Brain Research, 69 (1974) 115–124

ARITHMETICAL OPERATIONS PERFORMED BY NERVE CELLS

STEPHEN BLOMFIELD

Division of Cell Biology, Laboratory of Molecular Biology, Cambridge (Great Britain)

(Accepted September 26th, 1973)

SUMMARY

A simple neurone model is constructed and analysed mathematically to see what types of operation it can perform on its synaptic inputs. The neurone consists essentially of a soma together with semi-infinite dendrite. Provided their conductance changes are sufficiently small, excitatory synapses are linearly additive and inhibitory synapses are linearly subtractive, irrespective of location. Inhibitory synapses, producing large conductance changes and located preferably on the soma, are ideally suited to carry out division. The extension of this model to the more complex situation of branching dendritic trees is briefly examined and its relevance to real nerve cells is discussed.

Background

Several recent nerve-net models[1,2,9,10] require certain properties of nerve cells. In particular, they have to carry out the arithmetical operations of addition and subtraction and, in some cases, of division. The purpose of this paper is to examine what classes of arithmetical operations may be performed by a simple, idealized type of nerve cell.

The model

The nerve cell is taken to be a uniform, leaky cable extending to infinity in the positive direction and stopped off by a resistance R_0 at the origin. The cable has longitudinal resistance R_1/unit length and membrane resistance $R_m \times$ unit length. Its membrane potential is zero in the resting state. The firing zone of the cell membrane is situated at the origin. A synaptic input consists of an e.m.f. V_s applied across the membrane at the point $x = x_s$ in series with a resistance R_s ($s \epsilon \Sigma$, the set of synaptic inputs). We suppose that there are just two inputs, $<V_I,x_I,R_I>$ and $<V_E,x_E,R_E>$. This is essentially the synaptic model of Eccles[6] applied to a soma-dendritic cable. The firing rate of the cell is taken to be a function of the potential V_0 at the firing

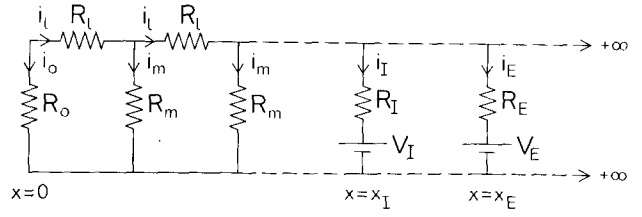

Fig. 1. Circuit diagram of the hypothetical nerve cell discussed in the text. The cell soma is at the origin. There are two synaptic inputs on the dendrite: an excitatory one at x_E and an inhibitory one at x_I. Synapses on the soma may alter the impedance R_0. The cell firing rate is a function solely of the potential at the origin.

zone, $x = 0$. The inputs are considered to be long-lasting, so that transient effects — due to capacitance and inductance — may be ignored.

Solution of the model

Let i_l be the longitudinal current, i_m the membrane current density, i_I and i_E the synaptic currents at $x = x_I$ and $x = x_E$ respectively and i_0 the leak current at the origin. Then the following differential equations and boundary conditions must be satisfied:

DEs

$$i_l = -\frac{1}{R_l}\frac{\partial V}{\partial x} \tag{1}$$

$$i_m = -\frac{\partial i_l}{\partial x} \tag{2}$$

$$V = i_m R_m \tag{3}$$

BCs

$$V \to 0 \text{ as } x \to +\infty \tag{4}$$

$$V = i_0 R_0 \text{ at } x = 0. \quad i.e.\ V_0 = i_0 R_0 \tag{5}$$

$$V \text{ is continuous at } x = x_I \text{ and at } x = x_E \tag{6) (7}$$

$$i_l = -i_0 \text{ at } x = 0 \tag{8}$$

$$[i_l]_{x_I^-}^{x_I^+} = -i_I = \left(\frac{V_I - V}{R_I}\right)_{x = x_I} \tag{9}$$

$$[i_l]_{x_E^-}^{x_E^+} = -i_E = \left(\frac{V_E - V}{R_E}\right)_{x = x_E} \tag{10}$$

