

Aphasiology



ISSN: 0268-7038 (Print) 1464-5041 (Online) Journal homepage: http://www.tandfonline.com/loi/paph20

The neural bases of prosody: Insights from lesion studies and neuroimaging

SHARI R. BAUM & MARC D. PELL

To cite this article: SHARI R. BAUM & MARC D. PELL (1999) The neural bases of prosody: Insights from lesion studies and neuroimaging, Aphasiology, 13:8, 581-608, DOI: 10.1080/026870399401957

To link to this article: <u>http://dx.doi.org/10.1080/026870399401957</u>

1	1	1	1

Published online: 31 Aug 2010.



🕼 Submit your article to this journal 🗹

Article views: 423



View related articles 🗹



Citing articles: 111 View citing articles 🗷

Full Terms & Conditions of access and use can be found at http://www.tandfonline.com/action/journalInformation?journalCode=paph20

Review

The neural bases of prosody: Insights from lesion studies and neuroimaging

SHARI R. BAUM* and MARC D. PELL

School of Communication Sciences and Disorders, McGill University, 1266 Pine Avenue W., Montréal, Québec H3G 1A8, Canada

(Received 2 September 1998; accepted 22 November 1998)

Abstract

This paper reviews the major findings and hypotheses to emerge in the literature concerned with speech prosody. Both production and perception of prosody are considered. Evidence from studies of patients with lateralized left or right hemisphere damage are presented, as well as relevant data from anatomical and functional imaging studies.

Introduction

The relation between brain and behaviour has fascinated researchers for many years. By investigating this relationship, we can independently inform models of neural organization as well as models of cognitive processing. For example, if we can determine that a particular cognitive operation is associated with two distinct brain regions, it may suggest a physiological connection between the two areas, permitting insights from processing to structure. Equally important, knowledge of neuroanatomy and neurophysiology can constrain theories of cognitive processing as well; models proposing operations that are incompatible with biological premises would naturally be ruled out (see Geschwind 1984).

In the search for the neuroanatomical correlates of behaviour, a great deal of attention has been focused on language processing. At the broadest level are asymmetries in the lateralization of cognitive functions. Every student of neuropsychology and neurolinguistics is familiar with the long-established association of the language processing centers with the left cerebral hemisphere and the emotion processing centers with the right hemisphere of the brain (e.g. Hughlings Jackson, 1915). But what of prosody, with its functions crossing these boundaries?

As is well known, prosody serves a variety of functions in language processing, from the conveyance of the speaker's emotions to the phonemic use of tone to differentiate lexical items in certain languages. Regardless of function, the same three acoustic parameters serve as primary prosodic attributes: fundamental

^{*} Author for correspondence; e-mail: c3cr@musica.mcgill.ca

frequency (F_0), duration and amplitude (Lehiste 1970). In most recent models of speech production (e.g. Garrett 1980, Levelt 1989), the prosody generator (Levelt 1989) is considered a distinct component of the speech production system or a subcomponent of the phonological system. Current phonological theories also posit a separate prosodic tier that specifies metrical structure (Levelt 1989, Liberman and Prince 1977, Selkirk 1984). To date, the majority of neurolinguistic research in this area has focused in some detail on the neural bases of the segmental aspects of speech (see for e.g. Blumstein 1991 for a review); far less attention has been devoted to speech prosody. Thus, despite its importance in communication, the neural systems responsible for the production and comprehension of prosody remain largely unspecified.

Among the types of evidence that have been brought to bear on this issue are data from lateralized stimulus presentation in normal subjects (e.g. Blumstein and Cooper 1974, Zurif and Mendelsohn 1972), results of studies of normal brain activity during specific tasks (e.g. Zatorre *et al.* 1992), and the patterns of performance deficits in unilaterally brain-damaged patients (e.g. Ross 1981, Danly and Shapiro 1982). For example, based largely on lesion studies, models of linguistic organization in the left hemisphere have long held that the comprehension of word meaning involves the posterior portions of the superior temporal gyrus (Wernicke's area) and that articulatory programming and implementation invoke activity in the inferior frontal lobe, as well as in pre-Rolandic motor cortex regions (at least; e.g. Kertesz 1994 for review). In addition, animal and human models have indicated that emotion is broadly associated with the limbic system, implicating subcortical structures (Borod 1993, Cancelliere and Kertesz 1990, Fuster 1989, Mesulam 1985, Rinn 1984).

Recently, much more detailed information has been gathered through the advent of functional neuroimaging techniques such as positron emission tomography (PET), regional cerebral blood flow (rCBF), functional magnetic resonance imaging (fMRI) and evoked response potentials (ERP). As an example, numerous PET studies have confirmed left superior temporal activation during word reading and recognition tasks (Chertkow and Bub 1994, Petersen *et al.* 1990). More directly relevant to prosodic processing, Zatorre *et al.* (1992) have found increased activity in Broca's area during tasks requiring phonetic judgments concerning CVC syllables; in contrast, tasks requiring pitch judgments of the same CVC syllables elicited right prefrontal activation, suggesting that identification of fundamental frequency is associated with right hemisphere mechanisms. These findings may support a dissociation in the lateralized processing of specific acoustic parameters.¹

Further evidence of right hemisphere control of fundamental frequency processing comes from a study of ERPs in right hemisphere-damaged and left hemisphere-damaged patients (Twist *et al.* 1991). Right hemisphere-damaged subjects were shown to exhibit abnormal ERP patterns in non-speech frequency discrimination tasks as well as in an affective prosody discrimination task. Left hemisphere-damaged subjects only displayed abnormalities in a semantic discrimination task. Twist and colleagues (1991) interpreted the ERP results as supporting the right hemisphere's role in prosodic processing, despite the absence of differences between the two brain-damaged groups on standard behavioural measures of prosodic abilities.

A recent PET study investigating the comprehension of emotion appears to confirm preferential activation of the *right* prefrontal cortex in tasks requiring

judgment of the affective prosodic content of a sentence; in contrast, bilateral prefrontal activation was found for tasks requiring judgment of the emotional propositional content of the same utterances (George *et al.* 1996). Interestingly, a PET investigation comparing auditory discrimination capabilities in native speakers of a tone language (Thai) to native speakers of English revealed significant activation in Broca's area (left hemisphere) only for Thai subjects when the pitch judgments were rendered in a linguistic context (Gandour *et al.* 1997). The investigators highlighted the functional role of the prosodic cues in determining lateralization of processing. Although much of these data are preliminary in nature, in combination with findings from psycholinguistic investigations of brain-damaged individuals, data from these emergent technologies promise to yield a clearer picture of the neural bases of prosody.

The findings to date have yielded several major hypotheses concerning the neuroanatomical regions active in prosodic processing. The most straightforward of the hypotheses contends that all aspects of prosody are processed in the right hemisphere and integrated with linguistic information via callosal connections (Klouda *et al.* 1988). A second hypothesis claims that affective or emotional prosody is controlled in the right hemisphere, whereas the left hemisphere is specialized for linguistic prosody—the functional lateralization hypothesis (Van Lancker 1980). Under this view, there is a continuum of linguistic 'load' along which processing shifts from left hemisphere to right hemisphere control as the tasks become less linguistically-based. A third major alternative posits that the comprehension and production of prosody are subserved largely by subcortical regions and are not lateralized to one or another hemisphere (e.g. Cancelliere and Kertesz 1990). Finally, several recent investigations have supported the theory that individual acoustic cues to prosody may be independently lateralized (e.g. Van Lancker and Sidtis 1992).

In the review that follows, we will consider the evidence that bears on these hypotheses by first discussing the production of prosody in both linguistic and affective domains and across levels of structure. We will then turn to the perception or comprehension of prosody considered from a similar perspective. Finally, we will attempt to summarize the findings to determine which, if any, of the current hypotheses is supported by the majority of the data. Despite a number of caveats that should be kept in mind in interpreting data from brain-damaged subjects (e.g. Geschwind 1984), by gathering converging evidence from a variety of sources, we may meet the challenge of devising a model of prosodic representation in the brain.

Production of prosody

One of the main catalysts for the development of theories of prosodic lateralization was the clinical observation that, subsequent to right hemisphere damage, many patients exhibit a generalized flattened affect with a concomitant monotonous speech production pattern. In striking contrast, subsequent to left hemisphere damage, despite myriad possible articulatory impairments, patients appear to produce relatively normal intonation and to retain the ability to produce appropriate melody and rhythm for singing (Hughlings Jackson 1915). As will be seen, although numerous investigators continue to rely on clinical judgments, many researchers have adopted more objective and reliable instrumental acoustic analyses to explore the prosodic patterns of speech produced by brain-damaged patients.

Affective prosody

Our review of the relevant literature will begin with the production of affective prosody and then turn to different types of linguistic prosody as well. One of the earliest systematic investigations of the production of affective intonation was that of Tucker *et al.* (1977). In their study, the authors examined the ability of 8 right hemisphere-damaged patients and 8 normal controls to repeat neutral sentences with specific emotional tones. The subjects' productions were then judged by a panel of three listeners who attempted to identify the emotion conveyed. Results revealed that the productions of the right hemisphere-damaged speakers were judged to show the intended emotion less often than those of the normal control subjects, supporting the right hemisphere's involvement in the production of affective prosody. It is important to point out, however, that there was a great deal of inter-subject and inter-judge variability and that no left hemisphere-damaged control group was included in the experiment, leaving open the possibility that brain damage, regardless of site, would have yielded comparable deficits.

As major proponents of the hypothesis that affective prosody is controlled in the right hemisphere, Ross and colleagues (Edmondson *et al.* 1987, Ross *et al.* 1986, Ross *et al.* 1988) have examined the production of affective language in a series of cross-linguistic investigations. One piece of evidence to support their hypothesis derives from a study of English-speaking epileptic subjects undergoing a right-sided Wada test (Ross *et al.* 1988). In this test, a sodium amytal solution is injected into the carotid artery to temporarily 'deactivate' one hemisphere of the brain. Perception and production tests may be conducted during the deactivation period in order to determine that hemisphere's role in a particular task. Five epileptic subjects were required to repeat a sentence following a model intoned in each of six emotional tones. Acoustic parameters were measured from productions recorded prior to and during the Wada tests. Results revealed that mean F_0 and F_0 standard deviation were reduced during the test as compared to before the injection, suggesting a flattened affect as a result of deactivation of the right hemisphere (Ross *et al.* 1988).

