Pathophysiology of consciousness

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Everything we know about ourselves, about our existence, our body and its parts, everything we know about surrounding physical reality is created in the brain and projected for us as an experienced reality. As brain’s awareness grows it allow us to „humanize“ everything, i.e. to consider that we are inevitable and to admit that other creatures are just actors in reality. Persistence of consciousness outside of the brain is unprobable. One’s consciousness however may persist in form of recorded thoughts, ideas, or imprinted in memories of others.

R. Benacka (2013)
Consciousness – general considerations

- **Consciousness** = various manifestation of neuronal assemblies of different size and complexity; no center in the brain; structural components of arousal, alertness, attentiveness, memory, emotion
- **Consciousness** = activation state achieved in the brain, maintained by the brain for the brain; outside of the brain consciousness has no real meaning
- **Consciousness** = continuum of behavioral states; changing throughout the night and day; also including somatic idiognosia
- **Consciousness** = morphed throughout the ontogeny; different in the kid, adulthood, man, woman; it is rather subjective than objective; similar to memory and emotions
- **Consciousness** = practical medicine through interviewing persons evaluates rather „awareness“ (Can you hear me ?, Where are you, Who are you ?)

Consciousness Tetrad (Singh & Singh, 2011)

- **Default consciousness**: basic manifestation of the life; differentiating living from dead; bilological principle form non-living physical principle
- **Aware consciousness**: continuum of behavioural states (lucidity, somnolency, sleep) po patologické (somnolencia, stupor, koma)
- **Operational Consciousness**: ability to perform motor, sensoric, cognitive, creative, emotional, esthetical manifestations
- **Exalted Consciousness**: connection with the source; God meditation, creativity
1 Neuronal representation of consciousness
Various levels of consciousness – older view

- **Consciousness components**
  - **arousal** = activity level, charge, energy level
  - **content** = awareness of self and awareness of environment

Continuum of behavioural states

GENERAL ALERTNESS

FOCALIZED ALERTNESS

ORIENTATION RESPONSE

ACTIVATION AROUSAL

FOCAL ALERTNESS

GENERAL ATTENTION

FOCALIZED ATTENTION

LEARNING, SPEECH THINKING

MOTOR OUTPUT

ROTATION OF HEAD

BRAINSTEM

THALAMUS PARietAL CORTEX

FRONTAL ASSOCIATION CORTEX

THALAMUS PARietAL CORTEX

RESPIRATION, POSTURATION CIRCULATION

FOLLOWING, GRASPING, ROOTING, STARTLE, POSTURE
Brainstem – arousal systems

"Consciousness “is required for us to survive – for breathing, heart activity etc."

Praventricular nucleus
vasopressin secretion

Locus coeruleus
Koeltler-Fuse area
Paraventricular nucleus

Brachium conjunctivum

Dentate nucleus

Bradycardia
Baroreceptors

Solitary tract

Ventrolateral
superficial reticular formation

Ventrolateral
superficial reticular formation

Bötzinger complex
Pre-Bötzinger cells: pacemaker

Ventral respiratory group: inspiration

Ventral respiratory group: expiration

Phrenic nerve contraction diaphragm

Intermediolateral column

Thoracic nerve

Vasoconstriction

Abdominal muscles

Cerv.

Cerv. 3/4

Central nucleus of the medulla oblongata: lateral subnucleus

Central nucleus of the medulla oblongata: medial subnucleus

Lateral reticular nucleus

Paramedian reticular nucleus

Retroambigus nucleus

Intermediate zone of the spinal cord

Nucleus paragigantocellularis

Nucleus gigantocellularis

Nucleus raphes obscurus

A5

Nucleus raphes medialis

Nucleus raphes magnus

Nucleus raphes linearis

Pediculopontine nucleus (Ch5)

Lateral parabrachial nucleus

Central superior nucleus

Locus coeruleus (A6)

