16. Inference Failure: The Underlying Impairment in Right-Hemisphere Communication Disorders

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The effects of right-hemisphere damage (RHD) on communication have been explored at the Clinical Aphasiology Conference for approximately 14 years. In 1976 Michael Collins noted that the speech of right-hemisphere-damaged patients was "bizarre and inappropriate" (Collins, 1976). Fourteen years later, their speech is still bizarre and inappropriate, but we are better able to describe it. In fact, almost all of our research efforts in the area of right-hemisphere (RH) communication disorders have been aimed at description. We have learned that for the most part RH-damaged patients are linguistically sound. We know that their deficits most typically occur in complex interactions in which meaning is dependent upon context. We have categorized their communication problems into two broad areas: prosodic and pragmatic or extralinguistic. We also have looked to various perceptual and attentional mechanisms as the possible roots of these problems.

After a number of years of research in an area, it is good to assess where we are going, and to reassess some of the assumptions we have made. We have specified and described, but a complete description of the symptoms of a disorder can only take us so far. A full understanding of the deficits will only come from developing, testing, and refining a model of the disorder. As yet, we have no well-developed model of RHD deficits to test. As a result, there are no standardized, theory-driven tests available for evaluating the described deficits, and our treatment remains at the level of managing symptoms rather than their underlying cause. In fact, the label we use for these disorders refers to anatomy, rather than to a cognitive deficit.

I would like to offer four hypotheses by way of addressing these issues. First, I would like to propose that RHD patients have difficulty with *inference*. This is nothing new. Second, I would like to suggest that gener-

ating inferences is not an activity restricted to the late stages of cognitive processing, but that it also operates very early in the system. Third, I would like to suggest that RHD can interfere with inference generation in this early stage as well as in later stages of processing. Finally, I would like to propose that inference failure may represent a "central" deficit underlying most, if not all, RHD communication disorders.

Inference has been defined as "the act of passing from one proposition, statement, or judgment considered as true to another whose truth is believed to follow from that of the former" (*Webster's New Collegiate Dictionary,* 1977). This process of passing from one proposition to another on the basis of belief gives us the ability to interpret intended meaning. It allows us to step beyond the face value of immediate sensory input and arrive at a meaning that is more than the sum of the given parts.

How does inference work? It requires an interaction between two types of recognition—the recognition of key elements and the recognition of their relationship to one another and to other contextual cues. It is difficult to know which comes first, the elements or their relationship. In either case, it is the relationship that creates an extension of the individual semantic content.

Inferred meaning comes from the recognition that mere sensation can represent more than it appears to. It is not based on fact so much as on *belief.* You see what you see or hear what you hear, and you combine that sensory input into a second proposition that you *believe* to be true based on the first, and so on into further propositions.

RHD COMMUNICATION DISORDERS AS INFERENCE FAILURES

How many RHD communication disorders can be considered failures of inference? Possibly all of them. What follows are examples of the communication deficits we typically associate with RHD looked at in the light of inference failure. As one looks down the list, it is important to recognize that the term *failure* does not necessarily refer to an absence of inference, but rather to faulty inference. There may be points at which the system fails to generate inferences or generates the wrong inference. Those points may vary with the nature of the input, the nature of the inference, the degree to which the system is stressed, and so on, just as "failure" to retrieve a word is reflected in a variety of behaviors in aphasia.

RH-damaged patients tend to gravitate toward the *literal meaning of metaphor, humor, idioms, indirect requests, and general conversation.* That is, they do not appear to process the contextual or situational cues that allow

them to infer the intended meaning of these communicative acts. These deficits can be considered cases of inference failure because they are in large measure based on failure to do what is required to go beyond the face value of the data—namely, to combine contextual cues into a belief about the intended meaning of the words.

In retelling stories, RH-damaged patients have two interesting tendencies. First, they tend to give *verbatim reports,* rather than paraphrasing. Second, they tend to justify *nonsensical or incongruous information.* In both cases their responses can be interpreted as deficits in responding to the overall intended or inferred meaning of the story.

