THE AGNOSIAS

Russell M. Bauer, Ph.D.

University of Florida

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Correspondence to: Russell M. Bauer, Ph.D., Department of Clinical and Health Psychology, University of Florida, PO Box 100165 HSC, Gainesville, FL 32610-0165 USA

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Russell M. Bauer, Ph.D.

University of Florida

The ***agnosias*** are rare disorders in which a patient with brain damage becomes unable to recognize or appreciate the identity or nature of sensory stimuli. Clinical examination of the patient reveals a profound, modality-specific recognition impairment that cannot be fully explained by problems in elementary sensory processing, mental deterioration, attentional disturbances, aphasic misnaming, or to unfamiliarity with the stimuli used to assess recognition abilities. Classically, a distinction between **apperceptive** and **associative** forms of agnosia has been made whereby the patient with **apperceptive** agnosia is said to have deficits in early stages of perceptual processing while the patient with **associative** agnosia either does not display such problems or does so to a degree not sufficient to substantially impair the ability to perform perceptual operations. The associative agnosic can typically draw, copy, or match unidentified objects, while the apperceptive agnosic cannot. This distinction has been clinically useful, though it is clear that nearly all agnosics have *some* degree of perceptual ("apperceptive") disturbance. It should be remembered that adequate copying or matching *by itself* does not indicate normal perceptual processing (see Farah, 1990; Bauer & Demery, 2003).

Clinical assessment of the putative agnosic patient has two fundamental goals. **First,** the possibility that the recognition disturbance exists because of elementary sensory disturbance, dementia, aphasia, or unfamiliarity with the stimulus should be ruled out with standardized neuropsychological testing instruments. **Second**, the scope and nature of the patient's recognition disturbance should be determined. Does the recognition disturbance exist only for certain stimuli or classes of stimuli? Is it restricted to a particular sensory modality? Under what conditions (if

any) can the patient recognize stimuli? This phase of the evaluation often requires detailed testing using specially formulated testing materials, and should be conducted from the point of view of cognitive models of recognition disturbance (see below). Appropriate referrals for neurologic, neuroradiologic, and basic sensory-perceptual (e.g., ophthalmologic, audiologic) testing are often important in formulating a clinical diagnosis.

I. BASIC DEFINITIONS

Several types of agnosia have been identified in the literature. References summarizing the basic subtypes, clinicoanatomic correlations, and neurobehavioral mechanisms producing disturbances in recognition include Bauer & Demery (2003) and Farah (1990). Humphreys & Riddoch (1987) provide an excellent book-length description of a visual agnosic written from a cognitive neuropsychology perspective. "Pure" forms of these disorders are quite rare and the etiology of the patient's disorder (e.g., whether from focal stroke vs. a more diffuse cause such as carbon monoxide poisoning) and/or the stage of recovery (if acute onset) will determine the observed pattern of deficits. Defining characteristics of the basic subtypes of agnosia are given below and in Table 1. The remainder of this section provides basic characteristics of agnosia in outline form. This method of presentation is intended to stimulate attempts at differential diagnosis, but should not discourage attempts at more in-depth analysis of presenting syndromes.

I.1. VISUAL AGNOSIAS

I.1.1. **Visual Object Agnosia.**

a. Key Features

1. cannot recognize the meaning of visually-presented objects
2. disorder is not restricted to naming (e.g., patient cannot point to the object when named or describe or demonstrate its use)

3. sometimes, recognition is better for real objects than for pictures or line drawings
4. can recognize objects when presented in other modalities

b. Varieties:

1. apperceptive: cannot demonstrate adequate perception of object through drawing, copying, or matching tasks
2. associative: drawing, copying, or matching tasks bring more success, though performance is sometimes 'slavish'

c. Sometimes recognition disturbance is worse for certain categories of objects (e.g., living things, tools, etc.); recognition testing should employ various categories of objects

I.1.2. **Simultanagnosia.**

a. Key Features

1. Patient cannot apprehend the overall meaning of a picture or stimulus, but may be able to appreciate and describe isolated elements.

b. Varieties

1. "dorsal" simultanagnosia (bilateral occipitoparietal lesions); cannot see more than one object at a time
2. "ventral" simultanagnosia (left inferior occipital lesions); may be able to "see" more than one object at a time

c. Often considered a variant of apperceptive agnosia

I.1.3. **Prosopagnosia.**

a. Key Features:

1. unable to recognize the identity of viewed faces
2. often can appreciate aspects of faces such as age, gender, or emotional expression.

b. Varieties: Apperceptive and associative forms have been identified on the basis of matching tasks.

c. Associated features: Within-class recognition of other types of visually similar objects (e.g., recognition of individual chairs, cars, animals, etc.) may be impaired

I.1.4. **Color Agnosia.** Because colors can only appreciated visually, the status of 'color agnosia' as a true agnosic deficit has been difficult to establish. Nonetheless, four classes of patients have been identified with disproportional impairment in recognizing, naming, or otherwise utilizing color information.

