A Neurobehavioral Approach to Dysprosody

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ABSTRACT

Much of the recent emphasis on prosody (the melody and rhythm of speech) and its disorders (dysprosody) has been on cognitive-affective functions attributed to cortical areas of the right cerebral hemisphere, with little further behavioral or neuroanatomical specification. This focus is inappropriately narrow both from the perspectives of neuropathogenesis and neurobehavioral phenomenology, and it is based on a limited view of prosody. Current models of brain organization for prosody propose lateralized representation based on functional (affective vs. linguistic) or featural (timing vs. pitch) properties of prosodic material. However, a role for subcortical structures in prosody is being increasingly described, and prosodic functions are now known to span a broad range in communication. In this article we describe normal prosody and present an overview of neurobehavioral disorders associated with acquired adult dysprosody. From these considerations we propose a neurobehavior-based approach to a more effective study of prosodic disturbance, and eventually, to better insight into normal prosody.

KEYWORDS: Prosody, basal ganglia, dysprosody, speech, affect

Learning Outcomes: As a result of this activity, the reader will be able to (1) describe the acoustic-auditory properties of prosody; (2) identify the various prosodic functions in communication; (3) delineate current models of brain-behavior relationships for prosodic behaviors and some of the limitations of those models; and (4) describe a neurobehavior-based approach that may be better suited to studying and describing disorders of prosody in the clinical setting than some of the currently popular perspectives.

Increased interest in the relationships between the brain and behavior over the past several decades has made prosody (the melody and rhythm of speech) a topic of study in disciplines like neurology, psychiatry, and neuroscience. Whereas the relationship between specific language abilities and particular regions of the brain has been the cornerstone of functional localiza-

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tion for over a century, recent attempts to place prosody in this framework have often yielded contradictory results. There are a number of likely reasons for this, but chief among them are a narrow, unsophisticated view of prosody, an overly simplistic approach to brain-behavior relationships, and a failure to consider the range of neurobehavioral syndromes that could yield insight into prosody and its disorders. In this article, we will address these limitations and propose a more inclusive approach to the study of dysprosody.

LANGUAGE STRUCTURE VERSUS PROSODIC CHARACTERISTICS

Details and theoretical implications are still debated, but there is no controversy about the essential building blocks of language: utterances representing sentences are made up of phones, phonemes, morphemes, words, phrases, and grammatical operations. Further, a consistent mapping between the acoustic events and the relevant language units, with their semantic and grammatical values, can be successfully performed. Although attempts have been made to create models of prosody in normal usage and some recent success has been achieved,[1,2] these models do not compare in viability and clarity with models of spoken language.

Fundamental differences between these two communication channels account for the discrepant scientific progress. Prosodic signals are graded and continuous, and, except for linguistic uses of prosody, do not easily form categories. This contrasts with linguistic entities, which are by nature discrete, unitary, and combinatorial. It is for this reason that prosody is also referred to as the "suprasegmental" or "paralinguistic" channel of communication. While linguistic information, as traditionally viewed, is decomposable into definable units and levels, the nature and characterization of the prosodic system remain elusive, not because the acoustic qualities cannot be measured, but because their correspondences in communication are rich and complex, interweaving linguistic, affective, attitudinal, psychological, pragmatic, and personal meanings.

FUNCTIONS OF PROSODY AND THEIR PHYSICAL PARAMETERS

Prosody can be defined as all the acoustic material not strictly comprising the phonemic inventory of vowels and consonants. This acoustic material is made up of fluctuations in pitch (or fundamental frequency), variations in loudness (or intensity), a number of durational features (e.g., phone, syllable, word, and phrase length; pausing, tempo, rhythm, and rate), and changing voice quality (e.g., murmured, harsh, clear). In the broad definition of voice quality, all of speech contributes to the product, inclusive of laryngeal tone and vocal and nasal tract effects, which encompass articulatory detail inherent in consonants and vowels.[3,4] For example, "baby talk" attains its effect in part because the lips are rounded and protruded, affecting the consonants as well as the vowels. An utterance spoken with imprecise approximation and closure of the articulators (slurring) will have a different communicative impact from the same message said with exaggerated articulation. Creaky voice, produced by irregularly and slowly vibrating vocal folds, can signal a victimized attitude in the speaker. These are just a few examples that illustrate that prosody, as generated by the range of physiological structures involved in vocal production, arises from the acoustic material enfolded in the texture of speech.