For $x_I \leqslant x_E$, the solution of these equations gives

$$\frac{V_0}{2} = \frac{\{1+(1-\varrho)G_E\}G_I V_I \alpha + G_E V_E \beta}{(1+\mu_0) + [(1+\mu_0) + (1-\mu_0)\alpha^2]\{1+(1-\varrho)G_E\}G_I + [(1+\mu_0) + (1-\mu_0)\beta^2]G_E}$$

where $\mu_0 = \lambda R_l / R_0$

$G_I = \lambda R_l / 2R_I$

$G_E = \lambda R_l / 2R_E$

$\alpha = e^{-x_I/\lambda}$

$\beta = e^{-x_E/\lambda}$

$\varrho = \beta^2/\alpha^2$

and $\lambda = \sqrt{R_m/R_1}$ is the space constant of the cable.

(See Appendix for the derivation of this formula.)

Significance of the constants

V_I and V_E are the inhibitory and excitatory driving potentials. α and β give the proportional decrements of V_I and V_E respectively that are due simply to the leaky cable properties of the soma-dendritic membrane. ϱ has a rather complex effect and provides a measure of the relative remoteness of the two synaptic inputs. μ_0 is the normalized soma conductance: it is the ratio of the actual soma conductance to the membrane conductance across one space constant. Likewise G_I and G_E are normalized synaptic conductances. G_I and G_E will depend on the number and/or firing rates of the inhibitory and excitatory inputs. If the synaptic conductance attributable to every inhibitory input is constant (and similarly for every excitatory input), then

$G_I \propto n_I$, the number of active inhibitory synapses

$G_E \propto n_E$, the number of active excitatory synapses.

Behaviour of the solution under certain conditions

There are four cases of special interest: (i) when the excitatory and inhibitory synapses are both located on the soma; (ii) when the excitatory and inhibitory synapses are situated close together on a remote portion of the dendrite; (iii) when the inhibitory synapse is located on the soma whilst the excitatory synapse is remote; (iv) when the inhibitory and excitatory synapses are situated on widely separated portions of the remote dendrite.

(*i*) $\alpha = \beta = 1$, $\varrho = 1$.

The expression for V_0 becomes $\dfrac{G_I V_I + G_E V_E}{\frac{1}{2}(1 + \mu_0) + G_I + G_E}$

If $G_I + G_E \gg 1 + \mu_0$, this approximates to $\dfrac{V_I + \dfrac{G_E}{G_I} V_E}{1 + \dfrac{G_E}{G_I}}$

I.e. V_0 is a function of G_E/G_I and hence the firing rate of the cell is determined by the *direct ratio* of active excitatory and inhibitory synapses.

If $G_I + G_E \ll 1 + \mu_0$, this approximates to $\dfrac{G_I V_I + G_E V_E}{\frac{1}{2}(1 + \mu_0)}$

I.e. V_0 is a linear function of G_E and G_I and hence the cell firing rate is determined by a *linear combination* of active excitatory and inhibitory synapses.

(*ii*) $\alpha = \beta$, $\alpha \ll 1$, $\varrho = 1$.

The expression for V_0 becomes $\dfrac{\alpha}{\frac{1}{2}(1 + \mu_0)} \cdot \dfrac{G_I V_I + G_E V_E}{1 + G_I + G_E}$

If $G_I + G_E \gg 1$, this approximates to $\dfrac{\alpha}{\frac{1}{2}(1+\mu_0)} \cdot \dfrac{V_I + \dfrac{G_E}{G_I} V_E}{1 + \dfrac{G_E}{G_I}}$

If $G_I + G_E \ll 1$, this approximates to $\dfrac{G_I V_I \alpha + G_E V_E \alpha}{\frac{1}{2}(1+\mu_0)}$

In the former case the cell firing rate is determined by the ratio G_E/G_I, whereas in the latter case it is determined by a linear combination of G_E and G_I.

