

Surveying Emotional Prosody in the Brain

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Abstract

Research has long supported a pivotal right hemisphere contribution to the decoding of emotional prosody, although a broader network of cortical and subcortical structures is now thought to support different components of this functional system during input processing. This paper highlights important work implicating the basal ganglia in emotional prosody decoding, especially in reinforcing key affective stimulus properties necessary for higher-order interpretative processes. The role of the right hemisphere in elaborating emotional-prosodic stimuli is then considered in reference to presumed ‘functional’ and ‘auditory-perceptual’ capacities of constituent regions. A broader description of the right hemisphere’s jurisdiction in social-emotive behaviour is advocated to advance future work in this area, and a new paradigm to tap on-line comprehension of emotional prosody in clinical populations is described.

1. Introduction

Researchers have long attributed a key role to the human right cerebral hemisphere in the processing of emotion, including its vocal-prosodic markers. Early conceptions of the right hemisphere as exclusively specialized to modulate expressive components of emotional prosody, and to construct a mental representation of these cues from speech, have been refined over the past decade; most researchers are now more explicit in favouring a distributed brain system devoted to emotional prosody, within which right hemisphere cortical regions serve a privileged, albeit shared, role [4]. Fortunately, empirical interest in the neural underpinnings of emotional prosody has increased in recent years, and is now being approached through varied perspectives using an increasingly broad range of investigative techniques.

One of the current challenges to research on emotional prosody in the brain is to define, and where possible isolate, physiological, behavioural, and cognitive components of emotional prosody for further study. At the level of cognitive control, there is still much indecision about the componential structure of emotional prosody, and thus, about how potentially separable processing components are linked to different areas of (hypothesized) network function in the brain. Working towards this level of detail will lead to more testable models of emotional prosody functions in the brain, and perhaps, help situate related capacities within a broader framework of social-emotive behaviour and communicative competence in humans [15], [25].

The following presents a sample of current issues and undertakings in the cerebral control of emotional prosody, concentrating largely on evidence from neuropathology. I will focus arguments on presumed cognitive-evaluative operations underlying the **decoding** of emotional meaning

from prosody, given indications that receptive and expressive aspects of prosody are to a considerable extent decoupled and require separate expository treatment [4]. In the first two sections, I outline ways in which subcortical (basal ganglia) and cortical (right hemisphere) areas of the cerebrum may be differentially recruited within a functional network devoted to emotional prosody decoding, supplementing conclusions made in each section with new observations from my lab [20], [22]. In a final section, I propose new directions for on-line, automatic evaluation of emotional prosody in the brain in an effort to stimulate alternative approaches to investigating emotional-prosodic phenomena in clinical populations.

2. Surveying the basal ganglia

The extensive involvement of limbic and paralimbic areas of the brain in coordinating emotional behaviour and integrating information on inner feeling states with external sensory data is well established. The human amygdala, with its direct interconnectivity with cortical (especially frontal lobe) structures, is known to play an especially critical role in affective evaluation and responsiveness [2]. Recent data suggest that the amygdala is a prominent component in a system that attributes emotional meaning to *facial* stimuli, although the participation of this structure in the recognition of emotion from prosody is far less certain [2], [3]. Somewhat overlooked in this sizable literature is the potential importance of the basal ganglia, a collection of grey matter nuclei situated deep in the cerebral hemispheres, to some of the operations underlying emotional prosody decoding.

The basal ganglia are best recognized for their central involvement in motor behaviour, although sectors of the neostriatum (particularly the caudate) also participate in a number of cognitive domains. Increasingly, it is coming to light that computational properties of the basal ganglia may be relevant to receptive aspects of emotional prosody, based on repeated reports of associations between prosodic deficits and acquired basal ganglia dysfunction. A critical summary of the two principal sources of this evidence is furnished below.

