A STUDENT'S GUIDE TO COGNITIVE NEUROPSYCHOLOGY

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PRINCIPLES AND ISSUES IN COGNITIVE NEUROPSYCHOLOGY

Chapter Overview

This chapter will provide an overview of some of the most important principles that allow researchers to study individuals with brain damage and make inferences about how healthy cognitive abilities function. In addition, it is important to consider some of the 'danger zones' when conducting such research so that theories are not based on highly specific individuals who are not representative of the general population. Finally, given that the studies are conducted on either specific individuals or groups, rather than a random sample of the general population as happens with most studies, there will be an overview of the research issues that need to be considered.

Chapter Outline

INTRODUCTION

A neuropsychologist has a number of approaches that are available when either assessing a patient clinically or conducting empirical research. Whichever of these two is the major aim, he or she needs to pay careful attention to principles and decisions about participants that are relevant when working with patients with brain damage. If the patient is being used for research purposes, how representative they are of the normal population is a very important consideration since the whole point of research is to generalise to intact cognitive processes. Similarly, in trying to compare two patients, the extent to which it is possible to compare any two individuals who have physically different brains and whose brain damage could differ either only subtly or greatly needs to be given careful thought. One of the major assumptions behind the use of damaged cognitive systems to infer normal intact processing, known as **subtractivity**, is an issue that a neuropsychologist needs to consider very carefully. Similarly, the appreciation that the brain may show some changes as part of its recovery after being damaged needs to be cautiously considered. A big question when conducting empirical research will be the choice and number of patients in a study – is it better to do a very fine-grained analysis of one patient or is it better to study a group of patients with a common deficit? Finally, when the patient suffered brain damage will have a large impact on both the profile that emerges and also how the findings can be interpreted. This is because damage early in life, when the brain is still forming, can have different consequences to damage that happens to a fully formed brain.

RECREATING THE 'COGNITIVE JIGSAW'

Subtractivity

If one of the aims of psychology is to understand human behaviour functions – and within cognitive psychology, to understand mental functions such as memory or face recognition work – it is reasonable to pose the question 'Then why are you studying people who have *problems* with those very abilities?' given that the majority of us can remember or recognise faces without even thinking about it. This brings up one of the fundamental

principles of neuropsychology, but to introduce it, it is useful to think of an important quote by Craik who said, 'in any well-made machine one is ignorant of the working of most of the parts – the better they work, the less we are conscious of them… it is only a fault which draws attention to the existence of a mechanism at all' (1943, p. 84). To understand why this quote is so vital, think about how a car works. It is a highly complex system whose design is known to engineers and mechanics but unknown to me and in all likelihood to yourself too. If my car broke down, it could be for a number of reasons and since I don't understand the internal workings, I would need to go to a mechanic to have it fixed. While there, I could ask them what is wrong and they might say that the fan belt was broken. Since I don't know what a fan belt is, I could then ask for a description of what it is and how it works within my car. Now if I was unfortunate enough, my car might break down again a few months later but this time it might not be the fan belt so I would have to go back to the mechanic who would work out that it is something called the gear box; again, I could ask them to explain to me how that works. Continuing on the theme of my bad luck, I could go round the entire car with different parts malfunctioning and eventually know how all the different parts work; however, initially looking at the smoothly running piece of transport gave me very few clues about its internal intricacy.

In this analogy, it should be noted that the design of the car is something that is *already known*, and therefore for someone to understand how or why it is malfunctioning isn't a difficult process. Also, whereas the mechanic might be able to repair the problem with the car, the same is not true in neuropsychology, since brain damage is, on the whole, irreversible. Further, the human brain and cognitive system are *much* more complex than a car and we do not know the design or internal processes until we conduct rigorous experimental research; however, the analogy of the complexity of a well-made machine and studying it when it is broken applies very well to the study of the human brain.

One way of conceptualising cognitive neuropsychology is to see it as a recreation of an originally intact jigsaw that has been broken up by some form of brain damage, and with some pieces now missing. Importantly, the rest of the brain is assumed to function in exactly the same way as that of a healthy brain-intact individual. This has been termed the **transparency** or **universality** assumption by Alfonso Caramazza (1986). In the healthy adult state, all elements of the complex aspects of a particular cognitive ability (such as memory for example) function normally. This system could be seen as an intricate jigsaw. Figure 3.1a is a conceptual representation of a rather simple nine piece jigsaw that would represent memory. However, due to the very efficient functioning of this system, despite the underlying intricacy, we are unaware of all the complex mechanisms and in reality (referring to the jigsaw) we only know the general outer shape, as seen in Figure 3.1b.

Now consider for example Patient A, who is seen by a neuropsychologist (Figure 3.2a). By carefully studying this patient, the neuropsychologist maps out what he or she can and cannot do. If done properly, this process will allow a general understanding of the outer 'shape' of A's memory functioning. From this, it is possible to infer the shape of the missing piece in A (Figure 3.2b). Using this knowledge, the neuropsychologist can make a hypothesis about

the shape of one of the pieces of the normal memory system found in all healthy adults; this is the principle of subtractivity. Using the same rationale, Patient B with a *different* memory problem might also be seen by the neuropsychologist (Figure 3.3a). From studying B, it is found that the 'shape' of his memory is quite different to that of A. Again, by carefully studying this new patient, it is possible to infer his 'missing piece' (Figure 3.3b). Using this rationale, depending on first of all the existence, and second the extremely careful study of different patients, cognitive neuropsychologists aim to infer the workings of the entire system and its many sub-processes (Figure 3.1a).

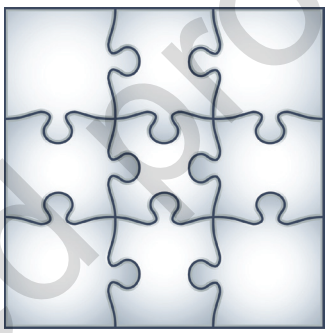

Figure 3.1a Cognitive neuropsychology: Recreating the jigsaw

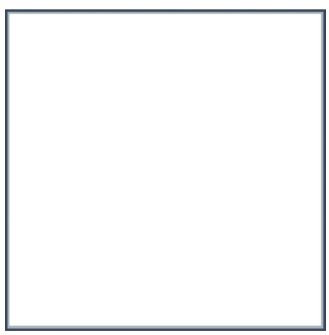

Figure 3.1b Due to the complexity of cognitive abilities, sometimes all that is known is the 'outer shape'

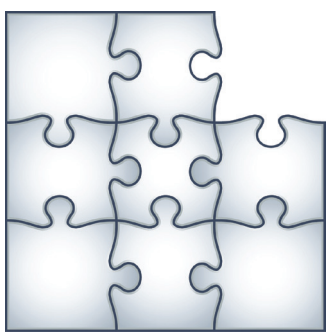

Figure 3.2a Patient A

Figure 3.2b Inferring what is missing in patient A

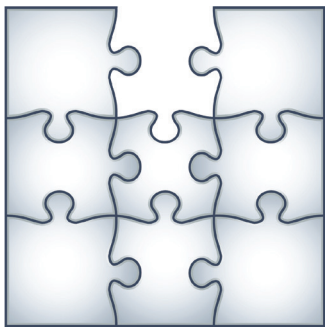

Figure 3.3a Patient B

Figure 3.3b Inferring what is missing in patient B

Associations, Dissociations and Double Dissociations

Associations

Once the neuropsychologist has used the various available standardised assessments and specially designed experimental techniques to study the deficits and intact abilities of a patient, the task now turns to making inferences from the evidence gained. How do neuropsychologists infer the existence of separate cognitive functions within the brain, and how do they then decide whether or not each of these contains separate processes? For example, is language a separate function from memory? Or within face processing are there different pathways for recognition of faces and recognition of the emotion in the face? Neuropsychologists look to see which cognitive problems tend to occur together and these are known as **associations**.

