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THE PASSIVE ELECTRICAL PROPERTIES OF THE MEMBRANE OF A MOLLUSCAN NEURONE

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SUMMARY

1. The passive electrical properties of the membrane of the gastro-oesophageal giant neurone (G cell) of the marine mollusc, *Anisodoris nobilis* were studied with small current steps.

2. The membrane transient response can be fitted with a theoretical curve assuming as a model for the cell a sphere (soma) connected to a cable (axon). The axo-somatic conductance ratio (ρ), determined by applying this model, is large (approximately 5) and the membrane time constant (τ) is long (approximately 1 sec).

3. When the actual surface area of the cell, corrected for surface infoldings, and the spread of current along its axon is taken into account, the electrical measurements imply a specific resistance of the membrane of approximately 1·0 $M\Omega.cm^2$.

4. Estimates of specific membrane capacity, either from measurements of the initial portion of the membrane transient or from the ratio of the time constant to the specific membrane resistance are close to the value of 1 $\mu F/cm^2$ expected for biological membranes.

5. Thus, our measurements of specific capacitance, time constant, length constant and axo-somatic conductance ratio all indicate that the value found for the specific membrane resistance of the G cell, while unexpectedly large, is valid.

6. The magnitude of this value suggests that the conductance (permeability) of its membrane to ions is much smaller than that previously assumed for nerve membranes; this small conductance may be related to the larger surface-to-volume ratio of the G cell.

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INTRODUCTION

Values for the specific membrane resistance and capacitance have been determined for a number of neurones (Cole, 1968). Molluscan nerve cells are unusual in that the reported values for the specific capacitance of their membranes, ranging from 5 to 60 μF/cm^2 (Fessard & Tauc, 1956; Maiskii, 1963; Murray, 1966; Hughes, 1967; Meves, 1968), are much larger than the value of approximately 1 μF/cm^2 found for most other cells (Cole, 1968). In contrast, the reported values for specific membrane resistance are in the range of those given for other preparations. These results, however, were obtained without accounting for the complex morphology of the molluscan neurone. The giant cell (G cell) of the gastro-oesophageal nervous system of the mollusc, *Anisodoris nobilis* (MacFarland), provides a convenient model for studying this problem. Mirolli & Talbott (1972) have given a set of estimates for the surface area of the soma and for the geometrical factor which determines the axonal conductance of the G cell. In the present paper, these results have been used in combination with electrical measurements to compute a set of values for the specific resistance and capacitance of its membrane. The major conclusion from these measurements is that the specific capacitance values are consistent with those expected for biological membranes, whereas the values for the specific resistance are at least two orders of magnitude larger than those reported for nerve cells. The magnitude of the specific resistance values suggests that the permeability of the *Anisodoris* G cell membrane to ions is much smaller than that previously assumed for neural membranes. A preliminary report of these findings has been published elsewhere (Gorman & Mirolli, 1971).

METHODS

Experimental procedures

The gastro-oesophageal ganglion and medial nerve of *Anisodoris nobilis* (MacFarland) were isolated and mounted in a chamber filled with circulating sea water maintained at a constant temperature (10–11° C; see Gorman & Mirolli, 1969). The major and minor axes of the G cell soma were measured under a dissecting microscope with a micrometer eyepiece and from these the equivalent diameter, $d = \sqrt[3]{(a^2b)}$ was calculated as previously described (Mirolli & Talbott, 1972).

Two independent micro-electrodes filled with 3 M-KCl (resistance between 5 and 12 MΩ), one for recording and the other for stimulating, were inserted into the soma of the G cell through the intact ganglionic sheath. The output of the stimulating circuit was connected to one of the micro-electrodes through a 10^9 Ω series resistor to assure constant current stimulation and the magnitude of the current was recorded as the voltage drop across this resistor. The other side of the circuit was connected to a larger, low resistance Ag–AgCl electrode in the bath. A rectangular pulse of current was passed through one of the micro-electrodes and the resulting displacement of membrane potential was measured differentially between the other micro-electrode and a second low resistance in the bath.

