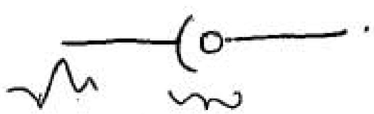
Lecture 9

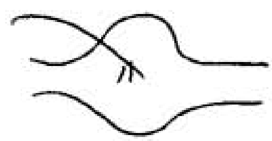
Last time: model of muscle spindle (vel + pos).

- γ carries additional info - sensitivity controlled.
- Ia Vs II
- Talked about Interneurons & their connectivity - Spatial
  - Temporal

Because the neural signal takes time to propogate, we can manipulate temporal info w/i spinal cord



chemical exch. = synaptic delay



chemical rxn = excitation to Contraction delay.

Side note: different neurons require diff. amount of input to fire

This is where memory may be located.

Spinal adaptation - changing synapse threshold

- stimulate a lot, lower threshold

We will talk about how delays could cause problems, but in general, they are also used for our advantage.

→ adding more interneurons, add more delays

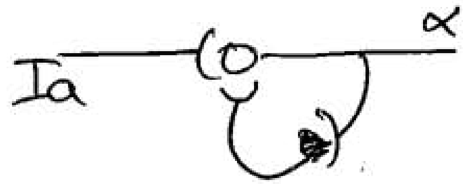
CPG: oscillating neur

~~we'll talk about how delays could cause problems, but in general, they are also used for our advantage.~~

One more impt. kind of neurons

Renshaw cells - negative feedback

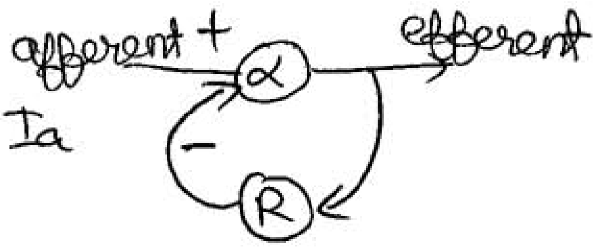
- inhibitory neurons that reverberate m.n s



add to the diag.

Adds negative delayed feedback

- causes transient depression of the reflex sensitivity for immediate future (protects from extreme acti



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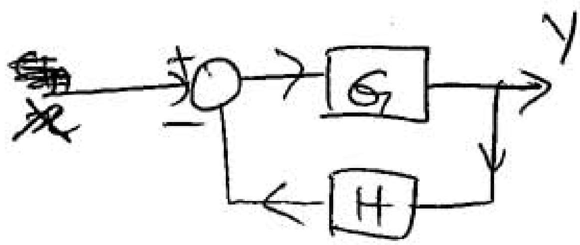


$$\frac{Y}{X} = \frac{G}{1+GH}$$

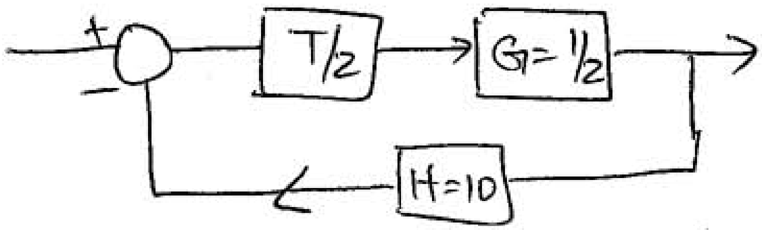
feedback gain can be changed modulating effectiveness of synaptic inpu