Pragmatic deficits in conversational speech may also be attributed to inference failure. If inference requires the apprehension and interweaving of key cues to arrive at meaning that goes beyond the superficial, then conversation is one of the greatest tests of that capacity. One must attend not only to words, but also to prior experience with the speaker and the topic, and to the speaker's affect, tone, and posture in order to recognize his or her intentions. It is possible that RH-damaged patients fail to make eye contact, interrupt at will, and generally fail to respond to the rules of conversation because the visual and verbal cues that suggest the speaker's intention are reduced to mere sensory data that do not combine into a proposition from which the patients can pass to another level of meaning. That is, they do not combine into a *belief* about the intended meaning. For example, RH-damaged patients may fail to make eye contact because information contained in the speaker's eyes does not suggest itself to them, and simply training them to make eye contact might not necessarily introduce content into that input.

Prosody helps us to convey and appreciate intended meaning, and it is not surprising that *compromise of emotional prosody* may be seen in RH-damaged patients. Interpreting and producing the prosodic features of speech requires at least three things: (a) recognition of the suprasegmental features themselves; (b) recognition of them as tools used to signal intention; and (c) the ability to apply them so that one can distinguish between sincerity and sarcasm and between neutrality and emotion. RHD may affect any one of these steps, but given that RH-damaged patients can interpret suprasegmentals used to signal word definitions, it is likely that the breakdown with emotional prosody occurs in the latter two steps. It may be that RH-damaged patients do not recognize the suprasegmental features as tools, or that their ability to relate them to a given context is impaired. Looking at prosody as potentially reflecting an inference deficit is a departure from the sensory-motor model used by Ross (1981) to explain the so-called "aprosodias."

The oft-quoted *emotional impairments* in this population may also reflect inference failure. Failure to use facial expression and body language to interpret and convey emotional content may be considered in the same

light as impaired prosody. That is, the signals we use for emotional expression require that we recognize them as tools and that we then use them to interpret intended meaning.

RH-damaged patients have been said to exhibit *impulsivity:* to respond immediately, rather than to reflect, as if response were the goal, rather than the furthering of knowledge. The very act of reflection involves weighing, combining, and relating various factors to arrive at a conclusion. Impulsive responses are superficial responses. They, too, could reflect failures of inference.

In the noncommunicative realm, close inspection of RHD *impulsivity in activities of daily living* suggests that it, too, could be the result of failure to account for situational cues, or a failure to use context to arrive at inference. *Denial of illness* itself can be looked at as a failure to assess the implications of motor, sensory, and cognitive impairment—that is, failure to combine the fact of illness with the context of events so that one can arrive at an accurate inference about its effect on one's present and future.

Other deficits associated with RHD that affect communication only indirectly include prosopagnosia and topological reduplication, also known as facial and location recognition impairments. Both of these deficits may be considered as impairments in the use of context and, hence, as inference deficits. In *topological reduplication,* available cues are not used to disambiguate location, so that patients may have difficulty distinguishing a hospital room from their bedroom at home.

Prosopagnosia, which can be extended to any class of ambiguous stimuli such as cars or one's clothes, is not a failure to recognize *what* an object is, but rather *whose* it is. It is likely that this deficit represents a failure to retrieve context—those contextual associations that tell the history of objects or faces. That is, it is a failure to infer something more about them than what they are by combining them with a particular contextual history in order to know what they mean or to enter into a second proposition about them.

RH-damaged patients have a tendency to be *verbose, tangential, and inefficient in discourse production.* In narratives, this inefficiency is often attributed to problems in organizing information into a hierarchy. For example, RH-damaged patients have been known to resort to chronological accounts of events when they are unable to give a direct answer to the question, "Why are you in the hospital?" Eventually, they arrive at the accurate response, but not before the examiner has been treated to an in-depth report of all the events, large and small, relevant and irrelevant, that happened to the patient on the day he or she was hospitalized.

