SBE II
TA Exam II Review Session

Your TAs
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3 April 2015

Disclaimer

These notes are for review purposes only.

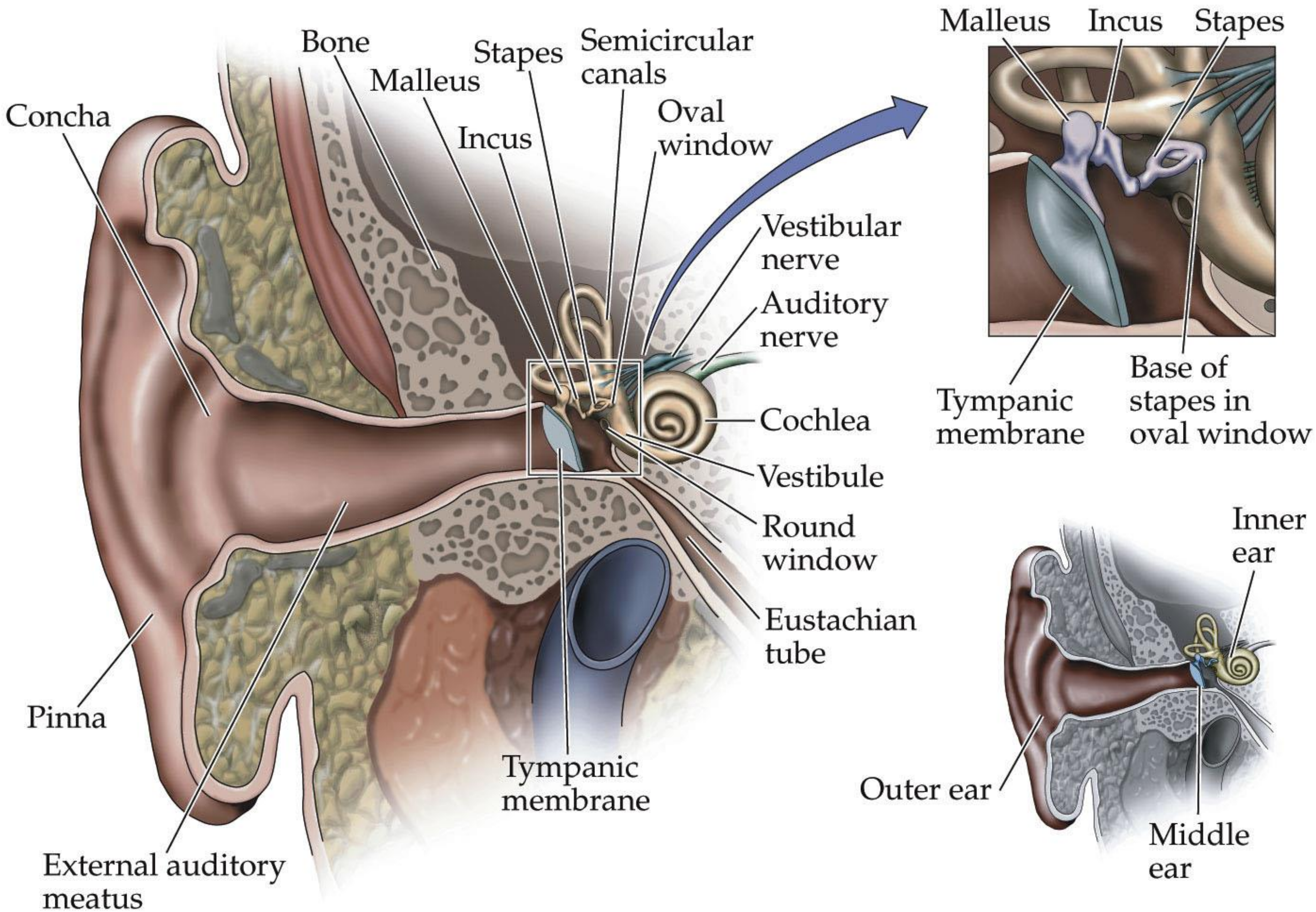
There is no guarantee that any of this material will be on the exam.

The exams will be developed from the lecture material.

Credit will not be given for any arguments derived from these notes.

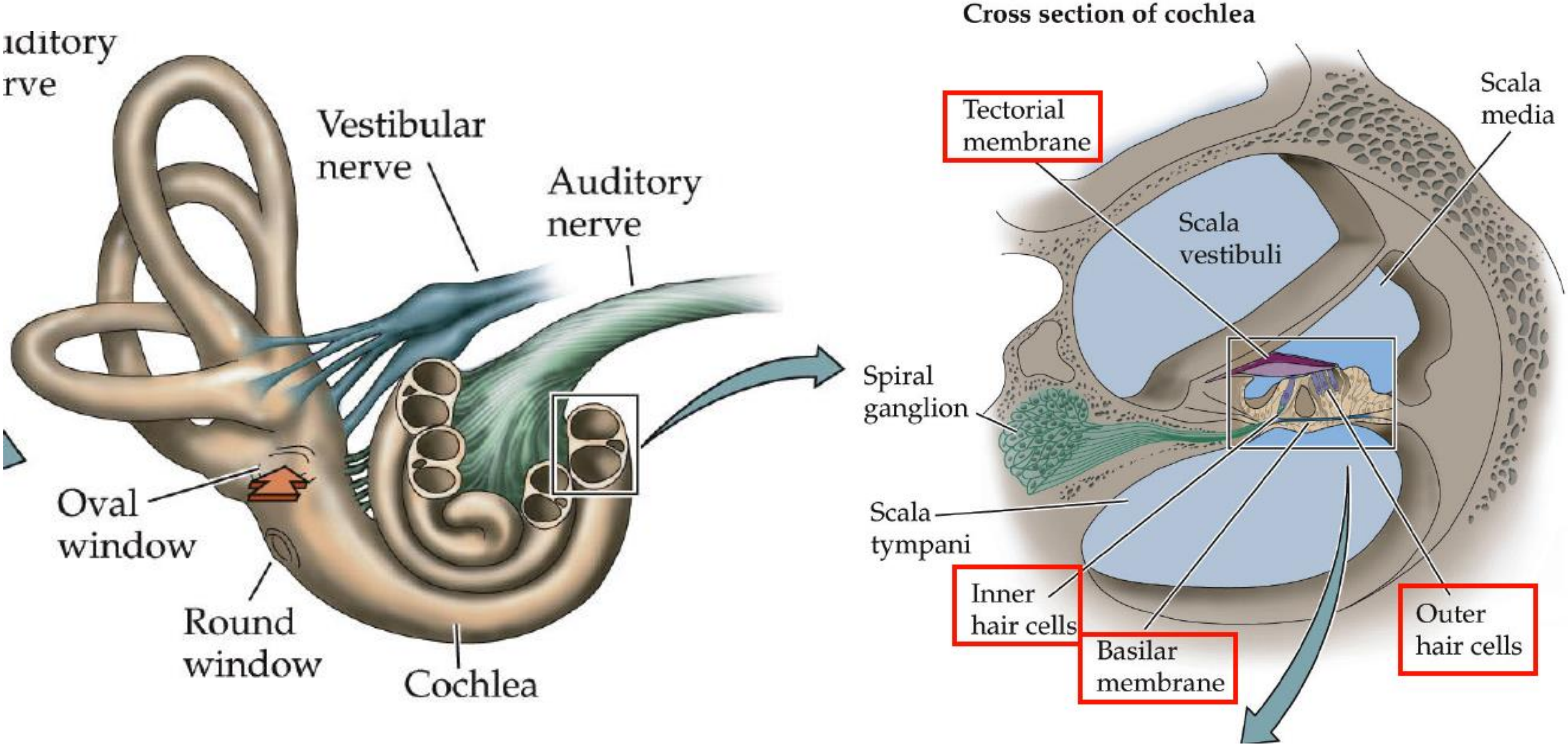
Dr. Wang’s Lectures

The Human Ear

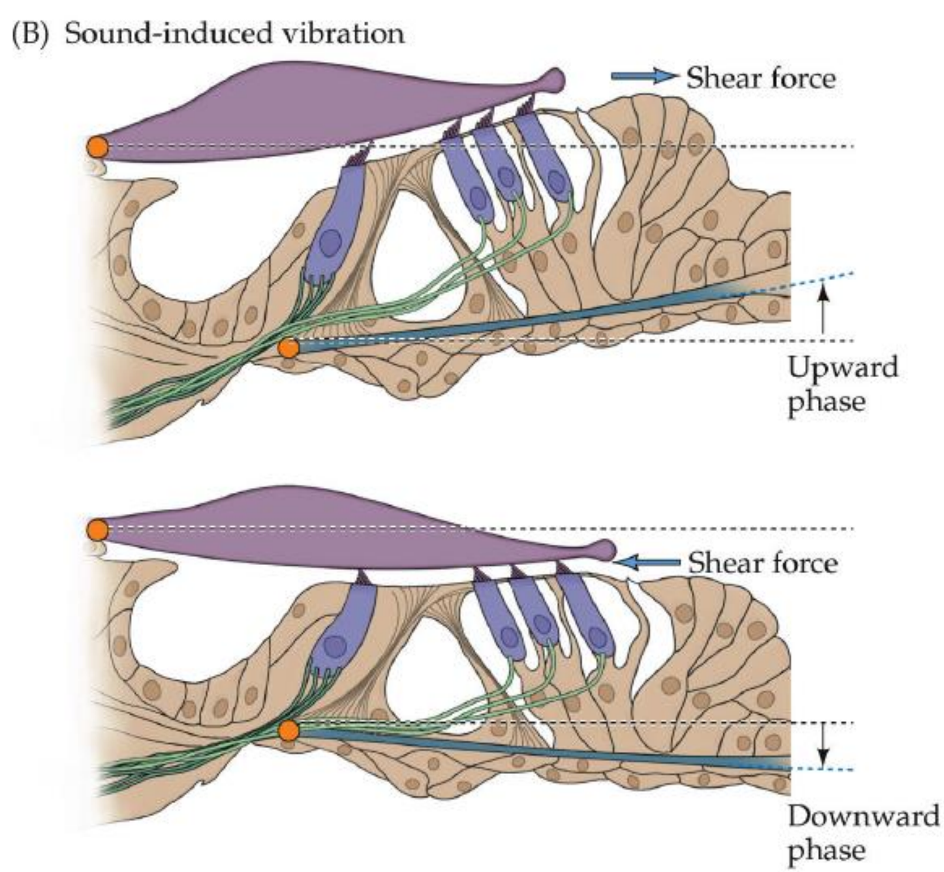


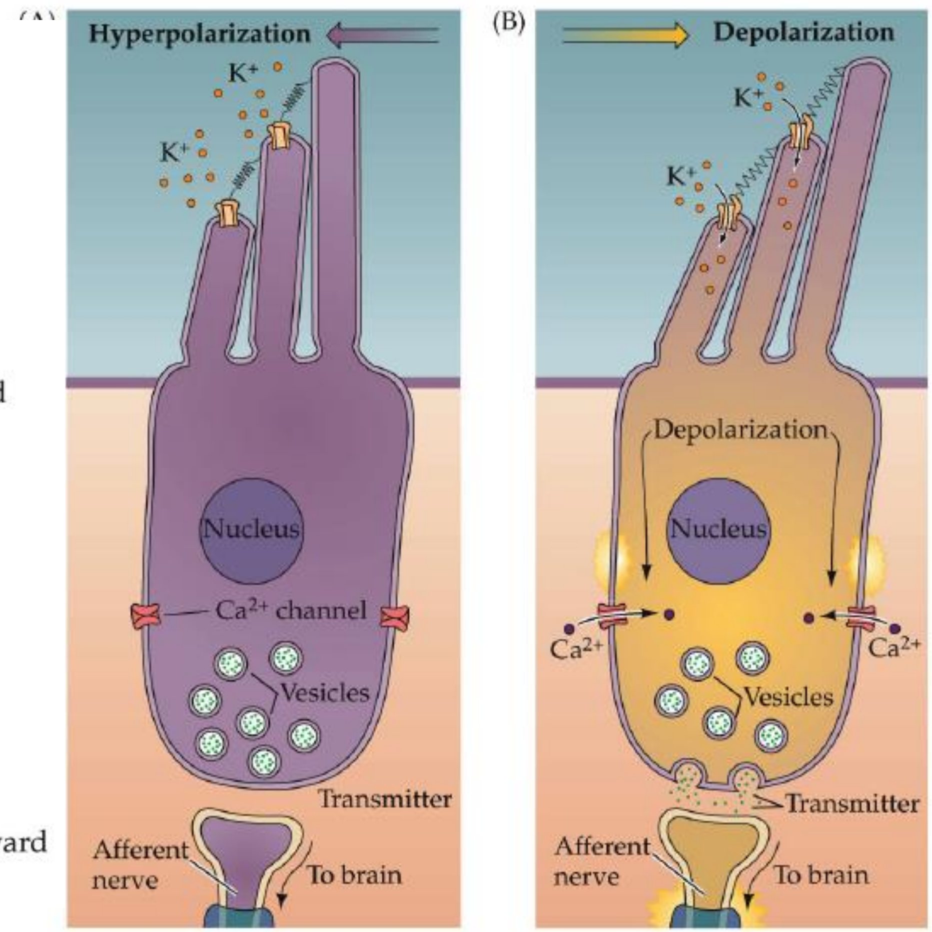
NEUROSCIENCE, Fourth Edition, Figure 13.3

The Cochlea

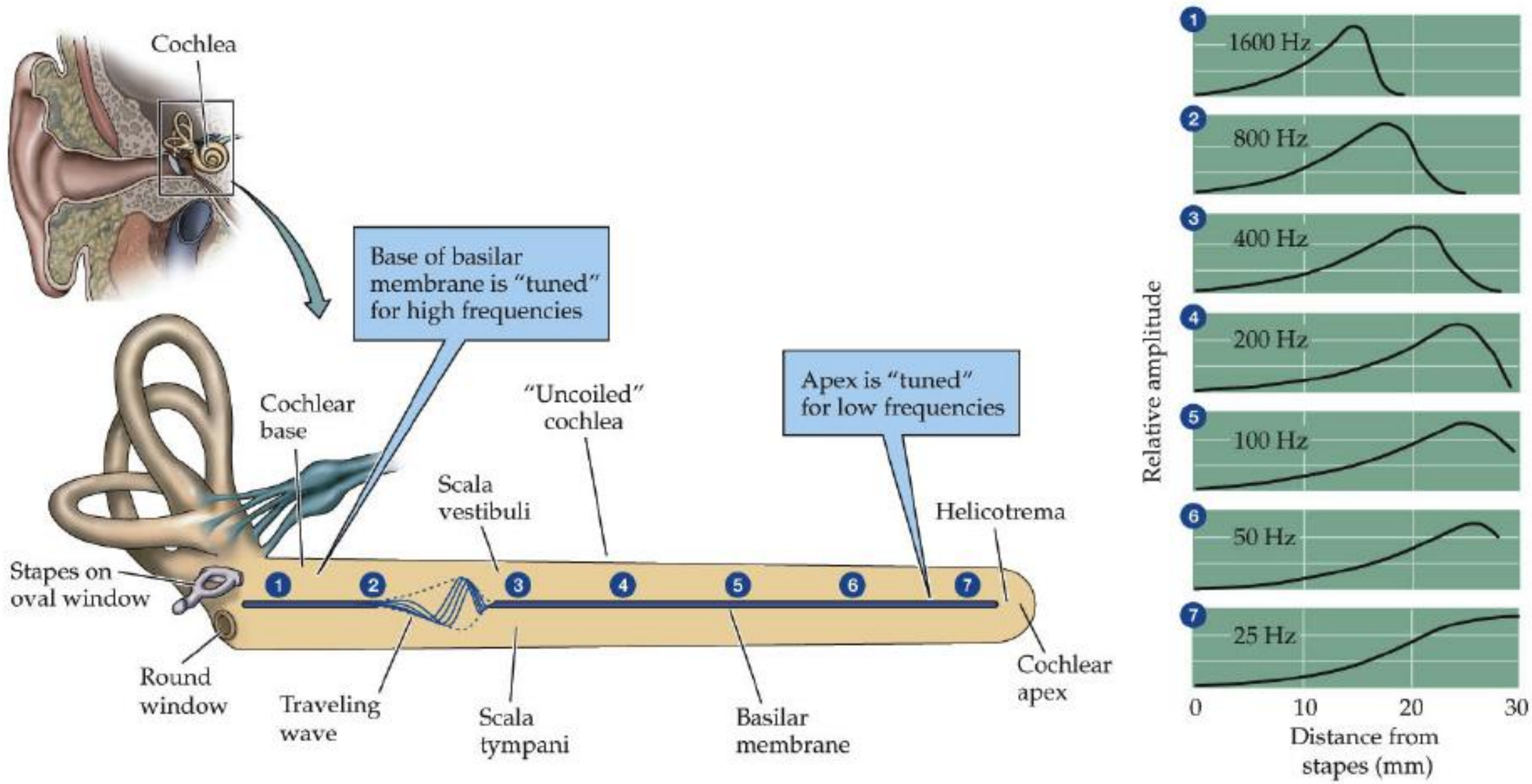

uditory
rve
Vestibular
nerve
Auditory
nerve
Oval
window
Round
window
Cochlea
Cross section of cochlea
Tectorial
membrane
Scala
media
Scala
vestibuli
Spiral
ganglion
Scala
tympani
Inner
hair cells
Basilar
membrane
Outer
hair cells

Mechanoelectrical Conduction in Hair Cells

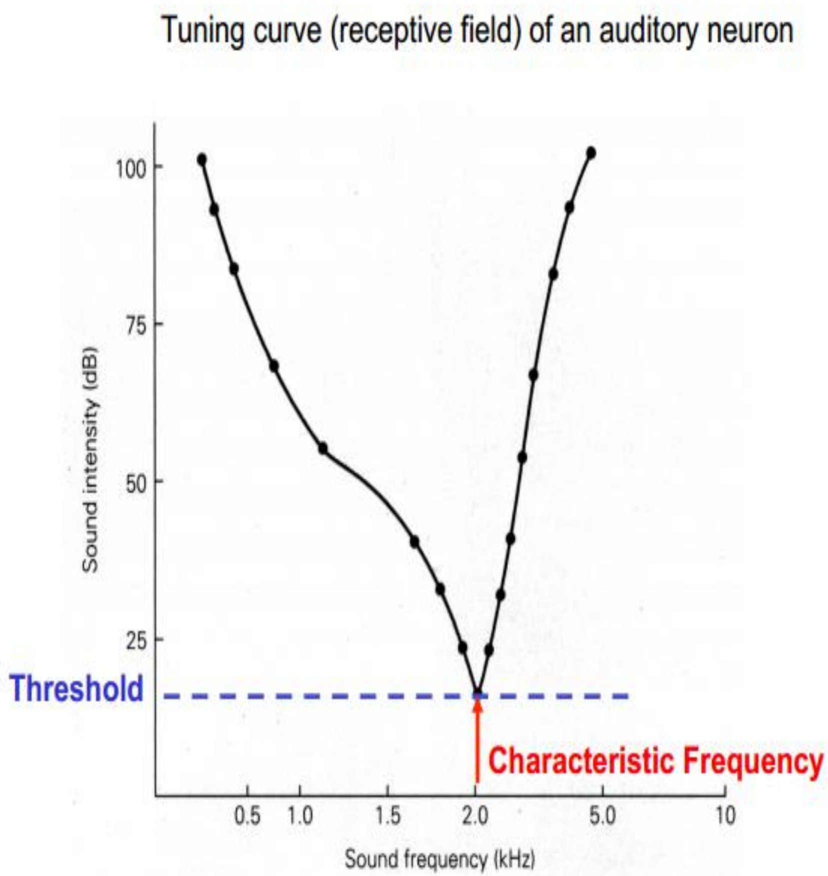




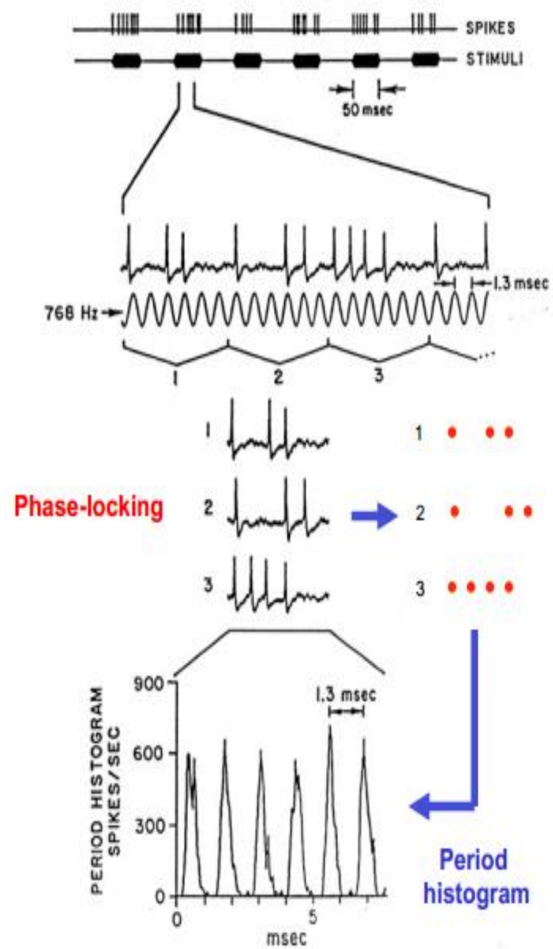
Sound-Tuning in the Cochlea



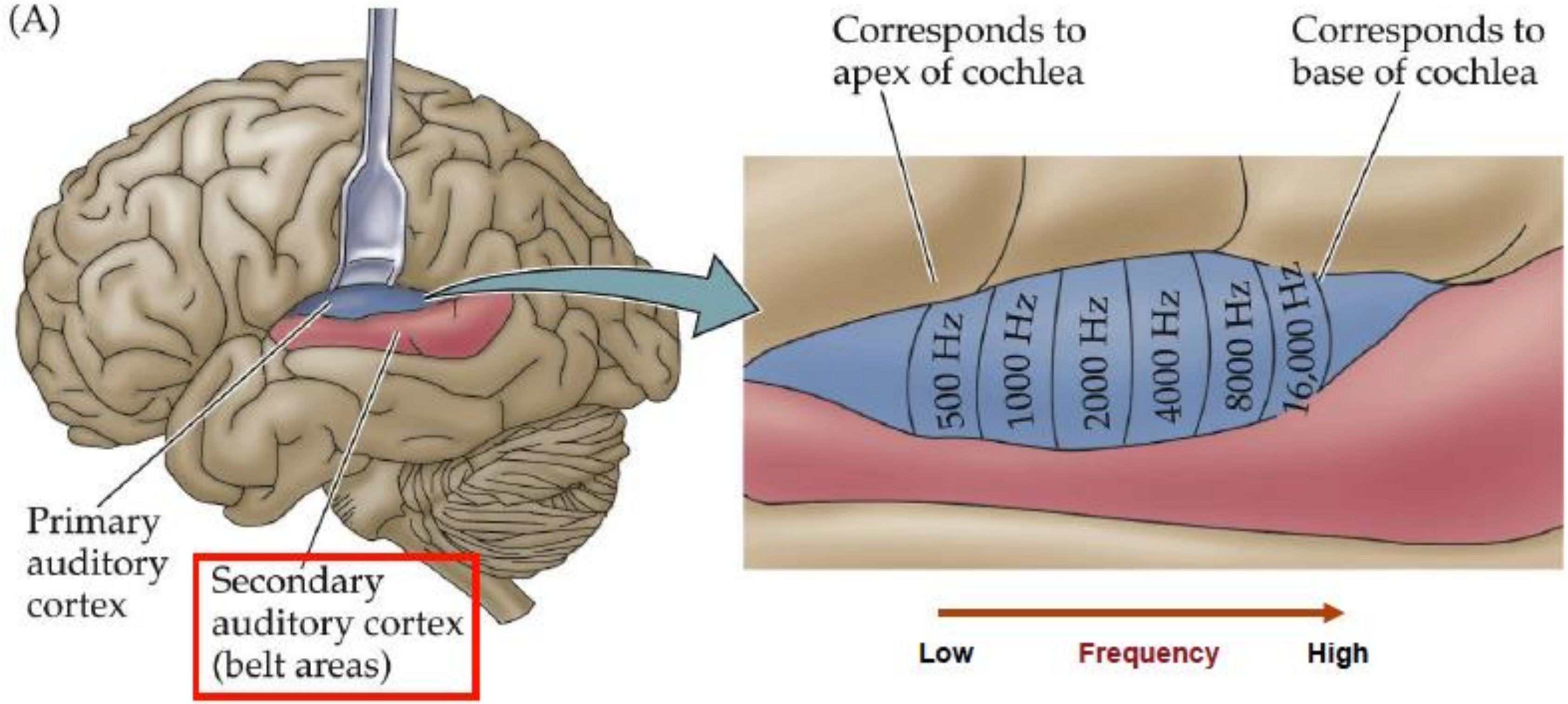
How does a tuning curve for an auditory neuron look like?



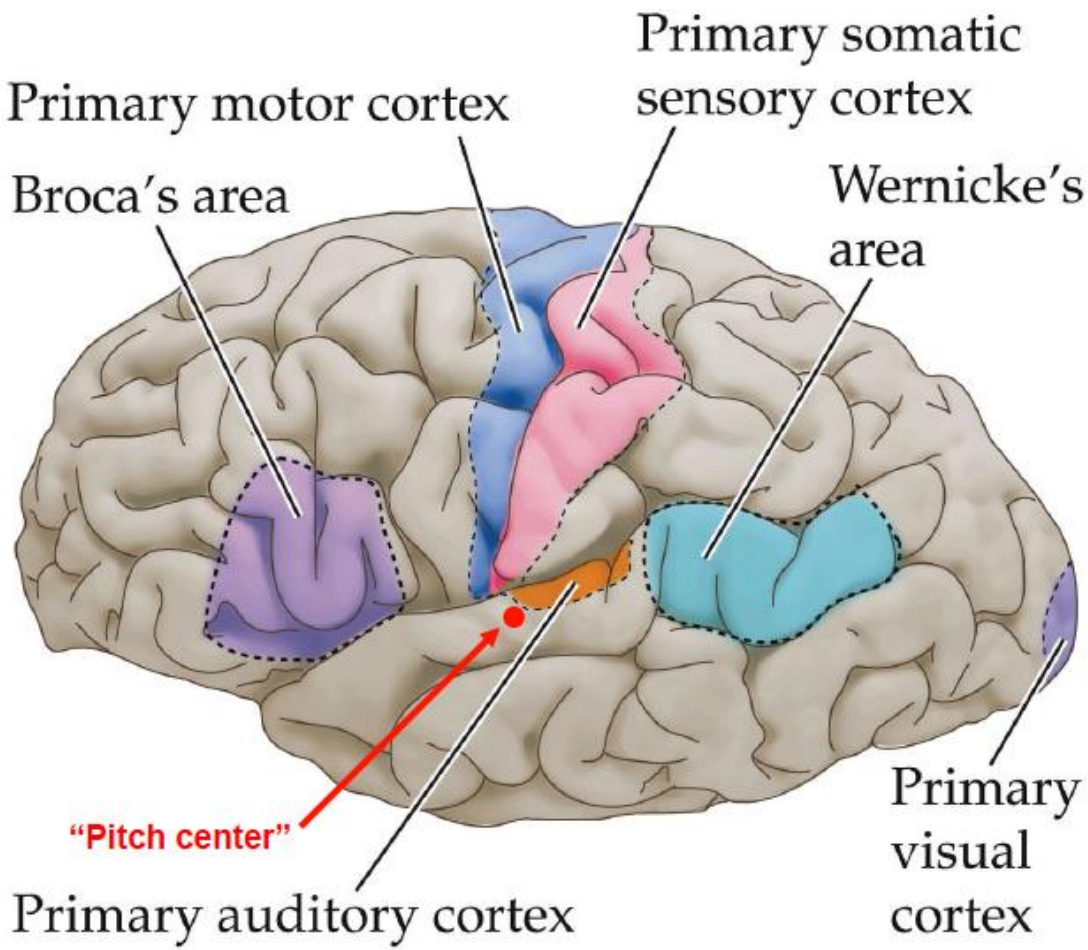
Period Histogram and Phase-Locking



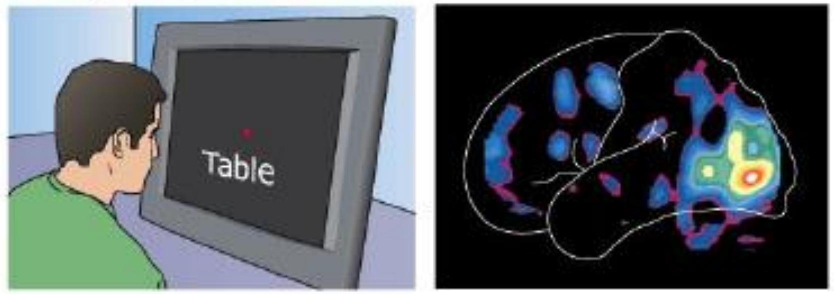
Tonotopic Organization in Auditory Cortex



