

BOOK REVIEWS

What's Etiology Got to Do With It?

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Cerebrovascular Disease, Cognitive Impairment and Dementia. John O'Brien, David Ames, Lars Gustafson, Marshal Folstein, and Edmond Chiu (Eds.). 2004. London: Martin Dunitz, 406 pp., \$79.95 (HB).

Reviewed by KERRY DONNELLY, Ph.D., ABPP-CN, VA Western New York Healthcare System, Buffalo, New York.

Cerebrovascular Disease, Cognitive Impairment and Dementia is the second edition of *Cerebrovascular Disease and Dementia* (2000). The new edition is over 160 pages longer than the earlier version, with several new chapters devoted to the pathophysiology of cerebrovascular disease [including provocative chapters on neurotransmitter changes in vascular dementia (VaD), the contributions of homocysteine and low vitamin B to VaD, and a good overview of hereditary forms of VaD], vascular mild cognitive impairment, noncognitive symptoms, and an expanded discussion of prevention and treatment. It is written for both clinical and scientific audiences. As in the first edition, there is a laudable comparative emphasis, with epidemiologic studies of vascular dementia in Europe, North America, Japan, and China. Indeed, the editors hail from the U.K, Australia, Sweden, and the U.S, and this affords the volume a useful global perspective. With five editors and 22 additional contributors, however, this book suffers a bit from the "too many cooks" syndrome. There is a good bit of redundancy. After the fifth or sixth description of classification criteria for vascular dementia, the reader begins to feel on the receiving end of some repetitive rehearsal therapy for her own dementia.

Perhaps most validating for those of us in clinical positions is the scientific confirmation that VaD and Alzheimer's disease (AD) are not necessarily the totally discrete entities described in textbooks. While historically we have been taught to rule out VaD in making a diagnosis of probable AD, evidence from the Nun Study (Snowdon et al., 1997) and the Neuropathology Group of the Medical Research Council Cognitive Function and Ageing Study (2001) showed that the two are often entwined. Holmes et al. (1999) suggested that AD and VaD occur together more commonly than VaD alone. Amyloid angiopathy is commonly associated with both AD and VaD. The apolipoprotein-E genotype is associated with the onset of both atherosclerosis and AD. Hypertension and vascular events are key in developing AD, and other biological markers for AD, such as increased tau protein levels, are also found in VaD. Similarly, where VaD was once character-

ized exclusively as having an abrupt onset with stepwise progression, we now know that such a syndrome is just one form of the disorder. Growing evidence suggests a large percentage of VaD cases are typified by microvascular changes that can lead to an insidious onset and gradual progression, as is typical of AD, further blurring the presentation of the two disorders.

Gustafson and Passant, in their chapter on clinical pathological correlates, describe the confluence of multiple factors in the expression of dementing disorders as follows: "Stroke may not only give rise to circumscribed, delineated neurological and psychological impairments but also global changes of mentation and behavior. These symptoms, however, may not only be the result of the vascular brain lesion but also of other concomitant brain disease and somatic disorders some of which, such as cardiac disease, blood pressure pathology and diabetes, are well known risk factors for cerebrovascular disease. Previous mental and physical health, medication, alcohol consumption, and socioeconomic conditions may also play an important role" (p. 197).

While it is increasingly clear that multiple systems are in play for the expression of all dementias, there is still value in understanding the specific forms of cerebrovascular disorders. The pathophysiological classification of VaD includes large vessel dementia, which can involve multiple lesions or a single, strategic infarct; small vessel dementia, in the subcortex or throughout the brain; hypoperfusive, hypoxic-ischemic dementia; venous infarct dementia; hemorrhagic dementia; and hereditary forms of VaD, such as cerebral autosomal-dominant arteriopathy with subcortical infarcts and leukoencephalopathy (CADASIL syndrome). A preclinical stage of these disorders, akin to mild cognitive impairment (MCI) as a precursor to AD, is now described as vascular cognitive impairment (VCI).

Hachinski and Bowler (1993) first described VCI as being all-encompassing of cognitive decline from the earliest stage through the most profound, late-stage dementia. More recently, the term refers to subtle cognitive changes resulting from cerebrovascular disease that signal a decline but

do not meet full criteria for dementia. Unlike MCI, memory might not be impaired in VCI. Depending on the lesion load and location, attention and/or executive changes might appear in the absence of memory impairment. As in MCI, early intervention in VCI often produces the best outcome.