Normal ranges of prosody are not well described or understood, and much individual variation exists across normal speakers. To complicate matters, the multiple acoustic cues are difficult to discern impressionistically; for example, what the ear hears as speech melody may in fact be an element of intensity or timing. Each of these cues contains multiple dimensions (e.g., mean, variation, range), and all of them, especially timing, take shape over a variety of unit sizes. The speech unit—phone, syllable, word, clause, breath group—over which the acoustic-prosodic attributes are measured must also be considered and the length of this unit has been found to be an important factor in prosodic perception and production.

The complexity of the acoustic parameters of prosody is further compounded by the multiple functions that prosody serves. For normal

speakers, the contribution of prosodic cues to the linguistic and pragmatic aspects of communication has recently received considerable attention.[5] There is a longer history of clinical and normal studies of prosody as a principal conveyor of emotional—"affective"—expression in speech than of its other roles. Attitudes expressed in prosody, less investigated in clinical settings but important in everyday language use, routinely affect the message: by prosodic tone, the speaker signals an evaluative stance toward self, the content of the utterance, or the listener. For example, voice quality changes relative to the speaker's habitual voice may be used signal irony or sarcasm.

Personal identity, the unique vocal pattern of each voice, is also transmitted by prosodic cues. People know the voices of many family members, friends, colleagues, and acquaintances, as well as culturally well-known actors, politicians, sports personalities, and entertainers. Many studies have examined the vocal characteristics of voice recognition.[6] When famous voices are played backwards or altered temporally or acoustically, recognition of some voices but not others is impaired. This demonstrates that individual voices differ in the characteristics critical to their identity, and that each familiar voice is recognized as a complex, unique pattern.[7]

Indexical information in a person's voice can include information such as age, psychological attributes, sexual orientation, and personality.[8] Judgments about truthfulness, competence, pleasantness, and mood are regularly made by listening to the voice. For example, statements uttered by a male voice were rated as more important than the same statements made by a female voice.[9] A recent study of the acoustic-prosodic features that influenced listeners' perceptions suggested that psychopaths project sincerity in part by speaking quietly and avoiding vocal emphasis of single words.[10] Psychiatric and medical conditions, such as depression, autism and Asperger's syndrome, specific language impairment, dementia, and schizophrenia are also characterized by distinctive prosodic patterns.[11]

Unlike the affective or attitudinal uses of prosody, its linguistic function naturally utilizes discrete categories. For example, a sentence is either a question or a statement, and a clause is either restrictive or nonrestrictive. In tone languages, prosodic shapes are used phonemically at the word level on stimuli of about 300 msec, a length comparable to that of phonological syllables. English has a similar process as word- and phrase-level stress, such as the noun-verb minimal pairs "ímport/impórt" and noun-noun phrase pairs such as "hótdog/hot dóg." Another important linguistic-prosodic cue is sentence accent, which lets the listener know the topic or theme. In addition, prosodic signals do much of the work of the pragmatics of communication, such as directing turn taking in conversation, differentiating types of discourse units, and indicating whether literal or nonliteral meanings are intended.[12] These linguistic uses of prosody are more easily studied and understood than the other prosodic functions, as the mapping from acoustic building blocks to their corresponding functions can be specified in a more straightforward manner.

