

International Encyclopedia of Rehabilitation

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Agnosia

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Abstract

Agnosia is a cognitive disturbance caused by neurological damage that affects associating knowledge with an intact percept within a single modality (vision, audition or somesthesia). Recently, neuroimaging research has led to a better understanding of how the brain processes perceptual information and lesion loci that contribute to the various agnosias. It is now thought that agnosia, in the classical sense, represents a top-down disruption in processing of perceptual information.

The late 1800s was a period when many neurologists were beginning to describe behavioral disturbances associated with brain injuries. At this point in medical history, little was known about the organization of the cerebral cortex. A prevailing view, connectionistic theory, posited that the brain processed in interconnected regions of specialization. However, many physicians of the time continued to reject the idea that specific brain regions were specialized for specific functions. Accordingly, the first description of agnosia was provided in 1890 by Lissauer. His initial description was of a single modality recognition deficit. He described two types of agnosia, apperceptive and associative agnosias, primarily distinguishing between disorders of perception and knowledge. This binary differentiation has persisted and is still used in neurobehavioral texts.

Lissauer stated that focal lesions and combinations of focal lesions could solely impair visual, auditory or somatosensory perception or recognition, leaving the other sensory modalities largely intact. The behavior displayed in a visual associative agnosia, for example, would be an inability to recognize a key from an array of objects, name a key from a picture or object display, or match a picture of one kind of key with another. Yet the individual would not have lost the ability to “know” what a key was and could demonstrate that knowledge through other modalities: i.e., he or she could name the key if permitted to feel it or respond appropriately to a question like, “What do you use to open a lock?”

In recent years, the development of sophisticated neuroimaging tools combined with careful cognitive-behavioral assessment has allowed revision of Lissauer’s model, an elucidation of top-down (feed-back) and bottom-up (feed-forward) processing systems, and an understanding of the complex ways in which processing networks can be disconnected (Mesulam) and disrupted (Mesulam, 2000; Damasio et al., 2000; Riddoch

and Humphreys, 2001). Although pure agnosias do exist, they are rare. Furthermore, careful diagnostic investigation often reveals idiosyncratic patterns of behavioral deficits that differ from one patient to another despite the initial appearance of a similarity (Riddoch and Humphreys, 2001). Prior to understanding the agnosias then, it is helpful to briefly describe the general cerebral organization of single modality processing. As a cautionary note, since neuroimaging technology is continuing to improve in its ability to provide a window into the human neurological processing, the description of human cerebral processing summarized in this chapter should be considered preliminary, at best.

Cerebral Organization of Visual, Somatosensory and Auditory Processing

The human brain is the organ of the body that allows us to adapt our internal body state to information and changes that occur in the environment (Mesulam, 2000). When we are hungry, the brain directs us to find food in the world around us. When we are tired, we seek sleep. To process information in the environment to meet our survival and personal needs, we need to process sensory information. This begins with a relay of visual, auditory, tactile and olfactory information from the sense organs (eye, ear, skin, nose) to primary sensory cells in the cortex (A1, V1 and S1 and the olfactory lobe respectively). Through early experience, the primary cortical cells become organized into a sensory map that represents salient stimulus qualities of the environment. For example, cells in the primary auditory cortex form a “tonotopic” map during the critical period of brain development that matches the range of frequencies an infant hears. These sensory maps are not specific to the human brain. In animals, exposure to a variety of animal calls results in an auditory map “tuned” to those calls while exposure to complex auditory signals like human speech result in a map that enables processing of speech stimuli (Chang and Merzenich, 2003). During early development those maps can be distorted by noise within the environment or nervous system or sensory deprivation. Although the sensory maps of the primary sensory regions are malleable after this early set up period, refining distorted maps requires attention to specific stimuli and timed reinforcement.

