Why do we need to know about Major Depression Disorder?

Common – 8-10% of men; 15-20% of women lifetime prevalence – 1 year prevalence about 7%; so 19 million Americans suffering

Incidence and prevalence increasing with time – cohort of women born in the 1970’s and 1980’s– as high as 25% prevalence

Burden of Disease – projected to be the 2nd leading cause of disability world wide by 2020 (WHO study) – heart disease #1 – Costs the US 100 billion dollars (2012) – equivalent to cost of cardiovascular disease or cancer

Treatable if someone seeks help; many families effected in many different ways

Criteria for Major Depression Disorder
(DSM 5 – Diagnostic and Statistical Manual)
Five or more of the following must be present for two weeks and represent a change from previous functioning. Must have 5 of the following 9 symptoms and must include either depressed mood or loss of interest or pleasure

(1) Depressed mood most of the day, nearly every day, as indicated by either subjective report or observation by others (In children and adolescents, can be irritable mood).
(2) Markedly diminished interest or pleasure in all, or almost all activities most of the day, nearly every day (as indicated by either subjective account or observation made by others).
(3) Significant weight loss when not dieting or weight gain or increased or decreased appetite nearly every day.
(4) Insomnia or hypersomnia nearly every day.
(5) Psychomotor agitation or retardation (Observable by others).
(6) Fatigue or loss of energy nearly every day.
(7) Feelings of worthlessness or excessive/inappropriate guilt.
(8) Diminished ability to think or concentrate—or indecisiveness.
(9) Thoughts of death or suicide.

Above cannot be due to a general medical condition (see below) or the direct physiological effects of a substance.
Must cause significant impairment or distress in social, occupation, or other areas of functioning

Definitions: Mood is what the patient states they are feeling in general
Affect is what we observe at the moment  Mood:Affect::Climate:Weather
Many students use the pneumonic **Sig. E. Caps** to remember the above 9 criteria.  
S(adness)  
I(nterest)  
G(uilt)  
E(nergy)  
C(oncentration)  
A(ppetite)  
P(sychomotor agitation or retardation)  
S(uicidal thoughts)

**Risk Factors:**
- Women 2:1 men – ratio narrows to 1:1 postmenopausal  
- Age onset: peak 20-30’s – this is decreasing with time – some studies suggest that 10% of adolescents may now have depressive symptoms  
- Family History – moderate genetic risk; if 1 parent 10-15%, 2 parents 20-30%; MZ twins 50%, DZ twins 15-20% - (if take into consideration “Affective Disorder Spectrum” – Major Depression, ETOH, Somatization, Antisocial personality then genetics more robust) Having a family history not only as a genetic base but also the psychological and environmental disruptions  
- Single, divorced, widowed > married (except in elderly males – high suicide group)  
- Income, profession, religion, geography have minimal impact  
- Cultural: some trends – lower in African-American men, Asians; higher in Hispanic women and American Indians  
- Childhood major negative events are a big risk factor – loss, neglect, abuse  
- Onset of illness usually a series of negative life events, but can also be one catastrophic event – death, loss, medical illness, etc.  
- 50% of people with only 1 episode, but if 2 episodes 70% of another, if 3 episodes or more 95% chance of another – for most it’s a reoccurring chronic illness  
- Triggers to relapse over time are less and less (stress vulnerability model)

**How to people develop a Major Depression Disorder?**

1. Genetic predisposition – smaller hippocampus, abnormal serotonin transport protein (now measurable through genetic testing) etc.  
2. Poor psychological coping strategies or skills- secondary to trauma, loss, dysfunction, societal situations, etc (lack of resiliency)  
3. Triggering events – Biological (could be medicines, substances, diseases), psychological, environmental  
4. Change in brain processes that cause us to interpret external or internal stimuli in different ways - negative cognitions, pessimism, physical changes, withdrawal, retreat, lack of rewards, altered self awareness  
5. These symptoms hinder our ability to reach our previous neurobiological homeostasis via neurogenesis through enrichment:  
   - Social connection
Mechanisms of Human Disease
Mood Disorders I and II and Suicide Risk
Tuesday, February 13, 2019 – 9:30 and 10:30am
Ralph Orland, MD