Another source of evidence that Ross and colleagues draw upon to support the right hemisphere's role in the control of affective prosody comes from investigations of brain-damaged patients. Based on data from English-speaking brain-damaged patients, Ross (1981, 1993, Ross and Mesulam 1979) not only contended that the right hemisphere was dominant for affective prosody, he further proposed a classificatory system for affective prosodic deficits (termed 'aprosodiæs') similar to that utilized for left hemisphere-damaged aphasic patients (e.g. Goodglass and Kaplan 1983). Specific constellations of symptoms were hypothesized to be associated with particular lesion sites. For example, impairments of affective comprehension were supposedly due to temporal lobe lesions, parallel to auditory comprehension deficits from left temporal lobe lesions, whereas disorders of spontaneous affective production were thought to arise subsequent to inferior frontal lobe damage, similar to the speech production deficits associated with left Broca's area lesions (Ross 1981, 1993). The majority of early data gathered in support of this classificatory system relied on bedside clinical judgments. For

instance, Gorelick and Ross (1987) examined 14 right hemisphere-damaged patients at approximately two weeks post-onset for spontaneous prosody and gestures, repetition of affective prosody and comprehension of prosody and emotional gestures. Judgments of accuracy were made by the neurologist at bedside, who also presented the stimuli. Patients were classified into clinical categories based on their performance and lesion sites for most patients were found to correlate with those expected for each aprosodia syndrome, paralleling presumed left hemisphere organization for language. Of course, findings from such a subjective bedside examination may not prove to be reliable upon replication.

Ross' (1981) classification system yielded a great deal of controversy, with numerous reports of frequent exceptions to the organization scheme (Brådvik et al. 1990, 1991, Cancelliere and Kertesz 1990, Darby 1993). Despite the questions raised, Ross and colleagues continued to pursue the localization of prosodic processing within the right hemisphere. They reasoned that, although the majority of research on speech prosody has been conducted in English, it was of particular interest to examine so-called tone languages in which prosody serves a phonemic function. In languages such as Thai, Chinese and Norwegian, a pair of lexical items may be distinguished solely on the basis of tonal contrasts, with, e.g. a rising tone on a given syllable yielding one meaning and a falling tone produced with that same string of consonants and vowels yielding a different meaning. In such languages, prosodic features serve a more basic linguistic function in addition to their use as cues to syntactic structure and emotional meaning. Because of the fundamental linguistic importance of tone in these language systems, one might predict a different pattern of prosodic control than is found in speakers of non-tone languages.

To examine this issue, as a first step, Ross et al. (1986) asked five native speakers of Mandarin, Taiwanese, Thai and English to produce translations of the sentence 'you are going to the movies' with five different emotional tones. The productions were digitized and acoustic analyses of F₀, duration and amplitude were conducted. From these measures, the authors derived a summary statistic which they termed 'emotional range' to characterize each language group. They reported significantly higher values of the emotional range statistic for native English speakers relative to the other groups, concluding that 'the presence of tone in a language adversely impacts the free use of F₀ for affective-prosodic signalling' (Ross et al. 1986, p. 298). To address whether the neural substrate of prosodic processing is similar in tone language speakers, Edmondson et al. (1987) performed a follow-up experiment in which 8 right hemisphere-damaged Taiwanese speakers (10-47 days post-onset) and 8 normal controls were asked to repeat a small number of affectively intoned sentences. Both acoustic and perceptual measures were undertaken. Results indicated that control of F₀ was impaired in the right hemisphere-damaged patients relative to the normal controls. In addition, normal listeners were less able to accurately judge the intended emotions from the right hemisphere-damaged speakers' productions compared to those of the normal controls; listeners rated the right hemisphere-damaged subjects' productions as relatively poor repetitions of the model stimuli. Edmondson et al. (1987) concluded that there is a loss of control of affective prosody due to right hemisphere damage across languages despite the finding that speakers of tone and non-tone languages may use somewhat different cues to signal affect (see also Hughes et al. 1983). Similar results were reported by Gandour et al. (1995) for Thai-speaking right hemisphere-damaged patients.

Interestingly, all of these studies utilized patients in a relatively acute stage, with the lengthiest time since stroke onset a maximum of three months for three of Gandour *et al.*'s (1995) 12 right hemisphere-damaged subjects. Time post-onset may prove to be a critical variable in the emergence of dysprosody.

In contrast to these results, numerous investigations have failed to find differences between right hemisphere-damaged patients and normal controls on tasks tapping the production of affective prosody; others have shown comparable patterns of deficit in both left hemisphere-damaged and right hemisphere-damaged patients. For instance, Brådvik and colleagues (1990, 1991) reported no significant impairments in the production of emotional prosody in a fairly large group of Swedish-speaking right hemisphere-damaged patients with cortical lesions. They contend that subcortical damage may be more likely to yield a prosodic production deficit than cortical right hemisphere damage. The authors note that their patients were mainly in a stable chronic stage, with most patients at least 4 months postonset at the time of testing; Brådvik *et al.* (1990) admit the possibility that the patients had had an acute dysprosody which had resolved during the period of spontaneous recovery (see also Darby 1993). Nonetheless, a significant lasting prosodic production deficit may not be associated with damage to the right cerebral cortex (Brådvik *et al.* 1990).

In a recent study of stable right hemisphere-damaged patients (>3 months postonset), Baum and Pell (1997) also found that right hemisphere-damaged patients, as well as left hemisphere-damaged patients, were able to signal different emotions in a manner comparable to normal control subjects. Both repetition and reading tasks were utilized to elicit emotional and linguistic prosodic contrasts based on stimuli with three degrees of linguistic structure. Model stimuli were either filtered of phonetic content, composed of nonsense syllables, or semantically well-formed and emotionally-biased. Acoustic analyses of productions in all conditions indicated similar patterns of acoustic cue manipulation in all groups tested. Overall, the right hemisphere-damaged patients did exhibit a lower mean $\tilde{F_0}$ than the normal controls (after normalization) and a somewhat restricted F₀ range, suggesting the potential for a right hemisphere role in the global control of F_0 ; however, the ability to signal affective prosodic contrasts was spared in the right hemispheredamaged patients (Baum and Pell 1997, see also Shapiro and Danly 1985). (These data will be discussed further in the section on linguistic prosody.) There are several potential reasons for the discrepancy between these findings and those of Ross and colleagues (Gorelick and Ross 1987, Ross 1981, 1993), including the difference in time post-onset and the reliance on objective acoustic measures as opposed to clinical perceptual judgments.

Yet, even in acute patients, not all studies yield results indicative of a right hemisphere specialization for affective prosody. In particular, Cancelliere and Kertesz (1990) found an approximately equal percentage of acute left hemispheredamaged and right hemisphere-damaged patients were classified as dysprosodic (but cf. Ross *et al.* 1997, for arguments contending that affective prosodic deficits subsequent to left hemisphere damage are a result of impaired callosal integration). Lesion sites were determined from CT scans obtained for 28 right hemispheredamaged and 18 left hemisphere-damaged patients. The investigators utilized Ross' (1981) classification system for aprosodias and examined the regions of greatest lesion overlap corresponding to each syndrome Analogous regions of the left and right hemispheres were generally implicated within each syndrome. Global

aprosodics displayed the greatest overlap in the basal ganglia, insula, and somewhat less in the perisylvian cortex. Lesions associated with motor aprosodia most often appeared in the inferior frontal lobes, insula and basal ganglia. The greatest lesion overlap for the sensory aprosodic patients was also in the insular cortex (Cancelliere and Kertesz 1990). (Interestingly, recent neuroradiological studies have demonstrated the importance of the insula (on the left) in the control of articulatory implementation (Dronkers *et al.* 1992)). The results do not support Ross' (1981) model of right hemisphere organization. Based on their review of lesion localization, the investigators concluded that the basal ganglia are most often implicated in dysprosodysyndromes; coupled with data on processing of emotional facial expression and gesture, the findings suggest that emotional expression is likely mediated subcortically (Cancelliere and Kertesz 1990).

The data considered above on the relationship between right hemisphere damage and the production of affective prosody appear equivocal. However, in order to draw firm conclusions on the issue of laterality, it is essential to investigate the production of linguistic prosody in order to determine whether any prosodic deficits found in right hemisphere-damaged patients extend to the linguistic domain as well. That is, one must establish whether right hemisphere damage results in a pervasive impairment in prosodic production, or a deficit limited to the communication of affect.

Linguistic prosody

Of the many studies of prosodic disturbance subsequent to brain damage, only a relatively small number have directly compared affective and linguistic prosody in the same patients. In several of the investigations of tone languages reviewed above, a clear dissociation has been found between impairments of affective prosody and intact abilities to process linguistic prosody subsequent to right hemisphere damage (Gandour *et al.* 1992, Gandour *et al.* 1993, Gandour *et al.* 1995, Hughes *et al.* 1983). Findings are much less clear-cut for non-tone languages. This dichotomy raises the important issue of the level or degree of linguistic function under investigation.

As noted earlier, in tone languages, variations in F₀ at the syllable level serve to differentiate lexical items and thus represent phonemic oppositions in those languages. In languages such as English, in contrast, tone does not in and of itself distinguish lexical items, but stress differences are a major component in the differentiation of noun/verb pairs (e.g. **con**vict/con**vict**) and compound noun/ noun phrase pairs (e.g. hotdog/hot dog). At a 'higher' level (e.g. Garrett 1980, Levelt 1989), stress can serve to emphasize or focus a specific word in an utterance, as in the contrastive 'John (not Bill) sold the car.' Finally, variations in intonation serve as cues to syntactic segmentation and sentence type, distinguishing declarative sentences from interrogatives and imperatives. This spectrum of the uses of linguistic prosodic cues has been described as a functional hierarchy by Van Lancker (1980 following Crystal 1969). In her consideration of the role of 'pitch cues', the affective level represents the least linguistically-structured extreme on the hierarchy, followed by the closely-related attitudinal level, and then the syntactic functions of intonation including perhaps emphatic stress. The most linguisticallystructured functions of prosody are the differentiation of lexical items and

phonemic uses of tone (Van Lancker 1980). In normal speech production, many of the same acoustic parameters serve to signal contrasts at all of these levels, demonstrating the necessity of their interaction and integration.

From this description of the functional hierarchy, it should be clear that not only does the degree of linguistic structure vary, but so does the domain over which prosodic parameters must be manipulated or controlled (Behrens 1989, Gandour *et al.* 1992, Van Lancker 1980). That is, phonemic distinctions apply at the segmental or syllable level while lexical contrasts apply at the word or phrase level and syntactic contrasts apply at the sentence level (Behrens 1989, Gandour *et al.* 1992, Van Lancker 1980). The domain of planning has been shown to be an important factor in determining the extent of speech production impairments in both left and right hemisphere-damaged individuals (Baum 1992, Behrens 1989, Gandour *et al.* 1994). Therefore, in examining prosodic production, all of these factors should be taken into consideration.

Numerous studies have examined the production of linguistic prosody, mainly investigating subjects who have suffered left or right hemisphere damage. One of the first systematic studies focused on the right hemisphere's potential role in linguistic prosody. Weintraub et al. (1981) presented to a single listener utterances that had been produced following a model by 9 right hemisphere-damaged subjects and 10 normal controls. The stimuli included declarative and interrogative sentences and sentences with emphatic stress. The listener was asked to judge how similar the subjects' productions were to the model stimuli. Results indicated that the right hemisphere-damaged subjects' utterances were consistently judged to be less adequate exemplars in relation to the model than those of the normal control group. Weintraub et al. (1981) concluded that right hemisphere damage yields a deficit in linguistic prosody, as well as affective prosody; they further predicted that a similar deficit subsequent to left hemisphere damage would be unlikely to emerge and therefore that the right hemisphere is dominant for prosodic production in general. The task upon which these conclusions are based is open to a great deal of criticism, rendering the data suspect. Listener judgments may be quite subjective and those of a single listener are even more prone to bias. Moreover, the absence of a left hemisphere-damaged control group raises questions about the veracity of the claims of right hemisphere dominance for both affective and linguistic prosodic production.

