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**Behavioral and electrophysiological study of binaural hearing
in patients with unilateral cerebrovascular accident**

**by
Tony Leroux, B.Sc./M.Sc.**

**A thesis submitted to the Faculty of Graduate Studies
in partial fulfillment of
the requirements for the degree of**

Doctor of Philosophy

**Carleton University
Ottawa, Ontario
January 11th, 1999**

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Acceptance of the thesis:

*"Behavioural and electrophysiological study of binaural hearing in patients with
unilateral cerebrovascular accident"*

submitted by

Tony Leroux

in Partial fulfillment of the requirements for
the degree of Doctor of Philosophy

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Carleton University
December, 1998

Abstract

The purpose of this study was to assess the potential of a new clinical procedure, based on sound localization, to identify central auditory dysfunction in patients with unilateral cerebrovascular accident (cva). Left hemispheric cerebrovascular accident often leads to aphasia. Aphasia is a communication disorder involving receptive and expressive language impairment. A lack of sensitive, valid and reliable clinical tests has made difficult the identification of the underlying components of speech comprehension problems related to aphasia. A large body of animal and human literature has demonstrated that various sound localization abilities are also impaired by cortical lesions. To document this non-linguistic auditory ability, binaural masking procedures were examined along with some of the more traditional speech tests to see how well they detect central nervous system disorders. Twenty-two patients with right or left unilateral cva, the experimental group, were tested using a binaural masking task employing behavioral and electrophysiological approaches. The results obtained from the experimental group were compared with those from a normal control group matched in age and hearing sensitivity. The relationship between the speech comprehension impairment and the performance on the binaural masking tasks was also studied. The severity of the speech comprehension problem was found to have a highly significant relationship to the performance of the patients on the behavioral task.

Acknowledgments

In 1994, I joined the brand new Audiology and Speech-Language Pathology Program of the University of Ottawa, chaired at that time by Dr. Andre Durieux-Smith. Dr. Durieux-Smith consistently strove to provide the new teaching staff with an adequate milieu which would support the development of a research career. To this end, she introduced me to Dr. Jack Kelly from Carleton University. This has been a great initiative and I owe her this first contact with Dr. Kelly. Since then, and over the last 4 years, Dr. Kelly has been a patient advisor and an invaluable resource available to discuss research methods and results. I have learned with him that simple presentation of data is a complex task, but a powerful exercise for the advancement of scientific knowledge. This is an esteemed gift that I will attempt to share with my own students. I am also indebted to the many people who were willing to listen, sometime for hours, to boring clicks and white noise. These people were students, colleagues, friends, family members and patients from the General site of the Ottawa Hospital. The Faculty of Health Sciences of the University of Ottawa supported my doctoral studies by an administrative workload reduction during one full year. This year was, in fact, one of the most productive. Thanks also to Caryn Bursch who enthusiastically accepted the task of revising drafts of this manuscript. Finally, my most important support came from my family. My wife Caroline who has taken during these years more of her share of the family workload. She listened and supported me through the unavoidable tough times. Caroline, you deserve a Ph.D. as much as I do. The little ones, Charles, Thomas and Batrice, you were the smiles, the laughs and the warmth of love that kept me going.

Table of contents

1.	Introduction.....	1
2.	Review of the literature.....	7
2.1.	<i>Speech comprehension deficits in aphasia.....</i>	<i>7</i>
2.1.1.	Assessment of the severity of the speech comprehension deficit using standardized tests	8
2.1.1.1.	Severity of the speech comprehension problem versus extent of the lesion and recovery	12
2.1.2.	Auditory capabilities and speech comprehension deficit.....	14
2.1.2.1.	Synthesis of the research on auditory capabilities and speech comprehension deficits	22
2.2.	<i>A sound localization approach in the identification of central auditory processing problems</i>	<i>24</i>
2.2.1.	Sound localization deficits and lesions of the central auditory system	24
2.2.1.1.	Brain stem site-of-lesion	25
2.2.1.2.	Distinctions between sub-cortical and cortical site-of-lesion.....	25
2.2.1.3.	Cortical site-of-lesion.....	28
2.2.1.4.	Corpus callosum lesions	33
2.2.1.5.	The effect of aging on sound localization abilities.....	35
2.2.1.6.	The effect of attention on sound localization performance	38
2.2.1.7.	Electrophysiologic means to assess localization abilities	41
2.3.	<i>Sound localization deficit in aphasic patients.....</i>	<i>46</i>
2.4.	<i>Synthesis of the review of literature</i>	<i>48</i>
2.5.	<i>Preliminary results with normal subjects using a binaural masking approach.....</i>	<i>50</i>
2.5.1.	Behavioral approach.....	50
2.5.2.	Electrophysiological approach.....	52
3.	Objective and Hypothesis.....	58
4.	Methodology.....	59
4.1.	<i>Subjects</i>	<i>59</i>
4.1.1.	Auditory status of the experimental subjects	62
4.2.	<i>Methods.....</i>	<i>62</i>
4.2.1.	Rationale applied to the development of the binaural masking procedures.....	63
4.2.2.	Behavioral procedure.....	65
4.2.3.	Electrophysiological procedure	72
4.2.4.	Speech comprehension measures.....	77
4.3.	<i>Data analysis</i>	<i>79</i>
5.	Results.....	82
5.1.	<i>Results from the behavioral procedure</i>	<i>82</i>
5.1.1.	Probability of signal detection	82
5.1.1.1.	Data reduction approach	82
5.1.1.2.	Results for the normal group.....	84
5.1.1.3.	Results for the left brain-damaged group.....	88
5.1.1.4.	Results for the right brain-damaged group.....	91

5.1.2.	Reaction time.....	95
5.1.2.1.	Data reduction approach.....	95
5.1.2.2.	Effect of the perceived spatial position of the signal on the RT	96
5.1.2.3.	Effect of the SNR on the RT.....	98
5.1.2.4.	Effect of the interaction of the SNR and the SITD on the RT	100
5.2.	<i>Results from the electrophysiological procedure</i>	107
5.2.1.	Data reduction approach.....	107
5.2.2.	Results of the initial analysis	109
5.2.2.1.	Amplitude	109
5.2.2.2.	Latency	112
5.2.3.	Interaction of the SNR and the SITD on N ₁ -P ₂ amplitude	118
5.2.3.1.	Results for the normal group.....	118
5.2.3.2.	Results for the left brain-damaged group	122
5.2.3.3.	Results for the right brain-damaged group.....	125
5.3.	<i>Results of the speech comprehension measures</i>	128
5.4.	<i>Results of the statistical analyses</i>	132
5.4.1.	Difference between groups on sound localization measures.....	132
5.4.2.	Relationship between behavioral and electrophysiological methods.....	134
5.4.3.	Relationship between sound localization-related and speech comprehension measures.....	134
6.	Discussion	137
6.1.	<i>Identification of deficits in central auditory processing in left brain-damaged aphasic patients</i>	139
6.2.	<i>Identification of deficits in central auditory processing in right brain-damaged non-aphasic patients</i>	143
6.3.	<i>Speech comprehension deficits and relation to change in performance on binaural masking procedures</i>	146
6.4.	<i>Limitations of the study</i>	147
7.	Conclusion	149

List of Tables

Table 1 - Identification of the site-of-lesion for subjects in the left and right mca cva groups

Table 2 - Mean pure-tone average hearing thresholds (dB HL) measured at the right and left ear for the normal, left and right brain-damaged groups

Table 3 - Example of the organization of the initial pre-testing block

Table 4 - Example of the organization of the data collection block

Table 5 - Parameters used for electrophysiological data collection

Table 6 - Example of the organization of the electrophysiological data collection block

Table 7 - Group means for the 4 dependent variables used in the MANOVA

List of Figures

- Figure 1 - Example of a Boston Diagnostic Aphasia Examination (BDAE)
- Figure 2 - Decision tree provided by the Western Aphasia Battery (WAB) to classify aphasias
- Figure 3 - Detection probability of a click signal in a binaural masking noise at 3 interaural time differences
- Figure 4 - Mean reaction time for the detection of a binaural target signal as a function of the interaural time difference for the signal at 3 signal-to-noise ratios ($n = 4$)
- Figure 5 - Mean latency values obtained for cortical responses P_1 , N_1 , P_2 , N_2 measured over left hemisphere ($n = 3$)
- Figure 6 - Mean amplitude values obtained for cortical responses P_1 - N_1 , N_1 - P_2 , P_2 - N_2 measured over left hemisphere ($n = 3$)
- Figure 7 - Schematic description of the TDT instrumentation used in the behavioral procedure
- Figure 8 - Description of the physical characteristics of the target signal and the masking noise and, their temporal relationship
- Figure 9 - Schematic description of the TDT instrumentation used in the electrophysiological experiment
- Figure 10 - Averaged probability of detection as a function of the interaural time difference for the signal (SITD) for the normal group ($n = 10$)
- Figure 11 - Overall probability of signal detection and index of binaural advantage as a function of the signal-to-noise ratio for the normal group ($n = 10$)
- Figure 12 - Averaged probability of detection as a function of the SITD for the left lesion group ($n = 9$)
- Figure 13 - Overall probability of signal detection and index of binaural advantage as a function of the SNR for the left lesion group ($n = 9$)
- Figure 14 - Averaged probability of detection as a function of the SITD for the right lesion group ($n = 10$)
- Figure 15 - Overall probability of signal detection and index of binaural advantage as a function of the SNR for the right lesion group ($n = 10$)

Figure 16 - Group mean reaction time as a function of the SITD

Figure 17 - Group mean RTs as a function of the signal-to-noise ratio

Figure 18 - Mean RT measured for the detection of the signal for 3 spatial positions as a function of the SNR for the normal group ($n = 10$)

Figure 19 - Mean reaction RT measured for the detection of the signal for 3 spatial positions as a function of the SNR for the left lesion group ($n = 9$)

Figure 20 - Mean reaction RT measured for the detection of the signal for 3 spatial positions as a function of the SNR for the right lesion group ($n = 10$)

Figure 21 - Overall amplitude of the N_1 - P_2 component measured at the C_z site as a function of the SNR for the 3 groups

Figure 22 - Overall amplitude of the N_1 - P_2 component measured at the F_z site as a function of the SNR for the 3 groups

Figure 23 - Overall latency of the N_1 component measured at the C_z site as a function of the SNR for the 3 groups

Figure 24 - Overall latency of the P_2 component measured at the C_z site as a function of the SNR for the 3 groups

Figure 25 - Overall latency of the N_1 component measured at the F_z site as a function of the SNR for the 3 groups

Figure 26 - Overall latency of the P_2 component measured at the F_z site as a function of the SNR for the 3 groups

Figure 27 - Mean amplitude of the N_1 - P_2 component measured at the C_z site as a function of the SNR at 3 perceived spatial locations for the normal group ($n = 10$)

Figure 28 - Mean amplitude of the N_1 - P_2 component measured at the F_z site as a function of the SNR at 3 perceived spatial locations for the normal group ($n = 10$)

Figure 29 - Mean amplitude of the N_1 - P_2 component measured at the C_z site as a function of the SNR at 3 perceived spatial locations for the left lesion group ($n = 9$)

Figure 30 - Mean amplitude of the N_1 - P_2 component measured at the F_z site as a function of the SNR at 3 perceived spatial locations for the left lesion group ($n = 9$)

Figure 31 - Mean amplitude of the N_1 - P_2 component measured at the C_z site as a function of the SNR at 3 perceived spatial locations for the right lesion group ($n = 10$)

Figure 32 - Mean amplitude of the N_1 - P_2 component measured at the F_z site as a function of the SNR at 3 perceived spatial locations for the right lesion group ($n = 10$)

Figure 33 - Group means for the 3 speech comprehension measures

Figure 34 - Group centroids on canonical discrimination functions and distribution of the cases

List of Appendices

Appendix A - Individual data / Probability of signal detection as a function of the SNR

Appendix B - Individual data / Reaction time as a function of the SNR

Appendix C - Individual data / Electrophysiological waveforms as a function of the SITD

Appendix D - Individual data / Scores on the speech comprehension tests

Chapter One

1. Introduction

Aphasia is a communication disorder that is related to a stroke or a traumatic injury occurring in the left hemisphere of the brain. Depending on the site of the lesion and its extent, the aphasic patient can present with a speech comprehension problems. At least 60 to 70 % of all aphasic patients present some degree of deficit in speech comprehension (Boller, Kim & Mack, 1977). Because of the high prevalence of this dysfunction, the assessment of speech comprehension is an essential part of most standardized aphasia batteries (see for example: the Boston Diagnostic Aphasia Examination (BDAE) (Goodglass & Kaplan, 1983), and the Western Aphasia Battery (WAB) (Kertesz, 1982). Typically in these diagnostic batteries, the patient is tested with verbal material presented live by the speech-language pathologist.

Standardized aphasia tests contain linguistic material and requests that vary in complexity from pointing to a specific picture representing a single word, to more complex demands using long sentences or short texts. Using these tests, the severity of the speech comprehension problem is determined solely from the specific level of linguistic complexity that the aphasic patient can or cannot successfully understand (Davis, 1993). Within this linguistically oriented framework, the possibility that the left-hemisphere lesion could also involve central auditory dysfunction is under examined (Divenyi & Robinson, 1989). Central auditory dysfunction such as reduced temporal resolution and impaired duration discrimination could also contribute to the severity of the speech comprehension problems (Schwartz & Tallal, 1980; Divenyi & Robinson, 1989; Thompson & Abel, 1992). Therefore, the patient performance on a speech comprehension task alone might not be an appropriate index of the severity of the problem.

In an integrated information processing model of auditory comprehension, McNeil and Kimelman (1986) suggested that observed speech comprehension deficits in aphasic patients could be secondary to primary deficits in the auditory processing of the rapid variations of the intensity, frequency and duration dimensions of speech sounds. Supporting this, there is evidence that some auditory non-linguistic capabilities are impaired in aphasic individuals and in dysphasic children (Lackner & Teuber, 1973; Tallal & Piercy, 1973a, 1973b; Tallal & Piercy, 1974; Tallal & Newcombe, 1978; Divenyi & Signoret, 1980; Schwartz & Tallal, 1980; Divenyi & Robinson, 1989; Thompson & Abel, 1992; Abel, Kimelman & Cohen, 1996). The affected auditory function are said to include, but might not be restricted to, frequency discrimination, frequency selectivity, discrimination of rapid frequency transition, duration discrimination, temporal resolution, temporal order discrimination, and detection of tone within broadband noise.

Divenyi and Robinson (1989) studied the relationship between non-linguistic auditory function and impairment of linguistic comprehension in aphasic patients. Their results revealed an orderly deterioration of 4 out of 7 examined auditory abilities as the level of language comprehension decreased in aphasic patients. Such a relationship suggests that specific central auditory dysfunction may parallel or even underlie speech comprehension problems. These findings also support the idea that even a mild impairment in central auditory functions may aggravate the genuinely linguistic impairment of an aphasic patient (Divenyi & Robinson, 1989). Therefore, a rigorous clinical assessment of the origin and severity of a speech comprehension problem would have to include both linguistic and auditory aspects (Musiek, Baran & Pinheiro, 1994; Phillips, 1995).

Aphasic patients presenting symptoms of hearing loss, or auditory-related complaints are typically sent to audiologists for evaluation. In most cases, the clinical evaluation will be limited to the peripheral abilities such as hearing acuity determined by an audiogram. More rarely, the central auditory abilities will also be assessed (Musiek et al., 1994). Central auditory capabilities are typically evaluated using sensitized speech

tests (Katz, 1962; Speaks & Jerger, 1965), temporal pattern recognition tests (Katz & Pack, 1975; Pinheiro, 1977; Musiek, Baran & Pinheiro, 1990) and auditory evoked response tests (Hall, 1992; Musiek et al., 1994; McPherson, 1996).

This global clinical approach, while attempting to describe the central auditory capabilities, presents severe limitations. First, the speech material used in sensitized tests has been criticized on the basis of its questionable validity and reliability (Huard, 1992; Amos & Humes, 1998). These tests were primarily designed to identify neurological site-of-lesions and are now being used to assess central auditory capabilities (Bamford & Saunders, 1991). This shift in the purpose of testing has also brought a change in the target population from brain-damaged adults to learning-disabled children. The above cited reasons cause clinicians and researchers to denounce openly a lack of standardization of these sensitized speech tests. In addition, a lack of control of linguistic bias often appears in these tests (Noffsinger & Kurdziel, 1979). For example, the vocabulary found in tests, first designed for brain-damaged adults, was not adapted when the tests began to be used with children. As well, with aphasic patients, the linguistic content of these procedures obscures the identification of specific central auditory processing problems. Second, the few non-linguistic tests that are available for clinical evaluation of aphasic patients are limited to identification of frequency or duration patterns, and identification of environmental sounds among background noise (Katz & Pack, 1975; Pinheiro, 1977; Musiek, Baran & Pinheiro, 1990). The relationship between the results of these non-linguistic tests and language processing is tenuous (Gollegly & Musiek, 1993; Colson, Robin & Luschei, 1990). Finally, the use of auditory evoked potentials in clinical settings is too often limited to site-of-lesion identification and infrequently directed to dysfunction assessment (McPherson, 1996). Under very strict technical and instrumental controls, auditory event-related potentials and mismatch negativity can be used to assess specific auditory capabilities such as frequency, intensity, or duration discrimination (Kraus, McGee, Carrell & Sharma, 1995; Näätänen, 1995; Näätänen & Alho, 1995). However, such sophisticated techniques are infrequently encountered and rarely used in clinical settings.

The exclusion or the identification of a central auditory processing disorder in an aphasic patient is of crucial importance in the selection of an appropriate rehabilitation strategy. On one hand, the current state of audiological practice and the lack of sensitive, valid and reliable tests prevents the identification of the precise origin of the comprehension problem in aphasic patients. On the other hand, the non-specific auditory component of a speech comprehension deficit is, most of the time, ignored by speech-language pathologists. As pinpointed by Divenyi and Robinson (1989), this void is so pronounced that no treatment has been designed so far to explicitly address auditory processing deficits in aphasic patients.

In this context, animal and human research have demonstrated that sound localization might be an appropriate vehicle for the evaluation of the central auditory system (Hochster & Kelly, 1981; Jenkins & Masterton, 1982; Jenkins & Merzenich, 1984; Al'tman, Rosenblum & Lvova, 1987; Cornelisse & Kelly, 1987; Kavanagh & Kelly, 1987; Cranford, Boose & Moore, 1990; Al'tman & Vaitulevich, 1993; Phillips, 1993; Poirier, Miljourns, Lassonde & Lepore, 1993; Poirier, Lassonde, Villemure, Geoffroy & Lepore, 1994; Zatorre, Ptito & Villemure, 1995). In animal studies, there is a large consensus that unilateral lesions of the primary auditory cortex cause a significant free-field sound localization deficit in the auditory hemifield contralateral to the lesioned hemisphere. In the same manner, human studies also suggest that cortical lesions disrupt sound localization performance. In addition, sound localization capabilities seem to be used, along with other cognitive mechanisms such as selective attention, in a non-optimal acoustical environment. An example of this occurs when a listener has to focus on one conversation while surrounded by background conversation (Soli, Nilsson & Gelnett, 1996). This effect, known as the cocktail party effect (Cherry, 1953), seems to rely on the ability of the central auditory system to achieve this by assigning a specific spatial channel to the desired speech source of information.

In a listening environment of multiple, simultaneous, speakers, voices come from various positions in space. Sound localization ability is involved in the selection of the desired voice, coming from a specific position in space, within a spatial channel

(Middlebrooks & Pettigrew, 1981; Rajan, Aitkin, Irvine & McKay, 1990). Other sounds coming from other surrounding positions in space, would be processed by different spatial channels. Segregated in this manner, the pertinent auditory information (figure) and the non-pertinent information (ground) are separated, helping to maintain the salience of the desired speech source for the linguistic central processor. An impairment in the spatial channel mechanisms would lead to difficulties in the identification and comprehension of the stream of language, even in weak background noise.

The non-linguistic nature of sound localization, and its well demonstrated impairment following cortical lesions, suggests that a test of sound localization has potential to be a useful clinical instrument to identify aphasic patients with deficits in central auditory processing. Although the unique central properties of sound localization are now well recognized, there is currently no available standard clinical test for sound localization deficits. The present work examines the potential of a clinical procedure, based on sound localization, to identify central auditory dysfunction in patients with temporal lobe damage and to compare the outcome with tests of speech comprehension.

The successful application of a sound localization paradigm to identify central auditory dysfunction in patients with temporal lobe damage could lead to changes in the focus of the therapy used in speech-language pathology. The sound localization paradigm is obviously not a speech comprehension test. Nevertheless, a deficit in sound localization abilities would demonstrate the presence of an auditory dysfunction that could coexist with the linguistic dysfunction. The simultaneous presence of these dysfunctions might influence the severity of a demonstrated speech comprehension problem. Patients with temporal lobe damage suffering from a central auditory dysfunction, as revealed by a deficit on a sound localization procedure, might benefit less from a treatment that emphasizes exclusively a linguistic approach. A therapeutic approach that includes attention to *both* the central auditory dysfunction and to the linguistic aspects of the speech comprehension deficit could probably improve the functional outcome for these patients as compared to the exclusively linguistic approach. Otherwise, brain-damaged patients that have normal central auditory processing while

showing a speech comprehension deficit would still benefit from the usual treatment focused on a linguistic-based therapeutic approach. The above examples suggest that the successful application of a sound localization paradigm to identify central auditory dysfunction in patients with temporal lobe damage could lead, over the long term, to an integrated battery of clinical tests and therapeutic approaches. The availability of these tests and approaches would allow the speech-language pathologist and the audiologist to delineate the contribution of auditory and linguistic underlying components to an observed speech comprehension problem and to select the most effective therapeutic approach.

Chapter Two

2. Review of the literature

2.1. Speech comprehension deficits in aphasia

There is a large body of evidence suggesting that lesions in the left posterior superior temporal lobe are associated with deficits in speech comprehension in aphasic patients (Wernicke, 1908; DeRenzi & Vignolo, 1962; Hecaen & Angelergues, 1964; Luria, 1964; Schuell, Jenkins & Jimenez-Papon, 1964; Geschwind, 1965; Benson & Patten, 1967; Benson & Geschwind, 1976; Kertesz, Lesk & McCabe, 1977; Naeser & Hayward, 1978; Kertesz, 1979; Kertesz, Harlock & Coates, 1979; Mazzocchi & Vignolo, 1979; Vignolo, 1979; Albert, Goodglass, Helm, Rubens & Alexander, 1981; Damasio, 1981; Kertesz, 1983; Selnes, Knopman, Niccum, Rubens & Larson, 1983; Naeser, Alexander, Helm-Estabrooks, Levine, Laughlin & Geschwind, 1982; Naeser, Helm-Estabrooks, Haas, Auerbach & Srinivasan, 1987; Gaddie, Naeser, Palumbo & Stiassny-Eder, 1989). Lesions in the right hemisphere are associated with relatively intact linguistic skills (Davis, 1993) but are associated with impairment of attention (Myers, 1986). In terms of speech comprehension, the majority of patients with right hemisphere lesions exhibit results that are statistically different from normal, but the size of the difference is often meaningless (Black & Strub, 1978; Coughlan & Warrington, 1978).

The severity of a speech comprehension deficit might be determined using: 1) tasks at different linguistic levels (words, sentences and paragraphs, for example) and 2) information regarding the site and extent of the lesion in relation to the recovery pattern. The next sections will examine both ways of determining the severity of the speech comprehension problem.

2.1.1. Assessment of the severity of the speech comprehension deficit using standardized tests

The Boston Diagnostic Aphasia Examination (BDAE) (Goodglass & Kaplan, 1983) and the Western Aphasia Battery (WAB) (Kertesz, 1982) were included in this review because of their widespread clinical use (Davis, 1993). Both batteries approach the assessment of the severity of a speech comprehension problem via subtests graded for linguistic complexity.

The Boston Diagnostic Aphasia Examination (BDAE)

The BDAE is used by clinicians and researchers for either identifying clinical syndromes (Broca, Wernicke, global, conduction, etc.) or as a basis for grouping experimental subjects according to syndrome. The BDAE has 27 subtests; and the score of each one is reported on a summary profile sheet in order to obtain a description of behaviors instead of a composite score for aphasia. An example of a BDAE summary profile sheet is found in Figure 1.

The second section of the BDAE deals with auditory comprehension. This section is subdivided into four subtests: word discrimination, body-part identification, commands, and complex ideational material. The subtests are presented using a simple to complex task gradient. In the first subtest, the patient is asked to point to pictures representing objects, actions, letters, numbers, colors and shapes. The next subtest involves right-left body-part identification. This subtest is not only used as a comprehension test but also serves to determine left-right discrimination. The third subtest uses one to five-stage commands. The patient's ability is rated according to the level of complexity successfully reached. In the last subtest, paragraphs of increasing length are read to the patient. The patient must afterwards answer a number of text comprehension questions. Again, the patient's ability is rated according to the level of complexity (paragraph length) successfully reached. In each subtest, the raw score is converted into percentiles. A mean percentile value for auditory comprehension can be computed using the percentiles

FIGURE 9-1 The BDAE's subtest summary profile of scores in relation to percentiles for the aphasic population. This is an example of Broca's aphasia with good auditory comprehension, few paraphasias, and expressive problems mainly in repetition. (Reprinted by permission from Goodglass, H., & Kaplan, F., *The Assessment of Aphasia and Language Disorders*. Chicago: Riverside, 1983, p. 78).

Taken from Davis (1993) A survey of adult aphasia and related language disorders.

Figure 1 - Example of a Boston Diagnostic Aphasia Examination (BDAE)

obtained from the four subtests. A low mean percentile (mean of around 25 and a range from 1 to 45) indicates a severe auditory comprehension problem whereas a high percentile (70 to 100) indicates normal comprehension or a mild deficit.

The BDAE itself was not designed to predict auditory comprehension recovery. However, it has been applied to document recovery in some formal studies (Naeser et al., 1987; Gaddie et al., 1989).

The Western Aphasia Battery (WAB)

The WAB is quite similar to the BDAE both in terms of the subtests included in the auditory verbal comprehension section and its clinical purpose, to classify patients with regard to the appropriate aphasia syndrome. In addition, the WAB was designed to serve as a measure of progress related to therapy (Kertesz, 1982).

In the auditory verbal comprehension section, the first subtest is a list of yes/no questions. The second subtest is an auditory word recognition task. The subject is asked to point to objects, pictures, and body parts. The third and last subtest involves more complex ideational material to assess multistage instruction comprehension. The subjects are asked to manipulate objects in front of them (pencil, book and comb). For example, a subject could be asked to put the pencil on the book while holding the comb in one hand. Again, the subtests follow a simple to complex task complexity gradient.