a. ***central achromatopsia:*** acquired deficit in color vision due to CNS disease. Cannot match, discriminate, or name colors. Suspect bilateral occipital lesions, but may be unilateral

b. c***olor anomia:*** specific difficulty in naming colors, usually found in the context of right homonymous hemianopia and pure alexia (Geschwind, 1965). Other aphasic signs generally absent; suspect posterior left hemisphere lesion

c. ***specific color aphasia:*** seen in the context of aphasia, represents a disproportionate difficulty in naming colors; suspect left (dominant) parietal lobe damage

d. ***color agnosia***: this is a residual category of patients who have difficulty appreciating the nature or name of color they see, but who do not fall within the categories above

I.1.5 **Optic Aphasia.**

a. Key Features:

1. patient cannot name a visually-presented object
2. *can* demonstrate its use by gesture, or can point to it when named

b. not regarded as a true agnosia

c. may represent a visual-verbal disconnection

I.2. AUDITORY AGNOSIAS

Subtypes of auditory agnosia have been distinguished on the basis of the type of auditory stimulus the patient has difficulty recognizing (Bauer & McDonald, 2003). Although much remains to be understood about these disorders (which have not been studied as exhaustively as cases of visual agnosia), three general classes of deficits have been described.

I.2.1. Cortical Auditory Disorder and Cortical Deafness.

a. Key Features:

1. difficulty recognizing auditory stimuli of many kinds, verbal and nonverbal.
2. Basic audiologic testing is abnormal

b. Varieties:

1. cortical deafness: complains of a subjective sense of deafness,
2. cortical auditory disorder: no subjective sense of deafness

c. Such patients may evolve to one of the more selective types of auditory agnosia described below; longitudinal assessment is important

I.2.2. <u>Pure Word Deafness</u>. (Buchman et al., 1986)

a. Key Features:

1. inability to comprehend spoken language but can read, write, and speak in a *relatively* normal manner.
2. comprehension of nonverbal sounds is relatively spared.
3. patient is relatively free of aphasic symptoms found with other disorders affecting language comprehension

I.2.3. <u>Auditory Sound Agnosia (Auditory Agnosia for Nonspeech Sounds)</u>.

a. Key Features

1. inability to comprehend meaning of common environmental sounds, with relative sparing of speech comprehension
2. far more rare than pure word deafness

b. Varieties (Vignolo, 1969):

1. perceptual-discriminative form: makes predominantly acoustic errors (e.g., “whistling” for birdsong)
2. semantic-associative form: makes predominantly semantic errors (e.g., “train” for automobile engine)

I.2.4. <u>Sensory (Receptive) Amusia</u>.

a. Key Features

1. inability to appreciate various characteristics of heard music

2. impairment in perceptual vs. conceptual aspects of music should be evaluated

b. impaired music perception occurs to some extent in all cases of auditory sound agnosia, and in most cases of aphasia and pure word deafness; exact prevalence unknown

c. probably underreported because a specific musical disorder rarely interferes with everyday life.

d. perception of pitch, harmony, timbre, intensity and rhythm may be affected to different degrees or in various combinations

I.3. TACTILE AGNOSIAS. Compared to visual agnosias, somatosensory (tactile) agnosias have received scant attention and are poorly understood. Several distinct disorders have been identified, and many classifications of tactile agnosia have been offered. Delay (1935) distinguished three disorders, including: (a) **"amorphognosia**", impaired recognition of the size and shape of objects, (b) "**ahylognosia**", impaired recognition of the distinctive qualities of objects such as weight, density, texture, and thermal properties, and (c) "**tactile asymbolia**", impaired recognition of tactile objects in the absence of amorphognosia or ahylognosia. Although only tentative, a clinically useful distinction can be made between "cortical tactile disorders" (which probably encompass the first two of Delay's deficit classes), and "tactile agnosia", which represents an inability to appreciate the nature of tactually manipulated objects.

I.3.1 Cortical Tactile Disorders.

a. Key Features

1. deficits in appreciating distinct object qualities such as size, shape, weight, or spatial configuration of tactually presented objects.

b. Varieties: some patients have especially obvious defects of size discrimination, while others fail in tasks which emphasize the spatial character of tactually manipulated objects

c. No hemispheric specialization exists in elementary somatosensory function, but patients with right hemisphere disease may have difficulty in performing the spatial component of many tactile discrimination tasks.

