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## Development of a Partial Balint's Syndrome in a Congenitally Deaf Patient Presenting as Pseudo-Aphasia

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### Abstract

We present a 56 year-old, right-handed, congenitally deaf, female who exhibited a partial Balint's syndrome accompanied by positive visual phenomena restricted to her lower right visual quadrant (e.g., color band, transient unformed visual hallucinations). Balint's syndrome is characterized by a triad of visuo-ocular symptoms that typically occur following bilateral parieto-occipital lobe lesions. These symptoms include the inability to perceive simultaneous events in one's visual field (simultanagnosia), an inability to fixate and follow an object with one's eyes (optic apraxia), and an impairment of target pointing under visual guidance (optic ataxia). Our patient exhibited simultanagnosia, optic ataxia, left visual-field neglect, and impairment of all complex visual-spatial tasks, yet demonstrated normal visual acuity, intact visual-fields, and an otherwise normal neurocognitive profile. The patient's visuo-ocular symptoms were noticed while she was participating in rehabilitation for a small right pontine stroke. White matter changes involving both occipital lobes had been incidentally noted on the CT scan revealing the pontine infarction. As the patient relied upon sign language and reading ability for communication, these visuo-perceptual limitations hindered her ability to interact with others and gave the appearance of aphasia. We discuss the technical challenges of assessing a patient with significant barriers to communication (e.g., the need for a non-standardized approach, a lack of normative data for such special populations), while pointing out the substantial contributions that can be made by going beyond the standard neuropsychological test batteries.

### Keywords

Balint's syndrome; Deafness; Simultanagnosia; Optic Apraxia; Optic Ataxia

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Balint's syndrome was originally described shortly after the turn of the 19<sup>th</sup> century (Adams, Victor, & Ropper, 1997; Balint, 1909). The full syndrome includes an inability to perceive

more than one object at a time (simultanagnosia), an inability to fixate and follow an object with one's eyes (optic apraxia), and severe misreaching for visual targets (optic ataxia). Balint's syndrome is believed to result from bilateral parieto-occipital lobe lesions that disrupt connections between the posterior visual association areas and the motor regions of the prefrontal cortex (dorsal stream "where" pathway) (Tranel, 1995). Given that this syndrome requires bilateral cortical damage, it tends to be rare in frequency of occurrence. Etiological causes typically include vascular insult and degenerative diseases (e.g., Alzheimer's disease, HIV encephalopathy) (Rizzo & Vecera, 2002). Other causes have included gunshot wounds and carbon monoxide poisoning. Udesen and Madsen (Udesen & Madsen, 1992) attributed the most frequent cause to an acute onset of severe hypotension that results in bilateral border zone infarctions in the parieto-occipital region. Partial cases of Balint's syndrome are more common and sometimes herald the onset of the full syndrome in the occurrence of degenerative disease (Davis, Panisset, De Agostini, & Boller, 1996).

Adams et al. (Adams et al., 1997) indicate that the primary feature of Balint's syndrome involves an inability to functionally direct oculomotor movements in the exploration of space. "Shaft vision" is a term that is sometimes used to describe the patient's fixation on one visual object. Studies have shown that Balint's patients have difficulty comparing two objects (H. Branch Coslett & Saffran, 1991). For example, they may not be able to discern which of two objects is smaller or closer, nor whether two lines differ in length. These visual limitations often impact the patient's ability to navigate their environment, and sometimes give them the appearance of being blind. Balint's patients often demonstrate impaired reading ability. They can sometimes read words easier than they can identify single letters, presumably because the word is treated as a single perceptual unit. However, some Balint's patients have been reported to have trouble grouping letters into words. Some patients will also complain that objects in their field of vision are disappearing. Of note, however, visual-field defects are not usually present and visual acuity may be normal. Patients suffering from this syndrome are sometimes mistaken to be demented or perhaps troubled by a psychiatric disorder by those lacking awareness of this condition (Juergens, Fredrickson, & Pfeiffer, 1986).

Explanation of the nature of the cognitive dysfunction underlying Balint's syndrome remains controversial. Early theories regarding a restricted visual-field or the impact of aphasia and other higher cognitive deficits have been discredited (Bauer, 1993; H. Branch Coslett & Saffran, 1991; Rizzo & Vecera, 2002). Object size does not seem to be a critical variable, as one might suspect if this disorder were to rest upon visual-field restrictions. Likewise, many persons exhibiting this disorder have not suffered from accompanying intellectual deficits. An area of promising research involves the possibility that Balint's syndrome primarily reflects an impairment of visual attention. Between-object attentional shifts are grossly impaired in Balint's patients. Furthermore, Rizzo and Hurtig (Rizzo & Hurtig, 1987) have shown that a defect of visual attentional processing impairs the ability of such patients to sustain awareness of visual targets. These findings seem consistent with research establishing the importance of the parietal lobes in maintaining and directing visual attention (Posner, Walker, Friedrich, & Rafal, 1984). Furthermore, bilateral lesions in the superior longitudinal fasciculus or the superior parietal lobes may disrupt areas that are vital for exploration and analysis of spatial relationships.

