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Historical Review

Anton, Balint, Charles Bonnet, and the Others: The ABC of Cerebral Visual Syndromes (A Historical Guide and an Update)

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ABSTRACT: Cerebral visual impairments have been of great interest to neurologists, ophthalmologists, and neuroscientists. Complicated or partial varieties related to cortical blindness are discussed in this review. They are a fascinating alphabet of eponymic clinical syndromes, bordering neurology, ophthalmology, and even psychiatry. Recent functional imaging and experimental studies have contributed further knowledge of cognitive visual organization in addition to the classical lesion evidence.

RÉSUMÉ : Anton, Bálint, Charles Bonnet et les autres : l'ABC des syndromes visuels cérébraux (guide historique et mise à jour). Les déficiences visuelles cérébrales ont suscité un grand intérêt chez les neurologues, les ophtalmologues et les neuroscientifiques. Les variétés complexes ou partielles liées à la cécité corticale sont abordées dans cet article. Elles constituent en effet une liste fascinante de syndromes cliniques éponymes qu'on peut situer à la frontière de la neurologie, de l'ophtalmologie et même de la psychiatrie. En plus des preuves lésionnelles classiques, des études expérimentales et des examens d'imagerie fonctionnelle récents ont par ailleurs permis d'approfondir les connaissances portant sur l'organisation visuelle cognitive.

Keywords: Visual cognition; neurosciences; neurology – behavioural; cortical localization

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Visual Syndromes Related to Cortical Blindness

Loss of vision due to impairment of the occipital cerebral cortex or geniculocalcarine pathways is called *Cortical Blindness*. Common causes are stroke, less frequently anoxia, trauma, encephalopathies, angiography, cardiac surgery, preeclampsia,[1] posterior reversible encephalopathy, and even Covid vaccination.[2] On examination, there is loss of visual sensations, including the perception of light and dark, loss of menace reflex, but the pupillary light reflex is preserved. The prognosis in cortical blindness is poor when caused by bioccipital stroke or anoxia.[1] Associated features, many described around the turn of the 19th century gave rise to eponymic syndromes, sometimes difficult to keep straight. Although these conditions are relatively infrequent, they should be in the knowledge base of every neurologist, particularly in the fields of behavior and vision. (Table 1).

Anton syndrome, also called *visual anosognosia*, is a denial of vision loss in the presence of relatively well-preserved cognition *associated with confabulation*, or making up experiences to compensate for cortical blindness. In 1899, the Austrian psychiatrist and neurologist Gabriel Anton described cases of patients with evident blindness who denied their deficits and similar cases of deafness with denial.[3] The first known description came from Seneca, in 63 CE, about Harpaste, in his household, who foolishly denied and argued about her blindness. Sometimes the name of Babinski is added because of his description of anosognosia.[4] Those affected behave and talk as if they had normal vision and *confabulate* to fill in the missing sensory input. When they collide with pieces of furniture or a closed door, they may explain that it is because of the lack of proper lighting. Anton suggested that intact areas of the brain are disconnected from the damaged visual pathways. Without visual inputs, the functioning speech areas may confabulate a response.[3] Others postulated that the secondary visual system, located in the superior colliculus, pulvinar, and temporoparietal regions, will continue to function in the absence of the primary one resulting in confabulation.[5] Lesions are described in the visual cortices, bilateral lateral geniculate bodies, posterior limbs of the internal capsules, optic radiations, and the corpus callosum.[6] Anton syndrome is considered rare, although unawareness of hemianopia is not uncommon.[7] The combination is seen in about 25% of cortical blindness in one large series.[1] Anton syndrome is described with additional deficits such as autotopagnosia or difficulty localizing one's body parts in space.[8] The prognosis is variable depending on the underlying condition.

