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Attention and Perception Related aspects in Balint syndrome-Neuropsychological and Neurophysiological prospects

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ABSTRACT

Balint syndrome is an uncommon and incompletely understood triad of neuropsychological impairments. Balint syndrome is characterized by dysmetria secondary to visual perceptive defect and inability to recognize more than one object at a time. Balint's syndrome most often occurs with an acute onset as a consequence of two or more strokes at more or less the same place in each hemisphere, therefore it occurs rarely. Disorders such as tumours, trauma, near drowning, Eclampsia, drug toxicity, HIV encephalitis, Alzheimer's can also leads to balint syndrome. It is a strange combination of optic ataxia, Gaze apraxia, Simultanagnosia. Although it has been generally constructed as a biparietal syndrome causing an inability to see more than one object at a time, other lesions and mechanisms are also possible. Key syndrome components are dissociable and comprise a range of disturbances that overlap the hemineglect syndrome. Balint syndrome usually from large and more or less symmetrical lesions involving the posterior parietal region, including extensively the superior parietal lobe, as well as part of the inferior parietal lobe and the superior part of the occipital lobe. Diagnosis of individual components and of the whole syndrome may remain difficult, particularly when elementary motor, sensory, and visual deficits coexist. Lack of awareness of this syndrome may lead to misdiagnosis and resulting inappropriate or inadequate treatment, therefore clinicians and other healthcare professionals should be familiar with the balint syndrome. The goal of this review is to explore a range of anatomical and psychological explanations for this disorder.

Keywords: Balint syndrome, Stroke, Optic ataxia, Gaze apraxia, Simultanagnosia

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INTRODUCTION

Balint syndrome is a clinical entity which combines variously a set of complex spatial behavior disorders following bilateral damage to parieto-occipital regions. The core syndrome refers to triad of manifestations –optic ataxia, gaze apraxia, and simultanagosia to which visuospatial perception deficits can be associated. Balint syndrome can be identified at the bedside examination and it allows roboust anticipation of lesion localization. Historically, balint provided in 1909 a thorough clinical and autopsy description of a patient who exhibited hitherto undescribed symptoms, he named seelenhmung des "schauens", optischeataxie, raumlichestorung der aufmerksamkeit, usually translated as "psychic paralysis of gaze, optic ataxia spatial disorder of attention" [1, 2] He related these symptoms to a bilateral area of ischemia in the, and posterior parietal and anterior occipital regions, including the angular gyrus and underlying white matter on both sides. In balint's view, psychic paralysis of gaze was a consequence of attention impairment and not of eye movement disorder meanwhile, optic ataxia was explained in terms of disconnection between visual and mortor centers. Independently Holmes [3] reported, under the heading of " visual disorientation", a series of six patients with penetrating missile wounds of the brain, stressing oculomotor disturbances and spatial misperception , without individualizing optic ataxia. It is not clear whether the descriptions balint and Holmes are truly different or merely reflect different focus on a range of symptoms [4.] Hecaen first coined the term "Balint syndrome" [5]. Among minor forms of balint syndrome ,those resulting from unilateral lesion mainly result in impaired visually guided manual reaching, a symptom ascribed to a general visual disorientation , in Holmes' view [6,7,] or as optic ataxia- a specific disorder following balint's view [8,9]. Balint syndrome is more than a rare neurological curiosity. It has gained a new interest from progress in visual neurosciences as it provides insight into the functional roles assigned to the neuronal populations of the occipital-parietal region. These functions include spatial perception, gating, and directing spatial attention, as well as spatial coding of eye and hand movements in the immediate extrapersonal space.