The results obtained with the auditory comprehension WAB subtests are rated on a severity scale. A severe comprehension problem would be indicated by a low score on the scale (less than 3.9 for global and isolation aphasia) whereas a slight comprehension problem would be indicated by a high score (between 7 and 10 for anomic aphasia). Figure 2 illustrates the decision tree provided by the WAB to classify aphasias. A high and significant correlation ($r=0.90$) has been found between the BDAE and the WAB for auditory comprehension performance (Wertz, Deal & Robinson, 1984).

FIGURE 9-3 Decision tree illustrating the manner in which the WAB classifies aphasia according to scores for fluency, auditory comprehension, and repetition.

Taken from Davis (1993) A survey of adult aphasia and related language disorders.

Figure 2 - Decision tree provided by the Western Aphasia Battery (WAB) to classify aphasia

2.1.1.1. Severity of the speech comprehension problem versus extent of the lesion and recovery

The use of powerful radioimaging techniques allows the examination of the relation between speech comprehension deficits and neuroanatomical correlates found in aphasic patients (Naeser & Hayward, 1978; Kertesz, 1979; Selnes, Niccum & Rubens, 1982; Selnes et al., 1983; Selnes, Niccum, Knopman & Rubens, 1984). It is generally accepted that larger lesions are related to poorer speech comprehension. Kertesz (1979) reported a significant negative correlation between speech comprehension and extent of the lesion in acute aphasic adults. When looking at the recovery pattern, using a difference compound score taken from the WAB, Kertesz (1979) found no significant correlation between the amount of recovery and the repeated WAB speech comprehension score. Early measures of severity of speech comprehension deficits, in the first week post onset, were also unrelated to amount of recovery in two earlier studies (Sarno & Levita, 1971; Gloning, Trappl, Heiss & Quatember, 1976). These results might be associated with the early post onset time window used in these studies. Such a short interval would not allow enough time to establish a neurophysiological recovery, which tends to stabilize in aphasic patients within the first few days to the first month after the cerebral accident (Rubens, 1977).

Selnes and colleagues have conducted carefully controlled prospective studies examining the relationship between recovery of speech comprehension problems and the site and extent of the lesion (Selnes et al., 1982; Selnes et al., 1983; Selnes et al., 1984). Globally, their results showed that most aphasic patients exhibited single word comprehension deficits at one month post-onset. However, the degree of the deficit was not predictive of the site of the lesion. Lesions located both inside and outside the posterior superior temporal lobe, around the auditory cortex, were associated with speech comprehension problems at one month post onset. For example, some patients with lesions sparing the posterior superior temporal lobe had severe speech comprehension problems at one month. At 6 months post onset, these patients regained near-normal speech comprehension at the single word level. For patients with lesions involving the

posterior superior temporal lobe, lesion size and location tended to be related to speech comprehension performance. Lesions, however small, located within the posterior superior temporal area were always related to more severe and persistent speech comprehension deficits.

Gaddie et al. (1989), using the BDAE, found two distinct recovery patterns in two groups of patients studied: one with fronto-parieto-temporal cortical lesions involving Wernicke's area, and the other with similar lesions, but no cortical involvement of Wernicke's area. At one to four months post onset, the two groups did not have significantly different scores of speech comprehension on the BDAE. At one year post onset, the patients that had cortical lesions sparing Wernicke's area had recovered substantially, whereas the patients within the other group had shown significantly less speech comprehension recovery. These results are in good agreement with a previous report by Naeser et al. (1987) who studied the relationship between severity of speech comprehension in Wernicke's aphasia and amount of of temporal lobe damage within Wernicke's area. Their results also showed that speech comprehension recovery is better six months to one year post onset when the damaged area included less than half of Wernicke's area regardless of the total temporoparietal lesion size. These results are in agreement with the proposal made by Selnes et al. (1982), who demonstrated that persistent speech comprehension deficits were associated with lesions in the posterior superior temporal and infrasyllvian supramarginal areas.

The conclusion emerging from the studies presented in this section tend to indicate that the acute evaluation of a speech comprehension problem in an aphasic patient by imaging or by speech tests, especially after the initial period following the onset, would be predictive of neither: 1) the site and the extent of the causative lesion, nor 2) the potential for recovery. The same research indicates that a better relationship between site, extent of the lesion and speech comprehension scores is observed when the severity of the speech comprehension problem is assessed at least 6 months post onset.

2.1.2. Auditory capabilities and speech comprehension deficit

One important factor that is frequently overlooked in the aphasia literature is the contribution of the auditory component to the speech comprehension process (Divenyi & Robinson, 1989). Numerous investigators considered aphasia to be essentially a language disorder, without involvement of auditory processing deficits (Carpenter & Rutherford, 1973; Varney, 1980; Varney, 1984; Riedel & Studdert-Kennedy, 1985; Blumstein, Katz, Goodglass, Shrier & Dworetzky, 1985). To the contrary, others found that auditory processing deficits could be the primary deficit in speech comprehension problems affecting aphasic patients (Efron, 1963; Swisher & Hirsch, 1972; Lackner & Teuber, 1973; Tallal & Newcombe, 1978; Schwartz & Tallal, 1980; McNeil & Kimelman, 1986; Divenyi & Robinson, 1989; Thompson & Abel, 1992; Phillips, 1995; Abel et al., 1996). This controversy is ongoing and the solution remains unclear even though the most recent pieces of evidence tend to demonstrate that non-linguistic auditory function is indeed related to the severity of speech comprehension problems in aphasic patients (Divenyi & Robinson, 1989; Abel et al., 1996).

Efron (1963), and Swisher and Hirsch (1972) studied the perception of the order of presentation of two non-speech sounds presented in rapid succession in brain-damaged patients. These subjects could only correctly perceive the order of the two sounds when there was a certain amount of time between them. Left or right brain-damaged patients required several hundred milliseconds between the two sounds before being able to determine the exact order of presentation. Trained normal subjects could correctly identify the order of presentation with an interstimulus interval of less than 20 ms (Hirsch, 1959). According to Swisher and Hirsch's results, the observed impairment was more pronounced in patients with left-hemispheric lesions. The ability to identify the order of presentation of non-speech signals could theoretically be related to the ability to form words from segmental units. However, the phonological and semantic context present in speech stimuli could compensate for an inability to perceive order of presentation. Therefore, it is not clear that this non-linguistic auditory dysfunction would cause a speech comprehension dysfunction.

Lackner and Teuber (1973) studied the ability of experimental subjects affected by right or left-hemisphere traumatic injury to discriminate the presence of one (fused) or two (distinct) clicks while listening to a binaural stimulus. The interaural time difference (ITD) between the clicks was varied from 0 to 1 second. The stimuli ranged from a single fused auditory image with simultaneous binaural presentation to the perception of two distinct images with a large ITD. The results showed that patients with left temporal lesions needed a significantly longer ITD to perceive a single fused click when compared to normal controls, or patients with lesions in the right hemisphere, or patients with lesions outside the temporal lobe. Patients with left-hemispheric lesions also needed a significantly longer interaural time difference before being able to perceive two distinct stimuli. When looking at the pattern of results, and the presence or absence of a dysphasic syndrome (sic), the left-brain injured dysphasic patients exhibited significantly impaired fusion thresholds, especially in the left-ear leading condition of stimulation. The fusion threshold impairment was significantly less in the non-dysphasic group.

The findings of Lackner and Teuber (1973) are valued for their implication that the left temporal area seems to be more involved than the right in duration discrimination tasks. However, the relationship between impaired fusion thresholds for non-speech signals and speech comprehension deficits was not clearly indicated in their paper. Formal aphasia testing was not performed, and no information was given regarding the degree of speech or language involvement for the studied patients.

Tallal and Newcombe (1978) studied a group of men with chronic, unilateral lesions of the right or left hemisphere caused by missile fragment wounds. The patients with left hemispheric lesions showed residual signs of aphasia as demonstrated by one aphasia test (Token Test), whereas patients with right hemispheric lesions showed no sign of aphasia. All subjects were submitted, along with a normal control group, to 1) a training procedure to identify contrasting pairs of three-formant synthetic syllables /ba - da/ and 2) a non-verbal sequencing task using two complex tones differing in fundamental frequency. For the first task, the transition in all formants was either short (40 ms), or long (80 ms). For

the second task, the interval between the complex tones was varied from 8 to 428 ms in a manner similar to that used by Efron (1963).

On the verbal task, for rapid formant transition (40 ms), the performance of the left hemisphere damaged group was again significantly poorer when compared to the two other groups. When the formant transition was increased to 80 ms, the performance improved for 40 % of left hemisphere damaged subjects. The patients showing the highest number of errors on the Token test failed to improve with extended duration of the formant transition. A significantly high correlation of 0.83 was obtained between the number of errors on the Token Test and the number of errors made on the non-verbal sequence test using short interstimulus intervals (8-305 ms). On the non-verbal sequence task, for short interstimulus intervals, the performance of the left hemisphere damaged subjects was significantly poorer when compared to the right hemisphere damaged, or normal, subjects. For long interstimulus intervals, the performance scores between the three groups were not significantly different. These results suggest a relationship between non-verbal auditory processing, phonetic perception, and speech comprehension in at least some aphasic patients. The most severely impaired subjects might possess both acoustic and phonetic processing deficits, which could explain why simple acoustic manipulation alone was not sufficient to modify their speech perception abilities.

The results reported by Tallal and Newcombe (1978) were challenged by Riedel and Studdert-Kennedy (1985). They argued that a dissociation between specific stimulus identification performance and the discrimination of difference/similarity in paired stimuli is frequently observed, both in aphasic patients and normal listeners. The identification performance was said to be related to the phonetic process, whereas the discrimination performance would be more closely associated with the auditory process.

Riedel and Studdert-Kennedy studied both the identification and discrimination abilities of 12 aphasic patients, 5 of whom were fluent, and 7 of whom nonfluent aphasics. The verbal stimuli used were similar to those of Tallal and Newcombe (1978). Unlike Tallal and Newcombe's findings, results for the identification task showed no

improvement with the extension of formant transition for either fluent or nonfluent aphasics. This emphasizes that acoustic changes do not lead to an improvement in the phonetic processing output. Additionally, as predicted by research on normal listeners, the extension of formant transition would usually lead to a perceptual shift from a stop consonant to a glide (for example: /ba/ to /wa/) (Miller & Liberman, 1979). The same observations were made with the aphasic subjects in the Riedel and Studdert-Kennedy study. Surprisingly, the paper by Tallal and Newcombe did not report such a perceptual shifts, but simply an improved stop perception from /ba/ to /da/.

As predicted, aphasic subjects performed better on the discrimination task than on the identification task (Riedel & Studdert-Kennedy, 1985). As opposed to Tallal and Newcombe's findings, the discrimination task was not influenced by the interstimulus interval, which was varied from 50 to 500 ms. For these aphasic subjects, a reduction in the interstimulus interval did not reduce the discrimination performance. However, the task in the Riedel and Studdert-Kennedy study was less demanding since it did not involve any ordering of stimuli.

According to the Riedel and Studdert-Kennedy results, the observed dissociation between identification and discrimination performance provides evidence against a common auditory processing origin. A common auditory origin would have caused a comparable deficit in both identification and discrimination tasks. Subjects performed at the same low level for both short and long formant transition duration in the identification task, whereas the performance was quite good in the discrimination task.

A more recent study by Schwartz and Tallal (1980) on normal college students tended to confirm lengthening the duration of formant transition reduced the classical right-ear advantage observed in dichotic or complex monaural listening tasks. Increasing the duration of the formant transition reduces the difficulty of the task, leading to a less demanding temporal task for the left hemisphere. These results, along with data collected by Tallal and colleagues from children with language learning impairments, suggest that the left hemisphere is not only involved in the linguistic processing of speech, but also in

the processing of brief acoustic signals juxtaposed in time similar to those carried by speech. The language comprehension problems exhibited by these children seem to be related to the processing of on-line speech (Tallal & Piercy, 1973a, 1973b; Tallal & Piercy, 1974; Tallal, Stark, Kallman & Melitts, 1981; Tallal & Stark, 1981; Tallal, Stark & Melitts, 1985; Merzenich, Jenkins, Johnston, Schreiner, Miller & Tallal, 1996; Tallal, Miller, Bedi, Byrna, Wang, Nagarajan, Schreiner, Jenkins & Merzenich, 1996). The processing of the succession of rapidly changing acoustic features likely plays a critical role in live speech perception and comprehension (Schwartz & Tallal, 1980).

The most comprehensive study of non-linguistic auditory capabilities in aphasic patients was conducted by Divenyi and Robinson (1989). Using a cohort composed of 11 left-cva (cerebrovascular accident) aphasic, 4 right-cva non-aphasic and 8 normal subjects, Divenyi and Robinson studied the relationship between 7 non-linguistic auditory abilities and impairment of speech comprehension as assessed by three standardized language tests (BDAE, PICA and Token Test). The examined auditory abilities included processes that were assumed to take place at the peripheral level, i.e., frequency discrimination, temporal gap detection, frequency selectivity, and at more central level, i.e., gap discrimination, discrimination of frequency transitions, temporal order discrimination, and detection of tones with and without frequency uncertainty.

The subjects were further divided according to side of lesion, type of fluent or non-fluent aphasia, and level of speech comprehension based on the results of the standardized tests (good, moderate, poor). The results of the tests for peripheral level auditory function were as follows:

Frequency Discrimination: A significantly poorer performance for the right-cva subjects, whereas control group and left-cva were not statistically different.

Gap Detection: There were no significant differences between the groups. However, there was a correlation between gap detection and tuning curves, which

suggests that gap detection covaries with other peripheral factors such as frequency selectivity.

Frequency Selectivity: All subjects except one exhibited normal frequency selectivity. No relationship was found between frequency selectivity and side of lesion, fluency, or speech comprehension.

The results of the more central tests were as follows:

Gap Discrimination: There were no significant differences between the groups. Therefore again, side of lesion, fluency, or speech comprehension were not significantly contributing factors in gap discrimination.

Discrimination of Frequency Transitions: Test results were similar to those found for the frequency discrimination tests, in that a significantly poorer performance was seen for the right-cva subjects, while control group and left-cva subjects were not statistically different. Fluency, and level of speech comprehension, were not significantly related factors.

Temporal Order Discrimination: The test design used to examine this auditory function was probably too difficult. Even normal subjects needed a very long test tone duration to achieve a correct discrimination of the order of the tones. This high level of difficulty led to a floor effect. However, right-cva subjects had significantly poorer performance when compared to the control group. Non-fluent left-cva aphasic subjects also showed significantly poorer performance when compared to fluent aphasic subjects.

Detection of Tones: The effect of frequency uncertainty on the detection of tones was significantly different for the right-cva subjects as compared to both control and left-cva groups. Within the left-cva group, fluency and speech comprehension were not significant contributing factors.

Regression and discriminant analyses were performed using the test results from measures of non-linguistic auditory capabilities and speech comprehension. Results of the regression analysis showed that the majority of the non-linguistic tests accounted for a significant proportion of the variance in aphasic patients, whereas only a small subset of the non-linguistic tests accounted for a significant proportion of variance in normal listeners. In aphasic patients, the subset of frequency sweep discrimination, frequency discrimination, and detection of tones of uncertain frequency accounted for 54.2 % of the regression variance.

The discriminant analysis was used to characterize the pattern of auditory performance using different levels of speech comprehension and fluency in the aphasic group. The canonical discriminant function was not significant for fluency, and therefore will not be examined here. Two canonical discriminant functions were found to segregate the three subgroups of left-cva aphasic patients according to their speech comprehension (good, moderate, poor). The first function carried 98 % of the variance ($p < 0.02$) and was strongly related to temporal order discrimination but no correlation was found with any other auditory measures. The second function was not significant. The speech comprehension subgroups within the left-cva group could therefore be distinguished based on time-related auditory tasks. Classification using the first discrimination function was successful for subjects with good and poor speech comprehension 90.0 % of the time (10 out of 11 subjects).

These findings reported by Divenyi and Robinson (1989) strongly suggest that the comprehension performance of left-cva aphasic patients can be predicted from non-linguistic auditory performance. Time-related tasks seem to be the most powerful classification tool to distinguish level of speech comprehension performance of left-cva aphasic patients.

In a related study, Thompson and Abel (1992) examined the effect of lesion on auditory detection, discrimination, and speech processing using 3 groups of ten patients

with confirmed lesions of the eighth nerve and right and left temporal cortex. Subjects were submitted to a series of frequency and duration discrimination and detection tasks. Speech intelligibility was measured using the Four Alternative Auditory Feature Test (FAAF) (Foster & Haggard, 1979). Subjects with left temporal lesions had the largest difference limen for both frequency and duration when compared to subjects of all other groups. In the FAAF test, the same subjects had the lowest consonant discrimination scores. Significant correlations were obtained in the left temporal lesions group between FAAF measures and duration discrimination of short duration pure tones. As pointed out by Thompson and Abel (1992), the ability to process basic acoustic parameters such as frequency and duration might be related to the discrimination of linguistic features such as place of articulation, formant transition, distinction of voice contrast in stops, timing of silent burst in fricatives, and discrimination of vowel length. These findings confirmed the importance of the contribution of non-linguistic auditory processing to speech processing, if only at the level of consonant discrimination.

The results of Thompson and Abel (1992) were extended by Abel et al. (1996) who examined the relationship between severity of the language processing deficit, and temporal processing deficits in 22 aphasic patients with left anterior (n=6) and left posterior (n=16) hemispheric lesions. Three standardized tests of speech-language performance were administered: the PICA, the BDAE and the Revised Token Test. The left posterior group showed significantly poorer results on each of the speech-language tests when compared to a normal group. Moreover, the difference limen for duration discrimination was significantly larger for the left posterior group when compared with the control group. The anterior group was overlapping with the posterior group in both speech and auditory measures. The severity of the language processing disorder, as evaluated by the PICA, was negatively correlated ($p < 0.01$) with the duration difference limen obtained by individuals with left posterior lesions. The relationship between duration discrimination ability, and the specific auditory comprehension subscale of either standardized tests of speech-language performance was not examined in this study. This study provides additional evidence that there might exist a relationship between non-

linguistic temporal processing and severity of language processing difficulties in aphasic individuals with left posterior hemispheric lesions.

Varney (1980) examined the relationship between speech perception and comprehension. In a cohort of 26 subjects, Varney found that 14 aphasic subjects with impaired perception also exhibited word comprehension deficit. In the remaining 12 subjects, 8 had a severe word comprehension problem but showed normal speech perception. These results illustrate clearly that some comprehension problems could be related to a primary auditory deficit, whereas other depends upon a linguistic processing disorder. Instead, Varney, because of the absence of a systematic relationship for *all* his cases, concluded that the auditory disabilities could not explain the speech comprehension problems.

2.1.2.1. Synthesis of the research on auditory capabilities and speech comprehension deficits

- 1) Efron (1963) and Swisher and Hirsch (1972) showed that the discrimination of the order of presentation of stimuli in rapid succession is affected by left hemispheric lesions. However, this deficit in auditory function could be compensated for using linguistic context (word, sentence). Therefore, the relationship between non-linguistic and linguistic tasks remains unclear.
- 2) Lackner and Teuber (1973) examined duration discrimination capability in patients with right and left-hemisphere lesions. Their results revealed that patients with left-hemispheric lesions had significant limitations in duration discrimination. However, the relationship between the auditory performance of these patients and their speech comprehension performance was not specifically examined by these researchers.
- 3) The work of Tallal and colleagues suggests that faulty processing of the succession of rapidly changing acoustic features is related to speech perception and comprehension problems, at least in very restricted subgroups of children. However, the speech

comprehension problems of these children identified as developmental aphasic or language-learning disordered, might be very different from the one presented by aphasic patients who had suffered a cerebro-vascular accident. Findings by Riedel and Studdert-Kennedy (1985) emphasize that both linguistic and auditory dimensions should be examined to fully describe a speech comprehension deficit in the aphasic patient.

- 4) The findings reported by Divenyi and Robinson (1989) strongly suggest that the comprehension performance of left-cva aphasic patients could be predicted from non-linguistic auditory performance. This study was the first to carefully evaluate the comprehension performance of aphasic patients using standardized aphasia batteries. Non-linguistic auditory tasks and time-related tasks seemed to lead to the most powerful classification criteria regarding level of linguistic comprehension performance of left-cva aphasic patients.
- 5) Thompson and Abel (1992) and Abel et al. (1996) confirmed the results of Divenyi and Robinson (1989), and showed deficits in the temporal discrimination tasks of left-cva aphasic patients that had speech comprehension problems identified with standardized aphasia batteries.
- 6) Results by Varney (1980) emphasized the importance of recognizing the existence of purely linguistic processing dysfunction in a fair proportion of aphasic patients presenting with speech comprehension problems. In these patients, non-linguistic auditory performance had remained intact.

2.2. A sound localization approach in the identification of central auditory processing problems

The current assessment of central processing disorders focuses on speech and most of the time fails to distinguish linguistic from acoustic aspects of it. Speech perception involves the processing of a succession of very brief events closely spaced in time (Phillips, 1995). The non-linguistic auditory functions that have been studied in aphasic patients suggest that time-related auditory tasks are the most powerful predictors of the severity of a speech comprehension deficit (Divenyi & Robinson, 1989). There is still a need for more acoustic tests of central processing disorders. Sound localization has been identified as a compound, non-linguistic, auditory ability that is supported by the cortical structures in the neighborhood of those supporting speech comprehension. The current audiological approach with patients with temporal lobe damage and aphasia is severely limited by the use of linguistic-related material, or inappropriate use of auditory evoked potentials. An approach based on sound localization abilities could help to resolve these limitations. The following section examines the advantages and limitations of this potential non-linguistic procedure for patients with temporal lobe lesion that were tested for speech comprehension problem.

2.2.1. Sound localization deficits and lesions of the central auditory system

A traditional site-of-lesion approach is necessary to help identify neuroanatomical sources of auditory dysfunction. Animal and human lesion studies have brought to audiology the neurophysiological basis for many of our routine psychoacoustic procedures. The development and application of a clinical test involving sound localization should proceed similarly. As already discussed, in the case of aphasic patients, the existence of a left hemispheric site-of-lesion is often related to speech comprehension deficits. However, it is important to note that for a small number of patients, neuroanatomic lesions remain undetected by modern neuroradiology instrumentation whereas neurological investigation clearly indicates the occurrence of unilateral brain damage. Therefore, identification based solely on dysfunction via non-

imaging techniques must also be considered as a valid tool for determining the presence of central auditory system lesions.

2.2.1.1. Brain stem site-of-lesion

Localization abilities, like many other auditory abilities, are dependent upon the integrity of the auditory pathways located in the brainstem. Research by Nordlund (1964), Bosatra and Russolo (1976), Newton and Hickson (1981), among others, has shown that lesions affecting the pons or the eighth nerve will impair localization abilities. This impairment might be explained by a neural disruption of the coding of interaural differences (Casseday & Neff, 1975). Currently, few clinicians will use a sound localization paradigm to make a differential diagnosis for suspected brainstem involvement. Auditory brainstem responses (ABR), masking level differences (MLD)(Olsen & Noffsinger, 1976), acoustic reflexes (Hall, 1985) and dichotic digits (Musiek, 1983) are preferred, and widely used by audiologists because of their specific sensitivity to brainstem lesions. Computerized-tomography (CT-SCAN) and magnetic resonance imaging (MRI) are also used in the neurodiagnosis of lesions that affect the central auditory system (Mafee, Meyer & Hill, 1985). With such an armada of tests available to assess the integrity of auditory brainstem structures, the significant contribution of a test for localization may be questioned.

2.2.1.2. Distinctions between sub-cortical and cortical site-of-lesion

There are only a few animal studies related to sound localization and subcortical structures. Some researchers have investigated the medial geniculate body (Strominger & Oesterreich, 1970; Al'tman, Syka & Shmigidina, 1970; Jenkins & Masterton, 1982; Kelly & Judge, 1985), other reserchers investigated cerebellar structures (Bechterev, Syka & Al'tman, 1975). In both cases, these subcortical structures appeared to be sensitive to real or simulated sound source movement in space, and less sensitive to stationary sound sources. Moreover, the nature of the response required from the animal, either attending to a sound or moving towards it, suggested that subcortical mechanisms are sensitive to

sound source location for simple tasks (e.g. head movement to a single fixed sound source) but that cortical structures were needed for complex localization behavior (e.g. head tracking of a moving sound source) (Heffner & Masterton, 1975; Heffner & Heffner, 1990; Beitel & Kaas, 1993). In agreement with this, several other studies also found that cortical areas were essential for complex localization tasks (Al'tman, 1968; Sovijarvi & Hyvarinen, 1974; Ahissar, Ahissar, Bergman & Vaadia, 1992; Stumpf, Toronchuk & Cynader, 1992; Toronchuk, Stumpf & Cynader, 1992; Takahashi & Keller, 1995).

A precise delineation between subcortical and cortical localization functions in humans has not been made so far. To date, there appears to be no report in the literature on sound localization or lateralization for isolated subcortical lesion in humans. With the exception of one patient, described by Gollegly and Musiek (1993), showing a large vascular lesion involving the right insula, lenticular process and external and internal capsules, the majority of the cases reported also showed some degree of cortical involvement. Poirier et al. (1994) examined at the relative implication of cortical and subcortical structures in three hemidecorticated patients using stationary and moving sound sources. Their results showed that the performance was worse in the latter task. However, Poirier concluded that: "the processing of moving sounds requires a stronger cortical involvement (...) but because of the degeneration that accompanies cortical lesions, subcortical structures such as the medial geniculate body, the lateral posterior thalamic group, and the inferior and superior colliculus would also be affected by the hemidecortication". Based primarily on site-of-lesion studies, it is difficult to clearly distinguish between the sound localization functions associated with the sub-cortical and cortical structures.

By demonstrating that lesions could affect differently the localization of stationary and mobile sound sources, many studies done on humans have shown that a high degree of central auditory integration is needed for moving sources (Cranford et al., 1990; Moore, Cranford & Rahn, 1990; Cranford, Morgan, Scudder & Moore, 1993; Poirier et al., 1994). Therefore, there is probably not a single level neither sub-cortical nor cortical that is responsible for the localization of mobile sound source, but a complex integration

of information at different levels that is requisite (i.e., brainstem *and* subcortical *and* cortical structures). Disrupting this integration at any level, by lesions or aging process for example, would lead to sound localization dysfunction. For a less integrative task, such as localization of a fixed sound source, a greater amount of damage, or greater dysfunction would be needed to observe a significant deviance. Therefore, as tasks involving localization or lateralization of stationary and mobile sound sources have different sensitivity regarding site-of-lesion or dysfunction identification, this differential sensitivity must be taken into account in the design of clinical sound localization tests.