(*iii*) $\alpha = 1, \quad \beta \ll 1, \quad \varrho \doteqdot 0.$

The expression for V_0 becomes $\dfrac{G_I V_I + \dfrac{G_E V_E}{1+G_E}\beta}{\frac{1}{2}(1+\mu_0) + G_I}$

If $G_E \gg 1$, the solution is uninteresting since it is independent of G_E.

If $G_E \ll 1$, then V_0 approximates to

$V_I + \dfrac{G_E}{G_I} V_E \beta \quad$ for $G_I \gg 1 + \mu_0$

$\dfrac{G_I V_I + G_E V_E \beta}{\frac{1}{2}(1+\mu_0)} \quad$ for $G_I \ll 1 + \mu_0$

In the former case the cell firing rate is a function of the ratio G_E/G_I, and in the latter case it is a function of their linear combination.

(*iv*) $\alpha \ll 1, \quad \beta \ll 1, \quad \varrho \doteqdot 0.$

The expression for V_0 becomes $\dfrac{1}{\frac{1}{2}(1+\mu_0)} \cdot \dfrac{G_I V_I \alpha + \dfrac{G_E V_E}{1+G_E}\beta}{1 + G_I}$

If $G_E \gg 1$, the solution is uninteresting since it is independent of G_E.
If $G_E \ll 1$, then V_0 approximates to

$\dfrac{V_I \alpha + \dfrac{G_E}{G_I} V_E \beta}{\frac{1}{2}(1+\mu_0)} \quad$ for $G_I \gg 1$

$\dfrac{G_I V_I \alpha + G_E V_E \beta}{\frac{1}{2}(1+\mu_0)} \quad$ for $G_I \ll 1$

I.e. the firing rate is dependent on the ratio G_E/G_I in the former case and on the linear combination of G_E and G_I in the latter case.

Implications of the model

Under certain conditions, *i.e.* G_I and G_E sufficiently small, the cell firing rate

$f(V_0)$ can signal their linear combination. For $f(V_0)$ to give a sensitive measure under these circumstances, αV_I and βV_E — the synaptic driving potentials as seen at the firing zone — must be of sufficiently large amplitude. V_E is known to be close to the short-circuited potential of the cell[5] and so βV_E will be fairly large — unless the synapses are too remote (which is unlikely even for the smallest dendritic branches). Addition is thus a feasible operation. However V_I is considerably smaller[4]: its magnitude appears to be only 1/5–1/10 that of V_E. In some cells it may even be zero.

Subtraction can only be performed to a significant extent if V_I is of sufficient size. In any case, V_I will be small compared to V_E, and so — to have comparable effects — either the inhibitory conductance G_I must be correspondingly greater than the excitatory conductance G_E or the inhibitory synapses must be located proximal to the excitatory synapses — thus making α large compared to β. Another possibility is that the dendrites are always depolarized relative to the soma: the shift in membrane potential would cause a relatively large increase in V_I and a relatively small decrease in V_E.

In other conditions (*i.e.* G_I or G_E sufficiently large in the case $\varrho = 1$, G_I sufficiently large and G_E sufficiently small in the case $\varrho \doteq 0$), the cell firing rate gives a measure of the ratio G_E/G_I. This will be a sensitive measure provided G_E/G_I is not too small compared to $1/\beta V_E$.

Extensions to the model

In the above analysis, μ_0 has been taken to be constant. However, μ_0 may be varied if there are synapses on the soma that cause a reduction in membrane impedance together with zero driving potential. Such synapses will reduce the size of the potential changes produced at the firing zone.

A combination of linear and divisor operations is thus feasible under the following conditions:

(i) $f(V_0)$ is a fairly sensitive function of V_0;

(ii) additive excitatory synapses have relatively low conductivity and large positive driving potential;

(iii) subtractive inhibitory synapses have relatively low conductivity and large negative driving potential;

(iv) divisor inhibitory synapses, sited preferably on or near the soma, have relatively high conductivity and zero driving potential.