In an effort to remedy this situation, Danly and Shapiro (1982, Danly *et al.* 1983) investigated characteristics of sentence intonation in left hemisphere-damaged aphasic patients. Following the methods of Cooper and Sorenson (1981), the researchers demonstrated that Broca's aphasics—whose nonfluent speech is characterized by hesitations and reduced melodic line (Goodglass and Kaplan 1983)—displayed abnormal F_0 declination patterns in long sentences, increased variability in F_0 (but cf. Colsher *et al.* 1987) and abnormal patterns of final-lengthening effects. The fluent, Wernicke's aphasics they tested exhibited increased F_0 variability relative to normal controls, but fairly normal patterns of declination overall. Danly *et al.* (1983) suggested that Wernicke's aphasic patients may have deficits in the control of F_0 that are not perceptible to listeners. These findings, coupled with impairments in sentence-level linguistic prosody in right hemisphere-damaged patients (Shapiro and Danly 1985) do not support a strict right or left hemisphere lateralization for prosodic production.

In one of the few studies of sentential intonation to examine both right

hemisphere-damaged and left hemisphere-damaged patients, Cooper et al. (1984) reported higher than normal mean F_0 in a reading task in both brain-damaged groups; the left hemisphere-damaged speakers deviated more from normal in terms of both timing and F_0 . However, due to the small number of subjects per group (4 right hemisphere-damaged, 5 left hemisphere-damaged, 4 normal controls), no statistical analyses were conducted. Behrens (1989) examined the acoustic characteristics of sentence intonation in a story-completion task. Eight right hemisphere-damaged subjects and 7 normal controls produced declarative, imperative, yes/no and WH-interrogative sentences. Results revealed that the right hemisphere-damaged patients produced the intended target sentence type in, on average, only 14 of 24 trials (as judged by a panel of listeners). Acoustic analyses demonstrated that the productions of the right hemisphere-damaged subjects exhibited less linear F_0 contours and flatter than normal slopes for declarative sentences. On the basis of these and other findings (to be discussed below), Behrens (1989) concluded that right hemisphere damage may lead to impairments in the production of sentence-level intonation, highlighting the domain over which prosody is programmed as an important factor in predicting whether or not a deficit would emerge.

Inconsistent with these results, Ryalls *et al.* (1987) found no significant differences among groups of anterior right hemisphere-damaged, posterior right hemispheredamaged, and normal control subjects in mean F_0 , F_0 range and a measure of slope extracted from sentences elicited in a repetition task. Although a larger group of subjects participated in this experiment in comparison to Behrens' (1989) investigation, a rather small number of utterances was utilized, possibly accounting for some of the contradictory findings. In an earlier study, Ryalls (1982) reported a restriction in F_0 range in a group of 8 French-speaking left hemisphere-damaged Broca's aphasics, leading to the tentative conclusion that the control of F_0 at the sentence level may be lateralized to the left hemisphere (consistent with the functional load hypothesis (Van Lancker 1980).

Most recently, Baum and Pell (1997) demonstrated that both left hemispheredamaged and right hemisphere-damaged patients were capable of signalling declarative, interrogative and imperative sentences utilizing normal patterns of prosodic cues; as found for affective prosody, these results emerged independent of the linguistic 'load' of the stimuli, with the same patterns found for repetition of filtered sentences, utterances made up of nonsense syllables and semantically and syntactically well-formed sentences. Baum and Pell (1997) suggested that prosody may not be functionally lateralized and that alternative hypotheses should be considered. Two of the possibilities noted were that particular acoustic parameters may be differentially lateralized (e.g. Van Lancker and Sidtis 1992, see Baum *et al.* 1997 for partially supportive data in the production of acoustic cues to phrasal boundaries) or that prosody may be under subcortical control (Blonder *et al.* 1989, Brådvik *et al.* 1991, Cancelliere and Kertesz 1990).

All of the investigations reviewed thus far have focused on sentential intonation. If, as suggested by Behrens (1989) and others, the domain or linguistic level at which prosody is functioning play a substantive role in the presence or absence of deficits subsequent to brain damage, it is critical to examine other aspects of linguistic prosody as well. Let us turn first to the comparatively few studies of production of emphatic and lexical stress, and then consider analyses of phonemic tone.

Behrens (1988) conducted acoustic and perceptual analyses of lexical stress pairs and pairs of sentences with emphatic stress contrasts produced by the same right hemisphere-damaged and normal control subjects who participated in the sentence intonation study described above. Duration, amplitude and F_0 measures were computed for stressed and unstressed syllables elicited in a scenario-completion paradigm. Results of the acoustic analyses revealed that the right hemispheredamaged subjects used fewer of the cues to lexical and emphatic stress than did normals, but that they were able to signal stress, as determined by perceptual identification scores. Behrens' conclusion was that the right hemisphere is probably not dominant for linguistic prosody at the word level.

Fairly comparable results were reported by Emmorey (1987) for her right hemisphere-damaged subjects' production of lexical stress pairs. An important addition in Emmorey's (1987) investigation was the inclusion of a comparison group of left hemisphere-damaged subjects. Interestingly, it was these speakers who exhibited marked deficits in signalling lexical stress. In particular, left hemisphere-damaged subjects failed to adequately utilize duration and F_0 parameters to differentiate the stress contrasts as determined both by acoustic and perceptual analyses. Emmorey (1987) interpreted her findings as indicative of a functional organization for prosodic lateralization, with an additional important determinant being the size or domain of the unit planned (see also Behrens 1988, 1989).

In a replication and extension of Behrens' (1988) study, Ouellette and Baum (1994) found that both lexical and emphatic stress were adequately produced by right hemisphere-damaged patients. Consistent with Emmorey (1987), left hemisphere-damaged subjects were impaired only in the use of durational parameters. Ouellette and Baum's (1994) interpretation of the findings focused on the temporal control deficit often reported in left hemisphere-damaged aphasic patients (see Blumstein 1991, Blumstein and Baum 1987 for reviews). The authors granted that the findings may be interpreted to support the functional lateralization hypothesis (Van Lancker 1980), with more linguistically-structured prosody controlled by the left hemisphere; nonetheless, the possibility of differential lateralization for specific cues to prosody was also underscored. In particular, these results support the hypothesis that the control of duration may be subserved by left hemisphere mechanisms (but cf. Dykstra et al. 1995). Other parameters (e.g. F_0) may be under the control of right hemisphere centers (Baum and Pell 1997, Van Lancker and Sidtis 1992). In general, there appears to be fairly good agreement in the data on word-level stress production by brain-damaged patients. The findings tend to support the role of the left hemisphere in the control of duration as a prosodic cue, with little role for the right hemisphere at this level.

As mentioned earlier, the most linguistically-structured function of prosody is the use of tone as a phonemic contrast in languages such as Norwegian, Thai and Chinese. Although cross-language data are not abundant, most of the studies that have been conducted indicate that the phonemic use of tone is a left hemisphere function. For example, Gandour and colleagues (1992) utilized acoustic and perceptual analyses to examine the control of F_0 in signalling tone contrasts in monosyllabic lexical items in Thai. Production data revealed minor differences from normals in the shape of the F_0 contour for both right hemisphere-damaged and left hemisphere-damaged speakers. Perception tests showed that the utterances

of the nonfluent left hemisphere-damaged aphasic speakers were least wellperceived by native Thai listeners, prompting the authors to conclude that, at this level or domain of prosodic function, the left hemisphere appears to be primarily involved. Packard (1986) reached a similar conclusion in his study of nonfluent left hemisphere-damaged aphasic speakers of Mandarin Chinese. Mono- and bi-syllabic productions were rated by three transcribers for accuracy of repetition of a model utterance. A much higher error rate was found for the left hemisphere-damaged aphasic speakers relative to a normal control group, leading to the claim that phonemic tone is controlled by the left hemisphere. Finally, results for left and right hemisphere-damaged speakers of Norwegian yielded impairments in tone production in the left but not the right hemisphere-damaged subjects (Ryalls and Reinvang 1986). As with the word-level stress phenomena, there is little controversy over the hypothesis that, due to their linguistic salience, the ability to produce phonemic tone contrasts is mediated by the left hemisphere.

Finally, to build upon studies reporting an association between focal brain damage and individual prosodic functions examined in isolation, a recent attempt was made to evaluate the success of brain-damaged patients in encoding prosodic representations which span different operational domains (word, sentence) and which assume different behavioural functions (linguistic, emotional) when expressed *in tandem*. For this undertaking, Pell (1999a, b) employed a story completion paradigm to elicit short utterances varying in contrastive stress placement, linguistic modality and emotional tone from matched groups of right hemisphere-damaged and healthy normal speakers (n = 10/group). Variations in the content of a 'priming scenario' preceding each trial biased specific combinations of the three prosodic variables when each target utterance was produced, without varying the segmental form of the utterance. Acoustic analysis of the data was then performed to determine whether right hemisphere-damaged and normal speakers differ in the ability to utilize duration or fundamental frequency to communicate specific prosodic target meanings.

Results of the acoustic analysis, for measures of both fundamental frequency and duration, pointed to a general sparing in the ability of right hemisphere-damaged patients to mark both word and sentence-level linguistic-categorical distinctions in their speech (e.g., contrastive elements displayed normal tendencies for vowel lengthening and increased F₀; declarative/interrogative distinctions were distinguished by a fall versus rise in F_0 in the terminal portion of the utterance). In contrast, acoustic distinctions among emotional categories, although demonstrating normal qualitative trends overall, tended to be fewer and smaller in magnitude when produced by the right hemisphere-damaged speakers. Moreover, the patients encoded certain linguistic constructs (contrastive stress, the terminal rising contour for interrogatives) with significantly less acoustic variation than that typical of normal speakers. The apparent failure of the right hemisphere-damaged speakers to supply a normal level of acoustic detail in conveying emotion and linguistic focus had a significant impact on the perceptibility of these parameters by a group of normal listeners (Pell 1998b). The author interpreted this pattern of findings as indicative of a possible motor disturbance in modulating graded aspects of prosodic stimuli subsequent to right hemisphere insult (Pell, 1999a, b), consistent with prior data collected for a single right hemisphere-damaged speaker (Blonder et al. 1995).

