tion on a copper block in the path of the air current, and allowed to stand for 30 minutes in order to attain steady-state conditions. It was then taken out and quickly weighed in an analytical balance weighing to 0.0001 g (*5*). Weighings were repeated at intervals of approximately 60 minutes. Monolayers of hexadecanol were spread at their equilibrium spreading pressure by addition of a small excess of crystals to the surface.

The retardation of evaporation by the hexadecanol monolayer is the same under comparable conditions of convection and is independent of the absolute rate of evaporation (Table 1). Thus, for these experiments, the hexadecanol monolayer produces no barrier to the vaporization step and exerts its effect by altering the hydrodynamic boundary conditions.

When air is passed over a liquid surface, there is always some movement of the surface as a result of the net stress due to the air. If a sufficiently incompressible monolayer is placed on the surface, the stress (τ) due to the moving air compresses the monolayer, producing a surface-pressure gradient ($d\pi/dx$) which opposes the stress. The back stress of the monolayer reduces the net stress on the surface (*6*) and therefore consequently increases the size of the boundary layer.

The highest air velocity (U_0) in these experiments was 480 cm/sec so that, at a value of 0.15 for the kinematic viscosity (ν) of air at 20°C, the highest value of the Reynolds Number ($\mathrm{Re} = U_0 \cdot x/\nu$) calculated at the far end of the tray ($x = 9.0$ cm) is 29×10^4. Laminar flow may therefore be assumed in all the experiments.

Values of δ were calculated from Eq. 1 (Table 2). For the experiments without monolayers, the average thickness of the hydrodynamic boundary layer δ_0 which occurs at about $x = 3.8$ cm, was calculated from (*7*)

$$\delta_0 = 4.64\ \mathrm{Re}_x^{-1/2}\ x \qquad (2)$$

Corresponding values of δ_0 for the experiments with monolayer covered surfaces were calculated by assuming δ to be proportional to δ_0 at a given air velocity (*8*). Finally, the net stress on the surface (*9*) was calculated from

$$\tau = \eta\ (U_0/\delta_0) \qquad (3)$$

where η is the viscosity of air (1.83×10^{-4} poise at 20°C).

If no net flow of the monolayer occurs under steady-state conditions, $\tau = d\pi/dx$. Thus, from the data in Table 2, it is seen that remarkably small surface pressure gradients can cause significant changes in boundary layer properties and consequently evaporation rates. The condition of no net flow is probably approached more closely by highly incompressible monolayers, thus explaining the effectiveness of these in reducing evaporation (*10*). More measurements of surface pressure gradients and flow patterns of monolayers in wind tunnel experiments would help considerably in the understanding of the effectiveness of different monolayers in reducing evaporation.

F. MacRitchie
C.S.I.R.O. Wheat Research Unit, North Ryde, Australia

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4. Thus, the "specific evaporation resistance" which has often been calculated for monolayers is not a material property as, for example, the specific electrical resistance of a metal. In fact, where a reaction sequence involves one or more equilibrium steps, as occurs in mass transport across interfaces and through membranes, the analogy with a simple electrical circuit breaks down and the calculation of resistances for different steps from kinetic data becomes of dubious validity.
5. This method of measuring evaporation rates is considered superior to that originally introduced by Langmuir and Schaefer [I. Langmuir and V. J. Schaefer, *J. Franklin Inst.* **235**, 119 (1943)]. It is easier to treat theoretically since it is essentially evaporation into a semi-infinite reservoir from an accurately known surface area. The conditions can also be varied more easily. The Langmuir-Schaefer method is complicated by the geometry of the system and by the introduction of additional diffusion and temperature gradients due to the presence of the absorbent. Steady-state conditions are therefore more difficult to attain.
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9. The assumption of a linear velocity gradient is an approximation and will give only rough values for the shear stress.
10. For certain surfactant films, it has been shown [L. F. Mockros and R. B. Krone, *Science* **161**, 361 (1968)] that a balance between the net stress on the surface and the back stress due to the film cannot necessarily be assumed owing to flow of the film.