As the brain matures, perceptual refinement of environmental stimuli occurs by development. During human processing, the information encoded in the primary regions drive a feed forward organization of adjacent categorical perception. Like layers of an onion, perceptual organization of the brain develops based on the integrity and “fine tuning” of the sensory system (Mesulam, 2000). For example, the auditory regions adjacent to A1 in the left hemisphere process phonemic classifications of one’s native language, while the visual modality regions adjacent to V1 process color, form, and motion of visual stimuli (Mesulam, 2000; Damasio et al., 2000). Thereafter, sensory information is processed by more complex unimodal associations and ultimately, transmodal projections to other modality associations, allow integration of stimuli into multimodal concepts, like words which can be processed auditorily and visually.

The Effects of Lesions on Single Modality Processing

Modality specific deficits can result from lesions that directly disrupt the unimodal perceptual regions. Thus, a lesion in V4, which processes color perception, will cause

acquired color blindness, or a lesion in V5 (which processes perception of movement) will result in, an inability to perceive motion (Mesulam, 2000). In individuals with small, localized lesions, detailed assessment can demonstrate that these specific impairments can occur although there is intact visual perception. (See Riddoch and Humphreys, 2001 for a detailed summary of sensory and perceptual dissociations.) In this case, an individual with acquired color blindness, who is shown a chair, would be able to perceive the object's shape, size, orientation in space, but not the color. She could match the chair to other similar and different chairs without difficulty. With larger lesions to the visual association cortex, several attributes of a stimulus might be misperceived.

When lesions instead disconnect the sensory/perceptual regions from multimodal association areas, individuals can have more pervasive single modality "associative" disturbances. This is the classic agnosia and most contemporary neuroscientists reserve the term "agnosia" to refer to these types of associative deficits. When the lesions isolate a unimodal association area from other unimodal and heteromodal association areas, it results in an intact perception of the stimulus but inability to attach meaning to the perception. The individual is characteristically able describe details of the object presented to him yet unable to name what she sees, hears or feels. Likewise, because the perception is intact, the individual should be able make a detailed drawing of the object or match two detailed drawings of the same object. But what will often perplex an examiner is that after drawing or matching the stimulus, the individual will still be unable to tell the examiner what he has just drawn. The inability to associate past experience or knowledge to the stimulus is due to the disconnection from other modality input and conceptual processing (Mesulam, 2000; Kandel et al., 2000).

Types of Associative Agnosias

Associative agnosias result from lesions that isolate in tact visual, auditory or somatosensory percepts from heteromodal processing networks, (associations) (Mesulam, 2000; Riddoch and Humphreys, 2001; Kandel et al., 2000; Devinsky and D'Esposito, 2004). Since agnosias affect a single modality one can describe them in terms of the modality affected.

The Visual Agnosias

Adjacent and just anterior to the primary visual cortex in the posterior occipital lobe, is the visual association cortex. This region is still a unimodal visual region, and contains several specifically mapped association areas. V4, and perhaps V8, are specialized for color recognition. A unilateral lesion in V4 causes contralateral *hemi-achromatopsia*, loss of color perception. When a lesion spares V4 disconnects communication between V4 and the language cortex, the result is a color anomia - the inability to name colors despite intact color perception (Mesulam, 2000; Damasio et al., 2000).

V5 and the middle temporal gyrus are visual association areas specialized for perception of movement. Damage to this region causes inability to perceive visual motion with relatively preserved acuity and color perception, "*akinetopsia*" (Mesulam, 2000). Motions appear as still shots, akin to the experience of a stroboscope, the perception of series of visual objects in different positions (Riddoch and Humphreys, 2001).

A region in the fusiform cortex, called the fusiform facial area (FFA), is specialized for face and, to some extent, object recognition. When damaged the disturbance in facial recognition is termed *prosopagnosia*. The “face” area of the brain is more strongly activated by faces than objects, with no differential responses for familiar vs. novel faces and more activated by upright and intact faces rather than upside down or scrambled faces.

Global impairment of object recognition, *pure object agnosia*, commonly results from lesions that isolate the visual association cortex from other unimodal and heteromodal association areas or a combination of broad based lesions in visual association areas (Mesulam, 2000).

