Lecture 13

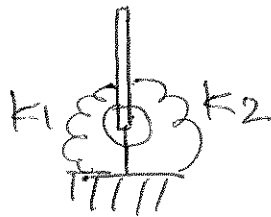Last lecture: Single joint control

       - effect of external load

       - " " co-contraction

       - Joint load on hip.

Today - More on single-joint control.

How do we control high-frequency input?

* inherent muscle stiffnesses add up.

~~Assume for simplicity a linear model~~

K_1 K_2 stiffnesses add up.

           (parallel.)

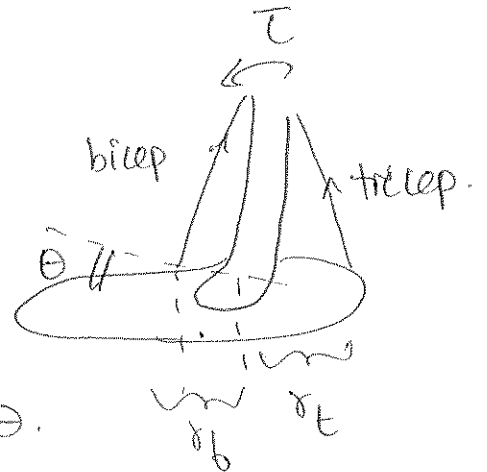   in addition to active ~~passive~~ stiffness

        due to α going up.

Let's do the math.

Suppose, muscle force has a linear model.

$$F = u + k(x - x_0)$$

contractile force

stiffness changes w/α

muscle length.

length @ rest.

$$\tau = F_t r_t - F_b r_b$$

Let's express $(x - x_0)$ in terms of θ.

$$x_b - x_{b0} \cong r_b \theta$$

$$x_t - x_{t0} \cong -r_t \theta$$

} approximation

$$\Rightarrow F_b = u_b + k_b r_b \theta$$

$$F_t = u_t - k_t r_t \theta$$

$$\tau = r_t(u_t - k_t r_t \theta) - r_b(u_b + k_b r_b \theta)$$

$$= (r_t u_t - r_b u_b) - (k_t r_t^2 + k_b r_b^2) \theta$$

force subtracts

stiffness adds

Ex. If $\tau_{ex} = 0$

      & muscle $k = 0$

$$\tau = I\overset{\circ\circ}{\theta} = r_E u_E - r_b u_b \qquad I s^2 \theta(s)$$

     $\downarrow$

    2 poles @ zero. $\Rightarrow$ sys unstable.

                       any perturbation would m
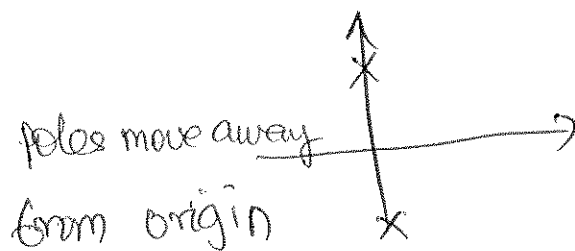
Even w/ damping in muscle & joint, still one pole @ ze

$$\tau = I\overset{\circ\circ}{\theta} + b\overset{\circ}{\theta} = r_E u_E - r_b u_b$$

But w/ stiffness

$$I\overset{\circ\circ}{\theta} = (r_E u_E - r_b u_b) - (k_E r_E^2 + k_b r_b^2)\theta$$

$$I\overset{\circ\circ}{\theta} + (k_E r_E^2 + k_b r_b^2)\theta = (\cdots -)$$

$$I s^2 \theta(s) + (\qquad\qquad) \theta(s)$$

poles move away
from origin
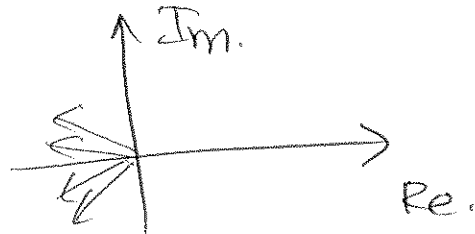
$\Rightarrow$ oscillates

Add damping.

