Nancy D. Chiaravalloti
Yael Goverover *Editors*

Changes in the Brain

Impact on Daily Life

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Preface: Laying the Framework

Often, we meet clients in our practice who have had a brain injury or another type of neurological illness. When trying to explain the importance of the brain, we tell them that our brain directs everything our body does. Without the brain, or to be more realistic, without its functions, we would not be able to do most of the things we do on a daily basis, from the mundane to the rare. Our brain is a central processing unit that translates our thoughts, feelings, memories, and opinions into a complicated nerve cell firing process and chemical release. These processes are responsible for our behaviors, and these connections are complicated and difficult to explain. Recently, there has been a growing awareness of the functional implications caused by the injured brain. Traditionally, professionals have used neuropsychological batteries or impairment-based assessments to document the patients' symptoms. These traditional assessments, however, do not focus on learning how the symptoms interfere with daily activities, or why.

We were inspired to compile this book because in the past 20 years we have seen numerous clients through clinical practice and research that share the common characteristic of a central nervous system that functions less than optimally, accompanied with diminished engagement in activities of daily living and social activities. This could be due to injury, illness, or just advancing age. When practitioners and researchers discuss the symptoms experienced, they discuss the impairments that were caused by the damaged brain. These impairments could be cognitive or motor in nature, among others. Our clients, however, speak about how these impairments have impacted their life. They often mention the fact that they cannot go to work anymore, and that they no longer receive social invitations, or drive their car. However, for some patients this link is not linear, which makes this connection between the brain and the behaviors even more complicated. For example, one may see two people with memory impairment, one diagnosed with a preclinical dementia and the other having sustained a TBI. The person with the dementia does not leave his house anymore and does not attend any social events. The person with the TBI went back to work and manages an active life. Why the difference in behavior? The observation of differences in such behaviors across various patient populations, coupled with the challenges affected individuals and their families have in understanding these differences, triggered the writing of this book.

This book includes 13 chapters, all with a common theme—the link between diagnosis, brain, and behavior as it plays out in everyday activities. We also sought to explore different causes of distinct behaviors. Is the diagnosis the essential element, or is it the course of illness, or perhaps cultural factors? Or, is it a combination of such factors that leads to distinct difficulties in daily life activities? In each chapter that follows the diagnosis and its characteristics are described, followed by the relationship between the symptoms and disability. In addition, factors such as culture and society are discussed. In most chapters we sought to illustrate the dynamic link between impacted brain structures, impairment, and participation in everyday life performance. We hope that the reader of this book will be aware of both the complexity in the functioning of the brain and, more importantly, how brain function/dysfunction affects the performance of everyday life activities. The purpose of the book is to educate the reader in regard to the changes in everyday life that are encountered with various mechanisms of brain insult/brain changes. A discussion of the impact of such changes from the perspective of the patient is also included. The book was designed to be useful to the professional, but also of interest to those directly affected by brain injury, brain illness, or brain changes that come with normal aging.

In order to present the most accurate information about the different types of changes in brain function resulting from the different etiologies, each chapter was written by experts in the topic areas. Thus, one can read the entire book or just the chapter of particular interest. While each chapter is a standalone chapter, some chapters such as Chaps. 1 and 11 are general and relate to all types of disability. We are very grateful to the authors who took their time to contribute to our understanding of their area of expertise. We are also grateful to the patients who contributed their portrayal of how the injury or disease affected their life.

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Neuroanatomy: The Brain–Behavior Relationship

1

Glenn Wylie, Erica Weber, Daniela Sacchetti, Silvana Acosta, and Helen Genova

Introduction

This chapter is designed to serve as a reference for the mapping of some of the more important human functions and abilities (e.g., vision) to their respective brain areas (e.g., the occipital lobe). Vision, audition, touch, motoric output, emotion, memory and language abilities are discussed. In each section, some common deficits that result when the relevant brain area is damaged are also discussed. This provides a framework within which the other chapters of the book, which cover particular issues in greater detail, can be understood.