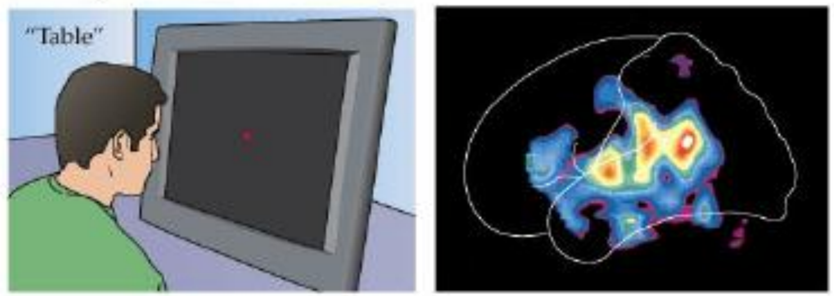
Cortical Representations of Senses



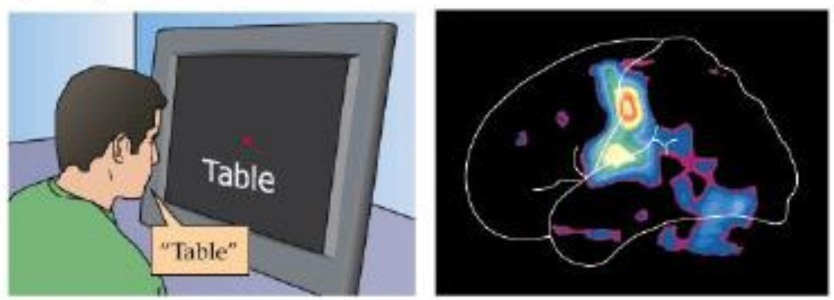
Passively viewing words



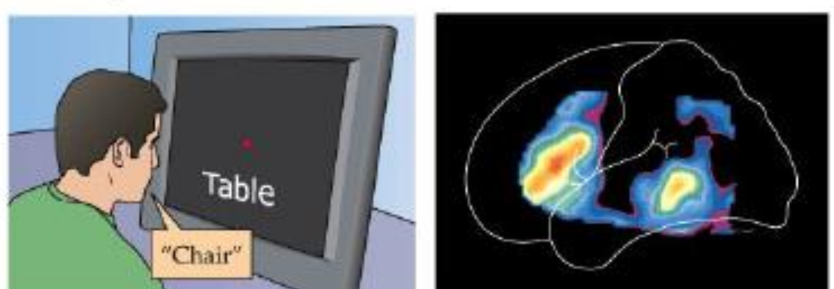
Listening to words



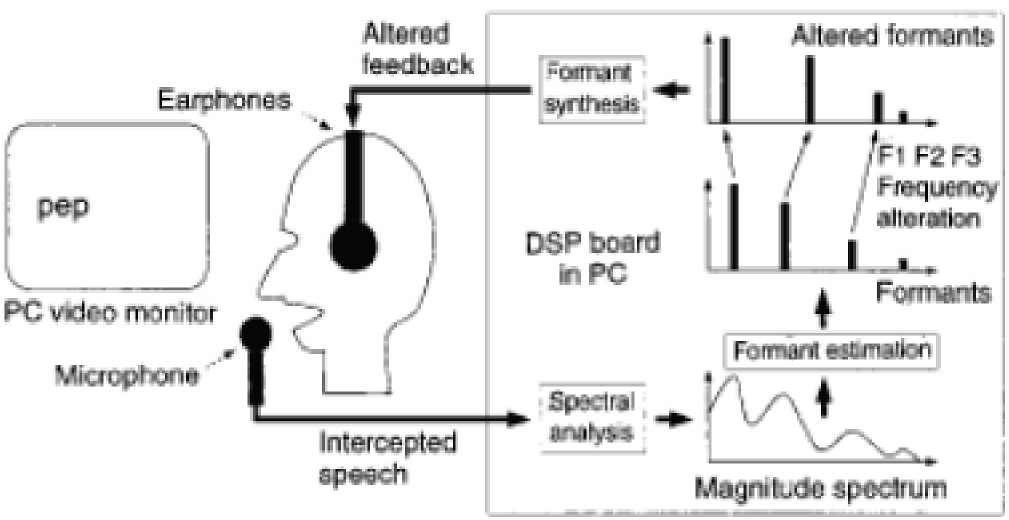
Speaking words

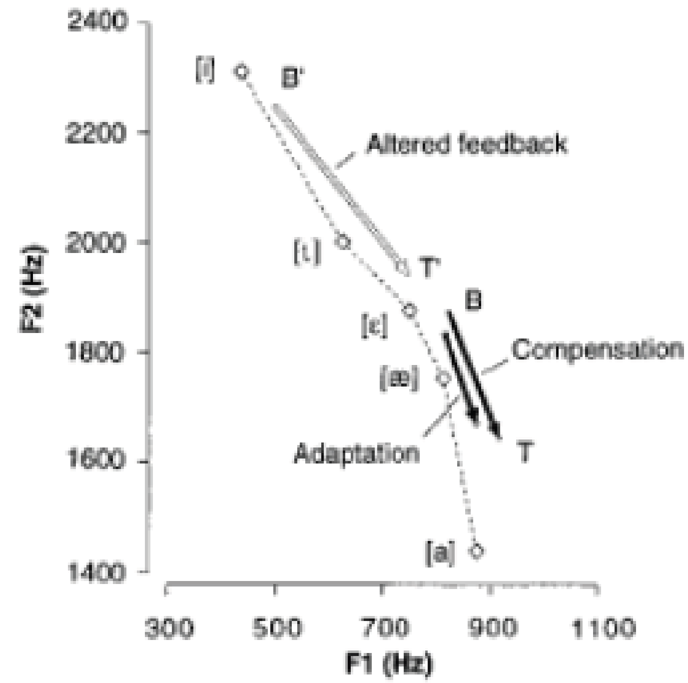


Generating word associations

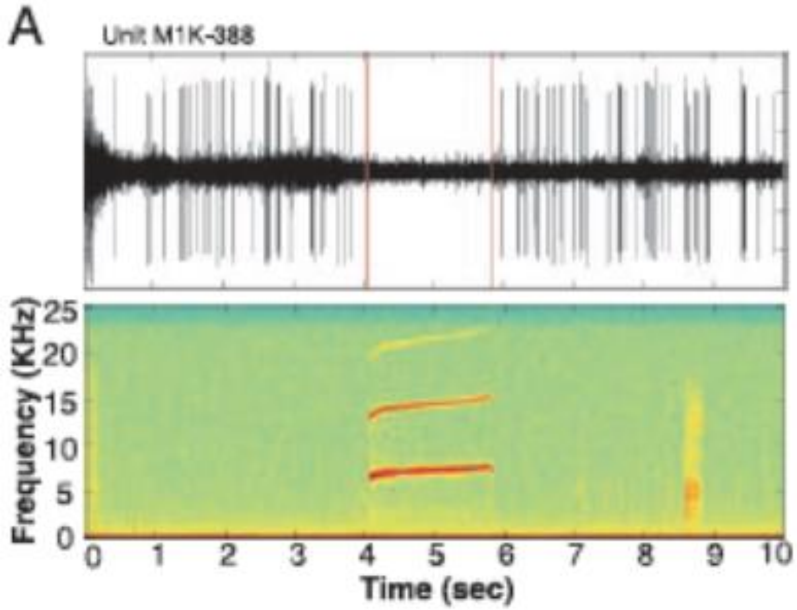


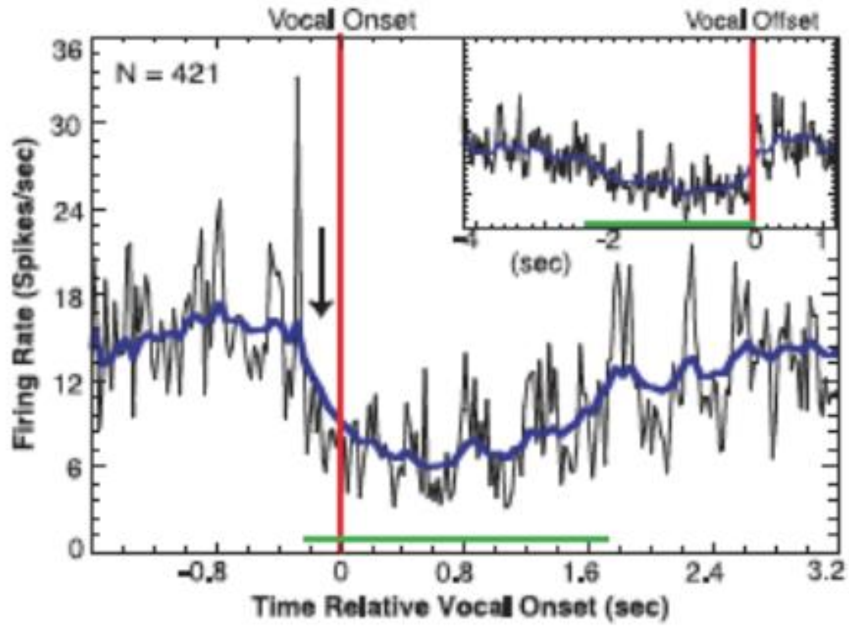
Feedback Alters Vocal Output



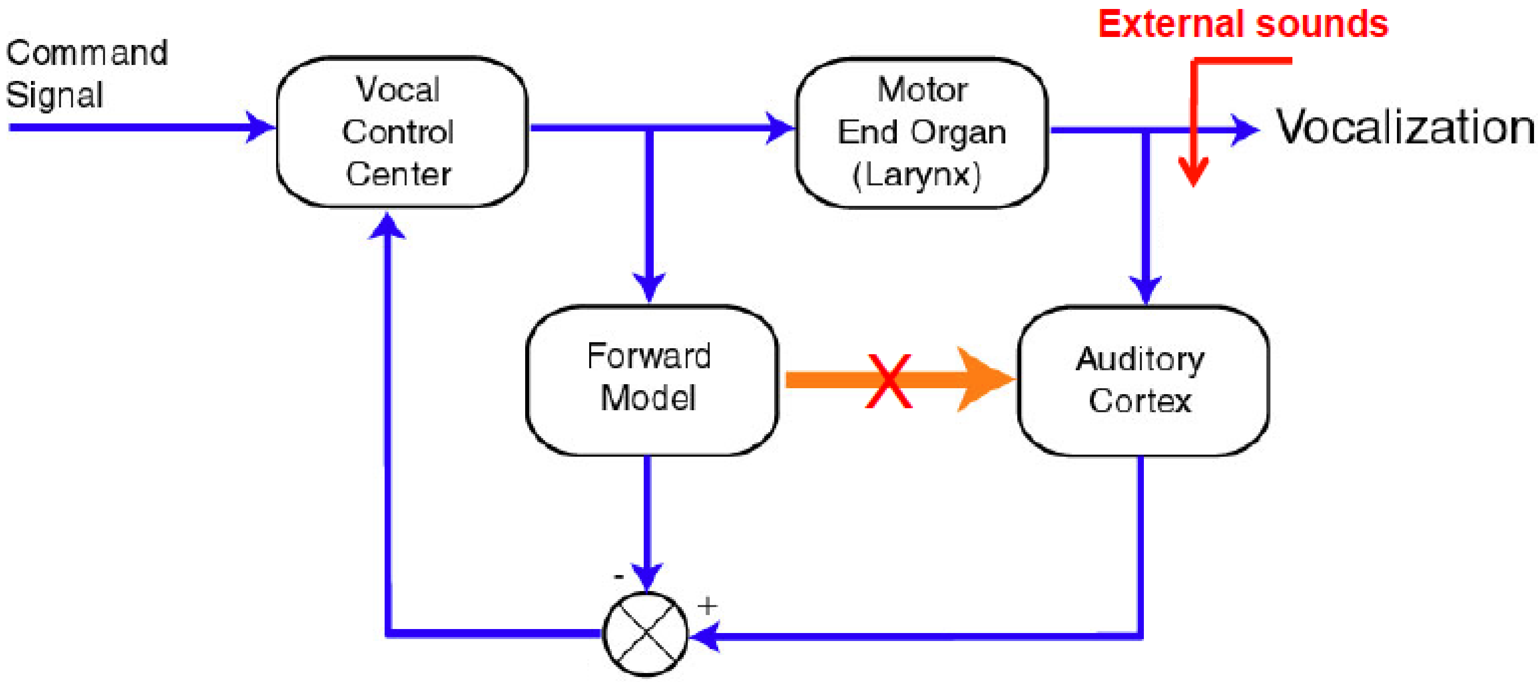


Auditory Cortex Activity Decreases During Vocalization

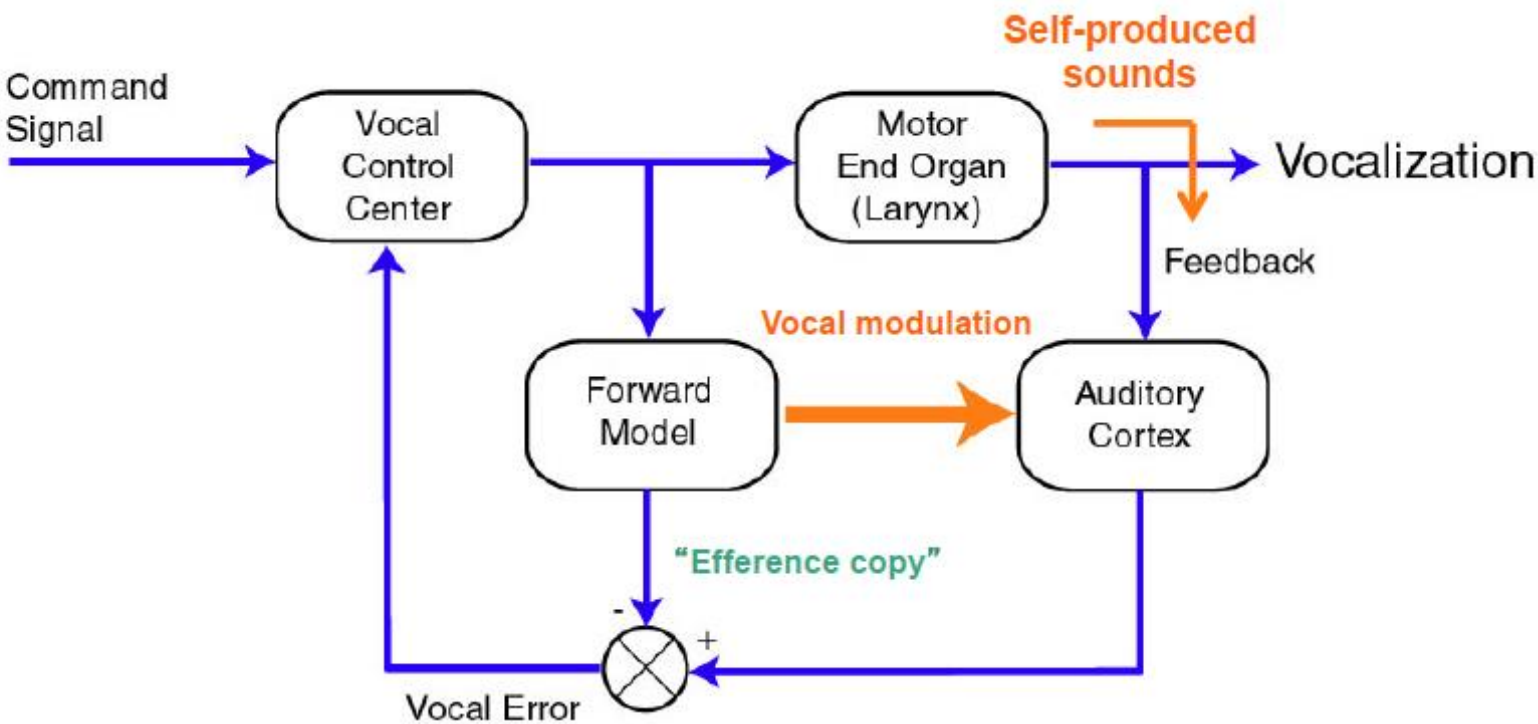




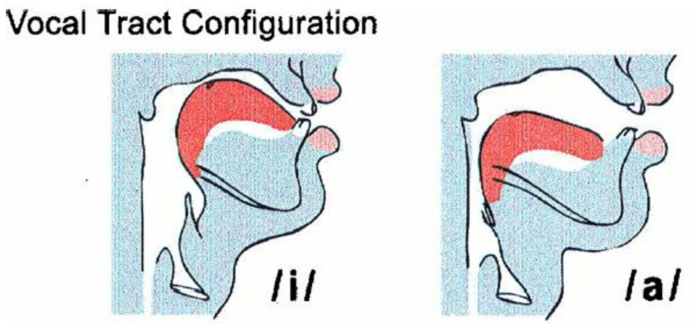
Auditory-vocal interactions in auditory cortex

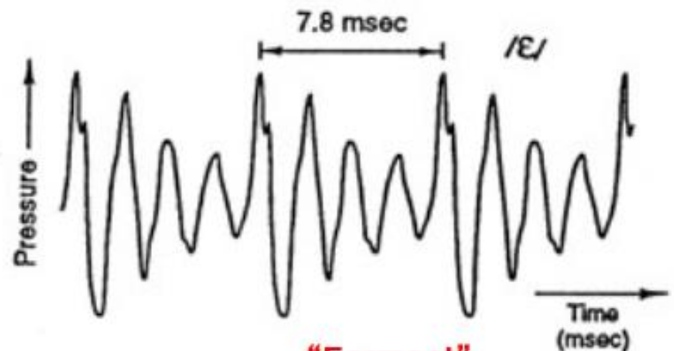


Auditory-vocal interactions in auditory cortex

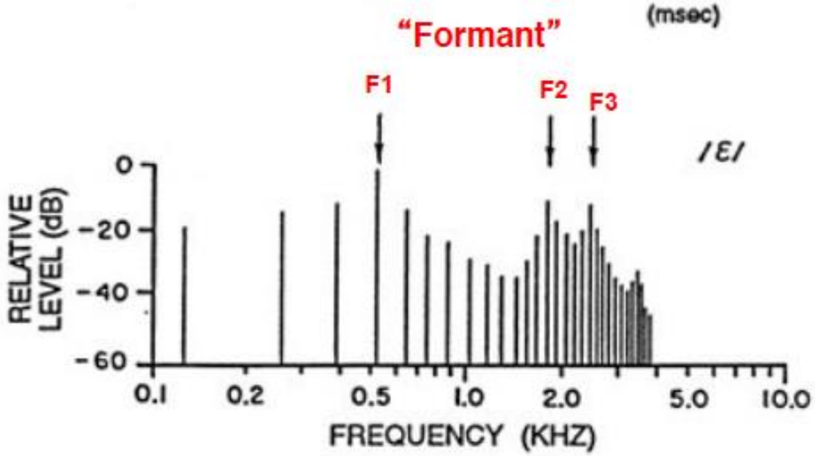


Spectral and Temporal Characteristics of Speech





Amplitude-time Waveform



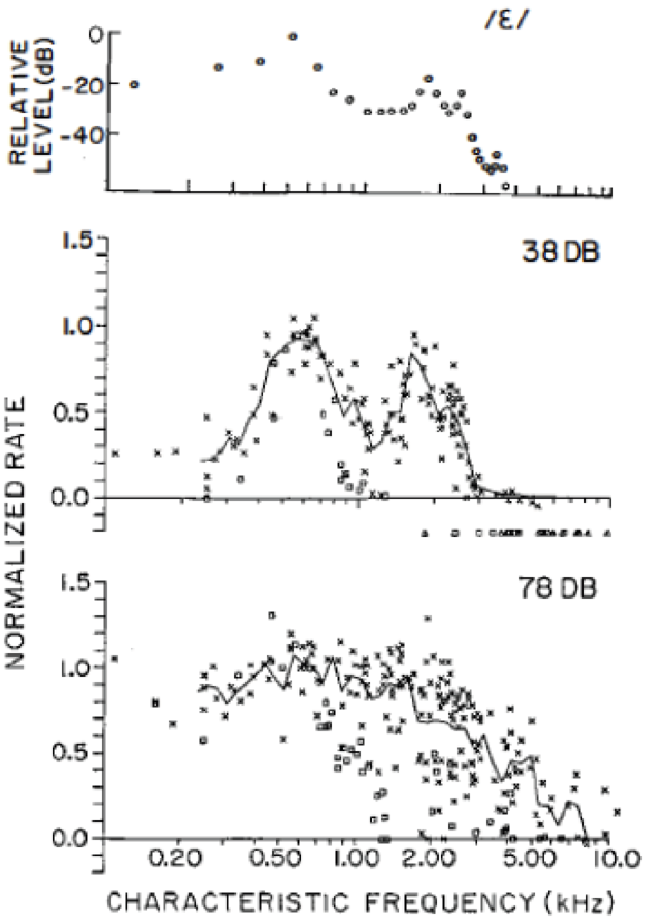
Spectrum

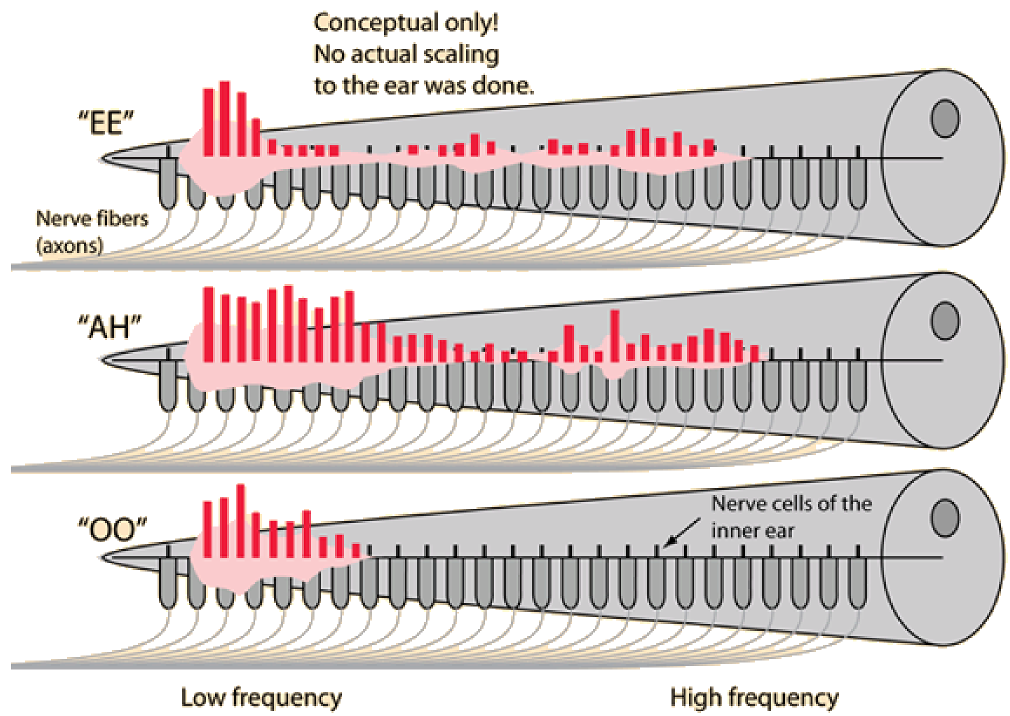
Average vowel formants[4]

Vowel (IPA)	Formant F_1 (Hz)	Formant F_2 (Hz)
i	240	2400
y	235	2100
e	390	2300
ø	370	1900
ɛ	610	1900
œ	585	1710
a	850	1610
ɶ	820	1530
ɑ	750	940
ɒ	700	760
ʌ	600	1170
ɔ	500	700
ɤ	460	1310
o	360	640
ɯ	300	1390
u	250	595

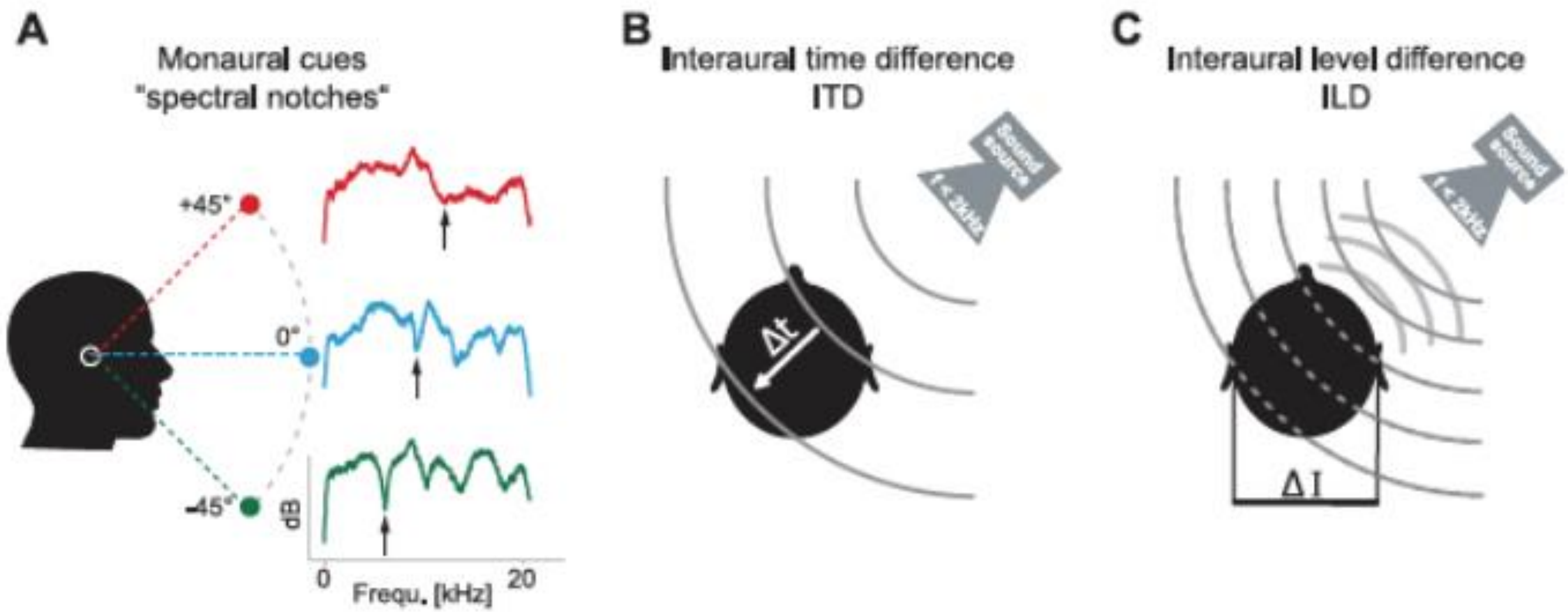
Auditory Nerve Representation of Speech Sounds

