Objectives

- At the end of this lecture, you will:
  - 1) Define dementia, mild cognitive impairment, and delirium
  - 2) Delineate between the various types of dementias with regards to their onset, clinical features, and unique qualities
  - 3) Describe how the medications used to treat dementia are believed to work
  - 4) Describe and utilize nonpharmacological interventions for dementia
Normal Memory

- The process by which information is stored, processed, and recalled
- When we are first born, our ability to store information is nearly at its peak but our capacity to recall and process it improves as we age
- Memory is often defined by storage technique
  - Immediate/Sensory
  - Short-term
  - Long-term

Long-Term Memory

- The most important aspect of memory is the ability to recall information long since lost from short-term memory
- Tends to take two forms:
  - Declarative - requires conscious recall in that some conscious process must call back the information; also called semantic memory
  - Procedural - not based on the conscious recall of information but on implicit learning
Memory and Aging

- As we age, our ability to recall information and store new information begins to decline
  - The decline generally is minimal and slow, sometimes it is not even noticeable
- In some cases though there is an accelerated impairment of memory consolidation and memory recall that may herald a dementing process
Mild Cognitive Impairment

- Refers to persons with significantly impaired memory who do not meet criteria for dementia
  - There is a memory complaint, preferably corroborated by an informant
  - Objective memory is impaired for age and education
  - General cognitive function is preserved
  - Activities of daily living are not impaired
  - May be a precursor to dementia

Mild Cognitive Impairment

- Can be disturbing for patients experiencing this condition, may provoke anxiety or denial
  - CBT can be helpful in allaying the anxiety and potentially slowing down the progression to dementia
Delirium

- Unlike dementia, this is mostly reversible
- Has a myriad of causes
  - Infection
  - Drugs
  - Hypovolemic states (dehydration and shock)
- Waxing and waning course
- Physical signs present far more commonly than cognitive signs
  - Exception: Hallucinations may be seen in more advanced delirium states

Delirium

- Treatment of delirium is predicated on determining the cause and either treating it (in the case of infection or blood salt abnormalities) or removing the offending cause (drugs)
- Delirium significantly increases morbidity and impacts mortality – patients who experience one episode of delirium are 50% more likely to develop it again and are more likely to die
Medications for Delirium

- **Haloperidol**
  - “gold standard” of treatment, reduces agitation and hallucinations
  - Can be administered IV or IM
  - Can precipitate NMS, no reversal agent available

- **Lorazepam**
  - Can be administered IV or IM
  - No NMS risks, can be administered more frequently
  - Can be disinhibiting, can be reversed with flumazenil

### Table 3: Delirium Vs Dementia

<table>
<thead>
<tr>
<th></th>
<th>Delirium</th>
<th>Dementia</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Onset</strong></td>
<td>Abrupt</td>
<td>Usually insidious; abrupt in some strokes or trauma</td>
</tr>
<tr>
<td><strong>Course</strong></td>
<td>Fluctuates</td>
<td>Slow decline</td>
</tr>
<tr>
<td><strong>Duration</strong></td>
<td>Hours to weeks</td>
<td>Months to years</td>
</tr>
<tr>
<td><strong>Attention</strong></td>
<td>Impaired</td>
<td>Intact early; often impaired late</td>
</tr>
<tr>
<td><strong>Sleep-wake</strong></td>
<td>Disrupted</td>
<td>Usually normal</td>
</tr>
<tr>
<td><strong>Alertness</strong></td>
<td>Impaired</td>
<td>Normal</td>
</tr>
<tr>
<td><strong>Orientation</strong></td>
<td>Impaired</td>
<td>Intact early; impaired late</td>
</tr>
<tr>
<td><strong>Behavior</strong></td>
<td>Agitated, withdrawn or depressed, or combination</td>
<td>Intact early</td>
</tr>
<tr>
<td><strong>Speech</strong></td>
<td>Incoherent, rapid/slowed</td>
<td>Word-finding problems</td>
</tr>
<tr>
<td><strong>Thoughts</strong></td>
<td>Disorganized, delusions</td>
<td>Impoverished</td>
</tr>
<tr>
<td><strong>Perceptions</strong></td>
<td>Hallucinations/illusions</td>
<td>Usually intact early</td>
</tr>
</tbody>
</table>