Sometimes, if a number of problems co-occur consistently, they can be grouped together as a **syndrome**. An example is **Wernicke-Korsakoff syndrome** which is a constellation of deficits that can occur following very chronic alcohol abuse (although there are other causes – see Chapter 7). The most striking sign is a dense inability to lay down

new memories. Coupled with this is a difficulty in retrieving memories laid down during earlier points in life before brain damage as well as a difficult walking gait. Between patients there can be variance in the extent of each of these individual deficits but by and large the association between them is strong, hence the syndrome label. It would be tempting to think that if the deficits in memory and walking gait occur together with such consistency following brain damage that they are all caused by one specific functional problem at the cognitive level, and this could result in localising each of these functions to the area of the lesions.

However, such a conclusion could be inaccurate and premature. For instance, the brain damage that causes the Wernicke-Korsakoff syndrome may be quite widespread affecting a number of different areas, and each of these is responsible for a different aspect of the syndrome. It may be that the initial disorder that initiates the brain damage (for example a thiamine deficiency that is caused by the poor diet of chronic alcoholics) triggers cell death in a number of different areas which in themselves are not related to one another. For this reason, **association of symptoms** needs to be interpreted with great care. McCarthy & Warrington (1990) state that some syndromes could occur simply due to the fact that separate functions may be controlled by brain areas that are very close to one another. A brain **lesion** that is large may therefore cover an area that controls a number of unrelated abilities. For example, in Gerstmann's syndrome there are four main symptoms: agraphia (deficiency in the ability to write), acalculia (difficulty in learning or comprehending mathematics), finger **agnosia** (inability to distinguish the fingers on the hand) and left-right disorientation. This does not, however, mean that these functions are all related to one another or even controlled by the same brain area but probably that a lesion has affected a number of different areas that are very close to one another and which control different functions.

Dissociations

A stronger inference about functions can be made when certain symptoms consistently occur in the *absence* of other problems. For example, it has been repeatedly observed that certain patients retain an ability to remember items for a few minutes, but lose their ability to remember the same information for longer periods (e.g. patient HM reported by Scoville & Milner, 1957). One initial conclusion could be that two types of memory are involved and that in these patients, they dissociate from one another – it is possible to lose one whilst retaining the other intact. This sort of **dissociation** has been used to support very influential early models of memory such as the **Modal Model** proposed by Atkinson & Shiffrin (1968) which has separate components for holding information for a brief amount of time, known as short-term memory (STM), or for much longer periods, known as long-term memory (LTM) (see Chapter 8).

Double Dissociations

However, even if this pattern of good STM and bad LTM is found very consistently, it could be argued that we don't actually have enough evidence to propose such a complicated system of two different forms of memory. If an easier explanation can be found it should be adopted instead – this is known as the 'law of parsimony', which means that we should look for the simplest explanation for an observation that we are trying to understand. For example, 'unidentified flying object' in the sky could well be an alien spaceship but it is more likely to be a simple weather balloon! Teuber (1955) argued that the neuropsychologist should consider the question: What if one of these two abilities was simply *easier* than the other? The pattern of observed behaviour could then be explained without suggesting a complicated multi-component system.

For instance, just hypothetically, suppose that there was just *one* memory system, and in that system the more 'effort' (conscious or unconscious) you put into retaining information, the more likely it was that you would retain it for longer periods. So for example, more 'effort' (such as more concentration or more efficient neural processing) is required to retain information for a few hours than it is to do so for just a few seconds or a few minutes (see Figure 3.4a). Now if this system was damaged by brain injury, perhaps rather than losing the entire memory system, only the ability to hold onto information for a long time, i.e. the 'difficult-to-hold-onto' memory could be affected. This does not affect 'easy-to-hold-onto' memory since that only requires a bit of effort/processing and therefore it could be possible to see the pattern described in patients such as Henry Molaison, who during his life for the sake of anonymity was known in the literature as HM (see Figure 3.4b). This situation would be similar to having a heavy illness for a few weeks which would generally reduce energy levels; following recovery, easy exercise such as walking would be possible but more strenuous activities such as running or swimming would prove more challenging.

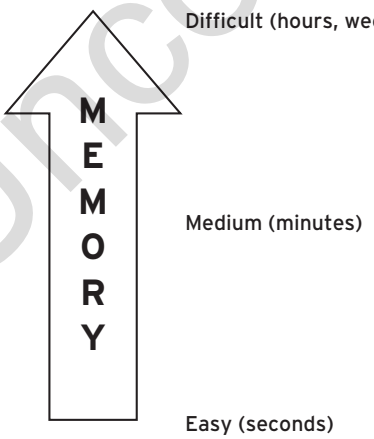

Figure 3.4a A hypothetical unitary memory system with different degrees of difficulty as the hierarchy is ascended

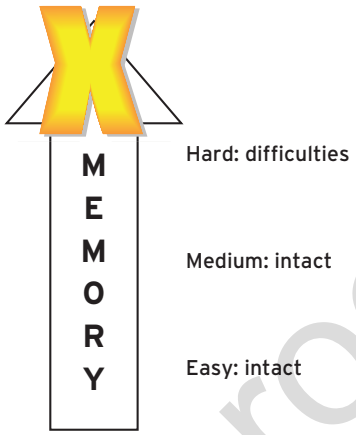

Figure 3.4b Damage to the hypothetical unitary system is most likely to affect the most difficult aspects of memory. This *could* explain cases such as HM who have an inability to remember information that happened more than a few minutes ago

The scientific rationale is that if a simpler explanation can suffice for all available data, it is not *yet* possible to suggest a more complex system – basically, nature and evolution prefers simplicity! Therefore, the evidence from amnesics such as HM does not *yet* allow us to suggest multiple forms of memory.