1) Firing rate
2) Temporal discharge pattern

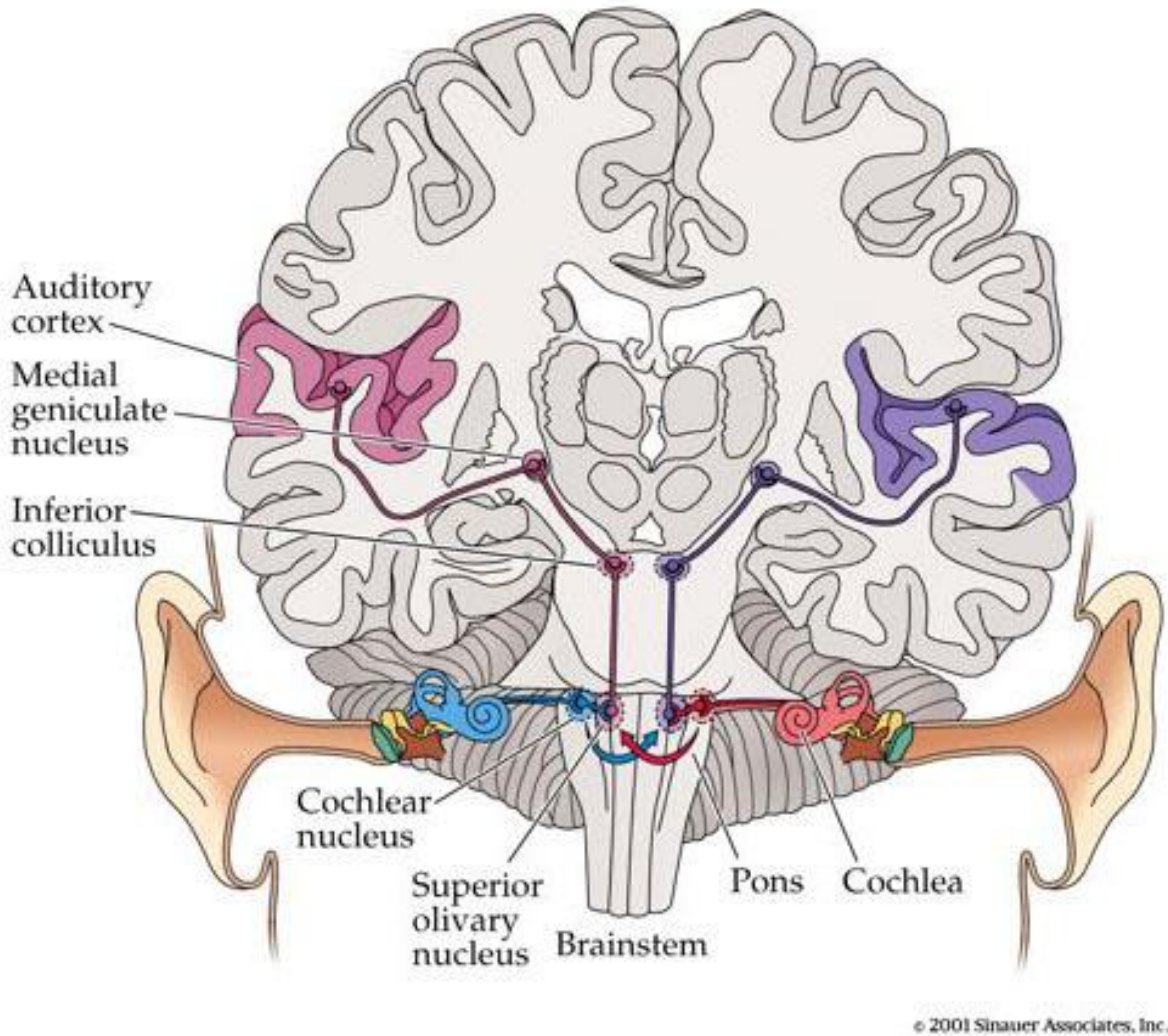




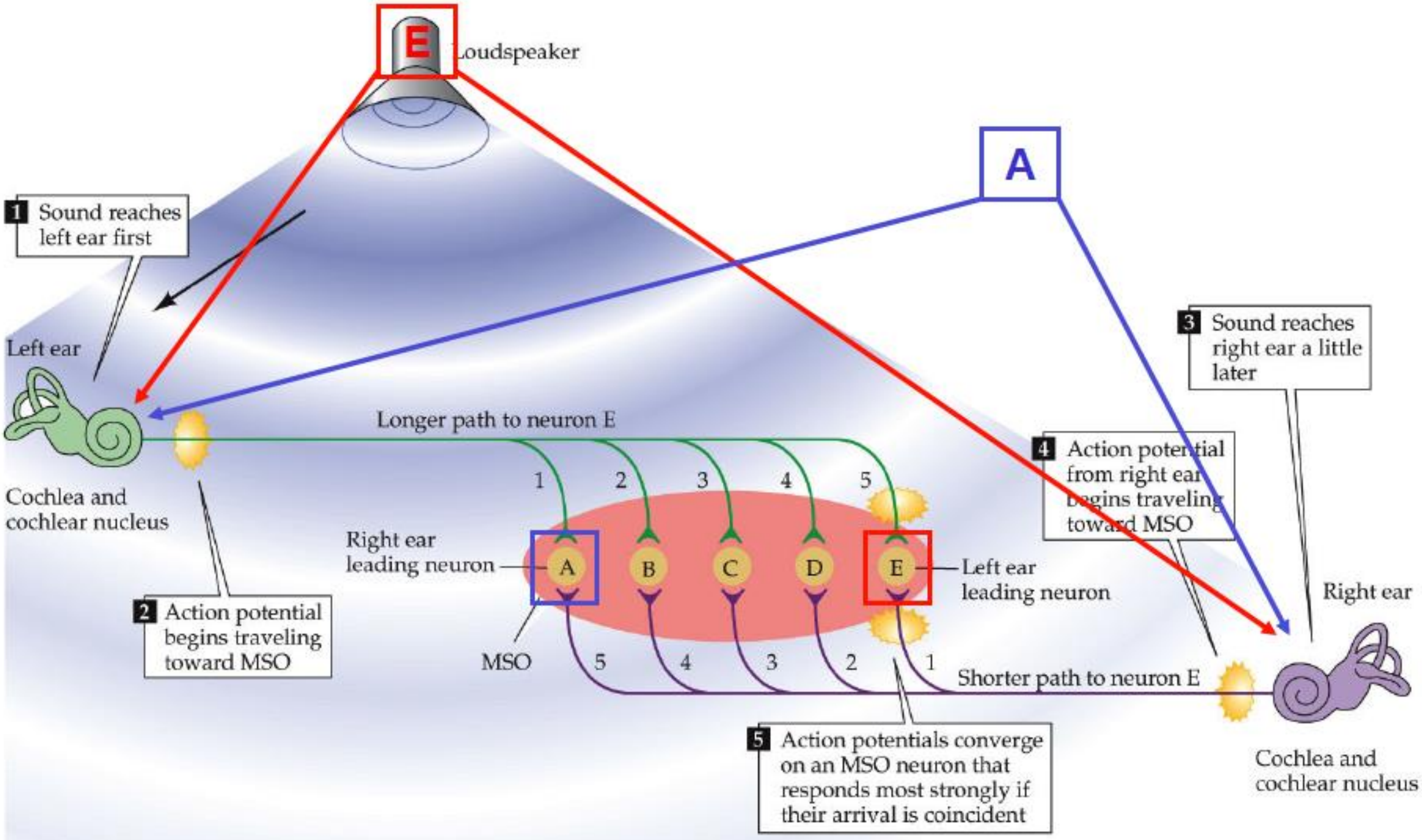
Sound Localization Cues



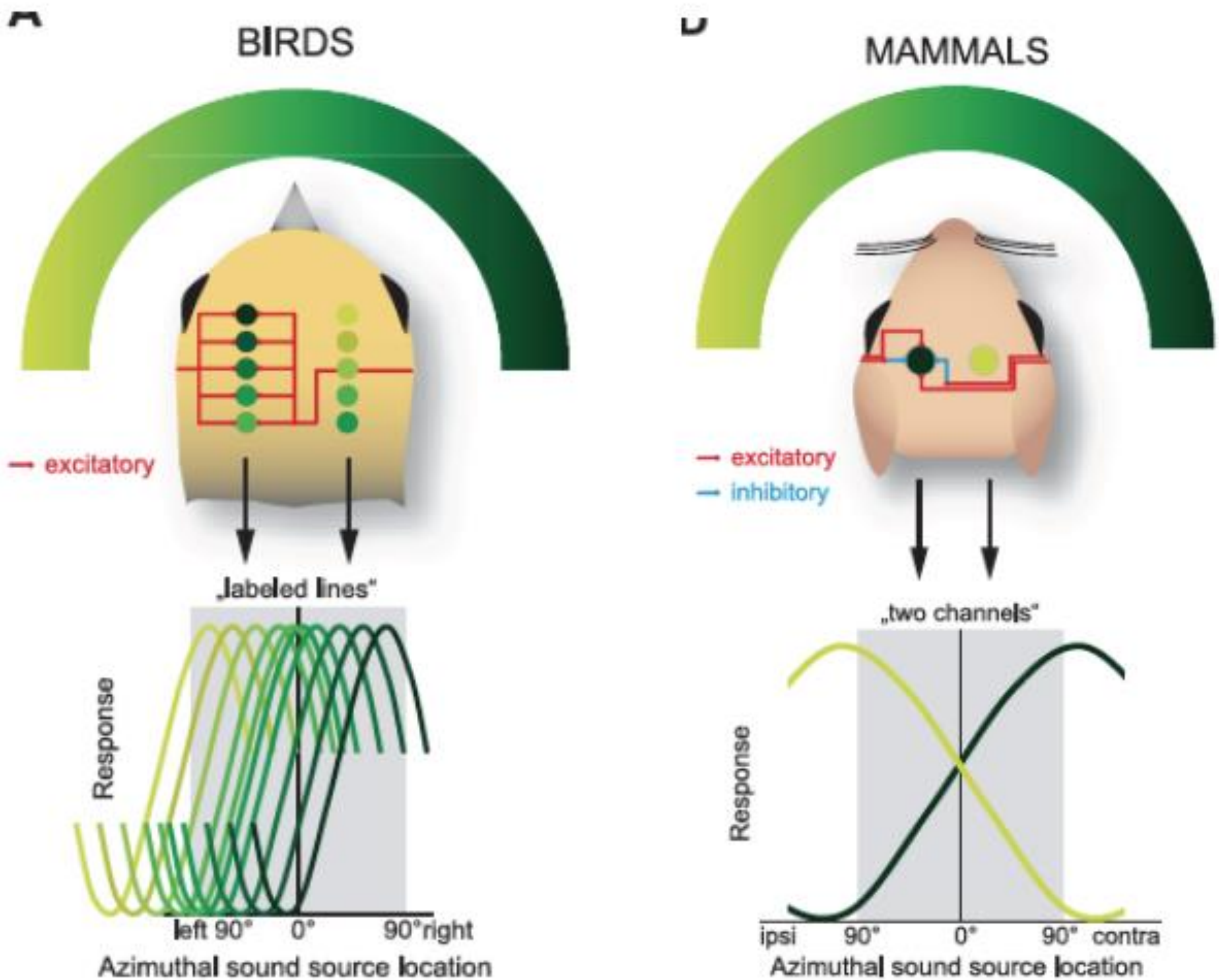
Auditory Pathway (CNS)



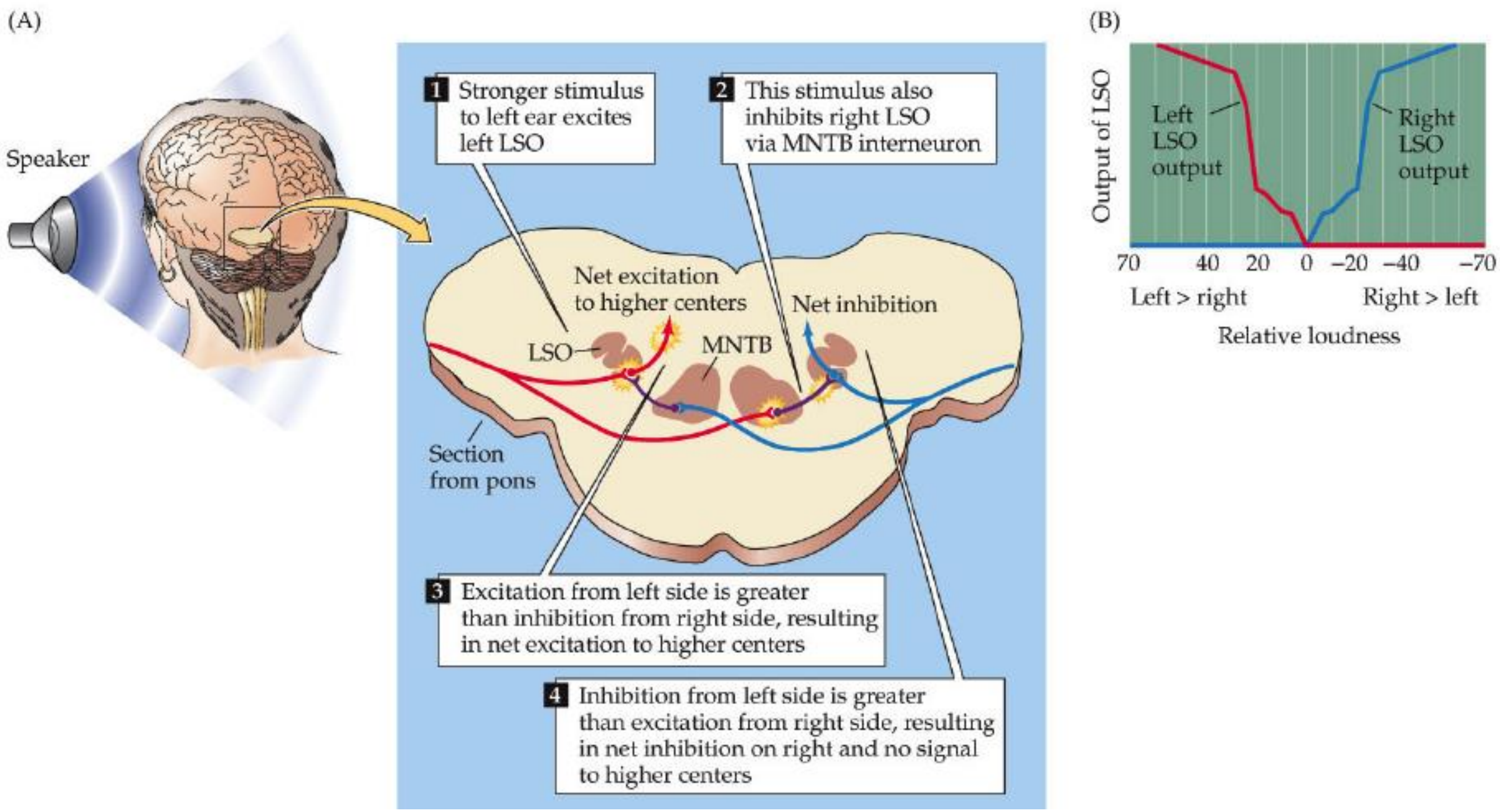
MSO Computes Sound Location with ITD



Two Mechanisms for ITD Computation



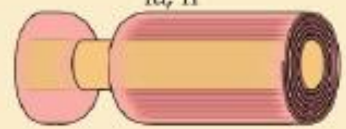
LSO Computes Sound Location with ILD

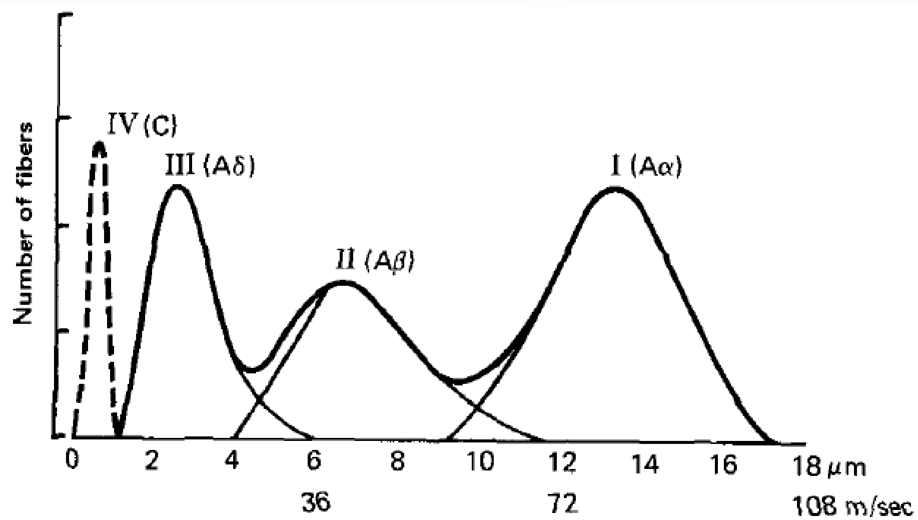


Somatosensory System Afferents

• Touch	Spatial form	(SA1)
	Texture	(SA1, PC)
	Movement	(RA)
	Flutter	(RA)
	Vibration	(PC)
• Pain	Pricking Pain (A –delta)	
	Burning Pain (C fiber)	
• Temperature	Cold	(A -delta)
	Warm	(C fiber)
• Itch	(C fiber)	

TABLE 9.1 Somatic Sensory Afferents that Link Receptors to the Central Nervous System

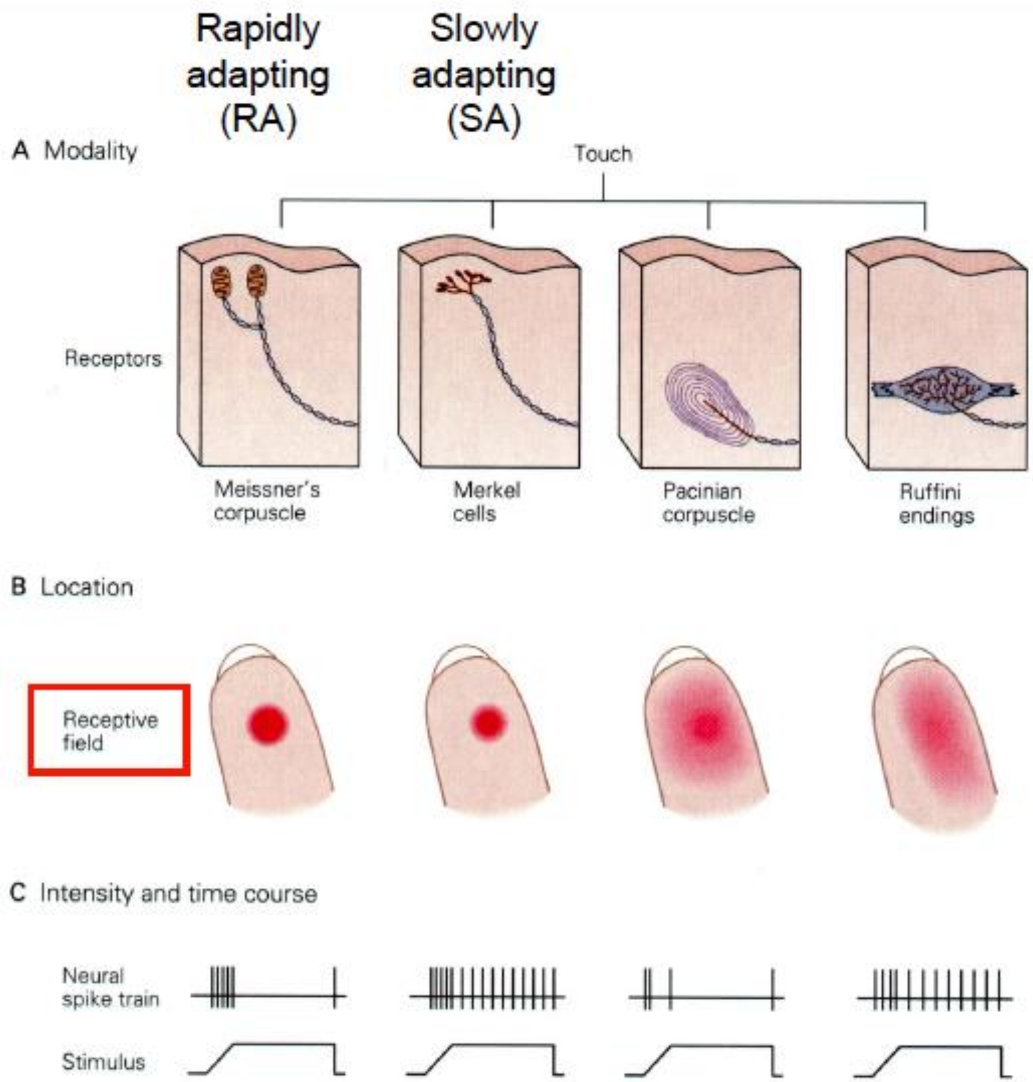
Sensory function	Receptor type	Afferent axon type[a]	Axon diameter	Conduction velocity
Proprioception	Muscle spindle	 Ia, II	13–20 μm	80–120 m/s
Touch	Merkel, Meissner, Pacinian, and Ruffini cells	 Aβ	6–12 μm	35–75 m/s
Pain, temperature	Free nerve endings	 Aδ	1–5 μm	5–30 m/s
Pain, temperature, itch	Free nerve endings	 C	0.2–1.5 μm	0.5–2 m/s



Diameter / Conduction velocity

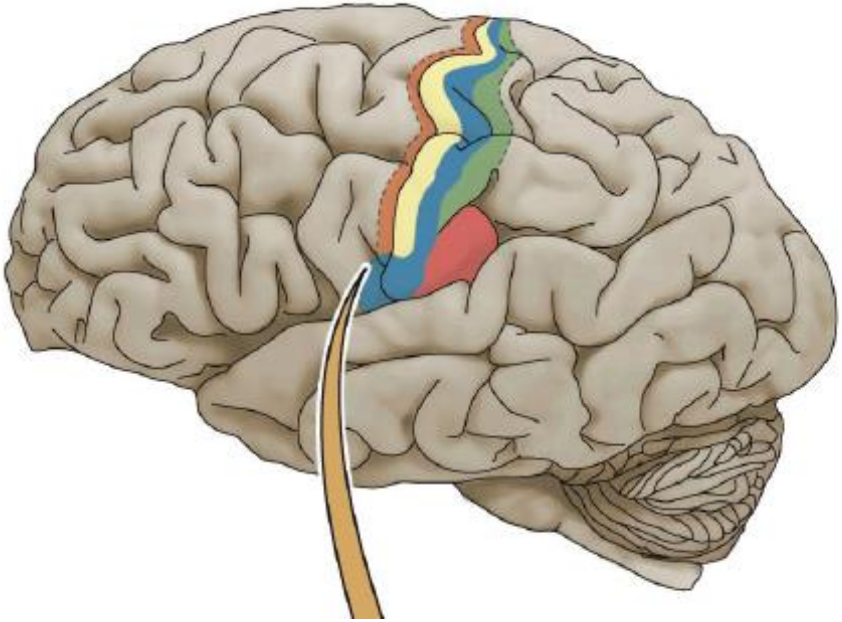
Cutaneous Mechanoreceptors

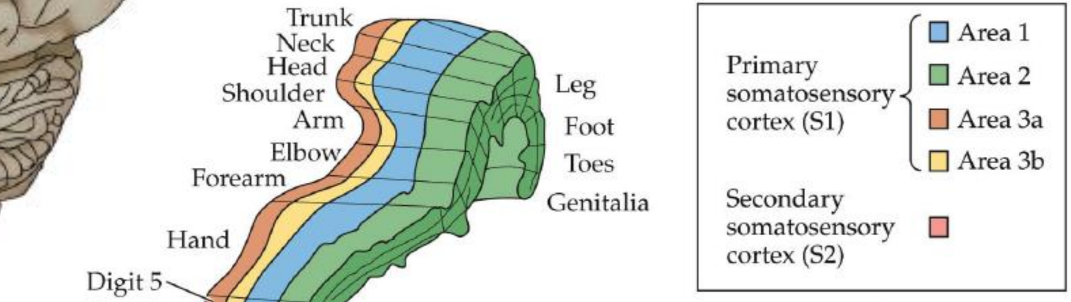
	Receptor	Diam	Density Fib/cm2	Resp	Percep. Function
SA1	Merkel	2mm	100	curvature	Form, texture
RA	Meissner	5mm	150	Motion	Motion, grip
PC	Pacinian	Hand	20	Vibration	Tools & probes

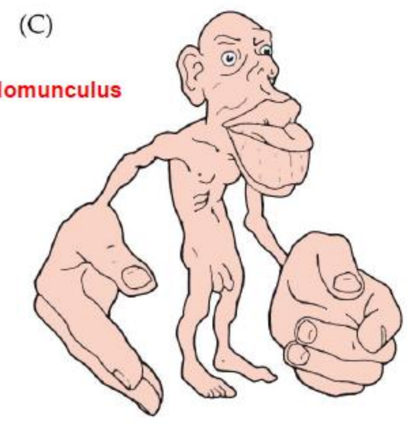


Rapidly adapting (RA)
Slowly adapting (SA)
A Modality
Touch
Receptors
Meissner's corpuscle
Merkel cells
Pacinian corpuscle
Ruffini endings
B Location
Receptive field
C Intensity and time course
Neural spike train
Stimulus

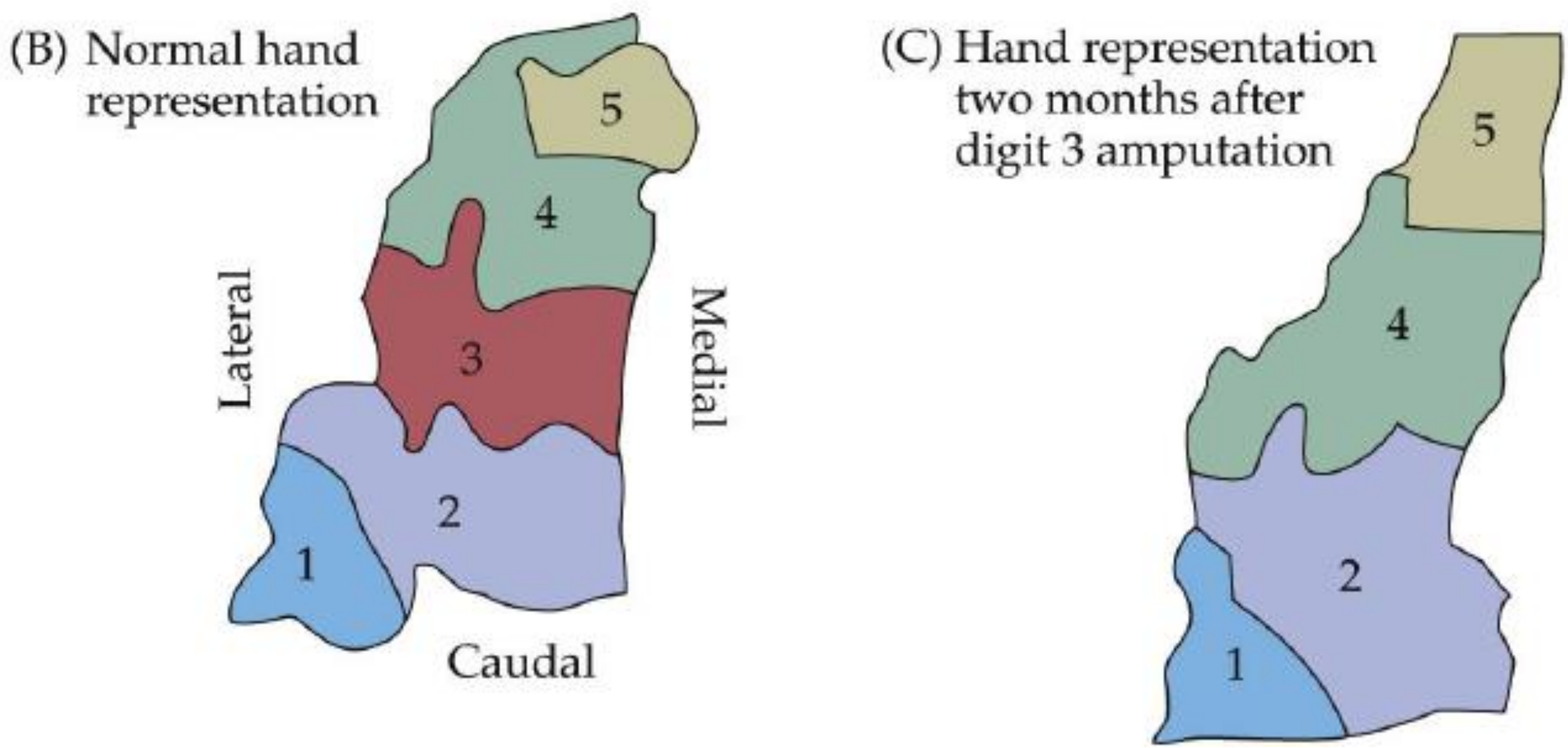
Somatotopic Organization





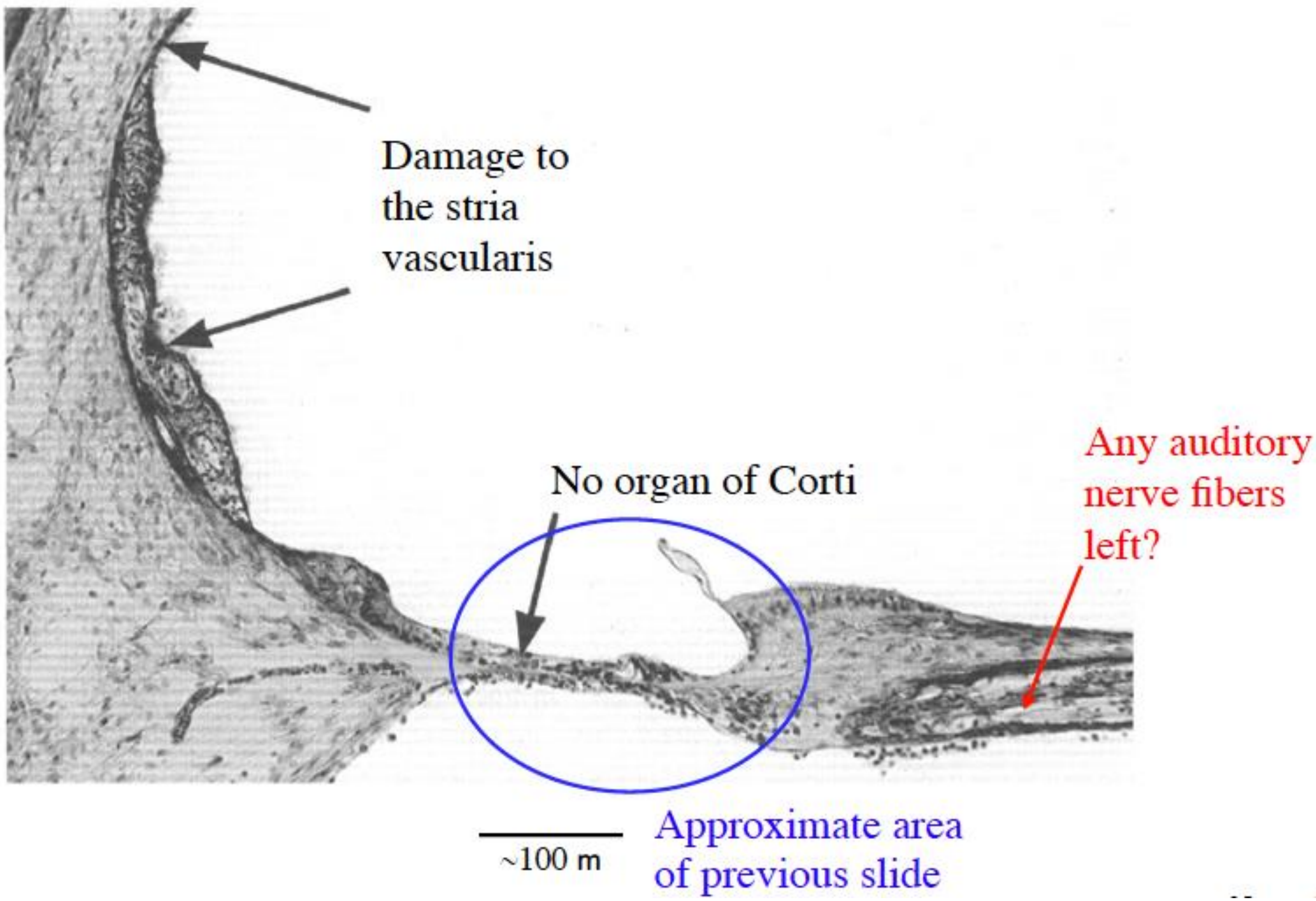


Plasticity


(B) Normal hand representation
5
4
3
2
1
Lateral
Medial
Caudal
(C) Hand representation two months after digit 3 amputation
5
4
2
1

Dr. Young’s Lectures

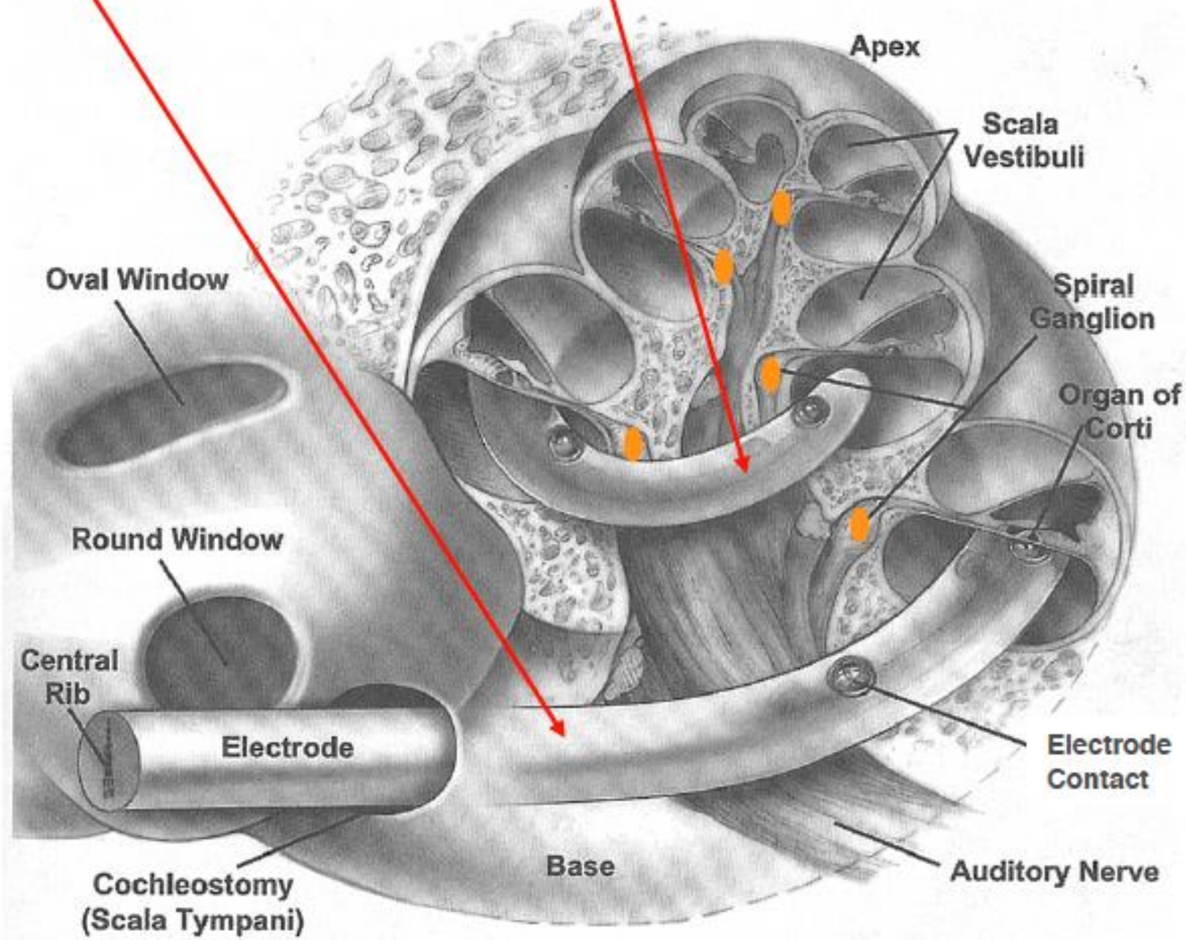
Damage to Hair Cells



The main practical technical challenge is the coiling of the cochlea.

