Introduction

Fluent aphasia is an interesting condition for various reasons. For a number of years, the existence of two types of aphasia that not only sound different but arise from different loci of cerebral damage has been used to support the notion of two independent domains of language, the grammar and the lexicon. Damage to cerebral tissue in the pre-Rolandic areas of the cortex is associated with damage to the grammar or to the computational aspects of language, while the mental lexicon, our vocabulary store, is spared. In fact it is not clear whether aphasia causes loss or damaged access to this domain of language, as we will discuss below. In contrast to this state, damage to the post-Rolandic area results in damage to the mental lexicon or access to that lexicon: the grammar, or access to the computational aspects of language, is assumed to be spared. So, these two pathological conditions that we will refer to as non-fluent and fluent aphasia, epitomise damaged grammar versus damaged lexis. To take idealised cases, non-fluent speakers exhibit grammatical errors whereas fluent aphasic speakers struggle with lexical recall. Of course, this can be seen as a gross over-simplification and we will examine the flaws in this description as we progress through the monograph, but it not only provides a good starting point but also encapsulates issues that will be explored.

This monograph is about fluent aphasia, a type of aphasia that is commonly seen in clinics but about which little is written. We will consider the language abilities of people with fluent aphasia, concentrating on their lexical and grammatical abilities. All aphasia textbooks contain descriptions of fluent aphasia (under various classifications) largely based on clinical observations rather than on empirical work. In the course of this monograph we will review various accounts of fluent aphasia. I aim, by drawing on connected speech data, to expand on the current descriptions and accounts that we have of fluent aphasic speech, concentrating on the language produced. I will discuss grammatical and lexical abilities revealed in these data and see how far lexical retrieval problems disrupt sentence structure. I will re-examine the claims that syntax remains intact. To this end, it will be necessary first to pull together available

descriptions, speculations and explanations of fluent aphasia. This information must then be viewed within the two contexts, of abnormal and normal language. Comparisons with abnormal language, aphasia, will focus on descriptions and explanations of non-fluent aphasia and, specifically, agrammatism. Comparisons of the features of these two types of aphasia include consideration of the explanations of agrammatism, especially explanations couched within the framework of Universal Grammar.

There are various perspectives from which fluent aphasia may be viewed. Aphasia is a condition that arises subsequent to brain damage and, as a result, changes occur to non-verbal as well as to verbal behaviour. Other consequences of brain damage may include impaired visual skills, spatial skills, memory and so on. Deficits of this nature impact on patients' ability to cope with aphasia, their ability to co-operate with testing and their chances of successful rehabilitation and, as a result, some aphasiologists and clinicians consider these behavioural deficits to be part and parcel of aphasia. I will not be taking this approach. This account focuses on the spoken output in fluent aphasia, although one chapter is devoted to the comprehension deficit, a characteristic of fluent aphasia.

Material in this monograph has been gleaned from many sources. I cite and discuss empirical research but I aim to cover or, at least, introduce the reader to various perspectives of study, including the use of continuous speech data. I have tried to give a broad view although I am aware that my selection will not be to everyone's liking. I have also tried to go beyond data and to give a flavour of how fluent aphasia touches and changes peoples' lives. The anecdotes I tell are based on my clinical experiences as a speech and language therapist and I hope they give some humanity to this study. I have learnt much from discussions I have had with other aphasiologists, not only speech and language therapists, but also neurologists, psychologists and, occasionally, some exceptional linguists. The study of aphasia seems to attract more psychologists than linguists, and linguistic analyses and linguistically motivated theories of aphasia are few and far between. I remain convinced, however, that there are rich data here for linguists to pick over.

This monograph does not adhere to any one school of aphasiology or any one theory within linguistic science: I write as a research speech and language therapist and for readers from a number of different disciplines. From time to time, I call upon theoretical linguistic notions but, in common with my fellow aphasiologists, do not follow or develop any one particular theory. I am writing for students of aphasia, whether based in psychology, linguistics, health studies, medicine or speech and language pathology. By students, I include

anyone who wishes to learn about the topic, regardless of their formal level of study. I hope that readers who come from a linguistics or psychology background will gain some insights into fluent aphasia that include the person as well as the data. For those readers who come from a clinical background, I hope that my linguistic descriptions will encourage them to think about aphasic data within some linguistic framework. There will be parts of the book that may be rather technical for some readers and some that will be too basic for others. It should be possible to turn the pages or go to the next chapter and pick up the story again or select the chapters of interest. I hope that this monograph will not only give a picture of what fluent aphasia sounds like but also help the reader to understand a little about what a person with fluent aphasia experiences.

