accident or disease. For example, a brain-injured patient suffering paralysis or aphasia following a stroke may, like any other patient, experience anger at the seeming unfairness of events. When the anger is misdirected at health care providers, not to mention friends and family, treatment may be disrupted and recovery prolonged. Nevertheless, the anger is understandable, even if unjustified, as the patient comes to terms with a painful event that makes little rational sense.

Such “normal” if misdirected anger must be distinguished from that which results more or less directly from injury to parts of the brain related to aggression. For instance, an injury that results in stimulation of the amygdala may induce an aggressive response that is interpreted post hoc by the patient as anger. As humans, we like to think that our actions are meaningful. When, for extraneous reasons (such as brain injury), we respond in ways that make no sense, we nevertheless impose meaning on the response. Interpreting a brain injury-induced aggressive response as anger is one such meaning-making device. (Recall the earlier discussion of the generic and specific senses of anger. As noted, anger is often used generically to refer to almost any aggressive response.)

One distinguishing feature between normal (albeit misguided) anger following injury and brain injury-induced anger is that the former is typically manifested soon after the injury’s occurrence, and it abates as the patient adjusts to life following the injury. However, differential diagnosis is difficult. Once an aggressive response is interpreted as anger, it is in a sense “normalized”; that is, it is made to conform to the beliefs and rules that help guide normal anger. The underlying condition that produced the aggression may thus be masked.

In short, the assessment and management of anger and aggression in neurologic rehabilitation requires careful exploration of the patient’s entire repertoire of behavior and the instigating factors. It is easy to be misled by focusing uncritically on a patient’s claim that he or she was simply acting out of anger.

The most efficacious treatment of anger and aggression following brain injury requires an individualized rehabilitation program that incorporates an array of neurologic, behavioral, and social therapies. In the same way that anger cannot be localized to any specific neural structure, the most effective anger management program will not rely exclusively on any one form of treatment.

See Also the Following Articles
AGGRESSION • COGNITIVE REHABILITATION • EMOTION • STROKE • VIOLENCE AND THE BRAIN

Suggested Reading

GLOSSARY
aphasia A disorder of language produced by damage to the language zone, usually in the left hemisphere of the brain. Aphasia is not due to the inability to articulate language but is caused by damage to language representations or mechanisms.
circumlocution When a target word cannot be retrieved, a multidimensional response describing the characteristics of the object it names. For example, “you write with it” for “pencil.”
confabulation naming A task used to evaluate a person’s ability to spontaneously name an object or an action; either a picture or an actual object is presented to the person for him or her to name, or a picture, paragraph, or video enactment of an action is presented to the person to elicit its name.
literal (or phonemic) paraphasia Sound substitutions or additions that result in either an unintended real-word utterance or a nonsensical utterance. For example, with the word target flower, the utterances live, drive, or flower would all be classified as literal paraphasias.
perseveration The unintended repetition of an idea, verbal utterance, or motor output.
verbal (semantic) paraphasia Substitution of a real word for a verbal target; if the substituted word is semantically related to a verbal target, such as log for the intended target tree, this would be considered a semantic paraphasia.

Anomia
LISA TABOR CONNOR*+ and LORRAINE K. OBLER§,‡

*Washington University School of Medicine; 1City University of New York Graduate School and University Center, and
§Boston University School of Medicine.

Anomia is a profound difficulty in coming up with words in the course of discourse and/or on a naming task, particularly those words that are heavily meaning-laden. This definition presumes both that a word being sought was originally known by the speaker and that the difficulty in retrieval is not due to failed articulation. Anomia is a hallmark of aphasic syndromes and occurs in aphasic persons with fluent, running speech and in aphasic persons with nonfluent, halting speech. That is, all aphasic individuals have word-finding difficulties to some degree. Such difficulties retrieving words are also characteristic of Alzheimer’s dementia and, to some extent, of normal aging. This review focuses on the three populations in which anomia, sometimes called dysnomia, is prevalent: persons with aphasia, persons with Alzheimer’s disease, and healthy older adults. A review follows of category-, modality-, and grammatical-class-specific anomias and of how these fractionations of word-finding into subtypes inform our understanding of the neuroanatomy of naming. Rehabilitation for anomia and recovery from it are discussed. Finally, a cognitive framework for naming is proposed to provide a means for discussing features of word-finding impairment.