Vision and Visuospatial Functions

The visual system in your brain accomplishes something extraordinary every time you open your eyes: it allows you to perceive—to see—energy. The energy you can see is light, and the light we can see is energy of a particular wavelength. This

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section will explain how the visual system manages to change energy into neural impulses that your brain can use.

Vision begins in your eye. On the back of your eyeball, a sheet of cells called the retina transduce (or change) light energy into neural impulses. There are two types of cells in your retina, which are named after their shape: rods which are cylindrical, and cones which are cone-shaped. Rods need less light to work, but are sensitive to only a limited range of light at the blue end of the spectrum. Cones are sensitive to a far wider range of light, and thus allow us to see in color, but require far more light to work. This is why you do not see the world in rich colors at night, as you do during the day. At night, there is not enough light for your cones to work, and so only your rods are active—resulting in a world of dark blues. The most sensitive part of your retina is your fovea, and the fovea is densely, and exclusively, packed with cones. During the day, this allows you to see things that interest you in high definition and in full color.

The difference between rods and cones in the retina illustrates an important principle that is found throughout the visual system, and throughout the brain: information is broken up and different systems handle different types of information. Another general principle of brain organization is shown as the information moves from the retina to the visual cortex. As the information is transmitted along the optic nerve, it is divided such that information about the left half of space (the left visual field) is delivered to the right half of the brain (the right hemisphere) while information from the right visual field is delivered to the left hemisphere. This division of information is seen throughout the brain: the right side of the brain receives input from the left side of the body and vice versa. Moreover, the right side of the brain controls the left side of the body (and vice versa).

In the visual cortex, which is located in the "back" of the brain, just above the nape of your neck, the information from the eyes is broken down even further. Lines and edges are represented in one part of the visual cortex, colors in another, motion in yet another, and so forth. We know this because if these areas in the visual cortices are damaged (e.g., by a stroke), then the function that was supported by that area is also lost. For example, when the area responsible for representing color is damaged by a stroke, the stroke survivor will be unable to see colors. He/she will perceive the world as comprised of shades of grey. Furthermore, and somewhat surprisingly, he/she will also be unable to remember colors. This points to another important aspect of how the brain is organized and how it works: memories are represented by the same areas that represent the information when it is perceived. Therefore, when you "look back" at your childhood, you really are looking back inasmuch as your childhood memories are represented in your visual cortex. Visual memories are represented in visual cortices, auditory memories are represented in auditory cortices, somatosensory memories (memories of touch) are represented in somatosensory cortices, and so on.

While someone who has suffered damage to color processing areas will be unable to see colors, someone who has sustained damage to visual motion processing areas will be unable to see motion. These individuals see the world not as a dynamically changing scene, but more like a series of still photographs. This can be

very debilitating, because the series of still photographs does not update quickly enough to avoid accidents. For instance, when crossing the street, one might see an empty street, then a car turning onto the street, and then the car might strike the person before the scene is updated.

As the visual information moves through the visual system, it is gradually reassembled as the different aspects of the thing one is looking at (its color, its location, its luminance, its motion, its depth, etc.) are associated with one another. One area that is specially tuned to this kind of higher order visual information is the "fusiform face area," which is located on the bottom of the brain. This area is highly active when we look at faces, regardless of whether the face is someone we know or not. This area is also active when we look at face-like stimuli, such as cars where the two headlights look like eyes and the bumper looks like a mouth. Indeed, this area is probably analyzing all visual information all the time to see if it is a face, and is probably responsible for things that are not faces (e.g., cars) appearing to look like faces to us. It is likely that we have an area devoted to face processing because faces are so important to our survival. It is important to be able to recognize your parents when you are young, it is important to be able to distinguish friend from foe as you get older, and it is important to be able to recognize subtle changes in faces (e.g., the difference between a bored face and an angry face) at all times. Indeed, the ability to recognize faces is so central to our lives that the deficits resulting from losing this ability are surprising.

