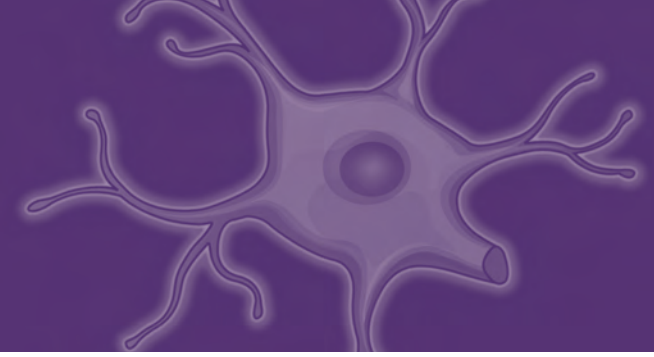
NEUROSCIENCE FUNDAMENTALS

for

Communication Sciences and Disorders



NEUROSCIENCE FUNDAMENTALS

for

Communication Sciences and Disorders

RICHARD D. ANDREATTA, PhD

Plural Publishing Inc.

PLURAL PUBLISHING
INC.

5521 Ruffin Road
San Diego, CA 92123

e-mail: information@pluralpublishing.com
Website: http://www.pluralpublishing.com

Typeset in 10.5/12 Adobe Garamond Pro by Flanagan's Publishing Services, Inc.
Printed in Canada by Friesens Corporation.

Library of Congress Cataloging-in-Publication Data:

Names: Andreatta, Richard D., author.
Title: Neuroscience fundamentals for communication sciences and disorders / Richard D. Andreatta.
Description: San Diego, CA : Plural Publishing, [2020] | Includes bibliographical references and index.
Identifiers: LCCN 2018049910| ISBN 9781597568890 (alk. paper) | ISBN 1597568899 (alk. paper)
Subjects: | MESH: Central Nervous System--physiology | Central Nervous System--anatomy & histology | Sensation--physiology | Perception--physiology
Classification: LCC QP376 | NLM WL 300 | DDC 612.8/2--dc23
LC record available at https://lccn.loc.gov/2018049910

Contents

PREFACE

The idea for this book has been running around in my mind since I first started university teaching back in 2000. All that was needed was an opportunity to get this idea off the ground and realized. That opportunity came in late fall 2014 when I received an email from Valerie Johns, Executive Editor at Plural Publishing, asking whether I would be interested in developing a high-quality neuroscience textbook for communication sciences and disorders students. As is evident by the book you are holding and reading right now, I grabbed that amazing opportunity and have spent the better part of the last 3 years working toward its realization and completion. I hope you enjoy the results!

Neuroscience Fundamentals for Communication Sciences and Disorders (NFCSD) was developed with three personal goals in mind. First and foremost, NFCSD was developed to provide senior undergraduate and graduate students in communication sciences and disorders (CSD) programs (as well as students in Doctor of Audiology [AuD] programs) with a richly illustrated, comprehensive, yet highly readable and accessible textbook covering the neuroanatomy and neurophysiology of the human nervous system. NFCSD was conceived and written as a "brain and behavior" style of textbook, while also ensuring coverage of essential neuroanatomy in an integrative fashion. My central motivation for producing this book was (and continues to be) to help students develop a deep understanding and appreciation of critical brain–behavior relationships that can impact and help inform their careers as rehabilitation specialists for patients and clients with speech, language, and hearing disorders. Valuing the critical role of the nervous system in the behaviors, perceptions, and cognitive functioning of someone with a communication disorder is something I believe is central to the development and implementation of effective and efficient evidence-based treatments.

Second, I aspired to write a textbook for practicing clinicians who want a comprehensive guide to the inner workings of the nervous system and its processes. A reference book of this type is always useful in one's professional library. Clinicians need a reference book that can be appreciated, read, and understood without having to take another formal course in the subject matter. My hope is that the accessibility of the writing and the illustrations can allow those who have been out of school for a while to use the material in this book to directly inform their practice. Aside from its direct use for informing patient care, the book can also be used for client and caregiver educational purposes; the illustrations and the explanatory analogies may be ideal for helping clients better understand the brain-based nature of their disorder.

Finally, my third personal goal was to write a textbook for faculty who are responsible for teaching neural bases of speech and language courses, but who do not themselves have specific expertise in this area. Larger programs in CSD usually have someone on their faculty with specific neuroscience experience, but medium- to smaller-sized programs may not. I wanted this textbook to be a resource or guide for any faculty colleague of mine who is in the challenging position of having to develop a class in and/or teach a neural bases course, but does not feel comfortable or confident in doing so. If this happens to describe your circumstances, then this textbook can certainly be of help to you. For faculty in this position, think of the textbook as more of a user's guide to help you develop lectures and course content. Feel free to use the analogies and examples scattered throughout the book as your own when teaching; customize them as you wish. The lecture slides that are available on the book's PluralPlus companion website provide a framework that you should feel free to customize and change as your circumstances dictate.

In closing, let me say that I believe textbooks should be dynamic and living things that evolve and change as we learn more about our subject matter. The contents of this book are as current as possible, and every effort has been made to check and cross-check its accuracy against several sources. Despite these efforts, there will be errors in the book, and for these I apologize in advance; the fault for these errors is mine, and mine alone. To students, clinicians, and adopting faculty alike, if you find errors in the book, please feel free to e-mail me at richard.andreatta@uky.edu, and I will compile a list of corrections to make upon any future editions of the book that may get published.

For faculty who choose to adopt this book for their own classes, I especially welcome your thoughts and opinions on the content of the book and its organization; I really mean this! If there are sections that you feel need to be expanded or reduced, areas that should be organized differently, or areas where my interpretation of the material doesn't conform to your own, please e-mail as well. I would welcome this kind of feedback and dialogue from those who are choosing to incorporate this textbook into their courses. I want this book to work for you in the best way possible, and the only way I can accomplish this is by getting your real-use perspectives.

Lastly, as a university professor I have always made it a point to maintain an open-door policy in my department and be available to students whenever they need me for help. Because I consider this textbook to be a direct extension of my teaching, I'd like to offer students or clinicians who are using the book an open invitation to contact me with questions that pertain to its content or for clarification on any explanation I may have used.

Some Suggestions for All Faculty Who Adopt NFCSD

To faculty who choose to adopt this book for their courses, the book was written to build a student's understanding and comprehension of neuroscience gradually from the workings

of the neuron to the broad functioning of systems underlying behavior and cognition. Let me offer the following suggestions for using and assigning readings from this book, to help you and your students get the most out of it. First, Chapter 1 sets the stage and rationale for the textbook and provides important study ideas and strategies for student use. In other words, don't skip over Chapter 1, as many of us are guilty of often doing. It will help orient students and contains important insights as to the grand scope of the book's content.

Chapters 2 through 5 should be read in the order presented because these chapters lay out the essential groundwork for the remainder of the book's content. Chapters 2 through 5 should be bookmarked and referenced *continuously* as students proceed through the later sections of the textbook. It is important to think of Chapters 2 through 5 as a reference core within the book itself. I have intentionally placed throughout the chapters parenthetical reminders to go back to this reference core to review critical concepts and descriptions. This was a pedagogical decision that I believe will help students cross-link different topical areas of information. Lastly, Chapter 5 should be assigned in small sections rather than as a whole. Use the major headings to subdivide the chapter's content and your reading assignments.

For the sensory section of the textbook, the first part of Chapter 6, covering sensation and perception, definitely needs to be read first because this content cuts across all the sensory systems equally. After this is accomplished, the remaining part of Chapter 6 and all other sensory chapters can be read in the order that best suits your needs. I ordered these sensory chapters based on my own view of their importance to CSD, with somatosensory and auditory being the most critical systems to appreciate, followed by the visual and chemical senses.

The motor systems section of the textbook should ideally be read in the presented order, but it is perfectly fine to move Chapter 10 on muscle tissue to a different content location if needed. This chapter can also be used as a self-study module if you don't want to spend class time on this material. The motor control systems content in Chapter 11 should be assigned in four separate segments: (1) direct motor systems, (2) the basal ganglia, (3) the cerebellum, and (4) the visceromotor system. Lastly, Chapter 12 on motor control theory is another chapter that can be used as a self-study module if so desired.

Finally, Chapters 13 through 15 can be read in any order because each is self-contained. I can envision some faculty using Chapters 13 through 15 immediately after Section 1 of the book, or perhaps using Chapter 15 on the central auditory pathway immediately after the content on the inner ear presented in Chapter 7. Although these three chapters were intended to be the culminating section of the book, use them as you think is best to meet your needs.

Now, while I consider all the chapters to be important (obviously because they are included in the book), let me offer some advice about what material can be safely trimmed. Chapter 3 is perhaps the content area that many will choose to trim because it deals with electrochemical signaling and includes some biochemistry. It is perfectly fine to do this as long as the student gets the "big picture" idea behind neural signaling and synaptic function. Using the analogies and the functional illustrations provided in the chapter will help your students achieve this broad level of appreciation. In Chapter 5, material on the hypothalamus and thalamus can be trimmed without losing too much understanding needed for later discussions. Within Chapters 6 through 9, the more neurobiologically flavored sections that contain some biochemistry content can be safely trimmed without loss of understanding, again, as long as the general summaries for the associated mechanisms are retained.

In the end, what you choose to use from the textbook will be your decision based on your needs and the realities of course time and scheduling. The book was designed to broadly meet the various needs of both students and faculty in different types of CSD programs. Depending on your department and how integrated your curriculum may be, the book can conceivably be purchased at the start of a student's program and then used throughout the curriculum as needed. Of course, the book is intended for use in neural bases classes, but parts of the book can potentially be adapted for use in several different courses. For example, the auditory system chapters can be held over for audiology courses, the neural bases of speech and language chapters can be used as review material for motor speech and normal language courses, the neuroanatomical content of Chapters 4 and 5 can be incorporated into aphasia or neurocognitive classes, and so on and so forth. Regardless of how you choose to use this textbook, I hope that it meets your needs and that your students find it informative and useful in their training.

ABOUT THE ILLUSTRATOR

Maury Aaseng graduated from the University of Minnesota Duluth with a BFA in Graphic Design and began working as a freelance illustrator in San Diego, California, creating graphics for young adult nonfiction. Shortly after, his work expanded into medical and anatomical illustration when he started collaborating with authors and experts in various medical fields to create vivid figures for publications that illuminate concepts necessary to understand the science of the body. His subject matter in this field includes: illustrations of intricate imagery of human anatomy, brain surgery, endoscopic views, cellular level structures, and assorted graphs and visuals that support text content. His style range also includes: creating vector drawn line art, cartooning, mechanical illustration, logo creation, and more traditional art media such as ink, pencil, and watercolor. With a special focus on watercolors, he has created promotional materials for opera productions, custom paintings, and illustrative signage for landscaping initiatives focusing on native plants, watersheds, and pollinator habitats.

Maury lives in Duluth, Minnesota with his wife and two children near the shores of Lake Superior. Much of his artistic inspiration is drawn from the outdoors, where he spends time observing and photographing the natural world for subject matter. His work won recognition in the juried exhibition Upstream People Gallery in 2008, and a collection of his watercolor work titled *Saturated Life* was displayed at the Great Lakes Aquarium gallery in 2016. He has taught classes covering scientific illustration and nature-inspired watercolor, and over the years he has reached into his broad experience of painting and drawing to create books that demonstrate his techniques to other budding artists. An online collection of his work can be viewed at mauryillustrates.com

CONTRIBUTORS

Richard D. Andreatta, PhD
Associate Professor
Department of Rehabilitation Sciences
Communication Sciences and Disorders
Rehabilitation Sciences Doctoral Program
College of Health Sciences
University of Kentucky
Lexington, Kentucky
Chapters 1–11

Sarah Grace H. Dalton, MSP, CCC-SLP
Doctoral Candidate
Department of Linguistics
Department of Speech and Hearing Sciences
Department of Neurosciences
University of New Mexico
Albuquerque, New Mexico
Chapter 14

Nicole M. Etter, PhD, CCC-SLP
Assistant Professor
Department of Communication Sciences and Disorders
Pennsylvania State University
University Park, Pennsylvania
Chapter 9

Patrick O. McKeon, PhD, ATC, CSCS
Associate Professor
Athletic Training Clinical Education Coordinator
Department of Exercise and Sport Sciences
Ithaca College
Ithaca, New York
Chapter 12

Anne D. Olson, PhD, CCC-A
Associate Professor
Division Director
Communication Sciences and Disorders
University of Kentucky
Lexington, Kentucky
Chapter 15

Jessica D. Richardson, PhD, CCC-SLP
Assistant Professor
Department of Speech and Hearing Sciences
Center for Brain Recovery and Repair
University of New Mexico
Albuquerque, New Mexico
Chapter 14

Stephen M. Tasko, PhD, CCC-SLP
Associate Professor
Speech, Language and Hearing Sciences
Western Michigan University
Kalamazoo, Michigan
Chapter 13

REVIEWERS

Plural Publishing, Inc. and the author would like to thank the following reviewers for taking the time to provide their valuable feedback during the development process:

Irene M. Barrow, PhD, CCC-SLP
Professor
Department of Communication Sciences and Disorders
James Madison University
Harrisonburg, Virginia

Margaret Lehman Blake, PhD, CCC-SLP
Associate Professor
Communication Sciences and Disorders
University of Houston
Houston, Texas

William R. Culbertson, PhD
Professor
Communication Sciences and Disorders
Northern Arizona University
Flagstaff, Arizona

Carol S. Deakin, PhD, CCC-SLP
Associate Professor/Program Coordinator
Communicative Sciences and Disorders
Alabama A&M University
Normal, Alabama

Jeremy J. Donai, AuD, PhD
Assistant Professor
Department of Communication Sciences and Disorders
West Virginia University
Morgantown, West Virginia

Daniel Furnas, PhD
Assistant Professor
Communication Sciences and Disorders
Jacksonville University
Jacksonville, Florida

Janis M. Jarecki-Liu, PhD, CCC/SLP
Professor/Chair
Department of Communication Sciences and Disorders
Clarion University of Pennsylvania
Clarion, Pennsylvania

Janie Park Magee, MA, CCC-SLP
Clinical Director and Instructor
Department of Speech and Hearing Sciences
Delta State University
Cleveland, Mississippi

Kimberly D. Mory, MA, CCC-SLP, CHES, CCM
Associate Clinical Professor
Department of Communication Sciences and Disorders
Texas Woman's University
Denton, Texas

Diane M. Scott, PhD, CCC-A
Professor
Communication Disorders Program
North Carolina Central University
Durham, NC

Lori L. Stiritz, MA, CCC-A
Senior Lecturer
Department of Communication Disorders
Texas State University
San Marcos, Texas

ACKNOWLEDGMENTS

To create and publish a book of this scope takes a village. I've been supported throughout this process by so many individuals that if I miss thanking someone in the next few paragraphs, please accept my sincere apologies; it was not intentional in the slightest.

