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Chapter 2.1

Hemispheric specialization for prosody perception: A meta-analysis of the lesion literature

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Abstract

It is unclear whether there is hemispheric specialization for prosodic perception and, if so, what the nature of this hemispheric asymmetry is. Using the lesion-approach, many studies have attempted to test whether there is hemispheric specialization for emotional and linguistic prosodic perception by examining the impact of left- versus right hemispheric damage on prosodic perception task performance. However, so far no consensus has been reached. In an attempt to find a consistent pattern of lateralization for prosodic perception, a meta-analysis was performed on 38 lesion studies (including 450 left hemisphere damaged patients, 534 right hemisphere damaged patients and 491 controls) of prosodic perception. It was found that both left- and right hemispheric damage compromise emotional and linguistic prosodic perception task performance. Furthermore, right hemispheric damage degraded emotional prosodic perception more than left hemispheric damage (trimmed *g =* −0.37, 95% CI [−0.66; −0.09], *N* = 620 patients). It is concluded that prosodic perception is under bihemispheric control with relative specialization of the right hemisphere for emotional prosodic perception.

1. Introduction

How we say something can be as important as *what* we say when conveying a message to our audience. This prosodic (supra-segmental) layer of speech uses a variety of acoustic cues such as speaking rate, pitch and intensity to convey different communicative functions. On the one hand, prosody can be used to convey information regarding the linguistic structure of an utterance (for a review of linguistic prosodic functions see Cutler, Dahan, & Van Donselaar, 1997). This 'linguistic prosody' can be used to stress syllables, group words into intonational phrases, emphasize importance of constituents in a sentence and to signal whether an utterance is meant as a question or a statement. On the other hand, prosody can be used to convey paralinguistic information such as the emotional state of the speaker (for a review see Scherer, 1986), which henceforth will be referred to as 'emotional prosody'.

Over the last four decades a considerable body of literature has accumulated on the question how the brain processes prosody (for recent reviews see Wong, 2002; Wildgruber, Ackermann, Kreifelts, & Ethofer, 2006; Kotz, Meyer, & Paulmann, 2006; Schirmer & Kotz, 2006; Alves, Fukusima, & Aznar-Casanova, 2008; Kotz & Paulmann, 2011). Understanding how prosody is processed in the brain is not only interesting from a fundamental cognitive neuroscience point of view but could also be clinically relevant as, for instance, impairment of prosodic processing has recently been found to be a core deficit in schizophrenia (Hoekert, Kahn, Pijnenborg, & Aleman, 2007). A central question that has remained unresolved so far is whether there is hemispheric specialization for prosodic perception, and if so, which mechanism drives this hemispheric asymmetry.

 Concerning lateralization of prosodic perception on the cortical level, four hypotheses have emerged:

(1) The right cerebral hemisphere is specialized in the processing of *all* prosodic information (Klouda, Robin, Graff-Radford, & Cooper, 1988);

(2) The *Right hemisphere hypothesis* posits that the right hemisphere is specialized in emotional prosodic processing (Ross, 1981; Blonder, Bowers, & Heilman, 1991; Borod et al., 1998);

(3) The *Functional lateralization hypothesis* (Van Lancker, 1980) proposes that hemispheric specialization is dependent on the communicative function of prosodic material: emotional prosodic information is processed in the right hemisphere while linguistic prosody is processed in the left;

(4) The *Cue dependent lateralization hypothesis* proposes that lateralization of prosodic processing depends on the acoustic cues that are critical for the extraction of meaning: the left hemisphere would be better adapted to processing of durational information while the right hemisphere is superior in spectral processing (Van Lancker & Sidtis, 1992). As variation in pitch is an important acoustic cue to the meaning of emotional prosody (but not the only cue; see Scherer, 2003), right hemispheric superiority for emotional prosodic processing could then be explained on the basis of rightward lateralization for pitch processing.

On the one hand, the first three hypotheses all assume a specialized (and lateralized) module for structuring of incoming acoustic information into *prosodic categories*. Note that these three "categorical" hypotheses need not necessarily be mutually exclusive; for instance, the functional lateralization hypothesis can be seen as a refinement of the right hemisphere hypothesis. On the other hand, cue-dependent hypotheses posit that lateralization of prosodic processing is determined by non-

prosody specific acoustic processes. The cue-dependent and the "categorical" lateralization hypotheses are not mutually exclusive either: they could represent different stages of prosodic processing which might be differentially lateralized (see Schirmer & Kotz, 2006).

One way to test these hypotheses is through the lesion approach. Typically, lesion studies compare a group of patients with acquired left or right hemispheric brain damage to a group of healthy controls on a prosodic perception task. If there is hemispheric specialization for prosodic processing then damage to the specialized hemisphere should (1) compromise performance on prosodic tasks as compared to controls (and equivalent damage to the non-specialized hemisphere should degrade performance relative to controls less) and (2) deteriorate performance as compared to equivalent damage to the non-specialized hemisphere. This approach provides information about which hemisphere of the brain is *necessary* for prosodic perception.

In order to differentiate between the "categorical" lateralization hypotheses it is necessary for lesion studies to have (1) a right hemispheric damage (RHD) group, a left hemispheric damage (LHD) group and a normal control (NC) group as well as (2) an emotional and a linguistic prosodic perception task. Unfortunately most lesion studies that have been published to date do not fulfill these criteria. The studies that did fulfill these criteria give an inconsistent picture with some presenting evidence favoring global right-hemisphere superiority for prosodic processing (Blonder et al., 1991; Borod et al., 1998) the right hemisphere hypothesis (Heilman, Bowers, Speedie, & Coslett, 1984) and the functional lateralization hypothesis (Walker, Daigle, & Buzzard, 2002) while others do not support any of the hypotheses (Pell & Baum, 1997; Pell, 1998; Breitenstein, Daum, & Ackermann, 1998; Geigenberger & Ziegler, 2001; Zgaljardic, Borod, & Sliwinski, 2002; Kho et al., 2007). When these studies and studies that included all relevant groups but imposed only one prosodic task find detrimental effects of hemispheric damage, many find that damage to *each* of the two hemispheres compromises emotional (Heilman et al., 1984; Van Lancker et al., 1992; Lalande, Braun, Charlevois, & Whitaker, 1992; Peper & Irle, 1997; Pell, 1998; Breitenstein, 1998; Zaidel, Kasher, Soroker, & Batori, 2002; Kucharska-Pietura, Phillips, Gernand, & David, 2003; Shamay-Tsoory, Tomer, Goldsher, Berger, & Aharon-Peretz, 2004; Pell, 2006; Kho et al., 2007, but for evidence of hemisphere specific degradation see Tompkins & Flowers, 1985; Bowers, Coslet, Bauer, Speedie, & Heilman, 1987; Blonder et al., 1991; Geigenberger et al., 2001; Walker et al., 2002; Charbonneau, Scherzer, Aspirot, & Cohen, 2003) and linguistic (Heilman et al., 1984; Pell et al., 1997; Baum, 1998; Geigenberger et al., 2001; Aasland & Baum, 2003; Seddoh, 2006b, but for hemisphere specific degradation see Bryan, 1989; Blonder et al., 1991; Perkins, Baran, & Gandour, 1996; Pell, 1998; Borod et al., 1998; Walker, Fongemie, & Daigle, 2001; Walker et al., 2002; Abada & Baum, 2006) prosodic perception performance, suggesting that both hemispheres provide necessary contributions to both prosodic functions.

