Geriatric Medicine
Survival Handbook
(revised edition)

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I would again especially like to single out and thank Dr. Christopher Patterson for his help in providing constructive criticism, priceless feedback, and common sense advice in the development of this; reviewing his marginalia and other pithy comments to the manuscript was a unique, humbling and yet ultimately invaluable and rewarding learning experience.
The cover illustration for this revised edition depicts an *inuksuk* (sometimes called *inukshuk*), a common feature of the Canadian Artic constructed by the Inuit living there. It is a directional stone marker or cairn often in the shape of a person (*Inuk*), and acts to help direct and guide lost travelers across the vastness of the North, else indicate areas good for hunting and fishing.

This handbook has a similar aim, to help guide medical learners across the vast topic of Geriatric Medicine. It is primarily aimed at medical students, Residents and other learners, and can act as a portable guide for approaching common problems encountered with older patients. It is neither fully comprehensive nor authoritative, but will hopefully provide interested learners with a starting place for their own journeys of self directed learning in this ever growing area of medicine.

There are many textbooks and reference materials that can be found in the appendices for the reader who wants a more thorough review of the material covered within.

If you note any errors in this text (be it facts, spelling mistakes, grammar, etc.), please do not hesitate to point these out so they may be corrected in any future editions of this handbook.

*Scattered inside the text are many boxed entries labeled PEARLS. These practical clinical tips, or “pearls of wisdom” are teaching points derived from experience and the “art” of medicine that have been backed up by evidence whenever possible.*

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What is Aging?

Aging is a multidimensional process and refers to the process of "…accruing maturity with the passage of time." It begins with conception and continues throughout life until death occurs. Aging is progressive, ubiquitous and inevitable to all living things.

Normal aging and diseases associated with aging are two separate entities. Normal aging refers to those normal deteriorative processes that all human beings will experience if they livelong enough, such as decreased bone mass, osteoarthritis, and lens cataracts. Diseases that are associated with aging, but not caused by aging and does not occur in all persons (i.e. probabilistic aging) include dementia, hypothyroidism, stroke, and congestive heart failure; while they are common they are not inevitable to all persons, and not all seniors will have them.

Homeostenosis is the concept where normal aging decreases the body's ability to withstand stress and challenges as homeostatic mechanisms decline over time. Our functional capacity and ability to respond to stress progressively declines in a linear or exponential fashion beginning as far back as the third decade. Each system’s decline is independent of changes in other organ systems, and is influenced by genetics, diet, environment and personal habits. The frailty seen in some older adults is a direct result of the attenuation of these normal reserves.
Canadian Aging Demographics

- Seniors are the fastest growing population group in Canada and the industrialized world.

- In 2000, there were about 3.8 million Canadians aged 65 and over, up 62% from 2.4 million in 1981. The senior population has grown about twice as fast as the overall population since the early 1980s. As a result, more than one out of every 8 Canadians is now a senior.

- In 2000, 13% of the populations were seniors, up from 10% in 1981 and 8% in 1971; this was 2.5 times the figure in 1921, when only 5% of people living in Canada were seniors.

- It is projected that by the year 2026 there will be over 7.7 million Canadian seniors, or 21.4% of the population.

- Seniors aged 85 and over represent the fastest growing segment of the senior population (see graph below); one in ten seniors now fall into this age category. In 2000, there were over 400,000 Canadians aged 85 and over, up from 140,000 in 1971 and 21,000 in 1921.

**PEARL:** Because of these demographic trends, how seniors are served by the health care system will become an increasingly important issue over time.

*Source: Statistics Canada*
DEBUNKING MYTHS ABOUT AGING...

Myth 1: To be old is to be sick and a burden on others

Fact: In 1998, there were about 3.7 million Canadians aged 65 and older. Most Canadian seniors living at home describe their general health as positive; in 1997, 78% said their health was either good (38%), very good (28%), or excellent (12%), while only 16% reported their health was fair and just 6% described it as poor. The majority of Canadian seniors also live at home; in 1996, 93% of all people aged 65 and over lived in a private household. 58% of them lived with either their spouse or a common-law partner. Another 7% lived with members of their extended family, such as the family of an adult child, while 29% lived alone and 2% lived with non-relatives.

Myth 2: Older Canadians contribute nothing to society.

Fact: The majority of seniors (58% in 1997) participate in informal volunteer activities outside their home, and a fair number continue full or part time paid work also. In 2000, close to three-quarters of a million Canadians aged 65 and over, 18% of the total senior population participated in some kind of formal volunteer activities. In 1996, 37% of all seniors provided some sort of household or personal assistance to others, 17% helped out with child care; 21% did household maintenance; another 21% helped with shopping, transportation or financial activities; 27% provided emotional support; and 35% checked up on others by visiting or telephoning. They all pay taxes, and have done so for many years.

Myth 3: The majority of older persons are senile or demented

Fact: The prevalence estimates from the Canadian Study on Health and Aging suggest that only 252,600 or just 8.0% of all Canadians aged 65 or over meet the criteria for dementia; that means 92% of those over 65 do not have dementia. Most seniors with Alzheimer's or other dementia live in a health-related institution. In 1995, 78% of all those aged 65 and over with this condition resided in long term care.

Sources: Health Canada: Division of Aging and Senior
Derived from Statistics Canada figures
Biological Theories of Aging

No one knows why we age, and the upper limits of the human life span, about 120 years, have not altered over the interval of recorded history despite advances in preventative health care and medicine that have occurred over the last few centuries. While many more persons can live longer today because of these advances (the so-called “rectangularization” of the survival curve where the % of individuals who live to a specific age approaches a more rectangular form over time) there still appears to be an upper limit of ~12 decades to our maximum life span.

Using the tools of molecular and cellular biology along with modern genetics various investigators have proposed a variety of hypotheses for why we age. Below you can find some of these current theories of aging, and none of them are mutually exclusive.

<table>
<thead>
<tr>
<th>Theory</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>DNA damage theories</strong></td>
<td>Various cellular mechanisms constantly repair ongoing occurring DNA damage (i.e. caused by radiation, mutation). The repair efficiency is positively correlated with life span, and decreases with age.</td>
</tr>
<tr>
<td><strong>Oxidative damage / Free radicals</strong></td>
<td>Life span is inversely proportional to the extent of oxidative damage caused by unstable &amp; reactive chemical compounds and directly proportional to antioxidant activity.</td>
</tr>
<tr>
<td><strong>Error catastrophe</strong></td>
<td>Faulty DNA/RNA transcriptional and/or RNA translational processes produce ineffective or toxic proteins.</td>
</tr>
<tr>
<td>Programmed Aging</td>
<td>Cells are programmed with a specific, finite number of divisions, &amp; cell death occurs when this number is achieved (i.e. the “Hayflick limit” of ~50 doubling of cells seen in tissue culture for fibroblasts). Telomeres (proteins that act like plastic ends of shoelaces to seal the ends of chromosomes, shorten with each division; once the telomere is gone, the end begins to &quot;fray&quot;). This has been suggested as the biological “clock” for aging</td>
</tr>
<tr>
<td>Apoptosis</td>
<td>Programmed cell death induced by extracellular signals or “gerontogenes” that tag a cell for removal by phagocytosis.</td>
</tr>
<tr>
<td>Cross Link Theory</td>
<td>Chemical bonds form between and within molecules and affect function (e.g. cross-linking in collagen causes loss of elasticity in blood vessels).</td>
</tr>
<tr>
<td>“Wear-and-tear”</td>
<td>Ordinary insults and injuries of daily living accommodate and decrease the organism's efficiency to subvital levels.</td>
</tr>
<tr>
<td>Immunological Theories</td>
<td>Damage to the immune-system makes the body vulnerable to disease. B and T cells are less effective and less numerous as we age.</td>
</tr>
<tr>
<td>Neuro-endocrine Theories</td>
<td>Failure of cells with specific integrative functions (in the pituitary, thyroid, adrenal, pancreas, and gonadal glands) brings about gradual homeostatic failure</td>
</tr>
<tr>
<td>Age versus Cancer theory (Jan 2002)</td>
<td>Aging may be a side effect of the natural safeguards that protect us from cancer. Over expression of tumor suppressor genes in transgenic mice (i.e. ( p53 )) causes premature aging</td>
</tr>
</tbody>
</table>

PEARL: The oldest living person with a valid birth certificate was a 122-year-old woman named Jeanne Calment from France who was born in 1975 and who died in 1997.
Life Expectancy

**Definition:** The number of years a person would be expected to live from the
day he or she was born (for life expectancy at birth) based on mortality statistics
at the time.

For a person was born in the mid 1960s (and having survived to age 20), the
average Canadian male would live to 71.5 years and the average Canadian
female would live to 76.7 years. In Canada, life expectancy has increased by an
average of seven years for men, and 13 years for women since 1920. The
reasons for this increase in the industrialized nations are mostly related to
improvements in public health (including sanitation and infection control), and
decreases in infant mortality.

Women outlive men, and twice as many women as men live to age 80. Reasons
for this include male employment hazards (job related accidents, exposure to
carcinogens), lifestyle choices (more male smokers and heavy consumers of
ETOH), and the protective effects of estrogen from cardiovascular disease (MI,
strokes). As a result of this discrepancy, many elderly women are widowed
(52%) and live alone (40%), while more older men are married (78%) and live
with a partner (85%) who is their caregiver.

Older adults are survivors. Many of them have managed to avoid or minimize
health problems that cut short others in their age cohort (i.e. MI, strokes,
cancer); ironically, the older you live, the tougher you seem to be, and you
increase your chances of living even older.

Because of this survivor effect, (and providing the person has no mortal
disease), as a rule of thumb:
- At age 65, life expectancy is ~15 years (male=14.2 years, female=18.6
  years).
- At age 75, life expectancy is ~10 years.
- At age 85, life expectancy is ~5 years.
- And if they live to age 95, they may have another 2-3 years of life
  remaining.
Normal Age-Related Physiologic Changes & Their Consequences:

Many normal changes can lead to clear clinical consequences (i.e. arterial stiffening leading to hypertension, absence of estrogen in post-menopausal females accelerating bone loss).

Not all these changes with aging are necessarily bad; for example, autoimmune disease may “burn out” in later life. However, you should be aware of some general themes, which may explain why disease presentation in the elderly can be atypical (i.e. diminished perception of acute pain may alter the presentation of pancreatitis, or myocardial infarction, etc.), and older adults are more likely to die because of a pneumonia (decreased immune system, decreased respiratory function and reserve).

PEARL: Please remember that aging is heterogeneous; although these changes occur with aging; there is much individual variation that occurs among and between individuals. There are moribund 65 year olds, and 93 year olds who run marathons.

General

- **Temperature regulation: Tendency to hypothermia:**
  - Decreased subcutaneous fat, decreased sweating, decreased shivering, decreased awareness to cold, less autonomic vasoconstriction, decreased metabolic rate, decreased heat generated in response to eating
  - Medical conditions: drugs (ETOH, TCA, ASA, acetaminophen), hypothyroidism, malnutrition, hypothalamic and Central Nervous System (CNS) dysfunction, dementia, Parkinson’s Disease, stroke.
  - Extrinsic factors; poverty (unable to afford heating oil),

- **Fluid Homeostasis: tendency to dehydration:**
  - Reduced Total Body Water (up to 30% more body fat)
  - Blunted thirst sensation, less drinking response, lower renal plasma flow, urine concentrating ability reduced
Aging Changes in Specific Organ Systems:

**Oral Cavity**
- 40% of those >65 are edentulous, mostly because of neglect rather than any natural age related process
- risk of caries increases with age as a result of gingival recession and loss of jaw bone density

**Voice**
- Ossification of the laryngeal cartilages causes stiffness; prevents vocal cords coming together while speaking, resulting in a weaker, breathy voice.
- In males, the vocal cords become thin and atrophied with age, resulting in a higher pitched conversational voice.
- In females, loss of hormonal influence leads to vocal cords becoming more edematous after menopause, resulting in a lower pitched voice.

**Eyes**
- **Presbyopia** (loss of lens accommodation) due to hardening & thickening of the lens (making it opaque) and decrease in muscle tone
- Decreased visual acuity because of narrowed pupil, fewer rods (cones spared) so poorer night vision; there is also the need for more light to reach the retina (on average, an older person needs 4x more light than a younger person); additional problems with depth and colour perception
- Flattening of the corneal surface (with diminished refraction) and clouding of lens

**Ears**
- **Presbyacusis**: high sound frequencies lost and impaired speech discrimination
- More prone to excess cerumen (ear wax) occlusion of ear canal, which becomes narrower and more tortuous.
Respiratory
- Age related changes resemble emphysema:
- Loss of elastic recoil
- Early airway closure (and more dead space where you are ventilating non-perfused lung)
- Decreased arterial P02 (-4 mm/decade)
- Decreased flow rates, FEV$_1$, and Vital Capacity
- Stiffer chest wall & weak muscles
- Increased dead space

Endocrine
- Progressive decline in carbohydrate tolerance and increasing insulin resistance
- Decreased aldosterone, renin,, calcitonin, and growth hormone.
- Slightly decreased (or no change) in thyroid hormones T3 and T4, cortisol, insulin, epinephrine, parathyroid hormone (PTH), and 25-hydroxyvitamin D.
- Increase in follicle-stimulating hormone (FSH), leutinizing hormone (LH), and norepinephrine.
- In women, decreased estrogen (post menopause) & prolactin
- In men, decreased testosterone in some (so-called andropause)

Cardiovascular
- Arterial wall stiffening (true and “pseudohypertension”)
- Increased L atrial size and prevalence of S4 heart sound
- Reduced LV compliance caused by: increased myocyte size, increased LV mass, and increased posterior wall thickness (infiltration with lipids, collagen, fat, amyloid)
- Cardiac output decreases at rest/exertion; maximal HR decreases (predicted max is 220-age in years), much less in physically active elders with disease
**Gastrointestinal**
- Reduced Lower Esophageal Sphincter (LES) tone
- Decreased acid production (leading to decreased emptying, less calcium absorption, and differential medication absorption)
- Reduced intrinsic factor
- Decreased liver mass and blood flow leading to reduced oxidative metabolism of some drugs (but not acetylation or sulfuronidation) and protein synthesis
- Increased transit time
- Increased rectal resting tone, decreased contracting pressure

**Genital/Sexual**
- As a rule of thumb, "Everything slows down!"
  - **Male**
    - More intense stimulation needed for erection
    - Erections less firm
    - Ejaculation takes longer, less volume, & intensity
    - Longer refractory period
  - **Female**
    - Estrogen dependent changes [vaginal lubrication slower atrophy (thin epithelium, honeymoon cystitis)]
    - Slower reaction of clitoris
    - Prolonged refractory period

**Hematologic & Immune systems**
- T cell: numbers decrease, delayed hypersensitivity reaction decreased, fewer natural killer and suppressor cells
- B-cell: numbers stable, but make fewer antibodies.

**Renal**
- Smaller kidneys (cortical renal mass decreases ~20%)
- Renal blood flow decreases
- Glomerular Filtration Rate (GFR) progressively decreases; average decline is 50% from age 20-80, but those 80+ show little decline.
- More prone to develop SIADH
Musculoskeletal
- Weight decreases, body fat increases, height decreases (in women especially)
- Sarcopenia (up to 80% decrease in skeletal muscle mass and quality in non active seniors)
- Osteopenia (decrease in bone mass)
- Total body calcium and potassium stores decreases

Dermatological
- Flattening of the dermal-epidermal junction, leading to more thin and fragile skin susceptible to tearing and sheer forces.
- Dermal atrophy, and progressive loss of elastic tissue subcutaneous fat leading to lines and wrinkling, and problems with thermal regulation.
- Loss of melanocytes, and retreat of the dermal plexus leading to pallor and increased vulnerability to sun damage and skin cancer
- Hair graying and hair loss.

Neurological
- Decrease in brain mass and selected loss of cortical neurons (1% per year loss after age 60)
- 20% decrease in cerebral blood flow from age 30 to 70
- Decreased smell and taste perception
- Reduced perception of acute pain
- Impaired postural reflexes
- Increased reaction time (up to 30% longer)
- Alterations of neurotransmitter levels:
  - Increased MAO levels
  - Decreased dopamine (and binding sites), norepinephrine, and a slight decline in GABA levels

Sleep:
- Less sleep required, but sleep latency increased
- Reduced slow-wave sleep (sleep stages 3 & 4)
- Increased REM but shorter; decreased REM latency (may compensate with am napping)
- Increased night awakenings and sleep fragmentation
Disorders More Common in Older Adults

A number of disorders are more common in older adults than in younger persons. They are not caused by aging itself, and do not occur in all older adults.

There is insufficient space to more than list a few of the more common disorders here (more trans-system problems can be found in the section on *Geriatric Giants*), and readers are advised to consult their favourite and trusted Internal Medicine textbook for more details on many of the items listed below.

**Anemia**
- Using WHO criteria (Hb <120 g per L in women and <130 g per L in men), the prevalence of anemia in the elderly ranges from 8 to 44 percent, with the highest prevalence in men 85 years and older.
- A cause can be found in ~ 80 percent of elderly patients.
- The most common causes of anemia are anemia of chronic disease and iron deficiency, but Vitamin B12 deficiency, folate deficiency, GI bleeding and myelodysplastic syndrome are also common causes.

**Atrial Fibrillation (Afib)**
- The prevalence of atrial fibrillation increases with age, about 3% in those in their early 60s, and is up to 10% in those older than 80.
- Afib is associated with a higher risk of cardiovascular death, congestive heart failure and peripheral embolic stroke in older patients.

**Cardiovascular Disease**
- Cardiovascular disease is the leading cause of death in older Canadian men and women.
- Hypertension (HTN), the best predictor of coronary artery disease, increases dramatically in prevalence with aging; isolated systolic HTN occurs in 34% of men and 38% of women aged 65 to 74.
- 50% of Canadian seniors are on no treatment at all for HTN.
- Congestive Heart Failure (CHF) is the most common cause of hospitalization among those aged 65+ in the US.
Cancer

- Lung cancer is the most common cause of cancer-related deaths in both men and women; 68% of cases occur in people over 65
- >50% of breast cancer patients are older than 65 at diagnosis
- Prostate cancer is the most commonly diagnosed cancer among Canadian men (excluding non-melanoma skin cancer) over 65, and is the second most common cause of cancer death (after lung cancer) in this same group.

Cerebrovascular Disease (Stroke)

- One Canadian study estimated 4.1% of people aged 65+ in the community are living with the effects of stroke (CMAJ Sept. 22, 1998; 159).
- Seniors who experienced stroke more often reported their health to be "poor" or "fair" than seniors who had not (69% v. 25%)

Chronic obstructive pulmonary disease (COPD)

- COPD is the fourth-leading killer disease of the elderly in Canada.
- Cigarette smoke is the underlying cause in ~80% to 90% of cases
- Prevalence of COPD for those aged 65-74 years is 5.0%; and for those over 75 years is 6.8%.

Dementia

- Alzheimer Disease (AD) is the leading cause of dementia in Canada (60-70% of all), affecting about 160,000 Canadians, or 3-11% of the general population over 60 years of age

Diabetes Mellitus

- Diabetes has a prevalence of ~13% in persons over 65.
- Type II diabetes mellitus is the most common form of diabetes in the elderly, accounting for about 92% of cases, and is the 6th leading cause of death in men over 65.
- The onset of Type II DM occurs 40% of the time after the age of 60, and there is often a long delay before diagnosis.
- Long-term studies have show that 35% of seniors with diabetes suffer from retinopathy 18% from cardiovascular disease, 30% from peripheral vascular disease and 12% from nephropathy
Hypothyroidism
- One US survey of community dwelling elders found 7% of women and 3% of men between 60 - 89 years of age with this hormone deficiency
- The Canadian Study on Health and Aging (CSHA) found 9% of their study population had subclinical hypothyroidism.

Osteoarthritis
- 85% of people over the age of 70 suffer from osteoarthritis
- It is the number 1 cause of long-term disability in Canada

Osteoporosis
- Estimates from the Osteoporosis Society of Canada suggest that 1.4 million Canadians have osteoporosis, a leading risk factor for bone fractures and death or morbidity after a fall.

Parkinson’s Disease (PD)
- Roughly 1/100 persons in North America are affected
- Average age of diagnosis is 60; the rates rise in persons >70.
- Dementia, a feared complication, increases in prevalence with age; it occurs in approximately 30% of patients with advanced PD.

Pneumonia
- Influenza/pneumonia is a major contributor to deaths and hospitalization in the elderly and is the leading cause of death from infectious disease in Canada.

Prostate Disease
- Symptomatic Benign Prostatic Hypertrophy (BPH) is very common, affecting 40 to 50% of men aged 51 to 60 years, and ~80% of men by age 80.

Skin Disease
- US datum show that 20% of all GP visits for those 65+ are motivated by a skin problem (i.e. rash, pruritis, photo aging, cancer)
- Surveys show that 2/3 of those 65+ have at least one dermatological disorder.
Physiological changes in aging skin when combined with immobility and incontinence predispose elderly persons to have pressure ulcers; prevalence rate in acute care range from 3.5% to 30% and in long term care facilities from 2.4% to 23%.

Sexual Dysfunction
- Erectile dysfunction (ED) is the most common form of sexual dysfunction in elderly men, affecting nearly 70% of men age 70.
- The prevalence of sexual dysfunction in elderly women is largely unknown, but reduced libido, inhibited orgasm and dyspareunia are the most common disorders, and are largely due to the decline of estrogen production.

Urinary Tract Infections
- Prevalence of asymptomatic bacteriuria in the elderly range from 15-60% depending on the study, with twice as many females as men affected.
- The annual incidence of symptomatic bacterial UTIs is estimated to be as high as 10% in those over 65.