Additional evidence on cortical/subcortical localization function was presented in a study by Pinek and Brouchon (1992). These authors raised the question of representation of the auditory space and its interaction with different motor output such as head turning and manual pointing to sound sources. In this study, four normal subjects and three brain-damaged subjects were asked to localize six auditory targets in a blindfolded condition (15° , 40° and 70° to the right and to the left of the middle of a semicircle). Subjects were instructed to indicate the location of the source by a manual pointing gesture or by a head turning gesture. Each subject was given 180 trials: 60 for head turning, 60 for each left and right hand manual pointing respectively. Results showed that there was a systematic difference between head turning and manual pointing performance across all normal and brain-damaged subjects. Manual pointing deficits tended to be restricted to unilateral hemifield whereas head turning seemed to produce bilateral deficits. These results suggested that differential spatial processes coexist. These differential processes could have different sensorimotor neural substrates: one head-centered system that could involve sub-cortical reflexes and another one that could be related to a cortical level involved in spatial coding (Paillard, 1971; Pinek & Brouchon, 1992).

The latter study, while adding further distinctions between cortical and subcortical sound localization functions, focuses on the crucial importance of the type of responses a clinical sound localization test would have to use. These could be head turning, manual pointing, visual pointing, or even verbal responses. As supported by these researchers, a

different mode of responses could involve different neural substrates. Therefore, the development of a clinical localization test must not only address this question, but also integrate the different modes of response in the assessment of different levels/functions of the central system.

2.2.1.3. Cortical site-of-lesion

Over the years, animal data have shown that permanent deficits in the localization of sound sources in the contralateral hemifield are found after primary auditory cortex lesions. It appears that the primary auditory cortex is necessary to achieve a certain level of sound localization accuracy at least under conditions designed to elicit the precedence effect¹, or for other complex localization behaviors (Cranford, Diamond, Ravizza & Whitfield, 1970; Cranford, Ravizza, Diamond & Whitfield, 1971; Whitfield, Cranford, Ravizza, Diamond, 1972; Ravizza & Masterton, 1972; Cranford et Oberholtzer, 1976; Neff & Casseday, 1977; Heffner, 1978; Jenkins & Masterton, 1982 and Jenkins & Merzenich, 1984). In human studies, the same pattern seems to emerge, but the picture is much more complex and still unclear: relative preservation of sound localization ability and asymmetric performances are common findings (Butler, 1994; Pinek, Duhamel, Cavé & Brouchon, 1989; Al'tman et al., 1987; Hochster & Kelly, 1981; Cornelisse & Kelly, 1987; Poirier et al. 1994; Zatorre et al., 1995; Griffiths, Rees, Witton, Cross, Shakir & Green, 1997).

Relatively good performance for fixed-sound localization was recorded in hemispherectomized patients by Poirier et al. (1994) and Zatorre et al. (1995). In both studies, patients aged 14 to 41 years underwent unilateral hemispherectomy for the treatment of epileptic seizures. The experimental samples included 6 patients with right lesioned hemisphere and 3 with left lesioned hemisphere. Sound localization performance remained similar to normal controls in the hemifield ipsilateral to the lesioned hemisphere. In the contralateral hemifield, localization performance was relatively well preserved especially for peri-central location. The performance dropped

down significantly for extreme locations of the contralateral hemifield (90°). The decrease in localization performance noted for extreme location of the contralateral hemisphere was similar in both left and right hemispherectomized patients. As opposed to Pinek, Duhamel, Cavé and Brouchon (1989), no left hemisphere dominance for sound localization could be identified in the above mentioned studies.

In a monaural and binaural paradigm, Butler (1994) studied the localization performance of normal subjects to sounds presented in the medial sagittal plane. The subjects were fitted monaurally with hearing protection (earplug and earmuff). They were then asked to localize a high-pass (3.5 kHz) filtered noise appropriate for localization in the vertical plane. By using a dummy loudspeaker arrangement in the horizontal plane, listeners were required to identify the vertical and horizontal coordinate of the sound source. The horizontal location was restricted to 0° azimuth, hence only vertical elevation was varied. Results obtained under this paradigm showed that monaural listeners made erratic judgment of location when listening with the right ear. When listening monaurally with the left ear, subjects also misjudged sound source location, but the difference was less pronounced and only laterally displaced towards the unoccluded ear when compared with binaural listening tasks.

In contrast to the results of Pinek et al. (1989), Butler identified a left ear-right hemisphere dominance for sound localization. These results suggest that the left hemisphere does not have a good spectral representation for location. However, in Butler's experiment, localization judgment was based mainly on spectral cues. In the Pinek et al. study, the localization judgment was made on short duration speech and white noise signals in which the high frequency content is less dominant. Differential dominance may take place depending on spectral complexity, and the need for a listener to rely on such cues and probably not on sound localization hemispheric asymmetry per se. Also, Jenkins and Merzenich (1984) revealed that contralateral deficits in localization were confined only to frequencies that corresponded to the ablated frequency band of the auditory cortex. This important finding emphasizes the idea that sound localization is

¹ The precedence effect is explained in page 30 of this text.

performed on a frequency specific basis by the cortical substrate regardless of which hemisphere is involved. The localization of a complex stimulus, such as a moving auditory image, or binaural stimulation occurring under precedence effect, could also involve both hemispheres and the contribution of the corpus callosum.

Moving auditory images have been examined in studies by Al'tman et al. (1987) and Poirier et al. (1994). In the study by Al'tman et al. (1987), asymmetrical performance in patients with focal damage of the temporal lobes was noted in a moving auditory fused image perception task. A deficit in the localization ability of moving auditory fused image was noted for 75 % of the patients in the cohort. However, depending on the side of the temporal damage, the pattern of deficit differed. When the deficit was located in the right temporal lobe, the perception of the fused image movement was disturbed only in the hemifield contralateral to the damage. When the deficit was located in the left temporal lobe, perception of the auditory image was disordered in both ipsilateral and contralateral hemifields. Using an array of loudspeakers in free-field, Poirier and colleagues recorded a sound localization deficit in 3 hemispherectomized patients. As hypothesized, the deficit was more pronounced in the hemifield contralateral to the lesioned hemisphere. An interesting finding is that a deficit was also noted in the ipsilateral hemisphere but to a lesser degree. Nevertheless, the deficit was significant in 2 out of 3 patients. Results from both studies suggest that localization of moving auditory image would be more sensitive to temporal lobe damage than a unique fixed sound source.

The sound localization abilities required under the precedence effect (PE) have been examined in adults by Cornelisse and Kelly (1987), and in children by Hochster and Kelly (1981).

The PE is created when the same signal is fed through a pair of matched loudspeakers located on each side of the subject's head, with one output leading the other by a few milliseconds. A midline virtual sound source is created by using an interspeaker time delay of 0 ms. The PE may be broken down into two different components.

The first PE process, sometimes called summing localization, is dominated by the fixed binaural time delay imposed by the distance between the two ears. A brief click coming from a left hand loudspeaker will first reach the left ear and then, after traveling around the head, will reach the right ear. The maximum delay associated with this additional traveling time is around $650\text{ }\mu\text{s}$ in humans (Kuhn, 1987). If a second click, coming from a right hand loudspeaker, is presented during this binaural delay, the listener will perceive a single click. In these conditions, the location of the sound source is perceived away from midline at a virtual position *toward* the leading sound source, in our example, toward the left hand loudspeaker. The virtual position of the source is influenced by the position of the loudspeaker. If the loudspeaker is moved away to the left (in fact also away from the other loudspeaker), the virtual source is also displaced in the same direction. On the other hand, if the loudspeaker is moved closer to the other loudspeaker, the virtual source will be displaced in the same manner. This phenomenon is explained by the ability of the central auditory system to resolve very small interaural time difference in a magnitude of approximately $100\text{ }\mu\text{s}$ (Klump & Eady, 1956).

The second PE process is involved when the interspeaker delay is extended to intervals between 1 and 8 to 16 ms., i.e. beyond the time period associated with the maximum binaural difference. Under these conditions, the location of the sound source is perceived *at* the location of the leading loudspeaker and the trailing loudspeaker is perceived to be inactive. When the interspeaker delay is further extended over 8 to 16 ms, the two sound sources are perceived independently. In other words, the lagging source is perceived as an echo of the leading source. The neural mechanisms involved in this second process seem to be mediated through a centrally active suppression of the echo. It is said to be active because the interaural time difference limen exhibited by the auditory system is well below, in the order of microseconds (Klump & Eady, 1956), the temporal delays involved in this second PE process, in the order of milliseconds. Studies conducted with animal models indicate that the temporal lobe and the auditory cortex are involved in the perception of a fused auditory image in this second PE process (Cranford

et al., 1970; Cranford & Oberholtzer, 1976; Whitfield, 1978; Whitfield et al., 1972; Whitfield, Diamond, Chiveralls & Williamson, 1978).

In an adult study (Cornelisse & Kelly, 1987), 14 unilaterally brain-damaged subjects aged from 28 to 73 years old were tested with both single click and paired clicks presented from loudspeakers on the left and right of midline. The presence of brain damage was objectively defined using CT-SCAN.

Of the cohort,

- 6 patients had no evidence of a discrete lesion although neurological diagnosis clearly indicated unilateral brain damage,
- 8 patients had a discrete lesion identified by the CT-SCAN (4 in the left hemisphere and 4 in the right), as well as clear neurological indications of unilateral brain damage, and
- 9 normal patients with no history that could suggest the possibility of a brain lesion, as a control group.

Subjects were asked to point with their preferred hand towards the perceived location of a single click or paired clicks. All subjects were able to easily localize the single sound source. When asked to repeat the task with paired clicks, the mean percentage of reversal errors (failure to identify the side of the leading loudspeaker) was less than 1% for the control group, patients with no discrete lesion as identified on the CT-SCAN and patients with a left hemisphere lesion. The right hemisphere lesion group showed a statistically significant 18.5 % of reversal errors ($p < 0.01$), with the number of reversals being greater when the left click was leading over the right click (i.e. when the leading click was located in the contralateral hemifield to the right hemisphere lesion). The errors occurred mainly with the use of delays of 8 and 16 ms between the paired clicks. These delays approximate the point at which the precedence effect starts to break down into the perception of two separated signals.

The results were similar in a study of children by Hochster and Kelly (1981) in which normal children, and children identified with a temporal lobe epilepsy focus were compared using the same tasks. Children with temporal lobe epilepsy also failed in greater number when paired clicks were presented. Again, the difficulties were located predominantly with the longer delays of 8 and 16 ms. For most subjects, the localization of the leading click was more difficult in the hemifield contralateral to the side of the brain damage.

Due to different cortical sites of lesion within left and right hemispheres involving more or less of the auditory areas, it is difficult to conclude on the basis of these adult and child studies that there is a right hemispheric dominance for sound localization. More data would be needed for both right and left sites of cortical involvement to thoroughly assess the hemispheric asymmetry theory. However, these studies illustrate that more complex tasks are definitively more sensitive to cortical lesions than a single source sound localization task.

The reviewed literature does not provide a definitive theory of hemispheric specialization for sound localization. However, this is not absolutely necessary to determine, as there is enough evidence to justify the use of complex stimuli or tasks that will involve the contribution of *both* hemispheres. The eventual finding that a specific complex task imposes a heavier workload on one or the other hemispheres is not so important as long as *both* hemispheres could be challenged at a certain proven level of sensitivity.

2.2.1.4. Corpus callosum lesions

Few studies, either on animal models or humans, have focused on the contribution of the corpus callosum to sound localization. In the cat, it has been suggested that callosal fibers are involved mainly in the fusion of the two horizontal auditory hemifields (Imig & Brugge, 1978; Imig & Reale, 1980; Imig, Reale, Brugge, Morel & Adrian, 1986). Electrophysiological studies conducted in the cat by Poirier, Richer, Lepore, Ptito and

Guillemot (1989) and by Poirier, Lepore, Provençal, Ptito and Guillemot (1995) showed that callosal fibers are mainly involved in the processing of null interaural time delays which simulate auditory midline localization. In a set of experiments done with humans, Poirier et al. (1993) examined the performance of four congenital acallosal subjects in a task involving the localization of fixed and simulated mobile sound sources. Based on animal studies, these investigators expected that the localization performance of acallosal subjects would be worst around midline and better at more lateral positions. In fact, the results showed the exact opposite; that the best performance was achieved for pericentral positions as one would expect in normal subjects. However, acallosal subjects showed a less precise performance for fixed sound sources in the whole auditory field when compared to a IQ-matched control group. Possible explanations brought forward by the investigators range from cerebral plasticity in the acallosal brain to an absence of midline fusion in the central auditory system as opposed to visual and somatosensory systems. Another possible explanation derived from an animal study by Poirier et al. (1995) is that callosal fibers are involved in tuning sound localization information more finely, therefore human subjects could combine information coming from the thalamo-cortical system and the corpus callosum to more accurately specify sound source location.

The findings of these studies are of great interest, but more research is needed to specify the contribution of the corpus callosum to sound localization. For example, there is no longitudinal data on the localization performance of subjects whom had undergone commissurotomy involving the corpus callosum. Such data would help clarify the question of whether the performance of these subjects could be similar to that demonstrated by congenital acallosal subjects (data from Poirier et al., 1993). The longitudinal nature of an investigation of this type would assess more adequately the effect of brain plasticity. A comparison between the evolution of localization performance between auditory and somatosensory systems could also be made longitudinally. This kind of study could shed more light on the hypothesis of midline fusion in auditory central system.

The idea that the corpus callosum serves as an enhancement system for cortical sound localization capabilities suggests also that only complex localization tasks would be affected by isolated lesions or dysfunction of this structure. Mobile sound sources have already been used to assess callosal sound localization function, but an even more complex task would be needed if one wishes to delineate between cortical and callosal function.

2.2.1.5. The effect of aging on sound localization abilities

The progressive decrease in hearing sensitivity that occurs with age is common knowledge, but not the effect of aging on most other hearing abilities (Willott, 1991). The effect of aging on various auditory localization tasks has been studied only in recent years (Cranford et al., 1990; Cranford & Romereim, 1992; Cranford, Andres, Piatz & Reissig, 1993). The examination of the age effect is also important in the context of aphasia, which is frequently caused by a cerebrovascular accident as it has been shown that the incidence of cerebrovascular disease is age dependent (Jerger & Jerger, 1981).

Using a sound localization task involving PE, Cranford and colleagues were interested in the performance of groups of persons that typically exhibit problems with auditory temporal processing. Like many other sound localization tasks, the PE task requires the integration of multiple binaural temporal cues. Multiple sclerosis (MS) sufferers and elderly subjects have been shown to present with auditory disabilities involving the treatment of temporal information (Kasten & McCroskey, 1982; Robin & Royer, 1989). It was hypothesized that MS patients and the elderly would exhibit a poorer performance on a PE task than a younger group of control subjects (Cranford et al., 1990). The results for the elderly subjects revealed that their performance was worst for interspeaker delays of less than 1 ms as compared to the performance of the younger group. However, this conclusion was limited by the fact that the effect of hearing loss was not controlled for in the older group.

This study was replicated in 1993 by Cranford, Andres and co-workers using four groups of subjects. Young subjects were divided into two groups, one with normal hearing and another one with hearing loss. Elderly subjects were similarly divided into two groups. The high-frequency pure-tone average thresholds were similar for the young and elderly groups with normal hearing, and the hearing-impaired sub-groups were matched as for the amount of hearing loss as well. Using the same PE task as in 1990, the results showed a similar trend, with the elderly subjects tending to have a lower performance for interspeaker delays below 1 ms. The differences in performance for interspeaker delays above 1 ms was not significant between all 4 groups. When hearing loss effect was controlled for, the elderly group still showed a greater performance deficit than the hearing-impaired young group. The detrimental effect of hearing loss on the performance of the PE task was more pronounced in the elderly group. As Cranford et al. (1993b) stated, it is possible that elderly people with hearing loss constitutes a sub-group of the elderly population. This group could have a greater incidence of associated cognitive or central auditory processing problems. However, this effect seems to be more related to sensory factors, as for example auditory temporal resolution, than cognitive factors.

In the above study, the vast majority of subjects showed a hearing loss of cochlear origin in both the young and elderly groups. As Cranford et al. (1993b) noted, the fact that the high-frequency pure-tone average thresholds (1, 2 and 4 kHz) were similar across individuals does not indicate similar underlying cochlear pathologies. It is not possible to disagree with that statement. Unfortunately, audiometric thresholds, which are a measure of only one auditory ability, are used predominantly to classify groups of subjects. A more rigorous approach would be to incorporate other kinds of measurement of the auditory ability in the study, and use it as a basis for comparison. In this case, a measure of auditory temporal acuity would probably be a better criteria of classification than the average of high-frequency hearing thresholds. This way, a better relationship could be established between the effect of loss of auditory sensitivity, and loss of temporal auditory abilities on tasks such as the PE in the elderly.

An example of this would be the Cranford and Romereim (1992) study who investigated the link between a localization task such as the PE and speech understanding in the elderly. The PE requires a complex neural integration of temporal information. Cranford and Romereim's study was designed to investigate whether elderly persons who showed deficits in the PE task would also exhibited speech comprehension problems. Normal speech comprehension depends on processing of rapidly changing temporal as well as spectral features of the ongoing speech chain (Cranford & Romereim, 1992). Twenty-two elderly persons ranging from 60 to 80 years of age and a young control group of 21 persons ranging from 19 to 40 years old were submitted to performance-intensity functions for a phonetically balanced word test (PI-PB including PB-max), a synthetic sentences with ipsilateral speech competition test (SSI-ICM), and a stationary PE task. The PE task was the same as described by Cranford et al. (1990). The SSI-ICM is built with 10 randomized versions of the same 10 third-order sentences. These sentences are successively presented with a continuous ipsilateral competitive speech message for which the message-to-competition ratio is varied. The subject has a numbered list of the 10 sentences and is asked to identify the perceived sentence.

Similar to previous studies, the groups differed significantly for the PE task for interspeaker delays of less than 0.7 ms. The elderly group's PE performance was significantly correlated with the SSI-ICM score ($r=.56$, $p=.007$), but not with the PB-max ($r=.21$, $p=.34$). Additionally, PE and SSI-ICM were both correlated with age and not with high-frequency pure-tone average. Unlike the SSI-ICM test, the PI-PB test is believed to be more sensitive to cochlear hearing loss (presbycusis), and less specific to central auditory processing disabilities (central presbycusis). These results, while identifying commonalities in the processes underlying speech comprehension and PE test performance (Cranford & Romereim, 1992), have to be viewed cautiously, because other cognitive factors could also be involved.

A similar relationship between how children process speech information in noisy or reverberant environments (such as classrooms), and performance on the PE test has also been suggested. While not examining the existence of this specific relationship, the

performance of children in a PE task was investigated by Cranford, Morgan, Scudder & Moore (1993). This study pointed toward an age effect on the PE task that might be related to maturational aspects. Such a maturational effect could be similar to the effect of aging in the elderly, therefore the same kind of link with speech understanding could be suspected. However, solid experimental data has yet to be collected in this area.

2.2.1.6. The effect of attention on sound localization performance

It seems obvious that attention interacts with sound localization abilities especially in situations like the cocktail party where a group of persons are simultaneously speaking (Cherry, 1953). In this situation, sound localization and selective attention probably contribute to the selection of the desired speech source of information in a specific spatial location.

Mondor and Zatorre (1995) recently conducted an extensive series of experiments on auditory spatial attention. Young normal-hearing subjects were submitted to six experiments in which the temporal relations between an auditory spatial cue and an auditory target were manipulated. These experiments were designed to explore the mechanisms underlying the allocation of auditory attention to specific spatial locations. The effects of 1) the distance of an attentional shift, 2) the time interval between the cue and the target, 3) the use of valid and invalid cues, and 4) the azimuthal position of the target were studied, along with the spatial distribution of auditory attention in an identification task. The results provided a solid explanation for the mechanisms by which auditory attention is shifted from one spatial location to another, and presented a precise description of the spatial extent of an attentional focus.

The main finding of Mondor and Zatorre's study is opposed to Rhodes' (1987) proposition that auditory attention moves analogically through space. According to Rhodes, auditory attention would work similarly to the well-known "spotlight" visual attention process (Johnston & Dark, 1986). This model predicts that a shift in spatial attention would procede in an all-or-none fashion from one spatial location to the other.

The time to shift the attentional spotlight would be related to the distance between the two spatial locations: the greater the distance, the poorer the localization performance.

Mondor and Zatorre used long or brief duration auditory targets to show that spatial attention varies progressively when the distance from cue to target locations is increased. For long-duration targets, the reaction time increased with increasing distance between cue and target. However, the steepness of the function was related to the time available to fully engage the attentional process at the cued location. For a longer target, the steepness of the function was greater, and simulated an analogic movement of the spatial focus. These results were similar to those of Rhodes for long-duration targets (Rhodes only used long-duration auditory targets). On the contrary, for brief duration targets, the density of the attentional focus, illustrated by reaction time and localization errors, clearly and progressively declined with increasing distance between cue and target location. This progressive decline is not appropriately represented by the all-or-none function proposed by Rhodes (1987). These results also suggest that the so-called “effect of distance” might be otherwise attributable to the time necessary to fully engage the attentional focus. The same temporal influence was also demonstrated when the time interval between the cue and the target was increased: with greater interval, the identification performance progressively improved (Mondor & Zatorre, 1995).

The same gradual decline of attention was reported by Teder and Näätänen (1994) using event-related auditory evoked potentials. Their paradigm involved selective attention to natural speech coming from two sets of 3 loudspeakers placed 66° from midline. Within each set, the middle loudspeaker delivered passages of Finnish literature. The subjects were asked to selectively attend to one of the competing messages. At the same time, tone pips were presented randomly at each of the 6 loudspeakers. Subjects were asked to ignore the tone pips. Event-related potentials were recorded with the tone pips under attended and unattended conditions. The results showed that the tone pips embedded in the attended message increased the amplitude of the N1 component when compared to the unattended condition. This enhancement of N1 amplitude did not followed an all-or-none function since a reduction of amplitude of 50 to 70 % was

observed for an azimuthal range of 3° which is in the range of the smallest minimal audible angle detectable by human listeners (Makous & Middlebrooks, 1990). An all-or-none function would have lead to a complete reduction of amplitude in the same 3° range.

The evidence provided by the literature, both from behavioral and electrophysiological approaches, demonstrate that the spatial auditory attention can be narrowly focused to enhance the perception of an acoustic signal. In addition, this focus does not follow an all-or-none function, but instead gradually declines to adjacent locations. This attentional process presents some similarities to the concept of spatial channel proposed in section 2.5.

In the context of this study on patients with a unilateral cerebrovascular accident, it is important to remember that right hemisphere lesions are frequently associated with left neglect or, more appropriately, hemi-inattention (Posner, Walker, Friedrich & Rafal, 1987). For example, hemi-inattention would cause a patient to ignore the left side of his body (shaving only right side of the face), or to draw only the right side of a happy face figure. It has been shown that in these patients, it is not the perception in the left hemifield that is affected, but instead a difficulty to disengage attentional focus from the right hemifield (Posner et al., 1987). Patients with left hemisphere lesions could also exhibit extinction of attentive responses to sound sources located in the right auditory hemifield when there are simultaneously active sound sources in both hemifields (Heilman & Valenstein, 1972). But whether this extinction, restricted most of the time to multisource conditions, should be considered as « auditory neglect » is still a matter of debate and controversy (Hugdahl, Wester & Asbjørnsen, 1991; Beaton & McCarthy, 1993; Hugdahl & Wester, 1994; Beaton & McCarthy, 1995).

These findings emphasize one important aspect of the effect of attention in perceptual tasks such as sound localization. In a sound localization paradigm involving a selective attention approach (more than one source of sound), it is essential that the attentional effect should be either carefully controlled or measured.

2.2.1.7. Electrophysiologic means to assess localization abilities

A few studies have focused on sound localization and electrophysiological correlates of behavioral phenomena:

- a) shift in lateralization: McEvoy, Picton, Champagne, Kellett and Kelly, 1990; McEvoy, Picton and Champagne, 1991a; McEvoy, Picton and Champagne, 1991b; Picton, McEvoy and Champagne, 1991;
- b) detection of deviant stimulus: Paavilainen, Karlsson, Renikainen and Näätänen, 1989;
- c) moving sound image: Al'tman, Belov, Vaitulevich and Malt'seva, 1984; Al'tman and Vaitulevich, 1988;
- d) masking level difference (MLD): Yonovitz, Thompson and Lozar, 1979; Fowler and Mikami, 1992a, 1992b, 1995, 1996.

These various experimental paradigms probably tapped different central processes, but shared a common ultimate goal in finding a more objective way to describe binaural interaction either at the brain stem level or at the cortical level. Some aspects of these procedures show high potential for clinical use in the identification of auditory dysfunction in patients with hemispheric lesions.

In a series of papers, McEvoy et al. (1990, 1991a, 1991b) and Picton et al. (1991) described an electrophysiological procedure involving binaurally perceived shifts in the lateralization of a continuous sound. The paradigm used involves abrupt changes in the interaural time delay of a binaural continuous noise to cause a shift in the perceived location of the sound from one ear to the other. Abrupt phase change, if not done properly, produces a click that would elicit an evoked potential in itself. In these studies, care was taken to ensure that the change in interaural time delay could not be perceived monaurally nor that it elicited any detectable click. Results were presented in parallel with a comparable onset stimulus in order to demonstrate that the binaural shift in lateralization was not contaminated by sound onset.

The shift in lateralization elicited an evoked potential with a positive peak (P_1) at 75 ms, a negative peak (N_1) at 135 ms and a second positive peak (P_2) at 220 ms. These late potentials were elicited, maximally at the vertex, consistently and reliably in normal-hearing subjects ranging in age from 16 to 49 years old. These responses were quite similar to normal late potentials evoked by stimulus onset but were longer in latency (by 30 to 40 ms). Usually, normal late potentials, such as P_1 , N_1 and P_2 would be obtained in the range of 40-70 ms for P_1 , 75-150 ms for N_1 and in the range of 150-200 ms for P_2 . Despite several attempts, the lateralization shift procedure had not been able to elicit any earlier potentials (McEvoy et al., 1990). The binaural processes involved here are based on very specific time-related information extracted from neuronal impulses. The task certainly involved the brain stem structures. The fact that the brainstem activity was not reflected in the electrophysiological recordings is puzzling and may be explained either by asynchrony of neuronal firing which tends to reduce the far-field recorded electrical activity, or by opposed bilateral dipoles at the superior olive level (McEvoy et al., 1990).