Chapter 1 starts by giving a short, general introduction to three topics: the description of fluent aphasic speech, the notion of aphasia syndromes, and the relationship between brain and language. I start by outlining the commonly recognised syndromes of aphasia. I then proceed to consider the original accounts of the disorder given by Wernicke in the nineteenth century and then move on to probably the most influential account of fluent aphasia given in the twentieth century, that by Harold Goodglass. Goodglass and his associates have been staunch advocates of the syndromic approach in aphasia research. That is, they hold that aphasia is not a unitary condition but comprises different types of aphasia and that these types constitute syndromes. While recognising the importance of syndromes, especially in aphasia research, we encounter and discuss some problems of this approach. This leads us to a short review of language-brain relationships.

In chapter 2, I extend the descriptions of fluent aphasia, especially descriptions of Wernicke's aphasic speech and the grammatical structures available to these speakers by taking into account a variety of test results and clinical observations. Test material is used extensively in research and in clinical practice and, as we will see in chapter 2, informs our views of fluent aphasic speech. However, the nature of the speech elicited and the assumptions that arise from assessment depend to a greater or lesser extent on the methods used in testing. Chapter 3 contains a discussion about the nature of testing, a variety of methodologies, and how a theoretical stance influences both methodology and interpretation of results. In chapter 4, I will present a characterisation of the speech of Wernicke's aphasia gathered from analyses of spontaneous speech samples. Some of these are from previously published studies and some are from new clinical studies at the University of Reading. I am indebted to the discussion I have had with colleagues and especially while working with Cindy Thompson

and her associates at the Aphasia Research laboratory, Northwestern University, USA, and acknowledge their contribution to my thoughts on analysis.

A brief description is given of agrammatism in chapter 1. In chapter 5, I examine descriptions that claim that agrammatism is a syntactic deficit and distinct from paragrammatism. This is a well-established view in the research literature but does not persist unchallenged. A contrary view is that these two disorders have much in common. In considering this afresh, we will need to have at least a brief overview of comprehension deficits in Wernicke's aphasic speakers. This is the content of chapter 6.

I am writing this monograph from two viewpoints, the clinical linguist's and the practising clinician's. Although the main aim of this book is to provide a new and more detailed description of fluent aphasia and, in constructing that description, to explore the nature of the lexical and grammatical deficits, I am always aware of issues of rehabilitation. Some readers may wonder how these deficits impact upon patients' lives. My experience is that those working in aphasiology but not engaged with aphasia therapy are interested in how aphasia affects the person as a whole. While these matters are not the focus of this monograph, our understanding of fluent aphasia can be enhanced by considering issues that go beyond data and theory. In chapter 7, I describe one speaker with fluent aphasia and his test results, and touch briefly on how these motivated therapy. These data allow us to consider language change in this type of aphasia and how one aphasic speaker coped with his reduced communicative capacities. In chapter 8, I endeavour to bring all these themes together presenting a description of the speech of those with Wernicke's aphasia.

1 *Fluent aphasia: identification and classic descriptions*

Uh we're in the in the kermp kerken kitchen in in the kitchen and there's a lady doing the slowing. She's got the pouring the plate watching it with with um. The water is balancing in the sink the (X) of the sink and the water is pouring all over the bowing bowing all over it.

Introduction

The text quoted above is a small section of a person's description of a picture of a woman washing dishes. The speaker has fluent aphasia. Fluent aphasia is an acquired language disorder that arises subsequent to brain damage. This chapter provides an introduction to the condition and a historical perspective.

