Exercises in Behavioral Explanation

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Abstract

Although behaviorism is no longer dominant in behavioral science, the emphasis on environmental variables that characterized behaviorism is still relevant for communication disorders. Through an examination of causal factors in stuttering, aphasia, and normal speech development, I attempt to demonstrate that explanations in our field are most useful when they invoke environmental causes, even for disorders with obvious organic components.

Specialists in communication disorders are often faced with practical examples of the esoteric problems that preoccupy philosophers of science. Philosophical questions concerning criteria for defining terms, or the role of theory, or the nature of proof take on special urgency in the context of a child or adult who has serious communication needs. In this paper, I will explore one such problem, the nature of causal relations, with emphasis on the practice of invoking physiological causes to explain the wide range of communicative disorders treated and studied in our clinics and laboratories.

Behavioral science has rarely experienced the profound revolutionary changes that Kuhn (1970) described for fields like physics. In contrast to the hard sciences, it is rare that a theory or approach to the study of behavior has completely and irrevocably preempted all others. Rather, accepted theories may wane in popularity but then reappear, perhaps somewhat altered or dressed in new terminology. Behaviorism is an example of an influential approach that was widely accepted but is now in decline (Zuriff, 1985). While behaviorism was dominant, its emphasis on functional relationships created a reluctance to adduce organic causes for behavioral problems. Wherever possible, explanations were framed in terms of environmental influences (Johnston & Pennypacker, 1980). Now, with the reduced influence of behaviorism and the increasingly sophisticated tools for the study of the human organism, there has been a return to organic explanations for a great variety of behavioral problems, such as autism, schizophrenia, depression, and learning disability, and also for communication disorders, such as stuttering. I will argue that the explanations that are most useful *for communication disorders* are behavioral.

Distal and Local Explanations

Distal explanations are concerned with the original cause of a disorder. They are, in a sense, ultimate causes and they often, though not necessarily, appeal to physiological or biological variables. Thus, we might look for the genetic roots of almost any communication disorder, or for evidence of a medical condition or a physiological predisposition that was present at birth or early in childhood.

Local or proximal explanations concentrate on current variations in behavior, focusing for example on why stutterers have difficulty on certain words and not others, or why children undergoing articulation therapy perform adequately in quiet but not in noise. Local explanations, too, can appeal either to physiological or to environmental causes. Thus, one may attribute the occurrence of a specific moment of stuttering to a momentary laryngeal spasm, or to the phonetic composition of the word in the sentence.

There is no logical requirement that distal and local explanations for a given disorder call on the same class of causal variables. The original cause may occur once, and then disappear, or at least no longer be an active contributor to the communication disorder. A disorder that was organic in origin may nonetheless be very susceptible to environmental stresses and reactions. Conversely, a learned vocal behavior pattern may cause an organic change that then causes further vocal change. As will be discussed later, it is important for a full theory of a disorder that the local and distal causes eventually be linked to each other.

The Context of Causality

Most phenomena can be described at several different explanatory levels. According to Einhorn and Hogarth (1986), the causal explanation is situationally determined. For example, Einhorn and Hogarth (1986) ask us to imagine that a watch face has been shattered when struck by a hammer. With no other contextual information, we are likely to state that the hammer caused the glass to shatter. However, if we know that the glass was being tested as part of a quality control procedure in a factory, we might say that the glass shattered because it was imperfect, and not because of the hammer. Similarly, Carnap (1966) describes the varying causes that would be

adduced for a car accident by a policeman, a highway construction engineer, a psychologist, a car designer, etc. The objective events are the same, but the context determines the causal explanation that is proposed.

Much the same process obtains in devising explanations for the phenomena of disordered communication. To a significant extent the causal explanation is determined by the context of concerns. In our own field, I believe the context requires that we focus on environmental rather than organic variables. I will pursue this argument by invoking examples in stuttering, aphasia, and normal performance.

Stuttering

The lability of theoretical approaches in communication disorders is probably nowhere so evident as in the area of stuttering, perhaps because no other communication disorder has been so thoroughly studied. Theories of stuttering have encompassed cerebral dominance, psychoanalysis, a variety of learning approaches, genetics, and many other fields. After an extended period of active exploration of the learning bases of stuttering, efforts now seem to have shifted back to a concentration on the organism. In the following paragraphs, I will develop the argument that even if we posit an underlying organic pathology for stuttering, specialists in communication disorders will still need to tum to environmental variables.

