Systems Neuroscience NOTES

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06 Mar 2021

INTRO

Readings / Tests / Course Outline

Predispositions / folk theory vs sci see how things are

10%

cell doctrin

smell brain

sex diffs

L/R

Taking notes

attn / mult-task analogy

type vs write

drawing

Separate anat

400 parts ..

repeat

3 planes / nissl-myelin

Golgi

Evolution

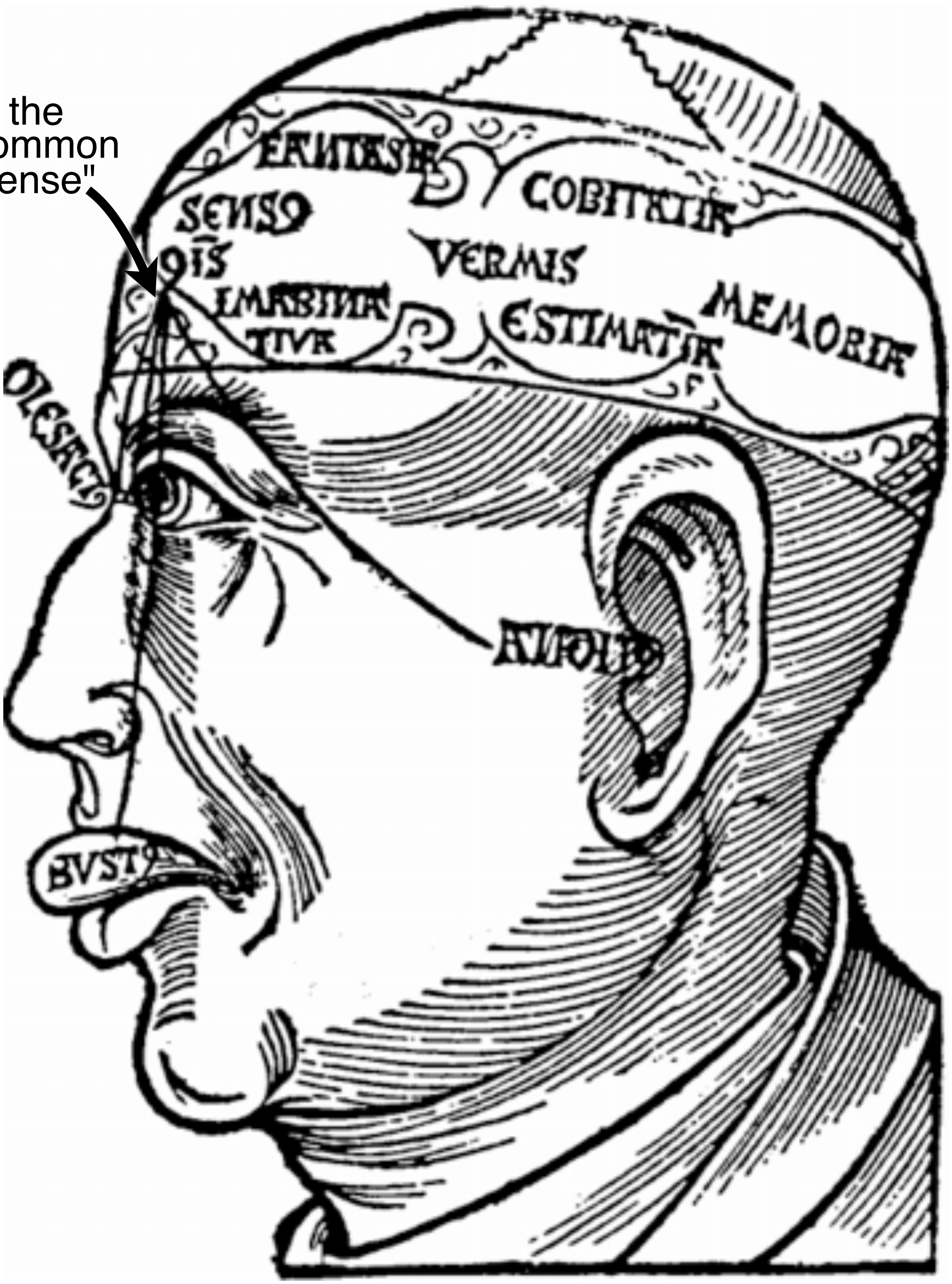
comparative function

Machines < not directly for machine learning
for engineer given biological raw material

- class summary
- signal/syst approch
  - machine learning (cf. multitask!)
    vs.
    engineer using bio material!
- folk theory
  - Aristotle (steam eng.)
  - Descartes (pineal cross bar)
  - *Cell Doctrine (cog sci)
  - Phrenology/Barnum
- modern folk theory
  - 10%
  - Smell brain
  - sex diffs]
  - L/R diffs]

The "Cell Doctrine"

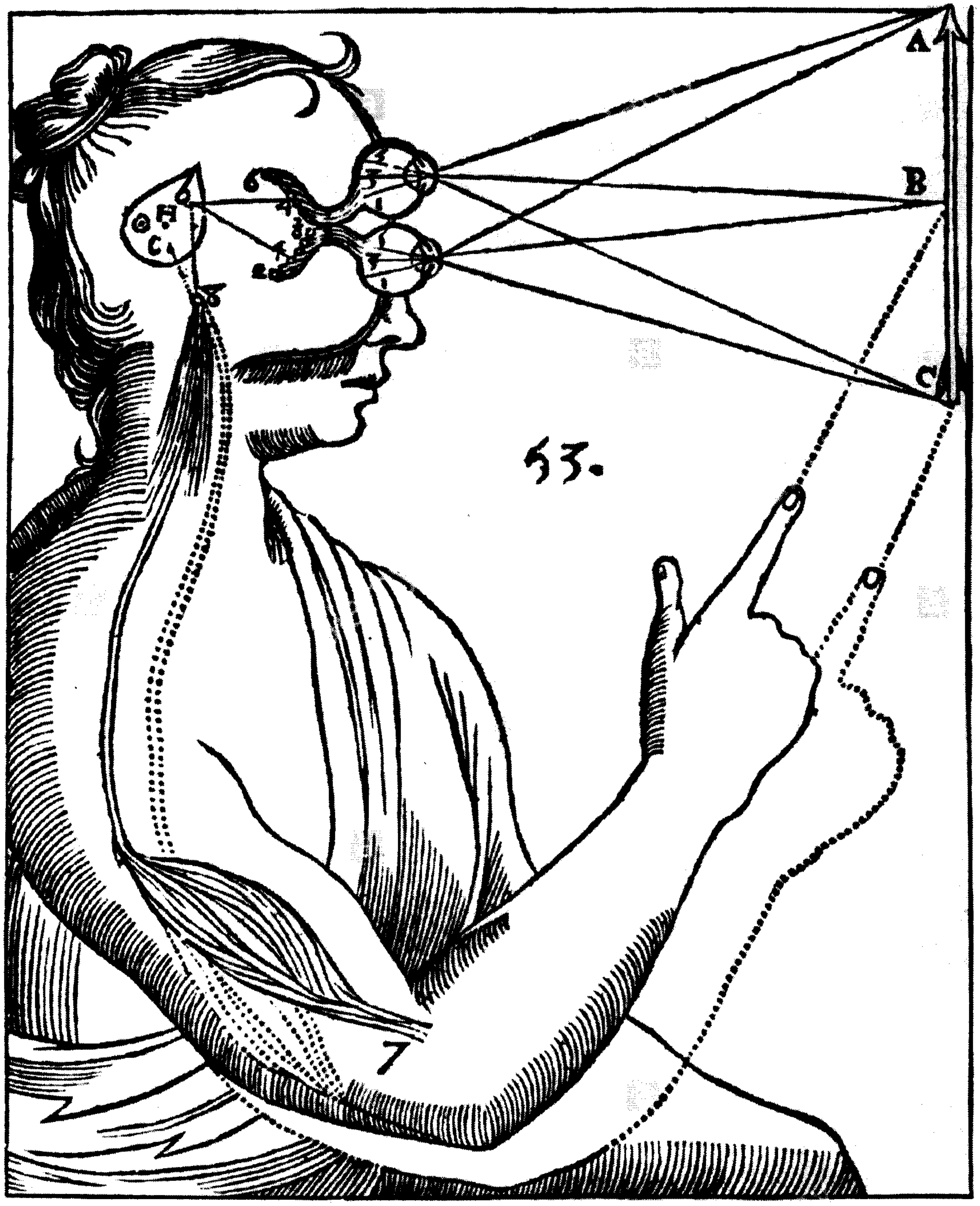
(default theory of human cognition in absence of any information about brain organization)



the "common sense"

from Descartes

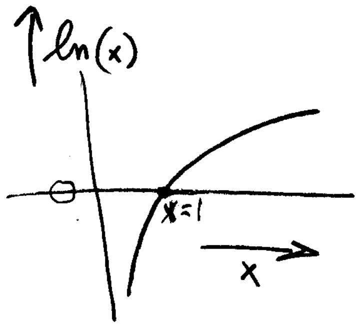
(a pneumatic crossbar switch in
the pineal controlled by the mind)



Nernst Potential

natural log

$$E_{ion} = \frac{RT}{F} \cdot \frac{1}{z} \cdot \ln \left[\frac{[Ion]_{out}}{[Ion]_{in}} \right]$$



$\downarrow$

Nernst eq: → reversal potential

or

equilibrium potential

or

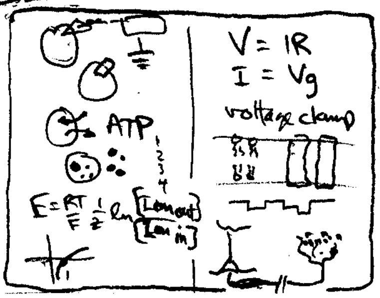
the membrane potential at which **no** current flows when channels for this ion are opened

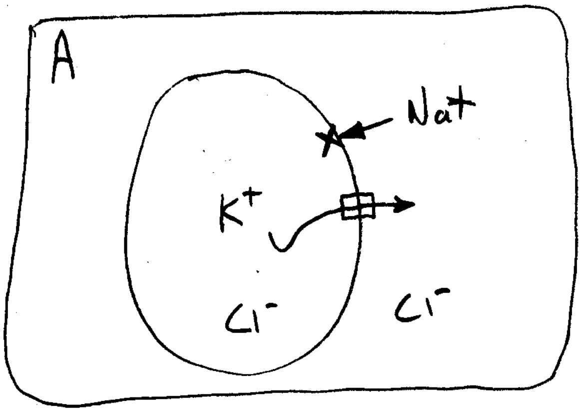
gas const
temp Kelvin

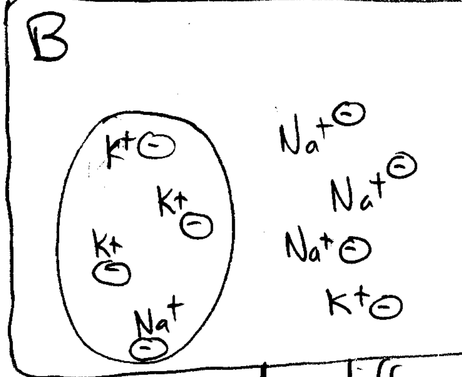
$$\frac{RT}{F} \approx 26 \text{ mv}$$

Faraday const

Summary







- start at 0, balanced
- a few K+'s leak out
- inside is negative

1 - differential concentration

2 - **semi** permeable membrane

3 - resting potential is balance between diffusion & potential diff

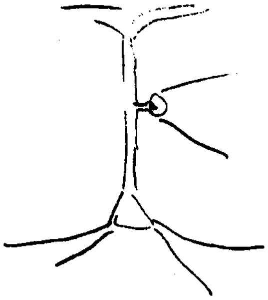
4 - requires membrane channels but **no** **voltage** or **neurotransmitter** gating

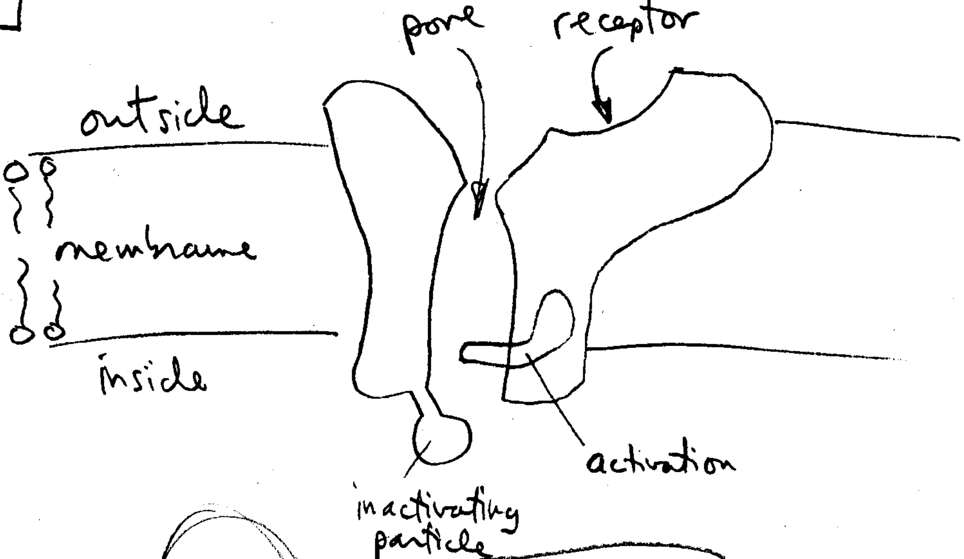
Important Point:

→ **Nernst** potential describes a target voltage for an ion

→ the present membrane potential is affected by other things!

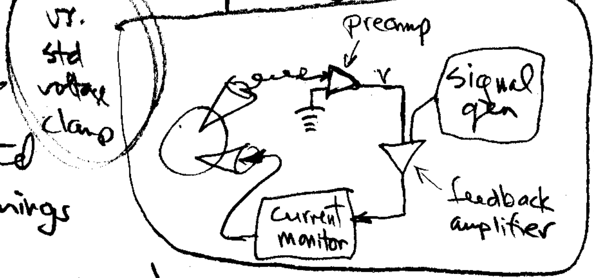
CHANNELS

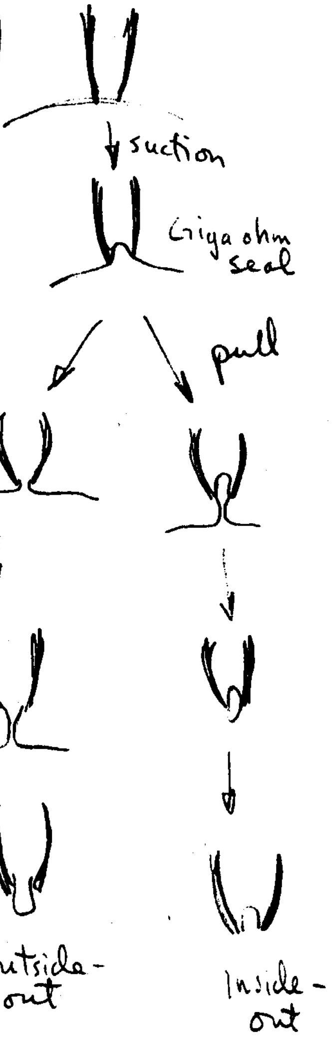


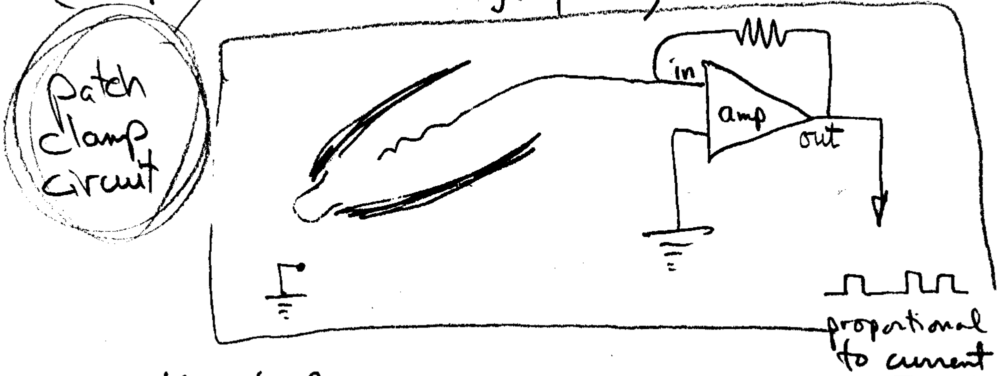


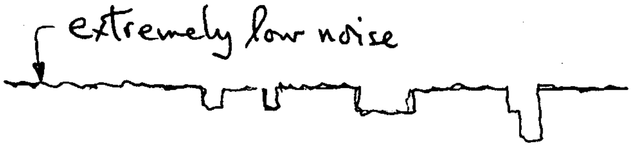
Patch clamp

- electric current associated w/ single channel openings
- (clamp for feedback reg. of V)





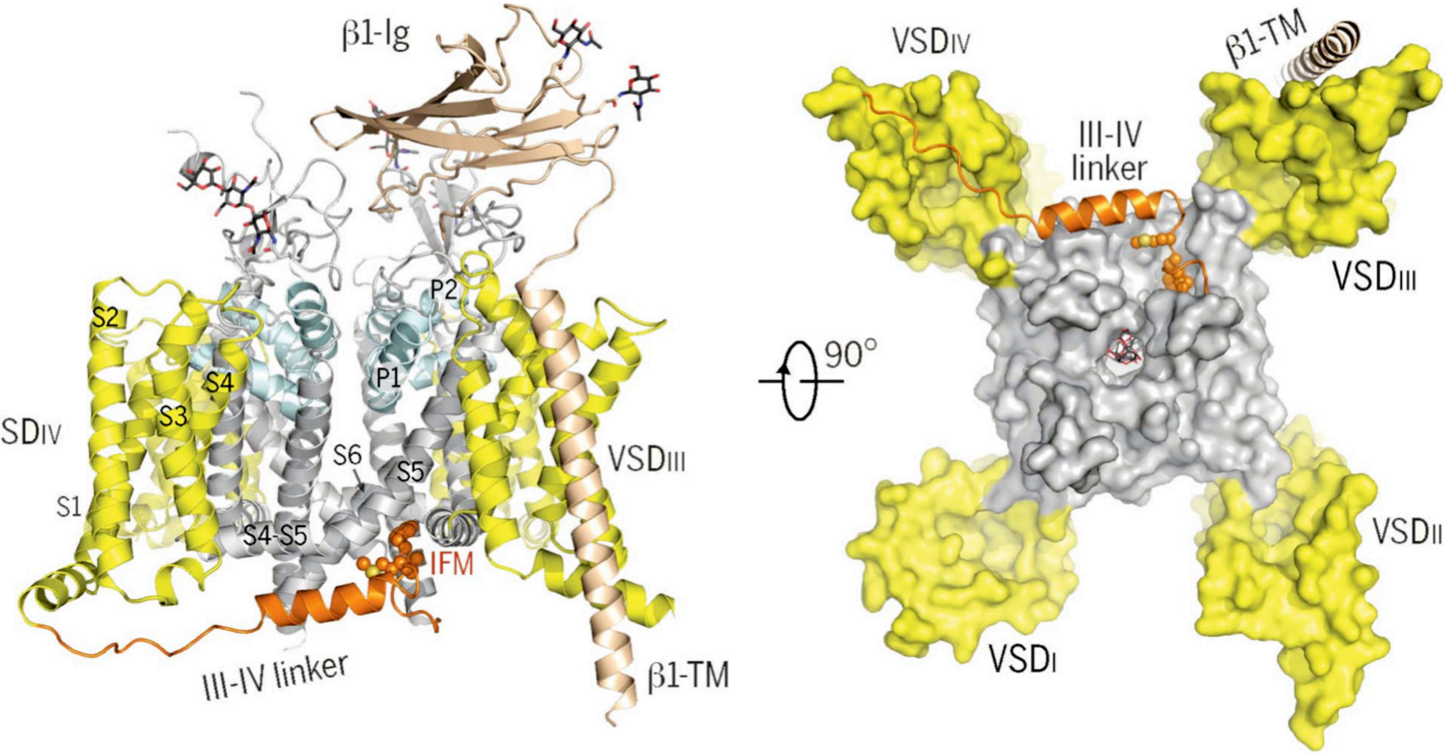




- individual channel openings visualized
- 7000 ions/event (10^7/sec)
- conductance: ~20 pS
- density: 100-1000/μm²

S is Siemens

$$\text{conductance (siemens)} = \frac{\text{current (amps)}}{\text{voltage (volts)}}$$



Structure of the human Na_v1.4-β1 complex. Two perpendicular views are shown. Left: Side view in ribbon cartoon. The VSDs are colored yellow, and the selectivity filter and supporting helices P1 and P2 are colored light cyan. The IFM motif is shown as spheres, and the III-IV linker is colored orange. The transmembrane segments in repeat IV are labeled. Right: Surface presentation for the bottom view to highlight the intracellular gate and the cavity that accommodates the IFM motif. The GDN molecule that penetrates the intracellular gate is shown as thin sticks.

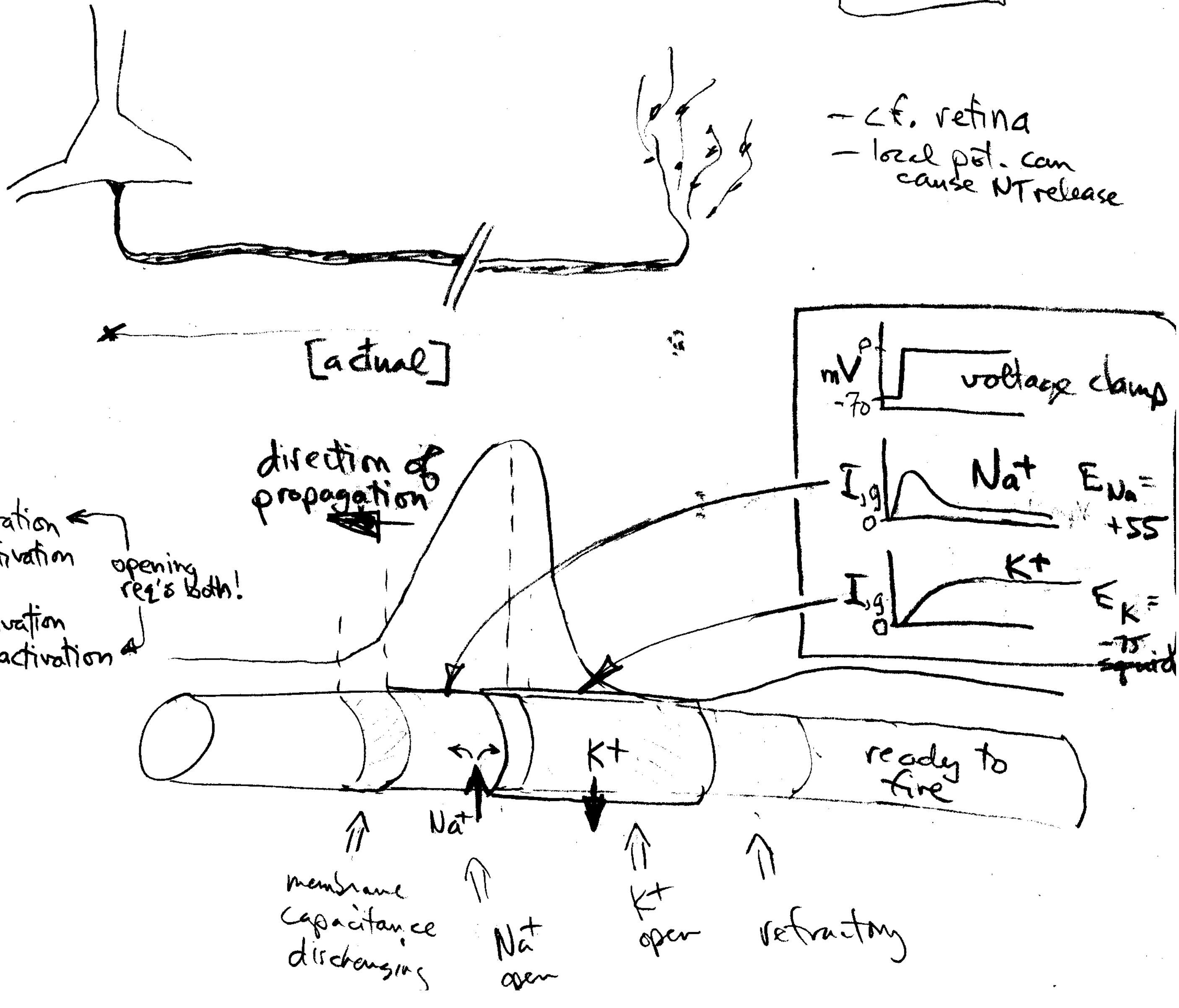
Action Potential

- reason fr action potential => passive flow dies out too quickly

- requires voltage-gated channels

[voltage-gated Na^+ (I_{Na})
voltage-gated K^+ (I_K)

Ohm's law
$V = IR$
$V = I/g$
$I = Vg$



- cf. retina
- local pot. can cause NT release

$E_{Na} = +55$

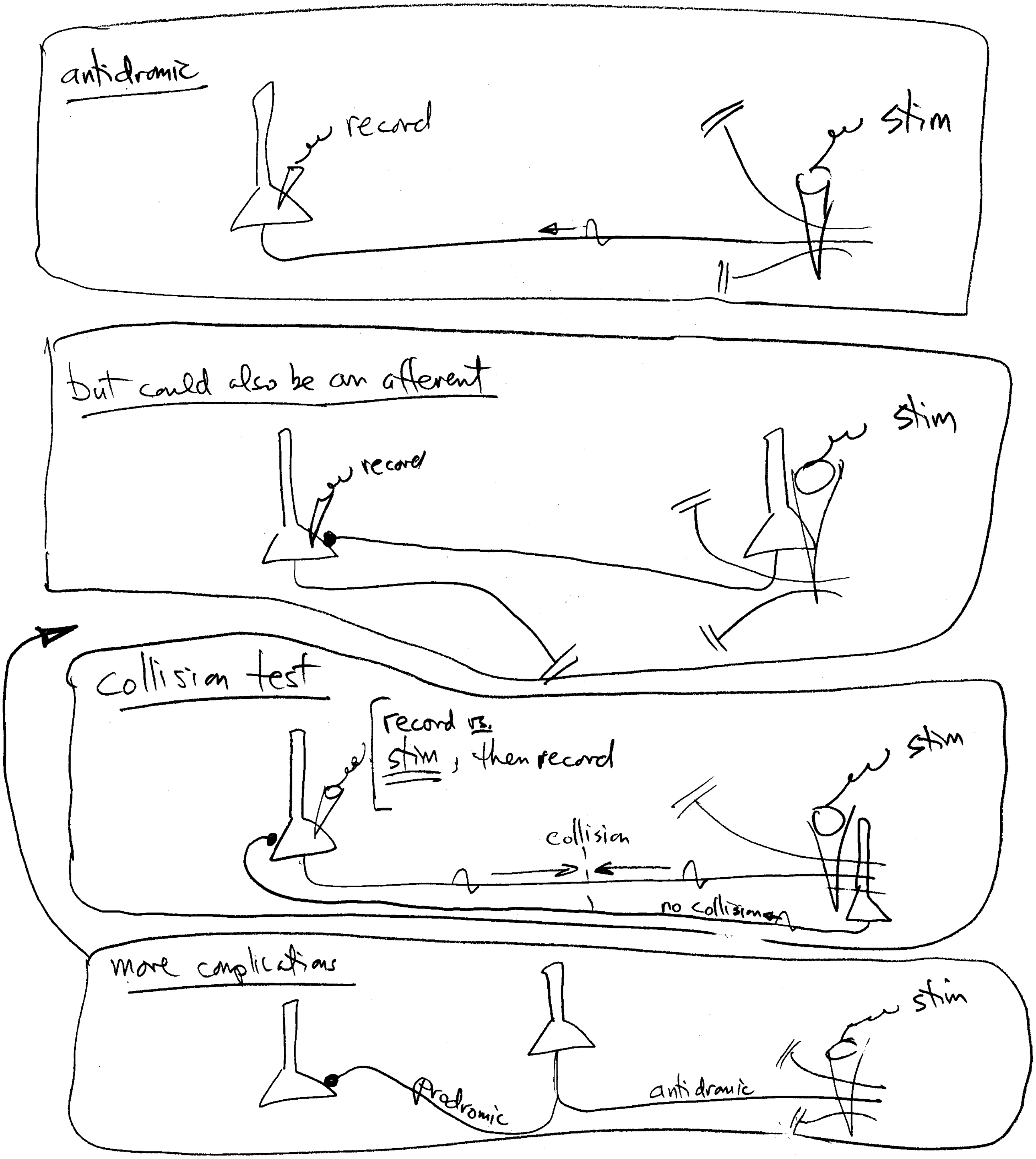
$E_K = $ ~~-75 squid~~

[activation
deactivation

[inactivation
deinactivation

opening req's both!

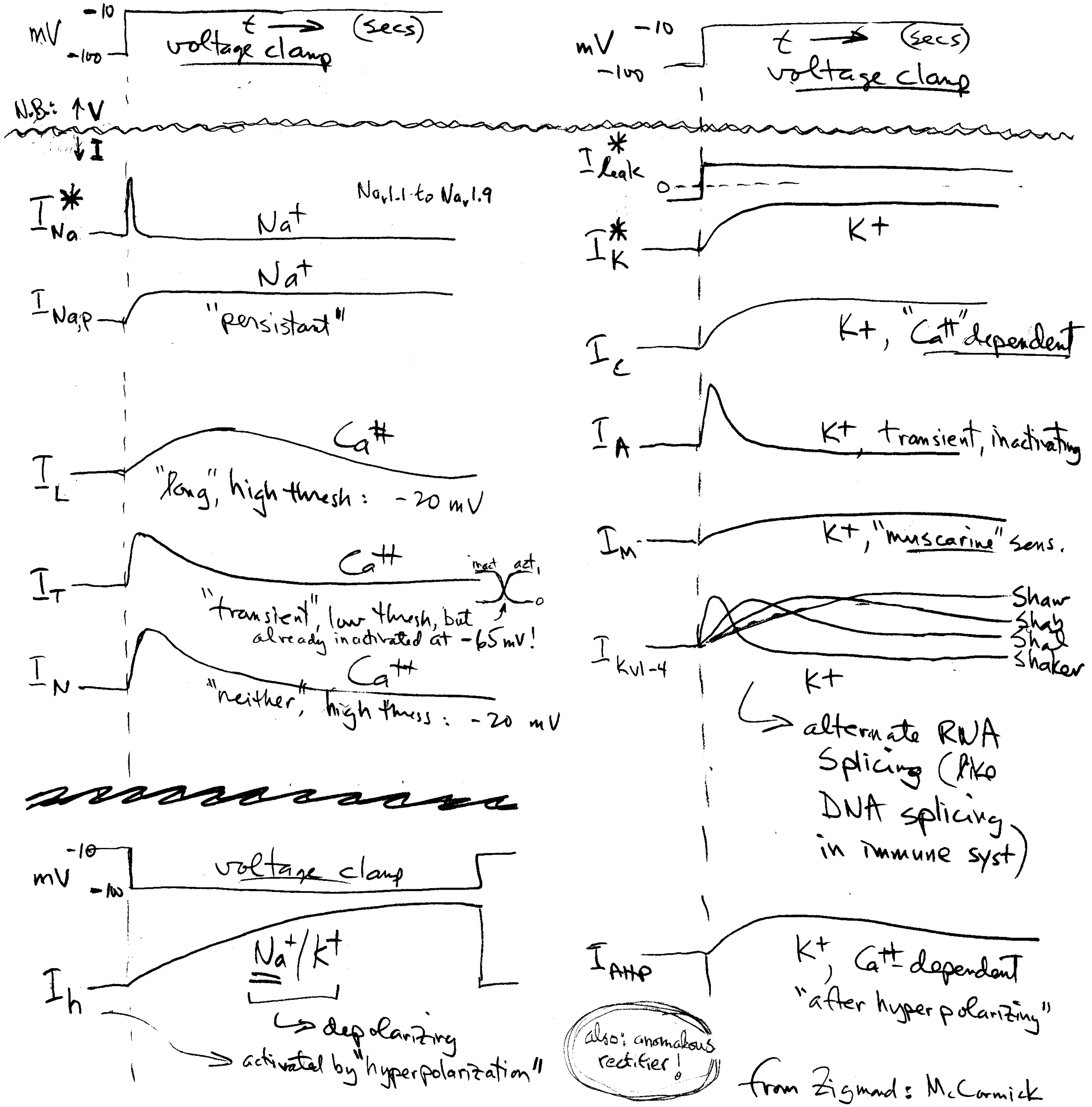
Collision test to verify antidromic



Other Voltage-Sens Neuronal Ioniz Currents

Excitatory ("inward") Na+, Ca++ currents

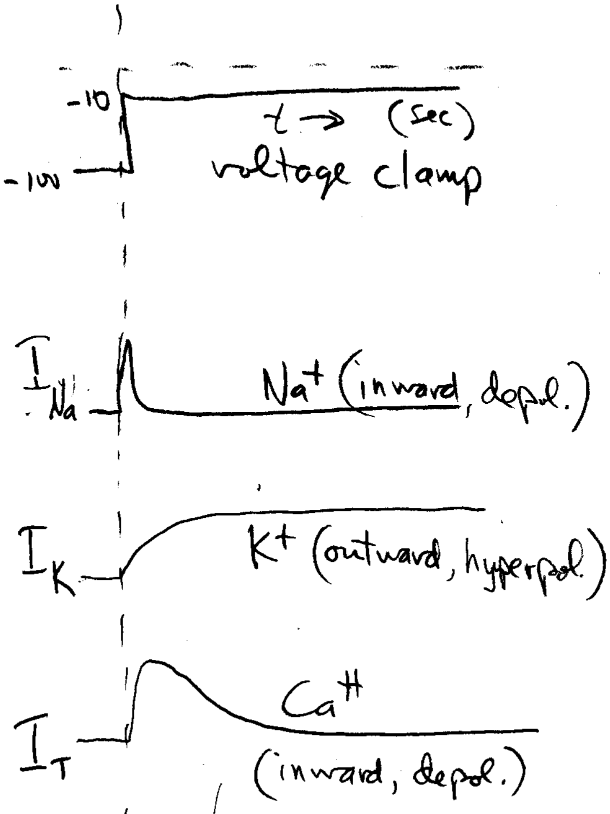
Inhibitory ("outward") K+ currents

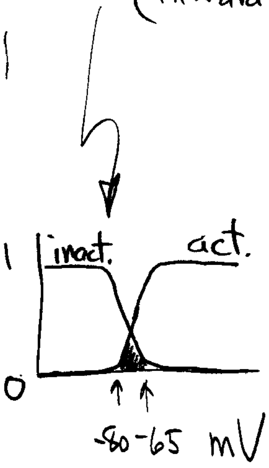


from Zigmond: McCormick

How I_T ("transient") leads to bursting

voltage clamp



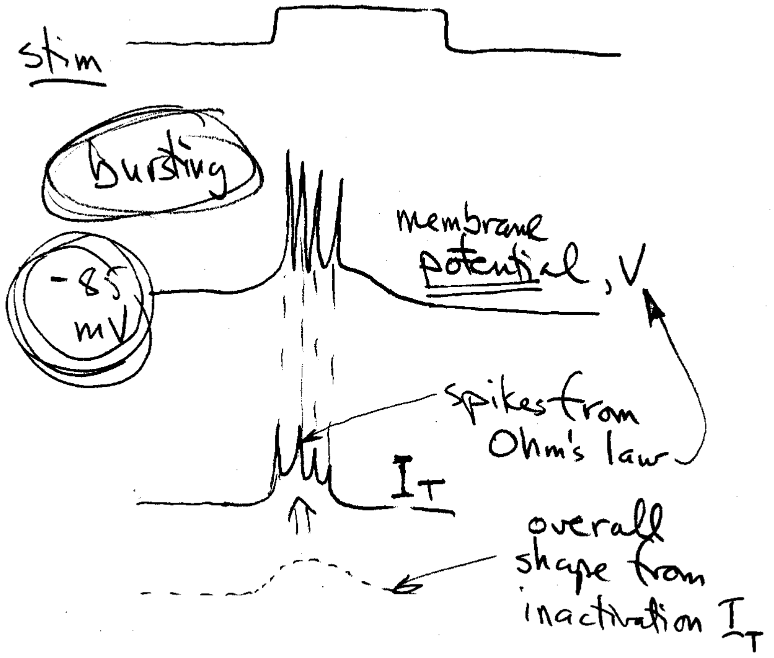


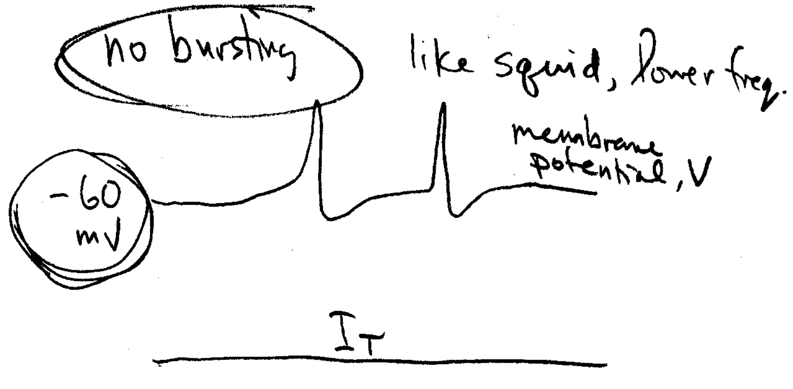
N.B. also time dep. diffs

H-H

vs. I_{Na}, higher volt before inact.

no voltage clamp
simple stim + 2 diff bias



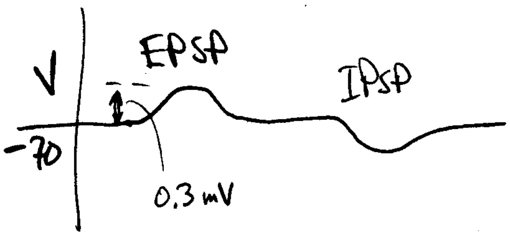


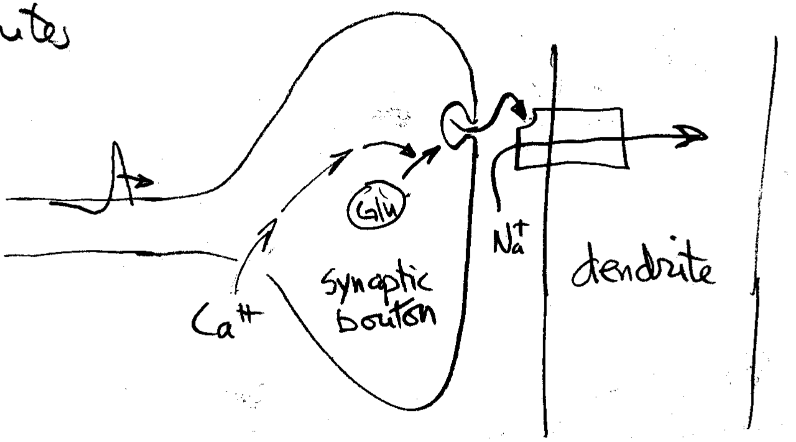
Another example:

I_A (outward) can cause delayed firing (firing starts after it inactivates)

Synaptic Potentials

- caused by neurotransmitter-gated channels
- mostly on dendrites





AMPA glutamate-gated Na^+

- "excitatory sodium"
- passes K^+ as well
- $\boxed{E_{Na} \approx 0 \text{ mv}}$
- not voltage-sensitive

NMDA glutamate-gated Na^+, Ca^{++}

- excitatory
- also passes Ca^{++}
- voltage-dependent
- blocked at rest

$GABA_B$ K^+

↳ note: if resting pot. due entirely to K+, then these would not by hyperpolarizing

- hyperpolarizing inhibitory
- slower than AMPA

$\boxed{E_K \approx -100}$ (mammalian)

$GABA_A$ Cl^-

- shunting inhibitory
- no effect at rest

$\boxed{E_{Cl} \approx -75}$ = rest

Channel Families

4+1

nAChR (nicotinic acetyl choline receptor)
- 5-HT3
- *GABA$_A$

4

G ionotropic
- non-NMDA ⇐
- *NMDA ⇐

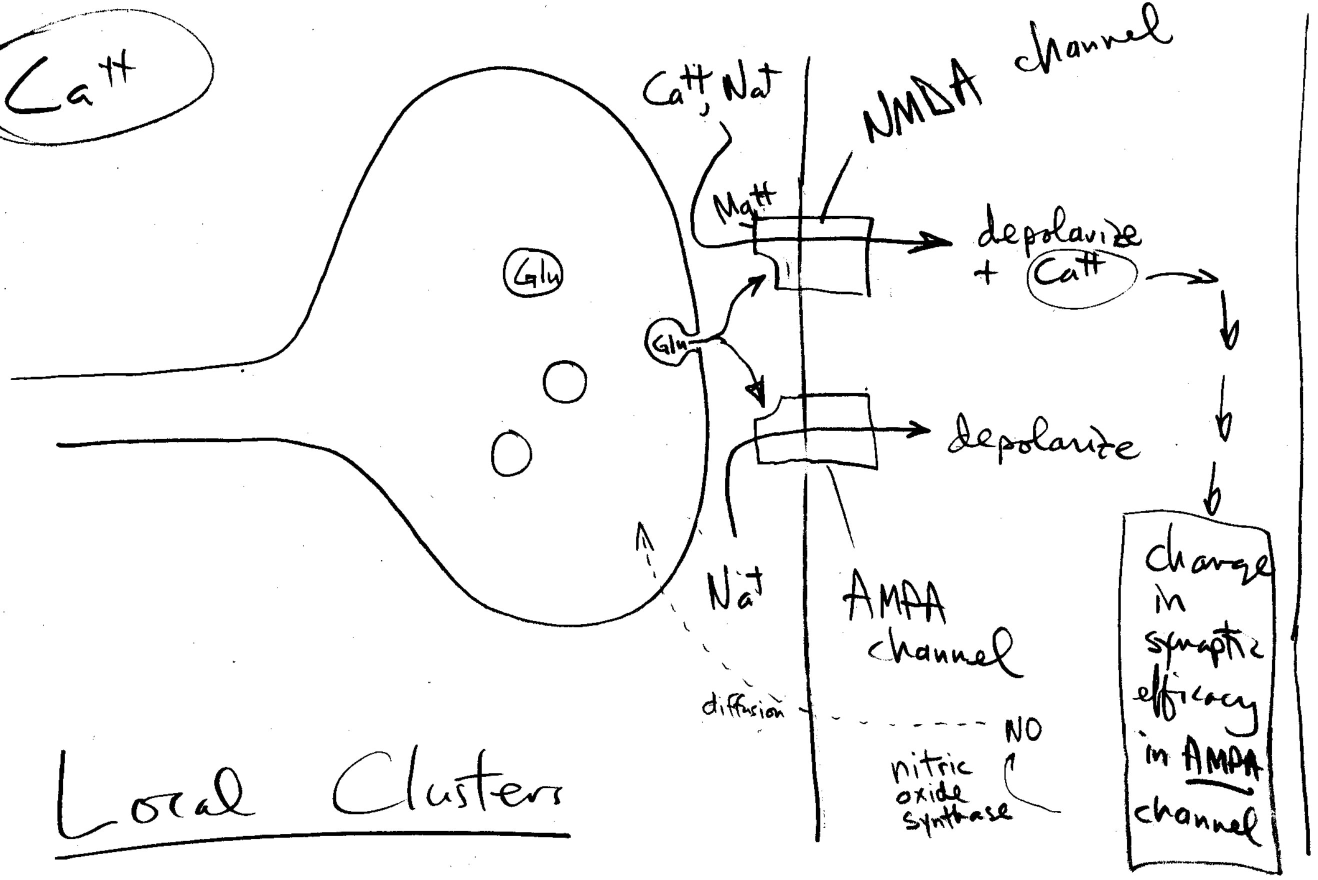
7

GPCRs – G-protein coupled receptors
- mAChR
- adrenergic
- dopamine
  - D1, D5
  - D2, D3, D4
- 5HT-1,3,4
- *glutamate
- *GABA$_B$
- neuropeptide

NMDA channels

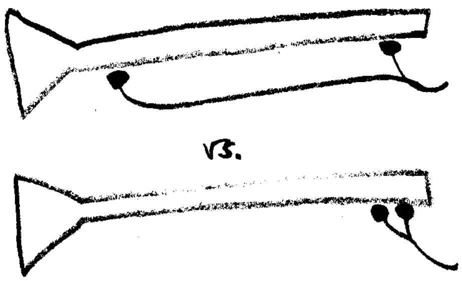
Detecting pre-post correlation

Ca^{++}



Local Clusters

Ca^{++}, Na^{+} ⇒ voltage change in cortex but not hippocampus ⇒



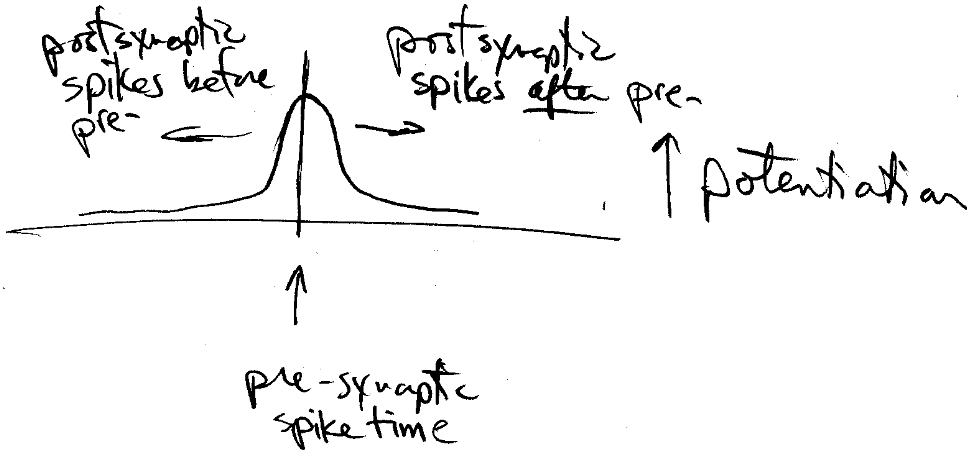
↓

synaptic strength change

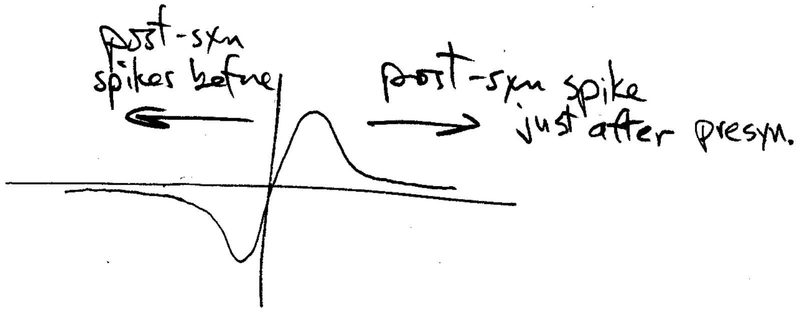
- N.B. one (e.g. this one!) synapse PSP not enough depol. for NMDA
- detect corr. across synapses

Spike-timing Dependent Plasticity

Old Idea



STDP



- idea: that synapses detect causality: if input caused cell to spike ↑

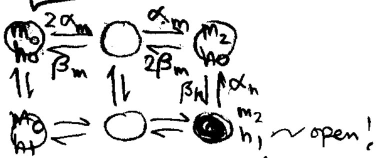
and

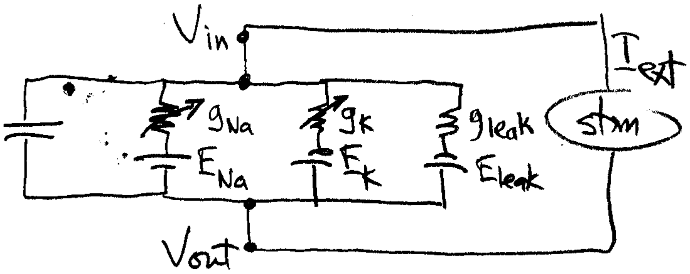
- if cell spiked but input just missed helping out, ↓

and

- diff by +/- 100 msec → no effect

H-H





$$C_m \frac{dV_{in}}{dt} + \underbrace{I_{ions}}_{\text{channels}} = I_{ext}$$

Ohm's law: $I = gV$

$$I_{ions} = \overset{\text{max}}{\bar{g}_{ion}} \cdot \overbrace{m^2 \cdot h}^{\text{gating vars } 0-1} \cdot (V_{in} - E_{ion}) + \overset{\text{other ions}}{\ldots}$$

voltage dep of fwd, backwrd time consts for m, h

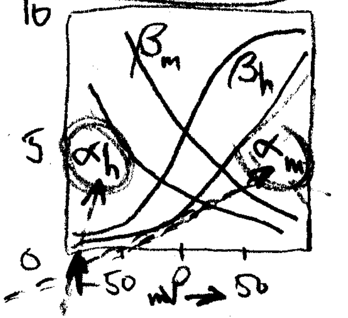
** fit V-clamp data get these curves

first order kinetics

$$\frac{dm^{(t,V)}}{dt} = \overset{\text{fwd rate const}}{\alpha_m(V)}(1-m) - \overset{\text{backward rate const}}{\beta_m(V)}\, m$$

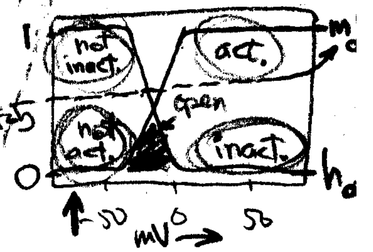
$$\frac{dh}{dt} = \alpha_h(V)(1-h) - \beta_h(V)\, h$$

rate const $\left(\frac{1}{\text{msec}}\right)$



alternate way of writing

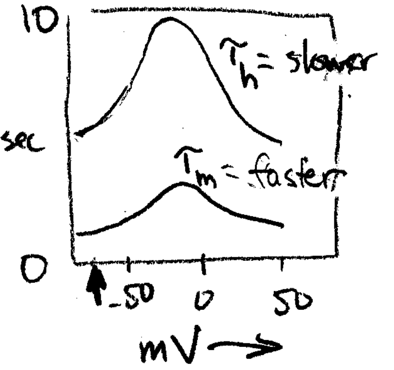
$$\frac{dm^{(t,V)}}{dt} = \frac{\overset{\text{steady state at partic. V}}{m_\infty(V)} - m}{\tau_m(V)} \qquad m_\infty(V) = \frac{\overset{\text{fit to data}}{\alpha_m(V)}}{\alpha_m(V) + \beta_m(V)} =$$



state of gating variables for partic V at $t=\infty$

time const

$$\tau_m(V) = \frac{1}{\alpha_m(V) + \beta_m(V)} \quad \text{msec}$$



Solve simultaneously

numerically integrate

$$\frac{dh^{(t,V)}}{dt} = \frac{\overset{\text{steady state rate const}}{h_\infty(V)} - h}{\tau_h(V)} \qquad h_\infty(V) = \frac{\alpha_h(V)}{\alpha_h(V) + \beta_h(V)}$$

time const

$$\tau_h(V) = \frac{1}{\alpha_h(V) + \beta_h(V)}$$

etc other ions

add

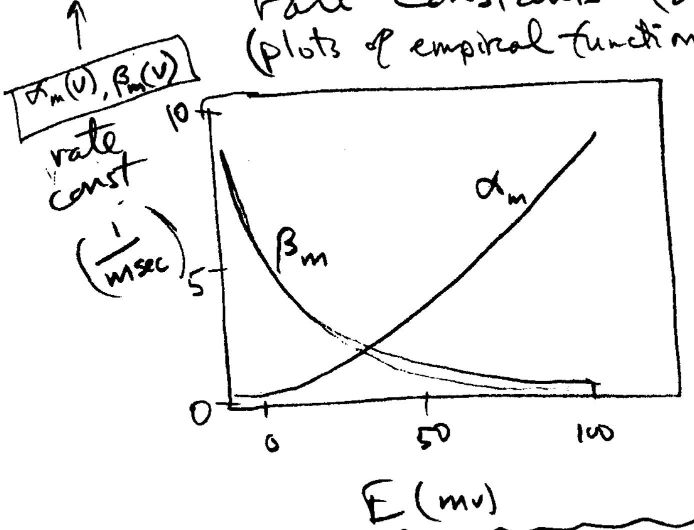
move eq's to left space for graphs

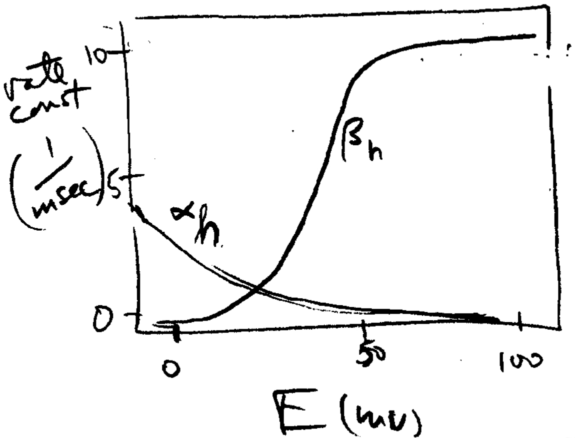
redraft one word time to separa

data fit mon

dy at

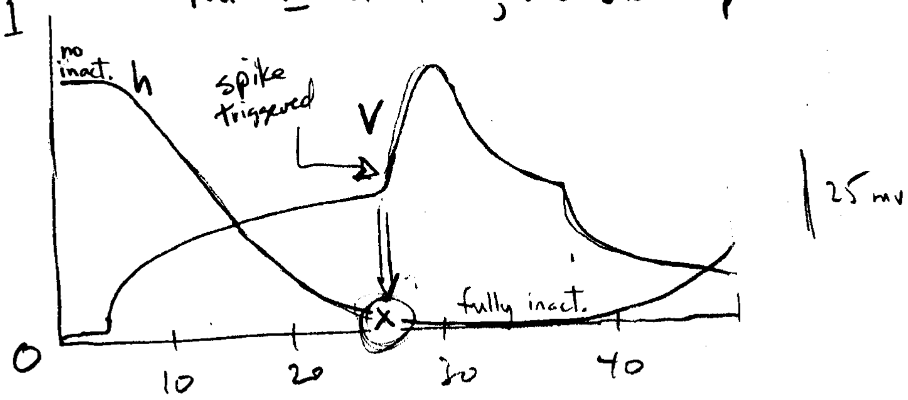
voltage dependence of forward & backward rate constants for Na activation & inactivation (plots of empirical functions)

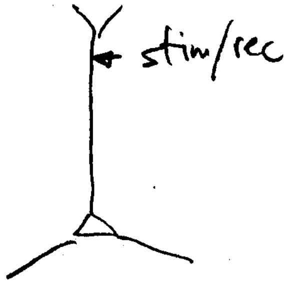


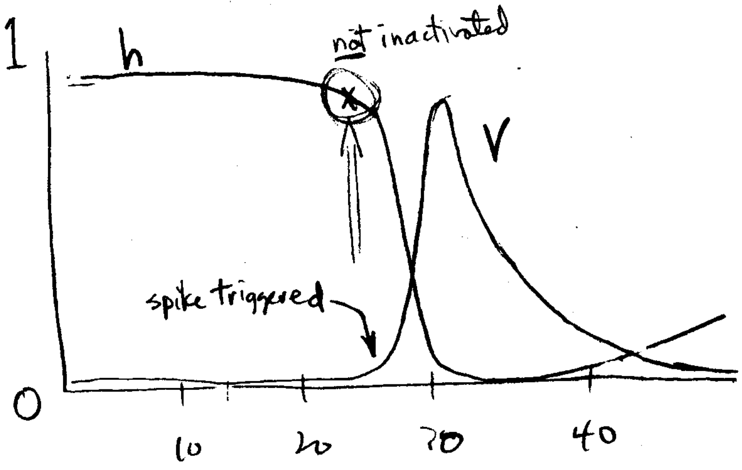


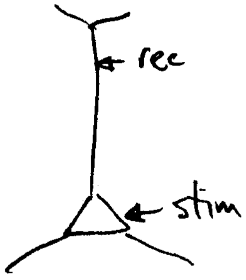
Why are spikes propagated backwards but not forwards in dendrites?

Na^+ inactivation, membrane pot.







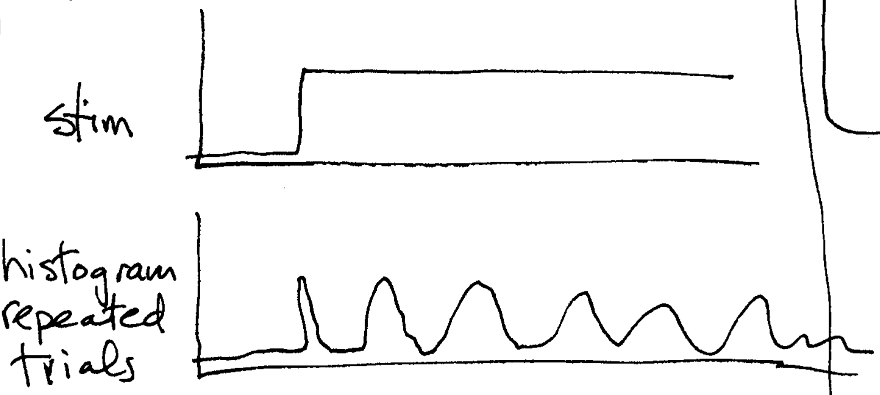


Noise, Stochastic Processes, Firing Patterns

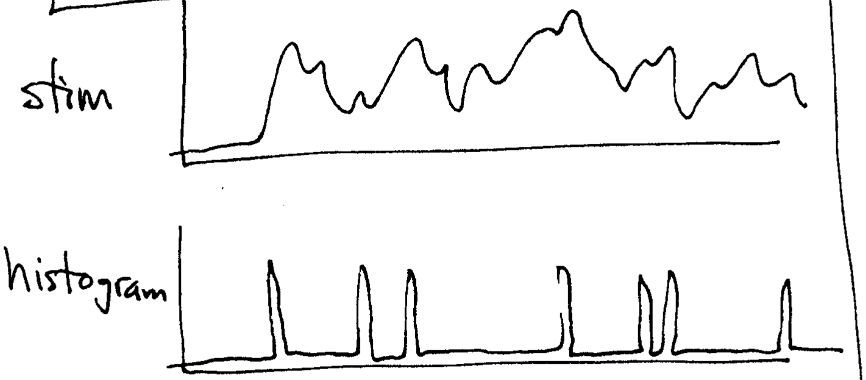
- cortical slices
- silent vs. background

- Hodgkin-Huxley neuron
- 10,000 Poisson distributed inputs
- very regular firing

+ step vs.



noise input



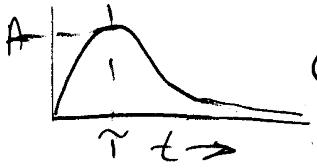
↳ spike generation process is deterministic (!)

- real neurons have highly variable firing patterns in the cortex

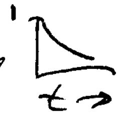
- to simulate cortical variability in firing of stimulated neuron in slice, must inject bursty non-Poisson distributed spikes - filtered - through - PSP

Integrate & Fire

1) P.S.P. conductance waveform



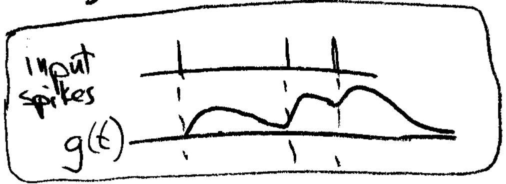
$$G(t) = \overset{\text{"weight"}}{A}\;\underset{\text{start at zero}}{t}\;\boxed{e^{-t/\underset{\text{time const for this P.S.P.}}{\tau}}}$$



2) spike function (vs. H.H)

$$S(t) = \begin{cases} 1 & \text{if } V(t) > \text{thresh } \underline{\text{and}} \text{ not refractory} \\ 0 & \end{cases}$$

3) Linearly add up conductance waveforms at one connection



$$g(t) = \int_0^{t-\text{delay}} G(\lambda)\, S(t-\lambda-\text{delay})\, d\lambda$$

4) add up for all conductances like this $\hat{g}(t) = \sum^{\text{fibers}} g(t)$

5)

$$\underset{\text{next time step}}{\Delta V} = \underset{\text{const}}{\frac{1}{C_m}} \sum^{\text{channels}} \Big(\underset{\text{current membrane pot}}{V(t)} \overset{\text{(reversed in Wilson \& Bower)}}{-} \underset{\text{reversal pot}}{E_{\text{channel}}} \Big) \underset{\text{from above}}{\hat{g}_{\text{channel}}(t)}$$

<u>from:</u> 1) Ohm's law $I = Vg$

2) $\Delta V = \frac{\sum I}{C}$

$\longrightarrow \Delta V = \frac{1}{c} V g$

Cable Theory → Compartmental Models

$$V = \overset{\text{space const}}{\lambda^2}\left(\frac{\partial^2 V}{\partial x^2}\right) - \overset{\text{time const.}}{\tau}\left(\frac{\partial V}{\partial t}\right)$$

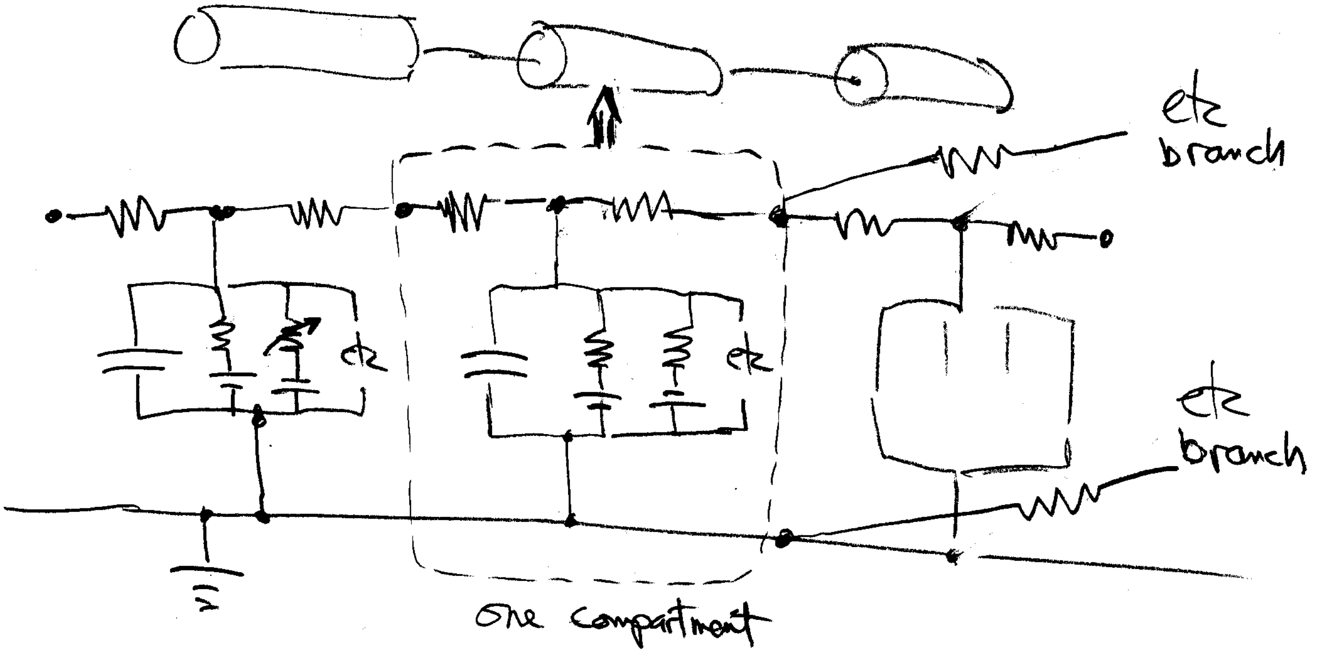
$$\lambda = \sqrt{\frac{r_m}{r_i}} = \sqrt{\frac{R_m/2\pi \overset{\text{radius}}{r}}{R_i/\pi r^2}} \qquad r_m c_m = (R_m/2\pi r)(C_m \cdot 2\pi \overset{\text{radius}}{r})$$

Cond vel:

$$\frac{dx}{dt} \propto \frac{\lambda}{\tau} \propto \sqrt{\frac{\overset{\text{radius}}{r}}{R_i R_m C_m^2}}$$

Compartmental models —

- replace cont. partial D.E. eq w/ ordinary
- each compartment isopotential
- non-uniformities between compartments
- assume extracellular ground



circuits-1

Definitions

Intro to Circuits

I – current ("amount of flow")

R – resistance

g – conductance (= $1/R$ = "diameter of pipe")

V – voltage, potential ("pressure")

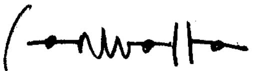
C – capacitance ("spring loaded storage jar", "balloon")

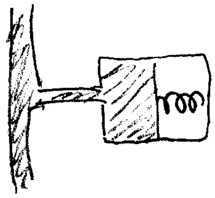
Q – charge ("what's stored")

Rules

V across parallel equal

I across series equal



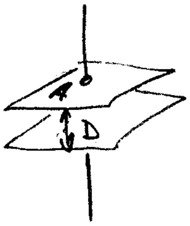


it takes <u>work</u> (and finite time!) to charge capacitors

$V = IR$ (Ohm's law)

$Q = CV$

$C \propto \frac{\text{Area}}{\text{Distance}}$



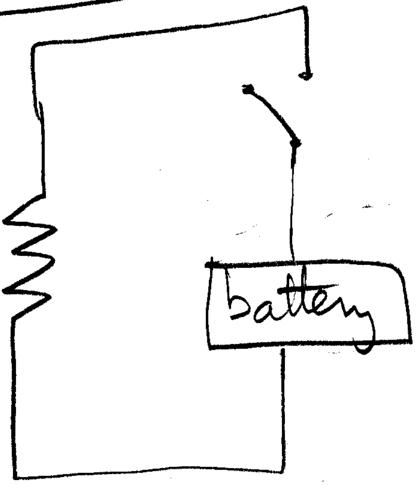
Do this for each
- a) hold one value constant
- b) vary second
- c) see how this affects third

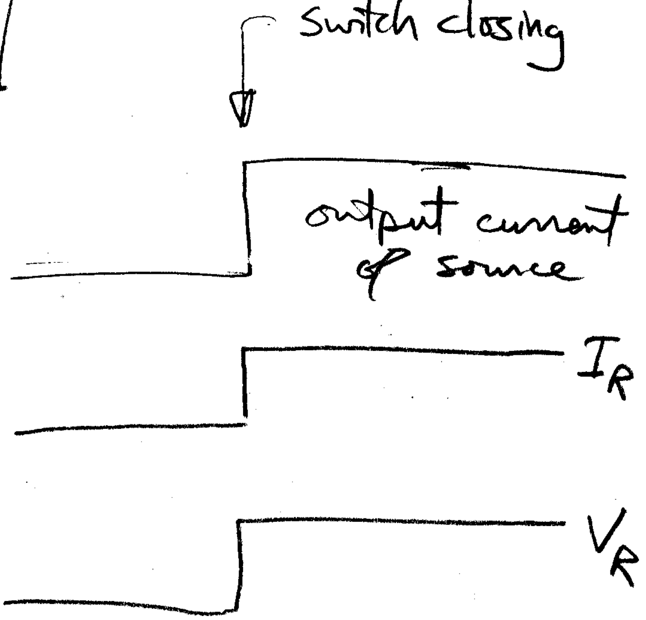
$$\frac{dQ}{dt} = C_m \frac{dV}{dt}$$

circuits-2

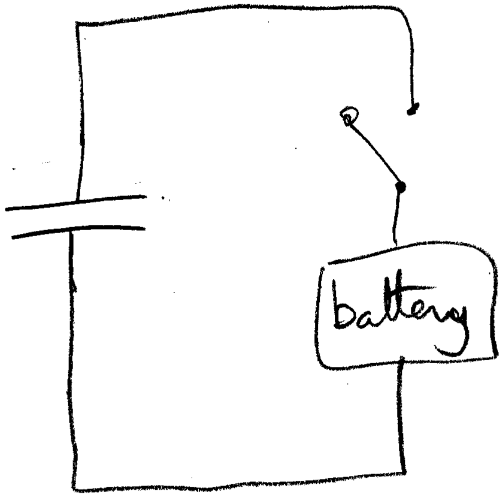
Simplest Circuits

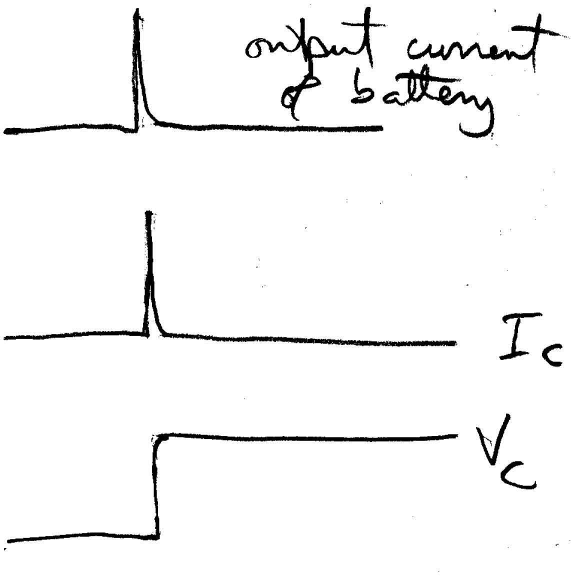
Resistor only





Capacitor only





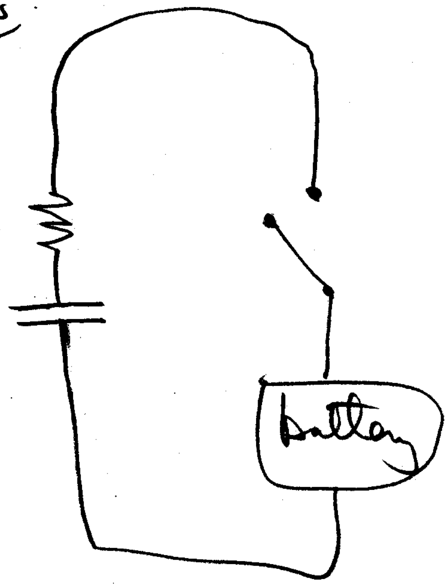
N.B.

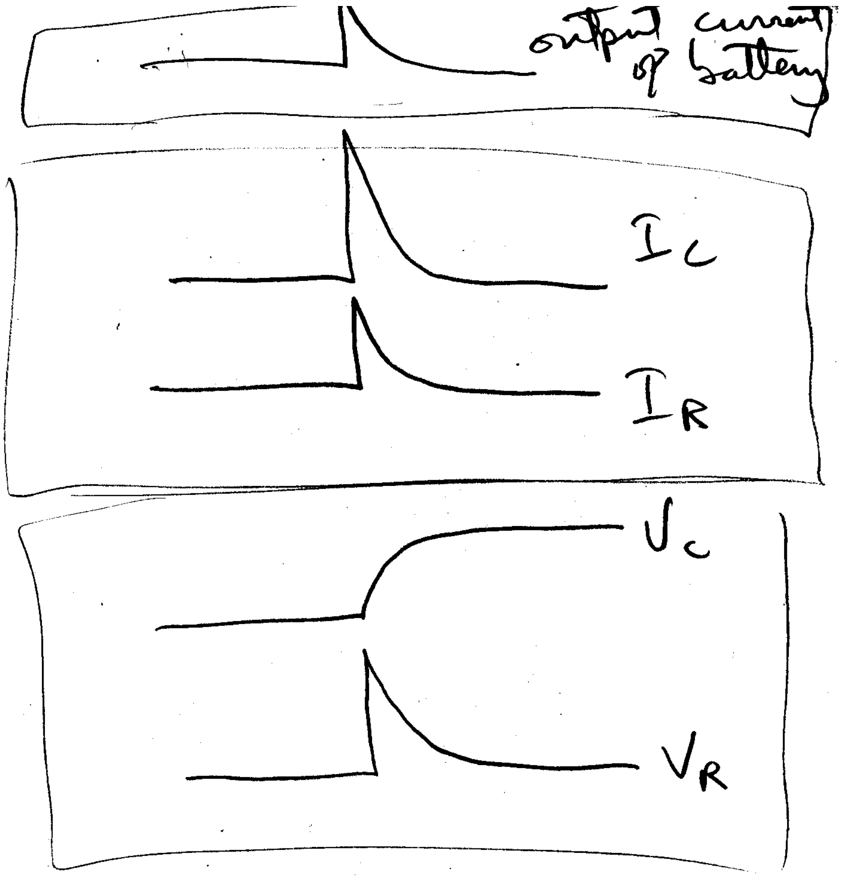
- no resistance so
  - current very high
  - capacitor charges almost instantly
- a partly-charged capacitor still non-infinite effective resistance

circuits-3

Simple Circuit

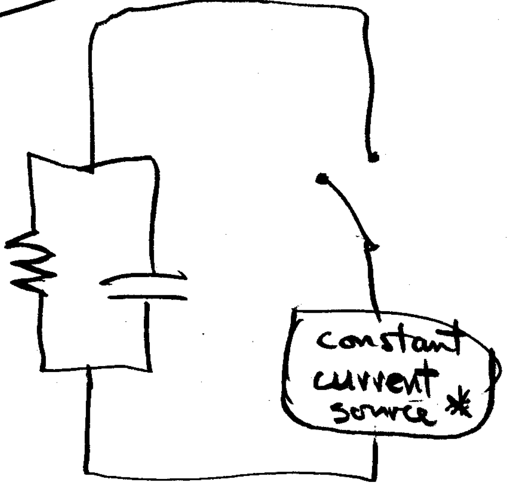
Series

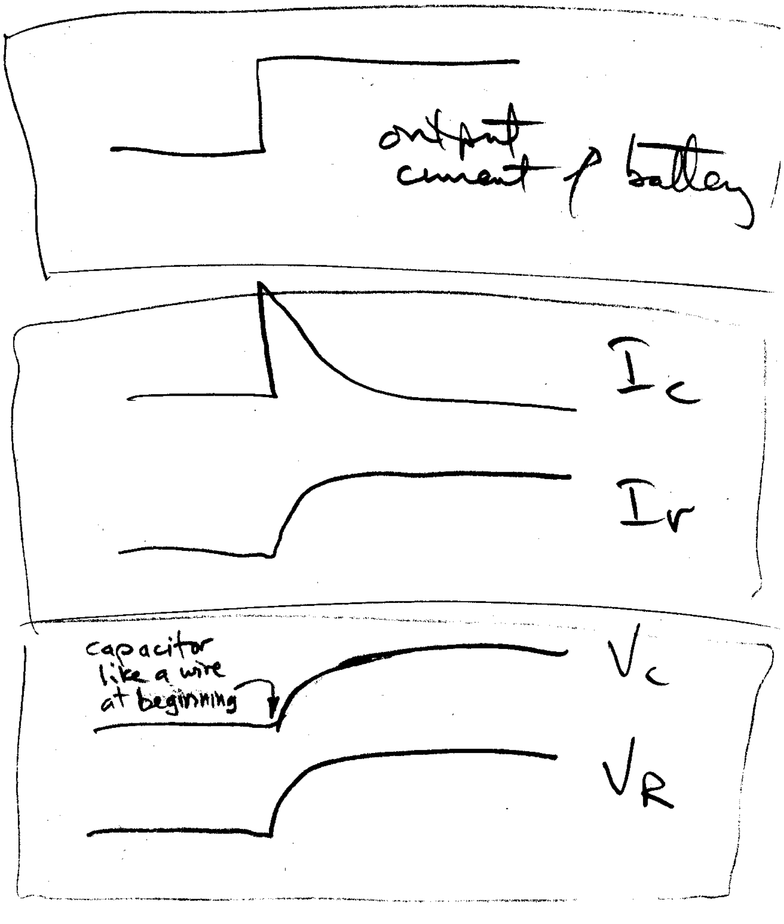




- C charges slower because max voltage limited by R. (voltage controls charging rate)

parallel **





- ditto (w/ finite current source)

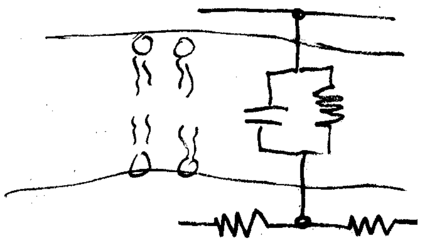
* if battery really big, would look instantaneous

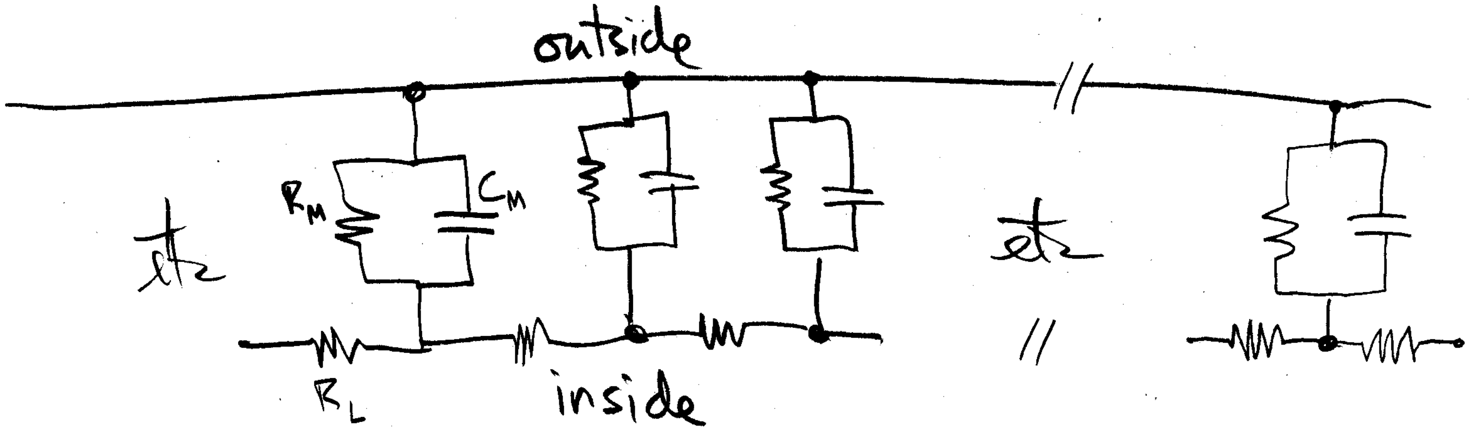
Circuits-4

#2

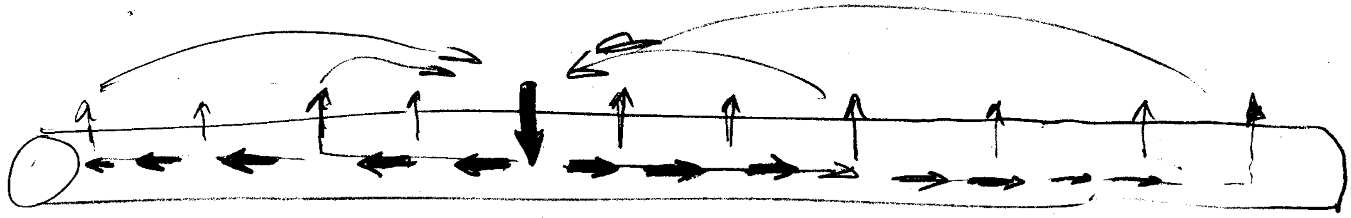
Electrotonic/Dendritic Current Flow

- where do delays come from?
- cable theory (differential eq.)
- assume resting potential $\underline{0}$
- cable theory → compartmental model



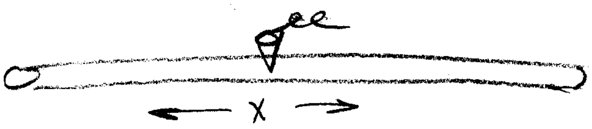


- why it's called cable theory $R_M \gg R_L$

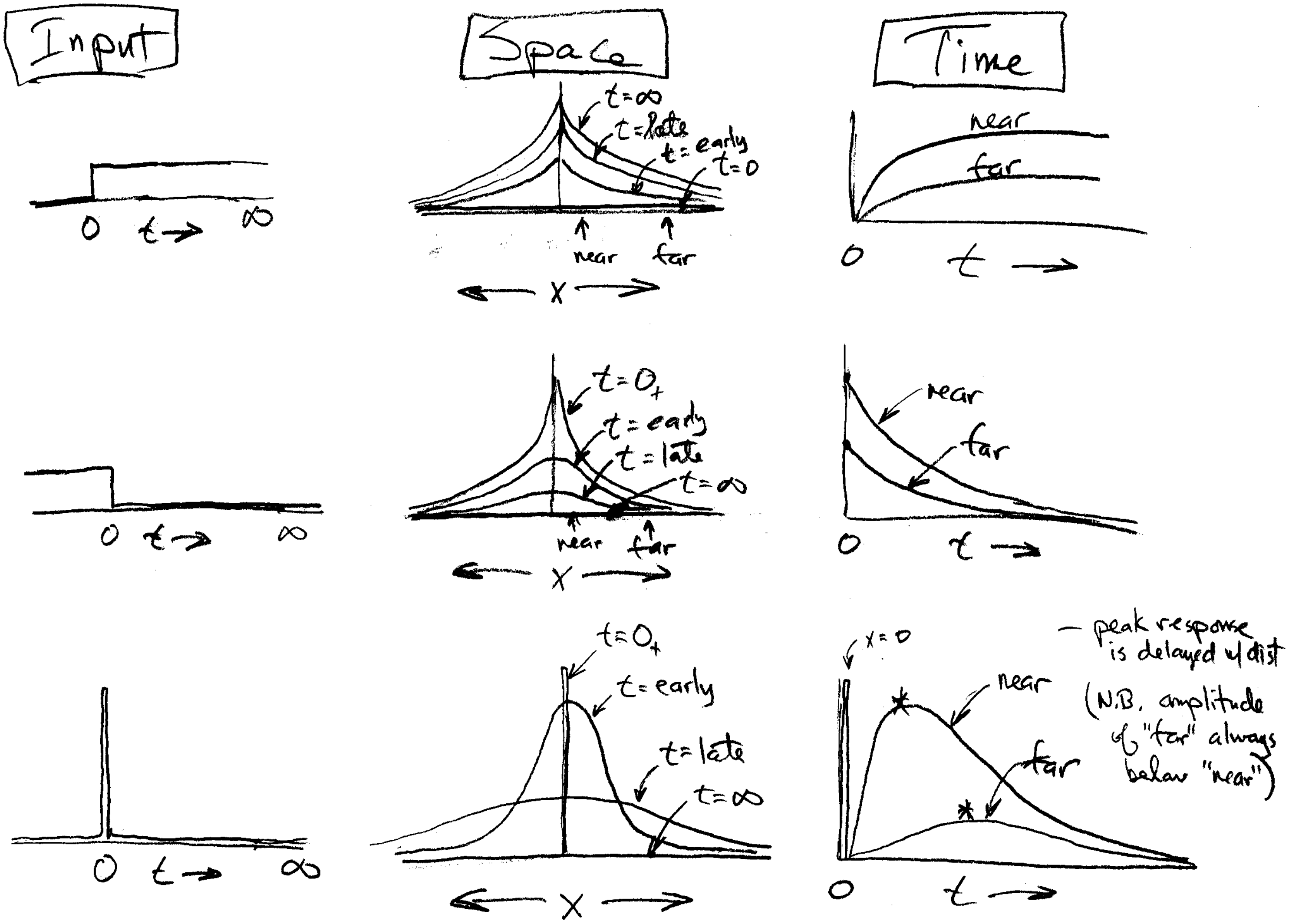


circuits-5

ON-STEP, OFF-STEP, SHORT PULSE IN SPACE & TIME



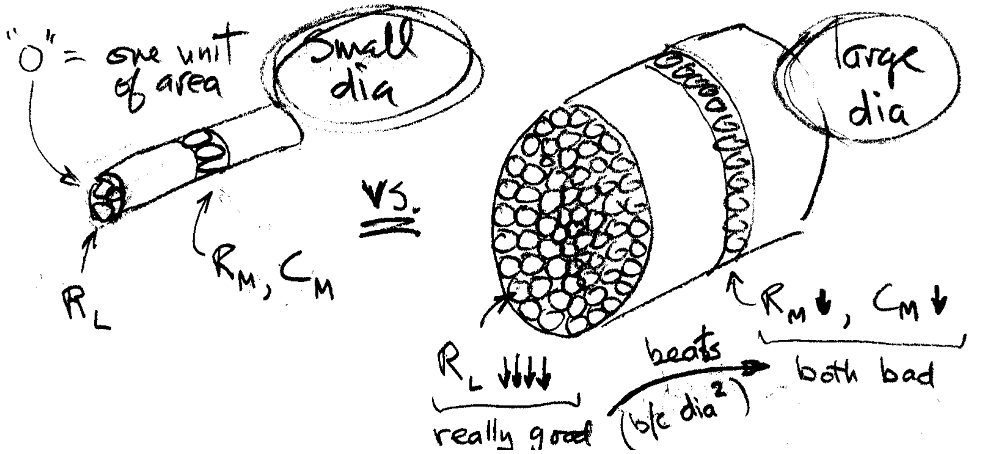
plot $\left<\begin{matrix}V\\I\end{matrix}\right.$ (conductance fixed)



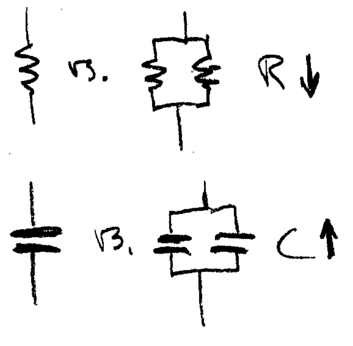
– peak response is delayed w/ dist

(N.B. amplitude of "far" always below "near")

WHY DOES INCREASED DIA INCREASE COND. VELOC.?