Dementia

- Evidence from the history and clinical assessment that indicates significant cognitive impairment in at least one of the following cognitive domains:
  - Learning and memory
  - Language
  - Executive function
  - Complex attention
  - Perceptual-motor function
  - Social cognition

Dementia

- The impairment must be acquired and represent a significant decline from a previous level of functioning
  - If they were Ph.D. level before and are now Pre-K level, much more significant than someone with MR experiencing a similar decline
- The cognitive deficits must interfere with independence in everyday activities
  - Specifically feeding, bathing, clothing, transportation, shopping, managing money
Dementia

- In the case of neurodegenerative dementias such as Alzheimer disease, the disturbances are of insidious onset and are progressive, based on evidence from the history or serial mental-status examinations.
- The disturbances are not occurring exclusively during the course of delirium.
- The disturbances are not better accounted for by another mental disorder (e.g. major depressive disorder, schizophrenia).

Dementia Impairments

- Through the course of the illness, patients may experience any or all of the following deficits:
  - **Prosopagnosia** – inability to recognize faces.
  - **Astereognosia** – inability to recognize objects when touched.
  - **Anosognosia** – inability to recognize illness or deficit.
  - **Phonagnosia** – inability to recognize familiar voices.
  - **Primary progressive aphasia** – gradual loss of language skills including sentence construction, grammar and syntax, and the ability to name objects when seen.
Dementia Impairments

- Additional impairments can include REM sleep behavior disorder, restless legs syndrome, insomnia, hallucinations, delusions and aggression.

Diagnosing Dementia

- Tends to be less lab-driven and more image driven
  - No specific labs for dementia although elevated ammonia or WBCs, evidence of UTIs or blood salt abnormalities may point to delirium
  - MRI superior to CT in precision but cannot be used alone to make a diagnosis of dementia
  - Definitive diagnosis comes from autopsied brain tissue
    - Researchers have found a new protein that lights up tau protein in the living brain, may allow for diagnosis before death
  - Neuropsychological testing sheds more light on a dementing process
Neuropsychological Testing

Screening tests:
- MMSE – Mini-mental Status Exam
- MOCA – Montreal Cognitive Assessment
- ADAS-Cog – Alzheimer’s Dementia Scale, Cognitive portion

Confirmatory tests:
- Graduate Record Examination - for well-educated individuals who test well on screens yet show signs of cognitive impairment
- Functional Capacity Assessment – helps measure a patient’s functional status and their cognitive impairment’s impact on their ADLs
- Clinical Dementia Rating – assesses function in memory, orientation, problem solving, judgment, community affairs, home and hobbies, and personal care

<table>
<thead>
<tr>
<th>Lecture 1 – Mini mental state examination (MMSE)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Temporal orientation (5 points)</td>
</tr>
<tr>
<td>What is the approximate time?</td>
</tr>
<tr>
<td>What day of the week is it?</td>
</tr>
<tr>
<td>What is the date today?</td>
</tr>
<tr>
<td>What is the month?</td>
</tr>
<tr>
<td>What is the year?</td>
</tr>
<tr>
<td>Spatial orientation (5 points)</td>
</tr>
<tr>
<td>Where are we now?</td>
</tr>
<tr>
<td>What is this place?</td>
</tr>
<tr>
<td>In what district are we or what is the address here?</td>
</tr>
<tr>
<td>In which town are we?</td>
</tr>
<tr>
<td>In which state are we?</td>
</tr>
<tr>
<td>Registration (5 points)</td>
</tr>
<tr>
<td>Repeat the following words: GAR, VASE, BRICK</td>
</tr>
<tr>
<td>Attention and calculation (5 points)</td>
</tr>
<tr>
<td>Subtract: 100 - 7 = 93.7 = 86.7 = 79.7 = 72.7 = 65</td>
</tr>
<tr>
<td>Remote memory (5 points)</td>
</tr>
<tr>
<td>Can you remember the 3 words you have just said?</td>
</tr>
<tr>
<td>Naming 2 objects (2 points)</td>
</tr>
<tr>
<td>Watch and pen</td>
</tr>
<tr>
<td>REPEAT (1 point)</td>
</tr>
<tr>
<td>&quot;NO IFS, ANDS OR BUTS&quot;</td>
</tr>
<tr>
<td>Stage command (5 points)</td>
</tr>
<tr>
<td>“Take this piece of paper with your right hand, fold it in half, and put it on the floor”</td>
</tr>
<tr>
<td>Writing a complete sentence (1 point)</td>
</tr>
<tr>
<td>Write a sentence that makes sense</td>
</tr>
<tr>
<td>Reading and obey (1 point)</td>
</tr>
<tr>
<td>Close your eyes</td>
</tr>
<tr>
<td>Copy the diagram (1 point)</td>
</tr>
<tr>
<td>Copy two pentagons with an intersection</td>
</tr>
</tbody>
</table>

