4. It's a Poor Sort of Memory That Only Works Backwards

John C. Rosenbek

Darley had only a little less trouble getting apraxia of speech admitted into the galaxy of neurogenic speech-language disorders than did Galileo the four moons of Jupiter into that of the heavenly bodies. Galileo was summoned before the Inquisition because Ptolemy and Aristotle had placed an unmoving Earth at the universe's imagined center and because the telescope and optics were new and heretical. After all, "God is light." Darley was resisted by those who believed that aphasia and dysarthria were worlds enough and that apraxia of speech was neither separate nor real but simply the result of misperception and misclassification. But whereas many were unwilling even to look through Galileo's telescope, especially if it were tilted toward the sky, nearly all of Darley's foes happily turned their ears toward the speech of a variety of brain-damaged speakers.

Indeed, it was perhaps the method—broad phonetic transcription in nearly all the early studies—more than any orientation or bias, that sparked the polemics about the existence and nature of apraxia of speech. Broad phonetic transcription is a gross tool. Its resolution is relatively poor and it is nearly defenseless against what might be called the tyranny of belief. Darley and his students posited that apraxia of speech was a motor speech disorder involving programming and affecting sound selection and ordering. In their view, speech sound substitutions were the conceptually comfortable errors to be expected from such a disorder. The early studies, most of them by Darley's students, confirmed the expectation (cf. Johns & Darley, 1970). Researchers committed to the notion that apraxia of speech was just another form of aphasia were elated. Substitutions are more readily explained by a variety of primarily phonological linguistic theories (cf. Martin, 1974) than by motor theories. Their own data (cf. Klich, Ireland, & Weidner, 1979) confirmed a high proportion of speech sound substitutions. Apraxic speakers made errors that could be explained linguistically; therefore, apraxia of speech was an aphasia.

Lewis Thomas, in an essay called "Alchemy" (1983), observes that science works because scientists "work, and *work together.* They become excited and exasperated, they exchange their bits of information at a full shout, and, the most wonderful thing of all, they keep *at* one another" (p. 33). Certainly the researchers interested in phonology, phonetics, and aphasia kept at one another about apraxia of speech. Clinical Aphasiology Conferences (CACs) offered a podium for both the shouting and the "keeping at" that Thomas describes as part of the scientific method. Occasionally the shouts turned to threats, and once several participants felt compelled to walk out of a CAC meeting during a heated discussion of apraxia. Usually, however, the shouting was more frustrated than mean.

Equally often, people with different visions worked together, as when Goodglass (1975), as an invited speaker to CAC, humbly and with numerous caveats stated his position on speech errors in aphasia this way: "I regard any organic breakdown of articulation as aphasic, provided that it varies with the communicative intent of the speaker" (p. 28). He said that the phonological errors of conduction and those of Broca-aphasic speakers reflected disruptions at different levels of the phonology, but both varied with intent and both were aphasic. Some of the data interpreted as supporting his position were presented at CAC as well. Bowman, Althoff, and Anderson (1982) reported being able to identify "linguistic regularities" in the errors of apraxic speakers that "challenge the concept of phonemic variability as supporting a motor programming interpretation of apraxia" (p. 242). The discussion that followed this paper seems from the summary to have been benign, but at the next conference Mlcoch and Beach (1983) argued, on the basis of their own process analysis of speech errors by two apraxic talkers, that apraxic articulatory behavior "did not appear to be rule-governed; at least not in a phonological sense" (p. 41).

Shankweiler and Harris (1966) warned researchers early on about the inability of broad phonetic transcription to do more than highlight a condition's major features. Certainly, they argued, it was inadequate to attempt to provide explanations about underlying pathophysiology. Unfortunately, most of the early researchers seem to have ignored Shankweiler and Harris's warnings because much of the early "keeping at" each other was over explanation. Perhaps, too, the fact that so many of the researchers were new and struggling to establish themselves was an influence as well. Wallace Stegner, in *Joe Hill* (1980), alludes to ideas strong as bars, by which he meant prison bars. But the young researchers in apraxia of speech seem, at least all these years later, to have been victimized by bars of a different sort, ones they were more likely to threaten with than to stare out from between.