What would the prediction be if this unitary hierarchical system was damaged at its midpoint? An impairment at the midpoint would allow easy levels of processing but would affect medium levels of difficulty (see Figure 3.4c). In our memory example, this would mean that a patient should be able to hold onto information for a few seconds but beyond that should have problems. Additionally, in such a hierarchical system, the patient should have problems in retaining anything at the 'hard' end of the spectrum, so the prediction would be that a patient who has a problem with holding onto information for anything more than a few seconds should *also* have problems holding onto information for any long periods of time such as days, weeks and years.

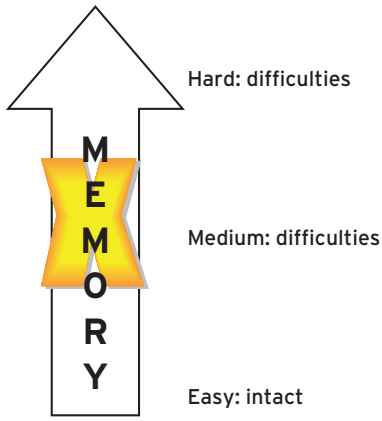

Figure 3.4c If the hypothetical unitary system is damaged in its middle portion the prediction would be that both 'medium' and 'hard' levels of memory would prove difficult

However, what if a patient was found who cannot repeat back a list of six single digit numbers after ten seconds and yet has no problems with remembering information from a few weeks ago? This is the pattern depicted in Figure 3.4d and has been found in some patients. Shallice & Warrington (1970) documented the case of patient KF who, following a motorcycle accident that caused brain damage, had problems with certain aspects of STM but no problems with his LTM. Can such a pattern be explained by our simple 'one-memory' model? The answer is 'no', and the result of such a finding would be that we would have to abandon the concept of a single unitary hierarchical model in favour of at least two separate memory systems, one which primarily caters for the medium-difficulty level whilst the other caters for the harder level of difficulty.

The reverse pattern between two patients, where HM had intact STM but impaired LTM, and KF had impaired STM but intact LTM, is known as a **double dissociation**. This type of finding is extremely useful for neuropsychologists because, as it cannot be explained in the one-system model that varies along 'ease' (Figure 3.4d), it seems to imply the existence of two separate systems (thus allowing both patterns of impairment to occur). Such a double dissociation allows researchers to then conclude that (at least to some degree) STM and LTM are separate processes. The existence of further double dissociations then allows for further fractionation of memory (see Chapter 8: Memory).

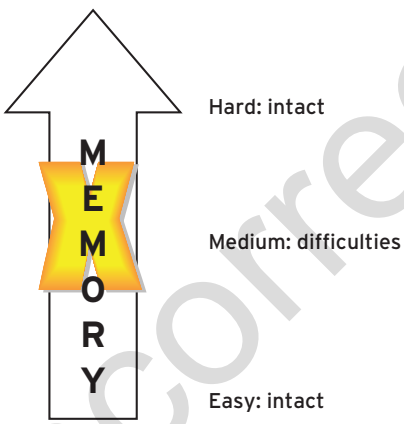

Figure 3.4d If the hypothetical unitary system is damaged in its middle portion resulting in problems with memory for information heard a few minutes ago, it is impossible to explain how a patient such as KF can still be able to remember what happened an hour, a week or many months ago

Other very clear examples of double dissociations in cognitive neuropsychology occur in face processing, between recognition of someone's identity and the emotion they are expressing (e.g. Adolphs et al., 1994; Jansari et al., 2015; see Chapter 7), and in reading, between phonological and **surface dyslexia** (e.g. Coltheart et al, 2001; see Chapter 10).

Generally speaking, if applied appropriately, double dissociations can be extremely useful in the development of cognitive models of normal functions. Researchers do not go looking for double dissociations since they are not common, but noticing patterns that may dissociate like this is useful as part of the 'tool kit'. For example, we (Jansari

et al., 2015; see Chapter 7) noticed that our patient's face-recognition problem was almost exactly the reverse of that of a patient with object recognition difficulties studied by Moscovitch et al. (1997). Therefore, we developed a study using their paradigm to demonstrate a double dissociation between the two patients. Following the discovery of a double dissociation, if specific anatomical differences exist between the two patients who demonstrate a double dissociation, it is possible to make claims about the localisation of particular functions. However, even in the absence of anatomical information, the functional significance is still very important.

Caution Regarding Double Dissociations

Double dissociations are a very powerful tool in cognitive neuropsychology but a number of authors suggest caution when interpreting such findings to conclude either functional or physical separation of processes (e.g. Young et al., 1993; Shallice, 1986; Law & Or, 2001). The issues revolve around how data from different studies is compared, how to judge a deficit and how to evaluate normality.

Comparison of Data from Different Studies

Patients who demonstrate double dissociations from one another are often studied in different laboratories by different researchers using methods that are not standardised. As a result, different stimuli may have been used to demonstrate the pattern of problems exhibited in the two patients. A classic example is in the field of object recognition where some researchers (e.g. Warrington & Shallice, 1984) have reported patients who have much greater problems visually recognising living things than non-living things whilst others (e.g. Sacchett & Humphreys, 1992) have reported the reverse case (see Chapter 6). This has led many (e.g. McCarthy & Warrington, 1990) to argue that the system responsible for storing conceptual knowledge (known as the **semantic system**) consists of at least two separate systems, split along categorical lines with a major sub-division between living and non-living objects.

However, many critics (e.g. Gaffan & Heywood, 1993) have argued that the double dissociation may be an artefact of the different studies using different materials for testing that were not matched appropriately. Since Warrington & Shallice will have used one set of stimuli to demonstrate that their patients had difficulties in recognising living things but not in recognising non-living things, while Sacchett & Humphreys used different stimuli to demonstrate the reverse pattern in their patient, a direct comparison is difficult. For some scientists, this brings into question the inference of a double dissociation.

However, there have also been studies reported that counter this concern. Hillis & Caramazza (1991) have shown a clear double dissociation between two patients, tested at similar times, in the same laboratory and on the same material.

Judgement of Deficits and Normality

How a deficit, dissociation or even 'normal performance' in a patient is judged can sometimes be a subjective matter. Law & Or (2001), for example, have shown that depending on whether or not appropriate data from healthy normal controls is used, it is possible to seriously misinterpret data. For example, imagine a patient who performs sub-normally on two tasks, where the average score for health individuals is 9 and the normal limits are between 7.5 and 10; however, the patient is better on task A than task B (see Figures 3.5a and 3.5b). If a second patient is found who also performs sub-normally on both tasks but shows the reverse pattern (i.e. better performance on task B than A), can these two patterns be interpreted as a double dissociation?

