Whole-wheat flour was found to be much richer than white flour in the factor stimulating pneumococcal growth *in vitro*. This suggested that mice eating whole-wheat bread might be more susceptible to pneumococcal infection than those eating white bread. The results of two experiments designed to test this possibility are shown in Table 1. The mice were maintained on either white or whole-wheat bread for 6 days before the injection of the pneumococci (10^{-4} or 10^{-6} ml. of a 17-hour culture). In both experiments the mice eating white bread were more resistant to the infection than those receiving whole-wheat bread. Thus, only 1 of 49 mice (2 per cent) on the whole-wheat diet survived the infection, whereas 20 of the 50 (40 per cent) eating white bread survived 6 days (and presumably indefinitely). In line with previous experience, the difference in response between the two dosages was small (*1*).

Obviously, these experiments provide insufficient evidence on which to decide the white vs. whole-wheat

TABLE 1

EFFECT OF DIET ON THE SURVIVAL OF MICE INFECTED WITH PNEUMOCOCCUS TYPE I

Exp. No.	Diet	Dose	No. of mice	Number surviving on day indicated						Average survival (days)
				1	2	3	4	5	6	
I	Whole-wheat bread	10^{-4}	24	24	9	4	3	1	0	1.71
	White bread	10^{-4}	25	25	19	16	14	13	11	3.92
II	Whole-wheat bread	10^{-6}	25	25	9	6	2	1	1	1.76
	White bread	10^{-6}	25	25	19	12	12	10	9	3.48

bread controversy or to base a dietary treatment of pneumonia. However, they do cast considerable doubt on one tenet of the nutritionist's credo. It is generally assumed that, when known essentials are present in equal amounts, a cruder foodstuff is to be preferred to a refined. This point of view implies that the unknown factors of the crude foodstuff are always beneficial. The assumption has an a priori validity which is supported by the history of the isolation of the vitamins and has not, up to now, been contradicted experimentally. The experiments described in this paper provide such a contradiction and cast doubt on the validity of the basic assumption. They show that, in one instance at least, the unknown factor of the crude foodstuff is more beneficial to the parasitic organism than to the animal consuming the food.

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The Production of Electricity by Nerve

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Five years ago we reported (*7*) that acetylcholine produces a pronounced phase-boundary potential of negative sign at the junction of oil and saline. It was suggested that this acetylcholine potential is the basis of the electrical negativity which arises in nerve during activity. A long series of additional experiments (*1–9*) have supported this theory that the chemical mediator of the nerve impulse, acetylcholine, sets up a negative phase-boundary potential in the lipoid layer of the nerve fiber—a theory which reconciles the "chemical" and "electrical" theories of nervous transmission. In a recent review, Feldberg (*12*) states that our experiments explain the "depolarizing" action of acetylcholine at synapses and at the end-plate. The term "depolarize" does not imply that acetylcholine renders an imaginary sieve membrane permeable to ions like potassium. We have previously shown (*6, 9*) that the old Bernstein concept of an ionic sieve membrane is untenable on both theoretical and experimental grounds. Actually, the lipoid-soluble acetylcholine dissolves in the oil layer to a much greater extent than the saline, thus establishing a true phase-boundary potential which we can demonstrate on an oil layer several centimeters thick, thereby eliminating any "permeability" change in an imaginary sieve membrane.

Recently the "oil-cell" model of the nerve impulse previously described (*7*) has been modified to conform to "physiological" conditions. For example, cholesterol (from spinal cord) or brain extract is added to the oil layer, thereby increasing the phase-boundary potential. Seventeen grams of cat brain extracted with 20 cc. of guaiacol at 80° C. was cooled and filtered. The resulting brain solution in guaiacol gave 45 mv negative with 0.05 per cent acetylcholine, in contrast to 30 mv established by 0.05 per cent acetylcholine on guaiacol without brain extract. Thus, acetylcholine can produce a phase-boundary potential with brain substance.

