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Medicine & Health
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The Aging Brain
Part II

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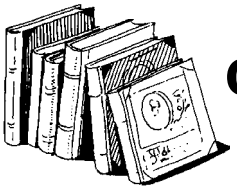


Commentaries

What Kind of Doctor Am I?

Like every other doctor, I often reflect on how I practice medicine. Like every other doctor, I like to think of myself as being above average. There is no "perfect" doctor. No one can be the ideal doctor for every patient. Some patients want an authority figure to lead them. Some want a sympathetic shoulder to lean on. Some want a doctor who is the leading expert in the field, and others want someone "nice" who is competent and happy to coordinate care among disparate experts. We know that we should always show respect and concern for our patients, no matter what their problems are or what their demeanor. Some of us are warm and sensitive types and some of us are more formal, but we all think that we project a sense of caring, with some measure of professional detachment; and, of course, some scientific understanding of the relevant medical issues and how to treat them.

A colleague of mine at another university introduced me before my lecture (on the use of antipsychotic drugs in Parkinson's disease) as an "atypical" neurologist, playing on the words, "atypical neuroleptic" used to label the "second generation" of antipsychotic drugs. The same colleague had introduced me once before, in a similar setting, as a "creative" neurologist. At the time, with the vocal inflections being what they were, I was unsure if these were generous words of acclaim, or, perhaps snide words of criticism. "Atypical" and "creative" are words that can be used in an ambiguous fashion, perhaps even euphemistically. " His understanding of the case was certainly creative," meaning,"This line of thinking is so stupid that you had to have been creative to figure out how to draw such a dumb conclusion." Well, the colleague is a friend, so I figure that the words were meant as compliments, but the edge of ambiguity remains lodged in my mind. Am I being defensive, overly sensitive, or is it good that I "challenge" myself occasionally? Could he be jealous?

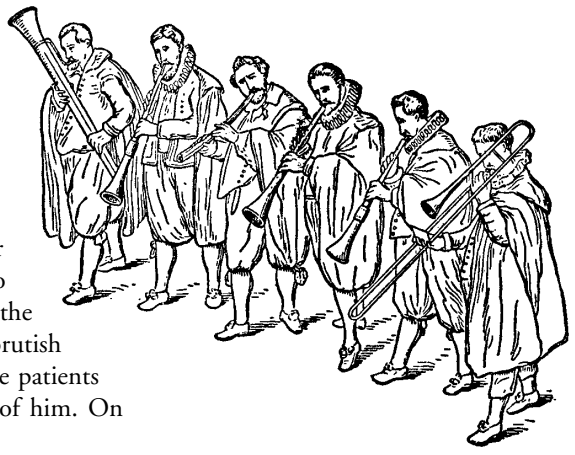
Recently my nurse practitioner, Steve, a super-nice guy, told me that he was getting overwhelmed with older men crying during their appointments. They were sad about divorces, deaths, grief over lost function; the usual stuff of debilitating disorders in older people. This doesn't happen to me. My patients, with rare exception, don't cry in my office, except maybe the first time when I give them a diagnosis that changes their life. Why do male patients cry in his office but not in mine? The answers to this question don't reflect that well on me. Do I rush them? Do I make them feel that I don't care? Am I too technical, focusing on a checklist of problems specific to their disease and not on their "soul," as Plato would have defined it? Am I too dour, unconcerned, cold-hearted? Of course I can't assess myself accurately. I like to tell myself that even if they don't unburden themselves to me, perhaps it's good that I, at least, think about it.

One of my patients told me recently that she liked visiting me, that I was a great comfort and she felt fortunate to have me as her doctor, quickly stating that she didn't feel that way when we first met. Not that I was cold or nasty, she confided, but "forbidding." Another patient told me, with a chuckle, that she had been at a PD exercise class in the morning and mentioned that she was going to see me in the afternoon, and another patient said, "You mean, Old Smiley?" making fun of my demeanor. She told me that she had told him, "He always smiles with me. He smiles a lot. I didn't know what he was talking about."

So, we're seen differently by different people. But the point of this commentary is not that, but to consider what image should I wish to convey to my patients? On the one hand I think it quite brutish to be seen as a doctor whose patients feel uneasy crying in front of him. On the other hand, I really don't want my patients to do this. If there is one thing I know about myself, it's that I'm not a "touchy- feely" kind of guy. I like to think that I'm kind, nice and sensitive, but how sensitive can I be if I give off the message, "Button up, check your emotions at the door."

On the other hand I admire Steve. I feel fortunate that he's part of our clinic, that middle-aged and older men feel comfortable sharing their feelings with him. He obviously provides a form of succor that I cannot. The patients are lucky to have this counterbalance. I hope that my patients don't feel my personal limitations are too limiting for them, but the difficult question is, how much do I really want or can change?

– Joseph H. Friedman, MD



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Teaching the World To Listen

"Language most shows a man: Speak, that I may see thee", implored Ben Jonson (1573 – 1637). Humans, according to Jonson, are essentially mute souls differing little from the vast array of lower vertebrate creatures except by the singular art of speaking; and thus arises their unique capacity to share their thoughts, inventions, speculations, even deceptions, with others. Speaking, therefore, has become the vehicle that we possess to teach each other, bring comfort to each other, even bind us together in times of adversity.

Life abounds with symmetries: for darkness, there is light; for wealth there is poverty; and for the art of speaking, there is the equally commendable art of listening. And yet when we assemble our generalizations extracted from the realities about us, speaking far outweighs the gentle but fervent skill of listening. The Oxford Book of Notable Quotations, for example, lists but five famous, quotable lines about listening but 58 priceless quotations about speaking. And so too does this proportion reflect every day reality: for every abiding listener, there seems to be at least ten earnest orators; surely then, many speeches must necessarily go astray since there are so few listeners to absorb their imparted wisdom. There is a sadness, then, that entire orations, embroidered with witty sayings, each filled with erudite insight, must flitter to nothingness, lost forever, for want of an eager congregation of avid listeners. In a world of symmetrical pairings, speaking may be paired with listening; or, it may be paired with not speaking, being silent. Yet surely listening is not the same as not speaking.

What type of preparation, what manner of advanced training is needed to transform a sentient human, from a non-speaker into a dedicated listener ? And are there educational centers – akin, perhaps, to those colleges teaching adults how to administer businesses – for formal training in advanced and abiding listening ?

Listening, listening in eager silence, is a sign of great forbearance and maturity. The wise man, listening in silence, says more than the endlessly talking simpleton. And even the Scriptural Proverbs declare: "Even the fool, when he holdeth his peace, is counted wise."

So, where, and under what circumstances, does one's education in listening commence ? Perhaps first in learning the rudiments of silence. "I have often repented speaking", said Xenocrates (396-315 BCE), "but never of holding my tongue." Silence is a magisterial presence, perhaps because it is so rare. Consider the many reasons for a human to stay silent. Firstly, to increase the likelihood of hearing something advantageous, something that might bring personal benefit to himself. And then, of course, one sometimes remains silent because one has nothing to say. And lastly, some interrupt their flow of words because their rare flashes of silence might bring wonderment and amusement to others.

Listening can be quite conventional especially when enhanced by periodic head-nods and barely audible sounds of approval. Listening can be analytical, with the expressed thoughts dissected, weighed and thoroughly debated thus indicating their inherent merit.

And then there is that mode of listening that requires an extra-corporeal organ, the third ear. Theodore Reik (1888-1969) discussed listening with the third ear as a crucial part of the armamentarium of the competent counselor: listening, and analytically absorbing not merely the uttered words of the speaker but also the words and ideas not openly expressed but nonetheless revealed by facial expressions, mood changes and the many inferred components of non-verbal communication collectively called body language.

Finally, listening to oneself is one of the great joys of life; it reaffirms the audacity of our thoughts and it gives credence to our dogmas. ("After all, if I said it, then it must be so !") But to the extent that genuine learning is principally accomplished through listening to others, listening rather than speaking should then be our dominant mode of learning. And the accrued benefits from listening ? Gladness, new thoughts, heretofore unrevealed secrets about the world and an endless sense of wonderment. Percy Shelley (1792-1822) said:

> Teach me half the gladness
> That thy brain must know,
> Such harmonious madness
> From my lips would flow
> The world should listen then –
> As I am listening now.

– Stanley M. Aronson, MD

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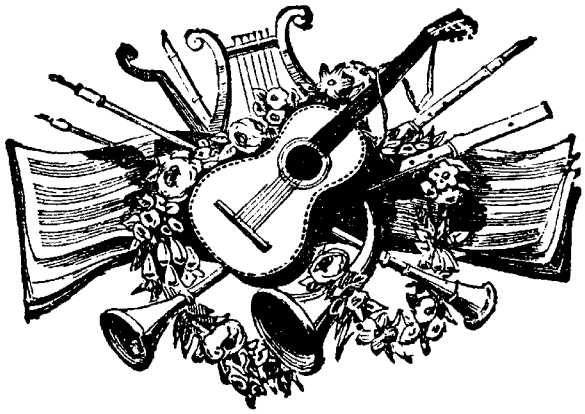

Office Screening for Dementia

Stefan Gravenstein, MD, and H. Edward Davidson, PharmD, MPH

Dementia is a common affliction in late life, affecting over 10% of US adults 65 and older, with prevalence doubling each five years of age thereafter. Dementia often goes unnoticed by primary care providers for several years after its onset. Not until sufficiently severe impairment such as difficulty with instrumental activities of daily living, including keeping appointments, impulsive purchasing, or getting lost driving prompts a caregiver to schedule a clinical visit or cognitive slip at the clinical encounter will the clinician realize a need to more carefully assess the patient. A public health threat with significant implications for patients, families and caregivers, dementia also produces a risk for increased health care utilization, including home health, nursing home and hospitalization; and, elder neglect and abuse. Those with mild cognitive impairment (MCI), but not dementia, incur a three to eightfold increased risk for ensuing dementia, depending on the definition used for MCI. Earlier recognition of MCI and dementia can help patients and those who care for them better prepare for later disability, and perhaps intervene to slow cognitive decline and keep patients and others safe. However, early recognition of cognitive impairment can only succeed if it is systematically implemented into clinical practice, a challenge for any busy primary care physician.

Cognitive Screening

Cognitive impairment often goes unnoticed because patients can cover defects by resorting to over-learned social skills and no real cognitive demands reveal the problems during usual social or clinical encounters. Busy clinicians will also miss cognitive impairment in many of their impaired patients unless they systematically look for the impairment. For a busy practice there are two complementary strategies that can help identify impaired patients or those at risk for impairment: having patients or their caregivers complete a screening questionnaire before the clinical encounter, such as in the waiting room; or, formally applying a dementia screening tool during the encounter. Of the several tools validated for screening, all have significant limitations (sensitivity and specificity) for identifying individuals with the least impairment. Also, more widely adopted tools for screening have the advantage of familiarity and ease of interpretation between providers who share patients, but such tools may take longer to administer or be less sensitive. Table 1 lists some commonly used screening instruments for cognitive impairment, including sensitivity and specificity for diagnosing dementia.

Of the available tools for screening, each has specific limitations, and the more comprehensive ones' greatest drawback has to do with the amount of time it takes to complete them. Of the ones that take more time to administer, the SLUMS has the potential advantage of greater sensitivity for mild impairment. The tools that take less time to administer generally miss important cognitive domains, whether language, spatial or executive function, and provide less utility in discriminating the source of the underlying pathology, but of these, we prefer the FAQ. The FAQ has the advantage of informing about functional impairment, directly informing potential interventions and types of community resources that can prove helpful to the patient and caregiver. The MiniCog has an embedded clock drawing activity, and is simple to score. A tool that can be consistently and efficiently applied can both provide a useful adjunct for systematic screening. More robust tools may offer added utility for monitoring progression of impairment, or response to interventions designed to stave off progression of impairment.

The approach to cognitive screening can mimic that of cardiac screening. Screening needs to include historical and clinical context, rather than just a screening instrument, and those patients who demonstrate risk or fail screening need further evaluation. If the patient has

Table 1. Cognitive screening: common tools for dementia.

Tool	Strengths	Weaknesses
Mini- mental status exam (MMSE)[1]	Well-known, best studied	Verbal, cultural bias; poor visuospatial, constructional praxis, problem solving; 5-10 m*
St. Louis University mental status (SLUMS)[2]	More sensitive for mild cognitive disorder than MMSE	Too complex for office use (10 min); age/education correction
Trails A[3]	Tests rapid visual search, 1-2 min	Not stand-alone; age/ education correction
Trails B[3]	Tests rapid visual search, 1-3 min	Not stand alone; age/ education correction
Mini-Cog[4]	Simple, 2-4 min, easy scoring	Not stand alone
Clock drawing test (CDT)[4]	1-5 min, minimal language, no prep	Not stand-alone
Time and Change[5]	Faster	Not stand-alone
Functional activities questionnaire (FAQ)[5]	Fast. Little skill to administer	Not stand-alone; needs informant
		Comparable to MMSE; may help distinguish MCI from Alzheimer's dementia

*time to administer

memory complaints or caregivers endorse memory issues, decline in instrumental activities of daily living—reduced ability to carry out routine daily tasks that require some cognition such as laundry, cooking, driving, shopping or balancing a checkbook—or are over age 65, brief cognitive screening is appropriate. If the screen is normal, consider rescreening every year or two. However, if the screen or clinical exam only establishes mild impairment, consider formal neuropsychological testing to establish a baseline, evidence of focal or more generalized dysfunction and whether further referral is needed to assist with additional evaluation. For patients who are clearly abnormal, evaluate for common causes of cognitive impairment, and consider formal neuropsychological assessment.

