steady inward flux. If most of the Ca^{2+} signal were due to a sudden surge at the beginning of the SAP, the arsenazo signal should not show this steady rise.
Furthermore, the time constant of the membrane ($\cong$ 4 msec at rest) is much too small to account for the SAP duration. Since the SAP is still observed in Na-free solutions and requires external Ca^{2+} (fig. 1), the increase in the arsenazo signal occurring during the long lasting depolarization is most probably due to the Ca^{2+} entering the cell rather than to Ca^{2+} released from cytoplasmic pools secondary to depolarization. Thus a more precise characterization of the Ca channels and of the mechanisms underlying their inactivation may be expected from the use of the arsenazo dye. The kinetics of Ca^{2+} entry and Ca^{2+} removal obtained from the arsenazo signal will be especially useful for studying whether internal Ca^{2+} concentration affects Ca conductance on this preparation as it does in other preparations[9,11].

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Nervous and behavioral responses to light in colonies of the free-living bryozoan, *Selenaria maculata* (Busk)[1]

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Summary. Bursts of electrical impulses which spread throughout the entire colony were recorded from individual zooids of *Selenaria*. Strong illumination caused a rapid increase in the frequency of bursts and also caused the colonies to start moving, usually towards the light. The recorded electrical activity may act to trigger locomotor behavior.

Bryozoa are generally small, colonial, sessile animals commonly found encrusting as flat sheets on rocks or seaweed[2]. Adjacent zooids in the colony are interconnected, and in some species at least there appears to be a colonial nervous system[3-5]. Thorpe et al.[6,7] recorded nerve impulses travelling across the colony of *Membranipora membranacea* (L.) in response to electrical or mechanical stimulation of the frontal membrane of a single zooid. The impulses were recorded from the surface of the colony and were found to influence retraction of the lophophores (ciliated food-collecting tentacles).
In contrast to these simple, sessile colonies, the family Selenariidae (Cheilostomata, Anasca) includes free-living species which show some remarkable behavior patterns[8,9]. The conical colony of *S. maculata* (Busk.) is illustrated in figure 1. The first reported observations of living *Selenaria* were made by Cook and Chimonides[8,9] who found that the peripheral avicularian setae (which in other Bryozoa act to clean the colony surface of detritus) can raise and support the colony, and propel it over the substratum at rates of up to 1 m/h. Colonies move laterally in a random manner except when strongly illuminated from one side when they move towards the light[8,9]. We have used electrophysiological techniques to investigate the neural basis of locomotion in *Selenaria*, and the response to light.
Methods. Colonies of *S. maculata* were collected from near Townsville (Queensland, Australia) and flown to Swansea where they were maintained in synthetic seawater (Instant Ocean) at 26 °C. Glass-tipped suction electrodes of about 100 µm diameter were used to record from the frontal membrane of individual zooids, and the electrical activity was amplified, displayed on a storage oscilloscope (Tektronix 5111) and photographed with a Polaroid camera. Strong illumination was provided by a 3 kW tungsten lamp, the light first passing through water to absorb the heat. The level of illumination was varied between 1.3 and 42 W/m^2 for wavelengths of 400–700 nm (behavioral responses[9] are produced by wavelengths of 400–560 nm).
Results and discussion. Under normal laboratory lighting (about 1.3 W/m^2) colonies usually showed no locomotion and little movement of the setae. Recordings from individual zooids showed bursts of impulses (fig. 2) occurring at irregular intervals of at least 1 min and often more than 10 min. In confirmation of the studies by Cook and Chimonides[8,9] increased irradiance (up to 42 W/m^2) initiated or increased setal movements within 2 min and colonies were observed to move (usually towards the light) within 10 min. Increased light intensity produced a rapid increase in frequency of bursts which persisted for the duration of the illumination and then returned to previous levels within

