

# 10 -

# 36\_Applied\_Neuroscience

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# 01 - 1. Lobar functions

## 1. Lobar functions

# 02 - A. Tests for frontal and parietal lobes

## A. Tests for frontal and parietal lobes

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1. Lobar functions A. Tests for frontal and parietal lobes Frontal tests Comments Similarities Comparing two objects to test the ability of 'categorisation' and not a description of common 'parts'. This is a test for abstract ability. Lexical fluency Naming items bought in a supermarket or animals (category fluency) or generation of words starting with alphabets FAS (word fluency). Tests not only the speed and accuracy but also the ability to shift from one set of objects to the next. e.g., supermarket list must include not only fruits, but also baked goods, drinks, cleaning items, etc. Luria motor test Fist palm edge - must not be verbally facilitated. Test for motor planning, execution and error correction. Go/on go test Tests response inhibition, the absence of perseveration and resistance to interference. Cognitive estimates test E.g. 'How tall is an average English woman?' Use questions that need abstract not mere factual thinking. Trail making test

Consists of two parts. In part, A simple number sequence is used to join the dots. Test B uses alternating numbers and letters and is thought to be more sensitive to frontal lobe dysfunction. Not specific for frontal lobe; tests visuomotor tracing, attention, conceptualisation and set shifting. Other tests Include alternate pyramids and squares drawing, proverb interpretation, and to some extent frontal release signs and digit span (normal:  $7 \pm 2$  forwards,  $5 \pm 1$  backwards) reflect frontal functions.

Parietal test Comments Copying shapes Ability to draw shapes and constructing geometrical patterns is a parietal (esp. nondominant) function. Identifying fingers Dominant parietal damage can cause finger agnosia as a part of Gerstmann syndrome. Test for the ability to recognise the touched finger when eyes are closed. Also test for the ability to correctly show one's index, middle and ring fingers. Interlocking fingers test (ability to copy examiner's interlocked fingers) is also a parietal test. Calculation ability Dominant parietal damage can cause acalculia as a part of Gerstmann syndrome. Test for simple mathematical functions. Mere recognition and use of numbers constitute arithmetic ability; this is often intact. Graphesthesia Ability to recognise what

number or alphabet is scratched on one's skin without seeing. Bilateral parietal function (somatosensory cortices) Right Left orientation Dominant parietal damage can cause right-left disorientation as a part of Gerstmann syndrome. Test for the ability to touch right ear lobe with the left index finger when eyes are closed. Stereognosis Ability to recognise objects by palpation, and without visual inspection. Bilateral parietal function (somatosensory cortices) Two point discrimination Cortical sensation; bilateral somatosensory cortical function Visual inattention Hemineglect is a feature of parietal lesions. Letter or star cancellation task, line bisection task, draw-a-person or draw-a-tree tasks are useful to identify hemineglect.

# 03 - B. Lobar lesions

## B. Lobar lesions

# 04 - Frontal lobe lesions

## Frontal lobe lesions

# 05 - Parietal lobe lesions

## Parietal lobe lesions

© SPMM Course B. Lobar lesions Frontal lobe lesions

### Parietal lobe lesions

Unilateral lesions Bilateral lesions Contralateral spastic hemiplegia Bilateral hemiplegia Slight elevation of mood, increased talkativeness, tendency to joke inappropriately (Witzelsucht) Spastic bulbar (pseudo bulbar) palsy Frontal release signs (grasp and suck reflexes) Abulia (indecisiveness, lack of drive) Anosmia Decomposition of gait and sphincter incontinence Motor speech disorder with agraphia, with or without oro-buccal apraxia (left) Varying combinations of grasping, sucking, oblique imitative movements, utilization behavior. Loss of verbal fluency with perseveration (left)

Specific frontal syndromes (pseudo depressive, dysexecutive and pseudo psychopathic) Unilateral lesions Bilateral lesions Corticosensory syndrome and sensory extinction Spatial disorientation & visual spatial defects Mild hemiparesis Bilateral ideomotor and ideational apraxia (more prominent with left-sided lesions) Homonymous hemianopia or inferior quadrantanopia (incongruent or congruent) Tactile agnosia (bimanual astereognosis) (more prominent with left sided lesions) Neglect of the opposite side of external space (right parietal lesions)

Anosognosia, dressing and constructional apraxias (may occur with lesions of either hemisphere are more frequent and severe with nondominant right lesions) Gerstmann syndrome (dysgraphia, dyscalculia, finger agnosia, right-left confusion) (left) Balint syndrome GELASTIC SEIZURE

An epileptic fit of incessant 'laughter', not necessarily euphoria, is called gelastic seizure. This occurs with left prefrontal seizures.

# 06 - Temporal lobe lesions

## Temporal lobe lesions

© SPMM Course Temporal lobe lesions

Unilateral lesions Bilateral lesions Homonymous upper quadrantanopia Auditory, visual, olfactory, and gustatory hallucinations Wernicke's aphasia Dreamy states with uncinate seizures Varying degrees of amusia and/or visual agnosia Emotional and behavioural changes Impairment auditory verbal learning Disturbances of time perception Dysnomia Korsakoff amnesic defect (hippocampal formations)

Apathy and placidity

Hypermetamorphopsia (compulsion to attend to all visual stimuli), hyperorality, hypersexuality, blunted emotional reactivity (KluverBucy syndrome; the full syndrome is rarely seen) •most common of auras, causing epigastric aura, salivation, sometimes vertigo etc. Autonomic sensations Autonomic sensations •The individual has a compulsion to think on a certain restricted topic. Forced thinking Forced thinking • Intrusion of stereotyped words or thoughts. Evocation of thought Evocation of thought •Similar to schizophrenic thought block is also reported. Sudden obstruction to thought flow Sudden obstruction to thought flow •Recall of expansive memories in incredible detail, as if running a video show of past. Panoramic memory Panoramic memory •Isolated auras with hallucinations, depersonalisations, micropsia or macropsia, déjà vu or jamais vu (especially if right sided origin) Psychic seizures Psychic seizures •Hallucinations of taste and smell associated with dream like reminiscence and altered consciousness. Uncinate crises Uncinate crises • Points to left hemisphere origin. Transient dysphasia Transient dysphasia •Fear and anxiety very common. Strong affective experiences Strong affective experiences •Ecstatic content in epileptic aura. Dostoevsky's epilepsy Dostoevsky's epilepsy GESCHWIND SYNDROME This is an uncommon type of personality change reported in epilepsy patients (esp. TLE). Symptoms include hypergraphia, circumstantiality, interpersonal viscosity, hyperreligiosity, and hyposexuality. It is thought to be result of lost connectivity among cerebral areas. This may also explain the personality features Psychopathology of the auras of Temporal lobe epilepsy

