Brain Damage, Behaviour and Cognition Series

MILESTONES IN THE HISTORY OF APHASIA

THEORIES AND PROTAGONISTS

JUERGEN TESAK AND CHRIS CODE

Milestones in the History of Aphasia

Milestones in the History of Aphasia surveys the history of aphasia from its earliest mentions in ancient times, to the turn of the new millennium in 2000.

The book takes a predominantly chronological approach starting with an examination of the earliest medical documents and medieval attempts to understand aphasia, to the momentous events of the nineteenth and twentieth centuries, up to the development of modern cognitive neuroscience in recent years. It traces the development of theory about and understanding of aphasia, and the role of significant individuals in this history. The result is a well illustrated introduction to the main events and personalities in the rich history of aphasia.

This accessible book provides a unique insight into the fascinating development of research in aphasia. It will be of great interest to undergraduates and postgraduates, researchers, teachers and clinicians in psychology, speech and language pathology and therapy, neurology and linguistics.

Juergen Tesak was Professor of Logopaedie at the Europa Fachhochschule Fresenius (EFF), Darmstadt, Germany.

Chris Code is Research Fellow and Visiting Professor in the School of Psychology, Washington Singer Laboratories, University of Exeter and Foundation Professor of Communication Sciences and Disorders (Hon) at the University of Sydney. He is also National Advisor on Aphasia for Speakability (www. speakability.org.uk), Patron of Aphasia Now and co-founding Editor of the international Psychology Press journal *Aphasiology*.

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Milestones in the History of Aphasia

Theories and protagonists

Juergen Tesak and Chris Code

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Series preface

From being an area primarily on the periphery of mainstream behavioural and cognitive science, neuropsychology has developed in recent years into an area of central concern for a range of disciplines. We are witnessing not only a revolution in the way in which brain – behaviour – cognition relationships are viewed, but also a widening of interest concerning developments in neuropsychology on the part of a range of workers in a variety of fields. Major advances in brain-imaging techniques and the cognitive modelling of the impairments following brain injury promise a wider understanding of the nature of the representation of cognition and behaviour in the damaged and undamaged brain.

Neuropsychology is now centrally important for those working with brain-damaged people, but the very rate of expansion in the area makes it difficult to keep up with findings from the current research. The aim of the *Brain Damage, Behaviour and Cognition* series is to publish a wide range of books that present comprehensive and up-to-date overviews of current developments in specific areas of interest.

These books will be of particular interest to those working with brain-damaged people. It is the editors' intention that undergraduates, postgraduates, clinicians, and researchers in psychology, speech pathology, and medicine will find this series a useful source of information on important current developments. The authors and editors of the books in this series are experts in their respective fields, working at the forefront of contemporary research. They have produced texts that are accessible and scholarly. We thank them for their contribution and their hard work in fulfilling the aims of the series.

CC and GH
Sydney, Australia, Exeter, England and Birmingham, UK
Series Editors

Preface

It is often said that someone who knows no history is always in danger of repeating the mistakes of the past. If only for this reason, the history of a field has relevance for the present. Sometimes history can provide or inspire answers for current questions, and often, important questions posed in history still remain unanswered. So an understanding of where a field's knowledge base came from and the context and circumstances of its development can provide an important foundation to any student of the field. Interest in aphasia comes from an amalgam of scientific fields and concerns. Interest originally emerged in ancient medicine and grew over the centuries with the development of neurology, psychology, linguistics, philosophy, and speech and language pathology and therapy. Aphasia has been at the core of the history of neurology and neuropsychology because of what it promised to tell us about how the brain works and the role it plays in language function.

When we set out to write an introduction to the history of aphasia, it soon became clear to us that we were examining the work of some of the greatest minds of their respective times. Many of these aphasiologists not only laid the foundations for aphasia, but also made significant contributions to the development of neurology, psychology, linguistics and psychiatry. The history of aphasia, like the history of any science, does not evolve in glorious isolation, but is significantly influenced not only by developments in other branches of science, but also by the social and political events and developments taking place around it. History is essentially a record of the ambitions, deliberations and actions of people engaged in events set against the background of their individual personal, cultural and political life. Therefore, in this introductory book we wanted to sketch some of the important cultural and political background to the development of aphasiology over time. We set out not to write a critical review evaluating in detail from a contemporary perspective the theoretical issues and developments from history – there are a range of such treatments to choose from – but we attempted a fairly neutral cataloguing of these theoretical developments and the events and personalities behind them. Where possible we wanted to support our presentation using the original writings or translations and commentaries on those writings by significant workers in the field. However, a position of neutrality is the

most difficult one to maintain, and we hope we are forgiven if the reader finds that our footing slips occasionally.

History evolves around us all the time and in a book like this we had to decide where, in effect, history ends. Reviewing the excellent range of past seminal works on the history of aphasia, it becomes clear that most end in the 1960s. However, a new century has begun: the second half of the twentieth century saw huge developments, such as the rise and continuing rise of neuroscience, and this more recent history has had significant relevance for aphasiology. We felt it necessary therefore to bring our introduction to the history of aphasia up to the year 2000, and this is where we drew our line.

Because we chose to bring our history of aphasia up to date, we have organised the book into two parts. Part I includes six chapters that cover the period from the earliest medical documents through to the 1960s and Part II sketches the more recent history of the second half of the twentieth century in four chapters. For the organisation of the book we adopted a mixed chronological and thematic approach, although a chronological approach predominates. We strove for a mapping of the ideas and contributions of individuals within their own time and politico-cultural frame as we attempted to highlight developments which cross time and space.

The historical literature on aphasia is vast, and often difficult to access. We have had the pleasure of reading a great deal of this literature and all citations in the book have been read by one or other of us. In the twenty-first century English is the international language in most contexts; but German, particularly, and French dominated scientific discussions in the nineteenth century, and Latin was the obligatory script for earlier European scholars. Before this the written languages of the ancient cultures that emerged around the Mediterranean provide some of the earliest records of aphasia. Most of the nineteenth-century European pioneers of aphasia enjoyed an education that resulted in the ability to read German, French and English, not to mention Latin, and even Greek and Hebrew. To a significant extent, contemporary argument can often pivot on what a writer actually wrote and in what context, but as so much of aphasiology's past is inaccessible to many readers, it was our expectation that many will not have delved too deeply into this history, if at all. The words of the protagonist, even in translation, should enable, we hope, more direct access to their ideas. We wanted therefore to provide actual quotations wherever possible and relevant, and have provided English translations where a good translation did not exist. This original literature appeared predominantly in French, German and English, which adds to the difficulties of access for the reader with limited background in one or more of these languages. When the source was English, the quotation is in English. Some German and French sources are quoted from published English translations and the rest were translated by us. So unless credited to another source, translations are by us.

In an introduction to the history of a discipline a writer is always in danger of missing out some individual or development in theory or practice, and

there are some we have not included, partly intentionally or in response to our reviewers of an earlier draft of the manuscript. Our concern was to include most important and relevant developments and individuals, although it may be inevitable that the reader finds that some aspect or individual contribution is not mentioned. Our criterion for exclusion was our judgement of the impact an individual or their ideas has had on the discipline. But there may be some genuine oversights resulting from our ignorance of a large and highly diverse literature.

This book is an introduction to the history of aphasia, and as such should be relevant to students, teachers, researchers and clinicians in neuropsychology, linguistics, speech and language pathology and therapy, and medicine. The main text of the book assumes some basic knowledge of the general field; a Glossary of basic terms is provided and an Appendix contains introductory material on aphasia and its neuroanatomy. The reader with little background in aphasia or neurology is directed to the Appendix for introductory material.

The six chapters that make up Part I cover the history of aphasia from the earliest records (Chapter 1) and the influence on emerging aphasiology of the Renaissance and 'the age of reason' (Chapter 2). Chapters 3 and 4 cover the pivotal nineteenth century period, which saw the naissance of 'dominance' theory and the birth of modern aphasiology. Chapter 5 maps the theoretical swings and geographical shifts of the early twentieth century and the re-emergence of a 'classical' model in the 1960s takes up Chapter 6. Part I is partially based on a completely revised, rewritten and augmented version of *Geschichte der Aphasie* (2nd edition, 2005) by JT. The four chapters of Part II attempt to examine the second half of the twentieth century until 2000. Important developments in linguistic aphasiology (Chapter 7) and cognitive neuropsychology and cognitive neuroscience (Chapter 8) are examined. Chapter 9 is an attempt to illustrate developments in theory and methodology from 1861 until 2000 through a historical sketch that examines evolving understanding of Broca's aphasia and Broca's area. Chapter 10 looks beyond the classic 'language area' of the left hemisphere and how this has influenced our current understanding of language and aphasia. We end with a short Postscript.

Our intention was not to examine in detail the development of the treatment of aphasia, a significant history in its own right, but to highlight only major themes in this development and their relation to progress in other fields. The development of approaches to treatment, therefore, figures in most chapters in the book. For readers who want a more detailed history of the treatment of aphasia, we can recommend Howard and Hatfield's excellent *Aphasia therapy: Historical and contemporary issues* (1987), which was an invaluable source for us.

We are grateful to the publisher of *Geschichte der Aphasie*, Schulz-Kirchner Verlag, who supported us through technical assistance. We thank our institutions for their support: the Europa Fachhochschule Fresenius (EFF), University of Applied Sciences and the School of Psychology, University of

Exeter. The EFF funded a working translation of *Geschichte der Aphasie*. We extend our gratitude also to Hans-Joerg Baehr, President of the EFF, who supported the project from the beginning.

We are particularly indebted to John Marshall and Jennifer Gurd for generous feedback on the early draft of the manuscript. We thank also two anonymous reviewers for detailed and useful comment on the early draft, and we gladly acknowledge Christy Ludlow and Jordan Grafman, who shared their direct knowledge of studies conducted in the USA on the brain-injured survivors of the Vietnam War. Finally, we thank our publishers, Psychology Press, for their support for this project and their understanding and forbearance when we were unable to complete the book on time because of illness.

Juergen Tesak and Chris Code
Idstein and Exeter

John Marshall died in August 2007, and we dedicate this book to his memory. John was a significant historian of aphasia with a keen understanding of the high relevance of history for the present.

Juergen Tesak
(1961–2007)
Aphasiologist

Juergen Tesak died in early June 2007 following the discovery of a tumour in his leg in the previous November. He was 45. Juergen took up the Chair in Language Therapy at the University of Applied Sciences (Europa Fachhochschule Fresenius) in Idstein, Germany, in August 2002. He became Dean of the Faculty of Health and built up the courses in speech and language therapy, occupational therapy and physiotherapy. He also introduced the first bachelors degree in logopedics in Germany.

We were colleagues for some time, but our work on this book brought us together a lot more and Juergen spent some weeks in England with my wife and me in the spring of 2006 as we worked to make progress with our book. It was good to get to know him better. We made excellent progress with our work at my dining-room table and we enjoyed each other's company.

Before Juergen arrived, I had warned my wife that he was very tall and we were worried that he would not fit into our spare bed. We were concerned too that he would not like our English food, but he was gracious and good company and took delight in trying English 'cuisine'. We visited several local pubs and he enjoyed steak and kidney pie, and sausage and mash washed down with Devon ale. Within a few months of his trip to England, a tumour was discovered in his leg.

I learnt a great deal from Juergen about the history of aphasia, especially the rich German literature on the subject, on which he was a leading expert. He is a great loss to aphasiology and I am not alone in having lost a good friend and a close colleague.

Chris Code
Exeter
September 2007

Part I

The older history of aphasia

1 Aphasia in antiquity and the Middle Ages

These days we think of aphasia and other cognitive impairments as a result of damage to mental processes represented in the brain and other parts of the central nervous system. This was not always so, and the route by which we came to our current understanding of aphasia and aphasiology is the subject of this book.

In this chapter we start with what the ancients knew of aphasia and the role of the brain in language, other cognitive processes and behaviour from the surviving writings that have come down to us. The period from antiquity to the Middle Ages stretches from the beginnings of recorded history to the fall of the Roman Empire in the fifth century AD, and seen from a Eurocentric perspective, the civilisations of the ancient Mediterranean (Egypt, Greece, the Roman Empire) and the Christian traditions of the Middle Ages played the central role. In ancient Egypt, and later in Greece, the heart rather than the brain was considered of central importance. In the Middle Ages the idea that human illness could be understood in terms of an imbalance of bodily fluids was the dominant assumption. When a role for the brain began to emerge, it was the large ventricular spaces in the brain, and the fluids they contained, rather than the substance of the brain that was seen as the seat of the human soul.

Aphasia has probably existed since humans have been able to speak, though when speech first emerged is a matter of hot debate, and may never be known with any reasonable certainty. True syntactically organised language probably did not emerge until 2 million years ago when early humans (*Homo erectus*) are thought to have left Africa to populate the world (Corballis, 2002). Modern humans enjoy a rule-governed capacity to generate an infinite range of utterances from a finite set of elements – what is called *recursive* syntax by many, but modern human language may have been preceded in its emergence by some form of *protolanguage*, which is seen as a stage preceding the development of full syntax (Bickerton, 1990). Bickerton (1990), for instance, argues that infant language, pidgin languages, and the languages taught to apes in captivity are all protolanguages made up of utterances comprising a few words, without syntactic structure beyond basic word order. Some form of aphasia, we can assume, has existed for as long as human beings have been able to use protolanguage.

The fossilised skulls and other bones of early humans (*Australopithecus*, *Homo erectus*, Neanderthal, etc.) provide only indirect indications of the evolution of brain size and jaw shape and movement, but fossilised skulls show different forms of partly healed cranial trauma (Finger, 1994: 3f.), and several millennia-old skull findings with trepanations (systematic drilling of varying geometric holes in the cranial bone as some form of treatment for illness) seem to suggest that it has been clear for a long time that humans have made a connection between the head, the brain, illness and behaviour, although the exact purpose of these trepanations remains unclear.

Naturally, the first mentions of speech and language impairments in history do not appear until after the development of systems of writing in the Middle East, which was around 3500 BC (Crystal, 1987).

Ancient Egypt

The earliest written traditions of medicine come from the Ancient Egyptian high culture. Many histories of medicine and surveys of the history of aphasia begin with the **Edwin Smith Papyrus** (Bouton, 1991; Critchley, 1964b; Gibson, 1962, 1967; Howard & Hatfield, 1987; O'Neill, 1980; Sondhaus & Finger, 1988), which is a later copy of a medical, surgery-oriented case collection that is approximately dated between 3000 and 2200 BC. Figure 1.1 shows a transliteration from the hieratic into the hieroglyphic, as printed in Breasted (1930: Table VIIa/VIIIa). One part of the text is attributed to the celebrated Egyptian physician and politician **Imhotep** (approx. third century BC). A total of 48 cases are presented in the papyrus, each following the pattern of examination, diagnosis, treatment, and is structured following the 'from head to toe' principle. A large number of the 48 cases described had head injuries (Ackerknecht, 1992: 21)

At least five of the cases display evidence of speech and language problems. Case 17 is described as 'speechless' because of a fracture of the upper jaw, and cases 19, 20 and 22 exhibit injuries to the temporal area of the skull and are described as 'speechless'. There is no differentiation between central (caused by brain injury) and peripheral (damage outside the central nervous system) disorders. Case 20 probably had a traumatic aphasia: one part of the description reads as follows:

> One having a wound in his temple, penetrating to the bone, [and] perforating his temporal bone; while he discharges blood from both his nostrils, he suffers with stiffness in his neck, [and] he is speechless. An ailment not to be treated.
>
> (Breasted, 1930: 286)

Figure 1.1 shows cases 19 to 21 in the original hieroglyphics and the framed part corresponds to the quoted paragraph. The double-framed part reads 'he is speechless'. Although it is stated that treatment is not possible, it is

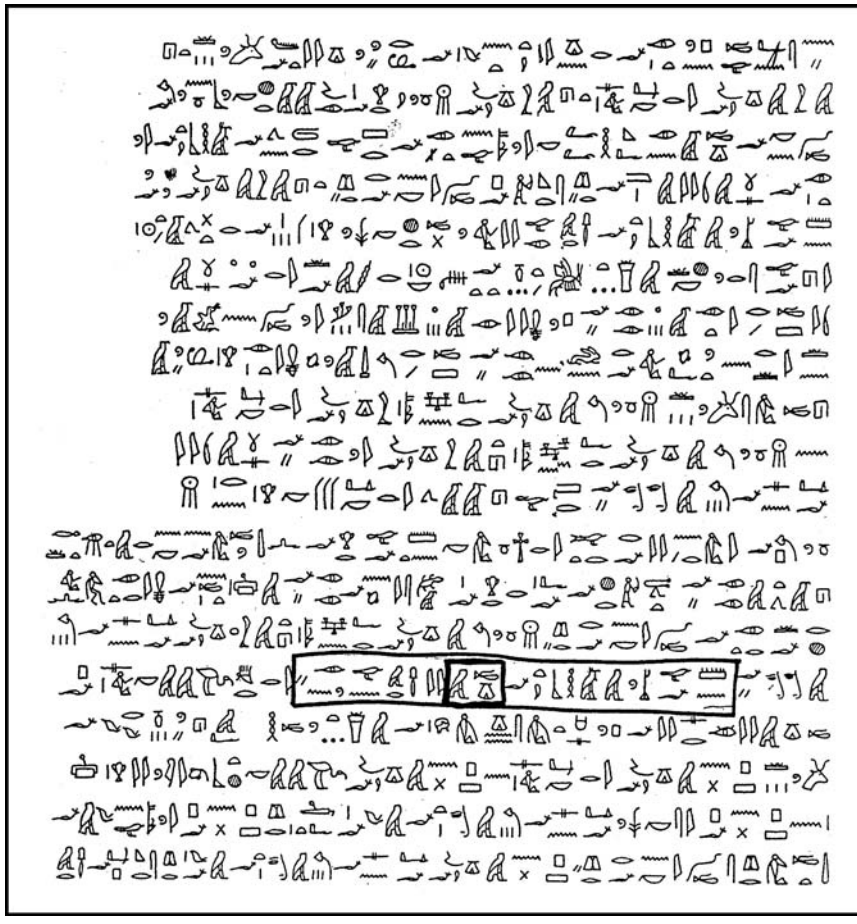


Figure 1.1 Excerpt from the hieroglyphic transliteration of the Edwin Smith Papyrus (Breasted, 1930: Table VIIa/VIIIa). This illustration shows cases 19 to 21 in hieroglyphic transliteration; the part in the single frame corresponds to the excerpt quoted in the text. The part in the double frame signifies '[and] he is speechless'.

proposed that the rubbing of ointment on the head and pouring a fatty liquid into the ears is a beneficial therapy.

Although it becomes clear from this observation and others that cranial and cerebral trauma can, for example, cause mobility, perception, and speech disorders, no connection is inferred between the brain and the symptoms. The brain was generally afforded no great importance in Egyptian medicine (or religion). While in the Egyptian mummification of the dead all the organs were stored, the brain was pulled out through the nose with a hook and discarded. This is, among other things, a reflection of the **cardiocentric** view (Joachim,

1890) dominant in Ancient Egypt, which considered the heart as the home of the soul, where a person's capacity for Good and Evil resides.

Ancient Greece and Rome

The Greek spiritual world played a prominent role in early European thinking and for the early history of medicine and early Greek medical science formed the basis of modern medicine in its current form (Ackerknecht, 1992; Bouton, 1991: 1–30). Roman medicine was a continuation of the Greek tradition (for example, 'Roman' physician Galen wrote in Attican Greek) and is therefore considered within the tradition of Greek medicine.

The theory of fluids

In the first pre-Christian millennium the notion that there were four fundamental elements (air, fire, earth, water) from which everything else is built, was inherited from Mesopotamia and Egypt. This four-element theory was developed further by different philosophers within the framework of natural philosophy (for example by Empedocles, 504–433 BC), in an attempt to understand the facts of nature and the essence of human nature. Certain physical characteristics and human bodily fluids were attributed to the basic elements as shown in Table 1.1.

Since bodily fluids played a crucial role, this approach is also called the **theory of fluids** (see Ackerknecht, 1992: 38–39; see also Finger, 1994: 12f.). Using this approach the causes of diseases were quite consistently considered as due to some imbalance of the bodily fluids. Attempts at healing hence consisted primarily in manipulating the balance of fluids through bloodletting, starvation, fluid deprivation, heat treatment, regurgitation, faecal evacuation and sweating. Epilepsy, for instance, was interpreted as an excess of cold phlegm, which treatment attempts to reduce by means of thermal cures. Deficits after cranial injuries were interpreted as an accumulation of undesirable life fluids. Cranial drillings – trepanations – were sometimes attempts at the evacuation of undesirable fluids, and in some cases may have been effective. This might have been due to a reduction of intracranial pressure. Without the theory of fluids the fundamental medical and early psychological thinking of

Table 1.1 The relationship between the elements, physical characteristics and human bodily fluids

Element	*Characteristic*	*Bodily fluid*
Air	Dry	Yellow bile
Fire	Warm	Blood
Earth	Cold	Phlegm (mucus)
Water	Moist	Black bile

the subsequent centuries is difficult to understand and the theory remained the basis of many model representations of human physiology and medical intervention until the eighteenth century. Ackerknecht (1970: 95) shows that the use of the 'unholy trinity: bloodletting, emetic (vomiting), and laxative' played a crucial role up to the nineteenth century. These purges often had negative consequences (not uncommonly the patient's death).

Early notions of localisation of mental faculties

Below we sketch the most important thinking of the Greco-Roman period concerning the connection between cognitive processing and a possible **localisation** in the structure of the human body. Essentially, the question centred on whether the human mind is represented in the brain or in the heart. Therefore, crucial findings of Greco-Roman anatomy and physiology are also briefly discussed.

Early writers **Alcmaeon** (approx. 500 BC) and **Anaxagoras** (500–428 BC) considered that the brain was the organ of perception and thinking. The philosopher **Plato** (428–347 BC) developed the ideas of Democritos and postulated a tripartite soul that also corresponded to anatomically different parts of the human being. Reason and mind were located in the head; higher characteristics such as pride, fear, courage, and wrath were in the heart, and lower characteristics such as lust and desire were located in the liver or in the abdomen. As human speech had been associated with the rational part of the soul since **Pythagoras** (580–428 BC), this was an important step for the examination of the relationship between speech, language and brain.

Anatomy was not very precise at this time because anatomical investigations were made neither on human beings nor on animals. Alcmaeon assumed, for instance, that the human nerves were ducts through which flowed the different fluids. Although Alcmaeon reported that he had conducted anatomical sectioning, these were not carried out systematically until the third century BC in Alexandria, mainly by Herophilos (335–280 BC, according to Finger, 1994) and Erasistratos (310–250 BC). Both carried out dissections on criminals (Finger, 1994: 14) who were still alive and put at their disposal by the first ruling Ptolemaeans, who were very interested in science and who set the course for the future development of medicine.

Herophilos (335–280 BC) is seen by many as the 'father of anatomy' and he had a distinct interest in the brain. He described the parts of the brain, the cortex, the cerebellum and the different ventricles of the brain and distinguished between sensory and motor nerve trunks. Herophilos is also considered the founder of **ventricular theory** where a connection was made between the 'psyche' (soul) and the ventricles of the brain (Gibson, 1967).

Erasistratos (310–250 BC), in contrast, attributed cognitive functions rather to the material substance of the brain than to the ventricles. Based on his comparative studies of the cerebellum and the cerebral surface, he maintained that there was a connection between the size of the cerebellum and the running

speed of animals, for instance. Likewise, he states that there is a connection between the complexity of the gyri and mental performance (including that of human beings). Erasistratos therefore was the first to develop a **theory of localisation** in its narrower sense, which relates specific cognitive *functions* to specific *structures* of the brain.

However, Erasistratos also developed the theory of the 'pneuma' on the basis of the theory of fluids. Here he proposed that inhaled air is transformed into 'vital pneuma' in the left ventricle of the heart and, together with blood, results in heat, energy, and life. Via what Galen called the ***rete mirabile*** (the 'wonderful net', a plexus at the base of the brain, subsequently found not to exist) a part of the vital pneuma penetrates into the brain, where it turns into 'psychic pneuma'. This psychic pneuma processes sensory perceptions and renders possible understanding and knowledge. The difference between animals and humans was established by the psychic pneuma especially, which only humans possess; animal life was the result of vital pneuma alone. But Eristratos also localised the soul itself in the fourth ventricle.

This short overview of the first emergence of early ideas on the role of the brain should not belie the fact that the dominantly widespread view at the time was the **cardiocentric perspective**, adopted from Egyptian culture, where the spirit (or soul) of a human being was located in the heart. Proponents of this view were Empedocles in the fifth and **Aristotle** (384–322 BC) in the fourth pre-Christian centuries. The works of Aristotle had a particularly significant impact in subsequent centuries. He did not follow the views of his teacher Plato, who localised reason in the brain (Clarke, 1963: 4ff.), but fervently argued that the heart was the home of all cognitive, perceptual, and associated functions. One argument put forward by Aristotle was that the heart feels warm and the brain cold to the touch and he surmised that the brain performs a cooling function for the blood. The human brain, he reasoned, was so large because the human body has a higher body temperature than animal bodies. Aristotle also discussed in detail why the sensory organs – ear, nose, and eyes – are attached to the head although, he suggested, they have no connection with the brain, only a direct connection with the heart.

Aristotle's conception of memory (which he also supposed to be located in the heart) was also central until the eighteenth century. He assumed that external events engender movements in the sensory organs which are then transported into the heart by the pneuma, where they remain (with reduced activity) as memory impressions. The structure of memory and the act of remembering required the **association** and correct order of the laid down impressions.

Aristotle's authority was so influential that the cardiocentric view allowed for little interest in the brain in Europe for over 1500 years. Equally influential until the seventeenth century was the Greco-Roman physician Galen from the second century AD (May, 1968; Singer, 1956; Temkin, 1973; Zimmer, 2004).

According to Ackerknecht (1992: 55) **Galen** (Galenus, AD 130–200; see Figure 1.2) is the 'most significant medical experimentator [. . .] of the entire history of medicine until the 17th century', particularly, from our perspective,

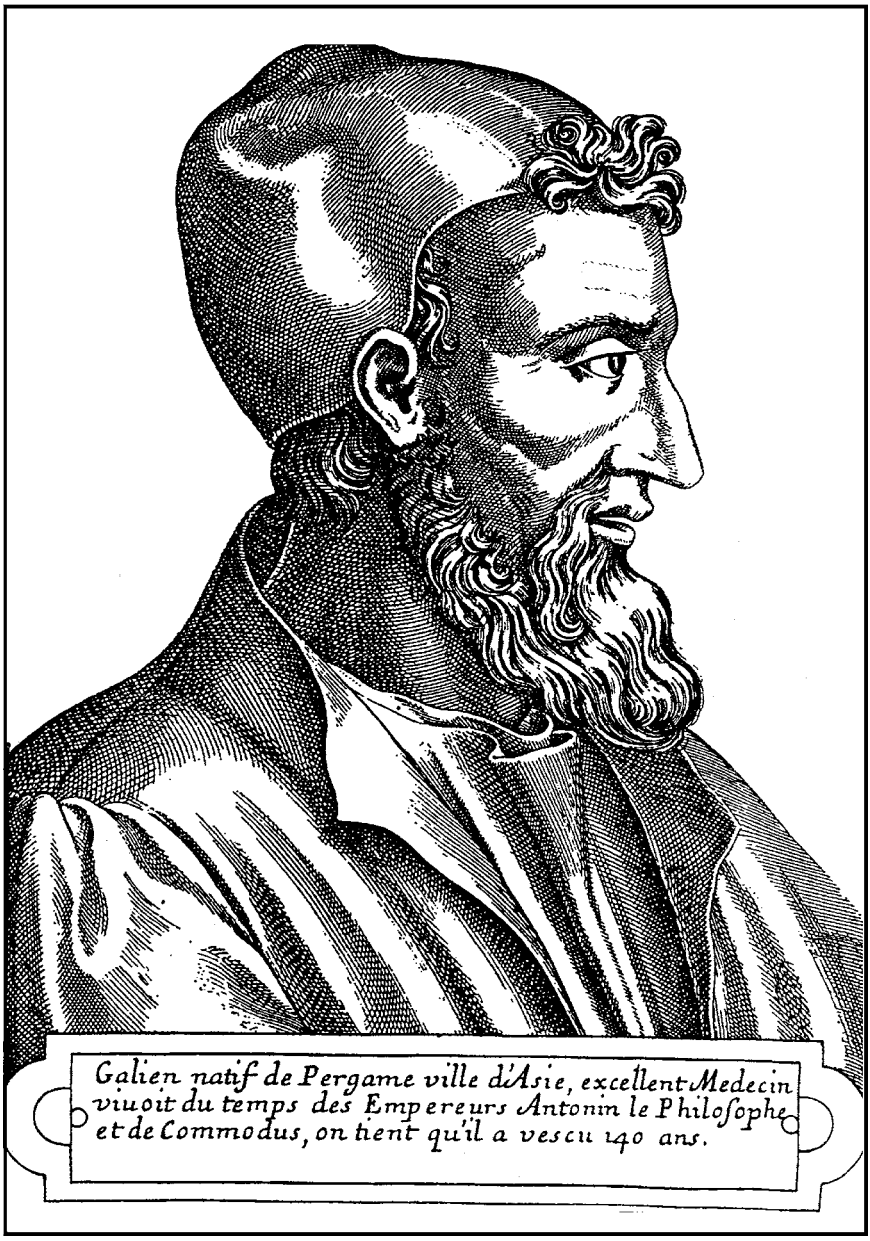


Figure 1.2 Greco-Roman physician Galen (AD 130–200), whose anatomical investigations and writings strongly influenced European medicine until the seventeenth century, and who proposed influential early ideas on the human brain.

for his pioneering work on the anatomy and physiology of the brain. Although Galen was a follower of Aristotle philosophically, he rejected Aristotle's 'brain theory' (especially the proposed cooling function of the brain).

Galen was empirically oriented and dissected different animals (cows, monkeys, pigs, dogs, cats, rodents, and at least one elephant), but no humans, a practice prohibited in Rome at his time. But as a physician to the gladiators, he certainly had access to patients with different injuries, including injuries to the brain. He discovered sensory and motor nerve tracts and attributed perception and motor function to different parts of the brain. The connection between gyri and intelligence (as maintained by Erasistratos) was rejected by Galen who pointed out that donkey brains are also very large and have many gyri.

Galen also described the ventricles and, like Herophilos one hundred years before him, suspected a connection between the ventricles of the brain and human intellectual faculties. This **ventricle theory** or **cell theory** would be the dominating one throughout the Middle Ages. Imagination was localised in the two lateral ventricles (understood as the first 'cell'), reason in the third ventricle (the second cell) and memory in the fourth (the third cell). Consequently, a brain injury could cause damage only in the sense of functional deficiency (in motor and sensory function) if the ventricles are also affected. The human soul (the spirit), Galen suspected, was either in the *rete mirabile* at the base of the brain, or also in the ventricles.

Galen's physiology also had a lasting impact. Via a complicated duct system (nerves were also considered as ducts) nutrients entered the liver, where the ***spiritus naturalis*** (the natural spirit) transformed them into blood. From there, the blood reached the right ventricle of the heart, among other things, where ***spiritus vitalis*** (life spirit) was produced. The enriched blood was transformed into ***spiritus animalis*** (animate spirit) by the *rete mirabile* and then reached the brain. Surplus *spiritus animalis* was stored in the ventricles until it was needed. From there, via the hollow nerves, it reached the muscles, in order to control movements, or the sensory organs, in order to process memory impressions.

Early descriptions of speech and language impairments

There were many observations of neurogenic speech and language disorders in antiquity (Benton & Joynt, 1960; Critchley, 1970; Finger, 1994; esp. O'Neill, 1980) but we can summarise in advance that the accounts from the Greco-Roman period show that although many observations were made and some symptoms described, there was nothing like a theory offering any explanation of the causes of speech and language disorders.

The **Hippocratic writings** were major milestone in the history of medicine (Ackerknecht, 1992; Chadwick & Mann, 1950). Hippocrates (460 BC–*c.* 370 BC) himself lived at the turn of the fifth to the fourth pre-Christian century, but the writings originate from different authors and from different periods (*c.* 420 BC to AD 100). What is relevant for us is that they contain a series of

descriptions of speech and language disorders (O'Neill, 1980: 21ff.), although it is unclear in most cases what exactly caused the problem, how the pathology manifested itself and whether it is a disorder of voice, speech or language that is being described. Often used Greek terms are *aphonos* and *anaudos* (literally 'without voice' and 'without hearing') and these terms may also mean muteness. One case was that of patient Piliscus, who lost his speech and died after a few days. A relatively clear case of transient aphasia was also briefly described in a young pregnant woman who apparently had a stroke and 'lost her power of speech, [and] the right arm was paralysed' (Chadwick & Mann, 1950: 53). On the fourth day after the event the young woman's speech was restored.

The connection between speech and language disorders and apoplexy (stroke) is noted in the Hippocratic writings (Chadwick & Mann, 1950: 230), as well as first indications of the connection between the side of the lesion and impairment of the opposite body half (Bouton, 1991: 14). But, because of the lack of relevant knowledge on the physiology and functionality of the brain, the concept of laterality did not yet develop. Altogether, the Hippocratic writings are rather unsatisfying with respect to aphasia (and also other communication disorders), mainly because observations are but vaguely described and because no cause–effect relationships are hypothesised.

The Roman **Valerius Maximus** (*c.* AD 30) described a case of *alexia* (reading disorder), and **Aurelius Cornelius Celsus** (25 BC–AD 50) suspected that most speech disorders were caused by paralyses and other impairments of the tongue, not to disorders of the brain. **Soranos of Ephesus** (AD 98–135) differentiated between disorders caused by paralysis of the tongue and disorders of articulation or loss of language from other causes (see Finger, 1994: 372; Sondhaus & Finger, 1988).

Plinius (*c.* 23–79 AD) described anomia, alexia and agraphia in his work *Naturalis historia*. In one case a man who was hit on the head by a stone could no longer read and write, but showed no other cognitive deficits. Another man fell from a building, injuring his head, and subsequently could not recall the names of his friends and relatives (O'Neill, 1980: 45). These symptoms are mentioned in the chapter on memory and memory disorders, implying that for Plinius they are **memory impairments**. Other cases of language disorder (e.g., the case of speaker Mesalla Corvinus) are probably due to dementia (Sondhaus & Finger, 1988: 89). Plinius is sometimes regarded as the original founder of modality-specificity and of the concept of selective impairment (of memory) (Critchley, 1970: 55). Galen also described speech and language disorders and stated in similar fashion to Plinius that lexical memory can be impaired by head injuries (Finger, 1994: 372).

Finally, **Caelius Aurelianus** (fifth century AD) differentiated between loss of voice, confused speaking in epilepsy, and speech and language problems in stroke: 'trembling voice, unclear articulation of words, interrupting the flow of speech without reason in the middle of speaking, or forgetfulness in terms of what was just said' (Drabkin, 1950: 329). Caelius Aurelianus also noted that language disorders do not affect motor speech function and that paralysis

of the tongue can affect swallowing as well as speech: 'when the tongue or the parts that are needed for swallowing are paralysed' (Drabkin, 1950: 481).

While **Sextus Empiricus** (AD 200) was allegedly the first to systematically use the Greek word **aphasia**, it was in a different philosophical context, meaning in a state of mind in which one expresses neither agreement nor rejection (Benton & Joynt, 1960: 207).

The Middle Ages

The Middle Ages is usually considered the period between the demise of the Roman Empire and the beginning of modern times with the emergence of the Renaissance. It approximately spans the period from the fifth to the fifteenth centuries. The Middle Ages are generally associated more with regression and chaos than with progress and the increase of knowledge, although there were significant contributions, for instance, the understanding of the calendar. Christian monasteries played a crucial role as keepers of antique knowledge and sites of learning, and, unlike European culture, Arabic culture flourished. For the topic of this book, the antique concepts of the nature of the human body and illness were passed down in the Middle Ages, but hardly anything new was added. Aristotle remained dominant in philosophy, and Galen in medicine. The ruling and most successful brain theory was ventricle or cell theory. If the temporal longevity of a theory is taken as the main criterion, this makes ventricle theory the most successful theory in the history of neuroscience.

Ventricle (cell) theory

As discussed above, ventricle or cell theory (Clarke & Dewhurst, 1972: 11–48) originated with Herophilos and Galen; the early fathers of the Christian church (Nemesius, Augustinus, Posidonius, et al.) adopted the tradition (Finger, 1994: 18f.). But many of the two Greek physicians' anatomical insights were lost and the ventricles were understood in the Middle Ages rather as theoretical concepts rather than anatomical quantities (and simply depicted as circles or similar – see Figure 1.3). Hence the label 'cell' theory is often considered preferable to ventricle theory, as the ventricles were conceived as cells, even though the terms are often used synonymously. Cell theory at this time existed in different forms. For example, the fathers of the church from the third to the fifth centuries discussed the attribution of discrete capacities to discrete cells, and the number of cells also varied (typically from three to five cells).

A crucial innovation of the tenth century was the introduction of a dynamic element into cell doctrine, arguably the first elementary **information flow diagrams** were created. In Figure 1.4, we see a depiction of a variant of cell theory from the Middle Ages which briefly sketches the supposed physiological progression from sensory input to cognitive processing in the frontal lobes.

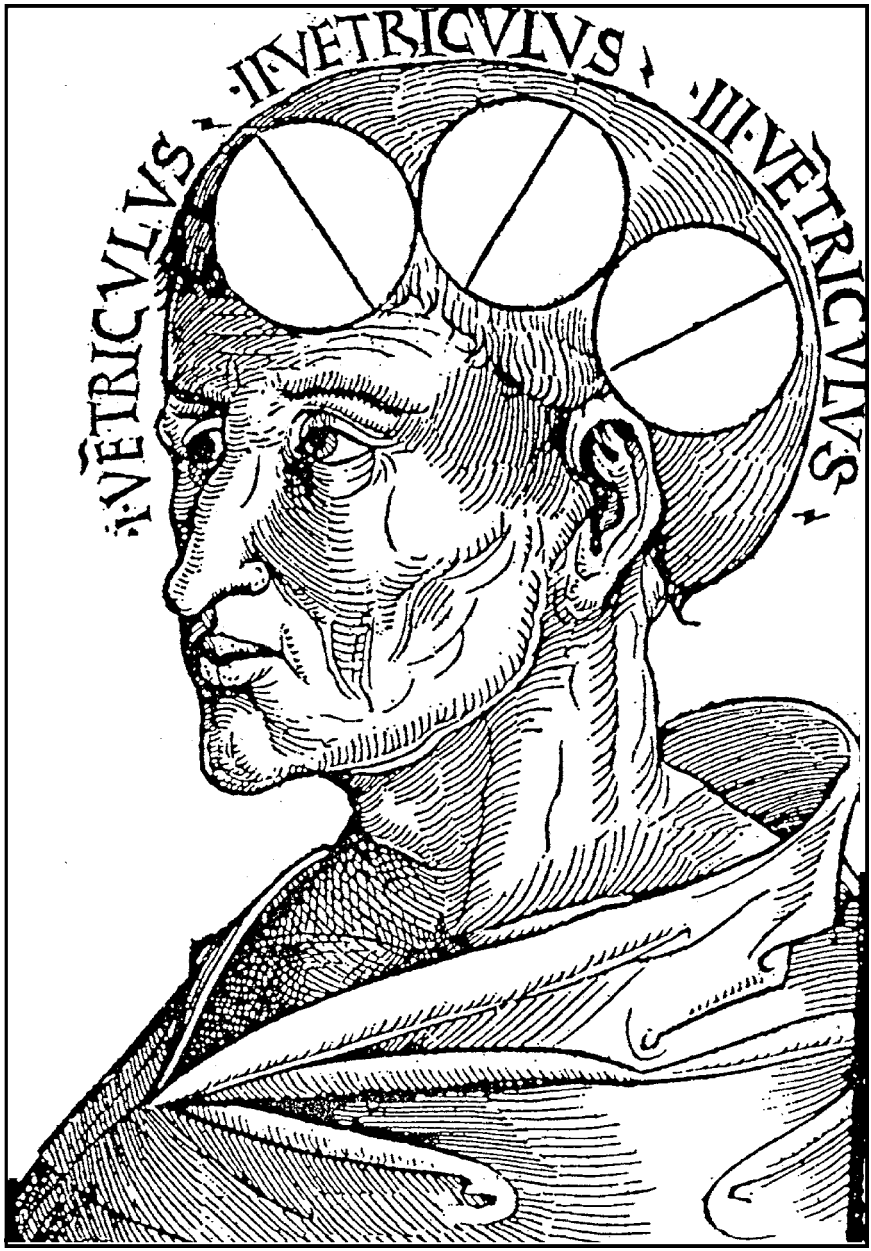


Figure 1.3 The ventricle from a medieval perspective (illustration following *Philosophia pauperum sive philosophia naturalis* from 1490).

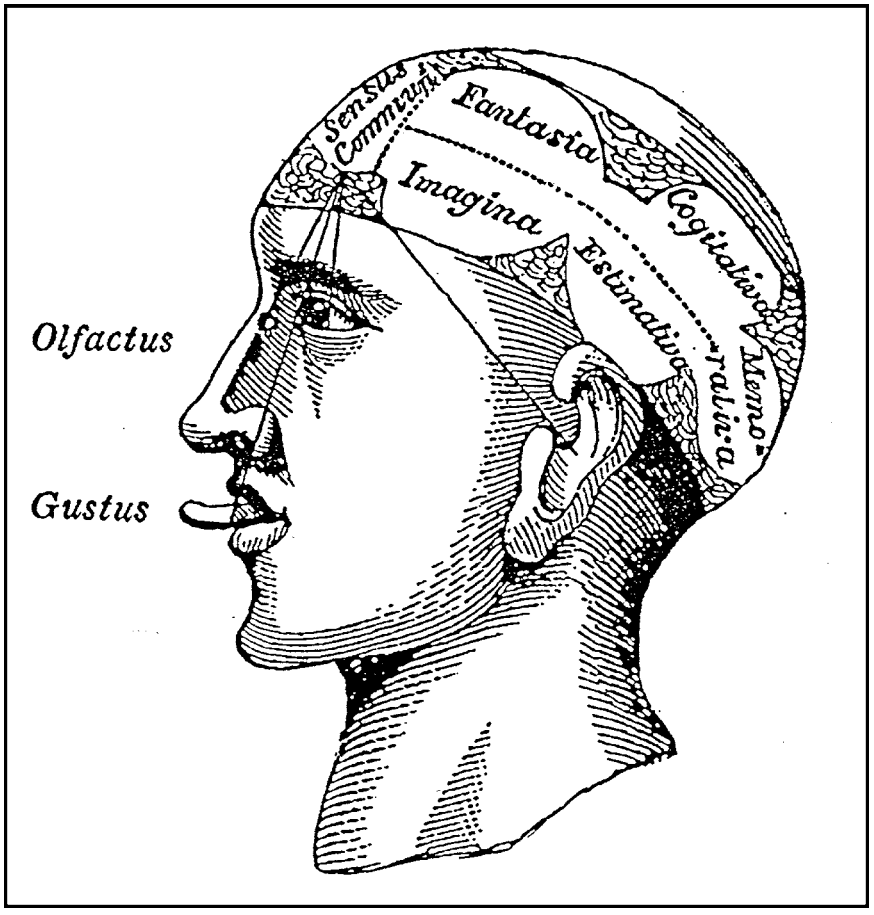


Figure 1.4 Cell theory in the Middle Ages (illustration following Blumenbach, 1840: 370).

Anterior to the cells, located in the frontal area of the brain, is the *sensus communis* ('common sense'), which receives information from the exterior sensory organs (e.g. gustus 'taste' and *olfactus* 'smell'). From these impressions, images are created in the first cell (*imaginativa* and *fantasia:* 'imagination'). In the second cell are contained rational characteristics: *(a)estimativa* (power of judgement) and *cogitativa* (thinking). The third cell contains memory (*memorativa*). Impressions from the outside are thus first transformed into images, then thought over and evaluated, in order to finally be stored in memory. In other models, motor function and will are also ascribed to the second and third cell.

Following damage to the brain, Nemesius and Posidonius, for instance, maintained that lesions of the frontal ventricle impair imagination, whereas

injuries in the occipital ventricle damage memory. It seems that aphasic symptoms are considered to result from damage to the third cell (the fourth ventricle) and conceptualised as **memory disorders** (Finger, 1994: 372). So functional localisation of higher functions is part of cell theory and the diagrammatic illustrations, as unrealistic as they may be anatomically, can still be interpreted as basic anatomical models.

Summary

From antiquity to the Middle Ages there were many early reports of aphasic symptoms as a consequence of damage to the brain, and the first interpretations of them developed in terms of localisation of some kind of damage or disturbance to some part of the body or its systems. Ventricle or cell theory emerged in the Middle Ages and established a connection between brain structure (the ventricles) and cognitive function (e.g., memory). Since language performance was understood as memory performance, aphasic impairments were attributed to a disorder of memory, i.e., damage to the ventricle that contains memories – often a ventricle located in the posterior part of the brain. Aphasia at the end of the Middle Ages was thus seen as a disorder of memory.

2 From the Renaissance to the eighteenth century

The Renaissance (the 'rebirth' of Antiquity) emerged as triumph over the darkness of the Middle Ages. It began in the fifteenth century in Italy and spread throughout Europe; we associate it with the beginnings of modern science and modern medicine. Crucial new insights were gained in the areas of anatomy and neurology, and although ventricle theory, for instance, remained highly influential for a while longer, the foundations for its demise were laid. The period from the Renaissance to the seventeenth century yields, for the first time, more detailed descriptions of aphasia and, in the eighteenth century, Gesner developed the first 'theory' of aphasia from his observations of people with aphasia.

The Renaissance to the seventeenth century

In the period from the Renaissance to the seventeenth century, crucial advances were made in the study of the anatomy and physiology of the brain and there were increasing attempts to connect behavioural and cognitive functions to specific structures of the brain. Descriptions of aphasia contained more precise elucidations of symptoms and first hypotheses on its causes.

The development of medicine in the Renaissance

First we present a few central personalities and their insights on medicine and philosophy. **Leonardo da Vinci** (1472–1519) is important because he made significant advances in anatomy. Using empirical methods (sections on animal and human corpses, wax castings, etc.), he produced exact anatomical sketches far superior to those of the medieval tradition. For example, he observed that there was only a vague connection between the medieval drawings of ventricles, and his own findings. Interestingly, however, Leonardo did not doubt the doctrine of ventricle theory.

In 1543, **Andreas Vesalius** (1514–1564) published his famous and beautiful book *De humani corporis fabrica* (*On the Fabric of the Human Body*), whose seventh and last volume is dedicated exclusively to the brain (see Figure 2.1). This book was a breakthrough in anatomical detail and neurology and it

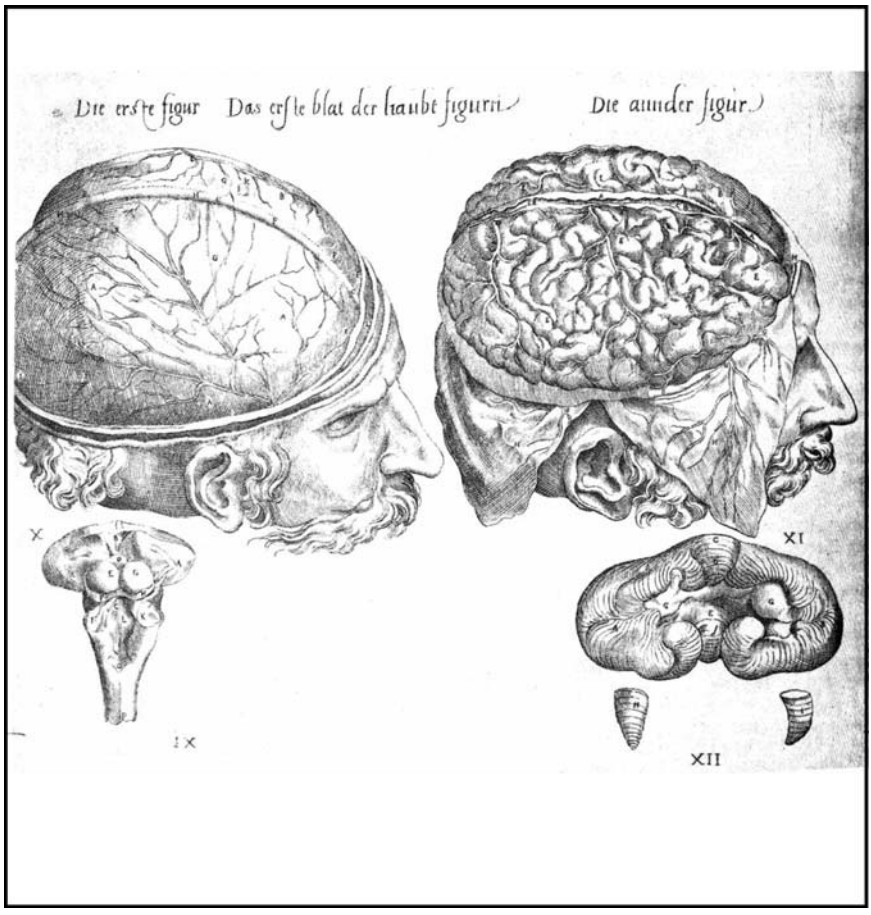


Figure 2.1 Brain picture from Vesalius' famous book *De humani corporis fabrica*, which appeared in 1543.

dismissed much of Galenian anatomy (for instance, the *rete mirabile* discussed in Chapter 1 was deemed non-existent in the human body). The ventricles are described in detail, and memory is not localised there, but in the cerebellum instead. Vesalius also emphasised that one could not fathom the functions of the brain by way of brain anatomy alone – a later often forgotten insight.

Thomas Willis (1621–1675; see Figure 2.2) was Professor of Medicine at Oxford and gained his knowledge of the brain from his observations of his patients with neurological impairments. He was of great importance for the developing neuroscience of the seventeenth century (Finger, 1994: 23ff.; Zimmer, 2004) and his great work *Cerebri anatome* (*Anatomy of the Brain and Nerves*, 1664) benefited from the anatomical drawings of the young architect

Figure 2.2 Thomas Willis (1621–1675), whose work *Cerebri anatome* represents a milestone in the history of neurology.

Christopher Wren. However, Willis was preoccupied not only by the anatomy, but also by the functions of the brain. He postulated that the **cerebral gyri** are responsible for **memory** and the **will**. He dismissed ventricular theory and concluded that mental life was essentially dependent on the cortex, providing the first cortical theory of the control of muscles and reflexes (Bennett & Hacker, 2003). He proposed a corporeal soul present in humans and animals and associated with vital spirits, a kind of distilled liquor, that was made in the brain and circulates in the blood. The soul was conceived by Willis as immortal, non-material and separate from the brain, with interaction between body and soul taking place in the corpus callosum (the connecting fibre between the two hemispheres) which was also concerned with imagination (Bennett & Hacker, 2003). The striatum (in the basal ganglia) is connected with perception and movement, while fundamental vital and involuntary systems are connected with the cerebellum. Those characteristics that figure in the discussion of the central nervous system during the Middle Ages were localised by Willis, but his localisation was performed on a more systematic anatomical basis. Finally, the size of the cortex also played an important role for Willis, an aspect later important for the ideas of phrenology.

During the Renaissance and the subsequent centuries, sectioning and examining human beings was still prohibited by the church, because man, as God's image, was not to be violated by the anatomist's knife. Many scientists therefore took a considerable risk in order to quench their thirst for knowledge. One solution to the problem comes from the work of French philosopher **René Descartes** (1596–1650) in the seventeenth century.

Mechanics and hydraulics were the most highly developed technologies in the seventeenth century. The French king had a garden with mechanical animals and people that could move in near life-like manner by means of complicated duct systems. Descartes was impressed by this and asked himself if human beings are not also steered by ducts (veins, nerves, etc.). As a matter of fact, Descartes described man as a machine and as a **mechanical automaton** in his work *De homine* (*On Man*). However, this automaton becomes a true human only by virtue of the divine soul and, when the body dies, the soul can nevertheless live on. The difficult question is where the soul has its home and Descartes suggested that it is the **pineal gland** lying at the base of the brain, through which the (immaterial) soul can enter and leave the body. For Descartes the unity between soul (*res cogitans*) and body (*res extensa*) is possible only in humans, a position called **Cartesian dualism** and it continues to have influence in current thought. This Cartesian separation of human body and soul allowed the church to lift its ban on anatomical sectioning and the basis for further development of medicine in the eighteenth and nineteenth centuries was established.

Descartes' **mechanistic world view** and physiology is rudimentarily illustrated in Figure 2.3. What we see mapped is the path of light (from an object ABC) onto the retina, upon which an image is produced. From there, the sensory stimulus is conducted to the ventricle walls across the ducts (which represent the optic nerve). So, ventricles still played an important role for Descartes. Through the liquid in the ventricles the information reaches the pineal gland (H), which acts as some kind of sensory-motor junction of transition, where is located the *sensus communis*, which then recognises the objects. The pineal gland also directs the distribution of the *spiritus animalis*, which is located in the ventricles and, from there, directs movement and motor function by streaming through hollow nerve ducts. Descartes' memory model is also built in. Memory impressions available to the soul are nothing more than pores in the brain in which the *spiritus animalis* originally flows.

Incidentally, the pineal gland was chosen by Descartes as the location of the soul because it is the only structure in the brain not to appear in double and because it is surrounded by cerebro-spinal fluid, precisely where the *spiritus animalis* was located. With his simple afferent (= leading to) and efferent (= leading away) conduits (with the *sensus communis* as interface), Descartes can be seen also as a forerunner of reflex theory and, with his memory model, as a forerunner of associationist theory. Discussion of the 'soul organ' began with Descartes too and lasted until the end of the eighteenth century (Hagner, 1997).

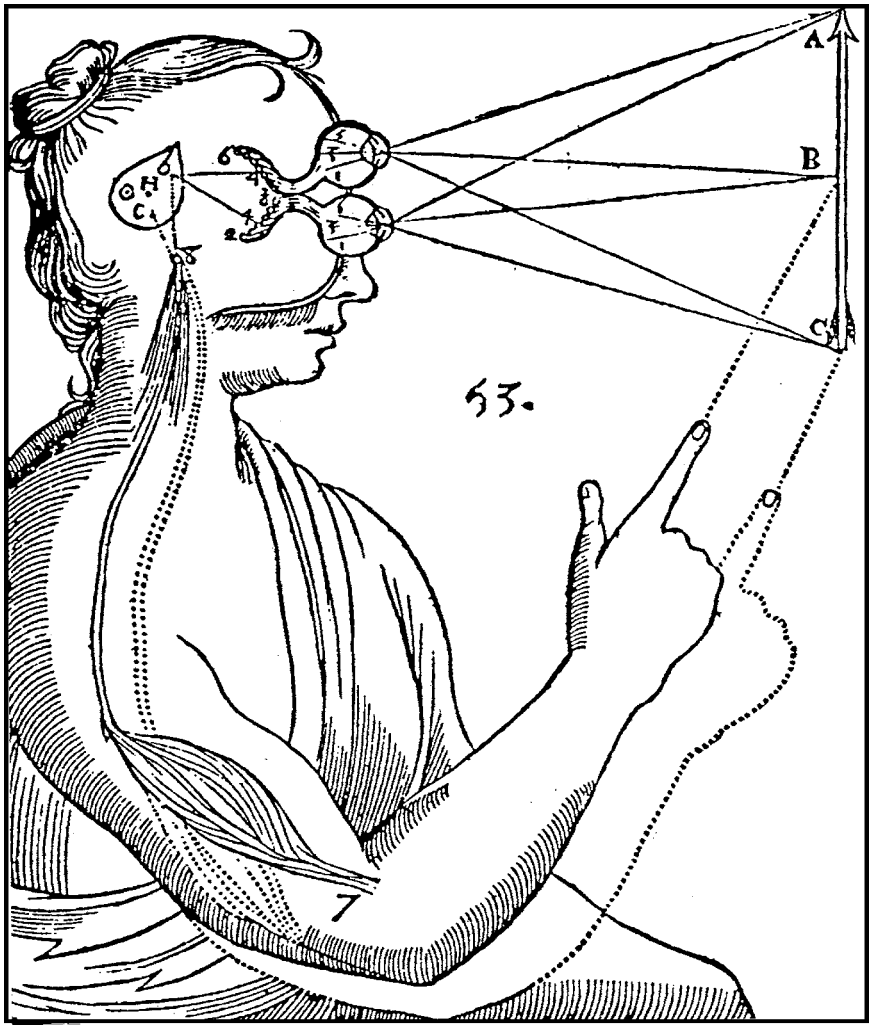


Figure 2.3 Drawing from *De homine* by René Descartes (1596–1650) (see text for explanation).

Descartes was heavily criticised, but the criticism typically related to anatomical aspects of his work and less to conceptual and methodological ones. One important critic was **Nikolaus Steno** (Niels Stensen, 1638–1686), who rightly found fault with the anatomical imprecisions in Descartes' work (Clarke & Dewhurst, 1972: 70ff.). In the second half of the seventeenth century the supposition is accepted that the cortex (and not the pineal gland) produces the *spiritus animalis* (as maintained, for instance, by Willis), a point of view that survived until the nineteenth century (Clarke & Dewhurst 1972: 83f.).

On aphasia

In the fifteenth century, **Antonio Guainerio** (died 1440) reported on two patients who were aphasic following head injuries, one of whom could say only a few words, another with paraphasic naming. Guainerio hypothesised that the cause of the aphasic symptoms was damage to the fourth ventricle (the third cell) in the back of the brain. Memory is impaired, he proposed, because the ventricle contains too much phlegm. Guainerio's explanation was thus based on the classical as well as within the medieval tradition, where language is a function of memory and a language disorder was seen as a memory disorder caused by damage to the fourth ventricle.

Cranial surgery was also performed in the Renaissance, for example, to retrieve a bone splinter from the brain and there are some early examples of recovery of language without language therapy. Howard and Hatfield (1987: 22) write that such a case was described by **Nicolò Massa** (1489–1569), concerning a man who lost his speech following a battle wound to the head. Although the other surgeons present disagreed, Massa is of the opinion that a bone splinter must have been left in the brain matter. He found it, pulled it out and immediately the patient called out: 'Ad Dei laudem, sum sanus!' (God be praised, I am healthy).

Another case of apparent recovery is described by Spaniard **Francisco Arceo** (1493–1573) (Finger, 1994: 372). A worker hit on the head by a stone was immobilised and without speech for several days. Arceo remedied the compression fracture and some days later the patient began to speak again and apparently recovered fully through a claimed *Spontanremission* (without therapeutic intervention).

Probably the first comprehensive presentation of aphasia can be found in the work of **Johannes Schenck** (1530–1598; see Figure 2.4) from Grafenberg, a German physician from the sixteenth century (Luzzatti & Whitaker, 1996). In his book *Observationes medicae de capite humano* (*Medical Observations on the Human Head*), Schenck rejected medieval ventricle theory. Among other reasons he described a case where there were no memory problems despite damage to the fourth ventricle. Schenck described at least 16 cases of speech and language disorders, most often cases of open skull–brain trauma and observed that speech or language were often impaired with brain damage, although the tongue is not paralysed. Schenck was therefore one of the first to grasp the essential fact that aphasia was a **language disorder** as opposed to a speech disorder. Schenck too views the cause of aphasia as a failure of memory. The idea that aphasia was an impairment of memory was expounded in the seventeenth century by **Johannes Jakob Wepfer** (1620–1695), who described at least 13 clear cases of language disorder in his work on head and brain injuries (Luzzatti & Whitaker, 1996) which he described as memory loss. And more clear cases of aphasia were documented in the seventeenth century (Benton & Joynt, 1963).

Figure 2.4 Johannes Schenck (1530–1598), who published one of the first comprehensive case presentations of aphasia.

In 1676, **Johann Schmidt** (1624–1690) described a patient who was paralysed on the right side after a stroke and had aphasia with severe reading problems but good writing:

> he substituted one word for another so that his attendants had difficulty in determining what he wanted [. . .] He could not read written characters, much less combine them in any way. He did not know a single letter nor could he distinguish one from another. But it is remarkable that, if some name were given to him to be written, he could write it readily, spelling it correctly. However, he could not read what he has written even though it was in his own hand.
>
> (Benton & Joynt, 1960: 209)

This is perhaps the earliest description of what we would now call alexia without agraphia. The report is also probably one of the first on treatment, bloodletting, rubbing oils and essences onto head and neck, which, unsurprisingly, did not help the patient. Schmidt was more successful with another case where the patient could 'put together letters and attain a level of perfection in reading after therapy' (Bernard, 1889: 68).

Thomas Willis also described at least two cases of aphasia with difficulties with naming and word identification (Critchley, 1970: 56). **Peter Rommel** (1643–1708) describes a further case with aphasia (Benton & Joynt, 1963). A female patient paralysed on the right side and with global aphasia was reduced to 'yes' and 'no' in spontaneous speech and had a repetition deficit, but could clearly recite the Lord's Prayer and other prayers fluently. However, short phrases from these prayers, given to the patient to repeat, could not be repeated by her and after several attempts she broke into tears. She could read, but without full understanding. Rommel also described his patient in great detail: the clear separation between automatic and non-automatic speech is also interesting, and was to form an important part of Hughlings Jackson's thinking on the functions of language in the nineteenth century (see Chapter 3). But Rommel called the disorder 'aphonia' (without voice), which is not atypical because, from the Hippocratic writings until the nineteenth century, an appropriate terminology for describing aphasic impairments did not exist.

The eighteenth century: Enlightenment, reason and nature

The philosophical movement known as the **Enlightenment**, which had its beginnings in the sixteenth century, was the critical feature of eighteenth-century thinking with the significant influence of such thinkers as René Descartes, Francis Bacon and John Locke. It sought to replace orthodox authoritarian beliefs with a rational approach to scientific inquiry and **reason** and **nature** were the watchwords for eighteenth-century thinking. At this time the term 'nature' had a wider meaning than it does nowadays and included human beings and their mental faculties. Important topics in

eighteenth-century thought were, among others, the order of animals and plants, the position of human beings in relation to animals, the position of humans in nature, the purpose and structure of the human brain, the location of the soul organ, and the relationship between the human races. In the second half of the eighteenth century the 'life sciences' emerged and the applied sciences (including medicine) were strongly supported, and the foundations for the great cataclysms in the nineteenth century were laid. The crucial political events that were closely connected to the Enlightenment were the writing and establishment of the American Constitution which came into force in 1788, and the **French Revolution** which began in 1789, and ended at the conclusion of the eighteenth century with Napoleon Bonaparte's coup d'état. The French Revolution marks the end of the Enlightenment.

The improvement of human living conditions was a further central feature of the time, and health care developed especially strongly. For instance, the general hospital (das Allgemeine Krankenhaus) was founded in Vienna in 1784, a then leading centre of clinical medicine. Practitioners became generally more deeply interested in the causes and the deficits at the root of diseases and mental diseases became a domain of medicine again and no longer a concern of religion (Ackerknecht, 1992: 97ff.).

Medicine in the eighteenth century

For speech and language and the brain, the work of Willis and Descartes still dominated. However, in the course of the eighteenth century the term *spiritus animalis* was replaced by 'nerve fluid', which was often claimed to be located in the ventricles and circulated through the hollow nerves, thus making motor and sensory function possible.

At this time a notion took hold that had sporadically been put forward earlier; namely that the nerves are not ducts, but rather firm fibres. **Isaac Newton** (1642–1727) had already supposed, based on Aristotle, that all human bodies contain a hidden, lightly vibrating 'ether' that, at the command of the will, moves through the nerves from the sensory organs to the brain and from there to the muscles ('vibration theory'). In the seventeenth century, the philosopher **John Locke** (1632–1704) considered the human mind a collecting point for sensory perceptions that are processed into increasingly complex ideas by combining, connecting and associating them with each other. This 'association of ideas' approach of Locke and the vibration theory of Newton were combined in neurophysiology by the English physician and philosopher **David Hartley** (1705–1757) in his attempt to explain memory. **Associationism** considers association and connection to be the fundamental principle underlying mental life. Ideas, sensations, movements, feelings are connected or associated in such a way that they are linked and succeed one another in mental processing. The idea is as old as Aristotle, but Locke is often, incorrectly, regarded as the founder of associationism, mainly on the basis of a short chapter in his *Essay Concerning Human Understanding* (1690) where

he discusses 'the association of ideas'. It was Hartley, and other members of the English School, who were responsible for the idea's early development.

Hartley supposed that every external stimulus creates vibrations that propagate through the sensory nerves until they reach the medula, where repeating stimuli will establish themselves over time. Based on association principles, complex idea systems build themselves through repetition over time. Hartley's **theory of association** was to enjoy long success, although not his vibration theory, and it was also to influence aphasiology in the closing nineteenth century. But Hartley's vibration theory too had successors. For instance, the Swiss philosopher **Charles Bonnet** (1720–1792) believed that every sensory, moral or intellectual ability of human beings (and even every word) had specifically vibrating fibre bundles in the brain.

One of the most influential figures of the eighteenth century was the Swiss **Johann Kaspar Lavater** (1741–1801) (Jaton, 1988; Lotsch, 1970; Weigelt, 1991), the originator of **physiognomy**, although its fundamental assumptions had been formulated since Antiquity. Physiognomy assumes that the characteristics of every human mirror themselves in the face. Lavater's chief work in four volumes has the title *Fragments on Physiognomy, for the Promotion of Knowledge and the Love of Mankind* (1775–1778). In Figure 2.5 we see the physiognomy of four boys, the respective interpretation by Lavater (1970) being

> a noble, open, happy, quiet, sensitive boy the first. The second does not have the lightness, freeness and openness of the first. Forehead and nose: sensible, at equal distance from genius and stupidity. His eye looks and marvels more than it observes and thinks. Coldness and calmness in the mouth. The third is far beneath all three on this table. Mouth and chin are even beneath the vulgarity of the eye, the forehead, and the nose. The fourth: delicate, fine, easily adaptive, clairvoyant, not audacious, not big, even less grand, but will become a useful, diligent, clever merchant clerk or skilled craftsman.
>
> (Lavater 1970: 31)

It is important to note that physiognomy was a European-wide mass movement that was accepted as a paradigm by amateurs and scientists alike without empirical proof. Physiognomy helped to fuel the European-wide discussion on the essence of humans and of human characteristics, a discussion that had tangible political consequences (the slogan of the French Revolution was the equality of all men). In some respects, a straight path leads from Lavater to Franz-Josef Gall, whose work on the development of craniology (or *organology*, and later termed *phrenology*) at the end of the eighteenth to the nineteenth centuries ultimately led to the breakthrough of the development of the notion of localisation (Whitaker, 1998: 33). Lavater also influenced Charles Darwin's (1872) work on *The Expression of the Emotions in Man and Animals*, leading eventually to a genuine science of facial expression.

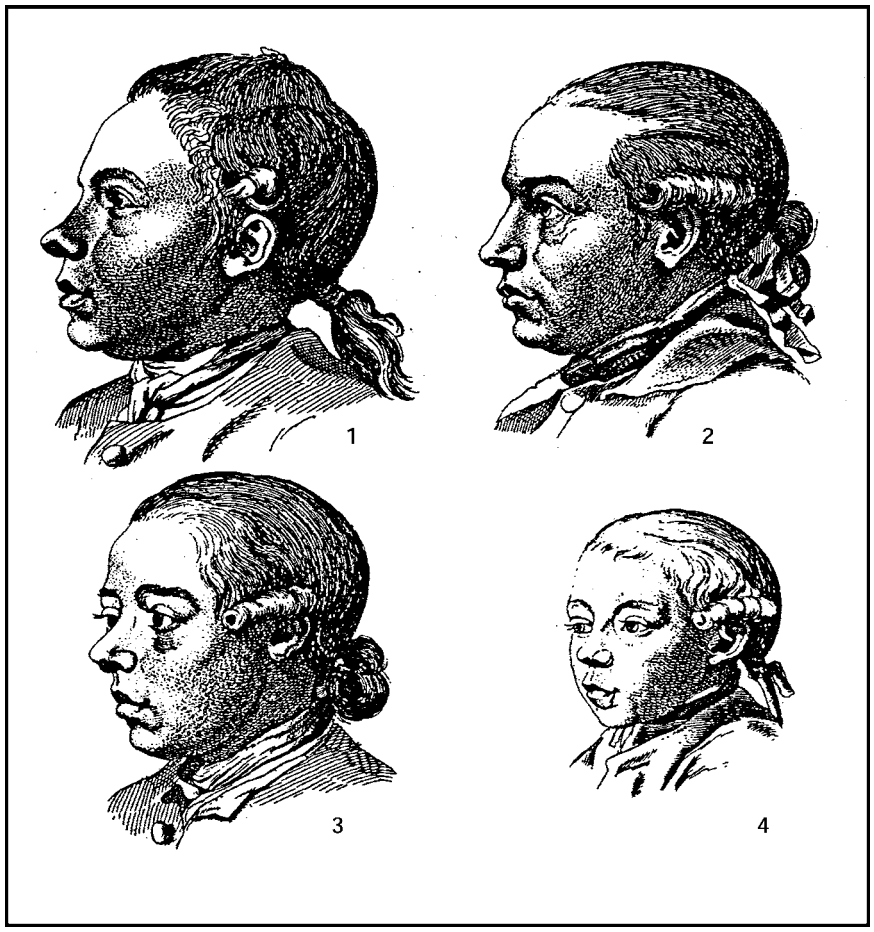


Figure 2.5 The physiognomy of the heads of four boys (see text for interpretation) following J. K. Lavater (1741–1801). Physiognomy was a Europe-wide success and one of the first popular 'scientific' mass movements.