Fluent aphasia is a distressing condition for the person who has it and for relatives and friends of that person. It is a relatively common type of aphasia, although there is little published about it compared with the literature on Broca's aphasia. Kertesz (1982:7) described Wernicke's aphasia as 'a common aphasic impairment' and Wallesch, Bak and Schulle-Mouting (1992) found that the majority of patients who survived for one year post-trauma had fluent aphasia. It is also one of the most common types of aphasia found in the first few weeks post trauma (Blanken, Dittmann, Grimm, Marshall and Wallesch 1993). Approximately a quarter of all referrals to our local hospital clinics are diagnosed as fluent aphasia by the speech and language therapist, but at our clinic at the University of Reading, approximately 80 per cent of all adult clients referred with aphasia have fluent aphasia.

Typically, this distressing condition presents problems for the hospital-based rehabilitation team because, for the majority of those referred, their problems are not physical but linguistic. Referrals to our university clinic often arise because it is not at all clear how rehabilitation or support is best provided. Fortunately, people with fluent aphasia are usually mobile in that they can get in and out of cars or on and off buses and therefore can make the journey to a university clinic without specialised transport. For some, though, the severity

of the language deficit makes independent travel impossible and they need to travel with a companion. Others, who are less impaired, may also always have a travelling companion, as spouses or carers may be reluctant to see them travel alone because of the language difficulty. Nevertheless, some complete journeys successfully and this is important in terms of rehabilitation and adds to a sense of independence. In some ways, then, people with fluent aphasia make an ideal group to refer on to a clinic situated on a large university campus. My knowledge of normal and aphasic language has been enhanced by working with this group of patients at our University clinic as well as within acute and rehabilitation services.

The identification and description of fluent aphasia occurred relatively late. Although descriptions of aphasia have been around for a very long time, at least since 400 BC, records of the disorder have concentrated on the type of aphasia in which production of words and sentences is seriously disturbed but comprehension remains intact, what is now known as Broca's aphasia. There seem to be no descriptions that match what is now recognised as fluent aphasia (Benton and Joynt 1960). It was not until the nineteenth century and the work of Carl Wernicke, a German neuro-psychiatrist, that a language disorder in which speech production was fluent, although often meaningless, was described. Fluent aphasia is the term I have chosen to use for this type of aphasia, a type that is recognised clinically, although difficult to delineate with a set of agreed criteria. The term 'fluent' refers to the characteristic noted by Wernicke and taken by Goodglass (one of the outstanding aphasiologists of the twentieth century) to be *the* defining characteristic of a group of aphasias. The speech of fluent aphasia is at a normal rate without the effort and hesitation associated with non-fluent aphasia. It is, however, often meaningless.

Taxonomy

Fluent aphasia needs to be thought about within the context of other aphasias, which I will now briefly summarise. (Those readers who are familiar with the field might wish to omit the next couple of paragraphs. For more detailed accounts of different aphasia types, the reader is referred to Goodglass 1993.) There are various ways of grouping aphasia types. This brief résumé is based on what is known as the 'neoclassical' school. This taxonomy has been used by a group of researchers publishing in the latter half of the twentieth century and it is 'neoclassical' in that it is based and refers back to terms used by nineteenth-century aphasiologists. The term 'fluent' denoted an important subdivision of

the aphasias and is used in the neoclassical terminology. The fluent/non-fluent division is especially associated with the 'Boston School', a group of clinicians and researchers, led by Harold Goodglass, at the Veterans' Hospital in Boston, USA. In the middle of the twentieth century, Goodglass and colleagues (Goodglass, Quadfasel and Timberlake 1964, Howes and Gerschwind 1964) observed that a major division between two types of aphasia could be made by a simple metric, namely, 'phrase length'. This became the basic feature by which two major groups of aphasias were distinguished. Each of these groups was then further subdivided.

