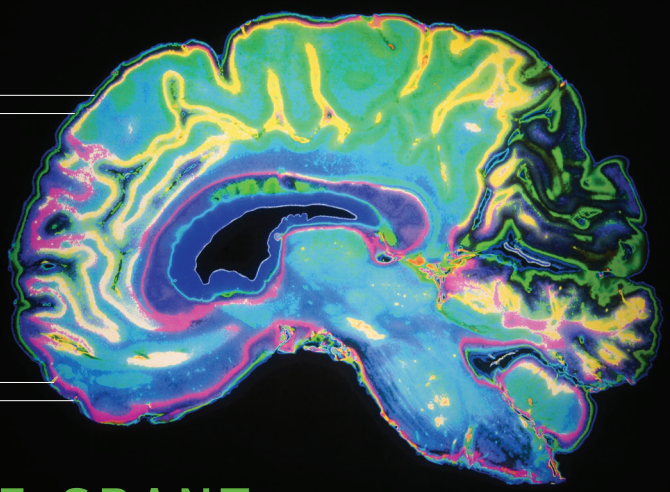
ULTIMATE REVIEW FOR THE NEUROLOGY BOARDS

THIRD EDITION



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Ultimate Review for the Neurology Boards

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Preface

Ultimate Review for the Neurology Boards, Third Edition, continues the tradition of providing a brief but comprehensive source for study or simply review. The authors and editors have tried hard to include the most up-to-date material while keeping the verbiage to a minimum. We have followed a point form outline style where possible, also including tables and lists where long paragraphs would be problematic. A brief Cheat Sheet at the end of most chapters provides a simple quick study section for key facts or potential "board question" information, often those tricky eponyms that we all learn and rapidly forget. We have included some suggested readings for those who want to dive deeper into a review, but have not exhaustively referenced the chapters for the sake of space and clarity. Finally, there are 50 all-new questions with answers and explanations at the end of the book for self-assessment.

The editors hope this text will provide a useful tool to students of neurology at multiple levels, and will help in review for whatever neurological examination looms in the future for the reader.

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Seby John
John A. Morren
Hubert H. Fernandez

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The editors would like to acknowledge the support of Christine Moore, our editorial assistant, who valiantly assisted in organizing authors, managing editors, modifying manuscripts, and generally making the entire contraption function.

Thanks go to our authors, who carefully reviewed and updated the chapters to reflect recent changes in understanding of disease and approaches to treatment within the bounds of the *Ultimate* review format.

We would like to acknowledge the support of the leadership of the Neurological Institute at the Cleveland Clinic for encouraging our authors and editors to contribute to clinical pedagogy.

We would also like to thank our editor, Beth Barry, at Demos for her assistance and support during the editing of this third edition of *Ultimate Review for the Neurology Boards*.

The editors would also like to thank their loving wives (Mary Bruce, Ritika, Divya, and Cecilia) and their wonderful children (Michael, Tucker, George, Sasha, Jordan, and Annella Marie) for their unconditional support and understanding during this process.

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INTRODUCTION

Preparing for Your Boards

I. How to Use This Book

Neurology covers a broad spectrum of disease processes and complex neuroanatomy, neurophysiology, and neuropathology. Moreover, your certification examination will also include psychiatry and other neurologic subspecialties such as neuro-ophthalmology, neuro-otology, and neuroendocrinology, to name a few. Covering all of the possible topics for these boards is not only impossible, it is impractical. Although this book is entitled *Ultimate Review for the Neurology Boards*, it is not intended to be your single source of study material in preparing for your examination. Rather, it presumes that throughout your residency training, or at the very least, several months before your board examination date, you will have already read primary references and textbooks (and, therefore, carry a considerable fund of knowledge) on the specific broad categories of neurology. Because you cannot possibly retain all the information you have assimilated, we offer this book as a convenient way of tying it all together. The point-form information will help you recall specific facts, associations, and clues that may help with answering questions correctly.

Ultimate Review for the Neurology Boards contains detailed chapters on subjects included on the neurology board examination.