- Exercise
- New learning
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Figure 1. A Diagram of Five Possible Outcomes (Response, Remission, Relapse, Recovery, and Recurrence) During the Three Phases of Treatment of Depression (Acute, Continuation, and Maintenance).*

Pathophysiology

Major Depression is a neurodegenerative brain disorder, but we do not yet have a biological marker! – The closest marker we have is the theory of chronic low level increases in cortisol secondary to stress which causes a disruption in healthy neurogenesis and may add to neurodegeneration. Genetics and epigenetic are involved and it is a very heterogeneous phenomenon.

Different theories arise:
Traditionally the theory of low levels of 3 major neurochemicals involved in emotions have dominated for over 50 years. The monoamines (norepinephrine, dopamine, and serotonin) have been widely implicated in depression. All present antidepressant drugs affect changes in one or more of these systems, and there are multiple other lines of evidence of changes in the monoamine systems being associated with depression. The data are particularly voluminous for serotonin. But this theory was based on antidepressant response which at best is 40% to remission and 65% for response. Newer theories are emerging.

Inflammatory theory:
Low levels of chronic inflammation from either active illness (think Lupus or CAD) or persistent heightened level of corticosteroids from “stress” produce a toxic inflammatory milieu where neurodegeneration increases and neurogenesis is inhibited. It is thought that interleukin – 6, which interferes with serotonin metabolism might be the main culprit. The cytokines that are involved also create disruption of other end organs and create higher risk for heart disease as well as Alzheimer’s. Hypersecretion of cortisol can cause acute and more severe depression (Cushing’s disease).

Structural theory:
Depression is caused by abnormal changes in brain areas that can be identified premorbidly and are exacerbated in active illness. Atrophy of the prefrontal cortex, amygdala, and hippocampus and enlargement of the insula and anterior cingulated cortex done via MRI suggest this. Enhancing neurogenesis in those areas that are atrophied (BDNF infusion into the rat hippocampus-quickly alleviated depression) or altering GABA (neuronal excitatory/inhibitory) in the insula are areas of exploration.

Network hypothesis:
It is not specifically an altered brain area that causes depression but aberrancies in the tracts between areas. Diffusion tension imagery has revealed white matter abnormalities in the tracts between the medial prefrontal cortex, amygdala, and hippocampus. Glucose activity is reduced in the hippocampus and dorsolateral prefrontal cortex and increased in the amygdala, ventral striatum, and subgenual cingulated gyrus (an area that is stimulated in the new technique of deep brain stimulation). Sertoninergic agents reactivate a juvenile like plasticity in the neuronal tracts which if also stimulated by normal external phenomenon or psychotherapy leads to recovery. Depression is therefore a result of miscommunication and misinterpretation of various brain regions involved with interpreting emotions. (Castren- JAMA psychiatry 70(9), p 983, 2013)
The longer the depression the greater the chronicity of illness – if <6 months 60% chance of remission; if >24 months 10 –15 % of remission

Depression leads to future risk factors for: 1) More Major Depression, 2) Other Co-Morbid Psychiatric illnesses (60%) of the time – Etoh and anxiety disorders are most common; 3) cardiac events (cytokines and inflammation); 4) CVA’, Parksonism, dementias

Should we treat Major Depression more aggressively?

- How long does it take for permanent changes to occur?
- Is degeneration related to severity of symptoms?
- How much is reversible with remission or treatment? (psychotherapy and medicines both promote neuronal growth and increased blood flow)

Let’s not forget other etiological theories- these may or may not lead to biological changes that we have just explored.