2.1. Evidence from focal damage to the basal ganglia

It has been noted on several occasions that stroke or other focal neurologic events that affect basal ganglia function are linked to difficulties understanding emotional prosody [11], [23], [27], [29]. Interestingly, repeated support for this relationship has often emerged in spite of most researchers’ focus on how neocortical (particularly right hemisphere) damage impacts on prosody judgements. In one important study, Cancelliere and Kertesz [11] examined a series of 46 acute stroke patients, characterizing each patients’ ability to produce, repeat, and identify prosodic contours representing four affective modes (happy, sad, angry, neutral). Patients

who displayed impairments in the ability to identify emotional prosody in relation to a control group were identified, and anatomical correlations of the prosodic impairments for this subgroup were derived using a CT overlap technique. The authors determined that damage to the basal ganglia, in addition to cortical damage in either hemisphere (especially when anterior temporal regions were affected), led to the most pronounced deficits in judging emotional tone from speech within their patient sample. They concluded that the basal ganglia are of “particular importance in the mediation of emotional expression and comprehension”, a position echoed by a later, related investigation of receptive prosody functions in 59 cerebrovascular patients [27].

In a report designed specifically to explore the effects of focal subcortical lesions on emotional behaviour, Weddell [29] also attributed a significant role to the basal ganglia in understanding emotional prosody. He observed that basal ganglia compromise was predictive of difficulties interpreting the happy, sad, angry, or neutral meaning of prosodic cues in semantically-neutral sentences, although it did not negatively impact on patients’ ability to interpret these meanings from verbal-semantic cues. Consistent with earlier findings [11], basal ganglia compromise in **either** hemisphere was related to the difficulties noted.

Thus, there are converging indications of the adverse effects of focal basal ganglia damage on operations necessary to interpret emotional prosody independent of verbal-semantic cues in speech. These investigations imply a central role for the basal ganglia in a neural system devoted to emotional prosody, although little of this evidence obtains from patients who presented exclusively with basal ganglia damage, mitigating the strength of these conclusions to some extent. Rather, more detailed inferences of this nature have been derived from the performance of individuals with idiopathic Parkinson’s disease on tasks of emotional prosody decoding.

2.2. Evidence from Parkinson’s disease

The early course of Parkinson’s disease (PD) is characterized by relatively focal degeneration of basal ganglia sectors which, over time, progressively deprive adjacent areas of the striatum, frontal cortex, and limbic regions of needed dopamine. For this reason, PD is often considered the “best neuropsychological model of basal ganglia functioning” [15], explaining the interest of many researchers in how affected individuals regulate speech prosody, among other phenomena. It is only now being recognized that PD leads to irregularities in how the emotional significance of vocal-prosodic cues is **decoded** from spoken utterances, in addition to impairing the expression of vocal emotion due to reduced physiological support for these processes, a sign commonly recognized in this population.

An investigation by Scott *et al.* [26] was the first to examine both expressive and receptive functions for prosody in non-demented PD patients. The authors concluded that their sample of 28 adults with PD displayed a “specific failure to react to patterns of intonation” based on their poor ability to recognize emotional and grammatical features of prosodic patterns when compared to healthy control subjects. Since that report, a correspondence between PD and reduced comprehension of emotional prosody in some form has been strongly indicated by the majority of studies [5], [7], [8], [16], [18]. Given the recent surge of interest in this area, it is now becoming possible to discern more specific relationships

between basal ganglia dysfunction and finer aspects of prosody decoding.

Following important work by Blonder and co-workers [5], Pell [18] assessed the ability of 11 PD patients to discriminate and identify the sad, happy, or angry meaning conveyed by different forms of prosodic stimuli. Discrimination tasks required subjects to judge whether pairs of prosodic contours low-pass filtered to obscure their segmental content sounded the same or different. Identification tasks required participants to label the prosodic tone encoded by ‘nonsense’ utterances (*Suh fector egzullin tuh boshent*) and by natural utterances containing congruent prosodic and verbal-semantic cues to the target meaning. Finally, subjects engaged in similar tasks that tapped their ability to decode the linguistic-pragmatic intent of utterances (statement, question, command) and to comprehend lexically-assigned stress.