I. ANOMIA IN APHASIA

As mentioned in the article on aphasia (this volume), it is a set of disorders of language. In adults, the disorders are typically caused by stroke in the left hemisphere of the brain in the perisylvian region. The perisylvian region is bordered by the third convolution of the frontal lobe and the angular gyrus of the parietal lobe.

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by the supramarginal gyrus, and by the superior border of the inferior temporal gyrus. Nearly all language deficits are produced by lesions inside the perisylvian region, and lesions outside of this region rarely produce language deficits, at least in right-handed individuals. Both areas traditionally associated with being the aphasia syndrome. Broca's area in the frontal lobe and Wernicke's area in the temporal lobe, lie inside the perisylvian region.

Aphasic individuals with lesions inside this "language zone" exhibit different characteristics depending upon the location and extent of the lesion. Language deficits may be characterized by severe limitations of verbal and or written production with relatively well-preserved comprehension (as in Broca's aphasia) or by severe limitations of auditory comprehension with relatively preserved verbal production (as in Wernicke's aphasia). Many other patterns of impairment and preservation of language faculties are possible, and particular combinations are characteristic of other aphasia syndromes. Regardless of whether the person with aphasia exhibits nonfluent speech or fluent speech, however, a common deficit is evident, that is, a difficulty accessing words, anomia. In fact, anomia was recognized in the late nineteenth and early twentieth centuries as a crucial component of aphasia by early aphasiologists such as Broca, Wernicke, Fried and Pals, Head, and Goldstein. Anomia may be a central feature of the aphasic picture or more peripheral to other features of language dysfunction. More specific to aphasic syndromes, however, most aphasia assessments begin with examination of the individual's ability to name in conversational contexts and to name an object or drawing of an object to confrontation.

Naming is first assessed by eliciting a sample of speech. The speech sample may be obtained by asking the patient to respond to the examiner's questions like, "What brought you here today?" or by showing a picture of a visual scene and asking the patient for a verbal description of it. An approach through a picture of the description task are specific targets for naming are present, providing the patient with more structure, and that there are no standards for quantifying the patient's impairment in naming. One picture description task commonly used in the assessment of aphasia is the Cookie Theft Picture Description from the Boston Naming Test. Naming to confrontation is the next step in assessing aphasia. The manner in which confrontation naming ability is tested is quite straightforward. Either an object or a picture or drawing of an object is presented and the patient is asked to say its name. Likewise, an action may be depicted, the examiner can pantomime an action, or a video clip may be shown portraying an action and the patient is asked to say what is happening. A commonly used test of confrontation naming of objects is the Boston Naming Test. To examine the nature of retrieval failures in confrontation naming, examiners probe the patient's accessibility to other information. For example, they may ask "Do you know what this is? Is there another word for that? What is the woman doing?" If the patient is still unable to respond, cues may be given that are related to the meaning of the pictured object. A meaning-based cue, called a semantic cue, is often given if it is unclear that the patient perceives the picture accurately. For example, if shown a picture of a beaver, a cue may be either general in nature, such as "It is an animal," or specific in nature, such as "It builds a dam." If the patient is still unable to respond correctly or if it is clear from the patient's comments that the picture is perceived correctly, a sound-based cue, called a phonemic cue, may be given to aid the patient in producing the correct target. Typically, a phonemic cue consists of a small fragment of the target, such as the initial sound and initial vowel. For instance, if the target is beaver, the cue "bee" would be spoken. In some instances, latencies to produce a picture name may be measured, such as the time it takes to comprehend the initiation or naming difficulties with particular categories of words. The significance of responding to cued and uncued naming deficits in naming will be discussed later in this article.

