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Dysprosodic speech following basal ganglia insult: Toward a conceptual framework for the study of the cerebral representation of prosody

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Abstract

Progress in understanding brain/behavior relationships in adult-acquired dysprosody has led to models of cortical hemispheric representation of prosodic processing based on functional (linguistic vs affective) or physical (timing vs pitch) parameters. These explanatory perspectives have not been reconciled, and also a number of neurobehavior syndromes that include dysprosody among their neurological signs have not yet been integrated. In addition to expanding the functional perspective on prosody, some of these syndromes have implicated a significant role of subcortical nuclei in prosodic competence. In this article, two patients with acquired dysprosodic speech following damage to basal ganglia nuclei were evaluated using behavioral, acoustic, cognitive, and radiographic approaches. Selective quantitative measures were performed on each individual's performance to provide detailed verification and clarification of clinical observations, and to test hypotheses regarding prosodic function. These studies, combined with a review of related clinical research findings, exemplify the value of a broader perspective on the neurobehavioral dysfunction underlying acquired adult dysprosodic speech, and lead to a new, proposed conceptual framework for the cerebral representation of prosody.

Keywords: Prosody; Basal ganglia; Neurolinguistics; Laterality

1. Background

In normal language use, producing and understanding prosodic nuances in speech is a prodigious ability and one of great importance and complexity. Prosody, the melody of speech, is made up of fluctuations in pitch or fundamental frequency, variations in loudness (or intensity) (Fairbanks & Pronovost, 1939; Lieberman & Michaels, 1962; Williams & Stevens, 1972), a number of durational features (Fairbanks & Hoaglin, 1941) (e.g., syllable, word, phrase, and breath group length, pausing, phrase final lengthening, tempo, and rate) (Apple, Streeter, & Krauss, 1979; Breitenstein, Van Lancker, & Daum, 2001; Hird & Kirsner, 2002) and changing voice quality (Bricker & Pruzansky, 1976; Laver, 1980, 2000; Remez, Fellowes, & Rubin, 1997; van Dommelen, 1990; Voiers, 1964).

Although many nuances provided by prosody remain to be described, five basic types of information within the communicative functions of prosody can be identified: emotional (affective) (Banse & Scherer, 1996; Hutter, 1968; Scherer, 1986); attitudinal (Cohen, Douaire, & Elsabbagh, 2001; Freese & Maynard, 1998; Protopapas & Lieberman, 1997), pragmatic (Cutler, Dahan, & van Donselaar, 1997; Douglas-Cowie & Cowie, 1998; Lonie & Lesser, 1983; Pell, 2001; Schegloff, 1998; Wells & Macfarlane, 1998); linguistic (including grammatical and semantic) (Baum, Pell, Leonard, & Gordon, 2001; Bolinger, 1986; Crystal, 1969; Ladd, 1996; Leonard, Baum, & Pell, 2001; Pike, 1945; Schafer, Speer, Warren, & White, 2000), and indexical (personal

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voice identity, including information about background and personality) (Alpert, 1982; Bricker & Pruzansky, 1976; Ellgring & Scherer, 1996; Hecker, 1971; Helfrich, 1979; Kreiman, Van Lancker-Sidtis, & Gerratt, 2005; Sapir, 1926; Scherer, 1979; Sidtis & Van Lancker Sidtis, 2003). Only a small part of this range of prosodic function in human communication has been examined in a neurological context: mostly emotional cues, a heterogeneous set of linguistic-prosodic contrasts, and more recently various pragmatic elements.

Many reports of the effects of brain damage on prosodic ability are contradictory and remain unconfirmed, as might be expected for such a complex topic (for reviews see Ackermann, Hertrich, & Ziegler, 1993; Baum & Pell, 1999; Van Lancker & Breitenstein, 2000; Wong, 2002). Nonetheless, some progress has been made. From the considerable effort that has been expended in studying prosody, two theoretical perspectives correlating brain function with prosodic behaviors have emerged, both of which pertain to cerebral lateralization: the functional-cognitive interpretation, which states that the function of the prosodic stimulus (whether linguistic or affective) determines laterality (Bryden & Ley, 1983; Bryden, 1982; Charbonneau, Scherzer, Aspirot, & Cohen, 2003; Chobor & Brown, 1987; Friederici & Alter, 2004; Kimura, 1967; Ley & Bryden, 1982; McNeely & Parlow, 2001; Van Lancker, 1980; Van Lancker & Fromkin, 1973, 1978; Walker, Pelletier, & Reif, 2004). A related parameter, degree (or presence) of structure, roughly classified as discrete (as in the tones of tone languages) versus graded (as in emotional intonation), has been considered (Gandour et al., 2000; Pell, 1999a, 1999b; Van Lancker, 1980; Van Lancker & Fromkin, 1978). The other prevailing model is represented by the physical stimulus approach, which distinguishes temporal from pitch elements in the prosodic signal (Breitenstein et al., 2001), and assumes hemispheric representation for those stimuli which are based more saliently on one or the other acoustic-prosodic parameter (Lalande, Braun, Charlebois, & Whitaker, 1992; Sidtis, 1984; Van Lancker & Sidtis, 1992; Zatorre, 1988). Variants of the second approach include the notions that variations in band width (Lakshminarayanan et al., 2003), or length (Boutsen & Christman, 2002; Pell, 2001) contribute to hemispheric side of processing (see Table 1).

Beginning with the claim that the right hemisphere (RH) is specialized for processing the "affective component" of language (Bowers, Coslett, Bauer, Speedie, & Heilman, 1987; George et al., 1996; Gorelick & Ross, 1987; Ross, 1981), ample support has emerged for the functional hypothesis (Blonder, Bowers, & Heilman, 1991; Blumstein & Cooper, 1974; Blumstein & Goodglass, 1972; Buchanan et al., 2000; Charbonneau et al., 2003; McNeely & Parlow, 2001; Schmitt, Hartje, & Willmes, 1997; Tucker, Watson, & Heilman, 1977; Weintraub, Mesulam, & Kramer, 1981). However, a number of studies have not supported the RH hypothesis of affective-prosodic mediation (e.g., Bradvik et al., 1990; Darby, 1993; Schlanger, Schlanger, & Gerstman, 1976; for review see Baum & Pell, 1999; Ryalls, 1988; Van Lancker, 2000; Van Lancker & Breitenstein, 2000). Instead, some results have led

Table 1
Schema of the current functional and acoustic models of hemispheric specialization for prosodic information in speech

Hemisphere	Prosodic functions	General properties		
Left	Tonemes	Timing	Structured	Shorter
	Word level stress			
	Sentence grammar			
	Sentence accent			
	Conversational turns			
	Discourse units			
	Literal/nonliteral			
	Attitudinal meaning			
	Emotional meaning			
Right	Voice identity	Pitch, quality	Graded	Longer

At least three properties of the stimuli (acoustic cue, structure, and length) have been proposed as influences on hemispheric side of processing. Whether all or some of the cognitively functional categories listed in the chart are selectively disrupted by focal neurological damage remains to be determined (adapted from Sidtis and Van Lancker Sidtis, 2003).

to physical models (Baum, 1998; Baum & Pell, 1997; Schirmer, Alter, Kotz, & Friederici, 2001), associating temporal processing with the left hemisphere (LH) and pitch processing with the RH (Sidtis, 1980; Zatorre & Belin, 2001; Zatorre, Evans, Meyer, & Gjedde, 1992). Damage to the LH has resulted in deviant speech timing features (Baum, 1992; Baum, Blumstein, Naeser, & Palumbo, 1990; Carmon & Nachshon, 1971; Danly & Shapiro, 1982; Gandour & Baum, 2001; Monrad-Krohn, 1947, 1963; Munson, 1994; Oepen & Berthold, 1983; Ouellette & Baum, 1994; Robinson & Solomon, 1974; Ryalls, 1986; Van Lancker et al., 1988), while RH patients have been primarily deficient in processing pitch (Baum & Pell, 1997; Hird & Kirsner, 1993; Johnsrude, Penhune, & Zatorre, 2000; Robin, Tranel, & Damasio, 1990; Shapiro & Danly, 1985; Sidtis & Feldmann, 1990; Sidtis & Volpe, 1988; Tramo & Bharucha, 1991; Tramo, Shah, & Braida, 2002). It has also been noted that these two perspectives—the functional and the physical hypotheses—are compatible and can coexist, probably contributing in varying degrees to prosodic processes, thereby, in many cases, accounting for apparently inconsistent clinical-experimental observations (Pell, 1998; Sidtis & Van Lancker Sidtis, 2003). Indeed, that both hemispheres are necessary for successful prosodic performance is further suggested by studies of congenitally acallosal persons (Paul, Van Lancker, Schieffer, Dietrich, & Brown, 2003). Table 1 schematically represents the notions that several disparate influences, including acoustic cue, stimulus mode and length, and others more recently under investigation, may all contribute to determining hemispheric specialization (see also Pell, 1998). Whether or not "cognitive" prosodic functions, such as those listed in Table 1, are selectively impaired in neurological disorders, independently of these other influences, remains to be clearly established.

The numerous reports that implicate subcortical structures in prosodic function in normal subjects (Kotz et al., 2003), following injury by lesion (Breitenstein, Daum, & Ackermann, 1998; Cancelliere & Kertesz, 1990; Karow, Marquardt, & Marshall, 2001; Ross, Thompson, & Yenkosky, 1997; Starkstein, Federoff, Price, Leigarda, & Robinson, 1994), and as

sequelae to progressive neurological disease (Benke, Bosch, & Andree, 1998; Blonder, Gur, & Gur, 1989; Breitenstein, Daum, & Ackermann, 1997, 1998; Breitenstein et al., 2001; Critchley, 1981; Lloyd, 1999; Pell, 1996; Speedie, Brake, Folstein, Bowers, & Heilman, 1990) have not been integrated into hemispheric models. Many such reports associate dysprosody with basal ganglia damage or dysfunction without respect to cerebral laterality, thus adding another dimension of brain–behavior correlations in prosody. The nuclei of the basal ganglia have recently been implicated in fluency and rate of speech in brain imaging studies (Alm, 2004; Liotti et al., 2003; Ludlow & Loucks, 2003; Santens, De Letter, Van Borsel, De Reuck, & Caemaert, 2003; Sidtis, Strother, & Rottenberg, 2003), but the role of the caudate nucleus, the putamen, and the globus pallidus in prosodic competence, as part of a frontal-subcortical circuit (Alexander, Crutcher, & DeLong, 1990; Alexander, Benson, & Stuss, 1989), although recognized as a strong participant in speech (e.g., Lieberman, 2002), has yet to be clarified.

Reports of prosodic deficits following adult-acquired brain damage also do not fit well with classic descriptions in the neurological literature on speech disorders, variously referred to as "impaired melody of speech," "monopitch," "monoloudness," "hypophonia," and "altered rate of speech" (Darley, Aronson, & Brown, 1975; Duffy, 1995; Goodglass & Kaplan, 1983; Kent & Kim, 2003; Kent & Rosenbek, 1982; Moen, 1991; Weniger, 1984). Impaired melody of speech was long held to be a feature of LH damage. Monopitch and rate changes described speech following subcortical dysfunction. This lack of correspondence between current and older descriptions of prosodic deficits arises at least in part from the current focus on group studies, where it is seldom determined whether prosodic abnormalities occur in the individual patients (Myers, 1999). More recently, clinical case studies have appeared, some confirming one of the prevailing models (Barrett, Crucian, Raymer, & Heilman, 1999), but most of these indicate a complexity in prosodic abilities not yet accounted for (Bertier-Marcelo, Fernandez, Celdran, & Kulisevsky, 1996; Blonder, Pickering, Heath, Smith, & Butler, 1995; Dykstra, Gandour, & Stark, 1995; Gandour, Larsen, Dechongkit, & Ponglorpisit, 1995; Niemi, 1998; Osmon, Panos, Kautz, & Gandhavadi, 1998; Patel, Paretz, Tramo, & Labreque, 1998).

In discussions of a RH role in expression and comprehension of affect, a fundamental question—whether affective-prosodic processing (an "affective component of language") exists at a conceptual level analogous to language—remains nebulous. This putative entity has not been characterized or described, and its ontological status is uncertain.[1] It is possible that the failure to consistently confirm the cognitive-functional model of linguistic and affective prosody in association with left, and right hemispheres, respectively (Baum, Pell, Leonard, & Gordon, 1997; Pell & Baum, 1997a; Schlanger et al., 1976) and the subsequent emergence of the physical feature model (associating temporal and pitch features with left, and right hemispheres, respectively) (Pell, 1999a, 1999b; Pell & Baum, 1997b; Van Lancker & Sidtis, 1992; Walker, Fongemie, & Daigle, 2001) is attributable to a basic conceptual error in postulating an ill-defined "affective component of language" (see Table 1).

A further limitation to premature models of dysprosody arises from the fact that normal adult standards for prosodic performance are not codified, and current theories of normal prosody are at the early stages of development (Hird & Kirsner, 2002; Le Dorze, Lever, Ryalls, & Brassard, 1995; Shattuck-Hufnagel & Turk, 1996; Tree & Meijer, 2000), leading to undue reliance on the expertise of the examiner. Considerable variability in ability to "hear" paralinguistic material can be assumed. Techniques or programs for training clinicians in prosodic testing skills are nonexistent. Selection of measurable variables is not straightforward. The significance of individual differences and the effect of speech contexts are challenging factors (Peppe, Maxim, & Wells, 2000), and, in the clinical setting, premorbid speech prosody, which may provide important clues to the effects of specific lesions or disease processes, is often difficult to obtain or is not sought. (A model study of pre- and post-morbid prosodic production in a single case of RH damage was provided by Blonder et al. (1995).) Acoustic measurement techniques for affective- and linguistic-prosodic cues are under development in research studies, but these are as yet too labor-intensive to be clinically applied at this time, and they are limited by uncertainties in how acoustic parameters correlate with prosodic meanings. A careful consideration of acoustic and behavioral detail is desirable to advance our understanding of prosodic competence and its disorders.