Nucleus of Koeltler-Fuse

Nucleus pontis oralis

Medial parabrachial nucleus

Nucleus raphes lateralis

Nucleus raphes lateralis

Nucleus raphes medialis

Nucleus gigantocellularis

Nucleus paragigantocellularis

Nucleus raphes obscurus

A5

Central nucleus of the medulla oblongata: lateral subnucleus

Central nucleus of the medulla oblongata: medial subnucleus

Lateral reticular nucleus

Paramedian reticular nucleus

Retroambigus nucleus

Intermediate zone of the spinal cord
Chemoreception and wakefulness

CO2 stimulates serotoninergic neurons. Respiratory motoneurons are stimulated via 5-HT, TRH and SP. Neurons in pre-BötzC are stimulated through 5-HT4a, 5-HT2a as well as neurokin1 (NK1).

- Reversal of respiratory rhythm uninduced by anaesthetics (Fentanyl)
- Projekcie do všetkých hlavných respiračných jadier (NTS, NA, preBötz, Bötz complex, XII i frenikové motoneuróny).
Arousing Diffuse Modulatory Systems (ADMS)

- Norepinephrine (NE): locus coeruleus (LC),
- Serotonin (5-HT): raphe nuclei,
- Dopamine (DA): substantia nigra (SN), ventral tegmental area (VTA), ventral periaqueductal grey (vPAG).
- Acetylcholine (Ach): laterodorsal, peduncular - pontine tegmental nucleus (LDT/PPT), basal forebrain (BF)
- Histamine (Hi): tuberomamilar nuclei (TMN)

Minimal neuronal substrate – experimental anesthesia

Principal parts of consciousness generator

Principle of internal and external awareness network

Continuum of behavioural states

Awareness of the self in the world around

- Myslenie (Thinking, reasoning)
- Explicit memory (Explicitná pamäť)
- Atentiveness (pozornost')
  - selective (selektívna)
  - general (všeobecná)
- Implicit memory Implicitná pamäť

Advanced consciousness (Pokročilé vedomie)

Awareness of the momentary world around (register)

- Alertness (čulost')
  - selective (selektívna)
  - general (všeobecná)
- Implicit memory Implicitná pamäť

- Orienting reflexes (Orientačné reflexy)
- Orienting responses (Orientačné odpovede)

Basal consciousness (Základné vedomie)

Wakefulness

Non-REM sleep

REM sleep
Disorders of consciousness
Disorders of consciousness

**QUANTITATIVE DISORDERS**
Decreased awareness

- Transient
  - Syncope
  - Epilepsy
- Brief
  - Absence

- Prolonged
  - Light forms
    - Somnolency
    - Obtundation
  - Advanced forms
    - Stupor (Precoma)
    - Coma

**QUALITATIVE DISORDERS**
Altered consciousness

- Light & Transient
  - Letargy
  - Disorientation
  - Confusions

- Advanced
  - Confusional state (Amentia)
  - Delirium
  - Obnubilation (Black out)

**SELF-IDENTIFICATION DISORDERS**
Depersonalisation
Acoenesthesia, Autotopagnosia
Anosognosia, etc.
Quantitative disorders of consciousness

- **Transient (sec- min)**
  - **Syncope (faintness)** short disorder of consciousness
  - **Systemic hypotension, resp. collaps** (postural/ orthostatic synkopa), kardiálne (ischémia srdca, vazovagálna synkopa, ASM)
  - **Disordered redistribution of blood** – changes in intrathoracic pressure (cough syncope, laugh syncope, food jedlo (postprabdial defecation), psychogenic factors, (neurogenic syncope)
  - **Changes in vessel lumen** (vertebro-basilar artery insufficiency, carotic stenotisation)
  - **Disorders in electric stability** (brain commotio, brain contusion, electrical current shock, epilepsy, electroconvulsions)