Contemporary neuroscientists identify two separate areas specialized for encoding words and word-like strings of letters –a region lateral to the “face” area and an area in lateral occipito-temporal region Mesulam (2000) has stated that it is likely words are handled as special forms of “objects.” *Pure alexia* sometimes referred to as *alexia without agraphia*, is a visual word recognition deficit that results from damage to or isolation of this region from other association areas.

The visual association pathways are often divided into ventral and dorsal pathways. Damage to dorsal visual areas or their connections cause visual spatial processing disorders but these are not considered agnosias. These include *hemispatial visual neglect*, *dressing apraxia*, the inability to align body axis with garment, *simultagnosia*, the inability to integrate visual detail into a coherent whole, *optic ataxia*, deficit in reaching toward a visual object, and *optic apraxia*, oculomotor exploration deficit. The former three may be seen as part of right hemisphere syndrome. The latter three are collectively known as Balint’s syndrome (Mesulam, 2000; Damasio et al., 2000; Devinsky and D’Esposito, 2004).

The Auditory agnosias

The primary auditory cortex (A1) is mapped for pitch and pure tone discrimination in all individuals who receive adequate auditory stimulation during early infancy. Bilateral damage to A1 has been termed central deafness, but usually results in problems with speech discrimination in noisy environments. The auditory association area adjacent to A1 in the left hemisphere responds to specific acoustic cues which allow for perception of phonetic parameters of spoken language. This region houses the following capacities, now often considered a prerequisite for phonemic awareness:

- Segmentation and sequencing of phonemes, perception of internal detail of syllable strings.
- Perception of polysyllabic and compound words.
- Presemantic encoding of phonemes.

Damage to these areas results in auditory perceptual impairments and cortical deafness. Disconnection of these areas from other association regions results in *pure word*

deafness, an agnosia characterized by the inability to repeat or understand spoken language despite good recognition of environmental sounds and no language deficit and *auditory agnosia* for environmental sounds. Often these agnosias have aperceptive and associative subtypes or components (Mesulam, 2000).

Phonagnosia results from damage to a right hemisphere auditory association area in humans that is analogous to facial perception in the visual cortex, and is the inability to recognize familiar voices despite preserved word recognition in tact ability to perceive environmental sounds (Mesulam, 2000; McCloskey, 2001).

The Somatosensory agnosias

S1 is the primary somatosensory (tactile perceptual) region of the parietal cortex. All primary sensory areas are mapped based on sensory experiences during early infancy. The mapping is similar to the homunculus mapping seen in the adjacent primary motor cortex in the left hemisphere. The adjacent association area S2 participates in pain perception and in some patients, lesions in S2 cause loss of pain perception. With additional damage to the insula and parietal operculum a painful contralateral disesthesia or “pain asymbolia” may result. The Somatosensory association areas Broadmann’s areas (Kandel et al., 2000 ; McCloskey, 2001) and perhaps also area 40 in the posterior insula are essential to:

- Localization of touch
- Exploration manually,
- Somatosensory guidance and coordination of reaching and grasping
- Somatosensory imagery

Lesions to this region or that disconnect this region from other association areas may result in:

- Problems with spatial orientation, tactile search, and ability to align the body axis with other solid objects during dressing, sitting in a chair or getting in bed.
- Hemispatial neglect
- Dressing apraxia

Other types of posterior parietal/insular lesions cause *tactile agnosia* (Mesulam, 2000).

Assessment of Agnosia

Assessment of agnosia necessitates determining that a disorder is limited to a single modality and does not affect other cognitive domains. It requires ruling out basic disorders of attention, sensation, impaired intelligence and aphasia (word recall problems, grammatical disorder (agrammatism or paragrammatism), phonological problems or verbal comprehension). There should be no evidence of disorientation, short or long-term memory limitations (Damasio et al., 2000).

