

Handed out: 2 March 06

Due: 9 March 06

**16.423J/HST515J/ESD65 Space Biomedical Engineering and Life Support Systems  
Muscle Homework Assignment**

Illustrations are highly desired in answers!

1. (1 points) From the readings and lecture, please describe the sliding filament mechanism.

**SOLUTION:**

- Muscle filaments are composed of actin and myosin proteins. The muscle myofibril contraction results from the relative sliding of actin and myosin.
- The contraction results from the myosin heads forming crossbridges on active sites of the actin. The isometric tension output is directly proportional to the number of crossbridges formed (T-L curve).
- The myosin heads go through a cyclic motion (attach on actin / pull / detach / attach...), allowing to create the pulling force while contracting
- Muscle shortening should be based on the relative motion of sliding filaments because:
  - A. A-band width stays constant during stretch and shortening
  - B. A-band disappears when myosin is dissolved away
  - C. Actin filaments begin at Z-line, run through I-bands into A-band, but stop before reaching H-zone (muscle at rest length)
  - D. I-band is entirely actin filaments
  - E. H-zone is myosin filaments
- In shortening, the relationship between the Force and Velocity is (Hill's Curve, Hyperbolic Form):

$$(T+a)(v+b) = (T_0+a)b$$

where  $T_0$  = isometric tension (velocity = 0)

- Muscles shorten more rapidly against light loads than they do against heavy ones.
- Muscles which are actively shortening can produce less force than those which contract isometrically.
- There is a discontinuity in the slope of F-V curve at zero velocity.
- Active muscle yields when the load exceeds about  $1.8 T_0$  ( $T_0$  – Tetanus tension).
- Hill's observations of the Fenn effect give a linear relation between total rate of energy liberation and tension.
- Fenn = muscle produces a certain extra heat when it shortens a given distance, whether shortening velocity is fast or slow.
- Muscular energy liberation should be based on the splitting of a high-energy phosphate as actomyosin attachments separate.

2. (1 points) Briefly discuss muscle coactivation.

**SOLUTION:**

Typically referred to as the  $\alpha$ - $\gamma$  (alpha-gamma) coactivation principle, it can be used to better understand movement control. More specifically,  $\alpha$ - $\gamma$  (alpha-gamma) coactivation can be described as muscle commands being signaled simultaneously with increases in both  $\alpha$  and  $\gamma$  activity by higher order motor centers. This alleviates the problem of contraction of the extrafusal muscle mass and a slackening of the spindles if only  $\alpha$  motor activity changes (say increased). The stretch reflex would then come into play as the Ia afferent activity drops, causing the  $\alpha$  motor activity to fall, thus, turning the muscle force off. In short,  $\alpha$ - $\gamma$  coactivation alleviates this problem.

Evidence from electromyography (EMG electrodes that measure muscle activity) from indwelling intrafusal and extrafusal electrodes record muscle fibers of the hand show that an increase in discharges from the spindle afferents occurs at the same time or only slightly after the beginning of electrical activity in the extrafusal muscle. The slight delay is accounted for by the time required for the intrafusal fibers to contract.

**More information (i.e., food for thought).** A motor command via the alpha motoneurons can only be effective in the presence of adequate facilitation from muscle spindles. If alpha activation leads to muscle shortening then the unloading of the spindle will reduce alpha motoneuron activity unless spindle afferent output is maintained by fusimotor activity. The logical way to maintain or increase spindle input (to alpha motoneurons) is to drive the static bag 2 fibers via the static gamma system. This biases Ia output. Dynamic bag 1 fiber biasing action is too small for use except in small slow movements. Appropriate action is therefore coactivation of alpha and gamma with the gamma receiving a modulated pattern based on the alpha pattern. As muscle shortens, Ia discharge is maintained. This explains the lack of change in Ia output seen in human movement. Compensation for spindle unloading could also come from the group II pathway by increasing static fusimotor output to chain fibers with secondary endings. There would be powerful biasing action in this case. Note that the above conditions keep the spindle responsive to unexpected length changes resulting from fatigue, change in load or obstruction of movement.

