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Medicine  Health  
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Geriatric Neurology

# What's in a Name???

GOOD - authentic, honest, just, kind, pleasant, skillful, valid

NEIGHBOR - friend, near

ALLIANCE - affiliation, association, marriage, relationship

CORPORATION - company, business establishment

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the Hope Alzheimer's Center. She has been par-  
ticipating at the Center since 2004 and lives in  
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The Hope Alzheimer's Center specializes in caring  
for people with memory loss and helping families  
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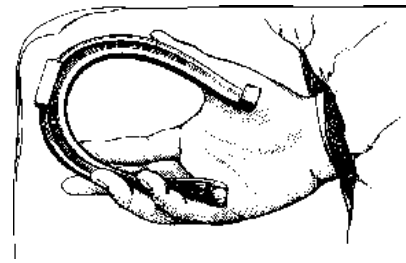
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## Commentaries

### Introduction To Geriatric Neurology Issue

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**Age takes a toll. Mathematicians' best work is behind them** by their late twenties, if not earlier. Chess champions are never elderly. No athletic records are held by the elderly. Yet some people get wiser. The great novels are products of skills honed with time, wisdom and experience, and, clearly a different sort of creativity than required in mathematics and chess. Our brains start to lose neurons in our twenties. As an elderly Houston Merritt, MD, the author of one of the standard American neurology texts, and a giant of twentieth century neurology, once commented, "when it's very quiet, I can sometimes hear the splash a dying neuron makes as it falls into the lacunar lakes in my brain."

In the 1930's the great British neurologist, MacDonald Critchley, published a series of papers pointing out that aging produced, as a normal consequence, many of the features we identify as pathological in Parkinson's disease. Yet these changes are not considered pathological. Perhaps they will be sometime soon. These "normal" changes often produce clinical challenges for physicians trying to distinguish pathological from

normal. Essential tremor in an elderly person, for example, can be indistinguishable from Parkinson's disease, but the prognosis and treatments are quite different. Gait disorders in an elderly person with widespread arthritis, multiple joint replacements and diabetic neuropathy, are often impossible to accurately classify, other than with the highly accurate designation, "multifactorial," which may, or may not be helpful to all concerned.

In the course of my 25 years of practice I have personally witnessed the astounding increase in the number of elderly and very elderly patients I treat. In a recent review I learned that I have cared for 43 patients with Parkinson's disease over the age of 90! Twenty years ago I probably hadn't ever seen a PD patient of that advanced age. As we all know, this is a mixed blessing. In Gulliver's Travels, one of the lands Gulliver visits has a small group of people who are immortal. He thinks this a great miracle but is informed that it is considered a curse because the people are not free of the diseases of aging, thus becoming crippled and demented, and never granted the freedom of death.

In this issue we grapple with the neurology of aging. Excepting pediatricians, we all deal with the elderly, and neurological problems become, like all other problems, increasingly common. Many non-neurologists are neurology-phobic and, because neurology used not to be a treatment oriented discipline, often tend to overlook neurologic problems, or else consider them part of the process of normal aging ("once you're 80 everyone has tremors or falls down once in a while"). In this issue we hopefully are "user friendly," and help you in your day to day care of the elderly.

These articles are summaries of a day long course on geriatric neurology given in Providence on Nov 3, 2007.

— JOSEPH H. FRIEDMAN, MD

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## Some Thoughts On Ethical Guidelines for the Neurologically-impaired Elderly

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**There is a widely accepted Inuit tale [denied as factual by many anthropologists]** that when their frail elders reach a point of senility requiring active nursing care, or when the food supply of their community becomes perilously low, the demented elders are reverently placed upon an ice-flow to drift away into the Eskimo equivalent of oblivion.

Two current social and geophysical realities have altered this aboriginal scenario: first, the intrusion of earnest ethical debate regarding the extent and merit of care for the elderly who are neurologically impaired [such care ranging from the very best technologically to utter abandonment]; and second, with the indisputable acceleration of global warming, there is the diminution in the number and survivability of arctic ice-flows.

There is little debate that a patient who is both elderly and neurologically impaired is effectively marginalized, certainly by contemporary society – and sometimes even by members of the healing community. Many patients with organic disabilities have a way of rehabilitating themselves; less so, though, for those with the loss of neural or cognitive function, thus making the elderly who are paralyzed or the elderly who are demented vulnerable to a more stringent standard of appraisal – outliers, perhaps – in the calculus of care for the elderly American citizen.

In an appraisal of moral norms and moral hopes in the field of geriatric neurology, there are substantially more questions than there are confident answers or even flexible guidelines. Geriatric neurology, particularly as it pertains to the demented elderly, presents an array of incorrigible problems

readily distinguishable from the other sub-disciplines of medicine by the frequent use of such alien words as triage, economic utilitarianism, equity, social justice, palliation, hospice care and the right to die; and the use of such needlessly inflammatory words as euthanasia and “pulling-the-plug.”

An ethical consideration - with any medical dilemma involving the elderly - certainly demands that a few basic questions be confronted:

1. Does the patient's age, per se, play any conscious role in determining the employment [or abandonment] of any therapeutic intervention? Should it? Is there such a thing as age-related rationing of institutional care or medical interventions?
2. Rewording the question, does age-conscious triage become operative in elderly, neurologically impaired patients more so than in orthopedically-impaired elderly?
3. Are stroke victims, beyond the age of 80, viewed differently than dementia victims of similar age? Rewording this question: Does future prognosis play a role in determining present medical intervention? Does the projected lifespan for the patient influence the type and intensity of the therapy?
4. Concerning diagnostic or clinical interventions of doubtful merit, whose vision [or will] prevails? The patient? The patient's assigned surrogate? The family? Clergy? Physician? And when differences arise, whose decision prevails?
5. Are there occasions when palliation replaces active intervention? And what are the thresholds when active intervention gets replaced?
6. When is the patient's plea to die listened to and acted upon?

Before confronting these questions, let us consider a bit of local history as well as certain demographic verities. In 1984, Rhode Island's General Assembly convened a task force assigned with the problem of determining the extent to which patients with enduring dementia imperiled the health care system of the state. Our first task was to define the clinical state of dementia [using standard criteria of duration of dementia, type and extent of memory loss, degree of disorientation, loss of social judgment, insight and the capacity to fulfill the minimal tasks of daily living]; then to survey each of the 107 registered nursing homes in the state to determine how many of their residents could be identified as organically demented; and finally, to extrapolate from these data to estimate the total number of the demented within the state [in hospitals, nursing homes and private homes]. We did not distinguish between dementia of the Alzheimer's type, multi-infarct dementia, Lewy body dementia or other dementias such as the heritable ones as those associated with Huntington's disease.

We concluded, conservatively, that in 1985 there were about 10,000 persons with dementia in a state population of about one million souls [about 1% of Rhode Island.] We presented our data to the State legislature. The first response to our oral presentation was: “Never heard of Alzheimer's disease. What is it? Something new like AIDS?” Our demographic data—and our projections into the 21<sup>st</sup> Century—were treated with a combination of wry amusement and skepticism. And, accordingly, our recommendations were largely ignored—although the State police did take to heart our recommendation that the police be

formally sensitized and educated in distinguishing between a demented elderly wandering the streets and an inebriated person wandering the same thoroughfares.

Years later the report was reviewed and our recommendations [regarding the assignment of state hospital beds for those with advanced dementia] were accepted.

There is little debate now that organic dementia is more than a medical problem; more than a public health problem draining much of this nation's health-care budget; and certain a compelling nightmare for those responsible for planning for this nation's future medical needs.

In 1950, 4% of this nation's GNP was invested in health care. By 1994, this had risen to 14%; it is now verging upon 20% - and rising. A decade ago this government was spending, on average, \$14,000 per year on each elderly American under the Medicare program; it is now approaching \$20,000. And as the nation - particularly its old-old segment - grows in number, and as the number of Americans burdened with dementia of the Alzheimer type increases exponentially, the cost of protecting and caring for these encumbered patients will grow at an alarming rate. Finally these staggering costs will outweigh the humanitarian beliefs in this country and—barring a medical miracle in discovering ways to prevent or cure the organic dementias—the rationing of care will become inevitable.

What may we expect in an elderly person with long-standing dementia? At best a stabilization of the sense of personal identity and cognitive awareness. Visitors, whose visits tend to be brief and cursory, may comment that they experience no communication with their sick relatives, that they are no longer recognized by the demented patients and view them as one step removed from a persistent vegetative state. Nursing aides however, may attest to a measurable degree of communication as well as preservation of some “selfness.” To exploit a common cliché, is the cognitive cerebrum half-full or half-empty?

Despite the notable advances in biotechnology in recent years, and despite some unsubstantiated claims that the progress of Alzheimer's disease may be slowed by certain medications, the professional care of such patients remains largely in the domain of care-takers such as nurses. Physicians continue to play an identifiable role in initiating more aggressive therapies for such complications as decubitus ulcers, opportunistic infections and renal or cardiopulmonic failure. What guidelines may then be established for a disease that is both progressive—often relentlessly so—and without effective therapy?

Certainly, comfort measures, good nursing, pain-control if needed and scrupulous attention to hygienic needs. But beyond these interventions, what then? Specifically, when in the course of this ailment might discussions be begun about a regime confined to palliative care? Daniel Callahan has suggested three standards to observe in such trying situations:

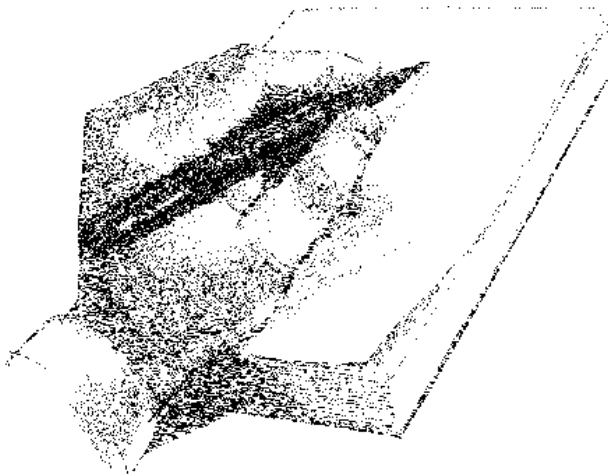
1. No one should, in the modern world, have to live longer in the advanced stages of dementia than he or she would have in a pre-technologic era.
2. The likely deterioration in individuals with advanced dementia should lead to a shift in the usual standard of treatment: that of stopping rather than continuing or extending treatment.

3. For the medical profession, there is as great an obligation to avoid a lingering, painful or degrading death as there is to promote health and life.

Which avoids the crucial question: What clinical features forewarn the attending physician that the time has arrived to warrant cessation of interventions beyond those designed for comfort, cleanliness, adequate hydration and freedom from pain? The single criterion continues to be whether there is any residual sense of conscious selfness in the patient, any awareness—even if only episodic—of himself or herself. Just as family members, visiting briefly, may observe nothing beyond a vegetative existence, so too with the physician visiting for a few moments. The insight into the patient's sense of social identity may come, more commonly, from the nurse or aide who feeds, bathes and interacts regularly with the patient.

The thoughts, wishes and religious observances of the family, the views of the concerned clergy must be listened to, but not heeded blindly. The wishes of the patient remains paramount, whether expressed in an advance directive, in a will or in any reliable document; or, expressed orally during his or her current institutionalization.

• • •



We, as a society, must confront the realities of a near-future in which an avalanche of impaired elderly with dementia will inundate the health care industry and its inpatient institutions, places ranging from nursing homes to tertiary care hospitals.

Our health-care professions are about to be overwhelmed by a tragedy of human aging called dementia. We assemble earnest papers such as this, make honorable declarations, admonish ourselves for alleged insensitivities, atone for our past negligences, pay obeisance to the ethical standards of our vocations, recall oaths to do no wrong, identify the physical needs of the demented, decry the insensitive insurance industry for wanting to set explicit thresholds for triage—but rarely do we listen to the secular voices of the demented. Let me end with a poem by Maya Angelou, a poem that says so much more than any powerpoint presentation might convey:

The print is too small, distressing me.  
Wavering black things on the page.  
Wiggling polliwogs all about.  
I know its my age.  
I'll have to give up reading.

The food is too rich, revolting me.  
I swallow it hot or force it down cold,  
And wait all day as it sits in my throat.  
Tired as I am, I know I've grown old.  
I'll have to give up eating.

My children's concerns are tiring me.  
They stand at my bed and move their lips,  
And I can't hear one single word.  
I'd rather give up listening.

Life is too busy, wearying me.  
Questions and answers and heavy thought.  
I've subtracted and added and multiplied.  
And all of my figuring has come to naught.  
Today I'll give up living.

— STANLEY M. ARONSON, MD

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Stanley M. Aronson, MD, has no financial interests to disclose.

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# Aging of the Human Nervous System: What Do We Know?

Richard W. Besdine, MD, FACP, AGSF, and Difu Wu

## LEARNING OBJECTIVES

Demonstrate the ability to identify and use in clinical care:

1. Nervous system changes with age
2. Differences between pure aging in the NS and the effects of common diseases
3. Age-related changes in key domains:
  - a. Cognition/Memory
  - b. Special senses
  - c. Strength
  - d. Balance
  - e. Somatic sensation

## OVERVIEW: AGING IN 21<sup>ST</sup> CENTURY AMERICA

Americans have gained >25 years of average life expectancy during the 20<sup>th</sup> century, and there is no evidence of slowing in the 21<sup>st</sup>. Although clinical and public health interventions have allowed gains in healthy life expectancy (average age of disability onset) to keep pace with the striking gains in longevity, the sheer numerical increases of older persons portend a burden of disease and disability that will overwhelm the social and financial capacity of our technologically advanced society to manage older persons' health and health care. And Congress dabbles by trying to trim 1-2% from the current Medicare growth rate. The tools to meet these care needs are biomedical science coupled with strategic changes in health care delivery for the very old and vulnerable; neurological problems of aging are a major contributor to the morbidity and healthcare costs for older adults.

## NEUROLOGY OF AGING

There is no greater fear among most Americans than loss of brain function – whether the loss of the very persona from dementia (usually Alzheimer's disease), the multiple other neurodegenerative conditions that are increasingly common with age (e.g., Parkinson's disease, ALS) or the sudden devastation of stroke.

## WHAT IS OBLIGATORY WITH AGING?

As we age, many neurological disorders become common. What are the changes occurring in the nervous system that are inevitable with age in the healthiest adults, even those who exhibit all known risk-reducing behaviors? Using the term **Pure Aging Syndrome** makes clear that no disease, environmental, life style or behavioral risk factor plays a role in the change. These are brain function changes that are inevitable, irreversible with current technology, and while mostly decremental, do not cause symptoms on their own. Although there is much in the literature about decrease in brain size and weight with age, secular trends of increasing size of humans make such conclusions from cross-sectional data hazardous. In addition, only in the past 50 years have large numbers of healthy adults survived into old age. Accordingly, great caution should be taken when concluding that brain shrinkage is due to age alone.

A useful concept is “**homeostenosis**,” the progressive restriction of physiologic reserve capacity in organ systems as a conse-

quence of the pure aging syndrome. The resulting reduced capacity to maintain homeostasis during stress often leads to early and unexpected decompensation under a variety of mild homeostatic perturbations. It is the superimposition of acute illness or drug toxicity upon the pure aging syndrome that results in “homeostenotic” organ crises. There is no better example than the extraordinary vulnerability of elders to delirium when they are stricken with many illnesses or adverse drug effects.

Once the changes of pure aging are understood, the impact, evaluation and management of superimposed disease in older adults can be appreciated. The complexity of these interactions of disease and aging defines the field of geriatrics. Nowhere are these interactions more complex and potentially confusing for the clinician than in the nervous system.

## I. COGNITION

**A. Attention** – There is a mild decline in overall accuracy, beginning in the 60s that progresses slowly, but sustained attention very good in healthy older adults. Older adults are more easily distracted, especially if irrelevant information is presented concurrent with important material.

Clinical point: When giving crucial information to older patients, stick to core data, repeat it and write it down.

## B. Learning and memory

1. Sensory memory is the earliest stage (visual, auditory, tactile); it is inherently unstable and decays rapidly. There is no age-related change.
2. Primary (short-term) memory is the stage after transfer of sensory memory. There is no loss with age.
3. Secondary (long-term) memory persists for hours, days and years. There is a decline with age, mostly in free recall; recognition is well preserved. The universal temporary decline in the ability to retrieve names generally begins early in middle age, and worsens over time. The lost name is almost always retrieved soon after the episode. This phenomenon is not predictive of any neurodegenerative disorder (e.g., Alzheimer's disease).
4. Encoding strategies help retrieval - mnemonics, mental hierarchies, clusters—but they are used less by older persons. Training gives long-lasting improvements.
5. Distraction interferes with learning more in older persons than in young.
6. Clinical point: Give instructions directly and simply, encourage encoding strategies, refer to reputable memory training.

**C. Language** – Vocabulary increases well into the 50s and 60s, and shows no decline with age in those who continue to be engaged in complex language use. Similarly, syntactic skills – the ability to combine words in meaningful sequences – show no decline with pure aging.

**D. Intelligence**, as measured by the Wechsler Adult Intelligence Scale, declines with age, but the biggest, earliest losses were reported in flawed studies. Cohort differences undermined these cross sectional studies, causing selection bias regarding education, gender, race, occupation and income. Summary scores of “intelligence” per se are less useful in adults. Measuring specific intellectual functions has become usual.

1. Crystallized intelligence (learning and experience) remains stable or improves with age until the late 70s or beyond, especially in those who remain healthy and engaged in cognitively demanding activities.
2. Fluid intelligence (problem-solving with novel material requiring complex relations) declines rapidly after adolescence.
3. Perceptual motor skills (timed tasks) decline with age.

### Special Senses

#### E. Vision

1. Pure aging includes decline in accommodation (presbyopia), low-contrast acuity, glare tolerance, adaptation, color discrimination and attentional visual field. Changes occur in components of the eye itself and in central processing. These numerous changes affect reading, balance and driving, but compensatory glasses and behavior can maintain safety.
2. The common eye diseases in old age (glaucoma, macular degeneration, cataracts, diabetic retinopathy) are superimposed upon these pure aging changes.