$$I\ddot{\theta} + b\dot{\theta} + (k_t r_t^2 + k_b b^2)\theta = 0.$$

Ex: $\tau_{ex} = \tau_{grav}.$

$$I\ddot{\theta} = \tau_m + mgh\, s\beta \sin\theta \quad \leftarrow \text{use small angle assumption}$$

$$I\ddot{\theta} + ((k_t r_t^2 + k_b r_b^2) - mgh\, s\beta\, c\theta_0)\theta$$

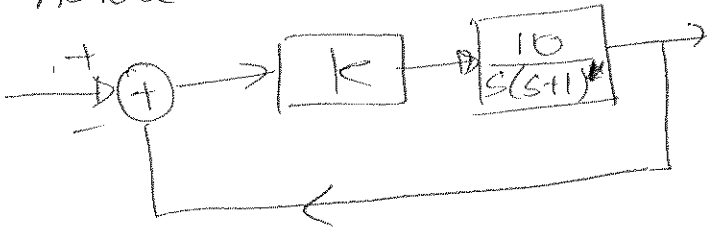$$= (r_t u_t - r_b u_b) + mgh\, s\beta\, s\theta_0$$

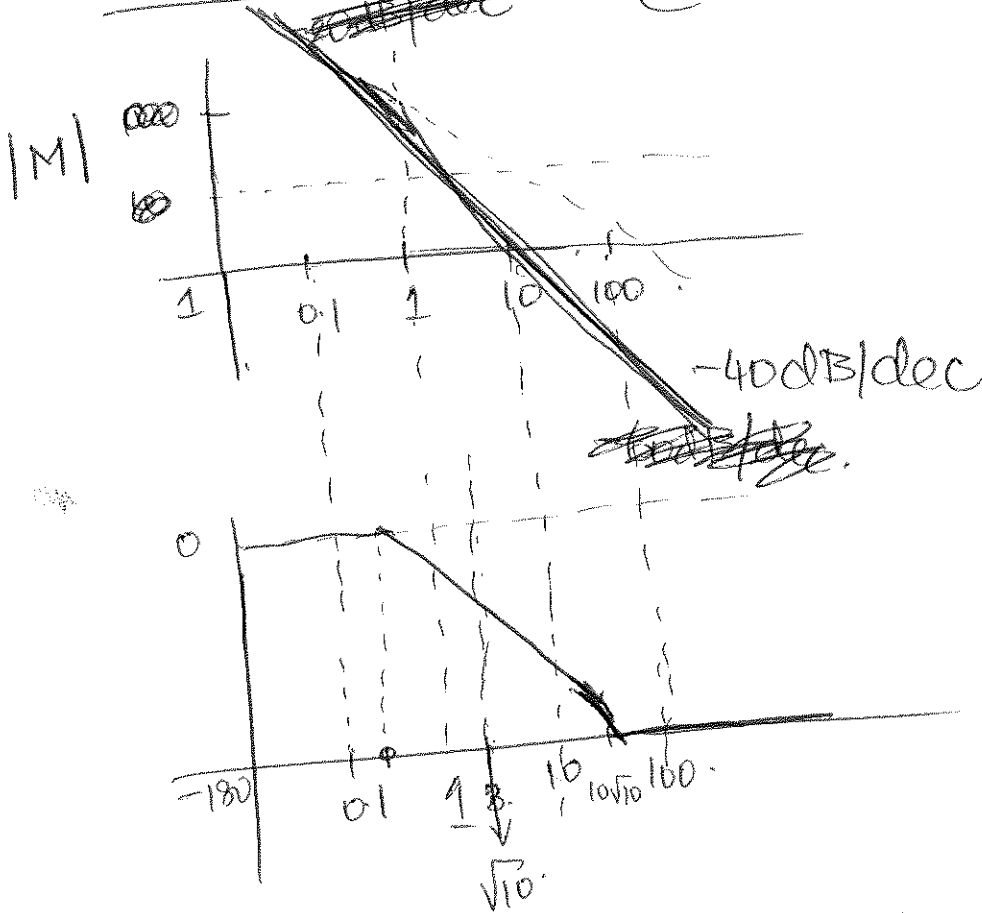If +ve $\Rightarrow$ stable.
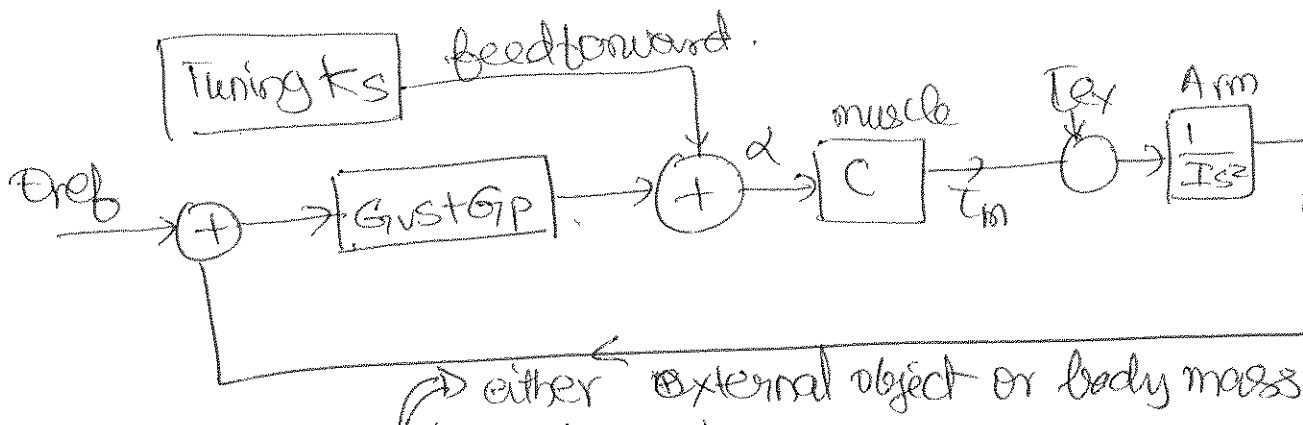
Note that as $m\uparrow$, k_t & k_b must go up.

