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Medical Conferences

I have had a long love-hate relationship with national and international neurology conferences and haven’t attended any in a while. Of course they’ve become more expensive, at a time when money is tighter than it used to be, which makes the decision to not go easier, but I wonder how much I’m losing touch. I reviewed a list of fellowship programs in my subspecialty and realized that I didn’t recognize almost half the names of the fellowship directors, and not because the number of programs has expanded. They’ve actually contracted as money has dried up. I used to know every director. Youth will be served, as it should be.

I read an article recently that was a complete surprise, reporting that one Parkinson’s disease (PD) medication reversed a particular and vexing side effect of another PD drug from a different chemical family. Since I use a lot of both drugs, and there is no current theoretical rationale for thinking the first drug would “cure” the second’s side effects, I don’t fault myself for not thinking of this “off label” use of the drug, nor do I kick myself for not having observed this unexpected benefit. Once the side effect of drug two was recognized, I would have advised against adding the first drug, rather than advising to add it.

I then wondered how many of my colleagues had known of this potential benefit of the first drug, so I checked the paper to see if it had been presented at a conference I had missed. It hadn’t, but nevertheless it may have been discussed at many informal lunches, coffee clatches and other meetings that typically occur at these meetings.

I find that meetings are good for learning and making one feel more up-to-date. An article published in Neurology, the most widely read journal in our specialty noted that chart reviews of epilepsy surgery showed that since official guidelines had been created and widely disseminated among neurologists and pediatric neurologists, no change had occurred in actual practice. The guidelines were intended to increase the number of referrals for treatment of intractable epilepsy, since most patients had delays of over 10 years, many years after all reasonable drug trials had failed. It turns out that for those patients who are epilepsy surgery candidates the surgery is usually amazingly effective. People had been seizing daily while chronically intoxicated on multiple anticonvulsants become epilepsy free on a single medication. Of course they never get their wasted years back, but certainly there’s good reason to encourage our colleagues to keep up on the guidelines to keep these missed good years to a minimum. I’ll bet that the neurologists who did follow the guidelines were either epilepsy experts or attended the national meetings.

National meetings are good for these extremely important issues. It is important to get up-to-date. The problem I have, and I’ve never discussed it with my friends, is intimidation. I always return from a meeting feeling that I’m so far behind in my field and such an ignoramus that I’m dejected for weeks. A whole session will be devoted to some biochemical entity I’ve never heard of, an observation in genetics that seems to explain half the neurodegenerative diseases I’m familiar with and a good percentage of many I’d never heard of. My colleagues are presenting posters and platform presentations and I’m simply in the audience. The stuff I’ve just read in the recent journals is out of date by the time the conference takes place.

Of course things are supposed to change. And of course the rate of change is increasing with ever increasing speed. This is good. What is troubling is my relationship to it. I am much more commonly deflated than elated. I don’t come back home saying, “Wow, is this field exciting. The developments are astounding. We’re going to get to the bottom of Parkinson’s disease soon. Things are really going to take off.” No. I generally return glum, thinking not only that the situation is even more complicated than it was last year, and that I not only don’t understand last year’s “breakthroughs” but I don’t understand this year’s any better. It’s sort of like a fractal. The deeper you look, the more the complications don’t change.

On the other hand, I’m a clinician, and one of the tenets I hold to is that although we don’t get smarter with time, some of us do get wiser. We clinicians learn every day, even if we don’t think too hard. How can we not learn from every story, from each time we review a topic to treat our last patient better? Experience touched with enthusiasm and inquisitiveness make for the best clinicians (leaving aside all aspects of humanism and compassion). This is what I tell myself.

Yet, while I think the clinical experience crucial, and I use it to buttress my ego, I wonder about all these colleagues of mine, and how much they understand. And if they do understand what I don’t, are they better doctors than me? Do they provide wiser counsel?

I need to attend more national conferences. Perhaps it’s not only good for the intellect but good for the ego as well. One popular motivational speaker at Parkinson patient support groups tells the audience, “Ask your doctor questions. Take charge. Too many doctors think that MD stands for “Medical Deity.”

Well, a good conference makes me always doubt if I should even be a medical doctor, let alone a medical deity. Two weeks later though I think I’m better for it.

– J OSEPH H. FRIEDMAN, MD

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Conflicts: In addition to the potential conflicts posed by my ties to industry that are listed, during the years 2001-2009 I was a paid consultant for: Eli Lilly, Bristol Myers Squibb, Janssen, Ovation, Pfizer, makers of each of the atypicals in use or being tested.
The Beckoning Emergency Room

Data from the emergency departments (EDs) of this nation are becoming accurate barometers of our country’s health. To know when, where, and of what intensity a new strain of influenza might be infecting the nation, for example, check the intake registries of the EDs rather than rely upon anecdotal tales or letters to the editor. To appreciate the magnitude of the killing of innocents by firearms, go no further than the statistics emanating from our nation’s EDs. And to appreciate the extent to which Americans are fond of—perhaps even dependent upon—certain mood-altering drugs, check out the records of the EDs.

Before World War II, American hospitals created back doors for the acutely injured, sick and weary arriving by ambulances. And the same back door, perhaps identified by an illuminated sign, also beckoned the ambulatory seeking help. Typically, it was a small, 24-hour facility monitored by a single nurse with an intern or two on call. It was used principally during the late night hours when physicians’ private offices and the neighborhood pharmacies were closed. The acutely distressed, the injured, and sometimes the very lonely passed through these doors, generally past midnight. Their numbers were small, the rendered care was rapid, responding solely to the emergent problems, and the majority of those seeking aid were visibly injured. Only the most sophisticated EDs had access to such ancillary aids as radiography or clinical pathology laboratories.

The decades following the mid-20th Century witnessed an exponential growth in ED activity. Where formerly it might have been a single, utilitarian room (called the Emergency Room) for the most desperate of injuries, it now had evolved, by necessity, into a sanctuary treating the entire spectrum of medical ailments. It became a free-standing department with its own staff, diagnostic facilities, holding beds, emergency surgical suites, and for its physicians, even their own specialty accreditation.

By the 21st Century, the health of the nation had improved significantly; people were living longer and insurance contrivances such as federally-sponsored Medicare were bringing the benefits of rational care to a larger segment of the American population; yet, paradoxically, a greater fraction of the populace was now depending upon the EDs, not only for their unanticipated emergencies but for their non-emergent medical needs as well. A former president declared that the nation needed no further governmental medical insurance since there always were the EDs to meet the public’s need. Truly, the nation’s EDs were no longer the clinic of last resort but America’s primary care facility, a function that they were not designed to fulfill.

Given their competency in profiling America’s health problems, what can the EDs tell us about chemical addictions? The United States Public Health Service recently created a new surveillance facility, Drugs Abuse Warning Network (DAWN), to monitor the nation’s EDs in terms of drug-related morbidities incident to “the nonmedical use of prescription drugs” such as oxycodone, hydrocodone and methadone.

During the most recent five year interval (2004 – 2008) the EDs confronted swelling numbers of patients compromised by the unwarranted use of opium-like medications. In 2004, 144,600 such cases were recorded; by 2008 this number of patients reacting adversely to opioid drugs such as oxycodone had risen to 305,900 patients streaming into EDs.

Oxycodone is not a newly created analgesic. Chemists were seeking an opium-like medication with fewer side-effects than morphine or heroin, both of which were derived from crude opium. Oxycodone was synthesized by a German pharmaceutical laboratory in 1916, and was derived from another opium derivative called thebaine. Its unwanted side effects include dizziness, confusion, stupor (and paradoxically, sleeplessness), nausea, severe constipation and visual changes caused by constricted ocular pupils (miosis).

What is the profile of the population taking illicitly derived oxycodone? Adult women slightly more than adult males. And about two-thirds of those seeking ED help admitted to taking more than one pharmacologically-active drug. In addition, about 15% had also consumed measureable amounts of alcohol. The symptoms in about 24% were sufficiently serious to warrant immediate hospitalization.

The second most frequent medication prompting ED visits was from the excessive use of one or another of the benzodiazepines, a family of mood-altering agents including valium and ativan. Excessive side effects include dizziness, incoordination (with a tendency to falling), confusion, diminished libido and irrational behavior. An estimated 271,700 ED visits in 2008 were caused by benzodiazepine toxicity.

DAWN estimates that well over two million ED visits, per year, are the result of the illicit overuse of addictive medications. Of these, about one million were occasioned by chemical dependency on cocaine and heroin.

In the words of the USPHS, “These findings indicate substantial, increasing morbidity associated with the non-medical use of prescription drugs in the United States.”