Clinical history should evaluate for risk factors for dementia: advancing age, history of loss of consciousness, vascular and cardiac risk factors including hypertension and hyperlipidemia, alcohol and drug abuse, metabolic disorders such as diabetes, and thyroid, other neurologic, psychiatric and infectious disease. Of neurologic disease, both familial history of dementia or cancer, and personal history of other neurodegenerative disease, falls, transient ischemic events, step-offs in cognition and cancer and can help. For psychiatric disease, depression and anxiety disorders can commonly affect cognition, especially for those who have a past history of these disorders. For infectious disease, history may identify risk for HIV, tuberculosis, or spirochetal disease, including syphilis or lyme disease. In any case, the history helps contextualize a differential diagnosis, and help target the exam for focal and other neurologic, vascular, infectious, metabolic, or findings. Always look for recent changes in medications, especially medications with anticholinergic effects (e.g, antimuscarinics, diphenhydramine), sedatives, and centrally active drugs.

In the screening activity, consider what other information the tool provides. For example, with visuospatial or executive function errors, consider whether a driving assessment may be indicated. With instrumental activities of daily living IADL dysfunction, consider whether the patient already has a power of attorney for finances and medical issues. Consider following abnormal cognitive screens with screens for depression, such as the geriatric depression scale (5-10 minutes), delirium using the confusion assessment method (CAM), and caregiver stress, like the one developed by Zarit and Zarit.[8, 9]

Patients who fail screening, in addition to a careful clinical exam, need laboratory evaluation to look for treatable conditions.

Screening is important to keep patients and their loved ones safe. In patients who fail screening, consider how you might assess whether they are safe with driving, judgment with managing a stove-top or electrical fire, taking medications independently, wandering or other accidents, such as falling or choosing an inappropriate temperature for food, home or bathing. A specific assessment of ability to manage medications, the Medi-Cog, combines the Mini-Cog with the ability to fill a pill box, has been studied.[10,11] Referral to an occupational therapist for a home visit can help elucidate the merit of such concerns, and even provide helpful interventions to the patient and caregiver.

Patients who fail screening, in addition to a careful clinical exam, need laboratory evaluation to look for treatable conditions. These will typically include a complete metabolic panel, complete blood count, TSH, B12 test, and may also include a lipid cascade, drug screen, ESR, cardiac evaluation (CNS perfusion), neuroimaging—especially for those patients with motor or focal findings, among other tests. Taken together, if laboratory testing is normal with the exception of cerebral atrophy, the clinical task remaining for most patients will be to distinguish mild cognitive impairment and the "three D's," depression, delirium and dementia from one another (Table 2).

Refer patients for whom the etiology remains unclear or who complement your clinical and diagnostic skill set, such as a neurologist, psychiatrist or geriatrician. The consultant will appreciate baseline information in cognitive and functional domains, and a copy of the screening tool you employed. Also, they can help address specific safety concerns you may have identified. The neuropsychologist can also help the physician consultants, especially in mildly impaired patients.

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Table 2. Distinctions between common causes of cognitive impairment

	Mild Cognitive Impairment	Depression	Delirium	Dementia
Confusion	Absent	Absent	Present	Variable
Attention	Good	Variable	Reduced	Good
Effort on tasks	Good	Reduced	Variable	Good
Consciousness	Clear	Clear, slowed	Clouded	Clear
Onset	Insidious	Recognized	Acute	Insidious
Duration	Months-years	Weeks-months	Acute	Months-years

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Clinical and Pathological Examples of Alzheimer's Disease, Dementia With Lewy Bodies, and Frontotemporal Dementia

Stephen Salloway, MD, MS

The differential diagnosis of dementia includes **Alzheimer's disease** (AD) (most common), **dementia with Lewy bodies** (DLB), vascular dementia, **frontal-temporal dementia** (FTD), Parkinson's disease, normal pressure hydrocephalus, and others. Post-mortem examination of patients with dementia reveals that mixed pathology is very common. Though much progress has been made in developing new diagnostic biomarkers, making a dementia diagnosis still requires a careful history from the patient and a knowledgeable informant. Determining the primary and secondary symptoms and the temporal course of the cognitive and functional decline are the keys to differential diagnosis. Has the onset of cognitive decline and progression been abrupt and step-wise, as seen with multi-infarct dementia, or insidious and gradual, consistent with AD? Knowing the patient's educational and occupational history can be helpful in estimating their level of cognitive reserve. Treatment of dementia is based on targeting the principal symptom, which may be memory loss, depression, Parkinsonism, or eliminating medications with deleterious side-effects.

AD is currently recognized as a progressive neurodegenerative disorder with preclinical, mild cognitive impairment, and dementia stages. There is increasing evidence supporting a AD pathological cascade with regional oligomeric and fibrillar amyloid extracellular deposits beginning ten to 20 years before the onset of cognitive symptoms, followed by accumulation of intracellular hyperphosphorylated tau protein, and later cortical and hippocampal atrophy on MRI.[1] Amyloid PET imaging and spinal fluid markers of amyloid and tau can detect the changes in amyloid burden during the prodromal period, providing an opportunity for detection and intervention before the full pathological and clinical expression of the illness.[2]

Clinical phenotypes of common dementia syndromes

Alzheimer's disease

Gradual onset and progression of episodic memory impairment is usually the cardinal feature of AD. The most common symptoms are misplacing items frequently, trouble keeping track of details, becoming repetitive, difficulty multi-tasking, and managing complex tasks such as balancing a checkbook, preparing a holiday meal, or navigating while driving. MRI may show diffuse cortical and hippocampal atrophy, and ventricular enlargement. Treatment with a cholinergic inhibitor tends to stabilize memory function during the first year of treatment and may make subsequent decline more gradual. However, the disease progresses despite treatment and disease-modifying treatments are needed.

The National Institute of Aging and an International Working Group have proposed new research diagnostic criteria for AD which include guidelines for the diagnosis of the mild cognitive impairment and preclinical stages of AD.[3-6]

Case example of MCI due to AD

A 66 year-old woman with a family history of dementia was evaluated for trouble misplacing items, being repetitive, and trouble recalling names. She was still managing her full-time job and driving without difficulty. Her Mini-Mental State Exam score was 28 and her **Montreal Cognitive Impairment Assessment** (**MOCA**) was 23.[7] Her MRI scan was normal. Apolipoprotein epsilon E genotype was 3,4. Detailed neuropsychological testing demonstrated an isolated impairment in episodic memory. The clinical diagnosis of mild cognitive impairment due to AD was supported by an elevated tau/amyloid ratio in CSF and elevated retention on amyloid PET scans consistent with AD. She was treated with a cholinesterase inhibitor and is participating in a clinical trial of an amyloid-lowering agent to try and slow the progression of AD.

Amyloid PET imaging can be used as a screening tool to detect the build up of cerebral amyloid in the preclinical stage of AD.

Case example of Preclinical AD

A 77 year-old man with no cognitive complaints responded to an ad for an Alzheimer's study because his mother had dementia at age 80. His MMSE was 29 with 3/3 recall. MRI showed "mild

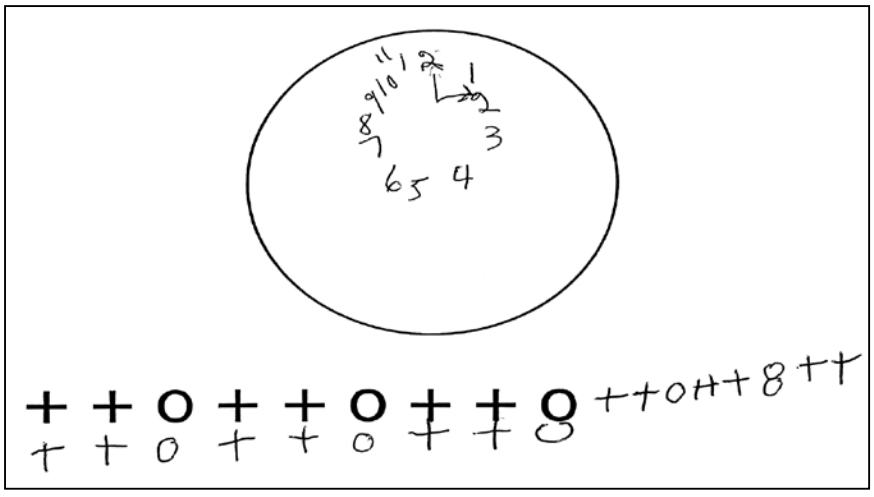

Figure 1. Clock drawing and copying of alternating sequences in the case of DLB. Clock drawing demonstrates evidence of visuospatial and executive impairment with central placement of numbers with mild micrographia. Copying of alternating sequences reveals executive impairment in continuing the sequence after the stimulus.

cortical atrophy, appropriate for age". His clinical dementia rating scale was 0. His amyloid PET score met the research criteria for preclinical Alzheimer's disease. He would be eligible to participate in a future clinical trial of an amyloid-lowering agent to delay the onset of cognitive symptoms.[8]

Clinical phenotype of dementia with Lewy bodies

The presenting symptoms of DLB vary widely and include amnesia, Parkinsonism, **REM behavior disorder (RBD)**, depression, hallucinations, delirium, and syncope. RBD is characterized by active sleep with thrashing, talking, or acting out of dreams. Patients may be injured crashing into furniture and dreams frequently have a violent quality. RBD is caused by onset of REM without atonia and may herald the presence of a Parkinsonian disorder greater than five years before the onset of cognitive or motor symptoms. RBD usually responds to a low dose benzodiazepine such as clonazepam given at bedtime.

Case example of DLB

A 70 year-old man began crashing into the walls at night during violent dreams two to three nights per week. He had no cognitive or neurological impairment. A sleep study revealed RBD which was successfully treated with low dose clonazepam. Six years later he developed mild cognitive symptoms and very mild Parkinsonian signs and seven years later he began seeing well-formed animals. MMSE was 26 and clock drawing showed central placement of the numbers with mild micrographia (see below). MOCA score was 18. A diagnosis of DLB was made. Treatment with a cholinesterase inhibitor stabilized memory symptoms and decreased the frequency and intensity of visual hallucinations for 12 months. After the first year his cognitive, behavioral, and motor symptoms progressed gradually despite treatment.

The diagnosis of probable DLB requires dementia plus 2/3 of the following, Parkinsonism, well-formed visual hallucinations, and fluctuating alertness. Executive and visuospatial deficits are often prominent in DLB and the MOCA is a more sensitive measure of cognitive impairment than the MMSE. It is important to identify the target symptom, cognitive, behavioral, motor, or active sleep, when treating DLB. There is a prominent cholinergic deficit in patients with DLB and these patients often respond well to treatment with cholinesterase inhibitors. Patients with DLB may be sensitive to side-effects of CNS medications, especially to neuroleptics. Low doses of CNS medications should be used with careful monitoring for side-effects.

The pathological diagnosis of DLB requires the presence of cortical cytoplasmic inclusions (Lewy bodies) composed of alpha synuclein protein. Post-mortem examination in patients with DLB frequently demonstrates mixed pathology with amyloid plaques in addition to cortical Lewy bodies.

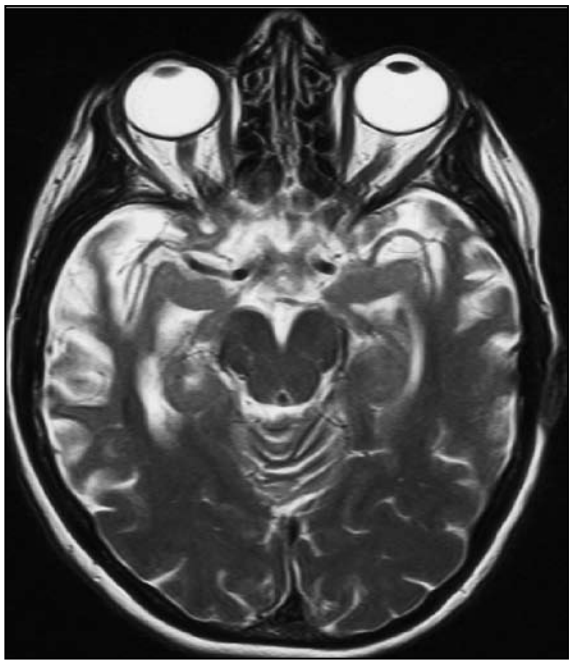

Figure 2 Axial T-2 non-contrast MRI in the case of FTD. The MRI shows significant anterior temporal lobe atrophy greatest on the right in the case of bvFTD.

Clinical phenotype of Frontal Temporal Dementia

There are three common subtypes of FTD, **behavioral variant (bvFTD)**, formerly Pick's disease, **progressive nonfluent aphasia (PNFA)**, and semantic dementia. The clinical and pathological spectrum of **frontal temporal dementia (FTD)** has recently been broadened due to discovery of new dominant mutations and now includes Parkinsonian conditions such as corticobasal degeneration and progressive supranuclear palsy as well as amyotrophic lateral sclerosis.

Patients with FTD are usually younger than patients with AD. Patients with behavioral variant FTD present with early decline in social conduct, emotional blunting, apathy, and compulsive behavior with relatively preserved episodic memory. Speech in PNFA is non-fluent and is characterized by at least one of the following, agrammatism, phonemic paraphasias, and anomia.[9] The cardinal feature of semantic dementia is failure to understand what common words mean.