# 07 - Occipital lobe lesions

## Occipital lobe lesions

# 08 - C. Neuropsychological tests

## C. Neuropsychological tests

# 09 - The Wechsler Adult Intelligence Scale (WAIS)

## The Wechsler Adult Intelligence Scale (WAIS)

© SPMM Course Occipital lobe lesions

C. Neuropsychological tests The Wechsler Adult Intelligence Scale (WAIS) □ Most widely used intelligence test in clinical practice. □ The latest revision, the WAIS-III, is designed for persons 16 to 89 years of age. Wechsler Intelligence Scale for Children-III [WISC-III] is used for <16. For ages, 4 to 6 1/2 years Wechsler Preschool and Primary Scale of Intelligence-Revised [WPPSI-R] is used. □ The WAIS is composed of 11 subtests made up of six verbal subtests and five performance subtests, which yield a verbal IQ, a performance IQ, and a combined or full-scale IQ. □ Verbal tests = similarities, arithmetic, digit span, vocabulary, information and comprehension □ Performance tests = picture arrangement, block design, picture completion, digit symbol, matrix reasoning (replaces object assembly) □ Certain tests are called 'hold tests' as they are supposed to be resistant to age-related decline; these tests may be sensitive for organic brain damage such as dementia. In WAIS, hold tests are vocabulary, information, object assembly and picture completion. Non-hold tests are block design, digit span, similarities and digit symbol. A deterioration quotient is derived from the difference between 'don't hold' and 'hold' test scores.

Unilateral lesions Bilateral lesions Contralateral (congruent) homonymous hemianopia which may be central (splitting the macula) or peripheral; also homonymous hemiachromatopsia Cortical blindness (pupils reactive) Elementary (unformed) hallucinations—usually due to irritative lesions Anton syndrome (visual anosognosia, denial of cortical blindness) If deep white matter or splenium of corpus callosum is involved, alexia and color-naming defect Loss of perception of color (achromatopsia) Visual object agnosia Prosopagnosia (temporo-occipital), simultanagnosia (parieto-occipital) Visual illusions (metamorphopsias) and hallucinations (more frequent with right sided lesions) Balint syndrome (parieto-occipital)

# 10 - Other cognitive instruments

## Other cognitive instruments

© SPMM Course Other cognitive instruments Raven's progressive matrix is a test for IQ that is independent of education and cultural influences. It taps on general intelligence with visuospatial problem-solving tasks (performance IQ). Reading is an ability that is seemingly resistant to organic brain damage. NART - National adult reading test taps on previous word knowledge before becoming ill. Hence, it is used to estimate premorbid IQ. Stroop test measures set shifting abilities and response inhibition. It is a test of frontal function and the ability to pay selective attention. The Wisconsin Card Sorting Test (WCST) contains stimulus cards of different colour, form, and number. These are presented to patients to sort into groups according to a single principle (e.g., to sort by colour, ignoring form and number). Persons with damage to the frontal lobes or to the caudate and some persons with schizophrenia give abnormal responses. Trail Making Test (TMT - B), Wisconsin Card Sort Test (WCST), Hayling test (Sentence completion), Brixton task, all test set-shifting ability, which is a part of executive functioning. The Wechsler Memory Scale-Revised (WMS-R) is the most widely used memory test battery for adults. The scale yields a memory quotient (MQ), which is corrected for age and generally approximates the WAIS IQ. In amnesic conditions, a disproportionately low MQ but a relatively preserved IQ is seen. WMS consists of the following tests: □ verbal paired associate □ paragraph retention, □ visual memory for designs, □ orientation, □ digit span, □ rote recall of the alphabet, and □ counting backward. Benton Visual Retention Test involves the presentation of a geometric figure for 10 seconds, after which the patient attempts to draw the figure from memory. (Short-term visual memory test) The Bender Visual Motor Gestalt Test is a test of visuomotor coordination that is useful for both children and adults. Halstead & Reitan developed a battery of tests that helps to determine the location of specific brain lesions. It consists of Category test, Tactual performance test, Rhythm test, Finger-oscillation test, Speechsounds perception test, Trail making test A and B, Critical flicker frequency, Time sense test, Aphasia screening test, Sensory-perceptual tests. NEUROCOGNITIVE DEFICITS IN SCHIZOPHRENIA Overall deterioration in IQ. Short-term memory disturbances. Deficits in higher order reasoning and perceptual difficulties. Frontal test deficits: Patients perform far more poorly than controls on category test, Wisconsin card sort test, paired associates verbal learning test, Trail B of the Halstead-Reitan battery.

