and 10 years (σ, 2.3). The college students were mostly freshmen and sophomores.

Thirty vocal excerpts selected from a previous study (*1*) were presented by tape recorder to groups of 10 to 15 subjects; they included opera singing, folk songs, college songs, and stage music, with instrumental backgrounds; each excerpt lasted about 60 seconds. Both category- and magnitude-scaling procedures, which yield ordinal and ratio scales, respectively, were employed (*2*). For the category scale, each subject was instructed to assign to each excerpt one of seven (for retardates) or nine (for normals) ordered responses ranging from most pleasant, through indifferent, to most unpleasant, in accordance with his impression. For the magnitude scale a standard excerpt was played first, and the subject was instructed to indicate his reaction to each subsequent excerpt by assigning a number proportional to that for the standard piece. The group scale value of each excerpt for the category scale is the mean category number; that for the magnitude scale is the geometrical mean of the assigned numbers. Both procedures were administered in a single sitting, with two presentations of each of the 30 excerpts. Extensive explanation of the procedures and some practice were necessary for the retarded groups.

The retardates' (as a group) preference for vocal selections closely resembled that of the normals. The product-moment correlations between retardates and aides and between retardates and students for the category scale range from .88 to .92; those for the magnitude scale are .84 to .94; correlations between aides and students are .86 to .98. In all groups the women tend to show higher correlations, but the sex differences are not significant. The correlations between men and women among the retardates (.86, .89) are slightly lower than among the normals (.94 to .97).

Since two different kinds of scaling procedure were applied to the same 30 excerpts, preference agreement between these two scales could serve as a measure of internal consistency. The retardates, as a group, again show high inter-scale correlations comparable to those of the normals (Fig. 1). In order to