INTRO

Readings / Tests / Course Outline

Pre-dispositions / folk theory vs sci; see how things are

10%
cell doctrine
smell brain
sex diffs
L/R

Taking notes
can / multi-task analogy
type vs write
drawing

Separate quit
400 parts
repeat
3 planes / nissl-myelin
Golgi

Evolution
comparative function

Machines < not directly for machine learning
for engine given biological raw material
The "Cell Doctrine"
(default theory of human cognition in absence of any information about brain organization)
from Descartes
(a pneumatic crossbar switch in the pineal controlled by the mind)
**Nernst Potential**

\[ E_{ion} = \frac{RT}{F} \cdot \frac{1}{2} \cdot \ln \left( \frac{[I_{out}]}{[I_{in}]} \right) \]

\[ \text{Nernst eq: } \rightarrow \text{reversal potential or equilibrium potential} \]

\[ \text{or the membrane potential at which no current flows when channels for this ion are opened} \]

---

**Summary**

A

- \( V = IR \)
- \( I = Vg \)
- voltage clamp

<table>
<thead>
<tr>
<th>ATP</th>
<th>( E_{ion} )</th>
<th>( V_{cell} )</th>
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B

- \( K^+ \)
- \( Na^+ \)
- \( Cl^- \)

\( Na^+ \) - start at 0, balanced
\( Na^+ \) - a few \( K^+ \)'s leak out
\( K^+ \) - 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

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**Important Point:**

- Nernst potential describes a target voltage for an ion
- the present membrane potential is affected by other things!
**Channels**

- Electric current associated w/ single channel openings
- (clamp for feedback reg. up V)
- Individual channel openings visualized
- 7000 ions/event \((10^7/\text{sec})\)
- Conductance: \(\sim 20 \text{ pS} \]
- Density: \(100 - 1000/\mu\text{m}^2\)

**Patch clamp**

- Extremely low noise

**Patch clamp circuit**

- S is Siemens

\[
\text{Conductance (Siemens)} = \frac{\text{current (amps)}}{\text{voltage (volts)}}
\]
Structure of the human Na$_{\text{1.4-\beta1}}$ 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 for 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 = V/g

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

[actual]

activation
detachment
opening sets off both!
inactivation
da inactivation

direction of propagation

ready to fire

membrane capacitance discharging

N⁺ open

K⁺ open

refractory
Collision test to verify antidromic

antidromic

Stim

record

but could also be an afferent

Stim

record

collision test

Record ± Stim, then record

collision

no collision

more complications

Stim

antidromic

prolentric
Voltage-Sensitive Other Neuronal Ionic Currents

**Excitatory (inward) Na⁺, Ca⁺ Currents**

- $I_{\text{leak}}$
- $I_N$
- $I_{\text{Na}}$
- $I_{\text{Kp}}$

**Inhibitory (outward) K⁺ Currents**

- $I_{\text{leak}}$
- $I_{\text{Na}}$
- $I_{\text{K}}$
- $I_{\text{L}}$
- $I_{\text{C}}$
- $I_{\text{m}}$

**Other Neuronal Ionic Currents**

- $I_{\text{L}}$
- $I_{\text{C}}$
- $I_{\text{K}}$
- $I_{\text{m}}$

**Voltage Clamp**

- $V_{\text{m}}$

From Ziemniak & M.C., 2019
How $I_T$ ("transient") leads to bursting

**Voltage clamp**

- No voltage clamp
- Simple stim + 2 diff bias

$I_Na$ (inward, depolar.)

$K^+$ (outward, hyperpolar.)

$I_{Ca}^+$ (inward, depolar.)

$I_T$

**N.B.** also time dep. drifts

- $-60$ to $-65$ mV

- H-H

- vs. $I_Na$, higher volt before inact.

$-85$ mV

Membrane potential, V

Spikes from Ohm's law

Overall shape from inactivation $I_T$

No bursting

Like squid, lower freq.

Membrane potential, V

$-60$ mV

Another example:

$I_A$ (outward) can cause delayed firing (firing starts after it inactivates)
**Synaptic Potentials**

- caused by neurotransmitter-gated channels
- mostly on dendrites

![Diagram of synaptic potentials]

**AMPA glutamate-gated Na⁺**

- "excitatory sodium"
- passes K⁺ as well
- \( E_{Na} \approx 0 \text{ mV} \)
- not voltage-sensitive

**NMDA glutamate-gated Na⁺, Ca⁺⁺**

- excitatory
- also passes Ca⁺⁺
- voltage-dependent
- blocked at rest

**GABAₐ K⁺**

- hyperpolarizing inhibitory
- slower than AMPA
- \( E_K \approx -100 \text{ (mammalian)} \)

**GABAₐ Cl⁻**

- shunting inhibitory
- no effect at rest
- \( E_Cl \approx -75 \text{ mV} = \text{rest} \)
Channel Families

4+1
mAChR (nicotinic acetylcholine receptor)

5-HT3

GABA_A

4
G protein-coupled receptors
non-NMDA

* NMDA

7
GPCRs - G-protein coupled receptors

mAChR

adrenergic dopamine - D1, D5
D2, D3, D4

5HT-1, 3, 4
* glutamate
* GABA_A

neuropeptide
**NMDA Channels**

Detecting pre-post correlation

\[ \text{Ca}^{++} \]

Local Clusters

\[ \text{Ca}^{++}, \text{Na}^{+} \rightarrow \text{vol} \text{tage change in cortex but not hippocampus} \]

- **N.B.** one synapse PSP not enough depol. for NMDA
- detect Corr. across synapses
Spike-timing Dependent Plasticity

Old Idea

\[ \text{postsynaptic spikes before} \rightarrow \text{postsynaptic spikes after pre-} \rightarrow \text{potentiation} \]

\[ \text{pre-synaptic spike time} \]

STDP

\[ \text{postsynaptic spikes before} \rightarrow \text{postsynaptic spikes just after presyn.} \]

- Idea: synapses detect causality: if input caused cell to spike \( \uparrow \)

- if cell spiked but input just missed helping out, \( \downarrow \)

and

- diff by \( \pm 100 \text{ msec} \rightarrow \text{no effect} \)
\[ C_m \frac{dV_{in}}{dt} + I_{ions} = I_{ext} \]

Ohm's Law: \( I = gV \)

