Parietal Lobes: Evolution, Neuroanatomy, and Function

Charles J. Vella, PhD

May 3, 2017

Intent of this lecture

- Parietal lobe syndromes are mostly domain of neurology, not NP
- But these syndromes will affect NP performance on multiple tests
- There are lots of anatomical references: you can always use the pdf online as reference text
- There are lots of unusual, sometimes rare, syndromes
- My objective is to have you be aware of when parietal lobe functioning is involved in something you are seeing clinically in a patient

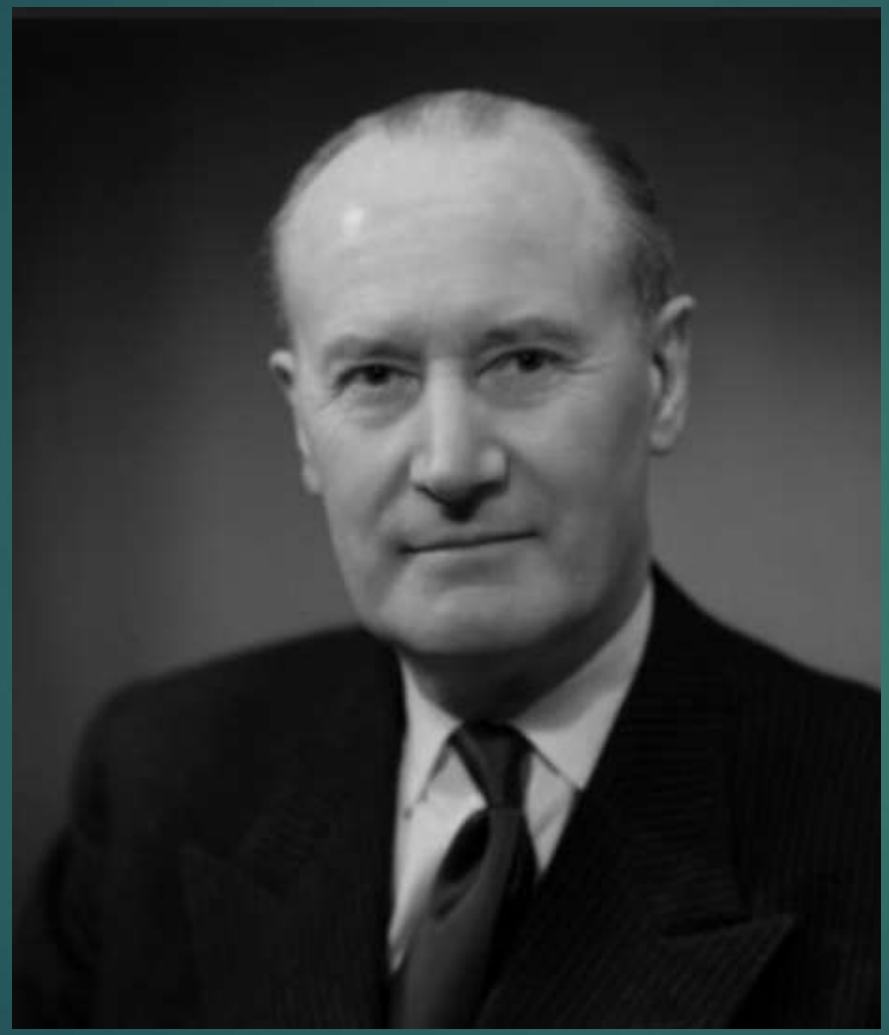
The discovery of the Parietal Lobe

- Franciscus Sylvius (1614-1672)
- Physician, physiologist, anatomist, and chemist
- Professor at the University of Leidon (1641)
- Was in the chemistry lab at the University when he discovered the deep cleft (Sylvian Fissure).

History of Parietal Lobe Discovery

- In 1874 Bartholow recorded odd sensation from legs on stimulating post central gyrus through skull wounds
- Djerine – alexia , agraphia -- angular gyrus lesion
- Hugo Liepmann--- ideomotor & ideational apraxia in (L) sided lesion
- Cushing in 1909 --- Electrical stimulation in conscious human beings — mainly tactile hallucinations; first homunculus
- Critchley (1953) – 1st monograph on " The Parietal Lobes"
- Siegel et al. (2003) – "The Parietal Lobes"

Macdonald Critchley: Parietal Lobes



Parietal Lobes

- No independent existence as anatomical / physiological unit
- Operates in conjunction with brain as a whole
- Strategically situated between other lobes
- Greater variety of clinical manifestations than rest of the hemisphere
- Ignored in Neuropsychology: dysfunction likely to be overlooked unless special testing techniques are used.

Functions of Parietal lobe

- Intermodal sensory/perceptual integration: Auditory, Vision, Touch
- Sensorimotor integration and visually guided movement: body in motion in space
- Interface of perception and action
- Virtual reality: brain-generated simulation of a three-dimensional image of environment
- Perceptual Imagination
- Higher Order Cognition: central to IQ

The Parietal Lobes – MacDonald Critchley, 1953

- His chapters regarding PC dysfunctions:
  - Disorders of Tactile Function
    - Epileptic sensations, tactile hallucinations, cortical sensory loss, hemi-anesthesia, postural loss, astereognosis, amorphognosis, tactile asymboly, graphesthesia, weights discrimination, inability to localize stimuli, tactile discrimination & inattention, asymboly for pain, Pseudothalamic syndrome,
  - Disorders of Motility
    - Apraxia
  - Constructional Apraxia
  - Gerstmann's Syndrome
  - Disorders of the Body-Image
    - Unilateral neglect, anosognosia (for hemiplegia)

The Parietal Lobes – MacDonald Critchley, 1953

- Visual Defects
  - Cortical blindness, hemianopia, color agnosia, achromatopsia, visual agnosia, prosopagnosia, metamorphopsia, dyslexia, visual field defects
- Disorders of Spatial Thought
  - Spatial neglect
- Disorders of Language and Symbolic Thought
  - Dyslexia, acalculia, dysgraphia

No other area of the brain surpasses the parietal lobes in the rich diversity of clinical dysfunctional phenomena

Acalculia: Inability to calculate

- **Agraphaesthesia**: Inability to identify number drawn on hand opposite to parietal lesion side
- **Agraphia**: Inability to write
- **Alexia**: Inability to read
- **Aphasia**: Loss of speech
- **Apraxia**: Loss of ability to carry out learned skilled movements to command despite normal sensory and motor function
- **Astereognosis**: Inability to identify shape and nature of object by touch alone (eyes closed)
- **Finger agnosia**: Inability to recognize and name fingers despite the retention of sensation
- And many more

Henry Head & Brain Maps

- About a hundred years ago, the great neurologist Henry Head suggested that the brain contains maps of the body, and that these maps – which he referred to as 'schemata' – can expand to incorporate clothes, tools and other objects.

- "Anything which participates in the conscious movement of our bodies," he wrote in a classic 1911 paper, "is added to the model of ourselves and becomes part of these schemata: a woman's [schemata] may extend to the feather in her hat."

Parietal Lobes: Peripersonal & Extrapersonal Space

- Peripersonal space: Parietal lobe produces a map of your body and of the space around it

- Spatial body map: This map incorporates anything you use or are in or on, i.e. car, bike, horse, tool use, baseball bat, etc.

- Body map: Tactile map of every inch of your body's surface mapped to opposite side of your brain.

Egocentric vs. Allocentric Frameworks

- In the egocentric reference frame, object locations are processed in reference to the person, and this self-based map is updated as a person moves through the environment; Allocentric frame: independent of person map
  - Updates spatial information on a moment-to-moment basis as we move through the environment

- Parietal cortex plays a critical role in the egocentric network and allocentric network (Burgess, 2008; Iaria et al., 2003)
- Egocentric frame of reference: dorsal caudate nucleus, posterior parietal cortex, precuneus, lateral frontal cortex (Iaria, et al., 2003)

(Ball, et al., 2009)

Somatic Senses processed by Parietal Lobes

- Receptors:
  - Touch (gentle, deep, sustained pressure, hair bend, vibration)
  - Thermoception (hot and cold, organ heat)
  - Nocioception (piercing, heat, chemical pain; joint, deep tissue pain; itch, tickle)
  - Proprioception (body position & movement in space: muscle stretch receptors for limb location; cartilage for joint slippage for limb speed)
  - Balance (inner ear)

Evolution of size of multimodal association area

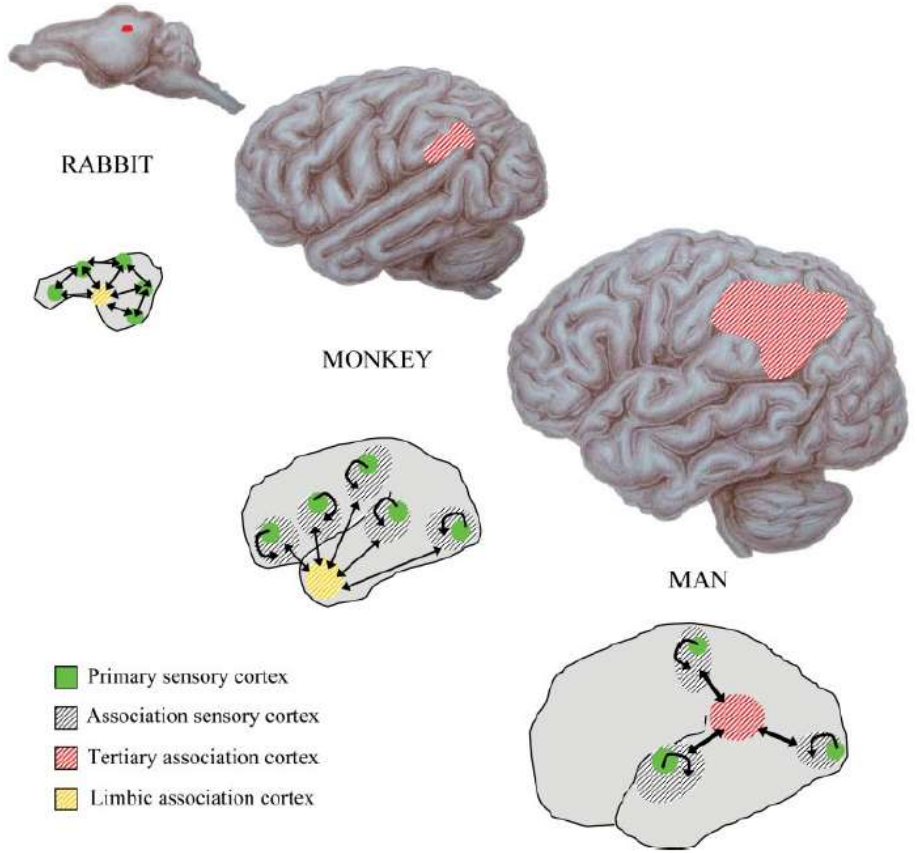


Fig. 4 Geschwind's view of the evolution of cross-modality associations. For simplicity only sensory cortex is illustrated. The top diagonal sequence shows the expansion of inferior parietal cortex from rabbit through monkey to man, considered by Geschwind as central for the development of language. The bottom diagonal sequence shows the differences in brain circuitry between the species. In the rabbit, Flechsig's rule does not apply and the primary cortices of different sensory modalities are connected directly to one another as well as through limbic cortex. In the monkey, primary cortices connect only to their association cortices with intermodality connections mediated by the limbic cortex. In man, the majority of intermodality connections are mediated by higher-order association cortex in the parietal lobe.

In humans, majority of intermodality connections are through the Parietal association area.

Parietal Development

- Superior and inferior parietal lobule and adjacent temporal occipital lobe larger in humans than in primates

- Develops by 6-7 years of age

Parietal bulge/Precuneus enlargement in Homo sapiens

- Parietal bulging is the major morphological variation in Homo sapiens.
- The size of the precuneus is the main determinant of the midsagittal brain geometry.
- Pattern is strictly determined by one single characteristic: the longitudinal extension of the precuneal area.

Evolution: Cost is Alzheimer's

- Our species is characterized by spatial changes at deep parietal areas like the intraparietal sulcus and the precuneus.
- Fossil endocasts: an increase in the parietal vascularization
- No intraparietal sulcus in non-human primates.
- In earliest stages of AD there is a pathological metabolic impairment in the deep parietal areas.
- Is Alzheimer's disease the evolutionary cost of a highly metabolic parietal lobe?

Emiliano Bruner, 2013

Neuroanatomy of Parietal Lobes

Neuroanatomy

- Occupies middle third of cerebral hemispheres
- Situated between frontal ,temporal, occipital lobes with anatomical & functional continuity

Boundaries of Parietal Lobe

- Anterior border - Central Fissure
- Lower/ventral border - Sylvan Fissure/Lateral Sulcus
- Posterior border - Parieto-occipital sulcus

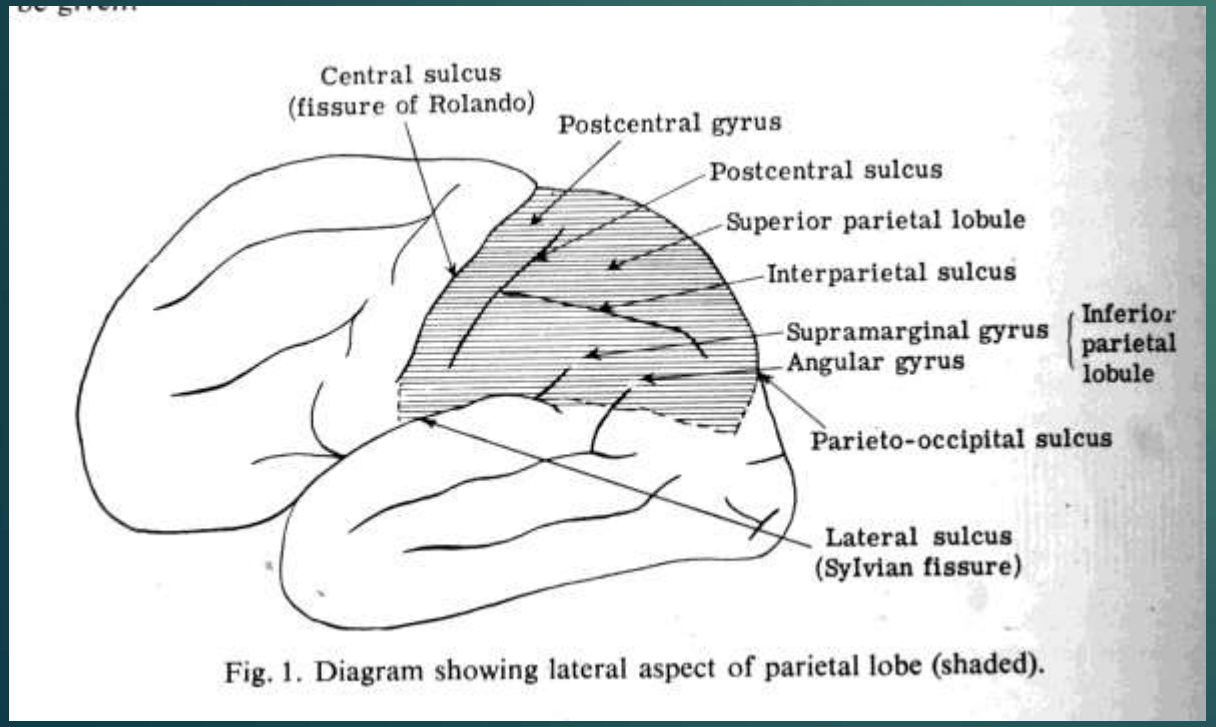


Fig. 1. Diagram showing lateral aspect of parietal lobe (shaded).

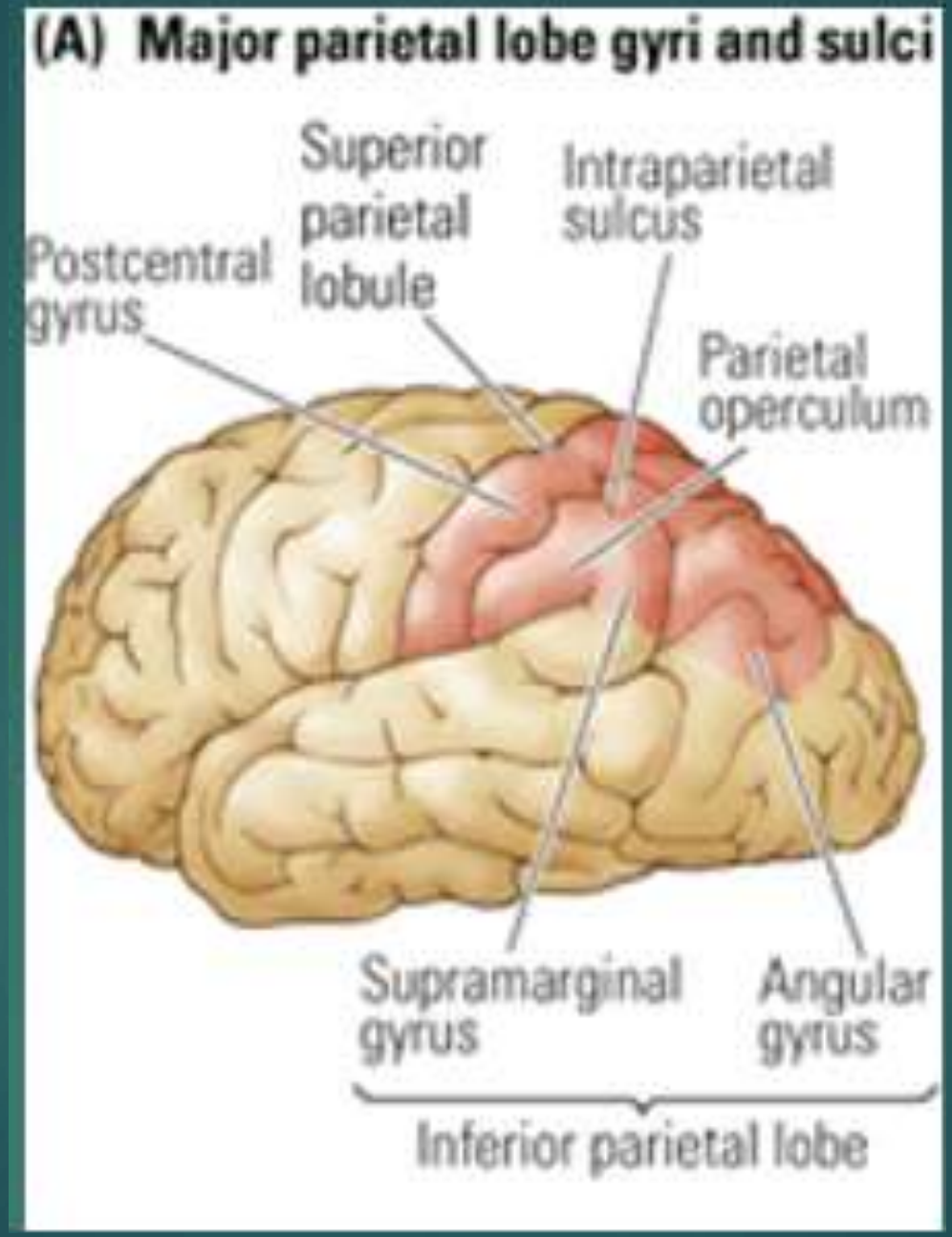
Lateral

Medial

Parietal lobe: Subdivisions

- Anterior part of the parietal lobe:
  - Postcentral gyrus (Brodmann area 3),
  - Primary somatosensory area: represents the skin area on the contralateral surface of the body.
  - Postcentral sulcus divides the above from posterior parietal cortex

- Posterior parietal cortex can be subdivided:
  - Superior parietal lobule (SPL) (Brodmann areas 5 + 7)
  - inferior parietal lobule (IPL) (39 + 40),
  - separated by the intraparietal sulcus (IPS). IPS is essential in the guidance of limb and eye movement


(A) Major parietal lobe gyri and sulci
Superior parietal lobule
Intraparietal sulcus
Postcentral gyrus
Parietal operculum
Supramarginal gyrus
Angular gyrus
Inferior parietal lobe

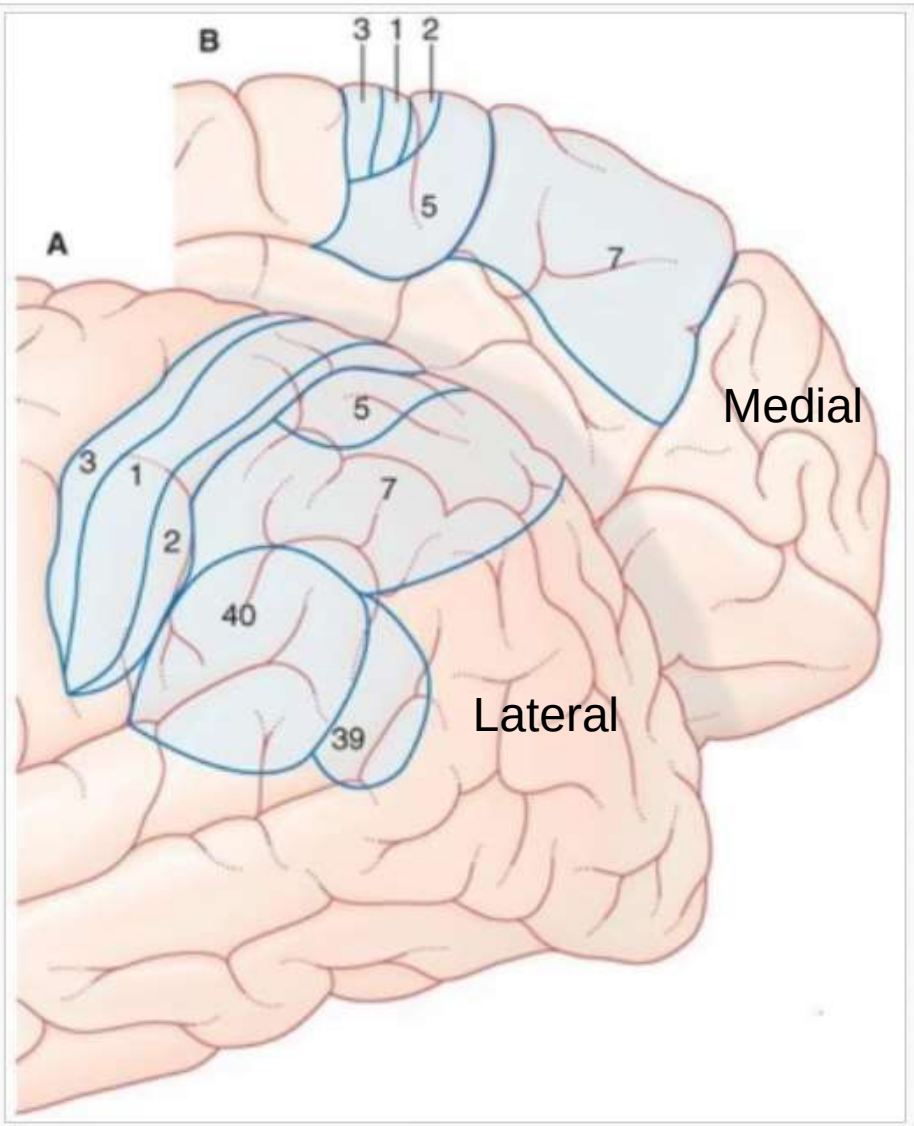


Figure 4: Brodmann areas. A (lateral), B (medial). Somatosensory cortex (Areas 3,1,2); Somatosensory association area (Area 5); Posterior parietal cortex (Area 7); Angular gyrus (Area 39); Supramarginal gyrus (Area 40)[8]

Postcentral sulcus:

3,1,2 - Somatosensory cortex

Superior Sulcus:

5 - Somatosensory Association area

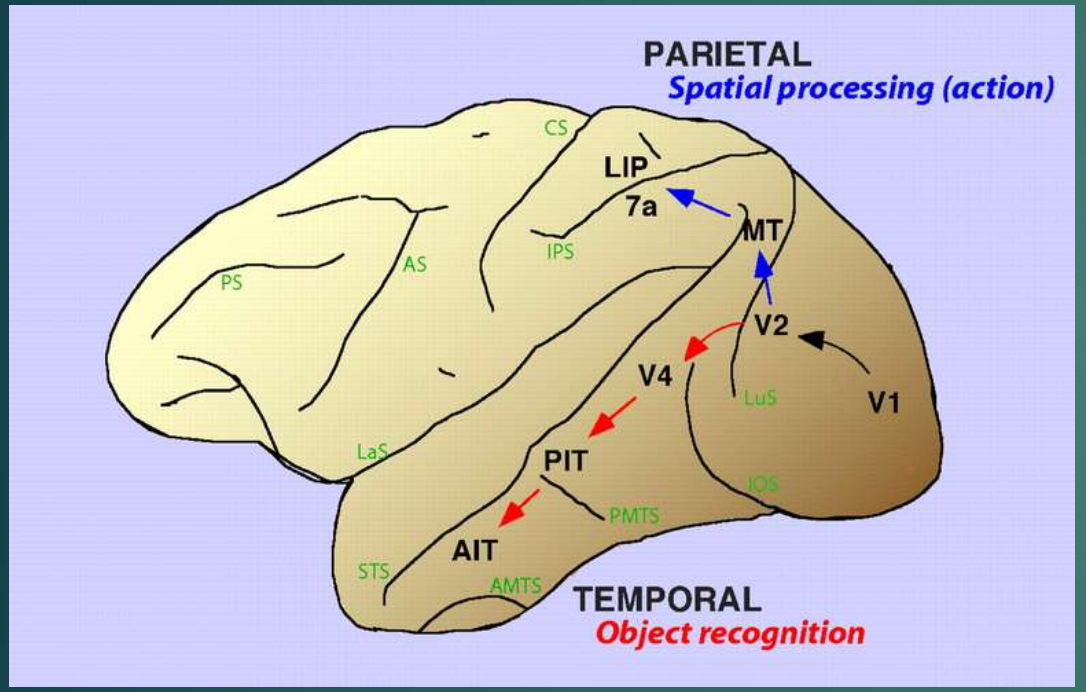
7 - Posterior parietal cortex (incl. dorsal path)