First, I wish to thank the crew at Plural Publishing for giving me the opportunity and the freedom to fashion together a book that met my vision for such a product and that I could be proud of creating. Thanks go out first to Valerie Johns, my executive editor, for recruiting me for this project and giving me the resources and freedom to make this project truly my own. Because of your kindness, support, and confidence in my work, I believe the project became everything that I hoped it would become. Next, my deep appreciation to Kalie Koscielak, my managing editor, for being my "go to" person whenever I had one of my infinite number of questions and concerns. During the last few months of the writing process, you helped me keep some semblance of sanity. I hope this textbook lives up to both of your expectations and confirms the confidence both of you graciously placed in me to deliver the goods. I'm very proud to be a member of the Plural family!

After the staff at Plural, the next person that truly needs my deepest sense of gratitude for helping me realize the true vision for this book is my illustrator Maury Aaseng. This book would be a fraction of what it is if it were not for his illustrating talents and gifts. Maury Aaseng is one of the finest illustrators I've had the pleasure of working with on any writing project. How he was able to translate my scribbles and mockups to create the vast majority of the beautiful illustrations in this book never ceased to amaze me. Thank you, thank you, thank you for being my partner in this project. I can't describe how lucky I was to have had you working with me on this book!

There are two sets of critical contributors to this book that I need to generously thank. First to my contributing authors: Steve Tasko, Jessica Richardson, Sarah Dalton, Anne Olson, Pat McKeon, and Nikki Etter . . . you guys have been terrific to work with and I cannot be happier with your contributions to this textbook. Each of you had a huge task in taking enormous bodies of literature and condensing them down to chapters that would give students an excellent scope of your topical area. I own each of you a rather large beer, or in the case of Nikki, a Bahama mama instead! Thank you all for agreeing to participate and for coming through for me to make this project a reality! The second group of contributors that I need to deeply thank are *all* my past and present students (you guys know who you are). I wish I could list all your names in the front part of the textbook, for you are all contributors and authors in this book. I sincerely mean this too! This book is the direct outgrowth of everything you have taught me throughout the years. You've showed me what worked, what didn't work, what needed better explanation and time to develop, what level of detail you could handle before revolting on me, and how to approach teaching each and every one of you in the most effective way possible. Being your professor has, and continues to be, the highlight of my career and my distinct honor.

As I said earlier, it takes a village to realize a project of this scope, and my village at the University of Kentucky, College of Health Sciences, is an awesome village to be a part of. Thank you to all my colleagues at UK for your constant encouragement, your cheerleading, and for taking over some of my committee duties to help me complete this book during its final stages. I wish to thank my college administration (Dean Scott Lephart and Associate Deans Carl Mattacola and Janice Kuperstein) for granting me sabbatical leave during the spring semester of 2017 to write the majority of this textbook. Without this dedicated time, this project would literally never have happened. Of my colleagues at UK, I wish to give a special and deep nod of appreciation to Joe Stemple, *Mister, you can do it!*; Anne Olson, my program chair; and Bob Marshall, my philosophical muse. To Joe: Thank you for being such a great friend and partner in crime! Thanks also for all your insightful advice, counseling, and constant cheerleading as this project took shape. To Anne: You have been such a dear friend and so generous with your willingness to grant me the time and resources needed to make this project a success. Thanks for all the little intangibles! Lastly, to Bob Marshall, who was the first to say to me very early on, "Hey, you should write a neuroscience book, you'd be great at it!" Even though the idea of writing a book was in my mind for quite a while, your encouragement made me seriously think to myself that I could actually do something like this. Thanks are also extended to Morgan Sandmann for her help in developing my reference files and to Korinne Henrickson for helping compile my glossary.

Reading through the pages of this manuscript while I was editing made me realize that so much of the spirit in my writing style and in the examples I used throughout the book had Steven M. Barlow imprinted all over them. Thank you Steve for having been such a profound influence in my life as an academic and university professor. My love and passion for all things neuroscience started with you, and I can't begin to thank you for opening my eyes to this amazing field of work. You continue to be one of the strongest mentoring forces in my career, and I'm proud to call you my colleague, but more critically, my friend. Thanks Hans!

Finally, I get to the part of the acknowledgments that holds the most value to me—my family. Without family, we are nothing, and I'm blessed to be a part of an incredible family of remarkable individuals. To Jane: I love you dearly and I'm so thankful each day that you somehow manage to see through my enormous number of faults and quirks and still choose to stay by my side. Your support, constant encouragement, and understanding of my various moods made this

entire project possible. No kidding . . . you are a saint! To Jonah, Joshua, and Madeline: Each of you played an important role in the development of this book. Thank you Jonah for taking several weeks out of your summer vacation to sit with Mom and proofread the entire book for me! You have made me a convert of the Oxford comma and have taught me more about the proper use of an ellipse than I could possibly imagine. Thank you Joshua for being the face of many examples that I used in the book and for helping me decide whether what I was writing made sense from a student's perspective. Thank you Madeline for being my creative and artistic consultant and guru. You told me very honestly whether a figure I was designing was any good or if it was just crappy. Finally (although I know you can't read this), thank you Stella (my golden retriever) for keeping me company and being by my side during those long and lonely sabbatical days. You made sure that I got up from my writing chair and away from my laptop at least twice a day to take you out for a walk around the neighborhood.

Finally, the fact that I can say that I am a professor at a major university in the United States and the author of a book is only possible because of the enormous sacrifices made by my parents, Ana and Oscar Andreatta. I am a first-generation American, and I am the proud son of Hispanic immigrants who left behind the countryside of Argentina to come to the United States to build a better life for their children. My parents came to this country with literally nothing, but through arduous work and perseverance built a wonderful life for me and my siblings. My parents, and my Dad especially, believed fervently in the power of education. Education was the cornerstone to developing into an honorable human being, one who was always kind and just. My dad passed away during the authoring of this book, but he knew of its writing and I know he would have been thrilled to hold a copy of it in his hands. Thankfully, my mom is still with us. When she holds this book and sees the name "Andreatta" printed on its cover, I know that she'll be thrilled not only for herself, but for my father as well.

Este libro está dedicado a
la memoria de mi papi, Oscar Andreatta,
"El Hombre de Sucre."

◆

(This book is dedicated to
the memory of my father, Oscar Andreatta,
"The Gentleman from Sucre.")

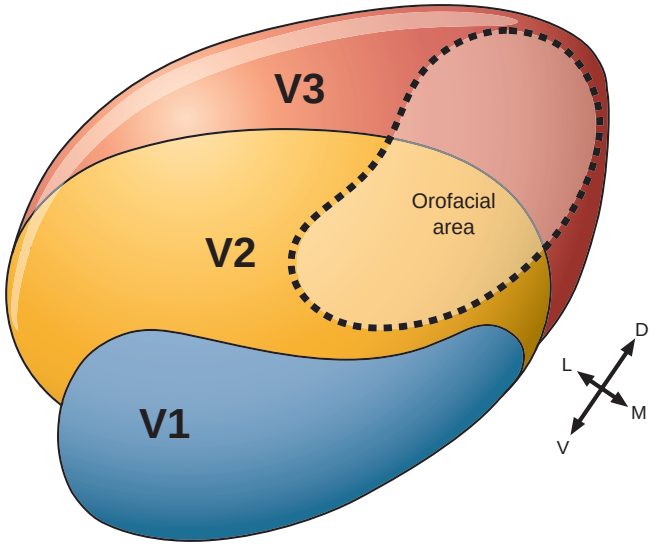


Figure 6–29. Schematic representation of the principal trigeminal nucleus. The principal trigeminal nucleus is somatotopically organized by structure within different regions of the nucleus. Facial structures from the ophthalmic branch (V1) are ventral, whereas structures from the maxillary (V2) and mandibular (V3) branches are more dorsal. Orofacial structures mediated by V2 and V3 are found in the medial aspect of the nucleus. The number of principal nuclei neurons that respond to orofacial stimulation far outnumber other neurons receiving inputs from less densely populated regions of the facial skin. [Legend: D (dorsal), V (ventral), M (medial), L (lateral)]

posterior limb of the internal capsule and the corona radiata (Capra & Dessem, 1992; Dodd & Kelly, 1991). These axons enter the cortex and form a synapse with the cortical neurons within layer IV of the ventral-most cortical tissue of the postcentral gyrus in the parietal lobe (Pais-Vieira et al., 2015). This general area is considered the primary cortical somatosensory representation for the face and oral anatomy (Fang, Jain, & Kaas, 2002).

The Somatosensory Cortex

We have finally made our way to the cortical somatosensory representation of the contralateral side of the body—the **somatosensory cortex** (S1) in the postcentral gyrus of the parietal lobe. S1 is considered the chief cortical input site for the modality of somatosensation and all associated submodalities (Amaral, 2013). Let's summarize where we are. Inputs to S1 originate from slowly and rapidly adapting mechanoreceptors, proprioceptors (muscle, joints, and tendons), nociceptors, and thermal afferent inputs out in the periphery. The somatosensory cortex lies three synapses removed from the input location (receptive field) of a peripheral stimulus event. Somatosensory stimuli transduced by peripheral receptors are transmitted along the DCML, ALS, and trigeminal sensory systems to the ventrobasal (VPL and VPM) complex of the thalamus (see **Figure 5–2**). From the thalamus, third-order neurons transmit tactile, proprioceptive, nociceptive, and thermal somatosensation to different locations in S1.

Structural and Functional Features of the Somatosensory Cortex

One of the most common ways to study the anatomical features of sensory cortical areas is to insert extremely fine microelectrodes into the cortex of a living animal and to record the electrical activity and responses of cortical neurons to controlled stimulation of peripheral receptive fields. In simpler terms, this method helps us identify which neuron or group of neurons in the cortex "light up" when a peripheral stimulus is applied to a receptive field of a given sensory receptor. This method has been a tried and true means in neuroscience to determine how the sensory cortex is "wired" to our peripheral sensors, helping us determine what exactly is connected to what. Through the use of this microelectrode mapping method, a number of key functional characteristics of S1 have been described.

By mapping the somatosensory cortex using neurophysiological methods, we have discovered that S1 possesses a highly ordered and detailed representation of the body's contralateral sensory receptive fields (Kaas & Garraghty, 1991). In other words, the neurons of S1 have an orderly arrangement that reflects the location of the body's receptive fields on (a) the skin (if we are referring to tactile, nociceptive, or thermal sense), and (b) in the musculoskeletal system (if we are referring to proprioception and deep forms of nociception from muscle tissue or joints). This orderly arrangement makes intuitive sense and, in fact, is critical for unambiguously identifying the peripheral location of any somatosensory stimulus event. Location of a somatosensory stimulus is unambiguously detected because there is a dedicated pathway from the sensory receptor in the periphery to the brain (our "labeled" line). This idea is the basic premise behind why we use microelectrode mapping methods and why they are effective to appreciate the correspondence between a stimulus event and central neural activity.

In effect, location can be thought of as somewhat "hard-wired" into the basic anatomical structure of the somatosensory system. We can test this notion by producing a sham perceptual condition in an individual by electrically stimulating his or her cortical S1 neurons. When S1 neurons are electrically stimulated, the participant will report perception of some form of sensation at the location mapped to the stimulated cortical S1 neuron. For example, electrically stimulating S1 neurons that obtain inputs from the skin of the lower lip will generate the false perception that something is actually touching your lower lip. This form of electrical assessment is

routinely performed during brain surgery to map the functionality of the cortical tissue before surgery begins so that critical cortical areas can be avoided as much as possible.

If we were to map every neuron in S1 through our microelectrode method, we would create a cortical representation of neurons that resembles the order of our own bodies, except inverted (Roux, Djidjeli, & Durand, 2018). Imagine taking a picture of yourself and draping it over the cortex of the postcentral gyrus in an upside-down manner, with the feet positioned over the edge of the longitudinal cerebral fissure in the midline and the head positioned over the ventral-most tissue that touches the lateral sulcus. Such a representation is shown in **Figure 6–30** and is called the sensory homunculus. The homunculus is a general-purpose visual analogy that helps us appreciate how the neurons in any cortical area are arranged and apportioned. You can create homunculi for any neural structure that uses spatial location and topography of the body as an organizing scheme. While homunculi are easily accessible ways for novice students and the lay population to understand the nature of the cerebral cortex, neuroscientists prefer to use more realistic and finely detailed maps of cortical areas derived from experimental data. This more precise form of cortical mapping is illustrated in **Figure 6–31** and represents the outcome of microelectrode recordings from literally thousands upon thousands of neurons (talk about a long experiment!). The lines drawn to demarcate once zone from another on these maps simply reflect the response of a group of cortical neurons when that specific peripheral body location is stimulated (Nelson, Sur, Felleman, & Kaas, 1980). In other words, tactilely stimulating the skin of the face will not normally activate cortical neurons mapped to the skin of the hand. Although these types of maps are not as visually appealing or intuitive as homunculi, they do carry and possess similar forms of information about a cortical area and its functionality.

