

Simonton, D. K. (1998). Career paths and creative lives: A theoretical perspective on late-life potential. In C. Adams-Price (Ed.), *Creativity and successful aging: Theoretical and empirical approaches* (pp. 3–18). New York: Springer. Summary of the work on creativity across the adult years by the major figure in the field.

U.S. General Accounting Office. (1998). *Alzheimer's disease: Estimates of prevalence in the United States (GAO/HEHS-98-16)*. Washington, DC: Author. A somewhat controversial report on the incidence of Alzheimer's disease in the United States; is free.

Ware, M. E., & Johnson, D. E. (Eds.). (2000). *Handbook of demonstrations and activities in the teaching of psychology* (2nd ed.). Mahwah, NJ: Erlbaum. Prepared by members of Division 2 of the American Psychological Association; a guide to activities that can be used in psychology courses. Includes several chapters on developmental psychology; presenting ideas for active learning in the area of adulthood and aging that can be incorporated into introductory psychology courses.

Whitbourne, S. K. (Ed.). (2000). *Psychopathology in later adulthood*. New York: Wiley. An edited volume containing current information on the epidemiology, theories, and treatment of psychological disorders in later adulthood.

Whitbourne, S. K. (2001). *Adult development and aging: Biopsychosocial perspectives*. New York: Wiley. Current reviews of the literature in the areas of brain and behavior, sensation and perception, cognition, personality, social, and abnormal psychology.

Whitbourne, S. K. (2003). *The aging individual: Physical and psychological perspectives* (2nd. ed.). New York: Springer. Comprehensive review of physiological changes associated with the aging process. Detailed information on health, sensation and perception, and the normal aging of major organ systems.

Until relatively recently, when the topic of underlying biological substrates was discussed in undergraduate and graduate classes of psychology, the course title was either "Physiological Psychology" or "Biological Psychology." With the growth of neuroscience and the application of clinical psychological principles to the assessment and rehabilitation of neurological disorders, neuropsychology has emerged as both the new title for these types of courses and a reconfiguration of the topics discussed. Whereas books such as Kalat's *Biological Psychology* continue to enjoy widespread appeal and acclaim, newer books (e.g., Zillmer & Spiers, 2001) have begun either to replace or change the curricular landscape relative to biological substrates of behavior. As a consequence and together with my own expertise, this chapter focuses on the inclusion of aging issues in neuropsychology.

3

NEUROPSYCHOLOGY: INTRODUCING AGING INTO THE STUDY OF BRAIN AND BEHAVIOR

ANTONIO E. PUENTE

Please direct correspondence to Antonio E. Puente, Department of Psychology, University of North Carolina at Wilmington, Wilmington, NC 28403; e-mail: puente@uncwil.edu.

Rather than simply provide a personal perspective on the topic, the initial starting point for this chapter was to examine the existing literature on how aging was introduced into courses involving biological substrates of behavior. To do this:

1. A review of the Advanced Placement (AP) psychology course, which is based on an introductory course at a university, was completed.
2. A review of the recently accepted guidelines for the teaching of introductory psychology by the American Psychological Association (APA) was done.
3. A review of the syllabi posted on the Web site of APA's Division 2, the Society for the Teaching of Psychology, was completed.
4. A review of the most recently published book on undergraduate teaching activities (Ware & Johnson, 1996) was accomplished.

LIFE SPAN DEVELOPMENT VS. BRAIN AND BEHAVIOR

The information on the AP course reflected the typical content of a 1-year "Introduction to Psychology" course. Although both life span and biological issues were covered, they were done so separately. For all practical purposes, the newly accepted guidelines for the teaching of "Introduction to Psychology" mirror those of the APA. For example, under the section entitled "Life Span Development," the closer the student would come to underlying neural substrates would be in the illustration of physical and cognitive changes (although those changes are not specified). The focus is clearly on social and behavioral changes. In contrast, under the section entitled "Biological Bases of Behavior," the closest a student would come to discussing developmental issues is presumably in the related discussion of evolution. In the Division 2 database of articles published in the *Teaching of Psychology* between 1974 and 1999, 15 articles were published under "aging" and 32 articles under "psychological." However, again the overlap between developmental issues in general and aging issues in particular with physiological ones were not present. Finally, the same could be said of the Ware and Johnson (1996) book *Handbook of Demonstrations and Activities in Teaching of Psychology*.

