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SOME PHYSIOLOGICAL AND BIOCHEMICAL CONSEQUENCES OF PSYCHOLOGICAL POSTULATES*

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(1) *Introduction.*—This note lists some psychological, physiological, and biochemical predictions that have been derived from simple psychological postulates. These psychological postulates have been used to derive a new learning theory,[1–3] which is called the theory of embedding fields. The mathematical variables of the theory have natural psychological labels—such as "presentation of a letter or spatial pattern at time t_1," "guess of a letter at time t_2," "stimulus trace," "associational strength," etc.—due to the fact that the theory is derived on a psychological basis. *Given* the psychologically derived theory, one then observes that its mathematical variables are *already* in a form that suggests a neurophysiological, anatomical, and in some cases biochemical labeling for these variables. For example, the theory contains geometrical objects which are readily identified with cell bodies, axons, synaptic knobs, and synapses. It also contains, associated with the geometrical objects, dynamical variables that readily call to mind membrane potentials, spiking frequencies, transmitter substances, various ions, and the like. Once the mathematical variables are labeled with these suggestive physiological and anatomical labels, the *psychologically* derived laws of the theory thereupon imply functional relationships between these empirical variables, as well as a psychological rationale for the existence of these relationships in terms of how the brain might learn, remember, and recall what it has learned.

Naturally the leap from mathematical to neural variables cannot be justified in a deductive way. It is governed, as is inevitable, merely by rules of prudence and the dictates of intuition. Fortunately, the simplest neural labeling seems often to yield functional relationships which represent, at least qualitatively, known and nontrivial neural data. In other cases, the functional relationships seem never to have been measured, and therefore stand as new predictions. The strength of such predictions is, of course, no greater than the correctness of the neural labeling, and an assessment of this requires a close scrutiny of the theory's development.[1–3]

We have also begun a rigorous mathematical analysis of the learning, memory, and recall capacities of the theoretical equations in various experimental situations.[4–10]

(2) *Some Qualitative Results.*—(*a*) The equations reduce in a special case to the Hartline-Ratliff equation for lateral inhibition in the *Limulus* retina.[11] Theoretical formulas for the empirical coefficients in the H-R equation are found, and various transients can be readily studied. A new phenomenon of "enhancement of associations" or "spontaneous improvement of memory," closely related to "contour enhancement" due to lateral inhibition, is found.[2] It shares many properties with the Ward-Hovland phenomenon, or "reminiscence."[12] The

"accumulation of inhibition" postulated by Hull[12] to explain bowing in serial verbal learning is identified with lateral inhibition.[2, 13]

(*b*) A unified formal explanation is given of various serial learning phenomena,[13] such as backward learning, bowing, anchoring, chunking, response oscillation, All-or-None versus Gradualist learning, and Gestalt versus Peripheralist learning.

(*c*) A unified formal explanation of the decrease of reaction time with increased learning and of spatiotemporal masking is found.

(*d*) The level of excitatory transmitter production is controlled *jointly* by presynaptic and postsynaptic levels of membrane excitation.

(*e*) Learning needs suggest the interaction of no fewer than two pairs of antagonistic ions, say (Na^+, K^+) and (Ca^{++}, Mg^{++}).

(*f*) Na^+ and Ca^{++} are bound as synergistic cofactors on the intracellular sites, or enzymes, which activate the production of excitatory transmitter, say acetylcholine.

(*g*) There exists a spiking threshold, greater than the cell body equilibrium potential, above which average spiking frequency is proportional to cell body membrane potential (after excitatory transients subside and before saturation sets in).

(*h*) Presynaptic spiking both mobilizes and depletes transmitter. Whereas the steady-state mobilized transmitter that is *released* per unit time increases as a function of steady-state spiking frequency and saturates at a finite value, the *total* steady-state mobilized transmitter decreases as a function of spiking frequency.

(*i*) A slowly varying form of post-tetanic potentiation occurs in the synaptic knobs.

(*j*) An excitatory transient in transmitter release occurs when presynaptic spiking is resumed after a rest interval.

