Bis jetzt gibt es noch keine gesicherten Hinweise dafür, dass diese Nervenzellen in der Darmwand die Darmmotorik steuern. Ferner fehlen noch Untersuchungen über eine motorische Innervation des Ösophagus. Die Ventrikularnerven enthalten Neuronen, die ebenfalls spontan aktiv sind[7].

Summary. Multiterminal nerve cells have been found in the foregut of crickets (*Acheta domesticus* (L.), *Gryllus campestris* L.) which are sensitive to stretching the wall of the gut. These cells are spontaneously active and they respond to stretch in a phasic-tonic way. Within the ventricular nerves there are also neurones being spontaneously active.

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[7] Die Untersuchungen werden durch ein Promotionsstipendium der Universität Köln und durch Mittel, welche die Deutsche Forschungsgemeinschaft Herrn Professor Dr. Huber zur Verfügung stellt, unterstützt.

Reactive Depolarization of Muscle Fibre Membrane with Slowly Increasing Middle-Frequency Current Flow[1]

KUMAZAWA[2–4], using intracellular recording at the site of stimulation, has shown that trains of sine-wave middle-frequency current (20 kc/sec) of variable strength and duration applied transversely to the curarized sartorius muscle of the frog evoke characteristic responses that may be summed up as follows: (1) The decrease of membrane potential, at first linear, towards the end of utilization time passes gradually into the upstroke of the conducted action potential. (2) The rate of the linear decrease of the membrane potential is not directly proportional to the voltage applied. The slope of the diminishing membrane potential is steeper than the slope obtained in direct relation. (3) Initiation of spike potential occurs as soon as a given degree of depolarization (approx. 20 mV) is reached. (4) Neither local decrease of membrane potential nor conducted action potential is observed if the sodium ions of the bathing fluid are replaced by choline ions. Both effects are fully reversible.

From these findings, it was concluded that the primary effect of middle-frequency current flow on the muscle fibre membrane cannot be explained on the basis of an electric depolarization process imposed on the membrane by an outward current flow. It is to all appearances due to some process initiated within the membrane, and has hence been qualified as 'reactive depolarization' (KUMAZAWA and WYSS[4]).

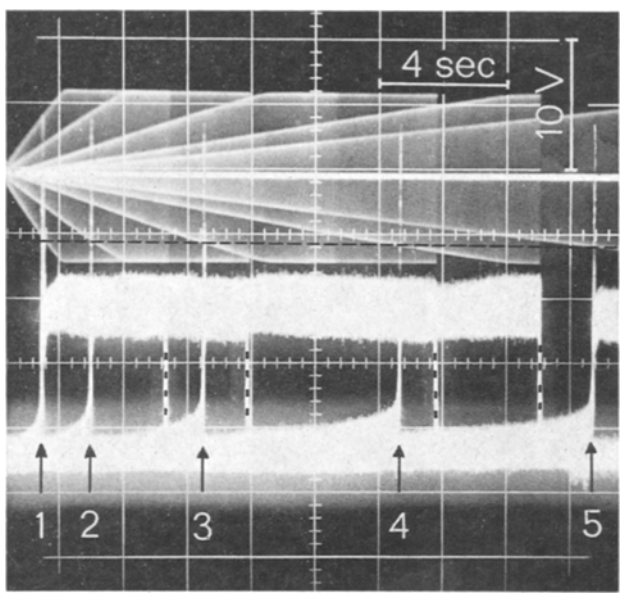

Fig. 1. Five stimuli records, each successive stimulus at lower rate of rise (upper beam). The 5 action potentials (1–5; lower beam) arise at the same stimulus threshold. The horizontal broken line connecting the 5 threshold values indicates that no accommodation occurs. (The vertical broken lines indicate the return of the membrane potential to its resting level after cessation of stimulus.)