Although I doubt that a common organic cause of stuttering exists, for the sake of argument let us assume that all stutterers suffer from a laryngeal problem of some sort. We would then know that the laryngeal problem is a *necessary* precondition for stuttering since no stutterer would be free of it; however, we would not have established that the laryngeal condition is also a *sufficient* cause of stuttering. In order to make the claim of sufficiency, we would also have to establish that all persons who have an affected larynx invariably develop stuttering. That would be even less likely than the claim that all stutterers have the same organic pathology. Organic theories of stuttering would rarely suggest that the populations of stutterers and normal speakers are at absolutely opposite ends of an organic continuum. Theorists with an organic tilt have generally proposed that the underlying condition interacts with environmental events to cause stuttering, thereby creating a predisposition to stuttering rather than an inevitability (e.g., West, 1958; West, Ansberry, & Carr, 1969; see Van Riper, 1982, for a review).

However, once the notion of an organic predisposition is introduced, the context of explanation shifts and the organic predisposition becomes less important than the external variables that call it out, especially, as is true with most communication disorders, if the presumed organic cause cannot be directly detected or treated. Attention is shifted to factors that activate the predisposition, such as early childhood illness, other speech or language disabilities, inappropriate parental expectations, etc., and research and clinical efforts are focused on finding ways to arrange the environment to block the predisposition. Thus, even if an underlying organic cause is posited for stuttering, attention is ultimately directed to environmental events that precipitate the disorder. These are the same events, moreover, that will enter into treatment and prevention programs since they represent the class of variables that clinicians can control.

The example of a laryngeal cause of stuttering dealt primarily with distal causes. It is also important to explain local manifestations of the problem. Stuttering varies in predictable ways as a function of the speaking situation, the audience, the materials spoken, etc. In addition, individual stutterers have highly reliable but idiosyncratic stuttering patterns that must be explained. Although distal and local causes need not be the same, an integrated and complete theory of stuttering would have to show how they are related. The theory should indicate not only how the stuttering developed initially, but also how that original cause led to the current forms of the disorder. If the original cause has little to offer in understanding the current forms of behavior, then it will recede into relative insignificance. Clinicians need explanations for the behaviors that are currently interfering with communication and are amenable to modification.

Aphasia

Damage to the brain is one of the defining criteria for aphasia. I believe that once the organic condition is granted, the context of explanation requires that we focus on external influences, despite the obvious organic component. The arguments in this condition are similar to those in the case of stuttering. Knowing that the condition was caused by a brain injury does not explain the language difficulties experienced by individual aphasic patients. Even if researchers are successful in finding a reliable relationship between the locus of a lesion and a general pattern of aphasic disturbance, such information is unlikely to account for the specific manifestations of the problem and will not identify the variables that currently control the client's performance. The lesion becomes a background consideration, like the hammer in the example of the watch factory.

It is tempting to attribute variations in aphasic performance to fluctuations in neurological state, but it is important to keep in mind the direction of reasoning. If neurological change is inferred because of changes in performance ("His behavior is unstable, so his physical condition must be in flux"), then the behavioral data are being used to explain the neurological events, not vice versa. This kind of circular reasoning is a potential problem in all areas of communication disorders that invoke underlying organic causes. If behavioral

fluctuations are to be explained in terms of neurological changes, a minimal requirement should be that the neurological events are capable of independent observation. We could then probe whether changes in neurological activity reliably cause certain behavioral patterns. Causal accounts of this sort, however, would require a far greater understanding of brain-behavior relationships than is now available.

It is especially tempting to explain aphasic performance during "spontaneous recovery" in physiological terms. The spontaneous recovery period usually lasts for several months immediately post-insult in aphasia, when the neurological situation is changing rapidly. Although environmental events may still have an impact on performance, it is very difficult for the clinician to trace their influence because the patient's behavior is changing rapidly. For the speech pathologist, the task during this period is to characterize and closely monitor the changes in behavior. Furthermore, if the danger of circular reasoning is to be avoided, it is important that the neurological instability should be independently observed.

The problems for the clinician in this instance may be more severe than most, but they are not fundamentally different from those that clinicians always face. Whenever behavior is unstable, regardless of the cause, it is difficult to evaluate a program of intervention because of the lack of a reliable baseline (Cook & Campbell, 1979). A severely retarded child who shows large fluctuations in behavior may also have an underlying physiological instability that affects performance. Usually, however, clinicians lack useful neurological approaches with such a child, and must intervene and conduct therapy before the baseline is established. I believe that the difference between the two situations is only that the organic damage is much more obvious in the case of aphasia. In both instances, behavioral instability defines the problem faced by the speech pathologist, and the solution involves applying methods and concepts already available in behavioral science.