- **Prolonged (hod- dni- týždne)**
  - **Somnolency, lethargy** – pathological sleepiness, waking up upon light stimuli (opening eyes, orientation), response are correct, targeted, make sense, but are slowed
  - **Obtundation** – communication is difficult, person spontaneously fall asleep, can be waken up by stronger mechanicâ stimuli (rarely verbal), responses are not so precised, limited, not comprehesive, disorientation
  - **Stupor (precome)** – deep unconsciousness; person reacts to painful stimuli pain stimuli; reactions are delayed little localized, sporadic movements, verbalisation
  - **Coma** - total unreactivity to outer stimuli, no spontaneous motor response, eyes are closed, breathing is shallow, vegetative responses present
## Causes of disordered consciousness

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<th>Causes</th>
<th>Lesions</th>
<th>Lesions</th>
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<td><strong>Supratentorial Lesions</strong></td>
<td>Epidural or Subdural Hematoma</td>
<td>Intraparenchymal hemorrhage</td>
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<td></td>
<td>Large Isquemic Infarction</td>
<td>Trauma</td>
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<td>Abscess</td>
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<td>Tumor</td>
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<tr>
<td><strong>Infratentorial Lesions</strong></td>
<td>Basilar artery thrombosis</td>
<td>Ischemic Cerebellar Infarction</td>
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<td></td>
<td>Pontine or Cerebellar Hematoma</td>
<td>Tumor</td>
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<td>Abscess</td>
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<tr>
<td><strong>Diffuse Encephalopathies</strong></td>
<td>Hypoglycemia</td>
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<td></td>
<td>Drug Intoxication</td>
<td>Meningitis and Encephalitis</td>
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<td>Hepatic Encephalopathy</td>
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<td></td>
<td>Hyperosmolar States</td>
<td>Myxedema</td>
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<td></td>
<td>Hyponatremia</td>
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<td></td>
<td>Global Cerebral Ischemia</td>
<td>Hypercarbia</td>
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<td></td>
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<td>Thiamine Deficiency</td>
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<td>Hydrocephalus</td>
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<td><strong>Psychogenic</strong></td>
<td>Catatonic States</td>
<td>Acute psychotic delirium</td>
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<td></td>
<td>Hysteria-malingering</td>
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</tbody>
</table>

Sir Graham Teasdale & Bryan Jennett (1926-2008) – Glasgow neurosurgeons, introduced Glasgow Coma Scale (GCS); 1974 Lancet, entitled "Assessment of coma and impaired consciousness: a practical scale"
Syncope – four stage management

Step 1: Electrocardiogram
All patients with syncope should undergo electrocardiography. If ECG is abnormal, confirmatory testing and appropriate therapy should be instituted.

Step 2: Echocardiography
In most patients without a diagnosis, a structural evaluation with echocardiogram is required.

Step 3: Head-up tilt-table test
Positive neurocardiogenic tilt-table test shows drop in BP and heart rate. Should be considered if steps 1 and 2 are negative.

Step 4: Monitoring for symptom-rhythm correlation
Normal tilt-table test shows maintenance of normal BP and heart rate.

Ambulatory monitoring recommended for patients with negative evaluation; duration of monitoring dependent on frequency of episodes; for daily symptoms, 48-hour monitor adequate.

Holter monitor
# Pupils

<table>
<thead>
<tr>
<th>Pattern</th>
<th>Description</th>
<th>Lesion</th>
</tr>
</thead>
</table>
| Dilated (bilateral)  | $\varnothing$ 7 mm, (-) reaction to light | • Transtentorial herniation of both medail temporal lobes  
• Intoxication: anticholinergics, sympathicomimetic drugs |
| Narrowed (bilateral) | $\varnothing$ 1-1.5 mm    | • Intoxication by opiates, organophosphates, cholinomimetics, miotic eye drops  
• Pontinne haemorrhage, Neurosyfilis |
| Asymmetric (anisocoria) | $\varnothing$ 1 mm difference | • Normal ~ 20% of population; (+) photoreaction  
• (-) photoreaction - dilation: ipsilateral pressure in mesencephalon + nucl..III (tumors, bleeding) |
| Fixed pupils         | $\varnothing$ 5 mm, (-) fotoreaction | • Mesencephalic laesions |
Qualitative disorders of consciousness