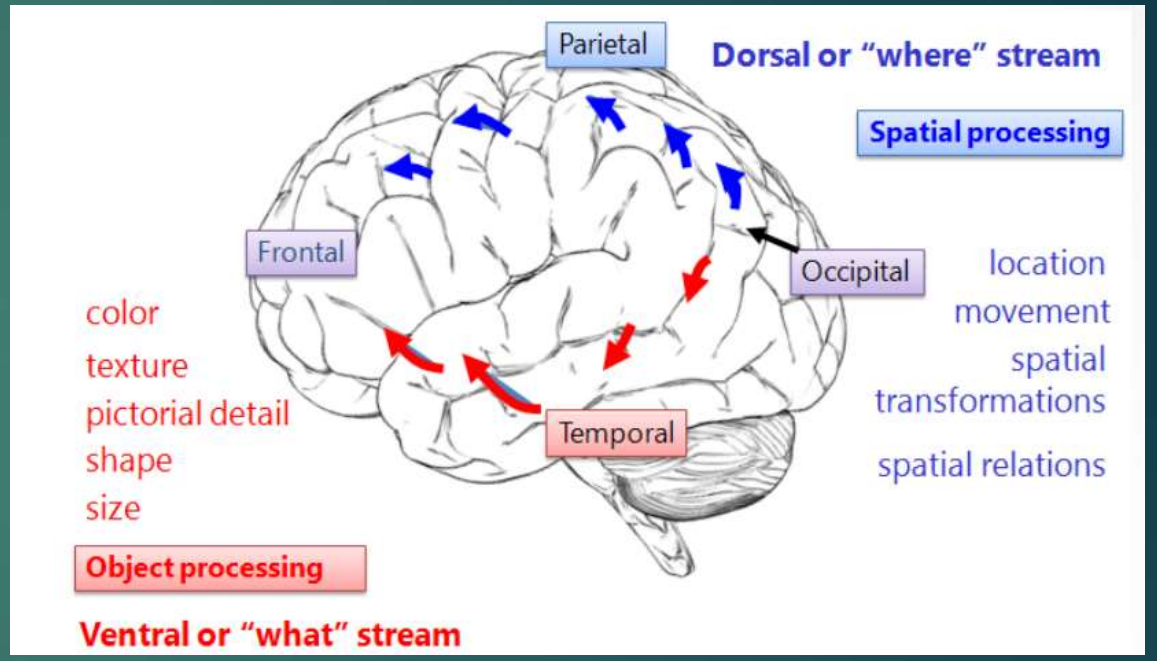
Inferior Parietal Lobule (IPL or ventral PC):

39 - Angular gyrus

40 - Supramarginal gyrus

Dorsal (action, spatial, where) pathway in Lateral Intraparietal Sulcus of superior Parietal Lobe



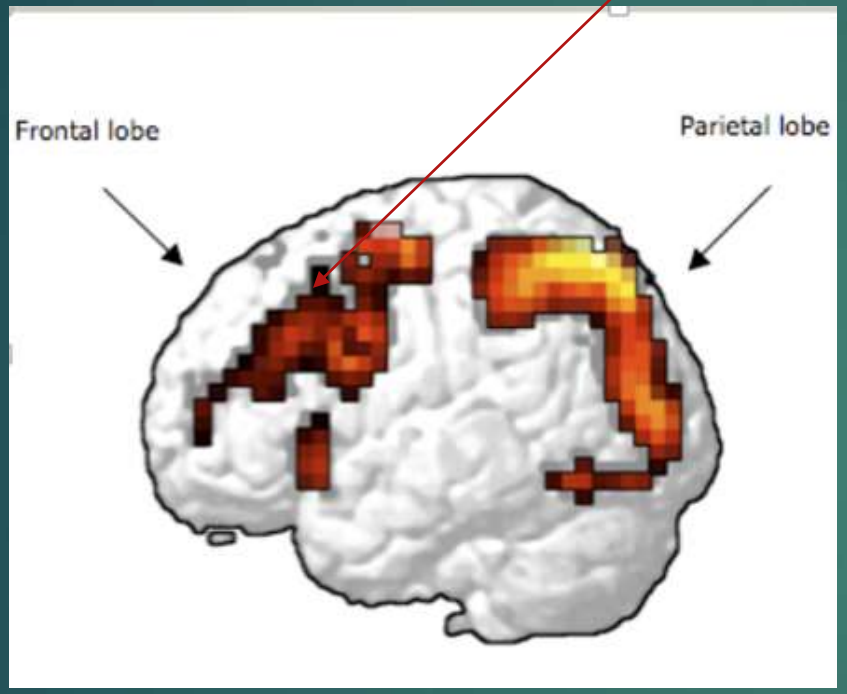


Lateral intraparietal sulcus (LIP) contains neurons that produce enhanced activation when attention is moved toward a stimulus

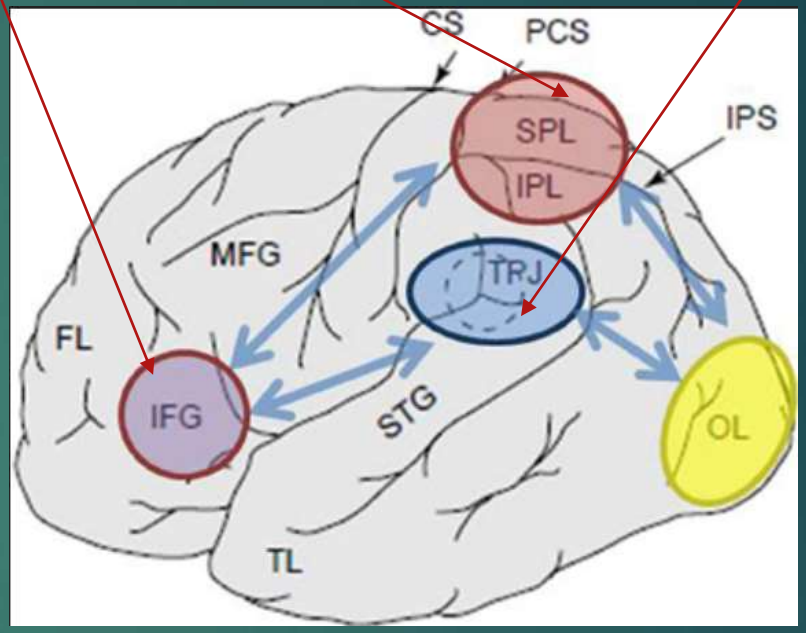
Attention = Prefrontal (goal) & Parietal (stimulus)

(top-down attentional orienting; task relevance)

bottom-up orienting: stimulus demand



Kingberg, et al., 2002



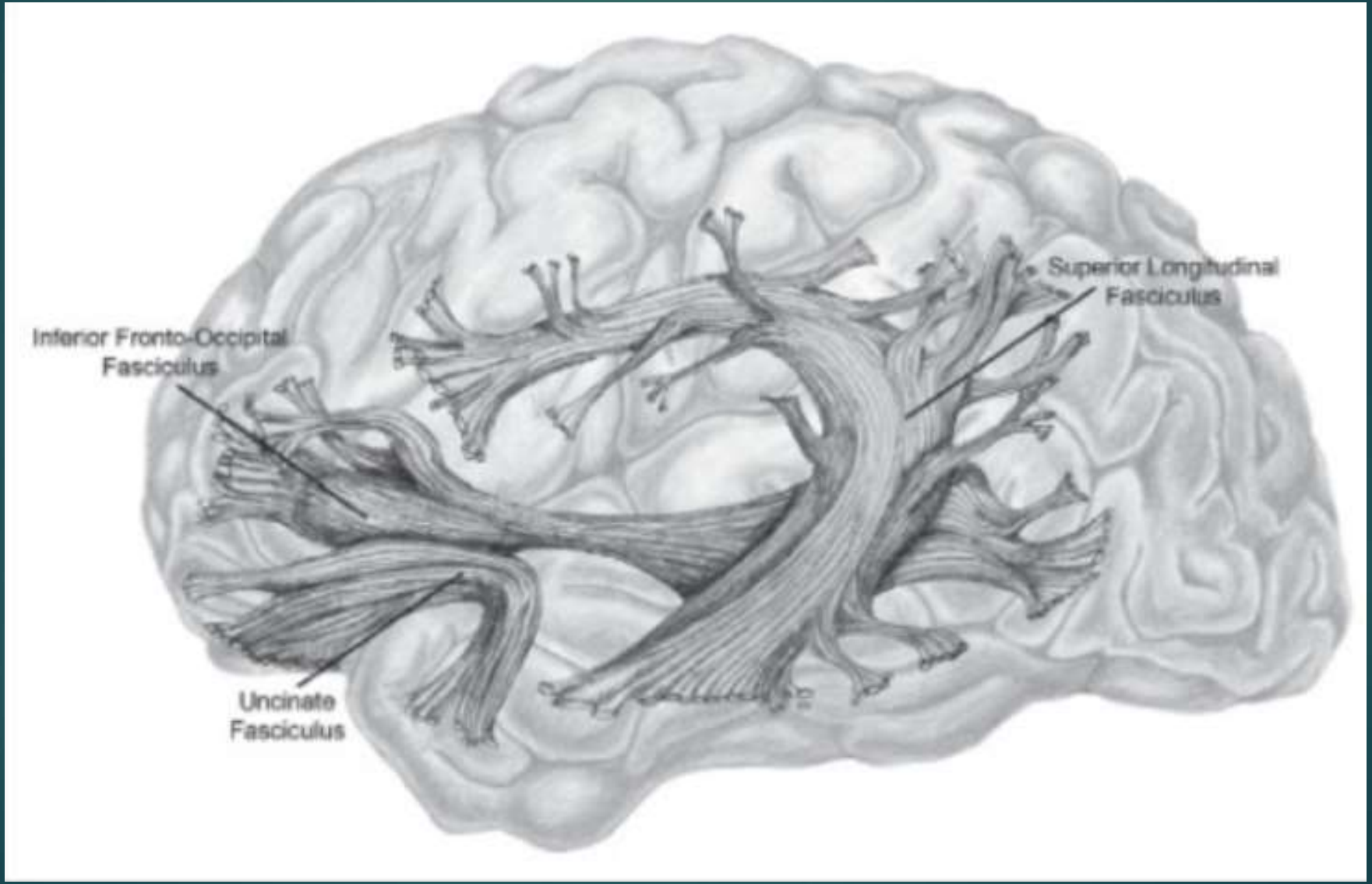
S. Shomstein, 2012

Prefrontal (top-down) & dorsal Parietal (bottom-up sensory input): attention can be controlled by intentions/expectation/goal direction of the observer as well as by the salience of the external physical stimulus;
IPL & TPJ = visual neglect;
TPJ: control signal that terminates the task at hand thus serving as a circuit breaker for attention

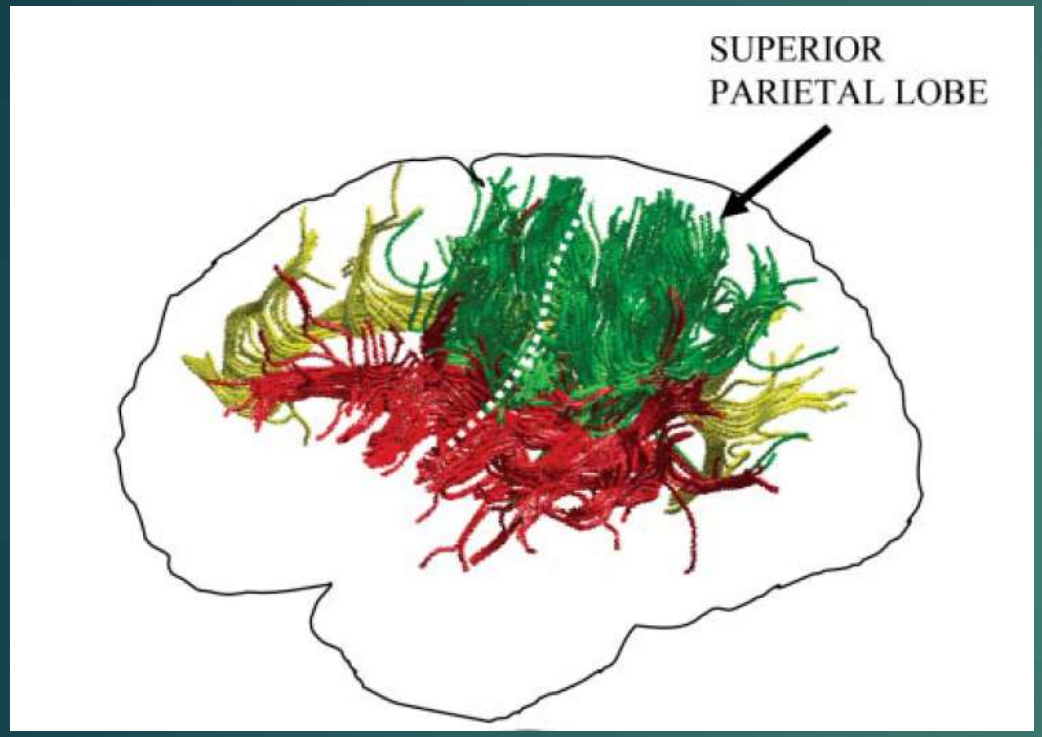
Partial lobe connections

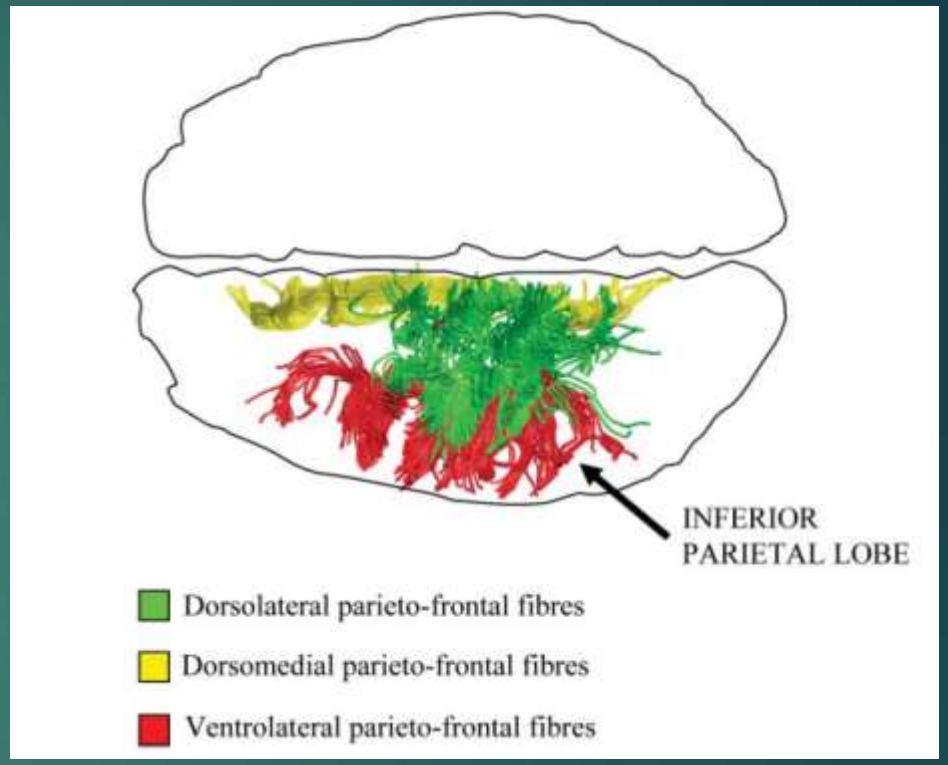
- Affrents (input)
  - Post central gyrus
    - VP thalamic nuclei
    - Spindle affrent -> 3a
    - Cutaneous affrent -> 3b + 1
    - Joint affrent -> 2
  - Association cortex
    - To frontal, temporal and occipital cortex of both side
- Effrents (output)
  - Somatosensory cortex -> area 5 superior parietal lobule
  - 1,3,5 (except hand and foot area) -> opp. somatosensory cortex

Associational fiber tracts connecting Parietal to all else



Frontoparietal circuitry of praxis network: Left hemisphere





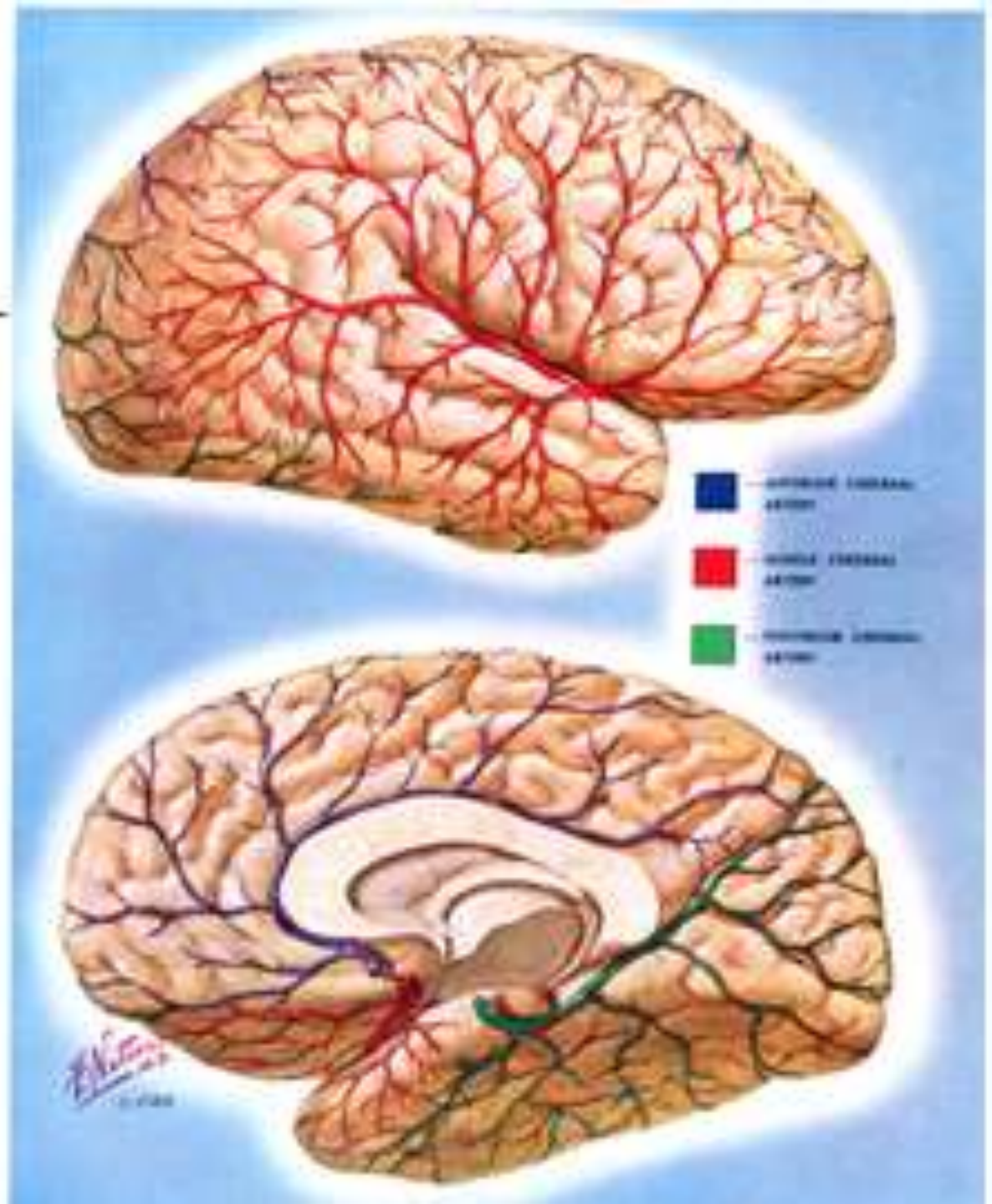
Green: dorsolateral fibers connecting the superior parietal lobe and dorsal motor and premotor cortex

Red: ventrolateral fibers connecting the inferior parietal lobe and ventrolateral motor and premotor cortex.

Yellow: dorsomedial fibers connecting the medial parietal lobe (precuneus) with the medial frontal lobe.
The dotted white line is the central sulcus.

Vascular supply

- Lateral - Middle Cerebral Artery
  - Artery of Rolandic fissure
  - Artery of inter parietal fissure
  - Artery of post parietal fissure
  - Inter opercular parietal artery
  - Artery to angular gyrus
- Medial - AnteriorCA mainly & PosteriorCA to a slight extent



Functions of Anterior Parietal Cortex

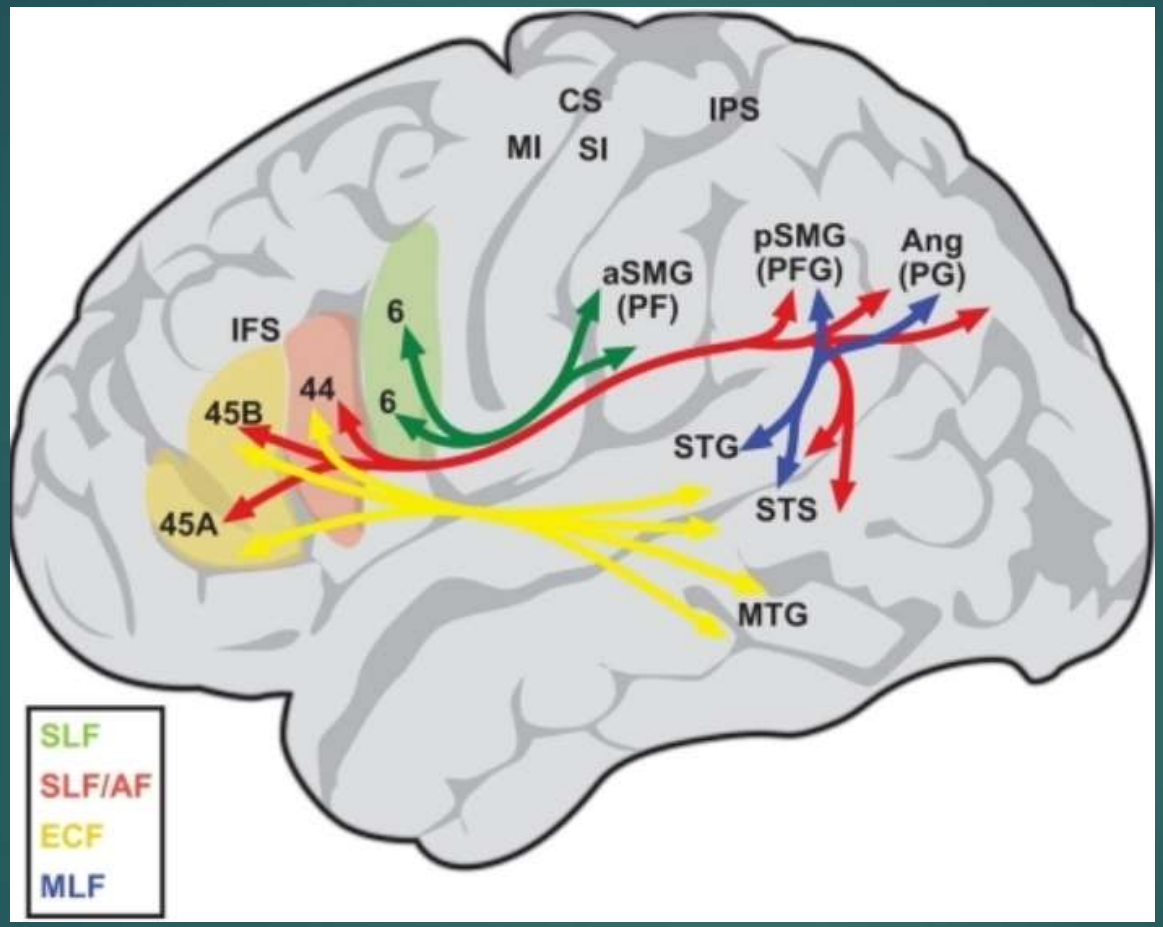
- Somatosensory perception
- Tactile perception
- Body sense
- Visual object recognition

- Classical sxs of PL lesions: tactile discrimination and stereognosis (tactile object recognition) deficits;
- Severe anterior lesion = sensory loss, complete anesthesia; resemble deafferentated states

Functions of Posterior Cortex

- Language
  - Reception of spoken language
  - Reading, writing
- Spatial orientation & attention
  - Spatial attention
  - Route following
  - L- R discrimination
- Calculation
- Praxis: Intentional movement
- Constructional ability
  - Drawing
- Auditory & visual working memory

Language and SMG and AG



Wernicke's Aphasia involves a "dorsal pathway" connecting auditory areas with parietal areas and with posterior ventrolateral prefrontal areas