Localisation theory in the nineteenth century was already pre-empted in the middle of the eighteenth century by a relatively unknown (in the history of neuroscience, but not of religion) Swede, **Emanuel Swedenborg** (1688–1772), who was an uncommon man – a mathematician and a mining director first before he turned to medicine and localisation theory. Then he became a theologian and founded the Swedenborgian Church, which still exists as the New Church of Jerusalem (see Schwedenberg, 1960). In his medical works he wrote in 1741 that different functions must be represented in different anatomical locations in the cortex. He offered two reasons: it had to be this way philosophically, so that the functions of the brain will not interfere with

each other and disturb each other in their effectiveness; clinically it was necessary in order to be able to explain the selective deficits of pathological phenomena. Swedenborg also wrote that the frontal lobes are responsible for will and memory, which is not dissimilar from our contemporary assumptions. But Swedenborg had little influence because his writings were not widely known.

Finally, **Franz Anton Mesmer** (1734–1815) must be mentioned as an important figure at this time, whose theory (**Mesmerism**) spread around Europe like Lavater's physiognomy (see Florey, 1995a, 1995b). Mesmer started from the assumption that the *spiritus animalis* flows through the human body and has an invigorating, organising effect. This *spiritus animalis* also has magnetic characteristics (**animal magnetism**), which can themselves be influenced by normal magnetism or by the world-ether that pervades the entire universe. Mesmer hence used a magnet during his treatment, but claimed also to have the ability to concentrate the world-ether in himself and to let it flow out through his hands, similarly to a magnet. Interestingly, his often surprising successes are attributed less to magnetic powers than to strong hypnotic suggestion, which plays an important role in mesmeric seances. This contributed to the development of hypnosis as a therapeutic tool (Ackerknecht, 1992: 150).

Mesmerism met with strong hostility from established orthodox medicine, but nevertheless found high acceptance outside orthodox medicine. This extra-scientific recognition may be best understood as some kind of reaction to the 'mechanical' era, which attributed great value to mathematical natural sciences and to a mechanistic understanding of diseases. This attitude of opposition between technical-scientific progress and less rational countermovements characterises European society and medicine from the eighteenth century.

With the end of the eighteenth century the 'soul organ' disappeared from neurology, and the last mention of soul localisation is found in Soemmering (1796) (Mann & Dumont, 1985). Thus, a further barrier had been overcome on the way to understanding the nature of human mental abilities.

On aphasia

In the eighteenth century, the number of descriptions of aphasic phenomena (and to some extent even of therapy for their remediation) increased significantly and self-descriptions of aphasia were also published. It began to become obvious that there were different kinds of aphasia. Additionally, a first 'theory' of aphasia developed through the work of Gesner at the end of the century, which saw aphasia primarily as a (lexical) memory problem.

The variety of descriptions of aphasic symptoms at this time provides early indications that aphasic people were presenting with different kinds of impairments and that separate features of language could fractionate into impaired and unimpaired.

The Swede **Olof Dalin** (1708–1763) described a patient whose speech was reduced to the word 'yes' but he could produce songs and hymns like a normal speaker (Benton & Joynt, 1960: 211f.). Another Swede, **Carl von Linné** (Carolus Linnaeus, 1707–1778; see Viets, 1943), reported a professor from Uppsala who had lost his 'memory for names', so that he could say neither his own name nor that of his children or his wife. When his communication partners attempted to guess what he wanted to say, he always responded 'Yes', and he always responded to invitations to repeat with 'I cannot do it'. Linné concluded his report as follows: 'he had lost two things: first of all the memory for all nouns, and second of all the ability to express nouns' (Viets, 1943: 329). We would now call these utterances **lexical speech automatisms** (Code, 1982a, discussed in Chapter 9) and 'I cannot do it' is a particularly common modal form (see Nespoulous, Code, Virbel, & Lecours, 1998). The king of Sweden also had aphasia; his 'memory loss' concerned the names of those personally and professionally close to him, with the exception of his state chancellor's name. All others were called 'doctor' (Benton & Joynt, 1960: 212f).

Giambattista Vico (1688–1744) was probably the first to mention a selective disorder of verbs (in contrast to nouns) in a patient who had a stroke (Denes & Barba, 1998). **Giovanni Battista Morgagni** (1682–1771) emphasised, in a comprehensive work, that the damaged anatomy must be examined together with the functional deficits. In addition, he noted that loss of speech often accompanies strokes, and he differentiated three kinds of problems: loss of voice, utterance of meaningless strings of sounds, and speaking problems due to pathological changes of the tongue. Morgagni's descriptions were short and leave a lot of room for interpretation (Benton & Joynt, 1960).

In the eighteenth century we see the emergence of **self-descriptions** of aphasia and interesting ones include de Fouchy, Samuel Johnson (Critchley, 1962), and **Johann Joachim Spalding** (Förstl, 1992). Spalding (1783) reports the following:

> I saw and recognised everything around me in its true shape; only the strange afflux and confusion in the head I was unable to shake off. I tried to speak, also as an exercise to see if something coherent could be uttered; but no matter how much I forced attention and thoughts together, and did this with utmost slowness, I noticed soon that shapeless and entirely different words ensued rather than the ones I wanted; my soul was now as little in command of the inner tools of speech as it had earlier been of writing. I therefore contented myself with the expectation naturally not joyful in itself that, were this state to constantly persist, I would not be able to read or write for the duration of my life, but that the principles and dispositions known by me would always remain [. . .] the same.
>
> (Spalding, 1783: 40)

What is interesting about this early description is that the aphasic impairments left the ability to think and the personality was intact, and that spoken as well as written language can be affected together. These are both observations that fit well with current understanding of aphasia.

It should be briefly mentioned that the German poet **Johann Wolfgang von Goethe** briefly described a character in his novel *Wilhelm Meister* with a hemiparesis on the right side and aphasia – moulded after the aphasia of his own grandfather – so the description is not entirely 'fictional'. What is interesting about this description is that the communicative consequences of a severe aphasia are clearly described for the first time by Goethe from the perspective of the aphasic person's communication partner. The communication partner in the novel does not succeed in getting the information that is crucial to her from the aphasic character. An emphasis on communication partner training is an important aspect of contemporary approaches to rehabilitation and social reintegration.

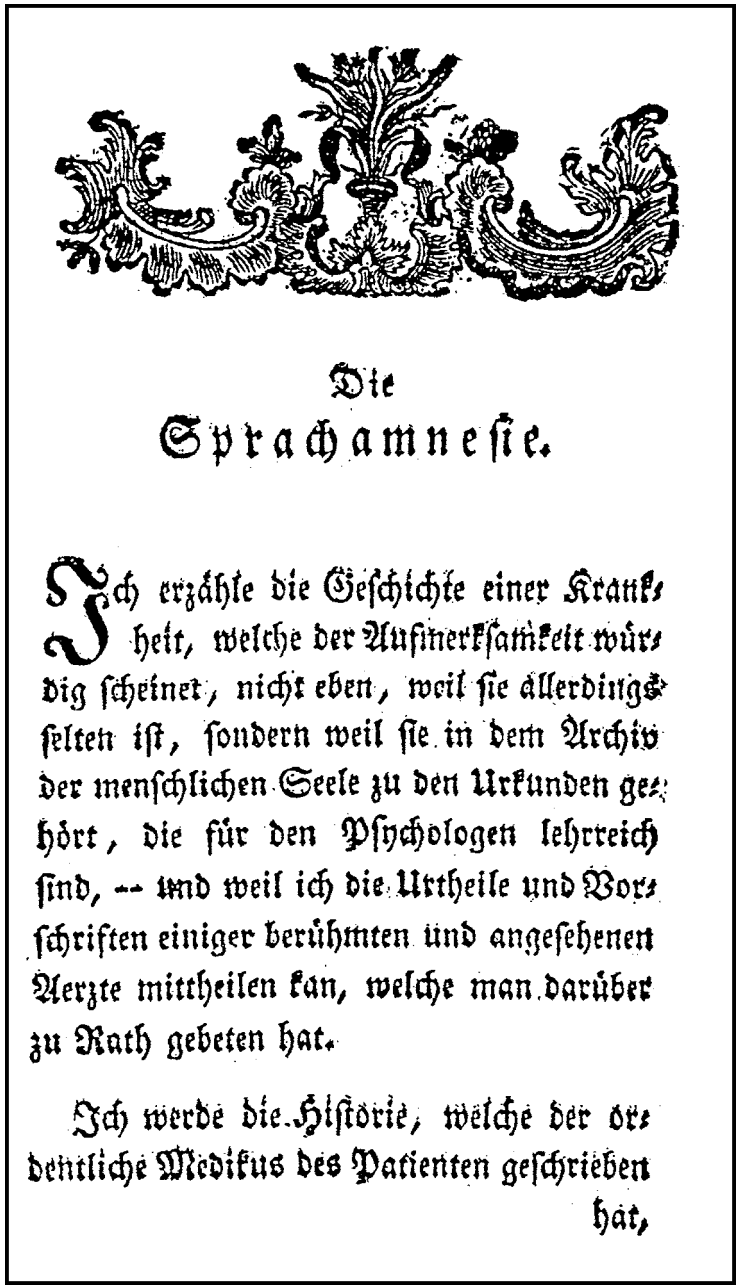
Johann Gesner and the first theory of aphasia

Of central relevance for the history of aphasia (not only in the eighteenth century) is a paper of more than 70 pages on aphasia in a multi-volume work by **Johann Gesner** (1738–1801). Under the telling title 'The Language Amnesia' (see Figure 2.6) a 73-year-old man, KD, was described in detail, and 'language amnesia' was subsequently discussed with reference to five further cases (from the literature and from Gesner's own observations). This was the most detailed description of aphasia thus far in its history. Gesner's conceptualisation of 'language amnesia' is remarkably close to present-day understanding of aphasia (the following quotations are translated from Gesner, 1789).

KD had 'a very particular impairment of language' (p. 111), which manifested itself in the following way: 'for although he spoke with ease and fluently, he expressed everything with quite uncommon and self-made words that no man could understand' (p. 111). KD appeared to speak fluent jargon and uttered many nonsense words, **neologisms**. The neologism 'zettejuset', for example, was 'used by him in such fashion and tempered with other words that his discourse thereby becomes incomprehensible' (p. 148). Automatic counting was also used in fluent speech and the neologisms that permeated his speech were used repeatedly:

> the words that Mister K.D. utters are mostly meaningless, thoughtless sounds. But their total number added up altogether is not too large, in that an incomprehensible word is often repeated one after another, and only once in a while replaced by other, equally incomprehensible ones.
>
> (Gesner, 1789: 146–147)

Stereotypies are stereotyped, automatically and fluently produced formulaic utterances (such as 'most obedient servant', 'good morning', 'I do not



Die
Sprachamneſie.

Ich erzähle die Geſchichte einer Krankheit, welche der Aufmerkſamkeit würdig ſcheinet, nicht eben, weil ſie allerdings ſelten iſt, ſondern weil ſie in dem Archiv der menſchlichen Seele zu den Urkunden gehört, die für den Pſychologen lehrreich ſind, — und weil ich die Urtheile und Vorſchriften einiger berühmten und angeſehenen Aerzte mittheilen kan, welche man darüber zu Rath gebeten hat.

Ich werde die Hiſtorie, welche der ordentliche Medikus des Patienten geſchrieben hat,

Figure 2.6 The first page of the first systematic essay on aphasia by J. A. P. Gesner (1738–1801), taken from his *Samlung von Beobachtungen aus der Arzneygelahrtheit* (*Collection of Observations from Medicine*) (source: Gesner, 1789).

want to', 'yes', 'no', 'oh my God') and were used repeatedly by KD – partly appropriately and partly inappropriately:

> these words are, for the most part, used by him in the correct meaning. But I have also noticed that, when I had drunk to his health, and he wanted to thank me, the word 'adieu' burst out instead of the common formula, that the patient would, against his will, say good evening of a morning, good morning of an evening, and that he therefore seemed to, in quite particular a fashion, have entirely lost control over his tongue.
>
> (Gesner, 1789: 149)

KD also had what appear to be similar problems with written output:

> writing and speaking are equally incorrect. The patient cannot even write his complete name. If he writes something else, which he is rarely persuaded into doing because he knows that he can't, then equally incomprehensible words are put on paper as when he utters them.
>
> (Gesner, 1789: 150)

The descriptions of KD's auditory language comprehension are less clear, but it can be safely assumed that it was also impaired, especially when what was said refers to 'abstract things' or when the conversation partner spoke unclearly. Reading comprehension was also impaired: 'thus he cannot read either, these symbols of ideas do not inspire any images in him, although more tactile objects all bring forth appropriate terms in him' (pp. 150–151). Although KD often read to himself, Gesner doubted that he understood what he was reading, among other things because 'the patient, who otherwise would have found his greatest pleasure in reading, for the most part, often grieves by weeping' (p. 151) when he was reading. KD was apparently aware of his errors: 'the patient also knows that he speaks incomprehensibly and has often laughed and joked about it by repeating some of these words' (p. 154).

Besides a detailed description of the aphasic symptoms, Gesner emphasised that there was no impairment of KD's knowledge of the world and his relationship to it or his social behaviour and discourse:

> the patient knows everyone he knew before, inquires after the situation of the individuals he deals with, respects all courtesy against them as would always have been his way otherwise, following their difference in status, age, gender, etc., he praises, reprimands, despises, derides what would arouse such judgement and emotions with such objects, and with reason.
>
> (Gesner, 1789: 151)

KD's 'normality' and the apparent disassociation of language from other aspects of higher cognitive processing was clearly captured by Gesner: it

> seems that, when one has seen the patient and spoken with him for a long

> time, he misses nothing but language, and I do not know if I cannot make his state best imaginable and understandable by saying: should one ignorant of the German language see and hear the patient speak without knowing of his sickness, then he would consider him quite a healthy, ordinary man and believe that he merely speaks a language unknown to him.
>
> (Gesner 1789: 156)

Gesner (1789: 132) described this 'language amnesia' as follows: 'the illness itself is thus the inability to make his thoughts understandable to others with spoken or written words in any language'. Gesner reasoned that the impairments cannot be due to motor dysfunction. First, because the tongue 'had enough skill to bring forth three times more words than the need would justify' (p. 114), and second, because, '[i]f it were a defect in the tongue, then the sickness would be an inability to speak, but not also to write' (p. 132). With this insight into how separate components of the speech, language and communication systems can be separately impaired, Gesner laid the foundations for a clear separation of general communicative competence, which was unimpaired, and language processing from speech programming.

For Gesner (1789: 156) the cause of the aphasia was **a selective disorder of memory**: 'as individual powers of the soul have been weakened in particular, without damage to the others, the memory especially has thus also suffered a greater or lesser loss with respect to certain classes of ideas.' This selective loss of memory for words was explained as follows: sensory perceptions and concepts (recognition of an object) are produced by means of **ideation** by the sensory nerves (in the *sensus communis*); the retrieval of words from memory follows ideation. Speaking and writing are functions of memory, while reading and understanding are functions of ideation:

> The dominance of the will over the imagination is the memory. Speaking and writing are performances of the same, reading and hearing are rather performances of imagination. In our case, more than the memory has thus suffered a loss which, following concurrent information, however, does especially only affect language.
>
> (Gesner, 1789: 162)

For Gesner, the concept of selective impairment to components of mental processing was quite clear. Brain damage can leave ideation intact, but impair lexical memory. According to Benton (1965) his was the first **associationist aphasia theory** to emerge, a precursor to the dominating associationist theories of the nineteenth century.

The organic cause of KD's language amnesia was seen in terms of a 'congestion' of the 'nerve ducts', and as outside the brain by Gesner. This led to a sluggishness of interactions in the brain and the cause, and consequently therapy, could be specified:

> the reason [. . .] seems to [. . .] lie in the fact that the residual viscous matter from the not yet abated catarrh has moved to the abdomen, has attacked the big nerve tangles located there precisely, wherefrom the stimulus has been transmitted towards the head. Therefore, one must particularly see to dissolving this viscous matter, expel it from the body little by little, and bring back into their correct order the nerves brought into disarray, and thereby also the performances that depend on them.
>
> (Gesner, 1789: 120–121)

Subsequently, the proposed underlying ailment was treated with bloodletting, foot baths, purging, enema, poultices, and tea augmented with, for example, tartar and saltpetre, 'in order to thereby lessen the impulsion towards the head and to dilute the viscous juices at the same time' (p. 123). The treatment was not successful.

Alexander Crichton (1763–1856), writing a few years later in Edinburgh, and with reference to Gesner's work, also characterised the cases of what he called **semantic paraphasia** observed by him (an example of 'semantic' paraphasia given by Crichton is *boots* for *bread*, not what we would call a semantic paraphasia these days as the words have no apparent semantic relationship), as a 'defect of the principle by which ideas and their corresponding expressions are associated, than as [a defect] of the memory' (Benton & Joynt, 1960: 217). Gesner had little impact on the scientific discussion of aphasia in his time, or since, apart from on Crichton, which seems remarkable given that his conceptualisation of aphasia seems quite modern from a present-day perspective.

Summary

From the Renaissance to the Enlightenment until the end of the eighteenth century, the foundations were laid for the great advances in medicine, neurology and aphasia of the nineteenth century. The brain was explored in more anatomical detail, the natural sciences received a strong boost, political and philosophical developments favoured the sciences and medicine. In neurology, the quest for methods for the localisation of function was gaining pace, and was to come to dramatic fruition.

More detailed descriptions of aphasia and aphasic symptoms appeared, and a first associationist theory of aphasia (without a clear anatomical basis) was developed by Gesner. Aphasia was still seen by most as an impairment of memory ('language amnesia', in Gesner's terms) but recognised by Gesner as a pure language processing disorder as opposed to a speech or communication disorder

3 The nineteenth century until 1880

The birth of a science

The nineteenth century is considered to be the most important period in the history of aphasia, mainly because hypotheses about the nature of aphasia and the localisation of language and speech emerged, which were to form the basis for the later investigations of Broca and others. It is probably fair to say that there was no real aphasiology, in the sense of a theoretical basis, until Gesner's work, and it was not really until the nineteenth century that the seriously systematic study of aphasia began.

Napoleon's reign in France dominated the beginning of the nineteenth century in Europe. At that time the scientific climate was notably more liberal in France than in the rest of Europe. It was not without reason then that the Austrian Franz Josef Gall (1764–1828), among others, moved from reactionary Vienna to a more open Paris, where language localisation, dominance theory and the beginnings of modern aphasiology would later flourish. After the confusion of the Napoleonic Wars, Napoleon suffered major losses in the Battle of the Nations at Leipzig. In 1815, Napoleon was ultimately defeated by a combined European army under Wellington at Waterloo, in 1815. Also in that year, a new national reorganisation of Europe was established at the Congress of Vienna.

Around the mid-nineteenth century colonial imperialism became the dominating political feature (e.g., 'the race for Africa') and scientific endeavours were also in progress to determine for example, and depending on political orientation, the inferiority or equality of black people in comparison to white ones. Anthropology played an essential role in this enterprise, and it was not a coincidence that questions of language localisation were under discussion in the Anthropological Society in Paris in the 1860s. It was at these meetings that the foundations were laid for the emergence of the classic doctrine in aphasiology in the middle of the nineteenth century. The main protagonists were Gall, Bouillaud, Auburtin, and Broca.

Gall's organology and its consequences

Franz Josef Gall (1764–1828; see Figure 3.1) and his work enjoy a central position in the history of aphasia, neuroanatomy and neuropsychology

Figure 3.1 Franz Josef Gall (1764–1828), the founder of the theory of language localisation and organology, which represented a turning point in aphasiology.

(e.g., in Benson & Ardila, 1996; Whitaker, 1998: 32f.). With him, the foundations of cerebral **localisation theory** began as a serious idea. As detailed earlier, before Gall there were various attempts at localising different mental faculties in various body parts, including parts of the brain, but no earlier attempt identified the central significance of the neocortex for human mental abilities and no earlier attempt had processed as great a wealth of empirical data from the most varied sources as Gall did. Within Gall's organology or craniology (**cranioscopy** was the skull measuring aspect of craniology) emerged also the foundation for the neuropathological basis of aphasias.

Phrenology (the term originated with his student Spurzheim – Gall's original terms are organology and craniology), which emerged from Gall's theories, had a profound impact on the development of evolutionary thinking. In some ways Gall was a forerunner of evolutionary thinking, and it was probably not a coincidence that Gall called man 'the most perfect animal' (Lesky, 1979: 86).

Franz Josef Gall and the idea of 'the language centre'

Gall first studied medicine in Strasbourg and then in Vienna, where he subsequently worked as a neuroanatomist and general practitioner. He also developed his theory of organology while still in Vienna. In 1801 Gall was forbidden by a decree of the church and the Holy Roman Emperor, Franz II, to spread his theory on grounds that it was 'materialistic', and politically highly dangerous. In 1805 he left Vienna on a two-year lecture tour across Europe, which earned him great attention, but also some hostility from a number of directions. The medical establishment was jealous and unhappy with scientific lectures where anyone who paid for a ticket could attend, and took exception to Gall's attacks on well known medical practitioners (e.g., Soemmering). However, most if not all money earned by Gall's tour went into funding his publications, which, because they included illustrative plates, were very expensive to produce. In 1807 he settled in Paris, where he died of a stroke in 1828 as a naturalised Frenchman.

The basic positions of Gall's organology were established in 1798. The essential tenet was that **mental faculties** (or 'organs' in his terminology) **are localised in specific parts of the brain**. In 1798 Gall wrote:

> The aim of all my investigations was to found a theory on the functions of the brain. [. . .] The possibility of a theory of the psychological and mental functions of the brain presupposes: [. . .] that the brain was the organ of all tendencies, all emotions and all faculties [. . . and] that the brain was composed of as many individual organs as there are tendencies, emotions, faculties, which essentially differ from one another.
>
> (reprint in Lesky, 1979: 73)

With this Gall established the foundations of localisation theory, the most influential theory that was to drive neuropsychology and cognitive neuroscience to the present day. Temkin (1947: 275) calls Gall the 'godfather of the principle of cortical localisation of mental faculties'.

Furthermore, Gall assumed that different parts of the brain are different sizes, depending on the level of development of the faculties associated with those parts. The better developed a faculty was, the larger the respective brain part would be. Gall was an expert anatomist, and was more than familiar with the indents found on the inner skull, which developed naturally to accommodate the outer shapes of the surface of the brain. This led Gall to his idea of cranioscopy: as the shape of the cranium adapts to the development of the brain during growth, the individual would consequently have a cranial shape and size that corresponded more or less to the parts of the brain located beneath:

> the form of the inner of the cranium is determined by the external form of the brain: consequently certain abilities and propensities can be

> concluded from the form of the outer surface of the cranium as long as it agrees with the inner form or does not diverge from the known deviations.
>
> (Gall, 1798, in Eling, 1994a: 22)

It would therefore be possible to detect the strength and development of particular human 'faculties' from the shape and size of the cranium.

Phrenology became the highly popular science of its time, with many practitioners emerging throughout the century in Europe and America. Phrenology became smart and fashionable, and the rich and famous would hire practitioners, many of whom were charlatans, to entertain their guests in their salons and drawing rooms. It is this part of Gall's craniology that was used to discredit his general theory, but it was not the crucial aspect that inspired the imaginations and investigations of subsequent scientists.

Gall localised altogether 27 organs or faculties of which humans incidentally have 19 in common with animals. The specifically human organs are localised mostly in the neo-cortex. Gall also postulated two faculties concerned with language: the faculty of words (*Wortsinn*) and the faculty of language (*Sprachsinn*), although he stated that the faculty of words was subordinate to the faculty for language.

According to Gall, the **faculty of language** is innate, is independent and autonomous of reason and intelligence, and its primary purpose was as a means of expression and communication. This separation and autonomy of faculties was an attribute which later formed part of the basis for the notion of 'modularity' (Fodor, 1983; Marshall, 1984), a significant feature of **cognitive neuropsychology** that was to develop in the 1980s (see Chapter 8). The faculty of language was, in other words, the 'ability to find the correct symbols and their correct combination to communicate what is thought' (Oehler-Klein, 1990: 140). The **faculty for words** (the lexical memory or **lexicon** in modern terminology) contains the words that the faculty of language can utilise. Although the faculty for words was described separately, he considered it was 'only a part of the faculty of language' (Gall in Lesky, 1979: 154). In later works, the faculty of language was often the only one mentioned. The faculties of language and for words are consequently localised in neighbouring brain regions (Figure 3.2).

The language organ was located in the 'brain section that rests on the rear half of the orbital roof' (Gall in Lesky, 1979: 154). This 'localisation' of the language organ was apparently based on an observation made by Gall in childhood. Gall had a fellow pupil in school who was very gifted in languages and could learn verbal material by heart especially well. The boy also has strongly protruding eyes, suggesting to Gall that the boy's brain was particularly well developed behind the eyes causing the protrusion. Hence protruding eyes signify a large and well developed organ for language. Thus, a 'language centre' was anatomically identified for the first time.

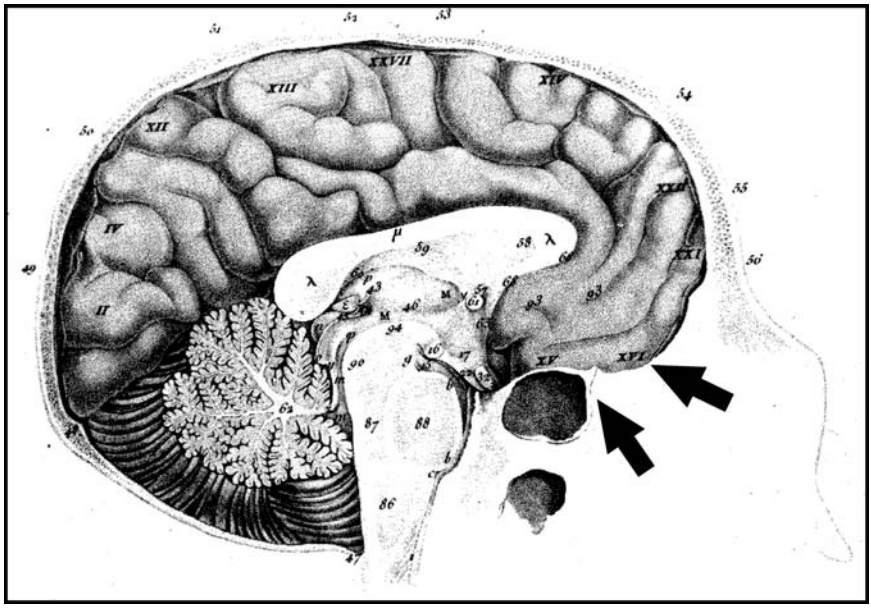


Figure 3.2 Brain picture from Gall's anatomical atlas (1810). Areas XV and XVI show Gall's localisation of the *faculty for words* and the *faculty of language* in the orbito-ventral region of the frontal lobes.

Gall supported his localisation claims with different arguments. His collections of human skulls are well known (one collection is housed in the Museé de l'Homme in Paris, including Gall's own skull, No. 19216, and the Rollett Museum, in Baden, Vienna). The skulls have particular behavioural or personality characteristics identified with them which have especially distinctive cranial bulges caused by the supposedly well-developed brain matter beneath. A further strand of reasoning was based on selective brain damage: 'In diseases and wounds of certain parts of the brain, certain qualities are deranged, irritated, or suspended' (Gall in Lesky, 1979: 51). Gall reported on selective impairments of the faculty for words and of the faculty of language in at least six aphasic patients (Gall, 1822–1825, vol. V: 16ff.; see also Williams, 1898: 193ff.).

In comments on disorders of lexical memory, Gall described an officer with a lesion to the frontal brain directly over the eyes who could no longer say the names of his friends. Another man had a fencing lesion, again above the eyebrows, and a loss of lexical memory was surmised because he no longer knew any names. Quite well known was the case of one Edouard de Rampan, whose ability to name things was impaired by a foil injury in the left prefrontal brain area. The patient could recognise and classify objects with ease, and he also showed signs of depression when reminded of the military.

Gall paid no attention to the fact that the lesion affected the *left* hemi-

sphere – a special role for the left hemisphere in language and speech did not become apparent until the middle of the century. For Gall, the localisation to the orbital region of the frontal brain took centre stage.

A disorder of the faculty of language was illustrated by the following patient:

> It was also following an apoplectic seizure that this man **sees himself incapable of expressing his feelings and thoughts by means of spoken language**. His face displays **no trace of mental impairment**. His spirit finds the answer to the questions addressed to him: he does everything he is asked to do. I showed him an armchair and asked him if he knew what it was; he answered by sitting down in it. He was incapable of articulating spontaneously a word that he is asked to repeat; but some moments later, this word unwittingly escapes his lips. In his embarrassment, he indicates the lower part of his forehead with his finger, displays impatience and, through gestures, he shows that his inability to speak was coming from there. **It is not his tongue that causes the embarrassment**, because he moves it with great agility, and he pronounces a great number of words very well when they are isolated. **His memory is not at fault either**, because he showed me very vividly that he was annoyed that he could not express himself on many things that he would have liked to tell me. **The only thing he has lost is the faculty to speak [‘la faculté de parler’]**. This soldier [. . .] could also neither write nor read.
>
> (Gall, 1822–1825, Vol. V: 37–38)

With such descriptions Gall characterised aphasia (for which he had no word yet); this characterisation would dominate until the twentieth century, and subsequently would become enshrined in the **classic model** with the work of Broca and Wernicke. Language processing takes places in specific language centres in the brain and if these places are damaged then functional language deficits will result. The language disorder should be examined separately: it was not a dysarthria, nor was it a memory disorder, and there was no reduction in intelligence. Likewise, it was clear that there were different forms of language disorder and that the ability to *communicate* without formal language could be well preserved. With regard to the eventual framework of the classic model developed later in the nineteenth century, all that was really missing was that the causative lesion should be left-hemispheric, and that language comprehension could also be impaired.

The phrenological movement: case descriptions

Gall developed his theory first alone and later in collaboration with his former assistant **Johann Caspar Spurzheim** (1772–1832), but they developed different views on the theory and separated as a consequence in 1813. Gall stayed in Paris and Spurzheim went to Edinburgh where he set up a flourishing phrenological society with the Combe brothers (Kaufman, 1998).

Consequently, **phrenology** (a term rejected by Gall) became a highly popular movement in England and Scotland and also took hold in North America, influencing literature and art, as well as science (Cooter, 1976; Davies, 1971; Temkin, 1947).

Spurzheim continued to develop phrenology and began to localise more and more faculties (altogether 37), so that the system departed further from Gall's original anatomical basis. Figure 3.3 shows a version of human faculties following Spurzheim, where the faculty of language and that for words become a united language faculty, localised beneath the eyes. Gall's assumptions are often dismissed as charlatanry, nonsense, and as being unscientific. The

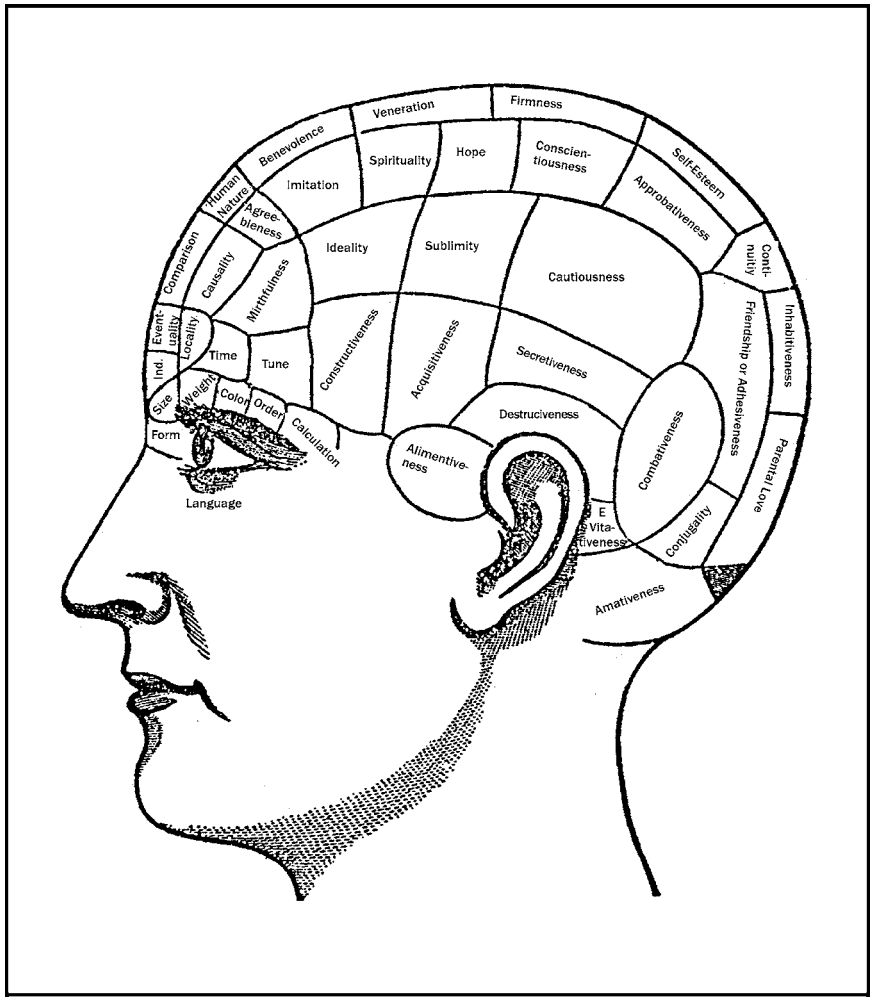


Figure 3.3 The 37 faculties (the phrenological organs) following Johann Kaspar Spurzheim.

cause for this not untypical assessment arises partly from Spurzheim's extensions and those of the Edinburgh based phrenologists **George** (1788–1858) and **Andrew Combe** (1797–1847), leading to the ascientific developments of phrenology during the course of the nineteenth century (Kaufman, 1998).

To jump forward in time to the second half of the nineteenth century for a moment, the phrenology of the Victorian era resulted in increasingly bizarre developments including, for instance, the 'automatic phrenometer', a device that measures the bumps on the skull, allowing the examiner to automatically determine the features of the character of the examined person. Equally problematic are the anthropometric views of **Cesare Lombroso** (1887) in Italy and Darwin's cousin the pioneering psychologist **Francis Galton** in England, that arose as a result of this extreme form of phrenology. Their confidence in the promise of their scientific methods was that it was possible to recognise criminals and criminal tendencies by means of measuring skull and face proportions. It is interesting to reflect that there still remain phrenological societies (Cooper & Cooper, 1983). But one should differentiate between the indisputable anatomical and scientific achievements of Gall and the ill-founded ideas of Spurzheim and other phrenologists (Clarke & Jacyna, 1987). Whatever the final assessment of Gall and Spurzheim may be (Kaufman, 1998; Kaufman & Basden, 1996; Marshall & Gurd, 1996), the phrenological movement gave rise to an animated discussion that also produced a range of important case reports on aphasia and ultimately led to the first language localisation debate in France.

In the phrenological case reports on aphasia (overview in Williams, 1898: 220ff.; see also Finger, 1994: 375), the following aphasic symptoms are described, among others: lost capacity of expression but retained acuteness of mind, semantic malapropisms but preserved articulation, speech automatisms, jargon and syntactic aberrances (partly with error awareness), anomia (naming disorders, word identification disorders). It was emphasised in all these reports that the patients do not have cognitive-mental impairments, and that the cause clearly was damage to the language organ, located in the frontal region of the brain. Localising lesions was partly based on the external injuries, but also confirmed through autopsies. For instance, the report by Dr Otto from Copenhagen, contains the following:

> When the head was opened, one of the hemispheres of the brain was found entirely diseased, and the other in a perfectly sound state, with the exception only of that part of which is held by phrenologists to be the organ of language.
>
> (Williams, 1898: 220)

Recurring indications in the case descriptions that the aphasic problems are caused by underlying *left* hemisphere damage was apparently not considered worthy of comment.

A case study from that time (but without reference to phrenology) by

Henry Dickson (1798–1872) was noteworthy because it was the first known presentation of an aphasic comprehension problem that goes beyond the presentation by Gesner. Dickson (1831) writes that the patient

> fancies, when he does not comprehend what is said to him, that he does not hear it, and complains much of this supposed defect. But that his hearing is perfect is proved by a thousand instances of acuteness in this particular. He hears a distant sound, the ringing of a bell, the striking, and even the ticking of a clock.
>
> (Dickson, 1831: 360)

The cases described by **Alexander Hood**, working in Edinburgh, occupy a special position among phrenological case descriptions (Whitaker, 1998: 34–37; Williams, 1898: 228ff.). The case of gardener Adam M'Conochie is often highlighted because it was apparently, and coincidentally, presented on the day of Broca's birth (24 July 1824). More importantly, though less romantically, in a comparison of different patients, Hood comes to the conclusion that the organ of language has three parts: the part that controls the organs of articulation and the parts already known from Gall – the faculty of language, which controls verbal expression, and lexical memory.

The first classic debate(s) on language localisation: Bouillaud and Auburtin vs. Flourens

In the last years of his life Gall had to witness the relative decline of his theory, but one aspect of his views retained fairly high scientific esteem, namely that of his localisation of language in the orbital region of the frontal brain. One follower of Gall in Paris was **Jean-Baptiste Bouillaud** (1796–1881; see Figure 3.4), who was a founding member of the French *Société Phrénologique* (Breidbach, 1997: 118ff.; Clarke & Jacyna, 1987: 262ff.; Finger, 1994: 375f.; Hagner, 1997: 232ff.; Lian, 1947; Stookey, 1963). Nevertheless, while he took a critical stance against phrenology as a whole, he was in favour of Gall's language localisation theory. In a book from 1825 with the revealing, if lengthy, title, *Clinical Examinations which Show that the Loss of Language Correlates with Lesions of the Anterior Lobe of the Cortex, and Confirm the Views of Mr. Gall on the Location of Articulated Language*, the connection between loss of language and frontal brain damage was made with a presentation of 15 case descriptions. But Bouillaud was more interested in the topic of motor function, and some of the presented cases seem to have what we would now call an **apraxia of speech**, because he also emphasised that 'the tongue' was not impaired in any case. Unfortunately, the description and presentation of the symptoms as well as of the location of the lesions was relatively vague (Finger, 1994: 37).

Bouillaud finally divided the disorders he found into two basic types: articulation disorders and language disorders. For him, language disorders

Figure 3.4 Jean-Baptiste Bouillaud (1796–1881), who initiated the first language localisation debate in France on the basis of Gall's theory.

were due to a memory problem, which presents in two different forms. In one words are no longer correctly organised or retrievable or usable (in the sense of Gall's faculty of language) and in the other, the memory form, the individual word itself was damaged. This parallels the current distinction between impairments of storage and retrieval. Bouillaud proposed two possible causes that could lead to the loss of language: first, a destruction of the memory for words, and second, a destruction of the neural foundations for controlling the organs of speech, which could be an apraxic speaking disorder.

Bouillaud published further studies up until the 1840s, describing more than 500 cases. He wanted to prove that speech and language are localised in the frontal brain. He was so convinced of his view that he famously offered a reward of 500 Francs if someone could show him a speech/language-disordered patient who did not display any lesion in the frontal brain (see Breidbach, 1997: 120; Buckingham, 1986).

Despite diverse publications (Bouillaud, 1839–1840, 1848) that kept the topic of language localisation under discussion (Nasse, 1851: 1f.; Schiller,

1992: 171ff.), Bouillaud was unable to really assert his views within the scientific community in Paris until the beginning of the 1860s (Hagner, 1997: 234ff.). He failed to make progress at first because scientists at this time distanced themselves from phrenology and the more generally accepted principle was the opposite position that functions are not localised, but **holistically** represented throughout the brain (Bogen, 1969).

The prominent and dominating representative of **holism** at this time (with respect to the brain) was **Pierre Flourens** (1794–1867) (Breidbach, 1997: 91ff; Finger, 1994: 35f.; Hagner, 1997: 114ff., 229ff., 248ff.; Olmsted, 1953). He was an experimentally orientated scientist and because he was able to replicate the results of his experiments producing the same or similar results, his conclusions were convincing. Flourens carried out many animal experiments (especially with birds and mammals). From a modern perspective his ablations studies of the cortex or parts of it were primitive (Riese & Hoff, 1951). He used 'spoons' for ablations and often took out huge parts of the brain so that the behavioural losses following ablations were often similar. In Flourens (1824), he summed up his experiments on the central nervous system of birds, dogs, cats, mice and moles using stimulation and ablation. In stimulation, the partially exposed animal brain was pinched or scratched and the reaction was recorded. He observed, for example, that irritation of the lower brainstem produced a muscular reaction, but that irritation of the cortex produced no reaction at all. In ablations, Flourens peeled off an animal's cortex or part of it and recorded reactions in behaviour. On the basis of his results Flourens came to the conclusion that the cortex cannot be divided into different functional regions but that functions are represented throughout the brain, a principle called cortical **equipotentiality**. But Flourens certainly admitted certain regional functional specialisations in the central nervous system, in so far 'as the cerebrum is the seat of intelligence, volition, perception, and instincts, the medula oblongata the seat of involuntarily triggered movement, the cerebellum finally the seat of coordination of body movements' (Hagner, 1997: 115; see also Clarke & Jacyna, 1987: 212f.). These general conclusions do not seem entirely out of place in modern times.

Returning to language localisation, **Gabriel Andral** (1797–1846) in the 1840s, in contrast to Bouillaud, presented a different interpretation of some cases of his own. He described patients with frontal brain lesions confirmed by autopsy but without speech disorders and drew the conclusion that 'loss of speech is not a necessary result of a lesion in the anterior lobe and furthermore it can occur in cases in which anatomical investigations show no changes in these lobes' (Gabriel Andral, 1840: 368, in translation by Finger, 1994: 376).

From Flourens' first publications in the 1820s until the 1870s, equipotentiality was the dominating paradigm of brain physiology (Clarke & Jacyna, 1987: 213) until the theory of functional localisation finally became established. However, the dispute between the localisers and the equipotentialists was not merely restricted to the question of functional localisation in the

brain. To begin with, it was a question of what was the right methodology: the clinical observation and case studies of the localisers, or the repeatable experiment (e.g., animal brain ablations experiments), the approach of Flourens' followers. Furthermore, argument centred on whether anatomy and pathology or physiology provided the most valid and reliable information. In addition, the two groups took different basic political and philosophical positions that influenced the neurological debate, Following the revolution of 1848 in France instigated by Napoleon III, the localisationists took the role of progressive liberals and the equipotentialists the role of conservatives.

In summary, a lively discussion on language localisation was taking place in France in the 1840s and 1850s. Some commotion was also created by the lectures of the French neurologist **Jaques Lordat** (1773–1870) on 'alalia', 'paralalia' and 'verbal amnesia', and in these lectures he described different forms of aphasia (Bay, 1969; Finger, 1994: 376; Lecours, 1993: 472ff; Riese, 1977: 15–18). It was especially noteworthy that he added a description of the aphasia that he experienced himself, where he emphasised several times that he had experienced no problem with thinking. Critchley (1970: 291) strongly doubts the value of this reminiscence (Lordat had had his aphasia 20 years earlier): 'Hindsight does not necessarily signify insight'; but for others (e.g., Goodglass, 1993: 17), Lordat's description was decades ahead of its time. The best source for the interested reader is Lordat (1843) himself.

The political developments in the 1850s alluded to above resulted in an expanded freedom of expression and assembly, which led to the foundation of many societies in France in which discussions on a variety of topics were conducted more openly and more liberally. Among the new foundations was the society for anthropology (*Société d'anthropologie*), one of its founders being Pierre Paul Broca. In this society, diverse topics of craniology and racial theory were hotly discussed, for instance the question of the connection between brain size and intelligence. The 'progressive' topic of the localisation of higher brain functions was also very much on the agenda of the society and another particularly significant 'activist' in the localisation discussion was Bouillaud's son-in-law, **Ernest Auburtin** (1825–1893), who was a follower of his father-in-law's views in general and of language localisation particularly. In the discussions of the society, the influential **Pierre Gratiolet** (1815–1865), who was a follower of Flourens (Stookey, 1954), represented the position of equipotentiality and Auburtin argued (with reference to Gall and especially Bouillaud) in favour of localisation of higher mental abilities, particularly language localisation, for which there already was ample empirical evidence.

The Paris language localisation debate (1861–1866)

It was with this background that modern aphasiology is traditionally considered to have begun in 1861. Benson and Ardila (1996: 13) call it the first epoch of the history of aphasia, Finger (1994: 377) speaks of the 'revolution

of 1861', and for Goodglass (1993: 18), the works of Broca are a 'turning point in the history of aphasia'. Caplan (1987: 43) even writes: 'The first scientific studies of patients with acquired disorders of language were presented in the last half of the nineteenth century. They began with an address by Paul Broca before the Anthropological Society in Paris in 1861.' This may be a little exaggerated as Broca's works were as (un)scientific as, for example, Gall's, Bouillaud's, and Auburtin's, and Broca directly referred to a debate on language localisation that had, after all, existed for a good while already.

As we will see, the idea that this single event was the 'beginning of aphasiology' in 1861 is a simplification of a more complex series of events.

Paul Broca: the breakthrough

Pierre Paul Broca (1824–1880; see Figure 3.5) was a versatile scientist (Bendiner, 1986; Castaigne, 1980; Eling, 1994b; Schiller, 1992; but see also Gould, 1996: 105–141). His over 500 scientific works range over anatomy, physiology, surgery, neurology, and anthropology (bibliography in Schiller,

Figure 3.5 Pierre Paul Broca (1824–1880), with whom aphasiology 'officially' began in 1861.

1992: 305–332). He saw himself as an anthropologist and surgeon, but his heart lay in anthropology. As already mentioned, he was co-founder of the Anthropological Society of Paris and of the *Bulletins de la Société Anthropologique de Paris*, which were both to play an important role in the language localisation debate. It was more by coincidence than design that Broca entered into the debate in 1861 that established him as the 'founder' of modern aphasiology. However, he contributed only between the years from 1861 to 1866, and the issue of aphasia was never his most central concern: it was localisation as a principle that drove him. Neither did Broca ever claim any special place in aphasiology. He emphasised several times in his writing that he responded only to the ideas of others, mainly Bouillaud's.

As already mentioned, this significant debate in 1861 was started by **Auburtin** on 4 April (Schiller, 1992: 177). He presented a patient named Bache, who had lost his speech but was said to understand everything and to be of sound mind (Stookey, 1954: 571). However, the patient was already very sick and his demise was imminent. Auburtin announced that he would publicly revoke his views on localisation if Bache's brain (or that of any other speech/language-disordered patient) displayed *no* frontal brain damage in a postmortem autopsy. It was this public announcement by Aubertin that triggered Broca's interest.

Things began to move swiftly. By coincidence, on 12 April a patient named Leborgne was transferred to the clinic of Bicêtre, where Broca was working at that time. Leborgne displayed symptoms similar to those of the case described earlier by Auburtin, Leborgne's chances of survival were also slight, and Broca invited Auburtin to examine the case together with him. Broca probably called in Auburtin because he had little or no experience with patients with speech or language impairments, whereas Auburtin was a recognised expert. A few days later, on 17 April, Leborgne died and his case was presented in the session of the Anthropological Society on the next day, 18 April, in an attempt to resolve the question raised by Auburtin. The case of Leborgne was briefly summarised by Broca (1861a, 1861b). The 51-year-old man had had epilepsy since his youth, the loss of speech had set in 21 years earlier, paralysis of the right arm was evident for 10 years and also of the leg for 4 years, and they increased in the intervening years. Leborgne's spoken output was reduced to the repeated nonsense syllable '*tan, tan*' and some expletives. Broca (1861b) states, without writing the profane words, that Leborgne occasionally uttered '*Sacre nom de Dieu*'. That Auburtin described a case just one week before Broca's case at a meeting of the same French Anthropological Society, with the same automatism, is interesting, to say the least (Lebrun, 1986). Some of those who knew him called Leborgne 'Monsieur Tan'. His comprehension seemed to be intact (although it is unknown whether Leborgne's comprehension was actually examined in any detail), and he could apparently make himself understood relatively well with gestures. Broca called Leborgne's problem ***aphemie*** **(aphemia)**, from the Greek meaning loss of articulated speech. Broca (1861b: 332) stated: 'I will

give to it therefore the name of "*aphémie*" (from the Greek . . . I speak, I pronounce).' Broca was probably surprised to find a disturbance in 'motor' speech with a cortical lesion. Buckingham (1986) points out that everyone knew, including Broca, that the highest known direct 'motor centre' was thought to be the corpus striatum.

Broca determined that Leborgne's lesion was in the left frontal lobe, which was heavily degenerated overall, but the centre of the lesion was the second and third frontal convolution (see Figure 3.6). But as Leborgne obviously had progressive brain degeneration, Broca developed a kind of staged model, where his aphemia was attributed to damage to the third frontal convolution. Broca (1861a: 238) concluded (very similarly to Gall), that aphemia occurred without intellectual deterioration or paralyses, aphemia was the consequence of anterior damage, and that Bouillaud's views on the location of the faculty of articulated speech to the frontal lobe were confirmed.

Remarkably, the side of lesion (left) played no part in the assessment or Broca's interpretation of the autopsy. The significance of the case is that Broca described a speech disorder and not a language disorder, and provided some anatomical evidence for his conclusion that the control of articulate speech is localised to the inferior frontal cortex. The description of the symptoms was relatively scanty, and the cause of the problem was degenerative (see Castaigne, 1980; Castaigne Lhermitte, Signoret, & Abelanet, 1980; Signoret et al., 1984). This said, it might seem that the case description of Leborgne was a somewhat dubious starting point for the 'birth' of modern aphasiology. Commenting on Leborgne's autopsy, Brain (1961: 145) notes 'how very slender today seems the anatomical basis for Broca's idea'.

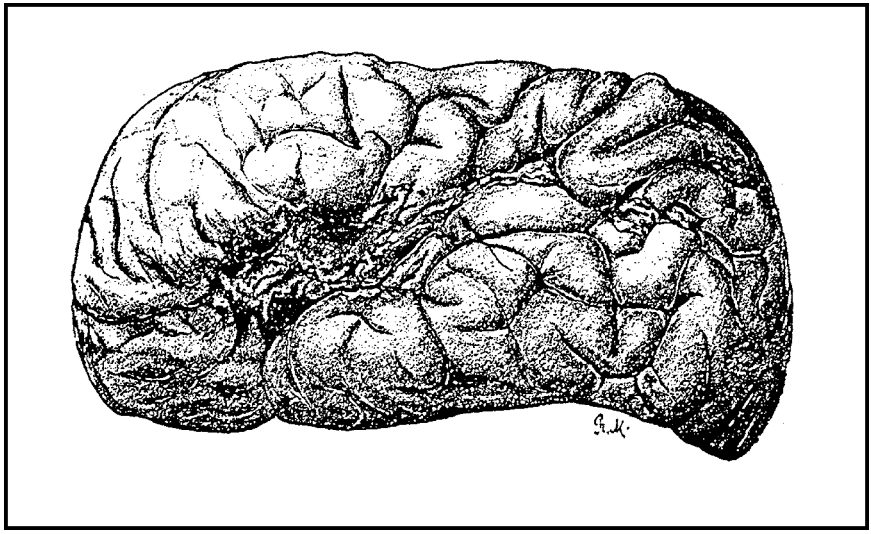


Figure 3.6 The brain of Leborgne (Monsieur Tan Tan), the famous case presented by Broca in 1861.

Yet Leborgne is one of the most discussed cases in neuropsychology and neurology.

Broca (1861c) presented a second case a few months later, Lelong, an 84-year-old working man who had had speech and language problems for about a year and a half as a result of a stroke. His speech was limited to only a few words (*qui, non, trios, toujour*), but he could answer simple questions with his few words and could use gesture. Broca concluded from this that intelligence as well as comprehension were unimpaired.

Aphemia, a disorder of articulated speech, was separated from the 'general faculty of language',

> which precedes all forms of expression of thoughts, and which can be defined as the ability to make a firm connection between an idea and a symbol, whether this symbol was a sound, a gesture, an illustration or any other expression.
>
> (Broca, 1861b: 331)

The general faculty of language would approximately correspond to a general notion of a symbolising faculty that processes symbols irrespective of modality.

At autopsy Broca noticed that the lesion was relatively circumscribed and the same inferior frontal gyri were damaged in Lelong's case as in Leborgne's, namely the second and third frontal gyri, although the third one was more severely affected. As a consequence, Broca developed the idea that the gyri themselves, rather than the wider region, must be the site responsible for speech production. As the causes of damage are so different between the cases of Leborgne and Lelong, Broca further concludes that the crucial point was precisely not the *cause* (i.e., epilepsy, as recorded by Broca in the case of Laborgne, or stroke) but instead the **localisation of the damage**. So, it was Broca's extension of the idea of functional localisation to specific gyri within larger regions that marks him as one of the most prominent figures in the history of neuropsychology in general and aphasiology in particular. Localisation really became 'local' with Broca.

Although the symptoms of Lelong too are described relatively superficially, they nevertheless resemble more a 'motor' aphasia as we would now describe it. But subsequent examination using modern technology showed that both hemispheres of Lelong's brain display the kind of atrophy that occurs in senile dementia (Castaigne, 1980). Thus Broca's second case too was a somewhat atypical case of aphasia (see further discussion of recent scanning of the brains of Broca's two original cases in Chapter 9).

We can wonder, with Lord Brain (1961) quoted above, how it was that these two cases could become the point of origin of the classic theory of aphasia, after similar attempts by Bouillaud and Auburtin had failed to have the same impact years earlier. Broca's cases are certainly anatomically better described, despite the superficial autopsy by present-day standards, but that

alone cannot have been the reason, because the clinical description of symptoms was no better than previous case descriptions, and in some respects not as good (Gessner, for instance) and the conceptualisation of aphemia as a speech disorder was something of a detour from questions established by Gall concerning aphasia (a language disorder), and indeed many of the phrenology case descriptions are much better examples of clinical research.

Probably the changed political situation, the spirit of the time (the *Zeitgeist*), had an important influence, where the localisationists cast themselves in the role of the scientific progressives. But it was especially due to further developments (described below) and to the discussion engendered by Broca's presentations, that Broca's two cases were singled out by subsequent aphasiologists. Sondhaus and Finger (1988) conclude:

> It thus seems clear that Broca's 1861 report is important not because he presented a novel type of case material, nor an entirely new theory, but because it had great impact in changing scientific opinion more than any previous report on the subject.
>
> (Sondhaus, & Finger, 1988: 106)

The subsequent discussion developed fast, and the years until 1866 saw remarkable events for the history of aphasia.

In 1863, Broca presented further cases of aphemia that all had left-hemisphere lesions and all – except for one – had damage to the third frontal gyrus. Broca (1863) remarked only that it was strange that the lesions were all in the left hemisphere, but made no theoretical connection or issue of the fact and so the beginnings of the idea that the left hemisphere was **dominant** for speech and language, and also for most other functions, began (Bogen, 1969; Code, 1987). **Jules Parrot** (1829–1883) presented a case to the Anatomical Society of Paris in 1863 in which the third frontal gyrus of the *right* hemisphere had been destroyed, but the patient had not suffered any loss of speech. Broca commented only that one exception does not invalidate the rule. Auburtin (1863) summarised the discussion up to this time and argued in favour of Gall, Bouillaud, and Broca.

In 1865, Broca (1865: 384) finally formulated the theory of **language lateralisation**, that is, language was represented in the left hemisphere. However, Broca's (1865: 384) famous sentence 'We speak with the left hemisphere' (*Nous parlons avec l'hémisphère gauche*) applies only to right-handers. For left-handers language may be lateralised in the right hemisphere. He discussed too the possibility that, in the case of damage to the left hemisphere, the right can compensate for the left hemisphere damage (Broca, 1865: 389) and that people with aphemia could actually be treated following the principles of child language acquisition under 'maternal' therapeutic guidance. These ideas were the first to entertain the possibility of brain–language **reorganisation** following brain damage (Code, 1987). With the introduction

of the concept of left-hemisphere dominance, the classic doctrine was developed further in a significant way (Bogen, 1969; Code, 1987).

The establishment of Broca as the founder of the idea of the connection between the impairment of language and left hemisphere damage is still controversial (Buckingham, 1986; Critchley, 1964b; Finger & Roe, 1996; Joynt & Benton, 1964; Schiller, 1992: 192ff.; Stookey, 1963: 1028f.). **Marc Dax** (1770–1837) had already written a paper for a regional physicians' meeting in 1836. This was one year before his death, but nearly 30 years before Broca's work. The connection between left-hemisphere lesions and speech disorders was clearly stated by Dax: 'There now remains a very interesting problem to solve: why does it happen that changes to the left cerebral hemisphere are followed by the loss of words, but not those of the right hemisphere?' (Dax, 1836/1865: 260)

Marc Dax's work remained unpublished, although it was submitted for publication at the *Académie de Médecine* by his son **Gustave Dax**, together with his own contribution as early as 1863, two years before Broca's 1865 paper, but the Dax contribution was not published until 1865, when Broca also argued in favour of left lateralisation. This led to a bitter conflict, with Gustave Dax claiming that his father was the first to discover the special role for the left hemisphere in the control of speech production (Schiller, 1992: 192ff.). So it is Marc Dax who should be credited with the finding that language is lateralised to the left, but Broca was already famous, and Dax was but a country doctor. We summarise the events of the Dax–Broca controversy in Table 3.1.

Hughlings Jackson, in a letter to the British Medical Journal in 1864 (1864: 572), noted that 'when defects of speech occur with hemiplegia, the hemiplegia is [. . .] invariably on the rightside . . . I have now seen thirty-one patients who have these curiously associated symptoms [. . .] M. Broca believes that diseases of the brain *on the left side only* produce loss of speech; and, if I were to judge from the cases under my own care, I should think so too'.

Interestingly, Broca understood well that people with aphasia have not only production problems, but also comprehension, reading and writing

Table 3.1 A summary of the original published claims of Dax and Broca that aphemia follows a left hemisphere lesion

1836:	Marc Dax completes and apparently presents his paper claiming that aphemia follows a left but not a right hemisphere lesion
1863	23 March: Gustave Dax deposits his paper, which includes his father's 1836 paper
1863	2 April: Broca presents his paper and notes that all eight of his cases of aphemia have left hemisphere lesions, but states 'I do not dare to draw any conclusion'.
1865:	Gustave Dax publishes his paper, which includes his father's 1836 paper.
1865	(Six weeks later): Broca publishes his paper confirming that the left hemisphere contains the seat of articulate language.

problems. Often neglected in historical surveys is that Broca had devised a first classification system for the aphasias, which also contains the later named sensory aphasia (though for Broca, it was *verbal amnesia*), the 'discovery' of which was wrongly attributed to Wernicke much later (Henderson, 1986). Broca's system comprised four forms of aphasia: alogia, verbal amnesia, aphemia, and mechanical alalia.

For Broca **alogia** was a language disorder caused by a general reduction in intelligence and **verbal amnesia** concerned cases 'where the patients can no longer understand normal connections between ideas and words', and these patients 'utter confused speech that often has no reference to what they wish to express'; moreover 'they have forgotten the meaning of the words that they utter and they do not understand the words any better that one addresses to them' (Broca, 1869: 255). So the essential characteristics of fluent aphasia with jargon were described by Broca (although in the framework of a reduction in intelligence). Those affected

> have lost all their intelligence, but they nevertheless continue to articulate without difficulties. They speak entirely randomly; they splutter chains of words that make no sense or they repeat mindless stereotypes; and although they do not understand at all what they are saying, they continue to articulate correctly.
>
> (Broca, 1869: 267)

Leaving aside the loss of 'intelligence', this was a very clear description of what would subsequently be called **Wernicke's aphasia**, but Broca's description of what was to be attributed to Wernicke is seldom acknowledged.

Aphemia, as already discussed at length, was the loss of the *ability* to produce articulated speech. The term is still used, mainly by neurologists, to describe what most speech and language pathologists and speech scientists call apraxia of speech. **Mechanical alalia** was a speech disorder caused by the inability to control the organs of articulation (now called *dysarthria*, or *anarthria* to indicate a complete loss). Broca emphasised that verbal amnesia and aphemia 'are entirely different from the point of view of physiological and pathological analysis' (Broca, 1869: 267), and he thereby pre-empted the dichotomies favoured up to the present day: motor or sensory, Broca's aphasia or Wernicke's aphasia, fluent or nonfluent aphasia. This dichotomy was not discussed any further by Broca.

This classification was also presented by Broca at a meeting in Norwich in England in 1868 (Critchley & Critchley, 1998: 94ff.), which we discuss below, and to which he had been invited by Frederic Bateman, himself a localisationist. The English neurologist John Hughlings Jackson also spoke at this meeting, but despite their mutual attendance, it is not known whether Broca ever met Jackson (Critchley & Critchley, 1998; Marjorie Lorch, personal communication).

'Aphasia'

Although Broca is regarded the founder of modern aphasiology for most, he was unable to establish his preferred term 'aphemia' (Henderson, 1990; Herrmann, 1990; Ryalls, 1984). The word 'aphasia' effectively replaced terms like alalia, language amnesia and, to a large extent, aphemia, although Broca fought fiercely for the term aphemia. The reason aphasia became established in preference to aphemia was due to **Armand Trousseau** (1801–1867), who was one of the most influential French physicians of the nineteenth century. Trousseau held the view that what he called aphasia was a **cognitive disorder** which also reduces the intellectual performance of those affected. This thought was also later to be expressed in similar fashion by John Hughlings Jackson. In 1864 Trousseau published an article with the pointed title 'On aphasia, a sickness formerly wrongly referred to as aphemia' (Figure 3.7), in which he criticised Broca's term with (pseudo-)philological arguments (*aphemia*, thought Trousseau, could imply 'infamy' – this homophonic effect works in French but not in English) and in which the term *aphasia* (from the Greek meaning 'without language') was proposed instead. Although Broca (1864) answered in detail and also proposed the alternative term *aphrasia*, he was ultimately unsuccessful, and even in Broca's 'house journal', the *Bulletins de la Société Anthropologique de Paris*, 'aphasia' prevailed. Broca (1869: 266) himself finally succumbed and used the term *aphasia* as the umbrella term.

Subsequently we arrive at the position accepted nowadays, where *aphasia* became the generic term that covers all acquired impairments of language processing in all modalities, with *aphemia* still retained and used by some to refer to *apraxia of speech*.

British aphasiology until 1870

In Britain, **John Hughlings Jackson (1835–1911)** was born in the Northern English county of Yorkshire to a farming family (see the biography by Critchley & Critchley, 1998). He attended a local village primary school and later was sent to boarding schools, one of which was in the Yorkshire town of Halifax. He left at 15 with a low opinion of formal education and his father paid for him to be apprenticed to general practitioners in York. His apprenticeship lasted five years during which time he enrolled at the small York Medical School in 1852 and three years later completed his medical training at St Bartholomew's Hospital in London. Jackson's provincial medical training was not unusual at that time; there was a network of medical schools unattached to universities around the United Kingdom. He considered that his lack of formal education was the opposite of a hindrance to him, being convinced that over-education was not conducive to the development of clear thinking. Despite some apparent early education in music, he had little interest in theatre or literature, and although he read a great deal, it was primarily non-fiction. He went on to become 'the father of English neurology' and his contribution to medicine included epilepsy and

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SOMMAIRE. — PARIS. Sur la séance de l'Académie des sciences. — Hôtel-Dieu (M. Trousseau). De l'aphasie, maladie décrite récemment sous le nom impropre d'aphémie. — Kyste de l'ovaire multiloculaire enflammé; ponction; canule à demeure; injection iodée; guérison. — Caillot fibrineux dans les cavités droites du cœur et l'artère pulmonaire; mort subite. — Académies et sociétés, séance du 4 janvier. — Nouvelles.

PARIS, LE 11 JANVIER 1864.

Séance de l'Académie des Sciences.

L'Académie, dans sa séance du 4 janvier, a procédé à l'élection d'un vice-président pour l'année 1864. M. le général Morin, vice-président en 1863, passant à la présidence. La majorité des voix s'est portée sur M. Decaisne, qui a été proclamé vice-président et a pris place immédiatement au bureau en cette qualité.

M. Velpeau, président sortant, a rendu compte à l'Académie, avant de quitter le fauteuil, de l'état de ses publications et des changements survenus, pendant le cours de l'année, parmi ses membres et ses correspondants.

Voici en quelques mots l'exposé de ces changements :

L'Académie a perdu trois membres titulaires et un associé étranger : M. Bravais, de la section de géographie et de navigation; M. Despretz, de la section de physique générale; M. Moquin-Tandon, de la section de botanique; et M. Mitscherlich, de Berlin.

Elle a élu trois membres nouveaux en remplacement des trois membres titulaires décédés : M. ...

particulièrement, parmi les communications qui ont été faites dans cette séance, une note de M. Schnepp sur le thé du Paraguay, note qui intéresse tout à la fois l'histoire naturelle et l'hygiène, et sur laquelle nous appelons l'attention de notre collaborateur du feuilleton scientifique; un travail de MM. Pécholier et Saintpierre (de Montpellier) sur la santé des ouvriers employés à la fabrication du verdet (acétate de cuivre); et une note de M. A. Claudel sur quelques phénomènes produits par la puissance de réfraction de l'œil.

On verra enfin dans le compte rendu que, sur la demande de M. Pasteur, l'Académie a nommé une commission chargée de suivre les expériences et de juger la question des générations spontanées. — Dr Brochin.

HOTEL-DIEU. — M. TROUSSEAU.

De l'aphasie, maladie décrite récemment sous le nom impropre d'aphémie.

M. Broca a décrit, il y a déjà quelques années, sous le nom d'*aphémie*, une maladie qui consiste à ne pouvoir prononcer certains mots ni exprimer ce qu'on a l'intention, la volonté de dire. C'est sous ce nom que depuis trois ou quatre ans plusieurs médecins ont étudié cette maladie. Mais, comme le mot est nouveau, bien que la maladie ne le soit pas, et qu'il faut désormais que nous adoptions un terme pour la désigner, j'ai voulu savoir, avant de me servir de celui qui a été proposé, s'il est correct et s'il ... bien la chose que l'on veut dire.

crivis quelques purgatifs, des frictions stimulantes aux tempes et à la nuque. Au bout de dix à quinze jours, le malade commença à bredouiller, puis à parler de plus en plus clairement, et il sortit, après un mois de traitement, complètement guéri. Il put nous raconter alors ce qui lui était arrivé, et il ajouta que pendant la courte maladie qu'il venait de faire, non-seulement il ne pouvait prononcer aucun nom, mais encore que les mots ne se présentaient pas à sa mémoire.

Ce fut là le premier fait de ce genre qu'il me fut donné d'observer. Je ne m'y arrêtai pas autrement. Mais le mémoire de M. Broca, qui parut quelque temps après, m'édifia sur sa signification.

L'année dernière, comme je causais de ce sujet avec un de nos éminents confrères, il me dit qu'il lui était arrivé à lui-même quelque chose de semblable. Voici ce qu'il me raconta, et que j'ai écrit en quelque sorte sous sa dictée :

Il était à la campagne et il s'y était donné une entorse qui l'obligea à rester au lit pendant quinze à vingt jours. Il a le bonheur d'être myope, par conséquent de pouvoir lire autant que cela lui plait. Aussi lisait-il toute la journée. Un beau matin, pendant qu'il parcourait les *Entretiens* de M. de Lamartine, il s'aperçut tout à coup qu'il ne comprenait pas bien le sens de ce qu'il lisait; il reprit le passage et ne comprit pas mieux la seconde fois que la première. Attribuant cela à la fatigue, il mit le livre de côté et se reposa. Un quart d'heure après il le reprit, relut le même passage et ne le comprit pas mieux encore. Cela l'étonna, l'inquiéta même. Il jette le livre et sonne son valet de chambre. Celui-ci ... il veut lui ... un ordre, il ... muet. Il s'épouva... fait de ...

Figure 3.7 The term 'aphasia' was first introduced in 1864 in an article by Armand Trousseau in this edition of the *Gazette des Hôpitaux Civils et Militaires.*

mental illness as well as aphasia. He was elected the first President of the Neurological Society of London in 1886 and with colleagues founded the journal *Brain* in 1878 (for further details of Jackson's life, see Critchley & Critchley, 1998).

Jackson developed his highly influential theory of the evolution and organisation of the central nervous system, supported by his observations of aphasic and epileptic symptomotology and significantly influenced by the evolutionary ideas of **Herbert Spencer** (1820–1903). Head (1926) commented that: 'Jackson derived all his psychological knowledge from Herbert Spencer, and adopted his phraseology almost completely. This has tended to alienate psychologists, blinding them to the truths underlying this somewhat uncouth nomenclature' (Head, 1963: 31). But his work on aphasia had little impact outside Britain and was relatively unrecognised until Henry Head's writings led to its recognition and rediscovery in the early twentieth century. Other participants in the aphasia discourse were William Ogle, Henry Charlton Bastian, as well as Frederic Bateman, who all, to some extent, maintained positions conflicting with each other.

John Hughlings Jackson: the evolutionary of brain and language

Nowadays Hughlings Jackson is often perceived as *the* prime opponent to the language localisation of his time, and the idea of pursuing a localised 'seat of language' was not something he considered useful; but he was more than just an anti-localisationist (Critchley & Critchley, 1998; Schulte, 1994). He introduced original views into aphasiology and developed his ideas while remaining relatively uninfluenced by the debates in the closing nineteenth century. Even though one of the leading neurologists of his time he had almost no influence on these European debates because his approach and his sources of insight were predominantly clinical, he employed a difficult style of writing, he stood against the localisation *Zeitgeist* after 1870 and, as a follower of popular and controversial evolutionist Herbert Spencer (Schmitz, 1983: 186ff.), was exposed to intense criticism. In this context, we should note that **Charles Darwin's** (1809–1882) *Origins of Species by Means of Natural Selection* was published in 1859 and the theory of evolution was being widely discussed beyond the biological disciplines and also had a massive public impact. Hughlings Jackson's first work on aphasia was published a few years later in 1864 and his last in 1894. Essential features were already contained in his earlier work and will be summarised here (see also summary in Caplan, 1987: 89ff.).