A number of good reviews have been written that describe the likely neuroanatomical lesions that underlie Balint's syndrome, and that also summarize the research attempting to more precisely determine the nature of its characteristic features (H. B. Coslett & Chatterjee, 2003; Rizzo & Vecera, 2002). While we hope that our case will contribute to this literature as well, we have decided to focus on the unique presentation that this visuo-ocular syndrome has created in a patient who is congenitally deaf (i.e., pseudoaphasia), and on the special challenges that this combination of deficits poses to assessment. Moreover, we hope that this case will highlight

the contribution that neuropsychology can make to the assessment of behavioral syndromes, even when we are forced to move beyond the comfort zone that can be afforded by a standard battery of neurocognitive tests.

## Case Presentation

Our patient is a 56 year-old, right-handed, congenitally deaf, white female who was participating in acute inpatient rehabilitation at the time of identification of her visual disturbance due to what appeared to be an unrelated cerebrovascular accident. The patient had initially presented with a moderate left hemiparesis and mild left-sided facial droop, which were attributed to a small right pontine ischemic stroke evidenced on CT scan of the brain. Medical history was remarkable for insulin dependent diabetes mellitus, peripheral vascular disease, and carotid artery disease. The patient had been successfully participating in rehabilitation therapies for a few days with her children acting as interpreters (she communicated using American Sign Language: ASL). The patient had not initially been referred for neuropsychological assessment due to communication issues as well as the belief that the patient's injury was restricted to a small brainstem infarction. However, her children became concerned that her cognitive status was worsening during her rehabilitation stay, which led to a consult for a neurocognitive evaluation. Her children reported that the patient seemed to be misidentifying ASL signs and noted that she was sometimes responding incorrectly to their queries (i.e., she would sign something back to them that was not an appropriate response to what they had signed). However, they also noted that she was often able to respond correctly with the repetition of signs. The patient was also complaining of difficulty reading. Her daughter noted that her mother was sometimes neglecting objects and persons located to her left side and indicated that she was also complaining of unusual visual phenomena. Positive visual phenomena included complaints of a persistent visual floater in the patient's lower right visual quadrant that moved from the periphery to her central field of vision and the intermittent occurrence of a multicolored band in this same region.

The first author (DLD) initially met with the patient and her family to review her history, to assess her complaints, and determine her ability to participate in formal testing given her congenital deafness. Her children served as interpreters during the clinical interview. During this initial interview, it was apparent that the patient was having difficulty processing her children's signs. Nevertheless, she was able to provide a coherent, thorough history. She used a combination of vocalizations and sign language to convey her history and complaints. The patient was widowed and was living with one of her four children at the time of her hospitalization. She indicated that she graduated from a high school for the deaf and had worked in the past in an unspecified position that appeared to involve general clerical work. It was also clear during the interview that the patient was neglecting objects and persons positioned on her left side. She bumped into objects when attempting to move about the room, did not respond to her children unless they moved from her left to the midline of her visual field, and could not locate objects that were positioned to her left side (e.g., the patient could not locate a pencil or book located on a table on her left-hand side). She described her reading problems as involving an inability to see the words on the page, while denying any problems with auditory or written comprehension. As it appeared that she was experiencing some degree of visual disturbance, an attempt was made to check her visual acuity with a pocket Snellen eye chart and to assess the integrity of her visual-fields to confrontation. Interestingly, when presented with the eye chart, the patient appeared to have difficulty seeing all of the letters on it. Each time we presented her with the card she would identify one or two letters and then indicate that she did not see anything else on the card. Her visual-fields appeared intact, as she responded to moving stimuli when presented to either visual-field in isolation. Of note, when presented with bilateral visual information (i.e., double simultaneous stimulation), she would only respond to stimulation on one side at a time. Nevertheless, there was no lateralized pattern to the extinction

exhibited on this task. In addition, no extinction to stimulation was observed in the tactile modality on this task. Finally, the patient was able to perform both simple and complex commands and instructions (e.g., inverted or otherwise complex syntax) without significant difficulty.

### Neurocognitive Assessment

Based on the initial contact with the patient, it appeared that visual dysfunction and partial neglect might be causing the majority of the problems that she and her family were describing (e.g., reading difficulty, problems responding to sign language). It was determined that further testing should be completed with the patient that would include a thorough screen of language and visual processing. Great care was taken in choosing tasks that would not be confounded by the necessity to work through an ASL interpreter. Some tasks, such as those requiring the patient to provide definitions to specific words or to identify objects based on a verbal description, really could not be considered with this patient. For example, conveying these test items using signs would often compromise the task, as the use of sign language sometimes provides the definition of a word when presenting the target stimuli. Scores and performance ratings for the formal neurocognitive tests administered appear in Table 1.