Visual areas of the Parietal Lobes

- Visual processing areas
  - 1 - Intraparietal sulcus (IPS)
    - Control of saccadic eye movements
      - Saccade - involuntary abrupt and rapid small movements made by the eyes when changing the fixation point
    - Visual control of grasping
  - 2 - Parietal reach regions (PRR)
    - Visually guided grasping movements

Saccadic eye movement dysfunction

- Patients with Right PC lesions have saccades dysfunction:
  - Saccades into contralesional field are hypometric (small), have longer reaction times than those into ipsilesional field
  - They have difficulty compensating for saccades into the contralesional field

Post Central Gyrus:
Kinesthetic Basis of Movement

- Primary projection area for somatosensory and kinesthetic (movement sensation); proprioceptive inputs.
  - topographically organized,
  - body areas for tactile analysis (hands, face, mouth, tongue) receive disproportionate representation.
- Precise motor control
- Injury: both sensory and motor impairment

Posterior Parietal Functions

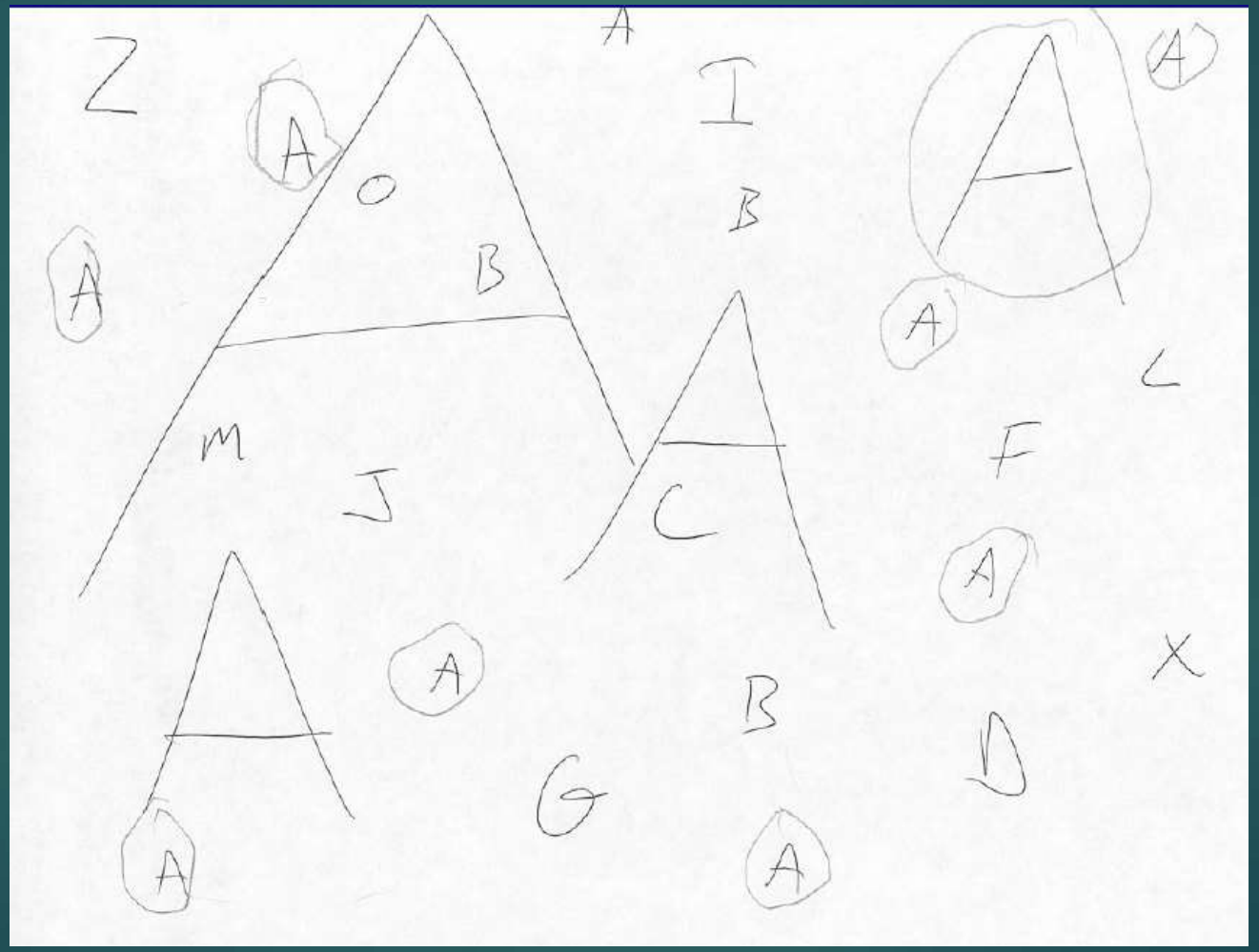
- Different parts of Posterior PC generate different sensory representations in different coordinate frameworks appropriate to different classes of actions, such as directing gaze, orienting, reaching, grasping
- There are multiple representations of external space used by sensorimotor control of specific motor systems
- Involved in visually guided reaching, monitoring body posture and movement, execution of reaching movements
- Classic deficit: optic ataxia, a reaching problem, based on loss of automatic online control of arm movements using currently available visual inputs

Disorder in visual control of action: Balint's syndrome

- Balint-Holmes Syndrome = disorder of spatially guided movements of eye and hand
- Bilateral Intraparietal sulcus lesion

  - Oculomotor apraxia: inability to move ocular fixation ("sticky fixation")
  - Simultanagnosia: inability to perceive more than a single object at a time; cannot bind the features of an object together; can see details but not whole (attentional disengagement deficit)
  - Optic Ataxia: difficulty in reaching toward objects in the visual field opposite to the side of the parietal damage with right hand

Simultanagnosia Disengagement deficit



Posterior Parietal Lobe Lesions

- Hallmark of PPC lesions is disturbance of specific attributes of perceptual feature extraction and elaboration; disturbance of higher-order processing of somatosensory information

- Tactile agnosia: Tactile form recognition, identification of object shape, shape discrimination, object naming deficit

- Deficient control of static (joint) force and posture

Posterior Parietal Lobe Dysfunctions

- Disturbance of body image:
  - tactile extinction,
  - Visual neglect,
  - anosognosia,
  - denial of hemiparesis,
  - asomatognosia (forgetting, ignoring, denying, disowning, or misperceiving the body (entirely or partially)
  - finger agnosia

- Apraxia

- Mirror Neuron deficits (IPL area): action observation deficit when objects are involved

Sensorimotor Integration in Posterior PC

- 19th Century: PPC as “association area” which associated different sensory modalities, with a purely sensory role
- Two functional pathways: Dorsal pathway (through PPC) involved in spatial (where) perception and a ventral pathway involved in object recognition
- Newer view: PPC & dorsal path are involved in sensorimotor integration i.e. shape is represented in PPC for action planning
- Lesions of PCC represent disconnects between visual sensory system and motor system: optic ataxia, apraxia, difficulty in correctly shaping hands in preparation to grasp objects

Sensorimotor Integration in Posterior PC

- Sensory extinction more common in superior PC lesions;
- Neglect more common in inferior PC lesions
- Role in sensorimotor attention: attention increases neuronal activation in PPC
- Separate visual maps for movements:
  - Lateral IP: specializes in saccade movement,
  - Parietal Reach Region (PRR): hand reaching
- PCC is engaged in most movement activation

Postcentral Gyrus Tests

1. Eyes closed - patient is to position hand to match position of other.
2. Passive finger detection.
3. Two point threshold.
4. Von Frey Hair threshold.
5. Vibration sense.
6. With lesion most severe changes are distal, coarse sensations return first
7. Height discrimination
8. Pinpoint vs. head.
7. Touch area on skin, have patient point to area on contralateral side.
8. Fine motor task: Fasten a button.
9. Tie a shoelace.
10. Localized lesion by deficit interactions.
11. Unusual speech. Consonant substitutions (especially of similar sounds), without broken or jerky speech typical of Broca's Aphasia. May see writing errors due to role of articulatory movements in analysis of words.

Postcentral gyrus (Brodmann areas 3,2,1): Primary Somatosensory Cortex

- Body image representation: postcentral gyrus is the primary somatosensory cortex (the sensory homunculus)
- Functions:
  - Representation of the body is contralateral, meaning that the right side of the body is represented by the left postcentral gyrus.
  - Proprioception and fine touch
  - Pressure and coarse touch
  - Temperature and pain (spinothalamic tracts) and general sensation from the head (trigeminothalamic tracts)
- Most of its afferent projections come from the ventral posterior nucleus of the thalamus, which is where the ascending somatosensory pathways terminate.

Somatosensory Areas

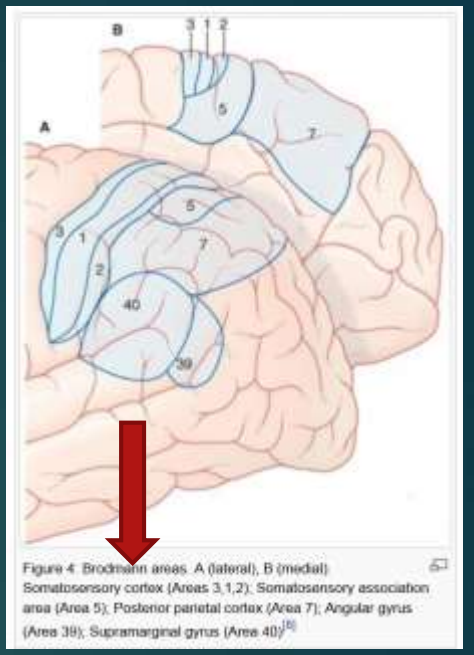


Figure 4: Brodmann areas. A (lateral), B (medial): Somatosensory cortex (Areas 3,1,2); Somatosensory association area (Area 5); Posterior parietal cortex (Area 7); Angular gyrus (Area 39); Supramarginal gyrus (Area 40)[6]

- Somatosensory Area I – S I.

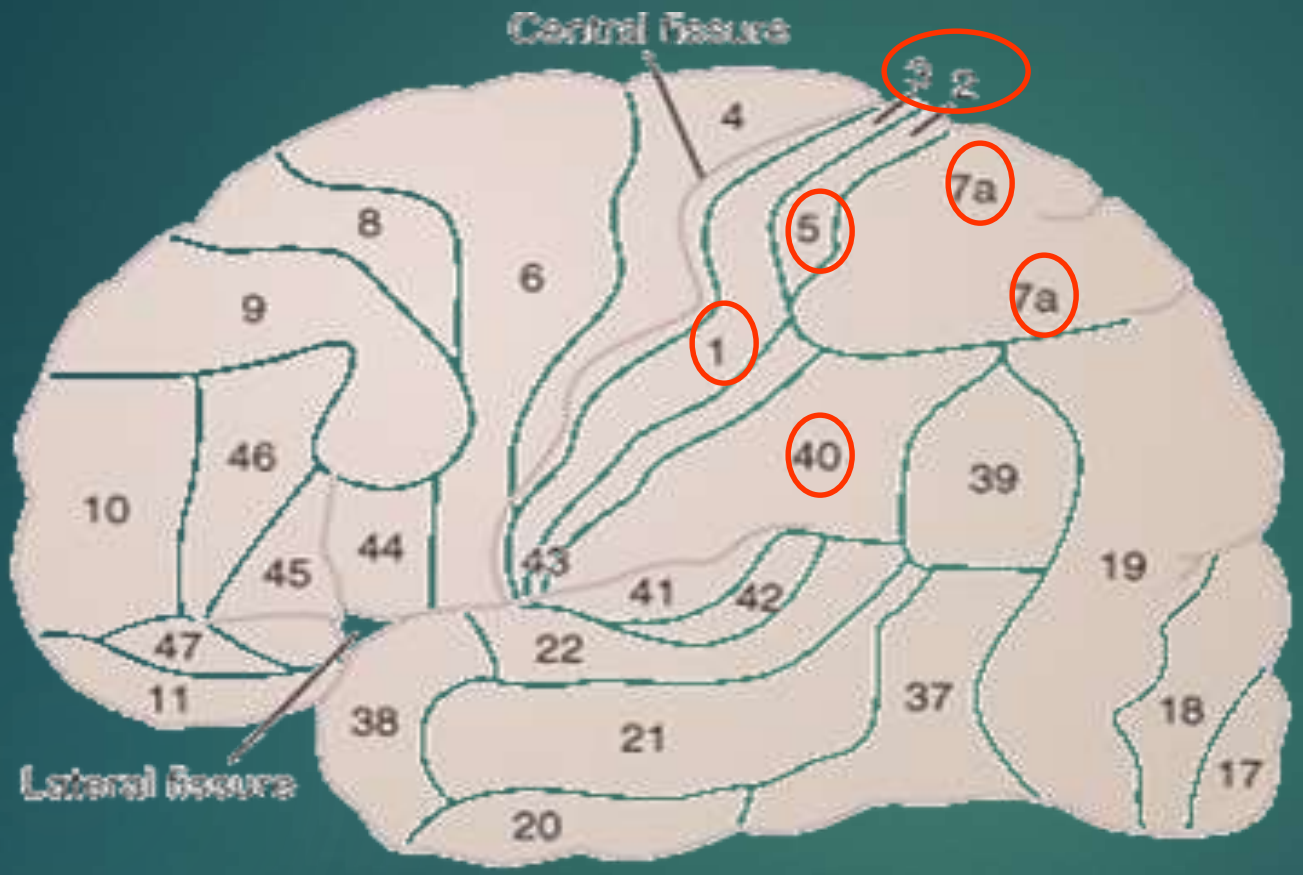
  (Brodmann area 1,2,3) – post central gyrus parietal lobe.

- Somatosensory area II – S II.

  (Brodmann area no. 40) in the wall of sylvian fissure which separate temporal lobe from frontal & parietal lobes.

- Sensory Association area (Brodmann area 5 & 7) located in parietal lobe behind S I.

SOMATOSENSORY CORTEX

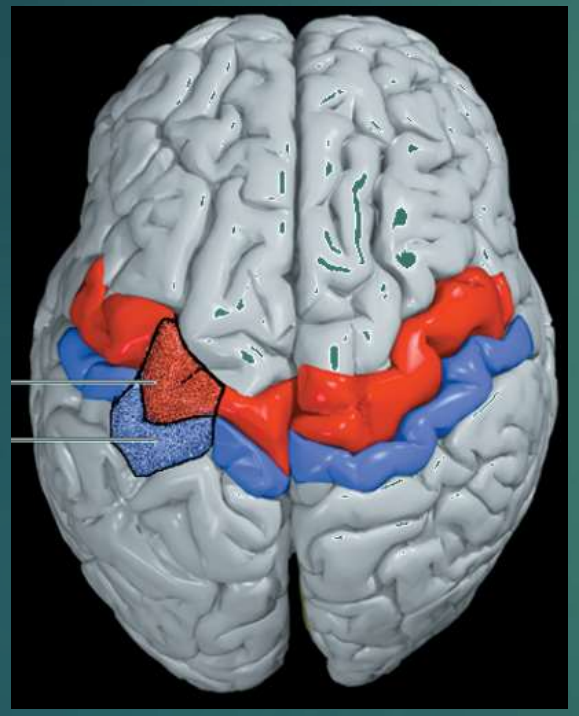


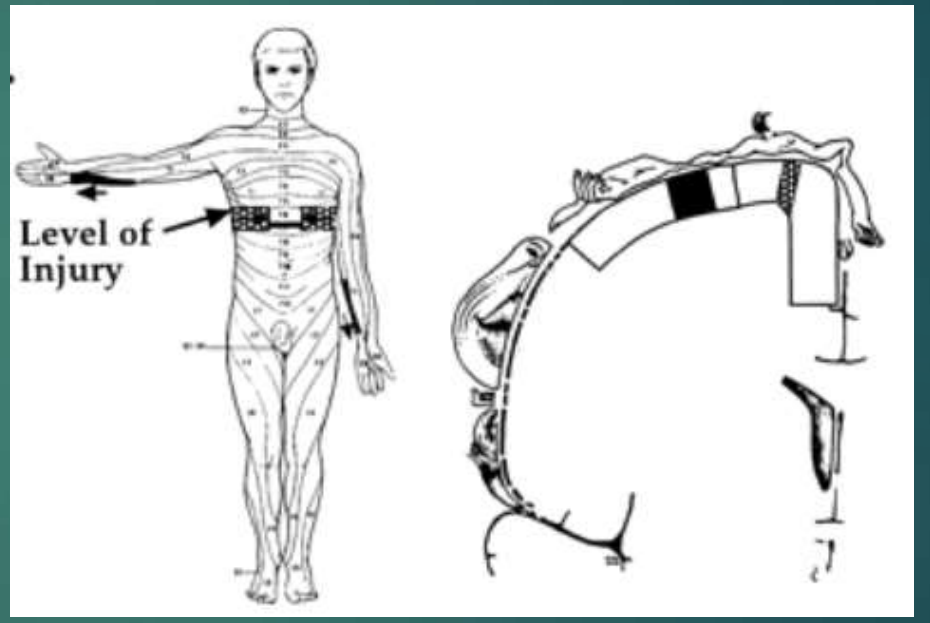
<u>Areas 1, 2, and 3</u>, which constitute PRIMARY SOMATOSENSORY AREA I

<u>40</u> is SECONDARY SOMATOSENSORY AREA II

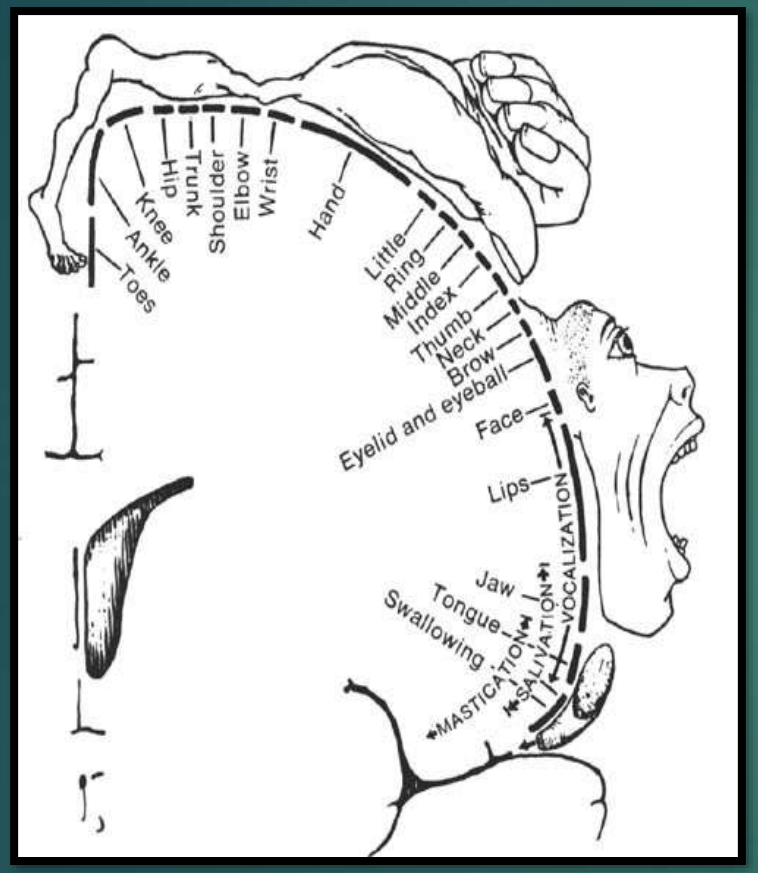
<u>5 and 7</u>, which constitute the SOMATOSENSORY ASSOCIATION AREA.

Representation of the different areas of the body in somatosensory area I



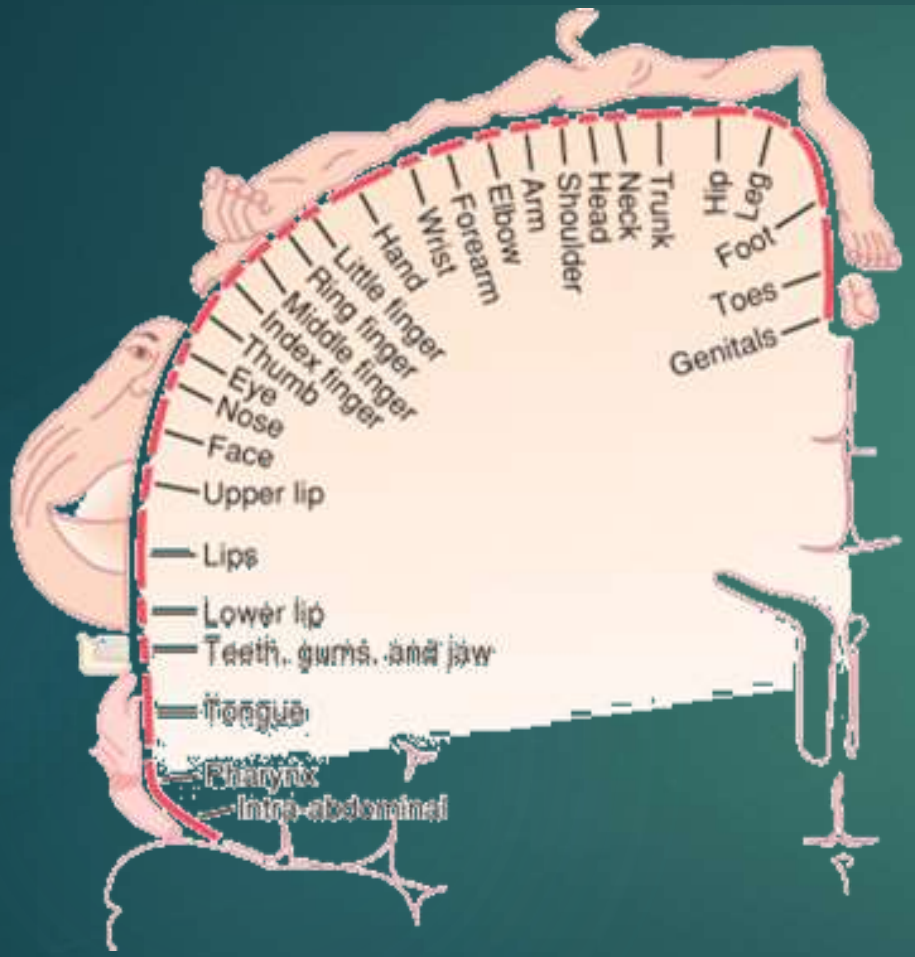


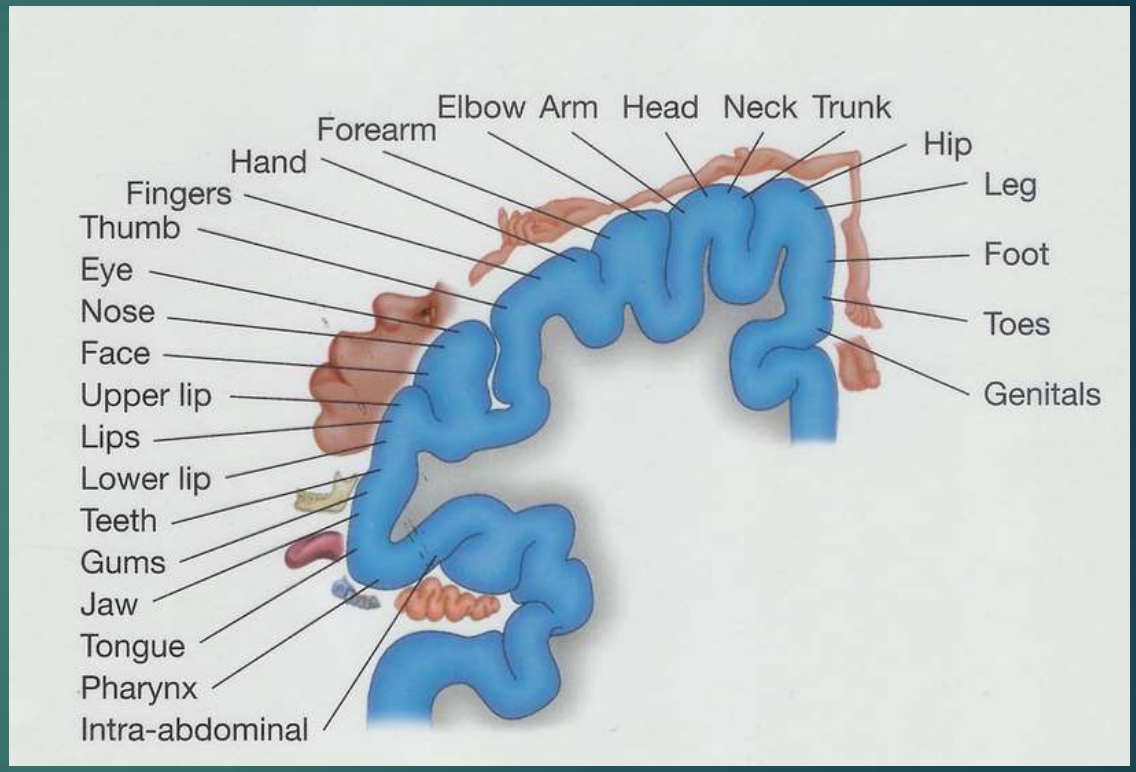
The idea of the cortical homunculus was created by Wilder Penfield.



Amount of cerebral tissue or cortex devoted to a given body region is proportional to how richly innervated that region is, not to its size.