According to Jackson both the ontogenic (individual development) and phylogenic (species development over time) evolution of the nervous system involved, first, a passage from the most to the least organised, from the lowest, well-organised centres up to the highest, least organised, centres; second, a passage from the most simple to the most complex (again from the lowest centres to the highest centres); third, a passage from the most automatic to

the most voluntary. *Dissolution* (a term originating with Spencer) of the nervous system, with loss of functions, would provide evidence of the reverse of evolution. Functions, according to Jackson's model, are organised hierarchically in the nervous system at different **levels of representation** from the oldest to the most recently developed in evolution and individual development, from the lowest to the highest, from the most primitive to the most complex.

Represented in the animal and human brain are mechanisms of control for individual levels varying in phylogenic age. Basic, primitive functions are controlled by phylogenetically older parts of the brain (e.g., the nervous system's control of breathing, heart rhythm) whereas higher, cognitive functions (such as higher aspects of language) are controlled by phylogenetically younger parts of the brain, that is, the neocortex. Higher functions are often able to consciously control, modify, and also **inhibit** the lower functions. One can, for instance, consciously inhibit a yawn and modify basic body movements into dancing.

From Jackson's perspective, damage done to the brain resulted not only in what he called **negative symptoms**, which occur as a *deficit* or a loss (e.g., not being able to write), but also in what he termed **positive symptoms**. These are new behavioural phenomena emerging from undamaged parts of the brain. He states: 'it is an abuse of language to say that the negative condition is the cause of the positive phenomena, for that implies that nothing causes something' (Taylor, 1958: 17). In general, Hughlings Jackson supposed a **reorganisation** of the processes in the brain after it had been damaged. Thus, positive (though undesired) symptoms can develop through a failure of higher inhibition, as an expression of lower levels. Neurological and neuropsychological symptoms could therefore be explained as the manifestation of primitive representations that emerge as the result of a failure of inhibition resulting from damage to levels higher up the hierarchy (e.g., cortical).

Jackson approached language disorders entirely differently from, for instance, Broca, Bateman, and others, who predominantly dealt with negative symptoms (deficits) at the lexical, single word, level. Jackson felt that the **function of language** should be essentially integrated and words and utterances cannot be considered in isolation: 'speaking is not simply the utterance of words. The utterance of any number of words would not constitute speech. Speaking is "propositionising" ' (Hughlings Jackson, 1874/1958: 130).

It was Hughlings Jackson's (1874) original observations of aphasic *recurring utterances* in the later nineteenth century, an example of a 'positive' symptom, that led him to propose the idea of **propositionality** in language. It will be recalled that Broca's first patient, Leborgne, produced the recurring nonsense utterance '*tan, tan*'. Non-propositional speech is produced automatically and the individual linguistic elements are not newly or individually generated. Non-propositional speech includes cursing and swearing, automatic rote-learnt serial verbal activities like automatic counting, nursery

rhymes, prayers and the recitation of arithmetic tables. Jackson distinguished this kind of speech from propositional speech where original ideas are being encoded into newly generated and novel referential utterances. Jackson (1874) also introduced the idea that the left hemisphere was responsible for processing propositional language, whereas both right and left hemispheres were engaged in the processing of non-propositional language. This was really the first developed expression of the idea that there may be a division of control between the hemispheres for different aspects of language. For Hughlings Jackson (1866/1958: 127) then, a sentence is more than 'a word heap', and a heap of words does not result in a proposition. For him, the **proposition** (statement) is a fundamental feature of language (Critchley & Critchley, 1998: 101f.).

Aphasia for Jackson was an impairment in the ability to build propositions: 'loss of speech is the loss of power to propositionise' (Hughlings Jackson in Schulte, 1994: 152). Jackson also differentiated between internal and external speech; external speech is what we produce and thought processes are internal speech. As speech is conceived of as a part of thinking, a loss of speech always affects the ability to build propositions in internal speech. Although this does not mean that speechless people can no longer think, the thought is limited however, and people affected can no longer learn or process complex thought:

> The speechless man can think, I suppose, because he has in automatic forms all the words he ever had; he will be lame in this thinking, because, not being able to revive words (to speak to himself), he will not be able to register new and complex experiences of things.
>
> (Hughlings Jackson, 1874/1958: 131)

What is preserved, however, in what Jackson called the *speechless man* is automatic, **non-propositional** speech as it occurs, among other things, in emotional speech (e.g., in cursing). Propositional speech is under conscious control and non-propositional speech is the product of deeper, automatic processes that are inhibited under normal circumstances. Following certain kinds of brain damage, non-propositional speech can become disinhibited, resulting in the automatic production of emotionally charged utterances. Jackson had – with acknowledgement to *Baillarger's* (1865) earlier distinction between *voluntary and involuntary speech* – observed that aphasic people can often produce complete phrases in particular contexts (curses, exclamations, stereotypies, etc.), even when they produce hardly anything in spontaneous speech. These automatically produced recurring utterances, however, are without conscious control. Hughlings Jackson (1874/1958: 133) wrote that 'the speechless man has lost speech (in the fullest sense that is, not being able to propositionise in any way), and that he has not lost the automatic use of words'. Jackson therefore dedicated much attention to the occurrence of **speech automatisms** (*recurring utterances*) and to **jargon**, and he also used these terms systematically. (There is further

discussion of speech automatisms within the context of Broca's aphasia in Chapter 9.)

Jackson did not support the assumption of a speech centre where the faculty of speech is localised, and he recommended caution in the interpretation of clinical findings: 'I think, then, that the so called "faculty" of language has no existence' (Hughlings Jackson, 1866/1958: 123), and 'I have never acceded to the opinion that speech is to be localised in any one spot' (Hughlings Jackson in Critchley & Critchley, 1998: 98). In addition, '**to locate the damage which destroys speech and to locate speech are two different things**' (Hughlings Jackson in Schulte, 1994: 139).

Despite these reservations, Jackson nonetheless supposed that the 'region of Broca's convolutions' played a special role in speech. In addition, Jackson argued in favour of a clear hemispheric specialisation. 'The right hemisphere is the one for the most automatic use of words, and the left the one in which automatic use of words merges into voluntary use of words – into speech' (Hughlings Jackson, 1874/1958: 131). Speech automatisms originate in the undamaged right hemisphere. Interestingly, Jackson attributed comprehension to the right hemisphere too, which may be for him a type of automatic processing. With comprehension processed by the right hemisphere, Jackson was able to support his opinion that 'the speechless man' understands well.

William Ogle: early descriptions of 'agraphia

William Ogle (1827–1912) is not to be confused with John William Ogle (1824–1905), who also wrote on aphasia (e.g. J. W. Ogle, 1874). William Ogle (1867) authored an article with the modern sounding title 'Aphasia and agraphia', which deals explicitly with verifying the assumption that the faculty for articulated speech can be localised in the posterior part of the third frontal gyrus. This question was answered in the affirmative through the description of 25 cases, and explanations for the exceptions were also provided.

Despite the fact that William Ogle is seldom considered in histories of aphasia, his work is important for various reasons: his work was probably read by other aphasiologists of his time, the idea of language centres is important in his writing, different forms of aphasias were postulated by him that corresponded to an impairment of the respective centres, he developed a simple word-processing model as a basis and the introduction of the term '**agraphia**' is credited to him.

Ogle (1867) conceptualised word production in the following way:

> What is necessary in order that a man shall communicate his ideas to another by speech? In the first place his ideas must evoke at once their appropriate symbols in his mind, each idea mentally clothing itself in the word or words which conventionally stands for it.
>
> (Ogle, 1867: 94)

Finally, the word is uttered.

The inability to convert existing concepts into the correct symbols represents a first form of aphasia that Ogle (1867: 95–5) called **amnemonic aphasia**, and it is characterised as follows:

> The patient in such a case suddenly in speaking stops short, unable to recall the words he requires. The moment, however, that he is prompted he is able to go on. [. . .] Sometimes, instead of actually stopping in his speech, he avoids the difficulty by some periphrasias, or frequently substitutes for the forgotten symbol some perfectly different one, with a perfectly different meaning. [. . .] often though the two words, that used and that meant, are utterly unalike, [. . .] there is a certain degree of similarity. [. . .] More frequently, however, the resemblance is one of sound. [. . .] In all cases, however, it appears to be a constant fact that grammatical form is observed; only substantives are substituted for substantives, verbs for verbs, numerals for numerals, proper names for proper names.

Ogle's description of 'amnemonic' aphasia resumed significance about one hundred years later under the name *amnestic aphasia* and, in modern terminology *anomia*, and Ogle describes above the main symptoms of semantic and phonological paraphasias and **deblocking** through assistance.

Ogle's (1867: 96) second form of aphasia is **atactic aphasia**, where the patient is sometimes entirely without speech, but more often some highly frequent words are available ('no', 'yes', 'my father', etc.), which are sometimes used correctly. The patient often switches to using pantomimes. Ogle considered *Broca's aphasia* an atactic aphasia. (Incidentally, Ogle was probably the first aphasiologist to use the term 'Broca's aphasia'.) This form of aphasia is most remniscent of the severe form of aphasia often called *global aphasia* nowadays, which is also characterised by significantly limited vocabulary, speech automatisms and non-verbal compensation.

Ogle is one of the first to note that it is possible that one form of aphasia can evolve into another form, thereby describing an early notion of **syndrome shift**. Furthermore, it is clear for Ogle that peripheral speech disorders (due to paralyses for instance) are not aphasias because aphasias are central neurogenic disorders and have a 'mental cause', as Ogle puts it: 'Were [the external organs of voice and articulation] entirely paralysed, there could be no speech; but such a case would not be one of aphasia. For this term is [. . .] limited to the loss of speech from mental causes' (Ogle, 1867: 98).

Ogle (1867: 99) appears to have coined the term **agraphia**, which also existed on his model in an amnemonic and an atactic form: 'Of this defect, for which [. . .] I would coin the name agraphia, there are moreover, as of aphasia, two forms – an amnemonic and an atactic.' With amnemonic agraphia the patient can write, but produces malapropisms or writes meaningless alphabetic strings. According to Bateman (1870: 98), Ogle's

term 'amnemonic aphasia' goes back to the 'amnesic aphasia' that a certain Popham is said to have introduced. With **atactic agraphia**, however, the ability to write letters has been lost almost completely. In such cases Ogle also tests if the patient can write words or their own names with letters on cards.

While Ogle recognises that aphasia and agraphia often occur together, they can also occur independently. From that Ogle concludes that **different cortical centres** must underlie speech and writing. He pre-empted contemporary modelling when he stated that 'Aphasia and agraphia are usually combined together. [. . .] The occasional separation of agraphia and aphasia points [. . .] to the existence of distinct cerebral centres for the faculties concerned in speaking and writing' (Ogle, 1867: 100). He suspected, however, that these centres are contiguous.

With the observation that aphasias are complex manifestations that can also occur selectively, and the assumption that there are cortical centres in the brain which can be affected selectively, Ogle took a decisive step beyond Broca. But the issue of comprehension deficit as part of aphasia remained strangely neglected by Ogle who considers that comprehension problems do not occur with aphasia.

Henry Charlton Bastian: his early work

Another British localisationist and admirer of Broca was **Henry Charlton Bastian** (1837–1915; see Figure 3.8) (Kalinowski, 1953; Marshall, 1994), who published an important article in 1869 where he divided neurogenic speech disorders into three groups which he illustrated with detailed case presentations. These groups were aphasia, aphemia and agraphia. Patients with **aphasia**, Bastian (1869: 218) suggested, 'can think, but cannot speak or write', with **aphemia**, the writing and thinking function is preserved, but not the speech function. Bastian (1869: 229) illustrates this: '[T]he patient was able to talk a meaningless jargon: and when attempted to read aloud gave utterance also to a series of articulate sounds, having no intelligible meaning or resemblance to those which he should have uttered'. With **agraphia** writing is affected in isolation, but not speech and thinking. It is noteworthy with Bastian that aphasia clearly is a rather abstract language disorder that concerns both expressive modalities, and that also modality-specific disorders like agraphia can occur.

Bastian (1869: 482) also contributed significantly to the discussion of **deficits in language comprehension**:

> In certain severe cases of Aphasia [. . .] it is distinctly stated that the patient either did not gather at all, or with difficulty and imperfectly, the import of words when he was spoken to, though he could be made to understand, with the utmost readiness, by means of signs and gestures.
>
> (Bastian, 1869: 482)

Figure 3.8 Henry Charlton Bastian (1837–1915), who was one of the first to notice aphasic deficits in comprehension.

Although Bastian (1869: 482) also referred to 'perceptive cortical centres', he did not yet make a connection between language comprehension deficit and a localisation of a 'faculty' for comprehension, as Wernicke was to do several years later

Where Wernicke later clearly separated comprehension from thinking, Bastian (1869: 216) equated thinking and language: 'We think in words, in fact, and these words are revived as sound impressions in the auditory receptive centres of the cerebral hemispheres'. Interestingly, **William Broadbent** (1835–1907) – later well known through his work on alexia and agraphia (see Finger, 1994: 400–401) – identified a cortical auditory centre in the temporal lobe following Bastian (and still before Wernicke) (Meyer, 1974: 570).

Brown (1984: ii) remarks that Bastian seemed to have been annoyed about the fact that the 'discovery' of sensory aphasia was attributed to Wernicke, and that Wernicke had not cited his work, although Howard and Hatfield (1987: 21) concluded that 'as Wernicke acknowledged in 1906, Bastian, in 1869, was the first to observe that aphasic disorders were not confined to language production, but there could also be difficulty in comprehension'.

Frederic Bateman and Byron Bramwell: doubts on localisation

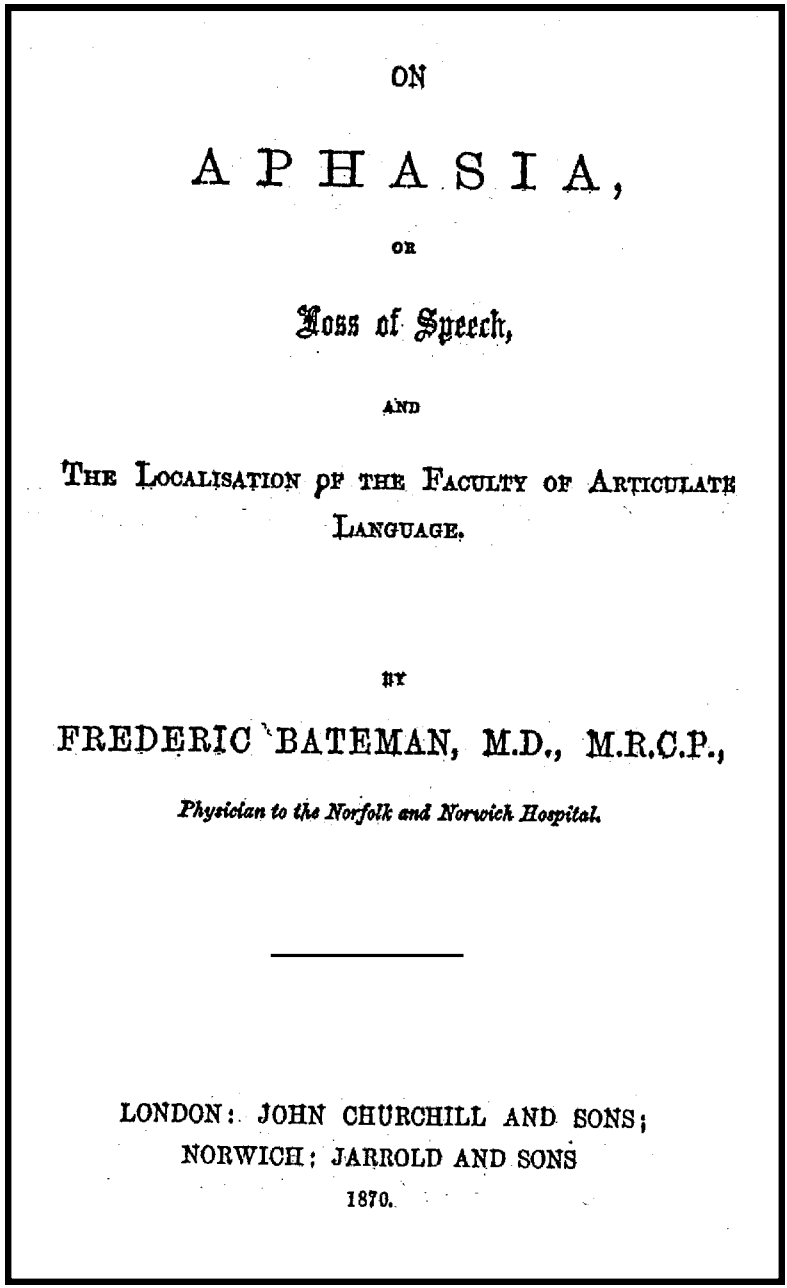
One of the first books dedicated exclusively to aphasia (Figure 3.9) was published in 1870 by **Frederic Bateman** (1824–1904), who is probably best known for bringing both Broca and Hughlings Jackson to speak at the conference in Norwich, England in 1868, where modern aphasiologists have been tantalised by the notion that they may have met. A recent investigation by Marjorie Lorch (personal communication) concludes that, partly because this was a very large meeting, it seems unlikely that they met. In Bateman's exemplarily systematic work altogether 72 cases (already published and his own) are discussed in order to clarify the question of localisation of speech. Bateman (1870: 130) used **aphasia** as an umbrella term: 'Having in this essay employed the word aphasia in its widest and most general sense, as applicable to loss of speech from whatever cause'. He also used – similarly to Ogle – the terms *amnesic aphasia* and *ataxic aphasia*. However, he sought less the prototypical of these forms, but rather emphasised repeatedly the variability of aphasic phenomena and the different compositions of symptoms because, according to him, the phenomenon could only be understood this way. Bateman is the first to clearly propose that aphasia could be described in terms of syndromes and the probability that each individual with aphasia has a unique pattern of symptoms (Breidbach, 1997: 127f.).

A range of symptoms and features of aphasia are presented in detail and with examples by Bateman. Aphasias can have different degrees of severity and deficits can be linguistically selective (e.g., specific word classes) and they can occur as modality-specific, with word finding impairments, searching behaviour and paraphrasing (meaning *to paraphrase*). Semantic and phonological paraphasias can occur and semantic and phonological jargon, echolalias, speech automatisms and stereotypies. Emotional (automatised) speech can often be preserved with aphasia and aphasic people can use non-verbal gestures expressively. In multilingual speakers, separate languages can be affected selectively. The presentation is impressive from a modern perspective and only disorders of language comprehension and syntax are missing from his extensive survey.

Bateman (1870: 115ff.) was also impressed by the range of possibilities in the presentation of the causes of aphasia, among which he also identified non-neurogenic, psychogenic ones.

On localisation, Bateman discussed the views of Gall, Bouillaud, Dax, and Broca and attached great importance to statistical testimony in his arguments, conscientiously checking supporting cases and exceptions. Bateman (1870) reached the following conclusions:

> That although something may be said in favour of each of the popular theories of the localisation of speech, still, so many exceptions to each of them have been recorded, that they will none of them bear the test of a



ON

APHASIA,

OR

Loss of Speech,

AND

THE LOCALISATION OF THE FACULTY OF ARTICULATE LANGUAGE.

BY

FREDERIC BATEMAN, M.D., M.R.C.P.,

Physician to the Norfolk and Norwich Hospital.

LONDON: JOHN CHURCHILL AND SONS;
NORWICH: JARROLD AND SONS
1870.

Figure 3.9 One of the first books exclusively dedicated to aphasia was published by Frederic Bateman in 1870: *On Aphasia, or Loss of Speech and the Localisation of the Faculty of Articulate Language*.

> disinterested and impartial scrutiny. [. . .] That I by no means consider it proved that there is a cerebral centre for speech at all.
>
> (Bateman, 1870: 178)

Another British physician interested in issues of localisation, working at the Royal Infirmatory in Edinburgh, was **Byron Bramwell** (1847–1931), who is now remembered as the first person to describe the condition of **crossed aphasia**, although, ironically, the patient he described in 1899 (Bramwell, 1899) did not have what we now call crossed aphasia, which is aphasia arising from a right hemisphere lesion in a right-handed person. Crossed aphasia was of particular interest as it raised questions about the localisation of language to the left hemisphere and the relationship of handedness to language dominance. In addition, it poses the possibility that the lateralisation of language could be completely reversed (Brown & Code, 1987). In fact, Bramwell's case was *left*-handed and had a *left* hemisphere lesion (for discussion of the impact of Bramwell's case and subsequent discussion of crossed aphasia, see Schweiger, 1996). Another case described by Bramwell (1897) was a particularly good description of **word-meaning deafness**, a rare disorder of auditory word recognition (for a republished version of Bramwell's paper with commentary, see Ellis, 1984). Bramwell's patient had no or few problems in expression, reading aloud, repetition and comprehension of written material, but could not understand words said to her. She was unable to understand questions put to her, but could repeat the question and after writing it down without error, could understand it.

Summary

The period from the beginning of the nineteenth century to the 1860s resulted in the establishment of the idea that aspects of language were localised, which was initiated by Gall who, among other things, postulated a language centre in the frontal cortex. Subsequently, language localisation was regularly and hotly debated in French scientific circles (instigated by Bouillaud and Auburtin), but partly due to more liberal social circumstances, Broca finally made a breakthrough with his case studies and identified the third frontal convolution as a centre for the control of articulated speech. With this, aphasia became established as an important topic for scientific discussion. Also through the work of Broca, though anticipated by Dax by nearly 30 years, the special role of the left hemisphere in language production was revealed, the ideas of lateralisation and cerebral dominance became established and the foundations of what we now call the classical model of aphasia were laid and the foundation for a new and more informed investigation of the nature of aphasia began. In 1864, the word 'aphasia' was introduced by Trousseau and was differentiate from aphemia. In Britain in the 1870s and 1880s Hughlings Jackson proposed non-localisationist ideas on aphasia and language and introduced a new evolutionary theory of the organisation of the central

nervous system based on the teachings of Herbert Spencer. Other British aphasiologists included staunch supporters of localisation and some who shared Jackson's scepticism. Because of the intense interest in aphasia at this time across Europe, the nineteenth century is regarded as the period when aphasiology became established as an important and respectable scientific field, giving rise to the development of other areas of neuropsychology. In Chapter 4 we continue to trace developments in the remainder of the nineteenth century.

4 Wernicke and the later nineteenth century

Following Broca and the others influenced by Gall's organology, a broader discussion of aphasia developed across European science (Bateman, 1870: 23–62), but an increased interest in language localisation and aphasia, mainly in England and Germany, did not develop until after the debate between Broca and Trousseau. We have seen in Chapter 3 that this discussion was concerned with wider and more important issues than correct nomenclature and ranged over the nature, scope and psychopathology of language disorders.

Developments in Germany marked the way for the further growth in aphasiology from the 1870s. This took place in the context of a time considered the 'heyday of German universities' (Müller 1990/1996: 86) following the Franco-Prussian War of 1870–1871, and German institutions were the leaders in many domains. One decisive breakthrough for localisation theory was led by the experimental work of Fritsch and Hitzig. The **Zeitgeist** was in favour of localisation of cognitive functions, especially in aphasia, and aphasiologists found it more appealing to Flourens' equipotentiality. The Wernicke–Lichtheim model and what became the classic principles of aphasiology emerged on the basis of associationist psychology. With the clear formulation of a model, counter-positions can, and usually do, develop. Aphasiology in German-speaking countries was going through its most important phase during the final quarter of the nineteenth century.

Developments in German-speaking countries until 1885–1886

With *The Symptom-Complex of Aphasia*, Carl Wernicke (1874) published the most important work on aphasiology in the latter part of the nineteenth century, notwithstanding the input of previous German contributors (especially Meynert and Schmidt, both discussed below). The emergence of the Wernicke–Lichtheim model during 1885–1886 is an important milestone, but Wernicke's model was met with opposition right from the beginning (e.g., Finkelnburg's *asymbolia*). In what follows we take a step back in time to map the developments leading up to Wernicke's famous localisation theory.

The forerunners of language localisation theory

Rudolph Wagner (1805–1864) was one of the few to argue in favour of cortical centres in German science before Wernicke. With reference to Broca's and Bouillaud's cases, Wagner (1863) wrote:

> By means of these clearly analysed cases, the fact is however stated that very specific deep disorders of the most complicated, immediate movements entirely dependent on mental impulses (language), as well as the faculty to store and reproduce past impressions (memory), are dominated by relatively small, locally confined sources in the grey substance of the convolutions of the cerebrum [. . .], and these are destroyed if they are lesioned.
>
> (Wagner, 1863: 23)

Also active in Vienna at this time was **Moritz Benedikt** (1835–1920) (Benedikt, 1865, 1871) who expected the following from aphasia research: 'highly interesting information on the connection of mental activity and on the significance of individual sections of the brain for certain mental activities and for mental activity overall' (Benedikt, 1865: 898). But more importantly, Meynert, Schmidt, Fritsch, and Hitzig prepared the ground for Wernicke's approach and also for its great impact.

Theodor von Meynert: fibre theory and the sound field

A groundbreaking development in neurology came from **Theodor von Meynert** (1833–1892) in Vienna with his **fibre theory** (Hagner, 1997: 269f.; Papez, 1953; Von Stockert, 1970; Whitaker, 1998: 45–47; Whitaker & Etlinger, 1993). Meynert's approach in turn originated in the second Vienna School of Medicine of Carl von Rokitansky (died 1878) (Lesky, 1965). He brought some order to the complicated fibre system of the brain by differentiating between projection fibres and association fibres. **Projection fibres** connect subcortical parts of the brain to the cortex, and **association fibres** connect cortical areas to one another. For instance, projection fibres communicate sensory information from the sensory organs to the cortex, and the association tracts transmit perceptions, ideas, and memory contents between areas. A frequent use of certain association tracts leads to a consolidation of the tracts, so that a functional differentiation emerges over time. Kleist (1970), one of Meynert's students, summarised Meynert's position as follows:

> The projection systems connect the entirety of sensory organs and the entire musculature with certain spots of the cerebral cortex, the so-called projection fields, whose cells acquire lasting changes through stimuli transmitted to them, which differ depending on the peripheral connection: the memory images of sensations and the memory images of movement

> patterns. The association systems connect the whole mosaic of memory images among themselves and thereby become the anatomical substrate of concept formation, of thinking as a whole.
>
> (Kleist, 1970: 106)

In addition, Meynert determined that the anterior part of the brain was responsible for motor function and the posterior part for sensory function.

In a 'Case of language disorder, anatomically founded', Meynert (1866) described (several years before Wernicke) a female aphasic patient who displayed phonological and semantic paraphasias as well as language comprehension deficits (see Whitaker & Etlinger, 1993). Meynert assumed that a language disorder can also affect comprehension and that thinking is clearly separated from language, but that comprehension is also part of language. The patient had a lesion of the left upper temporal gyrus. and Meynert (1866: 183) postulated a **sound field** close to the Sylvian fissure, in the area of the island of Reil in the temporal area, and he came to the conclusion that the entire peri-Sylvian region was important for language processing:

> The connection of the anterior wall with the acoustic nerve gives the walls of the Sylvian fissure the significance of a sound field, the connection of the same anterior wall with the arch systems running through the island's marrow and in the outer capsule makes this sound field a central organ of speech.
>
> (Meynert, 1866: 183)

Meynert also discussed the sound field in connection with 'auditory hallucinations' in 'lunatics'.

Meynert spoke of sound images and memory images, which combine by means of associations, thereby invoking neurological support for **association** psychological theory, which originated with Hartley. Meynert assumed that the anatomy of neural fibres could explain psychological processes and that association systems are the anatomical substrate of mental faculties. In his later works, Meynert applied his approach also to mental disorders. Meynert's contribution was therefore significant, identifying the anterior part of the brain with motor functions and the posterior part with sensory functions, postulating a 'sound field' later to develop into **Wernicke's area** and recognising the importance of the entire peri-Sylvian region for language processing and confirming the relevance of comprehension impairment in aphasia.

Johann Baptist Schmidt: the receptive language organ

Following Meynert, **Johann Baptist Schmidt** (1823–1884) (Boller, 1977) published a case of 'Hearing and language disorder in consequence of apoplexy' in 1871. The female patient produced phonological and semantic deficits and syntactic errors, and she 'had to make an effort in order to find a word and to

utter it [. . . and] she conjugated irregular verbs as regular and she frequently used the infinitive instead of the designated tense' (Schmidt, 1871: 304). The patient's hearing was intact, she looked up at the door when someone knocked, for instance, but she could not understand speech.

Schmidt (1871: 305) formulated the following hypothesis: 'Hence the organ in the brain that has the function to combine the sounds and to produce the sound image must also have suffered damage'. This problem also affects language production because the generation of sound images is a precondition for speaking:

> That spoken language now [. . .] has suffered with hearing is to be explained by the fact that the production of sound images must precede speaking; the word that I want to utter is first created in the sound field by mental influence and then transmitted to the language centre.
>
> (Schmidt, 1870: 305)

Thus a central feature of Wernicke's thesis, a receptive language centre, was foreshadowed first by Schmidt, and Wernicke actually refers to his work, but without citing a reference.

Eduard Hitzig and Gustav Fritsch: localisation theory takes hold

In 1870, with the works of **Eduard Hitzig** (1838–1907) and **Gustav Fritsch** (1838–1927), the stage was finally set for a breakthrough to enable localisation theory to become the dominant paradigm and the rejection of notions of equipotentiality, such as those of Flourens' (Breidbach, 1997: 242ff.; Finger, 1994: 38ff.; Hagner, 1997: 273ff.; Walker, 1957: 106ff.). This stage setting is both scientific and political (Pauly, 1983). Hitzig, a physician specialising in electrotherapy, and Fritsch, a neuroanatomist, carried out famous experiments with dogs on Hitzig's wife's dressing table in his own flat (Fritsch & Hitzig, 1870; Hitzig, 1874a). Their **electric stimulation** of regions in the anterior brain resulted in specific movements (bending/stretching) of the front or back paws of the test animals. In addition, it was shown by ablations, where the relevant part of the brain was surgically removed, that the ablation did not lead to paralysis, but to indeterminate, undirected movements of the paws, showing that the triggering of a movement and the 'idea' of a coordinated movement belong together. This work resulted in a real 'boom of electrophysiological mapping of functional areas of the brain' (Breidbach, 1997: 248) and an increase in interest in localisation among German scientists, which hardly existed before 1870, unlike in Britain and France. The growing interest in Germany also gained impetus from a changed political climate (Pauly, 1983). Pauly points out that there was the assumption abroad that Germany under Bismarck and the human brain were organised in the same way with a clear hierarchical structure and divided into distinct but cooperative regions. Interest and research in localisation became popular and equating what was

the cutting edge of science with the state produced clear political dividends and government support came readily.

While Fritsch and Hitzig established the presence of a motor cortex in dogs, Hughlings Jackson (1863; Bennett & Hacker, 2003) had concluded that there must be a motor cortex following his observations of patients with epileptic convulsions affecting one side of the body where subsequent autopsy revealed that the brain was damaged on the surface of the opposite side to the convulsions. He speculated too that the motor cortex must be organised somatotopically (Bennett & Hacker, 2003). **Charles Sherrington** (1857–1952) with Grünbaum 1902 (Grünbaum & Sherrington, 1902) unmistakably established the existence of the motor cortex and its boundary at the central sulcus, clearly distinguishing and separating it from the somatosensory cortex (Bennett & Hacker, 2003).

Oppositions to localisation

Before Wernicke published his famous book which completed the foundations for an anatomically based theory of aphasia begun by Broca, counterpositions were already being developed in Germany which did not conform to this theory and conceptualisation of aphasia. Finkelnburg, for instance, saw aphasia far more broadly and developed his concept of *asymbolia*, and Steinthal stressed a purely psychologically orientated approach to aphasia.

F. C. Finkelnburg: the concept of asymbolia

In 1870, **Ferdinand Carl Finkelnburg** (1832–1896) presented a significant paper on aphasia at a conference of the medical section of the Lower-Rhinein Society in Bonn (Birchmeier, 1984: 43–53; Duffy & Liles, 1979; Finger, 1994: 380f.). His view was that aphasic disorders are only a part of a more comprehensive problem that he called **asymbolia**, namely the inability to produce and understand symbols of any kind:

> That is, the loss of word formation represents only a part of the total disruption [. . .] the disturbance simultaneously encompasses more or less all those cerebral processes that convey meaning through learned sensory signs – that is, through symbols – of any kind. Furthermore, it is not only the utterance of one's own meaning through symbols which is demonstrably impaired in the victims, but also the interpretation of the symbols of others; thus, both the expression and reception of meaning through symbols are impaired.
>
> (Finkelnburg, 1870, in Duffy & Liles, 1979: 163)

Wallesch (1990: 517), in his summary of a case study by Leischner (1943), describes the intact symbol system as follows:

> The occurrence of semantic links between production in different modalities is interpreted as indicating the presence of one central 'main symbolia' (symbol system), which is superordinate to the other secondary systems and constitutes the code that is used in internal thought.
>
> (Wallesch, 1990: 517)

To illustrate Finkelnburg's notion of asymbolia and help make his views more comprehensible, we summarise below some of the cases he described in 1870. Finkelnburg reported on five people with language processing impairments. The second case, 'a 48-year old widow of a day-labourer', is summarised as follows:

> Important in this case are: the loss of the understanding of spoken as well as written words accompanied by substantially undamaged intelligence; further, the loss of understanding of other kinds of symbols, including those with which she had been extremely familiar since childhood; and, also, the impossibility of acquiring and retaining the meaning of new signs. Thus the disorder includes not merely the formation of words or written characters for concepts but also the other way around – the formation of ideas from words. Furthermore, the disorder includes the expression of concepts through visual signs which have no connection with word-formation. In brief, it is a pervasive, complete, comprehensive disruption of every type of symbolic usage. Also characteristic was that her memory for factual knowledge was intact from the beginning of the disturbance.
>
> (Finkelnburg, 1870, in Duffy & Liles, 1979: 160)

The patient could express herself only in automatisms ('bassa', 'ton'), could neither read nor write despite her premorbid literacy and 'often reacted to verbal stimuli in a confused manner, apparently owing to a lack of understanding of the words she heard' (Finkelnburg, 1870, in Duffy & Liles, 1979: 160). In addition, she could no longer make the sign of the cross (although a devout Catholic), and could no longer interpret the ring of the bell for meals.

In the third case, 'parallel to the loss of language and writing, the loss of another symbolic function, namely that of understanding for (musical) notes' could be observed. In the fourth case, an additional problem to the language problem was 'the loss of comprehension of money, that is, of a value symbol' and 'the pantomimic deficit, the forfeit of understanding for coins, meaning for value symbols' (Finkelnburg, 1870, in Duffy & Liles, 1979: 162). The fifth case was a civil servant who confused rank and insignia and 'his understanding of the symbols of the ritual was lost, as well as those of government service, and the expressions of social convention' (Finkelnburg, 1870, in Duffy & Liles, 1979: 163).

The idea that aphasia can be explained in terms of asymbolia is of course diametrically different to the localisationist's conception of aphasia, as

championed by Broca and his successors. Wernicke was also to dismiss Finkelnburg's conception of aphasia, but Leischner (1943, in German; see Wallesch, 1990, for a summary of this case study in English) and others working in German aphasiology continued to interpret aphasic and gestural impairments as asymbolia and continued to have some support right up to the 1970s (Duffy & Liles, 1979: 165ff; Leischner & Fradis, 1974). Finkelnburg considered examples of comprehension impairment as aphasia, and he supposed that a central faculty (the symbol faculty) was impaired, and that aphasia/asymbolia was a supramodal disorder.

Heymann Steinthal: a psycholinguistic approach

Aphasia was considered from a psycholinguistic perspective for the first time in 1871 by philologist **Heymann (Chajim) Steinthal** (1823–1893), who was Professor of General Linguistics at the University of Berlin (Steinthal, 1871: 453–487; see Eling, 2006). In his critique of the medical literature, Steinthal (1871: 464) reacted against the superficial and linguistically shallow coverage of language: 'The clinical pictures have been recorded by far too incompletely and imprecisely; our physicians have not understood what the function of language is' (see Figure 4.1). For Steinthal (1871), this resulted in the following demand:

> Physicians must attempt to recognise for what reason or to what extent or how at all there can exist a locally limited organ for mental functions in the brain. [. . .] To this end, it is especially necessary to observe the mental phenomena more closely, to analyse them more meticulously, to get to know them better in their content and form.
>
> (Steinthal, 1871: 472–473)

Steinthal made the point that it is meaningless to attempt to localise some language function if one does not have a clear psycholinguistic description of what it is one is attempting to localise, a feature of the contemporary critique of some brain imaging research.

On the basis of his more exact analysis of aphasic language, Steinthal subsequently differentiated between aphasia and what he called **acataphasia**. In aphasia the problem is at the lexical level (in the sense of a word memory retrieval problem) and in acataphasia, it is the **sentence level** that is impaired. Steinthal (1871) illustrated acataphasia in the following example:

> another patient said during the examination of his eyes: 'one eye – eye is always – tears – been teary – I can't – I could earlier – especially – of course – with the years little [. . .] writing – glasses. One understands what the patient wanted to say; [. . .] his wording betrays, it seems to me, more the inability to make sentences [. . .] than a bad memory.
>
> (Steinthal, 1871: 478)

EINLEITUNG

IN DIE

PSYCHOLOGIE

UND

SPRACHWISSENSCHAFT

VON

DR. H. STEINTHAL

A. O. PROFESSOR FÜR ALLGEMEINE SPRACHWISSENSCHAFT AN DER UNIVERSITÄT ZU BERLIN.

Motto:
Denken ist schwer.

BERLIN
FERD. DÜMMLER'S VERLAGSBUCHHANDLUNG
HARRWITZ UND GOSSMANN
1871.

Figure 4.1 Heymann Steinthal's (1871) book contained the first psycholinguistic approach to aphasia.

So Steinthal took a crucial step beyond the level of the single word in early aphasiology, an advance that was largely ignored (especially by Wernicke and his successors) and taken up again only in the twentieth century.

In contrast to the dominant anatomical approach to aphasia, Steinthal proposed a **psychological (psycholinguistic) model of language processing**, basically consisting of three parts. Steinthal (1871) insisted that one must

> certainly differentiate three things that play a part in speaking: organic mechanics, mental mechanics, [. . .] content in terms of opinion or concept. The purpose of language is the presentation and representation of content by means of mental and organic mechanics [. . .] only that these three moments must be thought of not as existing independently from one another, but instead as dynamically related to each other in their essence and existence.
>
> (Steinthal, 1871: 482)

Steinthal's *organic mechanics* corresponds to the articulatory apparatus, *mental mechanics* approximates language processing, and the *conceptual content* is approximately the intention to speak that precedes an utterance. Interestingly, this roughly corresponds to the main components of the currently dominant psycholinguistic model of Levelt (1989, 1993, 1995; Levelt, Roelofs, & Meyer, 1999).

The different language disorders were attributed to damage to one or other of these mechanisms: 'now each of these three mechanisms can be disordered. [. . .] This is how the actual diseases develop: first stammering, stuttering, and anarthria, second aphasia and acataphasia, third mental disorder' (Steinthal, 1871: 483).

Steinthal hence was the first to argue for a psycholinguistic characterisation of aphasia de-emphasising brain anatomy, a position that Kussmaul would also take some years later and would re-emerge more than one hundred years later with developments in cognitive neuropsychology. This tussle between those who saw the value of localisation of function and those who felt its claims were premature came to a head in a famous debate that took place in Berlin.

The 1874 Berlin language debate: Hitzig vs. Steinthal

In 1874, Hitzig and Steinthal clashed in the language debate of Berlin at a meeting of the Berlin Society for Anthropology, Ethnology and Prehistory (Hagner, 1997: 279–293; Jacyna, 1999) and the exchange was published in its entirety (Hitzig, 1874b, 1874c; Lazarus, 1874; Steinthal, 1874; Virchow, 1874; Westphal, 1874a, 1874b).

The debate started with a presentation by **Hitzig** (1874b: 44) on the 'Localisation of mental centres in the cortex'. As an example, he described an aphasia in which 'the memory organ for sound images' had supposedly been destroyed, and his conclusion (Hitzig, 1874b: 46) was 'that the most

excellent human faculty, that of independent language formation, possesses its organ in the front brain'. This he argued by comparing the motor activities in dogs with human language (equated with motor activity).

Steinthal (1874: 50) responded to this, as in his book, insisting that imprecise descriptions of symptoms will not do: 'If one wants to localise, then one must first know exactly what one can localise, which elements one is dealing with and, secondly, in which form their localisation can be conceived.' **Rudolf Virchow** (1821–1902), the so-called 'Berlin Broca' who dominated the society in which the discussion took place, arbitrated between them, and **Carl Westphal** (1833–1890) continued the discussion. Westphal (1874a: 101) presented three cases with a discussion of aphasic symptoms that were clinically very different and reached the following unsatisfactory, if convoluted, result for localisationists:

> From the material at hand, one may, namely under consideration of the undeniably verified pathological-anatomical findings in different locations of the brain with aphasia, and if one does not want to leave the scientific ground, it can only be concluded that apparatuses exist in different locations of the brain (respectively, of the cortex) whose destruction can impair the speech mechanism and the mental processes related to it in different ways, similarly to how one can bring an artificial mechanism into disarray by removing a screw or a spring in one place or another – not in all places. [. . .] So certain brain regions may also have a particular significance for aphasia because of their anatomical composition and the amalgamation of certain pathways, without being allowed to therefore now speak of a language centre and to exploit that fact for the localisation of mental faculties.
>
> (Westphal, 1874a: 101)

Lazarus (1874: 135) finally offered the insight (without knowledge of Wernicke's work): 'if understanding language belongs to language, then the term aphasia will not be exhausted by saying: it is a disorder of the ability to speak; instead it also consists in the inability to understand what has been spoken.' And he suspected that the idea of a single language centre is not sufficient and therefore one '[would] reach the categories for aphasia not more easily, but rather with even more difficulty' (Lazarus, 1874: 135).

In the closing discussion Hitzig (1874c: 138) distanced himself from his original statements and formulated more carefully:

> [I] would like to [. . .] point out that I have not claimed, and do not want to claim, that the conditions for language are all localised in a circumscribed place, the third frontal convolution. I only said that the faculty to speak is often destroyed by a destruction of this place and these are two different things. The language faculty can be composed of the function of many locations.

The Berlin language debate highlighted central issues in aphasiology that continued to concern aphasiologists in subsequent decades and persist to this day, including miscommunication between disciplines, superficial and incomplete observations, simplified conceptions of language localisation. Steinthal's put-down where he refers disparagingly to 'Messrs. Physicians' resonates even at the beginning of the twenty-first century. He was appalled by their lack of understanding of language and communication, contending that their observations must therefore be superficial and without theoretical value. However, Steinthal's pleas fell largely on deaf ears and localisationism was soon to witness its supreme dawn through the work of a young physician.

Carl Wernicke: *The Symptom-Complex of Aphasia*

It was also in 1874 that **Carl Wernicke** (1848–1905; see Figure 4.2) published the most renowned and most influential work in the history of aphasia: *The*

Figure 4.2 Carl Wernicke (1848–1905), whose work determined the aphasiology of the closing nineteenth century.

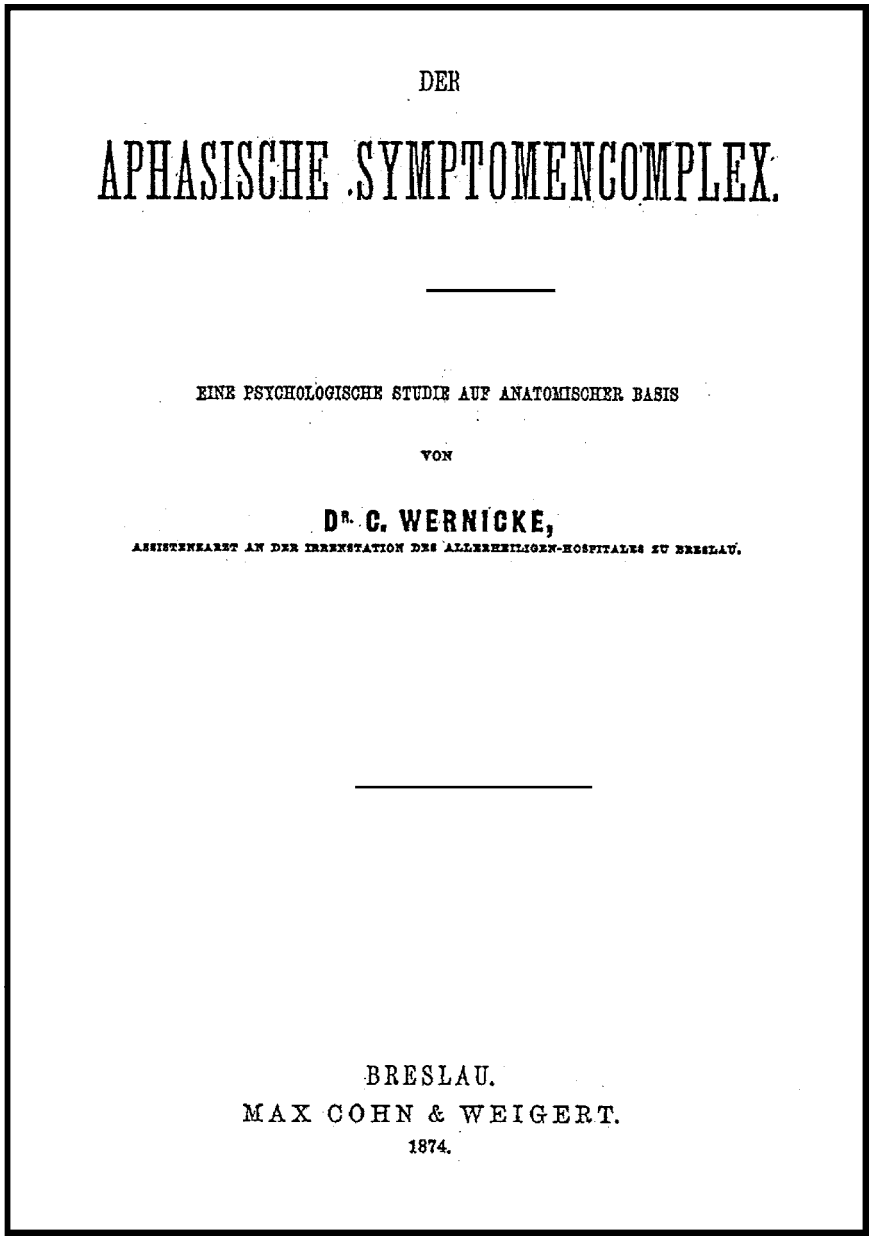
Symptom-Complex of Aphasia: A Psychological Study on an Anatomical Basis (*Der aphasische Symptomencomplex. Eine psychologische Studie auf anatomischer Basis*) (Arbib, Caplan, & Marshall, 1982: 10ff; Blanken, Dittman, & Sinn, 1993b, 1994; Cegelski & Dustmann, 1999; Eggert; 1977; Geschwind, 1966, 1967; Goldstein, 1953a; Keyser, 1994; Kleist, 1970; Leischner, 1981; Tesak, 2005; see Figure 4.3). With this work, the foundation for what became the **classic doctrine** was fully established. The **Zeitgeist** was amenable to language localisation and the book had an enormous impact: 'This epoch-making work was to set the tone for research in aphasia over the next 40 years' (Geschwind, 1966: 4).

Wernicke had spent some time as Meynert's student, and in his book Wernicke (1874) applied Meynert's views to the psychophysics of language and makes no secret of the fact that the crucial inspiration for his work is Meynert's anatomy and his idea of the sound field: 'In any case, everything that could be found to be of merit in the present work ultimately goes back to Meynert, because the opinion argued here results directly from his writings and preparations' (Wernicke, 1874: 3). The point of origin is the basic anterior–posterior separation of the brain:

> The whole surface of the cerebrum now falls into two large areas of functionally different significance: the frontal brain, the whole area situated in front of the fissure of Rolando of each hemisphere, and the common occipital temporal brain. The former is a motor area, i.e. it contains images of motions, the latter a sensor, i.e. it contains memory images of past sensations.
>
> (Wernicke, 1874: 5)

In his literature review Wernicke (1874: 15–16) states that 'Broca's area is not the only one to function as a language centre, because 'most cases of aphasia in which Broca's area was found to be unchanged, could display changes in the area claimed by Meynert'. And Wernicke (1874: 18–19) was then able to exactly place his own language areas anatomically (note that Wernicke counted convolutions from the middle of the brain towards the anterior, so that his first frontal convolution was Broca's third):

> The whole area of the first convolution circling the Fossa Sylvii together with the insular cortex serves as speech centre; and thus the first frontal convolution, because it is a motor area, is the centre of motor images, the first temporal convolution, because it is sensory, the centre for sound images; the fibrae propriae joining in the insular cortex constitute the connecting psychic reflex arc. The first temporal convolution would thus have to be considered the central ending of the acoustic nerve, the first frontal convolution (including Broca's area) the central ending of the speech muscle nerves.
>
> (Wernicke, 1874: 18–19)



DER

APHASISCHE SYMPTOMENCOMPLEX.

EINE PSYCHOLOGISCHE STUDIE AUF ANATOMISCHER BASIS

VON

Dr. C. WERNICKE,

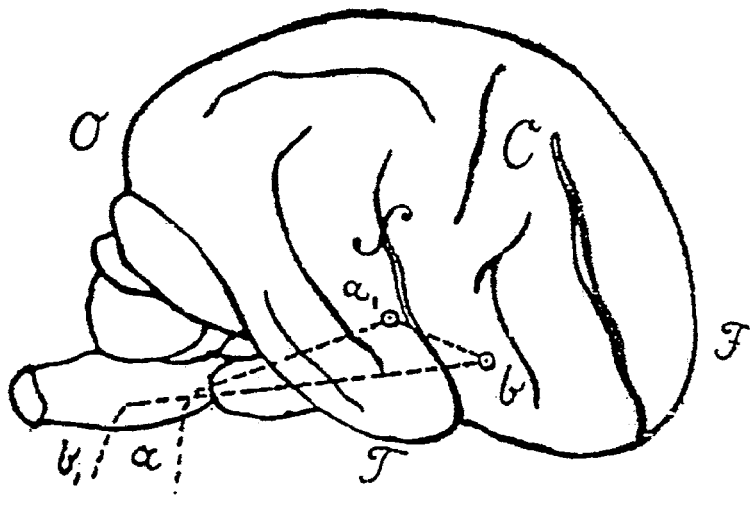
ASSISTENZARZT AN DER IRRENSTATION DES ALLERHEILIGEN-HOSPITALES ZU BRESLAU.

BRESLAU.
MAX COHN & WEIGERT.
1874.

Figure 4.3 Carl Wernicke's *The Symptom-Complex of Aphasia* was published in 1874. This book laid the foundations for the classic theory of aphasia.

This is illustrated in Figure 4.4 where Wernicke's simple **sensory-motor language model of the 'psychic reflex arc'** is represented. Strangely, the model is apparently drawn onto the right hemisphere of a primate brain (according to Poeck, 1998: 6)! For Wernicke, apparently, issues of precise anatomy and lateralisation seem unimportant and his diagrams are schematic, and became even more schematic in his later work.

Language was conveniently divided by this motor/sensory dichotomy. Words are stored as two types of memory images and **each word** is represented as (motor) **movement image** and (sensory) **sound image (memory image)**. Wernicke postulated two centres, one for movement images (**b**) and one for memory images ($\mathbf{a_1}$) and these become the two major centres of the model, now named Broca's and Wernicke's. The **reflex arc's** main contribution is in



F:	Frontal lobe	C:	Central fissure
O:	Occipital lobe	S:	Sylvian fissure
T:	Temporal lobe		
a:	Entry of the acoustic nerve into the medulla oblongata		
a_1:	Central ending of the acoustic nerve, center for memory images		
b:	center for movement images		
b_1:	Articulatory muscles		
a a_1:	Acoustic nerve		
a_1 b:	Association fibres running through the insular cortex		
b b_1:	Pathway of the articulatory motor nerves		

Figure 4.4 Wernicke's (1874) reflex arc model of language processing.

language acquisition (from **a** to **b**): to connect sensory memory images with motor movement images by creating associations (this is how Wernicke explained language acquisition). In human adults the reflex arc takes care of repetition. The route $\mathbf{aa_1}$ constitutes the acoustic nerve across which sound impressions are led to the centre **a** and route $\mathbf{bb_1}$ represents the pathways of the articulatory motor nerves. Breakdown in language, aphasia, is also understood on the basis of this very simple model.

As a precondition Wernicke pointed out that thinking and language are independent processes (in opposition to Bastian's position) and the sound images that are necessary for understanding are considered a part of language and no longer a part of thinking (as proposed by Lazarus). Language comprehension as a function can thus be selectively disturbed (disassociated).

Aphasia results from 'a disruption of the psychic reflex arcs used in the normal speech process' (Wernicke, 1874: 69) and 'aphasia can be caused by any disruption of the pathway aa_1 bb_1. But the clinical picture of the same will have to be different depending on the section of the pathway that is affected by the disruptions' (Wernicke, 1874: 19). Different aphasic profiles are therefore to be expected and the determining variable is the localisation of the damage.

If pathway $\mathbf{aa_1}$ is disrupted, there will be non-aphasic deafness ('simple deafness without any trace of aphasia'). If 'a_1, the location of the sound image itself, is affected by the destruction [. . .] a total loss of all sound images with totally preserved hearing [. . .] is observed in aphasia' (Wernicke, 1874: 21). Wernicke (1874: 22) suspected that the sensory nerves disperse themselves further and that

> thus the part of the central acoustic propagation can be destroyed that contains word sounds, while every sound or every musical tone still remains perceptible. [. . .] The ill person is hence neither able to repeat the spoken word – because that is the actual function of pathway aa_1 bb_1 – nor to understand the spoken word.

Wernicke called this disorder **sensory aphasia**.

The memory images of the *sensory language centre* are lost, not the concepts themselves. The connection $\mathbf{a_1b}$ has no direct function; but it is important to know that Wernicke (like Schmidt before him) supposed that memory images are a necessary precondition for language, and that this is explained through language acquisition: 'Because with normal speaking, as is easily understandable from the genesis of speech, the sound image seems to always be unconsciously innervated too, quasi hallucinating and thereby exercising a continual correction on the original process of motor images' (Wernicke, 1874: 23). From this it follows that the patient with sensory aphasia must also have problems with speaking: 'Apart from the lack of understanding, the ill person also has aphasic symptoms while speaking, caused by the lack of this unconscious correction exercised by the sound image. They consist of the easy confusion of words' (Wernicke, 1874: 23).

Although Wernicke is often considered the originator of sensory aphasia (e.g. Wilkins & Brody, 1970), as we have seen, there were earlier descriptions of comprehension disorders before Wernicke. Meyer (1974: 570–571) mentions Trousseau, Bastian, Moxon, Broadbent, Meynert, Schmidt, but he rightly states: 'It was Wernicke, however, who [. . .] made an almost immediate impact on the medical world, so that his name became eponymous with sensory aphasia.' Geschwind (1966: 4) also states 'Nevertheless, it was Wernicke's paper, not the observations of Bastian and Schmidt, which brought home to the neurological world the existence of the sensory varieties of aphasia.'

As Wernicke (1874: 25) suspects that written language acquisition is secondary to spoken language, in 'cases of [. . .] sensory aphasia [. . .] also an [. . .] agraphia can be expected.' In practised readers, for whom reading has disconnected itself from the perception of sound, problems with reading comprehension will rarely occur, but probably in an unpractised reader 'who even only [understands] what has been written when he hears himself speak, but both will again be as aphasic while reading out as in spontaneous speech.' With this it was clear that Wernicke understood aphasic disorders as affecting all modalities.

Conduction aphasia results from a disruption of the association fibres $\mathbf{a_1b}$, 'which connect the sound image with the corresponding motor image'. The patient understands everything and he can also speak, 'but the choice of words is disturbed in similar fashion to the form just described' (Wernicke, 1874: 26), sensory aphasia, because the sound images cannot exercise their monitoring and correcting function. Because of this, the patient has good error awareness, however, because he can understand what he says to himself and can compare this with what he originally intended to say.

The phenomena in **motor aphasia** are different (Wernicke's term for what later was called Broca's aphasia). In lesions of the frontal lobe

> caused by the destruction of the speech motor images of b, everything is understood, but the afflicted has suddenly become mute or has just a few simple words left at his disposal. The latter are mostly needed to indicate all sorts of things, but this does not happen from ignorance of their meaning, but instead from the need to react to questioning with any sound at all. [. . .] The majority of all cases of aphasia described until now, namely Broca's, belong here.
>
> (Wernicke, 1874: 31)

Finally, a disruption of pathway $\mathbf{bb_1}$ 'must have exactly the same effect as the destruction of the corresponding cortical areas themselves, the same motor aphasia must result', but it is hardly to be expected that there could be lesions so small 'that [they] thus could produce pure aphasia without any other paralysis' (Wernicke, 1874: 31). Consequently, Wernicke had 'no doubt that, between aphasia and alalia, there is [. . .] only a gradual, and no qualitative difference' (Wernicke, 1874: 32).

Wernicke substantiated his *theory of aphasia* with ten case descriptions, among which are three with sensory aphasia (e.g., the cases of Adam and Rother), three with conduction aphasia (Beckmann, Kuschkel, Zwettels), as well as five other cases (motor, mixed pathology). Of these ten, Wernicke had postmortem information on lesion localisation for only three for the first edition of his book (he added a fourth for the edition republished in 1893). The strength of Wernicke's work lies in the theory and not in the case descriptions. Marx (1966: 341) even writes that Wernicke 'refused to see that none of his cited cases supported his theoretical formulations', and Steinthal's reproach against the 'Messrs. Physicians' applies particularly to Wernicke, namely that the observations are extremely superficial and show little understanding of language. However, Wernicke (1874: 32) was almost prophetic in his justification of his few case descriptions in view of his theoretical approach: 'I believe it suffices to have directed the attention towards it in order to soon witness the publication of many quite relevant patient histories with autopsy findings.'

Although it had been claimed since Gall that aphasia does not impair intelligence and thinking (Bouillaud and Broca, for instance), and that they hence are not disorders of thinking or intelligence, this view only gained proper ground with Wernicke (1874: 35): 'Nothing worse could happen to the theory of aphasia than to conceive the disorders of intelligence occurring at the same time [. . .] as essentially belonging to the clinical picture.' Following Wernicke, asymbolia as Finkelnburg presented it, and the symptoms included in it, were equated with a *defect of intelligence* and therefore not aphasia.

Wernicke (1874) summarised his contribution as follows:

> The proposed theory of aphasia can subsume the so varied clinical pictures of aphasia. This diversity itself, which gave each new observer new puzzles to solve until now, will no longer be so striking, it will even be calculable following the laws of combination. But it is specific to all of them that a disruption of the psychic reflex arc used for the normal speech process is at their basis. Thereby, the reader has gained a clear definition of the term of aphasia.
>
> (Wernicke, 1874: 69)

With Wernicke's work the foundation is finally laid for the **classical model of aphasia**, in the last third of the nineteenth century:

- aphasias are the consequence of lesions of the language centres and/or their connections, input and output pathways
- aphasias are not disorders of intelligence
- they occur in symptom groupings; essential syndromes are identified (e.g., motor, sensory, conduction aphasia)
- the syndromes depend on the localisation of the lesion
- aphasias are disorders of the normal flow of speech/language processing (in the reflex arc model).

He also considered pure cases of aphasia as improbable and predicted that mixed forms would be observed.

This model is comprehensible and testable if one accepts association psychology. It strictly separated thinking and speaking, accounted only for single words and it regarded Meynert's fibre theory as correct. Wernicke (1874: 4) wanted to localise only the most elementary mental functions because, 'Only the most elementary psychic functions can be attributed to specific places of the cerebral cortex.' This is really as far as Wernicke's localisation goes, and he did not see higher cognitive functions as represented in circumscribed brain areas, but in mosaic-like association systems. While Wernicke was not an extreme localisationist, extreme localisationism took nourishment from his theory.

Even if Meynert's and Schmidt's essential ideas were adopted or formulated before, as for instance by Bastian, it was Wernicke's work that had such an enormous impact on the future development of aphasia. Mathews, Obler, and Albert (1994: 442) note that: 'It is Wernicke's 1874 monograph, however, that so powerfully determined a theoretical framework for aphasia that it set the tone of aphasia research for the next half century.' This is partly due to the expansion in ideas about localisation that emerged in the spirit of time, but also due to another fact of Wernicke's personality. As Geschwind (1966: 7) writes: 'The final reason for Wernicke's importance extends beyond his work on aphasia. He had the ability, not common to all distinguished figures, of developing great students.' Three of Wernicke's students who argued in favour of his approach were Bonhoeffer, Heilbronner, and Kleist, whose contributions are discussed later.

Beyond the single word and the anatomy: Adolf Kussmaul

Just three years later in 1877 Adolf Kussmaul (1822–1902) published a comprehensive work with the title *Disturbances of Language: An Attempt in the Pathology of Language* and developed a model of aphasia that went beyond the single word and was unconstrained by considerations of localisation. Kussmaul differentiated between disorders of articulation (dysarthria) and 'cortical language disturbances', which he subdivided into dysphasias or aphasias and dyslogias or dysphrasias. Dysphasias or aphasias are disorders of the 'organ of language', causing language processing problems, whereas dyslogias or dysphrasias are caused by some reduction of intelligence.

Kussmaul (1877) emphasised

> that a large number of symptoms of very varying natures are subsumed under the term aphasia in the clinical sense. One simply constructed a uniform clinical picture whose features were gathered without deeper understanding of the elementary processes that constitute language, gradually and from numerous individual observations that diverge from one another in many cases. – Mainly, the following dysphasic disorders

> are collected under the general name of aphasia: 1) *atactic aphasia* or the failure of motor coordination of words. 2) *amnestic aphasia* or the inability to remember words as acoustic sound complexes. 3) *word deafness* or the inability to understand words as well as in the past while retaining good hearing and sufficiently retained intelligence. 4) *paraphasia* or the inability to connect word images correctly with their concepts, so that, instead of the one with the corresponding meaning, wrong or entirely incomprehensible word constructs appear. 5) *agrammatism* and *acataphasia* or the inability to form words grammatically and to syntactically order them into a sentence.
>
> (Kussmaul, 1877: 154–155)

As we can see, Kussmaul distinguished between different aphasic manifestations at different linguistic levels. Importantly, he also included (with reference to Steinthal) the sentence level as well as the lexical level, and introduced the term **agrammatism** to describe impairments in grammatical formulation. Two features are essential in the sentence: word inflection (meaning morphology) and word position (syntax). Kussmaul (1877: 195–199) called the impairments in acataphasia '*dysgrammatical disorders or pathological agrammatism*', and he differentiates three types:

- **aphasic acataphasia**, caused by problems at the lexical level where words are omitted, and which leads to sentence truncation and sentence fragments
- **grammatical acataphasia**, for example the utterance 'Toni flowers got, guardian come, Toni hit', which corresponds to the modern concept of **telegraphic speech** (speech produced as in a telegraph or telegram, which nowadays is seen as a feature of agrammatism (sometimes the terms agrammatism and telegraphic speech are used synonymously, although erroneously))
- **syntactic acataphasia**, for example (translated from the German) 'I have as 1869 been a coachman and have a coach which me as I have serve the horses me as coachman with the servant been.'

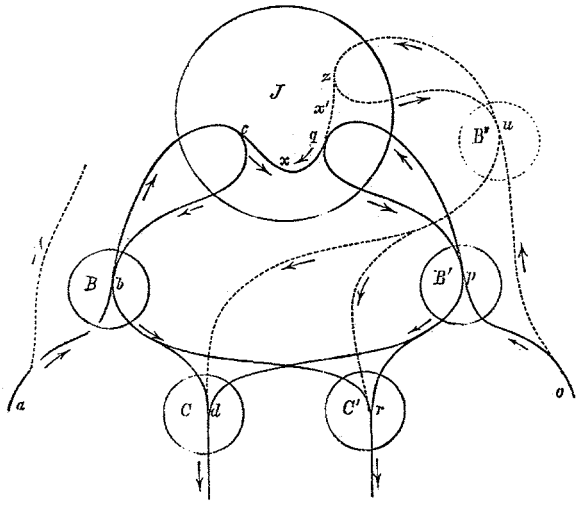
What Kussmaul identified for the first time as 'syntactic acataphasia', we now call **paragrammatism**. So not only did Kussmaul describe syntactic disorders and establish them as aphasic symptoms, but also he classified them in a way that still makes sense from a modern perspective.

Kussmaul also distinguished agraphias and paragraphias, which he saw as analogous to aphasias. The concept of word blindness was also used by him and he described a 'disorder of sign language', which he called 'dysmimia' (Kussmaul, 1877: 156).

As in modern cognitive neuropsychology, Kussmaul proceeded in a very **model-oriented** way and without reference to brain anatomy. 'Kussmaul understood, that a functional model can, indeed ought to, be developed without

being constrained by considerations of localisation, as long as the neurological foundations of language remain underspecified' (Jarema, 1993: 495).

For word processing Kussmaul (1877: 184ff.) developed a model unconstrained by anatomy (see Figure 4.5) in which the understanding, production, writing, and reading of words are represented in four centres (in accordance with the four modalities) and a centre for conceptions. These centres are connected to one another and to the senses by routes.



J:	Ideational centre
B:	Sound image centre
B':	Optical image centre
C:	Centre for coordination of sound images and motor images
C':	Centre for writing
a:	Acoustic nerve
o:	Optic nerve
abcbd:	Acoustic motor route for spoken language
opqpr:	Optic motor route for written language
abd:	Route for imitative speech of children
opr:	Route for the copying without comprehension
cbd:	Route for utterance generation
qpr:	Route for the writing of ideas
cxq:	Transfer from spoken to written language
abr:	Writing to dictation without comprehension
opd:	Reading aloud without comprehension
abcbr:	Writing to dictation with comprehension
opqpd:	Reading aloud with comprehension

Figure 4.5 'Schematic diagram of the centres and pathways of language' following Adolf Kussmaul (from Kussmaul, 1877: 182).

In atactic aphasia the centre for the coordination of sound–movements (*Lautbewegungen*, which roughly corresponds to Wernicke's motor images) (**C**) is impaired, so the patient understands everything but can neither spontaneously express him or herself nor repeat or read aloud. If route **bd** is damaged, then the patient cannot speak spontaneously, but can still read aloud. In amnestic aphasia, route **cb** is 'only temporarily disabled'. If no input comes from the centre for conceptions (**J**), prompting can be effective (heard across **a**; word image activated in **B**) or by written language (from **B′** across **C**). In word deafness route **abc** is impaired.

Because much of this model is reminiscent of Wernicke's model, the differences should be made clear. Unlike Wernicke, Kussmaul's centres have no anatomical significance: 'Wernicke made the mistake of plotting the centres in specific areas of the brain. The localisation of elementary functions of language is not mature enough for this' (Kussmaul, 1877: 183). In addition, Kussmaul points out that his is a model of *word* processing only and not language in general, whereas Wernicke displayed an incomplete understanding of language and did not even comment on the linguistic limitations of his model. Kussmaul clearly pointed to the heterogeneity of aphasic phenomena and a large range of aphasiological terminology (paraphasia, agrammatism, etc.) originated in his work – more or less as we use them nowadays. Finally, it is interesting that Kussmaul did not relate aphasic phenomena to brain-damaged patients only. In hysterias and intense emotion, according to him, a 'functional aphasia' can arise (Kussmaul, 1877: 200f.).

The Wernicke–Lichtheim model

In a long career, **Ludwig Lichtheim** (1845–1928) published just one article on aphasia, but it had such an impact that it was published in English translation in the new journal *Brain* in the same year it appeared in German, although as a shortened and slightly modified version. In his article he 'tries to determine the necessary innervation pathways for language, the functions closely related to it and their connections, and to determine the locations of the latter in the brain' (Lichtheim, 1885b: 204–205). From Wernicke, Lichtheim adopted localisation and Wernicke's model, and from 'the excellent work by Kussmaul' (Lichtheim, 1885b: 206) he took the terminology (e.g., paraphasia) and features of Kussmaul's model.