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Synaptic Activation of an Electrogenic Sodium Pump

Abstract. *An identified molluscan interneuron mediates different cholinergic synaptic actions by increasing the conductance of its follower cells to different ions. We have now found that this interneuron also mediates a new class of synaptic actions which does not involve a conductance change but the activation of an electrogenic sodium pump. This synaptic action results in a prolonged inhibitory synaptic potential which is dependent on metabolism and is selectively blocked by cooling and ouabain. In cells which have this synaptic potential, part of the resting membrane potential is also maintained by an electrogenic sodium pump. The same transmitter, acetylcholine, can independently stimulate both a chloride ion conductance and a sodium pump mechanism in the same follower cell by acting on two different postsynaptic receptors.*

The established mechanisms of chemical synaptic transmission involve an increase in the conductance of the postsynaptic membrane to one or more ion species which then move down their concentration gradients without requiring metabolic energy (for review, see *1*). Nishi and Koketsu (*2*) have recently suggested the existence of a new class of chemical synaptic transmission which is metabolically dependent and involves the activation of an electrogenic Na^+ pump. Their conclusions were based on studies of the slow inhibitory postsynaptic potential in the frog sympathetic ganglion using a sucrose gap technique for recording the activity of populations of nerve cells. However, the conclusions of Nishi and Koketsu have recently been challenged by the more direct studies of Kobayashi and Libet (*3*) using intracellular recordings. We now present independent evidence for the synaptic activation of an electrogenic Na^+ pump based on intracellular recordings from individual identified cells in the abdominal ganglion of the marine mollusc, *Aplysia*. Furthermore, our data indicate that the same transmitter, acetylcholine, can independently stimulate both the conductance and the Na^+ pump mechanisms for synaptic transmission.

An identified interneuron (cell L10) in the abdominal ganglion of *Aplysia californica* mediates three different synaptic actions—excitation, inhibition, and conjoint excitation-inhibition—via dif-

ferent branches to different follower cells (*4*). These synaptic actions appear to be produced by the release of a single transmitter, acetylcholine, which interacts with different receptors in the postsynaptic follower cells to produce different patterns of ionic permeability. The excitatory action produces an increase in Na^+ conductance, the inhibitory action an increase in Cl^- conductance, and the conjoint action an increase in both Na^+ and Cl^- conductance (*5*). In addition to these actions, we have now found that this interneuron produces another type of synaptic action which does not involve a conductance change.

A single action potential in the interneuron produces a monosynaptic hyperpolarizing inhibitory postsynaptic potential (IPSP), of about 800 msec duration, in six identified follower cells (L1, L2, L3, L4, L5, and L6) of the left rostral portion of the ganglion (*3*, and Fig. 1A, part 1a). When the interneuron discharges a train of action potentials, the duration of the hyperpolarization becomes disproportionately prolonged (Fig. 1A, part 1b). Even a single spike from the interneuron can sometimes trigger a prolonged IPSP, especially in cell L2 (Fig. 1B, part 1). This finding, and the pharmacological data to be described later, support the notion that the prolonged IPSP is also monosynaptically mediated. We can demonstrate with a number of different experimental procedures that the prolongation of the IPSP involves the activation of a second and independent synaptic process which we will refer to as the "late IPSP" to distinguish it from the early IPSP.