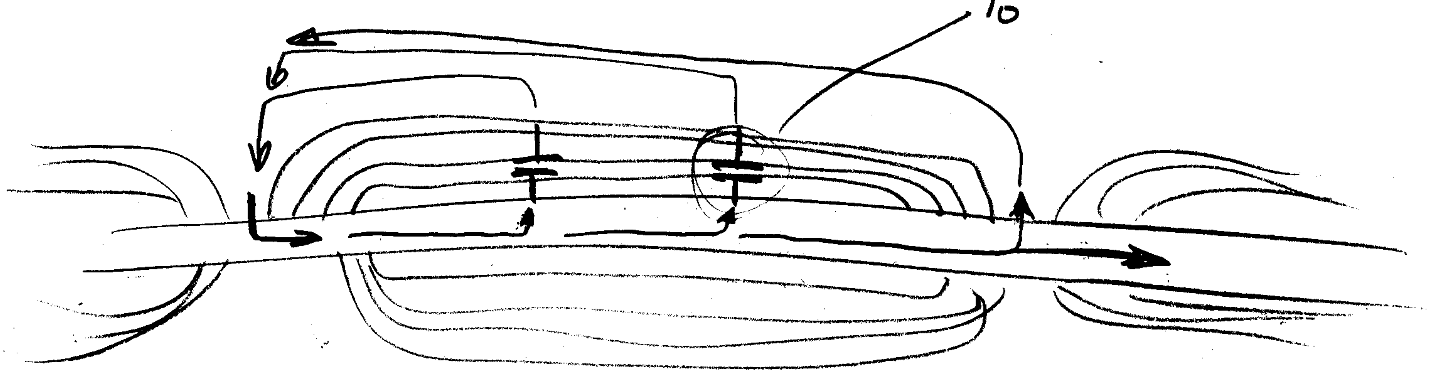
effects of expand area



Circuits-6

Myelination

- myelin has <u>low</u> C
- conduction velocity ↑
  electrotonic pulse <u>faster</u> as result



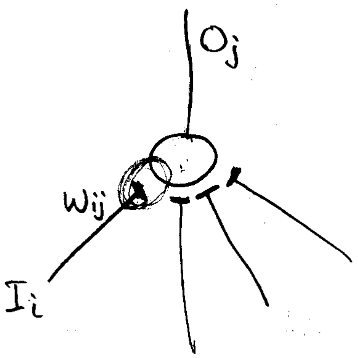
#6

6

Linsker (1) Basic Idea

1) linear summation

$$O_j = \sum^{i} I_i w_{ij}$$



2) simple Hebb

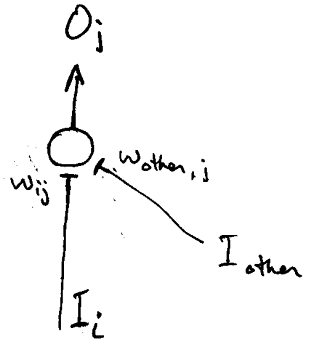
$$\Delta w_{ij} = \text{rate} * Q_{ij}$$
$$Q_{ij} = I_i * O_j$$
$$\Delta w_{ij} = \text{rate} * I_i * O_j$$

3) put together into one equation

$$\Delta w_{ij} = I_i \left(\sum^{\cancel{i} \to \text{other}} I_{\cancel{i} \to \text{other}} w_{\cancel{i} \to \text{other}, j} \right)$$

$$Q_{i,\text{other}} = I_i I_{\text{other}}$$

("other" ranges over all the other connections to the unit but <u>also</u> includes current connection i)



$$\Downarrow$$

$$\Delta w_{ij} = \sum^{\text{other}} Q_{i,\text{other}} w_{\text{other},j}$$

$$\Downarrow$$

$$\Delta w_{ij} = \overset{\text{weight decay}}{k_1} + \frac{\sum^{\text{other}} \left(Q_{i,\text{other}} + \overset{\text{set no-change point}}{k_2} \right) w_{\text{other},j}}{\#\text{inputs}}$$

[oneweight = Q <u>vector</u> · weight <u>vector</u>
weight <u>vector</u> = Q <u>matrix</u> · weight <u>vector</u>

<u>Q examples</u>

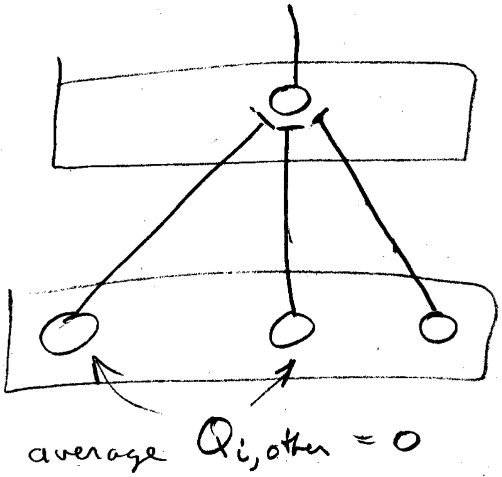
1 x 2
-2 x -5
3 x 1
-4 x 2

avg pos

→ thus, Hebb synapses detect and amplify 2-point correlations in input lines

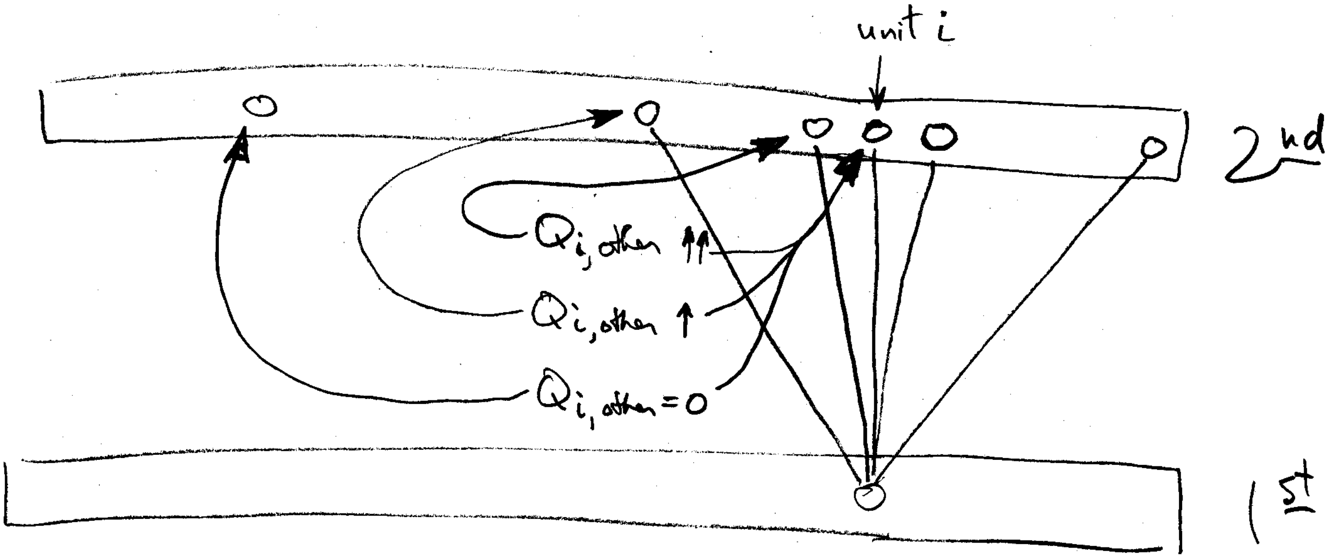
Linsker (2) What happens in layer 2

2nd layer input go all-neg or all-positive



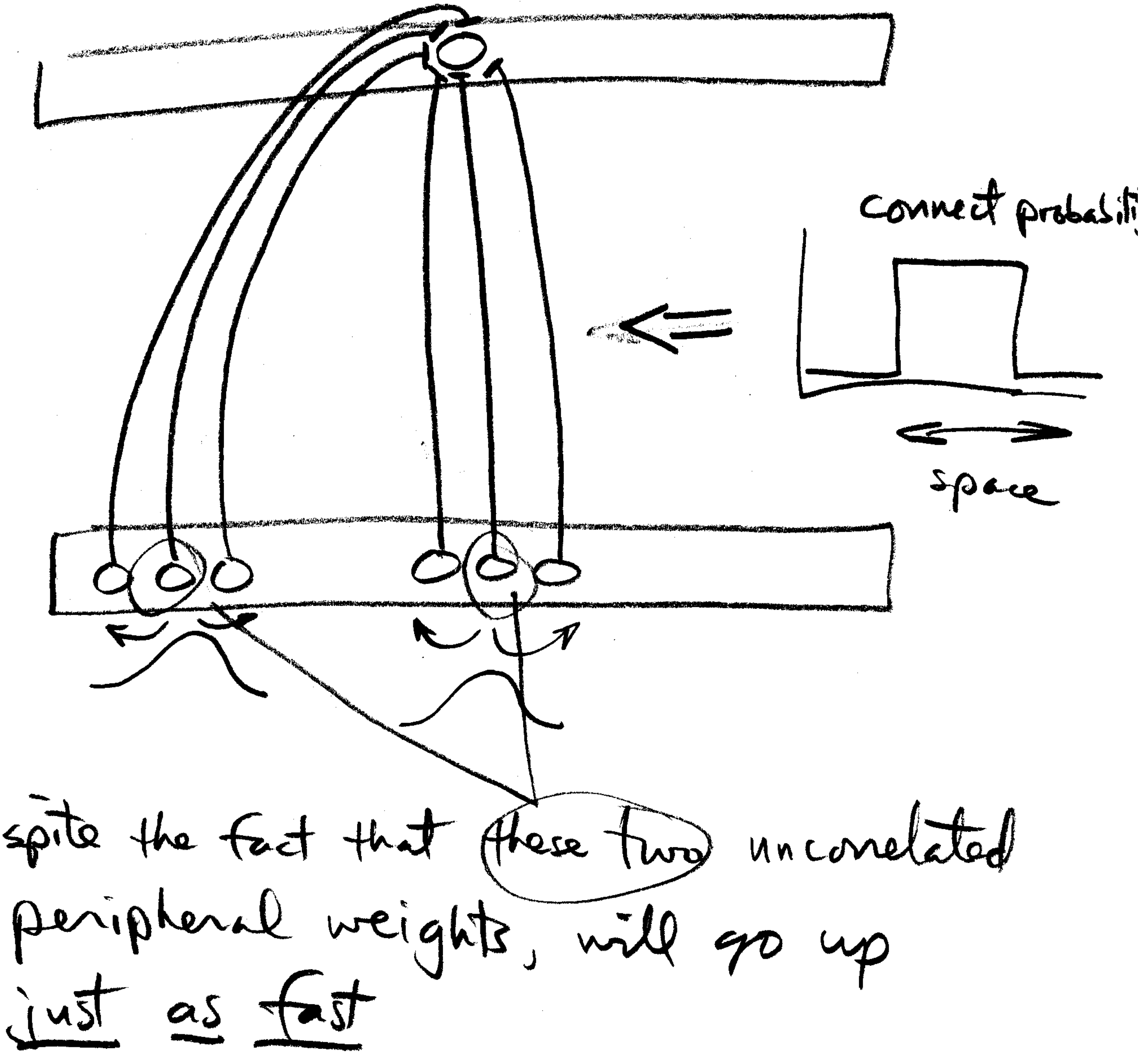
- as soon as average weight to a unit is slightly pos (or neg), Δw for current weight goes pos (or neg)
- the next cycle, tho $Q_{i,\text{other}}$ will still average zero, $\Delta w = \sum Q \cdot w_{\text{other}}$ will be more pos (or neg), since the <u>other weights</u> are more pos (or neg)

What endpoint $Q_{i,\text{other}}$ looks like for layer 2



Why the RF center weights go up

counter example



- despite the fact that these two uncorrelated peripheral weights, will go up *just* *as* *fast*

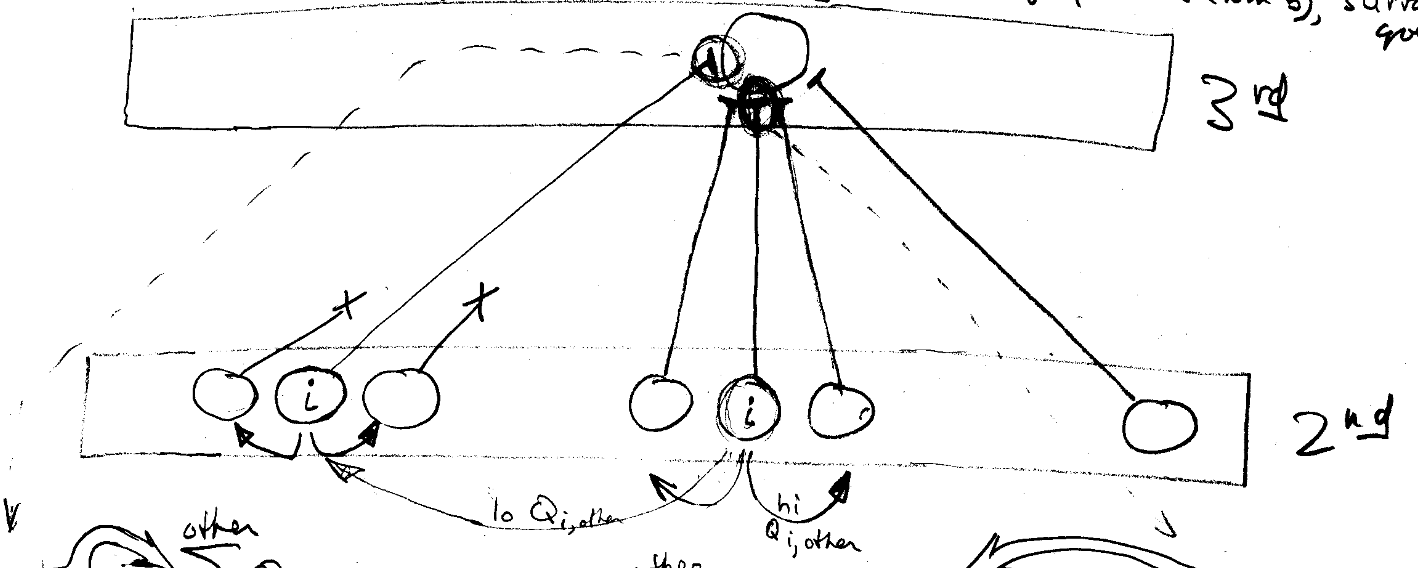
Linsker(3) What happens in layer 3

1) 3rd layer inputs go center-surround

a) k_1 causes increase in weights

b) because [nearby inputs from layer 2 correlated *and*, there are more units in center] → center weights go higher

c) k_2 drags down everybody; center stays positive from b), surround goes neg.



update for weight coming from receptive field periphery

$\sum^{other} Q_{i,other} * w_{other,j}$

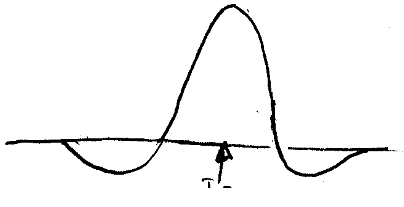
lower because few nearby correlated units hook up and add to sum (far away units that are connected are uncorrelated and add little)

$\sum^{other} Q_{i,other} * w_{other,j}$

high because many nearby correlated units hook up and add to sum

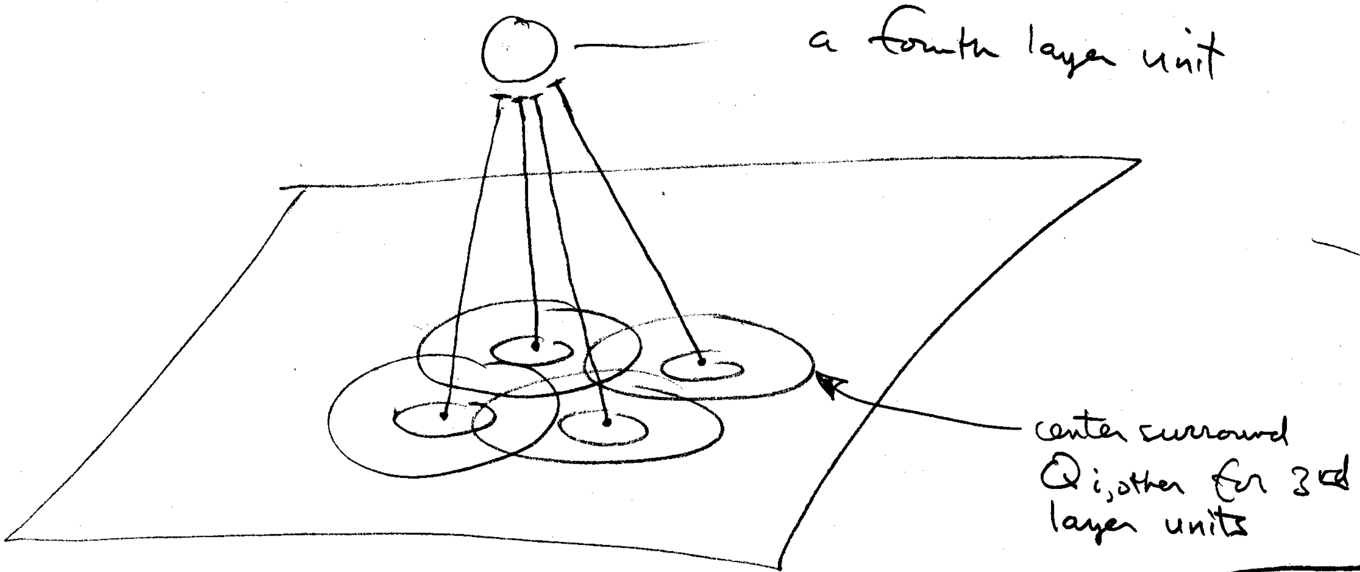
update for weight coming from receptive field center

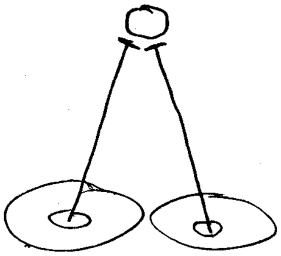
2) What the $Q_{i,other}$ looks like for layer 3



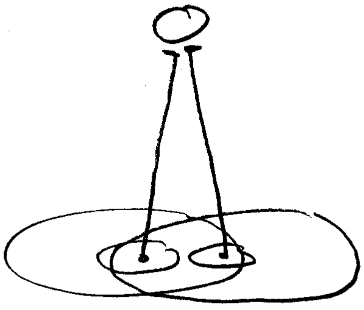
Linsker(4) What happens in layer 4

(symmetry-breaking)

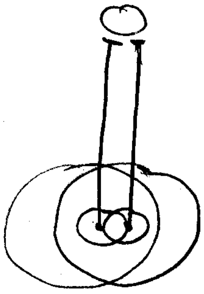




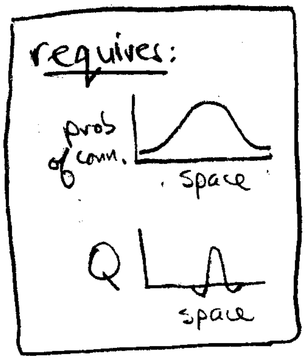
$\sum Q_{i,\text{other}}$ will be zero for these



$\sum Q_{i,\text{other}}$ will be negative for these



$\sum Q_{i,\text{other}}$ will be positive for these

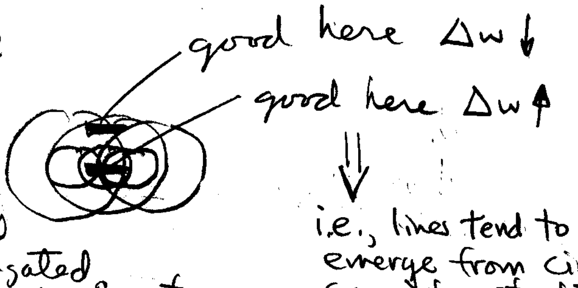


Correlation of nearby centers Δw ↑

anti-correlation of center and midrange surround Δw ↓

far away cells uncorrelated Δw = 0

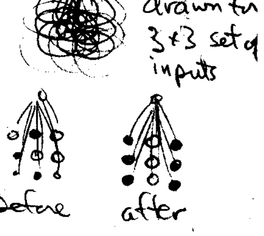
- it is possible to calculate a Hebb energy
- each Δw will always change to lower Hebb energy
- a receptive field with elongated excitatory & inhibitory regions has lowest energy



⇓

i.e., lines tend to emerge from circularly symmetric starting dist

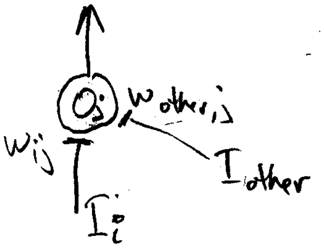
⇒



Why Eigenvectors?

$\Delta\vec{w} = \bar{Q}\vec{w}$ is a (system of) differential equations

Linsker



$$\Delta w_{ij} = K_1 + \frac{1}{\#inputs} \sum^{other} (Q_{i,other} + K_2)\, W_{other,j}$$

(ignore j b/c only one output unit) ↓

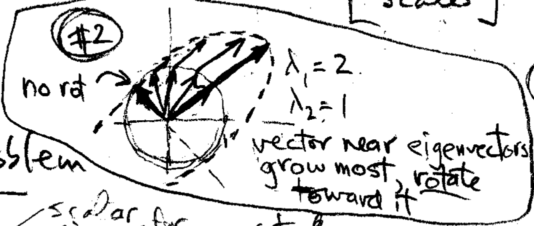
change in one weight Δw_i = **q** vector · **w** vector — dot prod ↑ if $\vec{q}, \vec{w}$ similar

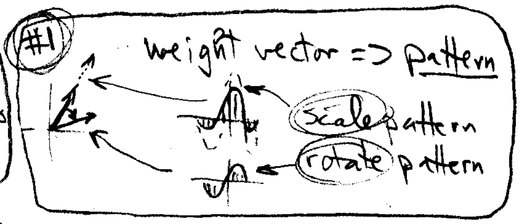
||

change in weight vector (different i's) $\Delta\vec{W}$ vector = $\bar{Q}$ matrix × $\vec{W}$ vector — same for all i's

Linear Transformations

$\mathbf{A}\mathbf{x} = \mathbf{b}$ matrix **A** [rotates scales] vector **x** = output vector **b**





Eigenvalue Problem

$\mathbf{A}\mathbf{x} = \lambda\mathbf{x}$

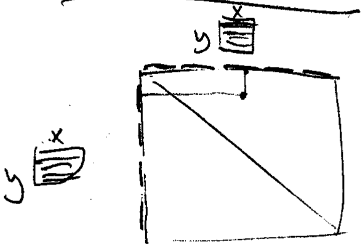
scalar eigenvector

solve for multiple **x**'s (eigenvectors)

find set of vectors $\mathbf{x}_n$ that when operated on by matrix **A** just grow (no rotation)

- λ_n is how much each grows
- vectors not in direction of $\mathbf{x}_n$ rotate most toward nearest $\mathbf{x}_n$

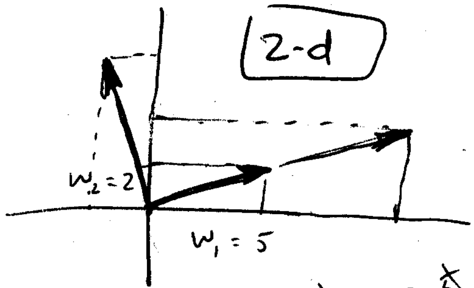
Covariance Matrix

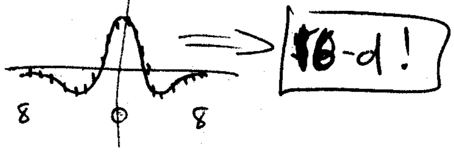


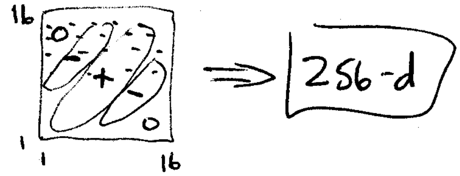
a vector in direction of eigenvector will just grow (no rot!)

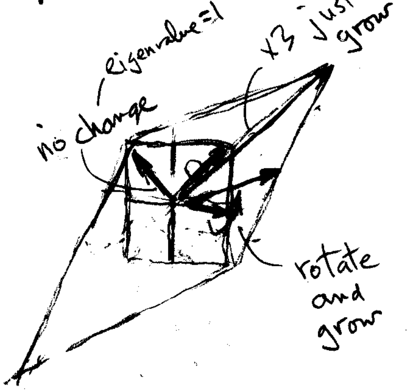
eigen vectors of stim covariance matrix tell which directions weight vectors will grow when operated on by covariance matrix during Hebb update

- vector is pattern of weights

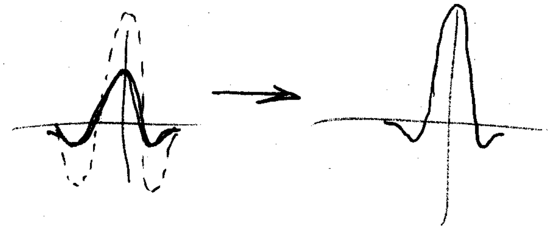




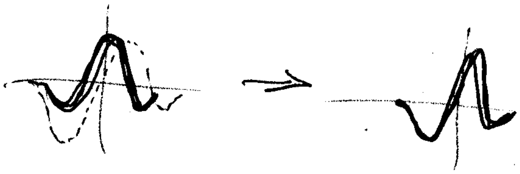


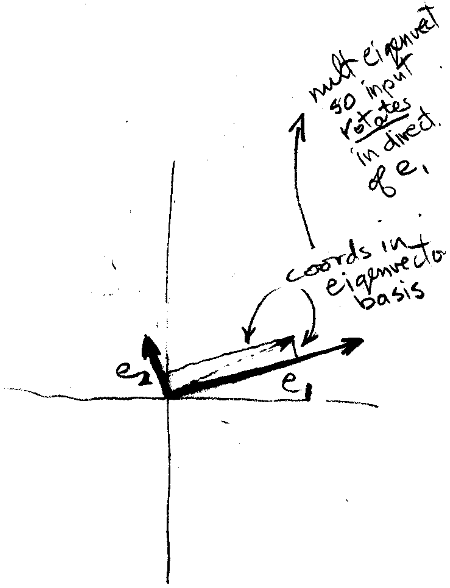


- scaling a pattern (multiplying by factor) doesn't change its "shape"



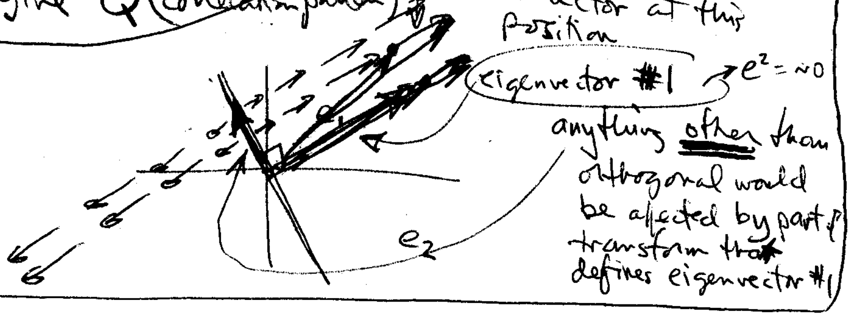
- rotating a pattern does change shape





- eigenvalue/eigenvector solution tells which directions (i.e. patterns) most likely to arise from give Q (correlation pattern)

eigenvectors are orthogonal!

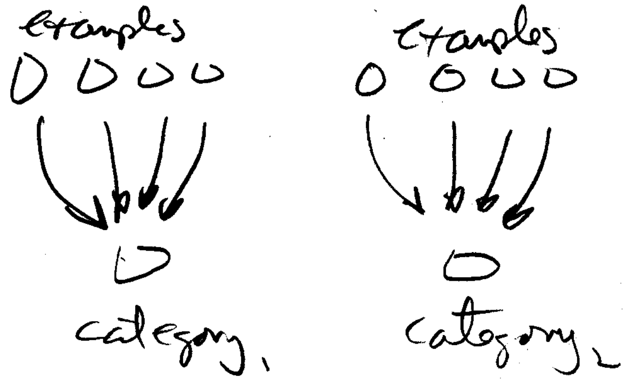


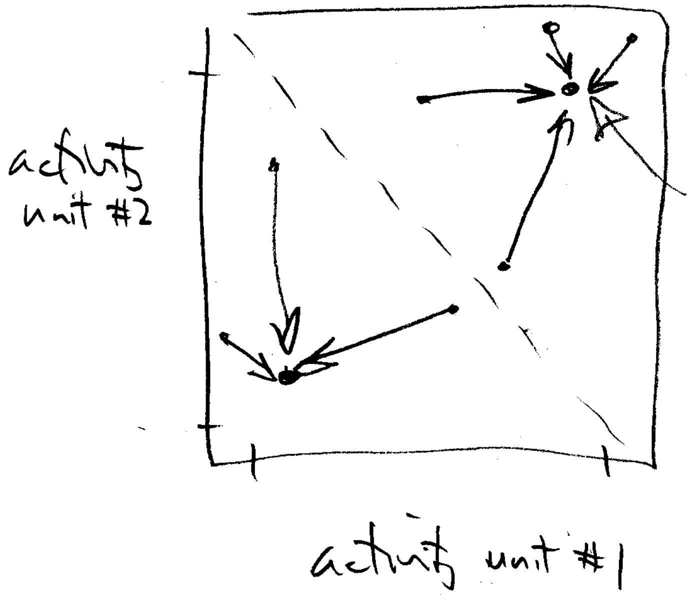
Recurrent Intro (1)

1) feedforward, learning (= weight change)

2) now: recurrent, dynamics w/o weight change

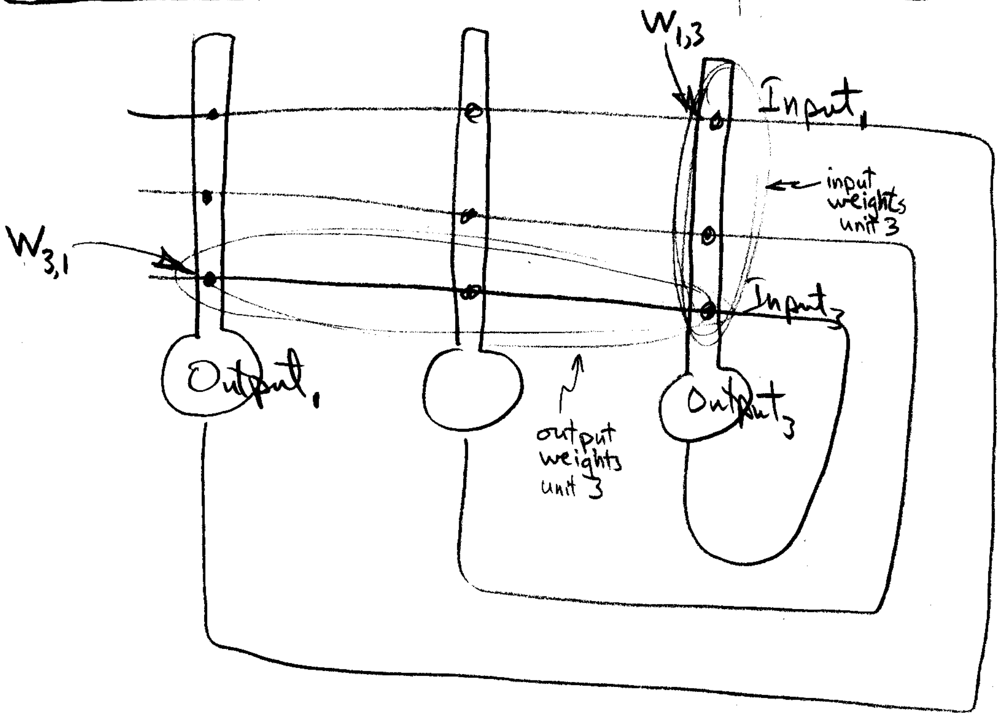
Attractor Networks





attractor pattern {unit 1 = 0.9, unit 2 = 0.1}

Recurrent – Why ΔE always $\downarrow$ or 0 (2)



Update

$$\underset{\text{(vector)}}{\text{Output}_j} = \left[\begin{array}{lll} 1 & \text{if} & \sum_i \text{Input}_i\, w_{ij} > 0 \\ -1 & \text{if} & \sum_i \text{Input}_i\, w_{ij} \le 0 \end{array}\right.$$

Energy

$$\underset{\text{(one number!)}}{\text{Energy}} = -\sum_i \sum_j \text{Input}_i\, w_{ij}\, \text{Output}_j$$

$(\text{where } \text{Input}_i = \text{Output}_i)$

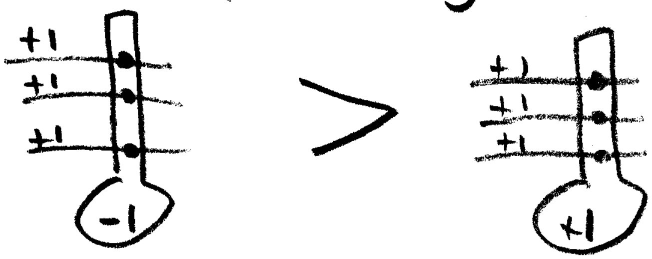
Lyapunov funct

show that update of any unit can only $\left[\begin{array}{c}\text{reduce} \\ \text{or} \\ \text{leave same}\end{array}\right]$ energy

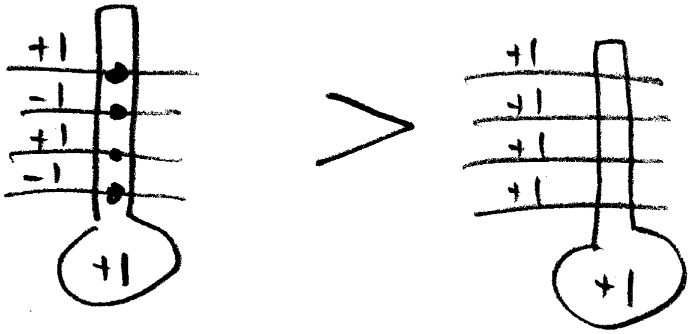
Recurrent - symmetry (3)

- intuitive idea of energy or "frustration"

1)



2)



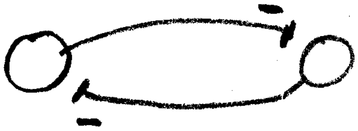
- update of one unit reduces (or keeps same) energy terms from its input connections
  ↳ (definition of update rule)

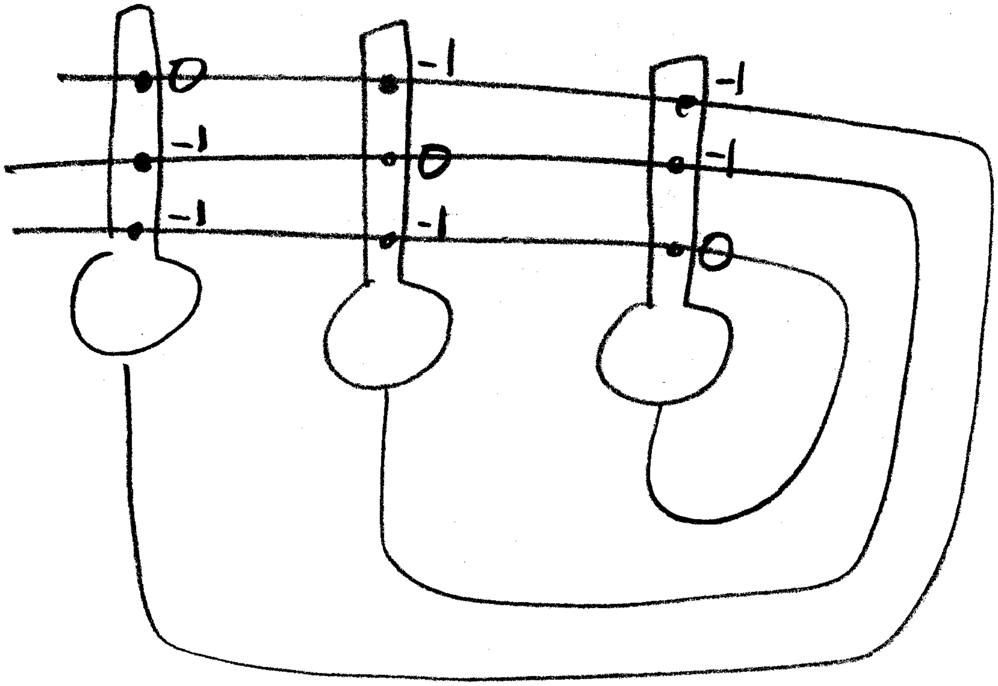
- output connection weights ⟶ unknown effect

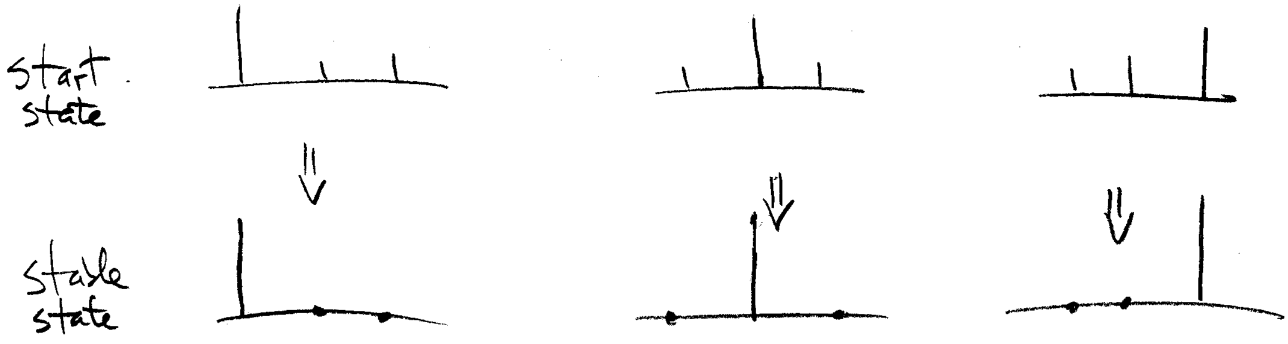
- if <u>symmetry</u>, output connection terms term-for-term same as input connection terms

- ∴ if <u>symmetry</u> ⟶ stable states

Winner-take-All

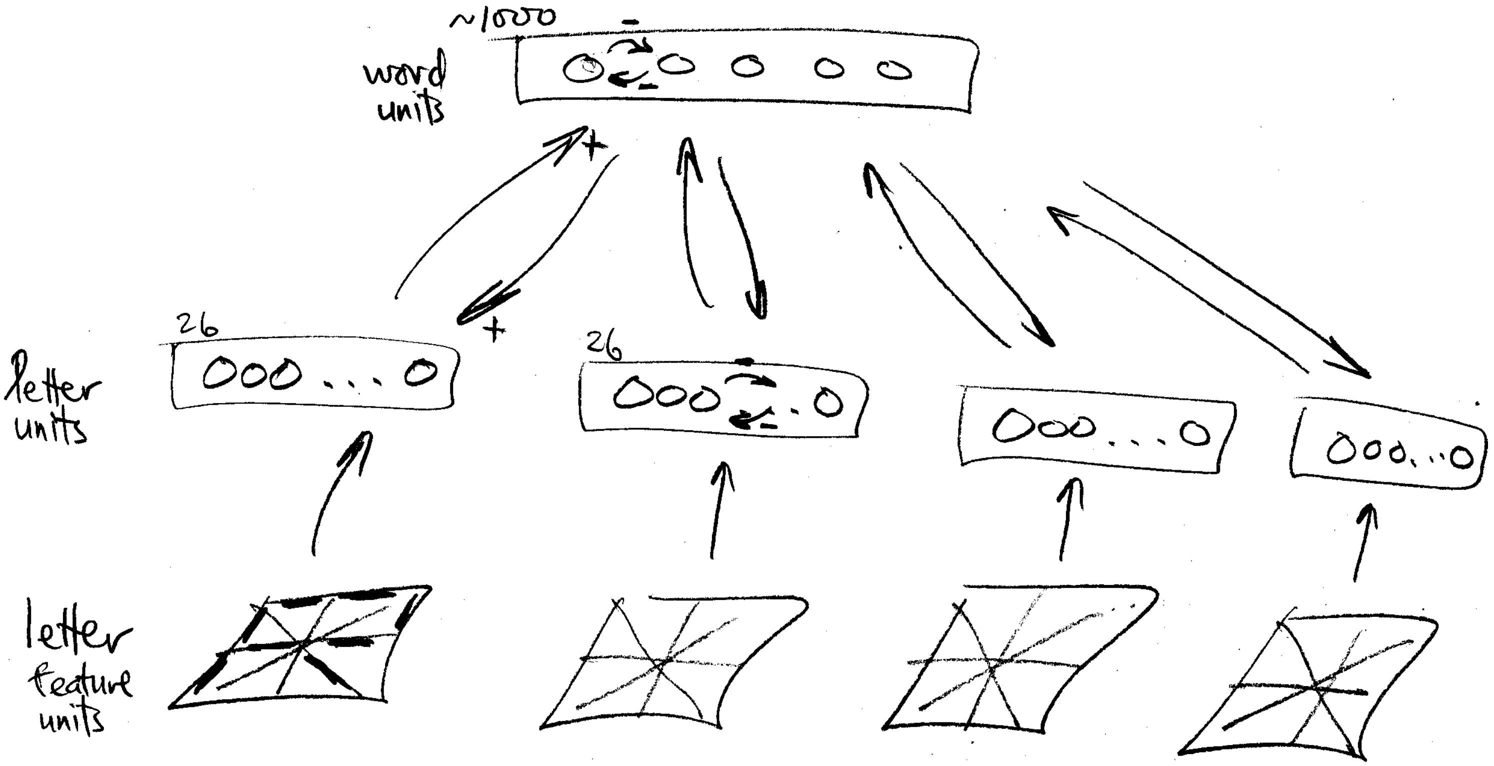


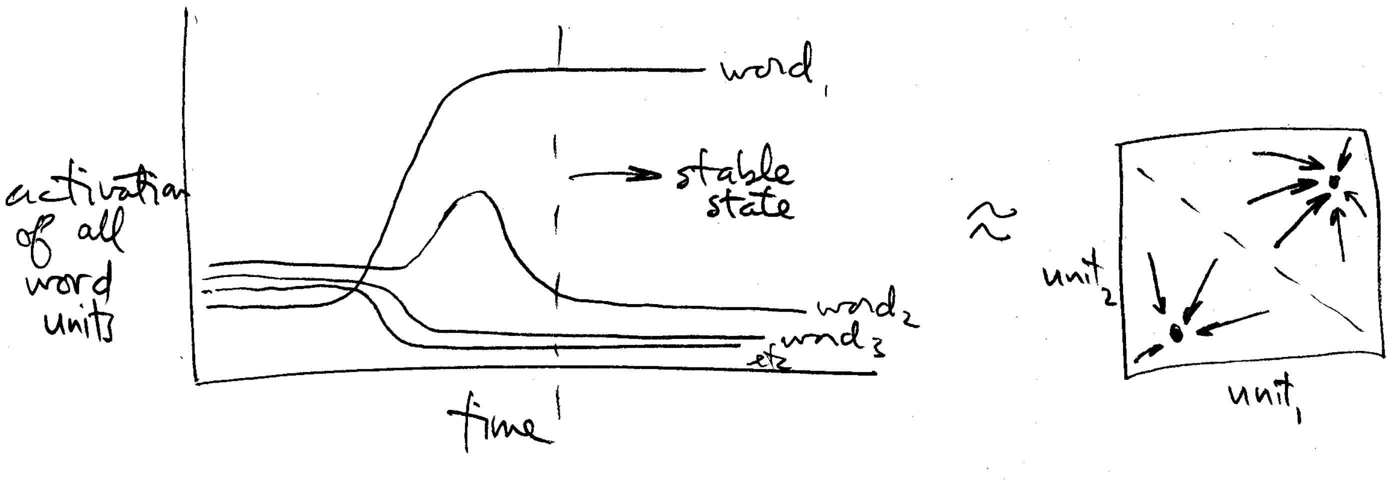




— 3 stable states

Word-Rec model as Hopfield





- word-like non-words speed letter rec.
- deal w/partial occlusion/noise

#5

Back Prop Summary

$$\text{linear\_output}_j = \sum_i \text{input}_i * w_{ij}$$

$$\text{squashed\_output} = f(\text{linear\_output}) \qquad (f(x) \text{ e.g. a sigmoid})$$

change weight to reduce error:

$$\Delta w = \overset{\text{learning}}{\text{rate}} * \delta * \text{input}$$

where:

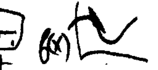
$$\delta_{output} = (\text{target} - \text{actual}) * f'(\text{linear\_output})$$

$$\delta_{hidden} = \left(\sum^{output} \delta_{output} w_{output}\right) * f'(\text{linear\_output})$$

gradient descent → what Δw = des. - act. does!

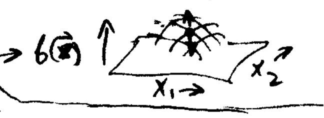
1

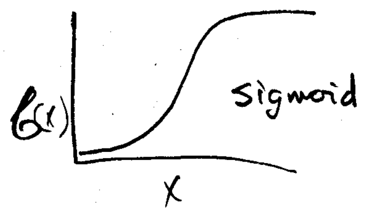
deriv. → get slope, take neg → go downhill

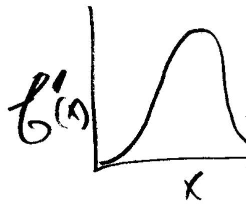


gradient → deriv. of $f(\vec{x})$ (generaliz. to vect arg)
get grad vector, take neg → go downhill

error →

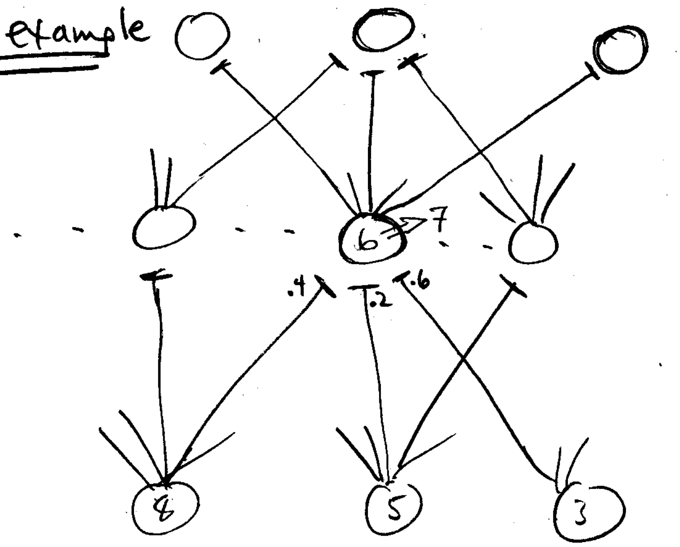


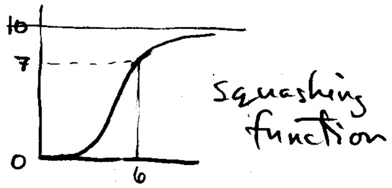




derivative of a sigmoi is a gaussia

Feedforward pass example





$$\text{linear\_output}_j = \sum^{i} \text{input}_i \, w_{ij}$$

$$= (8 \times .4) + (5 \times .2) + (3 \times .6)$$

$$= 6$$

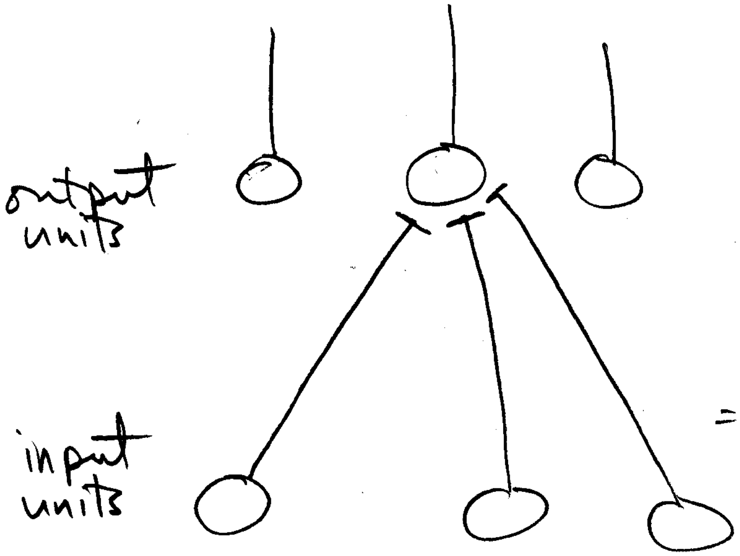
$$\text{squashed\_output}_j = f(\text{linear\_output}_j)$$

$$= f(6)$$

$$= 7$$

Feedforward

Weighted Sum (1)



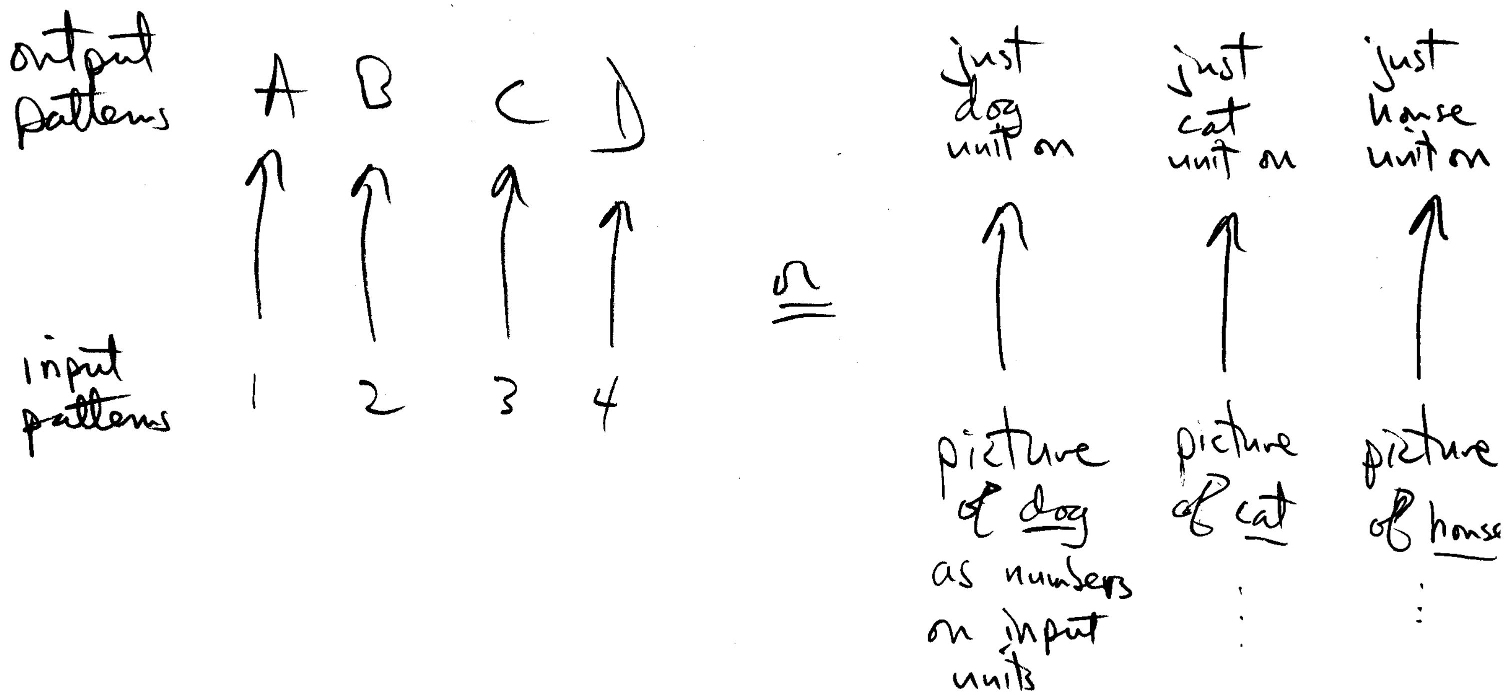
j's are outputs

$$\text{Output}_j = \sum_{\boxed{i}} \left[\text{Input}_i \times \text{weight}_{ij} \right]$$

= for all i,s

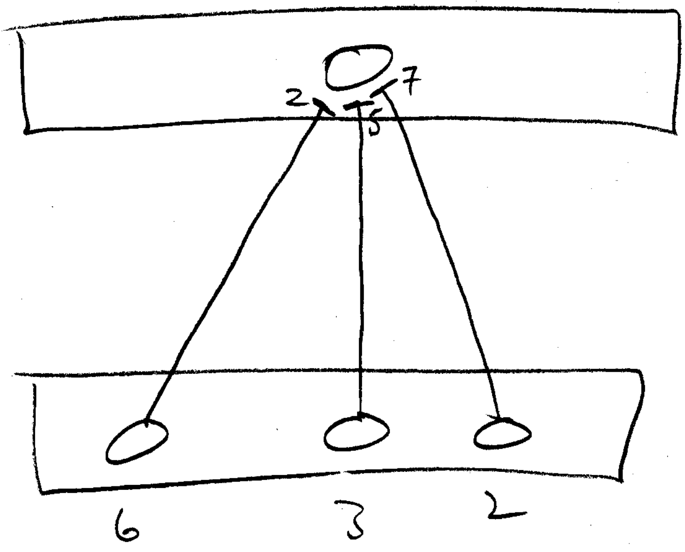
i's are inputs

Example of learning problem



How to get output error (2)

desired = 55
actual = 41

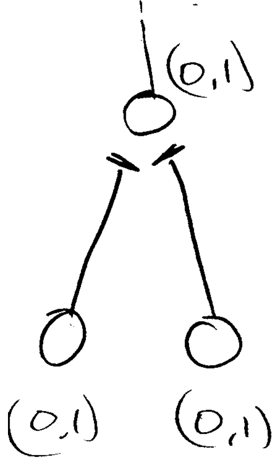


$$\boxed{\text{error} = \underset{\text{activation}}{\text{desired}} - \underset{\text{activation}}{\text{actual}}}$$

$$\text{output} = \sum(\text{inputs} \cdot \text{weights}) =$$
$$= 6 \times 2 + 3 \times 5 + 7 \times 2$$
$$= 12 + 15 + 14$$
$$= \boxed{41}$$

Problems w/ 2-layer

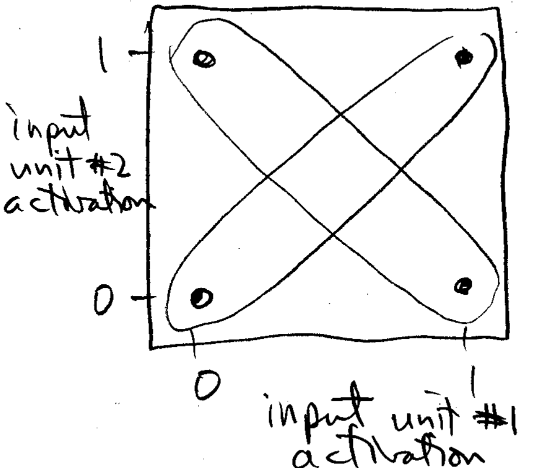
Network



Problem

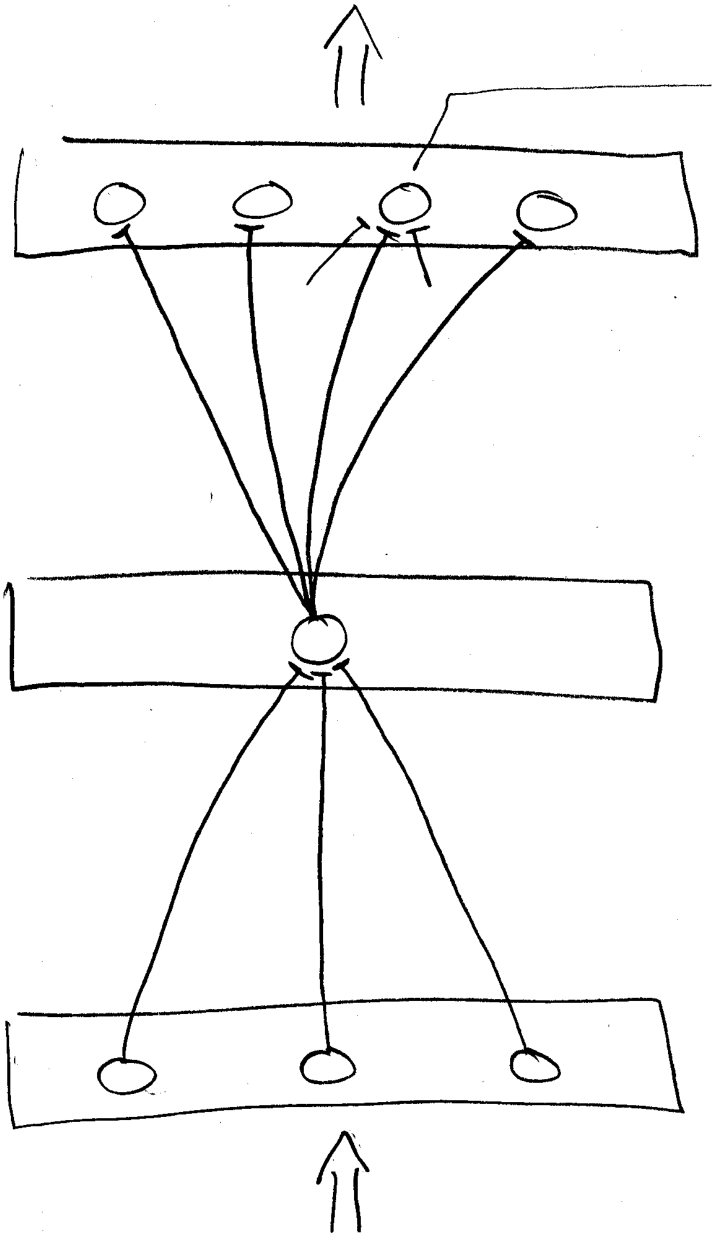
Input Patterns		Output Patterns
0 1	→	0
1 0	→	0
1 1	→	1
0 0	→	1

View a scatter plot of input space



How to get hidden error ③

output pattern calculated by
1) input → hidden Σ
2) hidden → output Σ



$\text{error}_{\text{output}} = \text{desired} - \text{actual}$

$\text{error}_{\text{hidden}} = \sum_{\text{outputs}} \left[\text{error}_{\text{output}} \times \text{weight}_{\text{to outp}} \right.$

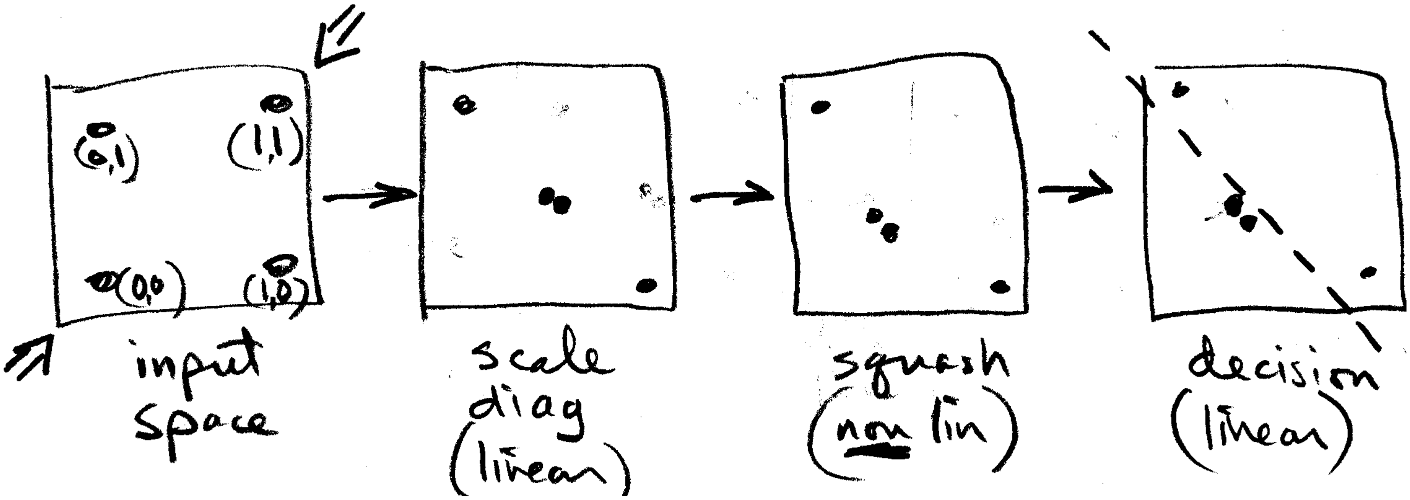
input pattern applied to input units

multiple layers
non-linearity
parity-like probs
(non-linearly separable)

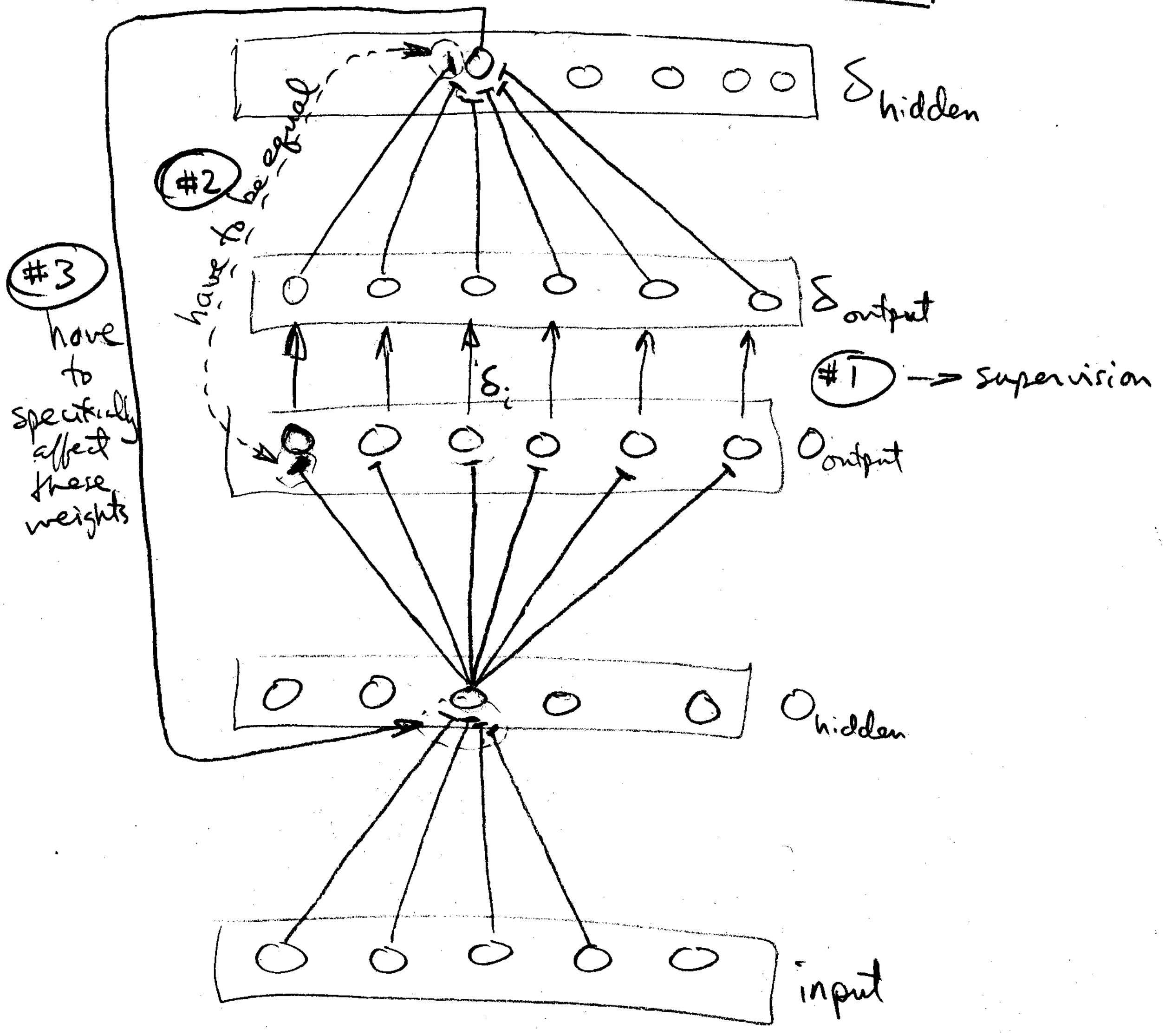
- if net is linear, the operations performed by n layers can be done all at once by one layer

- non-linearly separable require
  1) 3 layers
  2) non-linear in at least one

- how parity solved:



What's "bad" about backprop ("bad" → bio-implausible)



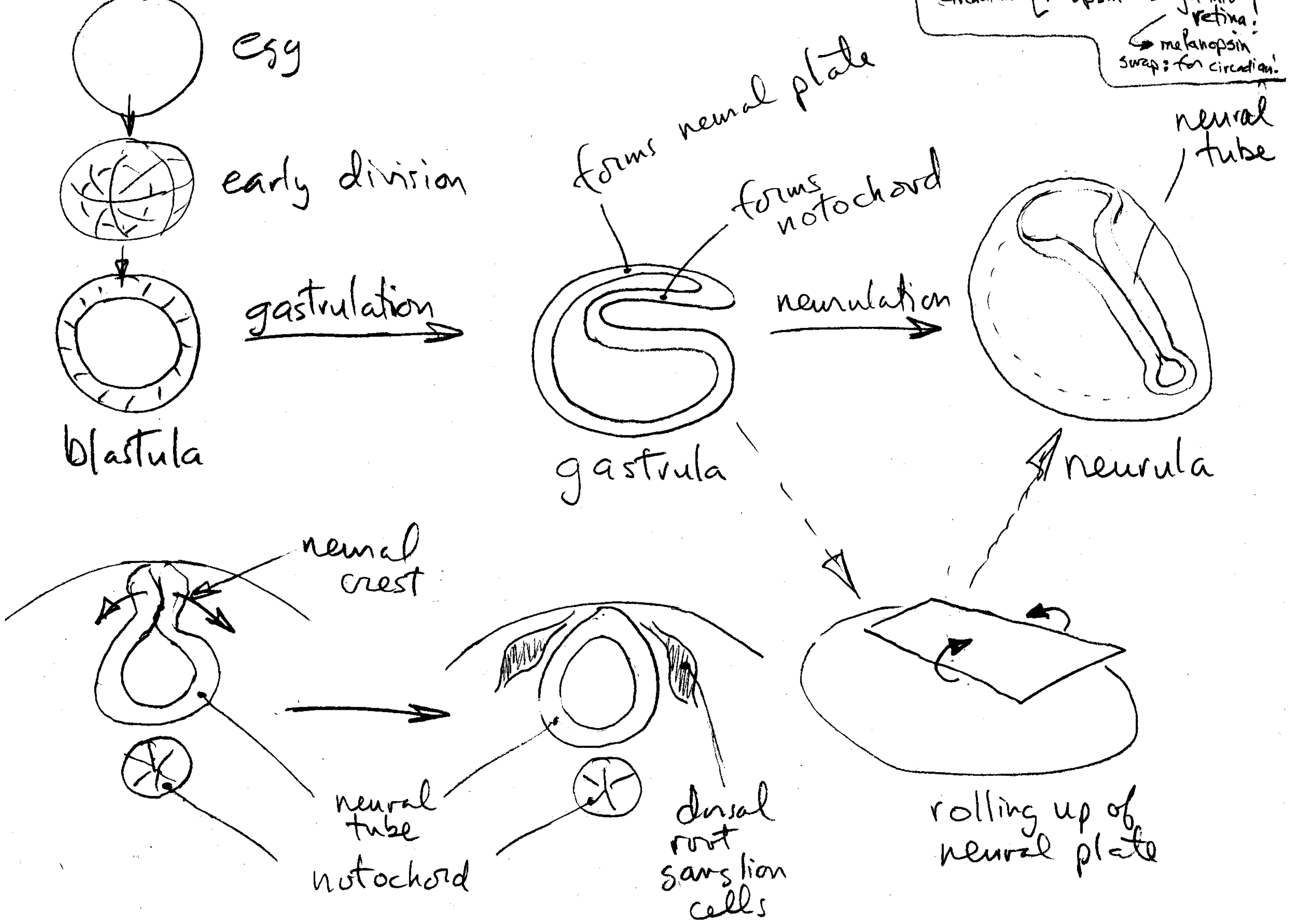
<u>eyes</u> pax6 ≈eyeless trans. fact, vitamin A, opsin

[rhabdomeric – → insects
[ciliary – → vertebrates

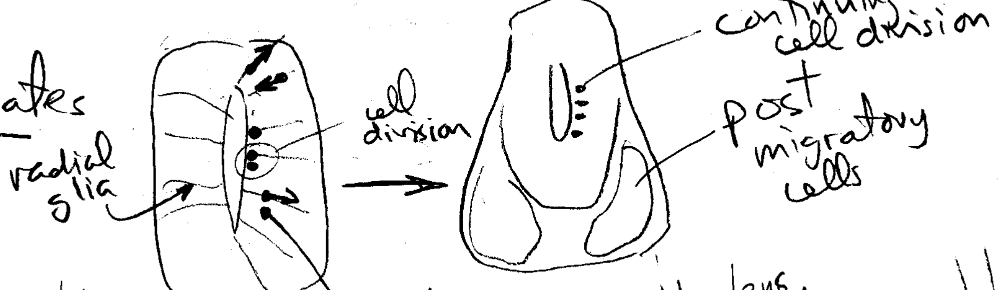
polychaete worm w/ both!

for circadian ← [c-opsin
[r-opsin → get into retina!
→ melanopsin swap: for circadian!