General Presentation of balint syndrome:

Complete Balint Syndrome:

Patients with full-blown syndrome are severely impaired. Their behavior is at first suggestive of blindness. Hence, their movements are hesitant and awkward; their gaze is kept mostly immobile, sometimes in the wrong direction with respect to the ongoing action, and patients are unable, for example, to pick up food from a tray or to walk on their own. However, examination

shows definitely that they can see. They demonstrate surprising ability to recognize objects, even of tiny size, although they may require time to spot them, particularly when several items are present. Further examination indicates their visual deficit is selective for visuospatial material. They are no longer able to reach for objects, to find their way in familiar surroundings, or to estimate size, orientation, or distance of objects. Patients are aware of their deficits, but usually they have much difficulty in conceptualizing them. Analysis of each component of syndrome may be rendered difficult by fluctuation of performance and possible interdependency between some of them, such as reciprocal interactions between optic ataxia, simultanagnosia, and visuospatial disorders, or between gaze apraxia and simultanagnosia [10,11] .

Optic Ataxia

It refers to inaccuracy in reaching and grasping visually presented objects, and more generally in exploring manually peripersonal visual space, that cannot be explained in terms of elementary visual, proprioceptive , or motor deficit. According to balint's view, optic ataxia is secondary to an inability to use visual information to guide movements, in the same way as tabetic ataxia results from loss of proprioceptive information. Alternative names have been proposed, such as visuomotor ataxia [12] or visuomotorapraxia [13], however the original term has persisted. Optic ataxia indicates a dissociation in brain function such that visuospatial attributes of a particular object are still processed in terms of perceptual conscious experience and verbal description but are no longer implemented to calibrate movement of the limb. Several spatiotemporal abnormalities of visual guidance of hand movement can be described from clinical observation and kinematic recordings [14, 15.] The whole movement is both delayed and slowed. The trajectory of hand movement is grossly impaired in terms of directions and amplitude, while the hand is held largely open, the posture grip being inadequate with respect to the form and orientation of the target object. Movement usually fails to reach the goal, although corrections may be possible with groping movements until a contact eventually elicits grasping.

It is evidenced in the peripheral field of vision-that is, when the eyes are not directed to the target to be reached. Spatial errors of reaching increase with the eccentricity of the target. In some cases, it may also be observed to a lesser degree in central vision. Typically, it is present for the two hands in the whole hands in the whole field of vision, but it can also be limited only to one homonymous visual field, or to one hand.

Limitation of optic ataxia to the peripheral field implies that the symptom has to be looked for in these patients, as deficit is not apparent when perhension is performed in the natural condition of foveal vision. Limitation of optic ataxia to one hand demonstrates that the symptom is

independent from any visuospatial defict and results from visuomotor incoordination, provided that proprioception is intact. Patients often exhibit striking decoupling of eye-hand coordination, in such a way that eyes are not aligned to the place the hand is reaching to. Some harbor abnormal eye-hand recoupling, resulting in attraction of hand movement toward the place the patient's eyes are foveating, a phenomenon called "magnetic misreaching" [16, 17.] Deficiency of visuomotor coordination observed in optic ataxia interferes with several daily activites, such as drawing, writing, eating, and filling a glass. For example, patients may be unable to draw aline between two points or to pick up a piece of food with a fork. Difficulty in navigating around and avoiding obstacles are related to the same dysfunction [18]. Beyond limb movements, whole-body movements may also be impaired, resulting in difficulty for these patients in properly orienting their body with respect to the chair they intend to sit in, or to the bed they want to get in [19.]. It may not be the sole cause of such an impressive disorder, and both axial apraxia and visuospatial misperception may also be contributive factors.

In contrast with movements directed to visual targets, those directed to body parts are accurate patients can point precisely with a finger to the body parts that are touched by the examiner, indicating that some aesthetic motor coordination is spared. Finally, although optic ataxia is described as a specific visuomotor transformation deficit, it may also be demonstrated for non-visually guided movements. For example, reaching without vision of hand may be even more impaired than reaching under visual feedback [20] and some patients may show in addition auditory ataxia [(21]. Performance may vary according to the hand or the sensory or cross-sensory modality, but also with the action space related to the body [22, 23]. These observations indicate that it may be understood as a dysfunction of supramodal sensorimotor transformation control of movements in the immediate peripersonal space.