- **Lighter forms** (often combined with quantitative disfunctions; patients are mostly aware of disorder)
  - Apathy, letargy – similar to tiredness (e.g. depression, toxic, infection, ictus, metabolic)
  - Disorientation – slowness, blunted attentiveness and preparedness, (e.g. altitude sickness, hypoxia, cold, starvation, hypoglycemia)
  - Confusion – a person is not orientated to time, place and/or person; responses or behaviours to situations may be inappropriate. agitation, restlessness with sleepiness (somnolence) or even stupor (difficult to arouse or state)

- **Progressive forms (altered state of consciousness, cognition)**
  - Acute confusion state – disordered perception, disorientation, disorders of memory (intoxication – posttraumatic, post-narcotic, inflammation)
  - Delirium – disorder of thinging, perception, hallucinations, disorders of memory, agitation, sleepness, amnesia, organic damage (nádory, toxic, abstinencia)
  - Obnubilation (blackout) – disorder of perception of reality; amnesia
  - Depersonalisation
Acute confusional state

(Alternatives: confusional state, organic brain syndrome, confusional insanity, transient psychotic reaction, organic psychosyndrome; Meynert-Korsakoff syndrome)

Characteristics: amentia (behaviorálna demencia) patrí aj ku kognitívnym poruchám)
- forma delíria s prevahou deficitu percepcie, pozornosti a orientácie, dezorientovaný v čase a priestore, často je nadmieru aktívny – úniky; aktivna obrana
- schopnost rozpadnutia (anterográdna, retrográdna amnézia), zasteté vedomie (neschopnost presunu a fokalizácie pozornosti)
- Môže sa prejaviti úzkost', strach, hnev, eufória, tras, potenie, búšenie srdca.

Causes:
- Intoxication (37% cannabis, marihuana), pooperative (probably post-narcotic or combination of effects)
  (in elderly suprisingly high incidency after hip fracture surgery), tramadol, infections,
- Diabetes – hypoglycaemia, cardiac decompensation, infarction,
- Confusional arousals (sleep terror),
- Alzeheimer disease, kidney failure (uremia)
- Dehydratation, disorders of electrolytes
Delirium

Delirium due to the substance withdrawal e.g., alcohol, benzodiazepines, or nicotine, etc.

- **Mechanisms:** imbalance of inhibitory and excitatory neurotransmitter systems in brain; alcohol consumption leads to *inhibition* of excitatory NMDA receptors and *activation* of the inhibitory GABA-A receptoric effect (*cerebral inhibition*); withdrawal leads to disinhibition of brain and reinforcement of *alarm response* -> dopaminergic and noradrenergic effects - predominant sympathetic activation and a tendency toward epileptic seizures.
- benzodiazepine withdrawal causes delirium by way of decreased GABA-ergic transmission; epileptic seizures may occur.

Delirium not due to substance withdrawal

- **Mechanisms:** many different; final common pathway of delirious states seems to consist of a *cholinergic deficit combined with dopaminergic hyperactivity*.
- significance of other neurotransmitters – serotonin, noradrenaline s less clear

**Figure 1. Factors Contributing to Changes in Neurotransmitters, Leading to Delirium**

**Delirium Subtypes**

- Hyperactive Delirium
  - Combative
  - Agitated
  - Restless
- Lethargic
  - Sedated
  - Stupor
- Mixed Delirium
- Hypoactive Delirium
  - Hyperactive Delirium
  - Mixed Delirium
  - Hypoactive Delirium
Delirium

Withdrawal symptoms in chronic alcohol abuse

Stage I: Personality changes, vacant stare
Stage II: Lethargy, flapping tremor, muscle twitching
Stage III: Noisy, abusive, violent
Stage IV: Coma