The second critical feature of the somatosensory cortex that we can discern from our sensory homunculus is that there are differences in the size of neural populations serving different regions of the body. This feature can also be seen in our more precise cortical maps by examining and comparing the sizes of demarcated cortical zones for different body parts (see **Figure 6–31**). Inspecting our homunculus more closely, you'll notice that the size of a cortical area allocated to a body part corresponds to that region's sensory sensitivity and the type of behavior the area is involved with (Nelson et al., 1980). For example, the lower face and hands, as seen in **Figures 6–30** and **Figure 6–31**, are disproportionately larger compared to all other areas of the body, indicating that these anatomical regions have the greatest number of neurons processing their sensory inputs. In a manner of speaking, cortical representation size can be used as an indicator of (a) the importance of sensory input from that region, (b) the frequency with which that body area is used, and (c) the degree of behavioral skill that body part participates in.

In humans, the hands and the lower face are sites of great skill, tactile sensitivity, and frequent use (see **Figure 6–30**). For

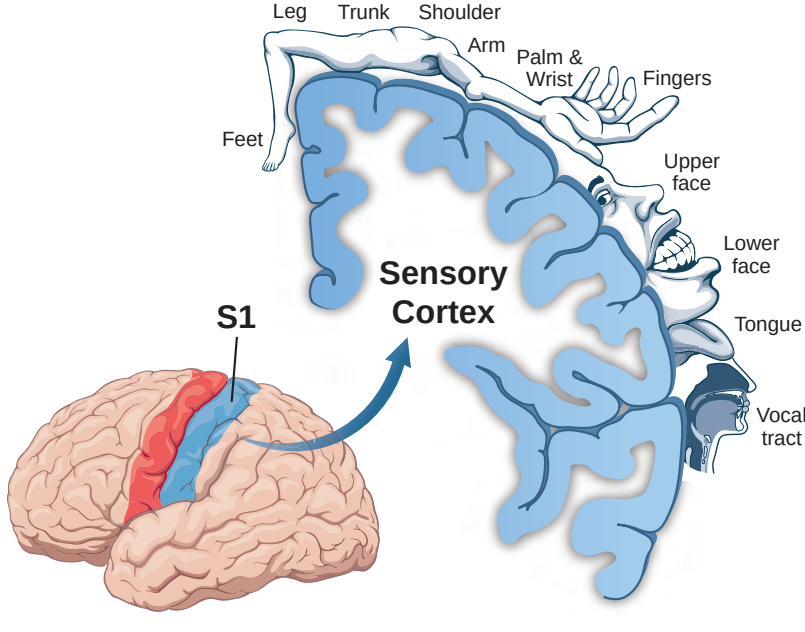


Figure 6–30. Sensory homunculus. The somatosensory homunculus is a visual analogy of how neurons in the primary somatosensory cortex are apportioned to different regions of the body. Enlarged regions of the homunculus correspond to body areas that require high degrees of sensory input to support skilled behaviors.

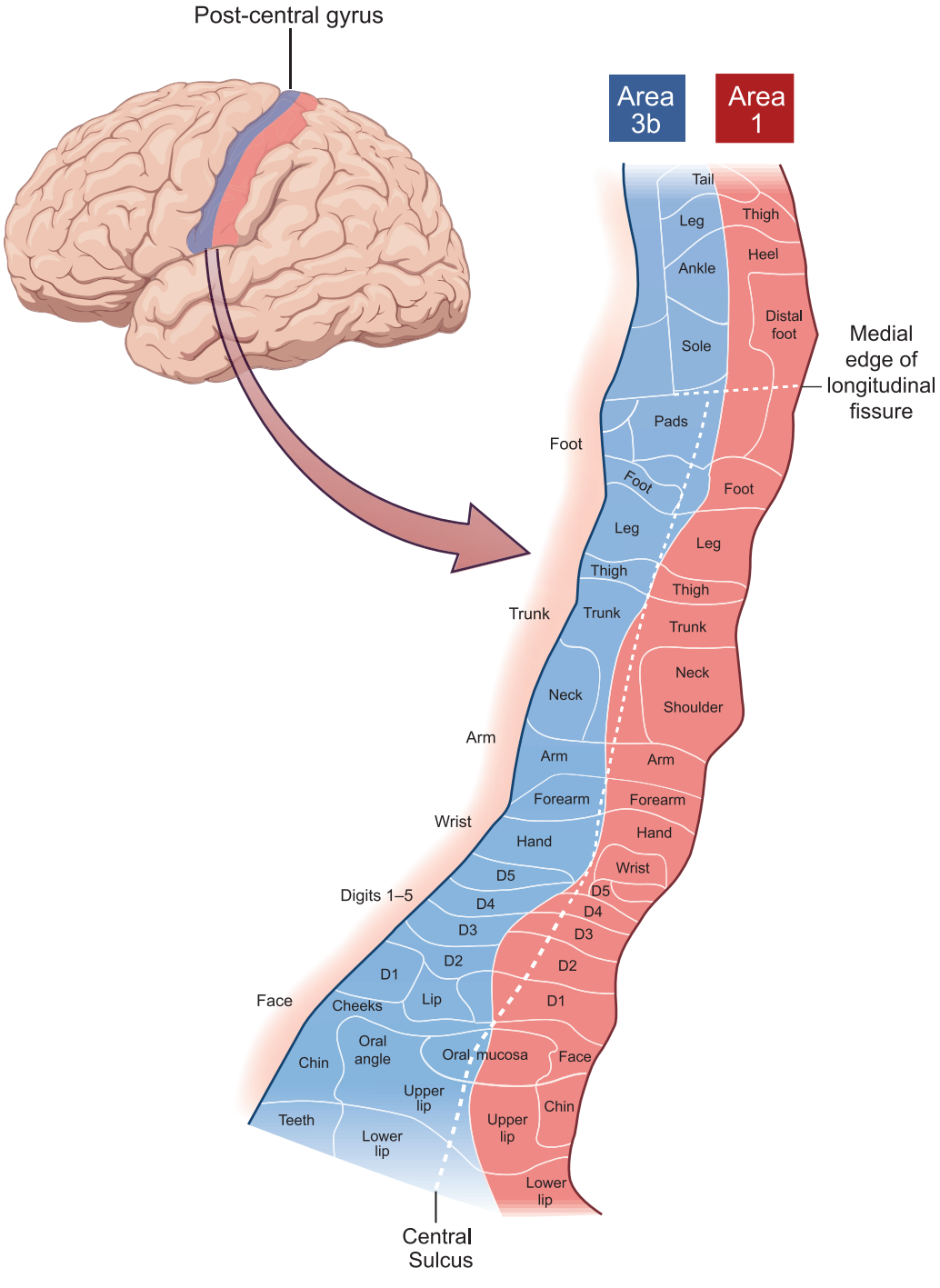


Figure 6–31. Realistic somatotopic organization of the mammalian somatosensory cortex in the postcentral gyrus. Demarcated areas (*white lines*) represent populations of neurons that become active when the corresponding region of the skin is stimulated by some form of tactile input. Note the detailed ordering and organization of the cortex to tactile inputs. Area 3b and Area 1 are two subregions of the expanse of tissue commonly referred to as the somatosensory cortex. Each subregion has its own complete somatotopic map of the body surface. (D = digit of the hand)

example, in humans, approximately 30% to 40% of the entire S1 cortical representation is mapped to structures related to vocalization and speech (Carey, Krishnan, Callaghan, Sereno, & Dick, 2017; Miyamoto et al., 2006). This is a truly staggering amount of cortical tissue, but completely understandable given that speech production is *the* most highly skilled

behavior performed by any animal on earth. Speech has no rival when it comes to its complexity, speed, adaptability, frequency of use, and need for sensory feedback to maintain its functional performance state (Guenther, 2016).

One final cortical feature that isn't obvious from cortical maps, but becomes clear when we start matching the activation of a cortical neuron to a specific form or quality of sensory stimulation in the periphery, is the cortical feature known as columnar organization. We introduced the concept of cortical columns, their origin, and functionality back in **Chapter 5**. To refresh your memory, recall that cortical columns are vertical collections of neurons that span all layers of the cortex (see **Figure 5–13**) (Mountcastle, 1997; Sur, Wall, & Kaas, 1984). Neurons within a column receive inputs from a common location of the body and respond to the same type of sensory receptor input. For example, a hypothetical cortical column could obtain information from the skin of the upper lip and from sensory receptors that transduce low frequency vibrations from that region.

In the somatosensory cortex, a columnar organization is clearly present. Within the somatosensory representation of a body part, cortical columns are arranged based on the adaptation property of the primary afferent. For example, as illustrated in **Figure 6–32**, digits of the hand are represented

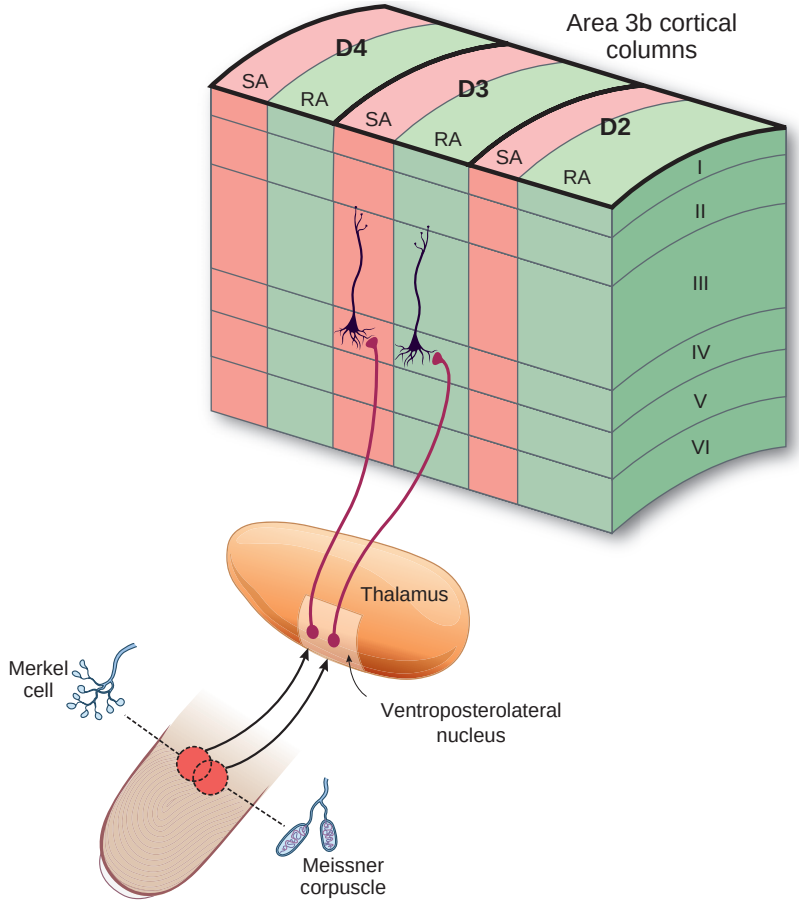


Figure 6–32. Columnar organization of the somatosensory cortex. Somatosensory cortical columns are arranged based on the original adaptation properties of the primary afferent. Shown in this block of cortical tissue are the representational zones for digits 2 (D2), 3 (D3), and 4 (D4) of the hand. For a given digit, alternating columns of cortical cells are present that receive either slowly (red shading) or rapidly (*green shading*) adapting inputs from the periphery. The origin of these inputs is from sensory receptors and primary afferents in the skin (see the Merkel and Meissner endings in the finger illustrated below the thalamus). The columnar organization of S1 reflects the segregation of slowly versus rapidly adapting responses in the VPL and VPM nuclei of the thalamus. Thalamic inputs to S1 are through layer IV of the cortex.

in S1 by adjacent areas of tissue in an orderly manner, such that digits that lie next to each other in the hand also lie next to each other in their cortical mapping. For a given digit, alternating cortical columns are present that receive either slowly (red shading) or rapidly (green shading) adapting inputs. The columnar organization of S1 is a reflection of the segregation of slowly versus rapidly adapting responses found downstream in the VPL and VPM nuclei of the thalamus. In turn, the segregation of SA and RA inputs to the thalamus can be traced further downstream until returning to the original primary afferent in the skin of the finger (Sur et al., 1984).

S1 Possesses Four Complete Cortical Body Representations

As shown in **Figure 6–33**, S1 consists anatomically of four Brodmann's areas (BAs): 3a, 3b, 1, and 2. Each subarea within the somatosensory cortex (a) receives unique forms of somatosensory information from different subdivisions of the ventrobasal complex (VPL and VPM) of the thalamus, (b) possesses different degrees of reciprocal connections with other cortical and subcortical targets involved in sensorimotor control (reciprocal means back-and-forth axon connections that allow for sharing of information between two areas), and (c) possesses a complete somatotopic and separate representation of the peripheral receptive field of the body (Kaas, Nelson, Sur, Lin, & Merzenich, 1979). These differences suggest a distinctive functional property for each of the four S1 areas.