Balint syndrome. In 1909, Rezso Balint, a Hungarian internist-neurologist, published a triad of "psychic paralysis of gaze, optic ataxia and spatial disturbance of attention."[9] The "psychic paralysis of gaze" manifests as the difficulty in shifting the eyes and scanning

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Table 1: Table of syndromes

Eponyms and synonyms	Main features	Etiology and structures affected
Anton syndrome	Denial of blindness	Disconnection from preserved subcortical connections in bilateral stroke
Balint syndrome	Simultanagnosia, integrative agnosia Oculomotor apraxia Optic ataxia	Biparietal syndrome of attention and oculomotor mechanisms Degeneration and rarely stroke
Charles Bonet	Visual hallucinations with blindness	Preserved subcortical structures to V5 and V6 cortex; stroke
Capgras delusion	Delusions of double (of a relative)	Degenerative disease of posterior cortex and underactivation of normal autonomic arousal
Cortical blindness	Loss of vision, but presence of pupillary reflex	The destruction of bilateral visual cortex or calcarine pathway, stroke, or anoxia
Fregoli syndrome	Delusion of an impostor of a famous person	Partial impairment of facial recognition, inappropriate arousal for viewing unfamiliar faces
Prosopagnosia	Inability to recognize faces	Right hemisphere temporooccipital mechanism, stroke, or congenital
Riddoch syndrome	Preservation of motion in blind field	Preserved parietal and V5 connections to subcortical structures
Subjective doubles	Seeing a "doppelganger" or mirror misidentification	Partial visual cortex R > L and facial recognition impairment, degenerative disease
Visual agnosia	Failure to identify objects with otherwise good vision and nonvisual semantic cognition	Visual-temporal impairment R*R*> L Stroke, carbon monoxide, and anoxia degenerative disease

(also called oculomotor apraxia). The inability to move the hand to a specific object by using vision is called optic ataxia. The third component, the inability to pay attention to multiple objects in visual space, was later labeled *simultanagnosia* by Wolpert.[10] The visual world is perceived erratically, as a series of single objects. Balint described bilateral parietal infarcts, but a complete Balint syndrome due to stroke is rare.[11] It is occasionally caused by a bilateral borderzone infarction in the occipito-parietal region due to sudden and severe hypotension. In posterior cortical atrophy,[12] the clinical presentation may encompass Balint syndrome as its key feature, and it may turn out to be the commonest presentation.[13] The restriction of the attentional window in simultanagnosia or inability to shift attention is tested by the description of a picture or the Navon figure (a large letter consisting of some other small letters). *Oculomotor apraxia*, what Balint called "psychic paralysis of gaze," consists of difficulty moving the eyes in the desired direction. Patients turn their head instead of using rapid eye movements or saccades to locate, perceive, and identify objects. It is related closely, even causally, to simultanagnosia. *Optic ataxia* is misreaching (but not towards oneself). Recent studies of the inability to adjust hand position for visually guided reaching and grasping further refined a distinction from the actual recognition of objects.[14]

Charles Bonnet was a Swiss naturalist and philosopher. In 1760, he described the vivid hallucinations experienced by his 87-year-old grandfather. He recognized that his grandfather had clear thinking, and that the hallucinations were associated with vision loss.[15] The syndrome is typically characterized by formed, nonthreatening, silent hallucinations of stereotyped images of objects and "lilliputian" small people.[16] The visual loss can be related to optic neuritis or macular degeneration, or damage to subcortical structures such as in peduncular hallucinosis, or cortical blindess.[16] In contrast to Anton syndrome, there is preservation of insight into the deficit. The phenomena tend to be underreported by patients, likely due to concern that they will be diagnosed with mental illness. Other causes of visual hallucinations include migraine with aura, occipital lobe seizures, neurodegenerative disease (Lewy body), hallucinogen use, alcoholic hallucinosis, delirium, and psychiatric disease. The persistence of visual dreams in cortical blindness is a similar phenomenon, although difficult to verify. It may signify potential recovery. Somewhere in the visual system, vision phenomena – the seeing of colors, brightness, depths, shades, and motion – is generated not only from the distribution of light on the retina, but also when the eyes are closed, in dreams, hallucinations, and imagery.[17] The exact mechanism is unknown, but the interaction of extrastriate and striate cortex is postulated and studied with functional imaging.