In all cases, one point was clearly evident. Aging was almost always introduced in life span development courses or sections of courses. In contrast, aging issues were rarely introduced in sections or courses involving brain and behavior. When aging was introduced into life span courses or sections, the

focus was usually on social and emotional issues. More recently, the focus has included or shifted to cognitive issues. However, it is rare for these cognitive issues to be grounded in neural and biological substrates.

The review of information about biologically based courses reflects a paucity of information involving aging. In fact, developmental issues are ignored as a rule. It is as if the organism that is being studied is an adult with no past or future.

As an alternative to this situation, two possibilities exist. In courses involving sections on developmental and biological issues, both could be covered in sequential fashion. Alternatively, an amalgamation of both issues might be in order. In other words, when developmental issues are being covered, they could either include or be grounded in biological or neural substrates. Another possibility would be to flavor an entire course from the other perspective. For example, a neuropsychology course, and not necessarily a developmental neuropsychology course, could be developmentally based.

NEUROPSYCHOLOGY COURSE

Overview

Several assumptions are made in outlining the course. First, the course is not truly a classical developmental course in that it focuses on the adult. Indeed, most neuropsychology courses do not even interface with developmental concepts. When developmental principles have been applied, the assumption is that three distinct and relatively stable developmental periods—child, adult, and aging—exist. Furthermore, although clear developmental changes through the adult cycle are evident, the course assumes that there is relative stability of both psychological and biological phenomena between the 20s and the 50s. Moreover, the most significant developmental changes pertinent to neuropsychologists have traditionally been those that occur in later years. Specifically, the changes during the early and middle adult years are less significant, although still present, than in later years. Thus, developmental perspectives in this and most neuropsychological courses, reflect a comparison between the relatively stable adult and the slowly changing aging years.

The ideas of Benton (Benton & Sivan, 1984) and Costa (1996) outlining the concept of life span neuropsychology and the interface of developmental and neuropsychological perspectives form the foundation for the orientation of a significant portion of such a course. Historically, neuropsychologists were not interested in developmental concepts. Benton and Costa indicated that neuropsychological functioning did not occur in a vacuum and that the organism went through significant developmental changes that

affected and were related to neuropsychological processes. Thus, the course reflects the concept that, although some developmental stability occurs for prolonged periods, especially during the adult years, significant changes are present, especially very early and very late in the life span of the person. Additionally, for reasons that are not well-explained in the scientific and professional literatures, early developmental changes and their interface with neuropsychological performance have been of greater interest to school psychologists. However, later developmental changes, especially those in aging, have been more the purview of clinical neuropsychologists.

Objectives

The main objective is to introduce the student to the relationship between psychological activity and neural substrates. Psychological activity primarily focuses on cognitive issues but also addresses emotional and social concerns, although they are less frequently measured in neuropsychology. Biological substrates are almost exclusively neural in scope, and specifically, brain focused. In addition, emphasis is on human rather than animal studies and models. Also, whenever possible, clinical rather than experimental concerns are raised. Thus, this is not a clinical neuroscience or an experimental neuropsychology course; instead, this course could be titled "(Clinical) Neuropsychology."

Another important objective is for the student to understand that the brain (and for that matter, the person) does not live in a vacuum. Thus, specific emphasis is placed on the neural substrates of behavior within a biopsychosocial context (Puente & McCaffrey, 1992). Of particular importance are such factors as medical status, gender, education, and age. Considering the typical clinical activities of the neuropsychologists and the personal experiences with family members of the students, a focus on aging issues is included.

Topics

The course is divided into two main sections: introductory and specific topics. The introductory section involves an introduction to a variety of topics, including educational and theoretical issues. In this section, attention is focused on models of brain functioning, clinical versus actuarial assessment, and historical and biopsychosocial contexts. Throughout, the dichotomy of adult versus aged is provided. The basic portion of the course involves the interviewing, testing, interpretation, diagnosing, and rehabilitation of brain-injured individuals. In the next section, general concerns about the interface between normal and abnormal aging and neuropsychology are addressed.