(neck holding head mass } maintain posture against gravity
lower back " torso

Aside on some Control Methods

$$\frac{G}{1+GH}$$

$$\frac{G}{1+G}$$

$$\frac{10/s(s+1)^2}{1+\frac{10}{s(s+1)^2}}$$

$$\frac{10}{s(s+1)^2+10}$$

$$\frac{10}{s^2+s+10}$$

Bode plot   @ K=1

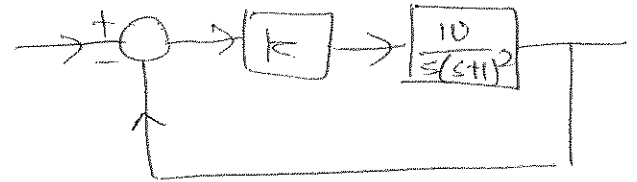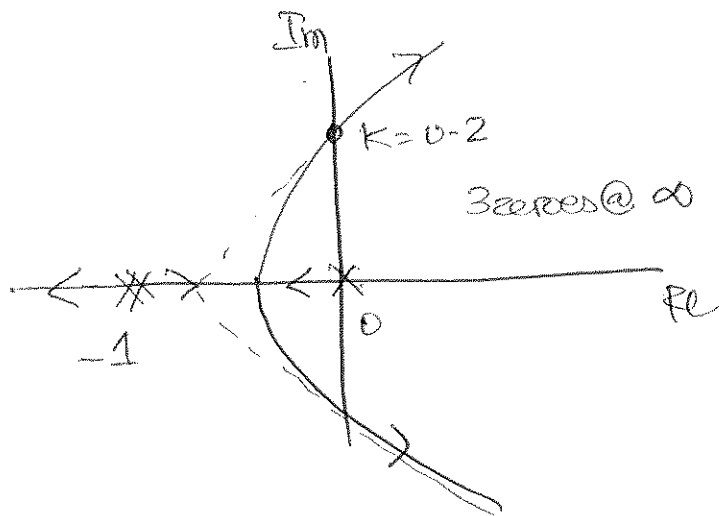$-40dB/dec$

$\sqrt{10}$

Find stability criterion for K:

~~find K such that~~

(gain margin, phase margin)

↓

The amt. of shift in magn. plot ~~such that gain=0dB~~ available

when phase = 1?

in order to make gain = 0

phase margin: the amt. of shift in phase curve

when gain = 0dB

Root locus : observe how the poles move around w/ varying k.

$K = 0.2$

3 zeros @ ∞

-1

Re

* Symmetric about x-axis

* ~~# of paths~~ ~~# pole~~

* start @ poles, end @ zeros.