Pathology and genetics of FTD spectrum disorders

Behavioral variant FTD is usually associated with cytoplasmic protein accumulations of tau (Pick bodies) or TDP-43. The tau pathology seen in bvFTD may be caused by a mutation in the tau gene on chromosome 17 or occur sporadically.[10] TDP-43 inclusions may be due to a mutation in the progranulin gene, located next to the tau gene on chromosome 17, be associated with an hexanucleotide repeat due to a mutation on chromosome 9p21, or are sporadic.[11-13] Semantic dementia is associated with asymmetric degeneration of the dominant temporal lobe and is primarily associated with TDP-43 inclusions that are usually sporadic. In general, two-thirds of cases of progressive aphasia are due to FTD and one-third to AD pathology. PSP and CBD are almost always associated with tau pathology. FTD-ALS is associated with ubiquitin positive TDP-43, tau negative, inclusions and may be associated a mutation on chromosome 9p21.[12,13]

Case example of bvFTD

A 55 year-old woman presented with gradual onset and progression of difficulty finding words and a change in behavior over one year. She was still working full-time as a business executive and driving

without difficulty. She was more anxious and impatient and had decreased regard for the feelings of others. Her MMSE was 25 with 3/3 on delayed recall. She had mild word finding difficulty and a normal sensorimotor exam. Her MRI revealed prominent temporal and frontal lobe atrophy. Her mother had a progressive dementia beginning in her late fifties with a similar pattern of asymmetric atrophy on MRI. Her mother's autopsy demonstrated FTD, tau + Pick type. Genetic testing revealed a mutation in the tau gene on chromosome 17. Our patient experienced a steady decline in behavior and language over six years and now requires full-time care. Trials of cholinersterase inhibitors, memantine, and a number of medications to control behavioral symptoms were ineffective. No medications are currently approved to treat FTD but future trials are likely to target the primary pathology such as tau or progranulin.

Summary

Dementia syndromes usually consist of distinct clinical and pathological phenotypes. A careful history is required to document the onset and progression of symptoms to generate the differential diagnosis. New biomarker tests can provide evidence to increase diagnostic certainty. Disease-specific interventions, based on advances in genetic and molecular biomarkers, are likely to have the greatest impact when given in preclinical and early symptomatic phases.

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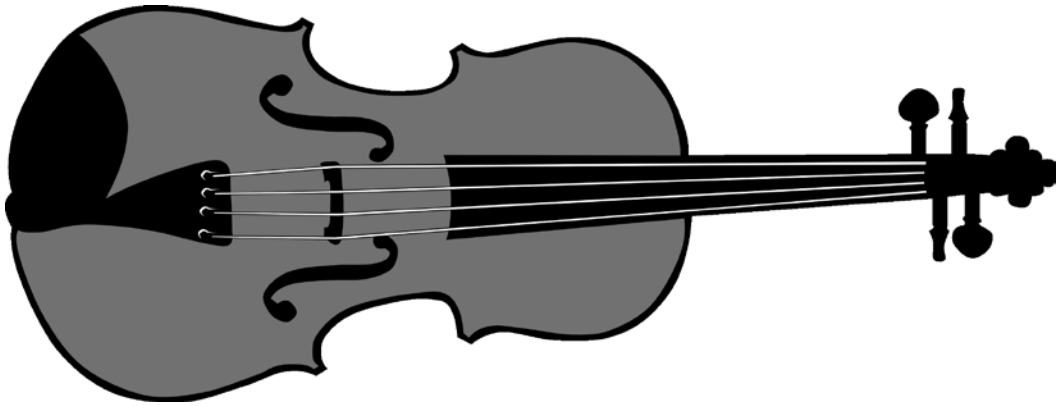

Depression and the Aging Brain

Laura Stanton, MD, and Robert Kohn, MD, MPhil

Impact of Aging on the Epidemiology of Depression

Depressive disorders among the elderly are a disabling illness that can result in death and need to be properly diagnosed and treated by clinicians. Although the rates of depressive disorders are lower in the elderly than in younger cohorts, prevalence rates are still high and accounts for nearly two percent of all disability in those over 60. Recent data shows that the 12-month prevalence of **major depressive disorder (MDD)** from ages 65-74 is 3.1% and the lifetime prevalence is 11.7%.[1] Similar to depressive disorders in the younger population, females are at higher risk for MDD with a 4.5% 12-month and a 16.9% lifetime prevalence from ages 65-74; whereas, men have a 1.4% 12-month and a 5.1% lifetime prevalence. The prevalence of MDD is highest among non-Hispanic white individuals with a lifetime prevalence of 13.2%, and lowest among African-American elderly, 5.1%.[2]

The lower rates of MDD among the elderly are perplexing to many clinicians. There are several factors to suggest why these rates are lower compared to younger cohorts: 1) older persons may have greater difficulty remembering past symptoms; 2) they may be less psychologically oriented in responding a mental health interview; 3) there may be a cohort effect in younger generations of an increased rate of MDD; and 4) persons who have had MDD may be less likely to live until older age, more likely to be institutionalized, and have more medical comorbidities precluding their participation in epidemiological surveys. Despite these lower rates among the elderly in the community, rates of MDD in primary care settings are high (five percent) and even greater among the nursing home population, 15-25%, and those in acute care hospitals, as high as 12%.[3]

Impact of Aging on the Risk Factors for Depression

Many stressors common to late life serve as important risk factors for developing major depression: physical illness, limited mobility, sensory deprivation from deafness or blindness, retirement, economic deprivation, poor living conditions, social isolation, rejection by children, as well as loss of spouse. A prior history of depression places elderly patients at substantial risk. It has also been hypothesized that the elderly may have specific physiologic changes that place them at particular risk; these include decreased acetylcholine, dopamine, and norepinephrine as well as increased monoamine oxidase. In addition, there are changes in the hypothalamic-pituitary-adrenal axis with increased levels of cortisol.

Other risk factors include commonly prescribed medications such as analgesics, antihypertensives, antibacterials, anti-parkinsonian drugs, cancer treatments, cardiovascular medications, estrogens, progestational agents, hypoglucemic agents, sedatives and steroids. There are many medical conditions that are frequently associated with depression such as stroke, Alzheimer's Disease, Parkinson's disease, cancer as well a chronic pain and many others.

Impact of Aging on the Clinical Presentation of Depression

The majority of elderly individuals who have MDD have suffered from early onset depressive disorder; only a minority have late-onset depression, first episode after the age of 50. Personality abnormalities, positive family history, family dysfunction, and guilt are more likely to be seen in early rather than late-onset depression. Anxiety, apathy, hypochondriasis, apathy, loss of interest, cognitive impairment and psychosis are associated more often with late-onset depression. Structural changes in the brain's subcortical structures, leukoencephalopathy, frequently seen in the elderly with MDD are more severe in late-onset depression and independently predict worsening of quality of life and disability.[4] Due to the association with leukoencephalopathy and cardiovascular risk factors, the vascular depression hypothesis has been proposed to explain the etiology of late-onset depression.[5] The clinical presentation of late-onset depression proposed by the vascular depression hypothesis include: reduced depressive ideation, greater psychomotor disturbance, apathy, executive dysfunction on neuropsychological testing, and neuroimaging abnormalities in the basal ganglia and white matter. Regardless of age of onset, the most important risk factors for developing depression in later age are being female, experiencing lack of satisfaction with life, feelings of loneliness, smoking and bereavement in the last six months of life.

The non-detection of major depression poses significant consequences including higher rates of nursing home placement, increased burden on caretakers, increased visits to physicians, and increased risk of physical disability. Some investigators believe that there may be increased mortality among the elderly who have depression, but this remains controversial. Unfortunately, detection of depression poses a challenge for many; nursing home staff recognize depression in only in 37%-45% of patients, and primary care physicians only diagnose half and treat less than half of those with MDD.

Impact of Aging on the Course of Depression

Clinical characteristics of depression may differ among the elderly and may manifest with more somatic complaints, anxiety, apathy and anhedonia. There may be unexpected functional decline and a resistance to care. Clinical screening scales, such as the Geriatric Depression Scale and PHQ-9, are helpful for clinicians to improve detection of depression among their elderly patients. Screening for suicide is also necessary, as suicide increases with age among men. For women, suicide rates do decrease slightly after age 55. Suicide attempts in the elderly need to be taken extremely seriously, as suicide lethality is exponential with age.[6]

Although, the median time to recovery from a MDD episode is no different among older and younger patients, the elderly are much more likely to experience

recurrence.[7] Unlike younger individuals with MDD, there are no good predictors for recovery or recurrence among the elderly, including medical comorbidity.

Impact of Aging on Treatment of Depression

Treatment of depression in the elderly must take into account pharmokinetic and pharmacodynamic changes in late life. Pharmacokinetic changes include decreased absorption, increased volume of distribution, decreased metabolism, and decreased excretion. Patients in later life may have age-related changes in drug sensitivity. Elderly patients may have pharmacodynamic changes that make them more vulnerable to anticholinergic and noradrenergic side effects of medication, due to age-related receptor sensitivity and age-related changes in cholinergic and monoaminergic neurotransmission.[8] Dosing, therefore, should start low doses and titrated slowly. If administration is a challenge, many antidepressants come in liquid form or have soluble tablets. Despite these concerns, clinicians should be attentive not to undertreat these patients and fail to provide adequate trials at therapeutic dosages.

Acute treatment of depression in the elderly frequently begins with a trial of a **selective serotonin reuptake inhibitor (SSRI)** for four to twelve weeks with the goal of remission. SSRIs are generally well tolerated in the elderly and have limited drug-drug interactions and less likely to be discontinued. A good trail is one that has achieved a therapeutic dose in at least eight weeks and about 60-70% of patients will respond. Once there is resolution of depressive symptoms, maintenance treatment should be continued for at least four to six months in order to consolidate remission and recovery. Compared to placebo, continued treatment with antidepressants is more efficacious in preventing relapses and recurrences.[9] In absence of maintenance treatment, 30-90% who achieved recovery will experience recurrence in eight to 48 months.

Unfortunately failure of response to SSRIs may be as high as 77%. Therefore, augmentation of the SSRI with buproprion, lithium or nortryptiline could be considered. For lithium, drug levels and renal function should be closely monitored. In addition, augmentation using other antidepressants, such as mirtazapine and venlafaxine, may be effective. There are several atypical antipsychotics that are FDA approved as augmentation strategies. The data on treatment of non-remission of depressive symptoms is limited and results are not optimal; this will hopefully be an area of greater clinical investigation in the future.

Treatment of depression in the elderly must take into account pharmokinetic and pharmacodynamic changes in late life.

First line treatment for concomitant psychotic symptoms is with antipsychotic medications with careful monitoring of extrapyramidal syndromes and tardive dyskinesia. Avoiding anticholingeric agents, monitoring QT interval, and the need to assess for Parkinson's symptoms, as well as for metabolic syndrome, should be considered in selecting an agent.

Electroconvulsive therapy (ECT) remains an effective treatment of for MDD in the older population, and a consideration among those with pharmacological treatment resistant disorders, concomitant psychotic symptoms, high suicide risk, and rapidly physically declining due to neurovegetative symptoms. There are few absolute medical contraindications to ECT. In addition, non-pharmacological interventions with emphasis on increased socialization, activity and exercise as well as psychotherapy have proven to be effective treatments for milder cases of depression in the elderly population.

Aging does affect the epidemiology, risk factors, clinical presentation, course, and treatment of depression in the elderly. Depression, however, remains the most treatable psychiatric disorder of late life.[10]

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Treatment of Neuropsychiatric Symptoms in Dementia

Michael Friedman, MD

Dementia is a disorder characterized by cognitive or behavioral deficits that interfere with ones ability to function at work or in their usual activities and represents a decline from previous level of functioning. Most patients diagnosed with dementia develop neuropsychiatric symptoms at some stage during their illness.[1] **Neuropsychiatric (NP)** symptoms are associated with an accelerated decline in overall patient functioning, increased use of medications, frequent hospitalization and earlier entry into nursing homes or supportive living environment. They are highly disruptive to caregivers and family members, greatly affecting quality of life of those caring for patients with dementia. Quality of life is found to be lowest in those caring for patients with symptoms of agitation, aggression and irritability.[2]

NP symptoms are heterogenous and not specific to dementia. While symptoms are associated with regional changes in glucose metabolism or neurotransmitter receptor populations, their presentation is shaped by interactions between the primary pathologic process, cognitive limitations, medical comorbidity, premorbid personality and coping style, environment and other psychosocial factors. The frequency of NP symptoms may vary depending the cause and stage of the dementia. Apathy, dysphoria and irritability are common in **mild cognitive impairment (MCI)** secondary to **Alzheimer's disease (AD)** while agitation, delusions and hallucinations tend to occur in later stages of dementia.[3] In dementia secondary to **frontotemporal dementia (FTD)**, aberrant motor behaviors and disinhibition may be common whereas in dementia with Lewy Bodies, visual hallucinations may be prominent in early stages. While symptoms may be transient, apathy for example, often persists through progressive stages of the illness.

NP symptoms not only lead to a lower quality of life of patients with dementia and their caregivers, they are also associated with progression of illness. Depression may manifest months before onset of clinically apparent cognitive symptoms and is a risk factor for MCI when present in the elderly without cognitive impairment. When present in those with MCI, it is found to translate to more than twice the risk of conversion to AD over those without depression.[4]

Depression is a common syndrome in patients with cognitive impairment. Symptoms, however, must often be obtained from family and caregivers as patients may not report them. History taking should include attention to sleep pattern, appetite, weight changes, hedonic capacity, irritability and agitation. As the disease progresses, language problems and reduced awareness of symptoms may further limit report of symptoms.

Depression is a common syndrome in patients with cognitive impairment.

Treatment of depression in the cognitively impaired patient should incorporate a multimodal approach tailored to the patient's cognitive and physical abilities. Developing a daily routine with structured activities, education of caregivers and assessment of social and support networks are critical first steps. Adult day care centers may offer structured socialization as well as physical and cognitive based activities and should be part of the treatment.