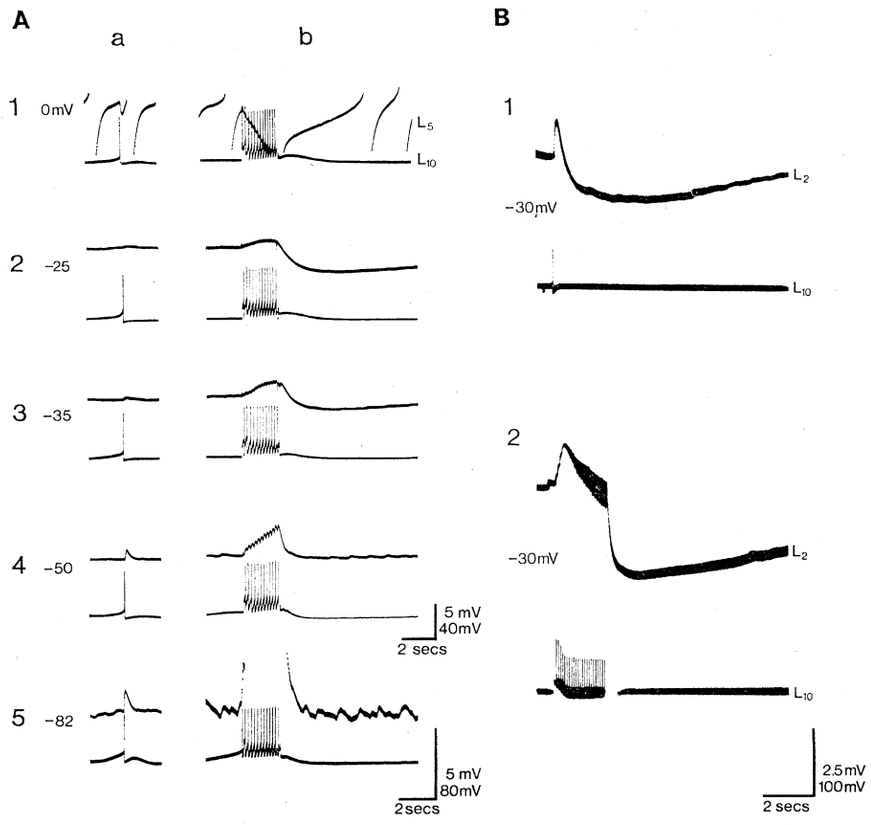

Fig. 1. Early and late IPSP's. (A) The amplitude of the early and late IPSP, following single spikes and trains of spikes in the interneuron, as a function of membrane potential. In this experiment and those illustrated in the subsequent records, a double-barrel microelectrode was inserted into the interneuron, cell L10 (bottom trace), (one barrel was used for recording and the other for passing current) and two independent microelectrodes were inserted into the follower cell, cell L5 (top trace). The tops of the spikes in L5 were cut off in photography in *1a* and *1b*. The changes in resting potential are indicated as millivolts of hyperpolarization ranging from 0 mv, at the resting level of membrane potential, to −82 mv. This represents a maximum change in the resting potential from −45 mv to −127 mv. (*a1–a5*) A single spike in the interneuron produced only an elementary (early) IPSP in the follower cell, which was nullified at an equilibrium potential of about −20 mv of hyperpolarization and became progressively more depolarizing beyond −25 mv. (*b1–b5*) When the interneuron was depolarized so as to discharge a train of spikes a second component became evident. At the resting level (0 mv hyperpolarization), this second component only manifested itself as a slowed return of the early IPSP and a delayed firing of the endogenously active follower cell. As the membrane potential was raised beyond the equilibrium potential of the early IPSP (−25 and −35 mv of hyperpolarization), the late component failed to invert and was clearly evident as a late hyperpolarization. The late IPSP decreased progressively in amplitude as the membrane was further hyperpolarized, but it failed to invert even when the membrane potential was brought to −127 mv (−82 mv of hyperpolarization). The voltage gain was increased in *b5* (only) to illustrate that no inversion of the late IPSP was detectable even at high gain. (B) Early and late IPSP in follower cell L2 following single spike (*B1*) and train (*B2*) in the interneuron (cell L10). The membrane potential was hyperpolarized by 30 mv so as to invert the early IPSP. In cell L2 the late IPSP is particularly well developed and even a single action potential can trigger it. In *B2*, the L10 record is a-c coupled.

The early and late IPSP's responded differently when the resting membrane potential of the follower cell, usually −45 mv, was increased by passing hyperpolarizing current through a second intracellular microelectrode. The early IPSP inverted to a depolarizing postsynaptic potential when the membrane potential was brought beyond the Cl^- equilibrium potential (Fig. 1A, part a). This dependence of the sign of the synaptic potential on membrane voltage is characteristic of a classical conductance IPSP (*1*). The late IPSP failed to invert, even when the membrane potential was increased by more than 80 mv (Fig. 1A, part b) bringing it to an absolute level of about −127 mv. This lack of inversion suggests that the late IPSP is not due to an increased conductance to K^+ or Cl^-, the two ion species which could produce a hyperpolarizing potential change. Indeed, the late IPSP was not affected by substituting proprionate for external Cl^-, although the early IPSP was reversed. The late IPSP was also not increased by reducing external K^+ (see below). To further exclude an ionic conductance change we also measured the membrane conductance directly and found that the early IPSP produced the expected increase in membrane conductance whereas the late IPSP did not.