I.3.2 Tactile Agnosia.

a. Key Features:

1. cannot identify objects placed in the hand despite
2. elementary sensory function intact

b. Varieties:

1. Deficit exists in both hands: an “agnosic” deficit (an inability to appreciate the nature of stimuli because of a central defect in processing the nature of a stimulus); cannot demonstrate use of object through gesture

2. Deficit exists in one (usually left) hand: a “visual-verbal disconnection”; can demonstrate use of the object, and can name the object if placed in the other hand

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Table 1 About Here

II. Neuroanatomical Correlates

Lesion localization based on individual case studies and recent reviews of the agnosic syndromes described above is presented in Table 2. In general, apperceptive agnosias involve more extensive damage to sensory association cortex while associative agnosias result from lesions of corticocortical pathways or from impairment in those areas where semantic representations of objects are stored. In most published cases, lesions are caused by ischemic stroke, though cases of carbon monoxide poisoning, post-traumatic hematoma, and neoplasm have been reported (Bauer & Demery, 2003; Farah, 1990). It is becoming increasingly recognized (most prominently in the visual domain) that apperceptive agnosia can result from degenerative disease, with particular attention being devoted to dementia syndromes presenting with predominant visuoperceptual disturbance (Biran & Coslett, 2003; Caselli, 2000; Jackson & Owsley, 2003; Mendez, Mendez , Martin, et al., 1990).

Table 2 About Here

III. Differential Diagnosis of Agnosia

A. Basic Decision-Making Process in Differential Diagnosis.

Diagnosis of the agnosias first proceeds by identifying the basic characteristics of the patient's recognition defect. The process of reaching a tentative initial diagnosis is outlined in flowchart form in Figure 1.

Figure 1a and 1b About Here

In applying the flowchart, clinicians should remember that "pure" forms of agnosia are not commonly encountered. The first part of the flowchart (Fig. 1a) presents three "streams" representing visual, auditory, and tactile agnosias, respectively, and outlines basic questions which should be asked in making a tentative initial diagnosis. The second part of the flowchart (Fig. 1b) deals specifically with visual agnosias, which are more common and are better understood than their auditory and tactile counterparts.

The flowchart assumes that simple materials for bedside testing are available (or can be manufactured) and that the clinician consults other disciplines in order to further document the extent of neuroanatomic damage and to better characterize sensory/perceptual function. In many cases, the physician and/or the treatment team make such referrals, but it should be remembered that the informed neuropsychologist can serve as a valuable advisor in insuring that appropriate referrals are made. In addition to an extended behaviorally oriented neurological examination, potentially useful referrals include neuroimaging consults (CT/MRI), evoked potential studies, and referrals to ophthalmology, speech pathology/audiology or other professionals for more detailed evaluation of sensory/perceptual and neurobehavioral status. Obviously, referral

decisions should not be made automatically, but should depend on the likely cost-effectiveness of obtaining the requested information.

B. Neuropsychological Assessment in Differential Diagnosis.

Once a tentative diagnosis has been reached (or once the clinician has narrowed the differential diagnosis to a subset of possible disorders based on clinical presentation) formal assessment of neuropsychological skills is indicated. As indicated earlier, neuropsychological assessment of the putative agnosic seeks to (a) rule out alternative explanations of the patient's deficit, and (b) characterize in more precise terms the nature of the patient's deficit so that its underlying mechanism and it relationship to pathological anatomy can be understood.

1. Ruling Out Alternative Explanations.

As suggested earlier, disturbances of "recognition" can occur in a variety of neurological conditions but are considered 'agnosic' only if they exist in the relative absence of aphasia, generalized dementia, impaired attentional capacity or other defect that nonspecifically impairs some or all of the information processing steps involved in object recognition. Therefore, one critical aspect of the assessment of the agnosic patient involves assessment of these 'bracketing' conditions in order to rule them out as explanations for the recognition defect. A review of available case reports reveals considerable variability in the methods used for this portion of the assessment. Table 3 presents a reasonable strategy for achieving this goal, though it is recognized that many other tests are available for achieving this purpose.

Table 3 About Here

In general, patients should receive basic neuropsychological examination designed to determine general intellectual status, memory function, linguistic competence, and to assess sensory-perceptual processing. The clinician may wish to perform a comprehensive neuropsychological battery in order to better understand the patient's cognitive strengths and weaknesses, to document 'baseline' functioning, or to assist in treatment planning. Assessment of language ability (naming, auditory comprehension, fluency, repetition, reading, writing, and praxis) is especially important in understanding the possible role that linguistic factors might play in the patient's recognition defect. A comprehensive aphasia battery (e.g., ***Boston Diagnostic Aphasia Examination***, Goodglass & Kaplan, 1983; ***Multilingual Aphasia Examination***, Benton & Hamsher, 1989; ***Western Aphasia Battery***, Kertesz, 1982) is useful for this purpose, though it may be necessary to perform supplementary tests to insure that naming and recognition are tested in all sensory modalities.

2. Characterizing the Nature of the Agnosic Deficit

Once the patient's general neuropsychological status has been determined, the clinician will want to perform further testing to more precisely characterize the nature of the patient's recognition deficit. At this stage, cognitive neuropsychological models of the perceptual-recognition process become helpful in guiding the approach to assessment. A representative model, adapted from Ellis & Young (1988), is presented in Figure 2. Consulting individual case reports contained in Tables 1 and 2 will also assist in planning an appropriate assessment.