**Psychological:** loss, abandonment, lack of nurturing, emptiness, anger turned inward, developmental arrest at a dependent stage with a disordered parent, low self esteem, failures, lack of self object stability and consistency(see following page)

**Environmental:** poverty, deaths, famine, wars, oppression, abuse, torture, drugs, learned helplessness, side effects of medications, chemical toxins, infectious diseases, medical conditions

**Conditions which may cause or mimic major depression:**

**Diagnosis: Depressive Disorder due to another medical condition**- once the condition is stable the depressive symptoms should dissipate –Epstein-Barr virus causing mono is typical example

- Infectious—(Mononucleosis, tertiary syphilis, toxoplasmosis, influenza, viral hepatic, HIV)
- Neoplasms—Abdominal malignancies (particularly pancreatic CA), brain tumors, lymphomas.
- Endocrine—Hyper or hypothyroidism/parathyroidism, hyper/hypoadrenocortical function (Cushing’s and Addison’s), diabetes.
- Metabolic/nutritional—Uremia, pellagra, anemia
- Neurologic—Frontotemporal dementia, Parkinson’s, Huntington’s, subdural hematoma, temporal lobe epilepsy, strokes, MS, head trauma
**Diagnosis: Substance/Medication-Induced Depressive Disorder**

- Medications—
  - Corticosteroids
  - Oral contraceptives
  - Antipsychotics
  - Interferons
  - Reserpine
  - Isotretinoin (Accutane)
  - Beta blockers
  - Central acting antihypertensives
  - All of the psychoactive substances: alcohol, cocaine, marijuana, opioids, sedatives/hypnotics, etc.

A Medical work-up is always a good first step – history, physical exam, labs – even if the symptoms are quite definitive for Major Depression. If there is an underlying medical condition it is usually quite obvious from other signs and symptoms - But remember to come back to the depression –“Whenever you hear hoof beats it’s most likely to be horses and not a Zebra”

In DSM 5 there is an attempt to add specifiers to diagnoses (like Major Depression) in order to better qualify them and possibly lead to different treatment – these include:

- With anxious distress
- With mixed features (anxiety and sadness)
- With melancholic features (mood worse in am, terminal insomnia, excessive guilt, marked weight loss, total lack of pleasure-anhedonia)
- With atypical features (wt gain, over sensitive mood reactivity, oversleeping, leaden paralysis- feel like can’t move arms or legs)
- With mood congruent psychotic features- about 10% of episodes-(hallucinations and delusions that have depressive content “I feel like I am rotting, the devil is telling me bad things”)
- With mood in-congruent psychotic features
- With catatonia
- With peripartum onset
- With seasonal pattern (20% of those of us in Chicago’s latitude have an element of this – worse in winter with wt gain and sluggishness and hyper and wt loss in spring)
# Psychological explanations for depression – from Comprehensive Psychiatry – Kaplan and Sadock