Given these contingencies, patient prosodic function is difficult to evaluate in the clinical setting. Prosodic performance is formally tested only rarely in clinical neuropsychology (Kiss & Ennis, 2001; Wymer, Lindman, & Booksh, 2002) or speech pathology (Duffy, 1995), and treatment of specific dysprosodic deficits is even less frequent (Stringer, 1996; Vance, 1994). Preliminary efforts to develop an instrument and treat to evaluate prosodic disturbance following brain damage are based on a formerly prevailing model, which associates "affective dysprosodia" with RH damage (Rosenbek et al., 2004). This model does not allow for consideration of specifically disordered elemental acoustic cues, the involvement of broader contexts of prosodic information such as linguistic and pragmatic cues, or behavioral disorders that may be associated with subcortical sites (Sidtis & Van Lancker Sidtis, 2003).

Addressing these challenges one by one is desirable to develop a composite model of prosody that is adequate in both description and explanation. A careful consideration

[1] Note that the "language component" is made up of phones, phonemes, morphemes, words, phrases, and sentences organized by linguistic rules (e.g., Denes & Pinson, 1993); in contrast, there is no such descriptive apparatus or nomenclature available for the "affective component" of language.

of detail in clinical presentation, placed in a broader context of prosodic functioning, is desirable to advance our understanding of this important aspect of communicative competence. The purpose of this paper is to discuss in detail two adult cases of acquired expressive dysprosody following subcortical damage, placing them in the broader context of the theoretical perspectives achieved thus far in prosody research. These studies are used to advocate certain methods for evaluating prosodic deficits in comprehension and production and to suggest approaches to confirming clinical observations using quantitative measures. A comprehensive review of these two cases leads to a broader view of the role of brain function underlying prosodic function than that which has typically appeared in the prosodic literature. In presenting these two patient cases, the larger aims of this paper are thus to address questions regarding: (1) measurement approaches to clinically observed dysprosody; (2) the integration of subcortically-based dysprosody into the larger picture of laterality models; (3) enfolding of traditional motor speech disorders into a neurobehavioral model of dysprosody. We present these two patients together, as they present important similarities, as well as differences, in the array of behavioral, acoustic, cognitive and radiographic approaches to prosodic measures chosen to explore these questions.

2. Case description: Patient 1

The first patient, a right-handed, 36-year-old female African American, was found unconscious on the floor of her bathroom by her mother and taken to the hospital. The period of unconsciousness was estimated at 18+ hours. At the time of hospitalization, partial paralysis of her left arm and leg was present. Previous medical history is significant for mitral valve prolapse, dilaudid abuse, and a 10 year history of headaches. She reported no hearing or visual problems and there was no significant family history of neurologic or psychiatric illness. Initially, she was diagnosed as having suffered a deep midline infarction affecting basal ganglia bilaterally. CT scan at time of admission revealed bilateral infarcts of the basal ganglia (see Fig. 1). A T2-weighted and proton-density MRI images taken one day and two months later showed involvement of globus pallidus and medial putamen bilaterally. An FDG PET study taken two years post-onset of injury revealed bilateral hypometabolism in the caudate and putamen, more marked on the right side. Upon careful review of the history, clinical symptoms surrounding the episode of unconsciousness and neuroimaging results, the diagnosis was probable hypoxia, possibly as a result of a drug overdose.

Patient 1 had held her current job as a psychiatric nurse for 10 years prior to her episode of unconsciousness and

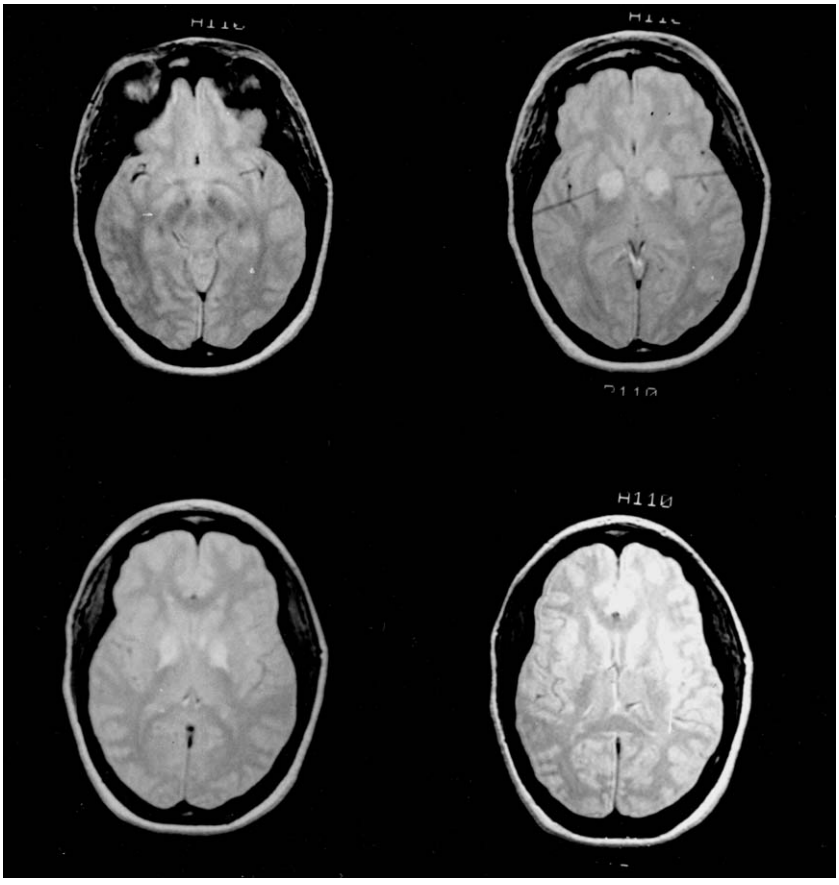

Fig. 1. A CT scan of Patient 1 revealed hyperintensity in the globus pallidus and medial putamen bilaterally.

returned to work 6 months later. The patient graduated from nursing school with good grades, but described her academic record as poor, especially in mathematics. No prior speech or language disturbance was reported, except for speech therapy in elementary school to correct a lisp. At examination, she denied problems finding her way, recognizing familiar faces, learning new faces, recognizing facial expressions (e.g., recognizing emotional states in her psychiatric patients); she also denied changes in color vision or hallucinations following her acute hospitalization.

While she no longer experienced physical weakness (limb weakness resolved within four months of the stroke), she described a general lack of interest in doing anything. Abulia, or "a lack of will or motivation to initiate… behavior," was a central feature of her neurobehavioral examination (Marin, 1990). She had no motivation, for example, for cleaning up the house, exercising, dating, conversation, social interaction, or sexual activity. She also reported a decrease in daydreaming and decreased recall of nighttime dreams. Her supervisor on the psychiatric unit counseled her about not interacting sufficiently with patients or staff, an admonishment that never occurred previously.

Patient 1 also has had mood alterations. Seven months following the incident she stated that her mood had "gotten better" since the incident, but on questioning an additional nine months later, she described her emotional state as "not happy," and perhaps more "sad," indicating further that she was not sure how she was feeling. Consonant with the abulia, she denied being fearful, and stated that she was experiencing generally less anxiety than previous to her hospitalization.

Patient 1 indicated that the most notable sequela of her injury was the change in her speech. Her conversational responses were strikingly monotonous, to the point of sounding like "synthetic speech." Her fiancé describes her as "sounding different… monotone," "passive and not as forceful" and being very different in her "ability to express her feelings in voice." He stated that her speech was more sparse, in that she "used to elaborate more." Indeed, she seldom initiated speech. She reported using fewer conversational expressions such as greetings and other speech formulas, and less swearing than in pre-injury communication. The fiancé described how he once induced her to "sound more like herself" by leading her to believe that his ex-girlfriend had called. Similarly, only a strong conversational stimulus induced a brief smile from Patient 1's otherwise expressionless face.

We were able to obtain pre-injury recorded telephone conversations of several minutes in length. Examination of an audio recording of a premorbid telephone conversation by Patient 1 revealed the voice of a playful, friendly young woman speaking Black American English dialect with a considerable dynamic range of expression (fundamental frequency (F_0) range from 135 to 420 Hz).

In summary, persisting neurobehavioral changes include monotone vocal expression and neutral facial expression, decreased overall verbal output with loss of formulaic expressions, difficulty initiating social speech, dramatically decreased motivation and initiative, fewer gestures with increased staring, and inconsistently reported dysphoria. According to her and her friends' reports, clinical examination, and study of several recordings of her speech, these symptoms are in stark contrast to her premorbid behavior, which was strikingly more ebullient. Following informed, written consent, Patient 1 was evaluated through clinical interviews, administration of standard protocols and specialized tests, and tape recording of speech, all performed between 7 months and 1 year post-onset of injury.

3. Neuropsychological evaluation: Patient 1

Table 1 summarizes the results of a number of neuropsychological tests administered to both patients. In general, Patient 1's performance on the Wechsler Adult Intelligence Scale-Revised (WAIS-R, Wechsler, 1981) was in the average to low average range (Verbal IQ = 90, Performance IQ = 77, Full Scale IQ = 83), and was consistent with her reported academic history. Performance was significantly below average only on the object assembly subtest (see Table 2).

Patient 1's performance was significantly below average on confrontation naming using the Boston Naming Test (Kaplan, Goodglass, & Weintraub, 1983), but her verbal fluency was within the normal range. Her abilities to generate word meaning (vocabulary), to recognize similarities, and to comprehend verbal material (comprehension) were all within normal limits. Her fund of general information (information) was within the normal range. Immediate and delayed recall on verbal memory tests was within the average range on the California Verbal Learning Test (Delis, Kramer, Kaplan, & Ober, 1987) as were the prose and paired associate subtests of the Wechsler Memory Scale-Revised (WMS-R) (Wechsler, 1981, 1987).

Performance on tests with visual-spatial demands was uneven. On the WAIS-R, the only significant evidence of below average performance was on object assembly. Performance was somewhat slow on both Trail Making A and B (Reitan, 1958). There was no evidence of visual neglect on line bisection (Lezak, 1983). Both copying and delayed reproduction were poor on the Rey Osterrieth Complex Figure (Osterrieth, 1944; Rey, 1941, 1964), but better on the immediate and delayed reproductions of the WMS-R.

Patient 1 also performed unevenly on tests of problem-solving or "executive functions." Performance was within normal limits on the digit symbol subtest of the WAIS-R, but below average on Trail Making A and B (Reitan, 1958). Her performance was abnormal on the Wisconsin Card Sorting Test (Heaton, 1981), with only three categories achieved and 54 (42.1%) perseverative errors, suggesting a perseverative set. However, perseveration was not a problem during her performance on the digit symbol subtest, nor on the Trail Making tests.

Table 2
Performance scores from neuropsychological testing for Patients 1 and 2

	Patient 1	Patient 2
Age	36	48
Education	16	20+
WAIS-R subtests (age-corrected scale scores: normal range = 10 ± 3)		
Information	7[b]	16[d]
Digit span	10	11
Vocabulary	9	14[c]
Arithmetic	6[b]	9
Comprehension	8	11
Similarities	10	18[d]
Picture completion	6[b]	17[d]
Picture arrangement	8	11
Block design	6[b]	[e]
Object assembly	4[a]	8
Digit symbol	7	[e]
WAIS-R summary scores (normal range = 100 ± 15)		
WAIS-R Verbal IQ	90	121[c]
WAIS-R Performance IQ	77[b]	112
WAIS-R Full Scale IQ	83[b]	118[c]
Boston Naming Test (number correctly named without phonemic cues)	46/60[a]	58/60
Verbal fluency test (FAS) (total words in 3 min)	42	32
Wisconsin Card Sorting Test		
Categories achieved	3[a]	6
Errors	66[a]	12
Failure to maintain set	1	0
Rey Osterrieth Complex Figure		
Copy	22/36[a]	36/36
Delay	10/36[a]	3/36[a]
Apathy Scale (higher score = greater apathy)	47[d]	18[a]
Affective-Prosodic Comprehension Test (percent correct)	94	94
Familiar and Novel Language Comprehension Test (FANL-C) (percent correct)		
Familiar phrases	65[a]	100
Novel phrases	90	100
Famous Voices Test (percent correct)		
Famous voice recognition	33[a]	92
Unfamiliar voice discrimination	96	98
Facial Recognition Test	43/54	54/54

[a] Significantly below average.
[b] Below average.
[c] Above average.
[d] Significantly above average, otherwise within the average range.
[e] Denotes missing data.

On extended special testing (see Table 1), the patient performed normally (94%) on the test of Affective/Prosodic Comprehension (Van Lancker, 1984; Van Lancker & Sidtis, 1992), in which she listened to sentences of neutral content spoken with happy, angry, surprised, or sad intonations, and responded by circling one of four responses. Comprehension of linguistic-prosodic contrasts, as in *green* house and green *house*, were also performed well. Moderately severe impairment in recognizing meanings of familiar non-literal expressions, idioms and speech formulas, was observed: on the Formulaic and Novel Language Comprehension Test (FANL-C), (Kempler & Van Lancker, 1988), Patient 1 identified only 65% of items correctly, while normal persons perform above 95% correct on this task. On the matched literal items, she identified 90% correctly.

On the Mini Inventory of Right Brain Injury (Pimenthal & Kingsbury, 1989), Patient 1's score was 34/43, placing her into the category of "mild right brain injury." She was impaired in recognizing familiar-famous voices (33% correct) but performed normally on unfamiliar voice discrimination (96% correct) (Van Lancker, Cummings, Kreiman, & Dobkin, 1988). Face perception tests revealed no deficit in processing familiar or unfamiliar faces: using a test of familiar-famous faces developed by Van Lancker and Nicklay (1992) (adapted from Albert, Butters, & Levin, 1979), she recognized faces at 93% correct, and her score on The Test of Facial Recognition was 43/54, in the average range (Benton & Van Allen, 1968; Benton, Hamsher, Varney, & Spreen, 1983).

With respect to mood and affect, Patient 1 was found to be profoundly apathetic, scoring 47 on the Apathy Scale (Marin, Biedrzycki, & Firinciogullari, 1991). This level of apathy was consistent with the patient's report of her reduced interaction with patients on her service, and in her reported loss of motivation in her activities of daily living. Repeated administrations of the Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961) at 7, 16, and 28 months after the injury indicated consistently elevated scores depression at the moderate to severe level (BDI scores of 24, 41, and 35, respectively). Unlike the apathy score, these measures were not entirely consistent with her subjective reports, in which she reported not being sure of feeling happy or sad, or she reported not being happy and also not being sad. Therapeutic trials of Elavil and Prozac were ineffective. In summary, neuropsychological testing was most notable for apathy and abulia.