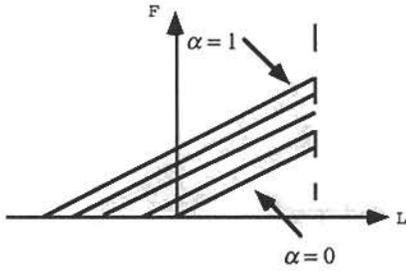
If you want to model planned movements, two messages are required.

1. A message to the spindle signaling the desired trajectory of the movement.
2. A signal to the extrafusal muscle that takes into account any load and fatigue. If the load proved unexpectedly great then the spindle would suffer a smaller degree of unloading, afferent discharge would increase and reflexes would assist movement. The discharge may also cause a central command to increase motor outflow. This is basically the  $\alpha$ - $\gamma$  (alpha-gamma) coactivation principle.

**Note:** This is a lot of text, which could be better captured using less text and nice illustrations (think about this before the Quiz).

**SOLUTION:**

(a) Muscle characteristics:



Muscle Force:  $F = kL + d\alpha$

$\alpha = e$

$F = -M\ddot{L}$  (-VE sign indicates that the muscle contracts (shortens))

$\therefore -M\ddot{L} = kL + de$

or  $M\ddot{L} + kL = -de$

(second order system; undamped; natural frequency =  $\sqrt{\frac{K}{M}}$ )

(b) Tendon organ:  $I_b = cF$

Thus:  $F = kL + d\alpha$ ;  $\alpha = e - I_b = e - cF$   
 (-VE sign as  $I_b$  feedback is inhibitory)

$\therefore F = kL + de - dcF$ ;  $F(1 + dc) = kL + de$   $F = -M\ddot{L}$

$\therefore -(1 + dc)M\ddot{L} = kL + de$   
 or  $(1 + dc)M\ddot{L} + kL = -de$

(second order system; undamped; natural frequency =  $\sqrt{\frac{k}{M(1 + dc)}}$ )

(c) Muscle spindle:  $I_a = aL + b\dot{L}$

Thus:  $F = kL + d\alpha$   $\alpha = e + I_a = e + aL + b\dot{L}$   
 (+VE sign as  $I_a$  feedback is excitatory)

$\therefore F = kL + d(e + aL + b\dot{L})$   $F = -M\ddot{L}$

$\therefore -M\ddot{L} = kL + de + daL + db\dot{L}$

$M\ddot{L} + db\dot{L} + (k + da)L = -de$

(second order system; damping ratio =  $\frac{db}{2\sqrt{(k + da)M}}$ )

natural frequency =  $\sqrt{\frac{k + da}{M}}$

(d) Both  $I_a$  and  $I_b$

$F = kL + d\alpha$   $\alpha = e + I_a - I_b = e + aL + b\dot{L} - cF$

$F(1 + dc) = kL + de + daL + db\dot{L} = -M\ddot{L}(1 + dc)$

$$\therefore M(1+dc)\ddot{L} + db\dot{L} + (k+da)L = -de$$

(second order system;

$$\text{undamped natural frequency: } \omega_n = \sqrt{\frac{(k+da)}{(1+dc)M}};$$

$$\text{damping ratio: } \xi = \frac{db}{2\sqrt{(k+da)(1+dc)M}}$$

Compare the natural frequencies and damping ratios. What have the spinal arcs contributed to “muscle management”?

The effect of increasing  $c$  is to lower both the natural frequency and the damping ratio. The effect of increasing  $a$  is to increase the natural frequency and decrease the damping ratio. The effect of increasing  $b$  is to increase the damping ratio.

Note that  $\omega_n$  can be rewritten as

$$\omega_n = \sqrt{\frac{(k/d+a)}{(1/d+c)M}}$$

and the damping  $\xi$  ratio as

$$\xi = \frac{db}{2\sqrt{d(k/d+a)d(1/d+c)M}} = \frac{b}{2\sqrt{(k/d+a)(1/d+c)M}}$$

Now suppose  $a$  and  $c$  are large enough to a first approximation  $a \gg \frac{k}{d}, c \gg \frac{1}{d}$ .