To disentangle the contribution of "categorical" versus cue-dependent hemispheric specialization in prosodic perception it is necessary to vary the function of the prosodic material while keeping acoustics constant or vice versa and observe whether there is differential impact of left- versus right-hemispheric damage on prosodic perception performance as compared to performance by NC. One approach has been to selectively remove durational or fundamental frequency $(F₀)$ variation in linguistic or emotional prosodic stimuli and to observe whether LHD or RHD differentially degrades perception performance as compared NC. Unfortunately these

studies (Pell, 1998; Baum, 1998; Aasland & Baum, 2003) have not consistently found differential degradation of performance after removal of F_0 variation for LHD and after removal of durational information for RHD, as would have been expected based on the Cue-dependent lateralization hypothesis (Van Lancker et al., 1992). Adopting a different approach, Van Lancker et al. (1992) used discriminant analysis to analyze which acoustic properties of emotional prosody could predict the pattern of errors made by LHD and RHD patients on an emotional categorization task. It was shown that the errors of the RHD patients could be predicted by misuse of F_0 variability. The authors concluded that the right hemisphere might contribute to emotional prosodic perception through a specialization in pitch processing. However, this conclusion must be considered with caution as Baum and Pell (1997) failed to replicate the result.

Several factors have been suggested in the literature that can moderate the impact of lateralized brain damage on prosodic perception performance. Ross, Thompson, and Yenkosky (1997) propose that apparent emotional prosodic processing deficits after LHD are not caused by emotional prosodic processing deficits per se, but that these patients have problems linking emotional meaning from the prosodic layer to the propositional layer of the speech signal. These authors predict that when the 'verbal-articulatory demands' (whether lexical meaning and syllables are present) of an (affective) prosodic perception task are increased LHD performance should degrade while RHD performance should remain unaffected. Secondly, as was already evident in our discussion of the non-mutual exclusivity of the cue-dependent versus "categorical" hypotheses of prosodic perception, prosodic processing can be conceptualized as a *process* consisting of several stages. For instance, in a recent review Schirmer and Kotz (2006) propose that there are at least three stages in prosodic perception (see also Kotz et al., 2006). In an initial stage, complex acoustical analysis of the speech signal is performed; in the second stage, emotional or linguistic information is identified; and in a final stage, this information becomes available to higher-order cognitive processes for further evaluation or integration with other layers of speech (such as the propositional content). This proposal implies that performance for prosodic perception tasks such as those used in the lesion literature reflects a combination (i.e. summation or even interaction) of these stages, each of which might be differentially lateralized (Gandour, 2004). Lastly, as Hoekert et al. (2007) have pointed out in a meta-analytic review of emotional prosodic impairment in schizophrenia, the quality of the prosodic perception task used might influence the findings. Tasks with high psychometric quality can be expected to give a better picture of prosodic performance degradation due to lateralized brain damage than tasks of low psychometric quality.

In sum, although a considerable body of lesion literature has accumulated, no consensus has been reached on the degree and nature of hemispheric lateralization for prosodic perception. Most studies that had the appropriate design for differentiating between the "categorical" lateralization hypotheses of prosodic perception find that both LHD and RHD can affect prosodic perception, suggesting that hemispheric specialization for prosodic perception is a matter of *degree* rather than type. The small number of studies that have manipulated acoustic cues in prosodic perception tasks have not consistently supported the cue-dependent lateralization hypothesis. The most important problems in testing the "categorical" lateralization hypotheses mentioned is that many of the lesion studies published to date do not have the appropriate design to disentangle the various lateralization hypotheses (Wong, 2002). Furthermore, studies

typically had fewer than 15 subjects per experimental group, limiting statistical power to detect effects.

In the present study, a meta-analysis was employed to review the lesion literature on hemispheric specialization for prosodic perception. By (1) including RHD, LHD and NC groups as well as (2) both emotional and linguistic perception tasks in the meta-analysis, it was possible to overcome the main weaknesses of previous individual studies and meta-analytically differentiate between the "categorical" lateralization hypotheses of prosodic perception (the number of studies manipulating acoustics while keeping prosodic function constant or vice versa was too low to contrast the "categorical" hypotheses with the cue-dependent lateralization hypothesis). For instance, although individual studies that have only included a NC group and a LHD or RHD group and one prosodic function (e.g. Baum, Daniloff, Daniloff, & Lewis, 1982; Wertz, Henschel, Auther, Ashford, & Kirshner, 1998; Seddoh, 2006; Weintraub, Mesulam, & Kramer, 1981; Harciarek, Heilman, & Jodzio, 2006) cannot give any definitive information on lateralization of prosodic processing (i.e. whether one of the hemispheres is *specifically* necessary for emotional or linguistic prosodic perception or both), such studies are valuable for discriminating between the "categorical" lateralization hypotheses when compared meta-analytically. Furthermore, by including a large number of subjects for each experimental group in the metaanalysis, the lateralization hypotheses of prosodic perception could be tested with high statistical power, making it possible to demonstrate subtle effects that individual studies cannot detect. By summarizing studies quantitatively a more precise and objective insight in the effect of lateralized brain damage on prosodic perception task performance can be gained as compared to the traditional qualitative reviews. Additionally, we tested the influence of factors that have been suggested to moderate the relationship between lateralized brain damage and prosodic perception performance by moderator-analysis. Moderating variables (such as whether patients and controls are matched on demographic variables) typically vary only between studies. Therefore, while most individual studies cannot test for such effects, meta-analytic moderator analysis can provide novel insights by assessing the impact of moderators. Finally, since the last qualitative review of the lesion literature on prosodic processing was published in 2002 (Wong, 2002), the current review additionally covers almost a decade of relevant research.

2. Methods

2.1. Literature search

The PubMed and PsycLit databases were searched for relevant articles published until January 2011 using the search string '*prosod** AND *brain* NOT *EEG* NOT *ERP* NOT *MRI*' (where the asterisk denotes a wildcard) in the title or abstract. Additionally, the reference lists of all articles included in the meta-analysis and available reviews (Wong, 2002; Kotz et al., 2006, 2011) were manually checked for previously published potentially relevant articles. This search yielded 80 publications that were considered for inclusion in the meta-analysis.

2.2. Study selection

Studies needed to fulfil the following set of criteria to be eligible for inclusion in the meta-analyses. First, (i) at least an adult group with acquired left hemispheric damage (LHD) and a group with right hemispheric damage (RHD) or (ii) at least one of these brain damaged groups and a normal control (NC) group had to be present. Second, objective (CT/MR imaging or surgical) evidence for the lateralized nature of the damage had to be presented for the majority of the patients. Third, the brain damage had to be primarily cortical and focal: lesions had to be clearly localizable and situated primarily in the cerebral cortex which implied that most lesions were caused by cerebrovascular damage or surgical intervention (such as tumor extirpation or resection for intractable epilepsy). We excluded non-focal (diffuse) etiology such as diffuse traumatic brain injury or Parkinson and Huntington pathology. As part of our general strategy to include as many lesion studies as possible in order to maximize the scope of our results and test for potentially moderating effects factors such as etiology rather than exclude studies, no further restrictions were imposed on etiology of the brain damage. However, as can be seen in Tables 1 and 2, the vast majority of included studies presented patients with brain damage of vascular origin. Fourth, at least one explicit linguistic or emotional prosodic perception task had to be reported and performance needed to be qualitatively interpretable (i.e. we excluded rating tasks that do not allow for the interpretation of test scores in terms of good/bad). Emotional prosodic perception tasks typically present a set of prerecorded utterances that are intoned in a variety of emotional categories by an actor; patients typically have to identify (or rate the intensity of) the intended emotion or discriminate between the emotional intonation of utterance pairs. Linguistic prosodic perception tasks also typically demand subjects to identify or discriminate between prosodic categories, but in this case prosody imparts linguistically relevant (and hence categorical) information such as sentence modality, sentential stress, syntactic structure through phrase marking or metrical stress. One of these studies (Grosjean, 1996) used an atypical task: the ability of subjects to predict from sentence prosody whether a sentence continues or stops at a certain target word. We considered this task to tap linguistic prosodic processing because it is a measure of the ability to perceive phrase structure on the basis of prosodic features (which can be considered a linguistic prosodic function, e.g. for a review see Cutler, Dahan, & Van Donselaar, 1997). Fifth, the study had to report original material (e.g., we excluded Ross & Monnot (2008), who reanalyzed data from their research database) in order to prevent data from entering a meta-analysis twice (which violates the assumption of independence) and had to be published in an English language international peer-reviewed journal. Implicit prosodic perception tasks (e.g., Wunderlich, Ziegler, & Geigenberger, 2003) were not included as they might engage different neural systems (for a review of this issue see Wildgruber et al., 2006) and by definition tap other processes in addition to prosodic processing (i.e. the patient is actively engaged in a different task than evaluating the prosodic information). Furthermore, there were not enough studies that reported implicit tasks to look at the moderating effect of the explicit versus implicit nature of the task. Sixth, we excluded studies on tonal languages as the literature suggests that these might have a different lateralization pattern than non-tonal languages (e.g., see Gandour et al., 2003). Finally, the study had to report sufficient information to be able to compute or accurately estimate the standardized difference in means. This required the study to report (i)

sample size and means and standard deviations or (ii) sample size and test statistics such as *Z-* or *T-* or *F*-values with means or (iii) sample size and exact or categorical *p*-values.