Why do they do this? It is as if they cannot produce their own intended meaning, so they compensate by verbalizing everything in an effort to get there. As one patient said, "My mind, like a vacuum cleaner, sucks up every thought and spews it forth." The key points are not isolated and

woven together. This oververbalization is analogous to the functional description of objects heard from aphasic patients who cannot retrieve object names. RH-damaged patients may know the inference they wish to generate (and it appears that they do), just as aphasic patients may know what they want to say, but they are inefficient at using the available tools in a way that creates economy of effort.

This same tendency is found in complex-picture interpretation. RH-damaged patients tend to label items without regard to their significance or relevance. *Slow, laborious, or inadequate achievement of inferred meaning* pervades most complex-picture description. A familiar example will serve as illustration.

An RH-damaged patient is describing the Cookie Theft picture from the *Boston Diagnostic Aphasia Examination* (Goodglass & Kaplan, 1983). First, he ignores the various disasters while describing irrelevant details such as shoes, cups, and the window curtains. When his attention is drawn to the children on the left, he may say that they are reaching for, not stealing, cookies. He fails to deduce that their activity is covert because he fails to link it to the somewhat dazed woman standing in a pool of overflowing water. He is drawn to the poorly depicted bushes outside the window and misinterprets them as various zoo animals or perhaps a corpse. He generates wrong inferences with ambiguous stimuli such as the bushes because he does not associate them within the context of the other objects. He fails, in other words, to recognize the significant cues and to weave them together into a proposition on which he can base a set of beliefs or further propositions.

This failure to infer is characteristic of RHD deficits in both the pragmatic and prosodic and perhaps even in the affective realm. The question is, why do they occur? What explanations have been invoked to explain them?

THE SEARCH FOR AN UNDERLYING CAUSE

In 1894 Lloyd Morgan said that one should not seek to explain a psychological fact by a mechanism at a higher level if it can be explained by one at a lower level (Morgan, 1894). His dictate has guided our efforts at understanding most aspects of brain dysfunction. In searching for earlier processes that might explain RHD communication impairment, we have focused primarily on the attentional disorders resulting from neglect and on various visuoperceptual impairments. For example, in evaluating RH-damaged patients we tend to test the presumed "higher-level" extralinguistic and pragmatic disorders, and then test for neglect and visual perception, as if neglect and visual perception somehow have an effect on

extralinguistic problems. In my mind, we have never successfully linked attentional and visuoperceptual impairments to the apparently higher-level cognitive impairments just described. They may be correlated, but the causal link has never been established—possibly because one does not exist.

Neglect

Neglect in RHD can be narrowly defined as the reduced capacity to respond to information on the left side of space despite adequate sensory processing. The definition may be expanded to include impairments in various types of attention in ipsilesional as well as contralesional space. Expanded explanations of neglect include deficits in arousal or preattentive processes (Heilman, Valenstein, & Watson, 1984), failure to disengage attention (Posner, Walker, Friedrich, & Raphal, 1984), impaired recognition of the boundaries of relevant space, the focus of attention within that space, the ability to be selective, and the motivational capacity to do so (Mesulam, 1981). It could be that impairments in attention account for failure to apprehend the contextual cues necessary to arrive at inference. However, inference failure can occur in RHD even when all the key items are accounted for by the patient.

Looking at a photograph of an isolated girl, sitting on a bench in front of a large dark wall, with her head bent upon her knees, the typical RH-damaged patient will say that she is resting. The patient will fail to infer the photographer's intention of creating a sense of loneliness or sadness by virtue of the subject's small size relative to the wall combined with her dejected posture. All of these items—the bench, the girl, her posture, and the wall—are noted in the typical RH-damaged patient's description of the picture, but the inference is not. A study by Myers, Linebaugh, and Mackisack (1985) found that non-brain-damaged and aphasic subjects, unlike RH-damaged subjects, categorized this picture with other scenes of sadness or depression. Why? The reason is that its accurate interpretation requires combinations of elements, not just their recognition in isolation. The failure of RH-damaged patients to accurately interpret the mood conveyed in the picture cannot be attributed to impaired sensory input or to failure to attend to or select the key items in the picture. All the items in the picture are significant, just as the words in a poem carry almost equal weight. All the items have been noted by the patient, but the relationship has not. The inference, the belief, is missing.