Reduction of capacitative coupling between the stimulating and recording circuits was achieved by inserting the micro-electrode into the cell at right angles to reduce interelectrode distance except at their tips and by reducing the fluid above the cell to a height of approximately 300 μ. When measurements were taken with the tips of both electrodes close together (*ca.* 50 μ) in the bath outside of the cell, coupling between the two electrodes was not detectable at the current intensities used in the present experiments ($0{\cdot}5$–$1{\cdot}0 \times 10^{-9}$ A). Measurements were also taken with both electrodes inserted close together inside the ganglionic sheath, but outside of the G cell. With these current intensities there was no measurable potential drop associated with the passage of current across the sheath.

Analytical treatment of the data

The shape of the voltage transient to an applied current step is determined by both the time constant of the membrane, τ, and the (steady-state) conductance ratio, ρ, between the axonal (or dendritic) and the somatic compartments (Rall, 1959, 1960). τ can be evaluated according to the procedure outlined by Rall (1960). If the early part of the transient can be analysed then ρ can also be determined. For this analysis, $\ln(\sqrt{t}\,\mathrm{d}V/\mathrm{d}t)$ is plotted *vs.* time t, and a value for τ is obtained from the slope of the straight line fitted to the latter part of the experimental points. The time scale is normalized in terms of τ and the data are replotted as $\ln(\sqrt{T}\,\mathrm{d}V/\mathrm{d}T)$, where $T = t/\tau$. The experimental curve is matched against the theoretical curves calculated for different values of ρ (Fig. 1). The computational task necessary to fit the appropriate theoretical curve to the data requires a series of successive approximations which may be quite laborious. If ρ is between 0 and 10 it can be determined, with sufficient accuracy, by finding the time at which the function $\ln(\sqrt{T}\,\mathrm{d}V/\mathrm{d}T)$ is at a maximum ($T_{\max}$) in an experimental transient. ρ can then be read directly in the graph shown in the insert of Fig. 1. This graph, a plot of ρ *vs.* $T_{\max}$, was obtained by using the general expression for $\mathrm{d}V/\mathrm{d}T$ calculated from eqn. (9) of Rall (1960), multiplying both sides of the expression by $\sqrt{T}$, taking the natural logarithm and differentiating:

$$F''_{(T)} = \frac{1}{2T} + \rho^2 - 1 - \frac{\rho \exp(-\rho^2 . T)}{\operatorname{erfc}(\rho\sqrt{T}) . \sqrt{(\pi T)}} \tag{1}$$

$T_{\max}$ was calculated by substituting different values of ρ in eqn. (1) equating to zero and solving for T.

If both τ and the specific membrane resistance, R_m are known, an estimate of the specific membrane capacitance, C_m can be obtained from the ratio τ/R_m. An independent estimate of C_m can also be obtained from the expression given by Lux & Pollen (1966).

$$\frac{C_s . \mathrm{d}V/\mathrm{d}t}{I} = \frac{\exp(\rho^2 . t/\tau) . \operatorname{erfc}(\rho\sqrt{t/\tau})}{\exp(t/\tau)}, \tag{2}$$

where C_s is the total somatic capacity and I is the current applied. For times close to 0, the right-hand side of eqn. (2) approaches unity, and the expression reduces to

$$C_s \approx \frac{I}{\mathrm{d}V/\mathrm{d}t}. \tag{3}$$

An approximate estimate of C_s can be obtained by plotting $I/(\mathrm{d}V/\mathrm{d}t)$ *vs.* t for very short times after the onset of the current pulse, fitting a curve to the experimental points and extrapolating to time zero. For times different from 0, the right-hand side of eqn. (2) will be less than unity, and therefore, C_s will be over-estimated, by an error which is directly related to magnitude of ρ and inversely related to magnitude of τ. For a value of $\rho < 10$ and of $\tau = 1$ sec, a plot of $I/(\mathrm{d}V/\mathrm{d}t)$ *vs.* t is practically

linear within the time interval of 1–5 msec and the intercept of a straight line fitted to the experimental point with the $I/(dV/dt)$ axis results in a maximum overestimate of C_s by no more than 20%.