PROSODY AND THE BRAIN

The classical approach to establishing functional localization in the brain is to determine that damage to a particular area results in a specific loss of function. In the case of aphasia, damage to more posterior portions of the left hemisphere results in language comprehension problems, while damage to more anterior portions of the left hemisphere results in language production problems in most right-handed individuals. While attempts to clarify the details of lesion localization and aphasia type have led to a century of research, this simple formulation is generally accepted. A model of lateralized function has been a standard approach in characterizing brain-behavior relationships, with language representation in the left hemisphere and various other neuropsychological abilities in the right hemisphere.[13] Our brief review of the multiple functions of prosody and the complexity of the underlying physical parameters may be sufficient to explain why simple dichotomies have not been productive in studying

disorders of prosody. Nevertheless, studies of dysprosody have provided a start in understanding how prosody is represented in the brain.[14]

Some findings in brain-damaged patients suggest the neuroanatomic independence of the two processing modes of prosody, perception and production. In other cases, a common underlying disability seems to link the two modes. For example, deficits in emotional-prosodic production accompany the motor speech disorders seen in Parkinson's and Huntington's diseases. In addition, individuals with these disorders may have deficient emotional-prosodic and linguistic-prosodic comprehension deficits. Adding to the complexity of understanding prosody is the fact that significant differences can be seen in prosodic abilities across the production modes of spontaneous speech, repeated speech, and singing. Thus, even the apparently simple distinction between perception and production may be inadequate.

Simple Hemispheric Models

While it is universally agreed that linguistic functions for phonology, syntax, and linguistic semantics (lexicon) are represented in the left hemisphere of right-handed persons, the question continues to arise regarding the cerebral representation of prosody. Whether the task is spontaneous production, repetition, or comprehension, the right hemisphere, the left hemisphere, and subcortical nuclei have all been identified as responsible brain areas.[15–20] Simple hemispheric models have distinguished between cognitive processes for linguistic versus emotional prosody and/or between acoustic-perceptual processes for timing versus pitch phenomena, associated with the left versus right cortical hemisphere, respectively. Although these models account for many of the observations of dysprosodic function, considerable uncertainty remains. Moreover, a significant role of striatal structures has been a recent focus in prosody research, raising additional questions about left/right asymmetries.[24]

One of the earlier simple hemispheric models proposed a global, unitary emotional-prosodic competence lateralized to the right hemisphere, varying by processing mode (production, repetition, comprehension) with the anterior-posterior hemispheric axis.[16] Unfortunately, this model has been only weakly supported or not supported at all. The notion of affective prosody as a unitary entity, modulated by the right hemisphere, is oversimplified and not substantiated, even though it has found its way into textbooks of neurology. Clinically, dysprosodic deficits are rarely seen in right hemisphere damaged persons.

No hemispheric differences were found for affective-prosodic tasks in numerous group studies, and in many earlier studies, patients with left hemisphere damage were simply not examined. When such patients were finally studied using the same method that led to the proposal for right hemisphere dominance for affective prosody, prosodic deficits were seen in left hemisphere damaged subjects as well.[21] Similarly, in several studies, no hemispheric differences were found for linguistic tasks. Further, type and difficulty of task were found to significantly influence performance.[22,23]

Looking Below the Surface

Difficulties for the cortical-hemispheric cognitive models for prosody were escalated when studies pointed to a significant role of subcortical nuclei in dysprosodic production and comprehension. In several studies, there was little or no effect of side of hemispheric lesion on any prosodic tests, but a common basis of basal ganglia damage associated with dysprosodic deficits across tasks was seen.[24,25]

Patients with lesions of the caudate nucleus, globus pallidus, or putamen, and concomitant mood or motivational disturbance have been observed to have deficits in emotional-prosodic production. From clinical descriptions, one can infer that "flat speech" is an indicator of "abulia," a motivational disorder.[26–29]

Neurodegenerative processes such as Parkinson's and Huntington's diseases are associated with difficulties in initiating output behaviors and can also result in a syndrome that includes alterations in psychological state, motivation, and prosody. Many Parkinson's patients have a mood

disorder which is independent of the motor disability, and which is not viewed as reactive to the disability. The observed dysprosody in Parkinson's disease may best be considered a feature of this frontosubcortical syndrome, which includes motor/movement initiation, mood, and speech deficits.[30]

Lateralization Based on Physical Features

Another approach to hemispheric specialization for prosodic ability focuses on auditory/acoustic processing features of perception and production rather than on a global, unitary competence like "emotional prosody." Most of the studies of acoustic features have focused on timing (rate) and pitch, which contribute to the various prosodic functions in different proportions.[31] In speech production and comprehension, timing control may be managed separately from pitch control. In a few studies of left hemisphere function, durational characteristics have emerged, although usually not for emotional-prosodic utterances. The source of the dysprosody may lie in timing or in pitch features.