Just as telescopes quickly improved over Galileo's original model (with Galileo's help), so too did methodology improve in apraxia of speech research with Darley's help. The results of a variety of acoustic and so-

called physiological measures of apraxic speech and other apraxic movements began appearing at CAC and other places. In 1975, Bauman, Waengler, and Prescott reported one of the earliest acoustic studies of apraxic speech at the American Speech-Language-Hearing Association's national meeting. Perhaps the earliest physiological study, at least in North America, was Shankweiler, Harris, and Taylor's electromyographic evaluation of two apraxic speakers, which appeared in 1968. Their rationale included this: "Precise information regarding which gestures of speech are defective and the physiologic basis of the defects are requirements for rational therapy" (p. 1). In 1976 Keatley and Pike published the findings of automated pulmonary testing of five apraxic speakers. No group discussion of that paper was provided, but both the methodology and the focus—respiratory function—may well have stirred the audience. In 1976 apraxia of speech was still considered primarily an articulatory disorder, and the articulatory functions of the respiratory system may not have been widely appreciated by aphasiologists.

Subsequent developments are well known to clinical scientists interested in apraxia of speech, by whatever name it is called. Acoustic studies proliferated (see Wertz, LaPointe, & Rosenbek, 1984, and McNeil & Kent, in press, for reviews). CAC considered some of the data, including those presented by Square and Mlcoch (1983); Colson, Luschei, and Jordan (1986); and Strand and McNeil (1987). Reports of a variety of physiological measures began appearing as well (cf. Itoh, Sasanuma, Hirose, Yoshioka, & Ushijima, 1980; Itoh, Sasanuma, & Ushijima, 1979). In the *Proceedings*, these included studies of surface electromyography (EMG) from the lips (Hough & Klich, 1987), measures of maximum strength (Crary, Hardy, & Williams, 1985), and, of course, the programmatic studies of lip kinematics and position and force control by McNeil and his colleagues (McNeil & Adams, 1991; McNeil, Caligiuri, & Rosenbek, 1989). One of the earliest physiological studies was by Fromm, Abbs, McNeil, and Rosenbek, who reported simultaneous auditory perceptual and lip kinematic findings for three apraxic speakers in the 1982 *Proceedings*. Even though it appeared 14 years after the benchmark study by Shankweiler, Harris, and Taylor, it was considered a seminal study. The authors, like their predecessors, argued that simultaneous measurement at several levels of observation "should lead to a more refined understanding of a disorder that has been subject to controversy and ambiguity" (p. 261). Unfortunately, replication of at least a portion of these findings has been impossible (Forrest, Adams, & McNeil, 1990).

Improvements in auditory perceptual analysis took the form of narrow phonetic transcription. CAC published one of the earliest, an analysis of apraxic speakers with different loci of lesion by Square, Darley, and Sommers (1982). These authors noted that "distortions were observed to be the predominant phonetic error" (p. 248). A more elaborate analysis by

Odell and her colleagues (Odell, McNeil, Rosenbek, & Hunter, 1990) confirmed the high proportion of sound distortion errors in purely apraxic speakers. These studies changed the description of apraxic speech, but they hardly stilled the clamor about the nature of apraxic errors.

Silence or murmured agreement would have been too much to expect. Procedures and instruments, no matter how elegant or complicated, do not necessarily resolve conflicts over interpretation and classification. Most of the early acoustic data, for example, were interpreted as demonstrating a phonemic deficit in all aphasic speakers (cf. Blumstein, Cooper, Zurif, & Caramazza, 1977). As Ziman, in his contribution to a book on the philosophy of science (Ziman, 1980), said, "Scientists . . . tend to look for, and find, in Nature little more than they believe to be there" (p. 37). One might add: The more sophisticated the tool that is looked through, the greater the confidence in the vision. Nonetheless, some positions have shifted. Even researchers with radically different political-scientific positions, such as Blumstein (Blumstein & Baum, 1987), can now write, "It is assumed that the articulatory disorder of Broca's aphasia corresponds roughly to the impairment known as apraxia of speech and will be treated as comparable disorders" (p. 7).