The addition of human serum which contains "serum cholinesterase" and small amounts of "cell cholinesterase" (*14*) to the saline phase brings the "oil-cell" model still closer to living nerve. Four cc. of human serum added to 200 cc. of 0.9 per cent NaCl with bicarbonate to make the pH 8.2 (at 37° C. for 8 hours) destroys the electrogenic activity of 0.05 per cent acetylcholine as tested in the "oil-cell." This experiment shows that it is the acetylcholine and not

the esterase which produces the electrical nerve impulse. These results help to explain the absence of severe symptoms in persons whose esterase is reduced to only 1 per cent of its normal value (*10*). In fact, Fraser (see *12*) nearly 80 years ago showed that eserine does not depress nerve-trunk activity. It is possible, however, that under certain conditions eserine may block impulse transmission by the negative phase-boundary potential established in contact with lipoids (*5*).

The descending part of the electrical wave in the electric fish and in the ganglion (*12*) is prolonged by eserine because the acetylcholine phase-boundary potential persists for a longer time, but no experiments have shown modification of the descending phase of the spike potential in nerve by eserine or other anticholinesterase drugs.

The rise and fall of the spike potential in peripheral nerve can be duplicated in the oil-cell by placing the acetylcholine first on one side and then on the opposite side of the oil layer. With resin in the oil a film less than 0.1 mm. thick can be formed by pressure on the saline on one side. Under these conditions a potential of 30 mv, produced by 0.05 per cent acetylcholine, fell to zero in 4 hours, due to the penetration of the alkaloid to the opposite side. With very thin films of oil the spike rose and fell too fast for measurement with the potentiometer attached to the "oil-cell."

Careful measurements of the phase-boundary potential of possible products of nerve metabolism (potassium, lactic acid, lactate, phosphate, acetate, choline, citric acid) have shown that acetylcholine has a much greater electrogenic property and is the only substance so far studied capable of producing the action current in nerve. For example, as much as 1 per cent KCl is necessary to produce 10-mv negativity on cholesterol in guaiacol, and 5 per cent lactic acid produces only 10-mv positivity in contrast to the pronounced effect of dilution of acetylcholine previously described (*7*). There remains the possibility that the small negative after-potential is set up by choline and the two small positive after-potentials by acetate and phosphate.

The distinction between cholinergic and adrenergic nerves receives its first explanation by the phase-boundary theory. Triglyceride oils (*2, 4*) establish potential with sympathomimetric but not with parasympathomimetric drugs. We have recently found that choline as well as acetylcholine is inactive on triacetin (which gives potentials with epinephrine, benzedrine, etc.). When present, the phase-boundary potentials of epinephrine and acetylcholine are both negative, which suggests that these substances may potentiate under certain conditions, which is indeed the case (*11*).

The study of phase-boundary potential can be applied directly to nerve. Frog sciatic nerve is immersed in isotonic glucose for 1–2 hours (to eliminate short circuits by salts). The ends are tied, and the nerve forms a loop between two watch glasses of isotonic glucose connected to our potentiometer as previously described for the oil-cell (*7*). Addition of 1:160,000 acetylcholine to the solution bathing the part of the nerve in one dish sets up a phase-boundary potential of 10-mv negativity (which decreases in magnitude with time). Lorente de Nó (*13*) did not detect electrical changes in nerve treated with acetylcholine, since he failed to use isotonic sugar as recommended by Netter (*15*).

The experiments reported above support the theory that the electrical nerve impulse is a phase-boundary potential produced by acetylcholine in contact with nerve lipoid.

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Scanning Science—

The American Physiological Society is holding its ninth annual meeting at Boston and Cambridge on December 29th and 30th. Headquarters will be at the Hotel Brunswick. Those who require apparatus or other necessities for making demonstrations may communicate with Dr. H. P. Bowditch. R. H. Chittenden is president of the Society and Frederic S. Lee is secretary.

The American Psychological Association will meet at the same time and Prof. G. S. Fullerton, its president, will make an address on the 29th.

The American Society of Naturalists, also meeting at the same time, will join with the Psychologists in a discussion of "Inheritance of Acquired Characteristics" in which J. M. Macfarlane, C. S. Minot, E. D. Cope and William James will take part. Prof. E. B. Wilson will lecture on "Recent Developments of the Cell Theory."

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