However, the specific nature of the cortical response remains unclear since the change in the stimulus can not be perceived monaurally. The response seems to be mediated uniquely by binaural processing. The response could reflect the cross-correlation process postulated by Sayers and Cherry (1957) the expression of which would be the delayed latency of the normal P_1 - N_1 - P_2 complex (McEvoy et al., 1991b). The response could also be viewed as a mismatch negativity (MMN). The MMN, an event-related brain potential, is believed to reflect neuronal stimulus traces left by subtle changes of the physical dimensions of a sound. Since the MMN can be elicited even when auditory stimuli are unattended, MMN seems to reflect an automatic brain responses to change in the stimulus (Näätänen, Gaillard & Mäntysalo, 1978). The MMN has been primarily examined using frequency, intensity, or duration changes in pure tones and also in speech stimuli (for a review, see Näätänen 1995). It has been, to a lesser extent, studied with regard to changes in spatial location of a sound source (Paavilainen et al., 1989). The stimulus used in the McEvoy et al. and Picton et al. studies is not typically one associated with MMN, but since there is a behavioral perception of change in the ongoing noise, it is not possible to rule out this possibility.

Paavilainen et al. (1989) conducted a MMN study in which auditory stimuli were presented in blocks to 12 normal subjects. Blocks were organized using 2 types; standard (90% of the presentation), and deviant (remaining 10%) stimuli delivered in a random order. The only difference between standard and deviant stimuli was their simulated and real spatial location. The main cues used for sound lateralization, the interaural phase difference for low frequencies and the interaural intensity difference for high frequencies, were alternatively manipulated to create the deviant stimulus. The same kind of difference was also more naturally created, in free-field, using differently positioned loudspeakers where both dimensions, phase and intensity, changed simultaneously.

The MMN was elicited regardless of whether location changes were produced on earphones or by free-field loudspeakers. The MMN recorded was largest in the frontocentral area, in agreement with the McEvoy and Picton studies, with latency values found in between 100 to 200 ms depending on experimental conditions. It is important to note that the MMN was detected in addition to the normal N_1 wave in this study, as opposed to the normal but delayed P_1 - N_1 - P_2 complex in the McEvoy and Picton studies. Additionally, differences were observed between lateralization and localization experimental conditions in the study of Paavilainen et al. (1989). The MMN growth curve, computed as a function of the distance between frequent and rare stimuli, was more progressive in the earphone condition while the slope was steeper in free-field indicating a tendency for an all-or-none type of response. These results strongly suggest that the MMN reflects a precise code of spatial origin of an auditory stimulus. Alho's review (1995) suggests that the MMN generators are located mainly in the auditory cortex. Additional generators have also been located in other brain structures, namely, the frontal lobe in humans, and the thalamus and hippocampus in some animal models. The results of Paavilainen et al. (1989) seem to rule out the possibility that the cortical response reported by the McEvoy and Picton studies would be a MMN. However there is no strong evidence that the MMN response recorded by Paavilainen et al. (1989) and that the delayed P_1 - N_1 - P_2 complex in the McEvoy and Picton studies reflect two distinct processes.

Al'tman et al. (1984) and Al'tman and Vaitulevich (1988) used increasing interaural timing delays with binaural clicks presented in series to create simulated movement of a sound source. This signal evoked movement of the binaural sound image from the midline to either right or left ear. Late evoked potentials were recorded for both stationary and moving sound sources. Various changes in the waveform were observed. The most salient and consistent change was the increase in the amplitude of the N_1 component when a moving source was used. This result should be considered with care since the onset nature of the moving source was not controlled. Even if the location of the sound was different from time to time, it was always locked to stimulus onset. Therefore it is not possible, in this paradigm, to separate a stimulus onset effect from the movement itself in the electrophysiological waveform.

Behavioral masking level difference (MLD) has been studied extensively, as it is believed to reflect the first coding step of the interaural phase difference at the level of the superior olivary complex. MLD is used clinically for the purpose of identification of a low brainstem site of involvement (Musiek et al., 1994) and in patients with multiple sclerosis (Jerger, Oliver, Chmiel & Rivera, 1986). A few studies were made to identify an electrophysiological expression of this behavioral phenomena (Yonovitz et al., 1979; Kevanishvili & Lagidze, 1987; Fowler & Mikami, 1992a, 1992b, 1995, 1996). Electrophysiological correlates that have been studied include latency and amplitude differences for auditory brain stem (ABR) wave V, various binaural interaction components derived off-line, late potential amplitude, and detection threshold differences. All of the studies which have looked for MLD-like characteristics in the ABR have failed to find any equivalent component (Kevanishvili & Lagidze, 1987; Fowler & Mikami, 1995).

Other studies successfully identified late components exhibiting MLD-like characteristics (Yonovitz et al., 1979; Fowler & Mikami, 1992a, 1992b, 1996). According to Fowler & Mikami (1992a, 1992b, 1996), all late latency components, P_1 , N_1 , P_2 and N_2 , exhibited an MLD-like effect. However, their studies focused on the P_2

component. The MLD parameters, masking noise pressure spectrum level and bandwidth, were examined. In both studies, a difference in P_2 detection threshold was recorded between S_0N_0 (signal and noise are homophasic) and $S_\pi N_0$ (signal is antiphasic while noise is still homophasic) conditions yielding to an MLD from 3.2 to 14.0 dB depending on parametric adjustment. The characteristics of the cortical electrophysiological responses were similar to behavioral MLD.

The results of Fowler and Mikami studies suggest that the auditory cortex is involved in the production of the MLD, along with structures in the brainstem (Noffsinger, Olsen, Carhart, Hart & Sahgal, 1972). The delineation of brainstem and cortical contribution to the production of the MLD is not resolved by these studies. More research is needed to 1) find an electrophysiological correlate of brainstem MLD-related activity, 2) extract the brainstem contribution from the MLD cortical correlate or 3) extract the cortical contribution from the brainstem MLD-related activity.

There are various ways to stimulate the central auditory system in a sound localization paradigm. Electrophysiological expressions of this behavioral phenomena have been found for the cortical area. No component has been identified so far at brainstem level, even if its contribution to the resolution of binaural differences is known. In the context of aphasia due to cortical lesions, the absence of a brainstem electrophysiological correlate is of less clinical importance. The electrophysiological research provides a solid basis which helps with the evaluation of aphasic patients with central auditory processing disorders. There is a clear need for careful clinical adaptation of these procedures and their integration with selected behavioral methods of assessment. Given the more objective character of the electrophysiological approach, it is surprising that none of these procedures received much attention from clinicians involved in the evaluation of aphasic patients with suspected central auditory disorders. The integration of such procedures into clinical routine would certainly improve the identification of central auditory processing disorders and would also provide evidence that such an evaluation is necessary.

2.3. Sound localization deficit in aphasic patients

Sound localization abilities are anecdotally reported in case studies of aphasic patients, and also in patients with a congenital absence of temporal lobes. Unfortunately, in most cases, the localization tasks used are quite unrefined, and important methodological details are missing.

Mark & Chobor (1990) reported on the case of a patient with bitemporal infarcts. The patient was complaining that he could hear but not understand speech. On standardized aphasia testing (WAB), he correctly responded to almost 50% of yes/no questions, and 50% of auditory word recognition items but was unable to give adequate responses to commands. To assess sound localization abilities, keys were shaken at various locations about a meter away from the patient. He was asked to close his eyes and point manually to the location of the key's sound each time he heard them. Even with such an uncontrolled stimulus and poor methodological controls, and despite normal hearing acuity, the patient was wrong for 33% of the presentations. The pattern of errors is not precisely reported, but it seems that the errors were related to front-behind confusions.

Kazui, Naritomi, Sawada, Inoue & Okuda (1990) reported a case of subcortical auditory agnosia associated with bitemporal subcortices infarcts. The patient reported that he was unable to hear anything but buzzing noises even when listening to his own voice. His speech comprehension, evaluated with pictures and letters was impaired at the single word level (80% correct), the sentence level (80% correct), and more markedly at the level of commands (50% correct). His recognition of environmental sounds was also impaired (28 to 56% correct over trials). His sound localization abilities were tested using a "soft" bell ringing. Also, the patient's abilities to point, eye closed, to the sound source was poor. No consistent pattern of errors is reported. The patient's audiogram revealed a moderate bilateral sensorineural hearing loss (pure-tone threshold average of 65 dB HL). The authors did not report whether the presence of the hearing loss was

compensated for by an increased intensity level for the bell ringing sound used in the localization task. Since it is reported that this sound was soft, the poor sound localization performance could have been influenced by the presence of peripheral hearing loss.

Lambert, Eustache, Lechevalier, Rossa and Viader (1989) reported a case of ventricular hemorrhage with no obvious parenchymatous brain damage. This patient behaved as if completely deaf. Forty days after onset, the patient was selectively impaired in sound and music identification without aphasia. However, when stop-consonant identification was tested, the subject made articulatory place confusions with the feature place. At the onset, the subject's audiogram revealed a bilateral severe mixed (sic) hearing loss. Sound localization abilities were also assessed at this time. The patient was unable to distinguish from left, straight in front and right but not from front to behind. The presence of the peripheral hearing loss could have strongly influenced the localization performance. Again, no information is provided for the nature of the stimulus or of its physical dimensions. The audiogram and the sound localization tasks were repeated three months later. The hearing sensitivity was better and only a mild bilateral hearing loss (15-20 dB) was present. For the localization task, the patient produced the same errors as previously. In this patient, the linguistic processing might have been relatively spared whereas the acoustic processing seemed to be impaired, especially at the level of the processing of rapidly changing temporal features.

Hood, Berlin and Allen (1994) reported the case of a patient with bilateral absence of temporal lobes. This patient has been followed for more than 20 years and despite a normal audiogram was unable to understand speech. When submitted repeatedly to a gross localization task (right-left, front-rear discrimination), this patient correctly identified the sound source location only in 40 to 80% of the trials. In contrast, normal individuals will easily obtain a perfect score of 100% with this simple task. In terms of speech comprehension, only a few details are reported, enough to say that this patient exhibited "a rather significant degree of aphasia".

2.4. Synthesis of the review of literature

Using standardized aphasia batteries, such as the BDAE and the WAB, acute evaluation of a speech comprehension problem would be predictive of neither: 1) the site and extent of the lesion, nor 2) the potential for recovery of speech comprehension. However, delayed post-onset evaluation (6 months to 1 year), using the same tools, *is* related to site and extent of the lesion in Wernicke's area within the posterior superior temporal lobe, and to the potential for recovery of speech comprehension.

Non-linguistic capabilities that are more often related to speech comprehension problems, at least in some aphasic patients and in delayed post-onset evaluation, involve the temporal dimension of the acoustic stimulus. The performance of aphasic patients on more complex time-related auditory tasks seems to lead to the most powerful classification criteria in terms of speech comprehension abilities (either good or poor) assessed by standardized aphasia batteries.

A time-related auditory processing deficit does not seem to be the primary underlying origin of the speech comprehension problem for *all* aphasic patients. There is clearly a population of aphasics for whom the comprehension problem is related more or less exclusively to a linguistic processing deficit.

Sound localization abilities might be affected by lesions at the various levels of the central auditory system. However, more complex localization tasks require a stronger cortical involvement. An example of a more complex localization task is provided by the precedence effect in which the response mode used within a localization task also involves various levels of the central nervous system. Manual pointing to a localized sound source seems to be related to a stronger cortical involvement, whereas head turning would involve subcortical reflexes.

The question of asymmetry of hemispheric functions in sound localization can not be resolved with the current literature at this time. However, it seems that more complex stimuli varying simultaneously in frequency and duration would require the intervention

of both hemispheres and of the corpus callosum. Sound localization of moving auditory images and sound localization under the precedence effect seems to be more sensitive than single fixed sound sources to temporal lobe damage.

Sound localization abilities under the precedence effect are affected by age, especially for interaural delays between 0 and 1 ms. This decreased ability might be related to changes in temporal resolution abilities. Age-related changes in the identification performance of speech presented in a competition background are significantly correlated to the sound localization abilities under the precedence effect.

Auditory spatial attention can be narrowly focused at a specific location to enhance the perception of an acoustic signal. The power of the focus has been shown to gradually decline towards adjacent locations. Attention is affected by the location of a lesion, with damage to the right hemisphere possibly leading to hemi-inattention.

Shifts in perceived sound lateralization have been shown to elicit cortical evoked auditory potentials similar to the conventional P_1 - N_1 - P_2 - N_2 waveform components. However, the components are longer in latency than their conventional counterparts. The masking level difference (MLD) paradigm can also be used to obtain electrophysiologic responses related to sound localization tasks.

Sound localization deficits have been reported in aphasic patients. In most reported cases, the lesion extended bilaterally and involved some degree of cortical deafness. Given the lack of a valid clinical test, there is no available data on sound localization abilities of well documented aphasic patients showing speech comprehension problems related to unilateral hemispheric lesions.

2.5. Preliminary results with normal subjects using a binaural masking approach

2.5.1. Behavioral approach

An exploratory study done by Leroux & Kelly (1995) used a binaural masking approach to demonstrate the existence of spatial channels in humans. The investigators used a binaural target signal, and a binaural masking noise, and varied their respective interaural time differences (ITDs). The target signal and the masking noise could share the same ITD or were respectively adjusted to different ITDs. The ITDs were varied from 0 to 1 ms in 0.1 ms steps. Young normal subjects were asked to detect the presence of the target signal from within the noise, and a probability of detection was computed from their responses for various ITDs of both target signal and masking noise. A detection probability of 1 meant that the target signal was always detected, whereas a score of 0 meant that the target signal was simply not perceived. A clear decrease in the probability of detection was shown when the signal and the noise shared the same ITD (Figure 3). This result was observed for 3 masking noise ITDs: in front ($0\ \mu\text{s}$), at the right ($-600\ \mu\text{s}$), and at the left ($600\ \mu\text{s}$) of the listener. Across subjects, this effect varied in shape and bandwidth with respect to the ITD of the target signal: some subjects had more powerful selective ability than others.

The narrower the shape of the obtained curve, the more powerful the selection of the target signal among noise. The subject illustrated in Figure 3 was able to detect the target signal with great accuracy except when it shared the same ITD as the background noise. For other listeners, the shape of the curve was wider, meaning that a greater ITD difference between the signal and the noise was needed in order to detect the target signal.

Taken from Leroux & Kelly (1995) Combined use of behavioral and electrophysiological methods to explore human sound localization abilities. *Canadian Acoustics*, 23, 11-12.

Figure 3 - Detection probability of a click signal in a binaural masking noise at 3 interaural time differences. The figure was drawn from the results obtained by a typical young normal subject.

To examine the effect of auditory spatial attention upon this specific binaural masking task, reaction times were also collected. Figure 4 shows the reaction time obtained with an ITD for the noise at 0 μ s. The signal-to-noise ratio was varied from 0 to -10 dB.

The reaction times show in Figure 4 were longer when both the signal and the noise shared the same ITD (at 0 μ s). For adjacent ITDs, the reaction time progressively decreased with increasing distance between signal and noise ITDs. The reaction time was linearly related to the signal-to-noise ratio, at least on the relatively small studied range of 10 dB. The longer reaction time at 0 μ s ITD suggests that the midline noise was perceived as an attentional auditory spatial distractor since the attentional focus improves progressively for more distant ITDs.

These exploratory results, both in terms of detection capabilities and reaction time, while based on ITD sensitivity, support the general concept of spatial channels (Phillips, Taylor, Hall, Carr & Mossop, 1997; Phillips, Hall, Harrington & Taylor, 1998; Boehnke & Phillips, 1999). This mechanism related to sound localization abilities is postulated to be representative of the primary auditory processing supported by the cortical structures (Boehnke & Phillips, 1999).

2.5.2. Electrophysiological approach

Electrophysiological data were also collected using the detection paradigm described in section 2.5.1. The main difference between the two approaches is the absence of any active participation of the subjects in the electrophysiological approach. The subjects were asked only to listen passively to sounds without having to respond to them in any manner.

Normal young subjects were submitted to repeated binaural stimuli for which the interaural time difference and the signal-to-noise ratio were manipulated. Cortical

Figure 4 - Mean reaction time for the detection of a binaural target signal as a function of the interaural time difference for the signal at 3 signal-to-noise ratios (4 young normal listeners)

auditory evoked responses were collected using a bipolar electrode arrangement: an active electrode at the vertex and inactive electrodes at right and left earlobes.

Amplitude and latency values were measured for P_1 , N_1 , P_2 and N_2 components of the late auditory evoked potentials. Figure 5 illustrates the mean latency values obtained over the left hemisphere for 3 subjects as a function of the ITD of the signal for a perceived midline position of the noise (midline is indicated by 0 μ s on the abscissa). Latency values were quite stable for all components. Slight variations are seen but might be due only to the small sample size. In Figure 5, the physical intensity of the stimuli remained unchanged, only the ITD of the signal relative to the noise was varied.

Figure 6 shows the amplitude values for P_1 - N_1 , N_1 - P_2 and P_2 - N_2 components measured over the left hemisphere. Again the amplitude is plotted as a function of the ITD of the signals for a perceived midline position of the noise (midline is indicated by 0 μ s on the abscissa). It can be seen that the amplitude of the N_1 - P_2 component is reduced for a shared ITD of the signal and the noise (0 μ s on the abscissa). The amplitude increased progressively for adjacent ITDs from -300 to -600 μ s. In Figure 6, as in Figure 5, the physical intensity of the stimuli remained unchanged.

The shape of this electrophysiological function is quite similar to the shape observed with the behavioral approach illustrated in Figure 3. In Figure 6, the amplitude of the response for all components was larger when the leading stimulus was perceived at the right ear (600 μ s on the abscissa) as compared to the left ear (-600 μ s on the abscissa). A similar measure made over the right hemisphere (not shown) also exhibits a larger amplitude when the leading stimulus was perceived by the right ear.

An increase in the amplitude of the P_1 - N_1 component can also be observed for the shared ITD in Figure 6 (0 μ s on the abscissa). Behaviorally, the signal is much more difficult to detect when it shared the same ITD with the noise. Therefore, a reduction in amplitude, and not an enhancement, is expected for the shared ITD. Nevertheless, an increased amplitude for the P_1 - N_1 component might be explained by an auditory spatial attention

effect. This effect could be triggered by the presence of the signal over the noise position. A similar effect has been also reported by Teder and Näätänen (1994). A larger sample size would be required to verify this hypothesis.

The preliminary cortical auditory evoked potential findings, both in terms of latency and amplitude, provide the basis for an electrophysiological correlate of the behavioral approach exposed in section 2.5.1. From a clinical point of view, an electrophysiological method to test sound localization would be very useful with difficult-to-test patients.

Figure 5 - Mean latency values obtained for cortical responses P1, N1, P2 and N2 measured over left hemisphere ($n = 3$)

Figure 6 - Mean amplitude values obtained for cortical responses P1-N1, N1-P2 and P2-N2 measured over left hemisphere ($n = 3$)

Chapter Three

3. Objective and Hypothesis

The objective of this study was to assess the potential of a clinical procedure, related to sound localization, to detect central auditory dysfunction in patients with unilateral cerebrovascular accident (cva). The presence of a unilateral hemispheric lesion could also have an impact on linguistic skills. For example, one might expect that left-hemisphere cva would lead, for a large majority of patients, to aphasia and speech comprehension problems. Right-hemisphere cva would likely lead to significantly less frequent and milder speech comprehension problems. To determine the possible coexistence between sound localization deficits and impairments in speech comprehension, all patients were given standardized tests of aphasia and speech processing as well as tests of binaural hearing underlying sound localization. When compared with a normal control group, it was hypothesized that the group of patients with unilateral cva would show deficits in the spatial channel mechanisms as measured both behaviorally and electrophysiologically.

Chapter Four

4. Methodology

4.1. Subjects

The experimental sample consisted of 22 English-speaking, right-handed patients with unilateral cva under the care of either speech-language pathologists of the Department of Speech-Language Pathology and Audiology, or neurologists at the Department of Neurology of the Ottawa Hospital (General Site). Subjects were selected on the basis of a chart review in order to document clinical evidence of a left or right unilateral cva. For each selected subject, a radio-neurological (CT-SCAN and/or MRI) or neurological examination revealed unilateral damage to cortical substrate. More specifically, patients with unilateral stroke, either by ischemia or hemorrhage, involving the middle cerebral artery (MCA) in the temporal lobe were selected. The MCA runs along the Sylvian fissure and provides the major vascular supply to the auditory receptive and association areas of the suprasylvian and posterior temporal lobe regions. These cortical areas, especially on the left side, are essential regions for speech and language.

Two groups were formed on the basis of a left ($n=12$) or right ($n=10$) MCA cva. For patients with left MCA cva, the results from the BDAE and the WAB were recorded as measures of acute speech comprehension difficulties. When available, the same measures were recorded for patients with right MCA cva. However, it is important to note that most patients with right MCA cva were not sent to speech-language therapy. Therefore, for most of them, a measure of acute speech comprehension was not available. Chronic speech comprehension measures were then performed to compensate for this lack of acute information and used in subsequent analyses (see section 4.2.3.).

The following exclusion criteria were applied for the selection of the experimental subjects: patients that had multiple cvas, were left-handed, older than 70 years old, presented a history of neurologic surgery, multiple sclerosis, dementia or other psychiatric

syndromes, alcohol or drug abuse, and use of psychopharmaceutical agents. All subjects were medically stable with a post-onset ranging from 6 months to 4 years. Prior to the definitive inclusion in the experimental protocol, subjects were submitted to an audiometric evaluation. The results of the audiometric evaluation were required to show normal hearing thresholds according to age to validate inclusion in the experimental protocol (ISO-DIS-7029, 1984).

The left MCA cva group was comprised of 5 females and 7 males aged between 39 and 66.7 years (mean: 54.9 ± 9.5). The right MCA cva group was comprised of 4 females and 6 males aged between 33.2 and 68 years (mean: 53.7 ± 12.6). All right MCA cva subjects were free from the hemi-inattention syndrome often seen in patients with right hemisphere cerebral lesion. A group of 10 normal adults, 5 females and 5 males aged between 39.5 and 68.3 years (mean: 53.3 ± 9.8) with no prior history of hearing and otologic problems served as control subjects. The subjects of the control group were also submitted to an audiometric evaluation to ensure normal hearing thresholds according to age (ISO-DIS-7029, 1984).

Table 1 describes the site-of-lesion for the brain-damaged subjects included in the behavioral and electrophysiological procedures. The table indicates whether the site-of-lesion information was obtained from Magnetic Resonance Imaging (MRI), Computerized Tomography Scanning (CTSCAN) or Neurologic Examination (NE). The table also gives gender, age and elapsed time after onset of the MCA cva for each subject.

The left and right brain-damaged groups were well matched for patients that had suffered temporal infarct (Left: $n = 4$; Right: $n = 5$), temporal hemorrhage (Left: $n = 1$; Right: $n = 1$), temporo-parietal infarct (Left: $n = 3$; Right: $n = 3$), temporo-parietal hemorrhage (Left: $n = 3$; Right: $n = 0$) and non-specific MCA territory stroke (Left: $n = 1$; Right: $n = 1$). Mean age, gender distribution and mean elapsed time after onset were also similar between groups.

Table 1 - Identification of the site-of-lesion for subjects in the left and right MCA cva groups

Ss ID	Age	Gender	MRI	CTSCAN	NE	Onset	Site-of-lesion
L1	63.2	M		X		2.3	Left temporal infarct
L2	50.5	M	X	X		0.8	Left temporo-parietal hemorrhage
L5	50.4	M		X		3.5	Left temporal infarct
L6	41.0	F		X		3.8	Left temporo-parietal hemorrhage
L7	45.3	F	X	X		1.5	Left temporo-parietal hypodensity (subtle)
L8	39.0	F		X		0.9	Left temporal infarct (large)
L9	62.5	M		X		1.1	Left temporo-parietal infarct
L10	56.0	M		X		0.8	Left temporo-parietal infarct (large)
L11	57.8	M		X		1.9	Left temporo-parietal hemorrhage
L13	66.7	F		X		2.3	Left temporal infarct
L14	62.8	M		X		1.5	Left temporal hemorrhage
L15	63.5	F			X	1.0	Left MCA territory stroke
Mean	54.9					1.8	
R2	47.5	F		X		2.9	Right temporo-parietal infarct
R3	33.2	M		X		1.5	Right temporo-parietal infarct
R4	62.0	M		X		1.3	Right temporal infarct
R5	67.5	F	X	X		0.5	Right temporal infarct (small)
R7	43.0	M		X		2.5	Right temporo-parietal infarct (massive)
R8	61.1	M	X	X		2.9	Right temporal infarct (small)
R10	57.0	M		X		2.0	Right temporal hemorrhage
R11	36.9	F		X		2.6	Right temporal infarct
R12	68.0	M		X		3.8	Right temporal infarct
R13	60.3	F			X	0.9	Right MCA territory stroke
Mean	53.7					2.1	

4.1.1. Auditory status of the experimental subjects

All experimental subjects had normal hearing thresholds according to age (ISO-DIS-7029, 1984). Table 2 shows the mean pure-tone average (500, 1000 and 2000 Hz) hearing thresholds (dBHL) at each ear for the three groups. Means and variances were submitted to a MANOVA (Stevens, 1992). As shown by Table 2, there was no significant difference between groups for hearing thresholds [$F(2,29) < 1$].

Table 2 - Mean pure-tone average hearing thresholds (dB HL) measured at the right and left ear for the normal, left and right brain-damaged groups

Group	Right Ear Hearing Threshold (dB HL)	Left Ear Hearing Threshold (dB HL)
Normal (n=10)	11.8	12.3
Left (n=12)	15.4	13.8
Right (n=10)	14.2	15.8
F-value (2,29)	0.40	0.34

4.2. Methods

Subjects from both the experimental and the control groups were submitted to behavioral and electrophysiological binaural masking procedures modified from Leroux and Kelly (1995). The rationale of these procedures and a detailed description of the procedures themselves are found in the following section. In addition, all subjects were given speech comprehension tests to document chronic speech comprehension problems.