First order kinetics

alternate way of writing

Solve simultaneously numerically integrate

steady state at particular \( V \)

state of gating variables for particular \( V \) at \( t=0 \)

\[ \frac{dm}{dt} = \frac{m_{\infty}(V) - m}{\tau_m(V)} \]

\[ \frac{dm}{dt} = \frac{m_{\infty}(V) - m}{\tau_m(V)} \]

\[ \frac{dh}{dt} = \frac{h_{\infty}(V) - h}{\tau_h(V)} \]

\[ \tau_m(V) = \frac{\alpha_m(V)}{\alpha_m(V) + \beta_m(V)} \]

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

\[ I_{ions} = \left( \frac{m^2}{g_{ion}} \right) h \cdot m \cdot (V_{in} - E_{ion}) \]

voltage gap of first, backward time constant for \( m, h \)

fit V-clamp data get these curves

add data fit mn data
Voltage dependence of forward and backward rate constants for Na activation and inactivation (plots of empirical functions)

Why are spikes propagated backwards but not forwards in dendrites?

Nat inactivation, membrane potential:

1

+ stim/rec

0 10 20 30 40

25 mV

not inactivated

spike triggered

stim

+ rec

0 10 20 30 40
Noise, Stochastic Processes, Firing Patterns

- critical slices
- silent vs. background

↑ step

stim

histogram repeated trials

noise input

stim

histogram

↑

spike generation process is deterministic (!)

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

- real neurons have highly variable firing patterns in the cortex
- to simulate critical variability in firing of stimulated neuron in slice, must inject bursty non-Poisson distributed spikes filtered through PSP
Integrate & Fire

1) PSP conductance waveform

2) Spike function (B. H.H.)

3) Linearly add up conductance waveforms at one connection

4) Add up for all conductances like this

5) \[ \Delta V = \frac{1}{C_m} \sum_{\text{channels}} \left( V(t) - E_{\text{channel}} \right) \tilde{g}_c(t) \]

From:
1) Ohm's law \( I = Vg \)
2) \( \Delta V = \frac{\sum I}{C} \)

\[ \Delta V = \frac{1}{C} Vg \]
Cable Theory → Compartmental Models

\[ V = \lambda^2 \left( \frac{\partial^2 V}{\partial x^2} \right) - \left( \frac{\partial V}{\partial t} \right) \]

\[ \lambda = \frac{T_m}{r_i} = \sqrt{\frac{R_m}{R_i}} \frac{1}{\pi r^2} \]

\[ r_mC_m = (\frac{R_m}{2\pi r})(C_m + 2\pi r) \]

Cond vel:

\[ \frac{dx}{dt} \propto \lambda \propto \sqrt{\frac{T_m}{R_i R_mC_m}} \]

Compartmental models

- Replace constant PDE eq w/ ordinary
- Each compartment isopotential
- Non-uniformities between compartments
- Assume extracellular ground

[Diagram of neuron with compartments and connections]
Definitions

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 work (and finite time!) to charge capacitors

\[ V = IR \] (Ohm's law)

\[ Q = CV \]

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

\[ \frac{dQ}{dt} = C_m \frac{dV}{dt} \]
Simplest Circuits

Resistor only

- Circuit diagram with switch closing, output current of source, $I_R$ and $V_R$

Capacitor only

- Circuit diagram with output current of battery, $I_c$ and $V_c$

N.B. 
- no resistance so current very high
- capacitor charges almost instantly
- a partly-charged capacitor still non-infinite effective resistance
Simple Circuit

Series

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

Parallel

- drifts (with finite current source)

* if battery really big, would look instantaneous
Electrotonic/Dendritic Current Flow

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

- why it's called cable theory

$R_m \gg R_L$
ON-STEP, OFF-STEP, SHORT PULSE IN SPACE & TIME

Input

Space

Time

plot $V_I$ (conductance fixed)

WHY DOES INCREASED DIA INCREASE CONDUCTION VELOCITY?

$0^*$ = one unit of area

$\frac{R_L}{M}, \frac{C_M}{M}$

Small dia

Large dia

effects of expanded area

$\frac{3}{3}$ vs. $\frac{R}{R}$

$\frac{V}{V}, \frac{C}{C}$

really good (big dia)$^2$

both bad

$R_L$ ---

beats

$R_L$, $C_M$ ---
Myelination

- myelin has low C
- conduction velocity of
  electrotonic pulse faster as result
Linsker(1) Basic Idea

1) Linear summation

\[ O_j = \sum_i I_i w_{ij} \]

2) Simple Hebb

\[ \Delta w_{ij} = \text{rate} \times Q_{ij} \]
\[ Q_{ij} = I_i \times O_j \]
\[ \Delta w_{ij} = \text{rate} \times I_i \times O_j \]

3) Put together into one equation

\[ \Delta w_{ij} = I_i \left( \sum_{i \neq \text{other}} I_i \left( \sum_{j \neq \text{other}} w_{ij} \right) \right) \]
\[ Q_{i, \text{other}} = I_i I_{\text{other}} \]
\[ \Delta w_{ij} = \sum_{\text{other}} Q_{i, \text{other}} W_{\text{other}, j} \]

\[ \Delta w_{ij} = K_1 + \sum_{\text{other}} \left( Q_{i, \text{other}} + K_2 \right) W_{\text{other}, j} \]

weight decay

set no-change point

\[ \text{weight vector} = Q \text{ vector} \times \text{weight vector} \]
\[ = \text{vector} \times \text{weight vector} \]

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-pos

- as soon as average weight to a unit is slightly pos (or neg), \( \Delta w \) for current weight goes pos (or neg)

- the next cycle, the \( Q_i, \text{other} \) will still average zero, \( \Delta w = \sum Q_i, \text{other} \) will be more pos (or neg), since the other weights are more pos (or neg)

What endpoint \( Q_i, \text{other} \) looks like for layer 2
Why the RF center weights go up

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) \( k_c \) causes increase in weights
   b) because nearby inputs from layer 2 correlated
      and, there are more units in center
   c) \( k_s \) drags down everybody; center stays positive from b),
      surround goes neg.