Whenever the potential V_0 is behaving linearly as a function of G_E and G_I, the effect of the soma conductance μ_0 is to reduce V_0 by the factor

$$\frac{1}{1 + \mu_0}.$$

Now μ_0 consists of two parts: a fixed 'resting' component, μ_r say, and a variable 'active' component, μ_a say, due to the divisor synapses.

$$\mu_0 = \mu_r + \mu_a$$

$$\therefore V_0 \propto \frac{1}{(1 + \mu_r) + \mu_a}$$

If division is to be proportional to the fraction p of active input fibres, then the divisor cells have to be driven so that

$$1 + \mu_r + \mu_a = kp, \text{ where k is constant}$$
$$i.e.\ \mu_a = kp - l, \text{ where } l = 1 + \mu_r.$$

Hence the total divisor cell activity should be linearly proportional to the input fibre activity. This can only work, however, in the range $p \geqslant l/k$, since μ_a cannot be negative. That is, division can only take place if the level of input fibre activity is sufficiently high.

Relevance to real nerve cells

The model has a highly idealized form. Real nerve cells have several dendritic processes, which branch repeatedly and gradually taper. The branching will increase the effective shunting conductances between a dendritic input and the soma, whilst the tapering will increase the remoteness of the input. However, the same qualitative results will hold. Thus linear behaviour will be closely approximated provided the normalized conductances remain small: and division will still be performed by the appropriate somatic synapses. To some extent, each main dendritic tree may function independently of the other dendritic trees, since the membranes and synapses of different dendritic trees are relatively remote from one another: this is particularly true if the cell soma contributes a large proportion of the total membrane area and if the dendrites have narrow diameters. In this case, the dendritic synapses may act in a linear fashion, providing a large e.m.f. in series with a high impedance (and thus behaving as constant current sources) whilst the somatic synapses perform the operation of division by shunting the synaptic currents through low impedance pathways. Even if one relaxes the constraint of low synaptic conductances in the

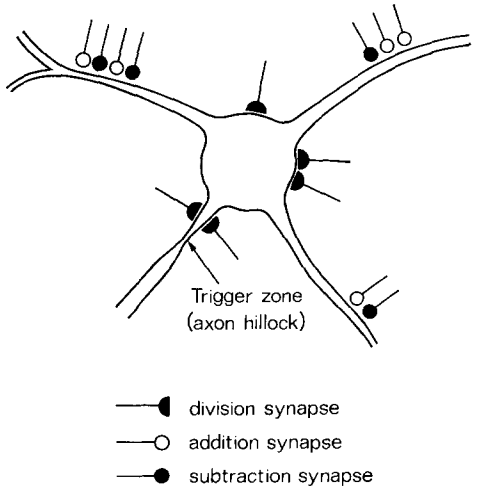

Fig. 2. Suggested distribution of the different classes of synapse on a nerve cell. Linearly interacting synapses, which behave as constant current sources, are located on the dendrites. Division synapses, which act as low impedance shunts, are located on the soma.

dendrites, so that individual dendritic trees no longer perform linear computations, it remains possible for the effects of separate dendritic trees to be combined linearly and for division to be performed by the somatic synapses.

Some nerve cells — *e.g.* spinal motoneurones, cells of Clarke's column, Purkinje cells of cerebellum — could fit into this pattern. Others cannot unless some modification is made to the theory. Neurones that have dendrites which generate spikes, *e.g.* hippocampal pyramidals[13] and lamina 4 cells of the spinal cord[14], cannot be accommodated within this theory unless one supposes there to be trigger zones — behaving as postulated in this model — sited in the convergent regions of the dendritic trees. The synapses on the main dendritic shafts would then have some further special action, *e.g.* signalling when to modify[15].