4. Prosodic production: Patient 1

Although Patient 1's spontaneous speech was strikingly monotone, she remained a good singer. She sang alto in her church choir, and was observed by the examiners on repeated occasions to sing "Amazing Grace" and "America" ("My Country 'tis of Thee") (two songs with large and difficult pitch intervals) with excellent voice quality, pitch and meter. In addition, while her spontaneous speech was devoid of affective or attitudinal nuances, she was observed to imitate prosodic contrasts in speech relatively well. Therefore, further study of the prosodic aspects of her speech was undertaken. This consisted of a subjective listening task in which subjects judged Patient 1's speech, and an acoustic analysis of the patient's speech samples.

5. Listening task

5.1. Subjects

The listeners were 22 students and colleagues (9 males and 13 females), with no hearing deficits, ages 24–78, with a

mean age of 36. All spoke fluent English and all but one were native speakers of English, having educational backgrounds from undergraduate to postgraduate degrees, with a mean education of 16.5 years.

5.2. Stimuli

Patient 1 was asked to produce affective-prosodic utterances, and her efforts were tape-recorded. She was asked to say designated sentences (emotionally neutral in semantic content) twice each with a happy, angry, sad, or surprised intonation, resulting in eight utterances with prosodic contrasts relatively spontaneously generated by way of elicitation. Next, she was asked to imitate an examiner's (DS) renditions of those sentences said in those four intonations. She produced two utterances of each affective category (happy, sad, angry or surprised), resulting in a set of eight repeated utterances.

5.3. Tasks

The two sets of utterances, elicited and repeated, were used to prepare two separate perceptual tests for normal listeners. In the evaluation task, listeners were told to listen to the sixteen utterances, and circle "yes" if it was a "good" example of the intended affective category, "?" if it was a questionable product, or "no" if it was a poor example. The eight elicited sentences were presented first, followed by eight repetitions of a model sentence. For the identification task, elicited and repeated versions were presented twice (32 utterances), in random order to listeners, who were asked to "objectively" identify the utterances as "happy," "angry," "sad," or "surprised." All subjects performed the evaluation task first, followed by the identification task.

5.4. Results

Subjects more often evaluated the elicited sentences as "questionable" or "poor" examples of the intended affective-prosodic type, with only a few "good" ratings (33/176, or 18.5%), while rating a larger proportion of the repeated utterances, 132/176 (75%), as "good." This difference was significant by a Pearson Chi-Square test (Pearson $\chi^2 = 111.8$, $p < .0001$). A similar contrast between elicited and repeated utterances was seen in the identification task. For the elicited utterances, only 151 of a total of 352 affective-prosodic utterances were correctly identified (42.9%), while 257/352 of the repeated utterances (73.0%) were correctly identified. This difference was also significant (Pearson $\chi^2 = 65.5$, $p < .0001$). Thus by both evaluation and identification, Patient 1's elicited affective-prosodic utterances fared poorly on listening tests, while her utterances repeated after a model were reasonably successful (see Fig. 2).

These findings matched the impressions of the examiners during interaction with the patient, that she was vocally "competent" to produce prosodic variation, but that she did not do so in her spontaneous speech.

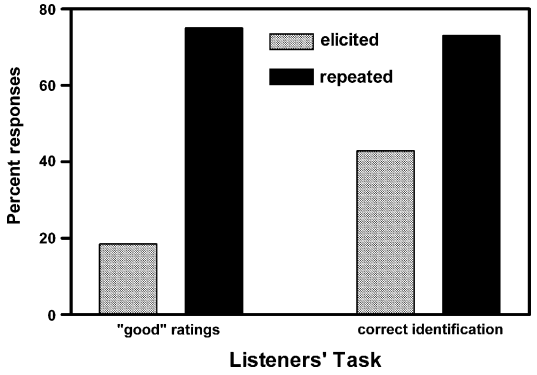

Fig. 2. Listeners' ratings of elicited and repeated utterances. Tasks were subjective evaluation of "goodness" of emotional type, and objective identification of emotion category.

6. Acoustic analysis: Elicited versus repeated sentences

Elicited and repeated sentences were subjected to acoustic analysis. Recorded material was digitized and fundamental frequency and timing measurements were carried out. Syllable boundaries were determined, and average fundamental frequency (F_0) was determined for each syllable. For each utterance, an F_0 mean and standard deviation was calculated using these syllable values. Mean F_0's produced during repetition were significantly higher than those elicited ($t(15) = 21.0$, $p < .0001$; see Fig. 3). Similarly, F_0 variability was significantly higher during repetition compared to those elicited ($t(15) = 7.0$, $p < .0001$; see Fig. 3).

Fig. 4 reveals this pattern in more detail. In Fig. 4, values represented by the open squares and broken lines reflecting F_0 means for each of affective utterance types, when elicited from Patient 1, show little difference in mean F_0. A one-way Analysis of Variance (ANOVA) comparing the four emotions resulted in a no significant F_0 differences between affective types. In contrast, the closed squares and solid lines, representing repeated utterances, reveal significant differences in F_0 across the four affective types ($F(3,7) = 7.0$, $p < .05$). A contrast is seen with Patient 2, who is described below.

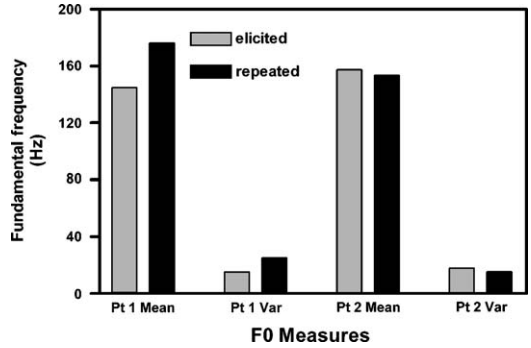

Fig. 3. Fundamental frequency (F_0) mean and variation for Patients 1 and 2 for elicited and repeated affective-prosodic utterances. Elicited versus repeated values differed for Patient 1 but not Patient 2.

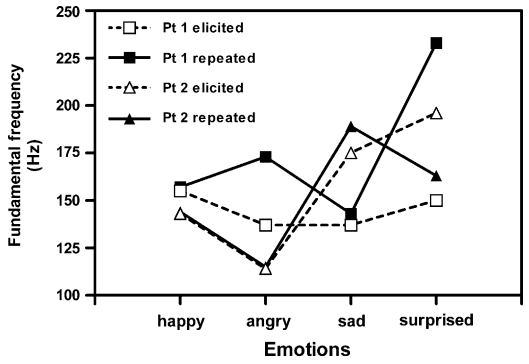

Fig. 4. Affective-prosodic utterances by emotion type (happy, angry, sad, surprised) of Patients 1 and 2 comparing mean fundamental frequency (F_0) measures for elicited and repeated utterances. Values for Patient 1 (squares) differ for elicited (open symbols) and repeated (filled-in symbols) modes, while values for Patient 2 (triangles) do not.

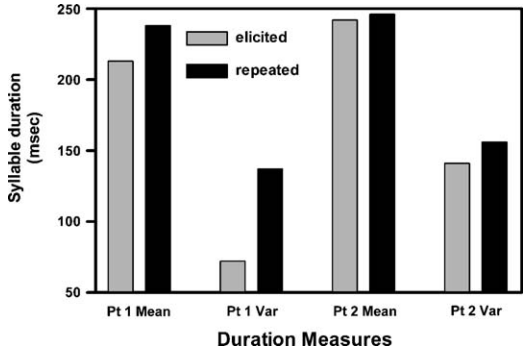

Fig. 5. Duration mean and variation comparing elicited and repeated utterances for Patients 1 and 2. Values differ for Patient 1 but not for Patient 2.

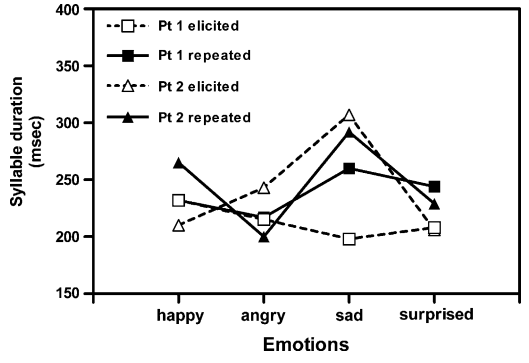

Fig. 6. Affective-prosodic utterances by emotion type of Patients 1 and 2, comparing mean durations for elicited and repeated utterances. Values for Patient 1 (squares) differ for elicited (open symbols) and repeated (filled-in symbols) modes, while values for Patient 2 (triangles) do not.

Syllable boundaries were also used to determine mean and standard deviations for syllable durations. Duration values showed a similar pattern, distinguishing between speech modes (see Fig. 5). Significant differences between elicited and repeated affective utterances were seen for duration mean ($t(15) = 38.0$, $p < .0005$) and in duration variation ($t(15) = 9.0$, $p < .0001$). Individual affective types suggest an effect of utterance mode on duration (see Fig. 6), but the differences were not statistically significant.

The premorbid speech sample, consisting of two brief conversations recorded on an answering machine, contained a broad range of prosodic variation (135–420 Hz), confirming the reports by the patient and her acquaintances of dramatic change in her vocal expression. Further direct comparison of this relatively limited set of utterances with the affective-prosodic utterances that were acoustically analyzed was not possible.

7. Case description: Patient 2

The second patient, a right-handed, 48-year-old male Caucasian with a Ph.D. in archeology, whose occupation is art museum executive, suffered a hemorrhagic infarct in the right basal ganglia, resulting in dysarthria and left hemiplegia. Early left-sided neglect resolved during the initial hospitalization. A T1-weighted MRI at the time of admission revealed an infarct of the right putamen, right globus pallidus and the posterior limb of the internal capsule on the right (Fig. 7). The patient had no prior history of neurological deficits. Persisting neurobehavioral symptoms included relatively monotonous vocal expression, increased irritability and disinhibition, more aggressive conversational style, and neutral facial expression with decreased facial gestures.

Patient and close acquaintances described his speech as significantly changed from his premorbid pattern, now being less expressive and sounding "irritable" following his stroke. With respect to turn-taking, in contrast to Patient 1, who suffered loss of self-initiated conversational speech formulas, Patient 2 complained of interrupting people more in conversation, and having difficulty controlling this new practice. He stated that he had less control over his speech and expression; that he previously could imitate dialects (e.g., British English) well enough to "pass" as a speaker of various different geographical areas, but that he had totally lost that ability since his stroke. He also reported losing his ability to sing as well as before. His voice was breathy and monotone and speech sounded hurried.

For this patient, also, we were able to obtain pre-injury speech samples. In this case, videotaped art history interviews televised during the two-year period previous to his stroke revealed that Patient 2 possessed an animated, charismatic quality in vocal expression, missing from his post-stroke speech. Videotaped programs recorded post-injury and conducted in similar format were obtained for comparative acoustic studies. Patient 2 was evaluated through clinical interviews, administration of standard protocols and specialized tests, and tape recording of speech, all performed between seven months and one year post-onset of injury.

8. Neuropsychological evaluation: Patient 2

In general, Patient 2's performance on the WAIS-R was in the average to above-average range, with a higher level on the verbal subtests than on the performance subtests.

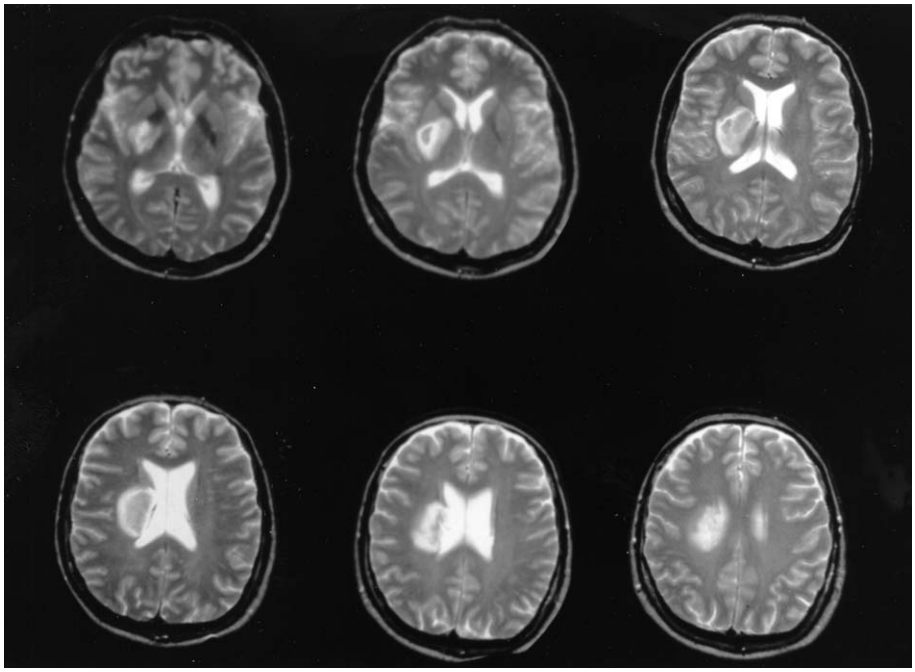

Fig. 7. A CT scan for Patient 2 revealing right-sided infarction of the putamen, globus pallidus and posterior limb of the internal capsule.

No significant abnormalities were noted on these measures (see Table 1).

In contrast with Patient 1, Patient 2's performance was better on confrontation naming than on verbal fluency, where performance was below average but not significantly so. His abilities to generate word meanings (vocabulary), to recognize similarities, and to comprehend verbal material were all within normal limits. His fund of general knowledge was better than average. Immediate recall was within the average range on the Rey Auditory Verbal Learning Test (Lezak, 1983; Rey, 1964). Delayed recall was below average, but not significantly so.

Like Patient 1, Patient 2 was able to normally identify affective-prosodic contrasts (Van Lancker & Sidtis, 1992), and he could both comprehend and produce linguistic-prosodic contrasts. He showed no difficulty with nonliteral utterances on the Formulaic and Novel Language Comprehension Test (FANL-C; Kempler & Van Lancker, 1988), but performed deficiently on the Inference Test (Brownell, Potter, Bihrle, & Gardner, 1986) (75% on inferential items, 94% on factual items). His performance on familiar voice recognition and unfamiliar voice perception protocols (Van Lancker & Kreiman, 1986, 1987) was normal.

Performance on tests with visual-spatial demands was generally intact. As noted above, performance on WAIS-R subtests was in the average to above average range. Copying was normal on the Rey Osterrieth Complex Figure but delayed reproduction was poor. No other visual memory tests were administered due to time constraints. Facial recognition was errorless for famous as well as novel faces.