Decrease in BAC results in:
- Hypertensive response
- Tremor
- Sweating
- Hallucinations
- Anxiety, cognitive defects, inner voice, confusion
- Generalized seizures

Electroencephalogram changes
- Ankle clonus
- Knee clonus
- Babinski sign

Stages in development of delirium due to the substance withdrawal
Postcomatous disorders of consciousness
Postcomatous disorders & coma like states

COMA

- RENEWAL OF CONSCIOUSNESS
- VEGETATIVE STATE
- LOCKED IN SYNDROME
- BRAIN DEATH

MCS (MINIMALLY CONSCIOUS STATE)

- PERSISTENT VEGETATIVE STATE (> 1 month)

RENEWAL OF LIMITED CONSCIOUSNESS

- PERMANENT MCS
- RENEWAL OF LIMITED CONSCIOUSNES
- PERMANENT VEGETATIVE STATE (> 1 year)
- BRAIN DEATH
Number of published papers per year on patients with disorders of consciousness and evolution of the terminology. Medline search (7/2013) keywords used were ‘coma’, ‘vegetative state’, ‘unresponsive wakefulness syndrome’, ‘minimally conscious state’ and ‘locked-in syndrome’.
Fred Plum (1924 – 2010) – american neurologist; he introduced the term "locked-in syndrome: together with Dr. Byron Jennett, they introduced the term "persistent vegetative state".


Schiff, N.D. (1997) tzw. deep brain stimulation

Laureys, S (2005): usage of fNMR, PET in research


Postcomatous disorders & coma like states

Vegetative state (Apallic syndrome 1940, Coma vigil 1952)
Unresponsive wakefulness syndrome (UWS) (2010)

**Etio:** traumatic/ atraumatic, drug dependencies/ independency
- severe global metabolic, toxic, ischaemic, traumatic cortical injury (cardiac arest, brain surgery, etc.)
- bilateral damage of frontal lobe, lesion of upper brainstem,

**Sy:**
- return to wakefulness (eye opening), but without awareness of self and environment; with no communication, no visual contact with persons, nor surroundings, no verbal response
- without paralysis, spontaneous movements exist, normal reflexes (breathing)
- bulbar reflexes present, eye-ball movement, swallowing, yawing
- occasionally decerebration or decortication rigidity, Babinski’s sign
- pupillary response to light often not present on both sides
- vegetative response normal or hyperactive (CVS – tachycvardias, hypertensive episodes, termoregulation, neuroendocrine, bowel movement)
- without sensory disturbances, reactions to pain are present but delayed

- **Persistent vegetative state** – lasting > 1 month
- **Permanent vegetative state** – lasting > 1 year after traumatic brain damage or > 3 month after atraumatic brain damage
Post-comatose recovery outcomes

- Persistent vegetative state
- Akinetic mutism

Patients can imagine various activities; there is lack of outer manifestations of awareness

Using NMR and PET scans in diagnostics
Locked in syndrome (LIS)
- **Alt.**: pseudocoma, deafferentation sy.
- **Etio**: rare clinical entity results typically from a ventral pontine infarction (rarely pontine tumours, haemorrhage, central pontine myelinolysis, head injury or brain stem encephalitis.) that damages cortico-spinal tracts below the level of the III.n. nuclei., leading to complete paralysis of voluntary muscles except for eye movements
- **Sy**:
  - total paralysis (tetraplegia loss of voluntary movement);
  - Bulbar parlysis (dysarthria, amimia, dysphagia) – artefitial feeding
  - patients can open their eyes and elevate and depress eyes to command. ; horizontal eye movements are usually lost
  - Patients are on artefitial ventilation
  - No sensoty defect; reactivity to pain present
  - Recovery is exceptional