### Visuo-Perception, Visual-Spatial Processing, and Constructional Praxis

We first attempted to assess visuo-perceptual and visual-spatial processing, as the patient had exhibited difficulty reading a Snellen eye chart and seemed unable to focus on more than one or two objects at a time. Using a wall-sized Snellen eye chart, we were able to present a single letter at a time from each line of the chart, while covering the other letters with construction paper. Using this method, we were able to demonstrate that the patient was exhibiting 20/20 visual acuity. The patient's inability to see more than one object at a time sounded like simultanagnosia. Therefore, we attempted to explore this construct in more detail. One standard way to accomplish this goal, is to present a complex visual scene, such as the Cookie Theft picture from the Boston Diagnostic Aphasia Examination (BDAE) (Goodglass & Kaplan, 1983). On this task, the patient could only describe a few elements of the picture and appeared to neglect the periphery of the picture in all directions. In addition, her responses tended to be fragmented and isolated. That is, she identified individual objects without specifying any relationship between them. Further, she failed to put together an integrated response even after her daughter was asked to prompt her to find a common theme or to explain the role of a particular character. We also assessed the patient's ability to perceive multiple visual stimuli simultaneously by having her attempt to draw a circle and then place a dot at the center of it. While the patient could draw a circle, she was unable to place a dot in its center. Likewise, we presented the patient with a series of items that we constructed which involved deciding which of two figures was higher than the other on the page (see Figure 1). The patient performed this task at essentially chance levels and sometimes reported that she did not see two figures on the sheet of paper. Finally, we provided her with a large array of letters of various sizes and asked the patient to circle all of the "A's" observed on this task. The patient was not able to locate all of the target stimuli, initially only identifying two of six potential targets. She was able to find more items when being presented with the page of stimuli a second time. However, she never recognized the target items that were larger in size (See Figure 2). We also assessed more complex visual-spatial and constructional abilities, which were all severely impaired secondary to these other primary perceptual deficits. For example, the patient could not accurately judge the spatial relationship between an array of lines and exhibited an impaired performance on a block design task requiring her to assemble colored blocks to match a stimulus design on a card. Overall, while the patient exhibited normal visual acuity and intact visual-fields, she displayed left visual neglect and simultanagnosia as well as impaired performances on all visual-spatial and constructional tasks.<sup>1</sup>

While assessing visual functioning, it was also clear that the patient was experiencing optic ataxia (misreaching). This was elicited using a task that required the patient to touch a sequence of blocks. The patient tended to under-reach for the blocks included in this paradigm. Although errors tended to be mild in nature, her attempts to touch the blocks revealed movements that were tentative, tremulous, and irregular. In contrast, movements that were not visually directed appeared smooth. The patient also had difficulty completing movements that were visually guided, although she could often successfully carry-out the same movements if not focusing her visual attention on them. For example, she made multiple errors signing the alphabet when paying attention to her own hand movements. However, she completed this task easily and without errors when asked to perform it with her eyes closed.

There was no evidence of optic apraxia (impairment of visual tracking), as the patient appeared to be able to focus on a single target and could track such an object through space. Extraocular movements were normal. There was also no evidence of achromatopsia or color agnosia. These latter color phenomena were evaluated by having the patient identify the correct name for color blotches and indicate the typical color of various objects (e.g., what color is a pumpkin?), respectively.

### Language Processing

The patient's language functioning appeared grossly normal. She had no difficulty performing commands and instructions, as long as she was able to perceive the signs presented to her. She was also able to correctly sign the name of single items presented in a visual confrontational manner without difficulty (Boston Naming Test = 55/60) (Kaplan, Goodglass, & Weintraub, 1983). As noted previously, the patient's visual deficits kept her from being able to describe pictures more complex than a single object. The patient's family felt that her ability to sign was as rapid or fluent as ever, although we had no standards for formal assessment in this regard. The patient was able to recognize single letters but appeared unable to reliably read entire words.

### Memory Assessment

This patient's memory functioning appeared intact for both recent and remote information presented in a visual format. This was true of information of a visuo-perceptual or visual spatial nature as well as verbal materials. The patient exhibited a low average to average ability to recall unfamiliar faces following immediate presentation and after a brief period of delay. We used the faces subtest of the 3<sup>rd</sup> edition of the Wechsler Memory Scale (WMS-III) (Wechsler, 1997b), which presents only a single stimulus at a time, as anything more would impair the patient's learning and recall for other perceptual reasons. The patient exhibited average recall of verbal information (short paragraphs) from the Logical Memory subtest of the WMS-III. Both the presentation of the paragraphs and the patient's recall was completed using sign. She recalled at least an average amount of information, despite the obvious encoding limitations resulting from her difficulty processing signs. Finally, general orientation was normal in all spheres of awareness and the patient exhibited an intact personal history and a high average fund of general knowledge.