Figure 2 About Here

Figure 2 draws on a diverse literature in perceptual psychology and neuropsychology (Ellis & Young, 1988) and is presented to the clinician because such models have succeeded in parsing the process of object recognition into distinct information-processing components or stages. The left side of Figure 2 represents dissociable stages of the object recognition process suggested by clinical and experimental research. The right side of Figure 2 presents the most important implications of the model for clinical assessment, and suggests some commonly available tests that can be utilized in "localizing" the defect at a particular processing level. Defects before the level of the "object recognition unit" can be roughly considered **apperceptive** in nature, while subsequent deficits correspond to **associative** forms of agnosia. The model presented in Figure 2 is obviously best suited to evaluating a visual recognition disturbance, but should provide guidance in assessing auditory and tactile agnosia as well. A comprehensive evaluation proceeds by evaluating all levels of the model, even in situations where 'early' deficits are found.

IV. Relevant Laboratory, EEG, and Neuroimaging Correlates

As a general neuropsychological classification, agnosia is not associated with any definitive pattern of abnormality in laboratory tests. EEG and neuroimaging findings vary with the type of agnosia, as might be anticipated from lesion localization data presented in Table 2. The most common etiologies of agnosia include CVA, tumor, carbon monoxide poisoning, closed head injury, and CNS infection, though as indicated earlier, it is becoming increasingly recognized that some cases of degenerative dementia with primary involvement of posterior cortex can present with prominent signs of (primarily apperceptive) agnosia. Medical findings vary with etiology and localization. Because of these considerations, it can be said that laboratory, EEG, and neuroradiological findings *per se* do not typically play an integral role in differential diagnosis. One exception to this rule is the occasional utility of auditory or visual

evoked potentials as a way of determining whether a defect exists in the sensory projection areas as opposed to the primary sensory or association cortex. Instead, the clinician should rely on behavioral factors and should consider the physical findings as confirmatory.

V. Psychological/Psychiatric Comorbidity

The lesions most likely to produce agnosic defects often spare limbic, paralimbic, or frontal regions that, when damaged, produce primary affective or personality changes. For this reason, specific forms of psychopathology are not obligatory accompaniments of agnosic syndromes. However, secondary emotional reactions to the real-life consequences of agnosia are common. Factors such as unemployment, changes in social life, dependency on others for help in everyday activities (i.e. dressing, transportation, eating), and boredom are seen. These major lifestyle changes may lead to depression or adjustment disorders in some individuals, while others may find adaptive ways to cope. As an excellent example, Humphreys and Riddoch (1987) describe in detail how their patient, John, and his wife both cope with John's visual agnosia. Their description contains evidence of both adaptive and maladaptive compensations. Although epidemiological studies have yet to be conducted, auditory and tactile agnosias seem less likely to produce major life changes so that is may be that such disorders have less deleterious consequences. Such speculations await definitive research.

Another trait sometimes seen in agnosics is sensory compensation. This is an interesting and as yet unresearched phenomenon reported in the animal literature (Horel & Keating, 1969), in which the agnosic comes to rely on intact sensory modalities (e.g., audition and touch in the case of visual agnosia) in exploratory activity. Whether this represents an attempt to achieve an optimal arousal level through sensory stimulation or an attempt to gain understanding of the world through an intact modality remains to be seen. For example, Bauer's (1984) patient with

severe visual agnosia listens to music constantly to lessen the boredom of living with the disorder. In our experience, substance abuse is a risk in the chronic period, possibly in response to the reduced stimulation that results from an agnosic deficit, and possibly a result of premorbid factors. It should be emphasized that one problem in understanding psychiatric comorbidity in agnosia is that the relative rarity of these syndromes complicates an analysis of whether such problems are caused or exacerbated by the underlying neurological impairment or whether the appearance of such problems reflects preinjury factors which would have exerted themselves in any event. Such issues await systematic research.

VI. Summary

Agnosia refers to an acquired impairment in the ability to recognize the identity or nature of sensory stimuli. It is a relatively rare disorder that can produce significant everyday impairment. No specific laboratory or neuroradiological marker exists, though orderly anatomic findings have been reported in the literature on visual, auditory, and tactile agnosia that should serve, if present, to raise suspicion about the diagnosis in the individual case. Key symptoms, characteristic neuroradiologic findings, and a general assessment approach based on cognitive neuropsychological models of object recognition were summarized in this chapter. Although significant progress has recently been made, much remains to be learned about these complex disorders, and clinicians are encouraged to take a hypothesis-oriented approach in order to enlarge the available knowledge base.

