Exploring Elder Psychiatry

Paul Kettl M.D.

EXPLORING ELDER PSYCHIATRY

By Paul Kettl, MD, MHA

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For Kathleen

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CHAPTER 1: INTRODUCTION

Welcome to this exploration of elder psychiatry, or geriatric psychiatry. The term "elder psychiatry" is deliberately chosen to reflect respect for those who spent their lives assisting all of us as we have grown with our families, and our roles. The book grows out of years of teaching the topic to medical students, psychiatry residents, as well as psychologists, nurses, nursing assistants and social workers who are so essential in the care of elders. I have tried to avoid medical jargon to make the subject more understandable, especially with the more complicated neuroscience involved in dementia and dementia care.

Because I get bored so easily reading text, the book is arranged in a series of "bullet points" hopefully arranged to convey the information quickly in a way that conveys the essential points. In this way, I hope you can gain access to the essential points of each chapter in as efficient a way possible. For each chapter, there is a brief introduction and at the end is a summary of the essential points conveyed. So, if you desire an even briefer review than the chapter provides—the summary at the end of each chapter would be a place to start for you. In addition, I have tried to include the information conveyed in the most recent articles, and the citation for the research article

discussed is displayed immediately after each point—again to make finding the article, and linking it with any particular topic easier.

So, then, the book is not meant to be a comprehensive textbook of geriatric psychiatry, but rather a quick overview of essential points of caring for the psychiatric problems of elders in a variety of topics. References for research articles are listed throughout the chapters for more full exploration as you desire.

The book also includes not only the typical topics in geriatric psychiatry such as care for elder depression, a discussion of the dementias, and delirium, but also in topics that are too often ignored in the study of elder psychiatry. A chapter on alcohol and drug abuse in elders is presented exploring the growing problem of elder drug abuse, especially prescription drug abuse, in this cohort of elders who came of age in the 1960's, when "peace, love and rock and roll", and drug use was more common. A chapter on intellectual disability in elders is also presented. The major public health success story you never heard of is the lengthening of the lifespan of those with Down's syndrome. In just a few years before the turn of the century, their lifespan doubled, and we in psychiatry are often presented with these individuals who are now surviving their caregivers. Finally, a chapter on ethical problems in caring for elders is presented. The problem in ethics is not just seeking the right thing to do, but in selecting which right principle to follow.

And, this is just a beginning. Elder psychiatry furnished me with a lifelong series of fascinating clinical experiences, working with people who share their lifelong insights with me. The only problem is how little we really know about the diseases that sap life and health from our elders, and how slow we are learning about those problems.

I hope you can change that.

CHAPTER 2: PSYCHOTROPIC PHARMACOLOGY IN ELDERS

It is helpful to begin our exploration of elder psychiatry by looking at the various ways elders are different in the way they metabolize drugs. Elders have a variety of chronic illnesses, including psychiatric illnesses. For these illnesses, they often receive a variety of medications. However, the combination of these medications can lead to troublesome or dangerous side effects. Medication use in elders should always be carefully evaluated, and treatment progress and presence of side effects should be continually monitored. The fact that elders frequently are prescribed medications, and take a combination of medications combined with our psychotropic medications makes it even more essential to monitor medications in elders.

The following pages present a brief overview of psychopharmacology in elders:

MEDICATION IN THE ELDERLY

Drug use in elders:

- Elders (over 65) are 12% of population

- They consume 25% of all drugs
- 87% of elders use over the counter meds
- 6% are taking 5 or more over the counter meds

Rules for prescribing for the elderly:

- For a new patient—the shopping bag test (i.e. bring in all pills from the medicine cabinet) is helpful. This allows you to see not only what they are taking, but what they have taken, and this gives you a good health history. Because elders may take meds that were prescribed in the past, and may share them with friends, bringing in the "shopping bag" also allows you to discard meds they no longer need, or meds that may dangerously interact with meds they are currently taking.
- Stop as many pills as you can (the more drugs, the more chance for drug interactions, and side effects of current drugs). …the good geriatric psychiatrist giveth, and the good geriatric psychiatrist taketh away.
- Start low, go slow (I will explain why in a moment)
- Monitor drugs on a regular basis
- Avoid polypharmacy
- Ask about drug side effects
- Are they compliant?? (Compliance in general is 25% to 50%

among all patients, because of cost, side effects, denial of illness, etc.). The most common reason why drugs don't work is because patients don't take them.

Start low, go slow is the cardinal rule.

- Elders have different metabolism
- They have DECREASED:
    - muscle mass
    - bone mass (So, if an elder is sedated, or dizzy or off balance from side effects of their medications, they are more likely to break a bone, and less able to catch themselves because they have less muscle mass.)
- Elders also have less body water. (We are mostly water, and less water means a smaller volume for the drug to be dissolved in. A smaller amount of volume means a greater concentration of the med that is put into that smaller concentration.)
- Elders also have less serum protein. (Many of the drugs we use are protein bound, and less serum protein means that less of the drug is "bound" in the blood, making more of the drug "free" to bounce around and act.)
- They have INCREASED:
    - body fat (Some drugs are fat soluble, and this also gives a bigger "storage area" for the drug to sit in.)

- All of these factors mean:
    - a lower volume of distribution, leading to a higher concentration of the drug
    - drugs hang around longer also leading to a higher concentration

Drugs are metabolized by the liver (for the most part) or excreted by the kidneys. Does the liver and kidney change as we get older? (everything does.)

Hepatic (or liver) drug clearance in the elderly:

- Hepatic blood flow is decreased in the elderly
- Serum protein binding is decreased in the elderly
- Capacity of the hepatocytes, or the individual liver cells, is about the same
- SO DECREASED HEPATIC DRUG CLEARANCE IS SEEN IN ELDERS

This means that the liver is slower in getting rid of drugs, so they build up more in elders.

Renal (or kidney) excretion of drugs in elders:

- GFR (glomerular filtration rate, or the rate of the kidney to

cleanse the blood) decreases 30% from the 3rd decade to the 8th decade of life

- Serum creatinine, the main measure of kidney function—which is a blood test—does not diminish as much. Muscle mass decreases too, at about the same rate. Serum creatinine comes mainly from muscle. So, kidney function decreases at about the same rate that muscle mass decreases. So, serum creatinine remains about the same.

- Serum creatinine overestimates renal function in the elderly

In summary, when considering medication use in elderly patients:

- Medications have a lower volume of distribution (i.e. a higher concentration)

- Medications hang around longer

- Medications are excreted slower

- THIS LEADS TO PROBLEMS

- Elderly are more likely to have any side effect

DRUG-DRUG INTERACTIONS

Now, lets look at drug-drug interactions. There are 3 basic types:

- Idiosyncratic—occur unpredictably in a small number of people

- Pharmacodynamic—interactions with a known pharmacologic effect mediated on receptors (additive, synergistic, antagonistic)

- Pharmacokinetic—involve a change in plasma level or tissue distribution of a drug rather than their pharmacologic activity (e.g. absorption, distribution, metabolism)

Pharmacodynamic drug side effects:

- Additive effects occur when the effects of one drug combine with the effects of another drug working in the same direction. Here, effects of one drug are added to the effects of another drug.

    - e.g. sedation, respiratory depression when sedative agents are combined, combined effects of different drugs which could be anticholinergic

- Antagonistic drug side effects—Here, the effects of one drug run counter to the effects of another drug.

    - For example, in Parkinson's Disease, dopamine agonists are used to treat the symptoms of the disease, but pushing dopamine can lead to psychotic symptoms. Then, antipsychotic meds are given, but these drugs block dopamine—leading to antagonistic drug-drug effects.

Pharmacokinetic side effects:

- Absorption of the medication in the gastrointestinal tract can be

affected by a wide variety of factors.

> • A drug could promote greater contact with and absorption from small intestine which could then lead to greater absorption of the drug, leading to higher concentrations. (e.g. metoclopramide, cisapride accelerate gastric emptying—dumping drugs into the small intestine to be absorbed)
>
> • drugs that diminish intestinal motility—anticholinergics, narcotics—can slow the passage through the gut, leading to increased absorption as well, in an unpredictable fashion
>
> • anything that alters gastric PH (that is—stomach acidity) can affect drug absorption

- Distribution of the drug (many drugs are protein bound. Psychotropic medications are 80%-90% protein bound. In the blood, they are "attached" to protein in the blood. If another drug comes along which can attach themselves to the same sites on the protein, the first drug can be "bumped off" and float free, leading to increased blood levels of the first drug. Often, these effects are minimal since this "freed" drug is absorbed by the liver, and excreted.)

- Metabolism changes can occur. Some drugs can affect the speed the liver enzymes "chew up" or degrade a drug. (e.g. in liver, cytochrome p450 enzyme system can be affected by a wide variety of drugs, and this can affect the half life of drugs for elders)

- Inhibitors of hepatic enzymes include: erythromycin, SSRI's,

many beta blockers, calcium channel blockers, divalproex sodium.

  - These agents ELEVATE blood levels since if the hepatic or liver enzymes are inhibited, they don't act as much and not as much drug is degraded. So, it sticks around longer.

- Inducers (i.e. speed up) hepatic enzymes include: alcohol, tobacco, barbiturates, carbamazepine, phenytoin . These drugs or factors can induce or accelerate the degradation of drugs leading to lower blood levels of the same drug over time.

SUMMARY

All of these factors combine to make giving medications to elders a tricky proposition. In general, these a typical dose of a medication given to younger adults leads elders to have higher blood levels. Elders with less body reserves often cannot tolerate these higher blood levels, and the side effects they engender. Elders also have less muscle mass, and are more likely to fall, and if they do, decreased bone density can lead to an increased risk of fracture.

SO, WITH A MEDICATION IN ELDERS, **START LOW AND GO SLOW**.

CHAPTER 3: EXAMINING DEPRESSION IN ELDERS

PREVALENCE

A debate about the prevalence of major depression in elders has been raging ever since the landmark Epidemiologic Catchment Area Surveys of the early 1980's, which showed a lower prevalence of major depression among elders than younger adults living in the general community. That was not the experience of geriatricians who frequently see depressive symptoms in the elders in their practices. More recent surveys show the prevalence of major depression in elders is similar to other adult groups in the general population, and that depressive symptoms which do not reach the level of a major depression are also common.

For example, a survey of depression in a general primary care practice shows:

- Survey of 224 pts (age 60 or over) in 4 primary care practices
- Major Depression in 6.5%
- Minor Depression in 5.2%

- EtOH abuse in 2.3%[1]

Cost of depression in elders:

The cost of caring for depression in elders is high, both in the cost of treating depression, and in the cost depression adds to the general cost of treating chronic illnesses in elders.

- Even though depression in elders may not occur more commonly, the topic receives a fair amount of attention as economists examine the general cost of medical care.

- Depression is associated with an approximately 50% increase in medical costs of chronic medical illness, even after controlling for severity of physical illness.[2]

And, any chronic illness can lead to major depression.[3]

- Any chronic condition can lead to increased rates of major depression. This includes psychological reactions or disorders such as grief, as well as any chronic "physical" illnesses such as heart disease— indeed any chronic condition

- HOW??

- Idea—chronic stress leads to overproduction of cortisol, which leads to shrinkage of hippocampus in the brain, which leads to vegetative signs, leading to the whole syndrome of major

depression

The following are risk factors can turn sadness into major depression:

- Female Sex
- Chronic medical burden, or disability
- Low social support
- CONSEQUENCES ARE: increased disability, greater healthcare utilization, and increased suicidal ideation

Depression is undertreated:

- Yet, even though depression is common, and very costly, unfortunately, it is not recognized as often as it should be in practice.
- In this survey of 15,762 adults:
    - A bit more than half had treatment
    - 1/3 of depressed individuals had used antidepressant meds, and only 1/3 of them had meds prescribed according to study criteria
    - In terms of cultural differences in treatment: Puerto Rican and Whites had the highest use of meds, and Mexican Americans and Blacks had the least use of antidepressant medications

• Only 21% had guideline concurrent therapy[4]

Any chronic illness may well be a risk factor for major depression, such as heart disease. In these illnesses, depression leads to a higher risk of death.

- After a myocardial infarction, or heart attack: the presence of depression is a significant predictor of mortality (even controlling for left ventricular function (the ability of the heart to pump), and a prior myocardial infarction or heart attack)

- The increased risk of death from depression has an impact at least equal to left ventricular function (the ability of the heart to pump), or a prior myocardial infarction or heart attack

- Even after an 18 month follow up, about a third of patients who had a heart attack were likely to get a major depression. Those patients with heart disease AND depression were more likely to die. The extra deaths were mainly centered in those with heart rhythm problems.[5]

Another chronic medical problem that is a risk for depression in elders: poor vision.

- A British survey of 13,900 people age 75 and older in primary care practices looked for the rates of major depression who had poor vision.