### Attention, Executive Functioning, and Mood Assessment

We also demonstrated that the patient exhibited normal mental flexibility, as she performed in the average range on the arithmetic subtest of the 3<sup>rd</sup> edition of the Wechsler Adult Intelligence Scale (WAIS-III) (Wechsler, 1997a) and was able to complete the sequencing tasks of the Oral

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<sup>1</sup>There was not enough time to arrange a neuro-ophthalmologic examination for the patient prior to her discharge. To our knowledge, she never pursued such an evaluation after returning home following her discharge.



Trailmaking Test (Axelrod, Ricker, & Konarzewski-Nassau, 1994), providing a signed response rather than a verbal response. Judgment and verbal abstract reasoning appeared normal using the Similarities and Comprehension subtests of the WAIS-III. No motor tasks were completed with the patient. Finally, the patient was signed a Beck Depression Inventory (Beck, Steer, & Brown, 1996), producing a mildly depressed score on this measure. She tended to report what appeared to be appropriate sadness related to her recent health problems and some mild neurovegetative features (i.e., mild sleep disturbance, heightened fatigue).

### Neuroimaging Findings

The patient underwent a clinical CT scan when presenting with left-sided weakness. This was interpreted as showing a right pontine stroke. There was also evidence of bilateral areas of decreased attenuation involving the occipital lobes and the white matter just beneath the occipital horns that were thought to represent microischemic change. As this scan was obtained for clinical purposes, it is limited in terms of its slice coverage and its spatial resolution, and could not be adequately converted into Talairach space. It included only 16 slices, with each being about 10 mm in thickness. Therefore we have provided a volumetric tracing of the bilateral lesions on the available CT slices on which they were present (see Figure 3). The lesion foci correspond to approximately  $\pm 35$ ,  $-40$ , and  $+25$  mm in Talairach coordinates. Figure 4 includes an approximate representation of these 3 CT slices represented on a healthy adult brain normalized to Talairach space. Of note, MRI images would have been more useful for exploring the white matter changes thought to be associated with this patient's visuo-ocular symptoms, but we were unable to obtain another scan during the patient's clinical stay.

### Course of Recovery, Interventions, and Outcome

The patient's positive visual symptoms (i.e., elementary visual hallucinations) continued for several days during her rehabilitation stay. One possible treatment option being considered was a trial of an antiepileptic medication in case that these symptoms represented aberrant electrical activity. Nevertheless, this phenomenon eventually resolved without treatment.

Over time, the patient seemed to adjust somewhat to her misreaching and the difficulty she initially exhibited judging distances. Nevertheless, she continued to exhibit simultanagnosia, impaired constructional ability, and limitations in reading ability throughout her stay in neurological rehabilitation. She also continued to exhibit difficulty completing visually guided tasks.

The patient and her family were fully apprised of her limitations as well as her intact areas of functioning. She no longer drove an automobile. They were encouraged to repeat signs when the patient seemed to have difficulty responding and instructed to keep visual targets in her right visual-field. In addition, she was coached to periodically explore her environment to the left in an exaggerated fashion. She was also shown that she could perform many rote motor tasks easier when she did not attempt to visually track her own performance. Given her vascular problems, the patient was encouraged to discontinue smoking cigarettes and to maintain tight control of her blood sugars. It turned out that she was not being followed regularly by any physician and that she was not monitoring her blood sugars routinely.

The patient and her family reported that her visual deficits appeared to resolve almost completely over time after returning to her home environment. However, she did not return for formal neurocognitive assessment and no further neuroimaging was ever obtained.

## Discussion

This case highlights a rare visual syndrome which is complicated by congenital deafness experienced by the patient. In addition to providing insight into the underlying neural substrates of this syndrome, this case provides an opportunity to explore a variety of clinical issues in neuropsychological assessment. Evaluation of a deaf patient relying upon sign language as their primary means of communication requires creativity and modification of standard test administration procedures, and appears akin to testing a patient in a second language. Moreover, combining auditory limitations with a disturbance of visual processing eliminated our ability to communicate with this woman through writing and compounded the need to modify existing procedures as well as to create new tasks to get at certain cognitive abilities. The patient's special needs in this area raise ethical issues regarding the assessment of patients for whom communication issues pose a potential barrier to performing a valid assessment (e.g., use of interpreters, translation of test materials, availability of appropriate normative data). We believe that this case demonstrates that useful data can be obtained even when such limitations appear substantial, as highlighted by our ability to rule-out a possible aphasia experienced by this individual while establishing her more primary visuo-ocular (perceptual) deficits.