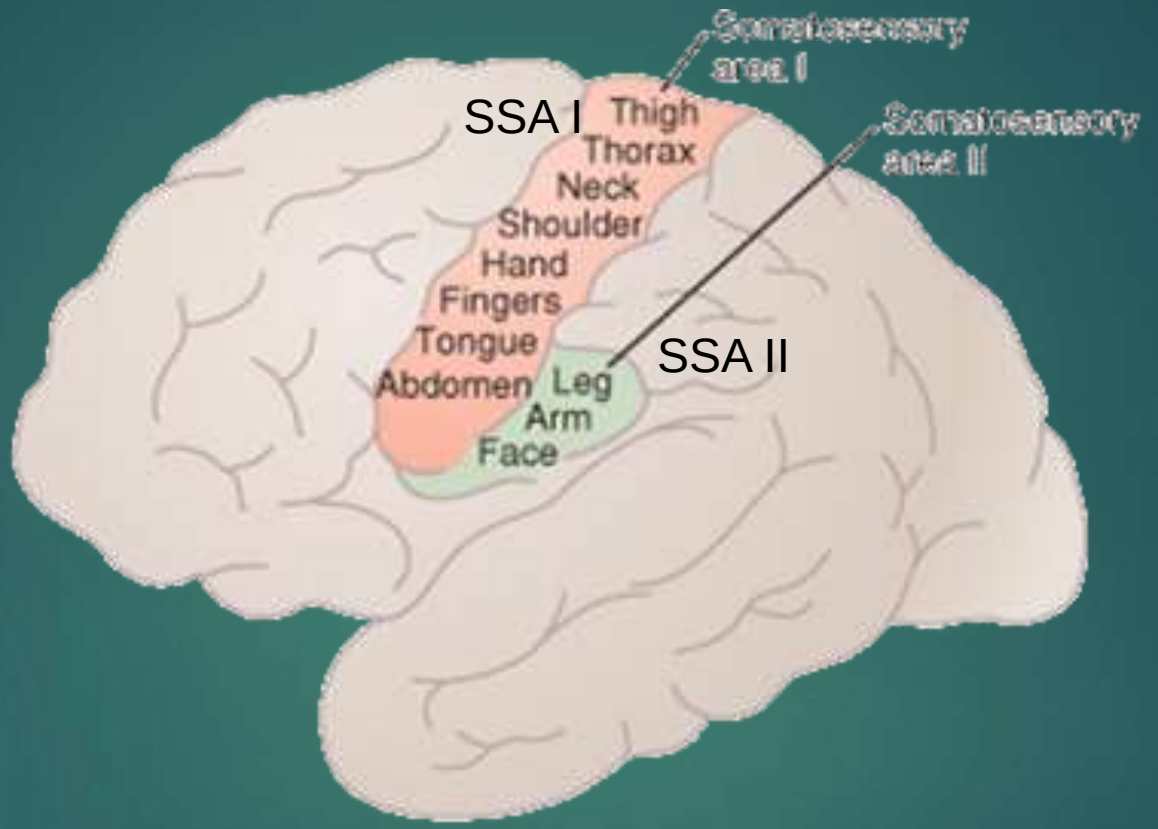
SI: Representation of the different areas of the body in somatosensory area I of the cortex





Size of cortical receiving area for impulses from a particular part of the body is proportionate to the number of receptors.

SOMATOSENSORY CORTEX



Somatosensory area I is so much more extensive and so much more important than somatosensory area II.
The term "somatosensory cortex" almost always means area I.

Dysfunctions of somatosensory area I

- Ablation (damaging) of SI area in animals causes loss of following types of sensory judgment;
  1. Loss of localization but still touch is felt.
  2. Loss of stereognosis (ability to judge size or shape of the object) = Astereognosis.
  3. Loss of fine touch, two point discrimination.
  4. Loss of proprioception.

Superior parietal lobule (Brodmann areas 5,7)

- Somatosensory association cortex: It is in charge of the interpretation of general sensory information and conscious awareness of the contralateral side of the body.
- The dorsal visual (where) stream also goes to the superior parietal lobe and it is concerned with control of visually guided arm and eye movements.

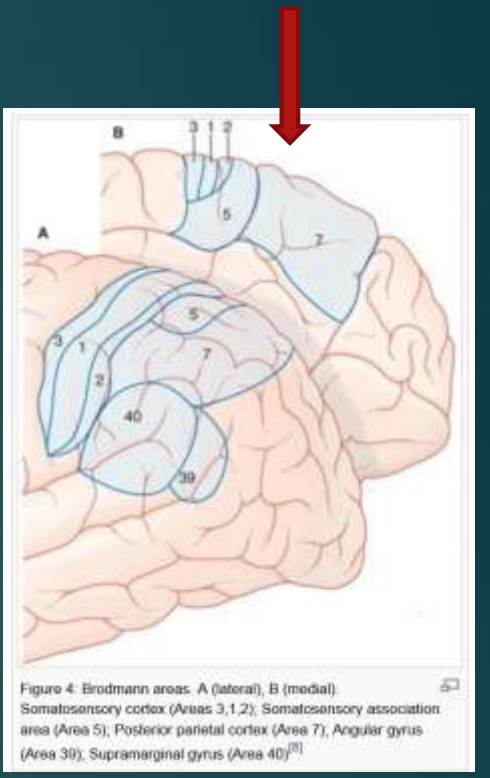


Figure 4: Brodmann areas. A (lateral), B (medial). Somatosensory cortex (Areas 3,1,2); Somatosensory association area (Area 5); Posterior parietal cortex (Area 7); Angular gyrus (Area 39); Supramarginal gyrus (Area 40)[8]

- **Symptoms and signs of dysfunction**
- Lesions may damage understanding and interpretation of sensory input.
- Optic ataxia may result, causing an inaccuracy of visually goal-directed movements of the hand (misreaching).

Amorphosynthesis: Effect of removing somatosensory association area (areas 5 & 7)

- Person loses these abilities:
  - Loss of recognition of objects felt on the opposite side of the body,
  - Loss of the sense of form of own body on the opposite side. Forget it is there.

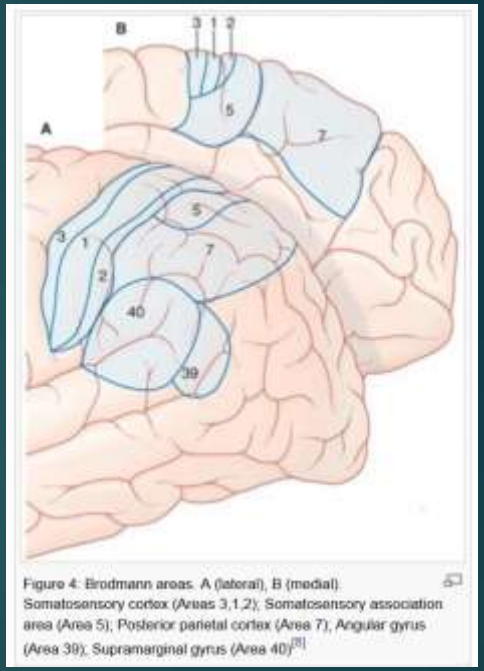


Figure 4: Brodmann areas: A (lateral), B (medial): Somatosensory cortex (Areas 3,1,2); Somatosensory association area (Area 5); Posterior parietal cortex (Area 7); Angular gyrus (Area 39); Supramarginal gyrus (Area 40)[3]

Sensory Perception without Postcentral Input

- In experimental animals & humans, cortical lesions of PPC do not abolish somatic sensations.
- Proprioception, fine touch are most affected by cortical lesion.
- Temperature sense is less affected
- Pain sensation is only slightly affected i.e. Pain & temperature is felt but poorly localized. Because of thalamus, brain stem and other basal regions of brain play role in discrimination of these sensations.
- Therefore some perception is possible in the absence of the sensory cortex.

Postcentral Dysfunction: Tactile deficits

- Paroxysmal localized paraesthesia (tingling sensations/'pins and needles'), affecting the contralateral side of the body. Also known as sensory seizure.

- Sensory deficits to the contralateral side can occur, leading to astereognosis, agraphesthesia, loss of proprioception, vibration and fine touch.

- The primary sensory modalities may return after a short time, however discrimination between sensations may not. Mild hemiparesis could also occur.

Clinical syndromes: either hemisphere

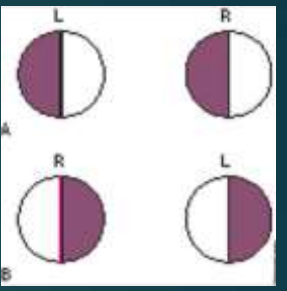
1. Cortical sensory syndrome & sensory extinction
2. Total hemi-anesthesia with large acute lesion of white matter
3. Mild hemiparesis, unilateral muscular atrophy in children, hypotonia, poverty of movements, hemiataxia
4. Homonymous hemianopia, visual inattention, sometime anosognosia, neglect of one half of body and extrapersonal space
5. Abolition of optokinetic nystagmus (occurs in normal response to a rotation movement) with target moving towards the side of lesion

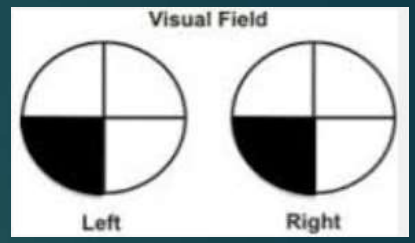
Somatosensory Symptoms of Parietal-Lobe Lesions

- Lesions to the postcentral gyrus produce:
  - Abnormally high sensory thresholds
  - Impaired position sense
  - Deficits in *stereognosis*, or tactile perception
  - Afferent paresis
    - Clumsy finger movements due to lack of feedback about finger position

Inferior PL (Brodmann areas 39,40) dysfunctions

- Thus lesions to this lobule may lead to:
  - homonymous hemianopia (visual deficits in the same vertical half of the visual fields of both eyes)
  - inferior quadrantanopia (visual deficits in the inferior quadrant of the visual field).





- Left IPL lesions: arrest of speech, alexia, agraphia, and acalculia. Also, finger agnosia, aphasia and left/right confusion may occur.

- Right IPL: contralateral neglect. There is a deficient response to stimuli on one side of the body and patients may fail to explore this space by limb or eye movements.

Parietal Lobe Functions

Functions

- Tactile perception (Anterior Parietal)
- Posterior SSA: tactile discrimination, position, tactile localization, stereognosis, graphaesthesia
- Spatial orientation
- Constructional activity
- Language - Understanding the grammatical & syntactical aspects of language
- Arithmetic, calculation

Dysfunctions

- Damage to the right hemisphere:
  - loss of spatial imagery
  - visualization of spatial relationships
  - neglect of left-side space and left side of the body. Drawings may be neglected on the left side.

- Damage to the left hemisphere:
  - problems in mathematics
  - long reading, writing, and understanding symbols.

Dysfunctions

- Gerstmann' s syndrome is associated with lesion to the dominant (usually left) parietal lobe; as well as damage of white matter fibers passing through angular gyrus
- Balint's syndrome is associated with bilateral lesions.
- Contralateral neglect is usually associated with large deficits of attention of the non-dominant (right) hemisphere.
- Optic ataxia is associated with difficulties reaching toward objects in the visual field opposite to the side of the parietal damage.

Inferior Parietal

1. *Multimodal assimilation* – capacity for organizing , labelling and conceptualizing , using all senses.

   Ex : recognize a chair

2. *Language capabilities*
   2. Left: visual recognition of symbols

      angular gyrus - anomia

      supramarginal gyrus – conduction & Wernicke’s aphasia

      visual cortex to IPC connections – word blindness

Inferior Parietal

3. *Agraphia* – left

Engrams for production and perception of written language are stored in IPC (misspellings, distorsions, and inversions occur.)

4. *Temporal sequential functions*

All sequential information (visual spatial functioning and temporal sequencing ability) i.e. either spatial sequential tasks lost – OXOXOX or sequential grammar relations are lost.

5. *Calculation (arithmetic) (Left) and computation (apply rules) (Right)*

Inferior Parietal Cortex Deficits

1. Apraxia for dressing.
2. Constructional apraxia (spatial apraxagnosia) - problems in motor integration in constructional tasks.
3. Spatial orientation deficit (more severe for right hemisphere lesions than left:).
4. Right-left disorientation.
5. Planto-pokinesia (disorganization of discriminations in spatial judgment).
6. Visuospatial agnosia.
7. Difficulty in performing reversible operations in extrapersonal space (difficulty in taking different perspectives) (more severe for right hemisphere lesions than left).
8. Inability to maintain visual image of patterned and verbal material.
9. Visuographic defects.
10. Contralateral neglect.
11. General intellectual impairment (lesions in left hemisphere).
12. Problems with writing and defective comprehension in reading.

IPC Testing

- 1. Inability to analyse positions of hands on a clock.
- 2. Confuses symmetrically arranged symbols (e.g., d & b).
- 3. Difficulty making rotations on a 2-D stick test.
- 4. Difficulty changing perspectives on a village scene test.
- 5. Difficulty with transformations on pool reflections test.
- 6. Problems on both visual and tactile route finding tests.
- 7. Difficulty in maze learning.
- 8. Inability to follow habitual routes.
- 9. Difficulty designating body parts on examiner.
- 10. Difficulty drawing common objects to demand.
- 11. Problems in visual memory for patterns and verbal matter.
- 12. Errors on the Bender.
- 13. Poor performance on Unknown Faces Test
- 14. Difficulty with simple addition, subtraction, multiplication, and division, both presented orally and written.
- 15. WAIS arithmetic subtest scores lowered.
- 16. Low test scores on Army General Classification Test.

Deficits in Either hemisphere

1. *Cortical tactile sensations*.
2. *Integration of sensory , motor and attention signals* (i.e. disengage attention - do other activity -immediately reengage correctly)
3. *Optic radiation* passes through.
4. *Constructional ability* – capacity to construct or draw 3D/2D figures or shapes; Black designs, clock drawing.

   Left – programming of movements necessary for constructional activity. (simplification of complex diagrams)

   Right – related to spatial relationships or imagery. (rotation of diagrams)
5. Working memory

   Lt. – immediate recall for digits and words

   Rt. – immediate recall for geometric patterns

Parietal lobe dysfunction signs (right or left)

1. Loss of cortical sensations.
2. Mild hemiparesis, hypotonia and hemiatrophy.
3. Visual field deficits: Hemianopia / quadrantanopia
4. Sensory and visual inattention; Visual spatial imperceptions
5. Astereognosis (inability to identify an object by active touch of the hands)
6. Agraphaesthesia (inability to read numbers or letters drawn on hand)
7. Failure of blind two-point discriminatory testing
8. Spatial neglect
9. Constructional and dressing apraxia
10. Optic ataxia (inability to reach and grab objects)

Table 14.1 Effects of left- and right-parietal-lobe lesions compared

	PERCENTAGE OF SUBJECTS WITH DEFICIT*	
	Left (%)	Right (%)
Unilateral neglect	13	67
Dressing disability	13	67
Cube counting	0	86
Paper cutting	0	90
Topographical loss	13	50
Right–left discrimination	63	0
Weigl's Sorting Test	83	6

*Note the small but significant overlap in symptoms of left and right lesions.
Source: Based on data presented by McFie and Zangwill, 1960.

Unilateral parietal dysfunction

- Sensory deficits
  - Astereognosis
  - Agraphesthesia
  - Sensory seizures
- Motor deficits: mild hemiparesis
- Visual disorders
  - Homonymous hemianopia
  - Inferior quadrantanopia
  - Visual inattention
- Contralateral neglect (more obvious for right sided lesions)
- Constructional and dressing apraxia

Somatoperceptual Disorders

- Astereognosis
  - Inability to recognize an object by touch
  - Unable to name objects, describe or demonstrate their use
  - Primary sensations intact

- Simultaneous Extinction
  - Two stimuli are applied simultaneously to opposite sides of the body
  - A failure to report a stimulus on one side is referred to as *extinction*
- Blind Touch
  - Cannot feel stimuli, but can report their location

Dominant/Left Hemisphere Parietal Deficits

1. Disorder of language
    - Wernicke’s fluent aphasia, alexia with agraphia, anomia
2. Gerstmann's syndrome (Angular gyrus):
3. Tactile agnosia (bimanual astereognosis)
4. Bilateral Ideomotor & ideational apraxia

Gerstmann's Syndrome: Posterior Parietal Lobe Damage

- Finger agnosia
- Right-Left Confusion
- Dysgraphia
- Acalculia

- Results from a left inferior parietal lobe (angular gyrus) lesion
- May be ass. with dyslexia or homonymous hemianopia / quadrantanopia.

Finger agnosia

- Inability to recognize, name & point to individualized fingers on self & others – usually middle 3 fingers
- Form of autotopagnosia: wherein one loses the ability to identify the parts of one's body to command or imitation.
- B/L lesion: Left inferior parietal lobe lesion, particularly in angular gyrus
- Central feature of Gerstmann syndrome

Tests for finger agnosia

- *Inability to name , point or recognize fingers on oneself or others.*

1. Non verbal finger recognition:

   With pt eyes closed, touch one of his fingers. Ask him to touch the same finger of examiner, with eyes open.

2. Identifying named fingers on examiner's hand:

   Examiner places hand in some irregular position and asks pt – " point to my middle finger"

3. Verbal identification (naming) of fingers:

   Either examiner's or pt's hand kept in an irregular position. Examiner points to a finger and asks him – "name this finger?"

Right-Left Disorientation

- Inability or loss of the ability to identify the right and left sides of one's own body or of another person.
- A special case of autotopagnosia
- Lesion of left IPC, esp. angular gyrus

Tests for right – left confusion

- Identification on self

  ex : show your left foot.
- Crossed commands on self

  ex : with your right hand touch your left ear
- Identification on examiner

  ex : point to my right elbow
- Crossed commands on examiner

  ex : with your left hand point my right foot.

Left Parietal: Language dysfunction

- Wernicke's Aphasia: defect in reception of spoken language & reading; timing of language
- Conduction aphasia
- Dyslexia: reading (left AG damage has been associated with some forms of developmental dyslexia)
- Agraphia: writing
- Spatial organization of words: "tap" vs. "pat"

Conduction aphasia: due to left arcuate fasciculus, SMG, and TPJ

- Classically, conduction aphasia (impaired repetition) is thought to result from a disconnection between sensory and motor speech areas caused by damage to the arcuate fasciculus.
- Recent work: Structural damage to the white matter beneath the left supramarginal gyrus, which includes the arcuate fasciculus, was the most strongly correlated region with repetition impairment.
- Perfusion imaging implicates a cortical zone that included the parietal operculum (inferior SMG) and a temporal-parietal junction region
- Speech repetition is "strongly associated with damage to the left arcuate fasciculus, supramarginal gryus, and TPJ"

Effects of unilateral disease of the nondominant (right) parietal lobe

A. Visuospatial disorders

B. Topographic memory loss

C. Anosognosia, dressing and constructional apraxias

D. Confusion

E. Tendency to keep the eyes closed, resist lid opening, and blepharospasm

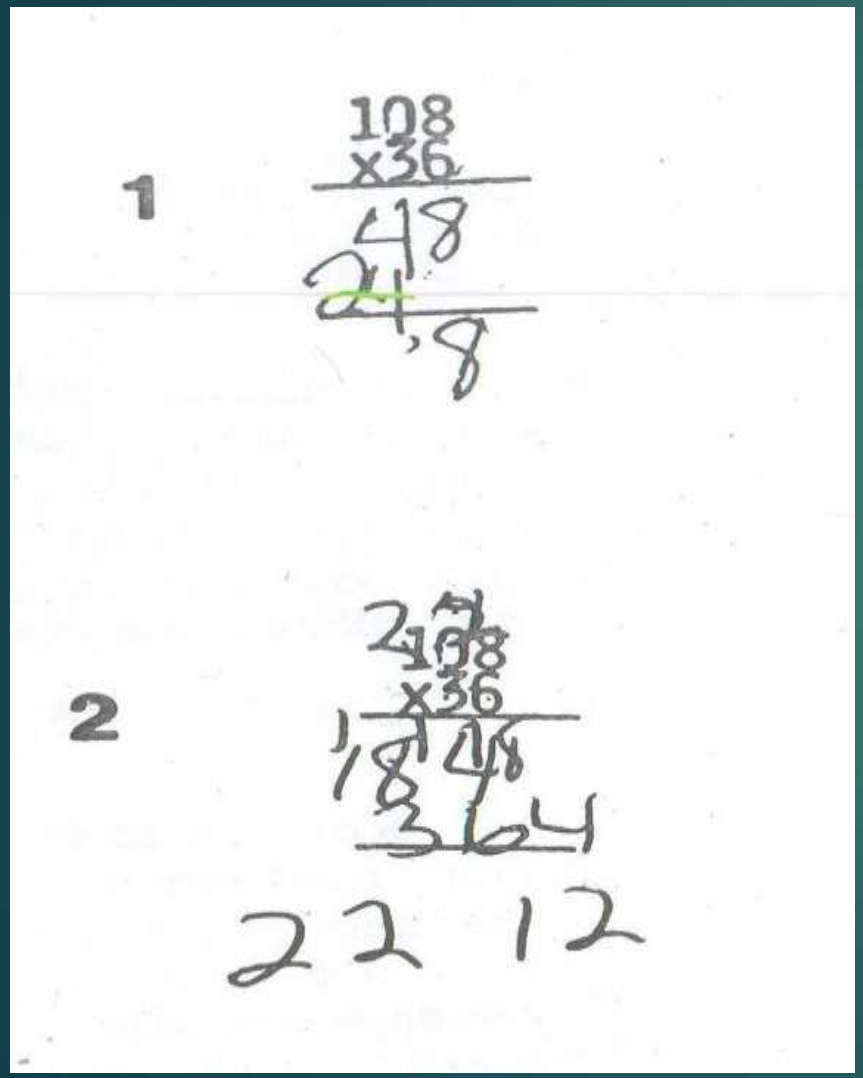
Left Parietal: Acalculia

- Difficulty or inability to do arithmetic
- Noted in parietal lobe patients
- Might result from the spatial properties of addition and subtraction
  - Two digit number occupy different spaces
  - “Borrowing” during subtraction

Right Parietal: Computation

1. Calculations –
    1. arithmetic concepts of carrying and borrowing
    2. spatial alignment of written calculations
    3. computational difficulty – inability to manipulate numbers in spatial relation, like using decimals, aligning numbers on paper, etc.
    4. ***But able to do problems in head***

Written complex examples: Right Parietal



- Pt with right hemisphere lesion: left neglect.
- Pt with right parietal hematoma – showing poor alignment and calculation errors.

Tests for Acalculia

- Components – Rote tables (add, multiply, etc.)

  Recognition of signs (+ , - , *)

  Basic arithmetic(carrying, borrowing)

  Spatial alignment of written calculations

- Verbal rote examples : what is 4 plus 6 ?

- Verbal complex examples : what is 21 * 5 ?

Visual Agnosia (loss of recognition (meaning) of visual objects)

Subtypes of Associative Visual Agnosia

Agnosia is a conceptual disorder and involves *both sides of the body and* extrapersonal space as a result of damage to the dominant hemisphere.

•Achromatopsia, an inability to distinguish different colors.

•Prosopagnosia, an inability to recognize human faces. Individuals with prosopagnosia know that they are looking at faces, but cannot recognize people by the sight of their face, even people whom they know well.

•Orientation Agnosia: an inability to judge or determine orientation of objects.

•Pantomime Agnosia: an inability to understand pantomimes (gestures). It appears that the inferior cortical visual cortex is critical in recognizing pantomimes.

•Oliver Sacks' book, *The Man Who Mistook His Wife for a Hat*

Original Sources: Major Symptoms and their Assessment

Table 14.2 Summary of major symptoms of parietal-lobe damage

Symptom	Most probable lesion site	Basic reference
Disorders of tactile function	Areas 1, 2, 3	Semmes et al., 1960 Corkin et al., 1970
Tactile agnosia	Area PE	Hécaen and Albert, 1978 Brown, 1972
Defects in eye movement	Areas PE, PF	Tyler, 1968
Misreaching	Area PE	Damasio and Benton, 1979
Manipulation of objects	Areas PF, PG	Pause et al., 1989
Apraxia	Areas PF, PG, left	Heilman and Rothi, 1993 Kimura, 1980
Constructional apraxia	Area PG	Benton, 1990
Acalculia	Areas PG, STS*	Levin et al., 1993
Impaired cross-modal matching	Areas PG, STS	Butters and Brody, 1968
Contralateral neglect	Area PG right	Heilman et al., 1993
Impaired object recognition	Area PG right	Warrington and Taylor, 1973
Disorders of body image	Area PE?	Benton and Sivan, 1993
Right–left confusion	Areas PF, PG	Semmes et al., 1960
Disorders of spatial ability	Areas PE, PG	Newcombe and Ratcliff, 1990
Disorders of drawing	Area PG	Warrington et al., 1966 Kimura and Faust, 1987

*STS, superior temporal sulcus.

Original Sources: Clinical Neuropsychological Assessment

Table 14.3 Standardized clinical neuropsychological tests for parietal-lobe damage

Function	Test	Basic reference
Somatosensory threshold	Two-point discrimination	Corkin et al., 1970
Tactile form recognition	Seguin–Goddard Form Board (tactile patterns)	Teuber and Weinstein, 1954 Benton et al., 1983
Contralateral neglect	Line bisection	Schenkenberg et al., 1980
Visual perception	Gollin Incomplete Figures Mooney Closure	Warrington and Rabin, 1970 Milner, 1980
Spatial relations	Right–left differentiation	Benton et al., 1983
Language		
Speech comprehension	Token	de Renzi and Faglioni, 1978
Reading comprehension	Token	
Apraxia	Kimura Box	Kimura, 1977

Note: These standardized tests have been validated on large samples of patients with known localized brain damage.