As illustrated schematically in **Figure 6–34**, BA 3a, 3b, 1, and 2 receive input from different regions of the ventral thalamus. BA 3a and 2 receive primarily proprioceptive inputs from the body via the VPL and VPM thalamus. BA 3b and 1 obtain inputs from slowly and rapidly adapting cutaneous mechanoreceptors via the VPL and VPM nuclei. As shown by the horizontal arrows in the middle panel of **Figure 6–34**, areas 1 and 2 also receive inputs, besides those from the thalamus, from areas 3a and 3b via short association fiber connections. This input arrangement suggests that areas 1 and 2 are higher-order processing zones of the somatosensory cortex that operate to combine raw information directly from the thalamus with similar, but slightly processed, information from areas 3a and 3b. The integration of these two different, yet related input streams may be the first steps in abstracting more complex tactile features from our raw somatosensory inputs. The more complex processing likely performed in areas 1 and 2 also suggests that these regions may hold more complex integrative operations for perception, motor control, and learning (Reed, Klatzky, & Halgren, 2005). For example, it has been observed that area 3b projections into area 1 are related to the perception of textures of objects, whereas 3b inputs to area 2 are related to the perception of size and shape of handled objects (Hsiao, 2008). Because of these processing differences, damage to S1 results in specific, yet unique perceptual deficits of sensory detection and discrimination corresponding to the loss.

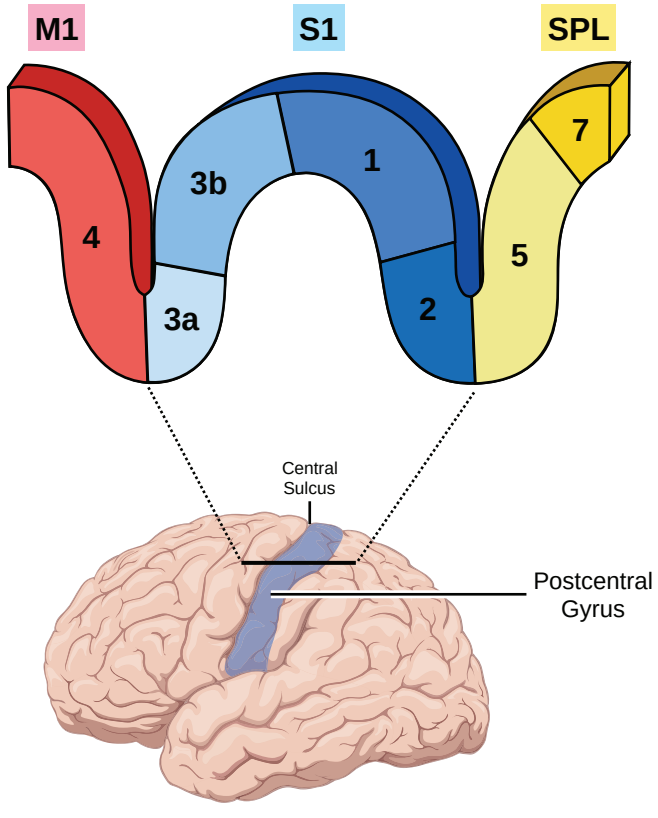


Figure 6–33. S1 consists anatomically of four Brodmann's areas (BA): 3a, 3b, 1, and 2. Each subarea (a) receives unique forms of somatosensory information from different subdivisions of the ventrobasal complex of the thalamus, (b) possesses different degrees of connectivity with other brain regions, and (c) possesses a complete somatotopic representation of the peripheral receptive fields. BA 3a and BA 2 receive primarily proprioceptive inputs from the body via the VPL and VPM thalamus. BA 3b and BA 1 obtain inputs from slowly and rapidly adapting cutaneous mechanoreceptors via the same thalamic nuclei. Adjacent to the somatosensory cortex is BA 4 (primary motor cortex), and BA 5 and 7 (superior parietal lobule).

Speech-Related Features of S1

With specific regard to speech, the representation of the contralateral side of the face and vocal tract occupies the ventral-most region of the postcentral gyrus (see **Figures 6–30** and **6–31**). This region of the cortex constitutes the primary receiving area for somatosensory information originating from slowly and rapidly adapting mechanoreceptors located within the skin of the face in both primates and humans. Thalamocortical neurons in the VPM have been found to project into area 3b with the orofacial representations located dorsal to that of the tongue and teeth (Iyengar, Qi, Jain, & Kaas, 2007; Kaas, Qi, & Iyengar, 2006). In humans, brain imaging studies have confirmed the existence of distinct somatosensory representations of vocal tract elements in the

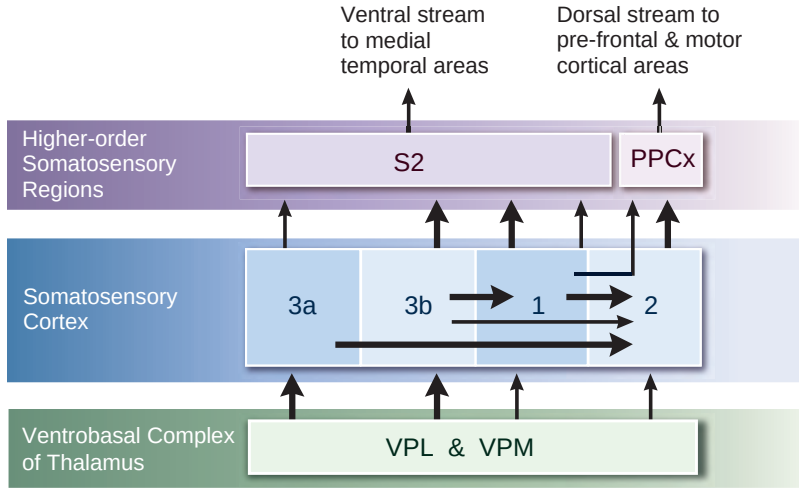


Figure 6–34. Information flow from the thalamus through S1 and beyond. The weight and direction of the arrows linking different parts of the figure represent the relative strength of contribution for an input to a target location. VPL and VPM project heavily to Areas 3a and 3b, respectively. Lesser projections from the thalamus target Areas 1 and 2. Horizontal projections between areas within S1 illustrate hierarchical processing of inputs. Projections from S1 to S2 form the entry to the ventral somatosensory processing pathway that runs through temporal association areas. Projections from S1 to the posterior parietal cortex (PPCx) form the entry to the dorsal somatosensory processing pathway that ultimately outputs to prefrontal and motor cortical areas.

ventral aspect of S1 on the postcentral gyrus (Carey et al., 2017; Miyamoto et al., 2006).

Neurons within the facial representation of S1 are known to alter their firing rate during a variety of oromotor behaviors (McClean et al., 1990). The changing activity of S1 neurons has been correlated with features of orofacial movement, including position, force, direction, and velocity. The importance of these sensory and motor correlations is highlighted by studies that have applied cold temperatures to cool down S1 neurons to levels where they are no longer active. This procedure, known as reversible cold block, effectively stops the influence of sensation on movement control. For example, reversible cold block application to S1 neurons conveying tactile information from the tongue profoundly degrades the fine motor control of tongue movements in primates. This outcome suggests that somatosensation contributes significantly to the normal operation of lingual motor control systems (Lin, Murray, & Sessle, 1993).

Outputs From the Somatosensory Cortical Areas

Output from S1 is directed to one of several different nearby cortical areas that represent a mixture of unimodal and multimodal association cortices (see **Figure 6–34**). Projections from S1 are directed serially to a unimodal area known as the **secondary somatosensory cortex** (S2) (Pons, Garraghty, Friedman, & Mishkin, 1987; Pons, Garraghty, & Mishkin, 1992) and to multimodal regions in the posterior parietal cortex corresponding anatomically to the superior parietal lobule (BA 5 and BA 7) and the inferior parietal lobule (BA 40 and 39) in humans (Romo & Salinas, 2001). If you recall from our discussion in **Chapter 5** on the dorsal and ventral processing routes for sensory areas in the cerebral cortex, somatosensory systems possess a ventral route directed toward the parietal association area that is related to spatial location and considered important for object recognition (Reed et al., 2005).

The secondary somatosensory cortex (BA 43) is located ventral to S1 and can be found on the upper lip and bank of the lateral sulcus, almost directly above the primary auditory cortex (see **Figure 5–8** for location). S2 is believed to activate strongly during touch and manual manipulation of objects for the purpose of recognition (Romo & Salinas, 2001). S2 activity has been observed related to perceptions of object features such as texture and shape. The firing of S2 neurons does not appear to provide very specific information about these object properties, but rather more generalized information related to the motivation of the animal and the context of the tactile exploration. This form of activity by S2 neurons is interesting especially if you take into account that S2 is reciprocally connected (information flow in both directions

between the connected sites) to the prefrontal cortex and the premotor areas (Rojas-Hortelano, Concha, & de Lafuente, 2014). It has been hypothesized that S2 sends object-related information to the frontal lobe. In turn, the frontal lobe feeds back to S2 and changes the responsiveness of these sensory neurons. The changes made by cognitive areas of the frontal lobe to S2 neurons are believed to allow us to make better informed decisions and judgments about the objects we are currently manipulating or will handle in the near future (Romo & Salinas, 2001).

S2 may also have a role in remembering object features. Anatomical studies have shown that S2 is not only connected to the frontal lobe, but also to the medial temporal lobe (via the insula) and specifically to the hippocampus (Romo, Hernandez, Zainos, Brody, & Salinas, 2002). As we have described, if S2 is integrating cognitive factors related to object recognition, it may also be the case that S2 is acting in the decision-making process of what tactile features to remember for future use (Newman, Klatzky, Lederman, & Just, 2005; Romo & Salinas, 1999). Here's an example to help you appreciate S2's two basic functions more easily. Let's say you're at the grocery store shopping for fruit to make a fruit salad for a party. You haven't quite decided yet which fruits to include, so you walk around the produce section picking up and inspecting different fruits you might buy. While inspecting the fruits, you are keying in and selecting a few important object features about each (texture, firmness, shape) based on your preferences and ideals for fruit. When you are ready to decide which fruits to buy, the remembered features you pinpointed during your inspection are used to make a series of judgments on which specific fruits to buy for making your fruit salad. S2 came in pretty handy (excuse the pun) in this particular situation. S2 is likely one of the origination points for the somatosensory ventral stream into the temporal association cortices (see **Figures 5–16** and **6–34**) (Reed et al., 2005). Its role in object recognition and memory are consistent with the general goals of ventral processing pathways in the cortex (Rojas-Hortelano et al., 2014).

Posterior Parietal Lobe Receives Inputs From Primary and Secondary Somatosensory Areas

The posterior parietal cortex is an expansive area of the parietal lobe directly behind the postcentral gyrus. Within this region are four key BAs of interest to our discussion on somatosensory processing: BA 5 and 7, found within the superior parietal lobule; and BA 40 and 39, of the inferior parietal lobule (see Brodmann's area cortical mapping in **Figure 5–12**). Neurons within BA 5 receive cutaneous inputs that convey information regarding body posture, and proprioceptive inputs that provide information on motion of the limbs (Pearson & Powell, 1985). Area 5 cells integrate this information and activate during intentional and goal-directed reaching motions, such as grabbing your soda off the table. Area 7 neurons receive a wide variety of somatosensory inputs and integrate these with visual signals that correspond to the timing and location of the somatosensory inputs. Area 7 becomes highly activated when an individual is looking at his or her own hand while simultaneously manipulating an object. In other words, these neurons are important in the development of hand-eye coordination. The neurons of both areas 5 and 7 project their output to prefrontal cortex, premotor, and primary motor areas of the frontal lobe to influence the decision and development of goal-directed actions (see **Figures 5–16** and **11–2** for information flow example).

Area 5 of the superior parietal lobule and the supramarginal gyrus of the inferior parietal lobule (BA 40) are regions known to simultaneously receive both motor control instructions directly from the frontal lobe and somatosensation from the postcentral gyrus. The motor control instructions from the frontal lobe are carrying a "copy" of the commands sent to lower motoneurons in the brainstem or spinal cord for an intended action. This copy of motor information is referred to as **corollary discharge** or, more colloquially, **efference copy** (Crapse & Sommer, 2008; Freedman & Ibos, 2018; Sommer & Wurtz, 2008). Corollary discharge operates to "tip off" or inform parietal regions as to what is about to happen (intent) based on remembered sensorimotor action plans developed through motor learning. Alternatively, the somatosensory inputs from S1 into areas 5 and 40 represent sources of sensory feedback that provide these same neurons with the information needed to verify that the goals (intent) of the action were met during its actual performance. The simultaneous presence of both the corollary discharge and sensory feedback signals allows neurons in areas 5 and 40 to compare the intent of an action with the sensory consequences (feedback) of the action's performance. Depending on the outcome of that comparison, if the intent and the sensory feedback from the performance match, the skill is strengthened. On the other hand, if a mismatch is noted between the intent and sensory feedback, a corrective response is triggered to restore the proper relationship between the two inputs and to help improve future instances of the action.

The one part of this story that is not entirely obvious to students is how exactly neurons in areas 5 and 40 perform the comparison between the corollary discharge and the somatosensory feedback signals. In other words, how is the neuron judging these two different forms of information? How is it comparing a motor signal with a sensory signal to arrive at a conclusion as to the accuracy of a match? To more fully understand how this comparison process works within areas 5 and 40, you have to appreciate that when we learn a new skill or behavior, we are learning not only how to perform the action motorically, but also what the action should *feel* like when it is performed. Motor control always produces some form of sensation that is correlated or synchronized in time with the performed movements. These sensations are subsequently used to help refine and improve future performances.

Learned action plans are believed to be represented in the frontal cortex as a combined "sensorimotor" plan. Thus, when the posterior parietal cortex receives the corollary discharge of an upcoming action from motor cortical areas, what these neurons are really getting is both the intended motor instruction *and* the intended sensations that have been learned and associated with that intended action (Romo et al., 2002). As such, the comparison being conducted by neurons in areas 5 and 40 is between the portion of the corollary discharge signal that reflects the learned "feeling" of the action and the real-time "feeling" produced by the performance attempt. Although areas 5 and 40 are performing similar comparative tasks, the action associated with each area is different. Area 5 uses this comparative process on behaviors related to hand and arm motion, such as reaching and grabbing, while area 40 performs these same actions during speech production.