While the authors are comprehensive in their descriptions of the current state-of-the-art in classification and diagnosis of VaD, focusing on imaging, biological markers, and clinical observation techniques, they also provide six chapters on the management of these disorders. In this final section of the book, as in the beginning, it becomes clear that there are more similarities than differences in the treatment and management of different dementias.

It turns out that the best prevention strategies for VaD are also salient for the prevention of AD. Controlling blood pressure appears to be crucially important in the prevention of both types of dementia. Additionally, controlling blood glucose levels, smoking cessation, cholesterol control, and limiting alcohol are targeted for primary prevention of VaD. Following stroke, secondary prevention measures such as anticoagulation therapy, carotid endarterectomy, and cerebrovascular angioplasty may interrupt further cerebrovascular disease and concomitant cognitive decline.

Until fairly recently, cholinesterase inhibitors were prescribed only for AD. These drugs (e.g., donepezil and galantamine) do not treat the underlying cause of AD. Rather, they treat the cholinergic deficit that causes some of the symptoms of dementia. In his chapter on treatment of cognitive impairment in VaD, Wilkinson indicates that the data now show that cholinesterase inhibitors have a significant impact on cognitive functioning in patients with VaD. The positive effects of NMDA antagonists (e.g., memantine) and neuroprotective agents (e.g., propentofylline) on VaD are less clearly established to date but are also under investigation. While more beneficial drug therapies are likely to emerge in the near future, it seems clear that cholinesterase inhibitors should now be considered in the management of mild to moderate VaD, just as they are now routinely used for AD and dementia with Lewy bodies (DLB).

The final two chapters of the book discuss family and long-term care issues in VaD. These are clearly universal issues that transcend specific dementia etiology. As we all know, once dementing disorders reach late stage, they all look pretty much the same, and the burdens on families and

society are indistinguishable. The authors argue persuasively for keeping patients at home for as long as possible and for employing multiple in-home supportive services to care for the patient and to ease the burden on families.

In conclusion, *Cerebrovascular Disease, Cognitive Impairment and Dementia* is a broad-based review of cerebrovascular disorders that offers new insights into their underlying causes, expressions, diagnosis, and management. It is definitely not written solely for an audience of neuropsychologists. In fact, several of the assessment-oriented chapters seem to gloss over the usefulness of comprehensive neuropsychological testing. Only one chapter specifically focuses on neuropsychological assessment.

Despite such a parochial concern, this book is a useful reference tool, and the section on pathophysiology of cerebrovascular disease is particularly strong. It is a good text for trainees, once they've made it through Lezak and Heilman & Valenstein. It is somewhat ironic that a nearly 400-page book focused on vascular dementia makes such persuasive arguments against the compartmentalization of dementing disorders, but this pan-diagnostic view of dementia is one of the book's primary strengths. This is a valuable addition to my bookshelf, and I'm sure I'll refer to it often in the future. While I believe that the volume would have benefited from more aggressive editing, the redundancy is really more of an annoyance than a problem. Better to have too much information than not enough.

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Imaging Epilepsy

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Magnetic Resonance Imaging in Epilepsy: Neuroimaging Techniques (2nd Edition). Rubin I. Kuzniecky and Graeme D. Jackson (Eds.). 2005. New York: Elsevier. 431 pp., \$149.95 (HB).

Reviewed by BRUCE P. HERMANN, Ph.D., ABPP-CN, *Department of Neurology, University of Wisconsin, Madison. Wisconsin.*

Readers of *JINS* are aware that the epilepsies are common, costly, and complex. Among the more common comorbid-

ities are cognitive and neuropsychiatric disorders. The potential etiologies of these disorders include factors related to

the cause, course, and treatment of the epilepsy, which may exert their effects, at least in part, through alterations in brain structure, metabolism, blood flow, and other dimensions of brain integrity. These effects can be captured and quantified by various neuroimaging techniques and greater familiarity with the diversity of neuroimaging approaches to epilepsy may open a variety of avenues of investigation.

