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Dissociating the performance of cortical and subcortical patients on phonemic tasks

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Abstract

To assess cortical and subcortical contributions to phonemic processing, patients with left frontal, temporal–parietal, or cerebellar lesions as well as those with Parkinson's disease were tested on phonemic identification and production tasks. In Experiment 1, patients and controls were asked to identify syllables on both a voicing and place of articulation continuum. Subcortical patients were relatively unimpaired at this task whereas cortical patients were less accurate at identifying the endpoints of both continua and exhibited little evidence of categorical perception. For Experiment 2, controls and patients were asked to produce syllables. Subcortical patients were able to produce contrastive voice onset times (VOTs) for voicing cognates although VOT of the voiceless phoneme was more variable for cerebellar patients. Cortical patients showed greater overlap in the production of both VOT and formant transition intervals. These results are discussed in terms of the type of computations hypothesized to originate from each neural area.

1. Introduction

Impairments of phonemic processing are associated with a variety of neurological conditions ranging from left frontal and temporal–parietal lobe lesions (Basso, Casati, & Vignolo, 1977; Baum, Blumstein, Naeser, & Palumbo, 1990; Blumstein, Baker, & Goodglass, 1977a, 1980, 1977b; Gandour & Dardarananda, 1984) to cerebellar (Ackermann & Hertrich, 1997; Gandour & Dardarananda, 1984; Ivry & Gopal, 1993) and basal ganglia pathology (Forrest, Weismer, & Turner, 1989; Lieberman et al., 1992). Dissociating the contributions of each of these neural areas to phonemic processing has been difficult as deficits are manifest for each of these groups on similar phonemic tasks. For instance, impairments in the production of voice onset time have been linked to both subcortical and cortical damage (see Ravizza, 2001; for a review). Part of the difficulty in establishing the specific phonemic operations provided by each neural area may lie in the fact that cortical and subcortical groups have seldom been compared in the same experiment. One aim of the research reported here is to provide a direct comparison of the performance on phonemic production and perception tasks between patients with cortical and subcortical damage.

The diversity of neurological areas associated with phonemic deficits seems paradoxical to modular accounts of language. It is unparsimonious to suggest that each of these areas provides the same computations to phonemic tasks. More likely, these tasks consist of a number of subprocesses that are instantiated in different neural areas, and that the type of breakdown in phonemic perception and production will reflect disruption of computations associated with these areas. In general, contributions to phonemic processing from subcortical areas are thought to be non-linguistic in nature whereas computations provided by cortical areas in the left frontal and temporal–parietal areas are claimed to be speech-specific (Darley, Aronson, & Brown, 1969; Duffy, 1995).

In terms of phonemic production, subcortical areas such as the cerebellum and basal ganglia are thought to aid in the execution of speech by providing accurate rate, timing and intensity parameters to motor structures. Similar to the role they play in the coordination of fine movements, the basal ganglia and cerebellum are believed to be important in the precise timing and

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modulation of articulation. Patients with subcortical damage who display impairments of phonemic production are classified as dysarthric—a collective name for all speech disorders that are due to disturbances in muscular control (Darley et al., 1969). In contrast, cortical damage is thought to impair phonemic production because of deficits in motor planning or selection that are specific to speech (Blumstein, 1995). Unlike subcortical patients, those with speech deficits due to cortical damage are not necessarily impaired at producing nonlinguistic movements of the oral musculature (Duffy, 1995).

Despite differences in the type of motoric or linguistic processing thought to be disturbed, similar phonemic production deficits have been reported following lesions to both subcortical and cortical areas (Ackermann & Hertrich, 1997; Baum et al., 1990; Blumstein et al., 1980; Forrest et al., 1989; Gandour & Dardarananda, 1984; Ivry & Gopal, 1993; Lieberman et al., 1992). Although rarely tested directly, performance on production tasks may vary depending on whether impairments are motoric or linguistic. For example, one dissociation between cortical and subcortical patients is suggested by research exploring VOT production. When producing a stop consonant, the interval between the release of the burst and the onset of vocal cord vibration needs to be timed appropriately for that phoneme—shorter for voiced phonemes like /b/ and longer for voiceless sounds such as /p/. Subcortical patients tend to show selective deficits in producing consistently timed movements (Ackermann & Hertrich, 1997; Ivry & Gopal, 1993) or initiating speech (Forrest et al., 1989) while still being able to maintain relatively distinguishable phonological categories In contrast, cortical patients tend to produce less distinctive phonemic contrasts as well as being more variable in their productions (Baum et al., 1990; Blumstein et al., 1980, 1977b; Gandour & Dardarananda, 1984). However, the performance of cortical and subcortical groups has not been compared in a single experiment, making it difficult to know how these groups perform in relation to each other.