(*k*) The amount of intracellular acetylcholine is regulated in part by a feedback inhibition within the synaptic knob of transmitter onto a previous stage of transmitter production. This inhibition affects an intermediate or terminal stage of transmitter production, rather than an initial stage.

(*l*) K^+ is more likely to be found in unbound form within the synaptic knob than are Na^+ and Ca^{++}.

(*m*) The ionic movements suggested by learning needs are compatible with some data concerning the pattern of ion translocation in the mitochondrion, and with the assumption that these movements make adenosine 5′-triphosphate available for production of acetyl-Co A, and thereupon acetylcholine, under the guidance of choline acetylase in the synaptic vesicles (see, e.g., ref. 14).

(*n*) A mechanism is found which makes plausible the distribution of synaptic vesicles and mitochondria near the synapse of the synaptic knob, rather than (say) uniformly distributed throughout the knob.

(*o*) In response to excitatory transmitter, there exists an inward flow of Na^+ through the cell membrane which is coupled at suprathreshold values to an outward flow of K^+.

(*p*) In response to inhibitory transmitter, there exists an outward flow of K^+ through the cell membrane.

(*q*) Acetylcholine release from synaptic knobs is coupled to the intracellular K^+ concentration.

(*r*) The sensitivity of RNA activation to Mg^{++} concentration is compatible with the need to guarantee control by membrane excitation of intracellular production levels, say of proteins, and thus

(*s*) membrane excitation due to learning experiments causes systematic variations in nuclear RNA, although individual RNA strands do not encode entire behavioral memories, which are spread over many cells.

(*t*) Learning needs suggest a cell nucleus which is localized in the cell body, rather than being spread throughout the cell. More generally, various functions performed by nerves as learning mechanisms seem to determine their shape, at least qualitatively.

(*u*) A system of intracellular tubules, such as in endoplasmic reticulum, is compatible with the need to carry chemicals used in learning between cell body membrane and nucleus and from the nucleus along the axon and to the synaptic knobs.

(*v*) In an *idealized* nerve cell (say without dendrites), cell body membrane area is proportional to nuclear volume and to the membrane area of axon and end-bulbs. This is a special case of the general property of spatiotemporal self-similarity, which is apparent in many biological shapes and interactions (e.g., shape of leaves, proportionality of axon diameter, and velocity of spike along the axon).

(*w*) The size of a cell in a given idealized cell type can, in principle, be controlled by a single gene whose activity is sensitive to the average total membrane excitation.

The theory can also be used to illustrate in various cases how particular anatomical cell distributions and multiple somatotopic representations might be used to perform particular tasks of learning and performance, such as in the sensory-motor cortex, cerebellum, and retina.

(3) *Postulates and Equations.*—The psychological postulates that lead to the equations which describe our learning machines M are quite simple. The following discussion heuristically describes these postulates in the case of learning a list of "simple" letters or events, such as the alphabet $ABC \ldots Z$.

(*a*) The letter A is never decomposed into two or more parts in daily speech and listening. It is a "simple" behavioral unit. Thus we assign to every simple behavioral unit r_i a single abstract point v_i in M, $i = 1, 2, \ldots, n$. (As the theory becomes more microscopic, even simple events create a space-time trajectory of excitation and inhibition that includes many points, which are ultimately "blown up" and identified as caricatures of nerves.)

(*b*) M must react to presentation of behavioral units at specified times. Hence a real-valued function of time $x_i(t)$ is assigned to each point v_i. The value of $x_i(t)$ at any time describes how recently r_i has been presented to M.

(*c*) Consider M's response to presentation of A, then B, and then C at a speed ω. If ω is small (say $\omega \cong 2$ sec), then the influence of A and B on M's response to C is substantial. As ω increases, the influence of A and B on M's response gradually changes and ultimately becomes negligible. Since the effects of prior

presentations of events wear off gradually, each $x_i(t)$ is continuous. Since our theory describes the macroscopic behavior of M, we can also readily assume that each $x_i(t)$ is differentiable.