2.3. Data analysis strategy

We tested the effect of RHD versus LHD and of lateralized damage versus NC performance for both emotional and linguistic prosodic tasks. For meta-analysis to be statistically valid, it is necessary that each study and each subject only contributes to the analysis once. This assumption of independence required six separate meta-analyses: three comparisons (NC vs. LHD; NC vs. RHD; LHD vs. RHD) for each of the two prosodic functions (emotional vs. linguistic).

Additionally, we wanted to assess differences between comparisons in the mean weighted effect size (ES). For instance, we asked whether damage to the specialized hemisphere compromises performance (as compared to NC) *more* than damage to the non-specialized hemisphere, or whether damage to a hemisphere *differentially* disturbs performance for linguistic or emotional prosodic processing. Therefore, the 85% confidence interval (CI) around the mean weighted ES under the random effects model was computed for each meta-analysis. Non-overlapping 85% CIs of two mean weighted effect sizes indicate a significant difference at the traditional 5% type I error threshold (Goldstein & Healy, 1995). This approach allowed us to assess whether there were differences in the mean weighted ES between meta-analyses and to present a graphical overview of the analyses.

Many studies reported multiple (and hence non-independent) measures of prosodic processing. To preserve independence we pooled multiple effect sizes to obtain one measure of ES per study for each of the six comparisons. This strategy had the following implications. First, we pooled across multiple measures of a prosodic function. However, if a study reported unimodal (only prosodic) and multimodal prosodic tasks (for instance, tasks in which emotional semantics can be congruent or incongruent with the emotional meaning of the prosody), only the unimodal task was used (as the former is a purer measure of *prosodic* processing). Second, if data were presented for subgroups with damage to different locations within a hemisphere we pooled to a mean ES for the whole hemisphere (since *inter*hemispheric differences are the focus of the meta-analysis). Third, if pre- and post-surgical data (tumor extirpation, resection of epileptic foci) were reported, we only analyzed the post-surgery data (preserving the overall logic of analyzing the effect of acquired brain damage on prosodic processing between groups).

2.4. Computation of effect size and meta-analytic procedures

Hedges's measure of effect size *g* (the standardized difference in means) was calculated for each comparison. Hedges's *g* is very similar to Cohen's *d* (the difference between means divided by the pooled standard deviation) but is less biased if the sample size is small (Hedges & Olkin, 1985). When comparing patient groups to NC we gave the ES a negative sign if patients performed worse than NC to indicate deterioration of performance due to brain damage. When comparing the LHD to RHD group we gave the ES a minus sign if RHD performed worse than LHD since most hypotheses of hemispheric specialization for prosodic processing predict performance degradation for the RHD group relative to LHD.

The included studies varied widely in the quality of the reported statistics. When possible, ES was calculated from the sample size and the (pooled) means and standard deviations. If the standard deviation was zero we entered the value 1·10-10 because the Comprehensive Meta Analysis package (Borenstein, hedges, Higgins, & Rothstein, 2005) does not accept standard deviations of zero. If the study reported sample size and either a t-test or sample size and a *p-*value, we used Comprehensive Meta Analysis (CMA) to convert to Hedges's *g*. When (one way) *F-*tests were reported, we used the "Effect size determination program" developed by Wilson (http://mason.gmu.edu/~dwilsonb/downloads/ES_Calculator.xls) to convert to *g*. When categorical *p*-values were reported, we entered the upper border in CMA (i.e. $p =$ 0.05 when $p < 0.05$ was reported), and when 'no effect' was reported, we entered an ES of 0, adopting a conservative approach.

Two additional sources of information were used to estimate *g*. When the main effect *F*-value of a multiple way ANOVA was reported, we used the 'Effect size determination program' to convert it to *g*. This procedure potentially overestimates the true ES as the error term of the *F*-test will be smaller due to the inclusion of an additional factor (apart from the factor of interest: lateralized brain damage versus control). We also entered *Z-*values (performance scores that have been normalized using the mean and standard deviation of the control group) as *g* in the meta-analysis for comparisons of patient groups versus controls. These measures of ES may also overestimate ES since it is not the pooled standard deviation but the (likely smaller) standard deviation of the control group that is used in the denominator. We later tested in a moderator analysis whether these last two sources of information gave a significantly larger ES than the other measures of ES but this test failed to reach significance. Therefore, we decided to include these measures of ES in the metaanalyses in order to increase the representativeness of the results.

For each meta-analysis, the distribution of effect sizes was checked for outliers by first converting all *g*'s to Fisher's *Z* and subsequently converting to standard normal scores. Outliers were defined as values outside the −3.29 to 3.29 range (corresponding to a probability that is lower than 0.001 in the normal distribution). As none of the study outcomes fulfilled this criterion all the included outcomes remained in the metaanalyses.

All further meta-analytic procedures were performed with Comprehensive Meta-Analysis (Borenstein et al., 2005). The mean weighted ES *g* and 95% CI were computed using the random effects model. The random effects model was chosen because it is reasonable to assume that the true ES varied among included studies (see Borenstein, Hedges, Higgins, & Rothstein, 2009) and it produced more conservative results. For each meta-analysis the mean weighted effect size and its confidence interval was computed using the inverse variance weight of each study under the random effects model, ensuring that the measurement precision of contributing studies was taken into account when computing the summary statistics. Subsequently, the *Q*statistic was computed to test whether there was significant heterogeneity in the ES distribution (Lipsey & Wilson, 2001). Follow-up moderator analyses were performed to explore whether theoretically or methodologically plausible factors (see below) could explain variance in the ES distribution.

Seven studies fulfilled all inclusion criteria but failed to report sufficient information to calculate *g*. In order to give a complete overview of the issue at hand these studies were analyzed through narrative review.

2.5. Publication bias

A threat to the validity of meta-analysis is the so-called 'file drawer problem' (Rosenthal, 1979). This refers to the phenomenon that studies that find statistically significant differences are published more easily than studies failing to reject the null-hypothesis (they remain in the file drawer). These statistically non-significant studies have a lower probability of being included in the meta-analysis than studies that do find statistically significant differences resulting in the risk of overestimation of the true ES by metaanalysis. This issue can be examined by plotting the ES of each study against the precision (defined as the inverse of the standard error) in a so-called 'funnel-plot' (Lipsey & Wilson, 2001). If there is no publication bias, this plot should look like a funnel where the less precise studies are scattered more widely around the point estimate than the more precise studies. If there is publication bias, studies with lower precision and a small ES (that did not get published) should be missing causing an asymmetry at the base of the funnel. For each of the six meta-analyses we checked the funnel plot for publication bias. Subsequently, we formally tested whether there was funnel plot asymmetry using Egger's regression test (Egger, Smith, Schneider, & Minder, 1997). This test aims to quantify funnel plot asymmetry by regressing the standard normalized ES against its precision (the inverse of the standard error). If there is funnel plot asymmetry the intercept of the regression line should be significantly different from zero (where $p = 0.10$ is chosen as the statistical threshold to compensate for the limited power of the test). Furthermore, we computed Orwin's fail-safe number of studies (Orwin, 1983) which is the number of unpublished and statistically nonsignificant studies that is needed to reduce the observed ES to a negligible effect (which we defined as a *g* of −0.20). A large fail-safe number of studies gives credence to the robustness of the observed ES. Lastly, we used the trim-and-fill method (Duval & Tweedie, 2000) to correct the observed ES for publication bias. This method iteratively trims small studies on the positive side of the funnel plot until it is symmetric, 'fills' the funnel plot with the trimmed studies and their mirror images (in order not to underestimate the variance), and recalculates an adjusted pooled ES. It is assumed that the adjusted ES is a more precise estimate of the true ES since it also incorporates unpublished studies.