Cerebral achromatopsia, the inability to discriminate between different hues or colors, was described in the last century by Verrey,[18] but it was doubted until modern studies demonstrated it with lesions of the ventromedial sector of the occipital lobe.[19] A unique color processing center was confirmed in the left hemisphere with a PET scan.[20] Congenital color blindness, on the other hand, is commonly caused by retinal cone cell deficiency, and it is selective for certain colors, red-green blindness being the most common.

Peduncular hallucinosis, described by Lhermitte,[21] is a syndrome whereby lesions in the cerebral peduncle give rise to visual hallucinations by disconnecting the brainstem from the central processing of what is being looked at. Lhermitte interpreted it as a "dream state intruding upon wakefulness."[21] A possible mechanism is interference by the lesions with the alerting system of the brainstem, identified as the brainstem reticular formation.[22] Lhermitte was also interested in other dream states with hallucinations, narcolepsy-cataplexy, hypnagogic, and parkinsonian visual phenomenon. In addition to dream states, psychiatric hallucinations, imagery during wakefulness, febrile or toxic delirium, and the blindsight phenomenon are also involved in the subcortical circuitry connections with the visual cortex.[17]

Prosopagnosia or face blindness has been separately defined relatively recently.[23] It is thought to be the result of impairment in

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the inferior occipital region, fusiform gyrus, and temporal cortex, at times only the right fusiform gyrus.[24] Prosopagnosia can result from stroke, traumatic brain injury, or certain neurodegenerative diseases. Up to 2.5 percent of people are born with some degree of congenital face blindness, often inherited in an autosomal dominant pattern. Oliver Sacks wrote about his own disabling inability to recognize faces.[25] Brad Pitt, Alan Alda, and Steven Wozniak are some of the famous individuals known to have it. Some degree of prosopagnosia is often present in children with autism and Asperger's syndrome and considered a contribution to their impaired social development. Often spatial or *topographical agnosia* is associated, resulting in getting lost,[25] but at times it is dissociated and results in the loss of topographical memory alone. Interestingly, expertise for cars and birds recruits brain areas involved in face recognition.[26]

Riddoch syndrome is perception of movements alone in cortical blindness.[27] Riddoch's war-injured soldiers saw moving objects in their blind field, but "they didn't appear to have any colour or shape; they look like shadows." Movement of objects may be perceived, but forms are not recognized. The cerebral activity occurs outside V1 in the peristriate cortex and functional magnetic resonance imaging (fMRI) activation in V5 was observed.[28] This is not the same, but similar to the blindsight phenomenon of visual functions that can be elicited in response to stimuli presented within fields of cortical blindness.[29] Those affected claim not to see the stimuli, while the "sight" refers to their residual or recovered ability to localize, detect, and discriminate between such unseen stimuli, with various degrees of awareness. These processes are of interest in the area of vision rehabilitation.

Motion blindness is the opposite of Riddoch syndrome, a rare condition named akinetopsia.[30] Patients will be able to see objects but will not be able to appreciate their motion. Objects would be seen in one place or another but not moving in between. This may be explained by the presence of projections from the lateral geniculate nucleus to both the primary visual cortex via the optic radiations and the motion-selective middle temporal area which is damaged.[30]