There is plenty of evidence for the linear addition of excitatory synaptic potentials[3,6,11] and for the division of excitatory synaptic potentials by inhibitory synaptic potentials[6,11,12]. Evidence for linear subtraction by inhibitory synaptic potentials is less common but nevertheless exists[11]. The postulated firing mode of the nerve cell receives support from experiments on the intracellular injection of constant currents into neurones[7,8]. In effect, a steady current corresponds to a constant potential at the trigger zone.

APPENDIX

Derivation of the solution for the case $x_I \leq x_E$

We start with the differential equations (1) to (3) and the boundary conditions (4) to (10).

From (1) and (2), $$i_m = \frac{1}{R_l} \frac{\partial^2 V}{\partial x^2} \tag{11}$$

From (11) and (3), $$V = \frac{R_m}{R_l} \frac{\partial^2 V}{\partial x^2} = \lambda^2 \frac{\partial^2 V}{\partial x^2} \tag{12}$$

where $$\lambda = \sqrt{\frac{R_m}{R_l}}$$

The general solution of (12) is

$$V = A e^{x/\lambda} + B e^{-x/\lambda} \tag{13}$$

Using (1), we get $$i_l = \frac{1}{\lambda R_l} (-A e^{x/\lambda} + B e^{-x/\lambda}) \tag{14}$$

Let the particular solution be

$$V = \begin{cases} A_0 e^{x/\lambda} + B_0 e^{-x/\lambda} & x \leqslant x_I \\ A_1 e^{x/\lambda} + B_1 e^{-x/\lambda} & x_I \leqslant x \leqslant x_E \\ A_2 e^{x/\lambda} + B_2 e^{-x/\lambda} & x_E \leqslant x \end{cases}$$

with corresponding equations for i_1.
Substituting for i_1 in (8), we obtain

$$\frac{1}{\lambda R_1}(-A_0 + B_0) = -i_0$$

$$i.e. \quad i_0 = \frac{1}{\lambda R_1}(A_0 - B_0) \tag{15}$$

Now $V_0 = (V)_{x=0} = A_0 + B_0$

Thus, using (5) and (15), we have

$$A_0 + B_0 = \frac{R_0}{\lambda R_1}(A_0 - B_0)$$

Putting $\frac{\lambda R_1}{R_0} = \mu_0$, we obtain

$$(1 - \mu_0) A_0 - (1 + \mu_0) B_0 = 0 \tag{16}$$

$$\therefore \quad B_0 = \frac{1 - \mu_0}{1 + \mu_0} A_0 \tag{17}$$

$$\text{and} \quad V_0 = \frac{2}{1 + \mu_0} A_0 \tag{18}$$

Condition (4) entails immediately that $A_2 = 0$ (19)

Condition (6) gives

$$A_0 e^{x_1/\lambda} + B_0 e^{-x_1/\lambda} = A_1 e^{x_1/\lambda} + B_1 e^{-x_1/\lambda}$$

Putting $\alpha = e^{-x_1/\lambda}$, we obtain

$$A_0 + B_0\alpha^2 - A_1 - B_1\alpha^2 = 0 \tag{20}$$

Likewise condition (7), together with (19), leads to

$$A_1 + B_1\beta^2 - B_2\beta^2 = 0 \tag{21}$$

where $\beta = e^{-x_E/\lambda}$.

Condition (9) gives

$$\frac{1}{\lambda R_1}(-A_1/\alpha + B_1\alpha) - \frac{1}{\lambda R_1}(-A_0/\alpha + B_0\alpha) = \frac{V_1}{R_1} - \frac{1}{R_1}(A_1/\alpha + B_1\alpha)$$

Multiplying through by $\alpha\lambda R_1$ and collecting terms,

$$A_0 - B_0\alpha^2 + \left(\frac{\lambda R_1}{R_1} - 1\right) A_1 + \left(\frac{\lambda R_1}{R_1} + 1\right) B_1\alpha^2 = \frac{\lambda R_1}{R_1}\alpha V_1$$

