Corneal thickness also showed little change when the endothelium was bathed in solutions of differing sodium concentrations.

These experiments reveal a direct relationship between the rate of transport of sodium ions and corneal thickness: as the rate of sodium transport decreases, corneal thickness increases. It was suggested from the previous work involving the use of applied voltages[3] that the rate of transport of sodium ions controls the sodium content of the stroma (which is primarily an anionic mucopolysaccharide) and in this way exerts a control on corneal hydration. The results reported here show that such a relationship between sodium ion transport and corneal hydration exists even without the application of extraneous (for example, electrical) forces. The transport–hydration relationship is physiologically fundamental to the control of corneal hydration and thickness. These findings also substantiate the role of the epithelium in the maintenance of corneal thickness.

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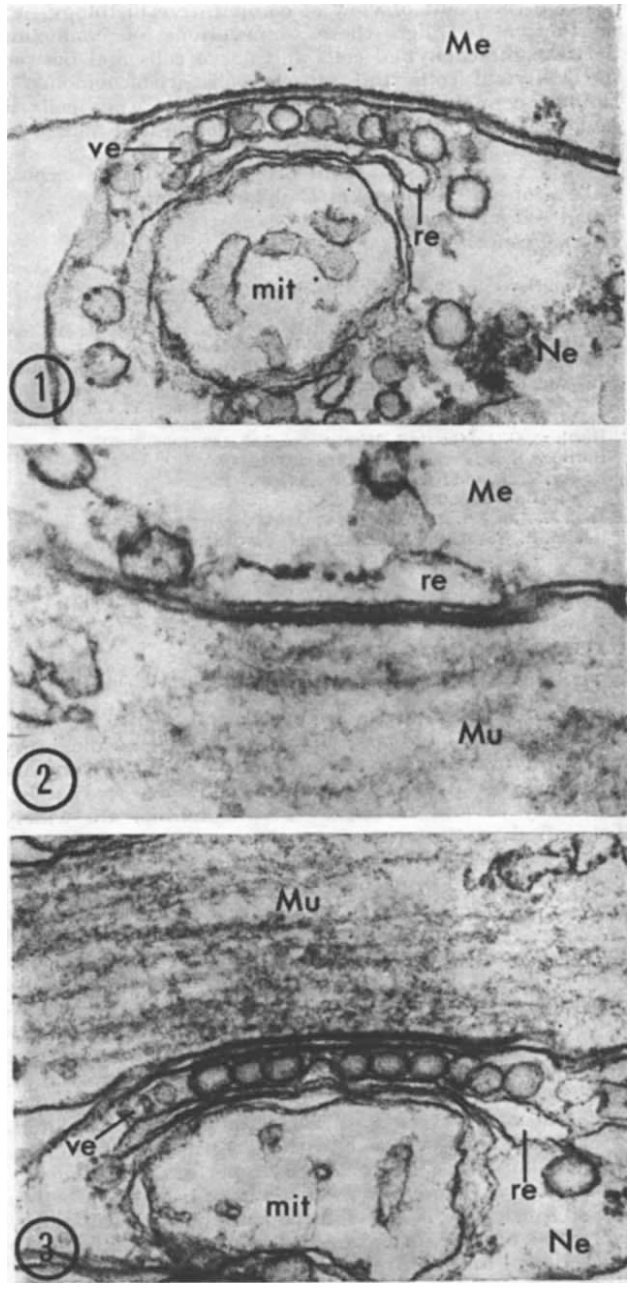
Specialized Connexions between Nerve Cells and Mesenchymal Cells in Ctenophores

THE mesogloea in ctenophores, as in certain coelenterates[1,2], is essentially a third cell layer containing muscle cells, nerve cells and so-called "amoeboid" cells[3,4]. Our unpublished studies on *Beroe* suggest that the "amoeboid" cells do indeed undergo a migratory phase during regeneration, but that there is no evidence of migration in the normal animal. Histological preparations show the cells with very long, thin processes connected together in the form of a net. We prefer to call them "mesenchymal" cells. We now have evidence from electron microscopy of specialized junctions between these mesenchymal cells and neighbouring muscle and nerve cells in *Beroe forskälii* and *B. ovata*.

