Peer Commentary on "Exercises in Behavioral Explanation" by Gerald M. Siegel

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In Support of a Physiological Approach to the Study and Treatment of Communication Disorders

In his article, Dr. Siegel argues that explanations of communication disorders are more useful when they involve environmental rather than physiological causes. I will argue that while identification of environmental variables which affect speech, language, and vocal behaviour provides information that is useful in understanding and treating communication disorders, such information is not more useful than that provided by identification of organic causes for, or relevant physiological variables associated with, disordered communicative behaviour. As part of his position, Siegel seems to equate behaviour and environment; for example, he uses the term "behavioral explanations" interchangeably with "explanations which invoke environmental causes." The position taken here is that it is physiological and behavioral variables which are equatable, given that both refer to events of organism function, while environmental variables constitute a separate category: a class of events which impinge on the organism, potentially affecting both behaviour and physiology.

Siegel points out that causes of communication disorders can be considered to be of two types: explanation of the original cause of a disorder, and explanation of current variation of its behaviours. Let us consider the roles of environmental and organic variables in each of these types of explanation and, further, the application of information about environmental or organic causes to intervention. I will begin by discussing an explanation for local variation in behaviour. We know that when charted over time the behaviour of a given disorder may be found to change with regard to its level of severity. Often, such variation can be associated with particular environmental conditions. When disordered behaviour can be demonstrated to change locally and systematically as a function of environmental stimuli, this relationship can be used in therapy in several ways. For one thing, response contingent stimulation in the form of positive or aversive events can be instituted to promote an increase in the frequency of occurrence of the more desirable, or least severe, form of the disordered behaviour and a decrease in frequency of the least desirable form. In this way, a person's communicative abilities can be improved by making use of existing behaviours rather than by learning new ones. In addition to this general application of a demonstrated relationship between environment and behaviour, specific information about how such a relationship is manifest in situations of daily living (i.e., identification of situations in which a person's communication proficiency is most significantly reduced) can be used to assist the client in coping most adaptively with his environment. For example, a person who stutters most severely in one particular circumstance such as talking with his boss, may want to develop special strategies for this situation.

In addition to explaining variation in severity of a given disorder we must also account for the fact of its occurrence at all, at any severity level. Explanations of why one person and not another exhibits a given disordered behaviour are explanations of original causes. Whereas local variation can be attributed to environmental variables (along with random variation), explanations of original cause of communicative disorders, when they are available, generally involve organic variables in addition to or instead of environmental variables. It is evident that identification of specific causal factors, be they organic or environmental, is most useful for treatment purposes when they can be eliminated. Notably, in the treatment of communication disorders it is seldom the case that factors of original cause can be immediately or completely eradicated, as is the case when, for example, a physician is able to cure an infection by administering an antibiotic. Nevertheless, causal factors which give rise to communication disorders can often be dealt with effectively. Consider known or presumed environmental precipitants of communication disorders; for example, delayed development of speech and lan-

guage exhibited by a child with a history of neglect may be attributed to lack of adequate speech and language stimulation, or the development of a pattern of chronic stuttering in a young child may be related, at least in part, to the effects of a communicatively stressful environment. In such cases, efforts are made to remove causal influences by replacing the harmful environmental conditions with those conducive to promoting desired behaviours. Most clinicians can cite instances where, over time, environmental changes of this nature have been associated with normalization of a child's communicative abilities. Certain organic conditions which are known to give rise to communication disorders can also be treated, though often they are not completely correctable. Notably, since these factors involve organic pathology, correction is usually carried out by members of the medical profession or by specialists in fitting corrective aids, or prostheses. Examples of organic conditions which may be treatable are hearing impairment, cleft palate and structural mass on the vocal folds. The critical first step in a total program for treatment of a communicative disorder having a known organic basis, then, is to undertake management which ensures that the structural/functional integrity of a person's communicative system is optimized. Where organic pathology can be clearly identified, as in the examples given above, management solutions which at least partially correct or compensate for the problem are usually forthcoming. However, where organic pathology is suspected but not identified in sufficient detail, corrective solutions are not a possibility. Hence, research efforts toward the goal of better understanding the nature of disorders with suspected organic bases are clearly warranted. Consider, for example, the pattern of language disability which persists into late childhood and adolescence and has been linked to possible auditory processing difficulties. Should such pathology be demonstrated in a subgroup of language disordered children, and if the specific nature of these difficulties could be delineated, some method of compensating for the disfunction, possibly in the form of a corrective prosthesis, might eventually be developed.