Once again, the important question to answer is why these processes are important for you to understand. Let's use an example to start addressing this question. Pretend that you are standing on a beach, and on the sand in front of you is a large inflatable beach ball. How do you go about picking up the beach ball from the ground? Because you have experience gained throughout your lifetime of picking up large objects, you automatically bend over, widely stretch out both of your arms and hands to the size of the ball, and lift it up. What if you had to pick up a small marble instead? How would your response need to change? You would still automatically bend over, but this time you would use one arm, a hand, and two or three fingertips to pick up the marble from the ground. For both conditions, your posterior parietal areas (specifically area 5 in these cases) use the comparison process described earlier to predict what the objects would feel like when you picked them up. The posterior parietal cortex used this prediction to help form the correct posture and reaching strategy to succeed in both conditions.

Now, imagine that you no longer had the ability to use these comparative processes to predict the appropriate response to a situation. Using our ball analogy, you would be left in a condition where every time you encountered a ball lying on the ground, you would have to go through the entire process of trial and error to figure out *de novo* how to produce the right reaching and grabbing action to pick up a ball of a given size. This would be enormously frustrating and slow! Everyday reaching actions that we take for granted would become arduous attempts at trying to figure out the exact way to accomplish a reaching goal. Put this example into the

Box 6–3. Clinical: **Cognitive reserve**

Cognitive reserve can be defined as resiliency to the development and/or effects of neuropathology in the brain (Antoniou, Gunasekera, & Wong, 2013). The theory of cognitive reserve originally arose to explain discrepancies noticed between levels of brain pathology and the high levels of clinical and cognitive functioning in some patients. One compelling example was of 10 cognitively healthy seniors who were discovered to have neurofibrillary plaques associated with Alzheimer's disease upon autopsy. The brains of these women were found to have greater mass and more gray matter than both their peers with dementia and their peers without any neuropathology. Their larger brains and extra neurons were hypothesized to have provided these individuals with a neural "reserve" that likely helped them cope with the Alzheimer's-induced plaques to the point that observable disease factors were virtually undetectable (Gold, 2015; Soldan, Pettigrew, & Albert, 2018).

Cognitive reserve postulates that certain variables and experiences may improve the ability of the brain to cope with damage, mitigating the negative effects of damage on cognitive functioning. Such variables may include educational level, occupational status, and the involvement in physical, intellectual, and social activities (Gold, 2015; Guzmán-Vélez & Tranel, 2015). Individuals who participate in intellectually demanding tasks over the course of their lives likely experience significant neuroplastic changes that underlie the mitigating effects of neuropathology. Such changes presumably operate to lessen the impact of damage by enabling the brain to make more efficient use of the reserve processing power of impaired cerebral resources, thus minimizing the impact of deficits on functional cognitive abilities (Schweizer, Ware, Fischer, Craik, & Bialystok, 2011).

Resources

Antoniou, M., Gunasekera, G. M., & Wong, P. C. (2013). Foreign language training as cognitive therapy for age-related cognitive decline: A hypothesis for future research. *Neuroscience & Biobehavioral Reviews*, *37*(10), 2689–2698.

Gold, B. T. (2015). Lifelong bilingualism and neural reserve against Alzheimer's disease: A review of findings and potential mechanisms. *Behavioral Brain Research*, *281*, 9–15.

Guzmán-Vélez, E., & Tranel, D. (2015). Does bilingualism contribute to cognitive reserve? Cognitive and neural perspectives. *Neuropsychology*, *29*(1), 139–150.

Soldan, A., Pettigrew, C., & Albert, M. (2018). Evaluating cognitive reserve through the prism of preclinical Alzheimer disease. *Psychiatric Clinics of North America*, *41*(1), 65–77.

Schweizer, T. A., Ware, J., Fischer, C. E., Craik, F. I., & Bialystok, E. (2011). Bilingualism as a contributor to cognitive reserve: Evidence from brain atrophy in Alzheimer's disease. *Cortex*, *48*(8), 991–996.

context of speech gestures, and you can begin to appreciate the critical importance that predictive abilities have for behaviors like speech, which are fast and complex. Without the ability to rely on your past experiences to help you predict future speech-related actions, speech production would grind to a halt. I think the point is clearly made that without the comparison processes described here, your ability to produce rapid and accurate actions would be severely limited.

Neuroplasticity: Changes to the Structure and Function of the Brain

The discovery that the mammalian brain is capable of lifelong adaptation based on the experiences an animal encounters and engages in within their environment has revolutionized the way animal behavior (which includes humans too) is conceptualized. A wide variety of factors that include features of normal behavior, enrichment, maladaptive experiences, injury, and disease have been shown to cause significant changes in the structure and functioning of the cerebral cortex in animals—a process often referred to as **experience-dependent cortical reorganization**. Supported by ample evidence from the cellular through the systems level, it is now well recognized that cortical reorganization falls under the broader umbrella of **neuroplasticity**, the general ability of the nervous system to continuously change and adapt (structurally and functionally) in a context-dependent manner to changing environmental conditions. Neuroplasticity is a fundamental neurobiological process that allows us to adapt sensorimotor performance and behavior throughout our lifetime (Hallet, 2005; Kleim & Jones, 2008; Nudo & Milliken, 1996). Our current appreciation of neuroplasticity is as a process that is not triggered by some specific situation, but can be thought of instead as the default operating state of the brain. Generally speaking, neuroplasticity, in its many forms, *is* the biological process underlying the way animals learn and continuously adapt to changing environmental conditions or novel situations.

The term neuroplasticity comes from the idea that objects that are "plastic" are moldable and flexible in their form and shape. Given this grand idea, how do we study changes in the cortical representations that underlie the sensory and motor behaviors performed by our bodies? When we discuss the idea of cortical representations, we literally mean the way in which neurons in the cortex are "wired" and functionally connected to our sensory endings in the periphery or to our muscles. The method typically employed to study the normal form and changes to cortical representations consists of creating a baseline understanding of how a given cortical area is "wired" to those sensory endings or muscles. What do we mean by the notion of creating a *baseline understanding*? It means something a little different depending on whether you are discussing sensory or motor systems. (For the purposes of this chapter, we'll focus only on the method used for studying sensory-related neuroplasticity. Methods used to study neuroplastic changes in motor systems are covered in **Chapter 11**.) For sensory systems, it means that researchers need to use microelectrodes to painstakingly record the electrical activity of thousands of individual neurons in the cortex while simultaneously using different forms of sensory inputs to excite peripheral sensory endings. The basic idea is to relate sensory stimulation occurring in the periphery with evoked neuron activity in the cortex. This method characterizes how and what inputs a cortical neuron is receiving and thus processing at any instant in time. As you can imagine, this method is akin to finding a needle in a haystack comprised of neurons; but because we know that S1 is somatotopically organized, the search area that a given neuron is mapped to in the periphery is usually fairly manageable. For example, if I'm interested in determining the firing properties of cortical neurons mapped to the lower lip for different forms of sensation, I need only to stimulate the skin of the lower lip and not the skin of the entire face.

After creating a baseline mapping for thousands of neurons, the next step in the process is to experimentally alter those inputs going to the region of the cortex being examined. Experimental modifications of sensory inputs can include factors ranging from intentional injury or the restricted use of a body part, to enhanced and enriched sensory experiences. The last step of the process entails reassessing the entire cortical area under investigation for any changes that might have occurred during the experimental condition using the same testing methods employed originally during the baseline part of the study. Factors that are often examined include: (a) changes in a given cortical neuron's response to a stimulus input, (b) changes in the number of neurons that are activated by a given sensory input, and (c) changes in the size of a neuron's receptive field. In essence, cortical neuroplasticity studies are a lengthy exercise in comparing the function and structure of a given cortical area *before and after* some type of experimental experience or condition has been applied. With a general appreciation of the method used to study sensory neuroplasticity, let's review a handful of classic experiments that helped define and inform our current understanding of the brain's remarkable ability to change itself. Specifically, we will review three studies that helped us understand the role of neuroplasticity after injury, as a consequence of enhanced experience, and as a function of normal use.

Somatosensory Cortex Receives Diffuse Projections From the Thalamus

Studies during the 1980s investigating the mechanisms and features of cortical neuroplasticity in adult primates revealed that sensory brain representations are capable of remarkable reorganization (structural and functional change) in response to different patterns of behavioral experiences transduced through our somatosensory system (Jenkins, Merzenich,

Ochs, Allard, & Guic-Robles, 1990; Merzenich et al., 1983; Schwaber, Garraghty & Kaas, 1993). These studies were performed mostly using the adult primate hand and its associated somatosensory representation in S1. As illustrated in **Figure 6–35 A & B**, under normal conditions, each finger or digit of the primate hand (**A**) is mapped to a discrete population of neurons within the S1 cortex (**B**). Zooming into the normal cortical representation of the digits in S1 (see **Figure 6–35C**), each colored zone represents a population of neurons that is responsive to tactile stimulation of the glabrous (nonhairy) skin of each finger as determined using the mapping method outlined earlier.

One of the earliest experiments revealing the possible magnitude of S1 cortical plasticity in the adult primate demonstrated the consequences of removing all afferent input from a given body part (**Figure 6–36**). As shown schematically in **Figure 6–36**, surgical removal of the entire third digit from a primate hand (D3) alters the normal cortical mapping of the fingers in a significant manner. Following the surgical amputation of D3 (see **Figure 6–36B**), it was found that the representations of adjacent fingers (D2 and D4) expanded into those regions of the cortex that were formerly occupied and mapped to the removed digit (Merzenich et al., 1984) (see **Figure 6–36C**). In other words, neurons that once responded only to tactile inputs from D3 were now responding to inputs coming from D2 and D4. This was a remarkable finding that would take investigators several years to unravel. The search for the underlying cause of this cortical expansion would lead to a fundamental discovery of how the thalamus communicates and innervates S1.

The rapid representation changes following surgical amputation of the digit were eventually found to have been caused by how the thalamus feeds information to the overlying sensory cortex and how neurons in the cortex manage those thalamic inputs (Garraghty & Kaas, 1991). Recall that the thalamus receives inputs from specific areas of the skin (see bottom part of **Figure 6–32**). It was once thought that the thalamus simply relayed these specific peripheral inputs to distinct areas of the S1 cortex, thus helping to account for the neat and highly organized appearance of S1 cortical maps obtained from older neuron recording studies (see top part of **Figure 6–32**). With the results of the amputation experiment, though, investigators realized that this explanation was simply not possible. If the thalamus did project to S1 in a neat and orderly manner, how in the world did the neurons that were once responsive to D3 suddenly become responsive to D2 and D4? The answer is that the thalamus does not project to the S1 cortex in a specific manner, but instead projects in a more diffuse pattern to several adjacent cortical representation zones at once (Garraghty & Kaas, 1991). Let's use **Figure 6–37** to illustrate what we mean by a diffuse projection pattern of thalamic inputs to the cortex.

Illustrated in **Figure 6–37A** are the cortical representations for D2 (red), D3 (blue), and D4 (black) with their respective thalamic inputs from the VPL nucleus color coded to each cortical zone. Notice that while most of the inputs from a given thalamic neuron innervate the corresponding location in S1 (red thalamic neuron innervates the red shaded D2 zone, blue thalamic neuron innervates the blue shaded D3 zone, and black thalamic neuron innervates the

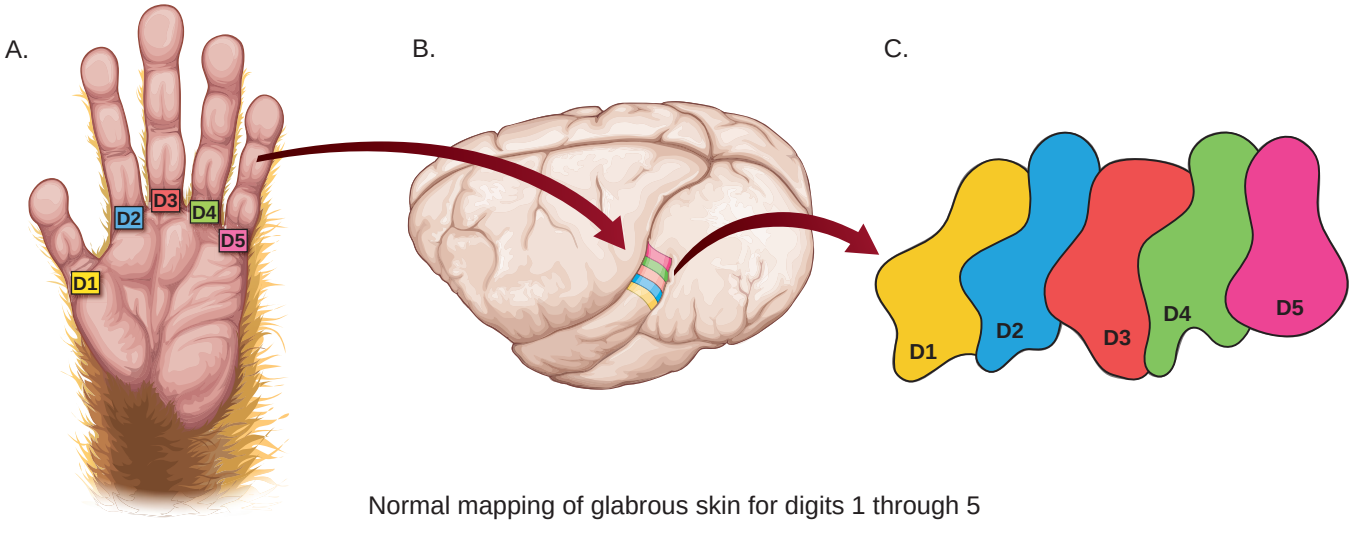


Figure 6–35. Normal somatosensory cortical representation of the primate hand. **A.** The glabrous (nonhairy) skin of the primate hand with each finger (digit) labeled from left to right, D1 through D5. **B.** Lateral view of a primate brain with the somatotopic organization of the digits illustrated on its surface and color coded to the digit labels from A. **C.** Expanded view of the digit S1 cortical mapping. Digit zones are color coded and labeled as in A.

