

# ANATOMO-CLINICAL CORRELATIONS (NEUROLOGICAL AND PSYCHOPATHOLOGICAL) IN THE CEREBRO-VASCULAR DISEASE

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**Keywords:** cerebral anatomy, psychopathological manifestations, functional areas

**Abstract:** Brain pathology due to cerebrovascular disease reflects the concerted effort of anatomists, neurologists and psychiatrists to accurately identify etiology, topography and different clinical forms of its expression in the common goal of ensuring optimal therapeutic management. Anatomic and functional, frontal lobe presents six distinct areas: primary motor area, premotor area, frontal eye field, supplementary motor field, prefrontal area and speech area of Broca. Parietal cortex comprises four functional areas: primary somato-sensory, secondary somato-sensory, gustatory and associative. Temporal lobes contain primary auditory area and areas associated to emotions and superior mental functions. Occipital lobes contain primary visual area and association area. Cerebral lobes lesions causes different neurological and psychopathological manifestations depending on location and affected vascular territory. In the present paper we tried to group these psychopathological phenomena into psychic life spheres for a complex understanding of manifestations associated to cerebrovascular disease.

**Cuvinte cheie:** anatomie cerebrală, manifestări psihopatologice, arii funcționale

**Rezumat:** Patologia cerebrală datorată bolii cerebro-vasculare reflectă efortul concertant al anomiștilor, neurologilor și psihiatrilor de a identifica cu acuratețe etiologia, topografia și diferitele forme clinice de exprimare în scopul comun de a asigura un management terapeutic optim. Anatomofuncțional, lobul frontal prezintă șase zone distincte: aria motorie primară, aria premotorie, câmpul ocular frontal, câmpul motor suplimentar, aria prefrontală și aria vorbirii a lui Broca. Cortexul parietal cuprinde patru arii funcționale: somato-senzitivă primară, somato-senzitivă secundară, gustativă și asociativă. Lobii temporali conțin aria auditivă primară, precum și ariile asociate emoțiilor și funcțiilor mentale superioare. Lobii occipitali conțin aria vizuală primară și de asociație. Leziunile lobilor cerebrali determină manifestări neurologice și expresii psihopatologice variate funcție de localizare și teritoriul vascular afectat. În lucrarea prezentă s-a tentat gruparea acestor fenomenologiei psihopatologice pe sfere ale vieții psihice pentru o comprehensiune complexă a manifestărilor asociate bolii cerebro-vasculare.

## SCIENTIFIC ARTICLE OF THEORETICAL PREDOMINANCE

The interplay of psychopathological phenomenology and neurological disorders is a phrase long debated by involved medical specialties. Brain pathology due to cerebrovascular disease reflects the concerted effort of anatomists, neurologists and psychiatrists to accurately identify etiology, topography and different clinical forms of its expression in the common goal of ensuring optimal therapeutic management. This paper proposes an anatomical-neuro-psychiatric synthesis of lesions in cerebrovascular disease, with the expectancy of an eloquent organization of treated topics.

**Frontal lobes** represent a third of cerebral hemispheres mass and are located anteriorly of Ronaldo fissure and above Sylvius fissure. Anatomic and functional, each frontal lobe presents six distinct areas: primary motor area, premotor area, frontal eye field, supplementary motor field, prefrontal area and speech area of Broca (3, 6).

**Primary motor area** corresponds to Brodmann area 4 and occupies the posterior part of precentral gyrus and adjacent part of paracentral lobe; it comprises ascending frontal convolution, Rolandic convolution, precentral area or primary motor cortex. In the motor cortex, somatotopic localisation of

contralateral movements is represented from top to bottom with inferior limbs in paracentral lobe, upper limbs in dorsal part of precentral gyrus and the face most ventrally. Fineness degree associated with a particular movement is directly proportional to the size of the area which represents different movements.

**Premotor area**, area 6, is responsible for complex motricity, being located in the anterior part of precentral gyrus, even though it increases dorsally and includes the more posterior part of superior frontal gyrus.

**Frontal eye field**, area 8, is located immediately anterior of area 6, mainly in middle frontal gyrus. Bilateral destruction of the field determines alteration of voluntary eye motricity and of visual research tests; unilateral lesion determines eye deviation to the lesion.

**Supplementary motor field** represents extensions of areas 6 and 8 into the medial part of superior frontal gyrus of paracentral lobe. This field is responsible for language initiation and complex movements which involve different segments of the body (postural responses, performance of sequential movements of fingers and even eyes) (3).

**Prefrontal area**, with 6, 8 and 45 areas, is located on lateral, medial and inferior surface of anterior frontal lobe. Being called also associative frontal cortex, it represents almost

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a quarter of the cerebral cortex and is divided into orbital and lateral regions. Orbital region is responsible for visceral and emotional activities, and the lateral region for certain intellectual functions as planning, conceptualization, reason and solving situations.