Cortical patients also exhibit impairments in the discrimination and/or identification of phonemes including voicing and place of articulation contrasts (Basso et al., 1977; Blumstein et al., 1977a, 1977b). However, perceptual difficulties are not restricted to cortical lesions. Cerebellar patients have also displayed deficits of phonological perception although their difficulty appears to be limited to phonemic contrasts that vary only in temporal characteristics (Ackermann, Graber, Hertrich, & Daum, 1997). For phonemic contrasts that vary in spectral parameters, cerebellar patients appear unimpaired (Ackermann et al., 1997; Ivry & Gopal, 1993) This is in accord with studies showing impairments for cerebellar patients in many tasks that require either the perception or production of precisely-timed intervals (Ivry & Keele, 1989). No studies have been conducted investigating the role of the basal ganglia in phonological perception, although it is doubtful that their cognitive and motor impairments would impact speech perception. Evaluating the performance of both cortical and subcortical patients on the same perceptual task would be instructive in identifying the effects of brain damage in general in comparison to the effects of damage to purely linguistic structures.

It is important to identify the contributions to phonemic processing from neural structures that are dedicated to linguistic computations compared to those that are involved in a wide range of motor and cognitive tasks. With such knowledge, the process of how phonemes are perceived and produced may become clearer. Toward this end, the experiments reported here focus on dissociating the performance on phonemic tasks of subcortical and cortical patients rather than emphasizing the differences within those groups. Patients with subcortical damage included those with focal lesions or atrophy of the cerebellum and those with basal ganglia pathology due to Parkinson's disease. The cortical group consisted of patients with damage to left frontal and left temporal–parietal speech centers.

It may be argued that the type of phonemic processing impairment within cortical and subcortical groups is substantively different and not amenable to generalization. For example, left frontal areas are conjectured to be more involved in motor speech planning than in phonological selection, and often patients with anterior damage display greater impairments on phonemic production tasks than temporal–parietal patients (Blumstein, 1995; Ravizza, 2001). However, motor planning and selection deficits have been reported for both frontal and temporal–parietal patients (Blumstein et al., 1980), and the distinction between the two cortical groups in both production and perception tasks has not been firmly established (Blumstein, 1995, 2000). Studies directly comparing patients with cerebellar damage and Parkinson's disease are more rare, but one study of the production of vowel length suggests that these patients performed similarly although cerebellar patients seem to be impaired to a greater degree (Ackermann, Graber, Hertrich, & Daum, 1999). Given the lack of distinctive patterns of phonemic deficits within either the cortical or subcortical group, it may be more beneficial to compare performance on phonemic tasks across these groups.

2. Experiment 1

The perceptual abilities of cortical and subcortical patients were examined in this experiment. Participants were asked to identify consonant-vowel syllables whose initial phonemes contrasted in either place of articulation or voicing. In accordance with other research

reporting deficits in phonemic identification tasks for frontal and temporal–parietal patients (Blumstein et al., 1977b), it is predicted that both cortical groups will be impaired at this task while the subcortical patients should be relatively unimpaired. Although cerebellar patients have shown perceptual deficits with temporal contrasts, the voicing and place of articulation contrasts employed here contain both spectral and temporal cues, and so these patients should not be impaired (Ackermann et al., 1997; Ivry & Gopal, 1993).

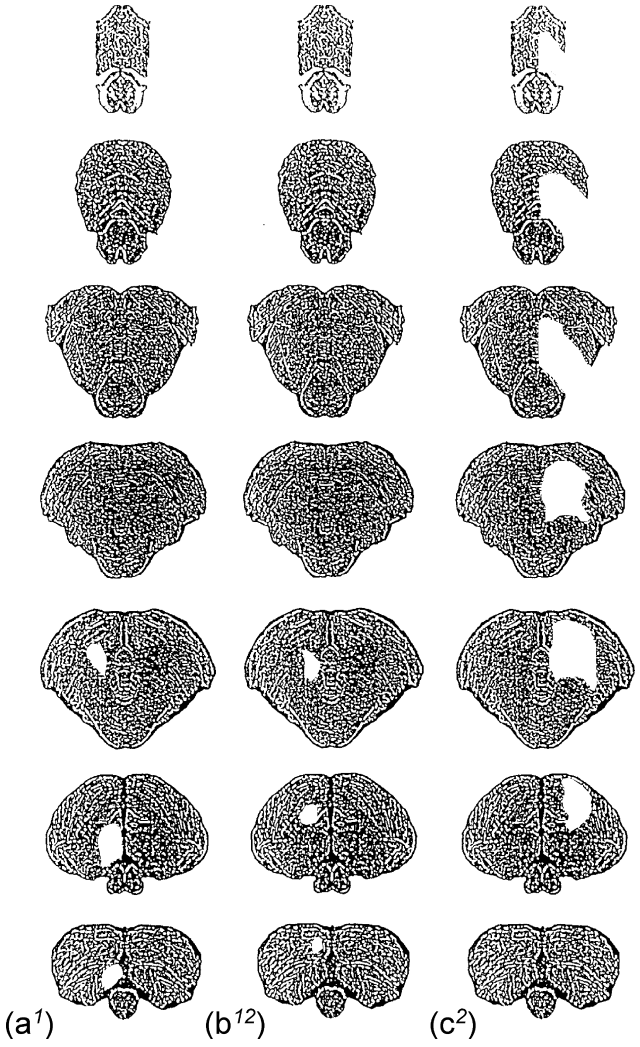

Fig. 1. Site and extent of focal lesions for cerebellar patients in Experiments 1 and 2. Subscripts indicate the experiment in which the patient participated.