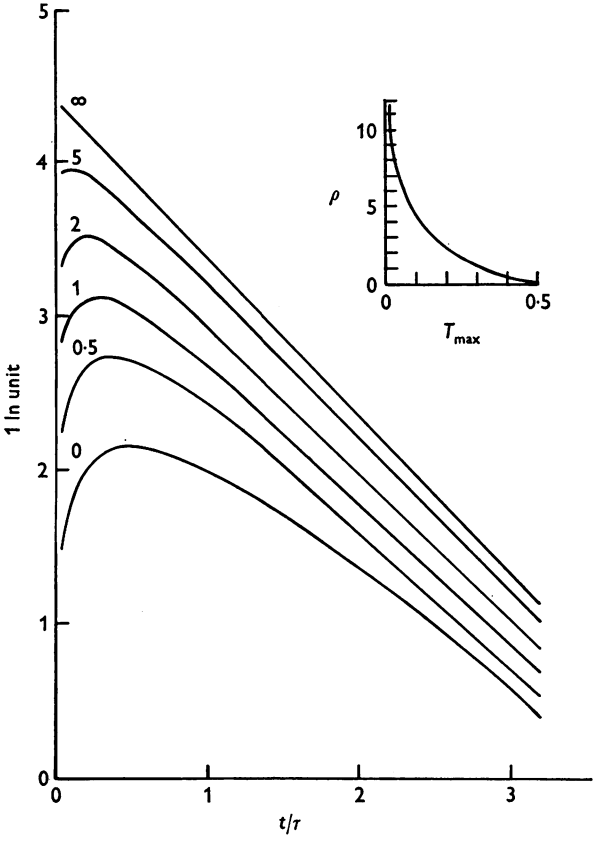

Fig. 1. The function

$$\ln\{\sqrt{T}.F'_{(T)}\} = \ln\left\{\sqrt{T}.\frac{d}{dT}\left(\frac{C_0}{\rho-1}\right)[\rho.\mathrm{erf}\sqrt{T} - 1\exp(\rho^2-1).T.\mathrm{erfc}(\rho\sqrt{T})]\right\}$$

plotted for the values 0, 0·5, 1, 2, 5, and ∞ of the axosomatic conductance-ratio, ρ. The constant, C_0 is assumed equal to 1 for all the values of ρ considered. The time scale has been normalized in terms of the time constant τ ($T = t/\tau$). These curves are comparable with those plotted by Rall (1960, Fig. 2) and Eccles (1961, Fig. 3). The inset shows a graph of ρ as a function of T_{max}. The method of computing T_{max} is given in the text.

RESULTS

Measurement of the specific membrane resistance

For most of the cells examined, the relation between applied current and membrane potential was not linear. This was particularly evident when

large currents were used, since the resistance of the membrane decreased markedly for both depolarizing (delayed rectification) and hyperpolarizing (anomalous rectification) currents (Gorman & Mirolli, 1970; Marmor, 1971*a*). For some cells, anomalous rectification occurred near the resting potential (Fig. 2). In others, it became apparent 5–15 mV below the resting potential level. For this reason the input resistance (total neurone resistance, R_T) was estimated from the slope of the current–voltage relation at $I = 0$.

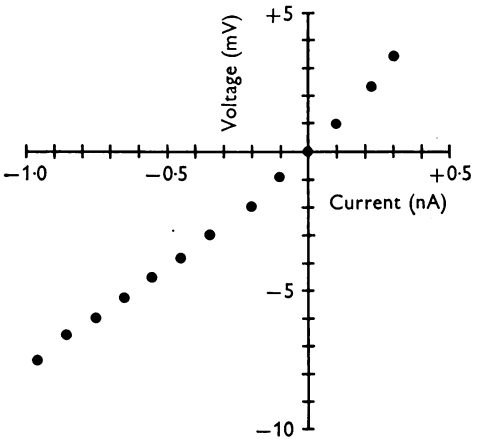

Fig. 2. Current–voltage relation of the G cell. Experimental points indicate the steady-state voltage produced by current steps. Resting potential of this cell was −57 mV.