Table 1 - Classification of the categories evaluated by the Clinical Dementia Rating.

<table>
<thead>
<tr>
<th>Impairment Level</th>
<th>None (0)</th>
<th>Questionable (0.5)</th>
<th>Mild (1)</th>
<th>Moderate (2)</th>
<th>Severe (3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Memory</td>
<td>No memory loss or slight inconsistent forgetfulness</td>
<td>Consistent forgetfulness, partial recall of events</td>
<td>Moderate memory loss, more marked for recent events; difficulty with daily activities</td>
<td>Severe memory loss; only highly learned material retained</td>
<td>Severe memory loss; only fragments remain</td>
</tr>
<tr>
<td>Orientation</td>
<td>Fully oriented</td>
<td>Fully oriented except with slight difficulties with time relationships</td>
<td>Moderate difficulty with time relationships, oriented in familiar areas</td>
<td>Severe difficulty with time relationships, almost always disoriented to place</td>
<td>Oriented to person only</td>
</tr>
<tr>
<td>Judgement &amp; Problem Solving</td>
<td>Solves everyday problems, such as financial affairs; judgement preserved</td>
<td>Slight difficulty in solving problems, similarities and differences</td>
<td>Moderate difficulty in handling problems, similarities and differences, social judgement maintained</td>
<td>Severely impaired in handling problems, similarities and differences, social judgement impaired</td>
<td>Unable to make judgements or solve problems</td>
</tr>
<tr>
<td>Community Affairs</td>
<td>Independent functioning in jobs, shopping, social groups</td>
<td>Slight impairment in these activities</td>
<td>Is not independent in these activities; appears normal to casual inspection</td>
<td>Is not independent outside home, appears well enough to be taken to events outside the home</td>
<td>Is not independent outside the home; appears too ill to be taken to events outside the home</td>
</tr>
<tr>
<td>Home and Hobbies</td>
<td>Daily life at home, hobbies and intellectual interests well maintained</td>
<td>Daily life at home, hobbies and intellectual interests slightly impaired</td>
<td>Slight impairment of tasks at home, more difficult chores, hobbies and interests are abandoned</td>
<td>Only simple chores are maintained, restricted interests, poorly maintained</td>
<td>No significant function at home</td>
</tr>
<tr>
<td>Personal Care</td>
<td>Fully capable of self-care</td>
<td>Fully capable of self-care</td>
<td>Needs assistance</td>
<td>Requires assistance in dressing and hygiene</td>
<td>Requires much help with personal care, frequent incontinence</td>
</tr>
</tbody>
</table>
Dementia Types

- There are numerous types of dementias, classified typically by their clinical features or their causes
  - Alzheimer’s Dementia (DAT)
  - Frontotemporal Dementia (FTD)
  - Dementia with Lewy Bodies (DLB)
  - Vascular Dementia (VaD)
  - Wernicke-Korsakoff Syndrome (WKS)
  - Normal Pressure Hydrocephalus (NPH)

Alzheimer’s Dementia

- Age of Onset: 60+
  - Exception: Autosomal dominant forms will occur much earlier, as early as the 5th decade of life (e.g. Down Syndrome patients)
- Initial Features:
  - Declarative memory becomes impaired first; procedural memory and motor learning rarely affected until late in the disease
- Later Manifestations:
  - Procedural memory and motor learning decline, primitive reflexes emerge (glabellar reflex, Babinski sign)
Alzheimer’s Disease

- Unique Features:
  - Neuritic plaques composed of beta amyloid precursor protein (BAPP)
  - Neurofibrillary tangles comprised of tau protein (a microtubule binding protein)
  - Nucleus basalis of Meynert becomes damaged (prevents the brain from manufacturing enzymes necessary to make acetylcholine)
  - ApoE4 often targeted as one of the primary genetic causes of plaque formation