2) What the \( Q_i, \text{other} \) looks like for layer 3

   update for weight coming from receptive field center

   \[ \sum Q_i, \text{other} \times \text{other} \]
   lower because few nearby correlated
   units hook up and add to sum (far away
   units that are connected are uncorrelated and add little)

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

   update for weight coming from receptive field center

   \[ \sum Q_i, \text{other} \times \text{other} \]
Linsker (4)  What happens in layer 4
(symmetry-breaking)

A fourth layer unit

\[ \sum Q_i, \text{other will be zero for these} \]
\[ \sum Q_i, \text{other will be negative for these} \]
\[ \sum Q_i, \text{other will be positive for these} \]

Correlation of nearby centers \( \Delta w \uparrow \)
Anti-correlation of center and midrange surround \( \Delta w \downarrow \)
Far away cells uncorrelated \( \Delta w = 0 \)

- It is possible to calculate a Hebb energy
- Each \( \Delta w \) will always change to lower Hebb energy
- A receptive field with elongated excitatory & inhibitory regions has lowest energy

Symmetric stimulus did not
Why Eigenvectors?

\[ \Delta w_i = k_1 + \frac{1}{\text{#inputs}} \sum_{\text{other}} (Q_{i, \text{other}} + k_2) w_{\text{other}, j} \]

Change in weight:
\[ \Delta w_i = \nabla \text{vector} \]

Change in weight vector:
\[ \nabla \text{matrix} \times \nabla \text{vector} \]

Linear Transformations

\[ Ax = b \]

Eigenvalue Problem

\[ Ax = \nabla \text{vector} \]

Covariance Matrix

Eigen vectors of stim covariance matrix 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 changes shape

- eigenvalue/eigenvector solution tells which directions (i.e., patterns) are most likely to arise from given Q (correlation matrix)

- eigenvectors are orthogonal!
**Recurrent Intro**

1) Feedforward learning (= weight change)
2) Now: recurrent, dynamics w/o weight change

**Attractor Networks**

Examples:
- Category 1
- Category 2

Attractor pattern:
- Unit 1 = 0.9
- Unit 2 = 0.1

Activity units:
- Unit #1
- Unit #2
Recurrent - Why $\Delta E$ always $\uparrow$ or $\Phi$ (2)

Update

\[
\text{Output}_j = \begin{cases} 
1 & \text{if } \sum_i \text{Input}_i \cdot w_{ij} > 0 \\
-1 & \text{if } \sum_i \text{Input}_i \cdot w_{ij} \leq 0
\end{cases}
\]

Energy

\[
\text{Energy} = -\sum_i \sum_j \text{Input}_i \cdot w_{ij} \cdot \text{Output}_j
\]

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

Lyapunov function

show that update of any unit can only $\text{[reduce or leave same]}$ energy
Recurrent symmetry

- Intuitive idea: $p$ energy or "frustration"

1) \[ \begin{array}{c}
+1 \\
+1 \\
+1 \\
-1
\end{array} \quad > \quad \begin{array}{c}
+1 \\
+1 \\
+1 \\
+1
\end{array} \]

2) \[ \begin{array}{c}
+1 \\
+1 \\
+1 \\
-1
\end{array} \quad > \quad \begin{array}{c}
+1 \\
+1 \\
+1 \\
+1
\end{array} \]

- Update of one unit reduces (or keeps same) energy terms from its input connections
  \[ \rightarrow \text{(definition of update rule)} \]

- Output connection weights $\rightarrow$ unknown effect

- If symmetry, output connection terms term-by-term same as input connection terms

- $\therefore$ If symmetry $\rightarrow$ stable states
Winner-take-all

Start state

Stable state

- 3 stable states
Word-Rec model as Hopfield

- word-like non-words speed letter rec.
- deal w/partial occlusion/noise
**Back Prop Summary**

linear_output_j = \sum_i input_i \cdot w_{ij}

Squashed_output = \sigma (linear_output)

\( \sigma(x) \) e.g. a sigmoid

change weight to reduce error:

\[ \Delta w = \text{learning rate} \cdot \delta \cdot \text{input} \]

where:

\[ \delta_{output} = (\text{target} - \text{actual}) \cdot \sigma'(linear_output) \]

\[ \delta_{hidden} = (\sum \delta_{output} \cdot w) \cdot \sigma'(linear_output) \]

---

**Feedforward pass example**

linear_output_j = \sum_i input_i \cdot w_{ij}

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

= 10

Squashed_output_j = \sigma(linear_output)

= \sigma(10)

= \sigma(6)

= 7
Feedforward

Weighted Sum (1)

Output \( j \) =

\[ \sum_i \left[ \text{Input}_i \times \text{weight}_{ij} \right] \]

j's are outputs

l's are inputs

Example of learning problem

output patterns A B C D
input patterns 1 2 3 4

picture of dog as numbers
just dog unit on
picture of cat
just cat unit on
picture of house
just house unit on
How to get output error

```
desired = 55  
actual = 41  
```

\[
error = \frac{\text{desired activation}}{\text{actual activation}}
\]

\[
output = \sum (\text{input} \cdot \text{weight}) = \\
= 6 \times 2 + 3 \times 5 + 7 \times 2 \\
= 12 + 15 + 14 \\
= 41
\]

Problems

\text{w/ 2-layer}

Netwrk

```
(0,1)  
```

\text{Problem}

\text{Input Patterns}  \quad \text{Output Patterns}

```
0 1 \rightarrow 0 \\
1 0 \rightarrow 0 \\
1 1 \rightarrow 1 \\
0 0 \rightarrow 1
```

View a scatter plot of input space
How to get hidden error

output pattern calculated by

1) input → hidden

\[ \sum \]

\[ \text{error output} = \text{desired} - \text{actual} \]

error hidden = \[ \sum_{\text{outputs}} \left[ \text{error output} \times \text{weight to hidden} \right] \]

input pattern applied to input units
multiple layers
non-linearity
parity-like class (non-linearly separable)

- if not 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:

input space
\[ \rightarrow \]
scale diag (linear)
\[ \rightarrow \]
squash (non lin)
\[ \rightarrow \]
decision (linear)
What's "bad" about backprop

("bad" → bio-implausible)

#1 → Supervision

#3 have to specifically affect these weights
**EARLY DEVELOPMENT**

- **Egg**
- **Early division**
- **Gastrulation**
- **Blastula**
  - **Neural crest**
  - **Neural tube**
  - **Notochord**