- Visually impaired people had a higher prevalence of depression compared with people with good vision. Of visually impaired older people, 13.5% were depressed compared with 4.6% of people with good vision.[6]

Any brain disorder is linked with a higher rate of major depression. High risk of depression is seen in those with almost any type of brain disorder. The "home" of mood is likely the brain, and when the brain is injured, the mood is injured as well, leading to a higher risk of major depression. Risks for major depression in the following conditions are:

- Any Stroke: 25%
- Left Frontal Stroke: 60%
- Parkinson's Disease: 50%
- Alzheimer's Disease: 25%

So, depression in elders is often a combination of physical illness and social and psychological risk factors.

- For example in a survey of over 400 primary care patients:[7]
    - "combination of risks, including minor or subsyndromal depression, impaired functional status, and history of major or minor depression, did identify a group at very high risk for incident depression"
    - "perceived family criticism had predictive

> characteristics comparable with that of functional disability"

Delirium is also associated with an increased risk of depressive symptoms.

- This study assessed 416 patients at medium or high risk for delirium:[8]

  > • 8.6% developed delirium during the first five days of hospitalization
  >
  > • On the Geriatric Depression Scale, those who developed delirium reported 5.7 depressive symptoms on average, whereas patients without delirium reported an average of 4.2 symptoms

With those suffering from dementia, the presence of depression is a poor prognostic sign.

- In dementia, the presence of depression corresponds to accelerated cognitive decline[9]

- Change in left hippocampal volume in those with dementia is associated with depression, and "Depressed patients with hippocampal volume loss are at greater risk of cognitive decline."[10]

Suicide rates are greatest among men who are elder.

- Among men, suicide rates are greatest among elders, and the

older you are, the higher the suicide rates become

- Among women, suicide rates are highest at mid age, with the "empty nest" thought to be the cause[11]

Why would there be such differences in suicide rates between the sexes? Emile Durkheim, a French sociologist wrote a book entitled "Suicide" (1900) and posited that social differences can help to explain suicide rates.

- He stated that rates of suicide increase the further an individual is separated from his social group. Thus, for men, who in earlier birth cohorts, may traditionally be defined by work or vocation—when that role is lost, suicide is at increased risk.

- For women in earlier birth cohorts, whose role may have been defined by children and family—when that role is lost, suicide is at increased risk.

RESPONSE TO TREATMENT

How do elders respond to treatment for major depression?[12]

- In psychotherapy, elders do as well as younger patients when treated with cognitive behavioral therapy

- And treatment with psychotherapy appears as effective as treatment with antidepressant medications.

But, how well does antidepressant medication work in elders?

- This is a study involving an evaluation or summary of ten treatment trials in treating depression for patients who are over age 60

- In those patients with a short duration of illness (i.e. less than 2 yrs!!) Antidepressant medication showed NO difference compared to placebo

- If patients had a long duration of depression (>10 yrs) antidepressants were more effective than placebo

- More severe depressions (Hamilton Depression Rating Scale >20) showed more efficacy for meds as well

- Age, sex, history of recurrence did NOT predict response of antidepressant medications[13]

In general, depression in elders is hard to treat.

- Survey of those age 85-89[14]

- If depressed at baseline, full recovery occurred at a rate of only 14% per year

- Annual risk of emergence of depression is 6.8%

- Among the oldest old, depression is frequent and highly persistent

And depression in elders can be quite persistent.

- In a British study at 2 year follow up[15]
- The incidence of depression was 8.4%, while depression persisted amongst 61.2% of those depressed at baseline

In these elders, does depression actually kill people?

- Survey of 13,097 British Elders aged 75 and above[16]
- Morbidity, disability, and lifestyle factors can explain most of the observed relationship between symptoms of depression and mortality
- BUT even after controlling for those factors, those who were depressed were 27% more likely to have died by the end of the follow-up period

Not every study shows such poor outcomes.

- In a survey of STAR*D patients (4041 pts) aged 18-75 showed no age group "clearly stood out as distinct from the others"
- "Age of onset was not associated with a difference in treatment response to the initial trial of citalopram."
- But … there were few elders in the study, so it is difficult to draw extensive conclusions about the treatment of elders from this study[17]

- And, another study showed that treatment of depression leads to faster decline of suicidal ideation in the patients treated
- And they had a better course of recovery from depression[18]

In medication treatment of elder depression, what is an appropriate length of a trial of an antidepressant med?

- In one survey of 472 elder depressed patients who were treated for depression for up to 12 weeks[19]
- Most who were partial responders at week 4 were full responders by week 8
- Few non-responders at week 4 were even partial responders at week 12
- So, you can get a good idea of how elders with depression are going to do by 4 weeks of antidepressant treatment. But a complete trial of an antidepressant med is usually thought to be 12 weeks.

Should we pay any special attention to side effects of antidepressants in elders?

- Antidepressant medications have been associated with an increased risk of osteoporosis in elders. Though, there is some debate whether osteoporosis comes from the antidepressant, or a lack of movement from the depression itself.

- In a survey of 2722 older women (mean age 78.5) living in the community
    - Use of SSRIs but not tricyclic antidepressants is associated with an increased rate of bone loss at the hip[20]
- And SSRI's have been shown to have an increased risk of falls, at least in those with dementia
- In a two year study of nursing home residents with dementia—mean age 82:
    - 61.5% had falls
    - Rate of falls tripled if on SSRI's (daily risk increased from 0.09% to 0.28%)[21]

And, still, the most successful form of treatment for depression is electro-convulsive therapy.

- Success rates with ECT in treating major depression in elders is around 60-80%
- And, it is safe. The death rate is less than 1 per 10,000[22]

Transcranial magnetic stimulation:

- Transcranial Magnetic Stimulation (TMS) is being investigated, and so far it is easier to pursue than electroconvulsive therapy since it does not require anesthesia. But, results are

not as good as with electro convulsive therapy. It is helpful in about half of those who receive it, compared to improvement in about 80% of resistant cases treated with electro-convulsive therapy.

- Now approved by the FDA (but usually NOT paid for by insurance)

- In the treatment, there is a placement of magnetic coils over the dorsolateral prefrontal cortex. Enormous electric currents in rapidly alternating on/off patterns producing intense magnetic fields, which move through tissues.

- It is theorized that it increases blood flow and metabolism in targeted areas of the brain—leading to an improvement in depressive symptoms.

- Typically 20 to 30 treatments are given over 4 to 6 weeks

- Each session lasts for about 45 min.

- ECT quicker and more effective

- BUT TMS is safer in that it does not require anesthesia. So, it is being considered for elders. Accordingly, there may also be a role for the treatment in pregnancy, since medications are not given, and general anesthesia is not required.

- Cost: $8,000 to $10,000 for a regimen of treatment

PREVENTION?

Enough about illness, what can we do to advise health promotion in elders—to prevent depression, or to treat depression?

- A study of 601 Australian men in their 80's sought to answer this question[23]
- In the survey, 76.0% enjoyed successful mental health aging
- Successful mental health aging associated with education, and lifestyle behaviors such as vigorous and non-vigorous physical activity

Daily physical activity (≥0.25 mile/day) is significantly associated with lower risk of 8-year incident depressive symptoms in another study of elderly Japanese-American men without chronic disease.[24]

Exercise, repeatedly, has been shown to be successful in decreasing depressive symptoms in elders.

- In a controlled study of sedentary elders—age 60-75
- They entered into a "fitness endurance activity"
- There was a significant decrease in depressive and anxiety scores and an improvement in the quality of life in the experimental group[25]

- And, exercise was effective in reducing depression symptoms in 45%-65% of elder pts compared to 25%-30% of controls in another study[26]

Is exercise always helpful for depression? The following study examined the use of exercise for depression in physically ill elders who were living in a nursing home:

- Study—exercise done in one British NH compared to a nursing home without an exercise program[27]

- The program—consisting of 45 min sessions twice a week—did not help in this frail population. So, exercise as a treatment for depression may need to be focused in those who actually can pursue aerobic exercise.

SUMMARY

Major depression is about as common in elders as in younger age groups. Chronic illness is a risk factor for the development of depression, and depression usually portends a poorer prognosis for those physical illnesses. Psychotherapy is as effective as it is in younger people. Medications carry side effects, but medication treatment is more helpful than placebo in chronic depressions in elders, and in more severe depressions. Exercise consistently has been shown to be helpful as treatment for depressive symptoms in elders. ECT or electroconvulsive therapy continues to be the gold standard for

treatment of depression, and transcranial magnetic stimulation is also helpful for resistant depression in elders.

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CHAPTER 4: ALZHEIMER'S DEMENTIA

PART 1—OVERVIEW OF THE ILLNESS

Dementia is a decline of cognitive abilities, sufficient enough to interfere with daily functioning. Simply, dementia is brain failure, and while there are multiple types of dementia, the most common type is Alzheimer's Disease. This disease presents with a very gradual decline of cognitive function over many years. Generally, from the time of first onset of symptoms to death is typically 7-10 years. It is generally accepted that the onset of the disease is many years before the onset of first symptoms. However, an easy way to determine those at risk, or those who will develop the disease is still under intense study. More recently, as care for those with Alzheimer's Disease has improved, the lifespan of those with the disease has lengthened.

The disease, like so many other components of brain function, remains shrouded in mystery. The cause, and hence the treatment for it remain unknown, hidden in the weeds of the complexity of human brain function. While many ideas about the disease and treatment for it exist, effective treatment continues to elude us.

Timeline Of Alzheimer's Disease Research

Examining the timeline of exploration of the disease over the last century is helpful to show just how little we know about Dementia, and

Alzheimer's Disease. Since the original description of the disease and the pathology of the disease in 1906, remarkably little progress has been made.

- 1906: Dr. Alzheimer describes the disease, and pathology in a 51 yr old female patient. The woman presented with "early onset", and thus was an interesting case. Dr. Alzheimer, being a careful researcher, did a pathologic examination of her brain, and described the hallmarks of the pathology of the disease in the brain: Plaques (which we now know are accumulation of Beta Amyloid protein) and neurofibrillary tangles (tangled pieces of dead neurons, which contain the protein tau).

- 1960's: syndrome of "Alzheimer's Disease" recognized

- 1970's: it was felt that there was no difference between AD and "senile dementia"

- 1980: Alzheimer's Association established

- 1978-1983: Cholinergic Deficit in AD identified (i.e. those who suffered with Alzheimer's dementia had deficits of acetylcholine in their brains, and this led to the first effective medication treatments, which attempted to boost acetylcholine. However, since dementia is total brain failure, there are deficits in many chemical or neurotransmitter systems, not just this one. Hence, the treatment has limited efficacy.)

- 1993: First acetylcholinesterase inhibitor approved

- 2004: First NMDA receptor antagonist approved (this is memantine, which is approved for use in severe or advanced Alzheimer's Disease)

- 20??: Next approved treatment for Alzheimer's Disease

- 20??: Cure or Prevention for Alzheimer's Disease

Public Health Impact Of Alzheimer's Disease

The prevalence roughly doubles every 5 year period over age 65 in the United States. That is, about 3% of those age 65 have Alzheimer's Disease. At age 70, the prevalence is about 6%. At age 75, it increases to roughly 12%, and at age 80 about one fourth of the US population suffers from the disease. Following this, of course, you cannot double forever, but it is thought that roughly half of those who live to ninety or above will get the disease.

The rise of the large number of aging baby boomers presents a looming public health disaster.

- The fastest growing segment of population are elders

- Number of elders over age 65 will double by 2050

- 5.1 million Americans current suffer from AD

- Current cost of AD care: more than $148 billion/year

- The future will bring larger numbers of elders, with larger numbers suffering from the disease, taxing our health care system, unless more effective treatments are found[28]

- More than half receive care at home

But, are rates of Alzheimer's Disease and dementia actually declining these days?

Two recent studies, one in Great Britain, and the other in Denmark show that the expected rates of Alzheimer's Disease and dementia may not be as high as was expected. The rates in these two studies are slightly lower than expected, but any change in a large number of the population can have significant impact on the public health, and in public health spending.

- Comparing Danish birth cohorts—those born in 1905 to those born in 1915:

- Those born later (in 1915) are living longer

- Generally, both groups of elders are physically as frail BUT…

    - The younger birth cohort is cognitively more intact (MMSE=22.8 vs. 21.4, p=0.0003)

    - Activities of daily living are also more intact in the younger birth cohort as well[29]

- And…a British study evaluating 7635 people, over age 65—rate

of dementia was 6.5% of that population (was predicted to be 8.3% based on prior estimates, studies)

- So, "Later-born populations have a lower risk of prevalent dementia than those born earlier in the past century."[30]

Looking at Alzheimer's Disease now:

- It is important to remember that the disease is gradually progressive over years (implies progressive for YEARS before onset of identifiable symptoms)

- Research to identify those at risk/or early identification of those with the disease (through the use of imaging, neurochemical studies is underway)

- It is important to remember that Alzheimer's Disease is a process which unfolds over years, not an event

- Medication therapy now involves either or both of cholinesterase inhibitor, and/or memantine. These medications at best slow the progression of the disease. They do not reverse the disease, or cure it.