The characteristics of anomia differ among aphasia classifications. As illustrative examples, consider the features of anomia in Broca's aphasia, Wernicke's aphasia, and atomic aphasia. Anomia plays a dramatic role in Broca's aphasia. In patients with Broca's aphasia, patients with Broca's aphasia have difficulty initiating speech, but once the fluent, agrammatic speech with poor prosody and phrase length, and have particular difficulty producing the "small" words of the language, such as articles and prepositions, as compared to substantive words. Their speech is hesitant and labored, with only critical items related to the meaning of the message being produced, such as nouns and some verbs. Their total output is severely reduced. During confrontation naming, the naming performance of Broca's aphasia may be severely impaired or normal difficulty encountered. Further, the Broca's aphasia may say the name of a previously produced picture even when a name is presented (a phenomenon called perseveration) or may produce a word that is semantically related to the target, such as leaf for the target tree. This latter type of substitution is called a verbal or semantic paraphasia.

Patients with Wernicke's aphasia, by contrast, have "fluent," but labored, fairly grammatical, speech with generally preserved prosody and phrase length. They often have difficulty with confrontation naming tasks, producing fluent aphasia and word sound substitutions called literal or phonemic paraphasias. It is often quite difficult to point to specific instances of anomia in the speech of a Wernicke's aphasic because there are no pauses to search for words and the intended meaning of the utterances in free conversational tension is often unclear. Wernicke's aphasic with severe impairment may produce neologisms, that is, nonsense words that have been hypothesized to reflect word-finding difficulties. A common feature of running speech in milder Wernicke's aphasia, like that of other fluent aphasia such as conduction, atomic, or transcortical sensory aphasia, is circumlocution, the verbal description of a target that the patient is unable to retrieve. In addition, Wernicke's aphasics fill their discourse with words "empty," of content (e.g., thing, something, do), again, probably reflecting difficulty in accessing the substantive nouns and verbs they intend. Severe Wernicke's aphasics are unaware that their speech does not make sense to the listener and may be frustrated that the listener is not comprehending or responding to the initiation or naming difficulties with particular categories of words. The significance of responding to cued and uncued naming deficits in naming will be discussed later in this article.

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representation in the brain; however, we are still quite a long way from understanding them.

**B. Grammatical-Class-Specific Anomia**

The early grammatical-class dissociation observed in the anomias was that between content-laden ("substantive") words and functions, those "small words" like "it," "is," and "who" that convey primarily syntactic information. Patients with a type of aphasia called agrammatism, of course, produce predominately substantive and few functions. However, agrammatism itself traditionally has not been considered primarily an anomia problem. Rather, problems with retrieving substantives (nouns, verbs, adjectives, and, perhaps, adverbs) in running speech or on confrontation naming constitute anomia.

Like the category-specific anomias, grammatical-class dissociations in naming have been reported that include noun-verb dissociations and proper-name/retieval failures. Unlike semantic-category dissociations, however, noun-verb retrieval dissociations are more tightly coupled with particular lesion territories. That is, individuals with selective impairment of object naming have left posterior language-zone lesions of the temporal cortex, whereas individuals with selective impairment of action naming have left anterior language zone lesions of the supramarginal cortex. The controversy in the domain of noun-verb naming dissociations lies in what these lesions represent in terms of the language system. If the anterior area is associated with verb problems and the posterior area is associated with noun problems, how can we account for the category-specific problems discussed earlier? One possibility is that they arise from smaller lesions than the one that results in general problems with nouns. A second possible explanation is that both the representations for semantics and phonology remain intact for nouns as well as for other substantives but that there is a disconnection between specific semantic centers and the store of the phonological shapes of names. The latter theory assumes that the distribution of semantic and phonological representations for nouns is separate from that for verbs, nouns being distributed throughout the temporal lobe and verbs being distributed throughout the frontal lobe.