Patient 2 performed well in problem solving or "executive function," achieving 6 categories with few perseverative errors on the Wisconsin Card Sorting Test.

With respect to psychological state, Patient 2 was found to have a significant lack of apathy, scoring 18 on the Apathy Scale. The Beck Depression Inventory was not administered secondary to an absence of subjective complaints of depression and time limitations with this patient.

In summary, neuropsychological testing was notable for poor delayed reproduction of figural information, and less pronounced reduced levels of performance in delayed verbal recall and verbal fluency. Apathy was not present.

9. Prosodic production: Patient 2

9.1. Acoustic analysis: Elicited versus repeated sentences

On formal clinical testing, Patient 2 was judged by the clinical examiners to be competent in producing elicited and repeated affective-prosodic utterances, so that tasks involving listening and identification responses were not conducted with normal listeners. This observation was confirmed by quantitative study of elicited and repeated utterances, which were subjected to the same acoustic analysis described for Patient 1. Fig. 3 shows no significant difference in F_0 mean or variation on elicited and repeated affective-prosodic utterances for Patient 2. In Fig. 4, values represented by the triangles reflecting F_0 means for each of the 4 affective utterances, when elicited from Patient 2, show a large difference in mean F_0 for both elicited (open triangles) and repeated (closed triangles) utterances, but there were no significant differences between elicited and repeated utterances across emotion type for either F_0 measure. One-way ANOVAs comparing the four emotions resulted in significant differences for F_0 mean, for both elicited ($F(3,7)=94.5$, $p<.001$) and repeated ($F(3,7)=27.4$, $p<.01$) utterances, but not for F_0 variation.

No significant differences between elicited and repeated affective utterances emerged for duration mean or variation (see Fig. 5). Duration values on individual affective types show a similar pattern (see Fig. 6), with significant differences in duration means between affective types (elicited: $F(3,7) = 11$, $p < .01$; repeated: $F(3,7) = 9$, $p < .05$), but no significant differences between emotions on the duration variability measure.

9.2. Acoustic analysis: Premorbid versus post-morbid speech

As stated above, a main complaint from the subject and his acquaintances was change in expressiveness in speech. Clinically, his poststroke speech was relatively monotonous, mildly slurred, breathy, intermittently dysfluent, and hypophonic. Audition of well-matched pre- and post-morbid speech samples suggested reduction in vocal expressiveness, focused on reduced intonation variability. Availability of comparable pre and post-morbid speech materials made possible an acoustic comparison, achieved by analyzing portions of the subject's speech recorded from three videotaped television shows (two shows recorded before the stroke and one show recorded six months following the stroke), all utilizing a similar format, in which a host (Patient 2) and two other persons discussed art works.

Nineteen contiguous breath groups from pre- and post-morbid speech samples were digitized and measures of F_0 mean and variation, as well as syllable and breath group durations were made as previously described. Premorbid mean breath-group durations (2.6 ± 1.3 s) were significantly longer than post-morbid breath group durations (1.8 ± 1.0 s), ($t(36) = 2.18$; $p < .05$). Although rate appeared to be somewhat faster, there were no significant differences in rate of syllable production (premorbid $= 5.6 \pm 1.3$ syllables per second; post-morbid $= 5.9 \pm 1.0$). Premorbid mean F_0 (121.4 ± 8.2 Hz) was significantly higher than post-morbid mean F_0 (113.6 ± 3.9 Hz) ($t(36) = 3.71$; $p < .01$). Similarly, premorbid F_0 variability (20.7 ± 7.2 Hz) was significantly higher than post-morbid F_0 variability (16.5 ± 3.0 Hz) ($t(36) = 2.35$; $p < .05$) (see Fig. 8).

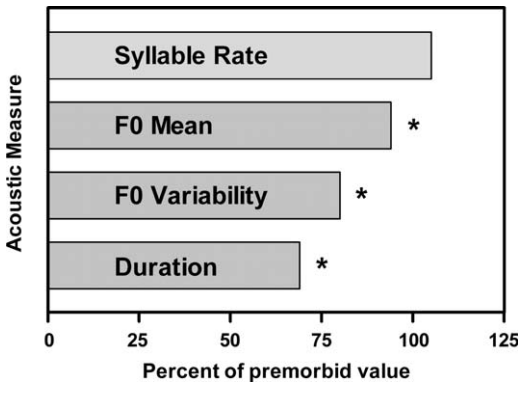

Fig. 8. Change in four speech measures following neurological damage for Patient 2: Fundamental frequency (F_0) mean and variation, speech rate and mean length of breath groups were compared from pre-morbid and post-morbid speech samples. The asterisks indicate a significant difference in speech comparing before and after injury utterances. F_0 mean and variability were significantly lowered and reduced, and syllable durations were significantly shorter. Speech rate was slightly (but not significantly) faster.

10. Discussion

Two patients with lesions of subcortical structures were brought to our attention because of alterations in speech prosody. Structural and functional neuroimaging studies revealed damage confined to subcortical areas, primarily globus pallidus and putamen bilaterally in Patient 1 (MRI, CT, and PET), and right putamen, globus pallidus, and internal capsule in Patient 2 (CT, MRI). Studies have shown a distant effect of subcortical infarcts in cortical blood flow, usually in association with aphasia or neglect (Hillis et al., 2002; Kang et al., 2000; Olsen, Bruhn, & Oberg, 1986; Weiller et al., 1993). However, no cortical involvement was in evidence in either patient. Although remote metabolic effects cannot be completely ruled out for these two cases, no evidence was seen in radiological studies, and no aphasia or persistent neglect was observed. Further, analysis of a resting PET scan revealed areas of hypometabolism only in the basal ganglia nuclei identified as sites of damage for Patient 1.

In Patient 1, speech was monotonous and spontaneous conversational expressions were lacking. In Patient 2, speech was reduced in expressivity, and communication style was impulsive. Comparisons of pre- and post-morbid speech samples from both patients supported the patients' assertions that these changes occurred as sequelae of the neurological incident. Despite these expressive-prosodic changes, neither patient suffered from impaired comprehension of affective- or linguistic-prosodic utterances.

Results of listening tests demonstrated that Patient 1's attempts to spontaneously produce affective prosody (via elicitation) were significantly less effective than her performance on repetition of affectively intoned statements. These differences were reflected in acoustic analyses of her utterances, which demonstrated acoustic differences between affective modes during repetitions but not during her spontaneous (elicited) productions. This individual had suffered a change in speech function that affected her social and occupational relationships. In contrast, Patient 2, by clinicians' judgements, produced effective affective utterances both spontaneously and during repetition. This ability was reflected in significant acoustic differences between affective modes for both types of utterances, elicited and repeated. Yet this individual also had suffered a change in prosodic expression which interfered with his communication in social and professional contexts.

Loss of motor control was not responsible for the patients' complaints. Patient 1 could accurately sing even difficult melodies, and she performed significantly better during repetition than during spontaneous expression. Patient 2 performed well in formal testing of affective expression. Their problems with prosody were not due to non-specific restrictions in vocal range or control

(Duffy, 1995; Sidtis, 1984). Their expressive dysfunction was also not restricted to affective prosody, since the changes in speech pattern did not occur only during attempts at emotional expression but, rather, were a consistent feature of their post-injury speech.

It is of interest to consider these two cases in the context of current models of cerebral representation of prosodic competence. These cases cannot be accommodated in either the functional or physical feature hemispheric models. While these models doubtless have some validity, subcortical cases require an amended or expanded model of brain/behavior relations for prosody. In this report, we examine how neurobehavioral mood and motivation changes following subcortical disease are associated with prosodic dysfunction. These considerations lead to a broader perspective for a model of brain–behavior relationships for prosody. We first consider two questions, the first involving quantification methods for prosody, and the second examining the role of neurobehavioral disorders in basal ganglia disease in dysprosody.

The first question addresses our ability to quantify and characterize a prosodic deficit with formal tests and analyses. The influence of task is one consideration. The ability of both patients in this study to perform better in selected structured test situations than in normal speech use, and of Patient 1 to produce adequate examplars during repetition but not during elicitation should provide a caution regarding post-injury experimental manipulations of prosodic demands, especially in the absence of a comprehensive conceptual framework for prosody. Pending a better understanding of prosody, a rigid approach to formal testing based on a narrow or simplistic view of prosody may well be insensitive to problems experienced by patients in their normal spontaneous speech, and it may also misrepresent a feature of prosodic expression as specifically "linguistic, " "affective," or "pragmatic" when it is, in fact, general. Studies of individual cases are desirable at this time, and comparisons between pre- and post-brain injury speech, whenever possible, have good potential for insights into prosodic disturbances.

In the pursuit of quantification standards, the several domains of prosody require careful consideration. Normal prosodic function involves a complex integration of the acoustic cues of pitch, timing, loudness, and voice quality. Although evidence for independent functions of prosodic elements has been reported (Ladd, Silverman, Tolkmitt, Bergman, & Scherer, 1985; McRoberts, Studdert-Kennedy, & Shankweiler, 1995), these elements are difficult to discern impressionically (Fry, 1970; Kent, 1996; Melara & Marks, 1990). In some cases, independent listening studies might be indicated to verify the clinician's impressions (as is done in intelligibility testing; see Yorkston & Beukelman, 1981). An impairment in expression or reception of prosodic elements—pitch or timing, for example, may account for the presentation of dysprosody. These elements all interact to functionally signal meanings on variously sized temporal units including phone, syllable, word, clause, and breath group (Hird & Kirsner, 2002). Some findings have depended on the size of the unit used in prosodic measurements—whether single word, "phrase," or breath group (e.g., Behrens, 1989; Danly & Shapiro, 1982; Danly, Cooper, & Shapiro, 1983; Gandour, Petty, & Dardarananda, 1989). Acquired dysprosodic disturbances can be expected to involve a combination of classes of prosodic function, and of a range of measurable acoustic components.

The second question, the relevance of internal neurobehaviorally describable states (mood, motivation, emotional experiencing) to prosodic performance, perhaps as involving subcortical structures, has begun to receive attention. Skin conductance studies suggested reduced emotional responsiveness in RH damage (Meadows & Kaplan, 1994), probably reflected in impoverished lexicon for emotional words (Borod, Bloom, & Haywood, 1998). Case studies revealed a fundamental emotional deficit in frontotemporal dementia (Perry et al., 2001), while related findings associate affective-dysprosodic disorders with emotional disturbances seen in frontal lobe damage (Hornak, Rolls, & Wade, 1996). In the present study, in neurobehavioral testing, Patient 1 was found to be profoundly abulic. In contrast, Patient 2 did not demonstrate abulia, but did evince increased irritability and disinhibition. Despite the alterations in motivation, mood and expressive prosody that followed subcortical damage, both patients in this study experienced unchanged ability to successfully identify affective states in others, to experience a range of affective states within themselves, and to mimic the affective prosody of others. Was the relatively greater severity of Patient 1's dysprosody related to her neurobehavioral changes, or were both (dysprosody and abulia) independent sequelae of her lesion? The question of causality versus correlation in the possible relationship between neurobehavioral disorder and dysprosody is not likely to be answered simply. This question was addressed when a dissociation between mood and affective prosodic comprehension disturbance in PD was reported by Pell and Leonard (2003).

Rather than simply viewing expressive dysprosody as an independent symptom, this disorder might be best appreciated in the context of a syndrome or symptom complex. In the patients presented in this report, expressive dysprosody was accompanied by reduced motivation or changes in psychological state (e.g., abulia, irritability, dysphoria), symptoms consistent with basal ganglia damage (see Cummings, 1993; for a review). In this respect, it is important to note that neither patient was incapable of the expression of affective prosody, but instead, their injuries disrupted the spontaneous, natural expression of prosody. The extent to which these changes represent more general problems with "initiation" or "motivation" needs to be addressed, but the answers will only be found within the broader context of understanding the neurological syndromes in which dysprosody occurs.

As in the present two cases, a role for subcortical structures in prosody (Ackermann et al., 1993; Cancelliere & Kertesz, 1990; Cohen, Riccio, & Flannery, 1994; Scott, Caird, & Williams, 1984) has been demonstrated. Similar

changes have been linked to observations of changes in psychological state and motivation (Bhatia & Marsden, 1994; Poncet & Habib, 1994), or as one group states, "loss of action initiation and maintaining" (Ali Cherif et al., 1984). Numerous descriptions in lesion studies appear which, on close examination of the clinical details, implicate prosodic disturbance in the neurobehavioral disability, in some cases utilizing the dysprosodic sign as a major clue to diagnosis.

Caplan et al. (1990), in discussing behavioral changes in motivational capacity following caudate damage, refer to "decreased speech" (p. 135). Caplan et al. (1990) characterize abulia as "lacking in spontaneity of action and speech … Verbal responses are late, terse, incomplete, and emotionally flat, but the intellectual content is normal" (p. 139). This exactly describes Patient 1 of this report. Lesions to orbital-frontal cortex, anterior cingulate (Cummings, 1993; Mendez, Adams, & Lewandowski, 1989) and the basal ganglia structures (Taylor, Saint-Cyr, & Lang, 1990) can produce behavioral changes including apathy, abulia and difficulty initiating and maintaining actions. Key to diagnosing these neurobehavioral disorders in many cases is dysprosodic speech.

The scientific literature on emotional processing points to a considerable role of the basal ganglia (Mayeux, 1983; Starkstein, Robinson, Berthier, Parikh, & Price, 1988; Weddell, 1994) and limbic system (Robinson, 1976; Van Lancker & Cummings, 1999). Bhatia and Marsden (1994) provided an analysis of disturbances in 240 patients with focal basal ganglia lesions of mostly vascular etiology. The most common behavioral symptom (28% of unilateral damage to the caudate nucleus) was a loss of spontaneous emotional and cognitive responses, or "abulia." Bilateral lesions of the globus pallidus were also associated with abulia (p. 868), for which a defining feature is the "loss of emotional affective expression" (p. 860; see also Fisher, 1983). "Dysarthria with dysprosody" (p. 863) was associated with small unilateral putamenal lesions, and "hypophonia" in a patient with unilateral caudate and lentiform lesions. In his review of behavioral changes following subcortical damage, Tranel (1992) associates "defects in articulation and prosody" with lesions of the putamen (p. 82). In their overview of clinical presentations, Saint-Cyr, Taylor, and Nicholson (1995, p. 14) used terms such as "emotional limitations" and "reduced drive or motivation" (p. 14), attributing to the basal ganglia "the establishment and selection of emotional responses" (p. 20). In three patients with bilateral basal ganglia lesions, affect is described as "disturbed" in the sense of an "indifferent attitude" (p. 377), "impaired" in the sense of "inactive," and dramatically "passive" (p. 379) (Laplane, Baulac, Widlöcher, & DuBois, 1984).