- So, focus is CARE, not cure

- Role for cognitive exercises?? Some suggest that keeping the mind active will help to slow the progression or onset of the disease. While it is true that early in life more educational opportunities help to prevent the disease, it is not clear if "exercising the brain" late in life will have the same effect.

Geneses Of Alzheimer's Disease

Research theories concerning the genesis of the disease:

- Acetylcholine hypothesis: This is the idea that the disease is caused by a deficiency of acetylcholine in the brain. We knew that medications that block acetylcholine lead to poor memory as a side effect. Also, it was discovered that acetylcholine was low in those with the disease (but later on it was discovered that MANY neurotransmitter systems were also affected in Alzheimer's Disease). The hope was, if we can increase acetylcholine, we can add memory and function to those who suffer from Alzheimer's. This led to the cholinesterase inhibitors. The most popular is donepezil (or Aricept).

- Amyloid hypothesis: Dr. Alzheimer described that the patient he saw with the disease, and he described the hallmark feature of protein plaques in the brain. It was later discovered that these plaques were accumulation of beta amyloid protein. Efforts to block accumulation of this compound, prevent its synthesis, "vaccinate" the brain to recognize it better to discard it, or to recognize it and have the body's immune system get rid of it have all proven unsuccessful.

- What is heart healthy is brain healthy: This is a newer idea. Consistently, it has been found that those who have a variety of cardiac risk factors (diabetes, high cholesterol, etc.) are at higher risk for the disease and conversely, those who have lower cardiac risk factors, or care for the risk factors they

have, are at lower risk. This idea holds that blood vessels in the brain can also be affected in the same way as heart blood vessels, and injury to these brain vessels can lead to injury to the brain. Then a cascade of events occurs in the injured brain tissue which leads to the gradual decline that is Alzheimer's Disease.

Is what is heart healthy, brain healthy?

- DM, elevated cholesterol, smoking put one more at risk for Alzheimer's Disease (does treating this decrease risk???—not clear)

- Psychological factors such as depression, never married, low social support (loss of a spouse) may be risk factors

- Omega 3 fatty acids may help prevent AD but diets, vitamins not useful

- APO E 4 a risk (Apolipoproteins are involved in carrying cholesterol, and one subtype is APO E 4. If you have two of this type of inherited proteins—i.e. one type inherited from your father's side, and one from your mother's side—you are more at risk to get Alzheimer's Disease.)[31]

- If you have a history of diabetes, you are twice as likely to develop dementia (AD as well as any cause dementia including vascular dementia)[32]

- And exercise can help to lower your risk—"A higher level of total daily physical activity is associated with a

reduced risk of AD." (p=0.007)[33]

- CETP V405 valine homozygosity is associated with slower memory decline and lower incident dementia and AD risk—boosts levels of HDL, the "good" cholesterol (about 70 percent less likely to develop Alzheimer's disease)[34]
    - Higher levels of HDL lead to lower risk of AD[35]
- those taking angiotensin receptor blockers (high blood pressure medications) were up to 24 percent less likely to develop dementia than patients on other medications AND patients already diagnosed with Alzheimer's disease were half as likely to be admitted to a nursing home and had a 17 percent reduced risk of dying if they were taking these medications.[36]
- Statins (medications which lower cholesterol): associated with a 79% decreased risk in retrospective studies (what's heart healthy is brain healthy)
- Why? It is not known, but the thought is that by taking care of your brain blood vessels, you may decrease injury to the brain, leading to:
    - Decrease plaque formation
    - Anti inflammatory effect
    - Prevent strokes, brain injury
- BUT, so far, statins don't look to be helpful in prospective

treatment for Alzheimer's Disease

Going along with the idea that what is heart healthy is brain healthy is the concept that exercise can be helpful to prevent or even treat symptoms of Alzheimer's Disease.

- Exercise reduces atrophy and white matter lesion load in the Lothian Birth Cohort 1936 (n = 691) which is a longitudinal study of aging of people in their 70's

- The physical activity associations with atrophy, gray matter, and WML remained significant after adjustment for covariates, including age, social class, and health status[37]

Overall evaluation of the idea of "what's heart healthy is brain healthy":

- Idea—up to half the cases of AD could be prevented with attention to 7 risk factors: diabetes, midlife hypertension, midlife obesity, smoking, depression, cognitive inactivity or low educational attainment, and physical inactivity[38]

    - BUT none of these factors is "proven to have a causal relation" to Alzheimer's Disease[39]

    - AND—a 2010 NIH panel—no evidence of any modifiable factor to reduce risk for Alzheimer's Disease!![40]

Exercise in nursing home residents can help maintain function.

- This is a study of 134 ambulatory French nursing home residents[41]

- They suffered from mild to moderate Alzheimer's Disease

- Assigned to exercise—1 hr twice/week, with walking ½ of the session

- Placebo—treatment as usual

- At 12 mo.—activities of daily living ratings declined less in exercise group compared to the placebo group

Could there be psychological risk factors for Alzheimer's Disease as well?

Depression has been postulated to be a risk factor. But, brain injury is a risk factor, and brain injury can lead to depression.

But, what about loss of a parent in childhood?

- Cache Co., Utah study of 570 demented elders compared to 3538 without dementia[42]

- Mother's death during adolescence and, to a lesser extent, father's death before age 5 a risk for Alzheimer's Disease (but not non-Alzheimer's Disease dementia)

- If widowed parent remarried—no effect

- Controlled for age, gender, education, APOE genotype, and socioeconomic status

Head injury is also a risk for Alzheimer's Disease.

- "Dementia pugilistica" or dementia in boxers has been observed
- Review of observational studies in brain injury in boxing: 10-20% of professional boxers have measurable long term brain injury[43]
- Another study followed up war veterans with head injury:[44]
    - 40 year follow up study of 548 WWII Navy and Marine vets
    - Mild head injury—inconclusive link
    - "moderate head injury (hazard ratio [HR] = 2.32; CI = 1.04 to 5.17) and severe head injury (HR = 4.51; CI = 1.77 to 11.47) were associated with increased risk of Alzheimer's Disease"

Clinically, what is seen in dementia?

- Usually you see in the patient a combination of etiologies of dementia leading to decline. That is, it is not uncommon for a patient to have both Alzheimer's Disease and stroke, or Alzheimer's Disease and Lewy Body Dementia
- Alzheimer's Disease—most common cause of dementia, followed by stroke (vascular dementia), and Lewy Body dementia

What is the pathologic hallmark of Alzheimer's Disease in brain histologic studies?

- Neurofibrillary tangles: intracellular in neurons; Tau protein is part of the microtubular structure of cytoskeleton of neurons, which becomes hyperphosphorylated in Alzheimer's Disease which leads to paired helical filaments which leads to neurofibrillary tangles

- Plaques: amyloid precursor protein is cut in different place in Alzheimer's Disease. In Alzheimer's Disease, the protein is cut into "alpha" and "beta" fragments. The beta fragment, or beta amyloid misfolds and the brain is not able to get rid of it effectively, and it accumulates in plaques or sections of protein "gunk" in the brain.

Genetics in Alzheimer's Disease:

- Small numbers (<1%) of those with Alzheimer's Disease have mutations on chromosome 21, 14 or 2—These patients typically have an early onset, and these very uncommon causes of the disease probably have an autosomal dominant type of inheritance (that is, about half of those with the gene will get the disease.)

- If you don't have that rare type of the disease, if you have a first degree relative with Alzheimer's Disease, your risk of getting the disease is double to triple that of the general population

- Apolipoprotein E-4 is associated with increased risk. Typically,

testing for this is not done routinely, and this continues to be mainly a focus of research at this time.

But, genetics also offers hope for another avenue of treatment.

Those who are genetically at high risk for the disease may be able to offer a source of information about the disease itself. In other words, families who get the disease more, or who have an increased genetic risk may be able to provide some answers about the origin of the disease, or its pathology once the disease gets started.

One such group of a family at high risk is in Columbia, in South America. They have a mutation in Presenilin 1 which leads to an accumulation of beta amyloid earlier in life. Studies are now being conducted in mutation carriers in Columbia from age 18-26. So far, these studies[45] show:

- No difference in neuropsych test or dementia rating scores, or Apo E4 presence
- Less gray matter in some right parietal regions of the brain
- Greater right hippocampal and parahippocampal activation in that part of the brain as seen on advanced brain imaging studies
- Higher Cerebral spinal fluid and plasma beta amyloid concentrations

Can MRI scans detect Alzheimer's Disease earlier than symptoms appear?

- Much research work is being devoted to this question now.
- Those who will develop Alzheimer's Disease seem to have cortical thinning in some parts of the brain such as:[46]
    - medial temporal lobe-temporal pole
    - superior frontal gyrus
- And, other studies have shown decreased hippocampal volume before development of Alzheimer's Disease. But the hippocampus shows smaller volumes in many types of psychiatric disorder.

So, if amyloid is found in the plaques seen in the brain, can we identify that early on, and would that help identify those at risk?

- "Trajectories of Aβ42 (beta amyloid) level in CSF, FDG uptake, and hippocampal volume vary across different cognitive stages. The longitudinal patterns support a hypothetical sequence of AD pathology in which amyloid deposition is an early event before hypometabolism or hippocampal atrophy, suggesting that biomarker prediction for cognitive change is stage dependent." That is, identifying markers for who may have the disease may change as the progression of the disease goes on, and the time that one has the disease progresses as well.[47]

So, much attention is being directed at labeling beta amyloid and identifying it as a means of focusing attention to those at risk for the disease.

PART 2—MEDICATION TREATMENT OF ALZHEIMER'S DISEASE

Obviously, the research about the course and clinical path of Alzheimer's Disease described in the first part of this chapter is important only if it would lead to proper treatment or prevention of the disease. While we know a great deal more about Alzheimer's Disease now compared to several decades ago, we still have a long way to go.

Currently, there are only two types of medication treatment available for the treatment of Alzheimer's Disease, and they are of limited efficacy. The available medications usually do not stop the disease, or reverse it. Rather, they slow the progression of the disease as time goes by.

Medication Treatment For Alzheimer's Disease

- FDA approved drugs: cholinesterase inhibitors (donepezil is the drug most commonly used), and memantine
- All work better at higher doses—so advance the dose gradually
- Gastrointestinal upset is the main side effect for all of them
- No head to head comparison on which one works best, but in general, the cholinesterase inhibitors are felt to be the most effective, and most people feel that the different types of this class are equally effective

- The cholinesterase inhibitors may delay progression by 1 year (?) (There is little data to guide us on how well the cholinesterase inhibitors work. The most optimistic among us feel that their use delays the progression of the disease by about a year. That is, instead of dying about 7 years after the onset of illness, you may die about 8 years out. Delaying the progression of a severe disease in elders is important—and can impact the burden of illness on the patient, but also in the medical care system as a whole.)

Cholinesterase inhibitors approved for use in Alzheimer's Disease

- Tacrine (cognex) associated with liver injury 25 % of the time, gastrointestinal upset in 25% (so seldom used)

- Donepezil (Aricept) much better tolerated, start at 5 mg, move to 10 mg hs at 4-6 weeks (most popular of this group)

- Rivastigmine (exelon) give with food, inhibits acetylcholinesterase, and butyrylcholinesterase (though it is not clear this gives any added benefit), dose is 1.5 mg bid to 6 bid, skin patch is available—which avoids stomach absorption, limiting stomach side effects

- Galantamine (razodyne)—well tolerated; now has ER or daily formulation available

Efficacy of the cholinesterase inhibitors:

- The cholinesterase inhibitors, we believe, work by slowing the

destruction of acetylcholine. In Alzheimer's Disease, there is less acetylcholine present. Less destruction of what is there, we think, leads to less memory loss.

- The medication does not stop the progression of the disease. It slows the progression.

- This can be difficult to gauge the success of treatment in any one patient. Since you don't know where the patient would have been without the med, it is difficult to gauge "progression" of the illness after the fact.

- However, in some patients, (probably about 5% to 10%) actually have more dramatic effects, where the disease actually does improve or stop progressing for a while with the use of these medications.

- Individual variability exists in us all—including the response to these medications. Response is on a "normal curve", with some individuals "at the other end of the curve" doing much better.

- Side effects are primarily stomach upset and diarrhea. However, muscle cramps and muscle weakness can occur as well. More severe reactions such as fainting are much less common.

- Side effects can be minimized by starting at a low dose (5 mg at night) and then increasing to 10 mg at night to give the body a chance to get used to the medication.

- Also, the effects of 10 mg are only slightly better than 5 mg. So, if cost is an issue, prescribe the 10 mg pill, and have the patient cut it in half.

Memantine—how does it work?

It is approved for use in moderate to severe dementia, and managed care agencies will often not approve its use for those patients less severely affected.