Then

$$\omega_n = \sqrt{\frac{a}{cM}}, \xi = \frac{b}{2\sqrt{acM}}$$

Thus the overall response of the entire effector (that is, the muscle and its spinal reflex arcs) is independent of the characteristics of the muscle, i.e., improved “muscle management”.

## 4.1

The original Simulink model sums up only 0.5 of a slow fiber, 0.3 of a fast fatigue resistant (ffr) fiber, and 0.2 of a fast fatigable (ff) fiber. To change from modeling just one fiber to modeling 1000 fibers, a simple gain is added immediately after each fiber's transfer function in Simulink. The value of each gain is set to 1000. Figure 4.1 below shows these added gain boxes, as well as the inputs converted from pulse generators to step functions.

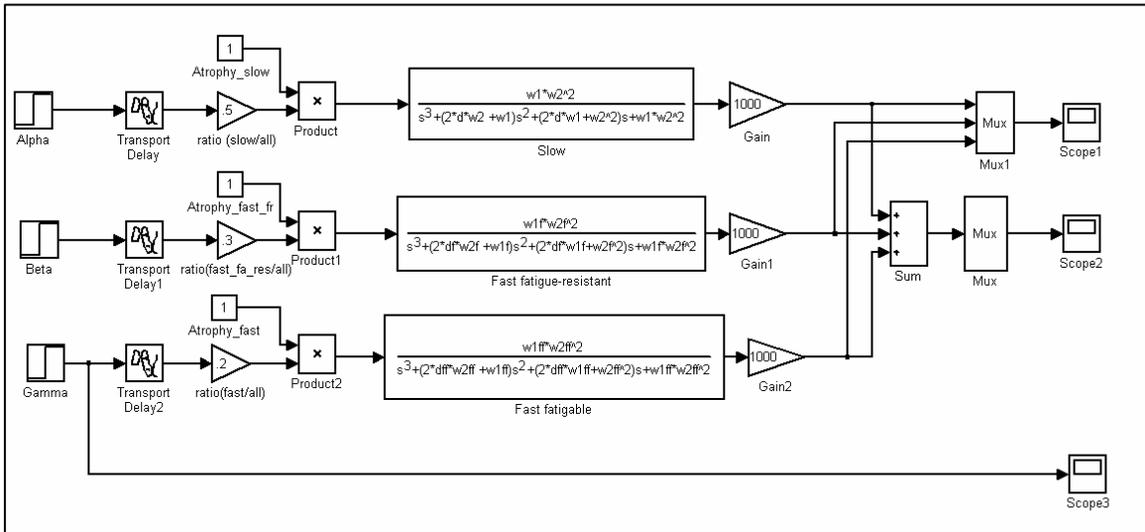


Figure 4.1 Simulink model that increases overall number of fibers to 1000

## 4.2

When the 1000 fibers are activated by step functions with amplitudes of 1, 2, and 3, for the slow, ffr, and ff fibers, respectively, the total steady-state force output is 1700 N. The form of this output is a step response with a rise time of about 0.25 seconds and a slight (~40 N) overshoot above 1700 N. To the steady-state response, the slow fibers contribute 500 N, the ffr fibers contribute 600 N, and the ff fibers contribute 600 N. These results correctly demonstrate the Henneman size principle: the slow fibers are the weakest of the three types.