To that end, this second edition of *Magnetic Resonance Imaging in Epilepsy: Neuroimaging Technique*, edited by Drs. Kuzniecky and Jackson, will probably become the standard reference text on neuroimaging in epilepsy. It follows the very well received first edition and captures the surge of interest in neuroimaging and the application of these techniques to epilepsy. The first three chapters are introductory in nature and provide a helpful introduction to the epilepsies, the principles of magnetic resonance (MR) imaging, and a wonderful review of brain anatomy written by Duvvernoy. The text then turns to a series of chapters that review temporal lobe epilepsy with particular attention to the hippocampus (77 pages) and extra-temporal lobe, frontal and occipitoparietal epilepsies (20 pages). The chapters that follow examine epilepsy from an etiological viewpoint (e.g., vascular/ischemic injury, infectious/inflammatory conditions), and with respect to various malformations of cortical development.

Various neuroimaging approaches are systematically reviewed, including advanced structural analytic techniques

as applied to epilepsy (e.g., shape analysis, cortical thickness, voxel based morphometry, and texture analysis) authored by Andrea Bernasconi. Michael Saling addresses the interface between neuroimaging and neuropsychology with a careful analysis of the impact of epilepsy on discrete memory systems and their neuroanatomical correlates in epilepsy, along with pertinent considerations in surgical planning and outcome. Binder and Detre then review the topic of functional magnetic resonance imaging (fMRI) in epilepsy with an expected emphasis on language and memory function.

Remaining chapters examine other techniques including MR neurophysiology [simultaneous electroencephalogram (EEG) and fMRI, Waites and colleagues], MR diffusion and perfusion (Connelly), MR spectroscopy (Hetherington and colleagues), single photon emission computed tomography (SPECT, Rowe), positron emission tomography (PET, Juhasz and colleagues), and magnetoencephalography (Knowlton and Sutherling).

The chapters are well written, authoritative, clinically significant, and the illustrations are generally excellent. The book as a whole presents a wealth of information regarding how these various techniques lead to better insight regarding the impact of epilepsy on brain integrity. The ways in which these techniques may lead to insights into the cognitive and neuropsychiatric complications of epilepsy should be clear. Overall, this edited text will serve as a very useful clinical and research reference text.

Disorder of the Natural Kind?

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Neuropsychology of PTSD: Biological, Cognitive, and Clinical Perspectives. Jennifer J. Vasterling and Chris R. Brewin (Eds.). 2005. New York: The Guilford Press, 337 pp., \$48.00 (HB).

Reviewed by MANFRED F. GREIFFENSTEIN, Ph.D., ABPP-CN, *Psychological Systems, Inc., Royal Oak, Michigan*.

Philosophically, neuropsychologists believe the disorders they evaluate are of a *natural kind*: biologically real and existing independently of our means of classifying them, much like viruses or atomic structures exist separately from cultural outlook (McNally, 2004). We do not like to believe historical influences affect our evaluations, and nobody likes the idea that cognitive disorders can be created by merely marketing their existence. Social and cultural factors cannot be avoided when neuropsychologists move away from well-defined cerebral disorders to the study of subjectively defined disorders. Shorter (1994) elaborated the fluid presentation of hysteria over time, and ill-defined “railway spine” syndromes emerged when railroad accidents became compensable in 1800’s Great Britain but not for orchard workers with similarly abrupt orthopedic strains. For a current controversy, consider how rising autism rates are believed to be biologically determined (e.g., mercury in

vaccines) even though research underscores a proportional decline in mental retardation rates and autism rates still climbed long after mercury preservatives were eliminated in Denmark (Madsen et al., 2003). I refer to diagnostic “bracket creep” (e.g., “defining deviancy down” or “up” in the case of retardation), and “medicalization of misery,” variants of the idea that cultural pressures influence the vocabulary and scope of our inquiries. This can lead to increasing heterogeneity of our diagnostic categories, becoming a potentially insurmountable obstacle for those determined to discover a specific neurobiology for psychological suffering. Posttraumatic stress disorder (PTSD) is a myriad symptom constellation with widening boundaries under increasing neurocognitive scrutiny.