2.6. Moderating variables

As discussed in the Introduction, the lesion literature suggests several variables that can moderate the relationship between lateralized brain damage and performance on prosodic perception tasks. These can be broadly categorized as either sample or task characteristics.

2.6.1. Sample characteristics

First, it is possible that hemispheric specialization patterns for prosodic processing differ between languages, especially for linguistic prosody (e.g., see Gandour et al., 2003). Therefore, we tested whether the mean weighted ES differed between studies using (American) English subjects and studies that used non-English speaking populations. There was not enough variation in languages between studies to further differentiate between specific languages. Second, studies varied in the proportion of the patient sample that had objective (radiologically or surgically confirmed) evidence for the lateralized nature of the brain damage. Studies that provide objective evidence for the lateralized nature of the brain damage for all patients will provide a more accurate

picture of the effect of LHD and RHD on performance than studies that do not (as in the latter case the possibility cannot be excluded that damage was in fact contralateral or bihemispheric for some patients). We therefore tested whether there was a difference in the mean weighted ES between studies that presented objective evidence for the lateralized nature of the damage for the total sample versus studies that did so for the majority of the sample (but not all patients). Third, when experimental groups significantly differ in demographic (such as sex or age) or neuropsychological variables (such as attention deficit) that could affect prosodic processing (see Fecteau, Armony, Joanette, & Belin, 2004; Schirmer, Striano, & Friederici, 2005), it is possible to erroneously conclude that differences between groups in prosodic processing are due to differences in lateralized brain damage while in reality the aforementioned confounders are (partially) responsible. Therefore, we tested whether there were differences in the mean weighted ES between studies that had matched groups on at least one demographic or neuropsychological variable versus studies that did not match experimental groups. Finally, it is possible that the nature of the brain damage influences the occurrence of prosodic perception deficits. Therefore we tested for a moderating influence of etiology of the brain damage by comparing studies that tested patients with only vascular damage, only other than vascular damage or vascular and non-vascular damage.

2.6.2. Task characteristics

First, similarly to Hoekert et al. (2007) we tested the following task quality parameters: (1) whether or not the article reported psychometric (reliability, validity) information about the task; (2) whether the actors that produced the prosodic material were professional actors or phoneticians or not; (3) whether at least six items per prosodic category were used for the task or less (since fewer than six items compromises the reliability of the task) and (4) whether six or more or less than six prosodic categories were used for the task (as more than six prosodic categories might tax working memory of the patients too much and confound degradation of performance due to *prosodic* processing deficits).

The influence of 'verbal-articulatory load' (Ross, Thompson, & Yenkosky 1997) was tested in multiple ways. First, it was tested whether there was a difference in the mean weighted ES for tasks that used speech material that contained lexical semantics versus tasks that used material without lexical semantics (such as pseudolanguage or low-pass filtered speech). Secondly, we tested whether there was an effect of the extent to which the response procedure taxed verbal abilities: we contrasted tasks that demanded a verbal response (such as pointing to a verbal label) versus tasks that required a nonverbal response (such as pointing to a facial expression) versus tasks that allowed for both modes of responding. This moderator is also interesting from a different perspective than the variation in verbal load: one could argue that tasks that allow for multiple ways of responding are a more valid measure of *prosodic* processing than tasks that only allow one kind of response as performance is less affected by response-specific (non prosodic) factors. Lastly, we compared identification with discrimination performance. Identification tasks tax verbal capacities to a greater extent than discrimination tasks (Pell, 2006) as in the former case prosodic information needs to be associated with verbal categories while in the latter case no such verbal categorization is required. Because many studies that used discrimination tasks also used identification tasks (forcing us to pool those tasks to preserve independence), we

decided to contrast studies that used both tasks to studies that only used identification tasks instead of comparing discrimination to identification orthogonally.

Contrasting the effect of lateralized brain damage on discrimination versus identification performance could also be interesting with regard to the proposed subdivision of the prosodic perception process in at least three stages (Schirmer & Kotz, 2006). One could argue that discrimination tasks primarily tap early processes (acoustic analysis, determination of emotional or linguistic significance) while identification tasks additionally tap the later evaluative processes. Therefore, contrasting these two tasks allows one to investigate whether lateralized brain damage differentially disturbs earlier or later prosodic processing stages.

Lastly, a number of variables were checked for moderating effects but these variables did not vary enough between studies to perform a sufficiently powerful moderator analysis (i.e. there were fewer than five studies per level of the moderator). These moderators included the size of the (prosody carrying) unit (e.g., Gandour et al., 2003) as most studies used sentences, the length of the lesion-onset-testing interval (nearly all studies tested patients in the 'chronic stage' i.e., used intervals of longer than 3 months) and whether lesion localization was only cortical or cortical and also subcortical (nearly all studies involved patients with cortical and subcortical damage). Similarly, we also aimed to test the hypothesis that perception of positive versus negative (or approach versus withdrawal) emotions is lateralized differently but the number of studies presenting data for these emotional categories separately was too small for a meaningful statistical analysis.

2.7. Explorative intra-hemispheric analysis

Although the current meta-analysis is focused on *inter*hemispheric differences in prosodic perception performance, several authors have proposed specific roles for different *intra*hemispheric loci. For instance, Schirmer and Kotz (2006) have proposed that the (bilateral) superior temporal sulcus and gyrus (STS/STG) are involved in the determination of emotional significance of prosodic cues while at a later stage prosodic information is evaluated and integrated with other cognitive processes in the frontal cortex. Ross and Monnot (2008) have also proposed a different role for temporal lobe and frontal lobe areas in emotional prosodic perception. These authors posit that the temporal operculum of the right hemisphere is crucial for adequate emotional prosodic perception and suggest that while damage to the right hemispheric temporal operculum differentially degrades emotional prosodic perception performance as compared to frontal cortical damage, for the left hemisphere the intrahemispheric cortical locus of the lesion does not predict prosodic perception performance.

 Given these intrahemispheric models of emotional prosodic perception it would be interesting to meta-analytically test whether the intrahemispheric locus of the brain damage influences prosodic perception performance. Unfortunately, there were not enough studies reporting performance measures separately for subgroups of patients with damage restricted to certain intrahemispheric cortical loci, preventing us from directly meta-analytically investigating this issue. However, a more explorative analysis was undertaken. For each study, the number of patients with temporal and frontal lobe damage (as reported by the authors) was counted and the percentage of patients with temporal and frontal damage was derived. Subsequently, for each patient group as compared to controls and for emotional and linguistic prosody, we performed a meta-regression analysis (e.g., see Lipsey & Wilson, 2001) to investigate the influence of percentage temporal and frontal lobe damage on effect size for prosodic perception performance. A weighted least squares regression analysis was performed with percentage temporal and percentage frontal damage as the predictors, the inverse variance under the random effects model as the regression weight and the effect size as the criterion. Similarly to Alink et al. (2008), we converted the effect size to Fisher's Z and used this metric as the dependent for the meta-regression analysis as it has superior distribution characteristics (Lipsey & Wilson, 2001). To test statistical significance of predictor beta-weights, a corrected standard error was used as suggested by Lipsey and Wilson (2001).