Agnosias: Somatoperceptual Disorders

- Agnosias
  - Asomatognosia
    - Loss of knowledge or sense of one's own body
  - Anosognosia
    - Unawareness or denial of illness
  - Anosodiaphoria
    - Indifference to illness
  - Asymbolia for pain
    - Absence of normal reactions to pain
  - Finger Agnosia

Disorders of the Body Image - Critchley

- Unilateral neglect
- Lack of concern over the existence of hemiparesis (Anosodiaphoria)
- Unawareness of hemiparesis (anosognosia)
- Defective appreciation of the existence of hemiparesis
- Denial of hemiparesis, with rationalization
- Denial of hemiparesis, with confabulation
- Loss of awareness of one body-half (Asomatognosia)
- Undue heaviness, deadness or lifelessness of one half of body
- Phantom third limb, associated with a hemiparesis

Remapping Somatosensory Cortex after injury: Phantom Limb

- Lesion induced plasticity: if arm cut off (deafferentation), somatosensory arm area can be invaded by neighboring areas, i.e. neuronal input from face area can activate previous arm area
- Result is phantom limb phenomena: referred sensation in amputated limbs; it is the perceptual correlate of cortical reorganization
- Longstanding or intense acute pain in limb prior or during amputation can lead to establishment of SS pain memory
- More SS reorganization, the greater the pain perceived
- Chronic low back pain can have same effect
- Ramachandran: virtual reality mirror box treatment
- Memantine significantly helpful

Ramachandran

Cortical Sensory Syndromes

- ASOMATAGNOSIAS
- APRAXIAS
- VISUAL DISORDERS
- AUDITORY NEGLECT

Somatagnosias

- Disturbances in general feeling pertaining to the existence of one's body or recognition of one's body schema

- Due to Parietal lobe dysfunction:
  - Astereognosis
  - Asomatognosia

Cortical Sensory Syndromes

- *The perception of pain, touch, pressure, vibratory stimuli, and thermal stimuli is relatively intact in parietal lobe lesions.*

- Cortical defect is essentially loss of ***sensory discrimination*** i.e. impaired ability to integrate and localize stimuli.

    1. Loss of position sense and passive movement.
    2. Topagnosia – loss of localization of tactile, thermal and noxious stimuli.
    3. Astereognosis.
    4. Agraphesthesia.
    5. Loss of ‘two point’ discrimination.

Other parietal sensory defects :

- Easy fatigability of sensory perceptions
- Inconsistency of responses to painful and tactile stimuli
- Pain sensations outlast the stimuli & hyperpathia
- Tactile hallucinations

Astereognosis Testing

- Pressure sensitivity
- Two point discrimination
- Point localization
- Position sense
- Tactual object recognition

Tests for cortical sensations

Graphesthesia :

Done with pencil or swab stick.

Sites – palms, fingers and face.

- ✓ Digits like 1-9 , or shapes/symbols used.
- ✓ Stand beside the pt and face the area to be tested (so that he will be familiar).

Stereognosis :

- ▶ no preliminary visual demo given.
- ▶ ex: key, pen or coin
- ▶ abnormal side done first and then normal side.

Double simultaneous stimulation testing:

- Pin prick used (both must be equally sharp).
- ✓ Eyes to be closed
- ✓ Pt is told to expect sensation either one side or both sides.
- ✓ After stimuli, ask to indicate site of stimulus and their nature.

POSITIVE test – stimuli on involved half is ignored or sharp stimulus interpreted as dull.

Nomenclature:

Sensory extinction

Sensory inattention

Sensory suppression

Sensory eclipse

Tactile inattention

Perceptual rivalry

Test of cortical sensation: Two point discrimination

- Use a compass / calibrated 2 point esthesiometer:
  - 1 mm tip of tongue
  - 2-4 mm finger tips
  - 4-6 mm dorsum (top) of fingers
  - 8-12 mm on palm
  - 20-30 mm on dorsum (top) of palm

Asomatognosia

- Acquired disturbance in the knowledge or sense of one's own body and bodily condition; *inability* to recognize a part of one's body.

- Lesions in bilateral parietal, or unilateral in right IPC bordering interparietal sulcus, suparamarginal gyrus, and angular gyrus or unilateral lesion of dominant parietal lobe, esp. angular gyrus

- Types
  - Anosognosia
  - Autotopagnosia: characterized by an inability to localize and orient different parts of the body

Testing Asomatognosia

Table 1: Cutting's (1978) verbal questionnaire used to assess for impaired body awareness.

Possible Disorder/Symptom	**Questions**
Anosodiaphoria	1. Is it a nuisance?
	2. How much trouble does it cause you?
	3. What caused it?
Nonbelonging	1. Do you ever feel that it does not belong to you?
	2. Do feel that it belongs to someone else?
Strange feelings	1. Do you feel the arm is strange or odd?
Misoplegia	1. Do you dislike the arm?
	2. Do you hate it?
Personification	1. Do you ever call it names?
Somesthetic/Kinesthetic Hallucinations	1. Do you ever feel it moves without your moving it yourself?
	2. How big or strong is it?
	3. How is the other arm?
Phantom supernumerary illusion	1. Do you ever feel as if there was more than one arm/hand?
	2. Do you ever feel a strange arm is lying beside you?
	3. Do you ever feel you arm as separate from the real one? Or from you?

Anosognosia: Denial of illness

- Ignorance of existence of disease
  - Deny weakness /sensory loss of affected limb
  - Extreme cases - disown limb

- Denial is more implicit than explicit, in many pts. (i.e. they may not actively deny that they are ill); and some may act as if nothing were the matter.

- 7 x more frequent with *Right sided lesions* than left.

Anosognosia: comorbidities

- Contralateral neglect may co-exist
- Associated with blunted emotionality – pts look dull, inattentive and apathetic. And also confused.
- Associated with hallucinations of movement and *allocheiria* (one sided stimuli are felt on other side).
- Anton-Babinski syndrome (occipital): "cortically blind", but affirm, often quite adamantly and in the face of clear evidence of their blindness, that they are capable of seeing

Autotopagnosia

- Impairment in localization / naming of parts of own body
- Patient unable to point to body parts named by examiner / move them
- May not be able to identify them on examiner's body / on diagram

Amorphosynthesis

- Amorphosynthesis is a inability to synthesize separate tactile sensations into perception of form
  - Lack of recognition of contralateral body & of space
- Left-sided lesions cause agnosia, a full-body loss of perception
- Right-sided lesions cause contralateral neglect, lack of recognition of the person's left side and extrapersonal space.
- The term amorphosynthesis was coined by D. Denny-Brown to describe patients he studied in the 1950s.

W.R. Brain: Contralateral Neglect

In 1941, the British neurologist W. R. Brain reported three patients with unilateral parietal lobe lesions with perceptual difficulty.

"Though not suffering from a loss of topographical memory or an inability to describe familiar routes, they nevertheless got lost in going from one room to another in their own homes, always making the same error of choosing a right turning instead of a left, or a door on the right instead of one on the left. In each case there was a massive lesion in the right parieto-occipital region, and it is suggested that this … resulted in an inattention to or neglect of the left half of external space. The patient who is thus cut off from the sensations which are necessary for the construction of a body scheme may react to the situation in several different ways.
He may remember that the limbs on his left side are still there, or he may periodically forget them until reminded of their presence. He may have an illusion of their absence, i.e. they may 'feel absent' although he knows that they are there; he may believe that they are absent but allow himself to be convinced by evidence to the contrary; or, finally, his belief in their absence may be unamenable to reason and evidence to the contrary and so constitute a delusion. W. R. Brain, 1941 (*Brain* 64: pp. 257 and 264)

This description is generally considered the first account of the link between parietal lobe lesions and deficits in attention or perceptual awareness.

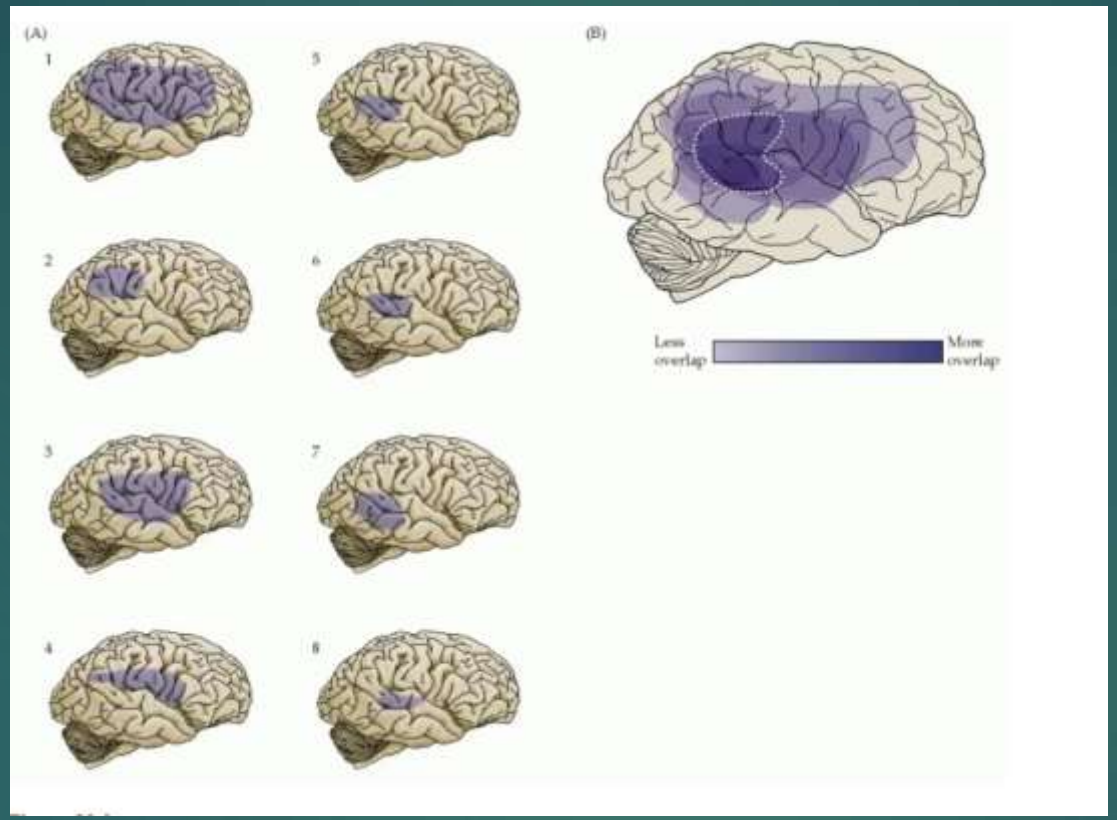
Right Parietal Specialization for Spatial Attention

- The parietal cortex, particularly the inferior parietal lobe, is the primary cortical region governing spatial attention.

- Contralateral neglect syndrome: damage to right intraparietal sulcus and the right angular gyrus

- RP: attention to both left and right halves of the body and extrapersonal space, whereas the left hemisphere mediates attention only to the right.
- LP lesions tend to be compensated by the intact right hemisphere.
- RP lesions: little or no compensatory capacity in the left hemisphere to mediate attention to the left side of the body or extrapersonal space. There is also increased activity in the right frontal cortex during such tasks.

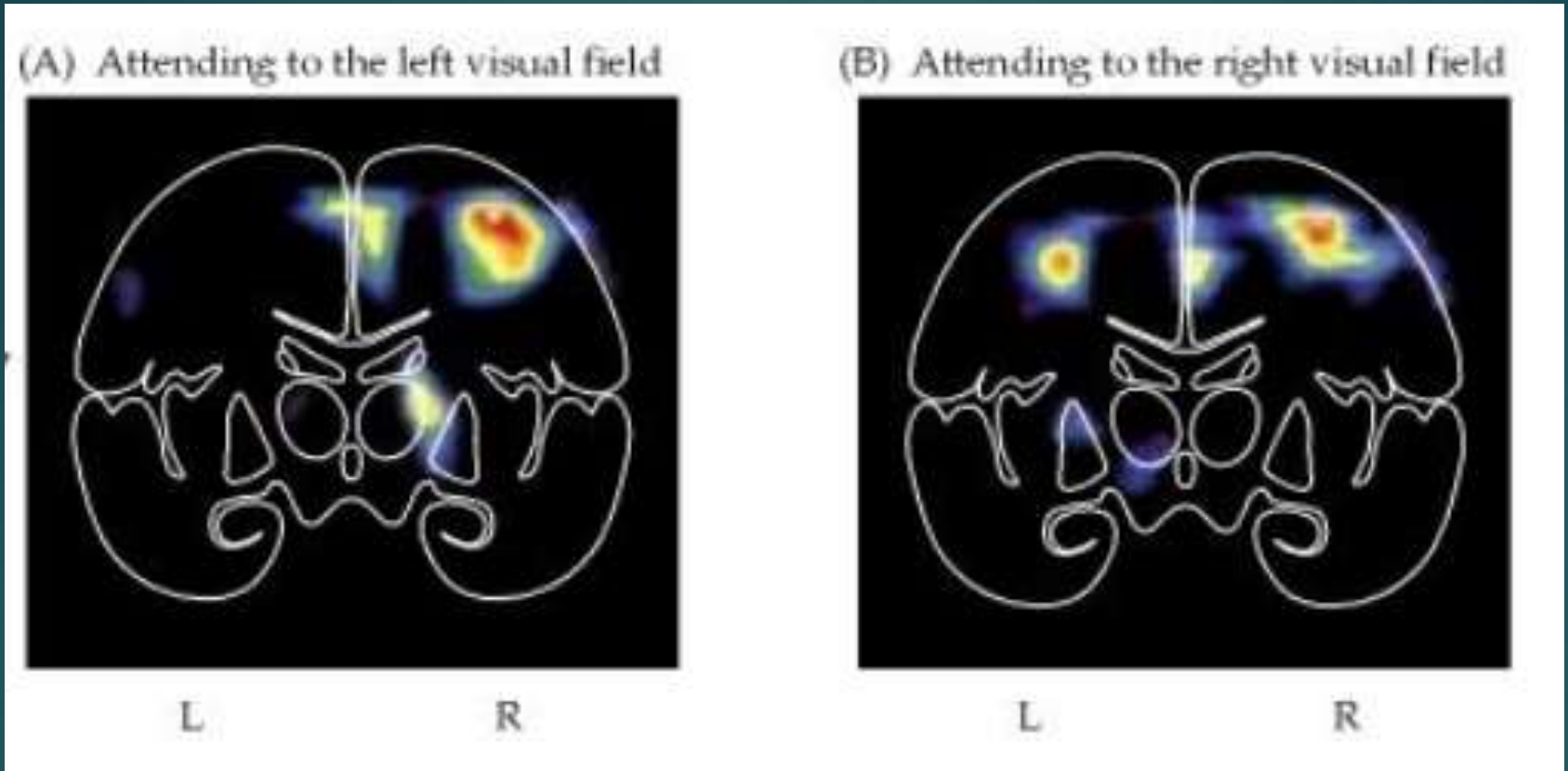
Right Parietal localization of Contralateral Neglect



8 pts with contralateral neglect; 1-5 parietal lobe; 6-8 mainly R temporal (pathway disruption to Parietal lobe; B.: region most affected in R parietal

Neglect can also be caused by Right DL premotor and medial frontal lesions.

Normal attention activation



RP cortex of **normal subjects** is highly active during tasks requiring attention.

(A) In attention to objects in the left visual field; only the RP parietal cortex is active.

(B) In attention to right visual field, RP cortex remains active, but activity is also apparent in the left parietal.

Implies that damage to the left parietal lobe does not generate right sided hemineglect because the right parietal lobe also serves this function. (After Posner and Raichle, 1994.)

Neglect

- Contralateral neglect: The most prominent attention disorder associated with PC lesions; characterized by defective detection of events and impaired exploratory activities in the contralesional part of space.

- Basic disturbance in these cases is an inability to summate a series of ‘spatial impressions’ - tactile, kinesthetic, visual, or auditory — a form of *amorphosynthesis*.
- The cause of the neglect: inability to disengage attention automatically from the intact region and direct it to the contralesional side.

- Can be extra-personal or peri-personal, within egocentric or object-based frame of reference.

Neglect

- A disorder of spatial directed attention
  - Neglect for visual, auditory, and somatosensory stimulation on one side of the body or space
    - Defective sensation or perception
    - Defective attention or orientation
  - Extinction to double simultaneous stimulation often present
  - During recovery patients go through *allesthesia*, begin to respond to the neglected stimuli as if they were on the other side of the body or space, and then *simultaneous extinction*

Contralateral Neglect

- Affected individuals fail to report, respond to, or even orient to stimuli presented to the side of the body (or visual space) opposite the lesion.

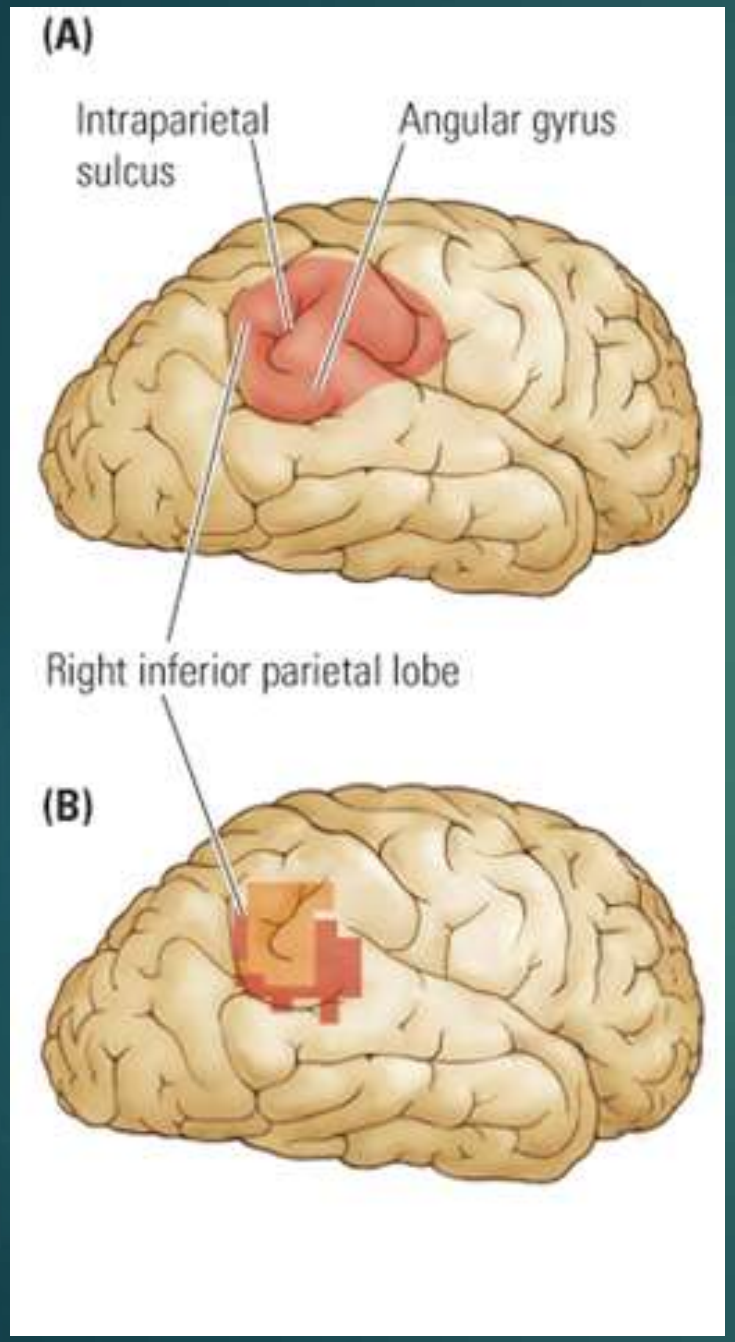
- Difficulty performing complex motor tasks on the neglected side, including dressing themselves, reaching for objects, writing, drawing, and, to a lesser extent, orienting to sounds.

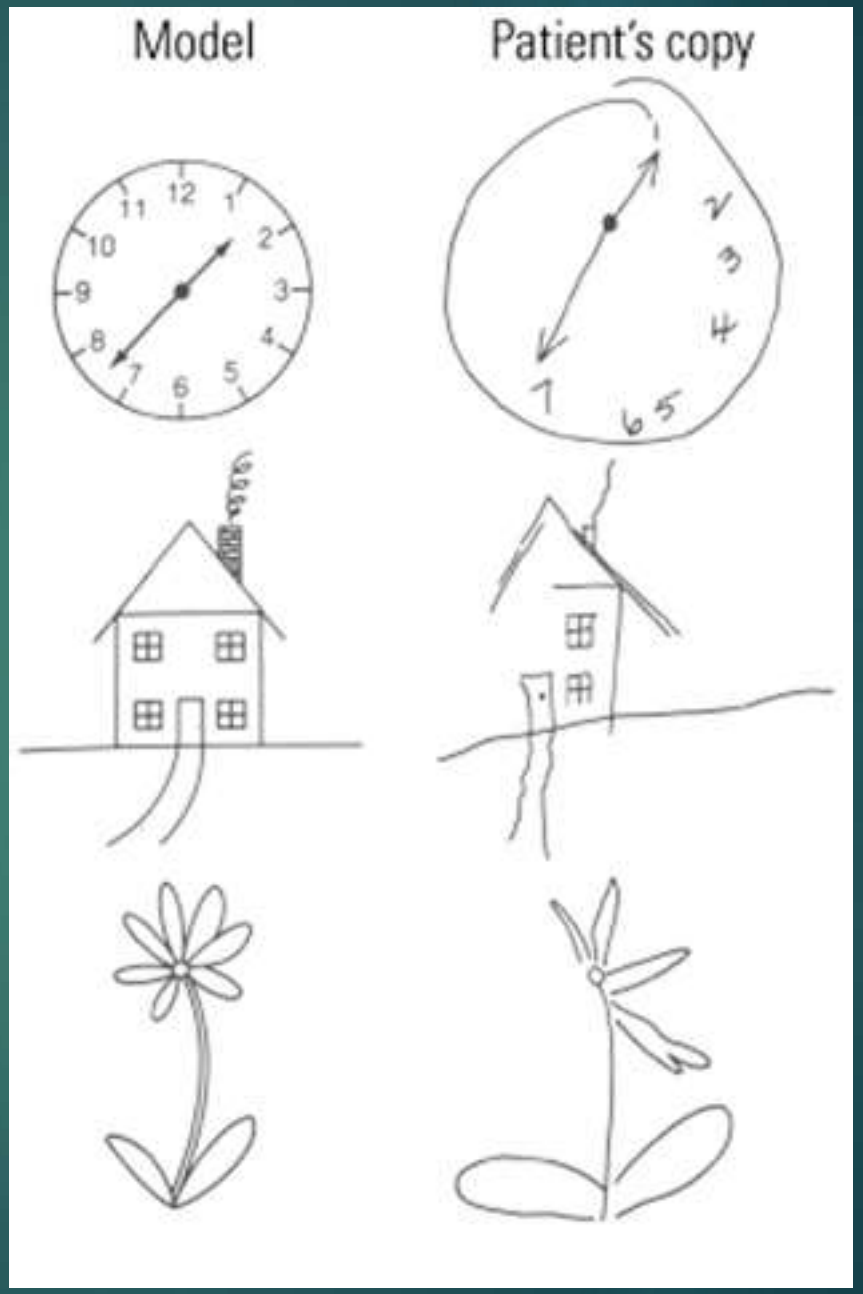
- The signs of neglect can be as subtle as a temporary lack of contralateral attention that rapidly improves as the patient recovers, or they can be as profound as permanent denial of the existence of the side of the body and extrapersonal space opposite the lesion.