GENERAL CONCERNs OF AGING AS APPLIED TO NEUROPSYCHOLOGY

Underlying Neural Substrates

Of utmost importance is the understanding of the biological and physiological changes occurring in the brain of the developing person. Specific focus is placed on understanding the changes between adulthood and aging, both normal and abnormal (Albert & Killiany, 2001). Also, as previously indicated, although these two epochs in a person's life are considered for the purpose of providing generalizations relatively stable, the concept of an evolving neural system is explained. Underlying cellular changes are described, but the primary interest is on macrocellular changes. Thus, attention to specific neural loss and the resulting impact of neurocognitive function would be appropriate. For example, recent evidence (Scheff, Price, & Sparks, 2001) has suggested that synaptic decline occurs in some but not all cortical regions of elderly people's brains. Furthermore, it appears that synaptic decline is seen primarily in individuals with disease process. Thus, when neural substrates are considered, specific attention must be placed on whether one is considering abnormal or normal aging. Attention must also be paid to the concept of neurogenesis across the life span. Related findings (Schaffr, 2000) indicate that neuronal production and differentiation continues to occur, although on a smaller scale in most nonhuman animals, through adulthood and aging. Thus, neurogenesis may not be a preadult phenomenon as previously thought.

Of particular importance is the concept of shrinkage of brain volume (combined with the expansion of ventricle volume) and the changes occurring in normal and diseased cerebrovascular function. Indeed, an error typically noted in clinical diagnostic work is the overdiagnosing of shrinkage of brain volume and the indirect assumption of the existence of neuronal tangles. Specific attention is provided to neurofibrillary tangles and senile plaques and their occurrence in the normal aging process. The loss of neurons in the frontal lobes, probably due to tangles and plaques, may be as much as 20% (Greenwood, 2000). At the same time, the majority of the older population does not exhibit appreciable neuronal atrophy. In contrast, there is a reduction of the importance of blood flow and supply to the brain, especially the cerebral cortex (and primarily the frontal and temporal lobes). Recent evidence has suggested that if plaque build-up occurs in any area of the cardiovascular system, there is strong likelihood of the same occurring within the cerebrovascular structures. Finally, it should be noted that the strong possibility exists that in most aging individuals, a decrease in brain volume (or increase in atrophy) and an increase in transient ischemic attacks (TIAs) resulting from cerebrovascular complications are likely to occur simultaneously.

Hence, underlying neural substrates pose a unique challenge for the integrator in that there seems to be an interaction of neurogenesis and disease (over normal aging), along with developmental changes seen in aging.

Cognitive Aging

The question of how aging occurs neurocognitively is a deceptively difficult one in that people do not age in similar manners. Although the aging process is most often categorized by biological or chronological age, aging could be characterized by a cognitive dimension (Park & Schwartz, 2000). Instead of considering a person as having a chronological age (e.g., 70), an alternative would be to consider the individual as having a cognitive quotient that could be understood as the ratio of cognitive abilities over chronological age. Cognitive abilities would be based on t or z scores or percentiles that would be derived from normative tables obtained from the performance of specific cognitive tasks across a relatively wide band of developmental stages (maybe ranging from 18 to 90). Thus, instead of considering a person as having a biological or chronological age, an individual would be considered to have a cognitive age or quotient.

The question remains as to which cognitive abilities would be measured in arriving at a cognitive quotient (Salthouse, 2001). The two most important cortical functions would be those mediated by the frontal and temporal lobes, which, in turn, are most likely to be affected by the aging process anyway. Specifically, frontal functions would be translated into executive functions, which would include but not be limited to, planning, organization, and problem solving, probably combined with persistence, pace, and follow-through. Temporal functions would be almost exclusively learning and memory. It may be worthwhile to consider related cortical functions, including sensory and motor activities. In general, however, the focus would be to present the idea that instead of viewing a person as having an age, the idea would be to consider the possibility that aging is best understood as cognitive capacity. Therefore, to understand a person's age, one should understand his or her cognitive, not chronological, status.

