pattern differs in several ways from the type 1 unit. First, there is no indication of the inhibition prior to the excitation. We have illustrated the responses to a vertical slit, $0.7° \times 5.6°$, to demonstrate that even when the stimulus is larger than the RFC, there is no indication of the inhibition. Second, the response amplitude is lower. And third, the inhibition when the stimulus leaves the RFC is weaker and shorter-lasting.

We have examined approximately 60 on-center units and have found about equal numbers of the 2 types of units. The question arises whether these 2 types of units correspond to the sustained and transient units described by earlier investigators. Examination of the responses elicited by a moving horizontal slit has provided evidence that the type 1 unit corresponds to the sustained type and the type 2 unit corresponds to the transient type of unit. The average response histograms of type 1 and type 2 units are shown in Figure 2. The responses in the upper row were elicited by a 0.7° square while those in the lower row were elicited by a $5.6° \times 0.7°$ horizontal slit. The stimulus intensity was 2.0 log units above threshold for all of the responses. The differences in the responses of the 2 types of units when a 0.7° stimulus was used are similar to that just described. With the horizontal slit an additional difference can be noted. For the type 1 unit, the high frequency burst of spikes is followed by a maintained firing level which is significantly higher than the spontaneous firing level. In the type 2 unit, the firing level decreases to the spontaneous level after the high frequency burst. The maintained firing level of the type 1 unit is in keeping with the response of the sustained type while the fast decay of the firing level ot the spontaneous level is what would be expected of the transient type of unit. Thus the type 1 units resemble the sustained type of units by showing a maintained firing while the stimulus is within the RFC, and a stronger and longer-lasting inhibition when the stimulus leaves the RFC.

What can account for the differences in the responses of the 2 types of units when a moving stimulus is used? The answer to this question may be provided by the response of sustained and transient units to a large annulus. The sustained type responds to an annulus flashed in the surround with a response characteristic of the surround component, while the transient unit responds to the same stimulus with a response characteristic of both the center and surround[6,7]. This difference has been attributed to a difference in the spatial arrangement of the center and surround components of the RF. For the sustained type, the diameter of the surround component is larger than the center component so that there exists a rim where only the surround component is present as in RODIECK and STONE's[8] model of the RF. For the transient type, the borders of the center and surround components are coincident so that both components are present throughout the RF. Thus for the type 1 or sustained unit, the moving stimulus will encounter first the rim of the surround component which will decrease the spontaneous firing rate. For the type 2 or transient unit, the stimulus will pass into a region where both the center and surround components are present so that the inhibition from the surround will not be noted. Thus our observations can be accounted for by the difference in the spatial arrangement of the center and sourrund components of the RF as proposed by earlier investigators[7].

Résumé. Deux Types de réponses apparaissent dans des cellules ganglionnaires du chat soumises à des stimuli mobiles. Ils correspondent aux unités répondant de façon transitoire ou durable et proviennent d'une différence dans la disposition spatiale du centre et de la périphérie du champ récepteur.

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Effect of Growth Hormone and Thyroxine on the Contractile Properties of Dystrophic Muscle

Strain 129Re muscular dystrophic mice at 3 weeks of age were injected daily with 1 μg thyroxine and 5 μg growth hormone. The effect of hormone treatment was tested by measurement of peak twitch tension, relaxation rate, and 'fatigue' in small strips of excised abdominis muscle, stimulated in oxygenated Ringers solution. While gross differences between normal and dystrophic muscle are reported, no change in the contractile behavior of the hormone treated dystrophic muscle was found. The possible relationship between hormone deficiency and muscular dystrophy is discussed.

Methods. Strain 129Re muscular dystrophic mice[1] were obtained at 3 weeks of age and injected for 14–17 days. Purified bovine growth hormone[2] was dissolved in 9% NaCl, pH 9.0. L-thyroxine[3] was dissolved in 0.01 *N* NaOH. Injections consisted of daily doses of 1 μg thyroxine in 0.025 ml of the NaOH solution and 5 μg growth hormone in 0.025 ml of the NaCl solution. Dystrophic mice were divided randomly into a hormone-injected group (average weight 7.4 g) and a control group (average weight 7.6 g), and injected daily with 0.05 ml 9% NaCl. A third group containing non-dystrophic litter-mates (average weight 11.9 g) were injected with 0.08 ml 9% NaCl, the higher volume being in proportion to the higher mean body weight.