Four types of aphasia are considered to be 'fluent' in the neoclassical classification scheme. These are *Wernicke's aphasia*, *conduction aphasia*, *transcortical sensory aphasia and anomia*, of which Wernicke's and conduction aphasia are the most common. Speech in transcortical sensory aphasia and conduction aphasia is fluent, as is the speech in Wernicke's aphasia. Characteristically, patients with conduction aphasia are differentiated from other types of fluent aphasia by their poor repetition abilities, while speakers with transcortical sensory aphasia are good at repeating speech. Comprehension is compromised in conduction aphasia and in transcortical sensory aphasia but less severely than in Wernicke's aphasia. (Classically, comprehension in conduction aphasia has been assumed to be intact although a number of studies from the latter half of the last century have demonstrated that this is not the case.) In conduction aphasia, the comprehension deficit is not as extreme as in Wernicke's aphasia and there are typically more errors in word form. Paraphasic errors, where phoneme substitutions occur, are commonly associated with this disorder. Conduction aphasia occurs rarely (de Bleser 1988:166). Although there is some agreement that lesions that result in Wernicke's aphasia involve the posterior perisylvian speech area, a variety of lesion sites have been reported for all aphasia types (Rapcsak and Rubens 1994). A type of fluent aphasia, often similar to transcortical sensory aphasia, can be observed in senile dementia, further undermining the notion of an isomorphic relationship between lesion site and language disorder. It would appear that there are a number of similarities, such as confusion with word selection and problems following commands, but the overall clinical picture differs. We discuss this further below.

All aphasic speakers (those with fluent and non-fluent aphasia) have difficulties with accessing lexical items. The diagnosis of anomia is given when this feature is predominant, and when comprehension is good or near normal and delivery of speech is fluent. These aphasic speakers have special difficulty in accessing nouns whereas they have less difficulty in accessing verbs. Patients

who make a good recovery from either a fluent or non-fluent aphasia may have a persisting residual anomia.

Two or three types of non-fluent aphasia are recognised. The most widely discussed and researched is Broca's aphasia. This is generally associated with lesions in the pre-Rolandic area of the left cerebral cortex. Speech production is reduced and grammar and access to vocabulary is compromised but comprehension is mainly intact. Speech typically consists of short utterances in which grammatical features such as determiners, auxiliary verbs and verb inflections are not always present in obligatory positions. Agrammatism is a term applied by some researchers to Broca's aphasia. For others, agrammatism constitutes a subgroup of Broca's patients who, as well as having production deficits, have problems understanding sentences with certain syntactic structures, specifically when sentence constituents have been moved from their canonical position. Strong claims have been made about the nature of these comprehension deficits (Grodzinsky 1990, 1995, 2000a and b): this condition is explored further in chapter 5.

A much less common non-fluent aphasia is transcortical motor aphasia. People with this type of aphasia have impoverished output, poor ability to produce either single words (in a naming task) or sentences. But, like their sensory counterparts, they are good at repeating both single words and sentences although Rapcsak and Rubens (1994:301) claim that this ability does not extend to repeating sentences with complex grammatical structures. Some researchers (e.g. de Bleser 1988 and Goodglass and Kaplan 1983) include global aphasia in the non-fluent category. People with global aphasia have severe impairment of both production and comprehension. Whether a lack of speech can be considered to be non-fluent is questionable but need not trouble us here. These then are the traditional categories or syndromes. Although they 'leak' in that characteristics are not exclusive to each syndrome, the concept of syndrome serves as a useful shorthand for constellations of features. I will be referring to these syndromes from now on and will use one further distinguishing feature, *fluency versus non-fluency*.

The use of *fluency/non-fluency* as a diagnostic criterion is not universally accepted. Poeck (1989) claimed that the dichotomy created merely reflects the idea that there are basically two distinct types of aphasia, *expressive* and *receptive*, *motor* and *sensory*, or, *Broca's* and *Wernicke's*. This idea, he says, has been around since Wernicke's early publications alerted the field to a type of aphasia which contrasted with Broca's. Despite criticisms, I find the concept of fluent versus non-fluent to have both clinical and pedagogic merit and it will serve as a good starting point. It is a metric that not only divides the aphasic

population into two categories which fit, by and large, clinical observations of surface features, but also relates to the neuro-anatomical claims about aphasia advanced by the two nineteenth-century giants of aphasia, Paul Broca and Carl Wernicke. However, this descriptor does not take us very far along the route of considering the linguistic and especially the grammatical features of aphasia. In order to gain some historical perspective, I will start at the beginning, with Wernicke's description of the condition.

