flowed from its site of secretion to neighbouring non-secretory areas. Thus, when the tubule was filled with oil, the flow resistance of the oil columns would prevent the primary secretion from growing and spreading over onto neighbouring non-secretory areas so that a sample of pure primary secretion could be obtained. During microperfusion, however, where the length of the injected column of perfusion fluid was many times greater than the largest column of primary secretion which we have ever observed, the perfusion fluid could be expected to come into contact not only with areas where primary secretion was being formed but also with areas where re-equilibration with plasma could take place. The composition of collected perfusate would thus come to be intermediate between that of primary fluid and serum.

Under free-flow conditions, when no unphysiological hinderance to flow of fluid along the tubules would occur, there would be a continuous influx of water and electrolytes into the tubules and equilibrium conditions would not develop. Whether this fluid mixes with a sodium-rich rete secretion by ebb and flow, to give rise to free-flow fluid as we have previously postulated[1] remains an open question. What the present experiments make clear is that the seminiferous tubules do indeed secrete a 'primary' fluid which is richer in potassium and poorer in sodium than free-flow fluid, the secretion which normally lies in the undisturbed tubule. We are at present attempting to study formation of primary fluid further by continuous microperfusion in vitro of tubule segments.

Zusammenfassung. Mit Hilfe der «stopped-flow»-Mikroperfusionstechnik wurden am Samenkanälchen Elektrolyttransportprozesse untersucht. Es wurde gefunden, dass die Kanälchen ein kaliumreiches Primärsekret bilden. Dieses unterscheidet sich in seiner Zusammensetzung von dem Sekret, das man gewöhnlich unter ungestörten Fliessbedingungen findet.

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'Compensatory' Muscle Hypertrophy in the Rat Induced by Tenotomy of Synergistic Muscles

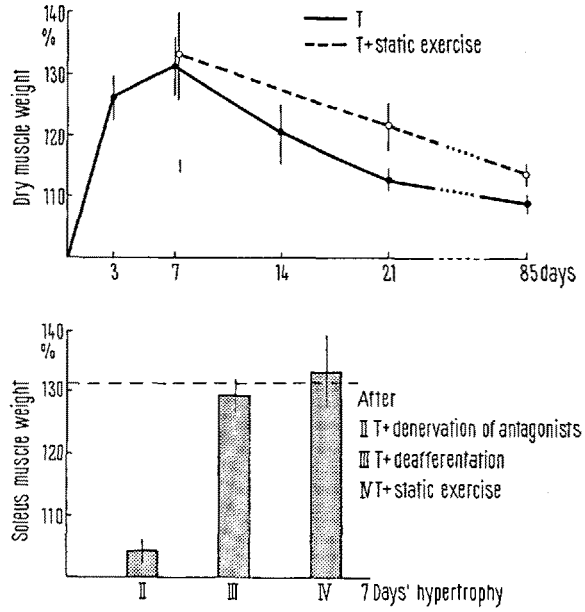
Several authors have described rapid hypertrophy of muscle following tenotomy[1] or denervation[2] of their synergists. Since the muscles undergoing hypertrophy could be expected to be under an increased functional load, compensatory hypertrophy seemed a reasonable term. However, it has recently been pointed out[3] that this particular type of hypertrophy is not apparently due to increased muscle activity, but rather to passive movements and mechanical tension of the hypertrophying muscles due to the action of their antagonists. The present report was intended to elucidate further the factors which are responsible for this type of hypertrophy.

In the 1st series of experiments (I) the tendon of the gastrocnemius muscle in 120–150 g male Wistar albino rats was sectioned under ether anaesthesia; a sham operation was performed on the contralateral side. Soleus muscles were excised 3, 7, 14, 21 or 85 days after the operation, respectively, weighed and then dessicated. The mean dry weight of the muscles on the operated side was expressed as percentage of the contralateral control.

In the 2nd series of experiments (II) tenotomy of the gastrocnemius was combined with section of the peroneal nerve which supplies the antagonistic muscle group. Tenotomy of the gastrocnemius was combined in the 3rd series of experiments with whole limb deafferentation (III) by ipsilateral section of L_1 – S_1 dorsal roots[4].

In the 4th series of experiments an attempt was made to increase muscle hypertrophy by an additional work load, either static or a combination of static and dynamic excercise (IV). Static training was achieved by making the experimental animal stand on a suspended wire ladder for 5 h a day starting on the day after tenotomy, while animals subjected to the combined training programme performed 85.5 h of static and 46 h of dynamic excercise by running on a tread-mill.

The maximum gain in weight in the soleus muscles was attained 7 days after tenotomy of the gastrocnemius (Figure 1, I). Thereafter, there was a relative loss of weight of the hypertrophied muscles, although their weight was still significantly higher than that of control muscles even after 85 days ($p < 0.001$). The initial rapid gain in weight is thus transient in character.