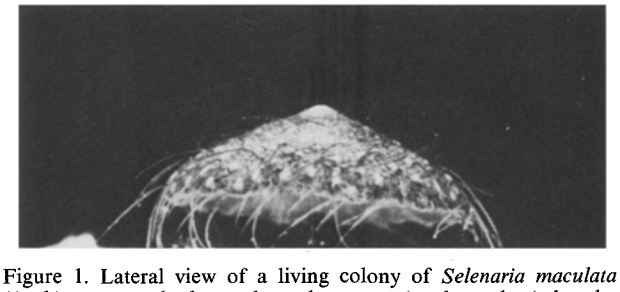

Figure 1. Lateral view of a living colony of *Selenaria maculata* (Busk) supported above the substratum (a glass plate) by the peripheral and subperipheral avicularian setae. Diameter of colony, 15 mm. (Photograph courtesy of Mr P.J. Chimonides).

a few sec (fig. 2, A). Each burst contained 2–18 impulses, this number tending to increase with increasing light intensity. Impulses could be recorded from any zooid and were found to spread throughout the whole colony. A burst of spikes at any point in the colony was accompanied by a burst at any other point (fig. 2, B); spikes often showed a 1:1 correspondence (fig. 2, C and D) but at times became desynchronized. At least 3 different spike amplitudes were observed, each spike spreading over the whole colony; bursts could be composed of single-amplitude spikes (fig. 2, C) or a mixture of 2 or more amplitudes (fig. 2, D). All 3 types of spike were increased in frequency by light. Conduction velocities of about 40 cm/sec were found by initiating impulses with electrical stimulation of single zooids and measuring the latency of response at known distances from the point of stimulation.

If the colony was split into fragments, bursts of impulses still occurred in pieces containing as few as 16 zooids, though their frequency was reduced, and bursts in different pieces were now out of register. Bursts were still increased in frequency by light, but there was usually a cessation of response if illumination continued for more than about 5 min; after a few minutes at low light intensity, however, transient responses were again obtained. This indicates that there is not a single, light-sensitive pacemaker for the colony. Periodic changes in the phase relationship between spikes recorded at different positions in the intact colony also suggests that the site of origin of impulse activity is variable.

It might be expected that the nervous activity responsible for coordinating locomotion would spread throughout the entire colony and would be sensitive to light. The recorded impulses have these properties but are evidently not directly responsible for setal movements, which they may precede by several minutes. The impulses did not appear to be associated with contraction of the lophophores, as in *Membranipora*[6, 7], and may act as an initiating ('command' or 'arousal') system. Electrical stimuli applied to individual zooids in dim lighting initiated bursts of such impulses and also produced prolonged, colony-wide setal movements.