In addition to noun-verb dissociations, evidence for speech deficits in proper-name retrieval has been reported primarily by Semenza and colleagues. Leisons of the left temporal pole are associated with this deficit. In individuals with proper-name impairments, common nouns are largely preserved. Even names that would have been well-known before the aphasia are markedly more difficult to access. Indeed, some patients have been reported to have a dissociation between their ability to retrieve proper names for people and that for geographic locations and landmarks. The explanation offered for such dissociations has been that proper names, especially those for people, have limited associations to other items in the lexicon. For example, the occupation baker has associations to other occupations and to what bakers do and produce, whereas the last name Baker refers more arbitrarily to the individual or family one happens to know with that last name. For geographical locations and landmarks, the name may be associated more strongly with other information about that place, for example, one may associate the Eiffel Tower with everything one knows about Paris and France.

**C. Modality-Specific Anomia**

Whereas the term anomia has classically been associated with problems locating the names of things presented visually, there is a substantial set of cases that have been reported in the literature in which modality-specific factors are evident in accessing or producing substantives. Among the different modalities of presenting the target words are those of touch, taste, smell, and hearing (e.g., the sound of a bell ringing to elicit the noun "bell"). Optic aphasia, for example, nicely demonstrates that naming impairments may be specific to a particular sensory modality. In optic aphasia, the person is unable to name an object presented visually but has minimal difficulty naming that same object presented through another modality. A picture of an apple may be met with naming impairment, but once the person has touched or smelled an apple the name is readily retrieved. As is the case with grammatical-class-specific anomia, a particular lesion along the pathway either from visual association areas and the semantic representations for objects or from visual association areas and motor output areas for speech is posited to explain the phenomenon. In addition, patients have been reported for whom it is not the modality of input that results in a dissociation but, rather, the modality of output. For example, an individual may be markedly more anomic when asked to speak the name of a target than when asked to write it on a piece of paper.

Cases of modality-specific anomia have lead to theorizing about the extent to which there is one underlying lexicosemantic system for all modalities. Instances with dissociations among modalities suggest, rather that there may be multiple systems of input in order to get to the phonological (or orthographic) shapes of words and for outputting them via speech or writing.

**III. ANOMIA IN ALZHEIMER'S DISEASE**

Alzheimer's disease (AD) is the most studied of the dementia-producing diseases that can have anomia as a salient symptom. Dementia (see the article on it in this volume) may be defined as a progressive cognitive decline resulting from a number of diseases. Such a cognitive decline characteristically may include more language disturbance (as in Alzheimer's disease in all but very late-onset instances) or less language disturbance (as in the dementia associated with perhaps one-third of the individuals with Parkinson's disease). When a language disturbance is evident, anomia is invariably a part of it. The naming problems associated with the dementias may or may not be termed anomia by different scholars; however, the phenomenon is quite similar to that found in the aphasias. What underlies the problem, however, appears to be different. First, the cognitive problems underlying the dementia regularly include a variety of memory problems, so one may argue that the difficulty with outputting a name results from the memory problem rather than from a more strictly linguistic problem. At the anatomical level, moreover, the dementias are associated with sizable, isolable lesions such as those of the aphasias but rather with multiple lesions at the cellular level, thus suggesting that it is a systemic problem rather than an area containing to some more or less specific aspect of naming that is impaired when anomia manifests itself.

Lacking the cognitive/decline crucial for a diagnosis of dementia is a marked component of semantic impairment evident in the dementias, most particularly in AD. The term "semantic" here is meant to be distinct from the term "phonological" (or "orthographic") in that the borders of meaning of a word seem to become more permeable, or less specific information about the meaning of the word is available. This can be well-demonstrated by a task such as one to probe the semantic attributes of a word. For example, when a patient cannot remember the name of a picture (e.g., a squirrel, a robin), one might ask about the information (e.g., is it a tree? an animal?). Coordinated information (e.g., is it a dolphin? a robot?), and subordinate information (e.g., does it eat fish? does it live in a cool climate?). Whereas people with anomia resulting from the aphasias can access all three types of information without difficulty, anomias with AD can, at one point in their cognitive decline, access only the superordinate information and later may even have problems with that. Such semantic problems, however, act in conjunction with more purely lexical-access problems. Those may be more severe than those associated with normal age-related problems that the patient would be expected to evidence.