Figure 4.6 shows Lichtheim's first two famous models. With the first model (known as 'Lichtheim's House' or 'The Wernicke–Lichtheim House', for obvious reasons), Lichtheim (1885b: 208) wanted to show 'that the assumed connections allow for seven different interruptions'. The numbers relate to the location of the interruption and correspond to the following numbering. Since writing and reading problems also occur with aphasia, they were integrated into the syndromes, thereby expanding the model by two centres (O, E) writing and reading. Lichtheim also developed his own terminology for his

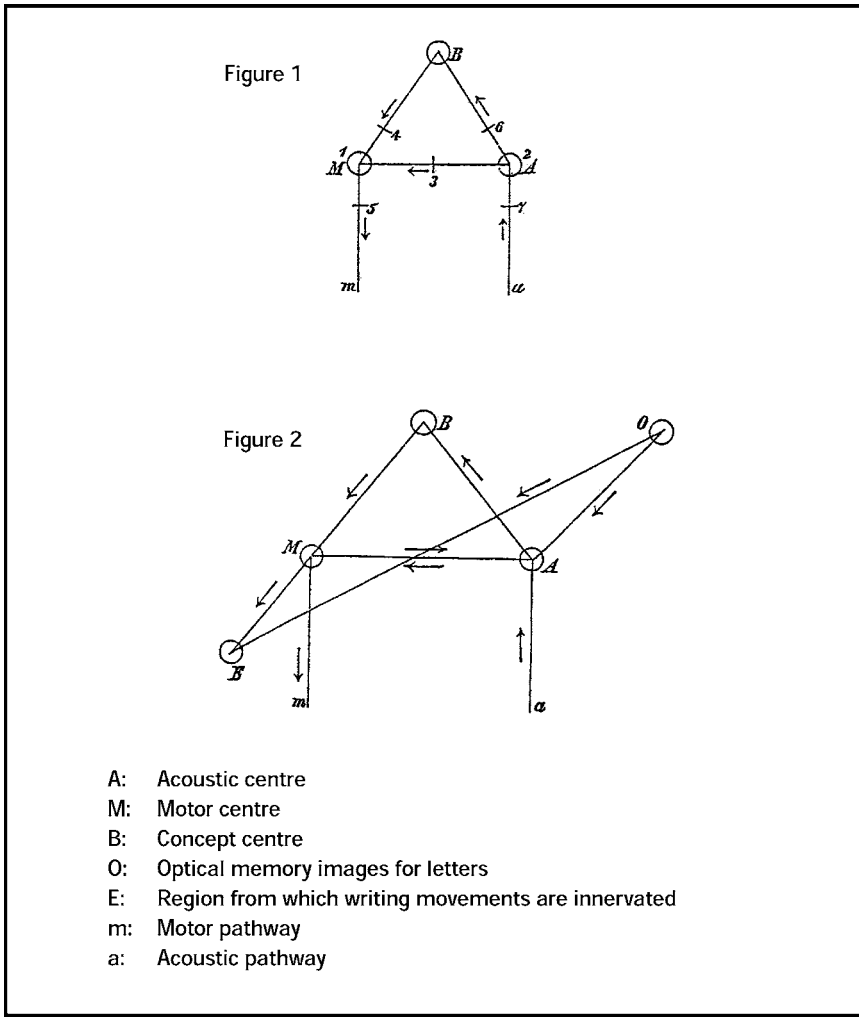


Figure 4.6 Lichtheim's (1885b: 207–208) language processing models.

classification system, a terminology later dismissed by Wernicke and the rest of the aphasiological community.

Figure 4.7 shows a compilation of disorders reflecting the respective interruptions or lesions to the model. Central aphasia corresponds to motor or Broca's aphasia and is caused by a lesion in the motor centre **M**. **Central language deafness** is caused by a disorder of the auditory centre **A** and corresponds to Wernicke's sensory aphasia. An interruption of the connection **AM** causes Wernicke's conduction aphasia, which is called **conduction paraphasia** by Lichtheim. If **MB** is interrupted, a 'variation of motor aphasia' is the result (Lichtheim, 1885b: 222), **central conduction aphasia**, in which

Interruption/ lesion	Central aphasia (M) 1	Central language deafness (A) 2	Conduction paraphasia (MA) 4	Central conduction aphasia (BM) 4	Peripheral conduction aphasia (Mm) 5	Central conduction language deafness (AB) 6	Peripheral conduction language deafness (AA) 7
Spontaneous speech BMABMm	-	+P	+P	-	-	+P	+
Repetition aAMm	-	-	+P	+	-	+	-
Audit. understanding aAB	+	-	+	+	+	-	-
Reading comprehension OAB	+	-	+	+	+	-	+
Reading aloud OAMm	-	-	+P	+	-	+	+
Writing BMAE	-	+P	+P	-	+	+P	+
Writing to dictation aABMAE	-	-	+P	+	+	+	-
Copying OE	+	+	+	+	+	+	+

P: paraphasic
+: present
–: strongly impaired

Figure 4.7 Summary of symptoms in the aphasia forms 'calculated' by Lichtheim (1885b), following Cegelski and Dustmann (1999: 74).

repeating, writing dictation, and reading aloud are possible. If **Mm** is interrupted, **peripheral conduction aphasia** results 'in which merely the ability to speak has been lost while the patients have retained the ability to express their thoughts by writing' (Lichtheim, 1885b: 224–225). Lesion 6 (**AB**) leads to **central conduction language deafness**. The interruption of connection **Aa** 'does not belong with the aphasic disorders because language is entirely intact with it' (Lichtheim, 1885b: 237). Lichtheim called this disorder **peripheral conduction language deafness** or **word deafness**.

Lichtheim (1885b) emphasised several times

> that the seven discussed forms only have simple interruptions at their basis, but there is no doubt that several pathways can be interrupted simultaneously. We possess an incontestable piece of evidence for the actual existence of such combined forms in *total aphasia*, i.e. in those forms of aphasia in which the total inability to speak [. . .] and language deafness exist simultaneously.
>
> (Lichtheim, 1885b: 243)

Lichtheim's identification of such combined forms is often overlooked. He supposed – like Wernicke – that the pure forms occur rather rarely. Lichtheim (1885b: 245) also noted 'that the individual components of the symptom complexes degenerate at very different speeds from one another.'

Lichtheim (1885b: 256) was aware of the hypothetical status of his classification: 'Also some will resent that the nomenclature relies on an interpretation of the disorders that is, after all, hypothetical.' Unlike Kussmaul, Lichtheim (1885b: 256–257) specified his model anatomically. The motor-image centre is localised in the '[part. . .] of the lowermost left frontal convolution lying against the Sylvian Fossa' and the sound-image centre in 'the temporal convolution lying on the opposite side.' The connection between the two centres goes through the insula or directly adjacent regions. The centre for conceptions represents the exception: 'My view tends to assume [. . .] that the concept formation is not linked to a location in the brain but is a common function of the entirety of sensory areas instead.'

Lichtheim thought that it might be difficult to determine an isolated disorder of pathways **AB** and **BM** if **B** were not an independent, anatomically fixed centre. He dismissed this objection by assuming that the many connections between **B** and **A** or **M** are bundled shortly before **A** and **M**, so that localisation should be sought at the location of the bundling. Lichtheim (1885b) supposed

> the lesion which causes a central conduction aphasia to be in the white matter sheath, namely closely below the beginning part of the lowermost frontal convolution. Analogously, the anatomical localisation of central conduction language deafness is to be assumed in the white matter sheath close to the first temporal convolution.
>
> (Lichtheim, 1885b: 258)

The localisation of pathways **Mm** and **aA** was not ultimately determined, but is discussed in detail.

Lichtheim's model became established, though his terminology did not, and for this, Wernicke (1885–1886) is responsible in a work where Wernicke discussed developments subsequent to his 1874 'symptom-complex of aphasia' book. He especially liked Lichtheim's approach, but the terminology did not please him. Based on Lichtheim's model, which he drew a little differently, Wernicke named his seven forms of aphasia (Figure 4.8) with reference to anatomical locations.

Disregarding written language, **Wernicke's classification system** can be summarised as follows (Wernicke, 1885–1886). **Cortical sensory aphasia** is characterised by comprehension problems and problems with repetition. Spontaneous speech is less impaired but characterised by paraphasias. Repetition and comprehension are also impaired with **subcortical sensory aphasia**, but with totally preserved spontaneous speech. Paraphasias in spontaneous speech, comprehension problems and preserved repetition characterise **transcortical sensory** aphasia. **Cortical motor aphasia** shows limited spontaneous speech and poor repetition, with intact comprehension. Similarly, patients with **subcortical motor aphasia** can still provide phonological information about words (e.g., number of syllables) because the speech motor images are still intact. In **transcortical motor aphasia** spontaneous speech is paraphasic but comprehension and repetition are intact. In **conduction aphasia** spontaneous speech and repetition are paraphasic, but comprehension is preserved. Finally, Wernicke identified **amnestic aphasia**, which he considered to be the result of a memory problem, and consequently he separated it from aphasias proper. As the centres are linked by connections, the model is

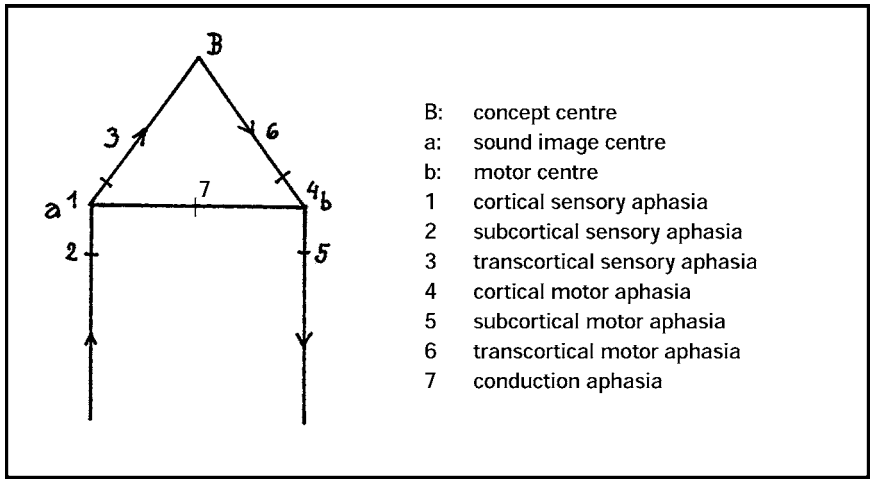


Figure 4.8 Wernicke's (1885–1886) model with the respective forms of aphasia caused by lesions in the marked locations.

sometimes referred to as the **classic connectionist model** (but should not be confused with modern connectionism, discussed in Chapter 8).

The **Wernicke–Lichtheim model** thus became established, although it has never been without its critics (Caplan, 1987: 47ff.; Laubstein, 1993; Von Block, 1992). Kleist (1970: 107f.) commented that with 'this work, the edifice of theory of aphasias in its major aspects was completed'. The model became the archetype for the term 'diagram makers', which was Henry Head's derogatory description of those aphasiologists whose models tended towards the diagrammatic. But the model would dominate the end of the nineteenth century. However, one should distinguish those diagram makers who anchor their model anatomically (e.g., Lichtheim) from those who do not (e.g., Kussmaul).

Wernicke died in 1905 following a bicycle accident, roughly a year after he had accepted a professorship at the University of Halle, where he also became head of a large clinic. A few years earlier he had married, and it was related by his assistants that for the first time he was something close to being happy and satisfied with life. Not yet 60 years old and close to his peak, he may have had a significant contribution yet to make, but his early death cut that possibility tragically short.

Aphasiology at the end of the nineteenth century

The Wernicke–Lichtheim model became the hub around which the classical doctrine of aphasia took shape, and heated discussions occurred. On one side were the localisationists of course (for instance Bastian and Charcot) and their approach enjoyed great popularity. On the other the Wernicke–Lichtheim model was sufficiently explicit to provide grounds for counter-positions (for instance Freud's).

A strengthening of the German Empire was developing and a military defeat of the French by the Germans in 1870–1871 resulted in the Germans marching into Paris. Because of this the French scientific community became rather closed to the revolution taking place in German aphasiology (Wernicke, Kussmaul, Lichtheim) and French aphasiology remained committed to Broca's mid-1860s findings (Bernard, 1889; Gelfand, 1999: 39f). Jean-Martin Charcot was a leading advocate of a reactively patriotic competition with German science (Gelfand, 1999) and it was only with Charcot and his pupils (for instance, Bernard) that aphasia became an important topic in Paris again, although there was significant reluctance to accept that things had moved on since Broca and the revolution in aphasiological thought coming from Germany was ignored. In England Hughlings Jackson published more on his evolutionary approach to aphasiology and was hardly influenced by the localisation debates going on in other parts of Europe, although as co-editor of the new journal *Brain*, he published Lichtheim's work in English in 1885. Bateman's work *On Aphasia, or Loss of Speech* appeared in 1890 in its second edition, in which Charcot, Kussmaul and others were included. But Bateman maintained his clinically oriented perspective, which was apposed to

classifications and localisation. At the end of the century an important contribution was made by Bastian, whose summary of his 30-year long interest in aphasia represented a culminating point in connectionism.

Another important critic of the Wernicke–Lichtheim model, and of connectionism in general, was Sigmund Freud from Vienna, who published an important monograph on aphasia. His contribution is discussed later in this chapter.

Jean-Martin Charcot's bell diagram

In the second half of the nineteenth century Paris developed into a centre for neurology, due not least to the activities and influence of **Jean-Martin Charcot** (1825–1893) (Goetz, Bonduelle, & Gelfand, 1995) at the *Hospice de la Salpêtrière*, also holder of the chair for nervous diseases, created for him at the University of Paris in 1882 (Brais, 1993; Gasser, 1994). Charcot was interested in localisation throughout his entire career, and a small but important part of his work was concerned with aphasia. In a series of lectures (in 1883 and 1884) with the title *On the Different Forms of Aphasia* (Charcot, 1884; Ballet, 1886; Bernard, 1885) he developed his famous **bell diagram** (Figure 4.9) (a reconstruction of it with current terminology can be found in Lecours (1993: 470). The model is essentially similar to Kussmaul's and was meant to permit a better understanding of normal and pathological language processing (Bernard, 1889: 36).

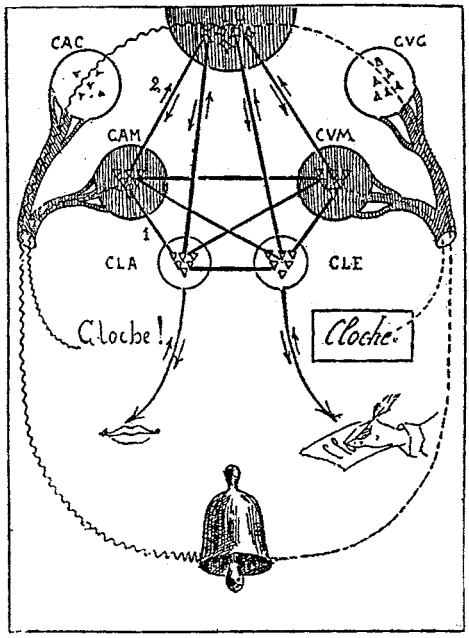
In this diagram, four centres for memory images (for speech, language, writing, reading) are attributed to an association centre. These are in turn connected to the outside world by general auditory and visual centres. This classification resulted from Charcot's idea of the nature of words, which traces back directly to William Hartley (Gelfand, 1999: 37).

Charcot (1890, in Rosenfield, 1992: 105) considered the word as 'a complex structure which consists of at least four basic elements: the acoustic memory image, the visual memory image, and two motor memory images, one for speaking (articulation) and one for writing.' Charcot derived these forms of aphasia from his model:

> When someone does not understand written words, one refers to this as verbal blindness or visual verbal amnesia, and when one does not understand the sound of spoken words, one calls this verbal deafness or acoustic verbal amnesia. In similar fashion, and following the same principles, one can say that someone suffers from motor verbal amnesia when the motor word images have been lost.
>
> (Charcot, 1890 in Rosenfield, 1992: 105)

To this he added agraphia in the case of loss of memory of images for writing.

On the basis of Ribot's (1881) memory theory, Charcot, like so many of his predecessors, thus saw aphasia as a **memory disorder**, with memory divided



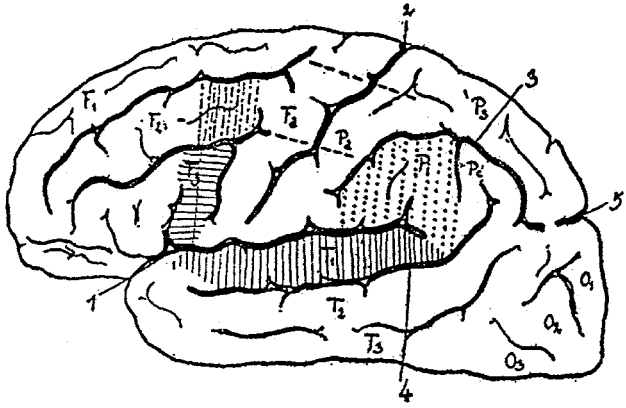
Cloche 'Bell'

IC	*centre d'idéation* Association centre
CAC	*centre auditif commun* General auditory centre
CAM	*centre auditif des mots* Hearing centre for words
CLA	*centre de langage articulé* Centre for articulated speech
CVC	*centre visuel commun* General visual centre
CVM	*centre visuel des mots* Visual centre for words
CLE	*centre du langage écrit* Centre for writing

Figure 4.9 Bell diagram of Jean-Martin Charcot (from Bernard, 1889: 37ff.).

into different subsystems in which he also supposed submemories for language, for understanding (*la mémoire auditive*), writing (*la mémoire graphique*), speaking and reading (Gelfand, 1999: 37ff.). The individual centres are linked to one another by many connections.

He attempted to anatomically anchor the localisation of aphasic disorders (Figure 4.10) and thought, after Broca, that motor aphasia was caused by a



1		*scissure de Silvius* Sylvian Fissure (lateral fissure)
2		*sillon de Rolando* Rolandic Fissure (central fissure)
F1/F2/F3		*première/deuxième/troisième circonvolution frontale* first/second/third frontal gyri
T1/T2/T3		*première/deuxième/troisième circonvolution temporal* first/second/third temporal gyri
01/02/03		*première/deuxième/troisième circonvolution occipital* first/second/third occipital gyri
Ps		*lobule pariétal supérieur* superior parietal lobe
Pi		*lobule pariétal inférieur* inferior parietal lobe
F2		*agraphia* agraphia
F3		*aphasia motrice (type Bouillaud-Broca)* motor aphasia
Tj1		*surdité verbale / aphasia sensorielle* word deafness / sensory aphasia
Pi		*cécité verbale* word blindness

Figure 4.10 The localisation of aphasias according to Charcot (from Bernard, 1889: 39).

lesion of the third frontal gyrus and suspected the cause of agraphia to lie in the second frontal gyrus. Word deafness was caused by a lesion in the first temporal gyrus, and **word blindness** resulted from a lesion to the lower parietal gyrus.

The two diagrams (Figures 4.9 and 4.10) are then summarised in a schema

(Figure 4.11; Cole & Cole, 1971: 15–22), in which the memory centres are directly plotted on to a left hemisphere. The association centre, however, is not included (like in Lichtheim), because, while Charcot localised the language memory centres, he also tended towards a purely psychological approach (Goetz et al., 1995: 133f.). This diagram became famous through

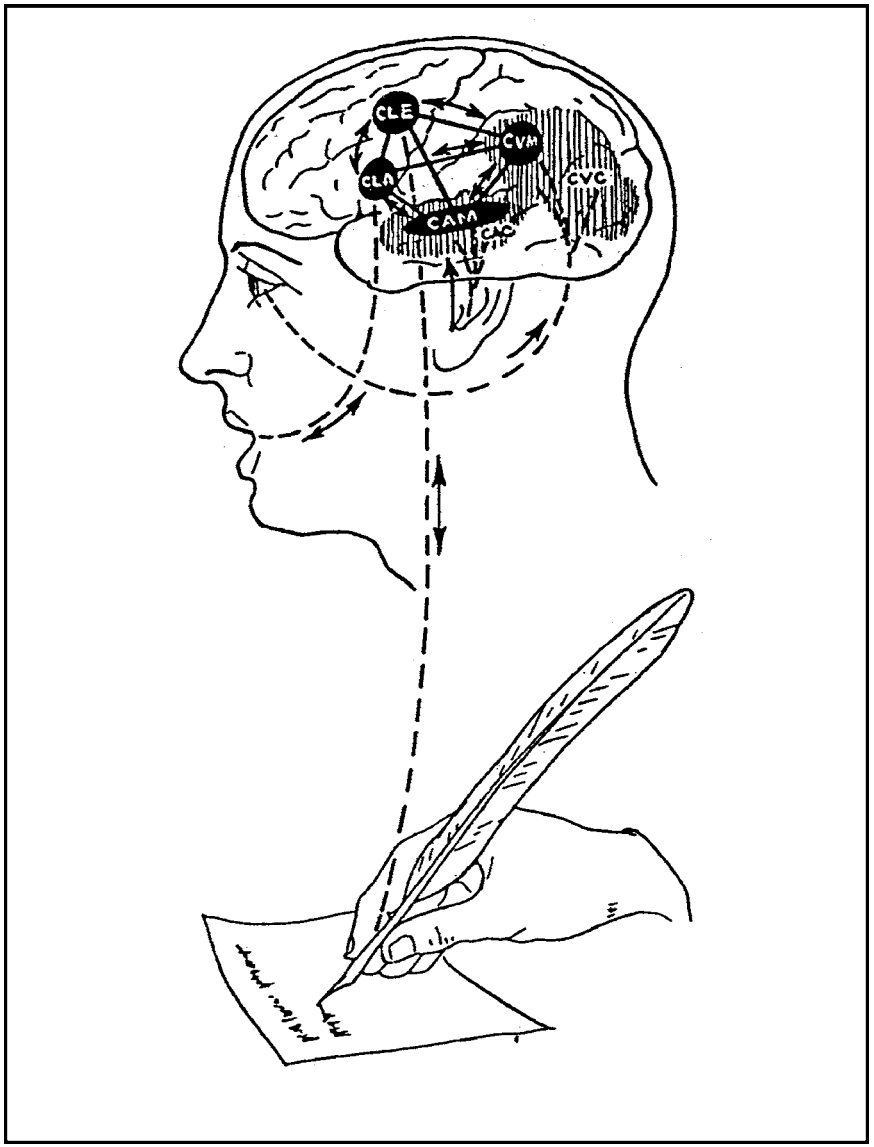


Figure 4.11 The localisation of the different language memory centres in the left hemisphere according to Charcot (adapted from Cole & Cole, 1971: 19). (For an explanation of the abbreviations, see Figure 4.9.)

the work of the young Pierre Marie, who joined Charcot at the Salpêtrière in 1885 and became one of his most famous pupils, although Sigmund Freud, who was to become far more famous than Marie for his work on the development of psychoanalysis, also spent time with Charcot in 1885. With the interest and work of Charcot, aphasia again became a topic of intense discussion in Paris.

Jules Dejerine on alexia with and without agraphia

The Swiss **Jules Joseph Dejerine** (1849–1917) was also a student of Charcot working in Paris (Bub, Arguin, & Lecours, 1993: 532ff.; Renier, 1994; Zabriskie, 1953), where he eventually became *Professeur de clinique des maladies du système nerveux* in the Faculty of Medicine in 1910. While he described a classification system of acquired speech and language disorders, he has become important mainly through two case descriptions of isolated writing and reading disorders. Dejerine (1891) described a 63-year-old man with **word blindness (alexia) and total agraphia** and (Dejerine, 1892) a 61-year-old educated woman with **word blindness without agraphia**, who could write spontaneously and to dictation, and had no difficulties with spontaneous speaking (Bub et al., 1993; Hanley & Kay, 2003).

The autopsies produced the following results: 'The first form is caused by a lesion in the language area (angular gyrus of the left side), the second form by a lesion in the general language area that separates it from the angular gyrus' (Dejerine, 1892, in Renier, 1994: 215). Dejerine suspected that the visual word images are stored in the angular gyrus, which he supposed is necessary for reading as well as for writing. With a lesion of the angular gyrus alexia and agraphia would therefore result. If only the access to the visual word images was impaired, then there would be an inability to write, but there would be an (isolated) alexia.

Three years later, Dejerine and Mirallié described a further form of alexia as it commonly occurs in motor aphasia. This 'third alexia' (Henderson, 1984) is explained with reference to Dejerine's **language zone** (*zone du langage*; see Figure 4.12). Within the language zone, Dejerine suggested, are specialised cortical regions – Broca's area, Wernicke's area and the angular gyrus, respectively responsible for production, auditory comprehension and written language comprehension. But these special regions are functionally integrated into the language zone. Interruptions of the connecting (subcortical) pathways lead to isolated phenomena. Cortical lesions of the language zone lead to a disorder of 'inner speech' and create supramodal disorders such as alexia in motor aphasia. Dejerine and Mirallié clearly differentiated in 1895 between word and sentence comprehension in written language comprehension and emphasised a role for function words (Henderson, 1984: 431).

Dejerine's model is clearly different from Charcot's. For one thing the functional architecture of language processing is structured differently, and for another the anatomical localisation is not at all congruent.

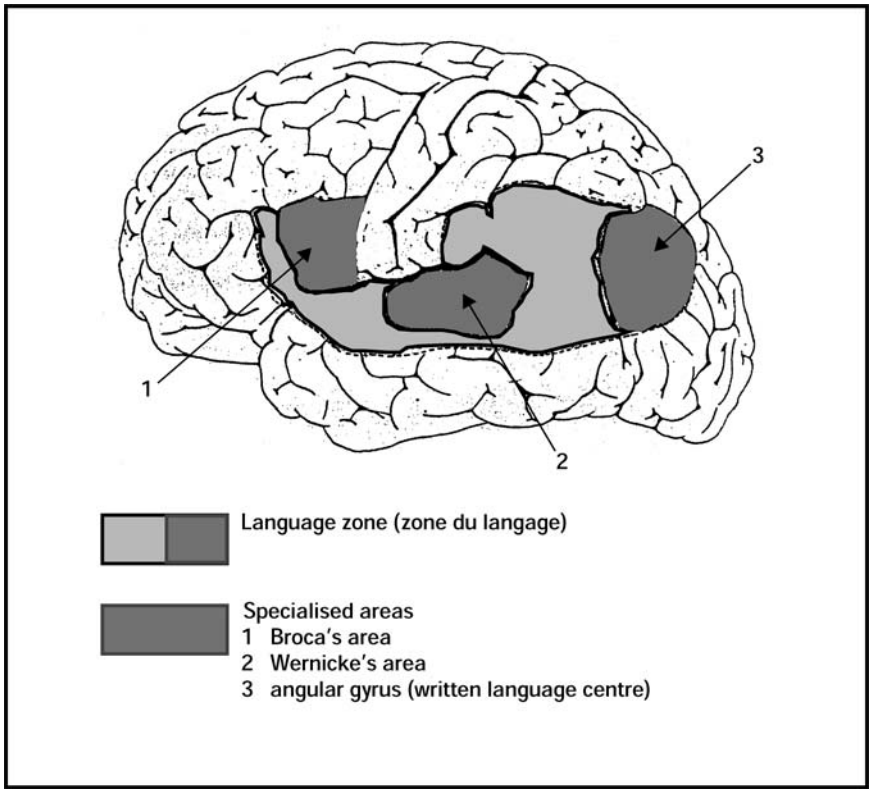


Figure 4.12 The language zone with specialised cortical zones following Jules Dejerine (1891).

Albert Pitres: amnesic aphasia and aphasia in polyglots

Albert Pitres (1848–1928) is well known in aphasiology from his early championing of **amnesic aphasia** and his book on aphasia in bilingual and multilingual speakers. The concept of **amnesic aphasia** was constantly under discussion from the 1860s and Pitres tried to establish it as an independent type of aphasia. He wrote articles and a book on the condition (Pitres, 1898). One case he described was a 38-year-old woman with good speech and reading comprehension who could write and communicate but had severe word finding impairments. She could often find the correct word, but sometimes she gave up or paraphrased what she wanted to say. Object naming was impaired in the same way. For Pitres amnesic aphasia was 'a form of aphasia in which the language difficulties consist in having forgotten the words that are necessary to express thoughts' (Pitres, in Benton, 1988: 210), and he distinguished three types. The first resembled the one described above; in the second form the speaker cannot create sentences because they have no verbs and connectors at

their disposal, and in the third, selective deficits in polyglots could be observed. Pitres emphasised that pure cases are rather rare. Pitres' amnesic aphasia would later play an important role in neoclassical conceptions of aphasia where it would re-emerge as what we now call **anomia** (Benton, 1988).

Ribot (1881) had suggested in his book on memory disorders that bilingual speakers who are aphasic will recover their native language first. This idea was in general support of his theory that recent memories are more vulnerable to loss than earlier ones (Paradis, 1981). Pitres (1895), who wrote the first important monograph on aphasia in bilingual speakers, stated categorically that the most recently learnt and most familiar language is the one that is recovered first. Unlike Ribot, he based his views on a detailed review of the research, and his analysis of eight new cases. A lively debate continued for some years with some attempting to support '**Pitres' rule**', that the most recently used and familiar language would recover first, and some '**Ribot's rule**', that the first learnt – the native language – would recover first (for relevant papers translated into English, see Paradis, 1983). Pitres' contribution included a strong opposition to the idea that different languages could occupy separate neuroanatomical locations in the brain and it is partly due to his pioneering work that it is now known that a wide range of recovery patterns can be observed in polyglot aphasia (Paradis, 1981).

Henry Charlton Bastian and neat and tidy connectionism

In Chapter 3 we discuss the earlier influence of Bastian, but 1898 saw the publication of his major work *Aphasia and other Speech Defects* (Figure 4.13), which is probably the most comprehensive monograph of its time on the topic. In it **association aphasiology** and **connectionism** are very clearly described, aphasias are classified and systematic testing methods developed.

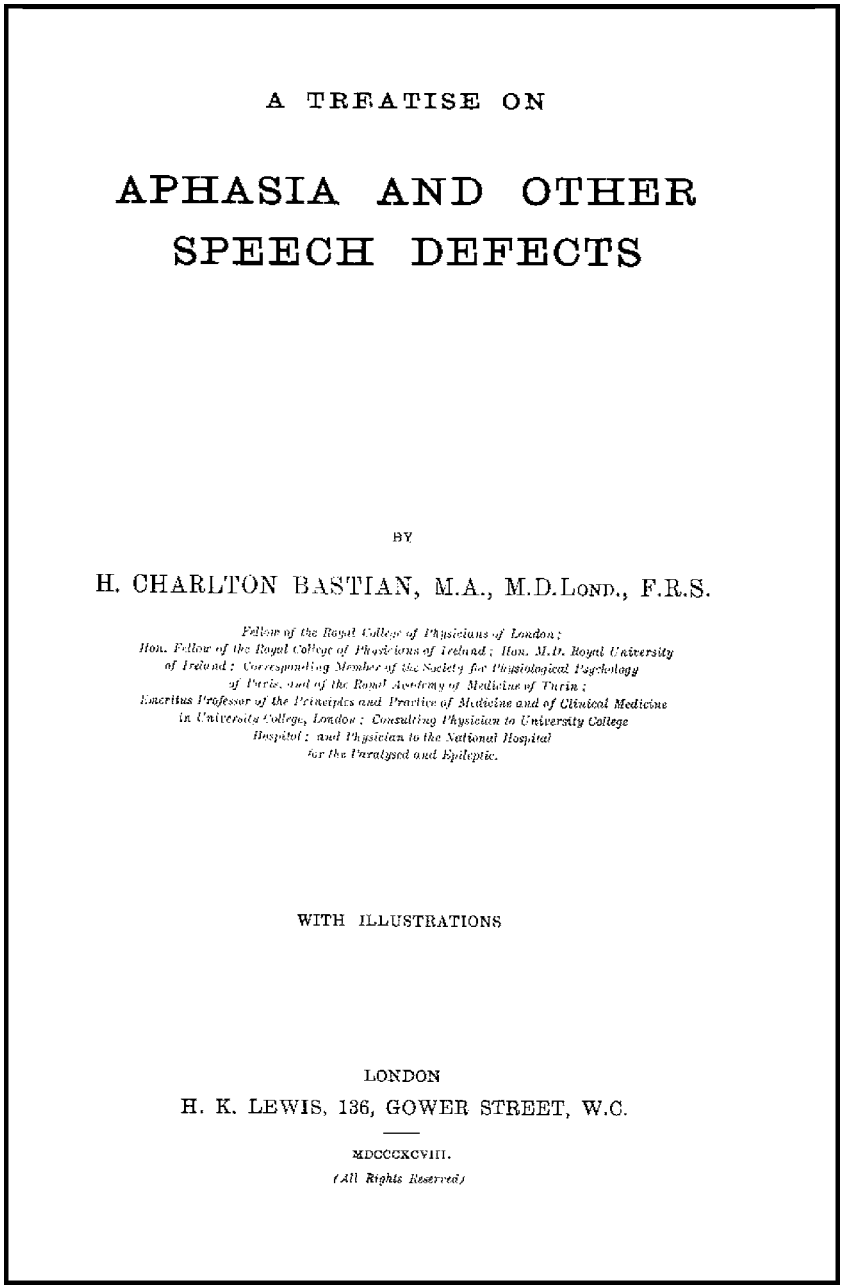
Bastian presupposed three things:

- thinking and language are not dissociated
- word processing takes place in word centres and in their connections with each other to and from the periphery
- aphasic phenomena are the result of a disorder of these centres and pathways (due to disconnections).

Bastian saw thinking and language as inseparable and rejected the idea of an ideation or concept centre as proposed by Lichtheim and Kussmaul.

He argued rigorously in favour of the empirical approach of falsifiability of models (Whitaker, 1998: 49). A major argument for his view was that he could not find evidence for a disorder of a concept centre, which was logical from his point of view because if there were such a centre then it would have to be selectively disturbable.

Bastian described four **word centres** with different connections (commissures) between them. The similarity to Charcot's bell diagram is obvious.



A TREATISE ON

APHASIA AND OTHER SPEECH DEFECTS

BY

H. CHARLTON BASTIAN, M.A., M.D.Lond., F.R.S.

Fellow of the Royal College of Physicians of London;
Hon. Fellow of the Royal College of Physicians of Ireland; Hon. M.D. Royal University of Ireland: Corresponding Member of the Society for Physiological Psychology of Paris, and of the Royal Academy of Medicine of Turin;
Emeritus Professor of the Principles and Practice of Medicine and of Clinical Medicine in University College, London; Consulting Physician to University College Hospital; and Physician to the National Hospital for the Paralysed and Epileptic.

WITH ILLUSTRATIONS

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MDCCCXCVIII.

Figure 4.13 Henry Charlton Bastian's (1898) book *Aphasia and other Speech Defects* is paradigmatic for association aphasiology and the 'diagram makers' of the nineteenth century.

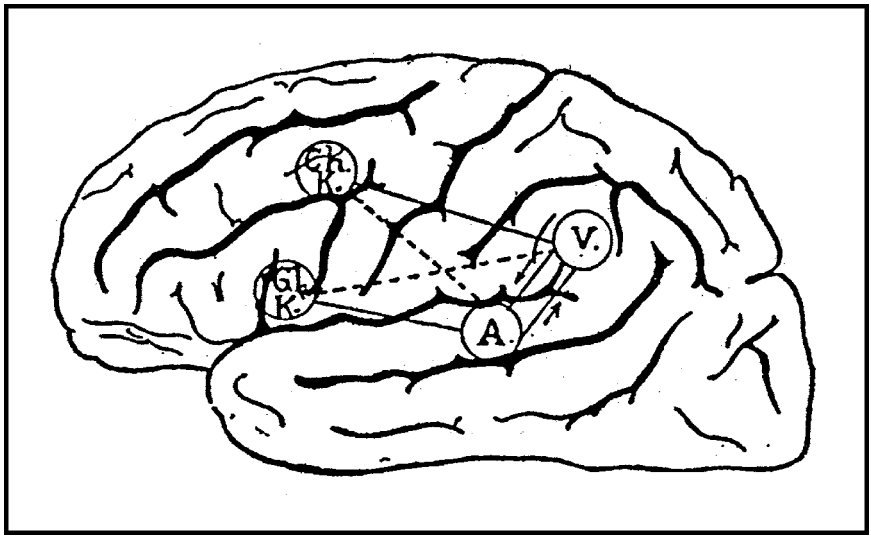


Figure 4.14 The word centres and their localisation in the brain following Bastian (1898: 19) (for explanation of abbreviations see text).

Here Bastian (1898: 45) claimed that he was actually the first to represent this approach: a 'diagram was also produced by Charcot [. . . that] adopts my view as to the existence of separate word centres'.

Two of these centres are sensory (auditory (A) and visual (V) word centres). The other two are the glosso-kinaesthetic centre serving oral production (GlK) and the cheiro-kinaesthetic (ChK) word centre for writing. These centres are localised similarly to Charcot (see Figure 4.14): the auditory word centre in the topmost temporal convolution, the cheiro-kinaesthetic word centre in the second frontal convolution, the glosso-kinaesthetic word centre in the third frontal convolution and the visual word centre in the inferior part of the parietal lobe.

It is worth noting that the processes in Bastian's centres with their many connections are presented relatively dynamically and with many 'active' processes, compared to the Wernicke–Lichtheim model, and his descriptions of the processes can be considered a precursor of contemporary interactive modelling (Whitaker, 1998: 48).

While clearly a connectionist model, and in the spirit of the Wernicke–Lichthiem model, Bastian's (1898: 311–314) system differs somewhat from it, not least in the absence of a concept centre. **Anarthria** is characterised as 'defective power of articulation – speech more or less unintelligible [. . .] understand all that is said to him. Can express himself freely by writing'. How anarthria differs from **aphemia** is less clearly explained, as comprehension and writing are also preserved, but the 'patient is absolutely dumb'. The difference evidently lies in the lesion localisation, which is in the

bulbar area in anarthria and in 'efferent fibres from Broca's centre' in aphemia.

In **aphasia** the lesion is in Broca's centre or in the audito-kinaesthetic commissure. The patient does not speak much (sometimes there are speech automatisms) but has preserved understanding ('understands everything said to him'). **Agraphia** typically accompanies aphasia, and **agraphia without aphasia** results from isolated damage to the cheiro-kinaesthetic centre or through the 'destruction of the audito-visual commissure'. From a 'lowered excitability of the auditory word centre' comes **verbal amnesia**, which is characterised by word finding disorders, paraphasias, well-preserved comprehension, and good repetition. Written speech is impaired in similar fashion.

Word deafness results from damage to the auditory word centre, causing comprehension impairment and aphasia and/or paraphasia. Repetition and writing (to dictation or spontaneously) are not possible. When the auditory word centre is only partially isolated with 'afferent fibres and audito-kinaesthetic commissure intact', repetition and spontaneous writing are possible. **Word blindness** comes from damage to or partial isolation of the visual word centre. In the former case the patient cannot understand, read or write and in the latter the ability to write is partially preserved. **Commissurial amnesia** results from the 'destruction of both commissures between the auditory and visual word centre'. The patient cannot write spontaneously or to dictation, nor read, nor name, but comprehension (auditory, visual), repetition and spontaneous speech are possible. The symptoms of **total aphasia** are aphasia, agraphia, complete word deafness and word blindness.

Bastian's summary of the medical causes of 'aphasia and other speech defects' sounds relatively current. The most frequent causes are 'embolism or thrombosis of the left middle cerebral artery, or of some of its cortical branches' (Bastian, 1898: 302) and when the stem of the medial cerebral artery is affected, total aphasia occurs. In addition, 'traumas often provoke speech disorders [. . .] In this category belong gunshot wounds, cranial fractures [. . .] local bleedings, abscesses or inflammation processes' (Bastian, 1898: 317).

Bastian called for uniform examination procedures that would enable a true comparison of different presentations. Moreover, the less experienced, he suggested, should be able to collect the relevant data. Bastian wrote,

> it will be seen to be highly desirable, if we are to frame a correct clinical [. . .] diagnosis, to submit all such cases to a complete examination in accordance with some uniform and definite scheme. By adopting such a course we can accurately ascertain the nature of the disabilities from which the patient is suffering; and, at the same time, those who are not accustomed to make such examinations may the more readily assure themselves that no important points which ought to receive their attention have been accidentally passed over.
>
> (Bastian, 1898: 307)

He developed a **schema for the examination of aphasic and amnestic patients**, reproduced in Figure 4.15 (Bastian, 1898: 307). His systematic assessment offered a solution to the problem that hindered the reading and interpretation of case descriptions from the nineteenth century, namely the often incomplete and cursory descriptions and examinations of symptoms. Bastian's schema was surprisingly versatile and comprised, beside the coverage of concomitant phenomena, linguistic features, automatic speech, reciting by rote, reading, writing, comprehension, gesture, facial expression and musicality. Bastian's contribution to the early development of treatment for aphasia is discussed in Chapter 5.

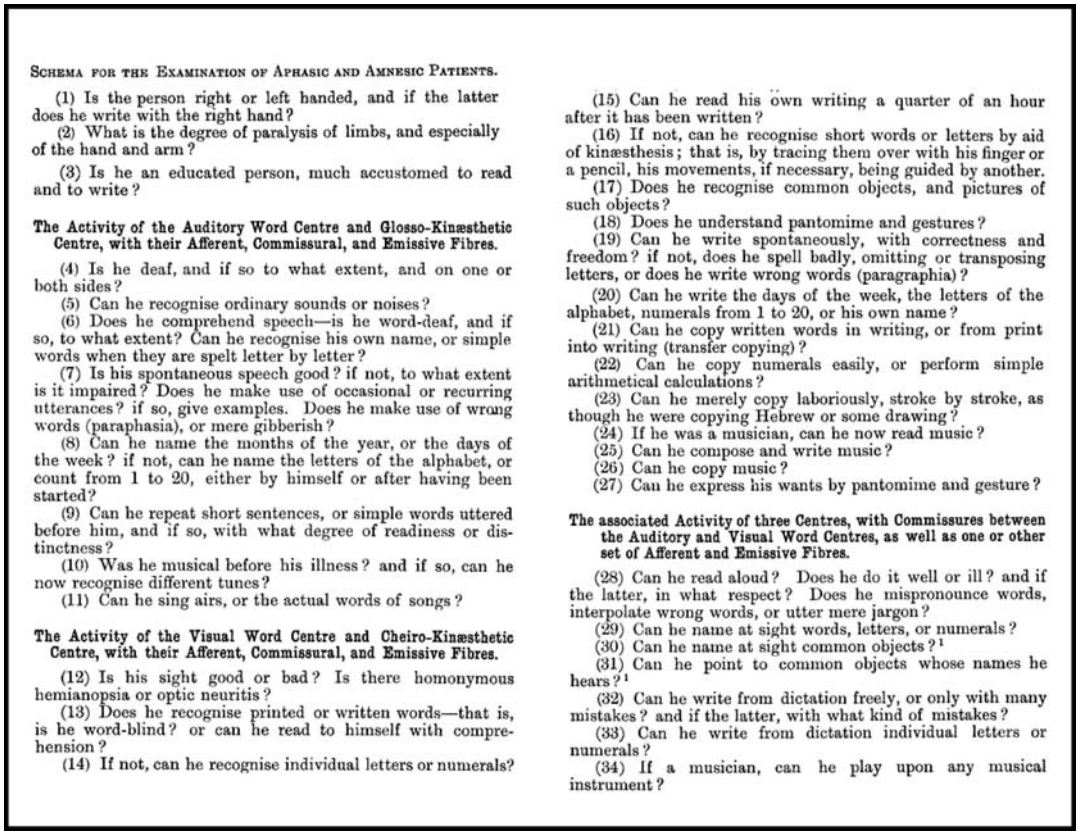
Sigmund Freud's criticism of connectionism

Sigmund Freud (1856–1939) (Buckingham, 2006; Hommes, 1994; Leuschner, 1992; Schultz, 1970; Wallesch, 2004; Wallesch & Bartels, 1996) was a significant critic of connectionism and the Wernicke–Lichtheim model (Caplan, 1987: 79ff.; Henderson, 1992; Marshall, 1974) and developed his own model and classification system. Freud was born in 1856 in Freiburg, Moravia, now in the Czech Republic but then in the Austrian Empire. He studied medicine in Vienna and was a junior or assistant professor in neuropathology at the University of Vienna until 1885, when, at the age of 29, he spent three months with Charcot in Paris, which was to be a turning point in his life (Hommes, 1994). He returned to Vienna as an aphasiologist and a neurologist before he developed his approach to psychoanalysis and helped found psychiatry, which continues to influence so many fields of modern thought. His book *On Aphasia*, with the telling subtitle *A Critical Study* (Buckingham, 2006; Greenberg, 1997), appeared in 1891, but was not translated into English until 1953.

Two particular features of Lichtheim's and Wernicke's models were singled out for criticism by Freud: first, the failure of the models to account for actually occurring phenomena sufficiently, and second, the tendency of the models to predict forms of aphasia that could not, in his view, exist. Concerning the first, Freud (1953) stated that clear cut dissociations did not occur:

> there is an [. . .] objection to Lichtheim's schema: whenever one attempts to fit an observed speech disorder into it, difficulties arise, because one finds the individual speech functions disturbed in various degrees, instead of one being completely lost and another remained intact.
>
> (Freud, 1953: 9)

In addition, Freud (1953: 9) added 'the combination of motor aphasia and alexia which is too frequent to be attributed to the coincidental interruptions of the two fibre tracts'.



SCHEMA FOR THE EXAMINATION OF APHASIC AND AMNESIC PATIENTS.

(1) Is the person right or left handed, and if the latter does he write with the right hand?

(2) What is the degree of paralysis of limbs, and especially of the hand and arm?

(3) Is he an educated person, much accustomed to read and to write?

The Activity of the Auditory Word Centre and Glosso-Kinæsthetic Centre, with their Afferent, Commissural, and Emissive Fibres.

(4) Is he deaf, and if so to what extent, and on one or both sides?

(5) Can he recognise ordinary sounds or noises?

(6) Does he comprehend speech—is he word-deaf, and if so, to what extent? Can he recognise his own name, or simple words when they are spelt letter by letter?

(7) Is his spontaneous speech good? if not, to what extent is it impaired? Does he make use of occasional or recurring utterances? if so, give examples. Does he make use of wrong words (paraphasia), or mere gibberish?

(8) Can he name the months of the year, or the days of the week? if not, can he name the letters of the alphabet, or count from 1 to 20, either by himself or after having been started?

(9) Can he repeat short sentences, or simple words uttered before him, and if so, with what degree of readiness or distinctness?

(10) Was he musical before his illness? and if so, can he now recognise different tunes?

(11) Can he sing airs, or the actual words of songs?

The Activity of the Visual Word Centre and Cheiro-Kinæsthetic Centre, with their Afferent, Commissural, and Emissive Fibres.

(12) Is his sight good or bad? Is there homonymous hemianopsia or optic neuritis?

(13) Does he recognise printed or written words—that is, is he word-blind? or can he read to himself with comprehension?

(14) If not, can he recognise individual letters or numerals?

(15) Can he read his own writing a quarter of an hour after it has been written?

(16) If not, can he recognise short words or letters by aid of kinæsthesis; that is, by tracing them over with his finger or a pencil, his movements, if necessary, being guided by another.

(17) Does he recognise common objects, and pictures of such objects?

(18) Does he understand pantomime and gestures?

(19) Can he write spontaneously, with correctness and freedom? if not, does he spell badly, omitting or transposing letters, or does he write wrong words (paragraphia)?

(20) Can he write the days of the week, the letters of the alphabet, numerals from 1 to 20, or his own name?

(21) Can he copy written words in writing, or from print into writing (transfer copying)?

(22) Can he copy numerals easily, or perform simple arithmetical calculations?

(23) Can he merely copy laboriously, stroke by stroke, as though he were copying Hebrew or some drawing?

(24) If he was a musician, can he now read music?

(25) Can he compose and write music?

(26) Can he copy music?

(27) Can he express his wants by pantomime and gesture?

The associated Activity of three Centres, with Commissures between the Auditory and Visual Word Centres, as well as one or other set of Afferent and Emissive Fibres.

(28) Can he read aloud? Does he do it well or ill? and if the latter, in what respect? Does he mispronounce words, interpolate wrong words, or utter mere jargon?

(29) Can he name at sight words, letters, or numerals?

(30) Can he name at sight common objects?[1]

(31) Can he point to common objects whose names he hears?[1]

(32) Can he write from dictation freely, or only with many mistakes? and if the latter, with what kind of mistakes?

(33) Can he write from dictation individual letters or numerals?

(34) If a musician, can he play upon any musical instrument?

Figure 4.15 Bastian's (1898: 307) systematic aphasia assessment using a questionnaire.

Freud's (1953: 11) second criticism, that the models predicted forms of aphasia that did not exist, was censured with reference to the non-existence of conduction aphasia (De Bleser, Cubelli, & Luzzatti, 1993) which, following Freud's interpretation of Wernicke's and Lichtheim's models, should mean that a lesion of association fibres would cause an abolition of repetition, but leave spontaneous speech intact, 'yet everybody will admit that such a dissociation of speech functions has never been observed nor is it ever likely to be observed'. Generally, Freud believed that there was no sense in a division into centres and pathways, and consequently the division into central aphasias and conduction aphasias (compare Lichtheim) ceased to be useful. Freud's view was that

> all aphasias originate in interruptions of associations, i.e., of conduction. Aphasia through destruction or lesion of a centre is to us no more and no less than aphasia through lesion of these association fibres which meet in that nodal point called a centre.
>
> (Freud, 1953: 67f.)

Freud (1953: 54) therefore also refuted the anatomical model that evolved 'under the influence of Meynert's teachings' which postulated that

> the speech apparatus [*Sprachapparat* = language apparatus] consists of distinct cortical centres; their cells are supposed to contain the word images (word concepts or word impressions). [. . .] One may first of all raise the question as to whether such an assumption is at all correct, and even permissible. I do not believe so.
>
> (Freud, 1953: 54–55)

It is interesting that Freud worked as an intern under Meynert, and Jones (1953: 36) highlighted Freud's problems with 'father figures' when he commented that Freud's striving for independence is, in every way (including scientifically), the basis for criticising a scientific giant like Meynert this sharply. Indeed, another dominating figure of neurology, Charcot, who Freud spent some time with, is similarly criticised.

Freud (1953) therefore

> rejected the assumptions that the speech [= language] apparatus consists of distinct centres separated by functionless areas, and the that ideas (memories) serving speech are stored in certain parts of the cortex called centres while their association is provided exclusively by subcortical fibre tracts. It only remains for us to state that the view that the speech area [= language area] is a continuous cortical region within which the associations and transmissions underlying the speech functions are taking place; they are of a complexity beyond comprehension.
>
> (Freud, 1953: 62)

Freud's (1953: 67) notion of the areas of the brain responsible for language was broad and he assumed that the **language area** (also called language field, *Sprachfeld*) is in the left hemisphere and occupies 'the space between the terminations of the optic and acoustic nerves and of areas of the cranial and certain peripheral motor nerves in the left hemisphere. [. . .] It probably covers [. . .] all the convolutions forming the Sylvian fissure.'

With Freud's (1953: 67) conception of a 'language field' (association area of language) 'the speech centres are [. . .] parts of the cortex which may claim a pathological but no special physiological significance'. In this way Freud was able to account for correlations between lesion locations and aphasic symptoms without having to localise centres. Like Hughlings Jackson, Freud knew that to localise the lesion is not equivalent to localising the function.

However, Freud (1893, in Kästle, 1987: 523) did acknowledge that the localisation of damage nevertheless plays a role:

> The destruction of the central area (to which belong the insular gyri) is tolerated quite well by the language function and only provokes the image of an indeterminate a[phasia]. [. . .] Whereas the destruction of the peripheral parts of the language field creates a[phasias] of a specific character. Depending on the situation of the peripheral lesion, one finds the motor, acoustic or visual element in the language associations to be damaged and can diagnose a motor a[phasia] (and agraphia), an acoustic (sensory) a[phasia] and a visual a[phasia] (alexia) and localise it with approximate certainty.
>
> (Freud, 1893, in Kästle, 1987: 523)

Freud has been considered the first 'neogrammarian neurolinguist' (Buckingham, 2006; Marshall, 1974) supporting, as he did, the emerging psycholinguistics of the period that was developing new analytical techniques and developing models of normal language and its development and evolution. Like Steinthal, Freud proposed a psycholinguistic approach to the investigation of aphasia that was concerned with describing impaired language using linguistic methods. Freud (1893) considered words as complex **association structures** (Figure 4.16). For him there were closed word associations (sound-image, images for reading and writing and motor-image) and there were open object associations (Rizzuto, 1990). Between the two association complexes Freud (1893, in Kästle, 1987: 522) claimed that there exists a 'symbolic' relationship, and 'to each object a word is associated as a "symbol" '. From this resulted the

> classifications of language disorders following a psychological viewpoint: disorders within the word associations themselves may be called **verbal a[phasia]**, disorders in the association between word and object **asymbolic a[phasia]**, and when there are speech disorders that result from a disorder

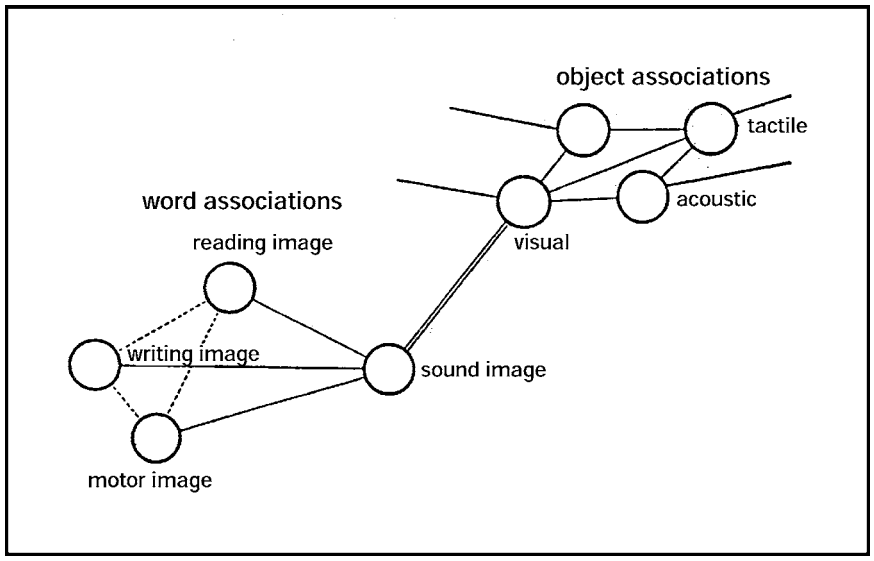


Figure 4.16 The word model of Sigmund Freud (following Kästle, 1987: 522).

> within object associations, then they deserve the denotation **agnostic a[phasia]**.
>
> (Freud, 1893, in Kästle, 1987: 522)

Freud's classification met with little acceptance although his introduction of the term and the concept of **agnosia** became well established in neurology.

To conclude Freud's contribution, two points remain. First, Freud is one of the few contemporary aphasiologists to absorb and value the views of Hughlings Jackson. Second, Freud supported the **continuity hypothesis**, which claims that normal and aphasic paraphasic errors only differ quantitatively, but not qualitatively (Buckingham, 1999) and he dealt with paraphasias in detail and considered them to be one, if not *the*, central symptom of aphasia (Lebrun & Stevens, 1976). Finally, he was one of the first aphasiologists to recognise the value of the application of psycholinguistic investigations in aphasia.

Freud's aphasiological ideas found little support in general. The original book *On Aphasia* seems to have met with very little interest and apparently only 257 copies were sold over nine years, with the remainder of the edition turned into pulp (Henderson, 1992: 35). Interestingly, Freud (1953) predicted at the end of his book:

> I am well aware that the considerations set out in this book must leave a feeling of dissatisfaction in the reader's mind. I have endeavoured to demolish a convenient and attractive theory of the aphasias, and having

> succeeded in this, I have been able to put into its place something less obvious and less complete.
>
> (Freud, 1953: 104)

Summary

Intense interest and discussion followed the localisation debates in Paris in the 1860s. In Britain Hughlings Jackson and others had introduced an evolutionary model of the brain and nervous system and argued, with Bateman, against localisation theorists like Bastian. In Germany the classical doctrine developed from the dominating Wernicke–Lichtheim model. At the same time theoretical positions developed, like asymbolia, which saw the essence of aphasia differently. The first linguistic, psycholinguistic and neuropsychological models emerged (e.g., Steinthal, Kussmaul). At the end of the nineteenth century a vigorous Europe-wide debate took place involving contributors like Charcot in Paris, Bastian in England, and Freud in Vienna. By the end of the century aphasia and the representation of language in the brain was an important and firmly established field of science.

5 The twentieth century until the Second World War

Following the momentous events of the nineteenth century, there was an enormous expansion in the study of aphasia at the turn of the twentieth century and hundreds of papers were published. For instance, a book by von Monakow (1914) lists more than 2000 references to aphasia. This was a time when a range of developments progressed in parallel. On the one hand, Wernicke's model continued to be dominant, and work inspired by localisationist–connectionist models culminated in the publications of Samuel Eberhard Henschen and Karl Kleist. On the other hand, powerful cross-currents emerged from the holists, like Pierre Marie and Henry Head, who also influenced developments. Within the scope of this development Hughlings Jackson was also (re-)discovered. At the same time association psychology was slowly superseded by other psychological schools (Ringer, 1983: 173ff.) and there was a rejection of aphasiology that focused on the word and a growing interest in impairments in sentence-level grammatical processing, especially in the German research.

One of the many legacies of the First World War was a large number of brain-damaged people, leading to the development of research on the psychopathology of higher brain functions and, for the first time, the impact of brain damage on individuals and the rehabilitation of aphasia began to occupy more workers (e.g. the works of Gutzmann and Goldstein in Germany and Hughlings Jackson in England). An estimated 10 million people lost their lives in the First World War and twice that number were injured. As Harrington (1987: 264) states: 'Cynical as it sounds, there can be no denying that the war was an exciting time for clinical neurologists.'

After the First World War a certain aversion to German science and inventions like connectionist explanations for aphasia developed, which facilitated, for instance, the reception of Head's work. Geschwind (1964) noted that

> Head (1926) had been shrewd enough to point out that much of the great German growth of neurology had been related to their victory in the Franco-Prussian war. He was not shrewd enough to apply this valuable historical lesson to his own time and to realise that perhaps the decline of the vigor and influence of German neurology was strongly related to the

> defeat of Germany in World War I and the shift of the center of gravity of intellectual life to the English-speaking world, rather than necessarily to any defects in the ideas of German scholars.
>
> (Geschwind, 1964: 214–215)

With the rise of fascism in the late 1920s, fruitful discussion on aphasia ended abruptly in Germany. One-third of academics lost their positions in German universities and, although medicine was less heavily affected, many in neurology (approximately half), psychology and psychiatry were divested of their office, banished or killed. Among the aphasiologists, Max Isserlin fled to England, Lotmar Fritz to Switzerland, Emil Froeschels to the USA, Kurt Goldstein emigrated first to the Netherlands and finally the USA, Egon Weigl to Romania via Prague. The already ill Adhémar Gelb did not survive the mental terror and Edmund Forster committed suicide. With the forced relocation of these individuals came the relocation of scientific aphasiology to the Anglo-Saxon world during the Second World War, especially to the USA (although some, like Weigl, fled East), which led to a shift in the centre of gravity in aphasia, as in other fields, from Europe to North America.

The 'assault' on the classic model

While the classic Broca–Wernicke–Lichtheim model established in the latter part of the nineteenth century was developed by Dejerine, Liepmann and others at the beginning of the twentieth century, Pierre Marie especially stood out as a forceful critic and took firm exception to the model; Constantin von Monakow's work on brain plasticity and reorganisation also did not sit well with the rather static classic model.

Pierre Marie, the iconoclast

Pierre Marie (1853–1940; see Figure 5.1) was an intern in Broca's and in Charcot's departments. Head (1926) dubbed him 'the iconoclast'; in addition, he appears to have been one of the most unpleasant men in the history of aphasia. Charcot and he were apparently close, as Charcot was a witness at Marie's wedding. He co-founded the *Revue Neurologie* in 1899 with Brissaud, was Professor of Pathological Anatomy at the University of Paris and followed Dejerine as Professor of Clinical Neurology (Lebrun, 1994). He was originally a localisationist like Charcot, but in 1906 initiated a major discussion, which continued without conclusion or agreement until 1908 (Brais, 1992; Lecours et al., 1992; Lhermitte & Signoret, 1982).

The bone of contention is an article by Marie (1906a) with the resolute title 'The third left frontal convolution plays no special role in language function', which was quickly followed by three other articles in which Marie (1906b, 1906c, 1907) further presented his position. In the period from 1906 to 1907, Marie was very productive, authoring at least 14 articles on aphasia, either

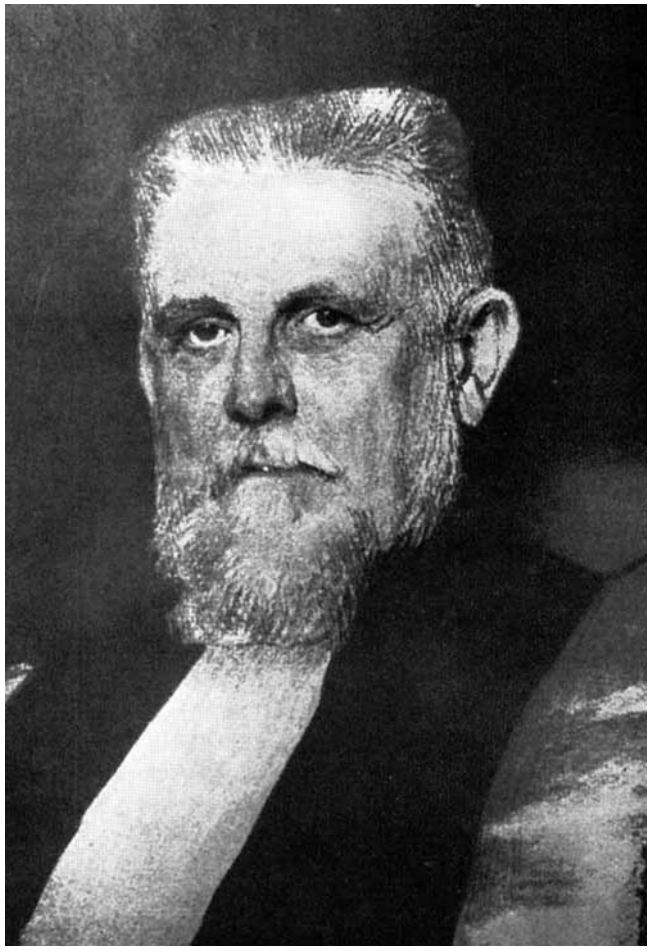


Figure 5.1 Pierre Marie, who attacked the classic aphasia doctrine of Broca, Lichtheim, and Wernicke in 1906.

alone or with François Moutier (Cole & Cole, 1971). 'Marie's first 1906 paper caused an immediate uproar' (Weisenburg & McBride, 1935: 20), and 'The Paris medical community was shocked by Marie's article' (Brais, 1992: 693).

The central point of attack is 'the dogma of the third frontal convolution', because Marie reported cases where severe damage to this convolution did not result in aphasia. In addition, he was able to show that Broca's aphasia could exist without lesions of the third frontal convolution (Marie & Moutier, 1906a, 1906b, 1906c).

Marie argued for an entirely different view of aphasia. For him, **language comprehension impairment** is the cardinal problem in aphasia, and in every form of aphasia, the first temporal convolution and/or the white matter

beneath, must be affected. For Marie, 'motor' (Broca's) aphasia is always the result of a lesion that affects both the first temporal convolution and what he called the 'lenticular zone'. Lecours and Caplan (1984) describe Marie's zone as

> quadrilateral extending between the island of Reil and the lateral ventricle and including the insula, the claustrum, the external and the internal capsule, and the caudate and lenticular nucleus. The third frontal convolution and its underlying white matter explicitly lay outside this zone.
>
> (Lecours & Caplan, 1984: 170)

According to Marie a lesion of this zone will lead to **anarthria**, his term for a disorder in the initiation and control of the complex movements required for the production of speech (see also *apraxia of speech*, *aphemia*, *dysarthria*), which approximately corresponds to subcortical motor aphasia, a term that Marie (1906a: 243) vehemently opposed because, according to him, 'l'anarthrie n'est pas de l'aphasie' – anarthria is not aphasia. Marie's use of the term anarthria is not the same as its current use, where it means a disorder of articulatory implementation because of paralysis or incoordination; Marie's anarthria is akin to what Broca called aphemia and most call apraxia of speech nowadays. Speakers with anarthria also understand without any problems, according to Marie. Thus Marie came up with his famous equation: Broca's aphasia = Wernicke's aphasia + anarthria. Consequently, Broca's aphasia is caused by a lesion compromising Wernicke's area and the lenticular zone, but without damage to Broca's area.

Marie (1906a: 244) was against the fractionation of aphasia, and his opinion was that 'l'aphasie est une' – there is only **one true aphasia** and that is sensory aphasia caused by a lesion of Wernicke's area (supramarginal gyrus, angular gyrus and the base of the first two temporal convolutions; see Figure 5.2). Obviously, this is a significantly different 'Wernicke's area' than Wernicke's own.

A new examination of the brains of Leborgne and Lelong took place as part of this provocative attack on Broca's views. Marie claims that these two cases did *not* support Broca's views. Marie suggested senile atrophy in Lelong's brain which also compromised Wernicke's area. In Leborgne's brain, he showed that the softening discovered by Broca reached into the first temporal convolution and the supramarginal gyrus.

Marie also attacked associationist aphasia, stating that there is little sense in holding on to word centres and stores. At the same time, he attempted to shake another pillar of the Broca–Wernicke model, the view that intelligence is totally unimpaired in aphasia. Marie believed that aphasic people always show a decline in their intellectual capacity, because thinking is closely linked to language and cannot be separated from it. This was supported by Moutier's enormous case collection (several hundred cases), which he,

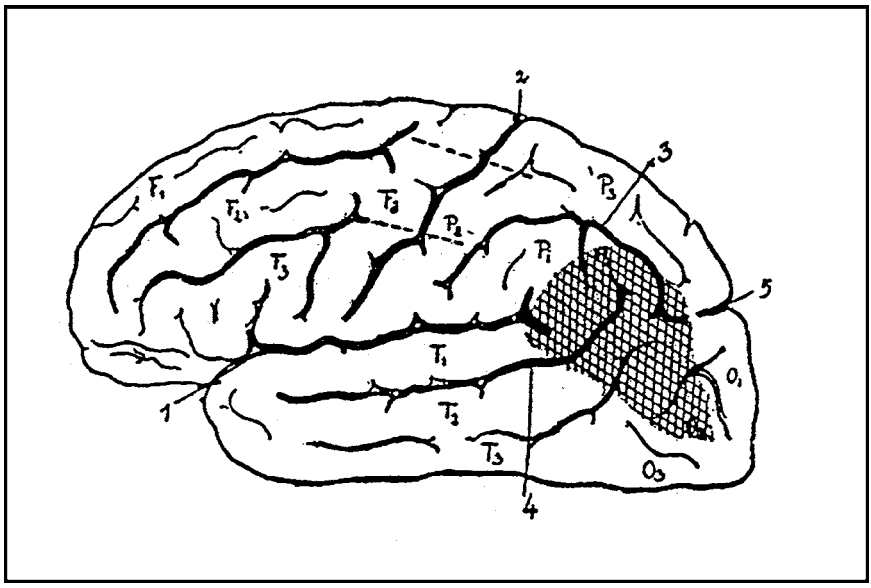


Figure 5.2 Wernicke's area according to Marie (1906a).

Moutier, submitted as a dissertation (Moutier, 1908). In exchange for his support, Marie promised advancement to Moutier, but when Marie was promoted he fired Moutier while the debate in the Neurological Society was still running (Lecours et al., 1992: 144).

The response to Marie's challenges came especially from Dejerine (1906a, 1906b), who had been a rival of Marie's for years (Brais, 1992: 693). There was an (also scientific) altercation between Marie and Dejerine as early as 1892, which ultimately led to Dejerine challenging Marie to a duel. After Charcot's death, Dejerine was given preference over Marie several times, because Dejerine was sponsored by the influential **E. F. A. Vulpian** (1826–1887) who had been Dejerine's teacher. When Charcot's academic chair became vacant again in 1910, Dejerine was called, and it was only after Dejerine's death that Marie could finally occupy the prestigious chair. One of Marie's first official acts, on his second day in post, was to fire Augusta Dejerine-Klumpke, an eminently respected scientist and the first woman to receive the title *interne des hôpitaux* in 1887 (Renier, 1994), but also Dejerine's widow.

As to be expected, Dejerine and his staff defended the classical tradition. Marie and Dejerine presented their positions at the concluding discussion of the *Société de Neurologique de Paris* which took place in 1908 (Lecours et al., 1992). The meeting ended without conclusion, but 'by the end of the debate most neurologists seemed to agree that there was a need to question the traditional teachings on aphasia' (Brais, 1992: 694).

Although Marie did not develop a substantial theoretical perspective of his own, his attack on connectionism and the classical theory still disturbed associationist aphasia, and his critique resonated throughout the western world. Besides Dejerine, Grasset and Mahaim took opposing positions to Marie (Brais, 1992: 694). Dieulafoy and Souques in France, Dercum and Lasalle-Archambault in the United States, and Markeloff in Russia argued in favour of Marie. Binet, Simon, Bernheim, Collier, Ladame, von Monakow and Meyer were partly convinced by Marie's arguments. But especially Henry Head in England and Kurt Goldstein in Germany would subsequently develop ideas which began with Marie that would also break with the classical dogma.

Marie's work became established because it sat well with the position that was developing in France through the philosopher **Henri Bergson**, who rejected associationist psychology. How far Bergson actually influenced Marie remains unclear, although Marie is sometimes called 'Bergson's neurological pupil' (Ombredane, 1951). In his book *Matter and Memory*, Bergson (1896) also dealt with aphasia, in the course of which he refuted association psychology and its (memory) images: 'It is the constant misapprehension of association psychology that it [. . .] replaces lively reality by a discontinuous multiplicity of lifeless juxtaposed elements' (Bergson, 1991: 128). Consequently, associationist notions of aphasia were not tenable either (Brandt, 1966).

Hugo Liepmann critique of Marie's 'false doctrine'

In the literature appearing in German, it was **Hugo Liepmann** (1863–1925), a former assistant of Wernicke, who dealt especially critically with Marie's views. Liepmann's (1909) essay was widely circulated; his critique of Marie's theory was accepted by many in Germany, and Marie played no significant role in the discussion in the German literature (Froeschels, 1914: 222).

Liepmann (1909) saw Marie's attack on classic doctrine as being totally excessive suggesting that Marie had simply set up a straw man:

> For scholars of literature on aphasia, it generally results that a so-called 'revision' of aphasia theory need not be stipulated now with pathos at all, but rather that it has been continuously under way for decades. When Marie speaks of *one* 'classic doctrine' against which he calls the field to battle, this seems to us a fantasy incomprehensible to one who overlooks the collective work of scientists of many nations. Where is the rigid dogma towards which the majority of authoritative minds were biased, and who should be met by the booming wake-up call 'on to revision'?
>
> (Liepmann, 1909: 450)

Liepmann referred to the comprehensive literature of the first years of the twentieth century, which, as we have seen, really did comprise very diverse positions. Liepmann (1909: 452–453) then discussed 'MARIE's false doctrine'

that 'the incapacity for articulated speech [. . .] has nothing to do with aphasia', but is instead only anarthria, and that 'aphasia [is] an intelligence disorder which shows itself in bad or abolished understanding, writing, reading, but furthermore in a widespread reduction of the intellectual faculty in general'.