At the neuro-mesenchymal cell junction the membranes are in close contact—separated by a gap of 130–150 Å—and show an obvious, densely staining thickening (Fig. 1). The nervous side of the junction is characterized by a row of vesicles tightly packed between the cell membrane and a sac of smooth endoplasmic reticulum, itself flattened against a single, huge mitochondrion (Fig. 1). The diameter of the vesicles ranges from 300 to 900 Å, with an average of 660 Å, which slightly exceeds the average size of "synaptic vesicles" of higher animals. After osmic fixation followed by block-staining with phosphotungstic acid (PTA) and section-staining with uranyl acetate and lead citrate, the vesicles look clear or are unevenly filled with a grey, amorphous mass. After fixation with glutaraldehyde, however, most of them show an electron-dense core of variable diameter.

Ultrastructural specializations such as those just described are also found at interneural, neuro-muscular (Fig. 3), neuro-glandular and neuro-sensory junctions in Beroids, Cydippids and Lobates (my unpublished results) and they correspond in essential features to the structures earlier described by Horridge[5,6] as synapses. If it is correct to regard them as such (and we assume that it is), it seems that the junctions between nerve cells and mesenchymal cells are also synapses, which implies that mesenchymal cells are effectors. It is noteworthy that mesenchymal cells receive large numbers of such synapses, in some cases receiving more than adjacent muscle cells. A single neurite may make synaptic connexions with both muscle and mesenchymal cells, which suggests that the two effectors could be excited at the same time.

In the absence of physiological evidence it would be inappropriate to speculate about the spread of excitation within or beyond the mesenchymal cell, or about possible effector action resulting from such excitation. It is of



Figs. 1–3. Electron micrographs from tangential sections of *Beroe forskälii* below the lip. Tissue fixed with OsO_4, buffered according to the method of Palade; embedded in 'Epon'; block stained by PTA; sections stained with uranyl acetate followed by lead citrate. 1, Neuro-mesenchymal junction (×59,000). 2, Junction between a mesenchymal cell and a muscular fibre (×82,000). 3, Neuromuscular junction (×59,000). *Me*, Mesenchymal cell; *mit*, mitochondrion; *Mu*, muscle cell; *Ne*, neurite; *re*, endoplasmic reticulum; *ve*, synaptic vesicles.

interest that specialized junctions of a distinct type occur between mesenchymal cells and muscle cells (Fig. 2) and between the processes of contiguous mesenchymal cells. These junctions show thickening and close approximation of the membranes (Fig. 2), but there is no accumulation of vesicles in the vicinity and the configuration is thus a symmetrical one. In junctions between mesenchymal cells and muscle cells a sac of endoplasmic reticulum does occur within the mesenchymal cell in direct apposition to the plasma membrane (Fig. 2). There is no evidence of membrane fusion and the junctions cannot be regarded as a type of "nexus"[7]. This does not, however, preclude the possibility that the junctions are somehow involved in information transfer between the cells in question.

From the point of view of comparative histology, it is instructive to align these observations of connexions between mesenchymal cells and nerve cells and between mesenchymal cells and muscle cells with evidence of similar connexions involving glio-interstitial cells in molluscs[8] and interstitial cells in rodents[9] and man[10].

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Prostaglandins and Toad Spinal Cord Responses

PROSTAGLANDINS have a wide distribution in the vertebrate body and have been shown to be pharmacologically active on many types of vertebrate tissues. Evidence that these compounds have a neurohumoral role is suggested by several findings. Prostaglandins are widely distributed in the central nervous system[1], and in the rat cerebral cortex they are localized in synaptic vesicles[2]. They are released from the feline cortex[3] and possibly cerebellum and ventricles[4,5]. In addition, prostaglandins have effects on the central nervous system of cats and chickens[6,7]. We decided to investigate the effects of prostaglandins on the isolated toad spinal cord because the preparation is convenient for studying direct actions of drugs on a neuronal pool. Furthermore, prostaglandins are released from the frog spinal cord on stimulation of the hind limbs[8], suggesting that they are involved in synaptic transmission in amphibians.