Behavioral results and neuroimaging data suggest that our patient experienced a partial Balint's syndrome resulting from damage to her bilateral superior occipital lobes and adjacent white matter (dorsal stream "where" pathway). There is likely some involvement of the posterior parietal lobe (i.e., the angular gyrus, portions of the superior parietal lobule) bilaterally, as several studies suggest that this region is critical in producing this constellation of symptoms (Coslett & Saffran, 1991; Kerkhoff, 2001; Rafal, 1997). Our patient exhibited both simultanagnosia and optic ataxia (misreaching and impaired movements under visual guidance). The absence of optic apraxia suggests that at least some connections with the prefrontal cortex were spared. However, the presence of left hemispatial neglect suggests that the lesion in the right hemisphere may encroach upon the temporoparietal junction based upon common findings observed in similar cases (Kerkhoff, 2001; Rafal, 1997). The etiology underlying the white matter changes believed to be responsible for the onset of visual disturbance is uncertain. However, it is possible that the patient experienced an unrecognized hypotensive episode or that these changes and her pontine infarction shared a common cause.

Given that the patient is congenitally deaf and relies upon sign language to communicate, her experience of visual dysfunction hindered her ability to recognize signs made by others. Both her partial left visual-field neglect and simultanagnosia would be expected to contribute to limitations in processing visual input. Her problem with communication was initially interpreted by the patient's family and hospital staff as evidence of possible aphasia. In actuality, however, the patient demonstrated no evidence of language disturbance.

The positive visual phenomena experienced by the patient may have resulted from electrical disruption caused by the patient's vascular abnormalities. Visual illusions are known to frequently result from electrical stimulation, lesion occurrence, and seizure activity involving the occipital cortex (Adams et al., 1997). Visual illusions may present as distortions of form, size, movement, or color. Similarly, lesions involving the occipital lobe are associated with elementary visual hallucinations. Elementary visual hallucinations include flashes of light, colors, stars, and geometric forms that can be stationary or moving. It is assumed that these phenomena resolved as the brain returned to a more normalized metabolic and electrical status.

To our knowledge, there are no previously published reports of Balint's syndrome experienced by a deaf patient. However, there have a number of studies over the years exploring co-existing visual and auditory deficits in various developmental syndromes and neurological conditions, and their impact on the development of communication skills (Nikolopoulos, Lioumi,



Stamatakis, & O'Donoghue, 2006). Causes of co-occurring deafness and ophthalmic disorders have included congenital infections (e.g., rubella, syphilis, cytomegalovirus, and toxoplasmosis), syndromic disorders (e.g., Moebius Complex, fetal alcohol syndrome), genetic conditions (Alport's syndrome, Usher syndrome), and a variety of injuries (e.g., such as those associated with a premature birth). The visual deficits associated with these conditions include blindness, visual acuity problems, or strabismus (misalignment of the visual axes of one's eyes) among others. In many of these cases, it has been clear that co-existing deafness and visual deficits can hinder language development and communication. Correction of visual defects in these individuals, whenever possible, can contribute to improved communication ability.

Little research exists regarding potential impact of rehabilitation upon the visual defects associated with Balint's syndrome (Perez, Tunkel, Lachmann, & Nagler, 1996). A few case study presentations have suggested that some improvement can be observed when Balint's patients are provided with visuo-perceptual training and functional strategies provided in their natural environment (Gillen & Dutton, 2003; Rosselli, Ardila, & Beltran, 2001). While our patient reportedly improved over time, she did not undergo formal testing to verify the extent and nature of her recovery.

This patient's combination of deficits posed a significant assessment challenge. Areas of caution included a lack of standardized norms for special populations, obvious communication barriers, and the difficulty involved in maintaining a valid and reliable approach when unable to conduct an evaluation in a standard procedural fashion. Many of these variables are common to conducting neuropsychological evaluations with patients for whom English is not their native language and for those with significant motor and/or sensory deficits. In recent years, much has been written about practicing within the scope of our abilities, pointing out that a grave disservice can be done to our patients when we fail to appreciate these limitations (Boone, Victor, Wen, Razani, & Ponton, 2007; Fortuny & Mullaney, 1998). For example, we can underestimate an individual's neurocognitive and functional capacity due to problems communicating our questions and test instructions accurately. Nevertheless, we believe that the current assessment demonstrates that some very useful data can still be obtained as long as potential assessment limitations are readily and transparently acknowledged. For example, although we clearly demonstrated the nature of the patient's visuo-perceptual deficits and her intact language functioning, we would have had difficulty providing a valid evaluation of her IQ. It was particularly difficult to query her about verbal knowledge, as her visuo-perceptual limitations made it troublesome to convey and clarify some questions (i.e., she had difficulty reading text as well as responding to sign language and finger spelling).