#### F. Hearing

1. Conductive and sensory hearing losses (presbycusis) occur with age; losses are primarily high tones, making consonants in speech difficult to discriminate.
2. Although impairment is defined as an auditory threshold greater than 25 decibels, the nearly half of Americans > 80 who don't reach the clinical threshold of 25 decibels still have diminution in acuity – pure aging effects.
3. Consequences include difficulty in localizing sound and understanding speech, usually accompanied by hypersensitivity to loudness.
4. Common diseases in old age are superimposed upon these changes, often resulting in worsening hearing impairment (e.g., cerumen impaction, otosclerosis, 8<sup>th</sup> N drug toxicity).

**G. Taste buds don't diminish**, but salt detection declines; perception of sweet is unchanged, and bitter is exaggerated. The volume and quality of saliva diminish. All changes combine to make eating less interesting. These aging changes are compounded by common diseases (periodontal) and medications.

**H. Smell acuity declines with aging.** There is atrophy of olfactory bulb neurons, and central processing is altered. The result is decreased perception and less interest in food. Again, these age-related changes are compounded by disease (e.g., AD and PD have diminution and alteration of smell).

## II. STRENGTH

**A. Muscle** – Disuse and disease, as in many systems, are major confounders of age effect.

1. Age-related changes include loss of muscle mass, though strength loss can be relatively preserved by exercise. Reduction in muscle fiber size occurs primarily in Type II ('fast') fibers, which are highly anaerobic; Type I ('slow') fibers, which are aerobic, tend to retain their size during aging.
2. Muscle wasting in frail older persons, a disorder known as sarcopenia, leads to higher incidence of falls and fractures and functional decline.

**B. Spinal reflex changes** include decline in amplitude of the spinal stretch reflex with normal aging, due in part to stiffness of tendons.

**D. Motor cortex changes** with age include decrease in the number of neurons and synapses; one hypothesis is that disuse atrophy occurs, arguing for a “use it or lose it” construct.

**E. Basal Ganglia** – Age-related changes in the striatum include decline in dopamine D1 receptor density in the caudate and putamen. Age-related loss of dopamine neurotransmission may play a role in vulnerability of older adults to extrapyramidal disorders. In the substantia nigra, pigmented neurons drop out; their loss is associated with motor dysfunction, including bradykinesia, stooped posture and gait disturbance. These aging changes mimic parkinsonian features, and may account for the increase in prevalence of PD with age.

## III. BALANCE

Balance function declines with increasing age, but is rarely the sole cause of falls in older persons. Strength, cerebellar integrity, vestibulo-cochlear function, hearing and vision all play a role in maintaining balance. Degeneration of the otoconia (granules of the otolith) is a mechanism for vestibulo-cochlear decline with aging. Many diseases affect the vestibular portion of the 8th N, and it is also sensitive to drugs. Finally, proprioception also contributes to maintenance of balance. Muscle spindle and mechanoreceptor functions decline with pure aging, further interfering with balance. Clinical position sense does not decline with age.

## IV. SENSATION

**A. Pain** – Typically painful disorders are often less or not at all painful in elders.

1. Some cortical processing capacity for pain sensation appears to decline with age. When functional **magnetic resonance imaging (fMRI)** was used to compare cortical nociceptive responses to painful contact heat in healthy young and older subjects, older subjects had significantly smaller pain-related fMRI responses in **anterior insula (aINS)**, primary **somatosensory cortex (S1)**, and supplementary motor area. Gray matter volumes in S1 and aINS were significantly smaller in the elders, suggesting reduced processing capacity in these regions, perhaps accounting for smaller pain-related fMRI responses.



**The Mini-Cog assessment instrument** combines an uncued 3-item recall test with a clock-drawing test (CDT) that serves as a recall distractor. The Mini-Cog can be administered in about 3 min, requires no special equipment, and is relatively uninfluenced by level of education or language differences.

**ADMINISTRATION – The test is administered as follows:**

1. Make sure you have the patient's attention. Instruct the patient to listen carefully to and remember 3 unrelated words and then to repeat the words back to you (to be sure the patient heard them).
2. Instruct the patient to draw the face of a clock, either on a blank sheet of paper, or on a sheet with the clock circle already drawn on the page. After the patient puts the numbers on the clock face, ask him or her to draw the hands of the clock to read a specific time (11:10 and 8:20 are most commonly used and more sensitive than some others). These instructions can be repeated, but no additional instructions should be given. If the patient cannot complete the CDT in =3 min, move on to the next step.
3. Ask the patient to repeat the 3 previously presented words.

**SCORING – Give 1 point for each recalled word after the CDT distractor. Score 0–3 for recall.**

Give 2 points for a normal CDT, and 0 points for an abnormal CDT. The CDT is considered normal if all numbers are depicted, once each, in the correct sequence and position, and the hands readably display the requested time. Add the recall and CDT scores together to get the Mini-Cog Score:

- 0–2 indicates positive screen for dementia.
- 3–5 indicates negative screen for dementia.

2. Endogenous pain inhibition is reduced with aging. Opioid-mediated endogenous analgesic systems are particularly susceptible to functional decline with aging. Additionally, older age is associated with reduced beta-endorphin levels.
3. Some pain thresholds show age-related changes. Although sensitivity to heat pain is decreased with age (above), sensitivity to pressure pain is enhanced.
4. Both Substance P and calcitonin gene-related peptide (CGRP), major neuro-transmitters of primary afferent nociceptive fibers, are decreased with aging, likely reflecting reduction in density or function of nociceptive nerves. The rate of CGRP axonal transport also decreases with advancing age.

**B. Sensory nerves lose myelin selectively,** perhaps predisposing to neuropathy. Vibration sensation perception diminishes with aging, especially in the legs. Since position sense is carried in the same tracts as vibration, diminished vibratory sense should be followed up by position sense testing.

**REFERENCES**

1. Beers MH (Ed). *The Merck Manual of Geriatrics*. 3th Ed. NJ: Merck & Co., Inc, 2006.
2. Boeve B, McCormick J, et al. *Neurol* 2003;60:477-80.
3. Borson S, Scanlan J, et al. *Int J Geriatr Psychiatry* 2000;15:1021-7
4. Burke SN, Barnes CA.. *Nat Rev Neurosci* 2006;7:30-40
5. Craft S, Cholerton B, Reger M. Aging and Cognition: What is Normal? In: Hazzard WR, Blass JP, et al, eds., *Principles of Geriatric Medicine & Gerontology*, 5th Ed. NY: McGraw-Hill, 2003:1370-1.
6. Galban CJ, Maderwald S, et al. *J Gerontol A Biol Sci Med Sci* 2007;62:453-8.
7. Gibson SJ, Farrell M. *Clin J Pain* 2004 ;20:227-39.
8. Keuker JI, Luiten PG, Fuchs E. *Neurobiol Aging* 2003;24:157-65.
9. Kirkwood TB. *Cell* 2005;120:437-47.
10. Lindeboom J, Weinstein H. *Eur J Pharmacol*. 2004;490:83-6.
11. Mattay VS, Fera F, et al. *Neurol* 2002;58:630-5.
12. Mrak RE, Griffin ST, Graham DI. *J Neuropathol Exp Neurol* 1997;56:1269-75.
13. Nanda A, Besdine RW. Dizziness. In: Halter JB et al, editors. *Principles of Geriatric Medicine and Gerontology*. 6th Ed. New York. McGraw-Hill; 2003.
14. Nusbaum AO, Tang CY, et al.. *AJNR Am J Neuroradiol* 2001;22:136-42.
15. Peters R. *Postgrad Med J* 2006;82:84-8.
16. Quiton RL, Roys SR, et al. *Ann NY Acad Sci* 2007;1097:175-8.
17. Raz N. The ageing brain: structural changes and their implications for cognitive ageing. In *New Frontiers in Cognitive Ageing*, Dixon R, Backman L, Nilsson L (eds). Oxford University Press: Oxford, 2004; 115-34.

18. Resnick SM, Pham DL, et al. *J Neurosci* 2003;23:3295-301.
19. Rodriguez-Aranda C. *Clin Neuropsychol* 2003;17:203-15.
20. Shaffer SW, Harrison AL. *Phys Ther* 2007;87:193-207. Epub 2007 Jan 23. Review.
21. Smith CD, Chebrolu H, et al. *Neurobiol Aging* 2007;28:1075-87. Epub 2006 Jun 13.
22. Smith DE, Rapp PR, et al. *J Neurosci* 2004;24:4373-41.
23. Tumeh PC, Alavi A, et al.. *Semin Nucl Med* 2007;37:69-87. Review.
24. Ward NS. *Ageing Res Rev* 2006;5:239-54. Epub 2006 Aug 14. Review.

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# Mild Cognitive Impairment, Healthy Aging and Alzheimer's Disease

Chuang-Kuo Wu, MD, PhD

**As predicted in 1976, the growth of the elderly population has resulted in a tremendous increase in Alzheimer's disease (AD).<sup>1</sup> In 2007, there were probably more than 5 million Americans suffering from AD.<sup>2</sup> The major advances include more clinical tools to diagnose AD and several medications approved by the FDA for treatment.**

Our goals are:

- Recognizing healthy, successful aging of cognition and developing strategies for primary prevention
- Developing new disease modifying agents to alter the course of AD.
- Defining mild cognitive impairment (MCI) as a diagnostic entity for early intervention.

## HEALTHY AND SUCCESSFUL AGING

Healthy aging is defined by the lack of a significant decline in physical and mental abilities.<sup>3</sup> These people are socially active and emotionally satisfied. One definition of "successful aging" is that the elderly who perform in the upper end of a distribution of test scores are deemed successful.<sup>4</sup> The effect of aging on cognition is a hot topic. People older than 65 continue to change through the rest of their lives in different ways. A wide variety of age-related phenomena have been described.<sup>5</sup> Many studies have documented various life styles leading to healthy or successful aging.<sup>3,4,5</sup> Learning from these studies, preventive strategies are postulated to delay the onset of dementia. Among them, education and participation in certain leisure activities are two areas which have demonstrated benefit.<sup>6,7</sup>

From epidemiological data, Katzman pointed out that elder people with poor or no education have an increased risk of developing dementia<sup>6</sup> compared to those better educated. It is postulated that education might generate brain "reserve", which can compensate the initial presen-

tation of dementia.<sup>7</sup> In a five-year, prospective, longitudinal study of healthy elderly adults (above 75 years of age), Verghese et al. reported that the elderly (n=345; age 78.9) who participated in three kinds of cognitive activities and one physical activity had a reduced risk of dementia.<sup>8</sup> Reading, playing board games and playing musical instruments are deemed beneficial cognitive activities; dancing is the only physical activity identified that is associated with a lower risk of dementia. They also observed that subjects (n=124; age 79.7) who eventually developed dementia (the majority of them had AD) were older individuals with lower levels of education (less than 12 grades) and relatively lower test scores (on two memory tests) at baseline. This landmark study provides evidence of risk-reducing activities to delay the onset of AD.

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**Currently the FDA approves no treatment for MCI because all the data indicate a clear lack of benefit.**

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## MILD COGNITIVE IMPAIRMENT (MCI)

The term, **mild cognitive impairment (MCI)**, was initially used as stage 3 of Reisberg's **Global Deterioration Scale (GDS)** in the staging of AD.<sup>9</sup> Petersen, et al then proposed MCI as a diagnostic entity for the transition between normal aging and AD.<sup>10</sup> In 2001, the American Academy of Neurology published its guideline for the diagnosis of MCI.<sup>11</sup> The criteria include 1) memory complaint 2) objective memory impairment 3) normal general cognitive function 4) intact ADLs 5) not demented by DSM IV criteria. The clinical tools recommended according to the guideline are the **Mini-Mental State Examination (MMSE)**, the **Clinical Dementia Rating (CDR)** scale

and other neuropsychological batteries, to be used as screening tests.

Clinicians encounter several pitfalls when diagnosing MCI. It is caused by a variety of pathologies other than just AD. To predict that a MCI patient is in the prodromal stage of AD, we have to consider other differential diagnoses. In the elderly, the two most common conditions which can mimic MCI are depression and stroke. Patients with late-life depression commonly complain of memory and cognitive problems. By sophisticated neuropsychological testing, their cognitive functions are usually within normal limits and subjective memory problem can be restored by adequate treatment. Older adults who had a sudden onset of cognitive impairment often had a cerebrovascular event. Recent studies have reported that patients with the amnesic-type of MCI often do have underlying AD pathology. For example, Morris has proposed that since so many amnesic MCI patients have AD pathology the diagnostic criteria need to be revised.<sup>12</sup> In contrast, the Mayo clinic reported that 71% of amnesic MCI brains displayed the AD pathology but that 29% of them showed non-AD pathologies, which particularly affected the mesial temporal regions.<sup>13</sup>

Neurochemical studies of MCI brains have demonstrated upregulation of the synthetic cholinergic enzyme, suggesting no decline of acetylcholine in MCI brains. This fits with the observation that all published clinical trials to date of cholinesterase inhibitors show no significant efficacy.<sup>14</sup> Currently the FDA approves no treatment for MCI because all the data indicate a clear lack of benefit.

## ALZHEIMER'S DISEASE

In the near future, we will witness major advances in the treatment of AD, such as the introduction of disease-modifying agents. None are yet available, however. The first crucial step in treating AD patients is to classify the clinical stage. (Figure 1). Today only symptomatic treatments are available for AD, including **cholinesterase inhibitors (CEIs)** and a **NMDA receptor**

# Flow Chart of AD treatment

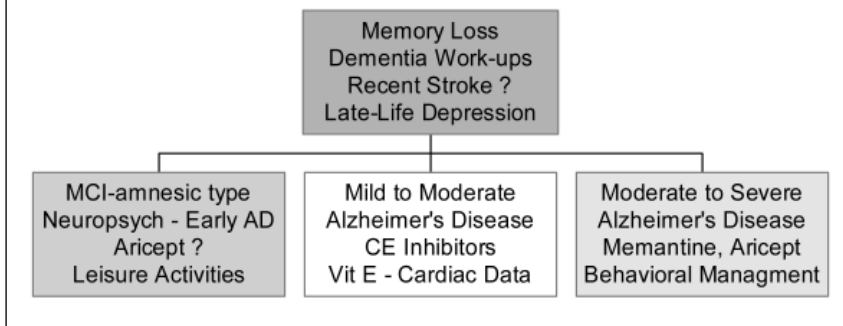


Figure 1.

antagonist. Since 1992, the FDA has approved five medications to treat Alzheimer's disease. We no longer prescribe tacrine because of significant side effects. Donepezil, rivastigmine and galantamine are approved for treating mild to moderate stage of AD. In 2004, memantine was approved for the moderate to severe stage of AD. In 2007, donepezil won approval for the severe stage of AD; rivastigmine has a skin patch approved to treat the mild to moderate AD.

In the clinic, patients can be evaluated simply by the MMSE as the first assessment tool. Many clinical scales have been developed to replace the MMSE. However, the MMSE yields a significant amount of data for interpretation. The first step in establishing the diagnosis of AD is to rule out other mimicking conditions, both treatable and untreatable. For example, a typical, high school-educated, 70 year-old patient comes in with a caregiver complaining of a memory problem, and scores 24/30. Based on the education level and age, the score suggests a high probability of dementia. Using a set of laboratory tests and a brain scan (CT or MRI), a clinician can confidently rule out other causes and establish AD as the cause of dementia (two-step approach). Scores of 12 to 23 are usually considered the mild to moderate stage of AD, as defined in the cholinesterase inhibitor clinical trials. A score below 15 indicates the moderate to severe range. However, there is no consensus about the defined score for the moderate stage of AD. The *Alzheimer's Disease Cooperative Study* (ADCS) group reported that the ability to perform a wide range of instrumental and basic ADLs correlates well with the MMSE scores of AD patients. A significant drop on the MMSE to below 20 correlates

with a significant decline in ADLs from the mild to moderate stage; likewise, a score below 15 indicates a further decline toward the moderate to severe stage.

Treating the cognitive problems of AD patients is limited to those approved by the FDA. When AD patients receive treatment, we should monitor progress of disease with the MMSE. A 4-point decline within a short period (6 months) deserves repeated clinical evaluation for other medical conditions which can worsen cognition. The combination of CEIs and memantine is recommended for treating the moderate to severe AD patient if side effects are tolerable. Memantine is not approved for mild AD or MCI diagnosis.

## REFERENCES

1. Katzman R. *Arch Neurol* 1976; 33:217-8.
2. Hebert LE, Scherr PA, et al. *Arch Neurol* 2003;60:1119-22.
3. Reichstadt J, Depp CA, et al. *Am J Geriatr Psychiatry* 2007;15:194-201.
4. Fritsch T, McKee MJ, et al. *Gerontologist* 2007;47:307-22.

5. Baltes PB, Baltes MM. Psychological perspectives on successful aging. In: Baltes PB, Baltes MM. Editors. *Successful aging*. Cambridge University Press; 1990:1-34.
6. Katzman R. *Neurol* 1993;43:13-20.
7. Zhang M, Katzman R, Dalmon D *Ann Neurol* 1990;27:428-37.
8. Verghese J, Lipton RB, et al. *NEJM* 2003;348:2508-16.
9. Flicker C, Ferris SH, Reisberg B. *Neurol* 1991;41:1006-9.
10. Petersen RG, Smith GE, et al. *Arch Neurol* 1999; 56:303-8.
11. Petersen RG, Stevens JC, et al. *Neurol* 2001; 56:1133-42.
12. Morris JC. *Arch Neurol* 2006; 63:15-6.
13. Jicha GA, Parisi JE, et al. *Arch Neurol* 2006; 63:674-81.
14. DeKosky ST, Ikonomovic MD, et al. *Ann Neurol* 2002; 51:145-55.
15. Jelic V, Kivipelto M, Winblad B. *JNNP* 2006; 77:429-38.

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# Gait In the Elderly

Joseph H. Friedman, MD

A number of impressive statistics on falls in the elderly are routinely cited in review articles.<sup>1,2</sup> Interestingly, none of them are recent.<sup>1,2</sup> My guess is that as the population ages, these numbers will worsen. In addition, abandonment of the “chemical straitjacket” routinely used for the demented twenty years ago which rendered poor walkers into non-walkers may have increased fall frequency.