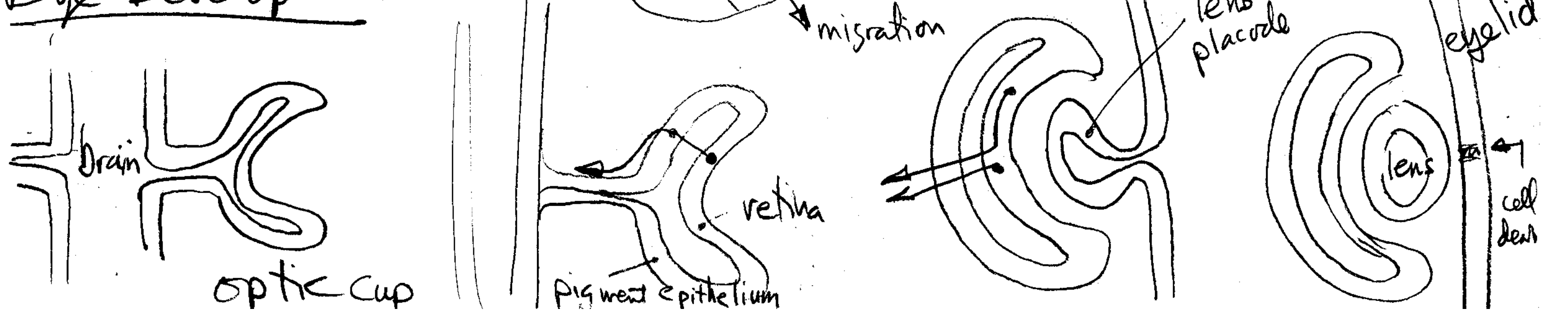
EARLY DEVELOPMENT



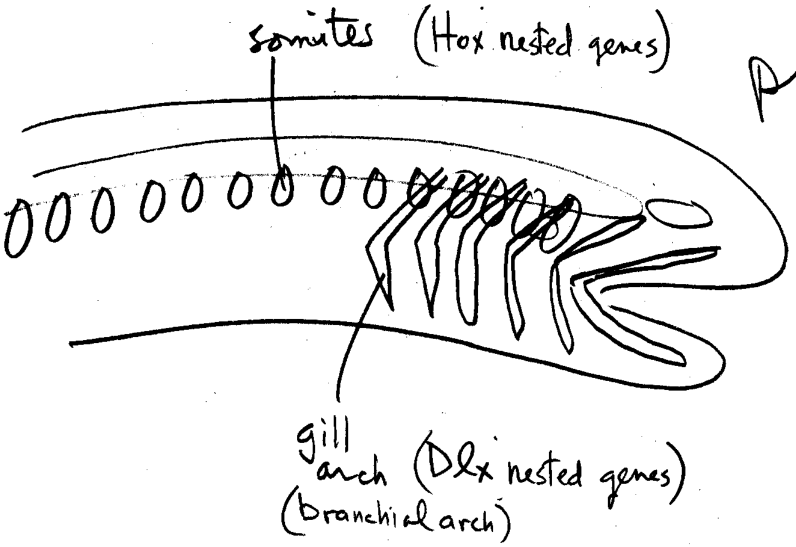
Migration/Birthdates



Eye Development

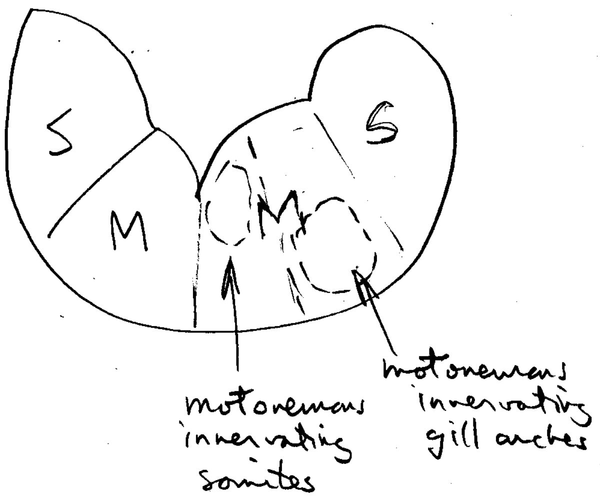


Somites & Gill Arches



primitive vertebrate

- antennapedia
- Hox DNA-binding proteins
- duplication
- tandem, like body order
- beware optimization

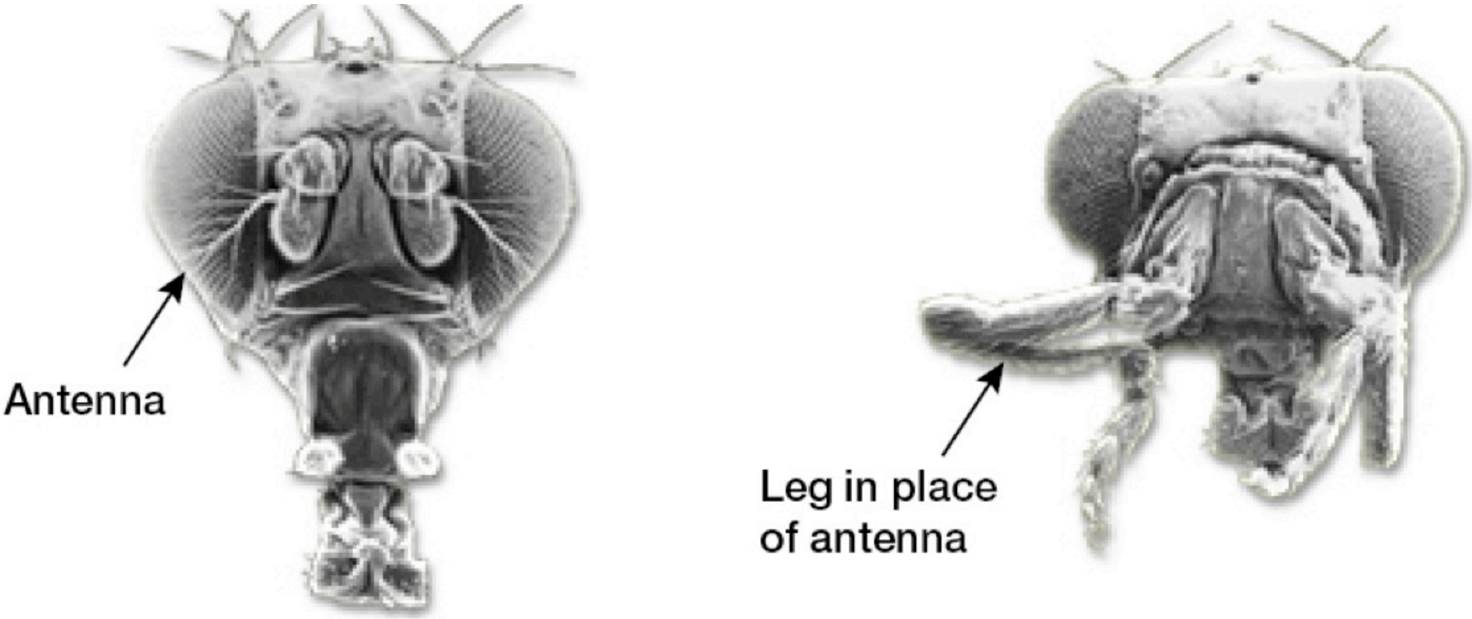


Somites

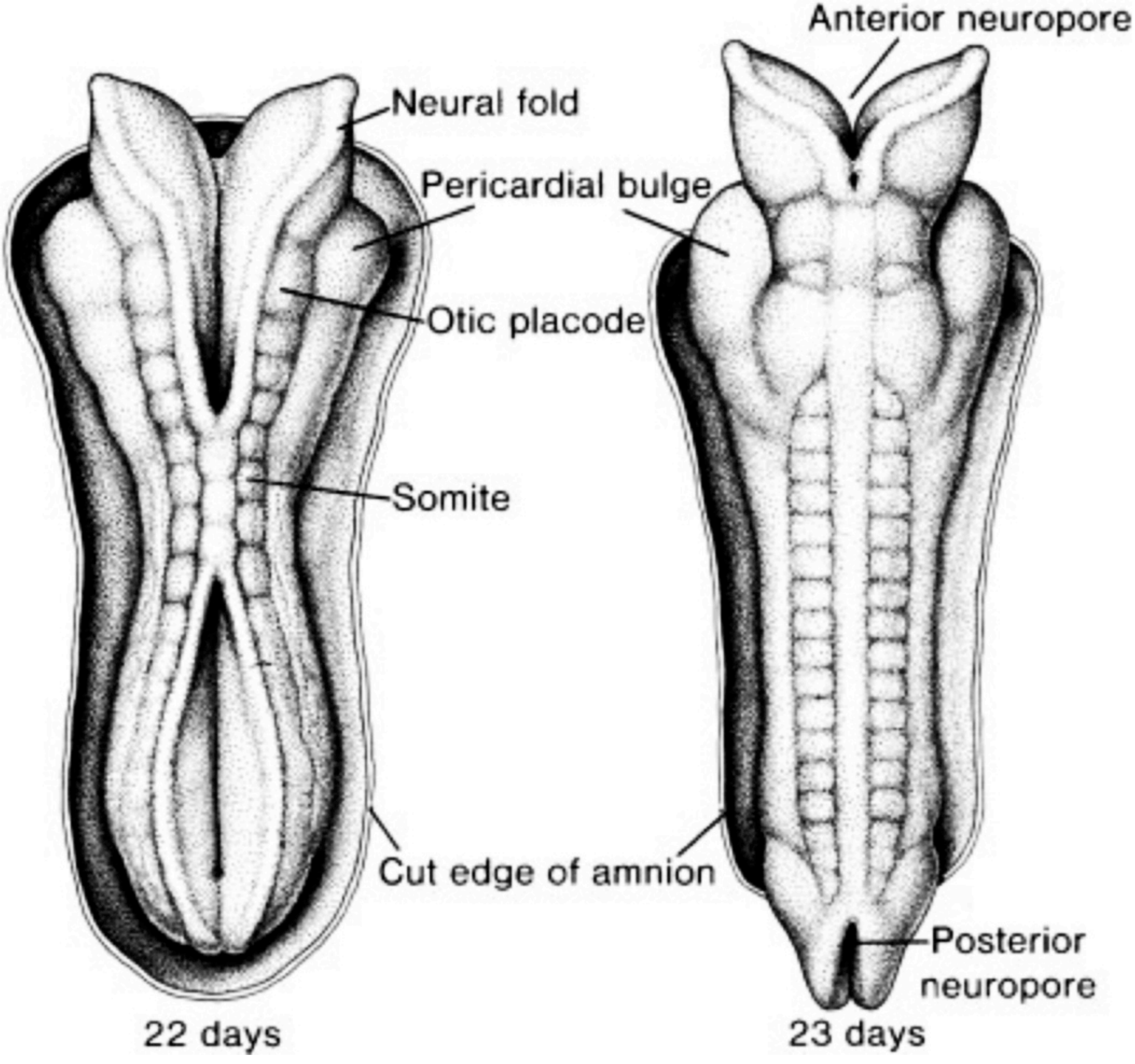
		location of motor nuclei in brain
1 2 3	⇒ 6 eye muscles	(4) III oculomotor (1) IV trochlear (1) VI abducens
4 5 6	⇒ tongue muscles	XII hypoglossal
7 8 9 ↓	⇒ body musculature	spinal motor neurons

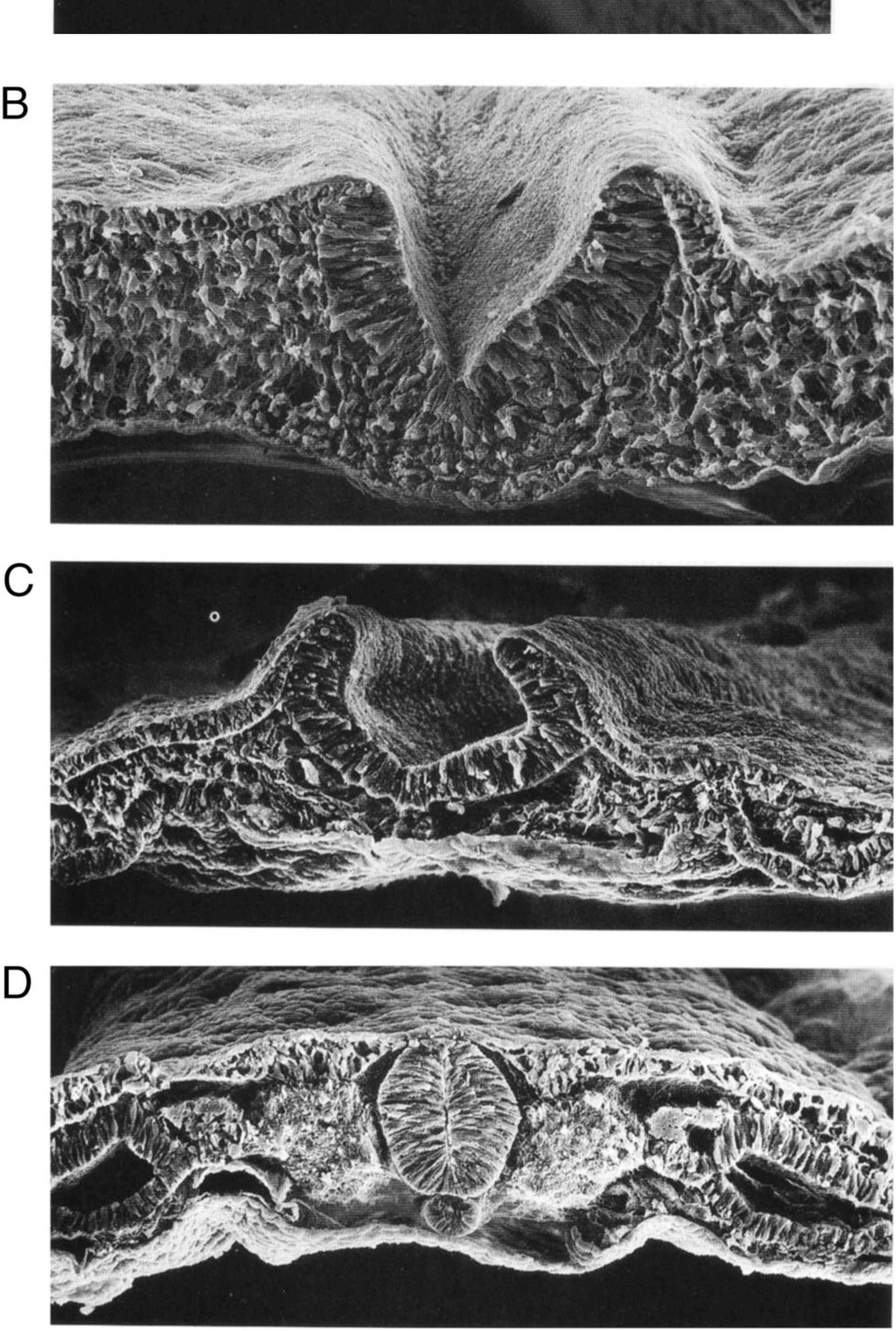
Gill Arches

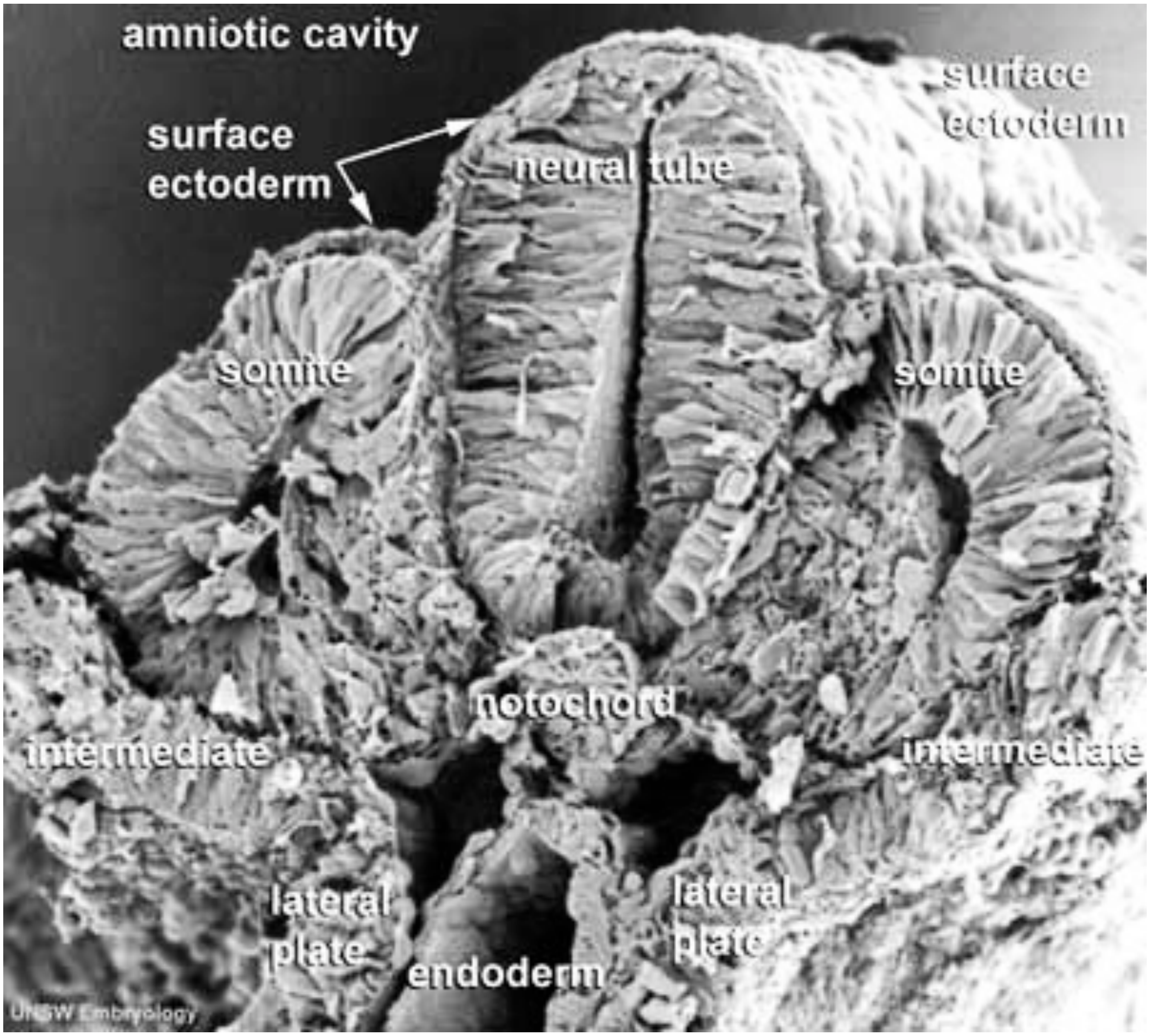
		location of motor nuclei in brain
1	⇒ jaw muscles	principle motor nuc. of trigeminal (V)
2	⇒ ear muscles musc. of facial expr.	facial motor nucleus (VII)
3 4 5 6 7	⇒ larynx, throat muscles	nucleus ambiguus

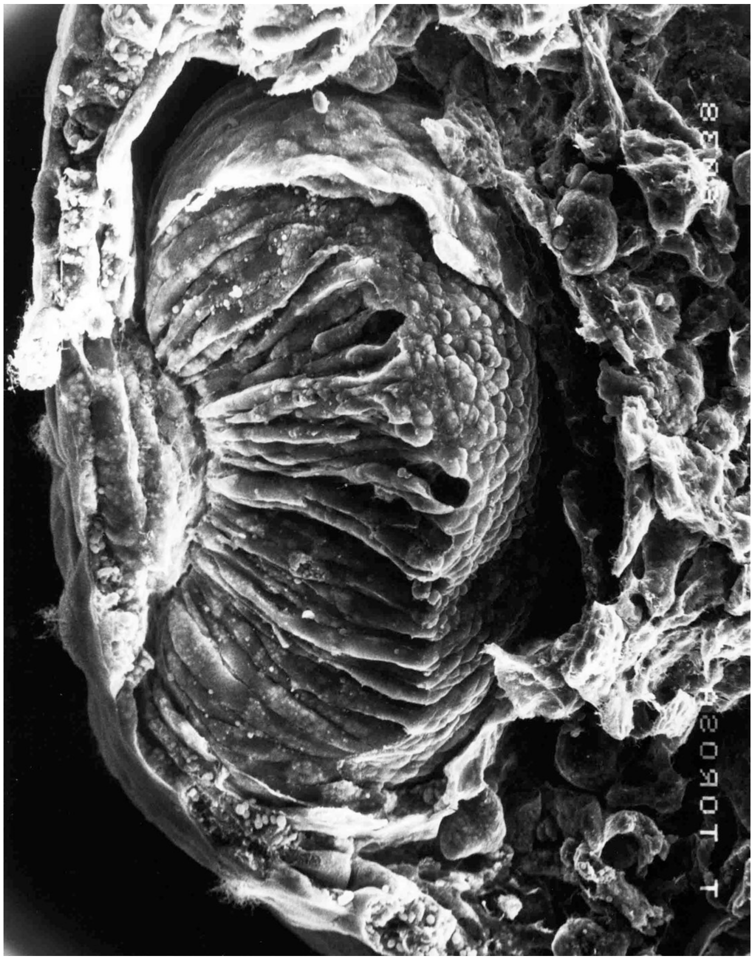

Antenna
Leg in place of antenna

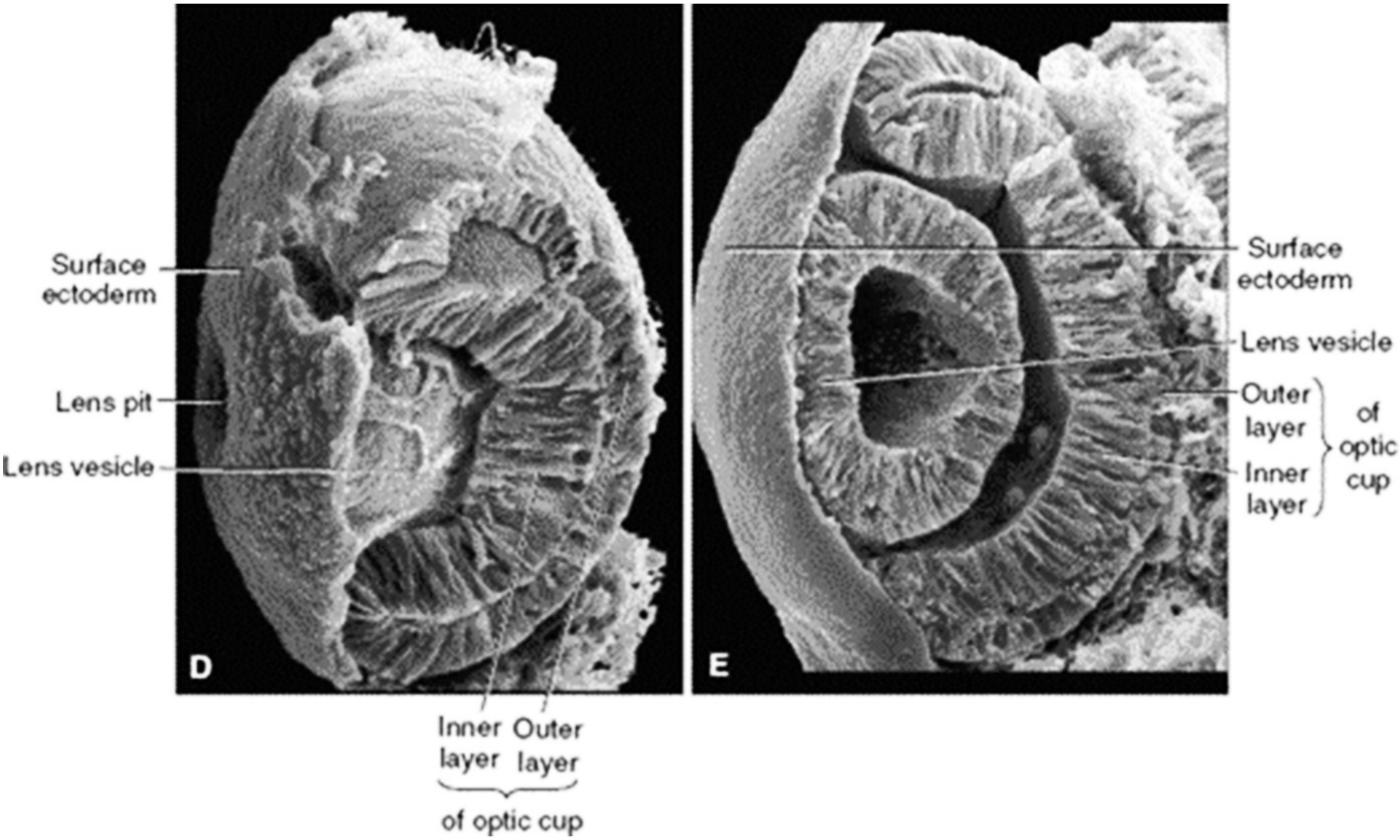

Haltere

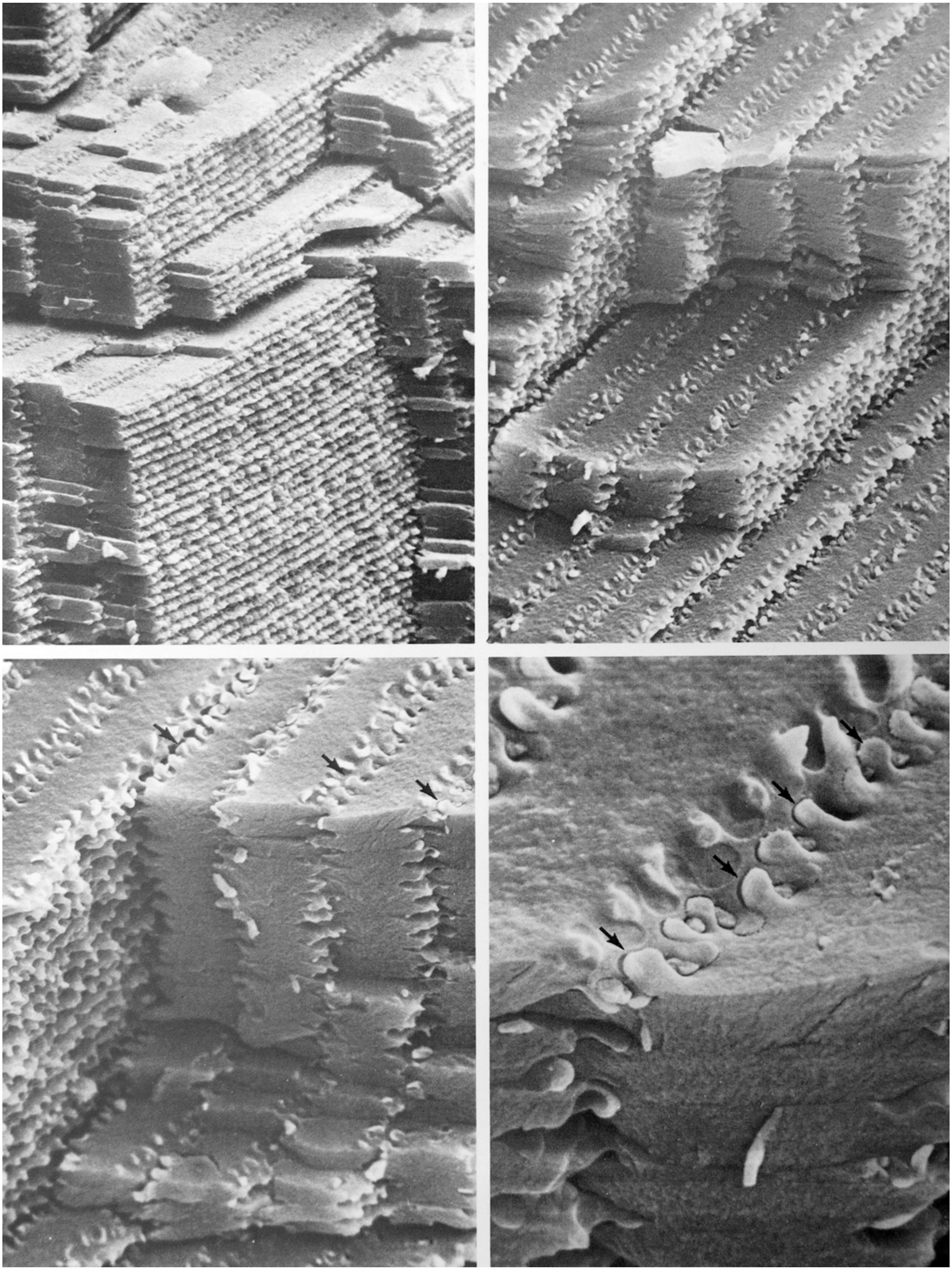

Neural fold
Pericardial bulge
Otic placode
Somite
Cut edge of amnion
22 days
Anterior neuropore
Posterior neuropore
23 days

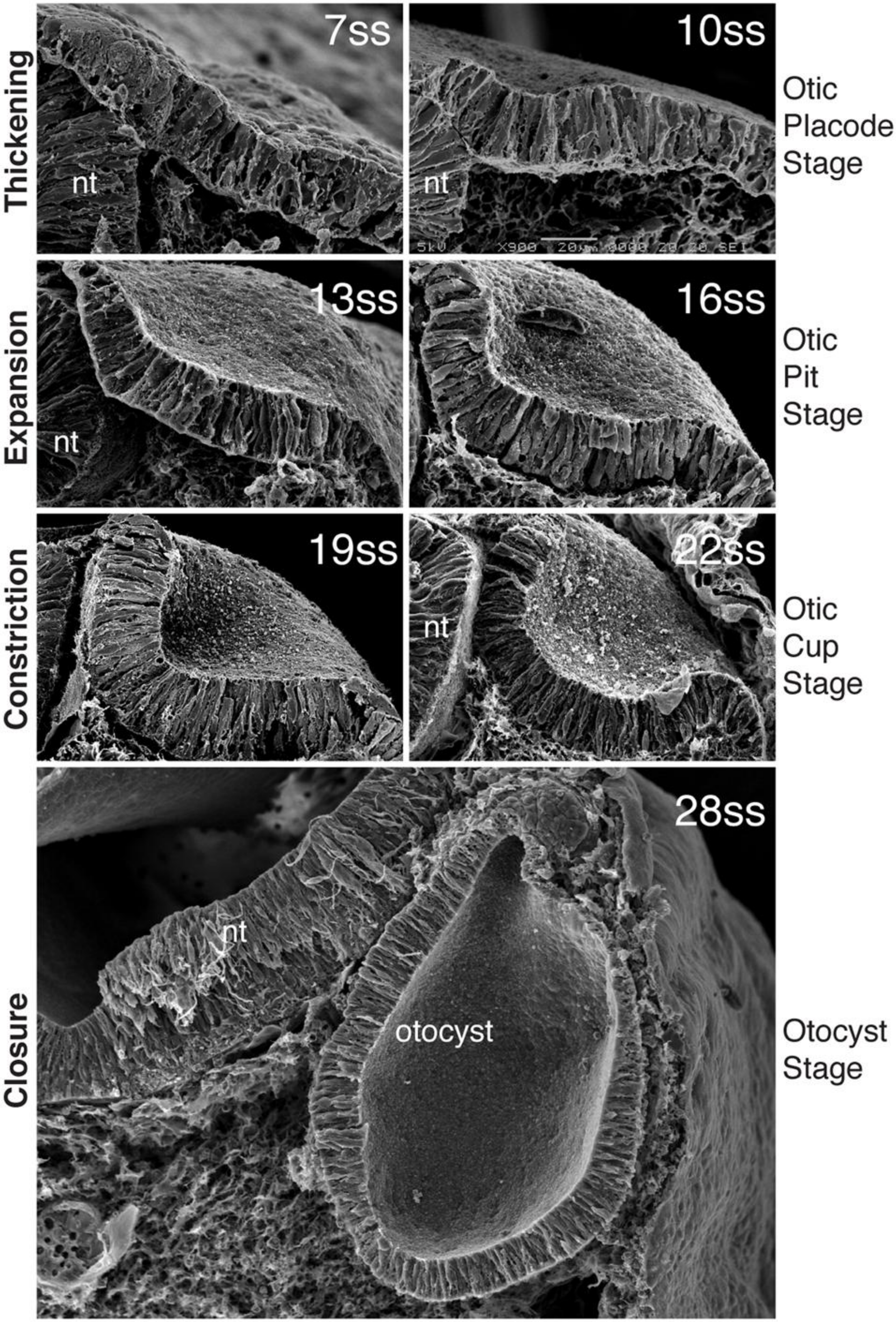

B
C
D


amniotic cavity
surface ectoderm
surface ectoderm
neural tube
somite
somite
notochord
intermediate
intermediate
lateral plate
lateral plate
endoderm
UNSW Embryology




Surface ectoderm
Lens pit
Lens vesicle
D
Inner layer
Outer layer
of optic cup
E
Surface ectoderm
Lens vesicle
Outer layer
Inner layer
of optic cup




Thickening
7ss
nt
10ss
nt
Otic Placode Stage
Basal Expansion
13ss
nt
16ss
Otic Pit Stage
Apical Constriction
19ss
22ss
nt
Otic Cup Stage
Closure
28ss
nt
otocyst
Otocyst Stage

"5 Minutes on Energy" :-}

Basic average numbers to remember for solar electric. Power hitting the atmosphere is 1366 watts/m^2. Practically available average power considering atmospheric losses, oblique, diurnal and weather variation *before* conversion to electricity is about 190 watts/m^2. Power available after conversion including defects, soiling, and inverter and spacing losses is about 15 watts/m^2 (=1.4 watts/sq ft). For scale, a standard sized car is a 100,000 watt device (135 horsepower) at maximum output, and can cruise smoothly at highway speeds on about 20,000 watts. Therefore, to directly power a car crusing at 60-70 mph (no storage), you need the average output of 14,000 sq feet of solar cells -- an array 120 feet by 120 feet, which is 1/4 the area of an American football field. At current prices, such an array (at volume discount) would cost over $300,000. For strong acceleration (like they do here in London trying to pass me on my bicycle on their way to a red light), you need a million-dollar full football field's worth of solar cells. That's why people are going to eventually be driving smaller, lighter cars at lower speeds, and accelerating less -- which is excellent news for cyclists

fossil fuel lifeblood → from localized bio failure to recycle

(instantaneous)
power = watts = I · V (kW)

energy = watts · time (kWh)
[time integral power]

barrel of oil

42 gal
- diesel (cargo cult)
- kerosine
- 20 gal gasoline

⇓

25% efficient

["bicyclist" ≈ 100 watts
SUV ≈ 200,000 watts]

↓

<u>one year</u> of 100 watts
@ 6 hours cont./day

— stomping on SUV accelerator
↳ 2 football fields solar cells

— all batteries produced in one current year
↳ 1-2 min grid storage

— grid ≈ cars

— wind + solar growth has not even kept up with overall demand growth

SCHEMA

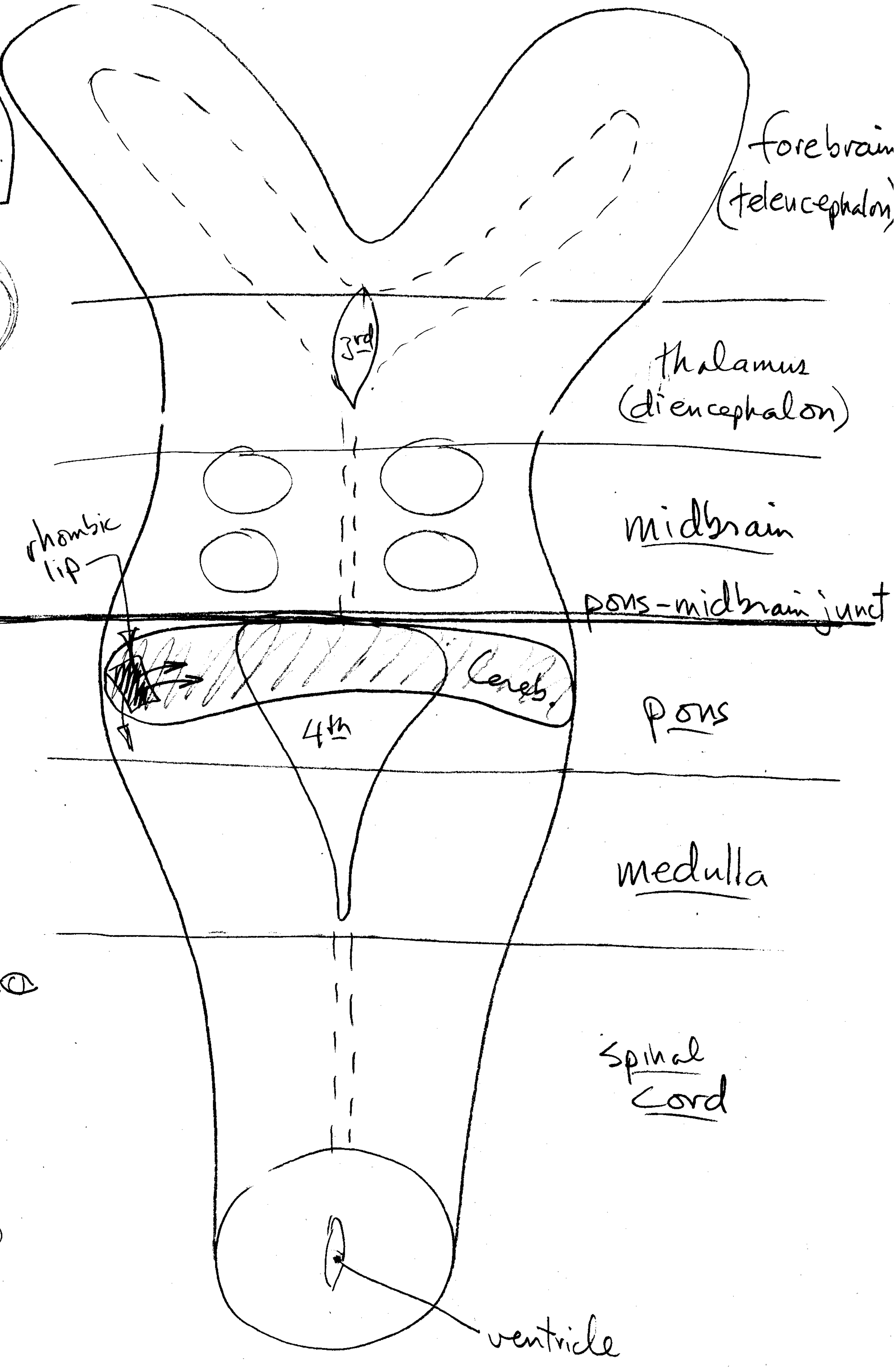
The "primal twist"

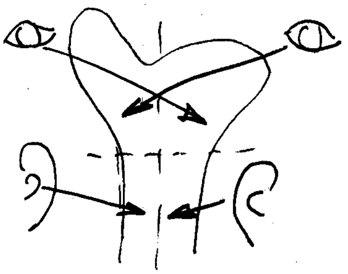
related to opposite side of body

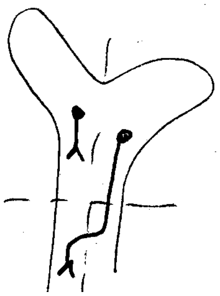
↑

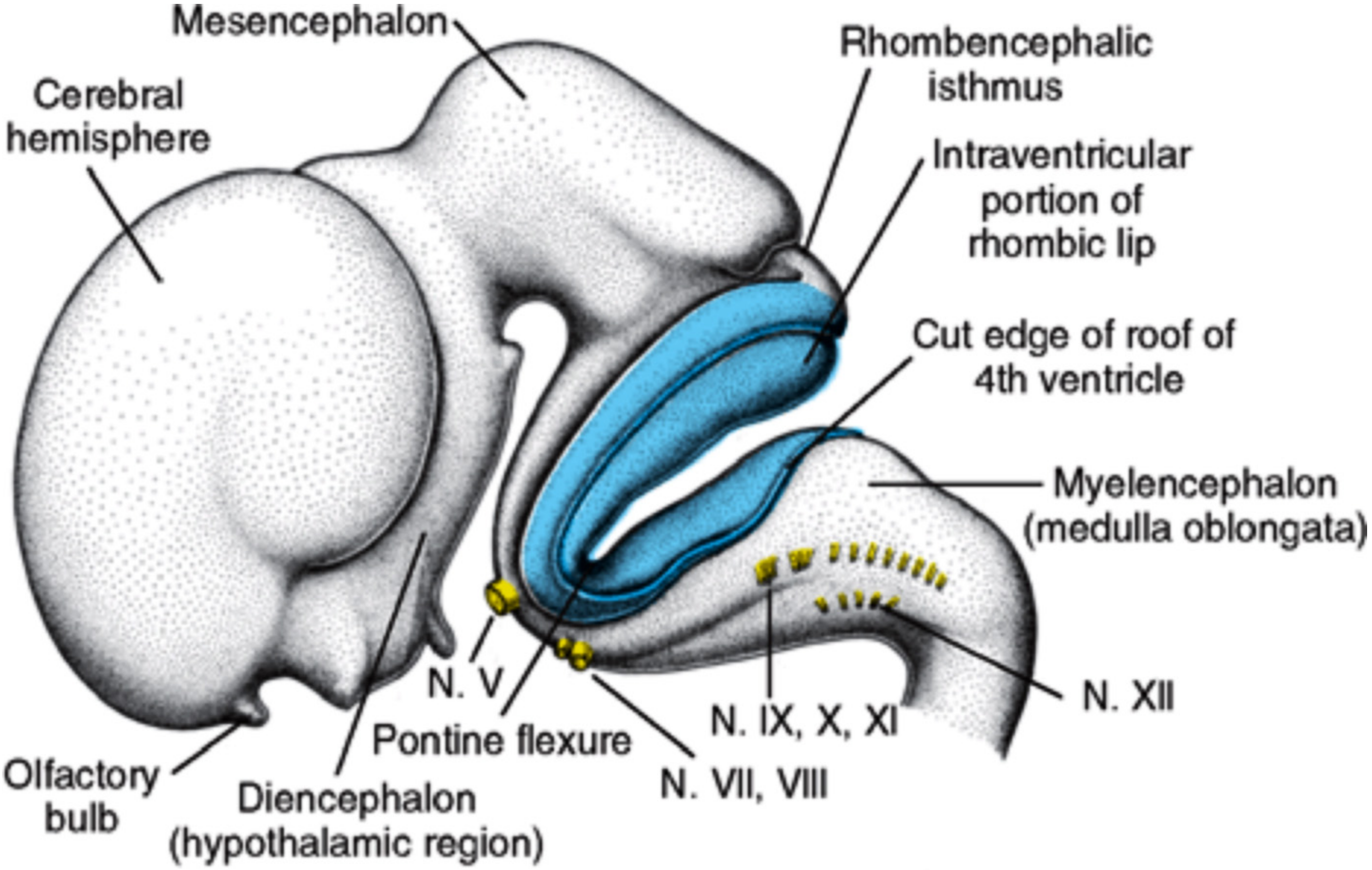
↓

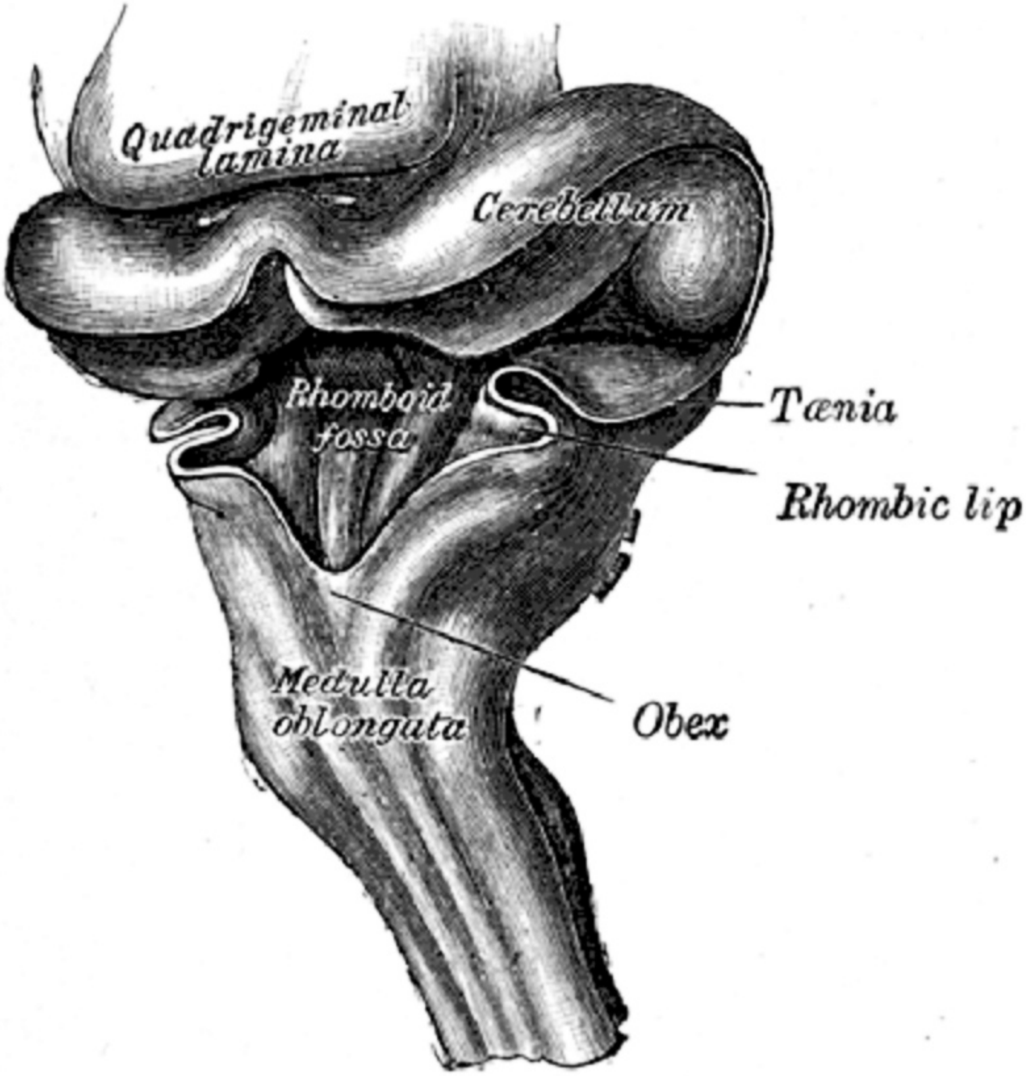
related to same side of body





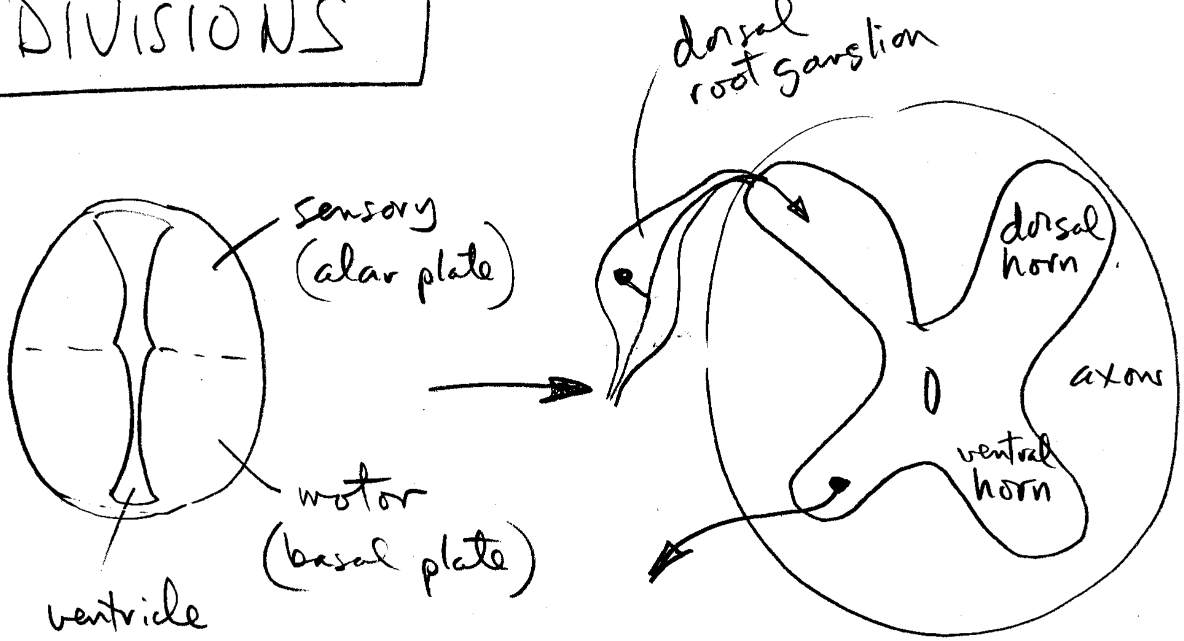



Mesencephalon
Cerebral hemisphere
Rhombencephalic isthmus
Intraventricular portion of rhombic lip
Cut edge of roof of 4th ventricle
Myelencephalon (medulla oblongata)
N. V
N. XII
N. IX, X, XI
Pontine flexure
N. VII, VIII
Olfactory bulb
Diencephalon (hypothalamic region)

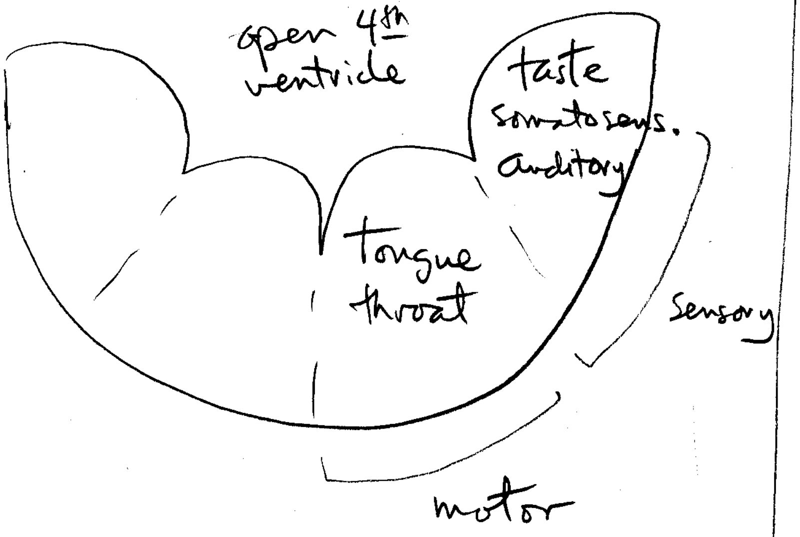

Quadrigeminal lamina
Cerebellum
Tænia
Rhomboid fossa
Rhombic lip
Obex
Medulla oblongata

MAIN DIVISIONS

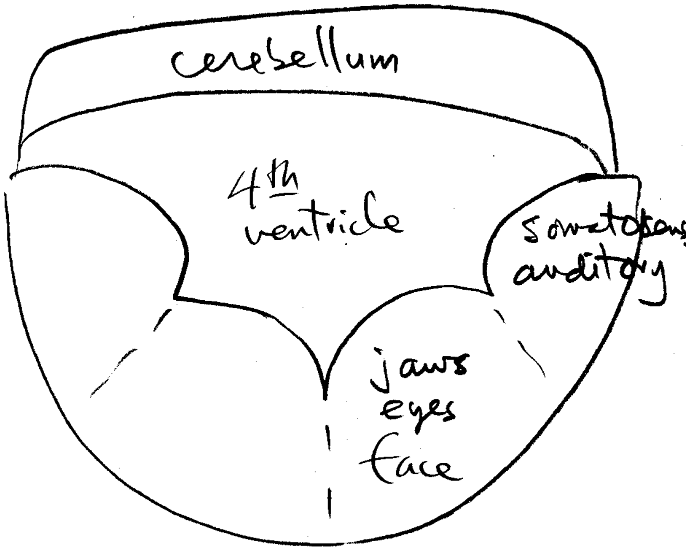
Spinal cord



medulla

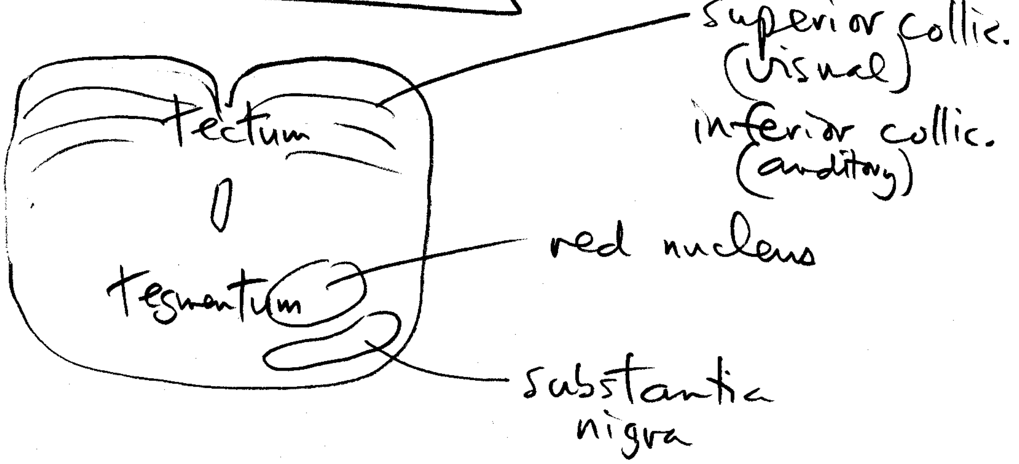


pons



MAIN DIVISIONS (cont)

midbrain



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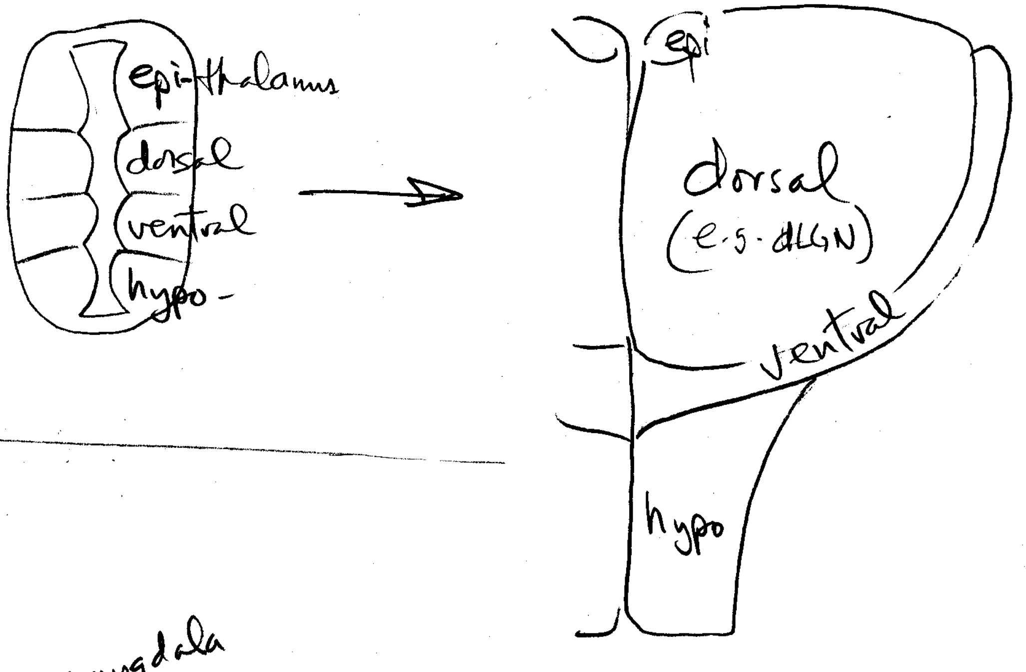
thalamus



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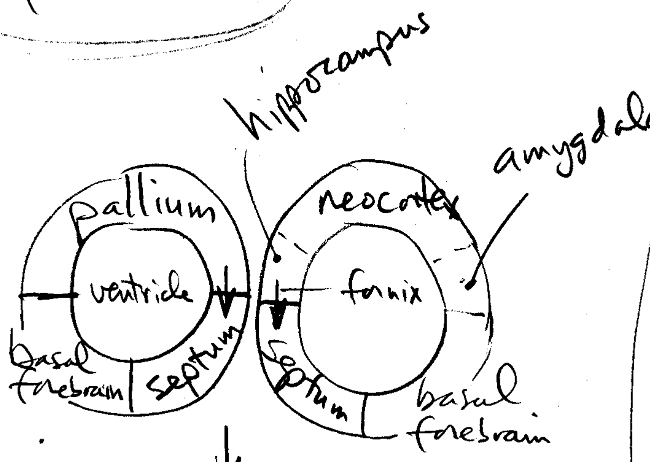
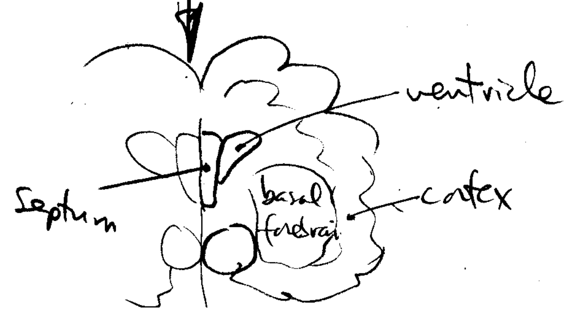
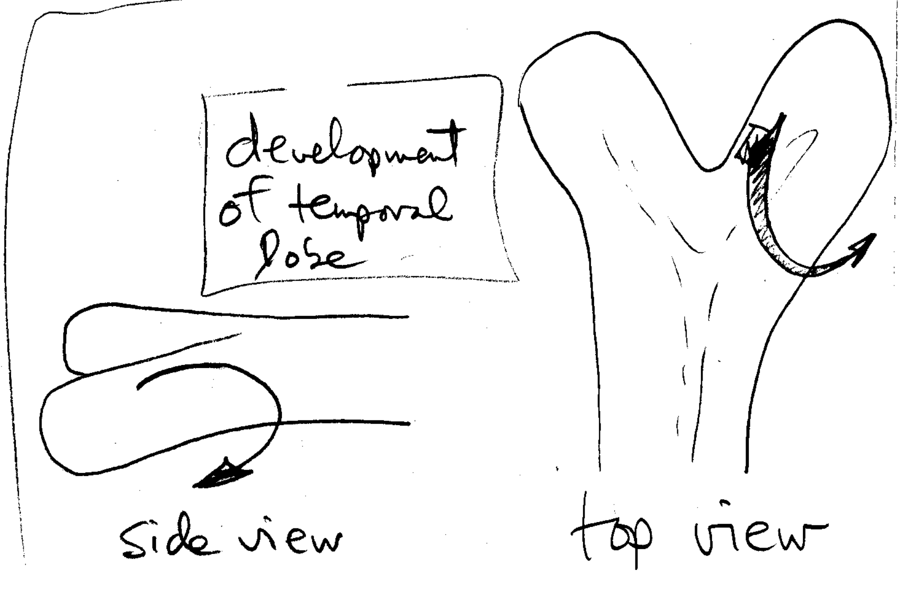
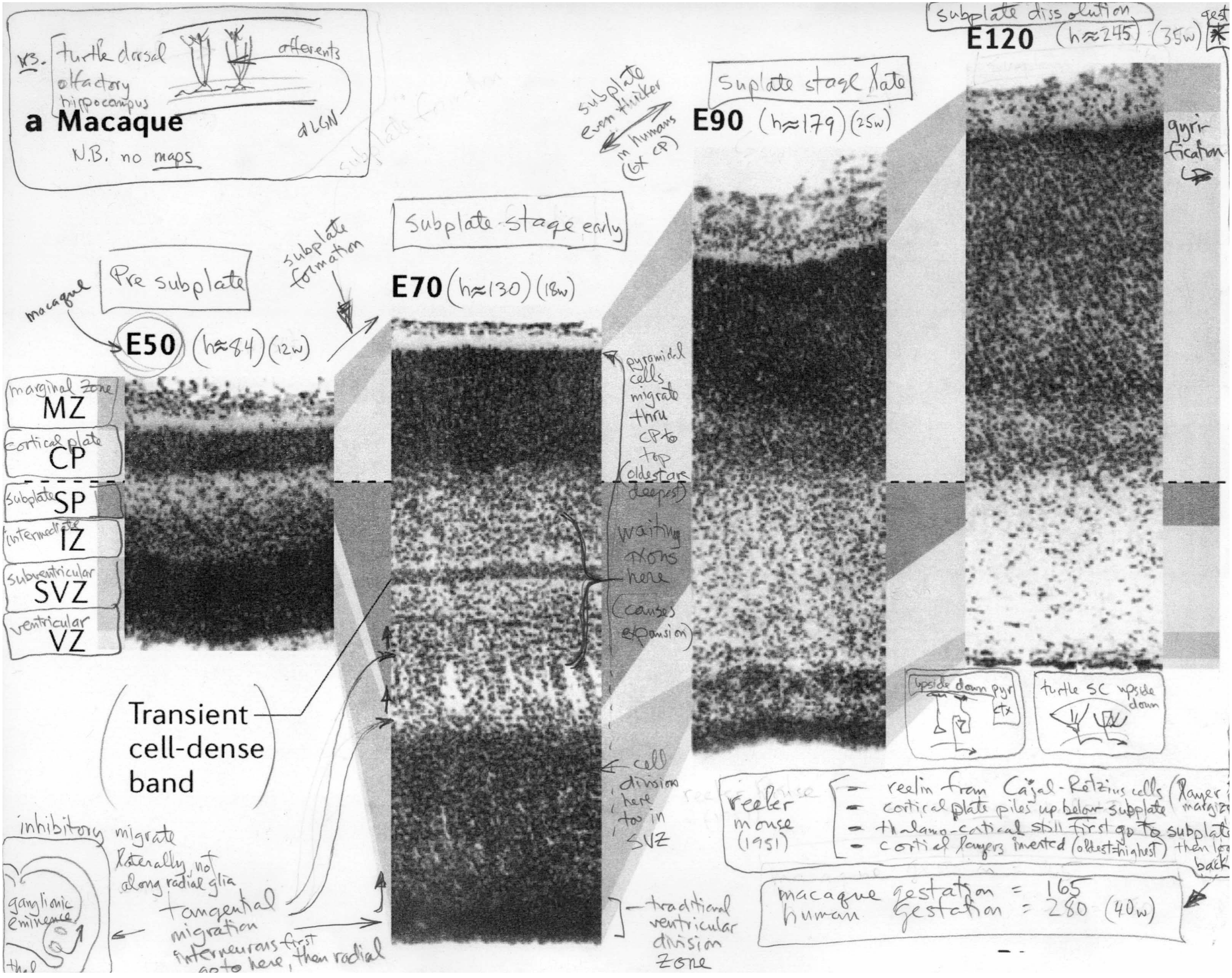
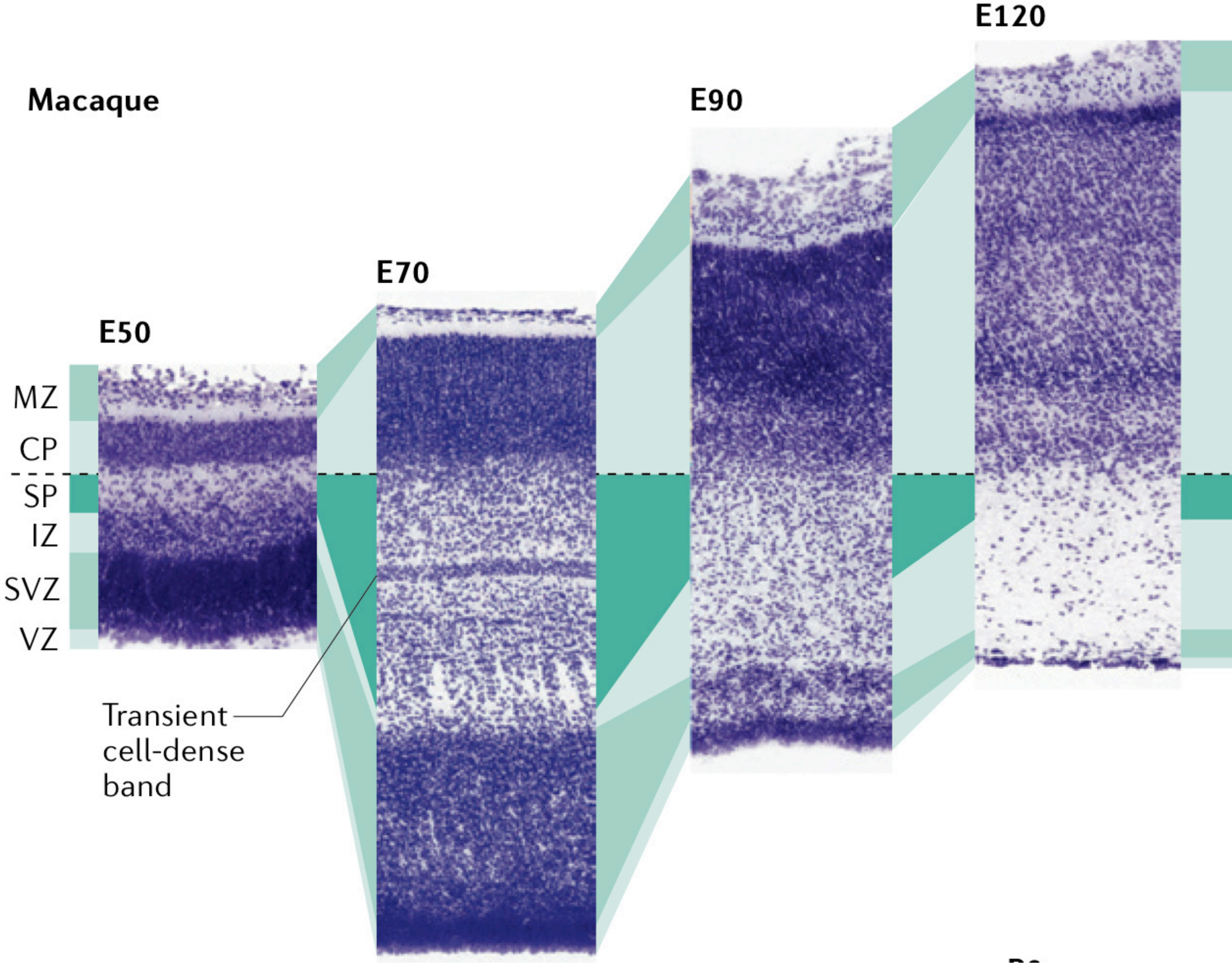
forebrain

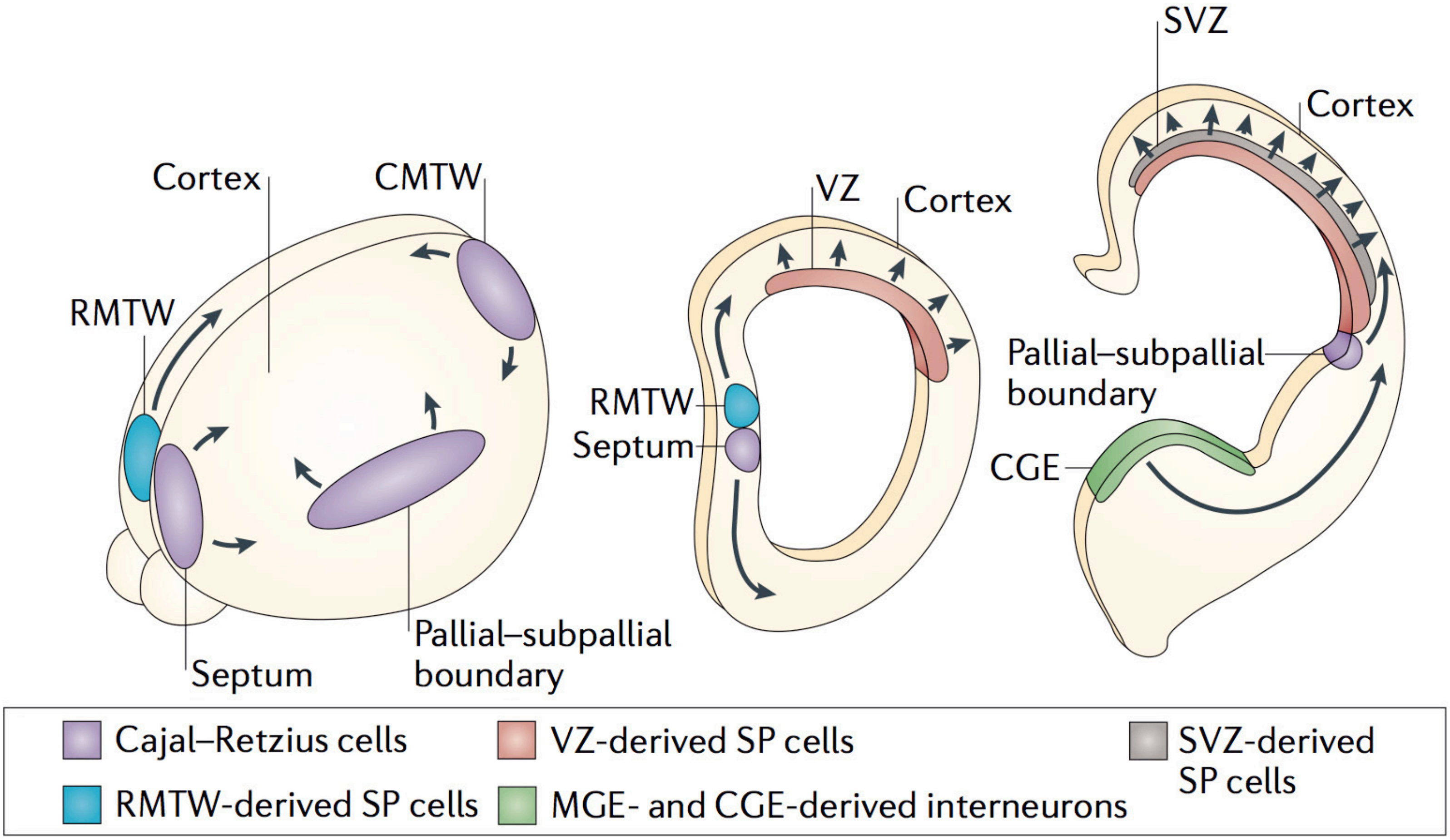





development of temporal lobe

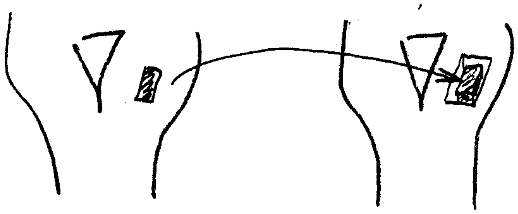

a Macaque
N.B. no maps
vs. turtle dorsal
olfactory
hippocampus
afferents
dLGN
macaque
Pre subplate
E50 (h≈84) (12w)
subplate formation
Subplate stage early
E70 (h≈130) (18w)
subplate even thicker in humans (6x CP)
Suplate stage late
E90 (h≈179) (25w)
subplate dissolution
E120 (h≈245) (35w)
gest
gyrification
marginal zone
MZ
cortical plate
CP
subplate
SP
intermediate
IZ
subventricular
SVZ
ventricular
VZ
Transient cell-dense band
pyramidal cells migrate thru CP to top (oldest are deepest)
waiting axons here (causes expansion)
cell division here, too in SVZ
traditional ventricular division zone
inhibitory migrate laterally, not along radial glia
tangential migration
interneurons first go to here, then radial
ganglionic eminence
thal
upside down pyr ctx
turtle SC upside down
reeler mouse (1951)
- reelin from Cajal-Retzius cells (layer
- cortical plate piles up below subplate
- thalamo-cortical still first go to subplate
- cortical layers inverted (oldest=highest) then
margin
back
macaque gestation = 165
human Gestation = 280 (40w)


Macaque
E50
E70
E90
E120
MZ
CP
SP
IZ
SVZ
VZ
Transient cell-dense band


Cortex
CMTW
RMTW
Septum
Pallial–subpallial boundary
VZ
Cortex
RMTW
Septum
SVZ
Cortex
Pallial–subpallial boundary
CGE
Cajal–Retzius cells
RMTW-derived SP cells
VZ-derived SP cells
MGE- and CGE-derived interneurons
SVZ-derived SP cells

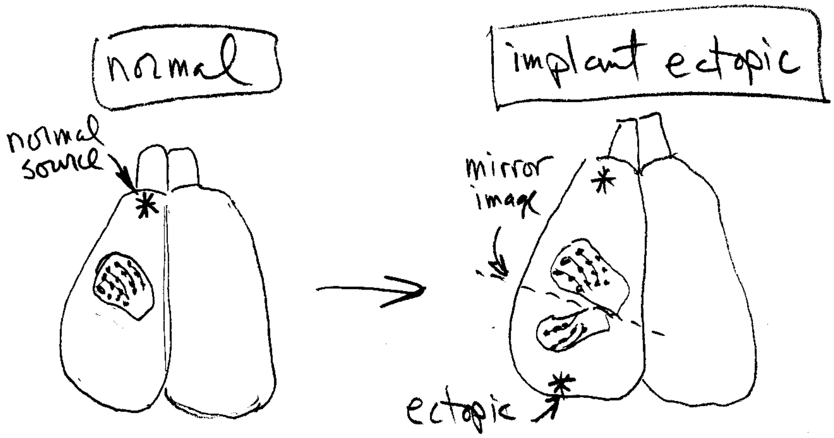
DEVELOPMENT EXPTS

Chick/quail embryonic brain grafts



- operate on egg
- transplant vocalization "sound"
- transplant vocalization head movement

ectopic fgf8 source implant (mouse) (fibroblast growth factor #8)



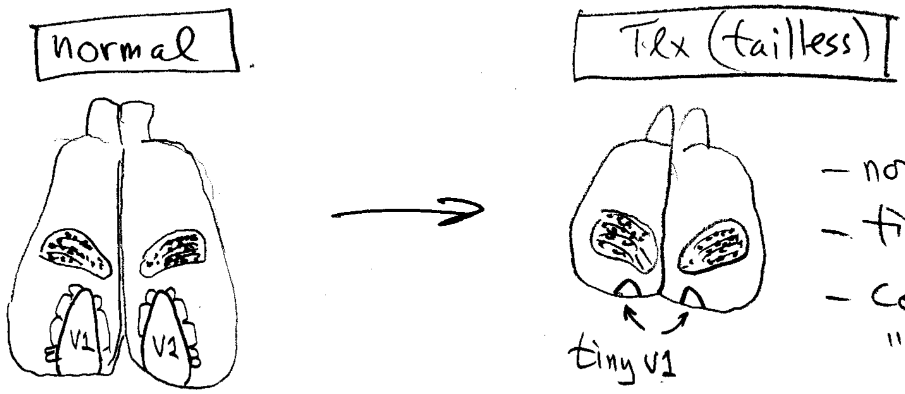
- many cortical areas are mirror-images, e.g.:
  - V1/V2
  - SII/SII
  - AI/R
- evol. of areas < duplication / fusion / splitting
- what is cortical area?
  - ↳ cf. ear bone evolution function can move!

tailless (mouse mutant)

fly = Tll
mouse = Tlx

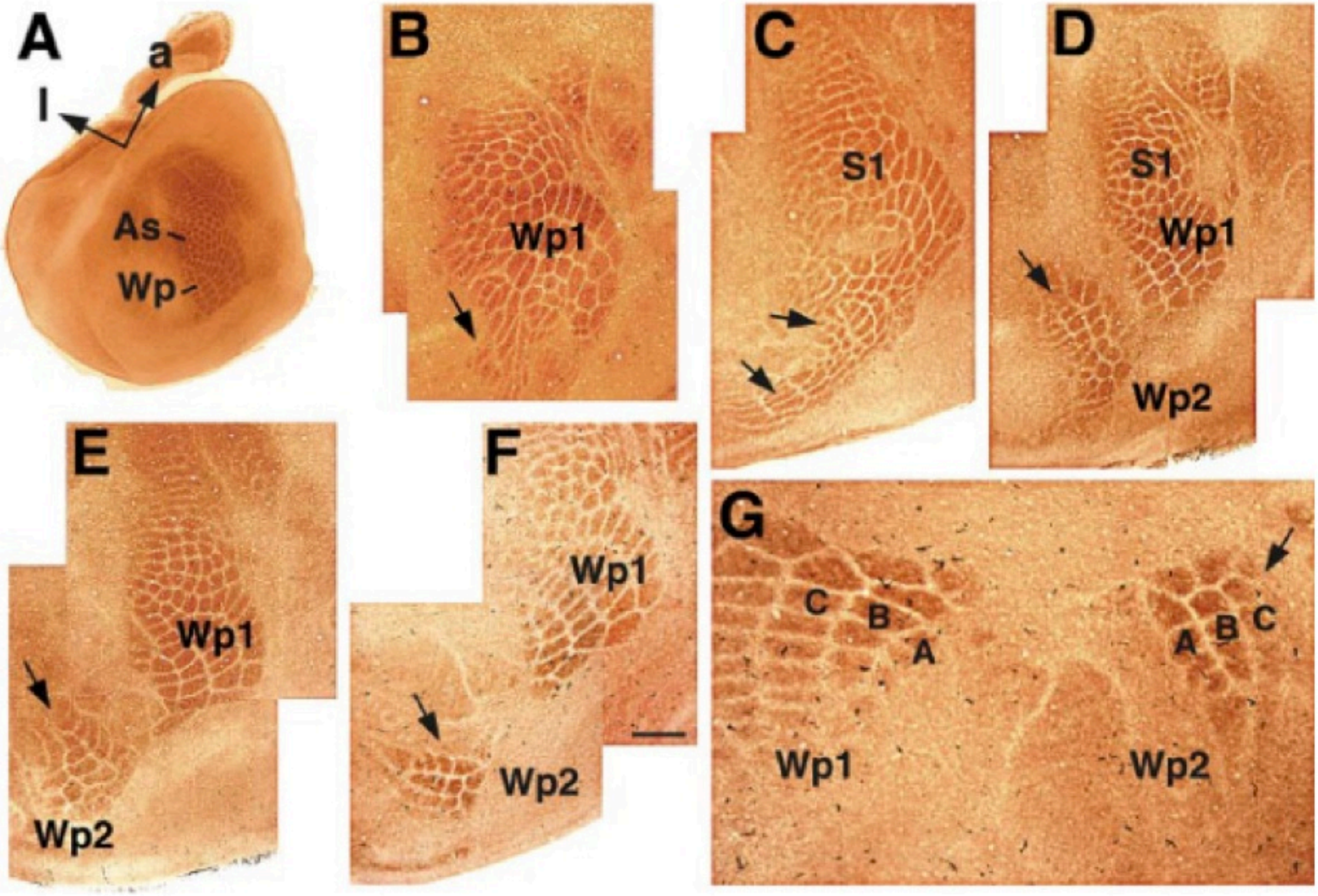
flies : structures from segments beyond 8th abdominal

mice : missing tail, small cortex, agressive behavior, no neurogenesis in subventricular zone



- normal size S-I
- tiny V1
- cortex does not "regulate" (adjust to give similar proportions)

fgf8: Duplication and Divergence



from E.A. Grove (2003)

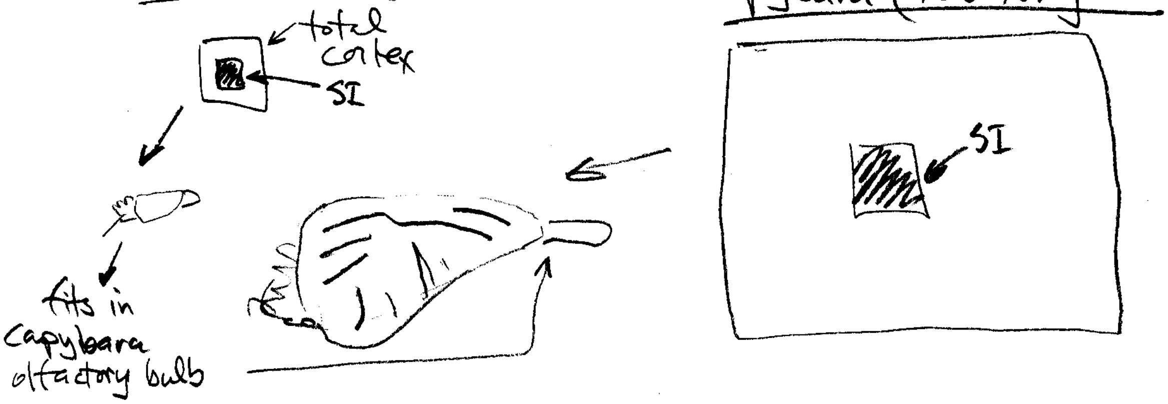
FORMATION OF GYRI/SULCI

brain size

- 95% of brain size variance explained by body size
- correlation body size and first PC of brain size measurements: 0.998
- 0.96 corr. of indiv. structures w/ total brain size
- as body/brain size increases, primary sensory areas occupy smaller percentages of total cortex

white footed mouse

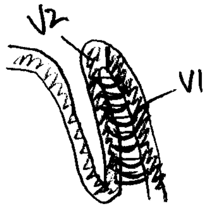
capybara (150 lb. guinea pig)



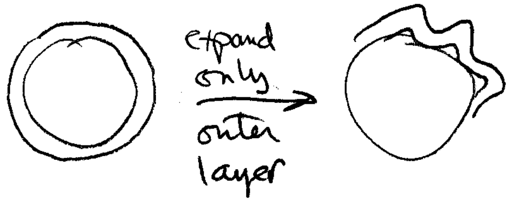
- larger bodied animals get more non-primary cortex 'for free' as it were!

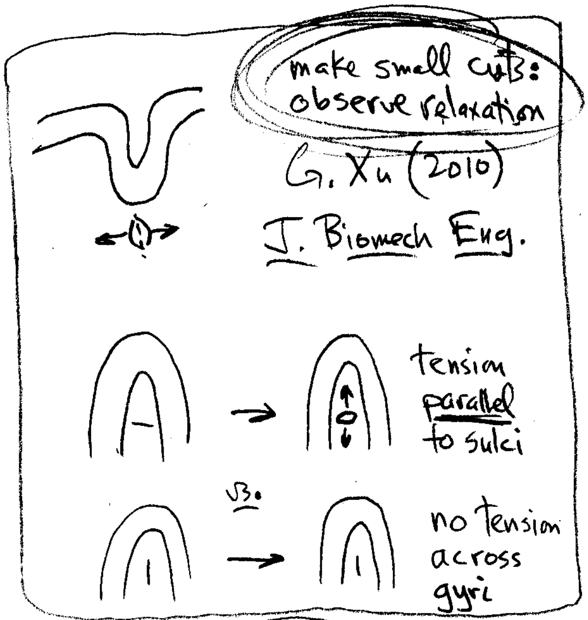
formation of gyri (Tallinen)

Van Essen tension idea



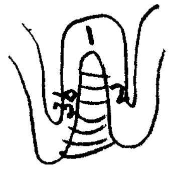
physical expts. suggest opposite

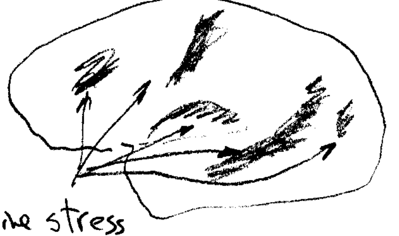




Tallinen (2016)

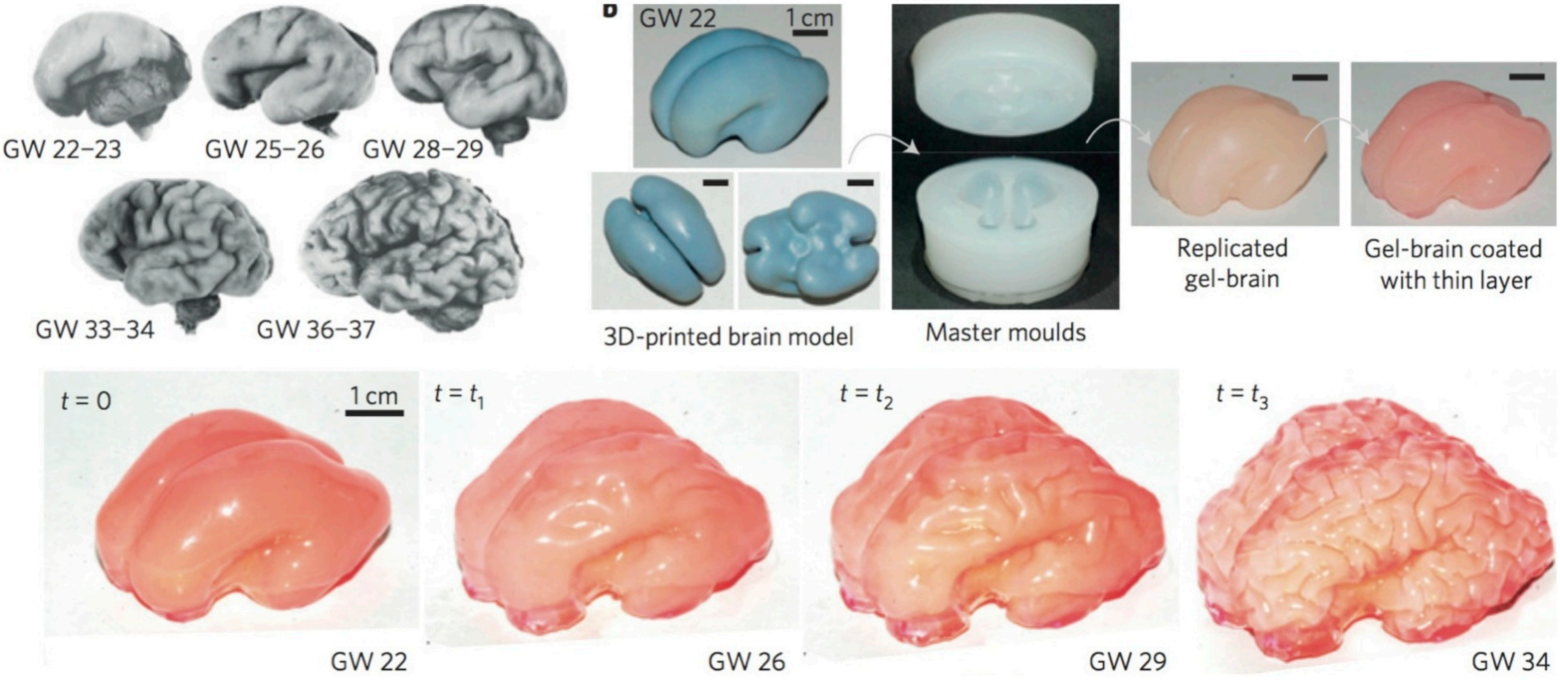
- coat expanding gel (ctx) over rigid WM-shaped non-expanding armature → human-like folding
- also, finite element models





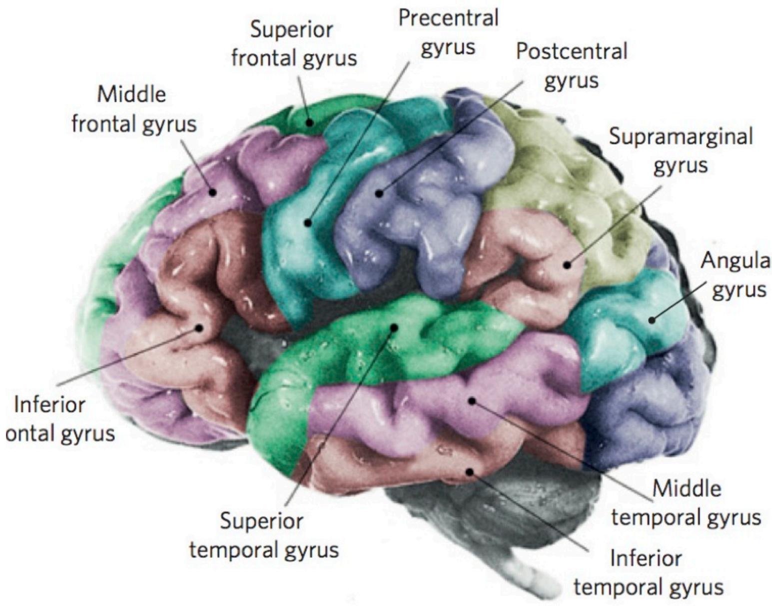
Physical Simulation

(thin outer elastomer gel layer expands
over non-expanding gel brain base)

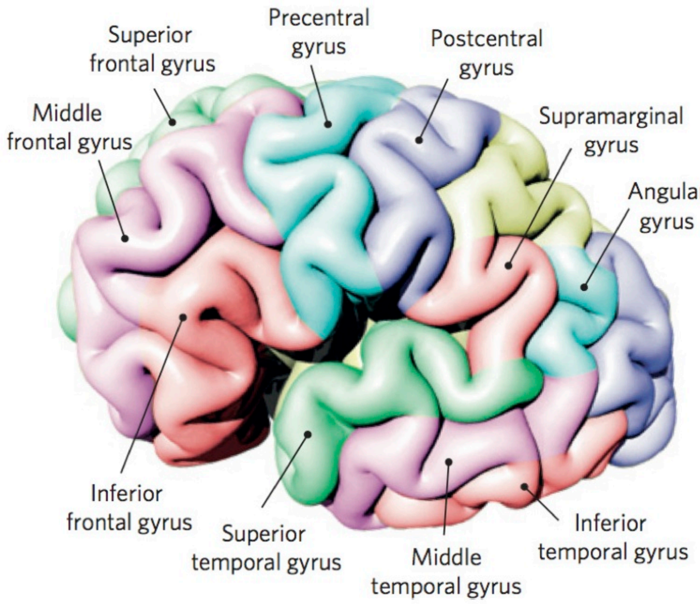


Tallinen et al. (2016) *Nature Physics* (doi:10.1038/NPHYS3632)

Real Brain

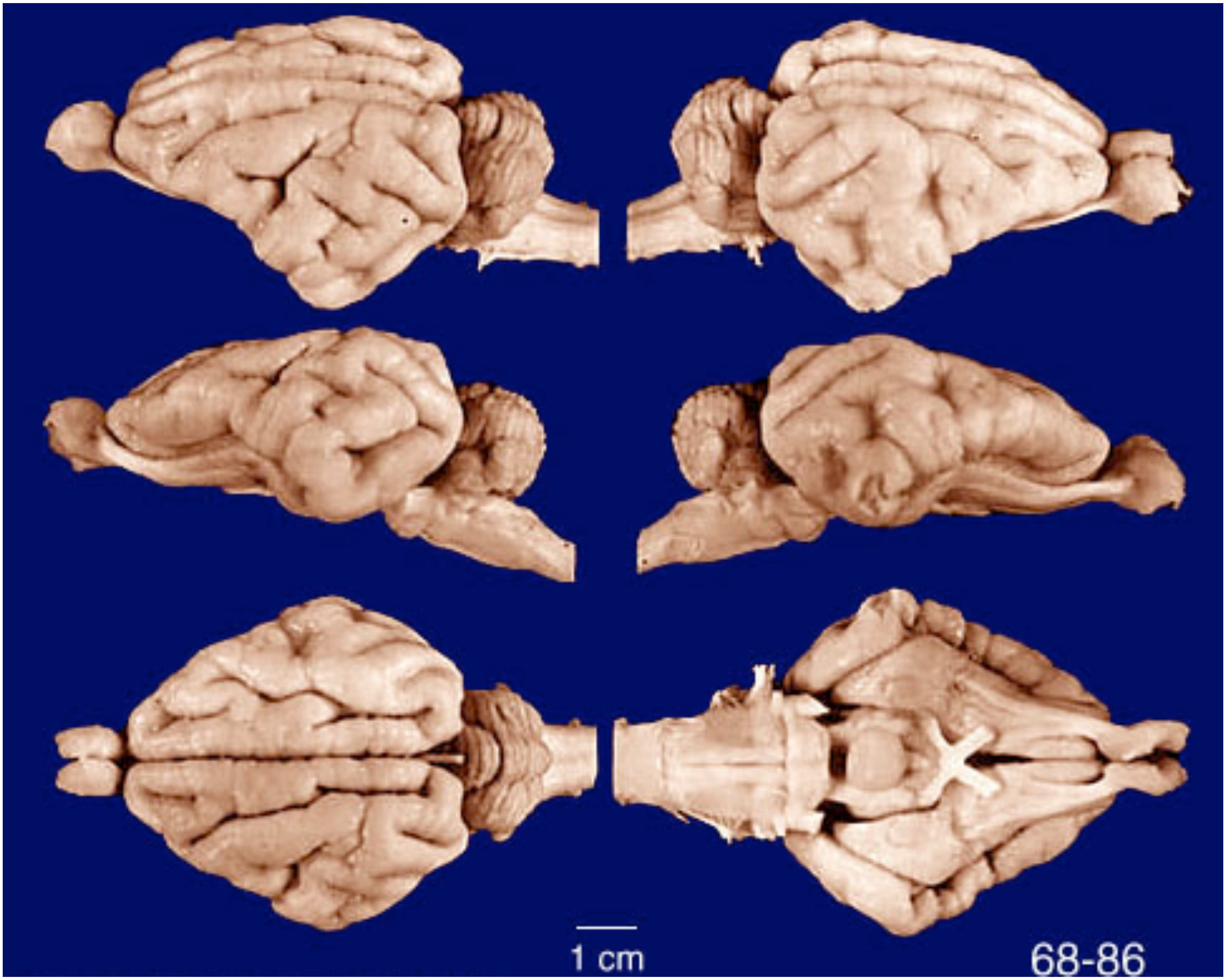


Numerical Simulation



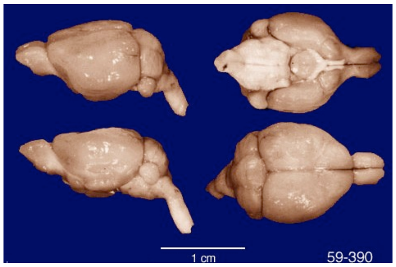
Capybara

(150 lb. guinea pig)



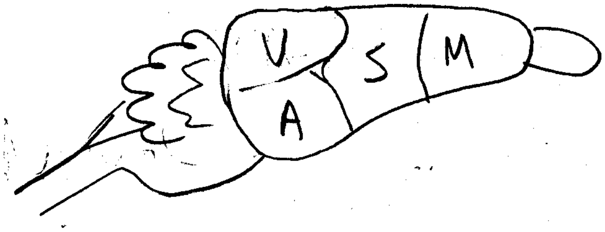
Lab Deer Mouse

(same scale)