We also counted the number of patients with subcortical damage and white matter lesions (as reported in the article) for each study. Unfortunately, the percentage of patients with white matter or subcortical damage did not vary enough between studies to allow for a meta-regression analysis. As white matter or subcortial lesions have been implicated in prosodic perception performance (e.g. see Ross & Monnot, 2008) we were concerned that cortical lateralization effects might be confounded with these lesions. Therefore, we used an independent t-test to check whether there was a systematic difference between the right and the left hemisphere in the percentage of patients with subcortical or white matter lesions.

3. Results

A total of 38 studies fulfilled the inclusion criteria: 28 studies provided data for the meta-analyses on emotional prosody (Table 1), and 20 studies for the meta-analyses on linguistic prosody (Table 2). An independent rater (DV) coded the part of the coding form involving the study, sample and task characteristics for 10 studies (26% of all included studies). The mean inter-rater agreement was 97.9% suggesting a high level of reliability of the coding process.

3.1. Emotional prosody

3.1.1. LHD vs. NC

A total of 21 studies (with a total of 287 LHD-patients and 399 NC) provided sufficient data for this comparison (see studies with an NC and LHD group in Table 1). The ES distribution with 95% CIs (the forest plot) can be found in Figure 1. The mean weighted ES *g* (95% CI) under the random effects model was −1.06 (−1.40; −0.71) suggesting a large effect of left hemispheric damage on emotional prosodic processing. Inspection of the ES to measurement precision scatterplot (the funnel plot: Figure 2) suggested publication bias. In Table 3 it can be observed that Egger's regression test confirmed the existence of publication bias. The number of statistically non-significant unpublished studies (Orwin's fail-safe N) to reduce the observed ES to a negligible effect was sufficiently large to give credence to the robustness of the observed effect. Trim and Fill did not result in an adjustment of the mean weighted ES.

The heterogeneity statistic was significant $(Q(20) = 81.23, p < 0.0001)$ suggesting significant unexplained variance in the ES distribution. None of the moderators explained a statistically significant amount of variance in the ES distribution.

Study	Etiology Language		CT/MR	N_{L}	N_{R}	Nc
Heilmanet al. (1984)	English	CVA	Most	9	8	15
Tomkins et al. (1985)	English	CVA	Most	11	11	11
Bowers et al. (1987)	English	ς	ς	10	10	12
Ehlers et al. (1987)	Danish	Mixed	All	5	11	
Blonderet al. (1991)	English	CVA	All	10	10	10
Brådviket al. (1991)	Swedish	CVA	Most	$\overline{}$	20	18
Lalande et al. (1992)	French	CVA	Most	10	12	16
Van Lancker (1992)	English	CVA	Most	24	13	37
Hornak et al. (1996)	English	Mixed	Most	$\overline{4}$	11	16
Pellet al. (1997)	English	CVA	5	10	9	10
Peper et al.(1997)	German	Resection	All	21	19	12
Ross et al. (1997)	English	CVA	All	10	12	16
Schmitt et al. (1997)	German	CVA	ç	25	27	26
Breitenstein et al. (1998)	German	Mixed	All	16	16	10
Pell (1998)	English	CVA	All	11	9	10
Wertz et al. (1998)	English	CVA	Most	$\overline{}$	20	18
Karow et al. (2001)	English	Mixed	All	10	10	5
Walker et al. (2002)	English	CVA	All	8	8	8
Adolphs et al. (2002)	English	Mixed	All	25	26	5
Zgaljardic et al. (2002)	English	CVA	L: Most	7	9	$\overline{7}$
			R: All			
Charbonneau et al. (2003)	French	CVA	Most	17	15	16
Hornak et al. (2003)	English	Mixed	Most	9	16	48
Kucharska et al. (2003)	Polish	CVA	All	30	30	50
Shamay-Tsoory et al.(2004)	Hebrew	Mixed	All	18	16	19
Harciarek et al. (2006)	Polish	CVA	All	$\overline{}$	30	31
Pell (2006)	English	CVA	All	11	9	12
Rymarczyk et al. (2007)	Polish	CVA.	All	$\overline{}$	37	26
Kho et al. (2008)	Dutch	Resection	All	16	15	47

Table 1. Overview of studies on *emotional* prosody.

Language = native language of the subjects; $CT/MR =$ objective $(CT/MR \text{ imaging})$ or surgical-) verification of lateralized damage for all or most patients; N_L = number of patients with left hemispheric damage; N_R = number of patients with right hemispheric damage; N_C = number of healthy controls.

Figure 1. Forest plot of the mean effect size (*g)* and 95% confidence interval for each of the studies included in the comparison of LHD to NC for emotional prosodic perception. Larger symbols indicate a larger number of subjects included in the respective study.

Figure 2. Funnelplot of the studies included in the comparison LHD to NC for emotional prosodic perception. The effect size *g* of each study (x-axis) is plotted against its standard error (yaxis). The vertical line represents the mean weighted effect size and the diagonal lines the 95% confidence interval.

3.1.2. RHD vs. NC

A total of 26 studies (with a total of 402 RHD patients and 508 NC) provided sufficient data for this comparison (see studies with an NC and RHD group in Table 1). The forest plot can be found in Figure 3. The mean weighted ES (95% CI) was −1.41 (−1.76; −1.05) suggesting a large effect of right hemispheric damage on emotional prosodic processing. Inspection of the funnel plot (see Figure 4) suggested publication bias. However, as can be observed in Table 3 Egger's regression test did not reach significance and Orwin's fail-safe gave credence to the robustness of the observed effect. Trim and Fill did not result in an adjustment of the mean weighted ES.

The heterogeneity statistic was significant ($Q(25) = 134.201$, $p < 0.0001$). The mean weighted ES was significantly larger ($Q_B(1) = 4.15$, $p < 0.05$) for studies that presented objective evidence for the lateralized nature of the brain damage for all patients (*g =* −1.72; k = 14) than for studies that provided objective evidence for the majority of the patients or for studies for which this was unknown ($g = -0.04$; k = 12).

Funnel Plot of Standard Error by Hedges's g

Figure 3. Forest plot of the mean effect size (*g)* and 95% confidence interval for each of the studies included in the comparison of RHD to NC for emotional prosodic perception. Larger symbols indicate a larger number of subjects included in the respective study.

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3.1.3. LHD vs. RHD

A total of 22 studies (with a total of 314 LHD and 306 RHD-patients) provided sufficient data for this comparison (see studies with an LHD and RHD group in Table 1). The forest plot can be found in Figure 5. The mean weighted ES (95% CI) was -0.47 (-0.74 ; -0.20) suggesting a significantly larger effect of right hemispheric damage than left hemispheric damage on emotional prosodic processing. Inspection of the funnel plot (Figure 6) suggested publication bias but as can be observed in Table 2 Egger's regression test did not reach significance and Orwin's fail safe N supported the robustness of the observed effect. Trim and Fill identified two missing studies and led to a slight downward adjustment of the mean weighted ES (95% CI) to -0.37 (-0.66 ; −0.09).

The heterogeneity statistic was significant $(Q(21) = 58.74, p \le 0.0001)$. None of the moderators explained a statistically significant amount of variance in the ES distribution.

Figure 5. Forest plot of the mean effect size (*g)* and 95% confidence interval for each of the studies included in the comparison of LHD to RHD for emotional prosodic perception. Larger symbols indicate a larger number of subjects included in the respective study.

Funnel Plot of Standard Error by Hedges's g

Figure. 6. Funnelplot of the studies included in the comparison LHD to RHD for emotional prosodic perception. The effect size *g* of each study (x-axis) is plotted against its standard error (y-axis). The vertical line represents the mean weighted effect size and the diagonal lines the 95% confidence interval.

Comparison	Observed g $(95\% \text{ CI})$	Fail- safe N	Egger's intercep	Trim and Fill (studies added)	Adjusted g $(95\% \text{ CI})$
$LHD - NC$	$-1.06(-1.40;$	78	$-3.22*$	0	
$RHD - NC$	-0.71 $-1.41(-1.76;$	146	-2.94	θ	
LHD - RHD	-1.05 $-0.47(-0.74;$	30	-0.13		$-0.37(-0.66;$
	-0.20				-0.09

Table 2. Publication bias analyses for emotional prosody.