Summary

In summary, investigations of prosodic production have yielded support for a weak version of the functional lateralization hypothesis (Van Lancker 1980). The majority of studies of phonemic and lexical tone contrasts have demonstrated an association of production deficits with damage to the left hemisphere; impairments in tone production subsequent to right hemisphere damage are uncommon, suggesting left hemisphere control (Behrens 1988, Emmorey 1987, Gandour et al. 1992, Ouellette and Baum 1994, Packard 1986, Ryalls and Reinvang 1986). At the other levels of the functional hierarchy, the findings have been much more equivocal, prompting the development of alternative theories (Behrens 1989, Pell 1998a, Van Lancker and Sidtis 1992). As we have seen, one possibility that has received some support in the literature posits that the size or domain of the utterance planned is a variable of importance in lateralization; segmental and wordlevel prosody may be controlled by the left hemisphere whereas sentence-level prosody, whether affective or linguistic, may require participation of both hemispheres (Baum and Pell 1997, Behrens 1989). The hypothesis that individual acoustic parameters are differentially lateralized has also received limited support in production studies (mainly with respect to duration) (Baum and Pell 1997, Baum et al. 1997, Ouellette and Baum 1994). Although, at present, no incontrovertible evidence is available to support or refute any one of the theories on the basis of production data, a more compelling case may be made following an in-depth consideration of the perception of prosody.

Perception/comprehension of prosody

Perceptual investigations of prosody, like the production studies described in the foregoing discussion, have tended to concentrate on either the affective or linguistic role of prosodic cues in speech (i.e. their assumed *functional* significance) or their operational domain (e.g. word vs utterance). Perhaps distinct from the production literature, a greater proportion of the evidence for the perceptual lateralization of prosody has been derived from studies of *non-pathological* performance using the dichotic listening technique (Kimura 1961). However, as will become evident from our review, analysis of prosodic abilities in brain-damaged individuals has remained the primary vehicle of inquiry into the neurological substrates of prosody in the receptive as well as the expressive mode.

Affective prosody

Studies of the perception and recognition of emotional attributes of speech have contributed greatly to the hypothesis that right hemisphere mechanisms are selectively engaged in the processing of affective prosody. In an early report that focused on the comprehension of affective speech Heilman *et al.* (1975) presented auditory stimuli to 6 left hemisphere-damaged and 6 right hemisphere-damaged subjects with temporoparietal lesions in two tasks: one in which subjects labelled the emotional mood of the speaker (happy, sad, angry, indifferent) and one in which subjects identified the semantic content of the same utterances. Judgments were indicated by pointing to line drawings of emotional facial expressions (emotion condition) or a graphic depiction of the semantic interpretation of the utterance (content condition) and the accuracy of each response was recorded.

Although both patient groups performed without error in interpreting the semantic meaning of the stimuli, results obtained in the emotion condition indicated that right hemisphere-damaged patients (who also presented with behavioural neglect) were significantly impaired relative to left hemispheredamaged aphasic patients in the ability to categorize the affective meaning of prosodic cues, performing at near chance level. In a replication and extension of Heilman et al.'s (1975) study, Tucker et al. (1977) obtained a similar pattern of results, reporting poorer comprehension of emotional prosody in right hemisphere-damaged patients with neglect than in left hemisphere-damaged aphasic patients (again, right hemisphere-damaged patients identified the four emotions at chance level). A disturbance in the ability to discriminate differences in prosodic patterns (i.e. make same/different judgments about paired stimuli differing in prosodic content) was also manifest in the right hemisphere-damaged but not the left hemisphere-damaged subjects (Tucker et al. 1977). Based on these data, both groups of investigators concluded that temporoparietal lesions of the non-dominant hemisphere in conjunction with behavioural neglect may lead to a selective impairment in the comprehension of affective prosody (Heilman et al. 1975, Tucker et al. 1977). However, the strength of these assertions is mitigated by the observation that left hemisphere-damaged patients were not error-free in their comprehension of affective meanings in either study, coupled with the absence of a non-neurologically-impaired control group in both paradigms.

The results of more recent experiments have also been interpreted in light of a privileged right hemisphere role in the comprehension of affective prosody. For example, Bowers et al. (1987) required 9 right hemisphere-damaged, 8 left hemisphere-damaged and 8 healthy control subjects to identify emotional-prosodic meanings from several different types of stimuli. These included utterances in which the semantic and prosodic message were either congruent or incongruent (e.g. 'the couple beamed at their new grandson' spoken in a happy or sad tone), as well as utterances that had been low-pass filtered of all identifiable linguistic content, preserving only the prosodic contour (i.e. 'speech filtered' stimuli). For all the conditions tested, the right hemisphere-damaged group exhibited significant deficits relative to both the left hemisphere-damaged and normal control groups in the recognition of the emotional tone of the stimuli, suggestive of right hemisphere control of these processes. Similarly, Blonder et al. (1991) reported a global decline in the ability of their right hemisphere-damaged patients to process the emotional significance of prosodic, facial and gestural communicative signals when compared to left hemisphere-damaged and non-neurologically-impaired control subjects; this outcome was interpreted as indication of the primacy of the right hemisphere in the modulation of perhaps *all* (nonverbal) aspects of emotional communication (see also Borod 1993).

To test the effects of 'associational-cognitive' demands on the processing of emotional prosody, Tompkins and Flowers (1985) presented emotionally-intoned, semantically-neutral phrases to 11 right hemisphere-damaged, 11 left hemispheredamaged and 11 control subjects in three tasks of presumably increasing cognitive complexity: a same/different discrimination task, an identification task in which subjects chose one of two possible emotional interpretations and an identification task in which subjects judged the emotion from four possible alternatives. Consistent with other findings (Blonder *et al.* 1991, Bowers *et al.* 1987, Heilman *et al.* 1975, Tucker *et al.* 1977), the authors found that their right hemispheredamaged patients performed at a significantly inferior level to matched normal subjects on all emotional prosody tasks. However, the left hemisphere-damaged patients' performance also broke down on the task in which the cognitive load was greatest (four-choice emotional identification). Thus, although their data appeared to supply further evidence that the right hemisphere subserves emotional-prosodic processing, the authors postulated that the left hemisphere becomes engaged in such tasks as cognitive demands increase, possibly due to greater need for comparative processes or short-term memory (Tompkins and Flowers 1985).

Evidence for right hemisphere superiority in recognizing emotional stimuli has also emerged from studies of prosody perception in normals. Employing the dichotic listening paradigm, Ley and Bryden (1982) paired emotionally intoned (happy, sad, angry, neutral) and monotone sentences of similar grammatical construction (e.g. 'the boy went to the store') for presentation to 32 young adults. Subjects were asked to attend to a specified ear and identify both the emotional tone and the verbal content of each sentence from a fixed set of alternatives. Analysis of subjects' accuracy for each type of stimuli yielded a significant left-ear (right hemisphere) advantage for judging emotions and a significant right-ear (left hemisphere) advantage for judging the verbal content, with the majority of subjects (n = 21/32) showing both trends simultaneously. Thus, normative data indicating differential lateralization of emotional and verbal processing in young normal subjects (consistent with a right hemisphere superiority in the comprehension of affective prosody) have come to light.

As noted earlier, Ross (1981) has not only advocated right hemisphere control of emotional prosody and gesture, but has elaborated a hypothetical model that places emotional-prosodic functions-both expressive and receptive-in circumscribed regions of the right cerebral cortex of the brain. In the receptive as well as the expressive mode, the work of Ross and his colleagues (Gorelick and Ross 1987, Ross 1981, Ross et al. 1981, Ross and Mesulam 1979) has relied to a large extent on bedside assessment of patients with acute right hemisphere lesions and suspected 'aprosodia'. To evaluate affective comprehension, the patient is asked to identify (either verbally or by means of a set list of alternatives) the emotion projected by the examiner who is positioned out of the patient's view (Ross 1981, 1993). This (rather uncontrolled) technique has been instrumental in rendering several case descriptions of right hemisphere-damaged patients with posterior (temporoparietal) lesions and 'receptive aprosodia' (i.e. impaired affective comprehension in the face of spared affective production and repetition), cases which have been cited as key in validating Ross' functional-anatomic organization of the aprosodias in the right hemisphere (Gorelick and Ross 1987, Hughes et al. 1983, Ross 1981, Ross et al. 1981).

Despite the valuable contribution of these clinical reports, there are now sufficient data derived from several different paradigms to warrant the abandonment of Ross' putative classification system for the aprosodias. Individual cases of receptive aprosodia reported by several investigators (Brådvik*et al.* 1991, Darby 1993, Heilman *et al.* 1984, Lebrun *et al.* 1985) clearly diverge from Ross' hypothetical model. For example, Cancelliere and Kertesz' (1990) study of the relationship between acute vascular lesions and disturbances of emotional expression and comprehension uncovered no evidence that aprosodic deficits in right hemisphere-damaged patients adhere to the anterior-posterior pattern described by Ross (1981); the authors attributed this discrepancy to their use of

standardized stimuli and a less biased assessment procedure. Of perhaps greater importance, Cancelliere and Kertesz reported emotional comprehension deficits of comparable frequency in both the right- *and* left-hemisphere-damaged adults they examined, calling into question the very notion that the right hemisphere is uniquely engaged in the processing of affective speech (but cf. Ross *et al.* 1997). Indeed, as noted previously (Van Lancker and Sidtis 1992), evidence that left hemisphere mechanisms possess some capacity to process emotional-prosodic stimuli would serve to explain purported cases of 'crossed aprosodia' or right hemisphere-damaged patients without the anticipated prosodic difficulties according to Ross' scheme (Gorelick and Ross 1987).

Cancelliere and Kertesz' (1990) observations that both the right and left cerebral hemispheres contribute to the processing of affective vocal cues obtain support from several additional investigations (Darby 1993, Pell 1998a, Schlanger et al. 1976, Starkstein et al. 1994, Van Lancker and Sidtis 1992). Schlanger et al. (1976) found no significant differences in the accuracy of right hemisphere-damaged (n = 20) and left hemisphere-damaged (n = 40) subjects in recognizing semantically neutral (e.g. He will come soon) or semantically anomalous (He will tuv roop) stimuli, indicative of bilateral control of emotional prosody. Similarly, in their examination of the prevalence of receptive prosodic deficits in 59 consecutivelyadmitted patients with cerebrovascular lesions, Starkstein and his co-workers (1994) reported that disturbed comprehension of affective prosody was a relatively frequent feature in both left hemisphere-damaged and right hemisphere-damaged acute stroke patients ($\sim 45\%$ of their subject pool). Interestingly, the authors did note that right hemisphere-damaged patients with basal ganglian or temporoparietal lesions exhibited a significantly greater incidence of such deficits in their sample. Thus, bilateral involvement in affective prosody comprehension is once again indicated, although Starkstein et al.'s (1994) findings intimate the possibility that right hemisphere mechanisms may play a more predominant role.