— Stanley M. Aronson, MD

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Practicing nursing home medicine can be both rewarding and challenging. Nursing homes provide a low overhead, flexible, self-designed practice setting with varied, complex patients. With recent changes in the health care system, nursing homes have become places of increasing medical acuity, and are no longer only places of long term residential care. Increasing efforts by Centers for Medicare and Medicaid Services (CMS) to improve quality of care have led to exciting initiatives. The current 9th scope of work (SOW) emphasizes continued quality of care, with a focus on transitions of care with continued focus on patient safety, and chronic disease management and prevention. Rhode Island’s quality improvement organization (QIO), Quality Partners of Rhode Island (QPRI), works in close partnerships with Rhode Island nursing homes to reduce rates of pressure ulcers, reduce rates of restraints, improve medication safety, and promote seamless transitions across the spectrum of care.

This issue reviews some recent areas of interest and change in nursing home medicine. To start, the 2008 Center for Medicare and Medicaid Services (CMS) nonpayment rules drew attention to pressure ulcer treatment and prevention. Now firmly considered a preventable condition, regulators scrutinize the standard of care around pressure ulcer prevention and treatment. The article in this edition reviews physician responsibilities around pressure ulcer treatment and prevention. The article in this edition reviews physician responsibilities around pressure ulcer treatment and prevention, and summarizes risk factor modification, ulcer identification, staging, documentation and treatment.

Dementia prevalence is on the rise and is one of the most common conditions treated in the long term care setting. Many patients will ultimately reside in nursing homes or special care units due to the inevitable, progressive functional and cognitive decline associated with dementia. Behavioral symptoms are often the most challenging symptom for physicians, nurses and families to manage. Two articles will address the management of behavioral symptoms from both the pharmacologic and non-pharmacologic viewpoints.

The increasing recognition of dementia as a terminal illness has led to a more prominent role for and the push to further involve palliative care in the nursing home setting. An article discusses the indications for referral to palliative care, and the symptoms of advanced dementia that can benefit from palliative interventions.

Finally, nursing homes are subject to the same trends and outbreaks that affect the general population. Last year’s pandemic of H1N1 was felt among nursing home residents and often with higher rates of morbidity and mortality. The final article reviews the effect of both seasonal influenza and pandemic influenza on the older adult population.

Hopefully, this special edition provides useful information and updates for those who practice in nursing homes, and for those who care for older adults in the community.

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Mrs. X, an 80-year-old woman with a history of insulin-dependent diabetes, high blood pressure, dementia, and peripheral neuropathy, was admitted to a nursing home for rehabilitation following repair of a fractured left hip. Upon admission she was assessed for pressure ulcer risk using the Braden Scale. For a Braden Score of 14 (moderate risk), the nursing staff initiated a plan of care to prevent the development of a pressure ulcer. This included placement of a pressure redistribution mattress on her bed and a cushion in her wheelchair. Three months later Mrs. X had completed rehabilitation. She could ambulate with a rolling walker and she transitioned to long term care.

The following year, Mrs. X became more confused and her oral intake decreased; she lost 5 pounds. During a weekly skin assessment the nurse noticed a blister on her left heel, possibly caused by pressure to her heel from walking in a new pair of shoes or from lying in bed. The nurse documented the occurrence in the record and reported the blister, as well as the resident’s overall decline, to the resident’s daughter and to her attending physician. The physician ordered laboratory studies, including a urine culture and serum albumin, dietary supplements, a foam foot elevator, and a treatment regimen. A clinician can expect the physician may round every 30 days, and be available when a problem is identified, periodic reassessment for risk of PU development and address any risk factors. 

Management of Pressure Ulcers in Nursing Homes

Since in many nursing homes a physician may round every 30 days, and be unavailable when a problem is identified, excellent nursing assessment and concise reporting are crucial. Nurses are required to report any open area in order to initiate a treatment regimen. A clinician can expect the nursing home nurse to include the following:

- The patient’s name and age
- Vital signs
- Degree of mobility, presence of contractures
- The wound’s location – (using proper anatomical terms such as anterior, posterior, medial, lateral)
- The wound’s dimensions - (in centimeters, proximal to distal, medial to lateral)
- Condition of the surrounding tissue – (ex. hyperkeratotic rim, open area in order to initiate a treatment regimen)
- Appropriate documentation guarantees effective communication between clinicians and other members of the care team and allows for full accountability and retrospective review by all interested parties.

The Centers for Medicare and Medicaid Services (CMS) require that all nursing homes receiving Medicare dollars perform a comprehensive resident assessment and develop a care plan. Skin conditions, including PUs, are a component of the assessment.

Nursing homes frequently use the Braden Scale to stratify an individual's risk for PU development on admission, quarterly and with any significant change in condition. This is documented in the record and in the Minimum Data Set (MDS)^ {6} (a detailed assessment tool associated with an interdisciplinary patient care plan). CMS require that an MDS be performed on all patients in nursing homes which receive Medicare/Medicaid reimbursement. The data are transmitted to the states electronically and from there to CMS. The Joint Commission on Accreditation of Health Care Organizations (JCHCO) requires practitioners in long term care to assess and periodically reassess each patient's risk for developing PUs and address any risks.

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Drainage

Undermining or tunneling
– look for and measure skin

– Location of wound

– Presence and strength of peripheral pulses when lower extremity ulcers are affected

– Incontinence of urine or feces

– Any recent diagnostic tests

– Presence of odor

CLINICIAN DOCUMENTATION

Clinicians are required to generate a progress note for each encounter. This record allows a clinician to compare past status to current status and serves as a link between clinicians and other members of the care team. This documentation, used for medical, legal and billing purposes, should include:

Patient’s name and date and time of visit

Vital signs

Presence or absence of fever, tachycardia, other signs of sepsis

The Dressing – Type of dressing and appearance - Is there drainage on the outside of the dressing material?

Location of wound - This is important for identifying the origin of the injury, such as pressure over a bony prominence, or friction from a positioning device.

Size – length, width, and depth measured in centimeters.

Use the face of a clock to describe location, 12 o’clock is the patient’s head and 6 o’clock is the feet. To measure depth, use a sterile cotton tip applicator. It is very important to avoid cross contamination of wounds by using the same gloves, instruments, measuring devices from wound to wound in a patient with more than one. Assess changes based on previous measurements.

Undermining or tunneling – look for and measure skin that overhangs the wound’s edges. Undermining is the destruction of tissue or ulceration extending under the surface of the skin edges so that the wound is larger at its base than at the skin surface. Tunneling is a course or pathway that can extend in any direction from the wound resulting in dead space with potential for abscess formation. The two can be differentiated: tunneling involves a small portion of the wound edge whereas undermining involves a significant portion of the wound edge. Both tunneling and undermining are caused by shearing and forces against the wound.

Drainage – Is there drainage on the contact layers of the dressing? What does it look like - serous, purulent, bloody, green, yellow, clear, thick, etc? Is the drainage a breakdown of the wound dressing (like a hydrocolloid) or actual drainage from the wound? For example, yellow purulent drainage could indicate staphylococcus involvement.

Odor – Foul or fruity

Necrotic tissue – Presence and percentage of necrotic tissue. Drawing a small diagram can be helpful for future comparisons

Infection – Consider infection when a wound is not progressing as expected. DO NOT swab wound drainage for culture and sensitivity. Culture of wound drainage can lead to incorrect diagnosis of the infecting agent. Wound drainage is contaminated with microorganisms which are often not the causative pathogen. Either perform a biopsy of the bed (gold standard) or cleanse the wound and swab multiple times in multiple directions with a culturette.

Stage pressure ulcers – refer to the table 1 on staging pressure ulcers for a complete review. An ulcer with an intact eschar should be noted as unstageable due to eschar formation. DO NOT reverse stage a healing ulcer. For example, an ulcer initially documented as a stage 4 should not be documented as a stage 2 or a stage 1 as it heals. This stems from the fact that skin over a healed ulcer is only 70 - 80 percent as strong as undamaged skin. A new health care professional on the case may look at the latest notes and only see a stage 2 in the assessment and not realize that this patient is at high risk. Instead, document that the wound is a healing stage 4 ulcer.

Past treatment – Note the past treatments and any changes in products. This will help new health care professionals on the case. Products that may not have produced the desired results won’t be accidentally duplicated.

Current treatment – Document the type of irrigation, products and secondary dressings used during the dressing change.

Consider – Consulting physical or occupational therapist for assistance with positioning and mobilization; dietician to improve nutrition; wound care specialist; plastic surgeon for grafting; general surgeon for debridement; vascular surgeon for non-healing ulcers of the extremities.

STAGING

Pressure Ulcer Stages (NPUAP, 2007)

Suspected Deep Tissue Injury

Stage I Non-blanchable erythema of a localized area

Stage II Partial thickness loss of the dermis (presents as shallow open crater)

Stage III Full thickness loss of through the dermis and into the subcutaneous tissue. Fascia remains intact.