Vision Loss
- Thirteen percent of Canadians over age 65 have some form of visual impairment.
- Almost 8% of seniors over 65 (and 11% if over 80) have impairment (blindness in both eyes) sufficient to meet the legal definition of blindness (visual acuity (VA) less than 20/200)
- 11% of Canadians between 65 to 74 years of age & 30% of persons over the age of 75 have Age Related Macular Degeneration (ARMD), the most common cause of irreversible vision loss in seniors.
- Diabetic retinopathy accounts for 35% of all cases of blindness; prevalence increases with age and the duration of the disease.
- The prevalence of lens cataracts sufficient to impair vision (visual acuity less than 20/30) rises from 1% by age 50 to 100% by age 90.
- Glaucoma is present in less than 1.5% of those under 65, 2-3% in those aged 65-74, and between 2.5-7% for those over 75.
Approach to Illness in the older patient:

In older adults, the presenting problem is the “tip of the iceberg” of a pathological process, which often takes careful diagnostic assessment to uncover.

The “iceberg” metaphor reminds us that we see only what is on the surface and what is only immediately obvious; however, in truth 9/10ths is hidden (like the underwater portion of the iceberg), and can only be found if you look for it.

For example, looking beyond the initial presenting problem of falls in an older adult (“the tip of the iceberg”), may reveal not only the reason for this problem, but also one or more diagnoses underlying this and other symptoms.

For example, a certain elderly patient is having recurrent falls. Why?
Under the Tip of the Iceberg…

After some history taking (the falls only occur after exertion), a physical examination (hypertension, dependent crackles, an S3 heart sound, and an elevated JVP) and some simple laboratory tests (oximetry after a short walk shows SpO2 changes from 91% at rest to 84% with exertion), you might discover that the falls are caused because of recurrent **exercise induced hypoxia**.

But why the hypoxia? Because of the true but hidden diagnosis of **Congestive Heart Failure (CHF)** that had not been diagnosed by anyone previously and requires treatment.

But you can’t stop there! Where did this particular iceberg come from (i.e. why is the person in CHF **now**)? And what further hazards need to be anticipated in its treatment?

In Geriatric Medicine you must see not just the isolated problem, but also how that older person’s problem fits in to the larger context of their life.
Geriatric Medicine Models

This all-inclusive way of thinking is in direct contrast to the classic medical model of diagnostic thinking based on an ever-narrowing upside down pyramid of taxonomy that obeys the “law of parsimony”, i.e.:

This conventional top-down “internal medicine” model does not recognize the psychosocial and behavioral dimensions of illness, or the interactions of multiple illnesses (i.e. there could be more than one gunman on the grassy knoll) and is often less useful as a paradigm in Geriatric Medicine.

**PEARL:** You don’t just want to know one reason a person is having falls at home, you want ALL their reasons!
Four Alternative Geriatric Medicine Models:

The Synergistic Morbidity Model (1+2=7)

- The gradual accumulation of ill health events, combined with social and environmental problems, each alone insufficient to impact dramatically on functional performance, can eventually take its toll on a frail older person.

For example, an older woman named Mrs. A (who has osteoarthritis gradually getting worse) is the caregiver for her demented spouse. Mr. A is now up at night wandering, and she cannot sleep and has become exhausted trying to manage him. He is also hoarding things, and has hid her glasses, which she cannot find. She has increasing OA pain, for which she begins to take Tylenol #3, prescribed by her MD. With her continual pain and caregiver burnout, it becomes more difficult to make meals at home, so she only prepares only soup and sandwiches (no vegetables) for the two of them. Depressed, she starts to drink alcohol more heavily than ever before. She is now starting to trip and fall at home, and while at the grocery store (after having locked her husband inside at home) she slips and breaks her wrist after falling on her outstretched hand...

The Misattribution Model (“A” not “B”)

- The problem is attributed to one cause (or even normal aging) whereas in actuality it is due to another cause.

For example, Mr. B, who lives alone and had a diagnosis of CHF (for which he is on a diuretic and digoxin), is having frequent stomach upset, for which is MD prescribes an H2-blocker. In actuality, he his having mild digoxin toxicity from his too high dose of digoxin (which has not been changed in 30 years). With the anticholinergic effects of the new medication, in combination with his...
mild digoxin toxicity he becomes acutely confused. He inadvertently stops all his medications, and is now SOB from acute heart failure; an ambulance is called...

The Causal Chain Model (A->B->C>X)

- A continual series of troublesome health problems in a previously independent older person can gradually wear them down to a point of seeming no return.

For example, Mrs. P, who has with poor balance 2° to a peripheral neuropathy, complains of insomnia and starts taking a benzodiazapine. She then has a fall and sustains a hip fracture, causing her admission to hospital. Immobility from the fracture leads to deconditioning and pressure ulcers, and now Mrs. P. is in too weak to move from her bed and becomes functionally incontinent. The medical staff is thinking about making her Alternate Level of Care (ALC) and discharging her to a nursing home.

The Unmasking Event (“aha!”)

- A sudden stressful external event—such as an injury, acute medical problem or a loss of a caregiver-- can reveal a previously unrecognized and hidden medical problem.

For example, the Smiths’s had been married for 65 years when Mrs. Smith suddenly dies of an MI. Suddenly the family notices that Mr. Smith is not coping at home and is failing badly, more than would be accounted for by grief alone. He doesn’t’ seem to be able to work the home security system, pay the bills, or work the microwave to heat up food his family is bringing over. He is burning pots in the kitchen, and mixing up his medications (if he takes them at all). He denies being depressed, and appears to be cheerful. While he has been somewhat vague over the last few years and poor with his memory, his wife has always been able to jump in as his “peripheral brain”. An assessment by his MD (whom he has not seen in 3 years) reveals signs strongly suggestive of Alzheimer’s dementia.

Please see the reference section for an article on these models
Atypical Presentations of Common diseases

Illness in old age often presents atypically, or is often masked; i.e.:

- Unrecognized dementia, delirium, and/or depression
- Depression w/o sadness, infection w/o fever, CHF w/o dyspnea
- Silent MI or Urinary Tract Infection (UTI) presenting as confusion
- Zoster-Varicella ("shingles") presenting as chest/back pain
- Dementia, depression, presenting as “failure to thrive”
- Iatrogenic illness is common!

Pearl: remember that old age does itself not cause disease, and confusion, incontinence, anemia, etc. are NOT normal parts of aging

Occam’s Razor: William of Occam (or Ockham) was a 14\textsuperscript{th} century Franciscan monk and philosopher who was famous for his use of economy in formal logic. To paraphrase his original writings*, "\textit{when you have two competing theories which make exactly the same predictions, the one that is simpler is the better.}"

In older patients, it is be far more likely that you will see common disorder presenting atypically (i.e. a UTI causing confusion, sepsis without fever) than you would find an uncommon or rare disorder.

In other words, "\textit{When you hear hoof beats, think of horses, not zebras.}"

* His original saying in Latin was "\textit{Pluralitas non est ponenda sine neccesitate}”, which translates into English as "\textit{Entities should not be multiplied unnecessarily}" Another version of his saying: "\textit{Entia non sunt multiplicanda praeter necessitatem.}"
In Geriatric Medicine, it is often more practical to think of problems or syndromes that cross several organ systems rather than starting with distinct disease diagnoses.

“Geriatric Giants” is a term coined by Bernard Isaacs, and the expression refers to the principal chronic disabilities of old age that impact on physical, mental and social domains of older adults. Many of these conditions, commonly misperceived to be an unavoidable part of old age, can in fact be improved. These “Giants” include:

- Cognitive Impairments (i.e. 2’ to dementia, delirium or depression)
- Incontinence
- Postural Instability and Falls
- Caregiver Stress & burnout
- Dizziness
- Iatrogenesis & “Polypharmacy”
- “Failure to Thrive” (often from the above)
- Frailty
- Elder Abuse

See the second section of this handbook for more details on these.
Geriatric Assessment (GA)

- Historically, the elderly (65 and over) accounted for a large proportion of the total hospital days in Canada. Of the 33.9 million hospital days in 1996/97, the elderly accounted for 62% (or 20.9 million days), although they represented only 12% of the population in that year.

- To avoid entry (or re-entry) into hospital of frail older persons, or simply to improve their quality of life by paying attention to matters that have been neglected by others, a Comprehensive Geriatric Assessment can prove very helpful.

- Comprehensive Geriatric Assessments decrease mortality, LTC placement, readmissions to hospital, and minimize the impact of “geriatric syndromes” such as cognitive impairment, urinary incontinence and falls.

- A Geriatric Assessment differs from the conventional medical assessment by its attention to many different functional and cognitive domains, as well as its attention to preventative health and their current socio-environmenal situation.

- Domains covered in an assessment include:
  - Medical history
  - Medications
  - Current living situation & social supports
  - BADLs & IADLs
  - Vision/Hearing/Mobility/Bowels/Bladder/Diet
  - Cognitive status
  - Emotional status

- Such assessments may be done by a Geriatrician working independently, or as part of a interdisciplinary team that often include specially trained nurses (RNs), Occupational therapists (OT), Physical therapists (PT), Social workers (SW), Dieticians, Speech Language Pathologists (SLP) and specialty Pharmacists.

- Assessments can take place in an outpatient clinic, a day hospital, or even home hospital, and treatment goal plans can be developed, changes in
medications can be made, tests arranged, other allied health services coordinated, and recommendations (if any) made for changes in their living arrangements

PEARL: Geriatric assessments only really work if there is follow up (i.e. by a case manager or a repeat visit) and there is medical control over the medications and other advice (i.e. don’t just make investigations and treatment recommendations, get your hands dirty and do them/order them yourself!). And remember to liaise with other physicians involved in their care, especially their Family MD.

- How to identify elderly patients who would benefit from such an assessment (i.e. the frail elderly)? Strongly consider if they have three or more of the following “Red Flags”:
  - >75 years
  - Needs help with ADLs/IADLs by CCAC or caregiver
  - Lives alone
  - Falls
  - Delirium/confusion
  - Incontinence
  - >2 admissions to acute care hospital/year
  - “Failure to thrive”

- Patients can be screened for the need of such a comprehensive assessment when seen in the ER, a Family MD’s office, a senior’s social center, etc.

The next few pages will elaborate on just how to do a comprehensive Geriatric Assessment yourself.
Approach to the Older Patient in Geriatric Medicine

Getting the History

History taking from older patients can be difficult because of deafness, vision impairment, dysarthria (from stroke, or lack of dentures), aphasia (from stroke or a degenerative condition), anxiety, confusion, or language barriers (if we don’t speak their language or vice versa). Otherwise, this is a similar process of information capture to that used for younger patients.

Be patient, and let the patient talk at first; if after 5 minutes you aren’t getting the information you need, change from open ended to closed ended questions.

Getting the History if the Patient has Memory Loss

If the referral is for memory loss, and there is no person with the patient to provide a reliable collateral history, all is not lost.

First ask if the patient knows why they are there to see you. If they deny any memory problem, ask them to tell them a little about themselves; where were they born? Did they have any brothers and sisters? What were their names? Did they go to school together? How much schooling did they complete? What did they do after that? Did they ever get married? How long did they know their spouse before they got married? When did they get married (the exact date). How long ago was that? Did they have any children together? What are their names? When were they born? How old are they now? Do they have any children of their own, and what are their grandchildren’s names? Where are
they living now? Whom do they live with? How long have they lived there? What is the address? And so on.

Keep track of their answers, and go back to verify statements (“So…your son John’s children’s names are what again?”). Gradually, you’ll find out if the person is struggling or not to come up with these answers. If you start to notice this struggle, and patients are having a hard time to account for themselves, or there are odd patches or gaps in their story, gently tell them, “I’m noticing that you are having a little trouble with my questions and with your memory. I’d like to test your memory, if that is okay with you.”, and then jump in with the SMMSE and the clock drawing test (and try and check up on the facts they told you later on with a telephone call to a caregiver or someone who knows the patient. If there is no family member, others can act as collateral witnesses; for example, the Family MD, their CCAC Case Manager, landlord, building superintendent, home pharmacist, etc.)

PEARL: Above is a picture of a highly underutilized investigative tool that is extremely helpful in history taking (although I strongly doubt you’ll be able to find one now with a rotary dial!).

If they admit that their memory is poor, ask them how long this has been a problem; “Is your memory as good as it was 5 years ago” is a good opener. Ask if they can recall any specific examples or problems with their memory. Then go back and use the technique above, exploring for gaps in long term and short term memory, ending with the SMMSE and other more sophisticated memory tests (see section on cognitive tests for Dementia).
If you are lucky enough to have a family member present, ALWAYS obtain collateral history from that person, especially if there are questions surrounding memory (you can do this apart from the patient, or in front of the patient; the former technique is more efficient, while the latter is more forthright and respectful of the patient).

Ask the caregiver if the dates and facts are accurate (with the patient’s permission first; while they are getting ready to change for your examination and as you step out, ask, “Is it okay if I have a quick word with your ___ while you are getting ready?”).

Ask the caregiver about explicit problem with memory loss (i.e. repetition, forgetting names of family members, forgetting to pay bills), evidence of geographic disorientation as well as safety problems because of memory troubles (forgetting to turn off taps or kitchen burners, forgetting to lock the door to the house, wandering)

Also ask about behavioral problems (becoming withdrawn, stopping activities, suspiciousness, thinks people are stealing from them, hallucinations, irritability, apathy and aggression), and how they are managing to cope through these difficult times.

**PEARL:** ALWAYS find out if caregiver stress/burden is playing a role. You can ask the caregivers themselves, in private. There are also a number of caregiver burden screens and questionnaires that may be available for you to have the caregiver fill out.
Other Hints for Easier History Taking

- Use an electronic amplifier (if available; there’s a picture of one below you can buy for about $40 Canadian at Radio Shack), or put in the patient’s hearing aids. Ensure that hearing aids are 1) in their ears, and 2) are working!

- Offer patients their eyeglasses if they seem hard of hearing (many unconsciously “speech read”), and ensure they are on their nose and clean.
- Stand or sit facing when you speak to an older person, and make sure they are aware you are speaking to them (use body language, such as touch, so that they know you are talking to them)
- Offer their dentures so they can talk!
- Speak in a low, deep voice (high frequency sound acuity decreases as we age, which is why many older persons will drop off the letter S when asked to repeat the phrase “No ifs, ands or buts” on the Standardized Mini Mental Examination).
- Make sure the room is well lit and free of other distractions
- Attempt to speak to their "good" ear

PEARL: Older patients aren’t all deaf; assume they can hear you and speak loudly only when asked to do so. Deafness by definition means the 1’ mode of communication is not speech!

PEARL: If they are missing their hearing aids, and you have no electronic amplifier handy, you use your stethoscope in a pinch. Place the ear pieces in their ears, and try speaking into the bell.
Past Medical History

Much along the same lines as you would obtain any other adult. In particular, asked about previous myocardial infarctions (when, how treated), CVA, DM (how long, are they seeing a diabetic specialist), hip fractures (how treated), memory troubles, or recent hospitalizations.

**PEARL:** Describing their current and past medical problems (diagnoses and dates of dx) is a useful way of testing remote memory (but only if you can verify this information by means of an accurate hospital chart or a knowledgeable family member)!

**PEARL:** Past surgical history is also useful (and may be prompted by finding unusual scars of the chest, abdomen, back or peripheries), including joint replacements, bladder lifts, etc. which may suggest problems such as incontinence, osteoporosis, etc.

Medication review:

Many patients don’t know the names or doses of their medications, and use over-the-counter (OTC) drugs, ETOH, herbs, etc. To avoid headaches in figuring what they are taking, use the “Brown Bag Technique” where all meds the patient is taking placed are into a paper (or plastic these days!) bag to be brought in and examined by the MD. Don’t forget lotions, potions, creams, eye-drops, puffers, insulin, oxygen, ear drops, vitamins, herbals, etc.

You can also call their pharmacy for collateral information, including when drugs were last refilled. Also find out what medications they have recently stopped taking, and when they had their last flu shot or pneumovax inoculation. This is also a good way to check for adherence to Rx.

**PEARL:** many medications prescribed to older medications are less than ideal (ie. NSAIDS for OA, Gravol for nausea, cogentin for EPS side effects of Haldol, etc.). Alternatively, many older adults are not on medications that they should be on (ie. Warfarin if A-Fib, Calcium/Vitamin D/Bisphosphonate for osteoporosis/hip fractures), ECASA if IHD, beta-blockers post MI.
ADRs (Adverse Drug Reactions)

This includes allergies (truly nasty stuff like anaphylaxis or Stevens-Johnson syndrome) and intolerabilities (i.e. bronchospasm with beta blockers, cough with ACE-Inhibitors, hyponatremia while on an SSRI, etc.), or predictable side effects (constipation on opioids, diarrhea on donepezil).

Ask what medications have caused true anaphylaxis (i.e. they had symptoms of shortness of breath (SOB), facial swelling, tachycardia, hives, etc.), rashes, GI discomfort (nausea, cramping, diarrhea), etc.?

Review of Systems (ROS):

Much along the same lines as what you would ask any other adult;

<table>
<thead>
<tr>
<th>CNS</th>
<th>Headaches/Syncope/Visual blurring/previous CVA/TIA/ sudden weakness/vertigo/confusion/poor memory/falls/sensory changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>CVS</td>
<td>SOBOE/Chest pain or heaviness/ankle oedema/orthopnea/PND/palpitations/previous MI or CABG or valve replacement</td>
</tr>
<tr>
<td>RESP</td>
<td>Cough/hemoptysis/SOB/Chest pain or tightness/using puffers more Wheeze/change in sputum colour</td>
</tr>
<tr>
<td>ABD</td>
<td>Nausea &amp; vomiting/Pyrosis/Dysphagia/dyspepsia/Melena/dyschezia Change in bowel habit/abdominal cramps</td>
</tr>
<tr>
<td>GU</td>
<td>Dysuria/Frequency/hematuria/Nocturia/sexual dysfunction</td>
</tr>
<tr>
<td>CON</td>
<td>Weight loss/appetite/fevers/chills/night sweats/pruritis/low energy/low mood/ahedonia</td>
</tr>
<tr>
<td>MISC</td>
<td>Any disorders that run in their family? Do they smoke? Did they ever? Current ETOH consumption/Past abuse?</td>
</tr>
</tbody>
</table>

**PEARL:** On a Geriatric ROS always ask about under-reported disorders, for older persons such as bladder or bowel problems, falls, pain, alcohol use (how much and how often). Also ask about vision (when did they last get their eyes checked?) or hearing impairments, recent and updated vaccinations, current diet, level of exercise activity and use of mobility aids (were they fitted for them, or where they just picked up at the Salvation Army thrift store on a whim).
Functional Inquiry:

BADLs  Basic Activities of Daily Living, (aka ADLs, personal self care)

Toileting, feeding, dressing, grooming, ambulation, & bathing.

For each ask if: independent, needs assistance, supervision or entirely dependent on others.

IADLs  Instrumental Activities of Daily Living (using tools & instruments)

Shopping, meal planning & preparation, housekeeping, laundry, transit, financial management, using a telephone, medication management, and driving

Independent, needs assistance, supervision, or entirely dependent?

PEARL:  Some of this material is captured in the FIM (see appendix for details)
Supports & Services:
  - Who specifically helps out at home?
  - Are their formal services in place?
  - Which ones? (i.e. Family MD who visits, CCAC, Case Manager PT, OT, SW, Veteran Affairs, DARTS, etc.).

Brief Biography

- Where were they born and when? Are their parents alive (when did they die)?
- Siblings (and current health)?
- How far did they complete their schooling (0, grade school, high school, college)?
- Their first language (English or? When did they come to Canada if not born here? Did they work, and at what, and for how long? When and why did they retire?
- Are they a war veteran (and so eligible for extra services)?

Current Living Situation:

- Type of dwelling (i.e. isolated farmhouse, apartment, 3 story walk-up?).
- Is it appropriate for their needs (near a bus route if no car, close to family)?
- Assess safety risks (i.e. fire, cold, appropriate clothing, falls, malnutrition, ability to call for help, abuse <financial, physical, emotional>).
- With whom do they live (alone, spouse, child, friend, or ?)
- Health of spouse (if appropriate). What is the diet like now? (makes meals, MOW, skips meals, etc.)

PEARL: for older adults, asking about the current living situation and support network is far more useful than asking about their past family history. Ability to do one’s own shopping correlates well with independence. Asking them how they pay for their rent, groceries and medications is also very helpful (but only if this, and all of the above, is answered accurately)..
**Power of Attorney (POA):**

A power of attorney is a legal document that allows the patient ("the principal") to designate someone else (an "agent") to perform certain duties on your behalf. It can also be drafted to become effective, or remain in effect, in the event they become incapacitated.

- Has this been formally given to someone? When was this done?
- Who is it or are they?
- For Finances, Personal Care, or both?

**PEARL:** This is not the same thing as a “living will” (see below); you need the assistance of a lawyer to draw one up.

**Advance Directives:**

Also known as “living wills”, these are a patient’s choices on what life-saving treatments they wish (or do not wish) to receive in the event that their life is endangered. They are ideally decisions made previously by a capable patient and charted and made known to others so that these wishes can be carried out if the patient is unable to voice their wishes at that time.

- Has this been discussed with the patient and their family?
- What are the patient’s wishes?
- Are these indicated on the chart anywhere?
Physical Examination of the older person:

Observation begins during your interview, and set-up for the physical examination.

Features such as the patient’s dress (neat, slovenly), presence of tremor or fidgety restlessness (? Akathesia= restlessness secondary to neuroleptics), condition of the skin (pallor, cyanosis, bronzing, lesions, bruising), ataxic, antalgic (painful), or bradykinetic (slow) gait when moving, etc., can all be noted.

Appearance: Comment if they are alert, look younger/older than chronological age, visible stigmata,(i.e. on O2/IV/foley, visible scars), have word dyspnea (needs to take a breath in between every few words when speaking), tremor, etc.

Vitals  
BP standing/seated/lying (pick at least two; BP drop on standing? dizzy symptoms?). Heart rate with each. Respiratory rate. Temperature. Current weight.