One of the ways one tests for the problem lying at the retrieval stage, as mentioned earlier, is by giving phonemic cues. For patients with AD, these may not be helpful as they are for normal elderly individuals with naming problems. Indeed, patients with AD may appear to "free associate" to the phonological cue: for a picture of a trolley, one says "..." and the patient may respond "trolley." Of course such a response does not positively assure that the problem lies at the lexical level; rather, it may be due to inattention to the task. An alternate indication is to look at the consistency with which the patient can name an item over time. If the patient cannot name the trolley one day but can the next, this argues that the item's representation itself is not impaired, but rather retrieval of it is. An additional factor that enters into the naming errors of patients with AD is perceptual difficulty. That is, pictures may draw inappropriate answers ("cucumber" for "escalator") as the patient is drawn to the overall shape or to a subcomponent of the picture.

**IV. ANOMIA IN NORMAL AGING**

Whereas there are clear-cut naming problems associated with old age, the term anomia may be even less appropriate to describe them than the naming problems of Alzheimer's disease, perhaps because they are so much more subtle than the naming problems of aphasia. Indeed the term "dysnomia" (here referring to a transient naming problem rather than a permanent disorder) may more accurately portray the naming problems of aging in that it implies a more fleeting problem rather than an enduring impairment. Phenomenologically, older adults report word-finding
difficulties as they age (including more frequent tip-of-the-tongue experiences), and, in fact, laboratory-based studies have confirmed this report. In particular, older adults find the retrieval of proper names increasingly difficult. Whereas the literature confirms that proper nouns are particularly difficult with advanced age, difficulties with nouns and verbs are evidence as well. There are some reports in the literature that noun retrieval is disproportionately impaired relative to verb retrieval with age, but more recent evidence suggests that, in carefully matched sets of nouns and verbs, the retrieval deficit associated with age, which becomes significant around age 70 years, is equivalent for both types of substantives.

Cross-sectional studies as a rule demonstrate significant naming problems for groups of older adults starting in the decade of the 70s. Longitudinal studies point to subtle declines in naming as early as the 40s. With normal aging, however, there is substantial variability across individuals within any given age cohort; some 80-year-olds perform like some 30-year-olds in a naming task. Education does seem to have long-term protective benefits in this regard. Results from the ability of older adults to rapidly choose a correct name on a multiple-choice task for items that they were unable to spontaneously retrieve, as well as being able to retrieve the correct name for an object once given a phonemic cue, suggest that the deficit in lexical retrieval for older adults lies in accessing the phonological shape of the target word. However, there is a suggestion that subtle semantic degradation may be involved as well from research examining the consistency of naming a given item over a series of test sessions.

Theoretical accounts for dementia in aging have centered around the well-described phenomenon of cognitive slowing in particular. Burke and her colleagues have put forward an account of age-related naming deficits that combines cognitive slowing with the clear-cut deficits in accessing the phonological shape of a target word. Their model suggests that aging produces slowing, or "transmission deficits," across the board in the semantic and phonological networks that underlie lexical retrieval. The phonological system is relatively more affected, however, because as activation spreads throughout the network, semantic and phonological representations in particular converge on the appropriate lexical node, but the activation must then diverge from the lexical node to the many phonological nodes that constitute the word. Because general slowing is at play in aging, the amount of activation that has accrued at the lexical node is less for older adults than for younger adults and, thus, when the activation spreads to the phonological nodes, it expires before crossing the threshold for word production.