Table 1 summarizes the average anatomical and electrical measurements taken from ten selected cells. Estimates of the surface of the soma, S, and of the geometrical factor for the axonal input conductance, M, were computed from the equivalent cell diameter, d, according to the procedures described by Mirolli & Talbott (1972). The specific membrane resistance, R_m, was calculated from the expression (see Appendix to the paper of Mirolli & Talbott, 1972)

$$R_m = \left(\frac{M+\sqrt{(M^2+4SG_TR_i}}{2G_T\sqrt{R_i}}\right)^2, \tag{4}$$

where G_T is the whole neurone conductance ($1/R_T$); and R_i is the specific resistance of the axoplasm. We have assumed R_i to be 100 Ω.cm, a value which is similar to those found for axons of other marine animals (Cole & Hodgkin, 1939; Hodgkin & Rushton, 1946; Hodgkin, 1947). Since the M factor can only be determined within a range of values (Mirolli & Talbott, 1972), two sets of values are given for R_m: the first R_{m_1} corresponds to the lower limit (M_0) and the second, R_{m_2} to the upper limit ($\bar{M}_0$) of the axonal

factor. Both values are at least two orders of magnitude larger than the values which have been calculated for other biological membranes.

The membrane transient response

The voltage transient of the G cell does not follow a simple exponential time course. This is shown in Fig. 3 where five transients produced by currents of different intensity are plotted as $\ln(dV/dt)$ *vs.* t. A straight line might be fitted to the later portion of each transient, but for the first 50–500 msec the points systematically deviate from this line, indicating the

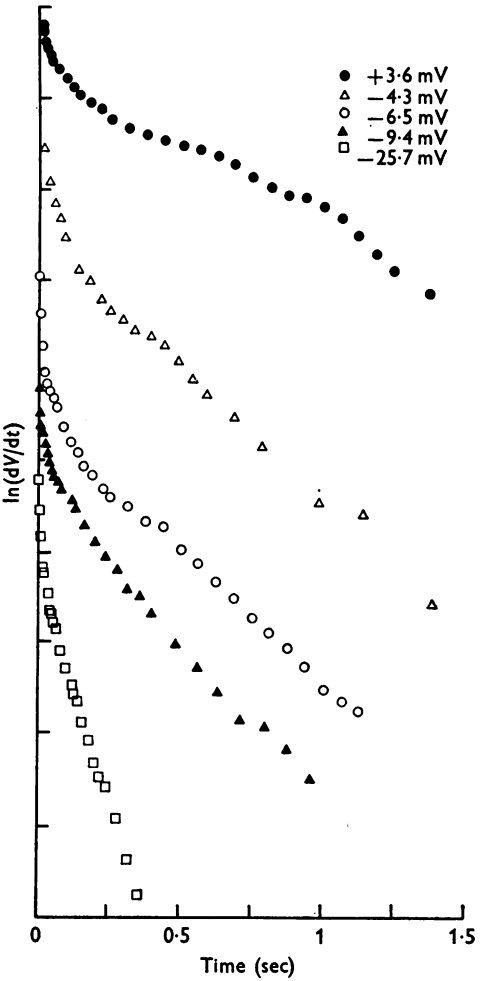

Fig. 3. Membrane voltage transients to applied current steps plotted as the natural logarithms of dV/dt *vs.* time. Five transients from the same cell (one depolarizing and four hyperpolarizing) are shown.

presence of at least one additional faster component. This type of deviation is expected when the axo-somatic conductance ratio is greater than zero. Fig. 3 also shows that the transient is faster when stronger hyperpolarizing current intensities are used. This effect is consistent with the decrease in membrane resistance which occurs when the G cell is hyperpolarized (Fig. 2). To reduce the magnitude of this effect, the analysis of the transient was restricted to small hyperpolarizing responses ranging between 2·5 and 6·5 mV in amplitude.