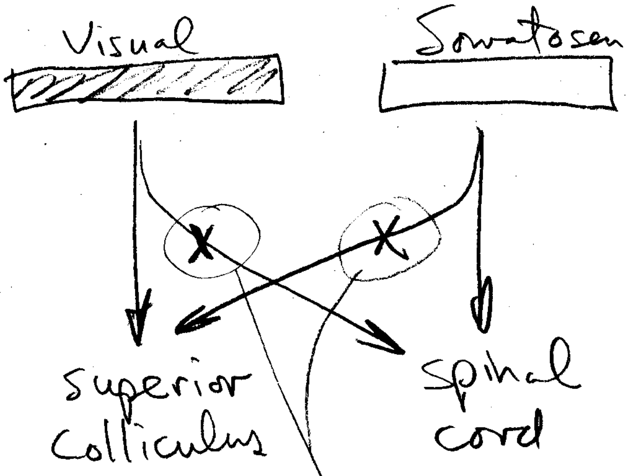


4) O'LEARY EXPT

also
- Shatz LGN Ramvitkn
- Cohen Tannoudji — gene
  \ expt

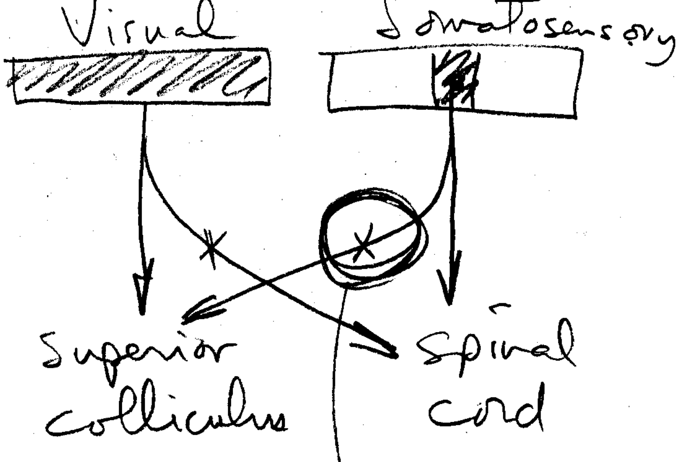


Normal

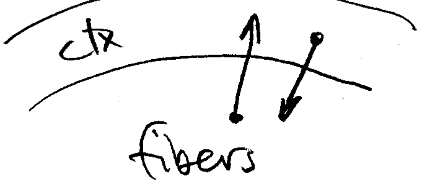


goes away later in development

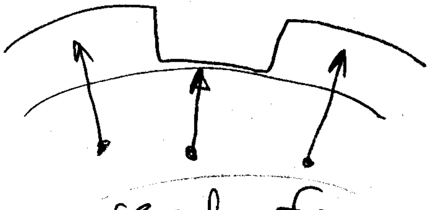
Transplant



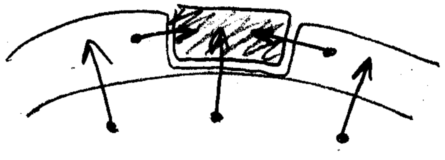
vis ctx is re-specified



intact cortex

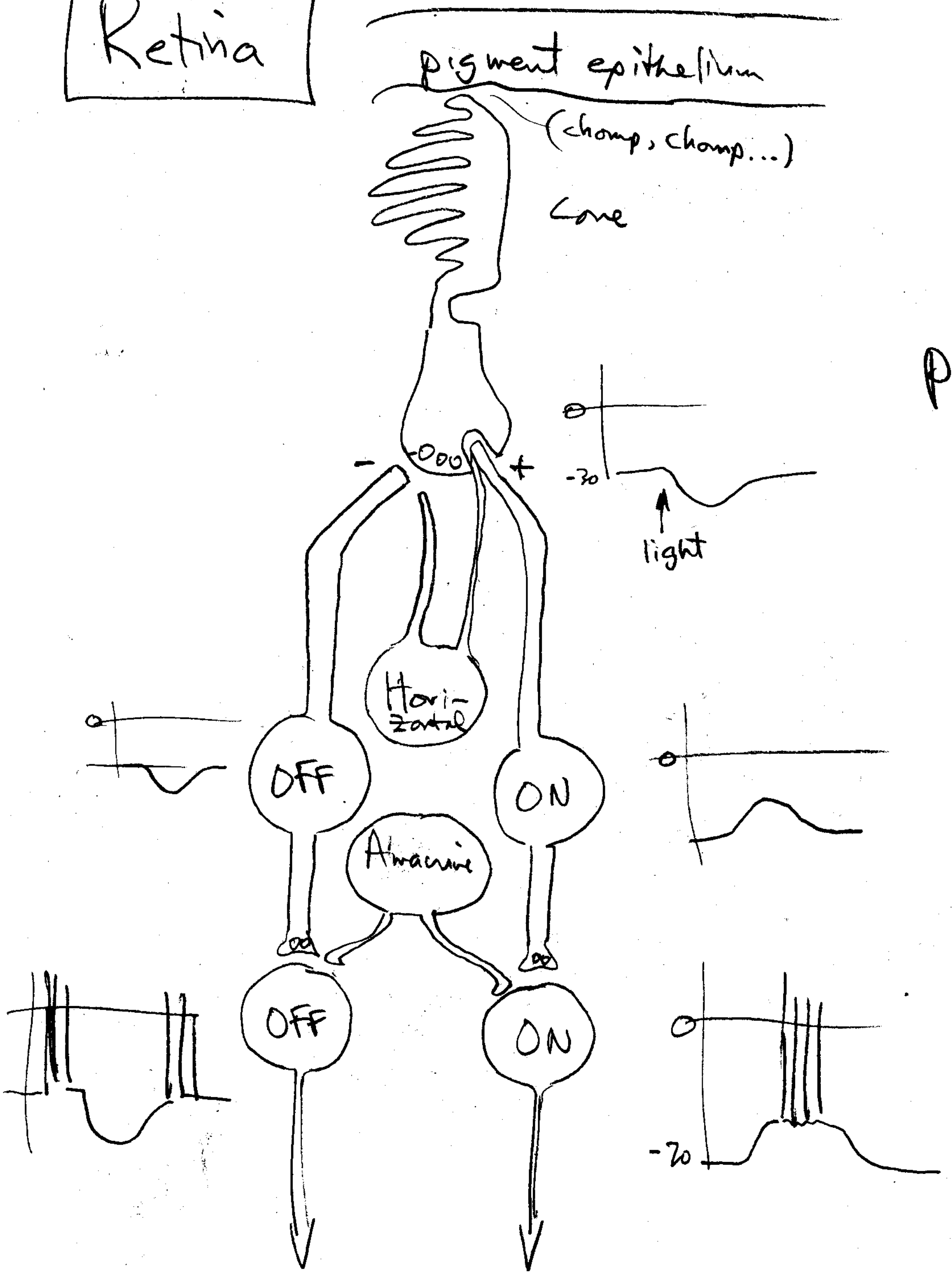


ready for transplant



fiber grow in an re-specifu

Retina



X - parvo
Y - magno
blue - konio

photoreceptors (4 types)

- dark current
- light closes channels → repol.
- repolarization → less neurotrans release

horizontal cells (2 types)

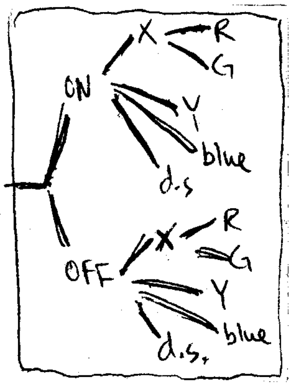
bipolar cells (3 types)

(ON, OFF, rod)

amacrine cells (20 types)

AII, A1-25...
starburst

ganglion cells (10 types)



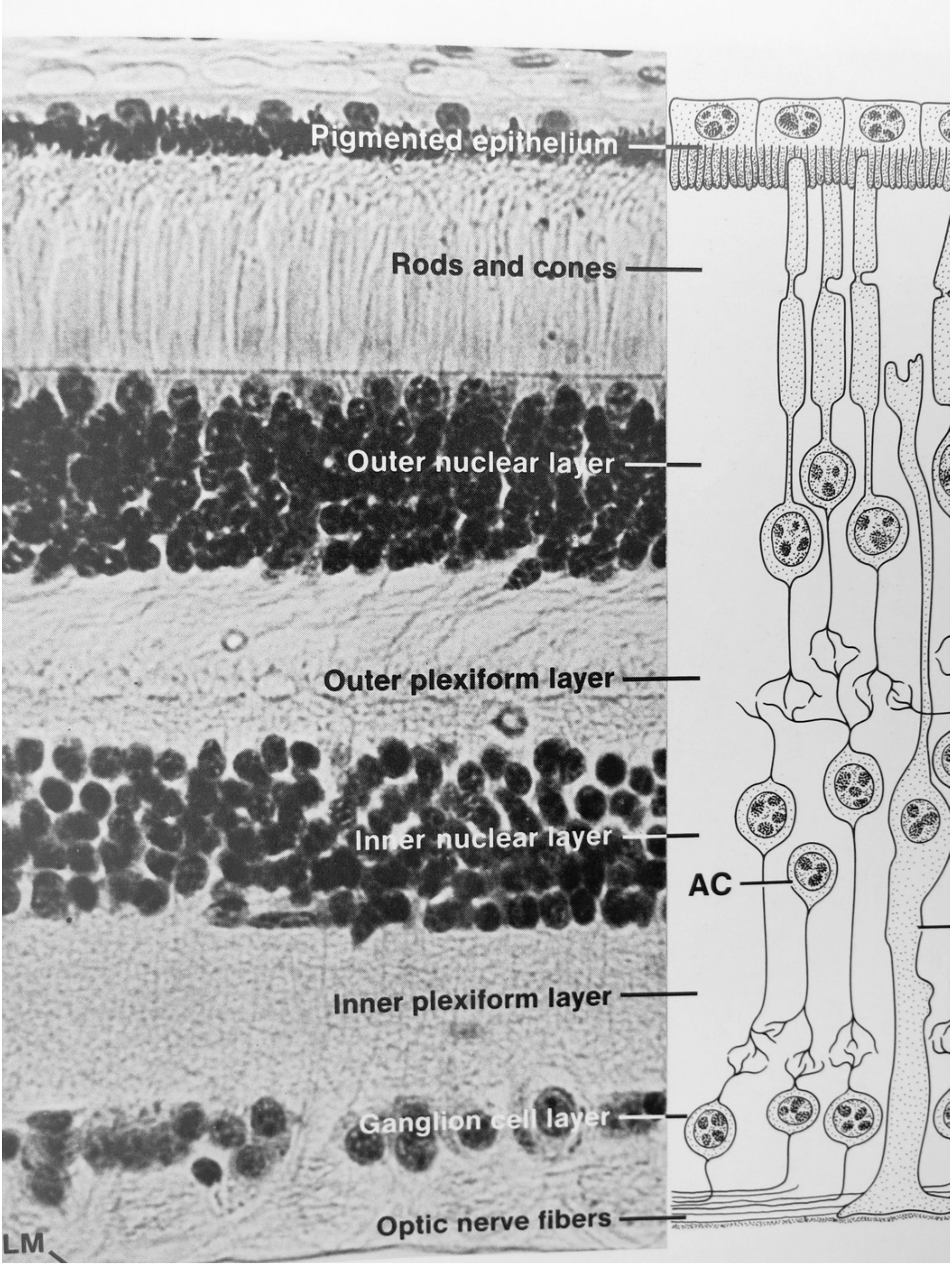
ganglion cell types

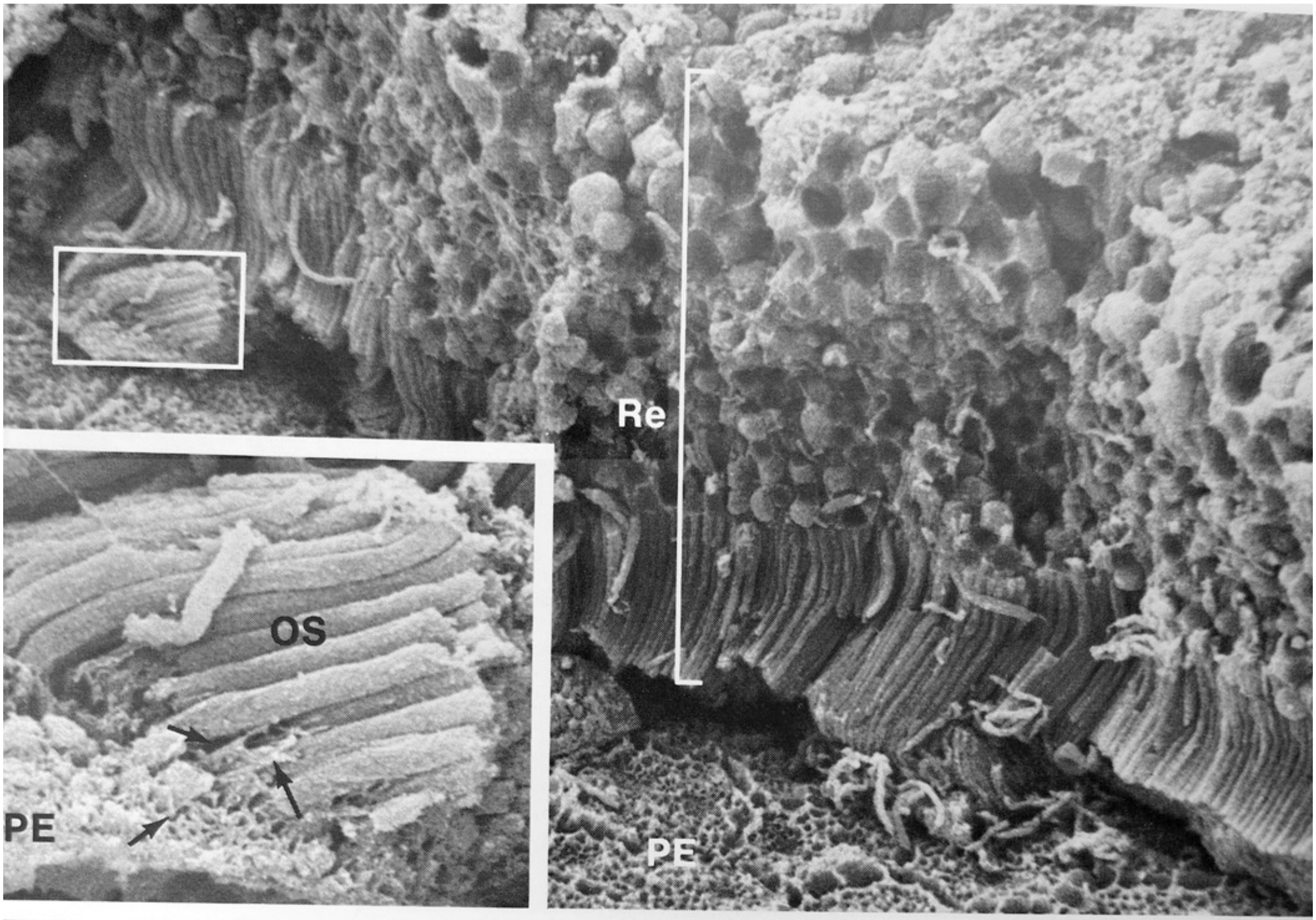
- (midget) X
  - (ON)
    - R cent
    - G cent
  - (OFF)
    - R cent
    - G cent
  - ⟹ LGN
- (parasol) Y
  - (ON)
  - (OFF)
  - ⟹ LGN, colliculus
- blue
  - ON
  - OFF
  - ⟹ LGN, colliculus
- W (cat) (ON/OFF) ⟹ LGN, colliculus
- direction selective ⟹ pretectum (MTN, LTN)
- thorn cells ⟹ colliculus

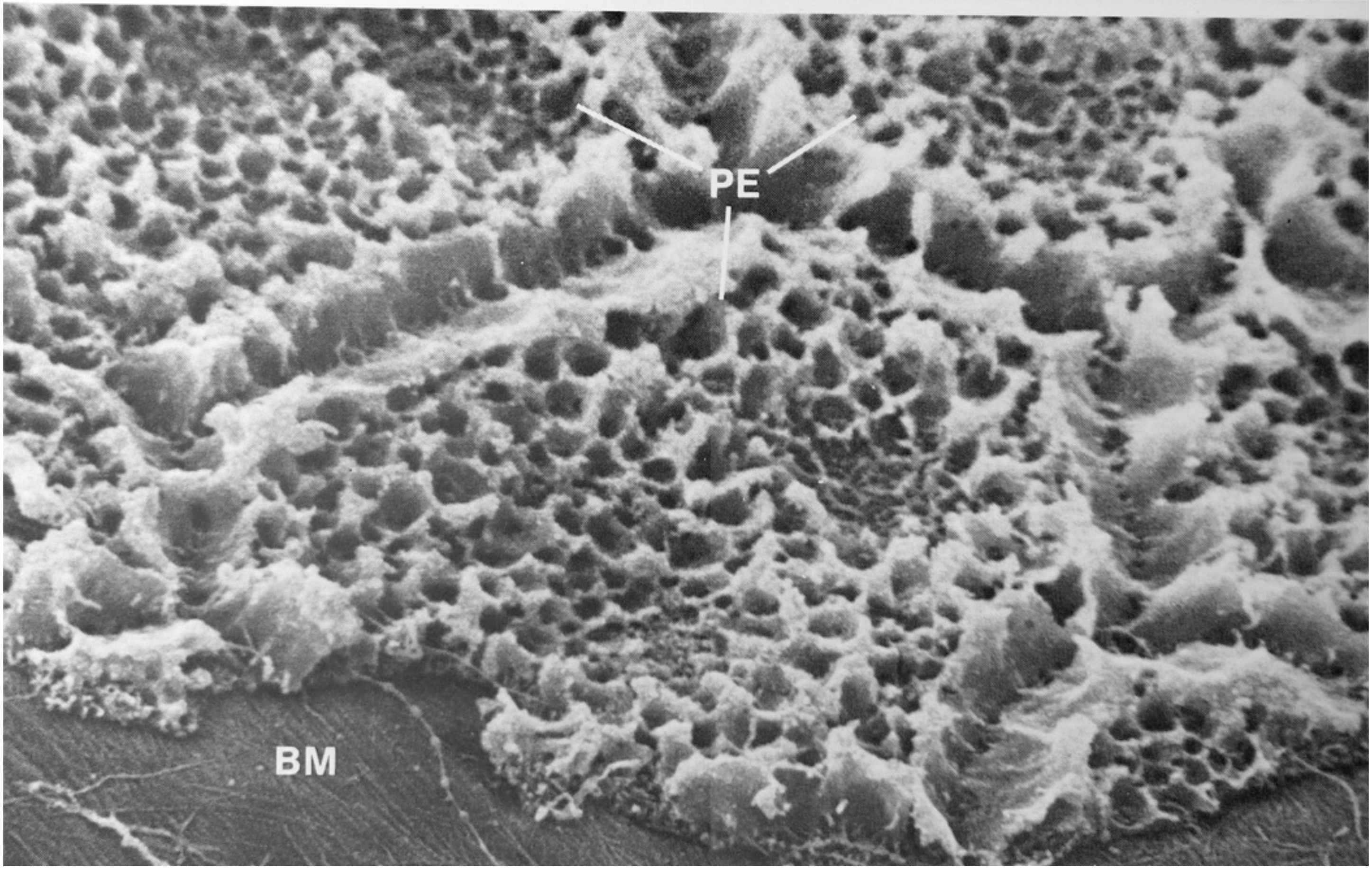
X
- sustained
- color selective
- smaller RF
- linear

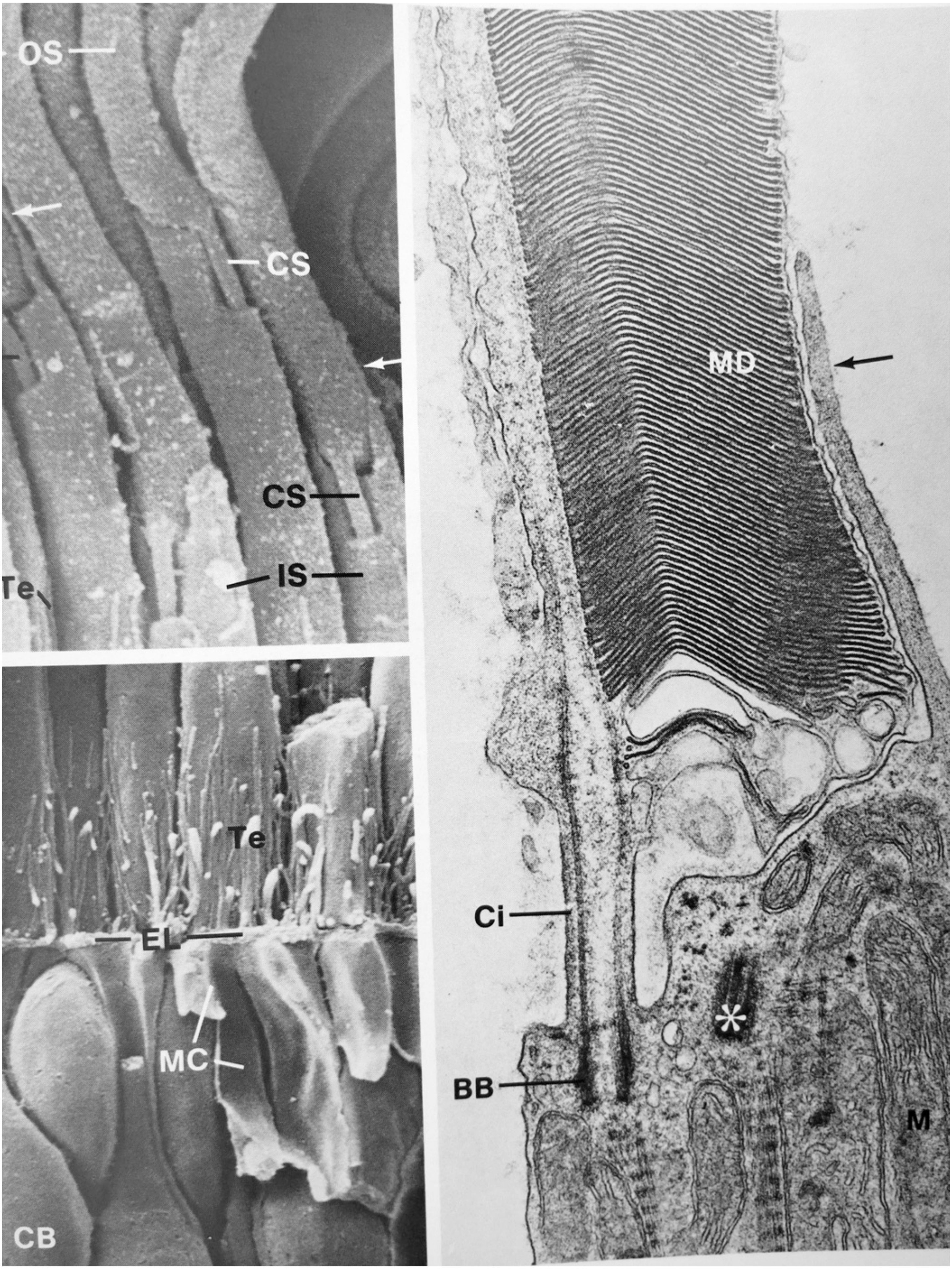
Y
- transient
- not color-selective
- larger RF
- non-linear

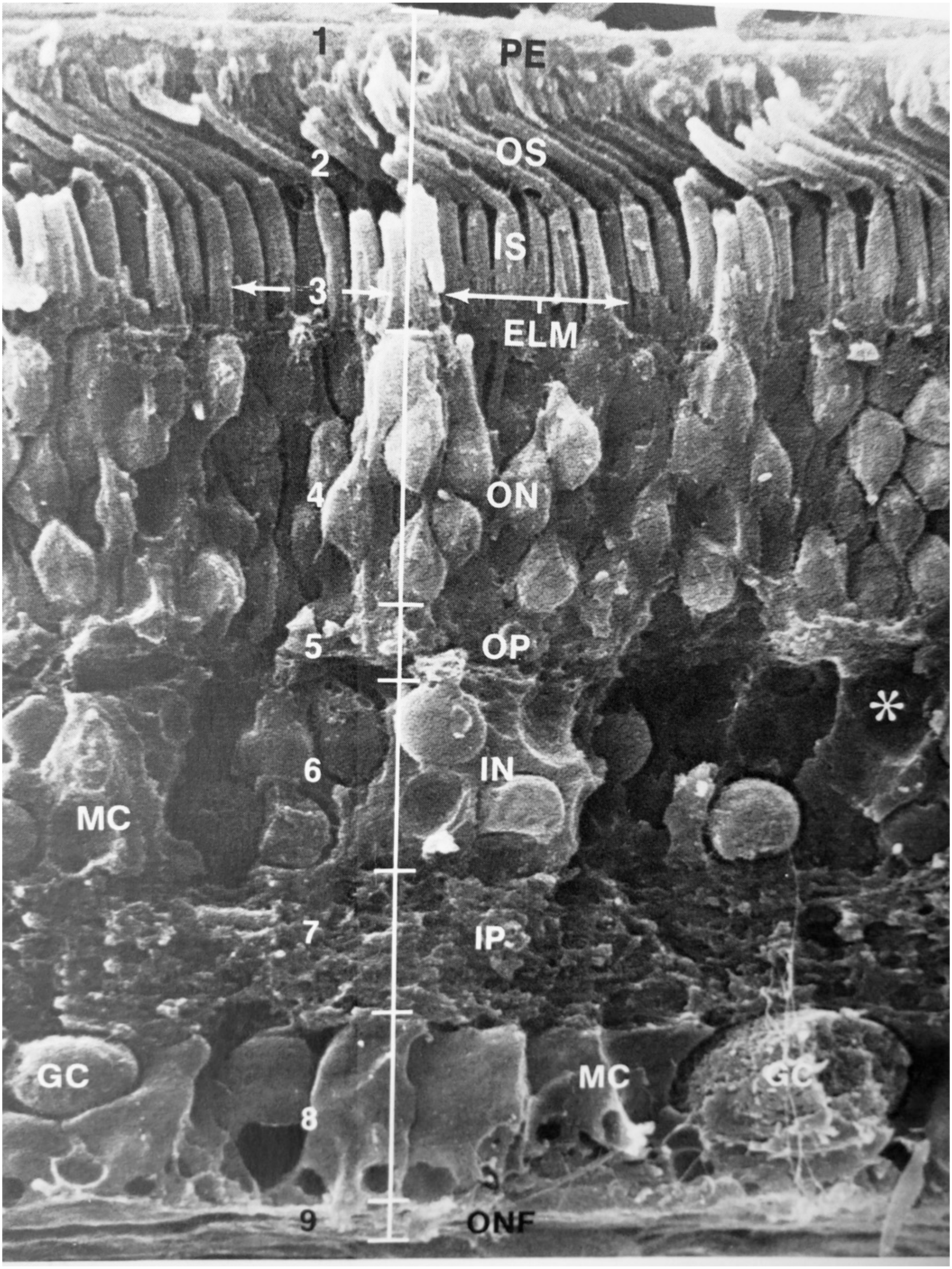
- cf. somatosens.


Pigmented epithelium
Rods and cones
Outer nuclear layer
Outer plexiform layer
Inner nuclear layer
AC
Inner plexiform layer
Ganglion cell layer
Optic nerve fibers
LM

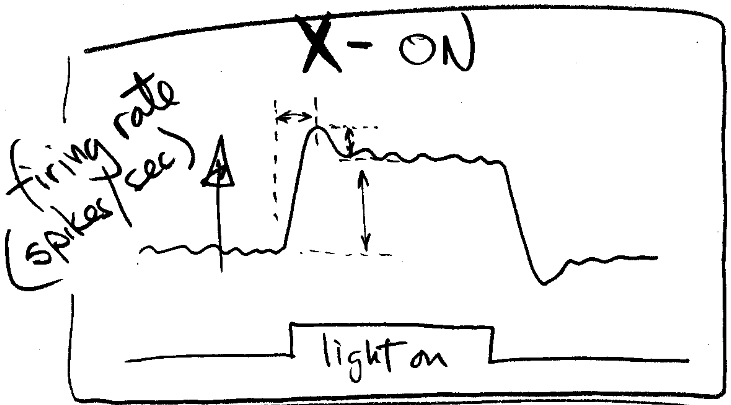

Re
OS
PE
PE

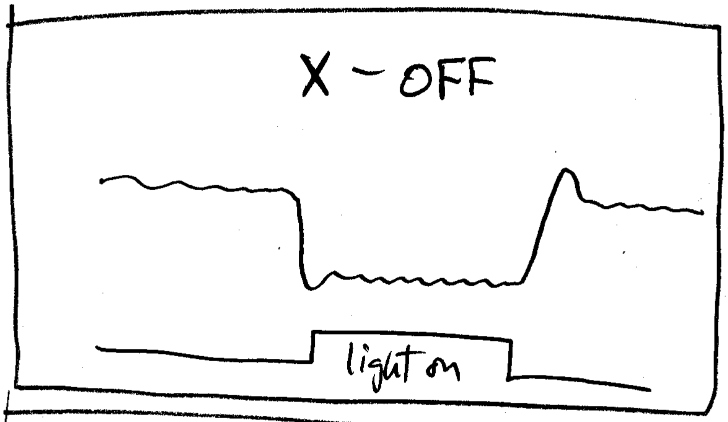

PE
BM

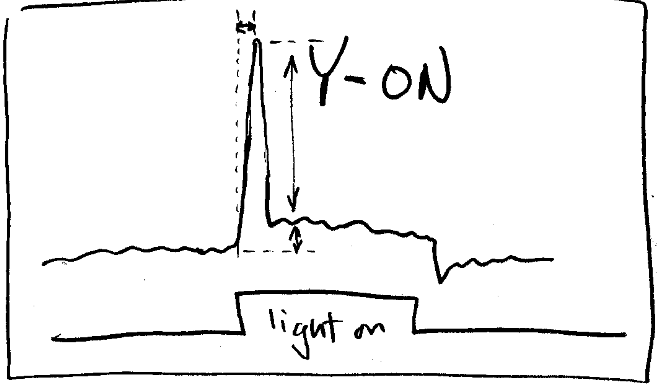

OS
CS
CS
IS
Te
Te
EL
MC
CB
MD
Ci
BB
*
M

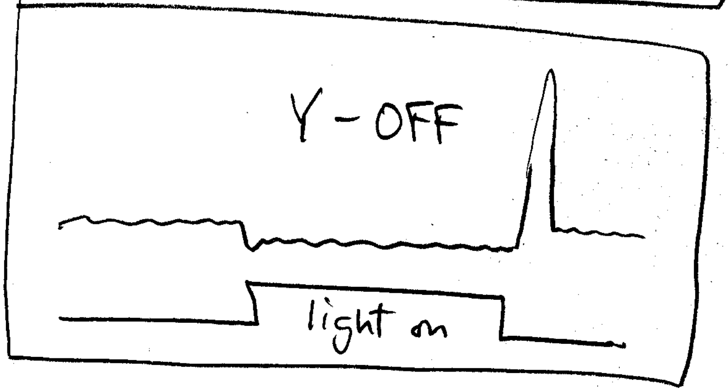

1
PE
2
OS
IS
3
ELM
4
ON
5
OP
*
6
IN
MC
7
IP
GC
MC
GC
8
9
ONF

Ganglion Cell Types









- this is like <u>brightness</u> & <u>derivative of brightness</u>

$f(x) =$ 

$f'(x) =$ 

↳ typical cells have both, but emphasize one

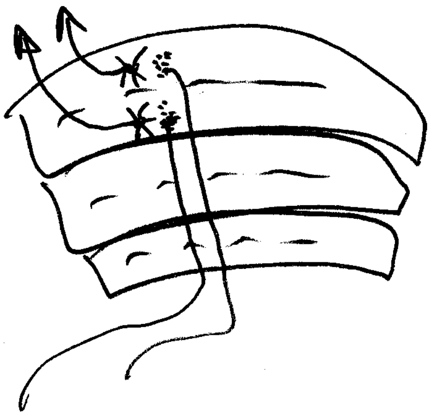
Other features of ganglion cells

- colliculus cell types.

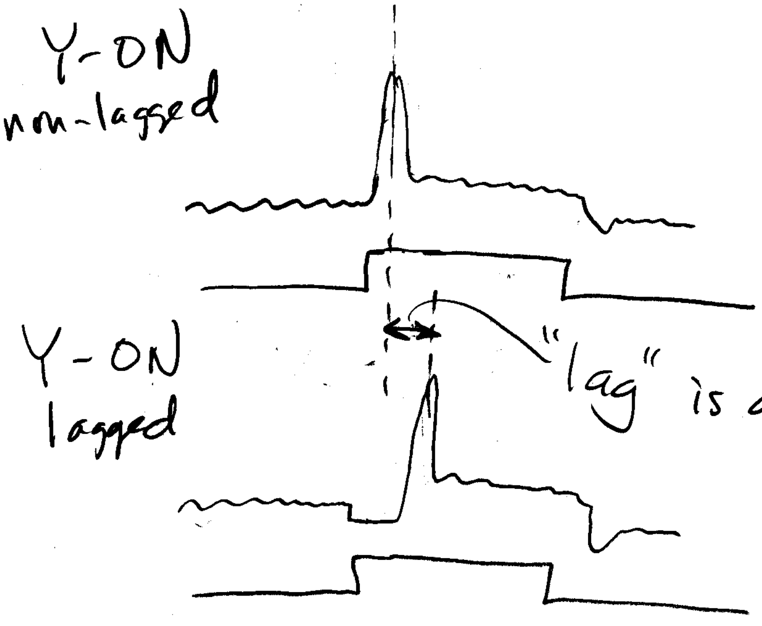
- red center / green surround
  green center / red surround
  blue
  luminance (Y cells — red vs. green equal if brightness

dLGN additions (not too many)

- cells are still monocular in dLGN despite being lined up

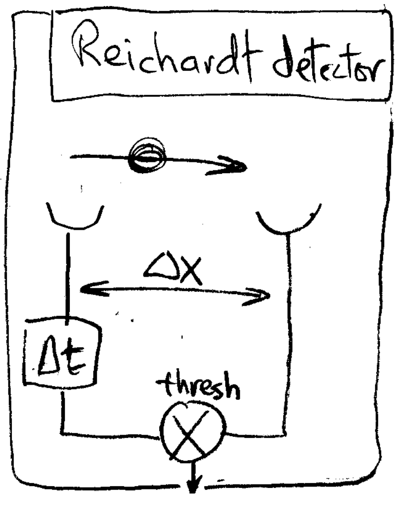


- non-lagged vs. lagged (cat dLGN)

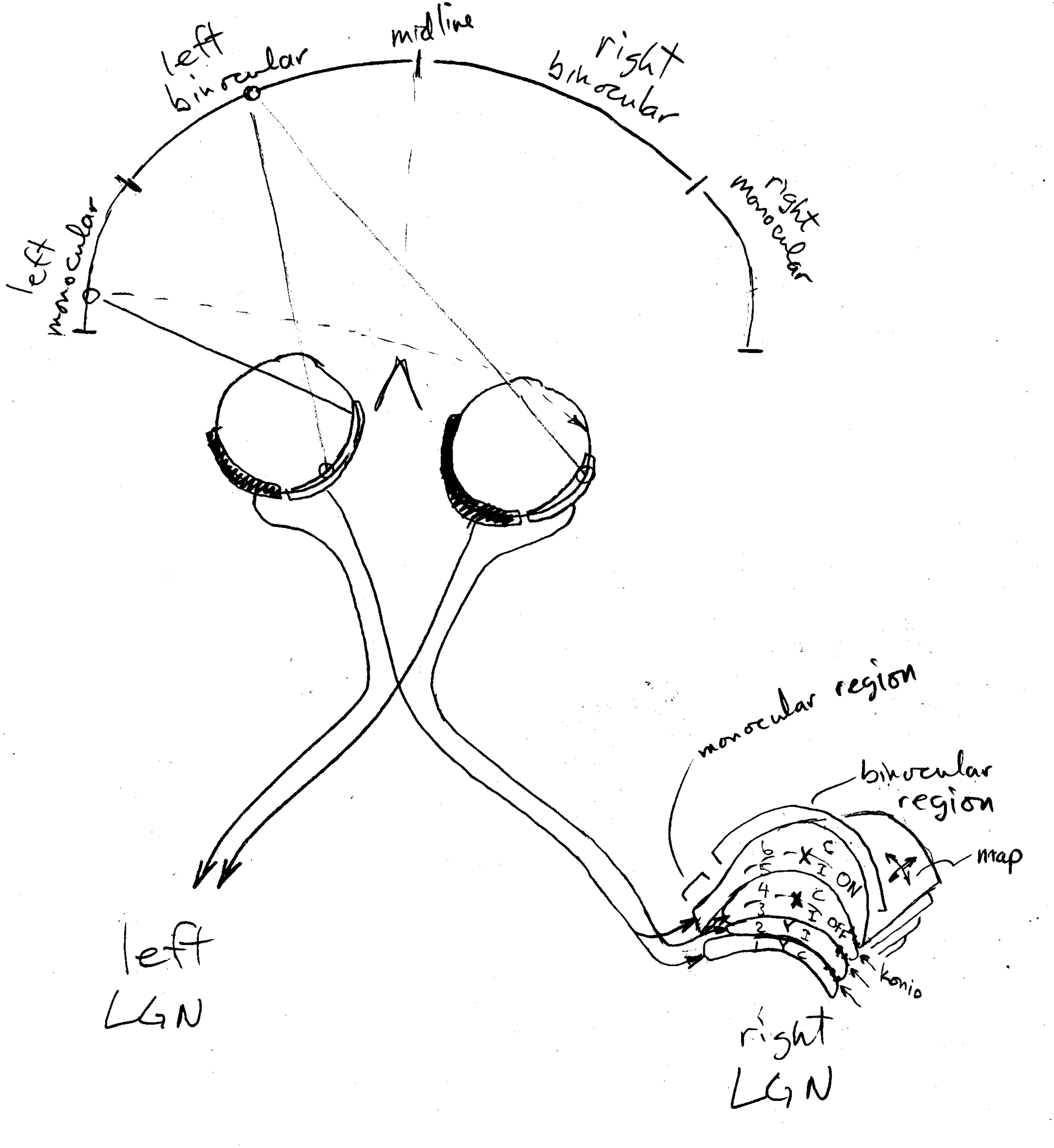


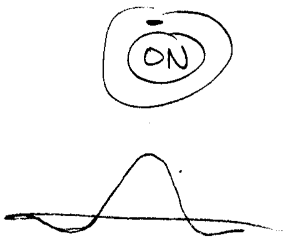
"lag" is about 50 msec

N.B.: in primates, this operation is postponed until the cortex (V1) 4Cα → 4B

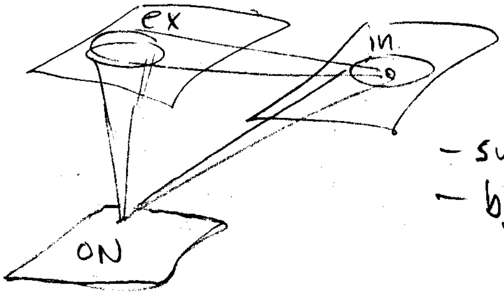


Retino geniculate

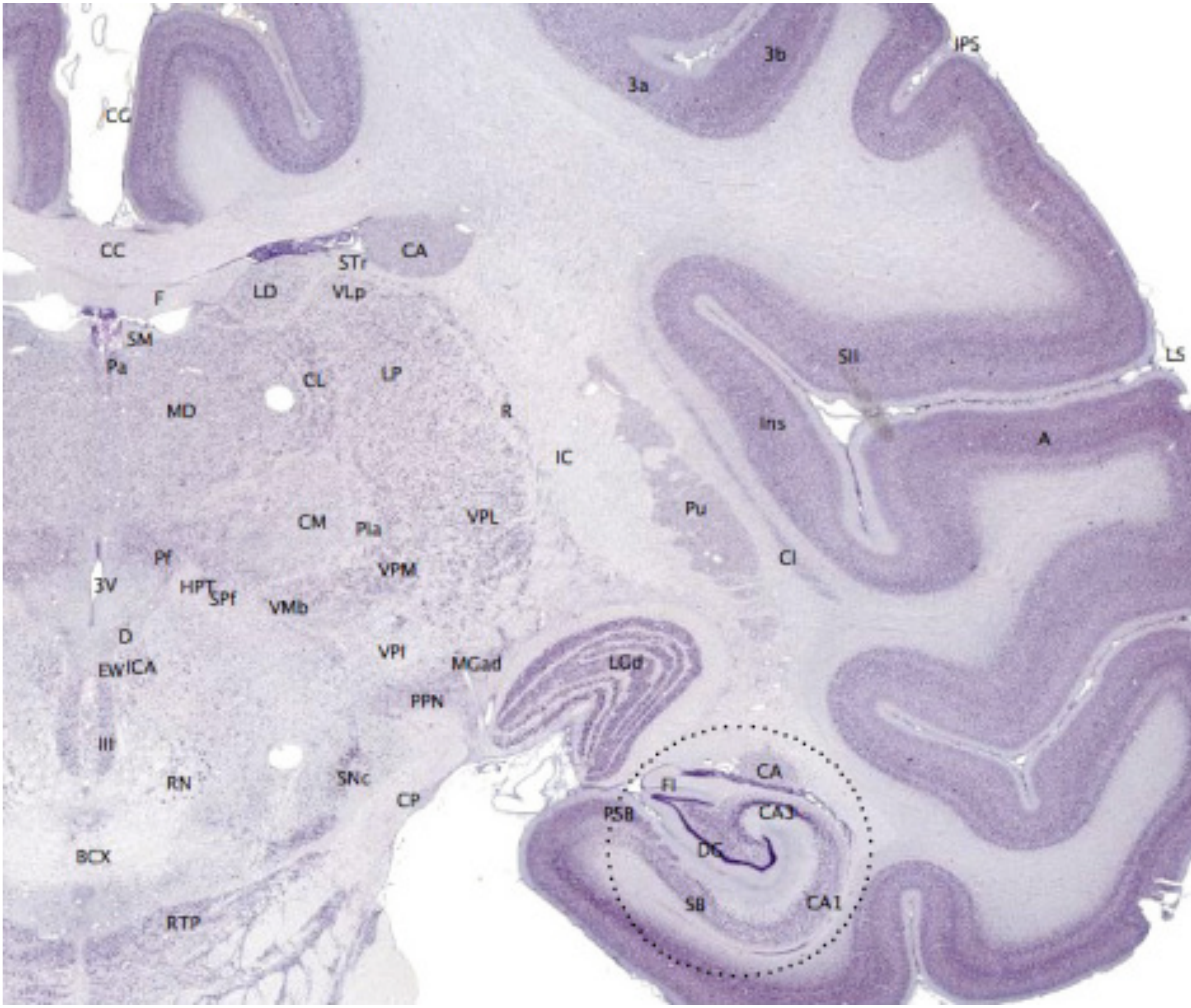




e.g.



- surround is inhib (not OFF)
- bigger because feedforward inhib. is disynaptic


CC
3a
3b
IPS
CC
CA
STr
LD
VLp
F
SM
Pa
CL
LP
MD
R
SII
LS
Ins
A
IC
Pu
CM
Pla
VPL
Pf
3V
HPT
SPf
VPM
VMb
Cl
D
EW
ICA
VPI
MGad
LGd
PPN
III
RN
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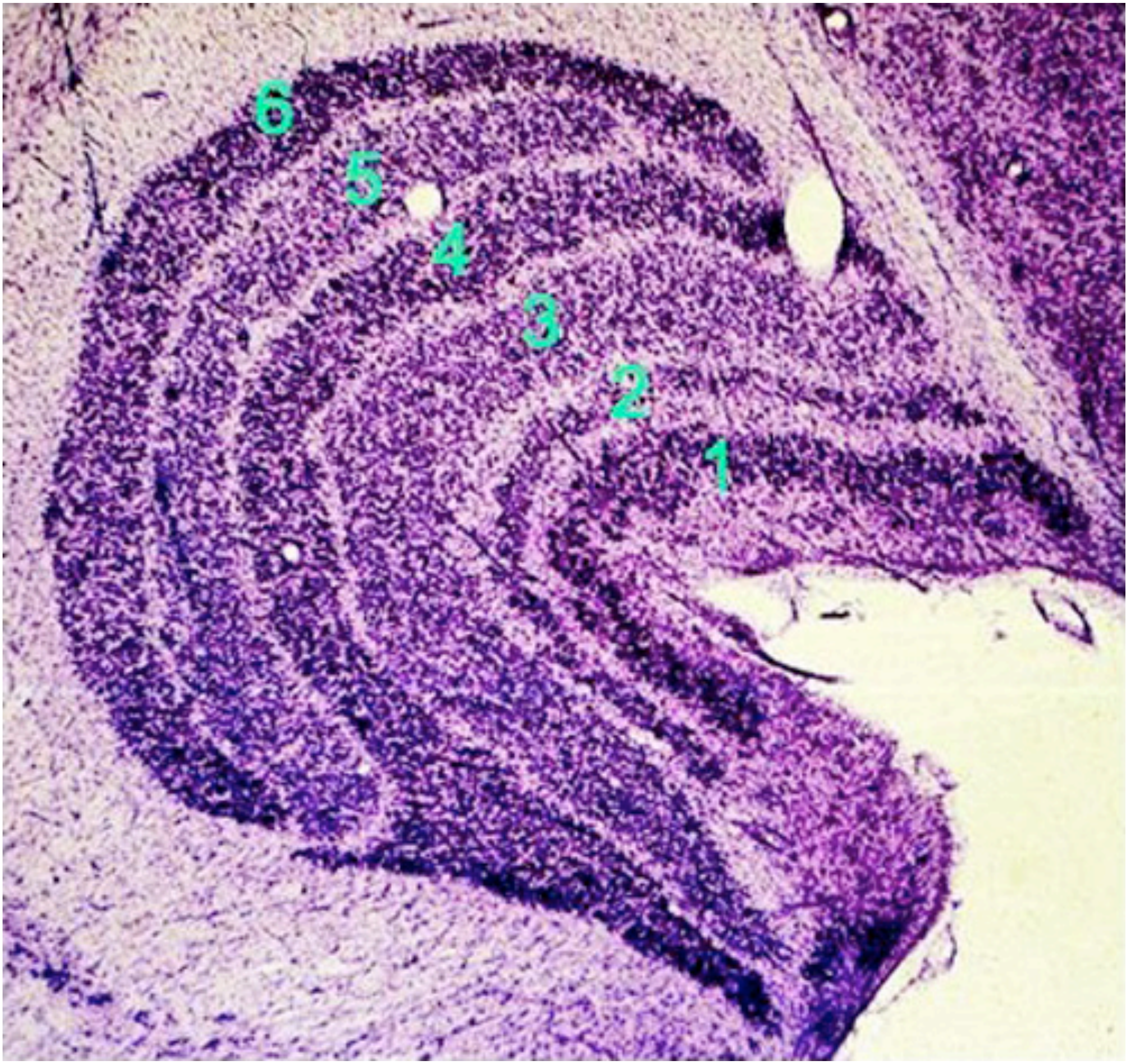
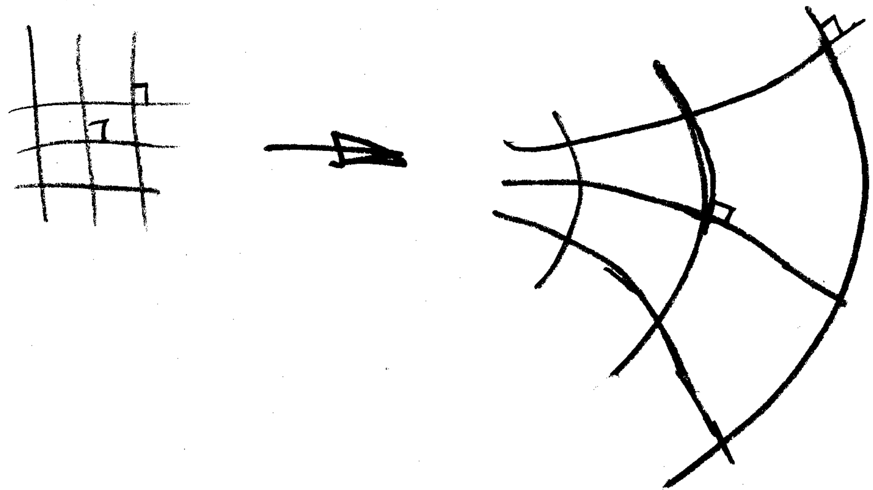
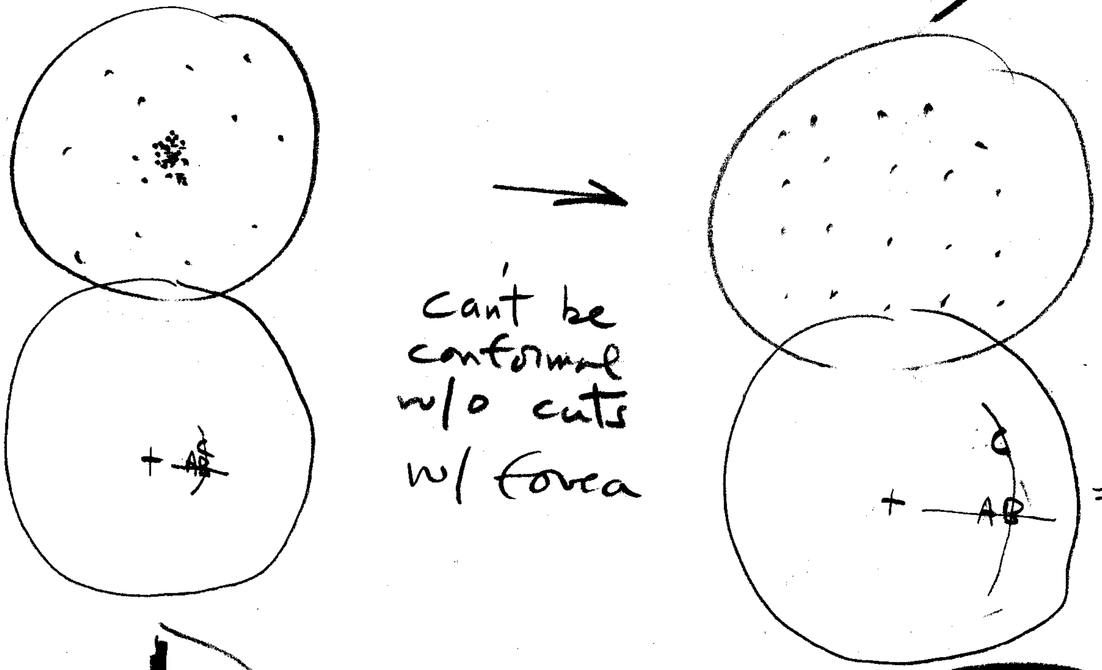
FI
CP
PSB
CA3
DG
BCX
SB
CA1
RTP


6
5
4
3
2
1

Maps

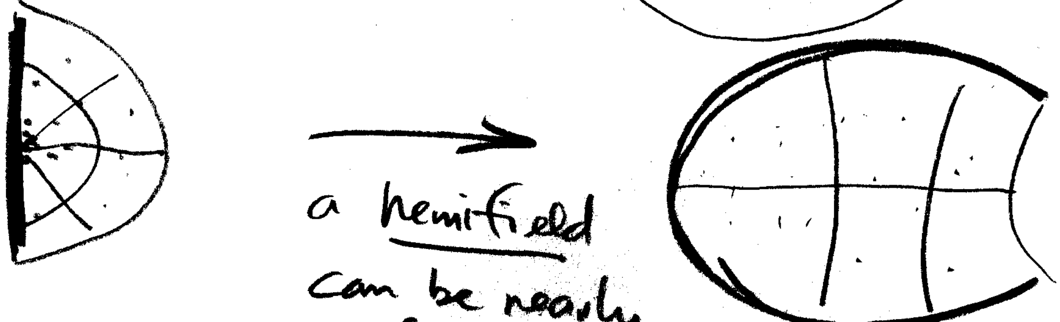
Conformal Maps — angle preserving



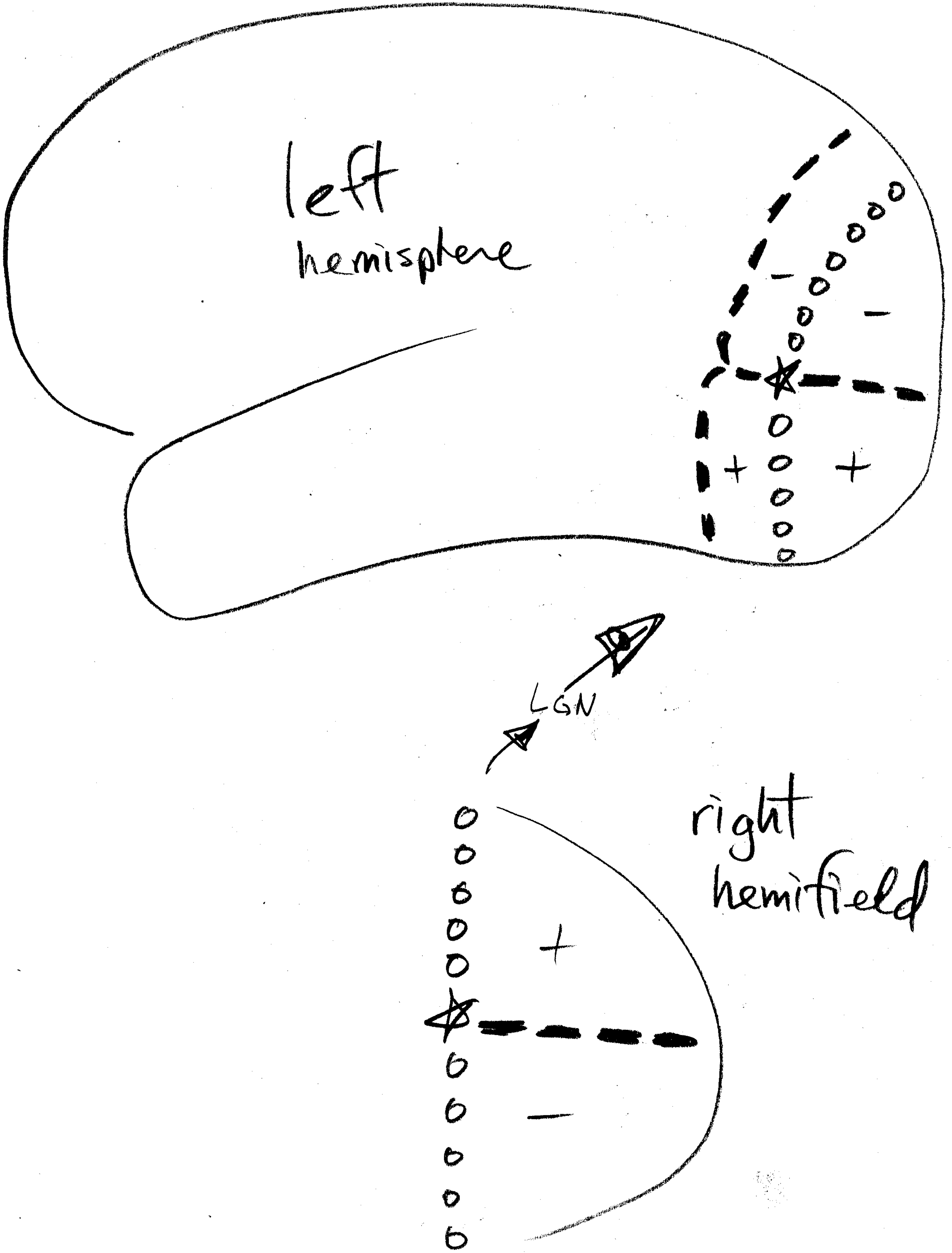


can't be conformal w/o cuts w/ fovea

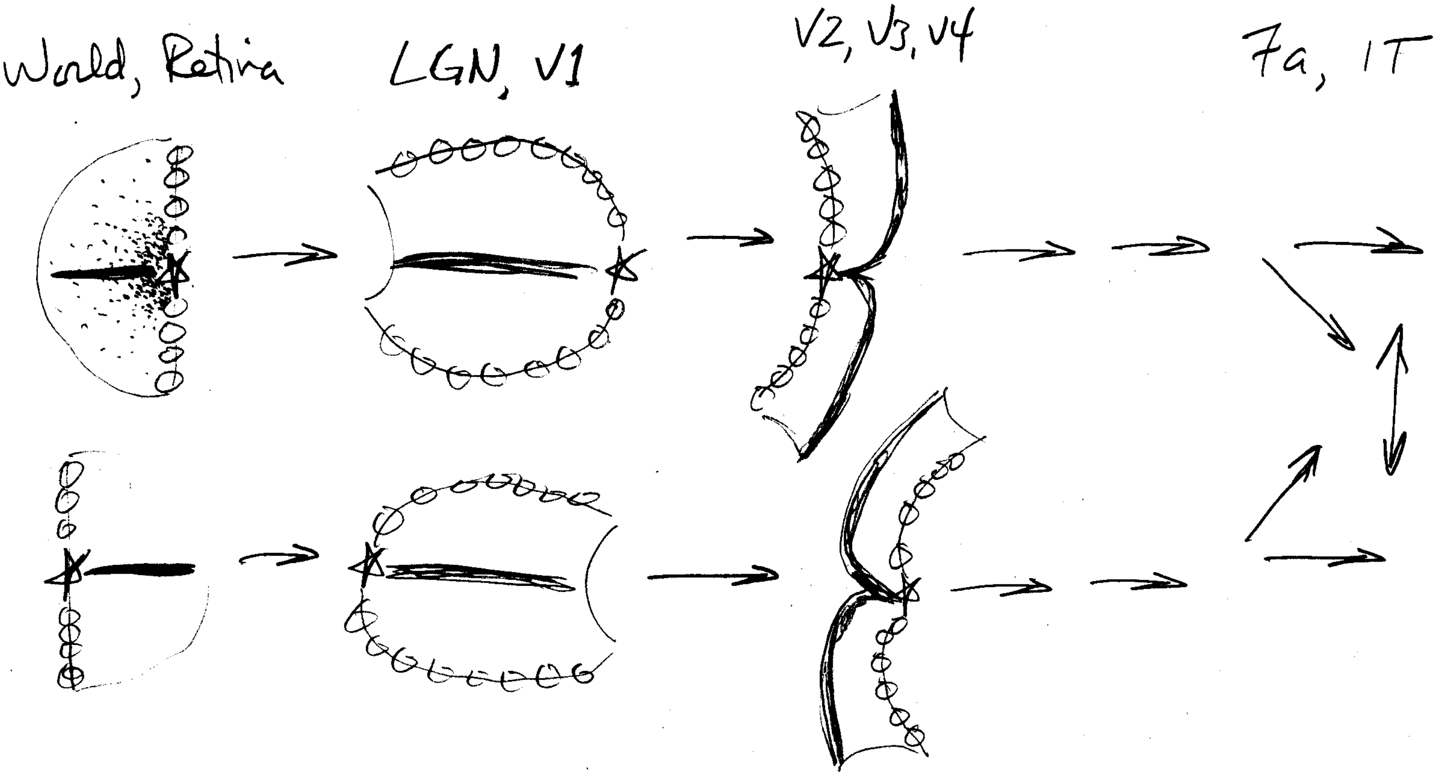
⇒ circumferential stretching greater than radial stretching



a hemifield can be nearly conformal


left
hemisphere
−
−
+
+
LGN
right
hemifield
+
−

Maps

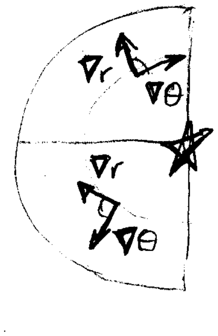


Mirror-image vs Non-Mirror Image

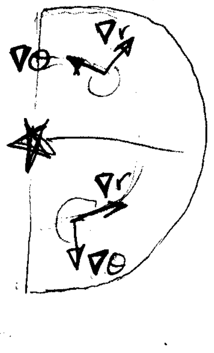
Map: Retinal Position (r, θ) $\Rightarrow$ Cortical Position (x, y)

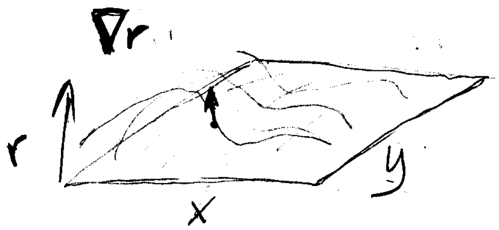
like $r = f(c)$
but r, c both have
two dimensions

non-mirror image cortical map of left hemifield

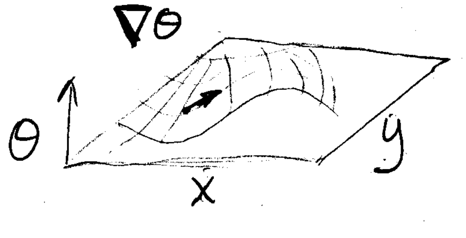


mirror image cortical map of left hemifield





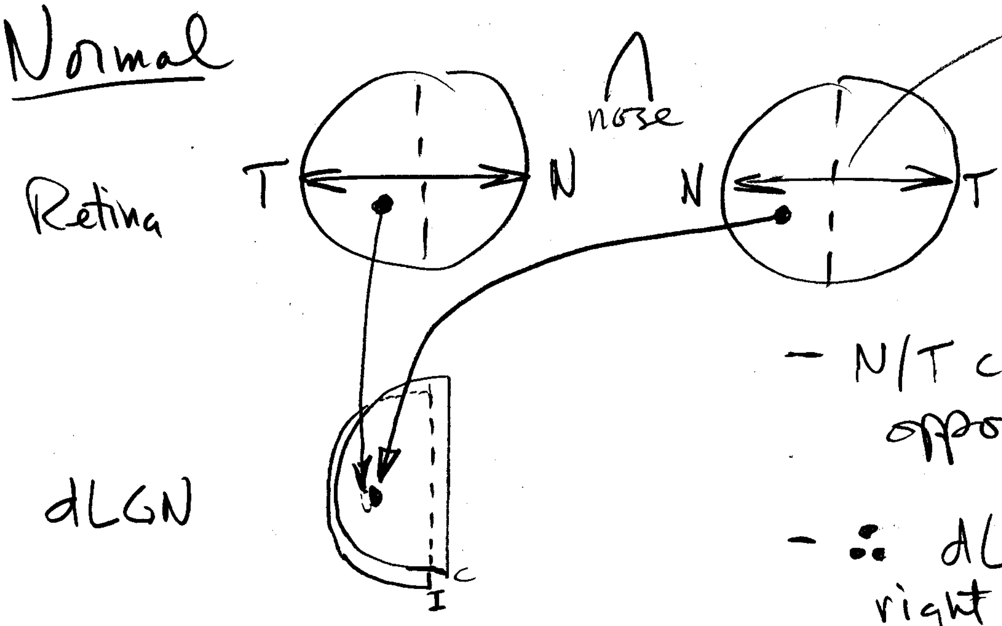
- steepest uphill direction of **r** at each **x,y**



- steepest uphill direction of **θ** at each **x,y**

Mutant Belgian Sheepdog

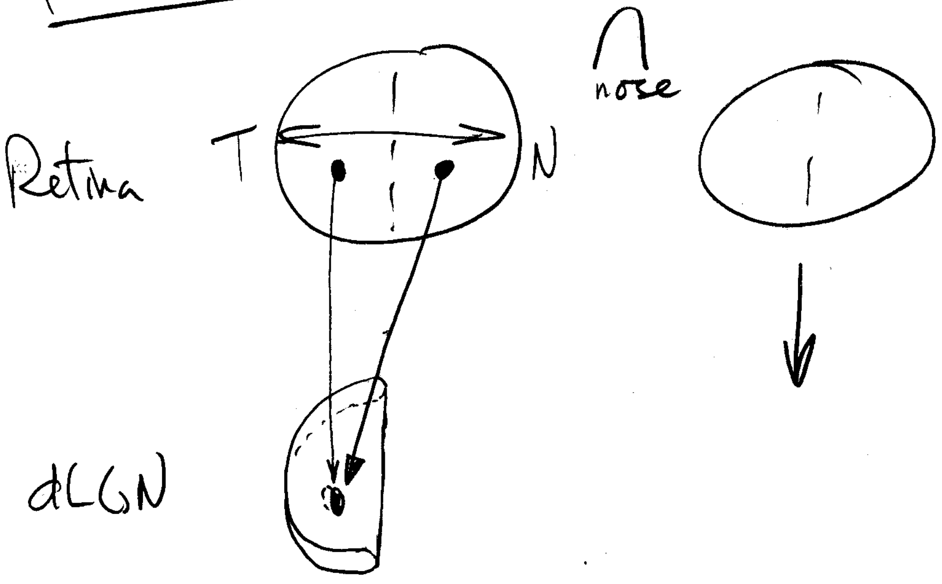
Normal



nasotemporal coordinate in retina

- N/T coordinate has opposite "sign" in two eye
- ∴ dLGN aligns left & right retinal points w/ <u>different</u> N/T coordinates in C & I layers

Mutant



- retina still goes to same N/T coordinate
- lack of chiasm then leads to a "taco"
- ∴ map generated by labels not activity

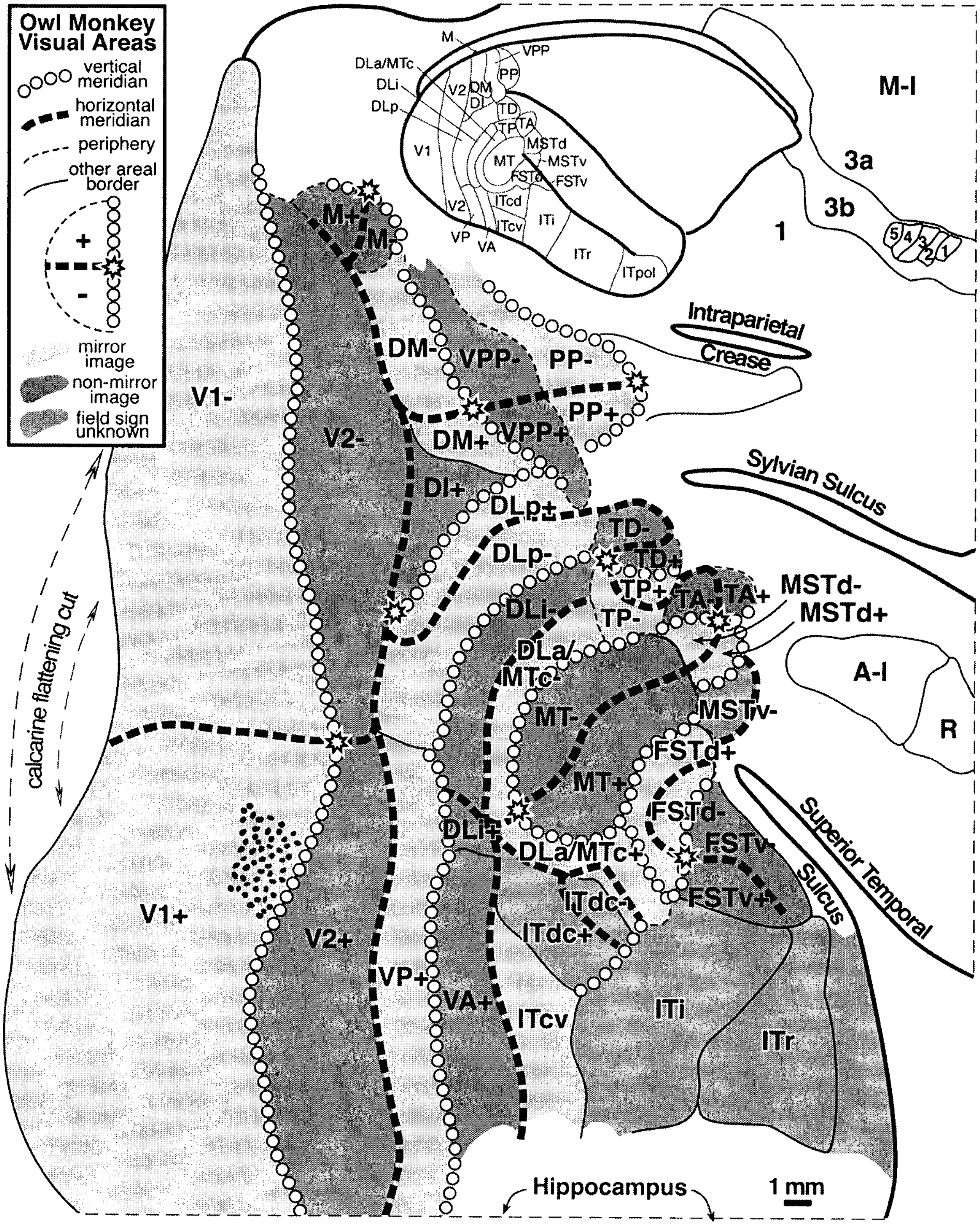
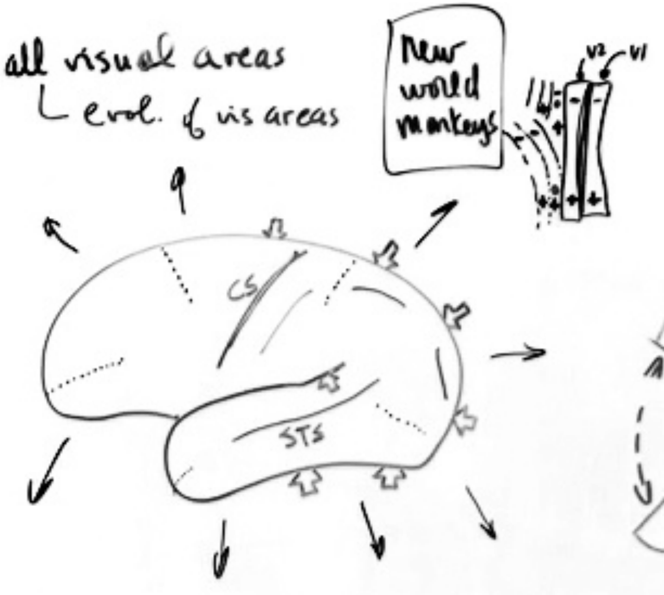
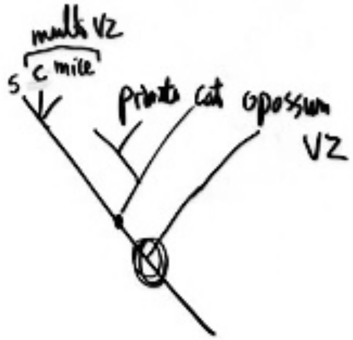
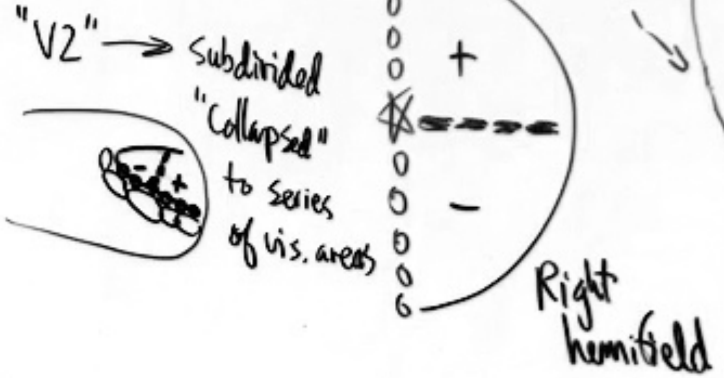


Fig. 2. Schematic proposal for the retinotopic organization of 24 owl monkey visual cortical areas drawn using a myelin-stained flat-mount. Anterior to V2 near the dorsal convexity of the brain are 3 areas with alternating field sign—DM, VPP, and PP. DM and VPP share a center of gaze and vertical meridian representation. VPP and PP share a periphery representation. The upper fields of DM and VPP curve anteriorly, away from V2. Just lateral to DM, there is another series of strip like areas with alternating field sign—DI (which unlike DM, has the same field sign as V2), DLp, DLi, DLa/MTc, and finally MT. DLa/MTc bends laterally away from MT along a shared vertical meridian border with FSTd. The complex topography anterior to MT is best visualized as two pairs of areas—TD/TP and TA/MSTd. Each pair of areas shares a center of gaze and a vertical meridian representation. In all four areas, the upper field is anterior to the lower field. Anterior and lateral to MT are FSTd and FSTv, which share a center of gaze and a vertical meridian. ITcd contains mainly an upper field representation lateral to DLa/MTc. Retinotopy of M, ventral VP and VA, and ITi, and ITr were taken from Allman and Kaas (1975), Newsome and Allman (1980), and Weller and Kaas (1987).

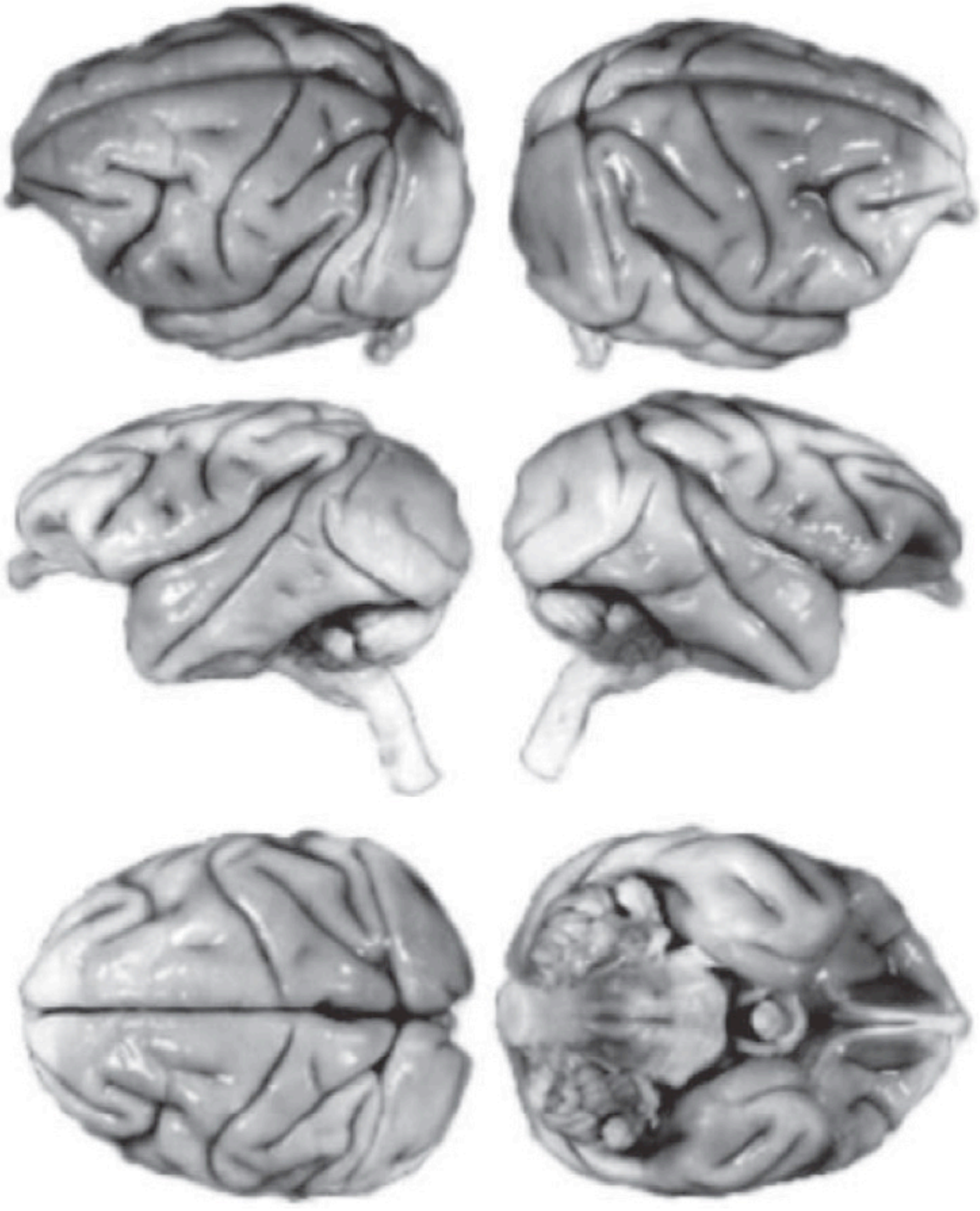
- all visual areas
  - evol. of vis areas

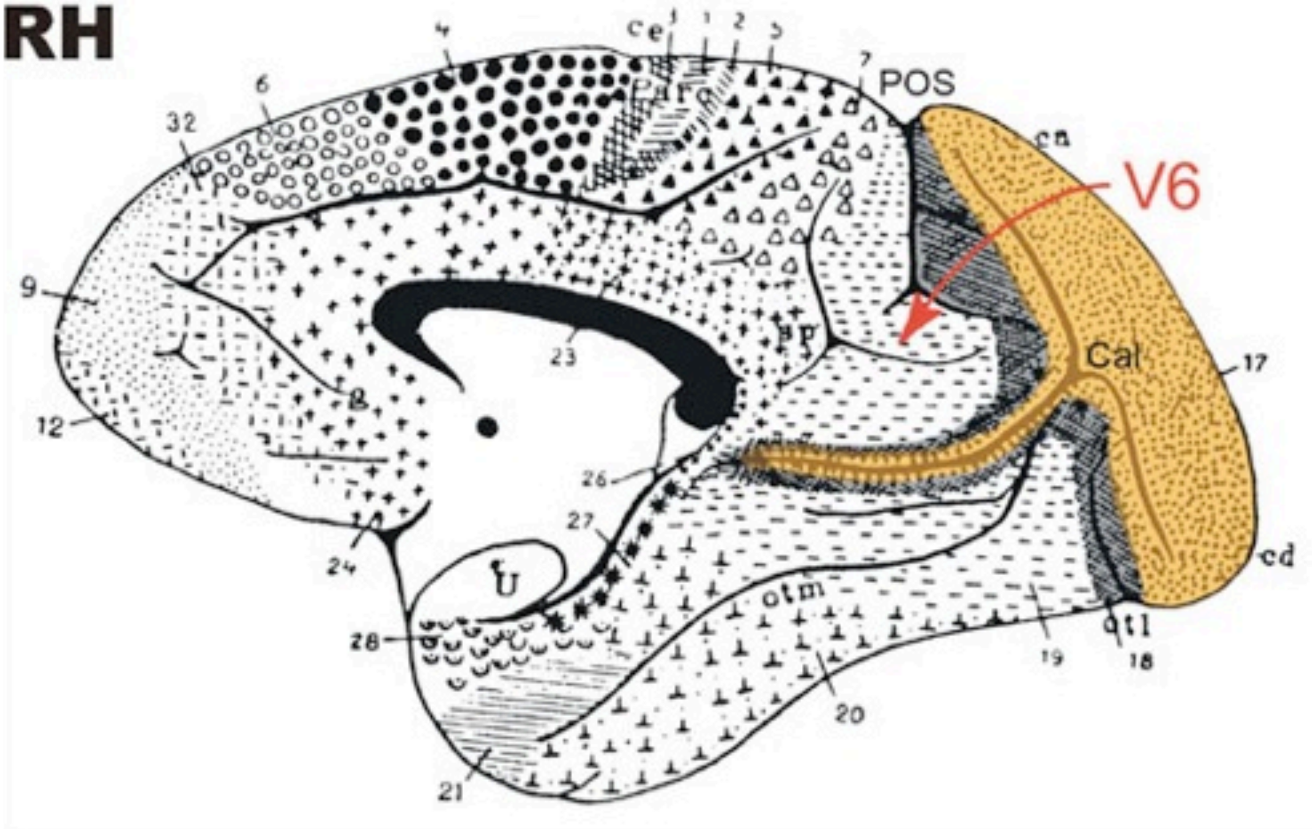






MTc
DLa
V4t




RH
ce
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POS
cn
V6
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pp
Cal
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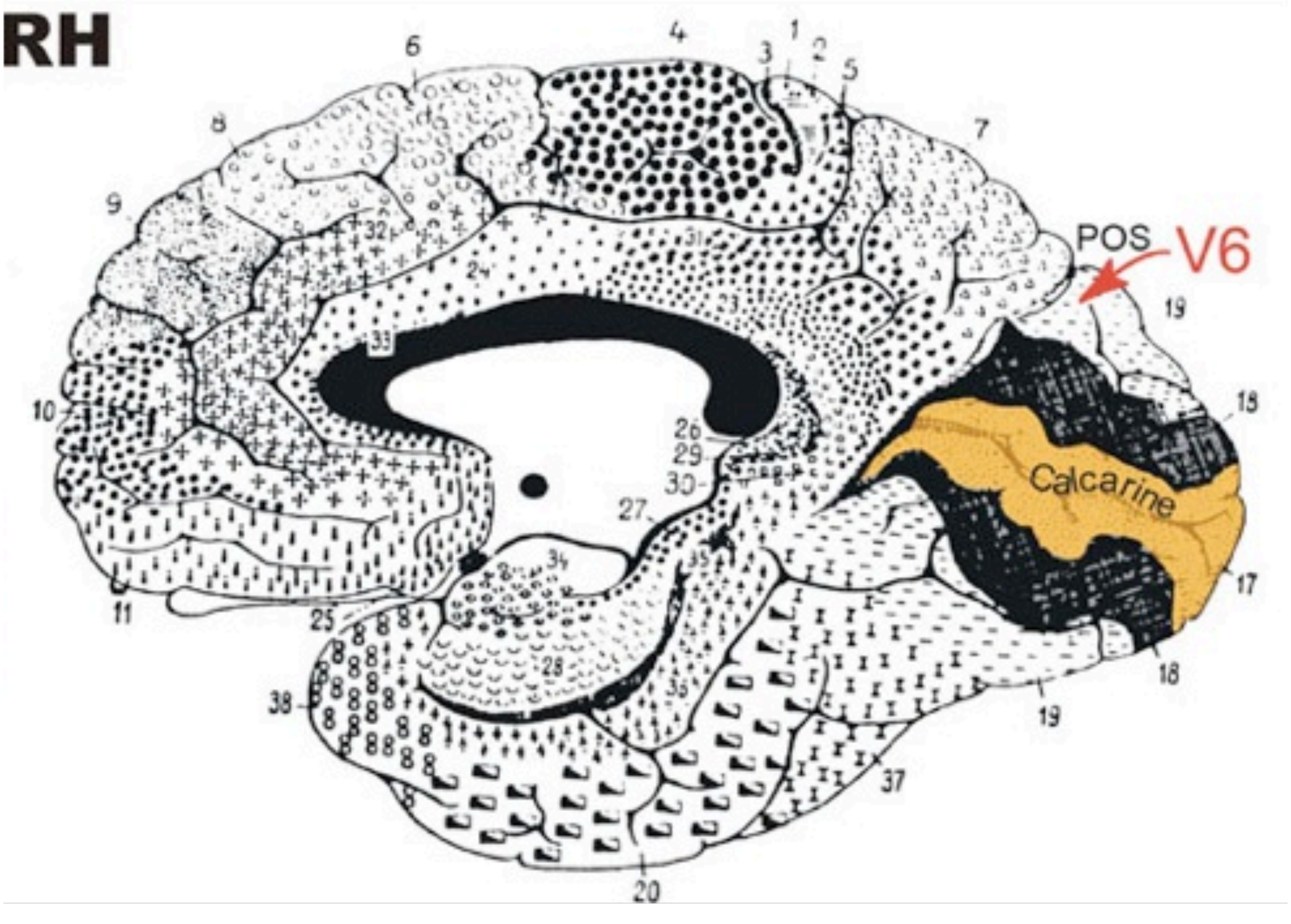
21