A specialization for temporal judgments has been associated with left hemisphere processing in normal subjects. Receptive disturbances of rhythm, a temporal parameter, have been seen in patients with left but not right hemisphere lesions and rhythm has been associated with left hemisphere function,[32] but the cerebellum also plays an important role in this function. Right hemisphere damaged subjects were not notably disturbed in use of temporal cues in processing affective or linguistic prosody,[33] while left hemisphere damaged patients' errors in comprehension of emotional prosody in speech were attributable more to mistaking timing cues than pitch cues in the sentences.[20] Deficient temporal processing accounted for a significant finding of affective-prosodic comprehension deficit in Parkinson's disease[34] and for deficient speech perception.

There is independent evidence that the right auditory cortex is specialized for complex pitch perception[35,36]; of course, pitch is a major cue in all functions of prosody. Deficient pitch processing may partially account for poor performance on prosodic stimuli by subjects with right hemisphere damage. In production, evidence from Wada testing and extreme left hemisphere dysfunction indicate a preserved ability of the right hemisphere to sing, that is, to manipulate motor control of pitch. Prosody, as the melody and rhythm of speech, is likely to have an important relationship to music abilities and should be considered in the context of musical function.[36,37]

Disruption in the control of vocal pitch simply in the context of vowel production was seen in a patient with left hemiparesis and monotonic speech resulting from a right hemisphere frontotemporal tumor.[38] A similar loss of motor pitch control was observed in a patient with right frontotemporal-parietal cortical and subcortical damage, who had formerly sung in musical theatre, but who postmorbidly spoke with severely reduced variation in pitch and was unable to sing even simple, familiar melodies. His speech disorder affected motor expression of emotional, pragmatic, and linguistic uses of prosody. This patient could recognize but not imitate prosodic intonations of various kinds. In these cases, expressive dysprosody reflected a loss of vocal pitch modulation independent of affective, linguistic, or other communicative demands.

Functional-Feature Lateralization

An early proposal about prosody and brain, the "functional lateralization" hypothesis stated that those acoustic/motoric features of prosody that are utilized functionally as linguistic (those at the level of phoneme, syllable, word, or clause) are lateralized to the left hemisphere, while those involved in attitudinal/emotional and personal voice quality recognition are lateralized to the right hemisphere[39] (see Fig. 1). This notion reflected the observation that language is lateralized to the left hemisphere, and was based on the discrete and compositional quality of linguistic uses of prosody, in comparison to the graded, patterned nature of paralinguistic information. This model incorporated findings for familiar voice recognition, complex pitch pro-