<table>
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<th>Proponents (Year)</th>
<th>Model</th>
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<th>Scientific and Clinical Implications</th>
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<tr>
<td>Karl Abraham (1911)</td>
<td>Aggression turned inward</td>
<td>Transduction of aggressive instinct into depressive affect</td>
<td>Hydraulic mind closed to external influences; non-testable</td>
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<tr>
<td>Sigmund Freud (1917)</td>
<td>Object loss</td>
<td>Disruption of an attachment bond</td>
<td>Ego-psychological; open system; testable</td>
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<td>John Bowlby (1960)</td>
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<td>Edward Bibring (1953)</td>
<td>Self-esteem</td>
<td>Helplessness in attaining goals of ego ideal</td>
<td>Ego-psychological; open system; social and cultural ramifications</td>
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<td>Aaron Beck (1967)</td>
<td>Cognitive</td>
<td>Negative cognitive schemata as intermediary between remote and proximate causes</td>
<td>Ego-psychological; open system; testable; predicts phenomenology; suggests treatment</td>
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<tr>
<td>Martin Seligman (1975)</td>
<td>Learned helplessness</td>
<td>Belief that one’s responses will not bring relief from undesirable events</td>
<td>Testable; predicts phenomenology; predicts treatment</td>
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<td>Peter Lewinsohn (1974)</td>
<td>Reinforcement</td>
<td>Low rate of reinforcement, or reinforcement presented non-contingently; social deficits might preclude responding to potentially rewarding events</td>
<td>Testable; predicts phenomenology; predicts treatment</td>
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<tr>
<td>Joseph Schildkraut (1965)</td>
<td>Biogenic amine (neurochemical)</td>
<td>Impairment or dysregulation of aminergic transmission</td>
<td>Testable; reductionistic; explains phenomenology and opposite episodes; suggests treatment</td>
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<tr>
<td>William Bunney and John Davis (1965)</td>
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<td>Alec Coppen (1968)</td>
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<td>I. P. Lapin and G. F. Oxenkrug (1969)</td>
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<td>David Janowsky et al. (1972)</td>
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<td>Arthur Prange et al. (1974)</td>
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<td>Larry Siever and Kenneth Davis (1985)</td>
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<tr>
<td>Bernard Carroll et al.</td>
<td>Impaired glucocorticoid and mineralocorticoid receptors</td>
<td>Testable; reductionistic; explains</td>
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Other Depression Disorders

**Persistent Depressive Disorder** (formerly Dysthymia): (2-3%) – 2 years of duration – depressed mood for most of the day on more days than not- course tends to be non-remitting
Two of the following Six:
- poor appetite or overeating
- low energy or fatigue
- insomnia or hypersomnia
- low self-esteem
- poor concentration or difficult decision making
- feelings of hopelessness
Has never been free of symptoms for longer than 2 months, no signs of other significant mental disorder that would explain symptoms, can have Major Depressive Disorder on top of this disorder

**Premenstrual Dysphoric Disorder:**

In the majority of menstrual cycles at least 1 of the following need to be present during the final week before start of menstruation:
  - Marked affective lability
  - Marked irritability and anger or interpersonal conflicts
  - Marked depressed mood, feelings, or hopelessness
  - Marked anxiety, tension, and/ or feeling on edge
At least 1 of the following also needs to be present
  - Decreased interest
  - Poor concentration
  - Lethargy
  - Change in appetite
  - Hypersomnia or insomnia
  - Sense of being out of control
  - Physical symptoms-breast tenderness, bloating, muscle pain
Must have a total of 5 or more of the above
Causes significant distress or impairment
Not an exacerbation of another similar disorder- i.e. Major Depression
**Bereavement/grief** (not classified as a psychiatric disorder)- For the vast majority of people a normal life reaction – it is expected that someone will feel empty and grieve the loss. They may have depressed mood, irritable, take some time off, not sleep well, etc. – These symptoms come in waves but the individual can still experience pleasure and joy. Much of grieving and mourning is cultural based- expectations are that by 3 months many of the symptoms have resolved and the person moves on with their life. However losses are a precipitant of MDD and one should not hesitate to treat as an MDD if symptoms are severe enough. Complicated (pathological) grief often involves many symptoms of PTSD on top of MDD and bereavement – these individuals get stuck on the loss and can’t progress – aggressive treatment is indicated

**Adjustment disorder with depressed mood**- some signs of depression that cause clinical concern but with an acute stressor that occurred within 3 months of the onset of symptoms. Criteria for other disorders are not met and the symptoms should abate by 6 months after the ending of the stressor. Usually brief therapy or social interventions are all that is needed

**Special Populations:**
Sometimes Major Depression Disorder may present in unusual ways that prevent doctors from making the diagnosis
Elderly: often masked depression – irritable, angry, don’t care, often somatic in presentation, don’t try, may be confused with dementia – depression is treatable