Not only have positions shifted but sources of exasperation and debate have changed. A change in the debate is visible in the discussion following Square et al.'s (1982) paper reporting the high number of sound distortions in apraxia of speech. Don't the distortions, it was argued, suggest that the apraxic talkers were also dysarthric? For after all, common wisdom has it (in some quarters even now) that a predominance of distortions means dysarthria.

Square et al.'s position was not idiosyncratic. Itoh and colleagues (cf. Itoh & Sasanuma, 1984) nominated speech sound distortion as the core sign of apraxia of speech. Data presented at CAC and elsewhere and at all levels of analysis support their nominee. In addition, some of the physiological data (Freeman, Sands, & Harris, 1978) suggest that the reason for the distortions, at least at one level of explanation, is dyscoordination across muscle groups and functional components of the motor speech mechanism (Netsell, 1986). The purposes of this new debate are not to deny apraxia of speech and dysarthria's separateness or to condemn some researchers for apostasy. The purposes are to improve and expand what we know. One result is that we are being required to reexamine our definitions and descriptions of these labels. Data emerging from the debate, including the presence of clinically significant dysphagia in some apraxic persons (Robbins & Levine, 1988), make continued examination of the underlying pathophysiology and confirming signs of both apraxia of speech and dysarthria (as well as dysphagia) more urgent as well. A 1974 CAC paper by Berry and his colleagues makes interesting reading in this regard.

Clinicians were as interested in treating apraxia of speech (21 treatment articles appear in the *Proceedings*) as the clinical scientists were in identifying its underlying nature. The issue of differentiating aphasia and apraxia of speech managements and treating both when they occurred together was addressed in a 1979 round table (Keith, 1979). Wiedel (1976) emphasized use of language in the treatment of apraxia. Florance, Rabidoux, and McCauslin (1980) developed an environmental manipulation approach to enhance carry-over. Modality influences (Berry & Newhoff, 1978; Simmons, 1978, 1980); the influence of pauses and rehearsal (Bugbee & Nichols, 1980; LaPointe & Horner, 1976; Warren, 1977; Wilson, 1977); and the influences of stress (Tonkovich & Marquardt, 1977), prolonged speech (Southwood, 1987), intoning (Hyland & McNeil, 1987), EMG-assisted biofeedback (McNeil, Prescott, & Lemme, 1976), a Handi Voice® (Rabidoux, Florance, & McCauslin, 1980), and gestures (Dowden, Marshall, & Tompkins, 1981; Rosenbek, Collins, & Wertz, 1976) were all reported. So were special programs such as Multiple Input Phoneme Therapy (Stevens & Glasner, 1983), Multiphonemic Articulation Therapy (Holtzapple & Marshall, 1977), PROMPT (Square, Chumpelik, Morningstar, & Adams, 1986), and special task continua unblessed by labels or acronyms.

Treatment in aphasiology proceeds along a course charted primarily by enlightened clinical experience and general treatment principles. Reading the CAC treatment literature gives one the impression that clinicians were only slightly attentive to the arguments over apraxia of speech's nature beyond the relatively simple notion—so simple, in fact, that no very sophisticated analysis was necessary to establish it—that some left-hemisphere-damaged patients have inordinate difficulty with verbal expression and that systematic practice of certain sorts seems to help. This is not to say that treatment in aphasiology, and especially in apraxia, is atheoretical, nor is it to deny that treatments can be enhanced by nontreatment data. It does imply, however, that the concerns of the linguists, physiologists, and speech scientists were not necessarily the concerns of clinicians. It suggests, as well, that not all data are or should be useful to clinical practice and that some of the most useful are generated by the treatment itself.

It would be cockeyed to deny that the path of treatment can be altered, even corrected, by discoveries or the resolution of disagreements about concepts in a related field of science. Medication for the treatment of Parkinson's disease is an example. Such a transfer from what might be called more basic science to clinical practice is not inevitable, however, and when it does occur, it does so slowly. Just as often, the one responsible for its transfer goes unacknowledged by peers (Jonas Salk did not receive a Nobel Prize) despite considerable popular acclaim. It probably is good for the science to be free to ask questions without worrying about what the clinicians and technicians think. It is also good for the clinic.