The *Neuropsychology of PTSD: Biological, Cognitive, and Clinical Perspectives*, edited by Jennifer Vasterling and Chris Brewin, is intended to examine neurocognitive corre-

lates of post-traumatic syndromes from diverse perspectives. The title implies PTSD is ahistorical and a disorder of the natural kind; and many of the thirteen chapters within five sections are variations on this theme. Some authors are strong advocates of a pure neurobiological model as if a well-established fact, others propose weaker somatic models that emphasize person–environment interactions, and some recognize that vexing validity problems make specific cause–effect statements inadvisable. Several chapters are highly technical, and the inexperienced reader may need *assists* to understand the biochemical and genetic terminology.

The first section's one chapter (Duke and Vasterling) reviews epidemiological and methodological issues as they bear on neuropsychological functioning. The authors do a masterful job of summarizing PTSD definitions, prevalence, risk and resiliency factors, natural history, and psychiatric comorbidities. They clearly catalog specific methodological and inferential issues that complicate PTSD research and offer concrete advice to correct problems during the design phase (e.g., don't do ANCOVA). Section II, Biological Perspectives, focuses on somatic modeling of PTSD. Chapter two (Southwick, Rasmussen, Barron, and Arnsten) summarizes studies of neurotransmitter and limbic system interactions during cognitive appraisal of threat. The authors advance a testable hypothesis, which I shall term the "frontal-amygdalar imbalance" model. That is, nor-epinephrine pulses suppress inhibitory outflow of the prefrontal cortex, thereby increasing amygdala excitability with attendant irrational fear reactions. This is a strong (albeit technically dense) chapter that ranges from gross anatomy to molecular analysis of stress neurophysiology. Especially interesting were methods for creating transient frontal lobe syndromes in volunteers, such as the tryptophan depletion technique. Chapter 3 (Shin, Rauch, and Pitman), less technically demanding, addresses structural and functional neuroimaging studies including resting and activated SPECT/PET/fMRI studies of the hippocampus, medial prefrontal cortex, and amygdala. These authors force divergent findings into the frontal-amygdalar imbalance model. One intriguing finding is that lower hippocampal volume predates trauma, suggesting it is a risk factor and not an acquired problem. Chapter 4 (Metzger, Gilbertson, and Orr) maintains a disciplined focus on event-related potentials (ERP) and argues ERP is well suited to investigating symptoms such as hypervigilance and reduced concentration. The authors render complex issues into understandable terms, and readily acknowledge lack of clinical specificity when ERP-PTSD findings are viewed in a broader psychiatric context. A problem with this approach is deciding which ERP predictions are valid; some ERP studies find reduced sensory gating (consistent with hypervigilance) but others find attenuation (consistent with avoidance). Hence, any group effects for ERP can be made consistent with PTSD in *post hoc* fashion.

Section III, Cognitive and Information-processing Perspectives, takes a purely cognitive psychology perspective. Premised on the theory that PTSD patients should show

attentional biases under mild threat conditions, author Constans (chapter 5) reviews cognitive psychology paradigms such as: dot probe following threat words; "emotional Stroop" reaction time; subjective biases of various types; and directed forgetting. Constans concludes that findings are contrary to expectations of involuntary perceptual biases. He reports unconscious attentional bias for persons with clinical anxiety and more pedestrian trait anxiety, but *not in various PTSD groups*. Instead PTSD–control differences appear with tasks requiring more deliberative processing, raising an issue of volitional contributions to performance. Brewin (chapter 6) focuses on traumatic memories from a cognitive perspective. Six pages are devoted to memory research from everyday life and PTSD patients, but the remainder is a theoretical exposition on single and multiple representation memory systems. Brewin provides some understandable context by discussing hippocampal and amygdalar roles in memory, but the speculative nature of his discussion is a problem throughout his three chapters.