2.1. Methods

2.1.1. Participants

Ten patients with subcortical damage, nine patients with lesions to the cortex, and eight age-matched controls gave their consent and were paid to participate in this experiment. Of the patients with subcortical damage, five were diagnosed with idiopathic Parkinson's disease (mean age = 68 years) while the rest (mean age = 64) exhibited atrophy ($n = 3$) or left-hemispheric focal lesions ($n = 2$) to the cerebellum (see Fig. 1 for cerebellar focal lesions). Five of the cortical patients had suffered damage to left frontal speech areas (mean age = 63) and four had lesions of the temporal–parietal language areas (mean age = 72; see Fig. 2 and Table 1). The scan of one anterior aphasic was unavailable for publication.

2.1.2. Stimuli

Syllables were created using SenSyn—a software package based on the Klatt synthesizer (Klatt, 1982). One continuum consisted of syllables that differed in place (/ba/-/da/) while a second continuum was comprised of syllables differing in voicing (/ba/-/pa/). Each continuum consisted of nine tokens. For the place continuum, the onset of the second and third formants of the /ba/ endpoint increased 100 Hz. for each successive token (/b/ endpoint: $F2 = 900$ Hz, $F3 = 1900$ Hz; /d/ endpoint: $F2 = 1700$ Hz, $F3 = 2700$ Hz), and then either linearly ascended or descended to a steady-state frequency in the first 40 ms (/ah/: $F2 = 1250$, $F3 = 2350$). To create the voicing continuum, the voice onset time

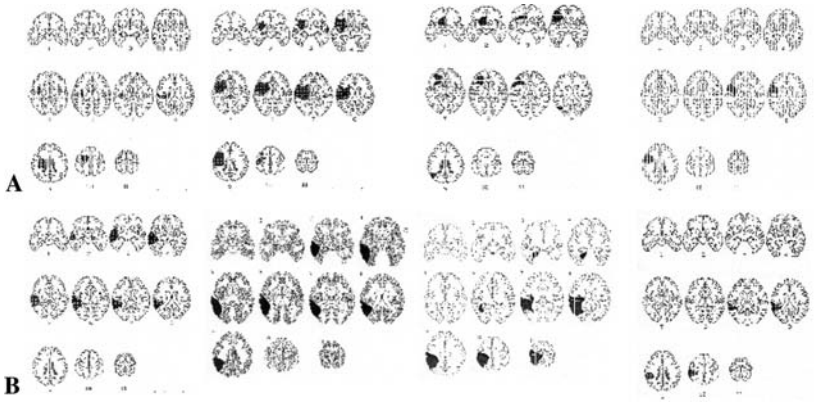

Fig. 2. Site and extent of focal lesions for (a) frontal and (b) temporal–parietal subjects tested in Experiments 1 and 2.

Table 1
Categorization of aphasic type and aphasia scores on the Western aphasia battery for each cortical patient

Patient	Experiment	Lesion site	Aphasia type	WAB score
1	1 and 2	Frontal	Anomic	96
2	1 and 2	Frontal	Unclassified	68.3
3	1 and 2	Frontal	Anomic	92.6
4	1 and 2	Frontal	Anomic	93.8
5	1 and 2	Frontal	Anomic	98.8
6	1 and 2	Temp–Par.	Wernicke	97.1
7	1 and 2	Temp–Par.	Unclassified	92.9
8	1 and 2	Temp–Par.	Conduction	77.8
9	1	Temp–Par.	Wernicke	51.5

(VOT) for each token was progressively raised by 10 ms starting with −10 ms of prevoicing for the endpoint /ba/ and ending with a VOT of 70 ms for the endpoint /pa/. All syllables were 500 ms in duration and were preceded by 300 ms of silence.

2.1.3. Procedure

All sounds were presented via a 16-bit sound card through headphones. Participants were first given a hearing test using tones of 500-, 1000-, and 2000-Hz, and any participant whose threshold was above 30 db for any of these tones was excluded from the study.

Before performing the phoneme perception task, participants listened to a demonstration of the two endpoints of the continuum for which they were about to be tested. Each continuum was tested in separated blocks of trials, and the order of presentation of the two continua was counterbalanced across participants. In each test block, a token from the continuum was presented and participants were asked to identify the syllable they heard. Depending on the severity of motor symptoms, participants responded either by pointing to one of the two syllables written on separate pieces of paper or by pressing the "z" and "/" keys. For participants making their own keypresses, pieces of paper with these syllables were placed on the appropriate sides of the keyboard to remind them of the key designations. The side that each syllable was assigned to was counterbalanced across participants. However, /ba/ was always assigned to the same key across the two continua for a given participant.