Results indicated that reduced capacity to decode prosodic stimuli in PD was limited to **recognition** and not discrimination of prosodic cues, and limited to contexts when only prosodic and not verbal-semantic cues could be employed to generate a target response. However, deficits in the PD group were not constrained to contexts of deriving the **emotional** significance of prosodic cues, as patients in this sample displayed concurrent difficulties gauging the linguistic-pragmatic intent of utterances, although not the meaning of word stress features (*cf.* [5]). This overall pattern was interpreted in light of the basal ganglia’s role in mapping prosodic markers in spoken utterances onto meaningful representations of their underlying intent [18]. The suggestion that prosodic functions in PD were deficient primarily for features encountered over the sentential (and not lexical) domain, while not fully consistent with all data [5], has also been made in a follow-up study of 11 new PD patients [16].

There are separate indications in this small literature that basal ganglia dysfunction in PD does not uniquely affect the ability to construct emotional representations of sentence prosody, although researchers seldom focus attention on this issue. Of those few studies that tested how PD patients decode both emotional and linguistic meaning from prosodic contours, a significant proportion revealed impairments in both contexts when processing spanned the sentential domain [5], [26]. Comparable difficulties to process prosody in both emotional and non-emotional contexts is also strongly indicated by a new investigation of 21 PD patients we have just completed, discussed below (see [22]). Collectively, these results imply that key computational properties of the basal ganglia attributed to emotional prosody decoding may, in fact, be equally relevant to other forms of prosody decoding, pending further comparison of performance in different ‘functional’ contexts in PD patients. The specificity of prosodic decoding impairments in PD to emotional components of these events is an issue that merits future clarification, as such data hold potentially important implications for explanatory models of the basal ganglia within brain systems devoted to receptive prosody.

Recent work by Breitenstein and her colleagues [7], [8] strengthens arguments that the basal ganglia (caudate), with connected circuitry to prefrontal cortex, help to successfully elaborate meaning from emotional prosody in spoken language. Interestingly, data reported in this pair of studies also imply that part of this (presumed) failure to map prosodic input onto meaning may revolve around imprecise regulation of specific acoustic parameters of the stimuli that contribute to their interpretation. For example, in their initial evaluation

of 14 non-demented PD patients [7], observed deficits in the ability to decode individual emotions from prosody appeared to revolve around difficulties with specific acoustic attributes of the stimuli (i.e., those indicative of arousal). A follow-up investigation of 20 PD patients [8], designed partly to test the idea of an ‘acoustic processing deficit’ in PD, uncovered additional evidence of problems mediating acoustic properties of the input in their sample. In this latter study the authors independently manipulated fundamental frequency or temporal parameters of emotional utterances presented for recognition, and noted that when compared to healthy adults, their PD group displayed selective irregularities in the processing of speech rate information. They concluded that difficulties activating or reinforcing prosodic meanings was partly explained by less efficient utilization of **temporal** properties of prosodic contours as a cue to their emotional significance, perhaps owing to a broader temporal processing disturbance that accompanies basal ganglia/frontostriatal compromise [8]. The researchers also highlighted that executive subfunctions of emotional prosody tasks such as working memory capacity were concurrently reduced in their PD patients and explained a significant portion of the observed decline in prosody comprehension measures (also [7]). The extent to which executive dysregulation in the absence of dementia impacts on prosody comprehension remains another issue to monitor in future work on PD.

Thus, it is increasingly clear that the basal ganglia are instrumental in facilitating input as well as output components of emotional prosody, although several more detailed issues are not fully resolved, such as whether basal ganglia contributions are restricted to emotional processing *per se*. We set out to test these issues in an investigation of 21 healthy adults and 21 non-demented PD patients currently being prepared for publication [22]. Experimental participants completed a broad range of receptive tasks tapping their ability to discriminate, recognize, and rate the intensity of emotional features encoded by nonsense utterances representing one of five primary emotions (happy, pleasant surprise, anger, disgust, sadness). Subjects were also required to discriminate utterance prosody based on the perceived location of intra-sentential focus, and performed tasks of discrimination, recognition, and feature rating for static **facial** displays of the five emotions tested in the prosody battery.