Although identification and modification of factors of original cause may be important in the treatment program of a communicatively disordered person as just described, it does not constitute the bulk of the work of speech-language-voice clinicians. Given that factors of original cause in communication disorders are either not completely eradicable or not identifiable, treatment requires that behaviour be modified directly to improve communication abilities. It is important to recognize, however, that a focus on behaviour does not preclude an interest in physiology. In fact, we frequently make use of physiological information to guide the development of procedures and instructions which help a client alter his or her behaviour. For example; in articulation therapy we may use information about incorrect placement, manner, or voicing features of misarticulated sounds to guide determination of appropriate therapy goals and teaching methods. In these circumstances we attempt to provide information to the client or arrange tasks which will assist in replacing incorrect movements of speech system structures with those which will result in production of perceptually normal phones. In voice therapy we also make use of information about normal and abnormal physiology to guide our conduct of therapy. Consider the case of a client who exhibits a chronic pattern of hard glottal attacks. In addition to the aesthetic problem presented by this unpleasant sounding pattern of speech start-up, hard glottal attacks are known to be abusive to the vocal folds because they are produced with extremely high levels of activity of the lateral cricoarytenoid muscles, which results in excessive medial compression of the vocal folds (Hirose & Gay, 1973). Over time, excessive medial compression can lead to structural changes of the vocal folds. On the other hand, both soft (simultaneous) attack and breathy (or aspirate) attack are performed with appropriate levels of lateral cricoarytenoid activity (Hirose & Gay, 1973). In choosing a method to replace the hard attack, a clinician may first teach soft attack since it is the most normal sounding pattern. However, if the client has difficulty adopting this behaviour, the clinician might choose instead to teach a breathy attack pattern; although breathy attack also sounds abnormal, it constitutes a pattern which is less abusive to the vocal folds than hard attack.

A third example to illustrate application of physiological data to treatment paradigms comes from stuttering therapy. Programs for the treatment of stuttering generally require clients to learn certain motor targets as a means of producing stutter-free speech; for example, they might learn to produce soft-articulatory contacts, which require that they perform oral articulatory movement with minimal muscular tension, or gentle voice onset which requires that they alter laryngeal activity at voice onset in ways that differ from that for normal modes of voice onset (see, for example, Van Riper [1973] and Webster [1979]). Use of targets such as these in therapy implies that we have identified the nature of motor system aberrancy in these speakers. However, the targets that are taught have been developed largely on the basis of client and clinician intuitions about what stutterers do differently from nonstutterers (and hence incorrectly) when they talk, and on trial and error experimentation, rather than on a firm understanding of their motor system disfunction. Interestingly, stuttering therapy programs are generally very effective in helping clients to achieve fluency, though frequently gains are made only on a temporary basis. Perhaps if we knew more about the physiology of stuttering and the physiological vulnerabilities of these speakers, we would be able to develop programs which yield more lasting results. Failing this, at least we would be better able to explain our program successes and failures. Notably, such information may be forthcoming since the primary research effort in stuttering today is directed toward improving our knowledge of motor system function in these

speakers during their production of both stuttered and fluent speech.