A recent investigation conducted by Pell (1998a) provides additional evidence of a bilateral substrate for emotional prosody comprehension, while simultaneously bolstering claims that the right hemisphere is somehow 'special' or dominant for specific (as yet undetermined) subcomponents of emotional prosody recognition. Short utterances distinguished solely by their prosodic features (stimuli differed with respect to emphasis assignment, linguistic modality, and emotional tone) were presented over headphones to 11 left hemisphere-damaged, 9 right hemispheredamaged and 10 normal individuals. Subjects listened to this common set of utterances over several conditions which manipulated the strength of particular acoustic parameters of the stimuli, and were required to independently judge either the location of emphatic stress within the sentence (initial, final, none) or the emotional tone (happy, angry, sad, neutral). Results indicated that although emphasis perception was uniquely disturbed in the left hemisphere-damaged sample, accuracy in recognizing emotional attributes of the same stimuli was significantly impaired in both right hemisphere-damaged and left hemispheredamaged subjects relative to age-matched controls. For emotional prosody, this pattern advances the position that distributed mechanisms in both hemispheres of the brain may be necessary for such processing. However, the observation that right hemisphere-damaged patients were selectively impaired in the emotion condition relative to the linguistic (emphasis) condition (the accuracy of the left hemisphere-damaged patients did not differ across conditions) implies that the

locus of certain operations inherent to emotional perception and evaluation may stem from a unique right hemisphere mechanism (Pell 1998a). Delineating the components within this functional system that favour right versus left hemisphere processing mechanisms remains a considerable challenge for future research.

A different perspective on the contributions of left and right hemisphere mechanisms in the comprehension of affective-prosodic stimuli has been formulated by Van Lancker and Sidtis (1992). They tested left hemisphere-damaged, right hemisphere-damaged, and healthy control subjects on an emotional prosody identification task, observing a similar level of impairment in the accuracy of both clinical groups. To further explore whether the comprehension errors of left hemisphere-damaged and right hemisphere-damaged patients could be predicted in terms of one or a combination of the acoustic parameters underlying emotionalprosodic meanings, the authors determined mean and variability measures of F₀, amplitude, and duration for the stimuli they had presented to patients for perceptual recognition. Discrimination function analyses were then performed to ascertain which of the acoustic cues served to signal the intended emotional meanings of the stimuli initially presented, and which cues predicted the comprehension errors made by each clinical group on the identification task; this procedure involved recoding each emotional stimulus according to the most frequent error response observed for that stimulus, independently for each group. In this way, the authors sought to determine the extent to which the left hemisphere-damaged and right hemisphere-damaged subjects' emotional comprehension deficits were related to impaired perception of specific acoustic features of the stimuli.

Despite the similar level of impairment of left hemisphere-damaged and right hemisphere-damaged patients on the emotional identification task, analyses performed on each group's recognition errors suggested that left hemispheredamaged and right hemisphere-damaged patients were using the acoustic cues to prosody differently in judging affective meanings (Van Lancker and Sidtis 1992). Interestingly, the discriminant analysis of the left hemisphere-damaged subjects' errors revealed that these patients may have been basing their decisions on fundamental frequency information (particularly F_0 variability), whereas an analysis of the right hemisphere-damaged subjects' affective misclassifications indicated a reliance on durational cues in identifying the stimuli. This pattern of results suggested to the authors that receptive disturbances of emotional prosody may be perceptual in nature, possibly reflecting the superiority of each hemisphere in processing different acoustic parameters that signal prosodic meaning (Van Lancker and Sidtis 1992). More generally, the authors concluded that the comprehension of prosody is best described as a multifaceted process subserved by distributed (i.e. bilateral) mechanisms that are not strictly localizable to the right hemisphere, contrary to previous assertions (e.g. Ross 1981).

The notion that brain-damaged patients are characterized by a basic disturbance in analyzing the acoustic structure of prosody complements reports that these patients often show deficits in using the same auditory cues in *nonlinguistic* tasks. More specifically (and as previously noted), right hemisphere-damaged individuals frequently appear to make errors on nonlinguistic tasks that require the processing of complex pitch information, indicating that this skill may rely predominantly on right-hemisphere auditory mechanisms (Robin *et al.* 1990, Sidtis and Feldmann 1990, Zatorre 1988, Zatorre *et al.* 1994). Interestingly, a left-hemisphere bias has been proposed for the processing of *temporal* cues on similar nonlinguistic tasks (Carmon and Nachshon 1971, Robin *et al.* 1990). Collectively, these data are consistent with the interpretation that each hemisphere may contribute independent auditory processing capabilities to the task of decoding emotional stimuli (Van Lancker and Sidtis 1992). However, in an attempt to replicate Van Lancker and Sidtis' preliminary findings, Pell and Baum (1997b) found no evidence that the emotional comprehension errors committed by their left hemisphere-damaged and right hemisphere-damaged patients were biased by specific acoustic features of the stimuli, despite careful adherence to Van Lancker and Sidtis' original methods. Thus, although intriguing, the hypothesis that individual acoustic cues to prosody are independently lateralized (Van Lancker and Sidtis 1992) remains speculative and awaits future elucidation.

Linguistic prosody

Thus far, our consideration of theories of receptive prosodic lateralization has concentrated on affective prosody, but the discussion may benefit from a review of the linguistic functions of prosodic cues as well. As noted in the section on production, prosodic features expressed over various domains signal differences in the illocutionary intent of an utterance (e.g. whether information is stated or requested), highlight items of relative importance in a spoken message (emphasis), or disambiguate the meaning of words with similar segmental structure (phonemic stress). Several investigators have explored the neural basis for comprehension of locally defined linguistic-prosodic features such as phonemic or emphatic stress. In response to contentions in the literature that right hemisphere lesions selectively disrupt affective prosody, Weintraub et al. (1981) tested 9 right hemispheredamaged and 10 normal control subjects for the comprehension, production, and repetition of linguistic prosody. One receptive task measured subjects' accuracy in discriminating phonemic stress contrasts (e.g. greenhouse vs green house) using a picture-identification paradigm and another measured their accuracy in making same/different judgments about sentence pairs differing in emphatic stress location (e.g. Steve drives the car vs Steve drives the car) or intonation contour (statement vs yes/no question).

Results obtained for each linguistic prosody task revealed significant impairments in the right hemisphere-damaged group relative to the control subjects, a pattern interpreted as evidence that the right hemisphere's role in prosody may extend beyond its affective components to the linguistic domain (Weintraub *et al.* 1981). More recently, Brådvik *et al.* (1991) compared the performance of 20 Swedish-speaking patients with stable right hemisphere lesions and 18 normal controls on tasks of both linguistic and affective prosody (e.g. emphatic stress perception, identification of linguistic and emotional intonation) and arrived at a similar conclusion: the inferior performance of their right hemisphere-damaged patients on both linguistic and emotional tasks pointed to an essential role for the right hemisphere in the processing of *both* (linguistic and affective) prosody, irrespective of the domain over which prosodic cues were perceived. The potential relationship between subcortical infarcts and a lasting disturbance of speech prosody, alluded to in the discussion of production, was also highlighted by their data (Brådvik *et al.* 1991).

The omission of a comparable left hemisphere-damaged patient group in the

latter two studies (Weintraub et al. 1981, Brådvik et al. 1991) again impedes an appropriate understanding of each hemisphere's potential involvement in prosodic perception. In a study that considered right hemisphere-damaged, left hemispheredamaged, and non-neurological control subjects simultaneously (n = 30/group), Bryan (1989) presented a battery of 13 linguistic prosody tests that incorporated stimuli of various perceptual domains (e.g., phonemic/emphatic stress discrimination, identification of declarative vs interrogative intonation). Bryan demonstrated that the right hemisphere-damaged patients were impaired on all 13 tasks of linguistic prosody relative to the normal group and on 8 tasks relative to the left hemisphere-damaged group, again favouring a right hemisphere basis for this processing. However, it is noteworthy that the left hemisphere-damaged group reported by Bryan (1989) was significantly impaired relative to the control group on 10 of the 13 tests as well, a finding the author conceded may be suggestive of bilateral control for at least some aspects of linguistic prosody. This pattern of results may be indicative of a superior (albeit not exclusive) role for the right hemisphere in the comprehension of linguistic prosody, along the lines suggested earlier for the comprehension of emotional prosody (Starkstein et al. 1994).

However, still more research has placed the receptive control of linguistic prosody-at least, the perception of locally-assigned stress cues-firmly in the left hemisphere of the brain. For instance, Baum et al. (1982), following Blumstein and Goodglass (1972), presented three tasks of stress comprehension to 8 left hemisphere-damaged nonfluent aphasics and 8 normal control subjects and reported a significantly reduced capacity to comprehend phonemic and emphatic stress in their left hemisphere-damaged patients, findings inconsistent with the notion that linguistic prosody is processed solely by the right hemisphere. Emmorey (1987) presented phonemic stress pairs (e.g. hotdog, hot dog) to 7 right hemisphere-damaged, 15 left hemisphere-damaged and 22 control subjects for perceptual recognition and observed a significant decrement in the performance of the left hemisphere-damaged subjects (both fluent and nonfluent aphasics) relative to control subjects on this task, but intact comprehension of the stimuli by right hemisphere-damaged patients. These data corroborate and extend those of Baum et al. (1982), indicating a left hemisphere substrate for the perception of linguistic stress.

Related findings are derived from Behrens (1985) in which the dichotic listening technique was used; she required 15 normal subjects to identify stress placement in phonemic stress pairs (e.g. hotdog, hot dog, as above) and demonstrated a significant right-ear (left hemisphere) advantage on this task. Filtering the same stimuli at 200 Hz for presentation or reducing the semantic content of the stimuli (e.g. botgog) did not lead to a right-ear advantage, however, suggesting to the author that left hemisphere mechanisms process stress contrasts *except* when those cues are of minimal linguistic import (as in the low-pass-filtered stimuli). Taken together, the results of these studies (Baum *et al.* 1982, Behrens 1985, Emmorey 1987) may be viewed as support for the 'functional load' hypothesis of prosodic lateralization, or the notion that the linguistic or emotional role of prosodic cues in speech determines the laterality of processing (Van Lancker 1980).

A recent study also tested the hypothesis that individual acoustic cues are differentially lateralized in the processing of phonemic and emphatic stress. Utilizing stimuli in which F_0 and duration cues were independently neutralized, Baum (1998) found that individuals with left hemisphere damage were most

severely impaired on these recognition tasks, particularly when deprived of F_0 information. However, right hemisphere-damaged patients also exhibited deficits compared to normal controls when not all acoustic cues were available in the stimuli. The findings are, in part, consistent with both the differential cue lateralization hypothesis (Van Lancker and Sidtis 1992) and the functional lateralization hypothesis (Van Lancker 1980), but suggest that both hypotheses require refinement or revision.

Further evidence of a left hemisphere substrate for the ability to perceive local, linguistically-assigned prosodic features is gleaned from perceptual investigations of languages in which pitch contrasts serve as a phonemic marker (e.g. Mandarin, Thai). The outcome of dichotic listening studies with normals (Van Lancker and Fromkin 1973) and of lesion investigations (Gandour and Dardarananda 1983, Hughes *et al.* 1983) are in general agreement, demonstrating a left hemisphere bias for the ability to discriminate tonal distinctions by native tone-language speakers. These reports, in conjunction with the English data reviewed above (Baum *et al.* 1982, Behrens 1985, Emmorey 1987), provide substantial evidence to suggest a privileged role for the left hemisphere in the processing of linguistically-assigned prosodic cues expressed at the syllabic level.