Patients who suffered bilateral globus pallidus lesions demonstrated "apparent affective indifference connected with a lack of spontaneous expression of the affects," yet were able to "bear witness to their feelings and emotions, but always in a cold and purely verbal manner" (Ali Cherif et al., 1984, p. 401). Poncet and Habib (1994), describing "motivational" (p. 588) disorders in association with damage to (bilateral) globus pallidus and adjacent structures, observe that "Spontaneous expression of feelings and emotions is absent or very impoverished" (p. 591). However, as in the previous example, these patients were able to say whether or not they are enjoying situations, indicating that mood was intact despite impoverished mood expression. Mendez et al. (1989) described a group of patients with lesions in the caudate nucleus, as being in an "apathetic state," with verbalization consisting of a few words. It likely that one of the indicators of emotional and motivational disorders described in these cases lies in "flat speech." The neurobehavioral description thus flows in part from the presentation of the speech pattern, which is dysprosodic—low in pitch and intensity mean, range and variability, but without articulatory disturbances.

These incidents of intonational failure may bear a significant relationship to comparable findings for other motor behaviors. Motor initiation deficits are seen in association with frontosubcortical damage. A syndrome of "gait ignition failure" (Atchison, Thomas, Frackowiak, & Marsden, 1993), an inability to initiate walking without other motor disorders, in cases with suspected frontal lobe vascular disease and/or focal degeneration has been described. Focal lesions to the basal ganglia and neurodegenerative processes such as Parkinson's and Huntington's diseases (Cohen, Laframboise, Labelle, & Bouchard, 1993; Cools, van den Bercken, Horstink, van Spaendonck, & Berger, 1984; Fraile, Masson, & Cohen, 1994; Kent & Rosenbek, 1982; Pillon, Dubois, Lhermitte, & Agid, 1986) are associated with difficulties in initiating output behaviors and can also result in a syndrome that includes alterations in psychological state, motivation and prosody (Pillon et al., 1986; Speedie et al., 1990). Mood disorders are associated with both subcortical (Masterman & Cummings, 1997; Santamaria & Tolosa, 1992) and cortical (Cummings, 1985; Robinsin, Kubos, Starr, Rao, & Price, 1984) insult, and must be considered in any clinical presentation of dysprosody.

We propose a conceptual framework for the study of dysprosody that encompasses the associated neurological signs and symptoms together with the brain regions affected (Table 3). In this framework, dysprosody may occur as a syndrome with widespread behavioral alterations, or as a relatively isolated deficit. Production or perception of any or some of the individual elements of prosody may be disabled by damage at various levels of the nervous system. Subcortical damage may result in syndromes involving alteration of motivation, initiation, psychological states, and/or motor ability, which may include clinically dysprosodic speech (Cummings, 1985). Subcortical injury has also been associated with rate dysfunction in spoken language in Parkinson's disease (Duffy, 1995) and in errors of timing perception in speech (Breitenstein, Van Lancker, Daum, & Waters, 2001; Gräber, Hertrich, Daum, Spieker, & Ackermann, 2002). Thalamic damage has led to specific timing abnormalities in speech (Canter & Van Lancker, 1985). Cerebellar damage is frequently

Table 3
Brain–behavior model for various sources of dysprosody

Chief feature of clinical abnormality	Typical neuropathogenesis
Motor control	
Paresis or paralysis	Focal cortical damage
"Prosodic dysarthria"	Focal cortical damage
Timing, coordination	Cerebellar disease
Motor control with behavioral sequelae	
Difficulty with movement initiation and maintenance; depression	Basal ganglia; e.g., Parkinson's disease
Adventitious movements; psychosis	Basal ganglia; e.g., Huntington's chorea
Behavioral syndrome	
Apathy, abulia, akinesia	Basal ganglia; anterior cingulate
Irritability, impulsiveness	Basal ganglia; orbitofrontal
"Representational"	
"Prosodic agnosia" (?)	Focal cortical (right hemisphere?)
"Prosodic anomia" (?)	Focal cortical (left or right hemisphere?)
"Prosodic apraxia"	Focal cortical (left hemisphere?)

This model presents a theoretical overview of proposed cortical-hemispheric and subcortical etiologies. This is a conceptual framework for studying dysprosody and some sites of damage or disease processes typically associated with its features. The extent to which a disorder of prosody is associated with a motor or perceptual deficit, a mixed motor-behavioral syndrome, or a behavioral syndrome depends on the site of the lesion and the disease process. A disorder of prosody may have more of an emotional flavor when a behavioral syndrome is present, but this likely reflects the nature of the syndrome more than any features of "affective" prosody. Compared to aphasia, the clinical presentation of disordered prosody can result from damage to a large number of brain regions, most of which are not homologous to language areas. "Prosodic agnosia" may theoretically include any of the functional prosodic categories in Table 1 (adapted from Sidtis and Van Lancker Sidtis, 2003).

associated with ataxia of speech, affecting mainly timing and coordination (Ackermann, Gräber, Hertrich, & Daum, 1997; Cole, 1971; Fine, Ionita, & Lohr, 2002; Le Dorze, Ryalls, Brassard, Boulanger, & Ratte, 1998). Thus, selective interference with the elements of prosody (pitch, intensity, timing, voice quality), whether in production or perception, may be central in the clinical presentation of dysprosody, and conferring a cognitive or functional interpretation in these cases would be in error. For example, Van Putten and Walker (2003) found that listeners were unable to categorize affectively intoned expressions in apraxic speakers, but while we can conclude that these LH damaged patients had some prosodic production deficit, no assay of other prosodic functions were attempted, nor were the acoustic deviations identified. From this study, we cannot tell whether the deficit was to a single or to multiple prosodic functions, and we cannot tell whether the deficit involved one or more of the prosodic elements (e.g., pitch control).

Cortical damage, theoretically, might produce agnosia for meanings of prosodic patterns in production or comprehension of any of the five major prosodic functions; apraxia for the execution of those patterns; or anomia for the labeling or lexical categorization of prosodic meanings. These conditions could arise from LH or RH cortical damage. One innovative approach to prosodic apraxia differentiates between intrinsic and extrinsic prosody in speech production (Boutsen & Christman, 2002). Cortical damage could also produce a global "dysarthria" of prosodic output (Sidtis, 1984). One promising direction is to examine preserved prosodic function in severe, congenital dysarthria (Patel, 2002).

It is unclear from published reports how to predict cerebral laterality for any of these clinical presentations. Claims in the literature for right cortical representation of prosody (Borod, 1992, 1993; Heilman, Scholes, & Watson, 1975; Heilman, Bowers, Speedie, & Coslett, 1984) presumably refer to a specific agnosia—inability to retrieve (access) or comprehend affective-prosodic meanings, resulting in impaired prosodic output, or to an anomia, difficulty in retrieving emotional words. For some time (Mills, 1912; Wechsler, 1973), the RH has been held responsible for much of emotional processing, in production and comprehension modes (Borod et al., 1996; Heilman, 1997), leading to notions of a RH specialization for "affective lexicon" (Bowers, Bauer, & Heilman, 1993; Rapcsak, Comer, & Rubens, 1993) and personal relevance (Cicone, Wapner, & Gardner, 1980; Cimino, Verfaellie, Bowers, & Heilman, 1991; Van Lancker, 1991).

In earlier reports describing these putative cortical dysprosodies, other possible clinical interpretations of impaired performance were usually not considered, and the incidence of these conditions following brain damage remains substantiated. Impaired "melody of speech" of an apraxic variety has traditionally been ascribed to the damaged left cortical hemisphere (De Bleser & Poeck, 1985; Goodglass & Kaplan, 1983; Luria, 1966).

Finally, this report highlights the importance of speech mode or task in the presentation of prosodic deficit. Table 4 presents a schema of expected prosodic deficit, based on the neurobehavioral model, as revealed in the modes of expression, repetition, perception and cognition. Careful examination of the array of prosodic functions and elements across these performance modalities may hold the promise of a true identification of dysprosody in the clinical setting.

11. Summary

Prosody has a range of functions in speech, of which at least five can be identified; at least four auditory/acoustic elements are woven into the speech signal. Whether and under what conditions brain damage affects these functions and elements selectively is as yet unclear. Normal ranges of prosody are not well described or understood, and much individual variation exists across normal speakers. Therefore, better understanding of normal prosody, as well as information about pre-injury speech patterns, as can be best derived from single case studies, are important to determining presence or absence of post-morbid dysprosody, its exact nature, and its likely etiology. Abilities may vary with speech performance

Table 4
Schema of prosodic competence coordinated with speech performance task

	Dysprosody in communicative tasks			
	Expression	Repetition	Perception	Cognition
Features				
Timing (rhythm)	xx	xx	x	0
Pitch	xx	xx	x	0
Intensity	xx	xx	0	0
Vocal quality	xx	xx	0	0
Motor control				
Movement disorders	xx	x	x	x
Depression, psychosis	xx	x	x	x
Behavioral syndromes				
Apathy, abulia, akinesia	xx	0	0	0
Irritability, impulsiveness	xx	0	0	0
Psychiatric disorders	xx	0	0	0
Representational deficits				
Prosodic agnosia	xx	0	0	xx
Prosodic anomia	0	0	0	xx
Prosodic apraxia	xx	x	0	0

Expected clinical presentation of dysprosody in expression, repetition, perception, and cognition, with "xx" and "x" standing for more and less severe, and normal as "0." Evaluation of prosodic competence in these various modes can aid in diagnosis. Examination of prosodic elements (timing, pitch, intensity, quality) and speech/language tasks (expression, repetition, perception, cognition), as well as representational systems (apraxia, anomia, agnosia) for prosodic functions (linguistic, affective, pragmatic, indexical) may lead to more accurate diagnosis of prosodic disturbance.

mode (expression, repetition, perception), such that differential performance on speech task (Hird & Kirsner, 2002; Kempler & Van Lancker, 2002; Wertz, Henschel, Auther, Ashford, & Kirshner, 1998) may be an important clue to diagnosis. Whether all or only some individual cognitively described prosodic functions (e.g., linguistic, pragmatic) are selectively affected by brain damage must be examined and studied in this broader context (Table 4). The case presentations reported here—as well as the review of prosody studies—lead to the conclusion that dysprosody has various etiologies; that classic neurobehavioral, auditory, or motor functions may account for many observed deficits; and finally, that acquired prosodic disturbance can result from damage to any of a number of cerebral subsystems.

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References

Ackermann, H., Gräber, S., Hertrich, L., & Daum, I. (1997). Categorical speech perception in cerebellar disorders. *Brain and Language, 60*, 323–333.

Ackermann, H., Hertrich, I., & Ziegler, W. (1993). Prosodische Störungen bei neurologischen Erkrankungen—eine Literaturübersicht [Prosodic disorders in neurological diseases—A review of the literature]. *Fortschritte der Neurologie Psychiatrie, 61*, 241–253.

Albert, M. S., Butters, N., & Levin, J. (1979). Temporal gradients in the retrograde amnesia of patients with alcoholic Korsakoff's disease. *Archives of Neurology, 36*, 211–216.

Alexander, G. E., Crutcher, M. D., & DeLong, M. R. (1990). Basal ganglia-thalamocortical circuits: Parallel substrates for motor, oculomotor, "prefrontal", and "limbic" functions. *Progress in Brain Research, 85*, 119–146.

Alexander, M. P., Benson, D. F., & Stuss, D. T. (1989). Frontal lobes and language. *Brain and Language, 37*, 656–691.

Ali Cherif, A., Royere, M. L., Gosset, A., Poncet, M., Salamon, G., & Khalil, R. (1984). Troubles du comportement et de l'activité mentale après intoxication oxycarbonée: Lesions pallidales bilaterales. *Review Neurologique, 140*, 32–40.

Alm, P. A. (2004). Stuttering and the basal ganglia circuits: a critical review of possible relations. *Journal of Communication Disorders, 37*, 325–369.

Alpert, M. (1982). Encoding of feelings in voice. In P. J. Clayton & J. E. Barrett (Eds.), *Treatment of depressions: Old controversies and new approaches* (pp. 217–228). New York: Raven Press.

Apple, W., Streeter, L. A., & Krauss, R. M. (1979). The effects of pitch and speech rate on personal attributions. *Journal of Personality and Social Psychology, 37*, 715–727.

Atchison, P. R., Thomas, P. D., Frackowiak, R. S., & Marsden, C. D. (1993). The syndrome of gait ignition failure: A report of six cases. *Movement Disorders, 8*, 285–292.

Banse, R., & Scherer, K. R. (1996). Acoustic profiles in vocal emotion expression. *Journal of Personality and Social Psychology, 70*, 614–636.

Barrett, A. M., Crucian, G. P., Raymer, A. M., & Heilman, K. M. (1999). Spared comprehension of emotional prosody in a patient with global aphasia. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology, 12*(2), 117–120.

Baum, S. R. (1992). The influence of word length on syllable duration in aphasia: acoustic analyses. *Aphasiology, 6*, 501–513.

Baum, S. R. (1998). The role of fundamental frequency and duration in the perception of linguistic stress by individuals with brain damage. *Journal of Speech, Language, and Hearing Research, 41*, 31–40.

Baum, S. R., & Pell, M. D. (1997). Production of affective and linguistic prosody by brain-damaged patients. *Aphasiology, 11*, 177–198.

Baum, S. R., & Pell, M. D. (1999). The neural bases of prosody: Insights from lesion studies and neuroimaging. *Aphasiology, 13*(8), 581–608.

Baum, S. R., Blumstein, S. E., Naeser, M. A., & Palumbo, C. L. (1990). Temporal dimensions of consonant and vowel production: An acoustic and CT scan analysis of aphasic speech. *Brain and Language, 39*, 33–56.

Baum, S. R., Pell, M. D., Leonard, C. L., & Gordon, J. K. (1997). The ability of right and left-hemisphere damaged individuals to produce and interpret prosodic cues marking phrasal boundaries. *Language and Speech, 40*(4), 313–330.