The object of the present investigation was to study the non-proportional relation between reactive depolarization and current strength. Experimental conditions were similar to those previously used. The following modifications were, however, introduced: (a) The middle-frequency currents were applied at a slow, linearly rising rate, the rising-time being extended up to several minutes; (b) the frequency was reduced to 5 kc/sec; (c) the sartorius muscle was not curarized.

With slow linear increase of middle-frequency current flow, reactive depolarization developed after an initial period during which a possible elevation from the base line of resting potential was hardly recognizable (Figure 2A). The decrease of membrane potential passed gradually into the conducted spike, and hence resembled KUMAZAWA's results with middle-frequency current pulses. The threshold values, however, remained below 10–12 mV in the present experiments. The rate of reactive depolarization depended on the rate at which stimulus voltage increased. The spike arose as soon as stimulus reached 'stimulus threshold' and reactive depolarization 'membrane threshold' (Figure 1). Hence no accommodation was ever shown to slowly rising stimulus intensity.

The fact that the critical value of membrane threshold was below the critical value obtained by KUMAZAWA, may be due to the technical modification adopted in the present investigation.

If, after linear rise, voltage was kept at constant level before membrane threshold was attained, reactive depolarization continued up to 7 mV, associated, however, with slow fluctuations of, regular or irregular in type, duration ranging between 2–30 sec.

The effect of successively replacing sodium by choline was to modify the reactive depolarization. A reduction of membrane potential exceeding 10–12 mV now was required in order to reach 'membrane threshold'. Finally the critical reactive depolarization was no longer reached, and occasional abortive spikes replaced the full action

[1] Presented at the First Meeting of the Union of the Swiss Societies for Experimental Biology, Berne, 17 May, 1969.
[2] T. KUMAZAWA, Experientia *22*, 393–394 (1966).
[3] T. KUMAZAWA, Helv. physiol. Acta *26*, 257 (1968/69).
[4] T. KUMAZAWA und O. A. M. WYSS, Helv. physiol. Acta *24*, C3 (1966).