Putting $G_I = \frac{\lambda R_1}{2R_I}$, we obtain

$$A_0 - B_0\alpha^2 + (2G_I - 1)A_1 + (2G_I + 1)B_1\alpha^2 = 2G_IV_I\alpha \tag{22}$$

Similarly condition (10), together with (19), leads to

$$A_1 - B_1\beta^2 + (2G_E + 1)B_2\beta^2 = 2G_EV_E\beta \tag{23}$$

Adding (20) and (22),

$$\begin{aligned} &2A_0 + (2G_I - 2)A_1 + (2G_I)B_1\alpha^2 = 2\ G_IV_I\alpha \\ \textit{i.e.}\quad &A_0 - (1 - G_I)\,A_1 + G_I\alpha^2B_1 = G_IV_I\alpha \end{aligned} \tag{24}$$

Multiplying (21) by $(2G_E + 1)$ and adding to (23),

$$\{(2G_E + 1) + 1\}\,A_1 + \{(2G_E + 1) - 1\}\,B_1\beta^2 = 2G_EV_E\beta$$

$$\textit{i.e.}\quad (1 + G_E)\,A_1 + G_E\beta^2\,B_1 = G_EV_E\beta \tag{25}$$

Substituting (17) into (20) gives

$$A_0\left\{1 + \alpha^2\left(\frac{1 - \mu_0}{1 + \mu_0}\right)\right\} - A_1 - B_1\alpha^2 = 0$$

$$\therefore\ \{(1 + \mu_0) + (1 - \mu_0)\alpha^2\}\,A_0 - (1 + \mu_0)\,A_1 - (1 + \mu_0)\,B_1\alpha^2 = 0 \tag{26}$$

Now (24), (25) and (26) are three simultaneous equations in the three unknowns A_0, A_1 and A_2.

The standard solution for simultaneous equations of the form

$$\begin{aligned} l_1x + m_1y + n_1z &= p_1 \\ l_2x + m_2y + n_2z &= p_2 \\ l_3x + m_3y + n_3z &= p_3 \end{aligned}$$

is $x = \dfrac{(m_3n_2 - m_2n_3)p_1 + (m_1n_3 - m_3n_1)p_2 + (m_2n_1 - m_1n_2)p_3}{(m_3n_2 - m_2n_3)l_1 + (m_1n_3 - m_3n_1)l_2 + (m_2n_1 - m_1n_2)l_3}$

etc.

Thus, substituting for the dummy constants above, we have

$$A_0 = \frac{\{-(1+\mu_0)G_E\beta^2 + (1+G_E)(1+\mu_0)\alpha^2\}G_IV_I\alpha + \{(1-G_I)(1+\mu_0)\alpha^2 + (1+\mu_0)G_I\alpha^2\}G_EV_E\beta}{\{-(1+\mu_0)G_E\beta^2 + (1+G_E)(1+\mu_0)\alpha^2\} + \{(1-G_I)G_E\beta^2 + (1+G_E)G_I\alpha^2\}\{(1+\mu_0) + (1-\mu_0)\alpha^2\}} \tag{27}$$

The numerator of (27) simplifies to

$$(1+\mu_0)\alpha^2\left[\left\{-\frac{\beta^2}{\alpha^2}G_E + (1+G_E)\right\}G_IV_I\alpha + \{(1-G_I) + G_I\}G_EV_E\beta\right]$$

$$\textit{i.e.}\quad (1 + \mu_0)\alpha^2\,[\{1 + (1 - \varrho)G_E\}G_IV_I\alpha + G_EV_E\beta] \tag{28}$$

where $\varrho = \beta^2/\alpha^2$

The denominator of (27) can be rewritten as

$$(1+G_E)(1+\mu_0)\alpha^2 - (1+\mu_0)G_E\beta^2 + \{(1+\mu_0) + (1-\mu_0)\alpha^2\}\{G_E\beta^2 + (\alpha^2+G_E\alpha^2-G_E\beta^2)G_I\}$$

i.e.