- It is a moderate to low affinity NMDA receptor antagonist. One theory is that affecting the NMDA receptor will interfere with brain "overexcitation" which can lead to brain injury or damage.

- (High affinity NMDA receptor antagonists didn't work in clinical trials—so the exact mechanism of action is not clear.)

- Studies have shown neuroprotection of neurons exposed to glutamate in-vitro. Glutamate is the main excitatory neurotransmitter in the brain, and blocking this, may block over excitation of the brain, leading to decreased breakdown.

- Another idea is that the medication may protect cholinergic cells in acute and chronic animal models

- Max serum concentration in 6 to 8 hrs

- Half life between 75 and 100 hours

- But, usually given twice a day
- Start 5 mg per day, increase by 5 mg per week to 10 bid
- The clinical folklore is: while cholinesterase inhibitors may be helpful for memory, memantine may well be more helpful in preserving activities of daily living.
- Memantine use led to less caregiver time
- And… longer time to institutionalization
- Some data shows that patients on this medication are more likely to remain autonomous[48]
- Side effects of memantine include agitation—and this can be difficult to determine in a disease that often has agitation as a side effect

Should memantine be routinely added in severe dementia?

- A recent study questions the wisdom of adding memantine in those with severe dementia[49]
- British study of 295 elders, SMMSE=5-13
- Community dwelling, one year follow up
- Continuing donepezil decreased decline by 1.9 pts (that is, donepezil was helpful in severe or advanced dementia in slowing decline)

- Adding memantine led to NO significant difference

- Donepezil had 23% less decline than placebo (but patients still continued to decline)

Are there other medications which may help?

- Vitamin E at a dose of 1000 bid slowed decline by about 9 months over a 2 year study—But more recent data does not show a clear help. And chronic use of Vitamin E over years leads to a slightly higher death rate (of about 5% increased deaths over a forty year period).

- Vitamin E offers no help in the change in progression to Alzheimer's Disease from mild cognitive impairment compared to placebo[50]

- Seligiline (a selective MAO inhibitor)—helpful (slowed decline by 5 mo.), but provides no added benefit when combined with Vitamin E.

- But, seligiline has an array of side effects—and can certainly cause dangerous drug-drug interactions—so it is not routinely used. Because MAO (monoamine oxidase) levels increase as one ages, the hope was that inhibiting this would decrease the cognitive effects in this disease associated with aging. But, it is thought that the risk of side effects outweighs any potential benefits of the drug.[51]

These compounds don't work in Alzheimer's Disease:

- Ginko Biloba not helpful[52]
- Low dose steroids (prednisone 10 mg)
- Estrogen replacement therapy
- NSAIDS (rofecoxib (cox-2 inhibitor) and naproxen (non-selective NSAID)
- The "vaccine". This was an attempt to treat Alzheimer's Disease by injecting beta amyloid into those already suffering from the disease. Here's the theory: Usually the amyloid protein is cut, and the pieces can be discarded by the body. However, in Alzheimer's Disease, amyloid protein is "cut" in a different place, leaving a beta amyloid fragment which the brain does not recognize, and it accumulates. As the theory holds, this accumulation of beta amyloid would cause problems in the brain, leading to Alzheimer's Disease. In animal studies, injecting beta amyloid in rats was associated with a decrease in amyloid plaque burden, with the thinking that injection of the beta amyloid would lead the brain to "recognize" it, and get rid of it easier—hence treating the disease. However, when beta amyloid, along with substances to increase the immune reaction, was injected into people, about 6% developed encephalitis, and the trial had to be stopped.
- "Fish oils" did not help stop the progression of Alzheimer's Disease either[53]

Other ideas for treatment:

- Could there be cellular implants delivering nerve growth factor? Nerve growth factor could serve as a "fertilizer" for the neurons in place, leading to growth and "healing" of the neurons in the brain.

- Stem cell transplant. Stem cells are cells which can differentiate into different cells needed in the body. Could a "transplant" of these cells lead to effective treatment for the areas of the brain affected by Alzheimer's Disease?

Medication Treatment For Behavioral Problems In Alzheimer's Disease

- There is no FDA-approved treatment for agitation associated with dementia

- This is a changing paradigm

- Double blind studies have failed to show a difference for antipsychotics, anticonvulsants, donepezil in the treatment of agitation in dementia

    - and memantine can make agitation worse)

- But, individual patients can be helped by individual drugs

- So, to use these medications, identify target symptoms

- Identify symptom cluster similar to well established drug-responsive syndrome

    - For example, if the patient is not sleeping, consider

trazodone; if the patient seems to be paranoid, consider an antipsychotic medication, etc.)

- Try behavioral interventions FIRST.

  - If the patient is agitated in a certain period of the day, focus care activities in the other part of the day. As always with agitation, if the patient is agitated, back off, and try again later.

- If this does not work—identify a target symptom, and try a medication that seems best suited for this particular symptom.

- Monitor for both effects of the medication and side effects.

- If things seem better—continue for a time. If things seem no better—discontinue that med, and try another.

Psychosis and agitation are common in Alzheimer's Disease, especially in moderate to severe dementia.[54]

- Psychotic symptoms in 30-50%

- Agitation in many—up to 70%

  - But, treatment with antipsychotics does not separate from placebo in discontinuation rates[55]

  - Meta-analysis shows a small statistical treatment effect for aripiprazole and risperidone in treatment trials[56]

- All antipsychotic meds are associated with an increased risk of death compared to placebo (the increased risk is about 2% over a six month period). This is true for haloperidol as well as the newer antipsychotic medications. So, before prescribing these meds, discuss this small increased risk of death with families. Most families elect to try treatment for the agitation or psychosis given the profound effect this symptom has on the patient and family as well as the unrelenting progressive nature of the disorder.

- Antipsychotics are also associated with an increased risk of falls in elders.[57]

Antipsychotics may also decrease cognition.[58]

- Study of 421 patients with AD with psychosis or agitation, treated with olanzepine, quetiapine or risperidone vs. placebo

- Those on drug had slightly more cognitive decline than placebo

So then, what should we think about using antipsychotic meds in those with Alzheimer's Disease?

- Increased risk of death is 1-2%

- Typical agents NOT safer

- Increased risk of stroke in the first few months (may return to normal after 3 months)

- Increased fall risk
- Increased cognitive decline
- Beware of prolonged QTc (lengthening of the heart beat), weight gain (Diabetes not a worry in nursing home pts.)
- Increased risk of pneumonia
- BUT—there is no FDA approved med for agitation; SO—use behavioral methods first, then … consider their use after talking with the family[59]

But patients who have improved on antipsychotics get worse when these meds are removed.

- Pts who responded to risperidone for aggression or agitation OR psychotic symptoms had higher rates of relapse than placebo (p=0.004)
- Rates of death and adverse events did not differ from placebo—but it was a small group[60]

What about using other meds—anticonvulsants?

- Some case reports and case series show some benefit
- BUT in controlled trials—NOT helpful
- Side effects: sedation, problems with gait, drug interactions

- May be worth a try in some patients

What about using antidepressants in patients with Alzheimer's Disease who are depressed?

- Sertraline and mirtazepine did not separate from placebo in depressive symptoms
- Drugs had more side effects than placebo[61]

What psychological approaches may be helpful in managing agitation in patients with Alzheimer's Disease?

- Helpful: cognitive stimulation therapy, behavior management techniques, caregiver education, residential care staff education, music therapy, sensory stimulation[62]
- Exercise may also be helpful (what is heart healthy is brain healthy)

But, there is little data that these approaches are helpful:

- reminiscence therapy, validation therapy, reality orientation therapy, simulated presence therapy, therapeutic activities, enforced social interaction, decreased sensory stimulation, mirrors, signposting, group living[63]

Present State Of Alzheimer's Disease Care

So, what is the present state of Alzheimer's Disease care?

- Diagnosis is reliable
- Treatment is symptomatic, palliative
- Treatment delays progression by about one year (at most??)
- Nursing Home stays at end of life cost about $85,000 per year
- When patients "die of Alzheimer's Disease", they typically die of pneumonia. As the brain fails, so does the body. Patients begin to lose the automatic ability to swallow, leading to aspiration of food or saliva into the lungs, leading to pneumonia.
- Insertion of feeding tubes into the stomach does not prolong life.

How to care for patients with Alzheimer's Disease at the end of life?[64]

- Meet with families and plan ahead
- Advanced directive should be what the ill person would have wanted
- Support family's decision
- Feeding tubes not helpful
- Role for hospice care

What about the future?

- Early identification of those at risk for the disease
- More effective treatments, and hopefully cure…
- Growth of home care to care for those at home "as long as possible"[65]
- …Prevention of the disease

SUMMARY

So, while the knowledge of the pathology and disease course of Alzheimer's Disease has improved dramatically over the last few decades, this knowledge has not yet led to effective treatments which would stop or prevent the disease.

There is not yet an effective management strategy to prevent the disease, but my best guess at this time is "what's heart healthy is brain healthy". That is, taking care of vascular risk factors such as high blood pressure, high cholesterol, being overweight, and exercising regularly may help. Also, keeping mentally active as well, in activities you enjoy may help.

Medication treatment of Alzheimer's Disease at this time involves treatment with cholinesterase inhibitors and memantine. Agitation and

depression symptoms are common in those who suffer from the disease. So far, no medication has been proven to be effective. Behavioral interventions are tried first to manage these symptoms. If these fail, symptomatic medication treatments are begun and carefully monitored with the hope that the symptoms will improve. If they do not, or if side effects are a problem, the drug should be removed, and see if another drug trial may be helpful.

We hope the future will bring more effective treatments.

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CHAPTER 5: NON-ALZHEIMER'S DEMENTIA

The most common forms of Non-Alzheimer's Dementia (that is types of dementia or global brain failure that are not caused by Alzheimer's Disease) are Vascular Dementia (that is dementia from stroke) and Lewy Body Dementia. In this chapter, we will review these forms of dementia, as well as other, less common syndromes. Treatment for vascular, or stroke related dementia is the use of aspirin, which serves to help to prevent further strokes. With other types of dementia, cholinesterase inhibitors may be tried, but it is not always clear how well they will work.

VASCULAR DEMENTIA

- 20% of all dementias
- Average age of onset—age 65
- Course—often sudden onset, stepwise deterioration (that is, someone will be stable until the stroke hits, and then will have a sudden decline, stabilize until the next stroke, and then have a sudden decline—giving a "step wise decline" in the clinical course)
- Risk factors: hypertension, smoking, vascular disease, diabetes[66]

- Imprecise relationship between volume of infarcted material (that is the amount of the brain affected by the stroke) and cognitive and psychiatric impairment (volume of the area of the brain affected by a stroke accounts for only about 60% of the variance)

- So, both volume AND location of stroke in the brain are important in determining the outcome for the patient

- 25% to 35% of late onset dementia have BOTH Alzheimer's Dementia and Vascular dementia on post-mortem exam—both are common disorders, and so it is not uncommon to have them both occur together[67]

NINDS-AIREN Diagnostic Criteria for Probable Vascular Dementia:[68]

- Dementia

- Cerebrovascular Disease

    - Focal neurological signs on physical exam (such as weakness or lack of sensation on one side of the body)

    - Neuroimaging evidence of EXTENSIVE vascular lesions (not "small vessel disease of aging")

- Relationship between stroke and dementia

    - onset of dementia within 3 months of recognized stroke, abrupt deterioration or stepwise progression

Other terms used clinically for vascular dementia:

- Multi-infarct dementia (implies many strokes)
- Binswanger's Disease (MANY small vessel ischemic changes leading to brain injury)
- Borderzone syndrome—"watershed" injury between blood supply areas, from periods of hypotension, brain ischemia

BUT…these descriptions are NOT vascular dementia:

- One Lacunar infarct (i.e. small lesion—lunar or fingernail shape of a small stroke on a report of a CT or MRI scan)
- "microvascular white matter changes of aging" description on a CT or MRI scan. This means chronic, small changes —not enough to lead to a dementia syndrome. This can be seen in normal aging.

Hachinski Ischemic Score:

This is an old, but tried and true method. This is a clinical scale helping to determine if a syndrome is a "vascular dementia syndrome."[69]

Symptom	**Points for the symptom**
Abrupt onset	2
Stepwise deterioration	1
Fluctuating course	2

Nocturnal confusion	1
Preservation of personality	1
Depression	1
Somatic complaints	1
Emotional incontinence	1
History of hypertension	1
History of stroke	2
Associated atherosclerosis	1
Focal neurologic symptoms	2
Focal neurologic signs	2

Score > or = 7—Vascular Dementia

Score < or = 4—Not Vascular Dementia

Treatment of vascular dementia:

- Prevention of further strokes—aspirin, dipyridamole, ticlopidine

- Treat psychiatric symptoms that may accompany a stroke
    - SSRI's for depression
    - Antipsychotics for psychotic symptoms
    - Agitation—use behavioral interventions first. Then consider antipsychotics, or anticonvulsants. There is no FDA approved drug for agitation.