All of the examples given above demonstrate that when behaviour is changed in therapy so also is physiology. Such dependence between physiology and behaviour must necessarily be the case because they refer to the same events of organism function; more specifically, physiology refers to the function of systems or organs of the body, while behaviour refers to the consequences (or aggregate) of systemic or organic events which are (is) manifest as observable activity by the organism. Thus, physiology and behaviour each refers to a different perspective of the same phenomenon. As well, in the sense that physiological events are component parts of a total process which is behaviour, they may be thought of as causal to, or responsible for, behaviour. In whichever of these ways one conceptualizes the relationship between physiology and behaviour, it is true that without a change in physical function, behaviour cannot change. In other words, changed physiology must underline the learning of any new behaviour. As such, physiological change is routine activity for an organism and is not restricted to therapeutic intervention. Moreover, when existing physiology is not modifiable, new behaviour will not be exhibited and a behavioral approach to treatment is not appropriate. For example, it has been found that behavioral therapy with spasmodic dysphonics is unsuccessful. Vocal spasms can be reduced temporarily while a client talks under such conditions as whisper, loud voice production or falsetto, but typically clients are not able to carry over use of the laryngeal patterns in these conditions to reduce spasms while talking at normal volume in chest register. As a result, medical management, in the form of surgery, has been undertaken with some of these clients in an attempt to alter existing laryngeal physiology another way. (See Friedman et al. [1987] for a review of such procedures.) Interestingly, the gains from this type of management have also proven temporary for many patients, demonstrating that even surgical changes in peripheral physiology may not be sufficient to permanently alter laryngeal function of speakers with this particular disorder.

A problem encountered in relating physiological information to behaviour is one of identifying those specific physiological events which underlie or are associated with a given behaviour. Peripheral systems such as the larynx, lungs and oral articulators are more accessible for observation and study than is the brain, and events of peripheral system function are more easily interpreted than neurophysiological events. Generally speaking, then, it is considerably easier to identify relevant physiology for speech and vocal behaviours than to identify relevant physiology for language behaviours. Nevertheless, identification of physiological events which are associated with any particular communicative behaviour can potentially be problematic. Siegel's clinical anecdote illustrates such difficulty. In describing the assessment of speech function of a child with a cleft palate, he states that behavioral and physiological data appeared discrepant: The child demonstrated adequate velopharyngeal function but poor speech performance. Although he indicated that measures of palatal movement and velopharyngeal closure were obtained, it is important to note that Siegel did not describe what equipment was used to obtain this information, nor did he specify the task that the child performed while the measures were being made. Such information is critical, since measurement accuracy may vary across methods of measurement and since velopharyngeal function may be different both across speaking tasks and for nonspeech as opposed to speech tasks. Given that the physiological and speech data were discrepant in the case Siegel describes it seems likely that at least one of these two situations applied: Either that physiological measures were not accurate because of instrumentation inadequacy or that evaluation of physical functioning was made during a different task context than that in which speech performance was judged. With regard to the latter explanation, evaluation of speech performance may have been based on production of continuous speech, for example, whereas measures of velopharyngeal function might have been obtained while the child produced single words or short units of speech, or possibly even during performance of nonspeech tasks. The only other explanation of a discrepancy between assessment of speech performance and velopharyngeal function is that quality of speech performance did not reflect the kinds of problems which occur as a function of poor velopharyngeal closure, but this seems unlikely in view of the fact that the child's speech improved when a bulb was inserted. The main point here is that if measures of speech performance and velopharyngeal function were accurate, they should not have been discrepant unless they were evaluating different phenomena.

In Siegel's clinical example, improvement in speech performance occurred only after the child was fit with a speech bulb, as previously stated. Apparently behavioral effort alone was not sufficient to evoke a change in physiology. Over time, however, bulb size was reduced and eventually removed without a decrease in quality of speech. Siegel concluded that introduction of a speech bulb was helpful to this child's progress in therapy "because it facilitated behaviour." I suggest, however, that the bulb was helpful initially because it reduced velopharyngeal port size and ultimately because it facilitated an improvement in velopharyngeal functioning. As such, a temporary change in structure followed by a permanent change in physical function of the client was associated with or underlay the behavioral changes in speech performance. It is further suggested that monitoring of physiological data might have aided the efficiency with which the bulb reduction program was implemented. For example, to assist in determining the next smallest bulb size, information regarding degree of velopharyngeal closure with bulbs of various sizes could

have been obtained. The smallest size that gave closure comparable to that already achieved would be a reasonable criterion for bulb size selection and would have provided a means of objectifying such a decision.