Section IV, Developmental and Population-specific Perspectives, details PTSD variants during developmental stages or in specific populations. Chapter seven (Bellis, Hooper, and Sapia) impressively catalogues studies of anatomical and biochemical correlates of early trauma exposure. Topics include associations between childhood trauma and brain development, MRI findings, limbic-hypothalamic responsivity, theory of mind, and cognitive function. This is the only chapter that addresses dose-response relations, albeit briefly. Although satisfying and informative, the chapter is vague about the nature and quantification of childhood stressors, casually lumping together the two distinct concepts of "neglect" and "abuse." Vasterling and Brailey (chapter 8) review neuropsychological findings in adults, using the familiar organization by domain: intellectual, attentional, executive, episodic, and implicit memory function. They do an excellent job of integrating this vast literature to propose a testable PTSD pattern: Mild sustained attention and initial memory acquisition problems against a background of intact executive, language, and perceptual-motor functioning, qualified by noting similar neurocognitive patterns in other psychiatric groups. They also report lower intelligence that correlates negatively with PTSD symptom frequency, a crucial consideration when faced with "subtle" deficit issues, common in neuropsychological populations. The contribution of Yehuda et al. in chapter 9 on aging and trauma is not a true chapter; they report a study of list learning in older trauma survivors. Contrary to claims elsewhere in this book, Figures 9.1–9.4 (p. 223) show that *older* Holocaust survivors who had entered death camps at young ages *had markedly better verbal learning scores than American veterans* reporting first traumas in their late 20's. Translation: A reverse dose-response effect. The authors futilely struggle to find a unified neurobiological explanation, but unfortunately there was no symptom validity testing. This chapter required a better description of circadian rhythms, as Yehuda et al. questionably assert

that aging PTSD patients show a “distinctive” attenuation of the daily cortisol cycle. But, dampened circadian rhythms (e.g., core body temperature) are the rule in normal aging. Chapter 10 is relevant to private practitioners: Dual diagnosis of PTSD and TBI. Brewin and coauthor write another overly theoretical piece, this time trying to explain away conceptual problems in dual diagnosis rather than carefully weighing the empirical evidence supporting/disputing it. The authors make the questionable argument that PTSD should be diagnosed in TBI patients not meeting conventional criteria. As hinted in my introduction, neuropsychologists concerned about how diagnostic elasticity affects research need look no further than this chapter.

Section V addresses Clinical Applications. Only Vasterling and Kleiner (chapter 11) offer useable advice. Their history-gathering recommendations are multi-faceted and nicely nuanced, but the plethora of comorbidities they list are daunting and raise an issue of over-reporting. Their neuropsychological test battery recommendations however are generic and more speculative. This is not the authors fault: Outside of expected “mild” relative weaknesses on generic memory and attention tests, there is no empirically demonstrated signature for PTSD on specific tests, and pretrauma *g* remains an issue (cf. chapter 8). Disappointingly, Brewin’s broad speculations on treatment implications (chapter 12) meander around with little direction. A discussion of cognitive-behavioral therapy, a natural fit for this book, is absent, yet space is given for the eccentric EMDR. Brewin does not attempt to map cognitive and biological psychology terms into the vocabulary of psychotherapy, and the esoteric language defeats translation into therapeutic technique, to wit: “From the perspective of dual-representation theory, hotspots may correspond to moments where there was maximal separation between visual-spatial and verbal processing, leading to a large discrepancy between the contents of the respective memory systems” (p. 285). Friedman brings the reader back to earth with pharmacological approaches (chapter 13). This chapter serves as a quick review of adrenergic and pituitary axis chemistry, followed by studies of psychotropic use in PTSD. At times, the list of in-text citations is numbingly long with insufficient detailing of the best study outcomes, but this chapter is useful for those desiring an exhaustive list of primary source material in disorder-specific pharmacology.

The book’s main weakness is lack of an integrative chapter distilling main ideas and conceptual problems. For example, the book did not inform about the best study population. This silence leaves an unsettling implication that traffic crashes minor and major, any head injury, rape, torture, robbery, one-time Sarin attacks, combat of unknown frequency, childhood abuse of any type and duration, and years brutalized in a Nazi death camp are interchangeable as “trauma.” Issues of severity stressor, its quantification, and its independent verification, as well as evidence for dose-response relations, are touched on but mostly neglected. There are necessary steps before considering