For example, data from 1988, still cited in recent articles,<sup>1,2</sup> state that one third of people over age 65 fall each year, and for half of them this is an ongoing problem.<sup>3,4</sup> Ten percent of these falls results in significant morbidity (data from early 1990s).<sup>5</sup> Presumably these numbers increase with age. Falls are the fifth most common cause of death in the elderly. Only 25% of patients who suffer hip fractures regain their former level of function. And falls frequently induce a fear of falling, which itself contributes to the problem.

Impairment in mobility affects 14% of those between 65 and 74 but half of those over 85.<sup>6</sup> This impairment occurs for a number of reasons, including brain changes, both normal and pathological, as well as changes in muscles and the sensory organs (eyes, vestibular apparatus, peripheral nerves). Normal aging produces physical changes that overlap with the signs of Parkinson's disease, except for tremor.<sup>7</sup>

The clinical importance of gait cannot be overestimated. In a nursing home evaluation, every single patient seen by this author had a significant gait abnormality. Of course, in many cases, that was why they were not living at home.

It is my hypothesis that gait abnormalities are often not identified by doctors because it has not been part of their training. In the outpatient setting, doctors, for efficiency, tend to see patients in examining rooms, partially undressed, seated on the examining table. In the hospital, it is difficult to get patients out of bed or off their stretchers, and a lot more difficult to get them back on them, especially in the emergency department where the stretchers are quite high.

The basic problem is the lack of a language for describing and classifying gait problems.

Gait disorders may occur for a number of reasons, and most are non-neurological in origin. Joint pain, muscle weakness, deformities, blindness, vestibular dysfunction, psychological factors, poorly fitting shoes (especially in those with edema and bunions) and deconditioning all may play a role. Pain, shoes, feet and blindness should be asked about.

Weakness and numbness may be peripheral nervous system contributions to gait disorders or may result from central nervous disorders. Increased tone, ataxia, weakness and abnormal “motor programs” are central nervous system abnormalities.

## COMPONENTS OF GAIT

### 1. Ability to stand

The patient should be asked to stand without using her arms. They may be kept folded across the chest or put into a praying position. If the patient cannot do this, she should try to stand pushing off from the armrests on her chair. A note should be made of how easily the patient was able to do this or whether the doctor needed to help.

### 2. Posture

Patients with an abnormal posture will generally have kyphosis, scoliosis or some degree of both. Rare patients will have a hyperlordotic posture from lower spinal muscle weakness or dystonia. Kyphosis is usually idiopathic, but may be due to compression fractures, Parkinson's disease, spondylitis. The curvature may occur anywhere from the lumbar spine up. In PD, for example, patients may have highly variable degrees of flexion in the thorax or the neck or both.

### 3. Base

The normal width of foot placement is considered the width of the shoulders. The base should be noted both when standing and when walking. Ataxic gaits, due to sensory problems, cerebellar or vestibular dysfunction will cause the base to increase or to vary. Spastic problems, due to corticospinal tract dysfunction, as seen in spastic forms of cerebral palsy, stroke or cervical myelopathies, cause a narrowing

of the base, with scissoring, the crossing of feet, being an extreme example, most often seen in people who were born with a form of cerebral palsy.

### 4. Stride and foot strike

The stride length depends on the speed of gait as well as the height of the subject. Excessively long strides are rare, and produce a “loping” quality, whereas short steps are common, especially in the elderly, seen in parkinsonian disorders as well as “fear of falling” in which people walk as if they are on ice. In asymmetric disorders, such as Parkinson's disease, one stride length may be shorter than the other, producing a limping gait.

In normal gait the heel strikes the ground first, but in Parkinsonian disorders there is a flat foot strike. In spastic disorders the ball of the foot hits first. These different foot strike patterns may be reflected in wear on the shoes.

### 5. Armswing

This is not important in the biophysics of walking but is extremely important in making diagnoses. The armswing is reduced in Parkinsonism, for example, generally more reduced on the worse side, possibly absent on one side and normal on the other. Armswing is absent after a stroke. In patients with ataxic syndromes the arms are often abducted, as if supplying extra balance. Tremor may be seen only during walking in Parkinson's disease, whereas tremor is not seen during walking in patients with essential tremor. In choreiform disorders, such as generalized tardive dyskinesia or Huntington's disease, the armswing is often excessive. It may also be excessive in some disorders in which there is a short stride, as if to compensate.

### 6. Turning

A normal turn requires a pivot. One foot is kept on the ground and rotation occurs on the ball of that foot. In Parkinsonism the patient turns “en bloc,” in one piece, using two or more steps. Sometimes the patient turns in a large circle, as if making a U-turn. Turning frequently causes loss of balance, in all gait disorders.

## 7. Pull test

This test, like the Romberg, tests balance. The pull test is performed by informing the patient that you are going to pull him off balance and that he should try to keep from falling, taking as few steps as possible. It is important to pull hard enough to make the patient take a step or two. Taking more than three steps is considered abnormal, indicative of a balance problem.

## INTERPRETATIONS

As in most aspects of the neurological examination, the cardinal feature is symmetry. When the stride length is different on the two sides, we usually see a limp or scuffing of one foot. Asymmetric armswing is obvious, once you look for it. When the gait looks abnormal, try to break it down into the component parts listed above to see if the abnormality can be localized. When the patient shifts weight suddenly, it is often an indication of joint pain, and this should be inquired about.

### Examples:

1. **Foot drop:** the affected foot is lifted higher than normal and then the ball of the foot is dropped onto the ground or is thrown forward and slapped.

2. **Hip weakness** produces a waddling gait. To lift the swing leg and move it forward, weight is shifted to the supporting leg by tilting the pelvis up on the swing side. This is usually a symmetric problem so that the hip swivels up and down.

3. **Parkinsonism** is one of the most common gait in the elderly, and not always pathological.<sup>7</sup> The posture is stooped, the stride length is reduced and there is a tendency for a flat foot strike. Armswing is reduced. Turning is without a pivot and balance is impaired on the pull test. As the gait deteriorates, the patient will often be flexed at the knees, which causes a major increase in weightbearing problem for the thigh muscles.

4. **Ataxic:** this may be from cerebellar dysfunction, vestibular impairment or sensory denervation. The stance may be normal or wide, but the gait is irregular and often lurching, with a tendency to veer to one side, or to either side, with increased problems on turning.

Armswing is normal, but sometimes reduced when the arms are abducted, acting like a balancing rod used for tight-rope walkers. Alcohol intoxication produces an ataxic gait.

5. **Post stroke:** armswing is lost on the affected side, and the arm is often flexed at the elbow, and wrist, while adducted at the shoulder. The affected leg is typically kept in extension, as if acting like a crutch. The leg is moved in a circular fashion as the knee is not bent. The ball of the foot usually hits the ground first.

6. **Spastic:** this is seen in children or adults with cerebral palsy, but also with other bilateral corticospinal dysfunction, typically after bilateral strokes, multiple sclerosis or cervical myelopathy, as occurs with cervical spondylosis. The posture is normal. The legs are stiff and do not bend normally at the knees. The base narrows. Because the legs are kept extended, the ball of the foot usually hits the ground first. Stride length is reduced. Armswing changes are determined by the level of the lesions so that in brain diseases, the armswing is reduced. When the spastic gait arises from a cervical process, it may or may not be reduced.

7. **Cautious gait.** This is the gait of someone fearful of falling, which, paradoxically, may increase the risk. The patient walks as if on ice, with slow, deliberate steps, placing each foot flatly and solidly on the ground before advancing.

8. **Astasia abasia.** This is an old term, meaning "can't sit, can't stand," used for conversion disorder, or psychogenic gait disorders. Although these are considered rare in the elderly, they do occur. Generally the gait is extremely bizarre, and, unlike other gait disorders where the body tries to minimize risk and effort, these generally maximize effort and produce convoluted postures, standing on one leg and other unusual stresses to the balance systems. Contrary to popular belief, these disorders are not generally associated with "belle indifference" or with the absence of falls or injuries.

## REFERENCES

1. Moylan CK, Binder EF. Fall in older adults. *Am J Med* 2007;120:493-7.
2. Tinetti ME. Preventing falls in elderly persons. *NEJM* 2003;348:42-9.
3. Tinetti ME, Speechley M, Ginter SF. Risk factors for falls among elderly persons living in the community. *NEJM* 1988;319:1701-7.

4. Nevitt MC, Cummings SR, et al. Risk factors for recurrent nonsyncopal falls. *JAMA* 1989;261:2663-8
5. Nevitt MC, Cummings SR, Hudes ES. Risk factors for injurious fall. *J Gerontol* 1991;46:M164-M170.
6. Odenheimer G, Funkenstein H, et al. Comparison of neurologic changes in "successful aging" persons vs the total aging population. *Arch Neurol* 1994;41:573-80.
7. Critchley M. Neurological changes in the aged. *Res Publ Assoc Res Nerv Mental Dis* 1956;35:198-223.

[http://medlib.med.utah.edu/neurologicexam/html/gait\\_abnormal.html](http://medlib.med.utah.edu/neurologicexam/html/gait_abnormal.html) contains information on gait including video examples of a doctor simulating and explaining common gait disorders as well as including examples. Other websites cited include information about gait, both for health professional and patient.

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# Parkinson's Disease In the Elderly

Marie-Hélène Saint-Hilaire, MD, FRCPC

*An 82 year old woman was admitted to a rehabilitation hospital after sustaining a pelvic fracture. She had a past history of osteoporosis and chronic lower back pain treated with epidural steroids. For the past two years she had had several falls associated with fractures. She also reported drooling, difficulties swallowing, gait shuffling and freezing, and tremor in the left hand, more at rest than with action. She had no cognitive impairment. She was started on a small dose of levodopa carbidopa 25/100, a half three times a day. She responded very well, stopped drooling, and had improvement in swallowing, gait, and tremor. However, her balance remained impaired and she continued to need a walker.*

This case exemplifies some of the challenges in diagnosing and treating elderly patients with **Parkinson's Disease (PD)**: 1) concurrent medical conditions, such as arthritis, can affect mobility, and symptoms can overlap with the symptoms of PD, thus delaying the diagnosis; 2) although treatment with levodopa is beneficial, it does not eliminate gait and balance problems, which are major causes of morbidity.

Age remains the single most important risk factor in PD. Although the average age of onset of PD is around 60, the incidence rates consistently increase through age 85.<sup>1</sup> Aging does appear to directly influence the clinical expression of PD, and late onset PD patients offer special challenges because of polypharmacy, multiple pathology, and coexisting cognitive problems. This article will review the specific aspects of the clinical presentation, differential diagnosis and treatment of PD and its complications in the elderly population.

## CLINICAL PRESENTATION

The diagnosis of PD is based on the history and the clinical examination. It requires the presence of two of the following: rest tremor, bradykinesia or ri-

gidity. Asymmetry of physical findings is important to support the diagnosis, as is a good response to levodopa.

Several clinical features help to distinguish idiopathic PD from other causes of parkinsonism. The presence of early falls, a poor response to levodopa, symmetry of signs at onset, or significant autonomic dysfunction should raise the suspicion that the patient may not have idiopathic PD.

In addition, significant cognitive decline and hallucinations, within one year of onset of the parkinsonian signs is suggestive of a diagnosis of dementia with Lewy Bodies. Concomitant PD and **Alzheimer Disease (AD)** are also possible in this age group. The diagnosis can be difficult, because some patients with AD have parkinsonian features. The presence of an asymmetric rest tremor, and improvement of the motor signs with levodopa lend support to a diagnosis of PD.

It is always necessary to review all the medications taken by the patient, because many have extrapyramidal side effects. Potential culprits include atypical neuroleptics (i.e. risperidone), antiemetics (i.e. metoclopramide), some antidepressants (i.e. fluoxetine) and some antiepileptics (i.e. valproic acid). Other conditions to exclude, especially in the elderly, are cerebrovascular disease and normal pressure hydrocephalus which usually present as a gait disorder or "lower body parkinsonism."

Patients with late onset PD progress at a greater rate and are more cognitively impaired than those with early onset disease. They also have more bradykinesia and postural instability<sup>3</sup>. Lack of tremor, male sex, and associated comorbidities are also associated with a more rapid rate of progression<sup>2</sup>.

## NON-MOTOR SYMPTOMS

Non-motor symptoms are increasingly recognized as an intrinsic feature of PD. Their prevalence is high: A survey found that 88% of PD patients had at least one non-motor symptom, and 11% had five.<sup>4</sup> With improvement in the treatment of PD motor symptoms, non-motor symptoms, such as dementia and depression, have become an important cause of dis-

ability.<sup>5</sup> They are however under recognized because their symptoms can overlap with the symptoms of PD.

Non-motor symptoms affect several domains: neuropsychiatric, autonomic, sensory, sleep, and dermatologic. Dementia, depression and autonomic symptoms are often the most problematic in elderly PD patients.

## DEMENTIA

The prevalence of dementia in PD varies between 10 and 44% depending on the diagnostic criteria used and the nature of the population studied. The risk increases with age, with one study finding that 65% of PD patients over the age of 85 were demented.<sup>6</sup> Risk factors include older age at onset, and initial manifestations of hypokinesia and rigidity.<sup>7</sup> The dementia in PD usually does not appear at the onset of the disease. It is characterized by impaired executive function, visuospatial abnormalities, impaired memory, and language deficits.<sup>8</sup> In elderly patients, superimposed cerebrovascular disease can contribute to cognitive problems. Dementia is a major factor in the management of PD, limiting the drug therapy that can be used, and leading to earlier nursing home placement and decreased survival.<sup>2</sup>

## DEPRESSION

Around 40% of subjects will have depression.<sup>9</sup> Although there may be a psychological response to living with a progressive neurological disease, there is evidence that depression in PD is related to the underlying pathology of the disease.

There is overlap between the symptoms of depression and those of PD which can make the diagnosis challenging. The nature of the depression in PD is more characterized by pessimism, hopelessness and poor motivation, with less feeling of guilt and self blame than in depressed elderly subjects without PD. Psychotic features are rare.<sup>10</sup>

## AUTONOMIC DYSFUNCTION

Symptoms of autonomic dysfunction become more prominent as PD progresses. They also increase with age and medication use.<sup>11</sup> They include bladder dysfunction, constipation, orthostatic hypotension, abnormal sweating and sexual dysfunction.

In addition, age itself affects autonomic function, as do concurrent diseases such as diabetes and hypertension, and medications, including some used to treat PD.

### Orthostatic hypotension

Falls in **blood pressure (BP)** occur particularly when getting up in the morning, or after meals. They manifest as dizziness when the patient stands, but can also present as fatigue or episodes of confusion. Critical review of all prescribed medications is necessary but sometimes specific treatment such as fludrocortisone or proamatine must be instituted.

### Bladder symptoms

Symptoms of urgency, frequency, nocturia, and incontinence are common in advanced PD. They result from detrusor hyperreflexia with or without detrusor/sphincter dyssynergia. In addition, they can be complicated by prostatic hypertrophy in males. Unfortunately medications for detrusor hyperreflexia are anticholinergic and can exacerbate confusion in elderly PD patients. Their risks and benefits must be carefully weighed.

### Constipation

Constipation is very common in PD, because of a combination of autonomic dysfunction with delayed transit time, and immobility, drug therapy, poor diet and lack of appropriate hydration. An aggressive bowel regimen may be necessary to avoid impaction.

### TREATMENT

Treatment must be individualized to each patient's needs, and the functional and cognitive status. Symptomatic therapy is introduced when the patient is functionally disabled. Levodopa/carbidopa is still the most effective medication for the motor symptoms of PD, and is better tolerated than Dopamine Agonists, amantadine or anticholinergics in elderly patients. It is initiated at a low dose, and increased slowly to minimize side effects. The optimal dose is the lowest one that will maintain adequate function. As the symptoms of PD progress, the dosage of the medication will need to be adjusted. However certain symptoms such as gait freezing, falls, hypophonia, and dysphagia do not respond well to drug treatment, and in these cases physical therapy and speech therapy may be helpful.<sup>12, 13</sup>

The treatment of the non-motor symptoms of PD must be addressed specifically and separately from the treatment of the motor symptoms. The only medication approved for the treatment of PD dementia is Rivastigmine.<sup>14</sup> There is no medication specifically approved for the treatment of depression, bladder or sexual dysfunction, constipation, or orthostatic hypotension in PD. For any treatment being considered, the clinician must weigh the potential benefit versus the risk of side effects.

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## Patients with late onset PD progress at a greater rate and are more cognitively impaired than those with early onset disease.

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### CONCLUSION

Elderly PD patients have more gait and balance difficulties, more depression, cognitive problems, and autonomic dysfunction, in addition to concurrent diseases such as cardiac and cerebrovascular disease. Drug therapy can be limited by neuropsychiatric side effects, and has marginal benefit for gait, balance, and swallowing difficulties. In this situation a non-medical approach involving physical and speech therapies becomes an important part of the management. A dietitian can also be involved to recommend strategies to maintain weight, and an occupational therapist can evaluate the home environment to improve safety. As the disease progresses, it may become increasingly difficult for patients to go to a specialty clinic. The primary care provider then becomes more involved in the management of the patient but must have access to consultation with the patient's specialist if necessary. The care of patients with advanced PD is complicated by the fact that the caregiver, usually a spouse, is also likely to be elderly and to suffer from a chronic illness.