Normal Performance

Although there are increasingly complex and elegant methods available to analyze physiological function, these methods are typically used to describe, rather than to explain, the behavior of persons presumed to be normal. They are offered as explanations only for abnormal behaviors. Once again, context determines the mode of explanation. For example, alcoholism is often regarded as a symptom of a disease, but the behavior of a recovering alcoholic is ascribed to will power. If two persons were known to have similar brain damage, but only one developed language problems, the tendency would be to explain the behavior only of the language disturbed patient in neurological terms. Similarly, all speakers are nonfluent at times, but it is only the fluency lapses of stutterers that are likely to be explained in terms of laryngeal function. That is, in the case of abnormal behavior, physiological variables are offered as explanatory whereas in normal behavior, the physiological variables are used to describe rather than explain performance.

During speech development, children make errors in articulation, semantics, or syntax that may be similar to the errors made by aphasic subjects, but we don't typically appeal to neurological explanations for these errors. The brain is involved in normal acquisition as well as in aphasia, but we are not even close to describing language development or function in neurological terms. Modern linguists like Chomsky (1975) espouse a strong nativist approach, but in fact their models deal with the structure of linguistic knowledge rather than the brain itself. Chomsky has likened language capacity to an organ of the body, but he doesn't describe the organ, only what it accomplishes.

The Uses of Physiological Explanation

A clinical anecdote recounted by a colleague may help to highlight the interplay between physiological and behavioral information. After an extended trial of speech therapy, a speech clinician working as part of a cleft palate team concluded that a young cleft palate girl would not profit from further therapy and that, given the structure of her oral cavity, she had reached the limits of her ability to change her speech. He counselled the team to consider a different form of intervention, either a prosthesis or surgery. They disagreed, because physiological measures of palatal movement and of velopharyngeal closure indicated the girl was improving under the speech therapy regime. The team persuaded the speech clinician to persist in the speech therapy program. However, after a considerable period in which no further improvement in speech was obtained, the physicians agreed to fashion a bulb for the young client. The result was immediate and dramatic improvement in speech and voice.

This anecdote provides several insights into the interaction between physiological and behavioral data. First, the two levels are not the same: The changes that were observed in the physiological measures did not guarantee corresponding improvement in speech. However, the treatment method that finally helped was a change in the architecture of the child's mouth. Furthermore, it was prompted by the speech clinician's judgment that behavioral improvement was not possible for this child without a physical change.

In recounting this episode, my colleague argued that the decision to change approach was based on an analysis of physiological data indicating that the child had reached the limits of her physical capacity, but I believe that the decision came, in fact, from behavioral data. I suggest that the

clinician's major contribution was in convincing the other team members that therapy was not likely to bring about any further improvement in speech, and that it took a behavioral and not a physiological analysis to come to that conclusion. The need to consider other alternatives arose when it was recognized that a skilled clinician had exhausted the available behavioral therapy methods. Interestingly, the story did not end there. Soon after the bulb was introduced, with its dramatic effects, another speech pathologist suggested a program of bulb reduction and this too was successful. After a surprisingly brief time, the bulb was removed entirely with no loss of speech quality. Thus, even the original conjecture that the child was physiologically incapable of producing normal speech was brought into question.

We might still want to question whether the bulb caused the change in speech. Certainly its introduction was followed by improvement after a long period of no gains. Because the clinical anecdote does not describe a controlled experiment, one cannot rule out the possibility that the bulb was helpful only because of the prior extensive speech therapy, or that improvement would have ensued even if the bulb had not been inserted. However, setting aside methodological concerns, I would argue that the bulb was helpful because it facilitated behavior. Behavior is the relationship between an organism and the environment and, quite obviously, without an intact organism certain behaviors are scarcely possible. Where organic factors are undeniable, they are best incorporated into a behavioral analysis, not as explanatory variables in themselves, but rather in terms of their influence on behavioral possibilities.

Conclusion

I have argued that the explanations likely to be most useful for communication disorders are behavioral, even for conditions with an acknowledged organic component. We use environmental stimuli to help clients change their behavior. We seldom can directly observe or modify the physiological variables that are involved in communication disorders. As clinicians, we attempt to arrange the environment in ways that facilitate improvement in performance. The changes that occur are functionally related to these environmental events, and are, therefore, properly explained through laws that focus on the interactions of the behaving individuals with their environment. Physiological explanations belong to another level of analysis. There is nothing in a complete behavioral analysis that should conflict with a physiological explanation. Ultimately, physiological and behavioral explanations should stand in perfect relationship to each other as parallel explanations. That undertaking can only be enhanced by a concerted effort to develop an independent and comprehensive theory of behavior to complement similar efforts in physiology.

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