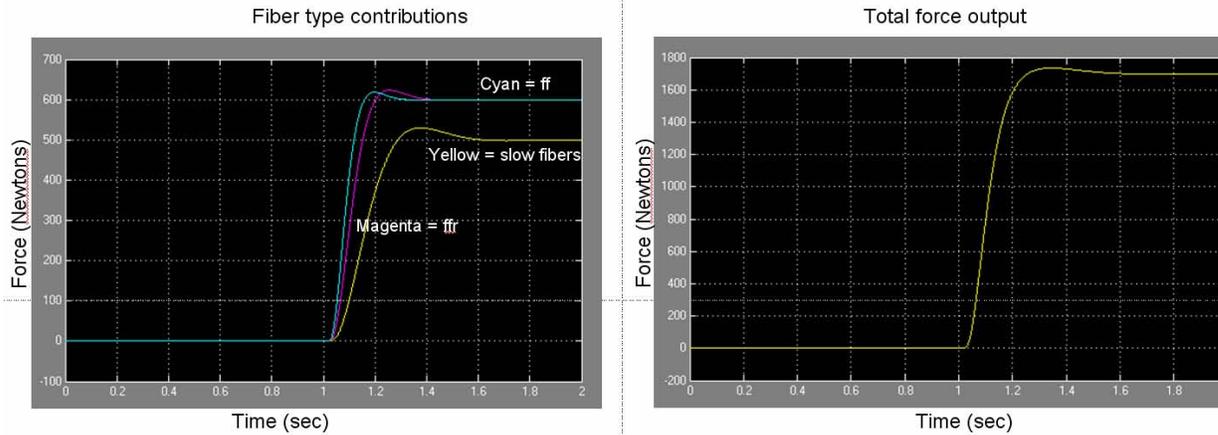


Figure 4.2 (LEFT) Force components from each fiber type at maximum activation levels, and (RIGHT) total force output at maximum activation levels

### 4.3

With only the slow fibers activated, the maximum steady-state force generated by the lower limbs is 500 N. The activation pattern to simulate only the slow fibers activated is  $[\alpha = 1; \beta = 0; \gamma = 0]$ . Figure 4.3 shows this result of 500 N. Notice that in this result, because the ffr and ff fibers are not activated, they contribute nothing to the 500 N force output. Because 500 N is greater than Jule's weight of 460 N, we know that she can maintain an upright position with her nominal activation pattern of  $[1 \ 0 \ 0]$ .

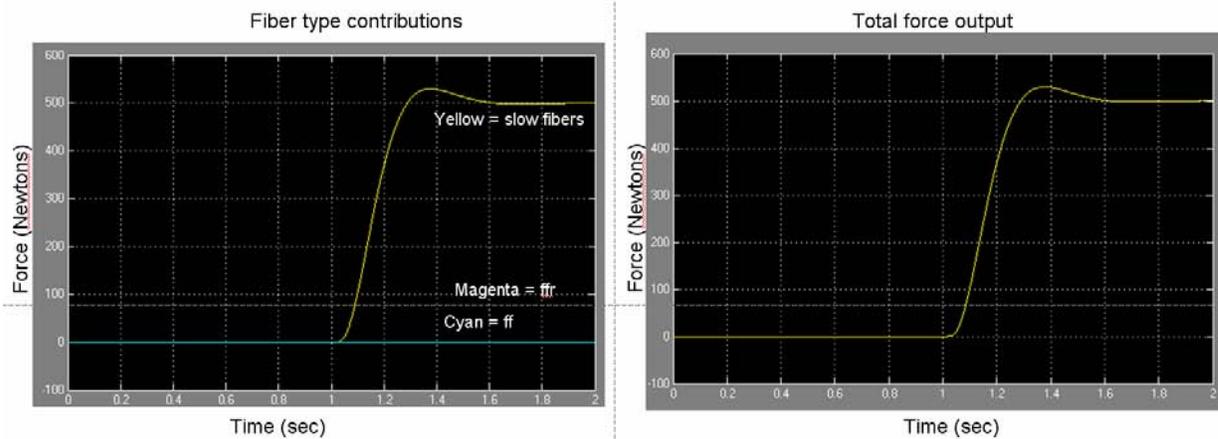


Figure 4.3 Force output with nominal activation pattern.

### 4.4

We can model the damage of 100 slow, 120 ffr, and 150 ff fibers by changing the values of the "atrophy" multipliers that feed into each of the fiber's "ratio" gains. There are 400 slow fibers remaining out of the initial 500 slow fibers, so the slow atrophy value is  $400/500 = 0.8$ . For the ffr fibers, 180 out of 300 remain undamaged, so the ffr atrophy

value becomes  $180/300 = 0.6$ . Finally, because 50 out of 200 ff fibers remain undamaged, the ff atrophy value becomes  $50/200 = 0.25$ .