With regard to the motor component of Marie's position, Liepmann (1909) referred to the fact that, among other things,

> on occasion, emotional utterances and remaining words of the patients prove: the neuro-muscular executive apparatus is not destroyed. A patient otherwise would become dumb, with the remaining words: 'good evening, good morning, oh God yes!' proves by the same token that tongue, lips and cheeks can take any necessary position upon cortical stimulation.
>
> (Liepmann, 1909: 460)

Marie's position seemed hardly compatible with these observations.

The notion that intelligence is impaired in aphasia was only an option for Liepmann if 'intellectual' was seen in a broader sense, that is, by 'intellectual' was meant selective disorders of memory and its associations. But that is not what was meant by Marie, who supposed 'a very widespread general reduction of intelligence' in aphasia. Liepmann (1909: 456) emphasised that reductions in intelligence, when they appear in aphasic people, are not causally related to aphasia, because 'aphasia is a symptom-complex that can exist *independently* from a general obscuration of the mind'.

On the question of localisation – Marie considered the third frontal convolution to be language-*irrelevant* – Liepmann (1909: 472) presented cases from the literature and new cases of his own 'with actual abolishment of articulated speech, a large ostensible focus in F3' was missing in only two cases. One case had a lesion 'immediately in F3 to the back', which Liepmann explains by a slight extension of the frontal speech area. The cases of Marie and those of his 'shield bearer' Moutier, which have no lesion of F3 but still have aphasia in Marie's view, do not have aphemia (Marie's 'anarthria') in Liepmann's analysis, or the aphasic disorder has its origin in an F3 lesion that simply had not been noticed by Marie and Moutier.

In conclusion, Liepmann (1909: 484) conceded that aphasia theory was far from being settled or complete and that it would be 'continuously revised further'. One thing was clear, however: 'A revision [. . .] like MARIE's [. . .], which [. . .] replaces small errors by bigger ones, ideas that are not yet definite but advancing by views that lock out insight, is to be rejected.'

However, Liepmann is known less for his contribution to aphasia than for his work in **apraxia** (Liepmann, 1900, 1908; see Rothi & Heilman, 1996). Apraxia is the inability to voluntarily plan and carry out actions and gestures in the absence of neuromuscular paralysis or coordination. Isolated observations of apraxias before Liepmann were classified either as 'motor asymbolia' or as 'agnosia'. It became clear only with Liepmann that apraxia is a motor

planning and programming deficit that cannot be explained by a primary motor dysfunction. In later writing Liepmann developed a classification of different forms of apraxias; a classification that still informs contemporary research and clinical work. Liepmann (1913: 56) described apraxia of speech as a variant of limb-kinetic apraxia, stating that 'the word limb here, refers to the tongue, palate, and oral mechanism' and that 'speech is a parasite making use of a preformed sensory-motor mechanism which is also involved in *other* functions, particularly that of eating, it likewise shares in any impairment sustained by this same mechanism'.

With Liepmann's work on the apraxias and development of research on impairments of perception, for which Freud introduced the term agnosia, the triad of aphasia, agnosia, and apraxia, emerged and became established as the major scaffolding for the classification of higher neurocognitive disorders following brain damage.

Constantin von Monakow and the concept of diaschisis

Constantin von Monakow (1853–1930; see Figure 5.3) was Russian and a naturalised Swiss who worked in Zurich and had studied under Eduard Hitzig and Theodor Meynert, among others (Harrington, 1996: 76ff.). In a comprehensive study of 1897 (second edition, 1905), he described his conception of the language region (Figure 5.4):

> The language region [. . .] comprises the cortical area of all convolutions involved in the creation of the Sylvian fissure, especially on the left side. It includes probably the entire third frontal gyrus [. . .], the whole insular cortex, the upper and lower lip of the Sylvian fissure and especially the cortical area of the first temporal gyrus. [. . .] Towards the occipital lobe, the language region goes over to the supramarginal gyrus and the angular gyrus.
>
> (Von Monakow, 1905: 827–828)

Von Monakow (1905: 828) was against a strict limitation, however: 'The borders of the language region are blurred and cannot be expressed by means of lines; this location probably abates very gradually in all directions, i.e. it reaches far across into the neighbouring gyri.'

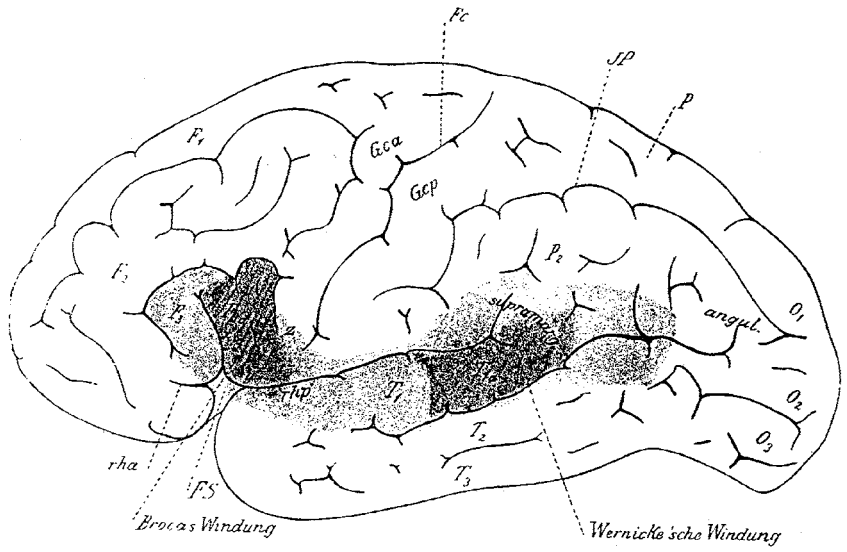
Von Monakow's basic aphasic disorders (still very much in the word-based traditional) were **anamnestic** aphasia (word recall deficit, *Wortvergessenheit*), word dumbness (*Wortstummheit*, motor aphasia), agraphia, word deafness (*Worttaubheit*, sensory aphasia) and alexia. These symptoms emerge from a disorder of the language field; but von Monakow (1905: 827) emphasised that a disorder of language, 'although lawful is not, under all conditions, a lasting occurrence', because 'long-range effects of various types (intercortical diaschisis, etc.) play a prominent role' here.

Von Monakow's notion of **diaschisis** describes a kind of cerebral 'shock'

Figure 5.3 Constantin von Monakow (1853–1930), whose concept of diaschisis (long-distance effects) has had a major influence on theoretical notions of brain organisation right up to contemporary times.

following brain damage that can lead to functional deficits from some interference to totally different parts of the brain and at some distance from the original lesion. Change or apparent 'recovery' in function with time after damage can be explained in terms of a reduction in the effects of diaschisis as the brain lesion heals and the diaschisis reduces. For von Monakow (in Harrington, 1996: 79), diaschisis was 'an essential dynamic principle, and it represents the bridge between localisable and non-localisable phenomena' and he (von Monakow, 1905) considered the processes specified by the Wernicke–Lichtheim tradition to be too restrictive:

> The anatomical substantiation of aphasic disorders to a sufficient degree, and their specific clinical forms completely, leave rather much to be desired, at the moment, despite the extensive material laid down in literature. It is certain [. . .] that the gyral regions that have been associated with aphasic language disorders have been conceived too narrowly by far up to now, and namely as far as the long association fibre tracts,



Lateral view of the left hemisphere with the language region. Language region dark, the relative fields shaded somewhat more lightly. *FS* Sylvian Fissure. *rha, rhp* anterior horizontal ramus, posterior horizontal ramus of the Sylvian Fissure. *Gca, Gcp* Anterior, respectively posterior central gyrus. F_1 F_2 F_3 First to third frontal gyri. *Fc* Central fissure. *JP* Interparietal fissure. O_1 O_2 O_3 First to third occipital gyri. T_1 T_2 T_3 First to third temporal gyri. *P* Superior vertical lobe. P_2 Inferior vertical lobe. *Supramarg* supermarginal gyrus. *angul* angular gyrus.

Figure 5.4 The language region according to von Monakow (1905: 828).

> commissures, and their areas of destination and origin respectively, all the way to their termini, which go from these regions **to other** gyral groups, i.e. the ones located outside of the language region, have been taken into account too little by far. The damaged cortical area, which often has macroscopically relatively small and well defined centres, goes **far beyond the visible borders of the lesion**.

The improvement of functional deficits is a central topic for von Monakow (1905: 912) which he tried to explain with reference to diaschisis and by supposing that the brain can reorganise itself through a process of plasticity, where new, compensatory connections can develop:

> That language quite often returns despite the persistence, aye, even despite moderate progress of the focus, explains itself by [. . . the fact that] the other [i.e. the right] hemisphere, which is habitually transitorily

> affected by long-range effect (diaschisis), gradually regains its ability to function. We may also think about the fact that, with time, under improved utilisation of the numerous [. . .] cortical connections (tracts located outside of the actual language area and those in the right language region), new supplementary connections to the basic sound and tone centres can be found, which, even if they function less promptly than the main language functions, are still possible to a certain degree.
>
> (von Monakow, 1905: 912)

The narrow notion of language localisation became increasingly incompatible with the introduction of von Monakow's concepts of diaschisis and plasticity. Goldstein (1927/1971: 155) summarised: 'Especially under the influence of v. Monakow's theories, the theory of circumscribed localisation has made room for a notion that attributes essential significance to the entire brain for the normal course of language processes.' Because von Monakow emphasised the complexity of the brain and its functioning as a whole, his work is typically considered together with other holistic conceptions and it is not by coincidence that von Monakow commented on the importance of Hughlings Jackson's work in his later writing (von Monakow, 1914; Harrington, 1996; 80ff.). Von Monakow subsequently developed a model of diaschisis that was graded hierarchically and developmentally where abilities that are acquired later in life are affected first by brain damage, and abilities acquired earlier are less affected. Diaschisis has developed into one of the most powerful explanatory mechanisms for recovery and modern imaging methods have provided support for its general theoretical assumptions, as discussed in Chapter 8. However, its strength is also its weakness, and its explanatory potency is seen by some as too powerful.

The ultra localisationists

Although the criticism of classic aphasia localisation theory by psychologists and holists was very influential, and although the voices of Marie, Pick, von Monakow, Head and Goldstein were clarion, localisation reached extreme realisation in the work of Henschen and Kleist, whose contributions are briefly described next.

Samuel Eberhard Henschen: the collector

After having become emeritus professor in 1912, **Samuel Eberhard Henschen** (1847–1930) (Weisenburg & McBride, 1935: 29–31; Winkelman, 1953) authored a comprehensive monograph on *Clinical and Anatomical Contributions on the Pathology of the Brain*, parts 5 to 7 of which are dedicated to aphasia (Henschen, 1920a, 1920b, 1922). Henschen's (1920b) credo is:

> Only on clinically-anatomically secured facts can the edifice of aphasia

> theory be built. Neither deductions a priori nor psychological reflections were able to present this theory with a firm basis. Modern endeavours to explain aphasic symptoms from psychological points of origin and under disregard of anatomy have led aphasia theory astray and emerged as fruitless.
>
> (Henschen, 1920b: preamble)

Henschen did not place much stock on the psychological approaches of Goldstein, Head and others, and he emerged as a dyed-in-the-wool localisationist. Weisenburg and McBride (1935: 29) wrote: 'Of all modern investigators, Henschen is the most extreme protagonist of the doctrine that knowledge of aphasia is possible through knowledge of localisation.'

Based on his documentation of no fewer than 1337 cases Henschen (1920b) tried to collect and systematise the

> clinically verifiable language debris – in so far as the corresponding autopsy findings were known – and, which seemed particularly important to me, to connect them to anatomical lesions, always guided by the intention of whether and in how far specific anatomical locations could be verified in terms of certain processes during language creation.
>
> (Henschen, 1920b: 112)

Henschen's findings generally confirmed the classic speech and language centres (third frontal convolution, superior temporal lobe, angular gyrus, among others). Generally, however, Henschen's work did not result in a coherent theoretical picture, which, among other things, may have been responsible for the low degree of acceptance of his ideas in aphasiology (Borenstein, 2005).

Karl Kleist's brain map

Karl Kleist (1878–1960) was yet another of Wernicke's assistants for about a year in Halle between 1903 and 1905, and Wernicke's influence on Kleist seems to have been enormous (De Bleser, 1987: 251). He was perhaps the most ardent localisationist and later in this chapter we discuss his contribution to the investigation of impairments of grammar. Here we describe the highly explicit localisation model he devised in 1934. Claus Wallesch (1988: 161) commented: 'Localisationism found its culmination in Germany in the work *Brain Pathology* by Karl Kleist.' With this comprehensive study Kleist took localisationism to an extreme position from which it could only fall perhaps. On the basis of Brodmann's (1868–1918) brain mapping, which divides the cortical surface into neatly numbered areas (see Appendix), Kleist sketched his famous brain map (Figure 5.5). On this map the higher mental functions are localised in their entirety to specific regions, and the brain cortex is presented as a 'mosaic of organs' (Wallesch, 1988: 161).

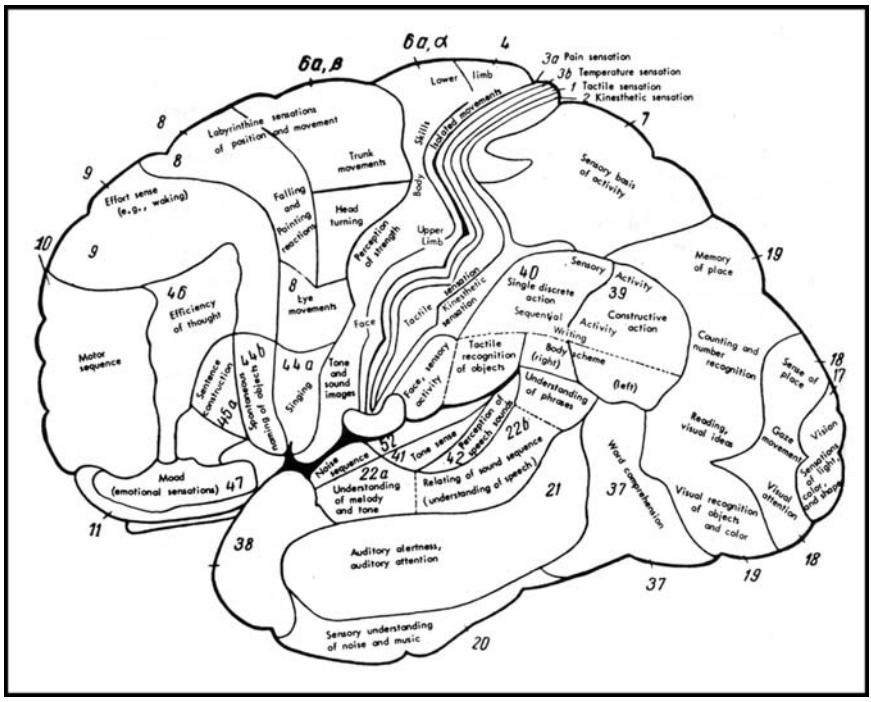


Figure 5.5 Karl Kleist's (1934) famous brain map (localisation chart) (following Luria, 1980: 12).

The foundation of Kleist's work is hundreds of brain-damaged soldiers from the First World War whose symptoms and lesion locations are examined in very detailed case presentations. The lesions are predominantly bullet wounds and therefore typically focal. In defence against the critical voices of the holists, Kleist (1934) wrote:

> The localisation of complex linguistic processes in circumscribed brain regions is refuted by MONAKOW as well as HEAD. All these objections coalesce in GOLDSTEIN'S theory with the attempt to better explain the disorders, which were association-psychologically conceived by the founders of aphasia theory, with the aid of Gestalt psychology.
>
> (Kleist, 1934: 803)

Kleist (1934: 804) rejected the holists' objections as 'premature as long as the simple clinical characteristics and types of speech dumbness have not been completely determined and as long as the anatomical findings are in such need of complementation and improvement as they are now'.

He explained inconsistencies in the cases published up to that point by saying

> that the researcher positioned a function into an entire convolution or into a diffusely limited part of a convolution while only a smaller field of this convolution is the carrier of the corresponding function. In addition, the areas do not respect the limits of the convolutions and fissures. The old material of aphasia literature with its coarsely described brain findings fails in this respect for the most part.
>
> (Kleist, 1934: 804)

Kleist hence assumed as a principle that the localisation of specific functions is possible and necessary and that localisation had been determined with inadequate methods in the past.

The proximity to Gall's brain mapping is not only visible but obvious (compare Kleist's model to Gall's in Chapter 3) and like the phrenologists, Kleist claimed that specific brain areas would be especially extensive in the case of the gifted. One example from many may serve as an illustration:

> Mrs Kowalewski [. . .] was [. . .] so [language] gifted that apart from Russian, her native language, she spoke German and French, learned Swedish in a very short amount of time [. . .] Moreover, she wrote novellas and stories. At the basis of this certainly rather great gift for languages lies an extraordinary development of the basal part at the left third frontal convolution, which mainly affects the anterior, ascending section. [. . .] The development of F3 [. . .] confirms our [. . .] conclusion following which the pars ascendens in particular is the organ of the highest language functions.
>
> (Kleist, 1934: 925–926)

Kleist adopted Wernicke's sensory and motor dichotomy and complemented it with his own classification, but 'Kleist's nomenclature never managed to prevail against Wernicke's earlier distinction' (Leischner, 1987: 58).

Under **sensory aphasias** Kleist discussed sound-deafness, word-deafness, syntactic deafness (by which he means temporal lobe paragrammatism) and agrammatism and amnestic aphasia or anomia. Kleist (1934) differentiated the steps of comprehension and their selective impairment:

> The particularly rich development of human language brought with it, however, that the individual forms of disorders are divided even more richly. In the field of language, there is not only the chronological sequencing of sounds that produce a word, the words also arrange themselves into phrases and sentence.
>
> (Kleist, 1934: 688)

Brodmann areas 42 (sound comprehension), 22b (sound sequence and sentence comprehension) as well as 37 (name comprehension) are held to be the brain regions responsible for comprehension.

Under **motor aphasias** Kleist described sound-dumbness, naming-dumbness and syntactic dumbness with the corresponding brain areas 44a (word formation), 44b (spoken naming) and 45a (syntactic speech). He revised his earlier views on agrammatic and paragrammatic disorders and emphasised that syntactic dumbness (broadly agrammatism) should be separated from telegrammic style (pure agrammatism) and that excessively fluent speech is not a necessary feature of paragrammatism. We discuss Kleist's contribution to the investigation of grammatical impairments in the next section.

Sentence processing disorders and grammar

Around the turn of the twentieth century something of a paradigm shift took place in psychology and theoretical developments from other schools began to replace the models and theories of association psychology and psychophysics (Kolk, 1994).

Wilhelm Wundt (1832–1920) is considered the founder of experimental psychology. Working in Würzburg, Germany, Wundt (1900: 194) broke with associationism and considered it as psychologically untenable that sentences are simply collections of words – a point already made by Hughlings Jackson.

Later, for other members of the **Würzburg School** (Kolk, 1994: 115–126; Lück, 1996: 65ff.; Mack, 1999; Thorne & Henley, 1997: 201f.) working with **Oswald Külpe** (1862–1915) (Hammer, 1999), the sentence (the statement) was granted significance and inner mental processes were studied through introspection. Thinking was conceptualised in an imageless way (*bildloses Denken*) that leaves no space for the memory 'images' of associationism (similarly to Bergson), and thinking was considered to be more than the manipulation of memory images. Another member of the Würzburg School, **Karl Bühler** (1879–1963), whose language theory later influenced linguistics substantially (Bühler, 1934), concluded that language comprehension is much more than the association of a sound image to a mental representation (Bühler, 1909).

These dramatic developments in psychology influenced aphasiology and grammatical disorders at the sentence level became a focus of interest (De Bleser, 1987). An important protagonist was **Arnold Pick** (1851–1924), who was directly influenced by the Würzburg School, as was the early work of Kurt Goldstein.

In his later work Goldstein, among others, was influenced by developments in another psychological theory, that of **Gestalt psychology** (Kolk, 1994: 91–113; Lück, 1996: 70ff.; Sprung & Sprung, 1999). The discussion of grammatical disorders became multifaceted and complex. On one side there were the proponents of the Wernicke–Lichtheim model and association aphasiology who attempted to incorporate the sentence within the classical single-word model, which, by definition, was difficult. Taking a different view were the representatives of the 'new' aphasiology in the tradition of the Würzburg

School and Gestalt psychology who, among other things, were opposed to Wernicke's view that thinking is unimpaired in aphasia.

Agrammatism and paragrammatism in German-speaking Europe in the early twentieth century

At the turn of the twentieth century in German-speaking Europe, **agrammatism** engendered lively discussion. Kussmaul (1877) had coined the term (see Chapter 4) but it was developed further by Arnold Pick to describe grammatical impairments, and detailed case studies appeared. Significant participants were Arnold Pick, Karl Kleist, Karl Bonhoeffer, Karl Heilbronner, Erich Salomon, Kurt Goldstein, Max Isserlin, and Edmund Forster (De Bleser, 1987; Stark & Dressler, 1990: 283ff.). The years 1913–1914 are pivotal because during this time Pick's (1913) book *Agrammatic Disorders* was published (Heilbronner, 1906: 667), the new aphasiology became firmly established, and Karl Kleist introduced the term **paragrammatism** for the first time, laying the foundations for the subsequent and contentious dichotomy of agrammatism versus paragrammatism.

In his earlier work Pick (1902: 82) proposed that the 'language disorder referred to as agrammatism [. . .] originates from a focal affection of the language area localised in the left temporal lobe', and that agrammatism was a language disorder and not a mental or intellectual disorder, as had been supposed in earlier conceptions of agrammatism since Steinthal.

Karl Bonhoeffer (1868–1948) was Wernicke's assistant in Breslau between 1893 and1898 and his successor in 1904 (Neumärker, 1990; Stertz, 1970), so it is no surprise that he worked in Wernicke's tradition. In contrast to Pick's localisation, Bonhoeffer (1902: 222–223) described two cases that suggest the possible frontal localisation of agrammatism, close to Broca's area, and his opinion is that 'the motoric language centre must be looked at as the "seat" of these grammatical concepts, if one wants to understand the disorder from a localisationist viewpoint at all'. The idea that the motor language centre is the seat of grammar was supported in cases where there was 'a disorder of morphological word structure and sentence construction upon its lesion'. Pick (1913: 254) would later classify Bonhoeffer's cases as 'pseudo-agrammatism' and try to show 'that these are not about real agrammatism'.

Karl Heilbronner (1869–1914) was another assistant of Wernicke's from 1893 to 1898. His opinion was that agrammatism was 'not a secondary result of the aggravation of the motor act of speech, but a primary deficit phenomenon' (Heilbronner, 1906: 683), which can also be observed in writing and sentence completion. But the localisation of agrammatism to the motor language area became plausible to Heilbronner (1906: 683) because his case investigations indicated that agrammatism could occur without comprehension deficit: 'considerable degrees of agrammatism are compatible with hardly damaged, maybe entirely undamaged, comprehension of small parts of sentences and hence of connected speech'.

Based on a detailed case study, **Erich Salomon** (1882–1923) came to the contrasting conclusion that agrammatism was a phenomenon not only of expressive speech, but also of comprehension. Salomon (1914) supposed a disorder of inner speech and of the grammatical system. He proposed two types of agrammatism, one with motor aphasia (localised frontally) and one with sensory aphasia (localised temporally).

Arnold Pick and the primacy of (psycho)linguistics

Arnold Pick (1851–1924; see Figure 5.6) (Brown, 1953; Friederici, 1994; Kertesz & Kalvach, 1996; Sittig, 1925; Spreen, 1973), like Freud, was born in

Figure 5.6 Arnold Pick (1851–1924), who attempted to establish aphasia theory on a new basis by drawing on new developments in psychology and linguistics.

Moravia and attended the University of Vienna. Like Freud and Wernicke, he worked with Meynert and in 1886 was appointed Professor of Psychiatry and Neurology at the German University of Prague (Friederici, 1994). He became a disciple of the Würzburg School and wanted to introduce the new psychology into aphasiology. It was important for Pick (1913) that the developments in psychology and linguistics must form the basis for a new theory of aphasia:

> Not only does the backwardness of the still authoritative psychology for aphasia theory urgently demand a revision, it is also the enormous progress that psychology itself has made [and] the situation in terms of linguistic science presents itself similarly to that of psychology [. . .] of which even the most recent presentations of aphasia theory have not taken notice.
>
> (Pick, 1913: 9)

It is clear for Pick (1920: 66) that 'aphasia theory is in need of a new foundation on the basis of more recent psychology and of the ancillary sciences collected within the psychology of language'. Consequently, Pick approached aphasic language from (in modern terminology) a psycholinguistic perspective.

Pick pointed out that the process of sentence comprehension must be much more than the activation of isolated meanings of words and more than just an association process. For him, 'language comprehension presents itself as a synergetic mechanism that combines a whole series of processes' (Pick, 1909: 30), and he proposed a model covering sound to sentence comprehension that had interactive features (Friederici, 1994: 256).

Pick wrote more on production than on comprehension. His monograph, *Agrammatic Language Disorders: Studies on the Psychological Foundation of Aphasia Theory* (1913; see Figure 5.7) was actually only the first part of a planned multi-volume work but, unfortunately, only the first volume was published. It was dedicated to Hughlings Jackson, 'the deepest thinker in the neuropathology of the last century' (Pick, 1913: III). In his book Pick developed a model of language production that consists of different stages and shares many features with current models (e.g. the contemporary models of Garrett and Levelt). Pick's model is sketched briefly below.

Through 'mental formulation' and 'attitude' (including pragmatic as well as emotional components) a **mental schema** develops, which we would call an intention to communicate or a 'preverbal message' today. Pick (1913: 229) stated: 'The mental schema [. . .] is ready when the linguistic formulation [. . .] sets in.' Subsequently, a **sentence schema** will be activated which takes place before the word choice and

> that the schematic formulation of the sentence precedes the word choice

MONOGRAPHIEN AUS DEM GESAMTGEBIETE DER NEUROLOGIE UND PSYCHIATRIE

HERAUSGEGEBEN VON
A. ALZHEIMER-BRESLAU UND M. LEWANDOWSKY-BERLIN

HEFT 7

DIE AGRAMMATISCHEN SPRACHSTÖRUNGEN

STUDIEN ZUR PSYCHOLOGISCHEN GRUNDLEGUNG DER APHASIELEHRE

VON

DR. ARNOLD PICK
PROFESSOR AN DER DEUTSCHEN UNIVERSITÄT IN PRAG

I. TEIL

BERLIN
VERLAG VON JULIUS SPRINGER
1913

Figure 5.7 Pick's book on *Agrammatic Disorders* was published in 1913. He saw the grammatical disorder as the central symptom of aphasia and analysed it from a psycholinguistic perspective.

> [. . .] is proved by the fact that the meaning of the individual word, which is a very different one, is only determined by the position in which it is used [. . .] thus the mental framework must essentially be ready in terms of grammar too, before the choice of words takes place.
>
> (Pick, 1913: 235)

Pick (1913) saw the evidence for this view in a case of jargon with

> often meaningless sentences, in which the familiar sentence form only serves as scaffolding. The [. . .] assumption that the formulation of word finding and word choice precede, makes it understandable [. . .] that also with disturbed word choice or when other signs are chosen instead of words, sentence formulation still takes place.
>
> (Pick, 1913: 247)

Likewise, the choice of word order and intonation precede word choice, in which Pick very carefully acknowledged the need for speaker intention, communicative context and also pragmatic factors as relevant influences on word order and intonation. At the end of this process grammatical and lexical words are then built into the sentence schema, where the specification of grammatical words (function words and inflections) precedes the specification of content words.

For Pick, agrammatism is the core symptom of aphasia, but he also discussed agrammatism in language acquisition and acquired agrammatism in non-organic psychiatric conditions. Pick (1913: 254ff.) differentiated separate forms of acquired agrammatism that are associated with different stages of the production process. In **isolated agrammatism** there is a disorder of grammatical processing where the sentence schema is not available (or dysfunctional) and utterances are consequently fragmentary (one- or two-word utterances). Additionally, the construction of the mental schema can be disturbed by a cerebral lesion and then no sentence schemata are constructed at all and utterances remain simple for this reason (e.g., interjections, exclamations). **Pseudo-agrammatism** occurs when only essential, emphasised words are produced despite intact thinking and syntactic processing because of a lack of speech drive or will to speak.

Pick also noted that word order is often retained correctly in agrammatic speakers, which he saw as an indication that the sentence schema may be intact. Nevertheless, in order to explain the deficit of grammatical elements (bound and free grammatical morphemes), Pick (1923a) supposed that **economy of effort** may play a role, and he discussed the concept of **emergency language** (*Notsprache*) in detail, which is like an adaptation of the system to post-brain damage. Pick was therefore responsible for introducing the concept of **adaptation** in aphasia. These mechanisms would, by and by, become autonomous and 'the whole mental language apparatus accommodates itself [. . .] extraordinarily fast with the situation created by the

illnesses' (Pick, 1913: 156). Similar views would later also be developed by Isserlin.

Karl Kleist's notion of paragrammatism

In 1914 Kleist described a 'grammatical' impairment he called **paragrammatism**, a second *word order disorder* distinct from agrammatism. Kleist (1914) described the disorder as a feature not only of aphasic language, but also of the language of psychiatric illnesses:

> The disorders at the highest echelon of language construction, of phrases and sentences, are divided into two types in the mentally ill: agrammatism and paragrammatism. Here too, the pathology of the mentally ill is superior to that of focal lesions. The word order disorders occurring in focal lesions have not yet revealed that there are two different types of 'grammatical' disorders. So far we have only spoken of agrammatism. We retain the term agrammatism for one of these two [. . .] word order disorders. The basic trait of agrammatism is the simplification and coarsening of word sequences. Complicated compound sentences (subordination of clauses) are not built. The patients only speak in small, primitive mini-sentences, if they continue to create sentences at all. All less necessary words, especially pronouns and particles, are reduced or eliminated. [. . .] Conjugation thereby also degenerates [. . .].But also the changes occurring in the words themselves, through conjugation, declination, and comparison (flexions in the narrower sense), are more or less omitted.
>
> (Kleist, 1914: 11–12)

In contrast to this pattern, in **paragrammatism**

> the ability to create word orders is not abolished, but phrases and sentences are often wrongly chosen and thereby amalgamate and contaminate each other. Very often, phrases and sentence constructions are not completed: anacolutha result. The spoken expression is not simplified overall; instead, also conditioned by a strong over-production of word sequences, it swells to confused sentence monsters.
>
> (Kleist, 1914: 12)

Kleist considered a mixed agrammatic–paragrammatic symptom pattern to be the rule, and pure cases to be rare.

He was very clear with regard to the anatomical basis (Kleist, 1914: 12): 'We will not go wrong if, contrary to frontal agrammatism, we localise paragrammatism in the temporal lobe or its immediate neighbourhood.' From Kleist's (1914: 12) perspective then, this settled the controversy of 'whether the grammatical disorders are to be relocated in the frontal lobe [. . .] or in the temporal lobe [. . .]. Probably both sides are correct.'

Later Kleist (1916: 170) modified his position and concluded that the cause of agrammatism was 'a loss or lowering of excitability of sentence and phrase formulae', which approximately corresponds to Pick's sentence schemata, and in paragrammatism 'sentence and phrase formulae [. . .] are aroused incorrectly'. The influence of Wernicke's engrams is noticeable when, according to Kleist (1916: 198), paragrammatism is caused 'by an incorrect arousal of acoustic sentence formulae'. Kleist (1916: 170) also discussed 'disorders of the understanding of grammatical word orders' and related those to the production.

Concerning localisation, however, Kleist (1916: 198) thought that all grammatical (i.e., agrammmatism and paragrammatism) disorders can follow a similar lesion in the temporal lobe:

> Disorders of grammatical comprehension as well as the aberrances in grammatical speech, meaning not only paragrammatism but also agrammatism, are caused by [. . .] injuries of the posterior temporal lobe. A connection with foci in the area of the motor speech centre has not yet been proved.
>
> (Kleist, 1916: 198)

Kleist (1918) stated that grammatical disorders never occurred with exclusive lesions of the frontal lobe or of the motor language area, concluding that grammatical disorders are caused by damage to the temporal language area.

Kleist (1916: 198) understood the underlying disorder primarily as a transcortical one resulting from a lesion between the 'acoustic "sentence formulae" and the cerebral tracts of non-linguistic thinking'. On Kleist's model, seeing agrammatism and paragrammatism as symptoms of transcortical disorder allowed him to explain how they could co-occur, how correct utterances were also possible and how language comprehension problems could be expected (De Bleser, 1987: 221).

Following Kleist's work on disorders of grammatical processing, the discussion continued on the topic in Germany. For instance, **Edmund Forster** (1878–1933) presented an interesting case where grammatical impairments occurred exclusively in production, but not in comprehension and without other impairments. The patient had good auditory comprehension, naming and repeating, but hardly any spontaneous speech and the lesion was in the second frontal convolution.

Max Isserlin and agrammatism as adaptation

In the 1920s **Max Isserlin** (1879–1941), also a supporter of the Würzburg School, attempted to bring some order into the extensive discussion of grammatical disorders. In his article 'On agrammatism,' he summarily states that there are 'without a doubt properly distinguishable forms of agrammatic speech' (Isserlin, 1922: 375). On the question of impressive (i.e., receptive)

agrammatism, Isserlin (1922: 376) comments 'that expressive agrammatism does not need to be connected to impressive agrammatism; the latter should rather to be examined as a separate disorder'. Isserlin (1922: 379) saw clear connections between specific syndromes and grammatical disorders: 'Motor aphasia [. . .] and telegrammic style as well as sensory aphasia and paragrammatism, belong together.' The descriptor 'telegrammic (or, more usually these days telegraphic) style' had been in circulation for several years, highlighting the similarities in the elliptic choice of language people used to use to compose a 'telegram' (where each word had a cost) and the pattern of language in agrammatic speech. (Usually the term is 'telegraphic' from US usage, as in 'a telegraph has arrived', and in British English authors sometimes use the term 'telegrammic'.)

In his discussion of underlying psychological mechanisms, Isserlin (1922: 388) stated that 'thought disorders as a cause of agrammatism do not come into consideration' in aphasic patients. Isserlin (1922: 379–380) referred to the observation that agrammatic patients can also show paragrammatism when the 'agrammatic person is forced to (school, special exercise) leave behind telegrammic style and to produce the normal form of language' which led him to develop the hypothesis that telegrammic style is an **adaptation** (Kolk, 1994: 125f.; Kolk, Van Grunsven, & Keyser, 1985):

> pure telegrammic style is not wrong or flawed language. It is rather a widespread form of language that normally appears in the development of human beings, a coarse, simplified language that still meets simple demands correctly. Telegrammic style is [. . .] the form of expression of the primitive, the deaf-mute, of children at certain development levels, and of the normal in certain situations of necessity, for instance with insufficient mastery of a foreign language or a constraint of having to say what is important with very few words (just like in a telegram). It is such a poverty of speech that the motor aphasic person faces, to whom the familiar currency of phrases, formulae, small language elements are not available. [. . .] Seen from such perspectives, telegrammic style would thus be an attitude phenomenon [. . .] originating from poverty of speech. We would thus assume that, on the way from speaking to thinking in telegrammic style agrammatism, the attitude to the 'schema' or the 'Gestalt' of the telegram exists a priori.
>
> (Isserlin, 1922: 394–395)

In favour of this view, Isserlin argued that agrammatic speakers are often able to express themselves grammatically correctly in writing or to classify abnormal constructions as grammatically incorrect.

The situation is different in sensory aphasia (Isserlin, 1922: 403–405): 'We cannot speak of poverty of language ("Sprachnot") in a sensory impaired as we would in a motor aphasic person. He can speak, sometimes even displays overflowing speech.' These patients are able to mentally construct 'in as far as

it is, broadly speaking, a precondition to the linguistic event. But also the linguistic sketch, the linguistic schema, is [. . .] adjusted to normal forms and succeeds, in essential traits, in terms of form.' For paragrammatic speakers, however, the problem is worse than the production of disordered sentences made up of paraphasias. Although the paragrammatic speaker can construct

> the general form of the sentence schema correctly [. . .] errors already take place [. . .] within and [. . .] during this overall structuring, in the sense that complex sub-forms are not determined, shaped, connected, and super- or subordinated in precise correlation with the mental intention, and that, accordingly, displacements, contaminations, substitutions of phrases and sentences disfigure and destroy the function of the sentence despite partially, sometimes even preserved, formal correctness.

Isserlin (1922: 405) explained the grammatical comprehension problems in sensory aphasia due to the deficits in auditory comprehension ('word sound comprehension') making it impossible to process the subtleties of inflections and small words that are required for grammatical comprehension. Isserlin (1922: 408) saw the agrammatic comprehension impairment in motor aphasia as caused by 'loss of grammatical sequence formation' which, according to him, is causally related to the motor language centre.

The demise of classic doctrine

Pierre Marie and Constantin von Monakow led the vanguard against classical connectionism in the early part of the century and Henry Head and Kurt Goldstein especially took up the banner in the period between the two world wars. It was not until the mid-twentieth century that localisationist aphasiology was resurrected by Geschwind.

Henry Head: 'chaos' and critique of the 'diagram makers'

Henry Head (1861–1940) (Critchley, 1961, 1963; Hudson, 1994; see Figure 5.8) occupies an important place in the history of aphasia, mainly because of his criticisms of 'the diagram makers'. Coincidentally, he was born in the same year, 1861, that Broca was describing his first patient Leborgne. He studied at the University of Halle and the German University of Prague, so his German was probably good. Incidentally, he is credited with introducing Association Football to Czechoslovakia and he also wrote poetry.

Already 60 years old, Henry Head published a comprehensive two-volume work on *Aphasia and Kindred Disorders of Speech* in 1926 (Figure 5.9), which has one primary aim, to remove the influence of the 'diagram-makers', as Head called them, from aphasiology.

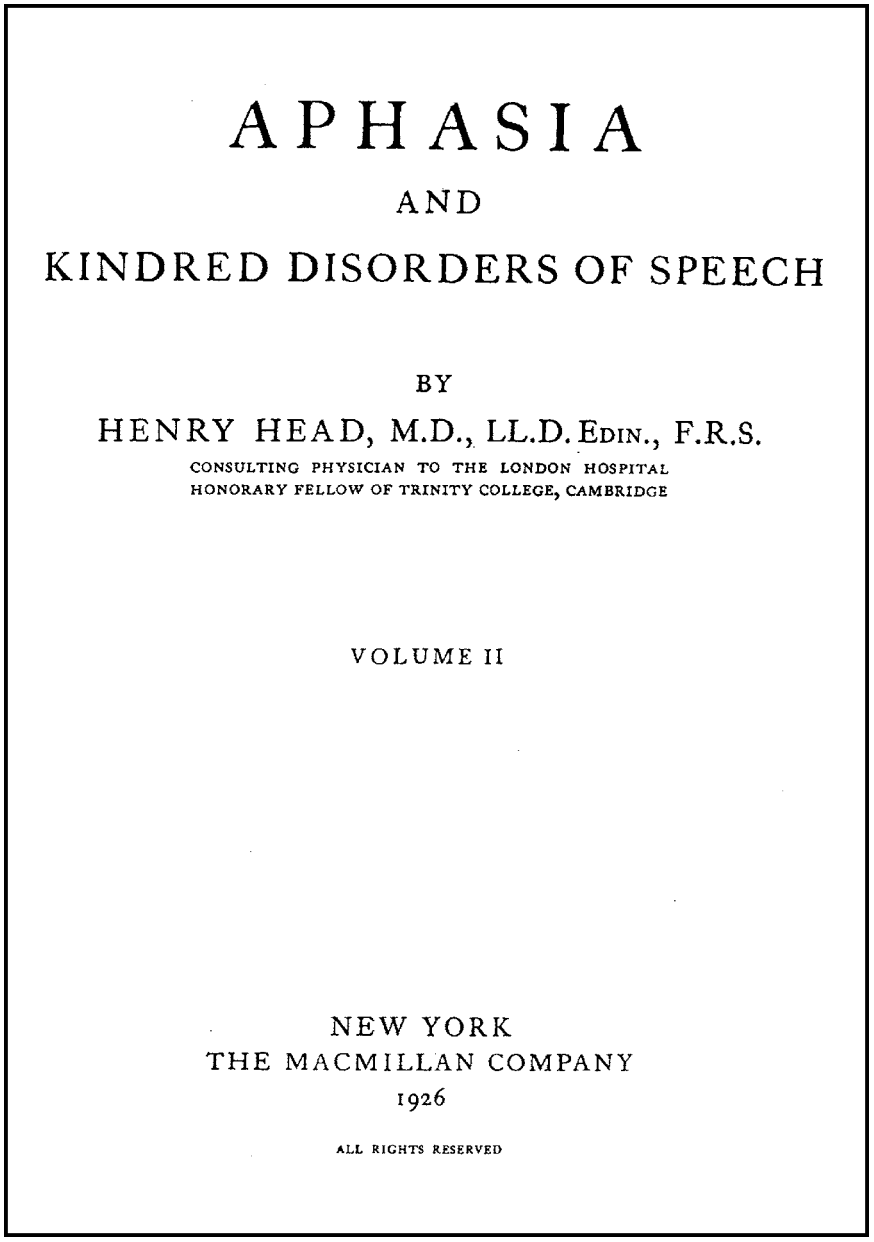
In the historical survey of aphasia that begins Head's book (1926/I: 1–141) the essential features of his views emerged. The headings are labelled: I. From

Figure 5.8 Henry Head (1861–1940), who considered the classic doctrine of the 'diagram-makers' to be worthless.

the schoolmen to Gall; II. Bouillaud to Broca; III. Hughlings Jackson; IV. The diagram-makers; V. Marie the iconoclast; VI. Chaos.

First, Head was a professed promoter of Hughlings Jackson and he had republished large parts of Jackson's papers, making them accessible to a broader readership, and in his book he devoted a long chapter to Hughlings Jackson alone.

Second, Head dealt very critically with the diagram-makers (Bastian, Wernicke, and others) and the conclusion is stark and clear to him: 'Most of the observations mentioned in this chapter [the diagram-makers] failed to contribute anything of permanent value to the solution of the problems of aphasia' (Head (1926/I: 65). Head accused the connectionists of simplified



APHASIA

AND

KINDRED DISORDERS OF SPEECH

BY

HENRY HEAD, M.D., LL.D. EDIN., F.R.S.

CONSULTING PHYSICIAN TO THE LONDON HOSPITAL
HONORARY FELLOW OF TRINITY COLLEGE, CAMBRIDGE

VOLUME II

NEW YORK
THE MACMILLAN COMPANY
1926

Figure 5.9 Henry Head's (1926) seminal book with which an holistic approach to aphasia was proposed as an alternative to the classic model.

assumptions that cannot do justice to the true clinical picture of aphasia. At the same time, the theoretical deductions from these models would lead to simply ignoring essential symptoms.

Third, Head referred to the contemporary situation of aphasia in 1926 as 'chaos'. Geschwind (1964: 215) commented on this: 'Chaos, as is well known, was Head's word and if chaos was perhaps not present before Head, it was certainly fully evident after him.' As illustration, Head lists irreconcilable positions (for example Henschen and von Monakow) or he criticises the concept of speech apraxia (proposed by Liepmann), which he considers to be entirely superfluous. The more holistic works of Kussmaul, Goldstein, Pick, and Marie were seen, with restrictions, more positively.

Head (1926/I: 211) himself described aphasia as a **disorder of the formulation and expression of linguistic and non-linguistic symbols**:

> By symbolic formulation and expression I understand a mode of behaviour, in which some verbal or other symbol plays a part between the initiation and execution of the act. This comprises many procedures, not usually included under the heading of the use of language.
>
> (Head, 1926/I: 211)

Head's notion of linguistic units as symbols is related to the tradition of Finkelnburg's asymbolia and Head also discussed problems of calculation (**acalculia)** and symptoms that others (e.g., Liepmann) had classified as apraxia.

Like Marie, Head thought that a **disorder of intelligence** occurred in aphasia. Head conceded, however, that it is not *general* intelligence that is impaired in aphasia, but rather that certain verbal mental activities are no longer fully possible:

> For a man who, in the course of general conversation, is unable to express his thoughts, or comprehend the full significance of words and phrases, cannot move freely in the general field of ideas. [. . .] Moreover, it must not be forgotten that the intellectual life of civilised man is so greatly dependent on speaking, reading and writing, that any restriction of these powers throws him back upon himself. [. . .] This inevitably leads to a diminished field of thought and many aphasics gradually deteriorate in mental capacity.
>
> (Head (1926/I: 211)

Head distinguished between four aphasic 'defects': verbal, syntactical, nominal and semantic. **Verbal defects** display themselves

> primarily through defective word formation. The patient is unable to find the words that he requires for normal day-to-day communication; in the worst cases, someone can be reduced to 'yes' and 'no', together with a

> few expressions that he uses automatically or only under the influence of emotion. Such a heavy disorder of spoken language is always accompanied by a loss of the ability to write and an impairment of the verbal memory for the content of sentences that have been read.
>
> (Head, 1926/I: 221)

A patient with verbal aphasia is able, however, to understand individual words (object names, colours) and can also follow a simple conversation, 'under condition that the sentences are not unusually long and complicated' Head (1926/II: x). Writing is also impaired.

Syntactic aphasia 'is characterised by a more or less severe disorder of rhythm and syntax. The patient speaks quickly, he speaks jargon, and prepositions, conjunctions, and articles tend to be omitted; polysyllabic words are slurred and pronounced badly' (Head, 1926/II: xiv). These symptoms occur in spontaneous language, while repeating, and while reading aloud. Writing to dictation is impaired and spontaneous writing is restricted to simple structures. The comprehension of spoken and written language is restricted because of the restriction of 'rhythmical aspects of symbolical forming of the symbolic expression' (Head, 1926/I: 240) which shows itself at the sound level as well as at the sentence level.

In **nominal defects** there appear to be more than just problems with language processing: 'we are not dealing with a difficulty in shaping word and phrases, but with a disturbance of their nominal significance' (Head, 1926/I: 240). The patient 'has many words at his disposal, but he cannot apply them exactly, and the verbal form can be affected by his attempts to find the correct term' (Head (1926/II: xvi). Written and spoken requests are only partially understood. Reading comprehension is generally limited; and writing also displays impairments. In addition, use of number and arithmetic functions are affected. Patients with nominal aphasia cannot interpret musical notes and they also have problems in drawing maps.

The last type is **semantic aphasia** which is 'characterised by a lack of recognition of the full significance of words and phrases apart from their immediate meaning. The patient fails to comprehend the final aim or goal of an action' (Head, 1926/II: xix). These patients also have orientation and planning difficulties, which significantly affect everyday life, although 'memory and intelligence can remain at a comparatively high level' (Head, 1926/II: xx).

Head acknowledged that his aphasia types were not a new classification, but rather that they represented pure forms that seldom occur in their pure form. For Head it was important to examine the performance of aphasic people comprehensively and also under varying conditions. The call for a comprehensive examination of the functions of aphasic patients was not new, but acknowledged by most as appropriate and accounted for much of the initial acceptance of Head's views. Head designed a comprehensive linguistic and non-linguistic test battery, although its development did not include comparison with a control group (which was not a common practice in the

psychometric research of the time in general). Critchley (1961) stressed exactly this fact:

> One particular defect of Head's battery was that, like so many other aphasiologists, he fell into the error of making no control studies. The unwisdom of this neglect is demonstrated when Pearson, Alpers and Weisenburg submitted a group of normal college students to Head's battery of tests, and found that a significant percentage scored badly.
>
> (Critchley, 1961: 558)

Head's attack on the classic dogma was most damaging although he contributed more than his famous critique of the diagram-makers. His terminology was used in Britain for some years, although it never became widely established, Head's critique may have modern relevance too according to Hudson (1994):

> Whether or not one agrees with Head's specific analyses and criticisms [. . .] Head's own words represent a refreshing, and readable, blast of fresh air. Perhaps the most convincing test [. . .] would be to replace the specific models and data attacked in his chapters with models and data drawn from today's work on aphasia [. . .] then perhaps we should consider carefully just what sort of traps and errors of thought we are propagating today.
>
> (Hudson, 1994: 288)

Kurt Goldstein's 'organismic' approach

Geschwind (1964: 218) wrote that Kurt Goldstein 'is often regarded as the greatest influence in the revolt against the classical school of thought about aphasia'. Oliver Sacks (1995: 7) wrote of Goldstein that he 'is one of the most important, most contradictory, and now most forgotten figures in the history of neurology and psychiatry'. This assessment is not really too absurd, since, for instance, among the 3000 references in a comprehensive survey of neurolinguistics (Stemmer & Whittaker, 1998), Goldstein does not get a single mention. Egon Weigl, a student and collaborator of Goldstein's in Frankfurt agrees with Luria (1966) that Goldstein should be 'regarded as the founder of modern neuro-psychology' (Weigl, 1968: 144)

Kurt Goldstein's (1878–1965; see Figure 5.10) life path parallels that of aphasiology from the nineteenth century until after the Second World War (De Bleser, 1994; Goldstein, 1971; Harrington, 1996: 140ff; Wallesch, 1988: 158f.).

Like so many influential aphasiologists, as we have seen, Goldstein studied under Wernicke but turned away from connectionism and what he called 'atomistic aphasiology' and under the influence of the Würzburg School and Gestalt theory developed his own holistic approach, **organismic aphasiology**. This approach was strongly affected by his work as head of the Institute for

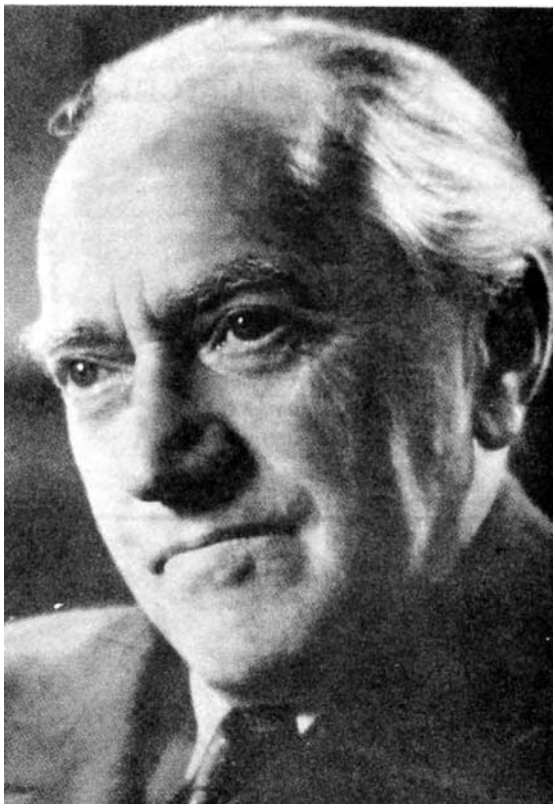


Figure 5.10 Kurt Goldstein (1878–1965), whose life path parallels that of aphasiology from the nineteenth century until after the Second World War.

Research on the After-Effects of Brain Injury in Frankfurt, where he dedicated himself to the rehabilitation of brain-damaged soldiers from the First World War. In 1933 Goldstein had to emigrate with his wife Eva Rothmann, as he had been denounced by the Nazis as a Jew and because of leftist sympathies (De Bleser, 1994), and went first to the Netherlands and then to the USA, where he remained until his death in 1965. Interestingly, his escape was made possible by Hermann Goering's cousin, who was a psychiatrist and a colleague.

In his early works Goldstein (1910) criticised Wernicke's approach, whose atomistic centre theory (with cortical word centres) he refuted as unsatisfactory. According to him, a word is significantly more than the association of auditory and motor memory images, and 'the distinction between sound memory images and speech motor images [is] to be rejected as unpsychological' (Goldstein, 1910: 17). Instead, Goldstein considered as central the

concept (the meaning, the linguistic idea) of the word, which (in the sense of the Würzburg School) does not entail a 'memory image':

> The essence of the linguistic image, which is common to all men, is something specifically distinct from the acoustic-motor, of whose peculiarity we are as aware as it is impossible to define it any further. It shares this peculiarity with all other specific elements of consciousness, the same as with the image of spatiality and it is, in my opinion, no more mysterious than the fact of simple sensation.
>
> (Goldstein, 1910: 17)

Because of the fact that the 'linguistic image' can be conscious without 'the acoustic and motor areas of the cortex being active, as it is the same whether we hear or speak, so it can not possibly be localised either acoustically or sensorially' (Goldstein, 1910: 18).

The implication for an approach like Wernicke's (with its different brain centres) was thus clear to Goldstein (1927):

> The theory of language centres in the form of a circumscribed localisation of isolated functions can be considered as invalidated. The entirely circumscribed localisation of aphasias has become highly problematic as well, after one has been forced to recognise that the assumption of circumscribed lesions as a necessary cause for the development of certain disorders certainly does not rightfully persist.
>
> (Goldstein, 1971: 155)

An underlying problem for Goldstein (1927) was the rash classification of insufficiently surveyed data on only superficially recorded anatomical details:

> If one described the symptom pictures at hand this insufficiently, [. . .] then the localisations had to turn out imperfect as well and the contradictions and discussions had to escalate. [. . .] the localisation by layers of the brain cortex was overlooked virtually completely; it was overlooked that the symptom picture depends on the individuality of the patient in psychological and physical terms, especially on the composition of the entire remaining brain; one lastly overlooked the extraordinary incertitude [. . .] that we actually do not know in which relationship a certain composition of the anatomical substrate stands to a certain function.
>
> (Goldstein, 1971: 171)

For this reason, classifications that rest on the occurrence of certain symptoms (core symptoms) in classical syndromes do not seem to Goldstein to be the optimal way to measure a patient's functions and behaviours and he is a proponent of individual measurement of performance in different situations and tasks: 'As a result his case descriptions show remarkable attention

to detail in the patients' performance in a wide range of tasks' (Howard & Hatfield, 1987: 46).

Goldstein considered Hughlings Jackson to be the first representative of the organismic approach, as Jackson had refuted the deficit-orientated approach of the connectionists (Goldstein, 1927, 1934, 1939, 1948). The reason for Goldstein (1927, in Goldstein, 1971: 164) is the following: 'one moment must not be overlooked here either. Man is a psycho-physical organism. Each disease changes him in his entirety.' For aphasia it follows that not only the linguistic deficit has to be considered, but much more than that:

> In pathological cases, we are not just dealing with an aphasic person as with a man with altered speech, but instead with a man whose alteration manifests itself to us in certain phenomena of his speech, but also in the most varied other phenomena. Thus one should never contemplate a phenomenon in isolation from the entire sick person.
>
> (Goldstein, 1927, in Goldstein, 1971: 163)

This integral (holistic) point of view also leads to an approach to symptoms without bias: 'One should take all phenomena into consideration that a patient displays, and not initially give any of them priority for assessment. There are no important, no unimportant ones at first' (Goldstein, 1927, in Goldstein, 1971: 164). Goldstein's approach is echoed in the contemporary World Health Organisation's International Classification of Functioning, Disability and Health (WHO, 2001).

Readjustment and **compensatory mechanisms** were important for Goldstein, as they were for von Monakow:

> These readjustments represent a resort that the diseased organism chooses in order to meet the demands still made of it despite the functional deficit. [. . .] Only the exact analysis of the detours [. . .] brings us [. . .] some understanding of some incorrect responses that are often erroneously perceived as direct consequences of damage to a specific function, while they are an expression of actually entirely normal functions, which only produce flawed effects because the sick person uses, i.e. must use, a path that would result in the same, effectively bad performances in a healthy person.
>
> (Goldstein, 1927, in Goldstein, 1971: 167)

This is related to Goldstein's concept of **catastrophic reaction** that can occur when a patient becomes overtaxed, for example when unable to solve some task (see Goldstein, 1942, 1948: 10ff., 1995). A catastrophic reaction can result in aggression, defence, denial, fear, and other reactions. Consequently, the person affected may display behaviour and functional impairments that strongly diverge from their usual abilities. And, 'in such catastrophic situations, not only the specific functions are impaired, but attentiveness, memory,

and the so-called basic functions are also impaired' (Goldstein, 1948: 12). Goldstein understood the human body as constantly and unconsciously attempting to avoid situations in which catastrophic reactions could occur. Conscious and unconscious adaptations and compensations are important for the organism to achieve its goal of normal functioning, or, with brain damage, as 'normal' as is possible. A protective mechanism with extreme effects would be a total withdrawal from the world, because every stimulus and experience could cause a catastrophic reaction.

Goldstein considered the **loss of 'abstract attitude'** as an essential 'basic disorder' of aphasia (Gurwitsch, 1971: XVIIIff.). Abstract attitude can be understood when contrasted with its antithesis, **concrete attitude**. Goldstein (1948) describes the difference as follows:

> We can distinguish normally two different kinds of attitudes which we call the concrete and the abstract. In the concrete attitude we are given over passively and bound to the immediate experience of unique objects or situations. Our thinking and acting are determined by the immediate claims made by the particular aspect of the object or situation. For instance, we act concretely when we enter a room in darkness and push the button for light. If, however, we desist from pushing the button, reflecting that by pushing the button we might awaken someone asleep in the room, then we are acting abstractively. We transcend the immediately given specific aspect of sense impressions, we detach ourselves from the latter and consider the situation from the conceptual point of view and react accordingly. Our actions are not so much determined by the objects before us as by the way we think about them; the individual thing becomes a mere accidental example or representative of a 'category'. Therefore, we call this attitude the categorial [. . .] attitude.
>
> (Goldstein, 1948: 6)

The abstract attitude makes the following functions possible:

- One can instigate a conscious action deliberately, for instance take the initiative or perform a function when ordered.
- One can deliberately change from one perspective to another and/or choose among different possibilities.
- Different aspects of a situation can be considered simultaneously, even if they do not directly belong together.
- The essence of a whole can be perceived but individual parts of the whole also considered in isolation.
- Common characteristics can be abstracted.
- One can plan in advance, and one can think and act symbolically.

For Goldstein aphasic people have a specific problem with abstract attitude. The difficulties an aphasic person has with carrying out desired functions

in linguistically complex tasks were understood by Goldstein as based on a reduction of abstract attitude.

Goldstein was one of the first to call attention to the 'difficult post-morbid situation' (the psychosocial consequences of aphasia) and to the difficulties of those affected in the clinical situation and was an advocate of the exemplary individual case study:

> Here too, we followed the principle that the years of dealing with psychopathological problems has led us to: to bring about the clarification of a problem not by summary examination of a large number of cases, but rather by understanding an individual case that we deem appropriate.
>
> (Goldstein & Gelb, 1924, in Goldstein, 1971: 62)

Despite his criticism of Wernicke and of the classic typology, and despite his emphasis on individual studies, readjustment, and the holistic changes of the organism caused by brain damage, Goldstein went along with Wernicke's paradigm to some extent, in that he acknowledged close relationships between symptoms and lesion location. And he used a classification system that was highly compatible with Wernicke's (Goldstein, 1948: 148ff.). This led to the view that Kurt Goldstein occupies a paradoxical position in the history of aphasia. Goodglass (1993) pointed out that

> Kurt Goldstein occupies a dual role in the recent history of aphasia. [. . .] even his latest major work on aphasia (1948) presents a classification and view of anatomic localisation that accepts that general framework. [. . .] At the same time, Goldstein is regarded as one of the strongest voices of the noetic [holistic] movement.
>
> (Goodglass, 1993: 25)

A new paradigm emerges from North America

Although a number of scientists in the United States had contributed individually to aphasiology (e.g. Mills, Meyer), their contributions had no great influence on the main thrust of international aphasiology until after the Second World War when the situation changed dramatically. However, there was a major exception, the book by Weisenburg and McBride (1935) heralded a new era in the history of aphasiology, and the beginning of a new paradigm emerged from North America in the 1930s in which there was little, if any, room for localisation.

Weisenburg and McBride: a clinical solution

Neurologist **Theodore Weisenburg** (died in 1935) and psychologist **Katharine McBride** approached aphasia from a purely clinical perspective and conducted the first large and comprehensively controlled study of aphasia. They

considered that established theoretical models were preoccupied with localisation and the differing syndromes of asymbolia, aphasia, apraxia, and so forth, and found especially that the obsession with classification was confusing and less than useful:

> Nowhere in aphasia literature is confusion more evident than in the classification of types of disorders. There are differentiations on anatomical, physiological, and psychological grounds and on various combinations of these.
>
> (Weisenburg & McBride, 1935: 47)

Weisenburg and McBride's study was developed between 1929 and 1935. Their 'psychological' paradigm entailed an appreciation of normal and disturbed information processing of behaviour using psychological rather than anatomical models. They acknowledge the inspiration of Hughlings Jackson: 'He is generally credited as being the first to realise the value of the psychological study of aphasia and the primary importance of the exact clinical findings' (Weisenburg & McBride, 1935: 19).

Weisenburg and McBride therefore attempted to detect, independently from established theoretical models, the features and phenomena of aphasia. To carry out this venture, the aphasic participants must of course be assessed with 'a satisfactory system of examination', acknowledging Head's recommendations.

Head's test battery was designed so that non-aphasic participants also produced errors. Hardly any attention had been paid in earlier studies to the question of what a 'normal' function or performance was, and how one could therefore determine with any accuracy the extent and quality of aphasic errors and patterns of performance across tasks: 'In the many performances where [. . .] errors appeared, the question became: What is the normal performance? And for the majority of tests, no answer to this question was available from previous work' (Weisenburg and McBride, 1935: 3).

For this reason, Weisenburg and McBride also tested a control group of healthy individuals to establish a normal level of performance. The idea to draw upon a control group matched to the aphasic samples in terms of age, education, and occupational background, was a novelty at this time in the history of aphasia:

> In order to interpret the results for the aphasic patients, therefore, it was necessary to extend research and include a control group of normal adults. The selection of these so-called normals was arranged to make the group as closely comparable to the aphasic group as possible. Educational and occupational data show that the normal group is not only similar to the aphasic, but that it represents a good sample of the population at large.
>
> (Weisenburg & McBride, 1935: 3)

In addition, a group of brain-damaged male and female participants without aphasia were also tested to determine the aphasia-specific and the aphasia-non-specific consequences of brain damage. Altogether 234 participants (male/female) and 85 adult controls were examined. Clear selection criteria were applied for all participants in all groups: all participants were aged below 60 in order to exclude any possible effects of old age, no participant had a mental health problem, and all were native speakers of English with no hearing or visual impairments.

Weisenburg and McBride (1935: 141–142) started with a broad and theoretically neutral definition of aphasia: Aphasia 'must [. . .] be understood to include a variety of psychological changes occurring with a unilateral cerebral lesion and appearing chiefly but not altogether in language processes'.

For this reason, their assessment battery comprised verbal and non-verbal tests. The tests are summarised in Figure 5.11, which shows that the battery contained sections testing speech production, naming, repetition, understanding, reading aloud, reading comprehension, writing, arithmetic, language intelligence, memory span, retelling a story, as well as non-verbal tasks.

Weisenburg and McBride (1935: 473) summarised their results for people with aphasia: 'The chief psychological changes consist of: a) predominant disturbances of language; b) disturbances in so-called non-language activities; and c) alterations in common everyday activities and social behaviour.' Furthermore, the authors devised a 'simple and relatively easily followed classification with the four divisions of expressive, receptive, expressive-receptive, and amnesic types of disorder' (Weisenburg & McBride, 1935: 472).

Patients with **predominantly expressive disturbances** display

> 1) defects in the articulation and formation of words with more or less marked alterations in the structure of the sentence; 2) verbal and structural confusions resulting in the erroneous use of words or grammatical forms; 3) difficulties in the evocation of words as names for objects, conditions, or qualities.
>
> (Weisenburg & McBride, 1935: 211)

In **predominantly receptive disturbances** are observed 'serious limitations in the understanding of spoken language or of printed material [. . .] disturbances in expression. The latter are chiefly verbal and grammatical confusions' (Weisenburg & McBride, 1935: 147), to which are added word finding disturbances and compensation strategies. In patients with **expressive–receptive disturbances** 'all speech functions are impaired more or less equally. Non-linguistic performances are usually only a little better in these cases, but sometimes they are far superior to the linguistic performances' (Weisenburg & McBride, 1935: 147). The difficulties of **amnestic cases** 'nearly exclusively come from the inability to recall the corresponding words for objects, situations or characteristics. The receptive functions are relatively intact'

SPEAKING

Records of spontaneous speech and reactive responses
Automatic word series: saying days of week and months of year; saying the alphabet; reciting a prayer or poem

NAMING

Naming of standard objects, colors, and line drawings

REPEATING

Words containing all English sounds, and short familiar phrases and sentences

UNDERSTANDING SPOKEN LANGUAGE

Response to everyday questions and comments
Comprehension of words and sentences, as shown by response to Gates Word, Phrase, and Sentence Reading Test, read aloud to the patient
Response to Test of Following Directions
Response to tests involving the comprehension of spatial terms and relationships, including Head's Hand, Ear, and Eye Test and the Abelson Geometrical Figures Test

READING

Oral: Gates Word Pronunciation Test
Gray Oral Reading Paragraphs
Reading comprehension: Gates Primary Reading Tests
Chapman Unspeeded Reading-Comprehension Test
Thorndike McCall Reading Scale

WRITING

Records of spontaneous writing
Writing to dictation
Word dictation: Morrison McCall Spelling Scale
Sentence dictation: Stanford Achievement Dictation Test
Oral Spelling: List of words from Gates
Copying: Standard paragraph

ARITHMETIC

Computations: Stanford Achievement Arithmetic Computation Test
Problems: Stanford Achievement Arithmetic Reasoning Test

LANGUAGE INTELLIGENCE TESTS

Controlled Association Tests
Opposites
Mixed Analogies
Sentence Completion Test: Kelley Trabue Completion Exercises
Absurdities
Vocabulary
Oral: Stanford Binet Vocabulary Test
Printed: Thorndike Test of Word Knowledge

REPRODUCTION OF VERBAL MATERIAL

Reproduction of digit and letter sequences, series of nonsense syllables, disconnected words, sentences, and a short story

NON-LANGUAGE TESTS

Formboards, imitation tests, digit-symbol substitution tests, drawing and picture completions and reconstructions: Pintner Paterson Performance Scale and Pintner Non-Language Mental Test
Drawing
Figures reproduced immediately after presentation
From model: drawing of a chair
Drawing of a man scored on the Goodenough Scale

Figure 5.11 Overview of Weisenburg and McBride's (1935: 135–136) test battery.

(Weisenburg & McBride, 1935: 147). Non-linguistic functions are almost entirely preserved.

Using this simple system, the 'classification problem' was thus solved satisfactorily for Weisenburg and McBride. With regards to localisation, although

lesions in certain regions (approximately the peri-Sylvian language area of the left hemisphere) cause aphasia, any significant brain damage has consequences for non-verbal processing too.

Weisenburg and McBride's seminal work in the 1930s established a group-study approach to research in aphasia, entailing carefully selected control groups and brain-damaged but non-aphasic groups. Theoretical development of aphasia did not advance with Weisenburg and McBride, and hypotheses on the causes of aphasic symptoms were not formulated. Eventually, their classification was seen to be too simplistic to be clinically or theoretically useful.

At the same time separate localisationist-neurological and clinical-therapeutic disciplinary groups with different objectives developed in North America: a trend that spread throughout the aphasiological world. Tikofsky (1984) stated:

> Weisenburg and McBride [. . .] created a division in the study of aphasia. That division led to a trend whereby psychologists and speech pathologists directed their attention to the behavioural aspects of aphasia, while neurologists continued to seek clues as to the localisation of the various forms of aphasia.
>
> (Tikofsky, 1984: 14)

The emergence and early development of therapy for aphasia

Although there had been sporadic attempts over the centuries to treat aphasia, aphasia therapy only really began to develop as the nineteenth century turned to the twentieth (for an excellent survey of the history of aphasia therapy, see Howard & Hatfield, 1987). In the German-speaking countries it was particularly phoniatricians, who are specialist otorhinolaryngologists with particular expertise in voice and speech disorders (for example Gutzmann in Berlin and Froeschels in Vienna), who, in developing treatment for people with voice and speech impairments, helped to put therapy for aphasia onto a firmer basis. The First World War left many survivors with brain damage and therapeutic approaches developed and increased dramatically as a result. Besides Berlin and Vienna, new rehabilitation centres appeared in other German-speaking cities or were created within existing institutions, significant ones were Munich (Max Isserlin) and Frankfurt (Kurt Goldstein).

Hermann Gutzmann and 'speech gymnastics'

Phoniatrician **Hermann Gutzmann** (1865–1922; see Figure 5.12) had been treating aphasia since the early 1890s (e.g., Gutzmann, 1896). He is regarded by some as 'the father of language therapy' (Howard & Hatfield, 1987) and was significantly involved in the foundation of aphasia therapy as a discipline. Leischner (1998: 353) stated that Gutzmann 'showed in his many works that aphasiology cannot fulfil its task without the simultaneous treatment of

Figure 5.12 Hermann Gutzmann (1865–1922), the 'father of language therapy'.

aphasic persons. Through the combination of diagnostics and therapy, he promoted it to the rank of clinical discipline.'