Other disorders of consciousness

Akinetic mutism
- **Etio:** first described in patients that suffered from diencephalic damage; lesions that interfere with reticular cortical/integration (but spare the corticospinal pathways); hydrocephalus, tumors close to 3rd ventricle; gross bilateral laesiaon of gyrus cinguli, frontal lobe, periaqueductal mesencephalon
- **Sy:** immobility, eye closure, little or no vocalisation; little movement to painful stimuli
- the relative paucity of signs indicating damage to descending motor pathways, despite the immobile state (as in LIS); spasticity and rigidity are not usually evident (as in vegetative state)
- Sleep/wake cycles can be seen, as indicated by eye opening.
- !! debate about whether or not the syndrome should be clearly differentiated from the vegetative state; indistinguishable from early stages of the vegetative state

Abulia = lighter form of akinetic mutism: hypokinesis (bradykinesia instead of akinesia (delayed verbal and other motor reactions)

Psychogenic coma (Hysteric pseudocoma)
- eyelids are kept firmly shut and are resistant to opening
- oculocephalic responses are unpredictable (nystamus is evident on caloric testing)
- motor tone is normal or inconsistent and limb reflexes retained.
- EEG shows awake rhythms
Catatonia

1874-Karl Ludwig Kahlbaum (Die Katatonie oder das Spannungsirresein)

**Etiology:**
- associated with psychiatric illness (affective) schizophrenia; metabolic/ drug induced disorders; no organic lesions

**Symptoms:**
- no spontaneous movement, patients seem unresponsive to their surroundings, but appear conscious. Neurological examination is normal. passive limb positioning in postures “waxy flexibility”.
- eyes open and unblinking, pupils dilated but reactive, oculocephalic responses absent or impaired, and caloric responses intact. EEG: low voltage, fast record rather than the “slowing” of true coma.
- difficult to distinguish from organic disease, particularly in lethargic unresponsiveness
## Comparison of coma-like disorders of consciousness

<table>
<thead>
<tr>
<th></th>
<th>Minimally conscious state</th>
<th>Vegetative state</th>
<th>Coma</th>
<th>„Locked in“ syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vigilant - communication</td>
<td>Reduced, partial</td>
<td>Absent</td>
<td>Lost</td>
<td>Full</td>
</tr>
<tr>
<td>Sleep - Wake cycle</td>
<td>Present</td>
<td>Present</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>Motor functions</td>
<td>Localized response to pain; touches and hold things;</td>
<td>Minimal spontaneous movements; withdrawal response to pain</td>
<td>Reflex and postural responses</td>
<td>Quadruplegia</td>
</tr>
<tr>
<td>Auditory functions</td>
<td>Localises source of sound; turn the head</td>
<td>Startle (orientation) after recovery some remember</td>
<td>None; after recovery some remember what they heared</td>
<td>Present</td>
</tr>
<tr>
<td>Visual functions</td>
<td>Visual fixation (prezerá; akoby do prázdna)</td>
<td>Startle (orientation), no focusing</td>
<td>No</td>
<td>Present</td>
</tr>
<tr>
<td>Communication</td>
<td>No words, sounds</td>
<td></td>
<td>No</td>
<td>Present; limited to vertical eye movement</td>
</tr>
<tr>
<td>Cognition understanding</td>
<td>Present but limited</td>
<td>Limited, little or missing</td>
<td>No</td>
<td>Present; cannot react</td>
</tr>
<tr>
<td>Emotions</td>
<td>Smiling, crying, clenching, mimics</td>
<td>Reflex smiling, crying</td>
<td>No</td>
<td>Present ; cannot react</td>
</tr>
</tbody>
</table>

Assessment

Standardized validated scales
- bedside assessment neurologist, internist
  - Glassgow coma scale (GCS)
  - Coma Recovery Scale-Revised (CRS-R)
  - Full Outline of Unresponsiveness scale (FOUR)

Specific assessment tools:
- mirror (to evaluate visual pursuit), patient’s own name (to assess auditory localization),
- self-referential stimulus (their own face)
- written commands (absence of response to oral commands)

What are not the signs of consciousness
- blinking in response to a threat (blink reflex may be elicited due to corneal stimulation by air flow)
- visual fixation (at least in patients with anoxia)
- resistance to eye opening is related to consciousness
Frontal-parietal disconnection