Some argue that it is necessary for *each patient* to be relatively intact, and to perform within the normal limits on one of the two functions to justify the use of the data from the two patients to claim a double dissociation. Shallice (1986) refers to a situation where at least one of the abilities is performed within normal limits as a 'classic dissociation'. In the hypothetical example above, since neither patient shows intact performance on at least one of the two tasks, some would suggest that the evidence is not strong enough for a double dissociation. However, if one patient scores 9 on task A and the other scores 8 on task B, then both meet Shallice's criterion for their performance on the two tasks to show a double dissociation (see Figure 3.5b). As an example, HM performed fully within the normal range for short-term memory tasks but extremely poorly on long-term memory tasks while KF was the exact opposite.

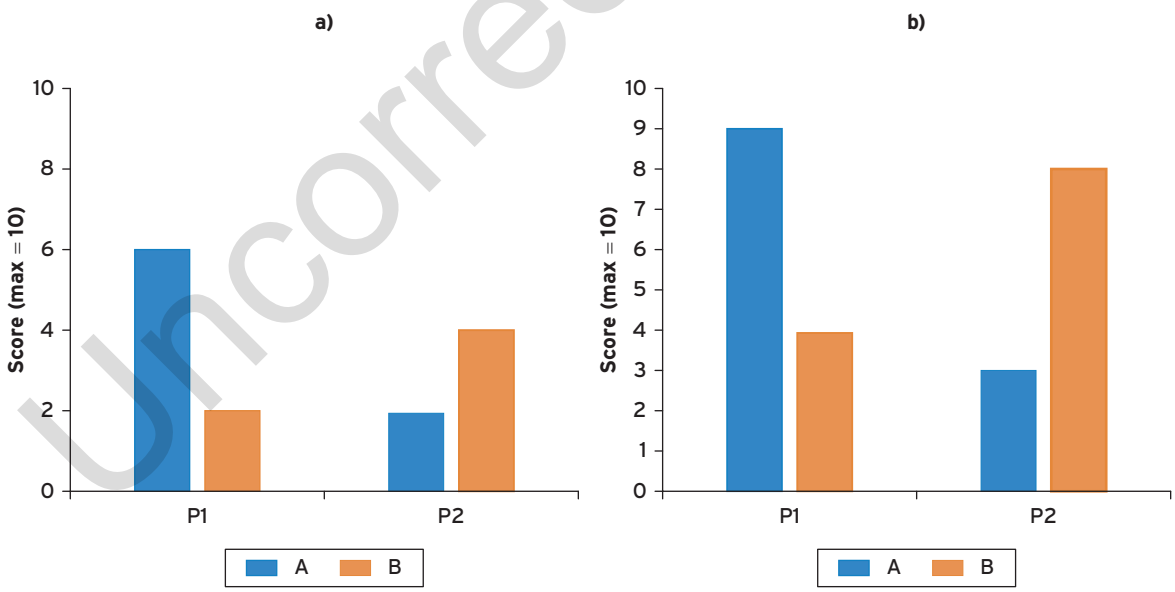

Figure 3.5 a) A 'weak double dissociation' with patients 1 and 2 both performing sub-normally on tasks A and B; b) A 'classic double dissociation' whereby both patients perform within normal limits on at least one of the two tasks and poorly on the second task

What Does 'Normal' Mean?

When we say 'normal' in neuropsychology, what do we mean? 'Normal' performance on a task does not necessarily mean normal cognitive functioning, since a patient may be using a compensatory strategy not found in the intact cognitive system (see below). For example, Humphreys & Riddoch's (2013 patient HJA seemed to be able to copy line drawings well, implying an intact ability to perceive the drawings properly. However, detailed analysis of his *strategy* when drawing showed a very laborious piecemeal line-by-line drawing, concentrating on different parts of the object selectively, rather than what one might expect if HJA perceived the object as a complete whole. Similarly, Jansari et al. (2015) examined a patient with face-recognition difficulties (see Chapter 7) but who was still able to recognise some famous faces. However, by not relying simply on his accuracy at recognising a famous face, but looking at how long it took him to name the face, they were able to show that the patient's 'recognition' was far from normal. It is therefore important to ensure that a patient is tested in a variety of ways to make sure that what seems 'within normal limits' is in fact 'normal'.

The above discussion shows that it is necessary to observe caution when interpreting dissociations and double dissociations. Since these interpretations can have a large impact on theories, it is important that patients are tested as rigorously as possible. Finally, as a cautionary note, Ellis and Young say that it 'would be unwise to regard the search for double dissociations as some sort of royal road to understanding the structure of the mind' (1988, p. 5). In other words, double dissociations are *one* of a set of tools but not the *only* tool that can be used.

Overall, it is clear that it is necessary to carefully scrutinise the evidence regarding the types of patients, the stimuli that are used and the judgements of the level to which an ability is intact or impaired to argue for double dissociations between functions. Therefore, anyone entering the field needs to ask themselves questions such as 'Although on the surface, two patients seem to be performing in quite opposite directions, were their abilities tested in sufficiently similar ways for the double dissociation to be convincing?'

General Caveats When Intrepreting from Damaged Brains

The Alien within Us

One of the major assumptions made in neuropsychology to allow the principle of subtractivity to be valid is that before brain damage, the patient's cognitive system worked in exactly the same way as any other person's. If this is the case, then it may be possible to extrapolate from this one person's damaged system to how the normal intact system functions. However, what if the assumption is invalid and, in fact, even before brain damage the patient had an unusual cognitive system, quite unlike the rest of the population; their system would be somewhat 'alien' to the normal one. The consequence of this would be that any inferences that are made from their damaged system for the intact system would

not satisfy one of the goals of neuropsychology which is to investigate the functioning of the normal intact system.

This of course, is a serious issue, especially since it is impossible to go back in time and check that the patient's intact system was indeed 'normal' before brain damage. This potential problem is particularly the case when there is an underlying abnormality. For example, if a patient complains of memory problems but it is discovered that they have suffered from epilepsy for 20 years, then it is important to consider that as a result of the physical abnormality that is the cause of the epilepsy, the patient's cognitive system has not developed normally over the last two decades, or they have developed 'compensatory strategies' that are not found in the normal state.

A famous example is that of Kim Peek, who was the inspiration for the Hollywood film *Rain Man*, which was about an autistic savant with an exceptional memory for numbers. Although Kim was seen as autistic and some of his cognitive abilities were indeed stunning, his brain had a large number of abnormalities which meant that trying to extrapolate from his abilities to 'normal' functions would have been inappropriate. Neuropsychologists are very aware of this potential problem and it is for this reason that each research study needs to be extremely detailed and rigorous and is usually followed up by replication in other patients.

COMPARING TWO INDIVIDUAL BRAINS

One of the basic ways that scientific research moves forward is by the replication and extension of studies that have revealed important findings. In the case of cognitive neuropsychology, this is done by testing out theories suggested from one patient on another patient who is exhibiting the same or similar problems. If one of the aims is to map function onto physical brain areas (see Chapters 1 and 2) then comparison of the two patients needs to be done very carefully. Even if two patients show similar behavioural profiles, (for example the same type of reading problem) does that necessarily mean that their brains are the same and show the same size and extent of brain lesions? Even small differences in sizes of lesions can have a major impact on differences that may be too subtle to see at a superficial level but which may nonetheless have a great impact on task performance.