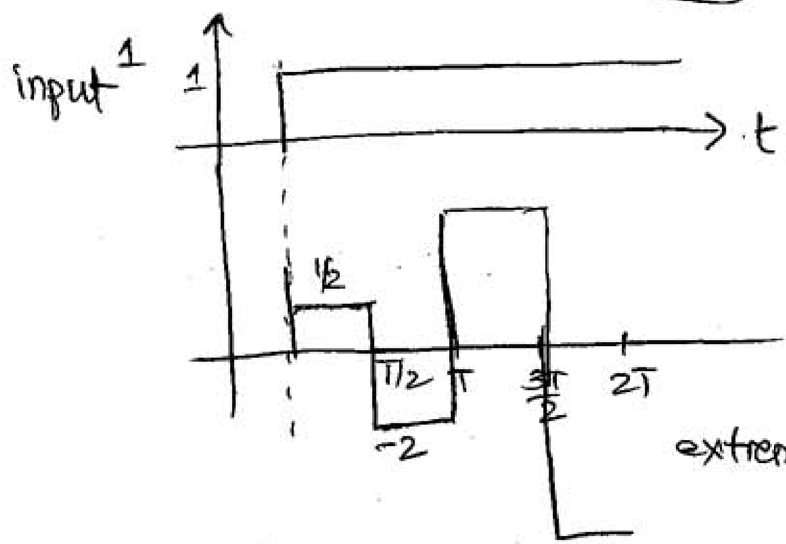
We already saw how the system attentuates to $1/3$

(depress sensitivity to input)

Reflex-related Disease

malfunction in feedback gain control.





extremely high output

Hypertonus – Usually caused by lesions in the brain causing high feedback gain @ the spinal cord.

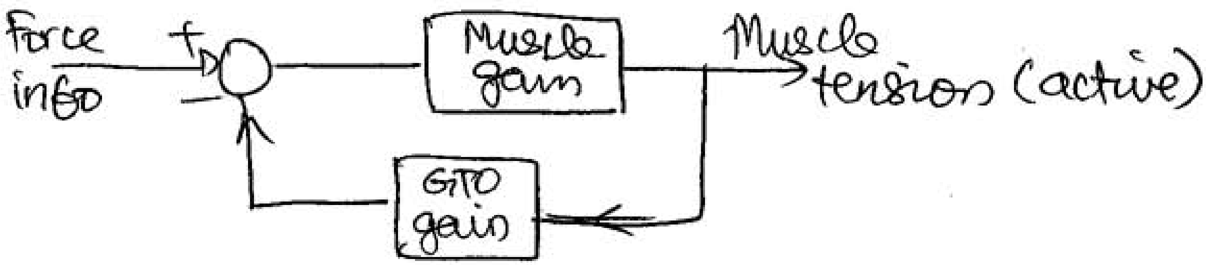
↳ stiff muscles

↳ most common hypertonus → spasticity in stroke patients

w/ any intentional mvts:

muscle move → stretch → tense up & join get locked comple

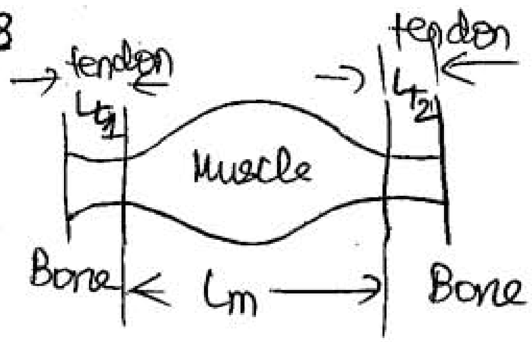
Another negative feedback sys: GTO



Because GTO detect only the active tension (rememb collagen stretch much less than muscle fiber)

- You'll see this in PS2

<u>Tendons</u>



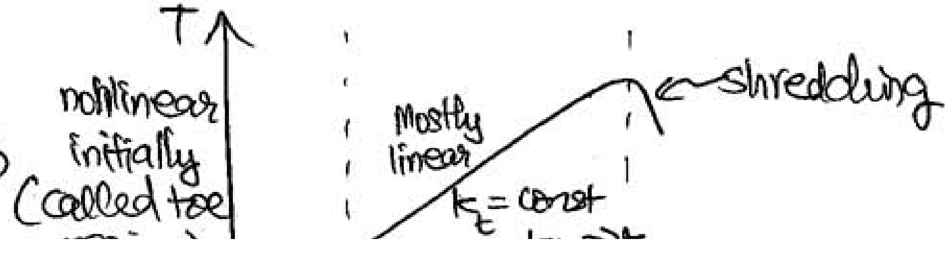
So far we have been calling $\Delta L_{total} = \Delta L_{muscle}$

because that's how most expts are done.