Alzheimer’s Imaging
Dementia with Lewy Bodies

- Age of Onset: 60+
- Initial Features:
  - REM sleep disturbances become more common, REM sleep behavior disorder may emerge
  - Parkinsonian features develop
- Later manifestations:
  - Significant proportion of DLB patients experience hallucinations (visual far more common than auditory)

Dementia with Lewy Bodies

- Unique Features:
  - Neurofibrillary tangles comprised of alpha-synuclein instead of tau
  - Sudden changes in alertness and cognition far more common than with DAT; noncognitive features predominate in early stages of DLB
DLB v DAT

Frontotemporal Dementia

- **Age of Onset:** 50+
- **Initial Features:**
  - Anomic aphasia, paraphasias, and reticence (seen in more severe stages as well)
  - Prominent apathy with disinhibition
- **Later Manifestations:**
  - Kluver-Bucy Syndrome
  - Broad decline in insight, social skills, interpersonal conduct, and executive function
Kluver-Bucy Syndrome and FTD

- A clinical constellation of signs and symptoms that is not unique to FTD but is often seen as being pathognomic due to its resultant effects stemming from temporal lobe damage.
- Consists of three of the following:
  - 1) Amnesia
  - 2) Docility
  - 3) Hyperphagia
  - 4) Hyperorality
  - 5) Hypersexuality
  - 6) Visual agnosia

Frontotemporal Dementia

- Unique Features:
  - Many features seen in late stage DAT are seen in early stage FTD (paraphasias, apathy)
  - Memory impairments are more subtle with FTD than with DAT, DLB, or VaD
  - Frontal and temporal lobe atrophy prominent on MRI; temporal and parietal atrophy seen more in DAT
Vascular Dementia

- **Age of Onset:** Variable
  - Very dependent on the severity of the brain damage due to CVAs, can occur as early as the 4th decade but more common in the 6th and 7th decades

- **Early Stages:**
  - Focal and lateralized signs dominate cognitive impairments
    - Dysarthria, hemiparesis, hemianopsia, and ataxia
    - Pseudobulbar palsy and aphasia may complicate the dementia and its expression

- **Later Manifestations:**
  - Physical and mental declines occur simultaneously, then progress in a stepwise fashion
Vascular Dementia

- Unique Features:
  - As noted under “Later Manifestations”, progresses asymmetrically (not stepwise, may skip steps or have them occur together)
    - Memory may not even be impaired at first, only executive functioning as seen with strokes
  - Has preventable risk factors
    - Hypertension
    - Diabetes
    - Smoking

VaD Imaging

A.  
B.  
C.  
D.
### TABLE: COMMON TYPES OF DEMENTIA

<table>
<thead>
<tr>
<th>Type of Dementia</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alzheimer’s disease</td>
<td>Represents up to 85% of all dementia, affects about 4.5 million Americans. Overall prevalence ~1%, climbing to 50% after age 85 years. Without new, effective treatments, 16 million Americans will be affected by 2050. Slow onset, steady decline. Involves neurofibrillary tangles, amyloid plaque, and brain atrophy.</td>
</tr>
<tr>
<td>Vascular dementia</td>
<td>Previously called multi-infarct dementia. Associated with vascular risk factors: hypertension, diabetes mellitus, arterial disease, and smoking. Many subtypes and etiologies (e.g., lacunar lesions, hemorrhagic lesions, mixed dementia).</td>
</tr>
<tr>
<td>Levy Body dementia</td>
<td>Represents 15% to 25% of all dementias. Often unrecognized as dementia because cognitive deficits fluctuate from hour to hour and day to day. Visual hallucinations during periods of confusion common.</td>
</tr>
<tr>
<td>Frontotemporal dementia</td>
<td>Originally called Pick’s disease. Associated with frontal and temporal anterior lobe atrophy. After age 35 years, onset after age 75 years rare. Behavioral changes and language problems are noteworthy, but visual-spatial and memory skills remain intact.</td>
</tr>
</tbody>
</table>

Adapted from references 7-16.