It is necessary to design a bundle of electrodes that can be inserted through the round window into scala tympani pushed as far up the cochlear coil as possible.

Two problems: how to avoid damaging the remaining cochlea, especially the nerve; and how to stimulate fibers in the apex, where the electrode won't reach.



Orange ellipses are the locations of spiral ganglion cell bodies (auditory nerve fiber cell bodies).

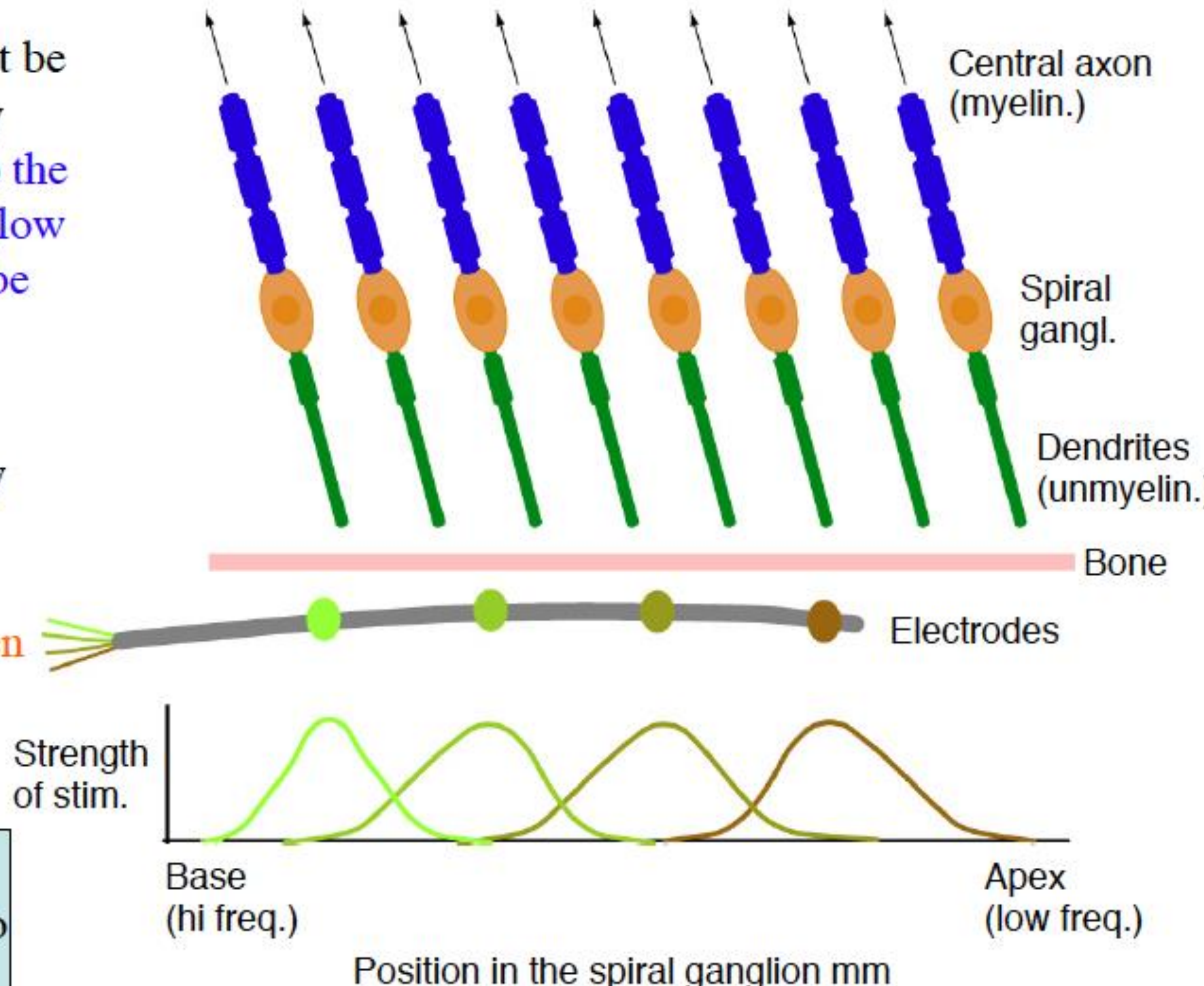
Loeb, 1985

Coding issues in implants reduce to how the stimulus is applied to the fibers and how independently different fibers can be activated.

This schematic sketch shows the problems:

1. The electrodes can't be inserted all the way into the cochlea, so the most apical fibers (low frequencies) can't be stimulated.

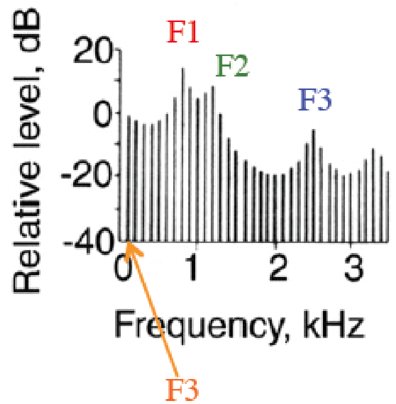
2. The electrodes are embedded in a very conductive fluid chamber causing interactions between electrodes.

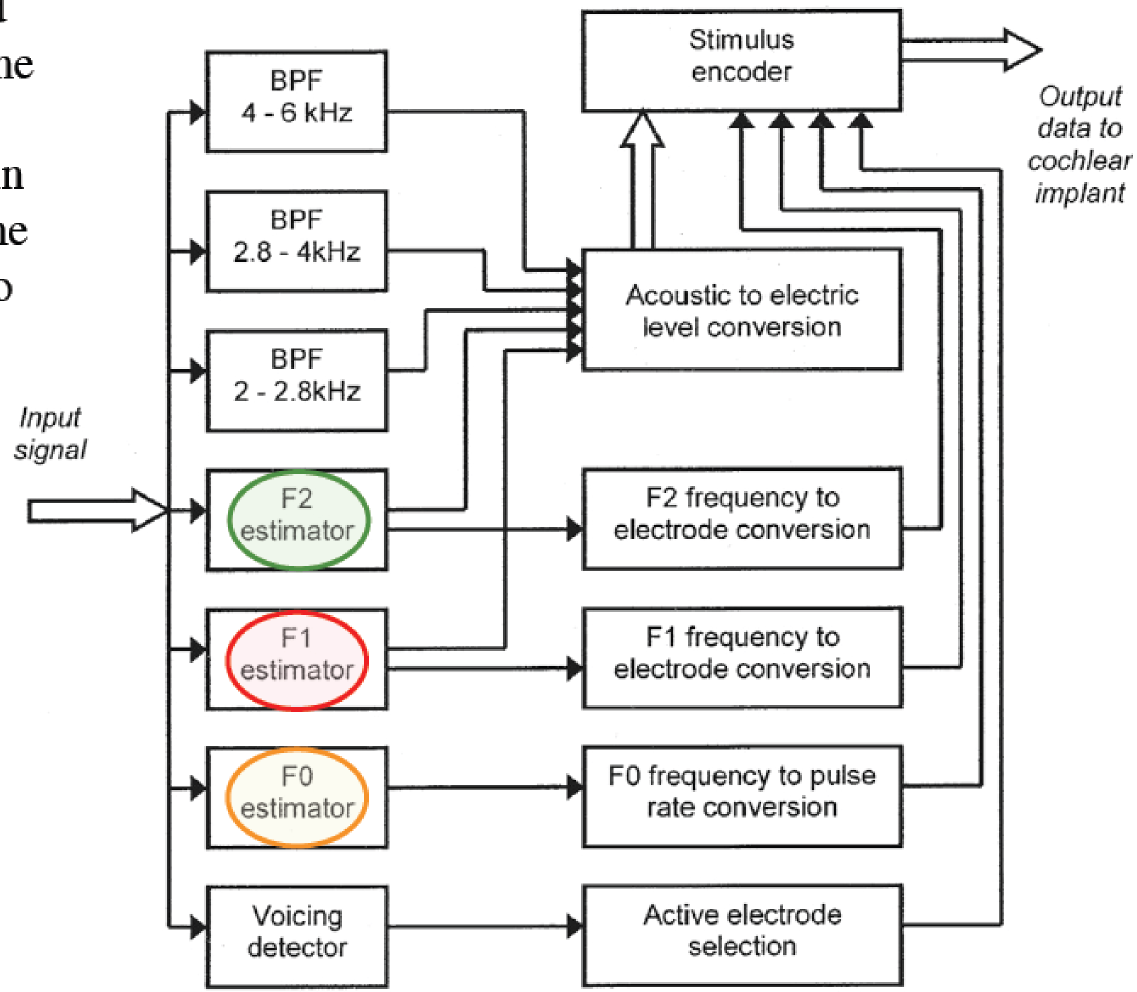


#2 limits the number of independent electrodes to about 8.

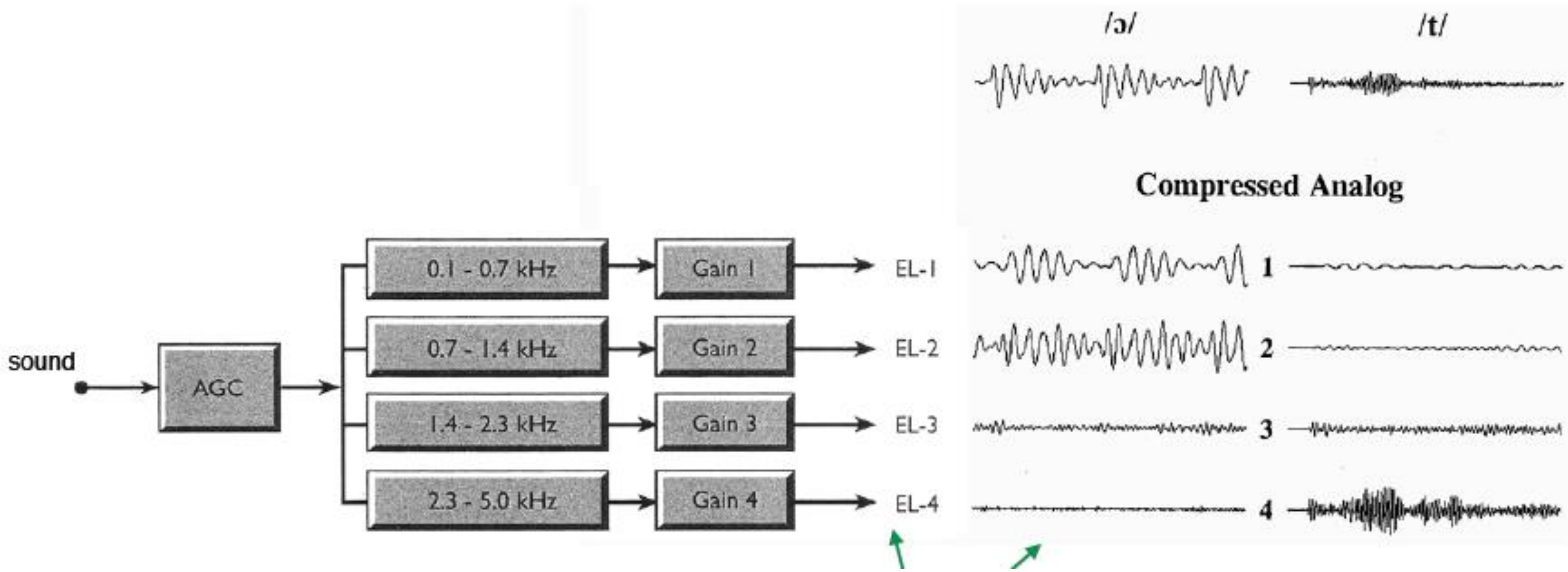
Cochlear Implant (CI) Attempts

A cool idea that turned out bad: coding both the general frequency content of the sound (in the first 3 channels), the frequencies of first two formants F1 and F2, and the voice periodicity (F0).





CI: Compressed Analog



Electrode interaction is a severe limitation on the performance of the CA processor.

CI: Continuous Interleaved Sampling

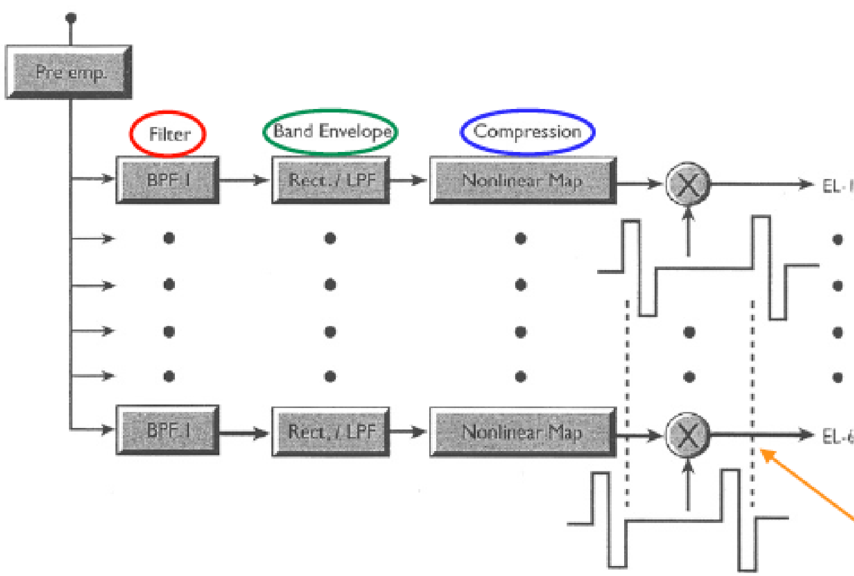
A bank of bandpass filters that do a tonotopic analysis of the speech.

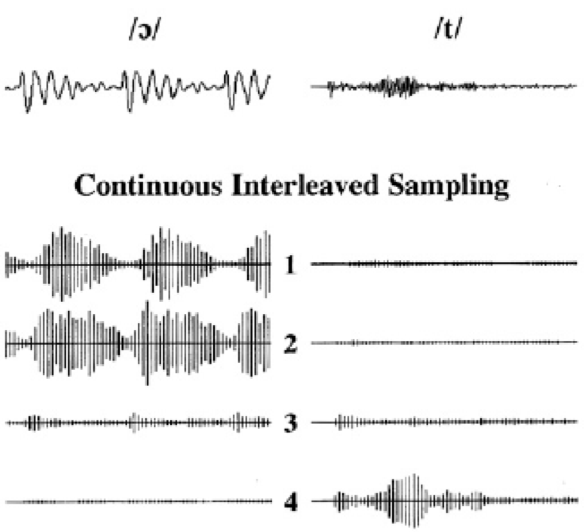
Rectification to extract the *envelope* of the signals from the filters.

The envelope sets the amplitudes of bipolar electrical pulses on the electrodes.

Compression is done in mapping the envelope to the pulse amplitudes.

Stimulation is done at a very high rate (>400 Hz, can be over 1 kHz), but non-simultaneously on the different electrodes to reduce electrode interaction.





Note the non-simultaneous electrode activation!

Some facts to ponder:

1. The implants can't be inserted all the way into the cochlea. This means that low frequencies can't be connected to the lowest-frequency auditory nerve fibers. Apparently the brain can overcome this problem.

2. Cochlear implants typically have fewer than 8 functional channels (although they may have as many as 22 electrodes). The normal human cochlea has 3,000 inner hair cells and 30,000 auditory-nerve fibers. There are 14-19 independent channels (critical bands) in the speech frequency range. Why do implants work with so few channels?

3. A big difficulty in implant design is electrode interaction, i.e. different electrodes stimulate overlapping populations of fibers. This limits how many independent electrodes there can be. Strangely bipolar electrode arrangements don't improve performance.

4. The implant doesn't activate fibers "naturally". Fibers usually discharge randomly and don't fire at rates above ~200 Hz. Would it make implants better if they activated fibers more normally?

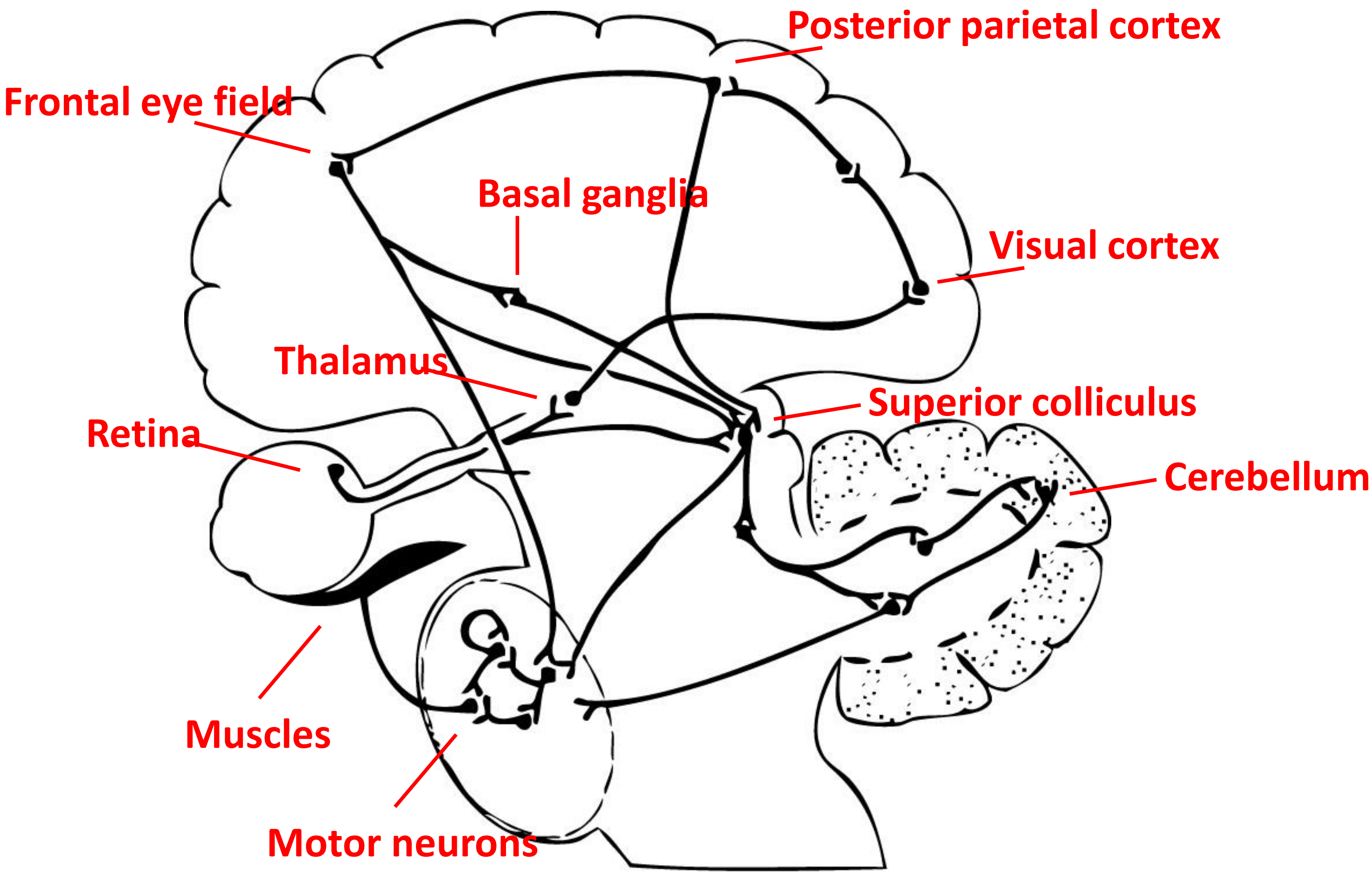
5. Plasticity in the central auditory system is essential. This means counteracting degeneration of central circuits. It can take 1-2 years to "learn" to use a cochlear implant.

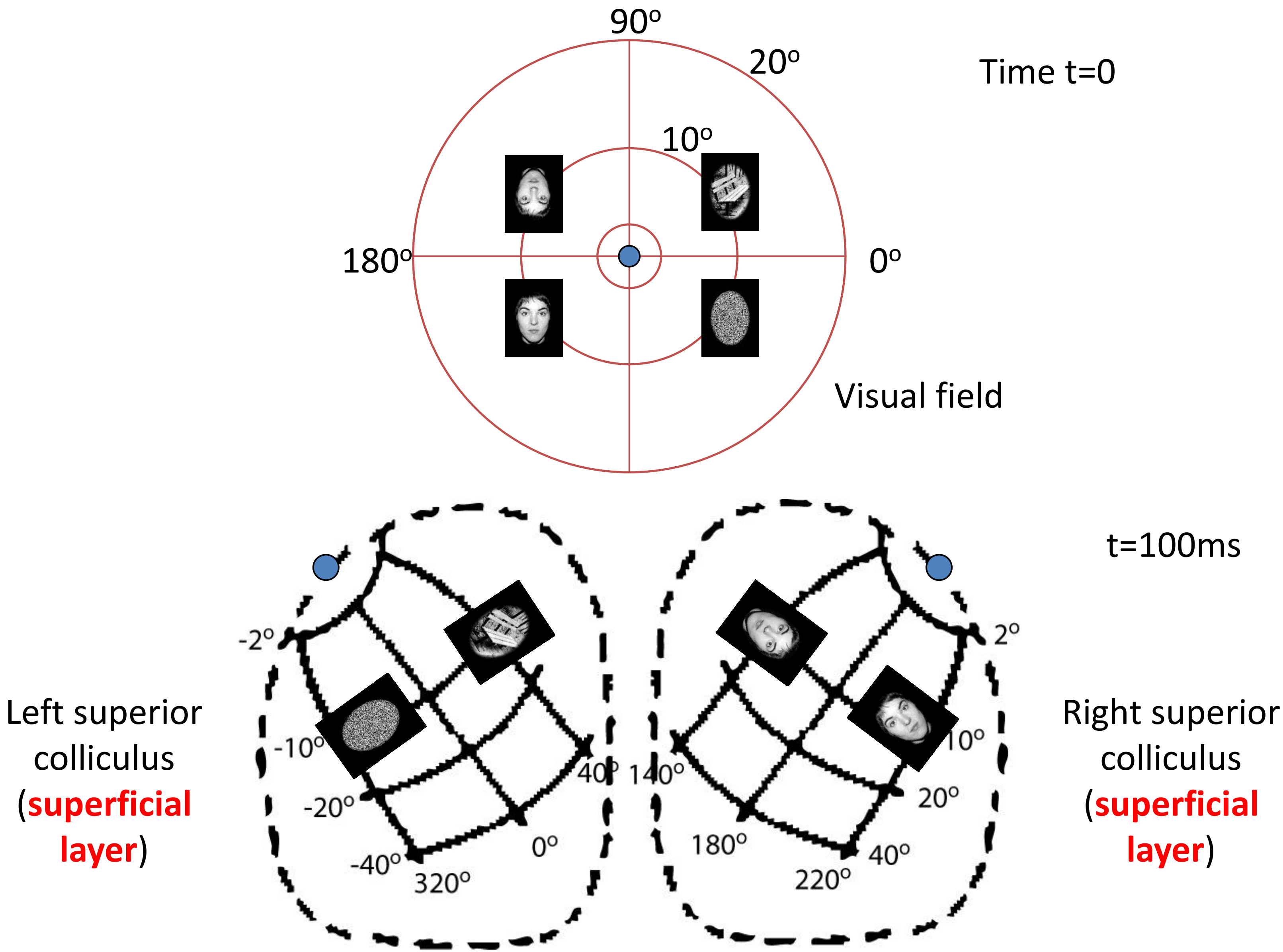
6. Implants only work for speech if the subject has adequate language. Thus persons who lost their hearing after learning language do much better than those who were deafened very early or were born deaf.

7. In many situations (e.g. background noise), we are helped by being able to compare the stimuli at two ears. Current CIs don't do that. Would it help if they did?

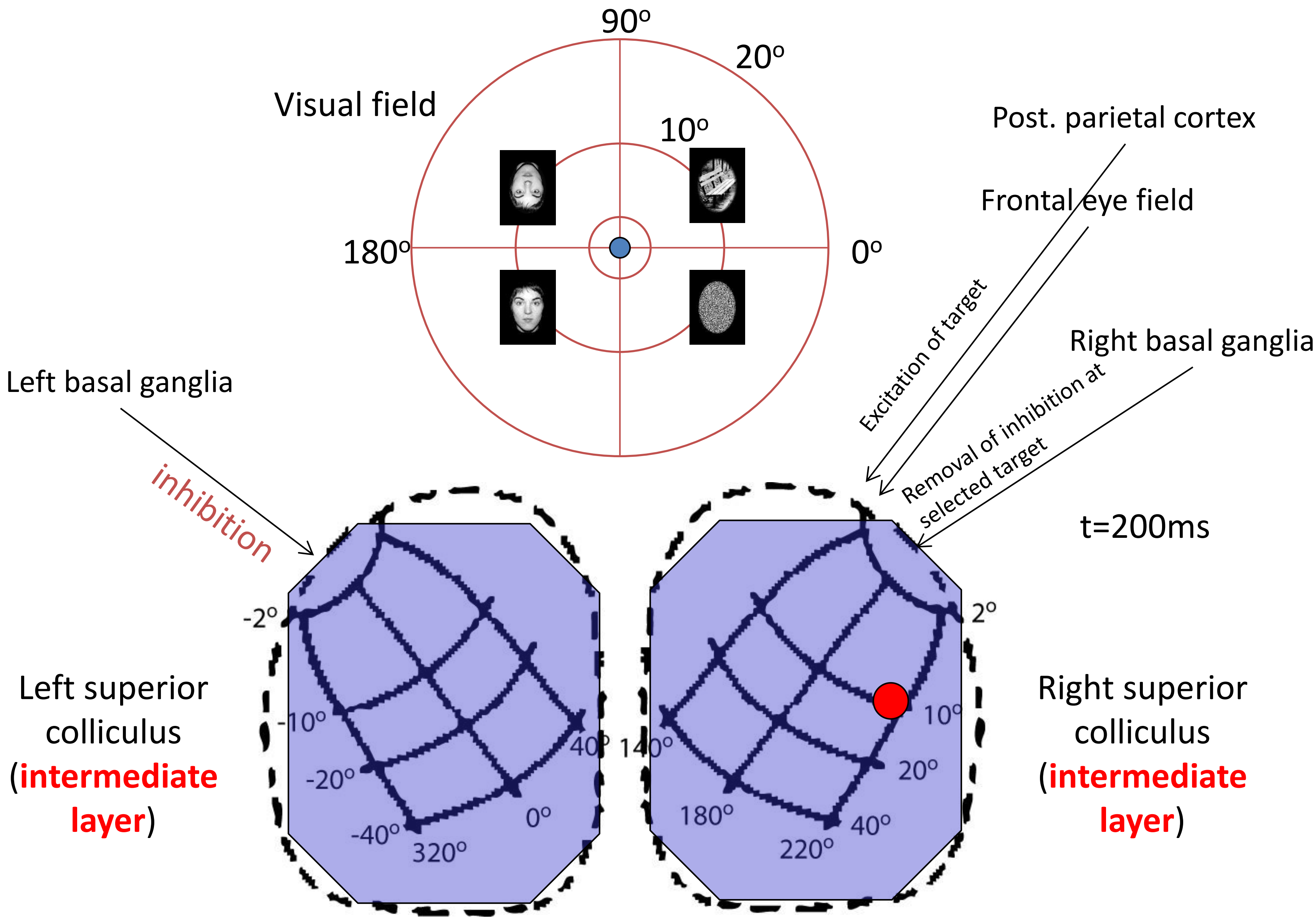
Dr. Shadmehr’s Lectures

Visual Pathway is Not As Simple As Previously Discussed





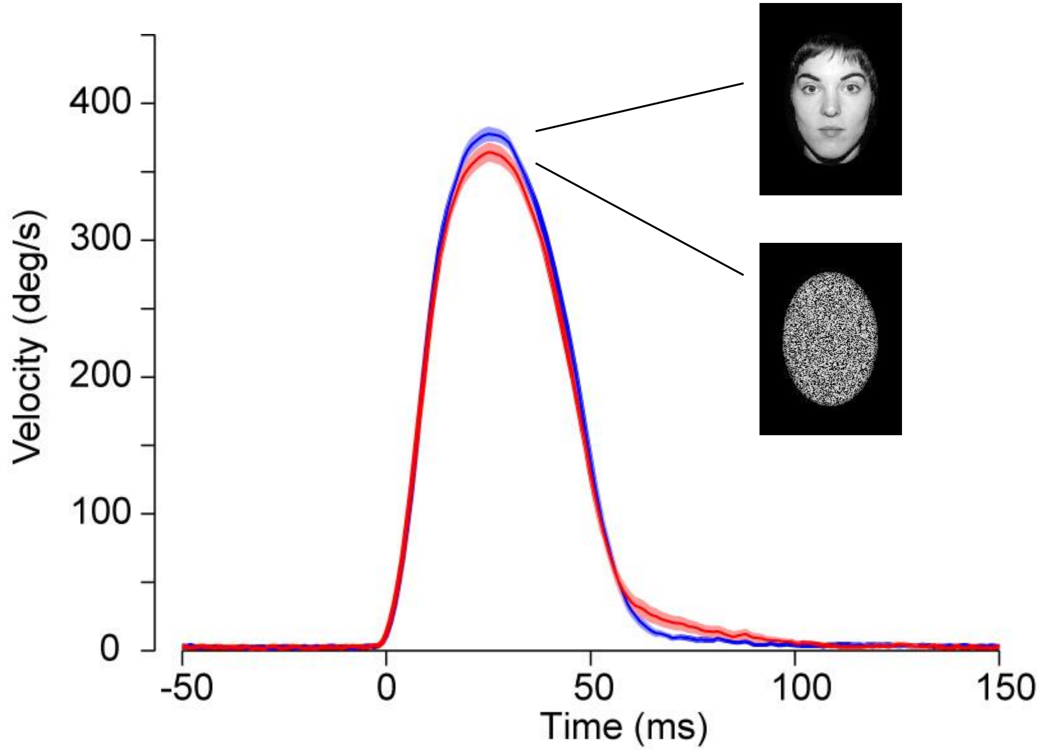
Superficial layers of superior colliculus (SC) have neurons that respond to visual information on the retina.
Right SC has a map of the left visual field. Left SC has a map of the right visual field.