(*d*) If r_i is never presented to M, then $x_i(t)$ remains at a fixed equilibrium value, which is (initially) set equal to zero. If r_i is presented to M at time $t = t_i$, then $x_i(t)$ must at least temporarily assume nonequilibrium values once $t > t_i$. We assume that $x_i(t)$ becomes positive after $t = t_i$, by convention. Since the effect of an event ultimately wears off, $x_i(t)$ eventually decays towards zero. (The choice of a zero equilibrium value tacitly assumes that all $x_i(t)$'s values are *observable* to a psychological experimenter. This assumption must ultimately be abandoned, for reasons that soon become clear.)

(*e*) *After* M has learned the list AB, a presentation of A to M at time t_A gives rise to the guess B by M a little while later, say at time $t_A + \tau_{AB}$, where τ_{AB} is positive. Thus a signal travels from v_A to v_B at finite velocity along a pathway e_{AB}.

(*f*) *Before* M has learned the list AB, other responses than B to A must exist, or else B would already be the only response to A. Thus a function $\dot{z}_{AB}(t)$ exists which can distinguish the presentation or nonpresentation of AB and lets only B occur in response to A after AB has been learned. Since $z_{AB}(t)$ grows only if A and then B are presented to M, $z_{AB}(t)$ *correlates* (prescribed) past values of x_A with $x_B(t)$. $z_{AB}(t)$ therefore occurs at the only position at which past x_A and present x_B values exist, namely, at the end of the pathway leading from v_A to v_B.

(*g*) The list AB is not the same as the list BA. Thus $e_{AB} \neq e_{BA}$, and $z_{AB}(t) \not\equiv z_{BA}(t)$. e_{AB} is drawn as an *arrow* from v_A to v_B with arrowhead N_{AB}. By (*f*), $z_{AB}(t)$ occurs in N_{AB}.

(*h*) If C is not said, then AB can be learned in first approximation independently of CB. Thus the signals received by B combine independently.

When the postulates (*a*)–(*h*) are translated into mathematical terms, the following equations are found as, perhaps, their simplest realization.

$$\dot{x}_i(t) = -\alpha x_i(t) + \beta \sum_{m=1}^{n} x_m(t - \tau_{mi}) p_{mi} z_{mi}(t) + I_i(t), \tag{1}$$

$$\dot{z}_{jk}(t) = -u z_{jk}(t) + \beta p_{jk} x_j(t - \tau_{jk}) \bar{x}_k(t), \tag{2}$$

where $i, j, k = 1, 2, \ldots, n$; α, β, and u are positive; all τ_{jk} are positive; all p_{jk} are nonnegative; and all initial data are nonnegative and continuous. The nonnegative and continuous inputs $I_i(t)$ often have the form

$$I_i(t) = \sum_{k=1}^{N_i} J_i(t - t_i^{(k)}), \tag{3}$$

where $t_i^{(k)}$ is the kth onset time of r_i, and $J_i(t)$ is a given nonnegative and continuous function that is positive in a finite interval of the form $(0,\lambda_i)$.

Equations (1) and (2) can be given a qualitative neural interpretation that includes cell bodies, axons, synaptic knobs, synapses, membrane potentials, spiking frequencies, and transmitter production and release.[1] These equations are not totally satisfactory because of the hypothesis (*d*) of observability. By including the following additional postulate, they can be improved without

violating (*d*) in the special case that all reaction times τ_{ij} have the same value τ.

(*i*) M can learn AB perfectly by practicing AB sufficiently often. This postulate is achieved by implementing the following property. *Increasing* the strength of the choice B, given an isolated presentation of A, *decreases* the strength of the choices C, D, E, ..., etc. In other words, a "set of response alternatives" to isolated presentations of A exists, and these alternatives *compete* with one another. This property has the effect of reducing behaviorally irrelevant background noise.