Spinal cords of the toad *Bufo marinus* were isolated, hemisected and perfused with oxygenated amphibian Ringer solution. Experiments were performed during the Australian summer. The methods have been described in detail elsewhere[9,10]. The eighth dorsal root was stimulated every 10 s and a.c. or d.c. recordings were made from the ninth dorsal and ventral roots using Ag/AgCl electrodes. A d.c. preamplifier and 'Grass Polygraph' were used to record changes in potential level. Prostaglandins PGE_1, $PGF_{1\alpha}$ and $PGF_{2\alpha}$ (supplied by Dr J. E. Pike) were prepared as 10 mg/ml. solutions in 95 per cent ethanol. Stoichiometric conversion to the sodium salt was effected by adding 0·9 ml. of a 0·2 mg/ml. solution of sodium carbonate for each 1 mg of prostaglandin. Immediately before application the stock solutions were diluted to an appropriate concentration with amphibian Ringer solution. L-Glutamic acid was applied regularly to the preparation in order to assess its level of excitability.

The three prostaglandins, PGE_1, $PGF_{1\alpha}$ and $PGF_{2\alpha}$, all produced distinct changes in reflexes and polarization level when perfused over the toad spinal cord for periods of 0·5–2 min. Simultaneous d.c. records from dorsal and ventral roots were comparable except that drug-induced effects recorded from the dorsal root were more pronounced than those from the ventral root.

Typically, the prostaglandins caused a slowly developing, long-lasting depolarization of both dorsal and ventral roots. During the application of PGE_1, this was frequently preceded by a hyperpolarization of both roots. This is illustrated in Fig. 1*A*. Two minutes before the application of PGE_1, the spinal cord was perfused for 30 s with L-glutamic acid (5×10^{-5} g/ml.) which induced a rapid depolarization with a simultaneous reduction in size of the dorsal and ventral root potentials. Recovery was complete within 1 min of termination of the glutamate application. PGE_1 (5×10^{-5} g/ml.) caused a hyperpolarization during its 50 s application, followed by a depolarization which lasted for several minutes. The dorsal root polarization level had not reached control value 20 min after the application. During the hyperpolarizing phase of the response, polysynaptic reflex discharges in the ventral root were markedly reduced. Tracings *B* and *C* in Fig. 1 were recorded from the same preparation. $PGF_{1\alpha}$ and $PGF_{2\alpha}$ induced a slowly developing and long-lasting depolarization which began during the application.

PGE_1 had the most pronounced effects, usually causing a greater depolarization than L-glutamate applied in the same concentration. $PGF_{1\alpha}$ was usually more potent than $PGF_{2\alpha}$. Desensitization occurred with repeated applications. Thus, although the first application of a prostaglandin always had a significant effect, a second or third application a few minutes afterwards had little or no effect. On washing for 0·5–1 h, the prostaglandin again had a significant effect.

The results of this investigation, together with those of Ramwell *et al.*[8], suggest that prostaglandins have a

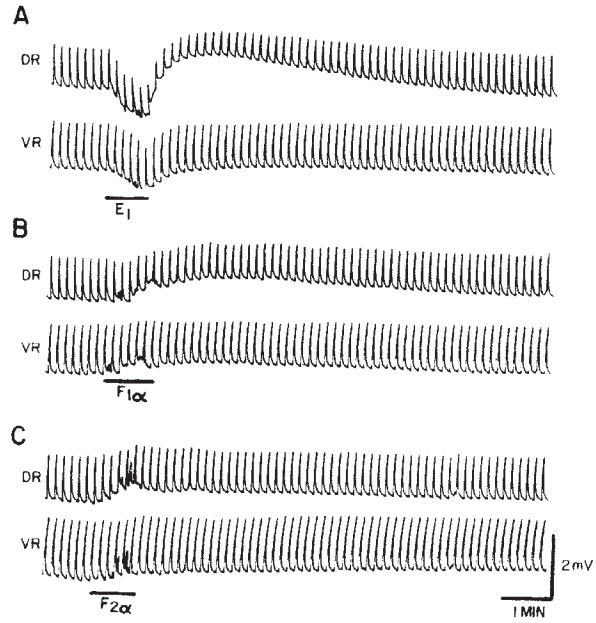

Fig. 1. d.c. Recording (negativity upwards) from dorsal and ventral roots of isolated hemisected toad spinal cord. *A*, *B* and *C* illustrate effects of three different prostaglandins on the same preparation, each applied in a concentration of 5×10^{-5} g/ml. Each pair of tracings represents simultaneous recordings from the ninth dorsal and ventral roots. The eighth dorsal root was stimulated at intervals of 10 s. Temperature was maintained constant at 20° C.