When the atrophy multipliers are reduced to these values, the total force output is 400 N for an activation pattern of [1 0 0]. 400 N is less than Jule's weight of 460 N, so we must conclude that she cannot hold her body upright upon return to Earth with the nominal activation pattern. Figure 4.4 shows this result of 400 N.

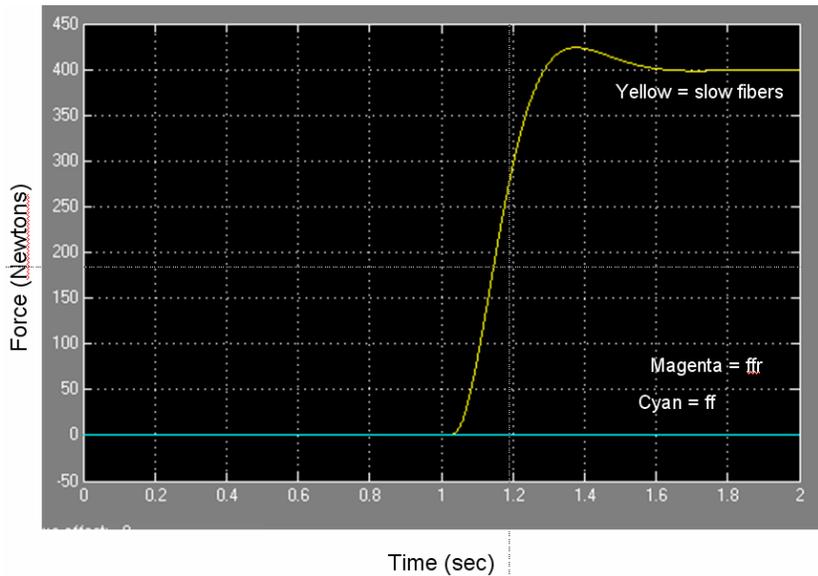


Figure 4.4 Force output with nominal activation pattern and “atrophied” muscles

If the activation pattern is changed to  $[\alpha = 1; \beta = 0.333; \gamma = 0]$ , then the total steady-state force output becomes 460 N, and Jule can just hold herself upright. This value of  $\beta = 0.333$  is determined in the following manner: first, we notice that 60 N additional output are required to bring the lower limb force up from the nominal 400 N to the minimum 460 N. The slow fibers are already completely activated, so we must activate the fast fatigue resistant fibers enough to generate the additional 60 N. We can run a simulation to see that if the ffr activation level,  $\beta$ , is set to 1, then the ffr fibers generate 180 N of force. The minimum  $\beta$  level, then is equal to  $60 \text{ N} / 180 \text{ N} = 0.333$ . The minimum activation pattern for standing is then  $[1 \ 0.333 \ 0]$ .