**Broca area** (44 and 45 areas of inferior frontal gyrus) is the motor centre of speech and contains motor programs for word generation. By projection of fibers to the motor cortex, it controls also the muscles that facilitate the articulation of words (2, 6).

Frontal lobe lesions cause neurological symptoms and psychopathological expressions, depending on the affected areas:

**A. Neurological symptoms:** contralateral paralysis and paresis, more pronounced in the distal parts of limbs and inferior part of the face (area 4). Middle cerebral artery thrombosis (sylvian artery) determines classic cortical hemiplegia, characterized by paralysis, hemiplegic posture, central facial paresis, hypertonia and spastic gait (4). The lesion of middle cerebral artery which irrigate the convex face of frontal lobe, determines contralateral hemiplegia with facio-brachial predominance, and the lesion of anterior cerebral artery (which irrigate the internal part of frontal lobe) determines hemiparesis predominantly crural, gripping reflex and mental disorders. The lesion of area 8 determines transient paralysis of conjugated eye movements of the opposite site. Nonfluent aphasia appears in lesions of 44 and 45 areas (Broca area). Other manifestations may be included in disorders of movement regulation: lack of use of the opposite limbs, difficulty moving from one type of motion to another, astasia-abasia, latero-compulsion, gait apraxia, ataxia.

**B. Psychopathological manifestations:** in 75% of cases these are early events and precedes the neurological manifestations.

**1. In psycho-cognitive area: attention disorders** – decreased ability to concentrate, decrease of attention volume and distribution, easy distraction of attention; **memory problems** – due to frontal lesions that suppress performance-based programs which involve memorization during the execution of tasks; it has been demonstrated the existence of a specific memory deficit in frontal lobe lesion during recent memory tests. One of the most recent findings shows the importance of dorsomedian medial frontal cortex in achieving memory. Prefrontal lesions due to a stroke can cause memory impairment through an inability to reconstruct a logical sequence of facts. In medial frontal lesions and bilateral lesions, patients have disorientation in time and space and confabulation phenomena (1). **Cognitive disorders:** loss of abstract thinking, with inability to make decisions and refuge in concrete thinking; bradypsychic-bradykinesia syndrome (akinetic mutism); Korsakov syndrome; catatonic episodes; pseudodementia syndrome (significant cognitive impairment with contrasting appearance with Q.I. which is much higher in reality); schizophreniform disorders – there is a relationship between frontal cortex atrophy and events that resemble the clinical picture with negative symptoms of schizophrenia; it seems that frontal hypodopaminergic activity would have a role in this (5). In the field of verbal expression - impoverishment and decreased spontaneity, anomie phenomena and echolalia. **Disorders of consciousness:** reduplicative **paramnesia syndrome**, Capgras syndrome, Cotard syndrome and alien hand syndrome.

**2. In the area of affectivity:** depression (pseudodepression) – in cortical and subcortical lesions; mania (pseudoe expansivity) – in fronto-orbital lesions. The manic syndrome (moria) seems to be due to a noradrenergic hyperactivity.

**3. In the emotional and behavioral area:** apathy, lack of initiative, inability to carry out the started activities, indifference towards the environment; stereotypies; persevering – with delay in movement initiation, a sudden stop of motion during execution and also a tendency to fixation in attitudes. Usage behavior (described by Lhermitte) – in which the simple presentation of certain objects to patients with frontal lesions induces them the order to grab and use this objects; imitation behavior – in which patients always imitate the examiner gestures without any order from them. **Personality change** – is quite evident in two directions: lesions of lateral or dorso-lateral face of frontal lobe causes rather akinetic changes and toward depression; lesion of orbital face (orbito-basal) of frontal lobe determines moriatic behavior – impulsive and foolish; sometimes a mixed picture can appear that combines symptoms of both groups described above (2).

**Parietal lobes** - higher developed structures on phylogenetic scale, are responsible for spatial and visual integration and mental map of space, representing more than one fifth of entire cerebral cortex. Parietal lobe is delineated anteriorly on external face by Rolando fissure and posteriorly by parieto-occipital sulcus and by an imaginary line that continues this sulcus on the external face of the brain; inferior limit is represented by sylvian fissure and an imaginary line that continues this fissure to the occipital lobe (3). Internal face of this lobe includes especially cadrilater lobe. Postcentral gyrus (3, 2, 1 areas) is delineated posteriorly by vertical branch of anterior parietal sulcus and anteriorly by Rolando fissure. Areas 5 and 7 (superior parietal lobe) are defined in relation to inferior parietal lobe through horizontal branch of anterior parietal sulcus. On the internal face of hemisphere, the anterior limit of parietal lobe is represented by an imaginary extension of Rolando fissure while posterior limit is represented by parieto-occipital sulcus.