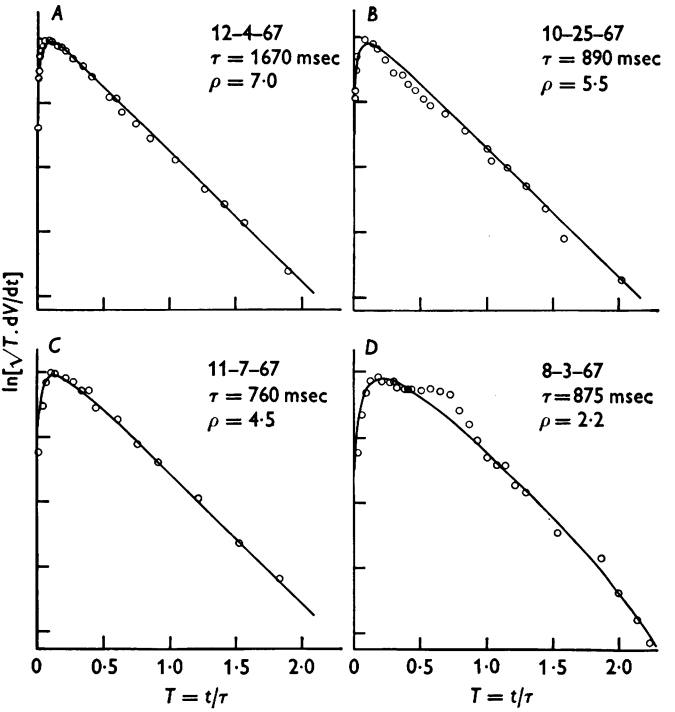

Fig. 4. Comparison of theoretical and experimental voltage transients. Membrane transients from 4 different cells plotted as the natural logarithm of $\sqrt{T}.\,\mathrm{d}V/\mathrm{d}t$ (open circles). The time scale has been normalized in terms of the time constant for each response ($T = t/\tau$). The continuous lines indicate the best-fitting theoretical curves calculated from eqn. (9) of Rall (1960). Values for the axo-somatic conductance ratio (ρ) and time constant (τ) are given above each plot.

Fig. 4 shows voltage transients from four different cells plotted as $\ln(t\,\mathrm{d}V/\mathrm{d}t)$ *vs.* t. The best-fitting theoretical curves, calculated from values of τ and ρ found experimentally (see Methods), are superimposed on the experimental points. With most of the cells examined the fit was good (Fig. 4*A–C*). The agreement between theoretical curves and the experi-

mental points was particularly satisfactory when ρ (determined from T_{max}) was greater than 4; when ρ was smaller, the agreement was less satisfactory (Fig. 4*D*). It was not possible to fit transient responses produced by depolarizing currents. The average values for τ and ρ are given in Table 1.

TABLE 1. *Anisodoris* G cell. Anatomical and electrical parameters*

Soma diameter	Soma surface	Axonal factor	
10^{-4} cm	10^{-3} cm^2	10^{-4} cm$^{3/2}$	
d	S	M_0	$\overline{M}$
330 ± 31	$26{\cdot}06 \pm 2{\cdot}07$	$11{\cdot}8 \pm 0{\cdot}6$	$17{\cdot}8 \pm 0{\cdot}8$

Input resistance	Specific membrane resistance		Axo-somatic conductance
$10^6\ \Omega$	$10^3\ \Omega \cdot$cm^2		Ratio
R_T	R_{m_1}	R_{m_2}	ρ
$5{\cdot}9 \pm 0{\cdot}6$	740 ± 90	1370 ± 190	$4{\cdot}8 \pm 0{\cdot}4$

Time constant	Specific membrane capacitance		
10^{-3} sec	10^{-6} F/cm^2		
τ	C_{m_1}	C_{m_2}	C_{m_3}
980 ± 140	$1{\cdot}36 \pm 0{\cdot}15$	$0{\cdot}75 \pm 0{\cdot}08$	$1{\cdot}14 \pm 0{\cdot}07$

* Means and S.E. of the means of values determined for ten cells.

Measurement of the specific membrane capacitance

Three sets of values for the specific membrane capacitance, C_m, are given in Table 1. The first two, C_{m_1} and C_{m_2}, were calculated from the time constant estimated from the slope of the best-fitting line through the linear portion of the transient response plotted as $\ln(\sqrt{t}\, dV/dt)$ *vs.* t (Fig. 4) and from the values of R_{m_1} and R_{m_2} respectively (Table 1). The difference between the two estimates reflects the difference between the values for specific resistance used in the computations. Since R_{m_1} and R_{m_2} are defined as a lower and an upper estimate of R_m. C_{m_1} and C_{m_2} are the upper and the lower estimates, respectively, for the specific capacitance of the G cell membrane.

The third estimate of specific capacitance, C_{m_3}, is the ratio between the somatic capacity, C_s, and the soma surface area. This estimate for C_m, unlike C_{m_1} and C_{m_2}, does not depend on the axonal factor, M. C_s was obtained by graphical analysis of the earliest part of the transient (see Methods). The values of C_s for the same cell were remarkably constant over a wide range of current intensities (Fig. 5). The three estimates for specific capacitance are in substantial agreement and are well within the range of the values expected on the basis of theoretical considerations (Cole, 1968).