Results of this most recent undertaking are highly consistent with the thesis that the intact basal ganglia form a critical component of a brain system devoted to prosody. PD patients with mild-moderate motor symptoms (i.e., relatively early in the disease) demonstrated reduced ability to use prosodic features to discriminate the emotional significance of paired utterances, and to provide verbal labels for these features in recognition tasks involving the same items. The patient group also displayed significantly reduced sensitivity to a wide spectrum of emotions present in individual vocal displays based on five separate tasks in which they judged the intensity of one pre-determined emotion on each occasion [2]. Initial impressions of these results point to a relatively generalized decline in the ability of PD patients to evaluate emotional prosody in the absence of verbal-semantic cues across several task-processing conditions—discrimination, recognition, and emotion feature intensity ratings. Neuropsychological measures indicated that performance on emotional prosody tasks was not due to underlying perceptual or obvious dysexecutive impairments in the PD group, although reduced working memory capacity did account for a

small amount of the variance in prosody scores for the patients [7], [8]. Emotional processing deficits were also not contingent on vocal features underlying specific target emotions in any of the processing conditions [22].

However, consistent with earlier discussion, the impact of basal ganglia compromise on prosody was associated with a concurrent impairment to decode **non-emotional** features of prosody in a discrimination task. Moreover, there was a marked discrepancy in the ability of the PD group to discriminate, recognize, and rate the intensity of the same emotional features from prosody than from visual attributes of static faces which was intact in all conditions (also [1]; see Figure). These function- and channel-related differences seem to again preclude explanations that basal ganglia involvement in prosody is confined to emotional components of the stimuli, or that these structures are critical to the evaluation of emotional events in a broad sense (i.e., encompassing prosodic, facial, and other nonverbal gestures). Findings do, however, reiterate that regions of the basal ganglia are indeed critical to systems underlying prosody decoding more generally. Since our study was initiated prior to reports concerning a selective temporal processing deficit in PD [8], we were not able to establish whether such difficulties may have accounted for part of the prosodic deficits noted in our data. Controlled investigation of the relationship between prosodic impairments and key stimulus dimensions of experimental tokens constitutes an especially promising area for future research on prosody and the basal ganglia.

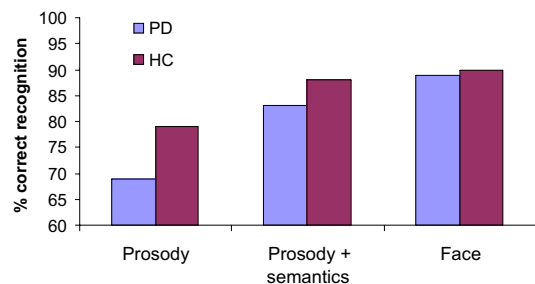


Figure 1: Recognition of five emotions by PD patients and healthy adults (HC) as a function of available cues.

2.3. Toward an understanding of how the basal ganglia contributes to receptive prosody

Based on data summarized above, it would appear that the prosodic “mapping failure” described by Pell [18] may be more constructively viewed as a failure of the damaged basal ganglia to somehow activate and reinforce key properties of prosodic stimuli needed for successful social-cognitive elaboration of these patterns by functionally contiguous brain regions (probably involving cortical, limbic, and paralimbic inputs) [8], [23]. In a recent synthesis of related work, Lieberman [15] emphasized the importance of basal ganglia structures, especially the caudate, in implicit learning and nonconscious or ‘intuition-based’ decoding of certain stimulus events. More precisely, he argued for the central involvement of the caudate in reinforcing the **temporal**, rather than conceptual, associations of incoming events and in assigning predictive value to their behavioural significance. Lieberman identified nonverbal decoding as a prime example of how the basal ganglia supports the nonconscious monitoring of rapidly changing temporal events, and notifies cognitive areas of the cortex about these predictive events.

Speech prosody, which is inherently temporal and which exploits finer aspects of temporal suprasegmental structure to encode discrete linguistic and emotional intentions, is certainly a behavioural system likely to benefit from the computational advantages of the basal ganglia described by Lieberman and others [8], [23]. This intriguing hypothesis converges with observations of subtle temporal processing deficits in PD [8] and may begin to explain why prosody decoding, rather than the decoding of static faces for example, is more frequently affected by even mild forms of basal ganglia dysfunction in experiments on PD [1], [22]. It may also explain why basal ganglia participation is not restricted to emotional processing of prosodic contours, pending further work on this topic. Forthcoming research will undoubtedly inform these preliminary views and contribute to detailed models of how the basal ganglia share the responsibility for speech prosody in both the expressive and receptive modality.

