unterscheiden sich signifikant ($p < 0,05$; U-Test von MANN und WHITNEY und nach dem FISHER-PITMAN-Test für unabhängige Stichproben).

Aus den Untersuchungen geht hervor, dass sich die gegenseitige Beeinflussung der Fische innerhalb der Gruppe synchronisierend auswirkt. Die Ergebnisse aus den Wintermonaten deuten darauf hin, dass neben dem Licht-Dunkel-Wechsel auch andere Synchronisationsfaktoren von entscheidender Bedeutung sein könnten. In den Monaten November/Dezember werden die Fische, während sie sich im Schwarm auf die Winterruhe vorbereiten, durch den natürlichen LD-Wechsel synchronisiert. Fällt unter Laborbedingungen dieser Steuerfaktor aus, ist bei den Einzeltieren das rhythmische Verhalten nicht mehr in der arttypischen Form ausgeprägt.

In Versuchen wurde die Bedeutung des optischen und chemischen Informationskanals für die Synchronisation von Einzeltieren untereinander analysiert. Überraschend war der deutliche circadiane Rhythmus, den sowohl linsen- als auch augenenukleierte Gruppen unter Licht-Dunkel- und Dauerdunkel-Bedingungen aufwiesen[12]. Somit konnte die optische Kommunikation nicht der alleinige Synchronisationsfaktor für die ausgeprägte circadiane Periodik innerhalb der Gruppe sein. Deshalb wurde in weiteren Experimenten die chemische Kommunikation ebenfalls überprüft. Die Streuung der Spontanfrequenzen bei den in Chemokommunikation stehenden Fischen war kleiner als bei den Tieren, die keine Möglichkeit zum Informationsaustausch hatten, da die optische Kommunikation im Dauerdunkel ausfiel.

Die Unterschiede in den Periodenlängen von 4, 66 und 140 Lux sind bei den Gruppen signifikant ($p < 0,05$; Rangvarianzanalyse nach FRIEDMANN). Bei den Einzeltieren lassen sich die Unterschiede der Spontanperioden von 4 und 140 Lux mit $p < 0,01$ sichern (t-Test). Nach diesen Ergebnissen gilt die Circadian-Regel auch für Fische. Der zusätzliche Einfluss sozio-ökologischer Faktoren für die Synchronisation wurde durch die Gruppenuntersuchungen deutlich.

Summary. In constant illumination fish groups and isolated fish exhibit a free running rhythm. The period length of the day active animals decreases with increasing light intensity and vice versa. The results correspond to the circadian rule, the validity of which has proved correct now for fish, too. The synchronization within fish groups is also influenced by socio-ecological factors.

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[12] R. SIEGMUND und D. L. WOLFF, forma et functio, im Druck.

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Role of Muscular Disuse in the Genesis of Fibrillation in Denervated Muscle

Fibrillation following denervation of skeletal muscle is generally[1–3] held to be due to cessation of a trophic effect of the nerve on the muscle, rather than to the muscular changes caused by disuse, since it is not observed in muscular disuse of different origin[4–7], even though muscle atrophy can develop to about the same degree as after denervation.

In the present paper it will be seen that fibrillation sets in much earlier than usually if spinal cord transection, or plaster cast immobilization of the limb, or tenotomy are carried out some days before denervation. These observations suggest that in the genesis of this type of muscular activity, muscular changes caused simply by disuse are likely to play a definite role.

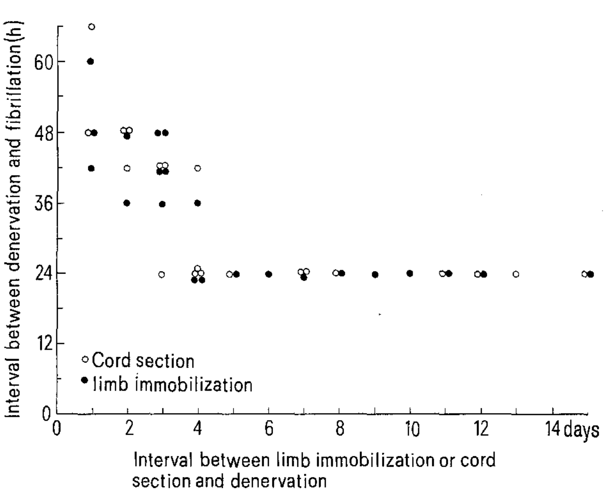

Fig. 1. Onset time of fibrillation in denervated muscles, as a function of the interval between spinal cord section, or the beginning of limb immobilization, and denervation. Individual values are presented from 42 animals.