A saccadic eye movement occurs where there is a burst of activity on the map of visual space on the intermediate layers of superior colliculus.

Saccades to faces are faster

Eye velocity



- The basal ganglia, along with the posterior parietal cortex and the frontal lobe, assign value to the various available actions (different movements that can be done).
- We tend to produce a movement toward the stimulus that has the highest subjective value.
- **IDEA #2: the relative value of the stimulus affects movement speed. We move faster toward stimuli that our brain assigns a greater value.**

Xu-Wilson et al. (2009)

Economics of motor control

Economics of motor control

We move to acquire a rewarding state. This is the **gain (reward)**.

However, we must pay for that action by consuming energy. This is the **loss (cost)**.

The utility of a movement is the sum of gains and losses of that movement, discounted by time.

We should move in a way that maximizes the utility.

Subjective value of reward

Energetic cost of the movement

Utility of the movement

$$J = \frac{\alpha - e(T)}{1 + \gamma T}$$

Temporal discounting factor

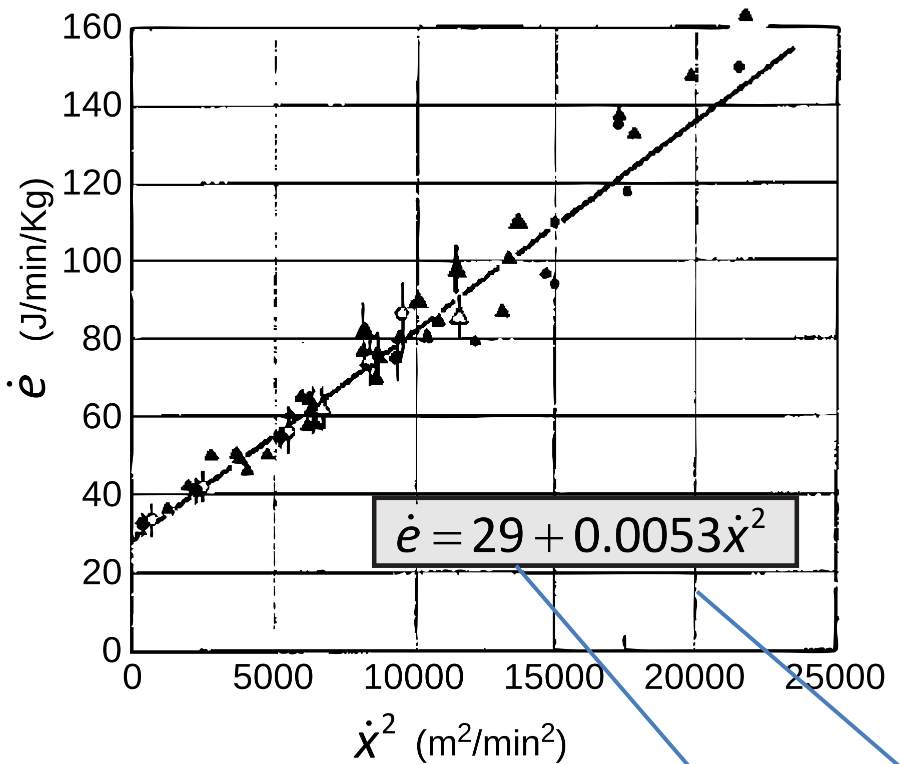
Duration of the movement

Use the utility equation to discuss:

- Walking
- Arm movement in a horizontal plane
- Perceived cost of applying a force

Animals move at approximately the speed that minimizes energetic cost.

Energy consumption per min per kg



$$\frac{\dot{e}}{\dot{x}}$$

Average speed of walking (squared)

Metabolic cost (per min) of walking

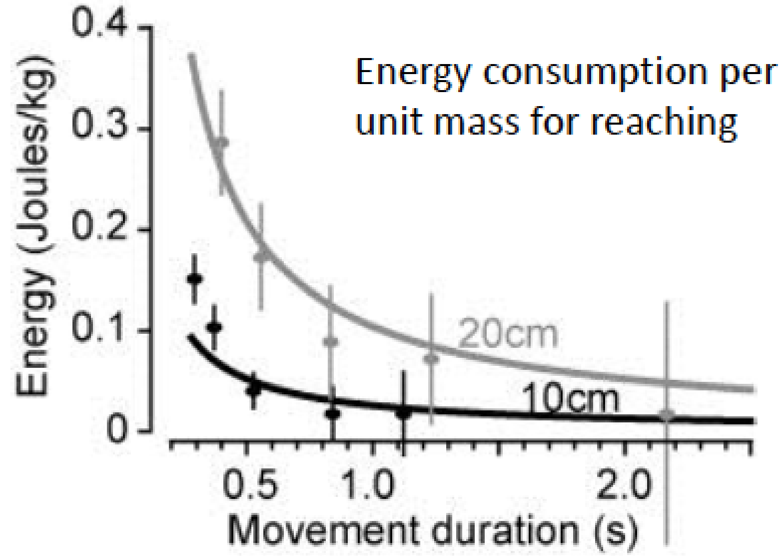
Metabolic cost (per min) of being alive

Movement involves expenditure of energy.
How does energy vary with duration and distance of a movement?

$\dot{e} = a + cE\left[\dot{x}\right]^2$ Total rate of energy consumption per unit mass during walking

$\dot{e}_m = cE\left[\dot{x}\right]^2$ Rate of energy consumption per unit mass for the act of walking

$e_m = c\dfrac{d^2}{T}$ Total energy consumption per unit mass as a function of distance and duration



$c = 2.6\ \text{J.sec} / \text{Kg} / \text{m}^2$

$e = mc\dfrac{d^2}{T}$ Total energy consumption as a function of mass, distance, and duration of the movement.

Movement utility as sum of reward and effort

Utility of the movement $$J = \frac{\underbrace{\alpha}_{\text{reward}} - \underbrace{mcd^2/T}_{\text{effort}}}{1+\gamma T}$$

We want to find the *T* (movement duration) that maximizes this utility.

$$\frac{dJ}{dT} = \frac{cmd^2\left(1+2\gamma T\right) - \alpha\gamma T^2}{T^2(1+\gamma T)^2}$$

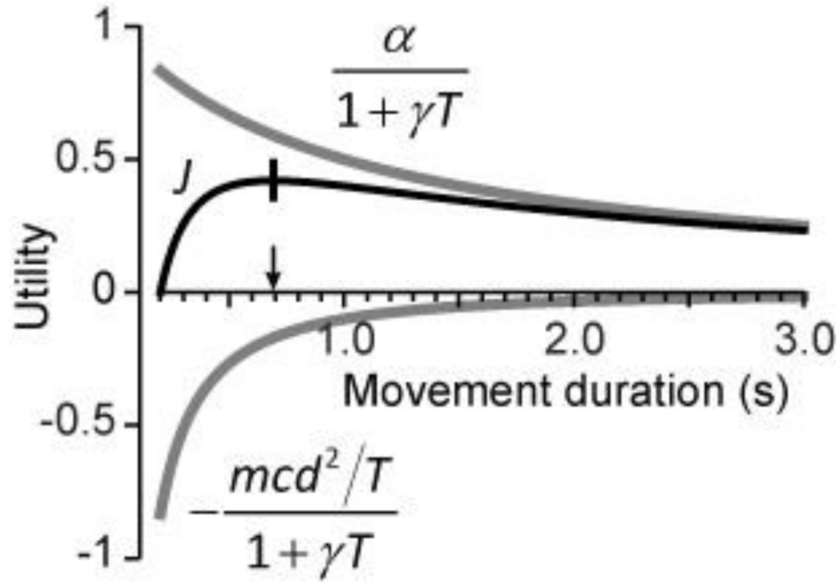
Duration of the movement that maximizes the utility $$T^* = \frac{cd^2m + \sqrt{c^2d^4m^2 + \alpha cd^2m\gamma^{-1}}}{\alpha}$$

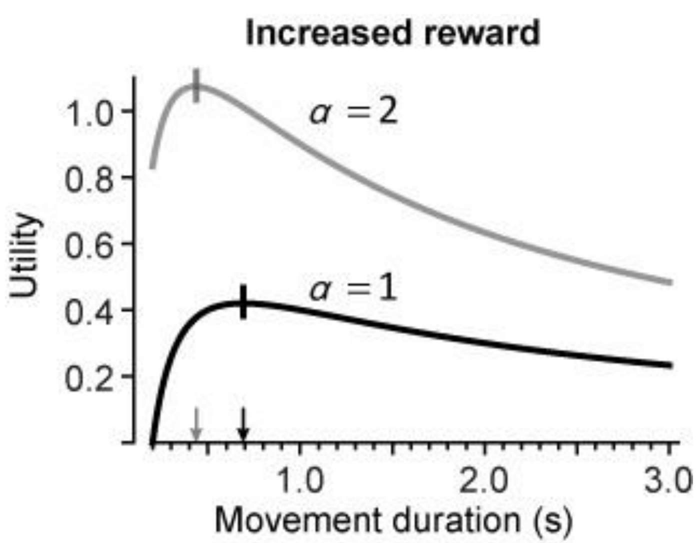
- As mass increases, utility decreases (option is less preferred), and duration of the movement increases (movement is slower).
- As reward increases, utility increases (option is more preferred), and duration of the movement decreases (movement is faster).

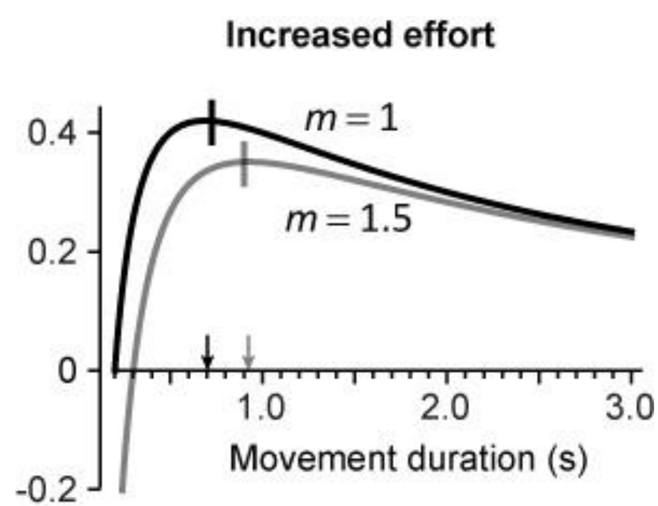
Movement utility as sum of reward and effort

reward effort

$$J = \frac{\alpha - mcd^2/T}{1+\gamma T}$$

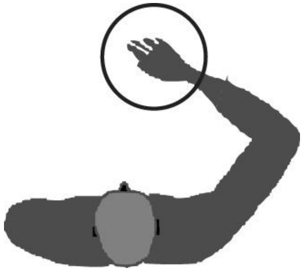






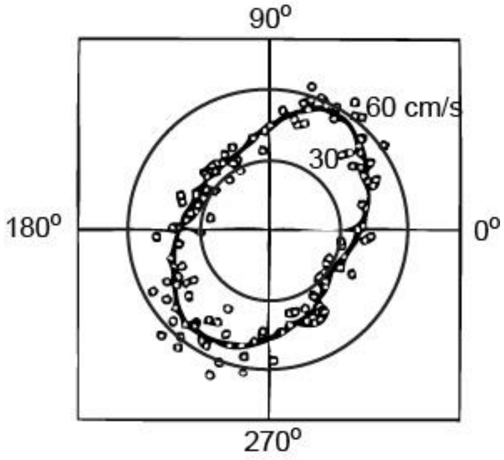
Reaching speed is slower for directions that have greater mass

Gordon et al. (1994)



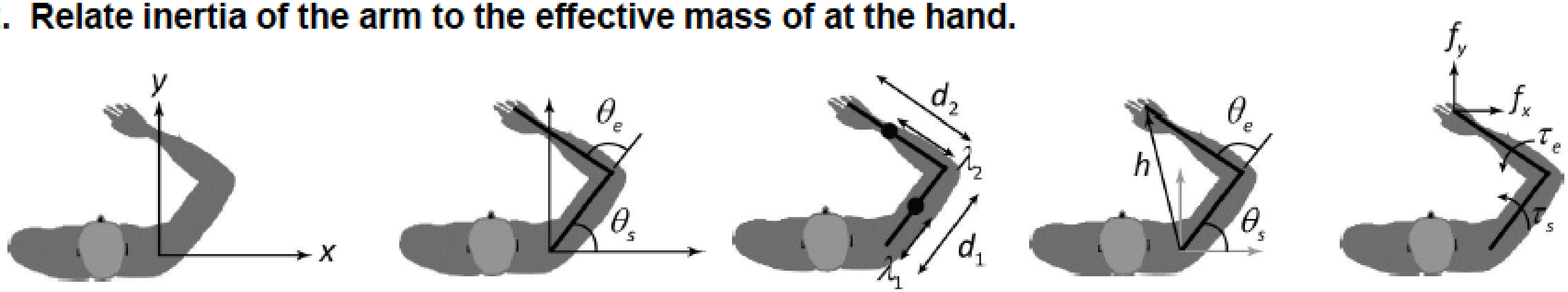
They asked their subjects to make a single, uncorrected movement to a target at 10cm, with no time constraints on the movement.

Peak velocity



Estimating the mass of the arm for movements in various directions

2. Relate inertia of the arm to the effective mass of at the hand.



$$\tau = \begin{bmatrix} \tau_s \\ \tau_e \end{bmatrix} \quad \theta = \begin{bmatrix} \theta_s \\ \theta_e \end{bmatrix}$$

$$\tau = H\ddot{\theta}$$

At rest, inertia describes the relationship between forces and torques in joint coordinates

$$h = \begin{bmatrix} d_1\cos(\theta_s) + d_2\cos(\theta_s + \theta_e) \\ d_1\sin(\theta_s) + d_2\sin(\theta_s + \theta_e) \end{bmatrix}$$

Location of the hand in Cartesian coordinates written in terms of joint angles

$$f = \begin{bmatrix} f_x \\ f_y \end{bmatrix} \quad f = M\ddot{h}$$

At rest, the mass matrix *M* describes the relationship between forces and acceleration in Cartesian coordinates

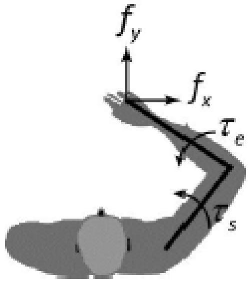
$$\Lambda = \frac{dh}{d\theta} = \begin{bmatrix} \frac{dh_x}{d\theta_s} & \frac{dh_x}{d\theta_e} \\ \frac{dh_y}{d\theta_s} & \frac{dh_y}{d\theta_e} \end{bmatrix}$$

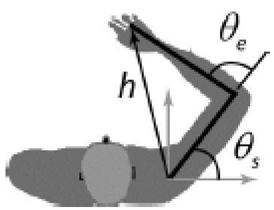
Jacobian matrix

$$= \begin{bmatrix} -d_1\sin(\theta_s) - d_2\sin(\theta_s + \theta_e) & -d_2\sin(\theta_s + \theta_e) \\ d_1\cos(\theta_s) + d_2\cos(\theta_s + \theta_e) & d_2\cos(\theta_s + \theta_e) \end{bmatrix}$$

Estimating the mass of the arm for movements in various directions

2. Relate inertia of the arm to the effective mass of at the hand.





$$\tau = \Lambda^T f$$

Force and torques are related by the Jacobian matrix

$$\dot{h} = \Lambda\dot{\theta}$$

$$\ddot{h} = \frac{d\Lambda}{d\theta}\dot{\theta}\dot{\theta} + \Lambda\ddot{\theta}$$

Motion at the hand is related to motion at the joints by the Jacobian

$$\left.\begin{aligned} \tau &= H\ddot{\theta} \\ \tau &= \Lambda^T f \end{aligned}\right\} H\ddot{\theta} = \Lambda^T f$$

$$f = \left(\Lambda^{-1}\right)^T H\ddot{\theta}$$

$$f \approx \underbrace{\left(\Lambda^{-1}\right)^T H\Lambda^{-1}}_{M}\ddot{h}$$

$$f \approx M\ddot{h}$$

$$M = \left(\Lambda^{-1}\right)^T H\Lambda^{-1}$$

The above equation relates inertia *H* to mass *M*.

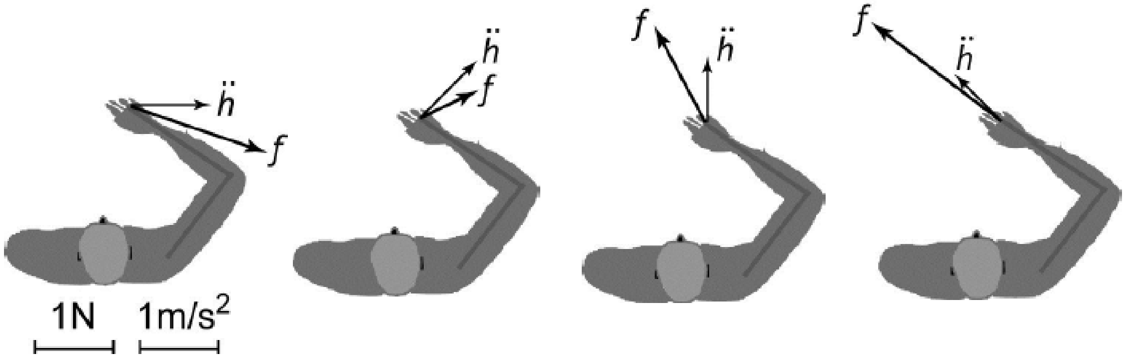
The mass at the hand *M* is a 2x2 matrix, relating acceleration of the hand to forces at the hand.

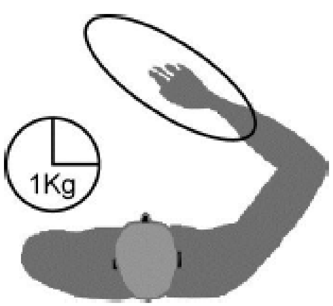
Reaching speed is slower for directions that have greater mass

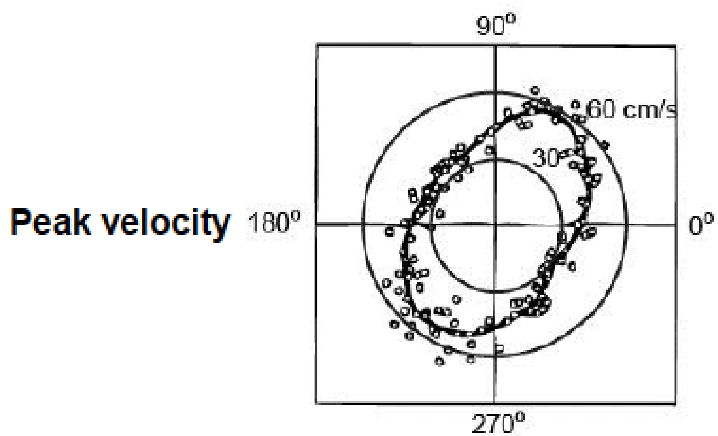
$$f \approx M\ddot{h}$$

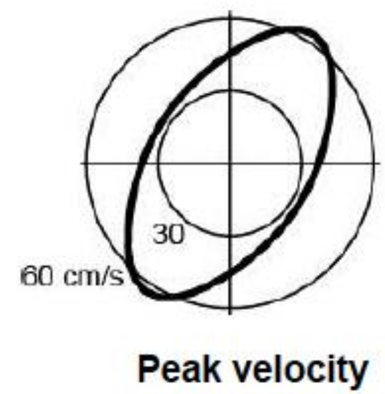
$$M = \left(\Lambda^{-1}\right)^T H \Lambda^{-1}$$

Pick a unit length acceleration of the hand in direction *p*. Using the mass matrix we can compute the required force. The magnitude of the resulting force vector is the effective mass *m* for a movement in direction *p*.

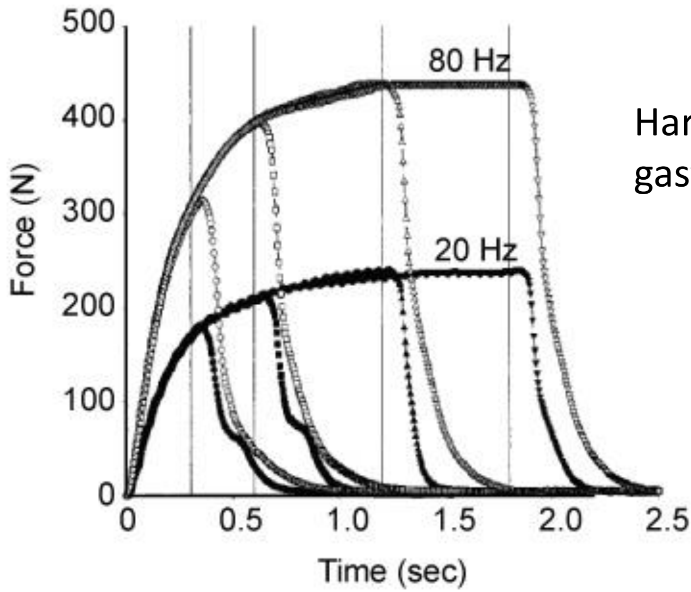








Metabolic cost of force production



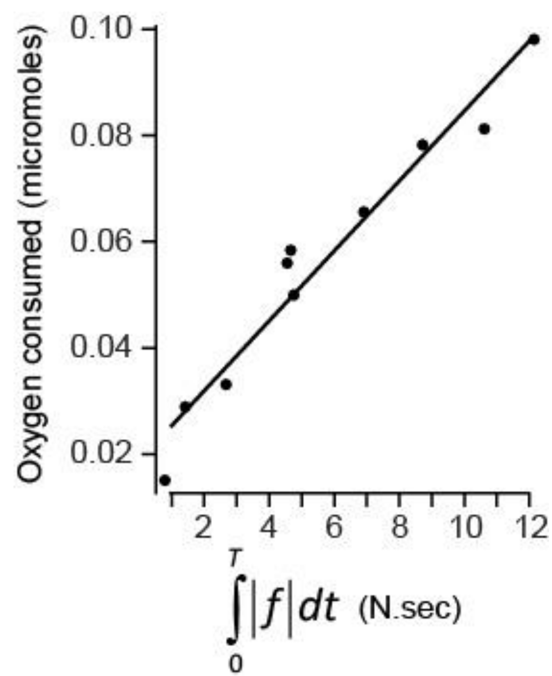
Harkema et al. (1997) stimulated the intact human gastrocnemius and used NMR to estimate consumption of ATP

$$f = k_i\left(1 - \exp\left(-t/\tau\right)\right)$$

$$e\left(f(t)\right) = a_1 \int_0^T \left|f(t)\right| dt + a_2$$

Energetic cost of force production

Kushmerick and Paul (1976): energy consumption in an electrically stimulated frog muscle



Perception of effort

The reward at stake

Energetic cost of force production

Utility of the action

$$J\left(f(t)\right) = \frac{\alpha - e(f(t))}{1 + \gamma T}$$

Duration of the force production

$$e(f(t)) = a_1 \int_0^T |f(t)| dt + a_2$$

Energetic cost of force production

Effort of the action

$$U(f(t)) = -\frac{a_1 \int_0^T |f(t)| dt + a_2}{1 + \gamma T}$$

$$U = -\frac{a_1 F T + a_2}{1 + \gamma T}$$

- As duration of force production increases, effort does not continue to grow, but reaches an asymptote.
- Prediction: perception of effort becomes indifferent to duration of force production as duration increases.

How limb movements are controlled

Control of a limb with antagonist muscles

1. Relating muscle force to joint torques using Principle of Virtual Work

$$\tau \, \Delta\theta \;=\; -f \; \Delta\lambda$$

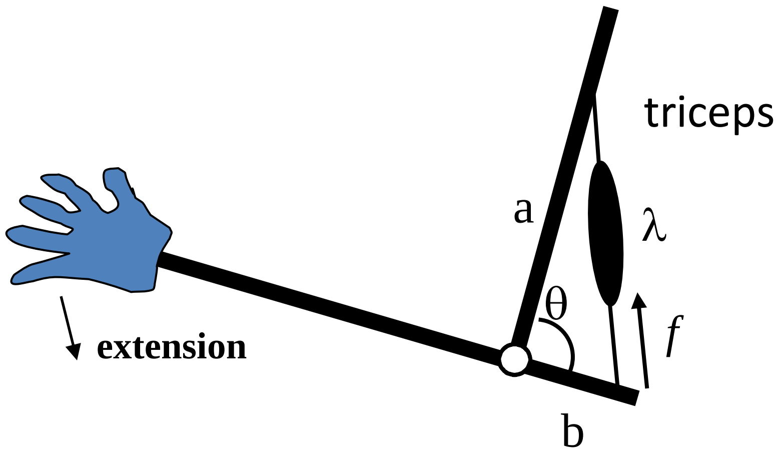
$$\tau = -\frac{\Delta\lambda}{\Delta\theta} \, f$$

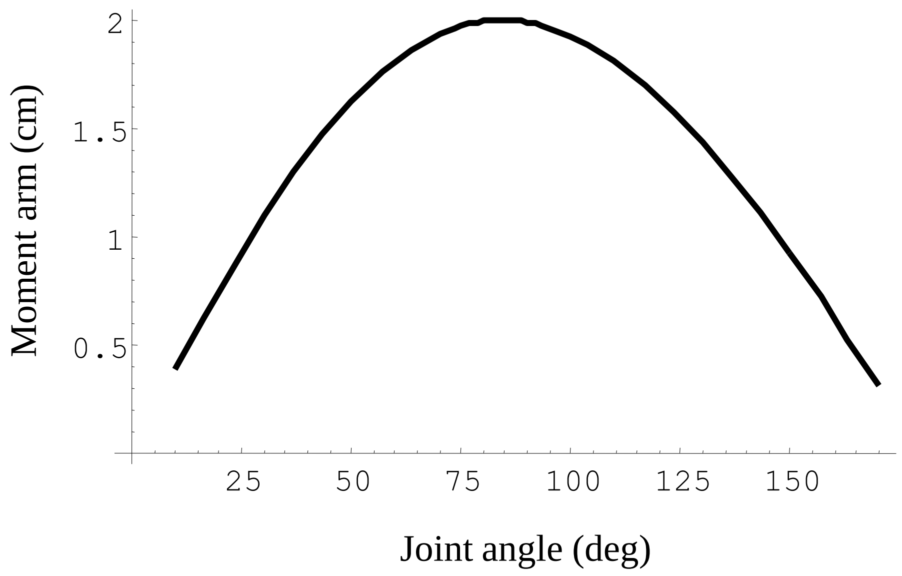
$$\tau = -\frac{d\lambda}{d\theta} \, f$$

$$\lambda = \sqrt{a^2 + b^2 - 2ab\cos(\theta)}$$

$$\frac{d\lambda}{d\theta} = \frac{ab\sin(\theta)}{\sqrt{a^2 + b^2 - 2ab\cos(\theta)}}$$

Moment Arm





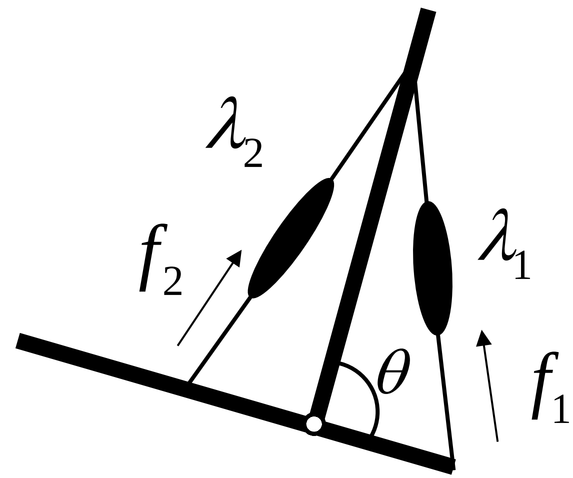
Control of a limb with antagonist muscles