RH
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POS
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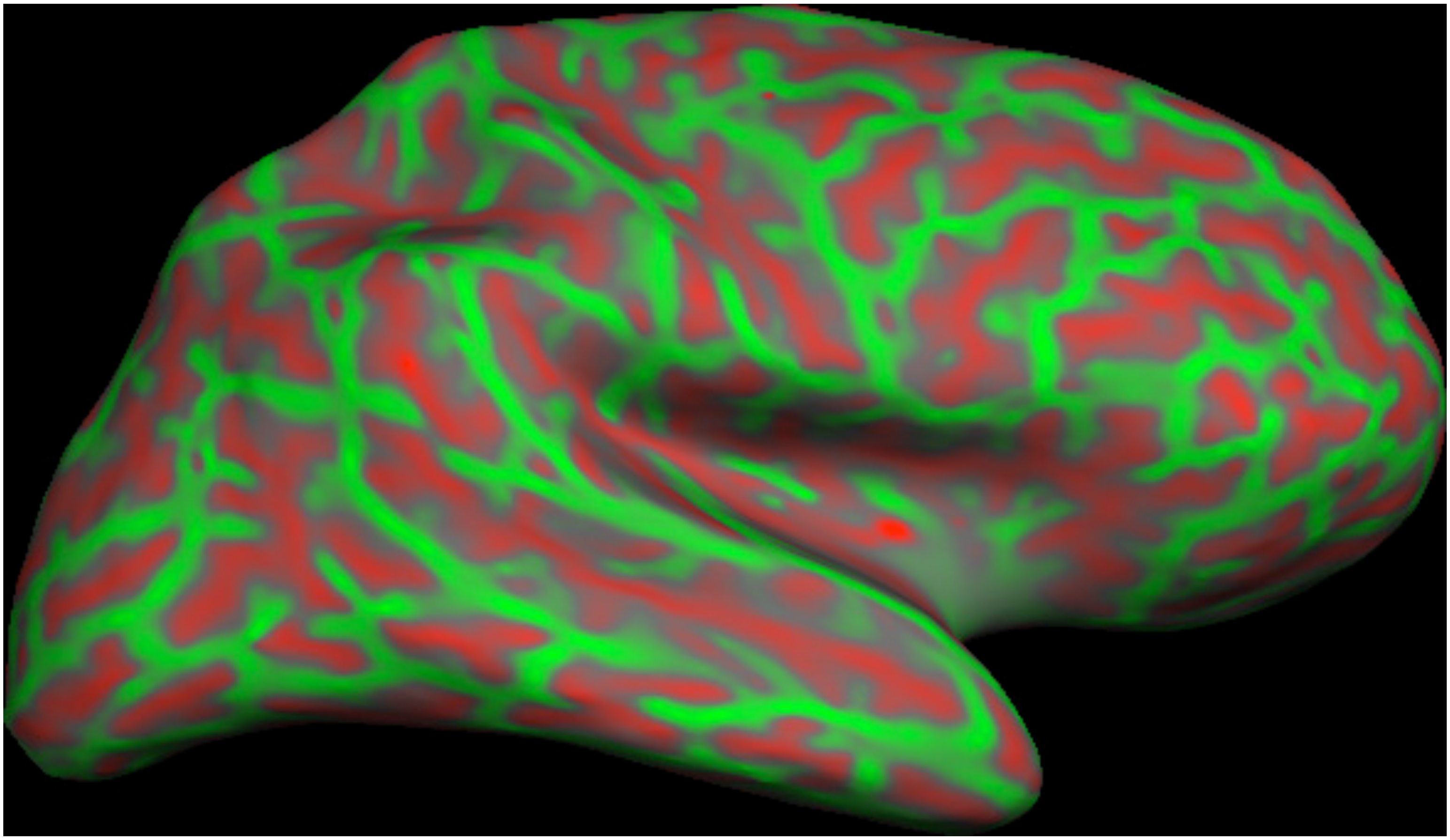
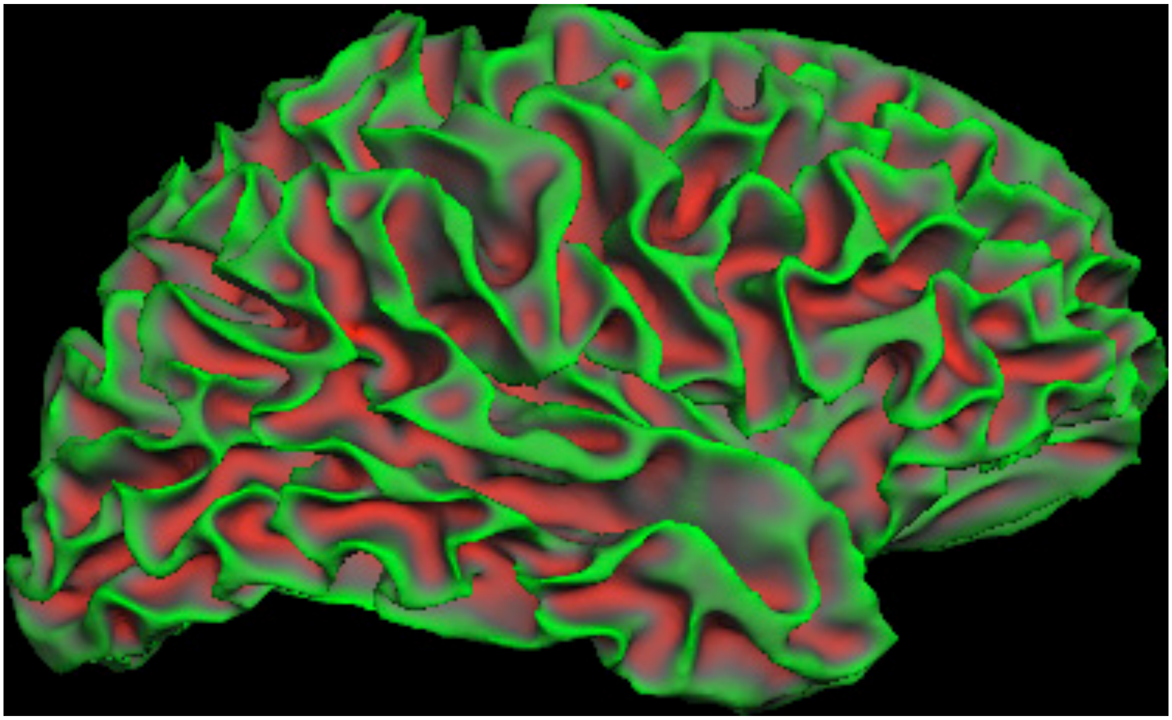
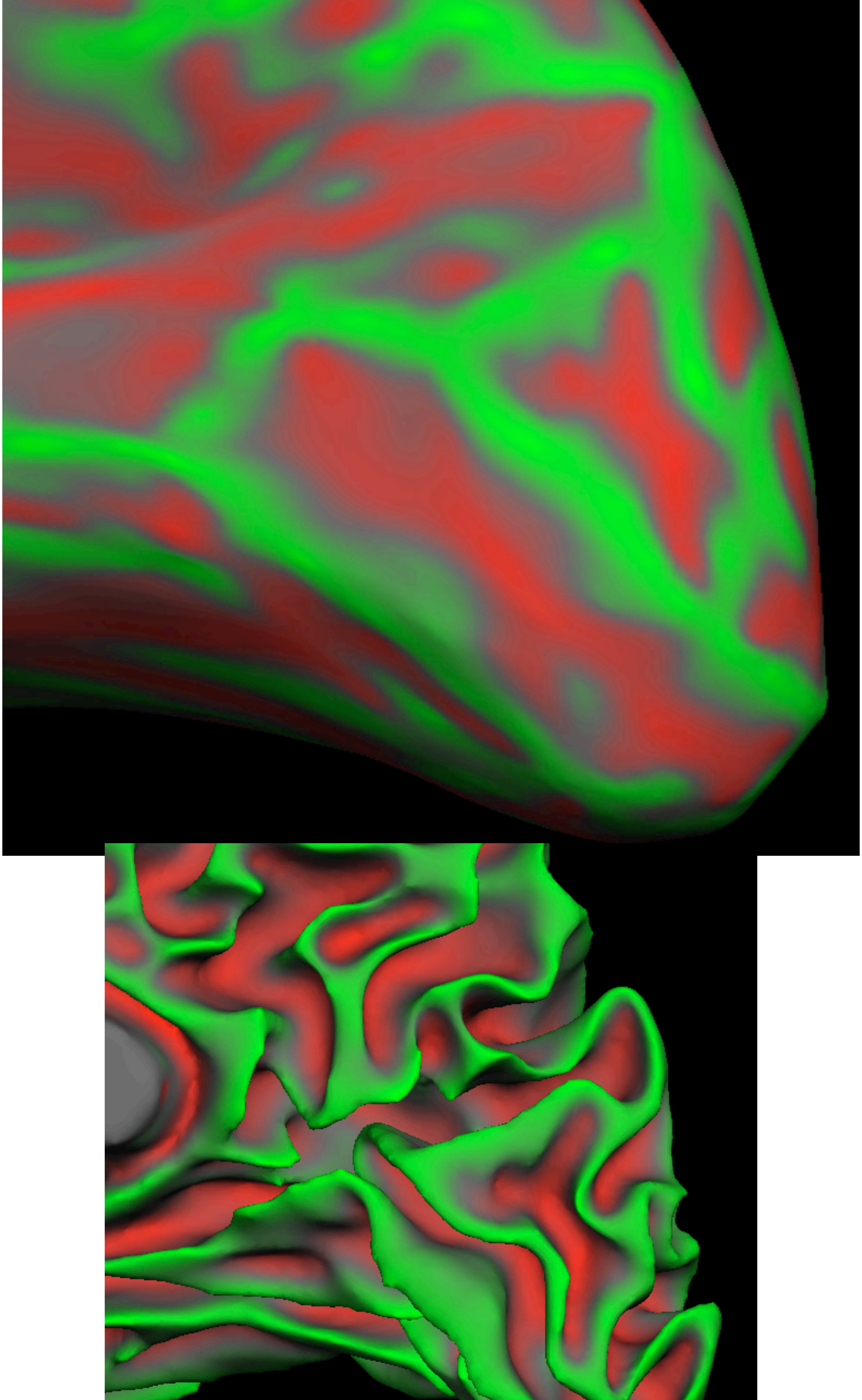






Visual System Overview

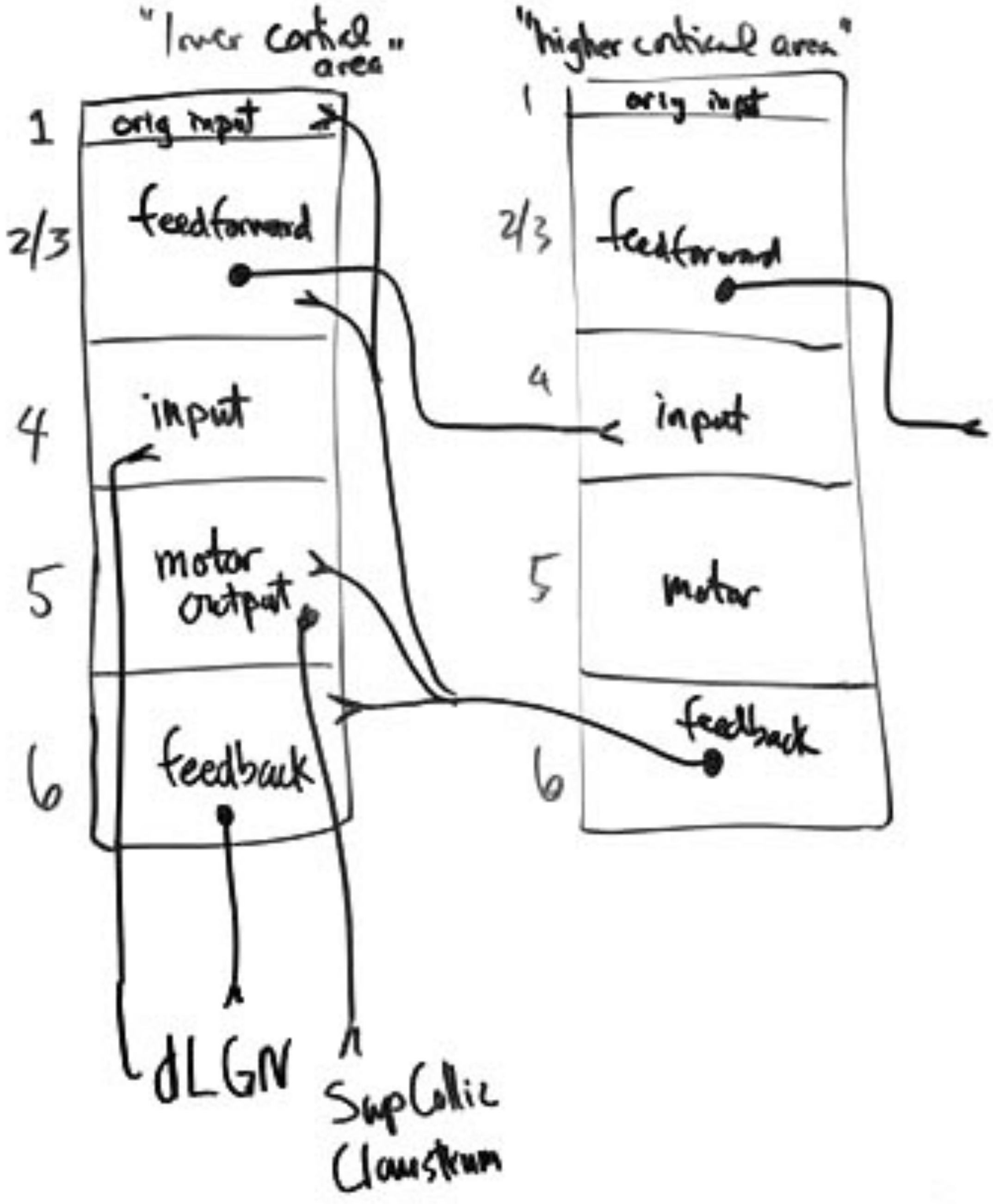
Areas

- 30 cortical
  - 50% of total neocortex
  - 10 - 100 million neurons each
- 10 thalamic
  - retic
  - sup pulv
  - inf pulv
  - IT pulv
- 10 midbrain

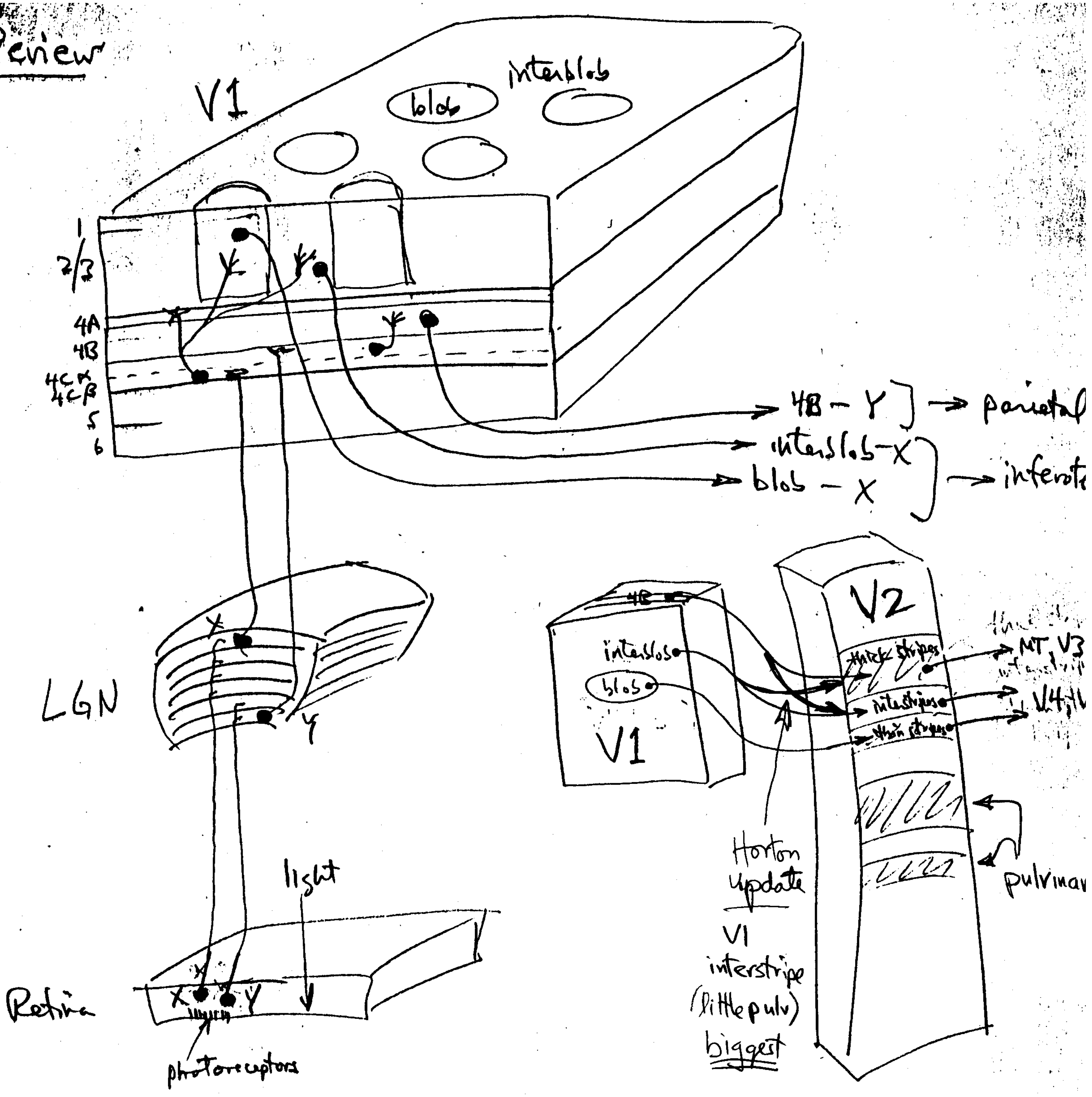
Connections

- each area connects to
  - ~10 cortical
  - ~5 thalamic
  - usu. reciprocal
- perhaps 800 interareal tracts
- each cortical area has outputs to several of these:
  - frontal
  - striatum
  - sup. collic
  - pontine nuc.

gen. scheme for cortical layers



Review

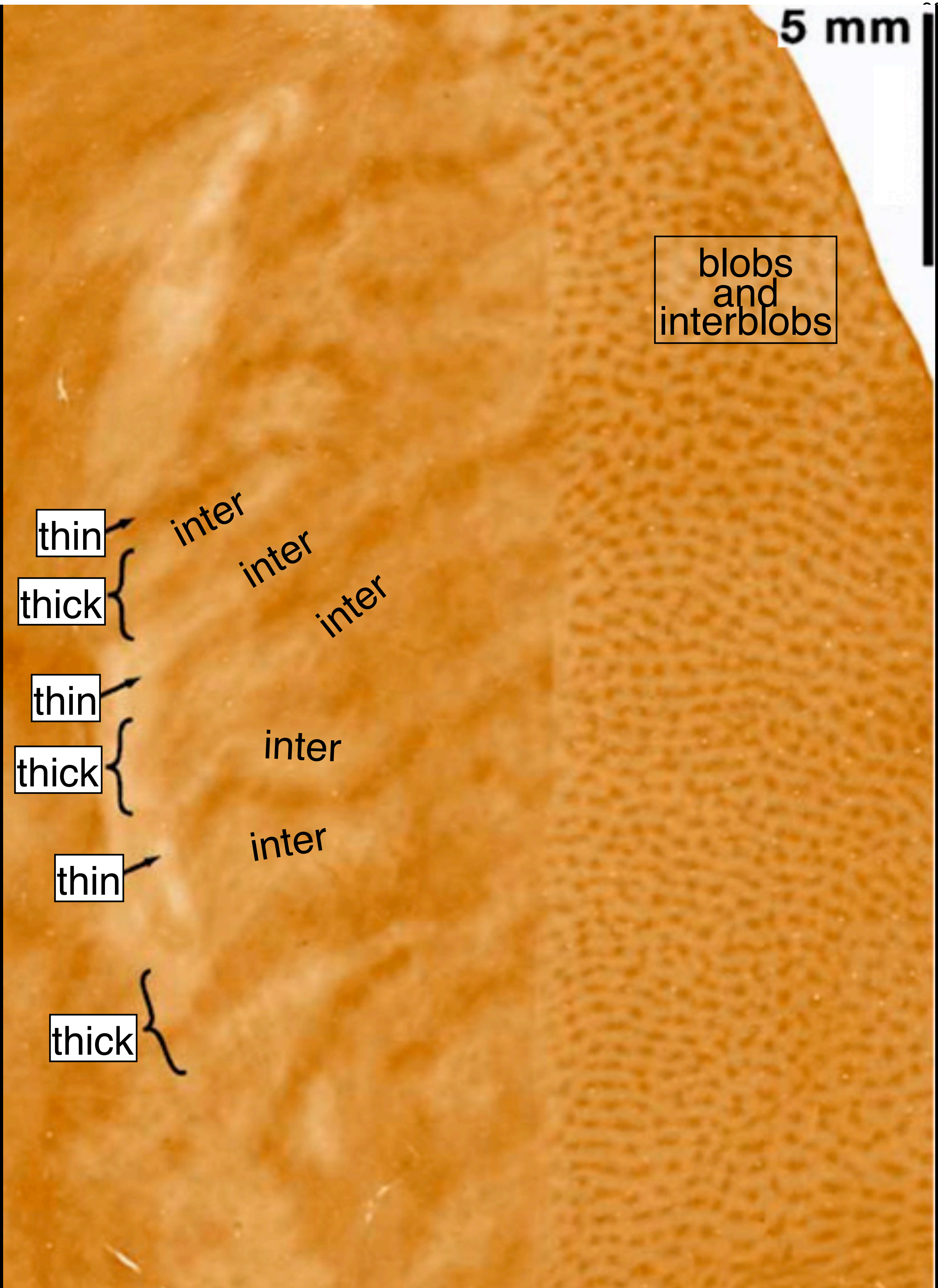


"dorsal" MT, V3 ⟶ MST ⟶ 7a

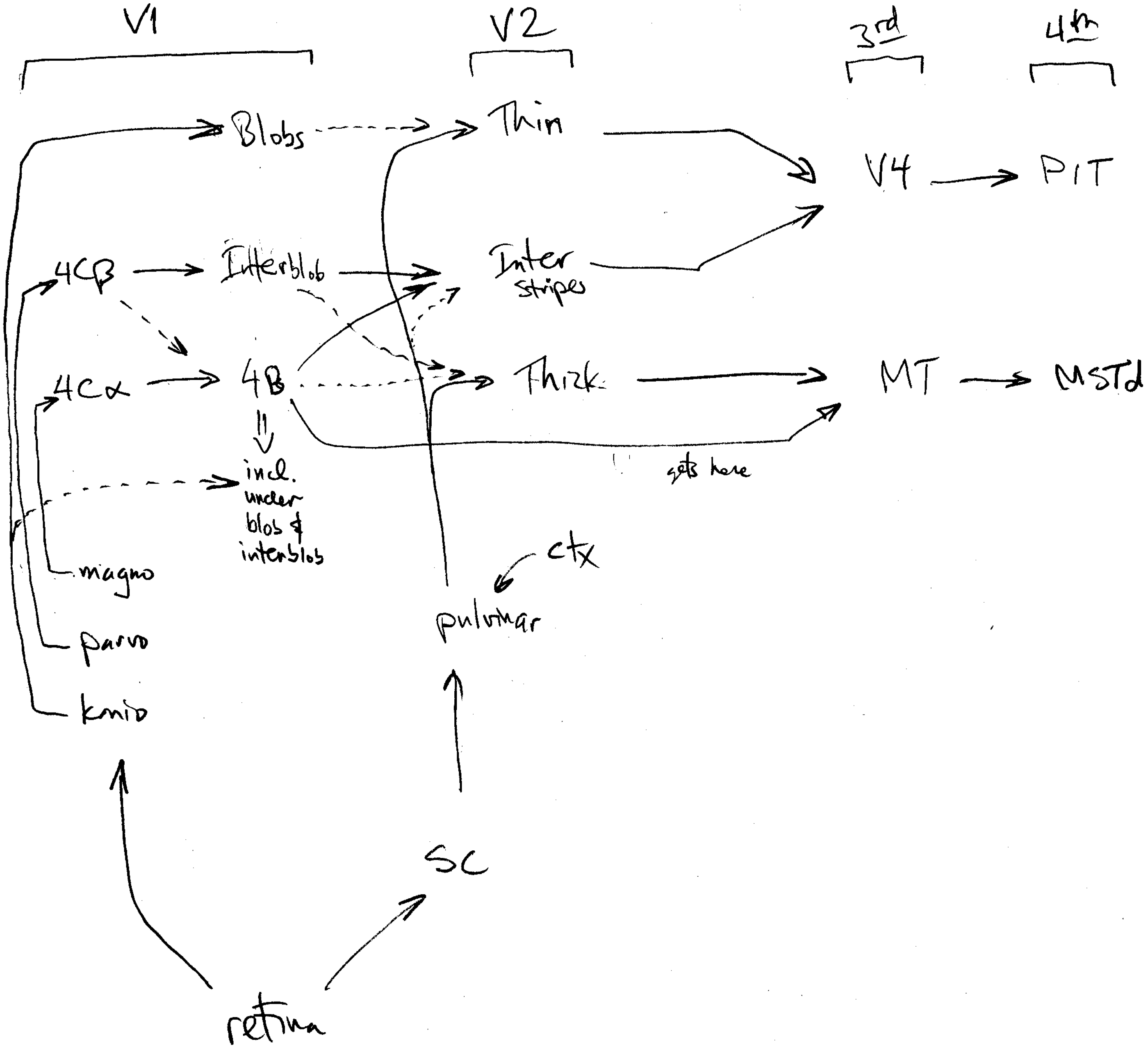
"ventral" V4, VP ⟶ PIT ⟶ AIT

– crude summary of ~300 corticocortical connection bundles between areas.

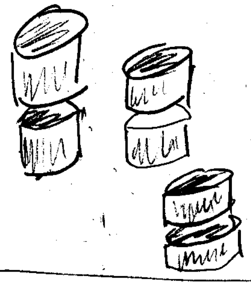
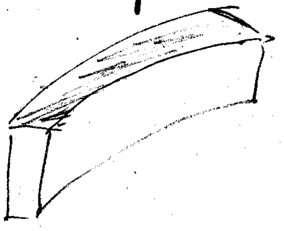
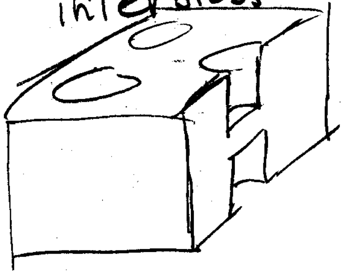
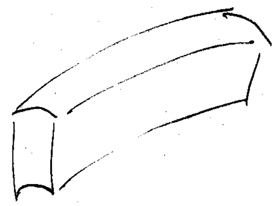
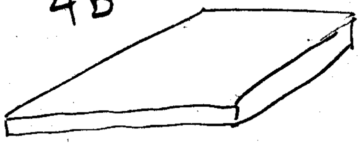
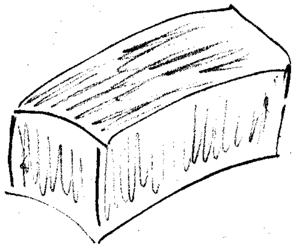
– *also*, remember the thalamus

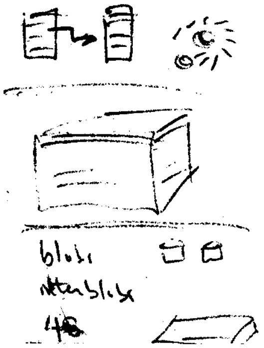

5 mm
blobs and interblobs
thin
inter
thick
inter
inter
thin
thick
inter
thin
inter
thick

Blobs, 4B, Interblobs / stripes — NEW



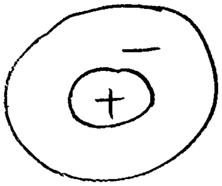
- differentiate, then remix
- motion OK w/ complete retinal parasol lesion (w/ contrast > 10%)

V1	V2	properties
blobs 	thin stripes 	- color selective (but cf. Galogo) ↳ **brightness** - large dynamic range (0-150)
interblobs 	interstripes 	- *orientation* selective - not color selective but can use color to detect orientation - small dynamic range (0-50
4B 	thick stripes 	- *direction* selective

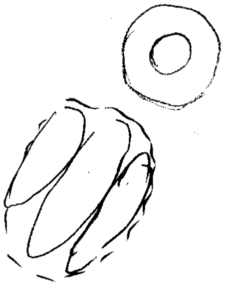


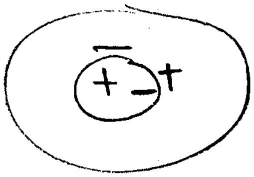
Basic Visual Physiology

non-opponent



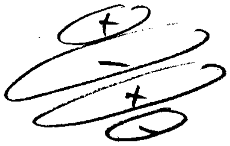
simple-opponent



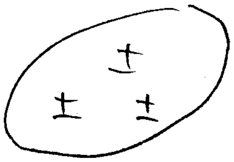


double-opponent

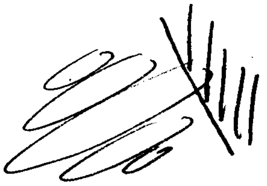
moon ≈ ½ deg vis angle



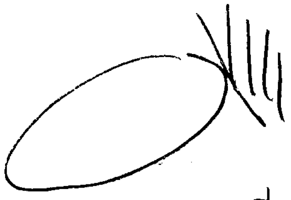
simple cells



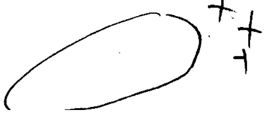
Complex cells



"simple
"hypercomplex" cells



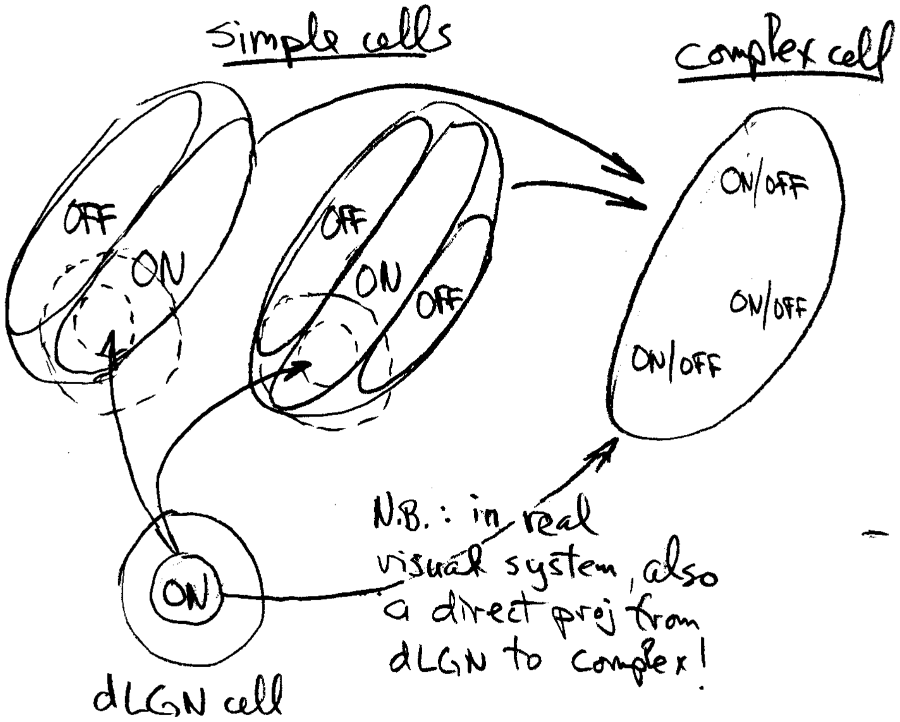
complex
hypercomplex cells



special complex (= anti-hypercomplex!)

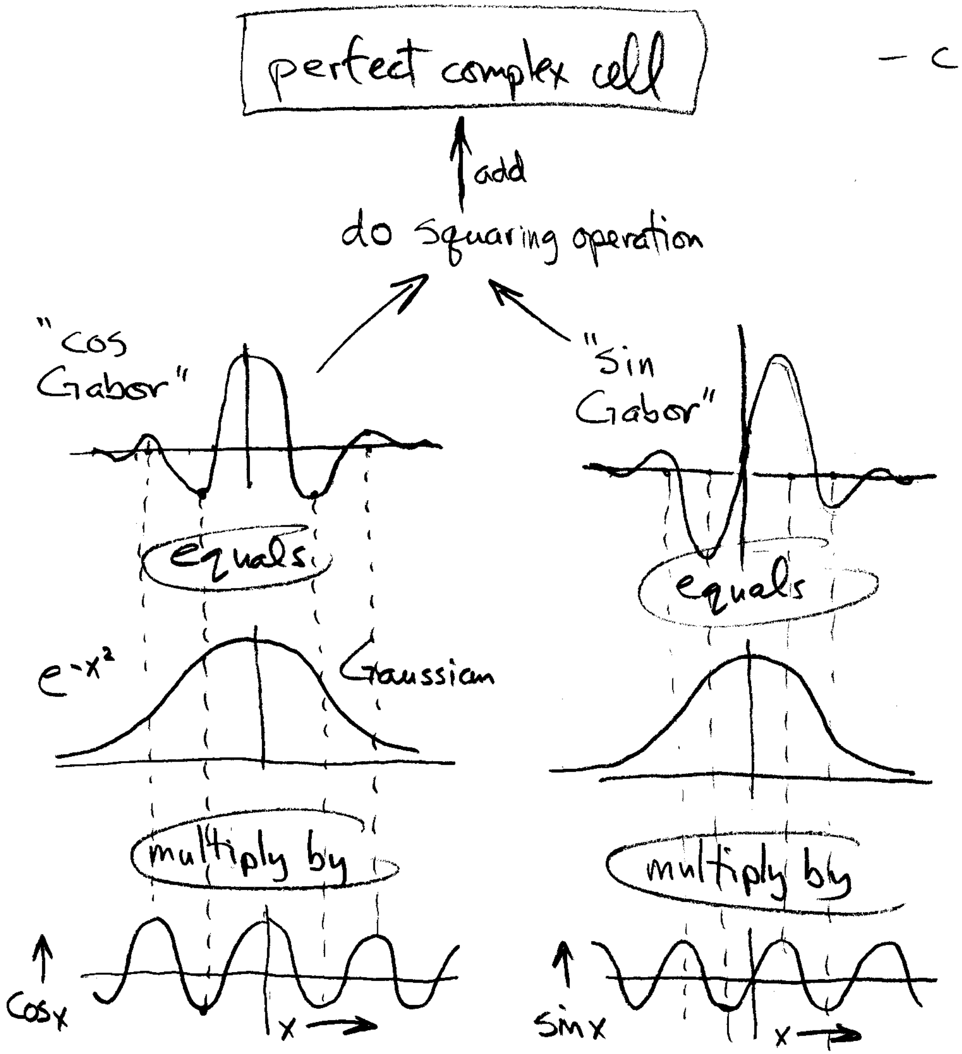
COMPLEX CELL MODEL — GABOR FILTERS

↳ orientation "energy" model



- N.B.: real simple cells have excitatory responses to ON & OFF (no negative responses in real cells)

- model simple cells by convolution of visual stim with Gabor filters (heavily used in machine vision)



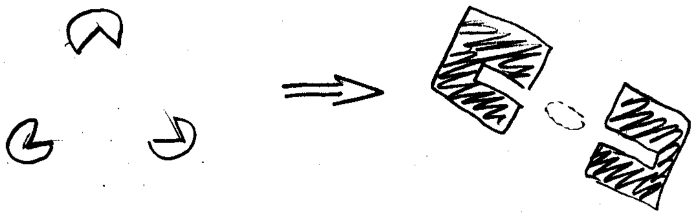
- convolving with individual Gabor finds oriented stripes in stim (cross prod = corr), but response varies with stim positioning in R.F.

- N.B.: trig identity $\cos^2 x + \sin^2 x = 1$

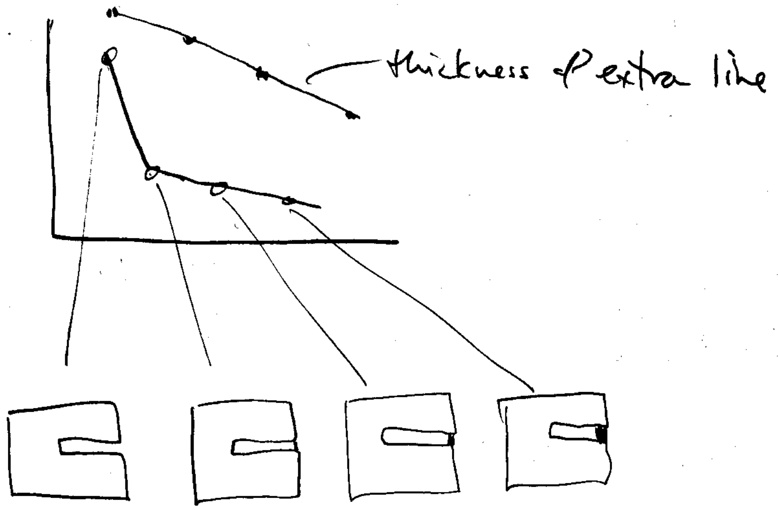
- Combining cos & sin Gabors eliminates response variation

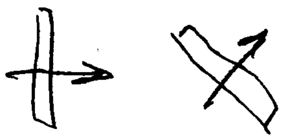
* output detects ripple-i-ness in stimulus independent of exact position of ripples in R.F.

Van der Heydt et al

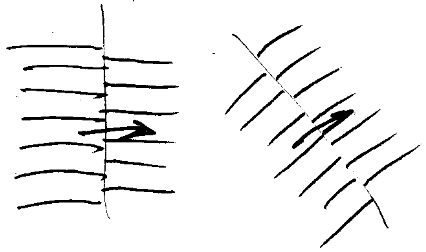


V1 – cells respond only to real contours
V2 – some cells respond to illusory





cf.

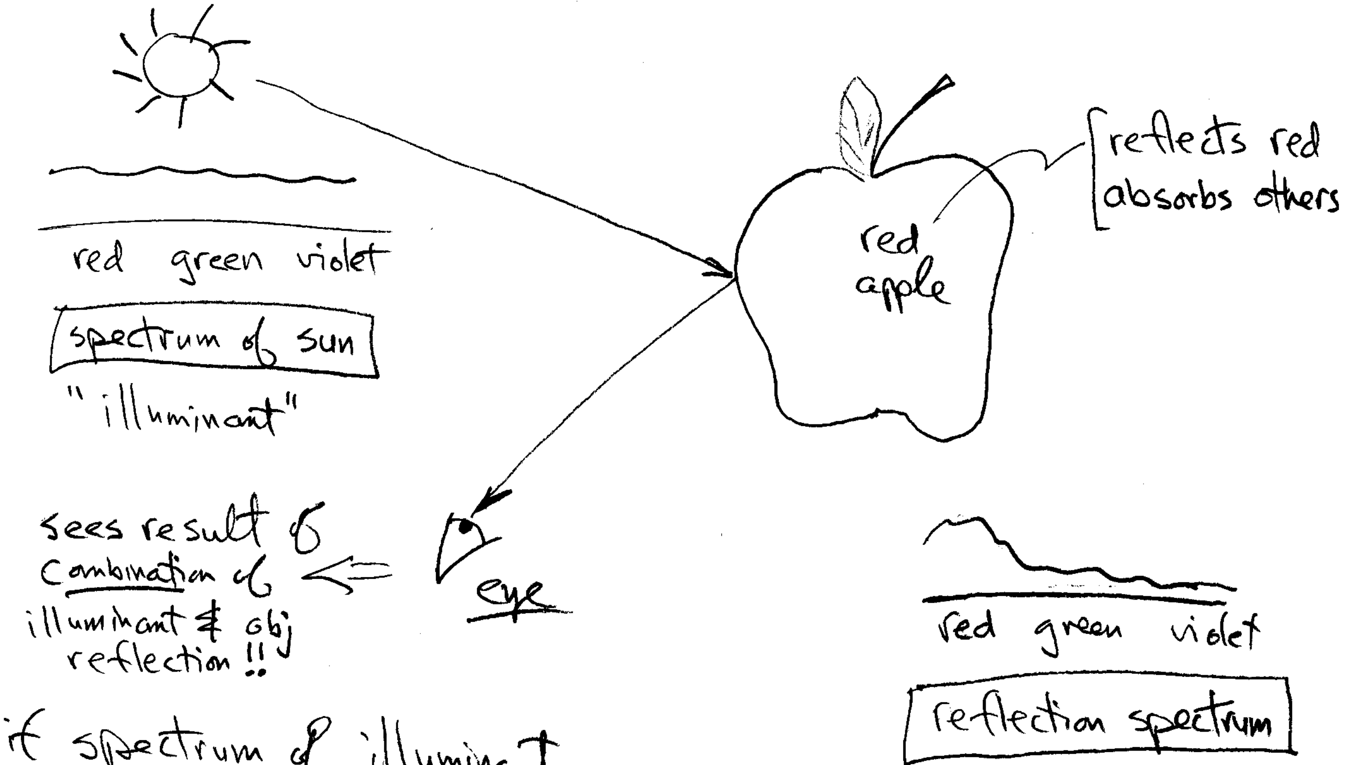


APERTURE PROBLEMS IN GENERAL

- to process local detail, need local views
- like viewing the world through straws
- V1 is "dumber" than you think
- a general problem
  - across modalities
  - across different stimulus features
  - across levels of processing
- other examples
  - object color
  - pattern motion
  - complex motion
  - Texture
  - ITD
- solved by combining information across space
  ↳ i.e., across receptor map space
- higher areas are "smarter" than they first look
  (e.g. often have "sloppy"-looking tuning curves
  to simple stimuli)

DETECTING COLOR AS APERTURE PROB

- "color" is spectrum of what is reflected (not absorbed)
- depends on properties of object (e.g. a ripe fruit)
- but spectrum of illumination affects final result



reflects red
absorbs others

sees result of combination of illuminant & obj reflection!!

↳ tells features of obj, indep. of illumination

- if spectrum of illuminant is biased (e.g. mostly orange light inside pub) even green objects will be mostly orange (tho less orange than orange obj)
- green objects still look green! → color constany
- this color constancy will fail in monochromatic light (e.g. Sodium vapor lamp) so req's some range of colors

Aperture Problem For Pattern Motion

Overview

Y cells → magno LGN → 4Cα → 4B → V2-thick → MT

4B → MT

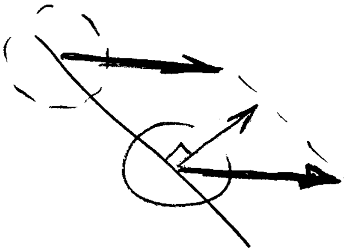
Y cells ⇓ center surround

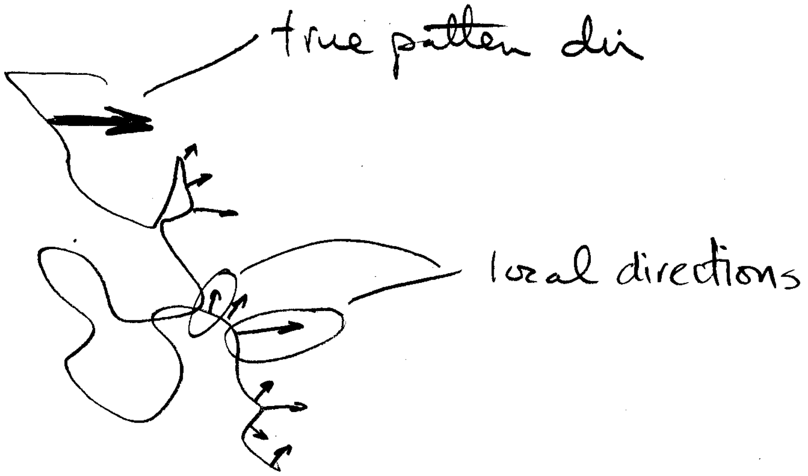
magno LGN ⇓ center surround

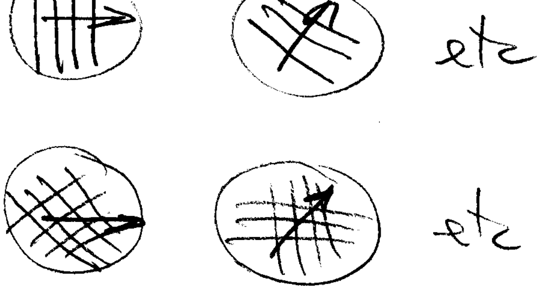
4Cα ⇓ center-surround & oriented

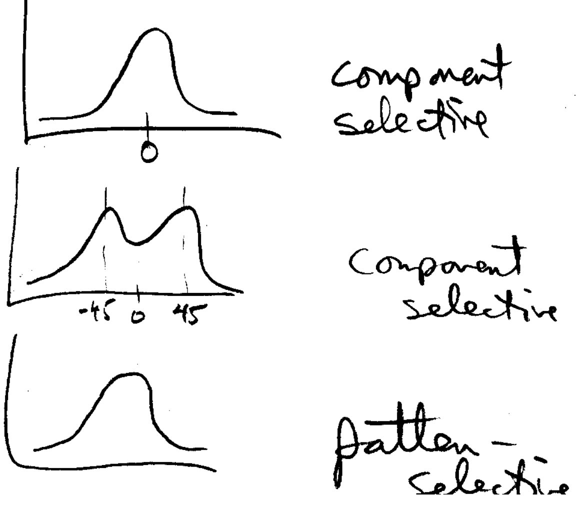
4B ⇓ oriented direction selective

MT: oriented dir. selective / pattern-selective

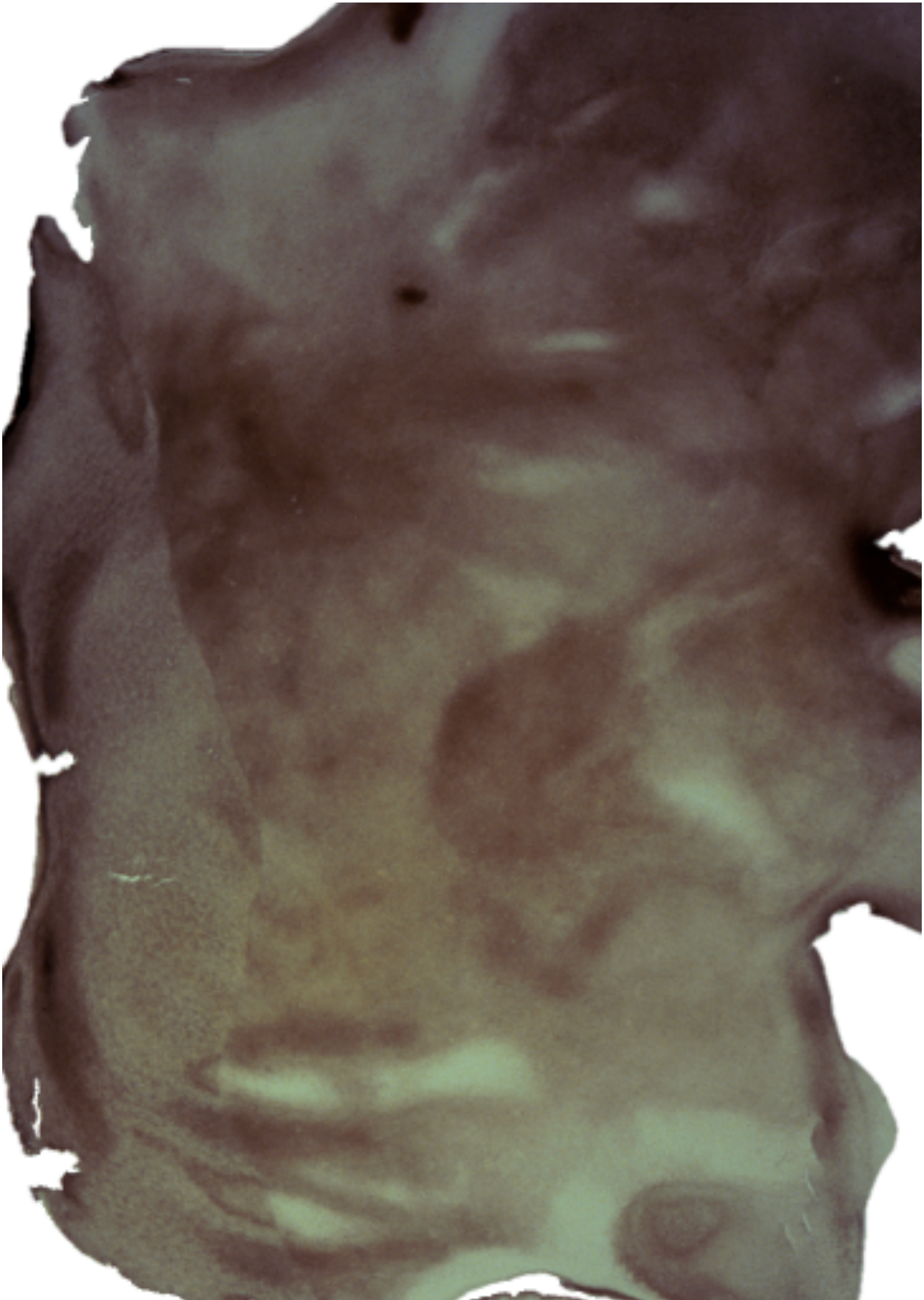






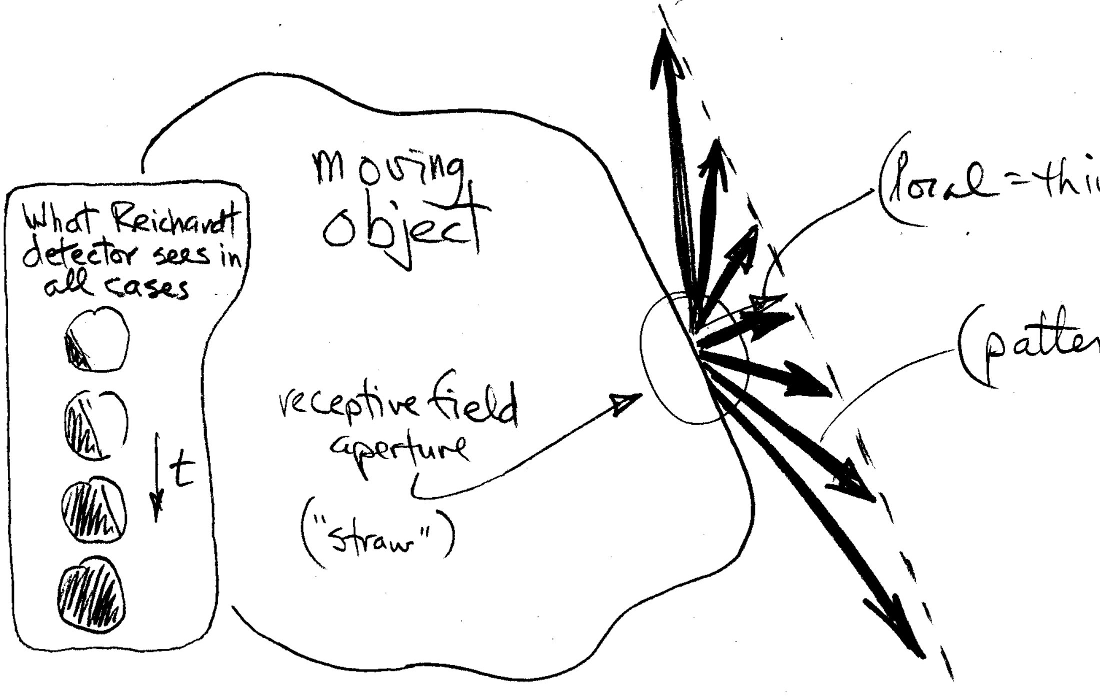


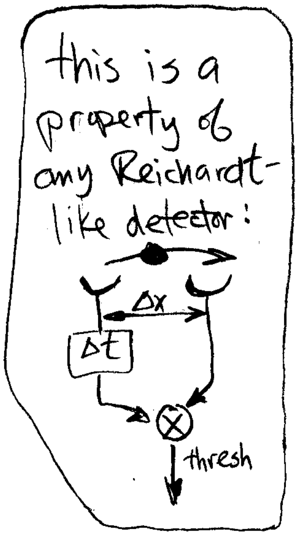
Saito

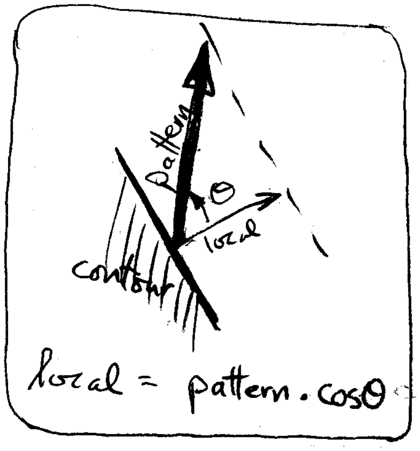


View from V1

Family of pattern directions that could have given rise to this one local direction



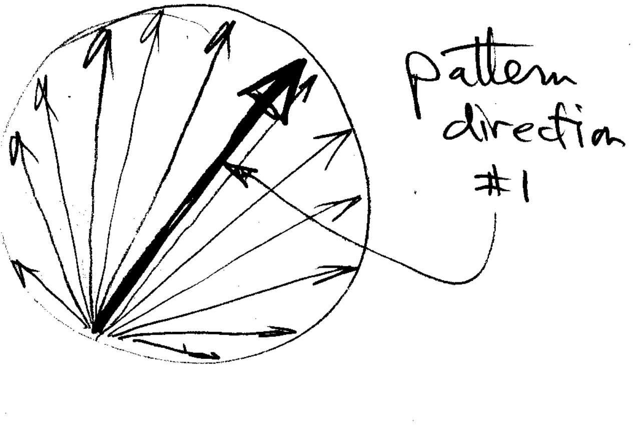




↳ i.e. V1,4B detects component of pattern motion ⊥ to contour

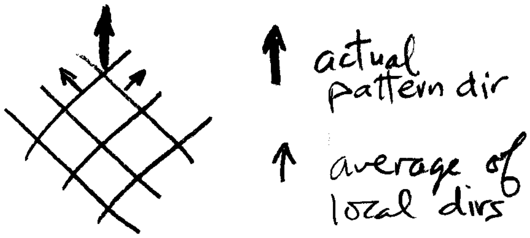
View from MT

Family of local directions consistent with one pattern direction

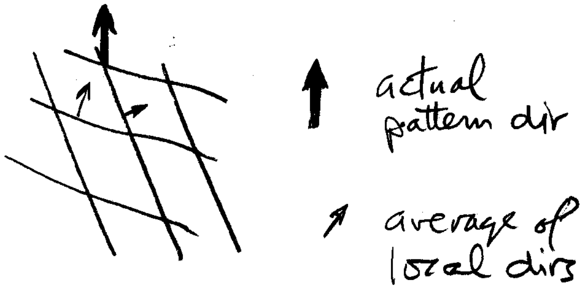


Why the average local direction won't work in general

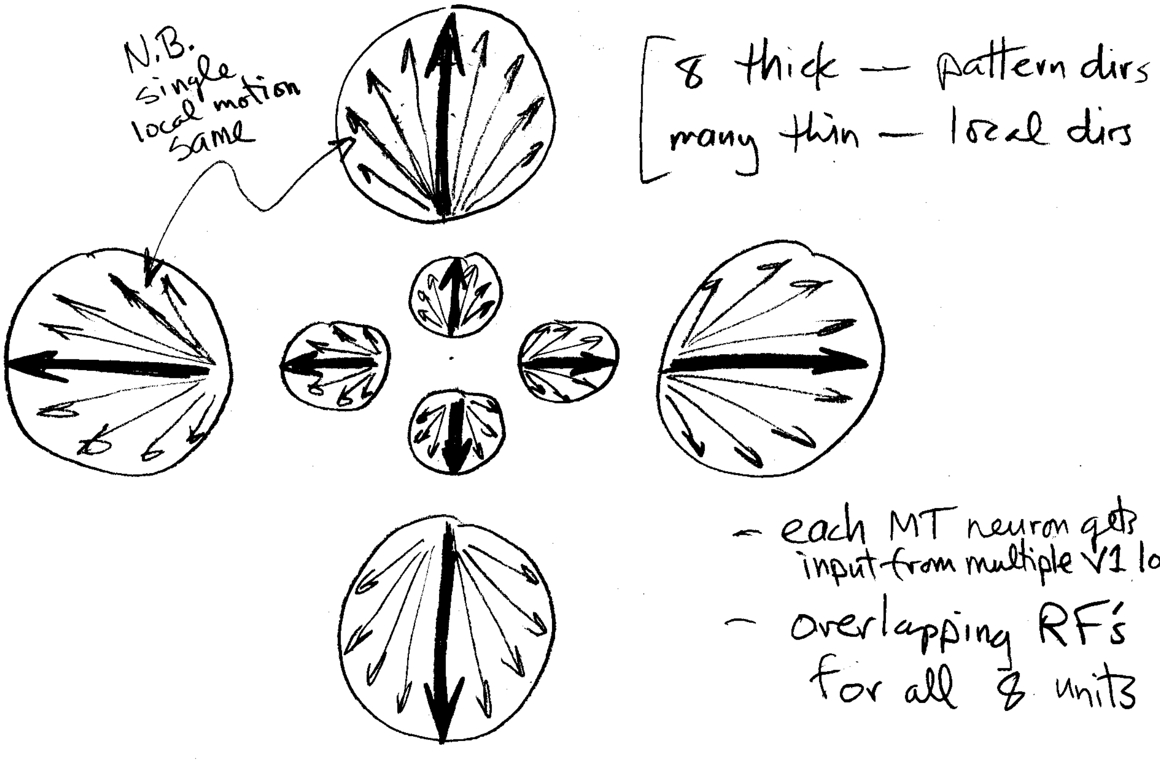
average works
(in same direction
tho speed wrong)



average doesn't work



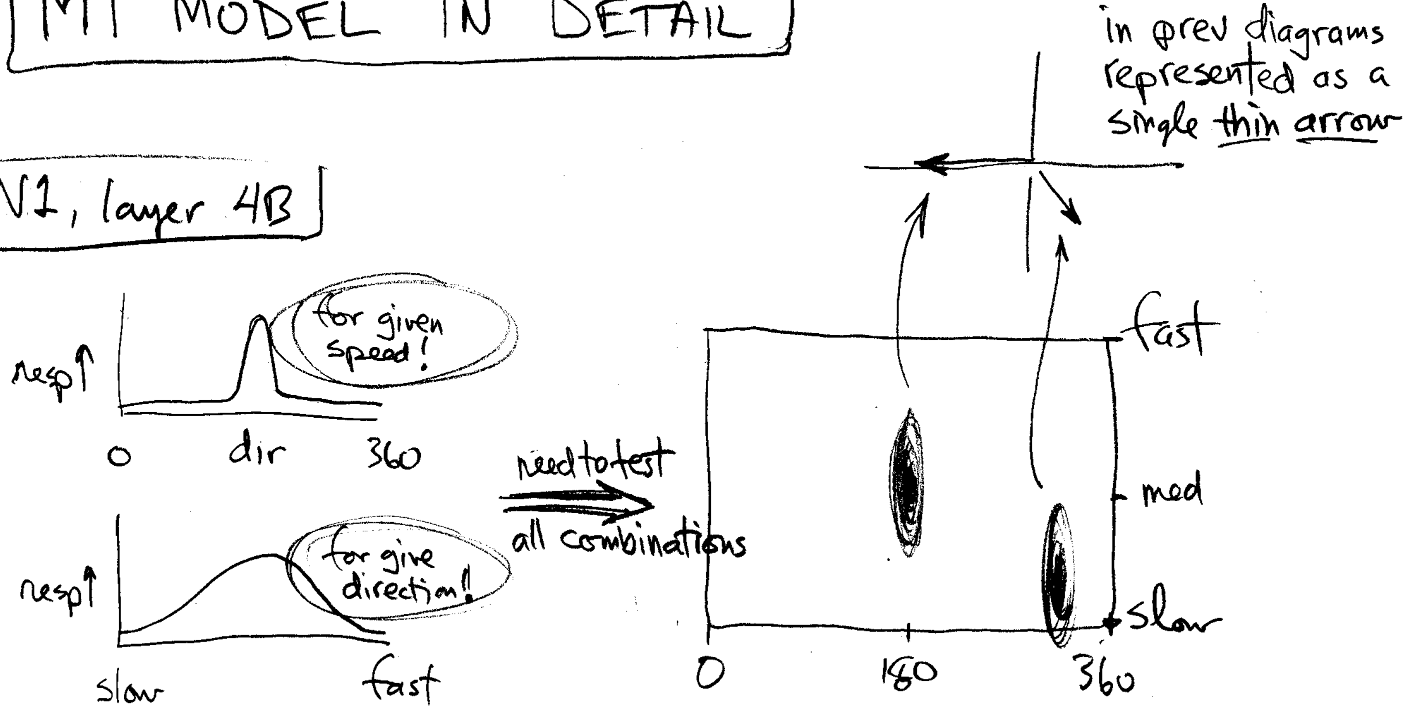
Model of map point in MT that can detect 8 different pattern directions

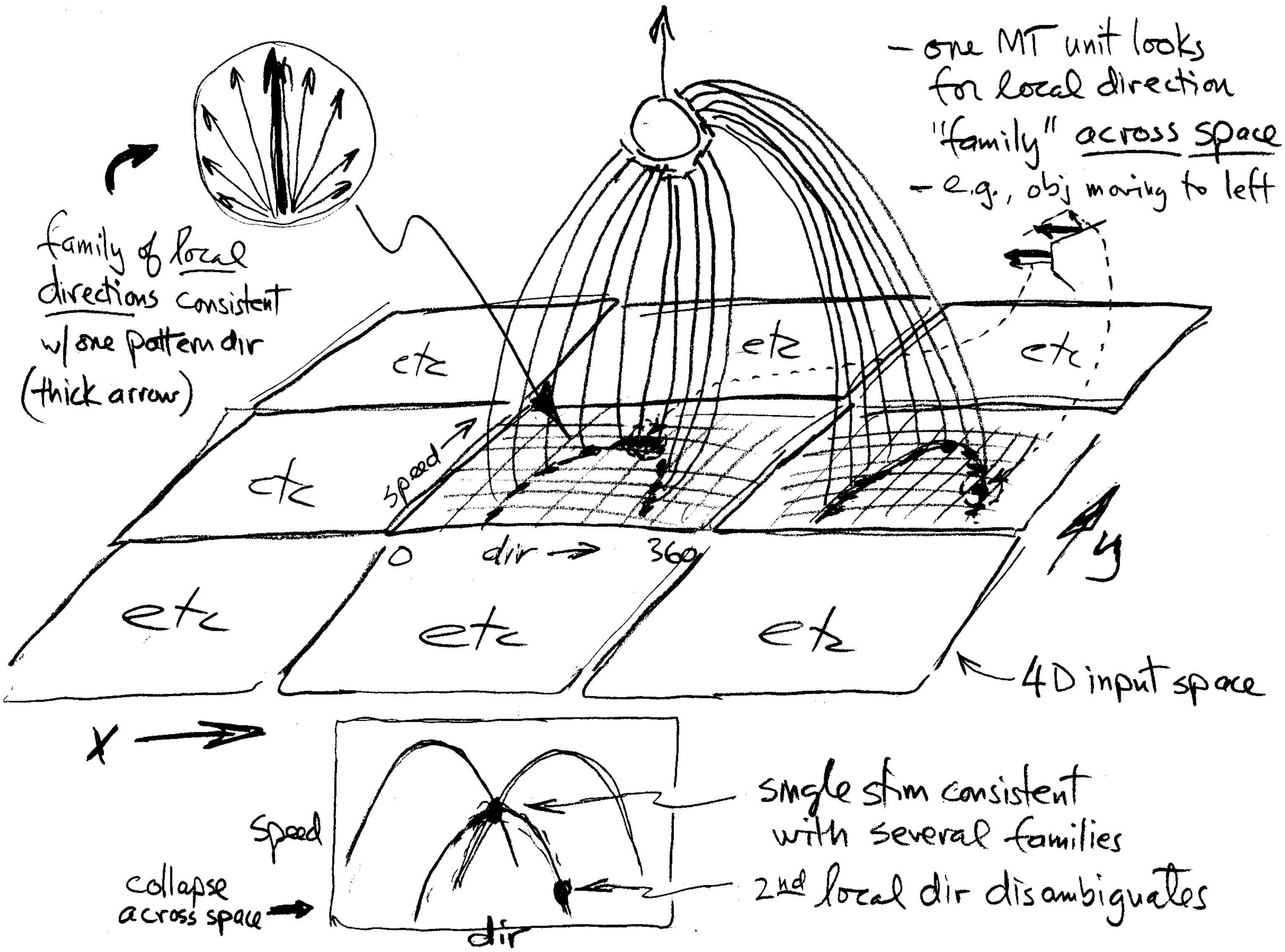


- each MT neuron gets input from multiple V1 locations
- overlapping RF's for all 8 units

MT MODEL IN DETAIL

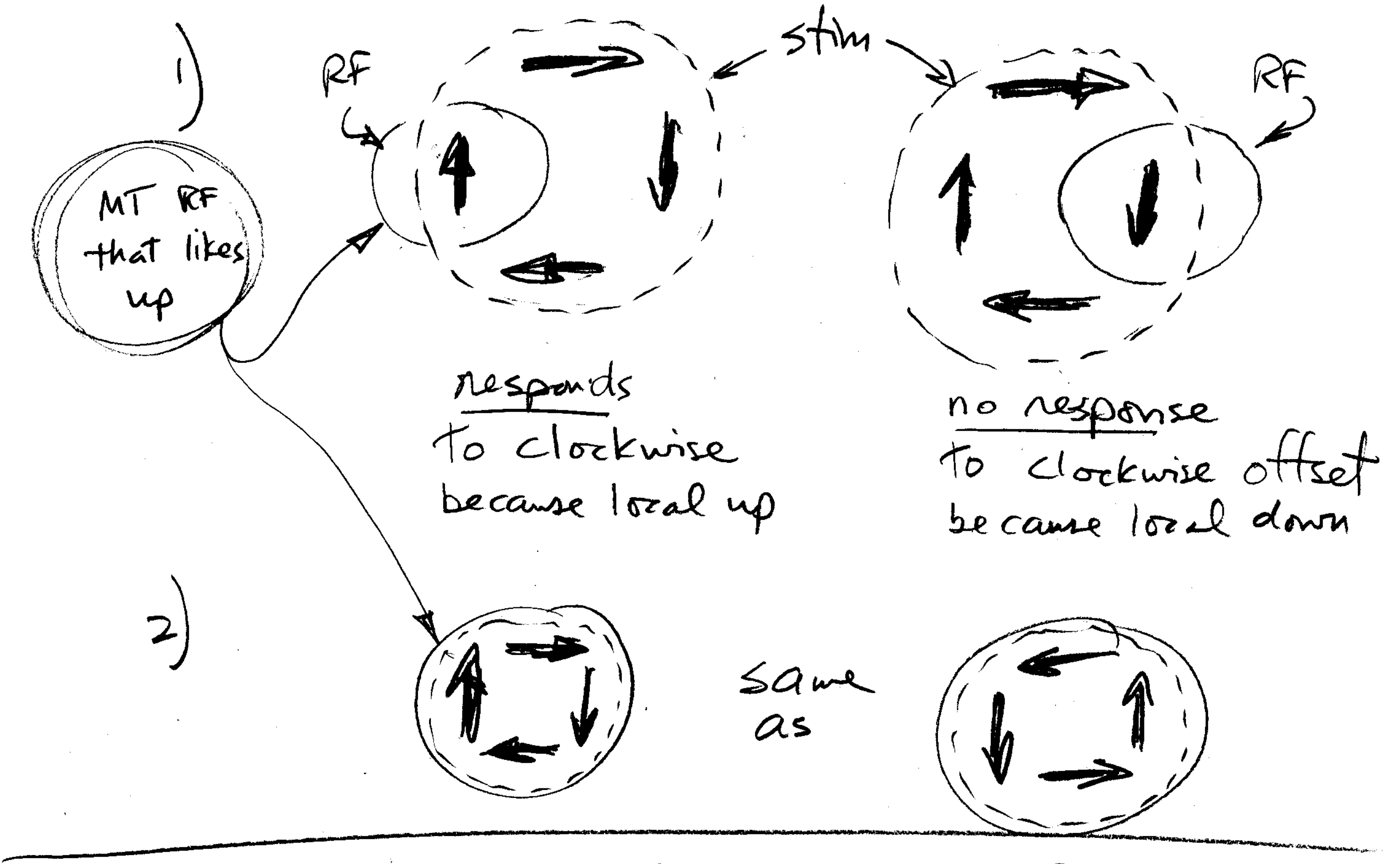
V1, layer 4B





MSTd

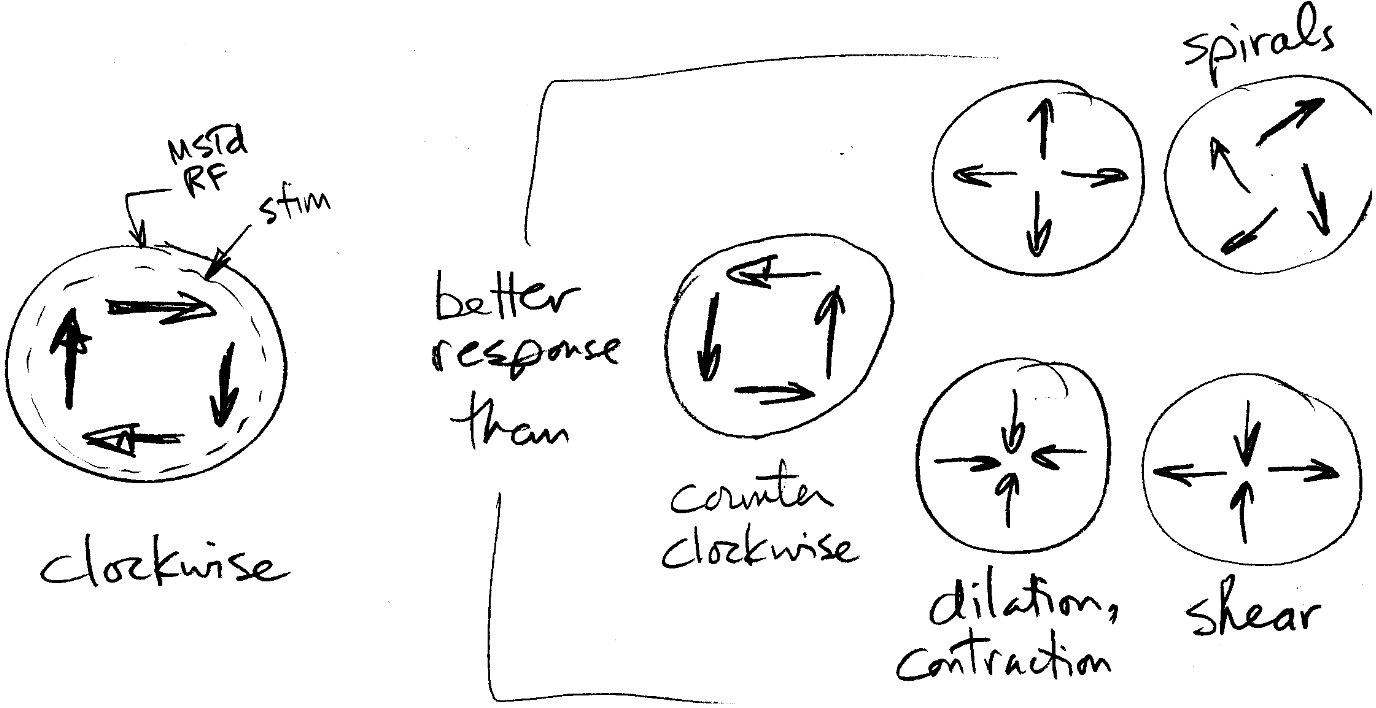
MT <u>responds</u> to rotation/dilation but <u>not selectively</u>



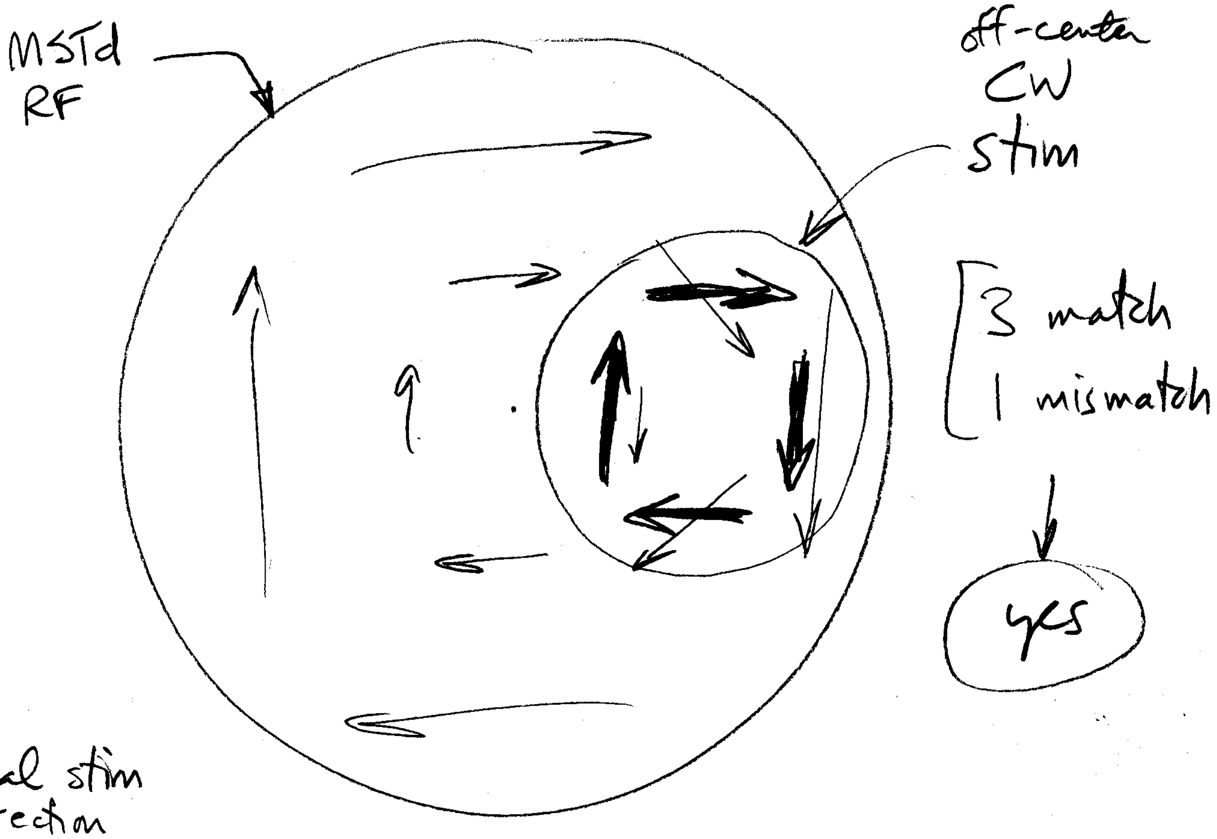
<u>responds</u> to clockwise because local up

no <u>response</u> to clockwise offset because local down

MSTd <u>distinguishes</u> different flow fields

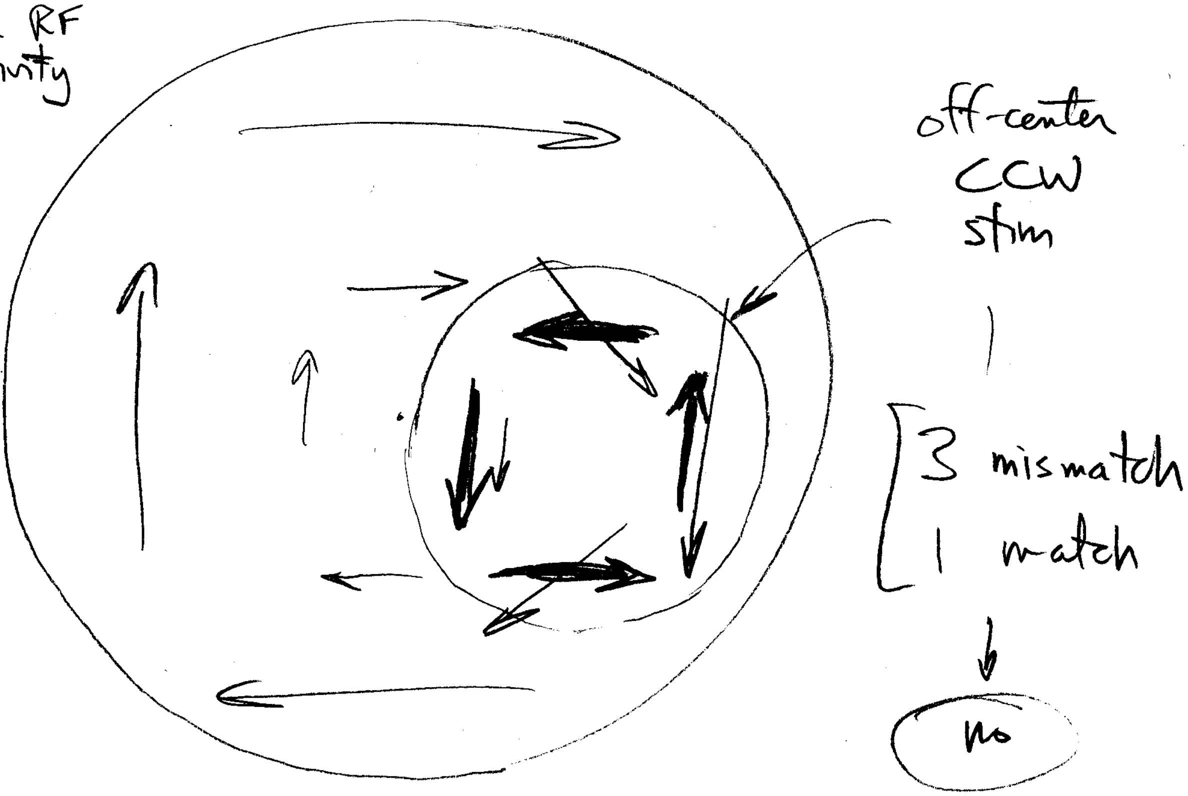


How a fixed template actually works



🡅 thick – local stim direction

↑ thin – local RF sensitivity



DETERMINING OPTICAL FLOW

Horn & Schunck, 1981
"gradient model"

brightness of image point $= E(x,y,t)$

assume brightness doesn't change w/move: $\frac{dE}{dt} = 0$

[no shadows
no lighting change

[movement $E(x,y,t) = E(x+\delta x, y+\delta y, t+\delta t)$

[Taylor expand around this point $E(x,y,t) = E(x,y,t) + \delta x \frac{\partial E}{\partial x} + \delta y \frac{\partial E}{\partial y} + \delta t \frac{dE}{dt} + \varepsilon$ ← higher order Taylor terms

[subtract $E(x,y,t)$
divide by δt
take limit $\delta t \to 0$

$$0 = \frac{\delta x}{\delta t}\frac{\partial E}{\partial x} + \frac{\delta y}{\delta t}\frac{\partial E}{\partial y} + \frac{\partial E}{\partial t} + \text{higher order}$$

→ ignore in limit of $\delta t \to 0$

$$\frac{\partial E}{\partial x}\frac{dx}{dt} + \frac{\partial E}{\partial y}\frac{dy}{dt} + \frac{\partial E}{\partial t} = 0$$

data — spatial gradient

data — temp. grad

unknowns

pixels: x, y

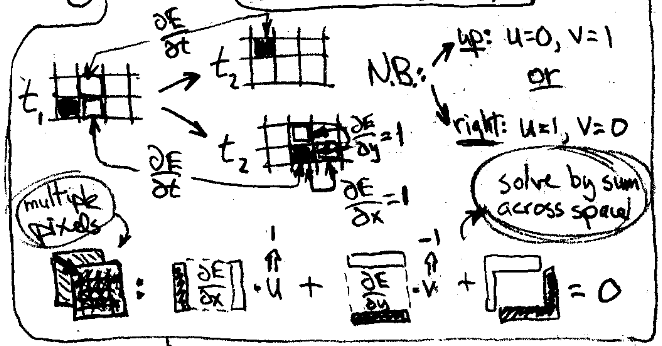
t_1 t_2

$\frac{dx}{dt} = u$

$\frac{dy}{dt} = v$

$$\boxed{\frac{\partial E}{\partial x}u + \frac{\partial E}{\partial y}v + \frac{\partial E}{\partial t} = 0}$$

finite difference example



Solve for $\frac{dy}{dt}$:

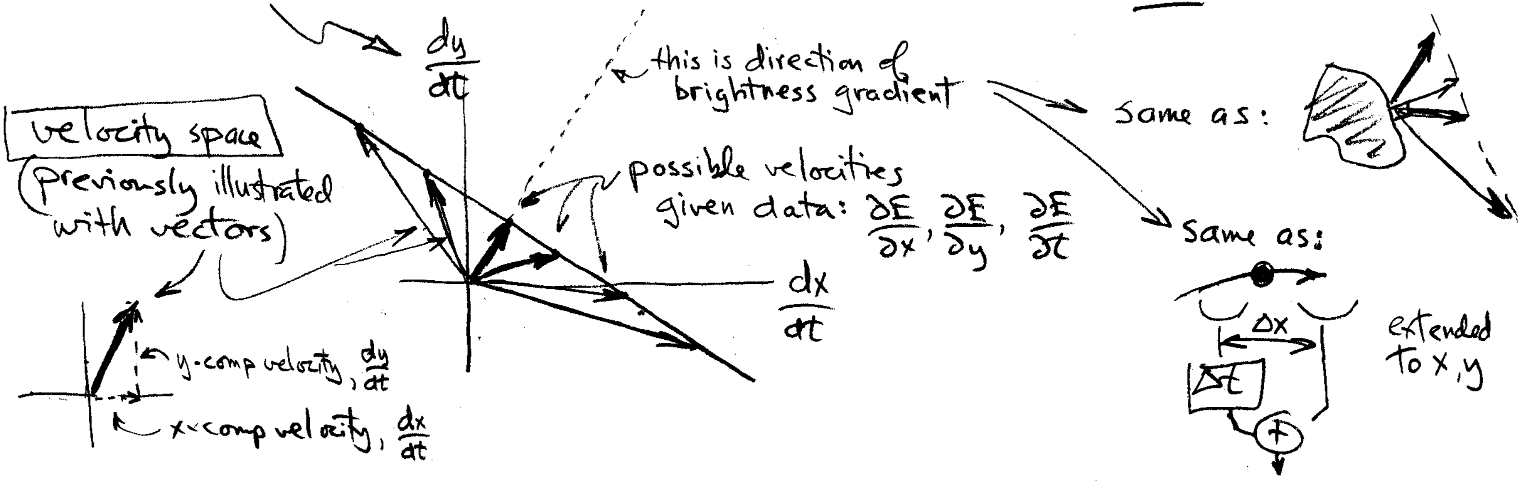
unknown!

$$\frac{dy}{dt} = -\frac{\frac{\partial E}{\partial x}}{\frac{\partial E}{\partial y}}\boxed{\frac{dx}{dt}} - \frac{\frac{\partial E}{\partial t}}{\frac{\partial E}{\partial y}}$$

$\frac{dy}{dt}$ is therefore dependent on unknown x-velocity (i.e., the aperture problem!)
↳ in Cartesian coord form

$$y = m x + b$$

⇒ this is equation for a line



COMPUTATIONAL MOTION

Horn & Schunk (2)

Smoothness
- since two variables at each point $\left(\frac{dx}{dt}, \frac{dy}{dt}\right)$ need additional constraints to solve even for rigid field
- add assumption of smoothness (gives completely smooth answer for rigid object, of course)

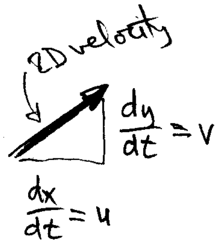
minimize: error + non-smoothness

should = 0

$$\text{error} = \iint \left[\frac{\partial E}{\partial x}\frac{dx}{dt} + \frac{\partial E}{\partial y}\frac{dy}{dt} + \frac{\partial E}{\partial t} \right]^2 dx\,dy$$

$$\text{non-smoothness} \iint \left(\left[\overset{\rightarrow\rightarrow}{\left(\frac{\partial u}{\partial x}\right)^2} + \overset{\rightrightarrows}{\left(\frac{\partial u}{\partial y}\right)^2} \right] + \overset{\uparrow\uparrow}{\left(\frac{\partial v}{\partial x}\right)^2} + \overset{\uparrow\uparrow}{\left(\frac{\partial v}{\partial y}\right)^2} \right) dx\,dy$$

$$\left(u = \frac{dx}{dt} \qquad v = \frac{dy}{dt} \right)$$



$$\boxed{\min: \quad e_d + \alpha e_s}$$

Examples

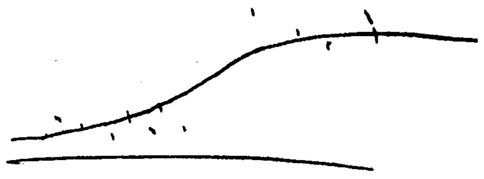
Iterative solution to minimization problem

stable in ~10-20 steps

Problems
- people can't detect movement of smooth brightness gradients
- smoothness smears edges

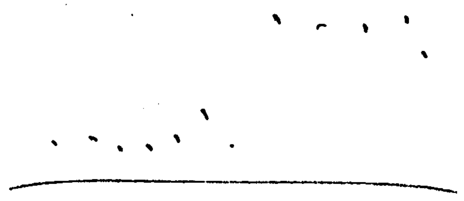
Line Processes

data

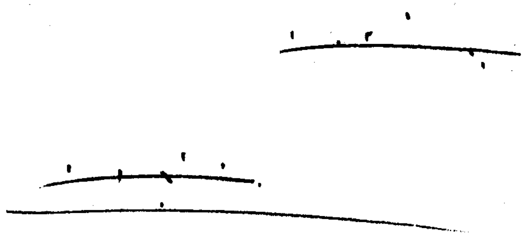


smoothness constraint

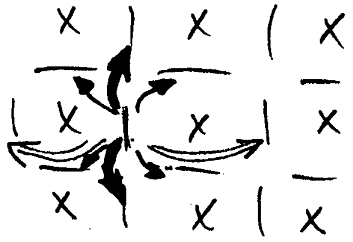
minimize → error + non-smoothness + penalty for discontinuity + penalty for non-collinear



data



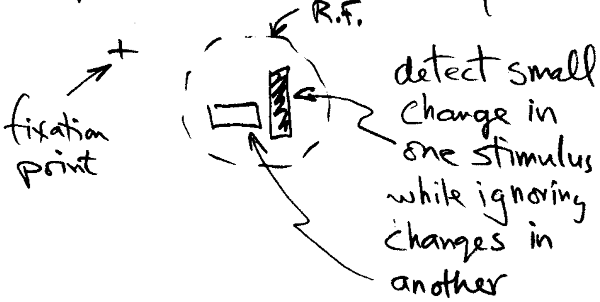
smoothness + line process



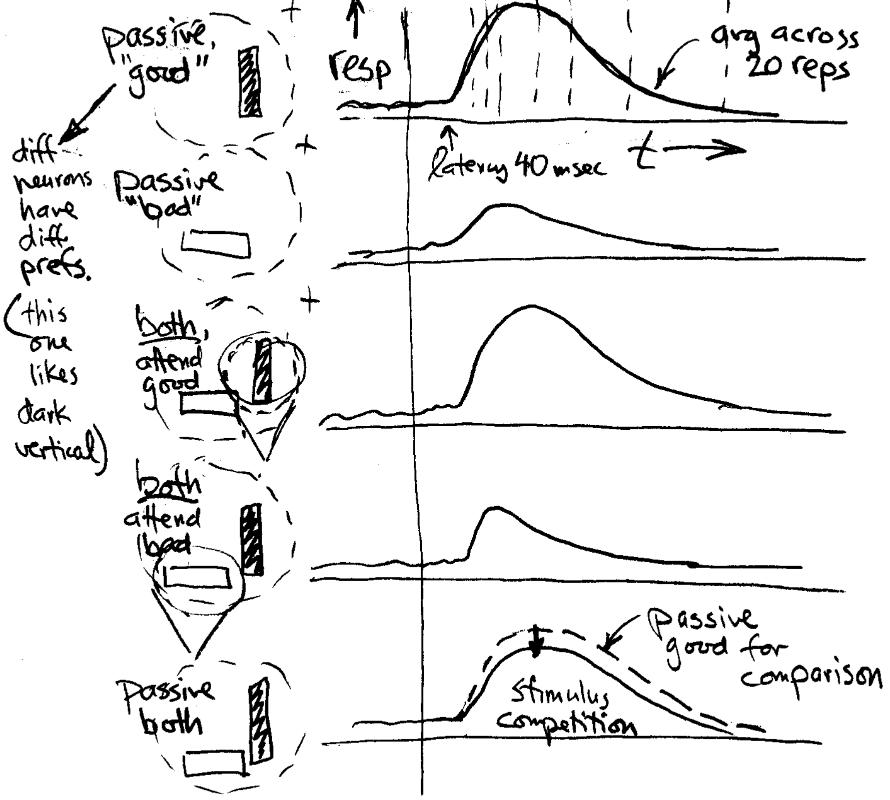
EFFECTS OF ATTENTION IN V4

Moran & Desimone, 1985

- standard model attention :
  - ↳ this is wrong
    attention is everywhere !
- macaque monkeys are
  pre-adapted for peripheral
  attention expts
  1) spend all day <u>not</u> looking
     directly at attended targets
     (e.g. dominant male)
  2) eat many very small
     ~~food~~ items
     grass root
- operational definition of attn:



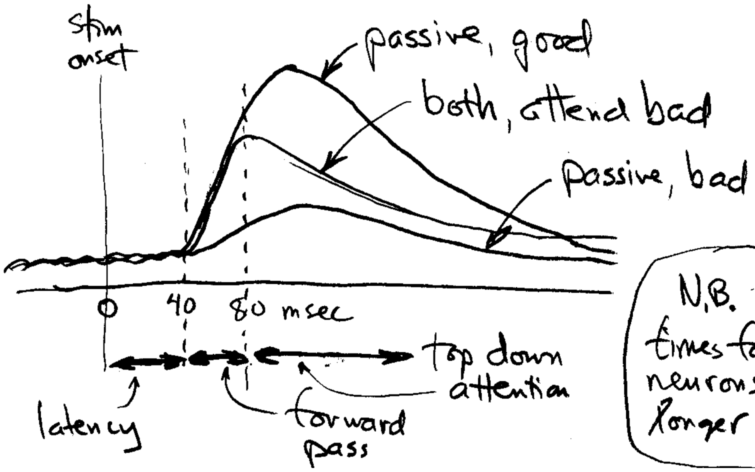
- much harder to train
  monkeys than undergraduates ☺



1) attention 'rescues' response to
   attended stimulus from the effect
   of stimulus competition

2) interpretation is ambiguous ;
   [attention to spatial location ?
   attention to features ?