Evidence based pharmacologic treatment for depression in patients with dementia is limited. The results have generally been mixed with some studies suggesting use of antidepressants only after non pharmacologic interventions have failed unless indicated by severity of symptoms or level of acute risk. SSRIs (e.g., sertraline or citalopram) should be considered first for treatment of depression associated with dementia.[5] Initial dosing should be conservative though titration to maximum dosing may be necessary depending on response. Tricyclic antidepressants are less well tolerated and and should be used with caution. Anticholinergic effects of the tricyclics may contribute to cognitive deficits making their use less practical. Cholinesterase inhibitors have been found to improve mood and other non-cognitive symptoms in patients with dementia though the results are not robust. Electroconvulsive may be considered in patients with severe or refractory depression or in those with risk of suicide. ECT is associated with delirium though it has been shown to improve cognitive function when successfully treating depression in patients with degenerative dementia. A trial of stimulants may be warranted for patients who respond partially to SSRI or who display prominent psychomotor retardation.

Apathy is defined as diminished activity due to lack of motivation. Behaviors associated with apathy include 1) decreased goal directed executive functioning, for example initiating daily chores or hygiene; 2) decreased goal directed behaviors, for example socializing; and 3) decreased emotional expression. Depression and apathy have symptoms such as diminished interest and psychomotor retardation in common making differentiation difficult.[6] Patients with cognitive decline may not complain of apathy. Apathy may be particularly frustrating for family members as patients with apathy may appear to be able to function to higher levels than they are. Family members and caregivers may mistakenly interpret their apathy as "lack of effort" or "oppositional."

Treatment of apathy should begin with education of caregivers and engagement in repetitive structures activities in a social setting. Acetylcholinesterase inhibitors have been found to have beneficial effects in some clinical trials and case reports have suggested benefits from psychostimulants. SSRI's may increase apathetic syndrome thus caution is advised.

Agitation, aggression and psychosis have the most profound negative effect on overall level of functioning of patient and quality of life of caregivers and family. It is necessary to consider a broad range of causative factors including reversible causes such as pain syndromes, infection, metabolic derangement, medication effect and other psychosocial/environmental factors. Agitation and aggression are

often triggered by misinterpretations or misperceptions and are associated with frank psychosis and delirium. Behavior intervention should be initiated early however if the symptoms are associated with potential risk of harm or significant distress to patient, caregiver or community, pharmacologic management is indicated.

Much has been written about use of second generation antipsychotics for treatment of neuropsychiatric symptoms associated with dementia. Meta analysis of placebo controlled trials using second generation antipsychotics for elderly patients with dementia related agitation has shown an increased risk of mortality (odds ratio 1.5 – 1.7.) [7,8] In 2005 the FDA issued a public health advisory warning against increased mortality for second generation antipsychotics for the treatment of behavioral symptoms in elderly patients with dementia.

Effectiveness studies of antipsychotics for patients with neuropsychiatric disturbances in dementia have shown mixed results. The CATIE AD trial studied effectiveness of olanzapine, quetiapine, risperidone and placebo in outpatients with AD and psychosis or agitated/aggressive behavior. Medications were discontinued in 83% of patients by week 36 due to intolerability, side effects or perceived lack of efficacy. Parkinsonism or EPS was highest in those treated with olanzapine and risperidone. Sedation and confusion were noted with all three medications though particularly likely with olanzapine. Increase in body weight and body mass index were noted with all three antipsychotic medications though most prominent with olanzapine and risperidone. Overall no significant differences were noted among the groups with regard to improvement on the Clinical Global Impression of Change scale at 12 weeks. The group concluded that adverse effects offset advantages in the efficacy of atypical antipsychotic drugs for treatment of psychosis, aggression or agitation in patients with AD.[9]

Subsequent studies have noted improvement on measures of specific clinical symptoms for example measures of hostility, suspiciousness, mistrust and uncooperativeness[10]. Although few studies report improvement in functional abilities or quality of life, improvement in psychiatric and behavioral symptoms may be clinically meaningful for individual patients without affecting overall function. Given the paucity of data supporting other classes of medications for treatment of agitation, aggression and psychosis in AD (anticonvulsants, mood stabilizers, benzodiazepines) and the frequent need for augmentation of non pharmacologic treatments, second generation antipsychotics continue to be frequently prescribed for neuropsychiatric symptoms in dementia.

Irritabilty/lability may be an early behavior symptom of cognitive impairment and may be influenced by underlying mood disorder, frustration with cognitive limitations, personality and coping style and quality of support network. Cholinesterase inhibitors and memantine, in addition to their cognitive enhancing effects, have been shown to have modest impact on irritability/lability of mood and should be considered along with SSRI's as pharmacologic intervention for irritability/lability in patients with AD. SSRI's have a broad range of effects including anxiolysis, anti-obsessional and anti-compulsive effects and should be considered for patients with cognitive impairment with anxiety syndromes. Benzodiazepines may worsen cognitive symptoms and are associated with increased risks of falls in the elderly population. Their use should be limited to situations that may require rapid onset with time limited effects while under close observation.

Personality changes may include coarsening or softening of personality characteristics. Disinhibition may present with impulsivity, tactlessness, violation of social boundaries, or sexually inappropriate behaviors. Primary treatment modality is behavior management aimed at identifying and avoiding triggers. Pharmacologic treatment has limited utility for personality changes or sexually inappropriate behaviors.

Non pharmacologic treatments should be considered first for NP disturbances associated with dementia. Such behaviors as wandering, hoarding, repetitive questioning, inappropriate behaviors will often respond to behavior therapy techniques though for limited periods of time. Day care centers, structured and semi-structured living environments will often provide an environment matched to the functional capacity while fostering independence, comfort and familiarity. A mulitidisciplinary approach with adequately trained staff providing mulitimodal treatment options that are patient specific can be invaluable in helping patient, family and caregiver cope with the behavior and psychological symptoms of dementia.

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Statement of Off-Label Discussion

All of the medications discussed here are off-label.

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Delirium In the Elderly

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Delirium is an acute organic mental syndrome characterized by disturbance in level of consciousness, disorientation, attentional impairments, perceptual disturbances, cognitive impairments and occasionally severe behavioral problems. The term "delirium" is based on the Latin roots *de, lira*, and *ium*, which literally mean "a going off the ploughed track, a madness." The term delirium has been known since 1 AD by the writer, Celsus, who described it in *De Medicina*. Nursing staff will often use the terms, "sundowning" or "ICU psychosis" to describe the acute mental status changes associated with delirium. Yet, neurologists prefer the term "encephalopathy", which literally means "disease of the brain." Regardless of the term used, delirium is not a benign condition and markedly extends hospital length of stay and increases the risk of further morbidity and mortality. ***The Diagnostic and Statistical Manual of Mental Disorders, 5th Edition*** **(DSM-5)** (due out in May 2013) will likely define delirium as a disturbance in level of awareness or attention (rather than consciousness as in the previous edition), marked by the acute or subacute onset of cognitive changes attributable to a general medical condition; and it tends to have a fluctuating course. DSM-5 will also likely add supportive features and subtypes, such as hypoactive, hyperactive, and mixed.[1]

Defining the Problem and Epidemiology

Delirium is one of the most common syndromes older patients develop and one that clinicians miss at the reported rate of 32% to 66%.[2] The prevalence of delirium upon admission to general medical units is between 10% and 31%, however most authorities place the estimate closer to 30% in the elderly population (65 years of age an older). In general, surgical patients have been found to have higher rates of delirium than medical patients, with coronary artery bypass graft patients having the highest risk of post-operative delirium (greater than 50% in five of 14 studies reviewed). The highest rates of delirium are found in the intensive care setting and in those with terminal illness, where rates of 80% or more have been noted. Delirium is especially common in nursing homes after brief hospital admissions because the average length of duration of the delirium is 21 days, while the average hospital length of stay at most general medical hospitals is approximately five days. Therefore, many patients are being discharged to nursing homes while still delirious. In one study, 72% of 214 patients in nursing homes who were hospitalized for delirium still had delirium at the time of discharge back to the nursing home. The delirium persisted for 55% of the patients at one month and 25% at three months after discharge.[3] The rates of delirium in patients who are ill and elderly, but cared for at home are much lower, than for those treated in the hospital.

The patients at the highest risk for delirium include older patients with severe dementia, who have multiple comorbidities.

Pathogenesis of Delirium

Delirium is a complex neuropsychiatric syndrome, often multifactorial in origin and likely affecting multiple domains of the central nervous system. The most widely accepted hypothesis involves the cholinergic neurotransmitter system, suggesting that deficiency may be one of the underlying factors causing delirium. It is well-known that anticholinergic drugs, such as atropine or diphenhydramine (Benedryl®) can elicit the symptoms of delirium in predisposed individuals. Other hypotheses include melatonin abnormalities, which would explain the term "sundowning", in which behavioral symptoms emerge as light levels decline and melatonin levels spike during the evening. Neuronal damage is an alternative explanation, secondary either to oxidative stress or inflammation. A link between inflammation and neurotransmission has been proposed, with inflammation-induced perivascular edema leading to hypoxia and subsequent reduced synthesis of acetylcholine.[4]

Electroencephalogram (EEG) is a sensitive but usually unnecessary test for the presence of delirium. EEG findings reveal a decrease in fast alpha frequencies and an increase in the slower theta rhythm. Unfortunately, this is a non-specific finding, but is telling of global brain dysfunction.

Risk factors for delirium

Delirium is often the initial manifestation of an underlying acute medical illness and may be present before signs such as fever, tachypnea, tachycardia, or hypoxia. The patients at the highest risk for delirium include older patients with severe dementia, who have multiple comorbidities. In these highly vulnerable patients, a medication such as an opioid narcotic may induce delirium. Older patients are more likely to have multiple vulnerability factors, therefore, they are disproportionately more susceptible to becoming delirious compared with younger patients. Dementia is probably the most consistently observed independent vulnerability factor for delirium across different clinical settings.[5] Marcantonio and colleagues identified seven predictors that could be used preoperatively to stratify an individual patient's risk of delirium. These factors include age greater than 70 years, self-reported alcohol abuse, poor cognitive status, poor functional status, abnormalities of serum sodium, potassium, or glucose, non-cardiac thoracic surgery, or abdominal aneurysm surgery. [6] Lower education, by reducing cognitive reserve, increases delirium risk, and when present, is of longer duration. Sensory impairments, especially visual loss, also greatly increases the risk of delirium in a vulnerable population.

Medications, especially polypharmacy, are a well-known cause of delirium in the elderly. Medications with anticholinergic properties, benzodiazepines, and narcotics are notorious for precipitating and exacerbating delirium. Medications with anticholinergic properties are more frequently associated with delirium than any other drug class; moreover, there are over 600 medications known to have these properties on the market. One-third of all of the elderly use over-the-counter sleep aids, most

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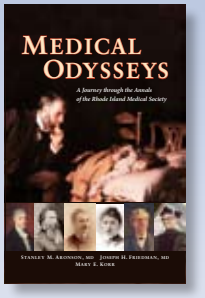

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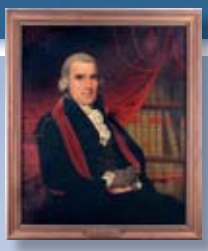

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The 1795 portrait of RIMS' first president, Amos Throop, was restored to optimal condition for public display.

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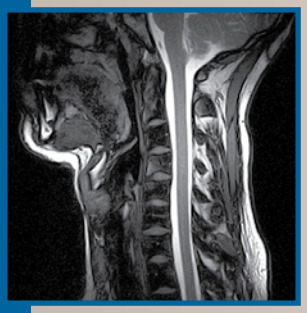

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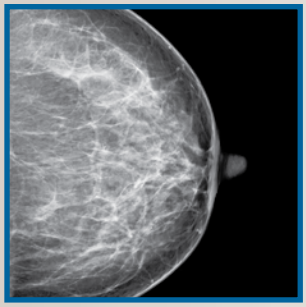

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of them antihistamines with anticholinergic properties. Delirium with mania may occur in patients exposed to parenteral steroids and occasionally even oral steroid doses.

At this point, there are no known genetic markers for predisposition to delirium. Although one study showed higher rates with those patients with apolipoprotein E4, this was confounded by the high rate of Alzheimer's dementia in this population. Another study on this gene showed no effect on the rate of delirium.

Interventions to prevent delirium

Multidisciplinary strategies have been implemented at many hospitals in order to prevent delirium and mitigate its duration. These strategies rely upon nursing care and the environment of care rather than additional pharmacotherapy. Patient safety is a high priority given the propensity for delirious patients to fall, aspirate, and develop pressure ulcers. At our institution, The Miriam Hospital, restraints are almost never used with the delirious elderly patient. In the case of delirium tremens, most studies indicate an increased mortality in those patients who required restraints during the admission. Instead, we have chosen to specially train certified nursing aids in a skill set that allows them to be both sitters and clinicians adept at handling delirious patients. They are usually assigned to one individual patient and can help mitigate falls, aspiration and skin breakdown. They provide frequent reorientation, access to sunlight, and they help optimize sensory losses that may be contributing to confusion. Urinary catheters are removed as soon as possible and the patients mobilize early in their stay. Delirium tool kits are also implemented when the delirious patient is found tugging at lines or medically necessary tubing. When these non-pharmacologic methods are unsuccessful, the team usually will obtain consultation from our geriatric psychiatry advanced practice nurse and/or the psychiatry consultation-liaison service. In fact, at our institution, approximately one-third of the psychiatry consultation-liaison consults are for evaluation and management of the delirious patient.

There have been some recent studies that have looked at preemptive use of antipsychotics to prevent delirium in highly vulnerable patients. At least one randomized, controlled trial addressed the issue of prophylactic haloperidol. In at-risk patients aged greater than 70 years, oral haloperidol 0.5mg twice a day was administered up to 72 hours preoperatively until the third post-operative day. The study found that prophylactic haloperidol use did not alter the incidence of post-operative delirium (15.1%) compared to placebo (16.5%).[7] Other studies on prophylactic haloperidol, in patients undergoing elective hip surgery, have showed a decrease in delirium duration and hospital length of stay but no difference in incidence of delirium. [8] Studies on risperidone and olanzapine were also conducted and seem to point toward decreased incidence of post-operative delirium, however, antipsychotic prophylaxis is generally not utilized at this point due to potential drug-induced side effects and lack of solid data.