---

### Normal Aging vs. AD vs. VaD vs. DLB vs. FTLD

<table>
<thead>
<tr>
<th>Normal Aging</th>
<th>AD (Alzheimer’s disease)</th>
<th>VaD (Vascular dementia)</th>
<th>DLB (Dementia with Levy bodies)</th>
<th>FTLD (Frontotemporal lobe dementia)</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Reduced speed of mental processing and choice reaction times</td>
<td>• Short-term memory loss, impaired executive function, difficulty with activities of daily living, time and spatial disorientation, language impairment, personality changes</td>
<td>• Impaired abstraction, mental flexibility, processing speed, and working memory</td>
<td>• Visual hallucinations</td>
<td>• Progressive behavioral and personality changes that impair social conduct (apathy, disinhibition, etc.)</td>
</tr>
<tr>
<td>• Benign forgetfulness that is mild, inconsistent, and not associated with functional impairment</td>
<td>• Verbal memory is better preserved</td>
<td>• Slower cognitive decline</td>
<td>• Spontaneous parkinsonism</td>
<td>• Language impairment</td>
</tr>
<tr>
<td></td>
<td>• Dementia occurs within several months of a stroke</td>
<td>• Executive function deficits are worse</td>
<td>• Cognitive fluctuations</td>
<td>• Possibly preserved episodic memory</td>
</tr>
</tbody>
</table>

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“Reversible” Dementias

- Somewhat of a misnomer
  - Though there is some reversibility, only a small percentage of patients with these forms of dementia actually experience a clinically significant reversal of cognitive decline
- Includes Wernicke-Korsakoff Syndrome and Normal Pressure Hydrocephalus

Wernicke-Korsakoff Syndrome

- Age of Onset: Variable
  - Dependent on the amount of damage done to the vermis and mamillary bodies via thiamine deficiency
- Early Stages:
  - Global confusional state
- Later Manifestations:
  - Gait ataxia (due to vermis damage)
  - Retrograde amnesia followed by anterograde amnesia
Wernicke-Korsakoff Syndrome

- Unique Features:
  - Primarily caused by alcohol consumption – causes thiamine deficiency
  - 25% of WKS patients can improve with IM/IV thiamine administration
  - May present with confabulation and an inability to form new memories

WKS Imaging

Magnetic Resonance Imaging of the Brain

Image courtesy of the National Institute on Drug Abuse
Normal Pressure Hydrocephalus

- Age of Onset: Variable (often 5th decade or later)
- Early Stages:
  - Gait apraxia is often the first sign – also the best prognostic sign, most likely to improve with intervention
- Later Manifestations:
  - Urinary incontinence
  - Dementia – resembles DAT

- Unique Features:
  - May be reversible via an intracranial shunt
    - If gait is the first symptom seen, success rate is fairly high
    - If dementia is the first symptom seen, success rate considerably lower
  - Cause is often idiopathic but can be due to meningitis or subarachnoid hemorrhage
  - Has a classic triad
    - “wet” = urinary incontinence
    - “wacky” = dementia
    - “wobbly” = gait ataxia
Severely depressed patients may experience a dementia-like syndrome in which they have impaired ADLs, cognitive decline, and difficulty finding words or describing emotions.

Differentiated from true dementia by its reversibility when exposed to antidepressant therapies (either pharmacological or psychological) and by its course:
- Pseudodementia – rapid onset, fluctuating cognition
- Dementia – insidious onset, stepwise cognitive decline
Pseudodementia v Dementia

<table>
<thead>
<tr>
<th>DEMENTIA</th>
<th>DEPRESSIVE PSEUDODEMENTIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Progressive onset</td>
<td>Rapid onset</td>
</tr>
<tr>
<td>Long term symptomatology</td>
<td>Short term symptomatology</td>
</tr>
<tr>
<td>Mood variations</td>
<td>Consistently depressed mood</td>
</tr>
<tr>
<td>The patient tries to answer to the questions</td>
<td>Short answers like “I don’t know”, negativism</td>
</tr>
<tr>
<td>Patient is concealing amnesia</td>
<td>Highlighting amnesia</td>
</tr>
<tr>
<td>Constant cognitive decline</td>
<td>Fluctuating cognitive impairment</td>
</tr>
</tbody>
</table>

Table 1: Differential diagnosis between dementia and pseudodementia

Pharmacological Treatment

- The pharmacological treatment of MCI and most dementias involves the use of acetylcholinesterase inhibitors or NMDA-antagonists
Acetylcholinesterase Inhibitors