4.2.1. Rationale applied to the development of the binaural masking procedures

As revealed by the literature review, one common finding in research devoted to the study of auditory capabilities and speech comprehension problems in aphasic patients is a decrease in temporal processing abilities for a significant proportion of these patients (Efron, 1963; Swisher and Hirsch, 1972; Lackner and Tuber, 1973; Tallal & Piercy, 1973a, 1973b; Tallal & Piercy, 1974; Schwartz & Tallal, 1980; Tallal et al., 1981; Tallal & Stark, 1981; Tallal et al., 1985; Divenyi and Robinson, 1989; Thompson and Abel, 1992; Phillips, 1995; Abel et al., 1996; Merzenich et al., 1996; Tallal et al., 1996). Divenyi and Robinson (1989) showed that the performance of aphasic patients on complex time-related auditory tasks was a powerful classification tool in terms of speech comprehension abilities. Normal performance on auditory temporal tasks were linked with better speech comprehension abilities. Conversely, poor performance on auditory temporal tasks were related to low scores on speech comprehension tasks. The cortical lesions usually associated with speech comprehension problems are often located within the temporal lobe.

The animal and human research have demonstrated sound localization deficits in the case of unilateral cortical lesions, in particular within the temporal lobe and the primary auditory cortex (Hochster & Kelly, 1981; Jenkins & Masterton, 1982; Jenkins & Merzenich, 1984; Al'tman, Rosenblum & Lvova, 1987; Cornelisse & Kelly, 1987; Kavanagh & Kelly, 1987; Cranford, Boose & Moore, 1990; Al'tman & Vaitulevich, 1993; Phillips, 1993; Poirier, Miljouis, Lassonde & Lepore, 1993; Poirier, Lassonde, Villemure, Geoffroy & Lepore, 1994; Zatorre, Ptito & Villemure, 1995).

Following this rationale, binaural masking procedures were developed by exploiting the temporal dimension of the acoustic signals while controlling for other dimensions (e.g. intensity). Target signal and background noise were presented to both ears using small interaural time delays (ITDs) varied from -600 to 600 μ s in 200 μ s steps. The coding and resolution of these ITDs are made at various levels of the central auditory

system (Irvine, 1986). However, animal and human lesions studies reviewed in Chapter 2 demonstrated that only complex tasks required the cortical involvement pertinent to our object of research.

The complexity of the task was reached by using two sounds, a gated white noise and a click, to which different ITDs were applied. The noise and the click signal could be perceived at the same or different positions in apparent location, as determined by ITDs between sound presented dichotically. The task of the subject was to detect the presence of the click signal in the background noise. This binaural task was not a sound localization task *per se* because the 2 sounds were not delivered from real spatial locations but instead interacted internally to form a simulation of space.

The click was preferred over other kinds of signals because it can be easily exploited in both behavioral and electrophysiological procedures. This is not the case for all acoustic signals and, in general, electrophysiological purposes are better served by the use of brief signals (Hall, 1992).

Another important aspect that influenced the development of the behavioral binaural masking procedure was the response mode required from the experimental subject. The response mode had to be simple enough to be used by *all* subjects with or without speech comprehension problems. To reach this goal, a detection response mode, similar to conventional audiometry, was adopted. The response mode was a 2 alternative forced choice. For each binaural stimulus presentation, the subject was asked to choose from 2 mouse buttons. One button, identified by a yellow «YES» tag, was associated with perceived detection of the target signal (click) through the masking noise regardless of its perceived spatial position. The other mouse button, identified by a red «NO» tag, was associated with perceived absence of the target signal and therefore the perception of a «noise-only» signal. The ability of the experimental subjects to use a button response mode was briefly assessed when hearing thresholds were obtained through conventional audiometry prior to the experimental procedures. All subjects involved in this research project successfully used the button response mode without any difficulty.

4.2.2. Behavioral procedure

Using Tucker-Davis Technology hardware (TDT) and a control software identified as Balance (Larivière, 1993), a target signal (2 ms alternating click), and a white masking noise (100 ms including 50 ms rise/fall time) were generated and transmitted binaurally to the subject through earphones (EAR-3A). The signal and noise were mixed together in a 100 ms temporal window. However, the target signal was started at the 49th ms of the temporal window. Balance allowed the independent control of: 1) signal interaural time difference (SITD) and noise interaural time difference (NITD), 2) the interaural intensity difference (IID) for both the signal and masking noise and 3) the signal-to-noise ratio (SNR). The manipulation of SITD, NITD, IID and SNR allowed the experimenter to move the sounds to different perceived positions within the head of the listener. Figure 7 illustrates the TDT instrumentation used in the behavioral experiment.

In this experiment, only a single masking noise condition was used with the NITD equal to 0 μ s, which was a location perceived in the middle of the head. We restricted the experiment to a single masking noise condition in order to limit the duration of the behavioral data collection for each subject to two hours.

The perceived location of the target signal was varied using SITDs from -600 to +600 μ s in 200 μ s steps in the presence of the noise. The perceived location of the signal was randomly varied from trial to trial. At all times, the IID was set at 0 dB for both the signal and the masking noise, therefore at an equal sound pressure level in the two ears. The SNR was randomly varied from -30 to +35 dB (in 5 dB steps). The noise level was fixed at 50 dB SPL (re: 0.2×10^{-5} Pa) whereas the click level was varied by 5 dB steps up to a maximum level of 90 dB SPL pe (peak equivalent). Figure 8 illustrates the various relations of the signal and the noise.

Figure 7 - Schematic description of the TDT instrumentation used in the behavioral procedure

Figure 8 - Description of the physical characteristics of the target signal and the masking noise and their temporal relationship

Each subject was given a pre-testing program prior to the data collection. The purpose of the pre-testing program was 1) to identify the lowest SNR for which perfect signal detection could be recorded; 2) to identify the SNR leading to a null signal detection performance; and 3) to familiarize the subject with the task and the response mode. The pre-testing program was comprised of 3 to 4 blocks of increasing level of difficulty in terms of signal detection (e.g. decreasing SNRs).

Each pre-testing block included 9 stimulus combinations (3 SITDs by 3 SNRs). The ITDs for the signal were randomly varied over 3 conditions 0 μ s, -600 μ s and + 600 μ s which corresponds to a perceived location in the centre of the head, right ear and left ear respectively. The ITD for the noise was fixed at 0 μ s which corresponds to a perceived location in the centre of the head. For the first block of the pre-testing program, 3 relatively high SNRs were purposely selected to obtain a perfect signal detection score and to familiarize the subject with the task. For each stimulus combination, 5 identical trials were presented to the subject. Catch trials (n=3), in which only noise was presented, were also introduced at each SNR to verify that the subject was correctly responding to both presence and absence of the target signal. Stimulus combinations and catch trials were completely randomized. A probability of signal detection was computed by Balance for each of the 9 combinations using the subject's responses to both stimulus and catch trials. Table 3 illustrates an example of the organization of one pre-testing block. Entries in the table are the number of identical trials presented for each of the 9 combinations of SNRs and SITDs.

Table 3 - Example of the organization of the initial pre-testing block.

	SITD	-600	0	600	Catch trials	Total N
SNR	20 dB	5	5	5	3	18
	15 dB	5	5	5	3	18
	10 dB	5	5	5	3	18
Total N		15	15	15	9	54

For each entry, the number indicates the amount of identical trials presented at each specific combination.

From one block of 9 combinations to the next, decreases in the SNRs were applied. The subject's performance on signal detection was successively evaluated until a floor effect was obtained (e.g. no detection of the target signal). For most subjects, this level of performance was achieved at SNRs between -10 and -15 dB. At this point, two subjects from the left MCA cva group were excluded from the behavioral procedure because they were unable to discriminate between signal-only and noise-only stimulus. For all the other subjects, once the floor effect was reached, data collection was begun.

The trials used for data collection were made up of twenty-eight different combinations (4 SNRs by 7 SITDs). The ITD for the signal was randomly varied from the right ear (SITD = - 600 μ s) to the left ear (SITD = + 600 μ s) in 200 μ s steps. Again, the ITD for the noise was fixed at 0 μ s. For each stimulus combination, 10 identical trials were presented to the subject. Catch trials ($n=7$), in which only noise was presented, were also introduced at each SNR to verify that the subject was correctly responding to both presence and absence of the target signal. The 4 SNRs were selected in order to cover, as far as possible, the entire range from perfect to null signal detection scores. However, in some cases, a second run at lower SNRs had to be conducted to reach the level of absolute null signal detection. In these cases, 35 to 42 combinations were obtained. Combination, stimulus, and catch trials were completely randomized within blocks made of different SNRs. A probability of signal detection was computed by Balance for each of the 28 combinations (or in some cases 35 to 42 combinations) using subject's responses to both stimulus and catch trials. Table 4 gives an example of the organization of one data collection block. Entries in the table are the number of identical trials presented for each of the 28 combinations of SNRs and SITDs.

Table 4 - Example of the organization of the data collection block. In some cases, additional combinations were needed to cover the entire detection range.

	SITD	-600	-400	-200	0	200	400	600	Catch	Total
SNR	0 dB	10	10	10	10	10	10	10	7	77
	- 5 dB	10	10	10	10	10	10	10	7	77
	-10 dB	10	10	10	10	10	10	10	7	77
	-15 dB	10	10	10	10	10	10	10	7	77
Total		40	40	40	40	40	40	40	28	308

For each entry, the number indicates the amount of identical trials presented at each specific combination.

The subject, wearing insert earphones (EAR 3A), was comfortably seated in an anechoic room at the Laboratory of Sensory Neuroscience at Carleton University. Using a serial computer mouse, the subject controlled the pace of stimulus presentation and chose his(her) detection response.

To examine the effect of auditory spatial attention upon this binaural masking task, reaction times (RTs) were also collected. To do so, Balance computed a mean RT for each of the 28 combinations. The mean RT was computed using all the RTs collected within a block of 10 trials regardless of the correct or incorrect nature of the detection response.

The choice to use all RTs, independently of the correctness of the response, was made on the basis that the experimental procedure was aimed at the identification of a specific low SNR for which the detection of the target signal would no longer be possible. At this point, the RTs should reflect a « fast » decision process related to the obvious *perceptual absence* of the target signal even if, in fact, the target signal was *physically present* and the behavioral response would be labelled as incorrect. In the same manner, the RTs should also reflect a « fast » decision process related to the obvious *perceptual presence* of the target signal at a high SNR, still a *physically present* signal but a

behavioral response that would be labelled as correct. Between these low and high SNR listening conditions were situations for which the task was influenced by the perceptual difficulty of detecting the target within the background noise, and therefore both the correct and incorrect detection responses would be made after a prolonged decision process. In these three listening conditions (low, high and intermediate SNRs), the tally of correct or incorrect nature of the behavioral response itself would not adequately reflect the perceptual process.

The only *truly incorrect* responses and their related RTs were associated with the catch trials. As described before, the catch trials, in which only noise was presented, were used at each SNR to verify that the subject was correctly responding to a physical absence of the target signal. For these catch trials, only the RTs related to correct responses, e.g. *perceptual absence* of the target signal for *physical absence* of the target signal, were averaged together. The RTs related to incorrect responses to catch trials, e.g. *perceptual presence* of the target signal for *physical absence* of the target signal, were rejected. This method allowed the examination of the RT associated with a non-contaminated correct perception of the real absence of the target signal .

The use of a serial Microsoft computer mouse to measure reaction time added a delay on each RT according to studies by Segalowitz & Graves (1990), Crosbie (1990) and Beringer (1992). This delay was equal as approximately 30 ms and included a time threshold to overcome bouncing key contacts and minimum transfer delay (Beringer, 1992). The RTs obtained in preliminary studies (Leroux & Kelly, 1995, see section 2.5.2) were well above the reported serial mouse delay to the order of 600 ms. The raw data were not corrected for this delay since all subjects and groups were affected by the same serial mouse delay.

It was shown by Leroux and Kelly (1995) that the probability of signal detection decreased when the target signal and the noise shared the same perceived spatial position and increased for signal positions away from the location of the noise. The variation in ability of the signal detection described a function referred to as a spatial channel. The

center of the spatial channel corresponded to the shared spatial position of signal and noise whereas the edges of the spatial channel corresponded to segregated spatial positions. This spatial channel function was determined from the signal detection probabilities given by Balance and then converted into a single number, the behavioral index of binaural advantage (BIBA). The BIBA was used to characterize the behavioral output of the measured spatial channel. A well-known analogy for the BIBA, although differently calculated, would be the Q factor used to characterize the filtering power of an acoustic filter. A high BIBA was obtained with high detection levels of the signal at SITDs only slightly different than 0 μ s (the ITD of the noise) that were associated at the same time to a lower detection level at 0 μ s. A low BIBA was obtained with detection levels of the signal that were similar for *all* SITDs. Therefore, the BIBA reflects the power of the signal detection in the presence of noise. A BIBA was computed for each SNR. The following equation [1] is the computational formula of the BIBA:

$$\frac{\text{Mean (PSD -600, PSD -400, PSD 400, PSD 600 } \mu\text{s}) - \text{Mean (PSD -200, PSD 0, PSD 200 } \mu\text{s})}{\text{Mean (PSD -600, PSD -400, PSD 400, PSD 600 } \mu\text{s)}} \quad [1]$$

where PSD is the Probability of Signal Detection at various SITDs.

4.2.3. Electrophysiological procedure

Using the same target and noise signals, subjects were also submitted to an electrophysiological procedure using a modified auditory evoked potential technique suited to the recording of N_1 and P_2 components. A preliminary study (see section 2.5.2) identified that, among a variety of components, N_1 and P_2 were the most clearly related to the spatial channel concept. Therefore, the procedure described here was designed to optimize the recording of N_1 and P_2 components.

A dual channel array of scalp-electrodes was placed on the subject's head: a *vertex* non-inverting electrode was referred to a linked inverting electrode applied to both ears (C_z-A_1/A_2) and a *frontal* non-inverting active electrode was referred to a linked inverting

electrode applied to both ears (F_z - A_1/A_2). Given the limited number of available recording channels (2), midline recording sites were selected for both frontal and vertex sites. It is well-known that cortical auditory evoked potentials are hemisphere-specific: the greater amplitude is usually observed over the hemisphere contralateral to the stimulated ear and, conversely, the smaller amplitude is usually observed over the hemisphere ipsilateral to the stimulated ear (Hall, 1992).

Electrodes and conductive gel were placed, after skin cleansing, along with a ground electrode on a frontal site (F_z). Subjects were asked to relax as much as possible. In addition, they were asked to pay attention to the signal heard in order to maximize cortical level responses. On-going EEG activity was monitored continuously during the recording. The TDT hardware and Auditory Evoked Potential (AEP) software allowed 2 channels of electrophysiological measurement. The parameters used for data collection are shown in Table 5. The SNRs used in the electrophysiological procedure were limited to the upper boundary of -5 dB. This limit was set in the light of preliminary measures which showed that a higher intensity of the rapidly presented click stimulus was associated with loudness discomfort for most of the normal subjects. This upper limit showed a difference compared to the upper limit used in the behavioral procedure (SNR of + 35 dB). The TDT hardware illustrated in Figure 7 was modified accordingly to allow auditory evoked potential measurement. The modifications are indicated in Figure 9.

Table 5 - Parameters used for electrophysiological data collection

Stimuli:	Binaural noise with 0 μ s ITD Binaural click with $\pm$ 600 μ s SITD (in 200 μ s steps)
Rate of stimulation:	1.7 presentations per second (pps)
Level of stimulation:	Signal level from 60 to 30 dB SPL pe (re: 0.2×10^{-5} Pa) Noise level fixed at 65 dB SPL (re: 0.2×10^{-5} Pa)
Amplification gain:	1×10^6 Grass amplifiers 1×10^6 AEP internal amplifiers
Filters settings:	1 Hz for high-pass filter and 100 Hz for low-pass filter 30 Hz low-pass filter off-line, roll-off rate: 48 dB/octave
Time window:	1- 300 ms
Artifact rejection:	55 % of threshold
Number of trials:	100 trials

Figure 9 - Schematic description of the TDT instrumentation used in the electrophysiological experiment

The data used in the electrophysiological procedure were taken from thirty-five different combinations (5 SNRs by 7 SITDs). The perceived spatial location of the signal was successively varied from the right ear (SITD = - 600 μ s) to the left ear (SITD = + 600 μ s) in 200 μ s steps. The perceived spatial location of the noise was fixed at the centre of the head (NITD = 0 μ s). For each stimulus combination, 100 identical trials were presented to the subject. The 5 SNRs were the same for all subjects and were selected in order to cover a wide dynamic range of signal detection. A very low SNR of -35 dB, where all subjects reported to hear only noise, was used as a baseline for random electrophysiological activity. Increasing levels of SNRs from -25 to -5 dB by 5 dB steps were then presented to replicate the behavioral procedure. The highest SNR, for which all subjects clearly reported the presence of the target signal at all perceived spatial positions, was used as an estimation of the electrophysiological activity related to the click signal regardless of its spatial position. The order of presentation from SITDs of - 600 to 600 μ s was pseudo-randomized from one subject to the other. For each run performed, 2 electrical waveforms, one for each electrode site, were obtained and stored. Off-line averages were computed at each SNR for 3 spatial locations: the right ear (SITDs of -600 and -400 μ s), the center (SITDs of -200, 0 and 200 μ s) and the left ear (SITDs of 400 and 600 μ s). These averages resulted in 3 averaged traces for each of the 5 SNRs at the 2 electrode sites for each subject. Each averaged trace was computed from 200 to 300 trials depending on the spatial location. Table 6 illustrates an example of the organization of one electrophysiological data collection block. Entries in the table are the number of identical trial blocks, each of 100 stimuli, averaged across the 3 groups of SITDs identified as Right, Center and Left.

Table 6 - Example of the organization of the electrophysiological data collection block.

Cz	SITD	Right	Center	Left	Fz	SITD	Right	Center	Left
	-35	3	2	3		-35	3	2	3
	-25	3	2	3		35	3	2	3
	-20	3	2	3		-20	3	2	3
	-15	3	2	3		-15	3	2	3
	-5	3	2	3		-5	3	2	3

The analysis was made on the individual averaged electrical waveforms. The purpose of the analysis was to identify pertinent late components (N_1 and P_2). For each component, latency (in ms) and amplitude (in nV) were measured and stored.

In addition, for the purpose of overall analysis, averaged curves were obtained for each group of subjects (normal, left and right) for each of the 5 SNRs at each electrode site. These averaged curves were computed from 7000 trials per SNR. The purpose of the group analysis was also to identify pertinent late components (N_1 and P_2). For each component, latency (in ms) and amplitude (in nV) were measured and stored.

In preliminary studies with young normal subjects (see section 2.5.2), it was shown that the amplitude of the N_1 and P_2 components reflected the behavioral signal detection. In other words, the N_1 - P_2 amplitude decreased when the target signal and the noise shared the same perceived spatial position and increased for signal positions away from the noise position. The variation of the N_1 - P_2 amplitude seemed to be related to the behavioral spatial channel concept. As in the behavioral procedure, the center of the spatial channel corresponded to the shared spatial position whereas the edges of the spatial channel corresponded to segregated spatial positions. A relationship, as defined by equation [2], was then determined from the N_1 - P_2 amplitude and then converted into a single number, the electrophysiological index of binaural advantage (EIBA). Similarly to the BIBA, the EIBA was used to characterize the electrophysiological output of the measured spatial

channel. A high EIBA was obtained with high amplitude of the N_1 - P_2 component at SITDs only slightly different than 0 μ s (the ITD of the noise) that were associated at the same time to a lower amplitude of the N_1 - P_2 component at 0 μ s. A low EIBA was obtained with amplitude of the N_1 - P_2 component that was similar for *all* SITDs. The EIBA reflects the amplitude variation of the N_1 - P_2 component as a function of the presence of noise. An EIBA was computed for each subject for each electrode site: EIBA(C_z) and EIBA(F_z). The following equation [2] is the computational formula of the EIBA:

$$\frac{\text{Mean (AMP Right, AMP Left)} - \text{Mean (AMP Center)}}{\text{Mean (AMP Right, AMP Left)}} [2]$$

where AMP is the Amplitude of the late component N_1 - P_2 at various SITDs.

4.2.4. Speech comprehension measures

In addition to acute speech comprehension measures provided in the chart review by the BDAE and the WAB, complementary measures were made to document chronic speech comprehension problems. These measures were carried out shortly before the experimental session.

This further examination of speech comprehension included three subtests of the Psycholinguistic Assessments of Language Processing in Aphasia (PALPA) (Kay, Lesser & Coltheart, 1992). Two subtests required the comprehension of a single word. The first of these subtests, the Minimal Pair Discrimination Requiring Picture Selection (MPDRPS), required the subject to match a single spoken word with a corresponding picture. All the words were monosyllabic with a Consonant-Vowel-Consonant structure. There was a choice of three drawings in which distractor objects had phonologically related names (e.g., coat, boat and goat). For this task, accurate selection of the target word was dependent upon both linguistic and auditory processing. In the MPDRPS, the nature of the errors made by the subject is retained for the following dimensions: location

of the error, initial/final consonant; voice, manner, place and occurrence of the target word, low/high.

In the one hand, intact auditory processing was needed to detect phonologically-related differences for this task. As suggested by Stevens and Blumstein (1978), Blumstein and Stevens (1979) and Blumstein and Stevens (1980), the place of articulation is cued by invariant acoustic properties found in the frequency spectrum in a 10-20 ms time window adjacent to consonantal onset or offset. According to Blumstein and Stevens (1979), the auditory system is endowed with detectors that are sensitive to these spectral changes. These detectors would therefore be involved in the classification process of speech sounds into their natural classes. On the other hand, intact linguistic processing was needed for phoneme identification. For example, one could acoustically characterize a spectral shape without being able to identify the exact stop-consonant. In the MPDRPS, additional phonological information was provided by the words semantic context to help the linguistic processing. The additional information provided by the linguistic context would limit the extent of a linguistically-related impairment. In contrast, for an auditory-related impairment, the linguistic context provided by the MPDRPS would be of no help (Kay et al., 1992).

In order to assess a more linguistically-dependent function of word comprehension, the second subtest, the Spoken Word-Picture Matching, was administered to the experimental subjects. This task used spoken word-picture matching to assess semantic comprehension ability. Four distracter pictures were included: a close semantic distractor from the same superordinate category (e.g., carrot-cabbage), a more distant semantic distractor (e.g., carrot-lemon), a visually similar distractor (e.g., carrot-saw) and an unrelated distractor (e.g., carrot-chisel). The unrelated and visually related distractors were related semantically to each other (but not to the target) to prevent the subject from making guesses based on the perceived semantic category (Kay et al., 1992). Subjects with impairments at the level of auditory processing may have performed at a higher level since there was no phonetically-similar items in this test whereas patients with a more

linguistic processing disorder would have a lower performance due to the semantic closeness of the test items.

The last subtest documented speech comprehension at the sentence level with the Sentence-Picture Matching: Auditory Version (SPMAV). This task used pictures to test comprehension of spoken sentences. A sentence level subtest was included in the chronic assessment of speech comprehension of our subjects because it has been repeatedly demonstrated that this ability is often one of the last to fully restore itself in aphasic patients (Davis, 1993). Moreover, recent work indicated that sentence processing deficits can occur for a variety of reasons, even in aphasic subjects who appear to have similar linguistic symptoms (Kay et al., 1992). One dimension that could explain these differences might be the presence of deficits with primary auditory processing skills. One of these auditory processings could be the perceptual segregation of successive acoustic events (Massaro, 1972).

This subtest contained four main types of sentences: reversible and non-reversible, gapped and converse relations. For each sentence there was a choice of three pictures: the target and two distractors. Distractor pictures varied with sentence construction and consisted of distractors in which the subject and object were reversed and/or distractors in which lexical substitutes were presented for the subject, object, verb or adjective (Kay et al., 1992).

All PALPA auditory items were recorded and played back using a high-fidelity UHER tape recorder. The items were spoken by an English native speaker. This was done to eliminate all visual cues (lip reading, facial expression, etc.) and to reduce the inter-subject variability usually introduced by the live spoken voice.

4.3. Data analysis

The data collected from the control group served as a normative database for comparison with the data obtained with the experimental groups. For each group, means

and variances were computed for BIBA, EIBA(C_z), EIBA(F_z), reaction time and speech comprehension measures. A series of analyses were used to answer three specific questions. First, was there a difference in sound localization abilities for the three groups ? Second, did the results of the behavioral and electrophysiological measures agree ? Third, was there any relationship between the sound localization measures and the speech comprehension tests and, if applicable, how strong was the relationship between these measures.

The first question was to determine whether the groups (normal, left and right cva) differed in terms of sound localization related measures used as dependent variables. BIBA, EIBA(C_z), EIBA(F_z) and the reaction time were submitted to a multivariate analysis of variance (MANOVA) (Stevens, 1992) using group membership as the factor (normal, left and right cva). MANOVA assumptions (normally distributed variables and homogeneity of variance-covariance matrices) were examined prior to analysis using the Box's M test. Univariate ANOVAs were also performed when permitted by the MANOVA results for each of the BIBA, the EIBA(C_z), the EIBA(F_z) and reaction time. Post-hoc univariate paired comparisons on groups were performed for significant dependent variables using a Bonferroni approach (Stevens, 1992).

The second question dealt with the similarity between the two sound localization measures. Thus, the pattern of results for both behavioral and electrophysiological methods were compared all together and for each group separately. In order to do so, Pearson-Product Moment correlation coefficients were computed involving BIBA, EIBA(C_z), EIBA(F_z).

The last question examined was the relationship between the measures of sound localization (temporal indices), and speech comprehension (PALPA). To achieve this purpose, a canonical correlation was computed, using BIBA, RT, EIBA(C_z), EIBA(F_z) and the speech comprehension measures across groups (Stevens, 1992).

To confirm our hypothesis, the statistical analysis would have to show a significant decrease either in the BIBA or EIBA for the experimental groups, reflecting a reduced ability to extract acoustic information coming from spatially overlapped locations. The statistical analysis of reaction time data should show a similar trend across right and left cva groups, suggesting that the binaural noise itself imposes an attentional distractor effect. The correlation coefficient analysis on BIBA and EIBA should validate the equivalency of the two measurements in terms of reflecting the same reduced ability to extract acoustic information from different spatial locations. The canonical correlation analysis using the sound localization related measures and speech comprehension measures should show at least one significant relationship. Establishing such a relationship would validate the hypothesis that the severity of the speech comprehension problem is related to the performance of patients on sound localization related tasks. Patients with right MCA cva should show better speech comprehension ability and better performance on sound localization tasks than patients with left MCA cva. Patients with left MCA cva should show impaired speech comprehension ability and poorer performance on sound localization related tasks.