Although he published the results of his therapeutic efforts, Gutzmann (1924: 357) was aware that it is unclear whether natural recovery or the therapist were responsible for improvements: 'Attempts at curing the aphasic have always been undertaken, sometimes with such noticeably swift success that the physicians justifiably said that [. . .] the aphasia most probably would have healed by itself without their assistance.' Gutzmann (1924) therefore advocated a now well known control to separate the effects of spontaneous recovery from therapy:

> For the therapy of aphasic states, it depends on which state the aphasic

> person is in. Language therapy as such can only ever be undertaken when a relatively quiescent state of the patient has been reached. [. . .] It is my own custom to take aphasic persons into my linguistic treatment at the earliest half a year after all violent symptoms have ended.
>
> (Gutzmann, 1924: 296)

At the basis of Gutzmann's therapeutic efforts lay a comprehensive diagnosis and he developed a comprehensive test battery, the nature of which can be gleaned from the **audit form** for his aphasia examination (Figure 5.13).

Gutzmann approached the therapy of motor, sensory and amnestic aphasia systematically (see Figure 5.14 for an example). His *Systematic exercise therapy* (Gutzmann, 1924: 308) distinguished between different steps in the individual modalities and involved a complex system with specific sequencing of exercises. The foundations for Gutzmann's approach were foreign language learning and primary school pedagogy, which is why the approach is often called 'speech gymnastics' and/or the 'didactic approach' (Howard & Hatfield, 1987: 28).

Like Gutzmann, **Emil Froeschels** (1884–1973) too was a phoniatrician (Black, 1980; Howard & Hatfield, 1987: 32ff.; Rieber, 1980). He worked in Vienna and was a follower of Liepmann's contemporary development of Wernicke's theory. Froeschels treated about 2000 brain damaged patients between 1916 and 1925 in his 'ambulatory (clinic) for speech and voice disorders', where aphasic people were also treated on a regular basis (Froeschels, 1913: 168ff., 1914, 1925). He collaborated closely with school teacher **K. C. Rothe** (1880–1931) and both used an approach similar to Gutzmann's.

For motor aphasia Froeschels (1913: 184) suggested strengthening of articulatory skills and in 'the treatment of sensory aphasic persons it is a matter of reawakening the understanding for sounds and words'. Froeschels' method features systematic hierarchical sequencing principles and repetition. It consists in

> saying individual sounds behind the patient and, [. . .] if he does not repeat the sound correctly, showing him the corresponding mouth position. When he imitates that, the correct sound, whose sound image he begins to memorise again, is thus produced. Through sufficiently frequent repetition of this exercise, one should, in the utmost number of cases, succeed in making the patient perceive the sound coming from the ear anew. After sounds follow sound combinations, then words and sentences in turn.
>
> (Froeschels, 1913: 184)

Developments in rehabilitation in English-speaking countries

In Britain too approaches to treatment of aphasia were based often on those developed by elocution and voice teachers. Bastian (1898) seems to have been an exception and emphasised the difference between compensation and

Name		Date
Age		Number
Profession	Profession of the father	
School, year		
Address		Diagnosis

I. Anamnesis:

1. Hereditary taint:
   Diseases of the parents:
2. Birth:
3. Siblings:
4. Prior diseases:
   Lifestyle:
5. Dentition:
6. Learning to walk:
7. Learning to speak:
8. Onset of illness:
9. Cause, reason:
10. Same or similar illness in the family:
11. Treated earlier:

II. Status:

A. General examination:

a) Physical characteristics:

12. General physical condition: (reflexes):
13. Scrofula, rickets:
14. Skull and face formation:
15. Body height and weight:
16. Physical dexterity:
    Apraxia test:
    Testing for sinistrality:
    Gait:
17. Bad habits:
    Parakinesia (and automatic movements):
18. Digestion:
    Sleep:
19. Heart:
20. Lungs:
21. Chest measurement:
    Chest space:
22. Respiration:
23. Duration of exhalation:
24. Respiration volume:
25. Pneumatometer measurement:
26. Lips, lower jaw:
27. Palate:
28. Teeth:
29. Tongue:
30. Nose:
31. Throat:
32. Larynx:
33. Hearing:
34. Eyes:

b) Mental abilities:

35. Predisposition:
    Intelligence test:
    Mirror writing left:
36. Fallen behind in school:
37. Attention:
    Fatigability:
38. Recognizing objects by view:
39. Recognizing objects by hearing:
40. Recognizing objects by touch:
41. Recognizing objects by taste: and smell:
42. Temperament:
43. abnormal tendencies:

B. Speech test:

35

c) Perception of speech:

44. Sound, vocal, word, sentence hearing:
45. Reading off speech movements:
46. Comprehension of what is spoken:
    a) Understanding of a request for actions:
    b) Indicating named objects:
    c) Indicating the number of syllables of spoken words:

Figure 5.13 Gutzmann's (1924: 124–125) audit form for his examination of aphasia.
(*Continued overleaf*)

47. Reading comprehension for what is written: Reading comprehension for what is printed: 48. Taking dictation: 49. Understanding facial expressions and gestures: b) Production of speech: 50. Repeating sentences: Repeating words: Repeating syllables: Repeating sounds: 51. Articulation of sounds: a) Vowels: b) Voiced plosives: c) Voiceless plosives: d) Fricatives: e) L-sounds: f) R-sounds: g) Nasal sounds: 52. Dexterity of speech musculature: 53. Speech respiration: 54. Speaking voice: Height of speaking voice: 55. Spontaneous speech: 56. Naming of indicated objects: 57. Reading aloud what is written: Reading aloud what is printed: 58. Spontaneous writing: 59. Copying: 60. Mechanical copying: 61. Whispering speech: 62. Singing: 63. Musical disorders: 64. Sign language:	V. Therapy: VI. Course of treatment: VII. Result of treatment:
III. Diagnosis:	VIII. Special remarks:
IV. Prognosis:	

Figure 5.13 Continued.

Step	(a) Speaking	(b) Writing	(c) Reading	(d) Calculating	(e) Drawing	(f) Games
I	**Imitating sound positions**; monophthongs, sound sequences, simple, meaningless syllable sequences. Repeating values. **Practising in front of a mirror.** For sensory and amnestic aphasic patients: **Lip Reading**	**Left-handed longhand** (also when right-handed longhand writing is possible): Single letters, upper and lower case, following models, into a copybook or onto cardboard tablets, and collects them.	**Print and handwriting,** single letters			Placing pieces (e.g., chess pieces) following models
II	**Repeating**: words, short sentences **spontaneous speech**: object and picture naming, at the same time training recognition and training the memory of amnestic patients. For sensory and amnestic aphasic patients: **sign language**, simple.	**Left-handed longhand: following a model** words, small sentences. Dictation: single letters, short words, phonetic writing.	**Print and handwriting,** words, simple word combinations, small sentences from a spelling book, letter games. Printed 1a	**Counting fingers**, counting beads on an abacus, addition and subtraction following Bohny's picture book.	**Left-handed drawing** and/or also right-handed **geometrically** following a model: line, triangle, quadrangle, rectangle, square, circle.	Token games from memory. Dominoes
III	**Spontaneous speech**: **simple syntax** following picture board, **naming small actions, Sign language**, more complicated.	**Left-handed writing**: Copying, sentences from a spelling book, dictation, small sentences, **spontaneous writing**, written naming of objects from nature and pictures	**Short reading passages, Ebbinghaus'** test reading passages with omissions	**Graphically**: multiplication division following B o h n y' s picture book. **In writing**: addition and subtraction, see II d. **Mental arithmetic**: Addition and subtraction, see II d, counting backwards	**Left-hand drawing** and/or also right-hand: **By memory** repeat exercise II e. **Following a model**: simple, perspective	Dice, nine men's morris, halma
IV	**Spontaneous speech: description** of images, **retelling a story**, **Description** of complicated actions	**Left-handed writing**: copying short reading passages, **dictation** of short reading passages, **Spontaneous writing**, small sentences, describing simple images	**Longer reading passages**, extended Ebbinghaus' test, **puzzle solving.**	**Mental arithmetic**, addition and subtraction, several-digit. **In writing**, multiplication, division several-digit. Applied arithmetic in simple examples	**Copying** simple model **Drawing** simple objects in perspective. **From memory**	Card games, chess, parlour games
V	**Spontaneous speech: narration** of longer reading passages from level IV e, **report** on experiences from holiday, past times, the field and similar. Telephoning.	**Left-hand writing: writing down reading passages** from memory. **Description** of experiences (see V a). Messages, writing short reports, writing letters, typewriting.	**Reading**, unknown handwriting, reports and letters, collate.	**Applied arithmetic**	**Freehand drawing**	Card games, chess, parlour games

Figure 5.14 Exercise plan for speech and language therapy of patients with aphasia (following Gutzmann, 1924: 299).

restitution. He described therapy for aphasic disorders in some detail (for detailed review, see Howard & Hatfield, 1987), which he claimed was based on the potential of reorganisation of the right hemisphere to take on language functions through the process of **functional compensation**, which he distinguished from **functional restitution**, which occurs with natural recovery. This fundamental distinction is still an axiom of modern neuropsychological approaches to recovery and rehabilitation (see Code, 1987, 2001), and not limited to a role for the right hemisphere. Bastian was impressed by a case treated by Bristowe (1880) using a 'full oral method', which subsequently spread to the *Salpêtrière* in Paris (Howard & Hatfield, 1987). Bastian's approach was also based on methods originally developed for teaching the deaf and delayed speech and language in children (Howard & Hatfield, 1987). Wyllie in Britain developed a phonetically based 'physiological alphabet' and in the USA the neurologist **C. K. Mills** and his assistant Weisenburg employed Wyllie's physiological alphabet and the methods of Gutzmann and Froeschels with an emphasis on articulatory drills and repetition.

There was little or no attempt to evaluate the effectiveness of therapy for aphasia at this time, but in California, **S. I. Franz** (1906, 1924) was developing more systematic methods of reporting the progress of the people he was treating by detailed description and recording of change. Howard and Hatfield describe his approach for naming problems, which entailed the selection of 60 objects, of which 20 would be chosen for treatment, with the remainder used for testing and re-testing following treatment. Treatment itself entailed naming by the patient, modelling of failed attempts by the therapist, and repetition by the patient. Repetition was a central feature of the approach and meticulous mapping of 'recovery curves' for each treated item.

Summary

Criticisms of the classic Wernicke–Lichtheim model continued until the Second World War, even though some sporadic but influential and vocal proponents of localisation continued to propose, in some cases, extreme scenarios of the localisation of language in the brain with contentious polarisations taking place in France and Germany. At the same time, workers became more aware that aphasia was too complex for the medical model to cope with alone and that psychological and linguistic models were clearly necessary to help explain aphasic phenomena and significant progress was made in describing impairments of grammar. The rise of Fascism in Germany caused a major exodus of aphasiologists, many of whom where Jewish, mainly to the English-speaking world, particularly North America. Scientific aphasiology began to develop in North America, new research methods were introduced and the controlled group study became established as the preferred approach to research. A more systematic approach to rehabilitation emerged from voice and elocution teachers in Europe and approaches to therapy spread to North America.

6 From the Second World War to Geschwind: neoclassicism and the return to localisation

After the Second World War, the centre of gravity had moved from Europe to North America in aphasiology, as in other areas of human endeavour. The influence of approaches established pre-Second World War was passed down and issues of localisation generally gave way to holism. A significant impact on development came from increased efforts in aphasia rehabilitation in those states engaged in the war, as Goodglass (1993: 26) stated: 'The Second World War produced several converging influences that accelerated both basic research on aphasia and the efforts to rehabilitate aphasic patients.' Similarly, Wepman (1951: 3–4) observed: 'The developing attention was greatly stimulated by the large numbers of brain-injured patients who needed therapy as an aftermath of the second war.' This is exemplified by the work of Luria in the Soviet Union, Wepman and Schuell in the USA and Zangwill, Butfield, Newcombe and Russell in Britain. Developments in aphasia and aphasia therapy were limited under fascism in Germany, but there were isolated attempts to continue with a traditional approach (for example by Bay and Leischner). In the 1960s there was a return to classical Lichtheim–Wernicke aphasiology triggered by the neoclassicism of Boston neurologist Norman Geschwind in the USA.

A. R. Luria and Soviet aphasiology: an attempt at synthesis

Russian psychologist and physician **Alexander Romanovich Luria** (1902–1977; see Figure 6.1) is considered one of the founders of modern neuropsychology (Métraux, 1994; Pickenhain, 1994; Vocate, 1987). Aphasia had an important place within his many publications (bibliography in Braemer & Jantzen, 1994). Hatfield (1981: 338) described his position in the following way: 'Luria is not only the pre-eminent figure in Soviet Russian aphasiology but he stands out monumentally on the international forum.' He was influenced by the pioneering work of fellow Russian psychologists Pavlov and Vygotsky and has been called the 'last giant' in the history of aphasia.

Luria's contribution to the general understanding of brain function, higher cognitive functions and their disruption, constitute the basis for his

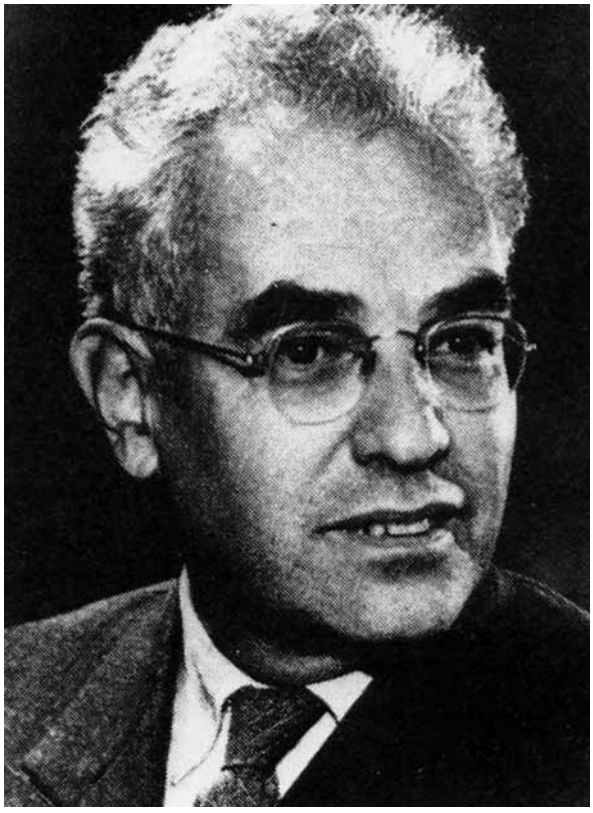


Figure 6.1 Alexander Romanovich Luria (1902–1977), the 'last giant' of aphasiology.

work on aphasia. The essential foundations are currently easily accessible in Luria (1973).

An important early work is Luria's *Traumatic Aphasia*, where insights essentially rest on data acquired from injuries from the Second World War. The book appeared originally in Russian in 1947, with a revision in 1959 (according to Luria, 1970: 5) and the English translation appeared in 1970. Of all the work on aphasia that has come from studying the victims of war, this book perhaps deserves to be recognised as the most inspired and influential. Extensions to his views on aphasia can be found in Luria (1976) and extensively retraced in *Higher Cortical Functions in Man* (1980) and *The Working Brain* (1973). A summary of Luria's aphasiology can be found in Kagan and Saling (1992) and in German in Sokolovsky (1997). Special issues devoted to Luria appeared in the *Journal of Neurolinguistics* (1989, vol. 4, no. 1) and in *Aphasiology* (1995, vol. 9, no. 2), the latter devoted to aphasia. Luria also published two little books about two unique individuals that became very popular. *The Mind of a Mnemonist* (1987) and a personal account, with

help from Luria, of the brain-damaged survivor of war, Lieutenant Zasetsky, *The Man with a Shattered World* (1972).

Luria attempted to create a **synthesis** of the localisationist approach, as it was represented by Wernicke or Kleist, with the holistic approach of a Flourens or a von Monakow. To Luria, neither approach seemed to be altogether appropriate to understand the functioning of the human brain. He states:

> While, on the one hand, the mechanistic view of strict localisation always leads the analysis of the cerebral basis of mental activity into an impasse, the holistic [. . .] opinions of mental processes were unable, on the other hand, to create the necessary preconditions for scientific progress.
>
> (Luria, 1992: 21)

The central terminology for this synthesis is **mental function** and the notion of a **functional system**. Every single mental function (like thinking, writing, arithmetic) should be understood not as a single, simple function, 'but as a complete functional system, embodying many components belonging to different levels of [. . .] motor and nervous apparatus' (Luria, 1973: 27). Therefore, 'there can of course be no question of the localisation of complex functional systems in limited areas of the brain or of its cortex' (Luria, 1973: 30).

Mental activity is a complex functional system 'involving the participation of a group of concertedly working areas of the cortex' (Luria, 1973: 35). In addition, functional systems are characterised by the variability and mobility of the participating mechanisms. If we consider writing, for instance, then this can also be achieved using the feet or the mouth if circumstances require. For this reason too, rigid allocation of functions to specific brain areas cannot be assumed.

Luria (1973: 43ff.) distinguished between 'three principal functional units of the brain'. The first unit regulates 'tone or waking', the second unit is concerned with 'obtaining, processing and storing information arriving from the outside world', and the third unit is for 'programming, regulating and verifying mental activity'. Seen anatomically, the first unit lies within the deeper layers of the subcortex and the brain stem. The second unit is composed of the parietal (for somatosensory), occipital (for visual), and temporal (for auditory) lobes, whereas the third unit is represented in the frontal lobe. Additionally, these basic units are hierarchically constructed and comprise three mutually overlapping cortical zones:

> the *primary* (projection) area which receives impulses from or sends impulses to the periphery, the *secondary* (projection-association), where incoming information is processed or programmes are prepared, and finally, the *tertiary* (zones of overlapping), the latest systems of the cerebral hemispheres to develop.
>
> (Luria, 1973: 43)

In other words, in the posterior sensory unit, there is an elaboration from sensation to symbolic process through the primary sensory zones, through the secondary to the tertiary zones. Only in the overlapping tertiary zones can information from separate modalities be integrated. While primary zones are directly linked to specific body parts, this is not the case for the tertiary zones, as this functions supra-modally.

For Luria language was also a functional system, which primarily used the second and third unit, and he attempted to decompose the individual language actions into their components and to attribute them to the corresponding fields or zones of the units.

For Luria the classification of aphasia resulted from localised injuries and their relationship to the respective components of language processing, as illustrated in Figure 6.2 (Luria, 1964, 1973: 303–322, 1980: 486ff., 506ff.). The primary, secondary, and tertiary fields are not illustrated graphically and mentioned only in the text. For details, consultation of the original texts is advised.

On Luria's model **dynamic aphasia** (**1**) (also called 'frontal' aphasia) is caused by a lesion of the left prefrontal lobe anterior to the premotor areas (tertiary zone of the frontal cortex, third unit). The main features are an apparent lack of a *will* to speak and a disturbance of inner speech, which is

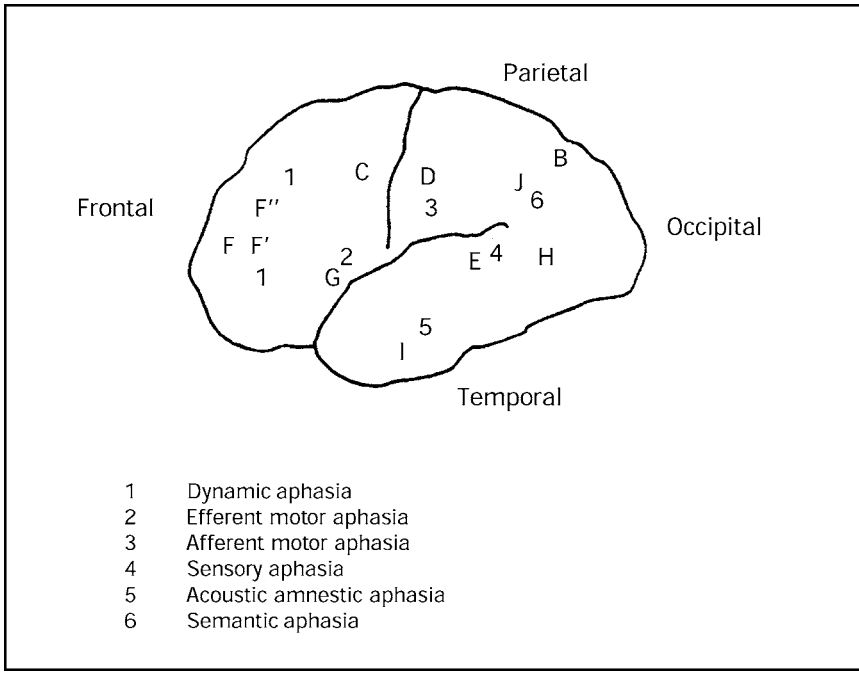


Figure 6.2 Localisation of the components of language processing and location of lesions causing aphasia following Luria (based on Caplan, 1987: 131; Kagan & Saling, 1992: 31).

responsible for the production of a verbalisable linear sentence scheme from a general plan. The aphasic person can no longer make predicative statements or propositions, and production is limited to empty phrases. The patient understands quite well and they can also name and repeat, although they initiate little speech without external stimulation.

A lesion of the inferior frontal areas of the left premotor zone (secondary zone of the frontal cortex, third unit, which corresponds to Broca's area, leads to **efferent motor aphasia (2)** (Luria, 1973: 183ff.). Individual sounds are not problematic, but the 'disturbance becomes apparent when such patients have to switch from one articulation to another' (Luria, 1973: 185). The individual has a problem with the production of linear schemes (G), which also has effects in other domains, so writing is also impaired in a similar fashion. In later stages of the condition, agrammatism emerges as a symptom: 'In later stages of recovery, the well known clinical syndromes of "telegram style" may appear' (Luria, 1964: 155). The reasons for that are not entirely clear and Kagan and Saling (1992: 32) suppose the following: 'The grammatical difficulty might be viewed as a problem with the connection of grammatical units in the same way that the articulatory problem relates to the connection of articulatory postures into a kinetic melody.' Luria also called this form of aphasia 'kinetic' motor aphasia Luria (1964: 153–155).

Afferent motor aphasia (3) is characterised by the 'inability to determine immediately the positions of the lips and tongue necessary to articulate the required sound of speech' (Luria, 1973: 174), and in milder forms there is confusion of similar phonemes. Phonemic confusions also occur in reading and writing. The lesion is in the inferior region of the left post-central parietal cortex (secondary zone, second unit), which, among other things, leads to an impaired interpretation of kinaesthetic feedback. For this reason Luria called this form of motor aphasia 'kinaesthetic' motor aphasia (Luria, 1964: 152–153). Thus, unlike most other theorists, Luria described two separate forms of 'motor' aphasia.

Sensory aphasia (4) is caused by a lesion of the superior and posterior regions of the temporal lobe (secondary zone of the temporal cortex, second unit), which approximately corresponds to Wernicke's area. In the secondary auditory cortex, Luria localised phonemic analysis (E). The individual has intact hearing, but they cannot discriminate between, analyse, or synthesise similar phonemes, which leads to comprehension difficulties at the lexical level. Luria attempted to derive paraphasias and the problems of written language from impaired 'phonemic hearing'.

An injury to the middle gyrus of the temporal lobe is the underlying cause (secondary zone of the temporal cortex, second unit) in **acoustic amnestic aphasia (5)**, which causes an impairment of verbal memory (I) affecting word repetition.

Finally, in **semantic aphasia (6)**, patients 'have a good understanding of the meaning of individual words, they cannot grasp the meaning of the

construction as a whole' (Luria, 1973: 153), and there is an impairment of 'logico-grammatical operations'. Not only the linguistic, but also the symbolical level is affected, so that additionally spatial deficits, apraxia, acalculia and other problems can co-occur. The common problem is 'a disturbance of simultaneous (and spatial) synthesis' (Luria, 1964: 157). The lesion is in the parieto-temporo-occipital region of the left hemisphere (tertiary zone, second unit).

On the face of it Luria's model seems similar to the Lichtheim–Wernicke model, but Luria has clearly different views on crucial points. First, he emphasises the individual processes (analysis, synthesis, integration) engaged in language; his is a process model. Second, the possibility of aphasic symptoms being connected at different linguistic levels on the basis of abstract principles is implied in Luria's work. The disturbance of the linear scheme, which shows itself in sound production, sentence production and in writing, is an example. Third, Luria's process model provides routes for the formulation of strategies for rehabilitation, because the model is flexible and dynamic in contrast to the static classical model and because the brain is conceptualised overall as an interactive system.

Luria's work did not become widely known in the West until it was published in English translations in the early 1970s, but in subsequent years interest increased (e.g. Kaczmarek, 1995). However, Luria had a major impact in the Soviet world. This is illustrated by the observation that while he had little influence in West Germany he was influential in East Germany.

Luria had his main impact in clinical rather than theoretical neuropsychology and aphasiology in Soviet countries and in Eastern Europe, the UK, Germany and Australia in particular. Luria's approach and materials for the assessment of brain-damaged individuals were translated into English by Anne-Lise Christensen in the 1970s, *Luria's Neuropsychological Investigation* (Christensen, 1974, 1975) and these became the basis for the development in the USA of a psychometric battery (Golden, Hammeke, & Purisch, 1980: *The Luria-Nebraska Neuropsychological Battery*) that met with some criticism, not least from Christensen (2002) herself, who doubted the wisdom of the attempt to impose psychometric criteria on Luria's clinical investigations. The tests were also translated into German as the *Tübinger-Luria-Christensen Neuropsychological Test Series* (**TÜLUC**) (Hamster, Langner, & Mayer, 1980). Luria's approach has made a significant contribution to rehabilitation (Hatfield, 1981; Sokolovsky, 1997: 41f.). While Luria claimed not to be a localisationist, but emphasised localisable 'functional systems', his model helped to reintroduce localisation and provide it with a more dynamic and multidimensional perspective, rather than the two-dimensional connectionist view of the neoclassical model (e.g. Goodglass, 1993: 31).

Also working within the Soviet school of aphasiology was **Egon Weigl** (1901–1979), who was born in Hamburg and following study at Munich and Berlin went to Frankfurt where he studied under Goldstein (Weigl, 1968). Like his teacher, Weigl was forced to flee Germany during the Second World

War, but unlike Goldstein and others, he headed East to Romania, where he spent 30 years and developed most of his important work before returning to East Berlin in 1961, when he was 60. Weigl's pioneering work on **inner speech** and **deblocking** (with Fradis and Kreindler) was completed within the framework of Soviet aphasiology (De Bleser & Marshall, 2005). His writings appeared in Russian, Romanian and German. Weigl developed deblocking (Weigl, 1961, 1968) as a method for investigation of dissociations in aphasic impairments and as a therapeutic method. It 'makes use of the fact that the capacity for decoding, coding, and recoding verbal information in not completely obliterated in most aphasic patients' (Weigl, 1968: 144). Among the many examples he gives, a speaker with an object naming impairment may be able to read aloud the object name. So while certain modalities of expressive or receptive language may be impaired, deblocking utilises unimpaired channels. The deblocking effect (D-effect) works by removing for a period a total or partial disturbance of language function and linking two semantically related examples of a preserved (e.g., reading the word aloud) and the blocked (e.g., confrontation naming of the object) function, where the preserved function is evoked before the impaired – blocked – function. Various forms of cuing used in therapy, and developed spontaneously by aphasic people, use variants of deblocking, like the individual who self-cues an elusive word by writing it down on paper, or sometimes in the air, before they can access it.

Weigl's other main contribution was on the concept of **inner speech**, a topic of significant interest in Soviet aphasiology and psychology (see De Bleser & Marshall's, 2005 translation of Weigl's 1964a paper with commentary). The irony of this fact does not escape De Bleser and Marshall (2005: 251) who comment that 'a passion for the study of inner speech was most highly developed in countries where the dangers of unguarded outer speech were most extreme'. The concept of inner speech has significance for the relationship between thought and language and has a long history in psychology and aphasiology: the question of whether though utilises language or proceeds independently of language, and whether aphasia impairs thought. In aphasiology, Goldstein (1948) suggested that inner speech accounts for all processing before the overt motor act and in his 'central' aphasia, inner speech is obliterated, but he did not believe that inner speech was equivalent to thinking (De Bleser & Marshall, 2005). A range of studies from Soviet workers employed physiological methods such as electromyography (EMG) to detect muscular and laryngeal activity during various covert, silent, speech tasks, including methods that prevented speakers using covert speech. Luria and Tsvetkova's (1970) investigations concluded that Luria's dynamic aphasia and efferent motor aphasia are directly caused by disturbances of inner speech. Weigl developed methods that used blowing down a straw into a glass of water to induce a suppression of subvocal speech activity, but his conclusion in his 1964a paper is that subvocal speech plays a minor role in normal or impaired language processing. In the latter part of the twentieth century

inner speech was to become a central component of leading models of verbal **working memory** (Baddeley, 1986); this development was independent of the earlier work in Soviet psychology and aphasiology (De Bleser & Marshall, 2005). Baddeley's model includes a central executive and the slave systems of the (verbal) phonological loop and the visuospatial scratchpad. In the verbal component, covert articulatory rehearsal forms the basis for retention of small units of information in working memory, that can be disturbed under articulatory suppression causing errors in working memory.

Aphasia therapy following the Second World War in Britain and North America

An historically significant study of the effectiveness of aphasia therapy was conducted in Edinburgh by psychologist **Oliver Zangwill** and speech therapist **Edna Butfield** and published in 1946 (Butfield & Zangwill, 1946). Howard and Hatfield (1987: 51) suggest that the paper 'was the first published attempt to evaluate the efficacy of therapy properly, and to assess also the significance of specific factors, such as the form of aphasia and its aetiology'. The short paper describes therapy for 66 cases of aphasia between the ages of 20 and 40 years. They were divided into two groups: in Group 1 treatment began within 6 months of the onset of aphasia, and in the other, Group 2, treatment began after 6 months. This was done in an attempt to examine the effects of spontaneous recovery in the second group. Patients were assessed in speech, reading, writing, and calculation and classified in terms of Weisenberg and McBride's expressive-receptive nomenclature and treatment was based mainly on Goldstein's methods. The amount of treatment varied between as little as 5 sessions to 290 per individual. Progress was measured fairly grossly by present-day standards in terms of much improved, improved or unchanged. 'Speech' was judged to be *much improved* in half of Group 1 and one-third of Group 2, but improvement in the other modalities was less satisfactory. Improvement was seen to be highest in traumatic cases, and the inclusion of Group 2 suggested that improvement was unrelated to spontaneous recovery.

In the USA, educational psychologist **L. Granich** (1947) developed therapy for 300 war veterans in Atlantic City Hospital, New Jersey, including 100 with aphasia and related disorders. Granich's therapy was also much influenced by Goldstein's work and he was not concerned with standardised testing or aphasic syndromes. He used drilling and not only believed in the beneficial effects of hard work by his patients, but also believed in the value of the strategies that patients produced themselves, although his approach was mostly uneven and patchy (Howard & Hatfield, 1987).

Joseph Wepman and Hildred Schuell: pioneers of therapy

Rehabilitation in the USA benefited from the creation of a system of military and veteran hospitals, the Veterans Administration hospital system

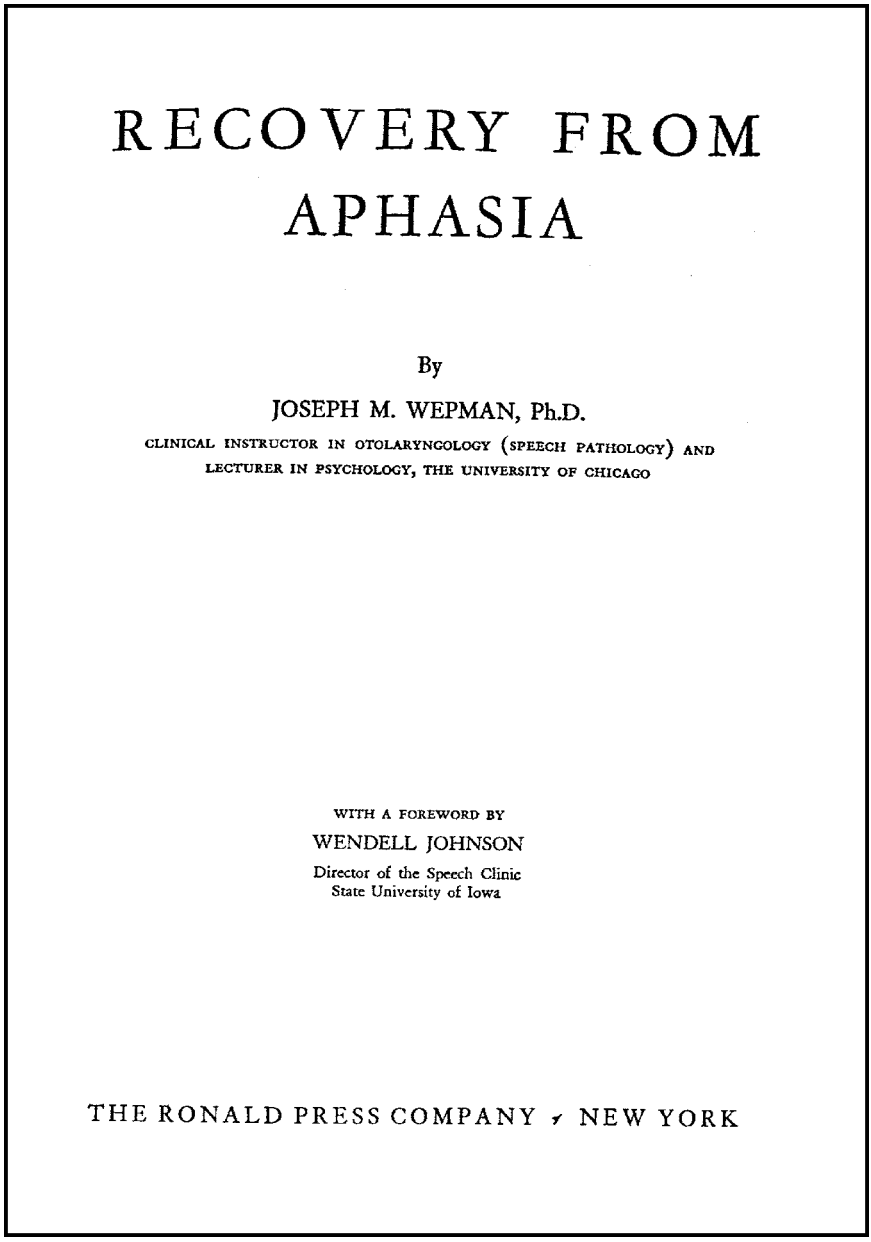
(Goodglass, 1993: 26). Two outstanding personalities who dedicated themselves to aphasia therapy in the post-war period in the USA are **Joseph Wepman** (born 1907) and **Hildred Schuell** (1907–1970). Both were successors to Weisenburg and McBride, who emphasised the importance of a valid and reliable assessment of aphasia and the irrelevance from the perspective of rehabilitation of localisation issues.

Although Wepman and Schuell are discussed together here, and although both are founders of the **stimulation approach** to aphasia therapy, they had very different views (Tikofsky, 1984: 19ff.).

Psychologist Joseph Wepman worked in the holistic tradition of Jackson and especially Goldstein; his 1951 book *Recovery from Aphasia* made a significant contribution to rehabilitation (see Figure 6.3). Wepman attached great value to the 'whole person' in his approach to aphasia therapy and defined aphasia and his therapeutic approach as follows:

> By definition, aphasia is any language problem resulting from organic disturbance of cortical tissue in which the defect is not due to faulty innervation of the musculature of speech, dysfunction of the peripheral sense organs, or general mental deficiency. The language problem manifests itself in the areas of symbolization, comprehension, and reproduction of concepts while the individual is using or attempting to use conventional spoken or written symbols. The brain defect which produces the aphasia is seen to produce also many other far-reaching symptoms beyond the realm of language. Many personality aberrations, many atypical modes of behavior that are not readily acceptable in our society, and various other symptoms of a functional nature are seen to be the direct or indirect result of the cerebral impairment. Recovery from the disorder of language, it is believed, is based upon over-all recovery of the individual with a new ability to function in society as a contributing part of that society. This, as is evident, is far beyond the concept of speech recovery as traditionally understood; it involves a total personality readjustment, a new-found stability as a person. It is a concept basic to the present book that recovery of the ability to speak, to read, or to write while the patient is still unable to adjust to society is a futile and useless goal. Language skill in a person unable to resume a normal or satisfying place in society is a wasted resource. A resumption of social intercourse in a manner mutually acceptable to the patient and to society, a controlled reduction of the effects of the personality aberrations which follow brain injury, stability of psyche, and insight into the physical limitations imposed by the brain insult seem to be the important goals. Language should be considered as the means of interpersonal relations, not the end of recovery.
>
> (Wepman, 1951: 4)

With this understanding of aphasia and aphasia therapy, Wepman was



RECOVERY FROM APHASIA

By

JOSEPH M. WEPMAN, Ph.D.

CLINICAL INSTRUCTOR IN OTOLARYNGOLOGY (SPEECH PATHOLOGY) AND LECTURER IN PSYCHOLOGY, THE UNIVERSITY OF CHICAGO

WITH A FOREWORD BY

WENDELL JOHNSON

Director of the Speech Clinic
State University of Iowa

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Figure 6.3 Joseph Wepman's (1951) book *Recovery from Aphasia* was an important inspiration for developing aphasia therapy.

clearly ahead of his time and his pointed demands for a comprehensive rehabilitation are resonant in the major themes of present-day approaches (like the WHO's International Classification of Functioning, Disability and Health and the 'social' approach). With a psychological approach as broad as Wepman's and comprising as many aspects, it is clear that localisation can play no role. Goodglass (1993: 30) wrote: 'Nowhere in Wepman's work is there any concern with the neuroanatomical correlates of aphasia.' Wepman (1951: 8–18) discussed the question of localisation in detail and feels that it is of little import for therapy:

> While not overlooking the contribution of the localisationists, the writer believes that a more hopeful prognosis can be made for aphasic adults with the acceptance of a nonlocalisationist viewpoint in accordance with which recovery follows reintegration of the remaining cortical tissue into a functioning whole.
>
> (Wepman, 1951: 8–18)

Wepman also developed a test of his own, the **Language Modalities Test for Aphasia (LMTA)** (Tikofsky, 1984: 15–18).

Speech pathologist Hildred Schuell, like Marie, saw aphasia as a single unitary condition, that could however occur with additional complications and symptoms (Schuell, 1974; Schuell, Jenkins, & Jiménez-Pabón, 1964; see also Duffy, 1994; Howard & Hatfield, 1987: 71ff.). She too attached great value to a detailed assessment in all modalities reflected in the **Minnesota Test for Differential Diagnosis of Aphasia (MTDDA)** (Schuell, 1955) developed with detailed psychometric evaluation by her for the examination of verbal and non-verbal performance in all modalities. This battery dominated the assessment for rehabilitation in the English-speaking world for many years. Schuell (1966: 138) deliberately recommended a long test so that nothing is overlooked: 'an adequate diagnostic test must sample relevant kinds of behavior in all language modalities over the entire range of aphasic deficit'.

The inclusion of non-verbal tests results from Schuell's definition of aphasia. For Schuell et al. (1964), aphasia is a general language impairment affecting all modalities that may be complicated by other effects of brain damage. Its main features are a reduction in vocabulary, impaired auditory-verbal retention span, and impaired language perception and production. It may also be complicated by impaired auditory, visual, and sensorimotor processes. Schuell emphasised that *all* aphasic persons have an auditory comprehension deficit as a common basic disorder and a reduced vocabulary, and that these are the core features of aphasia. She also emphasised, like Freud, that there was continuity between aphasic and non-aphasic errors in the use of language. Aphasic impairments were seen therefore as more serious and severe forms of normal language errors.

Although Schuell started with the notion, originating with Marie, that there is only one aphasia, she nonetheless divides aphasias into syndromes,

which result from the profile of performance on the five areas tested by the MTDDA: auditory functions, visual and reading functions, speaking and language, visuo-motor and graphical performances, numeric and arithmetic functions. Testing produces a profile that permits classification.

Schuell distinguished five basic aphasia syndromes determined on the basis of psychometric studies of performance of large groups on the MTDDA. **Simple aphasia** (aphasia without complications) is defined as reduction of available language in all modalities, is a relatively mild multi-modal language disorder without particular perceptual, sensorimotor or dysarthric components, and it has a very good prognosis. **Aphasia with visual involvement** is aphasia complicated by central involvement of visual processes, that is, visual discrimination, recognition, and reproduction. **Aphasia with sensorimotor impairment** is characterised by severe reduction in all modalities complicated by sensorimotor involvement. **Aphasia with scattered findings** has some residual language preserved, and it is marked by disturbances in reading and writing (caused by a disturbance of visual processes), and furthermore by disturbances in speech (dysarthria) and comprehension. Finally, **irreversible aphasia syndrome** is a global disorder that affects all areas and comprises an almost total loss of language skills. In her later work, Schuell sometimes added two further syndromes, **aphasia with persisting dysfluency** and **aphasia with intermittent auditory imperception**.

The syndromes (i.e., the performance profiles and the degree of severity) are essential for Schuell in order to plan targeted intervention and the therapeutic approach that Schuell (and Wepman) developed is called the **stimulation approach** (Duffy, 1994). Although this will not be presented in detail here, an important principle was that auditory stimulation is the bedrock of therapy. For Schuell aphasic people do not re-learn (as in a didactic approach), because nothing is 'lost' in the first place, but simply unavailable because of the brain damage. Thus, a reactivation of impaired functions underlies recovery.

The value of Schuell's classification was questioned by Goodglass (1993):

> Schuell did offer a typology of aphasia with five subcategories, but these were neither psycholinguistically nor anatomically based. Rather, Schuell's typology was based in part on severity and in part on how the language problem interacted with sensorimotor impairments. This typology had limited acceptance and did not survive.
>
> (Goodglass, 1993: 28)

As a representative of localisationist neoclassic doctrine, Goodglass may see it that way, inevitably, but Schuell's achievements seen from the perspective of those concerned with rehabilitation, rather than localisation, are different. For example, Duffy (1994: 146) stated that 'Schuell's work in aphasiology spanned two decades and included significant contributions in the areas of diagnostic testing, classification of aphasic patients, and theory

development regarding the underlying nature of aphasia.' Tikofsky (1984: 22) goes even further in his judgement of the MTDDA when he states: 'It is an instrument which has won relatively wide acceptance by many practicing speech-language pathologists, even those who do not accept Schuell's basic assumptions concerning the nature of aphasia or who are unaware of them.'

Whatever the theoretical or professional perspective, with the MTDDA a new era began (Tikofsky, 1984: 22). With the breadth of testing, the assessment of severity, the prediction of recovery and the valid and reliable evaluation of the success of treatment, the benchmark for the development of future assessment batteries of aphasia was defined. The therapeutic insights of Schuell and also Wepman are valuable even to this day and contributed to a new understanding of aphasia and its treatment. While there had been earlier approaches to treatment, as we have seen, with Wepman and Schuell the treatment of aphasia developed a more systematic and scientific basis. The growth of a purely therapeutic branch of clinical aphasiology which was independent of neurology began in North America,. It originated with Weisenburg and McBride and was consolidated with Wepman and Schuell. Parallel but relatively unconnected national associations developed later in the USA, including the Academy of Aphasia founded in 1960, and concerned mainly with neurological, linguistic and cognitive issues, and the annual Clinical Aphasiology Conferences, founded in 1969, concerned mostly with clinical issues.

Neoclassicism: the return of anatomy to aphasiology

Norman Geschwind was famously responsible for the reintroduction of the classical Wernicke–Lichtheim model to the international scientific community. Ironically, Geschwind's resurrection of the model contributed to a reintroduction of it to the German aphasiological world. Like the Wernicke–Lichtheim model, Geschwind's **neoclassicism** emphasised language centres and the connections (**connectionism**) and disconnections between them, and is sometimes referred to as neo-connectionism.

Norman Geschwind: the disconnection syndrome

Boston neurologist **Norman Geschwind** (1926–1984) (Geschwind, 1974; Kean, 1994) is mainly responsible for the return of language localisation as the fundamental assumption underlying aphasia theory, as **neo-connectionism** or neoclassicism. Benson and Ardila (1996: 21) call the third epoch of their history of aphasia 'Geschwind and the return of localisation'.

Geschwind resurrected the assumption that certain areas of the left hemisphere have a special function in language processing, among them especially Broca's area and Wernicke's area: 'Several [. . .] regions are more narrowly specialized. Broca's area and Wernicke's area are involved in the production and comprehension of language' (Geschwind, 1979: 113). The connection between Broca's area and Wernicke's area is via the **arcuate**

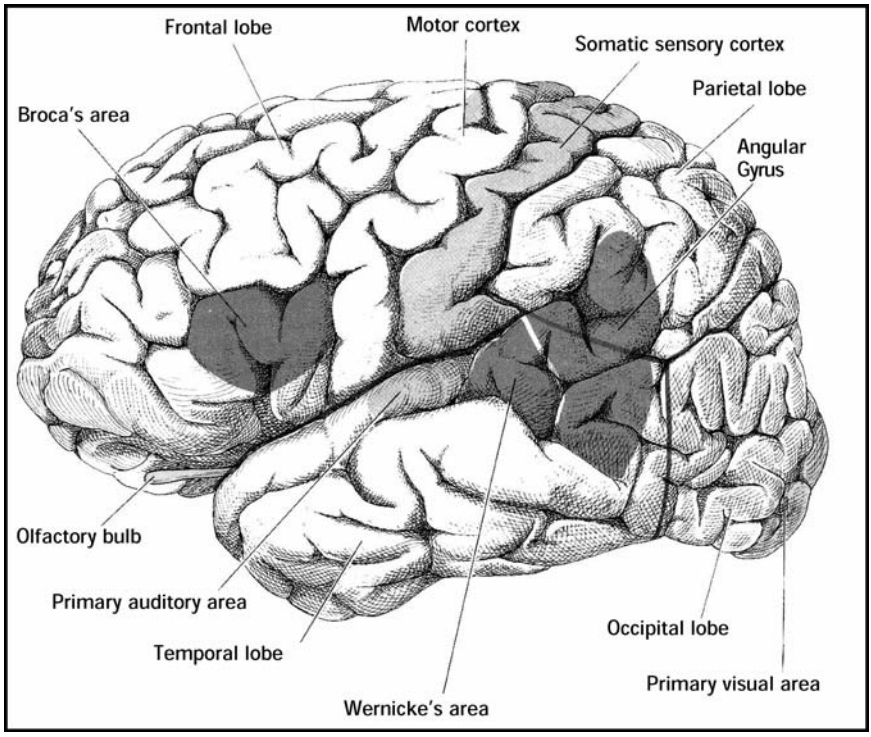

Figure 6.4 The human cortex with its functional specialisations according to Geschwind (1979: 113). From 'Language in the human brain', by D. Howard. Chapter 9 in *Cognitive Neuroscience*, edited by M. D. Rugg. Hove, UK: Psychology Press, 1997. Reprinted with permission.

fasciculus, and the **angular gyrus** mediates between visual and auditory information, which is important for written speech and for naming.

Geschwind (1979: 115) described language processing as a form of information processing with the participation of different brain areas. Visual information proceeds to the angular gyrus via the primary visual cortex, in which the visual form 'is associated with the corresponding auditory pattern', and when the word is required for speech, a representation is passed on to Broca's area via the arcuate fasciculus, 'where the word calls up a detailed articulation programme', which is implemented by the motor cortex. It is ironic that Geschwind, of all people, should reintroduce language localisationism, because he claimed to have been 'a member of the philosophically sophisticated Jackson-Goldstein-Head group' (Geschwind, 1964: 215).

Geschwind's change in theoretical outlook appears to have come about through the influence of his senior colleague **Fred Quadfasel**, who was Goldstein's former collaborator and Bonhoeffer's former assistant in Germany before he moved to Boston. Quadfasel possessed a good collection of essays

and books from the rich history of German aphasia research, to which Geschwind had access.

In a legendary essay on the **disconnection syndrome**, Geschwind (1965) developed his model based on the classical connectionists. This neoclassical aphasiology would ultimately become the dominating paradigm until contemporary time. 'Few works in the history of neurology match the intellectual robustness and scope of the "Disconnexion syndromes" or have had so profound an impact' (Kean, 1994: 356).

A case of **tactile aphasia** will be described to illustrate the concept of disconnection. Tactile aphasia is caused by a disconnection of the two hemispheres. Geschwind's patient had a 'disturbance characterized by an inability to name objects tactilely with preservation of the ability to name on the basis of visual or auditory stimulation and in the presence of intact spontaneous speech' (Geschwind, 1965, in Kean, 1994: 380). The lesion was in the **corpus callosum**, which connects the two hemispheres, so tactile information from the right hemisphere (via the left hand) could no longer reach the speech area in the left. The fact that the patient could recognise objects palpated in the hands is shown by the fact that he was always able to select the object from among several objects that he was unable to name. Geschwind referred to the fact that Liepmann had already discussed similar cases.

Important for Geschwind's position are two cases described by Dejerine, in which Dejerine argued 'that pure word blindness without agraphia was caused by a disconnection of the intact right visual cortex from the left angular gyrus in the patient, in whom the left visual cortex had been destroyed' (Geschwind, 1965, in Kean, 1994: 371). Disconnection syndromes can occur within, as well as between, hemispheres. For Geschwind (1962, 1965), cortico-cortical connections between cortical areas are important for language processing.

In their article 'Isolation of the speech area', Geschwind, Quadfasel, and Segarra (1968) argued that, because of the nature of brain physiology, lesions are possible that leave the speech areas intact but cut them off from the remaining brain areas and their functions. They describe the case of a young woman who has no language comprehension whatsoever and produced no spontaneous speech at all. However, she could produce **echolalia** and complete song lyrics, also songs or advertising slogans that she had learnt since her brain damage. The autopsy revealed that she had a massive lesion that left the auditory system, Broca's area and Wernicke's area and their connecting peri-Sylvian fibres intact, which then continued to execute their functions in isolation. Similarly, conduction aphasia is also understood as a disconnection syndrome.

The **fluent/nonfluent** distinction is associated with Geschwind and colleagues (Benson, 1967; Howes, 1964; Howes and Geschwind, 1964), although the idea originated with Wernicke (1874). Fluency divides the connected speech of aphasic speakers into two types: those with anterior damage with a

nonfluent aphasia, and those with posterior damage with fluent aphasia. The fluency dimension is discussed in more detail in Chapter 9.

With Geschwind's work at the Boston Veterans Administration Hospital, localisationist aphasiology made ground again and became the determining paradigm: 'The neurologists trained at the Boston VA Aphasia Research Center provided a rich and continuing influence on the understanding of aphasia' (Benson & Ardila, 1996: 22). However, psychological and linguistic approaches did not remain without impact in the new 'anatomical associationism of the waning 20th century either' (Goodglass, 1993: 31). As Benson and Ardilla (1996: 23) stated: 'During the 1960s and 1970s, in addition to the training program in aphasia, the Aphasia Research Center included an active psychology/psycholinguistics research unit under the direction of Harold Goodglass and Edith Kaplan.'

The Boston classification

Beginning with Geschwind's neoclassical approach in the 1960s, Wernicke's classification, which was now repackaged as the Boston classification, became internationally known. Beside considerable research activity in Boston, the influence of the Boston School was bolstered by a test battery that **Harold Goodglass (1920–2002)** and Edith Kaplan developed for the examination of aphasia: the **Boston Diagnostic Aphasia Examination (BDAE)** (Goodglass & Kaplan, 1972). This was to become probably the most popular and widely used aphasia battery ever produced, and still appears to dominate clinical assessment in English-speaking countries (Katz et al., 2000). Goodglass was Director of the Boston Aphasia Research Center from 1969 to 1996, renamed the Harold Goodglass Aphasia Center in his honour after his death. He established the American Psychological Association's Division of Clinical Neuropsychology and served as its first president (1979–1980).

With this battery different tasks assess language in all modalities. Simple questions (e.g., 'How are you today?'), open questions (e.g., 'Why are you actually at this hospital?') and the Cookie Theft picture description serve as the basis of assessment of connected spoken language. For comprehension the understanding of words, requests, and complex utterances are tested. Oral and written naming, reading aloud, repetition, reading comprehension (word, sentence, paragraph), writing to dictation, prosody and automatic speech are assessed. A range of functional profiles emerge from testing that allow the classification of an individual's aphasia into a neoclassical type. When the BDAE was developed, brain imaging was in its infancy, and it was assumed that certain aphasic symptoms indicated the location of certain lesions and a major aim of the battery was therefore to localise structural lesions from impaired functions.

The syndromes or aphasia types resulting from testing with the BDAE will be briefly described. The reader will note the close parallels between these types and those of the classic model. **Broca's aphasia**

> is the [. . .] aphasia depending on a lesion involving the third frontal convolution of the left hemisphere. Its essential characteristics are awkward articulation, restricted vocabulary, restriction of grammar to the simplest, most over learned forms, and relative preservation of auditory comprehension. Written language follows the pattern of speech in that writing is usually at least as severely impaired as speech, while reading is only mildly affected.
>
> (Goodglass & Kaplan, 1972: 54–55)

People with Broca's aphasia 'usually [have] lost the ability to recall syntactic patterns', so that 'syntax remains primitive' (Goodglass & Kaplan, 1972: 55).

Wernicke's aphasia 'usually depends on a lesion in the posterior portion of the first temporal gyrus of the left hemisphere. The critical features of this syndrome are impaired auditory comprehension and fluently articulated, but paraphasic speech' (Goodglass & Kaplan, 1972: 59). Syntax is often characterised as paragrammatic: 'Though the grammar of these patients is often incorrect, there is usually free use of complex verb tenses, embedded subordinate clauses, and other departures from simple declarative word order' (Goodglass & Kaplan, 1972: 59). Speech is fluent with not only normal melody of speech, but also literal (phonemic) and verbal (semantic) paraphasias that are also reflected in reading and writing. As theoretical background, Goodglass and Kaplan (1972: 59) emphasise 'that Wernicke's area is the crossroad for all meaningful associations to sound pattern and for performances (such as reading and writing) which have been learned in conjunction with the auditory component of words'. As a consequence, a lesion of Wernicke's area can 'reduce performances which depend on past and current auditory experience' (Goodglass & Kaplan, 1972: 60), but other functions, like syntax for instance, are left relatively intact. Paraphasias and a lack of error awareness likewise are consequences of impaired auditory control.

Anomia, or **anomic aphasia**, has as a 'major feature [. . .] the prominence of word-finding difficulty in the context of fluent, grammatically well-formed speech' (Goodglass & Kaplan, 1972: 61) and circumlocutions (periphrases) are produced quite often. Language comprehension is well preserved. Although the lesion is often in the angular gyrus on the model, anomia 'is the least reliably localisable of the aphasic syndromes' (Goodglass & Kaplan, 1972: 64).

Conduction aphasia 'is the name applied to the syndrome in which repetition is disproportionately severely impaired in relation to the level of fluency in spontaneous speech and to the near normal level of auditory comprehension' (Goodglass & Kaplan, 1972: 68). In accordance with Geschwind, the lesion is localised in the arcuate fasciculus, the fibre tract that connects Broca's and Wernicke's areas.

Transcortical sensory aphasia, a posterior aphasia, corresponds to the syndrome of 'isolation of the speech area' following Geschwind and appears as a

severe Wernicke's aphasia with a surprisingly well-preserved ability to repeat. The idea is that Wernicke's area fulfils its task of transferring the information to Broca's area via the arcuate fasciculus but 'the isolation of this portion of the speech system prevents any interaction between the knowledge, intention and perceptions of the rest of the brain and those of the isolated speech mechanism' (Goodglass & Kaplan, 1972: 72).

In **transcortical motor aphasia**, an anterior aphasia, 'repetition is particularly intact in a setting of otherwise limited speech. This syndrome is marked by an absence of spontaneous speech, with some recovery of the ability to make brief replies to questions and fairly good confrontation naming ability' (Goodglass & Kaplan, 1972: 73), and language comprehension is well preserved. **Alexia with agraphia** results from a 'lesion in the posterior margin of the language area, i.e. the angular gyrus' (Goodglass & Kaplan, 1972: 73), and spontaneous speech and comprehension remain virtually entirely preserved.

To these syndromes are added **pure aphasias**: aphemia, pure word-deafness, pure alexia or pure word-blindness, pure agraphia. Second, there are three **disconnection syndromes** (tactile aphasia, unilateral agraphia and apraxia, hemi-optic aphasia). For the record, beside multi-modal aphasic syndromes, unimodal or modality-specific aphasia types also occur.

Based on the same model, **Andrew Kertesz** (1979, 1982b) in Canada developed the **Western Aphasia Battery (WAB)** (Kertesz, 1982a) which, similarly to the BDAE, reaches a classification on the basis of testing of verbal and non-verbal functions according to classic, anatomically based doctrine. A controversial feature of the WAB, however, is that it permits, and even forces, a classification for all individuals with aphasia.

The BDAE has been translated into a number of languages and formed the model for the development of various offspring. In Germany neurologist **Klaus Poeck** and colleagues developed the **Aachen Aphasia Test** (AAT; Huber, Poeck, Weniger, & Willmes, 1983) a battery that was inspired by developments in Boston and resembles the Boston classification in many ways. The differences are that the AAT has no unimodal aphasia (like pure agraphia, for example), distinguishes 'standard' syndromes (more frequent) and 'non-standard' syndromes (less frequent). The AAT is widely used throughout Europe and has been translated and validated in several European languages, including English (Miller, Willmes, & De Bleser, 2000).

A recent survey in English-speaking countries showed that the most popular tests used in clinical practice are the BDAE and its variants and offspring (e.g., WAB) (Katz et al., 2000).

Summary

Developments following the Second World War led to increased endeavours for aphasic survivors of the war and the major conceptual thrust was holistic (in the tradition of Head, Marie, Goldstein). A significant Soviet aphasiology

emerged and aphasiologists like Luria attempted to bridge between holism and localisationism. In significant part as a result of the Second World War, major theoretical and clinical endeavour came predominantly from North America. Geschwind reintroduced anatomically based aphasia theory as neoclassicism in the 1960s, which would become the dominant diagnostic paradigm of its time spearheaded by Harold Goodglass and others at the Boston Aphasia Research Center.

Part II

Aphasiology to the millennium

Overview of Part II

> Let us be extravagant for just one moment: let us invent one other ology; let us speak of aphasiology.
>
> (Lenneberg, 1960: 97)

In the chapters that follow this short overview we examine the history of aphasia in the second half of the twentieth century, ending with the turn of the century, and briefly review some current developments that are so close to us in time that a 'historical' treatment may seem inappropriate; but the recent history of aphasia is history none the less. It is not our intention to critically review the current state of aphasiology, but to examine the relationship that twentieth-century developments have to the older history of aphasia. For contemporary discussion of current aphasiology there are a number of introductions and reference works which present the current situation comprehensively (e.g., Blanken et al., 1993a; Caplan, 1992; Davis, 2000; Denes & Pizzamiglio, 1999; Ellis & Young, 1988; Fabbro, 1999; Hillis, 2002; Kirshner, 1995; Lafond et al., 1993; LaPointe, 1997; Rapp, 2001; Stemmer & Whitaker, 1998).

There was an enormous expansion of research and increase in the number of publications after the Second World War with the founding of scientific journals, like *Brain and Language* (founded in 1974) and *Aphasiology* (founded in 1987) and the *Journal of Neurolinguistics* (founded in 1985) devoted to aphasia and related deficits in communication and their treatment. In addition, a number of societies dedicated to aphasia were formed, including the *Academy of Aphasia* (1960; Sarno, 1986), the annual *Clinical Aphasiology Conference* (starting in 1969) and the more recently instigated annual European *Science of Aphasia Conference*, as well as countless national societies holding yearly conferences and symposia. As Caplan (1987) put it:

> After World War II an international scientific community developed, complete with international societies, journals, academic structures, and related institutions, one of whose interests was linguistic aphasiology.

> Observations regarding the language abnormalities seen in aphasia became more numerous and more detailed.
>
> (Caplan, 1987: 143)

Our fascination with the processing of our own brains has produced a massive growth in human neuroscience, facilitated and even led by the development of sophisticated brain imaging techniques. We have seen in previous chapters that the functional deficits that follow brain damage provided a useful source of data in the search for the neural representation of language, but the improvements in imaging have meant that the changes in electrical activity and blood flow that take place in the healthy brain during cognitive and behavioural activities can be captured and measured to provide sometimes converging, and sometimes diverging, evidence. This overview will highlight only the major trends in this recent history.

Another more recent feature is an increased interdisciplinary interest. Medicine is no longer the leading discipline. Neuropsychology, neurolinguistics and speech and language pathology now contribute and interdisciplinary teams are engaged more than the individual investigator in taking the field forward and, while aphasia research is pursued all over the developed world, most originates from the English-speaking world, especially from the USA, but many countries, especially Italy, the UK, Germany, Canada, The Netherlands, Australia and Japan, punch well above their weight when population size and gross national product are taken into consideration.

As discussed in Chapter 6, neurologically orientated medical aphasiology (as represented, for instance, by the Boston School and fellow travellers) took the neoclassical syndrome approach of Geschwind and became influenced by the developments in brain imaging. The results of the explosion in imaging research in cognitive neuroscience in the later twentieth century began to build an expanded and more complex picture of the representation of language and speech in the brain. They showed that the lesion study approach is insufficient, but that some of the strongest evidence emerges where there is convergence of some kind between imaging with non-brain-damaged participants and studies with aphasic participants. Developments in imaging have, for instance, led to investigation of subcortical aphasia and have expanded understanding of language processed by the right hemisphere, and these developments are discussed in Chapter 10. In the 1960s there was a revolution in linguistics inspired by the work of Noam Chomsky in the USA, which had a significant influence in psychology and on aphasiology both directly and indirectly, reviewed in Chapter 7, and on the subsequent development of cognitive neuropsychology, discussed in Chapter 8. Neurolinguistic investigations of aphasia, particularly of agrammatism, attempted to identify the essential linguistic features of aphasic impairments and comparative studies of aphasic data in different languages began in earnest.

Psycholinguistics, a coming together of linguistics and psychology, developed out of the linguistic revolution of the 1960s and attempted to bring a

rigorous experimental methodology to investigations of language behaviour. In the 1970s another blending of disciplines emerged with cognitive neuropsychology, when cognitive psychologists became interested in testing and developing their new models of reading and writing with people with brain damage. Early psycholinguistic studies in aphasia took the neoclassic model as a theoretical base, but fierce theoretical and a clinical criticisms of the model and the research paradigms used to test it arose from both perspectives.

A central issue became a methodological one: how valid and reliable was data obtained from groups of people with aphasia classified into aphasic syndromes compared to data obtained from the detailed investigation of single cases using psycholinguistically controlled tests? Some cognitive neuropsychologists were interested too in whether treatments could be theoretically motivated from the models that were developing of the cognitive architecture of reading, writing, speaking and understanding. This led to the development of new approaches to assessing aphasia for rehabilitation.

With a gradual reduction of influence of Chomskian linguistics, many linguists and psychologists became interested in communication, discourse, pragmatics, conversation, and other aspects of language in social contexts and beyond the sentence, and studies with brain-damaged people began to show that lesions to areas of the brain other than the left peri-Sylvian language area produced impairments in language and communication. Aphasiologists too became aware that language was but a part of communication and this fact could be exploited in therapy. In parallel a clearer realisation emerged that brain damage did not only cause cognitive impairments, but that it could also have significant psychosocial consequences: the experience of aphasia could also have a negative impact on the emotional and everyday social life of the individual. The World Health Organisation developed an international classification of diseases and the impairments and disabilities they cause, and clinical approaches to aphasia were influenced by this framework in the same way that clinical approaches to other conditions were. So the study of aphasia was no longer solely concerned with a narrow focus on impairments of language arising from damage to a simple set of centres and routes between centres.

Geschwind's neoclassic model had become especially influential in medicine, and as discussed in Chapter 6, manifested in the Boston School and embraced the newly developing imaging methods. This led to investigations of subcortical aphasia, among other things, and more recently progressive aphasias. The influence of the neoclassical model was worldwide, as discussed in Chapter 6. In Germany, the classical model again became central, partly as a result of Geschwind's influence via **Klaus Poeck** (1926–2006) who gathered an interdisciplinary group of linguists, neuropsychologists and therapists around him in Aachen, where the Boston Test inspired the development of the influential **Aachen Aphasia Test (AAT)** in 1983 (Huber et al., 1983; Huber, Poeck, & Willmes, 1984). Geschwind's model had a similar impact in Italy,

where **Ennio De Renzi** and colleagues in Milan laid the foundations for the development of a vibrant neuropsychology that subsequently embraced cognitive neuropsychology in the 1980s. The **Token Test** was published in 1962 (De Renzi & Vignolo, 1962) and the influential journal of neuropsychology, *Cortex*, was founded by De Renzi in 1964.

War continued to produce many dead, and many survivors with brain injury. The Vietnam War (1954–1975) was no exception and 200,000 South Vietnamese soldiers, 1 million North Vietnamese soldiers and 500,000 civilians were killed. Between 1961 and 1975, 56,555 US soldiers were killed, one-fifth by their own troops (*Hutchinson Dictionary of World History*, 1994). A very large study was undertaken with funding from the US military and the US Veterans Administration and all of the head-injured soldiers from the Vietnam War, and non-head-injured controls from the same war, were taken to the Walter Reed Army Medical Center in Washington, DC over several years and received extensive cognitive, neurological, speech and language testing. Christy Ludlow designed the speech and language testing and Jordan Grafman the neuropsychology testing; CT scanning was conducted on all participants with analysis focusing on relating the CT findings to the cognitive and speech and language outcomes. A huge database was collected, partly because of the improved medical units in the field, which meant that the head injury survival rate was much greater than in any previous war (personal communications, Christy Ludlow and Jordan Grafman). A large number of published studies resulted, focusing on psychosocial (Salazar, Schwab, & Grafman, 1995), occupational (Kraft, Schwab, Salazar, & Brown, 1993), employment (Schwab, Grafman, Salazar, & Kraft, 1993), and cognitive and language outcomes; follow-up studies are still underway.

7 The rise of linguistic aphasiology

There had already been serious attempts in the nineteenth and early twentieth centuries to establish linguistics as a central discipline relevant for aphasiology (for instance by the linguist Steinthal and the physician Pick). At the beginning of the 1940s a seminal work on aphasia by linguist Roman Jakobson was published which had little impact until its translation into English in the 1960s. A broad linguistic aphasiology developed in the wake of Noam Chomsky's generative transformational grammar, which was to have a revolutionary influence in linguistics, cognitive psychology and philosophy.

Psycholinguistics, a coming together of linguistics and psychology, developed out of the linguistic revolution of the 1960s and attempted to bring a rigorous experimental methodology to investigations of language behaviour. In the 1970s another blending of disciplines emerged with **cognitive neuropsychology**, when cognitive psychologists became interested in testing and developing their new models of reading and writing with people with brain damage. Early psycholinguistic studies in aphasia took the neoclassic model as a theoretical base, but fierce theoretical and a clinical criticisms were levelled against the model and the research paradigms used to test it. In the latter half of the twentieth century philosophers of language, linguists, and psychologists began to recognise that language was more than a cognitive process. Language is also behaviour and has a social function and is not only more than single words, but is more than sentences. Research developed into the behavioural and social uses of language and these too were to have a significant impact on aphasiology.

Roman Jakobson

Russian literary and linguistic scientist **Roman Jakobson (1896–1980)** is often considered the first to seriously apply linguistics in aphasiology (e.g. Goodglass & Blumstein, 1973c) although Steinthal is probably more deserving of this position. Jakobson and fellow Russian, **N. S. Trubetskoy** (1890–1938), are the most well known founding members of the Prague Circle or School of Phonology, which was established in 1926 (Fischer-Jørgensen, 1975). When the Nazis entered Czechoslovakia, Trubetskoy died of a heart attack when

facing prosecution by the political police and Jakobson fled, first to Denmark, Norway and Sweden, and finally to the USA in 1941, where he eventually became professor at Harvard and The Massachusetts Institute of Technology (Fischer-Jørgensen, 1975). In his 1941 monograph, *Child Language, Aphasia, and Phonological Universals*, Jakobson focused mainly on phonology. He saw parallels between language acquisition and the language loss of aphasia and proposed a **regression hypothesis**, which holds that we can observe the same processes in both developing child speech and in the impairments of aphasic speakers, but in reverse. 'The dissolution of the linguistic sound system in aphasics provides an exact mirror-image of the phonological development in child language' and 'the order in which speech sounds are restored in the aphasic during the process of recovery corresponds directly to the development of child language' (Jakobson, 1968: 60, 62). Further, the 'development of child language [and] the decomposition of aphasic speech [. . .] display a series of common fundamental laws'. In phonology, Jakobson believed that the 'development moves from an undifferentiated initial form to increasing differentiation and separation [. . .] and the development begins with the higher layers' (Jakobson, 1968: 64).

He applied this general 'law' to grammar and syntax also, and predicted 'that, beside the phoneme system [. . .], also the grammatical components of language are subject to the same layered overlay'. Jakobson distinguished between primary and secondary grammatical components of grammar. A secondary component 'develops in children after the primary, disappears in aphasics before the primary' (Jakobson, 1968: 64).

For Jakobson it was obvious that aphasia should be described linguistically and understood in terms of linguistic theories. In addition, aphasia could test the validity of linguistic theories. Jakobson (1968) also attempted to contrast Luria's six aphasia types in terms of three linguistic dichotomies: encoding (combination, contiguity) impairments versus decoding (selection, similarity) impairments, limitation impairments versus disintegration and sequence (syntagmatic, successivity) versus concurrence (paragdigmatic, simultaneity). For instance, an encoding impairment, like Luria's dynamic aphasia or efferent motor aphasia, is characterised by problems with combination, whereas decoding impairments, like sensory or semantic aphasia, entail impairments in selection. While Jakobson is regarded a pioneer of linguistic aphasiology, his ideas had little impact on the mainstream and figure very little in contemporary linguistic research.