Although the late IPSP does not have an ionic equilibrium potential, it did decrease in amplitude with increasing

membrane potential (Fig. 1A, part b). Some of the decrease resulted from anomalous rectification in the extrasynaptic membrane (*6*) but the remaining decrease may represent a voltage dependence of the current for the late IPSP. Alternatively, additional rectification might be occurring in the synaptic region which was not detected by the electrode in the cell body.

The lack of an ionic conductance change indicates that the late IPSP cannot be explained by the usual mechanisms involved in chemically mediated synaptic inhibition. Instead, the properties of the late IPSP suggest that the transmitter may have stimulated the activity of an electrogenic pump.

A cell maintains a low intracellular concentration of Na^+ and a high intracellular concentration of K^+ by actively transporting these ions across its membrane against their electrochemical gradients (*7*). In some excitable membranes the outward movement of Na^+ is exactly coupled with an inward movement of K^+ and therefore the "pump" is electrically neutral (*7*). However, in other excitable cells the coupling ratio is not unity (for example, 3 Na^+ to 2 K^+) and some net charge is pumped across the membrane. In these cells the pump is electrogenic and may contribute to the resting potential (*8*). For example, in certain cells in *Aplysia* the electrogenic Na^+ pump accounts for as much as one-third of the resting potential (*9*).

The active transport of Na^+ and K^+ involves a Na^+ and K^+ activated adenosine triphosphatase (*10*). This adenosine triphosphatase and, consequently, the electrogenic Na^+ pump, is temperature-dependent (*11*), is inhibited by ouabain (*12*), and is sensitive to changes in extracellular K^+ and intracellular Na^+ concentration (*11*, *13*). We therefore examined the late IPSP with respect to these properties. A valuable control was provided by the early IPSP which should not be affected by manipulations which selectively inhibit the electrogenic pump.

As the temperature of the seawater solution bathing the ganglion was reduced there was a gradual and selective decrease of the late IPSP and an almost complete disappearance in the lower temperature range (10° to 7°C) (Fig. 2A, part 1). Similarly, when the ganglion was bathed in ouabain solution ($2 \times 10^{-4}M$) the late synaptic component was selectively inhibited (Fig. 2A, part 2). Both these results suggest that the late IPSP involves an active transport mechanism, perhaps an electrogenic Na^+ pump.

To produce the late IPSP an electrogenic Na^+ pump must transport Na^+ from the inside to the outside of the cell. Such a transport mechanism usually requires some external K^+ to exchange for the extruded Na^+ and can therefore be inhibited by removal of external K^+. In keeping with the suggestion of an electrogenic Na^+ pump, we found that prolonged washing with a K^+ free bathing solution blocked the late IPSP selectively.

Follower cells which showed electrogenic IPSP's became depolarized by about 10 mv when the bathing solution was cooled, when ouabain was added to it, or when external K^+ was re-