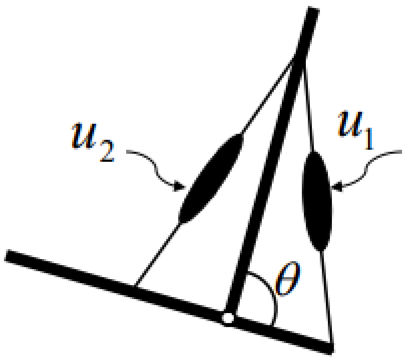
3. Torques produced by antagonist muscles sum at the joint

$$\tau_1 = -\frac{d\lambda_1}{d\theta} f_1$$

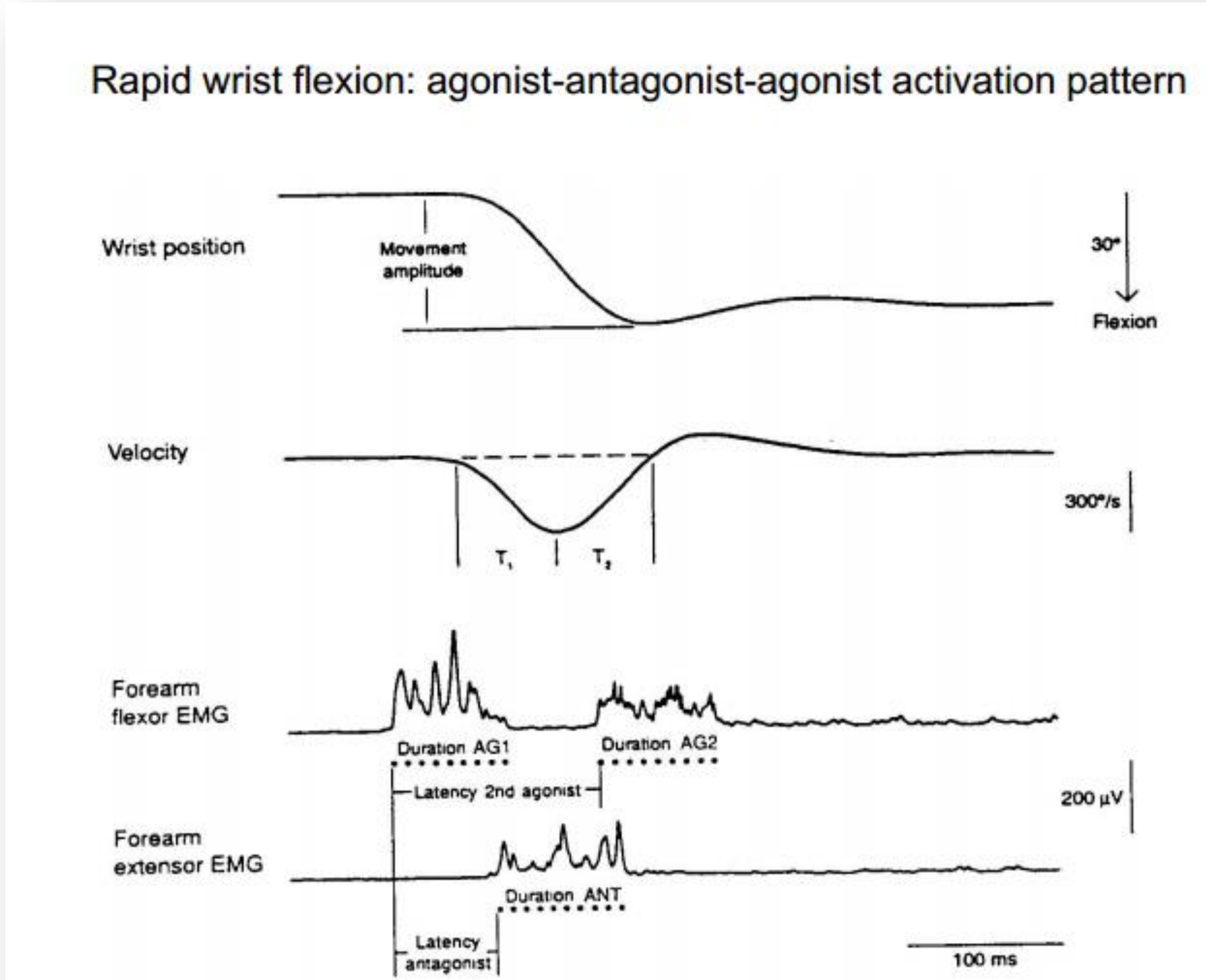
$$\tau_2 = \frac{d\lambda_2}{d\theta} f_2$$

$$\tau = \tau_1 + \tau_2$$





u_2
u_1
θ



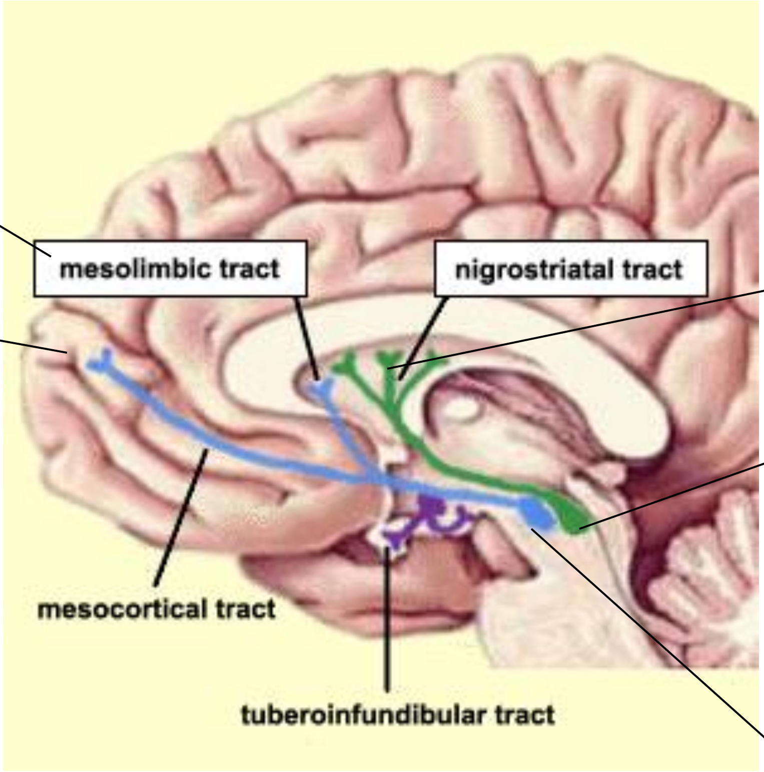
Rapid wrist flexion: agonist-antagonist-agonist activation pattern
Wrist position
Movement amplitude
30°
Flexion
Velocity
300°/s
Forearm flexor EMG
Duration AG1
Duration AG2
Latency 2nd agonist
200 µV
Forearm extensor EMG
Duration ANT
Latency antagonist
100 ms

Encoding of reward and effort by the neurotransmitter dopamine

Dopamine releasing neurons have their cell bodies in the brain stem: substantia nigra and the ventral tegmental area. They project to three main areas: the striatum (nigrostriatal tract), the hippocampus (mesolimbic tract), and the prefrontal cortex (mesocortical tract).

Limbic system: hippocampus and the medial temporal lobe

Prefrontal cortex



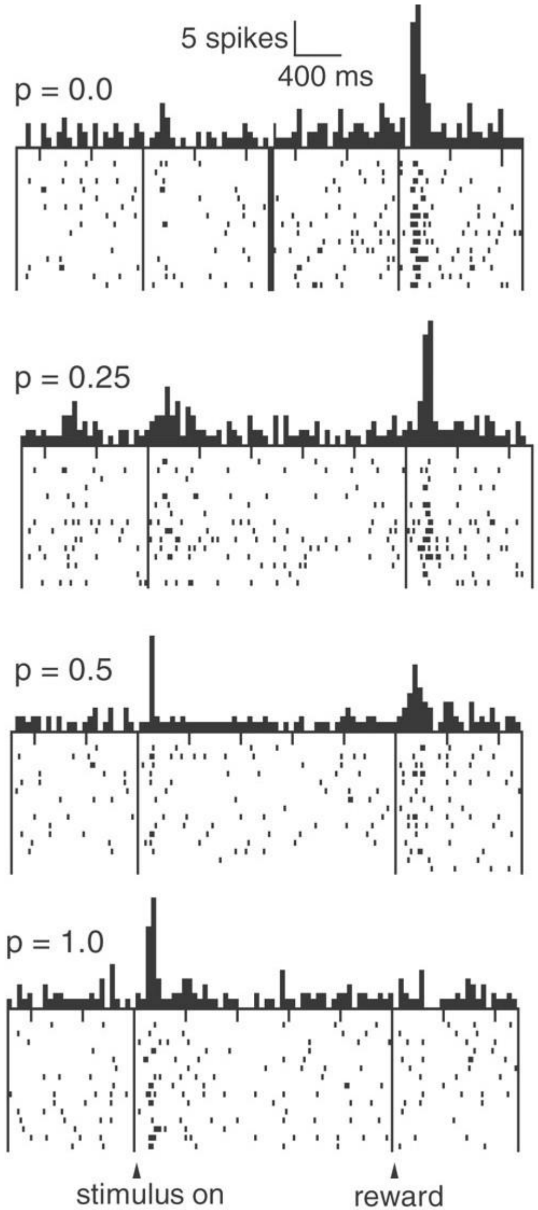
Striatum (caudate and putamen, parts of the basal ganglia)

Substantia nigra (part of basal ganglia)

Ventral tegmental area

Dopamine signals whether a stimulus is expected to be rewarding.

This figure shows a dopamine neuron in substantia nigra



Monkey was trained to associate each of 5 distinct visual stimuli with probability of getting juice.

p represents probability of reward. In this case it is near zero. Therefore, when reward is given, it is unexpected. The cell responds not at all to the stimulus, but strongly after the unexpected reward is given.

In this case p is near 1. Therefore, when reward is given, it is expected. The cell responds strongly to the stimulus, but not at all after the expected reward is given.

Fiorillo, Tobler, and Schultz (2003) Science

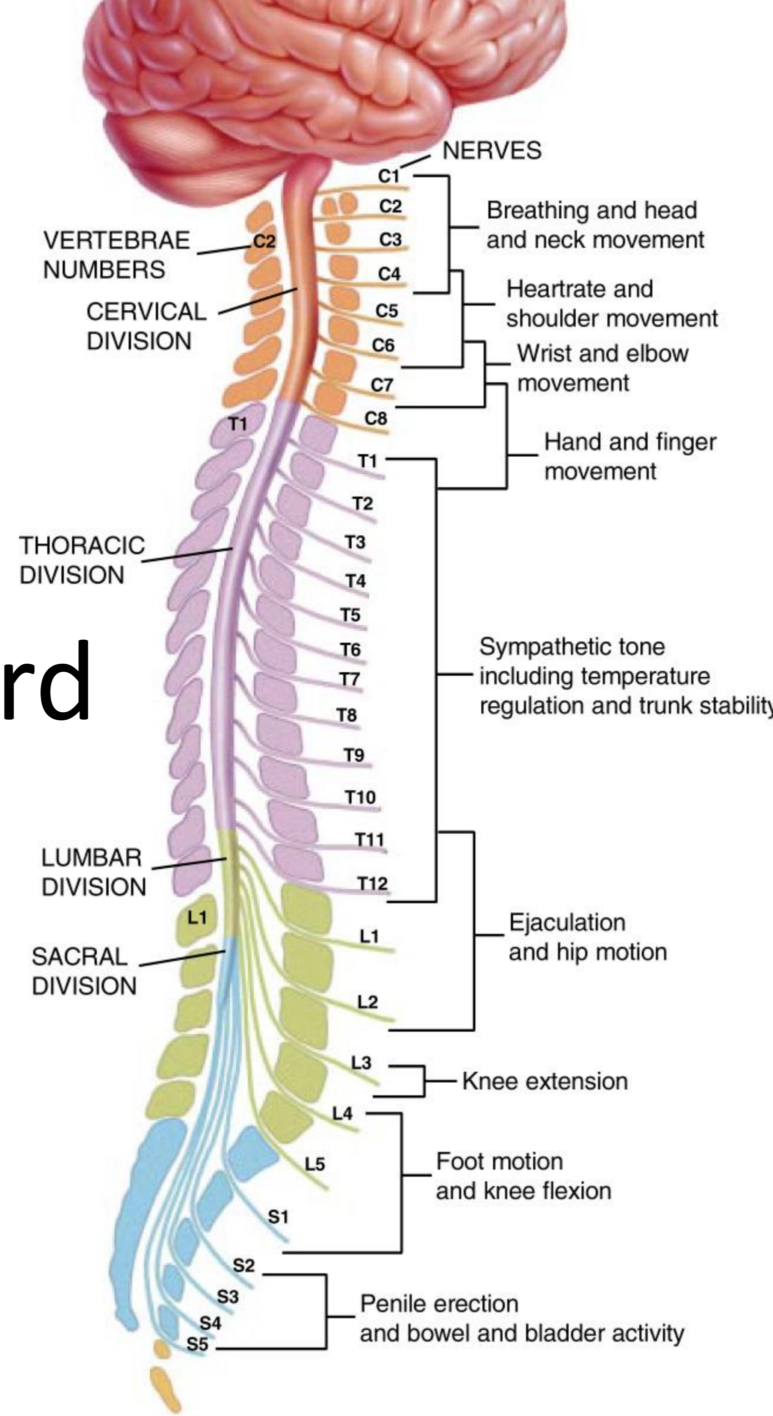
Parkinson's disease

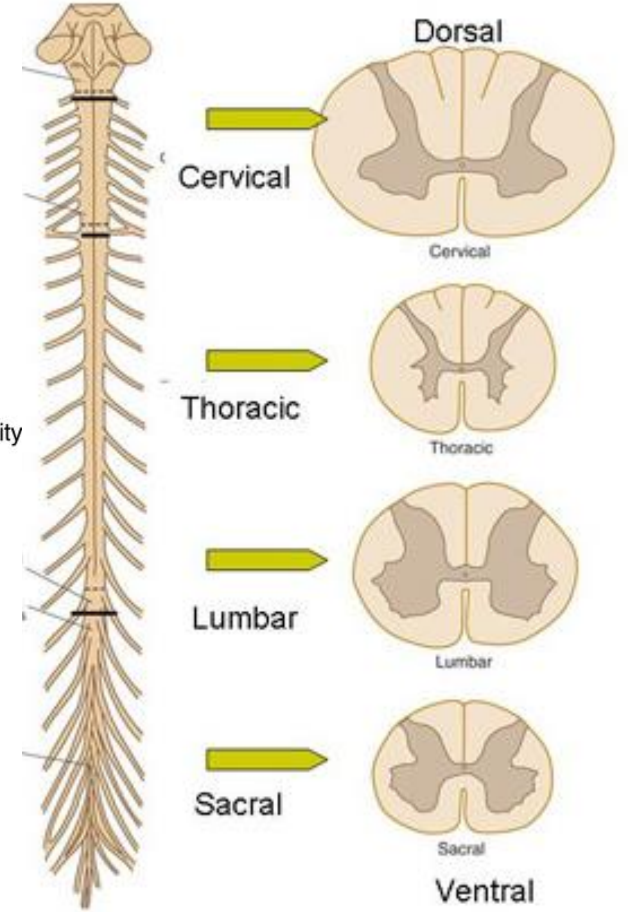
- In PD, dopamine neurons in the substantia nigra degenerate and die.
- Symptoms of the disease are slowness of movement (bradykinesia), resting tremor, rigidity of joints, and postural instability.
- The slowness of movement in particular appears to be context dependent: the patient may run out the building in case of a fire, but otherwise will have trouble moving at normal speeds.
- Treatments include dopamine replacement therapy.

Schizophrenia

- Symptoms include hallucinations and delusions, including unrealistic beliefs like superpowers.
- Pharmacological treatments that appear to help include dopamine antagonists, medication that blocks dopamine receptors.
- A component of schizophrenia appears to be an over-active dopamine system.

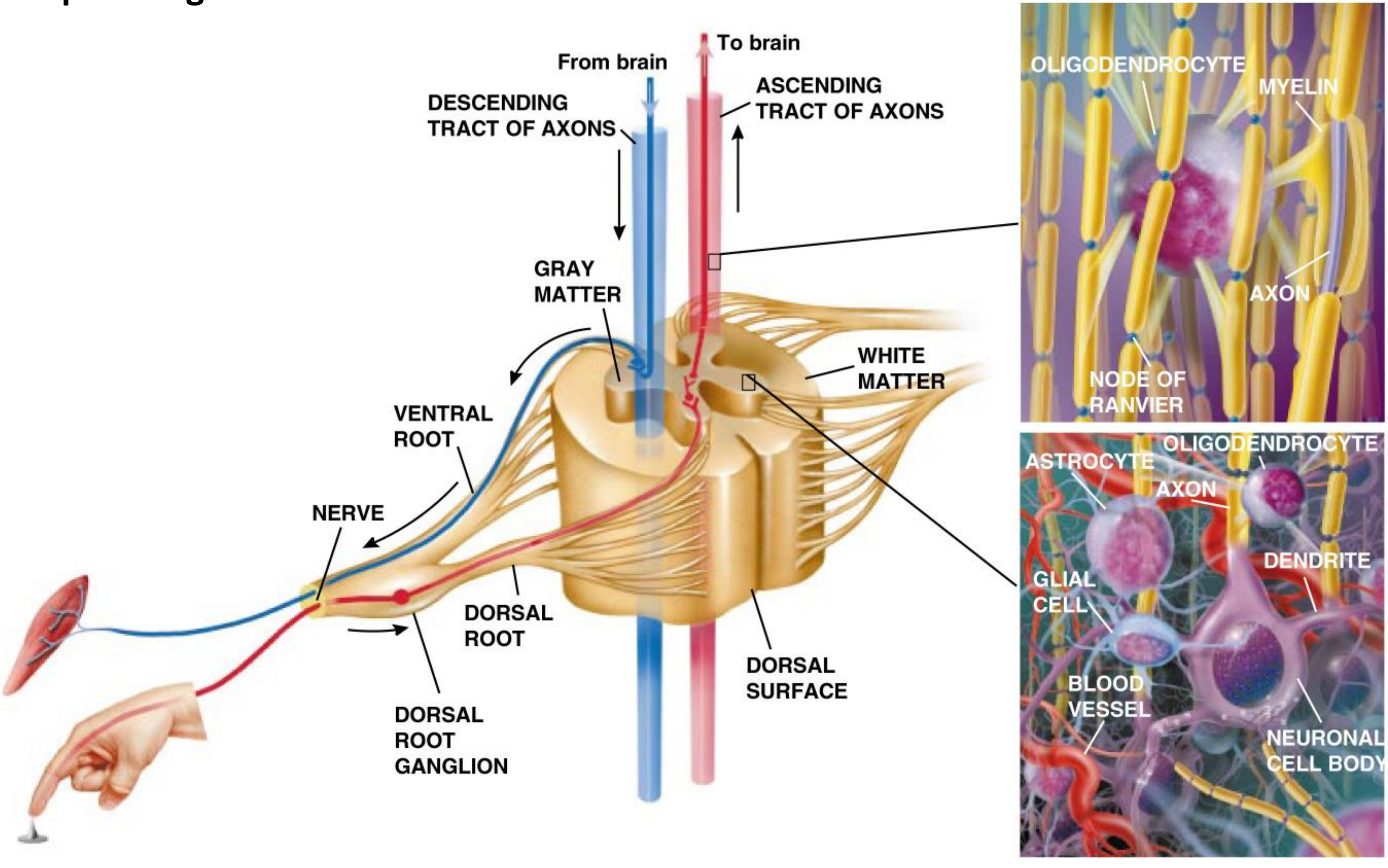
Spinal Cord





McDonald JW (1999) Sci Am 9:64-73

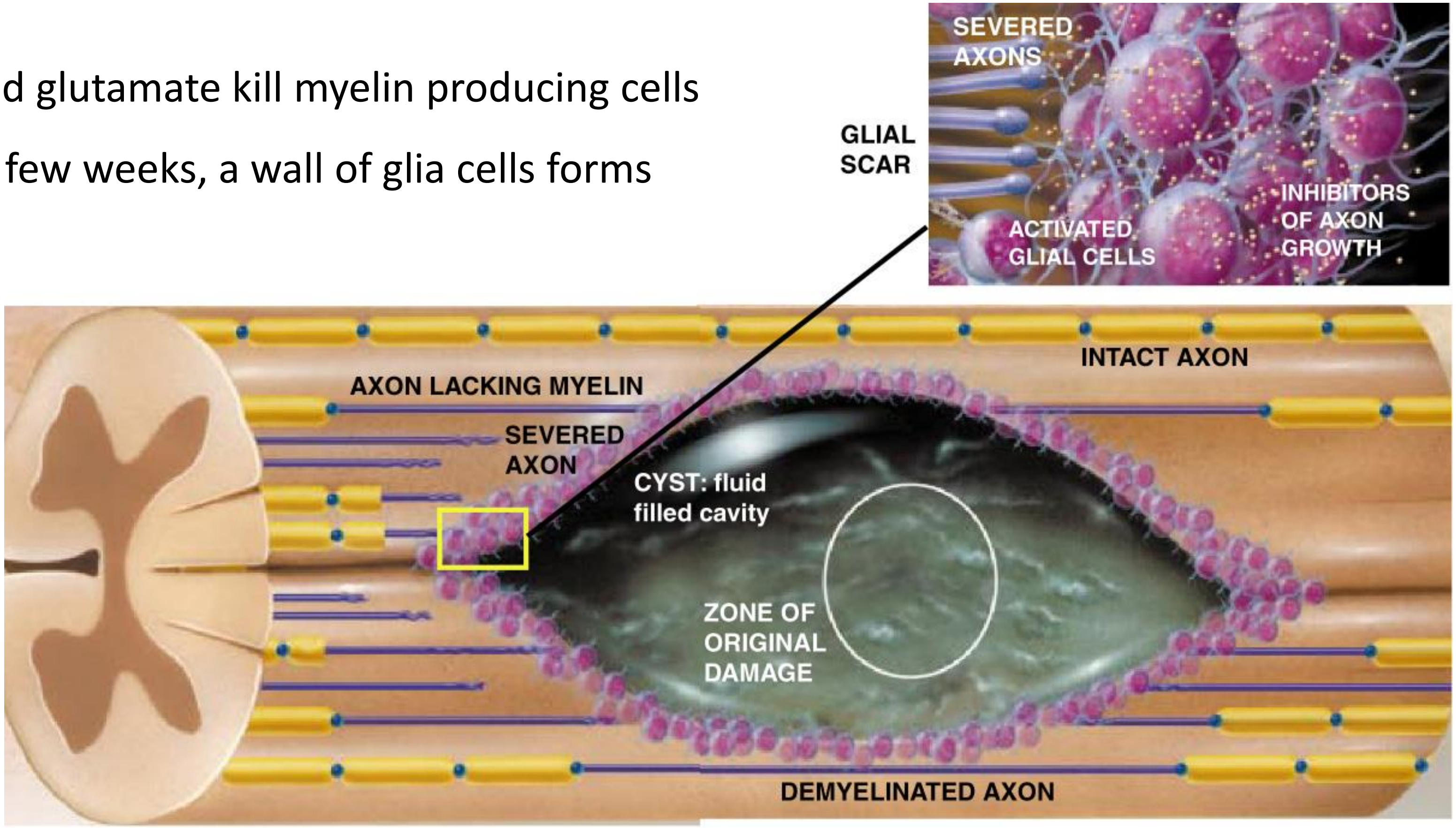
A spinal segment



McDonald JW (1999) Sci Am 9:64-73

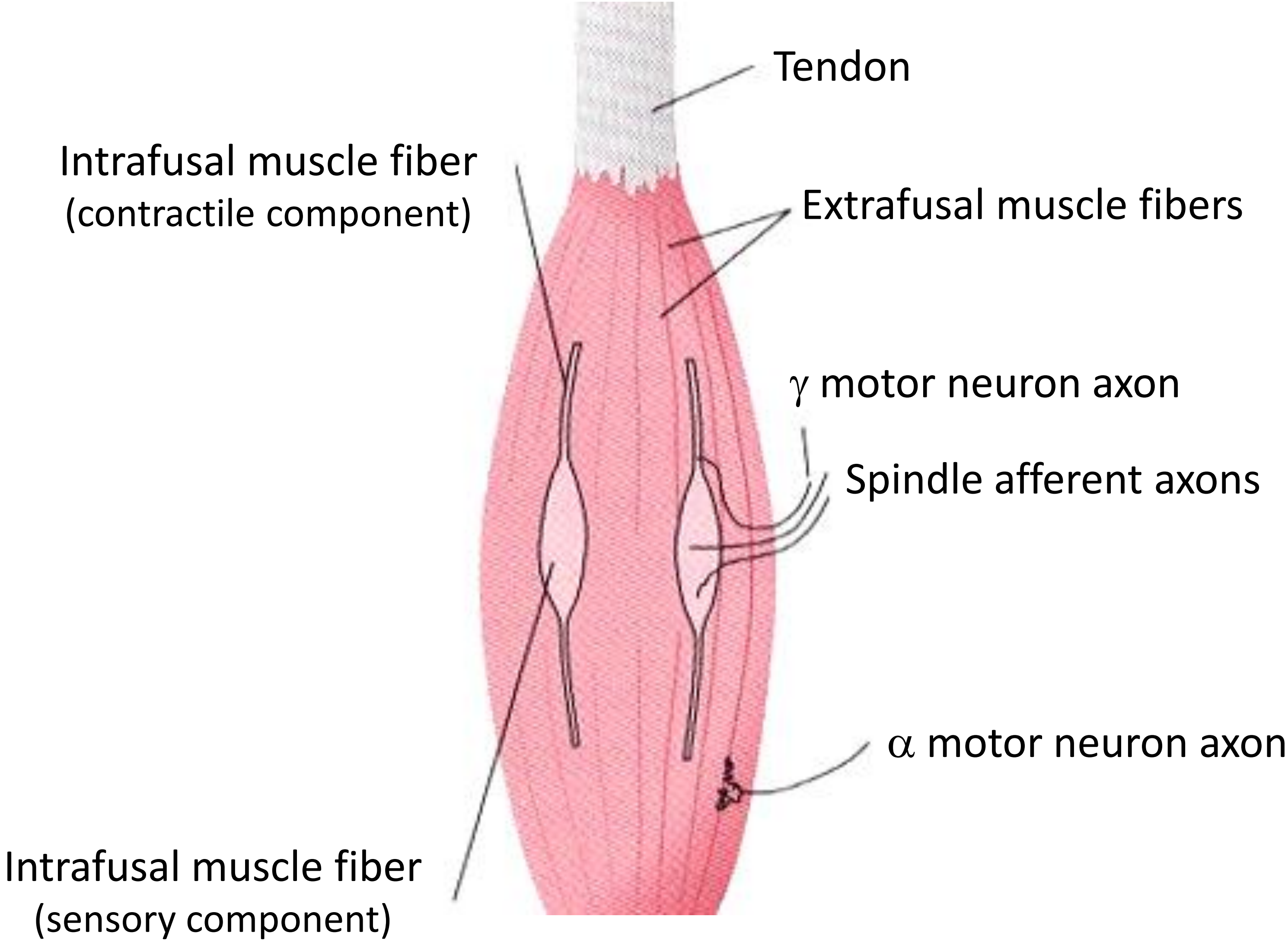
Spinal cord injury involves damage to both neurons and glia

- Initial damage is likely limited to a small region
- Hemorrhaging from broken vessels swells the cord, putting pressure on healthy neurons
- Injured neurons release glutamate at very high levels, over exciting neighboring neurons
- Cyst and glutamate kill myelin producing cells
- After a few weeks, a wall of glia cells forms

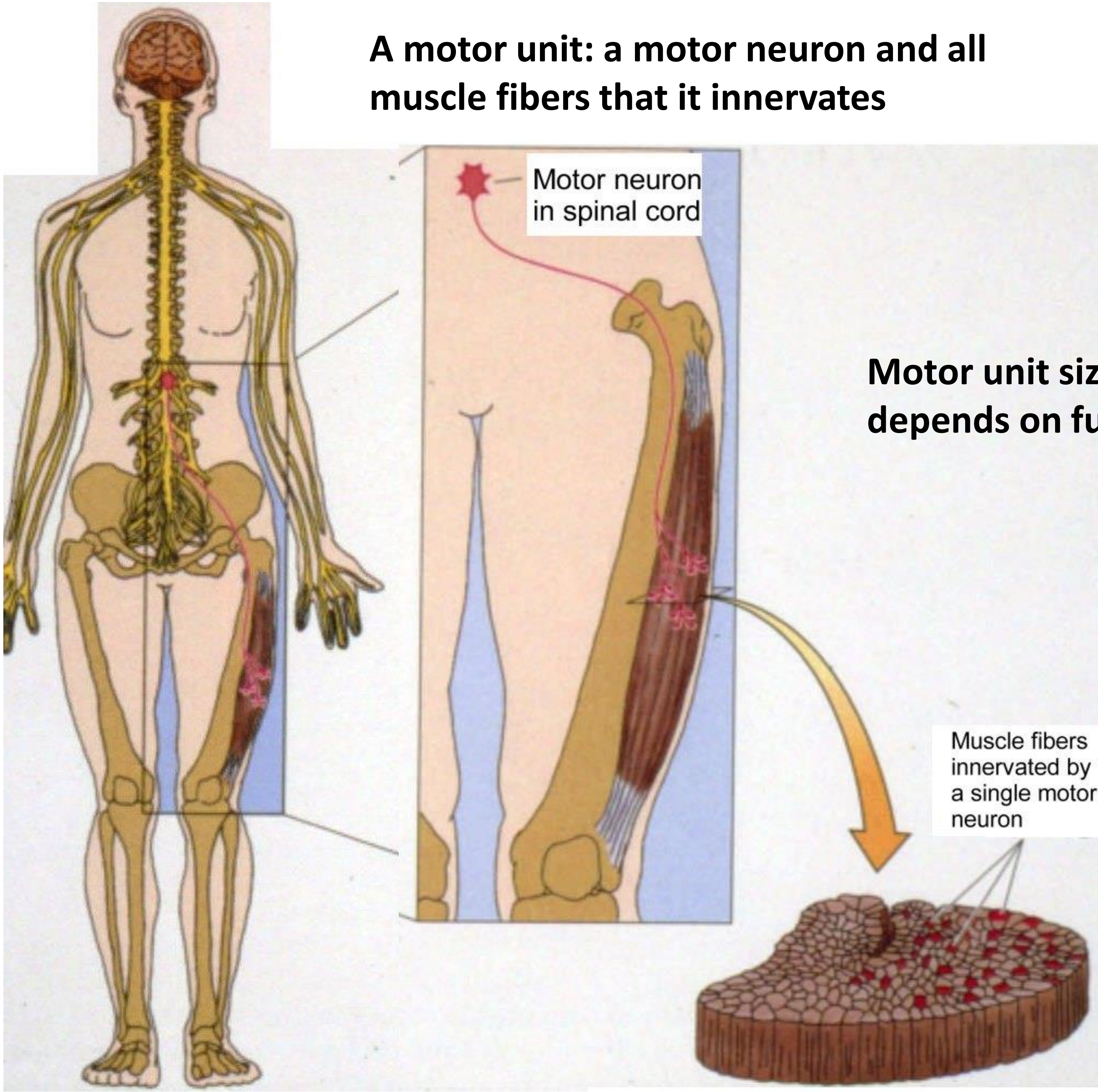


McDonald JW (1999) Sci Am 9:64-73

Muscle Components

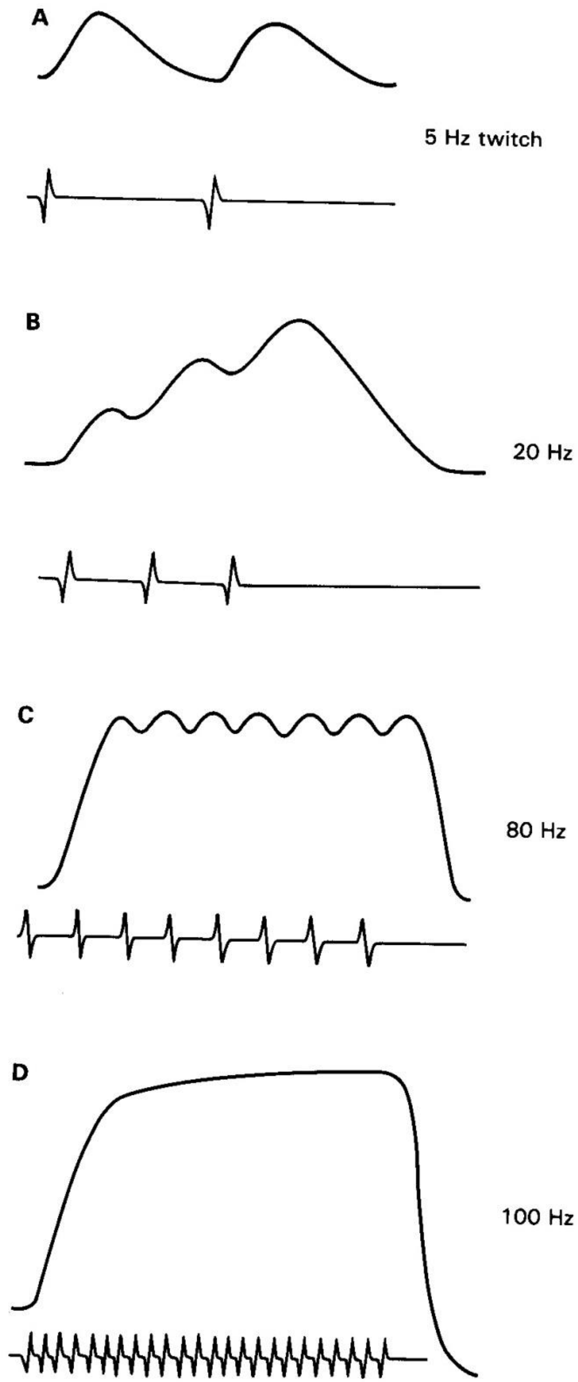


A motor unit: a motor neuron and all muscle fibers that it innervates



Motor unit size depends on function

Force produced by a muscle depends on the **rate** of action potentials from the motor nerve.