Each token was presented ten times at random within a block, making a total of 90 trials per block. Participants were tested on 2–3 blocks of each continuum depending on time constraints.

2.1.4. Data analysis

The computer recorded the number of times the participant identified each token as "ba." The dividing point between predominantly "ba" responses and predominantly "pa" or "da" responses was determined for each block of trials. The dividing point was defined as the first token after the /ba/ endpoint where the number of "ba" responses fell below 5. If the dividing point was different between blocks, the responses to each token were adjusted so that the point of division was the same. For example, if the dividing point fell between tokens 2 and 3 in one block and 3 and 4 in the next block, the response functions were aligned so that the dividing point was stable over each block of trials. The responses of the first block would be assigned to tokens 2–10 and the second block to tokens 1–9. In such a case, the number of "ba" responses to token 1 in the first block would be the same as token 2 and, for the second block, responses to tokens 9 and 10 would be identical. The percentage of /ba/ responses for each token was then calculated for each participant. This process of lining up crossover points was repeated when comparing the percentage of "ba" responses across participants within each experimental group. Thus, the voicing continuum was artificially expanded to contain 11 tokens and the place continuum was altered to include 13. Note that this procedure does not affect the analysis of endpoint performance, and will only allow for better examination of the steepness of the change in perception from one phoneme to the other.

2.2. Results and discussion

2.2.1. Voicing continuum

The percentage of "ba" responses to the endpoints of the voicing continuum was analyzed using one-way group (control, Parkinson, and cerebellar) ANOVAs to contrast the performance of each subcortical groups to the control group. The same test was conducted comparing both cortical groups with the control group. As can be seen in Fig. 3, both subcortical groups performed very similarly to controls ($ps > .1$). In contrast, patients with cortical damage were less accurate than controls at identifying both endpoints although the difference was not significant (/ba/—$F(2,14) = 3.01$, $p = .082$; /pa/—$F(2,14) = 2.04$, $p = .167$). The cortical group exhibited less evidence of categorical perception ($F(2,14) = 3.9$, $p < .05$). Independent t tests confirmed that the percentage change in "ba" responses at the crossover point was smaller than controls for both anterior ($t(11) = 2.31$, $p < .05$) and posterior aphasics ($t(10) = 2.3$, $p < .05$). Subcortical

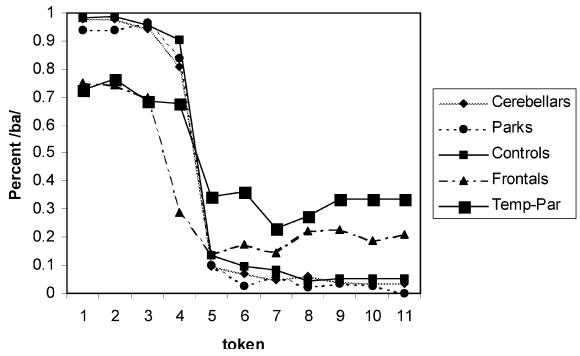

Fig. 3. Proportion of "ba" responses for each patient group on the voicing continuum.

patients, however, switched dramatically in their categorization of the phonemes and appear unimpaired at this task.

2.2.2. Place continuum

The same analyses that were used to investigate differences in the perception of the voicing continuum were also used here. Again, subcortical patients had no difficulty with the identification task. As can be seen in Fig. 4, both cerebellar and Parkinson patients performed similarly to controls at identifying the endpoints and they exhibited a steep division in the perception of /ba/ and /da/. In comparing the cortical patients to the controls, a group difference was obtained for performance on both endpoints (/ba/—$F(2,14) = 4.72$, $p < .05$; /da/—$F(2,14) = 8.82$, $p < .01$) and the steepness of the categorical function ($F(2,14) = 10.31$, $p < .05$). Post-hoc t tests showed that the difference in endpoint performance was significant only for those with frontal damage (/ba/: $t(5) = 3.04$, $p < .05$; /da/: $t(11) = 5.66$, $p < .01$), although both aphasic groups exhibited a more gradual switch between phonemes (frontals: $t(11) = 5.44$, $p < .01$; temporal–parietals: $t(10) = 3.1$, $p < .05$).