Chapter Five

5. Results

5.1. Results from the behavioral procedure

The behavioral procedure led to two different sets of data. The first set is based on the probability of detection, which provides evidence regarding the spatial channel mechanism using the binaural masking procedure. The probabilities of signal detection gathered under this binaural masking procedure are presented in section 5.1.1. The individual data showing the probability of signal detection as a function of the SITDs for each SNR are not presented in the core of the text but are available in Appendix A. The second set is derived from response latencies, which provide evidence regarding the auditory spatial attention involved in the binaural masking procedure. The reaction times obtained under the various experimental conditions are presented in section 5.1.2. The individual data showing reaction times as a function of the SITDs for each SNR are not presented in the core of the text but are available in Appendix B.

5.1.1. Probability of signal detection

5.1.1.1. Data reduction approach

As illustrated by Table 4 (see section 4.2.2), a minimum of 28 different probabilities of signal detection were computed for each subject by Balance (Larivière, 1993). These probabilities of detection were grouped under a specific signal-to-noise ratio. Seven probabilities of signal detection, reflecting the 7 ITDs of the signal that were used, were associated with each SNR. As described in section 4.2.2, the data collected for each subject covered the entire field of detection performance from perfect to null signal detection.

The dynamic range of the detection field varied from one subject to another in terms of the number of SNRs needed to cover perfect to null detection performance. For most subjects, the expected dynamic range of 20 dB (four different SNRs) was insufficient to cover the whole detection field. A wider dynamic range up to 30 dB (six different SNRs) turned out to be necessary to cover the whole detection field. For individual subjects, maximal and minimal performance levels were reached at different SNRs.

For each subject, the performance levels at SNRs above maximal performance were assumed to remain asymptotic. Therefore to obtain groups means for these values, the actual individual highest (perfect or near perfect) probability of signal detection for each perceived spatial location was used for the remaining levels up to the SNR of 35 dB, the highest level encountered across the three groups of subjects. In the same manner, the performance levels at SNRs below minimal performance were assumed to remain asymptotic and for these levels, the detection probability associated with the catch trials was used. The catch trials, trials which contained no signal but noise, can be assumed to be similar to regular trials presented at an extremely low SNR. Therefore their associated probability of signal detection could also be used for low SNRs. The actual individual probabilities of signal detection associated with the catch trials were used down to the SNR of -30 dB, the lowest level encountered across the three groups of subjects.

The missing data procedure described above allowed us to obtain a complete matrix of data for each of the seven ITDs from -600 to 600 μ s at each of the 14 SNRs from 35 to -30 dB for all subjects. As explained in section 4.2.2., the BIBA (equation [1]) was computed for each of the 14 SNRs. To reduce these values to a single number for multivariate statistical purposes (see section 5.4), the maximal BIBA value reached was retained along with the specific SNR at which it was recorded and combined into a new index named BIBAMAX. To solve the problem of having a 0 value in computation, the SNRs in decibels were converted into an arbitrary positive integral scale from 1 to 14, each end corresponding respectively to 35 and -30 dB. The following equation [3] is the computational formula of the BIBAMAX:

$$\text{BIBAMAX} = (\text{largest BIBA X corresponding rescaled SNR})^2 \quad [3]$$

5.1.1.2. Results for the normal group

Figure 10 shows the averaged probability of signal detection (on the Y axis from 0 to 1) computed for each of the seven ITDs of the signal (on the X axis from -600 to 600 μs) for the subjects belonging to the normal group ($n=10$). The probabilities of signal detection were averaged across the 14 studied SNRs (35 to -30 dB) and across all subjects for the normal group. As previously noted, a SITD of -600 μs was associated with a perceived spatial location toward the right ear whereas a SITD of 600 μs referred to a perceived location toward the left ear. For all conditions, the noise interaural time difference (NITD) was fixed at 0 μs . In Figure 10, for the sake of clarity of presentation, only the SNRs from 10 to -30 dB are given; in the normal group, the probabilities of signal detection for SNRs of 15 dB and greater were exactly the same as those recorded at 10 dB as the result of the replacement procedure for missing data.

Figure 10 shows a progressive reduction in the probability of signal detection with decreasing SNRs from 10 to -30 dB. At SNR of 10 dB, the mean probability of signal detection was close to a value of 1. This performance progressively declined from one SNR to the next, down to the SNR of -30 dB for which the mean probability of signal detection was close to a value of 0. However, the reduction was unequal from one SITD to the other. The amplitude reduction was greater when signal and noise shared the same ITD of 0 μs . This ITD-specific decrease on the probability of signal detection at 0 μs can be observed at SNRs between -5 to -20 dB. At these SNRs, on each side of the shared

Figure 10 - Averaged probability of detection as a function of interaural time difference for the signal (SITD) for the normal group ($n = 10$)

ITD, either toward negative SITDs or positive SITDs, the probability of signal detection increased as the SITD differed from 0 μ s. The greatest reduction in the probability of signal detection at the shared ITD was observed at the SNR of -15 dB.

In Figure 10, the solid gray line (filled squares) corresponding to the SNR of -15 dB resembles the operating characteristics of a spatial channel: the probability of signal detection decreases toward a value of 0 (0.15 at 0 μ s) when target signal and noise share the same ITD, and increases toward a value of 1 when the target signal and noise are greatly separated as determined by their respective ITD (for example, probability of 0.55 at -600 μ s). This function was in agreement to the one reported for young normal listeners by Leroux and Kelly (1995). The detection function described by the normal group mean was fairly symmetric on each side of the shared ITD.

Figure 11 presents the normal group mean overall probabilities of signal detection (filled triangles), averaged across the 7 different SITDs, obtained as a function of the SNRs from 35 to -30 dB. Figure 11 also shows the normal group mean for the behavioral index of binaural advantage (BIBA) (filled squares) obtained for each SNR. The function described by the overall probabilities of signal detection obtained from the normal subjects recalls the articulation function for spondees (Hudgins, Hawkins, Karlin & Stevens, 1947). In the same manner, the function showed a steep portion for which the probability of detection went from 0.7 to 0.3 in a 5 dB range. The SNR at the midpoint of the detection function, arbitrarily fixed at a 0.5 probability of signal detection, was estimated to -13 dB. The normal group BIBA maximal value was reached at the SNR level of -15 dB at a value of 0.62. As expected for the spatial channel mechanism, the BIBA increased when the signal overall detection probability decreased from 0.80 to 0.32. The maximal BIBA was obtained after the detection function had dropped below 0.5. For low signal detection probabilities (SNRs $\leq$ -20 dB), the BIBA decreased down to a value of 0.

Figure 11 - Overall probability of signal detection and index of binaural advantage as a function of the signal-to-noise ratio for the normal group (n = 10)

5.1.1.3. Results for the left brain-damaged group

Figure 12 shows the averaged probability of signal detection (on the Y axis from 0 to 1) computed for each of the seven ITDs (on the X axis from -600 to 600 μ s) for the subjects belonging to the left brain-damaged group ($n = 9$). The probabilities of signal detection were averaged across the 14 studied SNRs for all the subjects of the left brain-damaged group. Again, the noise interaural time difference (NITD) was fixed at 0 μ s.

Figure 12 shows a progressive reduction in the probability of signal detection with decreasing SNRs from 35 to -30 dB. At SNR of 35 dB, the mean probability of signal detection was close to a value of 1. This performance progressively declined from one SNR to the next, down to the SNR of -30 dB for which the mean probability of signal detection was close to a value of 0. In contrast to the normal group, the reduction was similar from one perceived spatial location of the signal to the other. No significant spatial-specific decrease in the probability of signal detection could be observed for midline shared ITD at any of the SNRs. None of the obtained curves reproduced the operating characteristics of a spatial channel.

Figure 13 presents the left brain-damaged group mean overall probabilities of signal detection (filled triangles), averaged across the seven different SITDs, obtained as a function of the SNRs from 35 to -30 dB. Figure 13 also shows the left brain-damaged group mean BIBA (filled squares) obtained for each SNRs. In this group, the probability of detection went from 0.6 to 0.3 in a 15 dB range compared to a 5 dB range in the normal group for the same perceptual decrease. The SNR at the midpoint of the detection function was estimated to -5 dB which was 8 dB higher than the normal group. The left brain-damaged group mean BIBA maximal value was reached at the SNR level of -10 dB at a value of 0.13. The BIBA was fairly stable around 0.10 between SNR levels of 0 and -20 dB leading to a plateau of performance. In contrast to the normal group, no clear maximal BIBA peak could be identified. Negative value BIBAs were also obtained for the left brain-damaged group between SNR levels of 20 to 5 dB. These negative values

Figure 12 - Averaged probability of detection as a function of the SITD for the left lesion group (n = 9)

Figure 13 - Overall probability of signal detection and Index of binaural advantage as a function of the signal-to-noise ratio for the left lesion group (n = 9)

are associated with an easier detection of the signal at the shared ITD ($0 \mu\text{s}$, $\pm 200 \mu\text{s}$) when compared to other ITDs (± 600 , $\pm 400 \mu\text{s}$). For low signal detection probabilities ($\text{SNRs} \leq -25 \text{ dB}$), the BIBA decreased down to a value of 0.

5.1.1.4. Results for the right brain-damaged group

Figure 14 shows the averaged probability of signal detection (on the Y axis from 0 to 1) computed for each of the seven ITDs (on the X axis from -600 to $600 \mu\text{s}$) for the subjects belonging to the right brain-damaged group ($n = 10$). The probabilities of signal detection were averaged across the 14 studied SNRs for all the subjects of the right brain-damaged group. For all conditions, the noise interaural time difference (NITD) was fixed at $0 \mu\text{s}$. In Figure 14, for the sake of clarity of presentation, only the SNRs from 15 to -30 dB are given; in the right brain-damaged group, the probabilities of signal detection for SNRs of 20 dB and greater were exactly the same as the one recorded at 15 dB as the result of the replacement procedure for missing data.

Figure 14 shows a progressive reduction in the probability of signal detection with decreasing SNRs from 15 to -30 dB . At the SNR of 15 dB , the mean probability of signal detection was close to a value of 1. This performance progressively declined from one SNR to the next, down to the SNR of -30 dB for which the mean probability of signal detection was close to a value of 0. Similar to the normal group, the reduction was unequal from one ITD of the signal to another. The amplitude reduction was greater when both signal and noise shared the same ITD of $0 \mu\text{s}$. This ITD-specific decrease of the probability of signal detection for a midline shared ITD was observed at SNRs between 0 to -15 dB . At these SNRs, on each side of the shared ITD, either toward negative SITDs or positive SITDs, the probability of signal detection increased as the SITD differed from $0 \mu\text{s}$. The greatest reduction in the probability of signal detection at the shared ITD is observed for SNRs of -10 and -15 dB .

Figure 14 - Averaged probability of detection as a function of the SITD for the right lesion group ($n = 10$)

The solid gray lines corresponding to the SNR level of -10 and -15 dB in Figure 14 reproduce the operating characteristics of a spatial channel: the probability of signal detection decreased toward a value of 0 (0.32 at 0 μ s for SNR of -10 dB; 0.09 at 0 μ s for SNR of -15 dB) when target signal and noise shared the same ITD and increased toward a value of 1 when the target signal and noise differed by their respective interaural time differences (for example: 0.61 at -600 μ s for SNR of -10 dB; 0.30 at -600 μ s for SNR of -15 dB). This function is similar to the one reported for young normal listeners by Leroux and Kelly (1995). The detection function described by the right brain-damaged group mean is fairly symmetric on each side of the shared ITD. In contrast to the normal group, the spatial channel curve obtained with the right brain-damaged group was wider in terms of interaural time difference range and not as deep as that for normals. This means that the presence of the noise at the 0 μ s ITD affected the right brain-damaged subjects' signal detection performance over a greater temporal range when compared to normals. Moreover, at the same SNR of -15 dB, the signal detection function was similar for both groups (e.g. both curves showed the operating characteristics of a spatial channel) but the right brain-damaged group exhibited a lower mean detection performance.

Figure 15 presents the right brain-damaged group mean overall probabilities of signal detection (filled triangles), averaged across the seven different SITDs, obtained as a function of the SNRs from 35 to -30 dB. Figure 15 also shows the right brain-damaged group mean BIBA (filled squares) obtained for each SNR. The function showed a steep portion for which the probability of detection went from about 0.7 to 0.3 in a 7 dB range which is fairly comparable to the normal group. The SNR at the midpoint of the detection function was estimated to -9 dB. The right brain-damaged group BIBA maximal value was reached at the SNR level of -15 dB at a value of 0.50. As expected with respect to the spatial channel mechanism, and similarly to the normal group, the BIBA increased when the signal overall detection probability decreased from 0.90 to 0.19. The maximal BIBA was obtained after the detection function had decreased to

Figure 15 - Overall probability of signal detection and index of binaural advantage as a function of the SNR for the right lesion group (n = 10)

below 0.5. For low signal detection probabilities ($\text{SNR} = -30 \text{ dB}$), the BIBA decreased down to a value of 0.

5.1.2. Reaction time

5.1.2.1. Data reduction approach

As illustrated by Table 2 (section 4.2.2), a minimum of 28 mean reaction times (RTs) were computed for each subject by Balance (Larivière, 1993). These mean RTs were grouped by SNR. Eight RTs, reflecting the seven ITDs for the signal that were tested plus one RT related to the catch trials, were associated with each SNR. As already described in section 4.2.2 and in section 5.1.1, binaural masking data were collected for each subject in order to cover the entire field of detection performance from perfect to null signal detection.

As already explained in section 5.1.1.1., for most subjects, the expected dynamic range of 20 dB (4 different SNRs) was insufficient to cover the whole detection field. A wider dynamic range, up to 30 dB (six different SNRs), was necessary to cover the whole detection field. For individual subjects, maximal and minimal performance levels were reached at different SNRs.

For each subject, the RTs at SNRs above maximal performance were assumed to remain asymptotic, therefore to obtain groups means for these values, the actual median RT (from the seven available mean RTs at each tested SNR) associated with the highest (perfect or near perfect) probability of signal detection was used for the remaining levels up to the SNR of 35 dB, the highest SNR encountered across the three groups of subjects. In the same manner, the RTs at SNRs below minimal performance were assumed to remain asymptotic and for these levels, the median RT associated with all the available catch trials was used (the number of catch trial RTs depended on the number of tested SNRs). The catch trials, trials for which no signal but noise was present, can be assumed to be similar to regular trials presented at an extremely low SNR. Therefore the RTs for

these catch trials can also assumed to be similar to RTs obtained at low SNRs. The median RT associated with the catch trials was used down to the SNR of -30 dB, the lowest level encountered across the three groups of subjects. In this procedure, the median RT was used because of its known robustness to extreme values which have been shown to influence the mean RT. Computations using the mean RT were also made and no significant differences were found in the individual results. However, note that group data was analysed using the mean RTs computed with individual median RTs. This choice was made after the data distribution was checked for significant skewness or kurtosis. To reduce all the individual values to a single number for multivariate statistical purposes (see section 5.4), an averaged RT was computed for each subject across all SNRs.

5.1.2.2. Effect of the perceived spatial position of the signal on the RT

Figure 16 shows the averaged RTs (on the Y axis from 0 to 1200 ms) computed for each of the seven ITDs of the signal (on the X axis from -600 to 600 μ s) for each group. The RTs were averaged across the 14 studied SNRs (35 to -30 dB). For all conditions, the noise interaural time difference (NITD) was fixed at 0 μ s. The means are shown with ± 1 standard deviation.

It can be seen in Figure 16 that the normal group exhibited, for all SITDs, the fastest average RTs whereas the left brain-damaged group presented the slowest RTs. The RTs collected for the right brain-damaged group were faster, for all SITDs, than the left brain-damaged group, but slower than the normal group. Within each group, the RTs seem to be fairly stable from one perceived spatial position to the other. No significant ITD-specific increase of the RT could be observed for a midline shared location for any of the groups. None of the obtained curves reproduced the operating characteristics of a spatial channel.

Figure 16 - Group mean reaction time as a function of the SITD

It was expected that the RTs would be slower when the target signal and noise shared the same spatial position (ITD of 0 μ s). A slower RT could be associated with the perceptual difficulty of detecting the target signal in the background noise. A faster RT could be associated either with a level for which the signal intensity is high and easy to detect (which would be the case for SITD $\neq$ 0 μ s) or with a level for which the signal is obviously absent (which would be the case at the shared spatial position at a low SNR). This analysis suggests that the RT at the shared ITD would depend on the SNR: at a high SNR, the signal would be easily detected leading to a fast RT (signal-present response type); at intermediate SNR, the signal would be difficult to detect leading to a slow RT (a mix of signal-present / no-signal-noise-only response types); finally at low SNR, the signal would be perceived as obviously absent leading again to a fast RT (no-signal-noise-only response type). The next sections examine the effect of the SNR on the RT and the effect of the interaction of the SNR and the SITD on the RT.

5.1.2.3. Effect of the SNR on the RT

Figure 17 shows, for each group, the mean RTs (Y axis from 0 to 1400 ms) averaged across the 7 different SITDs, obtained as a function of the SNRs from 35 to -30 dB (X axis from 35 to -30 dB). As expected, in all groups, the mean RTs progressively increased as the target signal became increasingly difficult to detect in the background noise. After the target signal reached the subthreshold level (at very low SNRs), the mean RTs progressively decreased.

For both the normal and right brain-damaged groups, the slowest RTs were reached at a SNR of -15 dB. The global value of the function was slower for the right brain-damaged group but the shape of the function was very similar to that of the normal group. Both RT functions are in good agreement with the BIBA functions already described for the normal and right brain-damaged groups (Figures 11 and 15). In both groups, the maximal BIBA was also reached at a SNR of -15 dB. For the left brain-damaged group,

Figure 17 - Group mean RTs as a function of the signal-to-noise ratio

the slowest RT was reached at a SNR of -10 dB, which is also in good agreement with the BIBA function (Figure 13). As with the BIBA function, the shape of the RT function exhibited a performance plateau between the SNRs of -5 and -10 dB. The slowest RTs for the right and left brain-damaged groups was similar (1168.7 vs 1211.2 ms) but occurred at a different SNR (-15 vs -10 dB).

5.1.2.4. Effect of the interaction of the SNR and the SITD on the RT

Within each group, RT data were combined to explore the effect of the interaction of the SNR and the ITD for the signal on the detection decision as reflected by the RT. RT data associated with SITDs of -600 and -400 μ s were averaged together and named according to the perceived side of the spatial position of the signal, in this case, the right ear ('Right'). In the same manner, RT data associated to SITDs of -200, 0 and 200 μ s were averaged together and named 'Center' whereas RT data associated to SITDs of 600 and 400 μ s were averaged together and named 'Left'. This approach allowed the examination of the RTs for each tested SNR at a specific perceived spatial position for the signal. It was believed that the detection decision differed from one perceived spatial position to the other because of the presence of the noise in the 'Center' position. It was expected that, for the listening conditions associated with the highest BIBA, slower RTs would be recorded for 'Right' and 'Left' positions while, at the 'Center' position, the RTs would be faster since the target signal would be barely detectable (no-signal-noise-only response type) because of the presence of the noise.

5.1.2.4.1. Results for the normal group

Figure 18 shows the normal group mean RTs (on the Y axis from 0 to 1400 ms) obtained for the detection of the target signal at the 'Right', 'Left' and 'Center' locations for each tested SNR (on the X axis from 35 to -30 dB). As expected, at the level for which the maximal BIBA was reached (SNR of -15 dB), the RT associated with the detection of the target signal in the 'Center' location was faster (882.8 ms) than

Figure 18 - Mean RT measured for the detection of the signal for 3 spatial positions as a function of the SNR for the normal group (n = 10)

the RTs obtained for the 'Right' and 'Left' locations (1018.6 and 1037.1 ms respectively). It is believed that, in fact, the target signal was for most normal subjects undetectable, which led to a fast response of the no-signal-noise-only type. At a SNR of -15 dB, the presence of the noise interfered with the detection of the target signal at remote locations ('Right' and 'Left') which was expressed by the slower RTs obtained at these locations. It is interesting to note that the RTs obtained for the detection of the target signal at the 'Right' and 'Left' locations were similar to each other, as was the case with their probabilities of detection.

At the SNR of -10 dB, the opposite pattern was observed: the slowest RT was obtained for the 'Center' location (793.5 ms) whereas the 'Right' and 'Left' locations led to faster RTs (735.6 ms and 679.5 ms respectively). At this SNR, it is hypothesized that the normal listeners experienced more trouble detecting the target signal at the shared spatial location (a mix of signal-present / no-signal-noise-only response types) than at other spatial locations (signal-present response type). At the SNR of -10 dB, the presence of the noise in the 'Center' location interfered, marginally, with the detection of the target signal at remote locations ('Right' and 'Left'), which was expressed by the faster RTs obtained at these locations.

At the SNR of -20 dB, the fastest RT was obtained for the 'Center' location (772.1 ms) whereas the 'Right' and 'Left' locations led to slower RTs (818.7 ms and 882.4 ms respectively). The differences observed between RTs from each perceived spatial location were smaller at this SNR when compared to the results obtained at -15 dB. This pattern of results was also in good agreement with the smaller BIBA obtained with the detection responses at the SNR of -20 dB when compared to what was obtained at -15 dB. At the SNRs of -25 and -30 dB, the observed RTs were similar for each location. At this point, the target signal fell under the detection threshold regardless of its location.

5.1.2.4.2. Results for the left brain-damaged group

Figure 19 shows the left brain-damaged group mean RTs (on the Y axis from 0 to 1400 ms) obtained for the detection of the target signal at the 'Right', 'Left' and 'Center' locations for each of the tested SNRs (on the X axis from 35 to -30 dB). As expected, at the level for which the maximal BIBA was reached (SNRs of -5 and -10 dB), the RT associated with the detection of the target signal in the 'Center' location was faster (1101.3 and 1169.4 ms for SNRs of -5 and -10 dB respectively) than the RTs obtained for the 'Left' locations (1230.3 and 1294.5 ms for SNRs of -5 and -10 dB respectively). For the SNR of -10 dB, the RT measured at the 'Right' location showed no difference from the RT obtained at the 'Center' location (1190.5 vs 1169.4 ms respectively). This absence of difference suggests that the target signal was not detectable when sent to either the shared position or the ear opposite to the side of the brain lesion, which led to a no-signal-noise-only response type. This trend was also confirmed by the observation that the RTs for 'Center' and 'Right' locations stayed the same from the SNR of -10 to -30 dB, at which point the signal was clearly at the subthreshold detection level.

One important observation is also the fact that the 'Center' location predominantly led to the fastest RTs across the vast majority of the tested SNRs. These fast RTs would be usually associated either with an obvious no-signal-noise-only or a clear signal-present response type.

5.1.2.4.3. Results for the right brain-damaged group

Figure 20 shows the right brain-damaged group mean RTs (on the Y axis from 0 to 1400 ms) obtained for the detection of the target signal at the 'Right', 'Left' and 'Center' locations for each of the tested SNRs (on the X axis from 35 to -30 dB). As expected, at the level for which the maximal BIBA was reached (SNR of -15 dB), the RT

Figure 19 - Mean RT measured for the detection of the signal for 3 spatial positions as a function of the SNR for the left lesion group (n = 9)

Figure 20 - Mean RT measured for the detection of the signal for 3 spatial positions as a function of the SNR for the right lesion group (n=10)

associated with the detection of the target signal in the 'Center' location was faster (1151.3 ms) than the RTs obtained for the 'Right' locations (1214.3 ms). The RT measured at the 'Left' location showed no difference from the RT obtained at the 'Center' location (1154.1 vs 1151.3 ms respectively). This absence of difference suggests that the target signal was not detectable when sent to either the shared position or the ear opposite to the side of the brain lesion, which led to a no-signal-noise-only response type. It is interesting to note that this pattern of results was also found for the left brain-damaged group.

At the SNR of -10 dB, the slowest RTs were obtained for the 'Left' location (1010.9 ms) whereas the 'Center' and 'Right' locations led to faster RTs (928.5 and 925.0 ms respectively). At this level, the presence of the noise seemed to selectively increase the duration of the decision process for spatial location at the left ear (contralateral to the right brain lesion). In contrast, this observation was not made for the normal group for which, as expected, the RT related to the 'Center' location was the slowest.

At the SNR of -20 dB, the pattern of the obtained RTs for the three locations was similar to the one observed with the normal group, with the fastest RT recorded for the 'Center' location and the slowest RT obtained for the 'Left' one. At the SNRs of -25 and -30 dB, the observed RTs were not significantly different for the three locations. At this point, the target signal fell under the detection threshold regardless of its location, an observation that was also made for the two other groups.

5.2. Results from the electrophysiological procedure

5.2.1. Data reduction approach

The initial analysis examined the effect of the signal-to-noise ratio on amplitude and latency of the the N_1 and P_2 components. This analysis was performed upon the characteristics of components that were measured on individual averaged electrical waveforms computed across tested SITDs from -600 to 600 μ s. For each subject, the averaged electrical waveform was comprised of 700 identical trials per subject. These individual waveforms were then averaged within each group to form a group averaged waveform. The group waveforms included 7000 identical trials per SNR obtained from 10 subjects per group. The amplitude and latency values are presented for each of two electrode sites: C_z and F_z . For both recording sites, for all groups, the amplitude of the N_1 - P_2 component was expected to grow with the SNR whereas the latency was expected to decrease as the SNR would increase. The overall amplitude was expected to be smaller at the F_z recording site when compared to the C_z site.

Subsequent analyses were made on the amplitude of the N_1 - P_2 component that was measured on individual averaged electrical waveforms to explore the effect of the interaction of the SNR and 3 perceived spatial positions of the signal. Individual electrical waveforms associated to SITDs of -600 and -400 μ s were averaged together (200 trials), and named according to the perceived side of the spatial position of the signal, in this case, the right ear ('Right'). In the same manner, individual electrical waveforms associated to SITDs of -200, 0 and 200 μ s were averaged together (300 trials), and named 'Center' whereas individual electrical waveforms associated to SITDs of 600 and 400 μ s were averaged together (200 trials), and named 'Left'. These individual waveforms were then averaged within each group to form group averaged waveforms related to the 3 spatial positions of the signal. These group waveforms were comprised of 10 000 and 15 000 trials at different spatial locations. Again, the group waveforms are presented for each of two electrode sites: C_z and F_z .

It was believed that the amplitude of the N_1 - P_2 component would differ from one perceived spatial position to another because of the presence of the noise in the 'Center' location. It was expected that, at low SNR, a greater amplitude of the N_1 - P_2 component would be recorded for 'Right' and 'Left' locations, whereas at the 'Center' location, the amplitude would be smaller since the target signal would be barely detectable. At high SNR, it was expected that the amplitude of the N_1 - P_2 component would be greater at the 'Center' location and smaller for 'Right' and 'Left' locations which is normally the case with binaural stimulation using a null interaural time difference at a midline recording site.