**Forms neural plate**

**Forms notochord**

**Gastrula**

**Neurula**

**Neural tube**

**Rolling up of neural plate**

**Migration / Birthdates**

**Eye Development**

- **Brain**
- **Optic cup**
- **Pigment epithelium**
- **Retina**
- **Lens placode**
- **Lens**
- **Eyelid**
Somites & Gill Arches

Somites (Hox nested genes)

Gill arch (DLx nested genes)
(Branchial arch)

Motor neurons
innervating
somites

Motor neurons
innervating
gill arches

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

Somites

1) 6 eye muscles
2) 3
3) 4
4) 5
5) 6
6) 7
7) 8
8) 9

location of motor nuclei in brain
(4) III oculomotor
(1) IV trochlear
(1) VI abducens

Gill Arches

1) jaw muscles
2) ear muscles
3) III hypoglossal
4) larynx, thyroid muscles
5) 6
6) 7
7) nucleus ambiguus

location of motor nuclei in brain

principle motor nerve (V)

facial motor nucleus (VII)
"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 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 cruising 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.

- Want to stamp on SUV accelerator? 
  $\rightarrow$ 2 football fields solar cells

- All batteries produced in one current year 
  $\rightarrow$ 1-2 min grid storage

- Grid = Cars

- Wind + Solar growth has not even kept up with overall demand growth

\[
\begin{align*}
\text{fossil fuel lifeblood} & \rightarrow \text{from localized bio failure to recycle} \\
\text{(instantaneous)} \quad \text{power} = \text{watts} = I \cdot V \quad (\text{KW}) \\
\text{energy} = \text{watts} \cdot \text{time} \quad (\text{KWh}) \\
\text{barrel of oil} & \leftarrow \text{diesel (cargo cult)} \\
42 \text{ gal} & \leftarrow \text{kerosene} \\
20 \text{ gal gasoline} & \downarrow \\
& \text{25% efficient} \\
\text{"bicyclist"} & \approx 100 \text{ watts} \\
\text{SUV} & \approx 200,000 \text{ watts} \\
\text{one year of 100 watts} & \circ 6 \text{ hours cont./day}
\end{align*}
\]
SCHEMA

forebrain (telencephalon)

thalamus (diencephalon)

midbrain

pons-midbrain junction

related to opposite side of body

related to same side of body

rhombic lip

4th ventricle

Spinal Cord

ventricle

Cereb.
MAIN DIVISIONS

Spinal cord

Sensory (alar plate)
Motor (basal plate)

Dorsal root ganglion
Dorsal horn
Axon
Ventral horn

Medulla

Open 4th ventricle
Taste, somatosensory, auditory
Sensory
Motor

Pons

Cerebellum
4th ventricle
Jaws, eyes, face
Somatosensory, auditory
**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
  - R1/R

- evol. of areas: 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, aggressive behavior, no neurogenesis in
subventricular zone

**Normal**

- 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 exists, suggest opposite

Tallinen (2016)
- coat expanding gel (ctx) over rigid WM-shaped non-expanding armature -> human-like folding
- also, finite element models

max compressive stress
before gyrification
Physical Simulation
(thin outer elastomer gel layer expands over non-expanding gel brain base)

Tallinen et al. (2016) Nature Physics (doi:10.1038/NPHYS3632)
Capybara
(150 lb. guinea pig)

Lab Deer Mouse
(same scale)
4) O'LEYAR EYPT

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**Normal**

- Visual
- Somatosensory

**Transplant**

- Visual
- Somatosensory

**Diagrams:**
- Superior Colliculus
- Spinal Cord

**Annotations:**
- Vis ctx is re-specified
- Goes away later in development
- Fiber grow in an re-specific

---

**Also:**
- Shatz
- LINDAMIRI
- Cohen Tamam
- gene
- expt
Retina

pigment epithelium
(chomping-chomping)
cone

X - parvo
Y - magno
blue - konio

Photoceptors (4 types)
- dark current
- light closes channels → repolarization → less neurotransmitter release

Horizontal cells (2 types)

Bipolar cells (3 types)
- ON, OFF, rod

Amacrine cells (20 types)
- AII, A1-25...

Ganglion cells (10 types)

Ganglion cell types
- OFF, somatotopic
- ON, OFF, blue

X (midget) → (ON) → R cent → G cent → LGN
Y (parvocellular) → (ON) → LGN, colliculus
W (OFF) → LGN, colliculus
direction selective → pretectum (MTN, LTN)
thorn cells → colliculus
Ganglion Cell Types

- this is like brightness & derivative of brightness

\[ f(x) = \begin{cases} 1 & \text{light on} \\ 0 & \text{light off} \end{cases} \quad f'(x) = \begin{cases} 0 & \text{light on} \\ 1 & \text{light off} \end{cases} \]

\rightarrow \text{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)

Y-ON
non-lagged

Y-ON
lagged

"lag" is about 50 msec

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

Reichardt detector
- Surround is inhib (
not OFF)
- Bigger because feedforward
inhib is diagnostic
Maps

Conformal Maps — angle preserving

Can't be conformal w/o cuts w/ force

Circumferential stretching greater than radial stretching

A hemi-field can be nearly conformal
Mirror Image ≠ Non-Mirror Image

Map: Retinal Position $(r, \theta)$ → Cortical Position $(x, y)$

Like $r = \theta(c)$ but $r, \theta$ both have two dimensions

- Steepest uphill direction of $r$ at each $x, y$
- Steepest uphill direction of $\theta$ at each $x, y$
Mutant Belgian Sheepdog

Normal

Retina

\[ T \quad N \quad T \quad N \]

\[ \text{naso-temporal coordinate in retina} \]

\[ \text{dLGN} \]

Mutant

Retina

\[ T \quad N \quad T \]

\[ \text{nose} \]

\[ \text{dLGN} \]

- N/T coordinate has opposite "sign" in two eyes

- \( \therefore \) dLGN aligns left & right retinal points w/ different N/T coordinates in C & I layers

- retina still goes to same N/T coordinate

- lack of chiasm then leads to "taco"

- \( \therefore \) 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).
Visual System Overview

Areas

30 cortical < 50% of total neocortex
10 thalamic < sup pulv
   ret
   inf pulv
   IT pulv
10 midbrain

Connections

- each area connects to ~10 cortical
- perhaps ~800 interareal tracts
- each cortical area has outputs to several of these:
  - frontal
  - striatum
  - sup. collic
  - pontine nuc.
gen. scheme for cortical layers