LHD - $NC =$ left sided damage vs. controls, $RHD - NC =$ right sided damage vs. controls, LHD - RHD = left sided damage vs. right sided damage. $*_p$ < 0.10

3.2. Linguistic prosody

An overview of the studies included in the meta-analysis of linguistic prosody perception can be found in Table 3.

3.2.1. LHD vs. NC

A total of 17 studies (with a total of 211 LHD patients and 249 NC) provided sufficient data for this comparison (see studies with a LHD and NC-group in Table 2). The forest plot can be found in Figure 7. The mean weighted ES (95% CI) was −1.05 (−1.39; −0.71) suggesting a large effect of left hemispheric damage on linguistic prosodic processing. Inspection of the funnel plot (Figure 8) suggested publication bias. As can be observed in Table 4 publication bias was confirmed by Egger's regression test but Orwin's fail-safe N gave credence to the robustness of the observed effect. Trim and Fill identified three missing studies and led to a downward adjustment of the mean weighted ES (95% CI) to −0.81 (−1.20; −0.43).

The heterogeneity statistic was significant ($Q(16) = 44.89$, $p < 0.0001$). None of the moderators explained a statistically significant amount of variance in the ES distribution.

Table 3. Overview of studies on linguistic prosody.

Study	Language	Etiology	CT/MR	$N_{\rm L}$	N_R	N_C
Weintraubet al. (1981)	English	Mixed	ς	$\qquad \qquad -$	9	10
Baumet al. (1982)	English	CVA	All	8		8
Heilmanet al. (1984)	English	CVA	Most	9	8	15
Emmoreyet al. (1987)	English	CVA	Most	15	7	15
Bryan(1989)	English	CVA	Most	30	30	30
Blonderet al. (1991)	English	CVA	All	10	10	10
Brådviket al. (1991)	Swedish	CVA	Most	\sim	20	18
Grosjean(1996)	French	Mixed	Most	10	10	20
Perkins et al.(1996)	English	CVA	All	8	8	8
Baumet al. (1997)	English	CVA	?	10	10	10
Baum et al. (1998)	English	CVA	All	12	10	10
Pell et al. (1997)	English	CVA	ς	10	9	10
Borod et al. (1998)	English	CVA	All	10	11	15
Breitenstein et al. (1998)	German	Mixed	All	16	10	10
Pell (1998)	English	CVA	All	11	9	10
Walkeret al. (2002)	English	CVA	All	8	8	8
Zgaljardic et al. (2002)	English	CVA	L: Most	7	9	7
			R: All			
Seddoh (2006)	English	CVA	All	21	\overline{a}	16
Rymarczyk et al. (2007)	Polish	CVA	All		37	26
Kho et al. (2008)	Dutch	Resection	All	16	15	47

Language = native language of the subjects; CT/MR = objective (CT/MR) imaging, or surgical-) verification of lateralized damage for all or most patients; NL = number of patients with left hemispheric damage; N_R = number of patients with right hemispheric damage; N_c = number of healthy controls.

Figure 7. Forest plot of the mean effect size (*g*) and 95% confidence interval for each of the studies included in the comparison of LHD to NC for linguistic prosodic perception. Larger symbols indicate a larger number of subjects included in the respective study.

3.2.2. RHD vs. NC

A total of 18 studies (with a total of 236 RHD patients and 271 NC) provided sufficient data for this comparison (see studies with an RHD and NC group in Table 2). The forest plot can be found in Figure 9. The mean weighted ES (95% CI) was −0.88 (−1.11; −0.64) suggesting a large effect of right hemispheric damage on linguistic prosodic processing. Inspection of the funnel plot (Figure 10) suggested publication bias. In Table 4 it can be observed that Eggers regression test confirmed publication bias but Orwin's fail-safe N supported the robustness of the observed effect. Trim and Fill identified four missing studies and led to a downward adjustment of the mean weighted ES (95% CI) to −0.73 (−0.99; −0.47).

The heterogeneity statistic failed to reach significance $(O(17) = 25.88, p > 0.05)$ suggesting a homogeneous ES distribution. None of the moderators explained a significant amount of variation in the ES distribution.

Figure 9. Forest plot of the mean effect size (*g)* and 95% confidence interval for each of the studies included in the comparison of RHD to NC for linguistic prosodic perception. Larger symbols indicate a larger number of subjects included in the respective study.

Fig. 10. Funnelplot of the studies included in the comparison RHD to NC for linguistic prosodic perception. The effect size *g* of each study (x-axis) is plotted against its standard error (y-axis). The vertical line represents the mean weighted effect size and the diagonal lines the 95% confidence interval.

3.2.3. LHD vs. RHD

A total of 14 studies (with a total of 172 LHD patients and 160 RHD patients) provided sufficient data for this comparison (see studies with an LHD- and RHDgroup in Table 2). The forest plot can be found in the Figure 11. The mean weighted ES (95% CI) was 0.12 (−0.29; 0.52) indicating a negligible (and nonsignificant-) difference between the two cerebral hemispheres in linguistic prosodic processing capability. The funnelplot (Figure 12) was symmetric. However as can be seen in Table 4 Egger's test did suggest publication bias but Trim and Fill did not identify any missing studies.

The heterogeneity statistic was significant ($Q(13) = 43.30$, $p \le 0.0001$). None of the moderator variables explained a significant amount of variance in the ES distribution.

Figure 11. Forest plot of the mean effect size (*g)* and 95% confidence interval for each of the studies included in the comparison of LHD to RHD for linguistic prosodic perception. Larger symbols indicate a larger number of subjects included in the respective study.

Figure 12. Funnelplot of the studies included in the comparison LHD to RHD for linguistic prosodic perception. The effect size *g* of each study (x-axis) is plotted against its standard error (y-axis). The vertical line represents the mean weighted effect size and the diagonal lines the 95% confidence interval.

Comparison	Observed g $(95\% \text{ CI})$	Fail- safe N	Egger's intercept	Trim and Fill (studies added)	Adjusted g $(95\% \text{ CI})$
$LHD - NC$	-1.05 (-1.39 ;	59	$-4.8*$	3	$-0.81(-1.20;$
	$-0.71)$				-0.43
RHD - NC	$-0.88(-1.11;$	58	$-2.4*$	4	$-0.73(-0.99)$;
	-0.64)				-0.47
LHD - RHD	$0.12 (-0.29;$		$3.7*$	Ω	
	(0.52)				

Table 4. Publication bias analyses for meta-analyses for linguistic prosody.

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3.3. Overview and comparison of the meta-analyses

The mean weighted ES and 85% CI under the random effects model for all six metaanalyses are presented in Figure 13. It can be observed that damage to each hemisphere compromises performance on both emotional and linguistic prosodic functions as compared to controls but that there is a non-significant trend for emotional prosody to be more disturbed following RHD than LHD while the reverse holds for linguistic prosody. Furthermore RHD compromises emotional prosodic processing more than linguistic prosodic processing. Finally, when comparing RHD to LHD directly there is evidence for right-hemispheric specialization for emotional prosody but no clear hemispheric specialization for linguistic prosodic processing.

Figure 13. The mean weighted effect size *g* (y-axis) with 85% CI is shown for each of the six meta-analyses (x-axis). It can be observed that both left hemispheric damage (LHD) and right hemispheric damage (RHD) compromises both emotional (circles) and linguistic (triangles) prosodic perception as compared to normal controls (NC). Furthermore, RHD degrades emotional processing more than linguistic processing. Lastly, RHD compromises emotional prosodic perception as compared to LHD while there is no significant difference between these groups for linguistic prosodic perception.

3.4. Qualitative mini-review of studies reporting insufficient data

A total of seven studies did not report enough statistics to compute Hedges's *g* but did fulfill the other inclusion criteria. To give a complete review of the literature on hemispheric specialization for prosodic perception, these studies will now be briefly reviewed.