As explained in section 4.2.3., the electrophysiological index of binaural advantage (EIBA, see equation [2]) was computed for each of the 5 SNRs. To reduce these values to a single number for multivariate statistical purposes (see section 5.4), the maximal EIBA value reached was retained along with the specific SNR at which it was recorded and combined into a new index named EIBAMAX. To obtain, with EIBAMAX, the same kind of scale used in the BIBAMAX computation, the SNR in decibels was also converted into an arbitrary positive integral scale from 1 to 5, each end corresponding respectively to -5 and -35 dB. The following equation [4] is the computational formula of the EIBAMAX:

$$\text{EIBAMAX} = (\text{largest EIBA X corresponding rescaled SNR})^2 \quad [4]$$

The individual data showing the electrophysiological curves obtained as a function of the SITDs are not presented in the core of this text but are available in Appendix C.

5.2.2. Results of the initial analysis

5.2.2.1. Amplitude

Figure 21 reveals the group mean amplitude of the N_1 - P_2 component of the cortical auditory evoked potential recorded at the C_z site (on the Y axis from 0 to 450 nV) as a function of the signal-to-noise ratio (on the X axis from -25 to -5 dB). The three groups are identified separately by filled black, left diagonal and right diagonal boxes respectively, for normal, left and right brain-damaged groups. In the same manner, Figure 22 gives the group mean amplitude of the N_1 - P_2 component of the cortical auditory evoked potential recorded at the F_z site.

At the C_z site (Figure 21), as expected, the overall amplitude of the N_1 - P_2 component grew fairly linearly from the lowest to the highest studied SNR for all groups. The overall amplitude was the largest, at each SNR, for the normal group, whereas it was the smallest for the right brain-damaged group. The overall amplitude of the N_1 - P_2 component for the left brain-damaged group was larger, at each SNR, when compared to the right brain-damaged group but smaller as compared to the normal group.

As expected, the overall amplitude for all groups and listening conditions was smaller at the F_z recording site (Figure 22) when compared to the C_z site (Figure 21). At the F_z site, for the normal group, the overall amplitude of the N_1 - P_2 component was fairly stable from SNRs of -25 to -15 dB, but increased sharply at the SNR of -5 dB. A linear growth of the amplitude of the N_1 - P_2 component was observed from one SNR to another for the left brain-damaged group. The same trend was seen for the right brain-damaged group except for the highest SNR at which an amplitude roll-over was measured. The same pattern of amplitude difference between groups was observed at both the F_z and C_z recording sites. At the lower SNRs (-25 and -20 dB), the amplitude of the N_1 - P_2 component was similar for both left and right brain-damaged groups. It is interesting to

Figure 21 - Overall amplitude of the N1-P2 component measured at Cz site as a function of the SNR for the 3 groups

Figure 22 - Overall amplitude of the N1-P2 component measured at Fz site as a function of the SNR for the 3 groups

note however, that for these SNRs, the absolute overall amplitude for brain-damaged groups was smaller than 100 nV.

Taken as a whole, the variations of the amplitude of the N_1 - P_2 component for both the C_z and F_z recording sites and for each group reflected the SNR increments from -25 to -5 dB. In subsequent analyses, the amplitude dimension was examined to explore the effect of the interaction of the SNR and the apparent location of the signal.

5.2.2.2. Latency

Figure 23 shows the group mean latency of the N_1 component of the cortical auditory evoked potential recorded at the C_z site (on the Y axis from 0 to 140 ms) as a function of the signal-to-noise ratio (on the X axis from -25 to -5 dB). The three groups are identified separately by filled black, left diagonal and right diagonal boxes respectively, for normal, left and right brain-damaged groups. In the same manner, Figure 25 reveals the group mean latency of the N_1 component of the cortical auditory evoked potential recorded at the F_z site.

At the C_z site (Figure 23), the overall latency of the N_1 component was stable from one SNR to the other for all groups. The only exception was the longest latency recorded at the SNR of -25 dB for the normal group which differed by a mean difference of 18.3 ms as compared to the other, higher, SNRs (-20, -15 and -5 dB). The mean difference between the latency of the latter levels was only 3.5 ms. Except for the lowest SNR, the latency of the N_1 component was shorter for the normal group as compared to the two brain-damaged groups. However, the amplitude of the between-groups averaged difference is small (8.6 ms) and not significant according to clinical criteria (Hall, 1992).

At the F_z site (Figure 25), a similar stability of the overall latency of the N_1 component was noted from one SNR to another for all groups. Unlike the latencies measured from the C_z site, this latency stability was without exception. Moreover, there was no significant overall latency difference from one group to the other.

Figure 24 reveals the group mean latency of the P_2 component of the cortical auditory evoked potential recorded at the C_z site (on the Y axis from 0 to 180 ms) as a function of the signal-to-noise ratio (on the X axis from -25 to -5 dB). The three groups are identified separately by filled black, left diagonal and right diagonal boxes respectively, for normal, left and right brain-damaged groups. In the same manner, Figure 26 gives the group mean latency of the P_2 component of the cortical auditory evoked potential recorded at the F_z site.

Figure 23 - Overall latency of the N1 component measured at Cz site as a function of the SNR for the 3 groups

Figure 24 - Overall latency of the P2 component measured at Cz site as a function of the SNR for the 3 groups

Figure 25 - Overall latency of the N1 component measured at the Fz site as a function of the SNR for the 3 groups

Figure 26 - Overall latency of the P2 component measured at Fz site as a function of the SNR for the 3 groups

At the C_z site (Figure 24), the overall latency of the P_2 component was stable from one SNR to the other for all groups. The only exception was the longest latency recorded at the SNR of -25 dB for the normal group which differed by a mean difference of 21.1 ms compared to the other, higher, SNRs (-20, -15 and -5 dB). The mean difference between the latency of the latter levels was only 1.3 ms. This pattern was also found for the N_1 component measured at the same recording site. Except for the lowest SNR, the latency of the P_2 component was similar among groups.

At the F_z site (Figure 26), a similar stability of the overall latency of the P_2 component was noted from one SNR to the other for all groups. This latency stability was without exception as opposed to the findings at the C_z site. The latency of the P_2 component was consistently shorter for the right brain-damaged group as compared to the other groups. However, the amplitude of the between-groups averaged difference is small (7.4 ms) and not significant according to clinical criteria (Hall, 1992).

The group data for the latency of N_1 and P_2 components did not seem to reflect the perceptual change related to the increase of the target signal amplitude. A latency decrease was expected with increasing SNR but this phenomenon was not demonstrated by our data. The variations observed here were small from one SNR to the other. These results however, are in agreement with a preliminary study (see section 2.5.2) using a smaller sample size ($n = 3$). In that study, small latency variations were also observed along with large amplitude variations related to the SNR increase. Due to these results, the latency dimension was not examined in subsequent analyses.

5.2.3. Interaction of the SNR and the SITD on N_1 - P_2 amplitude

5.2.3.1. Results for the normal group

Figure 27 gives the normal group mean amplitude of the N_1 - P_2 component of the cortical auditory evoked potential recorded at C_z (on the Y axis from 0 to 900 nV) as a

Figure 27 - Mean amplitude of the N1-P2 component measured at the Cz site as a function of the SNR at 3 perceived spatial locations for the normal group (n = 10)

function of the signal-to-noise ratio (on the X axis from -25 to -5 dB) for three locations ('Right', 'Center' and 'Left'). In the same manner, Figure 28 gives the normal group mean amplitude of the N_1 - P_2 component of the cortical auditory evoked potential recorded at the F_z site.

Figure 27 reveals a clear reduction in the amplitude of the N_1 - P_2 component for the 'Center' location at the SNR of -15 dB. At the same SNR, the amplitude of the N_1 - P_2 component for the 'Right' and 'Left' locations were clearly greater than what was obtained for the 'Center' location. These results suggest the presence of a spatial channel and are similar to those that were obtained with young normal listeners as described in section 2.5.2. The specific SNR at which the spatial channel operating characteristics appeared using the electrophysiological method was the same as with the behavioral method (-15 dB in Figure 11).

For SNRs of -15 and -5 dB, the amplitude of the N_1 - P_2 component stayed fairly stable for the 'Right' and 'Left' locations, whereas for the 'Center' location, the amplitude sharply increased with the SNR. This was also in agreement with the behavioral results for which a steep increase of the overall detection function was also observed between -15 and -5 dB (see Figure 11). At the SNR of -5 dB, the amplitude of the 'Center' location was clearly greater than each of the two side locations, which could be explained by the use of midline recording sites (C_z and F_z). As discussed in section 4.2.3., cortical auditory evoked potentials are hemisphere-specific: a greater amplitude is usually observed over the hemisphere contralateral to the stimulated ear and, conversely, a smaller amplitude is usually observed over the hemisphere ipsilateral to the stimulated ear (Hall, 1992). In the case of binaural stimulation using various interaural time differences, the greatest amplitude is expected over the midline for a null interaural difference.

Figure 28 reveals a clear reduction in the amplitude of the N_1 - P_2 component for the 'Center' location at the SNR of -25 dB, which was 10 dB lower than the response obtained at the C_z site. At -25 dB, the amplitude of the N_1 - P_2 component for the 'Right'

Figure 28 - Mean amplitude of the N1-P2 component measured at the Fz site as a function of the SNR at 3 perceived spatial locations for the normal group (n = 10)

and 'Left' locations was clearly greater than what was obtained for the 'Center' location. At the SNR of -15 dB, no clear reduction was detected for the 'Center' location. These results also suggest the presence of a spatial channel but at a lower level than at the C_z site. Similarly to the C_z site at the SNR of -5 dB, the amplitude obtained for the 'Center' location was clearly greater than each of the 2 side locations which could be explained by the use of midline recording sites (C_z and F_z). Globally, the amplitude of the N_1 - P_2 component measured at the F_z site was smaller than the amplitude obtained at the vertex recording site.

5.2.3.2. Results for the left brain-damaged group

Figure 29 reveals the left brain-damaged group mean amplitude of the N_1 - P_2 component of the cortical auditory evoked potential recorded at C_z (on the Y axis from 0 to 700 nV) as a function of the signal-to-noise ratio (on the X axis from -25 to -5 dB) for three locations ('Right', 'Center' and 'Left'). In the same manner, Figure 30 gives the left brain-damaged group mean amplitude of the N_1 - P_2 component of the cortical auditory evoked potential recorded at the F_z site.

Figure 29 reflects a clear reduction in the amplitude of the N_1 - P_2 component for the 'Center' location for almost all SNRs, with the exception of the SNR of -5 dB for which all locations showed similar amplitude. Taking into account the latter mentioned exception, the amplitude of the N_1 - P_2 component for the 'Right' and 'Left' locations was clearly greater than what was obtained for the 'Center' location. These results suggest the presence of a strong spatial channel for almost all studied SNRs in the left brain-damaged group. This finding is opposed to the behavioral data which failed to identify any significant spatial effect for this group (see Figure 13). The amplitude difference observed between 'Center' and 'Right' / 'Left' locations was greater in this group than in the normal group.

Figure 30, for the frontal recording site, does not clearly show a reduction in the amplitude of the N_1 - P_2 component for the 'Center' location for most SNRs with the

Figure 29 - Mean amplitude of the N1-P2 component measured at the Cz site as a function of the SNR at 3 perceived spatial locations for the left lesion group (n=10)

Figure 30 - Mean amplitude of the N1-P2 component measured at the Fz site as a function of the SNR at 3 perceived spatial locations for the left lesion group (n = 10)

exception of the SNR of -25 dB for which a small reduction was observed. Globally, the amplitude of the N_1 - P_2 component measured at the F_z site was smaller than the amplitude obtained at the vertex recording site.

5.2.3.3. Results for the right brain-damaged group

Figure 31 reveals the right brain-damaged group mean amplitude of the N_1 - P_2 component of the cortical auditory evoked potential recorded at C_z (on the Y axis from 0 to 900 nV) as a function of the signal-to-noise ratio (on the X axis from -25 to -5 dB) for three locations ('Right', 'Center' and 'Left'). In the same manner, Figure 32 gives the right brain-damaged group mean amplitude of the N_1 - P_2 component of the cortical auditory evoked potential recorded at the F_z site.

Figure 31 demonstrates a reduction in the amplitude of the N_1 - P_2 component for the 'Center' location at the SNR of -15 dB. At the same SNR, the amplitude of the N_1 - P_2 component for the 'Right' and 'Left' locations were greater than what was obtained for the 'Center' location. These results suggest the presence of a spatial channel that was, however, weaker as compared to the normal or the left brain-damaged groups. The specific SNR at which the spatial channel operating characteristics appeared using the electrophysiological method was the same to what was obtained using the behavioral method (-15 dB in Figure 15). This pattern of results was also obtained with the normal group.

For SNRs of -15 and -5 dB, the amplitude of the N_1 - P_2 component stayed fairly stable for the 'Right' and 'Left' locations, whereas for the 'Center' location, this amplitude sharply increased with the SNR. This is in agreement with the behavioral results for which a steep increase of the overall detection function was also observed between -15 and -5 dB (see Figure 15). At the SNR of -5 dB, the amplitude of the 'Center' location was clearly greater than each of the two side locations which could be explained by the use of midline recording sites (C_z and F_z).

Figure 31 - Mean amplitude of the N1-P2 component measured at the Cz site as a function of the SNR at 3 perceived spatial locations for the right lesion group (n=10)

Figure 32 - Mean amplitude of the N1-P2 component measured at the Fz site as a function of the SNR at 3 perceived spatial locations for the right lesion group (n = 10)

Figure 32 demonstrated a clear reduction in the amplitude of the N_1 - P_2 component for the 'Center' location at the SNR of -20 dB, which was 5 dB lower than the response obtained at the C_z site. At -20 dB, the amplitude of the N_1 - P_2 component for the 'Right' and 'Left' locations was clearly greater than what was obtained for the 'Center' location. These results also suggest the presence of a spatial channel but at a lower level as compared to the C_z site.

Contrary to the C_z site, at the SNR of -5 dB, the amplitude of the 'Center' location was not greater than each of the two side locations. A very large amplitude of the N_1 - P_2 component was obtained with the stimulus perceived in the 'Left' location. Globally, the amplitude of the N_1 - P_2 component measured at the F_z site was smaller than the amplitude obtained at the vertex recording site.

5.3. Results of the speech comprehension measures

Speech comprehension measures were made to document chronic speech comprehension problems. Three subtests of the Psycholinguistic Assessments of Language Processing in Aphasia (PALPA) (Kay et al 1992) were used: the Minimal Pair Discrimination Requiring Picture Selection (MPT in Figure 33), the Spoken Word-Picture Matching (SWT in Figure 33) and the Sentence-Picture Matching Auditory Version (SPMULTI in Figure 33). The individual data showing the performance for each subtest of the PALPA are not presented in the core of this text but are available in Appendix D.

Group means on the above mentioned tests are found in Figure 33, in which the percentages of correct responses (on the Y axis from 0 to 120 %) are presented for each test. The results of the MPT and the SWT correspond to a group mean computed directly from the raw data provided by the tests. In the case of SPMULTI, various dimensions of the sentence level speech comprehension items provided by the test (see section

Figure 33 - Group means for the 3 speech comprehension measures

4.2.4) were combined for each subject. This composite score was used afterward in the computation of the group mean. The scores obtained by the subjects for each subtest combined into SPMULTI are available in Appendix D.

As expected, when compared to the two other groups, the left brain-damaged group showed the lowest group mean speech comprehension score on all three tests, with the lowest mean score for this group recorded for the SPMULTI test. As predicted, the right brain-damaged group demonstrated a better group mean speech comprehension score on all three tests when compared to the performance of the left brain-damaged group. The normal group showed the best performance on all three tests.

These data were submitted to a MANOVA to identify significant differences among groups. A logarithmic transformation was applied to normalize variance on variables and to reach homogeneity of dispersion matrices. The transformed data were submitted to a Box's M test which turned out not to be significant [$F(6,2613) = 2.05, p = 0.06$]. The MANOVA was then applied and the multivariate test showed that the performance differences observed on all three variables were significant [$F(6,54) = 4.75, p = 0.001$].

At the univariate level, all variables showed at least one significant difference between groups [MPT: $F(2,29) = 7.06, p = 0.003$; SWT: $F(2,29) = 3.95, p = 0.03$; SPMULTI: $F(2,29) = 15.48, p < 0.001$]. The differences observed on the three speech comprehension tests among groups were analysed using a one-tailed post-hoc paired comparisons test. The differences observed between the normal and the right brain-damaged groups failed to reach statistical significance for two of the tests [SWT: $t(2,29) = -0.66, p = 0.51$; SPMULTI: $t(2,29) = -1.22, p = 0.23$]. For the MPT, the difference between these 2 groups reached statistical significance [MPT: $F(2,29) = 2.05, p = 0.05$]. The differences observed on the mean for the three speech comprehension tests between the left brain-damaged group and the normal group reached statistical significance for all the tests [MPT: $t(2,29) = -3.76, p = 0.008$; SWT: $t(2,29) = -2.68, p = 0.01$; SPMULTI: $t(2,29) = -5.27, p < 0.001$]. When left and right brain-damaged groups were compared,

only the SPMULTI test reached statistical significance [SPMULTI: $t(2,29) = -3.99$, $p < 0.001$].

The Appendix D presents the results of the MPT broken down into various error dimensions. The location of the error (initial/final consonant) and the linguistic trait of the error (voice, manner and place) are shown. The means computed on the number of incorrect responses obtained for each group suggested that the left brain-damaged subjects made more errors on the final consonant as compared to the initial consonant and had more trouble with the discrimination of the place of articulation, the voice and the manner (in decreasing order). The right brain-damaged subjects also made more errors on the final consonant as compared to the initial consonant and had more problem with the discrimination of the place of articulation. In contrast to the left brain-damaged group, the right brain-damaged subjects had less trouble with the voice. The differences observed across groups (brain-damaged groups only) on the error dimensions failed to reach significance on a MANOVA [$F(5,16) = 0.88$].

In the final analysis, it was established that on the basis of a single speech comprehension measure, it was not possible to discriminate between the normal group, the left and the right brain-damaged groups. This is mainly due to the difficulty in discriminating between the normal and the right brain-damaged groups. With this set of speech comprehension measures, the left brain-damaged group could be distinguished from the normal group. It is interesting to note that only the MPT test, which was the most auditory-related measure, distinguished the right brain-damaged group from the normal group. However, the pattern of errors on the MPT test did not reveal any significant trend. It is also important to highlight that the brain-damaged groups differed only on the complex linguistic task of sentence comprehension.

5.4. Results of the statistical analyses

The statistical analyses were applied to provide the answer to three specific questions: 1) is there a significant difference between the groups (normal, left and right brain-damaged) on sound localization related measures (BIBA, RT and EIBA(C_z), EIBA(F_z), 2) is there a significant parallel between results from behavioral and electrophysiological methods for each group and 3) is there a relationship between the sound localization measures and the speech comprehension measures. The results of the various statistical analyses used to answer to the above questions are provided in the following sections.

5.4.1. Difference between groups on sound localization measures

A MANOVA was performed to isolate significant differences between groups on sound localization related measures. The variables BIBAMAX, RT, EIBAMAX(C_z) and EIBAMAX(F_{pz}) were used as dependent variables and Group (Normal, Left and Right) was entered in the analysis as the factor. The Box's M test for multivariate homogeneity of dispersion matrices was not significant [$F(20, 1551) = 1.25, p = 0.20$]. The following Table 7 shows the group means for the 4 dependent variables entered in the MANOVA.

Table 7 - Group means for the 4 dependent variables used in the MANOVA

Dependent variables	Normal Group	Left Group	Right Group
BIBAMAX	98.4	31.0	37.1
RT	692.5	992.5	900.7
EIBA(C_z)	5.4	14.0	1.1
EIBA(F_z)	14.0	11.2	6.9

The MANOVA showed that the groups differed significantly on the dependent variables [$F(8,42) = 9.51, p < 0.001$]. The univariate F-tests showed that the groups

differed significantly on BIBAMAX [$F(2,24) = 10.84, p < 0.001$], RT [$F(2,24) = 4.49, p = 0.02$], EIBA(C_z) [$F(2,24) = 16.98, p < 0.001$] and EIBA(F_{pz}) [$F(2,24) = 4.61, p = 0.02$].

Post-hoc two-tailed paired comparisons on BIBAMAX showed that the normal group significantly differed from the brain-damaged groups [$t = -3.97, p < 0.001$ for the left lesion and $t = -3.97, p < 0.001$ for the right lesion]. The left and right brain-damaged groups were not statistically different on the same variable [$t = -0.36, p = 0.72$].

Post-hoc two-tailed paired comparisons on RT showed that the normal group significantly differed from the brain-damaged groups [$t = 2.82, p = 0.009$ for the left lesion and $t = 2.16, p = 0.04$ for the right lesion]. The left and right brain-damaged groups were not statistically different on the same variable [$t = 0.87, p = 0.40$].

Post-hoc two-tailed paired comparisons on EIBAMAX C_z showed that the normal group significantly differed from the brain-damaged groups [$t = 3.86, p < 0.001$ for the left lesion and $t = -2.14, p = 0.04$ for the right lesion]. The left and the right brain-damaged groups were statistically different on the same variable [$t = 5.80, p < 0.001$].

Post-hoc two-tailed paired comparisons on EIBAMAX F_z showed that the normal group significantly differed from the right brain-damaged group [$t = -3.01, p = 0.006$] but not from the left brain-damaged group [$t = -1.05, p = 0.31$]. The left and the right brain-damaged groups were not statistically different on the same variable [$t = 1.69, p = 0.10$].

These statistical analyses reveal that the behavioral measures (BIBAMAX and RT) presented a similar pattern of results across groups. Right and left brain-damaged group were different from normal, but not from each other.

The statistical analyses demonstrate a different pattern of results for the electrophysiological measures (EIBAMAX(C_z) and EIBAMAX(F_z)). For EIBAMAX(C_z), all groups are statistically different one from another whereas for EIBAMAX(F_z) only the right brain-damaged group significantly differed from the normal

group. In addition, the right and left brain-damaged groups were significantly different from each other. It is interesting to note that only the electrophysiological method, at least for the vertex recording site, led to statistically different results for each of the three groups. However, the results obtained by the electrophysiological method with the left brain-damaged group were opposed to those obtained with the behavioral method.

5.4.2. Relationship between behavioral and electrophysiological methods

A linear regression analysis was made between BIBA used as the dependent variable and EIBAMAX(C_z) and EIBAMAX(F_z) used as independent variables for the three groups together and separately. As suggested by the results presented in the preceding section, the overall regression analysis identified no significant linear relationship between the behavioral and electrophysiological methods [$r = 0.33$, $F(1,26) = 1.50$, $p = 0.24$]. When analysed separately, the normal group showed the highest degree of relationship between dependent and independent variables but the correlation, however, was not significant [$r = 0.68$, $F(2,7) = 2.97$, $p = 0.12$]. It must be noted however that this group analysis was not statistically powerful because of the small sample size. The two other groups showed correlation coefficients lower than 0.40.

The absence of any significant correlation tends to confirm the difference between behavioral and electrophysiological procedures at least in subjects with brain lesions. These results suggest that the behavioral and electrophysiological procedures might tackle different aspects of the same problem. However, the lack of a significant correlation could be explained also by a limited statistical power due to the small sample size of each group taken separately.

5.4.3. Relationship between sound localization-related and speech comprehension measures

A canonical correlation was performed to examine the relationship between two sets of data: sound localization-related measures BIBAMAX, RT, EIBA(C_z) and EIBA(F_z) and the speech comprehension measures MPT, SWT and SPMULTI. The assumptions of

univariate and multivariate homogeneity of variance were respected after applying a logarithmic transformation to the speech comprehension measures. The Box's M test was not significant [$F(21,830) = 1.05, p = 0.40$].

Two discriminant functions were identified. Both functions were significant: first canonical correlation [$R(2,14) = 0.84, p < 0.001$] and second canonical correlation [$R(2,6) = 0.80, p = 0.002$]. The first function explained 57.22 % of the variance and was correlated with EIBAMAX(C_z) ($r = 0.64$) and EIBAMAX(F_z) ($r = 0.35$) variables. This function could be identified as the electrophysiological function. The second function explained 42.78 % of the variance and was correlated with BIBAMAX ($r = 0.65$), SPMULTI ($r = 0.57$), MPT ($r = 0.49$), RT ($r = -0.45$) and SWT ($r = 0.30$) variables. This second function could be identified as the behavioral function. A high score on the first discriminant function was correlated with a high index of binaural advantage computed on the amplitude of the N_1 - P_2 component. A high score on the second discriminant function was correlated with a high index of binaural advantage computed on the probability of detection, a high score on the sentence comprehension test, a high score on the minimal pair test, a fast RT and a high score on the semantic contrast test.

Figure 34 illustrates the distance between groups on canonical discriminant functions along with the distribution of the cases. The electrophysiological function is located on the horizontal axis whereas the behavioral function is located on the vertical axis. As evaluated by group centroids, on the first function, the normal group mean was 0.90, the left brain-damaged group was 1.41 and the right brain-damaged group was -1.88. As already suggested by the preceding statistical analyses, a greater distance on the electrophysiological function was found between left and right brain-damaged groups while normal and left brain-damaged groups showed a smaller inter-group distance. On the second function, the group centroids were 1.45 for the normal group, -1.75 for the left brain-damaged group and -0.22 for the right brain-damaged group. On the behavioral function, the greater distance was found between the normal and the left brain-damaged group whereas the right brain-damaged group was at equal distance from both groups.

Figure 34 - Group centroids on canonical discrimination functions and distribution of the cases

Chapter Six

6. Discussion

The behavioral and electrophysiological results obtained for subjects in the normal group demonstrated operating characteristics of spatial channels. The probability of signal detection was markedly decreased at a low SNR (-15 dB) selectively at the ITD shared by the binaural signal and noise. At higher SNRs, the probability of signal detection remained stable across ITDs. These results agreed with the suggestion made by Yost (1997) that the binaural advantage supported by the spatial channel mechanism expresses itself only for low signal-to-noise ratios. As the signal becomes easier to detect with a higher SNR, the advantage of spatially separating the signal from the noise, on the basis of ITDs, disappears.