**PEARL:** Make sure you use the appropriate size BP cuff. Not writing these numbers down is the biggest error made in measuring BP. If BP extremely elevated, do Osler’s maneuver*. Finally, the proper way to auscultate is to use the **bell**, not diaphragm, to avoid falsely overestimate the diastolic pressure.

*Osler’s maneuver* is performed by palpating the pulseless radial or brachial artery distal to the point of occlusion of the artery by the BP cuff. When either of these arteries remains palpable (despite being silent in your ear pieces), the patient is described as "Osler positive".
Geriatric Physical Examination

**HEENT**
Evidence of previous cataract extraction (dysmorphic pupil)
Red reflex okay (if absent, think cataracts)?
Aniscoria (unequal pupil size; if small and occurs with ptosis (eyelid droop), think *Horner’s Syndrome*)
Pink conjunctiva?

**PEARL:** The “100/60” rule may help assess anemia. If the Hemoglobin (Hb) is less than 100, there will be conjunctival pallor. If lines on the hand are no longer pink, but white, the Hb may be less than 60).

Tympanic membranes (TM) visualized? Teeth/Dentures okay (take out the latter if they say they don’t fit right)?
Facial droop? Carotid bruits? Thyroidmegally?

**RESP**
Can they use their inhalers properly (and demonstrate this)?
Kyphosis (suggestive of osteoporosis)?

**PEARL:** When auscultating the lungs, start from bottom, and work up! You may only get one or two good breaths before they poop out.

**CVS**
Normal rhythm or irregular? Are they in atrial fibrillation?
JVP? Normal heart sounds? S3/S4 heart sounds?
Any Murmurs? What do they sound like? Where do they radiate?? (and what do you think it is???)

**ABD**
Ascites? Stigmata of liver disease? (spider angiomas, paper-money skin, leukonychia, caput medusa, ascites, organomegally)
Rectal exam (males for prostate), for occult blood (OB) if concerned

**GU**
Do they smell of urine? Why?
Is there a foley catheter in situ? If so, why? Can it come out?

**EXTR**
Pitting oedema? Chronic stasis changes (loss of hair, hemosiderin peppering)? Ecchymoses? Pressure ulcers? Xerosis/Dermatoses?
Dry axilla (one of the most specific signs of dehydration), +LR=2.8
Abnormal skin lesions (possible/probably skin malignancy)
Joint examination (evidence or OA, or RA)?
Peripheral pulses all present and accounted for?
Nail changes: clubbing, pitting, splinter hemorrhages, spooning (iron deficiency anemia)

CNS
Cranial Nerves? (Don’t forget visual acuity, visual fields, and hearing).
Primitive Reflexes (suggesting frontal lobe disorder)
Muscle wasting? Muscle Tone? Strength? Deep Tendon Reflexes?
Plantars downgoing (ie. Positive Babinski)?
Vibration sense in the toes with a 128 MHz tuning fork?
Foot sensation with a 10 gauge monofilament (if diabetic)?
Involuntary movements (i.e. resting tremor, tardive dyskinesias)?
Cerebellar function tests (touching nose to finger, rapid alternating movements, heel to shin)?
Romberg (balance with eyes open, then closed; if abnormal only with latter, it suggests problem in proprioception)?
Extrapyramidal signs?

GAIT
Stand up from seating without using hands? Shuffling? Poor turns?
“Get up and Go” Test (JAGS 1991; 39: 142-148); see FALLS section.

**PEARL:** Remember that fatigue is a big problem in older persons. Try to be organized so that your patients do not have to be acrobats to complete your assessment.

**PEARL:** Look what’s not there, and should be: gait aid, puffers, hearing aid, Dentures, etc.

**PEARL:** Look at the gait aid (is the rubber worn down on their cane? Brakes okay on rollator walker? What about shoes?)
Cognitive Testing

SMMSE (Standardized Mini Mental State Exam)

The Standardized MMSE is a validated and commonly used screening tool, scored out of 30, for cognitive impairments for whatever cause.

It does NOT diagnose dementia and is insensitive to early dementia or frontal lobe dementia.

A common cut-off is a score of 23 or less out of 30 (the caveat being the test is dependent on language, age, education and ethnicity). Such a cut off has a sensitivity of 69-100%, and a specificity of 78-99% (which translates into a +LR of 9, and a –LR of 0.2

**PEARL:** If the attention portion of the test (i.e. WORLD spelled backwards) is 0/5, this is highly suggestive for delirium.

If recall is 3/3 (perfect), yet the history is positive for cognitive decline this suggests a diagnosis other than Alzheimer’s dementia (such as Frontotemporal Dementia, or even depression).

**PEARL:** If, for whatever reason you cannot do a full SMMSE, there are several sub-score items you still may accomplish that may suggest a dementia (with Likelihood Ratios):

If does not know the year: +LR 37
If could correctly do 3 word recall: - LR 0.6

See appendix A for the actual test.
Clock Drawing Test (CDT)

This is another validated test for cognitive function and is particularly valuable when used with the SMMSE (which it is not part of, but is separate test). Draw a large circle, and ask the patient to place all the numbers in the correct position on this empty clock face, after which you will tell them a time to put the hands on their clock. For hand placement you must use the same time used in the original studies; “Please set hands of the clock to 10 after 11” (remember, the 10 is a virtual 10 represented by the 2 on the clock). Rate as Normal, struggle to complete, or abnormal)

![Clock Drawing Test](image)

**The Positive Likelihood Ratio** for cognitive impairments is 24 if their clock is abnormally drawn.

Below is an example of a “concrete” clock; persons who are concrete (i.e. only capable of objects and concepts perceived by the senses, and incapable of abstract or imaginary concepts) from Vad, FTD, or AD will often put the hands at the 10 AND the 11, which is incorrect. They are “stimulus bound” by the nearby 10.

![Concrete Clock](image)

**PEARL:** If you have only limited time for a quick cognitive screen, the CDT plus three item recall is a good compromise.
Frontal lobe testing

This is done if there is a history of personality and/or behavioral change more striking than memory changes, or if the history suggests greater executive functioning impairments than the SMMSE demonstrates (i.e. in vascular (VaD) or fronto-temporal dementias (FTD).

Tests which are sensitive to frontal lobe dysfunction (dysexecutive syndrome) include:

1. Word generation (abstract thought test): Ask the patient to name as many 4 legged animals as they can in sixty seconds (or vegetables, words beginning with the letter F, etc.); normal is 15+/- 5.

2. Verbal fluency tests with word pairs (“what is the difference between a lie and a mistake?”, river/canal?, child/midget?, etc.).

3. Problem solving: Ask “What would you do if you awoke in the night and smelled smoke?” , “What exactly would you do if you found a flood in your kitchen at home”


PEARL: Although suggested as a test, interpretation of proverbs, is fraught with peril because of differences in cultural upbringing. For example, what does the following proverb mean to you: “The golden hammer opens the door”?

PEARL: Several batteries of tests for frontal lobe syndromes have been developed, with scoring systems. Most recently, the Frontal Assessment Battery (FAB) was published in the December 12th 2000 edition of Neurology. It proposes a 6 domain, scorable bedside test that takes ten minutes to administer, and seems to distinguish patients with FTD from normal persons.
Laboratory tests & Special Investigations

Your choice of laboratory tests & investigations for older adults depends on the clinical context, and the range of diagnostic equipment available to you.

As a rule of thumb, the abnormalities seen in most blood tests have the same significance regardless of age (although healthy adults may have slightly abnormal tests, because most normal values are based on 2 SD from the mean of the local population).

**PEARL: Reference ranges for most laboratory tests follow a bell curve distribution so that 5% of tests will be abnormal even if the person is normal!**

Reference ranges of laboratory tests for the elderly seldom differ from those in the general population, the differences representing normal aging processes. Adequate reference ranges for laboratory testing in the elderly are generally lacking in most labs, as is specific information on persons over age 75, who, ironically, constitute the fastest-growing segment of the population.

Some lab values change with age: i.e. WBC (slight decrease), PaO2 (slight decrease), Creatinine Clearance (significant decrease), Alkaline Phosphatase (30% increase), Total Cholesterol/Lipids (~30% increase)

Some lab values do not change with age: i.e. Hemoglobin (Hb), Platelet count, Bilirubin, Liver Enzymes (ALT/AST), pH, Thyroid Stimulating Hormone (TSH).

**PEARL: older persons with infection or sepsis do not always mount the same WBC response (and may not even have a fever!). If someone is older and confused, but has a WBC is still in the “normal range,” look closely at the absolute neutrophil levels; if you see a rise in this (e.g. a doubling in the “band count”), they may have an occult infection despite having a “normal” WBC.**
Renal Function:

Creatinine Clearance (CrCl) usually declines with age, and many renally cleared medications (antibiotics like Gentamycin and Penicillin, drugs like digoxin, lithium, NSAIDs, ACE-inhibitors and allopurinol) have to be dose adjusted beyond the aphorism of “start low, go slow”.

To estimate a person’s CrCl, you can use the Cockcroft-Gault equation [adjusted below to use SI units which are used in Canadian medical labs for Creatinine, which is measured in µmol/L], which is:

\[
\text{ClCr (mL/second)} = \\
\frac{(140 - \text{age}) \times \text{(weight [in kg])} \times 1.23}{\text{Creatinine (in µmol/L)}}
\]

The above is the ClCr for men. You need to multiply it by a “fudge factor” of 0.85 to get a more accurate ClCr for females.

Note that normal values for serum creatinine range from 70-120 µmol/L and CrCl values range from 1.24-2.24 mL/second.

**PEARL:** For frail elderly patients, using their actual weight, rather than “Ideal Body Weight” as used in the original equation is usually “good enough”.

**PEARL:** A creatinine of 100 in a 90 yo female who weighs 45 kg is NOT normal. This calculates out to a CrCl of 0.39 mL/min. In contrast, a 65 kg man aged 25 with a creatinine of 100 has a CrCl of 1.53 mL/min, four times better clearance!

**PEARL:** many personal data assistants (PDAs) like the Palm Pilot, have available free medical calculators to make this calculation easy. MedMath is one I like to use.
Section 2: Geriatric Giants

Cognitive Impairments
  Dementia
  Delirium

  Depression
  Incontinence
  Falls
  Dizziness
  Orthostatic Hypotension
  Failure to Thrive & Frailty
  Polyphamacy Iatrogenesis, & Optimizing Medications
  Capacity
  Elder Abuse
Cognitive Impairments

**Definition:** A cognitive impairment is a change in how a person thinks, reacts to emotions, or behaves.

The most common differential diagnosis in older adults of an acquired cognitive impairment includes three major categories:

1. **Dementia,**
2. **Delirium**
3. **Depression (temporary and a reversible cause of “pseudodementia”)**

**Dementia**

A diagnosis of dementia is made when newly acquired cognitive impairments are sufficient to interfere with social or occupational functioning in a person without depression or delirium. More often it is other family members, rather than the affected person who notices the first symptoms of dementia.

There are over 70 different causes of dementia, and each has a particular pattern of decline, impairments, and underlying neurohistopathological processes. Alzheimer’s Dementia (AD) is the most common cause worldwide, and accounts for about 65% of cases. Vascular Dementia (VaD), mixed vascular and AD, Dementia Lewy Body (DLB) and Frontotemporal Dementia (FTD) each account for about 10% of cases.

**PEARL:** Remember that the cognitive impairments need to be *acquired* to meet the criteria of a dementia; there must be a decline from a previous level. Persons with mental retardation with impaired mental faculties are not demented unless they start having a decline from a previous level of achievement.

In AD, memory problems usually occur first (losing items, missing appointments), along with difficulties in the performance of complex tasks they could normally do (i.e. complete taxes, driving in strange places, cook a Thanksgiving meal). Other associated problems include word finding difficulties, difficulty with names, inability to follow the plot of a film or TV
show, geographic disorientation (getting lost driving, at the mall, etc.), apathy and disinterest in surroundings, sleep disturbance (sleeping much more in the day time) and difficulties in inhibiting behavior (impulsivity, socially inappropriate actions).

**Dementia Prevalence**

In Canada, dementia affects 8.0% of all individuals over the age of 65, and the prevalence increases with advancing age as per the following table (data from the Canadian Study on Health and Aging).

<table>
<thead>
<tr>
<th>Age range</th>
<th>All</th>
<th>Community</th>
<th>Long Term Care</th>
</tr>
</thead>
<tbody>
<tr>
<td>65-75</td>
<td>2.4%</td>
<td>1%</td>
<td>42%</td>
</tr>
<tr>
<td>75-85</td>
<td>11%</td>
<td>7%</td>
<td>53%</td>
</tr>
<tr>
<td>85+</td>
<td>34%</td>
<td>17%</td>
<td>66%</td>
</tr>
</tbody>
</table>

**Criteria for Dementia (based on DSM-IV):**

A. Impairment in short- and long-term memory
B. At least 1 of the following:
   1. Impairment in abstract thinking
   2. Impaired judgment
   3. Other disturbances of higher cortical function (agnosia, anomia, & visualospatial difficulties)
   4. Personality change
C. Memory impairment and intellectual impairment cause significant social and occupational impairments
D. Absence of occurrence exclusively during the course of Delirium
E. Cannot be accounted for by any nonorganic mental disorder*

- Older patients with depressive symptoms (i.e. hopelessness, excessive guilt, inertia and suicidality may be suffering from *pseudodementia* (i.e, major depression). When the depression improves with treatment, the cognitive impairments may resolve.

**PEARL:** Depressive symptoms are common in Dementia, and some have even suggested that a first time depression in an older adults heralds the transformation of dementia in some patients, a so-called “pseudodepression.”
Potentially Reversible Dementias

Dementias are usually progressive and irreversible. However, there are a number of conditions that may rarely mimic dementia and may reverse if identified and properly treated. These “exceptions to the rule” include:

- Delirium
- Depression: so-called “Pseudodementia”
- Electrolyte disorders (hyponatremia, hypercalcemia, etc.)
- Hypothyroidism
- Wilson’s Disease (rare, and very rarely seen in Geriatric patients!)
- Late onset Psychosis
- Medication side effects (e.g. sedatives, anticonvulsants, antihypertensives, anticholinergics, first generation neuroleptics)
- ETOH overuse/misuse
- Vitamin deficiencies (B-12, folate)
- Obstructive Sleep Apnea
- Normal Pressure Hydrocephalus (although few, if any actually reverse with shunting)
- Brain tumour
- Subdural Hematoma (SDH)
- Sub-acute CNS infections (i.e. syphilis)

**PEARL: More often, treatment improves but does not reverse the cognitive changes**

In one Canadian study [Freter et al. CMAJ 1998;159:657-62], of 196 patients seen in a tertiary care memory clinic, 45 (23.0%) were felt to have a potentially reversible condition identified by history, physical examination, blood testing or CT. However, in only 7 (3.6% of the total) did treatment result in improvement or resolution of the dementia.
PEARL: Misdiagnosis of a dementia does occur, and unfortunately all too often by physicians who only see a person in the throes of an acute delirium while sick in hospital, and mistakenly believe that they have always been this way. If you see a person labeled as Dementia, but there are atypical features, consider revisiting the diagnosis and widening the differential to include the above items.

In addition, there are subtle impairments that can often be only be detected and quantified with special neuropsychological instruments. It is thought that these may herald the beginnings of an early dementia, a form of “pre-dementia syndrome”:

Mild Cognitive Impairment (MCI)

- Refers to mild memory impairment alone (1.5 standard deviations below that of age matched controls) in the absence of global cognitive decline or functional disabilities; it can only be diagnosed by sophisticated neuropsychiatric testing.
- Approximately 30% of those with MCI will decline to dementia within three years (the remainder have stable, mild cognitive impairments that do not appear to progress.
- For these patients uncovering and management of risk factors such as hypertension, elevated lipids and smoking is crucial.
- Some clinicians recommend starting Vitamin E (400 iu po OD) and the active extract form of Ginko Biloba (Egb 761, beginning at 40 mg po TID).
- Clinical trials using COX-2 inhibitors and cholinesterase inhibitors for patients with MCI are also underway.

PEARL: There are other closely related terms for possible “pre-dementia” syndromes such a CIND (Cognitive Impairments Not Dementia) used by the Canadian Study on Health and Aging (and found on population screens) and ARCD (Age Related Cognitive Decline) used in the literature to label these types of persons. In this very hot area of current research, each term has its preferred champion, but MCI is emerging as the preferred designation.
Approach to a Person Suspected of Having a Dementia

The history is the key. When someone is in your office with memory troubles, find out how long there has been a problem, and how steep a decline. If it has taken place over a period of a few years, and is very gradual, it is likely Alzheimer’s Dementia (AD). If more steep a decline, and associated with walking problems, it more likely a Vascular Dementia (VaD)/ mixed VaD + AD. If behavior problems such as irritability and apathy are more prominent than memory issues, it may be a Frontotemporal Dementia (FTD). And if they have memory problems along with visual hallucinations and symptoms of Parkinsonism, it is likely Lewy Body Dementia.

PEARL: Memory in a person with dementia is like an ice-cube that is starting to melt from the top down. While old memories are frozen away and intact, more recent memories are fleeting and melt quickly. A mildly demented person may be able to tell you the name of their third grade teacher, but not be able to tell you what they had for breakfast today. Most recent memories melt earliest in the early stages of dementia, but as the dementia progresses, even those older memories will melt..

Reliable collateral witnesses are key (i.e. it may be the caregiver who has the dementia and is complaining about the other person’s poor memory when it is really their own!). Review the section on History Taking in a Patient with Memory Troubles. Ask for specific examples of memory troubles, and ask if they are getting better or worse as time goes on. Ask about safety issues, and find out if there is a Power of Attorney.

Ensure that you do at least an SMMSE and a Clock Drawing Test at each visit (or regularly every 3-6 months); as important is the absolute score, see how much of a struggle it takes on the test, and note any interval changes in the performance of these cognitive tests.

The physical examination may be completely normal. However, it may reveal focal signs suggestive of a stroke (i.e. up-going toe), a peripheral neuropathy (rule out B-12 deficiency), features of hypothyroidism (slowed reflexes) or primitive reflexes (suggestive of frontal or advanced diffuse cerebral atrophy).
Laboratory Work-Up for Dementia

You should always do screening blood work to look for the so-called “reversible dementias, or factors which may aggravate or even cause the dementia syndrome. The current Canadian Consensus Guidelines recommend only the following screening laboratory tests in the primary care setting:

**Complete blood count (CBC); measurement of thyroid stimulating hormone (TSH), serum electrolytes, serum calcium & serum glucose.**

Note that many other tests may be indicated in individual situations (e.g. liver function tests if excess ETOH, serum B-12 if deficiency is suspected, CXR if lung cancer is suspected, etc.)

Indications for Neuroimaging in Dementia

The 1999 Canadian Consensus Guidelines for the recognition, assessment and management of dementia (see the reference section), only suggest order a CT head in persons suspected of dementia only if one or more the following criteria were present:

1. Age less than 60 years
2. Rapid (e.g., over 1 to 2 months) unexplained decline in cognition or function
3. "Short" duration of dementia (less than 2 years)
4. Recent and significant head trauma
5. Unexplained neurological symptoms (e.g., new onset of severe headache or seizures)
6. History of cancer (especially sites and types that metastasize to the brain)
7. Use of anticoagulants or history of a bleeding disorder
8. History of urinary incontinence and gait disorder early in the course of dementia (as may be found in normal pressure hydrocephalus)
9. Any new localizing sign (e.g., hemiparesis or a Babinski reflex)
10. Unusual or atypical cognitive symptoms or presentation (e.g., progressive aphasia)
11. Gait disturbance
Diagnosis of Dementia

The diagnosis is made clinically, primarily from the history provided by the patient and the caregiver, but also supported by memory problems demonstrated by the patient.

**PEARL:** A diagnosis of dementia should not be conferred indiscriminately upon any patient, as it is a “terminal” diagnosis. Be very sure before making the diagnosis.

Management of Dementia: Breaking the News

- Break the diagnosis gently to the patient and the family/caregiver;
- Tell them “You have a memory problem that is not normal for someone your age. We call this type of memory problem a dementia. We think this type of dementia is (fill in the blank).”
- Give them time to ask questions, offer tissues, etc.
- Only if they ask, offer prognostic information given the type of dementia, stage of the disease, (or a simple “I don’t know” if you don’t know).
- Offer education and support.
- Suggest they contact the local chapter of the Alzheimer’s Society (who can also provide support for caregivers and patients; they do not limit their activities to individuals with AD, but also deal with persons with other types of dementia)

**PEARL:** You may not want to overwhelm the patient and family with all this information on the first visit. Offer a repeat visit(s) to go over this information again. You may wish to provide the family handouts regarding the diagnosis, and brochures on community resources that they can link up with, because they may not remember all the information you are giving them.
Management of Dementia Treatment and Follow-Up

- If no contraindications (i.e. bradycardia, asthma, active peptic ulcer disease or seizures), and if the person has an Alzheimer’s, Mixed, or Lewy Body Dementia, consider starting on a cholinesterase inhibitor (if no recent EKG, get one to ensure the patient does not have a bradycardia, or any conduction abnormality that would prevent them from being treated).
- Consider also starting Vitamin E (at least 800 iu po OD).
- If mild in severity, consider aggressively treating co morbid conditions that can accelerate the dementia (i.e. treat hypertension, hypercholesterolemia, start ECASA if previous strokes, etc.).
- Arrange follow up to see the effects of treatment (if any), and the development of any behavioral problems that need addressing. This should be done at least every 3-6 months under treatment (and more often if there are problems at home). Do a MMSE at least twice a year.
- If there is a case manager available, have them liaise with the caregiver.
- Watch out for caregiver burnout or depression, and ensure that the family know that Dementia support services (CCAC for homemaking, respite care) are available for them to utilize.
- If there is no POA for the patient, strongly suggest the family look into correcting this early on.
- Recommend driving cessation; if this causes problems, compromise that the patient stop driving until an on-road performance based test is available; notify the MOTC if you have any concerns.