This failure may reflect an attention allocation problem. Perhaps the effort to perceive what is in the picture consumes the resources available so that little attention can be devoted to recognizing relationships. If attention is consumed by the act of cataloging what is in the picture, why

is this so? What is it about perception that requires so much effort for RH-damaged patients?

Perception

We have heard again and again that RH-damaged patients have visuoperceptual deficits. And we have speculated that these impairments may affect RHD response to input such as complex scenes. Such speculation usually ends where it began. If a patient does not recognize the bushes in the Cookie Theft scene, we usually attribute the recognition error to some sort of visuoperceptual disorder and let it go at that. Without a deeper understanding of RHD visuoperceptual disorders this remains a hollow explanation.

It is important to recognize that most RH-damaged patients do recognize pictured objects both in isolation and in complex scenes. In fact, a study by Mackisack, Myers, and Duffy (1987) found that RH-damaged subjects labeled twice as many pictured objects as non-brain-damaged controls in their attempts to interpret scenes. In other words, they labeled almost everything in the pictures just as they tend to verbalize everything when they cannot get to the gist of their verbal responses. They did not have trouble perceiving objects.

But there are cases where they fail to recognize objects. RH-damaged patients tend to describe the images in Figure 16.1 as a man, a woman, and a cabbage. Most elderly and not-so-elderly non-brain-damaged subjects are initially confused by the middle item as well. But using the rest of the picture they eventually conclude that the picture is of a man, a woman, and a baby—that looks like a cabbage. Visual ambiguities are resolved through the use of context—that is, through the apprehension of the relationships among the items. Most RH-damaged subjects can label and recognize pictured information if it is not ambiguous. They fail, however, to use context to disambiguate the ambiguous and they thus fail to infer the meaning or identity of such items.

This is not to say that visual perception cannot affect inference generation. Research on visuoperceptual deficits in RHD indicates that RH-damaged patients do not have trouble perceiving isolated objects in prototypical views. Problems are manifested when subjects are presented with stimuli in degraded conditions. The nature of these degraded stimuli is crucial. Close inspection of the type of stimuli used over the past 30 years in documenting RHD visual recognition deficits suggests that they have something in common. They rely on various depth cues to be resolved.

Figure 16.1. Emotive picture. *Note.* From Farm Security Administration and Office of War Administration (FSA/OWI). Courtesy of the U.S. Library of Congress, Washington, DC.

DEPTH CUES AND INFERENCE

Why is a common depth cue deficit so important? It is important because assigning depth to our visual world is in itself an inference. Input to the visual system enters in the form of light rays, which are mapped onto the retina as a two-dimensional, flat image. We transform that initial image into a three-dimensional one containing depth through the use of specific cues. Among the cues necessary in most of the visual recognition tasks given to RH-damaged patients is the class of static monocular depth cues that include some of the following: (a) size disparity in which the larger the retinal size, the nearer the object is assumed to be; (b) shading and luminance changes; (c) changes in perspective such as linear perspective or the convergence of lines, texture gradient changes signaling the slant and distance of surfaces, and aerial perspective in which distant objects appear dimmer than near ones; and (d) occlusion or interposition, which helps us determine that one object is in front of another. This last cue is illustrated in Figure 16.2, which shows a brick in front of a cylinder. We probably make the assumption or infer that the brick is in front of the cylinder by virtue of differing types of line junctions.