Examples of Contralateral Neglect of the Body

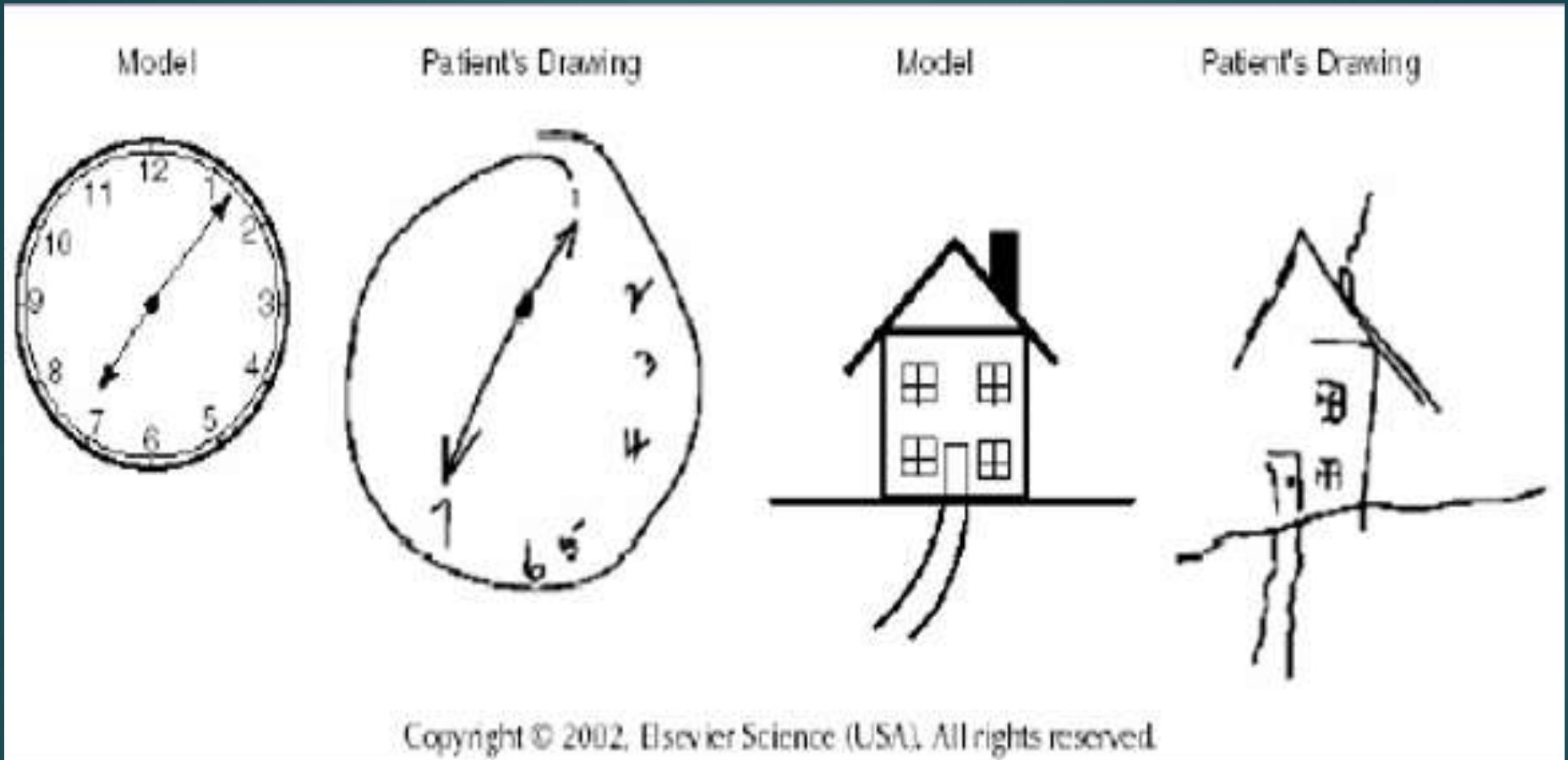
Neglect on one side of body in dressing and grooming.

- ✓ Shave only one side or use only one sleeve of shirt.
- ✓ Deviation of head and eyes to side of lesion.
- ✓ Torsion of body to the side of lesion.
- ✓ *Fail to use one side of body, even though paralysis is not present*
- ✓ Finds it impossible to wear eye glasses.


(A)
Intraparietal sulcus
Angular gyrus
Right inferior parietal lobe
(B)


Model
Patient's copy

Right parietal visual neglect



Critchley's Patients 1

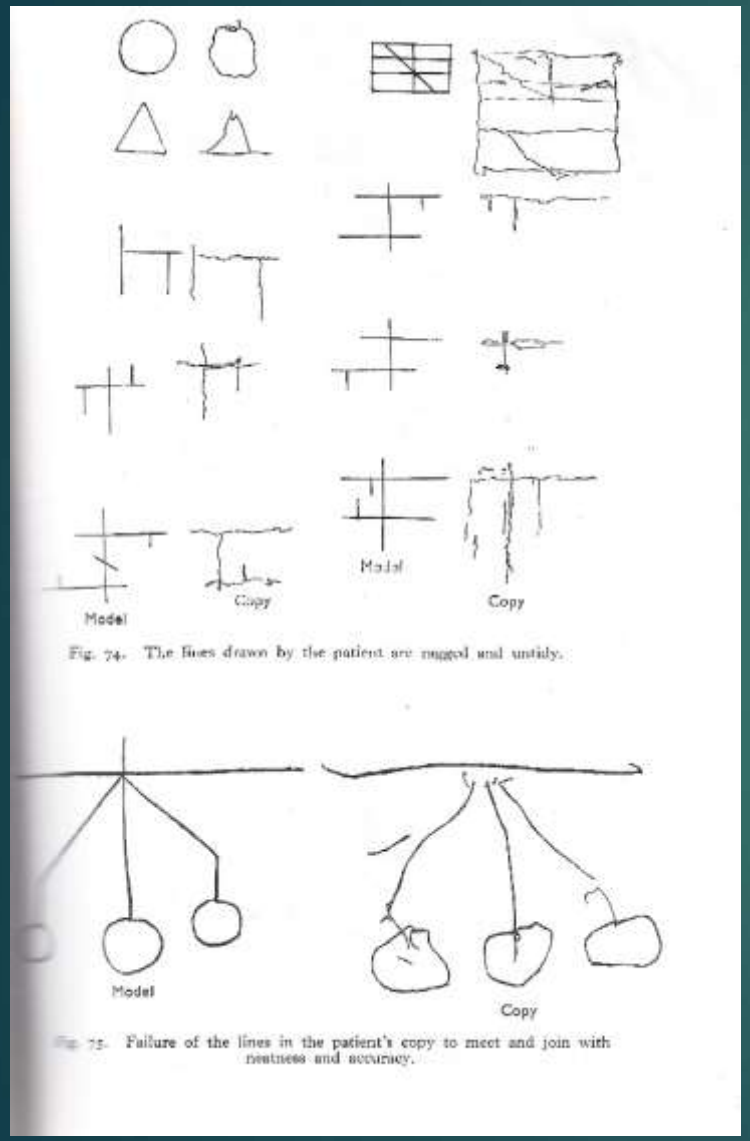


Fig. 74. The lines drawn by the patient are ragged and untidy.

Fig. 75. Failure of the lines in the patient's copy to meet and join with neatness and accuracy.

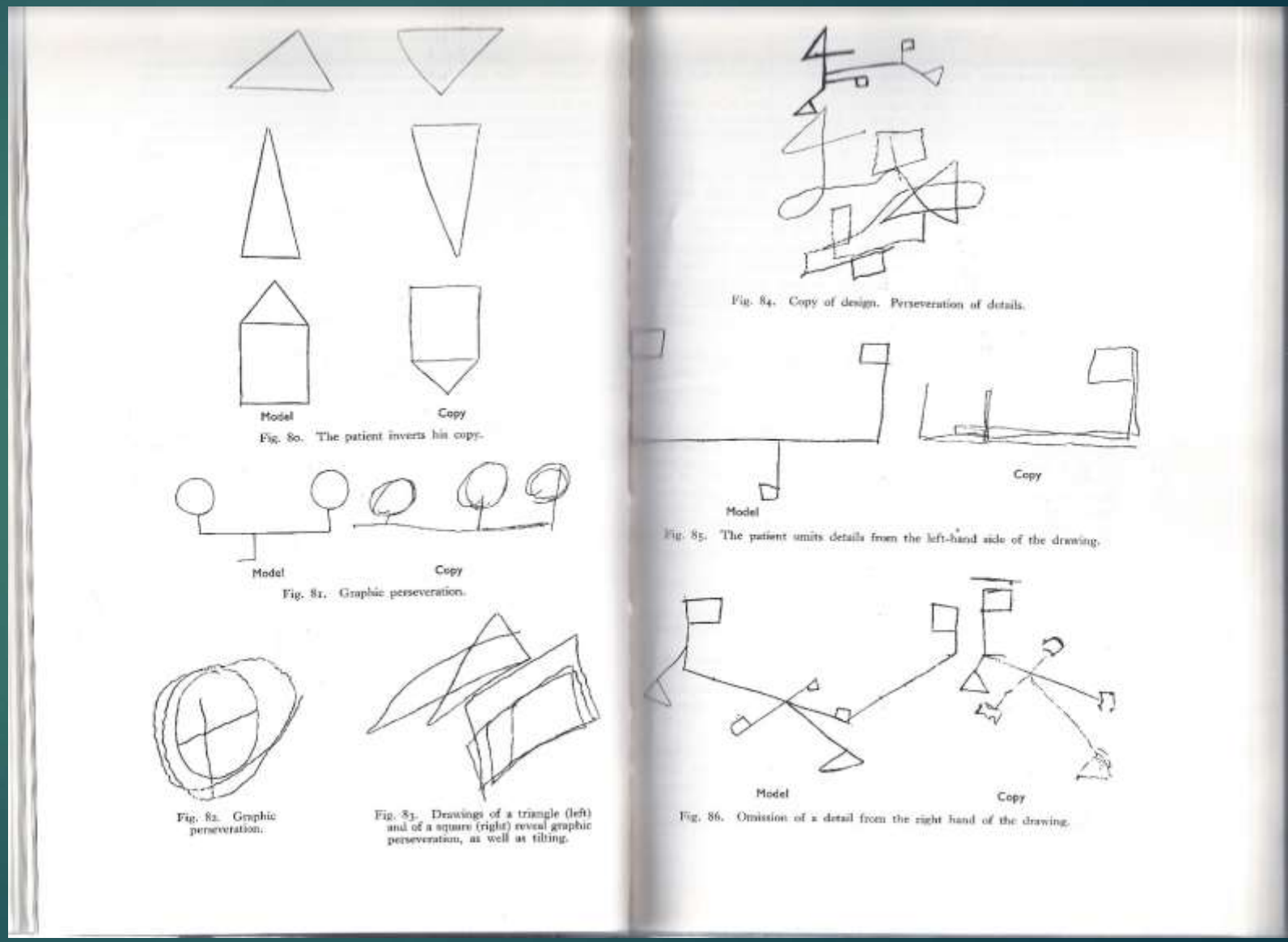


Fig. 80. The patient inverts his copy.

Fig. 81. Graphic perseveration.

Fig. 82. Graphic perseveration.

Fig. 83. Drawings of a triangle (left) and of a square (right) reveal graphic perseveration, as well as tilting.

Fig. 84. Copy of design. Perseveration of details.

Fig. 85. The patient omits details from the left-hand side of the drawing.

Fig. 86. Omission of a detail from the right hand of the drawing.

Critchley's Patients 2

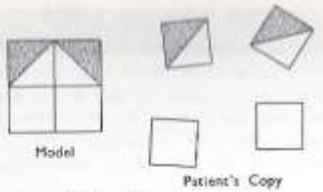


Fig. 90. Kohs' Blocks. The correct squares are selected, but an "open" square is constructed.

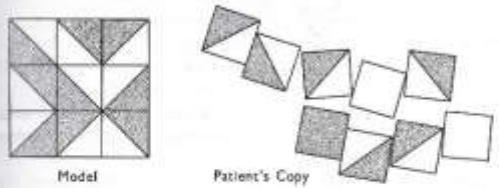


Fig. 91. Kohs' Blocks. The correct items are selected but the patient cannot put them together to form a symmetrical bi-dimensional square.

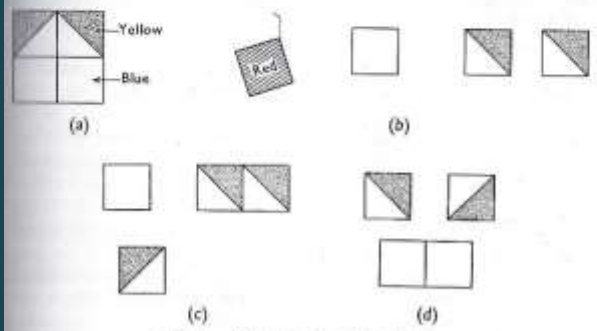


Fig. 92. Kohs' Blocks, design No. 3.
(a) Model. (b–d) Patient's three attempts. Left frontoparietal metastatic tumour.

Fig. 104. Drawing of a house, showing neglected left side and disorientation.

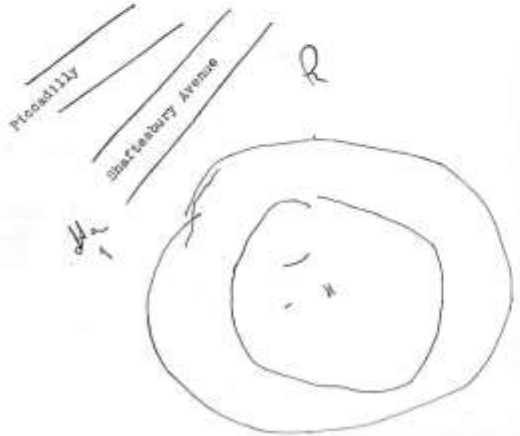


Fig. 105. Patient's drawing of Piccadilly Circus; note that the streets which should run in a radial fashion are depicted in an almost tangential manner.

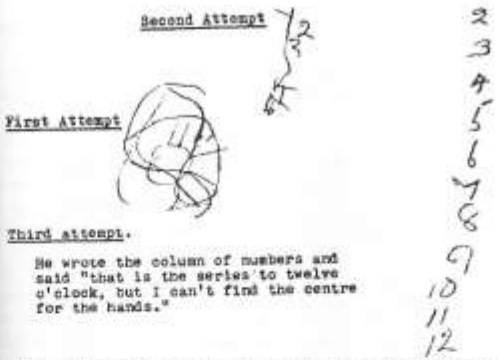


Fig. 106. The patient was instructed to draw a clock-face, complete with hands and numerals.

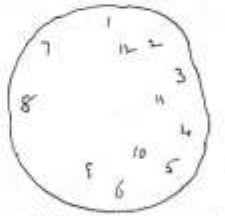


Fig. 107. The dial of a clock. The hands are omitted altogether and the numbers are arranged incorrectly.

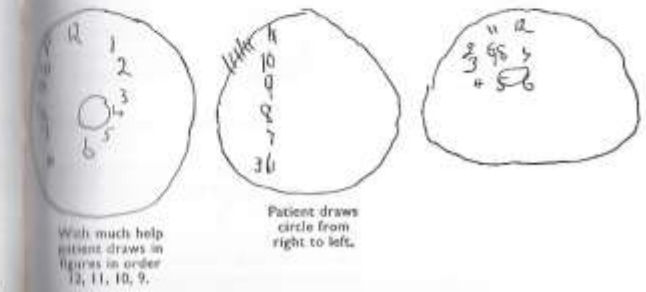
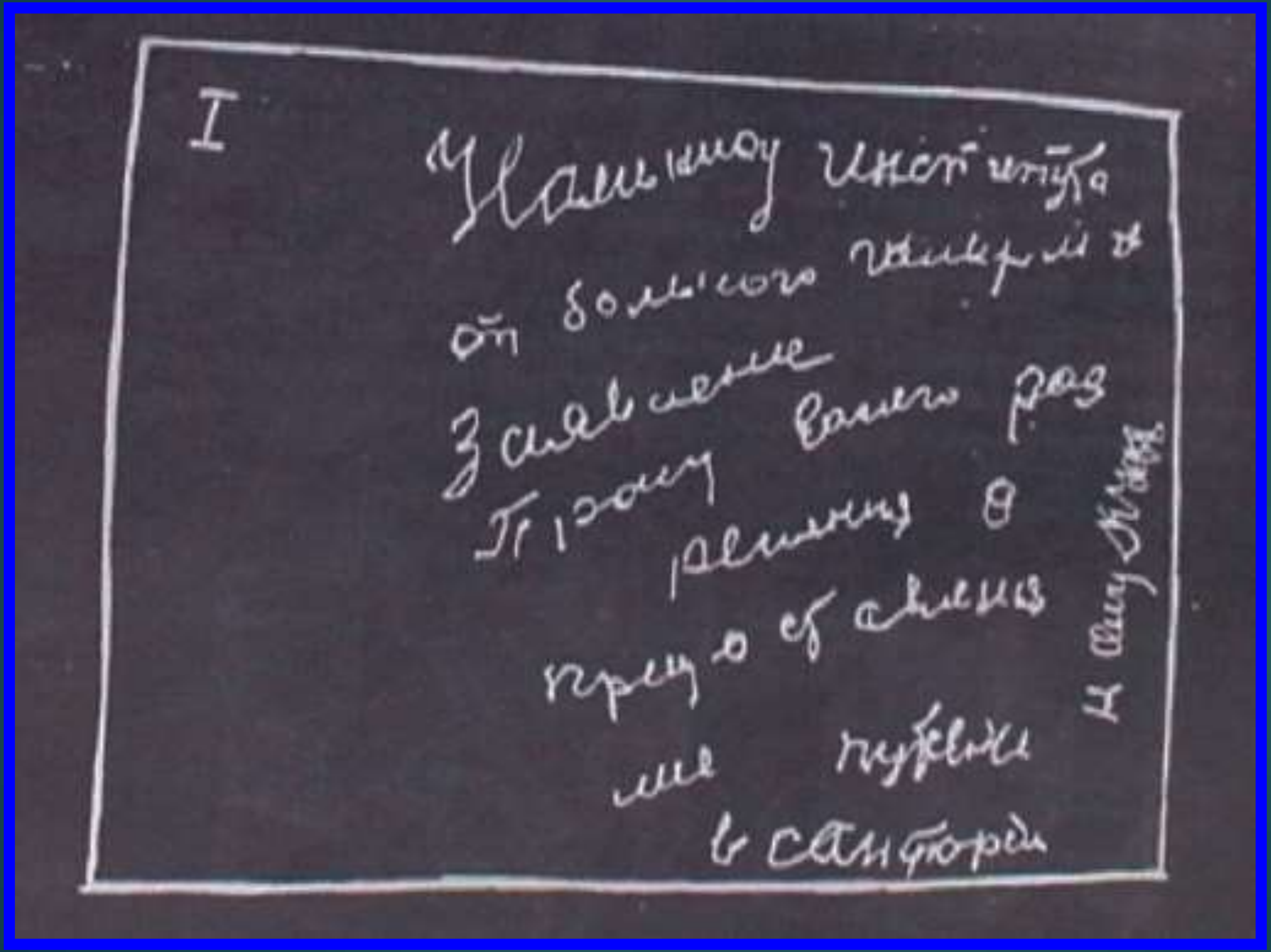
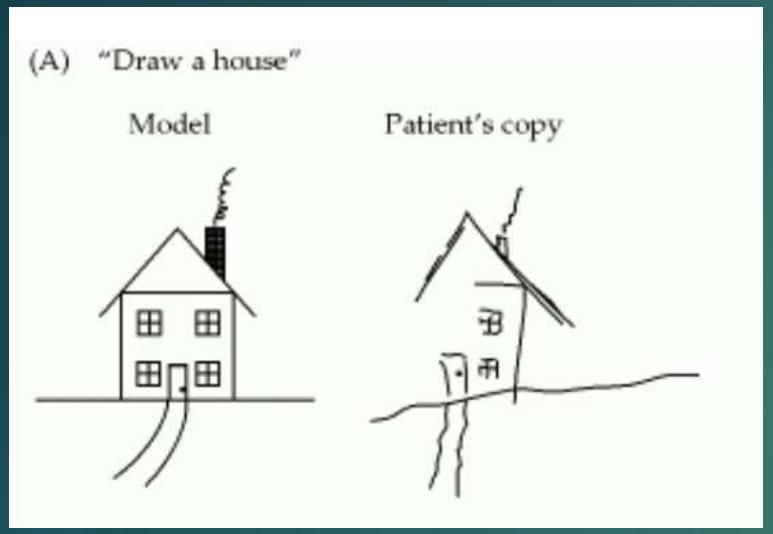
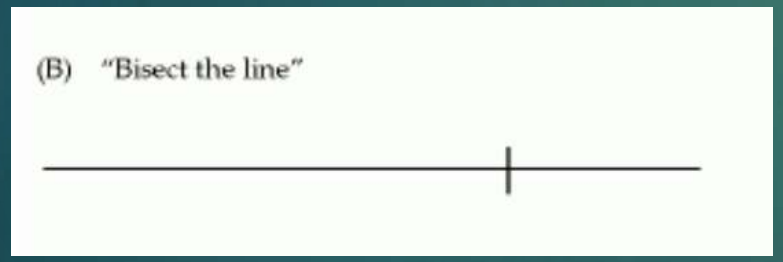


Fig. 108. Three attempts by the same patient at drawing a clock-face.


I
от больного
заявление
в санатори

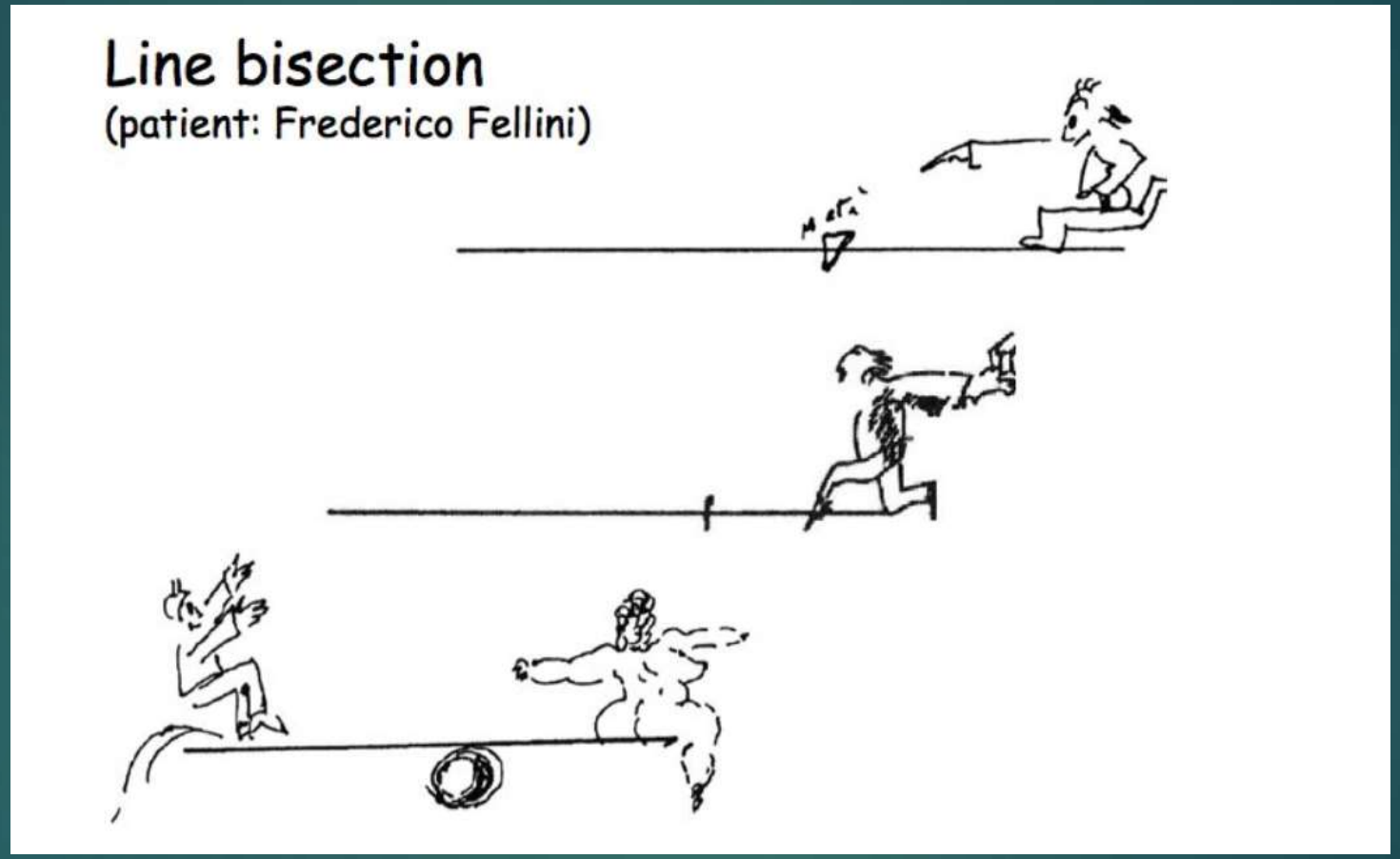
Classic Contralateral neglect



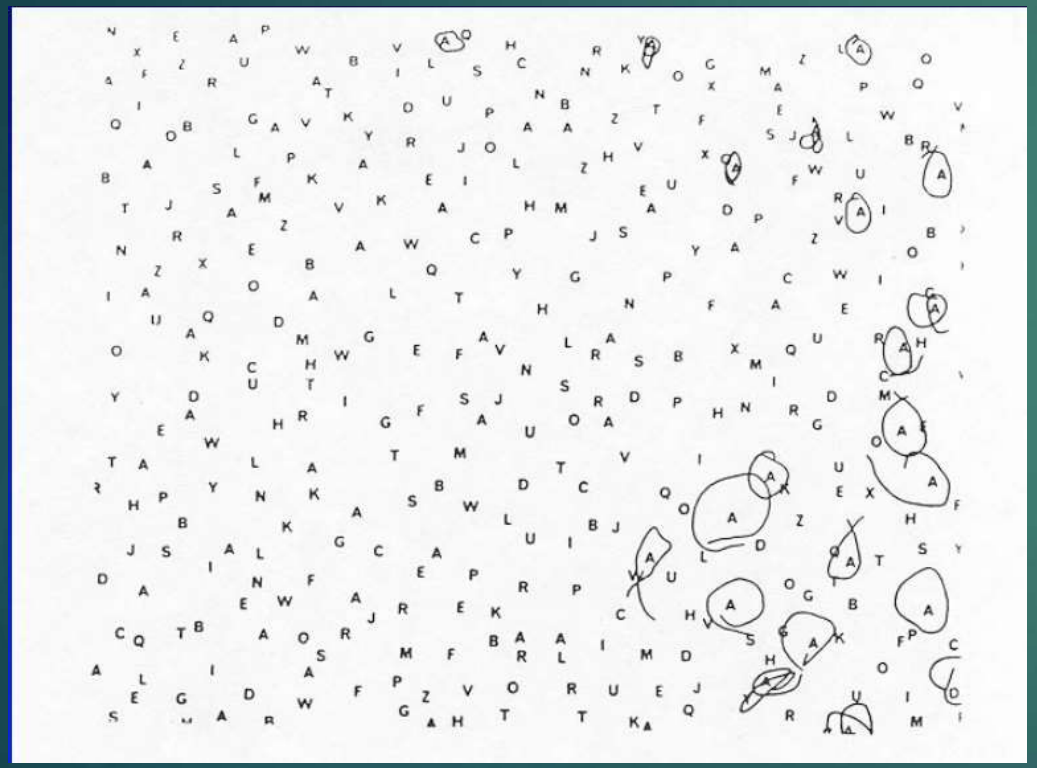


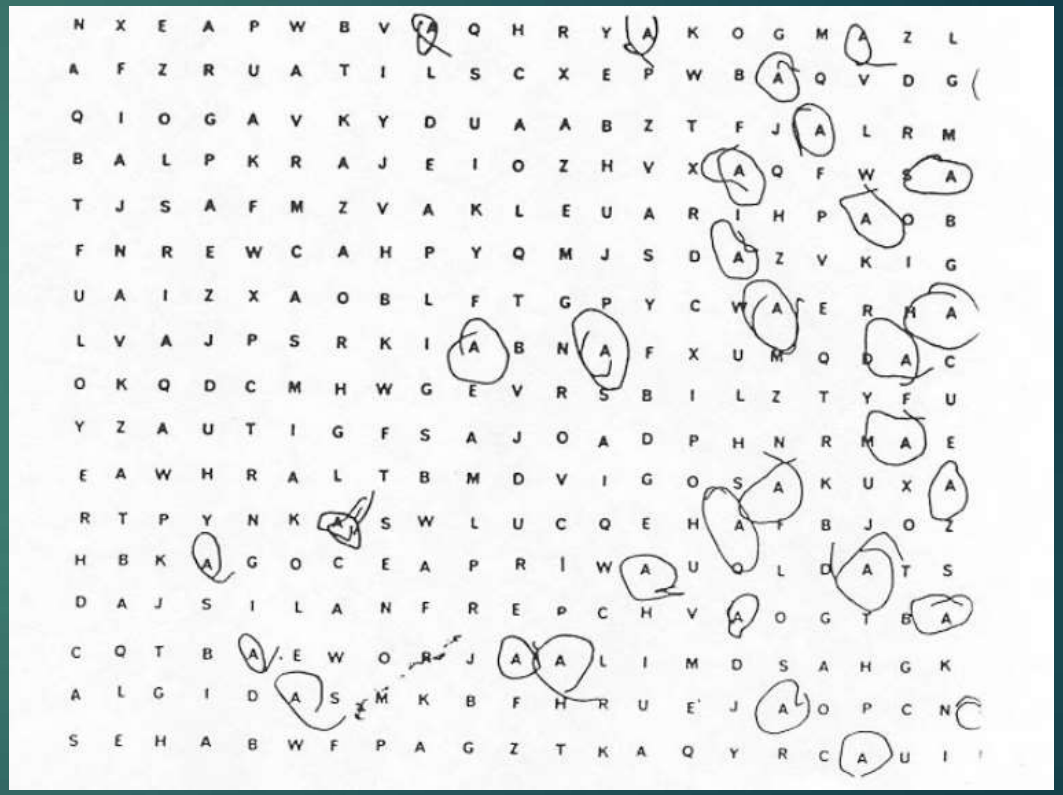
Artist Anton Raederscheidt over the period of his recovery from a right parietal stroke,