Wernicke's aphasia

In 1874 a German neurologist, Carl Wernicke, published a monograph *Aphasia Symptom Complex* in which he described a language disorder found subsequent to brain damage. This language disorder was strikingly different from the aphasia that Paul Broca had described ten years earlier. The aphasic speakers described by Wernicke had copious fluent speech, unlike the slow, halting, non-fluent and sparse aphasic speech described by Broca. Despite the fluency of speech observed by Wernicke, there were obvious problems with word retrieval and, as a consequence, meaning was diminished to a greater or lesser degree. The second distinguishing feature of these speakers was their conspicuous problem with understanding language despite their having normal hearing. Although the presence of deficient understanding was new and perhaps the most remarkable of his observations at the time, Wernicke also noted that impairments of speech were different from those observed by Broca.

Wernicke's 1874 monograph differed from the usual format in that his clinical findings were used to support his attempts to construct a model to explain the processes involved in aphasia (Eggert 1977:21). Wernicke was interested in the representation of language, the storage of images, the creation of lexical memory and the relationship between mental processes and neural structures. Although Wernicke's primary interest in this condition centred on the relationship between mental imagery, memory and the representation and retrieval of words, and it is in this domain that his discussion is focused, there was recognition that the structure of language was disrupted. Wernicke referred to this type of aphasia as *sensory aphasia*. There have been numerous classification schemes proposed and various labels suggested since that time but the term that predominates is, not surprisingly, Wernicke's aphasia.

Wernicke identified two further types of aphasia that arose subsequent to cortical damage in the post-Sylvian area of the brain: transcortical sensory aphasia and conduction aphasia, both of which were similarly characterised by fluent speech. All three types of aphasia are recognised today and (with

some minor variations) the same terminology is used both clinically and in much of the research literature. Our understanding of these three types of fluent aphasia has progressed in fits and starts in the intervening years but, as we shall see below, the descriptions used today bear an uncanny resemblance to the descriptions offered by Wernicke.

Wernicke's descriptions arose from his observations of a series of patients who had language disturbances following cortical damage with various aetiologies. Site of lesion was verified post-mortem. He brought attention to language disturbances that resulted from damage to the post-Rolandic cortex. In his 1874 monograph, Wernicke observed that aphasia in some patients resulted in impaired auditory comprehension despite the preserved ability of auditory perception. What was striking was that this deficit was associated with lesions in the left temporal lobe, as was the condition described by Broca. But, importantly, these two types of aphasia were distinguished, from the beginning, by the presence or absence of comprehension deficit and the two contrasting types of abnormal speech.

Wernicke did not actually list the characteristics of the aphasia he was observing but, in a review of Wernicke's work, Eggert (1977:47) notes that the symptom triad that characterises Wernicke's aphasia includes auditory comprehension loss, paraphasia and word-finding impairments. *Paraphasia* is a term that is frequently used in connection with Wernicke's aphasia but seldom defined satisfactorily. Word-finding difficulties are widely recognised as features of all types of fluent aphasia, although here Eggert notes that they are the 'chief characteristic of the final stages of sensory aphasia' (p. 54). 'Final stage' might refer to the mildest type of fluent aphasia or a late stage of recovery: the meaning is not clear. The presence of word-finding difficulties does not separate this type of aphasia from Broca's aphasia or, for that matter, any other type of aphasia. All aphasic speakers have difficulties with word retrieval. The class of word affected may be a distinguishing feature although the distinction probably a lot less categorical than has been suggested in the past (Luzzatti, Raggi, Zonca, Pistarini, Contardi and Pinna 2002).

Wernicke noted two essential features of what he called sensory aphasia. First, there was a loss of word sound perception and hence of access to word meaning. He observed that this loss of understanding was not caused by deafness, for these patients couldn't access meaning through other modalities. The second important symptom of this group of patients was that 'articulate speech remained intact' and, unlike the aphasia described by Broca a few years before, these speakers were 'surprisingly verbose' (Wernicke 1906:226–7, cited in Eggert 1977). Wernicke described these patients as having 'a fairly extensive