### REFERENCES

1. Mayeux R, Marder K, et al. The frequency of idiopathic Parkinson's disease by age, ethnic group, and sex in northern Manhattan, 1988-1993. *Am J Epidemiol* 1995; 142:820-7.
2. Suchowersky O, Reich S, et al. Practice Parameter Report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurol* 2006; 66: 968-75.
3. Diederich NJ, Moore CG, et al. Parkinson disease with old-age onset. *Arch Neurol* 2003; 60:529-33.
4. Shulman LM, Taback RL, et al. Comorbidity of the nonmotor symptoms of Parkinson's disease. *Mov Disord* 2001; 16: 507-10.
5. Weintraub D, Moberg PJ, et al. Effect of psychiatric and other nonmotor symptoms on disability in Parkinson's disease. *J Am Geriatr Soc* 2004; 52:784-8.
6. Mayeux R, Chen J, et al. An estimate of the incidence of dementia in idiopathic Parkinson's disease. *Neurol* 1990; 40:1513-6.
7. Miyasaki JM, Shannon K, et al. Practice Parameter Report of the Quality Standards Subcommittee of the American Academy of Neurology. *Neurol* 2006; 66: 996-1002.
8. Aarsland D, Andersen K, et al. Prevalence and characteristics of dementia in Parkinson disease. *Arch Neurol* 2003; 60:387-92.
9. Cummings JL. Depression and Parkinson's disease. *Am J Psychiatry* 1992; 149, 4: 443-54.
10. Brown RG, MacCarthy B. Psychiatric morbidity in patients with Parkinson's disease. *Psycholog Med* 1990; 20: 77-87.
11. Verbaan D, Marinus J, et al. Patient-reported autonomic symptoms in Parkinson disease. *Neurol* 2007; 69: 333-41.
12. de Goede CJ, Keus SH, et al. The effects of physical therapy in Parkinson's disease. *Arch Phys Med Rehabil* 2001; 82: 509-15.
13. Ramig LO, Sapir S, et al. Changes in vocal loudness following intensive voice treatment (LSVT) in individuals with Parkinson's disease. *Mov Disord* 2001; 16: 79-83.
14. Emre M, Aarsland D, et al. Rivastigmine for dementia associated with Parkinson's Disease. *NEJM* 2004, 351: 2509-18.

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# Epilepsy In the Elderly

Amanda M. Diamond, MD, and Andrew S. Blum, MD, PhD

People over age 65 are the most rapidly increasing segment of the population. The incidence of epilepsy rises significantly with age, starting with 25.8/100,000 person-years for the ages of 60-74 and increasing to 101.1/100,000 for the ages of 75-89.<sup>1</sup> The prevalence of epilepsy is 1.5% in the elderly, twice that of young adults.<sup>2</sup> Also, the prevalence of epilepsy in nursing home residents is even higher than that in the general population. Review of elderly nursing home residents reveals that up to 10.5% are receiving anticonvulsants, along with an average of 5.6 other medications.<sup>3,4</sup>

## ETIOLOGY AND SEMIOLOGY

In the elderly, cerebrovascular disease accounts for 30-40% of cases.<sup>2</sup> The risk of seizures within the first year after stroke has been estimated to be 23 times that of the general population.<sup>5</sup> Other causes include Alzheimer's disease, trauma, brain tumors and infection. About half of cases are cryptogenic, and it is presumed that vascular etiologies account for much of this group too.<sup>2</sup>

Further complicating the diagnosis is the sometimes vague history. The events are often unwitnessed and may involve confusional symptoms. Elderly patients do not always have classical seizure semiologies or auras as in younger patients. Symptoms that may suggest alternate diagnoses can further confound; these may include tremor, headache and dizziness, among others.<sup>6</sup> In a recent VA study, 27% of patients ultimately diagnosed with epilepsy were initially misdiagnosed as having syncope, altered mental status and confusion.<sup>6</sup> Postictal periods tend to be more prolonged in this age group, at times lasting for several days.<sup>7</sup>

## DIFFERENTIAL DIAGNOSIS AND WORK-UP

The differential diagnosis in this population is extensive and includes **transient cerebrovascular symptoms** (TIA), syncope (including convulsive syncope), confusional migraine, drug intoxication, infection, psychiatric disorders, transient

global amnesia and dementia. Multiple metabolic etiologies can also be considered, including hypo- or hyperglycemia, thyroid dysfunction, hypercapnia, uremia and hyponatremia.<sup>7</sup>

Electroencephalography can be pivotal, but one must be aware of potential false positive findings. Rhythmic runs of temporal theta activity may be seen in drowsiness as a normal or benign finding. Focal slowing may be present with underlying cerebrovascular insults. **Wicket spikes and subclinical rhythmic electrographic discharges in adults (SREDA)** are two other benign variant patterns that can be misinterpreted. **Long-term video-EEG monitoring (LTM)** may prove invaluable in evaluating recurrent spells. Brain imaging should be performed, preferably MRI. Other testing considerations include basic metabolic screens, sleep studies, Holter monitoring, echocardiogram, tilt-table and/or vestibular testing, where appropriate, to rule out competing diagnoses.

## Several medications more common in the elderly can pose significant interactions with some AEDs.

### MANAGEMENT

The assistance of family members and caregivers may be needed to ensure patient safety. In some, help with medications may be critical and the importance of compliance should be emphasized. Patients may need a home safety evaluation. Medication lists should be reviewed for potentially pro-convulsive medications.

Medical management of epilepsy in the elderly poses several distinct challenges. Pharmacokinetic and pharmacodynamic parameters change significantly with age, making the potential for side effects of **anti-epileptic drugs (AEDs)** greater and decreasing the predictability of dose – blood

level relationships.<sup>8</sup> Decreased protein binding is more common in the aged; this may lead to higher AED free fractions, which can lead to earlier toxicity with more highly protein bound AEDs such as phenytoin and valproate. Also, the elderly may have a lower volume of distribution. Decreased clearance due to hepatic or renal insufficiency may also promote higher than expected serum AED levels.<sup>9</sup> The CNS threshold for AED toxicity may also be lower in the elderly. Delayed esophageal and gastric emptying, altered postprandial gastric pH, and delayed intestinal transit all affect absorption in the elderly.<sup>10</sup>

Several medications more common in the elderly can pose significant interactions with some AEDs. For example, highly protein bound AEDs (e.g. phenytoin and valproate) may interact with warfarin and other highly protein bound concomitant medications, leading to complex untoward interactions. Cytochrome P450-inducing AEDs (phenytoin, carbamazepine, phenobarbital) accelerate the clearance of some hepatically cleared drugs, including some chemotherapeutics.<sup>9</sup>

Several AEDs are known to contribute to accelerated bone demineralization. The older generation of AEDs seems to be worse in this regard. The elderly are at a higher risk of osteoporosis and related fractures, so this may be relevant. Balance and cognitive concerns are also enriched in the elderly. Some AEDs appear to pose greater balance risks (e.g. phenytoin, carbamazepine)<sup>11</sup> or cognitive risks (e.g. phenobarbital, topiramate). Hyponatremia, a problem occasionally provoked by carbamazepine and oxcarbazepine, is more frequent in the aged, particularly with concomitant diuretic use.

The elderly are likely to have more medical diagnoses and take more medications than a younger population and thus are far more susceptible to drug-drug interactions.<sup>3</sup> AEDs that act as hepatic enzyme inducers or inhibitors will greatly augment this problem of drug-drug interaction burden in this age group. This is more often the case for the older AEDs. The newer (2<sup>nd</sup> generation) AEDs exhibit less hepatic effects and are more often



renally cleared, have lower protein binding, and hence have fewer drug interactions as a group. Additionally, the elderly may have more difficulty paying for AEDs, contributing to potentially poor compliance or restricted choice of agent.

### ANTIEPILEPTIC CHOICES IN THE ELDERLY

Little comparative efficacy data exist to help guide the use of AEDs in the elderly. Most clinical trials of AEDs have been conducted in younger and healthier adult populations. In the UK, Brodie et al<sup>12</sup> compared the efficacy and tolerability of lamotrigine vs. carbamazepine in elderly patients with new onset epilepsy. They found similar efficacy but better tolerability in the lamotrigine arm.<sup>12</sup> A similar US study compared gabapentin vs. lamotrigine vs. carbamazepine in the elderly and found better tolerability in the gabapentin and lamotrigine arms vs. carbamazepine with no significant efficacy distinctions.<sup>13</sup> However, a later international study that compared lamotrigine and carbamazepine using a more flexible dosing schedule and slow release carbamazepine did not observe as marked a tolerability difference.<sup>14</sup>

It is helpful when treating the elderly with epilepsy to keep the regimen as simple as possible. Medication choices should be tailored to seizure type, with most being partial onset rather than generalized in this population. Tolerability concerns should be considered carefully in AED selection and individualized accordingly. Doses should generally begin quite low and titration should be as slow as possible.<sup>15</sup> Levels may be helpful. Cost may be a deciding factor for some.

Fortunately, the majority of elderly cases are very responsive to treatment. Refractory epilepsy may be infrequently encountered in this group. Non-pharmacologic treatment options may be limited. Surgical resection is not as commonly used in this age group. Vagal nerve stimulation may prove an option for some when AEDs prove inadequate and surgery is not a viable option.

### SUMMARY

The incidence of epilepsy is higher in the elderly than in younger adults. This community presents a challenging set of considerations in diagnosis and management. Numerous other processes may mimic sei-

zures in the elderly. After careful diagnosis, AED choices should take into account metabolism, drug-drug interactions, co-morbidities, and side effect profiles while striving to achieve seizure freedom.

### REFERENCES

- Hussain SA, Haut SR, et al. Incidence of epilepsy in a racially diverse, community-dwelling, elderly cohort. *Epilepsy Res* 2006;71:195-205.
- Hauser WA. Epidemiology of Seizures in the elderly. In: *Seizures and Epilepsy in the Elderly*. Rowan AK, Ramsay RE, Eds. Boston, Butterworth-Heinemann, 1997:7-18.
- Lackner TE, Cloyd JC, et al. Antiepileptic drug use in nursing home residents. *Epilepsia* 1998;39:1083-7.
- Garrard J, Cloyd JC, et al. Factors associated with antiepileptic drug use among nursing home elderly. *J Gerontol Med Sci* 2000;55:384-92.
- So EL, Annegers JF, et al. Population-based study of seizures disorders after cerebral infarction. *Neurology* 1996;46:350-355.
- Ramsay RE, Rowan AJ, Pryor FM. Special considerations in treating the elderly patient with epilepsy. *Neurology* 2004;62(Suppl 2):S24-S29.
- Ramsay RE, Macias FM, Rowan AJ. Diagnosing epilepsy in the elderly. In "The Neurobiology of Epilepsy and Aging" (RE Ramsay, ed.) *Int Rev Neurobiol* 2007;81:1-14.
- Perucca E, Berlowitz D, et al. Pharmacological and clinical aspects of antiepileptic drug use in the elderly. *Epilepsy Res* 2006;68S:S49-S63.
- Boggs JG. Elderly patients with systemic disease. *Epilepsia* 2001;42:18-23.
- Gidal BE. Drug absorption in the elderly. *Epilepsy Res* 2006;68S:S65-S69.
- Fife TD, Blum D, Fisher RS. Measuring the effects of antiepileptic medications on balance in older people. *Epilepsy Res* 2006;70:103-9.
- Brodie MJ, Stephen LJ. Outcomes in elderly patients with newly diagnosed and treated epilepsy. *Int Rev Neurobiol* 2007;81:253-63.
- Rowan AJ, Ramsey MD, et al. New onset geriatric epilepsy. *Neurology* 2005;64:1868-73.

- Saetre E, Perucca E, et al. An international multicenter randomized double-blind controlled trial of lamotrigine and sustained-release carbamazepine in the treatment of newly diagnosed epilepsy in the elderly. *Epilepsia* 2007;48:1292-302.
- Leppik IE. Epilepsy in the elderly. *Epilepsia* 2006;47(Suppl 1):65-70.

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Discussion of off-label usage of any product: gabapentin and lamotrigine. Reference is made to studies performed on the use of these medications in elderly patients with epilepsy.

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# Peripheral Neurology: Special Concerns In the Elderly

George M. Sachs, MD, PhD

**Changes in the peripheral nervous system** produce a major impact on safety and quality of life in the elderly. The challenges of aging reflect alteration of both neuromuscular and sensory function. Of particular concern are motor and proprioceptive deficits increasing the risk of falls. In addition, changes in sensory pathways may predispose the elderly to neuropathic pain. This review will focus on aspects of aging in muscle and nerve relevant to these issues.

## SARCOPENIA

Sarcopenia refers to the degenerative loss of muscle mass and strength with increasing age. It begins in mid-life and accelerates significantly in the seventh and eighth decades. Both active and sedentary individuals suffer sarcopenia, though it is worsened by inactivity. Muscle mass decreases earlier in women but men experience greater overall loss, averaging 30-40% of their muscle mass by age 80.<sup>1</sup>

The causes of sarcopenia are multiple. They include loss of muscle cells as well as hormonal changes that influence the growth and regeneration of different cells within muscles. In addition, degeneration of spinal motor neurons has a profound effect on the muscles they innervate.<sup>1,2</sup>

Satellite cells are essentially stem cells residing in muscle. In response to physical trauma or even vigorous exercise, satellite cells proliferate to form new muscle fibers or fuse with damaged fibers to repair them. Most studies have shown reduction in the number of satellite cells with aging. This, along with changes in growth factor and hormone levels, decreases the regenerative capacity of muscle over time.<sup>3</sup>

**Testosterone (T), growth hormone (GH) and insulin-like growth factor (IGF)** all regulate protein synthesis within muscle. IGF appears to increase synthesis of actin and myosin (the principal contractile proteins in muscle), while GH and T promote protein stability. Decreasing levels for all three of these hormones in mid to late life presumably contribute to sarcopenia. The

role of estrogen is less well established, though its sharp perimenopausal fall may help explain the relatively early onset of muscle loss in women.<sup>1,2</sup>

The interaction of nerve and muscle can be described in terms of "motor units". One motor unit consists of a single spinal motor neuron and the muscle fibers it supplies through the branches of its axon. A motor unit in hand muscles typically includes about 100 muscle fibers, where calf and thigh muscle motor units include 1000-2000.<sup>4</sup> In addition to transmitting electrical excitation, each motor neuron provides trophic support to the muscle fibers in its motor unit. Once a muscle fiber loses its nerve input, it undergoes atrophy unless connection with another nerve terminal can be restored.

## Growth hormone supplementation has not demonstrated improvement in muscle mass or strength in elderly men or women.

Anatomical and physiological studies have shown that the number of motor units (i.e. the number of spinal motor neurons) supplying limb muscles remains fairly constant up to age 60. Beyond that, healthy individuals typically lose about half of their motor units between ages 60 and 80.<sup>5</sup> Sprouting of terminal axons from remaining motor neurons initially compensates for those that are lost, however as this process continues, muscle fibers are inevitably left to atrophy without nerve supply. This neurogenic atrophy is likely the major cause of sarcopenia.<sup>1,2</sup>

## CHANGES IN MUSCLE DYNAMICS

Two major types of motor units can be distinguished by their morphologic and physiologic characteristics. In Type 1 motor units, small motor neurons in-

nerve muscle fibers with slow contraction times. Type 2 motor units contain larger motor neurons and muscle fibers that contract more quickly. Aging muscles may show a shift toward the slower contractions of Type 1 units for a few reasons. First, aging leads to selective Type 2 fiber atrophy. Whether this simply reflects decreased physical activity or some other more specific feature of aging muscles is not clear. Secondly, muscle fibers may actually transform from Type 2 to Type 1 as a result of the denervation and reinnervation that follows loss of motor neurons.<sup>6</sup>

The predominance of Type 1 fiber contraction in aging muscles inhibits the ability to make quick, forceful movements. This poses particular problems for rapid postural reflexes important in maintaining balance. In addition to overall muscle weakening and proprioceptive deficits (described below), slowing of postural adjustments presents a major risk for falls in the elderly.

## THERAPEUTIC CONSIDERATIONS FOR THE AGING NEUROMUSCULAR SYSTEM

The literature regarding testosterone replacement for sarcopenia presents a mixed picture. Meta-analyses have suggested that injected testosterone can produce a moderate increase in muscle mass and strength in older men.<sup>7</sup> Concerns over adverse effects temper any current enthusiasm. In particular, no large, prospective studies have looked at rates of prostate cancer in older men receiving testosterone supplementation.<sup>8</sup>

Growth hormone supplementation has not demonstrated improvement in muscle mass or strength in elderly men or women. Furthermore, the addition of growth hormone to a resistance exercise program does not appear to confer additional benefit. Adverse effects including carpal tunnel syndrome, hyperglycemia, edema and orthostatic hypotension led to high drop-out rates in treatment groups of some studies.<sup>2</sup>

Relatively few clinical trials have evaluated the muscular effects IGF-1 administration. A study assessing a two

month course of IGF-1 complexed to a binding protein, found that grip strength improved by 11%.<sup>9</sup> The effect of estrogen replacement on sarcopenia has also received little study to date. Reported effects of estrogen on lean body mass have been mixed and changes in strength or contraction speed have yet to be investigated.<sup>10</sup>

Exercise, in the form of resistance training, appears to be the most effective treatment to counteract muscular decline in the elderly. Resistance training programs can achieve the same percentage gain in muscle mass and strength at age 80 as they do in young adults.<sup>2</sup> Although the mechanism is not understood, resistance training also increases whole muscle contraction speed with an associated improvement in balance.

This likely reflects adaptive coordination among different motor units since the contraction speed of individual muscle fibers may actually decrease with resistance training.

### AGING IN PERIPHERAL SENSORY NERVES

Signs of peripheral sensory loss become increasingly prevalent with advancing age. Loss of ankle reflexes and decreasing distal vibration sense are particularly common. One study revealed that at least one of these deficits occurred in 26% of individuals aged 74-84 and in 54% of those older than 85.<sup>11</sup> Correspondingly, anatomic studies have documented lower numbers of sural nerve fibers and physiologic studies have shown lower amplitudes of sural nerve response in "normal" older individuals.

Since these findings are so common, they are often considered part of "normal aging." This notion overlooks the impact of peripheral sensory deficits on balance and gait in the elderly. A number of studies have documented that peripheral sensory loss is an important risk factor for falls. In some studies, the existence of lower extremity neuropathy increased the frequency of falls about 20-fold.<sup>12</sup> Even modest loss of proprioception can pose significant risk when combined with slowed muscular response and visual or vestibular disturbances so often

seen in the elderly. Though neuropathy in older individuals is not frequently reversible, simple preventive measures including use of a cane, nightlights and shower chairs should not be neglected.