Bartley et al. (1997) carried out a study comparing the brains of identical twins with those of non-identical twins. Whilst finding that the brains of identical twins were much more similar in shape than those of non-identical pairs, they also found that the shapes of the individual gyri differed slightly between identical twins. Figure 3.6 shows three-dimensional images of my brain (AJ1) and that of my identical twin (AJ2). Without being a specialist in brain anatomy, you can see that although the two brains look similar (and more like one another than like a stranger who is the same age as us), our brains are far from *identical*. If this is the case for two genetically identical people, then two unrelated

people are likely to have even more differences in their brains before they become damaged. So for example, if hypothetically a lesion of one cubic centimetre was made on two brains that are not identical, the particular brain structures destroyed in them would differ at least slightly. The impact that this is going to have on the cognitive problems exhibited can be either subtle or substantial.

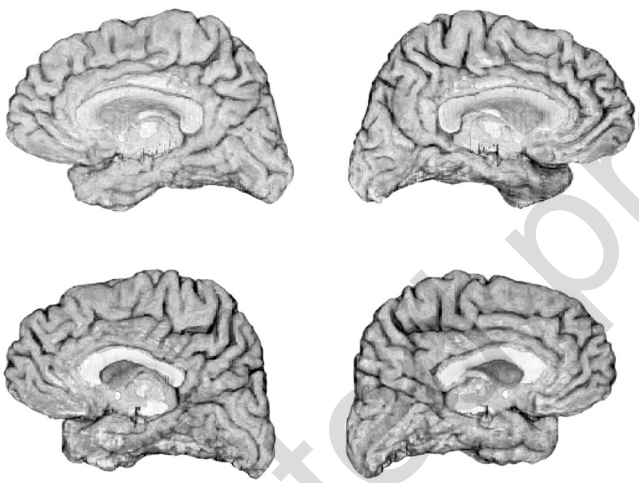

Figure 3.6 Brains of identical twins: my brain (bottom) and my identical twin's brain (top)

Even if all human brains were identical, natural brain damage or even neurosurgery does not result in 'clean lesions' which are restricted to distinct well-defined regions. For example, a blood vessel bursting may result in widespread damage affecting a number of different areas. If we think about research in language disorders as an example, researchers may be interested in production of spoken language which, as described above, is controlled by Broca's area in the frontal lobes. They could therefore study a patient who has had a stroke in this area. Although there may be a lesion in Broca's area, there may also be damage in adjacent cortical areas. The result is that as well as the difficulty in producing coherent speech, the patient may have a number of other cognitive problems either related to or unrelated to language. Therefore, the patient's speech problems could be because of damage to Broca's area only, or due to damage to the neighbouring areas only (because that interrupts the flow of information to Broca's area) or due to a combination of both. If this sort of issue can cause complications when studying one damaged brain, it is therefore not surprising that comparing two patients who may have differing damage that extends beyond Broca's area can greatly compound the problem. As a result, neuropsychologists need to be very careful and need take into consideration *all* of the damage suffered by a patient or patients rather than just the areas of interest to their research. Again, this requires very thorough analysis of the patient's intact and impaired abilities.

Plasticity

The logic behind the subtractivity argument has been applied very successfully to create or support complex models of normal cognition. However, it is important to note that the logic needs to be applied with caution under certain circumstances due to the **plasticity** of the brain. Plasticity refers to the ability of the brain to repair itself both at the neuronal and even cognitive level. Rose & Johnson state that 'far from being fixed, unchangeable and static, we now know that the brain is a dynamic and interactive organ, constantly changing in terms of cellular activity, neural circuitry and transmitter chemistry in response to demands placed upon it' (1996: p. 14). For example, it is known from work on animal brains that the brain can recreate neurons lost due to lesions. Under some circumstances, the brain can also 'move' the function from one area of the brain to another if there is damage in the original area where that function is usually found. For example, it is known in babies that suffer severe strokes in the left hemisphere (which is normally where language is processed) that language can sometimes develop in the right hemisphere (Bates & Roe, 2001).

In the adult brain, this near-complete switch of language to the right hemisphere is not found. However, if even *some* cognitive functions can move to another intact part of the brain, then the above assumption of subtractivity cannot be applied to use findings from such patients to infer the workings of the *normal* cognitive system. This is because the assumption of subtractivity is based on the whole premise of *removing* sub-components rather than *moving* or *creating* new ones, since under normal circumstances this does not occur.

For example, it is possible to use the assumptions of associations and dissociations to work out a simplistic model of the cognitive system that has modules for problem solving, object recognition, face recognition, reading and memory (see Figure 3.7a).

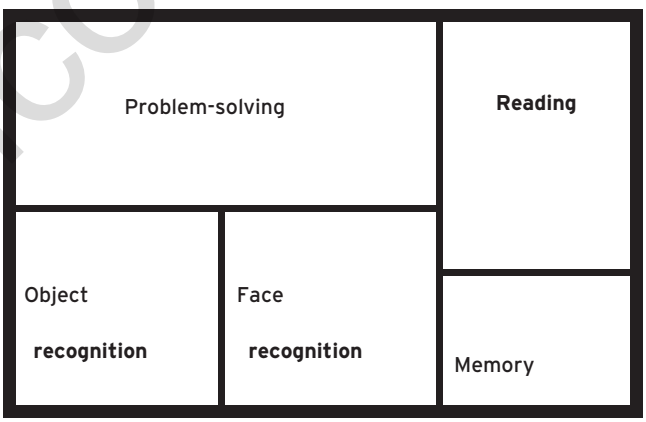

Figure 3.7a Hypothetical structure of the cognitive system sub-divided into major modules

Using the subtractivity assumption, it is possible to study healthy controls and patients who have suffered damage to the left hemisphere which results in different types of dyslexia (e.g. surface and phonological dyslexia) to sub-divide the overall reading system into a number of sub-sections such as A–H (see Figure 3.7b).