Thus, if the individual had significantly preserved cognitive abilities relative to his or her biological age, then that person would be considered to be cognitively younger. The opposite would also be true. That is, if a person has lost significant cognitive capacity relative to his or her chronological peers, then that person would be considered to be cognitively older. If a person would be cognitively challenged, relatively speaking, then the possibility is that the frontal lobe is compromised. Greenwood (2000) and Kaszniak and Newman (2000) suggested that of all the lobes of the cerebral cortex, the frontal is most sensitive to the aging process. Thus, age-related or normal aging is most quickly affecting frontal rather than other lobe functioning,

and thus, the deficits typically seen in older adults would mimic those seen in frontal lobe disorders.

In addition to the concept of cognitive ability (over chronological aging), the idea of cognitive reserve should also be considered. Satz (1993) argued that an individual with significant cognitive capacity before the aging or disease process sets in is more immune to either process. The issue of how to establish or increase such capacity is interesting of itself. The role of education as a prophylactic for the aging or dementing process should be considered. Timiras (1995) suggested that education both lengthens life expectancy and reduces disability and disease in old age. Specifically, he suggested that brain reserve capacity is built up during earlier years and affords the increased threshold to damage seen in both healthy and abnormal aging. In Ardila, Rosselli, and Puente's study (1994), norms for brain-damaged and non-brain-damaged older individuals with and without education were presented. Interestingly, two bell curves emerged when educated and non-educated individuals were compared. Indeed, the two curves (i.e., educated and noneducated) overlapped with brain-injured educated individuals, appearing quite similar to the noneducated and non-brain-injured individuals. Thus, it would appear that education might be a method to increase cognitive capacity or reserve, and therefore, the role of education as predicting and protecting cognitive capacity should be underscored.

In addition, recent evidence from neuropsychological and neuroimaging studies (Grady & Craik, 2000) has suggested that not all cognitive abilities are worse in older adults when compared with younger control individuals. Older adults displayed greater brain activation than their younger counterparts in some memory tasks. Grady and Craik also suggested that specific brain changes may be occurring as a coping mechanism for the effects of aging on cognition. The role of habit and overlearning of tasks as a deterrent to the aging process should be considered.

Although most neuropsychological and cognitive tests tend to favor the measurement of left hemisphere or language-based functions, the right hemisphere-mediated activities such as emotion also draw attention. If education increases cognitive capacity, what increases emotional capital? It would be worthwhile to consider the possibility that cognitive aging is but one part of the aging equation. Discussion at the end of the section on cognitive aging should consider the possibility that just as cognitive aging occurs, so does emotional aging.

Normal vs. Abnormal Aging

The assumption students have is that most people age with problems. Recent research (e.g., Reuter-Lorenz, 2000) has suggested that many adults age with neither significant cortical atrophy nor emotional and physical complications. As a consequence, differences between a normal trajectory

for biological aging should be established before understanding the effects of the disease process. Furthermore, aging is sometimes viewed, at least neuro-psychologically, as beginning at a cutoff point, typically age 65. Aging is an evolutionary process, whereas disease is a revolutionary one. In other words, normal aging proceeds in a fairly predictable trajectory from a biological perspective, with changes being relatively gradual and in some cases predictable. When a disease process or an injury (e.g., head injury) is introduced, that trajectory is altered, sometimes permanently, resulting in an unstable and less predictable trajectory. If it is a disease process, such as Alzheimer's disease, the trajectory is redefined but still somewhat predictable. Other disease processes such as TIAs are much less predictable and can, at best, be estimated indirectly by examination of other variables such as blood pressure. If an injury occurs, then the trajectory is haltingly altered with a modified S-curve recovery occurring. The question becomes, however, whether the return to preinjury baseline is ever achieved.

It then becomes imperative to establish some type of premorbid understanding of the aging process. In other words, a biopsychosocial history and context of the individual must be understood. If no premorbid trajectory is understood, the best one would be able to do is to use normative samples of diseased or injured cohorts to establish a statistically average estimate of expected trajectory.