Teenagers: not familiar with what depression means or feels like, don’t trust adults, we may dismiss as adolescent angst, irritable common as sadness, look for behavioral changes – friends, grades, experimentation with drugs – look for withdrawal – engage with the family – controversy as to whether antidepressants are potentially harmful in younger patients – area largely unstudied.
**Bipolar I Disorder**

Individuals with Bipolar Disorder suffer a life long illness that can devastate their lives along with their families’ lives. Although the classic forms of Bipolar I are relatively uncommon 0.6-0.8% of the general population, if Bipolar variants are included the prevalence may be as high as 4-6% of the population which makes this a significant national health issue. To be diagnosed you must have experienced at least one Manic episode, although most commonly there will be episodes of Major Depression and other mood states in the history.

What is a Manic episode?

DSM 5 criteria:

A. A distinct period of abnormally and persistent elevated, expansive, or irritable mood present for most of the day for at least 1 week duration

B. During the period of mood disturbance at least 3 of the following are present (if the mood is only irritable then 4 need to be present)

1. Inflated self esteem or grandiosity
2. Decreased need for sleep
3. More talkative than usual or pressure to keep talking
4. Flight of Ideas or subjective experience of racing thoughts
5. Distractibility
6. Increase in goal-directive activities or psychomotor agitation
7. Excessive involvement in pleasurable activities that have a high potential for painful consequences – buying, speeding, sexual indiscretions, foolish business ventures

C. Marked impairment in functioning in job, social activities, or relationships with others or there are psychotic features

D. Symptoms not caused by a substance or medical condition

Individuals usually cycle clearly from Mania to Depression over the course of weeks to months. Some patients will end up with a Mixed Bipolar state where they will have symptoms of both mania and depression occurring at the same time. Often these individuals are very refractory to treatment. When you look at a cohort of bipolar individuals, a good 40-45% of time will be spent in a depressive phase, only 5-10% is spent in the hypomanic or manic phase, and the euthymic state occurs the other 45% of the time. About 2/3 of patients never make it back to their pre-morbid level of functioning. Even when these individuals are euthymic there is evidence of brain function abnormalities. There math, reasoning, and informational processing abilities are impaired. Verbal memory, attention, and executive functioning are less than baseline. Life span is decreased by 8-10 years primarily due to metabolic syndrome co-morbidities and a 15-20x higher risk of suicide. Their insight into their illness is always suspect.
leading to issues of substance abuse, non-compliance with medications and treatment, and potentially challenging live courses.

**Bipolar II Disorder** - the patient must have experienced a Major Depressive Episode but in addition at least one period of time where they have symptoms of hypomania which is defined as a distinct period of abnormally and persistent elevated, expansive, or irritable mood and increased activity or energy lasting at least 4 days in a row for most of those days. You need 3 or more of the same criteria as for a manic episode. This episode is a change from normal functioning of the individual. It is observable by others, but it is not so severe to cause marked impairment in functioning or require hospitalization. There is no psychosis present. If there is psychotic thinking it is diagnosed as Bipolar I. Although Bipolar II has less severity than Bipolar I the course of illness is just as severe as for Bipolar I discussed above.

If the patient meets criteria for a manic episode then they have Bipolar I that overrides the diagnosis of Bipolar II

In both cases – the manic/hypomanic cannot be caused by the physiological effects of a substance (either prescribed medicine or alcohol or drugs of abuse).

If the manic/hypomania persists after the substance is removed then it is considered Bipolar.

Psychiatrists feel that it is extremely unusual to have a Manic episode without having had at least one episode of Major Depression so always take a good history or the depressive episode may be arriving in the future course of the illness (after the first Manic episode).

**Cyclothymic Disorder** - 2 years of minimum of mood cycling but never enough criteria for Bipolar I or II or Major Depressive Disorder.

**Bipolar Unspecified** – a diagnosis that encompasses many potential patients who have some symptoms of mania and hypomania at times but never enough to meet full criteria. Anyone with significant mood fluctuations and irritability that are not induced by substances may fall into this area. This diagnosis is largely speculative but may encompass 3-4% of the population who often are never diagnosed.