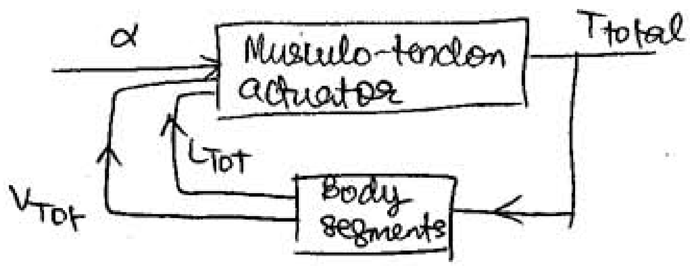
But in reality $\Delta L_{total} = \Delta L_m + \underbrace{\Delta L_{t1} + \Delta L_{t2}}_{\Delta L_{tendon}}$

What misinterpretation arises by ignoring tendons?

Isolated tendon tension-length curve.



Musculotendon Dynamics



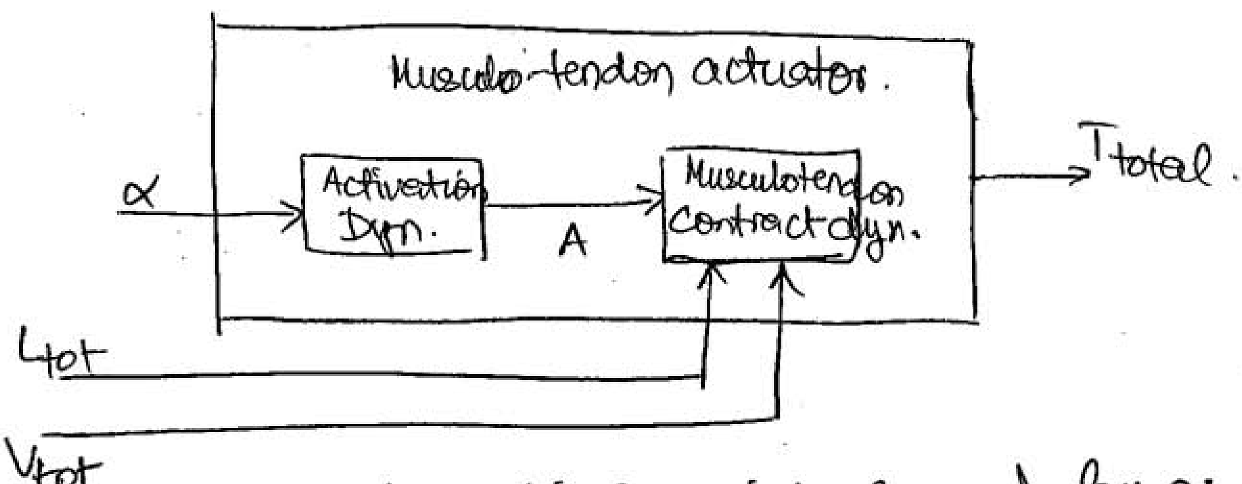
We've seen T Vs L

T Vs Vel.