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Table 1: Subtypes of Agnosia: Defining Characteristics and Key References

Subtype	Affected Stimulus Category	Varieties	Basis for Distinction	Suggested Reference
Visual Agnosias				Farah, 1990
Visual Object Agnosia	objects	a) apperceptive b) associative	a) drawing, matching - b) drawing, matching +	Benson & Greenberg, 1969 Rubens & Benson, 1971
Simultanagnosia	multiple objects or pictures	a) dorsal b) ventral	a) cannot see multiple items b) can see multiple items	Hecaen & Ajuriaguerra, 1954 Kinsbourne & Warrington, 1962
Prosopagnosia	faces	a) apperceptive b) associative	a) match, categorize faces - b) match, categorize faces +	DeRenzi et al., 1991 Pallis, 1955
Color Agnosia	colors	a) achromatopsia b) color anomia c) 'color aphasia' d) color agnosia	a) failure of color vision b) can succeed at nonverbal color tasks c) disproportionate deficit with color names d) residual category	Damasio et al. 1980 Geschwind & Fusillo 1966 Kinsbourne & Warrington, 1964
Auditory Agnosias				Vignolo, 1969
Cortical Deafness and Cortical Auditory Disorder	all sounds	a) cortical deafness b) agnosia	a) subjective deafness? b) patient claims not to be deaf	Michel et al., 1980 Kanshepolsky, et al., 1973
Pure Word Deafness	speech sounds	a) prephonemic b) phonemic	a) auditory acuity generally impaired b) disorder of phonemic discrimination	Buchman et al., 1986
Nonverbal auditory Agnosia	nonspeech sounds	a) perceptual b) associative	a) misidentifications primarily acoustic b) misidentifications primarily semantic	Spreen et al., 1965
Sensory (receptive) amusia	musical sounds		--	Bauer & McDonald, 2003
Tactile Agnosias				Delay, 1935 Caselli , 1991
Cortical TactileDisorder	tactually presented objects and object qualities	a) object-based? b) spatial?	a) fail on object discrimination tasks b) fail on tasks requiring spatial discrim	Corkin, 1978 Semmes, 1965
Tactile Agnosia	tactually presented objects	a) disconnection b) agnosic	a)unilateral; can demonstrate object use b) bimanual, cannot demonstrate object knowledge	Geschwind & Kaplan, 1965 Hecaen & David, 1945

Note: +=function is spared; -=function is impaired

Table 2. Lesion Localization for Various Forms of Agnosia

Disorder	Lesion Localization	Reference
VISUAL AGNOSIAS		
1)Apperceptive VOA	Diffuse, posterior damage to occipital lobes and surrounding regions	Benson & Greenberg, 1969
2)Associative VOA	Bilateral: Inferior occipitotemporal	Rubens & Benson, 1971
3)Simultanagnosia		
a) Dorsal	Bilateral parietal and superior occipital	Farah, 1990
	Localized bilaterally to either superior occipital or inferior parietal lobes	Hecaen & Ajuriaguerra, 1954
b) Ventral	Dominant occipitotemporal junction	Kinsbourne & Warrington, 1962
4)Prosopagnosia		
a) Apperceptive	Traditionally seen as bilateral in all or nearly all cases. Cortex and white matter in occipitotemporal gyrus or projection system.	Bauer & Demery, 2003
	More recently a few cases of what appears to be unilateral damage to right visual association cortices within occipital and parietal lobes.	Damasio et al., 1990 DeRenzi, 1986
b) Associative	Bilateral anterior temporal regions compromising hippocampal and other regions	Damasio et al., 1990
5)Color Agnosia		
a) Achromatopsia	Unilateral or bilateral inferior ventromedial region of occipital lobe - involves lingual and fusiform gyri - superior field defects	Damasio et al., 1982
b) Color Anomia	Dominant occipital infarction with corpus callosum involvement	Geschwind & Fusillo, 1966
c) Specific Color Aphasia	Dominant parietal damage coincident with posterior aphasia	Kinsbourne and Warrington, 1964
6) Optic Aphasia	Unilateral: Dominant occipital lobe and splenium	Riddoch & Humphreys, 1987 Geschwind, 1965
AUDITORY AGNOSIA		
1)Cortical Auditory Disorder	Variable- can involve superior temporal gyrus and efferent connections of Heschl's gyrus or bilateral subcortical lesions	Kazui et al., 1990 Oppenheimer and Newcombe, 1978
2)Pure Word Deafness	Bilateral: Symmetrical lesions of anterior section of superior temporal gyri - Most often bilateral disconnections of Wernicke's area from auditory input	Buchman, et al., 1986
	Unilateral (Rare): Deep subcortical in dominant superior temporal region damaging primarily auditory cortex and/or pathways to and from medial geniculate gyrus	Weisenburg and McBride, 1935/1964
3) Auditory Sound Agnosia		
a)Perceptual-Discrimination	Nondominant hemisphere	Vignolo, 1969

Type		
b) Semantic-Associative Type	Dominant hemisphere - linked with posterior aphasia	Vignolo, 1969
4)Sensory (Receptive) Amusia	Unilateral temporal lobe - if comorbid with aphasia, lesion is on dominant side.	Bauer and McDonald, 2003
TACTILE AGNOSIAS		
1) Cortical Tactile Disorders	Severe and Long-Lasting: Contralateral postcentral gyrus Less severe, bilateral lesions of SII	Corkin, 1978
2) Unilateral Tactile Anomia	Corpus callosum (affecting crossing somatosensory fibers (minimally; actual lesion may be more extensive)	Geschwind & Kaplan, 1962
3) Tactile Agnosia	Contralateral primary somatosensory projection area in postcentral gyrus	Caselli, 1991