Kandel ER et al. (1991)

Types of Skeletal Muscle Fibers

In adult humans, we find that a muscle may be made up of 3 distinct kinds of muscle fibers, where each fiber has a particular isoform of the myosin molecule.

- **Type I**: slow contracting fibers. Repeated stimulation results in little or no fatigue (loss of force).
- **Type II**: fast contracting fibers
    - Type IIa: fatigue resistant
    - Type IIx: easily fatigued

Composition of fiber types in a muscle depends on its function.

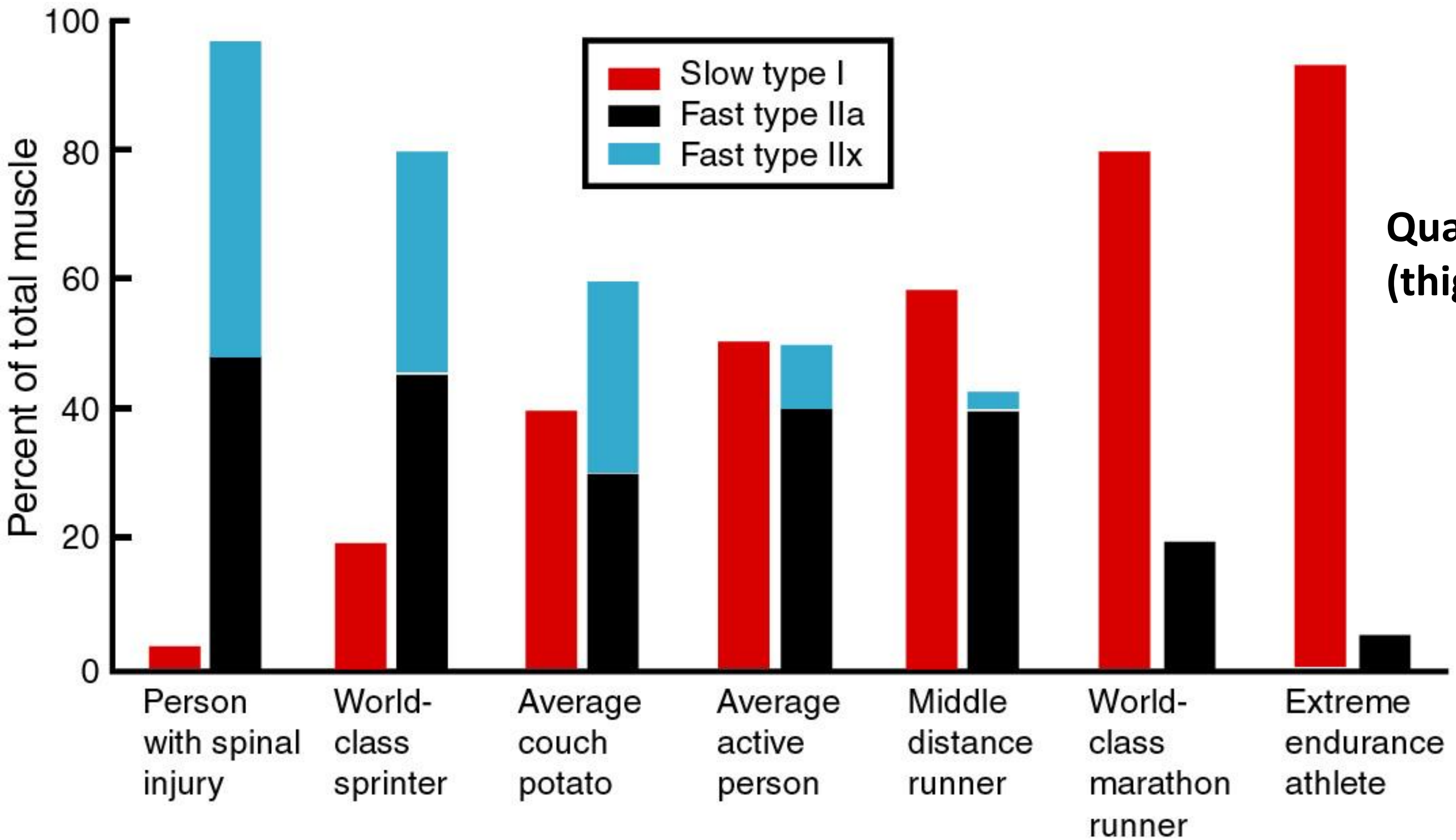
Properties of Motor Units

	Slow Type	FF Type
Axon Diameter:	Small	Large
Tetanic Tension:	Small	Large:
Speed of Contraction:	Slow	Fast
Fatigue:	Little	Rapid
No. of terminals:	Few	Many
Metabolism:	Aerobic	Anaerobic
Myoglobin:	Plentiful	Few
Glycogen:	Little	Many
Mitochondria:	High density	Low density
Capillaries:	Rich supply	Few
Muscle Fibers:	Small, red	Large, pale

Change in a Muscle: Spinal Cord Injury & Effect of Exercise

Strength training puts stress on tendons, signaling proteins to activate genes that make more myosin, resulting in the enlargement of muscle fiber. Type IIx fibers are slowly transformed into type IIa fibers.

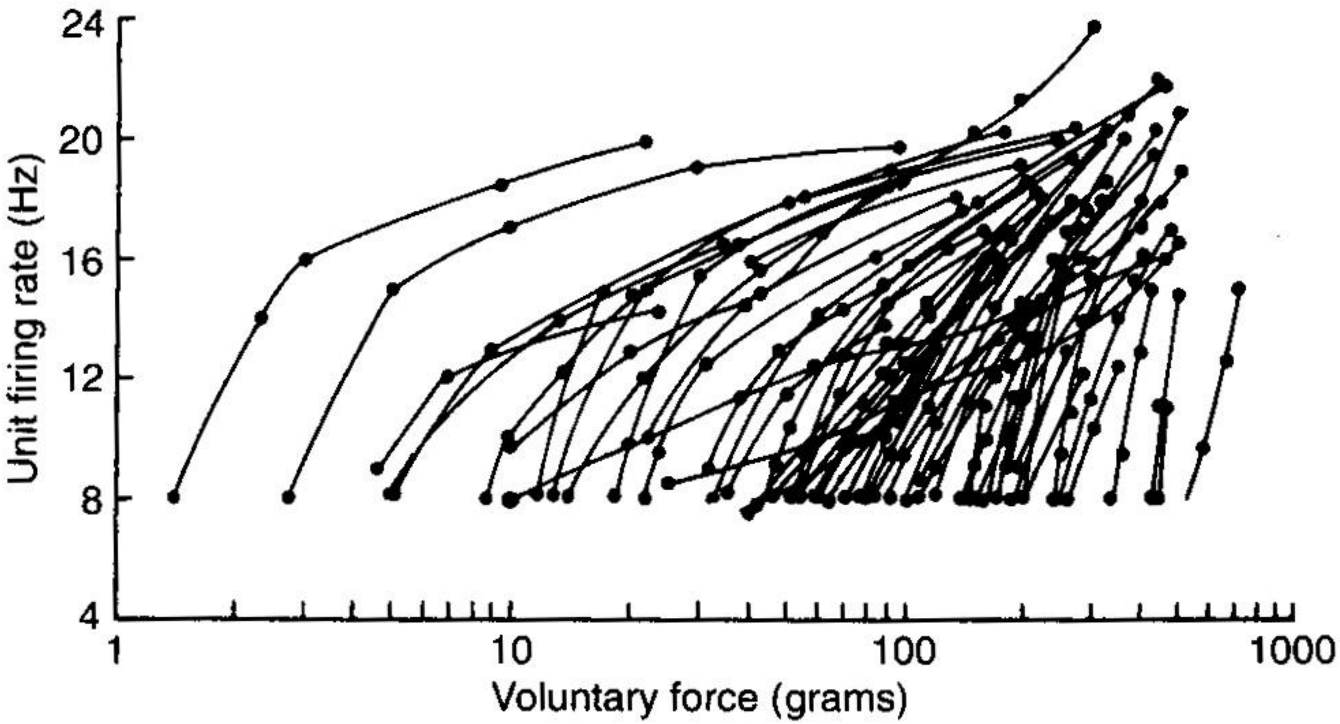
Paralysis: Transformation of type I fibers into type IIx.



Quadriceps (thigh muscle)

Control of Muscle Force

- As more force is needed, more motor neurons are recruited.
- Frequency of activation of motor neurons is increased.

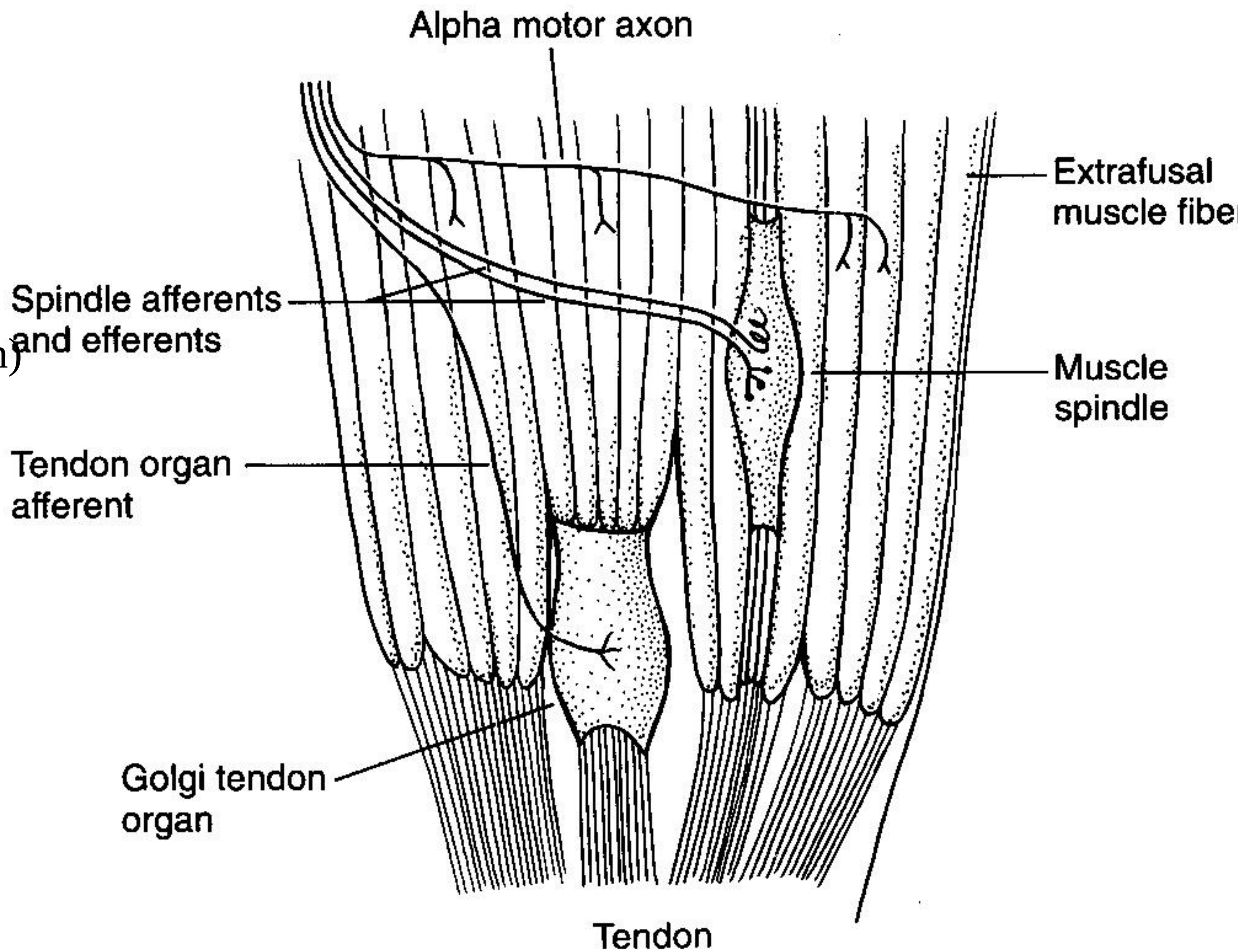


Monster AW & Chan H (1977) J Neurophysiol 40:1432

Muscle's sensory system allows the CNS to measure force and length of the extrafusal muscle

(Ia and II afferents)
(Gamma motor neuron)

(Ib afferents)

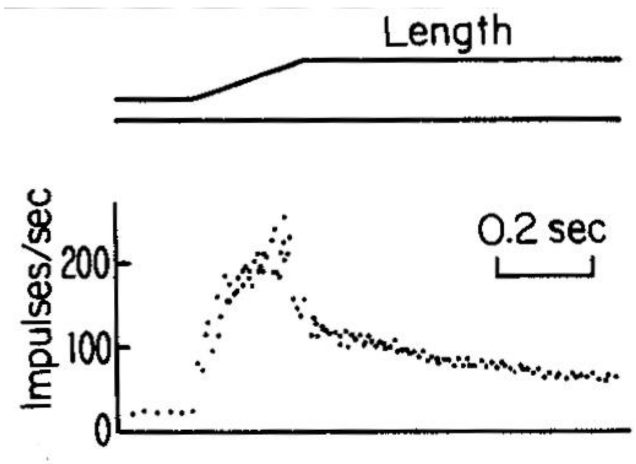


Houk et al. (1980)

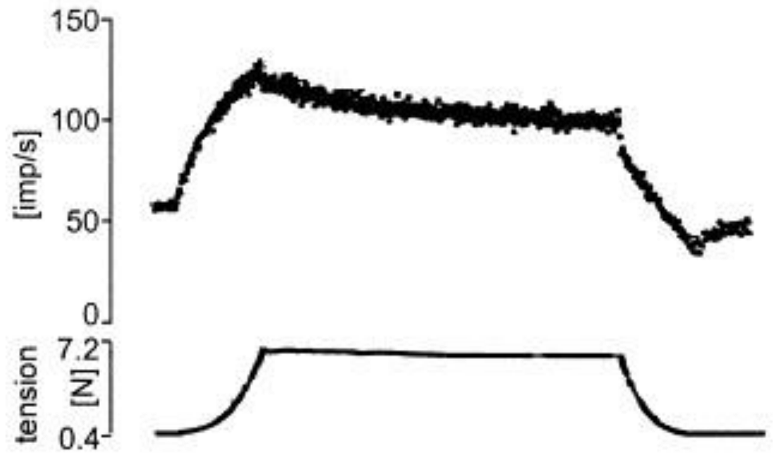
Spindle afferents signal length change in the muscle

Golgi tendon afferent signal force change in the muscle

Response of a muscle spindle afferent to an isotonic stretch

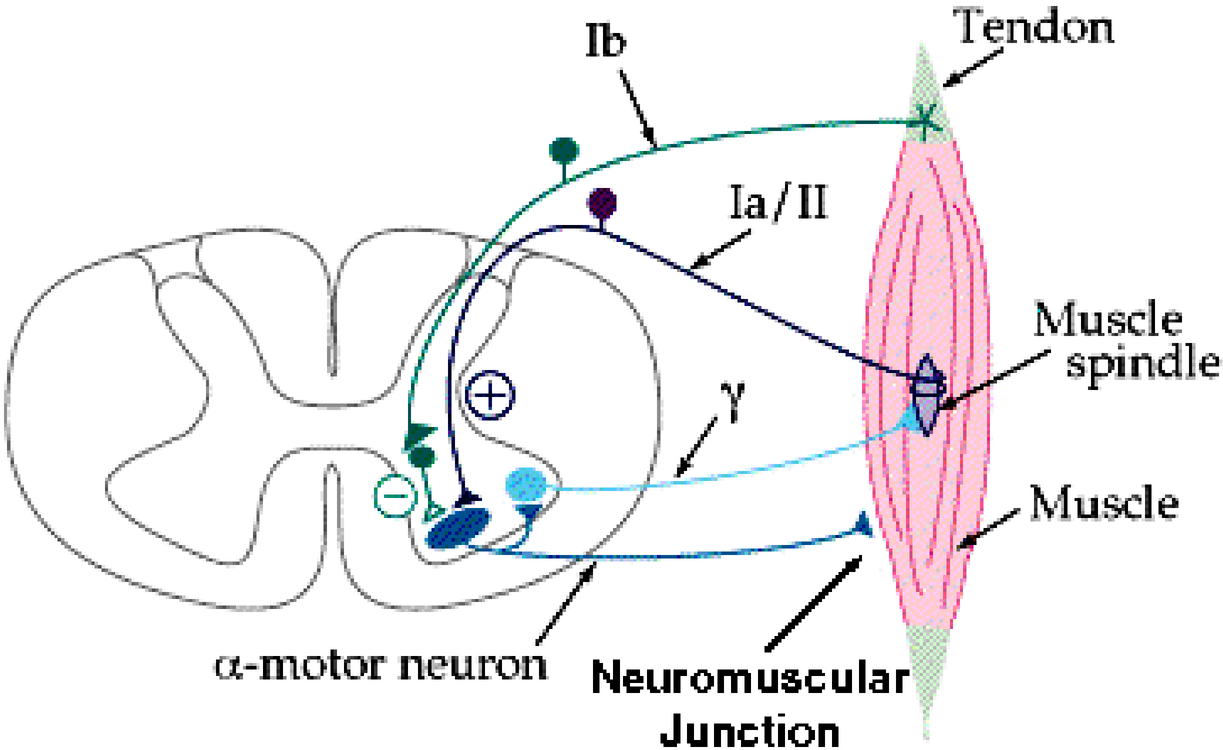


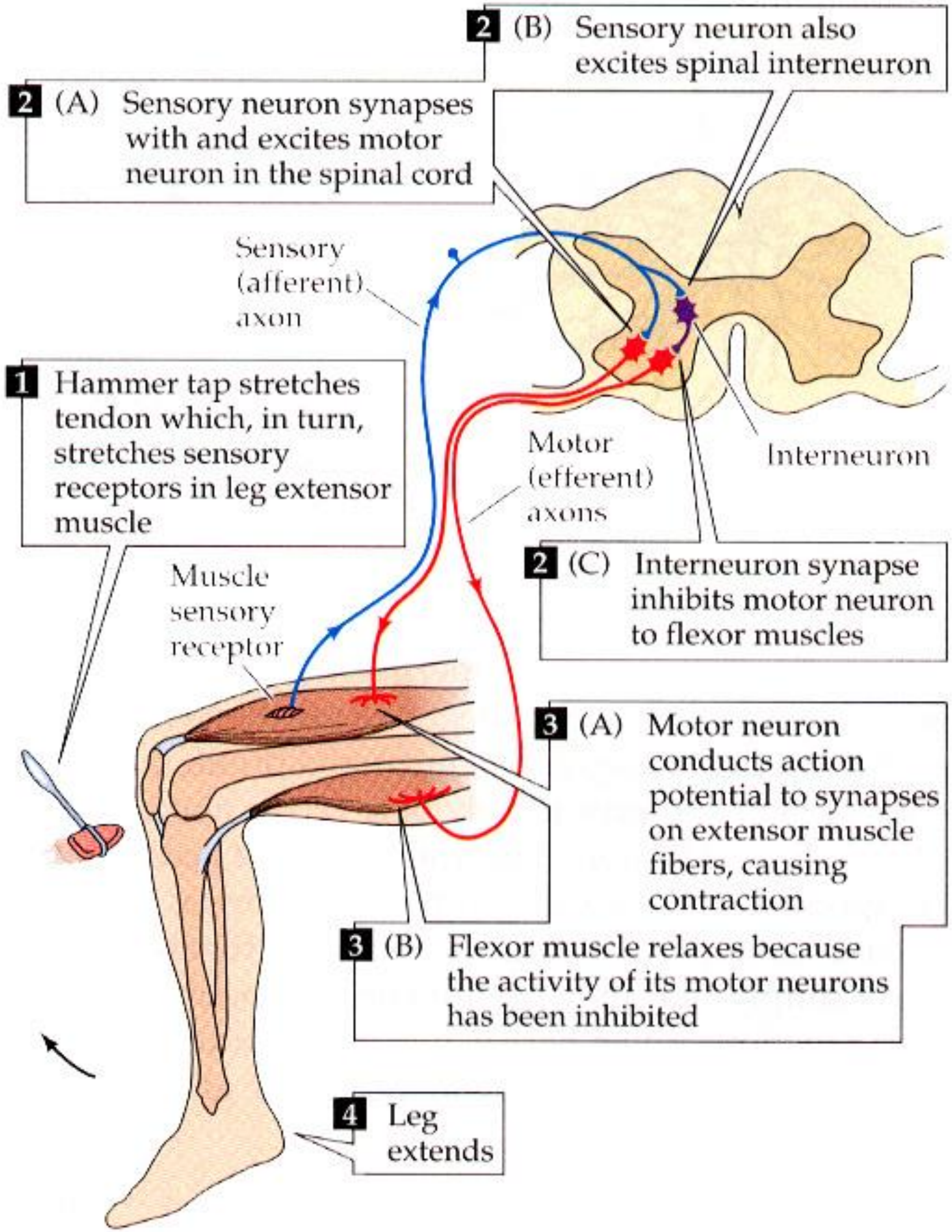
Response of a Golgi tendon afferent to an isometric increase in force



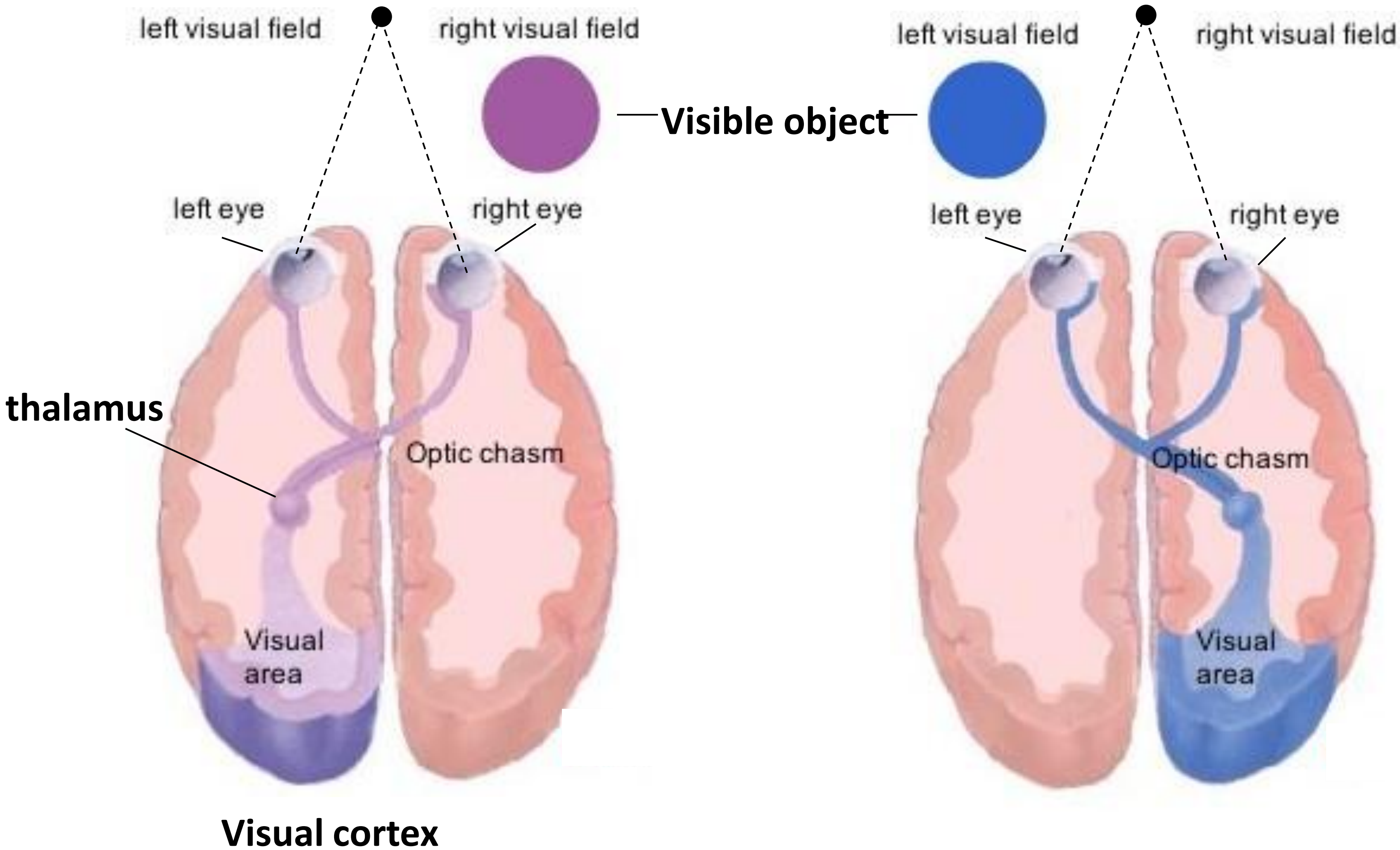
Spindle afferents excite α-motor neurons of the same muscle

Golgi tendon afferents inhibit (via inter-neurons) α-motor neurons of the same muscle




2 (B) Sensory neuron also excites spinal interneuron
2 (A) Sensory neuron synapses with and excites motor neuron in the spinal cord
Sensory (afferent) axon
1 Hammer tap stretches tendon which, in turn, stretches sensory receptors in leg extensor muscle
Motor (efferent) axons
Interneuron
2 (C) Interneuron synapse inhibits motor neuron to flexor muscles
Muscle sensory receptor
3 (A) Motor neuron conducts action potential to synapses on extensor muscle fibers, causing contraction
3 (B) Flexor muscle relaxes because the activity of its motor neurons has been inhibited
4 Leg extends

Objects to the right of fixation fall on the left hemi-retina, and are processed by the left visual cortex.



Left hemisphere has strong control over the contralateral arm and hand

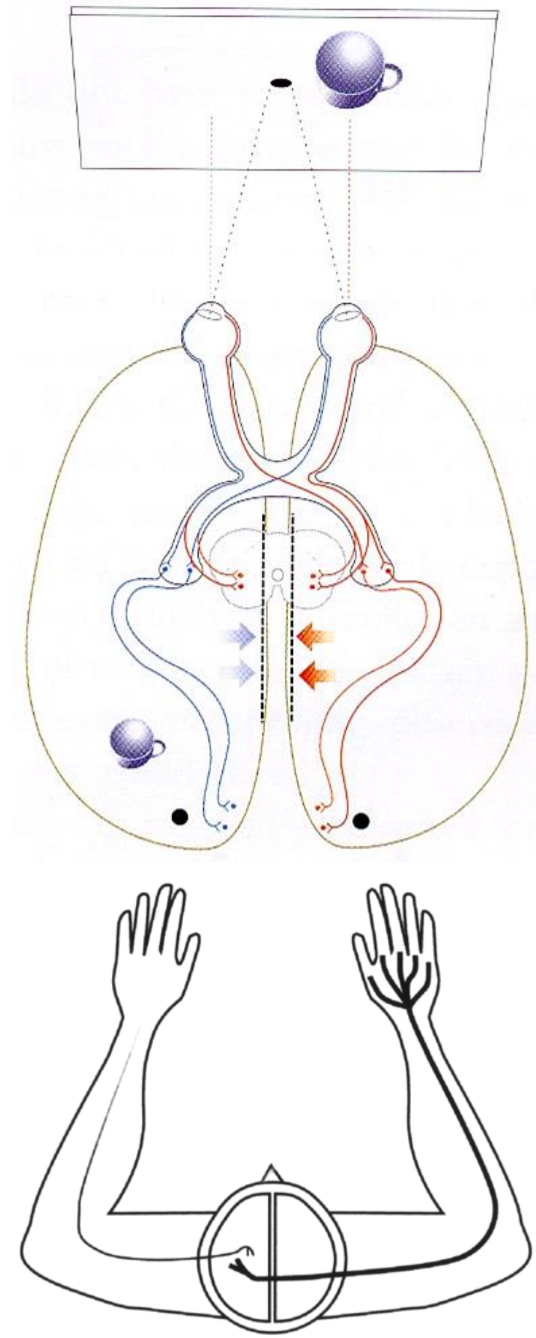
Left hemisphere some control over the ipsilateral proximal arm muscle

Left hemisphere very little control over the ipsilateral hand muscle

Split brain patient studies:

1. Left hemisphere has good control over the left proximal arm muscles: Subject is shown a cup in the right visual field. Information arrives in the left hemisphere. She is asked what she sees, and she answers "a cup". She is asked to reach with the left arm towards the cup. She can reach with left arm normally.

2. Left hemisphere has very poor control over the left finger muscles: Subject is shown a hand posture in the right visual field and asked to copy it with the left hand. She cannot do so. Correct responses are seen for only the very basic gestures like making a fist.