This case study also highlights the need to sometimes push beyond our standardized test batteries in order to explore unusual symptom presentations. While the backbone of neuropsychology is the application of standardized assessment procedures to the study of brain functions, situations do arise that call for approach modifications in order to obtain useful data. The ability to devise and implement atypical assessment strategies requires a strong knowledge of brain-behavior relationships (including both common and unusual neurological syndromes), and the ability to break complex neurocognitive tasks into their component parts.

Much of the modern focus on forensic applications to neuropsychology discourage variation from the standardized battery approach, yet in cases such as this one, a standard assessment would likely have missed the essential features of this patient's syndrome. Of course, even when departing from a standard battery, systematic, rigorous standards can yet be applied to the administration of an experimental measure. Specialized techniques to assess some of these more unusual deficits can be discovered through a careful literature search, including a review of some of our common assessment texts. Likewise, consultation with other neuropsychologists or related professionals who have specific expertise in an area relevant to an unusual

presentation is extremely helpful (e.g., in this case, this included colleagues with expertise in seizure disorders, visual processing and related neurologic phenomena, neuroimaging, stroke, and communication issues with the congenitally deaf). On a related note, as we develop novel test strategies, an effort to make these available to our colleagues should be undertaken as well.

Finally, this case highlights the contributions that neuropsychology can make in the assessment of acute behavioral or emotional changes as they occur in the setting of neurological or general medical treatment facilities. While this is also the domain of the behavioral neurologist, there are many settings where such professionals are lacking. Likewise, we can often provide complimentary data to such providers, adding a degree of quantification to what might otherwise be a qualitative judgment. This is another area where neuropsychology can make substantial contributions with real impact on patient care that is often overlooked despite a definite heritage in this arena (e.g., see the pioneering work of A. R. Luria, A. Benton).