3) some evidence that there
   is a "feedforward pass" before
   top down attentional filter kicks in



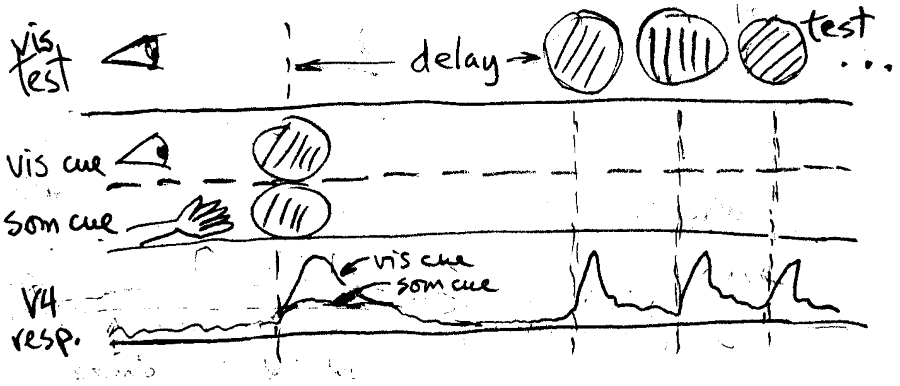
N.B. equiv.
times for human
neurons 2x
longer than here

Conclusion

- attention to location and
  features is present to
  some degree in <u>every</u>
  visual area (and somato.
  & aud areas, too)

Haenny & Mansell

somatosensory-visual & visual-visual



test responses

① test stim

cue

<table>
<tr><th></th><th>|||</th><th>///</th><th>≡</th><th>\\\</th></tr>
<tr><td>|||</td><td></td><td>✓</td><td></td><td></td></tr>
<tr><td>///</td><td></td><td>✓</td><td></td><td></td></tr>
<tr><td>≡</td><td></td><td>✓</td><td></td><td></td></tr>
<tr><td>\\\</td><td></td><td>✓</td><td></td><td></td></tr>
</table>

orientation-tuned

② test stim

cue

<table>
<tr><th></th><th>|||</th><th>///</th><th>≡</th><th>\\\</th></tr>
<tr><td>|||</td><td></td><td></td><td></td><td></td></tr>
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<tr><td>\\\</td><td></td><td></td><td></td><td></td></tr>
</table>

"cue-tuned"

③ test stim

cue

		✓	

both "cue"-tuned and orientation-tuned!

④ test stim

cue

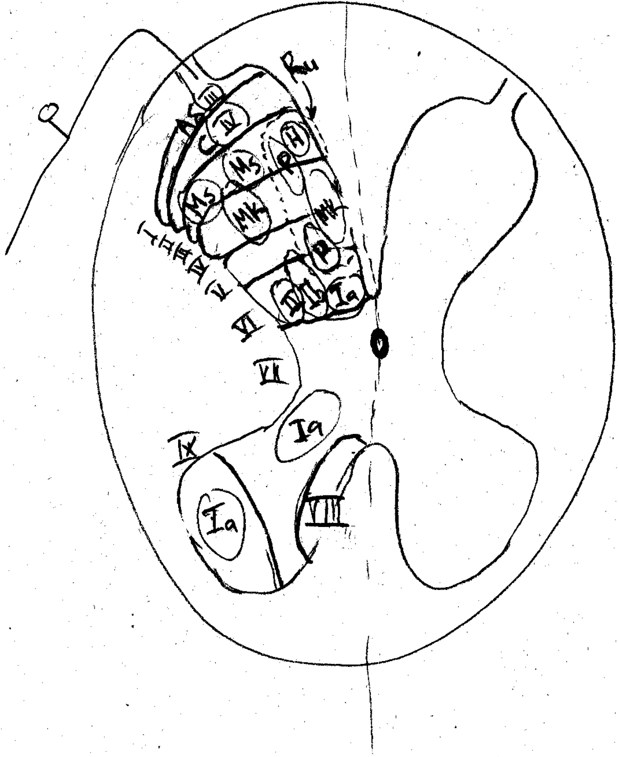
	✓		

"match cell"

Receptors

Unencapsulated

- heat
- cold
- pain
  - "x", C, sustained, small/unmyelinated
  - "y", Aδ, transient, large/myelinated
- slow stroking



Touch

Superficial

- Merkel disks — sustained (= slowly adapting)
- Meissner's corpuscles — transient (= rapidly adapting)

Deep

- Ruffini endings — sustained
- Pacinian corpuscles — transient

- hair follicle receptors — transient

Muscle receptors

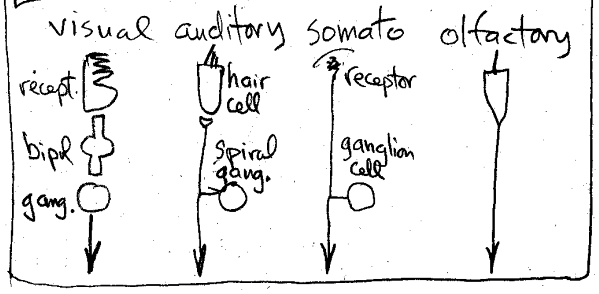
"Stretch" → length
↳ "ON" - only

- II spindles — sustained
- Ia spindles — transient
- Ib — Golgi tendon organs → force

pain topics

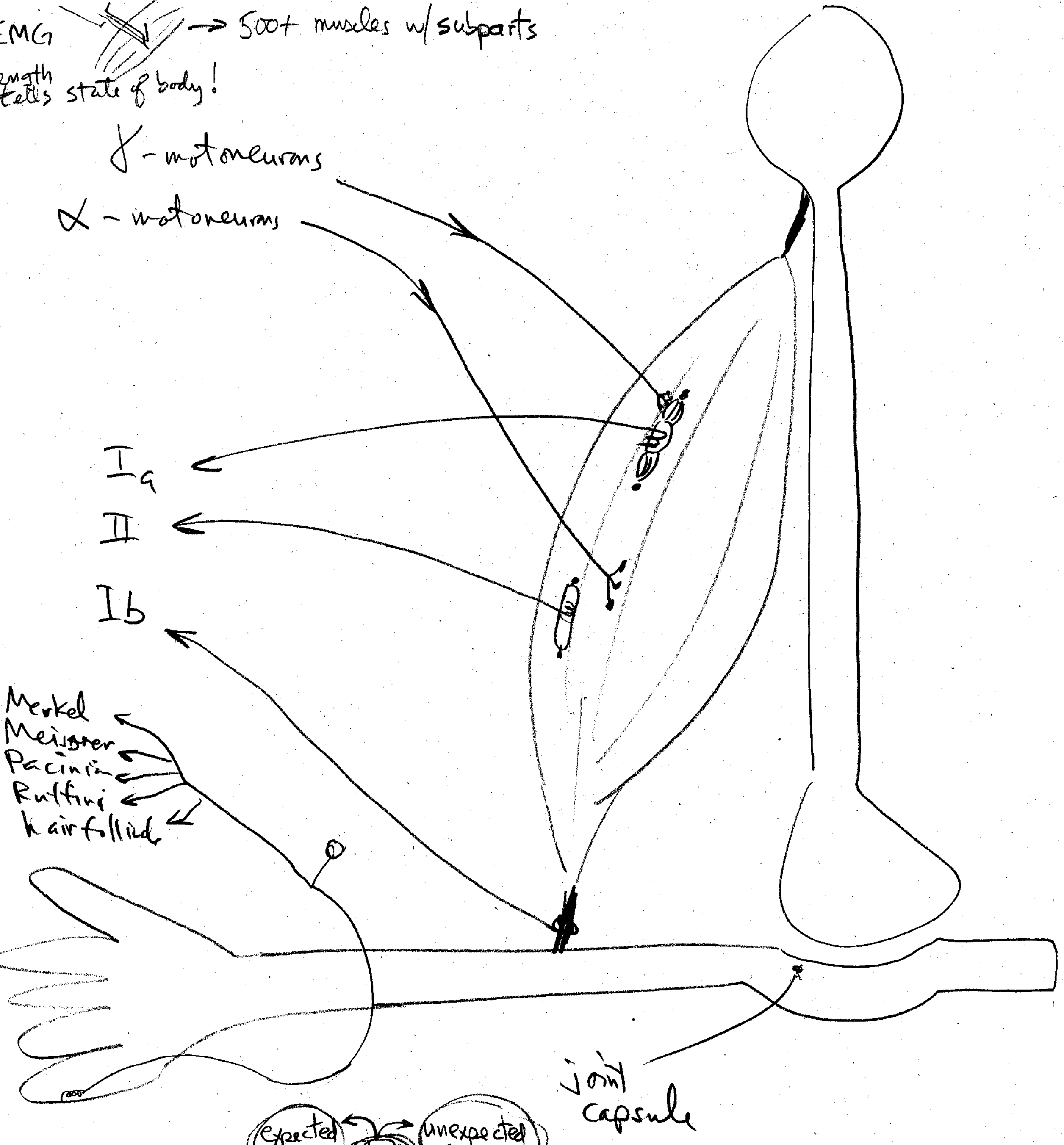
- no receptors a prob!
- cognitive pain ("good jam!")
- gating (e.g. machine shop)
- opiate - induced hyperalgesia
- opiate "don't care"

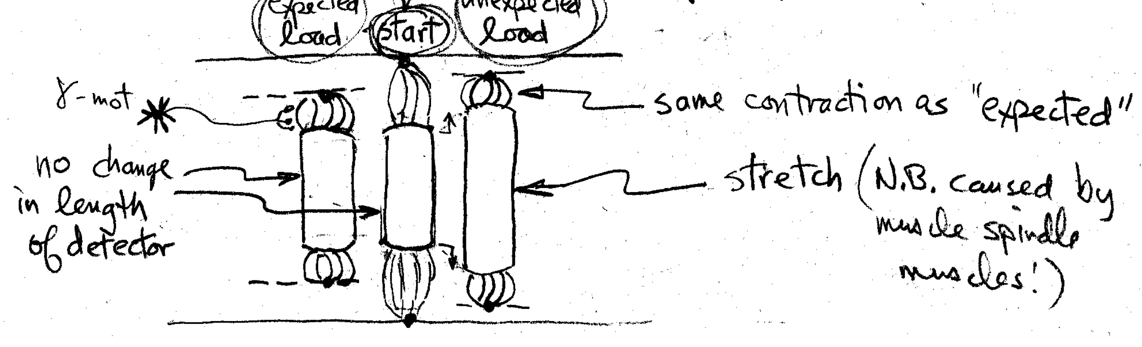
Intro



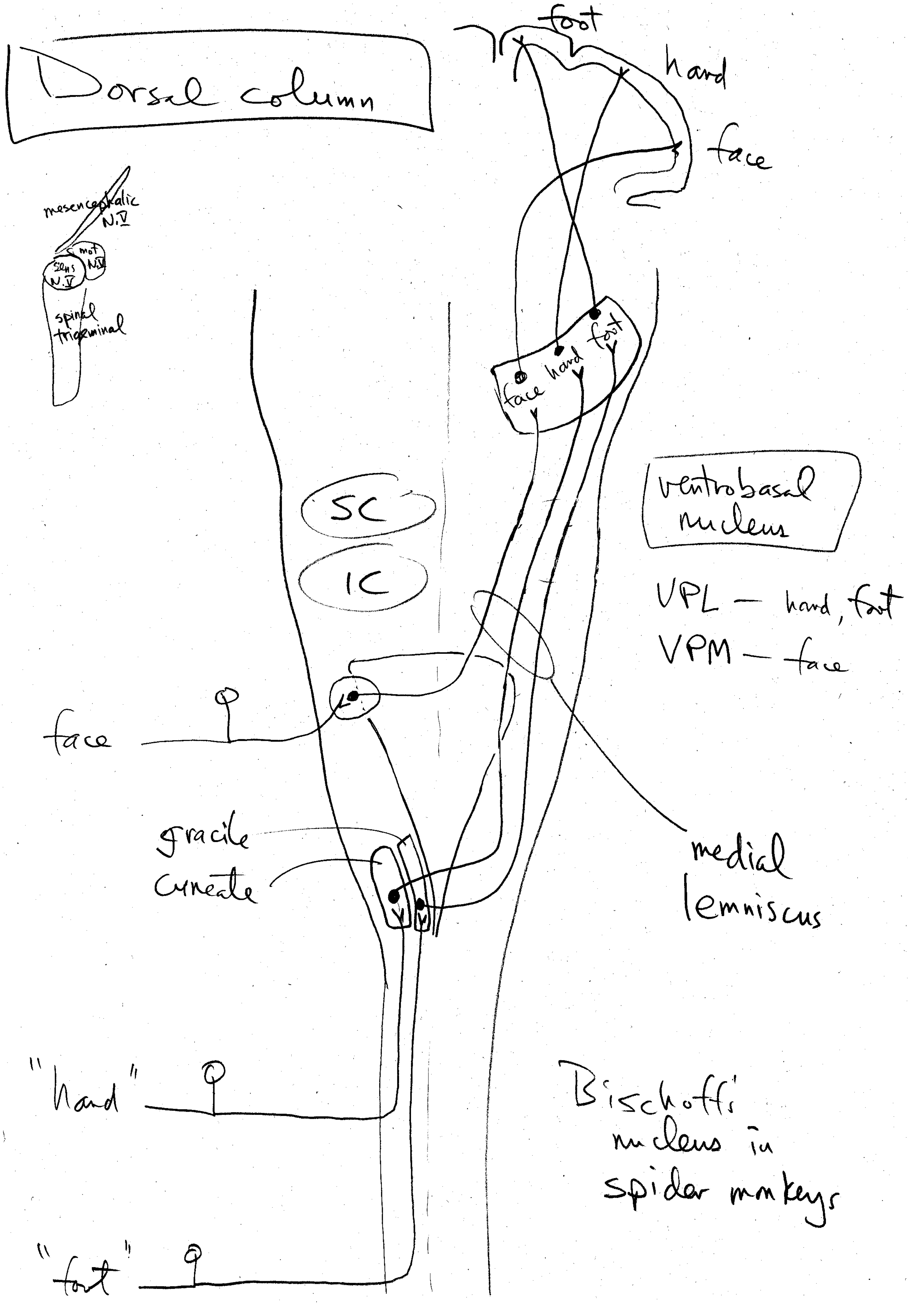
Co-contraction of α, γ motoneurons to detect deviation

[- EMG → 500+ muscles w/subparts
- length tells state of body!





Dorsal column

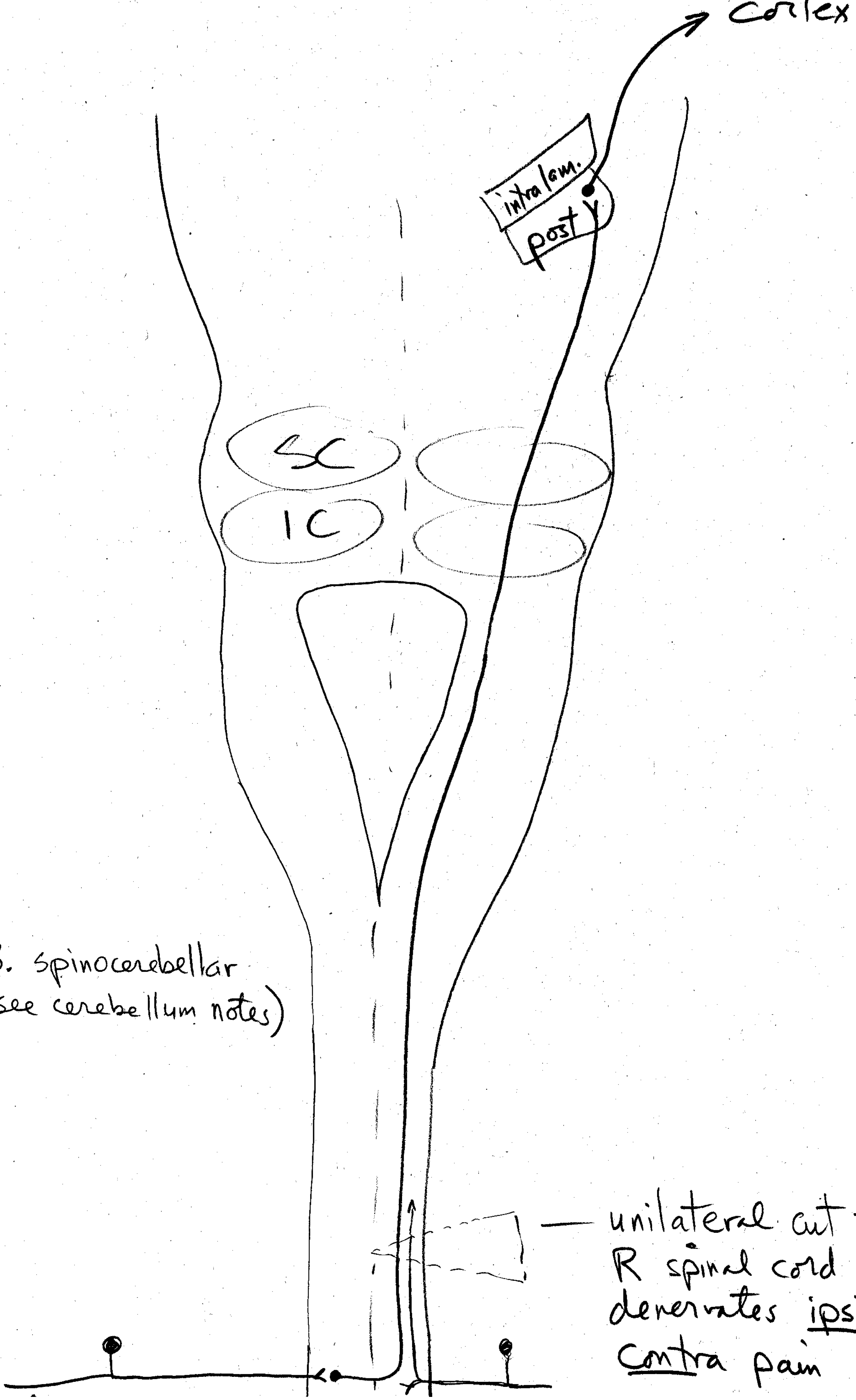


ventrobasal nucleus

VPL – hand, foot
VPM – face

Bischoff's nucleus in spider monkeys

Spinothalamic Pathway



- N.B. spinocerebellar (see cerebellum notes)

— unilateral cut to R spinal cord denervates ipsi touch contra pain

Somatosensory Cortex

visual maps

1) sm move ctx → sm move vis field R.F.

2) sm move vis field → sm move ctx
→ jump (eg. V2 HM)

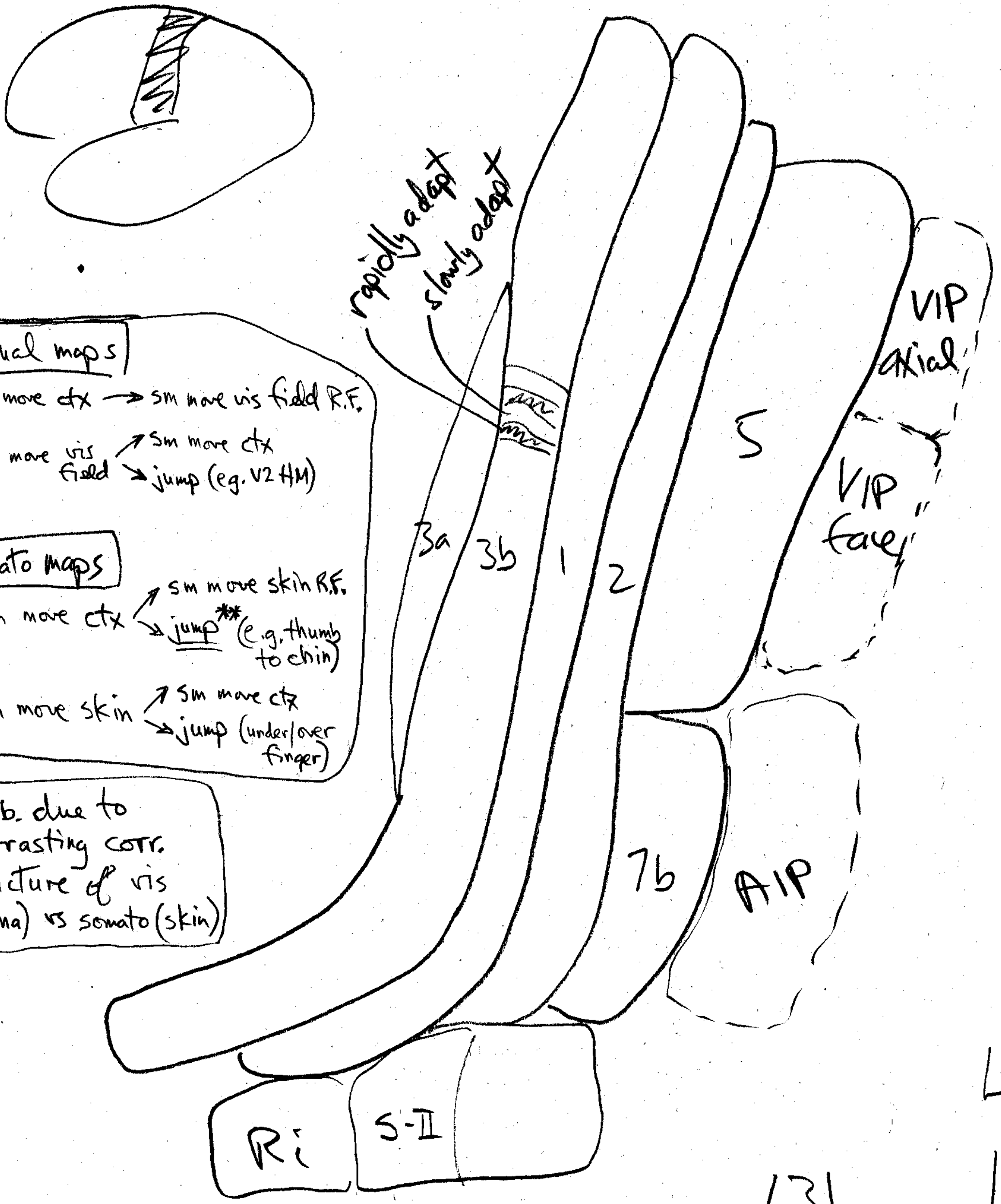
diff'l

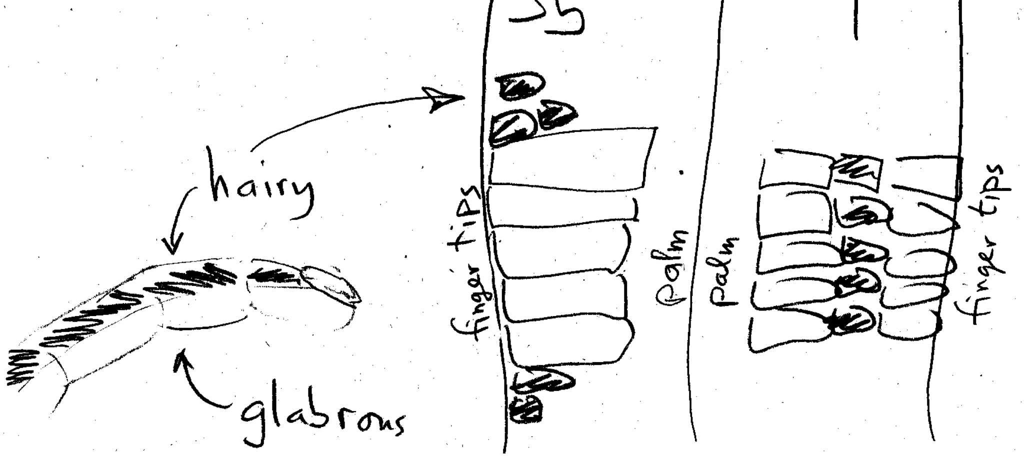
somato maps

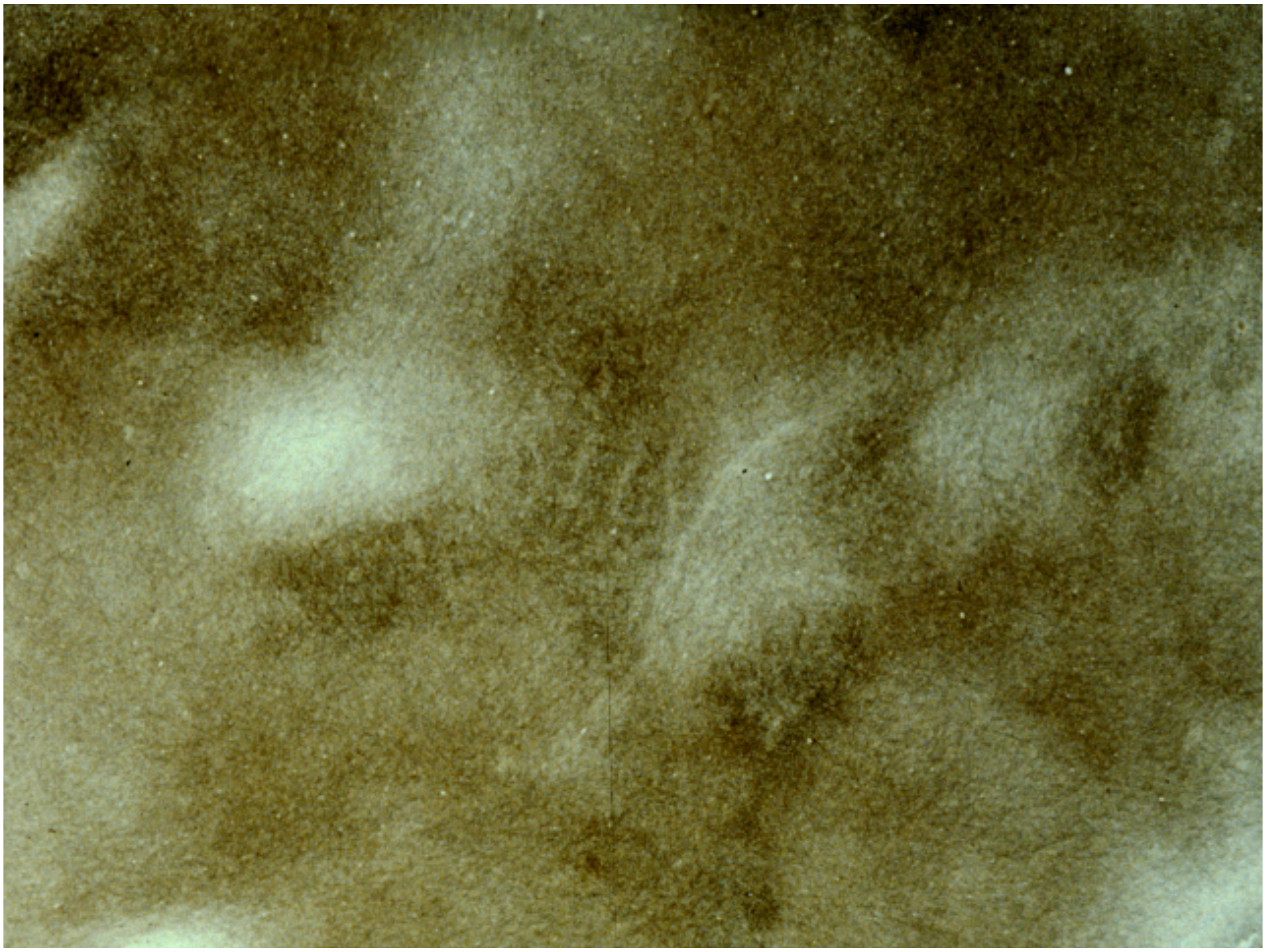
1) sm move ctx → sm move skin R.F.
→ jump** (e.g. thumb to chin)

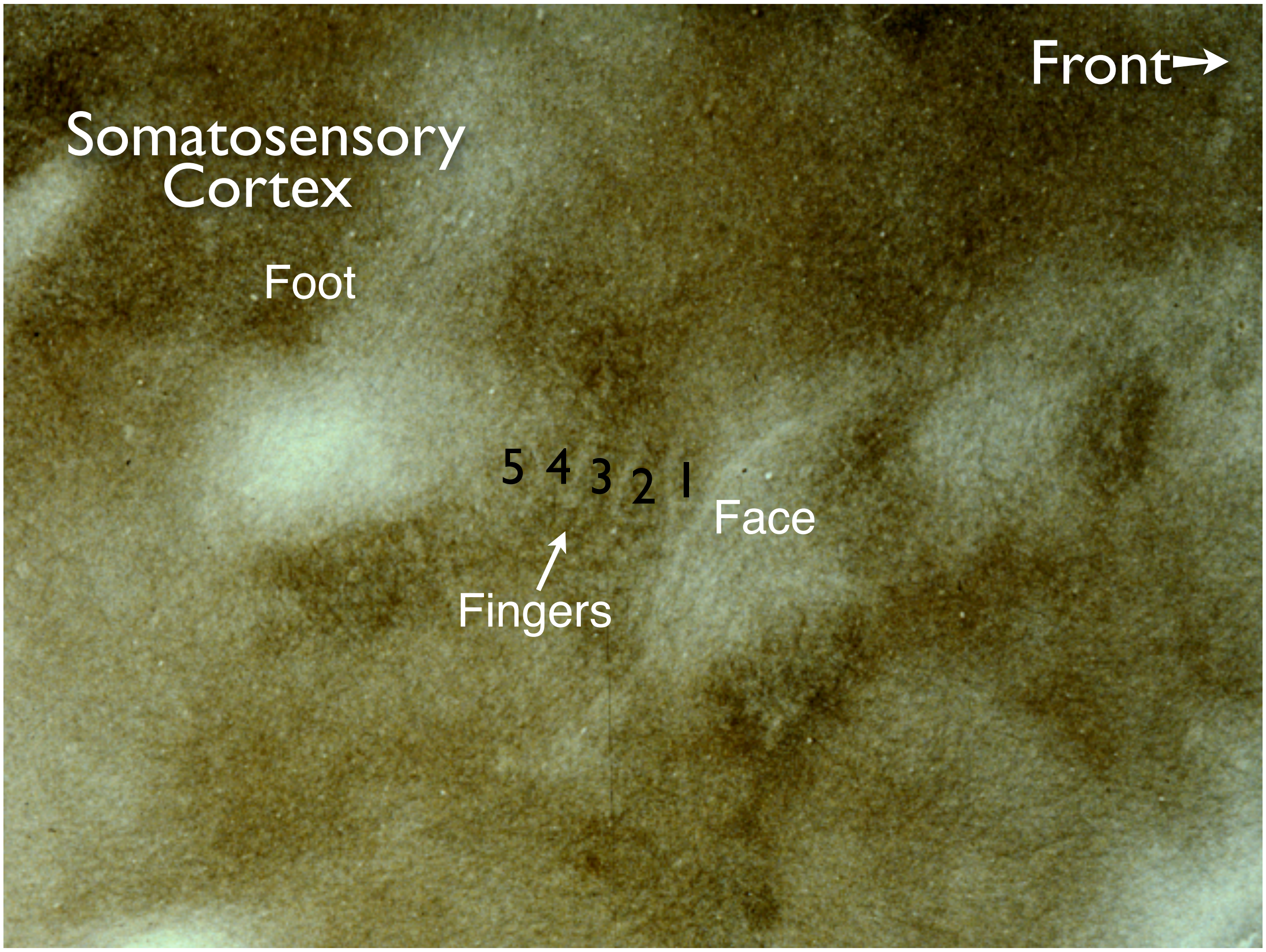
2) sm move skin → sm move ctx
→ jump (under/over finger)

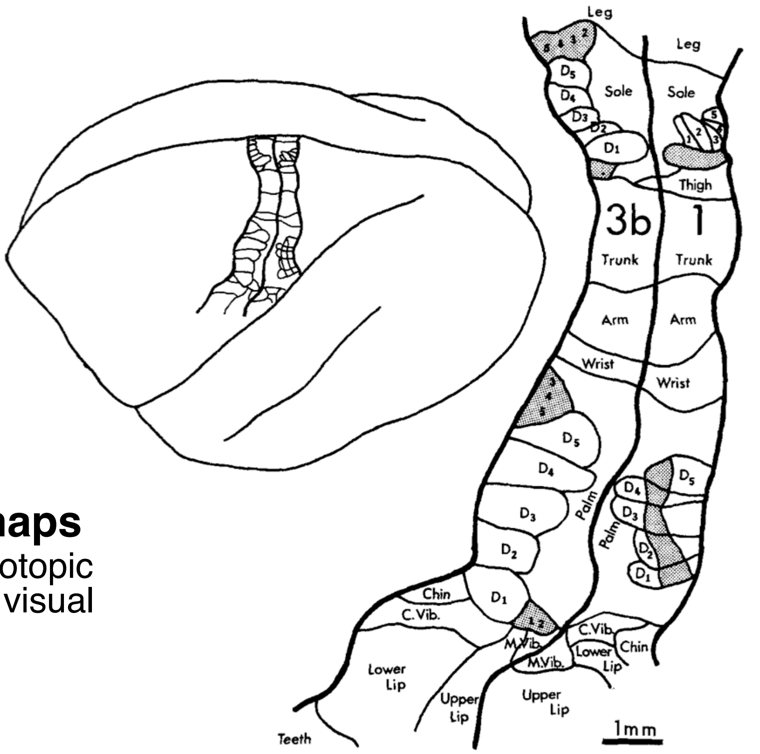
prob. due to contrasting corr. structure of vis (retina) vs somato (skin)



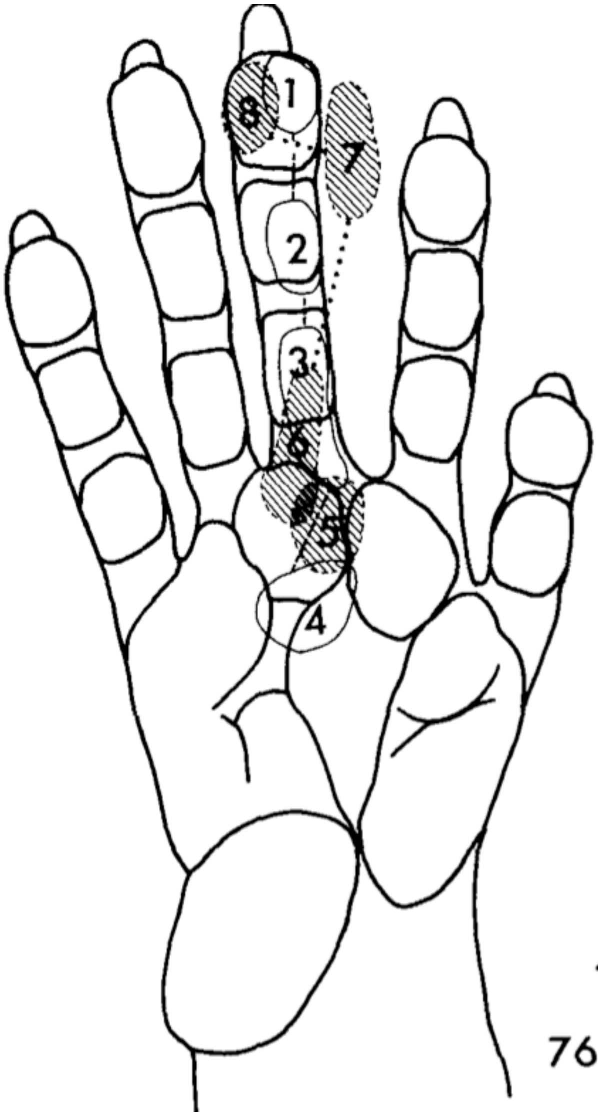


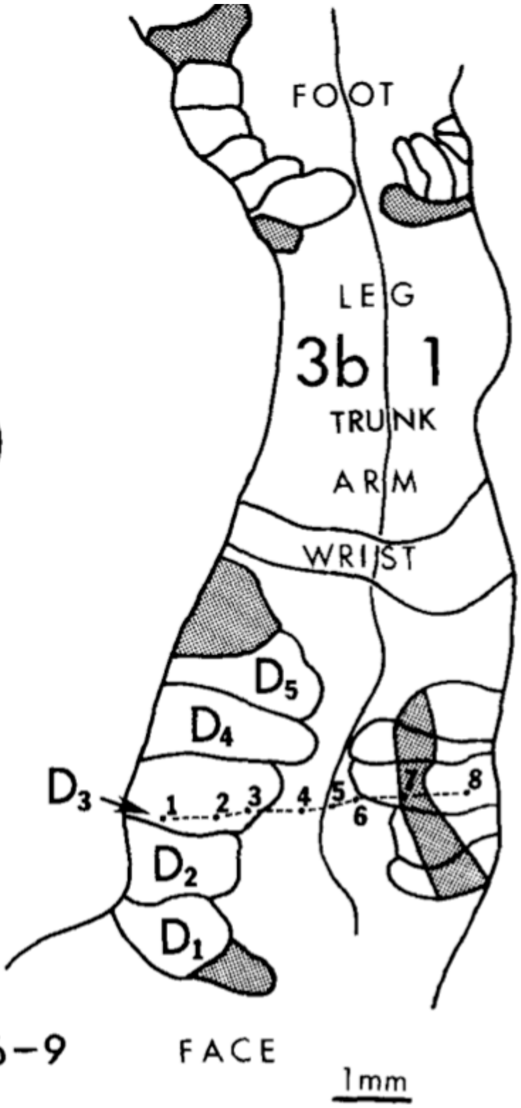



Front→
Somatosensory Cortex
Foot
5 4 3 2 1
Face
Fingers



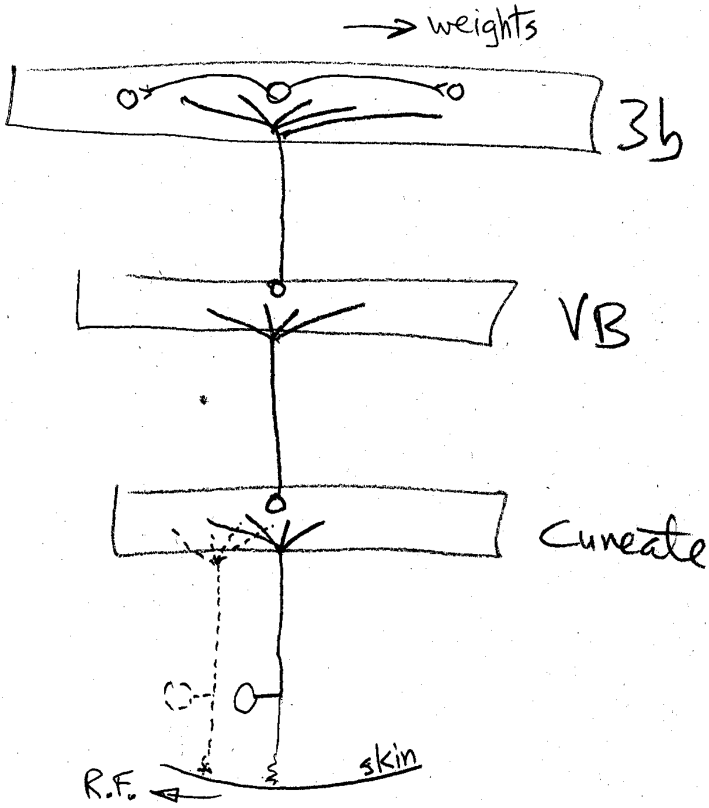
2D maps
somatotopic
versus visual





Plasticity Expts

- denervate radial
- transplant patch
- syndactyly
- just stim — cf. aud. Allard
- Silver Spring monkeys — face invades hand 1+ cm



- turn up terminals on R
- moves RF to L

- no plasticity corticocortical!!
- no plasticity VB → 3b!!

General Principles — Auditory

A) sensory surface is 1D

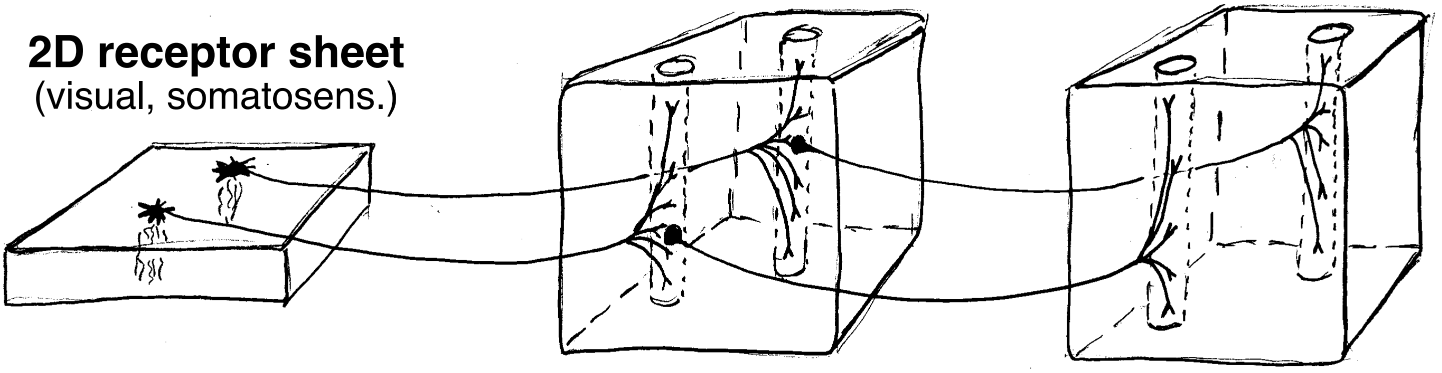
B) connection difference — convergent-divergent within isofrequency domains

C) fine-grained temporal info

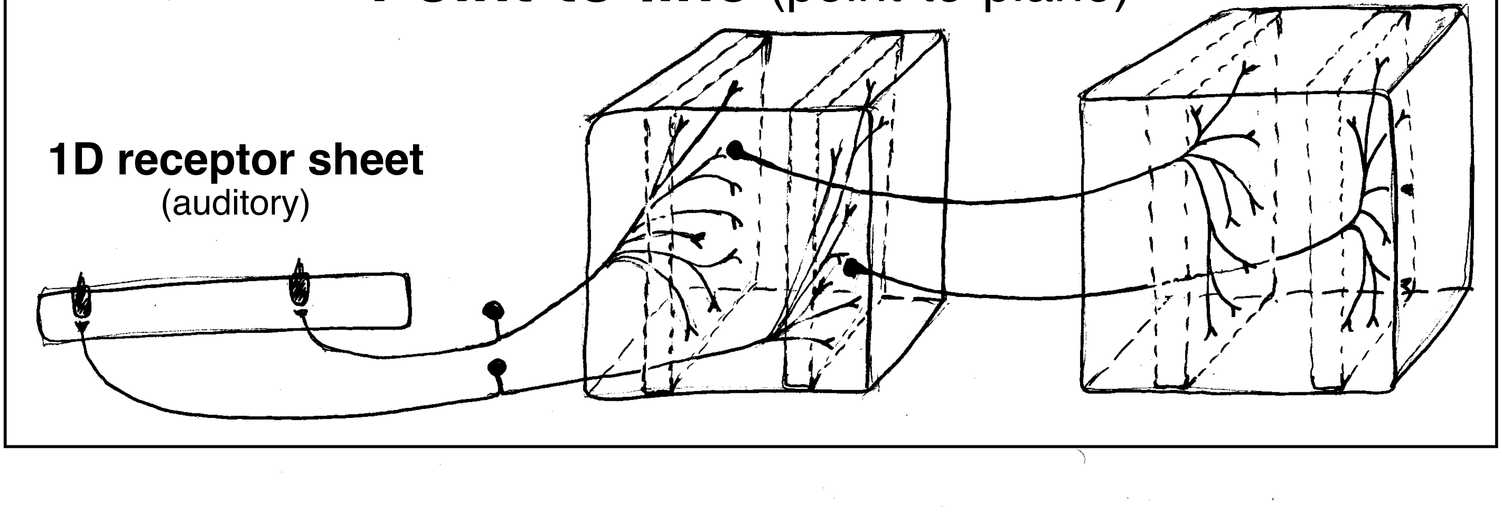
D) no "direct" rep of stim location

E) cortex is further away (6th order vs. 4th order)

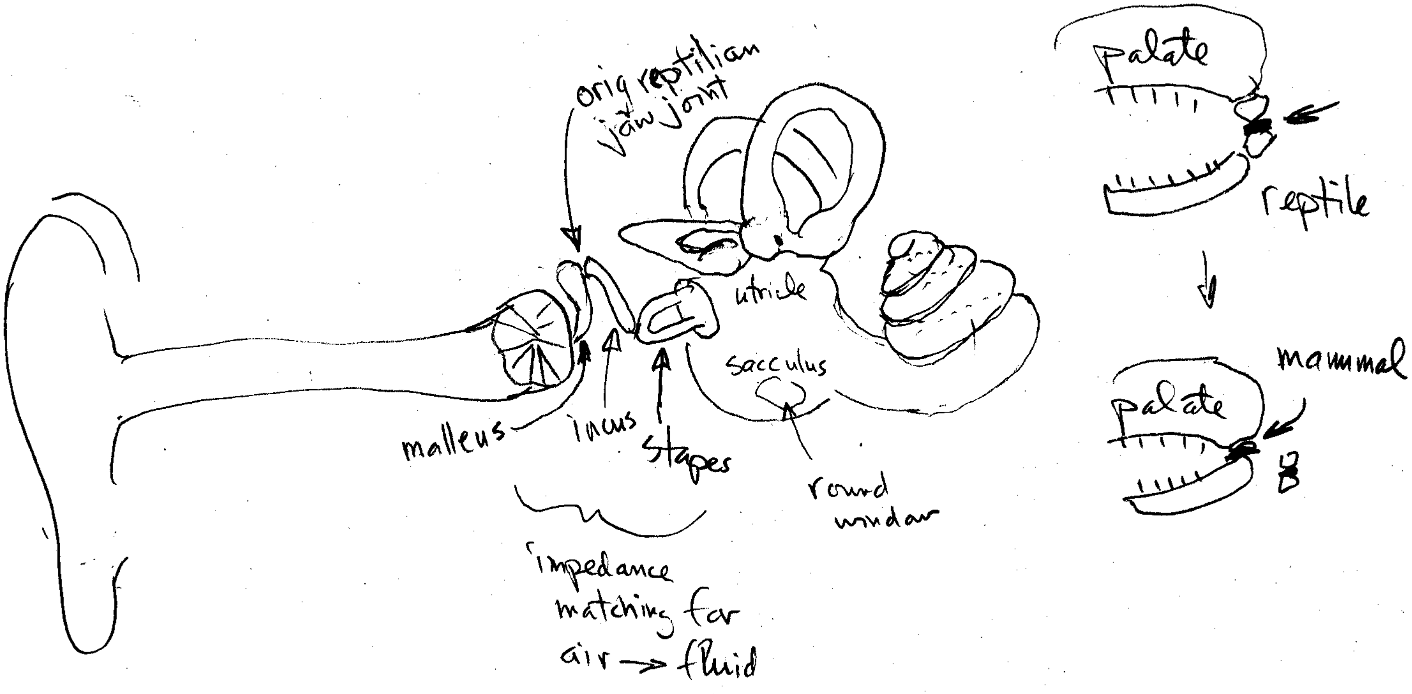
Point-to-point (point-to-line)



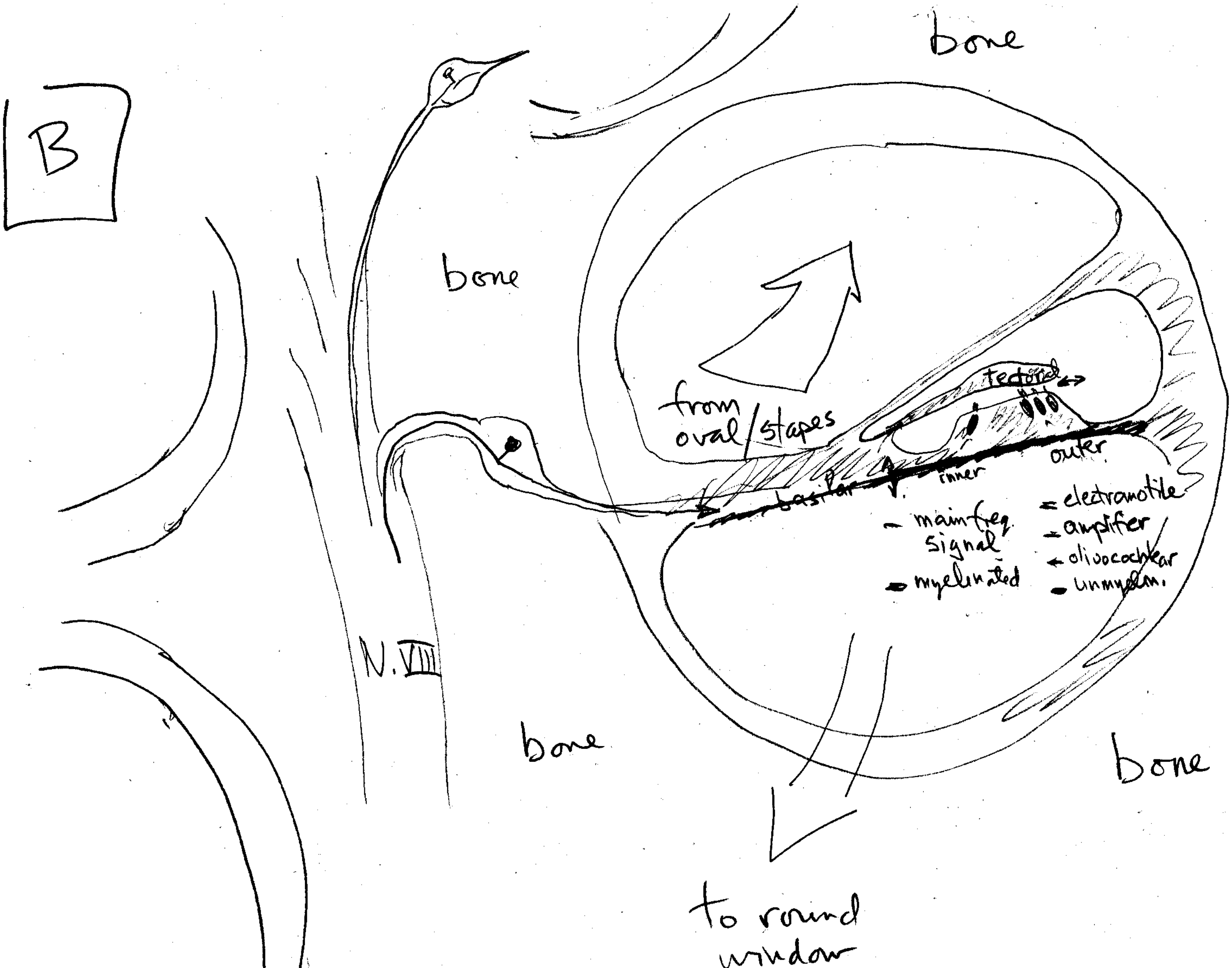
Point-to-line (point-to-plane)



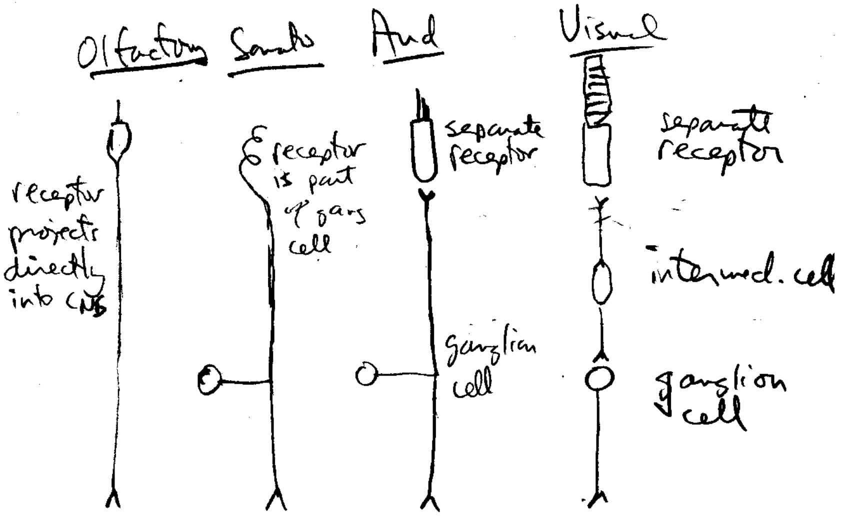
A


orig reptilian jaw joint
utricle
malleus
incus
stapes
sacculus
round window
impedance matching for air → fluid
palate
reptile
mammal
palate

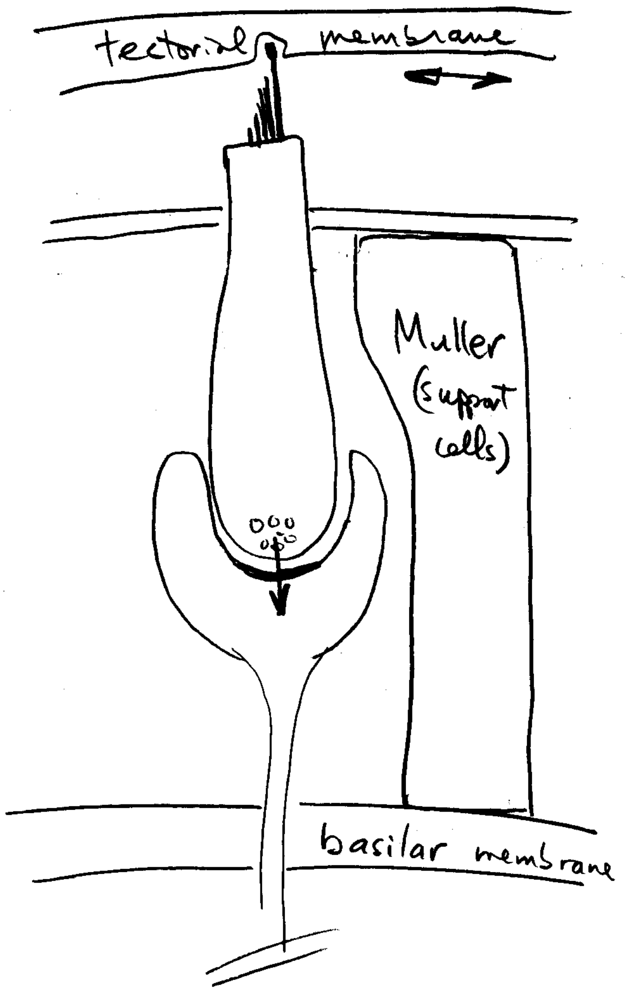
B

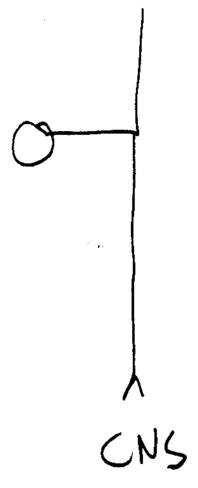

bone
bone
from oval/stapes
tectorial
outer
inner
basilar
- main freq. signal
- myelinated
- electromotile
- amplifier
- olivocochlear
- unmyelin.
N. VIII
bone
bone
to round window

C Receptors – Auditory



Hair cells

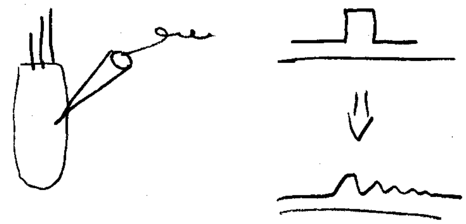




How hair cells become frequency selective

1) gradient in stiffness of basilar membrane
→ max in traveling wave

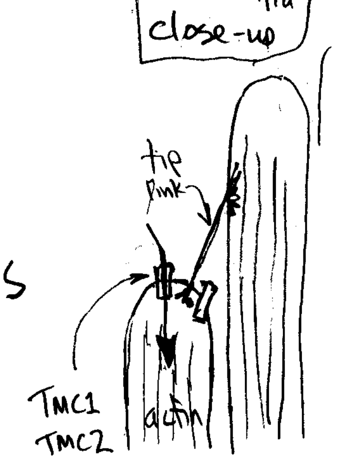
2) electrical tuning

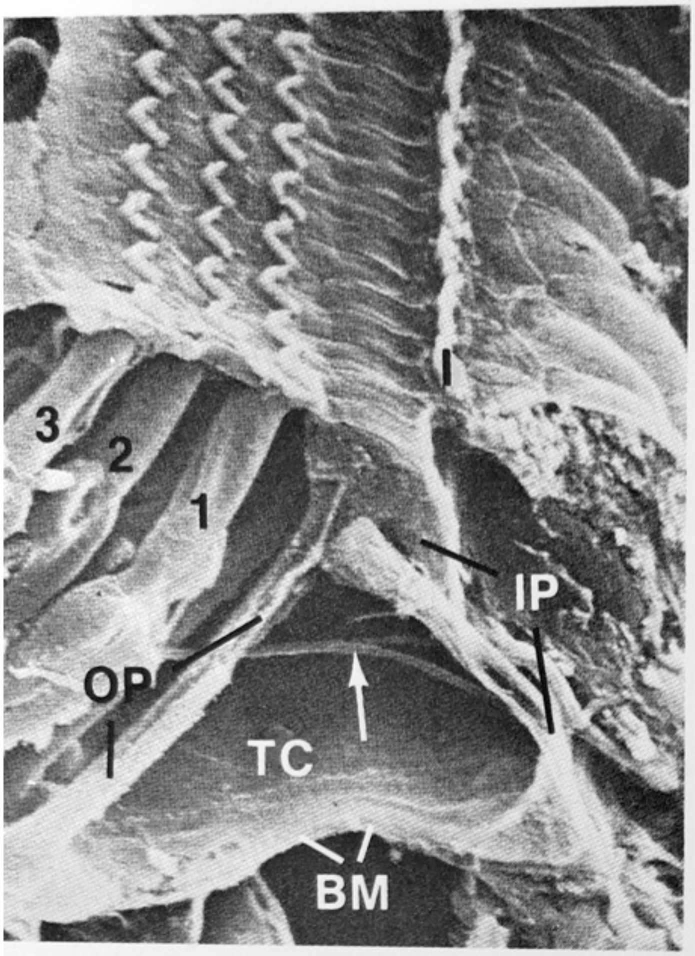


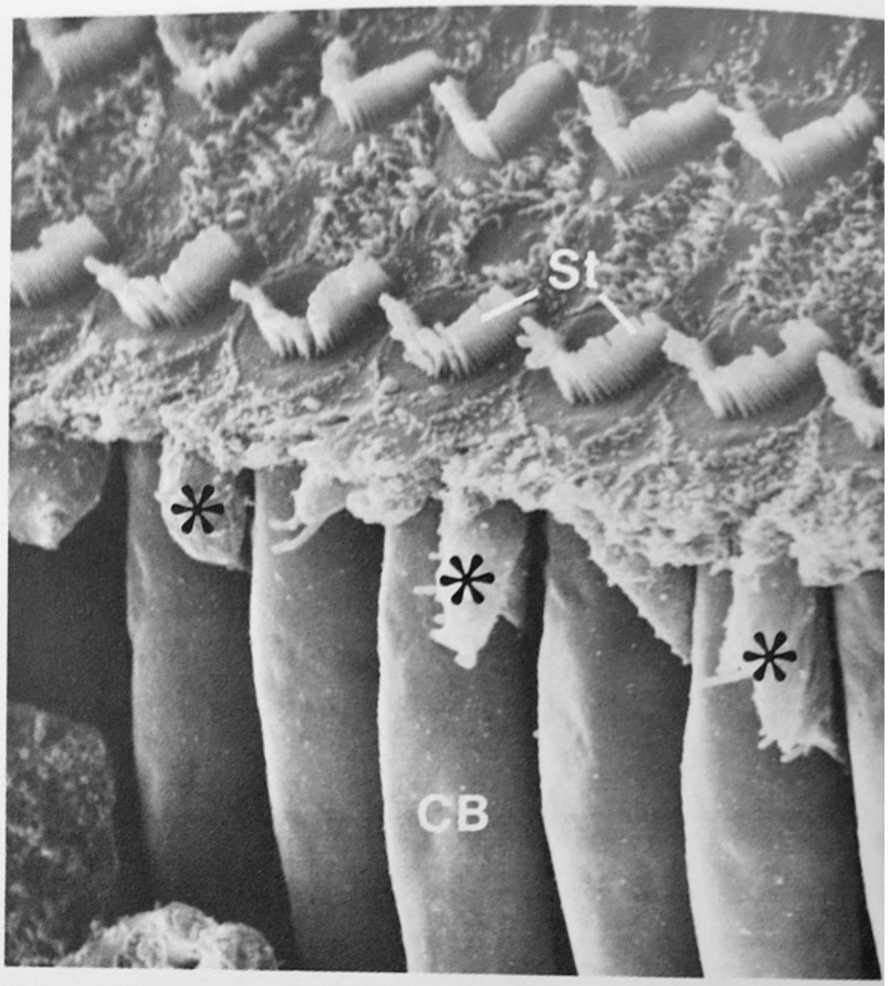
3) mechanical tuning of cilia base

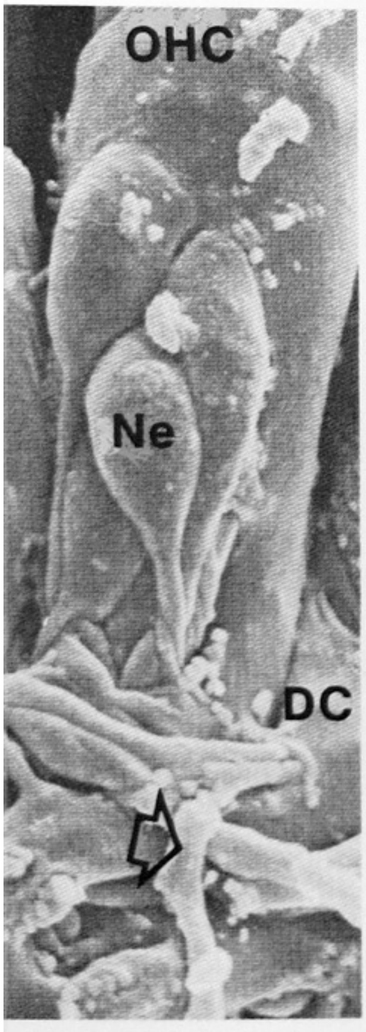
4) active physical responce to sound

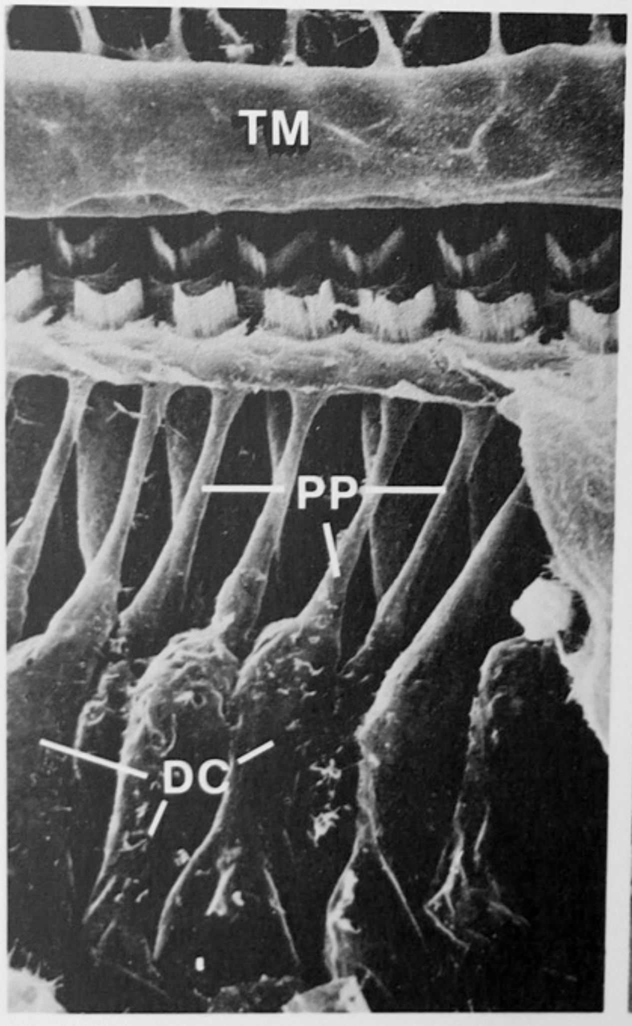
stereocilia close-up

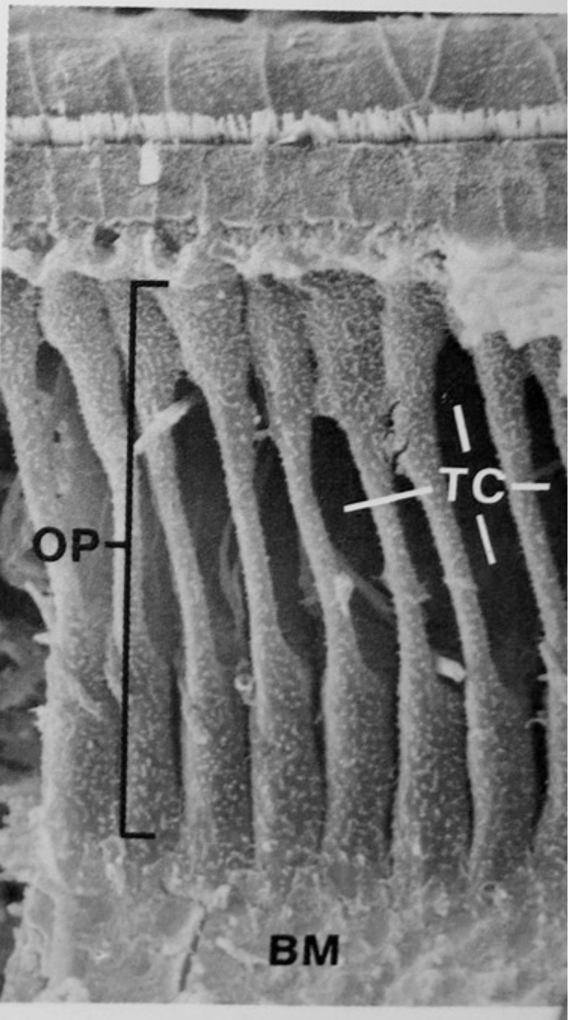


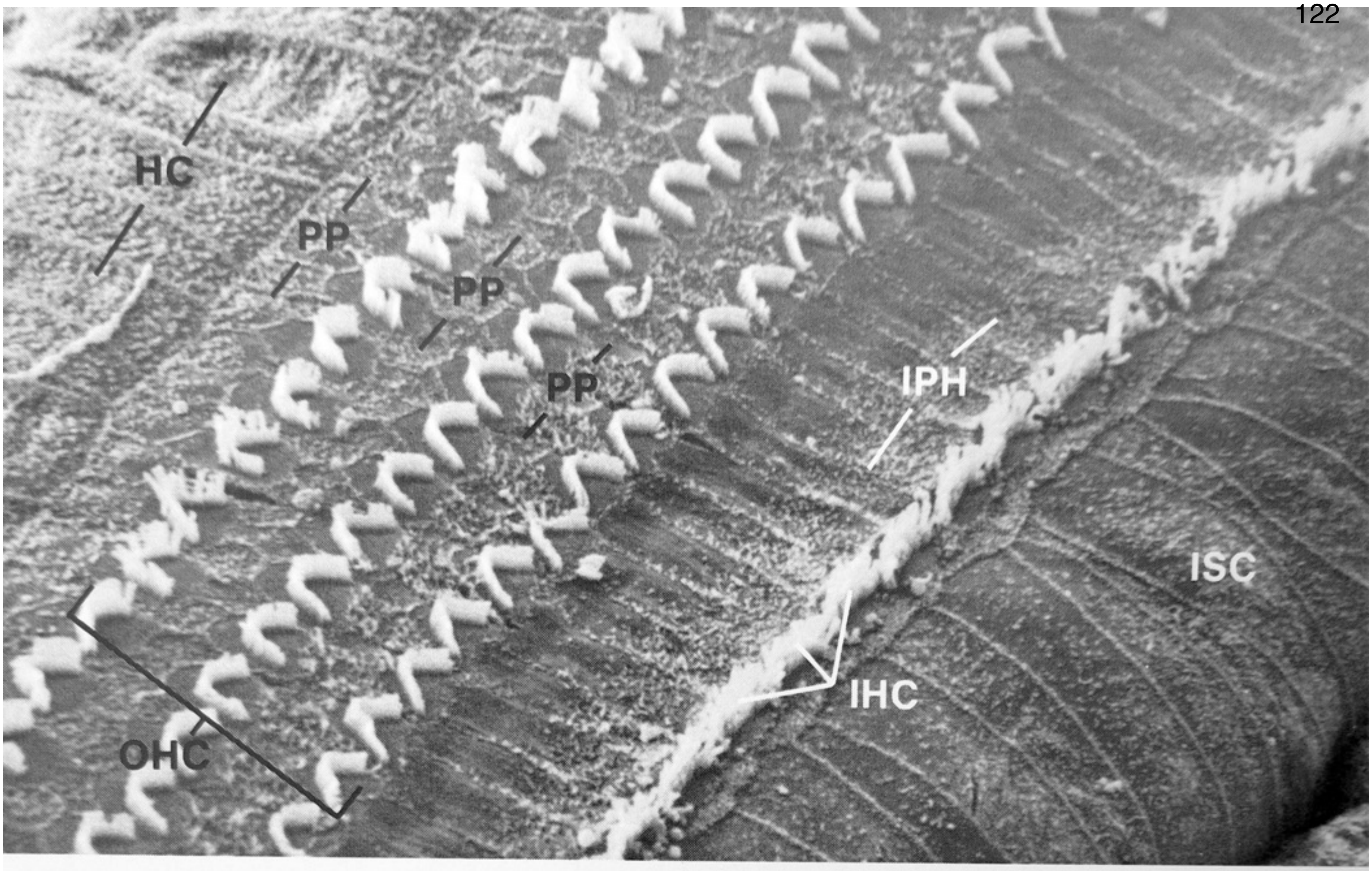

3
2
1
I
IP
OP
TC
BM

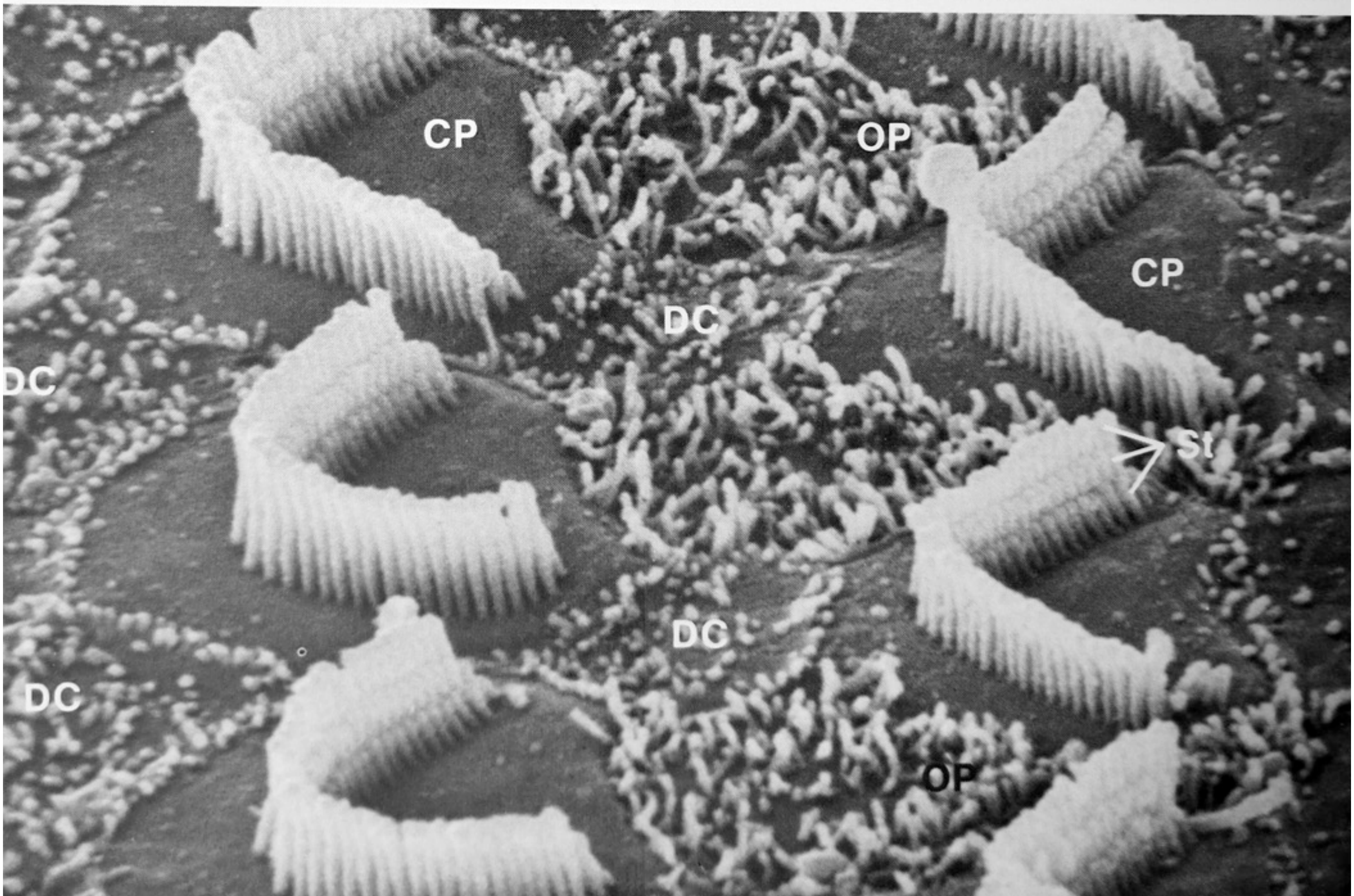

St
*
*
*
CB

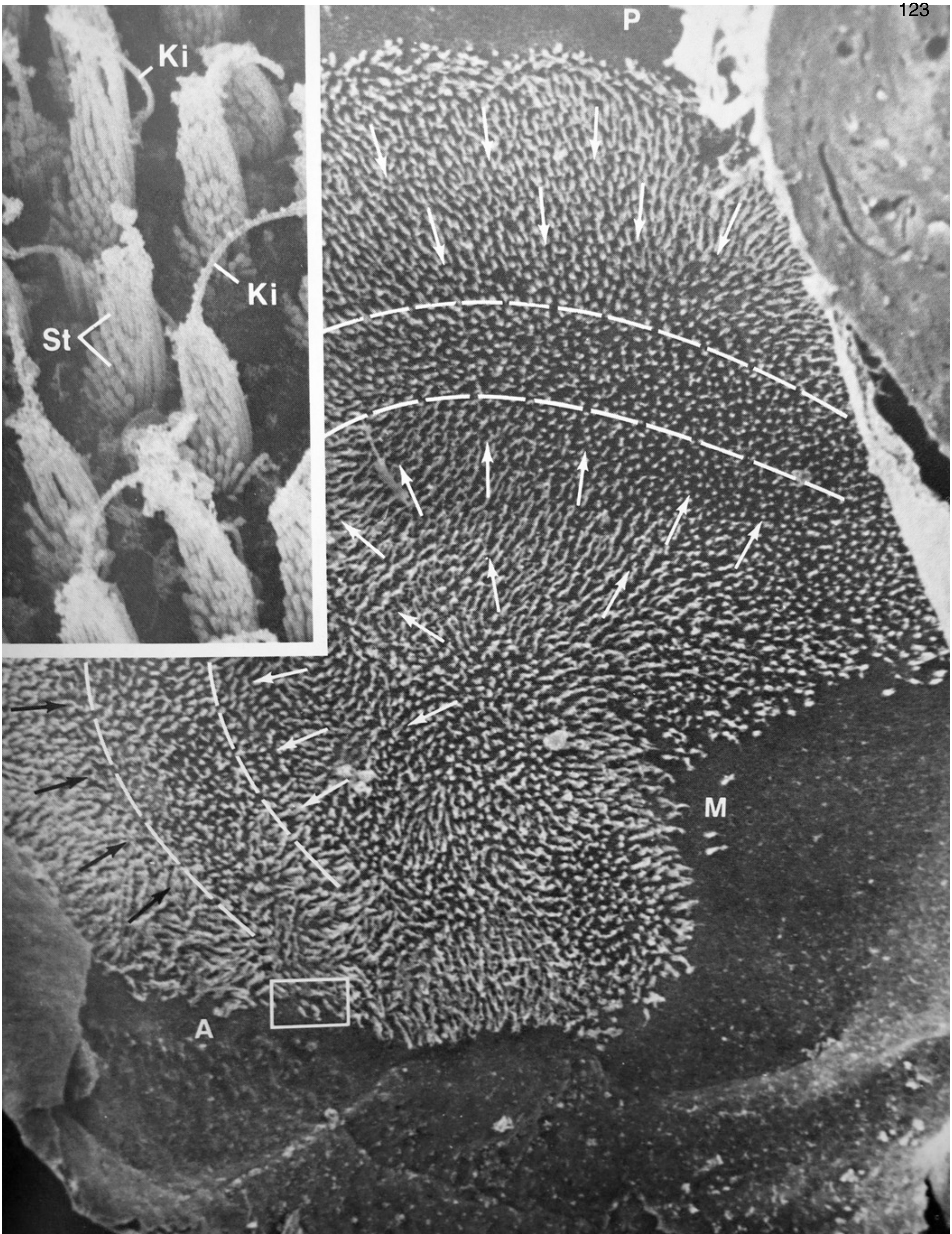

OHC
Ne
DC


TM
PP
DC


OP
TC
BM

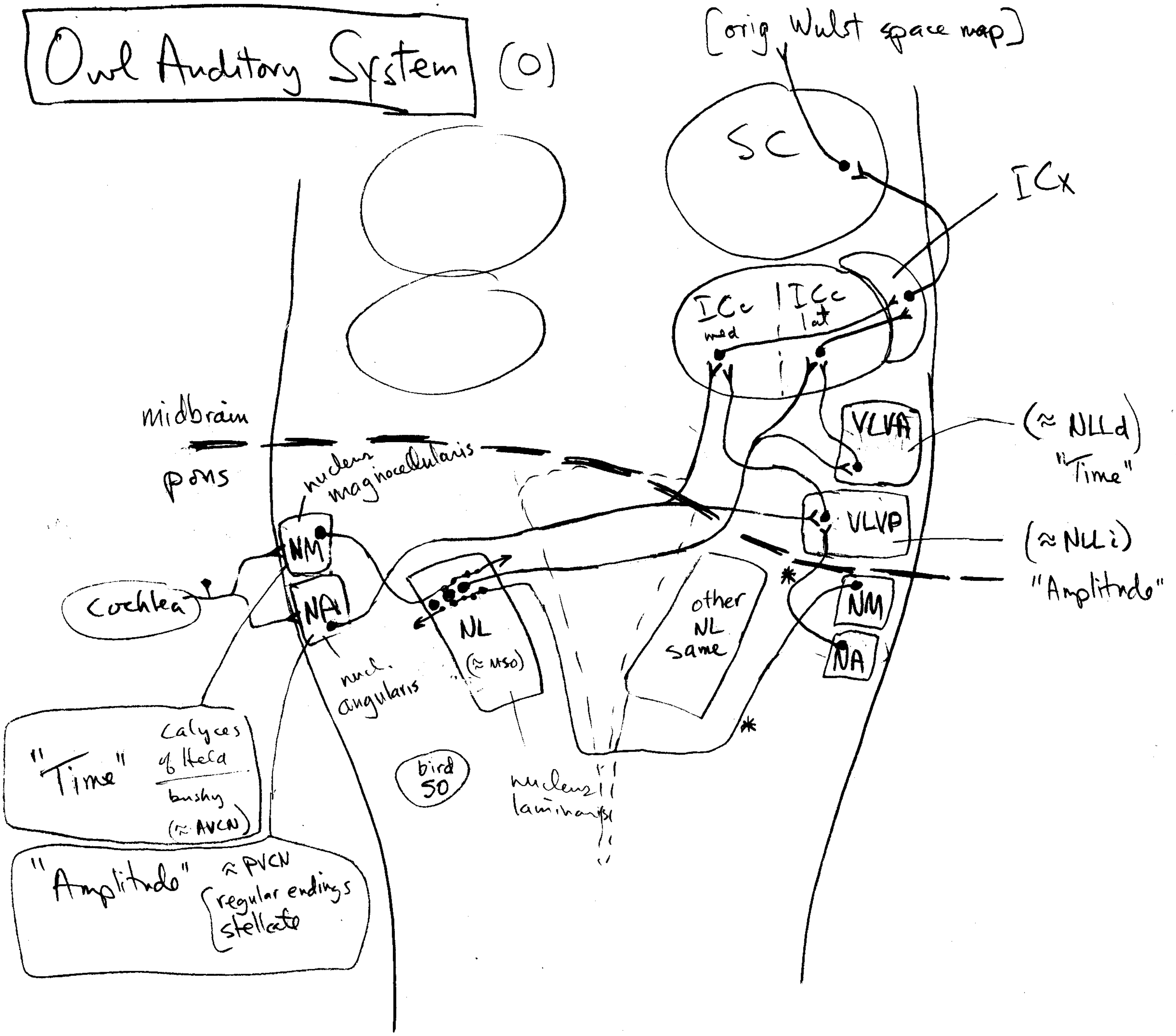

HC
PP
PP
PP
IPH
ISC
IHC
OHC


CP
OP
CP
DC
DC
St
DC
DC
OP


Ki
Ki
St
P
M
A

#11

Owl Auditory System (0)