Then (1) and (2) are replaced by

$$\dot{x}_i(t) = -\alpha x_i(t) + \beta \sum_{m=1}^{n} x_m(t-\tau) y_{mi}(t) + I_i(t), \tag{4}$$

$$y_{jk}(t) = p_{jk} z_{jk}(t) \left[\sum_{m=1}^{n} p_{jm} z_{jm}(t)\right]^{-1}, \tag{5}$$

$$\dot{z}_{jk}(t) = -u z_{jk}(t) + \beta p_{jk} x_j(t-\tau) x_k(t). \tag{6}$$

Both equations (4)–(6) and (1) and (2) can be described as cross-correlated flows on networks in a manner that has been previously described in this journal.[4, 5] *Bounded* versions of both (4)–(6) and (1) and (2) can readily be given.

(4) *Lateral Inhibition and Thresholds.*—Equations (4)–(6) improve the learning of (1) and (2) formally, but introduce a conceptual difficulty; namely, by (5), the value $z_{jm}(t)$ at the arrowhead N_{jm} of e_{jm} instantaneously jumps to the arrowhead N_{jk} where $y_{jk}(t)$ is computed. This "virtual" interaction must be replaced by a finite-rate and local interaction with the same qualitative properties. Since $y_{jm}(t) \geq 0$ and $\sum_{m=1}^{n} y_{jm}(t) = 0$ or 1, the mapping from $p_{jk}z_{jk}(t)$ to $y_{jk}(t)$ by which (4) replaces (1) describes an *inhibition* between the associations $y_{jm}(t)$, $m = 1, 2, \ldots, n$. The finite-rate analogue of this "virtual" inhibition requires the introduction of lateral inhibitory interactions and thresholds.[2] The finite-rate analogue, in the unbounded case, is given by

$$\dot{x}_i(t) = \alpha_i^{+}[P_i - x_i(t)]^{+} \quad \alpha_i^{-}[x_i(t) \quad P_i]^{+} + J_i^{+}(t) \quad J_i^{-}(t) + I_i^{+}(t) \quad I_i^{-}(t) \tag{7}$$

and

$$\dot{z}_{jk}(t) \quad u_{jk}^{+}[Q_{jk} \quad z_{jk}(t)]^{+} - u_{jk}^{-}[z_{jk}(t) - Q_{jk}]^{+} + \beta_j^{+}\gamma_{jk}^{+}p_{jk}^{+}[x_j(t-\tau_{jk}^{+}) - \Gamma_{jk}^{+}]^{+}[x_k(t) \quad \Lambda_k^{+}]^{+}, \tag{8}$$

where

$$J_i^{+}(t) = \sum_{m=1}^{n} \beta_m^{+}[x_m(t-\tau_{mi}^{+}) - \Gamma_{mi}^{+}]^{+} p_{mi}^{+}[z_{mi}(t) - \Omega_{mi}^{+}]^{+}, \tag{9}$$

$$J_i^{-}(t) = \sum_{m=1}^{n} \beta_m^{-}[x_m(t-\tau_{mi}^{-}) - \Gamma_{mi}^{-}]^{+} p_{mi}^{-}, \tag{10}$$

$I_i^{+}(t)$ and $I_i^{-}(t)$ are known excitatory and inhibitory inputs, respectively, and the notation $[\omega]^{+}$ denotes

$$[\omega]^{+} = \max(\omega, 0),$$

whereby various thresholds are described. P_i is the equilibrium value (or "potential") of $x_i(t)$, and Q_{jk} is the equilibrium value of $z_{jk}(t)$. The "spiking threshold" Γ_{ij} and the equilibrium value P_i satisfy $\Gamma_{ij} > P_i$. Similarly, $\Omega_{ij}^{+} \geq$

Q_{ij} and $\Lambda_j^+ \geq P_j$. Equations (7)–(10) can be given a neural interpretation which is substantially more quantitative than that of (1) and (2).[2] For example, consider (7)–(10) under steady-state excitatory inputs and let all interactions be inhibitory. Then (7) reduces in the steady state to the Hartline-Ratliff equation

$$r_i = e_i - \sum_{j=1}^{n} K_{ij}[r_j - r_{ij}^0]^+$$

for lateral inhibition in the *Limulus* retina if

$$K_{ij} = \frac{\mu_i^+ \beta_j^-}{\mu_j^+ \alpha_i^-} p_{ji}^-$$

and

$$r_{ij}^0 \quad \mu_j^+(\Gamma_{ji}^- - \Gamma_j^+),$$

when $\mu_i^+[x_i(t) - \Gamma_i^+]^+$ is the output from the retina to higher neural centers.