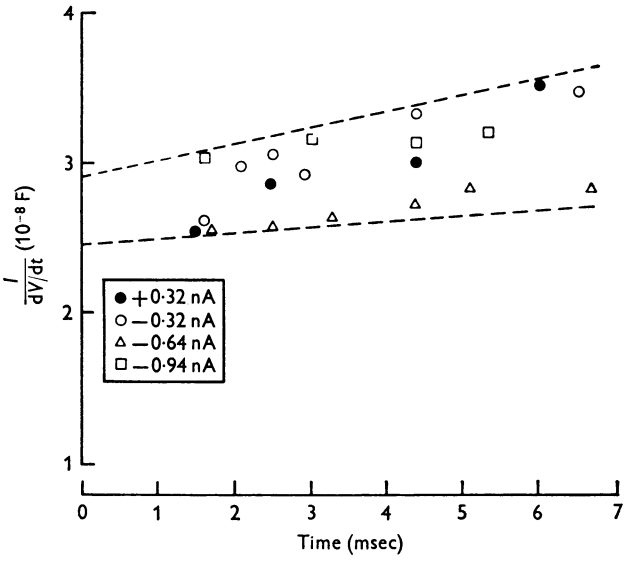

Fig. 5. Estimation of soma total capacitance, C_s. Plot of $I/(dV/dt)$ *vs.* time for the earliest portion of four membrane transients produced with different current intensities in the same cell. C_s is estimated as the value of $I/(dV/dt)$ at time 0. The lines shown above and below the experimental points measure the error for this estimate due to the graphical approximation.

Theoretical values for the G cell electrical parameters

An indication of the validity of our measurements can be obtained from a comparison of the theoretical values for the time constant (τ) and the axo-somatic conductance ratio (ρ) (Table 2) calculated from our combined anatomical and electrophysiological measurements with the actual values found from the analysis of transients. The mean values for τ and ρ determined from the membrane's transient (Table 1) fall between the upper and lower limits set by our anatomical estimates (Table II), but are much closer to the minimum values. This is not surprising, since half of the cells examined exhibited some degree of anomalous rectification which would tend to reduce both τ and ρ. The mean value of 4·8 found for ρ (Table 1), even if slightly underestimated, is much greater than previous estimates (0·4) given for other gastropod neurones (Magura, 1967) and clearly indicates a large contribution of the axon to the G cell's input conductance.

Table 2 also shows maximum and minimum estimates for the length constant (λ) based on our combined anatomical and electrophysiological data. The mean values agree with previous results (Gorman & Mirolli, 1968) which suggested that the length constant of the G cell axon is large.

Placement of a micro-electrode inside the axon to directly measure the spread of potential from the soma was only possible in one experiment, but clearly shows (Fig. 6) that a potential evoked by a somatic transmembrane stimulus is only slightly smaller in the axon at approximately 1·5 mm from the point of stimulation. This finding is consistent with our theoretical estimates for the G cells length constant.

TABLE 2. *Anisodoris* G cell. Expected values for time constant (τ), conductance ratio (ρ) and length constant (λ)

Time constant (10^{-3} sec)		Axo-somatic conductance ratio		Length constant (cm)	
Max.	Min.	Max.	Min.	Max.	Min.
1560 ± 250	860 ± 130	7·54 ± 0·65	3·97 ± 0·31	1·09 ± 0·08	0·81 ± 0·05

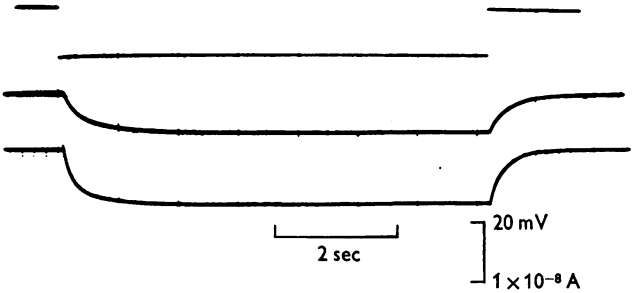

Fig. 6. Spread of potential from soma to axon. Potential displacement of the soma (middle trace) and axon (bottom trace) membranes produced by a current pulse (top trace) applied across the soma membrane. Time, voltage and current calibration are indicated. Resting potential was −59 mV.