Disturbances in the reassembly of visual material can result in *agnosias* (Farah, 2004), a Greek term for "without knowledge." At a more fundamental level, *apperceptive agnosia* describes the difficulty in forming a mental representation of what is visually sensed, such that individual visual characteristics (e.g., light/dark, size, color) are perceived but cannot be integrated into a meaningful whole. This syndrome typically occurs after diffuse damage to the occipital lobe and surrounding neural regions. A higher-level deficit is seen with *associative agnosia*, in which individuals can perform the perceptual integration that is deficient in apperceptive agnosia, but cannot attach semantic meaning to the visual percept. For instance, they may be able to copy a picture of an object accurately, but would be unable to name it or draw it from memory, due to the lack of meaningful information attached to what they see. Beyond these two broad distinctions, visual agnosias may be limited to a specific category of object. For example, patients who have lost the ability to recognize faces from vision are said to have *prosopagnosia*. Because the rest of their visual system is generally intact, they are perfectly able to distinguish (and faithfully report) the features of a face. They can tell you the color of someone's eyes, the shape of his nose and chin, whether his teeth are straight or crooked. However, they are entirely unable to put all this information together into a single representation—that is, into the percept that you or I might call "John's face." They cannot recognize people they have known and loved for decades; they cannot recognize famous people; they cannot tell you whether two faces are from the same person or from different people except by laboriously cross checking each feature one by one. However, while they cannot recognize friends and family by sight, as soon as they hear the voice of one of these people they immediately recognize

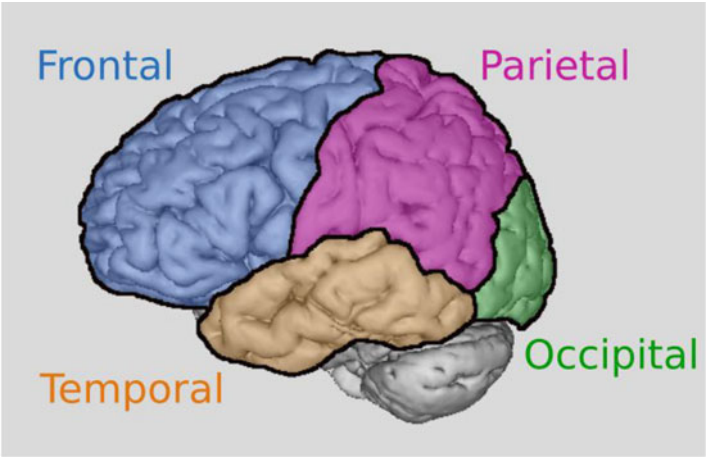

Fig. 1.1 The four primary brain regions, shown on the left hemisphere of the brain. The occipital lobe is shown in *green*, the parietal lobe is shown in *purple*, the temporal lobe is shown in *orange*, and the frontal lobe is shown in *blue*. The cerebellum is also shown in *grey*

them—thus showing that they have not forgotten these people, but rather that their facial recognition abilities have been damaged.

Much of the brain is devoted to vision. Indeed, the entire "occipital lobe" of the brain is given over to processing visual information. The occipital lobe is located at the back of the brain (see Fig. 1.1), and as we have seen, different aspects of visual information is processed by different parts of the occipital lobe. As the visual information is processed by the brain it moves from the back of the brain toward the front, and as this happens the information becomes increasingly complex. It follows two "streams" of processing: one stream results in representations about "what" we are seeing while the other results in representations about "where" the things are located in space. The "what" stream begins in occipital areas and ends in the temporal lobe, which is located under your ear and extends up to your temple (the face processing area is part of this stream). The "where" stream also begins in occipital areas, but extends up the back of your head into the parietal lobe, which is located between the back of your head and your crown. Visuospatial processing subserved by this "where" system describes a range of abilities, from basic (e.g., localization of points in space) to more complex (*constructional praxis*; Lezak, Howieson, Bigler, & Tranel, 2012). Fundamental parietal functions are critical in depth perception, orientation of lines, and perceiving motion—these represent many of the building blocks needed to perceive and interact with the spatial environment. More complex tasks include *constructional praxis*, which is the ability to manipulate objects based on spatial relationships. Examples of praxis include tasks like using your hand to turn a door knob and open a door, threading a needle, or copying a figure. Route-finding is another important use of visuospatial information that has great bearing on everyday functioning. These visuospatial abilities interact with other cognitive systems (e.g., attention) to produce more meaningful visual input

and directed output, such that seemingly visual disturbances may occur as a result of other cognitive deficits. One common example is that of *hemineglect*, an attention-based deficit typically caused by damage to the parietal lobe in which individuals essentially ignore one side of space.

