Journal of Phonetics (2000) **28,** 225–232
doi:10.1006/jpho.2000.0115
Available online at http://www.idealibrary.com on IDEAL®

Basic science at the intersection of speech science and communication disorders

Lynne E. Bernstein

House Ear Institute, Los Angeles, CA, U.S.A.

Gary Weismer

Department of Communicative Disorders, Waisman Center, University of Wisconsin-Madison, U.S.A.

Accepted 3rd August 2000

In this introduction to this special issue of the *Journal of Phonetics*, the concept of generalization of scientific explanations across normal individuals and individuals with identified clinical disorders that affect speech production and perception is discussed. Papers dealing with speech production in persons with hearing impairment, aphasia, dysarthria, voice disorders, and structural modifications of the vocal tract, as well as simulations of hearing loss and lexicons show how data from speech and hearing disorders may inform theory about normal processes. In this context, clinical disorders are seen as opportunities for basic scientific research. Associated with these opportunities are potential pitfalls for scientific experimental design. Chief among such pitfalls is the loss of randomized selection of individuals for experimental study. The effect of this problem on experimental design is discussed along with some suggestions for alternative research designs and methods for incorporating results from clinical disorders in basic scientific explanations.

This Special Issue of the *Journal of Phonetics* is focused on the use of results from experiments that exploit or integrate knowledge about clinical disorders of speech perception and production for the development of basic scientific knowledge. "Basic science" refers, minimally, to the search for fundamental explanations of mechanisms underlying the processes of speech communication. Frequently, when studies involve disorders/impairments, the research is reflexively labeled "clinical". However, the term "clinical research" should most appropriately refer to research in which scientific methods are applied to the evaluation of treatment outcomes or to the description of patient characteristics, and not to research focused on fundamental explanations. When basic science research is integrated with knowledge about clinical disorders, the disorders are viewed as natural experiments and opportunities to observe factors not ordinarily available for experimental manipulation in humans, and/or that cannot be viewed across a large enough range of settings under normal conditions.

The overall goal of this issue is to give readers insight into scientific opportunities that clinical disorders afford as well as scientific issues that arise precisely because study

0095–4470/00/030225 + 08 $35.00/0

participants exhibit clinical disorders. Contributing authors were asked to discuss explicitly how they view and use data from clinical disorders. They were asked to reflect on advantages and pitfalls associated with studying clinical disorders as a means to obtaining fundamental knowledge. Communication sciences has relatively few scientists conducting basic science that integrates knowledge of clinical disorders, and research results from studies of subjects with disorders are frequently omitted from the context of theoretical discussions among scientists who typically obtain their evidence from normal participants. Therefore, another goal of this issue was to provide the authors an opportunity to make the case for why the clinical disorders should be looked to on a more regular basis as sources of fundamental scientific knowledge.

All of the invited authors for this Special Issue are practitioners of basic science. Their goal is to integrate evidence from communication disorders into general explanations of speech production and/or speech perception.[1] The experiments they describe derive from deafness (MacEachern, 2000; Perkell, Guenther, Lane, Matthies, Perrier, Vick, Wilhelms-Tricarico & Zandipour, 2000), hearing impairment (Lum & Braida, 2000), dysarthria (Kent, Kent, Weismer & Duffy, 2000; Chen, Stevens, Hong-Kwang & Chen, 2000), aphasia (Katz, 2000), pathological voice quality (Gerratt & Kreiman, 2000), nasal surgery (Chen *et al*., 2000), and vocal-fold nodules (Chen *et al*., 2000). Technologies to ameliorate some of these conditions, such as cochlear implants (Perkell *et al*., 2000) and surgery (Chen *et al*., 2000), provide further opportunities for study.

Perkell and his colleagues present a model of speech production, and show how that model has been influenced by data collected from persons with various forms of hearing impairment. Importantly, several of the critical aspects of their model were conceptualized on the basis of data collected from persons with profound hearing loss, in some cases both before and after attempted management of the loss. Perkell and his colleagues consider various kinematic and acoustic aspects of sound class production as affected by hearing impairment, and in so doing highlight the influence of their findings on model development. *Kent and his colleagues* review the literature on brain mechanisms underlying the production of speech, and conclude from lesion studies that the classical understanding of the neural substrate for normal speech production is probably incorrect. The long-standing Mayo Clinic view of dysarthria categories as straightforward clinico-anatomic entities is called into question, and specific suggestions are made to show how further study of persons with particular types of neuropathology can illuminate general speech production models and theories. *Chen et al.* show how the acoustic theory of speech production can be refined and extended, based on the study of persons with hearing impairment, with dysarthria, and who have experienced either a surgical or natural modification of a speech mechanism structure. *Katz* summarizes the literature on coarticulatory data in persons with forms of aphasia that are known to affect speech production, and concludes that these data bear on the status of coarticulatory phenomena in theories of "normal" speech production. *Gerratt and Kreiman* review their work on reliability and validity of rating scales in voice disorders, and in doing so present the "inverse" case of how principles from the study of normal speakers may constrain the understanding of voice disorders; they also discuss how these constraints may operate in the opposite direction and hinder the development of general ("normal") models of voice disorders. *Lum and Braida* present a variant on the theme of this issue, that of simulating