Perfectly acceptable treatments may be needlessly sacrificed to the relatively more attractive interpretations of laboratory data just as otherwise excellent basic data may be distorted while being squeezed for their clinical significance. Most important, and perhaps this is the lesson of CAC's publications in apraxia rehabilitation, clinical practice can be its own science and its data can be useful to other sciences. Treatment can be enriched by the concepts and discoveries of others, but it does not require them for its support or need them for its validation.

Lewis Carroll (1980) has the White Queen tell Alice: "It's a poor sort of memory that only works backwards." Unlike Alice, who resisted trying to recall future events, I succumbed. Probably it is my age and lack of Alice's wisdom. Medawar (1979) seemed to have me at least partly right when he said that his view of older scientists "is of a committee of gray heads, all confident in the rightness of their opinions and all making pronouncements about the future developments of a kind known by philosophers to be intrinsically unsound" (p. 53). With apologies, then, here is my unsound recollection of the future.

One development has already been alluded to. The shouting over the differences between apraxia of speech and aphasia will be replaced, if not by shouts, then by urgent words over the diagnostically important differences between apraxia of speech and dysarthria and over the best tools and stimuli for revealing those differences. Distortions were always included among dysarthria's core signs. Now distortion appears to be a core sign of at least one form of apraxic speech as well. So how are dysarthria and apraxia of speech to be differentiated? One experimentally verifiable hypothesis is this: To the degree that distortions are the sole articulatory error type, the diagnosis of dysarthria is the one of best fit. To the degree that distortions are accompanied by distorted substitutions and a lesser number of substitutions, the diagnosis of apraxia of speech is the one of best fit. Also testable is the hypothesis that the distortions themselves differ. Distinguishing apraxic from apraxic-dysarthric speakers will continue as a challenge. Data on prosodic disturbances; nonspeech-movement integrity, including swallowing; localization; and number of nervous system lesions will help. Helpful, as well, will be experimental concern with the challenges of response variability in both apraxic and dysarthric speakers. We must stop acting as if one hypothesis, one test, one (or even three) repetitions, one study, and one conclusion are enough. Munhall's chapter (1989) on variability should be required reading for present and future researchers. The same experimental rigor will help to increase our confidence about the existence of clinically significant subtypes of apraxic speech as well. One final comment on the future of diagnosis. It may well turn out that what apraxic speakers do after they have begun to talk is more interpretable, and therefore more important diagnostically, than what they do to begin

talking. Difficulty with initiation, like perseveration, is too nearly ubiquitous to be a powerful differential diagnostic sign.

The adversaries in the early debates over apraxia of speech began primarily with models borrowed from behavioral neurology and linguistics. It is remarkable that the disorder whose name and nature were borrowed from the discussions of limb movement abnormalities largely by European, 19th-century behavioral neurologists and whose characteristics were interpreted primarily with methods and concepts created by scholars ignorant about abnormal speech and language survived into the present day. The energy generated by the term and by the hypothesized differences between *phonetic* and *phonemic* may have shed as much illumination on apraxia of speech as it is likely to, however. A new generation of studies is likely to be guided by models of motor control and by more elaborate linguistic models. Selected models have been described by Rosenbek, Kent, and LaPointe (1984); by Kent and McNeil (1987); and by McNeil and Kent (in press). One advantage of some of these is that they do not fractionate the cognitive, linguistic, and sensorimotor interactions responsible for human behavior, including communicative behavior. They do permit such reduction, however, and even guide it. As a result, the present nosology in the neuropathologies of speech and language is unlikely to be abandoned and may even be strengthened by the delineation of at least a few relatively strong syndromes. New models may help us explain unique and shared signs in the neuropathologies of speech and language. For example, recent evidence (McNeil, Liss, Tseng, & Kent, 1990) confirms similarities in the influences of rate on timing in conduction and apraxic speakers. Such data do not add bulk to one or the other group of participants in the old battles over the similarity, and therefore needless redundancy, of the terms *conduction aphasia* and *apraxia of speech*. They do provide ways of thinking about why dissimilar conditions may have some similar signs. The only casualty of the clash between old and future models is likely to be our lesion-label notion in which all of a speaker's abnormalities are assumed to be part of the label earned when the locus of lesion or disease entity (especially in the dysarthrias) has been established.