For maximal retention within the shortest amount of time, we have used an expanded outline format in this manual.

The main headings and subtopics are in **bold**. A few phrases or a short paragraph is spent on subtopics that we think are of particular importance. Crucial or essential data within the outlines are *italicized* or in **bold**. Thus, we present three levels of learning in each chapter. We suggest that you first read the entire chapter, including the brief sentences on each subtopic. After the first reading, you should go back a second time, focusing only on the headings and subtopics in **bold** and the *italicized* words within the outline. If you need to go back a third time to test yourself, or, alternatively, if you feel you already have a solid fund of knowledge on a certain topic, you can just concentrate on the backbone outline in **bold** to make sure you have, indeed, retained everything.

Whenever appropriate, illustrations are liberally sprinkled throughout the text to tap into your "visual memory." Quick pearls (such as mnemonics to remember long lists and confusing terminology, tables to organize a complex body of information) and high-yield topics are preceded with this symbol "**NB:**" (for *nota bene,* Latin for "note well"), to make sure you do not miss them. We have added a few suggested readings where pertinent to help you extend your learning both for the exams and for your education.

Some chapters overlap. For example, some diseases discussed in the chapter on pediatric neurology and the chapter on neurogenetics can also be found in the individual chapters of the Clinical Neurology section. This overlap is intended to maximize memory retention through repetition.

We have included 50 questions at the end of the book to help you practice for the tests. One of the best preparation methods for taking exams is practicing the exam situation over and over. We hope these questions will give you a chance to try out your hand at answering questions.

Good luck, and we hope you pass your boards in one attempt!

II. Preparing for Your Board Examination

Although most residents initially feel that after a busy residency training it is better to "take a break" and postpone their certification examination, we believe that, in general, it is best to take your examination right after residency, when "active" and "passive" learning are at their peak. There will never be "a perfect time" (or "enough time") to review for your boards. The board examination is a present-day reality that you will need to prepare for whether you are exhausted, in private practice, expecting your first child, renovating your newly purchased 80-year-old house, or burning candles in your research laboratory. You just need to squeeze in the time to study. Luckily, all the others taking these tests are in the same boat, so you are not alone!

Here are a few pointers to help you prepare for the board examination. All or some of them may be applicable to you:

A. Board preparation starts from day 1 of your residency training. Although most residency programs are clinically oriented and have a case-based structure of learning, here are some suggestions as to how you can create an "active" learning process out of your clinical training, rather than just passively learning from your patients and being content with acquiring clinical skills.

1. Imagine you are on your sixth month of a boring ward rotation carrying eight patients on your service. The following table contains the diagnoses of your patients in the neurology ward and the reading initiative we recommend. The point here is to use your patient caseload to suggest topic areas for review. Our experience is that case-based learning "sticks" better than starting on page one of any textbook.

PATIENT	DIAGNOSIS	READING INITIATIVE
1	Thalamic lacunar stroke	Master the anatomy of the thalamus.
2	Embolic stroke	Become familiar with the literature on the use of heparin versus aspirin.
3	Guillain-Barré syndrome	Master the differential diagnosis of axonal versus demyelinating polyneuropathy.
4	Amyotrophic lateral sclerosis	Master the differential diagnosis of motor neuron diseases.
5	25-year-old with stroke, unclear etiology	Master the data on stroke risk factors.
6	Seizure breakthrough for overnight observation	Know all the mechanisms of action of antiepileptic agents.
7	Hemorrhagic stroke	Know and be able to differentiate the MRI picture of a hyperacute, acute, subacute, and chronic bleed.
8	Glioblastoma	Know the pathology of all glial tumors.

2. Always carry a small notebook that fits in your coat pocket so you can write down all the questions and observations that may arise in the course of your day. If possible, do not sleep without answering those questions. Likewise, jot down all the new information you have learned. Read through these notes one more time before you call it a night.
3. Follow your grand rounds schedule. Read the topic(s) beforehand. This will help you in two ways: (a) the talk itself will serve as reinforcement because you already read about it; and (b) you can ask more intelligent questions that will, at the very least, impress your colleagues and mentors, if not make you learn and appreciate neurology even more.