3.4.1. Emotional prosody

Four studies used emotional prosodic perception tasks. Cancelliere and Kertesz (1990) focused on the influence of the intrahemispheric location of brain damage on emotional processing. Semantically neutral sentences that had been pronounced in happy, sad, angry, or neutral prosody were presented to 28 RHD, 17 LHD patients, and 20 NC. Participants were requested to identify the prosodic category. Because the focus was on *intra*hemispheric damage, the two lateralized damaged groups were not compared directly. Using the lesion-overlap method, the authors concluded that the basal ganglia are important for prosodic perception (irrespective of the side of the lesion).

Starkstein, Federoff, Price, Leiguarda and Robinson (1994) examined the effects of acute lateralized brain damage on emotional prosodic perception. A total number of 59 patients (numbers of LHD or RHD patients unclear) and 17 NC were presented with two prosodic perception tasks. For the first task, semantically neutral sentences were intoned in happy, sad, or angry prosody and participants were asked to identify the prosodic category. In a second task, the instructions were the same but the sentences contained emotional semantics that was either congruent or incongruent with the prosodic layer of the utterance. For the first task, LHD and RHD groups were not directly compared but for the second task the authors reported worse performance for RHD as compared to LHD.

Weddell (1994) investigated the effect of cortical and subcortical damage on emotional processing. Ten patients with damage to the wall or floor of the third ventricle (hypothalamus), 27 RHD and 24 LHD patients (which included cortical and subcortical lateralized damage) and 15 controls with spinal lesions had to identify the prosodic category (happy, sad, angry, surprised or neutral) of semantically neutral or congruent utterances. The authors reported poorer performance for the RHD group as compared to the LHD group but only when emotion was expressed solely through prosody. Furthermore, basal ganglia damage (irrespective of the side of the lesion) impaired emotional prosodic processing. The authors concluded that the bilateral basal ganglia are important for emotional perception.

Zaidel et al. (2002) administered an affective prosodic perception test to 23 RHD, 12 LHD patients and 21 NC among a set of tasks that tapped pragmatics. Participants were requested to identify the emotional prosodic category (happy, sad, angry, neutral) of semantically neutral sentences. Both brain damaged groups performed worse than NC but the authors did not report a significant difference between the brain damaged groups. The authors concluded that there is no clear hemispheric specialization for pragmatic aspects of language.

3.4.2. Linguistic prosody

Three studies used linguistic prosodic perception tasks. Aasland and Baum (2003) investigated the sensitivity of LHD and RHD patients to durational cues in determining phrasal boundaries. The duration of pauses and of pre-boundary words in the utterance "Pink and black and green" was systematically varied to obtain different phrase structures. In two experiments, 10 LHD, 9 RHD patients, and 10 NC were asked to identify the phrase structure by pointing to a picture that depicted the correct grouping of constituents (colors). This design allowed the authors to parametrically investigate the interaction of durational cues and lateralized hemispheric damage on phrasal boundary processing. Overall, both patient groups performed worse than NC in identifying phrasal boundaries, but the LHD group showed improved performance when temporal cues were exaggerated while the RHD group did not. The authors concluded that while a heightened temporal cue threshold might be responsible for the deficiency in phrasal boundary processing for the LHD group, the exact mechanism for the deficit in RHD group is unclear.

Abada et al. (2006) investigated whether the left and right hemisphere are differentially sensitive to metrical stress. Monosyllabic words (such as 'mint') were embedded in disyllabic nonwords where the second syllable was either weak (such as in "mintef") or strong (such as in "mintayf"). Previous research had shown that detection of the proper word is faster when the nonsense syllable is weak as compared to a strong nonsense syllable as there is a tendency to attempt lexical access at strong syllables. Groups of 10 LHD, 10 RHD patients and 10 NC were required to press a button as they detected a proper word. All three groups showed the strong syllable effect but overall the LHD group performed worse than NC while RHD patients did not differ from the other two groups which led the authors to conclude that LHD induces difficulties utilizing stress patterns.

Seddoh (2006b) presented meaningful and nonsense sentences that were intonated as statements or questions to 13 LHD, 8 RHD patients and 12 NC. Participants were asked to identify whether the utterance was a question or a statement. Both patient groups performed worse than the control group but only for utterances with question intonation. Seddoh suggested that the patients might have had difficulties in processing specific components of the pitch contour.

3.4.3. Conclusion of the mini-review

Taken together the studies on emotional prosodic perception indicate bilateral processing of emotional prosody with subcortical involvement and a possible relative right hemispheric advantage. This is in line with the quantitative analysis of emotional prosodic perception studies. The small number of studies on linguistic prosody generally points to bilateral processing, which is also in accordance with the quantitative analysis.

3.5. Explorative intrahemispheric analysis

Table 5 presents the percentage of patients with damage to intrahemispheric locations for each study. To explore a possible differential impact of temporal versus frontal damage on prosodic processing performance, a weighted least squares random effects meta-regression analysis was performed with the percentage of patients with temporal lobe damage and the percentage of patients with frontal lobe damage as predictors and the Fisher's *Z* transformed ES as the dependent variable. For none of the meta-analyses percentage temporal or percentage frontal damage explained a significant amount of variance in the effect size distribution (for all: $-0.3 < \beta < 0.7$, $p > 0.05$). A series of independent t-tests revealed no difference between the LHD and RHD groups in the

percentage of patients with subcortical damage or white matter lesions for any of the meta-analyses (for all: $-0.71 \le t \le 0.78$, $p > 0.05$).

Study	Used in comparison	Patient group	White matter	Sub-cortical	Temporal	Frontal
Weintraubet al. (1981)	Linguistic	Right	θ	11.11	66.67	22.22
Baum et al. (1982)	Linguistic	Left	5.	?	?	
Heilman et al. (1984)	Linguistic+	Right	$\overline{0}$	$\boldsymbol{0}$	$\overline{0}$	50.00
	Emotional	Left	θ	θ	22.22	55.56
Tomkins et al. (1985)	Emotional	Right	?	?	?	?
		Left	5.	?	ć.	?
Bowers et al. (1987)	Emotional	Right	Ç	P	ς	5.
		Left	5.	Ç	ς	ç
Emmorey et al. (1987)	Linguistic	Right	θ	28.57	14.28	28.57
		Left	θ	θ	6.67	60.00
Bryan(1989)	Linguistic	Right	θ	$\boldsymbol{0}$	36.67	26.67
		Left	θ	θ	23.33	13.33
Blonder et al. (1991)	Linguistic+	Right	θ	θ	60.00	60.00
	Emotional	Left	θ	$\boldsymbol{0}$	80.00	50.00
Brådvik et al. (1991)	Emotional	Right	5.	5.	?	?
Lalande et al. (1992)	Linguistic+ Emotional	Right	5	ς	?	P
Van Lancker (1992)	Emotional	Right	?	?	ς	P
		Left	5	ċ	ς	ς
Grosjean (1996)	Linguistic	Right	20.00	30.00	60.00	60.00
		Left	θ	70.00	50.00	70.00
Hornak et al. (1996)	Emotional	Right	9.09	27.27	27.27	72.72
		Left	50.00	θ	θ	50.00
Perkins et al.(1996	Linguistic	Right	12.50	25.00	62.50	62.50
		Left	25.00	37.50	50.00	50.00
Pell et al. (1997)	Linguistic	Right	?	?	ç.	?
	$+$	Left	5.	5.	ć.	5.
	Emotional					
Baum et al. (1997)	Linguistic	Right	20.00	0	20.00	0
		Left	θ	$\overline{0}$	10.00	40.00
Peper et al.(1997)	Emotional	Right	?	?	?	?
		Left	Ç	5.	5.	5

Table 5.Percentage of patients with damage to intrahemispheric loci for each study.