(5) *Symmetry-Breaking by Na^+ and K^+.*—The bounded analogue of (7) is

$$\dot{x}_i(t) = \alpha_i^+(M_i - x_i(t))(\gamma_i^+ + J_i^+(t) + I_i^+(t)) - \alpha_i^-(x_i(t) - m_i)(\gamma_i^- + J_i^-(t) + I_i^-(t)), \quad (12)$$

where $m_i \leq x_i(t) \leq M_i$ for all $t \geq 0$. There exists an obvious symmetry between the excitatory and inhibitory terms in (12). This symmetry can be made explicit by replacing (12) with equations for a *pair* of variables $x_i^+(t)$ and $x_i^-(t)$ which are positively and negatively "polarized," respectively. This symmetrization procedure must not, however, destroy the "excitatory bias" within (7)–(10) that makes learning possible. The result is, in first approximation,

$$\dot{x}_i^+(t) = \alpha_i^{++}(M_i^+ - x_i^+(t))(\gamma_i^{++} + J_i^{++}(t) + I_i^{++}(t)) - \alpha_i^{+-}\gamma_i^{+-}(x_i^+(t) - m_i^+), \quad (13)$$

$$\dot{x}_i^-(t) = \alpha_i^{-+}\gamma_i^{-+}(M_i^- - x_i^-(t)) - \alpha_i^{--}(x_i^-(t) - m_i^-)(\gamma_i^{--} + J_i^{--}(t) + I_i^{--}(t)),$$

and

$$\chi([x_m^+(t) \quad \Gamma_{mi}^{+-}]^+)(\beta_m^{+-}[x_m^+(t) \quad \Gamma_{mi}^{+-}]^+ - \beta_m^{--}[\Gamma_{mi}^{-+} \quad x_m^-(t)]^+) = 0,$$

where

$$J_i^{++}(t) \quad \sum_{m=1}^{n} \beta_m^{++}[x_m^+(t \quad \tau_{mi}^{++}) \quad \Gamma_{mi}^{++}]^+ p_{mi}^{++}[z_{mi}^{++}(t) - \Omega_{mi}^{++}]^+$$

$$J_i^{--}(t) \quad \sum_{m=1}^{n} \beta_m^{+-}[x_m^+(t \quad \tau_{mi}^{+-}) \quad \Gamma_{mi}^{+-}]^+ p_{mi}^{--}[z_{mi}^{--}(t) \quad \Omega_{mi}^{--}]^+,$$

and

$$\chi(\omega) \quad \begin{cases} 1, & \omega > 0 \\ 0, & \omega \leq 0. \end{cases}$$

Equations (13)–(17) can be interpreted to yield the "symmetry-breaking" properties (*o*) and (*p*) of section (2). The condition (15) is merely of qualitative interest, and will be made more quantitive in a later paper, along a pathway that is suggested in reference 2.

(6) *Transmitter Production and Release.*—In first approximation, the bounded equation for excitatory transmitter production $z_{ij}^{++}(t)$ in the collection of synaptic knobs N_{ij}^{++} is

$$\dot{z}_{ij}^{++}(t) = (M_{ij}^{++} - z_{ij}^{++}(t))(u_{ij}^{+++} + \gamma_{ij}^{++}F_{ij}^{++}(t)R_j(t)) - u_{ij}^{++-}(z_{ij}^{++}(t) - m_{ij}^{++}), \quad (18)$$

where

$$F_{ij}^{++}(t) = \beta_i^{++}[x_i^{+}(t - \tau_{ij}^{++}) - \Gamma_{ij}^{++}]^{+}p_{ij}^{++} \quad (19)$$

$$R_j(t) = [x_j(t) - \Lambda_j^{+}]^{+} \quad (20)$$

The inequalities

$$m_{ij}^{++} \leq z_{ij}^{++}(t) \leq M_{ij}^{++}$$

hold for all $t \geq 0$. All learning within (18) is due to the term

$$\gamma_{ij}^{++}(M_{ij}^{++} - z_{ij}^{++}(t))F_{ij}^{++}(t)R_j(t). \quad (21)$$