2.3. Conclusions

Although cerebellar patients' perceptual abilities have been tested in other studies (Ackermann et al., 1997; Ivry & Gopal, 1993), phonological perception has not been investigated for Parkinson patients. Interestingly, both groups of subcortical patients performed similarly to each other and to the control subjects. The intact performance of the cerebellar patients is in line with other research demonstrating proficiency at identifying phonemic contrasts (Ackermann et al., 1997; Ivry & Gopal, 1993). Indeed, Ackermann et al. (1997) demonstrated that cerebellar patients were only impaired at perceiving phonemic contrasts when their identification relied solely on temporal cues. As the voicing continuum used in this study contained both timing (VOT) and spectral (presence of aspiration) cues, cerebellar patients were unimpaired. Thus, the ability to establish distinct phonemic categories was intact for both subcortical groups. In spite of the differences in neural pathology between patients with cerebellar damage and Parkinson's disease, phonological perception was affected similarly in each group.

In accordance with much of the aphasic literature (Basso et al., 1977; Blumstein et al., 1977b; Itoh, Tatsumi, Sasanuma, & Fukusako, 1986), both cortical groups were impaired at identifying phonemes. Not only were they less accurate in identifying the endpoints, they performed poorly at all points of the continuum. Both aphasic groups exhibited difficulty in categorical perception while only the anterior patients were significantly impaired at identifying the endpoints. This may be due to the greater variability in performance across the temporal–parietal patients. Two of the four patients in this group performed similarly to controls on this task while the rest were severely impaired. In contrast, only one of the five anterior aphasics appeared to do as well as controls. Aphasics' performance on phonemic perceptual tasks is often variable with anterior aphasics outperforming posterior aphasics in some studies but not in others (Blumstein, 2000). However, the cortical patients performance on this task was quite different than that of the subcortical patients.

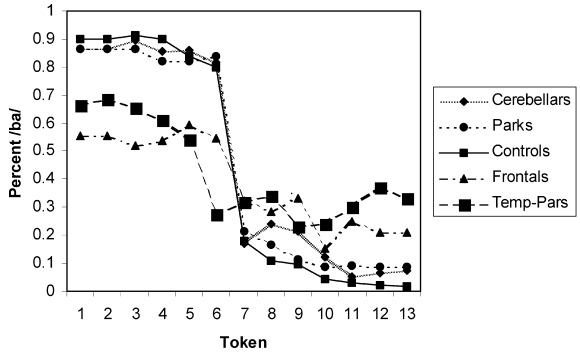

Fig. 4. Proportion of "ba" responses for each patient group on the place of articulation continuum.

3. Experiment 2

Production deficits were assessed by requiring participants to utter syllables that varied in terms of voicing and place of articulation. In general, mean values for two contrastive phonemes should be separable with non-overlapping distributions. Cortical patients who are hypothesized to have a linguistic deficit in articulatory implementation or phonological selection (Blumstein, 1990; Itoh et al., 1982) will be less able to produce phonemes that are contrastive. Due to the disruptions of phonemic representations, it is predicted that cortical patients will display impairments in both the mean and variance of temporal and spectral parameters that will result in overlapping phonemic categories. Given the

non-linguistic impairment claimed to underlie production deficits for subcortical patients, these patients are expected to demonstrate impairments only in the variability of production. On average, absolute values of temporal and spectral variables are predicted to be unimpaired such that distinct phonemic categories can be ascertained for these patients.

3.1. Methods

3.1.1. Participants

All of the patients with subcortical damage tested in Experiment 1 participated in this experiment with the addition of one new Parkinson and cerebellar patient (PD mean age = 65; cerebellar = 66). The cortical group also consisted of the same patients described in Experiment 1. One of the temporal–parietal patients (see Table 1) could not articulate any of the syllables correctly and so was dropped from the analysis. Six age-matched controls were also tested (mean age = 65). Patients and controls were paid for their participation.

3.1.2. Procedure

Participants were asked to read aloud a sentence presented to them on a computer screen in large type. The sentence consisted of a syllable embedded in the carrier phrase "That's a ____." The syllable set consisted of "ba," "pa," and "da," and each token was presented fifteen times at random. Participants were recorded on a cassette tape using a Sony Professional Walkman and a Crown unidimensional hands-free microphone.

3.1.3. Data analysis

The recordings were digitized at a rate of 10 kHz using the Computer Speech Lab (CSL) program from Kay Elemetrics. VOT was determined by measuring the interval from the burst to the onset of periodicity. This interval was measured by examining the spectrogram of the syllable for the absence of aspiration as well as determining the onset of periodicity in the wave signal.

The duration of formant transitions and their onset and offset frequencies were measured for the voiced, stop consonants /b/ and /d/. Impulse markers were first determined by the computer and corresponded to peaks in the fundamental frequency. These markers were placed both on the spectrogram and the wave signal to confirm that the computer was assigning them correctly. The first impulse marker was taken as the onset of the formant and the point at which the formant failed to change 20 Hz over 20 ms was established as the offset (Forrest et al., 1989). As the transition time of $F2$ is longer than $F1$ for the consonant /d/ (Kewley-Port, 1982), onset and offset points were determined by the second formant. In contrast, onsets and offsets were based on the transition of $F1$ for the consonant /b/ as $F1$ tended to be longer than $F2$. The interval between the onset of the consonant and the onset of the vowel portion of the syllable constituted the measure of formant transition duration. Frequency values for the onset and offset of the first two formant transitions were determined by the program using linear predictive coding. If the program was unable to determine the frequency value because of the weakness of the signal, no value was input for that sample. One cerebellar and one frontal patient did not have enough information to analyze $F2$ of /b/. In addition, both formants for /b/ and /d/ were too weak to be analyzed for one frontal and one cerebellar patient respectively.