### NEUROPATHIC PAIN

The age-related changes discussed above concern the largest sensory fibers. Evaluation of small caliber sensory fibers is more challenging but crucial to understanding neuropathic pain. This is an important issue for aging populations given the striking predominance of certain neuropathic pain conditions in the elderly.<sup>13</sup>

In contrast to the clear age-related loss of large sensory fibers, anatomic studies have demonstrated relative preservation of small-caliber, pain sensing fibers in the elderly.<sup>14</sup> This raises an interesting possibility regarding the development of post-herpetic neuralgia, a condition which is rare below the age of 50 but complicates 20-40 % of zoster cases past the age of 60. Studies testing sensory function in patients with post herpetic neuralgia have indicated that vibratory sensory loss (presumably due to large-fiber failure) is more prominent than small-fiber (pain and temperature) deficit.<sup>15</sup> This suggests that preferential large fiber loss with aging may be an important risk factor for the development of neuralgias and neuropathic pain. If small sensory fibers are relatively preserved, they may present an important target for treatments. The use of local or topical agents aimed at small fibers could supplement or replace systemic medications that are limited by adverse reactions in elderly patients.

### REFERENCES

1. Thomas DR. Loss of muscle mass in aging. *Clin Nutrition* 2007;26:389-99.
2. Borst SE. Interventions for sarcopenia and muscle weakness in older people. *Age Ageing* 2004; 33:548-55.
3. Shefer G, Van de Mark DP, et al. Satellite-cell pool size does matter. *Development Biol* 2006;294:50-66.
4. Feinstein B, Lindgard E, Nyman E. Morphological studies of human motor units in normal human muscles. *Acta Anat* 1955; 2:127-39.
5. Doherty TJ, Vandervoort AA, Brown WF. Effects of aging on the motor unit. *Can J Appl Physiol* 1993;18:331-58.
6. Doherty TJ. Aging and sarcopenia. *J Appl Physiol* 2003;95:1717-27.

7. Ottenbacher KJ, Ottenbacher ME, et al. Androgen treatment and muscle strength in elderly men. *J Am Geriatr Soc* 2006;54:1666-73.
8. Brand TC, Canby-Hagino E, Thompson IM. Testosterone replacement therapy and prostate cancer. *Curr Urol Rep* 2007;8:185-9.
9. Boonen S, Rosen C, et al. Musculoskeletal effects of the recombinant human IGF-1/IGF binding protein 3 complex in osteoporotic patients with proximal femoral fracture. *J Clin Endocrinol Metab* 2002;67:1593-9.
10. Thorneycroft JH, Lindsay R, Pickar JH. Body composition during treatment with conjugated estrogens with and without medroxyprogesterone acetate. *Am J Obstet Gynecol* 2007;197:e1-7.
11. Resnick HF, Vinik AI, et al. Age 85+ years accelerates large-fiber peripheral nerve dysfunction. *J Gerontol Med Sci* 2001;56:M25-M31.
12. Richardson JK, Hurvitz EA. Peripheral neuropathy. *J Gerontol Med Sci* 1995; 50:M211-M215.
13. Hall GC, Carroll D, et al. Epidemiology and treatment of neuropathic pain. *Pain* 2006;122:156-62.
14. Lauria G, Holland N, et al. Epidermal innervation. *J Neurol Sci* 1999;164:172-8.
15. Baron R, Haendler G, Schulte H. Afferent large fiber neuropathy predicts the development of postherpetic neuralgia. *Pain* 1997; 73:231-8.

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Discussion of off-label usage of any products or services

\*use of supplemental testosterone, growth hormones and IGF-1 to reduce sarcopenia is investigational

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# Sleep Disorders In the Elderly

Jean K. Matheson, MD

**Older patients frequently complain of** difficulty initiating sleep, sleep maintenance or excessive daytime sleepiness, while bed partners or caretakers are distraught by episodes of nocturnal confusion or belligerence.

Sleep disorders may represent a primary disorder of mechanisms regulating sleep or failure of a specific organ system manifesting in a unique way during sleep. Sleep complaints should not be ignored or treated empirically with pharmacologic agents without analysis of the etiology.

## POLYSOMNOGRAPHY

Polysomnography is the term applied to the simultaneous and continuous measurement of multiple physiologic parameters during sleep. In practice, the polysomnogram (PSG) has come to mean a specific type of polysomnographic study in which measurements allow for 1) the identification of sleep stage, 2) monitoring of cardio-pulmonary function and 3) monitoring of body movements during sleep.

## SLEEP STAGING AND ARCHITECTURE

Rapid eye movement (REM) sleep, sometimes called dreaming sleep, and non-REM (NREM) sleep are the two sleep states. NREM and REM sleep alternate in recurring cycles of approximately 90 minutes. NREM sleep had been divided into four stages (1-4), representing progressive deepening of sleep. A recent revision of staging nomenclature now identifies these stages as N1, N2 and N3.<sup>1</sup>

In what has been thought to be normal aging nocturnal awakenings and wake time increase. N3 sleep decreases and REM time usually remains relatively constant. Stage one sleep increases as a reflection of sleep disruption. Sleep efficiency, the ratio of time asleep to time in bed, decreases secondary to both increased time in bed and increased wake. However, many of these changes attributed to "normal" aging may also be exacerbated or induced by what we now know to be common and treatable sleep disruptors such

as sleep apnea and periodic leg movements as described below.

## SLEEP- RELATED BREATHING DISORDERS (SRBDs)

The most important cause of sleep disruption is sleep-disordered breathing. The term "sleep-disordered breathing" primarily refers to the sleep apnea syndromes, but also includes disorders that result in nocturnal hypoventilation and hypoxemia such as restrictive and parenchymal pulmonary diseases. While well known to cause excessive daytime sleepiness, the sleep apnea syndromes, both central and obstructive, are also important contributors to difficulty initiating and maintaining sleep because of frequent nocturnal arousals. Current definitions that have the most widespread clinical use are based on guidelines provided by the Center for Medicare and Medicaid Services (CMS):<sup>2</sup>

*Apnea:* An apnea is defined as the absence of airflow for at least 10 seconds.

There are three types:

*Obstructive apnea:* Absence of airflow for at least 10 seconds with evidence of persistent respiratory effort.

*Central apnea:* Absence of airflow for 10 seconds without evidence of any of respiratory effort.

*Mixed Apnea:* Absence of airflow for 10 seconds with initial absence of effort followed by a return of respiratory effort before resumption of airflow.

*Hypopnea:* The term hypopnea refers to a decrease in airflow. By CMS criteria: "Hypopnea in adult patients is defined as an abnormal respiratory event lasting at least 10 seconds with at least a 30% reduction in thoracoabdominal movement or airflow as compared to baseline, and with at least a 4% oxygen desaturation."<sup>2</sup>

*Apnea Hypopnea Index (AHI):* The total number of apneas and hypopneas are summed and divided by the number of hours of sleep. When used with the definitions above, the index is useful as a standardized measure that reflects sever-

ity of sleep disordered breathing  
*Respiratory Effort Related Arousal:* When airway resistance increases, oxygen saturation and airflow may stay the same as respiratory effort increases to overcome obstruction. This increased respiratory effort may induce an arousal that disturbs sleep termed, RERA, respiratory effort-related arousal. This event is NOT recognized by CMS and is sometimes called a "hypopnea without desaturation".<sup>1</sup>

The presence of 5 apneas/hour of sleep was previously deemed necessary to establish the presence of either "obstructive sleep apnea" or "central sleep apnea". It is now clear, however, that either incomplete obstructions or central hypoventilatory episodes without apnea induce physiologic changes of the same magnitude as apneas. CMS currently accepts a minimum AHI of 5 as evidence of sleep apnea that justifies treatment. However, RERAs, often occult contributors to sleep disruption, are not included in the CMS definition of sleep apnea, but are accepted by the American Academy of Sleep Medicine as determinants of obstructive sleep apnea syndrome.<sup>4</sup>

Well-documented risk factors for obstructive sleep apnea include obesity, large neck, upper airway structural abnormalities, nasal congestion, endocrine abnormalities, muscular weakness and sedating drugs.

In aging and degenerative neurological disease there are additional risk factors including laryngeal dysfunction and centrally-induced dysynergy of upper airway muscle activation in relationship to chest wall and diaphragmatic activation.

Central sleep apnea episodes usually represent Cheyne-Stokes breathing, a crescendo-decrescendo breathing pattern, common in the elderly, especially those with congestive heart failure. Periods of central apnea or hypopnea predispose to obstruction as well, because low airflow contributes to airway collapse. This type of breathing abnormality, once considered benign, may markedly disrupt sleep.<sup>4</sup>

## **TREATMENT OF SLEEP-DISORDERED BREATHING:**

Continuous positive airway pressure (CPAP) is the mainstay of treatment for obstructive sleep apnea and for some patients with predominantly central apnea. Airway pressure acts as a pneumatic splint to maintain upper airway patency during sleep. **Bi-level positive pressure (BIPAP)** is often better tolerated in the elderly because it provides an expiratory pressure always lower than inspiratory pressure, making expiration more natural and less effortful. The inspiratory–expiratory pressure difference also acts to augment ventilation in disorders associated with hypoventilation. New devices termed servo-ventilators are based on bi-level positive pressure technology, and can be used at home to treat patients with central components of their sleep-disordered breathing, especially Cheyne-Stokes respirations. These devices are an important addition to the treatment of sleep-disordered breathing in heart failure. Many new mask styles and variations on these devices including “smart” auto titrating machines can improve patient tolerance dramatically. Full face masks are now comfortable and useful in patients who may have rejected positive pressure previously because of nasal obstruction.<sup>3</sup>

Cheyne-Stokes respirations often respond to low-flow oxygen delivered through nasal prongs that generally are better tolerated than positive pressure. O<sub>2</sub> tends to decrease over-responsiveness to CO<sub>2</sub> that perpetuates the hyperventilation-apnea cycle. Obstructive episodes exacerbated by central apneas may also improve. Sedating medications, especially hypnotics and narcotics, can exacerbate sleep disordered breathing and are best avoided when sleep-disordered breathing is suspected.<sup>3</sup>

## **SLEEP RELATED MOVEMENT DISORDERS**

Sleep-related movement disorders include conditions in which simple stereotyped movements are present during sleep and induce sleep disruption. Difficulty initiating and/or maintaining sleep are the typical complaints.

### **Periodic Limb Movement Disorder**

The most prevalent of these disorders is **periodic limb movement disorder (PLMD)**, characterized by periods of repetitive stereotyped leg movements

that disturb sleep (PLMS). Many medications are implicated in the induction of periodic and aperiodic leg movements, most commonly SSRIs, SNRIs and tricyclic antidepressants.

### **Restless Legs Syndrome**

Restless legs syndrome (RLS) is characterized by 1) an urge to move accompanied by uncomfortable sensations, predominantly in the legs, that are 2) relieved by movement, 3) occur when sedentary and 4) are worse in the evening.<sup>5</sup> This syndrome is closely associated with PLMD. Restless legs syndrome, however, is a syndrome based on clinical, not polysomnographic criteria. The disorder is familial in approximately 50% of cases. Prevalence estimates vary between 5 and 20% of the general adult population in North America and the disorder appears to increase with age.<sup>5</sup> Abnormalities of both dopamine and iron metabolism are implicated in the underlying pathophysiology. Iron deficiency is known to exacerbate or precipitate restless legs and periodic leg movements in familial and non-familial cases. Secondary causes of RLS are subject to some debate due to limited data and include uremia, neuropathy, and medications, especially anti-dopaminergic drugs and SSRIs.

### **Management of Restless Legs and Periodic Limb Movements**

Periodic leg movements are often found incidentally on sleep studies and are not generally treated unless they are either accompanied by symptomatic restless legs, or clearly contribute to arousals. Recommendations for the management of restless legs have recently been published.<sup>6</sup> Serum ferritin should be checked in all patients; iron replacement is recommended in patients with ferritins below 45-50 ug/ml. Response to iron replacement may not occur in all patients and usually takes months to years; some clinicians advocate intravenous replacement in severe cases. Because antidepressant and antihistamine medications may exacerbate restless legs they should be avoided if possible. Some clinicians routinely screen for neuropathy with exam and metabolic studies. Ropinirole (Requip) and pramipexole, (Mirapex), both dopaminergic agonists FDA approved for RLS, are markedly effective. Because of the possibility of inducing a syndrome termed “augmentation”, it is best to use as low a single evening dose as possible. Augmen-

tation refers to the development of symptoms earlier in the day with increasing severity, further exacerbated by dosage increases. This phenomenon occurs most dramatically with the use of levodopa and less frequently with the dopaminergic agonists ropinirole and pramipexole. Gabapentin and low dose opioids are useful second line drugs that can be used alone or in combination with dopaminergic agonists for dosage sparing. Benzodiazepines are third line drugs because of limited efficacy and the development of tolerance.

### **REM Behavior Disorder**

Low muscle tone observed on PSGs during REM correlates with the normal paralysis that occurs during dreaming. This temporary paralysis, termed REM sleep atonia, prevents the dreaming subject from enacting dreams. REM Behavior Disorder is characterized by incomplete REM atonia associated with motoric activation during dreams.<sup>7</sup> Typically the patient, bed partner, or caretaker complains of violent, often injurious, activity during sleep. The patient may complain of a change in dream content with violence and running as typical themes. Sometimes the patient is able to incorporate ongoing conversation and activity into the dream, giving rise to the misperception that he is confused or hallucinating until he suddenly awakens and appear to “clear” his mental status. Underlying dementia or acute illness may impair the patient’s ability to report the perception of dreaming, resulting in misdiagnosis. Hospital caretakers usually dismiss even detailed dreaming reports as confusion. The disorder is frequently mistaken for “sundowning” or, because of the violent quality of the dreams, post-traumatic stress disorder.

REM behavior disorder occurs most frequently in older men with a mean age of 60. Over time it has become clear that RBD precedes other clinical signs or symptoms of some degenerative diseases, especially Parkinson’s disease, dementia with Lewy bodies and multisystem atrophy, (the “synucleinopathies.”) by years. Conversely, the prevalence of RBD in Parkinson’s disease is in the range of 33-60%. The combination of degenerative dementia and RBD is highly correlated with the diagnosis of dementia with Lewy bodies, based on clinical and pathologic criteria. There are rare published reports of RBD with a

“tauopathy” (e.g. pure Alzheimer’s disease, frontotemporal dementia, progressive supranuclear palsy). Some medications, especially NSRIs and SSRIs are known to precipitate and exacerbate the disorder. Idiopathic RBD also exists.<sup>7</sup>

### **Circadian Rhythm Disorders: Advanced Phase Sleep Disorder**

The tendency for elderly patients to spend more time asleep during the day and less time asleep at night raises the possibility that neurological dysfunction of the biologic clock within the suprachiasmatic nucleus of the hypothalamus mediates these changes. Advanced phase sleep disorder is a well-recognized circadian disorder in which patients complain of difficulty staying awake in the evening and early morning awakening.<sup>5</sup> This disorder is common in the elderly and may be mistaken for early morning insomnia.

Phase advance is exacerbated by visual impairment and low light exposure during the day; evening light exposure can therapeutically delay rhythms and improve sleep maintenance.<sup>8</sup>

### **REFERENCES**

1. The AASM manual for the scoring of sleep and associated events. Westchester, IL: American Academy of Sleep Medicine, 2007.
2. Medicare National Coverage Determinations Manual Chapter 1, Part 4 Section 240.4. [http://www.cms.hhs.gov/manuals/downloads/ncd103c1\\_Part4.pdf](http://www.cms.hhs.gov/manuals/downloads/ncd103c1_Part4.pdf)
3. Kryger MH, Roth T, Dement WC. *Principles and Practice of Sleep Medicine*. 4th ed. Philadelphia, PA: Elsevier/Saunders, 2005.
4. The international classification of sleep disorders : Diagnostic & coding manual, ICSD-2. 2nd ed. Westchester, IL: American Academy of Sleep Medicine, 2005.
5. Allen RP, Picchietti D, et al. Restless legs syndrome: Diagnostic criteria, special considerations, and epidemiology. A report from the restless legs syndrome diagnosis and epidemiology workshop at the National Institutes of Health. *Sleep Med* 2003;4(2):101-119.
6. Silber MH, Ehrenberg BL, et al. An algorithm for the management of restless legs syndrome. *Mayo Clin Proc* 2004;79:916-22.
7. Boeve BF, Silber MH, et al. Pathophysiology of REM sleep behaviour disorder and relevance to neurodegenerative disease. *Brain* 2007.
8. Campbell SS, Dawson D, Anderson MW. Alleviation of sleep maintenance insomnia with timed exposure to bright light. *J Am Geriatr Soc* 1993;41:829-36.

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## **Driving Safety Among Older Adults**

*Melissa M. Amick, PhD, and Brian R. Ott, MD*

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The number of individuals in the United States who are age 65 and older is expected to double by 2030.<sup>1</sup> As this population increases, so will the number of licensed older drivers. Increasing age is a significant risk factor for unsafe driving. The risk for crash involvement increases dramatically after the age of seventy; and drivers 85 and older have the highest driver fatality rate.<sup>2</sup> Drivers over the age of 70 have the highest annual fatality rate per miles driven compared to all age groups, except those aged 25 and younger.<sup>2</sup> In a study of mild dementia and non-demented drivers, baseline age significantly predicted performance on a road test independent of cognitive status.<sup>3</sup> These findings suggest that, as older people age, the risk of unsafe driving increases, and care providers will need to monitor their aging patients’ driving safety.

Research on driving safety in the elderly has mainly focused on drivers with dementia, who consistently perform more poorly on open road tests and simulated

driving tests compared to their non-demented counterparts.<sup>4</sup> For example, Duchek and colleagues found that 43% of participants with mild **Alzheimer’s disease (AD)** failed the road test, compared to 13% of patients with very mild AD and 3% of non-demented control participants.<sup>3</sup> Longitudinal data indicated that patients with mild AD experienced a more rapid decline in driving skills compared to the control group; however, patients with very mild AD did not differ significantly from either group.<sup>3</sup> Comparing crash rate records collected by the state registries, only one study has observed that patients with dementia are more frequently involved in accidents compared to a control group.<sup>4</sup>

There is also concern about the driving skills of patients with **Parkinson’s disease (PD)**, because of the motor and non-motor symptoms (visual changes and cognitive dysfunction). Interestingly, motor symptom severity and visual functioning do not consistently predict driving skills.<sup>5</sup>

While performance on neuropsychological measures does predict driving abilities, our research has found that PD drivers are infrequently observed to be unsafe drivers: only one of 25 participants assessed actually failed our road test. Rather, most participants received marginal or safe ratings.<sup>5</sup> Research in dementia and Parkinson’s disease emphasizes that mild degrees of motor slowing, cognitive dysfunction, and changes in vision may not adversely affect driving skills, and that these diagnoses alone are not absolute indicators of unsafe driving.