"lower cortical area"

1. orig input
2/3.
feedforward

4.
in
put
5.
motor
6.
feedback

"higher cortical area"

1. orig input
2/3.
feedforward

4.
in
put
5.
motor
6.
feedback

DLGN
SupCollic
Claustrum
"dorsal" \( \text{MT, V3} \rightarrow \text{MST} \rightarrow 7a \)

"ventral" \( \text{V4, VP} \rightarrow \text{PIT} \rightarrow \text{AIT} \)
- differentiate, then remix
- motion OK w/ complete retinal paraenchymal lesion (w/ contrast > 10%)
<table>
<thead>
<tr>
<th>V1</th>
<th>V2</th>
<th>Properties</th>
</tr>
</thead>
<tbody>
<tr>
<td>blobs</td>
<td>thin stripes</td>
<td>- color selective</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(but cf. Galago)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- large dynamic range (0-150)</td>
</tr>
<tr>
<td>interblobs</td>
<td>interstripes</td>
<td>- orientation selective</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- not color selective but can use color to</td>
</tr>
<tr>
<td></td>
<td></td>
<td>detect orientation</td>
</tr>
<tr>
<td>4B</td>
<td>thick stripes</td>
<td>- direction selective</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Basic Visual Physiology

- non-opponent
- simple-opponent
- double-opponent

Simple cells

Complex cells

"Hypercomplex" cells

Hypercomplex cells

Special complex (= anti-hypercomplex!)

moon x ½ deg vis angle
**Complex Cell Model - Gabor Filters**

- 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 (crossprod 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

vl - cells respond only to real contours
v2 - some cells respond to illusory

[Diagram of a graph with labeled axes]

[Diagram of circular symbols connected by lines]

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
  \( \text{across different stimulus features} \)
  \( \text{across levels of processing} \)
- other examples
  \( \text{Object color} \)
  \( \text{Pattern motion} \)
  \( \text{Complex motion} \)
  \( \text{Texture} \)
  \( \text{ITD} \)
- solved by combining information across space
  \( \Rightarrow \) 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

[Diagram: Sun → Red Apple]

- If spectrum of illuminant is biased (e.g. mostly orange light inside pub) even green objects will be mostly orange (though less orange than orange obj)
- Green objects still look green! → color constancy

- This color constancy will fail in monochromatic light (e.g. sodium vapor lamp) so reqs some range of colors
Aperture Problem For Pattern Motion

Overview

Y cells → LGN → 4Lx → 4R → V2-thick → MT

center surround

center surround

center-surround & oriented

oriented direction selective

oriented dir. selective

Pattern-selective

ture pattern dir

local directions

e etc

e etc

Saito
**View from V1**

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

What Reichardt detector sees in all cases:
- Receptive field aperture
- "Straw"

**View from MT**

Family of local directions consistent with one pattern direction.

Pattern direction #1

Pattern direction #2

\[ \text{local} = \text{pattern} \cdot \cos \theta \]

\( \Delta \text{x} \)

\( \Delta \text{t} \)

\( \text{thresh} \)

\( \text{local} \)

\( \text{contour} \)

\( \text{i.e., V1,4B detects component of pattern motion 1 to contour} \)
Why the average local direction won't work in general

average works
(in same direction tho speed wrong)

average doesn't work

actual pattern dir

average of local dirs

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

N.B. Single local motion same

[8 thick - pattern dirs
many thin - local dirs]
MT MODEL IN DETAIL

VT, layer 4B

[Diagram of neural activity patterns with directions and speeds]

For given speed:

neural activity

For given direction:

slow

fast

Need to test all combinations

Fast

Mod

Slow

One MT unit looks for local direction "family" across space.

E.g., object moving to left.

Family of local directions consistent w/ one pattern dir (thick arrow)

- 4D input space

Speed collapse across space

Dir

Single slim consistent w/ several families

2nd local dir disambiguates
MSTD

MT responds to rotation/dilation but not selectively.

1) MT RF that likes up.

2) Responds to clockwise because local up.

MSTD distinguishes different flow fields.

Spirals

MSTD RF

Stim

Clockwise

Better response than:

Counter clockwise

Dilation, contraction

Shear
How a fixed template actually works

MSTD RF

off-center CW stim

3 match
1 mismatch

yes

↑ thick - local stim direction

↑ thin - local RF sensitivity

off-center CCW stim

3 mismatch
1 match

no
Determining Optical Flow

Horn & Schunck, 1981

"gradient model"

Brightness of image point \( E(x, y, t) \)

Assume brightness doesn't change w/ time: \( \frac{dE}{dt} = 0 \) [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 \( E(x, y, t) = E(x, y, t) + \delta x \frac{\partial E}{\partial x} + \delta y \frac{\partial E}{\partial y} + \delta t \frac{\partial E}{\partial t} + \) higher order

Taylor terms [take limit \( \delta t \to 0 \)]

\[
\frac{\partial E}{\partial t} = \frac{\partial E}{\partial x} \frac{dx}{dt} + \frac{\partial E}{\partial y} \frac{dy}{dt}
\]

Data: spatial gradient

Data: temporal gradient

Unknowns

Solve for \( \frac{dy}{dt} \):

\[
\frac{dy}{dt} = -\frac{\frac{\partial E}{\partial x}}{\frac{\partial E}{\partial y}} \frac{dx}{dt} - \frac{\frac{\partial E}{\partial y}}{\frac{\partial E}{\partial y}}
\]

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

\[
y = mx + b \Rightarrow \text{this is equation for a line}
\]

Velocity space (previously illustrated with vectors)

Possible velocities given data: \( \frac{\partial E}{\partial x}, \frac{\partial E}{\partial y}, \frac{\partial E}{\partial t} \)

Same as:

Extended to \( x, y \)
COMPUTATIONAL MOTION

Smoothness

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

minimize: \[ \text{error} + \text{non-smoothness} \]
\[ \text{shown} = 0 \]

\[
\text{crm} = \int \int \left[ \left( \frac{\partial E}{\partial x} \frac{dx}{dt} + \frac{\partial E}{\partial y} \frac{dy}{dt} + \frac{\partial E}{\partial t} \right)^2 \right] \, dx \, dy
\]

non-smoothness
\[
\int \int \left[ \left( \frac{(\partial u)^2}{\partial x} + \left( \frac{\partial v}{\partial y} \right)^2 \right) + \left( \frac{\partial u}{\partial x} \right)^2 + \left( \frac{\partial v}{\partial y} \right)^2 \right] \, dx \, dy
\]

\[
u = \frac{dx}{dt}, \quad v = \frac{dy}{dt}
\]

Examples

Iterative solution to minimization problem
stable in \(\sim 10-20\) steps

Problems
- people can't detect movement of smooth bright/less saturated
- smoothness means edges
Line Processes

\[ \text{minimize} \quad \text{error} + \text{non-smoothness} + \text{penalty for discontinuity} + \text{penalty for non-constant} \]
EFFECTS OF ATTENTION IN V4

- Standard model attention:
  - This is wrong.
  - Attention is everywhere!