Table 3: Ruling out Alternative Causes of Recognition Disturbance

Condition or Problem	Assessment Instruments	Domains Tested	Reference
Generalized Dementia	Dementia Rating Scale	memory, attention/concentration, construction, initiation/perseveration	Mattis (1988)
Aphasia	Boston Diagnostic Aphasia Exam Multilingual Aphasia Exam Western Aphasia Battery	fluency, comprehension, naming, repetition, reading, writing, praxis	Goodglass & Kaplan (1983) Benton & Hamsher (1989) Kertesz (1982)
Disturbances of Attention/Orientation (e.g., delirium)	Temporal Orientation Test Visual Search and Attention Test WAIS-R Digit Span Sentence Repetition WMS-R Mental Control Line Bisection	time orientation visual search and selectivity focused attention span focused attention span (sentences) mental tracking, sustained attention spatial attention, hemispatial neglect	Benton, Sivan, et al. (1994) Trenerry et al. (1990) Benton & Hamsher (1989) Schenkenberg et al. (1980)
Unfamiliarity with Stimuli	Determined subjectively; the examiner needs to insure that failures of naming/identification are not based on experiential, cultural, or other factors that lead to the patient's unfamiliarity with stimuli tested; use of common or frequently-encountered items typically circumvents this problem	visual, auditory, and tactile object identification with common objects should be tested in each patient to determine familiarity statistics and to determine modality specificity; subjects who cannot name objects should be encouraged to divulge anything they know about it or to group items into familiar and unfamiliar categories	Familiarity must be determined, even informally, on an individual-subject basis. If creating in-house stimulus sets, general references containing relevant statistics on item frequency, imageability, etc. should be consulted in order to construct a balanced set of items

Figure 1a
Flowchart for Clinical Decision-Making
Differential Diagnosis of Agnosia