Do the cholinesterase inhibitors help with memory in vascular dementia?

- Galantamine effective with cognitive symptoms[70]
- BUT—donepezil linked with a slightly increased risk of death in vascular dementia—pooling 3 studies, death rate is 1.7% with donepezil, 1.1% with placebo[71]

Those with Alzheimer's Disease frequently have strokes.

Both Alzheimer's Disease and stroke are common diseases in elders, so it makes sense that they could co-occur. But, some wonder because they occur so commonly together if a stroke could pre-dispose to Alzheimer's Disease (by causing a brain injury, and then a reaction that in some way could kick off the Alzheimer's Disease process.)

- 60-90% of Alzheimer's Disease cases exhibit variable cerebrovascular pathology

- 1/3 of patients with Alzheimer's disease have infarcts

- co-existence of stroke and Alzheimer's Disease occurs more than by chance alone

- These authors in the study cited[72] "propose that cerebral ischemia or oligemia may promote Alzheimer type of changes in the aging brain"

LEWY BODY DEMENTIA

- Lewy Body Dementia typically presents with the clinical triad of dementia: often prominent visual hallucinations and parkinsonian symptoms (bradykinesia (slow movements), muscle rigidity, and muscle tremor)

    - (So, avoid the "typical antipsychotics" i.e. haloperidol in those with Lewy Body Dementia)

- 20% to 30% of patients with dementia on autopsy have Lewy Body Dementia

- The disease is slowly progressive, over 6-10 years[73]

How does Lewy Body Dementia compare to Parkinson's Disease?

Lewy Body Dementia typically presents with:

- Dementia

- Attention problems
- Visual spatial problems and prominent visual hallucinations

Whereas, Parkinson's Disease typically presents with:

- Movement problems
- Alert mental status
- Visual hallucinations can occur, but are not as common

In Lewy Body Dementia:

- Cholinergic deficits are present
- Increased numbers of Lewy Bodies in temporal lobe and amygdala are associated with visual hallucinations
- Reduced brain perfusion is present in the visual cortex (could this be the reason why visual hallucinations are so common?)[74]

Treatment of Lewy Body Dementia:

- Treat Parkinsonian symptoms with dopaminergic agents
- Treat psychiatric symptoms (75% have hallucinations, >50% have delusions)[75]

- Treat hallucinations with low potency atypicals (?)
- Treat depression with SSRI's
- Treat cognitive symptoms with cholinesterase inhibitors (case series support demonstrate use, but double blind trials are lacking)[76]

OTHER TYPES OF DEMENTIA

Parkinson's Disease

- Motor system disease
- Presents with tremor, stiffness, slowed movement, impaired balance or coordination
- Onset typically after age 50
- Course—typically gradual onset, slowly progressive (can vary)
- Affects 2% of US population over age 70[77]

Dementia in those with Parkinson's Disease:[78]

- Present in 40% of those with Parkinson's Disease
- Another 40% may have mild impairment
- More common if you present with akinesia (or slow movements)

- More common in elderly, or in chronic disease

Psychiatric symptoms in Parkinson's Disease:[79]

- Very common
- 1/3 have depression
- Psychotic symptoms are uncommon in untreated patients but in treated patients 15-40% have hallucinations. Therefore, it is more likely that psychotic symptoms come from the MEDICATION TREATMENT of the disease (which pushes dopamine) than the disorder itself.[80]
- More severe the disease, the more common and more severe these psychiatric symptoms
- Anxiety symptoms in 1/3
- Sleep problems in ½

Treatment of Parkinson's Disease can lead to problems…[81]

- Dopaminergic agents are the treatment…BUT
- Pushing dopamine can lead to
    - hallucinations in 1/3 (especially visual)
    - delusions in 6%-10%

- hypomania in 2%
- hypersexuality
- agitation

Treatment of Parkinson's Dementia:

- Treat the disorder
  - carbidopa, selegiline, amantadine, bromocriptine, etc.
- Treat the mood disorder
  - SSRI's first choice (stop selegiline if the patient is on it since the combination can lead to a dangerous reaction)
  - BUT, consider selegiline as back up treatment. ECT is helpful for both mood and movement disorder
- Treat the psychotic symptoms. Use the "low potency" atypical antipsychotics such as quetiapine, olanzepine, clozapine. These have a lower risk of parkinsonian or muscle side effects
- REMEMBER that side effects of some psychotropic drugs (tremor, rigidity) may worsen parkinsonian symptoms—continue to monitor

Treatment for cognitive problems in Parkinson's Dementia:

- Rivastigmine (exelon) has been approved by the FDA for Dementia associated with Parkinson's Disease[82]

- Other cholinesterase inhibitors may also be equally helpful

Fronto-Temporal Dementia

- Typically presents with behavioral disinhibition, coarsened interpersonal interactions, impulsivity, poor judgment. Typically, you will see misbehavior before clear cognitive impairment

- Onset in mid to early 50's

- 40% have positive family history in 1st degree relative

- Gradually progressive to death in about 10 years[83]

- Compared to Alzheimer's Disease, a patient with Fronto-Temporal dementia is LESS LIKELY to have

    - Psychosis

    - Depression

To help make the diagnosis of Fronto-Temporal dementia:

- Brain Imaging:

    - CT, MRI may show frontal atrophy (with more language problems may see more left sided atrophy)

- SPECT or PET scans can be helpful—and show decreased frontal perfusion (that is, less blood flow in the frontal lobes)

- Neuropsych testing—shows frontal lobe dysfunction

Treatment of Fronto-Temporal dementia:

- Try symptomatic treatment for psychiatric syndromes

- Is cognitive impairment treatable??

  - It is not clear that the usual cognitive treatments—the cholinesterase inhibitors—are helpful in the treatment of cognitive impairment in Fronto-temporal Dementia

Fronto-Temporal dementia is a dementia syndrome that focuses on the frontal and temporal lobes, but…

- You can also see dementia syndromes that affect other focal areas of the brain. For example, left sided brain syndromes will show language disturbances; and right sided brain syndromes can show behavioral disturbances. Other focal disturbances can also occur—for example showing only left or right frontal disturbances

Creutzfeldt-Jakob Disease

- Rare, progressive, fatal disorder

- Usually begins around age 60
- Course—90% die within one year, but can be more prolonged (several years)
- Caused by a type of protein called "prion"
- Can get it either from genetics (genetic loci is the short arm of chromosome 20), or infectious sources ("Mad cow disease") Bovine Spongiform Encephalopathy (BSE)[84]
- Usually ataxia, or depression or anxiety is seen clinically first
- Myoclonus very common
- Rapid progression—bed bound in 6-8 months
- EEG—periodic sharp waves are seen in the "sporadic", but not "Mad-Cow" types
- Brain biopsy only sure way of diagnosis
- Treatment—not at all clear what may be helpful

Alcohol Induced Dementia

- It has been debated if there is a clear "alcohol dementia". There probably is not a clear alcohol induced dementia. However, dementia seen with alcoholism does indeed occur. Dementia associated with alcoholism is either from head injury from alcohol associated misbehavior (head injury occurs in about

70% of patients with alcoholism who present to a mental health clinic) or Wernicke-Korsakoff syndrome, which is thiamine deficiency that is only very occasionally seen in those with alcoholism and a poor diet.[85]

- Wernicke's Syndrome—presents with confusion, ataxia, eye movement problems

- Then can progress to Korsakoff Syndrome (short term memory problem)

- CAUSE—Thiamine (Vitamin B1) deficiency. Heavy alcohol use interferes with metabolism of thiamine, and poor diet leads to lower intake too.

Wernicke's-Korsakoff Syndrome:

- Treatment: Thiamine—give intramuscular shots first then pills.

- Give thiamine before giving glucose. (If glucose is given first, it will speed metabolism making the deficiency of thiamine even more pronounced, making the symptoms worse.)

- Stop drinking to improve health.

- Short term memory loss (that is the inability to code new memories) occurs, leading to confabulation (making up stories to fill in the gaps of poor memory)

- If drinking stops with the addition of thiamine and a reasonable diet, FURTHER memory loss should stop too. But, you will

likely be left with the memory problems you already have.

Normal Pressure Hydrocephalus[86]

- Build up of fluid in brain
- Caused by "blocked outflow" of fluid in the brain, caused by a blocked "outflow" in the choroid plexus. The most common things that can block up the outflow are old blood (from prior trauma) or pus (from past infections)
- Trauma (blood), brain infections, or brain surgery puts one at risk
- This may occur in up to 5% of all dementias?
- Presenting symptoms: dementia, gait disturbance, incontinence subacutely over months
- Diagnosis—from brain imaging
- Treatment—shunt placement—50% improve
    - Best outcome in those with minor symptoms

AIDS Related Dementia[87]

- Before antiretrovirals, occurred in 25% of those with HIV disease, now in 10%

- Presents with "subcortical dementia" picture, that is social withdrawal, low energy, and low initiative to do things

- Most common cause of dementia in those 25-40

- Diagnosis is made by: HIV blood tests, brain imaging, cerebrospinal fluid exam

- Treatment: antiretroviral therapy, other AIDS treatment can be effective

Other Causes Of Dementia

- In up to 10% of dementia victims on autopsy, the pathology or exact reason of their dementia illness is not clear. They clearly had dementia clinically, but the pathologist is not able to clearly identify what sort of dementia they may have had on autopsy exam. So, nobody, even the pathologists, knows everything.

SUMMARY

So, there are a number of different types of dementia besides Alzheimer's Disease. Frequently, several types of dementia can co exist in elders. That is, elders can have several types of diseases at any one time.

The clinical care is the same. The disease should be identified, and the patient and his/her family should be educated about the disease and its

consequences. Proper clinical care, usually at home, is pursued. In those with vascular dementia, the best medication treatment is aspirin, which serves as a "blood thinner". For other types of dementia, a trial of a cholinesterase inhibitor or memantine can be tried with the hope that the progression of the illness can be delayed.

Behavioral problems are treated first with behavioral interventions. If these fail, a trial of medications can be initiated to symptomatically treat behavioral symptoms. No FDA approved medication exists for the treatment of agitation in dementia, so these trials should be carefully monitored for efficacy and side effects.

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CHAPTER 6: DELIRIUM

Delirium is brain "fogginess" which is a common problem in hospitalized elders. While occasionally a single cause can be found, usually it is caused by a combination of factors, such as infection, sedating medications, and chemical or electrolyte disturbance in a frail elder. Treatment involves the early recognition of the syndrome, removal of offending agents, and treating psychiatric symptoms that are seen.

- As opposed to dementia, which is brain failure which occurs, usually, gradually over time, delirium is acute brain failure with a subacute onset. It typically evolves over days to a week or so.

- It is important to identify the cause (meds, infection)

- Treat symptoms that emerge • Delirium is a poor prognostic sign. It is organ failure (i.e. brain failure) and indicates usually that a disease has progressed to the point that the brain can no longer manage usual insults.

DELIRIUM

Clinical features of delirium:

- Acute onset
- Fluctuating course
- Inattention
- Disorganized thinking
- Altered level of consciousness
- Cognitive deficits
- Perceptual disturbances (illusions or hallucinations in 30% of patients)
- Psychomotor disturbances (patients can be hyperactive or hypoactive)
- Altered sleep-wake cycle
- Emotional disturbances, including labile mood[88]

How often does delirium occur?

- 20% of inpatients 65 or older
- 36.8% of surgical pts[89]
- 50%-80% incidence in ICU[90]
- Prevalence is 1-2% in the community[91]

What is the cost of delirium in American society?

- Because delirium is often not recognized, it is hard to estimate
- One view of the cost of delirium in US: $38 million to $150 million[92]

What is the etiology of delirium?

- Occasionally, delirium has one etiology, but usually in a frail, ill elder, the cause is usually multifactorial
- Occasionally one prime factor, but usually a combination of:
    - illness (beware infection, sepsis)
    - meds (beware anticholinergic or sedating drugs)
    - labs (check glucose, lytes, CBC)
- Associated with decreased cerebral blood flow: prefrontally, thalamus, basal ganglia
- Associated with altered cholinergic system; inflammation;
- Associated with chronic "stress" which leads to hypercortisolism, as well as an increase in cytokines[93]
- Also consider…
    - weakened state (dementia)

- substance abuse

- sensory impairment which can cause an elder to misinterpret stimuli, contributing to confusion

- Post-operative care: With surgery, in addition to the physical insult to the body, the patient will have had anesthesia, which can linger, and contribute to delirium.

- Sleep deprivation (in hospital)[94]

Dementia and delirium are often seen together.

- Because patients with dementia already have problems with brain function, it doesn't take much to make their already burdened brains function less well.