- All prevent the action of acetylcholinesterase (AChE) from cleaving acetylcholine (ACh), leaving it to act on the memory centers of the brain.
- Tend to work on the cognitive amnestic deficits rather than the neuropsychiatric issues (delusions, hallucinations, agitation, insomnia).
- Includes:
  - Donepezil
  - Galantamine
  - Rivastigmine

Comparing the AChEIs

### Table 1. Pharmacological characteristics of cholinesterase inhibitors.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism of action</th>
<th>Half-Life</th>
<th>Protein-binding capacity</th>
<th>Metabolism</th>
</tr>
</thead>
<tbody>
<tr>
<td>Donepezil</td>
<td>Selective reversible noncompetitive inhibitor of AChE*</td>
<td>50-90 hours</td>
<td>96%</td>
<td>CYP 2D6, CYP 3A4*</td>
</tr>
<tr>
<td>Rivastigmine</td>
<td>Pseudo-irreversible inhibitor of AChE and BChE¹</td>
<td>2 hours</td>
<td>40%</td>
<td>Non-hepatic, metabolized by AChE and BChE</td>
</tr>
<tr>
<td>Galantamine</td>
<td>Reversible inhibitor of AChE, presynaptic modulator of nicotinic AChE</td>
<td>5-7 hours</td>
<td>16%</td>
<td>CYP 2D6, CYP 3A4*</td>
</tr>
</tbody>
</table>

*AChe = acetylcholinesterase  
†BChE = butyrylcholinesterase  
* CYP = cytochrome P-450  
† Adapted from Hsing YVR, Loy-English P.
**NMDA-Antagonists**

- In contrast to the AChEIs, NMDA-antagonists work differently to slow cognitive decline
  - In later stages of most dementias, most of the cells responsible for creating acetylcholine have died, making AChEIs generally ineffective
  - Some studies suggest mild reduction in agitation and insomnia with memantine (the only FDA approved NMDA antagonist for dementia)

**Namzaric**

- Newer combination agent (memantine with donepezil) introduced in 2015
- Only for patients who have been generally stable on both medications
  - One pill versus two or three (helps with once daily dosing)
  - More costly than combining individual medications
Many patients with dementia will experience periods of agitation due to confusion or illness that can be hard to manage without medications.

Most often used agents are the 2nd generation antipsychotics (none are FDA approved for this):

- Risperidone
- Olanzapine
- Quetiapine
Agitation in Dementia

- Side effects from antipsychotics
  - 1) Sedation
  - 2) Delirium
  - 3) Extrapyramidal side-effects/tardive dyskinesia
  - 4) Neuroleptic malignant syndrome
  - 5) Metabolic dysregulation (increased lipids, blood sugars, blood pressures, weight)

Benzodiazepines can be used in emergencies for agitation but rarely used routinely due to risk of falls and increased confusion/cognitive clouding

- Generally not recommended for this purpose
Agitation in Dementia

- Mood stabilizing antiepileptics can be used to lessen agitation
  - Valproic acid
  - Carbamazepine
  - Lamotrigine
- Data lacking for FDA approved use
  - However, many of these agents can be used for TBI patients to lessen agitation and many advanced dementias have the neurological equivalents to TBI

Agitation in Dementia

- Data suggests a combination of dextromethorphan + quinidine (Nuedexta) may be helpful if the agitation is caused by pseudobulbar affect
  - Study of 220 patients showed an improvement in the NPI scale of 1.5 points (one standard deviation) versus placebo
  - Only FDA approved for PBA, often used off-label for non-PBA patients, may worsen agitation in these cases
Psychosis with Dementia

- Often underreported by families and patients alike
- Clinicians should ask of both patients and families...
  - “Do you see or hear things that you question if they are there?”
  - “How do you sleep at night?”
- Presence of hallucinations associated with increased risk of decline and death

Psychosis with Dementia

- Fairly common in dementia, delusions more so than hallucinations (30% versus 10-15%)
- Antipsychotics tend to be the mainstay although there is no FDA approved agent for delusions or psychosis with dementia
Psychosis with Dementia

- Some evidence that classic mood stabilizers may provide benefit
  - Lithium most studied but also the most dangerous for the elderly
  - Lamictal/Depakote may be helpful in low doses (half-normal dosing as for bipolar disorder)
- Pimavanserin (Nuplazid) is an agent approved for Parkinson’s Disease-associated delusions and hallucinations
  - May only work for LBD or Parkinson’s dementia, data limited to studies in those patients.