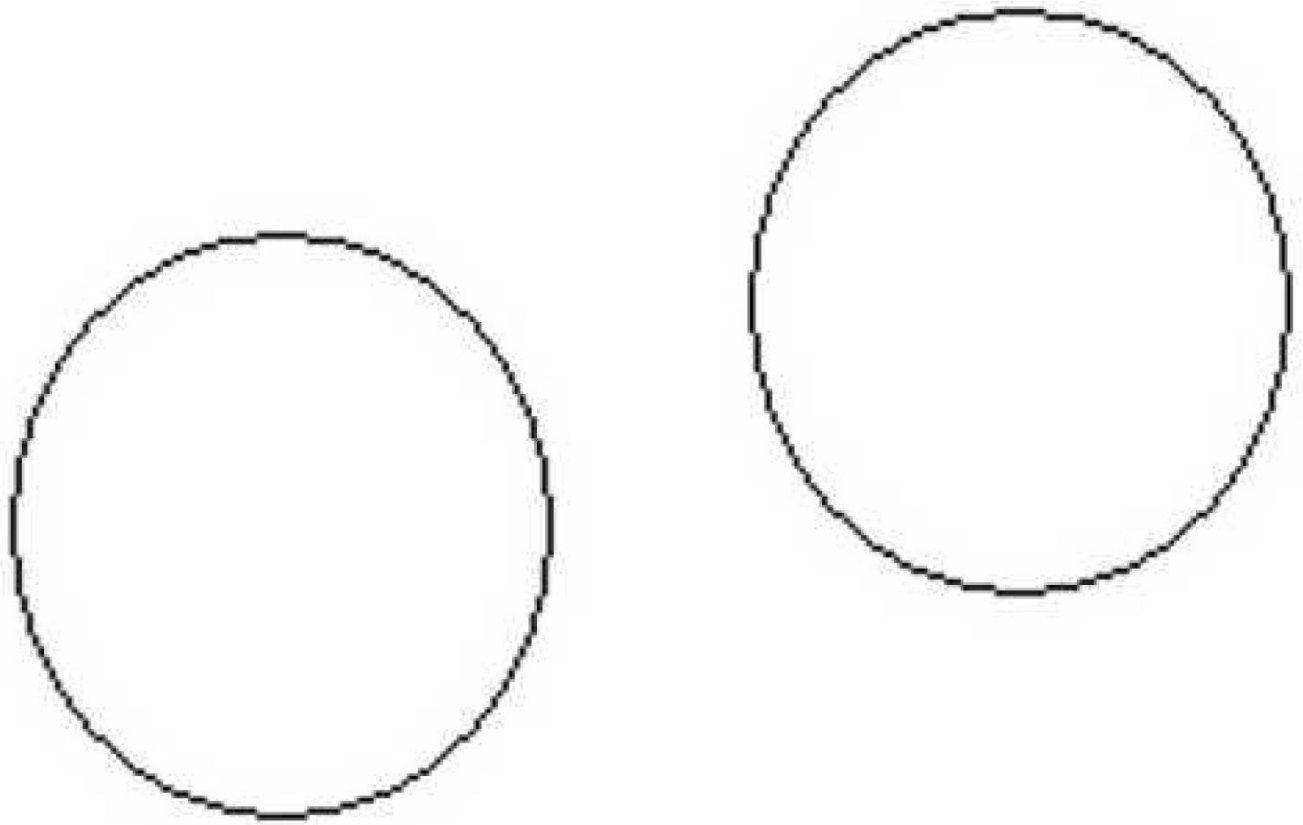
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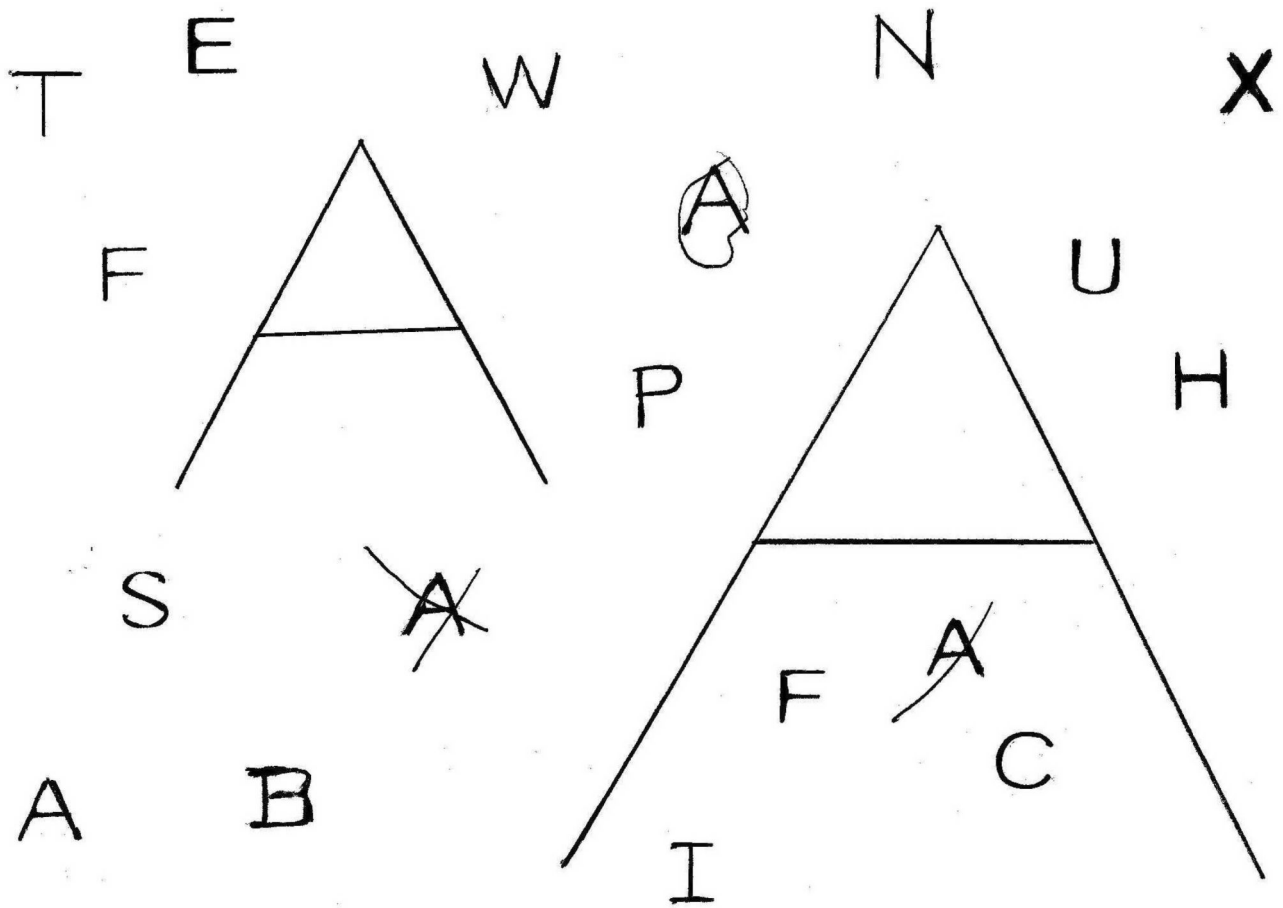
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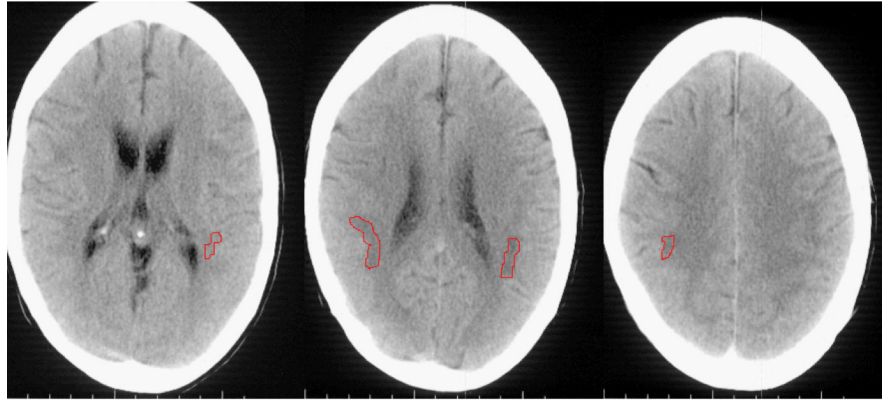
**Figure 1.**