Summary and Conclusion

The initial step of a comprehensive treatment approach for communication disorders is identification of possible factors of original cause. Where environmental factors are believed to be contributing to the development of disordered speech and language, efforts can be made to replace the negative influences with those which are more conducive to development of normal abilities. When an organic pathology is identified as causal or potentially causal to disordered communication, it can often be dealt with by medical management or application of a prosthetic device. Although complete correction of, or compensation for, a pathological condition may not be possible, treatment will often improve the structural/functional integrity of a person's communicative system. Given that in most cases of communication disorder either causal factors cannot be completely eradicated or the cause is unknown, the primary work of speech-language-voice clinicians lies in the direct management of behaviour rather than in the elimination of causal influences. Communication abilities can be improved by modifying behaviour in two ways primarily: by modeling or otherwise eliciting new behaviours, and by increasing the frequency of already existing desirable behaviours while decreasing the frequency of undesirable behaviours. In the former case, when patients are required to learn new behaviours to improve communicative performance, they must make changes in existing physiology to do so. In such cases, information about relevant normal and aberrant physiology can be useful to improve both the efficiency and effectiveness of the programs used for teaching the new behaviours. In the latter case, environmental stimulation can be used to assist the client in optimizing his current abilities.

Whereas Siegel argued that environmental variables are readily modified while physiological variables are not, I have attempted to show that physiological variables also are modifiable, and in fact, must be modified, if behaviour is to change. I have further attempted to show that information about specific environmental and organic factors of original cause, knowledge of an environmental explanation of local variation of behaviour, and data regarding abnormal and normal physiology can all be used in the development of a comprehensive treatment program. My main point is that knowledge of the specific organic pathology responsible for, and of relevant physiological variables associated with, disordered communicative behaviour, is no less essential to understanding and treating communication disorders than is knowledge of environmental causal influences. Research efforts in all of these areas are vital to our advancement as a discipline.
J.A.

References

Friedman, M., Toriumi, D.M., Grybaushar, V., & Applebaum, E.L. (1987). Treatment of spastic dysphonia without nerve section. *Ann. Otol. Rhinol. Laryngol., 96*, 590-596.

Hirose, H., & Gay, T. (1973). Laryngeal control in vocal attack. *Folia Phoniat., 25*, 203-213.

Van Riper, C. (1973). *The treatment of stuttering*. Englewood Cliff, NJ: Prentice-Hall.

Webster, R. L. (1979). Empirical considerations about stuttering therapy. In H.H. Gregory (Ed.), *Controversies about stuttering therapy*. Baltimore: University Park Press.

Peer Commentary #2

In this article Siegel deals with an issue that is central to the field of communication disorders—the relationship between theory and practice. This is an issue that is generally not given as much consideration as it merits. We sympathize with Siegel's frustration at the current tendency to ascribe causality to organic events, perhaps too quickly. Theories of behavioral disorders often become popular explanations for reasons that are not always related to the theory's ability to accurately explain psychological processes. During the 1970's there was a tendency for explanations to involve environmental stimuli. In neither case are these trends based exclusively, or even primarily, on the explanatory power of the theoretical positions.

Siegel argues that those "explanations" of greatest use to clinicians are environmental (or in his terms, behavioral) since the variables involved are more easily subject to modification. At first glance this argument seems valid and makes sense. Since practitioners in the area of Communication Disorders are required to devise immediate solutions to the communication problems of their clients, behavioral or environmental "explanations" of such disorders are of considerable value. Behavioral variables are more modifiable than brain lesions or morphological anomalies (although in light of modern pharmacology it would be wrong to assume that all organic variables are necessarily less modifiable than are environmental variables).