3. Surveying the right hemisphere

Far greater attention has been reserved for evaluating how the right hemisphere governs receptive aspects of emotional prosody [4]. As such, it is no longer an issue **if but in what way(s)** the right hemisphere contributes to component functions. Early notions that the right hemisphere accomplishes ‘all things prosodic’ at the cortical level have given way to evidence that this hemisphere dominates only certain, albeit important, aspects of prosody decoding. Unlike the basal ganglia, the right hemisphere appears to be differentially attuned to functional, and possibly auditory-perceptual, attributes of prosodic stimuli than the left hemisphere, yielding processing advantages in certain conditions. A look at some of the issues and controversies in this literature will provide a context for understanding the right hemisphere’s role within broader network system functions for prosody, and complement earlier arguments about subcortical involvement in emotional prosody.

3.1. Evidence from lesion studies and neuroimaging

Data collected over the past 25 years have continually reinforced the position that it is the right hemisphere that assigns emotional significance to prosody [4]. However, it is also strongly indicated that the right hemisphere is not responsible to elaborate all aspects of prosody (i.e. specialized to activate potential linguistic, pragmatic, and emotive associations of the suprasegmental content of an utterance). Rather, the cerebral hemispheres appear to be attuned to different **functional** attributes of prosody along a hypothetical continuum of ‘more or less linguistic’ or ‘more or less emotional’, depending on the view adopted. This notion of the ‘functional load’ of prosodic cues within an utterance has emerged as a central concept in describing the nature and degree of right hemisphere involvement in speech prosody at the level of the neocortex [12], [19]. This fact is best highlighted by research that compares hemispheric contributions to prosody in multiple domains simultaneously.

In a recent example, Pell and Baum [21] extended work by Heilman and colleagues [14], investigating how healthy, right-hemisphere-damaged (RHD), and left-hemisphere-damaged (LHD) adults interpreted the emotional versus the ‘linguistic-pragmatic’ relevance of prosodic contours in short utterances (choices were the same as described in [18]). Stimuli were presented for discrimination and in three emotion identification tasks that varied the type of cues

available to listeners. It was noted that attention to different functional properties of prosody was a significant determinant of how well the groups could identify prosodic meanings in the absence of semantic information (i.e., from nonsense or ‘speech-filtered’ utterances). Although the ability to recognize emotional or linguistic-pragmatic intentions of prosody was affected relatively equally by damage to either hemisphere (also [11], [27], [28]), direct comparison of each group’s performance between functional contexts revealed that damage to the **left** hemisphere was associated with a selective vulnerability in processing linguistic as opposed to emotional features of the stimuli. This pattern replicated Heilman et al.’s [14] findings, and accords with other frequent observations that deriving the linguistic or ‘predicative’ significance of prosodic cues in speech involves **relatively** greater contributions of the intact left hemisphere [4], [13]. Related research on the processing of phonemic stress and tone suggests in even stronger terms that the right hemisphere assumes a highly reduced role in linguistic analyses of prosody when compared to the left [4], [12].

Pell and Baum’s [21] report did not reveal complementary indications that deficits in their RHD group were significantly more pronounced for emotional than linguistic prosody, consistent with a strong functional interpretation of how the cerebral hemispheres subserve prosody. However, some of the other few reports that have collected performance measures on prosodic decoding in both linguistic and emotional contexts have described such a pattern [7], [19]. For example, Pell [19] observed a discrepancy in the ability of RHD patients to recognize emotion versus sentential focus from prosody, with markedly reduced performance in the emotion condition that was not exhibited by LHD or healthy control subjects. Again, however, findings obtained in this study represented the relative and not exclusive dominance of the right hemisphere to interpret emotional prosody, as LHD patients were also impaired in the emotion condition relative to matched control subjects without neurological compromise [11], [27], [28].