Treatment articles on apraxia of speech are not appearing at the same rate as previously. Probably the rate will not increase. Apraxia of speech is a relatively rare condition and one that is likely to improve spontaneously (Mohr, 1980). Treatment discussions of rare conditions are likely to be rare as well. In addition, clinicians seem to feel confident of their skills, predictions, and efficacy when faced with the infrequent apraxic talker. Many may even feel that apraxia treatment is pretty prosaic stuff for anyone but the neophyte. While I cannot support it with data, my feeling is that many clinicians were surprised, and perhaps even amused, by the decades-long struggle over the nature of apraxia of speech. Some may have wondered

what they were missing. Probably they did not miss much. While they may not have understood the pathophysiology of apraxia at the level that Shankweiler et al. (1968) or Abbs and Rosenbek (1985) said was essential, they understood human learning and responses well enough to make apraxic folks talk better.

The future of apraxia of speech treatment will not be its past, however. Future practitioners may even challenge the early arguments about how different apraxia and aphasia treatments are. Those differences were never fully developed and seem more like illusions to support apraxia and aphasia's putative differences than empirically based treatment principles. I think a future generation of clinical practitioners may find that only the stimuli differ significantly. The principles and the emphases on pragmatics and cognitive, linguistic, and sensorimotor interactions are similar or the same. Treatment studies of that hypothesis will be useful and forthcoming as will studies and discussions of the best measures of treatment efficacy with apraxic talkers.

William Kennedy, the author of *Ironweed* (1984) and other novels, said that a story begins with experience and is sustained by emotion and ideas. But only the unknown or the vaguely familiar will see it to the end. Literature, it seems to me, is not much different from conferences, associations, and other creative human enterprises. CAC's first 20 years were filled with experience, emotion, and ideas. My memory of its future is full of the surprising, the only vaguely familiar, and, best of all, the totally unfamiliar. And although that memory sometimes wavers and even blurs, it looks to me as if some of the surprises will come in the study of apraxia of speech. We have come far since Galileo, but the sky has not lost its mystery or romance. Neither has apraxia of speech since Darley.

REFERENCES

Abbs, J. H., & Rosenbek, J. C. (1985). Some motor control perspectives on apraxia of speech and dysarthria. In J. Costello (Ed.), *Speech disorders in adults* (pp. 21–57). San Diego, CA: College-Hill Press.

Bauman, J. A., Waengler, H. H., & Prescott, T. E. (1975). *Durational aspects of continuous speech: Comparative measurements based on vowel and consonant productions by normal and apraxic talkers.* Paper presented at the American Speech-Language-Hearing Association, Washington, DC.

Berry, W. R., Martin, A. D., & Rosenbek, J. C. (1974). Referral: "54 year-old male with severe expressive aphasia; please evaluate and treat as indicated." In B. Porch (Ed.), *Clinical aphasiology: Conference proceedings* (pp. 38–54). Albuquerque, NM: Veterans Administration Medical Center.

Berry, W. R., & Newhoff, M. (1978). The treatment of developmental apraxia of speech: A case report. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 8, pp. 295–303). Minneapolis, MN: BRK Publishers.

Blumstein, S. E., & Baum, S. (1987). Consonant production deficits in aphasia. In J. H. Ryalls (Ed.), *Phonetic approaches to speech production in aphasia and related disorders* (pp. 3–21). Boston: Little, Brown, a College-Hill Publication.

Blumstein, S. E., Cooper, W. E., Zurif, E. B., & Caramazza, A. (1977). The perception and production of voice-onset time in aphasia. *Neuropsychologia, 15,* 371–383.

Bowman, C., Althoff, L., & Anderson, N. (1982). Error patterns in apraxia of speech. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 12, pp. 239–244). Minneapolis, MN: BRK Publishers.

