Change of spike response induced by Refractive Error
Average for refactive errors of plus and minus 1 diopter

Example No.	Field type	Spike change per diopter	Response change per diopter (%)[a]	Example No.	Field type	Spike change per diopter	Response change per diopter (%)
1	on	1.5	−15	12	off	0.6	−22
2	on	2.0	+28	13	on	10.0	−76
3	on-	0	0	14	off[e]	1.7	−16
	off[b]	4.0	−27				
4	on-	0	0	15	on-	0	0
	off[c]	2.7	−17		off	0	0
5	off	2.0	−28	16	off[d]	8.5	−30
6	on-	0	0	17	on-	5.0	−40
	off	1.0	−25		off	0	0
7	on-	0	0	18	on-	0	0
	off	3.5	−37		off	2.5	−21
8	off[d]	2.3	−24	19	on-	2.5	−13
					off[f]	0	0
9	on-	0	0	20	on-	1.2	−17
	off[b]	0.7	−23		off	0.8	−16
10	off[d]	1.8	−20				
11	off	7.0	−58				

[a] A (+) sign indicates an increase in spike frequency, a (−) sign indicates a decrease. [b] These fields were giving on-off responses uniformally throughout. [c] This field had an on-off center and an off surround. [d] Response to an encroaching black edge only. [e] This field had an off center and an on-off surround. [f] This field had an on-off center and an off surround.

recruiting effect on the near-by surround; most commoly, however (all other cases shown in the Table) the opposite is true, suggesting that surround inhibition is being activated by the enlarging blur circle, in combination with a less strongly stimulated center area.

Dioptric degradation of the retinal image then can have a significant detrimental effect on the responsiveness of individual visual pathway neurons and such effects can vary over a wide range depending on the particular cell. Also, it should be noted that on and off response mechanisms of a cell can lose their response efficiencies independently of one another, suggesting controlled blur as one possible means of isolating the responses of the two, and perhaps more clearly identifying the individual missions of each.

Zusammenfassung. Bestimmung der Auswirkung von artefiziellen Refraktionsanomalien auf die Aktivität von Neuren in primären Sehzentren.

J. M. LEE and R. M. HILL[3]

College of Optometry, The Ohio State University, Columbus (Ohio 43210, USA),
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Electrical Responses of Cardiac Muscle in Na-Free High-Ca Solution

Frog atrial muscle immersed in Na-free high Ca solution relaxes completely after a transient contracture and then responds to electrical stimuli with strong twitches, which may be 'all or none' (BOZLER[1]). In the present work the changes in the membrane potential associated with these twitches were recorded using glass microelectrodes. Modified Ringer solution contained in m*M*: NaCl 115; KCl 3; $CaCl_2$ 1.5; *Tris* chloride (pH 7.2) 2. In Na-free high-Ca solution, all Na was replaced by 83 m*M* Ca. Rings of the atrium of the frog (*Rana pipiens*) were used.

In the Na-free High-Ca solution conducted action potentials could be obtained, most readily after epinephrine ($5.10^{-6}M$) was added. These action potentials have a large overshoot (membrane potential reversal), reaching an amplitude of 65 to 90 mV, as compared to 25–30 mV in Ringer solution. Including the resting potential, which was increased by the solution used, the total depolarization was as large as 185 mV. The potentials were not influenced by TTX (10^{-7} g/ml) and their duration was about half of that in Ringer solution. In the absence of epinephrine responses were generally local. If they were conducted, the drug increased the amplitude and the duration of the plateau.

These results strongly support views regarding the role of Ca in cardiac activity based on recent electro physiological studies. Voltage clamp experiments have indicated an influx of Ca during the cardiac action potential (REUTER[2,3], ROUGIER et al.[4]), although these results have been questioned by JOHNSON and LIEBERMAN[5]. The action potentials in Na-free solution reported here clearly demonstrate an influx of Ca during responses of cardiac muscle and show that the influx can be strong enough for con-