Standardized testing may result in confusing findings, since standardized tests are usually designed to determine cognitive and linguistic deficits of neurological origin, not assess a single modality or contrast that modality with others using analogous tasks. For example, a patient with a pure object agnosia will be unable to point to pictures on command on a picture pointing task on a standardized aphasia examination, despite the ability to follow commands and answer yes-no questions. The same patient will be unable to name pictures or match pictures to words while responsive naming will be intact. If an agnosia is suspected or possible based on a lesion, it is preferable to determine two essential components of agnosia before conducting standardized tests.

First is to determine whether all deficits exhibited are restricted to a single modality and if there is no evidence of a sensory deficit. The first step will be to obtain a substantial verbal description of what the individual sees (visual agnosia), hears (auditory agnosias), or feels (somesthetic agnosias) when presented with objects, pictures (visual agnosias) or sounds (auditory agnosias). Often when asked to name objects or sounds through the modality in question the patient will either misidentify the items or respond “I don’t know.” However, when the same object or sound is presented through the other modality the patient has no problem with naming or identification. For example, if the patient is unable to name a pen when seen, but names it easily when held, this is a sign that the problem does not have anaphasia but rather a problem with a single modality. Similarly, if a patient is unable to identify an environmental sound like a cell phone ring, see if the patient’s ability is normalized when they are able to feel the vibration or see the phone light up. This type of dissociation task should be repeated with several stimuli to make sure that the pattern holds.

Second, the clinician should attempt to distinguish between misperception, “aperceptive agnosia” versus the classical “associative agnosia”. This can be done using copying and matching tasks. Individuals with aperceptive agnosias cannot match two identical visual stimuli (visual apperceptive agnosia), auditory stimuli (auditory apperceptive agnosia), or objects held in the hands (somatosensory apperceptive agnosia). The person will not be able to repeat a word or imitate a sound (auditory) or match an object to its drawing or copy it (visual agnosias). In contrast, persons with associative agnosias will perform the above without error yet will not be able to match different examples of the stimuli. For example, with visual agnosia a patient would not be able to match a pine tree to a maple, a green apple to a red one, or a cursive to a printed word. In the auditory modality, an individual would not be able to match two different horns blowing or dog barks, or even a girl and woman saying the same word. In the tactile modality the person might have trouble matching a large and small hand held glass, or spoon.

With an associative agnosia an individual will also be unable to name the object seen, heard or touched (depending on modality affected) but they will be able to name it through the unaffected modality (i.e. from touch or from verbal description). Finally, they will be unable to point the object or word (visual), identify the sound source representation (auditory) or find the object through manual exploration (somesthetic) to command (Mesulam, 2000; Riddoch and Humphreys, 2001; Kandel et al., 2000).

Prosopagnosia, the inability to recognize and name familiar human faces, may be assessed through color or black and white photographs of family members, as well as contemporary politicians and celebrities taken from the internet and/or print media (Van Lanker and Canter, 1982). The examiner should use a variety of photographs and ask the person to name one set., then, using a different set of photos ask the patient to point to each as the clinician names them. Then, all photo(s) which the patient is unable to name or identify by name, check for knowledge by asking the patient a question pertaining to the individual, e.g., Who is our U.S. President now? Who starred in _____movie?

Other disorders of face processing such as facial emotion recognition deficits can occur with or without prosopagnosia. Such disturbances can be caused by bilateral damage to the amygdala or disconnection of the amygdala from other cortical regions (Damasio et al., 2000).

Pure Alexia, (also called alexia without agraphia or pure word blindness), is a reading and letter naming deficit with intact ability to write. The patient will be able to copy and trace words, during which (because of the tactile cues) they may be able to recognize the letters and/or words. This is often due to lesions in the corpus callosum as well as the left visual association areas (Dehan, 2001).