Baum, S. R., Pell, M. D., Leonard, C. L., & Gordon, J. K. (2001). Using prosody to resolve temporary syntactic ambiguities in speech production: acoustic data on brain-damaged speakers. *Clinical Linguistics and Phonetics*, 441–456.

Beck, A. T., Ward, C. H., Mendelson, M., Mock, J., & Erbaugh, J. K. (1961). An inventory for measuring depression. *Archives of General Psychiatry, 4*, 561–571.

Behrens, S. J. (1989). Characterizing sentence intonation in a right hemisphere-damaged population. *Brain and Language, 37*, 181–200.

Benke, T., Bosch, S., & Andree, B. (1998). A study of emotional processing in Parkinson's disease. *Brain and Cognition, 38*(1), 36–52.

Benton, A. L., & Van Allen, M. W. (1968). Impairment in facial recognition in patients with cerebral disease. *Transactions of the American Neurological Association, 93*, 38–42.

Benton, A. L, Hamsher, K. deS., Varney, N. R., & Spreen, O. (1983). *Contributions to neuropsychological assessment: A clinical manual.* New York: Oxford University Press.

Bertier-Marcelo, L., Fernandez, A.-M., Celdran, E. M., & Kulisevsky, J. (1996). Perceptual and acoustic correlates of affective prosody repetition in transcortical aphasias. *Aphasiology, 10*, 711–712.

Bhatia, K. P., & Marsden, C. D. (1994). The behavioural and motor consequences of focal lesions of the basal ganglia in man. *Brain, 117*, 859–876.

Blonder, L. X., Bowers, D., & Heilman, K. M. (1991). The role of the right hemisphere in emotional communication. *Brain, 114*, 1115–1127 [published erratum in Brain (1992) 115 654].

Blonder, L. X., Gur, R., & Gur, R. (1989). The effects of right and left hemiparkinsonism on prosody. *Brain and Language, 36*, 193–207.

Blonder, L. X., Pickering, J. E., Heath, R. L., Smith, C. D., & Butler, S. M. (1995). Prosodic characteristics of speech pre- and post-right hemisphere srtoke. *Brain and Language, 51*(2), 318–335.

Blumstein, S., & Cooper, W. (1974). Hemispheric processing of intonation contours. *Cortex, 10*, 146–158.

Blumstein, S., & Goodglass, H. (1972). The perception of stress as a semantic cue in aphasia. *Journal of Speech and Hearing Research, 15*(4), 800–806.

Bolinger, D. (1986). *Intonation and its parts.* Stanford: Stanford University Press.

Borod, J. C. (1992). Interhemispheric and intrahemispheric control of emotion: a focus on unilateral brain damage. *Journal of Consulting and Clinical Psychology, 60*, 339–348.

Borod, J. C. (1993). Cerebral mechanisms underlying facial, prosodic, and lexical emotional expression: A review of neuropsychological studies and methodological issues. *Neuropsychology, 7*, 445–463.

Borod, J. C., Bloom, R. L., & Haywood, C. S. (1998). Verbal aspects of emotional communication. In C. Chiarello & M. Beeman (Eds.), *Right hemisphere language comprehension: Perspectives from cognitive neuroscience* (pp. 285–307). Mahwah, NJ: Lawrence Erlbaum Associates.

Borod, J. C., Rorie, K. D., Haywood, C. S., Andelman, F., Obler, L. K., Welkowitz, J., Bloom, R. L., & Tweedy, J. R. (1996). Hemispheric specialization for discourse reports of emotional experiences: Relationships to demographic, neurological, and perceptual variables. *Neuropsychologia, 34*, 351–359.

Boutsen, F. R., & Christman, S. (2002). Prosody in apraxia of speech. *Seminars in Speech and Language, 23*(4), 245–256.

Bowers, D., Bauer, R. M., & Heilman, K. M. (1993). The nonverbal affect lexicon: Theoretical perspectives from neurological studies of affect perception. *Neuropsychology, 7*, 433–444.

Bowers, D., Coslett, H. B., Bauer, R. M., Speedie, L. J., & Heilman, K. M. (1987). Comprehension of emotional prosody following unilateral hemisphere lesions: Processing defect versus distraction defect. *Neuropsychologia, 25*, 317–328.

Bradvik, B., Dravins, C., Holtas, S., Rosen, I., Ryding, E., & Ingvar, D. (1990). Do single right hemisphere infarcts or transient ischaemic attacks result in prosody. *Acta Neurologica Scandinavica, 81*, 61–70.

Breitenstein, C., Daum, I., & Ackermann, H. (1997). Affective prosody in patients with Parkinson's disease. In W. Ziegler & K. Deger (Eds.), *Clinical phonetics and linguistics* (pp. 382–386). London, UK: Whurr Publishers.

Breitenstein, C., Daum, I., & Ackermann, H. (1998). Emotional processing following cortical and subcortical brain damage: contribution of fronto-striatal circuitry. *Behavioural Neurology, 11*, 29–42.

Breitenstein, C., Van Lancker, D., & Daum, I. (2001). The contribution of speech rate and pitch variation to the perception of vocal emotions in a German and an American sample. *Cognition and Emotion, 15*, 57–79.

Breitenstein, C., Van Lancker, D., Daum, I., & Waters, C. (2001). Impaired perception of vocal emotions in Parkinson's disease: influence of speech time processing and executive functioning. *Brain and Cognition, 45*, 277–314.

Bricker, P. D., & Pruzansky, S. (1976). Speaker recognition. In N. J. Lass (Ed.), *Contemporary issues in experimental phonetics* (pp. 295–326). New York: Academic Press.

Brownell, H. H., Potter, H. H., Bihrle, A. M., & Gardner, H. (1986). Inference deficits in right brain-damaged patients. *Brain and Language, 27*, 310–312.

Bryden, M., & Ley, R. (1983). Right-hemisphere involvement in the perception and expression of emotion in normal humans. In K. Heilman & P. Satz (Eds.), *Neuropsychology of human emotion* (pp. 6–44). New York: The Guilford Press.

Bryden, M. P. (1982). *Laterality: functional asymmetry in the intact brain.* New York: Academic Press.

Buchanan, T. W., Lutz, K., Mirzazade, S., Specht, K., Shah, N. J., Zilles, K., & Jancke, L. (2000). Recognition of emotional prosody and verbal components of spoken language: an fMRI study. *Cognitive Brain Research, 9*(3), 227–238.

Cancelliere, A., & Kertesz, A. (1990). Lesion localization in acquired deficits of emotional expression and comprehension. *Brain and Cognition, 13*, 133–147.

Canter, G. J., & Van Lancker, D. (1985). Disturbances of the temporal organization of speech following bilateral thalamic surgery in a patient with Parkinson's disease. *Journal of Communication Disorders, 18*, 329–349.

Caplan, L. R., Schmahmann, J. D., Kase, C. S., Feldman, E., Baquis, G., Greenberg, J. P., Gorelick, P. B., Helgason, C., & Hier, D. B. (1990). Caudate infarcts. *Archives of Neurology, 47*, 133–143.

Carmon, A., & Nachshon, I. (1971). Effect of unilateral brain damage on perception of temporal order. *Cortex, 7*, 410–418.

Charbonneau, S., Scherzer, B. P., Aspirot, D., & Cohen, H. (2003). Perception and production of facial and prosodic emotions by chronic CVA patients. *Neuropsychologia, 41*(5), 605–613.

Chobor, K. L., & Brown, J. W. (1987). Phoneme and timbre monitoring in left and right cerebral vascular accident patients. *Brain and Language, 30*, 278–284.

Cicone, M., Wapner, W., & Gardner, H. (1980). Sensitivity to emotional expressions and situations in organic patients. *Cortex, 16*, 145–158.

Cimino, C., Verfaellie, M., Bowers, D., & Heilman, K. (1991). Autobiographical memory: influence of right hemisphere damage on emotionality and specificity. *Brain and Language, 15*, 106–118.

Cohen, H., Douaire, J., & Elsabbagh, M. (2001). The role of prosody in discourse processing. *Brain and Cognition, 46*(1-2), 73–82.

Cohen, H., Laframboise, M., Labelle, A., & Bouchard, S. (1993). Speech timing deficits in Parkinson's disease. *Journal of Clinical and Experimental Neuropsychology, 15*, 102–103.

Cohen, M. J., Riccio, C. A., & Flannery, A. M. (1994). Expressive aprosodia following stroke to the right basal ganglia: A case report. *Neuropsychology, 8*, 242–245.

Cole, M. (1971). Dysprosody due to posterior fossa lesions. *Transactions of the American Neurological Association, 96*, 151–154.

Cools, A. R., van den Bercken, J. H. L., Horstink, M. W. I., van Spaendonck, K. P. M., & Berger, H. J. C. (1984). Cognitive and motor shifting aptitude disorder in Parkinson's disease. *Journal of Neurology, Neurosurgery and Psychiatry, 47*, 443–453.

Critchley, E. M. R. (1981). Speech disorders of Parkinsonism: A review. *Journal of Neurology, Neurosurgery, and Psychiatry, 44*, 751–758.

Crystal, D. (1969). *Prosodic systems and intonation in English*. Cambridge, UK: Cambridge University Press.

Cummings, J. L. (1985). *Clinical neuropsychiatry*. Orlando, FL: Grune & Stratton.

Cummings, J. L. (1993). Frontal-subcortical circuits and human behavior. *Archives of Neurology, 50*, 873–880.

Cutler, A., Dahan, D., & van Donselaar, W. (1997). Prosody in the comprehension of spoken language: a literature review. *Language and Speech, 40*, 141–201.

Danly, M., & Shapiro, B. (1982). Speech prosody in Broca's aphasia. *Brain and Language, 16*, 171–190.

Danly, M., Cooper, W. E., & Shapiro, B. (1983). Fundamental frequency, language processing, and linguistic structure in Wernicke's aphasia. *Brain and Language, 19*, 1–24.

Darby, D. G. (1993). Sensory aprosodia: A clinical clue to lesions of the inferior division of the right middle cerebral artery? *Neurology, 43*, 567–572.

Darley, F. L., Aronson, A. E., & Brown, J. R. (1975). *Motor speech disorders*. Philadelphia: W.B. Saunders.

De Bleser, R., & Poeck, K. (1985). Analysis of prosody in the spontaneous speech of patients with CV-recurring utterances. *Cortex, 21*, 405–415.

Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (1987). *California verbal learning test, research education manual*. San Antonio: Psychological Corporation.

Denes, P. B., & Pinson, E. N. (1993). *The speech chain*. New York, NY: W.H. Freeman.

Douglas-Cowie, E., & Cowie, R. (1998). Intonational settings as markers of discourse units in telephone conversations. *Language and Speech, 41*(3-4), 351–374.

Duffy, J. R. (1995). *Motor speech disorders*. St. Louis: Mosby.

Dykstra, K., Gandour, J., & Stark, R. (1995). Disruption of prosody after fronta lobe seizures in the non-dominant hemisphere. *Aphasiology, 9*(5), 453–476.

Ellgring, H., & Scherer, K. R. (1996). Vocal indicators of mood change in depression. *Journal of Nonverbal Behavior, 20*, 83–110.

Fairbanks, G., & Hoaglin, L. W. (1941). An experimental study of the durational characteristics of the voice during the expression of emotion. *Speech Monographs, 8*, 85–90.

Fairbanks, G., & Pronovost, W. (1939). An experimental study of the pitch characteristics of the voice during the expression of emotion. *Speech Monographs, 6*, 87–104.

Fine, E. J., Ionita, C. C., & Lohr, L. (2002). The history of the development of the cerebellar examination. *Seminars in Neurology, 22*, 375–384.

Fisher, C. M. (1983). Abulia minor versus agitated behavior. *Clinical Neurosurgery, 31*, 9–31.

Fraile, V., Masson, H., & Cohen, H. (1994). Deficits in temporal aspects of Parkinsonian speech. *Behavioral Neurology, 7*, 12.

Freese, J., & Maynard, D. W. (1998). Prosodic features of bad news and good news in conversation. *Language in Society, 27*(2), 195–219.

Friederici, A. D., & Alter, K. (2004). Lateralization of auditory language functions: a dynamic dual pathway model. *Brain and Language, 89*, 267–276.

Fry, D. B. (1970). Prosodic phenomena. In B. Malmberg (Ed.), *Manual of phonetics* (pp. 365–410). Amsterdam: North Holland.

Gandour, J., & Baum, S. (2001). Production of stress retraction by left and right hemisphere damaged patients. *Brain and Language, 79*(3), 482–494.

Gandour, J., Larsen, J., Dechongkit, S., & Ponglorpisit, S. (1995). Speech prosody in affective contexts in Thai patients with right hemisphere lesions. *Brain and Language, 51*, 422–443.

Gandour, J., Petty, J., & Dardarananda, R. (1989). Dysprosody in Broca's aphasia: a case study. *Brain and Language, 37*, 232–257.

Gandour, J., Wong, D., Hsieh, L., Weinzapfel, B., Van Lancker, D., & Hutchens, G. (2000). A crosslinguistic PET study of tone perception. *Journal of Cognitive Neuroscience, 12:1*, 207–222.

George, M. S., Parekh, P. I., Rosinsky, N., Ketter, T. A., Kimbrell, T. A., Heilman, K. M., Herscovitch, P., & Post, R. M. (1996). Understanding emotional prosody activates right hemisphere regions. *Archives of Neurology, 53*, 665–670.

Goodglass, H., & Kaplan, E. (1983). *The assessment of aphasia and related disorders* (2nd ed.). Philadelphia: Lea & Febiger.

Gorelick, P. B., & Ross, E. D. (1987). The aprosodias: Further functional-anatomical evidence for the organization of affective language in the right hemisphere. *Journal of Neurology, Neurosurgery, and Psychiatry, 50*, 553–560.

Gräber, S., Hertrich, I., Daum, I., Spieker, S., & Ackermann, H. (2002). Speech perception deficits in Parkinson's disease: underestimation of time intervals compromises identification of durational phonetic contrasts. *Brain and Language, 82*(1), 65–74.

Heaton, R. K. (1981). *Wisconsin Card Sorting Test Manual*. Odessa, FL: Psychological Assessment Resources.

Hecker, M. (1971). Speaker recognition: An interpretive survey of the literature. *ASHA Monographs 16*.

Heilman, K., Scholes, R., & Watson, R. (1975). Auditory affective agnosia: Disturbed comprehension of affective speech. *Journal of Neurology, Neurosurgery and Psychiatry, 38*, 69–72.