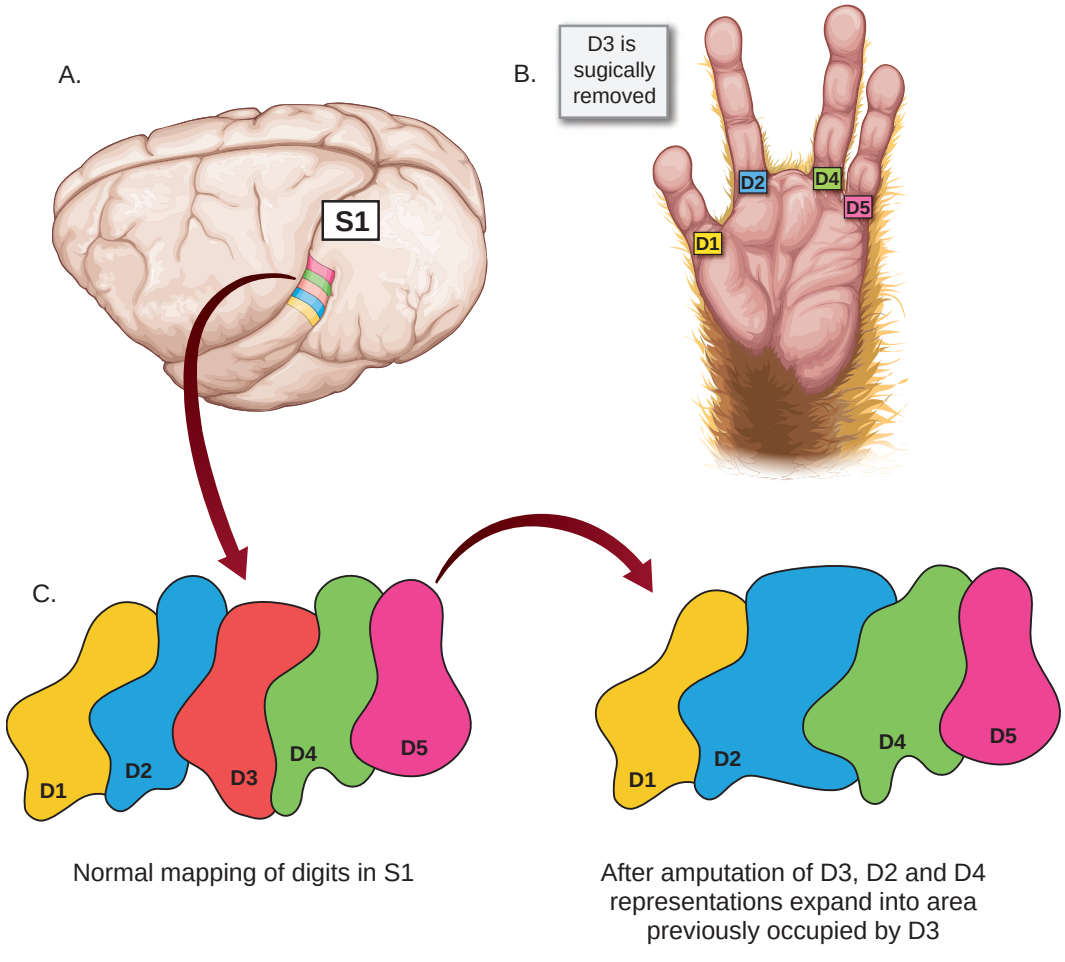


Figure 6–36. Somatosensory cortical representation of the primate hand before and after D3 amputation. **A.** Lateral view of a primate brain with the somatotopic organization of the digits illustrated on its surface and color coded as in **Figure 6–35**. **B.** The glabrous (nonhairy) skin of the primate hand with D3 surgically removed. **C.** Expanded view of the digit S1 cortical mapping before and after D3 removal. Digit zones are color coded and labeled as in A. Note the expansion of D2 and D4 into the cortical zone that was once occupied by D3.

black shaded zone for D4), some axon branches from each thalamic neuron cross over and innervate adjacent cortical areas in the following pattern: (a) axon branches of D2 overlap into the cortical zone for D3; (b) axon branches of D3 overlap into D4; and (c) branches of D4 overlap into D3. In other words, a thalamic neuron will have a dominant input or set of projections to its corresponding cortex (e.g., blue VPL neuron projecting mostly to the blue D3 zone) and several minor projections to cortical neurons in adjacent areas of cortex (e.g., the blue fibers that overlap into the red and black zones). Because the VPL and VPM thalamic nuclei are known to excite cortical neurons using glutamate, in order to maintain the normal pattern and form of the somatotopic organization in S1, the overlapping projections from a given thalamic neuron must be actively inhibited by those neurons within an overlapped cortical zone.

With this idea in mind, when D3 was amputated in the experiment described earlier, the thalamic input for D3 (blue neuron) was essentially silenced because the body area that fed sensory input into that thalamic neuron no longer existed. What this effectively caused in the D3 cortex were two events simultaneously: (a) the release of neurons in the D3 zone from their usual excitatory inputs (see **Figure 6–37B**), and (b) the release from inhibition of overlapping projections from D2 and D4. Together, these events allowed for the preexistent overlapping branches from D2 and D4 to now start exciting those deprived neurons in the former D3 zone (see **Figure 6–37C**). Thus, the apparent expansion of D2 and D4

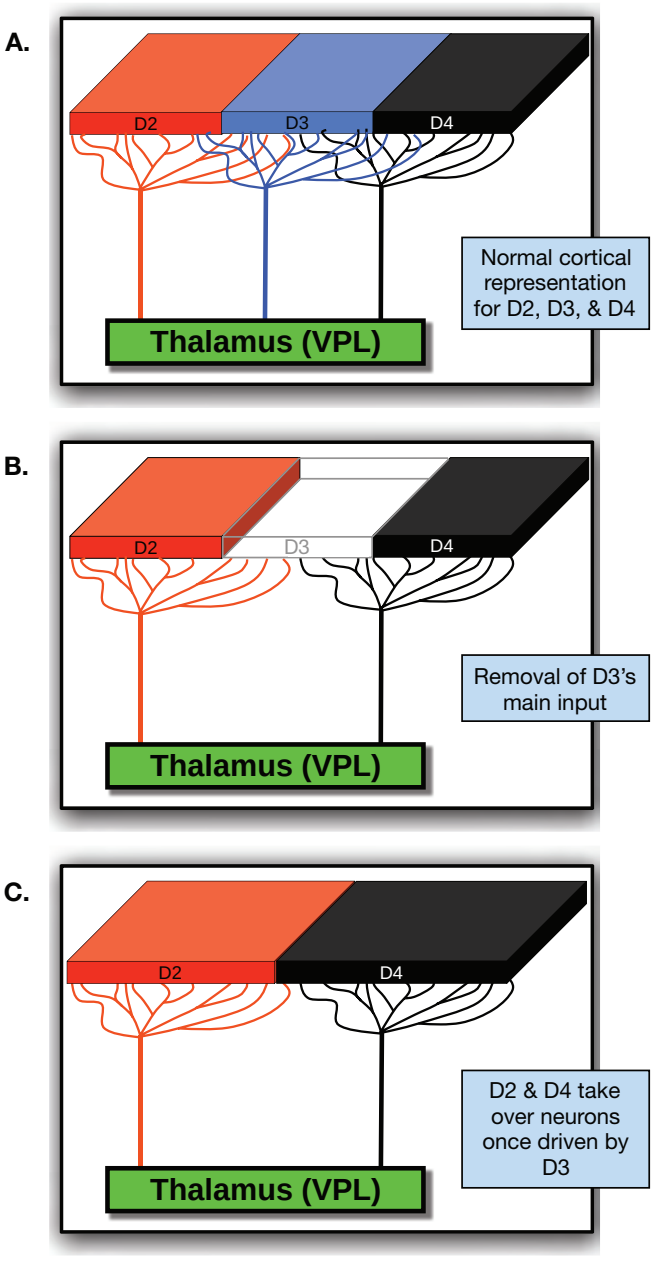


Figure 6–37. Thalamocortical projections from the ventroposterolateral nucleus (VPL) to the D2, D3, and D4 cortical representations in S1. **A.** Each cortical zone receives a dominant majority of input from a specific thalamic neuron (follow each thalamic neuron to its corresponding color in the cortical innervation zone). Note that each thalamic neuron's projections overlap adjacent cortical zones to some extent. **B.** When D3 is amputated, the sensory input to the blue thalamic neuron from A. is removed. This results in the loss of input to the cortical region that was responsive to the thalamic input from D3 (the blank area between D2 and D4). **C.** Existing overlapping branches are now exciting the cortical zone once mapped to D3, creating the apparent expansion of the D2 and D4 cortical zones noted in **Figure 6–36**.

into the neural zone that previously represented D3 was the result of (a) an existing or latent pattern of innervation from the thalamus, and (b) a change in the balance of excitatory and inhibitory activity among all these neurons. Discovering this more complex relationship between the thalamus and S1 has been instrumental in helping explain many past and current experimental findings in the neuroplasticity literature. It has also provided us with a possible mechanism for cortical reorganization as a function of learning and behavioral experience. The next experiment to be described demonstrates this particular idea.

S1 Plasticity as a Function of Enriched Experiences

Normally developing animals do not undergo radical changes to their anatomy except under extreme cases of injury. Instead, most animals are engaged in a process of continual exploration and tactile discrimination of objects in their environment. We pick up various things, hold them in our hands, and actively move our skin surfaces over them to explore and learn about their physical characteristics. These experiences are vital in helping us develop and maintain our perceptual skills (Allard, Clark, Jenkins, & Merzenich, 1991; Garraghty & Muja, 1995). A clue to discovering how perceptual skills emerge can be found in reports of neural plasticity that have demonstrated cortical reorganization solely as a consequence of enhanced sensory experience and training (Jenkins et al., 1990; Recanzone, Schreiner, & Merzenich, 1993).

In one of the early studies investigating how enhanced experience could alter sensory cortical representations, primates were trained to obtain a food reward by actively contacting a textured and rotating stimulus disk that tactilely stimulated the distal ends of a number of their fingers (Jenkins et al., 1990). To help you visualize this study, imagine an experimental setup akin to a primate sitting in a chair in front of an old-style vinyl-record player that is spinning a disc of fine grain sandpaper. While the disc was spinning, this animal would have to reach out and lightly place his or her fingertips on the spinning disc. The key part of this experiment was not just the tactile stimulation of the fingertips, but rather that the primate had to pay very close attention to which of fingertips were on the rotating disc and the force of the contact. The primate received a food reward only if the animal contacted the rotating disc very lightly with the fingertips of D2 and D3 for a set period of time. These requirements made the task much more challenging for the animal, necessitating high levels of attention to the behavioral conditions needed to receive the food reward.

Following several weeks of training, a dramatic enlargement in the representational area reflecting this discrete experience was recorded from SI. Shown in **Figure 6–38** is a representation of the cortical changes that emerged during this study. As can be seen, fingertip stimulation of D2 and

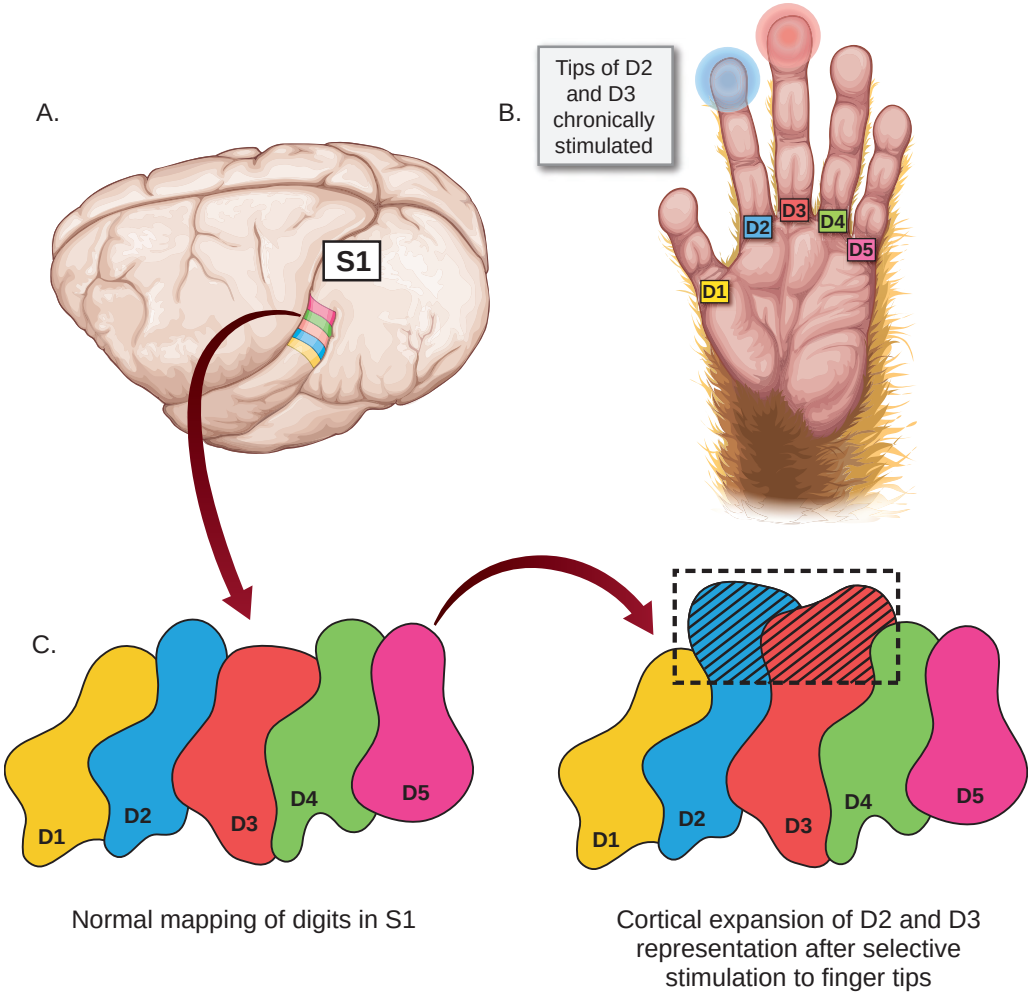


Figure 6–38. Somatosensory cortical representation of the primate hand before and after selective tactile stimulation to the fingertips of D2 and D3. **A.** A lateral view of a primate brain with the somatotopic organization of the digits illustrated on its surface and color coded to the digit labels from **Figure 6–35**. **B.** The glabrous (nonhairy) skin of the primate hand with site indicators where the tactile stimulation was provided during the study. **C.** Expanded view of the digit S1 cortical mapping before and after tactile experience. Digit zones are color coded and labeled as in A. Note the expansion of fingertip areas of D2 and D3 as illustrated by the hatched lines in the dashed box.