- PET studies hypometabolism in frontal-parietal regions
- Strata spojení medzi frontálnou a parietálnou kôrou pri VS a propofolovou anestéziou

5
Body - related agnosias
<table>
<thead>
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<th>Disorder</th>
<th>Description</th>
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<tbody>
<tr>
<td>Autotopagnózia</td>
<td>neschopnosť rozoznať dráždené miesto na povrchu tela</td>
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<tr>
<td>Dermoalexia</td>
<td>neschopnosť rekonštrukcie priestorovej mapy povrchu tela. Prejavuje sa tým že postihnutý nie je schopný rozoznať rôzne tvary, písmená ktoré sa mu kreslia na kožu</td>
</tr>
<tr>
<td>Alloestézia</td>
<td>je porucha stálosti lokalizácie podnetu. Pri opakovanom dráždení toho istého miesta cíti chorý dráždenie stále v iných oblastiach</td>
</tr>
<tr>
<td>Dyzestézia</td>
<td>je porucha, pri ktorej sa podnet jednej modality interpretuje ako vnem inej modality, napr. dotyk ako pálenie, chlad ako teplo a pod</td>
</tr>
<tr>
<td>Stereoagnózia</td>
<td>strata schopnosti rozoznať predmety hmatom pri zatvorených očiach. Táto porucha sa prejavuje dvomi, relatívne samostatnými formami</td>
</tr>
<tr>
<td>Amorfognózia</td>
<td>neschopnosť rozoznať tvary predmetov (napr. kocku, guľu, knihu). Porušená je centrálna integrácia podnetov z povrchových i hlbokových mechanoreceptorov i proprioreceptorov</td>
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### Qualitative disorders

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Description</th>
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</thead>
<tbody>
<tr>
<td>Ahylognózia</td>
<td>Neschopnosť rozoznávať látkovú podstatu ohmatávaných predmetov (napr. sáčok s vodou, pieskom a pod.). Porušená je centrálna reprezentácia podnetov z termoreceptorov chladu, tepla a povrchových mechanoreceptorov.</td>
</tr>
<tr>
<td>Akinestézia</td>
<td>Neschopnosť rozoznávať pohyb tela a jeho jednotlivých segmentov, napr. chôdzu, pohyby ruky nohy a pod.</td>
</tr>
<tr>
<td>Statanestézia</td>
<td>Neschopnosť rozoznávať statické postavenie tela alebo jeho jednotlivých častí, napr. stoj</td>
</tr>
<tr>
<td>Hypopalestézia</td>
<td>Neschopnosť vnímať hlúbkovú tzv. vibračnú citlivosť. Vzniká poruchou rýchlo sa adaptujúcich vibračných mechanoreceptorov v tlanive okolo svalov a kĺbov.</td>
</tr>
<tr>
<td>Hypobarestézia</td>
<td>Neschopnosť vnímať tupý, do hlúbky pôsobiaci tlak. Vzniká poruchou pomaly sa adaptujúcich nízkoprahových hlúbkových mechanoreceptorov.</td>
</tr>
<tr>
<td>Acoenesthesia</td>
<td>Neschopnosť vnímať vlastné telo a jeho jednotlivé časti. Vzniká integratívnom poruchou barorecepcie, termorecepcie, povrchovej a hlavné hlúbkovej mechanorecepcie, propriocepcie.</td>
</tr>
<tr>
<td>Amorphognosia</td>
<td></td>
</tr>
</tbody>
</table>
Self-location, self-consciousness

Gyrus angularis – speech processing (aphasia), acalculia, space cognition, attentiveness, memory (Brodmann 39)

Persinger, M: Stimulation of temporal lobe by weak magnetic stimuli may evoke special feeling and mystical experiences of encountering with God (well-being, absolute safety, endless love, http://en.wikipedia.org/wiki/God_helmet)