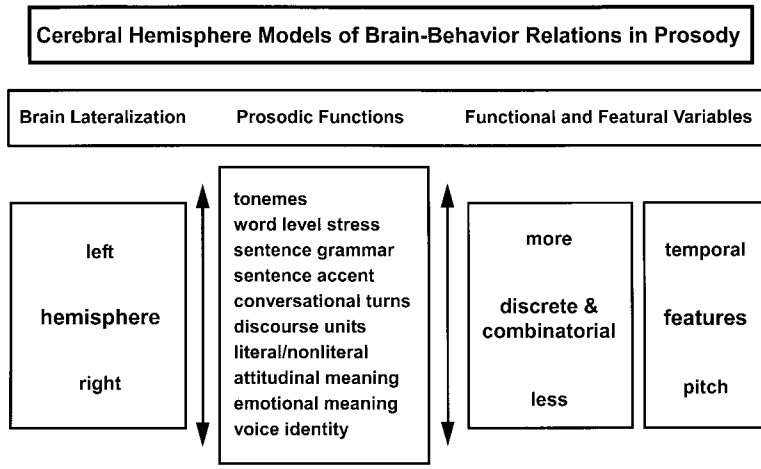

Figure 1 This continuum of functions can be contrasted along two dimensions: (1) more or less discrete and combinatorial, and (2) having greater emphasis on temporal or pitch characteristics. Note that the properties "less structured" and "more emphasis on pitch" are both associated with greater distance toward the "right hemisphere" end of the continuum. This confluence could account for much of the reported right hemispheric superior performance for prosodic utterances. Length also varies regularly on these continua. For shorter stimuli (around 300 msec), temporal and sequential processing becomes more prominent and processing will more likely involve left hemisphere capabilities. Stimuli made up of patterns or configurations in the "holistic" processing mode will more likely to involve right hemisphere capabilities.

cessing, timbre, and chords,[40] which are complex auditory-acoustic patterns compatible with preferential processing in the right hemisphere. The functional-feature lateralization model integrates both global, cognitive competencies (e.g., linguistic, pragmatic, affective, and personal stimuli) and physical features (e.g., pitch and timing cues) (see Fig. 1). Early support for the role of function in laterality of prosodic cues appeared in comprehension studies of linguistic tonal stimuli by speakers of a tone language (i.e., Thai) in normal[41] and neurologically impaired speakers,[42] but not by speakers of a nontone language (i.e., English).[43] Similar findings were reported for word- or phrase-level linguistic contrasts.[44,45]

These results are in agreement with laterality studies in normal, English-speaking subjects utilizing an array of experimental methods, suggesting that linguistically structured stimuli showed a preference for left hemisphere processing, while stimuli in the graded form characteristic of emotional or other nonverbal sounds show a right lateralized preference.[46–48] In their review of the neural bases of prosody for receptive and expressive prosody, Baum and Pell concluded that the left hemisphere may be particularly involved in the processing of phonemic and lexical tone contrasts (segmental and word-level prosody), whereas "the effects of the functional load of prosodic cues" becomes less clear at the sentential level.[49]

Casting some question regarding the association of linguistic prosody with left hemisphere function, linguistic-prosodic deficits following right hemisphere damage have been reported. In many studies, no differences between left hemisphere damaged and right hemisphere damaged groups using either linguistic or emotional stimuli have been observed.[20,50] Since multiple etiologies may underlie prosodic disturbances, it is not surprising that such inconsistencies occur in group studies. Patients sorted into left or right hemisphere damaged groups for prosody research often do not have clinically apparent prosodic deficits.[51] Or they may have specific prosodic deficits not detected by the array of research protocols.

In addition, the notion of preferential processing of emotional experiencing in the right

hemisphere, including verbal labeling of these experiences, has come to light.[52] This underlying cognitive function can be expected to influence production and comprehension of affective-prosodic material. Despite the bilateral neuroanatomical basis of structures known to be involved in emotional functions, there is evidence that emotional experiencing has greater representation in the right hemisphere, manifest in findings for lexical, facial, and gestural processing. The term "nonverbal affect lexicon" has been coined to describe the specialized abilities of the right hemisphere.[53] Borod et al.[54] recorded right and left hemisphere damaged subjects during the description of personally relevant emotional (seven emotions) and nonemotional (characteristics of people) experiences in an attempt to elicit spontaneous emotional expression. Right hemisphere damaged patients produced less emotional content than the control group, but did not differ from the left hemisphere damaged group. Related theories include observations that emotional disorders are often associated with right hemisphere damage,[55–58] and the notion that the right hemisphere is specialized for establishing, processing, and maintaining personal relevant stimuli, which naturally contain affective features.[59]

Preference for personally relevant stimuli and for integrated patterns may account for right hemispheric superior processing of familiar voices. Familiar voice percepts fall within the least linguistically structured of informational material in the prosodic signal, because each is made up of a unique pattern (see Fig. 1). Deficient familiar voice recognition (phonagnosia)[60,61] has been identified with focal damage to the right parietal lobe. Because the left hemisphere processes speech and speech carries all the voice identity information, patients with right temporal/parietal damage performed more poorly than those with left hemisphere damage when asked to recognize familiar voices. Not only did patients with left hemisphere damage recognize the voices, in many cases they performed as well as normal listeners. Even severely aphasic patients, who could not understand what was being said, knew who was saying it. Further, voice recognition (perception of familiar voices) and voice discrimination (perception of unknown voices) appear to be separate abilities because brain lesions can interfere with one ability while leaving the other unaffected.