3.2. Results and discussion

3.2.1. Voicing contrast

Average VOTs for the two patient groups were compared to those of the control group using independent-sample t tests. Given that VOT varies as a function of speech rate (Miller, Green, & Reeves, 1986; Summerfield, 1981), raw VOT values were not used in this comparison. Instead, the average VOT for each person was divided by the average duration for that syllable. All patient groups had shorter syllable durations than controls with the cerebellar patients speaking at the fastest rate (controls = 425 ms; cerebellars = 341 ms; PD = 370 ms; frontal = 388 ms, temp = 361 ms). This is surprising given that speech rate is usually found to be slower for cerebellar patients (Gandour & Dardarananda, 1984; Ivry & Gopal, 1993; Kent, Netsell, & Abbs, 1979). Although the subcortical groups tended to have shorter VOTs than controls for the voiceless syllable (see Fig. 5), a main effect of group was not obtained once the VOT scores were adjusted for overall speaking rate.

In contrast, VOTs for both voicing cognates tended to be longer for the cortical groups (/b/ - $F(2, 11) = 4.11$, $p < .05$; /p/ $F(2, 11) = 4.86$, $p < .05$). Independent t tests indicated that VOT for the voiceless syllable was significantly longer for temporal–parietal patients than controls and approached significance for frontal patients (temporal–parietal: $t(7) = 3.14$, $p < .05$; frontal: $t(9) = 1.91$, $p = .088$). For the voiced syllable, /b/, VOTs were marginally longer for frontal aphasics than

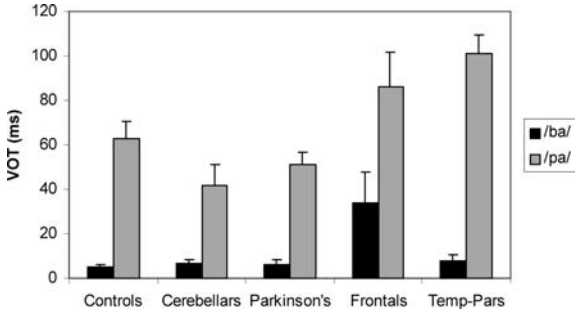

Fig. 5. VOT durations for /b/ and /p/.

Table 2
The range (mean ± one *SD*) of VOT productions in milliseconds in Experiment 2

	Controls	Cerebellars	Parkinson's	Frontals	Temp–Pars
/ba/	1.29	3.86	0.54	2.11	2.97
	8.43	9.85	11.44	65.27	12.59
/pa/	44.25	18.58	37.60	50.99	86.86
	81.58	64.60	64.96	121.18	115.16

controls ($t(4.22) = 2.33$, $p = .077$), but were normal for temporal–parietal patients.

Given that variance increases as segment duration lengthens (Crystal & House, 1988), variability in VOT production was examined by dividing each person's standard deviation by their average VOT duration. A one-way ANOVA indicated an effect of group on the variability of VOT production when comparing subcortical patients to controls ($F(2, 15) = 5.56, p < .05$). Of the subcortical groups, greater inconsistency of the voiceless VOT was displayed by the cerebellar patients ($t(10) = 3.59$, $p < .01$), while the Parkinson patients appeared unimpaired. Variability of both voicing cognates tended to be greater for cortical patients than controls (/b/—$F(2, 11) = 3.04$, $p = .089$; /p/ $F(2, 11) = 3.1$, $p = .086$).

Despite differences in pathology, cerebellar and Parkinson patients performed similarly when producing voicing contrasts. Average VOTs for both syllables were comparable to controls and the subcortical group was able to produce relatively non-overlapping VOT distributions (see Table 2). However, the cerebellar group did produce more inconsistent VOTs for the voiceless phoneme than controls. For cerebellar patients, this finding is in accord with other studies reporting impaired variability but not separability of VOTs (Gandour & Dardarananda, 1984; Ivry & Gopal, 1993) and reflects the non-linguistic nature of their speech deficits.

Studies of Parkinson patients are more rare, but two studies (Ackermann & Hertrich, 1997; Forrest et al., 1989) have reported that these patients can produce normal VOT durations except in the case of a sentence-initial syllables (Forrest et al., 1989). Given that the syllable in this experiment was the in the latter portion of the carrier phrase, the fact that the Parkinson patients produce normal VOTs is in line with previous results. Variability of VOT production has not been systematically measured in previous studies of Parkinson patients (Forrest et al., 1989; Lieberman et al., 1992), but the results of this experiment suggest that patients with basal ganglia damage can produce voiceless stop consonants consistently when not in sentence-initial position.