$$L_{tot} = L_{ten} + L_m.$$

$$V_{tot\cancel{a}} = V_{tot} + V_m$$

Inside musculo-tendon actuator



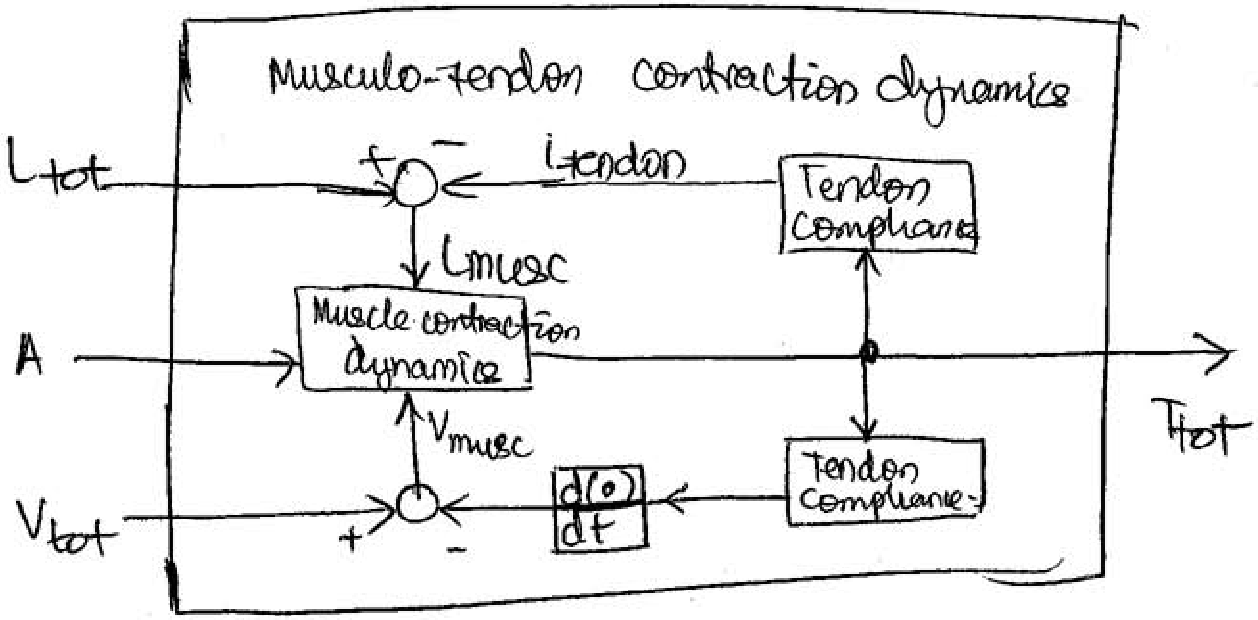
A is the activation state caused by α

(# of Ca^{2+}, efficiency of pumps, etc. can change the relnship btwn α & A)

A does not depend on L or V.

Contraction dynamics depend on L, V, & A.

Inside musculotendon contraction dynamics



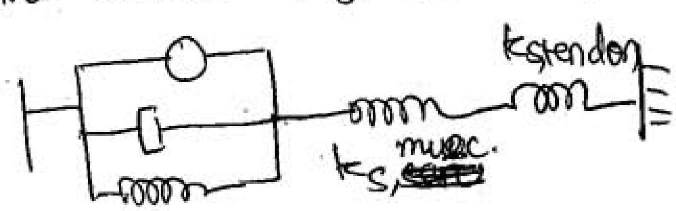
1) A changes muscle dynamics only.

2) Musc. tension is affected by A, L_m, & V_m.

3) Muscle tension = Tendon tension = T_{tot}

4) Tendon compliance does not affect T_{tot}

where is the tendon integrated in the model?



5) But tendon compliance changes L_t & V_t

↳ changes L_m & V_m, therefore changes muscle dynamics

Tendon compliance depends on ~~its~~ slack length (how long the tendon is)

Specified by ratio of the muscle they are attached to.

↳ slack length $L_t^{slack} = L_s = L_{tendon}$

L^s_t ranges from 0.12 to 16 for human.

Longest/largest ones are in human hands.

Remember we said

$$T = 0 \ @ \ 0.5\, l_0 \rightsquigarrow 0.5\, L_{mo}$$

In reality

$$T = 0 \ @ \ L_{tot} = 0.5\, L_{mo} + \underbrace{0.033\, L_{tendon}}_{\text{empirical}}$$

Normalize this:

$$\frac{L_{tot}}{L_{mo}} = 0.5\, \frac{L_{mo}}{L_{mo}} + 0.033\, \frac{L_{tendon}}{L_{mo}}$$

$$= 0.5 + 0.033\, L^s_t$$