Bugbee, J., & Nichols, A. (1980). Rehearsal as a self-correction strategy for patients with apraxia of speech. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 10, pp. 279–284). Minneapolis, MN: BRK Publishers.

Carroll, L. (1980). *The complete illustrated works of Lewis Carroll.* London: Chancellor Press.

Colson, K. A., Luschei, E. S., & Jordan, L. S. (1986). Perceptual and acoustic analyses of stress patterning in apraxic and normal speech. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 16, pp. 281–290). Minneapolis, MN: BRK Publishers.

Crary, M. A., Hardy, T., & Williams, W. N. (1985). Aphemia with dysarthria or apraxia of speech? In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 15, pp. 113–125). Minneapolis, MN: BRK Publishers.

Dowden, P. A., Marshall. R. C., & Tompkins, C. A. (1981). Amer-Ind sign as a communicative facilitator for aphasic and apractic patients. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 11, pp. 133–140). Minneapolis, MN: BRK Publishers.

Florance, C., Rabidoux, P., & McCauslin, L. (1980). An environmental manipulation approach to treating apraxia of speech. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 10, pp. 285–293). Minneapolis, MN: BRK Publishers.

Forrest, K., Adams, S., & McNeil, M. R. (1990). *Perioral EMG activity in aphasic, apraxic, and dysarthric speakers.* Paper presented at the Clinical Dysarthria Conference, San Antonio, TX.

Freeman, F. J., Sands, E. S., & Harris, K. S. (1978). Temporal coordination of phonation and articulation in a case of verbal apraxia: A voice onset time study. *Brain and Language, 6,* 106–111.

Fromm, D., Abbs, J., McNeil, M., & Rosenbek, J. (1982). Simultaneous perceptual-physiological method for studying apraxia of speech. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 12, pp. 251–262). Minneapolis, MN: BRK Publishers.

Goodglass, H. (1975). Phonological factors in aphasia. In R. H. Brookshire (Ed.), *Clinical aphasiology: Conference proceedings* (pp. 28–44). Minneapolis, MN: BRK Publishers.

Holtzapple, P., & Marshall, N. (1977). The application of multiphonemic articulation therapy with apraxic patients. In R. H. Brookshire (Ed.), *Clinical aphasiology: Conference proceedings* (pp. 46–58). Minneapolis, MN: BRK Publishers.

Hough, M. S., & Klich, R. J. (1987). Effects of word length on lip EMG activity in apraxia of speech. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 17, pp. 271–276). Minneapolis, MN: BRK Publishers.

Hyland, J. D., & McNeil, M. R. (1987). The effects of an intoning therapy on the speech of a developmentally apraxic adult. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 17, pp. 288–299). Minneapolis, MN: BRK Publishers.

Itoh, M., & Sasanuma, S. (1984). Articulatory movements in apraxia of speech. In J. C. Rosenbek, M. R. McNeil, & A. E. Aronson (Eds.), *Apraxia of speech: Physiology, acoustics, linguistics, management* (pp. 135–165). San Diego, CA: College-Hill Press.

Itoh, M., Sasanuma, S., Hirose, H., Yoshioka, H., & Ushijima, T. (1980). Abnormal articulatory dynamics in a patient with apraxia of speech: X-ray microbeam observation. *Brain and Language, 11,* 66–75.

Itoh, M., Sasanuma, S., & Ushijima, T. (1979). Velar movements during speech in a patient with apraxia of speech. *Brain and Language, 7,* 227–239.

Johns, D. F., & Darley, F. L. (1970). Phonemic variability in apraxia of speech. *Journal of Speech and Hearing Research, 13,* 556–583.

Keatley, M., & Pike, P. (1976). The automated pulmonary function laboratory: Clinical use in determining respiratory variations in apraxia. In R. H. Brookshire (Ed.), *Clinical aphasiology: Conference proceedings* (pp. 98–109). Minneapolis, MN: BRK Publishers.

Keith, R. L. (1979). Treatment of aphasia with coexisting apraxia. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 9, pp. 300–308). Minneapolis, MN: BRK Publishers.

Kennedy, W. (1984). *Ironweed.* New York: Penguin Books.