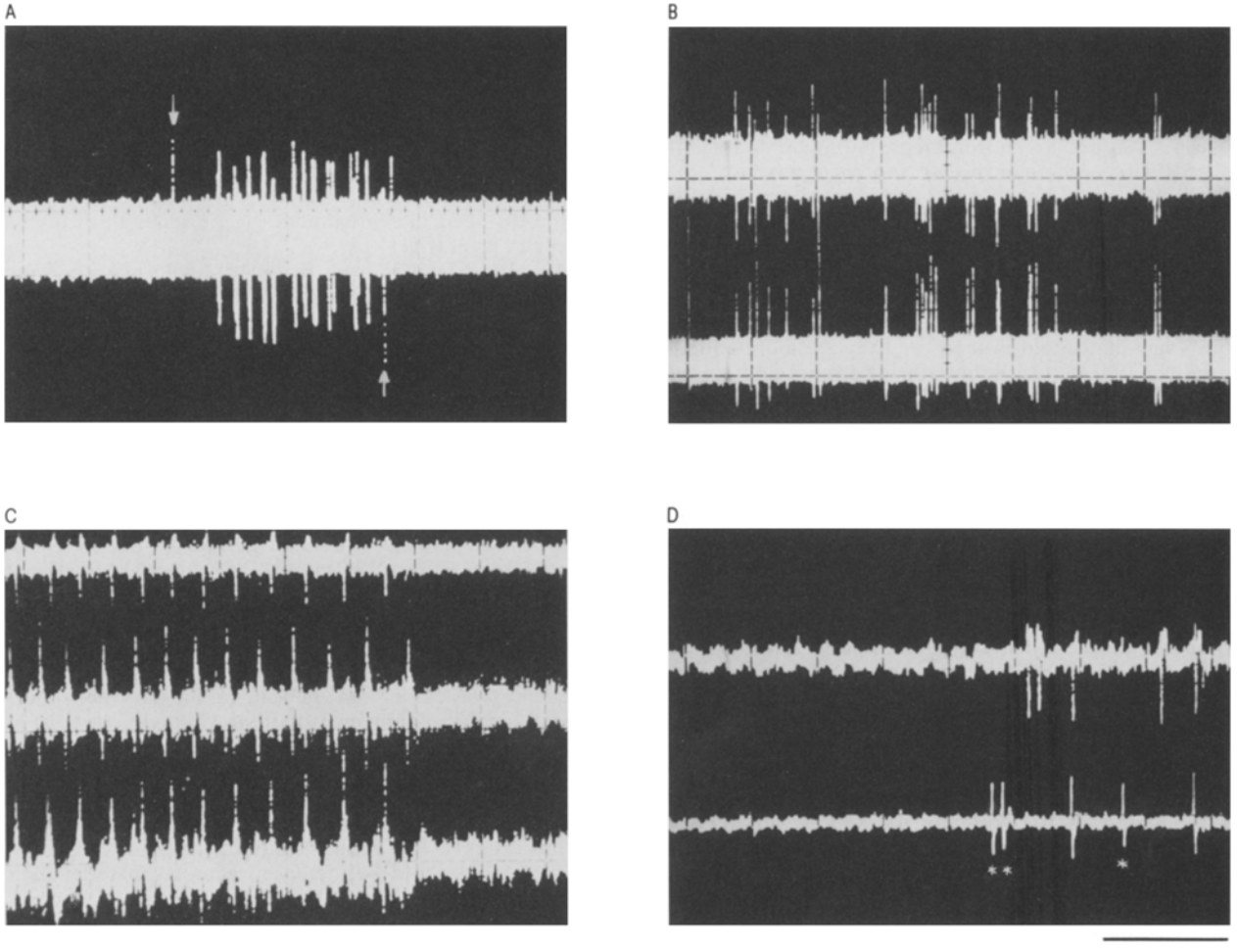

Figure 2. Examples of electrical activity recorded from the frontal membranes of individual zooids of *Selenaria* (4 different preparations). A and B were recorded at low sweep speeds and show bursts of impulses (each 'spike' may contain up to 18 impulses). *A* Effect of light. The colony produced bursts of impulses at very low rates (about 1 per 8 min) until strongly illuminated (34 W/m^2, arrow). When the light was switched off (2nd arrow) the colony quickly resumed its low firing rate. *B* Simultaneous recordings from opposite edges of a colony to show synchrony of bursts (light intensity 42 W/m^2). Similar bursts could be recorded from any point on the colony. *C* Simultaneous recordings at faster sweep speed from the middle (lower trace) and opposite edges of a colony to show spike composition of a burst. The sweep was triggered by the 1st impulse (not shown) on the lower trace. Note the constant amplitude of impulses and their 1:1 correspondence (firing rate was about 40/sec). *D* Simultaneous recordings from opposite edges of a colony showing a burst containing 2 types of spike. Spikes marked with asterisks on lower trace were followed with a latency of about 30 msec by spikes on upper trace. The 2 other, larger spikes occurred almost simultaneously with spikes on upper trace. Simultaneous recordings were essential for distinguishing different spike types in preparations where their amplitudes were similar. Time scale: A, 25 sec; B, 4 sec; C and D, 100 msec.

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Neuritic plaque-like structures in the rat cerebellum following prolonged alcohol consumption[1]

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Summary. Primitive neuritic plaques were observed in the inner third of the molecular layer of the cerebellar cortex of rats following chronic alcohol consumption. Neurites were identified as dystrophic parallel fiber boutons. Amyloid material dispersed among neurites was not clearly recognized, dystrophic some fibrils were frequently seen among them. Astrocytic processes were noted in the periphery of the plaque. Microglial reaction, however, was non-existent. The rarity of these lesions in the rat cerebellum and their probable relation to long periods of alcohol consumption is discussed.