4. For the driven resident: have a monthly schedule of books or book chapters to read. Maximize your reading on your light or elective rotations. On the average, a "good" resident reads 25 to 50 pages per day (from journals, notes, books, etc.). If you read more than 50 pages per day, you are driven and will be rewarded with an almost effortless board review period. If you read less than 10 pages per day, or, even worse, are an occasional reader, you are relying on passive learning and will need to make up a lot of lost time (and knowledge) during your board review.

B. Take your Residency In-Service Training Examination (RITE)/in-service examination seriously. If possible, prepare for it weeks in advance. People who do well every year are the ones who pass their written board examination on the first attempt.

C. Know all board examination requirements several months before you finish your residency training. Know all the deadlines. READ THE INSTRUCTIONS CAREFULLY! Check the name on your identification and the name on your admission slip to make sure they are identical. Contact the American Board of Psychiatry and Neurology (ABPN) if they are not. Ideally, you should be distracted as little as possible when your examination date approaches.

D. Start your formal board review midway (that is, January 2) of your senior year. Make a general, realistic schedule. Do not make it too ambitious or too detailed. Otherwise, you will find yourself frustrated and always catching up to your schedule. As we mentioned, there will never be a perfect time to study for your boards—you need to create your own time. Consider working with a study group, which will provide peer support and pressure to continue studying.

E. In general, start with topics you know the most about (and, therefore, are least likely to forget), such as clinical neurology, and end with topics you know the least about (and, thus, are more likely to forget in a short amount of time), such as neurogenetics, metabolic disorders, neuroanatomy, neurochemistry, and so forth.

F. Use your book allowance wisely. Read and underline books during residency that fit your taste and that you are likely to use for your board review. Underlined books are less overwhelming, provide a sense of security that you have already been through the material (even if you have forgotten its contents), make review time more efficient, and significantly reinforce learning and retention.

G. End your formal review at least 2 weeks before the date of your written boards. Earmark 1 week for the psychiatry portion (do not forget to read on child psychiatry topics) and 1 week for recapping high-yield topics, reviewing questions and answers, looking at radiology and pathology pictures, and reading the answers to past RITE/in-service examinations (they do repeat!).

H. Arrive at your examination site city at least 24 hours before the exam. You do not want to realize on the day of your examination that your hotel reservation was inadvertently misplaced or that your flight was canceled because of a snow storm. Make sure your cell phone is fully charged and that you have your driver's license with you. DO AS MUCH AS YOU CAN BEFOREHAND SO YOU DON'T HAVE TO WORRY ABOUT DETAILS.

I. You might consider bringing ear plugs, an extra sweater, and a reliable watch. When one of us took our boards in the basement of a hospital, there was a general announcement through the public-address system every 30 minutes. We have heard different stories: the heater was not working, a dog convention was going on in the next room, and so forth. **It is best to be prepared.**

J. If this is the second or third time you are taking the boards, consider the benefits of a small study group or having a study partner. You will be amazed that two or three people assigned the same topic to read will emphasize different items. It could very well be that you are underlining the wrong words and need someone to give you a different perspective. At the very least, a study group will keep you on pace with your schedule.

Basic Neurosciences

CHAPTER 1

Neurochemistry/Pharmacology

I. Neurotransmitters (NTs) and Receptors

A. Miscellaneous

1. *Three major categories of NTs*
   - *a.* **Amino acids**
     - i. *Glutamate*
     - ii. *γ-Aminobutyric acid (GABA)*
     - iii. *Aspartic acid*
     - iv. *Glycine*
   - *b.* **Peptides**
     - i. *Vasopressin*
     - ii. *Somatostatin*
     - iii. *Neurotensin*
   - *c.* **Monoamines**
     - i. *Norepinephrine (NE)*
     - ii. *Dopamine (DA)*
     - iii. *Serotonin (5-hydroxytryptamine [5-HT])*
     - iv. *Acetylcholine (ACh)*
2. Monoamine NTs are nearly always (with a few exceptions) inhibitory.
3. *ACh is the major NT in the peripheral nervous system (the only other peripheral NT being NE).*
4. *Major NTs of the brain are glutamate and GABA.*
5. Peptides perform specialized functions in the hypothalamus and other regions.