# 11 - 2. Consciousness

## 2. Consciousness

© SPMM Course 2. Consciousness □ Consciousness is a state characterised by an awareness of self and environment and an ability to respond to environmental factors; it is made up of two components - arousal (wakefulness) and awareness (attentional processing). □ Arousal depends on intact functioning of ARAS - Ascending Reticular activating System. Thalamocortical connections generate rhythmical bursts of neuronal activity (20 - 40 Hz) which are in desynchrony by default. ARAS acting via the thalamic intralaminar nuclei synchronises these oscillations. Arousal is directly proportional to the degree of such synchrony achieved. The absence of arousal produces stupor and coma. □ The maintenance of attention appears to require an intact right frontal lobe □ Small lesions of ARAS are enough to produce a stuporous state, but large bilateral lesions are required at the cortical level to cause the same depression in alertness. □ Stupor: In this state the individual appears to be asleep and yet, when vigorously stimulated, may become alert as manifest by eye opening and ocular movement (Cartlidge 2001). Most patients in stupor have diffuse organic cerebral dysfunction. Caloric testing in organic stupor will usually reveal tonic deviation whereas in a psychiatric stupor (catatonia/depression) ocular nystagmus will be seen (Cartlidge 2001). This is because the following tonic deviation in a conscious subject, a fast phase of correction appears resulting in nystagmus. □ Akinetic mutism: It is seen in patients with diencephalic or bilateral anterior cingulate damage. The syndrome is characterised by immobility and eye closure with little or no vocalisation. Sleep/wake cycles can be seen, as indicated by eye opening. There is little in the way of movement to painful stimuli, and the hallmark is the absence of spasticity and rigidity (Cartlidge 2001). Akinetic mutism can arise as a result of lesions that interfere with reticular/cortical integration but spare the corticospinal pathways. There is some debate about whether or not the syndrome should be clearly differentiated from the vegetative state. CJD can also present with akinetic mutism before death. □ Vegetative state: This results from the isolated actions of the ARAS and the thalamus in the absence of higher cortical influence due to extensive cortical damage. A patient in the fully established vegetative state will almost invariably show spasticity and rigidity of the limbs, which are absent in patients with the syndrome of akinetic mutism. In the early stages of the vegetative state, the two clinical syndromes are indistinguishable. □ Locked in syndrome: According to Cartlidge (2001), the ventral pontine or locked in syndrome describes a condition of total paralysis below the level of the third nerve nuclei. Such patients can open their eyes and elevate and depress their eyes to command. Horizontal eye movements are usually lost, and no other voluntary movement is possible. The diagnosis of this state depends on the recognition that the patient can open his eyes voluntarily rather than spontaneously in the vegetative state. This generally results from infarction of the ventral pons, pontine tumours, pontine haemorrhage, central pontine myelinolysis, head injury or brain stem encephalitis.



# 12 - 3. Attention and orientation

## 3. Attention and orientation

© SPMM Course 3. Attention and orientation □ Attention can be clinically tested using serial 7s, digit span, spelling “world” backwards, or asking to recite the months of the year or days of the week in reverse order. □ Although serial 7s is commonly used, it is frequently performed incorrectly by the elderly, as well as by patients with impaired attention. □ A reverse-order month of the year is a highly over-learned sequence and is a preferred measure of sustained attention. □ Digit span is a relatively pure test of attention that depends on working memory. Digit span is impaired in delirium, focal left frontal damage, aphasia, and moderate to severe dementia, but preserved in the amnesic syndrome (for example, Korsakoff’s syndrome or medial temporal lobe damage). Normal digit span of 7 +/- 2 varies with age and general intellectual ability. In the elderly, or intellectually impaired, 5 can be considered normal. Reverse digit span is usually one less than forward span. □ Orientation is usually assessed in time, place and person; it is worth noting that an intact orientation does not exclude a memory disorder. □ Time orientation is the most helpful test and should include the time of day. Many apparently healthy people do not know the exact date, and being inaccurate by two days or less is considered normal. □ Time intervals are often poorly monitored by patients with delirium, moderate to severe dementia, and in the amnesic syndrome, and are easily tested by asking about the length of time spent in hospital. □ Person orientation includes name, age, and date of birth. Disorientation to one’s own name is usually only seen in psychogenic amnesia. □ Orientation to place is affected in reduplicative paramnesia, seen in delirium.

# 13 - 4. Executive function

## 4. Executive function

© SPMM Course 4. Executive function □ This includes planning, initiation, sequencing, coordinating, error detection, error correction, set shifting, and termination. It is closely allied to other frontal functions such as judgement, problem solving, impulse control, and abstract reasoning. □ Executive function is generally believed to be a dorsolateral frontal lobe function and depends on intact frontal-subcortical circuits. □ Impulsivity is thought to reflect failure of response inhibition, and is seen in inferior frontal pathology. It can be assessed using the Go-No-Go task. The examiner instructs the patient to tap once in response to a single tap, and to withhold a response for two taps. This test can be made more difficult by changing the initial rule after several trials (for example, “tap once when I tap twice, and not at all when I tap once”). □ The ability to switch task, and the inhibition of inappropriate, or perseverative, responses can also be assessed by asking the patient to copy a short sequence of alternating squares and triangles, and then to continue across the page. Perseveration in drawing one or other of the shapes may be seen in frontal lobe deficits, but the test is relatively insensitive. □ The cognitive estimates test may prompt bizarre or improbable responses in patients with frontal or executive dysfunction. Although it is a formal test performed at the bedside by asking, for example, the height of the Post Office Tower, the population of London, or the speed of a typical racehorse. □ Questions about the similarity between two conceptually similar objects can be used to assess inferential reasoning, which may be impaired in the same way. Simple pairs such as “apples and oranges” or “desk and chair” are tested first, followed by more abstract pairs such as “love and hate” or “sculpture and symphony”. Patients typically answer, quite concretely, that two objects are “different” or that they are “not similar” instead of forming an abstract concept to link the pair. This often persists despite encouragement to consider other ways in which the items are alike. □ Testing of proverb meanings probably measures a similar skill, but it is highly dependent on educational and cultural background.

14 - 5. Visuospatial ability

5. Visuospatial ability

15 - Neglect

Neglect

# 16 - Dressing and constructional apraxia

## Dressing and constructional apraxia

© SPMM Course 5. Visuospatial ability □ Information from the visual cortex is directed towards the temporal or parietal cortex via one of the two streams. The dorsal (“where”) stream links visual information with spatial position and orientation in the parietal lobe, whereas the ventral (“what”) stream links this information to the store of semantic knowledge in the temporal lobes. □ The frontal eye fields are important in directing attention towards targets in the visual field. □ Neglect and constructional apraxia are disorders of visuospatial function. Neglect o Neglect of personal and extrapersonal space is usually due lesions to the right hemisphere— usually the inferior parietal or prefrontal regions. o Left side of personal and extrapersonal space is represented only on right parietal lobe, but right personal and extrapersonal space gets bilateral representation. Hence, a left-sided lesion rarely results in neglect, but right-sided lesion can result in left-sided neglect. o Deficits can be uncovered by simultaneous bilateral sensory or visual stimulation, or having the patient bisect lines of variable length. Letter and star cancellation tasks are similar, more formal tasks. o Visual neglect may produce a failure to groom one-half of body, or eat what is placed on one side of a plate. In extreme cases, patients may have anosognosia and deny they are hemiplegic or even that the affected limb belongs to them. Dressing and constructional apraxia o Although deficits in dressing and constructional ability are termed apraxias, they are best considered as visuospatial, rather than motor impairments. o Copying three-dimensional shapes such as a wire cube, interlocking pentagons (as in MMSE), or constructing a clock-face with numbers are good tests of constructional ability and may also highlight neglect if present. o Dressing apraxia is easily tested by having the patient put on clothing that has been turned inside out.

