EFFECT OF CERTAIN DRUGS ON THE ODONTOBLAST MEMBRANE POTENTIAL*

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Two physiological functions which are known to occur within teeth are the deposition of calcium salts within the organic matrix and the reception of irritant processes as pain. A cell, which, from an anatomical as well as a morphological viewpoint, could subserve either one or both of these physiological activities is the odontoblast. With the cell bodies lining the exterior of the pulp cavity, the odontoblasts send out long protoplasmic processes (Tome's fibers) through dentinal tubules to the dentino-enamel junction. Some workers have suggested that sensory function is mediated by the odontoblast[1,2], however, the majority opinion is that these cells are concerned only with dentin formation, and that sensory function is mediated by microneural filaments which extend through the dentin to the sensitive dentino-enamel junction (D.E.J.) or the dentino-cemental junction (D.C.J.).[3,4] To date there have been no studies regarding the physiological properties of the odontoblast to ascertain if this cell has properties which one would predict or expect for a sensory receptor or a glandular cell.

This is a report of the measurement of membrane potentials, using ultramicroelectrode techniques, of odontoblasts both in tissue culture (mouse) (Fig. 1a) and in the isolated mandibular preparation of the rat (Fig. 1b). The influence of certain labilizer and stabilizer drugs upon this potential as well as upon induced spike potentials in the isolated mandibular nerve have also been studied.

Because Avery and Rapp[1] speculated on the sensory role of the odontoblast largely on the basis of finding considerable quantities of cholinesterase both in the soma membrane as well as the Tome's fibers of human teeth, it was first necessary to determine if the cells used in this study possess this enzyme. Using the myristoyl choline technique of Gomori[5], Fig. 2 shows that the rat incisor odontoblasts have this enzyme. Because of low cell density of the mouse odontoblast in tissue

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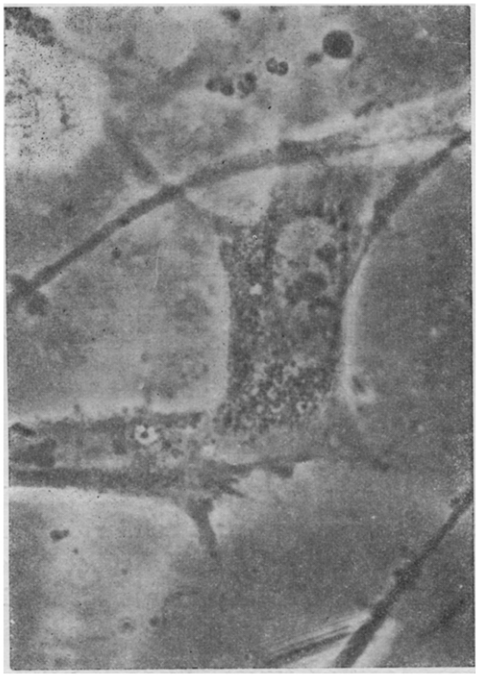

FIG. 1a. Phase micrograph of living polygonal cells (odontoblasts) in a 14-day old culture of molar tooth germ dental papilla from a 1-day old mouse. Only the initial segment of the protoplasmic process, Tome's fiber, is visible in the photograph. × 333.

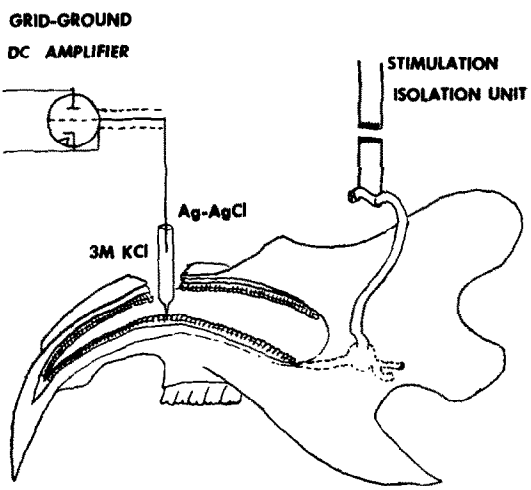

FIG. 1b. Schematic diagram of the *in vitro* rat mandible preparation with stimulation and recording instrumentation.

culture, it is not possible to state that these cells possess the enzyme; however, some indication of pigmentation has been observed.