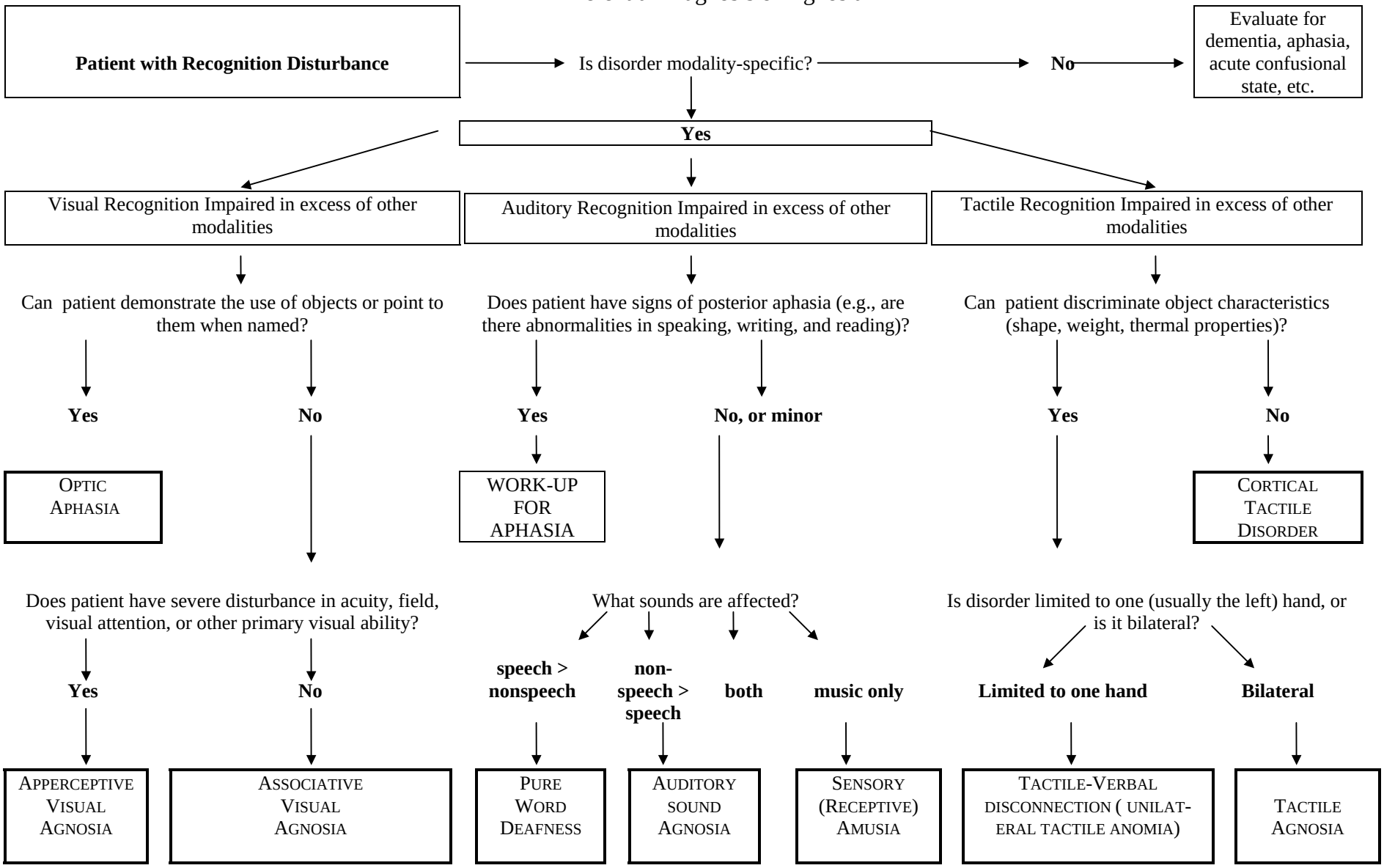


Figure 1b: Differential Diagnosis of Visual Agnosia

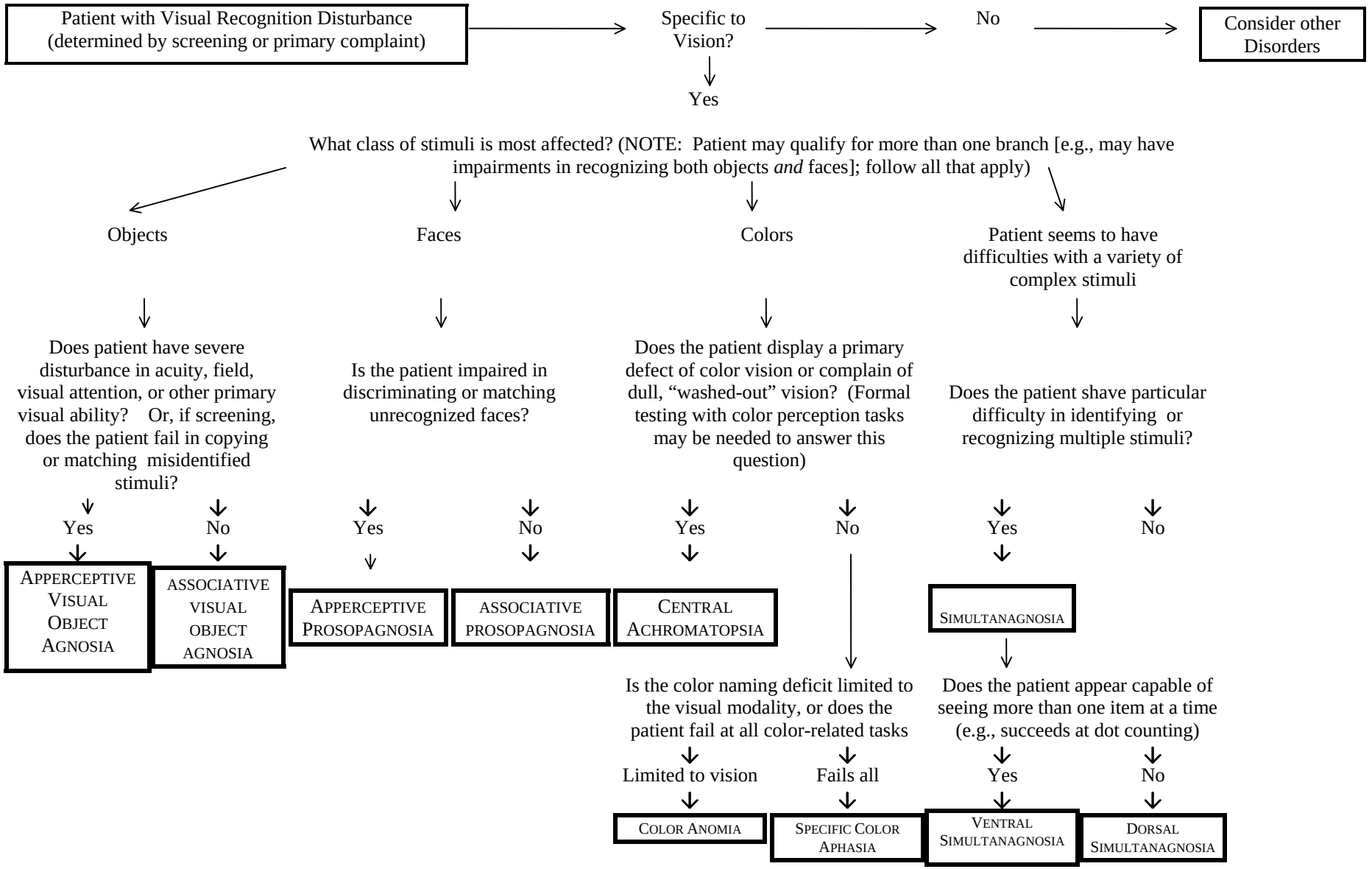
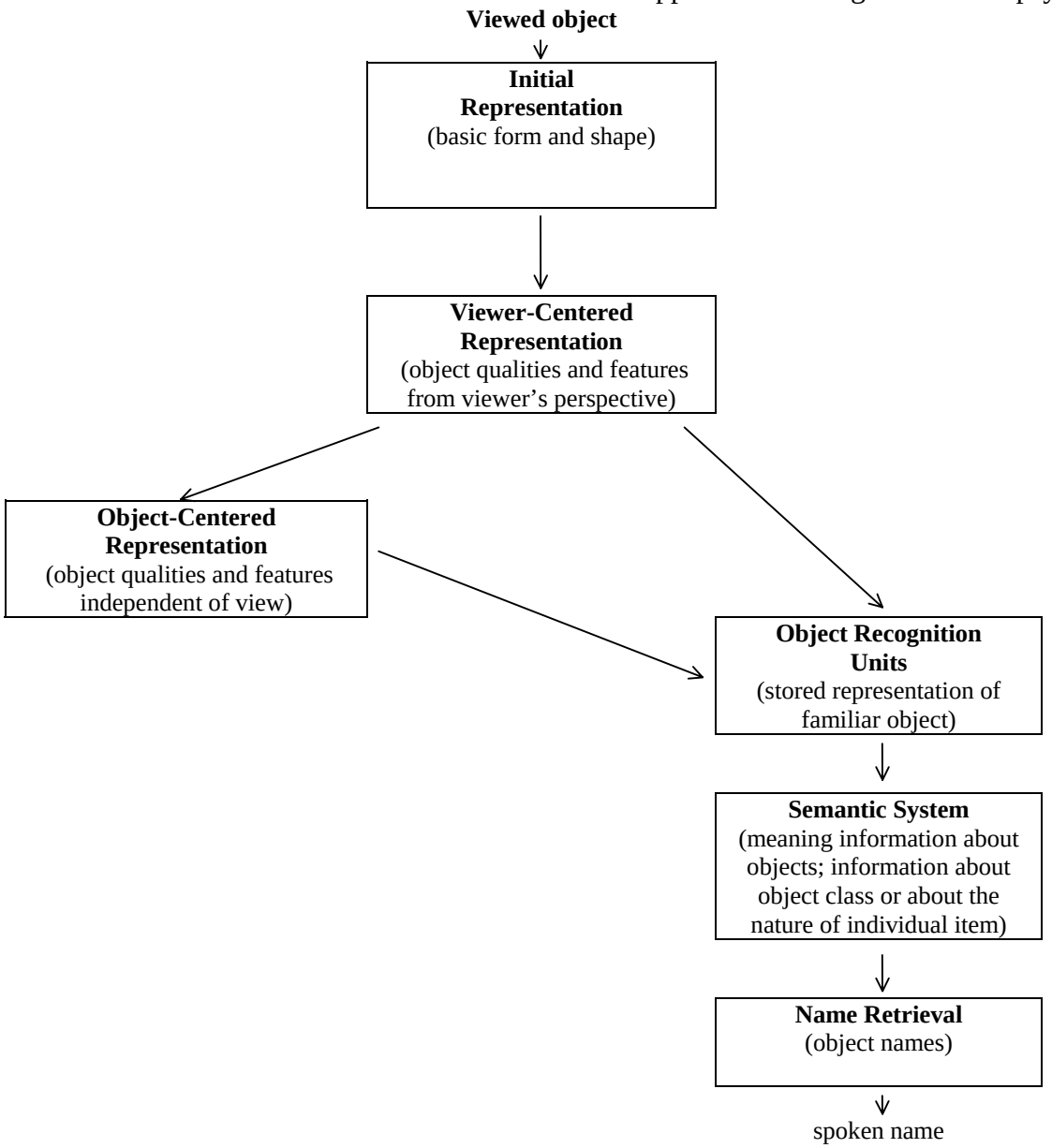