However, affective prosodic features are typically expressed and perceived over domains *larger* than the word, usually the phrase or utterance. It is at this level of linguistic structure—the sentence—that receptive studies of affective prosody have largely focused and accordingly, that we are best able to compare the perception of prosodic cues as an index of their linguistic or affective 'load' in speech. Regrettably, the perceptual literature on linguistic intonation is relatively small when compared to that on emotional intonation. In an early study to consider linguistic sentence prosody, Blumstein and Cooper (1974) presented dichotically paired utterances differing in intonational content to 40 young adults. In two separate experiments, subjects identified low-pass-filtered exemplars of the dichotic stimuli by their intonational meaning (declarative, interrogative, imperative, conditional) or matched the intonation pattern of filtered or nonsense (e.g. *padaka*) dichotic stimuli with a successively-presented foil. The accuracy of the subjects was then analyzed to determine the presence of an ear advantage on each task.

In general, the results revealed a 'small but consistent' left ear (right hemisphere) advantage for all tasks of perceiving and identifying linguistic intonation. The authors concluded from their findings that linguistic prosody, in the absence of meaningful segmental (i.e. semantic) structure, may be processed more efficiently by the right hemisphere, and that even when recognizable segmental information is present in the stimuli (as was the case for the nonsense stimuli), left hemisphere mechanisms are likely minimally implicated at best (Blumstein and Cooper 1974). These findings were contrary to the authors' original expectations that linguistic intonation may be processed by the left hemisphere in a way similar to other linguistic systems; the results provide tentative support to those investigators who have posited superior right hemisphere processing of sentence prosody generally, regardless of its function (Brådvik *et al.* 1991, Weintraub *et al.* 1981, but review Cancelliere and Kertesz 1990, Darby 1993, Schlanger *et al.* 1976, Starkstein *et al.* 1994, Van Lancker and Sidtis 1992 for data indicating left hemisphere control of emotional sentence prosody).

Few studies have attempted to explore how each hemisphere is specialized to process sentence prosody in *both* linguistic and affective contexts concurrently. In

one such study, Heilman and colleagues (1984) presented auditory stimuli to 8 right hemisphere-damaged, 9 left hemisphere-damaged, and 15 control subjects in two identification tasks, one in which intonation conveyed the linguistic modality of the utterance (declarative, interrogative, imperative) and another in which prosodic cues signalled various affective meanings (angry, sad, happy). Stimuli in both conditions were low-pass-filtered before presentation, rendering the segmental content, but not the prosodic contour, unintelligible to the listener. Subjects indicated their response either verbally or by matching the prosodic meaning with an appropriate graphic representation (facial expression or punctuation mark for the affective and linguistic stimuli, respectively) and the accuracy of each response was measured.

Right hemisphere-damaged patients made significantly more errors than both the left hemisphere-damaged and control subjects (who also differed significantly) in identifying the emotional meaning of low-pass-filtered utterances, whereas the right hemisphere-damaged and left hemisphere-damaged patients were equally impaired relative to normals in identifying the linguistic intent of the stimuli. Moreover, only the left hemisphere-damaged subjects' comprehension of prosodic meanings was affected by the type of prosody tested; specifically, the left hemisphere-damaged subjects performed at a significantly inferior level on the linguistic task when compared to the emotional task, a pattern not observed for either the right hemisphere-damaged or control groups. To account for these results, two hypothetical explanations were offered (Heilman et al. 1984). First, the processing of affective prosody may be lateralized to the right hemisphere of the brain (right hemisphere-damaged patients were most impaired on this task) whereas the processing of linguistic intonation may be achieved bilaterally (both patient groups were impaired relative to normals). Second, the right hemisphere may dominate all processing of sentence intonation (both linguistic and affective), but the left hemisphere becomes engaged on tasks as the need for linguistic processing increases (i.e. on non-affective tasks). The authors acknowledged that their data allowed for either interpretation; however, it is noteworthy that both proposals are inconsistent with previous assertions that the right hemisphere is specialized to decode only the affective features of prosodic stimuli (Blonder et al. 1991, Bowers et al. 1987, Ehlers and Dalby 1987, Heilman et al. 1975, Ley and Bryden 1982, Ross 1981, Tucker et al. 1977).

In a recent investigation, Pell and Baum (1997a) administered identification tasks for both affective- and linguistic-prosodic stimuli to 9 right hemisphere-damaged, 10 left hemisphere-damaged and 10 control subjects, testing the same target meanings employed by Heilman and colleagues (1984). To additionally address inconsistencies in the literature on prosody with respect to the type of stimuli presented (e.g. filtered, natural), linguistic and emotional stimuli were each presented in three distinct identification tasks: a semantically 'well-formed' condition, in which both prosodic and semantic information cued the (linguistic or affective) intonational target meaning; a 'nonsense' condition, in which phonetically-plausible but meaningless utterances were intoned to convey prosodic meanings corresponding to those presented in the well-formed stimuli; and, a 'filtered' condition, in which the well-formed utterances were low-pass filtered to obscure the linguistic content but retain prosodic cues. A task requiring subjects to make same/different judgments about pairs of low-pass-filtered utterances was also presented to test for an underlying perceptual deficit in the subjects' ability to process prosodic information (Van Lancker and Sidtis 1992). Both accuracy and response time data were collected.

Ålthough all three groups were shown to perform comparably in discriminating prosodic patterns, results of the identification tasks revealed that neither the left hemisphere-damaged nor right hemisphere-damaged subjects were impaired relative to normals in recognizing the emotional meaning of prosodic patterns (angry, sad, happy), but that both clinical groups exhibited deficient comprehension of linguistic-prosodic meanings (declarative, interrogative, imperative). Interestingly, comparing the performance of each group across linguistic and affective domains revealed a pattern qualitatively similar to that reported by Heilman *et al.* (1984); right hemisphere-damaged and control subjects each demonstrated similar capabilities on corresponding linguistic and affective tasks (reflected in both their accuracy and response times), whereas left hemisphere-damaged aphasic subjects *always* responded significantly slower and with less precision on the linguistic relative to the affective task (even though no semantic comprehension of the stimuli was required).

Thus, consistent with other studies (Heilman *et al.* 1984, Tompkins and Flowers 1985), a specific susceptibility to the linguistic load of prosodic stimuli was again noted in left hemisphere-damaged but not right hemisphere-damaged adults, although it is important to bear in mind that right hemisphere-damaged patients were also impaired for the linguistic stimuli. The perhaps surprising observation that neither clinical group was impaired in the comprehension of emotional prosody may have been due to clinical differences between Pell and Baum's (1997a) patients, who had been screened for behavioural neglect, and those tested elsewhere; indeed, the coincidence of lasting aprosodias and severe neurologic signs such as neglect have been noted previously on several occasions (Heilman *et al.* 1975, Starkstein *et al.* 1994, Tucker *et al.* 1977). Overall, the results of this study highlight the possibility that receptive prosodic functions, both linguistic and affective, may not be subserved by mechanisms lateralized to a single hemisphere of the brain, at least not when this processing occurs over larger domains such as the sentence (Pell and Baum 1997a).

Finally, other perceptual research suggests that the locus of mechanisms subserving prosody may not be limited to cortical regions, but rather, may be organized subcortically. In particular, the basal ganglia have been implicated as a structure of potential importance in several investigations of vascular patients with receptive aprosodias reviewed above (Brådvik et al. 1991, Cancelliere and Kertesz 1990, Ross and Mesulam 1979, Starkstein et al. 1994). These findings obtain further support from studies that have examined receptive prosody in patients with basal ganglia dysfunction as a result of Parkinson's or Huntington's disease (Borod et al. 1990, Blonder et al. 1989, Breitenstein et al. 1998, Cancelliere and Hausdorf 1988, Pell 1996, Scott et al. 1984, Speedie et al. 1990). For example, Blonder and colleagues (1989) and more recently Pell (1996), each demonstrated impaired comprehension of linguistic and emotional intonation in idiopathic Parkinsonian patients relative to healthy control subjects. Coupled with the cortical data on receptive prosody, the outcome of each of these investigations would appear to advocate a functional network dedicated to prosody consisting of both cortical and subcortical components (Blonder et al. 1989, Pell 1996, see also Breitenstein 1998). The issue of subcortical representation of prosody in both receptive and expressive behaviour is therefore worthy of pursual in future investigations.

Summary

In summary, our review of receptive investigations of prosody converges with that of production studies in its weak support of differential lateralization of prosodic cues as an index of their linguistic or affective communicative function in speech. To date, results emanating from studies of phonemic stress and pitch perception have demonstrated relatively consistent involvement of the left hemisphere and relatively infrequent involvement of the right hemisphere, signifying a left hemisphere neural substrate for linguistically-relevant prosodic cues operating over short domains (Behrens 1985, Emmorey 1987, Van Lancker 1980). However, it is at the sentential level that the effects of the functional load of prosodic cues become more opaque, and the issue of laterality becomes less certain. Although ample evidence has now accrued to suggest that the affective attributes of prosody are not processed uniquely by the right hemisphere (Cancelliere and Kertesz 1990, Darby 1993, Dykstra et al. 1995, Heilman et al. 1984, Pell 1998a, Pell and Baum 1997a, b, Schlanger et al. 1976, Seron et al. 1982, Tompkins and Flowers 1985, Van Lancker and Sidtis 1992), it remains unclear as to whether the right hemisphere serves a dominant (albeit shared) role in the processing of emotional and linguistic prosody (Blumstein and Cooper 1974, Heilman et al. 1984, Starkstein et al. 1994) or whether emotional and linguistic prosody functions are distributed bilaterally (Bryan 1989, Cancelliere and Kertesz 1990, Dykstra et al. 1995, Pell and Baum 1997a, b, Van Lancker and Sidtis 1992). Finally, subcortical structures may be critical in the regulation of prosodic functions in receptive and expressive modalities (Blonder et al. 1989, Brådvik et al. 1991, Breitenstein 1998, Cancelliere and Kertesz 1990, Pell 1996).

Concluding remarks

Summary of findings

The review of the literature detailed above leads us to several main conclusions. First and foremost is that the search for the neural bases of prosody is not a simple or straightforward one. The complexity of this issue is illustrated by the multiplicity of acoustic parameters that signal prosody (some of which remain undetermined), their necessary integration in speech production and perception across affective and linguistic contexts and the difficulty of examining prosody in isolation from other aspects of communication. In addition, of course, are the complications of interpreting evidence from brain-damaged patients, of parcelling out the effects of associated deficits, and of determining what level of neural activation or involvement constitutes dominance or control over a function.