- If you have dementia, and develop delirium, the one year mortality rate is 35-40%

- Dementia patients have 2-5 fold increased risk of developing delirium

- 2/3 of delirium pts also have dementia[95]

- For example, patients who develop delirium after cardiac surgery have lower MMSE score[96]

- Those patients with dementia and delirium die earlier than with dementia alone: They have an 80% higher death rate[97]

- A research survey of 263 hospitalized patients with Alzheimer's Disease shows:[98]

    - 56% developed delirium; rate of deterioration BEFORE hospitalization was not different

    - BUT…Those with delirium had "twice the rate" of deterioration in the year following admission, AND the increased rate of deterioration continued for FIVE YEARS

    - So, delirium is a poor prognostic sign

Evaluation of delirium:

- Evaluate meds (Remove the sedating meds, and any anticholinergic meds if at all possible)

- Check for infection: Vital signs, urinalysis, chest x ray

- Labs: check CBC, lytes, BUN, Cr, LFT's, ammonia, glucose, EKG, O2 sat

- MMSE or other cognitive screening tools can be widely varying. By definition, delirium presents with a waxing and waning level of consciousness. MMSE scores, then, can vary widely depending on the level of alertness and concentration.

TREATMENT OF DELIRIUM

- FIND THE ETIOLOGY!!

- (But, half the time, there is not one etiology. “Tuning up the patient” by removing unnecessary medications, correcting electrolyte imbalances, treating any infection, etc. is important. But, usually, there are a series of small insults that add up to cause delirium in a compromised patient.)

- Maintain patient safety

    - Manage the environment (reorient pt, clock, calendar are helpful); maintain hearing aids, glasses

    - Prevent hypoxemia, infection, constipation

    - Manage agitation (behaviorally intervene, use sedating meds judiciously)

    - Remember alcohol or drug intoxication or withdrawal can be a cause (including prescribed medication)—drug withdrawal can contribute to the cause of the delirium, or be the main cause of delirium.

    - Treat pain—this is under-recognized

Delirium takes days to weeks to clear. A supportive environment is always helpful. A clock in the room with a calendar is helpful. Identifying yourself as you enter the room is helpful as well. The presence of family, or familiar objects for the patient is always appreciated. And, make sure that eyeglasses, and hearing aids are available and working to make the environment less confusing.

Medication treatment of delirium:

- Combination of Haloperidol 5 and lorazepam 2mg for healthy adults is used. But, for geriatric patients, a smaller dose should be given. Haloperidol 1-2 mg IM and lorazepam 0.5 to 1 mg IM or PO is helpful.

- Can be put in the same syringe

- Less likely to develop EPS (6%) if both lorazepam and haloperidol are given together[99]

- Combination therapy with both of these drugs is superior to either alone[100]

- Atypical antipsychotics (quetiapine 50-50 bid) are commonly used, generally well tolerated, but no double blind trials demonstrate their usefulness or superiority[101]

PREVENTION?

Can we prevent delirium?

- Good nutrition is always important (pts with low albumin are more likely to get delirium)

- A medical tune up is useful to help to be sure that cardiac status, and blood pressure and blood sugar are optimal

- Meds? There has been a wide search for a medication which may help to prevent delirium. Largely this has been disappointing, but a few glimmers of hope are found in the

use of:

- melatonin 0.5 mg hs[102]
- risperidone 1 mg hs one time in PACU reduced delirium from 32% to 11%[103]
- donepezil, rivastigmine—not helpful in preventing delirium

SUMMARY

Delirium is a difficult condition to treat, and once it occurs it is a harbinger of a poor prognosis, usually in a frail elder, whose health is failing. Recovery can be slow—over weeks. The key to prevention is in limiting sedating medications as much as possible, and ensuring that the elder is in as good a medical condition as possible before initiating surgery or procedures.

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CHAPTER 7: ALCOHOL AND DRUG ABUSE IN ELDERS

Alcohol and substance abuse is sometimes an area of health concern neglected in elder psychiatry. The current group of elders, emerging from the "baby boom" years grew up in a culture which was more accepting of drug and alcohol use. Consequently, the prevalence of drug and alcohol abuse in this age group is more than in cohorts of elders in the past.

Finally, an emerging problem in drug and alcohol abuse in elders is the abuse or prescription medications, or over the counter medications. The misuse of these medications is growing, with results that can be tragic in elders who may have frail health and more susceptible to the effects and side effects of these medications, especially when combined with the other medications they are taking.

AGING AND ALCOHOL USE

Alcohol use grows in the teen years, and then reaches a peak when an individual reaches their twenties. There is a decline in alcohol use in the thirties, and then alcohol use remains relatively constant until the end of the fifties, when it again declines. Alcohol misuse follows the same pattern.

However, alcohol use and misuse can continue into the sixties and into old age, albeit at a lower rate. About half of those in their sixties use alcohol, and about half do not. Alcohol use and misuse rates continue to decline in the seventies and eighties.

Prevalence of alcohol abuse in primary care practices:

- Survey of 224 patients (age 60 or over) in 4 primary care practices showed:[104]
    - Major Depression in 6.5%
    - Minor Depression in 5.2%
    - EtOH abuse in 2.3%

So alcohol abuse continues to be a clinical problem in a small number of elders.

Will the next generation of elders have a higher substance abuse rate than their predecessors?

- Baby boomers are aging (Baby boomers are those born between '46 and '64)
- There is a concern that baby boomers, who during their youth may well have had a greater acceptance of drug and alcohol use or abuse in their culture, may well carry that same cultural belief into old age leading to higher rates of

substance abuse. This same factor may also bring higher rates of drug abuse into old age as well.[105]

- Rates of alcohol and drug abuse in elders are indeed rising[106]

Rates of substance abuse are rising in elders

- From early '90's to '02, prevalence of alcohol abuse or dependence tripled to 3.1%
- Heavy and binge drinking increased to 7.6%
- Rates of illicit drug use among those aged 50-54 increased from 3.4% to 6%

So, there is a projected increase in elders seeking care for substance abuse.[107]

- SAMSHA review (2010)—proportion of substance abuse treatment admissions:
- Those over age 50 were:
    - 6.6% of admissions in 1992
    - 12.2% of admissions in 2008
- During this period, there was an increase in marijuana, cocaine, heroin admissions, but the most common addiction leading to admission was for alcoholism

Another survey of elder drinking showed increase rates of alcohol abuse:[108]

- 13% of men and 8% of women reported at-risk alcohol use
- Binge drinking in 14% of men, 3% of women

Surveys in primary care practices show the same growth of substance abuse in elders:

- 24,863 elders greater than age 65 were screened for alcohol use in 6 VA's, and 5 non-VA primary care sites[109]
    - 70% reported no drinking in the past year
    - 4.5% had heavy drinking (>14 drinks/week) or binge drinking—this was associated with anxiety/depressive symptoms and perceived poor health
- In another survey of 23,000 patients in primary care who were over age 65, 6% were at risk for alcohol diagnoses[110]

A British survey of the "old old" also shows similar findings:[111]

- In the United Kingdom, 15,000 patients older than age 75 were surveyed, with the median age being 80
- 5% of men drank >21 drinks per week, and
- 2.5% of women drank more than 14 drinks per week

Risk factors for problem drinking in those over age 65:[112]

- MEN:
    - Higher income
    - Being divorced, separated or widowed
    - Problem drinking was associated with use of tobacco and illicit drugs
- WOMEN
    - Being employed
    - Non-medical use of prescription drugs
    - Problem drinking was associated with use of tobacco and illicit drugs

What is the prognosis for elder drinkers?[113]

- In the past, it was thought that elders with alcoholism fell into one of two groups:
    - About one third of those with drinking problems were thought to develop it later in life in response to grief or a personal crisis. This group was thought to have a better prognosis. Treatment of this group was simply grief work, and it was thought they responded well.
    - However, about two thirds of elder problem drinkers were thought to have simply grown older with pre-

existing alcoholism. These people, "the survivors" were thought to have a poor prognosis, since they had the disease for decades, and already survived into old age.

Reviews of current alcohol treatment for elders don't agree. The treatment prognosis in treating elders for alcoholism is at least as good.

- This study showed an examination of 12 month drinking trajectory in at risk drinkers over age 65:[114]
  - Improvements were made throughout the year
  - At the end of 12 months only about half of the "at risk" drinkers were still at risk

A follow-up phone survey shows the same:

- Compare 65 patients older than age 55 to 860 younger pts on 5 year follow up phone survey[115]
- Older pts did better (52% total abstinence from alcohol and drugs in last 30 days compared to 40% of younger pts)
- Older women did better than older men

Those elders who begin drinking to excess later in life (after age 45) do indeed have a better prognosis. They showed:

- Lower amounts of alcohol consumption

- Higher rate of abstinence at 12 month follow up than elders who had early onset (onset before age 25)[116]

Elders given naltrexone as treatment for alcohol dependence also did better than younger patients.

- Review of a naltrexone trial for alcoholism compared older vs. younger patients[117]
- Older adults had greater attendance at therapy sessions and greater adherence to the medication
- There was also less relapse for older patients

Another large survey also shows that older patients with alcoholism did well in treatment:[118]

- Reviewing 1358 admissions to a residential program—compare older pts to younger pts
- Older pts did as well (measured by one year abstinence rate)
- Older patients had lower severity of alcohol dependence on admission (but had more somatic problems)
- Older adults engaged in formal post-discharge aftercare less than middle-aged adults

So, alcohol and substance abuse can be a continuing problem in elders.

Those referred for treatment do at least as well as younger patients, so it is important to recognize and treat substance abuse in elders.

However, an emerging problem of drug abuse in elders is the abuse of prescription drugs—usually benzodiazepines and narcotic pain medications. The next series of studies shows the severity of this problem.

DRUG ABUSE IN ELDERS

Prescription drug abuse is a large, and growing problem among elders.

- Elders (over 65) are 12% of population
- Consume 25% of all drugs
- 87% of elders use over the counter meds
- 6% are taking 5 or more over the counter meds

In depressed elders, prescription drug abuse is also a big problem:[119]

- Survey of 154 pts over age 60, who were depressed (Beck >10) coming to clinic
- Half used alcohol in last month
- In prior 30 days:
    - Cannabis use in 12% of men and 4% of women;

- misuse of sedatives in 16% of men and 9% of women

- Cannabis use associated with higher Beck Depression Inventory scores

Among those in their fifties, drug abuse rates doubled over the last decade.[120]

- NIH reports from 2002 to 2010, substance abuse more than doubled (including NON-MEDICAL USE OF PRESCRIPTION DRUGS) from 2.7% to 5.8% of the population

- Among those over 65: 414,000 abused drugs

A survey from Florida shows an alarming rate of prescription drug abuse among elders:[121]

- 3497 elders screened for drug abuse in Florida

- Most common drug abuse problem in elders is prescription drug abuse (next is alcohol abuse, but next after that is over the counter drug abuse, with last being street drug abuse)

- Depression is common in elder drug abusers

Another survey shows 11% of elders abuse prescription drugs, and risk factors are female sex and loneliness:[122]

- 11% of older women abuse prescription drugs

- Factors associated with prescription drug abuse:
    - female sex
    - social isolation
    - history of a substance use or mental health disorder
    - medical exposure to prescription drugs with abuse potential.

However, not all surveys show an increase in prescription drug abuse among elders.

- In a national epidemiologic survey, 80,205 people older than age 65 were surveyed. Lifetime rates of substance abuse, and rates over the last year are presented:[123]

	Lifetime	last 12 months
Substance use disorder	21%	5.4%
Alcohol	16.1%	1.5%
Tobacco	8.7%	4%
Non Medical Drug Use	0.6%	0.2%

- Younger age and being divorced or separated were associated with greater odds of lifetime nonmedical drug use disorder

Street drug abuse is much less common in elders, but it can occur:

- Prospective survey of ER visits in those over age 60:
  - 5677 visits, urine obtained in only 911 visits
  - 2% were positive for cocaine[124]

SUMMARY

So, alcohol and drug abuse can continue into old age. In the past, alcohol and drug abuse problems among elders may have been ignored, or glossed over with the assumption that referral to treatment would not be successful. However, drug abuse treatment for elders is at least as useful as it is in younger patients.

An emerging problem in substance abuse with elders is the growing rate of prescription drug abuse. Elder women, especially with chronic medical conditions including pain, may be more vulnerable to this problem.

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CHAPTER 8: INTELLECTUAL DISABILITY IN ELDERS

One of the biggest public health success stories of the second half of the twentieth century was extending the lifespan of those with Down's Syndrome, the most common cause of intellectual disability. Attention to the health care needs of those with disability lengthened their life, and now, those with intellectual disability can usually live to their sixties or beyond. Because most of those with intellectual disability receive care at home, by their parents, this means that they are now surviving their parents—outliving their caregivers. In their old age, when they present with an accumulation of chronic health problems, we often meet them with an unclear medical history, and an unclear set of caregivers. Those who kept the chart of their lives—their parents—are no longer with us.