- Macaque monkeys are pre-adapted for peripheral attention exists:
  1) Spend all day not looking directly at attended targets.
     (e.g., dominant male)
  2) Eat many very small food items.
     (grass root)

- Operational definition of attention:
  - R.F.
  - Fixation print.
  - Detect small change in one stimulus while ignoring changes in another.

- Much harder to train monkeys than undergraduates 😊

3) Some evidence that there is a "feed-forward pass" before top-down attentional filter kicks in

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

2) Interpretation is ambiguous:
   - Attention to spatial location?
   - Attention to features?

Conclusion

- Attention to location and features is present to some degree in every visual area (and, of course, in areas, too).

N.B. equivalency times for human neurons 2x longer than here.
Haenny & Maniell
Somatosensory-visual & visual-visual

vis test
\[ \text{\longrightarrow delay} \]
\[ \text{\longrightarrow vis cue} \]
\[ \text{\longrightarrow som cue} \]
\[ \text{\longrightarrow V4 resp.} \]

\[ \text{\textbf{test responses}} \]

\[ \text{\textbf{test stim}} \]

\[ \begin{array}{c|c|c|c}
\hline
\text{cue} & 
\hline
\text{\textbf{\checkmark}} & 
\text{\textbf{\checkmark}} & 
\text{\textbf{\checkmark}} & 
\text{\textbf{\checkmark}} \\
\hline
\end{array} \]

"cue-tuned"

\[ \text{\textbf{test stim}} \]

\[ \begin{array}{c|c|c|c|c|c|c|c|c|c|c|c|c}
\hline
\text{cue} & 
\hline
\text{\checkmark} & 
\text{\checkmark} & 
\text{\checkmark} \\
\hline
\end{array} \]

orientation-tuned

\[ \text{\textbf{test stim}} \]

\[ \begin{array}{c|c|c|c|c|c|c|c|c|c|c}
\hline
\text{cue} & 
\hline
\text{\checkmark} \\
\hline
\end{array} \]

"match cell"

\[ \text{\textbf{test stim}} \]

\[ \begin{array}{c|c|c|c|c|c|c}
\hline
\text{cue} & 
\hline
\text{\checkmark} \\
\hline
\end{array} \]

both "cue"-tuned and orientation-tuned!
**Receptors**

- **Unencapsulated**
  - heat
  - cold
  - pain "x", C, sustained, small/myelinated
  - slow stroking

**Touch**

- **Superficial**
  - Merkel disks — sustained
  - Meissner's corpuscles — transient

- **Deep**
  - Ruffini endings — sustained
  - Pacinian corpuscles — transient
  - hair follicle receptors — transient
  - muscle receptors

"Stretch" → Length


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

**Intro**

- visual auditory somato olfactory

  - receptor
  - hair cell
  - ganglia
  - spinal ganglia
  - ganglion cells
Co-contraction of α, β motoneurms to detect deviation

- EMG
- Length tells state of body!

β-motoneurms
α-motoneurms

I_a
II
I_b

Merkel
Meissner
Pacinian
Ruffini
Hair follicle

Joint capsule

Expected load → Start
Unexpected load

γ-mot

No change in length of detector

Some contraction as "expected"
Stretch (N.B. caused by muscle spindle muscles!)
Dorsal column

Ventralbasal nucleus
VPL - hand, foot
VPM - face

medial lemniscus

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

Foot

Face

Fingers

1 2 3 4 5
2D maps
somatotopic
versus visual
Plasticity Exists
-
- denervate radial
- transplant patch
- syndactyly
- just stim — cf. and Allard
- Silver Spring monkeys — face
  invades
  hand
  1+ cm

\[ \rightarrow \text{weights} \]

\[ 3b \]

VB

Cuneate

[ - turn up terminals on \text{R} ]
[ - moves \text{RF to L} ]
[ - no plasticity corticocortical!! ]
[ - no plasticity VB \rightarrow 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 (6\textsuperscript{th} order vs. 4\textsuperscript{th} order)

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

2D receptor sheet
(visual, somatosens.)

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

1D receptor sheet
(auditory)
Impedance matching for air → fluid

Bone

From oval/stapes

Bone

Bone

To round window

Bone

Bone

Electrode

Motor

Signal

Amplifier

Otoscope

Myelinated

Unmyelinated

Main-freq
Receptors - Auditory

Hair cells
- Tectorial membrane
- Muller (support cells)
- Basilar membrane

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 response to sound
Owl Auditory System

[Diagram of neural pathways involving various brain areas such as SC, ICx, ICe, ICo, VLVA, VLVP, NM, NA, and nuclei magnocellularis and angularis. Notes include:

- "Time" field with note: Calyceal endings [NMLN]
- "Amplitude" field with note: Regular endings [stellate]

* breaks rule of Sereno]
Owl Physiology

- Introduction to sound
  - Frequency
  - Amplitude
  - Phase

NM — Phase-locked

Sound

Resp.