Heilman, K. M. (1997). The neurobiology of emotional experience. *Journal of Neuropsychiatry and Clinical Neurosciences, 9*, 439–448.

Heilman, K. M., Bowers, D., Speedie, L., & Coslett, H. B. (1984). Comprehension of affective and nonaffective prosody. *Neurology, 34*, 917–921.

Helfrich, H. (1979). Age markers in speech. In K. R. Scherer & H. Giles (Eds.), *Social markers in speech* (pp. 63–108). Cambridge, UK: Cambridge University Press.

Hillis, A. E., Wityk, R. J., Barker, P. B., Beauchamp, N. J. U., Gailloud, P., Murphy, K., Cooper, O., & Metter, E. J. (2002). Subcortical aphasia and neglect in acute stroke: the role of cortical hypoperfusion. *Brain, 125*(5), 1094–1104.

Hird, K., & Kirsner, K. (1993). Dyprosody following acquired neurogenic impairment. *Brain and Language, 45*(1), 46–60.

Hird, K., & Kirsner, K. (2002). The relationship between prosody and breathing in spontaneous discourse. *Brain and Language, 80*(3), 536–555.

Hornak, J., Rolls, E. T., & Wade, D. (1996). Face and voice expression identification in patients with emotional and behavioral changes following ventral frontal lobe damage. *Neuropsychologia, 34*, 247–261.

Hutter, G. L. (1968). Relations between prosodic variables and emotions in normal American English utterances. *Journal of Speech and Hearing, 11*, 467–480.

Johnsrude, I. S., Penhune, V. B., & Zatorre, R. J. (2000). Functional specificity in the right human auditory cortex for perceiving pitch direction. *Brain, 123*(1), 155–163.

Kang, D.-W., Roh, J.-K., Lee, Y.-S., Song, I. C., Yoon, B.-W., & Chang, K.-H. (2000). Neuronal metabolic changes in the cortical region after subcortical infarction: a proton MR spectroscopy study. *Journal of Neurological and Neurosurgical Psychiatry, 69*, 222–227.

Kaplan, E. F., Goodglass, H., & Weintraub, S. (1983). *The Boston Naming Test* (2nd ed.). Philadelphia: Lea & Febiger.

Karow, C. M., Marquardt, T. P., & Marshall, R. C. (2001). Affective processing in left and right hemisphere brain-damaged subjects with and without subcortical involvement. *Aphasiology, 15*, 715–729.

Kempler, D., & Van Lancker, D. (1988). The Familiar and Novel Language Comprehension Test (FANL-C). Copyright.

Kempler, D., & Van Lancker, D. (2002). The effect of speech task on intelligibility in dysarthria: case study of Parkinson's disease. *Brain and Language, 80*, 449–464.

Kent, R. D. (1996). Hearing and believing: Some limits to the auditory-perceptual assessment of speech and voice disorders. *American Journal of Speech-Language Pathology, 5*, 7–24.

Kent, R. D., & Kim, Y. J. (2003). Toward an acoustic typology of motor speech disorders. *Clinical Linguistics and Phonetics., 17*, 427–445.

Kent, R. D., & Rosenbek, J. (1982). Prosodic disturbance and neurologic lesion. *Brain and Language, 15*, 259–291.

Kimura, D. (1967). Functional asymmetries of the brain in dichotic listening. *Cortex, 3*, 163–168.

Kiss, I., & Ennis, I. (2001). Age-related decine in percetion of prosodic affect. *Applied Neuropsychology, 8*(4), 251–254.

Kotz, S. A., Meyer, M., Alter, K., Besson, M., von Cramon, D. Y., & Friederici, A. D. (2003). On the lateralization of emotional prosody: An event-related functional MR investigation. *Brain and Language, 86*(3), 366–376.

Kreiman, J., Van Lancker-Sidtis, D., & Gerratt, B. (2005). Perception of voice quality. In D. Pisoni & R. Remez (Eds.), *Handbook of speech perception*. New York: Blackwells Publishing Chapter 18..

Ladd, D. R., Silverman, K. E. A., Tolkmitt, F., Bergman, G., & Scherer, K. R. (1985). Evidence for independent function of intonation contour type, voice quality, and F_0 range in signaling speaker affect. *Journal of the Acoustical Society of America, 78*, 435–444.

Ladd, D. R. (1996). *Intonational phonology*. UK Cambridge: Cambridge University Press.

Lakshminarayanan, K., Shalom, B., van Wassenhove, V., Orbelo, D., Houde, J., & Poeppel, D. (2003). The effect of spectral manipulations on the identification of affective and linguistic prosody. *Brain and Language, 84*, 250–263.

Lalande, S., Braun, C. M., Charlebois, N., & Whitaker, H. A. (1992). Effects of right and left hemisphere cerebrovascular lesions on discrimination of prosodic and semantic aspects of affect in sentences. *Brain and Language, 42*, 165–186.

Laplane, D., Baulac, M., Widlöcher, D., & DuBois, B. (1984). Pure psychic akinesia with bilateral lesions of basal ganglia. *Journal of Neurology, Neurosurgery, and Psychiatry, 47*, 377–385.

Laver, J. (1980). *The phonetic description of voice quality*. Cambridge: Cambridge University Press.

Laver, J. (2000). Phonetic evaluation of voice quality. In R. D. Kent & M. J. Ball (Eds.), *Voice quality measurement* (pp. 37–48). San Diego: Singular Publishing Group.

Le Dorze, G., Lever, N., Ryalls, J., & Brassard, C. (1995). Values of certain prosodic parameters obtain with French-speaking probands without communication problems. *Folia Phoniatrica et Logopedica, 47*(1), 39–47 French.

Le Dorze, G., Ryalls, J., Brassard, C., Boulanger, N., & Ratte, D. (1998). A comparison of the prosodic characteristics of the speech of people with Parkinson's disease and Friedreich's ataxia with neurologically normal speakers. *Folia Phoniatrica et Logopedica, 50*(12), 1–9.

Leonard, C. L., Baum, S. R., & Pell, M. D. (2001). The effect of compressed spech on the ability of right hemisphere-damaged patients to use context. *Cortex, 37*, 327–344.

Ley, R., & Bryden, M. (1982). A dissociation of right and left hemisphere effects for recognizing emotional tone and verbal content. *Brain and Cognition, 1*, 3–9.

Lezak, M. D. (1983). *Neuropsychological assessment* (2nd ed.). New York: Oxford University Press.

Lieberman, P., & Michaels, S. B. (1962). Some aspects of fundamental frequency and envelope amplitude as related to emotional content of speech. *Journal of the Acoustical Society of America, 34*, 922–927.

Lieberman, P. (2002). On the nature and evolution of the neural bases of human language. *American Journal of Physiological Anthropology Supplement, 35*, 36–62.

Liotti, M., Ramig, L. O., Vogel, D., New, P., Cook, C. I., Ingham, J. C., & Fox, P. T. (2003). Hypophonia in Parkinson's disease: neural correlates of voice treatment revealed by PET. *Neurology, 60*, 432–440.

Lloyd, A. J. (1999). Comprehension of prosody in Parkinson's disease. *Cortex, 35*(3), 389–402.

Lonie, J., & Lesser, R. (1983). Intonation as a cue to speech act identification in aphasic and other brain-damaged patients. *International Journal of Rehabilitation Research, 6*, 4.

Ludlow, C. L., & Loucks, T. (2003). Stuttering: a dynamic motor control disorder. *Journal of Fluency Disorders, 28*, 273–295.

Luria, A. R. (1966). *Higher cortical functions in man*. New York, NY: Basic Books.

Marin, R. (1990). Differential diagnosis and classification of apathy. *American Journal of Psychiatry, 147*, 22–30.

Marin, R., Biedrzycki, R., & Firinciogullari, S. (1991). Reliability and validity of the Apathy Evaluation Scale. *Psychiatry Research, 38*, 143–162.

Masterman, D. L., & Cummings, J. L. (1997). Frontal-subcortical circuits: the anatomic basis of executive, social, and motivated behaviors. *Journal of Psychopharmacology, 11*, 107–114.

Mayeux, R. (1983). Emotional changes associated with basal ganglia disorders. In K. Heilman & P. Satz (Eds.), *Neuropsychology of human emotion* (pp. 141–164). New York, NY: The Guilford Press.

McNeely, H. E., & Parlow, S. E. (2001). Complementarity of linguistic and prosodic processes in the intact brain. *Brain and Language, 79*(3), 473–481.

McRoberts, G. W., Studdert-Kennedy, M., & Shankweiler, D. (1995). The role of fundamental frequency in signaling linguistic stress and affect: Evidence for a dissociation. *Perception and Psychophysics, 57*, 159–174.

Meadows, M., & Kaplan, R. (1994). Dissociation of autonomic and subjective responses to emotional slides in right hemisphere damaged patients. *Neuropsychologia, 32*(7), 847–856.

Melara, R. D., & Marks, L. E. (1990). Interaction among auditory dimensions: Timbre, pitch, and loudness. *Perception and Psychophysics, 48*, 169–178.

Mendez, M. F., Adams, N. L., & Lewandowski, K. S. (1989). Neurobehavioral changes associated with caudate lesions. *Neurology, 39*, 349–354.

Mills, C. K. (1912). The cerebral mechanism of emotional expression. *Transactions of the College of Physicians of Philadelphia, 34*, 381–390.

Moen, I. (1991). Functional lateralization of pitch accents and intonation in Norwegian: Monrad-Krohn's study of an aphasic patient with altered "melody of speech". *Brain and Language, 41*, 538–554.

Monrad-Krohn, G. H. (1947). Dysprosody or altered "melody of speech". *Brain, 70*, 405–415.

Monrad-Krohn, G. H. (1963). The third element of speech prosody and its disorders. In L. Halpern (Ed.), *Problems of dynamic neurology* (pp. 101–118). Jerusalem, Israel: Hebrew University.

Munson, B. R. (1994). An acoustic study of intonation in one aphasic patient. *Journal of the Acoustical Society of America, 95*(5), 3012–3013.

Myers, P. (1999). *Right hemisphere damage*. San Diego: Singular Publishing Co.

Niemi, J. (1998). Modularity of prosody: autonomy of phonological quantity and intonation in aphasia. *Brain and Language, 61*(1), 45–53.

Oepen, G., & Berthold, H. (1983). Rhythm as an essential part of music and speech abilities. *Revue Romane de Neurologie et Psychiatrie., 21*, 3.

Olsen, T. S., Bruhn, P., & Oberg, R. G. (1986). Cortical hypoperfusion as a possible cause of 'subcortical aphasia'. *Brain, 109*(3), 393–410.

Osmon, D. C., Panos, J., Kautz, P., & Gandhavadi, B. (1998). Crossed aphasia in a dextral: a test of the Alexander-Annett theory of anomalous organization of brain function. *Brain and Language, 63*(3), 426–438.

Osterrieth, P. A. (1944). Le test de copie d'une figure complex: Contribution a l'etude de la perception et de la memoire. *Archives de Psychologie, 30*, 286–356.

Ouellette, G. P., & Baum, S. R. (1994). Acoustic analysis of prosodic cues in left- and right-hemisphere-damaged patients. *Aphasiology, 8*, 257–283.

Patel, A. D., Paretz, I., Tramo, M., & Labreque, R. (1998). Processing prosodic and musical patterns: a neuropsychological investigation. *Brain and Language, 61*(1), 123–144.

Patel, R. (2002). Prosodic control in severe dysarthria: preserved ability to mark the question-statement contrast. *Journal of Speech, Language, and Hearing Research, 45*(5), 858–870.

Paul, L. K., Van Lancker, D., Schieffer, B., Dietrich, R., & Brown, W. S. (2003). Communicative deficits in agenesis of the corpus callosum: nonliteral language and affective prosody. *Brain and Language, 85*, 313–324.

Pell, M. D., & Leonard, C. L. (2003). Processing emotional tone from speech in Parkinson's disease: a role for the basal ganglia. *Cognitive, Affective & Behavioral Neuroscience, 3*, 275–288.

Pell, M. D. (1999a). The temporal organization of affective and non-affective in patients with right-hemisphere infarcts. *Cortex, 35*(4), 455–477.

Pell, M. D. (1999b). Fundamental frequency encoding of linguistic and emotional prosody by right hemisphere-damaged speakers. *Brain and Language, 69*, 161–192.

Pell, M. D., & Baum, S. R. (1997a). The ability to perceive and comprehend intonation in linguistic and affective contexts by brain-damaged adults. *Brain and Language, 57*, 80–99.

Pell, M. D., & Baum, S. R. (1997b). Unilateral brain damage, prosodic comprehension deficits, and the acoustic cues to prosody. *Brain and Language, 57*, 195–214.

Pell, M. D. (1996). On the receptive prosodic loss in Parkinson's disease. *Cortex, 32*, 693–704.

Pell, M. D. (1998). Recognition of prosody following unilateral brain lesion: Influene of functional and structural attribute of prosodic contours. *Neuropsychologia, 36*(8), 701–715.

Pell, M. D. (2001). Influence of emotion and focus location on prosody in matched statements and questions. *Journal of the Acoustical Society of America, 109*(4), 1668–1680.

Peppe, S., Maxim, J., & Wells, B. (2000). Prosodic variation in southern British English. *Language and Speech, 43*(Pt. 3), 309–334.

Perry, R. J., Rosen, H. R., Kramer, J. H., Beer, J. S., Levenson, R. L., & Miller, B. L. (2001). Hemispheric dominance for emotions, empathy and social behavior: evidence from right and left handers with frontotemporal dementia. *Neurocase, 7*(2), 145–160.

Pike, K. (1945). *The intonation of American English*. Ann Arbor: University of Michigan Pres.

Pillon, B., Dubois, B., Lhermitte, F., & Agid, Y. (1986). Heterogeneity of cognitive impairment in progressive supranuclear palsy, Parkinsons disease and Alzheimers disease. *Neurology, 36*, 1179–1185.

Pimenthal, P. A., & Kingsbury, N. A. (1989). *Mini inventory of right brain injury (MIRBI)*. Austin, TX: PRO-ED.

Poncet, M., & Habib, M. (1994). Atteinte isolée des comportements motivés et lésions des noyaux gris centraux. *Revue Neurologique, 150*, 588–593.

Protopapas, A., & Lieberman, P. (1997). Fundamental frequency of phonation and perceived emotional stress. *Journal of the Acoustic Society of America, 101*(4), 2267–2277.