Figure 2
Clinical Application of Cognitive Neuropsychological Model



Assessment Notes for Each Level

Key issue: can subject appreciate basic object form and shape qualities? Object and shape discriminations (e.g., Warrington & James, 1991), Visual Closure (e.g., Street , 1931), Visual Form Discrimination (Benton Sivan, et al., 1994)

Key issue: can subject match identical objects, or discriminate between same and different items? Birmingham Object Recognition Battery (Riddoch & Humphreys, 1993), Benton Face Recognition, items 1-6 (Benton et al., 1994)

Key issue: can subject match objects presented in different views? Birmingham Object Recognition Battery (Riddoch & Humphreys, 1993); Benton Face Recognition, items 7-22; (Benton et al., 1994)

Key issue: is the item familiar or not? Birmingham Object Recognition Battery (Riddoch & Humphreys, 1993); familiarity discriminations; famous face recognition from Albert Remote Memory Battery (Albert et al., 1979 and subsequent modifications)

Key issue: can subject recognize the general class to which item belongs? Have subject group like objects together (e.g., tools, office items) on the basis of semantic similarity; Birmingham Object Recognition Battery (Riddoch & Humphreys, 1993)

Key issue: can subject derive the specific name for the presented item? Confrontation naming tests (Benton et al., 1989; Kaplan et al., 1983)