Stage IV Deep destruction of the soft tissue exposing muscle, tendon or bone.

Unstageable A pressure ulcer is unstageable if the wound bed is obscured.
The coding guidelines present the following information on how to code pressure ulcers:

“Two codes are needed to completely describe a pressure ulcer: a code from subcategory 707.0, Pressure ulcer, to identify the site of the pressure ulcer and a code from subcategory 707.2, pressure ulcer stages, are to be used as an additional diagnosis with a code(s) from subcategory 707.0, Pressure Ulcer. Codes from 707.2, Pressure Ulcer stages may not be assigned as a principal or first listed diagnosis. The pressure ulcer stage codes should only be used with pressure ulcers and not with other types of ulcers (e.g. stasis ulcers/ Vascular or diabetic ulcers).”

Note: Staging implies that a wound is a pressure ulcer: only pressure ulcers should be staged.
LEGAL RISKS IN SKIN AND WOUND CARE

Eventually the case of Mrs. X went to trial. The jury found for the defendant based on documentation of early identification of the ulcer and the timely and ongoing assessment and treatment. Mrs. X’s co-morbid conditions were considered a significant factor in her outcome.

Nursing homes are justifiably concerned about the legal risks surrounding PUs. The American Medical Directors Association (AMDA) advises nursing homes to implement care plans that are consistent with the patient goals as well as the standards of care. Documentation and frequent communication between the physician, patient, family and relevant team members should include changes in the ulcer, changes in the care plan, and conditions which may interfere with healing.

REFERENCES

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Alzheimer’s disease (AD), the most common type of dementia, is estimated to affect 13% of persons over age 65 and almost 50% of those over age 85. Since age is the major risk factor, the number of persons afflicted will increase exponentially over the next several years.

As the disease advances, caregivers become increasingly involved with bathing, dressing, feeding, and toileting. Behavioral symptoms are common and often more distressing to caregivers than cognitive and functional decline. Caregivers experience high levels of stress and depression in caring for the person with AD at home. Nursing home placement typically occurs after families have exhausted their financial, physical and emotional resources.

The major predictors of nursing home placement in persons with dementia include severity of cognitive impairment, diagnosis of AD, dependencies in basic activities of daily living, behavioral symptoms and depression. Without a clear etiology or effective medical treatment for AD, the goals are to maximize functional and cognitive abilities for as long as possible, and enhance the safety and comfort of persons with AD and their families. Non-pharmacological interventions that reduce behavioral symptoms, maximize the person’s function, and improve quality of life are potentially cost-effective and safe alternatives to pharmacological treatments.

The behavioral symptoms described above are common throughout the course of dementia although it is controversial whether the symptoms increase or diminish as the disease progresses. Nevertheless, the clinician should identify problematic behaviors early.

**Management of Behavioral Symptoms in Alzheimer’s Dementia**

Before an inappropriate behavior is attributed to dementia, a comprehensive evaluation should be conducted. The first step is to carefully describe the behavior, including the time, location and frequency of occurrence. There are a number of instruments to assess and document behavioral symptoms in dementia. In the long term care setting, the minimum data set is routinely used to assess behavioral problems. This assessment often leads to the identification of social, environmental factors, or acute medical problems (e.g., infections, hypoxia, angina, metabolic disturbances and depression) as potential causes. The most common physical cause of NPS is unrecognized, untreated pain, often resulting from urinary retention, fecal impaction, pressure ulcers, or osteoarthritis.
fied and physical causes treated, the next step is to identify specific antecedents and consequences of the problematic behavior and suggest treatment strategies.

No one strategy can address all behaviors. Psychosocial interventions are considered first line-treatment and are often used in conjunction with pharmacological treatments. Medications have an important role, but need to be used judiciously with informed consent from the patient and/or surrogate as reviewed by Epstein-Lubow and Rosenzweig in this special edition.

**NON-PHARMACOLOGIC TREATMENTS FOR BEHAVIORAL SYMPTOMS**

Non-pharmacologic approaches to dementia care can enhance the quality of life and maximize the functional status of residents with AD in long term care. Non-pharmacologic strategies can be divided into four categories: behavior-oriented, stimulation-oriented, emotion-oriented and cognitive-oriented. These approaches reflect a person-center approach to care, emphasizing the individual. For many long term care residents several strategies may be used concurrently, and, when possible, delivered daily or weekly.

Behavioral strategies are the most commonly used intervention in dementia care. The general principles include creating a structured environment with a daily routine, simplifying tasks, providing one-step instructions and using a non-confrontational approach. Correcting hearing and vision impairments can optimize the resident's participation in activities. Repetition, positive reinforcement, verbal and visual cueing assist the resident in achieving maximal function. In addition, residents do better in familiar activities, rather than activities that require new learning. Some evidence suggests modest benefits of behavioral strategies, but additional trials demonstrating efficacy are needed.

Special Care Units are generally situated within the long term care setting. The units include structured programming, a modified physical environment, and staff trained to use non-pharmacological approaches to care. However, the efficacy of these units in reducing NPS has not been demonstrated, compared to traditional nursing home care.

Stimulation-oriented approaches include aroma therapy, bright light, movement, multi-sensory, music and touch therapies. These activities are generally offered as part of the therapeutic environment in long term care. Multisensory stimulation (MSS) environments or Snoezelen stimulate the senses through providing therapeutic objects of un-patterned visual, auditory, olfactory and tactile stimuli in a specially designed room or environment. Evidence demonstrates that MSS might help to reduce apathy in latter stages of the disease, but many of the positive results were not statistically significant and the benefits were not sustained over time.

Hulme et al. reviewed ten studies examining the effects of music therapy in reducing problematic behaviors. These studies scrutinized a wide range of behavioral symptoms including agitation, aggression, wandering, restlessness, and nutritional intake. Music and music therapy were effective in reducing behavioral symptoms, but the impact did not persist over time.

Physical activity and exercise can also reduce behavioral symptoms and improve functional ability. Additional benefits of a sustained walking or exercise program are decreased wandering and improved sleep quality and mood.

Emotion-oriented strategies include reminiscence therapy, validation therapy, supportive psychotherapy, simulated presence, and sensory integration. The efficacy of supportive psychotherapy has been demonstrated in the early stages of disease when patients are dealing with their prognosis, but has shown little benefit as the disease progresses. Simulated presence therapy utilizes video or audio recordings of family members' conversations, stories or shared memories to decrease agitated behaviors. This strategy is effective, easy to administer and well received by LTC residents. However, the effects are not sustained over time.

Hulme et al. reviewed four studies examining the efficacy of reminiscence therapy on mood, behavioral and psychological symptoms. This therapeutic model has a modest evidence base, but the results are based on studies with limitations in designs and method. There is less evidence supporting the efficacy of validation therapy and sensory integration in reducing NPS.

Less evidence supports cognitive-oriented strategies, which include reality orientation and cognitive retraining. These strategies focus on decreasing confusion and problematic behavior by orienting patients to time, place and person. Although there may be short term improvements in selected domains of cognition, this is not sustained over time. Short term emotional consequences such as frustration, agitation and depression have been reported with these interventions. External memory aids may be more effective than learning mnemonics for enhancing cognitive function of long term care residents with AD.

Overall, non-pharmacological strategies have demonstrated modest efficacy in improving mood and reducing behavioral disturbances in long-term care residents with dementia. In many cases, these strategies alone may not be able to manage the problematic behavior of the resident. In these situations, a multimodal approach that includes psychoactive agents may be necessary. More rigorous studies need to be conducted to examine the efficacy of non-pharmacological compared to pharmacological approaches in reducing behavioral symptoms and improving quality of life for the long term care resident with dementia.

**REFERENCES**


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The Use of Antipsychotic Medication In Long Term Care

Gary Epstein-Lubow, MD, and Andrew Rosenzweig, MD, MPH

This brief review frames the historical context surrounding antipsychotic medication for patients in nursing homes and other long-term care settings. The information is meant to describe, concisely, the evidence for and against the use of antipsychotic treatment. Every patient's experience is distinctive, and each treatment decision has potential benefits and risks.

The first antipsychotic medication approved for use in the United States was chlorpromazine. In the 1950s, the use of chlorpromazine allowed for deinstitutionalization of millions of residents from psychiatric hospitals. In the ensuing five decades, antipsychotics in the “first generation” with chlorpromazine and the “second generation,” beginning with clozapine, have been in and out of favor for patients in long-term care.

**Antipsychotic Medication and Nursing Home Reform**

In 1987, the Omnibus Budget Reconciliation Act (OBRA) incorporated nursing home reforms. OBRA required all nursing home residents to participate in a national database originally defined by the Resident Assessment Instrument, today known as the Minimum Data Set (MDS). The Resident Assessment Instrument led to significant improvement in care plan documentation, greater use of advanced directives and better behavioral treatments for problem such as bowel incontinence, while reducing problematic practices such as physical restraints and indwelling urinary catheters.