17 - 6. Memory

6. Memory

# 18 - Classification of memory

## Classification of memory

# 19 - Brain structures involved in memory

## Brain structures involved in memory

© SPMM Course 6. Memory Classification of memory □ According to duration: □ Immediate memory functions over a period of seconds; closely related to concept of working memory □ Recent memory applies on the scale of minutes to days; and □ Remote memory encompasses months to years. □ According to the type of encoding memory, can be classified into explicit or declarative memory and implicit or procedural memory. □ Explicit memory can be either semantic (meanings) or episodic (events). Episodic memory depends on the hippocampal-diencephalic system. It is the time-locked memory for personal events ('when and where' memory); it includes both anterograde and retrograde memory. Semantic memory involves memory for word meaning and general knowledge. □ The implicit memory includes skills and procedures e.g. car driving. □ Working memory refers to the very limited capacity that allows us to retain information for a few seconds.). It is made of a central executive system (attentional system, dorsolateral prefrontal) and at least 2 important buffer systems - the visuospatial sketchpad (right hemisphere) and phonological loop (left hemisphere). □ The term "short term" memory is applied, confusingly, to a number of different memory problems, but has no convincing anatomical or psychological correlate

Brain structures involved in memory □ Hippocampus □ Left hippocampus for encoding declarative verbal and right hippocampus for encoding nonverbal memories. □ Navigational memory and memory of object location in space are also served by the hippocampus. □ Animal studies have defined a hippocampal place code, a pattern of cellular activation in the hippocampus that corresponds to the animal's location in space. □ Unilateral hippocampal lesions are compensated well, and clinically significant amnesia does not occur. □ Amygdala has been suggested to rate the emotional importance of experience and to regulate the level of hippocampal activity accordingly. It is involved in emotional memory and emotional face processing. It helps in memory consolidation, depending on emotional input for the content of the memory. Amygdalar damage leads to loss of fear conditioning and in monkeys, loss of maternal behaviour has also been noted. Despite the amygdalar damage, learning and consolidation of memory can occur, especially in the absence of emotional valence and arousal. □ Diencephalic structures such as the dorsal medial nucleus of the thalamus and the mamillary bodies are associated with new learning; their damage leads to diencephalic amnesia seen in Korsakoff syndrome.



# 20 - Disorders of memory

## Disorders of memory

© SPMM Course □ Other regions: In most cases of memory loss procedural memory is intact. A deficit in procedural memory with preservation of declarative memory may be seen in persons with Parkinson's disease, in whom dopaminergic neurons of the nigrostriatal tract degenerate. Though speculative, cerebellum, striatum, amygdala and certain parts of the neocortex (including motor area) are thought to be involved in non-declarative procedural memory storage. The anterior temporal lobe is the key area for semantic memory. □ Long-term potentiation: Strengthening of the connection between two neurons on repeated communication is called long-term potentiation - LTP. This may be the neuronal basis of memory. It is mediated by NMDA mediated  $Ca^{2+}$  entry in glutamate neurons. Learning increases branching and synapse formation and may also influence neurogenesis.

Disorders of memory □ Amnesia is a term used either for pure memory deficits (mostly episodic) or cognitive deficits where memory loss is predominant and not congruent with the level of loss in other domains. □ Generally both anterograde and retrograde memory loss occur in parallel, such as in Alzheimer's disease or head injury. □ Relatively pure anterograde amnesia may be seen when there is hippocampal damage, e.g. herpes simplex encephalitis, focal temporal lobe tumours, or infarction. □ Confabulation—for example, in Korsakoff's syndrome—might be grandiose or delusional, but more often involves the misordering and fusion of real memories which end up being retrieved out of context. □ A transient amnesic syndrome with pronounced anterograde, and variable retrograde, amnesia is seen in transient global amnesia (TGA), while "memory lacunes", and repeated brief episodes of memory loss suggest transient epileptic amnesia (TEA). □ Ribot's Law of retrograde amnesia: 'The dissolution of memory is inversely related to the recency of the event'. Recent memories are more likely to be lost than the more remote memories in organic amnesia (not always the case though). □ Semantic dementia: It is a variant of frontotemporal dementia. Patients with semantic breakdown typically complain of loss of words. Vocabulary diminishes, and patients use substitute words such as "thing". There is a parallel impairment in appreciating the meaning of individual words, which first involves infrequent or unusual words. □ A word finding difficulty is common in both anxiety and aging, but variable and not associated with impaired comprehension. This is in stark contrast to the anomia in semantic dementia which

Anterograde amnesia Forgetting newly encountered information from the time of a lesion. Presents as forgetfulness regarding appointments, losing items around the home, inability to remember conversation leading to repeated questions etc. Retrograde amnesia Loss of memory of past events that happened before the lesion was sustained. Presents as loss of memory of past events such as jobs, holidays, not able to remember the topography of a route and getting lost.

© SPMM Course is relentlessly progressive and associated with atrophy of the anterior temporal lobe, usually on the left. □ Working memory deficits can present as lapses in concentration and attention e.g. losing one's train of thought, inability to process a complex task as the components are not retained long enough in memory to be processed. Basal ganglia and white matter diseases may present with predominantly working memory deficits. □ Dissociative amnesia is not an organic syndrome, but centred on the loss of memory of important recent events that is partial, patchy and selective. It can occur as a part of dissociative fugue. The characters of dissociative amnesia are episodic memory loss (retrograde only with no anterograde deficits) for events that happened in a discrete period of time from minutes to years. In dissociative amnesia, the problem is not inefficient retrieval but the strikingly complete unavailability of memories which were formed normally and were previously accessible. The forgotten events are generally traumatic or stressful.