Audition

There are some aspects of audition that are familiar. The keys toward the left of a piano's keyboard produce lower notes than those at the right. After being in a loud environment, our ears "ring" when we move to a quieter place. We are able to distinguish whether a sound is coming from the left or the right. When we are completely engrossed in an absorbing activity, we sometimes do not hear when people speak to us. This section will explain how these commonplace occurrences are achieved by the brain.

Sound enters the ear, and is transmitted—via the tympanic membrane—to a structure called the cochlea. The cochlea is shaped like a snail's shell, with an inner cavity that spirals in on itself getting smaller as it continues. This cavity is filled with fluid and the vibrations that sound produces on the tympanic membrane produce corresponding vibrations in this fluid. These are converted into neural signals by "hair cells" that are tiny, hair-like structures within the cochlea. Because of the way the cochlea is constructed, sounds of different frequencies cause vibrations in different parts of the cochlea; sounds of lower pitch cause vibrations near the one end of the cochlea (near the center of the spiral) and sounds of higher pitch cause vibrations at the other end. The neural signals produced by the hair cells are transmitted to the auditory cortex via the auditory nerve. Because sounds of different frequencies are registered by hair cells in different places in the cochlea, the brain is able to distinguish different sounds by where on the cochlea the information came from. If it came from the center of the cochlea's spiral, it was a low-pitched sound; if it came from near the outside of the cochlea, it was a high-pitched sound. In fact, the auditory cortex is "tonotopically mapped" such that higher pitched sounds are represented on one end of primary auditory cortex, low pitched sounds are represented at the other end, and intermediate pitches are represented in between—an organization reminiscent of a piano's keyboard. Thus brain deciphers the auditory world by converting the intangible world of sound into the far less difficult problem of where neural impulses come from on the cochlea (i.e., spatial location).

The hair cells on the cochlea are extremely delicate, and can be damaged by excessive vibration. Indeed, as we grow older, we gradually lose the ability to hear certain frequencies of sound, with the higher frequencies being the first to go. This is a particular problem for musicians, who not only need to hear the full range of sound, but also are constantly in environments that are full of sound. One possible consequence of hearing loss due to damaged hair cells is a condition called *Tinnitus*. This is a persistent "ringing" in the ears (though it can also be noises such as hissing, roaring, whistling, or clicking) that does not correspond to any external stimulation. As in disorders such as "*phantom limb*" (see sections on motor and

somatosensory cortices), *Tinnitus* may result from a loss of input to the brain. In the case of *Tinnitus*, it may be that the hair cells in a particular frequency range have been damaged sufficiently that primary auditory cortex no longer receives information about that frequency of sound from the external world. However, the cells representing that sound on the cortex continue to exist and continue to fire, and it may be that *Tinnitus* results from a continuous firing of these cells. The ringing in your ears after you have attended a loud concert is due to a related cause. In this case, there has also been damage done to the hair cells, but the damage is on a smaller scale and is not irreparable (taking about 24 hours to repair).

While our ability to locate sounds in space is not as good as that of other animals, such as a cat or a horse (because our ears are fixed on the sides of our head), it is nevertheless quite serviceable. We are able to tell where a sound comes from with sufficient precision to orient in the appropriate direction, at which point our eyes generally take over the task of identifying the source of the noise. The brain accomplishes this by comparing the time at which a given sound arrives at each ear. When a sound occurs on the left, it arrives at your left ear first and at your right ear an extremely short time later; when the sound comes from directly in front of you (or directly behind you), the sound reaches both of your ears at the same time; and when the sound comes from the right, it arrives at your right ear before your left. Although the difference in arrival time is minute, your brain is able to use this information to compute the origin of the sound (you can verify this by plugging one ear and trying to locate sounds). This fine-grained temporal analysis of auditory input is done in subcortical areas before the information reaches the primary auditory cortex, which is located on the top surface of the temporal lobe. Once the information reaches auditory areas, it appears that there is an auditory "what" system that extends down into the temporal lobe, and a "where" system that extends up into parietal areas (similar to the "what" and "where" systems in vision).