Recent neuroimaging and neurophysiological studies of prosody largely corroborate the opinion that each hemisphere is differentially equipped to process prosody along functional lines, and importantly, that this represents an incomplete specialization of each hemisphere to engage in respective processing [9], [12], [17], [24]. These collective findings build on arguments presented in the previous section that the cognitive regulation of prosody, including emotional prosody, is dependent on operations served by areas of the subcortex as well as cortical areas of both cerebral hemispheres [9], [11], [17], [19], [24], [27], [28]. For emotional prosody specifically, it is noteworthy that several of these new reports have highlighted the relevance of inferior prefrontal regions of both hemispheres in the possible integration and activation of emotion-related knowledge during prosody comprehension tasks [17], [24]. These findings imply a potential locus of high-order elaborative processes for emotional prosody within the cerebral hemispheres which accords with some evidence from pathology [8]. Interestingly, there are clues from available imaging data that prefrontal responses to emotional prosody are somewhat elevated in the right than in the left hemisphere in many subjects [9], [24].

What, then, dictates the right hemisphere’s bias for emotive components of prosody? Based partly on evidence supplied above, many in this field subscribe to a right hemisphere-based ‘nonverbal affect lexicon’ which houses

representational knowledge about how emotion is communicated, including its vocal-prosodic correlates [6]. According to this view, salient properties of affect-laden signals (prosody, facial expressions, gesture) that are initially activated and reinforced by a distributed emotion-processing network (including basal ganglia), are eventually compared with stored representational knowledge in the right hemisphere to derive the social significance of these cues. This idea of a right hemisphere ‘emotion processor’ that permits humans to assign social-emotive significance to sensory information for recognition, verbal identification, or in other high-order interpretative tasks has served as a working framework for research on emotional prosody and other aspects of emotive communication. This hypothesis may account for the relative dominance of the right hemisphere for emotional prosody in neuropsychological studies that almost invariably require patients to explicitly discriminate or label the intended meaning of the stimuli through **off-line** comprehension tasks. I will discuss some of the potential limitations of inferences based on off-line versus on-line measures of receptive prosody in brain-damaged patients in the final section.

There is another important factor that must be considered in models of right hemisphere prosodic function, which has proven somewhat difficult to reconcile with the presumed functional specialization of these cortical regions for emotion. Several detailed investigations of prosody decoding indicate that the right hemisphere preferentially attends to **pitch** rather than duration attributes of the stimuli [24], [28]. Some have related these findings to a broader right hemisphere predilection for pitch detection and complex auditory processing [9]. Accepting this, it is unclear how to disentangle right hemisphere effects stemming from functional considerations from those due to auditory-perceptual considerations, if such an enterprise is appropriate at all [12], [19], [24]. Certainly, there is sufficient evidence that pitch cues weigh heavily in the communication of emotional intentions in many languages, suggesting that future descriptions of emotional prosody are likely to appeal to both the functional and auditory-perceptual capacities of the right hemisphere that facilitate this form of processing, perhaps conjointly [19], [24].

3.2. Extending the right hemisphere’s role in prosody

As one may easily infer, psychological models ascribing to a set of ‘basic’, shared human emotions have largely directed how emotional prosody in the brain is characterized and studied. This theoretical position is perhaps overly restrictive and may be obscuring a broader purview for the right hemisphere in social behaviour and social-pragmatic judgements. In practical terms, emotional vocalizations are rarely intercepted in the absence of propositional speech; rather, prosodic features interact with linguistic-semantic information to convey a range of feelings and social attitudes about people or topics being discussed (e.g., doubt, sympathy, sarcasm). Even communication of ‘basic’ emotion states such as happy or sad occurs almost exclusively in **interpersonal** contexts and the precise form of representative prosodic cues is socially dictated to a considerable extent. Thus, understanding the intentions of emotional prosody, whether defined by the basic emotion states or more broadly, relies extensively on acquired **pragmatic** knowledge shared by the speaker-listener that guides expressive and receptive communicative behaviours. Determining the right

hemisphere’s role within the framework of social-pragmatic models of emotive communication (e.g., [10]) is therefore a useful direction for future undertakings on emotional prosody, and promises to better capture the type and **range** of knowledge that is usually applied to the problem of decoding underlying intentions of prosodic cues in speech.