Disorders of topographical orientation, sometimes called environmental agnosia, is the inability to locate a specific building in a city, find one's room in a home, or describe how to get to a specific location. The lesions are usually bilateral or in right hemisphere posterior regions. There are often also problems with route learning (Damasio et al., 2000). Environmental agnosia may occur with prosopagnosia and may also be seen in Parkinsonism. Epstein and Kanwisher (1988) have identified a parahippocampal region (PPA) that might be critical in recognizing the geometry of a local environment and some recent research suggests the hippocampus might also be important in some place memory (Nature Reviews Neuroscience, 3.08).

Disorders of color perception that result from brain injury are called central achromatopsias. They are commonly caused by focal damage to the visual unimodal association cortex and involve all or part of the visual field. The color loss can be complete or partial. In individuals with achromatopsia form perception is usually preserved, although there may be an accompanying object agnosia and prosopagnosia (Mesulam, 2000).

Simultagnosia is the inability to grasp the global view, focusing on one detail of a composition, then another. The patient with simultagnosia will attend to and describe details in the picture but often be unable to give a description of the scene as a whole. The best way to identify it clinically is through picture description. A room scene may be described one piece of furniture at a time; rarely would the patient name the room. Because the patient has trouble attending to the gestalt, if the object pictured is a face, the individual will perceive the eyes and the nose, but fail to mention the face. However, if the individual is viewing a human figure, they may perceive the head, the arms and the

legs. Patients may complain that objects, or portions of objects may appear to move around. A patient may say the words or objects appear “jumpy” (Mesulam, 2000).

Pure word deafness is the inability to understand the spoken word or repeat words or sentences with preserved ability to read words. Sometimes it is described as “Wernicke’s aphasia” without the aphasia. The lesions can be bilateral affecting Heschl’s gyrus and its association tracts or a corpus callosum lesion occurring in conjunction with a lesion in the left auditory association region (Dehan, 2001). The individual will not present as aphasic; they will not make grammatical or phonological errors in speaking and will not present with anomia. With pure word deafness an individual will understand and respond to prosodic contour and can recognize familiar voices (Mesulam, 2000).

Phonagnosia, first described in depth by VanLanker and Canter in 1982 (McCloskey, 2001), is the inability to recognize familiar voices. It is characterized by inability to recognize familiar voices and, thus, considered the auditory analogue of prosopagnosia. It may occur with pure sound agnosia, but it is rarely identified clinically because it is only a problem with recognition of familiar voices without a visual image, so it basically affects recognizing friends or well known persons by phone or on a radio.

Treatment

In acute stages the adult with agnosia may be intolerant of the therapeutic process because they are unaware of the problem. If there is additional involvement of the limbic system connections, often occurring with posterior cerebral lesions or with very deep lesions, this may decrease motivation to improve even if aware of the problems. Before attempts at remediation the clinician will need to increase recognition of deficits. Since agnosia is restricted to one modality, one way to build awareness of the deficit is to alternate presenting a stimulus in the impaired modality, then through the unimpaired modality. Over several repetitions the patient may gradually become aware of the difficulties experienced in the impaired modality. It might also be helpful to break down tasks into very small steps so that the patient can see each component of their problem.

Once the patient is aware of his or her difficulties, treatment goals should focus on developing compensatory treatment approaches that quickly enable the individual to function independently in their living environment.

Goals for increasing independence

Use of alternate cues

It is helpful to assist a patient in bypassing his or her perceptual limitations and learning to use alternate cues to improve recognition. Adults with prosopagnosia can use distinctive facial characteristics such as hair style, color or length or unique facial features. The patient can also learn to use contextual information or information that is revealed during a conversation, just as most of us do when we have trouble placing the person or thinking of a name.

Use alternate modalities

Patients may discover alternate modality compensatory strategies themselves. Often patients with prosopagnosia quickly learn that they can recognize a person as soon as they speak. In the same way, with pure word deafness, asking a person to write down what they are saying will help them to understand a verbal direction or communication. A person with pure word deafness can also learn to use shared contextual cues, intonation, gestures and facial expression to aid their comprehension. With pure word deafness, some patients report benefit from learning to use lip-reading to aid comprehension.