Normal aging could also be defined as healthy aging. Tranel, Benton, and Olson (1997) found that health status of older individuals was predictive of cognitive impairments. Aging alone, without an active disease process, does not reduce cognitive or mental capacities. The dichotomy of normal versus abnormal (or diseased) aging might be insufficient in describing very healthy older adults. Thus, instead of presenting a dichotomy, the idea of a third type of aging, healthy or hyper-healthy aging, could be presented. For these individuals a significant component of their aging process includes important cognitive activities such as travel, reading groups, and other creative endeavors. Research on this type of aging is often summarized in *The Positive Aging Newsletter*, available at <http://healthandage@newsboomerang.com>.

Depression

Estimates suggest that as many as one fourth of individuals referred for neuropsychological evaluation purposes may have direct or indirect symptoms of depression (Holsinger et al., 2002). Although controversial in scope, most neuropsychologists believe that depression affects the measurement of cognitive processes. It would then be prudent to take emotional states into account in the understanding of the aging process.

However, there is a misunderstanding of what could be considered either indirect or direct depression. Depression can be caused by some external or internal situation (e.g., diagnosis of Alzheimer's disease). Whether

the depression is long-standing or biologically based (as is often considered in the case of exogenous depression) is not important. Depression could be a direct result of underlying physiological changes, primarily in the right hemisphere, and could be manifested in two ways. First, symptoms that are measured in some scales (e.g., the Beck Depression Inventory; BDI) as apathy could be confounded by what actually is fatigue or lack of stamina. In other situations, a flattening or lack of appreciation of affective information or stimuli is directly caused by right hemisphere dysfunction. In both cases, the clinical presentation appears to be that of depression, but what is actually occurring are specific behavioral changes secondary to neuronal damage. Also as a rule, neuropsychologists are not in a good position to distinguish the two. The three most common tests used to measure these types of changes are Minnesota Multiphasic Inventory, Rorschach, and BDI (Camara, Nathan, & Puente, 2000), and none provide sufficiently detailed data to make the distinction between clinical depression and "depressive" symptoms secondary to neuronal dysfunction. Nevertheless, increasing evidence has shown that depression may not only complicate neuropsychological ability but may actually even predict it (Carmeli, Swan, LaRue, & Eslinger, 1997). Thus, depression may actually be measured incorrectly in that an increase in certain symptoms (e.g., fatigue) is often secondary to a disease process (e.g., multiple sclerosis) and not necessarily truly reflective of depression itself.

INCLUSION OF AGING IN A NEUROPSYCHOLOGY COURSE

Several different topics related to aging are typically included in a neuropsychology course. If the course is clinically focused, then a brief review of the major disorders is in order. Some neuropsychologists are categorizing dementia as being either cortical or subcortical or according to the type of disorder (e.g., anterograde vs. retrograde amnesia), so another approach might be more efficient. The most prudent approach for a course of this type is to provide an overview of the major dementias or disorders that, by definition, involve premature or rapid aging. In many respects, each disorder represents a unique version of how aging might occur if not affected by a disease or illness. In other words, these disorders represent the types of problems that will inevitably occur in the normal aging process.

Alzheimer's Disease

Impairment in cognitive capacity is becoming an increasingly difficult problem with the aging of the American population (Troster, 1998). Of these, progressive dementia or a rapid aging of the brain is clearly the most problematic and frequent. As described earlier, Alzheimer's dementia is secondary to an increase in neurofibrillary tangles. Additionally, these tangles

are not readily measurable on neuroradiological techniques such as computerized tomography and magnetic resonance imaging. Thus, early detection of Alzheimer's disease is often accomplished by neuropsychological assessment. In fact, there is growing evidence that certain patterns of performance occur in preclinical stages of the disorder. Metamemory appears to be intact in Alzheimer's disease, but a variety of related problems emerge and evolve. Of all the different problems noted in this disease process, the most notable is memory. The most profound difficulty involves the learning of new declarative memory, sometimes referred to as *anterograde amnesia*. For example, individuals have unusual difficulties in learning a short story or a list of 10 words, even when ensuring practice. In contrast, relatively well-stored information such as long term memory tends to remain intact (at least in the initial stages of the disease). Other problems are evident, including word finding and fluency, visual-spatial functioning, and complex problem solving. Less easily measured behaviors such as personality and mood are also affected and in some cases may be the most significant of the behavioral and neurocognitive changes. Finally, the progression of the disease is fairly predictable with faster changes occurring in the later stages.