Methods. Adult albino rats, weighing 250–300 g, were used throughout. All surgical procedures were performed under ether anaesthesia. In one group of animals, the spinal cord was sectioned at the mid-thoracic level, and one sciatic nerve was cut 1–15 days later, near the trochanter. In another group, both hind limbs were immobilized by a plaster cast, one limb in flexion and the other in extension, and after 1–15 days the cast was removed and the sciatic nerve was cut on both sides. In a third group, the tendo calcaneus was cut on one side, and 1–19 days later the sciatic nerve was cut on both sides. Electromyographic records were taken under ether anaesthesia, using a pair of needle electrodes which were introduced, through the skin, in several parts of the soleus-gastrocnemius muscular group. Records were first

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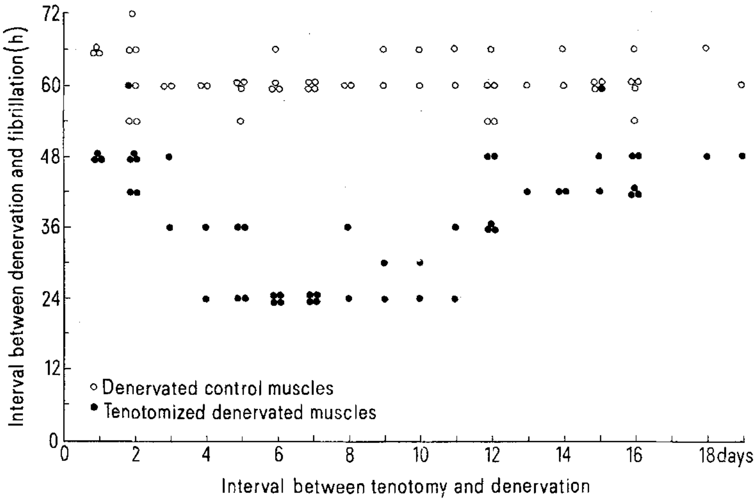

Fig. 2. Onset time of fibrillation in tenotomized denervated muscles, as a function of the interval between tentomy and denervation. Individual values are presented from 55 animals. Control data obtained from the contralateral, simply denervated muscles, are also reported.

taken 12–18 h after denervation, and afterwards every 6–12 h, until fibrillation had fully developed.

Results. In nearly all the muscles denervated without any other experimental procedure, the first fibrillation potentials could be detected only 60–66 h after denervation (see control data of Figure 2).

In the muscles denervated after cord section, limb immobilization or tenotomy, fibrillation appeared consistently earlier.

As shown in Figure 1, the average lag in the onset of fibrillation gradually shortened as the interval between cord section, or limb immobilization, and denervation was prolonged, reaching a minimum of 24 h when the interval was 5 days long; thereafter, the lag leveled off on this minimal value, even for intervals as long as 15 days. There was no difference between the effects of immobilization in flexion and in extension.

The minimal lag of 24 h was reached also in all the muscles denervated 6–7 days after tenotomy (Figure 2). When the intervals between tenotomy and denervation were longer than 6–7 days, fibrillation set in, on an average, less precociously: for intervals 13–19 days long, the lag ranged between 42 and 48 h. The latter result was obtained also when the scar tissue between the proximal and the distal stumps of the tendon had been cut 3–7 days before denervation. So it appeared to be independent of the re-establishment of the tendon, although some resumption of functional activity could not be excluded, because new adhesions rapidly developed between the tenotomized muscles and the surrounding tissues.

Discussion. The present results clearly indicate that some muscular change, not due to the lack of a trophic nervous influence, has an important role in the genesis of fibrillation.

The agreement between the data obtained in animals treated with different procedures, makes it likely that the change, or the changes, favouring the onset of fibrillation were mainly related to disuse, which in the 3 groups of experiments was the common feature preceding denervation.

It seems reasonable to assume that the alterations from disuse occur in denervated muscle as well, possibly at a faster rate and to a higher degree than in muscle put into disuse without a complete suppression of motor nerve activity. If this is the case, the longer interval between denervation and the onset of fibrillation in non pre-treated muscles as compared to the pre-treated ones, could reflect the time required for these alterations to develop[8].

Riassunto. Nei muscoli soleo-gastrocnemio di ratto la denervazione ha determinato insorgenza della fibrillazione dopo 60–66 h. Se eseguita dopo qualche giorno di disuso muscolare (conseguente a tenotomia, sezione del midollo spinale, immobilizzazione dell'arto) la denervazione ha determinato insorgenza della fibrillazione molto più precocemente, con un intervallo minimo di 24 h. Viene suggerito che nella genesi della fibrillazione dopo denervazione, il disuso muscolare abbia un ruolo importante.

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Neuron-Neuronal Attachments in the Parietal Cortex of the Rat

The parietal cortex of the rat fixed by perfusion of 100% formaline (12*M* formaldehyde) shows abundant closely apposed synaptic boutons[1]. Because the method of perfusion fixation employed had been specially designed to minimize hypoxia[2] no significant hypoxic reduction of the extracellular space (ECS)[3] was feared. As for the fixative, it was found to be so hypertonic that if perfused for more than 3 min the brain looked 'like a walnut in its shell', and the ECS was coarsely enlarged[1]. However, almost simultaneously, Brightman and Reese[4] reported failing in detecting interneuronal close appositions in mice, other than those related to hypotonicity and or poor preservation of different origins. These spurious entities they called 'labile appostions' and were described as 'five layered structures resembling tight junctions'... 'showing a middle laminae as dense as but wider than an individual outer leaflet of adjacent cell membranes'... 'their overall width was approximately twice that of an individual cell membrane'. These appositions lacked 'any associated cytoplasmic fuzz'. Lately, we have confirmed that,