Despite all of the interacting factors, the findings gathered to date have taught us a great deal. In terms of both production and perception of prosody, research has provided consistent support for the functional lateralization hypothesis (Van Lancker 1980) at the phonemic and lexical levels. The left hemisphere has been shown to be active in the production and comprehension of tonal contrasts and lexical stress, with minimal evidence of right hemisphere involvement. Yet, when other levels of the language code are considered, the results become less clear-cut. Few would argue that the right hemisphere is not implicated in the processing of emotion in general—be it language-based or otherwise (but cf. Ivry and Robertson

1998 for a different perspective on hemispheric specialization). However, the question of whether emotional prosody, in particular, is lateralized to the right hemisphere has not yet been resolved. What is clear is that models proposing a strict parallelism between left hemisphere organization for language and right hemisphere organization for prosody (e.g. Ross 1981) are not well-substantiated by the available evidence.

Among the intriguing hypotheses that merit further investigation are theories that posit subcortical involvement in both the production and perception of prosody (e.g. Cancelliere and Kertesz 1990), the notion that the size or domain of the utterance may determine which cerebral hemisphere is invoked for prosodic processing (e.g. Behrens 1989) and the hypothesis that individual acoustic cues are independently lateralized (e.g. Van Lancker and Sidtis 1992). All of these theories have received at least modest experimental support and, in all likelihood, aspects of each will need to be incorporated into a comprehensive model of the neural bases of prosody.

Considerations for future research

In this section, we briefly consider some of the variables that should be taken into account in order for research in this area to progress beyond its current state.

One critical factor in any investigation is subject selection; this is all the more true in studies of pathological populations. Differences in subject characteristics across investigations have undoubtedly contributed significantly to many of the inconsistencies in findings that we have discussed. Variables such as lesion site, time post-onset and the presence of associated deficits (such as depression, behavioural neglect and dysarthria) must be carefully controlled. Stimulus characteristics represent another important factor. It is essential that linguistically well-motivated structures be examined in sufficient numbers and under controlled testing conditions to obtain an adequate data sample. Anecdotal clinical reports must be replaced with carefully-designed experimental studies.

There is currently a dearth of cross-linguistic data concerning the neural substrate for prosody. Future investigations should sample from a variety of languages in which prosody serves different functions. It will also be important to explore the integration of affective and linguistic aspects of prosody; the dichotomy between affective and linguistic prosody may be an artificial one in spontaneous language processing and efforts are only beginning to be made to examine their integration in normal speech processing (McRoberts *et al.* 1995, Pell 1998a, 1999a, b).

The components and functions of prosody are obviously complex. It is, therefore, not surprising that the neural substrate subserving prosody may be equally, if not more, complex. With the increasing availability of advanced neuroimaging technology and meticulously-designed experimental investigations, we are confident that significant advances in our understanding of the neural systems that underlie prosodic processing will soon emerge.

Acknowledgements

Preparation of this manuscript was supported by grants from the Medical Research Council of Canada and the Fonds de la Recherche en Santé du Québec to the first author. The second author was supported by a scholarship from the Fonds de la Recherche en Santé du Québec. We gratefully acknowledge the helpful comments of Dr. Carol Leonard and Dr. William Katz.

Note

¹ There is a large literature on the lateralization of pitch in non-speech domains, generally supporting right hemisphere involvement in discrimination and memory for pitch (see e.g. Zatorre *et al.* 1994). Unfortunately, little research of this sort has been conducted to date on pitch processing in the speech domain.

References

- BAUM, S. 1992, The influence of word length on syllable duration in aphasia: acoustic analyses. Aphasiology, 6, 501-513.
- BAUM, S. 1998, The role of fundamental frequency and duration in the perception of linguistic stress by individuals with brain damage. *Journal of Speech, Language, & Hearing Research,* 41, 31–40.
- BAUM, S., KELSCH DANILOFF, J., DANILOFF, R. and LEWIS, J. 1982, Sentence comprehension by Broca's aphasics: effects of some suprasegmental variables. *Brain and Language*, 17, 261–271.
- BAUM, S. and PELL, M. 1997, Production of affective and linguistic prosody by brain-damaged patients. Aphasiology, 11, 177–198.
- BAUM, S., PELL, M., LEONARD, C. and GORDON, J. 1997, The ability of right- and left-hemispheredamaged individuals to produce and interpret prosodic cues marking phrasal boundaries. *Language & Speech*, 40, 313–330.
- BEHRENS, S. 1985, The perception of stress and lateralization of prosody. Brain and Language, 26, 332-348.
- BEHRENS, S. 1988, The role of the right hemisphere in the production of linguistic stress. *Brain and Language*, **33**, 104–127.
- BEHRENS, S. 1989, Characterizing sentence intonation in a right hemisphere-damaged population. Brain and Language, 37, 181-200.
- BLONDER, L., BOWERS, D. and HEILMAN, K. 1991, The role of the right hemisphere in emotional communication. *Brain*, 114, 1115–1127.
- BLONDER, L. X., GUR, R. E. and GUR, R. C. 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. and BUTLER, S. M. 1995, Prosodic characteristics of speech pre- and post-right hemisphere stroke. *Brain and Language*, **51**, 318–335.
- BLUMSTEIN, S. 1991, Phonological aspects of aphasia. In Sarno (Ed.) *Acquired Aphasia* (San Diego, CA: Academic Press, Inc.).
- BLUMSTEIN, S. and BAUM, S. 1987, Consonant production deficits in aphasia. In J. Ryalls (Ed.) *Phonetic Approaches to Speech Production in Aphasia and Related Disorders* (Boston, MA: College-Hill Press), pp. 3–22.
- BLUMSTEIN, S. and COOPER, W. 1974, Hemispheric processing of intonation contours. *Cortex*, 10, 146–158.
- BLUMSTEIN, S. and GOODGLASS, H. 1972, The perception of stress as a semantic cue in aphasia. *Journal* of Speech and Hearing Research, 15, 800–806.
- BOROD, J. 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., WELKOWITZ, J., ALPERT, M., BROZGOLD, A. Z., MARTIN, C., PESELOW, E. and DILLER, L. 1990, Parameters of emotional processing in neuropsychiatric disorders: Conceptual issues and a battery of tests. *Journal of Communication Disorders*, 23, 247–271.
- BOWERS, D., COSLETT, H., BAUER, R., SPEEDIE, L. and HEILMAN, K. 1987, Comprehension of emotional prosody following unilateral hemispheric lesions: Processing defect versus distraction defect. *Neuropsychologia*, 25, 317–328.

- BRÅDVIK, B., DRAVINS, C., HOLTÅS, S., ROSÉN, I., RYDING, E. and INGVAR, D. 1990, Do single right hemisphere infarcts or transient ischaemic attacks result in aprosody? *Acta Neurologica Scandinavica*, 81, 61–70.
- BRÅDVIK, B., DRAVINS, C., HOLTÅS, S., ROSÉN, I., RYDING, E. and INGVAR, D. 1991, Disturbances of speech prosody following right hemisphere infarcts. Acta Neurologica Scandinavica, 84, 114–126.
- BREITENSTEIN, C., DAUM, I. and ACKERMANN, H. 1998, Emotional processing following cortical and subcortical brain damage: contribution of the fronto-striatal circuitry. *Behavioural Neurology*, 11, 29–42.
- BRYAN, K. 1989, Language prosody and the right hemisphere. Aphasiology, 3, 285-299.
- CANCELLIERE, A. and HAUSDORF, P. 1988, Emotional expression in Huntington's disease. Journal of Clinical and Experimental Neuropsychology, 10, 62.
- CANCELLIERE, A. and KERTESZ, A. 1990, Lesion localization in acquired deficits of emotional expression and comprehension. *Brain and Cognition*, 13, 133–147.
- CARMON, A. and NACHSHON, I. 1971, Effect of unilateral brain damage on perception of temporal order. *Cortex* 7, 410–418.
- CHERTKOW, H. and BUB, D. 1994, Functional activation and cognition: the ¹⁵O subtraction method. In A. Kertesz (Ed.), *Localization and Neuroimaging in Neuropsychology* (San Diego, CA: Academic Press).
- COLSHER, P. L., COOPER, W. E. and GRAFF-RADFORD, N. 1987, Intonational variability in the speech of right-hemisphere damaged patients. *Brain and Language*, **32**, 379–383.
- COOPER, W., SOARES, C., NICOL, J., MICHELOW, D. and GOLOSKIE, S. 1984, Clausal intonation after unilateral brain damage. *Language and Speech*, **27**, 17–24.
- COOPER, W. and SORENSEN, J. 1981, Fundamental frequency in sentence production (New York: Springer Verlag).
- CRYSTAL, D. 1969, Prosodic systems and intonation in English (Cambridge: Cambridge University Press).
- DANLY, M., COOPER, W. E. and SHAPIRO, B. 1983, Fundamental frequency, language processing, and linguistic structure in Wernicke's aphasia. *Brain and Language*, **19**, 1–24.
- DANLY, M. and SHAPIRO, B. E. 1982, Speech prosody in Broca's aphasia. Brain and Language, 16, 171–190.
- DARBY, D. 1993, Sensory aprosodia: a clinical clue to lesion of the inferior division of the right middle cerebral artery? *Neurology*, 43, 567–572.
- DRONKERS, N., REDFERN, B. and SHAPIRO, J. 1992, The third left frontal convolution plays no special role in the function of language: Marie's quadrilateral space revisited. Paper presented at *Academy of Aphasia*, Toronto, ON.
- DYKSTRA, K., GANDOUR, J. and STARK, R. E. 1995, Disruption of prosody after frontal lobe seizures in the nondominant hemisphere. *Aphasiology*, **9**, 453–476.
- EDMONDSON, J., CHAN, J.-L., SEIBERT, G. and ROSS, E. 1987, The effect of right-brain damage on acoustical measures of affective prosody in Taiwanese patients. *Journal of Phonetics*, **15**, 219–233.
- EHLERS, L. and DALBY, M. 1987, Appreciation of emotional expressions in the visual and auditory modality in normal and brain-damaged patients. Acta Neurologica Scandinavica, 76, 251–256.
- EMMOREY, K. 1987, The neurological substrates for prosodic aspects of speech. Brain and Language, 30, 305–320.
- FUSTER, J. M. 1989, The Prefrontal Cortex: Anatomy, Physiology, and Neuropsychology of the Frontal Lobe (New York: Raven Press).
- GANDOUR, J. and DARDARANANDA, R. 1983, Identification of tonal contrasts in Thai aphasic patients. Brain and Language, 18, 98–114.
- GANDOUR, J., LARSEN, J., DECHONGKIT, S., PONGLORPISIT, S. and KHUNADORN, F. 1995, Speech prosody in affective contexts in Thai patients with right hemisphere lesions. *Brain and Language*, 51, 422–443.
- GANDOUR, J., PONGLORPISIT, S., DECHONGKIT, S., KHUNADORN, F., BOONGIRD, P. and POTISUK, S. 1993, Anticipatory tonal coarticulation in Thai noun compounds after unilateral brain damage. *Brain and Language*, 45, 1–20.
- GANDOUR, J., PONGLORPISIT, S., KHUNADORN, F., DECHONGKIT, S., BOONGIRD, P., BOONKLAM, R. and POTISUK, S. 1992, Lexical tones in Thai after unilateral brain damage. *Brain and Language*, 43, 275–307.
- GANDOUR, J., WONG, D., VAN LANCKER, D. and HUTCHINS, G. 1997, A PET investigation of speech prosody in tone languages. *Brain and Language*, 60, 192–194.