Neuritic plaques are a common finding in brains from patients with Alzheimer disease and aged persons[2,3]. Similar lesions have been observed in old dogs[4] and monkeys[5] and, after experimental induction, in a few other animal species[6-8]. It appears that neuritic plaques are rarely present in animals[9,10]; the most careful searches have been unable to detect their presence in many animal species[11]. It would seem that, for unknown reasons, these lesions are conspicuously age-dependent in man[9,10]. They are more abundant in the cerebral cortex, mainly in the frontal and temporal lobes, in the hippocampus and the amygdaloid nucleus[12], and infrequent in the cerebellum. The majority of plaques observed in the latter are morphologically different from those found in the cerebral cortex[13] and are probably related to forms of generalized amyloid angiopathy[14]. Classic neuritic plaques, however, have been described in the cerebellum of patients with familiar forms of Alzheimer disease[13], in aged patients with Down syndrome and in some cases of cerebellar atrophy in chronic alcoholics[15].

The purpose of this report is to describe the presence of primitive neuritic plaques in the cerebellum of rats following prolonged alcohol treatment; plaques which are probably related to the widespread alcohol-induced cerebellar deterioration.

Material and methods. 8-week-old male Sprague Dawley rats, weighing 200-220 g, were separated into 10 different groups of 6 animals each. Half of the groups were alcohol-fed for periods of 1, 3, 6, 12 and 18 months, and the others used as controls for the same periods.

Ethanol-treated animals were given unrestricted access to a 20% aqueous ethanol solution as the only available source of liquid. Food and fluid intake were measured every other day, and the amounts consumed were calculated for the alcohol-fed animals. Controls were given the same amounts of food and fluid, with sucrose isocalorically replacing ethanol. Details of this procedure have been described elsewhere[16].

The methods described by Palay and Chan-Palay[17] were used for fixation of the nervous system. Tissue blocks from the cerebellar vermal lobules 4-6 were embedded in epon[18]. Semi-thick sections were stained with toluidine blue and ultra-thin sections double-stained with uranyl acetate and lead citrate.

Results and discussion. In semi-thick sections stained with toluidine blue, aggregates of rods and dots, probably corresponding to abnormal neurites were often seen, after 12 months of alcohol treatment, in the inner third of the molecular layer (fig. 1).

At the ultrastructural level scattered single neurites were observed in all cerebellar cortical layers after 3 months of alcohol treatment (5-month-old animals). These neurites were seen in all sections studied; their number however, was greater in the molecular layer (fig. 2). Following 6 months of alcohol consumption (8-month-old animals), neuritic plaques of the primitive type[19] could be seen in the inner third of the molecular layer (fig. 3). This location is similar to that of the plaques described in the cerebellum of chronic alcoholics[15]. The number and complexity of the plaques are dependent on the duration of the experiment, Plaques with diameters between 20 and 30 µm were frequently seen after 12 months of alcohol treatment (14-month-old rats).

Plaques were formed by a variable number of neurites which were markedly distended by an accumulation of dense and lamellar bodies, lipofuscin granules and tubulovesicular profiles. Paired helical filaments were never noted, which is in agreement with previous descriptions in which it is affirmed that in the central nervous system these do not exist in animals[3,9,20]. However, it must be stressed that paired helical filaments have been demonstrated in neurons of the spinal ganglia in rats after chronic alcohol administration[21]. The careful study of the neurites showed that almost all could be identified as being parallel fiber boutons. In spite of the existing dystrophy, synaptic specializations were easily identifiable, as well as the Purkinje cell spines with which they maintained synaptic contacts (fig. 3). The presynaptic origin of neurites from plaques is in keeping with previous descriptions[22]. Dendrites and their spines in the proximity of the plaque seemed to be well-preserved, as opposed to those described in the cerebral cortex of patients with Alzheimer disease[23,24].

Although a central core of amyloid was never observed, filamentous material randomly oriented and dispersed among neurites, and whose size resembled amyloid filaments, was frequently seen (fig. 3). A similar finding has been reported by Terry and Wisniewsky[19] who showed that