OBRA led to increased monitoring of antipsychotic medication in nursing homes, but it is not clear whether overall usage declined or remained stable during the early 1990s. For patients receiving antipsychotic medication, the OBRA regulations mandate that all nursing homes report the following: an appropriate diagnosis for use of an antipsychotic medication, including specific target symptoms along with change in these symptoms over time; administration of the medication within a recommended 24-hour dosage limit along with routine monitoring for side effects; consideration of concurrent behavioral treatment; and, at least one attempt every six months to reduce the dose of antipsychotic (or documentation of a rationale for no dose reduction).

Soon after OBRA, serial introduction of second generation antipsychotics started. Early interest in these medications may have led to increased use in nursing homes. The original interest in the second generation antipsychotics for use in the elderly may have been driven by beliefs regarding reduced risk for parkinsonism, though most of these medications can impair gait and mobility. An estimated 25% of nursing home residents in the United States receive at least one antipsychotic medication in a given year. Though much of this use is for off-label indications, the vast majority of antipsychotic treatment is for evidence-based treatment. Nevertheless, the antipsychotic prescribing rate in nursing homes and the relationship between the use of antipsychotics and mortality remain concerns.

**Regional Attention to the Use of Antipsychotics in Long Term Care**

The January 2007 iteration of OBRA recommends Gradual Dose Reduction (GDR) of all antipsychotic medications in nursing homes. For patients newly admitted and receiving an antipsychotic medication, or established patients who begin an antipsychotic medication, during the first year of treatment staff must document at least two attempts to reduce the medication, with at least one month between the attempts. If the antipsychotic medication continues beyond the first year, staff must attempt one GDR every year unless clinically contraindicated. Documentation that a GDR is clinically contraindicated must include 1) notation that target symptoms worsened during the most-recent GDR attempt in the current facility along with 2) the physician's current medical opinion as to why additional attempts at GDR are likely to impair the patient's functioning or worsen the target symptoms.

In Massachusetts, the Department of Public Health has convened an Antipsychotic Task Force to raise awareness about antipsychotic use, including possible education campaigns and direct interaction with nursing home clinicians and administrators. Long-term care facilities are likely to welcome such activities because the new GDR guidelines carry devastating penalties for non-compliance. For example, F-Tags are regulations within the state operations manual; they are written by the Centers for Medicare and Medicaid Services. The F-Tags set guidelines for prescribing clinicians, pharmacists, nursing homes and state surveys. F-Tag 329 applies to unnecessary psychotropic medications. The goals of F-Tag 329 are to ensure that 1) medications are clinically required to treat a condition; 2) behavioral or other non-medication measures are used; 3) medication use is in an effort to promote the highest well-being; 4) actual or potential negative outcomes are avoided; and, 5) all negative outcomes are promptly identified and treated. If GDRs are not conducted according to the rules, the nursing home must undergo a cumbersome follow-up: a level 4 deficiency (immediate jeopardy) results if there is failure to monitor or reduce the dose of an antipsychotic in the presence of a side effect such as worsening gait and mobility due to parkinsonism, or if there is a failure to do a non-contraindicated GDR in the context of a significant negative drug effect such as tardive dyskinesia.

**Safety and Efficacy of Antipsychotic Medication Use for Elderly Individuals**

The OBRA legislation occurred 15 years before the US Food and Drug Administration (FDA) began generalized warnings regarding elderly patients' use of antipsychotic medication. In the years following OBRA, use of first generation antipsychotics declined; by 2004, prescriptions for second generation antipsychotic medications in nursing homes outnumbered first generation use 10 to 1. A considerable portion of this antipsychotic treatment is for behavioral disturbances...
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<td><strong>Total Cost</strong></td>
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associated with dementia; there has been moderate empirical support for this practice since the early 1990s with declining interest regarding these medications in the context of documented adverse events.

As antipsychotic medication prescribing continued, pharmaceutical companies invested in clinical trials directed at gaining an FDA indication for the use of antipsychotics in the treatment of behavioral aspects of dementia. In addition to continued evidence for efficacy, information became available regarding risks for medical adverse events (e.g., stroke, diabetes and death) related to use of second generation antipsychotics in elderly individuals. The first reports of these concerns appeared in 2003: a study of risperidone showed a non-statistically significant increased rate in stroke and mortality for individuals receiving active medication, compared with placebo.10 By the end of 2003, the FDA requested that all second generation antipsychotics include new warning information. In 2005 the FDA issued a black-box warning for all second generation antipsychotics based on data available from meta-analyses11 and soon after extended the same warning to all first generation antipsychotics. Today all antipsychotic labeling states: "Elderly patients with dementia-related psychosis treated with antipsychotic drugs are at an increased risk of death."

Occurring concurrently with the warnings regarding use of antipsychotic medications in dementia was the Clinical Antipsychotic Trials of Intervention Effectiveness – Alzheimer’s Disease (CATIE-AD) investigation of the second generation antipsychotic medications risperidone, olanzapine and quetiapine as treatment for behavioral disturbance associated with dementia due to Alzheimer’s disease in community-dwelling outpatients.12 The CATIE-AD study’s first reports have provided consistent evidence of adverse effects13 including significant metabolic changes in weight and cholesterol metabolism.14 Additional analyses have shown both positive and negative results regarding clinical efficacy; for example, some clinical symptoms in outpatients with Alzheimer’s disease do respond to antipsychotic treatment.15 This evidence must be weighed against the risk of adverse events such as those described above plus pneumonia.

The January 2007 iteration of OBRA recommends Gradual Dose Reduction (GDR) of all antipsychotic medications in nursing homes.

Considerations When Starting or Continuing Antipsychotic Medication Treatment in the Nursing Home

Residents in long term care continue to receive antipsychotic medication because, on a case-by-case basis, symptoms may improve, along with few adverse events. Residents may receive antipsychotic medication for behavioral symptoms associated with dementia or for an illness other than dementia. When considering use of an antipsychotic medication in the long term care setting, the first step is to clarify the condition in need of treatment. Antipsychotics are approved for use in schizophrenia, and several of the second generation antipsychotics have additional FDA-approved indications for the treatment of mood episodes such as acute mania or depression. Though considered “off-label” use, antipsychotic medication is reasonable for psychotic symptoms that occur in isolation (such as in delusional disorder) or in the context of other conditions such as delirium, Parkinson’s disease, psychotic depression and dementia. Regarding dementia, in addition to the treatment of psychotic symptoms such as hallucinations and delusions, antipsychotic medication has been used as treatment of other target symptoms such as irritability, aggression and disinhibition, as noted above.

Once a target symptom(s) has been established, the clinician should ask several questions before starting a new trial with an antipsychotic. First, is there a current behavioral emergency, or a high likelihood that intense target symptoms will recur soon and interfere with the patient’s or others’ safety? Secondly, is there a reason to suspect an urgent medical cause? If yes, then work-up and treatment might best occur in an acute-care hospital rather than the nursing home. If the target symptoms do not require hospital-level care, then several tasks can be considered concurrently.

What is the likely underlying disorder or condition contributing to the target symptoms? Are tests needed to confirm the diagnosis? Does any currently-prescribed medication carry risks or side-effects that might relate to the target symptoms? If so, could this medication be reduced or stopped? Has any change in environment or daily routine occurred recently; if yes,
could the surroundings be restructured? How are family members involved in the patient’s care? Can a surrogate decision-maker or primary caregiver assist with problem-solving regarding the symptoms?

After a diagnosis has been established, target symptoms fully characterized and environmental factors addressed, the clinician should ask several additional questions. Might any formal behavioral or psychosocial treatments be feasible for these target symptoms? Could medication include a trial of a drug that is not an antipsychotic?