All mice were sacrificed between 14–17 days of injection. Strips of excised abdominis muscle approximately $2 \times 6 \times 1$ mm. (1–3 mg dry weight) were clamped vertically in an oxygenated Ringers solution at 15°C. The apparatus used, which permitted automatic adjustments of resting length and tension has been previously described[4]. The

[1] Mice were obtained from Bar Harbor Laboratory, Bar Harbor, Maine.

[2] Growth hormone was graciously provided by Dr. E. BATES of The National Institute of Health.

[3] L-thyroxine was obtained from the Sigma Company.

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Table I.

Group	Peak twitch tension (g/g)	Fatigue index	Peak relaxation (g/sec)
Normal (8)	737 ± 52	0.76 ± 0.07	8.7 ± 0.9
Dystrophic control (6)	152 ± 28^{a}	0.79 ± 0.05^{b}	1.2 ± 0.24^{a}
Dystrophic injected (4)	85 ± 12^{b}	0.81 ± 0.07^{b}	0.49 ± 0.14^{b}

$\pm$ Standard error; ( ) number in group. $^{a} = p < 0.01$; $^{b} = p > 0.05$, when compared with above value in column, by Mann-Whitney U-test.

Table II.

Tissue	DNA/g muscle	Dry wt./wet wt.	Protein nitrogen	Phosphorylase activity
Hypophysectomized	Increases 18–21 days[7]	Lower than normal, increasing after day 17[7]	Low[10]	Low[7]
Dystrophic	Increases 18–21 days[8]	Lower than normal, increasing after day 17[9]	Low[11]	Low[12]
Normal	Decreases 18–21 days[7,8]	Decreasing after day 17	—	—

muscle was stimulated supramaximally every 2 sec for 30 min. Peak twitch tension and contraction times were recorded. The muscle strips were placed in an oven overnight at 105 °C., and peak twich tension in g/g dry weight calculated. An index of fatique was taken as the ratio of peak twitch tension at 30 min to the initial tension at time = 0. Relaxation time was calculated as the slope of the line tangent to the relaxation curve at its steepest point.

Peak twitch tension in g/g dry weight was shown to be almost 5 times greater in the normal mice than in the dystrophic control. Relaxation rate was 7.2 times greater in the normal, giving evidence for a previously reported observation by SANDOW[5] that the relaxation mechanism in dystrophic muscle may be defective. No difference in the fatigue pattern between normal and dystrophic muscle was found, an observation which greatly differs from a study by GABEL[6] showing dystrophic muscle fatigues less quickly than normal muscle. No myotonic response was recorded in any dystrophic muscle.

Differences between the dystrophic control and dystrophic injected group in regard to peak twitch tension, fatigue, and relaxation are not significant at $p = 0.05$, as determined by the Mann-Whitney U-test.

Dissussion. While it is apparent that our hormone treatment was not effective in this study, it is of interest to examine the hypothesis that muscular dystrophy may be related to an inability of some hormone to cause maturation of muscle fibers. Of particular significance are a series of biochemical aberrations which appear in skeletal muscle of both dystrophic and hypophysectomized chick embryos in a similar manner both quantitatively and chronologically. Table II summarizes this data.

From the Table, biochemical differences between normal and dystrophic muscle occur sometime around the 17th day of development. (ENEMAR[13] reports that the onset of pituitary activity in the chick embryo to be 15 days.)

DE LA HABA[14] has implicated growth hormone in the differentiation of muscle in vitro, and SPIRO[15] reports that thyroid hormone may be intimately related to the sarcoplasmic reticulum and intrinsic contractile properties to dystrophic muscle. The present experiment with thyroxine and growth hormone was not effective in restoring normal contractile properties to dystrophic muscle. Possible characteristics of dystrophic muscle which may have influenced our results are: a) membrane alteration, in which case hormones would leak out of cells, or not bind properly; and b) poor microcirculation which would prevent proper transport of hormone to muscle.

Résumé. Chez des souris ayant une dystrophie musculaire, l'injection d'hormone d'accroissement et de thyroxine n'a pas d'effet. On l'a observé en mesurant la tension maximum de la secousse, le degré de relâchement et les courbes de fatigue dans des lambeaux étroits du muscle abdominal excisé.

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