To impart depth to two-dimensional images such as pictures and line drawings, we must combine static monocular depth cues such as those

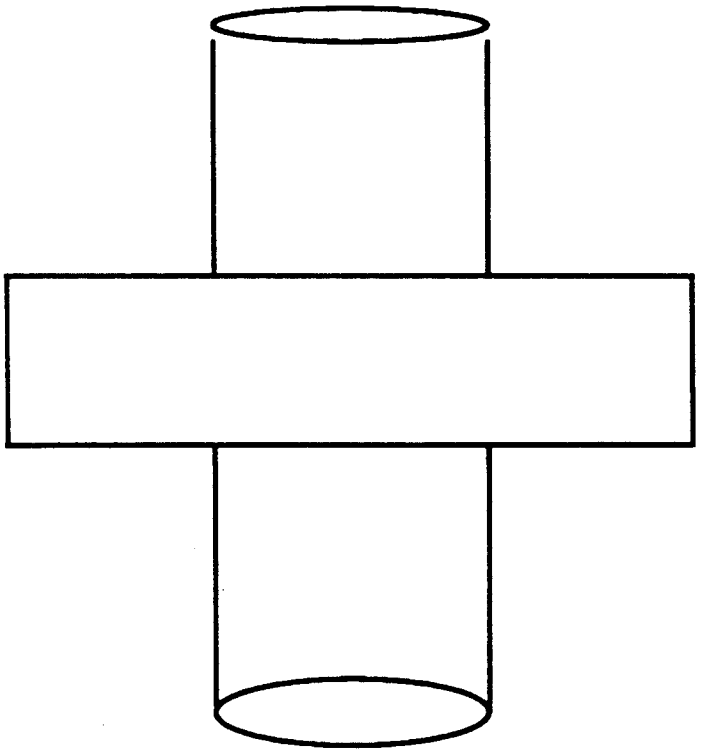

Figure 16.2. Example of the depth cue of interposition or occlusion.

described above, from which we generate an inference—the inference that depth exists in the two-dimensional input presented to our brains. In other words, inference, albeit unconscious, is operational at this very early level of visual processing. Cues are recognized and applied to sensory input to arrive at the inference of three-dimensionality.

It is in just this area that RH-damaged patients demonstrate visual recognition deficits. The type of test stimuli most frequently used in the literature includes the *Gollin Figures Test* (Gollin, 1960), the *Street Completion Test* (Street, 1931), the *Ghent Overlapping Figures Test* (Ghent, 1956), and various unusual-orientation tasks such as the ones used by Layman and Green (1988).

In most unusual-rotation tasks the subject must determine if two objects are the same or different. One object is presented in prototypic view and the other is either a different object or the same object rotated in depth. Shading and texture changes in one of the objects indicate that one end of the principal axis is farther away from the viewer and that the object has been rotated in depth. One must use two depth cues (shading and texture gradient changes) to infer depth and solve the task.

The line drawings of objects in the *Gollin Figures Test* appear incomplete. Actually, incompletion is the same thing as occlusion. Completing the

Figure 16.3. Examples of items from the *Street Completion Test*. Lower right, example from the *Ghent Overlapping Figures Test. Note.* From "Visual Recognition in Patients with Unilateral Cerebral Disease" by E. DeRenzi and H. Spinnler, 1966, *Journal of Nervous and Mental Disease, 142,* pp. 515–525. Reprinted by permission of Williams & Wilkins.

figures requires the assumption of depth in the picture. One must imagine that something is occluding or in front of the full figure to understand that it is incomplete. The same thing is true in the examples from the *Street Completion Test* (Figure 16.3). Resolving the figures requires the inference of depth—that is, the assumption that something is partially occluding the object, making it incomplete.

An example of the *Ghent Overlapping Figures Test* is in the lower right corner of Figure 16.3. Here the task is to pick out the objects such as the wineglass. Again, because the lines overlap, this task also relies on the depth cue of occlusion to be solved, just as in Figure 16.2.