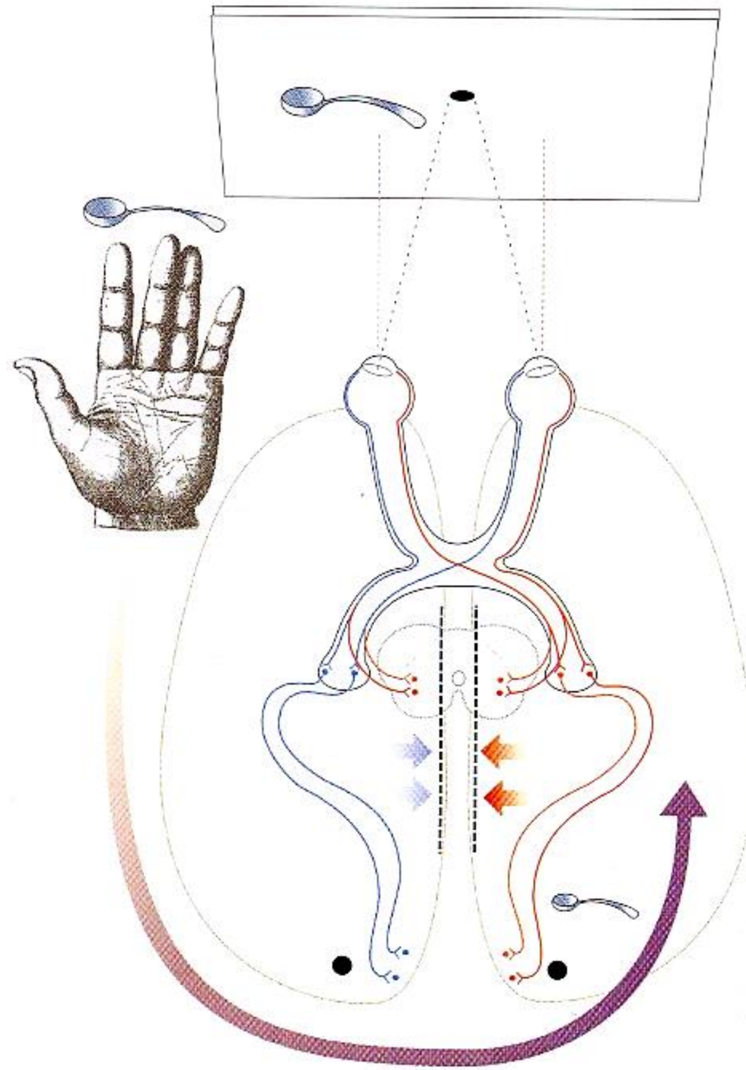


Gazzaniga (2000) Brain 123:1293.

The two hemispheres: language center is usually in the left hemisphere

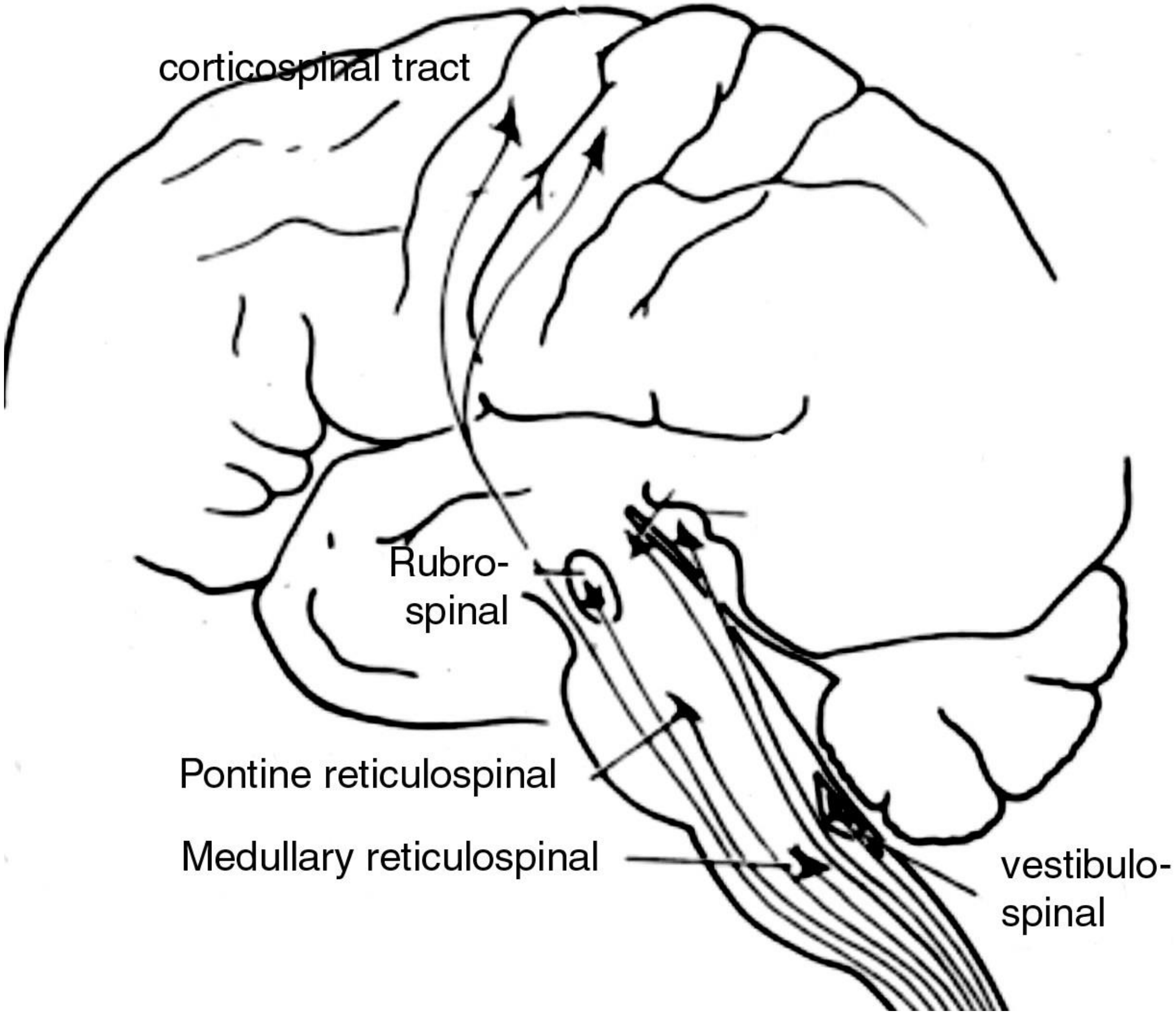
Now a picture of a spoon is shown to the left of the dot. The picture goes to the right hemisphere. She is asked what she saw, and she says “nothing”. She says this because in nearly everyone, the language centers are in the left hemisphere. Because the left hemisphere has not been given the visual information, it says that it has seen nothing.

However, when N.G. is asked to reach under a table with her left hand and select, by touch only, from among a group of concealed items the one that was the same as the one she had just seen, she picks a spoon. While she is holding the spoon under the table, she is asked what she is holding, she says “a pencil”. (R.W. Sperry 1968, American Psychologist 23:723-733)



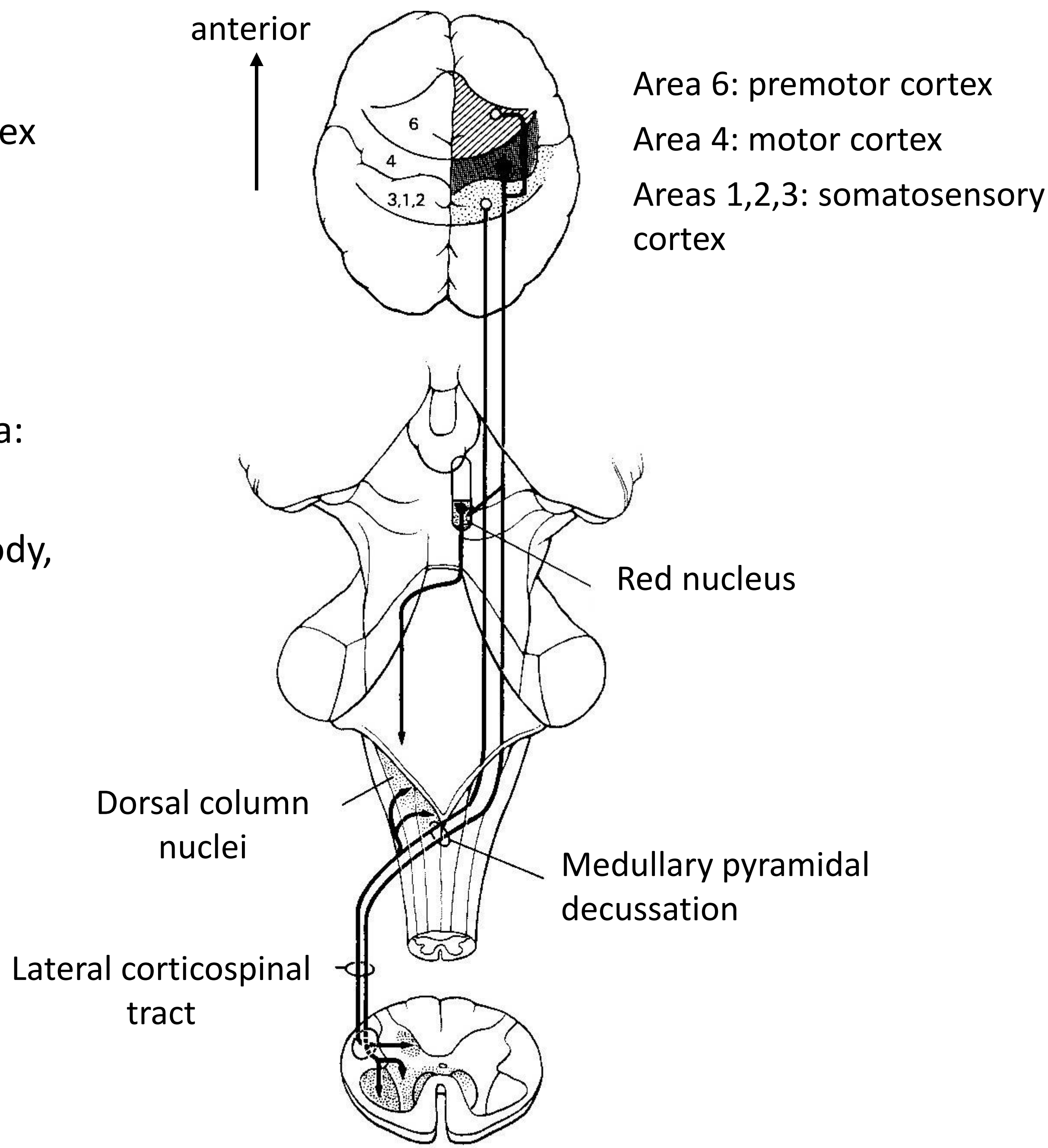
R. Carter (1998) Mapping the Mind

Descending tracts: send information from the brain to the spinal cord.



Corticospinal tract

- Origin: primary motor cortex (30%), Premotor (30%), somatosensory (30%)
- About 1 million fibers in humans.
- 90% cross at lower medulla: Right motor cortical areas control the left side of the body, specially distal muscles.
- 10% do not cross
- All are excitatory
- Small diameter, slow conducting fibers



Kandel ER et al. (1991)

Reticulospinal tracts

Large fiber axons.

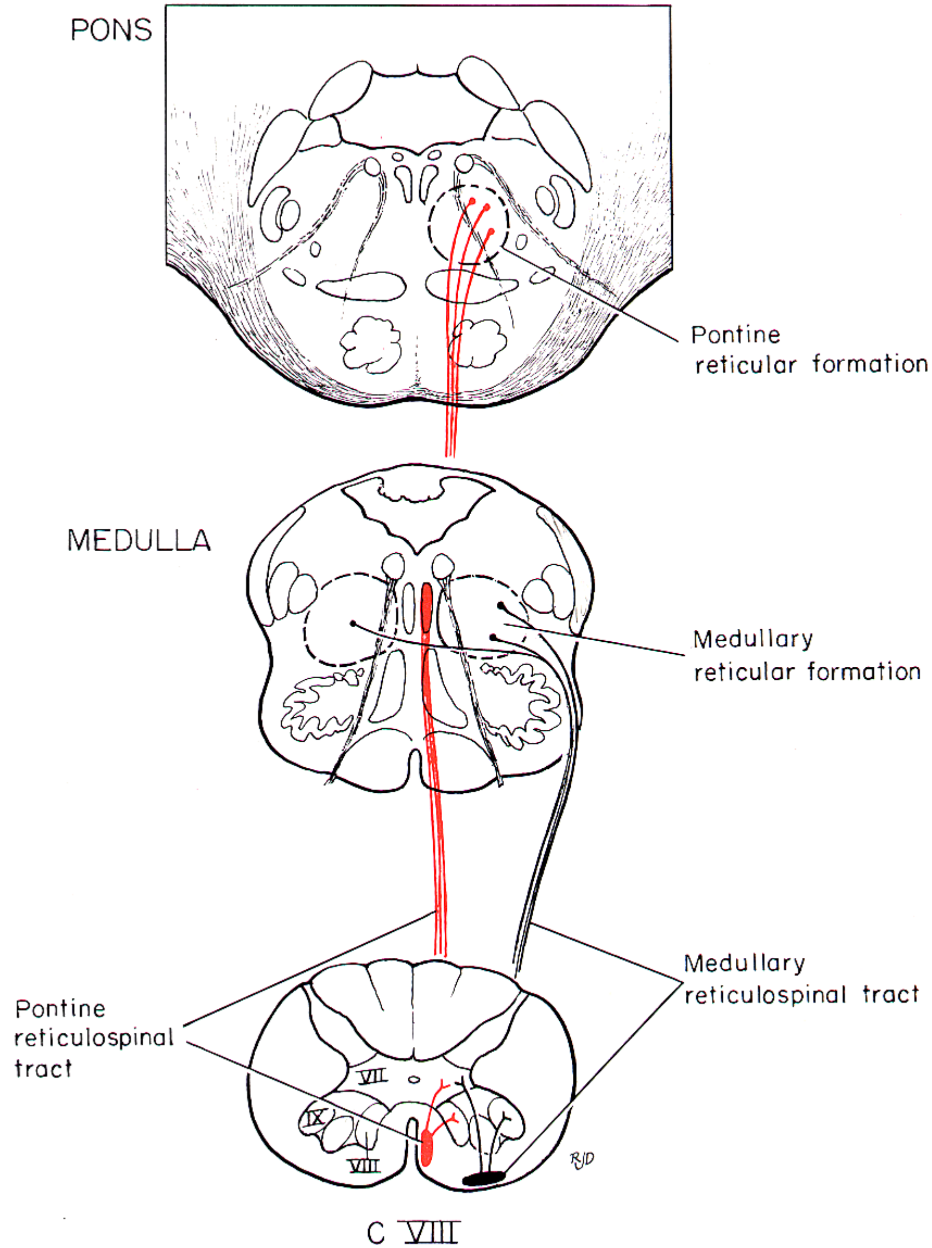
Control of posture and balance, acting on anti-gravity muscles.

Pontine reticulospinal tract

Excitatory synapses on leg extensors and arm flexors.

Medullary reticulospinal tract

Inhibitory synapses. Action is to reduce muscle tone for nearly all muscles of the upper and lower limbs.



Carpenter MB (1985)

Summary

Visual objects to the right of fixation are processed predominately by the left visual cortex. However, because of the corpus callosum, this information is shared with the contralateral cerebral hemisphere.

The corticospinal tract brings the output of the premotor cortex, primary motor cortex, and the somatosensory cortex. The corticospinal tract in the left brain controls the right arm, and the tract in the right brain controls the left arm.

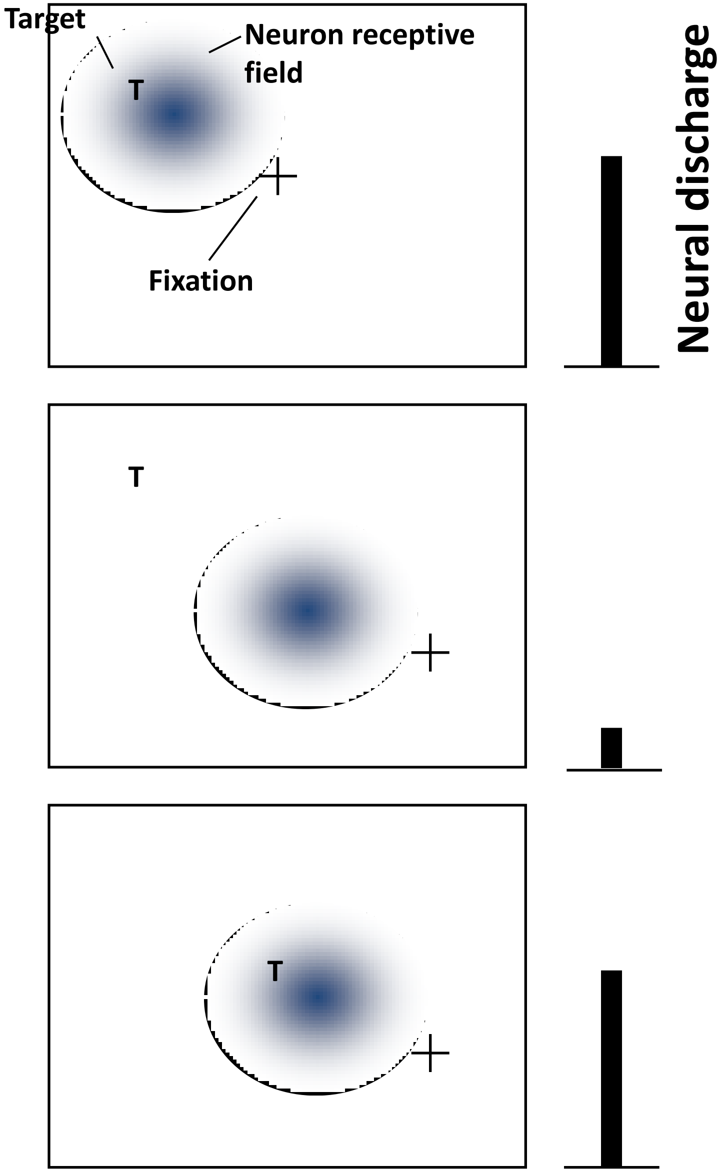
The function of the corticospinal tract is to control limb movements, particularly movements of the fingers.

In the brainstem we have two important motor centers: pontine reticular nucleus and medullary reticular nucleus. These centers sent their output to the spinal cord via the pontine and medullary reticulospinal tracts.

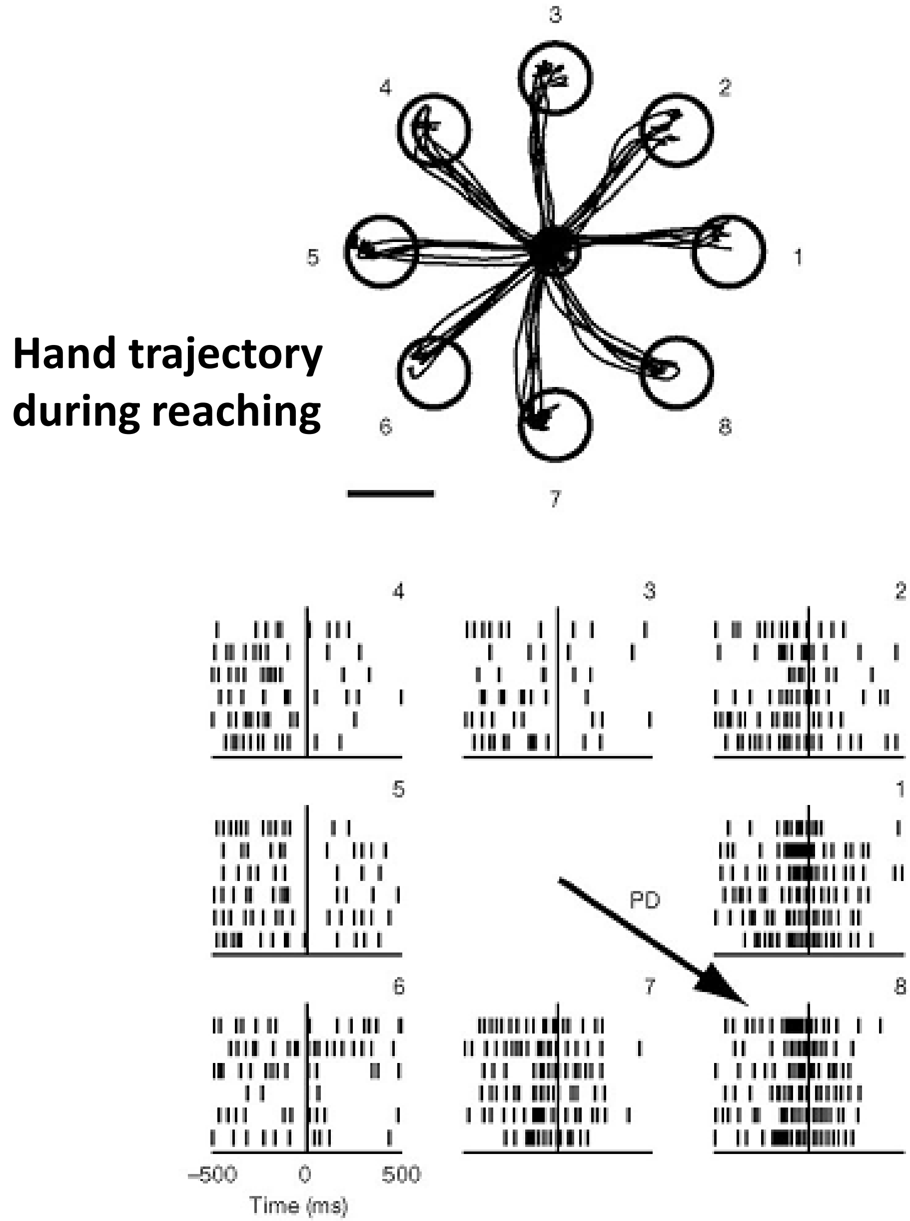
The function of the pontine center is to maintain our balance and posture.

The function of the medullary center is to inhibit muscles, particularly during rest and sleep.

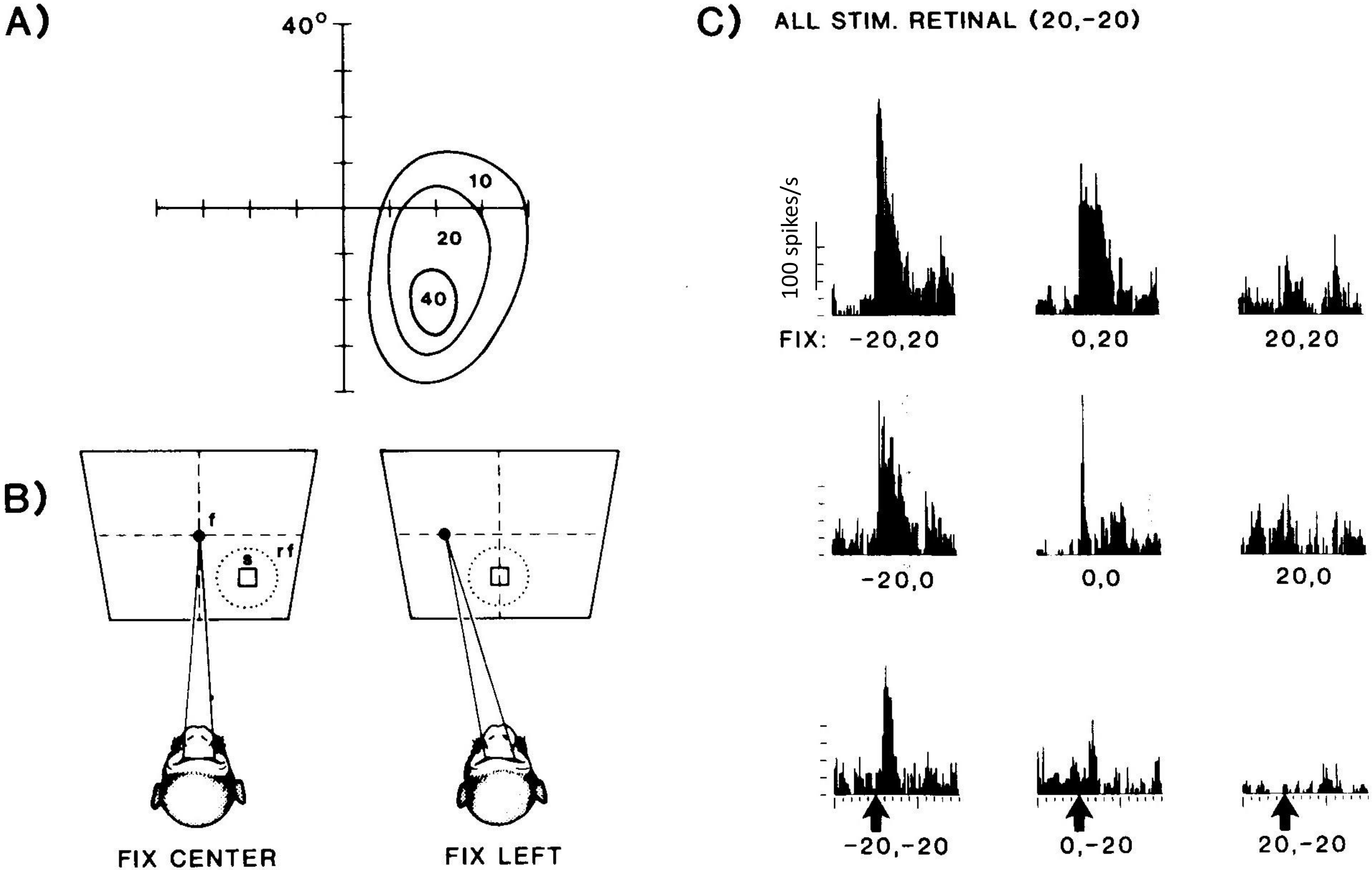
Cells in the visual cortex have a "fixation-centered" receptive field



Cells in the motor cortex encode direction of limb movement and forces

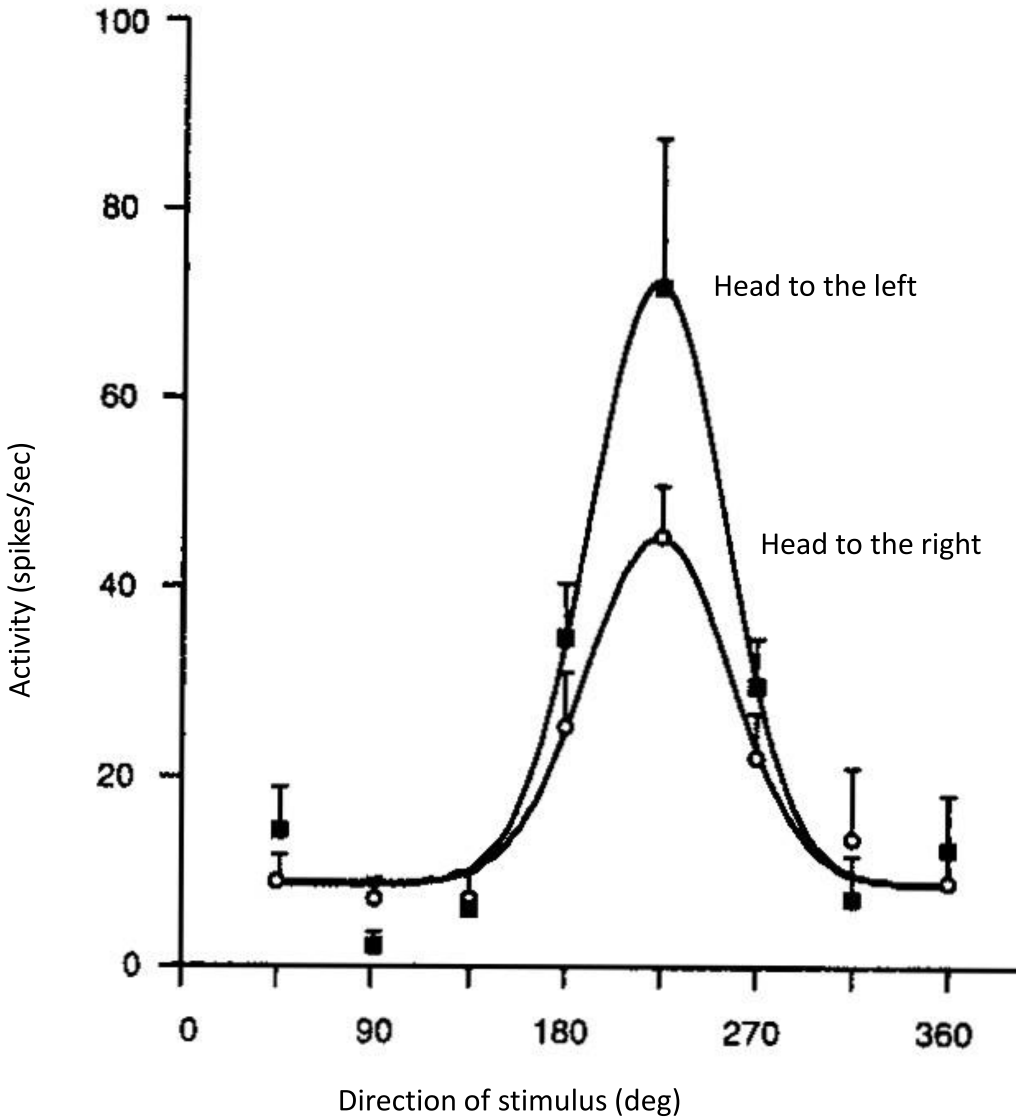


Neurons in the PPC encode both image location and eye position



Andersen RA et al. (1985) Science

PPC neurons are also sensitive to head position



Brotchie et al. Science 1995

Summary

Target location and hand position are computed by posterior parietal cortex cells in terms of vectors with respect to fixation point. These visual cues are represented with neurons that have receptive fields.

Proprioceptive information from the arm, head, and eyes are used to estimate hand position with respect to fixation.

Proprioceptive information from the head and eyes are combined with information about retinal location of the target to estimate target position with respect to fixation.

Posterior parietal cortex neurons combine visual and proprioceptive information as a gain field. In a gain field, neuronal response has a receptive field (usually a Gaussian) that is multiplicatively affected by a linear function that encodes proprioceptive information about location of the eyes or head.