**PEARL:** Once the diagnosis is made, consider suggesting planning for eventual relocation at a follow-up visit; this may be necessary if the patient lives along or if caregiver shows any sign of distress or burnout.

However, filling out LTC placement papers at a first visit for a patient with mild dementia is overkill and very threatening.
When to Suggest Long Term Care?

There are several indications that suggest that a person with dementia is no longer able to live in the community in their current place of residence:

- When the patient needs more supervision than the caregiver can provide (usually in moderately-severe or severe dementia, when both memory and personality is lost and the patient no longer recognizes the caregiver)
- Excessive caregiver burnout, depression, or other significant co-morbidity
- Safety issues at home (falls, medication noncompliance, abuse)
- Behavioral issues at home (delusions, hallucinations, wandering, or aggression.
- No control of bowels or bladder, and caregivers unable to manage.
- When home care support services and informal help cannot manage these and other behavioural issues.
Alzheimer's Dementia (AD)

In 1906, Alois Alzheimer first described the disease that bears his name. It is a progressive, neurological disorder that attacks the brain and results in cognitive problems, such as memory loss, impaired thinking, and unwanted behaviors. Alzheimer’s is the most common form of dementia (~65%) and the fourth leading cause of death in North American adults, after heart disease, cancer, and stroke.

Autopsies of the brains of Alzheimer’s patients reveal abnormal Beta-Amyloid plaques, and neurofibrillary tangles. Plaques are composed of abnormally processed amyloid precursor protein (APP) which has been converted to beta amyloid protein. Beta amyloid Protein (BAP) is the protein deposited in insoluble amyloid plaques sandwiched between neurons. When the beta amyloid protein builds up, synapses become “gummed up”, unable to transmit or receive messages.

Neurofibrillary tangles are abnormal fibres in the axons of neurons composed of abnormally phosphoralated filaments of microtubules and a stabilizing protein known as Tau. Normally fibres inside axons act as a form of two-directional cable-cars to shuttle materials from and to the nucleus; the abnormal tangles seen in Alzheimer’s appear to prevent materials from leaving the nucleus and they accumulate in the soma, leading to cellular dysfunction, and cell death.

**PEARL Which came first, nobody knows, but different investigators, dubbed “BAP-tists” and “TAU-ists” champion their own histopathology as being the inciting event leading to neuronal death and eventual Alzheimer’s dementia.**

**Risk Factors for Alzheimer’s Dementia:**

- Increasing age
- Positive Family Hx
- Apo E4 allele (susceptibility marker for AD; E3 is neutral, E2 protects)
- Head trauma ?
- Low education (<4 years; > 10 protective!)
- Systemic hypertension
- Female sex (even after adjusting for more female seniors)
- Down's syndrome (Trisomy 21)
Diagnosis of AD

There is no single clinical test or unique biomarker that can be used to identify AD.

A comprehensive patient evaluation includes a complete health history, physical examination, neurological and mental status assessments, laboratory screening tests, and other tests when indicated (i.e. CT of the brain).

Neuroimaging often reveal no changes, cerebral atrophy, or sometimes leukoariosis (non-specific white matter changes seen next to the ventricles of the brain)

In most cases the diagnosis is made by history, with other tests being done to rule out other causes of the cognitive decline (i.e. silent strokes, metabolic abnormalities) be they reversible or irreversible. While this type of evaluation may provide a diagnosis of probable Alzheimer’s (clinical diagnosis is correct 80% of the time), absolute confirmation requires examination of brain tissue at autopsy (the “gold standard”).

Prognosis of AD

At this point of time, there is no cure for AD. Available medications (cholinesterase inhibitors) appear only to delay the need for institutionalization by about 18 months in those who respond, but the “death date” does not change. Adjuvant agents such as Vitamin E and Ginko Biloba are thought to be somewhat neuroprotective, although the data is somewhat skimpy [See Treatment Section that follows].

Depending on which study you refer to, time from diagnosis to death is about 3-4 or 7-10 years.

**PEARL:** Many persons with Alzheimer’s die from pneumonia if they have no other co-morbid illnesses, and this is felt to be caused by the inability to prevent recurrent aspiration due to the global neurological decline.
Treatment of Alzheimer’s Dementia

Approved treatments for treatment of AD in Canada make use of the **Cholinergic Hypothesis of AD**: that loss of choline acetyltransferase & acetylcholinesterase causes at least some of the cognitive decline that occurs in patients.

This theory is supported by the following facts:

- in early AD, there is selective cell death in subcortical cholinergic neurons from the basal forebrain to the cerebral cortex and hippocampus in AD pts (Whitehouse et al., 1982)
- choline acetyltransferase (CAT), levels are also reduced in post-mortem tissue of AD patients (Bowen et al., 1976)
- decreases in acetylcholine release in AD patients (Nilsson et al., 1986)

Using the building blocks of Choline and ACo-A, the enzyme Choline Acetyltransferase (CAT), located within axon endings of neurons catalyzes the synthesis of Acetylcholine (ACh), which is stored in pre-synaptic vesicles.

Following an action potential, ACh molecules are released by pre-synaptic vesicles into the synaptic cleft, where they interact with post-synaptic receptors. These post-synaptic ACh receptors (muscarinic and nicotinic) eventually opens a post-synaptic ion “gate” (directly or via protein G), allowing cations flood in, and start a new action potential.

ACh in turn is broken down by hydrolysis in a reaction catylized by the enzyme **acetylcholinisterase (AchE)** [some is also broken down by butyrylcholinesterase (BuChE), whose primary substrate is butyrylcholine, but it also hydrolyzes Ach].

By blocking the breakdown of ACh by inhibiting the enzyme that chews it up it is hoped that there will be more ACh left in the synaptic cleft to be involved in normal cognitive processes at the cellular level.
Three acetylcholinesterase inhibitors are currently available in Canada: donepezil (Aricept), rivastigmine (Exelon), and galantamine (Reminyl).

These three agents are covered by under the Ontario Drug Benefits (ODB) for dementia and only when the SMMSE score is between 10 and 26). They are all very expensive, and cost $1875/year for those without coverage.

Each takes about 3 months before you can tell if there has been any therapeutic effect (i.e. lack of decline, or even improvement on cognitive testing such as SMMSE scores; improvement in function (BADL/IADL) or function).

Finally, an adjuvant agent, memantine (Ebixa) is available for patients with moderate to severe dementia. It acts on the NMDA (N-methyl-D-aspartate) receptor, and was recently as a treatment for AD. It’s as expensive as the above agents, and unfortunately not covered under ODB. It’s effects are modest on their own, but are synergestic with any one of the above.
Aricept (donepezil)

- Dosing is OD, starts at 5 mg, and increase to 10 mg after about 4 weeks.
- About 30-40% of people will improve with treatment
- Most common side effect is GI upset, but also bronchospasm in susceptible persons (i.e. those with asthma, COPD).
- Bradycardia can also be a problem; you can’t use if any heart block other than RBBB
- Use Limited Use (LU) code 347 to initiate donepezil, LU code 348 to continue it if it helps.

Exelon (rivastigmine)

- Acetylcholinesterase and Butyryl-cholinesterase inhibitor (found in plaque, but significance uncertain)
- Start at 1.25 mg po BID, and increase to 3 mg BID after 4 weeks, and titrate to 4.5 mg po BID if at all possible
- Not metabolized by the P450 system like donepezil or galantamine
- Probably similar clinical efficacy to others, but may have more GI side effects (nausea in up to 25%); suggest to patients take it with food)
- New topical patch formulation pending approval in Canada Summer 2008

Reminyl (galantamine)

- Another BID dosed drug, available in Canada as of September 2001
- Start at 4 mg BID, and increase to 8 mg after 4 weeks; take preferably with morning and evening meal.
- Derived from flower (Moly) alkaloid; [supposedly antidote for Circe’s atropine; Ulysses’ men thought they were swine when poisoned by her with it in The Odyssey]
- In addition to acetylcholinesterase inhibition, there is nictotinic modulation at both pre and post synaptic level; clinical significance is uncertain.
- Levels of serotonin, glutamate, norepinephrine, and acetylcholine all affected
- Probably similar clinical efficacy when compared to the others.
- Renal excretion; made need to adjust dose if renal impairment
- Not yet available for ODB
Other Possibly Helpful Medications (and will not do any harm)

- **Ginko Biloba (only extract EGb 761 felt active)**
  - A few small studies only
  - Felt to be helpful in MCI, rather than actual dementia
  - Metabolic effect, may be partial agonists to dopaminergic, serotonergic and noradrenergic receptors; can affect cAMP levels, and act as an anti-oxidant
  - Dose is 40 mg po TID of standardized extract
  - Suggest patients check with their pharmacist for appropriate preparation

- **Vitamin B complex?**
  - Individuals with Alzheimer's disease often have low blood levels of vitamin B-12; the reason for the association of low is not clear
  - A recent case control study of AD patients had higher blood homocysteine levels and lower folate and vitamin B-12 levels; measures of nutritional status indicated that the association was not due to dementia-related malnutrition.
  - If normal serum levels, unknown if supplementation useful (? Helpful role in MCI?).
Other investigational therapies for AD:

Many other agents are in development for the treatment of AD, ranging from anti-oxidants, modulators at other receptors (GABA, 5HT), vaccines, even green Mamba snake dendrotoxin, and brain neurogenesis via stem cells. Some of the more promising agents for treatment of AD currently in the pipeline include:

AN-1792

- vaccine against B-amyloid precursor protein
- Human trials started in September 2001 were halted January 2002 after several patients developed cerebral inflammation).

Talsaclidine & CI-1017

- Agents acting on muscarinic and nicotinic acetylcholine receptors; when stimulated extra stored acetylcholine is released.

COX-2 Inhibitors

- cerebral inflammation seems to hasten deposition of amyloid
- Three agents are being assessed in trials: celecoxib, rofecoxib, and GW253035 after initial promise shown with indomethacin and diclofenac in slowing loss of reasoning power (but alas, intolerable GI side effects) by blocking this harmful inflammatory response.

Antibiotics: Doxycycline & Rifampin

- A pilot randomized control study involving the above two antibiotics showed the intriguing results of slowing down the rate of dementia after just 6 months of therapy (D.W. Molloy et al, Journal of the American Geriatric Society, Volume 52 Issue 3 Page 381-387, March 2004).
- Mechanism not likely anti-microbial; may involve secondary anti-inflammatory effects, or possibly chelating away copper, needed in processing of Beta amyloid protein.
Vascular Dementia (VaD)

Vascular dementia (VaD) differs from AD by its history of onset (often sudden), clinical features (see below), and subsequent course (may be stepwise or saw-tooth decline). The prevalence of VaD is thought to be about 10% in Canada. It frequently co-exists with AD, a situation which is called Mixed Dementia.

In VaD, there is a history of transient ischaemic attacks (TIAs) or strokes, with brief impairment of consciousness, fleeting pareses, or visual loss. The dementia may also follow a succession of acute cerebrovascular accidents or, less commonly, a single major stroke. The pattern of cognitive impairments of the dementia may be patchy, so that there may be memory loss, intellectual impairment, and focal neurological signs. Insight and judgment may be normal or affected.

Associated features are gait disturbance, emotional lability with transient depressive mood, weeping or laughter, and brief episodes of clouded consciousness or delirium, often provoked by further infarction. Personality changes may be present in some cases with apathy, disinhibition, or enhancement (and coarsening) of previous traits such as egocentricity, paranoia, or irritability. If the vascular insults occur primarily in the frontal lobes, the features of a fronto-temporal dementia may be seen.

Different criteria have been published on dementia caused by stroke. The most widely used is the NINDS-AIREN criteria [National Institute of Neurological Disorders and Stroke and Association Internationale pour la Recherche et l'Enseignement en Neurosciences; two groups working together to develop criteria that was developed at an international workshop in 1992 involving 54 neurologists and neuroscientists]

NINDS-AIREN criteria for VaD (all the following)
1. Dementia
2. Focal signs on exam + evidence of cerebrovascular* disease by CT or MRI
3. A relationship of the two above, with dementia within 3 months of a recognized stroke and or abrupt deterioration in fluctuation, or fluctuating stepwise progression of cognitive deficits.
The specificity of NINDS-AIREN criteria is high (91%) for the exclusion of AD (Gold et al., Neurology 1997;49:690-694)

**Physical Examination in VaD**
- May show increased tone (especially in the legs), exaggerated deep tendon reflexes, and babinski responses (upgoing toes).
- Gait apraxia (“magnetic” gait) may be seen.
- Other focal signs may be detected

**Treatment of Vascular Dementia (VaD)**

Once VaD begins, there is no reversibility (although **Mixed Dementias** do respond to cholinesterase inhibitors just like they do in AD). In fact, little is lost in treating the same as in AD even if you are convinced it is pure VaD (the patient has little to lose and much to gain). However, one may slow VaD/Vad+AD progression by treating underlying comorbiditis. This includes identification and treatment of:
- Hypertension
- Atrial Fibrillation
- Carotid Artery Disease
- Elevated cholesterol
- And finally, tobacco smoking cessation

**PEARL:*** Needless to say, identification of stroke risk factors and treatment of the above modifiable factors BEFORE a stroke and its sequelae occur is crucial:

**Hypertension:** Aim for a BP less than 140/90. Simple diuretics such as Hydrochlorothiazide (HCTZ) starting at 12.5 to 25 mg po OD is reasonable to begin (but watch for orthostatic hypotension). Recent guidelines offer a choice of beta blocker/ calcium channel blocker. Choice of drug will depend on comorbidity:
- HTN + Angina= Rx with Beta blocker
- HTN + Asthma=Rx with anything BUT Beta-blocker
- HTN and DM=Rx with ACE-Inhibitor
**Atrial Fibrillation:** Start on warfarin (Coumadin) if there are no contraindications, or at the very least Enteric Coated Acetylsalicylic Acid (ECASA). Warfarin is far more effective than reducing risk of stroke than ECASA, but its use may be problematic. Frequent falls are NOT a contraindication as shown in a recent meta-analysis done by Man-Son-Hing (Archives of Internal Medicine, 1999;159:677-685)

**Carotid Artery Disease:** Carotid endarterectomy is beneficial in preventing stroke in selected patients and is indicated in symptomatic patients with carotid-territory transient ischemic attacks or minor strokes who have carotid artery stenosis of 70 to 99 percent. Unfortunately, once a VaD begins, you will be unable to find a vascular surgeon to treat this. Anti-platelet agents (ECSA, Plavix) and risk factor modification are recommended in symptomatic patients with less than 50 percent stenosis or those who cannot be surgically corrected.

**Elevated cholesterol:** Confirm Total Cholesterol levels, HLD, LDL and Triglycerides by fasting blood analysis. While diet modification can help, most elderly persons would benefit from starting on a ‘statin’ to lower their cholesterol levels.

**Prognosis of VaD/Mixed VaD + AD**

- Highly variable; if does not succumb to a devastating stroke, may have similar prognosis as AD patients
Lewy Body Disease (Dementia with Lewy Bodies)

A recently defined clinical entity, Lewy Body disease is a dementia where deficits on tests of attention, and visuospatial ability are be especially prominent. It is so named because of eosinophilic inclusion bodies (called Lewy Bodies, found primarily in the basal ganglia of patients with Parkinson’s Disease) are prominent histological findings in the cortices at autopsy.

For a Probable diagnosis of Lewy Body Disease you need at least two of:
- Fluctuating cognition with pronounced variations in attention and alertness
- Recurrent visual hallucinations which are typically well-formed and detailed
- Spontaneous motor features of parkinsonism

Features supporting the diagnosis:
- Syncope or transient LOC
- Neuroleptic sensitivity (i.e. new shuffling gait, tardive dyskinesia)
- Systematized delusions
- Hallucinations in other modalities (i.e. smell, hearing, taste)
- Falls

Diagnosis of DLB is less likely in the presence of:
- Cerebrovascular disease, evident as focal neurological signs or on brain imaging
- Evidence on physical examination and investigation of any physical illness or other brain disorder sufficient to account for the clinical picture

Aetiology
- Uncertain; overlaps between AD and Parkinson's.
- Genetic studies pending

Prevalence:
- Up to 15% of all cases of dementia
- Some investigators think this is the most common form of Dementia after AD
Investigations for DLB

- There are no specific diagnostic tests for DLB.
- Patients with DLB have great difficulties in not only the clock drawing test and other simple drawing tasks.
- EEG typically reveals generalized slowing of background activity; occasional patients with rapidly progressive dementia will show periodic complexes reminiscent of CJD.

Management and Treatment for DLB:

- There is no specific therapy; general treatment limited to managing neuropsychiatric disturbances and the associated movement disorders.
- Cholinesterase inhibitors may be useful in Rx, and there is now one open label RCT suggesting that rivastigmine may be helpful [Lancet. 2000 Dec 16;356(9247):2031-6].
- Small scale studies suggest that the newer atypical neuroleptics such as risperidone, olanzapine & quietepine may be able to treat psychotic symptoms without causing excessive parkinsonism.

Prognosis

- Shorter than in patients with AD
- Felt to be on average 4-5 years from time of diagnosis
Frontotemporal Dementia (FTD)

A dementia characterized primarily by problems with behavior, insight and judgment rather than memory loss; it is the “flip side” of AD in several ways:

- behavioral troubles >> memory; so-called “unpleasantly demented”
- anatomical deficits are 1º frontal-temporal versus bilateral parietal-temporal changes as seen in AD

Prevalence of FTD
- estimates vary from between 10-25%; most agree that it is the most common dementia after AD in the presenium (under 65s)

I. Core diagnostic features of FTD
- Insidious onset and gradual progression
- Early decline in social interpersonal conduct
- Early impairment in regulation of personal conduct
- Early emotional blunting
- Early loss of insight

II. Supportive Diagnostic Features

A. Behavioural disorder
- Decline in personal hygiene and grooming
- Mental rigidity and inflexibility
- Distractibility and impersistence
- Utilization behaviour Hyperorality & dietary changes
- Preservation and stereotyped behaviour

B. Speech and language deficits
- Altered Speech Output (Aspontaneity & Economy of Speech)
- Stereotypy of speech
- Echolalia
- Perservation
- Mutism
II. C. Physical signs

- Primitive Reflexes; at least one of grasp, snout and sucking
- Incontinence
- Akinesia, rigidity and tremor (rarely)
- Low and labile blood pressure

II. D. Investigations

- Neuropsychology
  - Significant impairment on frontal lobe tests in the absence of severe amnesia, aphasia or percutuospatial disorder
- EEG:
  - Normal despite clinically evident dementia
- Brain Imaging (structural or functional)
  - Predominant frontal and/or temporal abnormality

Relative diagnostic exclusion features of FTD

- Typical history of chronic alcoholism/
  - Sustained hypertension/
- History of vascular disease

Detection of FTD

- Frontal lobe dysfunction often unrecognized in its early phases
- May have normal neurologic testing when routine methods employed (ie. MMSE.)
- Unfortunately, there is no good test to reliably identify a dysexecutive function (several test batteries in use; FAB, FBI, ACE)

Common Misdiagnoses

- “Atypical” AD/ Schizophrenia/ Depression / Bipolar disorder/ OCD / Hypochondriasis/ Sociopathy

2º causes of FTD

- Head Injury / Post lobectomy or leukotomy/ Anoxic Brain Injury/ Huntington’s Disease/ ? ETOH
Closely Related FT Dementias
1) Primary Progressive Aphasia
   • non-fluent agrammatic speech
2) Semantic Dementia
   • fluent speech, lost semantic meaning of objects/words, repetition is good
   • memory relatively good for personal, autobiographical events

Genetics of FTD
• 45% of pts have a family member affected
• up to 18% of these have an abnormality on the short arm of chromosome 17 localized near gene for the microtubule associated protein, tau

Treatment of FTD

A: Medical Therapy of FTD
• theoretically there is no cholinergic deficit and would therefore will not benefit from cholinesterase inhibitors; however, as there is no good way to distinguish from AD, consider doing a trial of one of the three available agents.
• As there is a serotonergic deficit it may be worth trying an SSRI based on side effect profile (i.e. use fluoxatine if want to stimulate, if over stimulating, use fluvoxamine or paroxetine to sedate)

B: Non-Medical Therapy
• Behavioural modification
• Caregiver education, respite care and emotional support; early identification of caregiver depression

PEARL: Spatial orientation and praxis preserved (intact abilities to negotiate the environment). Can do well on SMMSE (often 27/30) despite being unable to perform certain tasks. Can alternatively do surprisingly well on driving tests despite a correspondingly low SMMSE (mostly on problems not understanding the questions).
Rare Dementias

Normal Pressure Hydrocephalus (NPH)
- Dementia 2° to a type of hydrocephalus which can occurs in older patients.
- NPH is an accumulation of cerebrospinal fluid (CSF), causing the ventricles of the brain to enlarge, yet do not cause consistently increased intracranial pressure, as is the case with obstructive hydrocephalus.
- The abnormal accumulation of CSF, causing enlarged ventricles, is thought to stretch the nerve tissue of the brain causing a triad of symptoms.
  1. Dementia
  2. Gait Disturbance
  3. Urinary Incontinence

Treatment of NPH:
- Often disappointing; short history & a precipitating event (e.g. meningitis or subarachnoid hemorrhage may predict better outcome to Rx).
- Ventriculoperitoneal shunting (shunt inserted to divert the cerebral spinal fluid away from the brain)
- About 1/3 improve [but only in terms of their gait], 1/3 no change, and 1/3 worse (27% get complications, 7% morbidity)

Korsakoff’s Syndrome: (aka Korsakoff’s Psychosis)
- Irreversible amnestic disorder caused by a deficiency of thiamine (Vitamin B1).
- The anterograde amnesia is commonly associated with vivid confabulation (i.e. the patient is unaware of the memory problem, and simply makes up false memories to fill the gap).
- Most commonly seen in alcoholism (although it can be found in severely malnourished or dialysis patients).
- It is related to Wernicke's syndrome, which often precedes Korsakoff's syndrome (and the two disorders are thought to be part of a spectrum of a single entity) but does not respond to thiamine therapy.
Huntington's Dementia

- Huntington’s Disease is a fatal autosomal dominant hereditary disorder characterized clinical triad of emotional, cognitive and motor disturbances:
  - Motor symptoms include chorea (dance-like involuntary movements), clumsiness, slurred speech;
  - Emotional symptoms include depression, irritability and apathy.
  - Cognitive losses include loss of intellectual speed, attention and short-term memory; personality changes are also common
- As the disease progresses, movements become severe and uncontrollable; cognitive impairments deteriorate to a dementia of the FTD variety.