Patients with pure alexia can learn to read fairly well through letter tracing. At first patients may trace the actual print letters, but often in a short period of time they begin tracing letters on their hand. In time, simply tracing the first letter of a word may trigger recognition, just as phonemic cueing can trigger naming in many Broca's aphasics.

Teaching the patient with visual object agnosia to feel an object to assist recognition and allowing the patient to practice using combined tactile and visual modalities to recognize objects in the environment will often be beneficial.

Patients with pure word blindness can be taught to trace letters to aid recognition of words.

Verbal strategies

Patients with prosopagnosia may learn to listen to a person's description of friends or events to aid in identification. Similarly, some patients with visual agnosias may use verbal descriptions to assist recognition. For example, as a person with problems recognizing whole objects describes details about an object or a face they may determine from their own verbalization what they are viewing. For obvious reasons, verbal strategies are not useful in auditory agnosias.

Alternate cueing devices

Color coding or large tactile or printed markers may aid patients with environmental agnosias or pure object agnosias. A piece of bright colored Velcro, sandpaper, or cloth that the patient is taught to associate with danger can be attached to a dial or touch pad of an oven or stove. Similarly tactile markers can be used warn a patient about impending danger on staircases or entryways to rooms that contain dangerous equipment. Placing child protection gates to block these areas or closing off the area with locked doors or partitions will provide even greater safety.

Organizational strategies

Objects that are used together can be stored in clearly labeled drawers, cabinets or storage bins. Organization of clothing will provide security and help patients make dressing choices more confidently. A closet that is organized so that garments that go together are placed next to each other or in the same section. Prepared foods can also be organized so that the patient knows where fruit, canned foods, bread, and other foods are always found. Most patients will be good at identifying fresh foods by touch and smell, but for

that reason keeping fresh foods available in consistent places will be very helpful for a patient who would like to fix his own cereal in the morning or make a sandwich at lunch. By pre-preparing drinks in non-spillable containers, the most adults with aphasia can eat healthy cold meals during the day without assistance.

Avocational goals

Quality of life depends on enabling adults with disabilities to participate in avocational interests, so it is essential that these features are built into any rehabilitation program from adults with agnosia. Audio files of books can be obtained at any library, and today there are many internet resources that provide audio streaming of newspaper and magazine articles as well. The Lighthouse for the Blind and Recording for the Blind and Dyslexic are resources for recordings of books and periodicals that are not commercially available. Adults with pure word deafness may enjoy movies with subtitles or closed-captioned TV.

Commercially Available Rehabilitation Programs

For milder patients who recognize their deficits and can tolerate the therapeutic process, treatment geared toward restoration might be feasible. There are many technological companies that now specialize in computerized reading programs. *Reading Assistant*, a product produced by Scientific Learning Corporation is an assisted reading program that allows an adult to read a passage aloud with voice verification software that corrects misread words. Dronkers et al. (1999) reported excellent improvement with receptive language deficits associated with aphasia after use of *Fast ForWord Language*. Battin et al. (2000) reported that the same intervention was useful for other types of auditory processing disorders in adults; however, these programs have not been specifically validated for use with agnosias.

Safety concerns

A concern with any neurologically impaired adult is the possibility that they may become separated from a caretaker at an inopportune moment or wander away from the home or a family unaccompanied. Preparing a patient for an outing by telling them before hand where they will be going and discussing situations they might expect to encounter may help to prevent unwanted confusion or misunderstanding. It is also important to avoid situations where the individual might be left alone in an unfamiliar setting for any period of time; individuals can take turns remaining in the presence of a person with agnosia when in a strange environment, providing an ongoing description of the events and activities going on around them. But even with adequate preparation and supervision, adults with visual agnosia should, always carry a clearly visible identification card that states that they have suffered a brain injury and may appear confused or disoriented when separated from family and friends. Contact information and other valuable information for seeking help should be included. Patients may also wear an electronic locator device or carry an emergency use cell phone so that family members can page or call that is labeled with information a stranger can use for calling a family member.

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