Insomnia in Dementia

- Sleep-wake cycles often perturbed in dementia
  - REM sleep behavior disorder and restless legs syndrome markedly increased in frequency and severity
- Nonpharmacologic treatments preferred as almost all sleep aids have significant side-effects in the elderly
  - No medications are FDA indicated for insomnia with dementia (most worsen behaviors instead)
REM Sleep Behavior Disorder

- Patients often have a violent dream enactment
  - May attack spouses while asleep, causing harm
- SSRIs, SNRIs and TCAs worsen this disorder
  - MAOIs in theory should eliminate it (MAOIs curtail the ability to achieve REM sleep) but very dangerous with polypharmacy
  - High dose melatonin (10mg) and low dose clonazepam (0.5mg) tend to diminish severity of symptoms

Nonpharmacological Treatments

- In addition to medications, other means may be employed to improve the quality if not the quantity of life in patients suffering from dementia
  - May be better tolerated than medications
- These include behavioral modification interventions (aromatherapy, reminiscence therapy, psychotherapy), nutritional therapies (dietary consults, exercise), and ADL-based therapies (physical and occupational therapy)
Aromatherapy

- A novel way to treat agitation associated with dementia using common scents
  - Some scents appear to trigger relaxation in healthy individuals like lemon oil and lavender oil
  - How they work remains unclear and not every patient will benefit from this treatment

Reminiscence Therapy

- As dementia progresses, short term memory develops marked voids but long term memory is relatively spared until later stages
- Reminiscence therapy is designed to strengthen the long-term memory connections to slow the progression
  - Often puts someone at ease when they can remember something
Psychotherapy

- Cognitive behavioral therapies are useful in reducing anxiety associated with memory loss and functional decline
  - Tend to work early in the dementia process
- Brief interpersonal therapies may also be useful as they focus on small issues that may or may not repeat themselves
  - More useful for later stages of dementia when cognition becomes more impaired

Nutritional Therapy

- Patients suffering from dementia often neglect their metabolic needs and are frequently undernourished or malnourished
- Appetite stimulants have limited benefit in these patients
  - They tend to be dysnosmic or anosmic so food odors don’t stimulate hunger as they should
Nutritional Therapy

- Oral supplements like Boost/Ensure do appear to improve body mass and weight but may not affect mortality, only morbidity.
- Medications like caprylidene may provide MCT (medium chain triglycerides), specifically caprylic triglyceride, the synthesis of which is impaired in DAT.

Exercise Therapy

- May be beneficial to any form of dementia, may provide most benefit for vascular dementias (lowers the number one risk – hypertension).
- Patients may not remember taking an exercise class but will preserve the muscle memory associated with the exercise (cerebellum stores procedural memory, remember?)
Occupational Therapy

- As dementia progresses, procedural memory (recollection of how to perform tasks) becomes more and more impaired; occupational therapy may serve to reinforce what is left and retrain the mind to perform tasks once again.
- Often entails placing the patient in a mock housing situation with a stove, a refrigerator, stairs, and other challenges that a cognitively impaired individual may find daunting.

Placement

- Patients suffering from late stage dementia processes may require placement at skilled facilities for their safety or the safety of others.
- The more severe dementia patients will lash out at loved ones perceiving them to be strangers or enemies, causing emotional and physical harm.
Placement

- These units are designed to excel at the treatment of demented patients but due to funding and training the quality of care will vary
  - Usually a locked unit to prevent wandering
  - May have activity stations in the hallways for patients
- Placement often requires declarations of incapacity from a physician and a court order of incompetence
  - Power of attorney

Conclusions

- Memory does decline slightly as we age but mild cognitive impairment and dementia are neither normal parts of the aging process nor are they inevitable
- Dementia and delirium, though similar, are distinct entities with different treatment modalities
- Psychopharmacology may provide some relief for dementia but the mainstays of treatment should also include nonpharmacological means
Questions?

Black Diamonds
Bunny Slopes
Ski Rentals