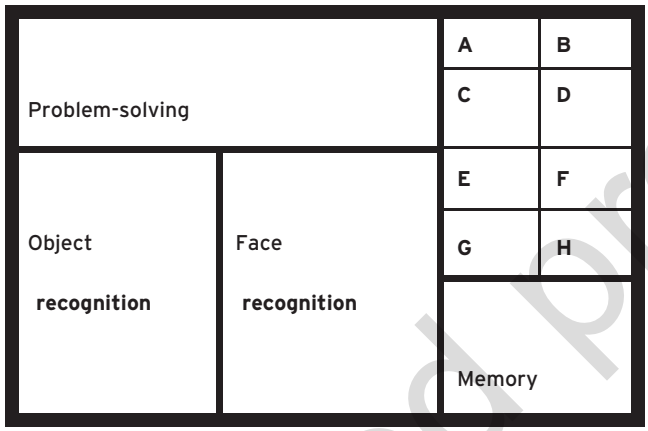

Figure 3.7b Sub-division of the reading module into a number of separate components

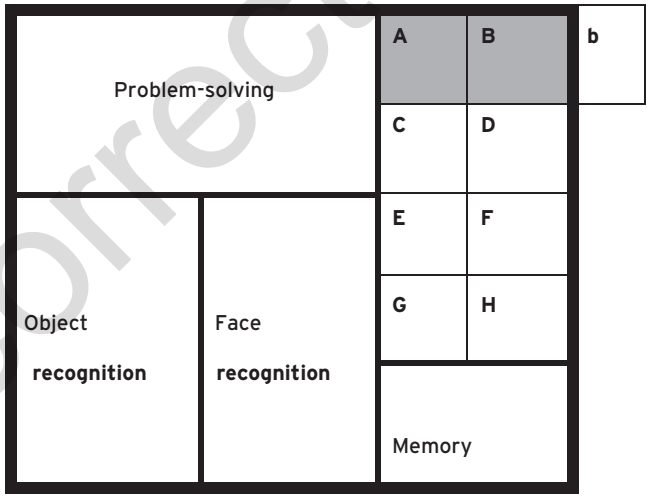

Figure 3.7c Hypothetical use of a 'silent' reading system 'b' within the right hemisphere following damage to the parts of the brain dealing with reading components A and B

In deep dyslexia, however, it is suggested that following more extensive damage to this system which is thought to be largely in the left hemisphere (e.g. to A and B in Figure 3.7b), some aspects of reading occur using a 'silent' reading system (e.g. sub-component b) in the intact right hemisphere which ordinarily is not used (Coltheart

et al., 1987; see Figure 3.7c). It is beyond the scope of this chapter to give a full account of the theory (see Chapter 10: Language) but it is claimed that there is a complex interaction between this right hemisphere reading system and those aspects of reading that are still intact in the left hemisphere. This interaction is thought to be sub-optimal resulting in a cluster of consistent reading errors in deep dyslexic patients which include visual errors (e.g. reading WHILE as /white/), semantic errors (e.g. reading the visual input SWORD as /dagger/) and difficulties in reading words that are more 'abstract' than more visualisable 'concrete' words (e.g. having more problems reading JUSTICE than TABLE). This theory of deep dyslexia involving a right hemisphere reading system is one that is still under debate, but if it is even somewhat accurate, how representative of the normal reading system is it? Module 'b' does not tell us anything about the normal reading system that may be made up of modules A–H only. To date, there is very little evidence that the right hemisphere becomes involved in normal intact reading. As a result, because of the possibility of plasticity resulting in the formation of new modules, researchers need to apply caution when interpreting findings.

RESEARCH DECISIONS

Single Case versus Group Studies

In standard experimental psychology, a large number of participants are studied to ensure generalisability, and an estimate of 'sampling error' is taken to extrapolate from the study participants to the general population. However, in neuropsychological research, this cannot always be possible since it is sometimes impossible to find more than a few patients with a rare condition. Even if it was possible to find a group of such patients, from the discussion on comparing two individual brains above, it is obvious that problems can arise due to the heterogeneity of brain damage across the group. The result is that a neuropsychologist is faced with the decision on whether to study a single patient in depth or to use a group of patients all with the same cognitive deficit. It is best to group the arguments by arguments for and against each approach and then look at the criteria that a researcher uses in their decision.

Arguments for Single Cases / against Group Studies

Grouping of subjects may be legitimate in standard cognitive research where the assumption is that all subjects start off roughly equivalent, but this is not possible in neuropsychology for a range of reasons. One of the most important reasons is that at the start of a particular branch of research, almost by definition, most research begins with single cases. Paul Broca's seminal work on patient Tan was monumental in terms of showing how selective brain damage can affect very specific cognitive difficulties, and this started

an explosion of replications by himself and other neurologists at the time. Similarly, Brenda Milner's work on patient HM (see Chapter 8: Memory) has been so important in the field of memory that the first paper (Scoville & Milner, 1957) has been cited a staggering 2500 times in other scientific articles; further, the insights on different aspects of this patient's memory impairments and preserved abilities set the agenda for more than half a century's work.

Tan and HM had cognitive disorders (aphasia and **amnesia** respectively), that can be caused by a number of different neurological disorders such as strokes, viruses and interruption of blood to the brain. Therefore, they are not uncommon disorders, with the result that it is possible to conduct group studies. However, there are some conditions such as prosopagnosia or **Capgras syndrome** (see Chapter 7: Face Processing) which are extremely rare. As a result, it is impossible to gather a large group of patients with these conditions making a group study unfeasible; single case studies are therefore the best choice. Sometimes, a patient is seen who because of their particular circumstances is rare even if the condition itself might be relatively common. For example, Jansari et al. (2010) studied a patient with a particular form of accelerated forgetting (see Chapter 8: Memory) which up to that point had been documented only in patients who had temporal lobe epilepsy. A 'confound' in all of these previous studies is that a number of the drugs that are used to control some types of epilepsy have as a side effect memory problems. Therefore, in studying the particular accelerated forgetting in this group, it is unclear whether it is their condition (the epilepsy) or their medication that is causing the memory problem. However, Jansari et al.'s patient RY had no clinical diagnosis at the time that the study was initiated, therefore his accelerated forgetting could not have been the side effect of medication. Indeed, as a result of the first set of studies with RY, it was discovered that he did indeed have a sub-clinical epilepsy that had been difficult to detect previously. Following the diagnosis, he was prescribed anti-epileptic medication and so a second study was conducted to look at whether this changed his memory performance. It was found that it didn't make any difference to his rate of forgetting; if the patients' medication in previous studies had been the *cause* of their forgetting, then RY would have been expected to get worse with the medication. Therefore, although in fact RY was not rare because he ultimately was in a relatively common group (patients with temporal lobe epilepsy), the timing of the studies before he had commenced medication made him rare.

Even if it is possible to gather a sizeable number of participants with the same cognitive disorder (for example, aphasia), the brain damage will vary across the group. For a start, as discussed above, before neurological damage the brain structure of two individuals will be different; added to this, even if all of them had a stroke in the same cerebral artery, the extent of damage will have varied across the individuals. As a result, there will be variance in the types and extent of lesions. Given the heterogeneity among the group caused by differing types of brain damage, there may be important subtle differences in task performance between subjects within the same group. In standard research methodology (such as trying to work out whether females are better on a linguistic task than

males), differences *between participants in a group* should be kept to a minimum to try to identify the differences *between the two groups*. Due to statistical principles, this allows averaging of the groups (e.g. the average female compared to the average male). However, in a neuropsychological study, this sort of averaging could mask (hide) subtle differences between two patients within the same group. Therefore, although a group of patients with aphasia performs differently to a control healthy group on a language-based task, there might be very subtle differences between two of the patients that could have very important implications for theories of language processing. Simply looking at the *average* of the group will hide these potentially important differences. As will be seen in some of the chapters (Chapter 4: Neglect; Chapter 5: Apraxia; Chapter 6: Object Recognition; and Chapter 7: Face Processing), it is sometimes the differences between individual patients that are the most revealing for theory building.