Cerebrovascular Dementia

What is often diagnosed as Alzheimer's disease is, in reality, probably cerebrovascular dementia. Many of the same problems noted in Alzheimer's disease are seen with this type of dementia. However, there is a prevalence of language and memory problems. A defining characteristic is the progression of the disease and its underlying etiology. In this case, the disease can wax and wane, sometimes even during the course of a day. In some instances, the person appears quite lucid and focused. Additionally, the underlying etiology involves changes in cerebrovasculature rather than anatomic functioning. Peaks, especially extended, of blood pressure or hypertension, for example, are often associated with the development of transient neuropsychological dysfunction. However, the patient does return to a "normal" baseline. Instead, the wanting of the significant dysfunction is followed by a change in the original baseline so that although a static state occurs, the resulting plateau is worse than the original baseline functioning. In other words, a return to a baseline never occurs; instead, a slow deterioration of cognitive abilities is marked with peaks and valleys of cognitive abilities.

Related Disease Processes

In addition to the preceding dementias, other disease processes are sometimes seen in older adults. These include Parkinson's, Huntington's, Pick's, and multiple sclerosis (MS). Discovered by James Parkinson 200 years ago, Parkinson's disease is a result of specific degeneration of the motor fibers

within the brain. The reduction in dopamine neurotransmitters results in initial but subtle changes in fine motor functioning. As time progresses, the disease evolves into a complex array of dementia-type deficits. These deficits include intellectual and executive function (e.g., organization and planning) problems and what is sometimes referred to as an inflexible personality. Also discovered in the last century, Huntington's disease similarly produces unusual changes in motor functioning. Specifically, uncontrolled, spastic, and non-goal-directed motor movements occur. Pick's disease was discovered by a neurologist and psychiatrist who noted that certain patients lost significant cortical cells. This loss resulted in a slow and irreversible reduction in expressive communication.

In contrast to these dementias, MS typically begins in early adulthood, most often in White women, and progresses gradually. Depending on the type of disorders (e.g., relapsing, remitting, or progressive), the symptoms can wax and wane or progress relatively steadily, resulting in early death. MS expresses itself in a variety of ways including motor (fatigue, slowing), sensory (numbness, visual difficulties), and cognitive (memory and executive functions).

CLINICAL NEUROPSYCHOLOGICAL ASSESSMENT

The differentiation between normal and abnormal aging (e.g., resulting from a disease process) almost always involves a clinical neuropsychological evaluation. Furthermore, students often consider the clinical assessment section to be the most interesting and applicable portion of the course. Thus, the most valuable section of the course for the students is the assessment section. At the same time, they are expecting a "cookbook" approach to understanding how assessment is completed. A typical stereotype is that an evaluation consists of matching testing instruments to specific neurological syndromes. In reality, almost nothing of the sort exists. Indeed, almost no neuropsychological test was developed with one specific disorder in mind, and most neuropsychological test items measure a variety of behaviors simultaneously. Hence, it is imperative for the student to come to a realization that the task of an evaluation is most often to have a thorough understanding of the aging and disease process, along with an understanding of the limits of the test instruments themselves.

Instead of thinking of an evaluation of a conglomerate of tests as has historically been the case (e.g., Halstead-Reitan Neuropsychological Battery and Luria-Nebraska Neuropsychological Battery; Reitan & Wolfson, 1993; Golden, Purisch, & Hammeke, 1991), a more prudent approach to the assessment of aging is to consider different categories of behavior to be assessed. Although the major category has traditionally been memory and

the most commonly used test for this has been the different editions of the Wechsler Memory Scale, other categories of behaviors should be measured too. Recently, Duke and Kaszniak (2000) proposed the major problems, which should be considered in understanding dementias as the executive control functions. Executive functions would include volition; planning and attention; purposive action/self-regulation, including productivity; cognitive flexibility; and shifting.