This chapter explores health issues in those elders with intellectual disability, as well as psychiatric problems with aging. Foremost among the psychiatric problems is the expanded prevalence of dementia in those with Down's Syndrome. A slightly increased risk of psychotic disorders in elders can also be seen. Substance abuse disorders may well be less in this population.

Whether you consider yourself expert in caring for this population or not, they are a growing segment of elders who present to our clinics and hospitals for care, and an awareness of their psychiatric care needs is helpful.

The DSM V criteria for Intellectual Disability (formerly mental retardation) have changed a bit from before. There is less reliance on the "IQ number" obtained on an IQ test (since testing is always a bit variable) and more reliance now on functional ability, which is more important to determine levels of care and support. Here are the new DSM V criteria for intellectual disability:

- Deficits in intellectual function determined by both clinical exam and IQ testing
- Deficits in adaptive functioning which cause problems in ADL's across multiple environments
- Onset during developmental period
- (Compared to DSM IV-less reliance on IQ and more on functioning, especially in social envt.)
- Specify severity as:
    - 317 (F70) Mild
    - 318.0 (F71) Moderate
    - 318.1 (F72) Severe

- 318.2 (F73) Profound

The American Association on Intellectual Disability and Developmental Disabilities developed a new description of level of disability based solely on function in 1992. They defined the following levels of support for those with intellectual disability:

- Intermittent support (need help only during times of stress or change)
- Limited support (may need some daily help)
- Extensive support (around the clock support)
- Pervasive support (daily support "in every aspect of daily routines")

CARING FOR INTELLECTUALLY DISABLED ELDERS

Psychiatric care for elders and for adults with developmental disabilities is often not available. Aside from dementia, it is estimated that about 10% to 40% (granted a wide range) of elders and adults with developmental disability have a psychiatric disorder requiring care.

- Those with Intellectual Disability have reduced access for psych services[125]
- In a British survey: 45% of 2711 adults accessed mental health

care. 33% had a psychiatric condition and 20% had 'behavioral disorders' (i.e. misbehavior of some kind requiring extra attention or medication)[126]

What is the rate of psychiatric disorders among adults with intellectual disability?

- Survey of 90 pts with intellectual disability in Britain (using ICD 10 criteria) showed higher rates of schizophrenia, but similar rates of depressive and anxiety disorders:[127]
    - Schizophrenia 4.4%
    - Delusional Disorder 1.1%
    - Depressive Disorder 2.2%
    - Phobia 4.4%

Substance abuse rates are not more common than in the general population:

- Review of 115 referrals in London. Only 15% had a hx of substance use, and only 8% of those were currently using alcohol or other substances.[128]

Comparing adult patients with intellectual disability to elders with intellectual disability, elders are much more likely to suffer from a dementia:

- Past history of any affective disorder:

- 16% in elders
- 10% in younger adults

- Schizophrenia or delusional disorder
  - 3% of elders
  - 3% of younger adults
- Autism:
  - 6% of elders
  - 7% of younger adults
- OCD:
  - 0% of elders
  - 3% of younger adults
- DEMENTIA:
  - 22% of elders
  - 3% of younger adults
- This study compared adults (20-65, n = 73) to elders (over 65, n = 134) with intellectual disability in Britain
- Psychiatric diagnosis was found in 48% of younger patients, and 69% of elder patients[129]

The care for adults and elders with intellectual disability over the last half century led to a true public health success story:

- Those with ID are growing older
- Average life expectancy now: 66.1 years
    - (average in U.S. is 77 years)
- Those elders now living with ID can expect to live a full lifespan, as do their peers in the United States

This success is especially true in those with Down's Syndrome:

- Most common cause of intellectual disability[130]
- Average lifespan doubled in only 14 years:[131]
    - 1983: 25 years
    - 1997: 49 years

The expanded lifespan of those with intellectual disability is bringing new problems (added cost, and caring for dementia in those elders with intellectual disability):

- Living longer is costing more
- Netherlands: Intellectual Disability is 9% of all disease specific costs[132]

- Netherlands: Intellectual Disability care is in top 5 costliest diseases[133]

This extended lifespan also brings more chronic illness in our elder patients with intellectual disability. For example, in vision care:

- Visual impairment: As severity of ID increases, prevalence of visual impairment increases[134]
- Down's: Age 50-59—half had moderate to severe visual loss[135]
- Down's: Over age 50—half had cataracts

Hearing problems are quite common in patients with Down's Syndrome:

- Down's: Age 50-59, in institutional setting—70% had moderate to very severe hearing loss[136]

And, in Down's Syndrome, cardiac and associated pulmonary problems continue as well in elder care:

- Congenital heart disease can continue
- Untreated cardiac or pulmonary conditions can surface or worsen[137]

Osteoarthritis and associated pain syndromes can be a big problem in

care:

- For patients with Down's:[138]
  - 1/3 had osteoarthritis
  - ½ had fractures of long bones
  - 30% had fracture of vertebral bodies
  - 8% had atlantooccipital instability
- Pain: as cognitive impairment increases, reporting of pain decreases. So, pain, especially in those who need total assistance can manifest as agitation or a behavioral disturbance.[139]

Thyroid disease is also common in those with Down's Syndrome, and this can mimic depressive symptoms:

- In a survey of 591 institutionalized Down's patients: 48% had increased TSH (hypothyroidism)[140]
- For patients with depressive symptoms, it is important to check TSH (thyroid stimulating hormone) with a blood test.

These problems with decreased vision and hearing as well as increased risk of arthritis leads to a bad combination of factors which can lead to agitation along with factors which make it harder to communicate these problems:

- Hearing and vision essential
- If not optimized—communication is impaired—thus:
    - Agitation may increase
    - Socialization may decrease
    - Quality of life decreases

So, do we turn to medication too often in these patients? It is difficult to determine this exactly. But, it is clear that the first medication to consider for agitation in elders with intellectual disability may well be acetaminophen.

- Survey of group homes in the Netherlands: 28% of pts. were "problematic"
- Psychotropics used by 53% of "problematic" patients, 23% of all pts.
- 3 or more psychotropics used in 17% of "problematic" patients, 7% of random group[141]
- And often specialist care is not available as we seek to care for these patients
- Survey of 80 patients with ID with "persistent challenging behavior" into adulthood—64% had NO specialist mental health care[142]
- Not likely to improve as elders

INTELLECTUAL DISABILITY AND DEMENTIA

The most common psychiatric condition bringing elders with intellectual disability to care is dementia. As those with ID age, especially those with Down's Syndrome, this is a growing clinical problem.

- Those with ID now are living long enough to get dementia
- Down's (trisomy 21): have high rates of Alzheimer's. At first, it was thought that all those with Down's Syndrome would get Alzheimer's Disease as they age. This is because deposits of amyloid protein is seen in the brains of those with Alzheimer's Disease.
- AND, the theory holds that—since amyloid precursor protein is coded for on chromosome #21—those with "trisomy 21" or "Down's Syndrome" have an added chance for more amyloid brain deposition, leading to Alzheimer's Disease.
- While not all those with Down's Syndrome develop a dementia as they age, their rates are much higher than in the general population. The rates of dementia in those with all types of intellectual disability are also higher than the general population. Perhaps this is due to the fact that brain injury is a risk factor for dementia.

Risk for dementia:

- In those with ID from all causes:[143]

- Age 65 and over: 20%
- Age 88: 52%

- U.S. population:
  - age 65: 3%
  - age 85: about 33%

The rates of dementia in Down's Syndrome are higher than the rates of dementia in the general intellectual disability population (and much higher than the rates in the general US population).[144]

Rates for dementia in those with Down's Syndrome are:

- Age 50: 42%
- Age 60: 56%
- Age 72: 67%

Diagnosing dementia in those with intellectual disability can be clinically challenging.

- The key may well be to document decline in abilities, and in activities of daily living over time.[145]
- New onset of delirium, depression—clue to a "change in mental status", which may herald the revelation of a dementing

illness[146]

Dementia care in intellectual disability:

- Treatment:
    - Cholinesterase inhibitor worth a try
    - Memantine not helpful (but the prospective study looked at cognition in both those with and without dementia)[147]
- Psychotic symptoms seen in 28%[148]
- Other behavioral symptoms common
- Patients with Down's and dementia—compared to patients with dementia and ID due to other causes— are more likely to have low mood, restlessness, disturbed sleep, hallucinations. But, they are less likely to be aggressive.[149]

Because the rates of dementia are so high, some have wondered if we should treat dementia before symptoms emerge in those with intellectual disability. Memantine was initiated in one trial.[150]

- Can it help memory in Down's before onset of dementia?
- Study of 40 pts, improvement in memory was seen in only one of the secondary measures and none of the primary measures or memory

- So memantine may well be of limited use in this population

The caregivers of those elders with intellectual disability are faced with unique problems.

- Elders with Intellectual Disability are usually being cared for by parents, or sibs
- Now, those with Intellectual Disability are outliving their caregivers
- "What will happen to him when I'm gone?" is a common question in clinics, and one for which there is often not a simple answer.
- In discussing these issues and problems, include patients in the decision making process when fashioning answers to the questions concerning care.[151]

SUMMARY

So, elders with an intellectual disability are an emerging group facing clinics and hospitals. The success of ongoing medical care to treat chronic health conditions led to a prolonged lifespan, and now, those with intellectual disability can often expect to live the same lifespan as other elders in our society. With those with Down's Syndrome, there is a remarkably high incidence of Alzheimer's Disease as they age. In these individuals, treatment

is the same, as we struggle with obtaining more effective treatments for those with Alzheimer's Disease.

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CHAPTER 9: ETHICAL PROBLEMS IN ELDER PSYCHIATRY

Ethical issues are typically posed as debating "right" vs. "wrong" ideas or attempts to deliver care. Unfortunately, life is seldom that simple. If all one had to do was to select the "right" approach, or "doing the right thing", there would be no real problems in ethics. Instead, ethical problems typically involve defining the issue and collecting opinions from a group of people on the topic. Usually, there are a series of "right" things to do. The "problem" in ethics is picking which right thing to do.

In addition, it is sometimes thought that ethical problems are simply decided by a wise, experienced clinician. While wisdom and experience are indeed good traits in making any decision, as always, having data to help guide decisions makes those decisions better informed, and indeed better for the patient or groups of patients involved.

ETHICAL PRINCIPLES

An example of "Right vs. Wrong" decisions:

- Before and during World War II, the Nazi's experimented on those they viewed as being "less" themselves. They did

horrible medical experiments on Jews in camps, and on political prisoners.

- Yet, at the same time in the United States, medical experimentation was going on among another group of people.

- 1932—US Public Health Service conducted experiments on a group of black men to study and follow the "course" of syphilis

    - The United States Public Health Service left them untreated (without the knowledge of the men being experimented on)

    - This went on for FORTY YEARS until it was exposed in 1972[152]

- AND in 1928, Dr. Wile at the University of Michigan bored holes into skulls of psychiatric patients with tertiary syphilis to extract brain tissue to see if the disease could be spread to rabbits

    - Study had absolutely NO therapeutic benefit

    - Patients could not give informed consent, families were not consulted[153]

These problems were identified, and a series of rules or general principles were advised for conducting research, and generalized to clinical care as a whole.

- In research: Nuremberg Code was proposed for the conduct of research studies.[154] Key components of the Nuremberg Code included the ideas that:

- VOLUNTARY CONSENT of research subject is essential

- Research should stop if harm is being caused

- The patient has right to withdraw at any time

Other ethical principles:

- BENEFICENCE: Hippocratic Oath: We will treat patients with their best interest at heart

- CONFIDENTIALITY is essential for proper treatment of patients, and in conducting research

- "PRIMUM NON NOCERE"—above all, do no harm

- Paternalism—looking out for the best interests of others

- Self Determination

- Looking Out for the Public Good

Following World War II, "the Nuremburg code of ethics" has received widespread support and praise. While it is generally accepted…

- "The Nuremberg Code" has not been officially adopted in its

entirety as law by any nation or as ethics by any major medical association."

- So, there are no hard and fast rules, but rather a series of guidelines, AND a sense that a continual examination of ethics is essential[155]

Continual examination of ethical ideas is essential, because both data and ideas about treatment evolve over time… But, it's not always clear what the right thing to do is…

EVOLUTION OF ETHICAL TREATMENT

Example) Antipsychotics for agitation/paranoia in elder dementia:

- Psychotic symptoms occur in 30-50% of those with Alzheimer's Disease

- Agitation also occurs in many—up to 70%—of those with Alzheimer's Disease[156]

- Over a ten year period, from 1996 to 2006, use of antipsychotics increased rather dramatically in US Nursing Homes. In 1996, 16% of US Nursing Home patients received an antipsychotic, and this grew to 26% of all patients by 2006[157]

- During this period, a series of "atypical antipsychotics" were presented to the US market. Not one was statistically significantly associated with an increased rate of death.