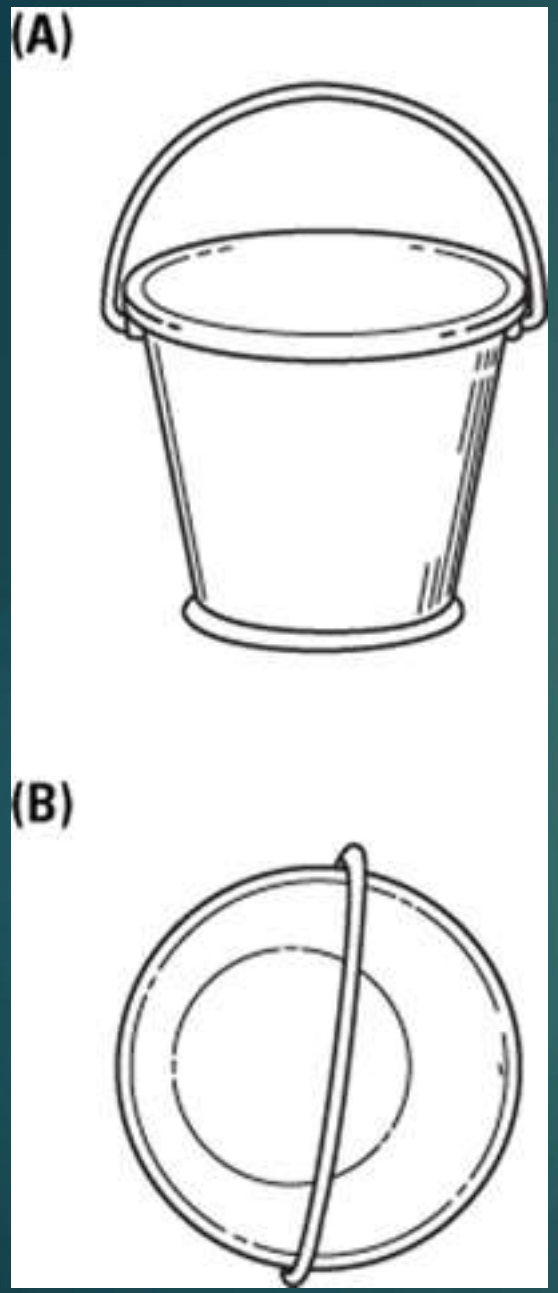
F. Fellini bisecting a line – left neglect



Left Neglect in Cancellation Task







Posterior Right Parietal Lobe Damage:

Impaired Object Recognition

Poor at recognizing objects in unfamiliar views

Tests for contralateral neglect

- Casual observation of pt.'s behavior.
- Drawings made by the pt.
- Line bisection
- Cancelation tasks
- Raven’s
- Block Design

Left Parietal Specialization for Motor Attention

- Left Parietal specialization for execution of and attention to movement
- Uniquely human; Primates prefer to use left hand for visually guided reaching & right hand to manipulate objects; Humans prefer right hand for both
- Deficits of sequencing motor actions & allocating attention to motor acts
- Examples:
  - Optic ataxia (inability to make accurate movements to objects with contralesional hand); LPC lesion – errors with R hand throughout entire visual field; RPC lesion – errors with L hand in L visual field
  - Apraxia (deficit in performing learned movements): results from LP damage and is deficit in motor attention
  - Only LH can generate voluntary facial expressions on command

Clock Drawing Test: RP and LF

- RP lesion: Problems with spatial organization and proper placement of numbers
  - impaired visuospatial processing in the SMG RP group
- LP lesion: problems with proper placement of clock hands (time-setting)
  - “10 after 11” is language and grammar reference
  - impaired language processing in the inferior LF group.

D. Tranel, et al., 2008

Clock Drawing

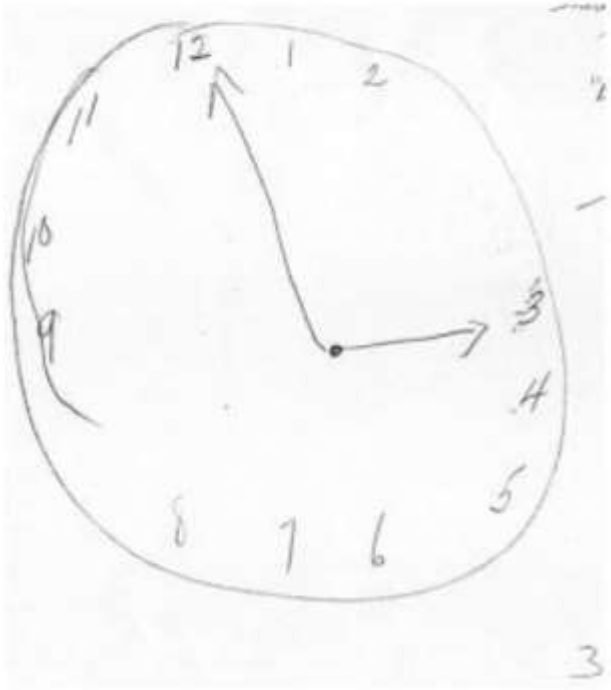
- RP: associated with CDT, WAIS Block Design & Facial discrimination;
- LF: assoc. with BNT, Cowat
- RP parietal lesions: typically occurs in the acute phase of brain injury, within hours or a few days of lesion onset, and then shows rapid recovery.

D. Tranel, et al., 2008

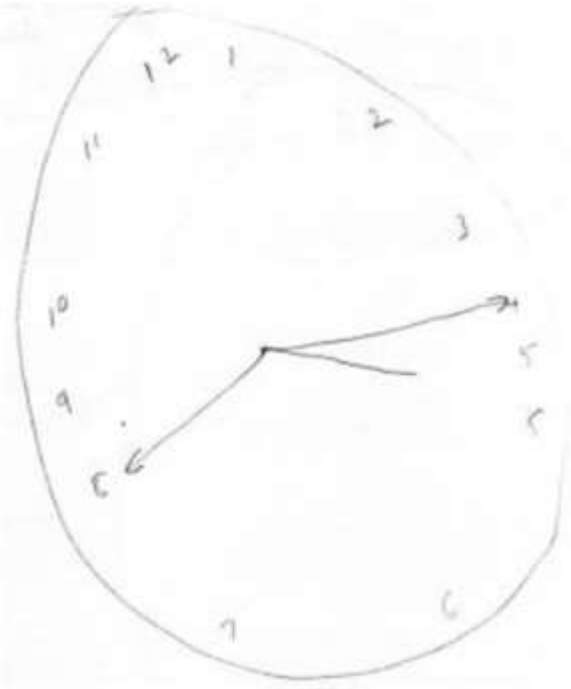
SMG RP: Impaired Spatial organization & number placement

A. Impaired spatial organization and number placement

A.1*



A.2



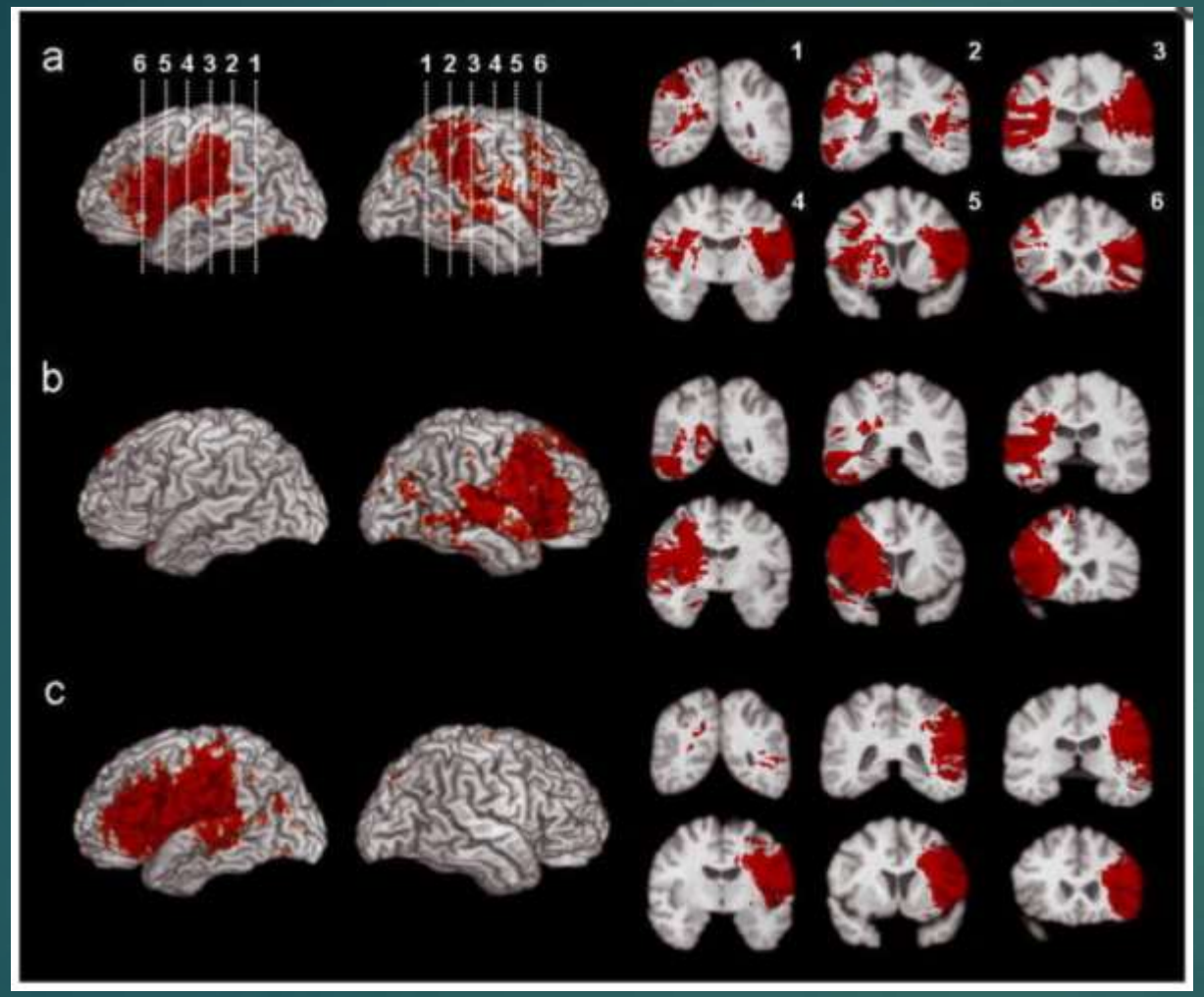
Inferior LF: Impaired time setting

B. Impaired time setting

B.1

B.2

Clock Drawing



(a) Maps for impairments irrespective of error type;

(b) Right: Maps for deficits in spatial organization and number placement

(c) Left LF: Maps for impairments in time setting

Cortical Sensory Syndromes

- ASOMATAGNOSIAS
- APRAXIAS
- VISUAL DISORDERS
- AUDITORY NEGLECT

The Apraxias

True Apraxias per Liepmann:

1. Ideomotor apraxia *
2. Ideational apraxia *
3. Buccofacial apraxia *

---

1. Constructional apraxia: visuospatial orientation
2. Dressing apraxia: *form of sensory extinction and loss of extra personal space*

Apraxia

- Apraxia: Impairment of the ability to carry out purposeful, skilled movements despite normal primary motor skills and normal comprehension of the act to be performed

- Interaction of abstract knowledge related to tools, objects, and actions & motor ability (praxis production)
- Ideomotor Apraxia (praxis production deficit; lack of representation & recognition of actions))
  - Inability to carry out a commanded movement; but can use the actual object (i.e. flip a coin)
- Ideational Apraxia (conceptual deficit)
  - Loss of ability to conceptualize, plan, and execute the complex sequence of motor actions involving the use of tools or objects in everyday life (i.e. prepare a letter for mailing)

Apraxia

- Cannot be explained by weakness, incoordination, sensory loss, impaired concentration, inattention, or intellectual impairment
- Can appear as an isolated disorder, but its common association with aphasic syndromes makes it difficult to differentiate from impaired comprehension
- Can manifest in relation to focal lesions, progressive degenerative disorders, or stroke
- Frequently associated with:
  - Anosognosia: pts may be unaware of their apraxic deficits,
  - Often have right hemiparesis, so they mistake their apraxic deficits with difficulty moving the affected limb

Apraxia: The Cognitive side of motor control

- Cognitive nature of apraxia comes to the fore most clearly in three manifestations of apraxia that are exclusively bound to left-hemisphere damage: imitation of meaningless hand postures, use of single mechanical tools, and pantomime of tool use.
- Their functional communality is a central role for segmentation and combination.
  - For imitation, visual features of the demonstrated gesture are segmented into distinct body parts which are combined for reproducing the posture.
  - For tool use the structures of tool and recipient are segmented into functionally significant traits which are combined to form mechanical chains,
  - For pantomime the compound image of hand, action, and object is segmented into distinctive features of the object and the acting hand which are combined to form a comprehensible image of the object and its use.

Disorders of Apraxia: Left PPC

- Lipmann: Left hemisphere controls praxis
- Left inferior PC controls programming of sequential action
- Sensory areas 5 and 7 in dominant parietal lobe, supplementary and premotor cortex of ***both*** cerebral hemispheres and their integral connections.
- Localizes generally to left IPC (sensory area) and frontal lobe systems, particularly the premotor cortex, supplementary motor area, and convexity, and as a result of disconnection of corpus callosum.

Testing for apraxia

- Pantomime of object use: show me how you would brush your teeth; watch me brush my teeth, then you do it
- Tests designed to evaluate transitive movements (those done as a goal-directed movement with an object): Here is a hammer. Show me how to use it.
- Tests of intransitive movements (those done without a specific goal and without use of an object, like hand gestures)
- Tests of ability to carry out serial acts (pretending to prepare a cup of tea or light a pipe)

Ideomotor Apraxia *("how to do")*

- Most common type of apraxia
- Impairment in imitating gestures, pantomiming tool use and making meaningful gestures to command, especially if the gestures require a series of sequential movements, or to imitate, although spontaneous production of the gestures may remain intact
- During pantomime, a patient with ideomotor apraxia will often use a body part as if it were an object (e.g., their hand as a "comb").
- Involved in tasks that require a sequence of movements & rapid reorienting of motor attention
- Usually involves lesions in the left IPC or supplementary motor area or lesion in the corpus callosum
- If movement is not in natural context, more left IPC

Buccofacial Apraxia

- Difficulty performing voluntary skilled motor movements of the face, tongue, lips, and cheeks on command.

- Testing:
  - intransitive buccofacial gestures include puffing out cheeks, sticking out tongue
  - intransitive buccofacial gestures include pretending to suck on a straw or sniff a flower

Testing Ideomotor Apraxia

i. Buccofacial apraxia (blowing a match)

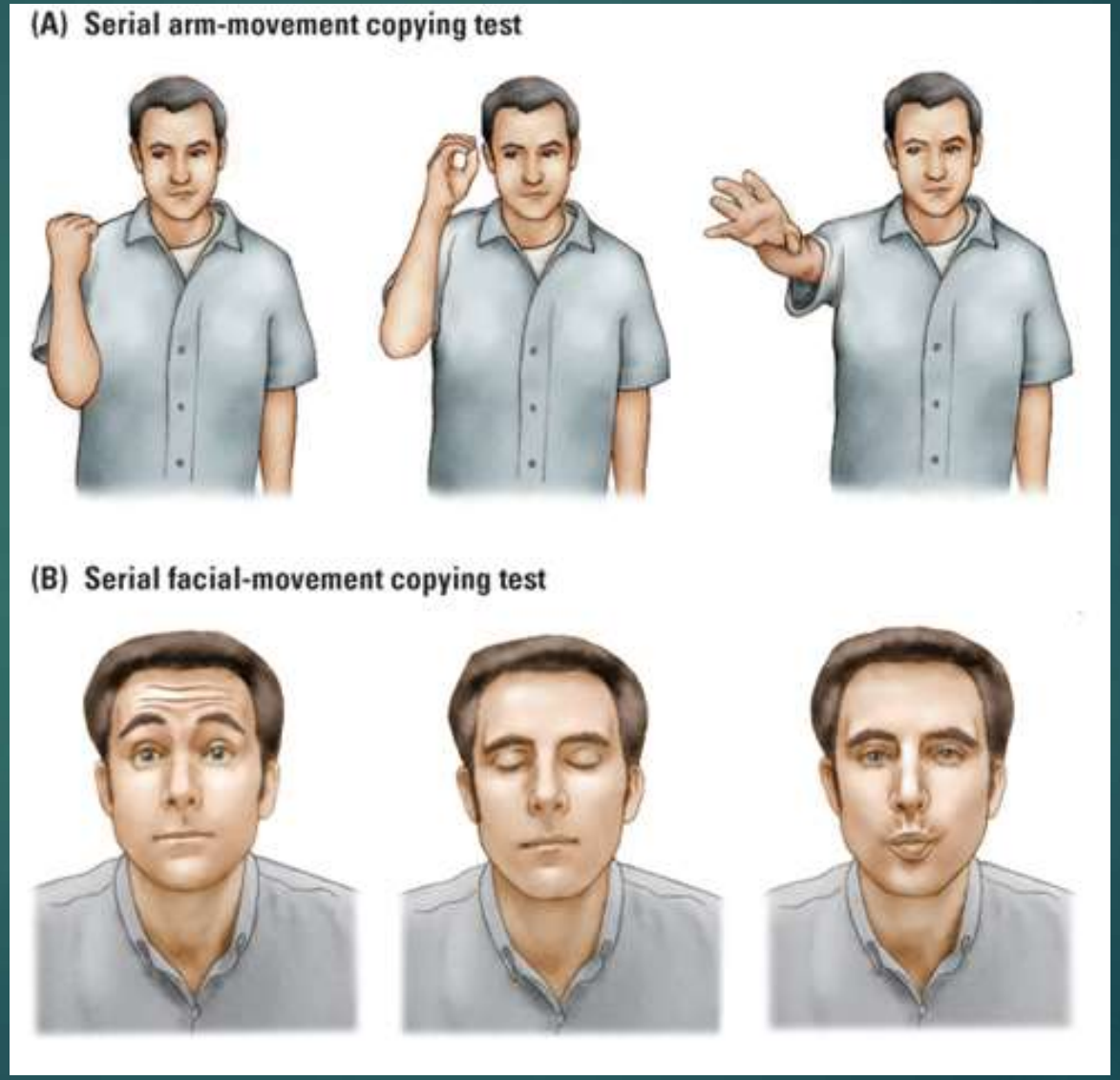
ii. Limb apraxia (flip a coin , comb hair)

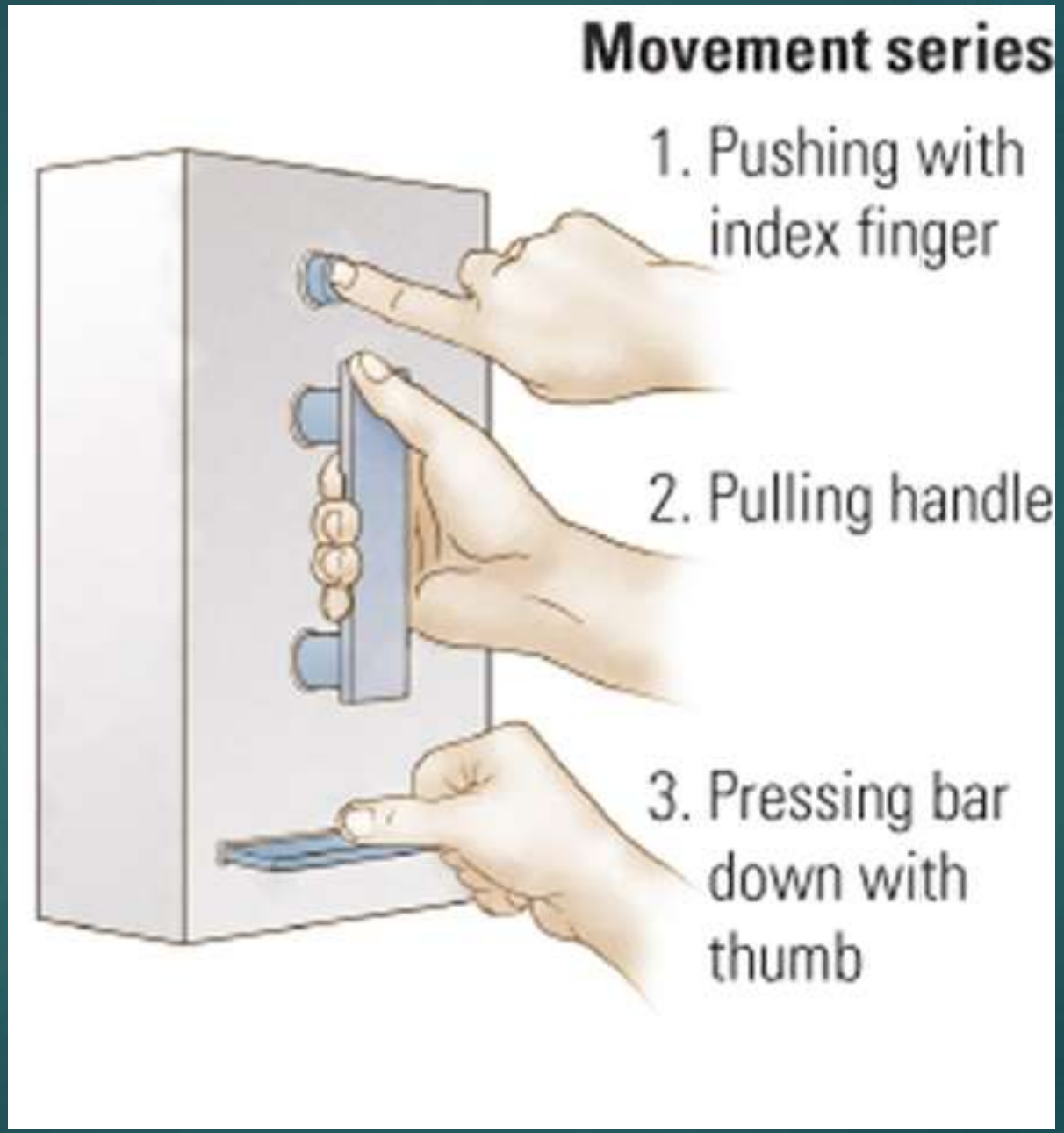
- Intransitive limb gestures: waving or saluting,
- Transitive limb gestures: pretending to use a comb or a scissors.

iii. Whole body apraxia (stand like boxer)

- Commands to be alternated b/w right and left limbs

Testing Ideomotor Apraxia




Movement series
1. Pushing with index finger
2. Pulling handle
3. Pressing bar down with thumb

Ideational Apraxia *("what to do")*

- Inability to carry out a series of acts involving use of an object; failure to correctly sequence a series of acts leading to an action goal; i.e. prepare a letter for mailing; can do elements, but not sequence

- A conceptual apraxia: loss of different types of tool-action knowledge.