Bipolar Epidemiology:

Bipolar I – lifetime prevalence 0.6-0.8%  
Bipolar II – lifetime prevalence 0.5-0.8%  
Cyclothymic Disorder/Unspecified Bipolar Disorder – lifetime prevalence 3-4%

Equal in men and women, age of onset - late teenage years – 1st episode usually begins with depression, childhood Bipolar is unusual unless there is significant genetic history, Highly hereditable – 65-85%; Concordant twins – 75-85%, Discordant twins – 15-20%, one parent with Bipolar – 10% chance of inheritance, two parents with Bipolar – 50% chance of inheritance.

Bipolar is unique amongst psychiatric illness as the only illness more prevalent in higher socioeconomic brackets. Having limited penetration of bipolar symptoms is a
highly sought after set of traits – less sleep, more energy, expansive mind, creative, talkative and these individuals tend to excess and are over represented in high functioning professions – physicians, researchers, lawyers, entertainers, business executives. There is a rich culture of creativity and madness.

Individuals with Bipolar have a much greater chance of becoming psychotic (hallucinations, delusions, disorganized thoughts) than those with unipolar depression.

The pathophysiology of the depressed phase of Bipolar looks surprisingly similar to Major Depressive Disorder on neuroimaging. The same cannot be said for the Manic phase that is very difficult to study (due to the pathology the patients are exhibiting) and has shown very mixed results other than an increased perfusion and glucose metabolism in all areas of the brain. Compared to controls, patients with Bipolar have enlarged ventricles and increased white matter hyperdensities. Due to the efficacy of lithium, many researchers have speculated on the neurochemical basis of mania, but nothing in conclusive. With recent biological treatments focusing on anticonvulsant medications the theories have moved to calcium and sodium channel regulation, secondary messaging signaling, and possibly some variant on serotonin dysregulation. Others believe there could be altered mitochondrial function, dysregulated dopaminergic/glutamatergic systems, and inflammation. A Nobel Prize awaits.

Separating Unipolar Major Depression from Bipolar Depression is paramount in that treatment and courses are very different and much co morbidity can be avoided. A recent research study, Almeida et al, British J of Psychiatry 2013, found that amount of blood flow to the Anterior Cingulate Cortex on a kind of fMRI called an arterial spin labeling was highly specific for Bipolar depression versus Unilateral Major Depression. Such biological markers in the future will enhance our diagnostic abilities. For now we rely on clinical findings.

**Hints that a person might be Bipolar when presenting with depression**

include:

- Early age onset(before age 20)
- Psychotic Depression
- 1st episode of depression is postpartum especially if psychotic
- Rapid onset and offset of depressive symptoms
- Recurrent depression with more than 5 episodes
- Bipolar family history
- Seasonal Mood Disorder
- Atypical Depression
- Hypomania associated with antidepressants
- Repeated loss of efficacy of antidepressant over time
- Trait mood lability, hyperthymic temperament
- Depression with mixed mood states
- Bipolar symptoms (hypomania) can at time be imitated by substances (i.e. cocaine, caffeine, prednisone) or general medical conditions (i.e. hyperthyroidism, closed head injury) and therefore a good medical work up is always a first step to diagnosis.
Suicide and Mechanisms of Diseases (all statistics are from American Foundation for Suicide Prevention web site)

Suicide is not only a tragic ending to one’s life but it affects a patient’s surviving family immensely as well as the caregivers taking care of the patient. Every religion (except for some peripheral cults) forbids suicide and makes strong statements against it as a way to exit life.

Suicide has been identified as a target of prevention by the surgeon general going back into the 1990’s. It is seen as a yardstick to measure the effectiveness of mental health services and interventions. The cost of loss of life secondary to suicide is upward of 34 billion in the US alone. Considering that it is a relatively common symptom in mental illnesses, it is essential that each physician be aware of the demographics and mechanisms that activate it in order to watch for red flags in a patient and the ability to do a good risk assessment. These skills need to be practiced by all physicians and not just Psychiatrists.