any cause–effect reasoning about neurobiological implications in individual cases. A related elephant in the room is the wildly varying PTSD prevalence rates reported in the book. The alleged prevalence of full and subclinical post-Vietnam PTSD is 50% (National Readjustment Study), yet only 15% of all veterans were assigned combat roles (Satel, 2004). It is reasonable to conclude that decidedly nonbiological influences impact symptom reporting and interpretation. Other contradictory findings are juxtaposed without irony or comment. For example, we are treated to the interpretation that PTSD is present in 27% of persons with moderate TBI (Bryant et al., 2000). Even though the Bryant et al. findings just suggest prominent irritability in TBI, the chapter authors jump to a *deus ex machina* conclusion that PTSD-TBI must regularly coexist to justify speculating about it. The symptom overlap between PTSD and other disorders is large and lower hippocampal volumes have been reported in other patient groups. The book omits the animal literature. For example, it is well known that tame animals have smaller amygdalas than their wild counterparts. In this light, a finding of slightly lower amygdala volume in PTSD patients (chapter 3) is of unclear significance. Animal studies might help to cut through comorbidity clutter and more directly address the hypothesis that stress by itself causes specific structural brain changes. My final issue is the questionable practice of relying on global chart diagnoses. There was little focus on core symptoms, and no investigation of “numbing” or exaggerated startle reflex. Given DSM-IV’s combinatory rules for seventeen PTSD symptoms, many patients sharing few symptoms can be diagnosed with PTSD. A consensus about which symptoms are core and which are peripheral is needed before human neurobiology even starts to enter the picture.

I recommend this book. Although in my view this volume provides better evidence for the nonspecificity of PTSD-Cognitive correlates and a need for better effort testing, the stronger chapters (e.g., any by Vasterling) carry the book and provide excellent overviews, informative descriptions of exciting new techniques of great interest to neuropsychologists, and comprehensive reference lists in this well-cited book. Clinicians can improve their differential diagnostic acumen when faced with individual PTSD patients, but treatment-oriented neuropsychologists will find little here.

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Recent and Relevant

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Traumatic Brain Injury in Sports: An International Neuropsychological Perspective. Mark R. Lovell, Ruben J. Echemendia, Jeffrey T. Barth, and Michael W. Collins (Eds.). 2004. Lisse, NL: Swets and Zeitlinger/Taylor and Francis, 510 pp., \$99.00, (HB).

Name your sport! Or, one of the internationally played sports commonly associated with traumatic brain injury that is given coverage within this 26-chapter volume. The book is divided into four sections, each the responsibility of one of the four editors. Section I, Basic Concepts, provides the historical context and reviews diagnosis, management, prevention, biomechanics, pathophysiology, the usefulness of neuroimaging, and genetic aspects of brain injury. Section II, Models of Neuropsychological Assessment, details each of eight highlighted sports. Section III, Methodological Issues, addresses concerns such as measurement of change, post-injury practical interpretation, issues related to testing of professional athletes, and the use of computerized assessment techniques. Section IV, Special Topics, includes important discussions related to gender and cultural issues, ethical issues, and psychotherapeutic aspects of recovery. Also included is advice regarding consultation with sports organizations, and return to play after injury. The editors recruited respected authorities in sports neuropsychology, or related areas, as authors. Together, they contribute to a well-integrated, thorough volume that reviews fundamentals, provides structure and guidance for clinicians, and elucidates the science that has enabled expansion of this area of practice with benefit accruing for players.

Also Arrived:

Technology in Cognitive Rehabilitation. Peter Gregor and Alan Newell (Eds.). 2004. New York: Psychology Press Ltd, 256 pp., \$80.00, (HB).

A selection of papers from the journal *Neuropsychological Rehabilitation* highlighting the use of information technology for cognitive impairment.

The Auditory Cortex: A Synthesis of Human and Animal Research. Reinhard Konig, Peter Hall, Eike Budinger, and Henning Scheich (Eds.). 2005. Mahwah, NJ: Lawrence Erlbaum Associates, 493pp., \$89.95, (HB).

An in-depth consideration of the auditory cortex inspired by presentations at an international conference in 2003 that emphasized auditory cortical fields and their functions, coding of sounds, and plasticity, learning and cognition.

Neurocognitive Disorders in Aging. Daniel Kempler. 2005. Thousand Oaks, CA: Sage, 329 pp., \$49.95, (PB).

Provides coverage of basics related to assessment and treatment of abnormal aging. This book is most appropriate for the student or person seeking an introduction that includes general knowledge along with reviews of specific neurobehavioral aspects associated with aging.