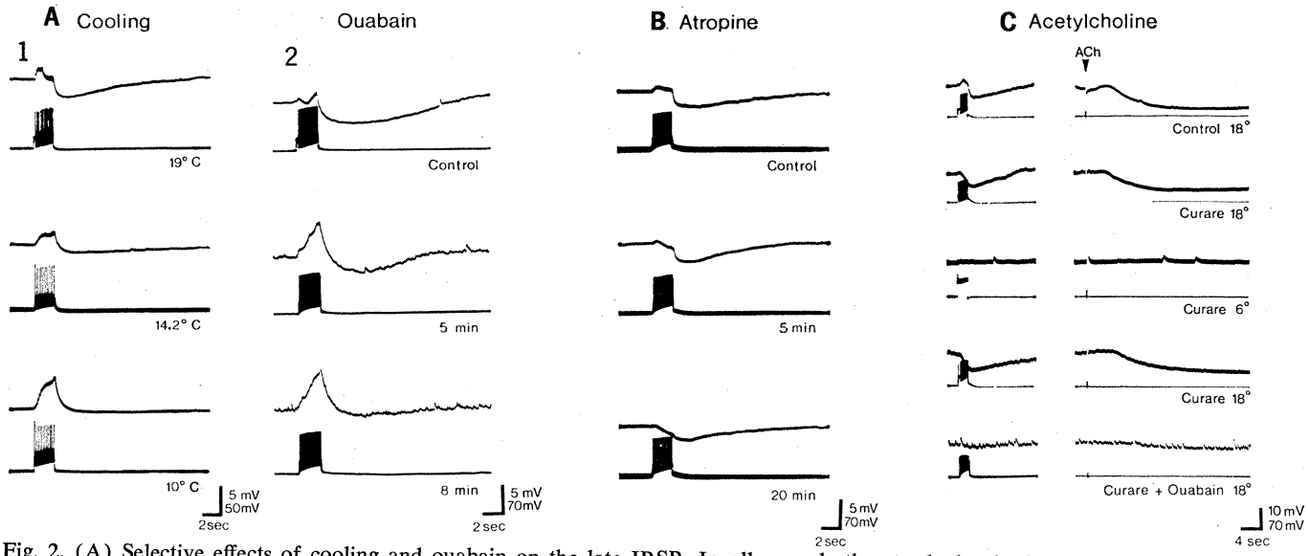

Fig. 2. (A) Selective effects of cooling and ouabain on the late IPSP. In all records the steady level of the membrane potential of the follower cell was kept constant and at a point which was beyond the equilibrium potential for the early IPSP. (1) Reducing the temperature from 19° to 10°C produced a progressive and selective decrease in the late IPSP. The increase in the early IPSP is an unmasking of its true size following the blockade of the late IPSP. (2) Ouabain ($2 \times 10^{-4}M$), a specific inhibitor of the Na^+-K^+ activated adenosine triphosphatase selectively decreased the late IPSP when added to the bathing solution. Because a component of the resting membrane potential is due to an electrogenic Na^+ pump, ouabain produced a depolarization in both the follower cell and the interneuron; only the change in the follower cell was compensated for. As a result the increase in the early IPSP represents in part a more effective train in L10 as well as an unmasking of the early IPSP. (B) Atropine (5×10^{-4} g/ml), a cholinergic blocking agent in this ganglion, selectively blocked the early IPSP and unmasked the actual time course and configuration of the late IPSP. Note that the late IPSP is present throughout the early IPSP. (C) Response of follower cell L2 to iontophoretic application of acetylcholine (ACh). In all of these experiments the membrane was hyperpolarized so that the early IPSP was inverted. The response of L2 to a train of spikes in L10 is illustrated on the left and the response of L2 to ACh iontophoresis is indicated on the right. Control, 18°C: the L2 response to L10 and to ACh is diphasic, first depolarizing and then hyperpolarizing. Curare, 18°C: the response of L2 to L10 and to ACh is only hyperpolarizing. Curare, 6°C: cooling the bathing solution to 6°C blocks the L2 response to both L10 and ACh. Curare, 18°C: warming the bathing solution to 18°C brings a return of the hyperpolarizing response in L2 to both L10 and ACh. Curare + ouabain, 18°C: ouabain blocks the remaining L2 response to both L10 and ACh.