We have recently adopted this approach in an ongoing experiment looking at how RHD patients decode prosodic cues to speaker confidence (‘evidentiality devices’, [10]). Initial results based on a sample of 5 RHD patients and 8 elderly control subjects do indeed encourage a broader view of the right hemisphere’s involvement in emotional prosody than previously envisioned; using a 5-point intensity scale, our patient sample displayed significantly reduced sensitivity to the underlying intent of prosodic attributes expressing high, moderate, and low degree of speaker confidence when presented in nonsense utterances. A pragmatic-interactive view of prosody in the right hemisphere has also been advocated in a newly published study of prosodic abilities following stroke [13]. Once confirmed, these and related findings may prove extremely helpful in linking functions that determine the social significance of (emotional) prosody with related operations that derive the **intended**, non-literal meaning of other forms of nonverbal and verbal stimuli which are also problematic for many RHD patients [25].

4. Surveying the survey: where do we go now?

Of course, one of the major challenges to inferring brain-behaviour relationships from neuropathological performance is to isolate operations of interest from executive or cognitive subfunctions that support these processes, or that reflect incidental task variables. This ongoing concern is highly relevant to investigations of prosody in individuals with PD or with right hemisphere cerebrovascular damage; each of these conditions is frequently associated with distinct forms of cognitive disturbance such as reduced working memory capacity, attentional difficulties, and/or problems with certain types of sensory recognition and recall. As noted earlier, there are already strong indications that some of these cognitive alterations are not entirely independent of difficulties understanding emotional prosody in generic off-line tasks that require brain-damaged subjects to discriminate or identify the meaning of prosody from a fixed set of response alternatives [7], [8]. The over-reliance on off-line performance measures in this literature—tasks which amplify demands for controlled processing, auditory working memory, selective attention, and verbal expression—is now becoming a barrier to advancing knowledge of emotional prosody in the brain based on inferences from neuropathology. One promising means of mitigating the potential confounds of traditional emotional prosody tasks is through development of **on-line** measures that will tell us which brain regions perform key analyses or functions underlying emotional prosody comprehension based on a more implicit or **automatic** analysis of stimulus features.

In a pilot study that is still ongoing, I have begun to test RHD, LHD, and healthy adults using a new on-line technique, the ‘facial affect decision task’. This paradigm, which resembles a nonverbal homologue of the cross-modal ‘lexical decision task’, is founded on the assumption of strong, overlapping connections between nonverbal systems devoted to emotional prosody and emotional faces [6]. Subjects are required to judge the value of a facial expression on a computer screen while listening to a nonsense utterance

produced in a happy or sad emotional tone. Facial stimuli represent prototypical happy or sad expressions ('true' emotional expressions) or a facial grimace that does not conform to a recognizable emotion ('false' expressions). A speeded yes/no decision about the candidacy of the face as a 'true' display of emotion is analyzed over a number of trials. Based on an initial study, young healthy subjects demonstrated markedly shorter response times when the sad or happy emotional prosody was congruent with the face than when the prosody was incongruent or neutral in tone [20]. This evidence was taken to reflect the implicit, mandatory effect of activated knowledge about the emotional significance of the prosody on facial judgements, despite the fact that participants were instructed to ignore the prosodic stimulus in making their judgements.

Initial impressions regarding the status of these processes in RHD patients are still forthcoming, but promise to illuminate whether knowledge structures associated with the basic emotions are automatically activated by prosody following specific right hemisphere lesions, and if so, whether this effect occurs to the same extent and follows the same timecourse as witnessed in healthy and brain-damaged control groups. Related experiments have been planned to ascertain whether difficulties observed in PD patients nonetheless reflect intact access to representational accounts of vocal emotions, as indicated by normal emotion congruency priming effects. It will be interesting to monitor how this and other new paradigms that tap the more automatic aspects of emotional prosody decoding are applied to different clinical populations and what specific inferences they will generate.

5. References

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