Find @ what k values, $\underset{\lambda}{\overset{C.L}{\text{poles}}}$ poles move to RHP.

Characteristic eqn:

$$1 + k \frac{10}{s(s+1)^2} = 0.$$

$\downarrow$

open loop poles / zeros

Nyquist Plot : Another way to tell stability from the loop gain function : Plot real and Im parts of GH for different frequencies

Unstable if $N + P_0 > 0$

$\uparrow$ $\uparrow$

# of poles of $GH(s)$ in RHP

# of CW
encirclements of
$\bigcirc$ $(-1,0)$

(CCW $\Rightarrow -$)

So   why don't we have high K's all the time?

Remember α must go up to get high K.

Not energy efficient.

So Ks must be tuned to be just enough to reject

instability

  — if that's what our nervous system cares to

optimize

Also by always using high α, you lose out on dyn

range.

  —Dynamic Optimization theory ... soon.

A quick summary of 1st half of the course.   (P8)

Anatomy

Mechanics

Muscle fiber mechanics —— tension-length.
—— tension velocity

Muscle spindle — velocity + position   fast-fatigue fiber
Ia & II   slow "   "

Golgi Tendon organ — Tension
Ib

All included in Hill-like models (linear, simple)
— good estimation, but not accurate
↳ more detailed models exist, but better to be simple
w/ 400+ muscles in the body.

Neural loops
● reflex path : —— mono-synaptic connxns | ●Forward path
—— interneurons
└ —— inhibitory neurons
└ —— reciprocal activation

● Delays — cause instability
└ compensation ~~by~~ through co-contraction (K↑).

--All lower models can be modeled, but the effect of them are highly variable & not fully understood yet

　　　- (variables are fatigue, adaptation)

Conclusions :

- Components + simple local neural control have simplifi models

- Combining them to describe higher level motor controls se hard

- Is that the only way to study higher level motor con

　　　↳ Don't include all , components + treat them as i
　　　　　　　↓
　　　　　　individual　　　　　　　　　　　　　　　un

　　　　- Find things that are Quantifiable/measurable the high-level & conduct those expts.

　　- This is what we will study in the rest of the cou

Example : • Dynamic Optimization Theory.

     • Higher-level single-joint control

     •   "   "   multi - "   "

     • Important quantifiable common mvt. features

     • Motor learning/adaptation

     • Cortical representation

          - What does brain encode?

          - Which parts of brain involved?

     o Lesion studies, motor-related disease

— Would be nice if we can draw a complete control diagram of motor control.
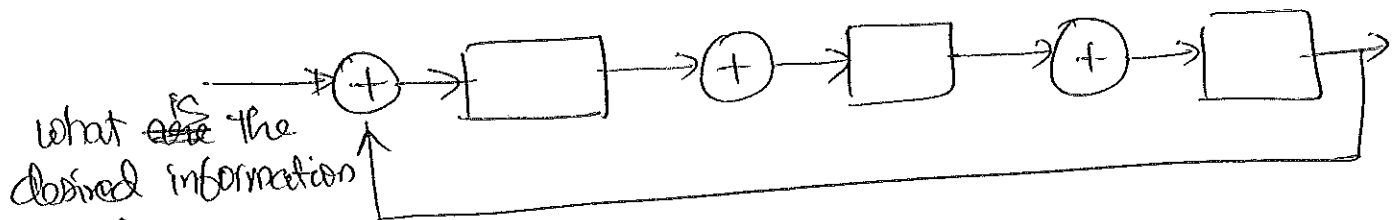
What is the desired information
θ_i?
x?
vel?
F?

    - What's being corrected?

    - What are in each box? How many boxes

    - What are the inputs/outputs?

First attempt in explaining how mvts are controlled.

- eq point theory.

- Feldman (1966-1974) then Bizzi (1981-83)

- Muscles have inherent K, b

↳ a well-behaved stability property

= there is an "attractor set" to which the sys. returns (or tends to return) when perturbed.

Single muscle → when $\alpha = 0$, "attractor set" @ rest.

→ As α changes, "a.s" changes.