- Exhibit content errors in performance of transitive movements (i.e. uses toothbrush as if it were a shaver); cannot associate tool and object with corresponding action or cannot associate tool with object of their action (show nail, uses scissors)
- Often co-occurring aphasia
- Causes life disability because use tools inappropriately, misselect tools, don't complete tasks
- Only Parietal lesions lead to defective tool use (ability to infer function from structure)

Ideational Apraxia: Serial action

- Loss of the ability to plan and execute complex gestures, as though one has lost the “idea” behind the gesture or use of a tool even though knowledge about the use of the tool is unaffected.

- Involves problems in motor planning and is manifest in errors in sequencing the necessary actions for a task
  - Example: lighting a pipe before putting in the tobacco.

- Assessment of serial acts is important for identification of this disorder.

- Associated with bilateral, nonfocal lesions and with left hemisphere lesions, especially the posterior temporal-parietal junction.

Praxis testing (done in an order)

1. Observe the actions – shaving, dressing, eating.
2. Carry out familiar acts – blow a kiss, wave goodbye.
3. Imitate the examiner (‘do this after me’)
4. How to use objects (pantomime)

    a) simple acts – hammer nail, comb hair .

    b) complex acts – light and smoke cigar; open soda bottle, pour in glass and drink.
5. Demonstrate use of actual items

(both limbs and orofacial commands to be asked)

Apraxia of Speech

- Not a language disorder
- An impairment in planning the movements necessary for speech production.
- Both acquired and childhood versions can be manifested in inconsistent articulation errors and difficulty with correct articulatory placement.
- A disorder of the planning and organization of articulatory movement, in contrast to *dysarthria*, which is a disorder of motor coordination.
- Localization is uncertain

Constructional Apraxia

- Constructional ability/praxis = visuoconstructive ability - high level nonverbal cognitive function.
- Perceptual motor ability involving integration of occipito – parieto – frontal connections.
- Non dominant parietal lobe is important for this.
- Inferior PC (kinesthetic analysis of visual patterns)

Tests of constructional ability

- Reproduction of drawings:

  given in order of complexity.

Testing Constructional Apraxia

- Drawings to command :
  1. draw a clock with 10:20 time
  2. draw a 2D figure - daisy in a pot
  3. draw a house – in way you can see two sides and the roof.

- Block designs:
  - Left sided lesions – simplification of complex diagrams, loss of details
  - Right sided lesions – loss of gestalt, rotation of diagrams

Drawing: Right Parietal deficits:

Loss of gestalt of whole: Scattered , fragmented

Loss of spatial relations

Faulty orientation

Energetic drawing

Addition of lines to make drawing correct

Drawing: Left Parietal deficits:

Gross lack of details

Coherent , simplified

Preservation of spatial relations

Correct orientation

Slow & laborious

Dressing Apraxia

- Not a true apraxia.
- Combination of spatial disorientation and visuospatial inattention.

Agraphia

- Written language disorder often associated with aphasia and alexia.
- It can be related primarily to motoric and spatial deficits
- Spontaneous writing & writing on command more affected than copy righting
- Irregular & tremulous script, misspelling , semantic & syntactial errors
- Site – inferior parietal cortex

Agraphia

- Apractic agraphia- agraphia despite normal sensory, motor & visual feed back, word & letter knowledge
  - Lesion- Dom (L) superior parietal lobule
- Visuospatial agraphia- neglect of right side of paper in writing
  - Lesion -- (R) temporoparietal junction

- Agraphic writing disorders can include writing impairments due to impaired sensory feedback, impaired written production secondary to errors of omission, or motor/sensory deficits, impaired letter formation, and impaired writing as a result of spatial deficits manifested in problems such as poor spacing and poor use of the space.

Recent Research of Parietal Involvement in Memory Functioning

- Researchers have found evidence linking the Parietal lobe to memory
- Different types of memory:
  - Visual short term memory
  - Working Memory
  - Episodic Memory

- Working Memory Index (WAIS3) maps to left posterior frontal and parietal cortex, not PFC (Glascher et al., 2009)

Cognitive Functions of ventral Prefrontal Cortex (or Inferior PC)

- vPC is lodged between the ventral perceptual stream and the dorsal action stream; VPC is comprised of the supramarginal gyrus (SMG) and the angular gyrus (AG). Temporo-parietal junction (TPJ) is part of SMG.

- VPC activations for many different cognitive functions:
  - perceptual and motor reorienting,
  - working memory
  - evaluation of relevant vs irrelevant distractors
  - language: semantic processing during word comprehension, discourse incoherence
  - number processing: conditions that require the retrieval of numerical facts, exact vs approximate calculations
  - theory of mind
  - episodic memory encoding and retrieval
  - confidence during recognition memory

Roberto Cabeza, et al., Trends Cogn Sci. 2012

Working Memory and Angular Gyrus

- WM deficits have been reported following AG lesions, with verbal and spatial short-term memory being impaired following damage to the left and right side, respectively.

- Although these deficits were initially attributed to impaired phonological or visuospatial WM buffers, more recently they have been linked to impaired attention whose operation is needed to activate and sustain the long-term memory representations that constitute the contents of WM

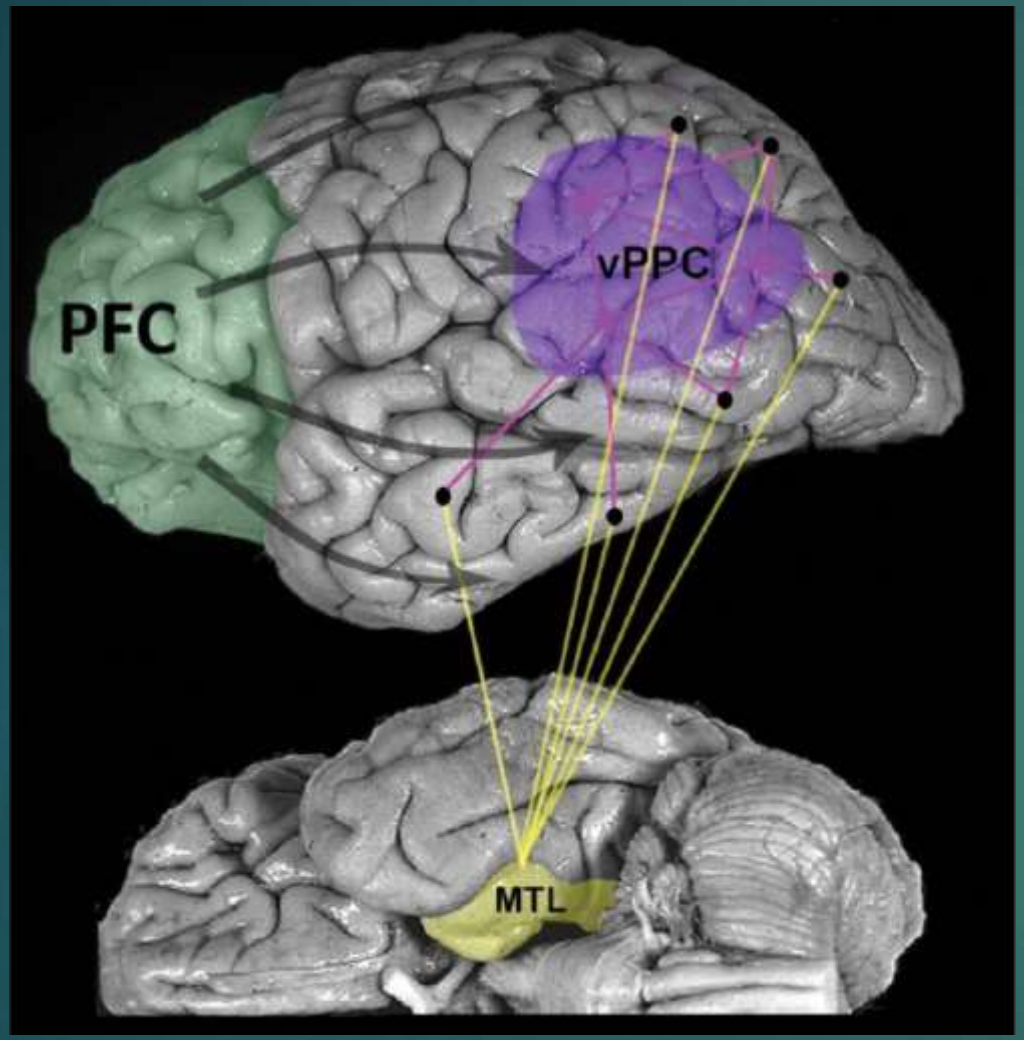
Function of vPC: Bottom-up attention (BUA) theory

- BUA hypothesis emphasizes the role that attention plays across all domains. Prefrontal cortex and ventral posterior parietal cortex are intricately connected.
- Bottom-up attention (BUA) hypothesis: VPC activity reflects the capture of bottom-up attention by information entering WM either from senses or from long-term memory.
- VPC activity is greater for recollection than for familiarity, for problems with known than unknown answers, increases with the amount of information recollected, and is stronger for high- than low confidence “old” responses
- Allows unexpected, but relevant information, to capture attention during goal directed action
- VPC activity is greater for retrieval than calculation strategies

VPC lesion effects: Memory neglect

- VPC lesion patients spontaneously reported fewer details in their autobiographical memories but they were able to provide the missing details when prompted
- VPC lesion patients subjectively rated their memories as impoverished but were able to recall source memory information when specifically questioned.
- This is analogous to the neglect syndrome, which does not affect perception per se but bottom-up attention to percepts.
- Memory deficits in VPC patients may be described as memory neglect.

Ventral PPC is Convergence Zone for Episodic Memory



Episodic retrieval starts by top-down search of stored event features in PFC. Search is facilitated by relational bindings in the MT. The ventral posterior parietal cortex (vPPC) acts as a convergence zone that links event features within the neocortex.

Activation usually bilateral but are more frequent in left VPC

Activations tend to increase as a function of recollection and confidence

Tracks the relevancy of recognition responses or cues & violation of expectations during retrieval rather than memory recovery per se.

Shimamura A P Current Directions in Psychological Science 2014;23:257-263

Episodic Memory and VPC

- VPC activity is associated with success during retrieval and with failure during encoding

- While VPC activations are generally associated with successful performance, in the episodic encoding domain they are associated with failed performance.

- In contrast with medial temporal lobe and ventrolateral prefrontal cortex, which typically show greater activity for subsequently remembered than forgotten items, VPC usually shows greater activity for subsequently forgotten than remembered items.

Cortical Sensory Syndromes

- ASOMATAGNOSIAS
- APRAXIAS
- VISUAL DISORDERS
- AUDITORY NEGLECT

Posterior Parietal: Object location/recognition

- Object recognition
  - Viewer centered object identification
    - Determines the location, location orientation and motion of an object

Spatial Navigation: Guidance of Movement

- Spatial Navigation
  - Relative position of the body
  - Cognitive spatial map
    - Route knowledge, unconscious knowledge of how to reach a destination
  - Medial parietal region (MPR)
    - Neurons show responses associated with making a specific movement at a specific location

Disorders of Spatial Cognition

- Mental rotation requires:
  - Mental imaging of the stimulus
  - Manipulation of the image
- Left PC deficit: inability to generate the image
- Right PC deficit: inability to manipulate the image
- Inability to use topographic information is associated with right parietal damage

White-Matter Organization and Spatial Cognition

- Mental transformations are carried out by the posterior parietal cortex
- Noted sex difference in the ability to perform mental transformations of objects
  - Men outperform women
- Mental rotations: white-matter organization near the anterior part of the intraparietal sulcus

Parietal Visual Disorders

- Incongruous homonymous *hemianopia or an inferior quadrantanopia*. (in practice, the defect is complete or almost complete and congruous) - Inferior part of the parietal lobe –

- Abolition of optokinetic nystagmus with target moving toward side of the lesion - Deep lesions -

- Left sided *visual neglect* - Right angular gyrus -

- Topographagnosia - visual disorientation and loss of spatial (topographic) localization. Pts are unable to orient themselves in an abstract spatial setting.

Parietal Visual Disorders

- Others –

  Deficits in localization of visual stimuli.

  Inability to compare the sizes of objects.

  Failure to avoid objects when walking.

  Inability to count objects.

  Disturbances in smooth-pursuit eye movements

  Loss of stereoscopic vision.

- "Spasticity of conjugate gaze" : eyes may deviate away from the lesion on forced lid closure.

- Optic ataxia : in reaching for a target, movement is misdirected and dysmetric. (distance to target is misjudged)

Geographic Orientation

- Geographic orientation is function of parietal lobe and its multimodal association area.

- Combination of processes – spatial orientation, right-left orientation, visual perception and its memory.

Tests for geographic disorientation

1. Get history from relatives :

   Does he becomes lost in work?

   Does he have difficulty in orienting to new environment?

2. Localizing places in maps:

   Adequate literacy level and historical knowledge is necessary.

   ex : to locate cities or states on maps.

3. Ability to orient self in hospital:

   By observing the pt's capacity to find their bed, ward and bathroom.

Cortical Sensory Syndromes

- ASOMATAGNOSIAS
- APRAXIAS
- VISUAL DISORDERS
- AUDITORY NEGLECT

Auditory Neglect

- This defect in appreciation of the auditory left side of the environment is less apparent than is visual neglect.
- Many patients with acute right parietal lesions are initially unresponsive to voices or noises on the left side.
- Main lesion usually lies in the *right superior lobule*.

Newer findings about Parietal Lobe

Precuneus: Newest brain area in H. sapiens

- Medial area of the superior parietal cortex
- 3 parts: Sensorimotor Anterior Region, Cognitive/Associative Central Region (executive functions, working memory and motor planning), Visual Posterior Region

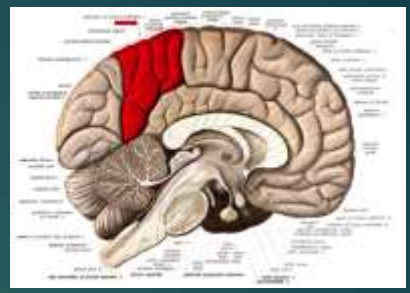
- Spatial enlargement of the deep parietal areas is the major morphological difference between modern and non modern human brains.

- More highly developed (i.e. comprises a larger portion of the brain volume) in human beings than in non-human primates or other animals; latest part of brain to develop in evolution

- This parietal bulging was localized in a very early post-natal period, in a stage which is absent in chimpanzees or in Neandertals.

- One of last regions to be myelinated.

Precuneus



- Self Referential Mental imagery/Self consciousness (anterior precuneus): rating ones own personality traits compared to those judged of other people.

- Episodic memory (posterior precuneus & inferior PF):
  - recall of past memories of self;
  - source memory (contextual associations): decides whether context information vs perceptual features exists for PF recall

Precuneus

- Visuospatial imagery: attention in space when in motion

- Mind reading: taking a third-person versus first-person visual point of view. Together with PF the precuneus is activated when people make judgments that requires understanding whether to act out of empathy and forgiveness.

- Consciousness (with Posterior Cingulate): cerebral glucose metabolism has its highest levels in these two areas in wakefulness but is most reduced during anesthesia in them; most deactivated during slow-wave sleep

- Default Mode Network central (ventral Precuneus): highest resting metabolic rates; day dreaming

P-FIT Model of Intelligence

- *Parietal-frontal network* is implicated as central to:
  - abstract human intelligence,
  - fluid intelligence (*Gf*)
  - working memory
  - attentional control

Autism: Parietal Advantage in detail focus

- The brain of those with autism also shows advantages. When some people with this condition are asked to complete detail-oriented tasks, such as finding a target shape in a design, they are quicker and more accurate.

- Additionally, those with autism generally exhibit less activity in the posterior parietal cortex, involved in visual and spatial perception, which suggests that their brain is performing the task more efficiently

FTD and PC: PGRN protein abnormality

- Study: All patients exhibited a clinical and radiologic phenotype compatible with frontotemporal lobar degeneration based on current consensus criteria.

- Parietal deficits, consisting of dyscalculia, visuoperceptual /visuospatial dysfunction, and/or limb apraxia, were a common feature, and brain imaging showed posterior extension of frontotemporal atrophy to involve the parietal lobes.

- **Conclusion** Parietal deficits may be a prominent feature of *PGRN* mutations and that these deficits may be caused by disruption of frontoparietal functional pathways.

When/Timing pathway: Right Parietal event timing

- Classic theory of two processing pathways of semantic (what) and spatial/action-oriented (where) information,
- Third Pathway: RTPJ activation increases when we need to temporally sequence information. TPJ is a crucial component of the "when" pathway.

- Battelli, 2008: Right inferior parietal lobe underlies this analysis of event timing.
- Judgment of temporal order, simultaneity and high-level motion are all compromised following right parietal lesions

Battelli L., 2008; Davis et al., 2009

Three pathways: Where, When, What

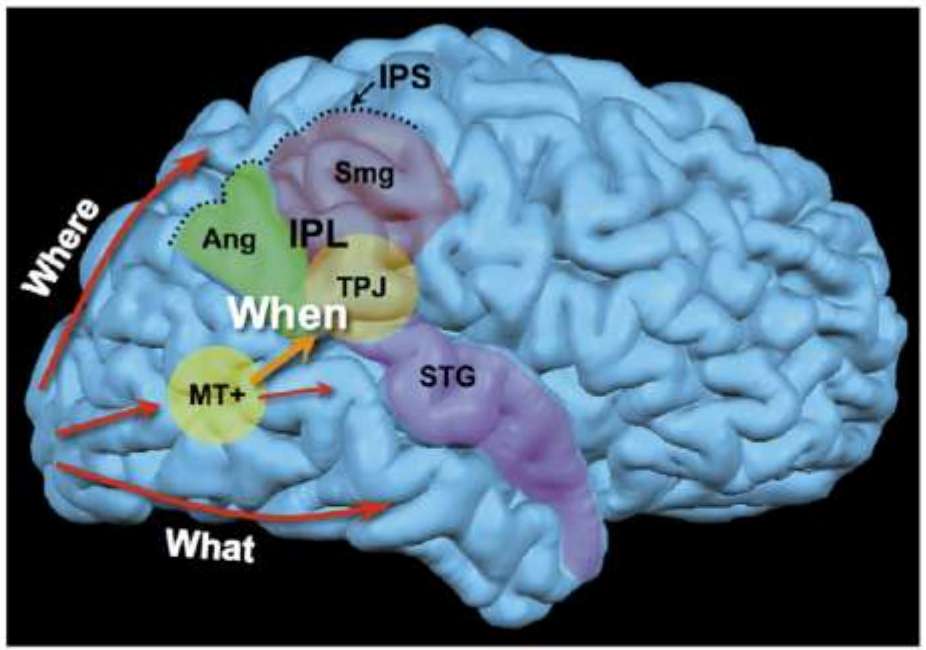


Figure 3. The *when* pathway. The *when* pathway is represented in the brain. This pathway is lateralized in the right hemisphere. Information from the primary visual cortex (V1) travels along the dorsal pathway (spatial perception, determining *where* objects are) or the ventral pathway (object recognition, determining *what* objects are), according to the classical subdivision that has been proposed based on animal models [1]. A third pathway coming from V1 is dedicated to using time information to identify objects (e.g. determining when objects appeared or disappeared). Here, the temporoparietal junction (TPJ; considered the most common substrate of neglect [16]) is identified as a core anatomical locus, within the inferior parietal lobe (IPL); however, the *when* pathway is likely to include a bigger network of areas, including the right angular gyrus (Ang), the supramarginal gyrus (Smg) and the posterior superior temporal sulcus (included in the superior temporal gyrus, STG). All these areas are often involved in the cortical lesion of right parietal patients. The intraparietal sulcus (IPS) separates the IPL from the superior parietal lobe (not labeled). The middle temporal area MT+ is reported in yellow (also called the motion area, highly specialized in detecting and discriminating moving stimuli).

www.sciencedirect.com

LTPJ: temporal order judgment; Wernicke's aphasia - integration of the order within and/or between phonemes or more generally in auditory temporal order judgment

Battelli et al., 2008

Left TP Junction

- Davis, et al., 2009: better study of fast temporal processing
- Important role of left temporoparietal junction in temporal order judgment

- Damage in the left TPJ causes Wernicke's aphasia, a syndrome characterized by language comprehension deficits, due to impaired integration of the order within and/or between phonemes or more generally in auditory temporal order judgment
- Dyslexic individuals often exhibit impaired temporal processing
- Left TPJ may comprise a multisensory temporal order processing unit within an extended “when” pathway.

Lucas Spierer, et al., 2009

Left side of time neglect

- IPL Lesions commonly give rise to spatial neglect.
- Deficits in spatial representation result in deficits in representing events along the mental time line.
- Patients with left hemispatial neglect have difficulty representing events that are associated with the past and, thus, fall to the left on the mental time line.
- Representations of space and time share neural underpinnings and that representations of time have specific spatial properties (e.g., a left and a right side).

Past is put on right of time line

- An intact ability to represent space is necessary for accurate temporal representation.

- A distortion in spatial representation (i.e., contralateral neglect symptoms) predicts a distortion in the way memorized events are represented along the mental time line.
- Patients with neglect tend to crowd past events to the right in their mental time map and consequently misattributing past events to the future.
- Patients with left neglect have difficulty representing events that fall to the left on the mental time line.

Numerosity: Right Superior PC

- Right superior parietal lobe maps numerosity, the set size of a group of items.
- One edge of medial SPC responds maximally to small quantities; opposite edge (closer to the outside of the brain) responded to the largest quantities.
- Parietal cortex map represented relative, not absolute, quantities. They are organized topographically; surface area devoted to specific numerosities decreases with increasing numerosity, and the tuning width increases with preferred numerosity.
- Disproportionately more area represented small quantities, and less area represented large quantities. The map was more selective for smaller than larger numerosities.

B. M. Harvey, et al., 2013

Lying & malingering: More active Inferior parietal

- PFC and parietal activation reflect GREATER brain activity in the deception condition (lying) relative to brain activity in the normal condition

- Malingered response times were associated with activity in the dorsomedial frontal, temporal and inferior parietal regions

Right Temporal Parietal Junction (vPC): Moral Judgment: Judging intentions

- 1 – Joan asks Susan to get coffee with sugar. Susan sees bowl labeled poison and puts it in coffee. But powder is actually sugar. Joan drinks coffee and is fine. (Bad intention; should be blamed based on outcome)
- 2 – Joan asks Susan to get coffee with sugar. Susan sees bowl labeled sugar and puts it in coffee. Powder is toxic poison. Joan drinks coffee and dies. (Accident: Cause harm but Good intention; can forgive)
- Question: In which condition is Susan to blame. People say Susan deserves blame in scenario 1. We interpret Susan morally by her intention. Adult capacity by age 12 (kids with older sybs do better)
- Impaired rTMJ: make decision on basis of outcome, not intention
- Action prediction: 5 year old & correct false belief ability; 3 year old poor at this

Rebecca Saxe

Temporal Parietal Junction (bilateral VPC): Theory of Mind (think about what others are thinking)

"I know you think you understand what you thought I said, but I don't think you realize that what you heard is not what I meant."

Lower RTPJ activation: harsh, outcome-based judgments of accidents
(e.g., she *poisoned* her friend; deliberate)
Higher RTPJ activation: more lenient belief-based judgments
(e.g., she *thought* it was sugar; accident)

Specific patterns in the RTPJ indeed allow a person to *identify* harmful actions as being either deliberate or inadvertent.

ASD: atypical, only outcome-based moral judgments, blame even for accidental outcome
Psychopaths: more likely to "forgive" accidental harms; blunted response to harmful outcome

TPJ: Mind reading

- Superior temporal sulcus (STS): ability to follow people's gaze and determine where another's attention is directed.
- TPJ linked to processing internal and external information, and the ability to distinguish between oneself and others.
- Active when people try to understand the minds of other people, as well as when people redirect their attention.
- TPJ Lesion: poor ability to interpret other people's actions and emotions,

Bibliography

- The Parietal Lobes – Macdonald Critchley, 1953 (1966)
- The Parietal Lobes – (eds) Adrian M. Siegel, et al., 2003
- The Body Has a Mind of Its Own: How Body Maps in Your Brain Help You Do (Almost) Everything Better - Sandra and Matthew Blakeslee, 2008
- The Visual Brain in Action – A. David Milner & Melvyn A. Goodale, 2006