DISCUSSION

Two immediate conclusions can be drawn from our study. First, the values given for the specific membrane capacitance are well within the limits expected for biological membranes from a consideration of their physical dimension and chemical structure (Cole, 1968). Secondly, the specific membrane resistance values are much greater than those reported for other neuronal membranes (Table 3).

It is conceivable that physiological as well as anatomical errors (see Mirolli & Talbott, 1972) affect our calculations. It is possible that the value used for the G cell axoplasmic resistance (R_i = 100 Ω.cm) differs from the actual value. This would be an important source of error if R_i was much smaller than the value used. For example, if R_i was one half of this value, the average R_m (1 MΩ cm^2) would be 1·7 times larger than found; conversely, if R_i was twice the value used, R_m would be 0·65 times smaller.

The recent results of Carpenter, Hovey & Bak (1971) suggest that the cytoplasmic conductance of some *Aplysia* neuronal somas may be 10–20 times that of sea water.

TABLE 3. Electrical properties of molluscan nerve cell

Species	Cell type	Soma diameter (10^{-4} cm)	R_T ($10^6\ \Omega$)	τ (10^{-3} sec)	R_m ($10^3\ \Omega.cm^2$)	C_m (10^{-6} F/cm^2)	ρ	References
Aplysia depilans	Abdominal RGC	500–700	0·1–0·22	100–200	11·3–2·5	12·4–56	—	Kandel & Tauc (1965) Hughes (1967)
Aplysia fasciata and *depilans*	Abdominal LGC	600	0·23–0·36	160	2·6–4	40–60	—	Hughes (1967)
Aplysia	Abdominal (small)	178	2·2	50 (10–80)	2·2	23	—	Fessard & Tauc (1956) Tauc (1955)
Aplysia californica and *punctata*	Visceral	80–500	1–10	20–50	0·5–10	3–20	—	Murray (1966)
Helix pomatia	Visceral	110	2·4	30	0·99	28·8	0·4	Maiskii (1963) Magura (1967)
Helix pomatia	Sub-oesophageal	80–140	12·1	54	8·3	6·5	—	Meves (1968)
Tritonia diomedia	Cerebropleural	300	1·9	50	5·0	1·0	—	Veprintsev, Krasts & Sakharov (1964)
Onchidium verruculatum	Oesophageal	100–300	—	—	2·5–4·5	8–12	—	Hagiwara (1960)
Anisodoris nobilis	Gastro-oesophageal	330*	5·9	977	1000	1·2	4·8	This paper

R_T, input resistance; R_m, specific resistance; τ, time constant; C_m, specific capacitance, ρ, axo-somatic conductance ratio.

* diameter, d, of G cell soma calculated from the relation $d = \sqrt[3]{(a^2b)}$, where a and b are the major and minor axes of the cell's optical outline.

This would imply a value for R_i which is 2–5 times larger than the one we have used. R_m would be smaller (approximately 0·4 times) by making R_i five times larger. However, this increase in R_i would not bring R_m within the range of values found for axonal membranes. There are, moreover, several problems which need to be resolved before employing a value for R_i based on their results. First, the low conductance given for the *Aplysia* soma cytoplasm suggests a binding of ions within the cell, yet the activity measurements given by Russell & Brown (1972) indicates that the major ions (K^+ and Cl^-) are not bound. Secondly, the Carpenter *et al.* (1971) analysis was restricted to the soma cytoplasm which because it contains large pigment molecules may have a very different conductance than the axoplasm. In our analysis it is only the resistance of the axoplasm which is important. Last, any reduction of R_m necessitates an increase in the value for C_m which is difficult to justify on theoretical grounds.

In addition, use of the cable equations in their present form (Rall, 1960; Mirolli, 1970) is based on the questionable assumption that the electrical characteristic of the membrane can be approximated by a linear model. There is clear evidence that the membrane has rectifying properties near resting potential and this fact introduces an additional source of uncertainty which, though difficult to evaluate, must be kept in mind.