However, upon closer scrutiny there is much with which we would disagree. Siegel states that "clinicians *need explanations* for behaviours that are currently interfering with communication and are amenable to modification" (our italics). While environmental variables might indeed be more useful for clinicians, as scientists we must ignore such concerns and concentrate on devising explanations of behavioral disorders that most accurately explain the phenomenon under study, regardless of extraneous considerations, even those as important as clinical utility. Clinicians indeed should focus on those

variables most amenable to modification, but to imply that science should skew its focus on the basis of such considerations is contrary to the nature of the enterprise and illogical. Furthermore, to do so would be very short-sighted. Variables not modifiable currently, may well be in the future.

Perhaps the problem lies in Siegel's use of the word "explanation." Behaviour therapists have long made arguments similar to those presented by Siegel. Their concern was to identify the contingencies or stimuli controlling (or maintaining) observed behaviour. Their methods were to collect baseline data on the frequency of the behaviour and then to modify the frequency of the behaviour by altering the reinforcement contingencies in the environment. There was no assumption made about the actual cause of the behavioral disorder, or whether relevant reinforcement contingencies might actually explain the emergence of the behavioral disorder. Behaviorism fell into disrepute because the learning theories on which it was based were simplistic and could not account for the rich cognitive processing occurring during learning. But the basis of the approach, and its sound methodology, lie close to Siegel's appeal, and the issue of scientific explanation is kept comfortably at an arm's distance. In a similar way, Siegel makes an important point for clinicians. It is essential to focus on the within-subject variance in the occurrence of behavioral events. A focus on such variance may well yield insights into environmental stimuli that regulate the frequency of undesirable behavioral events and may well be amenable to modification. This logic is, of course, at the heart of behavioral therapy.

We would argue that the problems with Siegel's argument emerge because he confuses this simple, rational appeal with the construction of scientific theories. For example, Siegel's treatment of physiology is specious, and he draws a dichotomy between physiology and behaviour that is frustrating and ignores contemporary neuroscience. Behaviour is a manifestation of physiology; it occurs as the function of the movement of muscle directed by neural signals. The simple fact that it is more readily observable (or modifiable) than the secretion of a hormone or the transmission of an electrical impulse from one cell to another does not logically imply that it requires "another level of analysis." Likewise, environmental stimuli are ultimately physiological events. A flash of light in the distance is clearly a stimulus of environmental origin, but it is processed in terms of the activity of retinal cells. In the absence of such retinal processing, the stimuli may just as well never have occurred. The distinction between environmental and physiological variables is false. There are stimuli whose origins are internal or external to the body, but this is hardly the logical basis for different "levels of analysis." Modern neuroscience has made considerable advances in understanding brain function largely because of its ability to recognize such a longstanding dichotomy as being false.

The issue dealt with in this paper is simple and should not serve to separate clinicians and scientists: Explain the variance in behaviour (note, we said the issue was simple, not the solution). Identify variables that account for this variance and devise an appropriate intervention. Clearly, some variables are more subject to modification than others. So, choose the ones that you can work with at the time. This, in very simple terms, is Siegel's point, and it is an important one. However, there is really no need to compromise the endeavors of scientists or to devise false "levels of analysis" in order to bring this message to the practitioner.

G.S.W. and M.J.M.

Response to Critiques of "Exercises in Behavioral Explanation"

Although there are a number of issues raised, the central idea that emerges from the critiques of Armson, and Waters and Meaney, and the one I will address in this response, concerns the relationship between behavior and physiology. According to Armson, behavioral and physiological events are equatable since both relate to functions of the organism. Waters and Meaney make a similar point, I believe, when they comment that behavior is a manifestation of physiology and that the distinction between environmental and physiological variables is false.