Visual agnosia (Freud's term), or "mindblindness" as originally described by Lissauer,[31] is an impairment in recognizing objects, despite otherwise normal primary vision. Patients only recognize objects using other sensory modalities. Apperceptive visual agnosia, with lesions to the parietal and occipital cortex, is an inability to recognize objects, draw, or copy a figure.[32] Those with associative visual agnosia with damage to the bilateral inferior occipitotemporal cortex can draw or copy but cannot identify the object, even though they can describe it and even draw it from memory.[33] As Tauber aptly defined it, "a percept deprived of its meaning." The dichotomy is often unclear, and many case descriptions have features of both and other features associated with the Balint syndrome such as spiraling field defect due to spatial inattention,[34] unawareness, or the Anton syndrome of denying the visual loss. There are partial varieties where colour, direction, motion, and depth continue to be perceived normally.[33] Visual agnosia is classically described in stroke or carbon monoxide poisoning but increasingly recognized to be frequent in posterior cortical atrophy[13] and frontotemporal lobar degeneration with predominantly right-sided temporal atrophy. Although performance dissociations of poorer understanding of names/words in left-predominant patients and of faces/pictures/objects in right-predominant cases of semantic dementia are observed,[35] the semantic deficit usually becomes multimodal. *Alexia without agraphia*[36] is visual agnosia for written words produced by a dominant occipital lesion combined with damage to the splenium of the corpus callosum, causing disconnection of the pathway from the intact right visual cortex to the language area.

Delusional Misidentification (Predominantly Visual) Syndromes

Capgras illusion is the belief that someone emotionally close, often a spouse, has been replaced by a visually similar impostor.[37] Although the Capgras illusion is mostly visual, other sensory input is often misinterpreted by the patient. This phenomenon has initially been considered a psychiatric disturbance observed in schizophrenia. It is often accompanied by reduplication of place, or the phantom boarder phenomenon, which adds an element of hallucination to the syndrome. At times it is a triplication, as in one of our patients who believed that there were three people replacing is wife. One was cooking for him, another going to football games with him, and the third spending his money. He also had a phantom boarder going in and out of his bathroom. This person turned out have Lewy body disease (LBD) where the visuo-cognitive symptoms preceded the movement disorder. In a large series from our unit, 15.8% of AD and 16.6% of LBD patients had some form of misidentification syndrome, predominantly the Capgras illusion and less frequently spatial reduplication.[38]

Fregoli delusion, the belief that some stranger or casual acquaintance is a familiar person in disguise,[39] is named after the Italian actor and mimic Leopoldo Fregoli because of his extraordinary ability to impersonate people on stage. Fregoli syndrome differs from those occasional misidentification errors that we all make when we mistakenly think that we see someone we know in that Fregoli patients believe that they see someone they know is in disguise. This also differs from a generic persecutory delusion, such as the neighbor being a spy or a secret agent.[40]

Intermetamorphosis is a delusional misidentification syndrome (DMS), where those affected believe that others change into someone else physically and psychologically, both in appearance and personality.[41] Although some of these people have face recognition problems and have organic disease, the paranoid flavor of the delusion often suggests psychiatric illness. *Reverse intermetamorphosis* is a belief that a stranger is transforming into yourself.[42] Persistent mirror delusion when a person reflected in a mirror believes it to be a stranger[43] occurs in the context of neurological illness, usually with right hemisphere impairment, and in dementia. *Subjective doubles* is a DMS in which a person believes they have a double (Doppelgänger) with the same appearance, but usually with different character traits, who is leading a life of their own.[42]

Reduplication Paramnesia In 1903, Arnold Pick described a case of a 67-year-old woman with a diagnosis of senile dementia, who developed a conviction that there was a duplication of the same clinic in Prague, an "old" one and a "new" one, both headed by Pick.[44] This is a visual misidentification of space or location rather than person and is probably the most common after the Capgras delusion.[38] Those with reduplicative paramnesia more frequently suffered from head trauma or cerebral infarction and showed more features of right hemisphere lesions on neuropsychological testing or computerized tomography scan than the patients with other misidentification syndromes.[41]

Updating Etiology, Investigations, and Mechanisms

These conditions are not only curiosities but provide an important source of knowledge of visual perception and processing. Visual

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agnosia and prosopagnosia, relatively common and the less frequent Balint syndrome and blindsight phenomena created a great deal of interest in cognitive neuroscience with new methods of functional imaging and experiments. The visual cognitive system has been described as the dorsal or "where system," connecting the occipital with the parietal areas for movement and attention and the "what system," connecting the occipital with the temporal areas providing meaning for object and face reognition.[45] Visual object agnosia and face agnosia are related to impairment of the "what" system, while reaching and movement related visual cognition to the "where" pathways. Hand movements to grasp an object under visual guidance are an example of the latter.[14]