Management of Delirium including behavioral problems

The single most effective treatment of delirium is to diagnose and treat the underlying cause. At this point, there are no FDA-approved medications for the treatment of delirium. The most commonly used drug for behavioral problems in delirium remains haloperidol. Haloperidol (Haldol®) is a commonly used antipsychotic and has been shown to improve delirium severity. Intravenous haloperidol should be administered cautiously in light of the black box warning regarding possible QT prolongation and subsequent torsades de points. There have been few studies on the newer atypical antipsychotics such as quetiapine (Seroquel®), risperdone (Risperdal®), olanzapine (Zyprexa®), and aripiprazole (Abilify®), however they are often used in the medical setting due to health provider fears over the black box warning on haloperidol. Until solid clinical studies are performed on these medications, there are few if any benefits over the traditional use of haloperidol in this setting. At least one caveat exists, that patients with parkinsonian disorders, and especially dementia with Lewy bodies, avoid haloperidol due to the possibility of irreversible motor damage. In these cases, quetiapine would be a safer option for treatment of the behavioral symptoms associated with delirium.

One randomized trial attempted to compare the efficacy of antipsychotic medications and lorazepam (Ativan®) in delirious patients, but was prematurely terminated because the lorazepam arm showed a higher prevalence of treatment-limiting side effects such as oversedation, disinhibition, ataxia, and increased confusion.[9] The typical antipsychotics studied, including haloperidol and chlorpromazine (Thorazine®), were found to be effective in controlling behavioral symptoms in these delirious patients. However, in the case of delirium tremens from alcohol or benzodiazepine withdrawal, lorazepam still remains the medication of choice.

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Disclosure of Financial Interest

The author and/or his spouse/significant other have no financial interests to disclose.

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Managing Older Adult Driving Safety in the Primary Care Setting

Melissa M. Amick, PhD

The number of drivers over the age of 75 will dramatically increase over the next few decades. Adults age 85 and older have the highest rate of driver fatalities.[1] Individuals age 70 and older have the greatest motor vehicle fatality rates per mile driven compared to all but the riskiest age group, those 25 years and younger. Advanced age is also a risk factor for motor vehicle crashes.[2] Since driving safety declines as older adults age, clinicians caring for this population may be increasingly called upon to evaluate the driving safety of their elderly patients.

Dementia and Driving Safety

Considering the high prevalence of dementia with increasing age, physicians are increasingly confronted with the challenge of assessing driving risk in those with cognitive compromise. In 2010, the **American Academy of Neurology (AAN)** published its practice parameters for evaluating driving safety among patients with dementia.[3] Previous studies have found that motor vehicle crashes and failure rates on road tests and simulated assessments all increase with increasing dementia severity, and generally, those with moderate stage dementia are considered unsafe to drive.[4] The AAN recommends considering dementia severity to guide clinicians' decision-making.

A high percentage of individuals with very mild or mild dementia, however, are able to pass a standardized road test.[2] Consequently, in those individuals with milder cognitive compromise, the AAN practice parameters recommend the consideration of additional information regarding risk factors for unsafe driving. Risk factors to assess include caregiver report of the patient's marginal or unsafe driving skills, as well as recent history of crashes or citations, reduction in miles driven per week, avoidance of complex driving situations, aggressive/impulsive driving habits, and a Mini Mental State Examination score of ≤ 24. Based on dementia severity and number of risk factors reported, the AAN recommends following state requirements for reporting unsafe drivers. Risk management options articulated by the AAN include counseling the patient for options for alternate transportation, discussing the need to relinquish driving privileges, and possibly referral for outside assessment of driving safety (e.g., DMV or professional driving instructor). In patients where risk is considered very low, follow-up every six months has been recommended.[4]

Considering the high prevalence of dementia with increasing age, physicians are increasingly confronted with the challenge of assessing driving risk in those with cognitive compromise.

Office-based assessment of older driver safety

In 2010, the **American Medical Association (AMA)** also updated their *Physicians Guide to Assessing and Counseling the Older Driver*, *a* free on-line publication.[5] The AMA's guide provides a description of how to evaluate a range of age-related medical risk factors for unsafe driving. A thorough review of all medical conditions that may affect driving safety including, but not limited to, seizures, syncope, respiratory disease, diabetes and cardiac events, as well as a consideration of medications that may have sedating side effects is recommended by the AMA's guide.

With regard to office-based testing to determine driving risk, the AMA's guide recommends the **Assessment of Driving-Related Skills Battery (ADReS)**. This battery includes assessment of visual functioning (acuity and visual fields), cognitive screening (clock drawing and the Trail Making test), and motor functioning (20 foot walk, tests of range of motion and motor strength). It should be recognized that brief office-based assessments to determine driving safety, such as those that comprise the ADReS, have been criticized for limited evidence to support their clinical utility for distinguishing safe from unsafe drivers. Importantly, the AMA's guide emphasizes that medical, sensory, and motor deficits should, if possible, be addressed and then followed up to determine if there is still persisting concern about driving safety. If issues still persist, then referral to a driving specialist is recommended. Care providers should know their state's requirements for reporting unsafe drivers, and the development of policy for reporting unsafe drivers should be reviewed by appropriate legal counsel.[4] According to the AMA's guide, Rhode Island does not mandate reporting of unsafe drivers. Physicians can, however, report patients thought to be unsafe drivers due to a medical conditions through the medical advisory board of the Department of Motor Vehicles. Physicians appear to be generally protected from legal reprisal, so long as the reporting is done in good faith and with due care. Please check with the Rhode Island DMV and the AMA's Guide (Chapter 8, page 58) for details on reporting.[5]

Older Driver Remediation

As described above, age-related declines in sensory and motor functioning, and to some degree cognitive functioning, does not mean that driving cessation is required. Considering the reality that within many communities there are limited alternate forms of transportation, it is important to try and maximize driving safety, when feasible. In addition to addressing any modifiable medical conditions, driving safety may be increased by referral to an occupational therapist or driving specialist. Adaptive equipment for

the vehicle is available for those individuals with some sensory and physical limitations. For example, hand controls for individuals with decreased lower extremity sensation and parabolic mirrors for those with reduced neck range of motion may allow some older adults to continue to safely operate a motor vehicle.

Beyond physical modifications of the vehicle, the most common form of intervention to improve driving safety has been older-driver education. Classroom-based and on-line education programs are offered by AARP, AAA, and other state run agencies. Few studies have examined the efficacy of education-based interventions. The results of these limited clinical trials have been mixed; one study reports that education is associated with fewer citations, one reports that education was linked to increased crashes, and three studies detected no influence of education upon crash risk.[6,7]

Use of a **cholinesterase inhibitor (ChEI)** may help drivers with early stage dementia prolong their ability to safely operate a motor vehicle. In a preliminary study with a small sample of participants with mild Alzheimer's disease (n=24), it was found that simulated driving performance improved following treatment with ChEI.[8] Furthermore, Daiello and colleagues observed that performance on the simulated driving assessment was better among those individuals who had been treated with a ChEI compared to individuals who had not yet started this treatment. These findings are preliminary but may have important implications for helping drivers with dementia maintain safe performance of this critical activity of daily living; however, the effect of ChEI upon actual driving habits has yet to be examined.

Cessation of Driving

In many cases, healthcare providers will need to recommend cessation of driving to their older patients. Cessation of driving has a number of serious and negative consequences linked to decreased community participation and increased risk of long-term care placement. Therefore, providers should be aware of alternate transportation options in the community such as the RIDE and para-transit for elders who cannot use public transportation. Other alternate forms of transportation may include friends, family members, religious groups, social clubs, or other volunteer programs. For those patients who cease driving, at follow-up providers should assess for regular attendance of appointments, renewal of prescriptions, as well as signs of depression, isolation, and other forms of self-neglect due to cessation of driving.

Conclusions

As the older population increases, clinicians in the primary care setting will increasingly be called upon to evaluate driving safety. Although research supporting an evidenced based approach to identifying and remediating unsafe older drivers is lacking, there are increasing recommendations to inform the primary care practitioner and help make this determination.

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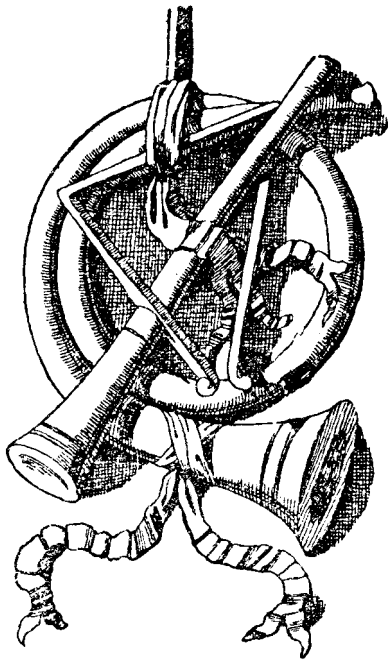


Family Caregiving During Healthy Aging and Illness

Gary Epstein-Lubow, MD

Family caregivers are inextricably nested in health care. Upon return home after an important physician visit, a patient may encounter questions from a spouse or other loved one. For many patients, family members provide direct accompaniment to the physician and actively participate in informed consent and other clinical decisions; for some, the caregiver is a designated legal proxy decision-maker. There is variety in how these patient-caregiver relationships form and develop over time; some individuals clearly identify who will help long before the help is needed whereas others are forced to recruit family caregivers amidst an unexpected and urgent crisis. Be it routine, a crisis or advanced care planning, there are always opportunities to enhance the process of patient, caregiver and clinician working together.

The **Institute for Healthcare Improvement (IHI)** proposes that all initiatives to enhance quality should concurrently address three facets of a "Triple Aim," including: "better care for individuals, better health for populations, and lower per capita costs."[1] In Rhode Island, estimates suggest that there are over 100,000 individuals providing substantive unpaid family caregiving, translating into millions of dollars of free health care to the seriously ill. Any quality initiative targeting an illness, such as post-stroke rehabilitation, or an environment, such as hospital service use, must attend to the role of the family caregiver as an essential component of improving health care quality. The question is: what opportunities currently exist for better integration of family caregivers into routine health maintenance and acute patient care settings?

Enhancing patient care by incorporating family caregivers must address the public health context of caregiving,[2] including health risks for family caregivers, and caregivers' instrumental needs. There is growing concern that the chronic stressors associated with protracted family caregiving pose health risks. Specifically, Family caregivers report worse health than non-caregivers,[3,4] engage in fewer health-promoting behaviors[3] and have worse medication compliance.[5] In 1999, Schulz reported a 63% higher mortality risk for strained spousal caregivers.[6] In other studies, caregiving for a hospitalized patient has been shown to be an independent risk factor for caregiver death; and, dementia caregivers have the highest risk,[4] possibly due to poor self-care combined with distress while faced with dementia-related behavioral problems in care recipients.[7] The majority of family caregivers assisting an individual hospitalized for behavioral disturbance associated with dementia report depressive symptoms suggesting clinical-level severity.[8] Based on data such as these, in their 2007 publication "Framing the Public Health of Caregiving," Talley and Crews propose a triadic model to conceptualize the role of the family caregiver as an equal and necessary collaborator.[2] In this model, the care recipient, the family caregiver and the clinician are partnered within a healthcare context constrained by limitations imposed by the illness, regional practice patterns, professional reimbursement and other system-level factors. This triadic model may help shape future public health attention specific to caregivers.

Empirical tests of the risks and potential benefits of strategies to build a stronger patient-caregiver-clinician alliance are on the near horizon.

How might the triadic model of patient-clinician-caregiver be actualized? Most clinicians likely see several family caregivers each day and devote a significant portion of patient-contact time to interacting with caregivers. Amidst these encounters, the clinician may informally note the family caregiver's functional capacity, current stress, and risk for succumbing to excessive burden or symptoms of depression.[9] While completing this informal assessment, the clinician might suggest new services for the caregiver and may also complete a brief chart notation as a reminder to follow-up regarding family resources at the next patient encounter; in addition, there are other methods by which the patient-clinician-caregiver alliance can be fostered.

Opportunities exist in both outpatient and hospital practice to conduct a formal assessment of caregiver health and functioning. In the outpatient setting, a screening measure for depression or burden can be completed prior to the clinician encounter;[10] the results of this screening tool can be reviewed by the clinician and incorporated into plans for providing the family caregiver with instrumental assistance, such as home care, or individualized clinical follow-up specifically for the family caregiver, such as a visit to primary care or a referral to social work or psychiatry if indicated. In the hospital setting, to complement the functional and mobility assessments that patients may complete in order to plan discharge services to home care or skilled residential treatment, similar tools can be used with family caregivers to determine whether the home environment is suitable for in-home nursing or physical therapy; such assessments could lead to improved aftercare matching and increased potential to transition patients directly home with enhanced support rather than through nursing home treatment. More importantly, a hospital-based caregiver assessment might prevent discharge to a homecare environment currently unprepared for success with such treatments; avoiding such a conflict could prevent excessive stress to the family caregiver and also reduce the risk of patient rehospitalization.

Increased educational opportunities are an alternative to completing formal or informal caregiver assessment during inpatient and outpatient clinical encounters. Education can build solidarity between patient, clinician and caregiver if opportunities are provided for the patient and caregiver to operationalize the information.