However, there are several checks on our measurement procedure which suggest that the values summarized in Table 3 for the passive electrical parameters of the *Anisodoris* G cell are valid. The analysis of the membrane's transient response provides a direct estimate, which is independent of morphological data, for both the time constant, τ, and the axo-somatic conductance ratio, ρ. The substantial agreement between these values and the theoretical values for τ and ρ obtained using estimates from R_m and C_m from anatomical and electrophysiological data suggests that the model of the G cell, advanced by Mirolli & Talbott (1972) is valid. Membrane rectification may complicate the evaluation of τ and ρ. However, recent evidence from our laboratory (Marmor, 1971*a*) has shown that anomalous rectification can be eliminated by cooling the G cell to 0°–5° C and its membrane properties examined under conditions without this complicating factor. In agreement with the values given in Table 1, Marmor's results show that near resting potential τ ranges between 600–1000 msec and the ρ is greater than 5. In addition, his values for R_m (0·1–1·5 $M\Omega.cm^2$) and C_m (0·5–1·0 $\mu F/cm^2$) are consistent with those given in this paper.

The strongest evidence that our measurements are valid comes from the membrane capacitance data which agree with the values reported for those axons where estimates of membrane-surface area are reasonably accurate as well as with the theoretical value for membrane capacitance (1 $\mu F/cm^2$) expected for a thin membrane (*ca.* 75Å) having a dielectric constant of 3–6.

The membrane resistance has been regarded as an index of the ion concentration and ion mobility of the membrane (Cole, 1940; Hodgkin, 1947). To account for the very high specific resistance of the *Anisodoris* G

cell, we must conclude that the product of ionic mobility and concentration in the membrane is extraordinarily low. The G cell is primarily permeable only to cations and at the temperature used in the present experiment (10–11° C), is about 33 times as permeable to K^+ as to Na^+ (Gorman & Marmor, 1970*b*). The resistance measurements, therefore, essentially provide an index of the K^+ permeability of the membrane and indicate that the G cell is approximately 1000 times less permeable to K^+ than the squid axon. Recent measurements of the K^+ conductance by Marmor (1971*a*) confirm that the permeability of the G cell membrane to K^+ is extraordinarily low.

Although it is possible that the conclusions reached for the G cell may apply only in part to other molluscan nerve cells (Table 3), the large difference between the value for R_m and C_m of *Anisodoris* and that of its close relative *Aplysia*, for example, suggests that both the electrical and anatomical data given for other molluscan cells should be re-examined. In considering the data of Table 3, it should be kept in mind that there is clear evidence that the giant abdominal ganglion cell in *Aplysia* has a highly infolded membrane (Coggeshall, 1967) and a large length constant (Tauc, 1962) data which are not easily reconcilable with the low value given for R_m. Measurements given for *Helix* neurones (Maiskii, 1963; Magura, 1967; Meves, 1968) indicate that the axonal contribution to input resistance is much less than the G cell. However, the larger values given for their specific capacitance (Table 3) suggest that even in these cells, the surface area of the soma membrane may have been significantly underestimated.

Our results may also have some relevance for non-molluscan nerve cells. Unlike the specific membrane capacitance, there is no theoretical reason for assuming a definite value for the specific membrane conductance. If a given ionic concentration must be maintained across the membrane, any 'passive leak' of the ion species must be compensated by work expended by the cell (see Gorman & Marmor, 1970*a*). In general, the amount of leakage must be proportional to the total cell surface, while the active ionic transport depends on production of both enzyme and energy yielding molecules, and thus, ultimately, is proportional to the cell volume. From this point of view, the very low value for the G cell membrane conductance is not extraordinary, but rather it appears to be dictated by metabolic economy. Thus, it is possible that the G cell represents a model for other cells having large surface-to-volume ratios, e.g. both vertebrate and invertebrate visual cells with their greatly infolded membrane, and the large cells of the vertebrate nervous system, such as Pyramidal cells, Purkinje cells and motoneurones with their characteristic extensive dendritic arborization.

Finally, it must be stressed that the values given for the G cell's passive

electrical parameters, other than for its capacitance, cannot be regarded as constants. Specific resistance, length constant, time constant and the axo-somatic conductance ratio all vary with both membrane potential and temperature (Gorman & Mirolli, 1970; Marmor, 1971*a*). The values given here, however, should represent a valid set of approximations of the electrical properties of the resting membrane of this cell under steady-state conditions when the net flux of ions across the membrane equals zero.

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