(Long term care facilities frequently contract with specialty service providers who are familiar with medication treatment options; these providers can, over time, become acquainted with the individual nursing home environment to determine which psychosocial or other treatments have the best likelihood of success.) As quality indicators regarding the treatment of dementia and other mental health conditions in nursing homes come under increased scrutiny, every resident with a mental health diagnosis may at some point be expected to receive mental health services.17

For now, each patient’s case must be treated uniquely. The FDA has approved antipsychotics for illnesses such as schizophrenia, but the use of these medications in the nursing home, irrespective of diagnosis, must follow standard OBRA and GDR guidelines. For the treatment of behavioral symptoms related to Alzheimer’s disease or another dementia, no pharmacotherapy is approved for these symptoms, though FDA-indicated treatments for memory loss in Alzheimer’s disease such as donepezil and memantine may provide some behavioral benefit. Also, psychosocial treatments for dementia have demonstrated benefits amidst few risks18 as reviewed by Curtin in this issue. Expert opinions regarding the management of agitation in dementia19 and the use of antipsychotic medication in the elderly20 are useful reference points for clinicians. Ultimately, if the clinician initiates medication for behavioral symptoms in a long-term care setting, particularly if the medication is an antipsychotic, it must be clear that the situation is likely to improve with the medication and that the treatment occurs under careful monitoring with a plan for discontinuation after short-term use.21

References

The prevalence of memory impairment, including dementia, increases exponentially with age. Of people with late-onset dementia, about half have Alzheimer’s, 16% have vascular dementia, and 30% have other forms of dementia. Alzheimer’s disease, the most frequent cause of dementia in Western societies, affects an estimated 5 million people in the United States and 17 million worldwide. The onset is insidious with manifestations evolving over a period of years from mildly impaired memory to severe cognitive loss. The course is progressive and terminates with mental and functional incapacity and subsequent death. As many as 90% of Americans with dementia will be institutionalized before death. Nursing homes are, therefore, important providers of end-of-life care. Several researchers have highlighted the need to improve the quality of palliative care in the nursing home setting.

Understanding the Stages and Trajectory of Dementia

Dementia is defined as a syndrome characterized by multiple impairments in cognitive function without associated deficits in consciousness. The domains affected include general intelligence, learning, memory, language, problem solving, orientation, perception, visuospatial skills, attention, concentration, judgment, executive functioning, personality, and social abilities. The acquisition of cognitive impairment after years of normal functioning differentiates dementias from developmental disorders; the persistence of the deficits differentiates dementia from delirium. The wide spectrum of involved domains helps to distinguish dementias from other neurologic conditions such as aphasia, amnestic syndromes, and neurocognitive manifestations of stroke. Dementia is associated with a wide variety of underlying conditions that affect the integrity of the central nervous system including primary neurodegenerative disorders, vascular injuries, infections, hydrocephalic conditions, drug-induced or metabolic conditions, and trauma.

Advanced dementia refers to progressive immobility and reduced capacity for self-care, poor nutrition resulting from reduced intake, infections, skin breakdown, and general debility. Many patients who develop dementia have other illnesses that contribute to overall decline in health. In particular, Alzheimer’s disease is an incurable and invariably fatal illness that has an average life expectancy of 4 to 7 years after diagnosis. Most dementias follow a disease course typical of other chronic illnesses, with gradual deterioration, punctuated by substantial cognitive and functional decline, usually as a result of an acute illness. Upon recovery from the acute illness, patients with dementia usually establish a new lower level of cognitive and physical functioning.

In the advanced stages of dementia, any acute stressor such as hip fracture, urinary tract infection, or pneumonia could become a terminal event. A study by Morrison showed that demented patients admitted to the hospital for pneumonia or hip fracture had a six month mortality of 50%, substantially higher than that of cognitively intact controls.

The cognitive and functional decline of Alzheimer’s patients usually follow a predictable pattern. Patients with mild dementia exhibit reduced memory of personal history, decreased capacity to perform complex tasks, and mild personality changes. As the disease advances to the moderate stage, patients become increasingly confused and disoriented, need assistance with most activities of daily living (ADLs), and have more noticeable declines in short-term memory. The severe stage is characterized by significant personality changes and behavioral symptoms, bladder and bowel incontinence, altered sleep habits, and dependence on all ADLs. Shuster describes the symptoms of this stage:

Neurocognitive – progressive worsening of memory and other cognitive deficits; profound confusion and disorientation; behavioral changes including combativeness or resistance giving way to apathy, then coma; progressive worsening of speech, inability to communicate; patient becomes incoherent, mute, then unresponsive.

Functional – independent mobility progressively lost; patient becomes bedbound; capacity for self-care progressively lost; and patient becomes totally dependent.

Nutritional – progressive loss of appetite and ability to swallow or eat independently; aspiration becomes an increasing risk

Associated Complications – fevers, infection, decubitus ulcers, aspiration pneumonia, urinary tract infections

Patients in the final stages of dementia are eligible to receive hospice services. The National Hospice and Palliative Care Organization (NHPCO) uses the Reisberg Functional Assessment Staging (FAST). Patients who meet FAST 7C exemplified by the inability to ambulate, dress, and bathe without assistance; intermittent or constant bladder or bowel incontinence; and the absence of meaningful verbal communication and/or use of fewer than six intelligible words. A FAST 7A demented patient with an ability to speak at least six words without necessarily having the other deficits of 7C may be eligible for hospice if the life expectancy is deemed to be less than six months and supported by one or more dementia-specific co-morbidities such as aspiration pneumonia, decubitus ulcers, persistent fever, greater than 10% weight loss, sepsis, and upper urinary tract infections.

Palliative Care For the Nursing Home Resident With Dementia

Noel S. C. Javier, MD
Palliative Care for Nursing Home Residents with Dementia

More than 90% of the nearly five million Americans affected by dementia die in the nursing home. Luchins reported that both relatives and health care providers believe that palliative care is an effective answer to terminal stage dementia symptom control. Thus, the provision of high-quality palliative care in the nursing home setting is essential. In the past, people with dementia received suboptimal end-of-life care. In 1996, only 1.5% of patients with dementia could access Medicare hospice programs while a great number were admitted to hospitals and invasively treated despite a short life expectancy. Many dementia patients died with feeding tubes in place, up to 44% of nursing home residents with dementia in some states, despite research suggesting little to no benefit from this treatment.

Over the next few years, there has been a steady increase in hospice admissions for dementia, from 12,829 in 1998 to 60,488 in 2008. This may in part be attributed to great strides in promoting palliative care either through research initiatives or increased awareness of the specialty such as fellowship training and education. In 2006, non-Alzheimer’s dementia became the most common diagnosis among Medicare hospice patients. The percentage of all Medicare hospice patients with a terminal diagnosis of cancer dropped from 52.8% in 1998 to 31.1% in 2008. Munn’s study showed that the rates of hospice use in nursing homes and assisted living were considerably higher than previously reported although persons with dementia may continue to be underrefered.

Barriers to Palliative Care in the Nursing Home

First, both health professionals and family members may have difficulty viewing dementia as a terminal illness. In a model demonstration project integrating palliative care into the ongoing care of dementia patients, 70% of families interviewed after the death of the patient believed that the patient was terminally ill and dying prior to death; more than two thirds of those family members who believed that the patient was dying believed it was from something other than dementia. Dementia differs considerably from cancer in that the time from diagnosis to death is usually longer with a length of survival typically measured in years. Given this protracted course and the gradual loss of cognition and function, physicians and families understandably struggle to view dementia as a terminal diagnosis.

Second, despite the models that attempt to predict death within 6 months, it can be difficult to determine when end-of-life care should be provided in an individual who may survive for years.

In 2006, non-Alzheimer’s dementia became the most common diagnosis among Medicare hospice patients.

Third, the provision of palliative care to dementia patients may be limited by the cognitive, communication, functional, and behavioral problems that arise with this disease. The evaluation of needs and the management of symptoms require a broad and thoughtful approach from health providers with input from staff and caregivers.

Fourth, Medicare’s policies create discontinuity in care if patients with dementia also have acute illnesses. For a typical example: a patient with dementia suffers an acute illness, which results in hospitalization, followed by a decline in cognition and function; home care services start, restart, or increase in intensity; the plateau of function is set at a new baseline typically below the pre-hospital baseline; Medicare discontinues reimbursement for nursing, therapy, and other services; the patient declines in function due to underlying dementia until the next acute illness restarts the cycle.

The financial incentives often work directly against the provision of palliative care, especially for long-term residents of nursing homes. Continuity of care both in terms of location and familiar staff is important for these patients, considering the risk for delirium and distress on transfer to the hospital. As staff spend more time on managing symptoms and providing comfort near death, the facility ends up bearing the increased cost without receiving additional reimbursement.

The provision of quality palliative care to dementia patients could potentially be addressed by systematic education and information dissemination among health providers, patients, families, and caregivers; more innovative programs integrating palliative and curative care including anticipatory grief and bereavement programs; modification of the payment system extending to the state and/or federal levels; support programs for caregivers and staff; and quality improvement programs at the organizational and health systems level.

Ethical Issues

Dementia patients with acute illnesses are frequently transferred to the hospital for aggressive treatment without consideration of their cognitive impairment, behavioral symptoms, and chronic co-morbidities, such as pressure ulcers and other chronic illnesses. Keene reported that autopsy reports of patients with dementia identified the main causes of death to be pneumonia, cardiovascular diseases, lung embolism, cachexia, and dehydration. As dementia patients develop infections such as pneumonia and urinary tract infections, they are subsequently hospitalized and managed with antimicrobial therapy. One study found an in-hospital mortality of 20% for advanced dementia patients receiving antibiotics, with a 6 month mortality rate of more than 50%. Furthermore, these patients may be subjected to painful daily blood draws and intravenous therapy, which in turn put the patient at risk of needing restraints.

