Welcome to Clinical Connections, an educational series sponsored by the UPMC Institute for Rehabilitation and Research. My name is Ellen Whyte, I’m an Assistant Professor of Psychiatry and of Physical Medicine and Rehabilitation and our presentation today is Psychiatric Aspects of Medical Rehabilitation. Before we begin I do wish to make a few disclosures. I have received research support from the National Center for Medical Rehabilitation Research and the National Institute of Mental Health as well as several pharmaceutical companies.

Our learning objectives today are to discuss the reciprocal relationship between depression and disability, to identify key symptoms of major depression, to learn techniques for eliciting symptoms of major depression from individuals with brain injury and to differentiate major depression from pathological crying.

Our topic today is the relationship between depression and disability. As the slide tried to illustrate there is a reciprocal relationship between these two phenomenon in that individuals who have suffered medical illness and now have residual disability are at increased risk for developing major depression and other depressive disorders. Furthermore, individuals with depression are at increased risk for both medical illness and its related disability. For the person who already has a physical disability the development of depression can further worsen that disability. The relationship between disability and depression may be mediated by both biological and psychological factors which we’ll discuss in detail.
However I first want to point out that when I refer to depression I’m referring to major depression, which is the most severe category of depression as diagnosed by the American Psychiatric Association. When we diagnose major depression we look for the presence of 5 of a possible 9 symptoms, these symptoms are listed on the slide and we will discuss these in more detail later on in this presentation. However please note that these 5 symptoms or more need to be present most of the day, most every day for at least 2 weeks to meet criteria for major depression. In addition they must cause significant emotional distress or functional impairment. Please note that the causality of the depression is not part of the diagnostic process, therefore even if a clinician thinks they understand why a particular patient may be depressed, for example they just had a devastating stroke, understanding why a person has depression is not part of the diagnosis.

So how common is depression in adults and in the elderly? If we look at all comers, the prevalence of major depression in adults is 5 to 6%, among community dwelling elderly, those individuals age 60 and above who are healthy enough to live in the community, the prevalence of major depression is actually much less, only 1 to 3%. In both groups of individuals the prevalence of depressive symptoms not meeting the full criteria for major depression is greater.

However focusing in on the elderly, the prevalence of major depression as well as the prevalence of depressive symptoms increases based on where we recruit our older sample from. So whereas in the community only 1 to 3% of elderly have major depression, if we would sample from primary care
practices 10 to 15% of older individuals in the waiting room for example would meet criteria for major depression. If we then look at the acute inpatient setting also 10 to 15% would suffer from major depression, and if we look in a long term care facility or a nursing home, 12 to 16% would have major depression. One possible explanation for this increase in both the prevalence of major depression and depressive symptoms among the elderly as we go from independent living settings to more supervised settings is that these individuals have experienced more medical illness and disability, hence necessitating admission to an acute care hospital or into a nursing home.

While some of this may be a psychological reaction to disability there is evidence that there is a biological component potentially to this increased risk of major depression among individuals as they require higher levels of care. So for example the prevalence of major depression in stroke has been estimated to be as high as almost 30%. As is the prevalence in Parkinson’s disease has also been estimated to be as high as 30%. Similarly, major depression is very common both in traumatic brain injury and spinal cord injury.

So let’s spend a little bit more time trying to understand the relationship between depression and disability and why there may be a reciprocal negative relationship between these two phenomenon. In this slide we are summarizing those factors that may contribute to understanding this relationship. First and foremost direct injury to the central nervous system may predispose a person both to physical disability as well as depression. Specifically there is evidence that certain medical illnesses are associated with a higher rate of depression than would be expected. So for example, as mentioned
earlier, about 30% of individuals who have stroke go on to develop major depression, and similarly about 30% of individuals with TBI also develop major depression. However, about 15 – 15% of individuals will develop major depression after a hip fracture. This is notable because hip fracture frequently is a sentinel event for severe long term disability among the elderly, disability that may be considered equivalent to that of stroke or traumatic brain injury but is associated with less depression. And this evidence has been used to suggest that there is a direct biological connection between CNS injury and the development of depression.

Specific neuroanatomical structures have been suggested that are associated with both depression and disability, large scale community studies of older individuals have demonstrated that basal ganglia strokes are highly associated with the development of depression. Again, 40 to 50% of people with Parkinson’s disease can develop significant depressive symptoms, and indeed major depression can be the presenting symptom in Parkinson’s disease. In these cases major depression is presents first and then only in the subsequent year or so do the classical motor symptoms develop.