Next is the issue of differential diagnoses. Once the data are in, the clinician is faced with the difficult task of determining what type of disorder or syndrome exists. To assist this differential diagnosis, an understanding of the different disorders and syndromes should be obtained. Rosenstein (1998) published a small monograph outlining a summary of neurocognitive functioning among the dementias. For each of the major dementias, the following categories of problems are considered: memory, attention, visuo-spatial functions, language, executive functions, reasoning, sensory-motor functions, psychiatric symptoms, and demographics. Table 1 of the Rosenstein (1998) monograph is invaluable in this regard.

The presentation of a case study and a comprehensive neuropsychological evaluation would assist in the illustration of the complexity of a clinical neuropsychological evaluation. Toward that end, presentations by a practicing neuropsychologist would enhance the information presented in classroom discussions. In addition, presentation of the preceding materials, followed by a carefully orchestrated visit to an adult day care center, assisted living facility, or nursing home, would prove useful in illustrating the different disorders.

SUMMARY

The study of neuropsychology has been applied for the past 2 to 3 decades to clinical settings and more recently to other ones (e.g., legal and sports). Traditionally, the presentation of clinical neuropsychology has focused on the (static) assessment of brain function and dysfunction. However, over the past decade, increasing emphasis has been placed on understanding the numerous variables that affect neuropsychological measurement. This chapter focuses on the increasing importance of a developmental and biopsychosocial perspective in neuropsychology. Of particular importance is the role of aging in this process. This chapter summarizes such issues as the difference between normal and abnormal aging and the concept of cognitive aging. As the population ages and the importance of neuropsychology in understanding the aging population increases, aging and neuropsychology will interface more in the scientific, clinical, and pedagogical realms.

REFERENCES

- Albert, M. S., & Killiany, R. J. (2001). Age-related cognitive change and brain-behavior relationships. In J. E. Birren, K. W. Schaie, & K. Warner (Eds.), *Handbook of the psychology of aging* (5th ed., pp. 161–185). San Diego, CA: Academic Press.
- Ardila, A., Rosselli, M., & Puente, A. E. (1994). *Neuropsychological evaluation of the Spanish-speaker*. New York: Plenum.
- Benton, A. L., & Sivan, A. B. (1984). Problems and conceptual issues in neuropsychological research in aging and dementia. *Journal of Clinical Neuropsychology*, 6, 57–63.
- Camara, W. J., Nathan, J. S., & Puente, A. E. (2000). Professional psychological test usage. *Professional Psychology*, 31, 141–154.
- Carmeli, D., Swan, G. E., LaRue, A., & Eslinger, P. J. (1997). Correlates of change in cognitive function in survivors from the Western Collaborative Study. *Neuropidemiology*, 16, 285–295.
- Costa, L. (1996). Lifespan neuropsychology. *Clinical Neuropsychologist*, 10, 365–374.
- Duke, L. M., & Kaszniak, A. W. (2000, June). Executive control functions in degenerative dementias: A comparative review. *Neuropsychology Review*, 10, 75–99.
- Golden, C., Purisch, A., & Hammek, T. A. (1991). Luria-Nebraska Neuropsychological Battery. Los Angeles: Western Psychological Services.
- Grady, C. L., & Craik, F. L. (2000). Changes in memory processing with age. *Current Opinions of Neurobiology*, 10, 224–231.
- Greenwood, P. M. (2000). The frontal aging hypothesis evaluated. *Journal of the International Neuropsychological Society*, 6, 705–726.
- Holsinger, T., Steffens, D. C., Phillips, C., Helms, M. J., Havlik, R. J., Breitner, J., et al. (2002). Head injury in early adulthood and lifetime risk of depression. *Archives of General Psychiatry*, 59(1), 17–22.
- Kalat, J. W. (2001). *Biological psychology* (7th ed.). San Francisco: Wadsworth.
- Kaszniak, A. W., & Newman, M. C. (2000). Toward a neuropsychology of cognitive aging. In S. H. Qualls & N. Abelese (Eds.), *Psychology and the aging revolution: How we adapt to longer life* (pp. 43–67). Washington, DC: American Psychological Association.
- Park, D. C., & Schwarz, N. (2000). *Cognitive aging: A primer*. Philadelphia: Psychology Press/Taylor & Francis.
- Puente, A. E., & McCaffrey, R. (1992). *Handbook of neuropsychological assessment: A biopsychosocial perspective*. New York: Plenum.
- Reitan, R. M., & Wolfson, D. (1993). The Halstead-Reitan neuropsychological test battery. Tucson, AZ: Neuropsychology Press.
- Reuter-Lorenz, P. A. (2000). Cognitive neuropsychology of the aging brain. In D. C. Park & N. Schwarz (Eds.), *Cognitive aging: A primer* (pp. 93–114). Philadelphia: Psychology Press.