Limits of Functional and Featural Models of Prosody

Many reports either fail to support the functional or the featural models of prosody, or provide simultaneous support for both types.[62] Acoustic analyses of speech of patients with left or right hemisphere damage have yielded diverse results. Abnormal intonational characteristics have been found in fluent as well as nonfluent aphasic patients. Most observers agree that in most cases of severe aphasia, some prosodic output function remains, in contrast to "flat" speech observed in other clinical conditions; but the prosodic forms do not attain normal range, shape, or functionality.[63] Shapiro and Danly,[64] who obtained linguistic and emotional-prosodic utterances by having right hemisphere damaged patients read aloud sentences embedded in paragraphs, reported reduced fundamental frequency mean and variability in association with anterior damage, while posterior damage was associated with elevated values. For the proposal that left hemisphere damage is associated with deficits in prosodic timing, inconsistent results also appear, with abnormalities reported for both left and right hemisphere damaged subjects.

Persons with agenesis of the corpus callosum and normal intelligence were significantly impaired on a test of affective-prosody comprehension, suggesting that communication between cerebral hemispheres during language development is required for successful development and execution of this task.[65] However, adult patients who have undergone surgical section of the corpus callosum for the control of epilepsy have normal conversational prosody. These observations, which are difficult to interpret, are more likely to find a place in a model that is finely and realistically tuned to the actual array of prosodic elements in normalcy and disease.

A MODEL BASED ON A SEGMENTAL NEUROBEHAVIORAL APPROACH TO PROSODY

A basic question underlying the various approaches to cerebral representation of prosody is whether there are distinct, modular competencies, such as the ability to process emotional information in speech, or whether a combination of motor, perceptual, and superordinate organizational operations (e.g., linguistic, affective, motivational, etc.) is coordinated in orchestrating the set of prosodic behaviors. The question is an important one, because focal lesions or neurological deficits can be expected to affect a "unitary" competency differently from a constellation of disparate functions.

The literature reviewed in this article strongly suggests that prosodic behaviors are brought about by a constellation of perceptual, motor, and organizational factors, which can be differentially dysfunctional following brain damage in relatively simple or complex ways. Thus, a variety of "causes" can lead to a clinical presentation of dysprosody, or to inferior performance on prosody tests. In this framework, it is possible to fully accommodate the hemispheric models, physical feature approaches, and functional lateralization, with expansion to hypotheses based on observations in subcortical damage relating to motor organization as well as neurobehavioral mood and motivational states.

We propose that the discrepant results across group studies are due to the fact that dysprosody arises from disruption of a complex system that can occur following damage or dysfunction at multiple levels of the central nervous system. Our conceptual framework for the study of prosody recognizes these levels of neurobehavioral organization in the central nervous system. Table 1 summarizes our neurobehavioral approach to the study of dysprosody. The chief clinical features of dysprosodic speech are on the left side of the table, and the major divisions of the central nervous system associated with those features is on the right. Clinical presentations are typically complicated, but the general approach is to distinguish between abilities at the level of features versus more complex cognitive, linguistic, behavioral, and affective functions. In this framework, dysprosody may occur as a syndrome with widespread behavioral alterations, or as a relatively isolated deficit.