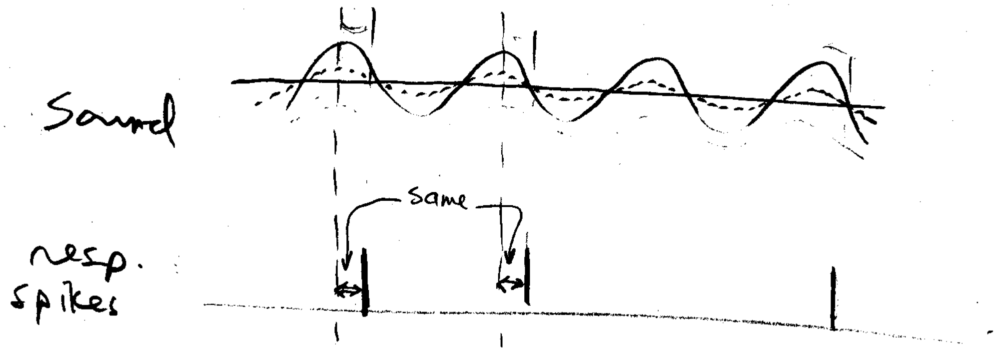


*breaks rule of Sereno

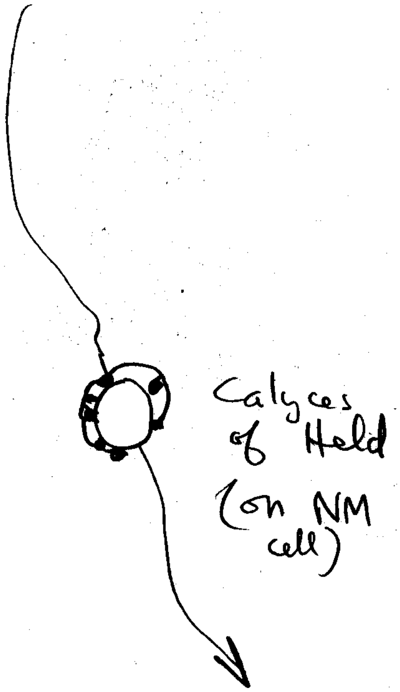
Owl Physiology (1)

intro to sound <
- frequency
- amplitude
- phase

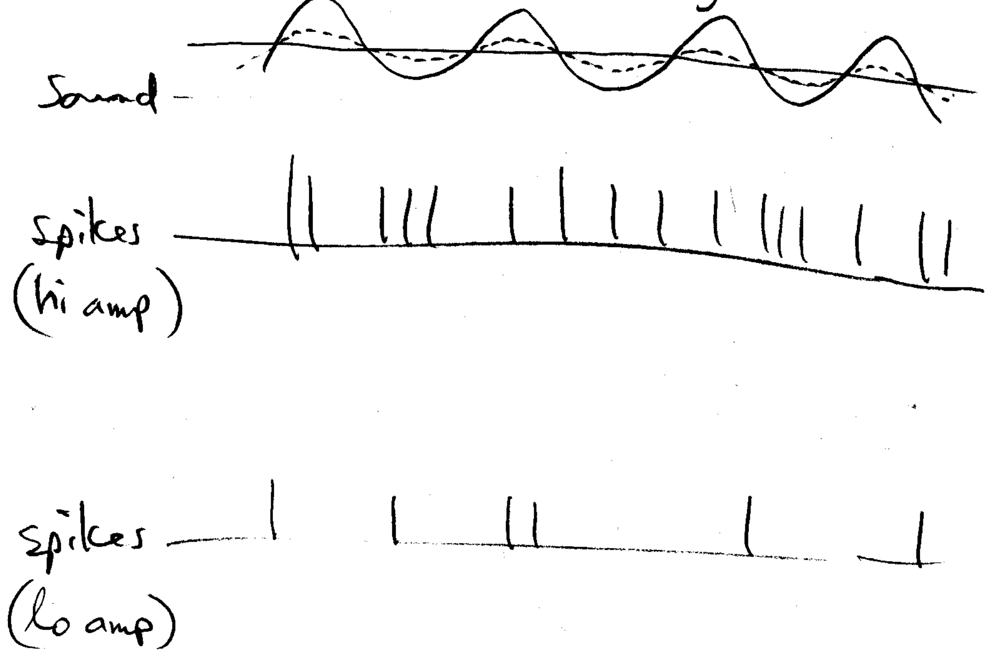
NM — phase-locked

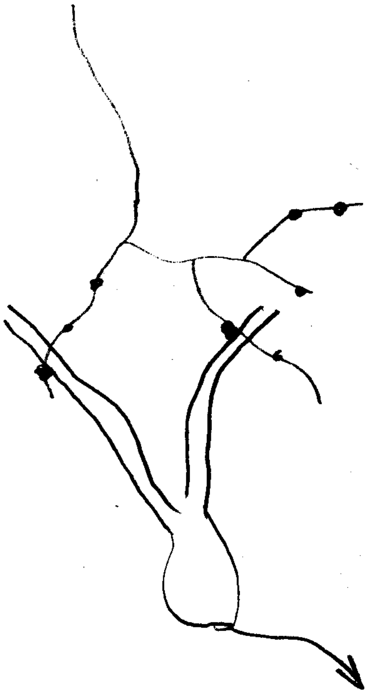


↳ also: phase delay not ampl.-sens

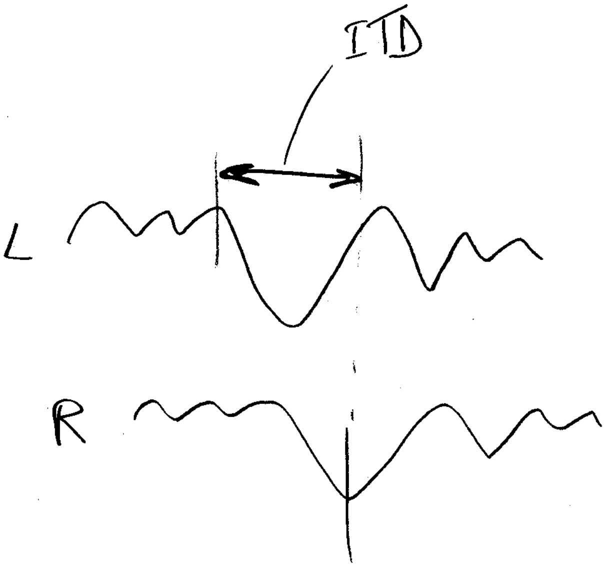


NA — amplitude coding



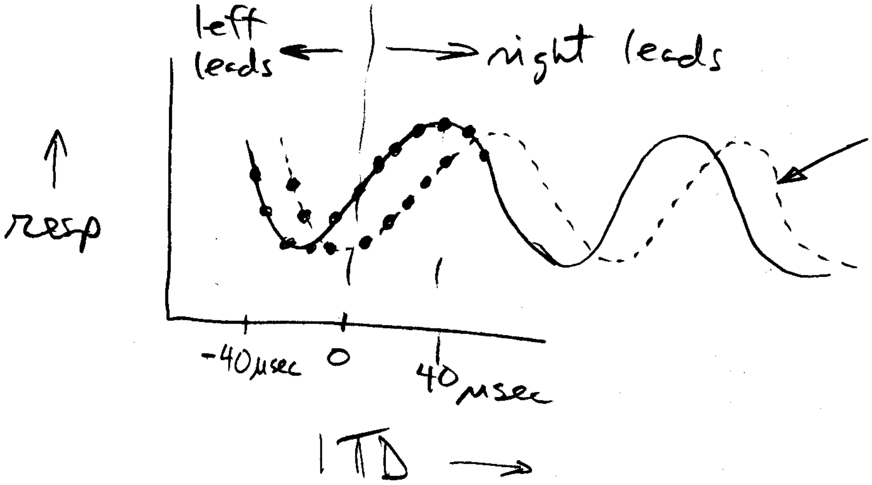


Owl Physiology (2)



NL (≈ MSO)

- first binaural
- ITD's

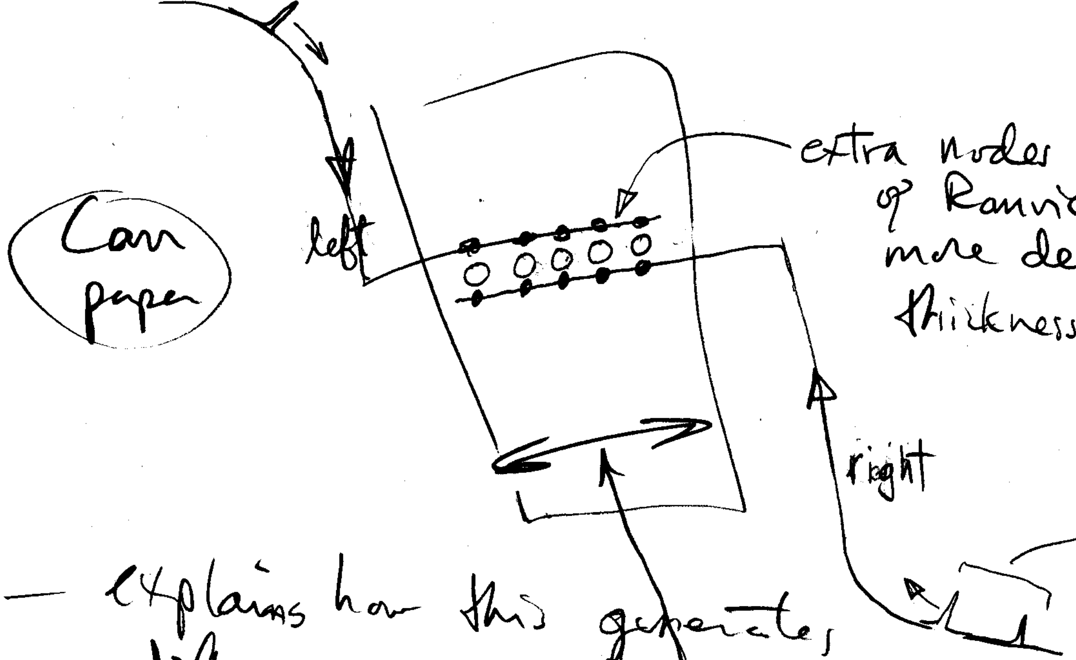


what a different characteristic delay looks like at same freq

---

VLVP (≈ NLLi)

How diff. char delays start in NL



Carr paper

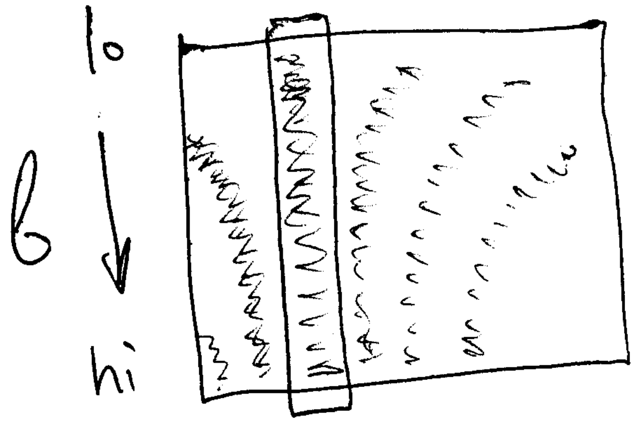
extra nodes of Ranvier for more delays through thickness of nucleus

2 spikes from this NM, 1 from other NM

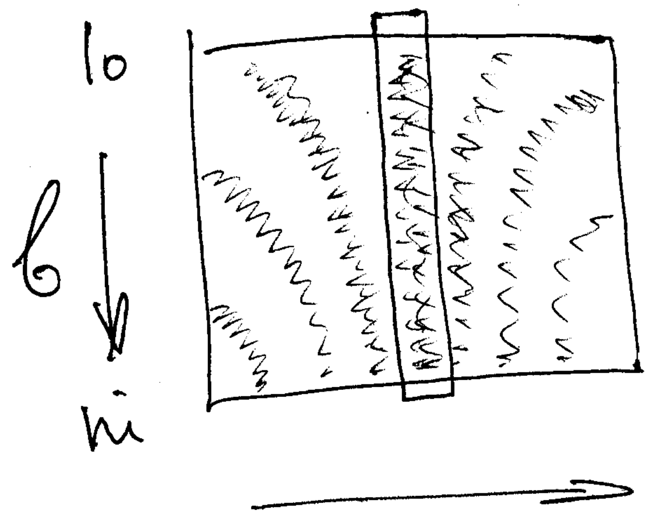
— explains how this generates diff. char. delays

Owl Physiology (3½)

Patterns in ICc lat w/white noise & different ITD's



– white noise at one ITD



– white noise at a different ITD

char. delay
(same as ITD if summed across f)

– this is view of one ITD across space

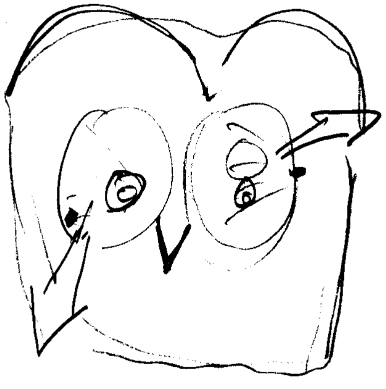
versus previous:

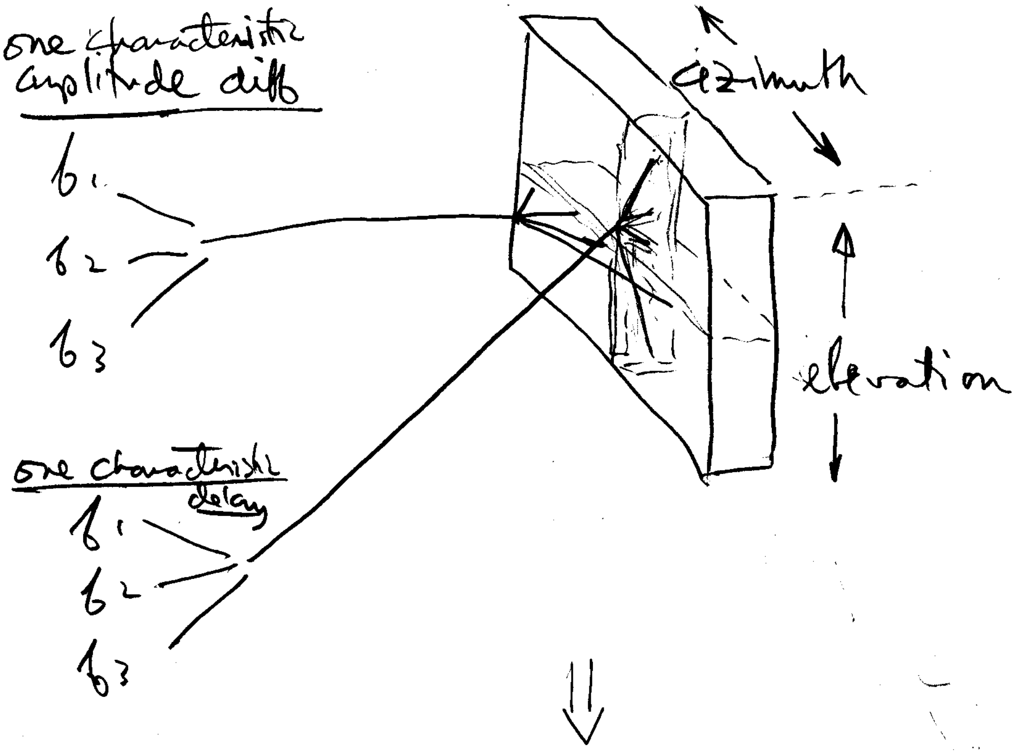
graph of many ITD's for one NL/ICc lat neurons

Owl Physiology (4)

ICx

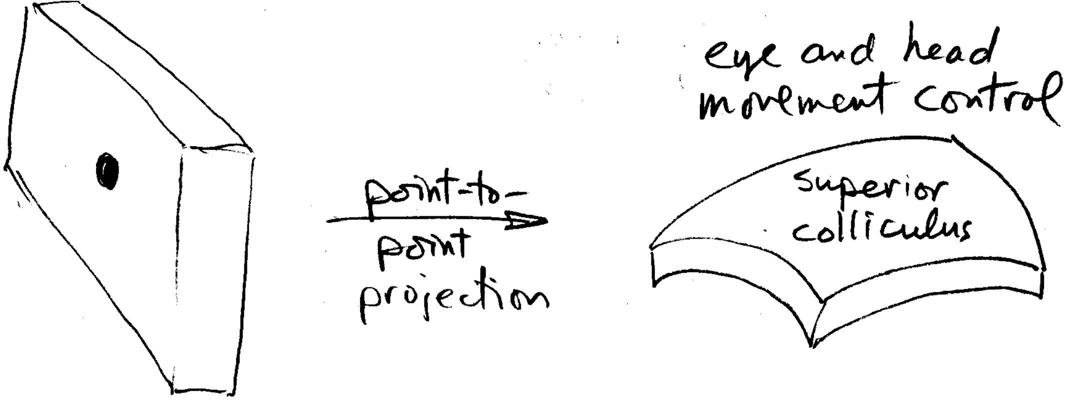
- space map
- amplitude vs. true delay



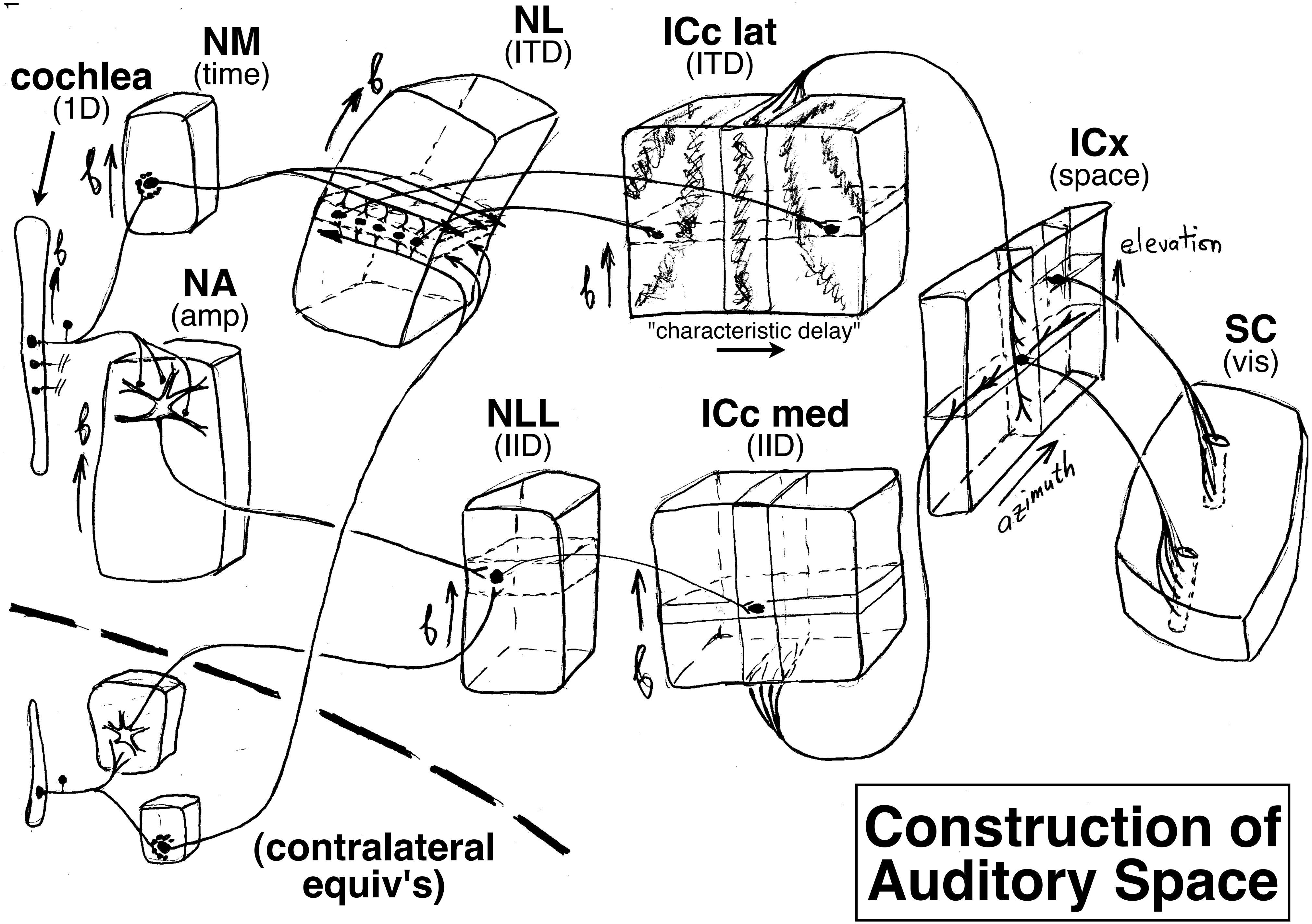


voilà!
a space map

- activation in response to white noise coming from a small speaker



Construction of Auditory Space



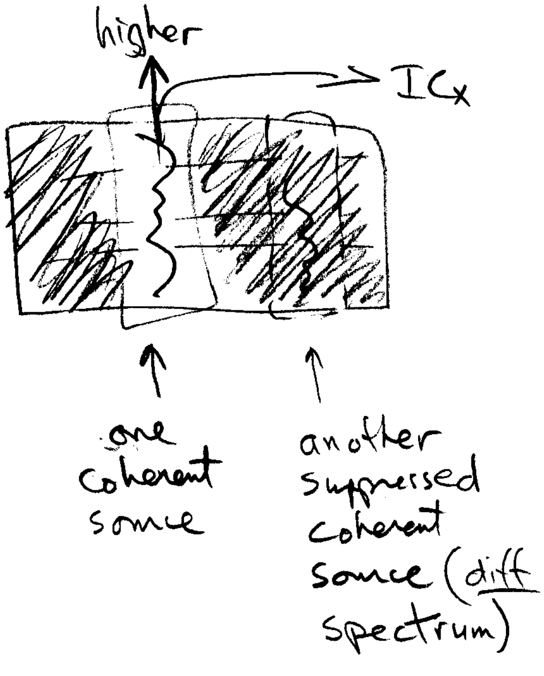
Implications

Why study?

- not all owls do elevation by asymmetric ears

- why study, then?
- → well-worked out example of how brain computes w/ maps
- → likely many other examples not yet worked out

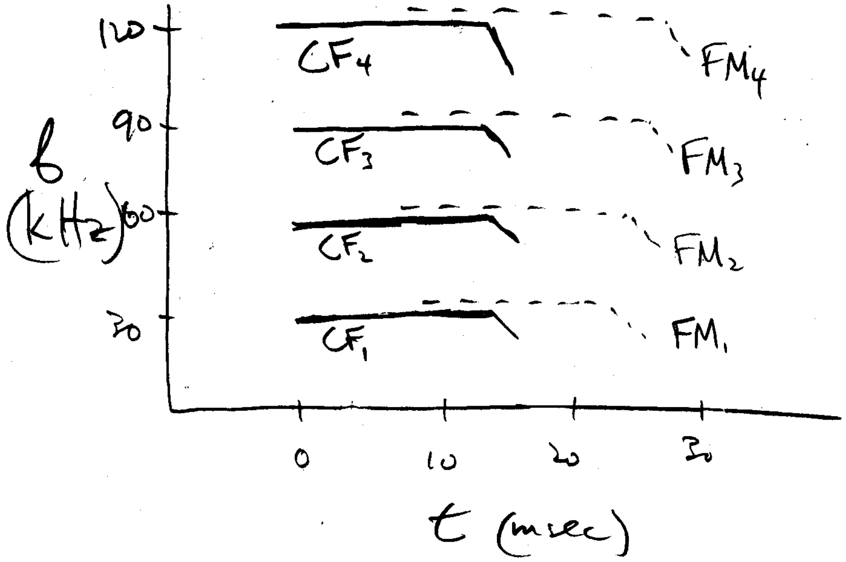
Spatial Auditory Attention



- selectively amplify signals coming from particular spatial position
- depends on spectral diffs between sources

#12

Bats (1) the signal

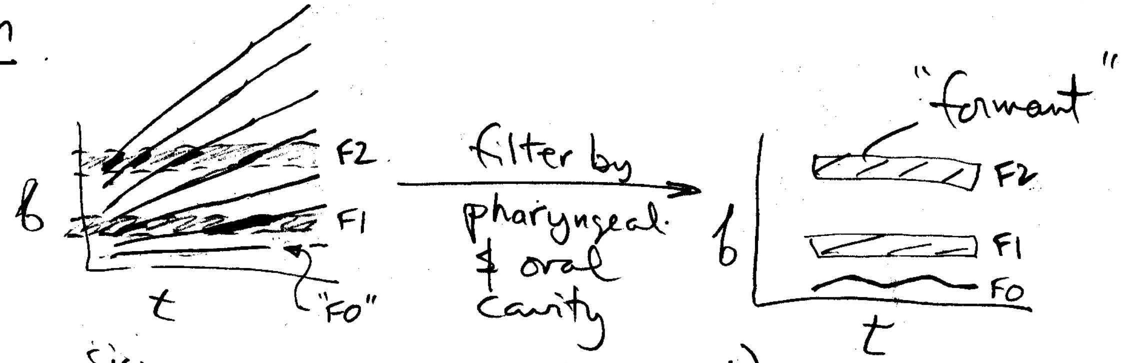


- this is approximately what the auditory nerve sees

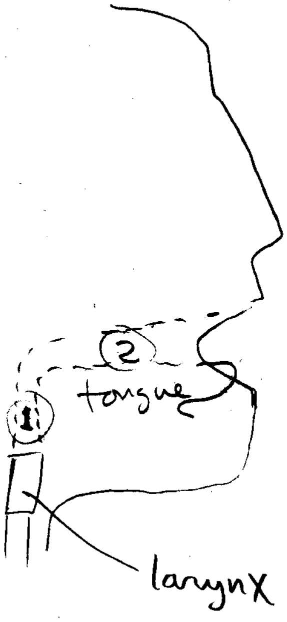
- bat emits scream while "muffling its ears" then releases muscle on stapes to hear echo

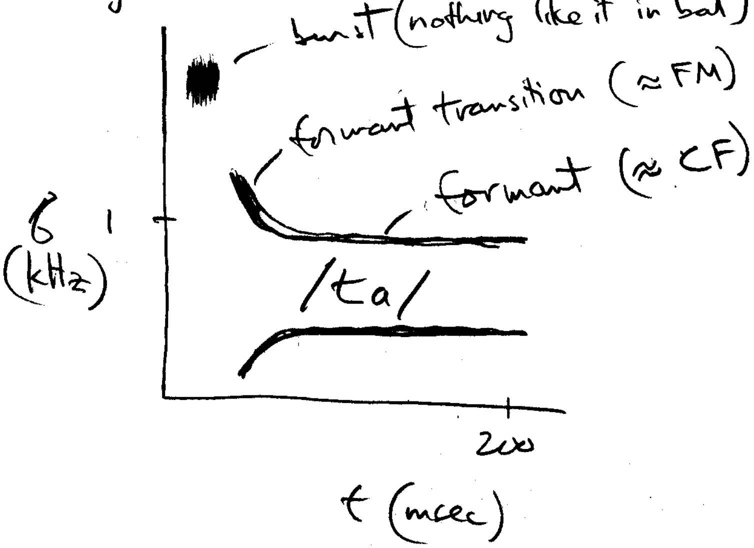
---

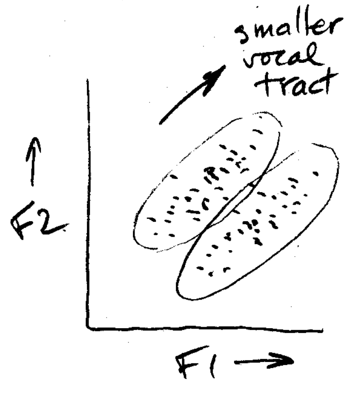
cf. Speech



sign

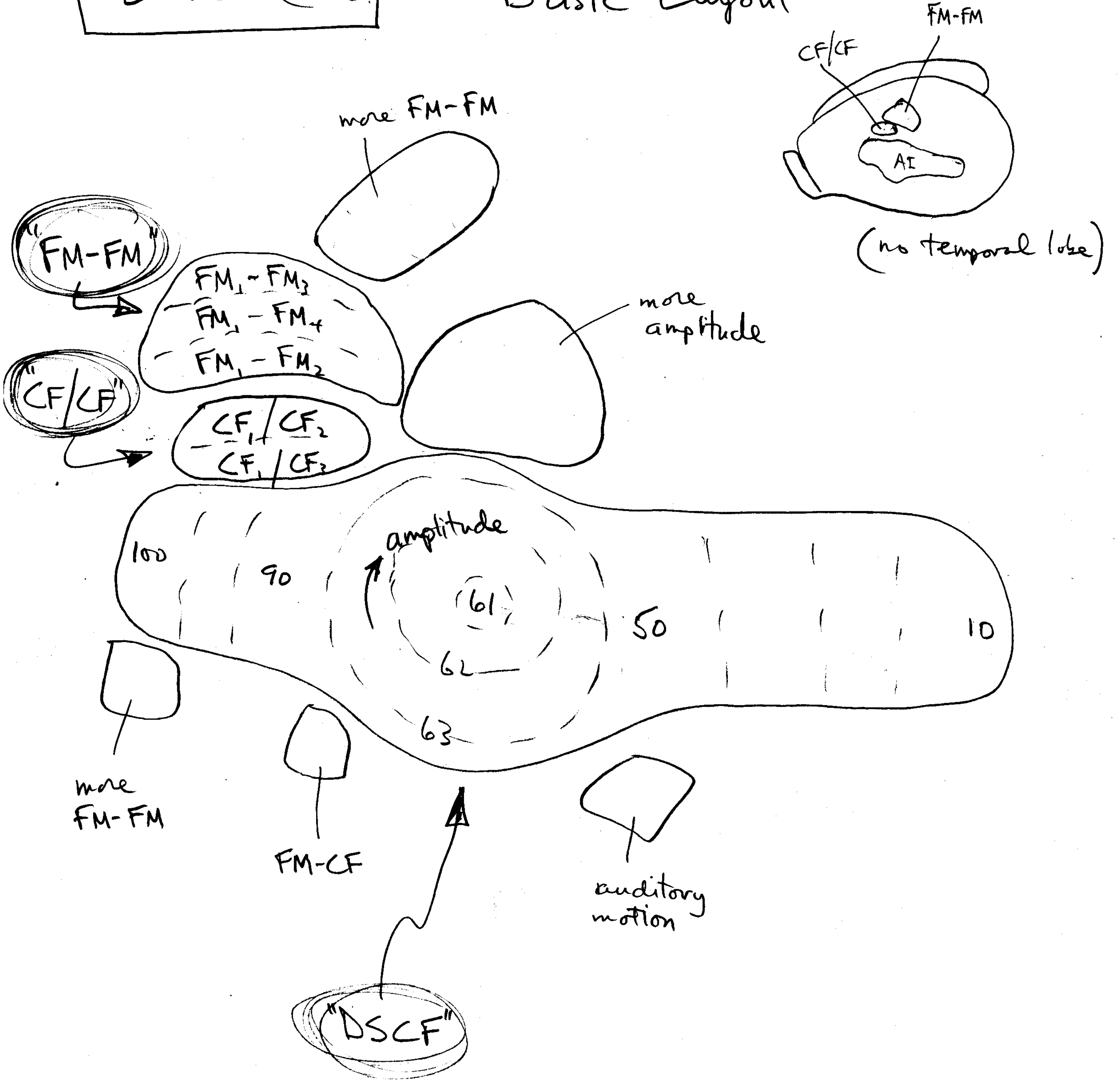






Peter Frampton
Bon Jovi

Bats (2) – Basic Layout



~15 mm² of auditory cortex

(≈ 1,500,000 neurons)

(≈ 15,000,000,000 connections)

Bats (3.)

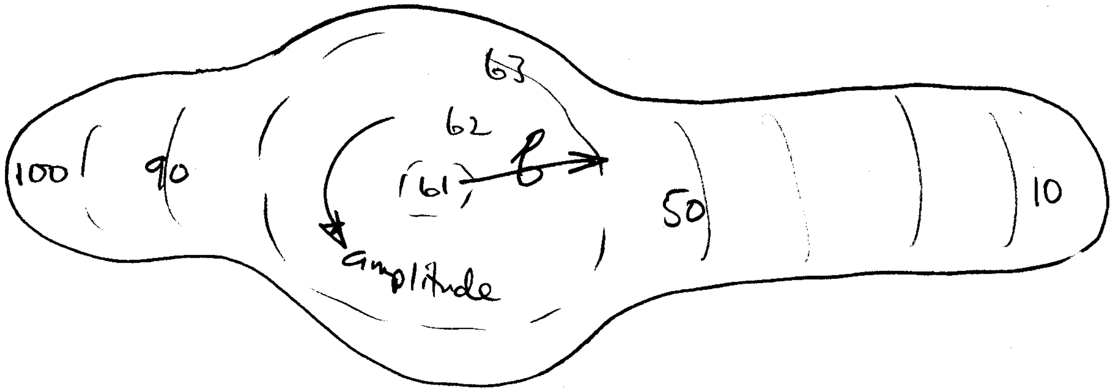
DSCF & Dopp. Shift Comp.

Bat behavior

- what bats do
  - catch insects (e.g. dist. textures)
  - navigate
  - communicate
- Doppler-shift compensation behavior
  - compensation
  - increase rate at attack
- the "acoustic fovea"
  - starts in cochlea
  - by cortex, amplitude is separated from frequency

DSCF area

- frequency & amplitude axes at CF_2 frequency

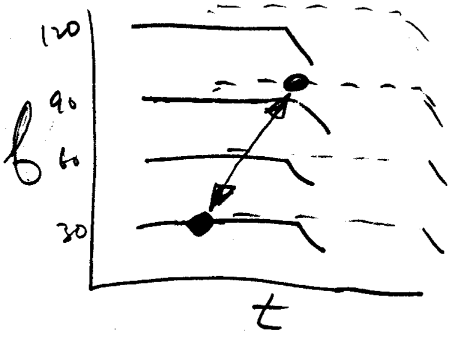


Bats (4) CF/CF

Doppler shift components

- bat movement
- flutter

→ first just consider bat-target relations

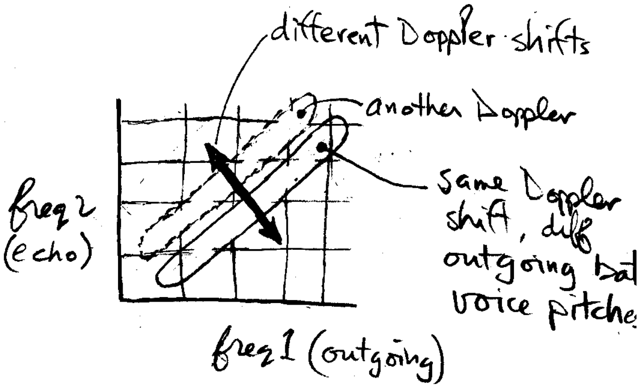


e.g. CF_1/CF_3

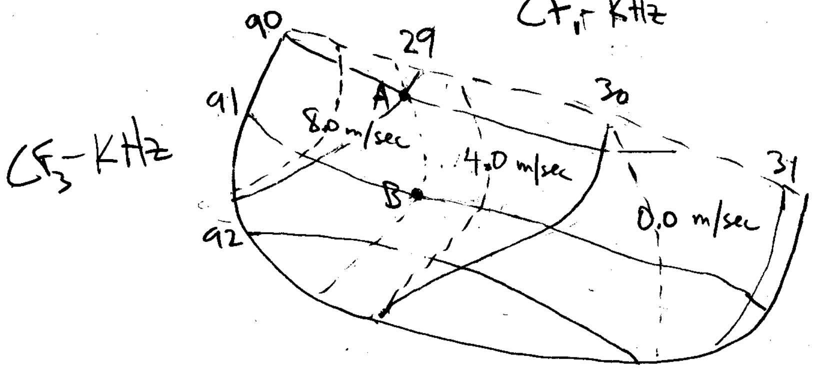
$CF_{emitted} - CF_{echo}$ ~ relative velocity (not distance)

$y = x + 2$
$y = x$
$y = x - 2$

CF/CF area

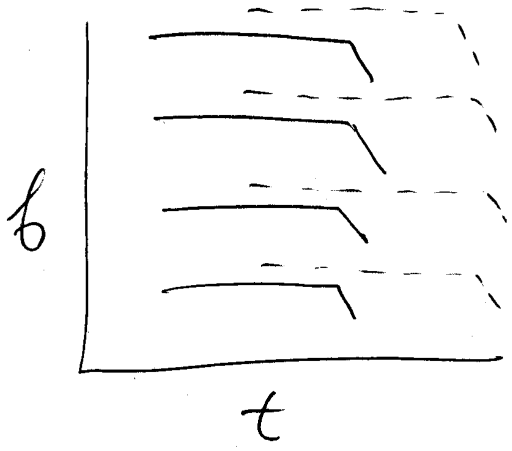


A: 90 − (29×3) = 3 kHz
B: 91 − (29.6×3) = 3 kHz



- units are relatively level-insensitive
- respond only to pairs of CF's (**AND** gate)
- pairs of CF's correspond to particular target velocities (shift = $CF_3 - 3 * CF_1$)
- cannot detect distance (delay in sensitive)

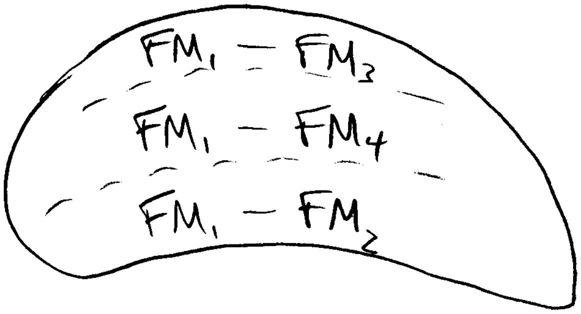
Bats (5) FM-FM

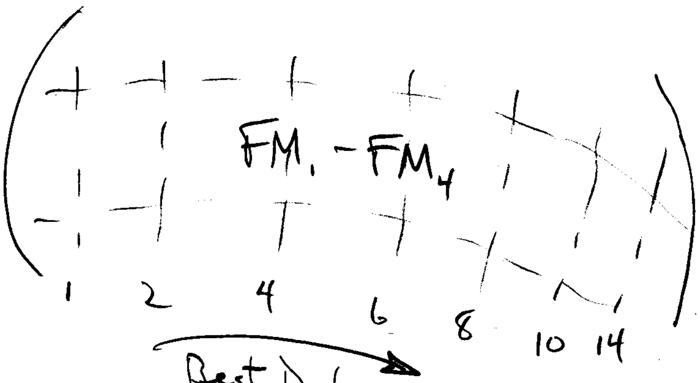


FM info

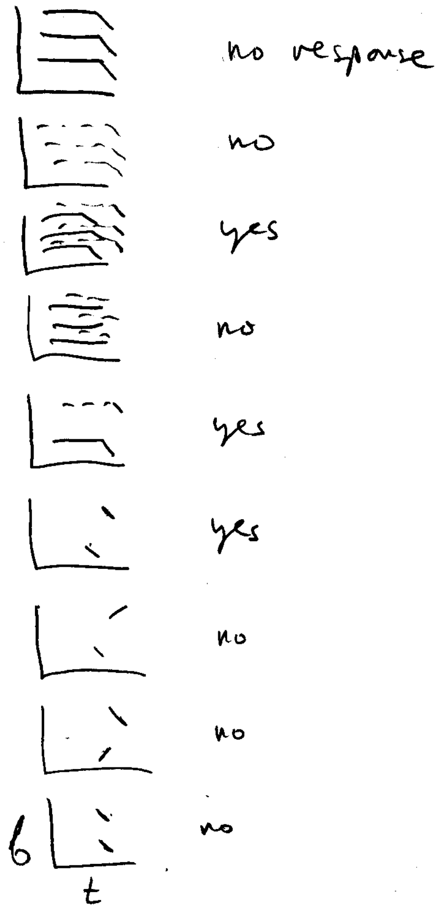
- good because delays are represented (contrast CF)
- by comparing different harmonics, f overlap reduced
- monaural delay detectors (contrast owl)

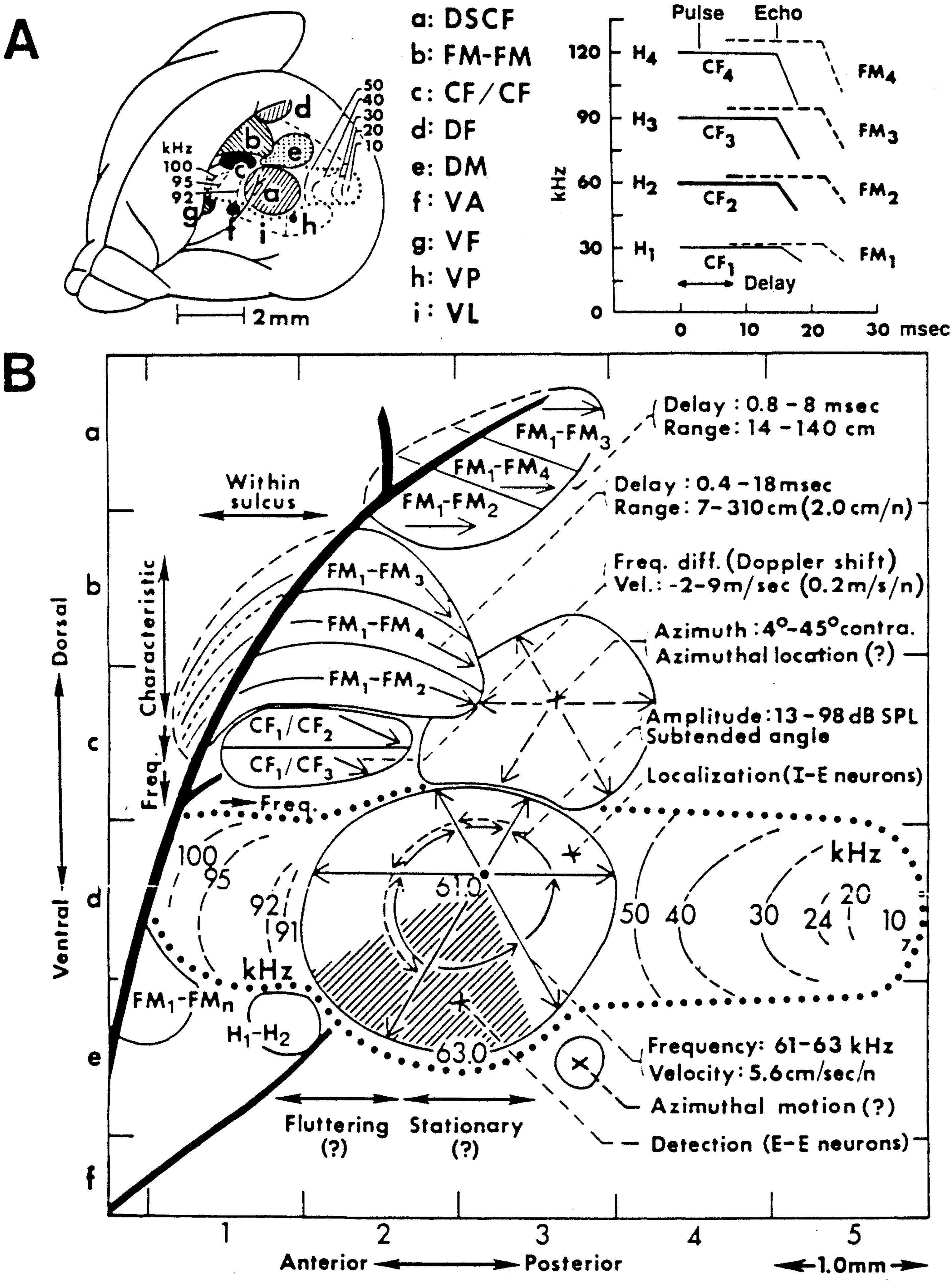
FM-FM area





Best Delay - msec (not μsec!)




A
kHz
100
95
92
50
40
30
20
10
a
b
c
d
e
f
g
h
i
2mm
a: DSCF
b: FM-FM
c: CF/CF
d: DF
e: DM
f: VA
g: VF
h: VP
i: VL
Pulse
Echo
kHz
120
90
60
30
0
H4
H3
H2
H1
CF4
CF3
CF2
CF1
FM4
FM3
FM2
FM1
Delay
0
10
20
30 msec
B
a
b
c
d
e
f
Dorsal
Ventral
Within sulcus
Characteristic
Freq.
Freq.
FM1-FM3
FM1-FM4
FM1-FM2
FM1-FM3
FM1-FM4
FM1-FM2
CF1/CF2
CF1/CF3
Delay : 0.8 - 8 msec
Range : 14 - 140 cm
Delay : 0.4 - 18 msec
Range : 7 - 310 cm (2.0 cm/n)
Freq. diff. (Doppler shift)
Vel.: -2-9 m/sec (0.2 m/s/n)
Azimuth : 4°-45° contra.
Azimuthal location (?)
Amplitude : 13 - 98 dB SPL
Subtended angle
Localization (I-E neurons)
100
95
92
91
61.0
63.0
kHz
50
40
30
24
20
10
7
kHz
FM1-FMn
H1-H2
Frequency: 61-63 kHz
Velocity: 5.6 cm/sec/n
Azimuthal motion (?)
Detection (E-E neurons)
Fluttering (?)
Stationary (?)
1
2
3
4
5
Anterior
Posterior
1.0mm

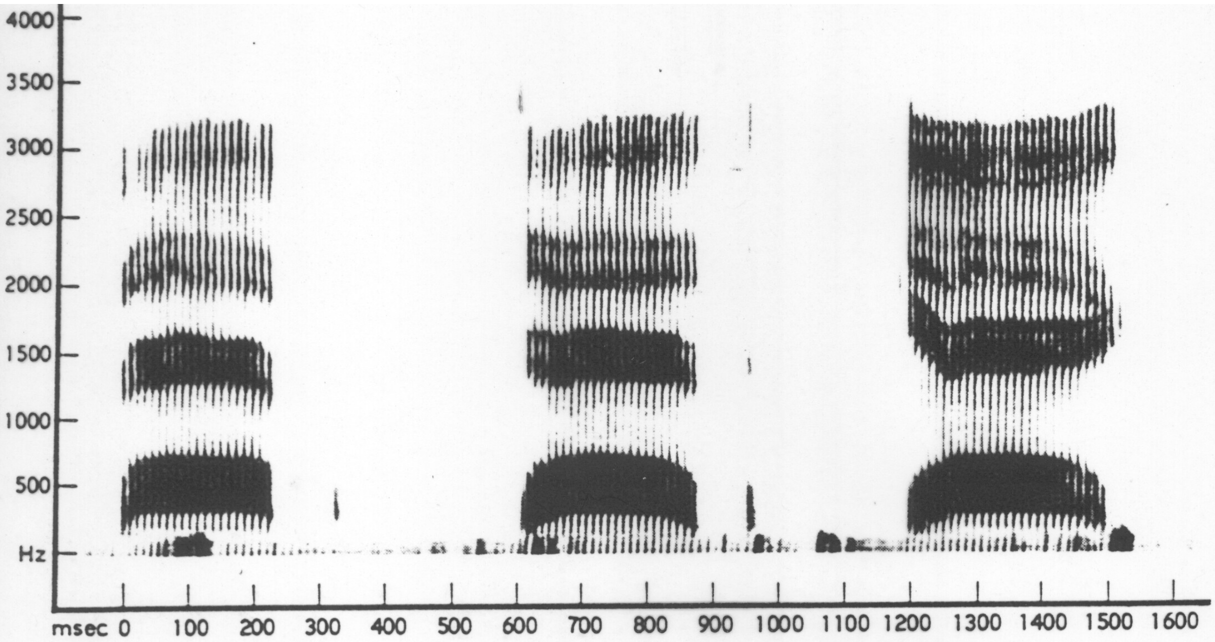


Figure 8.9 *A spectrogram of the words "bab, dad, gag" (British accent).*

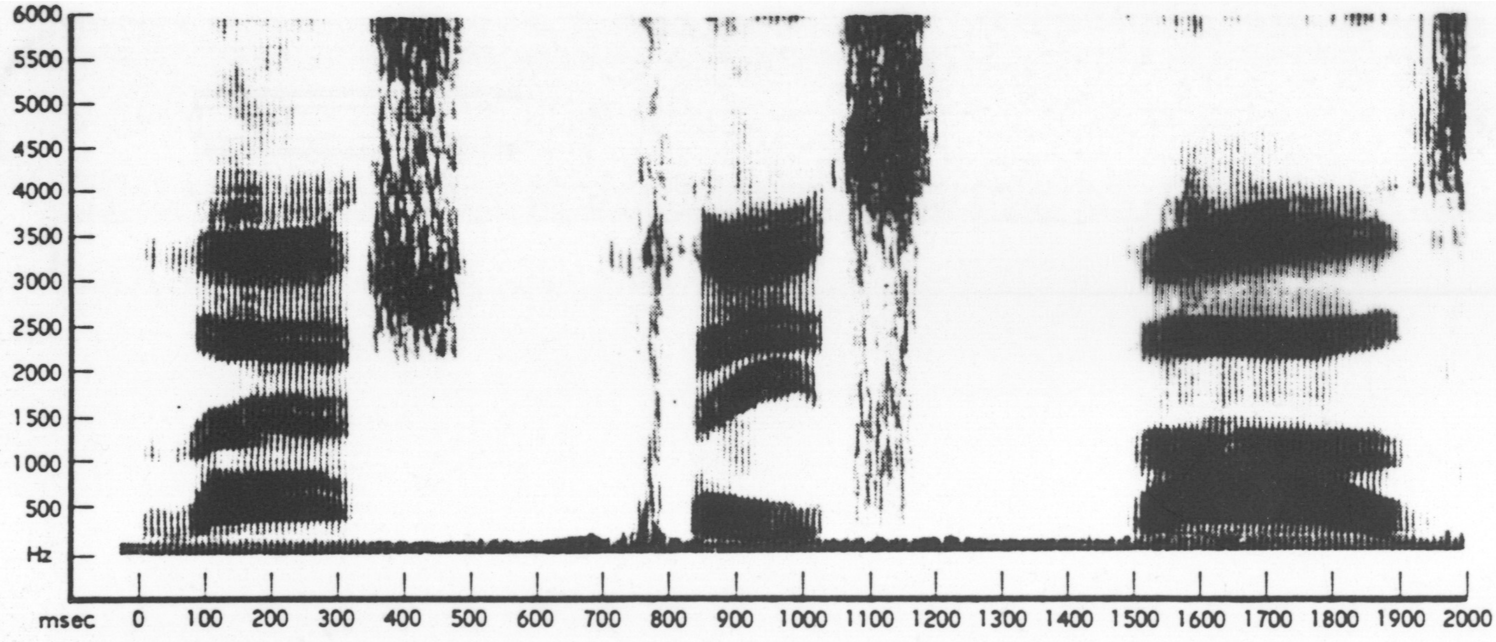
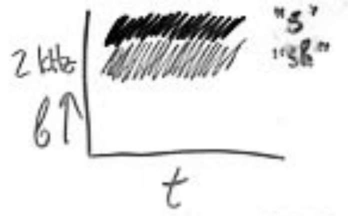
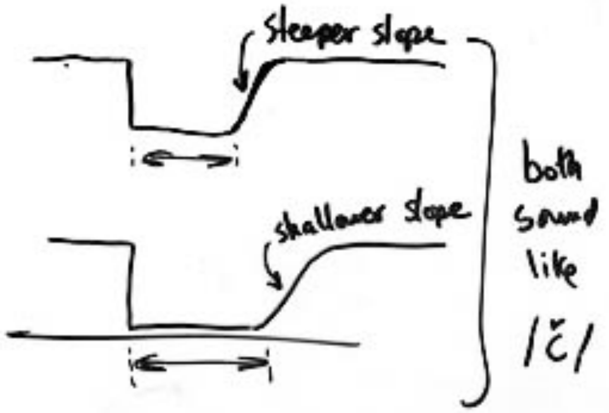
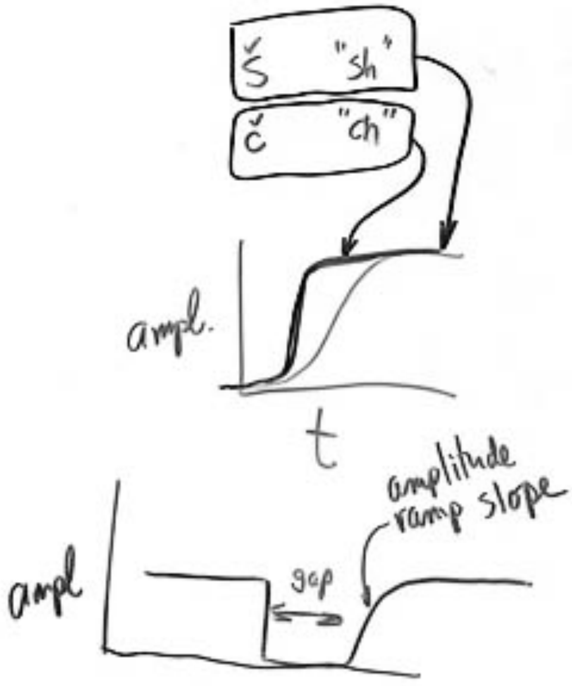


Figure 8.11 *A spectrogram of "lash, face, vase" (British accent).*

f "trading relations"
- aud ctx
- complex snds





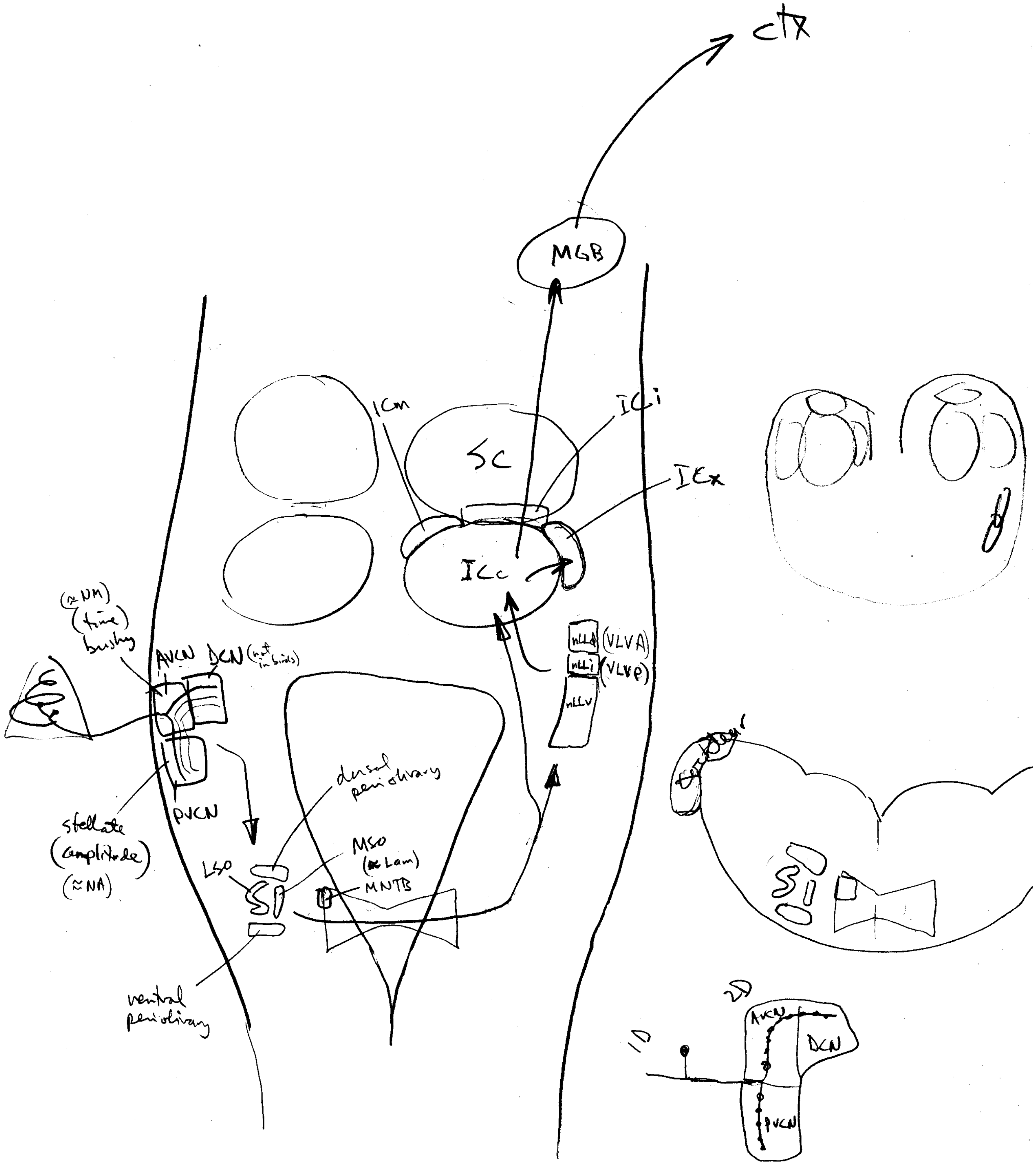


N. VIII

anesthetized cat

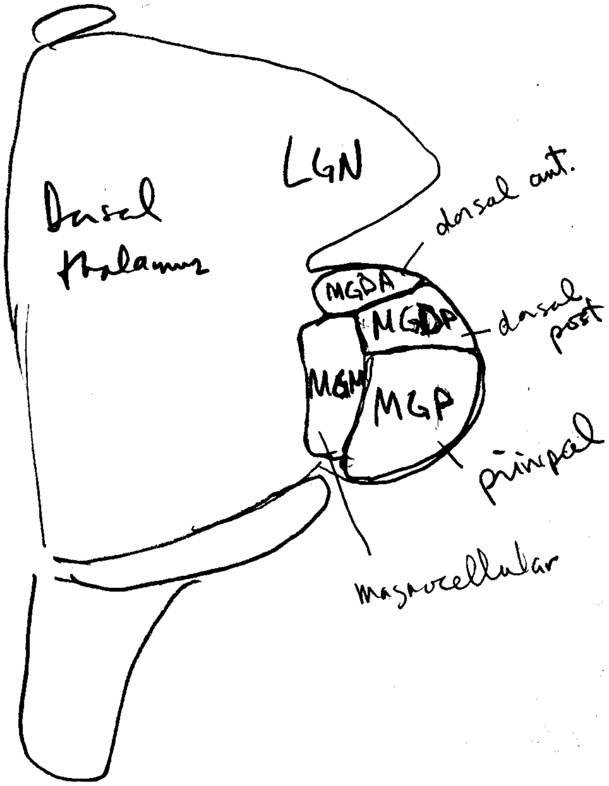
larger gap
→ dishabituation

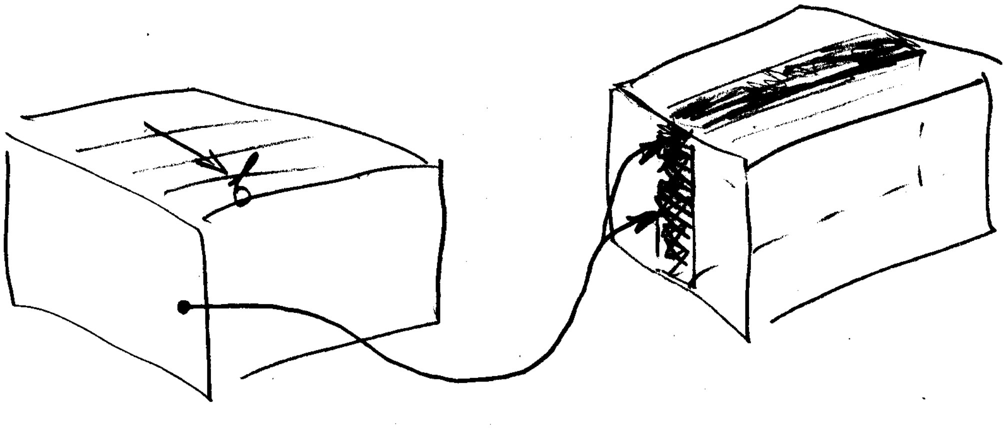
Auditory Brainstem & Ctx in Mammals



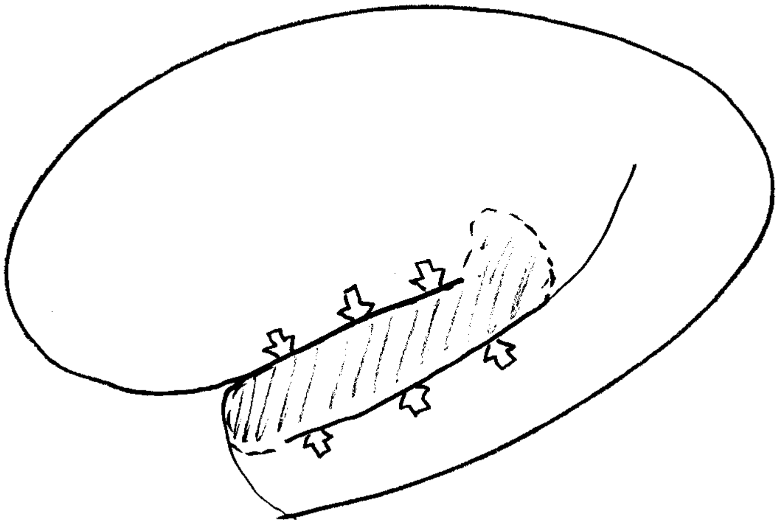
Auditory Thalamus

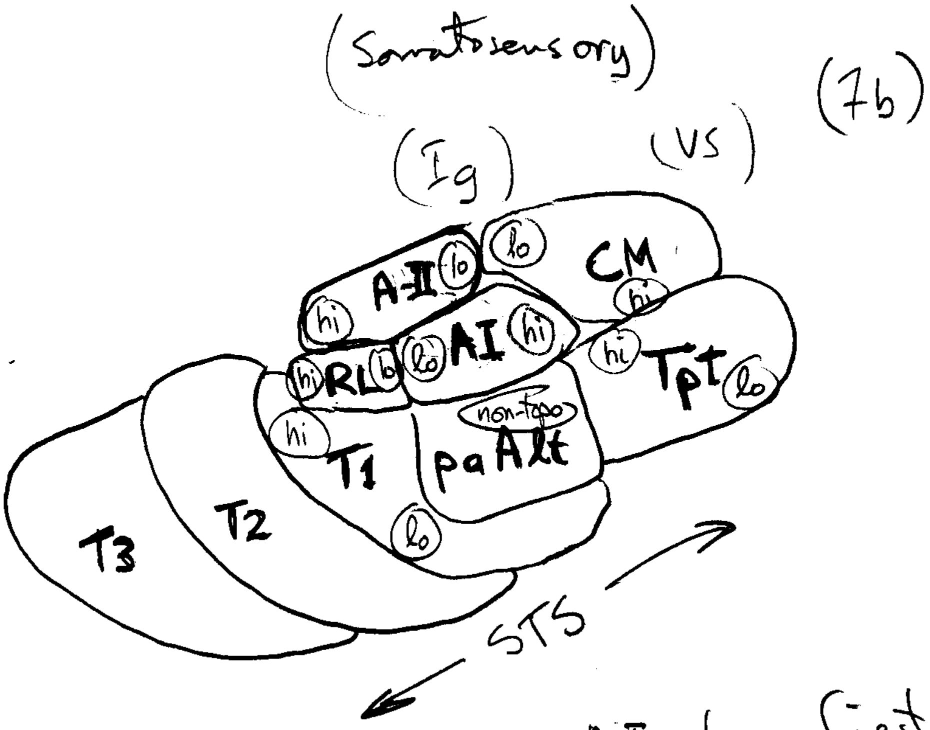
medial geniculate ⇒ posterior & lateral in thalamus





Cortical Areas — Auditory



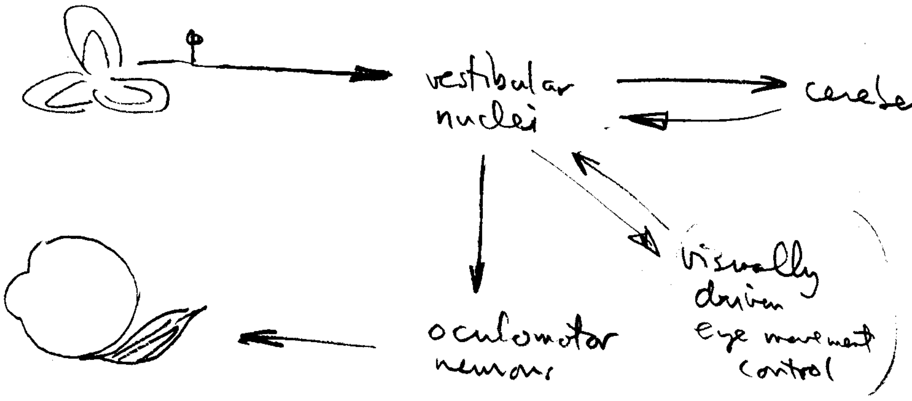


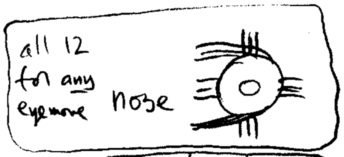
AI has finest-grained f map

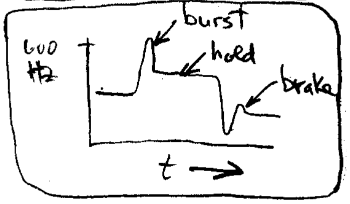
Eye Movements Anatomy

1) Vestibulo ocular reflex

compensating for head movements

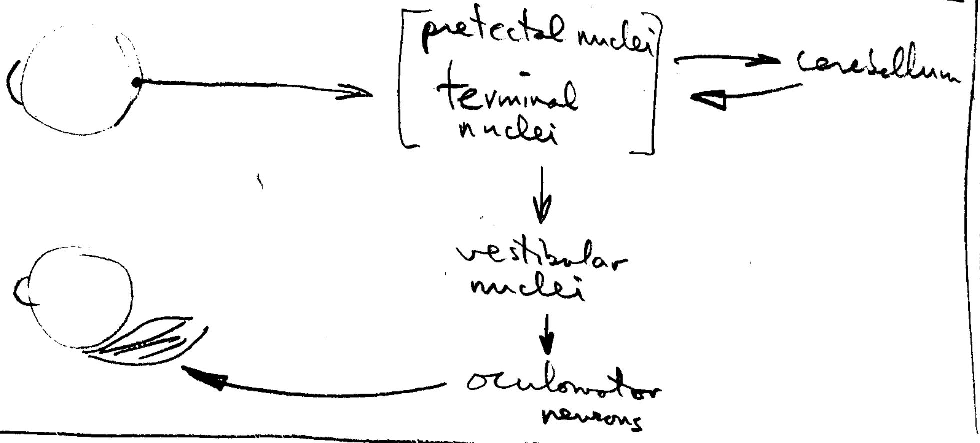






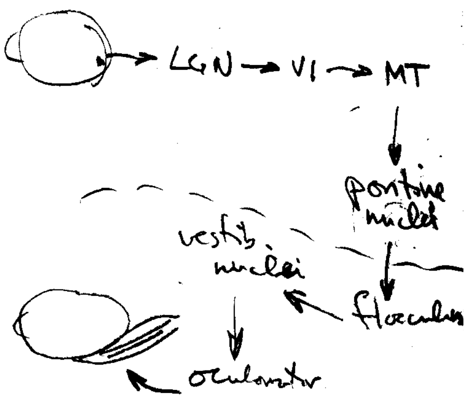
2) Optokinetic Nystagmus

Compensating for visual field movements



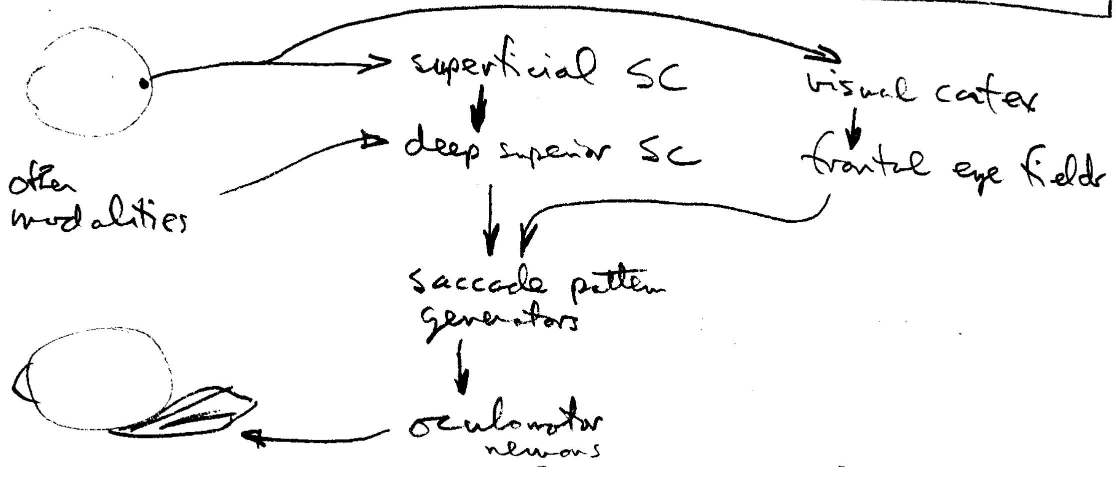
2a) Pursuit

following a small object

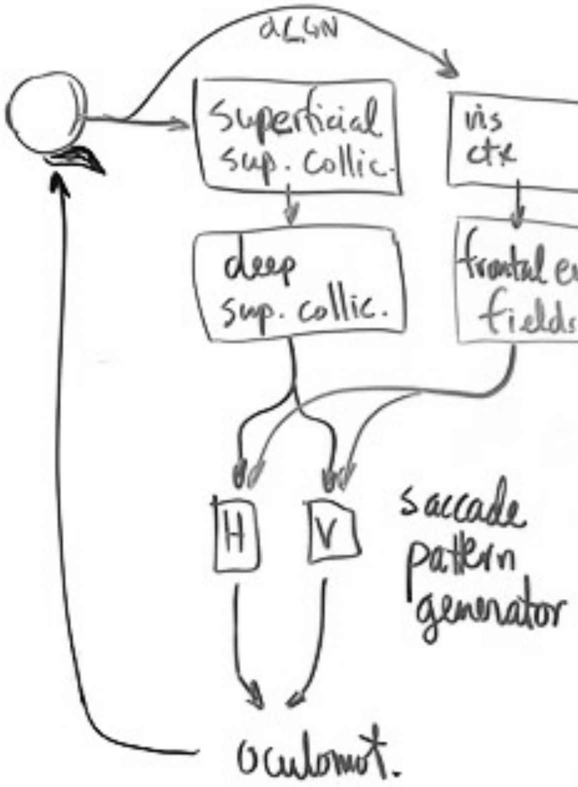


3) Orienting Eye Movements

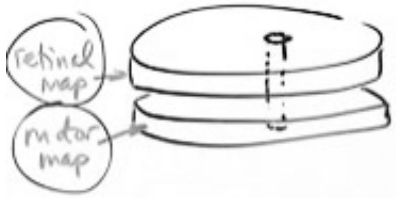
looking at new things



Orienting Eye Movements



sup. collic.
- 50 cell types
- many layers



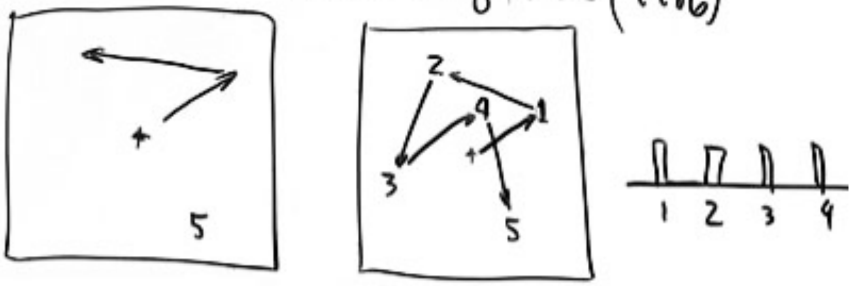
- double-step saccade

1) record receptive field

2) record during eye move

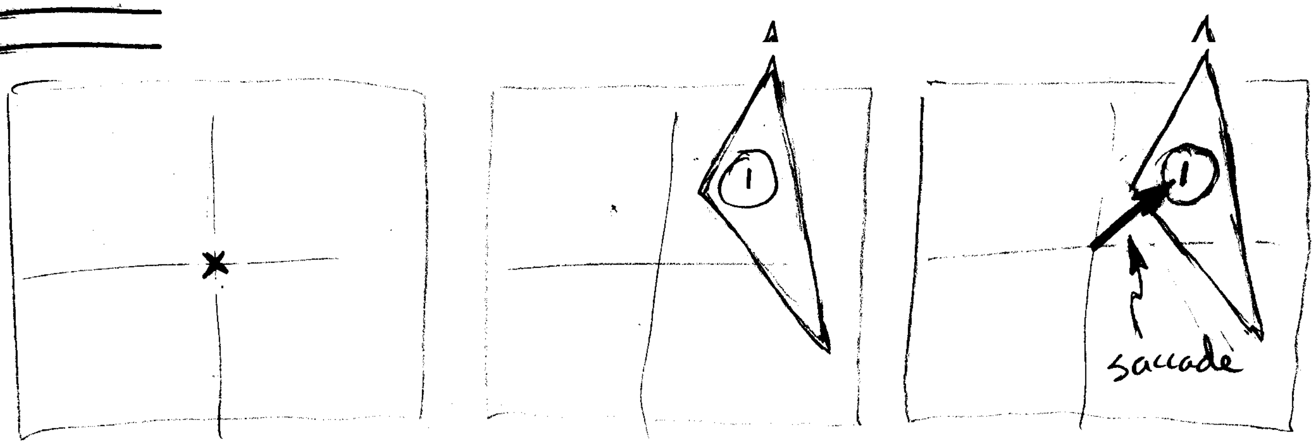
3) stim

Hallett & Lightstone (1976)



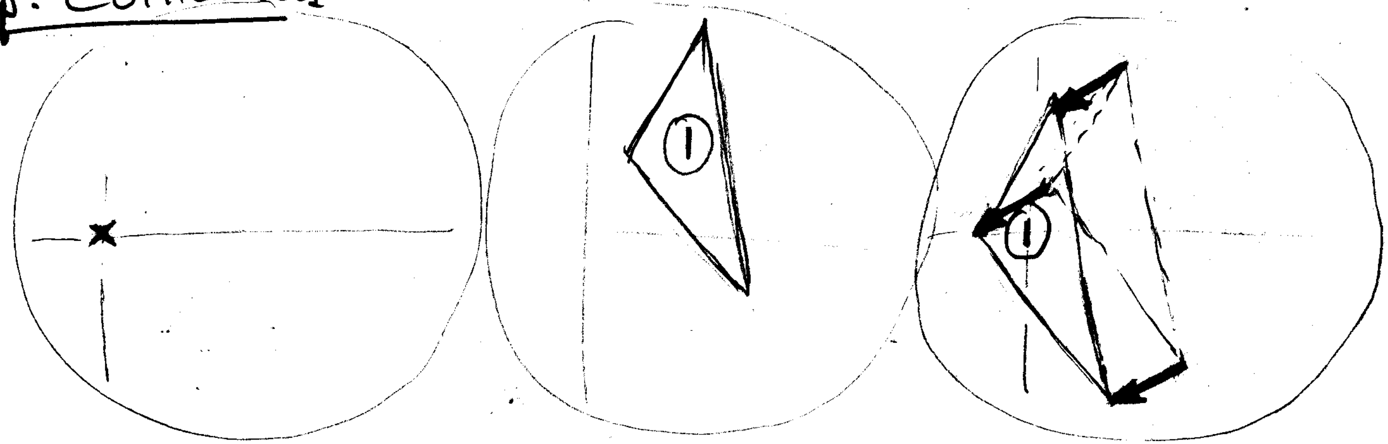
How saccades update stationary targets

World



fixation target saccade

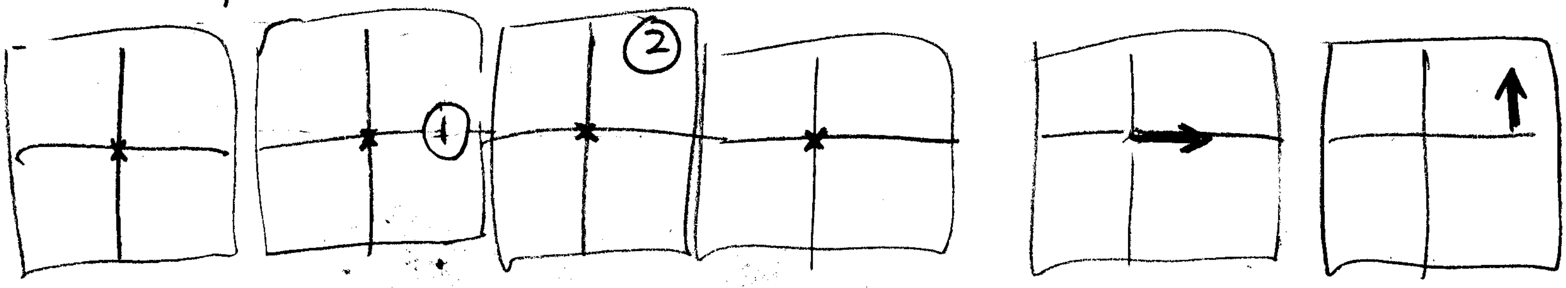
Sup. Colliculus



fixation target saccade

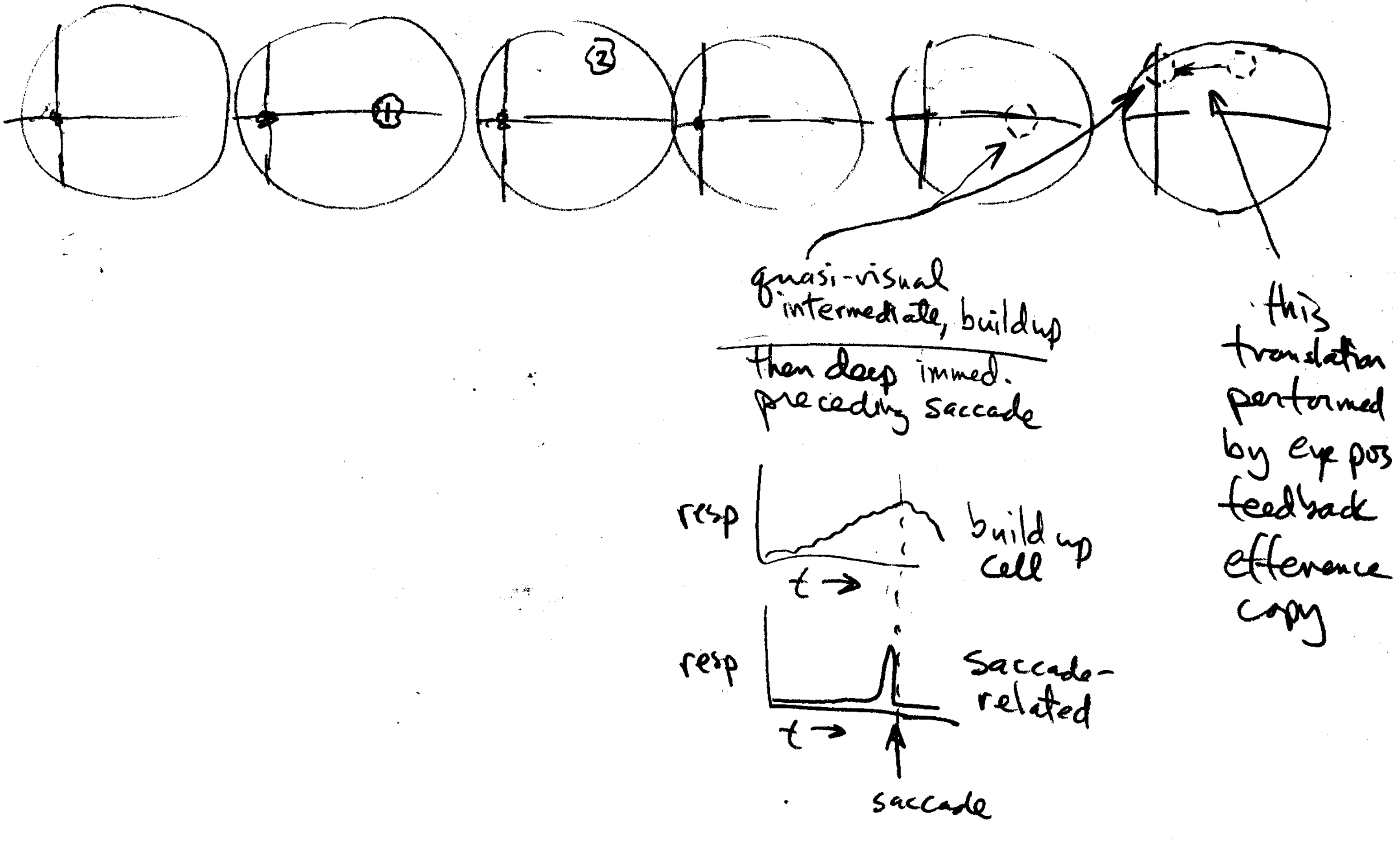
Double - Step Saccade Remapping

World/Behavior



Sup Collic

for clarity, reversed (actually right hemifield is represented in *left* SC)

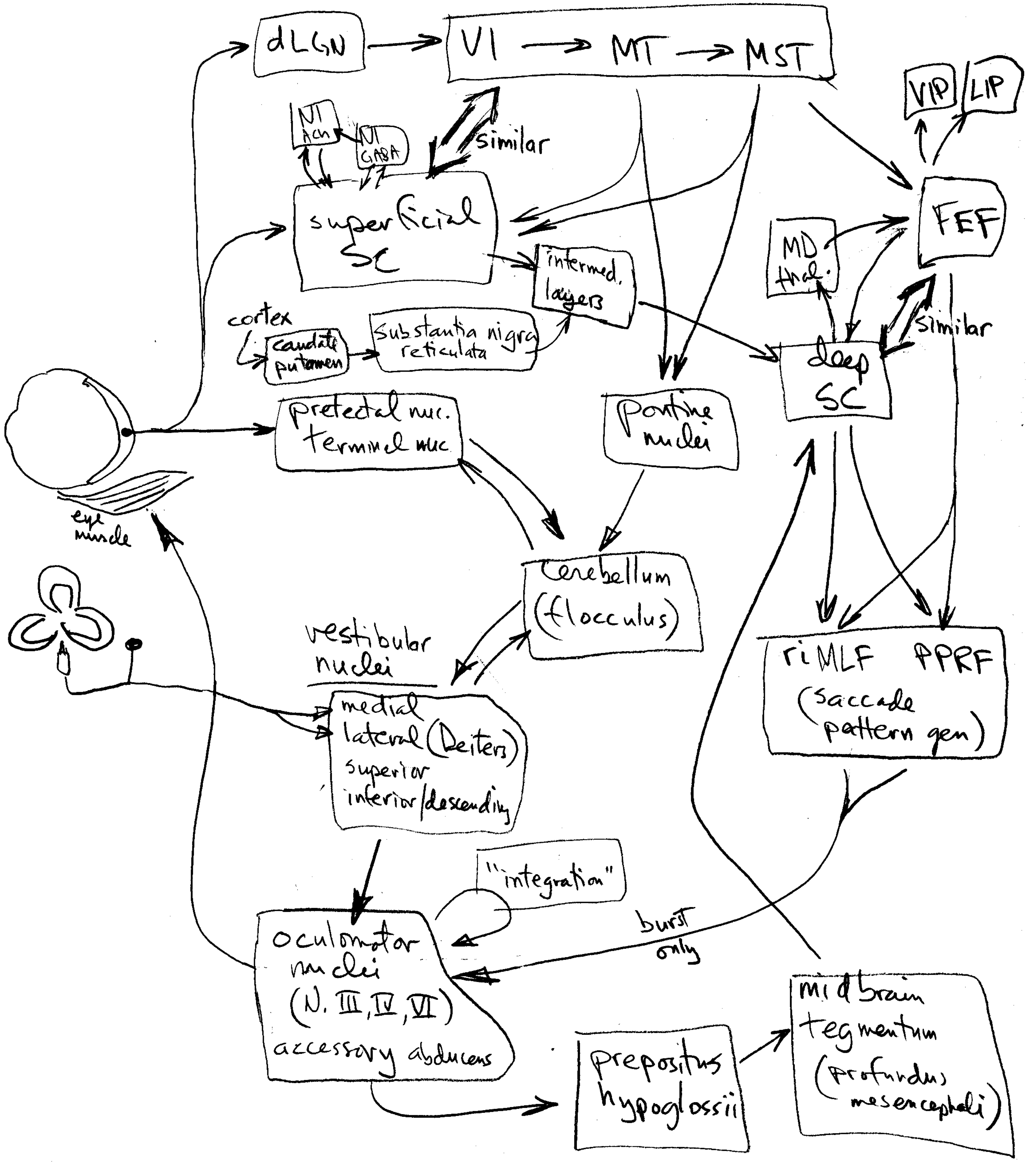


quasi-visual intermediate, buildup

then deep immed. preceding saccade

this translation performed by eye pos feedback efference copy

All-In-One (do after indiv.)