Spikes

NA — Amplitude Coding

Sound

Spikes (hi amp)

Spikes (lo amp)
Owl Physiology

NL (≈ MSO)
- first binaural
- ITD's

↑ RESP

left leads ↔ right leads

what a different characteristic delay looks like at same freq

VLVP (≈ NLLi)
How diff. char. delays start in NL

extra nodes of Ranvier for more delays through thickness of myelin

- explains how this generates diff. char. delays

2 spikes from this NM, 1 from other NM
Owl Physiology (3½)
Patterns in ICc lat w/ white noise & ITD's

different

- white noise at
  one ITD

- white noise at
  a different ITD

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

- this is view of one ITD
  across space

versus previous:

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

ICx

- space map
- amplitude vs. true delay

one characteristic amplitude diff

b1
b2
b3

one characteristic delay

b1
b2
b3

voilà!

a space map

elevation

gazimuth

activation
in response
to white noise
coming from a
small speaker

point-to-point projection

eye and head movement control

superior colliculus
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 drifts between sources

higher

one coherent source

another suppressed coherent source (diff spectrum)
Bats (1) the signal

- This is approximately what the auditory nerve sees
- Bat emits scream while "sniffing its ears" then releases muscle on stapes to hear echo

CF. Speech

Peter Frampton
Jim Soui
Bats (3)

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

DSCF area
- frequency & amplitude axes at $CF_2$ frequency

Diagram with axes and numbers indicating frequency and amplitude values.
Bats (4) CF/CF

Doppler shift components
- bat movement
- flutter

First just consider bat-target relations

\[ CF_{emitted} - CF_{echo} \sim \text{relative velocity} \quad \text{(not distance)} \]

\[ y = x + 2 \]
\[ y = x \]
\[ y = x - 2 \]

CF/CF area

\[ CF_1/CF_2 \]
\[ CF_1/CF_3 \]

A: \[ 90 - (29 \times 2) = 3 \text{ kHz} \]
B: \[ 91 - (27 \times 3) = 3 \text{ kHz} \]

\[ CF_1 \text{ kHz} \]

\[ CF_2 \text{ kHz} \]

\[ CF_3 \text{ kHz} \]

\[ 90 \]
\[ 29 \]
\[ 91 \]
\[ 8.0 \text{ m/sec} \]
\[ 4.0 \text{ m/sec} \]
\[ 0.0 \text{ m/sec} \]
\[ 92 \]
\[ 39 \]
\[ 31 \]

- units are relatively level-insensitive
- respond only to pairs of CF's (AND gates)
- pairs of CF's correspond to particular target velocities \( (\text{shift} = CF_3 - 3 \times CF_1) \)
- cannot detect distance (delay is sensitive)
Bats (5) FM - FM

**FM info**
- Good because delays are represented (Contrast CF)
- By comparing different harmonics, 6 overlap reduced
- Monaural delay detectors (Contrast and)

**FM - FM area**
- FM₁ - FM₂
- FM₁ - FM₄
- FM₁ - FM₆
- FM₁ - FM₈

Best Delay: *m sec (not usec)*
Figure 8.9  A spectrogram of the words "bab, dad, gag" (British accent).

Figure 8.11  A spectrogram of "lash, face, vase" (British accent).
- trading relations
- and cxs
- complex facts

N. VIII
[anesthetized cat]
larger gap
[dishabituation]
Auditory Brainstem &Ctx in Mammals
Auditory Thalamus

medialgeniculate $\rightarrow$ posterior & lateral in

[Diagram of thalamic structures labeled with brainstem nuclei and connections]
Cortical Areas — Auditory

(Somatosensory)

(T3) (T2) (T1) paACC

AI has finest-grained 6 map
**Eye Movements Anatomy**

1) **Vestibulo ocular reflex**

- Vestibular nuclei → Cerebellum
- Oculomotor neurons

2) **Optokinetic Nystagmus**

- Preceding nuclei → Cerebellum
- Terminal nuclei → Vestibular nuclei → Oculomotor neurons

3) **Orienting Eye Movements**

- Superficial SC → Visual cortex → Deep superior SC → Frontal eye fields
- Oculomotor neurons

2a) **Pursuit**

- Following a small object

Additionally, a diagram illustrates the control over eye movements and the neural pathways involved, including the role of the cerebellum in compensating for head movements.
Orienting Eye Movements

Sup. collic.
[50 all types many layers]

R.F. movement field
saccade caused by stim here

- double-step saccade

1) record receptive field
2) record during eye move
3) stim

Hallett & Lighthorne (1976)
How saccades update stationary targets

World

fixation target saccade

Sup. Colliculus

fixation target saccade
Double-Step Saccade Remapping

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

resp \( t \rightarrow \) build-up
resp \( t \rightarrow \) saccade-related

this translation performed by eye pos feedback efference copy

quasi-visual intermediate, build-up
then deep imm. preceding saccade
Coordinate Systems for Multi-Sensory Fusion

- SC codes target position by position, not amount of firing
- Saccade path gen. code position by amount of firing < reward

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

VIP

Auditory / SC

LIP pre-saccadic update

Update can go into opposite hemifield
Motor System
Basic Plan

Cortex

Striatum

Red nucleus

Cerebellum

 Vestibular

Retic. Fun.

Spinal cord

Excludes SC

Visually directed orienting movements

Eyes

Head

Ears

Whiskers

Shoulders

Outside inputs
Motor System "Principles"

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

2) Pattern generators - autonomous lower level sensory motor transformers (spinal cord)

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

4) Higher level pattern generators may have independent access to motor neurons (cingulo-spiral, striato)

5) Many "lateral" connections to mediate conflicts between different info sources (Suprasp VOR during present)
Descending Paths

Reticulospinal
- Pontine & Medullary retic. Fm. — uncrossed
- Midbrain Retie Fm — crossed

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

Rubrospinal
- red nucleus in mid
  - crossed

Corticospinal (only mammals)
Pattern Generators

- Spinal cord generates rhythmic, coordinated muscle movement w/o cortex (requires sens. skin)

- 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

  **Cats**

  **Minks, raccoons**

  **Cortex (new pattern gen.)**

  **Pattern gen.**

  **Motonurons**
Cerebellum - Basic Layout

deficits
- decomposition of movement
- inaccuracy movements
- oscillations
  - intention tremor
- titubation
  - trunkal tremor!
- lack of VOR adjustment

"Fractured Somatotopy"

folium top view

⇒ in granule cell layer and P-cell layer!
⇒ ascending ⇒ parallel
CEREBELLUM & CONDITIONING

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

Behavior

UR, CR

US (casea)

US (tone)

- conditioned response (CR)
- conditioned stimulus (CS)
- US

- lesion: no CR but UR remains
- lesion: no eyelid CR to auditory
- lesion: CR goes to extinction

Small part of interpositus nucleus

Pontine nuclei

Lesion: CR generated

Lesion: no eyelid CR to auditory

Stim: can be used as CS

Stim: can be used as US (60-400 mA !!)

cf. Saussure, an arbitrary connection
"Up & Down State"

- spiny stellate CP neurons

- "anomalous" rectifier
- K⁺ current activated by hyperpolarization
- cf. fast Na⁺ spike current
- perhaps a mechanism for switching between motor programs

Basal Ganglia defects

easy for Parkinson's

prog #1

prog #2

hard for Parkinson's

prog #1

prog #2
Striatum (= Basal Ganglia)

also used confusingly

caudate

putamen

globus pallidus

midbrain

substantia nigra

etc.