The time course and some factors affecting 'compensatory' muscle hypertrophy in the rat soleus after tenotomy of the gastrocnemius muscle. Above: I. dry muscle weight expressed as percentage of contralateral control (solid line), broken line indicates an analogous situation under additional static work load. Below: soleus muscle weight 7 days after tenotomy of the gastrocnemius and peroneal nerve section (II), limb deafferentation (III) and additional static work load (IV). Horizontal broken line represents soleus hypertrophy after 7 days (not combined with other procedures). Vertical bars denote standard error of the mean.

However, the soleus muscles did not gain any appreciable weight when their synergists were tenotomized, if their antagonists were excluded from function by denervation (Figure 1, II). In this type of hypertrophy muscle afferents are apparently not involved, since in deafferented soleus muscles the weight gain was comparable to that in muscles with intact sensory innervation (Figure 1, III).

When 'hypertrophying' muscles were subjected to static training, there was no increase in weight 7 days after the operation (Figure 1, IV), even though the residual hypertrophy at longer time intervals was somewhat enhanced (Figure 1, upper I, broken line). Combined excercise had a similar small effect.

It thus appears that the rapid hypertrophy which the soleus undergoes within a week after tenotomy of the gastrocnemius is transient in character and regresses almost completely by the 3rd postoperative week. This hypertrophy is not predominantly due to excessive use of the muscles, but rather to mechanical passive stretching by the antagonistic group, since denervation of the latter prevents the development of muscle hypertrophy. Additional exercise did not further increase the muscle weight increment at its peak as compared with the non-exercised animals. A reflex mechanism is apparently not involved, since even complete limb deafferentation did not prevent the development of muscle hypertrophy.

However, it cannot be excluded that part of this weight gain is actually true working hypertrophy, since a certain weight increment persists for longer periods of time (Figure 1, I) and has also been found in the plantaris and extensor digitorum longus muscles under analogous conditions[5].

Zusammenfassung. Nachweis, dass die schnelle Hypertrophie des M. soleus nach Tenotomie des synergistischen M. gastrocnemius durch passive Spannung des antagonistischen Muskels verursacht wird. Denervierung dieser Antagonisten verzögert eine Hypertrophie des M. soleus, während die Deafferenzierung des ganzen Gliedes ohne Effekt bleibt; auch zusätzliche Arbeitsleistung führt auf dem Höhepunkt der Hypertrophie zu keinerlei Gewichtszunahme.

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Electrophysiology Studies on the Cardiac Non-Function Mutation in the Mexican Axolotl *Ambystoma mexicanum*[1]

HUMPHREY[2] reported the discovery of a recessive and lethal gene *c* (for cardiac nonfunction) in *Ambystoma mexicanum*. Myogenic activity of cardiac muscle was absent in the homozygote of gene *c*, although early morphological developments were similar to those observed in normal embryos[3]. Mutant embryos later became edematous and their developing hearts became thin-walled, elongated and tortuous. Mutant hearts at Harrison stage 40 were only one cell layer thick, had excessive accumulation of yolk and lipid material, and lacked trabeculae which were present normally at stage 38. Mutant animals expired within 21 days after the time when hearts would normally demonstrate myogenic activity.

Transmembrane potential responses of in vivo embryonic hearts of normal *Ambystoma mexicanum* recorded prior to and immediately after onset of myogenic activity have been reported[4]. Typical resting potentials and action potentials, only present after onset of myogenic activity during Harrison stage 34 were recorded (Table). Acetylcholine, norepinephrine and electrical stimulation were without effect on quiescent hearts, in contrast to modification of cardiac activity after onset of myogenic activity[4].

The present study was undertaken to compare the electropharmacological induced responses of in vivo embryonic hearts of normal and homozygous cardiac mutants of *Ambystoma mexicanum*.

Materials and methods. Fertilized eggs from *Ambystoma mexicanum* were incubated until development approximated Harrison stage 34. All embryonic hearts of normal *Ambystoma mexicanum* begin to beat during and after stage 34, whereas cardiac mutants do not show myogenic activity during any stage of development. Individual embryos were transferred to a Syracuse watch dish layered with paraffin and 10 ml Steinberg's solution[4] maintained at 25 ± 0.1 °C. Wire restraints immobilized anesthetized embryos on the dorsal surface, and hearts were then exposed. Animals required 10 min to completely recover from anesthesia and experimental manipulation.

Transmembrane potentials[4] were recorded from 29 mutant homozygous for gene *c* and 29 heterozygous developed to stages 34–38. Hearts were directly stimulated 60–120/min and stimulation parameters were continually monitored. The influence of acetylcholine, norepinephrine and serotonin (0.1 µg/ml to 0.1 mg/ml) on myogenicity was determined. Only one drug was used with each embryo.

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