[1]Although the term "explanation" is notoriously slippery, in general it can be said that an explanation identifies and elaborates the cause of some process, performance, or other phenomenon (Gasper, 1991).

disordered hearing in persons with normal hearing; the obvious advantage of this approach is control over experimental conditions that cannot be realized when the disorder occurs in the natural state. The encouraging nature of Lum and Braida's results suggests that important issues in speech perception theory may be addressed experimentally using such simulations and the control they afford. Finally, *MacEachern* presents another form of simulation, but in this case of an English-like lexicon. MacEachern uses the simulation to show that visual distinctiveness of the English lexicon for speechreading is a property of the segment inventory and sound sequencing characteristics of the language, rather than of the lexicon itself.

What follows is a consideration of some issues that are relevant to the use of data from persons with speech and hearing disorders in the construction of models and theories of speech communication. The individual contributions summarized above, however, contain the best lessons about the theme of this special issue.

1. What should be the scope of theoretical explanations about speech production and perception?

Science is the process of exposing the underpinnings of, or truths about, nature. A generally accepted notion is that the goal of laboratory research is to make verifiable statements about phenomena that occur outside of the laboratory, that is, to be able to generalize. We propose that the explanatory responsibility of theories for which claims of generalizability are made extends to *any* individual speaker or listener, even if the speaker/listener is identified with a clinical disorder of speaking or listening. We also propose that evidence obtained from studying clinical disorders can inform understanding of normal behavior.

That theories should have a scope of generalization across individual variation in individuals *without* clinical labels (e.g., "deaf", "dysarthric") is not a particularly provocative assertion. However, the assertion that the long-term goal is to develop theories whose scope extends to individuals in both normal and clinical populations probably *is* provocative. Why would a theory that accounts only for individuals who do not qualify for clinical labels not be general enough? The answer is that clinical disorders usually represent variations or perturbations of normal speech perception and production systems, not transformations of systems into new or entirely different systems (cf. Caramazza (1986), for a discussion of conditions under which a disordered system might no longer provide evidence about a normal one). For example, deafness does not result in a type of perception that requires an entirely new set of assumptions about hearing, nor does dysarthria transform a speech mechanism into something that generates sequences of sounds in an entirely new way. Under clinical conditions, compensations, enhancements, and plasticity of listening and speaking strategies can be observed. But these types of effects almost always appear to be influenced by, and adapted to, system constraints that are inherent in the underlying biology, physiology, and perceptual and cognitive organization. Therefore, a complete theory of speech production and/or perception should account for the processes and structures that are weakened, amplified, distorted, or destroyed by disorders. In addition, understanding those processes and structures that are changed by disorders can inform an understanding of normal processes and structures. An example of this from the speech production literature concerns the equilibrium-point hypothesis of the control of articulatory movements (Ostry & Munhall, 1985), which has figured prominently in certain dynamical systems modeling efforts (Saltzman

& Munhall, 1989). The hypothesis holds that parameterization of articulator stiffness plays a major role in the determination of movement durations (e.g., Ostry & Munhall, 1985), which might lead one to expect very fast movements in neurological disorders characterized by elevated muscle tone, such as the spasticity of cerebral palsy or acquired upper-motoneurone disease, or the rigidity associated with Parkinson disease. The articulatory movements in these disorders, however, are actually slower than normal (Weismer, 1997). This result is clearly a case where notions tested (and largely confirmed) in normal speakers emerge as inadequate for a generalized theory of speech production behavior, suggesting that changes are needed in the model.

Consideration of the data on articulatory movements in these neurologically-disordered individuals might indicate ways in which the equilibrium hypothesis needs to be modified or embedded in a broader, generalized theoretical perspective.