Parkinson’s Dementia

- About 30% of patients with Parkinson’s Disease (PD) will develop dementia (some studies say up to 60%)
- Problems include deficits in visual/spatial ability, visual hallucinations, and memory
- May overlap with symptoms seen in Lewy Body Dementia, and because PD and AD are both common, they may occur together by chance

Dementia associated with acquired immunodeficiency syndrome (AIDS) [aka AIDS Dementia Complex].

- Adults over age 50 now account for at least ten percent of reported AIDS cases, and it is currently estimated that 8 to 16% of people with AIDS will develop dementia as a direct effect of the AIDS virus on the brain
- The intellectual and cognitive decline in patients with AIDS dementia is remarkably similar to those in Alzheimer’s, and autopsies show plaques similar to those found in Alzheimer’s
Creutzfeldt-Jakob Disease (CJD)

- A very rare and fatal neurological disorder caused by a prion (a proteinaceous infectious particle) that was first described in the 1920s.
- Its pathophysiology is not completely understood. Although it can occur in younger persons, peak age of onset of classic CJD is 60 to 70 years.
- Worldwide incidence ~0.5-1.5 cases per million per year, and most cases are sporadic. CJD has been considered infectious since the mid-1960s, but its transmissibility through the transfusion of blood or blood products is controversial.
- One strain, new variant Creutzfeldt-Jakob Disease (nvCJD), is linked to bovine spongiform encephalopathy (i.e. Mad Cow Disease) in the UK, but there have been no documented cases of human transmission of BSE in Canada.

Classical Symptoms of CJD

- a rapidly progressive presenile dementia with progressive myoclonus and progressive motor dysfunction.
- Early symptoms can include cognitive impairment, behavior changes, visual disturbances, and lack of coordination.
- Pyramidal and EPS symptoms, along with akinetic mutism develop late.

Diagnosis of CJD

- CSF fluid analysis for the presence of 14-3-3 protein
- Brain biopsies looking for spongiform changes (but don’t do it yourself!)
- EEG shows a typical pattern of periodic sharp-wave complexes
- an autopsy is the only definitive way of diagnosing CJD. In most cases of human prion diseases the histological features are distinctive; spongiform change, neuronal loss, astrocytosis and amyloid plaque formation.

Treatment of CJD

- There is no treatment, and the duration of CJD from the onset of symptoms to death is usually less than a year.
Behavioural Problems in Dementia

Up to 90% of demented patients will develop significant behavioral problems at some point in the course of their illness

- Agitation (definition=a range of purposeless verbal, motor behaviors that put the patient or others at risk of harm) is the most commonest problem, and is seen in up to 75% of patients with dementia.
- Wandering in up to 60%
- Depression in up to 50%
- Repeated stories and statements in ~32%
- Psychosis in up to 30%
- Hoarding & Rummaging in up to 30%
- Screaming in up to 25%
- Aggression and violence in up to 20%
- Hypersexuality in up to 10%

These problems result from the progressive inability of the demented patient to remember, reason or solve problems. Because they are associated with frontal involvement, they occur early in FTD and DLB, and in the later stages of AD. They arise from the disease process and are rarely enacted to be manipulative.

**PEARL:** Think of dementia as a form of unwinding of learned abilities, or retrogenesis with what was most recently learned lost first. It’s like Jean Piaget’s stages of childhood development running in reverse, with the mind of a demented person becoming more childlike and primitive in its logic and abilities as the disease progresses. Remember that while a demented person’s intellectual processes are deteriorating, their emotions and feelings may be preserved.

Behavior problems caused by dementia are often disruptive, and can lead to social isolation, caregiver burnout, accelerate the need for LTC placement, iatrogenesis, and the increased use of restraints (and their own problems)
Assessment of Behavioral Problems

Establish or revisit the medical diagnosis

- Accurate diagnosis of type of dementia?
- Is it really a dementia (consider life long personality disorder, chronic psychiatric problems, etc.)

Assess and Reverse Aggravating Factors

- Pain or discomfort that cannot be verbalized (fatigue, distended bladder, constipation, compression ulcers, dental abscess, PND at night)?
- Anxiety or Depression?
- Medications contributing?
- Physical limitations, or functional disabilities?
- Medical illness, delirium (from infection, drugs, etc.)?
- Hearing impairment or vision loss?
- Boredom, isolation, loneliness
- Environmental sources (staff interaction, under stimulation, over stimulation)

Document the specific behaviors

- Use a behavior chart to document, using **ABC analysis**
- **A is for Antecedents** (i.e. what happened just before)
  - Triggers for behavior (i.e. physical treatments, pain, bathing, mealtimes, company, loneliness, noise level, vision or hearing problems)?
- **B is for Behavior**
  - Be specific! The words “Aggression” or “Agitation” are not helpful. Were they screaming, biting, or merely calling out because they were lonely and wanted attention? Did they really hit someone, or were they simply waving their hands about and someone got in the way?
  - Note the frequency, timing and location of the behavior; related to some intervention (e.g. giving care, bathing, meals) or spontaneous?
  - Note which persons or staff were involved (or just one person?)
• C is for Consequences
  o Events that maintain the behavior?
  o How did the staff handle it?
  o What has worked before? What has failed?

Ask yourself, is intervention really necessary?
• Is this a matter of patient safety versus staff convenience?
• Is the behavior distressing to the patient (i.e. hallucinations/delusions?)
• Is the behavior interfering with BADLs or socialization?

Attempt individualized Non-pharmacolgical Approaches

Behaviors are rarely random, unpredictable or meaningless events. Behavior itself is a way of communicating feelings and needs that cannot be adequately verbalized by a patient with a dementing illness. Management of problem behaviors can be shifted from trying to change the patient to trying to change the triggering or exacerbating factors. Three problems and their non-drug approaches follow below:

<table>
<thead>
<tr>
<th>Agitation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Possible Trigger</td>
</tr>
<tr>
<td>Discomfort, pain</td>
</tr>
<tr>
<td>Fatigue</td>
</tr>
<tr>
<td>Medication ADRs</td>
</tr>
<tr>
<td>Overstimulation (noise, overhead paging, beepers, people, TV, activities)</td>
</tr>
</tbody>
</table>
### Wandering

<table>
<thead>
<tr>
<th>Possible Trigger</th>
<th>Management Strategy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restless, bored, no stimuli.</td>
<td>Provide meaningful activity (folding towels, sorting buttons), provide safe circular path, fenced in yards.</td>
</tr>
<tr>
<td>Escaping from stress (noise, clutter, crowding, activity—&gt;“Run away!”)</td>
<td>Reduce excessive stimulation.</td>
</tr>
<tr>
<td>Lost—looking for something or someone familiar, “There’s no place like home, Auntie Em!”</td>
<td>Provide familiar objects, pictures of toilet on bathroom door, signs, reassure.</td>
</tr>
<tr>
<td>Eloping stimuli; exit signs, people leaving “Hey, wait for me!”</td>
<td>Camouflage exits, use stop signs or barrier tape, code access doors.</td>
</tr>
</tbody>
</table>

### Inappropriate or Impulsive Sexual Behavior

<table>
<thead>
<tr>
<th>Possible Trigger</th>
<th>Management Strategy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Decreased judgment and social awareness</td>
<td>No not overreact or confront; respond calmly and firmly; distract or redirect.</td>
</tr>
<tr>
<td>Misinterpreting caregivers interactions (i.e. bathing, toileting)</td>
<td>Avoid mixed sexual messages, distract while performing personal care, bathing; be consistent.</td>
</tr>
<tr>
<td>Uncomfortable clothing; too tight or to hot</td>
<td>Check room temperature. More appropriate clothing.</td>
</tr>
<tr>
<td>Need for attention, affection, intimacy, hugs.</td>
<td>Model appropriate touch, offer soothing objects (stuffed animal), provide back massage, provide privacy</td>
</tr>
</tbody>
</table>
General Psychotropic Approaches to the Management of Behavioral Problems in Dementia.

Non-drug therapies work best, and should be tried first. Drugs are a last resort, and only if the above strategies and analysis fails.

- Scheduled, not prn, medications
- Use monotherapy if possible
- Consider and avoid drug-drug interactions
- Plan duration of trial (not open ended)
- “Start Low, go slow” (ie. ¼ normal starting dose), but avoid under dosing
- Chose target symptoms, and reasonable end points.
- Document outcomes
- Monitor for ADRs, especially those that impact on cognitive functioning (i.e. sedation, anticholinergic effects), movement (sedation, EPS, orthostatic hypotension, tardive dyskinesias or dystonia), and sexuality.

PEARL Remember that drugs work only if there is:

- Psychosis and/or Aggression
- Depression
- Anxiety
- Sleep disturbance

PEARL: Psychotropic drugs do not help: Repetition or, Wandering
What Psychotropic Medication to Use?

Choice may be influenced by the urgency of the situation:

- If requires rapid control of grossly disturbed or unsafe situations, use neuroleptic and benzodiazepine combination (ie. IM haloperidol + lorazapam; start with 1 mg of each for a robust person, and 0.5 mg of each for someone frail)
- If more time available, look at what psycho-behavioral “metaphors” (not diagnoses, but what the behavior “kind of looks like”) that the patient is exhibiting (chart below)
- More and more cholinesterase inhibitors are found to be useful but not specific Rx for behavioral problems.

<table>
<thead>
<tr>
<th>Behavioral “metaphor”</th>
<th>Agent to try</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Depressive Features</strong>&lt;br&gt;(i.e. an agitated patient who is withdrawn, apathetic, irritable, negativistic, ruminating and/or dysphoric)</td>
<td>Antidepressant (i.e. citalopram, sertraline, venlafexine) Anticonvulsant (i.e. carbamazepine, divalproex)</td>
</tr>
<tr>
<td><strong>Manic Features?</strong>&lt;br&gt;(i.e. an agitated patient who is hyperactive with pressured speech, irritable mood, and sexual preoccupation)</td>
<td>Anticonvulsants (i.e. divalproex, carbamazepine) Beta-blockers (i.e. propanolol)</td>
</tr>
<tr>
<td><strong>Anxious/Obsessive Features</strong>&lt;br&gt;(i.e. an agitated patient relentlessly checking her purse for pocket money)</td>
<td>SSRI Antidepressants (i.e. citalopram, sertraline, paroxetine) Anticonvulsants (i.e. divalproex) Benzodiazepines (i.e. short term oxazepam)</td>
</tr>
<tr>
<td><strong>Psychotic Features</strong>&lt;br&gt;(i.e. a patient screaming and striking out because she thinks that aliens are experimenting on her)</td>
<td>Neuroleptics (i.e. Risperidone, Olanzapine, Quetiapine)</td>
</tr>
<tr>
<td><strong>Sexually Aggressive Features</strong></td>
<td>Neuroleptics (i.e. low dose haloperidol)</td>
</tr>
</tbody>
</table>
Delirium

Delirium, or acute confusion, is a common problem. Up to 60% of older persons hospitalized for surgery will become delirious. 35% to 65% of elderly patients with hip fractures will become delirious after surgery.

**Why is delirium important?**

Mortality is 20%/year for those >70 years (independent of all other co-morbidities); Delirium is therefore a MEDICAL EMERGENCY.

**DSM IV definition:**

Decreased attention & disorganized thinking, with at least 2 of:

- reduced LOC
- perceptual disturbances
- altered sleep-wake cycle
- disorientation
- memory impairment
- altered psychomotor activity
- with acute onset and fluctuation over time (hours).
Clinical diagnose of Delirium

Method 1. Use the CAM (Confusion Assessment Method); best, see appendix.

Is there:

1. acute onset and fluctuating course?
2. lack of attention?
3. disorganized thinking?
4. altered Level of Consciousness?

If 1 and 2 are present, and either 3 or 4, the patient is probably delirious.

Method 2. Use the Digit Span Test

Ask the patient to repeat a sequence of numbers that you present to them at a rate of one per second in your normal voice. Start off with a sequence of 3, then 4, then 5, etc. up to a maximum if 7 random number sequences. (i.e. 1-8-5, 6-4-3-7, 2-0-7-1-5, etc.) Do not use duplicate numbers or group them into obvious patterns (i.e. phone #).

Persons who are not delirious should be able to repeat 7 (+/- 2) numbers from the sequence you initially dictate to them.

Risk Factors for Delirium

- Pre-existing dementia (such brains are more vulnerable and less able to withstand any form of insult)
- Poor vision (ARMD, cataracts, no glasses)
- Poor hearing (absent hearing aid, wax blocked ears, etc.)
- Dehydration

If all 5 are present, the patient will likely become delirious if they become sick enough to need to become hospitalized! And if they are already in hospital, additional risks for delirium include:

- Use of restraints
- Foley catheter in situ
- >3 new medications
- NPO status
PEARL: Mnemonic of risk factors commonly associated with DELIRIUM:
- Dementia,
- Electrolytes,
- Lungs and other organs,
- Infection,
- Rx (medications),
- Injury (pain and stress),
- Unfamiliar environment, and
- Metabolic problems.

PEARL: Another mnemonic for causes of delirium is DEMENTIA
- Drugs
- Endocrine
- Metabolic, organ failure, etc.
- Epilepsy (post seizure)
- Neoplasm
- Trauma (head, surgery, etc.)
- Infection
- Apoplexy or other vascular event.

Causes of Delirium:

Anything that affects the brain and its normal functioning can provoke a delirium. A brief listing includes:

- Drugs (or drug withdrawal; especially anticholinergics, sedatives, opioids)
- Metabolic disturbances (hypoxia, hyponatremia, hypo & hypercalcemia, hypovolemia, Hepatic or renal failure, Cushing's Syndrome, etc.).
- Infection (obvious or occult; may include UTI, or subacute bacterial endocardidits (SBE)).
- CHF, MI, tachy- or brady-arrhythmias
- ETOH (toxicity, withdrawal, or thiamin deficiency)
- CNS (strokes, trauma, etc.)
- Pain (fecal impaction, urinary retention, recent surgery).
Management of Delirium (see also appendix):

- Quiet environment; pagers on vibrate, dim lights at night, PT & OT to attempt to mobilize the patient as early as possible
- Regular reorientation
- Improve vision & hearing if possible
- IV boluses rather than continuous IV fluids if at all possible
- One-on-one care
- Px including rectal to rule out fecal impaction, and a post-void residual to rule out urinary retention
- Lab screen: CBC, lytes, BUN\Cr\glucose, ECG, CXR, pulse oximetry or cap gases, urinalysis (also consider doing “extras” including Ca++, Mg++, TSH, LFTs, albumin & B12
- Stop medications that may be maintaining the delirium (i.e. opiods, sedatives, anticholinergics, H2 blockers)
- Keep well hydrated; maintain fluids and nutrition.

Management of Uncontrolled Agitation with Delirium

If agitated, violent and combative, the immediate goal of treatment of treatment is to reduce symptoms to a level where the patient is not a physical danger to himself or others. The cessation of hallucinations and/or delusions (assumed to be the basis of the dangerous agitated behavior), is the ultimate goal. Current management of agitated patients involves the use of neuroleptics and benzodiazepines either alone or in combination.

- Low dose po haloperidol (Haldol) 0.25 mg po bid to 1 mg bid; may also give IM haloperidol 0.5 mg or IM olanzapine (Zyprexa) 2.5-5 mg QHS which should work within 30 minutes when combined with an short acting benzodiazepine (1-2 mg lorazepam, either po or IM, can be given q 1 hour in addition to the neuroleptics above until sedation is
- Low dose Risperidone (Risperidal) 0.25 BID works well if not needed acutely; it is less anticholinergic and has less hypotensive ADRs than seen in older neuroleptics.
- Sublingual Olanzapine (Zyprexa Zydis) in a gelatin wafer that instantly dissolves when placed under the tongue is now available in many Ontario hospital formularies (one new possible ADR to watch out for is hyperglycemia.
Depression

Depression is the most common psychiatric illness in the elderly. Although common, it is NOT a natural part of aging.

The prevalence in community dwelling elders range from 8-15%; it rises to as much as 30% of those in long term care facilities. Depression and suicide are common in the elderly (especially older males; those over 75, have the same risk of suicide as 20-24 year old depressed males).

Depression is NOT present in ALL older adults, but is under recognized & under treated. Depression is common in:

<table>
<thead>
<tr>
<th>Long Term Care</th>
<th>Dementia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bereavement</td>
<td>Disabilities</td>
</tr>
<tr>
<td>Stroke</td>
<td>Poor education</td>
</tr>
<tr>
<td>Parkinson’s Disease</td>
<td>Poverty</td>
</tr>
<tr>
<td>Any Chronic Illness (i.e. DM, COPD,)</td>
<td>Social Isolation.</td>
</tr>
<tr>
<td>Alcoholism</td>
<td>Chronic Pain (i.e. Cancer (Ca), RA, OA)</td>
</tr>
</tbody>
</table>

To Dx a **Major Depression**, you need at least 5 of the below for 2+ weeks

<table>
<thead>
<tr>
<th>Anhedonia (absence of usual pleasure)</th>
<th>Psychomotor changes (slow…)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depressed Mood</td>
<td>Excessive guilt</td>
</tr>
<tr>
<td>Sleep Disturbance</td>
<td>Nihilistic thoughts</td>
</tr>
<tr>
<td>Unexplained wt loss or gain</td>
<td>Indecision</td>
</tr>
<tr>
<td>Loss of appetite</td>
<td>Concentration and memory problems</td>
</tr>
<tr>
<td>Thoughts of Suicide and hopelessness</td>
<td></td>
</tr>
</tbody>
</table>
PEARL: Mnemonic “Sig. E Caps” (Sig.=from the old Latin way to prescribe, E=“Energy”, Caps=“Capsules”) as a check list for Depression:

- S uicidal Ideation
- I nterest, lack of
- G uilt
- E nergy, none
- C oncentration, poor
- A ppetite(s), altered
- P sychomotor changes (slowed or revved up)
- S uicide

Bereavement or Grief reaction is commonly misdiagnosed as depression. A normal grief reaction after the death of a spouse or a loved one lasts about 2 months in time, with the mourning process being complete in under 2 years. Feelings of sadness and preoccupation with the deceased are not helped by anti-depressant medications during this time of mourning.

However, grief can become pathological when it persists past 2 months, when the grieving person is unable to function, and constantly expresses nihilistic thoughts of death. Suicidal thoughts are also felt and may be verbalized, and these are never normal, and merges into a diagnosis of true Major Depression.
Screening for Depression

- Ask, "What is wrong?" Or, “Do you often feel downhearted or sad?”
- 15 or 30 item GDS (see below)
- ROS shows up with many somatic complaints in many domains
- Ask about suicidal or pessimistic thoughts

The **Geriatric Depression Scale** (GDS), is a screening tool for depression, and does not itself diagnose depression. There are various forms about, including a 30 question, a 15 question short form (see Appendix) a four question, and a single question screen (i.e. If you have time to ask only 1 question to screen, ask “Do you feel that your life is empty?”

For short form (15 questions) GDS; >5 points are suggestive for depression, and scores >10 points are very suggestive for depression [See the appendix for all the questions].

For the 30 question GDS, >13 points are suggestive, and >15 very suggestive.

**PEARL:** The GDS has not been validated in patients with Cognitive Impairments, and needs to be interpreted with caution.

**PEARL:** If positive on screen, ask about such depressive symptoms as listed above. Also ask if they are consuming ETOH, since ~4% of older adults abuse it.

**PEARL:** Also, if positive for depression on both a screen and subsequent interview, discuss treating this without delay.
Treatment of Depression in the Elderly

Non-Pharmacologic

Psychotherapy, in particular behavioral and cognitive based therapies appear to be helpful.

Short-term psychodynamic therapy (such as cognitive behavioral therapy or interpersonal therapy) has been reported to be effective in the treatment of depression in elderly.

Pharmacological Agents

Selective serotonin reuptake inhibitors (SSRI’s) are the friendliest antidepressants to use in older adults, because of their safety in overdose, and because they do not interact with as many other medications as older agents.

They are also not anticholinergic as the older TCAs, and have fewer adverse cardiovascular effects and do not cause orthostatic hypotension. They are also useful in patients who have comorbid dementia, and this has been demonstrated in a number of studies.

Common ADRs with SSRIs include nausea, and other GI symptoms, headaches, and dizziness.

Some caution needs to be followed in using SSRIs, as there is the risk of SIADH (hyponatremia caused by an as yet unknown mechanism). They can also cause sexual dysfunction, which your patients may not report (and you may not remember to ask!).

Good all purpose SSRIs for older adults currently available in Canada are Zoloft (Sertraline ) and Celexa (Citalopram):
**Sertraline:** The initial starting dose is 25-50 mg po OD, given in the morning. Target dose by week three is 50-100 mg/OD. By week five, the target dose is 50-150. Full effective dose in those over the age of 65 after week 7 is 50-200 mg/OD [with the upper dose range reserved for the robust older adult; for the frail elderly the range is 100-150 mg po OD] Commonly reported side effects include tremor, restlessness, and gastrointestinal complaints, but no more than placebo in RCTs.

**Citalopram:** The full effective dose is 20 mg po OD. You can start off at 5-10 mg a day for 3-7 days, watching for tolerance and adverse side effects. If tolerated, continue doubling the dose every week until the maximum dose of 20 mg/day is achieved. It will take at least 6 weeks before any change of depressive symptoms will be achieved. Citalopram appears to inhibit P450 minimally, if at all.