The American Medical Association’s Physician’s Guide to Assessing and Counseling Older drivers lists acute medical events such as myocardial infarction, stroke, syncope, seizure, surgery, and delirium as well as chronic conditions such as disease affecting vision, cardiovascular disease, neurological disorders, psychiatric illnesses, metabolic conditions, musculoskeletal impairments, and respiratory disease as risk factors for unsafe driving.<sup>6</sup> Detailing the

specific conditions that may be associated with unsafe driving is beyond the scope of this brief review. Interested readers are referred to chapter 2 of the AMA's guide.

### **OFFICE BASED ASSESSMENTS FOR DRIVING SAFETY**

In a sample of 460 primary care physicians in Canada approximately 72% indicated that physicians should be legally responsible for reporting unsafe drivers to state licensing authorities.<sup>7</sup> Strikingly, only 55% of the surveyed physicians believed they were most qualified to make this decision, and 88% felt that they would benefit from additional training in this area.<sup>7</sup> The AMA's guide recommends that physicians assess visual function (acuity and visual fields), cognition (Clock drawing test and Trails B, a visual motor task requiring participants to alternate between connecting numbers and letters) and motor function (20 foot walk and manual test of range of motion and motor strength), with cutoff scores for each measure.<sup>6</sup>

While office-based tests can assist clinicians in making recommendations about driving safety, some have argued that there is not enough research linking these measures to unsafe driving. For example, Molnar and colleagues performed a systematic review of research studies (1984-2005) examining the predictive utility of office-based screening measures for determining driving safety.<sup>8</sup> They found only sixteen articles were of high enough quality to include in the review and only one study provided cutoff scores for determining driving safety. The review indicated that Trails B was variably related to driving performance; and none of the studies examined clock-drawing performance. At the present time clinicians are expected to make recommendations, without empirical evidence to support office-based assessments.

Ott and colleagues examined the accuracy of physician assessments (based on chart review) for determining a professional driving instructor's rating of AD patients' standardized road test performance.<sup>9</sup> Physicians' accuracy ranged from 62% to 78%.<sup>9</sup> Clinicians also indicated which portions of the evaluation they relied upon for making their decision.<sup>9</sup> Raters with higher accuracy emphasized dementia duration, dementia severity (CDR and MMSE), neuropsychological measures of praxis, visuospatial ability, executive function, attention, history of

accidents and traffic violations, whereas less accurate raters emphasized dementia history, global neuropsychological performance, eye examination results, general medical history, and language skills.<sup>9</sup> Taken together, these findings suggest that driving safety is best not determined by performance on a single measure, but rather based on consideration of many patient characteristics. Importantly, compared to physical examinations or neuropsychological tests, a road test conducted by a professional driving instructor or certified occupational therapist appears to be the gold standard for determining driving safety.

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### **OLDER DRIVER RE-EDUCATION**

Changes in sensory, motor, or cognitive functioning do not always mean that the older patient should retire from driving. In some cases evaluation and training by an occupational therapist or private drivers' education program may help older adults become safe drivers again. Modifications can be made to vehicles to make them easier to use. For example, older adults with limited range of motion in their necks may benefit from parabolic mirrors. Drivers with limited motion in their arms may need a knob on their steering wheel.

The most common driving re-education program is the AARP Driver Safety Program, which is run as a classroom course and available on-line. AAA and state agencies also offer informational materials as well as classroom education. Research on the benefits of older driver re-education is very limited. Kua and colleagues performed a systematic review of the older drivers re-education literature and found that only eight studies demonstrated sufficient internal validity to be included.<sup>10</sup> Limited benefits of physical (range of motion exercises

or at home physical therapy exercises) and vision interventions (speed of information processing training or at home exercises to improve visual perception) were reported.<sup>10</sup> Educational programs were associated with some improvements in driving safety behaviors.<sup>10</sup> Unfortunately the two studies (self monitoring program and California's mature drivers program) that examined the benefit of driver education programs upon crash rates found no significant effect.<sup>10</sup>

The limited research should not completely dissuade clinicians from recommending these "refresher courses" to their older drivers. Driver education programs can help individual drivers, and some insurers provide discounts to older adults who participate in AARP's driver reeducation program. Until these programs are empirically validated, however, it is difficult to judge their relative benefit as well as which elements of the course are associated with the best remediation of driving skills.

### **CESSATION OF DRIVING**

The AMA Guide has compiled each states' reporting procedures for easy reference.<sup>6</sup> In Rhode Island "Any physician who diagnoses a physical or mental condition which, in the physician's judgment, will significantly impair the person's ability to safely operate a motor vehicle may voluntarily report the person's name and other information relevant to the condition to the medical advisory board within the Registry of Motor Vehicles. Any physician reporting in good faith and exercising due care shall have immunity from any liability, civil or criminal. No cause of action may be brought against any physician for not making a report."<sup>6</sup> Massachusetts is a self-reporting state. It is the responsibility of the driver to report to the Registry of Motor Vehicles any medical condition that may impair driving ability. However, physicians are encouraged to report unfit drivers to the Registry of Motor Vehicles. The law does not provide any protection from liability, nor does it promise confidentiality due to the "Public Records" law which states simply that a driver is entitled to any information upon receipt of written approval"<sup>6</sup> pg30-31.

Clinicians who recommend driving cessation to their patients should suggest alternative transportation. In Rhode Island patients can be referred to the Department of Elderly Affairs Pocket Manual of Elder Services,<sup>11</sup> which lists the different RIDE programs as

well as a phone number to schedule paratransit for individuals unable to use public transportation. Care providers will want to closely monitor these patients for signs of depression, self-neglect, and isolation as all can occur as a result of loss of driving privileges (see the AMA guide for specific monitoring methods).<sup>6</sup>

### FUTURE DIRECTIONS

There is much research to be done in the area of assessing older driver safety. As Molnar and colleagues point out, the establishment of empirically validated cut scores for visual functioning, motor skills, and cognition is necessary to help physicians confidently identify truly at risk drivers.<sup>8</sup>

A second priority is to establish clinically meaningful outcome measures. Certainly poor performance on a simulated driving test or pre-determined driving route is an indication for concern and monitoring, but there is not a perfect correlation between these measures and crash risk. In this regard, a new study examining risk factors for poor drivers in a naturalistic setting has begun at Rhode Island Hospital. In this study, funded by the National Institute of Health, older drivers both with and without dementia will be examined by video camera recordings in their own cars and driving in their neighborhoods. These recordings will be compared to performance on a standardized road

test and computerized office tests. For more information about participation or referrals, contact Lindsay Miller at 444-0789.

A third and final priority is to examine the benefits of driver education programs. If physicians are going to recommend these interventions, as with any other treatment, efficacy trials are necessary.

### REFERENCES

1. US Interim Projections by Age, Sex, Race, and Hispanic Origin. Table 2a. Projected Population of the United States, by Age and Sex: 2000 to 2050. <http://www.census.gov/ipc/www/usinterimproj/>
2. US Department of Transportation National Highway Traffic Safety Administration. Traffic Safety Facts 2000. <http://www-nrd.nhtsa.dot.gov/Pubs/2000OLDPO.PDF>
3. Duchek JM, Carr DB, et al. Longitudinal driving performance in early-stage dementia of the Alzheimer type. *J Am Geriatr Soc* 2003;51:1342-7.
4. Man-Son-Hing M, Marshall SC, et al. Systematic review of driving risk and the efficacy of compensatory strategies in persons with dementia. *J Am Geriatr Soc* 2007;55:878-84.
5. Amick MM, Grace J, Ott BR. Visual and cognitive predictors of driving safety in Parkinson's disease patients. *Arch Clin Neuropsychol* 2007;22:957-67.
6. The American Medical Association. Physician's Guide to Assessing and Counseling Older Drivers. <http://www.ama-assn.org/ama/pub/category/10791.html>
7. Jang RW, Man-Son-Hing M, et al. Family physicians' attitudes and practices regarding assessments of medical fitness to drive in older persons. *J Gen Intern Med* 2007;22:531-43.
8. Molnar FJ, Patel A, et al. Clinical utility of office-based cognitive predictors of fitness to drive in persons with dementia. *J Am Geriatr Soc* 2006;54:1809-24.

9. Ott BR, Anthony D, et al. Clinician assessment of the driving competence of patients with dementia. *J Am Geriatr Soc* 2005;53:829-33.
10. Kua A, Komer-Bitensky N, et al. Older driver retraining. *J Safety Res* 2007;38:81-90. Epub 2007 Feb 14.
11. The Rhode Island Department of Elderly Affairs. Pocket Manual of Elder Services. <http://www.dea.state.ri.us/documents/2007%20Pocket%20Manual.pdf>

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## Geriatric Neurorehabilitation In the New Millenium

*Stephen T. Mernoff, MD*

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**Rehabilitation interventions have changed** little in the last few decades, aside from improvements in materials and medical care. Most neurorehabilitation research involves geriatric patients since most disabling neurologic disease occurs in older populations. Proving rehabilitation intervention efficacy is difficult for methodological reasons.

New technologies and neuroscience advances allow us to foresee development of evidence-based neurorehabilitation interventions improving functional outcomes. The need for such interventions will increase as the population ages. Developing *patient-specific* rehabilitation programs using *selected tools* at *selected times* during recovery now seems within reach.

### REHABILITATION SETTINGS

Approximately 6-8% of Medicare patients admitted to acute care hospitals will

need inpatient rehabilitation. Medicare recipients comprise 75-80% of admissions to **acute rehabilitation facilities (ARFs)**. In 2007, the average age of patients admitted to ARFs in the US was 67 years. After an average stay of 16 days,<sup>1</sup> 73% of these patients returned home. With limited staffing and ill patients, acute care hospitals usually provide one or two brief therapy treatments a day. Immobilization for even only a few days causes deconditioning which takes longer to reverse than to develop; therapy should be initiated as soon as possible. **Long-term acute care (LTAC)** facilities manage patients with persistent intensive nursing and medical care needs. Acute rehabilitation hospitals admit 50-60% of their patients with neurologic diagnoses, generally providing the most intensive rehabilitation programs available for patients who can tolerate and

benefit from at least three hours of therapy per day. Subacute rehabilitation units provide programs for patients who cannot tolerate, or will not benefit from, more intensive therapy. Home therapy may often be suboptimal due to lack of equipment and inefficient scheduling. Outpatient therapy varies between one and five sessions a week for medically stable patients. Patients may move between these settings, depending on medical status and rehabilitation needs.

### ROLE OF PHYSICIANS IN REHABILITATION

Primary care physicians and/or physiatrists provide general medical management and help to prevent complications. Immobility increases risks of infection, deep venous thrombosis, and skin breakdown, which can usually be effectively prevented.



Inadequate sleep interferes with therapy, possibly contributing to cognitive impairments. Obesity and depression must be treated early and aggressively. Reassessment of medications is critical; patients often come to rehabilitation on medications they no longer need.

The role of the neurologist has expanded as patients survive acute neurologic disease at progressively higher rates and more is known about neurologic recovery. First and foremost is accurate diagnosis; some patients arrive to rehabilitation with incorrect diagnoses. Patients with traumatic brain injury must be monitored for hydrocephalus and subdural hematomas. The neurologist's role in educating therapists, patient, and family, by describing impairments, expected course of recovery, and prognosis, is critical to program design and realistic goal-setting.

## **EVOLUTION OF NEUROREHABILITATION AS A CLINICAL SPECIALTY<sup>2</sup>**

Rehabilitation Medicine as a medical subspecialty, the disciplines of Physical, Occupational, and Speech Therapies, and dedicated Rehabilitation Units developed in the early to mid 20<sup>th</sup> century as a result of war injuries (amputations) and polio. The "traditional" rehabilitation model was based on static impairments and compensation for, rather than restoration of, function. In the late 20<sup>th</sup> century, improved survival from acute neurologic injury and an emphasis on evidence-based practice resulted in improved outcome measures and a "neurologic" model of rehabilitation incorporating pathology and prognosis. Powerful technologies and greater understanding of plasticity present the possibility of enhancing natural recovery processes and evaluating these techniques. Clinicians will be able to develop the rehabilitation "holy grail": evidence-based patient-specific rehabilitation programs using selected techniques at selected times during recovery.

## **MECHANISMS OF RECOVERY AND THE CONCEPT OF NEUROPLASTICITY**

A brief summary of recovery mechanisms in stroke with a three-stage model is instructive. In the first stage, reduction of edema (causing mass effect and metabolic depression) over days to weeks improves the function of noninfarcted tissue as intercellular communication improves. The second and third stages of recovery are marked by spontaneous neurophysiologic adaptations referred to as neural plasticity. In the second stage, over weeks

to months, cells in the ischemic penumbra recover somewhat. These cells may have greater potential for synaptic plasticity via increased expression of genes for neurotrophins and angiogenesis.<sup>3</sup> The third stage is thought to consist of distant undamaged tissue taking on the functions of lost tissue. Several mechanisms may subserve neural plasticity including synaptogenesis, axonal regrowth, neurotrophins, and neurogenesis (stem cells).<sup>4</sup>

These mechanisms seem to be inhibited in more mature tissue. Enhancing natural mechanisms of plasticity may improve recovery. For example, antibodies to MAG promote axonal regrowth.<sup>5</sup> Although motor control is virtually fixed after adolescence, the reorganization occurring in damaged neurologic tissue may present an opportunity to intervene. Potential interventions, including neurotrophins, stem cells, and pharmacologic potentiation, are technically challenging and expensive. Elegant, noninvasive, and less expensive methods that may enhance neural plasticity are being investigated. Different approaches will likely be applicable in different situations.

## **NEW NEUROREHABILITATION TECHNOLOGIES UNDER INVESTIGATION**

Neurorehabilitation research is coming of age. Improvements in outcome measures and study design allow new techniques to be investigated in the context of randomized, controlled, often multi-centered trials (RCTs). The EXCITE trial, a study of Constraint-Induced Movement Therapy, demonstrates that multicenter RCTs for rehabilitation interventions are feasible, and is serving as a model for design of future studies.<sup>6</sup>

The Center for Restorative and Regenerative Medicine (a collaboration of Brown University and the Providence VA Medical Center led by Roy Aaron, MD, and John Donoghue, PhD), in collaboration with MIT and Harvard, is becoming a major center for such research. Other local entities, including Cyberkinetics, Inc. (Foxboro, MA), Afferent Corporation (Providence, RI), and Rehabilitation Hospital of Rhode Island (RHRI, North Smithfield), are at the forefront of these efforts. Some of these investigational interventions are briefly described below.

### **"LOW TECH" INTERVENTIONS**

Constraint-Induced Movement Therapy (CIMT) is based on the concept of "learned nonuse" in monkeys, a behavioral suppression in which lack of success with use

of an impaired arm leads to preferential use of the unaffected arm.<sup>7</sup> CIMT is a "forced use" paradigm. The use of the unaffected arm is limited (by a sling or mitt) and the affected arm/hand undergoes intense therapy. Multiple animal and human studies, including the landmark EXCITE trial, have demonstrated the technique to be quite effective in certain populations. There is some evidence that measurable cortical reorganization results. CIMT has been investigated mainly in patients with stroke and cerebral palsy, but is also being applied to lower limb impairment, traumatic brain injury, and even aphasia.

## **STROKE INPATIENT REHABILITATION REINFORCEMENT OF WALKING SPEED (SIRROWS)<sup>8</sup>**

Gait speed may be a surrogate marker for gait quality, often the major limiting factor for home discharge. Faster gait improvement in the inpatient rehabilitation setting might result in more efficient recovery with shorter lengths of stay. The World Federation of Neurorehabilitation (WFNR), in collaboration with the American Society of Neurorehabilitation (ASNR), is running the SIRROWS trial to determine if this is true. This elegant and simple RCT involves giving patients daily feedback about their walking speed, with encouragement to walk faster, safely. SIRROWS is the first attempt to develop an internationally controlled multicenter trial in neurorehabilitation. Twenty centers, mostly outside of the US, are involved. RHRI will soon become a SIRROWS study site.

## **"MEDIUM TECH" INTERVENTIONS Sensory Enhancement**

Afferent Corporation (Providence, RI) has several devices under development. Afferent's technology involves applying subthreshold mechanosensory stimuli to affected limbs to enhance afferent information flow to the brain. The premise is that sensory impairment contributes to functional impairment after nervous system injury. Improving sensory function could improve recovery by enhancing plasticity. Studies show the system to be effective in sensory-mediated gait disorders.<sup>9</sup> A pilot study, using the technology in patients with arm weakness after stroke, is being conducted at Spaulding Rehabilitation Hospital in Boston.

## **"HIGH TECH" INTERVENTIONS Robotics**

Various robotic devices are being studied for use in rehabilitation settings. The MIT-

MANUS robots, under development over the last 15 years at the MIT biomedical engineering department, are devices designed to provide consistent doses of intense limb exercise. They provide decreasing levels of assistance as the patient's own abilities improve. The devices also provide kinematic data that measure effectiveness of the technique and give insight into natural recovery. Initial studies are encouraging, and demonstrate improved motor function in involved limbs which appears to be sustained for years after a therapeutic trial of 4-8 weeks.<sup>10</sup> A VA multicenter trial (supervised by Albert Lo, MD, PhD, of the Providence VA) utilizing various versions of MIT-MANUS robots for stroke patients is currently underway.

### Treadmill Devices

Several devices combining a treadmill with partial-weight-bearing-support have been developed over the last 10 years. The **Lokomat** (Hocoma, Inc., Switzerland) device has the added feature of a lower body exoskeleton providing partial assistance for stepping, and has shown benefit in patients with spinal cord injury. Studies of benefit to patients with stroke have been mixed. Results of the first study of its use in patients with multiple sclerosis (Albert Lo, MD, PhD) should be available shortly. The **Autoambulator**, a similar device developed for HealthSouth, is also being studied.