Figure 1: The Clinical examination of Optic ataxia patients

The clinician stands behind the patient and asks them to fixate straight ahead. The clinician then successively presents target objects in the patient's peripheral field of vision to be grasped with

one hand or the other. This patient with right optic ataxia exhibits a gross deficit when reaching to right-sided objects with his left hand. Once he has missed the object, he exhibits exploratory movements comparable to blind subjects. This poor visuomotor performance can be contrasted with the ability of the patient to describe the object and his normal ability to reach towards central targets [24].

Gaze Apraxia

Eye movement disorders have been variously labeled "psychic paralysis of gaze," oculomotor disorders [25] or "ocular motor apraxia" [26]. These nebulous terms reflect the difficulty in assigning ocular motor deficits to a well-defined dysfunction, as well as the spectrum of eye-movement problems observed from patient to patient. As no single term is satisfactory, the term gaze apraxia will be used here to indicate that the defict of saccade function may be dissociated according to cognitive context (apraxia). Gaze apraxia should not be confounded with "oculomotor apraxia", a developmental syndrome of horizontal saccades deficit in children. Gaze apraxia is characterized by severe abnormalities of generation of eye movements in response to visual targets in space, in the absence of ocular motor palsy, as curtained by full reflexive eye movements. The more constant observation in these patients is poverty of eye movements, which may culminate in a condition often referred to as "spasm of fixation" or "visual grasp reflex". Patients stare open eyed, with the gaze being locked to the place they are fixating and they may be able to disrupt such sticky fixation only after a blink. In more severe cases patients are not able to initiate intentional saccades in response to a visual stimulus or a verbal command. Only occurrence of some automatic saccades to novel stimuli, such as unexpected sound or sudden touch to their own body and sparing of REM sleep, may distinguish this condition from saccadic paralysis [27, 28.] Usually patients with balint syndrome keep moving their eyes "spontaneously" or on verbal command, while they are impaired in performing visually guided saccades. The more attention and complex visual processing the eye movement requires the less it is likely to be performed. In this regard visual such behavior of a particular target among distracters is particularly vulnerable. Hence, when the patient is asked to move their eyes to a target suddenly appearing in the peripheral field, they may generate no movement or they may initiate wandering eye movements that results in erratic displacement of eyes in space, ending with incidental acquisition of the target. Even at this point the target may be lost through the liability of perception and instability of fixation, and a new search pattern has to be activated. Scaccadic behavior is abnormal in a rich (natural) environment array that requires continuous selection between concurrent stimuli [29], but it may be normal in a simplified context,

for example when the task is to direct eyes to a peripheral light emitting diode in the dark [30]. In addition to saccades m other visually guided eye movements are impaired, such as smooth fovealpursit and optokinetic responses. Convergence on to a visual target is altered and blink to visual threat is usually abolished. In addition to ocular movement abnormalities some patients loss spontaneous blinking [31]. Eye movement recordings usually show several abnormalities, such as prolonged latency, fragmentation and hypometria of saccades, fixation drift, and absence of smooth pursuit [32, 33]. The pattern of oculomotor scanning is highly abnormal during scene exploration [34]. Both of accuracy of fixation and saccadic localization are impaired, and spatio temporal organization of eye displacement does not fit with the spatial configuration of the scene to be analyzed [35] based on the facts that eye movement deficit is enhanced whenever attentional demand is high that a gradient of declining deficit exists between visually guided , pure intentional , and reflexive movements, it could be suggested that gaze apraxia is the product of both visual- and attention- processing deficits in eye movements.