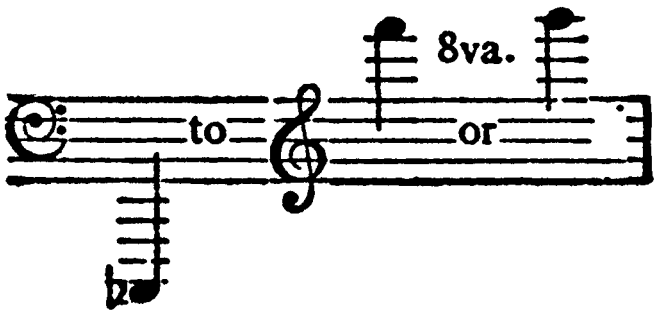
For example, if upon admission to a new outpatient program or hospital care environment, the patient and family caregiver are provided with an education brochure *plus* notification of a designated time at which the information will be reviewed in detail with a clinician; then, an iterative process can develop, leading to enhanced communication and care coordination.

Empirical tests of the risks and potential benefits of strategies to build a stronger patient-caregiver-clinician alliance are on the near horizon. Already there have been substantial efforts dedicated to improving post-hospital care transitions for patients at risk for rehospitalization due to illness such as congestive heart failure or hip fracture. This transitions intervention research has led to best practices that are now being adopted by the **Centers for Medicare and Medicaid Services (CMS)** as well as private insurance carriers, and mandated use of quality indicators are being written into new hospital contracts. Meanwhile, there is a growing spotlight on vulnerable patients who, due to cognitive impairment or other frailty, are less able to self-advocate during aftercare transitions. For these populations, there has been relatively sparse empirical testing of transition programs; however, new announcements from CMS and elsewhere are placing renewed emphasis on family caregivers as partners in quality improvement initiatives, particularly for vulnerable patients at risk for high utilization of health services.

Rather than wait for government entities and private insurers to issue guidelines to improve coordination of service use, several illness-specific advocacy groups such as the **American Cancer Society (ACS)** are developing new models now that enhance patient care by building patient-caregiver-clinician alliances. One such example is the Patient Navigator Program developed by ACS; this initiative seeks to match every newly-diagnosed cancer patient with a specially-trained clinical "patient navigator." The navigator's job is to provide education, instrumental aid for communication with key clinical services, and emotional and cognitive support as the patient adjusts to the cancer staging and treatment process. The navigator seeks to complete these tasks in coordination with family caregivers; for those patients without a family caregiver to assist with instrumental support, the navigator accepts added responsibility.

When clinical outcomes are positive and a family caregiver is able to "retire" from a formal caregiver role and return to being spouse or adult child, there can be both relief and satisfaction. Unfortunately, many illnesses are progressive and caregiving responsibilities persist into the last chapters of a patient's life. For some caregivers, entering bereavement is a relief whereas for others the death of the family member brings new unforeseen challenges regarding return to their own life anew. For clinicians who provide end-of-life care, there are opportunities here as well to promote caregiver health by encouraging caregiver self-care in anticipation of the terminal illness phase and after death.

In summary, there are many opportunities to enhance patient care by formally strengthening triadic patient-caregiver-clinician communication. Success with such endeavors has the potential to reduce health risks for caregivers, improve the quality of patient care, and result in lower health delivery costs. While awaiting the empirical testing of new models, there are steps clinicians, patients and family caregivers can take today. These steps include increased attention to educational materials for family caregivers, formal assessment of caregiver functioning prior to key role changes, and attention to developing new opportunities for caregivers and patients to collaboratively discuss care and treatment with clinicians.



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Rhode Island Children's Hospital Emergency Department Visits for Oral Health Conditions, 2006 – 2010

Junhie Oh, BDS, MPH, Laurie Leonard, MS, Erin Walsh, and Deborah Fuller, DMD, MS

Hospital emergency departments (EDs) provide critical and highly-demanded services to communities, including treatment for emergency oral/dental problems. EDs also serve as dental safety net points of access for a significant number of low income and uninsured Rhode Island children and adults who have limited access to oral health care due to lack of dental insurance, immigration status, or a number of other reasons. However, reliance on the ED for less severe, or non-emergent oral/dental conditions results in significant health care spending and increased pressure on the already crowded and overburdened EDs throughout the State.

Despite the fact that most children's dental problems are preventable with age-appropriate and effective disease management through regular dental visits, significant numbers of children experience dental decay. According to the National Health and Nutrition Examination Survey, more than a quarter of US young children age two to five years, more than half of the children age six to eight years, and 60% of adolescents 12 to 19 years had dental caries in the period of 1999–2004.[1] Similarly, the 2010-11 Rhode Island Third Grade Oral Health Survey found about half of Rhode Island third graders have experienced dental decay.[2]

Evidence suggests that regular preventive dental care visits beginning in early childhood can reduce the need for restorative and emergent care, particularly for children at high risk of developing dental caries.[3] However, regular preventive dental care is not equally accessible for all children. Parents may bring their children to the ED for non-urgent or traumatic dental/oral health concerns. For children and families without a dental home and/or an affordable source of dental care, EDs are the last resort to obtain dental care.[4] However, most non-traumatic and non-urgent dental care needs are more adequately addressed and treated in primary outpatient dental offices or clinics.[5,6]

The objectives of this report are to (a) document the extent of Rhode Island children's hospital ED visits for oral/dental conditions that are mostly preventable and treatable in primary care settings; (b) assess ED visits by children's age, insurance status, and primary diagnosis; and (c) discuss how to assure optimal and regular dental care for all Rhode Island children and decrease unnecessary hospital ED visits.

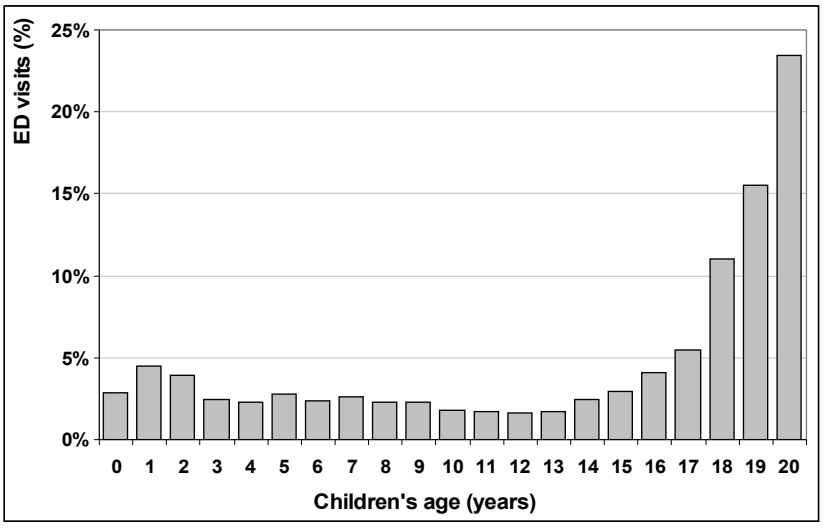
Methods

The data used for this analysis were obtained from the Rhode Island **Hospital Discharge Database** (**HDD**). Since 1989, Rhode Island hospitals are required to submit financial and statistical data using the statewide uniform reporting system to the Rhode Island Department of Health pursuant to their licensure authority.[7] Data on hospital inpatient and ED encounters are submitted by all 14 Rhode Island non-federal acute-care and specialty hospitals. HDD provides information on patient demographic characteristics, insurance, hospital admission and discharge related details including admitting diagnoses and clinical procedures rendered.

Data extracted and summarized for this report were all ED visits between January 1, 2006 and December 31, 2010 for children (20 years old and younger) with primary admitting diagnoses related to oral/dental conditions (i.e., ICD-9-CM codes of 520.0–529.9) that did not result in hospital admission. Children under 21 years of age were included to align with the age eligibility covered by Rhode Island Medicaid, which provides dental benefits for eligible children through the **Early Periodic Screening, Diagnosis, and Treatment** (**EPSDT**).

Using SAS® v9.3, descriptive statistics of the ED visits were generated by children's age, insurance type (or expected source of payment identified in hospital's initial admission records), and primary diagnosis.

Figure 1. Children's visits to EDs at Rhode Island hospitals for oral/dental conditions by children's age in years, RI Hospital Discharge Data 2006–2010. (Total ED visits = 5,460)



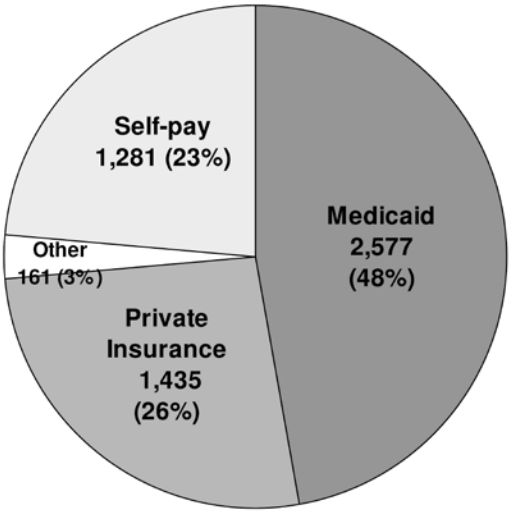
Results

From 2006 through 2010, 5,460 children's visits to the EDs at Rhode Island hospitals were primarily attributed to oral/dental conditions (identified with primary admitting diagnoses of ICD-9-CM codes 520.0–529.9). Noticeable trends or differences in the ED encounters by year were not observed over the five-year period.

Figure 1 depicts the oral/dental condition-related ED visits by children's age. Older children were the most frequent ED users; children age 18–20 years, combined, accounted for half of ED encounters (18 years: 11%, 19 years: 16% and 20 years: 23%).

Medicaid (RIte Smiles or Medicaid fee-for-service) was the most common payment method for oral/dental complaints in the EDs, accounting for approximately half of all the ED visits (48%, Figure 2). Visits by children who were privately insured, and under- or un-insured children (whose payment sources were identified as "self-pay") accounted for 26% and 23% of ED visits, respectively (Figure 2).

Figure 2. Children's visits to EDs at Rhode Island hospitals for oral/dental conditions by expected payment source*, RI Hospital Discharge Data 2006–2010. (Total ED visits = 5,460)

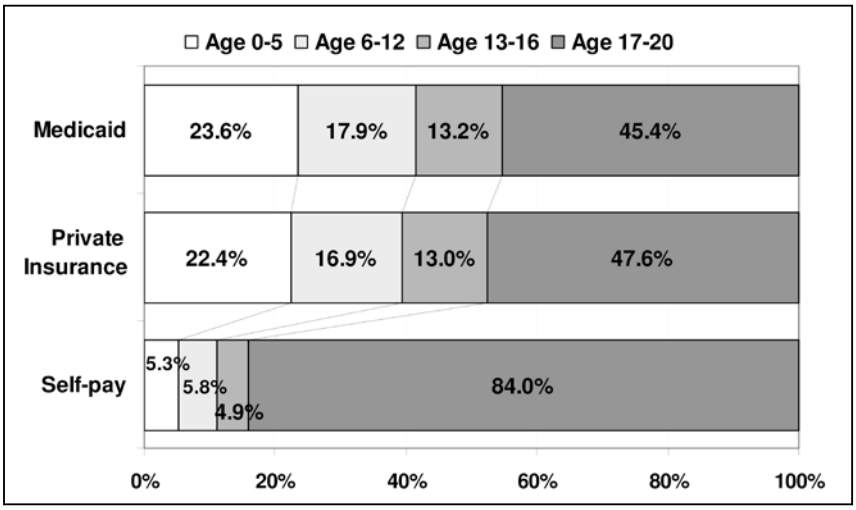


* The payment source information is based on the "expected" source of payment identified in hospital's initial billing records. Final and actual payer may change and are not necessarily the same as the "expected" payer.

Figure 3 summarizes the ED visits by child's age (categorized as 0–5, 6–12, 13–16, and 17–20 years) for the three major expected payors: Medicaid, private insurance, and self-pay. ED visits by children age 17–20 years occurred most frequently for all types of payors. No difference was observed in children's age distribution between Medicaid and private insurance coverage. Most of the children reported as under- or un-insured were within the oldest age group (17–20 years).

Table 1 shows the distribution of ED primary admitting diagnosis related to oral/dental conditions. A third of these primary diagnoses were dental caries or inflammatory pulp and periapical lesions originated by tooth decay (ICD-9-CM codes 521.00–521.09 and 522.0–522.9: 32%). Less specific conditions recorded as "unspecified disorders of the teeth and supporting structure", such as toothache of undefined cause (ICD-9-CM codes 525.8 and 525.9), comprised 30% of the primary diagnoses.

Figure 3. Children's visits to EDs at Rhode Island hospitals for oral/dental conditions by age group and expected payment source, RI Hospital Discharge Data 2006–2010.



Discussion

Many children (under age 21 years) sought care at Rhode Island hospital EDs for acute signs and symptoms of oral health problems that are mostly preventable, given access to earlier and optimal dental care. ED use for preventable oral/dental disease is a significant public health problem. EDs typically offer only temporary relief of pain and palliative care that may require return visits or further dental services.[8] Because EDs are not equipped with the resources to offer definitive diagnosis and treatment for oral/dental conditions, patients usually must seek alternate follow-up care elsewhere to receive more appropriate dental services, resulting in delay of needed treatment. Significant numbers of "unspecified" primary diagnosis reported here can be explained by the fact that a majority of cases were diagnosed by ED physicians or nurses who had not been trained to offer appropriate dental counseling or services. Authors could not evaluate specific treatments rendered to resolve oral/dental complaints in EDs, due to incomplete record keeping of clinical procedural codes in the database. An empirical study showed that most of the pediatric patients presenting for ED dental treatments received only symptom-relieving treatments, such as prescriptions of analgesics or antibiotics.[9]

This analysis of Rhode Island HDD found Medicaid to be the most common payor for children's ED visits for oral/dental conditions within the study period of 2006-2010. The predominance of Medicaid patients seeking care for non-emergent or traumatic dental/oral conditions at Rhode Island EDs suggests that (1) dental problems are more prevalent and severe among children from low-income families, and (2) children with Medicaid are less likely to obtain preventive and restorative dental care than those with private insurance coverage.[10–13] Postponing needed dental care may lead to an ED visit if a patient's disease progresses to a more complex condition.