Having asserted the goal of complete generalization of explanations, it must be acknowledged that research involving clinical disorders does have pitfalls and challenges for scientific methods. A main challenge comes from requirements of designs for scientific experiments, that is, that there be random assignment of study participants to experimental conditions (Campbell & Stanley, 1963). It is important to be clear about the status of this requirement when considering results from studies involving individuals with clinical labels, such as "deaf" or "dysarthric". Below is a brief discussion of the issue.

1.1. *Experimental designs*

Effective experimental design requires control over independent variables, as well as the random selection and assignment of study participants to conditions. With those requirements met, random error variance (variation not due to the controlled experimental factors) can be assumed. Statistical hypothesis testing uses estimates of the random error to test for the significance of the experimental factors. When experimental designs involve selecting individuals based on their clinical status, the experiment is considered *quasi-experimental* (even when the conditions or tests are administered in a factorial manner, and the statistical analyses are ones such as ANOVA or ANCOVA) (Campbell & Stanley, 1963). The reason for quasi-experimental status is that the possibility exists that the error variance is meaningful. That is, some factors not directly controlled in the experiment, associated with the difference in status between the experimental groups, contributes to the error variance. For example, if deaf children were compared with hearing children on a visual perception task, it is possible that differences in their educational training (not their hearing status) could affect the outcome of the comparison between them and the hearing group. It would be correct to assert that there was a correlation between group assignment and group performance; however, it could be wrong to infer that the difference is due to deafness *per se*. One solution to this problem is to simulate clinical disorders in otherwise normal individuals (e.g., Lum & Braida, 2000). The goal of this approach is to replicate characteristics of the clinical disorder without the potential occurrence of correlated, uncontrolled factors.

Many studies are conducted in communication sciences with grouping based on clinical labels, which renders the research designs quasi-experimental (Campbell & Stanley, 1963). Obviously, the data emerging from such studies are necessary for a full understanding of the disordered phenomena, and are no less scientific for being collected in a quasi-experimental framework.

1.2. *Identification of study participant*

Caramazza (1986) has asserted that, for the case of work in neurolinguistics with brain-damaged patients, there is a significant problem associated with using data from disordered individuals to explain normal processes. The problem lies with uncertainty as to the true nature of the disorder. Caramazza argues that grouping study participants is only justified when it can be known with certainty that all of the participants have experienced the same brain damage. If the goal of the research is to explain how damage perturbs a system, but experimentation involves isolating the damage, then the best that can be done, according to Caramazza, is single participant designs tested against a model. That is, grouping is never justified until the nature of the disorder has been understood (see Kent *et al*., 2000; Katz, 2000). Arguably, this is less of a problem for some disorders than for others. The isolation of damaged cortical structures in relation to specific syntactic processes may be more problematic for grouping study participants than is, for example, the study of adult onset deafness involving only the peripheral auditory system. But Caramazza's point is well taken and should be kept in mind in interpreting results involving clinical disorders, particularly when study participants are grouped on the basis of clinical labels.

1.3. *Types of individual differences*

The requirement of randomly distributed individual variation for true experimental designs (Campbell & Stanley, 1963) can be accommodated fairly easily when the study participants come from a group such as putatively normal undergraduates. We have already acknowledged that grouping based on descriptions of clinical disorders makes it difficult to meet this requirement. On the other hand, certain types of individual differences can be the focus of interest in working with clinical disorders, and in testing the application of theories derived from data collected from normal individuals. Therefore, here we present a simple taxonomy of individual differences and their status in research.

Observations of differences among individuals in performing a task (such as speech perception or production) can (1) result from factors irrelevant to the experimental question, (2) be part of an overall associational pattern (which may or may not be relevant to the task itself), and/or (3) be caused by theoretically interesting variation in the aspect of the system under investigation. The first type of individual difference is typically assumed to be incorporated in the error variance when study participants come from the same population and are randomly assigned in an experimental design. For example, irrelevant (trivial or uninteresting) differences might be due to age, or levels of arousal or fatigue in a group of young adults.

The second type of individual difference—an overall associational pattern—is typically what is used for describing and categorizing individuals and/or groups. Establishing that there are reliable associational patterns in a group of individuals is an important descriptive stage clinically and in basic science. For example, level of hearing impairment in infancy is generally associated with rate of language and speech production development (Downs & Gerkin, 1988). Children can be grouped as potentially exhibiting speech production deficits *vs.* having normal speech production by considering their audiological status. However, as noted above, once associational patterns are used to group study participants, the important property of random selection has been lost. That is, no longer

is it possible to assume that the uncontrolled individual variation is randomly distributed across groups.

