Tier 1 im 8/8-Std-Tag, beim Tier 11 im 10/10-Std-Tag und bei den Tieren 2 und 10 im 16/16-Std-Tag auf (Tabellen I, II, III). Diese Perioden wichen teilweise deutlich etwas von 24 Std ab, in einigen Fällen (im 8/8- und 16/16-Std-Tag) waren sie gleich 24 Std. Im ersten Fall kann ihr endogener Ursprung als erwiesen gelten; im zweiten Fall könnte man an den Einfluss eines nicht ausgeschalteten, unbekannten 24-Std-Zeitgebers denken. Möglicherweise liessen sich diese Käfer aber durch die 8/8-beziehungsweise 16/16-Std-Tage zu einer 24-Std-Periodik synchronisieren. Dies ist insofern denkbar, als beide Periodizitäten mit der 24-Std-Periodizität in relativ kurzen Abständen koinzidieren. Dann aber wäre die Synchronisation zur 24-Std-Periodik ein weiterer Hinweis auf eine zugrunde liegende endogene Circadian-Periodizität.

Die Ergebnisse zeigen, dass *C. cancellatus* einerseits einen ungewöhnlich grossen, innerhalb anderer Tierordnungen noch nicht beobachteten Mitnahmebereich hat[3]: sowohl im 8/8-Std- als auch im 16/16-Std-Tag ist noch Synchronisation möglich. Andererseits lösten sich einige Käfer im 8/8-, 10/10- und 16/16-Std-Tag mehr oder weniger vollständig vom Licht-Dunkel-Wechsel und zeigten eine circadiane Eigenperiodizität. Damit ist die Existenz einer selbst-erregten Circadian-Schwingung auch bei Carabiden erwiesen.

Summary. Most of the active individuals of *Carabus cancellatus* (Coleoptera) investigated were synchronized by 'zeitgeber'-lengths of 8/8 10/10 or 16/16 h. On the other hand, a smaller number of beetles lost the periodicities of these 'zeitgebers' and showed a circadian activity rhythm. According to these experiments, the existence of a self-sustained circadian oscillation in carabid beetles is demonstrated. As we believe, a special numerical method of computing the length of activity periodicities was used for the first time.

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The Influence of Cations on Contracture in the Superfused Ventricle of *Helix aspersa*

Methods. Ventricles were removed from hydrated snails[1] and superfused in the apparatus of Lamb and McGuigan[2] by means of downward jets of saline which entered each ventricle through a hole in its upper aspect. Contractures were induced by stimulation with alternating current of 6 V, 50 Hz. About half the ventricles beat spontaneously. The basic (control) saline contained 40 m*M* NaCl, 4 m*M* KCl, 4 m*M* $MgCl_2$, 5 m*M* $CaCl_2$, 15 m*M* $NaHCO_3$. It was equilibrated with 1.5% CO_2 in oxygen to give a pH of 7.6–7.8. In solutions containing different concentrations of calcium or magnesium, the osmotic pressure was kept constant either by adjustments in the amount of sodium present or by addition of sucrose, similar results being obtained either way. To investigate the possible influence of sodium, the bicarbonate buffer was replaced by one of *tris*(hydroxymethyl)aminomethane (16 m*M*, adjusted to pH 7.7 with HCl).

The procedure used to compare contractures in the presence of different concentrations of calcium was as follows: The heart was superfused with calcium-free saline for about 1 min, within which time the response to stimulation would disappear. The test solution was then applied and, after equilibration, 3 or 4 contractures were induced. Then the ventricle was equilibrated with the control solution and 3 or 4 more contractures were induced. The cycle was repeated for other test solutions. The effects of variations in magnesium and sodium were studied in a similar way, except that the ventricles were not equilibrated with calcium-free saline. The isometric contractures were of 3 characteristic forms, as shown in Figure 1. Contracture tensions were measured, as indicated, as the differences between resting or diastolic tensions and the steady values attained during stimulation and were expressed as percentages of control values obtained immediately afterwards.

Results and discussion. The relations between contracture tension and concentration of calcium and magnesium are shown in Figures 2 and 3. (A relation similar to that of curve A was found when contractures were induced by replacing 50 m*M* of the sodium with potassium.) Varying the concentration of sodium (as chloride) over the range 0–230 m*M*, while keeping the osmotic pressure constant with sucrose, had no significant effect on contracture tension in 6 ventricles.

Between periods of electrical stimulation, non-beating ventricles seemed to be in a state of tonus and, on a few occasions, stimulation at lower than usual intensity caused a slight fall in tension, perhaps through an effect on inhibitory nerve endings. Moreover, in the quiescent heart, there was a slight fall in tension when the level of calcium was lowered. In contrast, as reported for *H. pomatia*[3], and associated with partial depolarization[4], a beating ventricle deprived of extracellular calcium would immediately increase its rate and amplitude of beating and then gradually stop in systole. If calcium were then replaced, the tension would at once fall, though not always with resumption of beating, but, if superfusion with calcium-free solution continued, tension would fall gradually over a period of several minutes.