Patients with advanced dementia have difficult eating. Weight loss and poor oral intake are a hallmark of the later stages of dementia. Those patients have difficulty swallowing, lose oral motor coordination, and their interest in food. Staff frequently discuss the need for a feeding tube, citing: decreased mortality, prevention of aspiration, improved nutritional and functional status, and
healing of pressure ulcers. A landmark review by Finucane and colleagues demonstrated that none of these indicators are reversed or improved with tube feeding. No randomized trials compare hand-feeding and tube-feeding for end-stage dementia patients, but cohort and cross-sectional studies show no improvement in patient outcomes with tube feeding. Mortality is high with or without tube feeding: 30-day mortality averages 20%; 6-month mortality, 50%. Additionally, feeding tubes can lead to complications (leaking, clogging, and replacement if dislodged), which can lead to more medical care, as well as the use of restraints.

In addition to worsening nutritional status as a result of dysphagia related to progressive dementia, these patients also experience functional impairment and physical debility. They eventually become bed-bound, increasing the chances for falls and hip fractures. The in-hospital mortality rate for repairing a fractured hip in persons with advanced dementia is relatively low, about 5%; however, the 6-month mortality rate remains high (greater than 50%). Sadly, 76% of these patients did not receive a standing analgesic regimen for pain.

Finally, the polypharmacy related to multiple morbidities is a concern. To date, clinicians pay little attention to discontinuing inappropriate therapy in persons nearing end of life. A recently published article suggests a decision-making model that takes into consideration remaining life expectancy, time until benefit is derived, goals of care, and treatment targets.

Recognition of dementia as a terminal illness can assist providers in caring for these patients as the course advances. Supportive and palliative treatment goals can aid in feeding difficulties, decreased functional status, polypharmacy and pain management.

REFERENCES
CASE (PART 1)

One Saturday morning, October 2, 2009, the nursing home ward nurse reported that one of our patients developed new respiratory symptoms: a dry cough and sneezing but no fever. At age 84, her medical history included heart failure, diabetes, osteoporosis and dementia—the reason for her institutionalization. The previous night, new onset restlessness made it difficult to get her to stay in bed. She repeatedly got up to wander or find a new seat, intermittently sleeping in a chair by the nurses’ station or by her bed. Her pulse oximeter showed 95% saturation, and she was tachycardic at a rate of 100 beats per minute. Blood pressure remained in her usual range. She did not complain of breathlessness, with her respiratory rate of 18.

CLINICAL PRESENTATION OF INFLUENZA

The immediate differential appears broad, but the clinical information reported by the nurse remains incomplete without the epidemiologic context. Knowing which pathogens are circulating in the community can help in ranking certain viral and bacterial diseases in the differential diagnosis. Influenza presents with a variety of symptoms. In a healthy younger population, fever plus respiratory symptoms can clinch the diagnosis in 80% of adults, when influenza is known to be circulating in the community.1 By October 2, 2009, the Rhode Island Department of Health had already reported local circulation of the new pandemic A/H1N1 influenza strain, similar to that reported by the Centers for Disease Control and Prevention (CDC) for much of the rest of the country.2 Epidemiologists described symptomatic presentation for this strain as typically affecting young, rather than old patients, and new risk factors for complicated disease included obesity and pregnancy, but not age per se. In children, diarrhea and abdominal symptoms were sometimes the main presenting symptoms.

Because of both the apparent predilection of this new virus for illness and more severe disease in younger adults and the initial limited availability of vaccine, the pandemic A/H1N1 vaccination of older adults, especially institutionalized older adults, was a low priority. But could this patient’s symptoms be influenza? (Table 1)

The presence of fever adds specificity to the diagnosis of influenza, but can also substantially reduce the sensitivity: the greatest reduction in sensitivity occurs in frail elderly adults, where the majority typically do not develop an oral temperature greater than 99°F during the course of their illness, (Figure) In addition, elderly nursing home residents have multiple morbidities, and the presenting influenza symptoms may take the form of exacerbations of pre-existing conditions. Typically more than half of the residents in US nursing homes have dementia and may be unable to report the complaints we typically associate with an influenza-like illness, such as malaise, dyspnea or chest discomfort.

In the US people aged 65 or older represent about 13% of the population. Yet influenza causes more than 90% of the overall influenza-related mortality annually in this group. Indeed, the risk of influenza-related death increases after the age of 50 years and exponentially after the age of 65.7 However, to reflect on influenza mortality or even the 200,000 plus hospitalizations8 grossly understates the impact influenza has on older individuals, especially the frailest old. Influenza can produce significant functional decline in elderly patients.9 Over one-third of hospitalized patients age 70 or older leave the hospital more disabled than when they arrived;10 older patients endure more

<table>
<thead>
<tr>
<th>Symptom</th>
<th>SEASONAL INFLUENZA</th>
<th>PANDEMIC INFLUENZA</th>
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<tbody>
<tr>
<td>Cough or chest discomfort</td>
<td>Common, can become severe</td>
<td>Common, often lasts 2-3 weeks</td>
</tr>
<tr>
<td>Fever</td>
<td>Common, often high, abrupt onset, 3-4 days</td>
<td>Common, often with oral temperature</td>
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<td>Sneezing</td>
<td>Often</td>
<td>Sometimes</td>
</tr>
<tr>
<td>Malaise</td>
<td>Can be extreme, lasting 3 weeks</td>
<td>Common, may precede vascular event</td>
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<td>Gastrointestinal</td>
<td>Rare</td>
<td>Occasional</td>
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<td>Symptom duration</td>
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<td>2-4 days</td>
</tr>
<tr>
<td>Incidence</td>
<td>3-10 week period in late Fall to early Spring</td>
<td>All year; more severe in Winter</td>
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</tbody>
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Table 1. Influenza Symptoms in Adults. Adapted from references 3 and 4.
frequent and longer influenza-related hospitalizations; thus influenza likely contributes significantly to this functional loss.

(Table 2) Influenza vaccine can protect individuals from influenza and influenza-related morbidity, such as pneumonia, and still reduce the occurrence of fever when it fails to prevent infection. (Figure) However, and more importantly, the vaccine reduces the likelihood of other complications such as cardiac and cerebrovascular collectively, even better than preventing pneumonia. In the context of our case, we have insufficient information to propose influenza as the likely diagnosis. The nonspecific symptoms of our patient could represent a variety of conditions, including any one of a number of respiratory viruses.

**CASE (PART 2)**

Questions posed to the nurse calling for direction on our patient included the following:

1) Is the patient the only one who is newly sick (respiratory or other symptoms) in the past week; if others are ill, have they had close contact (share a room, activities, dining table, sit together) with our patient?

2) Have any other residents gone to the hospital for evaluation for cardiac, cerebrovascular, pulmonary, or fever issues in the last few days?

3) Are there concomitant behavior changes in other residents? If so, do some or most of them come in close contact with each other or our case patient?

4) Do any staff have new respiratory symptoms, or did staff call in sick?

5) Is the nurse reporting about the patient symptomatic (is there hoarseness, sniffing, sneezing or other audible respiratory abnormality present)?

6) Has the caller noticed if tissue boxes are now on the medication cart, or facial tissues in the trash containers in the rooms or nurse’s station?

The caller indicated another resident was taken to the emergency department the night before for evaluation of chest pain; that patient was admitted for an acute myocardial infarction; another had received a chest radiograph which the radiologist read as consistent with a pulmonary infiltrate. The resident who usually sits next to our patient has taken to bed in the last day, and two other residents who sit together had mild coryza, one with diarrhea; neither of these two typically sit with the resident our caller has reported on. The caller also sniffled twice while giving this report on the phone. No other staff members had reported in sick, and the caller denied being ill, explaining she had seasonal allergies. The residents had received their seasonal influenza vaccine the week before but not the pandemic vaccine because it was unavailable to them.

**INFLUENZA IN NURSING HOMES**

Our case is ensconced with others who have taken ill. Any one of the other residents, or even the audible symptoms of the staff member reporting on our

![Figure legend. Proportion of residents with laboratory-confirmed influenza who have an oral temperature over 99°F by number of days following symptom onset. Two groups of 400-500 nursing home residents were prospectively monitored over three years (1997-2000) for the onset of new clinical symptoms occurring when A/Sydney/5/97 [H3N2]-like influenza was known to be circulating in the community. When such symptoms were detected, the individual was tested for influenza by culture and PCR, and was clinically monitored daily until symptom abatement. Influenza was detected 1/4 as frequently among the group who received the vaccine that was well-matched to the circulating strain than the two comparator groups. (Previously unreported, these data are drawn from the populations monitored that included participants of two influenza antiviral prophylaxis trials, and reported in abstract form).