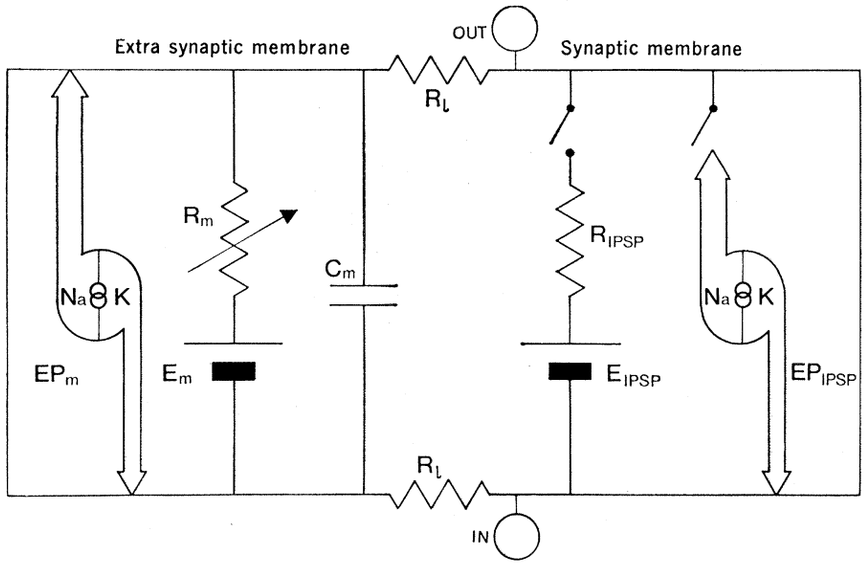

Fig. 3. Schematic diagram depicting the membrane components responsible for the resting potential (extrasynaptic membrane) and for early and late IPSP's (synaptic membrane). The generators for the resting membrane potential are depicted as consisting of two components: a conductance channel—a battery (E_m) in series with a variable, anomalously rectifying resistor (R_m)—in parallel with an electrogenic Na^+ pump (EP_m). C_m and R_l refer to the membrane capacitance and the longitudinal resistance of the membrane, respectively. The generators for the two PSP's are depicted as being in parallel with each other and operated by two independent switches (receptors). The early IPSP is generated by the usual conductance increase to Cl^- which results from the throwing of a switch connecting a battery (E_{IPSP}) and its series resistance (R_{IPSP}) into a parallel arrangement with the generators of the resting membrane potential. The late IPSP is generated by an electrogenic pump (EP_{IPSP}) which acts as a charge generator because it transports more Na^+ out than K^+ in.

moved (see *9*). Moreover, when we increased the intracellular Na^+ concentration by injecting Na^+ iontophoretically, the membrane hyperpolarized by 10 to 20 mv; comparable injections of K^+ produced much smaller potential changes. These data indicate that in cells showing the late IPSP the Na^+ pump is normally electrogenic and that the properties of the late IPSP are similar to those of the electrogenic component of the resting membrane potential. These data are consistent with the suggestion that the transmitter acts merely to increase the number of pump sites or the Na^+ pumping rate. Alternatively, the transmitter could produce a change in the Na^+-K^+ coupling ratio (*14*).

Acetylcholine, the transmitter implicated in the actions of this interneuron (*4*), can trigger both the conductance IPSP and the pump IPSP by acting on two different postsynaptic receptor mechanisms. The conductance IPSP was blocked by bathing the ganglion in atropine or *d*-tubocurarine (curare) (5×10^{-4} g/ml), which block known forms of cholinergic transmission in this ganglion (*4*, *15*), whereas the pump IPSP was unaffected (Fig. 2B). Iontophoretic application of acetylcholine on the synapse-free cell body, or adding acetylcholine to the perfusing solution, simulates both actions of the interneuron (Fig. 2C). Acetylcholine produces an early conductance component which is selectively blocked by curare, and a late hyperpolarizing component which is selectively blocked by cooling and ouabain (Fig. 2C) and is not affected by removing extracellular Cl^- (*16*).

The two types of mechanisms generating inhibitory synaptic potentials at this synapse can be compared by means of a schematic diagram (Fig. 3). The early IPSP is actived by acetylcholine acting on a receptor which increases the permeability to Cl^-. This mechanism can be depicted as a switch bringing a Cl^- battery of -65 mv and its low series resistance in parallel with the resting membrane potential (*1*). The late IPSP is activated by acetylcholine acting on a different receptor which increases the activity of the electrogenic pump. This second and independent mechanism can be depicted as a switch bringing a constant current generator with its high internal impedance in parallel with the resting membrane potential.

The two types of synaptic channels have very different properties. The synaptic current flowing in the conductance channel can be activated only by the appropriate chemical transmitter acting on the external surface of the membrane and not by voltage or ionic changes within the postsynaptic cell. By contrast, the pump channel triggered by the transmitter appears identical to that which contributes to the resting potential. This pump can be activated in two quite different ways: (i) on the external surface of the membrane by the transmitter released by the presynaptic neuron, and (ii) on the internal surface by increases in intracellular Na^+ concentration such as those produced by action potentials in the postsynaptic cell (*17*).

The present demonstration of synaptic activation of an electrogenic Na^+ pump and previous demonstrations that electrogenic Na^+ pumps can contribute to resting membrane potential (*8*, *9*), to prolonged posttetanic afterpotentials (*17*), and to receptor potentials (*18*) in different neurons suggest that the distribution of electrogenic Na^+ pump mechanisms may be widespread and may complement conductance mechanisms in performing a variety of signaling functions for nerve cells. In addition to these signaling functions, electrogenic Na^+ pump activation may have other functional consequences. The active transport of Na^+ and K^+ not only controls cell volume (*19*), but also provides a "pacemaker" for metabolism because about 50 percent of the energy metabolism of neurons is devoted to ion transport (*20*). A neuron which synaptically activates an electrogenic pump could therefore exert a continuous control over the metabolism of the cells it innervates via the transmitter substance it releases from its presynaptic terminals.