- GARRETT, M. 1980, Levels of processing in sentence production. In B. Butterworth (Ed.) Language Production (London: Academic Press).
- GEORGE, M., PAREKH, P., ROSINSKY, N., KETTER, T., KIMBRELL, T., HEILMAN, K., HERSCOVITCH, P. and POST, R. 1996, Understanding emotional prosody activates right hemisphere regions. *Archives of Neurology*, 53, 665–670.
- GESCHWIND, N. 1984, Neural mechanisms, aphasia, and theories of language. In D. Caplan, A. R. Lecours and A. Smith (Eds) *Biological perspectives on language* (Cambridge, MA: MIT Press), pp. 31–39.
- GOODGLASS, H. and KAPLAN, E. 1983, *The assessment of aphasia and related disorders* (Philadelphia, PA: Lea and Febiger).
- GORELICK, P. and Ross, E. 1987, The aprosodias: further functional-anatomical evidence for the organisation of affective language in the right hemisphere. *Journal of Neurology, Neurosurgery, and Psychiatry*, **50**, 553–560.
- HEILMAN, K., BOWERS, D., SPEEDIE, L. and COSLETT, H. 1984, Comprehension of affective and nonaffective prosody. *Neurology*, 34, 917–920.
- HEILMAN, K. M., SCHOLES, R. and WATSON, R. T. 1975, Auditory affective agnosia: disturbed comprehension of affective speech. Journal of Neurology, Neurosurgery, and Psychiatry, 38, 69–72.
- HUGHES, C., CHAN, J.-L. and SU, M. 1983, Aprosodia in Chinese patients with right cerebral hemisphere lesions. Archives of Neurology, 40, 732-736.
- HUGHLINGS JACKSON, J. 1915, On affections of speech from diseases of the brain. *Brain*, **38**, 101–186. IVRY, R. and ROBERTSON, L. 1998, *The two sides of perception* (Cambridge, MA: MIT Press).
- KERTESZ, A. 1994, Localization and neuroimaging in neuropsychology (San Diego, CA: Academic Press).
- KIMURA, D. 1961, Cerebral dominance and the perception of verbal stimuli. Canadian Journal of Psychology, 15, 166-171.
- KLOUDA, G., ROBIN, D., GRAFF-RADFORD, N. and COOPER, W. 1988, The role of callosal connections in speech prosody. *Brain and Language*, **35**, 154–171.
- LEBRUN, Y., LESSINNES, A., DE VRESSE, L. and LELEUX, C. 1985, Dysprosody and the non-dominant hemisphere. *Language Science*, 7, 41–52.
- LEHISTE, I. 1970, Suprasegmentals (Cambridge, MA: MIT Press).
- LEVELT, W. 1989, Speaking: From Intention to Articulation (Cambridge, MA: MIT Press).
- LEY, R. G. and BRYDEN, M. P. 1982, A dissociation of right and left hemispheric effects for recognizing emotional tone and verbal content. *Brain and Cognition*, 1, 3–9.
- LIBERMAN, M. and PRINCE, A. 1977, On stress and linguistic rhythm. Linguistic Inquiry, 8, 249-336.
- MCROBERTS, G., STUDDERT-KENNEDY, M. and SHANKWEILER, D. 1995, The role of fundamental frequency in signaling linguistic stress and affect: evidence for a dissociation. *Perception and Psychophysics*, **57**, 159–174.
- MESULAM, M. 1985, Patterns in behavioral neuroanatomy: association areas, the limbic system, and hemispheric specialization. In M. Mesulam (ed.) *Principles of behavioral neurology* (New York: F. A. Davis).
- OUELLETTE, G. and BAUM, S. 1994, Acoustic analysis of prosodic cues in left and right-hemispheredamaged patients. *Aphasiology*, **8**, 257–283.
- PACKARD, J. 1986, Tone production deficits in nonfluent aphasic Chinese speech. *Brain and Language*, **29**, 212–223.
- PELL, M. 1996, On the receptive prosodic loss in Parkinson's disease. Cortex 32, 693-704.
- PELL, M. 1999a, Fundamental frequency encoding of linguistic and emotional prosody by right hemisphere damaged speakers. *Brain and Language*, submitted.
- PELL, M. 1999b, The temporal organization of affective and non-affective speech in patients with right-hemisphere infarcts. *Cortex*, **35**, 163–182.
- PELL, M. 1998a, Recognition of prosody following unilateral brain lesion: influence of functional and structural attributes of prosodic contours. *Neuropsychologia*, 36, 701–715.
- PELL, M. 1998b, Some acoustic correlates of perceptually 'flat affect' in right-hemisphere-damaged speakers. *Brain and Cognition*, in press.
- PELL, M. and BAUM, S. 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. and BAUM, S. 1997b, Unilateral brain damage, prosodic comprehension deficits, and the acoustic cues to prosody. *Brain and Language*, **57**, 195–214.
- PETERSEN, S. E., FOX, P. T., SNYDER, A. Z. and RAICHLE, M. E. 1990, Activation of extrastriate and frontal cortical areas by visual words and word-like stimuli. *Science*, **249**, 1041–1044.

- RINN, W. E. 1984, The neuropsychology of facial expression: a review of the neurological and psychological mechanisms for producing facial expressions. *Psychology Bulletin*, 95, 52–77.
- ROBIN, D. A., TRANEL, D. and 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.
- Ross, E. 1981, The aprosodias: functional-anatomic organization of the affective components of language in the right hemisphere. *Archives of Neurology*, **38**, 561–569.
- Ross, E. 1993, Nonverbal aspects of language. Behavioral Neurology, 11, 9-22.
- Ross, E., EDMONDSON, J. and SEIBERT, G. 1986, The effect of affect on various acoustic measures of prosody in tone and non-tone languages: a comparison based on computer analysis of voice. *Journal of Phonetics*, 14, 283–302.
- Ross, E., EDMONDSON, J., SEIBERT, G. and HOMAN, W. 1988, Acoustic analysis of affective prosody during right-sided Wada test: A within-subjects verification of the right hemisphere's role in language. *Brain and Language*, **33**, 128–145.
- Ross, E., HARNEY, J. H., DELACOSTE-UTAMSING, C. and PURDY, P. D. 1981, How the brain integrates affective and propositional language into a unified behavioral function. *Archives of Neurology*, **38**, 745–748.
- Ross, E. and MESULAM, M.-M. 1979, Dominant language functions of the right hemisphere?: Prosody and emotional gesturing. *Archives of Neurology*, **36**, 144–149.
- Ross, E., THOMPSON, R. and 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. 1982, Intonation in Broca's aphasia. Neuropsychologia, 20, 355-360.
- RYALLS, J., JOANETTE, Y. and FELDMAN, L. 1987, An acoustic comparison of normal and righthemisphere-damaged speech prosody. *Cortex*, 23, 685–694.
- RYALLS, J. and REINVANG, I. 1986, Functional lateralization of linguistic tones: acoustic evidence from Norwegian. *Language and Speech*, **29**, 389–398.
- SCHLANGER, B., SCHLANGER, P. and GERSTMAN, L. 1976, The perception of emotionally toned sentences by right hemisphere-damaged and aphasic subjects. *Brain and Language*, 3, 396–403.
- SCOTT, S., CAIRD, F. and WILLIAMS, B. 1984, Evidence for an apparent sensory speech disorder in Parkinson's disease. *Journal of Neurology, Neurosurgery, and Psychiatry*, 47, 840–843.
- SELKIRK, E. 1984, *Phonology and Syntax: The Relation Between Sound and Structure* (Cambridge, MA: MIT Press).
- SERON, X., VAN DER KAA, M.-A., VANDERLINDEN, M., REMITS, A. and FEYEREISEN, P. 1982, Decoding paralinguistic signals: effect of semantic and prosodic cues on aphasics' comprehension. *Journal of Communication Disorders*, 15, 223–231.
- SHAPIRO, B. and 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.
- SIDTIS, J. and FELDMANN, E. 1990, Transient ischemic attacks presenting with a loss of pitch perception. *Cortex*, 26, 469–471.
- SPEEDIE, L., BRAKE, N., FOLSTEIN, S., BOWERS, D. and HEILMAN, K. 1990, Comprehension of prosody in Huntington's disease. Journal of Neurology, Neurosurgery and Psychiatry, 53, 607–610.
- STARKSTEIN, S., FEDEROFF, J., PRICE, T., LEIGUARDA, R. and ROBINSON, R. 1994, Neuropsychological and neuroradiologic correlates of emotional prosody comprehension. *Neurology*, 44, 515–522.
- TOMPKINS, C. A. and FLOWERS, C. R. 1985, Perception of emotional intonation by brain-damaged adults: the influence of task processing levels. *Journal of Speech and Hearing Research*, 28, 527–538.
- TUCKER, D., WATSON, R. and HEILMAN, K. 1977, Discrimination and evocation of affectively intoned speech in patients with right parietal disease. *Neurology*, **27**, 947–950.
- TWIST, D., SQUIRES, N., SPIELHOLZ, N. and SILVERGLIDE, R. 1991, Event-related potentials in disorders of prosodic and semantic linguistic processing. *Neuropsychiatry*, *Neurosurgery*, and *Behavioral Neurology*, 4, 281–304.
- VAN LANCKER, D. 1980, Cerebral lateralization of pitch cues in the linguistic signal. International Journal of Human Communication, 13, 227–277.
- VAN LANCKER, D. and FROMKIN, V. 1973, Hemispheric specialization for pitch and 'tone': evidence from Thai. *Journal of Phonetics*, 1, 101–109.
- VAN LANCKER, D. and 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.

- WEINTRAUB, S., MESULAM, M.-M. and KRAMER, L. 1981, Disturbances in prosody: A righthemisphere contribution to language. *Archives of Neurology*, 38, 742–744.
- ZATORRE, R. 1988, Pitch perception of complex tones and human temporal-lobe function. *Journal of the Acoustical Society of America*, 84, 566–572.
- ZATORRE, R., EVANS, A. and MEYER, E. 1994, Neural mechanisms underlying melodic perception and memory for pitch. *Journal of Neuroscience*, 14, 1908–1919.
- ZATORRE, R., EVANS, A., MEYER, E. and GJEDDE, A. 1992, Lateralization of phonetic and pitch discrimination in speech processing. *Science*, **256**, 846–849.
- ZURIF, E. and MENDELSOHN, M. 1972, Hemispheric specialization for the perception of speech sounds: The influence of intonation and structure. *Perception & Psychophysics*, 11, 329–332.