<table>
<thead>
<tr>
<th>Biologic change with age</th>
<th>Clinical Consequence</th>
</tr>
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<tbody>
<tr>
<td>Reduced IL-6</td>
<td>Reduced fever, less efficient viral clearance</td>
</tr>
<tr>
<td>Impaired mucociliary function in respiratory tract</td>
<td>Reduced cough, less efficient viral clearance and mucus clearance</td>
</tr>
<tr>
<td>Reduced TNF-α</td>
<td>Less malaise</td>
</tr>
<tr>
<td>Delayed increase in inflammatory cytokines</td>
<td>Fewer symptoms at disease onset</td>
</tr>
<tr>
<td>Delayed decline in inflammatory cytokines</td>
<td>Greater area-under-the-curve during which inflammatory cytokines produce a pro-thrombotic state (risk for thrombo-embolic stroke, myocardial infarction)</td>
</tr>
<tr>
<td>Reduced T-cell help</td>
<td>Reduced protection from vaccine; reduced longevity of protection from vaccine</td>
</tr>
<tr>
<td>Reduced nutrition</td>
<td>Reduced physiologic reserve, more difficult rehabilitation</td>
</tr>
<tr>
<td>Brain aging</td>
<td>Greater likelihood of delirium, sleep and appetite disturbance with cytokine storm</td>
</tr>
</tbody>
</table>
patient alone would hardly raise the index of suspicion about influenza in our patient, let alone an influenza outbreak. The CDC had not reported seasonal influenza, but showed the pandemic A/H1N1 strain circulating widely throughout the country, including Rhode Island. No other viruses were reported to in wide circulation in Rhode Island at that time.

Collectively, this presentation suggests a disease outbreak. As the only widely circulating virus appears to be the pandemic strain of influenza, it moves to the top of the differential diagnosis for our patient. In our case, a specimen was ordered for PCR confirmation of pandemic influenza in our patient and several other residents. Antiviral prophylaxis with a neuraminidase inhibitor was initiated for those without signs or symptoms of new illness, and antiviral treatment in those who were symptomatic, both residents and staff willing to receive medication.

Nursing home residents run a greater risk of influenza complications than their younger counterparts. The residents are most susceptible to influenza, given their multi-morbidities and greater exposure risk through close-living quarters and shared caregivers. Clinicians have under-appreciated the context of pandemic influenza and the risk to residents of nursing homes.

In contrast to seasonal influenza, the majority of symptomatic infections during pandemic influenza have been reported in young adults. Experts have offered persistent immunological memory from prior exposure to similar viruses decades earlier as an explanation; 1/3 of the adults born before 1950 had cross-reactive antibodies to the pandemic H1N1 virus at the beginning of the 2009 pandemic. Nevertheless, absence of recognized influenza symptoms with the pandemic strain have not kept elderly individuals from getting infected and seroconverting to it. In a small study of elderly people in Shanghai, the seropositive rate climbed from 9.4% to 42.5% from April to September in 2009, although none of these elderly subjects had any influenza-like symptoms during the study time. The observation is intriguing, raising the possibility of an unrecognized reservoir of potential pandemic transmission among the elderly with sub-clinical influenza.

Although disproportionately fewer elderly people developed clinical influenza, their mortality during the 2009 H1N1 pandemic remained substantial. For example, an analysis of patients hospitalized with pandemic H1N1 virus in California found that persons ≥ 50 years of age had the highest influenza fatality rate (18-20%). As with seasonal influenza, presence of underlying chronic diseases increased the mortality risk. Also, several nursing home outbreaks with pandemic H1N1 influenza have been reported during the 2009-2010 influenza season, indicating that even if older patients harbor cross-protective antibodies, as with seasonal influenza following vaccination, they remain uniquely susceptible and continue to experience greater morbidity and mortality overall than their younger counterparts.

**Summary**

Seasonal and pandemic influenza clinically remain remarkably similar in long-term care populations. Clinicians cannot distinguish clinical influenza, whether seasonal or pandemic H1N1, from other respiratory viral infections in individual patients. Part of the difficulty in the clinical diagnosis relates to fewer clinical features that might help with diagnostic differentiation, such as fever. However, the nursing home provides an epidemiologic context that can prove helpful to clinicians who inquire—by considering illness patterns among others in the facility, both staff and residents. This can lead to more timely diagnosis and treatment in the resident, and prophylaxis—an opportunity to protect the remaining residents and staff. Check out the treatment guidelines posted on the CDC website to be sure to select the best agents, because antiviral resistance patterns have been rapidly changing.

**References**


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Stefan Gravenstein MD, MPh, and Aurora Pop-Vicas MD, MPh, and/or spouses/significant others have no financial interests to disclose.

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Medications have long been associated with acute lung injury. We describe a case of a patient with acute fibrinous and organizing pneumonia (AFOP) after receiving decitabine.

CASE PRESENTATION

A 64-year-old man with a history of cardiac transplant in 2000 secondary to idiopathic nonischemic dilated cardiomyopathy presented with pancytopenia in July 2009 and was diagnosed with the myelodysplastic syndrome refractory anemia with excess blasts-2 (RAEB-2). His blast count bordered on qualifications for acute myeloid leukemia, and he was initially treated with standard induction consisting of seven days of standard-dose cytarabine followed by three days of daunorubicin. A repeat bone marrow showed persistent blasts and he was then treated with high-dose cytarabine which he also failed. Subsequently, his treatment regimen was changed to decitabine at the standard dose of 20 mg/m² days 1-5 dosed every 4 weeks. He received 3 cycles of decitabine. His only other medications were cyclosporine 75 mg daily, prednisone 5 mg daily, and omeprazole 20 mg daily.

Within three months of the first dose of decitabine, his chest radiographs began to show bilateral diffuse opacities. On the day of admission, he presented to an outpatient cancer center with worsening fatigue, dyspnea on exertion, a nonproductive cough, and tachypnea. On examination, temperature was 37.8 degrees Celsius, blood pressure 130/80 mmHg, respiratory rate was 30/minute, and his oxygen saturation was 100% on room air. Lungs were clear to auscultation but during the course of his hospitalization he developed diffuse rales bilaterally.

High-resolution computed tomography (CT) of the chest (Figure 1) showed innumerable bilaterally centrilobular nodules most prominent in the dependent regions of the lung. There were subpleural reticular changes with thickening of the interlobular septae but no evidence of architectural distortion or honeycomb cystic change.

Bronchoscopy was performed and microbiological studies for bacterial, fungal, and viral etiologies were negative. A transbronchial biopsy showed no evidence of infection or leukemic infiltrate but was concerning...
for diffuse inflammation. An open lung biopsy was performed which was consistent with acute fibrinous and organizing pneumonia (Figure 2). The patient was started on high-dose intravenous methylprednisolone (250mg IV every 6 hours for three days) and subsequently transitioned to oral prednisone (40mg daily) with significant improvement in his symptoms. At a follow-up visit several weeks after discharge, the patient's respiratory symptoms had resolved and he was tapered to his baseline prednisone dose of 5mg daily.

**DISCUSSION**

**Myelodysplastic syndromes (MDS)** have been reported with a higher frequency in patients receiving solid organ transplants.¹ Decitabine is a cytosine analog that serves as a hypomethylating agent (HMA) by inhibiting DNA methyltransferase and is indicated for the treatment of myelodysplastic syndromes including refractory anemia with excess blasts (RAEB).² Commonly, it is associated cytopenias although pneumonia and respiratory failure have also been reported.

**Acute fibrinous and organizing pneumonia (AFOP)** was first described in 2002 as a variant of diffuse alveolar damage.³ Characterized by intra-alveolar fibrin balls and organizing pneumonia, the pathologic specimens are notable for absence of granulomatous inflammation and hyaline membranes. In a series of Canadian patients diagnosed with the severe acute respiratory syndrome (SARS) a significant number of patients were noted to have AFOP on post-mortem analysis.⁴ AFOP has also been described after hematopoietic stem cell transplantation (HSCT) as well as after decitabine administration.⁵ Treatment usually includes steroids, although antibiotics and cyclophosphamide have also been used. Recently, use of mycophenolate mofetil has been used as an adjunct to corticosteroids in treating AFOP.⁶ In patients who have received decitabine, particular attention should be paid to the patient's respiratory status and cessation of decitabine considered if the patient develops clinical or radiographic findings to suggest acute lung injury.