A. Miscellaneous (*cont'd*)

6. *Peripheral nervous system has only two NTs:*
   a. ACh
   b. NE
7. *Excitatory NTs*
   a. Glutamate
   b. Aspartate
   c. Cystic acid
   d. Homocystic acid
8. *Inhibitory NTs*
   a. GABA
   b. Glycine
   c. Taurine
   d. β-Alanine
9. *Excitatory/inhibitory pairs*
   a. Glutamate (+): GABA (−) in the brain
   b. Aspartate (+): glycine (−) in the ventral spinal cord

B. ACh

1. Miscellaneous
   a. First NT discovered
   b. The major NT in the peripheral nervous system
      i. *Provides direct innervation of skeletal muscles*
      ii. *Provides innervation of smooth muscles of the parasympathetic nervous system*
   c. Major locations of ACh
      i. *Autonomic ganglia*
      ii. *Parasympathetic postganglionic synapses*
      iii. *Neuromuscular junction (NMJ)*
      iv. *Renshaw cells of spinal cord*
   d. Roles of ACh
      i. *Thermal receptors*
      ii. *Chemoreceptors*
      iii. *Taste*
      iv. *Pain perception (possibly)*
   e. Primarily (but not always) an *excitatory* NT
   f. Main effect of ACh on pyramidal cells is via muscarinic receptor-mediated depletion of K^+ currents, which results in hyperexcitability
   g. Most dietary choline comes from phosphatidyl choline found in the membranes of plants and animals.
   h. Phosphatidyl choline is converted to choline, which is then transported across the blood-brain barrier.
   i. Acetylcoenzyme A and choline are independently synthesized in the neuronal cell body and independently transported along the axon to the synapse in which they are conjugated into Ach.
2. Synthesis: *Rate limiting: supply of choline*
3. Release

a. Voltage-gated calcium channel is open as the action potential (AP) reaches the terminal button of the presynaptic neuron, producing an influx of calcium ions that allows exocytosis of presynaptic vesicles containing ACh into the synaptic cleft.

b. The activation of postsynaptic ACh receptors results in an influx of Na^+ into the cell and an efflux of K^+, which depolarizes the postsynaptic neuron, propagating a new AP.

4. Receptors
   a. Muscarinic receptors
      i. *Subtypes*
         (A) *M1, 3, 5: activate phosphatidyl inositide hydroxylase*
         (B) *M2, 4: inhibit adenyl cyclase*
      ii. *Agonists*
         (A) Bethanecol
         (B) Carbachol
         (C) Pilocarpine
         (D) Methacholine
         (E) Muscarine (from *Amanita* mushroom)
      iii. *Antagonists*
         (A) Atropine
         (B) Scopolamine
         (C) Trihexyphenidyl
   b. Nicotinic receptors
      i. *Antagonists (nondepolarizing)*
         (A) Tubocurarine
         (B) Atracurium
         (C) α-Neurotoxin of sea snakes
         (D) Procainamide
         (E) Aminoglycoside antibiotics
      ii. *Antagonists (depolarizing)*
         (A) Succinylcholine
      iii. *Receptor inactivation*
         (A) Myasthenia gravis
      iv. *Receptor deficiency*
         (A) Congenital myasthenia gravis
      v. *ACh release augmentation*
         (A) Black widow spider latrotoxin
      vi. *ACh release blockade*
         (A) Botulism
         (B) Lambert-Eaton syndrome
         (C) Tick paralysis
         (D) β-Neurotoxin of sea snakes
   c. Specific locations of muscarinic and nicotinic receptors
      i. Both nicotinic and muscarinic
         (A) *Central nervous system (CNS) (muscarinic > nicotinic receptor concentrations)*

i. Both nicotinic and muscarinic (*cont'd*)