This is a sample of one of several test items used to assess the patient's ability to estimate the spatial relationship between pictured objects. She was instructed on this task to indicate which of the two figures is higher.



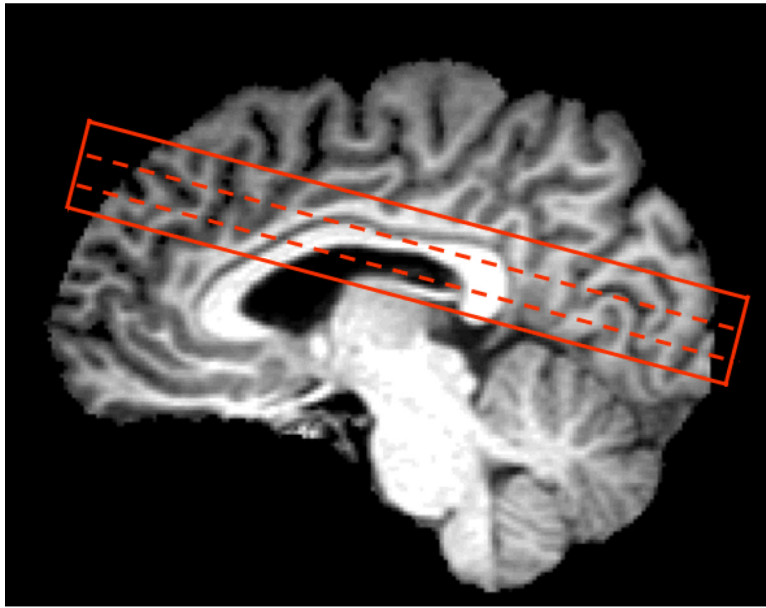
**Figure 2.**

This represents the patient's attempt to circle all letter "A's" pictured on a sheet of letters. On her first attempt she identified only two of the six target letters. She identified one additional target when prompted to scan again. However, she missed a number of target letters, particularly those that were larger in size.



**Figure 3.** This is a volumetric tracing of the bilateral lesions on the available CT slices on which they were present. The lesion foci correspond to approximately  $\pm 35$ ,  $-40$ , and  $+25$  mm in Talairach coordinates.





**Figure 4.** Depiction of the 3 CT scan slices from Figure 3 on a healthy adult brain normalized to Talairach space.

**Table 1**

Scores obtained on both formal and qualitative neurocognitive measures administered.

Tests Administered	Scale Score or Raw Score	Performance Rating
WMS-III General Orientation	13/14	Normal
Boston Naming Test	55/60	Average
Oral Trailmaking Test – Part A	5.5 seconds	Average
Oral Trailmaking Test – Part B	26.3 seconds	Average
WAIS-III Arithmetic	SS = 8	Average
WMS-III Spatial Span	SS = 3	Impaired
Visual Acuity (modified Snellen Eye Chart)	20/20	Normal
Drawing a Circle/Placing Dot in Center	Not able to perform task	Impaired
Judgment of Line Orientation Test	4/30	Impaired
Judging the Relative Spatial Relationship between objects	Patient missed greater than 80% of test items	Impaired
WAIS-III Block Design	SS = 3	Impaired
WMS-III Faces I – Immediate Recall	SS = 7	Low Average
WMS-III Faces II – Delayed Recall	SS = 8	Average
WMS-III Logical Memory I – Immediate Recall	SS = 9	Average
WMS-III Logical Memory II– Delayed Recall	SS = 10	Average
WAIS-III Information	SS = 12	High Average
WAIS-III Similarities	SS = 9	Average
WAIS-III Comprehension	SS = 8	Average
Beck Depression Inventory	10/30	Mildly Depressed

Note. The Snellen eye chart was modified by exposing only a single letter at a time. Otherwise, the patient would become fixated on only a single letter. WMS-III = Wechsler Memory Scale (3<sup>rd</sup> Edition); WAIS-III = Wechsler Adult Intelligence Scale (3<sup>rd</sup> Edition).