There is a sense in which these concerns are valid. The boundary between environment and physiology is not a fixed one. Technological advances make it possible to observe and record events that occur "under the skin." At least some of these events can be studied in relation to environmental changes in the same way that movements of the tongue and lips can be studied. My argument is not that physiological explanations are invalid or that physiology doesn't exist. I am convinced that physiological explanations are very important for a full understanding of the behavior of organisms. I become uneasy, however, when I see an implication that explanations that are framed in physiological terms are somehow more basic, or fundamental, or correct than those at the behavioral level. To state that all behavior is physiology is, I think, to miss the point. By the same token, it would be reasonable to argue that all physiology is biochemistry, and to embark on an endless search for the "ultimate" level of description.

The distinction between physiology and behavior is no more false than is the distinction between psychology and sociology. They represent different levels of analysis. Of course the two levels have to be brought into consonance for a complete understanding of the behavior of organisms. For physiological and behavioral explanations to be most profitab-

ly related to each other, it is important to explicate each system fully. Problems arise when we too readily slip into physiological explanations to fill the gap in our behavioral understanding rather than refining or modifying behavioral theory. It would be equivalent to introducing such concepts as motivation, or disappointment, or frustration to explain a gap in physiological theory. Treatments that invoke mind over matter are of this sort, and they are unsatisfying because they wander out of the theoretical framework that physiologists can deal with. This is not to say that emotional factors can't influence physiological states; but if they are to be included in a physiological theory, they must be reinterpreted into physiological terms. Otherwise, they don't fit into established theory and are of limited utility in advancing that theory.

The temptation is always great in our field, because we draw on so many other disciplines, to invoke physiology or neurology when out behavioral analyses seem inadequate. Rather than pushing our theories to the limit and working to improve on them, we shift into a different mode. When we have difficulty understanding and treating recalcitrant articulation problems, it is tempting to ascribe the problems to developmental apraxia. When we find children with complex language problems, there is always available the diagnosis of developmental aphasia. When all else fails, we can ascribe difficulties to auditory processing deficits. All of these so called explanations take refuge in vague physiological inferences that are not capable of independent verification, and that unfortunately pose as explanations at just the points when we should be pursuing problems. My discomfort with such explanations is that they cut off further inquiry; they are answers when what we really need are better, more refined questions.

When physiological variables are used to explain problems in behavior, the variables take on an autonomous existence, as though these variables are not themselves the product of some process. If we think of stuttering moments as the expression of a physiological deficit, then we must ask why the physiological deficit expressed itself at just that moment. If behavior is lawful, then we are obligated to seek out the causal chains where we are best equipped to integrate them into our theories. Physiological explanations are neither more nor less real or valid than behavioral ones. Returning to stuttering, if stuttering is due to a physiological response, we still need to know why that physiological response tends to be diminished over repeated readings of the same passage, or why that physiological response is more likely to occur on certain parts of words than on others, or why in certain situations than others.

I said in my original article that physiological and behavioral explanations are parallel, but they are not the same. It is important that both levels be explored. And I certainly won't deny that the more we understand about the physiology of behavior, the better we will be able to serve our clients and expand our understanding of communication disorders. However, I don't believe that one type of analysis will obviate or preempt the other. Waters and Meaney give a nice example of how a behavioral pattern of vocalizing can induce vocal pathology. A physiologist, however, would not be satisfied with that kind of explanation of vocal abuse. The physiologist will want to know just how vocal abuse is translated into physiological terms, and which physiological mechanisms are engaged when the folds become swollen or irritated or ulcerated. A complete behavioral explanation of how those physiological processes were initiated will not complete the physiological task. Nor will a complete physiological explanation complete the behavioral task. Both are needed.

I have argued that the arena in which our discipline can contribute most usefully, because of our own scientific training, and the unique requirements of our professional practice, is the behavioral. This is not to diminish the importance of other kinds of inquiry. Problems arise, however, when physiological variables are adduced to explain difficult behavioral problems. This practice, rather than promoting scientific inquiry, tends to discourage it, and it is a concern with this tendency that motivated me to write my original article.

G.M.S.