Unlike vision, where you can stop visual input into the brain by closing your eyes, your auditory cortex receives information continuously. You cannot close your ears in the same way you can close your eyes. However, we are able to stop auditory information from impinging on our thoughts through the use of auditory attention. At its most basic level, we can use auditory attention to simply inhibit all auditory input. You may do this when reading an absorbing book, for example. However, auditory attention also allows you to follow one conversation out of many in a crowded room (e.g., during a party). In this case, the environment is full of speech sounds, but only one stream of speech—coming from the person you are speaking with—is relevant, and you are able to selectively hear that person while the other conversations are ignored. While the information from other conversations is ignored, your auditory system is however still processing it. This is shown by the "cocktail party effect": when you are in a crowded room, carefully attending to one conversation out of many, you do not "hear" the other conversations at all. You are aware of noise, but not of any of the words spoken by the other people in the room. However, if someone calls your name, your attention is immediately attracted to that person. This could not happen if you had not been processing the auditory information from other speakers all the time.

Touch

Imagine you are baking cookies and absentmindedly touch the hot cookie sheet without an oven mitt. You pull your fingers back in pain. An important somatic sensory (touch) signal was just sent to your brain to relay information to help you determine your next move. We are always receiving such sensory information: the feel of your fingers on the keyboard, an itchy tag in your shirt, or a cold glass of water in your hand. Somatosensory information is responsible for the perception of different sensory signals: tactile signals (touch, vibration), where your limbs are in space (proprioception), pain, and temperature. These signals are conveyed along two different pathways in the spinal cord, both of which terminate in the thalamus (located deep in the center of the brain) before information is sent to the primary somatosensory cortex (a strip of cortex extending from the top of your head down towards each ear). These pathways are called the *dorsal column-medial leminiscal system* (DCML) and the *anterolateral system*.

Tactile Sensation and Limb Proprioception

The *dorsal column-medial leminiscal* (DCML) system is primarily responsible for perception of tactile sensation such as touch and vibration. The name of the pathway is derived from two structures along the pathway: the *posterior (dorsal) columns* of the spinal cord, and the *medial lemniscus* in the brain stem. From there, the pathway continues to the thalamus and thence to the primary and secondary somatosensory cortices (in the post-central gyrus) and posterior parietal cortex. This pathway is responsible for the localization and sensation of touch, and enables someone to determine not only what is being touched, but also which part of the body is doing the touching. This ability can be selectively lost. Indeed, in *astereognosis*, individuals cannot recognize an object in their hands (e.g., a comb) when relying on the sense of touch alone. However, when they are allowed to see the object, they are immediately able to identify it.

Limb proprioception is perceived via the same pathway (the DCML), and can be defined as the perception or awareness of your own limbs relative to the rest of your body. In everyday life, we are often unaware of proprioception except when we accidentally turn an ankle or have to quickly react to avoid colliding with someone else. However, there are certain clinical populations in which proprioception is negatively impacted including multiple sclerosis (MS). In populations such as MS, individuals may experience the sensation of walking "lop-sided" or feeling tipsy, which may increase the risk of falling. Thus, individuals may need to be more aware of the placement of their limbs to ensure balance and coordination.

Pain and Temperature

The *anterolateral system* plays a major role in the perception and processing of pain and temperature, and a minor role in tactile sensation and proprioception. There are three major pathways of the anterolateral system: spinothalamic, spinoreticular, and

spinomesencephalic. Noxious and temperature sensations are transmitted by the spinothalamic and spinoreticular tracts. The sense of pain is largely mediated by the spinomesencephalic pathway. Similar to the DCML pathway, the anterolateral pathway ultimately terminates in the primary and secondary somatosensory cortices and posterior parietal cortex.