Chomsky's generative linguistics and the consequences

American linguist **Noam Chomsky** (born 1928; Newmeyer, 1980: 33f.) laid the bedrock of **transformational generative grammar** at the end of the 1950s and the beginning of the 1960s (Chomsky, 1957b, 1965) and subsequently linguistic science emerged as a dynamic enterprise in the 1960s. Chomsky's (1957a) famous critique of **B. F. Skinner**'s book *Verbal Behavior* dismissed

behaviourist accounts of language development as due simply to stimulus–response learning. For generative linguistics, children do not learn a set of utterances through a process of imitation and reinforcement, but learn a set of rules and have an innate capacity for language acquisition.

Chomsky proposed that the crux of understanding language lies in linguistic **competence**, contrasted with linguistic **performance**. Competence is the abstract system of mental representations and processes that constitutes the basis of language, and performance is the actual realisation of language through use. Generative linguistics has at its heart the notion of a **universal grammar** which developed during the 1960s and 'by 1970, if not before, it was clear that transformational generative grammar had become the "established" linguistic theory in the United States' (Newmeyer, 1980: 20) and became influential in linguistics departments worldwide.

In addition, Chomsky identified linguistics as a branch of cognitive psychology, because the theory of a language 'is a psychological model of an area of human knowledge' (Newmeyer, 1980: 42), and is subject to scientific laws. It is hence not surprising that psycholinguistics and the development of experimental investigations of language processing grew and that the interest in mental representations and processes led to a dynamic interchange on how the psychological reality of linguistic constructs might be verified (see for instance Miller, 1964). Among other things, there was a realisation that aphasia was relevant for linguistics and psycholinguistics.

The connection between linguistics and aphasia is complementary. On one hand, linguistics has methods for describing aphasic errors (aberrant language is also language), and on the other, aphasic data can be used as 'external' evidence for linguistic hypotheses and models (of competence). Grodzinsky (1984: 101), for instance, argues as follows: 'Since most language deficits are selective, and the selectivity presumably runs along some grammatical lines, formal grammars must serve as the framework for the description of the functional impairment.' For theoretical linguistics, the structurally based deficits of aphasia are of great interest because they provide evidence for the cognitive reality of linguistic structures.

> So, if a case can be found, where the brain damaged patient is able to understand sentences of some syntactic type, but unable to correctly interpret others, this has some potential interest for the theory of syntax. What we might have in this case is selective impairment to language mechanisms, where the selectivity is governed by structural principles.
>
> (Grodzinsky, 1986: 180f.)

A case was made for the correlation between the neoclassic aphasic syndromes with certain linguistic deficits using group studies. Goodglass and Blumstein (1973b) describe their work from the 1960s as follows:

> Goodglass and his co-workers were the first to introduce experimental

> and quantitative methods into the study of syntax of agrammatics to identify those particular linguistic operations which best discriminated between the clinically agrammatic patient and the non-agrammatic, including the paragrammatic. Just as there is for syntax, so there is for each of the other levels of linguistic organisation – phonology, morphology, lexicon, semantics – a corresponding type of aphasia in which that level is most, if not exclusively, impaired.
>
> (Goodglass & Blumstein, 1973b: 6)

In sentence comprehension, for instance, research suggested, among other things, that the person with Broca's aphasia understands sentences via content words, but can have problems comprehending syntactic markers. People with Wernicke's aphasia, on the other hand, can process syntactic markers, but they can have difficulties in understanding the semantics of content words. This led to the position that the speaker with Broca's aphasia and agrammatism has a central syntactic deficit (e.g., Berndt & Caramazza, 1980; Caramazza & Zurif, 1976), whereas the speaker with Wernicke's aphasia has a semantic deficit with allegedly preserved syntactic capabilities (e.g., Caramazza & Berndt, 1978). Much (psycho)linguistic research in aphasias at this time involved studies of groups formed according to the classic taxonomy (Broca's, Wernicke's, conduction aphasia, etc.), who were examined with reference to different linguistic components, including phonology (e.g., Whitaker, 1988), morphology (e.g., Nespoulous & Villiard, 1990), semantics and syntax (e.g., Kolk, 1998).

These linguistic analyses result in a clearer description and understanding of aphasic performance and 'significantly deepen our understanding of language breakdown' (Caplan, 1987: 149). However, it gradually became clear from these studies 'that different patients who had been classified into a syndrome display different language disorders in reality' (Caplan, 1987: 149). Thus these studies resulted in a re-evaluation of the idea of aphasic syndromes. Caplan (1987: 151) concluded: 'There are many problems for the traditional clinical taxonomy which are raised by the introduction of qualitative linguistic and psycholinguistic descriptions of aphasic symptoms.' The issue was taken up by others, especially with the development of cognitive neuropsychology.

Aphasia in context: beyond the sentence

As detailed in previous chapters, the study of aphasia tends to confirm the view that the left hemisphere is responsible for those aspects of language that can be characterised through a formal unit-and-rule linguistic model providing an account of the linguistic structure of sentences. Aphasic individuals have problems at the structural linguistic levels of phonology, morphology, syntax and lexical semantics.

But there is more to language than its componential and combinatory

structure – syntax, morphology, phonology. The behavioural and social 'context' is a significant feature of language use. **Pragmatics** concerns the interface between language use and other aspects of behaviour and is concerned with the total behavioural–social context in which communication takes place. There has been an increased interest in pragmatic uses of language in brain damage in recent years reflected in developments in linguistics, sociolinguistics, social psychology and speech and language pathology. The 1960s saw Chomsky's revolution in cognitive perspectives on language, but this overshadowed to some extent other **functional linguistic** theories emerging around the same time, such as the development of **M. A. K. Halliday**'s **Systemic Functional Linguistics**, a theory of language that focuses on language *function*. It does not ignore syntactic structure, but it considers the function of language as central and is concerned with what language does, and how it does it, and with the social context of language use (Halliday, 1961, 1985). Other developments in the 1960s in the philosophy of language that had an impact in aphasia included Grice's **Speech Act** theory (Grice, 1968), which is concerned with the application of the rules of logic to the analysis of conversation.

Paradis (1998: 7) stated: '[I]t has become apparent that language is not just the language system, which has been the focus of attention of linguists and aphasiologists over the years, but linguistic competence plus pragmatic competence.' In studies with brain-damaged people the interest has come from researchers concerned with the communicative abilities of those with aphasia from left hemisphere damage, from right hemisphere damage (Code, 1987; Joanette, Goulet, & Hannequin, 1990) and the impairments and disabilities of those with traumatic brain injury (McDonald, Togher, & Code, 1999).

'Communication' was barely considered in historical approaches to aphasia, but interest in aphasia comes from a realisation that non-combinatory aspects of language and communication can be affected by brain damage, and should not be considered *secondary*, as they were historically, if considered at all. However, aphasic people can also have substantially retained non-combinatory language and communication, and this appreciation has been integrated into therapeutic approaches to aphasia.

So pragmatics (Leech, 1983; Smith & Leinonen, 1992) is concerned not with the cognitive function of language, but with its social function and its communicative use (Feyereisen, 1991). Research has examined the relationship of verbal and non-verbal communication and the interaction of conversation partners with one another (conversation analysis, discourse structure).

Paralinguistic and non-verbal communication

For classic aphasiology, by definition, only the componential and combinatory aspects of language are affected in aphasia and it is assumed that aphasic speakers are not communicatively impaired (Holland, 1982). In other words, aphasic speakers communicate better than they speak (Feyereisen, 1991). The

more severe the aphasia, the more necessary it can become to use non-verbal communication like gesture and pantomime, drawing and facial expressions, as compensation. Recall that Broca's patients Leborgne and Lelong could both apparently convey essential contents non-verbally, despite the nearly total absence of speech. According to Broca both could make themselves understood through gesture, though examinations of gesture were not described.

We have seen in earlier chapters that the definition of aphasia oscillated around the issue of the role of non-verbal features of communication in aphasia; Finkelnburg's asymbolia is an example (Chapter 4). Research revealed that aphasic communicators could have more difficulties in understanding and producing non-verbal communication (pantomime, gestures, facial expressions) than non-aphasic brain-damaged people, which led to some revival of the idea of aphasia as a form of asymbolia (Duffy & Buck, 1979; Duffy & Liles, 1979; Feyereisen & Seron, 1982a, 1982b). The term **aprosody** had been introduced by Norwegian neurologist **Monrad-Krohn** in the late 1940s to describe impairments in prosody, 'the melody of language', although prosody provides more than melody to spoken language, covering the role of stress, rhythm and intonation in speech. Monrad-Krohn (1947) introduced the term to explain the most famous case of someone sounding like they had a foreign accent following brain damage. The term **foreign accent syndrome** was coined by Whitaker (1982) to describe this rare phenomenon (Moen, 1996, 2000; see the special issue of the *Journal of Neurolinguistics*, vol. 19, no. 5, 2006). Pick (1919) had described a case of a Czech butcher who sounded like he had a Polish accent following a stroke, and this seems to be the first description. Monrad-Krohn described a Norwegian woman, Astrid L, who developed a German-sounding accent following a shrapnel wound to the brain. Sounding like a German to listeners in German-occupied Norway during the Second World War would cause significant social and personal problems so the condition had serious consequences for her. Monrad-Krohn explained her condition as due to altered prosody and a number of cases have been described, although the condition is rare, and there is clear heterogeneity between cases, with altered vowel and consonant production compounded by prosodic stress assignment impairments (see Gurd & Coleman, 2006, for review and Moen, 1996, for discussion of Monrad-Krohn's case.)

However, despite limitations in people with aphasia, the non-verbal communication of the more severely aphasic person can still be superior to their linguistic communication. The observation is particularly relevant for therapy because a major component of modern approaches to intervention is the enabling of aphasic people to participate as independently and autonomously as possible in the communicative interactions of everyday life. It is not the means by which a communicative goal is achieved that counts, but its success in achieving communication.

The interest in pragmatics spurred the development of new approaches to

therapy that focused on communicative goals and their achievement, and less on the grammatical or articulatory correctness of language use. An early approach developed by Davis and Wilcox (1981, 1985) that had significant impact is **PACE** (Promoting Aphasics' Communicative Effectiveness). A brief presentation can be found in Edelman (1987) and an extensive discussion in Carlomagno (1994). The central feature of PACE is that every possible channel for communication is permitted, and even desired, in order to convey messages.

Therapy following brain damage can take two broad approaches: therapy that aims for **restoration** of lost functions and therapy that aims for **compensation** for lost functions. One view has been to see these approaches as relevant to different stages in recovery from aphasia and to different severities of aphasia. Thus a clinician may decide that therapy should aim to restore lost syntax or naming during the early months following the stroke or where the impairments are relatively mild or moderate, but that a general compensatory approach which aims to maximise communicative effectiveness is more relevant at later chronic stages post-stroke or with more severely impaired individuals.

Conversation analysis and discourse

A feature of human interaction that strongly reflects the social function of language use is conversation. Often great ideas are not exchanged in conversations, which can be used to share gossip and engage in 'social grooming', made up often of significant amounts of formulaic and nonpropositional talk. Conversations follow specific rules, however, and have specific structure. An early inspiration to the investigation of conversations was the philosopher Grice's work on what he called the **cooperative principle** (Grice, 1968). **Conversation analysis** is a range of methods that identifies and investigates these rules and structures and became increasingly important in the observation of aphasic speech behaviour in the 1990s (Goodwin, 2003a; Hesketh & Sage, 1999; Perkins, 1998). We briefly discuss three selected topics that have been investigated in aphasia below. These are turn-taking, repair, and the role of the aphasic person's communication partner. For a range of studies on conversation in aphasia, the special issue devoted to conversational analysis in *Aphasiology* (1999, vol. 13, nos. 4–5) and Goodwin (2003a) are recommended.

Participants in a normal conversation speak in turns and the taking over of the speaker role is called **turn-taking**. The relinquishing of a conversational turn is normally indicated by the speaker (through pauses, falling intonation, decreasing gestures, eye contact) or attributed (through questions, requests). When a listener wishes to take a turn to speak, the request is normally accepted, or the right to speak is claimed (for instance by interrupting). Although most aphasic people know the rules of turn-taking and they have no primary deficit in this connection, their impairments often interfere with

attempts to end or take a turn because they may produce too long a pause (for instance during word finding) which is misunderstood by the communication partner as a request to take a turn, or because the communication partner interrupts because they are unable to understand the aphasic conversationalist's speech (Ahlsén, 1985; Lesser & Milroy, 1993). Aphasic speakers are also often too slow for their conversational partners when it comes to taking over or taking back the right to speak. But studies show not only that aphasic conversationalists are given the right to speak more often than non-aphasic speakers, but also that their relatives often speak *for* them, taking away their opportunity to take over the speaker role as appropriate for the situation.

Another important feature of conversation examined in aphasiology is called **repair**, which refers to a variety of behaviours in conversations that deal with 'troubles' in talk (Lesser & Milroy, 1993; Perkins, 2003). A repair may be initiated when a conversationalist feels that a problem has occurred in their own or the other's talk or due to a failure of understanding. A range of studies have been conducted into repair behaviour in aphasic speakers and their conversational partners and the extent to which conversation participants are able to solve problems together (Clark & Schaefer, 1989; Ferguson, 1994; Goodwin, 2003b; Lesser & Algar, 1995). Conversation analysis 'sees interaction as a result of cooperation between the aphasic and the non-aphasic conversation participants' (Perkins, 1998: 77) so the **communication partner** of the aphasic person makes an important contribution to the success or failure of the conversation. (For the collaborative construction of repair in aphasic conversation, see Perkins, 2003). It has been found that simple behavioural steps taken by the communication partner contribute much to the success, such as when speaking slowly and clearly and using simple sentence structures, avoiding abrupt changes in topic, and minimising background noises. When the non-aphasic partner is the listener, then the conversation is helped when the partner indicates non-comprehension and provides support in case of word-finding difficulties.

The appreciation of the importance of natural conversation for people with aphasia and their conversation partners and its analysis provided a new dimension that has had an important impact on compensatory approaches and on the social reintegration of aphasic people into their communities.

Discourse refers to a continuous stretch of language, and usually spoken language. So, technically a conversation is an example of discourse, although they can be, and are, separated, mainly because separate methods of analysis have been developed to examine conversation, as discussed above. ('Text' is another term sometimes used to mean the same thing, but technically refers to a stretch of spoken or written language *recorded* in order to conduct an analysis.) Impairments in discourse are mostly associated with speakers who have right hemisphere damage (Code, 1987; Joanette et al., 1990) or traumatic brain injury (McDonald et al., 1999). Right-hemisphere-damaged speakers often have difficulties with linking contextual information

in utterances, structuring narratives or stories, procedural texts, like describing how one wallpapers a room, and understanding metaphors and the punchlines of jokes, although the componential aspects of language (phonology, morphology, semantics, syntax) are intact. Joanette and colleagues summarise the problems as follows: 'Although right-hemisphere damaged patients master the elementary aspects of speech, they seem to be impaired in the use of language in context' (Joanette et al., 1990: 186).

Assessing functional communication

The gradual appreciation that pragmatic aspects of communication are important in rehabilitation led to the development of a range of assessments of 'functional' aspects of communication which attempt to measure less the structural language deficits and more the communicative functions. This is no place to review all the measures of communication that have been developed, but several important developments will be mentioned briefly.

The **Functional Communication Test** (**FCP**) was an early measure developed by **Martha Taylor Sarno** (Sarno, 1969). She argued that standard batteries give a false impression of a person's communicative abilities because they measure *clinical* performance of linguistic aspects of language rather than the person's ability to cope with everyday communicative situations. A measure of functional communication for Taylor Sarno is where 'the conditions or stimuli used are informal and they sample language usage without task presentation' (Taylor, 1965: 102). The difference between a clinical and a functional assessment is contrasted when a patient is asked to write their name to command in the clinic and when they sign a cheque in a bank. The FCP is not concerned with aphasia types or diagnostic categories and has no specific theoretical base, except its distinction between clinical and functional aspects of assessment. The original FCP contained 45 everyday communicative functions, like ability to indicate 'yes' and 'no', reading street signs and recognition of names of familiar objects, and used first-hand observation by the tester wherever possible gathered during conversational interaction. Abilities are rated and converted into percentages and the overall score is expressed in terms of pre-aphasic abilities. An overall score of 59 per cent, for instance, suggests that the aphasic person is performing at 59 per cent of their pre-aphasic communicative ability.

The **Pragmatic Protocol** (Prutting & Kirchner, 1987) consists of 30 items divided into three major aspects and seven subgroups. Users are required to determine whether a person's interactional behaviours are *appropriate* or *inappropriate* on such communicative acts as speech acts, topic use and turn-taking, as well as prosody, body posture, gesture and facial expression. It uses Grice's rules of conversational behaviour as the theoretical basis of the protocol and the integration of verbal with paralinguistic and non-verbal behaviours. Another development was the **Communicative Abilities in Daily**

Living (CADL), developed by **Audrey Holland** (1980), a test of functional communication for aphasic patients.

In addition, the relatives of aphasic people became involved in functional assessment in the **Communicative Effectiveness Index (CETI)** (Lomas et al., 1989). This is a questionnaire for the relative who answers 16 questions about the aphasic person's functional communication abilities, which they rate on a semantic differential scale from 'not at all able' to 'as able as before the stroke'. A more recently developed functional measure is the **Inpatient Functional Communication Interview (IFCI)** (O'Halloram et al., 2004) that uses a natural interview to assess the functional abilities of hospital in-patients. For further review of pragmatic assessments in aphasia see Manochiopinig, Sheard, and Reed (1992).

The social and psychosocial consequences of aphasia

Closely related and influenced by developments in pragmatics and social models of language, workers became interested in the social uses of communication in people with aphasia and the psychosocial consequences of aphasia. The emphasis was on dealing with aphasic disability rather than repairing aphasic impairment. In 1980, the World Health Organisation developed a new version of a framework that considers disease from four perspectives: causes, symptoms, functional deficits resulting from symptoms, as well as psychosocial consequences of a disease. Social approaches to aphasia emerged from social models of disability that emphasise that disability arising from failures in society which set up barriers to inclusion for people with aphasia; the problem lies in society's failure to accommodate people with aphasia not in the aphasic individuals' impairments giving rise to social barriers and oppression (Jordan, 1998) and problems accessing services and engaging in authentic inclusion. The main aims of the social approach are: to increase successful participation in authentic communication events; to focus on communication at the level of conversation; to provide communicative support systems within the speaker's own community; to increase communicative confidence and empower speakers with aphasia (Simmons-Mackie, 1998).

Social approaches differ from functional approaches. The functional approach concerns itself mainly with 'transactional' information exchange, in activities like ordering a meal, buying a ticket or phoning a bank (Worrall & Frattali, 2000), whereas a social approach is concerned with the 'interactional' exchange in the aphasic person's use of communication to establish and maintain relationships and everyday conversational interaction with others. This could entail things like chatting in a restaurant, expressing emotions to a partner or gossiping with neighbours.

Several centres in different countries have been established on a not-for-profit basis to develop social approaches for people with aphasia. At the **Pat Arato Centre** in Toronto, Canada (www.aphasia.ca/about/centre.shtm)

Aura Kagan and colleagues have promoted **supported conversation** (Kagan, 1998; Kagan et al., 2001), which provides training for the conversation partners of people with aphasia and stresses the importance of providing the aphasic conversation partner with the support they might need to engage in conversational exchange. The **Aphasia Centre of California** (www.aphasiacentre.org) established by Roberta Elman and colleagues provides a range of socially relevant approaches, including a supportive group approach (Elman, 2004). In London, Sally Byng headed a group who set up **Connect** (www.uk connect.org) which is a focus for socially inspired services, research and professional training (Parr, Duchan, & Pound, 2003). The social experience of people with aphasia appears to have a marked influence on their psychosocial and emotional life.

The term **psychosocial** refers to the grounding of emotional experience within social context. There had been little interest in the psychosocial consequences of aphasia until the 1960s and 1970s, apart from the work of Kurt Goldstein discussed earlier (see Chapter 5), when a few investigators, recognising that aphasia could have significant psychological, as well as linguistic, impact on the individual and their families, began to conduct research. We will summarise only briefly some of this work, but the interested reader is referred to Sarno and Gainotti (1998) for an outline of the history of the topic, Müller and Code (1989) for introductory discussion of some of the background research up to 1979, and to Lafond et al. (1993).

Our personal sense of well-being comes from our current experience of life as a whole and the positive and negative aspects of our emotional life both come from our interaction with others in society. How we perceive this determines the quality of our life experiences. Most of our happiness and sadness comes from our interactions with others, whether directly or indirectly through music, reading, TV, art, and so on. The psychosocial consequences of aphasia affect professional, social and family activity and relationships and can produce psychological changes (Code & Herrmann, 2003; Code, Hemsley, & Herrmann, 1999).

Coping with aphasia and the depression that can co-occur for aphasic people and their relatives have been areas that have received attention from researchers. There have been attempts to model the stages or phases that affected people experience in coming to terms with aphasia (Tanner, 1996). These stages have been conceptualised in terms of the **grief model** by Tanner and Gerstenberger (1988), devised originally to describe the stages that a bereaving person goes through. The first stage entails crisis management and an unwillingness to accept the situation may occur, most often connected to the (most often unjustified) hope that language abilities will return. In the second stage, when the permanence and extent of the problems are recognised, irritation, anger, and frustration occur. In the third phase, the individual becomes depressed, discouraged, desperate, and without drive. The fourth stage is characterised by acceptance and the new state of affairs is accepted.

Depression after stroke and with aphasia are very common, with up to 30 per cent experiencing severe depression at some time (Robinson & Starkstein, 1990). Other authors, for example Williams (1996), even suggest that it is higher (up to 80 per cent). Herrmann, Code and colleagues (Code & Herrmann, 2003; Herrmann, Bartels, & Wallesch, 1992, 1993a) have developed a model that describes the different types and causes of depression at different stages post-stroke. Primary depression is the consequence of neuro-endocrinological, neuro-chemical or anatomical changes in the brain and characterises the acute stage (0–3 months post-stroke). Secondary and tertiary forms of depression are reactive to psychosocial, neuropsychological and functional impairment and disability. Secondary depression typically occurs within the first six months after the stroke, when the person affected recognises the full extent of his or her impairments (for instance during a period in rehabilitation). Tertiary depression occurs later, when individuals fully realise the consequences of their disabilities on their everyday life, which they were unaware of while in rehabilitation.

Summary

Linguistic aphasiology developed methods of linguistic and psycholinguistic analysis that have revealed that the classic syndromes display very heterogeneous symptom combinations, and provided a sophisticated range of methods for the investigation of aphasic speech and language. The linguistics revolution of the 1960s provided theoretical models of language and led to the development of psycholinguistics and eventually to cognitive neuropsychology. In the 1970s and 1980s aphasiologists began to be influenced by developments in the social functions of language use and began to appreciate that the social function of language was an important dimension with significant implications for clinical aphasiology. Their investigations revealed that the social functions of language too can be impaired with brain damage, but for many people with standard forms of aphasia the social functions of language could be substantially unimpaired. This knowledge became the basis for the development of new methods of assessment and approaches to treatment.

8 Cognitive neuropsychology of language and the rise of cognitive neuroscience

Neuropsychology is concerned with the relationships between brain structure and mental function and Gall's and Broca's investigations can be seen as the beginnings of neuropsychology. In classical neuropsychology the attempt is always made to clarify the relationship between neuronal substrate and mental function (e.g., language processing). In the 1970s a paradigm called **cognitive neuropsychology** developed that had a significant impact on neuropsychology in general and aphasiology in particular and it differed from classic neuropsychology in some fundamental respects (Coltheart, Patterson, & Marshall, 1980; Coltheart, Sartori, & Job, 1987; Denes & Pizzamiglio, 1999; Denes, Semenza, & Bisiacchi, 1988; Ellis & Young, 1988), and was to some extent a reinvention of neuropsychology. This contention is supported by the fact that its three major features – modularity, an information processing metaphor, and a single case methodology – are not new, but already existed in the nineteenth and beginning of the twentieth centuries. Additionally, psycholinguistics and linguistic aphasiology used some of the same approaches as cognitive neuropsychology. The approach developed mainly as a result of experimental cognitive psychologists wanting to test and develop their **information processing** models of various aspects of human cognition on brain-damaged individuals. Contributors came from North America, Europe and Japan, but the approach developed originally in the UK, and mainly through the early work of John Marshall, John Morton, Tim Shallice, Elizabeth Warrington, Karalyn Patterson and Max Coltheart, and in 1985 the journal *Cognitive Neuropsychology* was founded by **Max Coltheart**. So, the separate features of the enterprise are a coming together of the information-processing metaphor, the notion of a mind organised into modules and the development and testing of models against the broken cognition of brain-damaged individuals using psycholinguistically controlled (in the case of language studies) tests. Early models, such as Morton's logogen model, came out of the psycholinguistics lab, but it was the clinic that provided the data that drove the enterprise.

While it is unwise to pinpoint the beginnings of any movement in science to one study, there is some justification for tracing the beginnings of cognitive neuropsychology back to a genuinely seminal paper by **John Marshall**

Figure 8.1 John Marshall (right) and John Morton in 2005. Photo courtesy of Professor Jennifer Gourd.

(1939–2007) (see Figure 8.1) and **Freda Newcombe**, who described the acquired impairments in reading we now know, after Marshall and Newcombe (1973), as deep dyslexia, surface dyslexia, and visual dyslexia, and for introducing a dual-route information processing model of reading aloud. It was with this important study that some of the essential features of the enterprise can be identified: the hypothesis-driven foundation for the work of an information processing model and a single-case approach to research using psycholinguistically controlled tests designed to test components of the model. We can note that the title of the article (not to mention the title of the journal in which it appeared) indicates that the authors were working within the province of psycholinguistics.

The assumptions of cognitive neuropsychology

There are a number of assumptions and axioms that serve as the basis for cognitive neuropsychology. Cognitive neuropsychology attempts to understand cognitive functions like language processing on the basis of information processing models. Among the data relevant for modelling, impaired functions play an important role, because it is assumed that impairments to a function provide evidence about its normal functioning. So aphasic deficits can be used

to test models of normal cognitive function and theories of normal cognitive function can be used to help explain disorders (Ellis & Young, 1988).

Models are usually based on the assumption that information processing is performed by a limited number of mental processing components, which work in an autonomous fashion uninfluenced by other steps in the process. This assumption is called the **modularity hypothesis**. The modularity hypothesis originated particularly with Jerry Fodor (1983), who drew on Gall's organology (Fodor, 1983: 131).

The modularity hypothesis supposes that the components of cognition, like language processing, action processing, face processing, are organised and represented in the brain within a modular architecture. Modules are domain-specific, in the sense that processes performed by them are specific to that module only; they are computationally autonomous and independent of other cognitive processes, probably genetically determined and associated with specific neural structures. The echo of Gall's voice is clear. In diagrammatic representations of such models, as in Figure 8.2, modules are represented by the boxes and interactions between modules by the arrows. Deficits in function can be caused by damage to one or more modules, or to the routes between modules.

Furthermore, there is an assumption that the pathological function is disturbed in a selective, 'meaningful' way. This is called the **fractionation hypothesis**, 'the belief that brain damage can result in the selective impairment of components of cognitive processing' (Caramazza, 1984: 10). This also means that unimpaired modules operate normally. Additionally, the approach assumes that the observable behaviour of aphasic people can be interpreted directly using models of normal processing. This is called the **transparency assumption**, which claims that the damaged cognitive system is incapable of creating 'new' cognitions: 'That relation (between impaired performance and normal cognition may be transparent in the sense that the hypothesized modifications of the normal processing system are traceable within the proposed theoretical frameworks' (Caramazza, 1992: 82) and the 'resulting behavior patterns do not represent the creation of new subsystems, rather, they reflect a reorganisation that emphasizes intact subsystems' (Saffran, Schwartz, & Marin, 1980: 221).

Naturally, the fractionation and transparency assumptions have not remained uncriticised, because 'such an undynamic conception of disease [is] highly problematic' (Kelter, 1990: 20), since observable changes in function in aphasia are described, as in recovery with time since onset, and the ability of the brain to reorganise is an established principle of neuropsychology. A further problem, as Caplan (1987) emphasised repeatedly, is that our knowledge of normal language processing remains meagre and it remains unclear how a specific language deficit reflects a disorder of the system.

The transparency assumption may present serious problems for radical variants of cognitive neuropsychology, therefore, especially as it would appear to be untestable, and brain damage produces patterns of behaviour that can

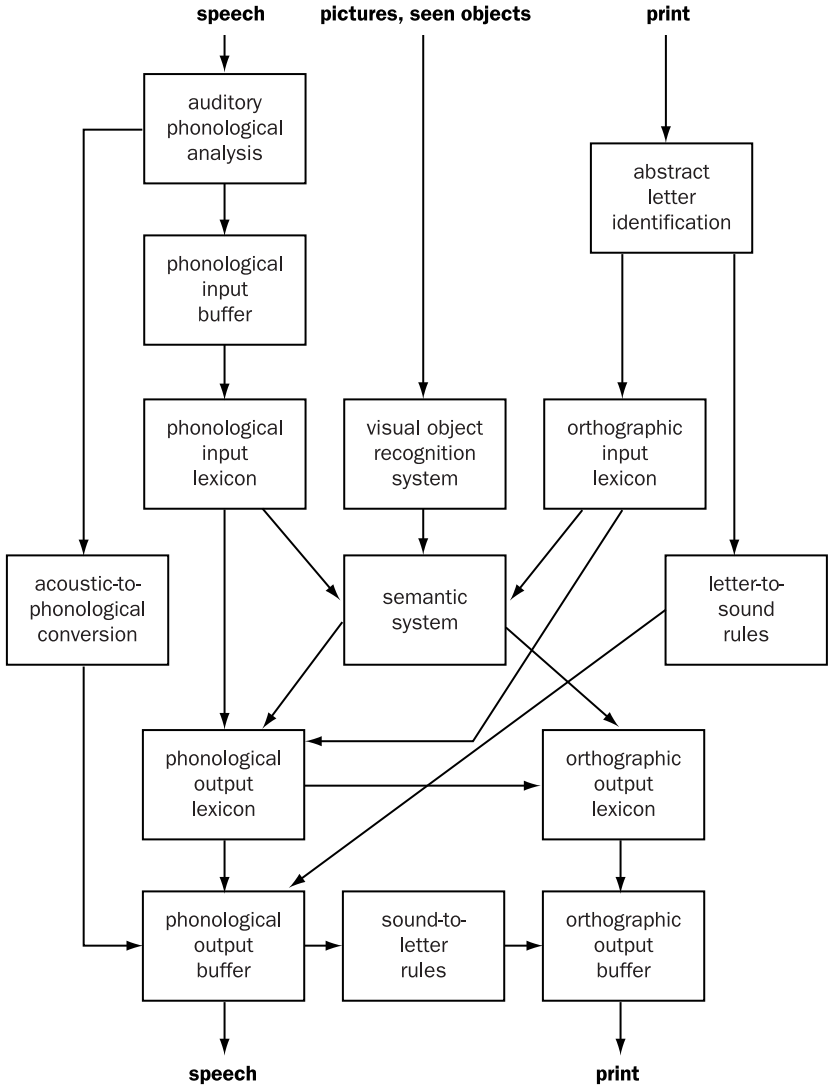

Figure 8.2 The *Psycholinguistic Assessment of Language Processing in Aphasia* version of the cognitive neuropsychological model for reading, writing, speaking and understanding single words from *PALPA: Psycholinguistic Assessment of Language Processing in Aphasia* by J. Kay, R. Lesser, and R. Coltheart. Hove, UK: Lawrence Erlbaum Associates, 1992. Reprinted with permission..

be interpreted as resulting from functional reorganisation or modification of intact cognitive functions (Kosslyn & Intriligator, 1992; Kosslyn & Van Kleek, 1990).

Finally, association and dissociation play an important role in cognitive neuropsychology, as they did in pre-cognitive neuropsychology. Associations concern the common appearance of symptoms, and the dissociation of a function occurs when it can be disturbed in isolation: that is, if a patient can be reliably shown to have a deficit in function X but a retained ability in function Y. A double dissociation, which goes back at least to Tueber (1955), occurs when two functions can fail independently of one another; when, for instance, we can observe the opposite to the above dissociation in another patient, who has impaired function Y, but intact function X. Such evidence is interpreted to suggest that function X is represented separately from function Y in the brain. Separate processing stages or processing modules can be implied using double dissociations. Cognitive neuropsychology subsequently developed into a cornerstone of modern cognitive neuroscience.

The logogen model

In contrast to classic neuropsychology, cognitive neuropsychological models are typically formulated without concern for brain anatomy, being centrally concerned with the nature of cognitive processes divorced from neurological processes. The **logogen model**, and its many variants and expansions, became the standard way of representing the processing of single words. The information processing metaphor has its origins in the way digital computers process information (Morton, 1968, 1970) and the first models were concerned with reading (Marshall & Newcombe, 1966, 1973) and other aspects of language soon followed. In subsequent years similar models were developed for other aspects of cognition, like face processing, limb apraxia and calculation, but these developments will not concern us. Figure 8.3a shows a very early version of the logogen model that was originally developed by **John Morton** (see Figure 8.1) in 1968. Figure 8.3b shows the post-1977 version. The logogen system

> was founded on the premise that the production of a single word response should be mediated by the same element irrespective of the origin of the information which led to the response. For this reason the results of visual analysis and auditory analysis of verbal material were fed into the same system where they interacted with contextual ('semantic') information from higher-order processes (variously called semantic system, cognitive system, etc).
>
> (Morton & Patterson, 1980: 91)

The 'cognitive system' depicted in the diagram is not part of the original logogen model itself (hence its cloud-like appearance), the logogen system itself contained no semantic information and had only two outputs, to the cognitive system or to the response buffer. Because of the first of these outputs, it was able to account for the semantic, 'paralexic', errors made by

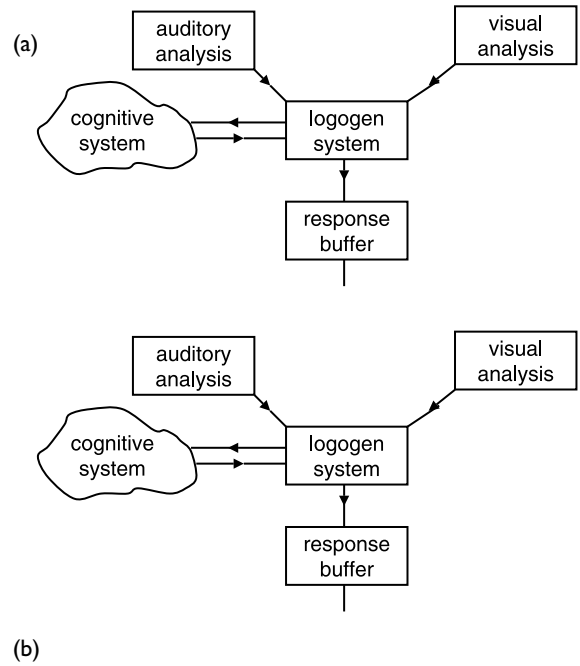

Figure 8.3 Logogen models: (a) 1977 and (b) post-1977 versions from Figure 4.1 and 4.2b in 'A new attempt at an interpretation, or, an attempt at a new interpretation', by J. Morton and K. Patterson. Chapter 4 in *Deep Dyslexia*, edited by M. Coltheart, K. Patterson, and J. C. Marshall. London: Routledge & Kegan Paul, 1980. Reprinted with permission.

individuals with deep dyslexia (e.g., reading *storm* as 'thunder'). In the 1977 version of the model, separate input and output logogens and a grapheme to phoneme conversion (GPC) mechanism were added to account for new evidence. The mechanism became necessary to deal with reading non-words aloud which are experienced visually for the first time. Later an acoustic-phonological mechanism was added to allow the repetition of auditorily presented non-words.

The more recent variant illustrated in Figure 8.2 is taken from the **Psycholinguistic Assessments of Language Processing in Aphasia (PALPA)** (Kay, Lesser, & Coltheart, 1992), a collection of tests designed to test the integrity of separate modules and routes, and is a much more detailed model. In this variant, the term 'logogen' has been replaced by **lexicon** and a semantic system is now included as a component of the model. This information processing metaphor lies at the centre of the cognitive neuropsychology enterprise as applied to language processing, and provides a characterisation of how cognitive representations are related one to another within modules. As noted above, it is more than superficially reminiscent of the diagrammatic models produced by Wernicke, Lichtheim, Kussmaul, and Charcot discussed

in previous chapters. Graves (1997), for instance, is not alone in drawing parallels between the aims and the diagrammatic representations of the cognitive neuropsychological model and the Wernicke–Lichtheim model. So cognitive neuropsychology saw a return to what Head (1926) referred to disparagingly as 'the diagram-makers', but, importantly, made no claims for localising function to brain structure, and indeed was concerned only with cognitive, rather than anatomical, architecture. The model deals with the processing of single words in reading, writing, naming, and repetition. There is a central semantic system, and separate lexicons that store input and output information, various buffers that store information for very short periods, acoustic to phonological and letter to sound (or grapheme to phoneme) conversion components and connecting routes between these various components. We will not describe these here in detail, but recommend Ellis and Young (1988) for an introduction.

The power of one: single case versus group studies

We have seen in previous chapters that aphasiology, and neuropsychology more generally, is predominantly founded on the study of individuals with brain damage, prime examples being Broca's original cases (for examples of classic cases in neuropsychology, see Code, Wallesch, Joanette, & Lecours, 1996, 2003), but the group study has an established role in experimental psychology. The advantage is that, through good design and statistical analyses, results from group studies can be related to the population from which the test sample is drawn, provided they are a truly representative sample. As mentioned above, many group studies were conducted in the 1960s in aphasiology where groups of participants with, for instance, Broca's, Wernicke's or conduction aphasia were compared. The assumption in such group studies is that significant differences in group means on some experimental tasks reflect real differences between the groups, and by implication, differences between impairment profiles in different groups provide some basis for clustering cognitive functions together and some basis for claiming neural representation for these clusters of functions. This logic rests on the assumption of the homogeneity of the examined groups. The notion of a syndrome can be variously defined, but an essential element is that a syndrome is established where more than one symptom is observed in an individual with all the symptoms assumed to have a single cause. It may be that a syndrome can be claimed where one or more 'cardinal' symptoms are observed but with other more optional symptoms also occurring. Thus Coltheart's (1980a) early discussion of deep dyslexia contained the idea that a cardinal and necessary symptom of the condition was the presence of semantic paralexias in reading aloud. However, many cognitive neuropsychologists adopted the view that progress in the investigation of the nature of cognition could be achieved best by careful examination of individuals with brain damage through a process of hypothesis testing using tests controlled for a range of controllable and

manipulatable variables. Cognitive neuropsychology embraced the traditional single case approach and a common central view for most cognitive neuropsychologists was that a theory-driven approach to the investigation of individuals is preferable to attempts to compare heterogeneous groups of patients categorised according to classical syndrome models.

The assumption of group homogeneity was strongly doubted, especially in early cognitive neuropsychology (Caramazza, 1984, 1986; Caramazza & Martin, 1983; Caramazza & McCloskey, 1988; Shallice, 1979). The view was that group means, or other indicators of central tendency, mask individual differences and that associations found within the syndromes show marked variability, and are not systematically attributable to components of a language processing model (Schwartz, 1984; Shallice, 1979).

However, there are opposing views within cognitive neuropsychology on the value of syndromes (Caramazza, 1984; Schwartz, 1984) and strong arguments for the retention of the syndrome have been advanced (Coltheart, 1980a; Shallice, 1979). Indeed, syndromes have been developed in cognitive neuropsychology since its earliest investigations, like **deep dyslexia**, **surface dyslexia**, **phonological dyslexia**, **deep dysphasia** and so on, although traditional syndromes were often atheoretical or based on flimsy theoretical foundations, whereas the contemporary dyslexias have been built up with reference to a systematic model of reading. In more recent years too there has been a return to group comparisons within cognitive neuropsychological approaches (Graham, Patterson, & Hodges, 2004; Hodges, Patterson, Oxbury, & Funnell, 1992; Jefferies, Crisp, & Lambon Ralph, 2006; Lambon Ralph, Moriarty, & Sage, 2002; Patterson et al., 2006).

So while the single case study has survived and a large number of experimental single case studies have been conducted, the field appears to be moving in the direction of combining single case series and group studies.

Further developments in cognitive neuropsychology

The information processing model is a 'serial' model where separate individual steps follow each other successively and the individual steps do not interact with each other. In recent years other models and paradigms have developed that have challenged the dual-route models of reading and writing. These are interactive models, which propose that language processing occurs in interactive and parallel networks that can be activated simultaneously and which act with one another. These models are, coincidentally, also called 'connectionist models', but are not to be confused with the connectionist models of classical aphasiology.

Developments in **connectionist** network modelling are still having a major impact on cognitive neuropsychology (Hinton & Shallice, 1991; McClelland & Rumelhart, 1988; Patterson, Seidenberg, & McClelland, 1989; see Harley, 1995 for details). These artificial **neural networks** use ways of processing information diametrically different from the orthodox, serial information-

processing method utilised so widely in the past as a basis for explanation in the cognitive sciences. Neural networks run on computers, but simulate fairly grossly certain features of biological neuronal organisation and are based on knowledge of the behaviour of real neurones. Conventional computation is serial and linear, whereas neural network models are nonlinear and parallel. Where serial computation is based on logic and syntax and systems of rules to manipulate symbols, connectionist networks represent knowledge as patterns of activation across 'neurones', or units, in the network. Like neurones, these units can be excitory or inhibitory and are arranged in connected layers. These layers are either input layers, output layers or so-called 'hidden' layers, and form a network of interconnected units. They have no in-built facility to process input as conventional computer programs do, but they learn through training using a process of associative learning and some include a sort of feedback called back-propagation. Values of the weights on units can be set or are initially set to random values, and determined through learning where actual values are compared to desired values and propagated back from the output layer during training to the input layer to adjust the excitory or inhibitory weights. Once trained, the network is ready to perform its task.

Brain function is nonlinear and it is suggested that linear information processing is unlikely to be an accurate model for cognitive processing (Kosslyn & Intriligator, 1992). The future of neurocognitive research therefore appears to require a continued interaction between this computer metaphor and brain damage, where models of cognitive processing can be developed within a nonlinear connectionist paradigm and tested with brain-damaged individuals. The cognitive neuropsychological model had a major impact on the assessment and treatment of aphasic impairments too.

The efficacy of aphasia treatment and the influence of cognitive neuropsychology

Attempts were made in the 1980s to design and carry out more closely controlled group studies of the efficacy of aphasia treatments, and with varying degrees of sophistication, a small range of randomised clinical trials (RCTs) were carried out in Europe and North America. Outcomes varied but most studies suffered from significant limitations, although their results had an impact on perceptions of the effectiveness of treatment (for relevant discussion, see Greener, Enderby, & Whurr, 1999; Greener, Enderby, Whurr, & Grant, 1998; Robey, 1998; Robey & Schultz, 1998; Wertz, 1995). In their systematic review Greener et al. (1998: 160) concluded that 'based on a systematic review of randomised trials of speech and language therapy for aphasia following stroke, it is not possible to reach a conclusion about the effectiveness of these treatments'. The problems of designing and carrying out large group studies that would produce unambiguous results had been clear earlier than this, not least the heterogeneous nature of the group samples (Howard,

1986; Pring, 1986), and in reaction many therapists turned to the developing methodology of cognitive neuropsychology for approaches to the treatment of impairments. The increasing desire to focus on the social consequences of aphasia outlined in Chapter 7 was a further reaction to the failure of RCTs to provide satisfactory outcomes.

Therapists were excited about the potential of the treatment approach promised by cognitive neuropsychology that was theoretically motivated, used carefully controlled single case methodology (therapists work predominantly with individuals after all) and had a model for detailed assessment of an individual's deficits and retained abilities. Early studies were conducted in Britain and probably the earliest is one by **Frances Hatfield** (1983) describing the treatment of three individuals with deep dysgraphia and one with surface dysgraphia using treatment inspired by the dual-route model of writing heard words. The general thrust towards the single case influenced treatment and clinical researchers and therapists working on aphasic impairments were quick to develop designs that would allow the unambiguous examination of the efficacy and efficiency of individually tailored treatments. Coltheart (1983) described basic single case designs that could test the success of treatments. Early studies were Patterson, Purell and Morton (1983), Byng and Coltheart (1986), and De Partz (1986). Other relevant studies are Coltheart and Byng (1989) (see Howard & Hatfield, 1987 and Edmundson & McIntosh, 1995, for a review).

Howard and Patterson (1990) made the case for cognitive neuropsychological treatment and described three broad strategies for therapy that logically flow from cognitive neuropsychological research. The first is to devise methods that can re-teach the missing information, missing rules or procedures based on a detailed hypothesis-testing approach to assessment; the second is to attempt to teach a different way to do the same task; and the third is to attempt to facilitate the use of impaired access routes. Promising results began to show that patient-specific and deficit-specific treatment based on the general approach could significantly improve performance in patients which could not be accounted for by spontaneous recovery or non-specific effects like attention or novelty. With the development of the PALPA resource (Kay et al., 1992), a collection of assessments designed to test the integrity of separate modules and routes of the model became available. These subtests are controlled for crucial psycholinguistic variables like word frequency, concreteness, imageability, spelling regularities and irregularities and length, and examine mainly single word processing in speech, writing, comprehension and reading.

Seeing the working brain: the rise of cognitive neuroscience

The closing twentieth century saw the emergence of a new interdisciplinary endeavour in **cognitive neuroscience**. This was a coming together of cognitive neuropsychology and the development of neuroimaging methods that allow

investigators to determine the location of brain damage, see the activity in areas that were undamaged, and visualise 'activation' in the healthy brain while individuals are completing cognitive tasks. Thus imaging can be **structural**, where it can be used to answer questions about localisation of damage, and it can be **functional**, where it seeks activation in the brain during cognitive processing. Early activation studies were primitive, but investigators began to realise that well developed models of cognition were required that could generate constrained and well-conceived questions. Currently the field seeks **convergence** (see Ellis & Young, 1988) between findings from studies and a significant issue for the field, especially in language, is the degree to which there is agreement between localisation data obtained from brain damaged participants and data obtained from healthy participants using functional neuroimaging.

Until the beginning of the 1970s, brain autopsy was the only reliable method to obtain information on the location of lesions (Damasio, 1989: 3). This changed with the development of methods that detect visual changes in brain tissue arising through damage in the living brain. However, the sodium amytal (Wada, 1949) method, still regarded as the most reliable method for determining lateralisation of function, was and still is widely used. The method was associated mainly with the Montreal Neurological Institute (Milner, Branch, & Rasmussen, 1968) and involves the injection of a fast-acting barbiturate like sodium amytal into the carotid artery in the neck on one side and then on the other on separate occasions. The barbiturate anaesthetises most of the hemisphere on the side of the injection for between five and ten minutes and allows the cognitive testing of the patient to determine which hemisphere is in control during various language and other cognitive tasks. The method involves risk and is used only when medically justified.

Brain imaging technology and experimental manipulation are developing rapidly and we can present only a brief introduction here. Perani and Cappa (1999) is recommended for recent technical and neuropsychological reviews. Computerised tomography emerged in the 1970s and is a structural method that provides information on the localisation of lesions. It was followed by other techniques (MRI, SPECT, PET) in the 1980s that allowed investigators to examine functional correlates of actual cognitive activity in the brain. **Computerised tomography** (CT) is based on radiological technology and can provide monotonic structural images of a brain taken at several different levels – typically eight to ten cross-sectional X-ray 'slices' are imaged so that different cortical and subcortical structures can be seen. Pathological processes (especially necrotic tissue) become visible as darker areas. So CT studies were able to correlate lesion sites and sizes with functional deficits seen in people with aphasia and other neurological conditions. Following the development of the CT scanner, a range of methods based on measuring blood flow to different regions of the brain were developed based on the observation that blood flow through brain tissue varies as a function of metabolism, which underlies all cortical function. Cognition requires metabolism, which

in turn requires oxygen and it is the blood that carries the oxygen. The higher the blood flow to a particular area during a specific task therefore, the more important is that area in the processing of that task. An early method developed in the late 1970s measured **regional cerebral bloodflow (rCBF)** on the cortical surface of the brain. It entailed the patient inhaling or being injected with a radioactive isotope of the inert gas *xenon* and the course of the isotope was tracked for about one minute using a gamma-ray camera. The information obtained is processed by a computer and the detail is represented as coloured images on a monitor. The method is limited by the fact that it was able to show activity only on the brain's surface.

Single Photon Emission Computer Tomography (SPECT) and **Positron Emission Tomography (PET)** developed in the 1960s and 1980s respectively; they use the injection or inhalation of radioactively marked tracers that travel through the blood and are able to cross the blood–brain barrier. These markers emit positrons that can be captured by a scanner. SPECT and PET allow the detection of increases (hyperperfusion) or decreases (hypoperfusion) in blood flow to specific parts of the brain. They provide more than structural information, and can show the behaviour of blood flow in three-dimensional detail so that a whole brain and the behaviour of its blood flow can be imaged and measured. SPECT is particularly useful in investigations of cerebrovascular disease as it can show areas of hypoperfusion (reduced blood flow) that are larger than the area of structural damage shown in CT scanning, indicating that reductions in blood flow in undamaged areas may be the result of diaschisis (see Chapter 5), a 'deactivation' caused by a distant lesion (Perani & Cappa, 1999). It can measure the regional cerebral blood flow, the regional cerebral blood volume, and various metabolic values (glucose consumption, oxygen consumption). The method has made a significant contribution to the detection of damaged brain that CT cannot show, as well as spearheading the use of **functional imaging**, that allows an experimenter to examine areas of cortical activation. PET can detect the interaction of positrons with electrons creating two gamma photons travelling in opposite directions allowing the measurement of local tracer concentrations and representing these as images of activity (Perani & Cappa, 1999).

Magnetic resonance imaging (MRI) is the most recently introduced imaging method and can provide a high definition structural image and its functional variant, **functional MRI (fMRI)**, can image brain activation associated with cognitive function. It exploits the way oxygen carried by the haemoglobin in the blood affects the magnetic properties of the blood. Changes in blood flow can be detected and 'functional' activity can be measured. MRI uses the magnetic characteristics of atomic nuclei in order to measure the electromagnetic waves that escape from the body when it is briefly brought into a strong magnetic field. Atomic nuclei spin on their axes and have a positive electronic charge and any spinning, charged, particle will act as a magnet with 'north' and 'south' poles located on the axis of spin, pointing randomly. When placed in a powerful magnetic field the axes line up with the field with

the 'north' poles all pointing 'south'. This creates an average vector of magnetisation and a radiofrequency (RF) pulse is broadcast towards the object causing the axes to tilt with respect to the magnetic field, producing a 'resonance' of the magnetisation vector with a cycle of 20–300 ms. fMRI is safer than PET as it entails no injection of nuclear substances, and it possesses better spatial (visual) resolution than PET providing clearer and more detailed images. Where PET allows only a short period where data can be collected concerning activation (as long as it takes for the nuclear carrier to flush out from the brain – typically about 90 seconds), MRI has no such limitations. Modern fMRI includes **Echo-Planar Imaging (EPI)**, an ultra-fast image acquisition technique. A disadvantage is that MRI produces a loud and unpleasant noise and does not allow metallic objects, including recording devices, into the area of the scanner, limiting investigations of speech processing.

CT and MRI are more appropriate for examining the localisation of damage, and we are no longer dependent on postmortem examinations. With PET and SPECT the effects of specific brain lesions on other brain regions can be examined by measuring the behaviour of blood in undamaged regions, for instance glucose consumption or rCBF. PET and fMRI are used in experiments designed to examine the activation of different parts of the brain during different cognitive tasks.

In the 1970s and 1980s many investigations were carried out using CT scanning (Naeser & Palumbo, 1995). Attempts to determine the lesion locations of the classic syndromes partly resulted in extensions of the aphasia-causing brain regions. For instance, the 'anatomical definition of the classical "centers" has also changed. The "anterior language center" has suffered considerable expansion. [. . .] The same applies to the posterior language region' (Damasio, 1989: 43).

Although there were interesting confirmations for lesions assumed or verified by autopsies, the limit of anatomically orientated aphasiology was soon revealed. Some studies suggest that prediction of a lesion site from aphasia type has a success rate of about 83 per cent using CT scanning (Basso et al., 1985), and Poeck, De Bleser, and Von Keyserlingk (1984: 85) were led to the conclusion that '[I]ndividual consideration of single patients in groups shows that there is by no means a one-to-one relationship between specific syndromes and particular regions within the language area.' Furthermore, 'one has recognized more negative cases than expected by the classic doctrine on the differential localisation of aphasic syndromes within the left hemisphere' (Poeck et al., 1984: 88).

Studies with SPECT and PET show that brain regions that appear normal in CT images can display altered metabolism (Kuhl et al., 1980). The 'functional' lesion visible in PET is often shown in the immediate vicinity of an actual lesion (ischemic penumbra). It is therefore possible that 'a function in undamaged tissue can be aberrant, which is possibly responsible for individual aspects of an aphasic language disorder' (Metter, 1995: 199); rCBF

has also been observed to increase in the region analogous to Broca's area in the right hemisphere (Metter et al., 1984). Unsurprisingly, a leading PET expert, Jeffrey Metter, summarised the results of PET and SPECT studies in 1995 as follows:

> The studies reviewed suggest that language requires the interaction of numbers of highly integrated systems of the brain. This interaction involves both hemispheres as well as cortical and subcortical structures. [. . .] The excitement from SPECT, PET, and xenon studies is in the observation of the importance of brain regions other than the classical language cortex in normal language and aphasia.
>
> (Metter, 1995: 206–207)

In sum, imaging studies clearly show that the simple correlation of aphasic syndromes with anatomical lesions is insufficient. A more dynamic notion began to replace the static assumptions: 'The units are not mere independent centers linked by cable pathways. They are, rather, richly interconnected functional regions which form overlapping networks' (Damasio, 1989: 43–44).

More recently methods of investigation that exploit the electrical activity of the brain have developed. **Electroencephalography (EEG)** is not new, but in combination with **magnetoencephalography (MEG)**, a powerful method of investigating brain activity has developed in recent years. Neurones produce tiny amounts of electrical activity (action potentials) when they fire, caused by the transmission of ions from the axon to the cell. **Event-related potentials (ERPs)** are changes in EEG in response to sensory stimuli (e.g., the word 'car' or a picture of a car) detected with electrodes on the scalp. The tiny signals are averaged over hundreds of measurements to provide information about the electrical activity over regions of the brain. MEG takes advantage of the fact that neuronal electrical currents generate tiny magnetic fields that can be identified with very sensitive detectors – **Superconducting Quantum Interference Devices (SQUIDs)**. MEG can detect the source of magnetic activity during the cognitive tasks devised by experimenters. MEG has excellent temporal resolution, 1/1000 of a second (e.g., deciding that '4' is less than '5' takes 200ms) although its spatial resolution is low. Currently investigators are conducting experiments that combine the good spatial resolution of MRI with the good temporal resolution of MEG to provide information on *where* cognitive processing is taking place and *when* it is taking place. The promise is that this will provide information that will allow investigators to build valid and reliable neural networks underlying language and related aspects of cognition.

Currently there is disagreement about what neuroimaging can tell us about cognition in general and language in particular. Poeppel (1996) asked, 'What has functional neuroimaging told us about the mind so far?' and concluded that it has told us nothing in the domain of language. Many cognitive psychologists believe that we still need a more complete theory of cognition before

we can begin to reliably interpret the images that we see, and we do not have that yet.

Harley (2004: 10) summarises the issue for many cognitive psychologists: 'There is a level of psychological theorising – the cognitive level – that can only be studied at this level, and information from lower levels will tell us nothing about what happens at the cognitive level.' Coltheart (2006) has asked if any imaging studies have successfully tested any psychological theories or helped adjudicate between competing psychological theories. His conclusion is in the negative. For further discussion of these issues see Coltheart's (2006) 'What has functional neuroimaging told us about the mind (so far)?' and the accompanying replies to his question.

Other developments in cognition

While cognitive neuropsychology was the main theoretical thrust in the final third of the twentieth century, there have been developments and advancements in a variety of areas that either have been utilised in aphasia research or have used aphasia to test and develop better models. These include the sentence production model developed by Garrett (1975, 1980, 1984, 1993), which aphasiologists have used to examine sentence level impairments like agrammatism, and the influential psycholinguistic model developed by Levelt and colleagues (Levelt, 1989, 1993). Both of these models account for syntax and sentence processing and their foundations are data from healthy speakers. While both are psycholinguistic models, Garrett's was built on data from slips of the tongue research and Levelt's on reaction time research – very different from the data obtained from the performance of people with brain damage.

In addition, the notion of **working memory** has been identified and developed (for an introduction, see Baddeley, 1986; for a collection of papers on impairments to auditory-verbal short-term memory, see Vallar & Shallice, 1990). Working memory deals with the short-term storage of a very limited amount of information for a very short time (around 4 seconds or less according to many). The model includes a phonological store, an articulatory loop (sometimes called a phonological loop) for the rehearsal of material in inner speech, and a central executive. A separate visual scratchpad or sketchpad is conceived to deal with visual information. Language processing depends on properly functioning auditory-verbal working memory, and a number of aphasic impairments have been investigated from this perspective. For instance, judging whether two words rhyme or not or making a judgement on syntactic comprehension tasks like deciding who kicked who in a sentence like 'The boy who the girl kicked was angry', depends for successful processing on being able to hold the two words or the sentence in working memory. For instance, impairment to auditory-verbal working memory has been commonly suggested to be the underlying deficit for problems with reversible sentences. Sentence comprehension is discussed further in the sec-

tion on the history of Broca's syndrome later in Chapter 9. For discussion of these aspects, see Caramazza, Basili, Koller, and Berndt (1981), Romani (1994), Saffran and Marin (1975), and Vallar and Baddeley (1984).

Summary

Cognitive neuropsychology represented a coming together of cognitive psychology, psycholinguistics and neuropsychology and for many replaced anatomically specified models of brain and language in favour of an explicitly cognitive model. The paradigm dismissed classic and neoclassic aphasia classifications on theoretical grounds, although it developed its own classifications and types, albeit somewhat more refined than the traditional ones, and it spearheaded a return to single case investigation. The paradigm and its methodology influenced developments in treatment for aphasic impairments, initially in the English-speaking world. Clinical researchers conducted randomised control trials of treatment with large groups of aphasic participants, but the significant limitations to interpretation of the outcomes of such trials were identified.

Sophisticated imaging methods developed and cognitive neuroscience emerged as one of the most influential fields of science. This meeting of neuroscience and cognitive psychology thrived as new methods for imaging the brain were developed. The simple idea that clearly defined aphasic syndromes develop from lesion-specific, anatomically determined brain regions became untenable. Brain regions outside the classic cortical 'language centres' in the left brain were identified as being crucially involved in speech and language processing, but despite enormous amounts of research, many of the old questions about the representation of language in the brain remained. Neuroscience promises to shed light, but at the end of the century, the illumination appears to mainly expose more clearly the extent of our continuing ignorance.

9 Broca's aphasia and Broca's area

The journey from 1861 to 2005

We have attempted in this book to highlight the development of the main features of aphasia and its forms and variations and the classifications and typologies that have emerged from various theoretical standpoints over the centuries. This is a rich and varied history and we could have gone into further detail on the development of ideas and research on any number of topics. But such detail would be inappropriate in an introductory text like this one. However, we can perhaps provide an illustration of the historical development of the forms of aphasia by sketching the evolution of the topic that has attracted the most attention in aphasiology – Broca's aphasia and Broca's area.

Broca's aphasia

The sands beneath Broca's area and Broca's aphasia have been shifting ever since 1861. We saw in Chapter 3 that Broca's original description identified a speech production problem he called *aphemia* with a non-lexical speech automatism (*tan tan*), with unimpaired comprehension and gestural abilities caused by a lesion to the third frontal convolution. While Leborgne's lesion was on the left, Broca drew no attention to that fact and did not recognise that aphasia tended to be associated with a left lesion until 1865. From this beginning the syndrome and the area of the brain named after Broca have had a chequered history. However, as we move into the twenty-first century, both syndrome and area appear to be very much alive, though the range of impairments subsumed under the syndrome and the range of cognitive functions that the area serves have widened enormously.

First, the syndrome of Broca's aphasia. We cannot do justice to the sheer amount of work that has appeared over the years on the syndrome named after Broca and we will track only the main highlights. In the twentieth century Broca's aphasia had come to mean a syndrome that can include an apraxia of speech (or aphemia), one or more speech automatisms, agrammatism, and syntactic comprehension impairments, together with reading (deep dyslexia and/or phonological dyslexia) and writing (deep dysgraphia) problems. Severity can vary from virtual muteness to relatively mild articulation and

syntactic processing difficulties. We consider mainly developments in speech production, speech automatisms and syntactic processing.

As we have seen (Chapter 3), later development saw the decision taken by most in the field to adopt the term *aphasia* introduced by Trousseau in 1864 to refer to the language disorder, although many retained the term *aphemia* for the speech production disorder central to Broca's aphasia. The use of the term **phonetic disintegration** to describe the speech of those with Broca's aphasia is associated with the French and French-Canadian tradition, particularly **Théophile Alajouanine** (1890–1980), and was introduced as the title of the book published by Alajouanine's research group in 1939, *Le Syndrome de désintégration phonétique dans l'aphasie* (Alajouanine, Ombredane, & Durand, 1939). This book, the result of studies conducted by Alajouanine's group in France before the Second World War at the *Hospice de Bicêtre*, focused mainly on the phonetic description of Broca's aphasic speech (Lecours & Signoret, 1981).

The American speech pathologist **Frederic L. Darley** (1918–1999; see Figure 9.1), working at the Mayo Clinic in Minnesota, is mainly remembered for his attempts at redefining **apraxia of speech** (AoS) (Darley, 1968; Rosenbek, 2001), a term he coined (he also used the term 'oral verbal apraxia' in earlier writing [Darley, 1967]), and his work on laying the foundations for research

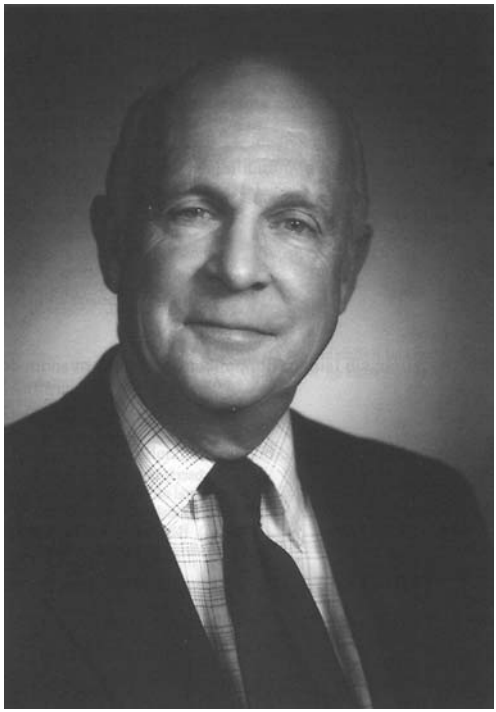

Figure 9.1 Frederic Darley.

into the efficacy of language rehabilitation in aphasia (Wertz & Irwin, 2001). But apraxia of speech was probably originally described by Liepmann (1900, 1913), who saw it as a variant of limb-kinetic apraxia (Liepmann, 1913: 56) stating that 'the word limb here, refers to the tongue, palate, and oral mechanism' and that 'speech is a parasite making use of a preformed sensory-motor mechanism which is also involved in (*other*) [Liepmann's emphasis] functions, particularly that of eating, it likewise shares in any impairment sustained by this same mechanism'.

Before Darley a bewildering array of terms had been used to describe the disorder (for a review of this terminological history, see Johns and LaPointe, 1976; Lebrun, 1989), including aphemia, anarthria, cortical anarthria, phonetic disintegration, verbal a(dys)praxia, motor aphasia. These terms still survive and are being sustained by different professional and national traditions. Anarthria and aphemia are still used by medical writers and researchers, and as mentioned above, phonetic disintegration is still used by French authors. For Darley AoS was the result of a lesion to Broca's area (Rosenbek, 2001) and in 1975, Darley, Aronson, and Brown (1975: 255) defined AoS as 'an articulatory disorder resulting from impairment due to brain damage of the capacity to program the positioning of speech musculature for the volitional production of phonemes and the sequencing of muscle movements'. This definition entails two elements – positioning and sequencing of articulatory muscle movements – and implies that the apraxic symptoms to be observed are incorrect positioning of articulators resulting in incorrect speech sound productions and speech sounds in inappropriate order. We can note that it is phones at the phonetic level that are produced by muscular activity, and not **phonemes**, which are abstract phonological entities. The case for AoS as a disorder of phonetic planning and/or programming rather than as a central linguistic disorder arising from some impairment at the phonological level, gained support from a range of clinical and laboratory sources in North America and Europe, but particularly in the research produced in North American speech pathology.

For Darley and most of his students, it was inappropriate to use a range of terms to describe speech production disorders that were not aphasic and were not dysarthric, when everyone was describing the *same* non-dysarthric, non-aphasic speech disorder. The term apraxia of speech is utilised these days by the majority of researchers and speech and language clinicians, although verbal or articulatory a(dys)praxia are used synonymously.

Discussion of the nature of AoS developed over the latter half of the twentieth century, with some consensus emerging. Rosenbek, Kent, and LaPointe (1984) listed the following main features: errors in articulation increase as the complexity of the motor task increases, with vowels easier than consonants and single consonants easier than clusters of consonants; errors occur more often on low frequency than on higher frequency consonants; errors increase with word length and occur more on imitation than in spontaneous speech; errors occur less often on automatic than on more

propositional speech. A striking feature of AoS, developed by Hughlings Jackson, with acknowledgments to earlier work by Baillarger (1865), is a disassociation of voluntary and involuntary speech actions. The speaker is typically able to carry out involuntary and automatic actions which are relatively preserved. Thus a patient with an apraxia which affects buccofacial-oral mechanisms may be unable to lick his or her lips to command or even to imitation, but will automatically lick their lips while drinking. This dissociation implies that voluntary and involuntary control over the same movement patterns, carried out by the same muscle groups, are initiated and/or organised separately.

In the 1960s the dimension of **fluency** was firmly established by Geschwind and Howes (Howes, 1964; Howes and Geschwind, 1964; see also Benson, 1967) to characterise the significant differences observed between anterior and posterior aphasias, although the terms 'fluent' and 'nonfluent' were coined originally by Wernicke (1874; see Benson & Ardila, 1996; Poeck, 1989) and were clearly described by Hughlings Jackson who divided his patients into two groups – those who were speechless, or nearly speechless, and those who spoke a great deal but made many errors. **Frank Benson** (1928–1996; see Figure 9.2) gained his early inspiration from Geschwind and was Chair of the Aphasia Research section at the Boston VA Hospital and Vice-Chair of

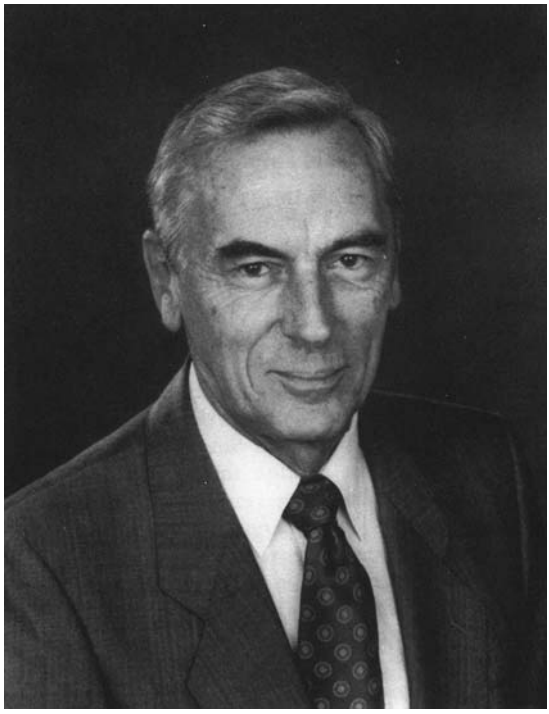

Figure 9.2 D. Frank Benson.

Neurology at Boston School of Medicine until 1979 (Cummings, 1999). He made many contributions to aphasiology and was responsible for the earliest work utilising the emerging brain scan techniques in aphasia (Mega, 1999). An important contribution was his radioisotope brain scanning study of the features of aphasic fluency and nonfluency (Benson, 1967), pointing out the localising deficiencies of the Weisenburg and McBride expressive–receptive dichotomy, and establishing a *posterior* (fluent)–*anterior* (nonfluent) dichotomy with localising implications in its stead. The parameters of speech production that characterised fluency–nonfluency that the work of Geschwind, Howe, and Benson had identified included the rate of utterance, phrase length, articulatory agility, speech effort, verbal stereotypes (speech automatisms). These features appeared to distinguish fluent from nonfluent aphasia, and a subsequent study by Kerschensteiner, Poeck, and Brunner (1972) confirmed that aphasic speakers could be grouped into fluent and nonfluent. It was these features that are mainly used to distinguish between the classic anterior–posterior syndromes on the Rating Scale Profile of Speech Characteristics of the Boston Aphasia Exam, rather than scores on the subtests of the battery. However, it is difficult to separate the 'phonemic paraphasic' errors arising in fluent posterior aphasia from the speech errors observed in AoS. MacNeilage (1982) suggested that two separate processes may underlie the finding that high-frequency unmarked consonants replace low-frequency marked ones in the speech errors of aphasic speakers, irrespective of the presence of AoS. He suggested that it is motorically difficult consonants that are substituted by nonfluent speakers with AoS and high-frequency consonants that are often favoured intrusions by fluent speakers with paraphasia.