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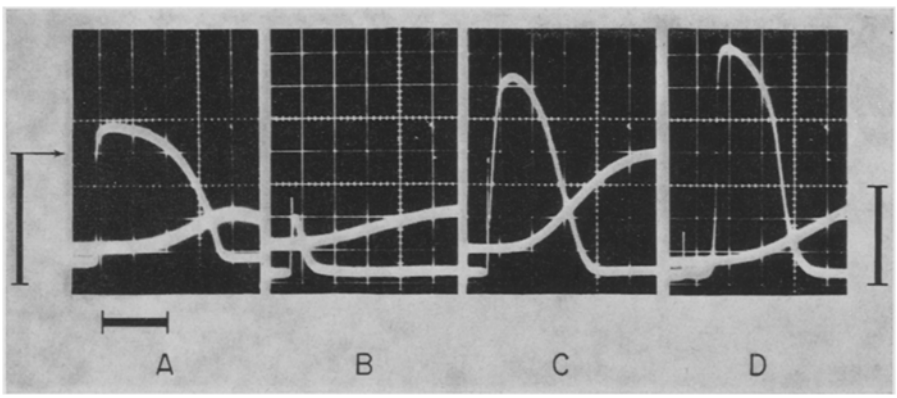

Electrical and mechanical responses of frog atrium in Na-free high Ca (83 mM) solution in presence of epinephrine ($5\times10^{-6}M$) and TTX (10^{-7} g/ml). A) Control in Ringer *tris* solution; B) and C) local responses; D) conducted response. O· reference is indicated by arrow; left vertical bar: 100 mV; right vertical bar 0.25 g; horizontal bar: 200 msec.

duction to take place. Furthermore, the fact that epinephrine increases the magnitude and duration of the potentials strongly supports the assumption that the drug increases P_{Ca} as concluded from voltage clamp experiments (REUTER[3], VASSORT et al.[6]).

The change in membrane potential during conduction permits conclusions to be drawn regarding the internal Ca^{++} concentration. If a maximal value of 90 mV for the overshoot is taken as the equilibrium potential of Ca, the internal concentration at the beginning of contraction, calculated from the Nernst equation, is $6.5\times10^{-5}M$. However, as it is unlikely that an equilibrium for Ca is reached, this value must be considered merely as a maximal value. Thus, the results suggests that intracellular Ca^{++} concentration remains low inspite of the high concentration in the medium.

Résumé. L'activité électrique de préparations atriales de grenouille soumises à un milieu de Ringer dont tout le Na^{+} est remplacé par du Ca^{++} a été enregistrée à l'aide de microélectrodes. Dans ces conditions, il a été possible d'obtenir des potentiels d'action propagés. Ces réponses sont caractérisées par une très grande inversion de potentiel dont l'amplitude, augmentée par la présence d'adrénaline dans le milieu, peut atteindre 90 mV pour une dépolarisation totale de 185 mV, du fait d'une hyperpolarisation de la membrane. Ces résultats démontrent clairement le rôle du Ca^{++} dans l'activité électrique cardiaque.

J. F. DELAHAYES[7]

Department of Physiology, Ohio State University, 333 West 10th Avenue, Columbus (Ohio 43210, USA),
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Vesicle Hypothesis: Effect of Nerve Stimulation on the Synaptic Vesicles of Motor Endplates

Ever since they were first described, the synaptic vesicles of axon terminals have been regarded as the morphological correlate of quantal transmitter release. This so-called 'vesicle hypothesis' has become almost axiomatic in the neuro-sciences.

To test the hypothesis, various attempts have been made to find whether the concentration of synaptic vesicles is altered by experimental conditions which influence either transmitter release or synthesis. The results seem somewhat confusing. Thus, some authors have reported decreased number of vesicles following stimulation[1–3], whereas others have found an increase[1,4–5]. Exposure to hemicholinium alone[6] has resulted in decreased number of vesicles, and hemicholinium combined with stimulation has caused a decrease in vesicles[4–5] or no change[7].

The present experiments were performed with a similar purpose to those just referred to. Distinctive features of our experiments are firstly that the stimulation of the nerve outlasted fixation of the muscle and the nerve terminals, and further, that the stimulation frequencies were so high as to put an exhaustive load on the presynaptic structures.

Methods. Phrenic nerve-diaphragm preparations from male 200 g albino rats were removed under ether anesthesia, fixed by threads to a glass fork, and placed horizontally in Tyrode solution[8] at 37 °C, carbogen bubbling through the solution. The phrenic nerve was stimulated by supramaximal square wave pulses of 0.1 ms width, contraction observed visually. Towards the end of the stimulation period the fork was carefully transferred to the surface of the fixing solution (2% paraformaldehyde + 2% glutaraldehyde in isotonic phosphate buffer[9], pH 7.3), the muscle being fixed from the abdominal side. In the course of 2–3 min, the diaphragm was lowered gradually into the fixation medium until it was completely

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