Interest in the third type of individual variation—variation due to an underlying theoretically interesting factor—is what motivates scientific study with clinical populations. In clinical research, the disorder/impairment itself is the primary interest; describing the disorder/impairment is the goal of the research. Clinical research usually does not have as its goal generalization beyond the disorder/impairment. Research with clinical populations whose goal is furthering basic knowledge about underlying mechanisms starts from a theory and asks how particular characteristics of a clinical population or individuals within the population might be related to some hypothesis generated from the theory. Individuals are selected for study, because they potentially possess certain characteristics that may lead to further understanding of the speech perception and/or speech production system. For example, work with individuals who have cochlear implants to restore some degree of hearing lost to deafness affords a unique opportunity to investigate the role of auditory feedback in speech production (Perkell *et al*., 2000). Until the development of the cochlear implant, it was not possible to investigate within the same individual the effects of alternating between hearing and nonhearing (i.e., deaf) conditions.

1.4. *Scientific approaches to individual differences due to clinical disorders*

A standard factorial type of experimental design that includes a sufficiently large number of subjects without clinical labels is of course not the only way to gather information under "true" experimental conditions. Another approach is to model the disorder computationally (e.g., MacEachern, 2000). Yet another approach is to use speech synthesis or resynthesis techniques to modify natural speech samples so that normal speech achieves the perceptual or acoustic attributes of disordered speech or *vice versa* (cf. Gerratt & Kreiman, 2000; Kent *et al*., 2000).

One approach to experimental design with participants who have been identified clinically is to use each individual statistically as a replicate in the experiment. There is a history of this approach in the study of normal speech production (e.g., Ostry & Munhall, 1985; Hertrich & Ackermann, 1995), and this has been extended to the study of persons with speech disorders (e.g., see Katz, 2000; Tjaden, 1999). Then the question becomes the reliability with which the experiment can be replicated across individuals. A factor is generalizable when it is reliable across replicates, independent of whether the individuals have received clinical labels (and, arguably, whether they have been randomly assigned to conditions). A different approach is to develop an explicit model and test the model against results that have been obtained from individuals with clinical labels (Chen *et al*., 2000; Perkell *et al*., 2000). When components of a model correspond to dysfunctioning components of a talker or listener, then a clinical disorder "is an opportunity to deepen our knowledge of how this component might function in the overall speech production [or perception] processes" (Chen *et al*., 2000). Furthermore, because disorders frequently result in more extreme component settings, opportunities to establish or strengthen evidence for functional relationships across individuals arise, in both normal subjects (Chen *et al*., 2000) and those with clinical labels.

Another approach to overcoming the random assignment problem is to use regression analyses. Here the goal is to account for the dependent factor(s) in terms of measures applied to both subjects with and without communication disorders. That is, if clinical

disorders amplify or attenuate the phenomena under study, it should be possible to incorporate all study participants along the same measured dimensions. An example of this kind of analysis is found in Weismer, Tjaden & Kent (1995), who related formant frequency variation to speaking rate fluctuation in individual speakers with apraxia of speech. The goal of this study was to better understand the disruption of coarticulatory processes in individuals with presumed speech motor programming deficits. A finding of greater variability in these processes among individuals with apraxia revealed the putative effect (Saltzman & Munhall, 1989) of rate variation on articulatory overlap, which is not easily observed in the clinically unlabeled (i.e., normal) subjects.

Alternatively, one might use regression to estimate the variance associated with the clinical entity. For example, Bernstein, Demorest & Tucker (2000) investigated visual speech perception scores with sentence stimuli in deaf ($n = 72$) and hearing ($n = 96$) adults. Potential predictors included group (deaf *vs.* hearing), phoneme identification in nonsense syllables, isolated word identification (from a different set of stimuli), and the interactions of group with each of the remaining predictors. Only group and isolated word performance were significant predictors of sentence performance. Phoneme identification in syllable performance did not contribute significantly to the prediction of sentence performance, once word performance was taken into account.[2] This analysis showed that individual variation in lipreading sentences was mostly accounted for by the ability to lipread isolated words, and considerably less variation was accounted for by being labeled "deaf" *vs.* "hearing".

2. Conclusion

Work with clinical disorders challenges our thinking about science in general and speech perception and production in particular. Work restricted to nonclinical populations can lull us into thinking that we know more than we do. Communication sciences can benefit from regarding data from disordered speakers and listeners as having the same value as data from normal speakers and listeners in the development of models and theories.