In the latter half of the twentieth century, speech motor theorists working on AoS characterised the problem as one of speech programming, speech planning or both. Darley et al. (1975) saw the problem as a motor speech programming problem in their three-stage model, a position agreed by Clark and Robin (1998). Van der Merwe's (1997) model of sensorimotor control involving linguistic-symbolic planning, both motor planning and programming and execution, where planning entails the retrieval of the invariant core motor plan that specifies spatial and temporal parameters of individual speech phones, and programming specifies muscle tone, resistance, and force. McNeil and colleagues (McNeil, Robin, & Schmidt, 1997; McNeil, Doyle, & Wambaugh, 2000) have developed a more circumscribed position where the diagnosis of pure AoS is restricted to the four kernel characteristics of sound distortions, prolonged durations within segments, prolonged inter-segment durations and disturbed prosody arising at the planning stage of speech.

Leborgne's *tan tan* is often cited as the first example in the literature of an aphasic speech automatism, another optional symptom of Broca's aphasia, but Lebrun (1986) cites a patient with the expletive automatism '*Sacre nom de Dieu*' described by Auburtin just one week before Broca's case at a meeting of the same French Anthropological Society, a case also cited by Broca (1861b) in his article '*Remarques sur le siège de la faculté du langage articulé,*

suivies d'une observation d'aphémie (perte de la parole)'. Indeed, Broca appears to be saying that Leborgne also occasionally produced the expletive '*Sacre nom de Dieu*' when he became outraged. We saw in Chapter 3 that it was Hughlings Jackson's (1874) original observations of aphasic speech automatisms in the later nineteenth century that led him to propose the idea of **propositionality** in language. Jackson's term for these utterances was *recurring* (sometimes called *recurrent*) *utterances*; the term *speech automatism* was introduced by Huber, Poeck, and Weniger (1982). Non-lexical automatisms are utterances like *tan tan* or /tu tu tu/, /bi bi/, /di di/ (examples from Code, 1982a). These recurring utterances contrast with a lexical speech automatism, examples being *Sacre nom de Dieu* above, *I can't, I can talk, I can try, I want, bloody hell* (latter examples from Code, 1982a). Both forms of speech automatism can occur in Broca's aphasia, although rarely are they mixed. The non-lexical form is associated with more severely aphasic speakers with Broca's aphasia and, as with Leborgne, the patient may have little other speech. These non-lexical forms are made up of high frequency phones from the speaker's language and are invariably repeated, often with relatively intact prosody, so that the utterance is produced fluently. The lexical form is made up of a limited range of high frequency words and phrases, made up of 'modals' and auxillaries like *I want . . . I can't*. Expletives and swear words are particularly common too. Alajouanine (1956) completed a study of 317 cases of aphasia of whom 30 had a speech automatism (9.4 per cent). He considered an impairment of awareness to be a significant factor in patients with speech automatisms. He suggested that those who do make recovery go through four stages where attempts to check the utterance signals the re-establishment of awareness and the last of which is where agrammatic but propositional speech emerges. It was Alajouanine who identified the two basic (lexical and non-lexical) types and that nearly all those with the non-lexical type had an apraxia of speech, although the utterances themselves are produced with no sign of apraxia. In the 1980/90s a series of studies were done on speech automatisms, mainly in Germany (Blanken, 1991; Blunk, De Bleser, Willmes, & Zeumer, 1981; Brunner et al., 1982; Code, 1982a, 1982b, 1987, 1994b; Haas, Blanken, Mezger, & Wallesch, 1988), which examined the anatomical location of lesions causing speech automatisms and developed neurolinguistic and cognitive models. Models incorporating the notion of an articulatory buffer developed, which could be the locus of continued production of the utterance and could be responsible for the failure of the speaker to move on to a new utterance (Blanken, 1991; Code, 1994b).

We discussed the early progress in investigations of impairments in the use of grammar in Chapters 4 and 5, noting that it was Kussmaul (1877) who introduced the term agrammatism and Kleist (1934) who developed further the idea of a specific impairment of syntax. These days we use the term agrammatism to describe a variety of impairments apparently to syntactic processing where (in English at least) there may be a paucity or absence of **function words** in contrast to content words, omission of auxiliary verbs,

impaired inflection of verbs, nominalisation of verbs, impaired theta-role assignment from semantics and mapping from semantics to syntax and tense marking (for review see Caplan, 1987; Grodzinsky, 1993; Howard, 1985; Lorch, 1989). It is characterised by shortened sentence length, simple syntactic structures, problems with function words and morpho-syntactic elements but with relatively intact use of content words.

While agrammatism in now seen as part of the Broca's syndrome, the model developed by Wernicke and reintroduced by Geschwind was essentially limited to single words, and impairments in sentence processing were not readily accommodated within it. In Chapter 5 we saw that in his earlier work Pick (1902: 82) proposed that the '*language disorder referred to as agrammatism [. . .] originates from a focal affection of the language area localised in the left temporal lobe*'.

Bonhoeffer (1902: 222–223) described two cases that suggest the possible frontal localisation of agrammatism, close to Broca's area, and his opinion was that 'the motoric language centre must be looked at as the "seat" of these grammatical concepts, if one wants to understand the disorder.' The idea that Broca's area was the centre for grammar was supported in cases where 'a disorder of morphological word structure and sentence construction' where found. Pick (1913: 254) later described Bonhoeffer's cases as 'pseudo-agrammatism' and tried to show 'that these are not about real agrammatism'.

Heilbronner viewed agrammatism as 'not a secondary result of the aggravation of the motor act of speech, but a primary deficit phenomenon' (Heilbronner, 1906: 683), which could be observed in writing and sentence completion. The localisation of agrammatism to Broca's area became plausible to Heilbronner (1906: 683) because agrammatism could occur without comprehension deficit: 'considerable degrees of agrammatism are compatible with hardly damaged, maybe entirely undamaged, comprehension of small parts of sentences and hence of connected speech.' Salomon (1914) came to the contrasting conclusion that agrammatism was a phenomenon not only of expressive speech, but also of comprehension, a theme that was to be taken up in the later twentieth century, and discussed below. Salomon proposed a disorder of 'inner' speech as well as of the grammatical system and described a frontal and temporal type. In Chapter 6 we described how agrammatism emerges as a symptom of Luria's efferent motor aphasia: 'In later stages of recovery, the well known clinical syndromes of "telegram style" may appear' (Luria, 1964: 155).

Linguistic explanations for agrammatism have been numerous (for at least eight different theoretical viewpoints, see Kean, 1985) and by the 1980s the copious range included syntactic, morphological and phonological theories, employing an assortment of theoretical models. Syntactic descriptions have always been of particular interest to linguists because syntax holds a central position in linguistics and the development of linguistic models saw a number of attempts to characterise agrammatic language (production and comprehension) in terms of different theoretical models in the latter part of the

twentieth century. Kean (1977, 1978), for instance, proposed that agrammatism in language production can be understood by summarising those elements which remain intact in agrammatism (nouns, verbs, adjectives) and those elements that are disturbed using Chomsky and Halle's (1968) generative phonology theory which differentiates between 'phonological' and 'clitic' elements, where the first group consists of autosemantic, stressed words and the second group of synsemantic, unstressed clitics and endings (inflectional affixes). On this 'phonological' hypothesis, agrammatic speakers reduce their sentence structures to stressed phonological words. The unstressed clitics are mostly skipped. In similar fashion, but utilising different Chomskian models (Chomsky, 1981, *Lectures on Government and Binding*), Grodzinsky (1984) attempted to characterise agrammatism structurally. The structure in this model has two word classes: main word classes, which are lexically specified, and others, which are underspecified. The hypothesis is that agrammatic language can be represented exactly at the syntactic level and that the form it takes can be predicted in its appearance. Agrammatic speakers would be able to process lexically specified elements but not the others.

These approaches were subsequently modified several times partly because the underlying theory changed, in part because it became clear that the theory is incompatible with agrammatic data. Beside the approaches of Kean and Grodzinsky, there was a series of studies and hypotheses on agrammatism inspired by theoretical linguistics, which cannot all be mentioned here (for summaries see Fromkin, 1995; Kolk, 1998).

While linguistic theory and linguistic aphasiology in the 1960s and 1970s were dominated by studies in the English language, it became apparent that this provided only part of the story, because there is a desire to develop general theories that are, in principle, universal and applicable to all human languages. However, it has been pointed out (e.g., Howard, 1985; Lorch, 1989) that the early case descriptions were in German and by the 1930s there were few descriptions in English. German (and French) are highly inflected languages, having a much larger number of grammatical word endings than English, and agrammatism is therefore more obvious in speakers of highly inflected languages. There was an increase in **cross-linguistic** studies of agrammatism in the 1990s (Menn et al., 1996a; Menn et al., 1996b), a significant example was the **CLAS study** (Cross-Linguistic Aphasia Study) of agrammatism, coordinated by Lise Menn and Loraine Obler (Menn & Obler, 1990), where comparable agrammatic samples from fourteen languages were collected and analysed in order to be able to differentiate between language-specific and general aspects of agrammatism. The results show a rather complicated picture of agrammatism, which eludes any simple linguistic characterisation by current theoretical models.

However, there were also contributions to developments in agrammatism originating from psycholinguistics and cognitive neuropsychology (Whitaker, 1997). One of these is **adaptation theory** (Kolk, 1998; Kolk et al., 1985; Kolk, Heling, & Keyser, 1990), which refers back to the work of Isserlin and

Hughlings Jackson, and postulates that the agrammatic surface structure does not directly reflect the underlying deficit, but is the result of adaptation of the system. Kolk (2000) compared agrammatic output with the that of the developing speech of 2- to 3-year-old boys and found significant similarities in the elliptic speech of the agrammatic speakers and developmental ellipsis, suggesting, rather than a regression in agrammatism to developmental patterns, the operation of a choice by agrammatic speakers for normal elliptical speech that Kolk hypothesised functions to prevent computational overload.

The motor element to agrammatism has been considered central to the disorder, either directly or implicitly, by a number of theories and in the 1970s, Lenneberg (1973) and Goodglass (Goodglass, 1962; Goodglass, Fodor, & Schulhoff, 1967) developed motor theories of the disorder. Lenneberg's (1973) view was that agrammatic speakers employed an 'economy of effort', where the speaker's articulatory system was so impaired that their options were limited to only producing the important information carrying elements, like content words. Goodglass developed a stress-saliency hypothesis for what he called *motor agrammatism* (Goodglass, 1962), based on the experimental observation that the words omitted by speakers with Broca's aphasia are the unstressed function words occurring in sentence initial positions. On this hypothesis Broca's aphasic speakers have particular difficulties with the initial mobilisation of the speech production system.

Agrammatism continues to enjoy the attention of many linguists and psycholinguists and theoretical discussion and research is still hotly pursued. At the close of the twentieth century agrammatism was viewed by most as a non-unitary syntactic disorder, and a range of alternative underlying impairments have been posited, including verb and other lexical access deficits, theta-role assignment impairments, systemic adaptation to impaired mechanisms, impaired working memory, among others (for a review of the contenders, see Lorch, 1989). But an appreciation began to emerge that different forms of agrammatism can evolve with recovery and time, changing the pattern of syntactic deficit with recovery from severe to milder forms (e.g., Guasti & Luzzatti, 2002) with features more likely reflecting systemic adaptation and compensation at more chronic stages.

Despite what must have been a rudimentary assessment, Broca (1861a) was clear that Leborgne understood almost everything said to him and so was established the notion that comprehension was intact in Broca's aphasia. However, the idea that agrammatic speakers could have sentence comprehension problems was originally proposed by Salomon (1914) and later by Isserlin (1922). Contemporary views emerged in the 1970s with a seminal study by Caramazza and Zurif (1976) who showed that agrammatic speakers can indeed have problems in the comprehension of syntax (for a critique, see Caplan, 2003). They examined the sentence–picture matching abilities in agrammatic speakers and found that there were conditions where they could and conditions where they could not match the sentences to the pictures. They could match sentences to pictures where the *thematic roles* in the sentence

(identifying the agent or actor and the recipient of the action) could be inferred from the word meaning and real world knowledge and where the sentences were syntactically simple, but they could not match them when the sentences were syntactically complex and the thematic roles in the sentences could not be inferred from word meaning and real world knowledge.

The participants could match simple active sentences like 'The boy is eating a red apple' and simple passive ones like 'The apple the boy is eating is red', but not semantically reversible sentences like 'The girl the boy is chasing is tall.' The authors concluded that the patients had lost the ability to assign the syntactic structure of a sentence and determine thematic roles by integrating the lexical meanings into the structure, but their ability to interpret the sentences and assign thematic roles on the basis of the likely interactions of the words in the real world was intact. The explanation for the deficit was essentially an impairment in syntactic parsing. The study spurred many others into the sentence comprehension of aphasic participants (for a review, see Caplan, 2003). Linebarger, Schwartz, and Saffran (1983a, 1983b) demonstrated that four agrammatic speakers could make successful grammaticality judgements about many sentence types, suggesting that they had no impairment of parsing, but the deficit was a 'mapping' deficit, and impairment in using intact syntactic structures to determine aspects of meaning.

The parsing versus mapping debate continues into the twenty-first century and some researchers into syntactic comprehension in aphasia looked to auditory-verbal or phonological **working memory** (see Baddeley, 1986) for support. Working memory deals with the temporary storage of a limited amount of information and is considered to be necessary for the manipulation of that information in a short-term cognitive space (see Chapter 8 for further discussion). Making a judgement on syntactic sentence matching tasks like those above depends for successful processing on being able to hold the sentence in working memory in order to make the necessary manipulations. Impairment to phonological working memory has been suggested to be the underlying deficit for problems particularly with reversible sentences in a number of reports (Romani, 1994; Saffran & Marin, 1975; Vallar & Baddeley, 1984), although a review by Caplan and Waters (1990) found little or no good evidence to support the view (see Romani, 1994, for discussion).

In an extensive review of the available literature on the symptomology of Broca's aphasia, Berndt and Caramazza (1980) attempted another redefinition, and conclude that the nonfluent output characteristic of the syndrome and the problems with sentence comprehension are accounted for in terms of impairment to two separate components, an impairment to the articulatory mechanism and an impairment to the syntactic parsing component. They state,

> Broca's aphasia is the result of a combination of two primary deficits and the operation of compensatory mechanisms that are employed in an attempt to establish communication despite existing deficits. The two

> primary problems involve the syntactic parsing system with resulting agrammatism. [. . .] The second primary deficit is an often severe impairment of the physiological mechanism responsible for the articulation of speech. These two deficits are theoretically and practically separate and, in principle, need not occur together.
>
> (Berndt & Caramazza, 1980: 271)

The syndrome of Broca's aphasia has come a long way since 1861, and on the way has inspired theory and experiment way beyond the original concerns of nineteenth-century aphasiologists. Whether the commonly occurring impairments that define Broca's aphasia can be considered a syndrome or not constitutes a significant theoretical issue. Apraxia of speech, speech automatisms and agrammatism have been described in pure forms, and can be observed separately at different stages in the recovery process. Such dissociation is usually cited as evidence for separate cognitive representation. But the nonfluent character of agrammatic speech and the implication of the motor system suggests a relationship between articulatory production and syntax, and also gesture, supporting claims for a motor-gestural basis to syntax (e.g., Armstrong, Stokoe, & Wilcox, 1994). This is suggested by the high co-occurrence of aphasia, apraxia of speech and other facial and limb apraxias (for a review, see Code, 1998), although, again, these impairments can also dissociate. These observations converge also with evidence for a common genetic base for motor immaturity and specific language impairment in children (Bishop, 2002) and evolutionary bonds between them (e.g., Arbib, 2005: Corballis, 2002; Greenfield, 1991; Kimura, 1976).

The close relationships between acquired agrammatism and speech apraxia converge with behavioural and genetic investigations of the KE family. Some members of this family share an inherited impairment of speech and facial praxis, syntactic processing and more general language impairments, due to damaged expression of the FOXP2 gene for family members with the condition, but not those without the condition (for a discussion, see Corballis, 2004; Marcus & Fisher, 2003). The behavioural studies identified facial and speech apraxia as core elements of the condition and these studies appear to have implications for a close evolutionary relationship between facial action, speech action, and syntax.

The neural location for the control of articulate speech identified by Broca too has undergone revision and extensive expansion since first identified, and we sketch that journey below.

Broca's area

Recall from Chapter 3 that Ernest Auburtin took it upon himself to show that his mentor and father-in-law Jean-Baptiste Bouillaud was correct in his claim that the frontal lobes were the seat of the faculty of language. Auburtin had presented cases of frontal damage with language impairment, but in

1861 he accompanied his colleague Broca in his examination of Leborgne at Bicêtre Hospital in Paris with subsequent autopsy. We can note at this point that Broca's autopsy did not entail a detailed sectioning of the brain, as would be conducted nowadays, but a grosser examination. His examination revealed a large lesion, the size of a hen's egg, involving part of the first temporal convolution, the insula, the striatum and the centre of the damage was in the second and third frontal convolutions. Broca announced that the area sustaining most damage, the second and third frontal convolutions, was the centre for the motor control of speech at the subsequent meeting. This area, made up of the pars triangularis and pars opericularis, Brodmann's areas 44 and 45, is the one now known as Broca's area. Broca pointed out that, while the lesion had probably grown over the 21 years that Leborgne's condition had developed, it had probably started in the third frontal convolution and that his first symptom had been a loss of articulated speech. The first major criticism of Broca's location for the faculty of articulated speech was in a series of short articles by Marie (1906a, 1906b, 1906c), discussed more fully in Chapter 5. It will be recalled that Marie considered the third frontal convolution to be language-*irrelevant* and having no special role in speech production. He reported cases where severe damage to this convolution did not result in aphasia and was able to show that Broca's aphasia could exist without a lesion of the third frontal convolution. For Marie, 'motor' (Broca's) aphasia was always the result of a lesion that affects both the first temporal convolution and what he dubbed the 'lenticular zone'.

For Marie a lesion of this zone will lead to anarthria, his term for what most now call apraxia of speech, and for him *'l'anarthrie n'est pas de l'aphasie'*, anarthria is not aphasia. His examination of the brains of both Leborgne and Lelong led to his claim that these two cases did *not* support Broca's conclusions. He described atrophy in Lelong's brain which also compromised Wernicke's area. In Leborgne's brain, Marie pointed out that the softening discovered by Broca reached not only into the first temporal convolution but also, he claimed, the supramarginal gyrus in the parietal lobe.

In truth, problems with the identification of 'Broca's area' permeate the history of aphasia. In his Hughlings Jackson Lecture delivered to the British Royal Society of Medicine in 1961, Brain (1961) wrote

> We may note in passing how very slender today seems the anatomical basis for Broca's ideas, for the patient from whom he inferred that the lesion of the frontal lobe was responsible for the loss of speech, had a softening of the left cerebral hemisphere which not only involved the greater part of the frontal lobe but produced a cavity the size of a hen's egg, which extended backwards as far as the ascending parietal convolution, below to the 'marginal convolution' of the temporosphenoidal lobe, and in its depth involved the insula and 'extraventricular nucleus' of the corpus striatum.
>
> (Brain, 1961: 145)

The brain of Leborgne was rediscovered in the 1970s, and further investigations were conducted in Paris, including a CT scan (Signoret et al., 1984). The investigators found extensive damage beyond areas 44 and 45 that extended subcortically into the insula and the basal ganglia, especially the lenticular nucleus, but, despite Marie's claim, there was no damage to the supramarginal gyrus.

In the 1970s influential work on the redefinition of Broca's aphasia and Broca's area was conducted by Mohr and colleagues (Mohr, 1976; Mohr et al., 1978). They conducted a study of 20 cases from the past records at the Massachusetts General Hospital using autopsy, CT scan or arteriogram and neuropsychological examination and Moutier's (1908) and von Monakow's (1914) literature surveys of 125 published cases since 1820 to examine the nature of the symptoms and the extent of damage to Broca's area and surrounding brain. Mohr et al. (1978) concluded that Broca's aphasia as conventionally defined in 1978 did not result from a lesion limited to Broca's area, but resulted from a large lesion involving the area of supply of the upper division of the left middle-cerebral artery which produces a global aphasia. The damage includes the operculum, the third frontal convolution, the anterior parietal region, the insula, and both sides of the central Rolandic fissure, extending deep into the underlying white matter. This produces what they defined as 'Big Broca's aphasia' or the *operculum syndrome*, with a severe and persisting apraxia of speech with either mutism or a non-lexical speech automatism with the later emergence of agrammatism and severe reading and writing problems if there is any recovery. A lesion limited to the classic Broca's area can cause the patient to be mute at onset, but within days this can improve to a milder form of apraxia of speech. Other studies using the then new CT scanner showed that the correlation between lesions in Broca's area and Broca's aphasia was poor (e.g., Basso et al., 1985; Naeser & Haywood, 1978). For instance, Basso et al. (1985) found that the prediction of lesion site from aphasia type was something in the region of 83 per cent. Findings like these led to dissatisfaction with studies of aphasia using group methodology, as discussed in Chapter 8, and a rediscovery of the value of single-case investigations.

Investigations of the role of the insula continued to engage researchers. It will be recalled that it has been known that the insula was damaged in Leborgne's brain since 1861 and Meynert, de Boyer, Wernicke and Freud, among others (for a review, see Ardila, 1999), maintained that it was implicated in speech and/or language disorder, and a range of modern anatomical investigations discussed above confirmed that it was implicated in 'big' Broca's aphasia. Benson had long advocated a role of the insula in language, speech and other functions (Ardila, 1999; Benson, 1979), but it was in apraxia of speech, the impairment to the planning and/or programming of speech, that it became the focus of more contemporary studies. An influential lesion-overlap study was conducted by Dronkers (1996), where she compared the CT or MRI scans of 25 speakers, all at least 12 months post-onset of stroke and

with relatively large lesions diagnosed with AoS, with 19 stroke survivors without AoS. In the AoS speakers she found lesions that all included the precentral gyrus of the anterior insula of the left hemisphere, but the precentral insula was spared in all 19 speakers without AoS. Damage to the left anterior insula, she concluded, causes AoS and is involved in articulatory planning. More recently Dronkers, Plaisant, Iba-Zizen, and Cananis (2000) were able to re-examine both Leborgne's and Lelong's preserved brains, now housed at the *Musée Dupuytren* in Paris, using structural MRI (the brain of Lelong for the first time). They found extensive damage well beyond Broca's area, including into the basal ganglia. In the case of Leborgne, insula, superior longitudinal fasciculus, calostrum, putamen, globus pallidus, caudate head and internal and external capsule. However, the insula in Lelong was spared, but the superior longitudinal fasciculus was damaged. As noted, Broca's area (BA 44/45) is considered to be the posterior third of the inferior frontal gyrus, interestingly, Leborgne's brain had damage to the middle third and the inferior frontal gyri with some damage to the posterior third, and Lelong's lesion spared two-thirds of Broca's area, with damage only to the posterior half of the pars operculus (BA 45).

A study by Hillis et al. (2004) of AoS and the anterior insula points out the limitations of the lesion-overlap methodology used by Dronkers with chronic stroke survivors with large lesions. Hillis et al. (2004) examined areas of damage and areas of brain dysfunction indicated by low blood flow (hypofusion) in participants within 24 hours of stroke, to minimise the possibility of brain reorganisation, who had insula damage (N = 40) and without insula damage (N = 40). In this way they were able to identify the probability of the lesion causing AoS as well as the probability of AoS simply being associated with the lesion. They found no association between AoS and lesions of the left insula, the anterior insula or the precentral region of the insula. Indeed, they found an association with structural damage or low blood flow in Broca's area for those patients with AoS speech.

Towards the end of the twentieth century there was a significant expansion of experiment and theory on the involvement of Broca's area in a range of cognitive processes that began to emphasise that 'it would be a serious mistake to assume that Broca's area is a language-specific area' (Hagoort, 2005: 161) and studies began to highlight its contribution to brain-wide distributed networks subserving cognitive function.

Paulesu et al. (1997) conducted an fMRI study using variants of the classic phonemic and semantic *speech fluency* tasks (used clinically to examine the integrity of the left frontal lobe by asking patients to generate words beginning with a specific phoneme or as many words as they can in a semantic category, like words beginning with F, A, or S, in the case of the former or animals for the latter). They found that the posterior opercular area of Broca's area was activated during phonemic fluency and a separate, but overlapping, area of Broca's area (the retrospherical region) was activated during semantic fluency. More recent work (Arbib, 2005; Corballis, 2002) emphasises

the close relationships between speech, language and gesture/action and the emergence of articulate speech, syntax, action/gesture and lateralisation are seen to be closely related in evolution (Arbib, 2005; Corballis, 2002; Greenfield, 1991). Greenfield (1991), in an influential article, drew attention to the role of Broca's area in the organisation of developing speech and the capacity to combine objects like tools manually in the first two years of life, which, she hypothesised, establishes a foundation for the evolution of language. Some of the symptoms of Broca's aphasia have been utilised by a number of studies in language and speech evolution. MacNeilage (1998) proposed the Frame/Content theory to explain the evolution of speech from the close–open cycle of the mandible that originally evolved for mammalian chewing, sucking and licking to the emergence of consonant–vowel (CV) basis for speech, and MacNeilage and Davis (2001) confirmed the major similarities between the development of the CV syllabic characteristics of babbling in children and the non-lexical speech automatisms made up of recurring CV syllables discussed above. The relationship between the lexical speech automatisms of Broca's aphasia and their eventual recovery to agrammatism has been suggested as a possible model for the evolution of *proto*-syntax to full recursive syntax in evolution (Code, 2005). Imaging and electrophysiological studies have identified an enlarged frontal region engaged in speech and language and Hagoort (2005) has called this enlarged area *Broca's complex*. Activation of this region is seen while experimental participants engage in semantic (BA 47 and 45), syntactic (BA 45, 44, 46) and phonological processing (BA 44, 6) with substantial overlap. Hagoort's review identifies a cognitive selection and binding or unification role for information retrieved from the mental lexicon stored in the temporal lobe for the enlarged region, and emphasises its role in maintaining information on-line while the binding operations can take place. Thompson-Schill (2005) stresses the importance of Broca's area in the selection of information from competing sources. Working memory, as alluded to above, is seen to be necessary for most language, speech, and other cognitive tasks. It becomes necessary for the holding of information for short periods so that the information can be manipulated within cognitive space, and the enlarged Broca's region has been widely implicated in working memory. For instance, Paulesu, Frith, and Frackowiak (1993) found BA 44 and bilateral insula activation during a working memory task that required letter rhyming and remembering. More recently in an extensive review, Cabeza and Nyberg (2000) established that neuroimaging studies have found activation during working memory tasks in Broca's area and other prefrontal cortex in nearly 60 studies.

Summary

The anatomical area and the syndrome named after Broca have both expanded unrecognisably since they were first identified: the brain area from the third frontal convolution to one encompassing an enlarged frontal complex

responsible not just for 'the faculty of articulated speech'; the syndrome now includes a nonfluent amalgam of apraxia of speech, speech automatisms, agrammatism, sentence comprehension impairments, deep dyslexia, phonological dyslexia and dysgraphia. Research suggests that Broca's enlarged area is engaged in a wide range of language and language related cognitive functions in a networked relationship with many regions of the cortical and subcortical brain. We now have a broader and a deeper understanding of aphasia and of how the brain organises language, and this is due in no small part to the fallout engendered by Broca's original interpretations of what he found. In retrospect, we know that his views were influenced by the bias of his collaborator, Ernest Auburtin, who in turn was influenced by Jean-Baptiste Bouillaud, who in his turn was concerned with localising 'the language organ' to the frontal lobes, where Franz Joseph Gall had proposed it resided. We now have a broader understanding of how Broca's area, the most famous yet still mysterious part of the human brain, contributes to human mental life. Broca's area and its syndrome endure into the twenty-first century, and so do some of their mysteries, despite the ingenious methods we can devise to probe the anatomy and physiology and despite the experimental tasks and techniques we can invent to explore the impairments that result.

10 Beyond the left peri-Sylvian language area

Developments in imaging in the 1970s began to confirm that areas of the brain outside the classic left peri-Sylvian region could produce impairments in language processing. Classic aphasic symptoms could arise following subcortical damage and damage to the right hemisphere could produce other, apparently non-aphasic impairments in language use. Additionally, researchers became aware that various forms of language impairments could accompany progressive neurological damage. Consequently, a broader appreciation of the involvement of the whole brain in language processes began to emerge. In this, the final chapter, we present an overview of the main thrusts of these developments in the latter half of the twentieth century. In so doing it becomes clear that aphasiology has expanded beyond the recognition of eighteenth and nineteenth century physicians, and even in the past 100 years. But, despite our sophisticated methods of investigation, there is still much to know about the nature of language and its breakdown following damage to the brain.

Beneath the cortex: subcortical aphasias

Although there had been signs that lesions to subcortical structures participated in language and speech disorders (e.g., Alexander, 1989: 47 mentions at least Marie, Dejerine, and Liepmann), and Wernicke had discussed the concept of subcortical aphasia with reference to the white matter fibre connections located directly beneath the neocortical surface, the discovery and exploration of **subcortical aphasias** only became possible with the development of methods that could visualise subcortical structures and show subcortical lesions (Cappa & Abutalebi, 1999: 321ff.; Crosson, 1992: 1ff.; Crosson & Nadeau, 1998: 432).

The **basal ganglia** and the **thalamus** are subcortical structures that have received the most attention in studies of language processing and subcortical aphasia (Cappa & Abutalebi, 1999; Crosson, 1992). The basal ganglia are, among other things, formed by the globus pallidus and the putamen, which are together referred to as the lentiform nucleus. Together, the caudate nucleus and the putamen form the corpus striatum and the thalamus is a complex

formation consisting of many nuclei. These neuronal structures have many connections with frontal and parietal regions and are engaged in motor and sensory processing and the normal processes that translate thought into action, including speech action.

A range of studies have shown reliable correlations between subcortical lesions visible on brain scanning and speech and language problems. As Alexander, Naeser, and Palumbo (1987: 984) put it: 'Current evidence from aphasia assessment and CT scan lesion studies suggests a coherent relationship between subcortical lesions and language/speech disorders.' Damage to the basal ganglia often leads to semantic and verbal paraphasias, word-finding disturbances and writing problems (Cappa & Abutalebi, 1999; Cappa et al., 1983). Sometimes fluent aphasia, echolalia and perseverations also occur. With damage to the thalamus the deficits seem to be more uniform and the 'thalamic aphasia syndrome' (Wallesch, Johannsen-Horbach, Bartels, & Hermann, 1997: 408) displays 'relatively fluent speech behaviour with semantic paraphasias and word-finding disturbances, intact faculty to repeat, and preserved auditory comprehension' (Cappa & Abutalebi, 1999: 323).

The neoclassic model can accommodate **subcortical aphasic syndromes** where specific syndromes can be attributed to specific lesion locations (Cappa & Abutalebi, 1999: 324). Researchers subsequently identified a series of subcortical aphasic syndromes. Helm-Estabrooks and Albert (1991: 41ff.), for instance, distinguish anterior capsular/putaminal aphasia, posterior capsular/putaminal aphasia, global capsular/putaminal aphasia, as well as thalamic aphasia. Others (e.g., Kertesz, 1994) assume the existence of subcortical aphasias, but they see no difference between cortical and subcortical aphasias: 'In clinical terms, despite much variation within each group, subcortical lesions, on the whole, impair language abilities to the same extent that cortical lesions of similar volume do' (Kertesz, 1994: 76).

Imaging studies have shown that specific areas of subcortical structures are connected to individual symptoms (e.g., Démonet, Puel, Celsis, & Cardebat, 1991) and significant activation in the thalamus and basal ganglia in normal participants when processing single words, but display no clear activity patterns during phonological tasks (e.g., Frith et al., 1995; Herbster, Mintun, Nebes, & Becker, 1997). The basal ganglia and its participation with the ancient frontal limbic system, is known to be involved in the processing of emotionally charged communications, as between mother and infant and in the production of sexually explicit language, expletives and in the **copralalia** of Gile de la Tourette's syndrome and in the production of aphasic speech automatisms (Van Lancker & Cummings, 1999).

What research has not yet unravelled is the extent to which subcortical aphasia is caused directly by disruption to speech and language processes represented in subcortical structures (e.g., Wallesch & Papagno, 1989; Wallesch et al., 1997), or whether the disorder is caused by the interruption of pathways through subcortical regions from one cortical area to another or through

diaschisis. The significant current question therefore is, in what ways do subcortical and cortical structures cooperate in the processing of complex speech and language functions?

Crosson and Nadeau consider that language deficits after basal ganglia damage are always of a cortical nature, and only a consequence of the interruption of the blood flow to cortical regions by a subcortical lesion (Crosson & Nadeau, 1998; Nadeau & Crosson, 1997). Wallesch and co-workers, on the other hand, have formulated the hypothesis that the striatum plays an important role for the integration of different inputs (situation, emotion, motivation) that are crucial for the choice from different lexical-semantic entries. The thalamus has a steering function that is involved in choice from lexical alternatives, critical for language processing, and with damage causing semantic impairments.

Work on the neurophysiology of language and speech processing received a boost from investigations of subcortical aphasias (e.g., *Brain and Language*, issue 58/3). Investigation of the participation of subcortical structures in speech and language processing came not only from studies of subcortical aphasias. Data from imaging studies with speech-healthy participants and investigations of Parkinson disease, the effects of stereotactic surgery and electrical stimulation studies were contributing to the development of new insights (e.g., in Wallesch & Wyke, 1985). As Cappa and Abutalebi (1999: 324) write: '[I]t must be acknowledged that these efforts to build neurologically plausible models of language organisation in the brain represent serious attempts to move beyond the simple "correlational" approach which has until recently characterised aphasiological research.'

The existence of subcortical aphasia indicates that assumptions that language is processed exclusively by the cortex could no longer be sustained and the earlier focus solely on cortical structures declined. If the goal of cognitive neuroscience is, among other things, 'the development of an explicit neurological model of speech implementation in the brain, then specific hypotheses on the linguistic task of subcortical structures must be formulated within this conceptual framework' (Cappa & Abutalebi, 1999: 325).

Language from the other side of the brain: right hemisphere damage

Both Broca and Hughlings Jackson more than entertained the idea that the undamaged right hemisphere was involved in language and in the recovery of language in aphasia. For Jackson, as we have seen in Chapter 4, 'the right hemisphere is the one for the most automatic use of words, and the left the one in which automatic use of words merges into voluntary use of words – into speech' (Hughlings Jackson, 1874: 130). But for Geschwind's (1965) neoclassical model, the right hemisphere had no role; it was mute, word deaf and word blind. However, as one of us wrote in 1987, 'until relatively recently one of the strongest myths of neuropsychology has been that unilateral right

hemisphere damage does not produce language problems' (Code, 1987: 44) and a significant literature emerged on right hemisphere language in the post-1945 era continuing into the latter part of the twentieth century. This literature has a number sources. It came from the commisurotomy and callosotomy studies originating with Roger Sperry and Michael Gazzaniga in the early 1960s and the studies of people who had undergone **hemispherectomy**, a complete, or nearly complete, surgical removal of the left hemisphere. We shall not review this work in detail (for a review, see Code, 1987), but focus attention on the research that has examined language impairments resulting from right hemisphere damage.

Early observations by Eisenson (1962) raised the first questions regarding the intact quality of language following damage to the right hemisphere. He found marked deficits in vocabulary and sentence completion involving abstract words. These impairments are not noticeable in normal discourse nor were they picked up on standard aphasia testing. Lesser (1974) found errors on tests of semantic comprehension in a group with right hemisphere damage, but not in syntax. As discussed in Chapter 8, there is more to language than that which can be handled by a strictly componential linguistic model, but at that time such models had not been developed in aphasiology. However, by the 1980s it was becoming clearer that while aphasic individuals have problems with the structural linguistic components of phonology, morphology, syntax and lexical semantics, what appeared to be an essential feature of the right hemisphere's involvement in language processing was not captured by structural linguistics. It was assumed that underlying the componential processing of the left hemisphere was a serial, analytical, segmental processing capability. In comparison, some suggested that the right hemisphere's fundamental processing mode was holistic and parallel. So left hemisphere damage produced problems in context-free linguistic processes, like syntax and phonology, whereas right hemisphere damage produces impairments in context-dependent, complex linguistic entities like verbal jokes, metaphors, narratives, indirect speech acts, as well as semantic discrimination and intonation (for reviews see Code, 1987; Joanette et al. 1990; Wapner, Hamby, & Gardner, 1981), and by the 1980s there was a large body of research showing that the right hemisphere was involved in the processing of various suprasegmental features of prosody, emotional prosody, and emotional language (Ley & Bryden, 1981).

Recovery from aphasia and the right hemisphere

A common observation in aphasiology is that the initial consequences of brain damage are more severe than the eventual consequences and that most people make some, at least, spontaneous recovery with time. As we have seen, the dogma of left hemisphere dominance took hold in neurology (and later in neuropsychology) following the impact of Broca's studies in the 1860s, and it was with Broca that the role of the right hemisphere in the recovery of

aphasia was first entertained. This is a logical implication of the dominance model: if one hemisphere is responsible for language then it is necessary to account for recovery following left hemisphere damage, especially widespread damage. So some form of the **lateral shift hypothesis** has a history originating in the latter half of the nineteenth century.

But the idea of lateral or hemispheric shift takes several forms, and Brown (1979) articulates the variants as follows:

> Does the right hemisphere account for the various degrees of insufficiency in aphasia or is the right hemisphere truly aphasic? Is the right hemisphere effect a compensatory one, in which a mirror system either limited in capacity or differing in design 'takes over' for the damaged side, or are structures in the right hemisphere part of a unitary bilateral organisation mediating language?
>
> (Brown, 1979: 137)

Sir William Gowers (1845–1915) qualified in medicine at University College London in 1867, receiving his MD in 1870. In 1880, he was appointed physician to the National Hospital in Queen Square, London in 1880, and later became professor of clinical medicine at University College Hospital, London. Gowers (1887) was the first to make the claim that the aphasic individual who makes some good recovery, but then becomes aphasic again following a second stroke but this time to the right hemisphere, is powerful evidence that the right hemisphere must have been responsible for the initial recovery following the first stroke affecting the left hemisphere:

> Loss of speech due to permanent destruction of the speech region in the left hemisphere has been recovered from, and that this recovery was due to supplemental action of the corresponding right hemisphere is proved by the fact that in some cases, speech has been again lost when a fresh lesion occurred in this part of the right hemisphere.
>
> (Gowers, 1887: 131–132)

Henschen (1922) too argued for the right hemisphere's role in recovery, based on the same 'second lesion' argument put forward by Gowers. In fact, the idea that the right hemisphere is responsible for recovery in aphasia is sometimes called **Henschen's Axiom** (Kertesz, 1979).

In the period following the Second World War, American neurologist **Johannes Nielsen** (1890–1969) described several cases where 'the minor cerebral hemisphere assumes the function of the major in language with great facility in some instances, with great difficulty in others, and not at all in some persons' (Nielsen, 1946: 155). With the advent of the CT scanner, a number of studies of globally aphasic people with massive damage to the classic language areas but who had made remarkable recoveries suggested to the investigators that the right hemisphere must have been responsible for the

observed recovery (Cummings, Benson, Walsh, & Levine, 1979; Pieniadz, Naeser, Koff, & Levine, 1983).

A few studies used the Wada technique (see below) to examine speech and language functions in the right hemispheres of aphasic participants. With this technique investigators are able to inject the barbiturate sodium amytal into the carotid artery in the neck to anaesthetise most of the hemisphere on the side of the injection. Kinsbourne (1971) tested three aphasic participants and found not only impairments in speech when the right hemisphere was anaesthetised, but also some slight speech difficulties when the left was anaesthetised. One patient 'was totally unable to phonate or even to move his tongue and lips to command. Afterwards he reported that he had tried to speak, knew which words he wanted to use, but found himself unable to exert control over his speech musculature' (Kinsbourne, 1971: 303). This sounds like a complete anarthria, which may have been induced by the combination of left hemisphere damage and the temporary right hemisphere 'damage' caused by the barbiturate. A larger study of 22 patients was conducted by Czopf (1972, 1979), who reported that under right hemisphere anaesthetic 10 participants produced very severe impairments in language functions; in a further group it had a moderate effect and in three individuals it had no effect. These latter three individuals had milder aphasia. Czopf (1979: 29) concludes that the effect of right hemisphere anaesthetisation 'was extensive in those cases whose aphasia had originally been severe and had been present for a long time'.

With the development in the 1960s of experimental methods like **dichotic listening** and **tachistoscopic viewing**, that attempt to measure lateral preference or advantage for verbal and non-verbal material presented to the ears and the eyes, studies developed into the 1970s and 1980s that examined the responses of aphasic participants. Results were initially promising, and some aphasic participants were shown to have increased left ear or left visual field advantages for verbal material (indicating increased right hemisphere involvement in the processing of the tasks). The most interesting finding was that there appeared to be an increase in left ear advantage on verbal dichotic tasks with time post-onset of aphasia and with increased severity of aphasia, providing some support for the earlier finding that the right hemisphere became more engaged in more severely aphasic people and with time since the original damage. But results were unreliable because of the probability that brain damage itself induces a so-called 'lesion effect', where damage to any part of the auditory verbal processing system produces an impairment or a complete extinction of the dichotic signal in the ear opposite the lesioned hemisphere (for a review, see Code, 1987). Dichotic listening to examine hemispheric processing, while cheap and non-invasive, became increasingly obsolete for examining people with brain damage as the new imaging techniques came on stream in the 1990s.

One variant of the lateral shift hypothesis is that some of the symptoms observable in aphasia – agrammatism, paraphasia, speech automatisms,

dyslexia, and so forth, are produced not by the damaged left hemisphere but by the intact right. The evidence for this view is sparse and mainly inferential. Right hemisphere hypotheses have been developed for some kinds of speech automatisms (Code, 1987, 1991a) and some of the features of acquired dyslexia (Coltheart, 1980b; Landis, Graves, & Goodglass, 1982; Weniger, Kitteringham, & Eglin, 1988). The strongest position holds that following left hemisphere damage there is a shift of control and the right hemisphere is released from the inhibition of the dominant left. Related to this is the notion that linguistic abilities that have been 'latent' can emerge from the right hemisphere following damage to the left (Moscovitch, 1973). The weakest claim is that the right hemisphere's contribution following left hemisphere damage is a simple compensatory one where, for instance, visuospatial processing is utilised by the patient to compensate for lost linguistic skills. These two last broad forms inspired ideas in the 1980s on how to employ the right hemisphere in treating aphasia (for a review, see Code, 1994a).

As the twentieth century closed, there were strong indications from a range of studies that the involvement of the right hemisphere in aphasic individuals varies as a function of severity and time since onset: the more severe the aphasia and the more time has elapsed since the damage, then the more involved the right hemisphere appears to be (Cappa & Vallar, 1992; Code, 1987; Gainotti, 1993; Weniger et al., 1988). In 1992, Cappa and Vallar felt able to conclude from their review of the role of both hemispheres in recovery, that the right hemisphere was involved, dependent on time post-onset and lesion-related and individual factors. There was, however, converging evidence for the contribution of both hemispheres. Similarly, from their functional MRI study, Cao et al. (1999) found that language activity significantly increased in the right hemisphere and nonsignificantly decreased in the left in recovered patients. However, bilateral functional language networks were observed which included partial restitution of damaged functions in the left and activation of areas in the right. Where there was failure to restore any left hemisphere language function, this led to mainly right hemispheric language networks. So better language recovery was associated with both left and right hemisphere involvement in aphasia.

Progressive aphasia

The pattern of lesioning to the brain caused by stroke is a reflection of the impairment to blood supply caused by the stroke. Thus, aphasia arising from inferior frontal stroke is caused by impairment to the middle cerebral artery that supplies the anterior brain. Lesions can also often be large. Such facts suggest that lesions thus caused have a *horizontal* impact and cross cognitive systems (Croot, Patterson, & Hodges, 1998). The implication is that aphasia following disturbance in blood flow can never provide a clear picture of the role of affected brain structures in language processing.

Extensive study since the early 1980s has revealed that language and speech

can be impaired in a variety of ways by progressive neurological damage in the absence of significant impairments to other cognitive processes (Croot et al., 1998; Duffy & Petersen, 1992; Garrard & Hodges, 1999; Harasty, Halliday, Kril, & Code, 1999; Jefferies et al., 2006; Mesulam, 1982; Nestor et al., 2003; Patterson, Graham, Lambon Ralph, & Hodges, 2006; Snowden, Goulding, & Neary, 1989). It has been known since at least the 1890s that aphasia is part of the widespread cognitive deterioration associated with dementia, and the original case described by Arnold Pick (1892) had a progressive aphasia in addition to other progressing impairments. **Primary progressive aphasia** was first described in modern times by Mesulam (1982), although a case of primary progressive pure word deafness was described by Sérieux in 1893 (Sérieux, 1893; see also Ceccaldi, Soubrouillard, Poncet, & Lecours, 1996).

The interest of researchers arises because progressive neural degeneration is said to follow more *vertically* represented functional systems compared to stroke or traumatic damage, implying that a gradually progressive decline follows more closely the ontogenic and phylogenic development of functional neural systems (Croot et al., 1998). This assumption provides a new appreciation of the relationships between cognitive systems and their neural representation, as well as evolutionary relationships between different modular architectures of cognitive systems and their neural representation.

Different forms of progressive speech and language impairment have been identified and two broad types began to be described in the 1990s (e.g., Duffy, 2006; Kertesz, Hudson, Mackenzie, & Munoz, 1994; Patterson et al., 2006; Snowden et al., 1989). **Semantic dementia** (SD) (Garrard & Hodges, 1999; Snowden et al., 1989) arises from progressive damage either restricted to the mainly anterior/inferior temporal lobe or temporal lobe plus some frontal parieto-temporal damage, and is characterised by fluent speech with significant impairment of semantic processing. **Progressive nonfluent aphasia** (PNFA) arises mainly from progressive left frontal damage extending into subcortical areas, and can include varying severities of motor speech impairment, naming impairments, syntactic deficits and comprehension impairments. While the differential emergence with time of impairments in people with progressive conditions allows the possible examination of relationships between different components of the language and cognitive system, significant variability in the pattern of emerging deficits would appear to be an impediment to reliable attempts to reveal general relationships between functions. The emergence of symptoms may be purely coincidental and explainable better in terms of the gradually progressing and increasingly extensive neural damage, than as a reflection of presumed relationships between functions or systems. Nevertheless, longitudinal investigations of progressive conditions have the potential to test the idea that this progression can reveal something about the relationships between cognitive functions.

Summary

Early observations suggested that lesions to areas outside the classic peri-Sylvian language region could result in impairments of language processing. The subsequent development of neuroimaging confirmed that aphasic symptoms could arise from subcortical areas of the brain. Such findings raised questions about the cardinal role of the classic language areas. Investigations of patients with damage to the right hemisphere showed that they too could have language problems, although these problems were different to the kinds of impairments that follow left hemisphere damage. In addition, researchers became more interested in progressive aphasia, especially when aphasia was the primary condition, as it was reasoned that aphasia caused by progressive neurological disease could reveal something about how separate aspects of the language system were cognitively organised and neurologically represented.

Such data clearly confirmed that the more we learnt about language and its breakdown following neurological damage the more complex the representation of language in the brain became.

Postscript

At the beginning of the twenty-first century we know that language is more than grammar, syntax, and phonology; we know that the breakdown of language and communication following brain damage is more than 'aphasia'; we know that there are no language centres, but complex interrelated networks partially associated to motor and sensory systems in the brain and entailing the interactive collaboration of ancient and modern, cortical and subcortical structures. We suggested in our preface to this book that knowledge of the past is an important foundation to understanding the present, and our hope is that our efforts met at least some of the reader's expectations in this regard. As we would expect, aphasiology and aphasia are different at the turn of the twenty-first century to how they were at the beginning and further distance from events and developments will always provide a different picture. In this postscript we attempt to look a little way into the future and try to assess what aphasiology will be like in the next 20 years or so. Does a knowledge of the history of aphasia allow a more reliable prediction of its future?

The future unfolds in our peripheral vision as we walk backwards into it as a seamless merging with the present. Making predictions about the future, therefore, maybe a foolhardy enterprise, and the, often dramatic, failures of economists to predict how the stock market or national inflation will behave are examples of the difficulties of prediction, even when reliable figures from the past are available.

In the Western world, at least, predictions are that, as our diets become healthier and we accept that exercise and self-moderation become more a part of individual lives, the incidence of stroke should reduce over the coming years, and therefore the incidence of aphasia. But as the incidence of stroke in the 40 to 60 age range may indeed reduce, better medical interventions will see an increase in the number of survivors from stroke, many with aphasia. In addition, as people live longer, the incidence of progressive neurological conditions, including aphasia, is likely to increase. Therefore, we find it hard to predict either a reduction or increase of aphasia in the population. Predictions suggest that medical treatments available for people with aphasia will improve (Small, 2000, 2004) as pharmaceutical and neurophysiological approaches

refine, but that such treatments will have to be carefully combined with behavioural treatments conducted by experienced therapists.

But will aphasia still provide a testing ground for the advancement of understanding of the representation of language in the brain? Clinical endeavour is said to benefit from advances in basic knowledge and non-clinical research to benefit from the willingness of aphasic people to act as participants in non-clinical research. We see no reason to think that this mutually beneficial relationship will not continue. Computer simulations of aphasic conditions and increasingly sophisticated imaging studies with non-aphasic participants have been a feature of development in the later twentieth century, but we can ask if such studies make research with aphasic participants redundant? We would predict not, not over the next few decades at least, because simulations will need to be tested on real-life aphasic data and neurological models of brain activity are unlikely to merge into cognitive models of mind over this time frame either.

What would Broca, Wernicke, and Hughlings Jackson make of the current state of aphasiology nearly 160 years since Leborgne was first described? Broca and Hughlings Jackson would not be surprised perhaps that the right hemisphere has been shown to make a significant contribution to language and Broca, who wrote extensively about recovery and rehabilitation, would be interested to see that recovery from aphasia involved the right hemisphere, at least for some people with aphasia, and that rehabilitation now encompassed approaches beyond the treatment of aphasic impairments. Hughlings Jackson might be forgiven some self-satisfaction in knowing that his assertion that there was more to language than the propositional and referential had been borne out, and that there was more to the neural control of speech and language than Broca's area, Wernicke's area and the connections between them. He might feel similarly smug to find that cognitive neuroscience had been unable to find an anatomical 'faculty of language' confirming his contention that 'the so-called faculty of language has no existence'. Broca, and the others, would be fascinated to note that the area named after him had acquired more functions than just the control of the production of speech.

They would all be fascinated with our new methods for imaging activation associated with language processing in the brain and be excited by the development of the cognitive neuroscience of language, although they too would have reservations. Jackson might be heard muttering under his breath that functional activation imaging is all very well, but might share the view of some that much of it was little more than a technically sophisticated form of phrenology, and he might get slightly annoyed with studies that appeared to have forgotten, or perhaps never knew, that 'to locate the damage which destroys speech and to locate speech are two different things' (Hughlings Jackson, 1874: 130).

The founders of aphasiology might all be pleased, and maybe surprised, that their original ideas and models still inform research and theory and

interested to know whether research with people with aphasia will still be making a contribution in 20 or 50 years' time. As medical men, they might be disappointed that, despite significant improvements in the effectiveness of treatment for people with aphasia, aphasia still exists at all for a large proportion of people.

Appendix
Brain and language

An appreciation of the history of aphasia requires some understanding of the neural anatomy and physiology of language and speech and of the effects of brain damage on speech and language, and the main text of this book assumes some knowledge of these topics. The purpose of this Appendix is therefore to provide some basic introductory background for readers who are new to or unfamiliar with aphasia and its basic neuroanatomy. More detailed discussion can be found in the main text and in the references at the end of the Appendix.

The brain

Figure A.1 depicts a *lateral* view of the left hemisphere that shows the four major lobes – frontal, temporal, parietal, and occipital, and the main *gyri* or convolutions on the cortex or surface of the brain, together with the *sulci* or fissures, the valley-like structures between the convolutions. This convoluted structure of the cortex has evolved in order to extend the surface area of the cortex while remaining compact enough to fit within the skull. The diagram also shows the *central sulcus* or *Rolondic Fissure* that separates the frontal from the parietal lobes and the *lateral sulcus* or *Sylvian Fissure* separating the temporal lobe from the frontal and parietal lobes.

The figures in this Appendix provide a descriptive terminology developed by anatomists over the years for navigating around the three-dimensional structure of the body and brain. *Anterior* refers to structure in front of and *posterior* to structure behind the central sulcus. *Superior* and *inferior* refer to the areas at the top and bottom of the brain respectively. *Lateral* describes the external side of a hemisphere and *medial* the inside surface of a hemisphere. *Dorsal* refers to the top surface, the 'back' of the animal, and *ventral* to the 'belly'. These terms are used relatively, not absolutely. So a structure can be described as 'anterior to' or 'dorsal to' another structure and terms can be combined in various ways. For instance, the classic Broca's area, shown in the figure, is represented in the inferior frontal brain, the visual cortex as *posterior occipital* and the premotor area of the frontal lobe as *dorsofrontal*. The *poles* of the frontal and temporal lobes are their respective

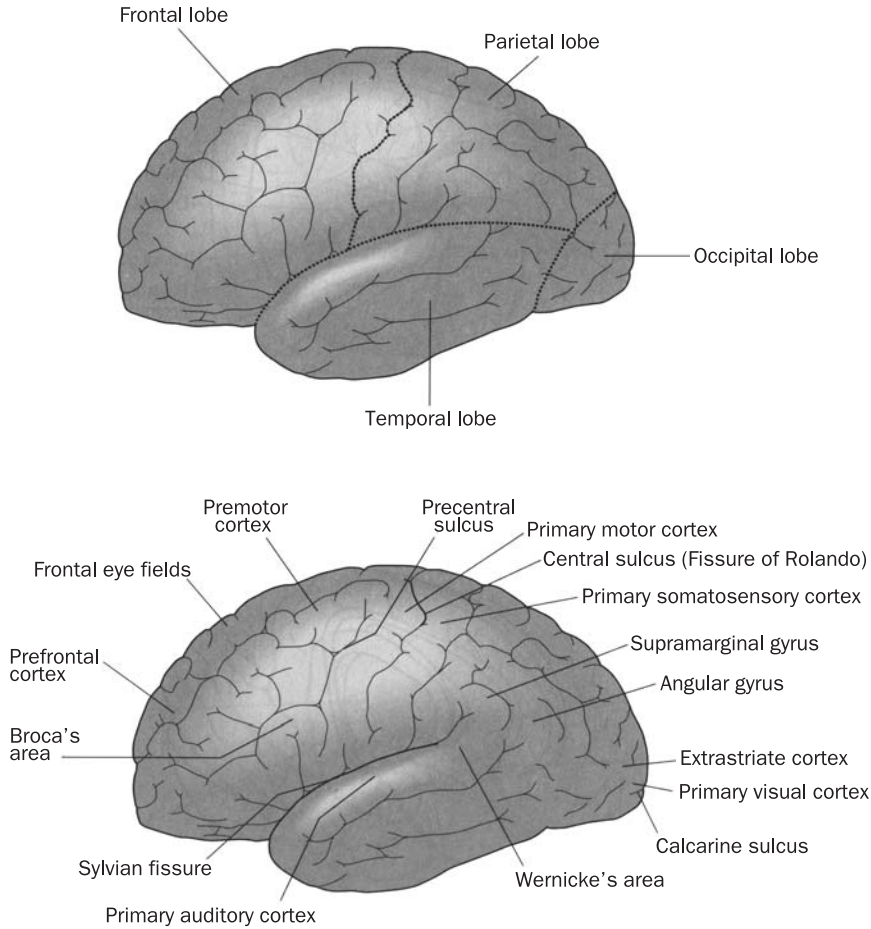

Figure A.1 Lateral and medial brain. Copyright © 2001 from *The Handbook of Cognitive Neuropsychology*, edited by B. Rapp. Reproduced by permission of Routledge/Taylor & Francis Group, LLC.

most anterior portions, and the most posterior portion of the occipital lobe. On the border of the most posterior part of the frontal lobe with the parietal lobe, is *the primary motor cortex* or *motor strip*. On the posterior side of the central sulcus, in the most anterior border of the parietal lobe, is *the sensory cortex* or *sensory strip*. Primary motor and somatosensory processes from the body are represented in these regions.

Figure A.2 shows the surface numbering system developed by Brodmann (1909), which is widely used as a further aid to brain navigation, Brodmann attempted to identify particular cortical sites, now called *Brodmann's areas* (BAs) with the layers of neurones immediately underlying the cortical surface. The structure of these layers entails different types and densities of neurones

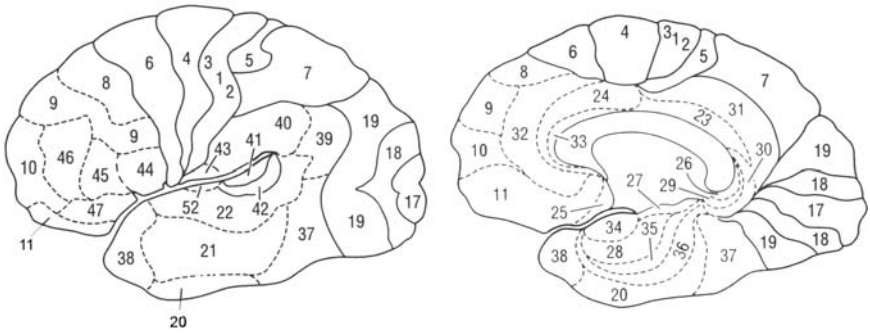

Figure A.2 Brodmann's areas. Copyright © 2001 from *The Handbook of Cognitive Neuropsychology*, edited by B. Rapp. Reproduced by permission of Routledge/Taylor & Francis Group, LLC.

and different connections between layers and between structures. For example, Broca's area is often described as including, at least, BA 44 and 45.

Each lobe of the brain appears to be concerned with some major domain of responsibility. The frontal lobes are concerned with planning, thinking, executive action and motor function, including motor speech. The parietal is concerned with the processing of somatosensory perception, which includes pain, pressure, temperature, and joint and muscle position sense (*proprioception*). The lobe's responsibilities include the integration of visual and somatosensory information. The temporal lobe is responsible for auditory perception and processing, including speech and language, and aspects of long-term memory. The occipital lobe's responsibility is for visual perception and processing. The anterior brain, in front of the central sulcus, is characterised as essentially a motor and executive system with the posterior brain, behind the central sulcus, described as a sensory and receptive system, although in normal functioning their activities are highly integrated.

Aphasia

The history of aphasia tells a story of disagreement and controversy, a story not uncommon in the evolution of a science. Controversy is also a significant feature of the contemporary study of aphasia, and, any attempt at a definition goes where angels fear to tread. But we will attempt a working definition here that allows a reader access to the contents of this book. The main text discusses in detail different attempts at definition but for the purposes of this introduction we prefer a fairly neutral, inclusive and generic use of the term *aphasia*.

Aphasia is the term most workers in the field would use to describe impairments of the *use* of language, the expression and comprehension of language in any modality – whether through speech, writing, or linguistic signing – and is caused by some acquired form of damage to the brain.

So aphasia is not an impairment of articulation (*dysarthria*) or of voice (*aphonia*).

One view is that the term aphasia should be reserved for the observed impairments or symptoms of aphasia that arise from damage to the left hemisphere that can be described in terms of the core domains of a standard linguistic model. These domains are semantics (word and sentence meaning), syntax (grammatical structure), morphology (word structure), and phonology (sound structure). This model deals with *componential* aspects of language (e.g., words, phonemes, syllables, morphemes) and their *combinatory* features (syntactic and morphological combination, for instance) and provides an account of many of the features of aphasia accompanying stroke, but is less satisfactorily dealing with *discourse* and everyday *conversation*, which can also be impaired, particularly following traumatic head injury, right brain damage and some progressive language conditions. For instance, the model excludes some complex aspects of language use, and consequently language impairments arising from damage to the right hemisphere, which is established as being heavily involved in processing aspects of language not described by the components of a standard linguistic model. These complex features of language include such things as using verbal jokes, understanding inferences and metaphors, using pragmatic aspects of language concerned with the behavioural context of language use, and prosody, the meaning carried by stress, rhythm, intonation and emotional tone.

But conventionally the term aphasia does not include the impairments to language use accompanying right brain damage, traumatic brain injury, although is commonly associated with the language impairments that develop in a range of dementias and other *progressive* conditions. The term also includes what is most often called *apraxia of speech*: impairments in the fluent production of speech arising from damage to the mechanisms controlling the planning and programming of speech before articulation is attempted. In apraxia of speech, there is no muscular paralysis or in-coordination and therefore apraxic impairments to motor speech are not due to dysarthria.

The features of aphasia

A dictionary of some length could be filled with the array of terms that have been introduced over the years to describe the symptoms, characteristics or features of aphasia arising from left hemisphere damage. The meaning of most of the main terms is explained in the main text or can be checked in the Glossary, and here we shall mention only the major features of an aphasic problem arising from left hemisphere damage.

An aphasic condition is said to be *fluent* or *nonfluent*. A person is nonfluent if their speech is hesitant with many pauses, is slow and with a lack of articulatory dexterity and melody in speech. Some speakers are significantly nonfluent with severe apraxia of speech. This fluency dimension is commonly used as an initial demarcation, and comes with an implication that an aphasic

speaker can be assigned to a fluent or nonfluent classification. What are usually called the *classic* aphasia types arising from left hemisphere damage below are often grouped according to whether they are fluent or nonfluent forms of aphasia.

Anomia is a term that can have several meanings. It can refer to *a type of aphasia*, or it can be used to refer to a symptom. It is a particularly common symptom of aphasia and describes a problem with retrieving or accessing common words like the names of things, actions and events. Such naming or word-finding impairments are usually referred to as examples of *anomia*. There appear to be several forms of naming impairment producing different symptoms and apparently arising as a result to damage to different brain–language components or connections.

Also considered to result from an underlying anomia are *semantic paraphasias* where a speaker incorrectly produces a word that is semantically related to the intended word, like saying *table* for *chair*. Semantic paraphasia is sometimes called *verbal* paraphasia, although a verbal paraphasia is not necessarily a semantic paraphasia if it is not semantically related to the intended or target word. *Phonemic paraphasia* is where an individual produces incorrect speech sounds and appears to have difficulties with accessing or retrieving the appropriate phonemes for a planned utterance. So the speaker may produce *pat* instead of *cat*. Phonemic paraphasias can also result in verbal paraphasias, where the resulting utterance is an existing word (*hat* for *cat*), often called *formal* paraphasias.

If an aphasic speaker produces many paraphasic substitutions in their speech then the severely impaired result, that may be mostly unintelligible, is most often described as *jargon aphasia*. A speaker who produces phonemic paraphasia and/or jargon can be highly *fluent*, with no signs of hesitation or articulatory difficulty. Phonemic paraphasia is sometimes referred to as *literal* paraphasia.

Agrammatism is used to describe impairments in the use of syntax and can disrupt a number of syntactic processes. Common features of agrammatism in English may be impairments in verb inflections and a *telegraphic* or *telegrammic* style of speech; in telegraphic speech the small 'function words' like *the*, *a*, *and*, *but*, are omitted, which have a grammatical function, but the speaker retains the 'content' words, the meaning-carrying words in the sentence. Agrammatism can occur in writing also.

Alexia/dyslexia and *agraphia/dysgraphia* refer to reading and writing impairments respectively and are common and varied in aphasia, affecting single word and connected sentence processing: a wide range of types have been described.

Repetition impairments are difficulties repeating the speech of others. While this may seem to present few problems in everyday communication, it can have significant diagnostic and theoretical implications.

Speech automatisms and *stereotypes* are common in aphasic speech and can account for the majority of some severely aphasic speakers' utterances.

Various examples include expletives, sentence stems, *yes* and *no*, family names, high frequency and automatic expressions and simply repeated non-meaningful syllables like *ta*, *ta*.

Comprehension impairments, or impairments in understanding the speech of others, are common for aphasic people and can occur for sentence meaning, single word meaning and in reading.

As detailed in the main text, there have been many attempts to seek patterns and dissociations in the symptoms of aphasia and to identify types of aphasia within some classification or other. Many researchers and clinicians prefer not to use aphasia classifications at all but to describe the impairments they observe in objective terms without reference to a classification system. We would not argue with this approach, all classifications have theoretical implications, but for someone coming new to aphasia, some basic groupings can help provide a basis for understanding. We content ourselves therefore with a basic division, which should not be seen as the end of the story but simply the beginning.

Two major types of aphasia distinguished by the fluency dimension are *Broca's* aphasia, the major form of nonfluent aphasia, which is sometimes described as an *expressive* or *motor* aphasia, and *Wernicke's* aphasia, the archetypical fluent aphasia, a *receptive* or *sensory* aphasia. Broca's aphasia can be mild with some mild articulatory problems, word finding difficulties and mild agrammatism, or severe with prominent apraxia of speech, speech automatisms and severe agrammatism. In milder forms comprehension may be well preserved. Wernicke's aphasia in contrast can include significant

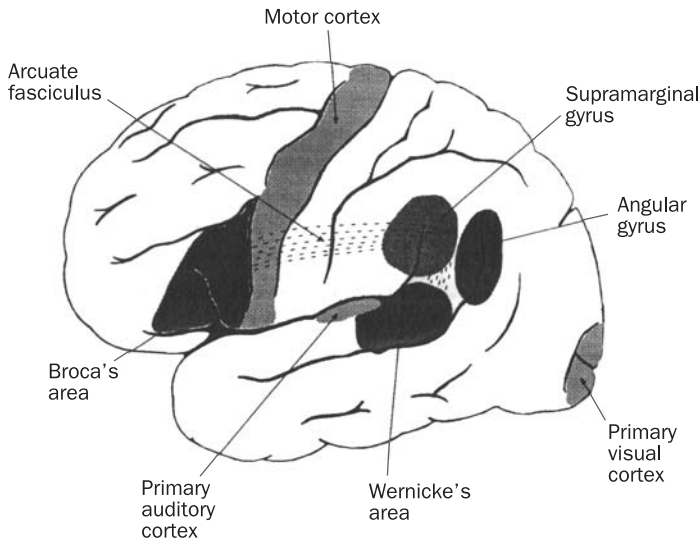

Figure A.3 Geschwind brain and language model from 'Language in the human brain', by D. Howard. Chapter 9 in *Cognitive Neuroscience*, edited by M. D. Rugg. Hove, UK: Psychology Press, 1997. Reprinted with permission.

fluently produced paraphasias and comprehension difficulties with jargon characterising more severe forms.

Figure A.3 presents a version of Norman Geschwind's brain model from the 1960s, discussed in more detail in the main text. It identifies the major anatomical areas of the left brain associated with classic aphasia types.

Contemporary accounts of aphasia can be compared in Davis (2000), Hagoort (2005), Hillis (2002), and Rapp (2001).

Glossary

abstract attitude: contrasts with **concrete attitude**; according to Kurt Goldstein, brain damage could produce a lack of an abstract attitude in an individual

agrammatism: a variety of impairments apparently to syntactic processing characterised by shortened sentence length, simple syntactic structures, problems with **function** words and morpho-syntactic elements but with relatively intact use of content words

agraphia or **dysgraphia**: writing disorder

alexia or **dyslexia**: reading disorder

anarthria or **dysarthria**: impairment in speech production caused by muscular paralysis or in-coordination

angular gyrus: see Figures in the Appendix

anomia: a relatively milder type of aphasia in which the speaker has particular difficulties accessing the content words they need; also, a term used more generally to describe word finding difficulties

aphemia: Broca's original term for the impairment in speech production he described

aphonia: a disorder of voice production

apoplexy: an archaic term for stroke

apraxia of speech: impairments in producing and combining speech sounds and movements in the absence of neuromuscular impairments

arcuate fasciculus: fibre tract linking Wernicke's and Broca's areas (see Figures in the Appendix)

association fibres: fibre tracts connecting cortical areas to one another

catastrophic reaction: can occur when a patient becomes overtaxed, for example when unable to solve some task, which can lead to aggression, defence, denial, fear, and other reactions

concrete attitude: contrasts with **abstract attitude**; according to Kurt Goldstein, brain damage could result in the individual employing a conceptually concrete attitude with an accompanying lack of abstract attitude

conduite d'approche: repeated phonemic paraphasias during speech where repeated attempts at a word result in closer approximations to target

connectionism and neo-connectionism: (a) referring to the connectionist models of aphasia originating with Wernicke's model; (b) referring to connectionist neural network modelling

content words: words like nouns and verbs that have a full semantic content and contrast with **function words**

copralalia: uninhibited, obscene, expletive speech automatisms associated with Giles de la Tourette's syndrome

corpus callosum: the great fibre track that connects the two hemispheres

cortical anarthria: term sometimes used for **apraxia of speech** or **aphemia**

crossed aphasia: aphasia following a right hemisphere lesion in a right-handed person

dysarthria: impairments of articulation and voice (*dysarthophonia*) caused by muscular paralysis or in-coordination

echolalia: impairment where the speaker echoes back the speech of an examiner

function words: small grammatical words that are low in semantic meaning, and contrasted with **content words**

hemispherectomy: the surgical removal of an entire hemisphere of the brain

jargon (aphasia): fluent but partly or mostly meaningless speech made up of a mixture of neologisms, paraphasias and correctly produced words

neologism: a non-word produced in speech with so few correct phones that the 'word' is unrecognisable

ontogeny: describing individual life development and contrasted with **phylogenic** development

paragraphia: paraphasia in writing

paralexia: paraphasia in reading aloud

phone: a speech sound

phoneme: an abstract phonological entity

phonetic disintegration: another term describing the speech of those with Broca's aphasia and associated with the French and French-Canadian tradition, particularly Théophile Alajouanine

phylogeny: describing evolutionary development or pedigree

pragmatics: the interface between language use and other aspects of behaviour; is concerned with the total behavioural–social context in which communication takes place

projection fibres: fibre tracts connecting subcortical parts of the brain to the cortex

tactile aphasia: an inability to name objects through touch

thematic roles: the agent or actor and the recipient of the action in the sentence

trepanations: cranial drillings

ventricle: a fluid filled cavity in the brain

word blindness: a rare disorder of reading

word-meaning deafness: a rare disorder of auditory word recognition

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