Rapcsak, S. Z., Comer, J. F., & Rubens, A. B. (1993). Anomia for facial expressions: Neuropsychological mechanisms and anatomical correlates. *Brain and Language, 45*, 233–252.

Reitan, R. M. (1958). Validity of the Trailmaking Test as an indication of organic brain damage. *Perceptual Motor Skills, 8*, 271–276.

Remez, R. E, Fellowes, J. M., & Rubin, P. E. (1997). Talker identification based on phonetic information. *Journal of Experimental Psychology: Human Perception and Performance, 23*, 651–666.

Rey, A. (1941). L'examen psychologique dans les cas d'encéphalopathie traumatique. *Archives de Psychologie, 28*, 286–340.

Rey, A. (1964). *L'examen clinique en psychologie*. Paris: Press Universitaire de France.

Robin, D. A., Tranel, D., & Damasio, H. (1990). Auditory perception of temporal and spectral events in patients with focal left and right cerebral lesions. *Brain and Language, 39*, 539–555.

Robinsin, R. G., Kubos, K., Starr, L., Rao, K., & Price, T. (1984). Mood disorders in stroke patients: Importance of location of lesion. *Brain, 197*, 81–93.

Robinson, B. W. (1976). Limbic influences on human speech. *Annals of the New York Academy of Sciences, 280*, 761–771.

Robinson, G. M., & Solomon, D. J. (1974). Rhythm is processed by the speech hemisphere. *Journal of Experimental Psychology, 102*, 508–511.

Rosenbek, J. C., Crucian, G. P., Leon, S. A., Hieber, B., Rodriguez, A. D., Holiway, B., Ketterson, T. U., Heilman, K., & Gonzales-Rothi, L. (2004). Novel treatments for expressive aprosodia: a phase investigation of cognitive linguistic and imitative intervention. *Journal of the International Neuropsychological Society, 10*, 786–793.

Ross, E. D. (1981). The aprosodias: functional-anatomic organization of the affective components of language in the right hemisphere. *Archives of Neurology, 38*, 561–569.

Ross, E. D., Thompson, R. D., & Yenkosky, J. (1997). Lateralization of affective prosody in brain and the callosal integration of hemispheric language functions. *Brain and Language, 56*, 27–54.

Ryalls, J. (1986). An acoustic study of vowel production in aphasia. *Brain and Language, 29*, 46–67.

Ryalls, J. (1988). Concerning right-hemisphere dominance for affective language. *Archives of Neurology, 45*, 337–338.

Saint-Cyr, J. A., Taylor, A. E., & Nicholson, K. (1995). Behavior and the basal ganglia. In W. J. Weiner & A. E. Lang (Eds.), *Behavioral neurology of movement disorders. Advances in neurology* (Vol. 65, pp. 1–28). New York, NY: Raven Press, Ltd.

Santamaria, J., & Tolosa, E. (1992). Clinical subtypes of Parkinson's disease and depression. In S. J. Huber & J. L. Cummings (Eds.), *Parkinson's disease: Neurobehavioral aspects* (pp. 217–228). New York NY: Oxford University Press.

Santens, P., De Letter, M., Van Borsel, J., De Reuck, J., & Caemaert, J. (2003). Laterlized effects of subthalamic nucleus stimulation on different aspects of speech in Parkinson's disease. *Brain and Language, 87*, 253–258.

Sapir, E. (1926). Speech as a personality trait. *American Journal of Sociology, 32*, 892–905.

Schafer, A. J., Speer, S. R., Warren, P., & White, S. D. (2000). Intonational disambiguation in sentence production and comprehension. *Journal of Psycholinguistic Research, 29*, 169–182.

Schegloff, E. A. (1998). Reflections on studying prosody in talk-in-interaction. *Language and Speech, 41*, 235–263.

Scherer, K. R. (1979). Personality markers in speech. In H. Giles (Ed.), *Social markers in speech* (pp. 147–201). Cambridge: Cambridge University Press.

Scherer, K. R. (1986). Vocal affect expression: a review and a model for future research. *Psychological Bulletin, 99*, 145–165.

Schirmer, A., Alter, K., Kotz, S. A., & Friederici, A. D. (2001). Lateralization of prosody during language production: a lesion study. *Brain and Language, 76*, 1–17.

Schlanger, B. B., Schlanger, P., & Gerstman, L. J. (1976). The perception of emotionally toned sentences by right hemisphere-damaged and aphasic subjects. *Brain and Language, 3*, 396–403.

Schmitt, J. J., Hartje, W., & Willmes, K. (1997). Hemispheric asymmetry in the recognition of emotional attitude conveyed by facial expression, prosody and propositional speech. *Cortex, 33*(1), 65–81.

Scott, S., Caird, F., & Williams, B. (1984). Evidence for an apparent sensory speech disorder in Parkinson's disease. *Journal of Neurology, Neurosurgery, and Psychiatry, 47*, 840–843.

Shapiro, B. E., & Danly, M. (1985). The role of the right hemisphere in the control of speech prosody in propositional and affective contexts. *Brain and Language, 25*, 19–36.

Shattuck-Hufnagel, S., & Turk, A. E. (1996). A prosody tutorial for investigators of auditory sentence processing. *Journal of Psycholinguistic Research, 25*(2), 193–247.

Sidtis, J. J. (1980). On the nature of the cortical function underlying right hemisphere auditory perception. *Neuropsychologia, 18*, 321–330.

Sidtis, J. J. (1984). Music, pitch perception, and the mechanisms of cortical hearing. In M. S. Gazzaniga (Ed.), *Handbook of cognitive neuroscience* (pp. 91–114). New York: Plenum Press.

Sidtis, J. J., & Feldmann, E. (1990). Transient ischemic attacks presenting with a loss of pitch perception. *Cortex, 26*, 469–471.

Sidtis, J. J., & Van Lancker Sidtis, D. (2003). A neurobehavioral approach to dysprosody. *Seminars in Speech and Language, 24*(2), 93–105.

Sidtis, J. J., & Volpe, T. B. (1988). Selective loss of complex-pitch or speech discrimination after unilateral cerebral lesion. *Brain and Language, 34*, 235–245.

Sidtis, J. J., Strother, S. C., & Rottenberg, D. A. (2003). Predicting performance from functional imaging data: Methods matter. *NeuroImage, 20*, 615–624.

Speedie, L. J., Brake, N., Folstein, S., Bowers, D., & Heilman, K. (1990). Comprehension of prosody in Huntington's disease. *Journal of Neurology, Neurosurgery, and Psychiatry, 53*, 607–610.

Starkstein, S. E., Federoff, J., Price, T., Leigarda, R., & Robinson, R. (1994). Neuropsychological and neuroradiologic correlates of emotional prosody comprehension. *Neurology, 44*, 515–522.

Starkstein, S. E., Robinson, R. G., Berthier, M. L., Parikh, R. M., & Price, T. R. (1988). Differential mood changes following basal ganglia vs thalamic lesions. *Archives of Neurology, 45*, 725–730.

Stringer, A. Y. (1996). Treatment of motor aprosodia with pitch biofeedback and expression modeling. *Brain Injury, 10*(8), 583–590.

Taylor, A. E., Saint-Cyr, J. A., & Lang, A. E. (1990). Subcognitive processing in the frontocaudate "complex loop": The role of the striatum. *Alzheimer's Disease and Associated Disorders, 4*, 150–160.

Tramo, M. J., & Bharucha, J. J. (1991). Musical priming by the right hemisphere post-callosotomy. *Neuropsychologia, 29*, 313–325.

Tramo, M. J., Shah, G. D., & Braida, L. D. (2002). Functional role of auditory cortex in frequency processing and pitch perception. *Journal of Neurophysiology, 87*, 122–139.

Tranel, D. (1992). Neuropsychological correlates of cortical and subcortical damage. In S. C. Yudofsky & R. E. Hales (Eds.), *Textbook of neuropsychiatry volume II* (pp. 57–88). Washington, DC: American Psychiatric Press, Inc.

Tree, J. E., & Meijer, P. J. (2000). Untrained speakers' use of prosody in syntactic disambiguation and listeners' interpretations. *Psychological Research, 63*(1), 1–13.

Tucker, D. M., Watson, R. T., & Heilman, K. M. (1977). Discrimination and evocation of affectively intoned speech in patients with right parietal disease. *Neurology, 27*, 947–950.

van Dommelen, W. A. (1990). Acoustic parameters in human speaker recognition. *Language and Speech, 33*(3), 259–272.

Van Lancker, D. (1980). Cerebral lateralization of pitch cues in the linguistic signal. *Papers in Linguistics: International Journal of Human Communication, 13*, 201–277.

Van Lancker, D. (1984). Affective Prosody Test. Copyright.

Van Lancker, D. (1991). Personal relevance and the human right hemisphere. *Brain and Cognition, 17*, 64–92.

Van Lancker, D. (2000). Brain structures in verbal communication: a focus on prosody. *Contemporary Issues in Stroke Rehabilitation, 7*, 1–23.

Van Lancker, D., & Breitenstein, C. (2000). Emotional dysprosody and similar dysfunctions. In J. Bougousslavsky & J. L. Cummings (Eds.), *Disorders of behavior and mood in focal brain lesions* (pp. 326–368). Cambridge, UK: Cambridge University Press.

Van Lancker, D., & Cummings, J. L. (1999). Expletives: Neurolinguistic and neurobehavioral inquiries into swearing. *Brain Research Reviews, 31*, 81–104.

Van Lancker, D., & Fromkin, V. (1973). Hemispheric specialization for pitch and 'tone': Evidence from Thai. *Journal of Phonetics, 1*, 101–109.

Van Lancker, D., & Fromkin, V. A. (1978). Cerebral dominance for pitch contrasts in tone language speakers and in musically untrained and trained English speakers. *Journal of Phonetics, 6*, 19–23.

Van Lancker, D., & Kreiman, J. (1986). Preservation of familiar speaker recognition but not unfamiliar speaker discrimination in aphasic patients. *Clinical Aphasiology, 16*, 234–240.

Van Lancker, D., & Kreiman, J. (1987). Unfamiliar voice discrimination and familiar voice recognition are independent and unordered abilities. *Neuropsychologia, 25*, 829–834.

Van Lancker, D., & Nicklay, C. (1992). Comprehension of personally relevant (PERL) versus novel language in two globally aphasic patients. *Aphasiology, 6*, 37–61.

Van Lancker, D., & Sidtis, J. J. (1992). The identification of affective-prosodic stimuli by left- and right-hemisphere-damaged subjects: all errors are not created equal. *Journal of Speech and Hearing Research, 35*, 963–970.

Van Lancker, D., Cummings, J., Kreiman, J., & Dobkin, B. H. (1988). Phonagnosia: A dissociation between familiar and unfamiliar voices. *Cortex, 24*, 195–209.

Van Lancker, D., Kempler, D., Hanson, W., Jackson, C., Lanto, A., & Metter, E. J. (1988). Prosodic changes in speech following brain damage. Acoustic and neuroradiographic measures. *Journal of the Acoustical Society of America*, abstract BB.

Van Putten, S. M., & Walker, J. P. (2003). The production of emotional prosody in varying degrees of severity in apraxia of speech. *Journal of Communication Disorders, 36*, 77–95.

Vance, J. E. (1994). Prosodic deviation in dysarthria: a case study. *European Journal of Disorders of Communication, 29*(1), 61–76.

Voiers, W. D. (1964). Perceptual bases of speaker identity. *Journal of the Acoustical Society of America, 36*, 1065–1073.

Walker, J. P., Fongemie, K., & Daigle, T. (2001). Prosodic facilitation in the resolution of syntactic ambiguities in subjects with left and right hemisphere damage. *Brain and Language, 78*(2).

Walker, J. P., Pelletier, R., & Reif, L. (2004). The production of prosodic structures in subjects with right hemisphere damage. *Clinical Linguistics and Phonetics, 18*, 85–106.

Wechsler, A. (1973). The effect of organic brain disease on recall of emotionally charged vs. neutral narrative texts. *Neurology, 23*, 130–135.

Wechsler, D. (1981). *Manual for the WAIS-R*. New York: Psychological Corporation.

Wechsler, D. (1987). *Wechsler Memory Scale-Revised*. New York: Psychological Corporation.

Weddell, R. A. (1994). Effects of subcortical lesion site on human emotional behavior. *Brain and Cognition, 25*, 161–193.

Weiller, C., Willmes, K., Reiche, W., Thron, A., Isensee, C., Buell, U., & Ringelstein, E. G. (1993). The case of aphasia or neglect after striatocapsular infarction. *Brain, 116*(6), 1509–1525.

Weintraub, S., Mesulam, M.-M., & Kramer, L. (1981). Disturbances in prosody: A right-hemisphere contribution to language. *Archives of Neurology, 38*, 742–744.

Wells, B., & Macfarlane, S. (1998). Prosody as an interactional resource: turn-projection and overlap. *Language and Speech, 41*, 265–294.

Weniger, D. (1984). Dysprosody as part of the aphasic language disorder. *Advances in Neurology, 42*, 41–50.

Wertz, R. T., Henschel, C. R., Auther, L. L., Ashford, J. R., & Kirshner, H. S. (1998). Affective prosodic disturbances subsequent to right hemisphere-stroke: a clinical application. *Journal of Neurolinguistics, 11*(1-2), 89–102.

Williams, C. E., & Stevens, K. N. (1972). Emotion and speech: Some acoustical correlates. *Journal of the Acoustical Society of America, 52*, 1238–1250.

Wong, P. C. M. (2002). Hemispheric specialization of linguistic pitch patterns. *Brain Research Bulletin, 59*(2), 83–95.

Wymer, J. H., Lindman, L. S., & Booksh, R. L. (2002). A neuropsychological perspective of aprosody: features, function, assessment, and treatment. *Applied Neuropsychology, 9*(1), 37–47.

Yorkston, K. M., & Beukelman, D. R. (1981). *Assessment of intelligibility of dysarthric speech*. Tigard, Oregon: C.C. Publications.

Zatorre, R. J. (1988). Pitch perception of complex tones and human temporal lobe function. *Journal of the Acoustical Society of America, 84*, 566–572.

Zatorre, R. J., & Belin, P. (2001). Spectral and temporal processing in human auditory cortex. *Cerebral Cortex, 11*, 946–953.

Zatorre, R. J., Evans, A., Meyer, E., & Gjedde, A. (1992). Lateralization of phonetic and pitch discrimination in speech processing. *Science, 256*, 846–849.