The papers in this issue provide an initial glimpse of how basic scientific issues can be investigated fruitfully in subjects with communication disorders. It also shows that such work may contribute to a deeper understanding of those disorders, which is a major long-term objective of research funding. We hope that the material collected here can serve as inspiration to other scientists who are in the process of developing models and theories of speech perception and production.

3. Author notes

This Special Issue was an outcome of a day-long workshop at the Meeting of the Acoustical Society of America (December, 1997) at which presenters were asked to respond to the following questions: (1) How does research with clinical populations uniquely contribute (or not contribute) to increased knowledge in your area? (2) What

[2]These results do not mean that phonetic perception is not important in explaining individual differences in lipreading sentences. They mean that the word identification measures account for the same variance as the syllable identification scores and for additional variance not accounted for by the syllable scores.

are the particular methodological issues/pitfalls that arise in your research with clinical populations? and (3) What are the special data analysis problems you face?

This research was supported in part by grants to the authors from NIH/NIDCD (DC02107 and DC00698, Bernstein; DC00319 and DC03723, G. Weismer).

References

Bernstein, L. E., Demorest, M. E. & Tucker, P. E. (2000) Speech perception without hearing, *Perception and Psychophysics*, **62,** 233–252.

Campbell, D. T. & Stanley, J. C. (1963) *Experimental and quasi-experimental designs for research*. Chicago: Rand McNally.

Caramazza, A. (1986) On drawing inferences about the structure of normal cognitive systems from the analysis of patterns of impaired performance: the case for single-patient studies, *Brain and Cognition*, **5,** 41–66.

Chen, M. Y., Stevens, K. N., Hong-Kwang, J. K. & Chen, H. (2000) Contributions of the study of disordered speech to speech production models, *Journal of Phonetics*, **28,** 303–312.

Downs, M. P. & Gerkin, K. P. (1988) Early identification of hearing loss. In *Handbook of speech-language pathology and audiology* (N. J. Lass, L. V. McReynolds, J. L. Northern & D. E. Yoder, editors), pp. 1188–1201. Toronto: B.C. Decker, Inc.

Gasper, P. (1991) Causation and explanation. In *The philosophy of science* (R. Boyd, P. Gasper & J. D. Trout, editors) pp. 289–297. Cambridge, MA: MIT Press.

Gerratt, B. R. & Kreiman, J. (2000) Theoretical and methodological development in the study of pathological voice quality, *Journal of Phonetics*, **28,** 335–342.

Hertrich, I. & Ackermann, H. (1995) Coarticulation in slow speech: Durational and spectral analyses, *Language and Speech*, **38,** 159–187.

Katz, W. F. (2000) Anticipatory coarticulation and aphasia: implications for phonetic theories, *Journal of Phonetics*, **28,** 313–334.

Kent, R. D., Kent, J. F., Weismer, G. & Duffy, J. R. (2000) What dysarthrias can tell us about the neural control of speech, *Journal of Phonetics*, **28,** 273–302.

Lum, D. S. & Braida, L. D. (2000) Perception of speech and non-speech sounds by listeners with real and simulated sensorineural hearing loss, *Journal of Phonetics*, **28,** 343–366.

MacEachern, M. R. (2000) On the visual distinctiveness of words in the English lexicon, *Journal of Phonetics*, **28,** 367–376.

Ostry, D. J. & Munhall, K. G. (1985) Control of rate and duration of speech movements, *Journal of the Acoustical Society of America*, **77,** 640–648.

Perkell, J. S., Guenther, F. H., Lane, H., Matthies, M. L., Perrier, P., Vick, J., Wilhelms-Tricarico, R. & Zandipour, M. (2000) A theory of speech motor control and supporting data from speakers with normal hearing and with profound hearing loss, *Journal of Phonetics*, **28,** 233–272.

Saltzman, E. & Munhall, K. (1989) A dynamical approach to gestural patterning in speech production, *Ecological Psychology*, **1,** 333–382.

Tjaden, K. (1999) Can a model of overlapping gestures account for scanning speech patterns?, *Journal of Speech, Language, and Hearing Research*, **42,** 604–617.

Weismer, G. (1997) Motor speech disorders. In *The handbook of phonetic sciences* (W. J. Hardcastle and J. Laver, editors), pp. 191–219. Oxford, U.K.: Blackwell.

Weismer, G., Tjaden, K. & Kent, R. D. (1995) Can articulatory behavior in motor speech disorders be accounted for by theories of normal speech production?, *Journal of Phonetics*, **23,** 149–164.