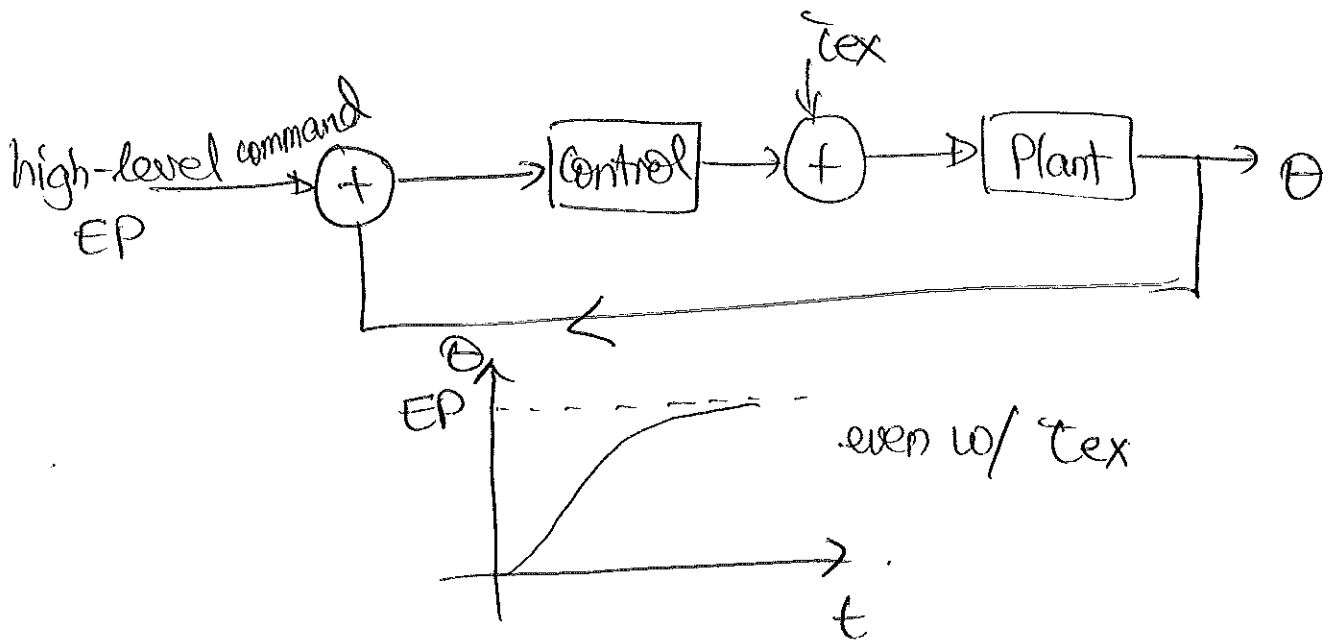Single joint — driving an inertia w/ neuromuscular sys. (w/ springs + dampers)

Equilibrium point : the pos'n to which the neuromuscular
‾‾‾‾‾‾‾‾‾‾‾‾‾(EP) sys. is driving the limb.

Tension

EP

Tension-length curves for biceps

joint angle.
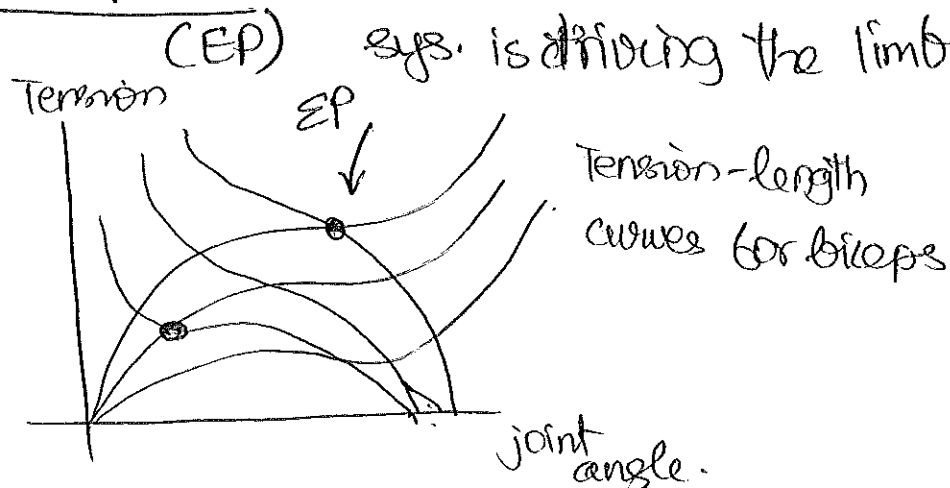
— Final position control Hypothesis

"High-level commands" specify the EP & the neuro-mus[c]

sys. (k & b) gets the joint there.