The spiking frequency term $F_{ij}^{++}(t)$ is interpreted as an antagonistic coupling between Na^{+} and K^{+} at suprathreshold values, whereas $R_j(t)$ is interpreted as an antagonistic coupling between Ca^{++} and Mg^{++}. Na^{+} and Ca^{++} act synergistically in (21) to activate $z_{ij}^{++}(t)$.

It is readily seen that the coupling between $F_{ij}^{++}(t)$ and

$$G_{ij}^{++}(t) = [z_{ij}^{++}(t) - \Omega_{ij}^{++}]^{+}$$

in (16) describes a transmitter release process in which the depleted transmitter is instantaneously replenished. The finite-rate analogue of this coupling is given by the pair of equations

$$\dot{Z}_{ij}^{++}(t) = \lambda_{ij}^{+}(\delta_{ij}z_{ij}^{++}(t) - Z_{ij}^{++}(t)) - \lambda_{ij}^{-}F_{ij}^{++}(t)[\tilde{Z}_{ij}^{++}(t) - U_{ij}^{++}]^{+} \quad (22)$$

and

$$\dot{\tilde{Z}}_{ij}^{++}(t) = \omega_{ij}^{+}(Z_{ij}^{++}(t) - \tilde{Z}_{ij}^{++}(t)) - \lambda_{ij}^{-}F_{ij}^{++}(t)[\tilde{Z}_{ij}^{++}(t) - U_{ij}^{++}]^{+} - \omega_{ij}^{-}[\tilde{Z}_{ij}^{++}(t) - V_{ij}^{++}]^{+}, \quad (23)$$

with

$$U_{ij}^{++} = \delta_{ij}\Omega_{ij}^{++} > V_{ij}^{++}$$

and

$$0 \leq \tilde{Z}_{ij}^{++}(t) \leq Z_{ij}^{++}(t).$$

$^{+}(t)$ = the total amount of excitatory transmitter in the synaptic knobs N_{ij}^{++} at time t,

$\tilde{Z}_{ij}^{++}(t)$ = the total amount of *mobilized* transmitter at time t,
$z_{ij}^{++}(t)$ = the total number of *active* transmitter-producing sites at time t.

A simple physical interpretation of (22) and (23) yields properties (h), (i), (j), (k), and (q) of section (2). Equations (22) and (23) can be solved explicitly for the transient responses of $Z_{ij}^{++}(t)$ and $\tilde{Z}_{ij}^{++}(t)$ when (say) $F_{ij}^{++}(t)$ is a steady-state spiking frequency F for $t \geq 0$, $\lambda_{ij}^{+} = \omega_{ij}^{-}$, and $U_{ij}^{++} = V_{ij}^{++} = 0$. Then, ignoring slow variations of $z_{ij}^{++}(t)$,

$$\tilde{Z}_{ij}^{++}(t) = \frac{\delta_{ij} z_{ij}^{++}(0)\omega_{ij}^{+}}{\omega_{ij}^{-} + \omega_{ij}^{+}} \left[\exp\left(-(\lambda_{ij}^{+} + \lambda_{ij}^{-} F)t \right) + \frac{\lambda_{ij}^{+}}{\lambda_{ij}^{+} + \lambda_{ij}^{-} F}\left(1 - \exp\left(-(\lambda_{ij}^{+} + \lambda_{ij}^{-} F)t\right)\right) \right], \quad (24)$$

and the amount of mobilized transmitter which is *released* from N_{ij}^{++} at time t is $\lambda_{ij}^{-} F \tilde{Z}_{ij}^{++}(t)$, as (22) and (23) show.

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