Beginning with the expression and perception of the elements of prosody, damage to the central nervous system from the brain stem to the cerebral cortex can result in abnormalities in pitch, timing, coordination, intensity, and vocal quality. For example, damage to the medulla can produce hoarse voice. Cerebellar dam-

Table 1 A Summary of a Neurobehavioral Approach to the Study of Dysprosody

Chief Clinical Features	Typical Lesion Sites*
Feature expression and perception	
Pitch, timing, coordination (rhythm), intensity, vocal quality	Brain stem, cerebellum, diencephalons (thalamus), striatum, cortex§
Motor control with behavioral sequelae	
Difficulty with movement initiation and maintenance, involuntary movements, depression, psychosis	Striatum, thalamus, cortex
Behavioral syndromes	
Apathy, abulia, akinesia, irritability, impulsiveness, primary psychiatric disorders	Striatum, cortex
"Representational" or ideational deficits	
"Prosodic agnosia" (?)	Cortex
"Prosodic anomia" (?)	Cortex
"Prosodic apraxia" (?)	Cortex

*These sites are not meant to be exhaustive but rather indicative of levels of organization of prosodic functions in the central nervous system (see text).
§Cortex refers to cerebral cortex and includes underlying white matter.

age can produce ataxic speech, a condition in which timing and articulatory coordination are affected. Striatal (basal ganglia) damage can produce movement disorders including tremor, athetosis, chorea, and ballism that alter prosody, and thalamic injury can produce extrapyramidal movement disorders affecting speech as well. Possibly in association with its role in motor planning and monitoring, subcortical dysfunction can be associated with receptive dysprosody. Cortical damage, too, can affect elements of prosody as a result of weakness or spasticity, perceptual failure, planning problems, or other organizational deficits. Although cortical deficits are more typically associated with ideational disorders (see below), damage at this level can produce a deficit in expressive prosody that could be characterized as a cortical dysarthria.

Disruptions in motor control for the elements of prosody can also occur in combination with neurobehavioral syndromes. Damage to the striatum or certain cortical areas may result in syndromes involving alteration of motivation, initiation, psychological states, and/or motor ability, which may include clinically dysprosodic speech in production, repetition, or comprehension. Parkinson's disease and Huntington's disease both result in dysprosodic speech in expression and reception and are typically associated with behavioral sequelae including depression and psychosis.

Less understood than movement disorders, primary behavioral disorders such as apathy, akinesia, and abulia can produce significant alterations in prosody without obvious movement disorders. These conditions are also associated with damage to the striatum and specific cortical areas. Primary psychiatric conditions would fall into this classification category as well, although the responsible neuropathophysiologies are likely more functional than structural.

Disorders of prosody that could be characterized as purely representational or ideational (i.e., that exist while the ability to express or perceive the physical features of prosody remain intact) would be expected at the cortical level. If such ideational deficits exist, cortical damage could be expected to produce agnosia for meanings of prosodic patterns in production or comprehension; apraxia for the execution of those patterns; or anomia for the labeling or lexical categorization of prosodic meanings. We include a category of prosodic anomia, as recent studies attribute right hemisphere prosodic deficits to specific anomia, invoking a deficient "affect lexicon."[53] Claims in the literature for right cortical representation of prosody presumably refer to a specific agnosia—inability to retrieve (access) or comprehend affective-prosodic meanings, resulting in impaired prosodic output and/or recognition; or they refer to an apraxia (e.g., sometimes termed "motor aprosodia"), impairment in formulating the form of prosodic utterances. Impaired "melody of speech" of an apraxic variety has traditionally been ascribed to the damaged left cortical hemisphere. Disorders in any of these ideational capacities could result in faulty prosodic expression or comprehension. Prosodic agnosia or apraxia may be seen to differentiate into linguistic (tones, word-stress contrasts), affective-prosodic, pragmatic, and personal indexical varieties, although this remains unclear.

Our approach emphasizes that attention should be paid to the level and scope of disorder in dysprosody. As has been noted, some cases of dysprosody that might easily be characterized as affective or linguistic may well be entirely attributable to deficiency in pitch and/or timing control, affecting prosodic function across the various categories, including affective, linguistic, attitudinal, pragmatic, or personal functions. In such instances, characterizing a dysprosody as a loss of an ideational competency would be analogous to misclassifying a dysarthria as an aphasia. The neurobehavioral, syndrome approach to prosodic disorders also allows for accommodation of dimensions of dysprosody seen in association with a wider variety of disorders like Alzheimer's disease, autism, and schizophrenia, which have not been typically considered in discussions of prosody.