A related issue is that grouping subjects according to a syndrome (a cluster of symptoms) such as Broca's versus Wernicke's aphasics, which was the standard method in the early days of aphasia research (see Chapter 10) can be misleading. This is because although the patients may *share* some symptoms, the averaging and treatment of the individuals as a homogenous group can overlook many *differences* which may be the most revealing. The research in the 1940s and 1950s on aphasia suffered because of this, and eventually researchers had to find more fine-grained groupings to be able to explore the language difficulties more successfully.

Finally, there are those who say that it doesn't make sense to study many people if one very compelling case demonstrates an important point and might be the exception to the rule. So if we assume that the cognitive architecture is generally the same across us all, unless we *know* that someone is an 'alien among us', generalising from the one patient should suffice. Put simply, if I showed you one flying pig, how many more pigs would I need to show you to demonstrate that pigs could fly?! *Nature Neuroscience* uses the metaphor of finding a talking pig when discussing how important a case study needs to be in order to be accepted (cited in Medina & Fischer-Baum, 2017, p. 445). It should be remembered that the field of cognitive neuropsychology effectively traces its roots to the documentation of a single patient, Tan, while the work on the amnesic patient HM has been *the* bedrock for so many discoveries. As talking pigs go, they told us a lot…

Arguments against Single Case Studies / for Group Studies

As discussed above, due to the possibility of a patient being an 'alien among us' with a brain architecture that was unusual even before brain damage, there is a question of generalisability from a single individual to normal cognition. For example, the brains of patients with temporal lobe epilepsy may have developed non-conventionally due to their epilepsy and may therefore have created compensatory strategies not found in the intact brain.

In terms of resources, single case studies can take a substantial amount of time if the researchers want to look in great detail at a patient's abilities. Often experiments within a single case study are developed based on the findings from the initial experiments with the patient and so there is a stepwise evolving process in exploring particular issues. This method of developing a series of separate experiments – testing the patient and matched controls, and then based on these findings, spending time developing the next set of experiments – can mean that it can take a long time to get a coherent set of data. For example, it took us four years to collect the data for the nine experiments on our patient with prosopagnosia (Jansari et al., 2015: see Chapter 7); this makes one wonder how long it took Moscovitch et al. (1997) to conduct 19 experiments on their patient and matched controls! Further, given that one individual is the focus of all of this work, there is also the risk that if something happens to the patient, then the research can no longer continue; in a group study, it would be possible to replace a patient who was no longer available for study.

A possible concern with single case studies that can be avoided with group studies is that patient selection can sometimes be biased towards patients who fit into a particular theory – the result being that what is known as **theory-driven research** can be rampant because researchers are only studying the patients whose data goes along with their theoretical framework! Therefore sometimes researchers find it very difficult to replicate the findings from other published research studies due to the original patient having been specifically chosen to prove a theory; as discussed in Chapter 2, the replication crisis is prevalent in psychology and, sometimes, single case research can contribute to that. With a group study where there are objective inclusion criteria for being part of the research (just as would occur for other areas of psychology), there is less chance of only selecting patients that fit one's theories.

How Do You Decide between Single and Group Studies?

With such a diversity of issues, how should a neuropsychologist decide which methodology to choose? The first consideration depends on resources. Some research institutions (for example hospitals interested in research) have access to large groups of patients, whilst others only have limited numbers. A second consideration is the particular field of research, since patients with some types of deficit are extremely rare, while those with other problems are much more available. For example, Capgras syndrome (see Chapter 7), which results in a patient with a face-recognition problem feeling that his or her family have been replaced by imposters (e.g. Alexander et al., 1979), is *so* rare that it would be very difficult to find two patients to study at the same time. In contrast, Wernicke-Korsakoff syndrome, which is a cluster of deficits (the hallmark symptom being classical amnesia), is much more common, meaning that group studies are relatively feasible (e.g. Albert et al., 1979; Kopelman, 1989). Finally, the strictness of methodology and the vigour that is applied by researchers play a large part, because if all the caveats of studying single

cases are borne in mind, and a patient is also tested very thoroughly, then a single case study can be the most informative approach available.

Recently, a new approach has been to combine the strengths of the two different approaches by studying 'case series'. In these designs, a group of patients or special participants is studied but their performance is analysed individually in addition to standard group analysis. This way, the richness of data that can come from studying an individual in depth is complemented by making a stronger statement about a group of individuals. For example, in the early days of research on **synaesthesia** (see Chapter 12), a lot of the research was on single cases, but there was growing understanding regarding the heterogeneity between individual **synaesthetes**. Therefore, Jansari et al. (2006) conducted a case series of three synaesthetes that demonstrated that each of them *did* in fact see 'coloured numbers' in their mind's eye when performing simple mathematical calculations, but also that the three of them differed from one another with variations in whether they were looking at the calculations visually or hearing them. This way, they were able to use the strength of case studies while avoiding some of the limitations by demonstrating that this was not occurring in only isolated individuals.

Overall, most fields have seen an evolution over time. For example, Broca's single case approach eventually led to group studies by many aphasiologists, and then more recently, respecting the subtle differences between individual patients, case series have become more popular. Therefore, it's not a case of 'Which is better?', but more so 'Which is better for this particular stage of this area of research?', 'How available are the types of research participants?' and 'What resources are available?'; it's perfectly possible to contribute to a research area with a single case study in a field where group studies are the norm as long as the research is conducted carefully.

DEVELOPMENTAL VERSUS ACQUIRED NEUROPSYCHOLOGICAL DISORDERS

Brain damage can occur at any time in someone's life – it could happen pre-natally in the womb, post-natally as a result of complications or due to a premature birth, during childhood due to accidents or during adulthood. To further complicate matters, the cognitive system may have problems not due to brain damage but because of a genetically inherited disorder.

A difficulty due to potentially genetic reasons is known as a developmental disorder, whereas one caused by physical brain damage is referred to as an **acquired disorder**. Therefore, a child who has always had a reading problem of a particular type could be classified as having developmental dyslexia, whereas someone who could read before brain damage but then shows impairment following a stroke would be classed as having **acquired dyslexia**.

Historically, more research has been carried out on acquired disorders than developmental ones for a number of reasons. One of these is that within cognitive neuropsychology, an attempt is being made to try to infer how the normal system works by looking at one that used to work normally but has now been damaged – this is the principle of subtractivity described above. An adult who used to be able to read normally who suffers brain damage that affects their reading has acquired dyslexia, and it is possible to use the subtractivity principle to hypothesise about the missing components as described above. However, since a developmental dyslexic's reading system may not have formed normally from the outset, making conclusions from examples of it about how an adult reading system works can be difficult.