D3 (see **Figure 6–38B**) resulted in an expansion of the cortical area mapped to these specific regions of skin only (see the hashed areas of the D2 and D3 zones in **Figure 6–38C**). No other areas of the cortical zone demonstrated any change. Thus, focused tactile experiences produced specific neuroplastic changes only for those populations of neurons that were directly mapped to the peripheral sensory endings being chronically stimulated by the textured disc.

Curious about the possible role that attention could have played in these results, the investigators repeated the entire experiment on a new group of primates, but this time did not require that the primates maintain attention to the task conditions. In the repeated experiment, the hand of the monkey was braced over the spinning textured disc so that the fingertips for D2 and D3 would rest lightly and passively on it. Under this condition, the primate did not need to pay any attention to the task. After thousands of trails, can you guess what happened within the cortical S1 representation for the stimulated fingertips? Nothing. Absolutely nothing. The cortical representation for the fingertips of D2 and D3 showed no change at all, even though the nature of the sensory experience was the same as in the first experiment. The only factor that was different was the attention paid by the primate to its performance (Jenkins et al., 1990).

These results were stunning and clearly pointed to the notion that neuroplastic mechanisms require active engagement and attention by an individual to develop. Passive experiences do virtually nothing to alter brain networks and cortical representations. It was hypothesized that the observed changes in S1 during the first part of the study were driven by the enhanced input coming from the skin of the fingertips during the training. More importantly, attention of the primate to the task conditions may have operated as the catalyst for shifting the balance of activity (excitatory vs. inhibitory) between the thalamic neurons and the cortical cells with which they form a synapse. As you can venture to guess, the implications of these data as they pertain to typical learning and rehabilitation practice are enormous. These results argue strongly for the need and application of therapeutic tasks and learning conditions that engage the patient's attention and that are motivating and challenging as well (Jenkins et al., 1990). Conditions that are passive or that allow the individual to "tune out" will not be effective in creating the necessary brain changes that are needed to alter behavior in any significant manner.

The Timing of Sensory Inputs Are Critical Factors in Changing Cortical Representations

While the enhanced-experience study described in the previous section helped us appreciate the critical role of attention and the specificity of cortical changes to chronic sensory stimulation, we are still left with one last factor to review: How do normal behaviors and experiences operate to change brain representations? In a unique study by Allard et al. (1991), these investigators decided to tackle this question in a most creative manner. To allow for a clear interpretation of how the normal use of one's hand can modify central sensory representations, these investigators decided to alter the anatomy of a primate's hand from having five digits to having only four. Importantly, they accomplished this change in anatomy without injuring or removing any part of the hand, but instead by surgically connecting two digits (D3 and D4) to form an artificial syndactyly (**Figure 6–39B**). A **syndactyl** is a congenital defect of the fingers and toes whereby two or more digits are fused to one another and used as one. By creating what amounted to a "super" finger, the researchers had effectively fashioned a brand new anatomical appendage that the primate now had to learn how to use and that the S1 cortex had to incorporate into its existing normal representational pattern of the fingers.

After the surgery to create the syndactyl, the primates in the test group were returned to their natural enclosures and were free to do whatever primates like to do daily. No training or exceptional experiences were additionally provided; the animals simply learned to use their new hand anatomy on their own through typical everyday experiences. From an experimental standpoint, the connected fingers created a situation whereby sensory inputs produced through normal everyday use would now occur coincidentally on skin surfaces that had distinct and separate cortical representations (see the normal S1 representation in **Figure 6–39C**). Because nothing was damaged or removed from the hand, any changes in the S1 hand representation that ensued from the animals' natural use of the new finger could now be directly examined.

After several months of allowing the primates to learn how to use the new anatomy of their hand, the S1 representation was reexamined. The results were dramatic, to say the least. The once sharp boundaries that existed between D3 and D4 prior to the finger fusion were completely eradicated in the syndactylized hand map (see **Figure 6–39C**). The S1 cortex had effectively come to represent the two fingers as a single anatomical unit. In other words, the S1 cortex now looked and responded as if a primate's hand normally has only four fingers rather than five. In a subset of the primates tested, the investigators decided to separate the fused fingers and return the hand anatomy back to its original condition. After several months of allowing the primates to relearn independent use of the fingers, you can probably guess what happened in the cortex. Strikingly, when the fused digits were surgically separated, the sharp somatotopic borders of each previously fused digit reappeared; the hand representation in S1 returned to normal.

Besides being a really clever and cool experiment, the work by Allard et al. (1991) taught us an important conditional factor that affects and drives cortical plasticity. Temporal correlation of sensory inputs (the coincidental timing and presence of sensory experiences) is believed to be a key factor that alters sensory cortical mappings during normal use. With the digits fused, the primates no longer had independent control over the fingers' movements and sensory experiences. The primates *had no choice* but to learn how to use the two fingers normally and functionally together. This resulted in the two fingers experiencing coincidental degrees of sensation and motion while the primates lived their lives in their enclosure. Simply put, the synchronization of skin use and sensory experiences forced the cortex to change and represent what were once two different anatomical elements as one.

The broader implication of these results is that normal cortical representations are likely maintained and adjusted as a function of the timing (temporal) pattern of sensory inputs to cortical networks of neurons. A typical experience and/or use of a body part during a functional activity generates a wide variety of sensory information simultaneously with the experience or performance of the action. These coincidental sensory inputs are not only transduced simultaneously through our somatosensory system, but are likely used to create, maintain, and "tune" the more extensive and widespread cortical representations that underlie these experiences and behaviors.

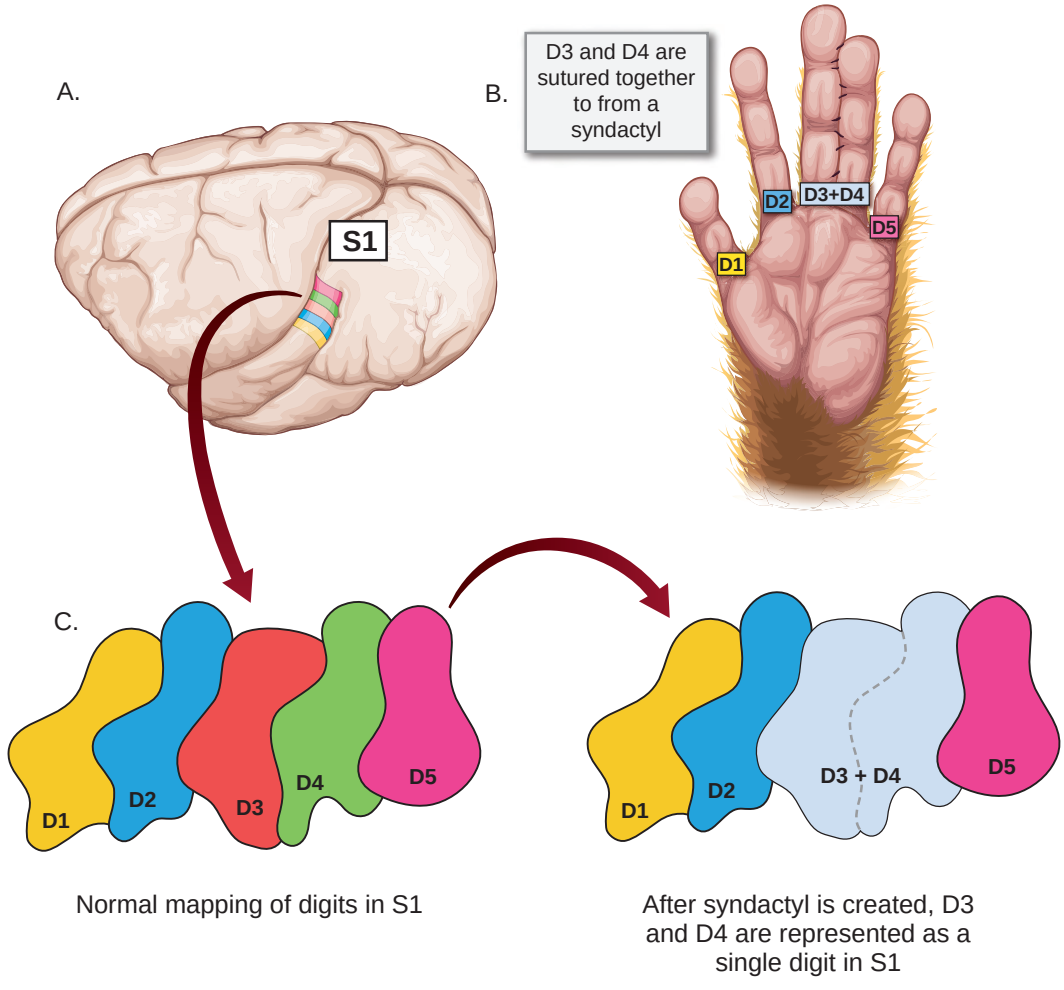


Figure 6–39. Somatosensory cortical representation of the primate hand before and after the suturing together of D3 and D4. **A.** Lateral view of a primate brain with the somatotopic organization of the digits illustrated on its surface and color coded to the digit labels from **Figure 6–35**. **B.** The glabrous (nonhairy) skin of the primate hand with D3 and D4 sutured together to form an artificial syndactyl. **C.** Expanded view of the digit S1 cortical mapping before and after digit fusion. Digit zones are color coded and labeled as in A. Note the merging of the cortical zones for D3 and D4 into a single area. The cortex now represents D3 and D4 as a single anatomical unit.

Implication of Neuroplasticity Literature to Rehabilitation

Consistent with recent ideas in rehabilitation neuroscience, therapeutic-related neuroplasticity of the cerebral cortex after surgery, injury, disease, or resulting from a congenital disorder is likely driven by a variety of behaviorally related sensory events similar to those reviewed in the previous sections (Byl, Merzenich, & Jenkins, 1996). Adaptations in an animal's behavior are thought to exploit mechanisms of neuroplasticity, allowing the CNS to modify and adapt its behavior to integrate new skills while maintaining competency in existing skills (Thelen & Smith, 1996). To support such changes, cortical representations must be able to adapt in a use-dependent and task-specific manner (Nudo, 2003; Nudo, Milliken, Jenkins, & Merzenich, 1996). A variety of different mechanisms are believed to underlie such adaptive plasticity and are likely responsible for the ability of the nervous system to adjust to new environmental conditions, learn new skills, and, most importantly from our perspective, recover from injury (Nudo, 2003).

Current research in the neurosciences has made great strides in unraveling these adaptive mechanisms, discovering that the mammalian brain functionally and structurally

Box 6–4. Clinical: **Top ten principles of neuroplasticity**

Experience-dependent neuroplasticity is an extensive field of study with great relevance to understanding how the brain learns and how it recovers from injury. In a now classic review by Kleim & Jones (2008), the authors successfully distilled decades of research in neuroplasticity into a convenient "top ten" list of major principles. Following is an encapsulated summary of their 10 principles of experience-dependent neuroplasticity. Be sure to check out their entire paper at the reference provided at the end of this box. It is one journal article that is definitely worth your time to read and think about as you train to become a practitioner in communication sciences and disorders (CSD).

Principle 1: Use it or lose it. Neural networks that are not actively engaged and used in behavior become weakened and even lost over time.

Principle 2: Use it and improve it. The opposite of Principle 1, the more we engage neural networks with demanding tasks, the stronger and more resilient neuronal connections become.

Principle 3: Repetition matters. Practice, practice, practice! Plasticity depends on providing enough high-quality experience to neural networks to "outcompete" the brain's usual way of functioning.

Principle 4: Specificity. The form of neuroplasticity that is produced in the brain is determined by the quality of the information that a person engages in regularly. Practicing a skill poorly leads to learning that skill poorly. If I want to learn how to play the piano, flapping my hands for three hours a day isn't going to turn me into a famous concert pianist. In short, practice doesn't make perfect; only perfect practice makes perfect.

Principle 5: Intensity matters. Not only do you have to practice a skill regularly, but you also must practice that skill with high levels of attention and effort during training sessions.

Principle 6: Time matters. Development of changes in the brain as a function of neuroplasticity take time to realize and to establish firmly in the network structure of the underlying behavior being learned.

Principle 7: Salience matters. Neuroplasticity mechanisms are heightened if what you engage in and practice is relevant and important to your life. Doing something with high value leads to greater attention on your part. With greater attention and motivation comes stronger and more comprehensive synaptic changes.