- Rosenstein, L. D. (1998). Differential diagnosis of the major progressive dementias and depression in middle and late adulthood: A summary of the literature of the early 1990s. *Neuropsychology Review*, 8, 109–168.
- Salthouse, T. A. (2001). Structural models of the relations between age and measures of cognitive functioning. *Intelligence*, 29(2), 93–113.
- Satz, P. (1993). Brain reserve capacity on symptom onset after brain injury: A formulation and review of evidence for threshold theory. *Neuropsychology*, 13, 273–295.
- Scharff, C. (2000). Chasing fate and function of new neurons in adult brains. *Current Opinion of Neurobiology*, 10, 774–783.
- Scheff, S. W., Price, D. A., & Sparks, D. L. (2001). Quantitative assessment of possible age-related change in synaptic numbers in the human frontal cortex. *Neurobiology of Aging*, 22, 355–365.
- Timiras, P. S. (1995). Education, homeostasis, and longevity. *Experimental Gerontology*, 30, 189–198.
- Tranel, D., Benton, A., & Olson, K. (1997). A 10-year longitudinal study of cognitive changes in elderly persons. *Developmental Neuropsychology*, 13, 87–96.
- Trotter, A. (1998). *Memory in neurodegenerative disease: Biological, cognitive, and clinical perspectives*. New York: Cambridge University Press.
- Ware, M., & Johnson, D. E. (1996). *Handbook of demonstrations and activities in teaching of psychology*. Mahwah, NJ: Erlbaum.
- Zillmer, E. A., & Spiers, M. V. (2001). *Principles of neuropsychology*. Belmont, CA: Wadsworth.

4

USING AND CONDUCTING AGING RESEARCH IN EXPERIMENTAL METHODS AND STATISTICS COURSES

RAYMOND J. SHAW

ANNOTATED BIBLIOGRAPHY

- LaRue, A. (1992). *Aging and neuropsychological assessment*. New York: Plenum. Provides one of the first comprehensive (and authored) presentations on the topic of neuropsychological assessment of normal and abnormal aging.
- Nussbaum, P. D. (1997). *Handbook of neuropsychology and aging*. New York: Plenum. A "reference source" for the understanding, assessment, and treatment of older adults.
- Parks, R. W., & Zec, R. F. (1993). *Neuropsychology of Alzheimer's disease and other dementias*. New York: Oxford University Press. Provides a somewhat outdated review of most of the dementias and their pathophysiology and assessment.
- Poon, L. W. (1986). *Handbook of clinical memory assessment*. Washington, DC: American Psychological Association. An important initial publication on many of the topics discussed in this chapter.
- Woodruff, D. S. (1997). *The neuropsychology of aging*. Malden, MA: Blackwell. Analyzes the impact of aging on brain function.

¹The author thanks Diane Apriile for comments on an earlier version of this chapter. This chapter is dedicated to the memory of Sandra K. Beck. Please direct correspondence to Raymond J. Shaw, Department of Psychology, Merrimack College, North Andover, MA 01845; e-mail: raymond.shaw@merrimack.edu.