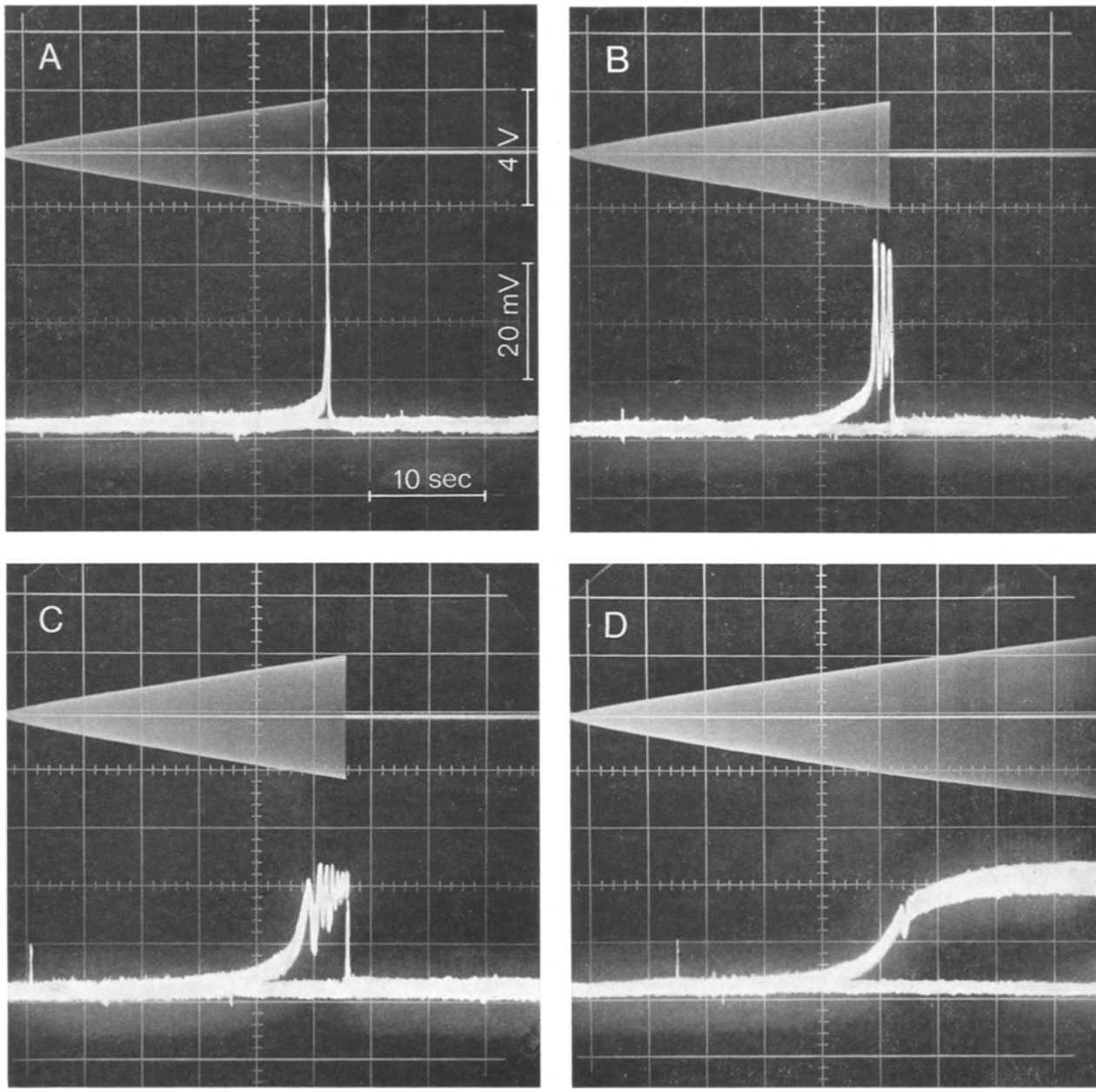

Fig. 2. Modification of the reactive depolarization to increasing middle-frequency current-flow after replacing sodium by choline. From (A) to (D): Upper beam: Each middle-frequency stimulus increases at the same linear rate. Lower beam: Intracellular-recorded membrane potential at high amplification (10 mV/div). Note that the stimulus mid-line does not represent the zero-level of membrane potential. The resting value of the latter remains at approximatively 90 mV throughout the experiment. (A) Mode of action of the reactive depolarization at onset of replacement of sodium by choline. (Continuous supply approximatively 30 drops/min. Bath solution content about 11.5 ml.) The reactive depolarization is followed by an ordinary action potential. (B) After 12 min; (C) after 18 min; (D) after 21 min. From (A) to (D), the membrane threshold rises above 10–12 mV, whereas the height of the responses falls.

potential. Further reduction of sodium concentration only led to initial progressive increase of depolarization, followed by decrease of slope, until a ceiling of up to 20 mV reduction of membrane potential was attained (Figure 2, B–D). This ceiling in spite of continuing current flow, showed a tendency to decrease, i.e., to spontaneous repolarization. With further reduction of sodium concentration, ceiling depolarization was also reduced.

All these findings confirm previous observations that reactive depolarization depends on extracellular sodium ions. Furthermore, there is strong evidence that the phenomenon represents a process elicited within the excitable membrane.

Zusammenfassung. Die Mittelfrequenz-Querreizung (5 kHz) wird am Sartorius des Frosches mittels intrazellulärer Ableitung am Reizort untersucht, wobei die Wechselstromamplitude linear langsam über Minuten zunimmt. Die als reaktiv zu bezeichnende Depolarisierung ist ihrem Wesen nach ein eigenständiger Vorgang. Der hervorstechendste Unterschied gegenüber der physikalisch aufgezwungenen Gleichstromdepolarisierung ist, dass keine Akkommodation nachweisbar ist.

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