In leprosy, a disease caused by a chronic bacterial infection, patients can lose the ability to feel pain due to the deterioration of the nerves of the somatosensory system. Limbs therefore can become subject to various injuries because the patient does not experience pain as he/she should. For example, when you get a blister on your finger after spending an afternoon raking leaves, you avoid using that finger for several days because the blistered area hurts when touched. This allows it to heal. If you were unable to feel pain from that finger, you would not favor it, and the damaged area would not be given the opportunity to heal. Eventually, this could lead to irrevocable damage. A similar disorder, which is genetic rather than infectious, is *hereditary sensory neuropathy type 2* (HSN2), in which the patient is unable to feel pain, or in some cases, any sensation of touch whatsoever. These patients are at risk for innumerable injuries: they burn themselves in the bathtub, break bones, suffer infections, without knowing or sensing that anything has occurred. On a daily basis, HSN2 affects their functioning in that they may have difficulty with tasks of every day living such as self-care or care of others. Damage to fingers or hands may make it so that it is difficult to dress oneself, brush one's hair or prepare food for one's self or family.

Sensory Homunculus

Primary somatosensory cortex is organized such that the parts of your body that are next to each other (e.g., your hand and your wrist) are represented in adjacent places on the cortex. That is, your primary somatosensory cortex is organized like a little person (an homunculus), with the head represented next to the neck, which is represented next to the chest, and so on. This is also true of primary motor cortex (see below). A neurosurgeon named Wilder Penfield conducted a series of experiments during operations on patients with epilepsy and other brain disorders (Penfield, 1950). Penfield stimulated the post central gyrus of patients (under local anesthesia) and asked where in the body they felt the stimulation. He found that during these stimulations, sensations of pressure, tingling and numbness were reported from different parts of the body in a pattern that he used to create a somatosensory map (or homunculus). This map revealed that the left side of the body was represented in the right hemisphere of the brain, and it also showed that the amount of cortical area devoted to different parts of the body are not equal, nor are they proportional to the size of the body part. For example, the amount of cortex devoted to the face is far greater than the area devoted to the back of the head.

Somatosensory Disorders

There are two major types of disorders of the somatosensory system. The first is *Primary Tactile disorder* in which damage occurs to the primary somatosensory cortex, the thalamus or subcortical regions of the somatosensory pathways. This damage results in loss of the ability to perceive basic aspects of somatosensory information, such as pressure sensitivity, perception of vibrations, two-point discrimination or deficits in proprioception. Primary tactile disorder can result in one type of impairment (lack of pressure sensitivity) while retaining other abilities (proprioceptive knowledge or temperature perception) (Corkin, 1978).

Higher order touch disorders involve damage to the secondary somatosensory cortex, the posterior parietal cortex or the insula. These disorders involve impairment of the ability to perform more higher order processing of touch including object recognition and feature discrimination. Individuals with *amorphognosia*, for example, have difficulty perceiving the size and shape of an object. *Ahylognosia* results in the inability to perceive the texture, weight, or temperature of an object (Delay, 1935; Denes, 1989). *Tactile agnosia* results in an inability to identify an object based on its somatosensory properties even though the patient has not lost his/her sense of touch (Caselli, 1991). A patient with this type of agnosia would be unable to identify an object in their hand if they were not allowed to see or smell it, for example.

Damage to the somatosensory system can cause disorders related to one's perception of their own body. Our perception of our body is based on somatosensory input, proprioception, and visual feedback. Many disorders involving body image and body awareness can occur, including *asomatognosia* whereby the patient is not aware of certain body parts, and feels that they are "missing." In *somatoparaphrenia*, the patient does not believe that he/she owns a part of their body that is paralyzed. In severe forms of these disorders *misoplegia* can occur, where the patient can abuse their own body part because they feel strong resentment or hatred of it. These types of disorders can occur after damage to premotor, parietal and posterior insular regions (Vallar & Ronchi, 2009). The experience of "phantom limb" refers to the situation in which an amputee patient has the sensation that a removed limb is still present (Ramachandran & Hirstein, 1998), discussed in further detail in the next section.

Movement

Movement is one of the key ways that we interact with our environment. We are constantly being exposed to objects and we must decide what to do with them. Making decisions about our actions are often based on intentions and goals. For example, if you are feeling tired you may decide that you want to have a cup of coffee, so you walk into your kitchen, reach for a mug, grab the coffee pot, and pour