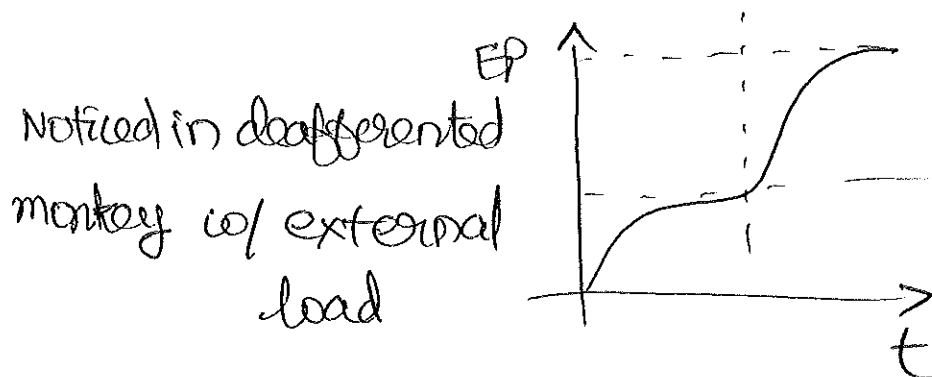high-level command
EP
$\rightarrow \bigoplus \rightarrow \boxed{Control} \rightarrow \bigoplus \rightarrow \boxed{Plant} \rightarrow \theta$

τ_{ex}

.even w/ τ_{ex}

Use error-signal to correct for τ_{ex}

w/o feedback loop

EP $\rightarrow \bigoplus \rightarrow \boxed{C} \rightarrow \bigoplus \rightarrow \boxed{P} \rightarrow \theta$

τ_{ex}

$\tau_m + \tau_{ext} = \tau_{tot}$

If τ_{ex} is suddenly removed:
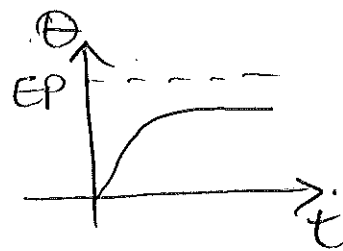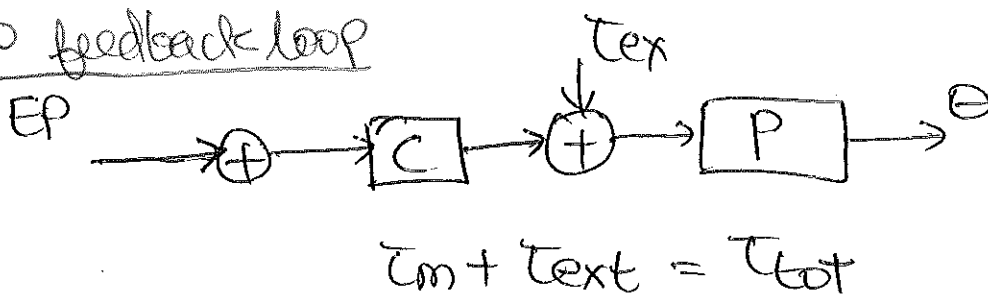
Noticed in deafferented
monkey w/ external
load

But deafferented monkeys could also move fast or slow
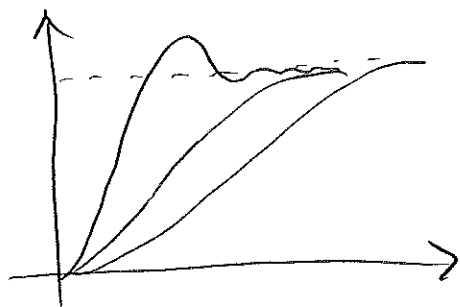
So this could not be explained by just one EP.

"Virtual Trajectory theory"

- the CNS specifies a series of EPs and the limbs spring-like behavior take care of the dynamics.

Why ~~EP~~ "Virtual"?

- Actual location of the limb may not be @ EP if

① EP is fixed + external load/constraints are applied

② EP moves w/ time + the limb does not catch up w/ virtual trajectory.

Quantitative analysis shortly.