# 21 - 7. Language

## 7. Language

© SPMM Course 7. Language □ Aphasia refers to a higher-level language defect despite intact hearing, sound production, articulation mechanisms. □ Aphasia is almost always organic. Naming defects (anomia) accompanies any aphasia in various degrees. □ To understand aphasia, consider the following facts □ Sound received by ears is transmitted to Wernicke's area and auditory association cortex that processes the language component. □ Arcuate fasciculus connects Wernicke's area to Broca's area. (NOTE: this is different from the uncinata fasciculus that interconnects the anterior temporal and inferior frontal gyrus) □ Broca's area is the higher motor area of language production. Signals from Broca's area are relied on onto the motor area to coordinate the delivery of language via the tongue, lips and vocal cords. □ Three important components of language are □ Fluency depends on intact Broca's area and its forward connections. □ Comprehension depends on intact Wernicke's area and its connection with association cortex and sensory input □ Repetition requires no high-level processing. Repetition can occur if Broca's, Wernicke's and arcuate fasciculus are intact. Repetition does not need relay of signals from either Broca's or Wernicke's areas to higher association areas. Adapted from Harrison's Textbook of internal medicine; 15 e

□ In Broca's aphasia the speech is nonfluent; it often appears laboured with any interruptions and pauses. Function words (propositions, conjunctions) are most affected though the good degree of meaning-appropriate nouns and verbs are still produced. Abnormal word order and a characteristic agrammatism are noted. Speech is telegraphic. Harrison's Textbook of Medicine quotes the following example: "I see...the dotor, dotor sent me...Bosson. Go to hospital. Dotor...kept me beside. Two, tee days, doctor send me home". □ In Wernicke's aphasia, the comprehension is impaired for both spoken and written language. Language output is fluent but is highly paraphasic, sometimes with string of neologisms and circumlocutions. Hence, it is also termed as "jargon aphasia." The speech contains large numbers of function words (e.g., prepositions, conjunctions) but few substantive nouns or verbs that refer to specific actions. The output is, therefore, voluminous but uninformative, mimicking schizophrenic speech disturbance at times. Type of aphasia Fluency Repetition Comprehension Naming Wernicke's sensory aphasia Intact Lost Lost Lost Broca's motor aphasia Lost Lost Intact Lost Conduction aphasia Intact Lost Intact Lost Transcortical sensory aphasia Intact Intact Lost Lost Transcortical motor aphasia Lost Intact Intact Lost

© SPMM Course □ Pure word deafness: Patient can speak read & write fluently, but comprehension is impaired only for spoken language. Bilateral (or left sided with disrupted connections to non-dominant circuit) damage to the superior temporal pole is suspected. □ Pure word blindness (alexia no agraphia): Here the patient can speak normally and comprehend what is spoken; he can

also write spontaneously and to dictation, but reading comprehension is impaired. It almost always involves an infarct to the left posterior cerebral artery affecting splenium of the corpus callosum and left visual cortex. So the affected person, who is still able to see with the right visual cortex, cannot undertake lexical word processing making him unable to read. □ Pure word dumbness: Spoken language cannot be produced clearly, but the patient can comprehend language well, can read and write. □ Pure agraphia: This is an isolated inability to write while other faculties of language are preserved.

22 - 8. Apraxia

8. Apraxia

# 23 - Types of apraxia

## Types of apraxia

© SPMM Course 8. Apraxia □ Damasio and Geschwind (1985) defined apraxia as a condition with varying combinations of the following disturbances in order of progressive dysfunction: o A failure to produce the correct movement in response to a verbal command, o A failure to correctly imitate a movement performed by the examiner, o A failure to perform a movement correctly in response to a seen object and o A failure to handle an object correctly □ Although a number of categories, such as limb kinetic, ideomotor, and ideational, exist, these labels are seldom useful in clinical practice. It is more helpful to describe the apraxia by region (orobuccal or limb), and to provide a description of impaired performance, recording both spatial and sequencing errors on several different types of task. □ Apraxia is of limited localizing ability, but the left parietal and frontal lobes appear to be of greatest importance. □ Progressive, isolated limb apraxia is virtually diagnostic of corticobasal degeneration. Types of apraxia Functional classification: Apraxia type Definition Localization Constructional apraxia

Inability to construct elements into a meaningful whole. e.g., inability to draw or copy simple diagrams or figures. Right cerebral hemisphere, often parietal lobe. Ideational/concept ual Impairment in carrying out sequences of actions (multiple-step task) requiring the use of various objects in the correct order to achieve an intended purpose. The patient does not know 'what' to do. Left parieto-occipital and parietotemporal regions Ideomotor (most common type among all apraxias) The disorder of goal-directed movement. The patient knows what to do but not how to do it. Impairment of pantomiming ability to use tool. Abnormalities include the use of body-part-as-object substitution, e.g. the patient uses his own finger to represent a toothbrush when asked to brush his teeth and abnormal orientation of body part performing the action. Improves on imitation and with the use of the actual tool. Tool use is more affected than gestures. Mainly in the left hemisphere; frontal and parietal association areas. Unilateral lesions of the left hemisphere in right-handed patients produce bilateral deficits, usually less severe in the left than in the right limb

Regional classification: Buccofacial apraxia (aka facial-oral apraxia) Inability to coordinate and carry out facial and lip movements such as whistling, winking, coughing, etc. on command. The most frequent type of all focal brain lesion related apraxia syndromes. Associated with left inferior frontal lobe and the insula, and commonly accompanies the aphasia caused by lesions of Broca's area. Limb-kinetic Loss of hand and finger dexterity resulting Dominant frontoparietal or primary motor

© SPMM Course from the inability to connect or isolate individual movements. Affects use of tools, gestures, especially distal fingers movements. Can be either ideomotor or ideational type. cortex Other variants Apraxia of speech, apraxia of eyelid opening and apraxia of gait.



# 24 - 9. Agnosias

## 9. Agnosias

# 25 - Visual agnosia

Visual agnosia

# 26 - Prosopagnosia

## Prosopagnosia

# 27 - Colour deficits

## Colour deficits

© SPMM Course 9. Agnosias

- o Visual agnosia
- o Visual object agnosia refers to a failure of object recognition despite adequate perception.
- o Patients with apperceptive visual agnosia have normal vision, but cannot identify and name objects. But these subjects have preserved semantic representation of the object, as evidenced by their ability to name objects in description or touch. This is seen in patients with bilateral occipitotemporal infarction.
- o In associative visual agnosia, the stored semantic knowledge is affected. Lesions of the anterior left temporal lobe are often seen.
- o To test for visual agnosia, it is important to assess visual object naming/description and tactile naming, naming described objects, and providing semantic information about unnamed items.