**REFERENCES**


Nathan T. Connell, MD, is Chief Resident and Assistant Instructor in Medicine.
Heather M. Cassidy, MD, is House Staff Officer in Medicine.
David Berz, MD, PhD, MPH, is Assistant Professor of Medicine.
Eric S. Winer, MD, is Assistant Professor of Medicine.
All are at the Warren Alpert Medical School of Brown University.

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Physician’s Lexicon

The Ubiquitous –nyms of English

An earlier Greek word, meaning the father’s name, evolved into the Latin, *patronymicus*, an adjectival noun meaning from the father’s name. * The Latin, *pater*, meaning father, appears in words such as patrician, patricide, patrimony, patriot and patrilineral. A name descended from the mother, on the other hand, is called a metronym. The Greek, *metro-* , ultimately meaning uterine, appears in words such as parametrium, endometritis and metropolis (mother city). A wide range of English words stem from the older Greek root, *-nym*, both in medical and general usage.

An acronym defines a word composed of the initial letters of a well-known phrase such as FEMA or NATO. The Greek prefix, *akron-*, meaning an extremity or utmost, appears in numerous medical terms such as acrodynia, acromegaly and acромion (the lateral extremity of the scapula) and in general terms such as acrobatic, acrophobia and acropolis. But, contrariwise, words such as acrimony, acridine and acrid are descended from the Latin, *acer*, meaning sharp or bitter; and is earlier related to the Greek *aker*, meaning fields or pastures leading to such English words as acrid, agrarian and acre.

An eponym is a real or mythical name used to describe a larger social unit such as a clan, tribe or nation in words such as Napoleonic or Caesarian. And, in the case of medicine, the name of a disease, procedure or biological group named after its discoverer such as Pavlovian psychology. Fractures such as Colles’, Duverney’s and Potts’ are named after their medical discoverers.

A homonym, from the Greek root meaning the same or similar, as in medical words such as homeopathy, homeostasis and homocentric; and general terms, defining words that sound alike but provide different meanings such as aisle/isle and altar/alter. The list of such words is wondrously long and reflects the many languages which had coalesced to form standard English.

The –nyms of English also include the synonyms, the antonyms and even the contronyms (those words which are spelled alike and pronounced alike but may lead to sharply divergent meanings. The word, cleave, for example, may mean to bring together; or, alternately, to split apart. And the word, awful, may mean filled with awe and wonder or, alternately, something worse than terrible.

And finally, a pseudonym defines a fictitious or false name to hide the real authorship; a pen-name. The Greek prefix, *pseu-*, means false or feigned, as in clinical words such as pseudarthrosis, pseudopod, and pseudotabes. Anatole France (1844-1924) once declared: “Chance is perhaps the pseudonym of God when he did not want to sign.”

– STANLEY M. ARONSON, MD

VITAL STATISTICS

Edited by Colleen Fontana, State Registrar

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

<table>
<thead>
<tr>
<th>Underlying Cause of Death</th>
<th>Reporting Period</th>
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<tbody>
<tr>
<td></td>
<td>December 2009</td>
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<tr>
<td>Diseases of the Heart</td>
<td>Number</td>
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<tr>
<td>Malignant Neoplasms</td>
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<tr>
<td>Cerebrovascular Diseases</td>
<td>201</td>
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<tr>
<td>Injuries (Accidents/Suicide/Homicide)</td>
<td>38</td>
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<tr>
<td>COPD</td>
<td>43</td>
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<td></td>
<td>44</td>
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</table>

<table>
<thead>
<tr>
<th>Vital Events</th>
<th>Reporting Period</th>
</tr>
</thead>
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<tr>
<td></td>
<td>June 2010</td>
</tr>
<tr>
<td>Live Births</td>
<td>Number</td>
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<tr>
<td>1,076</td>
<td>12,153</td>
</tr>
<tr>
<td>Deaths</td>
<td>791</td>
</tr>
<tr>
<td>Infant Deaths</td>
<td>(10)</td>
</tr>
<tr>
<td>Neonatal Deaths</td>
<td>(8)</td>
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<tr>
<td>Marriages</td>
<td>716</td>
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<tr>
<td>Divorces</td>
<td>276</td>
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<tr>
<td>Induced Terminations</td>
<td>391</td>
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<tr>
<td>Spontaneous Fetal Deaths</td>
<td>41</td>
</tr>
<tr>
<td>Under 20 weeks gestation</td>
<td>(39)</td>
</tr>
<tr>
<td>Under 20 weeks gestation</td>
<td>(2)</td>
</tr>
</tbody>
</table>

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

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

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

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

* Rates per 1,000 estimated population
# Rates per 1,000 live births

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

VITAL STATISTICS

Diseases of the Heart
Malignant Neoplasms
Cerebrovascular Diseases
Injuries (Accidents/Suicide/Homicide)
COPD

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– STANLEY M. ARONSON, MD
Ninety Years Ago, December 1920

Elliott Washburn, MD, in “A Wider Use of Tuberculosis Clinics and Hospitals,” described two new kinds of clinics: the occasional, traveling, or itinerant, clinic, and the consultant clinic.” Neither was connected with a dispensary or a hospital outpatient department. The occasional clinic traveled to rural areas and small communities. In New York State, Dr. Washburn noted: “We found in these small communities and in rural districts an astonishingly large amount of active tuberculosis, a very considerable proportion of which was under no supervision.” Clinic staff referred those cases to county tuberculosis hospitals. The consultant clinics grew out of the Framingham Tuberculosis Experiment, where “They induced 12,000 of the total population of 16,000 to come in for a through and complete examination…They found…that for every death from tuberculosis there were 19 living cases, 9 of which were active, and 10 were old cases which probably had never been active.” The TB Consultation clinic relied on outside tuberculosis specialists (“so that the physicians of the community have no hesitancy in sending their cases…for consultation”). Dr. Washburn noted: “The consultation and examinations are entirely free. In most cases the physician comes with the patient and Dr. Bartlett [Chief Examiner] in a great many cases, is able to point out to the physician some point in the diagnosis which the physician has overlooked.”

An Editorial, “Diphtheria in Providence,” recounts the statistics: 80 deaths in 1917, 56 in 1918, 77 in 1919, up to 61 by October 1920. The editorial stressed the efficacy of antitoxin, given on the first day of the disease. “Although there is no doubt that the chief factor in the high mortality…is due to the neglect of parents in calling a physician, not a little blame attaches to physicians.” Dr. Richardson from Providence City Hospital cited the first error: “failure to make a diagnosis.” “Although diagnosis is not always possible on the first call, suspicion is always possible and the patient should always be given the benefit of the doubt.”

Fifty Years Ago, December 1960

“Rhode Island has the Lowest Infant Mortality Rate,” Statement from the Children’s Bureau, Social Security Administration, US Department of Heath, Education, and Welfare, November 2, 1960. The US infant mortality rate (under one year) was 26.0 deaths per 1000 live births in 1956, then 27.1 in 1958. (In 1915, the rate was 99.9; in 1941, 47.0.) “No state within the US matched the low infant mortality rate for Sweden – 15.8.” The rates in the US ranged from 21.3 in Rhode Island to 41.0 in Mississippi.

Roman R. Pe’er, MD, Head, Department of Surgery, Poriah Government Hospital, Israel, and Surgeon-in-Chief, Pro Tempore, Miriam Hospital, contributed “Personal Experience in Management of Hydatid Cyst, with Report of Unusual Complications.” A 37-year old immigrant from Morocco had arrived at the hospital in Israel, complaining of severe pain. After surgery, he improved, but it was not clear why. One year later, the pain recurred. “He came to us complaining bitterly about the state of Israeli medicine, stating that in his native Morocco, he would already have been dead two years ago, but here, because of us, he was neither fully dead nor fully alive.” After surgery and treatment with cortisone, the patient recovered. The conclusion: he had a foreign-body granulomata in fibrous tissue.

Alex M. Burgess, MD, Chair, Publications Committee of the RI Medical Society, announced the appointment of Seebert J. Goldowsky, MD, as editor-in-chief of the journal.

Twenty-Five Years Ago, December 1980

Wendy J. Smith, Editor, described “A New Payment Schedule.” Two Boston surgeons had developed a reimbursement schedule “based on relative difficulty of performing a procedure rather than the amount of time required.”

Ian R.H. Rockett, PhD, in “A Profile of Injury Mortality in Rhode Island, 1980-82,” reported “…trauma accounts for 38.3% of all potential productive years of life lost.” The key causes of injury deaths were traffic accidents, suicide, falls, and homicides.

Manuel E. Soria, MD, and Thomas A. Jordan, MSW, discussed “Clinical Prediction of the Violent Patient,” a discussion spurred in part by the Tarasoff ruling. “While no accurate and specific criteria for predicting violent acts exist, their incidence can be reduced.”
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