Suicidal ideation- a relatively common symptom (20% of adolescence will experience this at some point and about 10% of adults in any given year)- it is usually present when someone is under a lot of stress and feels there is no way out. This is illustrated by statements such as “I wish I were dead”, “The world would be a better place without me”, I wouldn’t mind if I developed cancer and died”. These thoughts are often fleeting but can begin to persist on a daily basis.

Suicidal intent- Thought have moved to thinking about how someone would commit suicide. Looked on websites, asked other, have begun to secure the mean to commit suicide or thought out when and how they would do it.

Suicide attempt- the actual carrying out of an act that could end one’s life. The degree to which the attempt might be lethal depends a lot on the means and the place of action. The ratio of attempts to completions is 12:1. There are about 460,000 attempts each year in the United States that are evaluated in the ED. There are probably many more that never seek treatment. Determining whether someone really wanted to die versus just relieve pain or get help is essential in psychiatric triage.

34% of those with suicidal ideation plan an attempt
And 72% who plan an attempt actually try to commit suicide

Completed suicide- 10th leading cause of death in US. – over 43,000 people in 2013.
51% firearms
25% suffocation- usually hanging
17% poisoning

There are probably many more deaths due to suicide that are not documented- single passenger accidents, drowning, accidental over doses, stigma preventing MD from listing true reason on death certificate because of the family desire or insurance reasons.
4:1 male to female for successful suicide
3:1 female to male in terms of attempted suicide

Self harm – no desire to want to die but instead an attempt to relieve pain or feel something real – but overlap does exist and needs to be explored

Epidemiology of suicide:
12.9/100,000 per year in the Untied States
Males – 19.9
Females-5.5
Caucasions-14.1
Asians-6.2
African-American-5.1
Hispanics-5.9
Adolescents- 10.5 (but often give the least warning and most impulsive)
Elderly- 17.6 (often sudden and told of a terminal disease)
Middle age (45-64)- 18.6 – now the top risk group demographically
Veterans returning from war- 34.9 – transition back makes them vulnerable

Suicide in an individual is hard to study because the actual chance of suicide is quite low but risk factors for potential suicide attempts have been well documented. The following is list with the most serious risk factor listed first:

- currently has a feasible plan in mind
- history of prior attempts
- psychosis (especially command hallucinations)
- high anxiety
- impulsivity
- presence of a mental disorder
- substance abuse (especially if intoxicated)
- hopelessness
- lack of support
- family history of completed suicide
- significant negative life event in the last 3 months
- presence of guns in the house

There are also protective factors that need to be considered when assessing for suicide but the presence of a protective factor does not make the patient immune from taking action.

Protective factors:
Supportive family
Strong faith or religion
Being on medications for the psychiatric illness

No harm - suicidal safety contracts have little to no value

**Pathophysiology:**
Lower serotonin receptor numbers
Lower serotonin levels in the CNS

Both of these are done post mortem which does not help us in clinical assessment- some recent PET scans claim that they can identify which individuals are more likely to resort to suicide attempts – this ability to predict will be very helpful

Lack of effective coping strategies, presence of impulsivity also have strong correlation with attempts. It is estimated that 70% of patients who ultimately complete suicide make the decision within that last 5 minutes- this makes intervention near impossible. Most people who call or show up in the ED for suicidal thoughts are wanting to live and are asking for help.

Prevention:
1. Ask- you will never put the thought of suicide into someone’s mind
2. Use behavioral incidents (as taught in interviewing skills) – details are important in such a shameful area- “What exactly were you thinking?”, “Did you actually go out and buy a gun? – What did you plan to do with it?”
3. Do a risk analysis based on each patient’s unique situation
4. Remove the means; make sure the patient is safe