Simultanagnosia

"Spatial disorder of attention" [36], "restriction/ shrinkage of attention field" [37], "bilateral neglect syndrome," or disorder of simultaneous perception" [38] are equivalent terms to designate a complex symptom that can be viewed as a limitation of visuospatial attention resources. Patients do not seem to perceive visual targets located away from a small area, which is usually the area of current foveation. They exhibit a reduction of "useful field of vision", operationally defined as the field of space that can be attended to while keeping central fixation [39]. This can be tested by asking patients either to direct their e eyes or their hand to or to name, an object presented extrafoveally. Evaluation using a motor response may be affected by concurrent optic ataxia and gaze apraxia, while verbal response more directly probes conscious or attentive perception. Generally responses are more likely to be given after verbal encouragement, a finding that indicates that the deficit is not a consequence of a reduction of the visual fields but of attention scanning for non-central events. Shrinkage of the attention field reduces detection of multiple objects and may render patients able to perceive just one item at a time. For example, a patient with balint syndrome was not able to perceive the light of a match while focusing on a cigarette until he felt a burning pain. This limited capacity of attentive vision for only one object at a time does not depend upon the size of the object. This is another distinction from a visual deficit. In addition single object attention is reduced, as perception may even have difficulty in binding several attributes of one object such as color and form [40]. As a general consequence, patients fail to perceive at any time the totality of the items forming a visual scene [41].

Description or copying of complex figures is laborious and slow, patients focusing serially on details, understanding portions of the pictures with a piece meal approach but failing to switch attention from local details to global structures [42]. Similarly performance for counting objects is altered. Patients explore working space in a disorganized fashion and may focus several times on the same object, their "revisiting" behavior betraying impairment of short-term memory for spatial location of items already explored. Alexia is often prominent, with difficulties in assembling words in a text, while letters and isolated words are usually well identified. In all these tasks the severe perceptual deficit linked to attention capacity limitation increases when visual environment is complex and when stimuli are presented for a brief period. Although blinking in these patients is often reduced, some patients learn unconsciously to close their eyes either to enhance perception of a vanishing object being fixated or to break fixation from one object so they can look at another one[43]. Finally, this attention- related deficit may be limited to a part of the spatial field resulting in hemispatial neglect [44] or in altitudinal neglect [45]. Spatial attention of disorders may have impact on the other components of balint syndrome, and for some authors constitute the central core of the syndrome [46]. They also play a role in hand misreaching by increasing visual misperception of the target which may "vanish" whenever the hand is approaching the goal [47].

Associated deficits of balint syndrome:

Visuospatial perception disorders can variously be found, depending on the patient and on both the way the examination is performed and the way the results are analyzed. In most severe cases, patients have lost cardinal orientation in space resulting in an inability to distinguish the right from left side (or up from down) in egocentric space or an inability to discriminate whether a given object is oriented to the right or to the left (orientation agnosia). Other aspects include difficulty in judging absolute distance in estimating positions of objects relative to the subject, indepth perception and stereopsis, in visual motion perception in perception of egomotion and in directional heading [48, 49, 50]. Additional symptoms depend on the extension of the lesion in the parietal lobes and in the adjacent occipital and temporal regions. They include anomic or transcortical sensory aphasia, pure agraphia, alexia without agraphia, alexia with agraphia, Grestmann syndrome, hemispatial neglect, constructional aparxia, dressing apraxia, or appreceptive agnosia [51, 52]. Visual fields are typically full [53, 54].

Minor forms of balint syndrome:

Those with minor forms of balint syndrome have either the whole spectrum of symptoms but in attenuated form [55, 56], or only a limited range of symptoms. In the latter form has both theoretical

and practical importance as it indicates that each symptom can be isolated or predominant. A bilateral lesion may result in almost isolated simultanagnosia, in isolated optic ataxia [57, 58], or in associated optic ataxia and simultanagnosia, without gaze apraxia [59, 60]. By comparison a unilateral lesion of the posterior parietal region results in near-isolated optic ataxia [61, 62, 63, 64, 65, 66]. Optic ataxia from a unilateral lesion resembles that after a bilateral lesion [67]. However visuomotor deficit is observed only in the peripheral visual field as it is corrected with foveation [68]. In addition the symptom is lateralized mainly affecting both the hand and the visual field contralateral to the lesion. Such a lateralization of optic ataxia excludes an explanation in terms of motor, sensory, or visual deficit. In the involved hemifield, it can be demonstrated that optic ataxia can occur independently from perceptual disorder of spatial position or orientation discrimination [69, 70, 71]. Finally additional psychophysical studies in patients with optic ataxia have provided new insight into some visuomotor operations performed in the parietal lobe. First, it was shown that patients who have recovered from optic ataxia in central vision are not able to adjust movement if the target location is unexpectedly changed upon movement initiation [72]. Moreover, other studies have shown that hand reaching accuracy much improves whenever a few seconds delay is introduced between target presentation and movement onset [73, 74]. These observations strongly indicate that optic ataxia more likely results from a deficit of real time automatic trajectory adjustment depending from a fast and visuomotor processing operated in the dorsal-most part of the posterior parietal lobe than from a deficit of initial motor planning [75] or from a global impairment of goal directed actions. They show that alternative visuomotor routes, using more cognitive and intentional context, enabling a partial compensation for the deficit [76, 77)]. Second, evidence has been provided to indicate that optic ataxia does not result from processing of erroneous visual spatial information about the target but from a faulty reconstruction of the target location in a stable spatial representation [78].