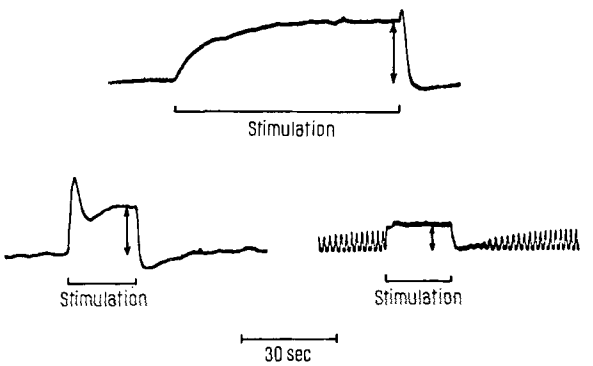

Fig. 1. Characteristic changes in tension in isolated ventricles on stimulation with alternating current. The manner of measuring steady contracture tension is shown for each.

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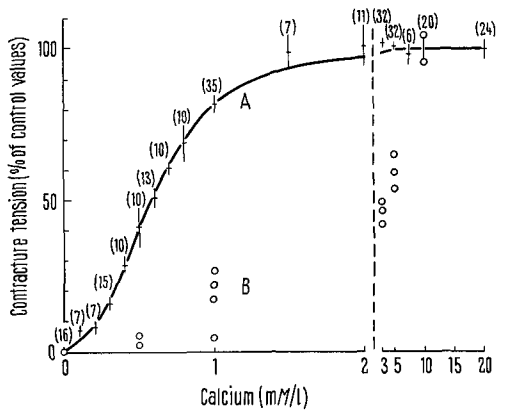

Fig. 2. The influence of the concentration of calcium in the superfusion medium on contracture tensions, the latter being expressed as percentages of a control value. Curve A: Magnesium, 4 mM. Results (from 12 ventricles) expressed as means $\pm$ standard errors of means; number of data averaged to give each point given in brackets. Curve B: Magnesium, 20 mM. Individual data from one ventricle.

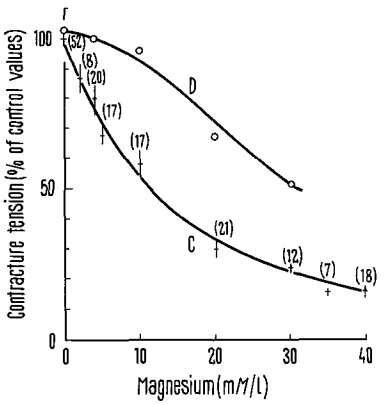

Fig. 3. The influence of the concentration of magnesium in the superfusion medium on contracture tension. Curve C: Calcium, 1 mM. Results (from 6 ventricles) expressed as means $\pm$ S.E. of means; numbers of data for each point given in brackets. Curve D: Calcium, 5 mM. Individual data from one ventricle.

Responses to changes in concentration of calcium or magnesium were immediate when these occurred actually during a contracture, and complete in about 5 sec. This rapidity is more suggestive of changes near the cell surface than of gross alterations in the amounts of calcium or magnesium inside and the results can be interpreted in terms of a competition of the two cations for superficial binding sites, with the tension depending on the proportion of sites occupied by calcium: On this view, the failure of tension to rise indefinitely with extracellular calcium concentration would be due to saturation of the sites. A comparable competition between calcium and magnesium is seen in uterine muscle of the rat[5], but, in frog ventricle, the action of calcium is antagonized by sodium[6,7].

The concentration of calcium in the blood of *H. aspersa* is usually 4–16 mM and, in hydrated snails, magnesium averages about 3.6 $\pm$ S.D. 1.2 mM[1]. Though some of the calcium is not ionized[8], there is enough present that even large variations can have only negligible influence on excitation-contraction coupling. Normal fluctuations in magnesium should have a greater effect, but, here too, the data suggest it would be unimportant.

Zusammenfassung. Die isometrische Kontraktionsspannung in der isoliertenHerzkammer – mit Wechselstrom gereizt – nimmt mit der Kalziumkonzentration im Medium bis zu einem «Plateau» zu. Dem Kalziumeffekt wird durch Magnesium entgegengewirkt während Veränderungen der Natriumkonzentration keinen entscheidenden Einfluss haben.

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On the Mechanism of Compensatory Hypertrophy in Skeletal Muscles

It has recently been shown that in the rat, following tenotomy of the gastrocnemius muscle, a rapid and striking hypertrophy of the synergistic soleus and plantaris muscles occurs[1,2]. In a preliminary attempt to identify the physiological mechanisms operating during compensatory hypertrophy, it seemed important to test the role of the neural influence or at least the necessity for intact neural circuits in inducing compensatory hypertrophy. Two alternative working hypotheses may in fact be considered to explain how the overload imposed on skeletal muscles following tenotomy of synergists might determine compensatory hypertrophy. On the one hand, the increased strain, signalized by the stretch receptors, may induce through reflex pathways an increased functional activity. In this case the compensatory hypertrophy that follows would be essentially similar to that due to physical exercise, in both cases the causal agent inducing hypertrophy being increased 'active' muscle work dependent on increased neural stimulation. Alternatively one can envisage a direct effect of mechanical tension on skeletal muscle, not dependent on nerves: the hypertrophy, in this case, would be produced by the increased 'passive' stretching of the loaded muscles. A similar mechanism has been suggested to explain the transitory hypertrophy of the denervated hemidiaphragm

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