Visuospatial attention and orientation, impaired in Balint syndrome, depend on a network of structures that includes occipital areas 17, 18, and 19 connections with temporal and parietal lobe regions, frontal eye fields, and prefrontal cortices.[11] The ability to perceive objects in simultaneous visual presentations and in space depends on "where" pathways and their parietal connections. The various perceptual phenomena reported in Balint syndrome include "vanishing" objects, tilted vision, metamorphopsia (distortion, a straight line becomes wavy), and palinopsia (persistence of an image) are explained as a variety of combined deficits from lesions of the dorsolateral visual association cortices. Executive switching of attention between visual objects and tasks depends on areas in the prefrontal cortex that influence the dorsal and ventral visual pathways.[11] The ventral "what" pathway, which is crucial for object representation and memory, is relatively preserved in Balint syndrome.

Clinical investigation of the higher-order visual syndromes in addition to neuroimaging should include tests of object and facial recognition as well as the more elementary visual functions of pupillary and menace reactions, extraocular movements, visual pursuit, and detailed cognitive assessment. An Object, Face Colour Agnosia screen (OFCAS), focusing on the ventral "what stream," and Complex Picture descriptions testing of global versus local processing for the "where stream" are examples of comprehensive, yet practical tests.[15] Cognitive neuroscientists examine single patients extensively with an exhausting array of experiments.

A clinical rule of thumb is that cortical blindness, acquired prosopagnosia, visual agnosia, Anton, and Charles Bonnet syndromes are likely caused by focal lesions, most likely vascular, in the basilar artery territory. Neoplasms, lobar hemorrhages due to amyloid angiopathy, multiple sclerosis, preeclampsia, autoimmune disorders, mitochondrial disease, and posterior reversible encephalopathy syndrome (PRES) have also been described in these conditions. The misidentification syndromes of Capgras and Fregoli are more likely to be associated with degenerative conditions, such as Lewy body, Alzheimer's disease, and frontotemporal degeneration. Visual agnosia and Balint syndrome can be related to either focal or degenerative brain disease.

Facial recognition is based on knowledge and familiarity. These have different neural bases and can be dissociated. In cases of dementia, faces appear familiar but cannot be identified. In Capgras syndrome faces, though recognized, no longer generate a sense of emotional familiarity and may appear as impostors. Capgras and Fregoli have originally been described in schizophrenia with paranoid content being common. The Fregoli delusional content is presumably generated when hyperexcitation from the cognitive system causes the misidentification while in Capgras the problem is the underactivation of normal autonomic arousal, possibly the amygdala.[46] However, a report where the two syndromes were combined makes this idea of activation in opposite patterns unlikely.[40] Facial recognition is a complex cognitive processing skill not unique in humans, but demonstrably present in many animals, albeit confounded by smell and motion perception and other visual cues. Recent years have seen the computerized use of facial recognition algorithms for security, fighting crime, and many other purposes, even opening your iPhone.

Functional magnetic resonance revealed the activation of motion-specific areas, such as the supramarginal gyrus, middle temporal area, and subthalamic activations within the superior colliculi and the pulvinar. These results reveal the role of secondary pathways bypassing the primary visual area in residual vision.[28] Subcortical pathways to extrastriate visual cortex underlie residual vision comprising blindsight following bilateral damage to V1.[47] The pathology that destroys the visual cortex causes massive degeneration of the lateral geniculate nucleus and leads to transneuronal degeneration of many retinal ganglion cells. The survivors continue to project via further subcortical way stations, such as the superior colliculus of the midbrain, to visual cortical areas beyond V1. fMRI activations in extrastriate visual cortex were found in patients who retained or recovered vision after V1 destruction.[17] This has implications for potential therapeutic intervention.

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