Location of lesions and functional correlations:

Balint syndrome usually from large and more or less symmetrical lesions involving the posterior parietal region, including extensively the superior parietal lobe, as well as part of the inferior parietal lobe and the superior part of the occipital lobe. Depending on the causes, cortex of BA 7, BA 39 and BA 19, as well as underlying white matter are involved. Extension of lesions is however quite variable. This is particularly true when balint syndrome results from infraction in the watershed areas. In such cases lesions can extend anteriorly to the frontal lobes or posteriorly to the occipital lobes. Functionally a lesion in the posterior parietal cortex also disconnects its cortical and subcortical targets namely several areas in the premotor/prefrontal cortex and deep

layers of the superior colliculus. Diversity in the clinical expression of the each component of the syndrome reflects variability of lesion extension and functional disconnection. There is no minimal lesion sufficient for producing the whole syndrome and it is likely that anatomical correlation for each component is relatively independent. Simultanagnosia may follow bilateral lesion of the superior aspect of the occipital lobes and the superior parietal lobe [79]. This region is part of a dorsal cortical network of visuospatial attention [80] that also includes the dorsolateral prefrontal cortex. This network may contribute to attentive vision for recognition of multiple objects distributed in space by selecting objects of interest enhancing pertinent sensory information maintaining a representation of information for a brief period of time in working memory system for spatial location, disengaging attention, and shifting from one object to another one in a different location [81]. The core lesion for optic ataxia after a unicus and the nearby parietal occipital junction [82, 83]. A functional MRI study showed that visually guided pointing movements activate a frontal parietal cortical network, including two specific areas for visuomotor control forming the "parietal reach region" namely the medial part of the intraparietal sulcus and the parietal occipital junction depending as to whether pointing is performed in central or in peripheral vision [84, 85]. Deficit in visually guided saccades may be the consequence of a lesion in the "parietal eye field" a region in the superior parietal lobe and intraparietal sulcus that may include several areas identified by functional MRI studies [86, 87]. However extensions of the lesions to the frontal lobe encroaching the "frontal eye field" may explain additional loss of intentional, self-generated saccades is closely related to the network obtaining spatial attention processing. The posterior parietal cortex is fed with visual inputs from the dorsal stream and processes spatial information, using different routes for spatial perception and action organization [88]. Some populations of neurons respond to concomitant hand reaching, manipulation, saccade, or combined eye-hand movement [89].

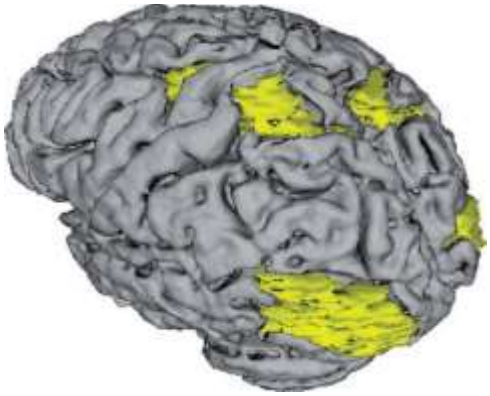

Figure 2: A typical bilateral lesion responsible for Balint's syndrome.