Parietal cortex comprises four functional areas: primary somato-sensory, secondary somato-sensory, gustatory and associative.

**Primary somato-sensory area:** primary projection area of elementary sensitivity (Brodmann areas 2, 3, 1, represented by three longitudinal areas), occupies postcentral gyrus and adjacent part of paracentral lobe. Area 3 comprises the cortical tissue from the floor and posterior wall of central sulcus; area 1 is located in the anterior two-thirds of the convex surface of postcentral gyrus and area 2 in the remaining third of convex surface and adjacent anterior wall of postcentral sulcus. Somatotopically, representations is contralateral with parts of the head located ventrally, inferior limb medially in the posterior part of the paracentral lobe and upper limb dorsally in postcentral gyrus (6).

**Secondary somato-sensory area:** it extends from the parietal operculum (cortical tissue contiguous with postcentral gyrus which form the superior wall of lateral sulcus) to the posterior part of the insular cortex. Painful sensation is perceived in this site.

**Primary gustatory cortex:** it comprises the anterior part of parietal operculum and is located in area 43. It extends along the wall of lateral sulcus to the insular cortex and is adjacent to tongue region from primary sensory and motor areas (2).

**Parietal association areas:** consist of inferior and superior parietal lobes. Inferior parietal lobe includes two gyri, area 40 and 39, which receive input both from parietal lobe and frontal, occipital, temporal and limbic lobes. Superior parietal lobe consists of areas 5 and 7, which receive input from primary somato-sensory area and also from visual and motor areas of the cortex. These areas process tactile and visual information being

intimately related to cognitive function over the body and surrounding objects, and have important implications for sequentiality of performing tasks. Their lesion determines astereognosia and denial syndrome.

Classic, it is estimated that right and left parietal lobes respect the general functioning of cerebral hemispheres, namely left cerebral hemisphere controls memory and symbolic or abstract thinking, and right hemisphere is responsible for spatial thinking and memory.

Parietal lobe lesions may determine neurological disorders (sensitive, of discriminatory and motor sensitivity) and psychopathological manifestations:

**A. Neurological manifestations:**

Motor disorders that consist of motor hemiaspontaneity and retrorolandic forms of motor neglect. True motor deficits can occur only in the case of combination of the lesion with a lesion of ascending frontal convolution. It can occur: parietal ataxia (by disturbance of deep sensibility), parietal pseudoathetosis, ataxic hemiparesis. Posterior parietal lesions and lesions of parieto-occipital intersection (nondominant) lead to dressing apraxia and constructive apraxia with hemiasomatognosia (4). As well, ideational and ideomotor apraxia has been reported. Parietal lobe lesion may lead to moderate contralateral muscle atrophy especially at the shoulder and hand, mnestic aphasia and dyslexia.

Disorders of body scheme: parietal lobe plays a fundamental role in complex spatio-temporal integration which is the base of our body image. Parietal connections with the other lobes permit a unified synthesis of body image. Temporal lobe lesion leads to the occurrence of Gerstmann syndrome (digital agnosia, acalculia, left-right disorientation, agraphia, constructive apraxia), Anton-Babinski syndrome (anosognosia, anosodiaphoria, asomatognosia).

Taste disorders: lesion of area 43 leads to loss of taste and contralateral gustatory anesthesia.

Sensitivity disorders: primary cortex lesion determines the loss of discriminative tactile sensitivity and position sense on the contralateral side. Stimulation of this area leads to numbness or paresthesias. Lesions of areas 5 or 7 determine sensitivity disorders in the opposite hemisphere that comprises especially astereognosia, epicritic sensitivity disorders, tactile apraxia.

Sensory disturbances: Partial somato-sensory epilepsy – it consists of paresthesias occurrence, rarely pain and sensation of electric shock that starts contralateral to the lesion. The lesion can be located clinically: if it is located on the internal face of parietal lobe, paresthesias occur initially in the leg; if the seizure comprises initially the hemiface and thumb, the lesion is located on the external face of contralateral parietal lobe. If the focal sensory seizure irradiates to the motor cortex, it can be followed by a jacksonian seizure initially which can become generalized afterwards, causing the grand-mal seizure.

**B. Psychopathological manifestations:**

Memory disorders: in unilateral parietal lesions, short-term memory or working memory are affected, long-term memory being rather affected in temporal lobe damage. While left parietal lobe lesions determine in righthanded persons retaining disorders of verbal material, lesions of right parietal lobe determine retaining disorders of nonverbal material. Bilateral lesions may cause gradually the development of a dementia with variable severity.