4. Discussion

The current meta-analyses suggest that both cerebral hemispheres are necessary for adequate explicit emotional and linguistic prosodic perception. Within Cohen's (1988) framework for qualifying effect size, the detrimental effect of both RHD and LHD (as compared to NC) on both linguistic and emotional prosodic perception performance can be considered large (i.e., *g* was larger than 0.80 for all comparisons). However, when comparing the detrimental effect of LHD and RHD (as compared to NC), it was shown that while LHD degrades emotional and linguistic prosodic perception to a similar degree (−1.06 and −1.05 respectively) RHD degraded emotional prosodic perception performance more $(g = -1.41)$ than linguistic prosodic perception performance $(g = -0.88)$. Finally, when comparing both patient groups directly, RHD degraded emotional prosodic processing more than LHD ($g = -0.47$) while there was no differential degradation of lateralized brain damage for linguistic prosodic perception performance. These results are therefore compatible with the notion of bihemispheric control over the perception of linguistic and emotional prosody with a *relatively* greater contribution of the right hemisphere to emotional prosodic perception.

Strong versions (absolute lateralization) of the "categorical" lateralization hypotheses mentioned in the introduction can therefore not be supported. The results of the present study clearly do not support even a weak version of global control of the right hemisphere over *all* prosodic perception (Klouda et al., 1988) since LHD compromised linguistic prosodic perception performance to a comparable degree as RHD did. A relative version of the Functional lateralization hypothesis (Van Lancker, 1980) can not be supported either. Although RHD compromised emotional perception more than LHD no significantly larger performance degradation for LHD than RHD was found for linguistic prosodic processing, as would have been predicted by this hypothesis. However, our results are compatible with a weak version of the right hemisphere hypothesis (Ross, 1981; Blonder et al., 1991; Borod et al., 1998) where there is bilateral processing for emotional prosodic perception but with larger right than left hemispheric contribution. As discussed in the introduction, the current design does not permit us to differentiate between the "categorical" and cue-dependent lateralization hypotheses of prosodic perception. Hence, it is unclear whether the right hemispheric superiority for emotional prosodic processing originates in superior processing of acoustics necessary for adequate emotional prosodic perception (Van Lancker et al., 1992) or superiority in processing of emotional prosodic categories (in partial support of the Functional lateralization hypothesis) or both (for an informative discussion of this issue see Pell, 1998).

Another important line of evidence regarding hemispheric specialization for prosodic perception is provided by the neuroimaging literature. Activation maps of fMRI/PET studies are highly dependent on the exact experimental and control condition (i.e. contrast) used. For instance, contrasting emotional versus linguistic decisions for the same prosodic material in a categorization task is mainly sensitive to later stages of the prosodic perception process (identification of emotional or linguistic information), whereas contrasting emotional prosody to neutral prosody additionally taps into earlier stages (acoustic analysis). Hence, comparing neuroimaging studies is not without difficulty. With this caveat in mind, imaging studies to date have found bilateral temporofrontal (STG/STS, IFG) activations for emotional (Buchanan et al., 2000; Mitchell, Elliott, Barry, Cruttenden, & Woodruff, 2003; Kotz et al., 2003;

Grandjean et al., 2005; Ethofer et al., 2006a; Ethofer et al., 2006b; Mitchell & Ross, 2008; Ethofer et al., 2009; Ethofer, Van de Ville, Scherer, & Vuilleumier, 2009) and linguistic (Gandour et al., 2003; Wildgruber et al., 2004; Doherty, West, Dilley, Shattuck-Hufnagel, & Caplan, 2004; Humphries, Love, Swinney, & Hickok, 2005; Aleman et al., 2005) prosodic perception tasks. Some of these imaging studies have additionally found more (extended) right than left hemispheric activation for emotional prosodic processing (Buchanan et al., 2000; Mitchell et al., 2003; Ethofer et al., 2006a; Ethofer et al., 2009) while for the limited number of studies that used linguistic prosodic perception tasks, the results are mixed with some finding more (extended) activation in the left (Wildgruber et al., 2004; Aleman et al., 2005) and others in the right (Gandour et al., 2003; Doherty et al., 2004) hemisphere. These studies therefore suggest that there is a bilateral temporofrontal network of areas involved in the perception of linguistic and emotional prosody with possibly a relative right hemispheric superiority for emotional prosodic perception. The results of our metaanalysis are clearly in keeping with the neuroimaging literature and additionally suggest that within this bilateral network of sufficient areas both hemispheres are also *necessary* for the perception of prosody. Moreover, our results suggest that the stronger activation of the right hemisphere found by some neuroimaging studies might reflect relative superiority of the right hemisphere in the perception of emotional prosody. Our explorative meta-regression analysis of intrahemispheric contributions to prosodic perception did not reveal a differential impact of temporal versus frontal damage on prosodic perception performance. However, due to the indirect nature of this analysis this result should not be taken as conclusive.

Although we tested in multiple ways whether the 'verbal-articulatory demands' of prosodic perception tasks (Ross et al., 1997) moderated the effect of lateralized brain damage on prosodic perception performance, we failed to find such effects. Increased verbal demands on prosodic tasks (such as the presence of lexical semantics or the need to respond verbally) did not increase the effect of left hemispheric damage on emotional perception performance. However, one moderating variable yielded a statistically robust effect. Studies that provided objective evidence that brain damage was indeed right lateralized for all patients, found a significantly larger effect of RHD on emotional prosodic perception performance than studies that did not provide such evidence for all patients. It is therefore possible that in the latter case patients with left or bilateral brain damage had been included, which might have decreased the ES (and hence the reported mean weighted ES for the effect of RHD on emotional prosodic perception reported here might represent an underestimation). A clear recommendation for future studies, then, is that presumed lateralized brain damage for all patients should be radiologically confirmed in order to get an accurate measure of the effect of lateralized brain damage.

There were some limitations to the present investigation. First, the study design does not permit us to conclude that the observed division of labor between the hemispheres is *specific* to prosodic processing as we have not included non-prosodic control tasks. Hence, although we cannot exclude the possibility that the results might generalize to linguistic and emotional processing in other modalities, we can conclude that the results are *at least* valid for prosodic processing. A second and related issue that has already been pointed out is that the current design does not permit us to differentiate cue-dependent versus "categorical" lateralization (which might be modality-independent) explanations of the right hemispheric advantage for emotional

prosodic processing. Thirdly, to maximize sensitivity of our meta-analysis to hemispheric specialization we were forced to pool effect sizes over the levels of moderators. This may have decreased the power of our moderator analyses so that the (absence of) effects in our moderator analysis should not be taken as conclusive. Fourth, an often mentioned critique on meta-analysis is that it quantitatively combines studies that are incomparable and therefore should not be combined. In this light, it might be possible that different linguistic prosodic functions follow a separate pattern of hemispheric lateralization and that the net bilateral control that was found in the current meta-analysis reflects this heterogeneity in hemispheric specialization. Unfortunately, the number of studies per prosodic linguistic phenomenon was too small for a statistically robust test of this issue leaving this a matter for future investigation. Finally, although we focused on cortical lateralization of prosodic perception, many of the studies that were analyzed in the current meta-analysis included patients with cortical *and* subcortical damage. Therefore, we cannot rule out a contribution of subcortical structures to the observed pattern of hemispheric involvement in prosodic perception (indeed many studies have suggested involvement of subcortical structures in prosodic processing such as the basal ganglia, see e.g. Cancelliere & Kertesz, 1990; Blonder, Gur, & Gur, 1989). Hence, when assessing the effect size of lateralized brain damage on prosodic perception performance as compared to controls in isolation it should be kept in mind that the effect likely reflects both cortical, subcortical, and white matter damage. However, as it seems likely that subcortical involvement was present in the right and left hemisphere to a similar degree (which was confirmed by the analysis of the percentage of patients with subcortical damage as reported in each paper) this does not complicate our comparisons of left with right hemispheric damage.

In sum, we conclude that the network of necessary areas dedicated to the perception of linguistic and emotional prosody is *bilateral* with only *relative* right hemispheric specialization for emotional prosodic perception at best.

5. References

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