Kent, R. D., & McNeil, M. R. (1987). Relative timing of sentence repetition in apraxia of speech and conduction aphasia. In J. H. Ryalls (Ed.), *Phonetic approaches to speech production in aphasia and related disorders* (pp. 181–220). Boston: Little, Brown, a College-Hill Publication.

Klich, R. J., Ireland, J. U., & Weidner, W. E. (1979). Articulatory and phonological aspects of consonant substitutions in apraxia of speech. *Cortex, 15,* 451–470.

LaPointe, L. L., & Horner, J. (1976). Repeated trials of words by patients with neurogenic phonological selection-sequencing impairment (apraxia of speech). In R. H. Brookshire (Ed.), *Clinical aphasiology: Conference proceedings* (pp. 261–277). Minneapolis, MN: BRK Publishers.

Martin, A. D. (1974). Some objections to the term *apraxia of speech. Journal of Speech and Hearing Disorders, 39,* 53–64.

McNeil, M. R., & Adams, S. (1991). A comparison of speech kinematics among apraxic, conduction aphasic, ataxic dysarthric and normal geriatric speakers. In T. E. Prescott (Ed.), *Clinical aphasiology* (Vol. 19, pp. 279–294). Austin, TX: PRO-ED.

McNeil, M. R., Caligiuri, M. P., & Rosenbek, J. C. (1989). A comparison of labiomandibular kinematic durations, displacements, velocities, and dysmetrias in apraxic and normal adults. In T. E. Prescott (Ed.), *Clinical aphasiology* (Vol. 18, pp. 173–193). Austin, TX: PRO-ED.

McNeil, M. R., & Kent, R. D. (in press). Motoric characteristics of adult aphasic and apraxic speakers. In G. R. Hammond (Ed.), *Advances in psychology: Cerebral control of speech and limb movements.* New York: Elsevier/North Holland.

McNeil, M. R., Liss, J., Tseng, C.-H., & Kent, R. D. (1990). Effects of speech rate on the absolute and relative timing of apraxic and conduction aphasic sentence production. *Brain and Language, 38,* 135–158.

McNeil, M. R., Prescott, T. E., & Lemme, M. L. (1976). An application of electromyographic biofeedback to aphasia/apraxia treatment. In R. H. Brookshire (Ed.), *Clinical aphasiology: Conference proceedings* (pp. 151–171). Minneapolis, MN: BRK Publishers.

Medawar, P. B. (1979). *Advice to a young scientist.* New York: Harper & Row.

Mlcoch, T., & Beach, W. (1983). The efficacy of phonological process analysis for apraxia of speech. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 13, pp. 35–41). Minneapolis, MN: BRK Publishers.

Mohr, J. P. (1980). Revision of Broca aphasia and the syndrome of Broca's area infarction and its implication in aphasia theory. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 10, pp. 1–16). Minneapolis, MN: BRK Publishers.

Munhall, K. G. (1989). Articulatory variability. In P. Square-Storer (Ed.), *Acquired apraxia of speech in aphasic adults* (pp. 64–83). London: Taylor and Francis.

Netsell, R. (1986). *A neurobiologic view of speech production and the dysarthrias.* San Diego, CA: College-Hill Press.

Odell, K., McNeil, M. R., Rosenbek, J. C., & Hunter, L. (1990). Perceptual characteristics of consonant production by apraxic speakers. *Journal of Speech and Hearing Disorders, 55,* 345–359.

Rabidoux, P., Florance, C., & McCauslin, L. (1980). The use of a Handi Voice in the treatment of a severely apractic nonverbal patient. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 10, pp. 294–301). Minneapolis, MN: BRK Publishers.

Robbins, J., & Levine, R. L. (1988). Swallowing after unilateral stroke of the cerebral cortex: Preliminary experience. *Dysphagia, 3,* 11–17.

Rosenbek, J. C., Collins, M. J., & Wertz, R. T. (1976). Intersystemic reorganization for apraxia of speech. In R. H. Brookshire (Ed.), *Clinical aphasiology: Conference proceedings* (pp. 255–260). Minneapolis, MN: BRK Publishers.