Confusional syndromes: confusional states may occur abruptly after a parietal lesion, that can evolve as a temporal disorientation and especially in space disorientation, nocturnal restlessness, confabulations, sometimes even paranoid delusions. A characteristic of these symptoms is the variability, occurring 2-3 hours per day, otherwise the patient presenting an

adequate behavior (it occurs especially in right parieto-occipital lobe damage).

Depressive syndromes: occur in posterior parietal lobe damage, while the damage of anterior area causes apathy (5, 1).

Personality disorders: occur in parietal lobes damage (left – disturbance of abstract symbolic integration; right – temporo-spatial orientation) and consist of irritability phenomena alternating with depression status, psycho-motor instability, aggressiveness, pseudo-psychotic behavior (schizo-paranoid syndromes accompanied sometimes by hallucinations) (2).

**Temporal lobes:** represents a quarter of entire cortex and contains primary auditory area and areas associated to emotions and superior mental functions (memory and speech). It has the following functions: sensorial, visceral-vegetative, affective-motivational, prosexic, cognitive.

Primary auditory cortex (areas 41 and 42) is found in transverse temporal gyrus of Heschl. Area 41 is mostly located in anterior gyrus. Area 42 is located adjacent to area 41, and joined there is associative auditory part of area 22. Electrical stimulation of the auditory area leads to continuous ring-type sound and stimulation of the adjacent part of area 22 produces bell-sound. A unilateral lesion produces difficulty in recognizing the distance and direction from which sounds are generated, and bilateral lesions lead to significant hearing loss. Posterior parts of temporal lobe are designed to record experiences (2, 3). Stimulation of this area produces illusions of past events. Left posterior temporal lesion can affect verbal learning, while the right can affect visual information learning. Bilateral lesions of areas 20 and 22 can lead to prosopagnosia.

**A. Neurological manifestations:** sensory disorders – cortical deafness, acoustic agnosia, sensory aphasia Wenicke, olfactory, gustatory (rare) and auditory hallucinations; balance disorders; hemianopsia; visceral-vegetative disorders – appear as aura or accompanying phenomena in clinical form of temporal epilepsy; aphasia (2). Nondominant temporal lobe is considered clinically silent, its lesion causing just hemianopsia by involving geniculo-calcarine radiation.

**B. Psychopathological manifestations:**

**a. Affective-motivational disorders:** stimulation of temporal cortex and amygdala nucleus is anxiogenic causing intense fear reactions. Their partial stimulation determines anger reaction. It is also met: persevering, indifference towards the environment, exacerbation or inhibition of primary biological needs.

**b. Prosexic and vigilance disorders:** temporal lobe, due to multiple connections, sets the relationship between waking state and prosexic function. Stimulation of temporal cortex can produce visual and auditory hallucinations, déjà-vu, déjà-connu, jamais-vu, jamais-connu states and its lesion can induce uncinate seizure (5). Lesions of temporal neocortex induce the so called dreamy-state and complex hallucinations and those of allocortex – visual and olfactory hallucinations.

**c. Cognitive disorders:** mnestic disorders (especially fixation memory), disorders of thought process development (in dominant lobe damage), inability to use visual and spatial information (in the non-dominant lobe damage).

**Occipital lobes** represent approximately one eighth of the cerebral cortex; they contain primary visual area and association area.

Primary visual cortex (striate area) is located in the walls of calcarine fissure gyri and receives optic radiations. Unilateral lesions of primary visual cortex (area 17) lead to contralateral homonymous hemianopsia.

Association areas are parastriate cortex (area 18) and peristriate cortex (area 19); they receive visual information from

striate areas and have a role in complex visual perception (color, movement, objects, direction); they mediate accommodation reflexes and eye movements, thereby decoding visual afferents (3). Lesions of these areas induce visual agnosia.

### ***A. Neurological manifestations***

Lesion of dominant occipital lobe leads to alexia, color agnosia, visual agnosia. Lesion of nondominant occipital lobe causes visual and spatial agnosia associated with prosopagnosia and metamorphopsia. In bilateral lesions may occur: Ballint syndrome (psychic paralysis of gaze, optic ataxia, visual hypoprosopia or simultagnosia), cortical cecity with agnosia of visual deficit and pupillary light reflex preservation without optic atrophy (4).

Occipital epilepsy may be accompanied by tonic and clonic, head and eyes movements, fluttering eyelids, vegetative disorders, thymic disorders and automatism, delusions and hallucinations.

***B. Psychopathological manifestations:*** delirium, psycho-motor agitation accompanied by aggressiveness (occurring more frequently in the temporo-occipital lesions); antero- and retrograde amnesia, temporo-spatial disorientation due to the loss of topographic memory - in some cases can lead to irreversible dementia; visual hallucinations, visual delusions (5).

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