Last time poroelastic → electrokinetics

$$\boxed{\frac{\partial u_1}{\partial t} = Hk' \frac{\partial^2 u_1}{\partial x_1^2} + U_0 + \frac{k_{12}}{k_{22}} J_0}$$

(1) $\sigma_{11} = (2G + \lambda)\,\varepsilon_{11} - p$

(2) $\frac{\partial \sigma_{11}}{\partial x_1} = 0$

(3) $U_1 = -\frac{\partial u_1}{\partial t} + U_0$

(4) $U_1 = -k_{11}\frac{\partial p}{\partial x_1} + k_{12}\frac{\partial V}{\partial x_1}$ Darcy ...

(5) $J_1 = k_{21}\frac{\partial p}{\partial x_1} - k_{22}\frac{\partial V}{\partial x_1}$ Ohm ...

(6) $\frac{\partial J_1}{\partial x_1} = 0$ charge relaxation is $\tau \sim$ ns

- neglect inertia - solid $L/\lambda \gg 1$
  - fluid $Re \ll 1$

$k' \equiv k_{11} - \frac{k_{21}k_{12}}{k_{22}} > 0$ positive diffusivity

- new effects - electroosmotic flows flows generated by ΔV
  - streaming potentials ΔV generated by flow

electrokinetics ⇒ fixed charge density (cartilage, cornea, skin, artery, extracellular matrix ECM, ...)
e.g. $[NH_3^+]$, $[COO^-]$, $[SO_3^-]$ from GAGs

- not covered: osmotic pressure, dependence of k, λ, G on concentration

Today: transition to cell mechanics

molecules (nm, pN) — statistical mechanics, single molecules

tissues (cm, mN - N) — continuum mechanics, poro / visco-elastic materials

- cells
  - molecular motors
  - ion channels
  - cytoskeletal filaments

continuum mechanics
CSK as a continuum
CSK filaments

- mechanics: why cells?

cartilage: its cell content doesn't really affect its stiffness, its mechanical behavior
but cells respond to mechanical deformation "cells go along for the ride"

arterial wall: mechanotransduction (response of endothelial cells to fluid shear stress → atherosclerosis)
smooth muscle cells (contractile, can redistribute the flow)

muscle: cardiac / skeletal muscle cells, smooth muscle cells

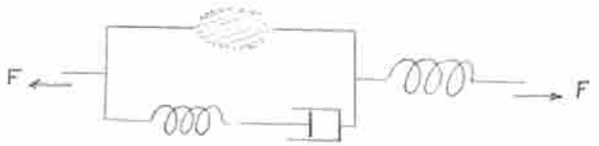
airway wall: mechanotransduction (epithelial cells)

▷ Force / biology interaction

- Prototypical example: muscle
  - anatomy / macroscopic behavior
  - activation / contraction
  - cross-bridge dynamics (Huxley)

see slides

- Macroscopic functions of muscles
  - tetanus = maximum contracted state
  - exponentially stiffening behavior $\frac{d\sigma}{d\varepsilon} = \alpha(\sigma + \beta) \Rightarrow \sigma = C\exp(\alpha\varepsilon) + \beta$



contractile + viscoelastic

  - how much force can be generated depending on the velocity on force development ?
  zero if too fast
  Hill's equations $\frac{v}{v_{max}} = \frac{1 - \frac{F}{F_{max}}}{1 + C\frac{F}{F_{max}}}$ purely empirical, $\left\{\begin{array}{l}\text{true for all muscles} \\ \text{normalized}\end{array}\right.$

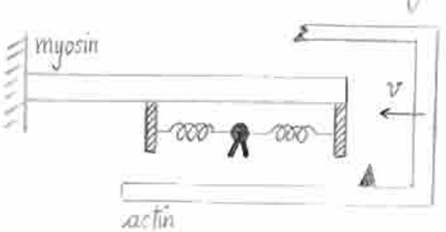
  efficiency $\eta \equiv \frac{\text{mechanical work}}{\text{chemical energy use}} \simeq 25\ \%$ (comparable to car)

  - $\Delta G \simeq 25\ k_B T$ per molecule
  step size (myosin on actin) $\Delta x = 5$ nm and $F \sim 3\text{-}4$ pN

- Activation / contraction
  - depolarization (by nervous cell) conducted transversely $\Rightarrow$ Ca^{2+} ions released $\Rightarrow$ contraction
    ↳ from sarcoplasmic reticulum all around
  after contraction is completed, Ca^{2+} sequestration in SR.
  - sliding filament model thick filament ≡ myosin, walks along the actin
    thin filament ≡ actin

  dark = overlap (A band) + light = actin only (I band)
  A Huxley - Niedergerke
  H. Huxley - Hanson simultaneously in Nature in 1954
  ATP-dependent conformational change → power stroke and displacement toward the (+) end of actin
  Ca^{2+} removes the $\left\{\begin{array}{l}\text{tropomyosin} \\ \text{troponin}\end{array}\right.$ hindrance $\Rightarrow$ walk possible

  Model by Jonathan Howard (following article by Pate, 1993)



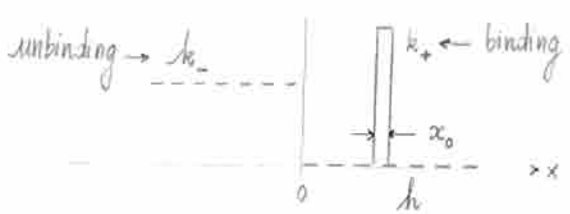
myosin head

actin binding site

$n(x,t)$ probability of binding

$dn = \cancel{\frac{\partial n}{\partial t}}\, dt + \frac{\partial n}{\partial x}\, dx$

0 at steady state



$\frac{dn}{dt} = \frac{dx}{dt}\frac{\partial n}{\partial x} = -v\,\frac{\partial n}{\partial x}$ for different x regions