V. TREATMENTS AVAILABLE FOR ANOMIA

Naming abilities in aphasia recover over time. Indeed, it appears that there may be slow progressive recovery in the ability to name objects and actions over time even years after the aphasia-producing incident and long after treatment has ended. However, some aphasic who have, according to all aphasia tests, fully recovered from their aphasia, nevertheless report feeling that they cannot always find the words they need in conversation. Despite the apparent spontaneous recovery of naming abilities over time, treating them is quite difficult. Attempts at drilling items, which work well for second-language acquisition in some normal individuals, have no long-term effects for aphasic patients. More successful, by contrast, are efforts to affect the processes that interfere with naming. For example, perseveration of a previous verbal response may prevent access to an otherwise intact name. Therefore, treatments that focus on reducing perseveration may be helpful in "deblocking" verbal naming. One such strategy developed by Helm-Estabrooks and colleagues involved bringing perseveration to the awareness of aphasic individuals and teaching them to actively suppress the item that they were about to incorrectly produce and to then ask the therapist for a cue as to the correct name for the target. This treatment is not, however, a treatment for naming per se but rather an means to prevent an incorrect response from intruding upon the production of the correct name.

Another deblocking technique for severely impaired aphasic is the Voluntary Control of Involuntary Utterances program also developed by Helm-Estabrooks and colleagues. This method is appropriate for use in individuals who have quite restricted verbal ability, perhaps limited to a few stereotypical responses but whose productions are inappropriate to the context. This type of off-target responding occurs only in cases of severe verbal production deficit. An example of this would be the patient who says "cat" when shown a picture of a dog. The fact that the patient clearly has access to the name "cat" indicates that it is a candidate for remapping to its appropriate semantic representation. The idea underlying this program is that speech that is generated automatically and without voluntary control can be retrained to be uttered voluntarily in an appropriate context. The clinician starts with a very small vocabulary of items, perhaps as few as five or six, for which the patient can successfully read the name of an object presented with its picture (e.g., the patient says "cat" to a picture of a cat) and expands the functional vocabulary over time by first introducing emotionally salient words and expanding outward from there to include new items. This technique tends to deblock other vocabulary, enabling the clinician eventually to expand functional naming to as many as a few hundred items.

Techniques have also been put forward to enable a less severely impaired patient to use partial utterances to cue a correct response. Often it is the case that, once a phonemic cue is given, an aphasic patient will be able to use that cue to generate the remainder of the word. Because this technique is only appropriate for a particular target, the goal is to get the patient to apply a systematic approach to retrieving the appropriate cue that will "bootstrap" the target response. An alphabetic search strategy is one means by which to generate the appropriate cue (and many aphasic have spared ability to recite fixed sequences like the alphabet), or manually generating semantic associations to the target may lead to the recovery of the initial phonemes.

VI. COGNITIVE FRAMEWORK FOR NAMING

In order to arrive at a cognitive framework for naming that accounts for a majority of the available data from aphasia, aging, and Alzheimer's disease, a basic three-stage model of naming like that of Levelt consisting of a visual object-recognition stage, an access-to-semantic-information stage, and finally a phonological-realization stage needs to be expanded considerably. First, a lexical level needs to be inserted between the semantic and phonological levels to satisfy constraints of mapping distributed semantic and phonological representations onto one another. In addition, due to modality-specific deficits such as those found in optic aphasia, it is clear that a single semantic system blind to input modulation will not suffice. Therefore, it is necessary to propose two levels of semantic analysis: one dealing with modality-specific features of an object and a second dealing with supramodal features such as an object's function. Moreover, on the basis of data from normal aging, it is necessary to include in a model of confrontation naming a means to describe the process by which the information is passed from one level of representation to another and how that information could be disrupted or affected by changes in the processing abilities of the individual. Finally, information about semantic-category dissociations and the degradation of semantic knowledge with Alzheimer's disease requires that there be a hierarchial representation of semantic attributes. Therefore, what initially appeared to be a simple and straightforward three-stage model of naming is complicated by what we know about how the components and processes of naming are affected by aging, disease, and neurological accident. Even with all of the additions to and modifications of the simpler model, many behavioral phenomena associated with anoma, as well as their neurological underpinnings, are as yet unexplained.

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