Using 3M KCl-filled ultramicroelectrodes, 10–40 MΩ resistance, connected to a d.c. preamplifier (Grass P6 with M.E.P.-6 probe), it has been shown that the membrane potential of the odontoblast in tissue culture or in the *in vitro* rat mandible preparation possess a resting membrane potential of 35–40 mV[6,7]. Stimulation of the peripheral end

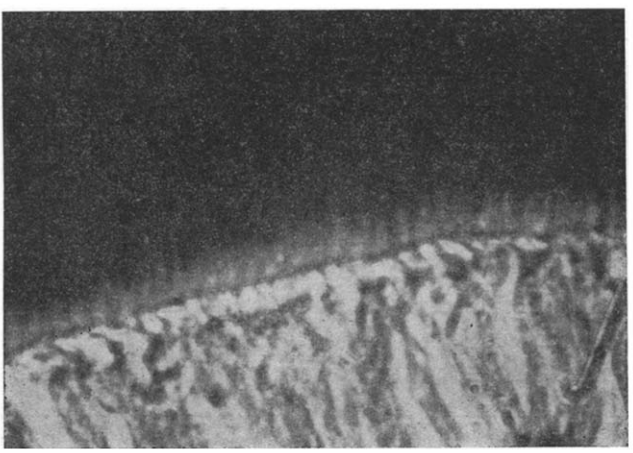

FIG. 2. Photomicrograph showing the presence of non-specific cholinesterase in the odontoblastic layer of the mandibular inscisor toth of the rat. Both the cell bodies and the fibers in the dentinal tubules exhibit enzyme. × 1000.

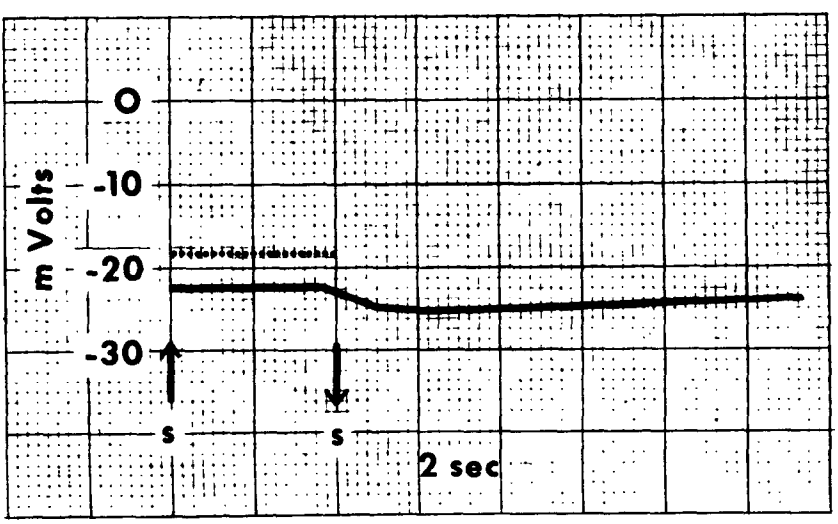

FIG. 3. Oscilloscopic trace of the membrane potential obtained from an odontoblast in the rat mandibular inscisor showing the effect of mandibular nerve stimulation (modified).

of the mandibular nerve in rats or the application of acetylcholine (10^{-7} moles) to the tissue culture perfusion medium causes a slight increase of the membrane potential of approximately 3–4 mV lasting 15–25 sec (Fig. 3). Epinephrine, histamine, and 5-hydroxytryptamine have not been observed to cause this effect. Atropine (0·01%) appears to prevent this transient hyperpolarization of the cells in tissue culture, while the local anesthetic, procaine (0·01%) blocks the change both in the tissue culture and the rat mandibular preparation.

Electrical stimulation applied to the exposed pulp is capable of inducing spike potentials in the mandibular nerve, but as yet concurrent change in the membrane potential of the odontoblast, from which we were recording, have not been observed.

The slight increase in membrane potential of the odontoblastic membrane with stimulation of the mandibular nerve or adding acetylcholine to cells in tissue culture would not seem to be sufficient to be classified as a hyperpolarization phenomenon seen with certain other cells such as those reported by Lundberg for the salivary glands[8]. Since the extent of hyperpolarization is largely dependent upon the existing membrane potentials[9], further work is needed to clarify this observation.

The inability to obtain any alteration of the membrane potential of the odontoblast with the formation of spike potentials in the mandibnular nerve would seem to indicate that this cell plays no part in the mediation of pain impulses. There are, however, several technical reasons which could account for this negative result, such as poor distribution of the drug to the impaled cell and the ability to study only a limited number of odontoblasts which may or may not be physiologically connected to the fibres showing excitation.

It is not possible from these studies to determine the physiological role of these pulpal cells. The moderately high membrane potentials recorded for the polygonal cells, odontoblasts, indicate that they might be classfied with such cells as smooth muscle[10] and glandular epithelium[8]. The anatomic position of the odontoblast, the presence of cholinesterase in its membrane, and a moderately elevated membrane potential is suggestive of some physiologic role which remains to be clarified.

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