This proposed approach to the study of prosody is based on several facts and conclusions. Prosody has a range of functions, of which at least five can be clearly identified (linguistic, affective, attitudinal, pragmatic, personal). Each of these has complex, multiple subtypes, and individual languages have unique ways of structuring these functions. Whether brain damage

affects these functions selectively is as yet unclear. We believe it is clear, at this point in our understanding, that considering any of these complex functionalities of prosody as a unitary, monolithic cognitive module, without further examination, is dangerous.

The model proposed in Table 1 is based on the view that dysprosody has various etiologies—that classic neurobehavioral, auditory, or motor dysfunctions may account for many observed deficits; and finally, that acquired prosodic disturbance can result from damage to any of a number of brain subsystems. Before the phenomenon of dysprosody can be understood, a formal, thorough conceptualization of functional and acoustic varieties of prosody must be developed, along with a systematic mapping between acoustics and function. Correspondingly, a more appropriate view of neuropathogenetic processes must be incorporated to advance the study of brain-behavior correlations in dysprosody.

The degree to which a dysprosody is associated with a relatively "pure" motor or perceptual deficit, a mixed motor-behavioral or perceptual-behavioral syndrome, a relatively "pure" behavioral syndrome, or with an ideational competency depends on the site of the lesion and the disease process. Dysprosody may have more of an "affective" flavor in those situations in which a behavioral syndrome is present, but this likely reflects the nature of the syndrome more than any features of prosody. Compared to aphasia, the clinical presentation of dysprosodic phenomena can result from damage to a relatively large number of brain regions, most of which are not homologous to language areas.

IMPLICATIONS OF THE NEUROBEHAVIORAL APPROACH TO PROSODY FOR REHABILITATION

Patients with "flat speech" due to various causes are communicatively handicapped. Linguistic, emotional, and pragmatic cues, normally produced in speech, are lacking. We have discussed the multiple etiologies of dysprosody. These patients may sound "depressed" or "disinterested" when the problem lies in their motor output, not their psychological state. Or the dysprosody may be due to perceptual or cognitive dysfunction. Identification and evaluation of prosodic disturbance in clinical settings remain undeveloped and largely unpracticed. Although several protocols are available, none is used routinely. Understanding the source of the speech disorder is the first step in designing appropriate therapy. This important service to identified patients deserves broader attention and official recognition within the treatment delivery system.

For treatment, once a prosodic deficit is identified, visual displays of the intonational contour providing a model to match are available for speech-language therapy. One study of treatment of motor dysprosody used voice pitch biofeedback and modeling of emotional utterances.[66] We believe that a neurological model accommodating the widest range of clinical presentations is necessary to fully depict clinical observations of dysprosody, which is a first step in developing proper methods of evaluation and treatment.

SUMMARY

In normal language use, utilizing and understanding prosodic nuances in speech is clearly a prodigious ability and one of great importance and complexity. Despite a fair amount of literature on the topic, many claims about the effects of brain damage on prosodic ability remain in uncertainty. Conflicting or discrepant brain models have suffered from (1) focusing on one dimension of prosodic function, (2) attempting to characterize dysprosodic behaviors as based in solely cognitive or perceptual/motor abilities, or (3) adhering to strict notions of hemispheric lateralization of function. The purpose of this article is to suggest a comprehensive neurobehavioral model of brain representation underlying prosodic function, leading to a new approach to the study and understanding of dysprosodic speech. Rather than simply viewing expressive dysprosody as an independent sign or symptom, this disorder is often best appreciated in the context of a syndrome or symptom complex. This comprehensive model accommodates pre-

vious approaches and at the same time offers a broader format for accounting for clinical observations of dysprosody.

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