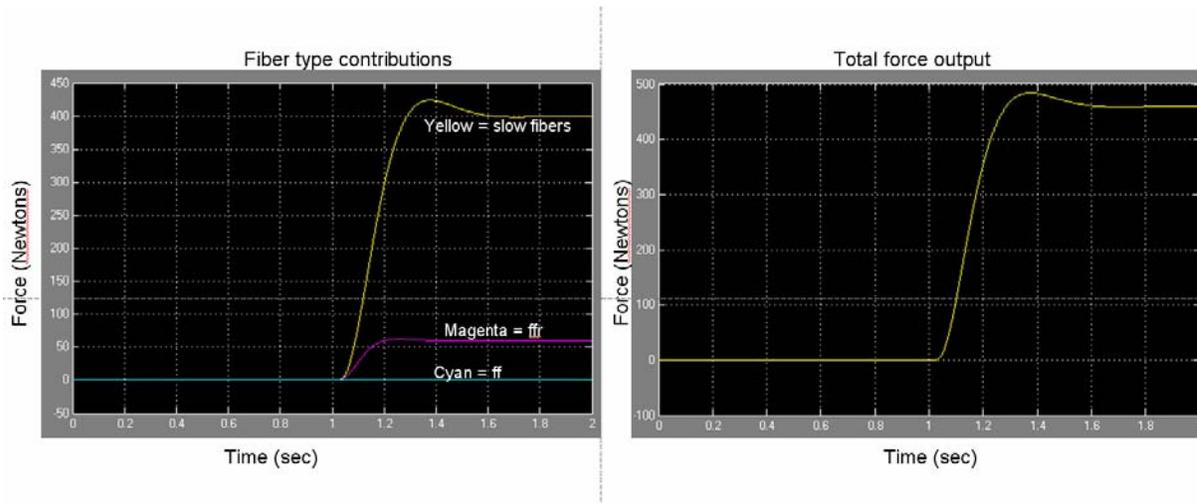


Figure 4.5 (LEFT) Force output contributions from slow and ffr fibers, and (RIGHT) total force output with  $[1 \ 0.333 \ 0]$  activation pattern and “atrophied” muscles

#### 4.5

The maximum force Jule can generate with her atrophied muscles is 910 N. This is the total steady-state output when the activation pattern (step inputs) is returned to  $[\alpha = 1; \beta = 2; \gamma = 3]$ . The slow fibers contribute 400 N, the ffr fibers contribute 150 N, and the ff fibers contribute 360 N. Figure 4.6 shows these individual contributions and the total force output.

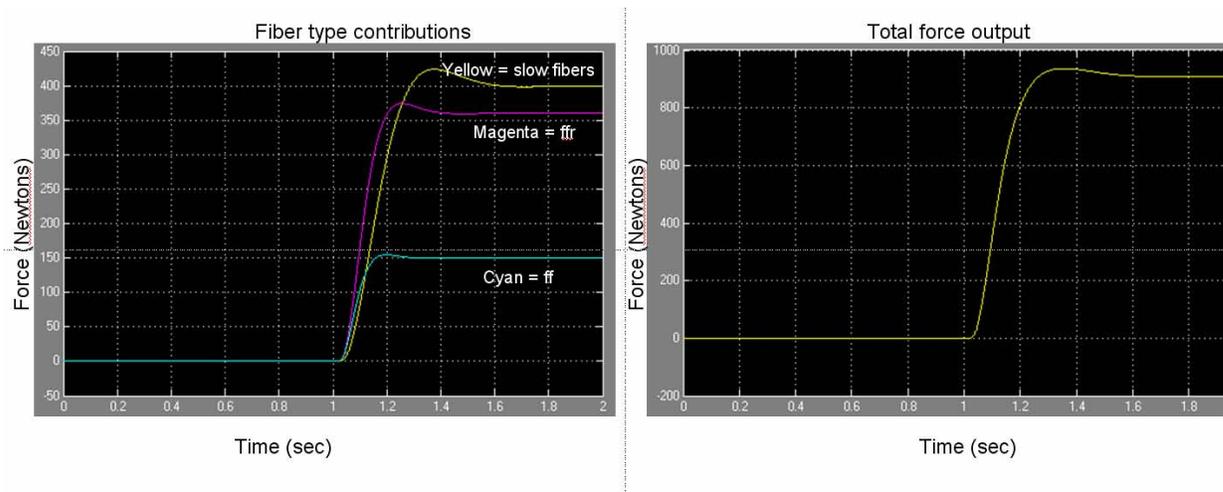


Figure 4.6 (LEFT) Contributions from individual fiber types and (RIGHT) total output with  $[1 \ 2 \ 3]$  activation pattern and “atrophied” muscles

#### 4.6

To model Jule’s severe atrophy and damage of 200 slow fibers, 240 ffr fibers, and all ff fibers, we reduce the atrophy multipliers in the Simulink model to 0.6, 0.2, and 0. Then, under the nominal activation pattern, the total steady-state force output is 300 N (as usual,

under the nominal pattern, all of this force comes from the slow fibers). Jule cannot stand up under the nominal pattern. Figure 4.7 shows this result.