(B) *All sympathetic and parasympathetic preganglionic synapses*

ii. *Muscarinic only*

(A) *All postganglionic parasympathetic terminals*

(B) *Postganglionic sympathetic sweat glands*

iii. *Nicotinic only*

(A) *NMJ*

(B) *Adrenal medulla*

iv. In brain: muscarinic > nicotinic

5. Vesicle transport

a. SNARE proteins:

i. Mediate docking of synaptic vesicles with the presynaptic membrane

ii. Targets of the bacterial neurotoxins responsible for botulism and tetanus

b. SNAP-25:

i. Synaptosomal-associated protein 25 (SNAP-25) accounts for the membrane fusion (bringing the synaptic vesicle and plasma membranes together).

ii. Botulinum toxins A, C, and E cleave SNAP-25, leading to muscle paralysis as intended in clinically induced botulism.

6. Inactivation

a. Metabolism

i. Within synaptic cleft by acetylcholinesterase

ii. Acetylcholinesterase found at nerve endings is anchored to the plasma membrane through a glycolipid.

7. *Cholinergic agonists*

AGONISTS	SOURCE	MODE OF ACTION
Nicotine	Alkaloid prevalent in the tobacco plant	Activates nicotinic class of ACh receptors, locks the channel open
Muscarine	Alkaloid produced by *Amanita muscaria* mushrooms	Activates muscarinic class of ACh receptors
α-*Latrotoxin*	Protein produced by the black widow spider	Induces massive ACh release, possibly by acting as a Ca^{2+} ionophore

8. *Cholinergic antagonists*

ANTAGONISTS	SOURCE	MODE OF ACTION
Atropine/ scopolamine	Alkaloid produced by the deadly nightshade, *Atropa belladonna*	Blocks ACh actions only at muscarinic receptors
Botulinum toxin	Eight proteins produced by *Clostridium botulinum*	Inhibits the release of ACh
β-*Bungarotoxin*	Protein produced by *Bungarus* genus of snakes	Prevents ACh receptor channel opening
d-Tubocurarine	Active ingredient of curare	Prevents ACh receptor channel opening at motor end plate

9. *Specific agonist/antagonist action*
   a. *Presynaptic NMJ release blockade*
      i. Botulinum toxin: block presynaptic vesicle mobility (see Sections 5.a and b)
      ii. Lambert-Eaton syndrome: block presynaptic Ca^{2+} channels
      iii. Sea snake venom
   b. *Postsynaptic NMJ receptor blockade*
      i. Myasthenia gravis: ACh receptor antibody
      ii. Succinylcholine: depolarizing blockade
      iii. Curare: nondepolarizing blockade
      iv. α-Bungarotoxin: irreversible ACh receptor blockade
10. *Anticholinesterases*
   a. *Reversible*
      i. Neostigmine
      ii. Pyridostigmine
      iii. Physostigmine
      iv. Donepezil, galantamine, rivastigmine, tacrine
   b. *Irreversible*
      i. With irreversible anticholinesterases, receptors can be regenerated with pralidoxime (peripherally) and atropine (centrally).
      ii. Agents
         (A) Organophosphates
         (B) Carbamates
         (C) Nerve gas
11. *Conditions/medications that increase ACh concentration*
   a. Acetylcholinesterase inhibitors
      i. Pyridostigmine
      ii. Physostigmine
      iii. Edrophonium
      iv. Donepezil, galantamine, rivastigmine, tacrine
      v. Organophosphates
      vi. Black widow venom
      vii. β-Bungarotoxin
   b. Enhances of neurotransmission
      i. Pyridostigmine
      ii. 3,4-diaminopyridine

C. Catecholamines

1. Miscellaneous
   a. *Principal catecholamines*
      i. *NE*
      ii. *Epinephrine*
      iii. *DA*
   b. Synthesis
   c. Tyrosine (TYR) transported to catecholamine-secreting neurons in which it is converted into DA, NE, and epinephrine