Table 1. Children's visits to EDs at Rhode Island hospitals for oral/dental conditions by primary diagnosis (ICD-9-CM Codes), RI Hospital Discharge Data 2006–2010. (Total ED visits = 5,460)

Primary Diagnosis	ICD-9-CM code	Number of visits	Percent
Dental caries, pulpitis and periapical lesions	521.0, 522	**1,745**	**32.0%**
Unspecified disorders of the dental/supporting structure	525.8, 525.9	**1,595**	**29.2%**
Soft tissue and tongue lesions	528, 529	**688**	**12.6%**
Disorders of the TMJ/jaw and malocclusion	524, 526	**503**	**9.2%**
Gingival and periodontal lesions	523	**439**	**8.0%**
Other (including eruption/tooth development anomaly, erosion, abrasion, tooth loss, cracked tooth, restoration fracture, etc.)	520, 521.2-521.8, 525.0-525.7	**288**	**5.3%**
Diseases of the salivary gland	527	**202**	**3.7%**
Total		**5,460**	**100%**

ED utilization was particularly concentrated within the adolescent ages, even among those with Medicaid coverage. Since the implementation of RIte Smiles (Rhode Island's Medicaid dental managed care program) in 2006, significant gains in access and utilization of preventive and treatment dental care among Medicaid-enrolled children age ten years and younger have been reported.[14] However, adolescent children born prior to May 1, 2000 are not covered by the RIte Smiles program. Children over age 12 are currently covered by traditional fee-for-service Medicaid, which has a different reimbursement/fee schedule and benefit structure. More efficient use of Medicaid dental benefits for adolescent children should be considered to better coordinate these older children's oral health needs and promote preventive and regular dental care in primary dental care settings. These efforts would help reduce emergency dental care treatment needs and generate Medicaid cost-savings by reducing the provision of more expensive dental care at hospital EDs.[15]

Most of the children reported as under- or un-insured were adolescents who were most likely lack of access to a regular source of oral health care. Public and private dental insurance that is more affordable and includes an expanded scope of dental benefits would allow more children to access routine dental care.

EDs provide crucial safety net dental access to a significant number of low income and uninsured Rhode Island children who have limited access to oral health care. The reliance of Rhode Island children on EDs for preventable, or non-emergent oral/dental conditions should be addressed by policy makers and oral health advocates to ameliorate significant health care spending and increased pressure on the overburdened hospitals, insurers and patients throughout the state.

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The authors and/or their spouses/significant others have no financial interests to disclose.

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Images In Medicine

Eastern Equine Encephalitis: MRI Findings In Two Patients

Matthew Ethier, MD, and Jeffrey Rogg, MD

A PREVIOUSLY HEALTHY 20 YEAR-OLD MALE PRESENTED TO THE Rhode Island Hospital Emergency Department with 24 hours of worsening abdominal pain, vomiting, and fevers. On physical examination he appeared diaphoretic and febrile, with an oral temperature of 104.3°. The patient was noted to have tenderness to palpation in the epigastric region. Initial laboratory evaluation revealed a white blood cell count of 23,000/µl. A CT scan of the abdomen and pelvis was normal, with no evidence of appendicitis as was suspected clinically. The patient was started empirically on IV antibiotics and admitted for observation. On the first night of admission, the patient developed a severe headache, with subsequent rapid deterioration of his mental status. Status epilepticus soon followed. He was intubated for airway protection and transferred to the Medical Intensive Care Unit.

A non-contrast CT scan of the head showed diffuse cerebral edema and effacement of the basilar cisterns. Brain MRI performed shortly thereafter revealed abnormal T2 and FLAIR signal hyperintensity involving the bilateral thalami in a symmetric manner, as well as the left external capsule and tegmentum pons (Figures 1, 2, and 3; white arrows). Prominent perivascular congestion was also noted (Figure 4; white arrows). No associated restricted diffusion or post-contrast enhancement was seen in these areas.

Due to diffuse cerebral edema and cisternal effacement a lumbar puncture could not be safely performed. A small amount of CSF obtained from placement of a ventriculostomy shunt yielded negative Herpes Simplex Virus (HSV) 1 and 2 PCR analysis. The patient expired 3 days after admission.

One month later, a 21 year-old male was transferred to the Neurology Intensive Care Unit at RIH from an outside hospital after being intubated for status epilepticus. The patient initially presented with 24 hours of headache and vomiting, and was febrile to 103.8°. His course was rapidly progressive, with mental status deterioration to the point of obtundation and onset of seizures beginning less than 12 hours after admission to the outside facility. His history noted exposure to mosquitoes while golfing in southeastern Massachusetts seven days prior to admission.

An initial brain MRI at the time of admission revealed abnormal T2/FLAIR signal hyperintensity in the mesial temporal lobes bilaterally in a pattern suggestive of viral encephalitis, possibly HSV based on these imaging findings. However, CSF analysis revealed negative HSV 1 and 2 PCR, in addition to negative gram stain and culture. The patient showed only

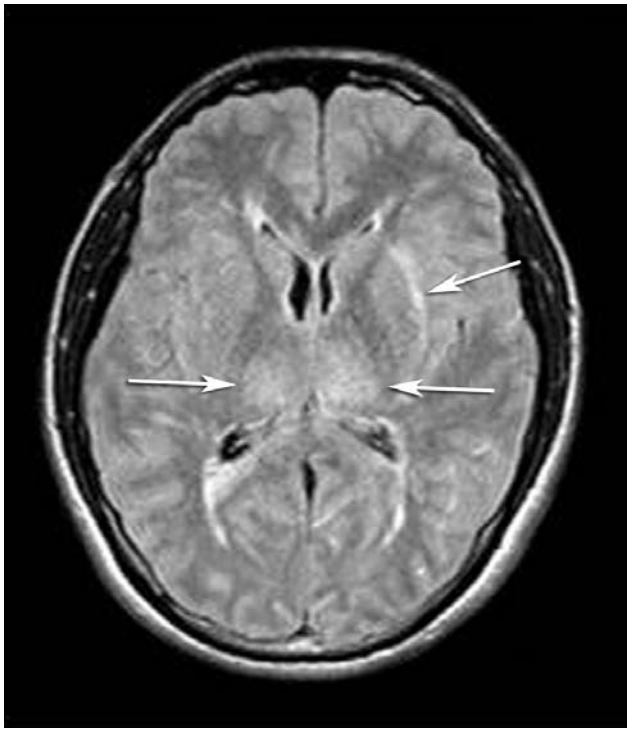
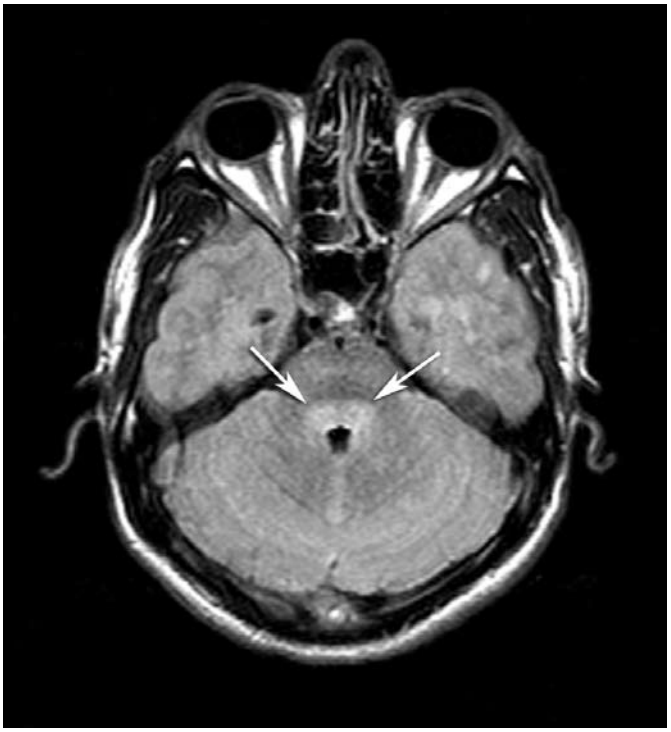


Figure 1.



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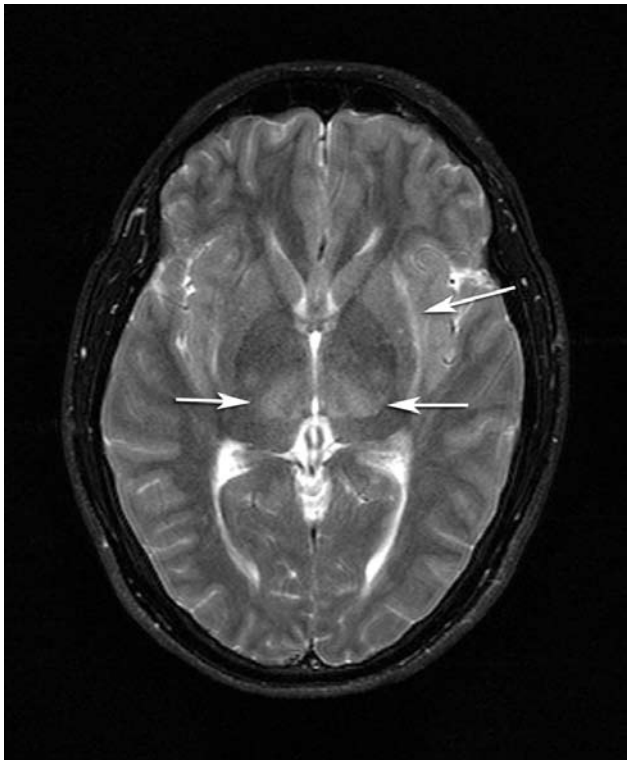

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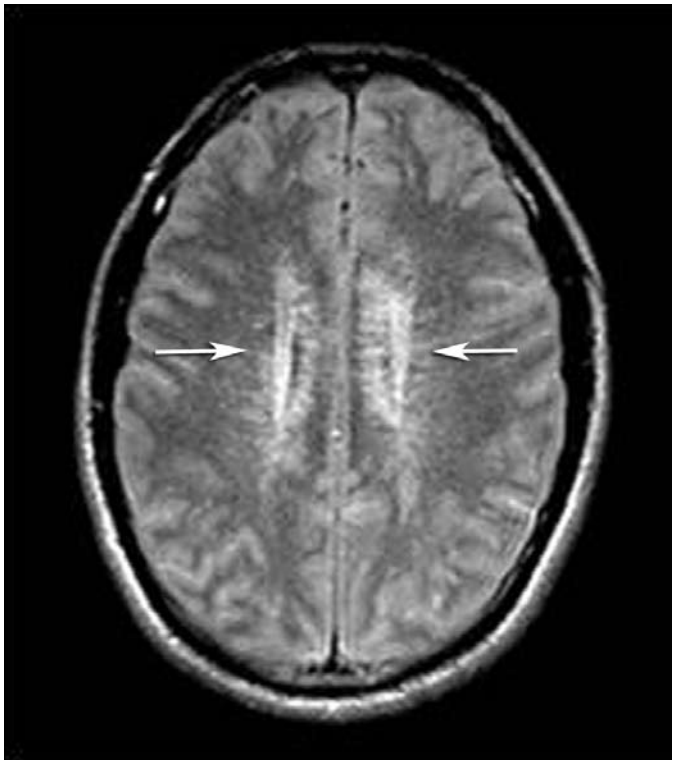
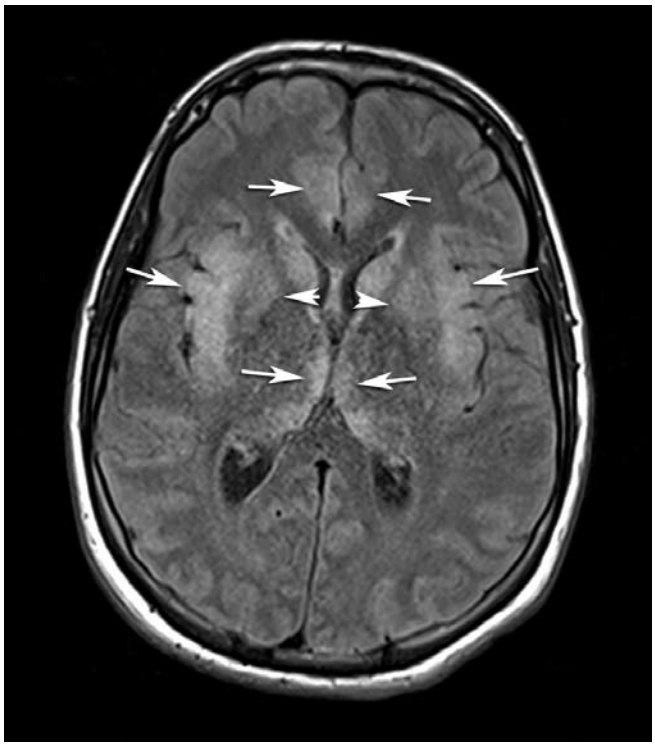


Figure 4.

minimal improvement clinically while on anti-viral therapy, so a subsequent brain MRI was performed 10 days later. This showed abnormal T2 and FLAIR signal hyperintensity within the pulvinar nuclei of the bilateral thalami, the medial aspects of the thalami, the bilateral insular cortex, bilateral putamina, and inferomedial frontal cortex bilaterally (see Figures 5 and 6; white arrows). Repeat CSF analysis performed within 24 hours of the second brain MRI showed negative gram stain, culture, and negative HSV 1 and 2 PCR analysis, but anti-EEE IgM anti-body were positive. Anti-EEE IgG anti-body was negative. In addition to anti-viral therapy and steroid administration, this patient also received IVIg therapy. His neurologic status



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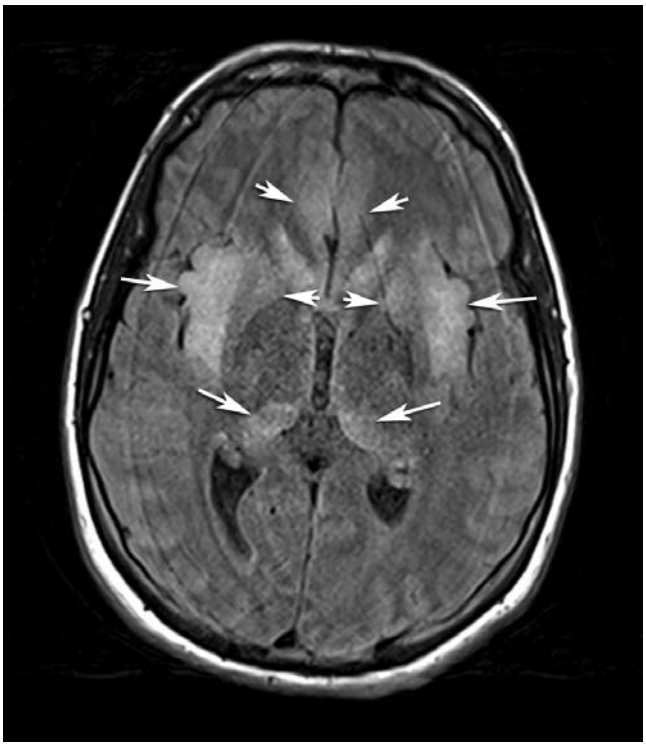

Figure 6.

improved gradually over the course of the admission and he was eventually discharged to a rehabilitation facility.