In terms of the reaction time needed for making the detection decision (target signal present or absent), the normal group exhibited, at a low SNR (-15 dB), a faster RT at the shared ITD. This faster RT was found at the shared ITD along with the lowest probability of signal detection. For the segregated ITDs, at the same low SNR, the RTs were longer and associated with a probability of detection close to 0.60. This reduced probability of detection could express a difficulty to disengage from the attentional focus provided by the midline noise. It was suggested by Good and Gilkey (1996) that the presence of a localized masking noise could induce a bias toward the masker. In our study, the binaural noise could attract the listener's attention at the midline location. Such a bias could in turn increase the difficulty of the detection task at segregated ITDs which could lead to prolonged RTs at the cost of a less accurate detection response. The RT results obtained with the normal group at the shared and segregated ITDs support the concept of the spatial channel. Similar RT results were reported for auditory selective attention tasks studied by Teder and Näätänen (1994) and by Mondor and Zatorre (1995). Their results established that attentional focus could be described by a gradient that declines with distance from an attended location. Such a gradient is compatible with the operating characteristics of a spatial channel.

In terms of electrophysiological results, the amplitude of the N_1 - P_2 component measured at the C_z site was reduced at a low SNR (-15 dB) for the shared ITD as compared to segregated ITDs. As expected, at a high SNR (-5 dB), the shared ITD gave rise to a larger N_1 - P_2 component as compared to segregated ITDs (at the midline recording electrode). These results were similar to those reported by Al'tman & Vaitulevich (1988) and by Vaitulevich & Pak (1991) for hemisphere-specific recording sites. Using hemisphere-specific electrode sites, a higher amplitude of the N_1 - P_2 component was recorded with the sound-image shifted either to the right or left side. The results of these studies also showed that at a high SNR, the difference in the amplitude of the N_1 - P_2 component between midline and off-midline perceived spatial locations tended to level off on specific electrode sites (midline spatial position on vertex recording site vs off-midline spatial position on parietal recording site). The equalization of the amplitude of the N_1 - P_2 component at different perceived spatial positions and across the recording sites at a high SNR is also in good agreement with the suggestion made by Yost (1997) that the spatial channel would not be activated when the signal is easily detectable.

Consistent results were provided by the subjects of the normal group across investigation methods. Characteristics of a spatial channel were demonstrated in both behavioral and electrophysiological methods. This similarity of results, both in terms of pattern and specific SNR involved, certainly supports the concept of the spatial channel. The linear regression analysis made on this group failed to identify a significant relationship between behavioral and electrophysiological results. However, this result could be due to a small sample size. Nevertheless, the results of the discriminant analysis identified a significant relationship between the specific pattern of performance of the normals on the binaural masking tasks and on the speech comprehension measures.

6.1. Identification of deficits in central auditory processing in left brain-damaged aphasic patients

For the left brain-damaged patients, the results presented in Chapter Five provided a consistent picture of deficit across the dimensions studied through the behavioral method. However, the observed effects in the data differ across investigation methods. While the behavioral method failed to identify any correlate for a spatial channel, the data obtained with the electrophysiological method suggested a strong spatial channel effect.

As will be discussed in the following sections, for the behavioral aspects of the binaural masking task, the deficit presented by the left brain-damaged subjects might be summarized as 1) a reduced steepness of the detection function, 2) an elevated threshold for a 50 % detection probability, 3) a plateau of small positive index of binaural advantage between SNRs of 0 to -20 dB and 4) a slow overall RT.

The reduced steepness of the detection function seemed to be related to a greater variability among left brain-damaged subjects in the probability of detecting the signal. In this group from one subject to the other, the highest and lowest points of the detection function varied significantly in terms of the specific SNR at which they were obtained. When averaged together, the spread of individual results led to a less steep group detection function. There is some indirect evidence in the literature that supports this finding of a larger variation of the detection performance in left brain-damaged patients. Looking at intra-subject variability, Divenyi & Robinson (1989) examined the consistency on detection tasks of normal, right-cva and left-cva subjects. The normals showed the highest consistency, whereas the left-cva subjects were the least consistent from trial-to-trial. An ANOVA revealed a significant side-of-lesion effect, but the reported differences failed to reach statistical significance on post-hoc comparisons between any pair of the three groups. The results reported by Divenyi & Robinson (1989) suggested a higher variability of detection performance for left brain-damaged subjects. It is not clear, however, if this wider variation of the results on detection tasks is

exclusively related to the side of the lesion, or to the interaction between the side of the lesion and the nature of the auditory task.

In terms of the 0.50 criteria, the overall detection function revealed an elevated threshold for the left brain-damaged group (-5 dB) when compared to the right brain-damaged (-9 dB) and the normal (-13 dB) groups. These results were similar to those reported by Thompson & Abel (1992) who used a 0.75 criteria to establish the detection threshold of a target signal, alternatively of short and long duration, in a silent background. Their results showed that subjects with left temporal pathology had significantly higher detection thresholds than the normal subjects but were not different from subjects with right temporal pathology. In terms of absolute results, for a short duration 500 Hz signal (50 ms), the normal group had the lowest threshold at 28 dBSPL, followed by the right temporal pathology group at 33 dBSPL. The left temporal pathology group had the highest threshold at 43 dBSPL. It is interesting to note that our results, and these of Thompson & Abel (1992) were obtained with subjects who presented normal hearing thresholds according to age within each group. Therefore, it can be assumed that auditory sensitivity, as evaluated with long duration pure tones, can be excluded as a contributing factor to the observed threshold differences. However in either study, it cannot be ruled out that other peripheral auditory abilities, which were not measured, could have influenced signal detection. One of these abilities, frequency selectivity, could have certainly influenced the detection of a signal in a background noise.

The stability of the probabilities of signal detection across SITDs in the left brain-damaged group merits some attention. Regardless of the size of the difference between the ITD of the signal and the noise, the probability of detection remained the same. This observation could be interpreted in two ways.

First interpretation: the presence of the noise has no effect on the detection performance. This interpretation receives little support when the shape of the detection function and the elevated detection threshold are considered. As explained earlier, these

demonstrated alterations of the detection function are difficult to link to a single cause but is it certainly not possible to completely exclude the influence of the masking noise on the detection response.

Second and most plausible interpretation: the presence of the noise at the midline location interfered with the detection of the signal over a large portion of the studied range of SITDs. Regardless of the ITD of the signal relative to which of the noise, the left brain-damaged listeners seemed unable to filter out the noise. This difficulty to filter out the noise might be explained by temporal processing deficits. The fact that small and similar indices of binaural advantage were obtained over a 20 dB range suggested that increasing or decreasing the SNR had little effect on the filtering of the noise. Therefore, the lack of a large binaural advantage is not related to the variation of the SNR but to a largely independent mechanism. In other words, binaural processing deficits are apparent and independent of the signal-to-noise ratio.

Precise discrimination of temporal characteristics is essential to resolve the specific position of a binaural target signal in masking noise. Low interaural time difference (ITD) discrimination thresholds are usually associated with a high capability to separate a target signal from a noise located in the same spatial area. Elevated ITD discrimination thresholds could be associated with an inability to separate a target signal from a noise located in the same spatial area. For example, with an ITD discrimination threshold of 415 μ s, the presence of a signal presented with an ITD of 400 μ s could not be differentiated from a masking noise at ITD of 0 μ s. Conversely, with an ITD discrimination threshold of 45 μ s, the same signal and noise could be easily differentiated. Elevated ITD discrimination threshold is a frequent finding in studies which have examined this auditory ability in unilateral left brain-damaged subjects (Rosenhall, 1992; Yamada, Kaga, Uno & Shindo, 1997). The mean ITD discrimination threshold reported by Rosenhall (1992) for 13 patients with left temporal lobe lesions was 187 ± 33 μ s using phase audiometer method I and 147 ± 27 μ s using phase audiometer method II. The mean ITD discrimination threshold reported by Yamada et al. (1997) for 12 patients with left hemisphere lesions involving the auditory cortex was 418 ± 160 μ s.

In our study, ITD discrimination thresholds were not measured, therefore, we can only speculate about elevated ITD thresholds in our sample of left brain-damaged subjects.

The left brain-damaged group exhibited the slowest overall RTs. In other studies on brain-damaged patients, slow RTs were reported for various auditory tasks, and especially for discrimination tasks (Dee & Van Allen, 1973; Thompson & Abel, 1992). Thompson and Abel (1992) reported that subjects with either left or right cortical brain lesions responded as fast as the control subjects on detection tasks. In the current study, when further examining the RTs related to signal detection at shared and segregated ITDs, the pattern across groups was less clear and only trends could be discussed.

For the left brain-damaged group, as it was also for the normal group, at the SNR corresponding to the maximal index of binaural advantage, the RT obtained for the detection of the signal at the shared ITD was faster, compared to positive SITD (left ear). The RT associated with the negative SITD (right ear) was as fast as the one obtained for the shared ITD. This result suggests an asymmetry in the perception of the target signal that was not identified with any other measures. At low SNRs, faster RTs are probably related to a subthreshold target signal (noise-only response type). This was the case for the shared ITD at the SNR of -10 dB. But this was also the case when the target signal was perceived at the right ear-contralateral to the lesioned hemisphere. The size of this effect was, however, small.

Given the behavioral results for the left brain-damaged group, it was expected that the amplitude of the N_1 - P_2 component would be stable across SITDs. A stable amplitude across SITDs would lead to the conclusion of a null binaural advantage. On the contrary, the amplitude of the N_1 - P_2 component was greatly reduced at the shared spatial position for almost all SNRs. Moreover, at the highest SNR (-5 dB), the amplitude of the N_1 - P_2 component for the shared spatial position was still smaller than the one obtained for left and right ear locations. This pattern of results strongly demonstrated spatial channel characteristics.

The electrophysiological results and their behavioral counterparts seemed difficult to reconcile for the left brain-damaged subjects. According to Loveless, Vasama, Mäkelä & Hari (1994), dissociation between perceptual processing and evoked responses is the rule rather than the exception. Our evoked electrophysiological responses could easily reflect some other process than the measured behavioral perception. Moreover, in our study, the exact sources of the measured electrophysiological activity were not identified in terms of cortical locations.

6.2. Identification of deficits in central auditory processing in right brain-damaged non-aphasic patients

The results presented in Chapter Five provide a consistent picture of deficits for the right brain-damaged group with both behavioral and electrophysiological methods. The size of the observed deficits was small but often statistically significant.

For the behavioral aspects of the binaural masking task, the deficit presented by the right brain-damaged subjects might be summarized as 1) a positive index of binaural advantage that is significantly smaller than the one recorded for the normal group and 2) a slower overall RT. As opposed to the left brain-damaged group, the presence of the binaural noise did not create a differential effect on behavioral and electrophysiological measures. Both the behavioral and electrophysiological results obtained from subjects in the right brain-damaged group demonstrated operating characteristics of a spatial channel as defined in this study.

With the behavioral method, the probability of signal detection markedly decreased at low SNRs (-10 and -15 dB) selectively at the ITD shared by the binaural signal and noise. At higher SNRs, the probability of signal detection remained stable across all ITDs. For the normal group, these results reflect a binaural advantage as described by Yost (1997).

The right brain-damaged group exhibited slower overall RTs compared to the normal group but faster RTs when compared to the left brain-damaged group. As already

reported in the preceeding section, slow RTs were reported on various auditory tasks for brain-damaged patients but especially on discrimination tasks (Dee & Van Allen, 1973; Thompson & Abel, 1992). In the current study, the specific examination of the RTs related to the detection of the signal at shared and segregated ITDs showed a pattern similar to the left brain-damaged group but with an inverse configuration. At the SNR corresponding to the maximal index of binaural advantage, the RT obtained for the detection of the signal at the shared ITD was faster compared to the negative ITDs (right ear). The RT associated with the positive ITDs (left ear) was as fast as the one obtained for the shared ITD. As with the left brain-damaged group, this result suggests an asymmetry in the perception of the target signal that was not identified with any other measures. At low SNRs, faster RTs are probably related to subthreshold target signal (noise-only response type). This was the case for the shared ITD at SNR of -15 dB. But this was also the case when the target signal was perceived at the left ear-contralateral to the lesioned hemisphere. As was the case with the left brain-damaged group, the magnitude of this effect was small. However, the fact that a contralateral configuration appeared in both groups lends additional support to a possible perceptual asymmetry.

In terms of the reaction time needed for making the detection decision (target signal present or absent), the right brain-damaged group exhibited, at low SNR (-15 dB), a faster RT at the shared ITD. This faster RT was found under conditions that produced the lowest probability of signal detection with signal and noise at the same central locus. For the segregated ITDs, at the same low SNR, the RTs were longer and associated with a probability of detection close to 0.60. As with the normal group, this reduced probability of detection could express a difficulty to disengage from the attentional focus provided by the midline noise as suggested by Good and Gilkey (1996). The RT results obtained with the right brain-damaged group at the shared and segregated ITDs support the concept of a spatial channel.

Given the behavioral results for the right brain-damaged group, it was expected that the amplitude of the N_1 - P_2 component would decreased at the shared ITD. In this group, the behavioral and electrophysiological results were fairly consistent. The

amplitude of the N_1 - P_2 component measured at the C_z site was reduced at a low SNR (-15 dB) for the shared ITD as compared to segregated ITDs. As expected, at a high SNR (-5 dB), the shared ITD gave rise at the midline recording electrode to a larger N_1 - P_2 component as compared to segregated ITDs. These results are similar to those of the normal group and those reported by Al'tman and Vaitulevich (1988) and by Vaitulevich and Pak (1991) for hemisphere-specific recording sites.

Consistent results were provided by the subjects of the right brain-damaged group across investigation methods. Characteristics of a spatial channel were demonstrated in both behavioral and electrophysiological methods. This consistency of results, both in terms of pattern and specific SNR involved, certainly supports the concept of a spatial channel. The pattern of results for this group is similar to that obtained with normal subjects. The main finding with right hemisphere brain-damaged subjects was a small but statistically significant difference on all studied dimensions compared to the normal group. This observation coincides to what is usually obtained with right hemisphere brain-damaged subjects on speech comprehension tests. The right brain-damaged subjects exhibit results that are often statistically different from normal, but the size of the difference is functionally questionable (Black & Strub, 1978; Coughlan & Warrington, 1978).

6.3. Speech comprehension deficits and relation to change in performance on binaural masking procedures

The results of the canonical correlation analysis indicated that a high score on the first significant discriminant function ("electrophysiological") was correlated with a high index of binaural advantage computed on the amplitude of the N_1 - P_2 component from both C_z and F_z sites. A greater distance on the electrophysiological function was found between left and right brain-damaged groups while normal and left brain-damaged groups showed a smaller inter-group distance. A high score on the second significant discriminant function ("behavioral") was correlated with a high index of binaural advantage computed on the probability of detection, a high score on the sentence comprehension test, a high score on the minimal pair test, a fast RT and a high score on the semantic contrast test. On the behavioral function a greater distance was found between the normal and the left brain-damaged group, whereas the right brain-damaged group showed an equal distance from both groups.

These results, and those discussed in preceding sections tend to confirm our hypothesis that patients with unilateral cva would show deficits in spatial channel mechanisms as measured both behaviorally and electrophysiologically. The results also suggest that the sound localization paradigm supported by the binaural masking procedures could be useful in predicting the severity of a speech comprehension problem and to identify underlying auditory dysfunction. As highlighted by Divenyi and Robinson (1989) and McNeil and Kimelman (1986), speech comprehension deficits in aphasic patients could be secondary to primary deficits in the auditory processing of rapid variations of intensity, frequency and duration dimensions of speech sounds. The binaural masking tests examined one specific duration dimension of the auditory processing that could be different than the ones underlying speech comprehension. As for an example, the pattern of errors on the speech comprehension tests, especially on the minimal pair discrimination test, did not reveal a strong trend of errors that could be

associated with time-related phonological aspects such as discrimination of consonantal place and voicing.

Our results demonstrated that the binaural masking procedures developed in this project could be used successfully to evaluate the central auditory system of brain-damaged patients. These procedures could complement the few non-linguistic tests that are available for clinical evaluation of aphasic patients (identification of frequency or duration patterns, and identification of environmental sounds in background noise (Katz & Pack, 1975; Pinheiro, 1977; Musiek, Baran & Pinheiro, 1990). Before any wide clinical application, however, additional experimentation would be needed to improve the quality of the procedures and address some of their limitations.

6.4. Limitations of the study

The use of only one ITD for the noise at the midline probably limited the power of the method especially with unilateral brain-damaged subjects. We believe that the use of three different perceived spatial positions for the noise (-600, 0 and 600 μ s) would allow us to obtain a more hemisphere-specific information. The data collected with our current method could not be used to examine the controversial question of function asymmetry because of its focus on the midline as a source of noise masking. For example, our findings did not show any strong contralateral deficit to the lesioned hemisphere which is often found in sound localization studies.

The discrepancy observed between the behavioral and electrophysiological results with the left brain-damaged group poses a problem for clinical interpretation. The use of a mismatch negativity (MMN) method instead of the conventional cortical evoked auditory responses might give a better reflection of the perceptual process. The MMN response is evoked by the activity of a higher-order process associated with detection of change in stimulus parameters. The MMN does not generate results in subjects who are unable to perceive the targeted change. This method has been used successfully in the normal population to study changes in sound source location (Teder & Näätänen, 1994; Schröger & Wolff, 1996, 1997).

A thorough examination of the various dimensions of what is called temporal processing would be useful to reach a better characterization of the brain-damaged subjects that would be asked to participate in further studies. This characterization should include measures of temporal resolution, gap detection and temporal ordering. Azimuthal location identification should also be made in the free field using single, multiple, fixed and mobile (real or virtual) sound sources. In the same manner, the speech comprehension problem should be better characterized, in terms of temporal aspects, using, for example, measures of categorical perception of stop consonants.

These suggested changes in the methodology should increase the sensitivity of the binaural masking procedures in the detection of central auditory dysfunction in brain-damaged subjects.

Chapter Seven

7. Conclusion

We have shown that patients with unilateral brain-damage resulting from cva (mca) present deficits on a binaural masking paradigm. The most sensitive measure of neurological impairment was the probability of signal detection as measured behaviorally. The left brain-damaged subjects exhibited the lowest performance on the binaural masking task. The detection of a signal in a background noise is strongly related to the temporal processing of interaural timing differences. Our results show that the severity of the binaural masking deficit is correlated with the severity of the speech comprehension problem. This study, however, did not clarify whether the two deficits reflect disruption of common mechanism or two separate mechanisms disrupted by the same lesion. One way of advancing knowledge on this question is to seriously consider the possibility that aphasia could coexist with other auditory processing deficits. A more complete assessment of the aphasic patient could lead to changes in the focus of the therapy used in speech-language pathology. Aphasic patients suffering from an auditory processing problem, as revealed by a binaural masking procedure, might benefit less from a treatment that emphasizes only a linguistic approach. The findings of this research project underscore the importance of an integrated battery of clinical tests and therapeutic approaches that would allow the speech-language pathologist and the audiologist to delineate the unique contribution of auditory and linguistic components of an observed speech comprehension problem.

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Appendix A

Individual data / Probability of signal detection as a function of the SNR

Appendix B

Individual data / Reaction Time as a function of the SNR

Appendix C

Individual data / Electrophysiological waveforms as a function of the SITD

Appendix D

Individual data / Scores on the speech comprehension tests

Results of the Spoken Word-Picture Matching Test (PALPA, 1992)

Ss ID	Total	Close Semantic			Distant Semantic			Incorrect responses (%)			Visual	Unrelated
		Correct (%)										
L01	100.0		0.0			0.0		0.0		0.0		0.0
L02	97.5		0.0			0.0		0.0		0.0		2.5
L05	100.0		0.0			0.0		0.0		0.0		0.0
L06	100.0		0.0			0.0		0.0		0.0		0.0
L07	100.0		0.0			0.0		0.0		0.0		0.0
L08	100.0		0.0			0.0		0.0		0.0		0.0
L09	65.0		27.5			7.5		7.5		0.0		0.0
L10	92.5		2.5			2.5		2.5		0.0		0.0
L11	100.0		0.0			0.0		0.0		0.0		0.0
L13	100.0		0.0			0.0		0.0		0.0		0.0
L14	72.5		12.5			5.0		7.5		7.5		2.5
L15	70.0		22.5			7.5		7.5		0.0		0.0
R02	100.0		0.0			0.0		0.0		0.0		0.0
R03	100.0		0.0			0.0		0.0		0.0		0.0
R04	100.0		0.0			0.0		0.0		0.0		0.0
R05	100.0		0.0			0.0		0.0		0.0		0.0
R07	100.0		0.0			0.0		0.0		0.0		0.0
R08	97.5		0.0			0.0		0.0		2.5		0.0
R10	100.0		0.0			0.0		0.0		0.0		0.0
R11	97.5		0.0			2.5		2.5		0.0		0.0
R12	97.5		0.0			0.0		0.0		2.5		0.0
R13	100.0		0.0			0.0		0.0		0.0		0.0
N01	100.0		0.0			0.0		0.0		0.0		0.0
N02	100.0		0.0			0.0		0.0		0.0		0.0
N04	100.0		0.0			0.0		0.0		0.0		0.0
N05	100.0		0.0			0.0		0.0		0.0		0.0
N06	100.0		0.0			0.0		0.0		0.0		0.0
N07	100.0		0.0			0.0		0.0		0.0		0.0
N08	100.0		0.0			0.0		0.0		0.0		0.0
N11	100.0		0.0			0.0		0.0		0.0		0.0
N13	100.0		0.0			0.0		0.0		0.0		0.0
N14	100.0		0.0			0.0		0.0		0.0		0.0

Results of the Sentence-Picture Matching Test (PALPA, 1992)

Ss ID	Multi	Active Form	Passive Form	Reversible	Correct responses (%)		Gapped	Converse
					Reversible	Non-reversible		
	Correct (%)							
L01	94.3	100.0	83.3	95.0	93.8		93.8	100.0
L02	89.3	100.0	83.3	90.0	100.0		87.5	75.0
L05	85.9	91.7	83.3	90.0		93.8	93.8	62.5
L06	72.9	75.0	58.3	60.0	100.0		93.8	50.0
L07	100.0	100.0	100.0	100.0	100.0		100.0	100.0
L08	68.5	83.3	50.0	65.0	87.5		62.5	62.5
L09	42.2	58.3	50.0	45.0	43.8		18.8	37.5
L10	87.2	100.0	83.3	90.0	100.0		75.0	75.0
L11	73.8	83.3	58.3	70.0	93.8		62.5	75.0
L13	74.7	66.7	66.7	65.0	87.5		87.5	75.0
L14	52.7	58.3	41.7	60.0	37.5		56.3	62.5
L15	52.8	50.0	33.3	40.0	68.8		50.0	75.0
R02	95.8	100.0	100.0	100.0	100.0		100.0	75.0
R03	96.9	100.0	100.0	100.0	100.0		93.8	87.5
R04	95.4	91.7	91.7	95.0	93.8		100.0	100.0
R05	100.0	100.0	100.0	100.0	100.0		100.0	100.0
R07	97.9	100.0	100.0	100.0	100.0		100.0	87.5
R08	85.4	91.7	91.7	85.0	87.5		93.8	62.5
R10	93.8	100.0	100.0	100.0	100.0		100.0	62.5
R11	99.0	100.0	100.0	100.0	100.0		93.8	100.0
R12	86.1	91.7	83.3	85.0	93.8		75.0	87.5
R13	100.0	100.0	100.0	100.0	100.0		100.0	100.0
N01	97.8	100.0	91.7	95.0	100.0		100.0	100.0
N02	97.9	100.0	100.0	100.0	100.0		100.0	87.5
N04	94.1	83.3	100.0	100.0	87.5		93.8	100.0
N05	100.0	100.0	100.0	100.0	100.0		100.0	100.0
N06	97.9	100.0	100.0	100.0	100.0		100.0	87.5
N07	100.0	100.0	100.0	100.0	100.0		100.0	100.0
N08	100.0	100.0	100.0	100.0	100.0		100.0	100.0
N11	100.0	100.0	100.0	100.0	100.0		100.0	100.0
N13	93.6	91.7	100.0	95.0	100.0		100.0	75.0
N14	100.0	100.0	100.0	100.0	100.0		100.0	100.0

Results of the Minimal Pair Discrimination Requiring Picture Selection Test (PALPA, 1992)

Sa ID	Total	Initial Consonant	Final Consonant	Metathetic	Incorrect responses (%)						Place	Low Occurrence	High Occurrence
					Correct (%)	Voice	Manner	Place	Low Occurrence	High Occurrence			
L01	97,5	0,0	5,6	0,0	8,3	0,0	0,0	0,0	0,0	5,0			
L02	92,5	5,6	11,1	0,0	0,0	0,0	7,1	14,3	5,0	10,0			
L05	95,0	0,0	11,1	0,0	0,0	0,0	0,0	14,3	5,0	5,0			
L06	100,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0			
L07	100,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0			
L08	97,5	0,0	5,6	0,0	8,3	0,0	0,0	0,0	5,0	0,0			
L09	80,0	16,6	27,8	0,0	33,3	14,3	14,3	14,3	20,0	20,0			
L10	85,0	0,0	27,8	25,0	0,0	21,4	21,4	21,4	15,0	15,0			
L11	77,5	5,6	38,9	25,0	25,0	14,3	14,3	28,6	30,0	15,0			
L13	100,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0			
L14	70,0	38,9	22,2	25,0	33,3	28,6	28,6	28,6	35,0	25,0			
L15	77,5	16,7	16,7	0,0	8,3	7,1	7,1	28,6	15,0	15,0			
Mean	89,4	7,0	13,9	6,3	9,7	7,7	7,7	12,5	10,8	9,2			
R02	100,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0			
R03	100,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0			
R04	95,0	5,6	5,6	0,0	0,0	0,0	0,0	14,3	5,0	5,0			
R05	95,0	5,6	5,6	0,0	8,3	8,3	8,3	0,0	0,0	10,0			
R07	97,5	0,0	0,0	25,0	0,0	0,0	7,1	0,0	0,0	5,0			
R08	87,5	0,0	27,8	0,0	0,0	7,1	7,1	28,6	10,0	10,0			
R10	95,0	5,6	5,6	0,0	0,0	7,1	7,1	7,1	5,0	5,0			
R11	100,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0			
R12	85,0	5,6	27,8	0,0	8,3	21,4	21,4	14,3	15,0	15,0			
R13	100,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0			
Mean	95,5	2,2	7,2	2,5	1,7	5,1	5,1	6,4	3,5	5,0			
N01	100,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0			
N02	100,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0			
N04	100,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0			
N05	100,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0			
N06	100,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0			
N07	97,5	0,0	5,6	0,0	8,3	0,0	0,0	0,0	0,0	5,0			
N08	100,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0			
N11	100,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0			
N13	100,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0			
N14	100,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0	0,0			
Mean	99,8	0,0	0,6	0,0	0,8	0,0	0,0	0,0	0,0	0,5			