Sensory Striatum

motor cortex

Sensory cortex

+ 

caudate/putamen

+ 

substantia nigra

- 

globus pallidus

- 

thalamus

+ 

spinal cord

Limbic Striatum

limbic neocortex/amygdala

+ 

nucleus accumbens

+ 

ventral pallidum

- 

pedunculopontine & reticular nuclei

spinal cord
New Striatum

motor cortex

sensory cortex

Pallidal loop

GPe (2)

GPi (2)

SN

Premotor FEF

face brain

Thalamus

Midbrain

GPe

STN
dorsal
Skeletal

STN ventral
expulse

GPe

SNr

GABA

"pars reticulata"

GPe (2)

SNc

Dopamine

"pars compacta"

Chol.

Redunctolopentine

SNr

"pars reticulata"

Superior colliculus

Dorsal col. nucl.

Spinal cord

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

GPi / SNr

in similar functional positions

Dorsal col. nucl.
Motor Cortex

- Versus SI: Single muscles
- Unlike SI, multiple reps of muscle groups (not cut & peel)
- M-I & M-II have own FEFs

- SMA
- DLPFC
- IC
- FEF
- PZ
- Premotor
- PCA
Georgopoulos (1) - bad

- M-I
- Population vector

Caminiti

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

Georgopoulos (2) - good

- Like mental rotation
- Think of as higher level programming leaking through into primary mot.
- notation:

1 2 3
MotorCtx (parietal) Stim

- muscles vs. movements
- EMG of 'everything' is hard
- Graziano
  \[
  \begin{align*}
  & \text{long stim trains} \\
  & \text{position in extra personal space} \\
  & \text{cf. frog spinal cord!}
  \end{align*}
\]

- Stepniewska/Kaas

- rodent vs. primate
  \(\rightarrow\) major diff thalamic afferents
  \[
  \begin{array}{c|c|c}
  \hline
  \text{rodent} & \text{primate} \\
  \text{somato-motor CTX} & \text{M-I} | \text{S-I} \\
  \text{VL} & \text{VB} & \text{VL} | \text{VB} \\
  \hline
  \end{array}
  \]

- Sulci (human: principal + arcuate)
  \[
  \begin{array}{c}
  \text{principal} \\
  \text{arcuate} \\
  \text{macaque} \\
  \text{human}
  \end{array}
  \]
BASIC LIMBIC CIRCUITS

Septum (6) Basal Forebrain

Hypothal amm (20)
LH subthal. nuc.

Hypophysis (pituitary)
Post. mammo/ventromed. varioespin

Hippocampus (14)

Amyg (10)

Perforant pathway
fornix (H)

Fornix (H) stria terminalis (A)

Epiphysis (Pineal)

Olf. spilial. (Vomeronasal organ)

Sensory

MFB

Midbrain

Septum (6)

Motor nuclei

CorticoPWN

PPN

SN

SNr

VST

SNc

Substantia nigra

Pons

Rexed

Cir. stratum
Hippocampus

Anatomy

- MT → MST → PFC
- V1 = V2 = V4 → PIT → AIT
- Entorhinal
- Subiculum
- CA1
- CA3
- Dentate gyrus
- Cingulate & orbitofrontal
- Somatosensory
- Auditory
- Vis
- Superior Temporal Sulcus
- Inferotemporal cortex (e.g., PIT, AIT)
- Rhinal fissure
- Parahippocampal

Physiology

- Place cells in CA fields
  - History of discovery of place cells
  - Modern study of place cells
    - Rodent arm maze < Distal cues to place
      - Works in dark, too
    - Cue-card enclosure
    - Spatial deficit (Morris water maze)
    - Cue card scales up to larger enclosure
      - Less firing in different shape
      - Barrier < clean opaque
      - No head direction
- Head direction cells in "Postsubiculum"
  - No place fields
  - Incredible inertial guidance
  - Distal cues to head direction
1) James Olds
   missed discovery
   theory-driven observation

2) Basic place cell

3) Water maze

4) Radial arm maze < local
4b) Move cue card in view \[\rightarrow\] reset!

5) Head dir cell

6) Grid cell
   - Med entorhinal dst
   - Size: deeper \[\rightarrow\] larger
   - Shape: \[\rightarrow\] spiral

7) Elaboration
   - 2 rooms
   - Barriers

8) Head fixed
   VR navigation: place cells \[\text{OFF!}\] \[\rightarrow\] reg. vestib/real movement

\[\text{eta phase process}\]

\[\text{Theta phase process}\]
Evidence

- no new memories
- retrograde memory loss for 1 year
- preserved ‘motor’ learning

Interpretations

1. explicit/cognitive memory/declarative
   
   input → iconic → STM → LTM

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

H.M. vs. place cells??

- ITM vs. "you are here now"
- effects of lesions disambiguate:
  - what H.M. looks like!
  - hippocampal proper
  - parahippocampal

H.M.

Entorhinal ctx
Parietal

PIT
LTP

V4

Hippocampus

Amyg.
Anatomy

- Inputs from 3° sensory areas
- Outputs to 3° sensory areas
- Limbic connections

Physiology

- Klüver-Bucy (Amyg)
- 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
- birdsong vs. calls, sexual selection, whales
- hominin vocal theory

- main distal sense
  - blinds
- "picture theory" (p3, 17)
- word recog. (Peter pic expl3)
- Lakoff/Fauconnier/Jackendoff

#2
- fictive scene comprehension
- film
- polysemy → "line" < tiny context (cf. alanine)
  - anaphora → "that fictive scene comprehension stuff"

Squirrel, monk, vocal:
- core vocal.
- area
- control gray
- nuc. ambigu.
- nuc. retromvng.
- larynx

DNA, protein self-assembly