Eastern equine encephalitis (EEE) is an illness caused by the mosquito-borne arbovirus *Alphavirus togaviridae* which occurs predominantly along the East and Gulf coasts of the United States. Although only about 5% of infections lead to EEE, morbidity and mortality are high with 33-36% of affected patients dying from the illness.[1] In a study of the clinical and imaging manifestations of EEE performed by Deresiewicz et al in 1997, only 1 of the 36 patients with EEE made a full recovery.[2] The early presentation of EEE can be similar to any common viral illness, with symptoms of fever, headache and abdominal pain. Rapid deterioration of neurologic status and seizure activity then occurs due to severe encephalitis, often despite anti-viral and steroid therapy. There is limited evidence suggesting that IVIg may be of some therapeutic value.[3] Definitive diagnosis is made by CSF or serum analysis for anti-EEE IgM and IgG antibodies. While EEE titers were positive in the second case, there was an insufficient quantity of CSF to send for EEE titers in the first case, though this entity became the leading diagnostic consideration based on the imaging findings and the patient's rapidly progressive course. While encephalitis from herpes simplex virus can have a similar clinical course to EEE, the MR imaging findings can aid in distinguishing this entity from EEE. The basal ganglia, brainstem and bilateral thalami demonstrate abnormal T2 signal on MRI early in the course of EEE. These areas are often spared completely in HSV encephalitis or become involved only later in its course. In both of these cases, the imaging patterns were most consistent with EEE rather than HSV infections.

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Disclosure of Financial Interest

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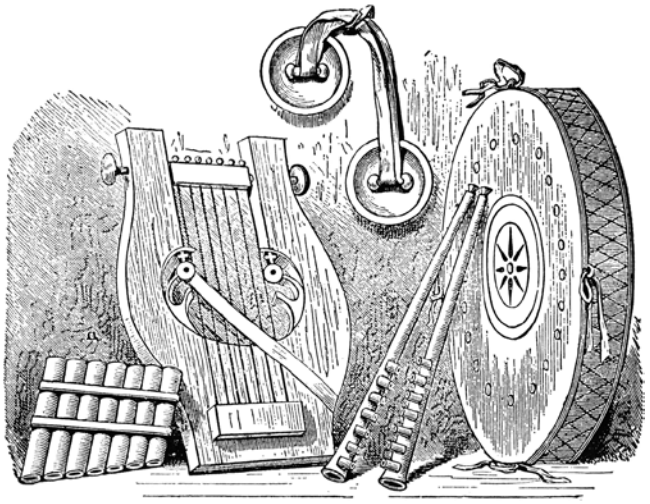

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Contributions report on an issue of interest to clinicians in Rhode Island: new research, treatment options, collaborative interventions, review of controversies. Maximum length: 2500 words. Maximum number of references: 15. Tables, charts and figures should be submitted as separate electronic files (jpeg, tif, or pdf). Each submission should also be accompanied by a short (100-150 words) abstract.

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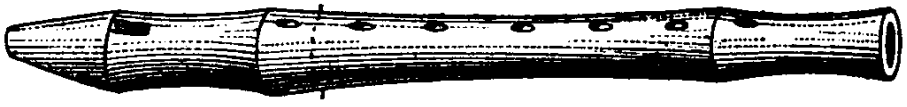

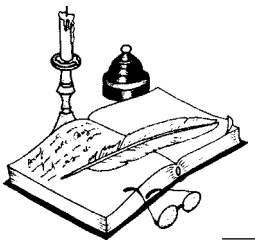

Physician's Lexicon

The Generous Words of Heredity

In the beginning was Genesis, the inaugural book of the Scriptural Pentateuch. The first word of the book, *bereshith*, (Hebrew, meaning "in the beginning"), was then translated to the Greek, *genesis*, which had earlier been derived from the Greek *genos,* meaning producing or coming into being, and still earlier from *gignesthai,* meaning to be born. And in the millennia since, there has been an abundance of words based upon, *gens,* the Latin descendant of the Greek, *genesis*, now meaning a tribe, a clan or a race.

At one philologic extreme is the current word, genocide, meaning the extermination of an ethnic group, a term newly devised by Allied jurists in 1944 to define the Holocaust. The *–cide* root is from the Latin, *caedere*, meaning killer (as in words such as suicide, fratricide or homicide.)

In general, the many descendants of the Indo-European roots *gen-, gon-,* and *gn-* have all conveyed the sense of "that which produces" or, "that which comes to pass."

The Latin, *gens*, has specifically given birth to a plethora of English words such as genus, gender, generation, generic, eugenics, generous, genus, genie, ingenious and genius. Yet another brood of derivative words, often via Old French, include ingénue, gendre, genteel, gentile, gentle, and gentry (and even jaunty).

The *gon-* and *gn-* roots have taken on a somewhat male meaning as in the word, gonorrhea, literally meaning "the flow of semen", with the Greek suffix, *-rrhea*, meaning to flow (as in words such as sialorrhea, diarrhea or rhinorrhea.) Medical terms employing the *gon-* root include gonocele (a cyst of the epididymus), gonococcus, geneogenous (congenital, meaning, "derived from the parents") and gonocyte (primordial germ cell). The *gn-* root should not be confused with words employing the Greek root, *gnathos-*, meaning pertaining to the jaw, as in gnathic, gnathalgia (painful jaw), gnathology (the science of jaw dynamics) and even the English verb, gnash.

The current English vocabulary is sprinkled generously with additional words that harken back to *gens*, including: genealogy, genes, genitive, genitals, gendarme, ingenuity and ingenuous.

– Stanley M. Aronson, MD

Rhode Island Department of Health
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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			
	July 2011	12 Months Ending with July 2011		
	Number (a)	Number (a)	Rates (b)	YPLL (c)
Diseases of the Heart	179	2,418	229.6	3,532.5
Malignant Neoplasms	229	2,274	215.9	5,611.5
Cerebrovascular Diseases	35	450	42.7	724.5
Injuries (Accidents/Suicide/Homicide)	56	660	62.7	9,587.5
COPD	33	551	52.3	432.5

Vital Events	Reporting Period		
	January 2012	12 Months Ending with January 2012	
	Number	Number	Rates
Live Births	917	11,695	11.1*
Deaths	882	9,642	9.2*
Infant Deaths	(7)	(77)	6.6#
Neonatal Deaths	(6)	(58)	5.0#
Marriages	182	6,245	5.9*
Divorces	309	3,393	3.2*
Induced Terminations	327	4,104	350.9#
Spontaneous Fetal Deaths	55	631	54.0#
Under 20 weeks gestation	(46)	(534)	55.4#
20+ weeks gestation	(9)	(95)	8.1#

(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 1,052,567. (www.census.gov)

(c) Years of Potential Life Lost (YPLL).

Note: *Totals represent vital events that 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

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Ninety Years Ago, July, 1922

P.E. Truesdale opens this issue with an article cancer of the stomach with a report of end results in 65 cases. Noting that stomach cancer is often more insidious than other forms of cancer, detection is crucial—particularly in getting patients to accept operations early. Detection gets easier as patient awareness of symptoms grow. The situation is far from perfect, however. The problem isn't so much surgery, but lateness—lateness in warnings, recognition and in timing of operation.

Roy Blosser, MD, discusses diagnosing diseases of the scalp. While common, the medical profession as a whole tends to not give it much attention. Consequently, patients who suffer from these diseases usually drift around from one to another of the self-constituted hair specialists, beauty parlors, barber shops, and such. The author goes on to outline various conditions from hair loss to psoriasis of the scalp, neurodermatitis, and sycosis barbae.

Accepting the presidency of the Rhode Island Medical Society, Frank E. Peckham, MD, addresses the membership. Among his remarks, he states: "To me the profession of medicine is a big challenge, composed of very many smaller of sub-challenges. The men making up this grand profession are men of varying ability. As each man develops his particular bent, he naturally fits in somewhere, because there are so many avenues in which he can accept the challenge which most appeals to his personal characteristics."

An editorial observes that with the recent opening of the Jane Frances Brown Building, Rhode Island Hospital has been placed among the first in the eastern United States in the way of modern hospital facilities. It is also observed that the obstetrical ward, an entirely new departure of the hospital, lends added attractiveness to the general hospital facilities.

Fifty Years Ago, July, 1962

In the Caleb Fiske Prize Essay for 1961, Lester L. Vargas, MD, discusses the current status of cardiac surgery. In the wrap-up, the author states: "Clinical cardiac surgery, only a little more than twenty years old in America, has progressed to include operations which once seemed impossible. Its present status has been reached through the development of physiological concepts and apparatus which have made open-heart surgery a reality. Hypothermia induced with a heat exchange has added to the safety of extracorporeal circulation. Cardiac arrest and profound hypothermia provide a relatively dry, motionless, operative field. Under these optimum operating conditions, it is now technically possible to correct a number of complex intracardiac lesions. This progress has been so rapid that merely keeping up with events has been likened to the dilemma of the Red Queen in Alice's dream who had to run as fast as she could only to stay in the same place."

Boating and boating hazards are addressed in a editorial that makes reference to Coast Guard guidelines, but finishes with a sensible call to common sense. Don't overload; be sure there is a life preserver for everyone aboard; don't overlook an adequate first aid kit; and don't venture off shore unless you know how to plot a course and have the latest weather information. A little courtesy and common sense will keep boating accidents to a minimum.

Continuing with a theme on summer pastimes, another editorial looks at swimming pools. After waxing poetic on the idyllic aspects of swimming pools, the topic turns toward such hazards as staphylococcus infections, impetigo, otitis media, broken necks, and drowning. What steps can be taken? The RIDOH will furnish a copy of their swimming pool rules and regulations upon request, and the Division of Sanitation will advise anyone with a swimming pool on the best methods for maintaining it in a sanitary condition. In addition, it's suggested that one know the swimming pool being used, and who maintains it. Gate locks, life-saving rings, reaching poles and safe walkways are also mentioned. Likewise, a safe pool is a clean pool.

In an article by John B. Lawlor, MD, Roger G. Berard, MD, and Ernest K. Landsteiner, MD, entitled "Acute Renal Failure Complicating Salicylate Intoxication: Role of the Artificial Kidney," the authors discuss various case report related to the title and conclude: "Severe acute salicylate poisoning responds favorably to early dialysis with the artificial kidney. Subsequent renal failure caused by the nephrotoxic effect of salicylates, although it occurs but rarely, is a grave complication with a poor prognosis. A case of acute salicylism complicated by oliguric renal failure with complete recovery is therefore reported."

Twenty-five Years Ago, July, 1987

Jacques G. Susset, MD, discusses the challenge of assessing and treating female incontinence and stresses the need for a complete urodynamic evaluation preceding intervention. "A physio-pathological assessment of the factors involved in female incontinence is now possible through a thorough urodynamic evaluation which appears to us essential for the understanding of the underlying condition. Such an approach should increase the accuracy in the choice of treatment. This in turn leads to improvement of the therapeutic results and many times to conservative management."

In light of the Challenger space shuttle disaster, Shawn Cooper, PhD, MPH, suggests that a strategy should be developed for dealing with children and traumatic national events. He reports on a survey sent to pediatricians which supported the idea for trauma counseling both through families and in schools. The survey "implies that the medical community, particularly that part of it which deals with children, may need to develop a general strategy for responding to adverse reactions to traumatic national events, however rare they may be."

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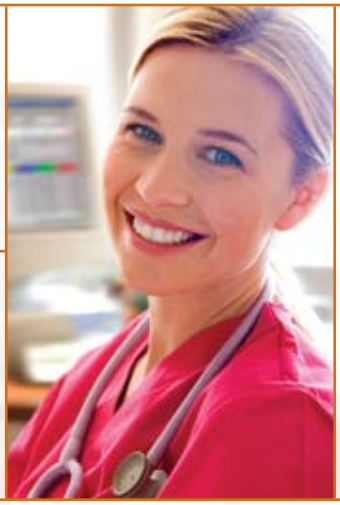

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