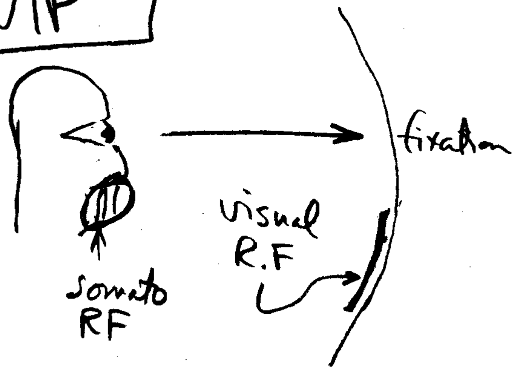


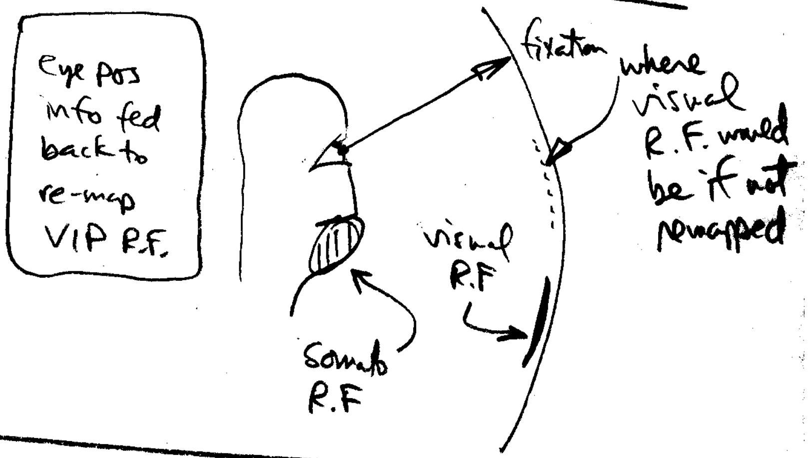
Coordinate Systems for Multi-Sensory Fusion

- SC codes target position by <u>position</u>, not <u>amount</u> of firing
- saccade patt. gen. code position by amount of firing < horiz / vertical

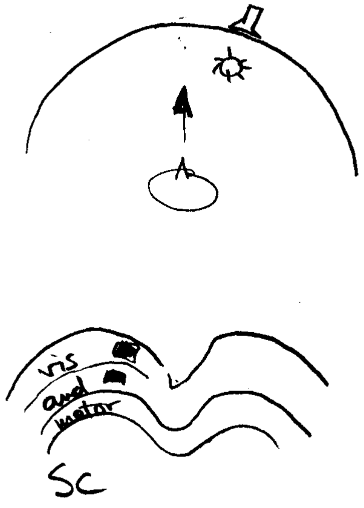
- SC has abstract notion of target — potential target whether or not it's actually there
  ↳ saleency map

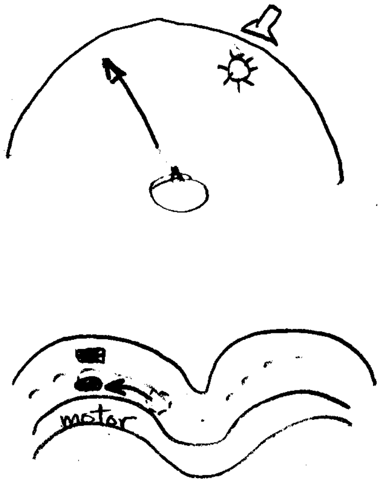
VIP



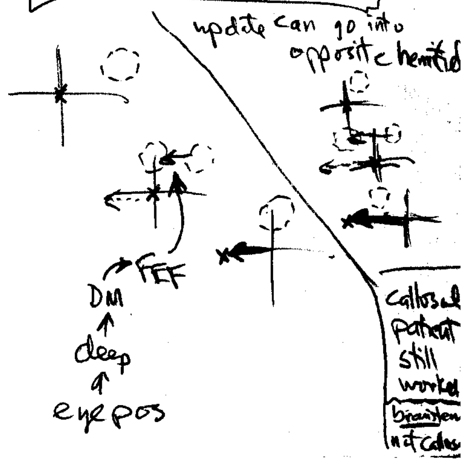


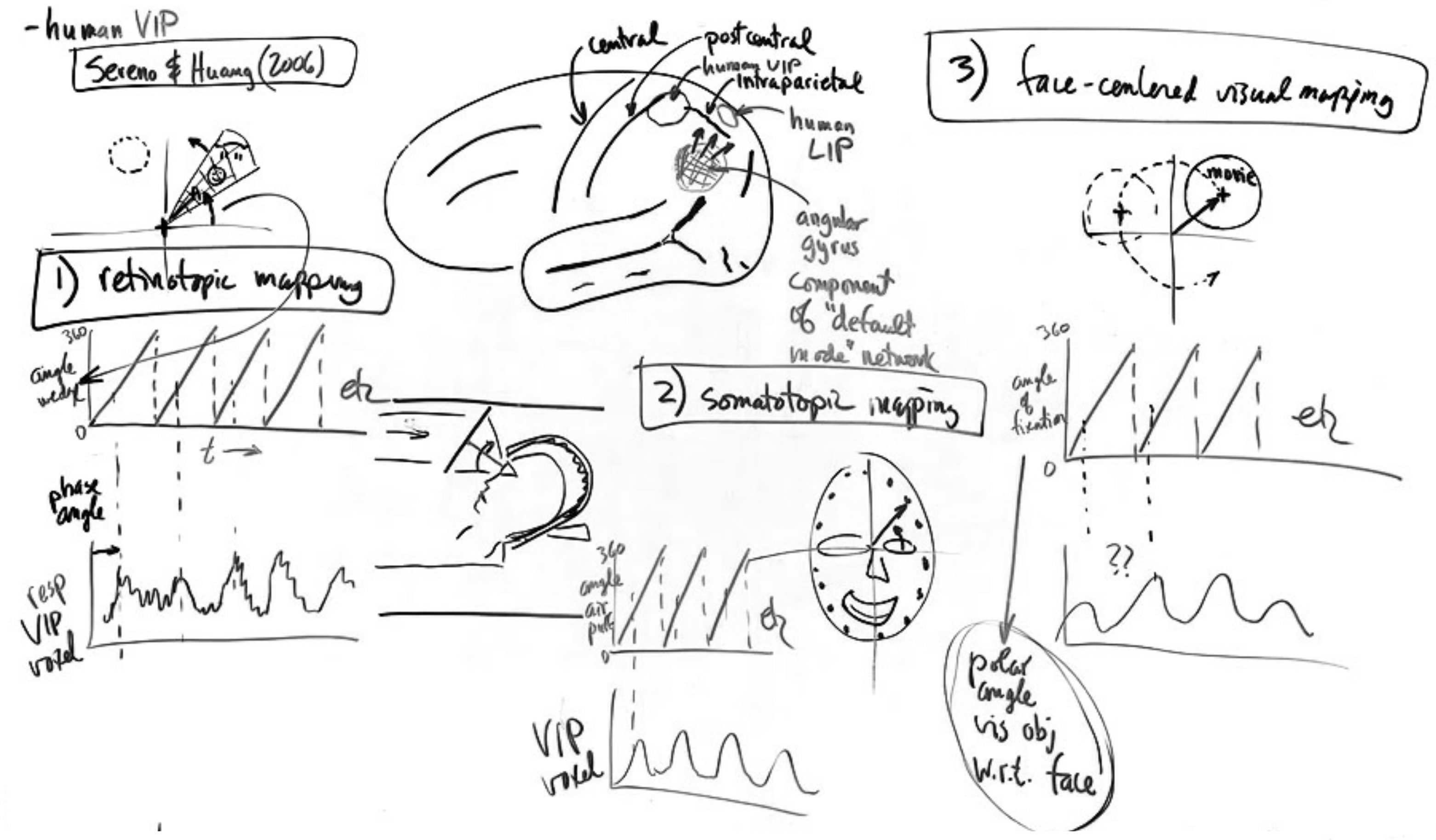
Auditory / SC

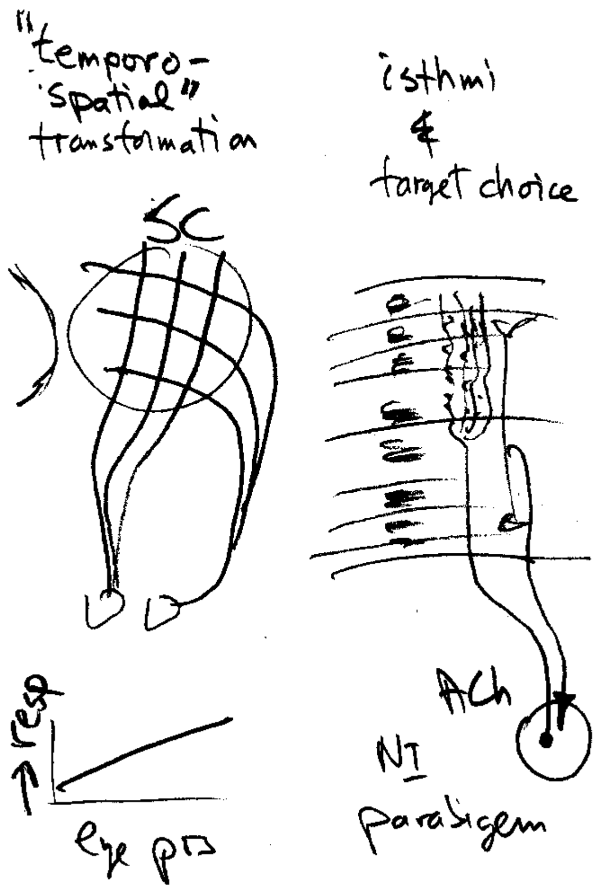




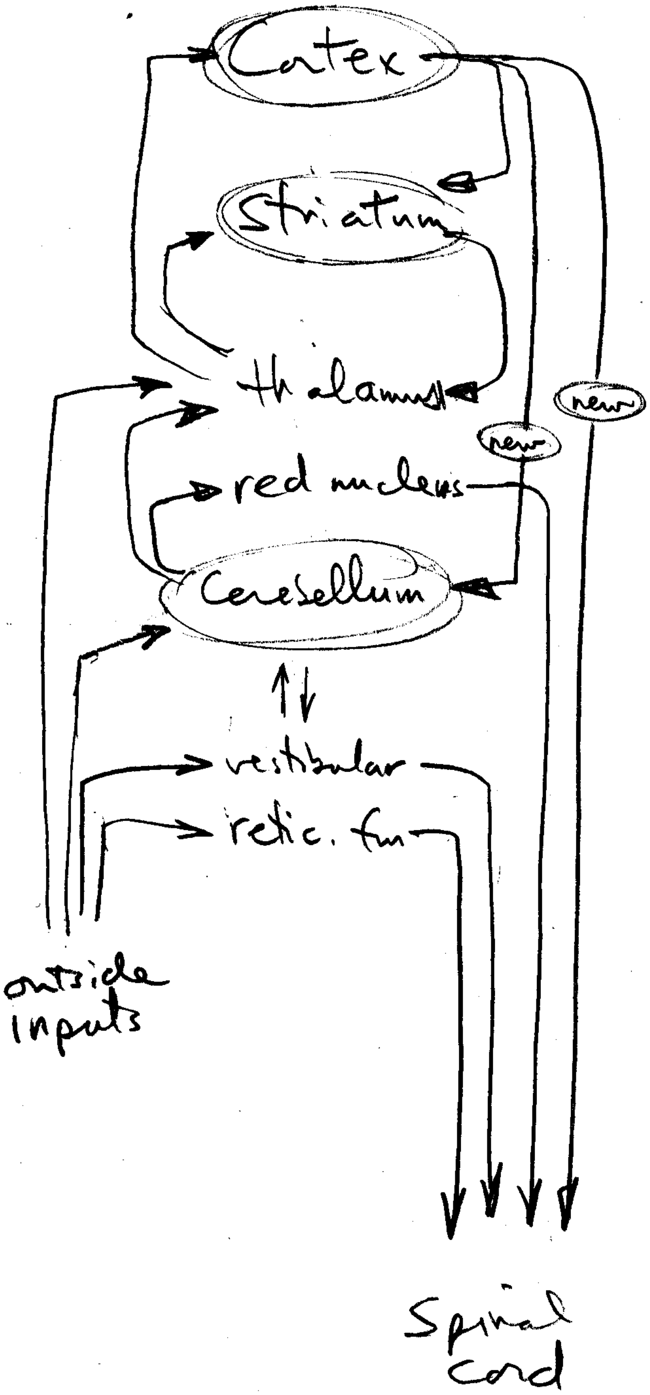
LIP pre-saccadic updating




-human VIP
Sereno & Huang (2006)
1) retinotopic mapping
360
angle wedge
0
t →
ett
phase angle
resp VIP voxel
central
postcentral
human VIP
intraparietal
human LIP
angular gyrus
component of "default mode" network
2) somatotopic mapping
360
angle air puff
0
ett
VIP voxel
3) face-centered visual mapping
movie
360
angle of fixation
0
ett
??
polar angle vis obj w.r.t. face


"temporo-spatial" transformation
SC
resp
eye pos
isthmi & target choice
ACh
NI
paradigm

Motor System Basic Plan



excludes SC

↳ visually directed orienting movements
- eyes
- head
- ears
- whiskers
- shoulders

Motor System "Principles"

1) Short number of synapses from input to output (retic sp. vestib. sp. rubro sp.)

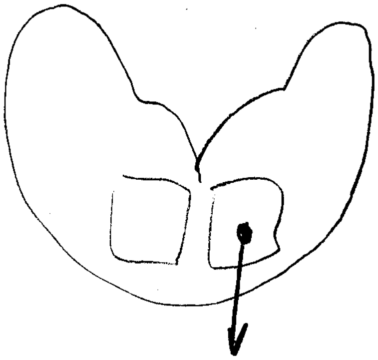
2) Pattern generators — autonomous lower level sensorimotor transformers (spinal cord)

3) Additional systems added as input to pattern generators (cereb striatum)

4) Higher level pattern generators may have independent access to motorneurons (corticospinal → fingers ↓ speech)

5) many "lateral" connections to mediate conflicts between different info sources (Suppress VOR during pursuit)

Descending Paths

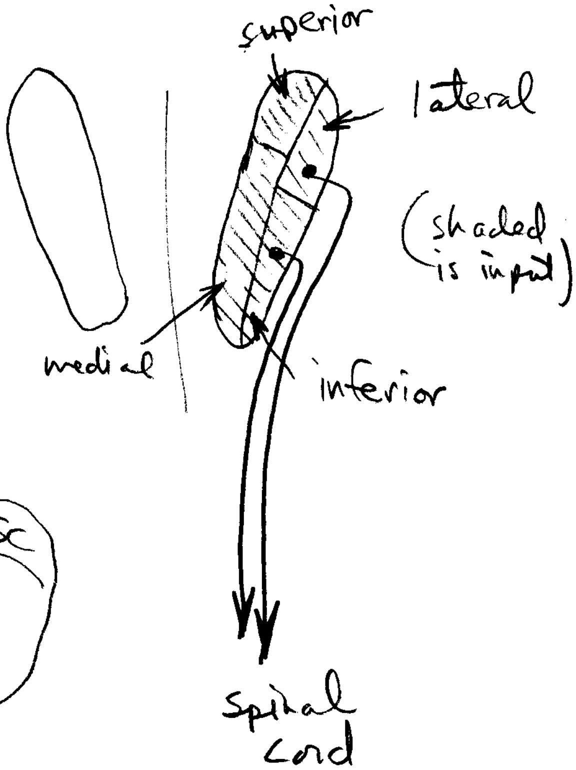


Reticulospinal

anti-gravity "extensors"

Pontine & Medullary retic. fm. — uncrossed
midbrain Retic fm — crossed

Vestibulospinal

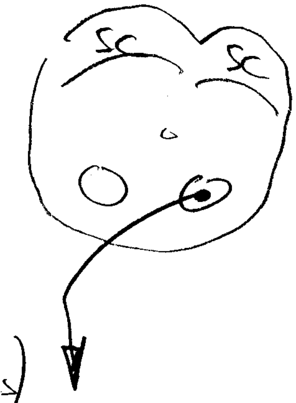


vestibular nuclei (in pons)
- all receive semicircular or otolith
- main descending outputs from lateral & inferior

Rubrospinal

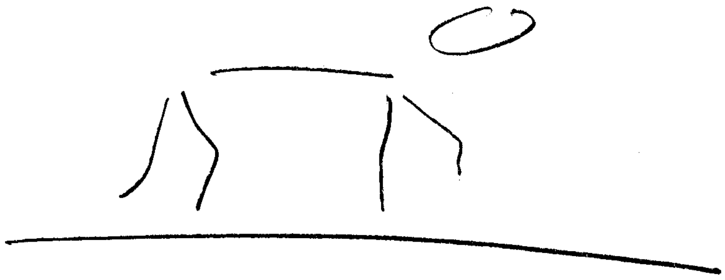
active movement "flexors"

- red nucleus in mi
- crossed

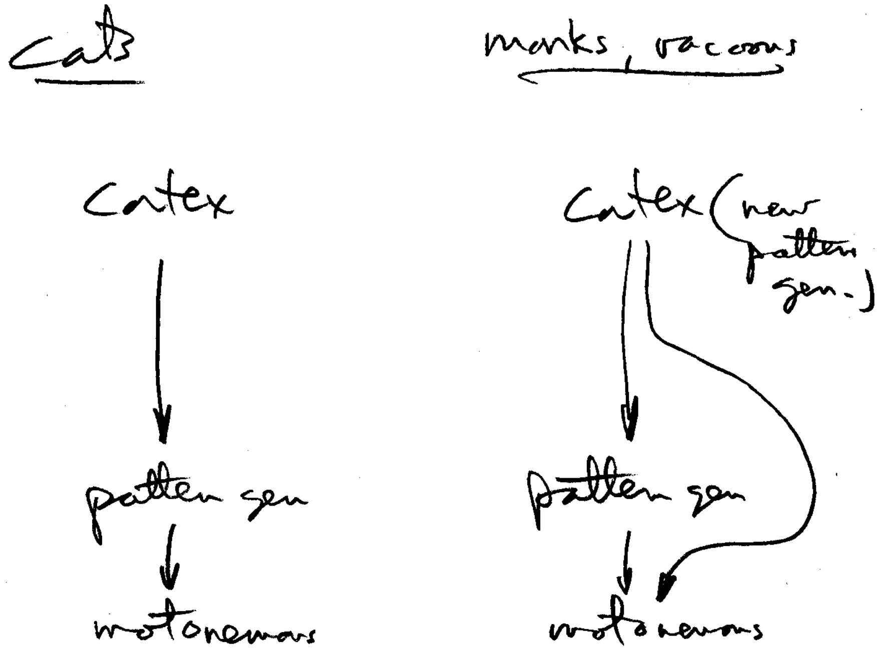


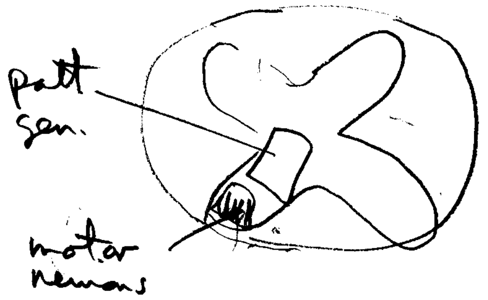
Corticospinal (only mammals)

Pattern Generators



- spinal cord generates rhythmic, coordinated muscle movement w/o cortex (requires sens. stim)
- cortical control has been superimposed on existing I/O systems
- in primates, raccoons, cortical control bypasses spinal pattern generator
- same thing in birds for song and humans (but not chimps!) for speech

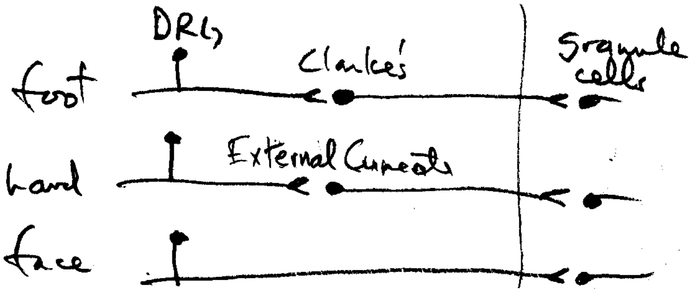


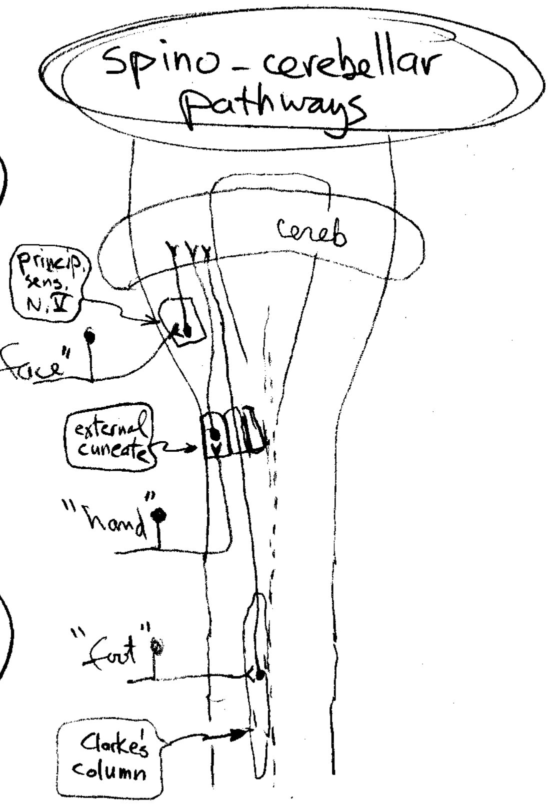


Cerebellum inputs

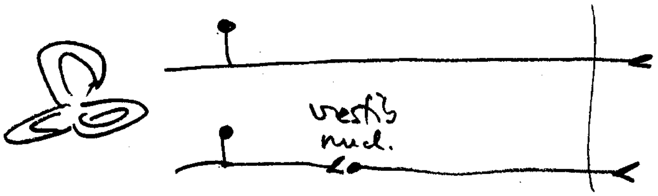
mossy fibers

Spinal cord (somatosensory input)

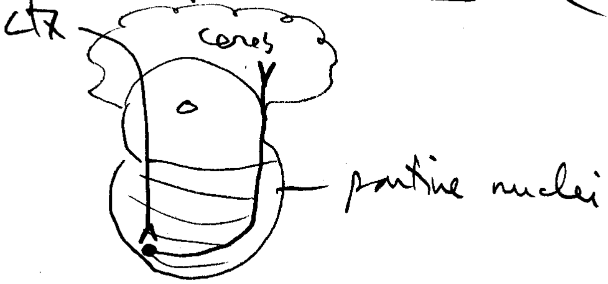




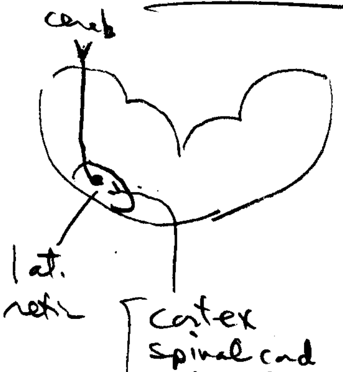
Vestibular system (vestib. input)



pontine nuclei (cortical input)

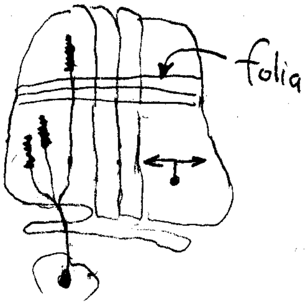


lateral reticular (mixed)



cortex
spinal cord
red nucleus
vestibular nuc

climbing fibers



inferior olive (spinal, vis. and aud. input)

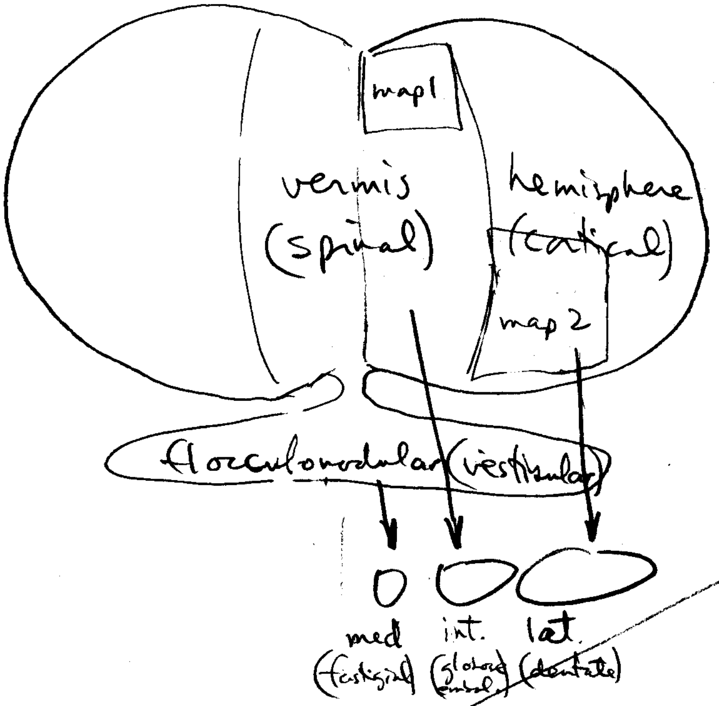
- main
- medial accessory
- dorsal accessory

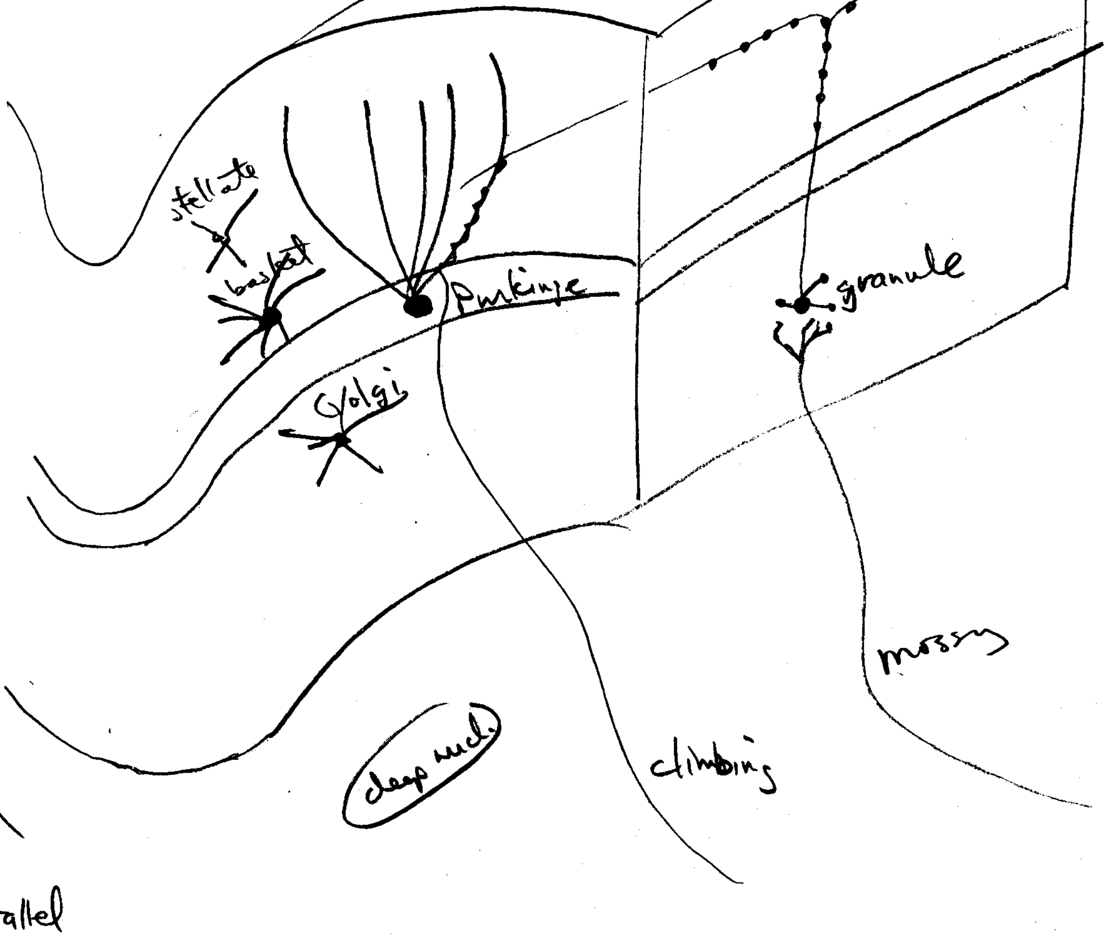
Sensory inputs → IO → climbing fibers

Cerebellum - Basic Layout

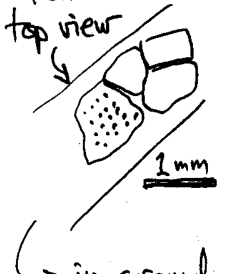
defects

- decomposition of movement
- inaccurate movements
- oscillations
  - intention tremor
- titubation
  - trunk-al tremor!
- lack of VOR adjustment





"fractured somatotopy"



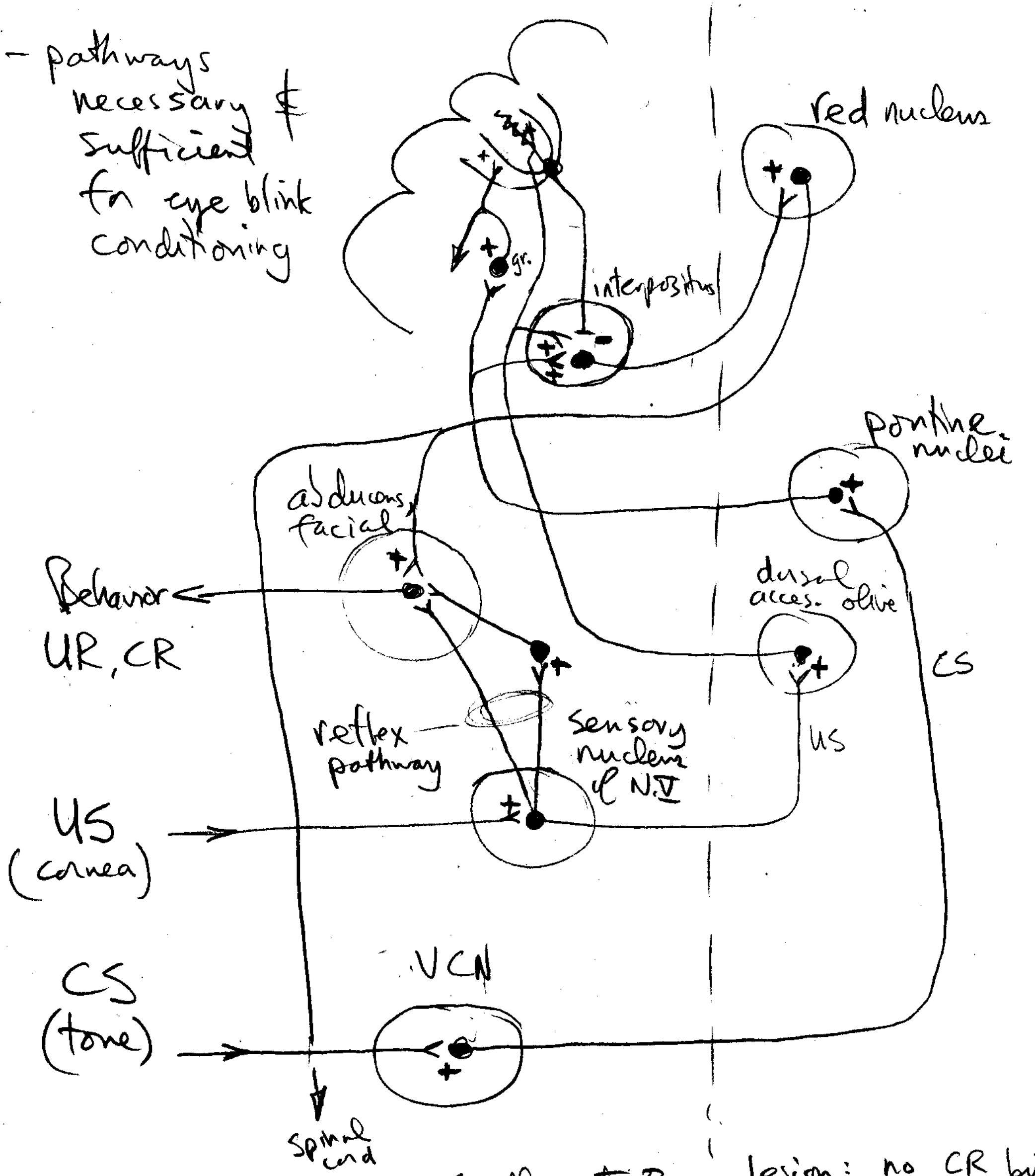
↳ in granule cell layer and P-cell layer!

↳ ascending > parallel

CEREBELLUM & CONDITIONING

Thompson

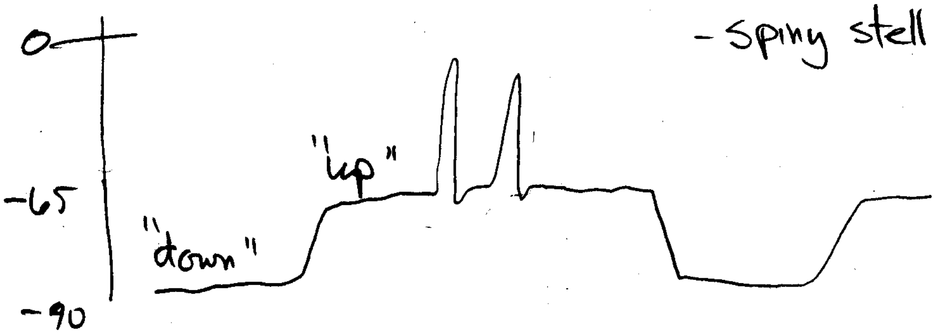
- cf. Lashley
- pathways necessary & sufficient for eye blink conditioning



cf. Saussure! an arbitrary connection

- conditioned response (CR) → small part of interpositus nucleus
  - lesion: no CR but UR remains
  - stim: CR generated
- conditioned stimulus (CS) → pontine nuclei
  - lesion: no eyelid CR to auditory
  - stim: can be used as CS
- US → DAO
  - lesion: CR goes to extinction
    - ↳ trace not here
  - stim: can be used as US (60-400μA !!)

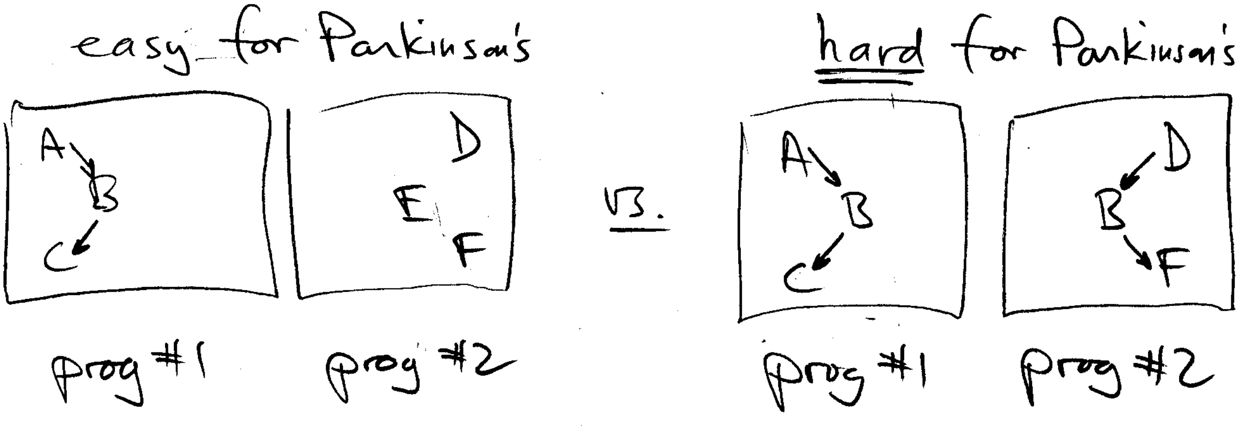
Up & Down State



- spiny stellate CP neurons

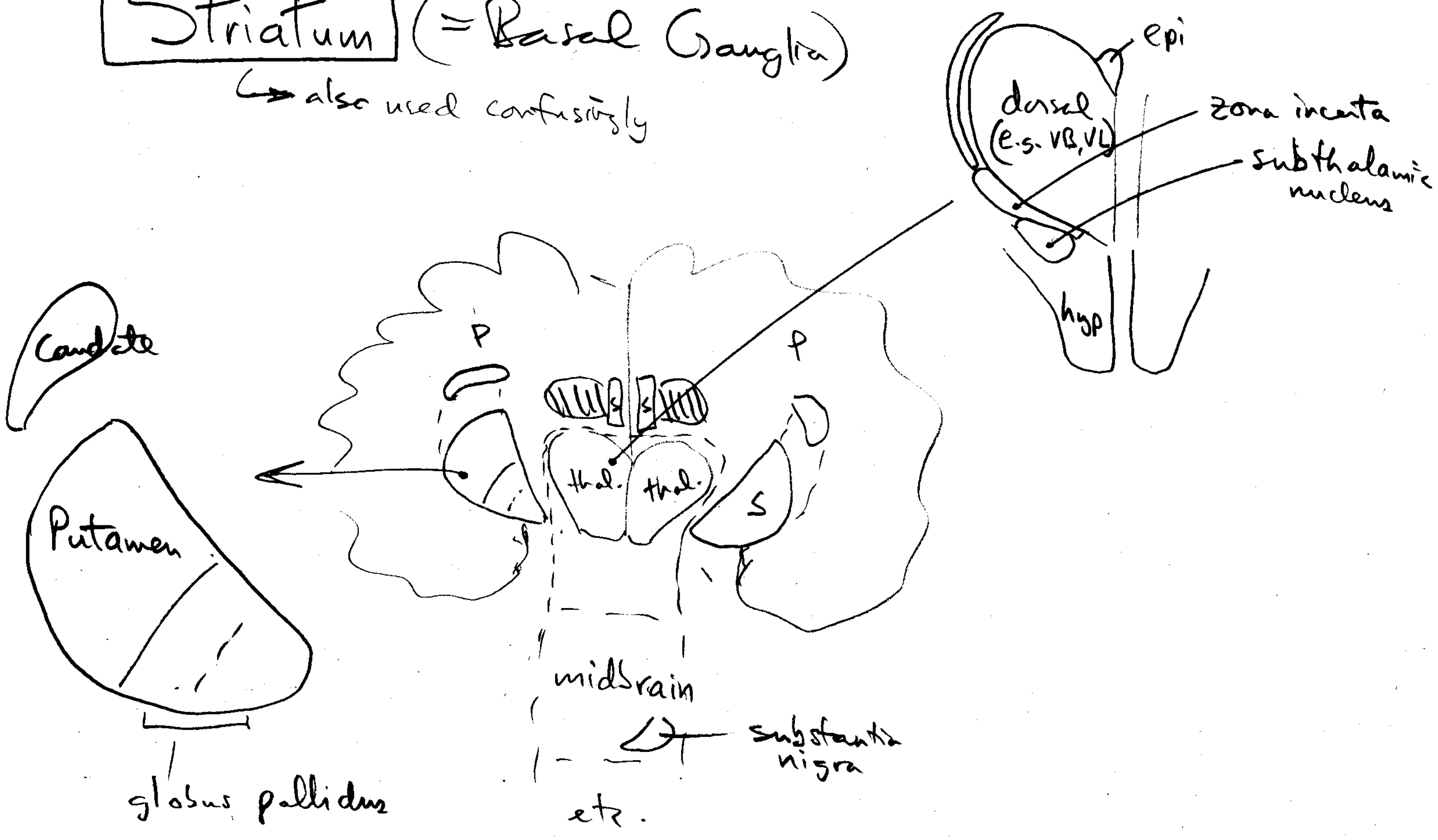
- "anomalous" rectifier
- K+ current activated by <u>hyper</u>polarization
- cf. fast Na+ spike current
- perhaps a mechanism for switching between motor programs

Basal Ganglia deficits



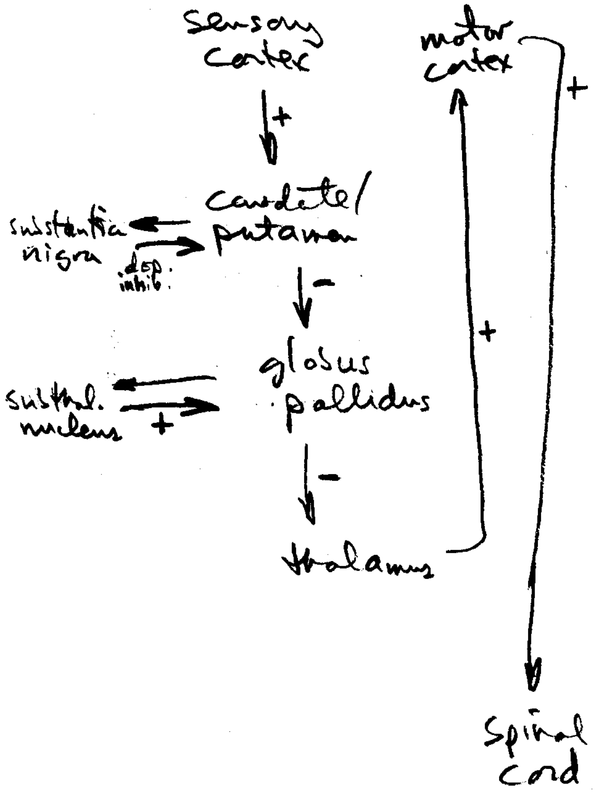
Striatum (= Basal Ganglia)

↳ also used confusingly

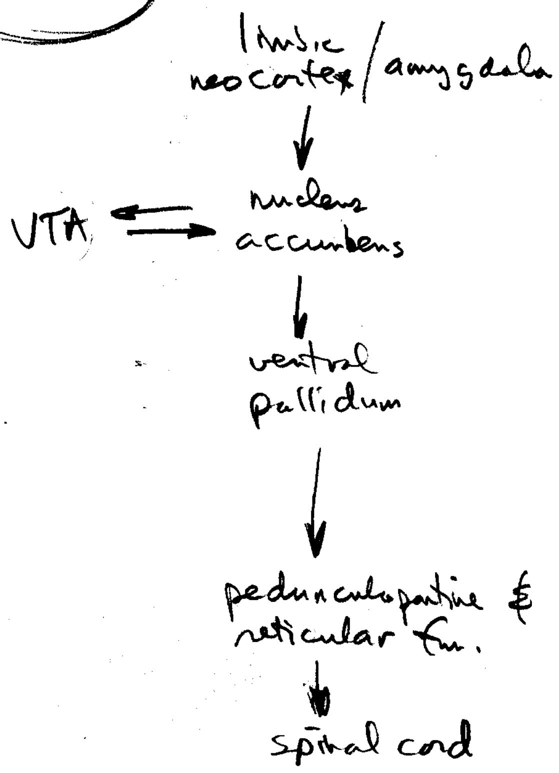


Sensory Striatum

very coarse overview

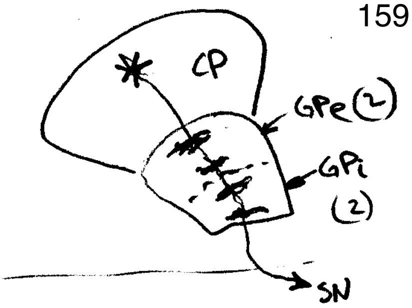


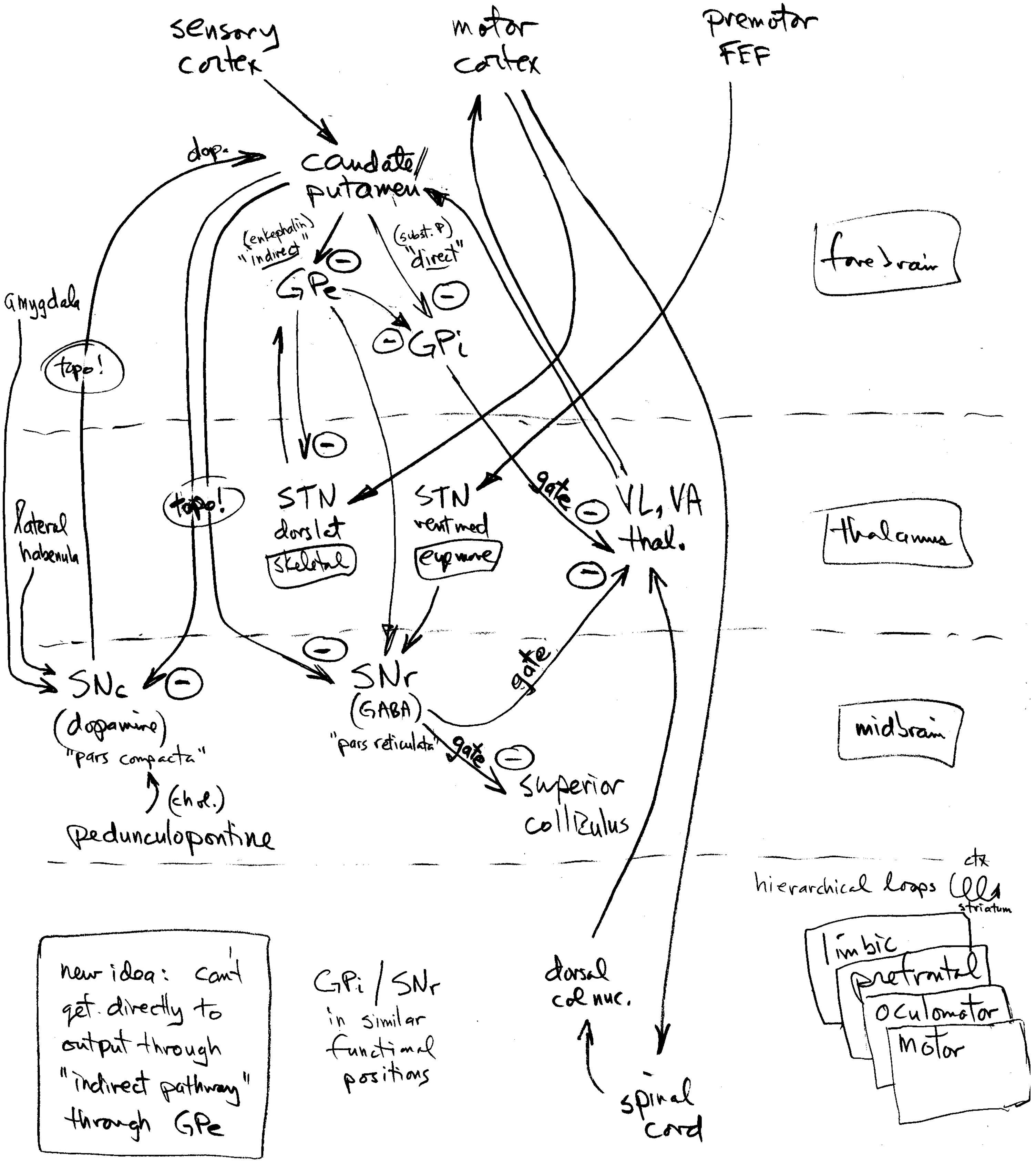
Limbic Striatum



New Striatum

(new A Parent)

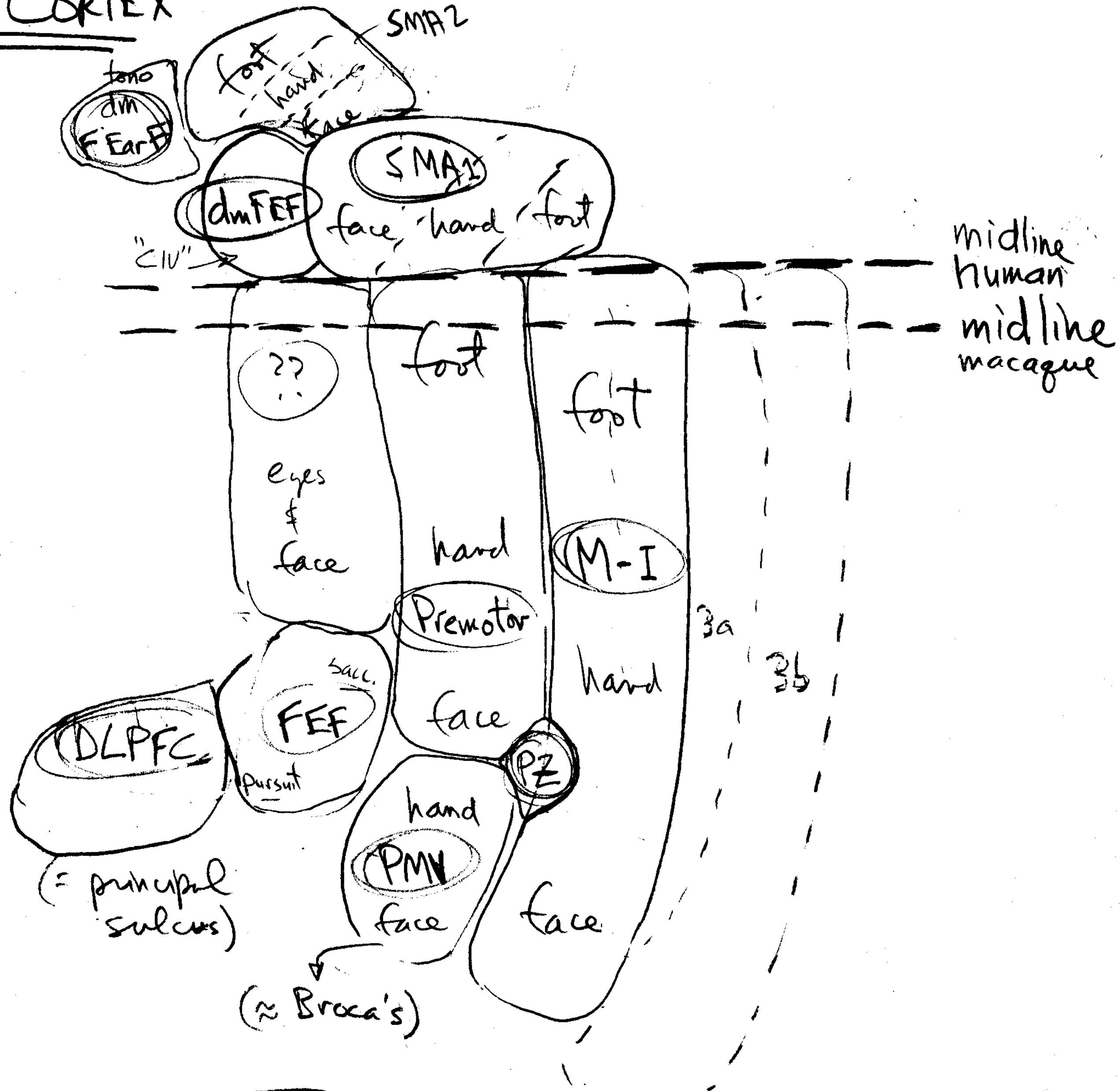


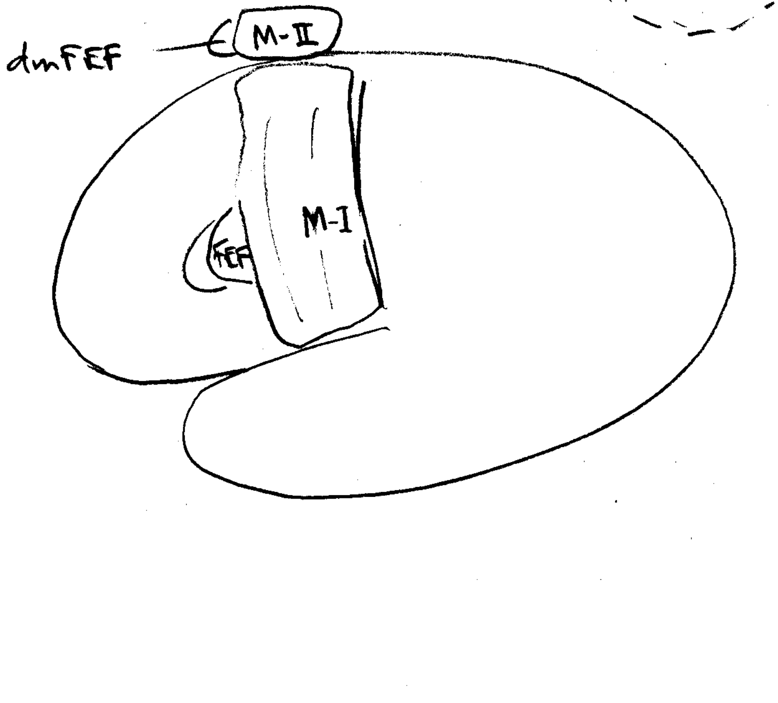


new idea: can't get directly to output through "indirect pathway" through GPe

GPi / SNr in similar functional positions

Motor Cortex



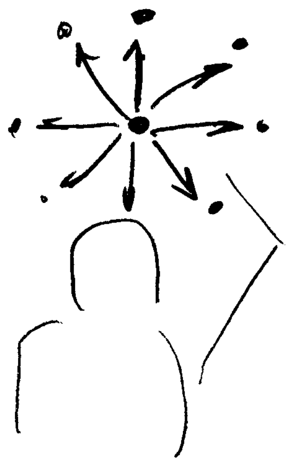


- versus SI: single muscles

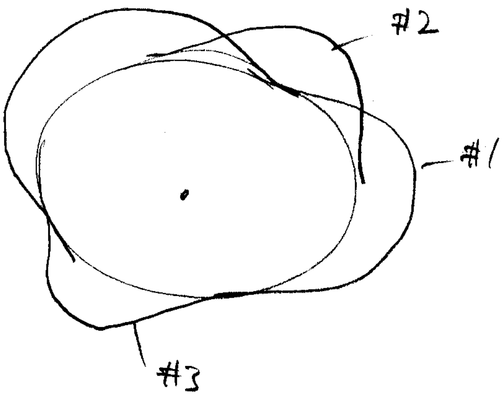
- unlike S-I, multiple reps of muscles & muscle groups (not cut & peel)

- M-I & M-II have own FEFs

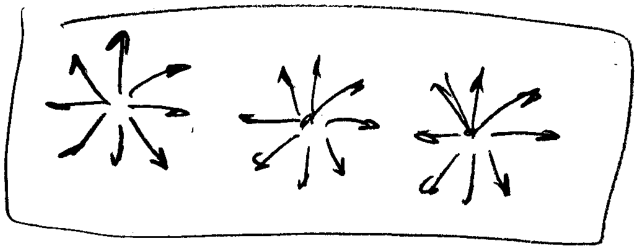
Georgopoulos (1) — bad



- M-I
- population vector



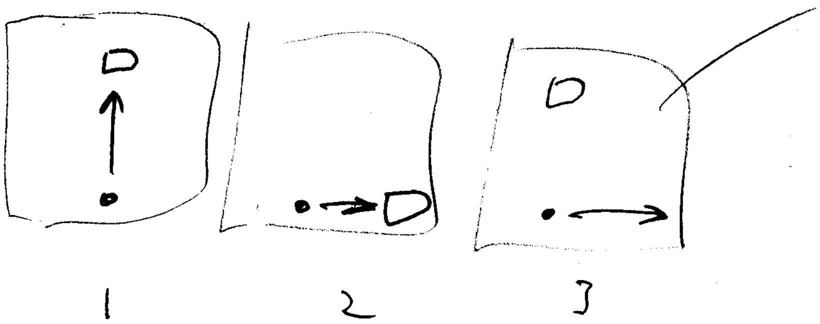
Caminiti

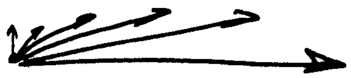


→ *differences suggest that neurons are probably coding muscle pulling direction, not direction in space

---

Georgeopoulos (2) — good

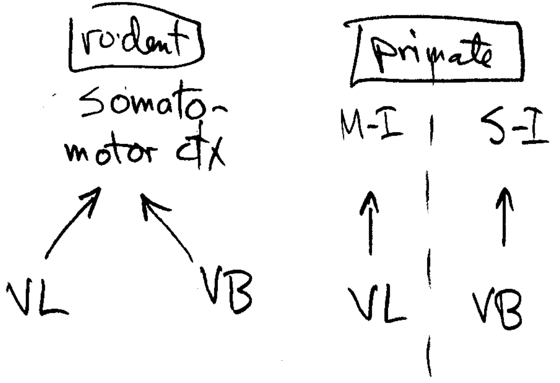




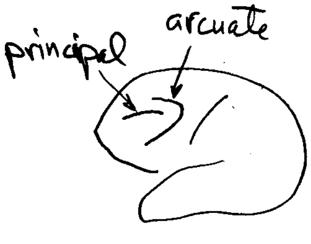
- like mental rotation
- think of as higher level programming leaking through into primary mot.
- rotation vs. ampl: ↳ →

Motor Ctx (parietal) Stim

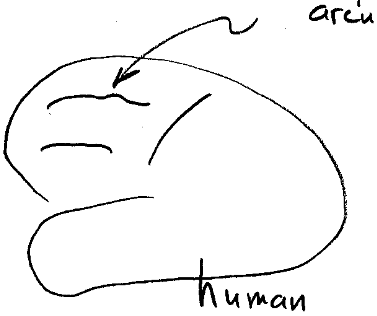
- muscles vs. movements
- EMG of 'everything' is hard
- Graziano
  - long stim trains
  - position in extra personal space
  - cf. frog spinal cord!
- Stepniewska/Kaas
- rodent vs. primate
  ↳ major diff thalamic afferents



- sulci (human: principal + top arcuate)

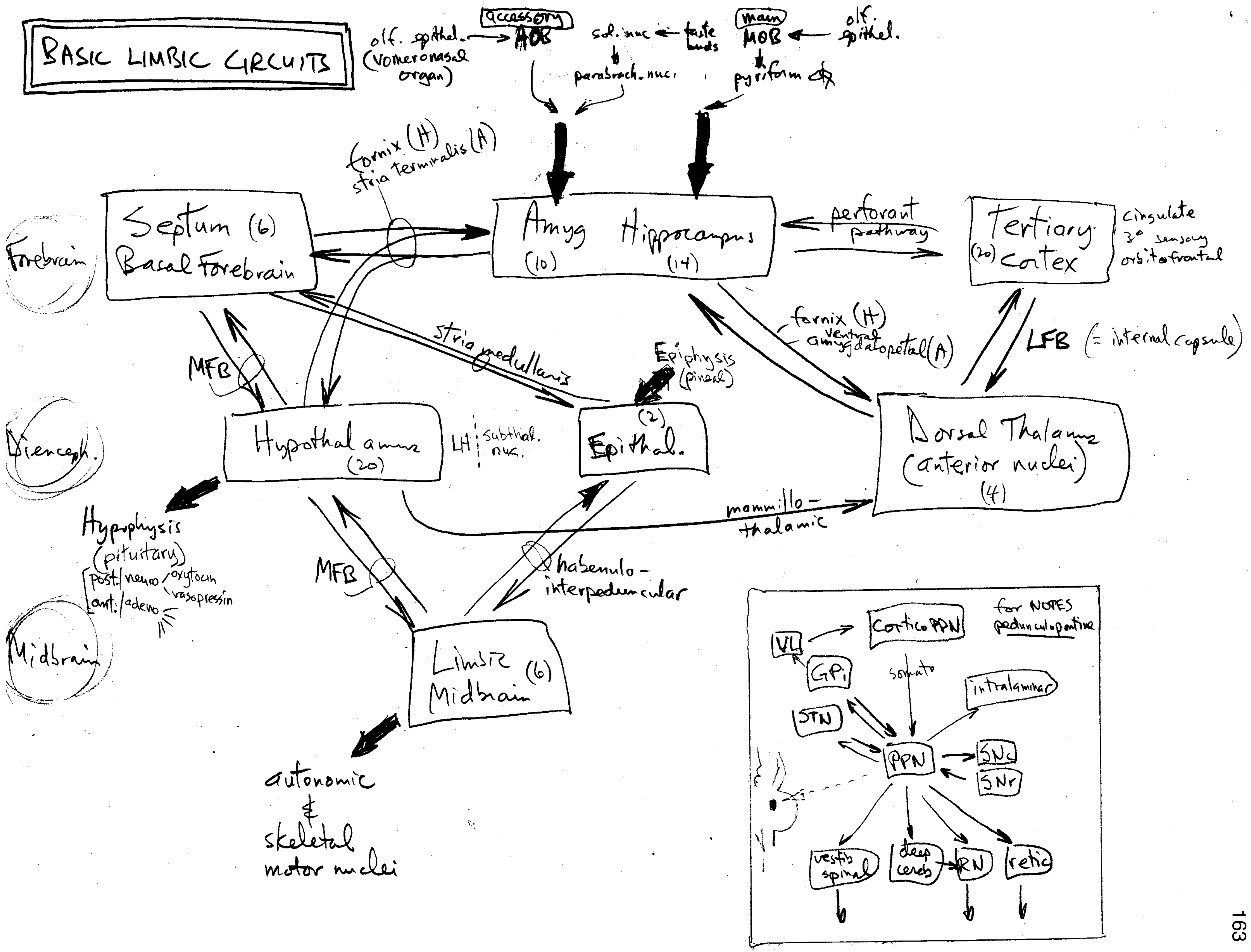


macaque



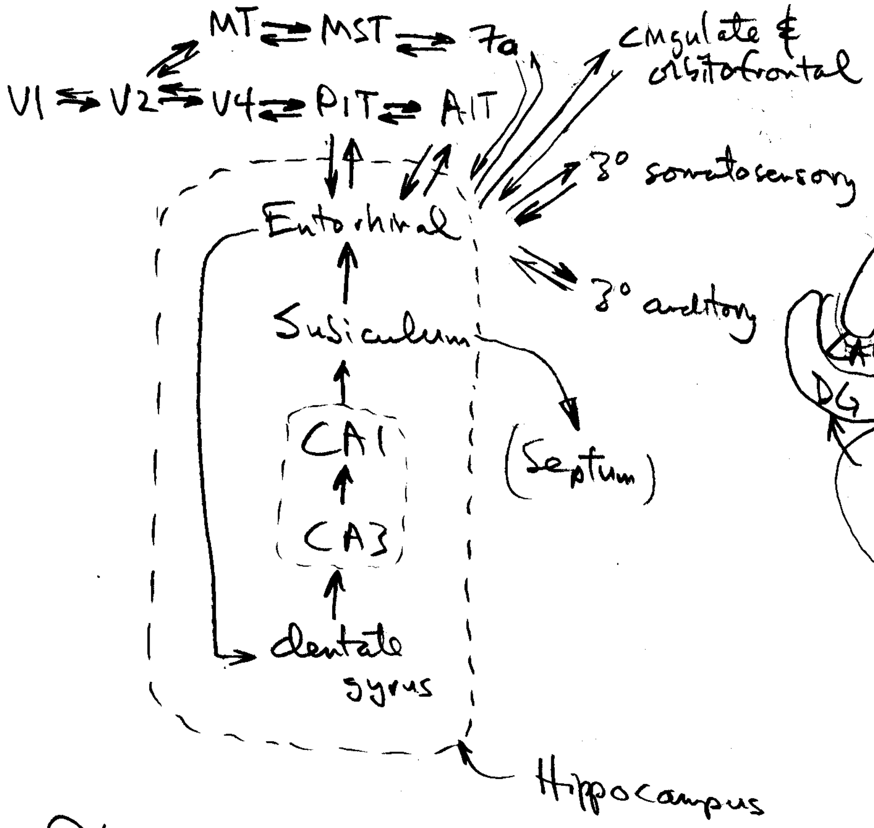
human

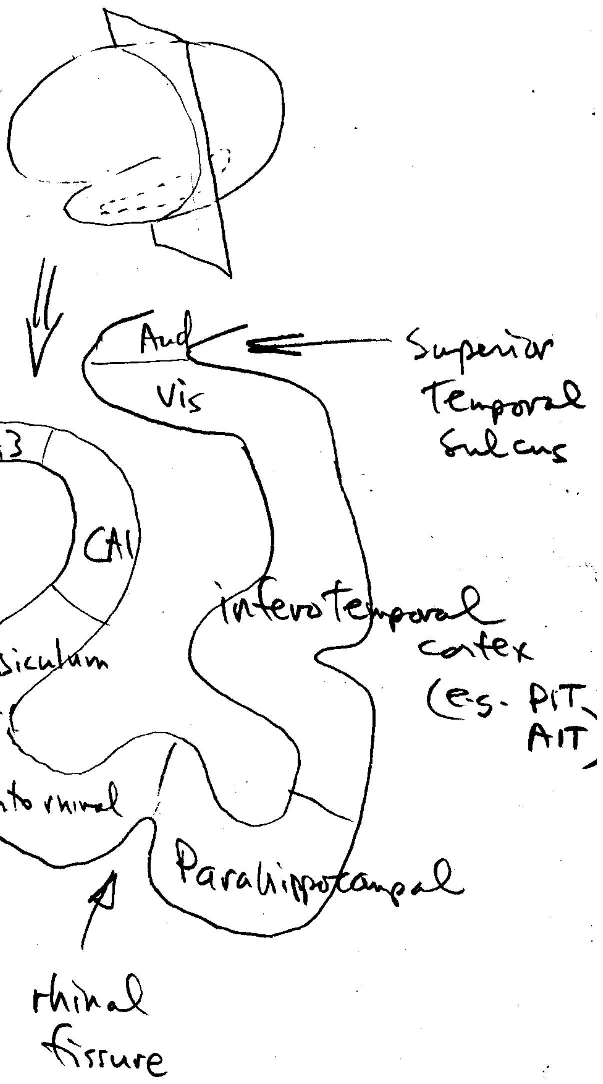
BASIC LIMBIC CIRCUITS



Hippocampus

Anatomy





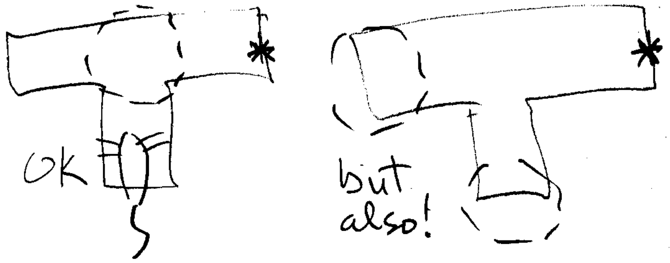
Physiology

- place cells in CA fields
  - history of discovery of place cells
  - modern study of place cells
    - radial arm maze < distal cues to place; works in dark, too
    - cue-card enclosure
      - cue card
      - scales up to larger enclosure
      - less firing in different shape
      - barrier < clear / opaque
      - no head direction
  - spatial deficits (Morris water maze)
- head direction cells in "post subiculum"
  - no place fields
  - incredible inertial guidance

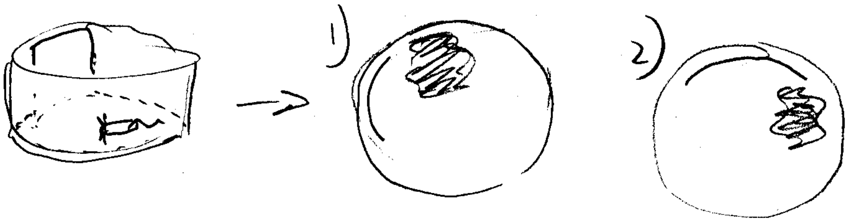
  - distal cues to head dir

Place / Headdir / Grid

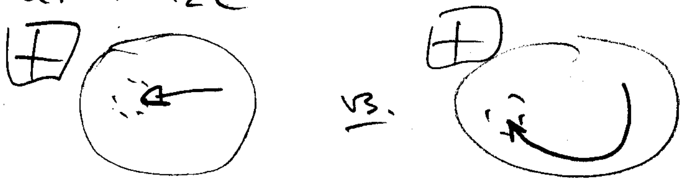
1) James Olds
   - missed discovery
   - theory-driven observation



2) Basic place cell



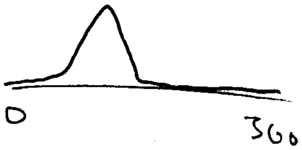
3) Water maze

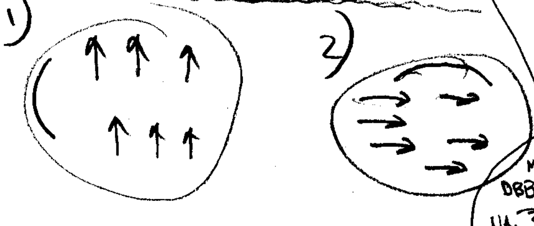


4) radial arm maze < local / distal

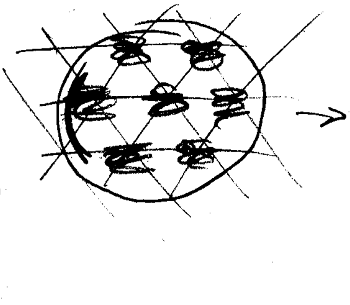
4b) move cue card in view → reset!

5) Head dir cell





6) Grid Cell
- med entorhinal ctx
- size: deeper → larger

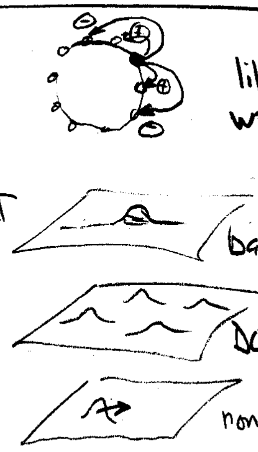


7) Elaboration
- shape □ → ○
- 3D spiral
- 2 rooms
- barriers

8) head fixed VR navigation: place cells <u>OFF</u>! → req. vestib./real movement

*

Attractor Network



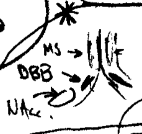
1D ring — like WTA

2D sheet — basin

DOG

non-mono

*



hip outp → lat septal
↳ med septal

theta from ↳ diagonal band Broca

*

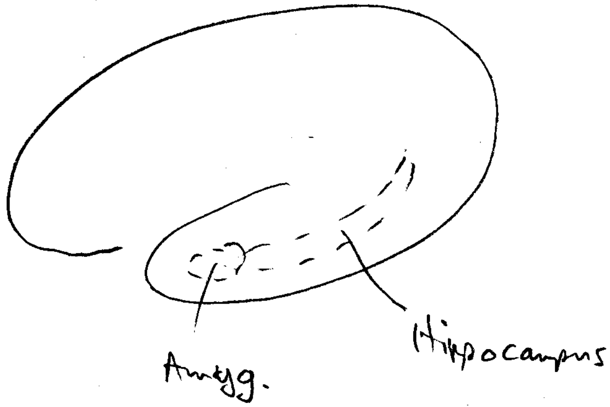
Theta phase precess

outside place field
↳ in phase

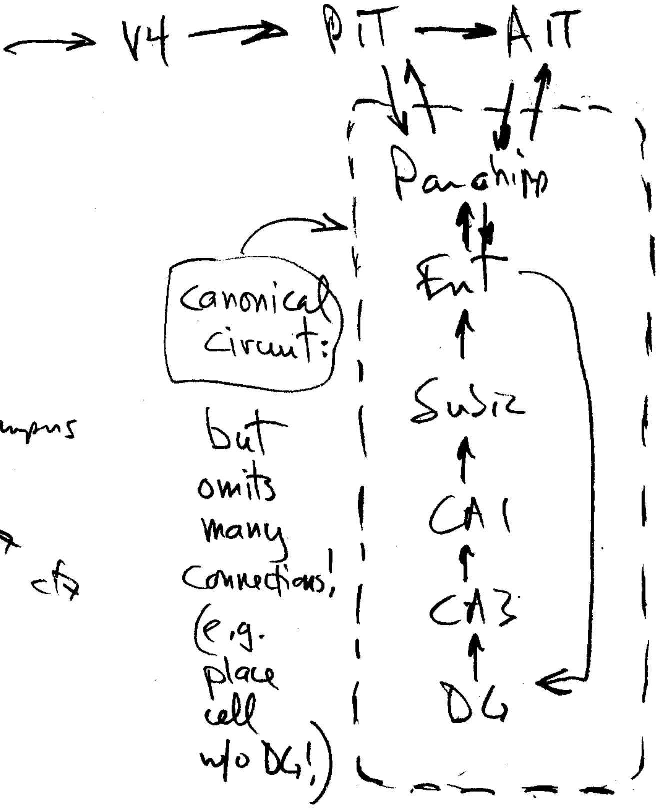
crossing place field
↳ more and more in advance of theta
↳ antiphase in middle
↳ in phase leaving

N.B. can have place fields w/o theta!

H.M.



Entorhinal ctx
parahippocampal ctx



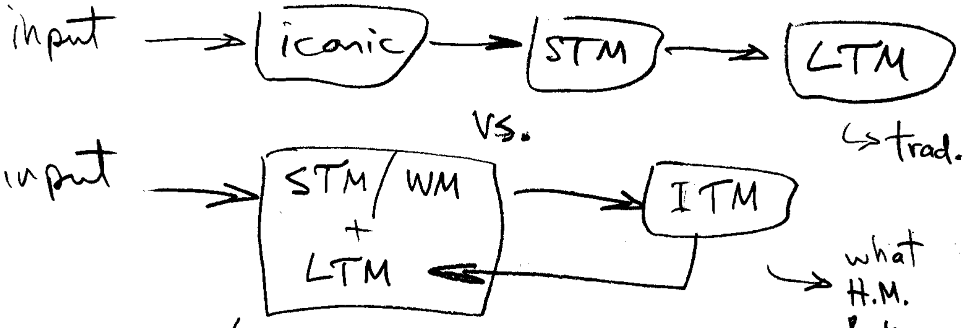
but omits many connections! (e.g. place cell w/o DG!)

Evidence

- no new memories
- retrograde memory loss for a year
- preserved 'motor' learning
  - maze → explains animal expts
  - abstract → Tower of Hanoi

Interpretations

1. explicit/conscious memory/declarative



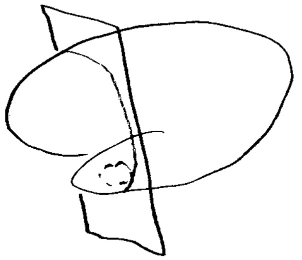
→ what H.M. looks like!

2. procedural/~~motor~~ separate
   ↳ new pathways: → striatum, → cerebellum!

H.M. vs. place cells??

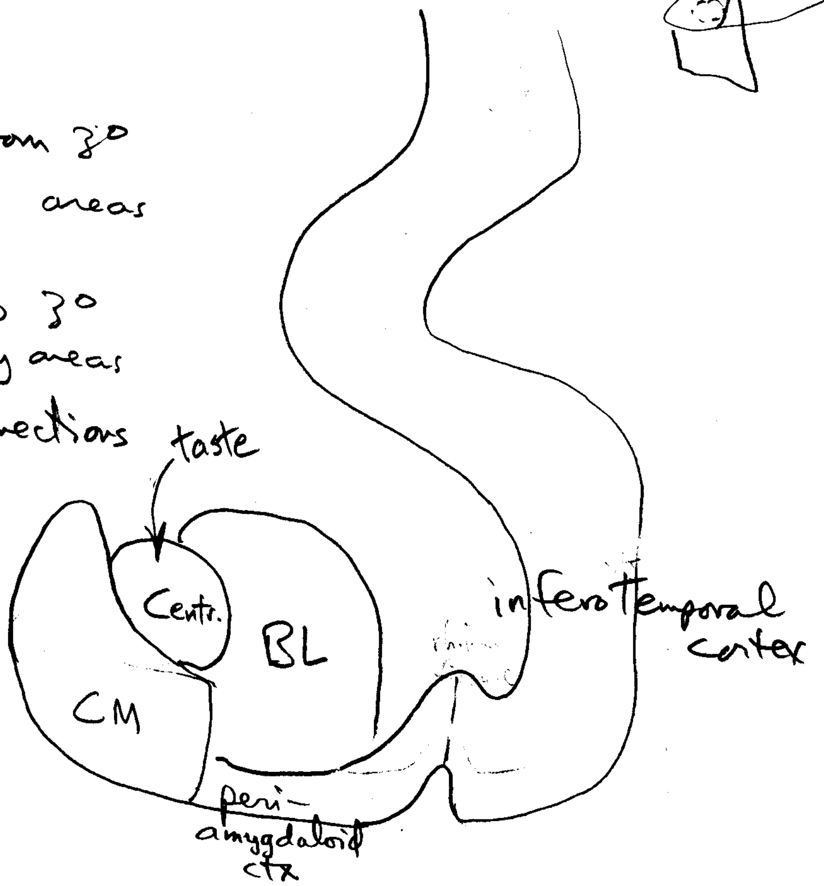
- ITM vs. "you are here now"
- effects of lesions disambiguate:
  - hipp. proper ⇓ "inertial guidance syst"
  - pararhinal parahippocamp ⇓ "ITM"

Amygdala



Anatomy

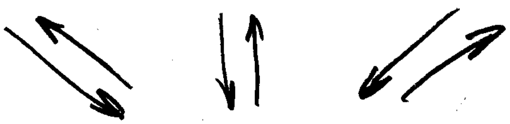
- inputs from 3° sensory areas
- outputs to 3° sensory areas
- limbic connections



3° Somatosensory (e.g. 5)

3° Aud (e.g. TpT)

3° Visual (e.g. PIT)



Basolateral Amygdala

Physiology

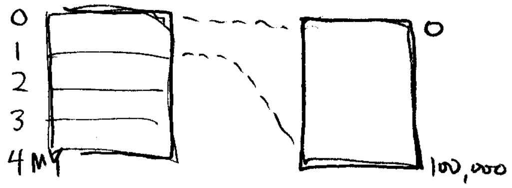
- Kluver-Bucy
  - amyg
  - IT
- limbic connections
  - "violence"
  - "not very good wine" cells ☺
- Mishkin & cross-modal matching task deficits

Origin of LANGUAGE

2 pre-adaptations

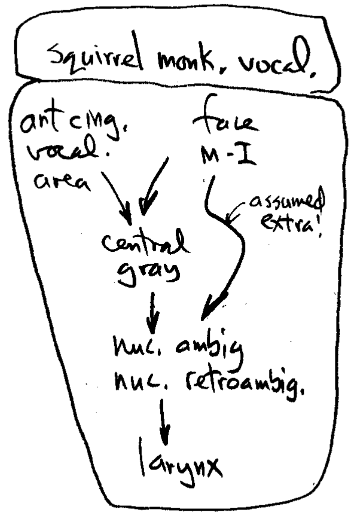
- primate evolution

- paleoanthropology



- ape language — Washoe / Sherman/Austin / Kanzi, Alex
- Gallup mirror test

#1 - birdsong vs. calls, sexual selection, whales
- hommin vocal theory



- main distal sense — primates / bats / electric / snakes / catfish / platypus
  - └ blind
- "picture theory" (vs. IT)
- word recog. (Potter pic expts)
- Lakoff / Fauconnier / Jackendoff

#2 - fictive scene comprehension
- film
- [polysemy → "line" < tiny context (cf. alanine) / context is key
  anaphora → "<u>that</u> fictive scene comprehension stuff"

DNA, protein self-assembly