### Cortical Stimulation

Brain stimulation might enhance neural plasticity. Noninvasive devices (including **Transcranial Magnetic Stimulation** and **DC Current Stimulation**) designed to stimulate the cerebral cortex during therapy are under investigation. A multicenter controlled trial is currently underway to determine if an epidurally implanted electrode (Northstar Neuroscience), providing subthreshold stimulation during physical therapy, enhances recovery (Spaulding Rehabilitation Hospital in Boston is a participant).

### Brain-Computer Interfaces

Patients with severe neurologic disabilities usually have some residual muscle function (finger, eyelid, or eye movements) allowing at least rudimentary communication. Some patients (e.g., ALS, brainstem stroke) are completely paralyzed despite having normal or only mildly impaired cognition ("locked-in"), resulting in inability to communicate or influence the environment. **Brain-computer interfaces (BCI)** are devices

that detect and decode brain signals, allowing control of external devices. The **Braingate** (Cyberkinetics, Inc, Foxboro, MA) device, developed in John Donoghue's laboratory at Brown University, is an electrode array implanted on the cerebral cortex. A computer decodes detected signals to determine the individual's intended movements. A pilot trial with human subjects has demonstrated that patients can operate cursors on computer screens with their thoughts,<sup>11,12</sup> allowing control of external devices such as televisions and robotic arms.

A less invasive device, the **Wadsworth BCI Home System** (Laboratory of Nervous System Disorders, The Wadsworth Center, NY State Dept of Health), uses surface electrodes and software to detect and compile EEG signals which patients learn to modulate to select characters on a computer screen. This system has enabled some patients with ALS and other severely disabling disorders to communicate and send email. A multicenter trial is being planned to better assess the effectiveness and efficacy of this system with ALS patients on a larger scale.

### CONCLUSIONS

Plasticity exists particularly in the post-injury period, even in the older population. Many possible new treatments are being developed. Particular epochs during the postinjury period may be windows of opportunity for intervention. Optimization of therapies for different types of patients is a major challenge. Ongoing research will determine which treatments should be done for which patients, and when. New models of recovery are under development to enable us to capitalize on advances in neuroscience and technology to improve rehabilitation outcomes.

Several landmark studies (recent, ongoing, and upcoming) are demonstrating that neurorehabilitation research is no longer in the back reaches of anecdotal evidence and unprovable theories. Both low-tech but elegant interventions and sophisticated technologies clearly have roles in improving the functioning of patients with both mild and severe neurologic disabilities, by enhancing the nervous system's natural plasticity. This goal is a huge challenge, particularly in the geriatric population. We now have techniques to perform valid and reliable studies of these interventions. Neurorehabilitation is worthy of being considered a medical subspecialty subject to the standards of evidence-based medicine.

### REFERENCES

1. eRehabdata.com and Bazemore, Lisa (eRehab) personal communication 10/17/07.
2. Dobkin BH. *The Clinical Science of Neurologic Rehabilitation*, 2nd edn. Oxford University Press, 2003
3. Wei L, et al. Collateral growth and angiogenesis around cortical stroke. *Stroke* 2001; 32:2179-84.
4. Kempermann, et al. *J Neurosci* 2002;22:635-8.
5. Lehmann M, et al. Inactivation of Rho signaling pathway promotes CNS axon regeneration. *J Neurosci* 1999; 19:7537-47.
6. Wolf SL, et al. Effect of CIMT on upper extremity function 3-9 months after stroke. *JAMA* 2006; 296:2095-104.
7. Taub E, et al. Technique to improve chronic motor deficit after stroke. *Arch PM&R* 1993; 74:347-54.
8. Latham N, et al. Physical therapy during stroke rehabilitation for people with different walking abilities. *Arch PM&R* 2005; 86:41-50.
9. Priplata AA, et al. Noise-enhanced balance control in patients with diabetes and patients with stroke. *Ann Neurol* 2006;59:4-12.
10. Volpe B, et al. Is robot-aided sensorimotor training in stroke rehabilitation a realistic option? *Curr Op Neurol* 2001;14:745-52.
11. Serruya MD, Hatsopoulos NG, et al. Instant neural control of a movement signal. *Nature* 2002;14;416:141-2.
12. Hochberg LR, Serruya MD, et al. Neuronal ensemble control of prosthetic devices by a human with tetraplegia. *Nature* 2006; 442: 164-71.

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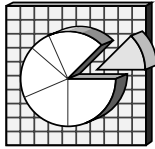
### Disclosure of Financial Interests

The author has no financial interests to disclose.

Discussion(s) of off-label usage of any products or services: Several devices described in the article are investigational but are approved by the FDA for investigational use: devices by Afferent Corporation, MIT-MANUS robot, Locomat, Autoambulator, Northstart device, Braingate (Cyberkinetics, Inc.), and Wadsworth BCI Home System.

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## The Value Equation: Costs and Quality of Rhode Island's Health Plans

Bruce Cryan, MBA, MS

Two health plans, Blue Cross and Blue Shield of Rhode Island (Blue Cross), and United Healthcare of New England (United), provide health coverage to a large majority of Rhode Island (RI) residents who are commercially insured. To assess whether the purchasers of these plans' products are receiving value, one must necessarily examine its two components, cost and quality. For Rhode Islanders to receive good returns from their expenditures for health insurance, that coverage should be equally or less expensive and deliver the same or better quality services than similar plans elsewhere. Information about how these two plans perform is therefore essential to evaluating their relative value.

In response to this need for information, the RI General Assembly passed the Health Care Accessibility and Quality Assurance Act in 1996 (Rhode Island General Laws 23-17.13).<sup>1</sup> The Act instituted health plan performance reporting in the state, which is summarized annually, and most recently in the *Rhode Island Health Plans' Performance Report (2006)*.<sup>2</sup> The information presented here is derived from that report.

### METHODS

The Rhode Island Department of Health's Center for Health Data and Analysis conducts an annual health plan data collection from three primary audited sources: Statutory Filings to the state's Department of Business Regulation and Health Plan Employer Data and Information Set (HEDIS) reports and Consumer Assessment of Healthcare Providers and Systems (CAHPS) survey data submitted to the Department of Health.<sup>3</sup>

From these data, 32 measures are evaluated, comprising eight separate dimensions of performance (enrollment, costs, utilization, prevention, screen-

ing, treatment, access, and satisfaction). For definitions of these measures, see the source report.<sup>2</sup> To ascertain relative performance, the measures for each plan are compared to the average of all commercial health plans in New England.<sup>3</sup>

### RESULTS

Of Rhode Island's 342,000 commercially insured population, most are covered by two carriers, Blue Cross, with a 2006 market share of 64.8%, and United, with a share of 14.6%. The remainder of the market (20.6%) consists of a

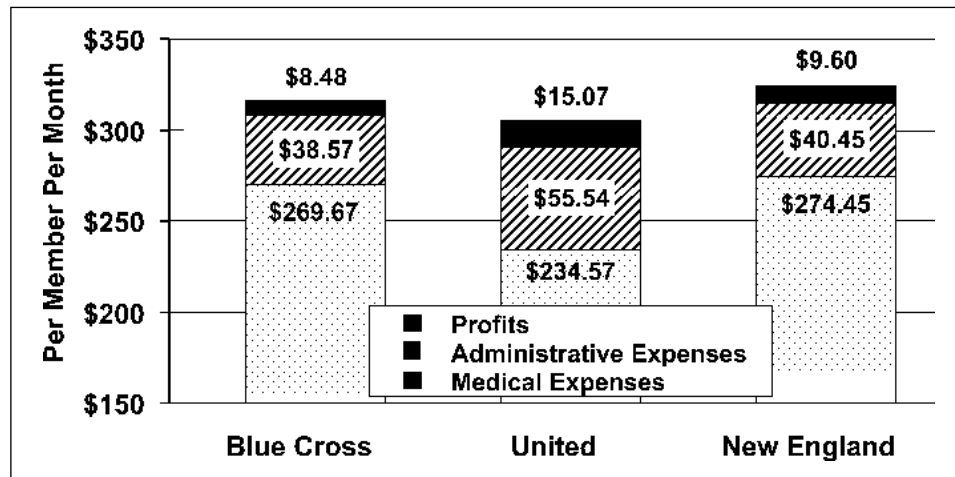


Figure 1. Average health plan premium per member per month, by component, Blue Cross of Rhode Island, United Healthcare of New England, and New England aggregate, 2006.

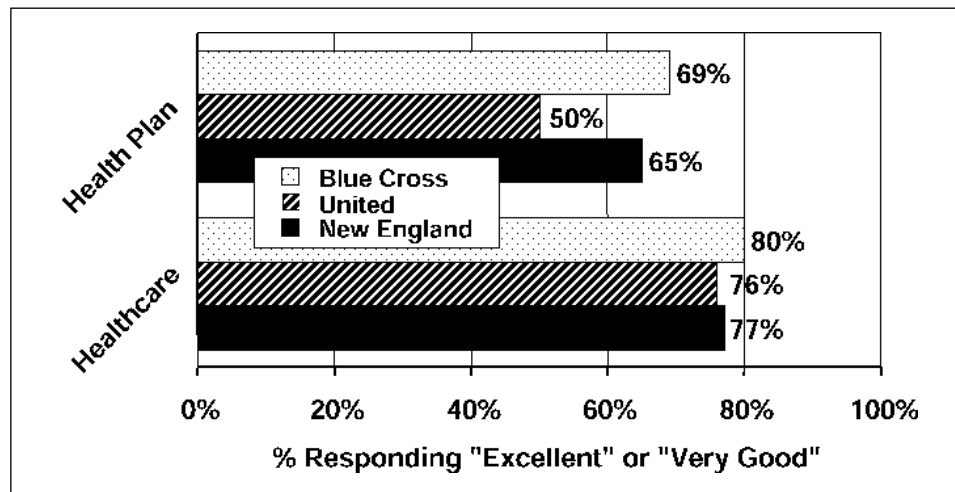


Figure 2. Health plan member satisfaction with health plan and health care, Blue Cross of Rhode Island, United Healthcare of New England, and New England average, 2006.

**Table 1.**  
**Health Plan Clinical Performance Measures, Blue Cross of Rhode Island,**  
**United Healthcare of New England, and New England Average, 2006**

Dimension/Measure	New England Average	Relative to New England Average	
		Blue Cross	United
<b>Prevention</b>			
Childhood Immunization	82.3%	=	=
Adolescent Immunization	80.5%	-13%	-12%
Smokers Advised to Quit	78.5%	=	+7%
Smokers Advised on Medications	52.4%	-5%	-5%
Smokers Advised on Methods	52.0%	=	+15%
<b>Screening</b>			
Colorectal Cancer Screening	64.9%	=	-8%
Breast Cancer Screening	78.8%	=	=
Cervical Cancer Screening	85.7%	=	=
Chlamydia Screening	44.4%	-11%	-12%
Diabetes: Eye Exam Screening	68.7%	-7%	-8%
Diabetes: HbA1c Tested	91.5%	=	=
<b>Treatment</b>			
Beta Blocker Treatment	99.3%	=	=
Cholesterol Controlled	62.4%	-5%	-14%
Appropriate Asthma Medications	91.6%	=	=
Antidepressant Medication Management	27.4%	-7%	-12%
<b>Access</b>			
Follow-up for Mental Illness	85.2%	-7%	=
Prenatal Care	96.1%	=	-12%
Postpartum Care	85.5%	+5%	-15%
Well Child Visits	84.0%	=	=
Adolescent Well-Care Visits	60.0%	=	=

Note: "=" indicates that the relative difference from the New England average is less than +/- 5%.

number of smaller plans, none of which are domiciled in Rhode Island. [Note that these data include only the insurers' "fully-insured" members and exclude members of plans where the purchaser (employer) is self-insured.]

On average in 2006, commercial health insurance cost slightly less in RI than in New England. (Figure 1) Blue Cross' monthly premiums were 2% lower than regional premiums (\$317 vs. \$325), and United's premiums were 6% lower (\$305 vs. \$325). In addition, both RI plans spent relatively less on medical services for their members (2% less for Blue Cross and 15% less for United).

With few exceptions, both Blue Cross and United generally performed at or below average when their clinical quality measures were compared to the New England values. (Table 1) For Blue Cross, 12 of its 20 quality measures were equivalent to the regional averages, one measure was better, and the remaining seven were worse than these comparables. For United, nine of its 20 quality measures were equivalent to the regional averages, two measures were better, and the remaining nine were worse than these comparables. Given that New England health plans consistently post some of the highest quality (and satisfaction) scores in the country, this regional comparison provides a rigorous benchmark for RI plans.

In addition to an individual plan's relative performance on the clinical measures, the absolute values on some of the clinical

measures are worth examination. For example, the low rates of chlamydia screening (Blue Cross: 40%; United: 39%) and antidepressant medication management (Blue Cross: 26%; United: 24%) highlight the need for further improvement in these areas.

Proxy measures of whether members perceive value in their plans may be obtained from member satisfaction surveys. Member satisfaction with Blue Cross' performance as a health plan was 4 percentage points higher than the regional rate in 2006 (69% versus 65%), while member satisfaction with United was 15 percentage points below that comparable (50% versus 65%). (Figure 2) There was little difference in members' satisfaction with their healthcare services between the two plans and in comparison with the New England rate. Interestingly, regardless of geographic area or health insurer, more members were satisfied with their healthcare services than with their health plans.

## DISCUSSION

Increasingly, the public, purchasers, providers, and policymakers are requiring meaningful information about health plans. Since 1998, the Department of Health has tracked the performance of this industry and produced annual reports on the subject.<sup>2</sup>

With the small number of health plans in the state and the market dominance of Blue Cross, most Rhode Islanders have limited

choice of carrier. The lack of widespread selective contracting also means that most plans deliver services through a similar network of physicians, hospitals, and other providers, and the lack of differentiation between the two plans in their healthcare satisfaction rates bears this out.

Therefore, the real value in publishing this information is less in aiding consumer choice and more in fostering accountability of the industry. Purchasers deserve to know how well the plans are performing and policymakers need empirical evidence to inform their efforts. An added benefit is that the performance of health plans will likely improve if for no other reason than the results are made public.

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## Disclosure of Financial Interests

The author has no financial interests to disclose.

## REFERENCES

1. See <http://www.rilin.state.ri.us/Statutes/TITLE23/23-17.13/INDEX.HTM>.
2. Cryan B. *Rhode Island Health Plans' Performance Report 2006*. Providence RI: Rhode Island Department of Health and Office of the Health Insurance Commissioner. In press.
3. For information on the HEDIS and CAHPS programs, see the website of the National Committee for Quality assurance (NCQA) - <http://www.ncqa.org>.



## Rhode Island Leads In Regulating Office-Based Cosmetic Surgery

Shaun Najarian

**A recent decision by the Rhode Island Director of Health** to close an unlicensed ambulatory surgery center and to suspend the medical license of the owner underscores the need for the appropriate regulation of cosmetic surgery.

In September 2007, the **Rhode Island Department of Health (HEALTH)** closed an ambulatory surgery center owned and operated by a physician licensed in the State of Rhode Island after the Rhode Island Board of Medical Licensure and Discipline (“the Board”) concluded that the owner/operator had performed a variety of cosmetic surgeries in an unlicensed outpatient facility. As well, the Board concluded that the physician had allowed unqualified employees to deliver anesthesia to patients at the facility.

Public Health is invested in the accreditation, licensure, and regulation of physicians, surgeons and their facilities. Many states have considered legislation regarding the licensure and regulation of office-based surgery, but few have followed through. At present, only six states have statutes of this type in place: Rhode Island, California, Florida, New Jersey, Pennsylvania, and Texas.<sup>1</sup> In Rhode Island, legislation was passed in 1999 requiring physicians to be specially licensed by HEALTH in order to perform office-based surgery, and in 2001, the law was amended, requiring that the facilities be accredited for Level II and Level III anesthesia (intravenous and general) within 24 months of licensure.<sup>2</sup> To ensure the highest level of patient safety, all states should consider similar legislation.

Under Rhode Island law, as specified by “Rules and Regulations for the Licensure of Physician Ambulatory Surgery Centers and Podiatry Ambulatory Surgery Centers (R23-17-PASC),” surgery is defined as follows:

1.21 “Surgery” means the excision or resection partial/complete, destruction, incision or other structural alteration of human tissue by any means. Surgery shall have the same meaning as “operate.”

Certain procedures are exempt from R23-17-PASC:

1.8 “Exempt procedures” means: a) Minor surgical procedures such as excision of skin lesions, moles, warts, lipomas and repair of lacerations, incision and drainage of superficial abscesses, or surgery limited to the skin and subcutaneous tissue performed under topical or local anesthesia not involving drug induced alteration of consciousness other than minimal pre-operative tranquilization of the patient; b) Procedures not requiring or using conscious sedation techniques or pre-operative medications other than minimal pre-operative tranquilization of the patient; c) Procedures requiring or using only local, topical, or no anesthesia.”

All medical laser procedures fall under the aegis of the Rhode Island statute, including ablative and non-ablative skin rejuvenation procedures, tattoo removals, treatment of pigmented and vascular lesions, and laser hair removal. The reason for regulating *all* medical laser procedures, including laser hair removal, is the sheer variety (and changing variety) of laser devices – including a wide

range of output – and the potential for lasers to do irreparable harm to a patient if used incorrectly. Thus, in Rhode Island, individuals who wish to perform any of these cosmetic laser *surgeries* in an office setting must apply for a special license.

In December 2003, the Rhode Island Board of Medical Licensure and Discipline strengthened its regulatory position on the use of medical lasers with the following decision:

12/15/2003 - Policy statement on office based esthetic procedures:

It is the position of the Board that office based cosmetic or esthetic procedures that require the use of medical lasers, high-frequency radio waves, or injection of sclerosing chemicals or biologically active compounds [e.g. Botulinum toxin A, Botox] are medical procedures.