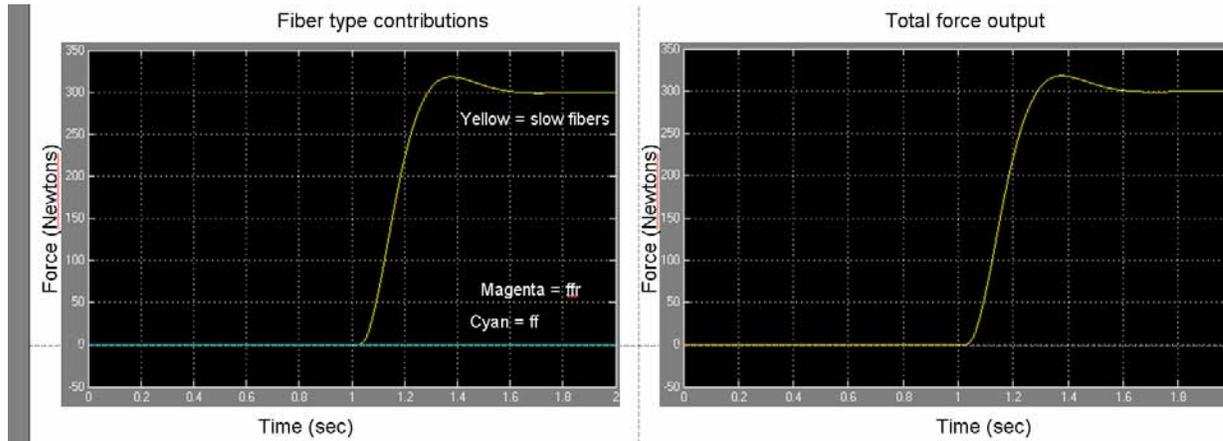


Figure 4.7 Insufficient force output with severe atrophy and nominal activation pattern

If the activation level of the ffr fibers is increased to its maximum of  $\beta = 2$ , then total force output is still only 420 N. This value is 40 N less than Jule's weight, so she *cannot stand up at all* with atrophy this severe. No activation pattern will enable her muscle fibers to produce 460 N of force output. (Since all the ffr fibers are damaged, changing gamma will not change the output at all.) Figure 4.8 shows this still insufficient force output of 420 N.

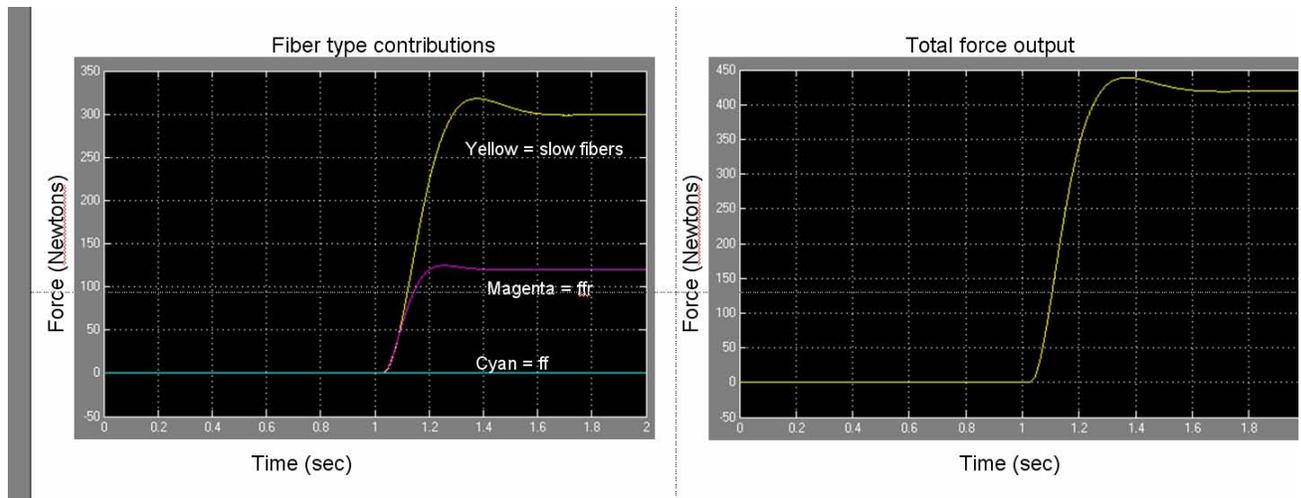


Figure 4.8 Insufficient force output with severe atrophy and maximum activation pattern.