Effects of the SSRI to improve depression may begin by week 4-6, should be seen by weeks 7-9. If no improvement by this point (or an adverse side effect), consider switching to another SSRI or a Serotonin and Noradrenaline Reuptake Inhibitor (SNRI) after first allowing the original medication to wash out. If improvement, continue for at least 12-24 months after the remission of symptoms in older adults. If there is a history of previous recurrent depression, consider treatment indefinitely.

**Non SSRI Anti-Depressants**

Other agents proven to be safe in older adults include **Effexor XR** (Venlafaxine) which is a SNRI (think of it as a mixture of a TCA and an SSRI), and **Bupropion SR**. A relatively new agent now available is **Mirtazapine** (Remeron), which is classed as an NASSA (noradrenaline and selective serotonin antidepressants).

Adjuvant agents include short term use of short acting benzodiazepines (i.e. lorazepam, oxazepam) for treatment of anxiety until the antidepressant kicks in, and mood stabilizers such as Lithium, valproic acid and carbamazepine may also be useful. Augmentation with triiodothyroxine can be considered.
Electroconvulsive Therapy (ECT)

- ECT uses electricity to cause a grand mal seizure to improve the symptoms of major depression; the mechanism of the antidepressant effect of ECT is unknown, but it is thought that the whole brain seizure provokes changes in endogenous neurotransmitter systems.
- It is a safe, proven, yet underutilized method of treating major depression, especially in older adults.
- It is usually given 2-3 times a week for a period of several weeks, then maintenance ECT is used.
- Quicker onset than conventional antidepressants, and fewer side effects.
- It is often used in medication resistant depression, in patients with psychotic features with their depression, or in patients who have previously responded to ECT therapy, or in urgent situations where life is in danger.
- Improvement of symptoms seen in ~80% of those treated with ECT.
- Besides treating major depression, ECT is used in severe bipolar depression, schizoaffective disorder and chronic psychotic disorders such as schizophrenia and bipolar mania.
- There are no absolute contraindications to ECT, only situations of increased complications; ECT in patients with brain masses and recent stroke (less than three months) is a concern, as is ECT in patients who have had a recent MI (less than three months) because of the risk of further cardiac problems with the combined actions of the anesthetic used and the increased demands on the heart experienced during the procedure.
When to refer Depression to a Geriatric Psychiatrist?

• The patient is suicidal or homicidal, and/or refusing to eat or drink
• Uncertainty of the diagnosis or when the differential diagnosis of depression and neurologic disorders overlap
• Treatment resistive depression that has failed to respond to one or two adequate trials (8 to 12 weeks at a therapeutic dose) of antidepressants and/or gets worse during treatment
• Antidepressant ADRs or drug-drug interactions that limit effective therapy.
• When the patient requires maintenance of complicated treatment
• When you know or suspect the patient has Bipolar Disorder (either early or late onset)
• When the patient requires evaluation for ECT
Orthostatic Hypotension (OH)

- Also known as postural hypotension, orthostatic hypotension (OH) is a major and often hidden cause of falls in the elderly!
- Not a specific disease as much as it is a syndrome of abnormal postural blood pressure response due to one or more underlying conditions that can affect the normal autonomic reflex arc.

**Definition:** An excessive drop in Blood Pressure (BP) seen when changing from a lying/sitting position to assume a standing and upright posture;

Typically the BP drop is at least 20 mmHg systolic / 10 mmHg diastolic

**Warning “Red Flags” for the possibility of OH**

- OH symptoms (dizziness, faints and near falls/falls) that occurs early each morning
- Symptoms of OH that occur on consecutive days

**Aetiology of Orthostatic Hypotension (in order of frequency)**
• **Hypovolemia:** diarrhea, hemorrhage, salt losing nephropathy, ETOH, adrenal insufficiency.

• **Drugs:** Antihypertensives, Diuretics, Vasodilators (nitrates, hydralazine), Alpha- and beta-blocking agents, barbiturates, opiates, Tricyclic antidepressants, and ETOH.

• **CNS problems:** Parkinson’s disease, Parkinson’s Plus syndromes (multisystem atrophy, Shy-Drager), Stroke.

• **Autonomic problems associated with Diabetes Mellitus (DM)**

• **Deconditioning** and prolonged bed rest

• **Post-prandial** (“face down in the corn flakes!”)

• **2° systemic arterial hypertension**

• **Idiopathic= Bradbury-Egglestone syndrome** (this can be distinguished by measuring supine norepinephrine levels, which are low)

**Non-drug Tx of OH:**
- Elevate head of bed at night
- Instruct the patient to first dorsiflex and plantar flex feet before arising slowly out of bed
- Increase salt intake (ie. canned soups, vegetables)
- Increase oral fluid intake (or even IV if necessary)
- 40 mmHg Jobst compression stockings (hard to put on, use only if they can get them on themselves otherwise they will not be used once they are home alone!)
- Caffeine helpful if taken between meals!

**Drug Tx of OH:**
- Florinef (fludrocortisone); a volume expander
- Midodrine (alpha vasoconstrictor activity on resistance and capacitance blood vessels)
- Indomethacin (but only with gastric protection; not a good idea for the oldest old)
- Erythropoeitin (useful only if low RBC mass)
Osteoporosis

**Definition:** progressive low bone mass & micro-architectural deterioration that leads to skeletal weakness and an increased risk of bone fracture; specifically it is a Bone Mineral Density (BMD) T-score of 2.5 or less (which is the equivalent of a bone density lower than 648mg/cm²).

Bone is a living organ, tissue mostly made up of collagen that has been mineralized with calcium phosphate, which provides strength and hardness to the skeleton. Bone remodeling occurs throughout life, and osteoporosis results from an imbalance between osteoclast resorption (“chews up bone”) and osteoblast remodeling (“builds up bone”) activity. Normal bone has a high bone density; once it become demineralized because of the excess action of osteoclasts, it is first becomes osteopenic (“poverty of bone), and once it is further demineralized it becomes weak and is at risk of fractures, a term called osteoporosis.

Osteoporosis may be **primary** (a combinational of post-menopasual loss of estrogen in females, and/or related to aging losses of osteoblasts seen in both men and women) or **secondary** to some other disorder (such as osteomalacia, hyperparathyroidism, hyperthyroidism, glucocorticoid excess, renal failure, liver disease, etc.).

**PEARL: Low bone mass is the single best predictor of fracture risk**

Normal **cortical** bone (Latin cortex=bark, and in medical use it means the outer layer of an organ) is the dense and compact outer part of bone that imparts strength to the skeleton, especially the long bones, and has a slower turn over; although osteoporosis can thin cortical bone, it is less affected by post-menopausal osteoporosis than trabecular bone. Normal **trabecular** bone (Latin, trabeculae= “a little beam”) has a spongy, honey-comb appearance. It has a high turn over, and is mostly present in the spine, distal radius. Because of its higher turn over, the imbalance of osteoclast activity (especially in the absence of estrogen) leads it to become progressively demineralized and osteoporotic. As these “little beams” start to melt, they can no longer impart their normal internal structural support to bone, making them weak.
Grim statistics for hip fractures:
- 40% lifetime risk if female, 13% risk for men
- 25% of persons will die post hip fracture, a similar % will transfer to LTC

Equally grim statistics for vertebral fractures:
- Prevalence ~12% for both men and women in one European study [J Bone Miner Res 1996 Jul;11(7):1010-8]
- Mortality doubles with increasing numbers of vertebral fractures (from 0 to 5), and is related to the risk of subsequent cancer & pulmonary death
- Survival rate for a clinically significant fracture the same as for hip fractures
- Chronic functional impairments also similar for hip and vertebral fractures!

PEARL: most vertebral fractures occur at +/- T12 level. If you get a compression fracture far from this location, suspect bony metastases)
Risk factors for osteoporosis

- Female gender
- Advanced age
- Early menopause
- Amenorrhea
- Women not on HRT 5 years after menopause
- Poly Cystic Ovarian Disease (PCOD)
- Hypogonadism (in men)
- Hypothyroidism and hyperparathyroidism
- Maternal history of fractures
- Sedentary lifestyle
- Poor "get up and go" test
- Any previous fracture
- Prolonged corticosteroid therapy
- post chemo patients (especially breast cancer or leukemia/lymphoma)
- Low body mass

When to do Bone Mineral Density (BMD):

- See above!
- If you suspect osteoporosis (patient kyphotic, or has shrunk over the years
- Falls or at risk of falling.

The purpose of doing a BMD is to

- Quantify bone mass
- Assess fracture risk
- Follow effects of intervention

PEARL: While a BMD can determine the quantity of bone mass present, it cannot tell the quality of the bone.
How to interpret a BMD?

- The “T score” is exactly the same as the Standard Deviation
- Normal is in bell curve of +1 to –1 SD;
  - below –1 is Osteopenia,
  - below –2.5 is Osteoporosis.
- If osteopenia, discuss lifestyle modifications (exercise, increasing dairy products); start Calcium/Vitamin D; monitor BMD once every two years.
- If Osteoporosis, start definitive treatment (see following section), and repeat BMD yearly for first two years.

Other lab tests:

- CBC
- Calcium & albumin (only the ionized calcium is biologically active; ignoring pH effects, you can roughly calculate ionized calcium by using:  
  Corrected Calcium = for each fall (or rise) of albumin by 4 g/L add (or subtract) 0.1 mmol/L to the plasma calcium.
- TSH level
- Alkaline Phosphatase
- Serum Phosphorous and Magnesium
- Creatinine
- Consider serum protein electrophoresis if you suspect multiple myeloma.
- Consider serum bioavailable testosterone level in a male with a fracture

The purpose of these tests is to rule out other 2° causes of bone demineralization

<table>
<thead>
<tr>
<th></th>
<th>Se Ca⁺⁺</th>
<th>Alk Phos</th>
<th>Se P04⁻⁻</th>
<th>Other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Osteoporosis</td>
<td>Normal (N)</td>
<td>N or Increased</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>Osteomalacia</td>
<td>Decreased</td>
<td>Increased</td>
<td>Decreased</td>
<td>25-OHD decreased</td>
</tr>
<tr>
<td>Primary PTH</td>
<td>Increased</td>
<td>N or Increased</td>
<td>Decreased</td>
<td>PTH increased</td>
</tr>
<tr>
<td>Multiple Myeloma</td>
<td>Increased</td>
<td>N or Increased</td>
<td>--</td>
<td>Protein electrophoresis</td>
</tr>
<tr>
<td>Renal Osteodystrophy</td>
<td>Decreased</td>
<td>Increased</td>
<td>Increased</td>
<td>Creatinine Increased</td>
</tr>
</tbody>
</table>
Treatment of Osteoporosis:

Pearl: Purpose of Rx in Osteoporosis is to prevent fractures, maintain function and reduce pain (if any).

A: Non drug treatment
- Diet (to improve calcium rich foods, such as dairy products and green leafy vegetables)
- Exercise (you need not only calcium, but exercise to “pound it” into the bone)
- Lifestyle modifications to minimize risk of falling (proper footwear, improved lighting, activities that improve balance and strength like Tai Chi.

B: Drug Treatment for Osteoporosis
- Calcium supplementation: 1000 mg of elemental calcium
  = Calcium Carbonate 1250 mg PO BID
- Vitamin D: 800 iu PO OD (aids the absorption of Calcium & Magnesium)
- Estrogen: 0.625 mg po OD (or 50-100 microgram transdermal patch/day)
  - important for prevention & treatment in post-menopausal woman; but cannot use if h’ of Breast CA, unexplained vaginal bleeding, active thrombosis or liver disease!
C: Specific Therapies for Osteoporosis:

**Raloxifene (Evista)** [Ettinger study, 50% reduction vertebral # after 1 year]
- Also beneficial is the reduction in breast cancer
- Good for 50-75 year old females; but can increase numbers of Deep Vein Thromboses (DVTs)

**Calcitonin** [PROOF study]
- Reduces incidence of vertebral #, & 40% of persons obtain analgesia

**Bisphosphonates (three available)**

**Etidronate (Didronel)**
- Only one on ODB at this point in time, its cheap, but takes ~4 years to see improvement (therefore, if life expectancy not this much, don’t start!)
- Often combined in kit-form with Calcium (Didrocal), where it must be taken cyclically 2 weeks on, 3 months off.
- Need to show decline in BMD or new fracture while on etidronate before Ontario’s Drug Benefit Program (ODB) will fund more potent bisphosphonates (alendronate or risedronate):

**Alendronate (Fosamax)**
- More rapid acting than etidronate
- Reduces vertebral fractures ~50-60%
- Help build bone mass mostly in the spine; somewhat less so in the hip
- Not currently covered on ODB except as limited use

**Risedronate (Actonel)**
- Best single agent, reduces risk within 1 year by 50% and has slightly better hip data than Fosamax; unfortunately, not covered on ODB
- Can use “Pagets Disease dose” (once/week) if dosing causes intolerance.
Urinary Incontinence

Definition: The involuntary loss of urine in sufficient amount or frequency to constitute a social or health problem.

Even though there are aging associated changes in the bladder and the urinary tract which make the elderly person more prone to urinary incontinence, the problem is not and should not be considered as part of ageing.

Normal urination (“To pee or not to pee…”)

There are three nervous-system loops for control of urination, including the cerebral hemispheres, the spinal cord, and the local nervous system. Interruption of any of these loops can cause urinary dysfunction.

- Reflex in sacral micturition center: as the bladder fills, sympathetic tone closes bladder neck (alpha), relaxes the detrusor muscles making up the dome of the bladder, and inhibits parasympathetic tone (via acetycholine). With urination, sympathetic and somatic tone decreases, and parasympathetic impulses causes the bladder to contract allowing voiding to take place.
- Cerebral cortex exerts inhibitory reflex on this process
- Brainstem facilitates urination, and acts as an intermediary between the cortex and the bladder in the spinal cord at the S2, S3 & S4 level.
Causes of Acute, Reversible cause of Urinary Incontinence: DRIP

D  delirium
R  restricted mobility, retention.
I  infection, inflammation, impaction (faecal).
P  polyuria, pharmaceuticals

Causes of Persistent Urinary Incontinence:

**Stress:** Involuntary loss of urine (usually small amounts) with increase in intraabdominal pressure (e.g. cough, laugh, or exercise).

**Urge:** Leakage b/c of inability to delay voiding after sensation of bladder fullness is perceived.

**Overflow:** Leakage (small amounts) resulting from mechanical forces on an over-distended bladder or from other effects of urinary retention on bladder and sphincter function.

**Functional:** Leakage assoc. w/ inability to toilet because of impairment of cognitive and/or physical functioning, psychological unwillingness, or environmental barriers.
Evaluation of incontinence:

Focused history:
- Active medical conditions: CNS disease, diabetes, congestive heart failure
- Medications (especially loop diuretics)
- Fluid intake pattern (use a diary if necessary)
- Past GU h_{x} (childbirth, surgery, dilatations, urinary retention, radiation, recurrent UTIs.).
- Symptoms - onset and duration, type (stress vs. urge vs. mixed vs. other), frequency, timing and amount of incontinence and periods of continent voids.
- Other urinary tract symptoms: Irritative (dysuria, frequency, urgency, nocturia); Voiding difficulty (hesitancy, slow or interrupted stream, straining, incomplete emptying); Other (hematuria, suprapubic discomfort).
- Psychiatric symptoms (apathy, inertia, depression)
- Bowel problems (constipation, stool incontinence).
- Perceptions of incontinence (“Who’s problem is it?”)
- Environmental factors.
- Volume expanded states (liver cirrhosis, CHF, nephritic or nephrotic syndrome)

Targeted Physical examination
- Mobility and dexterity; gait disturbance (? NPH)?
- Mental-status; cognitive function, motivation, mood and affect
- Neurological: focal signs? Signs of Parkinsonism? Sacral arc reflexes?
- Pelvic: perineal skin condition? perineal sensation? Atrophic vaginitis?
- cystourethrocele or pelvic prolapse? pelvic mass ?
- Other: Lower extremity edema or signs of CHF.
Lab work-up of Urinary Incontinence

Step I: start with the following
- Urinalysis & urine C&S
- Blood glucose and calcium levels
- Post void residuals (preferably done with bladder scan, or in-and-out catheterization)

Step 2: Other tests if the diagnosis not clearly established:
- Simple urodynamic tests
- Complex urodynamic tests
- Lab studies: renal function, urine cytology,
- Radiological studies: renal U/S, voiding cystourethgraphy
- urological or gynecological evaluation

Non-Drug Therapy
- If secondary to overflow incontinence, may need intermittent catheterization, in conjunction with bethanacol (remember to stop any anticholinergic drug if this diagnosis is made)
- If 2’ to urge incontinence, try regular toileting (q2 hours when awake), and common sense measures such as reduction of oral fluids, caffeinated beverages, and bedside commodes/urinals within easy reach.
- If 2’ to a functional incontinence (ie. dementia, OA) try frequent and regular timed toileting, and have bedside commodes/urinals nearby
- If 2 to stress incontinence, try smoking cessation (and other measures to eliminate cough), Kegel’s exercises, weight loss in obese patients.
### Drug therapy

<table>
<thead>
<tr>
<th>Control/ (drug examples)</th>
<th>Detrusor</th>
<th>Sphincter</th>
<th>Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetylcholine (Bethanechol)</td>
<td>Contracts</td>
<td>Weakly relaxes</td>
<td>Urinary retention, and obstruction to outflow</td>
</tr>
<tr>
<td>Anticholinergic (Oxybutynin) (Tolteridine) (Imipramine)</td>
<td>Relaxes</td>
<td>Weakly contracts</td>
<td>Urinary frequency and Urge incontinence</td>
</tr>
<tr>
<td>Alpha sympathomimetic (Phenylpropanolamine)</td>
<td>-</td>
<td>Contracts</td>
<td>Stress incontinence; may cause urinary retention.</td>
</tr>
<tr>
<td>Alpha sympathetic Blocker (doxazosin) (terazosin)</td>
<td>-</td>
<td>Relaxes</td>
<td>Prostatism</td>
</tr>
<tr>
<td>Calcium Channel Blockers (any)</td>
<td>Relaxes</td>
<td>-</td>
<td>Urge (theoretical);</td>
</tr>
</tbody>
</table>

For **Anticholinergics** use the minimal dose to assist preservation of cognition
- Oxybutynin (Ditropan): use pediatric syrup form (1 mg) or 2.5 BID
- Detrol (Tolterodine): less dry mouth, ? crosses into brain less than Oxybutynin
- Tricyclic Antidepressants (Imipramine): not used as much now

**PEARL**: Acetylocholine = constricts bladder detrusor muscles causing release of urine; therefore anticholinergics may cause urinary retention (as well as contribute to delirium!); Calcium Channel Blockers may also causes retention
Failure to Thrive (FTT)

NIA definition (National Institute of Aging): "a syndrome of weight loss, decreased appetite, poor nutrition and inactivity, often accompanied by dehydration, depressive symptoms, impaired immune function and low cholesterol"

Failure to thrive, a term more commonly seen in the pediatric literature, has multifactorial causes in the elderly with a DDx that could fill a textbook of internal medicine; you need careful and detailed Hx and a systematic approach of 4 major domains:

- Functional: BADL/IADL
- Malnutrition: wt trend, BMI, lymphocytes and albumin
- Depression: +/-GDS and Cornell
- Cognition: CAM, SMMSE

Potential contributors to all domains:

- Medications
- Orthostatic Hypotension
- Deconditioning
- Unrecognized illness (stroke, endocrine abnormalities, carcinoma, infections)
- Renal impairment
- Vision impairment
- Hearing impairment
- Elder abuse
- Pain
- Fear of Falling
Approach to Failure to Thrive

An interdisciplinary approach should be made, with involvement of the Geriatrician, Family MD, and a Geriatric Assessment team. Depending on the acuteness of the decline, admission to a medical ward or a Geriatric Assessment Unit is recommended for the initial assessment.

**PEARL: An excellent problem for a Comprehensive Geriatric Assessment.**

**Questions to ask on history:**

- Temporal profile of the decline; when did it start, and how fast? (if with days or weeks, suggests an underlying medical cause)
- Last weight before decline? Current weight?
- Changes in medical health?
- Alterations in sense of taste or smell?
- Is the problem that the patient cannot (dysphagia with problems swallowing, new stroke, cognitive impairments), won’t eat (depressed, anorexic, food phobic), or losing weight despite normal intake and appetite (occult malignancy, infection, hyperthyroidism)
- Who does the cooking? Has this changed?
- Any medication changes (prescription or OTC) that preceded the decline?
- Change in living situation, legal status or interpersonal relationships?
- Alteration of finances (loss of income, loss of spouse’s income)
- Is elder abuse or neglect occurring?
- Are their depressive symptoms, or ETOH or other substance misuse?

**Physical Exam**

- Vitals, including accurate height, weight (to calculate BMI) and temperature.
- Focus on ruling out malignancy, occult infection, anemia;
- Check wallowing and test gag reflex
- Abdominal exam; look for masses, jaundice
- Thyroid exam; evidence of hyper or hypothyroidism
- Pelvic and rectal exam (for masses, blood)
- CNS exam; any focal deficits? Problems with gait, or mobility?
Screening Tests for FTT

- Cognitive testing with SMMSE, CDT, or more sophisticated neuropsychiatric instruments.
- GDS to screen for depression

Laboratory investigations

- Basic Geriatric Medicine screen, including CBC, electrolytes, BUN, Creatinine, Albumen, Calcium, TSH, folate, B-12
- Liver enzymes (AST, ALT, Alk Phos, Bilirubin, GGT)
- Nutritional indices that include serum Magnesium, Phosphorus, and lipids and cholesterol (biomarkers for malnutrition)
- ESR (if suspecting occult infection)
- Chest X-ray (if suspecting malignancy)

Functional assessments

- Kitchen and home safety assessments by OT
- Mobility, balance and strength by PT
- Adequacy of current diet and education by Dietary
- Self Administered Medications (SAM) program by Pharmacy
- Speech language pathology referral and video-fluoroscopy swallowing study if suspecting dysphagia

Management

Specific management depends on the specific clinical entities uncovered. It is possible that one or more of the so-called “Geriatric Giants” will be discovered and will require specialized treatment and referral to allied health services. If felt to a Major depression, consider referral to a Geriatric Psychiatrist for consideration of ECT. The treatment recommendations of the Dietician are very important (i.e. meal supplements, referral to “Meals on Wheels” (MOW), placement of feeding tube if deemed necessary, etc.)