Cortical patients also performed as predicted. These patients had longer VOTs than controls and tended to be more variable as well. This result accords well with previous findings that VOT duration and variability are disrupted by cortical lesions (Baum et al., 1990, Blumstein et al., 1977a, 1980, 1977b). Moreover, VOTs falling within one standard deviation from the voiced mean fell within the equivalent range of the voiceless phoneme for the frontal patients although this was not true for the posterior patients (see Table 2). The difference in performance between frontal and temporal–parietal patients may reflect the greater involvement of the frontal areas to speech motor planning (Blumstein, 1990; Duffy, 1995). In contrast, posterior areas may be more involved in selecting the correct phoneme (Ravizza, 2001) rather than specifying how to articulate it.

3.2.2. *Place of articulation contrast—transition time*

Paraphasic errors of place (b ↔ d) were eliminated from the analysis and ranged from an average of 0–1.2 syllables per group. The number of place of articulation errors was not shown to differ by group. In line with research reporting formant transition durations for healthy adults (Kewley-Port, 1982), transition times were longer for the second formant of /d/ than the first formant of /b/ in the control group(see Fig. 6). The duration of the formant transitions for /b/ and /d/ was examined by dividing this interval by the overall duration of the syllable in order to account for differences in speaking rate.

In comparing the subcortical groups to the control group, the duration of the transition for /d/ was found to be marginally longer for subcortical patients ($F(2, 14) = 2.75$, $p = .099$) than the controls even when overall syllable duration was taken into account. The cortical groups displayed normal transition durations. Thus, all patients were relatively unimpaired at producing transitions of an appropriate length.

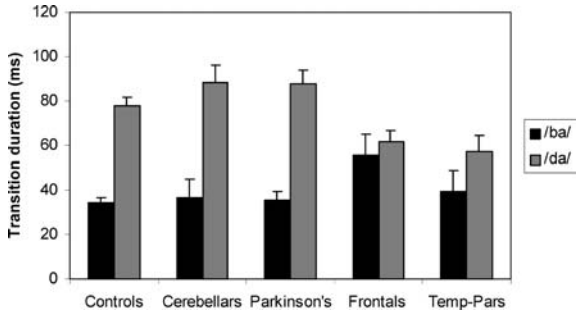

Fig. 6. Transition durations for /b/ and /d/.

As can be seen in Fig. 6, transition durations for /ba/ were about 30-40 ms shorter than for /da/ for both control subjects and subcortical patients. Cortical patients, however, produced transition durations that were much less contrastive for each syllable ($F(2,10) = 7.09$, $p < .05$). Independent-sample t tests confirmed that the difference in transition duration between /ba/ and /da/ was smaller for the frontal group ($t(8) = 4.64$, $p < .01$). No significant difference was obtained when comparing subcortical patients or posterior aphasics and controls.

As in the analysis of VOT, the standard deviation of the formant transition duration was divided by the duration itself. Although the variability of transition duration tended to be higher for subcortical patients, it was not unduly high given their longer transition durations. Cortical patients were also able to produce consistent formant transition durations, however, some overlap across phonemic boundaries was displayed by both cortical groups (see Table 3). The degree of overlap was assessed by means of the following equation normalized for speaking rate differences:

$$(\text{Transition time}[/\text{d}/] - SD[/\text{d}/]) - (\text{Transition time}[/\text{b}/] + SD[/\text{b}/])$$

A main effect of group was obtained when comparing cortical patients to controls ($F(2,10) = 6.42$, $p < .05$). Independent t tests confirmed that both cortical groups showed more overlap of transition times across phonemic boundaries than controls (frontal: ($t(8) = 3.63$, $p < .01$; temporal–parietal: $t(7) = 2.95$, $p < .05$)). The difference between controls and each subcortical group was not significant.

3.2.3. Place of articulation contrast—onset and offset frequencies

Given that formant frequencies will vary as a function of the fundamental frequency, analyses were conducted upon the difference in frequency between the onset and offset of $F1$ and $F2$ for /b/ and /d/. One-way group ANOVAs confirmed that $F1$ and $F2$ ascended or descended to the same degree for all patient groups and controls. Variability did not differ between groups.

One difference between /b/ and /d/ is in the onset frequency of the second formant. For /b/ this formant rises to the steady-state portion while for /d/ it descends (Allen, 1987). To determine whether patients were less able to make this distinction, the difference in frequency between the onsets of $F2$ for /b/ and /d/ was examined. However, all the patient groups performed similarly to controls.