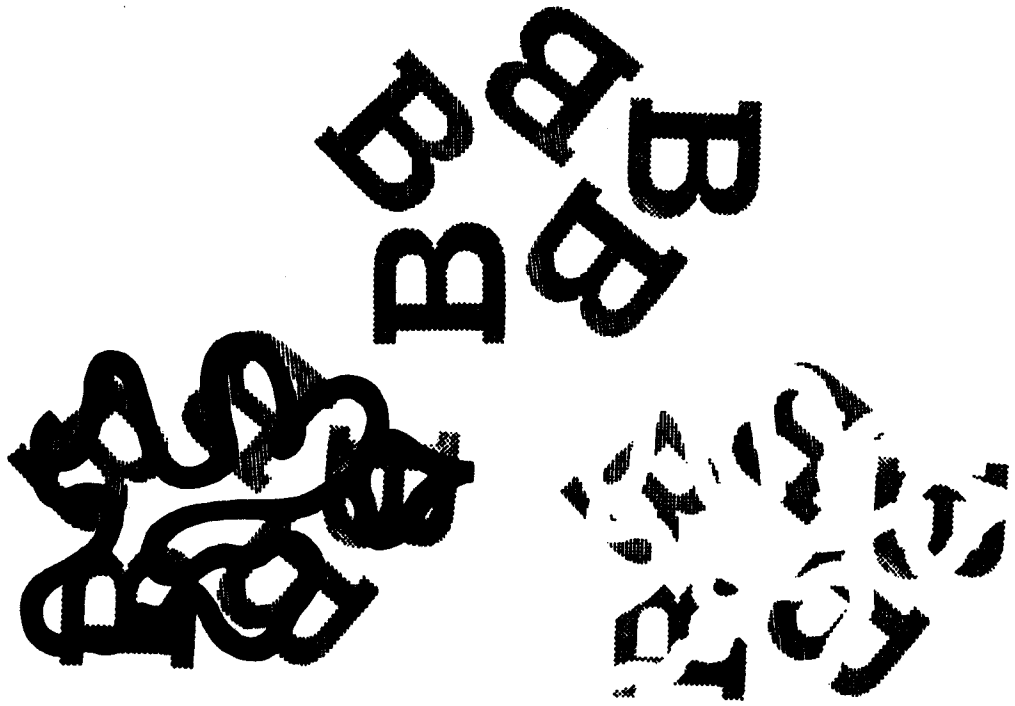

Figure 16.4. The *B*'s in the lower left are occluded by a black snake-like structure. In the lower right the occluder is the same color as the background. The exposed fragments of the *B*'s are identical to those in the lower left but are more difficult to see. *Note.* From "Stereoscopic Depth: Its Relation to Image Segmentation, Grouping, and the Recognition of Occluded Objects" by K. Nakayama, S. Shimojo, and G. H. Silverman, 1989, *Perception, 18,* pp. 55–68. Reprinted by permission of Pion, Ltd.

We are used to thinking of such tasks, particularly the *Street Completion Test* and *Gollin Figures Test,* as tests of completion. But to solve the task we must assume that there is a complete figure to be found. Inherent in that assumption is the belief that the object is partially occluded and thus currently appears incomplete, even though we cannot see the occluder.

Figure 16.4 illustrates how we rely on depth cues to help resolve an incomplete figure. A group made up of the letter *B* is in the lower left of the figure. The *B*'s are occluded by a black snake-like structure and appear to be behind the occluder. It is easy to pick out or complete the *B*'s because we use depth cues to infer that the snake or occluder is in front of the *B*'s.

In the bottom right of the figure, the *B*'s are also occluded, but they are more difficult to make out because the occluder is the same color as the background. We do not immediately see the occluder, and the *B*'s therefore appear to be in fragments. That is, we do not immediately infer depth

in the picture as we did when we could see the occluder in the lower left part of the figure. It is more difficult to complete the fragments without imagining something in front of them—without the sense of three-dimensionality in the picture. The visual system uses the cue of occlusion and thus applies depth to the picture, but it is slow to do so because the occluder is the same color as the background.