Such follow-up and after care services should be monitored by the case manager assigned to the patient.
Frailty

Although MDs often talk about the “frail elderly” there are unfortunately no clear consensus on the definition of this term.

The American Geriatric Society defines it as the “…clinical expression of cumulative biologic changes with aging which result in decreased ability to maintain homeostasis and lead to vulnerability to stressors.”

In other words, “frail seniors” are those older adults who actively demonstrate failure of homeostasis.

This is an important area of health care still requiring further study for the frailest seniors, about 3% of the total, consume ~30% of Canadian health care resources, often in the final years of their life.

Features of fraility include:

- Unexplained weight loss (>5% over a year)
- Poor endurance and energy (self reported)
- Poor strength (in lowest 20th percentile)
- Slow walking speed (Poor “Get up and Go” test).
- Low physical activity (lowest 20th percentile)

Work-up for frailty is identical to that for Failure to Thrive.
Pain in the Elderly

- Pain is an unpleasant subjective and personal experience associated with actual or potential tissue damage; it is the way your body tells you to “stop doing that!”.
- Pain increases in incidence and prevalence after age 60 because of the accumulative burden of such aging associated illnesses such as osteoarthritis, compression fractures, diabetic neuropathy and cancer.
- It is under reported by seniors, who may not tell their doctors because they fear being labeled as bothersome, hypochondriacal, or becoming addicted.
- Twenty-five percent to 50% of elderly people in the community experience pain on a regular basis, and as many as 85% of the elderly in residential facilities report continual pain.
- Unfortunately, pain is under treated in the oldest-old (>85) and in patients with dementia who may not adequately describe their pain.

The most common causes of pain in the elderly stem from:
- Osteoarthritis (prevalence is 49.5% in those 65+)
- Other joint disorders (Rheumatoid arthritis, gout/pseudogout, PMR, SLE, etc.)
- Osteoporosis (e.g. vertebral compression fractures)
- Cancer
- Cardiac ischemia
- Diabetic neuropathy
- Varicella Zoster and post herpatic neuralgia

Assessing Pain in the Elderly (OLD CART mnemonic)
- Onset: When did it start
- Location
- Duration
- Characteristic of the Pain
- Aggravating Factors
- Relieving factors
- Treatment’s taken or tried
Pain Control in the Elderly: Treatment Guidelines

Non Drug Rx

- Icing (maximum 20 minutes/hour, over each hour)
- Heat (but avoid electric heating pads, which can cause burns)
- Hot bath
- Massage therapy
- Unloader braces (knee)
- Use of canes, walkers
- Exercise/Weight training
- Hylan GF 20 (Synvisc); synthetic biopolymer injected into an osteoarthritic joint (usually knee) to restore the shock absorbing, protecting, and lubricating properties of the synovial fluid

Drug Rx

Non-narcotic analgesics

- Acetaminophen (given regularly; maximum dose is 4 grams a day, which translates to 2 Tylenol Extra Strength QID).

PEARL: The "trick" to using medications for pain control is to schedule it regularly, so that patients have the medication on board just prior to when they experience pain. Think of the analogy of brushing your teeth; you don’t wait for a cavity, but brush your teeth to prevent a cavity.

- NSAIDS (only helps if there is an inflammatory pain component; not recommended for OA unless flare)
  - Conventional NSAIDS (extreme caution given the risk of GI bleeding if used without gastroprotection; can also cause such renal side effects, fluid and salt retention, and provoke CHF and renal failure)
  - COX-2 Inhibitors (newer agents, with less associated gastritis, but still have renal side effects can occur)
• Capsaicin cream (Zostrix)
  o Derived from red chile peppers
  o Depletes substance P, and works on Gate control theory of pain by acting as a counterirritant
  o Useful for MSK pain (i.e. osteoarthritis) and some neuropathies

Narcotics (opioids)
  • Tylenol #1/#2
  • Morphine sulfate
  • Hydromorphone

PEARL: Avoid meperidine, in older patients as its metabolites can accumulate in patients with impaired GFR.

PEARL: When starting narcotics, don’t forget to start them on a bowel regimen to prevent constipation! And consider starting long acting dosage forms for chronic pain (i.e. MS contin)

• Adjuvant agents to Rx pain:
  o SSRI Antidepressants (citalopram, sertraline)
  o Tricyclic Antidepressants (nortryptyline)
  o Anticonvulsants (valproic acid, cabamazapine)
  o Calcitonin (for vertebral fractures)
  o Gabapentin (useful for comorbid anxiety)

WHO pain ladder

<table>
<thead>
<tr>
<th>Degree of Pain</th>
<th>Analgesic</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild</td>
<td>Non-opiod +/- adjuvant</td>
<td>Acetaminophen (max 4 grams /24hours) Or NSAID (preferably COX-2)</td>
</tr>
<tr>
<td>Moderate</td>
<td>+/-Weak opiod +/- adjuvant</td>
<td>Codeine 30-60 mg q4h added to above</td>
</tr>
<tr>
<td>Severe</td>
<td>Potent opioid +/- non opioid +/- adjuvant</td>
<td>Morphine 1-5 mg q4h Or Hyrdromorphone 1-2 mg q4h</td>
</tr>
</tbody>
</table>
Elder abuse is defined as any action or inaction that threatens the well being of an older person. A 1990 national telephone survey of 2,000 seniors showed that up to 4% of Canadians over the age of 65 may be abused or neglected. A recent study of 31 Canadian nursing homes found that 36% of nursing home staff had witnessed the physical abuse of an older adult in the preceding year and that 81% had witnessed psychological abuse. A 1988 Health and Welfare Canada study found that financial abuse accounted for over 50% of the documented cases of elder abuse.

Abuse can take many forms, but is usually grouped in five major categories:

**Physical & Sexual abuse**
- Any act of violence or rough treatment, whether or not actual physical injury results
- i.e. slapping, punching, kicking, pinching, burning, restraints.

**Emotional and psychological abuse**
- Any act that diminishes dignity and self worth
- i.e. confinement, isolation, verbal assault, humiliation & infantilization.

**Financial Abuse & material exploitation**
- Any improper conduct that results in monetary or personal loss for the older adult.

**Abandonment & Neglect**
- Active Neglect - intentional (deliberate) withholding of basic necessities and/or care for physical or mental health.
- Passive Neglect - not providing basic necessities and care. There is no conscious attempt to inflict distress.
Medical abuse

- Any medical procedure or treatment done without the permission of the older person or their Power of Attorney or substitute decision maker.

Who are the abusers? Three categories

- Domestic elder abuse (maltreatment by a caregiver in 2/3 of all cases, financial abuse by a distant relative in 90% of cases)
- Institutional elder abuse (abuse in a residential facility)
- Self-neglect or self-abuse.

Reasons for abuse

- Caregiver Stress/burnout & social isolation
- Impairment of Dependent Elder (i.e. dementia)
- Transgenerational “Cycle of Violence”
- Material or other gain

Abuse indicators

- Unexplained physical injuries: burns, cuts, bruises, grip marks, rope marks, burns, cuts, head injuries pain, bruising or bleeding in the genital area
- Unexplained malnourishment or decubitis ulcers
- Unkempt appearance (inappropriate clothing, signs of infrequent bathing or incontinence, a lack of glasses, hearing aids or dentures when they are needed)
- Failure of a medical condition to improve or the continued presence of pain that might indicate under medication.
- Fear of certain family members, friends or caregivers
- The older person is largely ignored or treated passively by caregivers or others
- Caregivers who are entirely ignorant of the medical problems or treatments for the older person they are directly caring for.
Physician Legal Responsibilities

There are no specific laws against elder abuse in Canada, and no legal requirement for physicians to report elder abuse in the province of Ontario. Many believe that laws intended for the general public are adequate for cases of abuse of older adults, with some of the behaviors performed by the abuser can lead to charges of criminal offense (i.e. physical assault, sexual assault, forced confinement theft, fraud, forgery, extortion and the wrongful use of a Power of Attorney).

PEARL: However, physicians who suspect abuse still have the moral duty to act in the best interests of their patients.

What must you do?

An interdisciplinary team approach appears to work best. Physicians (ideally Family Physicians, Geriatricians and Geriatric Psychiatrists) RNs, social workers, lawyers, law enforcement officials, is essential.

If abuse is suspected, interview the patient alone. Begin with open ended questions about , then zero in on direct questions about possible maltreatment and abuse. If abuse is acknowledged, document the nature, frequency, and severity of the abuse (and don’t forget to explore other domains of possible abuse).

Also interview the caregiver. Do not be confrontational. Sensitively enquire about stressors they are undergoing in the performance of their role, and ask what they understand about the medical problems of the older adult in their charge. Ask how many hours they are involved in their care giving role, and if they are getting supports from anyone. If they are responsible for such treatments or medications, ask how this is done and how diligent they are following the prescription’s dosing regimen. Also ask about the possibility of help being provided in the home via CCAC.

If abuse is denied, rigorously document your reasons for abuse (physical signs such as burn marks, rope marks, bruising). Don’t forget to do a SMMSE, as cognitive impairments could cloud the reliability of the older person’s story.
KEY POINT: If the patient is capable of making their own health care decisions, they should decide on their next action (pressing legal charges, changing living arrangements) depending on the severity of the abuse. If they are not competent to make these decisions, immediately notify law enforcement officials if obvious signs of abuse are observed.

Management of Elder Abuse

If the patient is capable:

- Educate re: options (shelter, pressing charges, support services, legal advance
- Support their decision, even if it is one we would not make.

If the patient is incapable:

- Defuse the situation if at all possible and take steps that matters do not worsen; involve CCAC SW and, and see if a case manager can be assigned to follow
- Remove the patient if necessary on a Form 1 if you are concerned about immediate risk or harm
- Meticulously document your reasons for abuse (history, physical exam, abnormalities on X-rays and blood work); consider taking photographs.
- Plan relocation or alternative care with another caregiver
- Involve the police

PEARL: Prevention of abuse is the key, along with early identification of seniors and caregivers at risk, and proper support services started before the abuse begins.

PEARL: There are specific police officers with the Hamilton Regional Police Department who are assigned to crimes involving seniors. Get them involved if you have the patient’s permission, or they are incapable of providing this.
Falls & Poor Balance

Falls are a leading cause of morbidity and mortality in seniors. In Ontario, falls cause 600 deaths annually for those over the age of 65. Falls in the elderly are also the leading cause of injury admissions to Ontario acute care hospitals.

It has been reported that:
- 30% of community dwelling elderly persons fall each year
- 1/2 of these have multiple falls
- 45% of falls occur in residents in Long Term Care facilities

Why else are falls important?
- Fear of falling restricts activities
- Falls are often marker for underlying disease
- 6th leading cause of death
- 5-15 % falls lead to serious injury
- 1-2% of falls lead to hip fractures.

Aetiology (often multifactorial)
- Accidents and environmental hazards
- Drugs
- Dementia
- Syncope
- Seizures
- Lower limb weakness and deconditioning
- Dizziness and/or vertigo
- Vestibular disease
- Orthostatic hypotension (see OH section for more detail)
- CNS disease
Falls Assessment:

Ask about:

- Past medical history
- How long falling; how much, and how serious?
- Exact circumstances of fall (accident, trip, “black out”, “legs just gave way”)
- Previous fractures with falling.
- Medications (Meds that can contribute include diuretics, antihypertensives, neuroleptics, antihistamines, antidepressants, long acting benzodiazepines)
- Do they consume ETOH? How much and how often?
- Collateral history can be very useful!

Physical Examination

- Look for an orthostatic drop (about 25% in patients who fall!)
- Pay attention to cardiovascular system; evidence of arrythmia on taking their pulse? Aortic stenosis murmur?
- Concentrate on CNS examination; tremor and bradykinesia suggestive of Parkinson’s disease? Focal signs of a previous stroke (weakness, upgoing toe, asymmetrical reflexes)? Impaired vibration sense or proprioception (suggestive of a peripheral neuropathy)
- Include gait assessment; examine the speed at which they move, distance between ankles/knees when they walk, if they shuffle when walking, or do not swing their arms. See if they touch the wall for balance, or wobble away from a straight line. Are their turns smooth, or do they trip over their feet
- If they use a mobility aid, examine it. Is their cane the right size for them (and is the rubber worn down?)? Do the brakes work on their walker?
- Don’t forget cognitive testing (i.e. dementia impairing judgement, leading to falls)
Specialty Testing for Falls

Timed “Get up and go” test,
- This is the time it takes for a person to stand up from a straight back chair, walk 3 m, and return and sit down.
- 10 seconds is normal, 11-20 is normal for a frail or disabled patient, and >20 seconds is abnormal and warrants further assessment


Functional Reach
- The maximal distance one can reach forward beyond arm's length, while maintaining a fixed base of support in the standing position.
- An older subject stands relaxed, extending their arm against a wall; a mark is placed at the knuckle position; they are then asked to stretch out and extend their arm as far as possible without losing their balance, and a second mark is done.
- Older persons with a good reach have a better chance of avoiding falls if they can reach out 25 cm (10 inches) or greater; between 15-25 cm (6-10 inches), their odds ratio of falling is 2, if under 15 cm (6 inches), their risk of falling is 4, and if they cannot reach out at all, their odds ratio of falling is 8.
Laboratory investigations for falls:

- CBC, electrolytes, BUN, creatinine, glucose, calcium, albumen, TSH, serum B-12, and folate.
- Do an EKG
- Consider EEG if you suspect seizure activity
- Consider EMG’s if you suspect a nerve conduction problem leading to a peripheral neuropathy

When to order a CT Head?

- Gait ataxia
- Focal neurological signs
- Primitive reflexes
- Cognitive Impairments

Management of Falls

- Treat underlying causes (i.e. if suspect Parkinson’s Disease a trial of levodopa/carbidopa, improve blood sugar control if DM, start on anticoagulation for 2’ stroke prophylaxis if in atrial fibrillation, if B-12 deficient, start replacement therapy, etc.)
- Review medications and consider their necessity (this may involving contacting other physicians, such as the Family MD and other specialists) and whether if they can be reduced or discontinued.
- Taper sedatives gradually before stopping (with avoid withdrawal), reduce neuroleptics (or change to a 2nd generation atypical neuroleptic, etc.), stop dimenhydrate (Gravol), etc.
Management of Falls (continued)

- Cut down or stop ETOH if this is playing a role (falls while inebriated, peripheral neuropathy)
- Rehabilitation with referral to OT (home visit to examine hazards and to see whether devices need to be installed or have the environment modified), PT (for strengthening or balance exercises), Day Hospital or Falls clinic referral (for complicated patients who need interdisciplinary approach)
- Enhance mobility (cane, walker, rails), improve vision, improve environment
- Consider hip protectors (either hard plastic shell or soft foam “hockey pants” that protect the femoral heads from a direct blow if there is a fall).
- Consider getting a BMD, and starting on calcium/vitamin D; if osteoporosis, start on a bisphosphonate.
- Recommend a “Lifeline” or other device that the older person can use to signal for help if they’ve fallen and can’t get up.
- If poor balance or deconditioning, train for preventing falls, by recommend they start Tai Chi, or joining a seniors fitness group
Dizziness

Often multi-factorial in aetiology:

- Vertigo
- Presyncope
- Dysequilibrium
- Anxiety and or depression may be playing a role

Presbystasis: minor age related changes in proprioception, vestibular function, neuromuscular function and age that can contribute to unsteadiness in gait.

Evaluation of dizziness: Questions to ask on Hx:

- What do they mean by “dizzy” (light headed, room spins, unsteadiness of feet but head okay, loss of balance or veering to one side, etc.)?
- Onset + duration? Single attack or multiple?
- Are the spells becoming more frequent, or lasting longer?
- Any warnings it’s about to start?
- Symptoms between attacks?
- Is it brought on by standing, lying, rolling over in bed, neck movements, when walking, walking in the dark.
- Any fainting episodes?
- Past Medical history
- History of TIA, stroke, neglect?
- Concurrent illness
- Neurological review
- CVS review
- History of Head trauma or Falls?
Recent viral infection or ear infection? Recent hearing loss?
Recent medication change?
Any recent change in eye-glasses?

Medications that can contribute:
- Diuretics (loop especially)
- Antibiotics (e.g. gentamycin)
- Antihypertensives
- Neuroleptics (antipsychotics)
- Antihistamines
- Long acting Benzodiazepines
- ECASA/NSAIDS (can also cause tinnitus)
- ETOH (also ask about depression and/or anxiety)

Physical Examination for Dizziness:
- Vitals: any orthostatic changes?
- Vision/hearing screen
- Otoscopy
- CVS exam
- Neck ROM, head turning
- Cranial Nerve (CN) nerve exam
- LE tone, strength, power
- Proprioception testing.
- Cerebellar function testing.
- Dix-Hallpike maneuver: Starting with the patient in a sitting position on the examination table, rapidly lie them backward with their neck extended and their head turned to the left; repeat again, with the head turned to the right. Each time observe if they demonstrate nystagmus or a replication of their dizzy symptoms (see picture).

PEARL: do not do the Dix-Hallpike in elderly patients with significant carotid artery disease; listen for bruits first, and if positive (or positive on history), do not do.
**Laboratory investigations:**
- Potassium, renal function, blood glucose, B-12, CBC.
- EKG
- Consider EEG if suspect seizure
- Consider also VDRL, digoxin and ASA levels (only if taking).

**Management:**
- Treat underlying causes
- Consider trial of antidepressant if nothing can be found
- Review meds, and reduce or stop contributors
- Rehabilitation with referral to OT, PT, Day Hospital
- Enhance compensation mechanisms- improve vision, cane, walker
- If suspect *Benign Paroxysmal Positional Vertigo* (BPPV) from a (positive Dix-Hallpike) consider referral to an ENT specialist

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*Dix-Hallpike*
Although seniors represent 12% of the Canadian population, they consume 28% to 40% of all prescriptions. Unnecessary prescribing, misuse of medication and inappropriate prescriptions can contribute to the risk of drug-related illness and result in unwarranted costs in health care delivery.

An estimated 5% to 23% of hospital admissions are due to drug-related illness. Physical impairments and death (estimated 200,000 per year in the US) are all too common tragic results.

Polypharmacy literally means “Many Drugs”, and it reflects the problems that occur when persons are taking too many (or simply too many of the wrong drugs, which is iatrogenesis); it is an older term that is falling out of use. Previously defined as 5 or more new drugs during a given 3 month period, it is estimated that 15% of patients are at risk of from polypharmacy.

A more current term is Medication Optimization (since many patients truly need the many medications for their plethora of problems), and the thinking has now shifted from simply reducing medications, to ensuring than an older person be on the optimum number and dose of medication for their problems.

Medications that may have been relatively benign when the patient was younger, can accumulate risks as the person ages (and with that, changes in drug metabolism and pharmacokinetics). The main concern in the elderly is that side effects should not outweigh beneficial effects.
Frequent drug side effects in the elderly include:

- Falls
- Arrhythmias
- Confusion (from sedation, anticholinergic effects)
- Dehydration from diuretics
- Hypotension from many cardiac medications
- NSAID gastropathy
- Digoxin toxicity (even with so-called therapeutic dig levels)
- Insulin Hypoglycemia

Medications causing unwanted side effects may be prescribed by different well-intended MDs (Iatrogenesis), be purchased over the counter or even “borrowed” from another person. What can complicate matters are patients who obtain their medications from different MDs, or from different pharmacies.
Drugs to avoid in the Elderly

Amitriptyline (Elavil)
- Use less anticholinergic agent; change to nortriptyline (same dose), or use an SSRI antidepressant instead.

Benztropine (Cogentin)
- Used to treat EPS side effects of older neuoleptics, as well as in Parkinson’s, but itself can cause confusion and delirium itself; taper gradually and observe.

Cimetidine
- Use a less anticholinergic H2 blocker (i.e. Ranitidine). Ask, do they need it, and if the need is established, consider an alternative

Dimenhydrinate (Gravol)
- Too anticholinergic (and a major cause of delirium in older adults); try using Domperidone instead for nausea.

Fluoxetine (Prozac)
- Very long half life; try newer SSRI antidepressants.

Indomethacin (Indocid)
- Safer agents less likely to cause NSAID gastropathy are readily available, such as COX-2 Inhibitors (or Acetaminophen if non inflammatory pain).

Long-acting Benzodiazepines (Valium, Dalmane, Librium)
- Use short-acting agents (i.e. Lorazepam, Oxazepam) as can cause excess sedation and cognitive impairments. Avoid entirely if possible.

Methyldopa (Aldomet)*
- Safer agents available for controlling blood pressure

Pentazocine (Talwin)
- Safer, more effective agents available

Propranolol (Inderal)*
- Other beta-blockers cause fewer central CNS effects (i.e. atenolol)

Theophylline
- Can cause nausea, vomiting, and seizures in seniors, especially if liver impairment. Other safer asthma medications are available

* Note: These agents may be acceptable in some instances. Although they cause CNS side effects frequently in the elderly, they have been proven to decrease morbidity and mortality in some circumstances.
Optimizing Medications for the Elderly

A. Ask yourself, “Why they are on the following medication?”:
   - Benztropine (Cogentin)
   - Haloperidol (Haldol)/ Chlorpromazine (CPZ)
   - Long Acting Benzodiazepines
   - Narcotics (ie. Tylenol #3)
   - NSAIDs and hypertension/renal impairment

   *If no good reason, titrate down and STOP.*

B. Ask why yourself why they are NOT on the following medications
   - Calcium
   - Vitamin D
   - Bisphosphonate (if has osteoporosis or a clinical fracture)
   - ECASA (if previous CVA, MI or IHD)
   - Warfarin (if in A-fib)
   - Antidepressant (if depressed)
   - ACE Inhibitor (if has CHF or HTN with DM)
   - Cholinesterase Inhibitor (if diagnosed with mild-moderate AD and living at home)

   *If no good reason, consider starting!*

Capacity Assessment is the formal assessment of a person's mental capacity (competence) to make decisions. Medical treatment, management of property and personal care are areas where capacity assessment is often necessary.