3.3. Conclusions

The results of Experiment 2 accord well with the predictions delineated in the introduction. It was hypothesized that patients with damage to purely linguistic structures would show more overlap across phonemic boundaries than those with non-linguistic or no impairments. Both cortical groups displayed some overlap in formant transition duration across phonemic categories, and frontal patients exhibited greater variability in both transition duration and VOT. Although these measures may be disrupted for different reasons, the pattern of impairments displayed by the frontal and temporal–parietal patients were more similar to each other than to the subcortical group.

Cerebellar patients also performed as expected on measures of VOT production given their motor deficits. For these patients, variability was increased, but average duration was unimpaired. These patients were able to produce distinctive durations for each phoneme which accords well with the non-linguistic nature of their phonemic deficit.

Parkinson patients did not display abnormalities in any aspect of production. The intact performance of these patients on this production task lends weight to hypotheses suggesting that these patients will be primarily impaired on sentence-initial phonemes (Forrest et al., 1989). When a phoneme is placed in the final position of the carrier phrase, Parkinson patients are able to produce stop consonants that are not temporally or spectrally abnormal.

4. General discussion

These experiments constitute the first direct comparison of phonemic production and perception across a number of cortical and subcortical groups. Although each of these groups has been tested in separate studies of phonemic processing, there has been no systematic comparison using a common methodology.

Table 3
The range (mean ± one SD) of formant transition durations in milliseconds in Experiment 2

	Controls	Cerebellars	Parkinson's	Frontals	Temp–Pars
/ba/	28.53	17.47	25.05	33.31	22.87
	40.08	56.37	45.65	77.48	56.06
/da/	68.06	69.89	73.97	50.26	45.33
	87.37	107.18	101.95	72.99	69.24

The experiments reported here demonstrate that patients with damage to left-hemisphere speech centers perform in ways that are dissociable from those with damage to non-linguistic structures.

In accord with their dedication to purely linguistic processing, damage to left frontal and temporal–parietal cortices was associated with a pattern of performance that indicated disruptions of phonetic or phonological representations. On perceptual tasks, these patients displayed a gradual rather than a steep alternation between phonemic categories. Moreover, cortical patients were less accurate at identifying unambiguous instances within a phonemic category. When producing phonemic contrasts, both groups exhibited VOT and/or formant transition durations that were abnormal and overlapped across phonemic boundaries. Thus, cortical patients exhibited classification schemes and productions of phonemes that were less distinguishable in terms of place of articulation and voicing. Although, frontal aphasics appeared more impaired at production than temporal–parietal patients, both exhibited deficits that indicated difficulty with maintaining phonemic categories.

Damage to subcortical areas was associated with more selective deficits in phonemic production and perception. Both subcortical groups were relatively unimpaired at perceiving phonemic contrasts. They displayed evidence of categorical perception and were as accurate as controls in identifying unambiguous tokens on the voicing continuum. Both subcortical groups did far better on this task than either of the cortical groups. Non-overlapping distributions of VOT and formant transition durations were produced by cerebellar and Parkinson patients indicating intact phonemic representations. The cerebellum and basal ganglia appear to be important at executing intact phonemic representations rather than specifying what those representations should be.

An interesting dissociation occurred between the production of VOT and that of formant transition duration for the cerebellar patients. For VOT, cerebellar patients were impaired at producing consistent intervals whereas the variability of formant transition durations was normal. This is especially noteworthy given that both intervals are on approximately the same scale, being less than 100 ms. It may be that VOT is an interval that is explicitly programmed whereas formant transition duration is an indirect consequence of the force and trajectory of the articulators. When it is necessary to coordinate the sequence and timing of two articulators as in VOT, subcortical patients will be less able to execute these movements consistently. In contrast, formant transition duration may not be programmed, but be a product of other parameters that are explicitly specified by phonetic representations such as the trajectory of the movements needed to produce a certain phoneme. Indeed, cerebellar patients were able to produce formant durations that were contrastive for /b/ and /d/.

None of the spectral analyses were sufficient to identify dissociations between cortical and subcortical contributions to the production of phonemes. The lack of effects may have more to do with the current state of speech-analysis software rather than being a true reflection of patients' capabilities. It was difficult to estimate stable spectral measures for some of the participants when articulation was creaky or breathy. Moreover, the analysis program had difficulty extracting formant information for the most severely dysarthric or apraxic patients. Instead of going with less reliable estimates of formant frequencies, those trials were discarded, possibly obscuring differences between groups.

On the whole, these experiments have demonstrated that cortical and subcortical patients display dissociable patterns of performance on phonemic tasks. Given that performance on phonemic tasks often appears identical for cortical and subcortical groups across studies, it was deemed necessary to contrast phonemic abilities on the same set of experiments. By assessing the performance of several neurological groups on identical phonemic tasks, it is possible to determine the contribution of various neural structures to these processes. Both linguistic and non-linguistic neurological sites function together to perform the operations needed to perceive and produce phonemes. Investigating the contributions of neurological structures to phonemic processing will allow further delineation of the function of several neural areas, and allow a model of the computations involved in phonemic processing to be developed.

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