Causes of balint syndrome:

There are numerous diseases leading to balint syndrome. Traumatic brain lesions have been historically described after war injuries [3, 8], but currently also result from traffic accidents in patients who have suffered severe ischemic/anoxic or hemorrhagic insult in the posterior part of the hemispheres. Vascular origin is frequent and has yielded most clinical-anatomical correlation studies, because of the relatively discrete topography of lesions and the number of patients examined at autopsy [2, 23, 28].

Main causes of Balint Syndrome [78, 79]:

- Infarction
- Eclampsia
- Hemorrhage
- Amyloid angiopathy
- Central venous thrombosis
- Cerebral vasculitis
- Hypertensive encephalopathy
- Posterior reversible encephalopathy syndrome
- Tumors (Butterfly parietal malignant glioma, bilateral metastases)
- Degenerative cortical lesions (Alzheimer disease, posterior cortical atrophy, corticobasal degeneration , diffuse lewy body disease)
- Infection (Creutzfeldt - Jacob disease , progressive multifocal leukoencephalopathy, HIV encephalitis)
- Inflammatory disease (Balo concentric sclerosis)
- Adrenoleukodystrophy
- Drug toxicity
- Traumatic brain injury
- Cerebral anorexia
- Carbon monoxide intoxication

Differential diagnosis:

Thorough examination of the patient should eliminate the erroneous diagnosis of blindness, visual object agnosia, or psychogenic disorder. However, diagnosis of individual components and of the whole syndrome may remain difficult, particularly when elementary motor, sensory, and visual deficits coexist. This is the case when balint syndrome is found after recovery from

cortical blindness, at a stage when patients keep degraded low-level vision with visual field defect and appreceptive agnosia caused by occipital lesions. Association of hemispatial neglect with contralateral hemianopia can mimic simultagnosia [89].

Assessment of balint syndrome:

Diagnosis of balint syndrome is often difficult because of the complexity of symptoms, which can hardly be analyzed at the beside. Actually the syndrome is under diagnosed and often missed. Diagnosis of isolated optic ataxia may also be missed, but for a different reason, as the symptom is usually convert and only identified through specific search. Assessment needs evaluation first of elementary motor and sensory functions, such as strength and coordination, touch sense, and kinesthesia, as well as "low –level" vision. Bedside examination of vision would indicate only that objects or colors are recognized provided they are presented centrally and time is allowed for evaluation, and that visual fields are full in confrontation testing. Formal testing is necessary. Visual acuity is usually normal, ophthalmologist should be aware that simultanagnosia and gaze apraxia may compromise reading of the letter strings. Visual fields are typically full but they may show homonymous deficits such as lower quadrantanopia or paracentral scotoma or they may exhibit constriction of peripheral isopters on goldmann perimetry as a consequence of fluctuating spatial attention [60].

Prognosis and Management of balint syndrome Patients:

In patients with unilateral lesions who are optic ataxia, functional consequence is modest as visuospatial functions are mostly spared and reaching is normal when performed in natural, central fixation [51]. In full balint syndrome, functional prognosis is poor because the diffusion of lesions usually precludes significant recovery, even in these patients recovery is partial leaving impaired simultaneous perception and reading difficulties [80, 84]. Rehabilitation strategies are largely empirical and individually tailored based on thorough clinical analysis of impaired and spared functions particularly in terms of patients "lower" and "higher" visual spatial abilities [85,86]. An adaptive strategy uses preserved skills to compensate for problems and it aims at altering the patients environment to lessen his or her disabilities [31].Exercises usually include eye movement fixation, saccades and persuit of visual targets with the intial help of additional auditory or proprioceptive cues, manual reaching and pointing as well as perception of a growing number of simultaneously presented objects [85, 86, 87, 88].

CONCLUSION

Balint syndrome is not common and is difficult to assess with standard clinical tools, the

literature is dominated by case reports and confounded by case selection bias, inadequate study of basic vision, poor lesion localisation and failure to differentiate between defects in acute and chronic phases of recovery. Finally the validity of balint syndrome is has been questioned by some.

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