Clinical Neuroradiology
To my parents, with boundless love and limitless gratitude. I owe you a debt I can never repay.
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Preface

One of my early mentors in radiology told me the following aphorism: “You see what you look for, and you look for what you know.” In the past two decades, the increasing spatial and contrast resolution of modern imaging techniques has vastly expanded the scope of what we can see, and hence, of what we must know. The past dozen years of teaching radiology residents, neurology residents, and neuroradiology fellows has convinced me that this is particularly true in neuroimaging, and applies both to the depth and the scope of our requisite knowledge base. The ability of MRI to show precise neuroanatomical details has made the practices of neurology and neuroradiology more intertwined than ever before. This, in turn, makes it necessary for neuroradiologists to learn more neurology and for neurologists to learn more neuroradiology than practitioners of a generation ago.

This book seeks to fulfill that aim. Focusing on the intersection between these two closely related specialties, it attempts to bridge a gap sometimes found in the many excellent standard textbooks in both fields – an insufficient stress on the overlap between them. To give a rudimentary example, when a neurologist comes down to radiology stating that he has a patient with internuclear ophthalmoplegia (INO), the neuroradiologist needs to recognize the syndrome, understand its underlying neuroanatomic substratum, and look carefully in the brainstem tegmentum for the small lesion involving the medial longitudinal fasciculus which might otherwise be missed. Conversely, when a neuroradiologist proclaims to his neurology colleagues that a parkinsonian patient’s MRI scan shows the stigmata of multiple system atrophy (MSA), they would like to be familiar with the particulars of MRI to recognize those stigmata. Thus, this book attempts to provide imaging correlates for typical cases seen in neurology and clinical correlates for the findings made with neuroimaging.

The book is divided into individual chapters, from the cerebellum through the brainstem, diencephalon, basal ganglia, and cortex. Each chapter provides a discussion of the clinically relevant neuroanatomy of that part of the brain. Following this introductory discussion, structure–function correlations in the CNS are illustrated through consideration of actual clinical cases. The cases are presented in an interactive question–answer “noon conference” format, leading from the clinical history to a presentation of imaging findings and a discussion of the relationship between those findings and the patient’s clinical deficits. This format allows neuroanatomical details to take on an immediate clinical relevance, thus making them easier to remember, and also allows the clinician to appreciate the elegance and specificity of modern neuroimaging. By its very nature – i.e., a case-based approach – this is not meant to be a comprehensive text. However, it attempts to present many of the common entities seen in a hospital-based neurology practice in some detail, and to enhance these presentations with discussions of the relevant neuronal circuitry, pertinent neurochemistry and sometimes the basic therapeutic approaches to particular syndromes of the CNS. Since many of the structure–function correlations we discuss are best displayed with stroke cases, the book ends with a detailed chapter on imaging in stroke and the role of imaging in stroke therapy.

As you read through the book, you will notice that I have tried to keep it light-hearted and informal in style, with sporadic attempts at humor which I hope will be neither feeble nor offensive. If they are either, or both, please accept my apologies in advance.

I need to credit several individuals for their help, and to thank others for their support. For the most part, I will do that in the Acknowledgments section, as is customary. Here, however, I must both thank and credit Dr. Tanya Ferguson for her invaluable help. She not only provided the many excellent neuroanatomical illustrations, which are the crux of the structure–function correlations, but also edited the midbrain chapter and helped edit the neuroanatomy sections throughout the book, keeping me honest with her exquisite knowledge of neuroanatomy. The manuscript is better for her participation.
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References

At the end of many case discussions, I have provided one or several references, which serve both as footnotes for some of the cited facts and as sources for further reading on particular topics. At this juncture, though, I would like to credit several excellent sources which I have heavily relied on throughout the writing of this book. This makes more sense than referencing them again and again at the end of each case. Those sources are:


Fig. 2.16. Schematic diagram of brainstem structures involved in Wallenberg’s syndrome.

Fig. 2.19. Schematic diagram of the structures involved in Dejerine’s syndrome.

Fig. 3.44. Schematic diagram showing the blood supply of the pons.
Fig. 4.10. Vascular distribution of the midbrain.

Fig. 5.23a. Fluorodeoxyglucose PET scans at the level of the striatum our patient.

Fig. 5.23b. Fluorodeoxyglucose PET scans at the level of the striatum with normal control.
Fig. 8.33. Pre- and post-thrombolysis images of a patient with right MCA infarct. The pre-treatment images show a matched DWI–PWI defect. Post-treatment, both the PWI and the DWI abnormalities have resolved. (Image courtesy of Dr. Michael Waters, Director of the Stroke Program, Cedars-Sinai Medical Center, Los Angeles, CA.)

Fig. 8.38c. CT perfusion study in a patient with recurrent TIAs, but without acute infarct. MTT image shows a clearly prolonged MTT in the left MCA territory consistent with hypoperfusion. CT can also provide direct quantitation for specified regions of interest.
Fig. 8.38d. CT perfusion study in a patient with recurrent TIAs, but without acute infarct. Quantitative MTT estimates show an MTT of 2.9 seconds on the right, and a markedly prolonged MTT of 6.5 seconds on the left.

Fig. 8.38e. CT perfusion study in a patient with recurrent TIAs, but without acute infarct. Quantitative CBF mapping shows a normal CBF of 64 ml/100 g per min on the right, and a significant reduction to 29 ml/100 g per min on the left.

Fig. 8.38f. CT perfusion study in a patient with recurrent TIAs, but without acute infarct. The quantitative CBV map shows a value of 2.1 ml/100 g on the right and 2.8 ml/100 g on the left, reflecting the known compensatory increase in CBV in the face of hypoperfusion.