It is a process, not a result, and the purpose of capacity assessment to see where a person lies on a continuum between autonomy and beneficence

- In Ontario, one can be judged capable or incapable for issues surrounding health care and finances alone
- However, other domains exist for some physical tasks or functions (driving) versus cognitive (i.e. advanced directives); most health care decisions are cognitive; a diabetic self administering meds has both a cognitive and functional components

PEARL: Capacity assessments are intrusive and can have extensive outcomes; one can take away more fundamental rights given to a prisoner in jail. Making someone incapable should be the last thing you do for a person.

In Ontario, MD’s can assess financial and personal care capacity only in a Schedule 1 Hospital, but not outside of hospital. This is where a capacity assessor may be needed. If a person has a signed POA and becomes incompetent, an MD may simply write to the lawyer with their opinion whether they believe they are capable or not (and reasons why).
How to measure capacity? 4 Step Process

A. Trigger
- Something that puts a person or others at significant risk
- Have impaired decision making
- A new change or shift; making choices at odds to previous personality
- Only if there will be a concrete change

B. Assent
- The person must participate in the process because it is in their best interest

**PEARL:** If they don’t consent to the assessment, you cannot assess capacity!

C. Assessment

1. Information gathering: talk to family, lawyers, nurses, etc.
   - does the patient understand the following parts of each individual decision?
     - context
     - choices
     - consequences
   - Education: must not mistake ignorance from incompetence
Action

- Decision made of competent or not; if not needs POA or some form of substitute decision maker.

There are many dilemmas and ethical questions surrounding matters of competency and capacity. While any MD can make a determination of capacity, few are trained in this process (or want to be involved in this medical legal process).

Formal assessors can be available (for a steep fee) to help make this determination, and they can be arranged via the Capacity Assessment Office of the Ontario Ministry of the Attorney General.

PEARL: If one has full understanding of the choices they have, disagreement with the advice of their MD’s does not necessarily mean incompetence. It is the process that counts, not the result. The analogy is that of a person living in a condemned house: if they know that the place may fall about their head any moment, while we may not agree with their living situation, we must abide by it. In contrast, if one is living in a condemned house but denies that there is anything wrong with such an unsafe living arrangement and denies that they may come to harm, in this instance they are not capable of understanding the risks of their current situation and are therefore not capable of making such living decisions on their own.

Capacity to Decide, D.W. Molloy, P. Darzins, D. Strang, 300 pp., 1999
Appendix A

Screening Tools and Instruments used in Geriatric Medicine:

Confusion Assessment Method (CAM)

Standardized Mini Mental State Examination (SMMSE)

15 Item Geriatric Assessment Scale (GDS)

Functional Independence Measure (FIM)
CONFUSION ASSESSMENT METHOD (CAM)

The Confusion Assessment Method (CAM) as developed by Sharon Inouye, was designed specifically to diagnose delirium.

There are four key features to the CAM: After seeing the patient, and reviewing the history of cognitive changes, was there

1. acute onset and fluctuating course?
2. lack of attention?
3. disorganized thinking?
4. altered Level of Consciousness?

If 1 and 2 are present, and either 3 or 4, the patient is probably delirious.

Gold Standard: Geriatric Psychiatry Interview
- Sensitivity: 46-94%, Specificity: 92-98%
- + Likelihood Ratio (LR) is 8.8; -LR is 0.2

PEARL: Caveats about using the CAM are in patients with dementia (chronic confusion), or patients with primarily psychotic symptoms. The CAM was also not validated in younger populations.

STANDARDIZED MINI-MENTAL STATE EXAM (SMMSE)

Based on the copyrighted Mini-Mental State Examination (MMSE), as developed by Folstein, Folstein, and McHugh (1975), it has undergone refinement and changes over time to the current form. The currently used version is (scoring in parentheses):

“Now I’d like to ask you some questions to check on your memory; this is a standard test we do on all our patients.”

ORIENTATION TO TIME
(1) What year is this?
(1) What season of the year is it?
(1) What is the month?
(1) What is the date?
(1) What day of the week is it?

ORIENTATION TO PLACE
(1) What is the name of this place?
(1) What floor are we on?
(1) What city are we in?
(1) What province are we in?
(1) What county is this?

IMMEDIATE RECALL
(3) I am going to say 3 objects. After I say them, I want you to repeat them. They are: "Car" "Ball" "Man" Now you say them. Remember what they are because I'm going to ask you to name them again in a few minutes.
[Interviewer: Repeat until all 3 are learned]

ATTENTION (either item)
(5) a) Subtract 7 from 100, then subtract 7 from the answer you get and keep subtracting 7 until I tell you to stop.
   Or b) Spell the word "WORLD" backwards [DLROW].

NAMING
(2) Show patient wrist watch and pen and ask to name them.
REPETITION
(1) Repeat the following sentence exactly as I say it. "No ifs, ands, or buts."

3 STAGE COMMAND
(3) Now I want to see how well you can follow instructions. I'm going to give you a piece of paper. Take it in your right hand, use both hands to fold it in half, and then put it on the floor.

READING
(1) Read the words "Close your eyes" (provided on a sheet of paper prepared previously) silently to him/herself, and then do what it says.

WRITING
(1) On same sheet of paper, ask patient to write a complete sentence.

COPYING
(1) Give patient clean sheet of paper and ask him/her to copy the design of intersecting pentagons.

DELAYED RECALL
(1) What are the 3 words I asked you to remember earlier? (At least a 3 minute delay, 1 point for each, score out of 3)

Total Maximum score = 30
A score of 23 or less is probably abnormal by this screening test and suggests further cognitive testing and a search for the reason are needed; scores 24-30 may need further evaluation based on patterns of deficits (i.e. recall 0/3), or previous educational an intellectual achievements (e.g. a 65 year old lawyer with a SMMSE of 26/30 is a red flag that something is wrong). If a person is blind, cannot read or write, or has aphasia, note their deficits and provide scores out of this new denominator (i.e. 24/27)
Geriatric Depression Scale (GDS)

This scale was developed as a basic screening measure for depression in older adults, and is in the public domain. While there are 4 point scales and 30 point scales in circulation, there is no advantage in having more questions asked than on the 15 item version below (Pomeroy et al, Int J. Geriatric Psychiatry. 2001; 16:321-6).

Ask the person you are screening to choose the best answer for how they have felt over the past week or so:

1. Are you basically satisfied with your life? **YES** / **NO**
2. Have you dropped many of your activities and interests? **YES** / **NO**
3. Do you feel that your life is empty? **YES** / **NO**
4. Do you often get bored? **YES** / **NO**
5. Are you in good spirits most of the time? **YES** / **NO**
6. Are you afraid that something bad is going to happen to you? **YES** / **NO**
7. Do you feel happy most of the time? **YES** / **NO**
8. Do you often feel helpless? **YES** / **NO**
9. Do you prefer to stay at home, rather than going out and doing new things? **YES** / **NO**
10. Do you feel you have more problems with memory than most? **YES** / **NO**
11. Do you think it is wonderful to be alive now? **YES** / **NO**
12. Do you feel pretty worthless the way you are now? **YES** / **NO**
13. Do you feel full of energy? **YES** / **NO**
14. Do you feel that your situation is hopeless? **YES** / **NO**
15. Do you think that most people are better off than you are? **YES** / **NO**

Items in **BOLD** get one point; others get no points.

**Scoring:** For most clinical purposes a score > 5 points is suggestive of depression and warrant a follow-up interview. Scores > 10 are almost always depression.

Sensitivity of a score of >10 of the 15 item GDS is 82%, specificity is 60%.
Functional Independence Measure (FIM)

The FIM instrument was derived from the Barthel Index and uses seven levels of function, two in which no human helper is required and five in which progressive degrees of help from another person is needed. It is regarded throughout the world as one of the most reliable and uniform methods for documenting the severity of an inpatient’s disability and is used to evaluate functional outcomes of adult inpatients undergoing medical rehabilitation.

Specifically, the FIM is made up of 18-item, rated on a seven level scale (7=independent, 1= total assist), and was originally designed to track the improvement of patients during rehabilitation.

The higher the FIM score, the more the person is able to do independently; the lower the FIM, the greater the functional disability. The range of scores ranges from 18 (total care) to 136 (totally independent).

A FIM completed by a person trained to use this instrument at the beginning of Rehab to document his or her level of independent functioning versus the need for assistance from another person. Following the course of rehab treatment, the FIM instrument is used again at discharge and for follow up.

A minimal FIM instrument score of 60 or greater increases the chances of successful discharge in the community, and is the minimum level used by patients treated at the Hamilton Wentworth Geriatric Assessment Unit at the Hamilton General campus.

7 Level Scale Used:

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>7</td>
<td>Complete Independence (timely, safely)</td>
</tr>
<tr>
<td>6</td>
<td>Modified Independence (extra time, devices)</td>
</tr>
<tr>
<td>5</td>
<td>Supervision (cuing, coaxing, prompting)</td>
</tr>
<tr>
<td>4</td>
<td>Minimal Assist (performs 75% or more of task)</td>
</tr>
<tr>
<td>3</td>
<td>Moderate Assist (performs 50%-74% of task)</td>
</tr>
<tr>
<td>2</td>
<td>Maximal Assist (performs 25% to 49% of task)</td>
</tr>
<tr>
<td>1</td>
<td>Total Assist (performs less than 25% of task)</td>
</tr>
</tbody>
</table>
Domains on the FIM:

SELF CARE ITEMS

*Feeding/Grooming/Bathing/Dressing Upper Body/Dressing Lower Body
Toileting/Swallowing*

SPHINCTER CONTROL

Bladder Management/Bowel Management

MOBILITY ITEMS (Type of Transfer)

Bed, Chair, Wheelchair/Toilet/Tub or Shower/Car Transfer*

LOCOMOTION

Walking/Wheelchair (circle)/ Stairs/ Community Access*

COMMUNICATION ITEMS

Comprehension-Audio/Visual (circle)
Expression-Verbal, Non-Verbal (circle)
Reading*/ Writing*/ Speech Intelligibility*

PSYCHOSOCIAL ADJUSTMENT

Social Interaction/Emotional Status*/Adjustment to Limitations*
Employability*

COGNITIVE FUNCTION

Problem Solving/Memory/Orientation*/Attention*/ Safety Judgment*

Appendix B

Interesting Syndromes

Work-up and \( R_x \) of Delirium and Agitation
Some Curious Syndromes You May Encounter

Anton’s Syndrome: Cortical blindness, and the bizarre denial of blindness in the blind person (i.e. not only do they not know they are blind, but they don’t know that they don’t know, and fabricate what they think they are seeing). Occurs when bilateral occipital strokes are combined with lesions in the parietal lobes.

Charles Bonnet Syndrome: Vivid cinematic hallucinations in older adults with normal cognition (i.e. insight into the unreality of the visions must be full and prompt). Felt to be 2° to vision problems (the ocular equivalent to “phantom limb pain”). An unknown number may progress to Lewy Body Dementia.

Diogenes Syndrome: A syndrome characterized by 1) Extreme self neglect 2) domestic squalor 3) syllogomania (excessive hoarding) 4) social withdrawal and 5) lack of concern/shame about living conditions. Not a well understood entity, and covers a mixed bag of possible diagnoses ranging from dementia, late onset psychosis to simple eccentricity. More of a social problem than a medical one. (aka Senile Squalor Syndrome or better, Social Breakdown Syndrome).

Gerstmann’s Syndrome: Finger agnosia, right-left confusion, dyscalculia, and dysgraphia. Seen in lesions of the association area of the dominant parietal lobe of the brain.

Wernickes Syndrome: Thiamine deficiency leading to an encephalopathy characterized by a triad of confusion, ocular dyskinesias (diplopia and nystagnus), and ataxia. It responds to thiamine therapy. It is mostly commonly seen in alcoholism, and may proceed to Korsakoff’s Syndrome).
Work Up for Confusion/Delirium:

If an older person comes into the ER confused, and the aetiology is not immediately clear, get a history from the family if the confusion is acute or chronic. Get as much background info as possible (i.e. do they have an underlying dementia? Do they consume ETOH? Recent hospitalization or medication change?).

Try to get a list of medications from the ambulance attendants). If it’s the daytime, contact the Family MD’s office, or the patient’s pharmacy for clues for past medical conditions.

There are several screening tests that have a high diagnostic yield in sick, older adults:

- CBC with differential (may indicate occult infection, anemia, malignancy)
- Electrolytes, urea, creatinine (renal failure, hyper/hypo kalemia/natremia)
- Blood sugars (hyper/hypoglycemia, occult DM)
- Serum albumin, Calcium, Alk-Phos, ALT (looking for liver failure, malnutrition, malignancy)
- Get a blood ETOH level if you suspect delirium tremens from withdrawal
- Cardiac markers i.e. CK, Troponin (occult MI, coronary syndrome)
- SpO2 (hypoxia from occult pneumonia, PE, CHF)
- EKG (occult cardiac ischemia, arrythmia)
- CXR (occult pneumonia, effusion, malignancy, CHF)
- CT (occult CVA, SDH, intracranial bleed, tumour)
- Urine for white cells, nitrite, blood and protein (occult infection, nephrotic/itic syndrome)
- Urine specimen for Gram stain, culture and sensitivity ie C&S (may indicate occult UTI)
- Don’t forget PUS (Pneumonia/Urine/Skin) is the cause of 90% of infections in older people.
Management of Delirium

• Treat the underlying cause if you discover it.
• Do frequent CAM to follow the progress of the delirium.
• Provide supportive care (IV fluids if not drinking, O2 if hypoxic).
• Move the patient to a quite, well lit room.
• Have OT provide orientation measures; calendar, clock, and message board (i.e. a placard stating “It is Tuesday February 5th, 2002, and you are in room 405 at the Hamilton General Hospital”).
• If problems sleeping because of with the delirium (“up all night”), consider starting Trazadone, beginning at 25 mg po QHS.
• Minimize the use of restraints (which can both cause or maintain a pre-existing delirium); get one-on-one care if at all possible, or at least have a family present to soothe and console if possible.

Management of Acute Agitation in Delirium

• If agitated, violent and combative, the immediate goal of treatment of treatment is to reduce symptoms to a level where the patient is not a physical danger to himself or others. The cessation of hallucinations and/or delusions (assumed to be the basis of the dangerous agitated behavior), is the ultimate goal. Current management of acutely agitated patients involves the use of neuroleptics and benzodiazepines either alone or in combination.
• Consider an oral second generation neuroleptic; Risperidone (Risperidal) 0.25 mg BID, or olanzapine (Zyprexa) 2.5 to 5 mg QHS (there is now a fast-acting gelatin sub-lingual wafer version of olanzapine that immediately dissolves under the tongue that will be shortly released).
• If they unwilling to take medications orally, use IM haloperidol (Haldol) 0.5 mg or IM olanzapine (Zyprexa) 2.5-5 mg QHS which should work within 30 minutes if combined with an adjuvant benzodiazepine.
• 1-2 mg lorazapam, either po or IM, can be given q 1 hour in addition to the neuroleptics above until sedation is achieved (e.g., unresponsive to verbal stimuli, but responsive to a noxious stimulus).
Appendix C

References & Recommended Reading
Textbooks
Other books
Journals
Original articles

Final Geriatric Pearls

Abbreviations and Acronyms Used
**References & Recommended Reading List**

**Textbooks:**

PEARL: Don’t buy these for your Geriatric Medicine clerkship experience; you can find them in most medical & hospital libraries; the first two are the two leading authoritative textbooks in Geriatric Medicine


**Geriatric Medicine Journals:**

- *Age and Aging*
- *The American Journal of Geriatric Psychiatry*
- *Archives of Gerontology and Geriatrics*
- *Canadian Journal of Aging*
- *Journal of the American Geriatrics Society (JAGS)*
- *Geriatrics*
Other Useful Books

- Parenting Your Parents (2nd edition) by Bart Mindzenthy & Michael Gordon [as the above, but with a Canadian & multicultural perspective]

Original Papers/Articles:

The Geriatric Medicine Model


Geriatric Assessment

Cognitive Testing


Pre-Op Assessment in the Elderly:

2. Clark. "Preoperative Assessment: primary care work-up to identify surgical risks" *Geriatrics 56 (7) 36-40, July 2001*

Dementia

2. Canadian Study of Health and Aging: study methods and prevalence of


4. Patterson, CJS and Gauthier, S. (Editors) Canadian Consensus Conference on Dementia. Can J Neurol Sci 2001-28 (Supplement 1) SI-S 123 (Supplement on Dementing Disorders)


Delirium


Depression

Frailty & Failure to Thrive

Falls and Risks of Falls

Bone Disease


5. 2002 clinical practice guidelines for the diagnosis and management of osteoporosis in Canada. *CMAJ November 12, 2002; 167 (90100)*

**Polypharmacy, Iatrogenesis & Optimizing Medications**


**Pain & Pain Control**


**Elder Abuse**


Miscellaneous Topics

2. Hogan, DB. Imposed Activity Restriction for the Elderly *AnnaIs RCPSC* 198 5; 18:410-412
FINAL GERIATRIC PEARLS

The longer we live, the more important basic functions are (BADLs & IADLs) in order to maintain persons in their own homes as long as they can; a nursing home costs $40k a year, but if you can maintain seniors at home with CCAC supports you can save half that cost (and more importantly, improve their quality of life).

Remember, if you are taking care of an older person in an acute care hospital:
• Get PT, OT & CCAC involved early for discharge planning.
• Strongly consider getting SW, involved, too if discharge will likely be an issue.
• Always screen for cognitive impairments (if only the year, or a clock) and depression
• Always get a collateral history, and find out if caregiver stress is present; the most underutilized diagnostic test is the telephone.
• Anemia is never normal in an older person; work it up!
• Incontinence is not part of normal aging; work it up!
• Think thrice about any drug you give an older person; will it do more harm than good? Will it interact with other medications they are on, and impact on their function?
• Older persons are more sensitive to medications; when starting new drugs, start low, and go slow (i.e. ¼ the usually starting dose for psychotropics). Also, look over their old meds, and see if they really need all what they are taking.
• Look for the atypical presentations of typical diseases in older persons
• Listen, look and touch your patients; don’t be a “foot of the bed” MD.

And finally, strongly consider doing an elective in Geriatric Medicine, to gain in-depth exposure to the treatment and management of older persons in many different settings (GAU, out-patient, Day Hospital, Outreach, Geriatric Rehab, etc). Persons interested in Family and Internal Medicine would certainly benefit (since a good many patients will be categorized as geriatric), but no matter what branch of medicine you go into (even radiology or pathology!) you will have aging relatives who would benefit for your Geriatric Medicine knowledge

Good luck on the Wards!

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Abbreviations and Acronyms Used

AD: Alzheimer’s Dementia
ADR: Adverse Drug Reaction
ALC: Alternate Level of Care
ASA: Aspirin
BADL: Basic Activities of Daily Living
BMI: Body Mass Index
BP: Blood Pressure
CA: Cancer
CAM: Confusional Assessment Method
CBC: Complete Blood Count
CCAC: Community Care Access Centre (previously “Home Care”)
CDT: Clock Drawing Test
CHF: Congestive Heart Failure
CJD: Creutzfeldt-Jakob Disease
CNS: Central Nervous System
CXR: Chest X-ray
CVA: Cerebrovascular Accident
Dx: Diagnosis
DDx: Differential Diagnosis
DLB: Dementia with Lewy Bodies
DVT: Deep Vein Thrombosis
ECASA: Enteric Coated Aspirin
ETOH: Ethanol, alcohol.
FIM: Functional Independence Measure
FTD: Frontotemporal Dementia
GAU: Geriatric Assessment Unit
GARU: Geriatric Assessment and Rehab Unit
GFR: Glomerular Filtration Rate
GI: Gastrointestinal
GDS: Geriatric Depression Scale
Hx: History
HCTZ: Hydrochlorothiazide,
HTN: Hypertension
IADL: Instrumental Activities of Daily Living
Ix: Investigations
IHD: Ischemic Heart Disease
LTCF=Long Term Care Facility
MI: Myocardial Infarction
MOTC: Ministry of Transportation & Communication
MOW: Meals on Wheels
NPH: Normal Pressure Hydrocephalus
nvCJD: new variant Creutzfeldt-Jakob Disease
ODB: Ontario Drug Benefits
OA: Osteoarthritis
OT: Occupational Therapy
OTC: Over The Counter
P_x: Physical Examination (aka P/E)
PD: Parkinson’s disease
PLT: Platelet count
PMH_x: Past Medical History
PND: Paroxysmal Nocturnal Dyspnea
POA: Power of Attorney
PT: Physiotherapy
PTH: Parathyroid hormone.
RN: Registered Nurse
R_x=Treatment
RA: Rheumatoid Arthritis
SSME: Standardized Mini Mental State Exam
SBE: Subacute bacterial endocarditis
SOB: Shortness of Breath
SOBOE: Shortness of Breath on Exertion
SW: Social Work/Social Worker
TCA: Tricylic Antidepressant
TD: Tardive Dystonia
TIA: Transient Ischemic Attacks
UTI: Urinary Tract Infection
VaD: Vascular Dementia
VA: Visual Acuity
WBC: White Blood Cell
3MS: Modified Mini-Mental State Examination