Prosopagnosia

- The ability to recognise familiar faces is affected in prosopagnosia. But clues such as voice, gait, etc. can aid identification.
- The deficit is often not just restricted to faces; fine-grained identification within categories may also be impaired (e.g. types of fruits and flowers).
- The underlying semantic knowledge associated with a particular person is not disrupted; so when asked to describe the facial features of a named person, the patient can usually describe this well.
- Face processing is a bilateral function; more key areas may be present on the right hemisphere.
- Acquired prosopagnosia is usually associated with bilateral or right-sided lesions of the occipital

- temporal junction (FUSIFORM GYRUS). In rare cases of prosopagnosia after left-sided lesions in left-handed subjects, it is attributed to a reversed hemispheric specialization for face processing.

### Colour deficits

Achromatopsia Colour agnosia Colour anomia Loss of ability to discriminate colours. (Often associated with pure alexia) Loss of the ability to retrieve colour information stored in semantic knowledge base (E.g. "What colour is a banana?") Disorder of colour naming despite intact perception and colour knowledge ("What colour is this?") Medial occipitotemporal damage due to left posterior cerebral artery infarction Left occipito-temporal damage Disconnection of the language structures in the temporal lobe from the visual cortex

# 28 - 10. Other neurological deficits

## 10. Other neurological deficits

© SPMM Course 10. Other neurological deficits Acalculia refers to the inability to read, write, and comprehend numbers. It is NOT the same as anarithmetica, which is the inability to perform arithmetical calculations. Acalculia can be tested using the simple calculation, writing numbers to dictation, copy numbers and read them aloud, and give reasons for calculated answers. Balint's syndrome results from bilateral superior-parietooccipital damage (disruption to the dorsal "where" stream linking visual and parietal association areas). The triad of symptoms is shown in the attached figure. Possible causes include carbon monoxide poisoning, infarction, and Alzheimer's disease. Gerstmann syndrome is characterized by four primary symptoms: dysgraphia/agraphia, dyscalculia/acalculia, finger agnosia and left-right disorientation. The full presentation of tetrad is rare but occurs with lesions in the dominant angular and supramarginal gyri (parietal lobe). Anton's syndrome occurs in bilateral occipital damage. The patient denies any deficit and may even attempt to walk and navigate without success. Marchiafava-Bignami disease is due to symmetrical demyelination and necrosis of corpus callosum and adjacent anterior commissure. It is mostly seen in alcoholics using red wine excessively (not clear whether some impurities are implicated). Patients present with sudden onset of stupor or coma and seizures. A chronic onset of dementia and/or gait problems with spasticity is also reported.

- inability to attend to more than one item of a complex scene at a time simultanagnosia
- simultanagnosia
- inability to guide reaching or pointing despite adequate vision optic ataxia
- optic ataxia
- inability to voluntarily direct saccades to a visual target oculomotor apraxia
- oculomotor apraxia

29 - 11. Cranial nerves

11. Cranial nerves

30 - Olfactory nerve CN I

Olfactory nerve CN I

# 31 - Optic nerve CN II

## Optic nerve CN II

© SPMM Course 11. Cranial nerves Olfactory nerve CN I □ Only sensory nerve to have no thalamic relay □ Unilateral anosmia should raise the suspicion of a lesion affecting the olfactory nerve filaments, bulb, tract, or stria. □ Because the cortical representation for the smell in the piriform cortex is bilateral, a unilateral lesion distal to the decussation of the olfactory fibers (i.e. temporal/uncinate) causes no olfactory impairment. □ Frontal meningiomas can cause unilateral anosmia. □ Head injury is probably the most common cause of disruption of the olfactory fibers Hyposmia is an early feature of Parkinson's disease and Alzheimer's dementia and may precede motor and cognitive signs respectively. □ Impaired sense of smell is seen in some patients at 50% risk of Parkinsonism.

**Optic nerve CN II Syndrome Lesion**

**Unilateral one eye blindness** Lesion anterior to optic chiasm e.g. optic nerve itself or retina

**Bitemporal hemianopia** Optic chiasmatic lesion e.g. craniopharyngioma, pineal tumors

**Homonymous hemianopia - left** Lesions of the right sided optic tract, lateral geniculate body, optic radiations and striate cortex (any retro chiasmatic structure)

**Homonymous hemianopia - right** Lesions of the left retro chiasmatic structures

**Enlargement of the blind spot** Any process causing disc swelling

**Superior quadrantanopia** Optic irradiation lesion at temporal lobes of contralateral side

**Inferior quadrantanopia** Optic irradiation lesion at parietal lobes of contralateral side

**Cortical blindness** Occipital cortex lesions □ Hemianopia is a field defect covering roughly half of the field. Vertical hemianopia can be nasal or temporal. Horizontal or altitudinal hemianopia can be superior or inferior. If only one-fourth of the field is affected, this is called quadrantanopia. □ Bilateral field defects are homonymous when they affect the identical portion of vision in both visual fields □ Funnel vision: In patients with organic visual system defect, the visual field projected at 2 metre distance is larger than the field at 1 m. This is seen in glaucoma, retinitis pigmentosa, post papilledema optic atrophy, bilateral occipital infarcts with macular sparing. □ Tunnel vision refers to the absence of disparity between 2m and 1m fields on confrontation test. The presence of patchy spirals of field loss is seen in hysteria or malingering. □ Cortical blindness often results from simultaneous bilateral posterior cerebral artery occlusion. Patients often have a bilateral homonymous hemianopia with the small central field around the point of fixation (macular sparing or keyhole vision) or complete blindness. Occasionally, patients with cortical blindness deny their visual defect (Anton's syndrome). □ The following testing is appropriate for optic nerve:

# 32 - Pupillary light reflex

Pupillary light reflex

33 - The convergence  
accommodation reflex

The convergence /  
accommodation reflex

34 - Oculomotor nerve CN III

Oculomotor nerve - CN III

# 35 - Trochlear nerve CN IV

## Trochlear nerve - CN IV

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1. Acuity using the Snellen chart (near and distant vision)
2. Visual fields using confrontation test or perimetry
3. Colour vision using Ishihara chart
4. Fundoscopy Pupillary light reflex Afferent fibres in each optic nerve (some crossing in the chiasm) pass to both lateral geniculate bodies and relay to the Edinger-Westphal nuclei (midbrain) via the pretectal nucleus. Efferent (parasympathetic) fibres from each Edinger-Westphal nucleus pass via the third nerve to the ciliary ganglion and thence to the pupil. Light constricts the pupil being illuminated (direct reflex) and, by the consensual reflex, the contralateral pupil. The convergence / accommodation reflex Fixation on a near object requires convergence and is accompanied by pupillary constriction. Afferent fibres in each optic nerve, which pass through both lateral geniculate bodies, also relay to the convergence centre. This centre receives muscle spindle afferent fibres from the extraocular muscles - principally medial recti - which are innervated by the third nerve. The efferent route is from the convergence centre to the Edinger-Westphal nucleus, ciliary ganglion and pupils. Pupils that accommodate but do not react are said to show light-near dissociation. Two important types are Argyll Robertson pupil, seen in neurosyphilis and diabetes (more common these days), and Adie pupil due to peripheral pupillary defect producing a tonic pupil. ARP (note: Accommodation Reflex Present - light reflex absent) is due to an afferent defect in pupillary reflex pathway - possibly pretectal. Oculomotor nerve - CN III □ The oculomotor nucleus of the nerve is located in the midbrain □ Supplies the levator palpebrae superioris; the superior, inferior, and medial recti; and the inferior oblique muscles. □ Lesions of CN III result in paralysis of the ipsilateral upper eyelid and pupil, leaving the patient unable to adduct and look up or down. The eye is frequently turned out (exotropia). □ Lesions of the nucleus of the third nerve cause bilateral ptosis, in addition to the findings mentioned above. □ Paralysis of CN III is the only ocular motor nerve lesion that results in diplopia in more than 1 direction. □ Pupillary involvement is an additional clue to the involvement of CN III. □ Pupil-sparing CN III paralysis occurs in diabetes mellitus, vasculitides of various etiologies, and certain brainstem lesions such as due to multiple sclerosis. Trochlear nerve - CN IV The nucleus of the nerve is located in the midbrain. It innervates the superior oblique muscle. Trochlear nerve typically allows a person to view the tip of his or her nose.

36 - Trigeminal nerve CN V

Trigeminal nerve - CN V

37 - Abducens nerve CN VI

Abducens nerve - CN VI

38 - Facial nerve CN VII

Facial nerve - CN VII

# 39 - Vestibulocochlear nerve

## CN VIII

# Vestibulocochlear nerve - CN VIII

© SPMM Course Trigeminal nerve - CN V □ The nucleus of the nerve stretches from the midbrain (i.e. mesencephalic nerve) through the pons (main sensory nucleus and motor nucleus) to the cervical region ( a spinal tract of the trigeminal nerve). □ It provides sensory innervation for the face and supplies the muscles of mastication. □ Divisions: ophthalmic; V1, maxillary; V2, mandibular; V3. □ Corneal reflex: □ Afferent - V nerve □ Efferent - facial nerve □ Complete paralysis of CN V results in sensory loss over the ipsilateral face and weakness of the muscles of mastication. Attempted opening of the mouth results in deviation of the jaw to the paralyzed side. □ Acoustic neuroma can press on 5th nerve leading to loss of the corneal reflex. Abducens nerve - CN VI The nucleus of the nerve is located in the paramedian pontine region on the floor of the fourth ventricle. It innervates the lateral rectus, which abducts the eye. Patients complain of double vision on horizontal gaze only. This finding is referred to as horizontal homonymous diplopia. Paralysis of CN VI is a false localising sign as it may result from increased intra cranial pressure. Facial nerve - CN VII □ Motor supply to facial muscles from the motor nucleus. □ Though it is considered a predominantly motor nerve, it also innervates a small strip of the skin of the posteromedial aspect of the pinna and around the external auditory canal. It serves to conduct taste sensation from the anterior two-thirds of the tongue and relay to sensory nucleus tractus solitarius. □ Secretomotor functions include parasympathetic relay to lacrimal, lingual and submandibular glands. □ A lower-motor-neuron lesion of the nerve, results in complete ipsilateral facial paralysis; the face draws to the opposite side as the patient smiles. Eye closure is impaired, and the ipsilateral palpebral fissure is wider. This is called Bell 's palsy where the cause is idiopathic. □ In an upper motor neuron lesion, only the lower half of the face is paralyzed. Eye closure is usually preserved. Vestibulocochlear nerve - CN VIII □ 2 components - vestibular for balance; cochlear for hearing. □ Auditory part tested using 512 Hz - Weber's test and Rinne's test. □ The Weber test involves holding a vibrating tuning fork against the forehead in the midline. The vibrations are normally perceived equally in both ears because bone conduction is equal. In conductive hearing loss, the sound is louder in the abnormal ear than in the normal ear. In sensorineural hearing loss, lateralization occurs to the normal ear. □ In the Rinne test, the vibrating tuning fork is placed over the mastoid region until the sound is no longer heard. It is then held at the opening of the ear canal on the same side. A patient with normal