HAROLD PINSKER
ERIC R. KANDEL
Departments of Physiology and
Psychiatry, New York University
Medical School, New York 10016

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6 January 1969

Boron Modifications Produced in an Induction-Coupled Argon Plasma

Abstract. *Most of the small particles (50 to 100 micrometers in diameter) of microcrystalline β-rhombohedral boron that quickly transit an argon plasma maintained within a radio-frequency induction-coupled torch emerge as better crystallized spheroids of the same crystalline form and nearly the same size as the starting material. A few crystals of each of four distinctive, well-faceted habits are formed along with the general product. Three of these types are monocrystals of the β-rhombohedral polymorph, of the tetragonal-III modification, and of an unreported cubic form of boron. Specimens of the fourth type are polycrystals of another unreported form of boron, apparently consisting of many hexagonal platelets stacked in an imprecise fashion.*

Several different crystalline modifications of boron are recognized (*1–4*), but the structures of only three of these have been determined (*1, 3*). The large number of forms of the element and of structurally similar crystal species rich in boron (*5, 6*) has given rise to numerous discussions of the modes of formation of the structural variants, the nature of their bonding, and the ranges of their thermodynamic stabilities (*6*).

Many of these problems persist because it has been extremely difficult to prepare crystals of known composition which are of a sufficient size (100 μm in diameter) to allow the needed structure determinations to be made. Generally, the preparation of boron specimens has involved contact with other, supposedly inert, solids at high temperatures (*1–4, 6*), with the consequent risk of contamination and the formation of an ambiguous product.

In order to avoid such contamination during crystallization we dropped finely powdered (50 to 100 μm in diameter) polycrystalline β-rhombohedral boron through an argon plasma maintained within an induction-coupled radio-frequency torch (*7*). We used relatively pure starting material of widely varying isotopic content without significant effect on the results. By proper adjustment of the operating conditions of the torch, a large proportion of particles emerging from the plasma could be made spheroidal with some small flattened faces, but never truly spherical. This is in contrast to the commonly produced shape for oxides and metals after passage through such a plasma (*8*). Because the size range of the spheroids corresponded closely to that of the feed material and because vaporization appeared insufficient to produce the quantity of product obtained (a major fraction of the input sample), we believe that the spheroids were formed directly from individual feed particles by means of a liquid or solid-state conversion. X-ray diffraction photographs usually indicated one or at most a few β-rhombohedral crystallites per spheroid.

The overall purity of the product was improved by passage through the plasma, some of the more volatile foreign atoms having been removed. X-ray diffraction patterns from powder samples of the product material indicated its improved crystallinity and a predominance of the β-rhombohedral polymorph. However, a few faint lines foreign to that pattern were also present.

Careful microscopic examination revealed a small number of beautifully faceted crystals of four distinctive habits, each approximately 50 μm in diameter, within the bulk of the product (Fig. 1). X-ray diffraction photographs showed these specimens to be very well-ordered internally. When all of these distinctive specimens had been handpicked from a typical portion of product, the remainder gave a typical powder pattern for β-rhombohedral boron without extraneous lines.

We determined the densities of these well-formed crystals with a density-gradient column, using specimens of varied, but known, isotope ratios (assuming that no change occurred as a result of passage through the plasma). The density, adjusted to the normal isotope ratio of boron (*9*), was 2.367 ± 0.002 g cm^{-3} for each type of crystal.

Single-crystal x-ray diffraction techniques indicated that crystals of type (*a*) are simple tetragonal with lattice parameters $a = 10.061 \pm 0.005$ Å, $c = 14.210 \pm 0.005$ Å. The axial ratio obtained from optical observations ($c/a = 1.401 \pm 0.001$) is in good agreement with these values. Systematic absences in the diffraction patterns of these crystals limit their possible space groups to $P4_12_12$ (D_4^4) or $P4_32_12$ (D_4^8). The lattice parameters approximate those for a structurally unelucidated boron modification previously prepared only in the microcrystalline state (*2*); a powder pattern from our sample is in substantial agreement with that of this modification. The lattice type and parameters are also similar to those for α-AlB_{12} (*10*) and a form of SiB_6 (*11*) whose structures are unknown, although