- But then, Lon Schneider at USC coalesced data for a number of studies, for a number of the new antipsychotics—the drug companies agreed to give him the data to review

- He found that although any one drug did not show an increased risk of mortality in any one single study, if all of the studies were combined as a group, they showed a slightly greater risk of mortality (2%) and stroke. This is because having a larger number of patients allowed smaller differences to become statistically significant.

- So…what to do now…new information showed that these drugs which were commonly used to treat a common symptom in those suffering from Alzheimer's Disease led to an increased risk of death.[158]

- And, further data showed that these new drugs were not all that effective in treatment of the symptoms of psychosis and agitation.

- There were no statistically significant differences among treatment groups in time to discontinuation BUT time to discontinuation due to lack of efficacy favored olanzapine and risperidone, whereas time to discontinuation due to adverse events favored placebo[159]

- So, treatment that once was viewed as appropriate, "good" and "ethical" and quite common in American nursing homes, was now suspect.

- So, over time, because of data, treatments changed from being a

usual course of care to being viewed as dangerous or even life threatening. Research data not only changed treatment, but also ethical views of this problem.

- "If we knew what it was we were doing, it would not be called research, would it?" –Albert Einstein

So, the "Right vs. Wrong" decision can change over time with new data. Another way to view ethical problems is the problem of the "slippery slope", or that ethical decision making is not deciding which good thing to do, but rather how much of a good thing is good.

"SLIPPERY SLOPE"

For example, in elder psychiatry, the question of "physician assisted suicide" presents how far a "good thing" can be pushed.

- Physician Assisted Suicide is now not only a medical question but is also a political question

- The question is how far we are willing to go to "end suffering" vs. suicide

- The ethic principles being advocated are "beneficence" (i.e. ending suffering) and "Patient self determination" where a patient makes his own decision about care, and indeed about life itself, and the value of one's own life. This occurs against a backdrop of a seeming overwhelming bureaucracy of

medical care[160]

- The "Death with Dignity Act" passed in Oregon in 1994
    - Of all the patients since the act was passed who asked for and were given prescriptions to kill themselves 2/3 of those who get prescriptions take them to kill self
    - The number of request increased gradually until '02-'03 and then stabilized, at about 60 requests per year
    - now 1 in 1000 deaths in Oregon are due to "assisted suicide"[161]
    - Interestingly, 5 patients took lethal a lethal dose of medication, but did NOT DIE. 2 died within 38 hours, BUT 3 died 5 days to 3 months later of their underlying illness. NONE of the patients who woke up after their failed attempt to kill themselves took more meds to kill themselves once again after their failed attempt.
- Netherlands passed a law in 2002 allowing euthanasia, and physician assisted suicide
    - over the last 17 years, the number of euthanasia and physician assisted suicide deaths are stable[162]

So, is physician assisted suicide "ethical"? It is a debate between two competing ethical concerns.

- "Primum non nocere"—"First, do no harm" to the patient VS.

- "Self determination" of patients in a health care system sometimes viewed as being too big, and too out of control

- How far to take "self determination" is a "slippery slope". How far does it become a problem?

And, how a question is framed can affect the range of decisions.

- For example, should we pursue "Assisted" suicide, or "killing ill patients"? It's the same fact, but approached by different ethical angles.[163]

- Another example of the way questions are framed is the question of "Death Panels". In the original health care reform law, "death panels" was actually a change in Medicare regulations which would allow physicians to discuss advanced directives with their patients and bill for this time. However, in the heated political arena of modern Washington, DC, this was reframed as "Death Panels".

- So, are you in favor of "death panels", or being able to direct your own health care in advance ("Self determination") if you are too ill to communicate your decisions (Advanced directive discussions)? It's the same thing, presented in different ways.[164]

- (Just so you know, my belief (bias?) is that physician assisted suicide is wrong, and "primum non nocere", or "first do no harm" is the ethical principle which takes precedence here. And my belief is that "Self Determination" is the ethical

principle which takes precedence in the "death panel" discussion over the fear of government intrusion in health care decision making.)

USING THE DATA

Can we get any help in ethical decision making…can ethical decisions among elders be "studied"?

- For example, in elder psychiatry, we often see suicidal ideation, and even suicide attempts. Should we allow patients to go ahead and die in a suicide attempt ("self determination") or should we intervene?

- Can data help?

- Medicare mandates that patients admitted to hospital be informed of their right to give advanced directives. On our inpatient geriatric psychiatry unit, we asked those patients admitted with suicidal ideation what they would want us to do if their hearts suddenly stopped. 40% of those suicidal patients admitted with major depression or suicidal ideation wanted full "code" or full resuscitation (this is compared to 56.7% without suicidal ideation— $p<0.05$). So, less suicidal patients would want to be resuscitated ($p<0.05$), BUT a full 40% of suicidal elder patients severely ill enough to be admitted to a psychiatric facility would want full resuscitation efforts.[165]

What can the study of advanced directives tell us about end of life decision making by families with an ill family member with Alzheimer's Disease?

- Alzheimer's Disease is a difficult illness
- Prognosis uniformly poor—leading to death
- What about advanced directives—what to do if your ill relative's heart suddenly stopped?
- What to advise?
- Again, we decided not to "decide" this for our families, but rather asked the families what they would want, or "direct" for their ill family member in such a case. We measured how severe the dementing illness was in each patient with the use of the Mini Mental Status Exam.
    - Families who said do all measures to save their family member—their demented family member had an average MMSE=14
    - Families who said "no code", or do nothing if the demented patient's heart would stop had an average MMSE=14 (THE SAME!!)
- Therefore, doctors or staff on a geriatric psychiatry unit, could not predict advanced directive decision making on the basis of how demented the patient was. Families make decisions based on a variety of principles, and the doctor cannot

"know" without asking.

- Therefore, here, "Self determination" by the family member making decisions outweighs the "beneficence" of the doctor who does not know the patient as well as the family, or what ethical principles the family is using to make decisions—even though they know much more accurately how demented, or how severely ill the patient is.[166]

Another example where data is helpful in ethical decision making is in addressing suicidal ideation or intent in those admitted to rehab following a spinal cord injury.

- Spinal Cord Injury is a devastating injury, leading to permanent disability…

- Who wouldn't be suicidal immediately on admission to a rehab facility?

- 81%

  - That is, 19% of spinal cord injured patients admitted to a rehab facility expressed suicidal ideation. So, suicidal ideation in this group is uncommon, and is a symptom, or sign of a treatable illness—depression.[167]

So, data is always helpful in informing ethical decisions, and can help to define and solve them.

FINDING COMFORT IN CONCLUSIONS

Another way to view ethical decisions with competing ethical concerns is to explore the degree of comfort in coming to a conclusion:

- 1) fully worked out
- 2) debated
- 3) not even recognized

Fully Worked Out Ethical Decisions In Elder Psychiatry

- Involuntary Psychiatry admissions
    - paternalism vs. self determination (or public safety)
- Can admit involuntarily if a danger to self or others (or likely to come to harm in 30 days)
- Emergently or non-emergently

Another example of ethical decisions which have been "fully worked out" is "forced medication treatment".

- Patients can accept OR REFUSE medication treatment (if competent)
    - (self determination vs. paternalism)

- This is true even in patients who are involuntarily committed to an inpatient psychiatry unit

- AND patients can accept or refuse treatment even in advance (advanced directive)

Another example of an elder psychiatry ethical problem which has been fully worked out is whether or not to report elder abuse:

- It is generally recognized that one should report even a suspicion of elder abuse

    - Paternalism wins vs. self determination or confidentiality of the patient

- And, not reporting a suspicion of abuse is a serious offense (e.g. Penn State's handling of Jerry Sandusky child abuse case)

Yet another example of an ethical issue that is fully worked out is organ donation in elders.

- Organs for transplantation can be donated only if the individual agrees before hand

- (here, self determination wins vs. beneficence—or taking the organs anyway because they are desperately needed)[168]

Elder Ethical Psychiatric Issues That Are Debated

- Self Determination vs. "Primum Non Nocere"

- e.g. “assisted suicide”

- Physician Assisted Suicide (Self Determination vs. “Primum Non Nocere”)

- Another “grey area”—ability to give informed consent, or “competence” to consent to procedures (Beneficence vs. Self Determination)

- “Obamacare”: should we mandate all citizens to have health care insurance? (“paternalism” vs. “self determination”)

Unrecognized Ethical Issues

Yet, there are ethical issues that are not recognized. Many concern how money in health care is spent.

- Money: For many years, there were unequal payments in Medicare for psychiatric vs. “medical” problems. Was it “ethical” for the government to pay less in reimbursement for different health problems that involved mental health?

- Money: “Carve out” of psychiatric care to for-profit companies to manage psychiatric needs in insurance policies. Is it “ethical” to make a profit on illness?

- State budget: Should we have “state hospitals”? Is it the responsibility of state government to provide facilities for mental illness?[170]

Another unrecognized ethical problem—a "new" health problem—the growing population of elders with intellectual disability.

- Most common cause of intellectual disability is Down's Syndrome[171]

- Average lifespan for those with Down's Syndrome doubled in only 14 years. This is truly one of the great health achievements in the latter half of the twentieth century…that nobody knows about:

    - in 1983, the average lifespan was: 25 years

    - in 1997, the average lifespan was: 49 years[172]

- Now, care for those aging with ID is confronting the American health care system

- Many are outliving their parents. Where is proper place to care for them "once I'm gone" from the home?

- What say do they have in care ("paternalism vs. self determination")? Do those with intellectual disability have the "right" to determine where they live? To determine the amount of assistance they receive?[173]

What about dementia care? Should we pay for cholinesterase inhibitors for treatment?

- Britain has a national health service, whose role is to use the

budget to provide care for all

- Focus is on the Public health not just individual health

- At first, donepezil was not available in Britain. It was felt cost of these meds were not justified. That is, they did not work well enough to justify the expenditure. There was an outcry from the public to provide these meds.

- Now, they are available —and for advanced dementia can choose donepezil OR memantine (but not both) OR nothing[174]

- The decision on not providing both medications was helped by data— a British study of 295 elders with advanced dementia, SMMSE = 5-13

- The study was done on those residing in the community. On one year follow up:

    - Continuing donepezil decreased cognitive decline by 1.9 pts

    - Adding memantine made NO significant difference

    - Donepezil had 23% less decline than placebo (but these patients still continued to decline)[175]

- Again, data helps to make the decision

So, how do you figure out ethical problems?

- Is it an emergency?

- (In an emergency, act to save the person first—can debate it later)

- Usually, Beneficence rules

- If not an emergency, time is an advantage. Take advantage of that time:

  - Look for data

  - 2 heads are better than 1 (i.e. Refer to "ethics committee")

- Keep looking for ethical problems (they'll be there)—especially those which are not recognized

- Talk about it, "The role of a health care provider is to comfort the afflicted and afflict the comfortable."

- Recognize that money, politics will take a role[176]

SUMMARY

- We have been using ethical principles in medicine since Hippocrates (or before)

- Ethical concepts are often competing. That is, usually it is not a question of "doing the right thing", but rather "which right thing to do"

- Some ethical problems have been fully worked out

- Other ethical problems—not recognized
- When a problem hits
    - if emergent, pursue beneficence,
    - if not—take time and consider—ask for help

Data can help settle the question.

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CHAPTER 10: CONCLUSIONS

This has been a brief exploration of geriatric, or “elder” psychiatry. The burgeoning population of this group brings elder psychiatry into every hospital, and every psychiatric clinic. Exploring the many facets of elder psychiatry helps to have a broader understanding of how to recognize these problems, and to hopefully do a better job of caring for these disorders.

In elder psychiatry, patients often present to us at the worst part of their lives. In dementia care, the individual who inhabited the body, and pursued dreams and work that cared for the people bringing them to the clinic, is often slipping away. Depression robs the life force from an individual. Substance abuse can continue to lead the elder individual away from their ongoing potential. Delirium can suddenly take the individual out of focus, and present difficult psychiatric problems.

Better medical care and better psychiatric care can extend these lives, leading to healthier and more fulfilling times for our patients. Nowhere is this more evident than in the care of elders with intellectual disability.

Finally, an examination of ethical principles can be helpful when we are met daily with concerns and questions about care that continue to circle in

our head.

This brief book hopes to convey information about these important topics, and at least an outline in how to think about them. One thing is for sure. We don't have good enough answers for these many conditions now. The future will bring better ideas, better treatments, and better answers for these conditions.

You are part of this future.

ABOUT THE AUTHOR

Paul Kettl, MD, MHA is a psychiatrist at the Philadelphia VA Medical Center and is Clinical Professor of Psychiatry at the Perelman School of Medicine of the University of Pennsylvania.

He treasures the time he spends teaching medical students and residents, and the outlines in this book are a result of the questions and concerns raised over the years by his students.

Dr. Kettl also dabbled in politics, and won the democratic nomination for US Congress from his home district before losing the general election to the republican incumbent. He explored the interface between psychiatry and politics in his first novel, *The President's Secret*, which is available now on Amazon as an E book or paperback.

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