40 - Glossopharyngeal nerve  
CN IX

Glossopharyngeal nerve - CN  
IX

# 41 - Vagus nerve CN X

## Vagus nerve - CN X

© SPMM Course hearing should continue to hear the sound. In conductive hearing loss, the patient does not continue to hear the sound since bone conduction, in that case, is better than air conduction. In sensorineural hearing loss, both air conduction and bone conduction are decreased to a similar extent. □ The vestibular portion transmits information about linear and angular accelerations of the head from the utricle, saccule, and semicircular canals of the membranous labyrinth to the vestibular nucleus. □ The Romberg test is performed to evaluate vestibular control of balance and movement. When standing with feet placed together, and eyes closed, the patient tends to fall toward the side of vestibular hypofunction. Results of the Romberg test may also be positive in patients with polyneuropathies, and diseases of the dorsal columns, but these individuals do not fall consistently to one side as do patients with vestibular dysfunction. □ Provocative tests include caloric testing. Normally on cold water testing, nystagmus is noted to the opposite side; warm water elicits nystagmus towards the same side. (Remember the mnemonic COWS) Glossopharyngeal nerve - CN IX □ The nucleus of the CN IX is anatomically indistinguishable from the CN X, therefore, known as nucleus ambiguus. Its main function is the sensory innervation of the posterior third of the tongue and the pharynx. It also innervates the pharyngeal musculature, particularly the stylopharyngeus, in concert with the vagus nerve. □ Vascular stretch afferents from the aortic arch and carotid sinus travel via glossopharyngeal nerve to the nucleus solitarius - important for neural control of blood pressure. □ Lesions affecting the glossopharyngeal nerve result in loss of taste in the posterior third of the tongue and loss of pain and touch sensations in the same area, soft palate and pharyngeal walls. □ CN IX and CN X travel together, and their clinical testing is not entirely separable. Vagus nerve - CN X □ Starting in the nucleus ambiguus, the vagus nerve has the longest peripheral course of all cranial nerves - it stretches up to splenic flexure of the colon. □ Provides motor supply to the pharyngeal muscles (except the stylopharyngeus and the tensor veli palati), palatoglossus, and larynx. □ It innervates the smooth muscles of the tracheobronchial tree, esophagus, and GI tract up to the junction between the middle and distal third of the transverse colon. □ The somatic sensation is carried on the back of the ear, the external auditory canal, and parts of the tympanic membrane, pharynx, larynx, and the dura of the posterior fossa. □ The pharyngeal gag reflex (ie, tongue retraction and elevation and constriction of the pharyngeal musculature in response to touching the posterior wall of the pharynx, tonsillar area, or base of the tongue) and the palatal reflex (ie, elevation of the soft palate and ipsilateral deviation of the uvula on stimulation of the soft palate) are decreased in paralysis of CN IX and CN X. □ In unilateral CN IX and CN X paralysis, touching these areas results in deviation of the uvula to the normal side.

42 - Spinal accessory nerve

CN XI

Spinal accessory nerve - CN

XI

43 - Hypoglossal nerve CN  
XII

Hypoglossal nerve - CN XII

# 44 - 12. Traumatic brain injury

## 12. Traumatic brain injury

© SPMM Course Spinal accessory nerve - CN XI □ Spinal root supplies trapezius and sternocleidomastoid. Hypoglossal nerve - CN XII □ It provides motor innervation for all the extrinsic and intrinsic muscles of the tongue. To test the hypoglossal nerve, have the patient protrude the tongue; when paralyzed on 1 side, the tongue deviates to the side of paralysis on protrusion.

12. Traumatic brain injury □ Traumatic brain injury is the result of mechanical forces applied to the skull and transmitted to the brain. This may lead to focal and/or diffuse brain damage. □ Focal lesions often result from a direct blow to the head and include brain laceration, contusion, intracerebral hemorrhage, subarachnoid or subdural hemorrhage, and ischemic infarct. □ Concussion causes transient coma for hours followed by apparent complete clinical recovery. Brain contusion leads to prolonged coma, focal signs and lasting brain damage. Pathological support for the distinction between concussion vs. contusion is poor. □ Contusion occurs directly beneath (coup injury) or contralateral (contrecoup injury) to the site of impact. Contre-coup is most common in the orbital-frontal area and the temporal tips, where acceleration/deceleration forces cause the brain to impact on the bony protuberances of the skull. A frontal behavioural dyscontrol syndrome occurs in cases of bilateral orbitofrontal injury. □ Mechanisms of TBI include axonal and neuronal damage from direct trauma, shearing and rotational stresses on decelerating brain, brain oedema and raised intracranial pressure, brain hypoxia and ischaemia. □ The differential motion of the brain within the skull can cause shearing and stretching of the axons resulting in diffuse axonal injury (DAI). DAI related damage occurs over a more widespread area with extensive lesions in white matter tracts than in focal brain injury. DAI is more often associated with persistent vegetative state and coma. □ Two types of amnesia can occur after head injury: □ Post-traumatic amnesia (PTA) includes anterograde amnesia for the period of injury and the period following injury until normal memory resumes. □ Retrograde amnesia includes dense amnesia for the period between the last clearly recalled memory prior to the injury and the injury itself. The duration of PTA is mostly in minutes, and with increasing time after the injury, the duration of PTA reduces gradually. □ GCS (Glasgow coma scale) at 24 hours after injury is widely used to assess severity. Apart from GCS other indices of TBI severity include the length of coma (LOC), duration of post-traumatic amnesia (PTA), and the Abbreviated Injury Scale (AIS) scores. LOC and PTA have been used exclusively to predict the functional outcome, but the AIS has been used to predict survival. Most investigations have found LOC or PTA to be more predictive of functional status than GCS. □ Poor prognostic factors with respect to psychiatric morbidity following head injury includes long duration of loss of consciousness, long PTA, elderly, chronic alcohol use, diffuse brain damage, new

onset seizures and focal damage to dominant lobe.

© SPMM Course Duration of PTA Classification Functional outcome PTA less than 60 minutes Mild injury May return to work in <1 month PTA between 1-24 hours Moderate injury May return to work in 2 months PTA between 1-7 days Severe injury May return to work in 4months PTA greater than 7 days Very severe injury May require > 1 year for return to work □ Late sequelae o Cognitive impairment is common especially after closed head injuries with PTA lasting >24 hours. o Personality changes are most likely after a head injury to the orbitofrontal lobe or anterior temporal lobe. o Depression (most common sequelae) and anxiety occur in roughly 1/4 of head injury survivors. Suicide risk is also higher post head injury. o Post-concussional syndrome is characterized by headache; dizziness; insomnia; irritability; emotional lability; increased sensitivity to noise, light, etc.; fatigue; poor concentration; anxiety; and depression. o A schizophrenia-like psychosis with prominent paranoia is associated with left temporal injury while affective psychoses (esp. mania in 9% patients) are associated with right temporal or orbitofrontal injury. There is also an increased prevalence of schizophrenia post head injury (-2.5% develop the disorder). o Post-traumatic epilepsy is seen in 5% closed and 30% open head injuries (usually during the first year) and worsens the prognosis. o Less psychopathology in children after head injury due to increased brain plasticity.

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