This is the same effect found in the completion items from the tests just described. The items from the *Street Completion Test* are occluded by a white snake-like object the same color as the background. Recognizable occlusion makes such pictures easier to complete. But whether or not the occluder is visible, we rely on the inference of depth to complete incomplete figures and to match objects to their counterparts that are rotated in depth.

In the real world, we rely on multiple cues for inferring depth: the static monocular ones just described as well as various binocular, motion, and oculomotor cues. This sort of redundancy may help to disambiguate stimuli just as redundant information helps the aphasic person to comprehend language.

Inferring depth in the real world does not appear to be a problem for RH-damaged patients. They are able to reach for and distinguish objects adequately. Their errors in navigation appear to be related specifically to left-sided neglect rather than to impaired depth perception. In addition, they have little difficulty with pictured objects presented in prototypic view.

Problems arise when objects are degraded, and, as stated earlier, the type of information reduction or degrading typically used in research makes reliance on individual monocular depth cues crucial. In effect, such tasks are like the *Token Test* of visual recognition (DeRenzi & Vignolo, 1962). The system is stressed by dependence on limited, nonredundant information.

Visual recognition deficits in this population may be partly attributed to an inference deficit. This is not to suggest that inference failure in communication is the result of failure to apply depth cues. Rather, it suggests that failure to apply depth cues in degraded object recognition tasks is an *instance* of impaired inference early in the system.

We generally think of inference as operating only in the late stages of cognitive processing. But inference may operate at both early and later stages and it may be affected by brain damage in early as well as later stages. In all levels of processing it appears dependent upon an ability to identify available cues, integrate them with one another and with other sensory input, and form relationships that specify meaning beyond the sensory data. This appears to be the case in something as seemingly automatic as appreciating depth, and in something as complex as interpreting a scene or story.

CONCLUSION

I have offered four hypotheses in this article: first, that RH-damaged patients suffer from inference failure; second, that inference failure can occur at all levels of cognitive processing; third, that RHD can affect inference generation at early as well as late stages of cognitive processing; and fourth, that, as in aphasia, inference failure may be a central deficit.

What are the implications of the hypothesis that the fundamental problem in RHD communication disorders is inference failure? First, inference is fundamental to communication. To communicate adequately, we must be able to understand intent and to convey our own intended meaning using the conventions or tools available to us.

Second, we can look at inference failure in very much the same way that we look at a language impairment as the fundamental deficit in aphasia—that is, as damage to a central process. Like aphasia, inference failure may be disproportionate to impairments of other intellectual functions. Inference failure may be considered a multimodal disorder with an emphasis on the visual modality, just as aphasia is multimodal but with an emphasis on the auditory modality. In this sense we can think of inference as a horizontal function that crosses modalities.

We can also think of inference as a vertical function that crosses levels of processing and one that can break down at all levels subsequent to the right hemisphere. This gives us a new perspective not only on inference, but also on the possible link between at least one type of visuoperceptual disorder (i.e., recognition deficit) and the so-called "higher-level" extralinguistic deficits associated with RHD. Both types of disorder may be manifestations of a central deficit in generating inference. Conceiving of inference failure as a central deficit may make it worthwhile to study inference in the same way we have aphasia. We may want to look for a common set of rules that operates at low and higher levels of processing for the phonology, syntax, and semantics of inference. Exploring how the process breaks down may help us understand how it works and vice versa.

Finally, the term *inference failure* gives us something to hold on to, a label, a name. The patient suffers from "inference failure." It gets us away from anatomical specificity in a way that the term *RHD communication disorders* does not. Inference failure may be associated with RHD, but just as we have cases of crossed aphasia, we may find cases of inference failure associated with left-hemisphere or diffuse damage. We can refer to inference failure as *IF.* IF is conditional, a question, a hypothesis. If the hypothesis is correct, the term may survive. If it is not, it will go the way of alchemy, phrenology, and 3-D movies.

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