Needless to say, the study of developmental disorders can still be extremely useful for understanding the problems that certain groups of young children and adolescents face in an attempt to find ways to help them; this is the field of developmental neuropsychology. In recent years, for example, dyslexia, autism and attention deficit hyperactivity disorder (ADHD) have been taken much more seriously such that in some school systems (for example in the UK), children who have any of these conditions are given access to support them with the difficulties they experience As knowledge grows about how to study such groups, this area will develop greatly.

Due to the issue of plasticity mentioned above, *when* an individual suffers their brain damage has important implications both theoretically and clinically. If damage occurs very early in life, then some level of neuronal repair and/or a certain amount of reorganisation may be possible. Damage later in life makes this unlikely, meaning that the same type of brain damage in childhood and adulthood can have very different results in the permanent problems that the patient may be left with. For example, some babies who are born very prematurely and whose lungs are not yet strong enough to circulate blood around the body sufficiently suffer damage to the brain because it is starved of oxygen for a brief but crucial period of time. In one such baby, the only damage caused was in a very discrete but important part of the **hippocampus**, an area that is vital for memory (Vargha-Khadem et al., 1997). Despite this, the baby grew up relatively normally and it was only at around the age of six that it was noticed that he had significant memory problems. Although he has quite severe problems with his memory, he is still able to have a job and look after himself to a certain degree. Adults who suffer the same brain damage to the hippocampus, however, have much greater difficulty in learning to cope because it is impossible for the brain to 'rewire' at that age and difficult to develop new coping strategies. For example, two very well-documented patients with severe amnesia, HM (Corkin, 2002) in North America and CW (Wilson & Wearing, 1995) in Britain, both have to live in care homes because of the impossibility of them living independent lives.

Chapter Summary

- If one cognitive ability is impaired in a patient (e.g. language) whilst leaving another intact (e.g. memory), these two abilities are said to dissociate and are governed by separate cognitive processes.
- It is possible to explain some patterns of observed behaviour with unitary systems rather than having to invoke two separate processes. In this simpler system, the more effortful aspects of processing (e.g. remembering events from many years ago) can be impaired whilst leaving more basic aspects (e.g. retaining five digits in memory for a minute) intact. If such an explanation is possible, researchers should adopt it.
- However, if two patients are found, one of whom is impaired on an ability A (e.g. ability to recognise emotion shown in a face) but has no problems with ability B (e.g. ability to recognise people) whilst another patient shows the reverse pattern (impaired on B but intact on A), these two abilities are said to show a double dissociation. A finding like this can be very strong evidence for the separation of cognitive processing.
- There are some caveats about the use of double dissociations that need to be borne in mind when interpreting data.
- Researchers need to be aware of the possibility that a patient may *not* be representative of the general population, and therefore to make statements about normal cognition it is important to replicate findings.
- Due to physical differences between any two brains and the fact that brain damage can often be diffuse rather than neatly localised, it is important to compare data from patients systematically both at the behavioural and neurological level.
- The assumption of subtractivity centres around the idea that what is observed in a patient is the effect of a whole cognitive system which has had certain modules (e.g. long-term memory) impaired or removed whilst leaving the rest of the system intact. By applying this assumption systematically and with care, neuropsychologists aim to construct an understanding of the entire intact system.
- Researchers need to be aware of the possibility of plasticity both neurally (regrowth of brain tissue if damage occurs *very* early in life) and cognitively ('movement' or adaptation of a function) following brain trauma.
- Due to the confounding and sometimes opposing factors of representativeness of single individuals and the heterogeneity of any two brains (as well as the extent of brain damage), whether to study single individuals or groups of patients is a very important research issue. There is no correct answer and there are many factors which determine the choice of methodology.
- Since brain damage can occur at any point in life and brain abnormalities can occur due to genetic factors as well, there is a difference between developmental disorders and those that are acquired following trauma. Applicability of methodologies and interpretation of results will therefore be determined by whether the behavioural problem is a developmental or acquired one.

Important Research Study

KF (Shallice & Warrington, 1970)

KF was involved in a motorcycle accident at the age of 19 and the resulting brain damage brought on epilepsy which he suffered for many years. This study was extremely important since Tim Shallice and Elizabeth Warrington were able to demonstrate that KF suffered from a very selective deficit in *only* his verbal STM; his STM for visual materials and his LTM were completely unaffected. This finding was seismic for a number of reasons. First, it demonstrated that it was possible to have impairments within STM but still transfer information into LTM; this is something that went against the way that the Modal Model was formulated. Second, it demonstrated that even the STM of the Modal Model was simplistic since there was no differentiation between different types of information, and yet the fact that KF could remember *visual* information within his STM but not *verbal* suggested that this part of memory was more complex than simply one store. Finally, this specific difference was what led to the development of the Baddeley & Hitch Working Memory Model which was able to explain how it was possible to have an impaired ability to remember words or numbers while still being able to remember visual information.

Important Researcher

Alfonzo Caramazza

Alfonso Caramazza is an Italian neuropsychologist and neuroscientist based at Harvard University in the United States. His main areas of interest are the nature of language processing and conceptual representations in the brain. Besides his theoretical contributions in these areas, one of Alfonso's most important contributions is his defence of the single case study. Since in the rest of psychology, groups are usually used, the focus on a single individual seemed 'wrong' by many. In addition to this, with the growth of neuroimaging in the 1980s, many researchers went to 'the new kid on the block', abandoning work on neuropsychological patients. Therefore, Alfonso's strong defence of the single case study was extremely important at that time in history.

Questions for Reflection

- What is the cognitive neuropsychology jigsaw and why is it important for helping us understand intact healthy cognitive functions?
- Why are double dissociations such a powerful conceptual tool in neuropsychology?
- When deciding on the number of participants for a neuropsychological study, why is it not a simple case of 'single' or 'group'?
- Why are inferences about healthy cognitive systems easier to make from patients with acquired rather than developmental disorders?

Further Reading

- Medina, J., & Fischer-Baum, S. (2017). Single-case cognitive neuropsychology in the age of big data. *Cognitive Neuropsychology*, *34*(7–8), 440–448.
- Caramazza, A. (1986). On drawing inferences about the structure of normal cognitive systems from the analysis of patterns of impaired performance: The case for single-patient studies. *Brain and Cognition*, *5*(1), 41–66.
- Lambon Ralph, M. A., Patterson, K., & Plaut, D. C. (2011). Finite case series or infinite single case studies? Comments on 'Case series investigations in cognitive neuropsychology' by Schwartz and Dell (2010). *Cognitive Neuropsychology*, *28*(7), 466–474.