$$[(1+\mu_0)\alpha^2 + (1+\mu_0)\alpha^2 G_E] + (1-\mu_0)G_E\beta^2\alpha^2 + \{(1+\mu_0) + (1-\mu_0)\alpha^2\}\{\alpha^2+(\alpha^2-\beta^2)G_E\}G_I$$

Taking the common factor α^2 outside and rearranging the terms, we get

$$\alpha^2[(1+\mu_0) + \{(1+\mu_0) + (1-\mu_0)\alpha^2\}\{1 + (1-\varrho)G_E\}G_I + \{(1+\mu_0) + (1-\mu_0)\beta^2\}G_E] \tag{29}$$

Hence, using (18), (28) and (29), we finally obtain

$$\frac{V_0}{2} = \frac{A_0}{1+\mu_0} = \frac{\{1 + (1-\varrho)G_E\}G_I V_I\alpha + G_E V_E\beta}{(1+\mu_0) + [(1+\mu_0)+(1-\mu_0)\alpha^2]\{1+(1-\varrho)G_E\}G_I + [(1+\mu_0)+(1-\mu_0)\beta^2]G_E} \tag{30}$$

REFERENCES

1 BRINDLEY, G. S., The classification of modifiable synapses and their use in models for conditioning, *Proc. roy. Soc. B*, 168 (1967) 361–376.
2 BRINDLEY, G. S., Nerve net models of plausible size that perform many simple learning tasks, *Proc. roy. Soc. B*, 174 (1969) 173–191.
3 BURKE, R. E., Composite nature of the monosynaptic excitatory postsynaptic potential, *J. Neurophysiol.*, 30 (1967) 1114–1137.
4 COOMBS, J. S., ECCLES, J. C., AND FATT, P., The specific ionic conductances and the ionic movements across the motoneuronal membrane that produce the inhibitory postsynaptic potential, *J. Physiol. (Lond.)*, 130 (1955) 326–373.
5 COOMBS, J. S., ECCLES, J. C., AND FATT, P., Excitatory synaptic action in motoneurones, *J. Physiol. (Lond.)*, 130 (1955) 374–395.
6 ECCLES, J. C., *The Physiology of Synapses*, Springer, Berlin, 1964, Ch. IV, VII and X.
7 GRANIT, R., KERNELL, D., AND LAMARRE, Y., Algebraic summation in synaptic activation of motoneurones firing within the 'primary range' to injected currents, *J. Physiol. (Lond.)*, 187 (1966) 379–399.
8 GRANIT, R., KERNELL, D., AND SHORTESS, G. K., Quantitative aspects of repetitive firing of mammalian motoneurones caused by injected currents, *J. Physiol. (Lond.)*, 168 (1963) 911–931.
9 MARR, D., A theory for cerebral neocortex, *Proc. roy. Soc. B*, 176 (1970) 161–234.
10 MARR, D., Simple memory: a theory for archicortex, *Phil. Trans. B*, 262 (1971) 23–81.
11 RALL, W., BURKE, R. E., SMITH, T. G., NELSON, P. G., AND FRANK, K., Dendritic location of synapses and possible mechanisms for the monosynaptic EPSP in motoneurones, *J. Neurophysiol.*, 30 (1967) 1169–1193.
12 SMITH, T. G., WUERKER, R. B., AND FRANK, K. J., Membrane impedance changes during synaptic transmission in cat spinal motoneurons, *J. Neurophysiol.*, 30 (1967) 1072–1096.
13 SPENCER, W. A., AND KANDEL, E. R., Electrophysiology of hippocampal neurons. IV. Fast prepotentials, *J. Neurophysiol.*, 24 (1961) 272–285.
14 WALL, P. D., Impulses originating in the region of dendrites, *J. Physiol. (Lond.)*, 180 (1965) 116–133.
15 MARR, D., A theory of cerebellar cortex, *J. Physiol. (Lond.)*, 202 (1969) 437–470.