Rosenbek, J. C., Kent, R. D., & LaPointe, L. L. (1984). Apraxia of speech: An overview and some perspectives. In J. C. Rosenbek, M. R. McNeil, & A. E. Aronson (Eds.), *Apraxia of speech: Physiology, acoustics, linguistics, management* (pp. 1–72). San Diego, CA: College-Hill Press.

Shankweiler, D., & Harris, K. S. (1966). An experimental approach to the problem of articulation in aphasia. *Cortex, 2,* 277–297.

Shankweiler, D., Harris, K. S., & Taylor, M. L. (1968). Electromyographic studies of articulation in aphasia. *Archives of Physical Medicine and Rehabilitation, 49,* 1–8.

Simmons, N. N. (1978). Finger counting as an intersystemic reorganizer in apraxia of speech. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 8, pp. 174–179). Minneapolis, MN: BRK Publishers.

Simmons, N. N. (1980). Choice of stimulus modes in treating apraxia of speech. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 10, pp. 302–307). Minneapolis, MN: BRK Publishers.

Southwood, H. (1987). The use of prolonged speech in the treatment of apraxia of speech. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 17, pp. 277–287). Minneapolis, MN: BRK Publishers.

Square, P. A., Chumpelik, D. A., Morningstar, D., & Adams, S. (1986). Efficacy of the PROMPT system of therapy for the treatment of acquired apraxia of speech: A follow-up investigation. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 16, pp. 221–226). Minneapolis, MN: BRK Publishers.

Square, P., Darley, F., & Sommers, R. (1982). An analysis of the productive errors made by pure apractic speakers with differing loci of lesions. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 12, pp. 245–250). Minneapolis, MN: BRK Publishers.

Square, P., & Mlcoch, A. (1983). The syndrome of subcortical apraxia of speech: An acoustic analysis. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 13, pp. 239–243, abstract). Minneapolis, MN: BRK Publishers.

Stegner, W. (1980). *Joe Hill.* Lincoln: University of Nebraska Press.

Stevens, E., & Glasner, L. (1983). Multiple input phoneme therapy: An approach to severe apraxia and expressive aphasia. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 13, pp. 148–155). Minneapolis, MN: BRK Publishers.

Strand, E. A., & McNeil, M. R. (1987). Evidence for a motor performance deficit versus a misapplied rule system in the temporal organization of utterances in apraxia of speech. In R. H. Brookshire (Ed.), *Clinical aphasiology* (Vol. 17, pp. 260–270). Minneapolis, MN: BRK Publishers.

Thomas, L. (1983). *Late night thoughts on listening to Mahler's Ninth Symphony.* New York: Viking Press.

Tonkovich, J., & Marquardt, T. (1977). The effects of stress and melodic intonation on apraxia of speech. In R. H. Brookshire (Ed.), *Clinical aphasiology: Conference proceedings* (pp. 97–102). Minneapolis, MN: BRK Publishers.

Warren, R. (1977). Rehearsal for naming in apraxia of speech. In R. H. Brookshire (Ed.), *Clinical aphasiology: Conference proceedings* (pp. 80–90). Minneapolis, MN: BRK Publishers.

Wertz, R. T., LaPointe, L. L., & Rosenbek, J. C. (1984). *Apraxia of speech in adults: The disorder and its management.* Orlando, FL: Grune & Stratton.

Wiedel, I. M. H. (1976). The basic foundation approach for decreasing aphasia and verbal apraxia in adults. In R. H. Brookshire (Ed.), *Clinical aphasiology: Conference proceedings* (pp. 209–223). Minneapolis, MN: BRK Publishers.

Wilson, P. (1977). Temporal relationships in therapy for apraxia. In R. H. Brookshire (Ed.), *Clinical aphasiology: Conference proceedings* (pp. 119–123). Minneapolis, MN: BRK Publishers.

Ziman, J. (1980). What is science? In E. D. Klemke, R. Hollinger, and A. D. Kline (Eds.), *Introductory readings in the philosophy of science* (pp. 35–54). Buffalo, NY: Prometheus Books.