Principle 8: Age matters. Although plasticity has been discovered to be a lifelong and continuous operation of the brain, the younger you are, the better the outcomes are to specific training and learning.

Principle 9: Transference. You can think of this principle as generalization to related learning activities. My son, the professional bassoonist, once told me that learning to play his instrument allows him to play the saxophone and clarinet pretty well because the key patterns are all similar.

Principle 10: Interference. Sometimes learning one skill can actually interfere with your learning of another behavior. You can think of this idea as the "bad habit" principle. Developing compensatory or maladaptive behaviors often makes it much more difficult to learn a skill "the right way" or more adaptively.

Resource

Kleim, J. A., & Jones, T. A. (2008). Principles of experience-dependent neural plasticity: Implication for rehabilitation after brain damage. *Journal of Speech, Language, and Hearing, 51*, S225–S239.

changes to meet the specific performance features of an action (Kleim et al., 2002). As we've seen in the last few sections, sensory cortical reorganization is observed when the patterns of sensory inputs are chronically changed by natural experience or via surgical manipulation (Allard et al., 1991; Jenkins et al., 1990). Collectively, these reports demonstrate that task-specific and use-dependent factors play a major role in helping to shape and maintain the structure and function of the CNS. All these ideas can be exploited intentionally to develop interventions for all forms of normal and disordered conditions.

As suggested by Barlow (1998), our traditional view of speech pathology will require a dramatic shift to embrace and incorporate different, yet related scientific areas. Specifically, the neurosciences are fully capable of contributing much to our profession's conception of therapeutic practices. Behavioral therapy programs, in general, either intentionally or unintentionally take advantage of principles of experience-dependent neuroplasticity whereby patterns of behavior are remodeled and trained through graded and targeted activities taught by the intervening therapist. At their essence, clinical interventions function to organize the sensory and

motor experiences of the patient by controlling the complexity, frequency, and form of various therapeutic tasks (Barlow, 1998).

From the point of view of someone directly involved in the training of students, it is evident that speech pathology needs to make up much ground to more deeply understand and embrace the power inherent in neuroplasticity principles. As I often tell my students, they are "practicing neuroscientists," changing the very structure and function of the brains of those they are working with, regardless of diagnosis. From simple articulation errors to the complex deficits associated with neurological or congenital disorders, all available evidence points to the conclusion that treatments provided by a therapist directly impact the individual's nervous system. Principles of neuroplasticity form the means through which the therapist's impact on the client's nervous system will be realized.

(Previous section was modified from Andreatta, R. D. (2008). Sensorimotor elements of the orofacial system: Reviewing the basics. Perspectives on Speech Science and Orofacial Disorders, *18, 51–61. Used with permission from the American Speech-Language-Hearing Association.)*

TOP 10 The Top Ten List

1. Sensation is the processes by which specialized neural structures, known as sensory receptors, located throughout the body, are activated by environmental and/or internal stimuli. Sensation is a two-step process that transduces and transmits real-world energy of a stimulus into and through the nervous system. Perception is the interpretation of "raw" inputs once they reach the CNS. Perception is at its essence an integrative process that is actively constructed by the brain and dependent on memories of past experiences with a given stimulus or set of stimuli. Perception requires us to possess and use mechanisms that can filter the barrage of sensory information we encounter every moment.
2. The experimental appreciation of sensory systems is made possible through two means of investigation, psychophysics and sensory physiology. Psychophysics is the science relating physical properties of a stimulus to our internal percepts. Sensory physiology is concerned with neural activity associated with a stimulus and how the stimulus is transduced and processed by the nervous system. The combination of psychophysics with sensory physiological approaches has further advanced our understanding of human perception by bridging the gap between our subjective experience and neural activity.
3. All sensory systems are sensitive to and transmit to the CNS four essential attributes that are necessary to fully appreciate any sensory experience: modality, location, intensity, and duration. Modality reflects the different types of environmental energies that are transduced by specific forms of sensory receptors. The transduction of sensory input produces the first abstraction of the real world by the nervous system. Localization of a sensory event is dependent on the receptive field. A receptive field is a discrete region of the body that is "monitored" by a single sensory ending. Intensity and duration factors are based on the magnitude of change in the membrane potential of a sensory ending and the rate of action potential firing it creates. Intensity and duration are based on appreciating the nature of neural codes and their associated patterns of action potential firing.
4. Unlike other sensory systems that mediate one form of real-world energy and that are located in a given area of the body, such as the eye or ear, the somatosensory system mediates a variety of stimulus energies (mechanical, thermal, chemical, and nociceptive) throughout the entire body surface, viscera, and musculoskeletal systems. Anatomic pathways that comprise the somatosensory system include the dorsal-column medial lemniscal pathway, the anterolateral system, and the trigeminal system. These pathways form three parallel routes allowing for the simultaneous transduction and processing of all aspects of an environmental experience involving somatosensation.
5. All somatosensory pathways start with sensory receptors located in the body that are tuned to transduce different forms and types of stimulus energies. Inputs are transmitted centrally along axons with different diameters, degrees of myelination, and conduction velocities. The primary afferent represents the first leg of a three-neuron system responsible for transmitting somatosensation from the outside world into the cerebral cortex. Receptors differ along many dimensions depending on the submodality of somatosensation in question. Differences include the form of energy transduced (mechanical, chemical, or thermal), the form and structure of the receptor, and the distribution of the receptor in the body.
6. The dorsal column–medial lemniscal (DCML) system is a somatotopically organized pathway that mediates tactile sensation and proprioception from all regions of the body via entry through the spinal cord. The

The Top Ten List *continued*

pathway is named for the two major axon tracts that transmit sensory inputs from the spinal cord to the cerebrum. The DCML consists of first-, second-, and third-order neurons. First-order neurons contain the sensory receptor and are responsible for transmitting transduced inputs from the PNS to brainstem nuclei in the CNS. Second-order neurons convey the transduced input through the remainder of the brainstem and to the contralateral thalamus. Third-order neurons transmit input from the thalamus to the cerebral cortex.

7. The anterolateral system (ALS) is not a single pathway like the DCML, but rather a collection of different pathways that mediate nociceptive signals, thermal inputs, and crude forms of touch and deep pressure. The ALS gets its name from the two zones in the anterior and lateral white matter of the spinal cord that house the axons that transmit these forms of sensory inputs. Similar to the DCML, the ALS consists of a three-neuron pathway from the periphery to the cerebrum. The lateral segment of the ALS is needed for the perception of sharp pain, burning pain, cool, warmth, itching, and visceral sensations. This segment sends its output to a wide range of subcortical and cortical areas for integration with other sensory signals. The anterior segment of the ALS transmits inputs related to crude touch and deep pressure to the thalamus and other subcortical locations.
8. The trigeminal sensory system is comprised of three main nerve branches: the ophthalmic (V1), maxillary (V2), and mandibular (V3) nerves. All branches mediate cutaneous tactile inputs, nociception and thermoreception from the facial skin, oral and nasal mucosa, the anterior two thirds of the tongue's surface, the dura mater, the gums, and the tooth pulp. The trigeminal system also transduces proprioceptive inputs from stretch-sensitive receptors found within muscles of the mandibular system and the periodontal ligaments. Proprioceptive inputs are directed to the mesencephalic nucleus. Cutaneous tactile inputs project to the principal nucleus of trigeminal. Nociceptive and thermal inputs project to the spinal trigeminal nucleus.
9. The somatosensory cortex (S1) in the postcentral gyrus of the parietal lobe is considered the chief cortical input site for the modality of somatosensation and all its associated submodalities. The first organizational feature for S1 neurons is that they have an orderly arrangement that reflects the peripheral location of the body's receptive fields. Such an arrangement is critical for unambiguously identifying the peripheral location of any somatosensory stimulus event. The second organizational feature of the somatosensory cortex is that there are differences in the size of neural populations serving different regions of the body. The size of a cortical area allocated to a body part corresponds to a region's sensory sensitivity and type of behavior in which the area participates (skilled vs. gross).
10. Output from S1 is directed in a parallel manner to one of several different nearby cortical areas that include the secondary somatosensory cortex (S2) and multimodal regions in the posterior parietal cortex corresponding to the superior parietal lobule (BA 5 and BA 7) and the inferior parietal lobule (BA 40 and 39). S2 is believed to activate strongly in response to manual manipulation of objects during recognition and in the perception of object features such as texture and shape. Somatosensory inputs from S1 into Areas 5 and 40 are sources of sensory feedback that provide these neurons with the information needed to verify that the goals (intent) of an action were met during performance. All parietal areas project to the frontal lobe to influence the decision and development of goal-directed actions.
11. *(This Top 10 List is special. It goes to 11!)* A wide variety of factors, including features of normal behavior, enrichment, maladaptive experiences, injury, and disease can cause significant changes in the structure and functioning of the cerebral cortex in animals—a process called experience-dependent cortical reorganization. Neuroplasticity is the general ability of the nervous system to continuously change and adapt in a context-dependent manner throughout our lifetime. Sensory cortical reorganization is observed when the patterns of sensory inputs are chronically changed by natural experience or via artificial means. Research demonstrates that task-specific and use-dependent variables play a major role in helping to shape the structure and function of the CNS. From simple articulation errors to the complex deficits associated with neurological or congenital disorders, all available evidence points to the conclusion that treatments provided by a therapist directly impact the individual's nervous system. Principles of neuroplasticity form the means through which the therapist's influence on the nervous system of a patient will be realized.

Study Questions and Activities

- Distinguish between sensation and perception? Why are these ideas are complementary but different from each?
- What do we mean by the idea that "perception is a cognitive event"?
- What is meant by the term "abstraction" in terms of the perception of sensory inputs?
- What factors does the brain use to "build a perception"?
- What are sensory receptors and how do they operate to "transduce" our physical world into a code understood by the nervous system?
- Describe the general physical features of a typical sensory receptor?
- Create a set of flash cards describing the different sensory receptor types, the stimuli needed to excite them and what modality each belongs to?
- Define the different types of axons available in the nervous system. Why is the size and diameter of an axon an important factor in perception?
- Define, describe and provide examples for the four basic attributes that every stimulus event possesses: Modality, Location, Duration, Intensity
- Define and describe the importance of the "receptive field". Create an example of your own to explain this concept.
- What is the relationship between the receptor potential and the magnitude and spatial features of a stimulus?
- Compare and contrast slowly adapting vs. rapidly adapting receptors. Why are both needed to fully characterize the features of a complex stimulus event?
- Label and define the response features and functions of cutaneous mechanoreceptors, muscle spindles, and GTOs
- What are dermatomes? What diagnostic information can a dermatome provide you?
- Describe and distinguish (1) differences in the pathway organization, (2) the specific anatomy, and (3) function of the DCML, ALS, and Trigeminal Systems.
- Describe the specific sensory inputs, features and organization of the VPL and VPM.
- Describe the organization of and functional relationships among somatosensory structures of the brainstem, thalamus and the primary sensory cortex.
- Describe the organization of the primary somatosensory cortex.
- How does S1 interact with related sensory association areas during behaviors?
- What is neuroplasticity and why is it a vital concept underlying learning and behavior throughout our lifetimes?
- What general operating characteristics of neuroplasticity did we learn about by examining the three primate experiments reviewed in this chapter?
- Explain how principles of neuroplasticity can impact our clinical practice.

References

Allard, T., Clark, S. A., Jenkins, W. M., & Merzenich, M. M. (1991). Reorganization of somatosensory Area 3b representations in adult owl monkey after digital syndactyly. *Journal of Neurophysiology*, *66*(3), 1048–1058.

Amaral, D. G. (2013). The functional organization of perception and movement. In E. R. Kandel, J. H. Schwartz, T. M. Jessell, S. A. Siegelbaum, & A. J. Hudspeth (Eds.), *Principles of neural science* (5th ed., pp. 356–369). New York, NY: McGraw-Hill.

Andreatta, R. D. (2008). Sensorimotor elements of the orofacial system: Reviewing the basics. *Perspectives on Speech Science and Orofacial Disorders*, *18*, 51–61.

Andreatta, R. D., Barlow, S. M., Biswas, A., & Finan, D. S. (1996). Mechanosensory modulation of perioral neuronal groups during active force dynamics. *Journal of Speech and Hearing Research*, *39*, 1006–1017.

Andreatta, R. D., & Etter, N. M. (2018). Sensory systems. In B. Rousseau & R. Branski (Eds.), *Anatomy and physiology of speech*. New York, NY: Thieme Medical.

Aramideh, M., & Ongerboer de Visser, B. W. (2002). Brainstem reflexes: Electrodiagnostic techniques, physiology, normative data, and clinical applications. *Muscle and Nerve*, *26*(1), 14–30.

Baars, B. J., & Gage, N. M. (2010). *Cognition, brain, and consciousness: Introduction to cognitive neuroscience* (2nd ed.). Oxford, UK: Elsevier.

Bandell, M., Macpherson, L. J., & Patapoutian, A. (2007). From chills to chilis: Mechanisms for thermosensation and chemesthesis via thermoTRPs. *Current Opinion in Neurobiology*, *17*(4), 490–497.

Barlow, S. M. (1987). Mechanical frequency detection thresholds in the human face. *Experimental Neurology*, *96*, 253–261.

Barlow, S. M. (1998). Real time modulation of speech—Orofacial motor performance by means of motion sense. *Journal of Communication Disorders*, *31*(6), 511–533.

Basbaum, A. I., & Jessell, T. M. (2013). Pain. In E. R. Kandel, J. H. Schwartz, T. M. Jessell, S. A. Siegelbaum, & A. J. Hudspeth (Eds.), *Principles of neural science* (5th ed., pp. 530–555). New York, NY: McGraw-Hill.

Bell-Krotoski, J., Weinstein, S., & Weinstein, C. (1993). Testing sensibility, including touch-pressure, two-point discrimination,