Therefore, prior to undergoing such procedures patients must receive a medical evaluation for appropriateness by a licensed and qualified physician or other practitioner acting within his/her scope of practice. Although these procedures may be performed by an appropriately trained nonphysician working under the supervision and direction of a physician or other practitioner acting within his/her scope of practice, it is the supervising physician’s [or other practitioner acting within his/her scope of practice] responsibility to assure that procedures are conducted appropriately; with appropriate assessment, consent and follow-up; and upon appropriate patients; and that all patient records are maintained according to standards applicable for medical records; and that patient privacy is protected. The supervising physician or other practitioner acting within his/her scope of practice is responsible for any procedures carried out by nonphysicians under his/her direction.

Physicians [or other practitioner acting within his/her scope of practice] who perform and supervise such procedures must be able to demonstrate appropriate training and experience. Such training and experience may include, but is not limited to, residency or fellowship. The physician or other practitioner acting within his/her scope of practice is responsible to assure and document adequate training for individuals under his/her supervision.

Additionally, other cosmetic procedures such as dermabrasion or the application of potentially scarring chemical treatments [e.g. so-called chemical peels] should also meet this same standard.

[Rhode Island Board of Medical Licensure and Discipline. “Policy statement on office based esthetic procedures.” December 15, 2003. <http://www.health.ri.gov/hsr/bmld/positions.php>]

Despite increased vigilance such as this, office-based laser hair removal remains especially troublesome to regulate throughout the United States. Many unlicensed facilities perform the work, unaware of potential dangers and of existing safety regulations, e.g., those that limit maximum laser exposure or require eye protection. At least one death in the United States has been attributed to an overdose of anesthetic cream before laser hair removal.<sup>3</sup>

Several national organizations seek to improve the quality of office-based surgical practices:

- The American Association for Accreditation of Ambulatory Surgery Facilities (AAAASF)
- The Accreditation Association for Ambulatory Health Care (AAAHC)
- The Joint Commission for Accreditation of Healthcare Organizations (JCAHO)

The JCAHO recently released safety goals for office-based surgeries.

Despite the best efforts of HEALTH, some health professionals may continue to perform cosmetic surgical procedures in unlicensed facilities. It is important for healthcare providers throughout the state to be vigilant for this possibility. At present, in addition to licensed hospitals and licensed freestanding surgical centers, only five "office operatories" are licensed in Rhode Island. Those who wish to verify the license status of a facility or

of a service provider may consult HEALTH's website: <http://www.health.ri.gov/hsr/professions/license.php>.

## REFERENCES

1. Lapetina EM, Armstrong EM. Preventing errors in the outpatient setting. *Health Affairs* 2002; 21: 28.
2. Springer R. An update on office surgical regulations. *Plastic Surgical Nurs* 2004;24: 172.
3. On February 18, 2005, National Public Radio's (NPR's), Adam Hochberg discussed the dangers of an unregulated laser industry on his show "All Things Considered." Hochberg reported on the case of a North Carolina woman who died from an overdose of anesthetic cream that she applied before her laser treatment in January.

*Shaun Najarian is an undergraduate at Providence College who served as an intern for the Rhode Island Department of Health, Office of Medical Licensure and Discipline.*

## Disclosure of Financial Interests

The author has no financial interests to disclose.

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# Point of View

## Creative and Sensory Therapies Enhance the Lives of People With Alzheimers

*John Stoukides, MD*

Today physicians have access to an enormous body of research and knowledge regarding the pathophysiology of Alzheimer's disease. Pharmacologic treatment options have also increased significantly over the past few years. However, pharmacologic treatment is only one part of the overall care plan for improving the quality of life of patients suffering from Alzheimer's and related dementias.

Long-term care facilities and adult day centers throughout Rhode Island use a variety of creative and sensory therapies to enhance the lives of people with Alzheimer's including aromatherapy and massage; painting, pottery, sculpting; music and dance; exercise and cooking.

The "Memories Fade... Love Inspires" art exhibit, recently held at the Bellini Ruggeri Gallery in Providence, showcased a collection of watercolor paintings and pottery pieces created by participants at the Hope Alzheimer's Center. The cover of this journal features the beautiful work of Theresa Aiello, a long time participant in the therapeutic art program at the Hope Center. Although Theresa, age 91, has had no formal art training, she is able to express herself through vivid color

Since opening its doors in 1995, art therapy has been a centerpiece of the adult day program at the Hope Center. Through the years, the Center has reported seeing many withdrawn and quiet participants begin to engage in lively conversations about a special place or time in their life reflected in the painting.

Research conducted in the field of Alzheimer's shows clear evidence that art therapy is a powerful, nonmedical way to engage minds in the grip of this disease. For people with memory loss, creative and sensory activities can help:

Promote well being	Help maintain skills
Aid communication by using sensory rather than cognitive pathways	Enhance relationships
Facilitate decision making	Utilize past skills
Combat depression	Express emotion
	Encourage cooperation with others

Although treatment is not available today that can delay or stop the deterioration of brain cells in Alzheimer's disease, studies have consistently shown that active medical management of Alzheimer's and other dementias can significantly improve quality of life through all stages of the disease for diagnosed individuals and their caregivers. Active medical management includes the integration of support services like adult day services that offer creative and sensory therapies into the overall treatment plan.

Most of the participants in the Hope Center's art therapy program live at home, where they are cared for by family and friends. Even when care is provided at home, most families also seek other sources of help, particular as the disease progresses. As a trusted information source for family caregivers, physicians can discuss the benefits of choosing an adult day program or long-term care provider that offers creative and sensory therapies for people with Alzheimer's and other forms of memory loss.

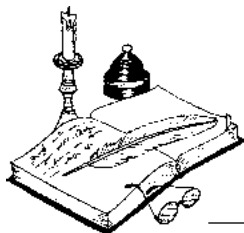
*John Stoukides, MD, is Medical Director of the Rhode Island Mood & Memory Research Institute and Director of Geriatric Medicine at Roger Williams Medical Center.*

## Disclosure of Financial Interests

The author has no financial interests to disclose.

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# Physician's Lexicon

## The Prefixes of the Past

**The prefix, particularly when employed** for the technical terms such as those which serve medicine, informs us about the root of the noun: its size [eg, *macro-*, *micro-*], its spatial relationship [eg, *juxta-*, *infra-*, *supra-*], as well as an array of attributes to define for us more precisely the modified meaning of the root word. A special group of prefixes, either Latin or Greek, also tells us whether or not the noun is old, ancient, backwards or primitive. These prefixes include the following: *ante-*, *paleo-*, *palin-*, *pre-*, *presby-*, *pretero-* and *proto-*.

*Ante-*, Latin signifying before or prior to, is seen in words such as antecubital and antenatal [but not antigen or antacid.] *Paleo-*, from the Greek meaning primitive or ancient, appears in such technical terms as paleopathology, paleontology, paleo-striatum [a synonym for the globus

pallidus] and Paleocene [the geologic epoch preceding the Eocene.] *Palin-*, in Greek meaning backwards, appears in words such as palindrome [a word or phrase reading the same backward or forward], palingenesis [meaning regeneration or rebirth] and palinopsia [a recurrent visual disorder.]

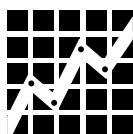
The *pal-* prefix such as in palinopsia, however, can be confusing. Palisade, for example, is derived from the Latin word, *palus*, meaning a stake. Palliation, meaning to extenuate, to cloak [and, specifically, to lessen pain] comes from the Latin, *pallium*, meaning a cloak. Pallid, meaning ashen or pale, is from the Latin, *pallidus*, meaning pale or colorless [as in globus pallidus.] Paludism, an archaic name for malaria, is from the Latin, *palus*, meaning marsh or swamp. And pallesthesia, mean-

ing the appreciation of vibration, is from the Greek, *pallo*, meaning to quiver.

The Greek prefix, *presby-*, meaning venerable or ancient, is seen in words such as presbyopia, presbycusis, an older euphemism for deafness, and Presbyterian. The Latin prefix, *pretero-*, meaning coming before or beyond, appears in words such as preternatural [a synonym for supernatural] and preterition.

Medical terms beginning with the Latin prefixes *pro-* and *pre-* are too numerous to list. Precocity, for example, is from the Latin *praecox*, meaning to ripen early, thus explaining the phrase, dementia praecox, an older term for schizophrenia. Precipitation is from the Latin, *praecipitare*, meaning to cast down headlong, and ultimately derives from *capitus*, meaning head.

— STANLEY M. ARONSON, MD



RHODE ISLAND DEPARTMENT OF HEALTH  
DAVID GIFFORD, MD, MPH  
DIRECTOR OF HEALTH

## VITAL STATISTICS

EDITED BY COLLEEN FONTANA, STATE REGISTRAR

### Rhode Island Monthly Vital Statistics Report Provisional Occurrence Data from the Division of Vital Records

Underlying Cause of Death	Reporting Period			
	May 2007	12 Months Ending with May 2007		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	223	2,730	255.2	3,601.5
Malignant Neoplasms	201	2,261	211.4	6,077.5
Cerebrovascular Diseases	36	388	36.3	562.0
Injuries (Accidents/Suicide/Homicide)	43	565	52.8	9,003.0
COPD	50	434	40.6	392.5

Vital Events	Reporting Period		
	November 2007	12 Months Ending with November 2007	
	Number	Number	Rates
Live Births	942	13,521	12.7*
Deaths	826	9,943	9.3*
Infant Deaths	(9)	(108)	8.0#
Neonatal Deaths	(6)	(74)	5.5#
Marriages	394	6,879	6.4*
Divorces	187	3,026	2.8*
Induced Terminations	386	4,919	363.8#
Spontaneous Fetal Deaths	49	994	73.5#
Under 20 weeks gestation	(45)	(920)	68.0#
20+ weeks gestation	(4)	(74)	5.5#

(a) Cause of death statistics were derived from the underlying cause of death reported by physicians on death certificates.

(b) Rates per 100,000 estimated population of 1,067,610

(c) Years of Potential Life Lost (YPLL)

*Note: Totals represent vital events which occurred in Rhode Island for the reporting periods listed above. Monthly provisional totals should be analyzed with caution because the numbers may be small and subject to seasonal variation.*

\* Rates per 1,000 estimated population

# Rates per 1,000 live births

# THE RHODE ISLAND MEDICAL JOURNAL

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## NINETY YEARS AGO, MAY, 1918

Frank J. McCabe, MD, in "Eye Strain as Related to General Practice," discussed types of asthenopia: accommodative, "due to errors of refraction and to strain of the...muscle;" muscular, "due to an abnormal condition of the...muscles of the eye;"; nervous, "due to some faulty condition of the nervous system;" and reflex, "due to abnormalities in the organism outside of the eye and the nervous system." Accommodative strains were the most common.

Frederick V. Hussey, MD, in "A Review of 100 Consecutive Cases of Acute Diseases of the Appendix, Gall Bladder, Duodenal and Gastric Ulcers Which Have Come to Operation," faulted general physicians' "lack of courage - reluctance to advise radical measures in the beginning, without first trying out some of the older methods of treatment, which brings his patient into an extreme condition." The author conceded that often patients wanted to avoid surgery. Of 46 cases of acute gangrenous appendicitis, all drained, three died; the average hospital length of stay for those patients was 3 weeks.

In a War Department memo, the Office of the Surgeon General, Washington, reminded physicians of the rule forbidding publication of professional papers related to official records or military service, without permission.

An editorial, "Illegal Operations," described the case of a woman recently convicted of an "illegal operation." "The woman had enjoyed a lucrative clientele for some time, pursuing her work in a respectable residential section of Providence." The editor assumed that this was the first case of a woman convicted in Rhode Island for the "crime of performing abortions, and, furthermore, ... the conviction did not depend upon the death of the unfortunate patient." The editorial congratulated both the police and the Attorney General. "...decent public opinion cannot fail to approve of the outcome."

## Medical Office Building

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(401) 821-6800

## FIFTY YEARS AGO, MAY 1958

Eugene T. Lothgren, MBA, general agent, Northwestern Mutual Life Insurance Company, contributed "Tax Savings in Estate Planning for Physicians." He urged: "action is essential to security and peace of mind."

The Division of Vital Statistics of the Rhode Island Department of Health contributed "Acute Poliomyelitis in Rhode Island, 1948-57." Many Americans, including Rhode Islanders, were not yet vaccinated. "Unless there is a speedup in the polio vaccination program it is the opinion of the Surgeon General, Leroy E. Burney, that serious outbreaks of poliomyelitis could occur this summer." The State traced wide fluctuations in incidence: in 1948, 8 cases; in 1949, 157 cases; in 1950, 55 cases; in 1951, 15 cases. The toll rose to 295 in 1953, dropped to 123 in 1954, peaked at 421 in 1955.

Robert W. Hyde, MD, in "Butler Health Center Today," described the 1957 reorganization of Butler Hospital into a Health Center, with outpatient and day services.

David S. Liang, MD, and Asdrubal De Carvalho, MD, in "Leiomyoma of the Prostate," described an 87 year-old man, admitted to Joseph's with "painful gross hematuria for 2 days." Surgeons found and removed an intravesical tumor.

Bencil L. Schiff, MD, in "Dermatitis from Acetazolamide (Diamox)," discussed the case of a 45 year-old woman who had been given 250 mg daily for treatment of glaucoma, and the case of a 57 year-old man, treated for hypertension and left ventricular failure.

## TWENTY-FIVE YEARS AGO, MAY 1983

Carl H. Critz, MD, W. Martin DeLuca, PA, and Arun K. Singh, MD, in "Iatrogenic Extra-Corporeal Hemolysis during Cardiac Surgery in a Child: A Case Report," postulated "shear-stress in transfusion filter as the cause of the hemolysis." A four year-old girl had been admitted to Rhode Island Hospital for surgical correction of both an atrial septal defect and pulmonary valvular stenosis.

Frank Newman, PhD, President, The University of Rhode Island, delivered the 1982 Fiorindo A. Simeone oration at The Miriam Hospital: "Notes from Underground." His subject was the anatomy of recovery. He detailed his experience of hospitalization, including "coping with fear," and "role of the self in healing."

A.J. Migliaccio, MD, FACS, and A.V. Migliaccio, MD, FACS, contributed "The Use of a Feeding Gastrostomy as a Means of Preventing Staple Line Disruption in Gastric Operations for Morbid Obesity."



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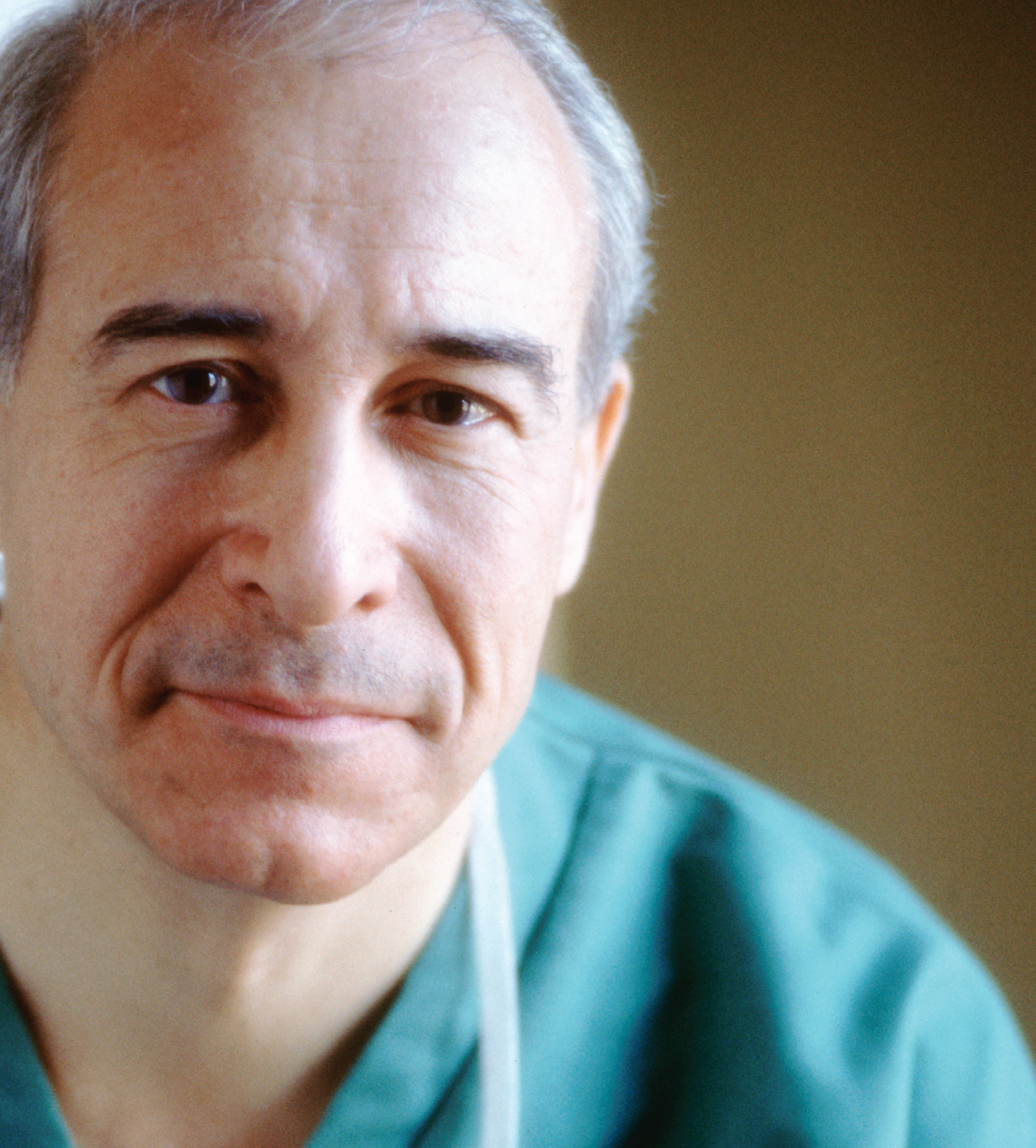
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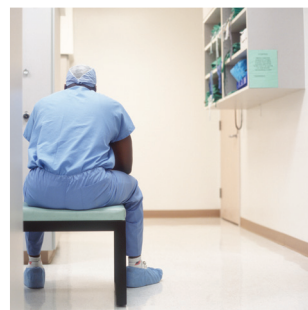
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