Additional evidence suggesting that brain injury can directly lead to depression is the evidence in late-life major depression that older individuals with major depression tend to have more white matter hyperintensities, especially in the frontal steroideal circuits than older individuals without major depression. White matter hyperintensities are felt to represent some clinical ischemic injury to the brain. In addition there is evidence from the stroke literature that those individuals who have had a stroke effecting the left frontal lobe have a higher incidence of depression than individuals who have
had strokes affecting other parts of the brain. Certainly a person who has had a stroke in another part of the brain other than the left frontal lobe may also go on to develop depression but the fact that the occurrence of depression is more likely after left frontal strokes seems to suggest that there is a particular mood regulating function in that area of the brain.

There are many other factors though besides biology that may affect or may explain the link between depression and physical disability. Certainly external factors may be a role. Individuals who have chronic financial stress, or poor social support may be at increased risk for developing depression, or for developing medical illness and subsequent physical disability. Certainly the depressed person may not have the motivation to engage in healthy health behaviors and therefore may develop medical illness and subsequent disability. And in addition if they are already physically disabled the depressed individual may not engage in those activities that would maximize their function.

In addition, the presence of major depression in and of itself increases the risk of cerebral and cardiovascular illness, both of which can lead to physical disability. For example, there is strong evidence that poststroke depression can increase mortality. The current slide is from a study done by Williams and colleagues, they reviewed the records of over 51,000 veterans who had a stroke and identified those individuals who developed depression after their stroke. While less than 5% of the stroke population in this study developed depression, those who did had a much higher mortality rate over the follow-up period than those who had not developed depression. The figure that is currently on the slide is based on the subset of this population in which individuals with poststroke depression
were matched to individuals who did not develop depression after stroke, based on their initial NIH stroke scale score. Again, those individuals who developed depression had a much higher mortality rate over the follow-up period than those who did not develop depression. Now clearly there were other factors associated with mortality including older age and burden of medical illness. But this slide nicely illustrates that poststroke depression is an independent risk factor for death after stroke.

It is not only major depression that is a risk factor for mortality after stroke, but the presence of any depressive symptoms is a risk factor as well. And this study by House and colleagues illustrates that depression as measured by the GHQ28 at baseline predicted mortality both at 12 months and 24 months. What I do not have a slide to show you is that this relationship between major depression, depressive symptoms and mortality is – and after stroke is parallel to that seen after both CABG and heart attack, so there is a strong relationship between depression and mortality from vascular diseases.

So what’s the possible mechanism? The presence of significant depressive symptoms is associated with several physiological changes that may explain this increased mortality in cardio and cerebral vascular disease. First off individuals who are depressed have increased platelet activation, and therefore their platelets are more sticky and likely to form a thrombus. In addition, during an episode of major depression people have decreased heart rate variability. This decreased heart rate variability is actually a risk factor for cardiac arrhythmia. During major depression people also have increased activity of the hypothalamic pituitary adrenal axis, and are bathing their body in higher levels of
cortisol which may have damaging effects. In addition, individuals with major depression have increase – have evidence of increased inflammation in their body. As we now know, inflammation is – has a pathogenetic role, excuse me, is involved in the pathogenesis of both cerebral and cardiovascular disease. Individuals with major depression may have increased sympathetic tone and of course they may also engage in poor health behaviors. Interestingly, remission of depression whether through pharmacological treatment or spontaneous remission or use of SSRI antidepressants are associated with normalization of these physiological changes suggesting that once a person’s major depression is treated the physical medical effects of depression are lessened.

Moving on, there is also evidence that depression can worsen physical disability as impaired motor function is a key feature in the diagnosis of major depression. Typically in a clinical setting we look for evidence of psychomotor slowing or psychomotor agitation as one of the possible symptoms of major depression. Psychomotor slowing may actually impair the person’s ability to function with a physical impairment. Indeed there has been a study demonstrating that there is increased swing time variability in middle aged subjects with mild to moderate symptoms of a mood disorder. This – in this study these individuals were asked to walk over a gait mat and their gait characteristics were recorded. This increased swing time variability was independent of any concurrent psychiatric medications these individuals were receiving. The authors of this study suggested that this change – these changes that they demonstrated in this mid-life sample was actually consistent with changes that are seen in early Parkinson’s disease and may actually contribute to falls in older depressed individuals. I should mention that it is commonly observed that in older individuals those who have
depression are more likely to fall. So I think it’s reasonable to believe that if there are these gait changes associated with the presence of depression, that if somebody already has a motor impairment affecting their gait the additional impairment caused by the depression could lead to additional functional limitation.

And then finally, we have to consider what many people would consider the obvious that having a physical disability can lead to depression. Clearly the answer to disability increases the risk of onset of depressive symptoms or major depressive episode, and in older individuals we expect 9 to 35% to develop major depression after a life disabling event. Interestingly physical disability predicts poor depression treatment outcomes and perhaps this is due to the fact that the disabled person may be more socially isolated, and those social contacts would have initially either protected them from depression or helped them recover from depression. In addition, the physical disability may be making – may be creating additional barriers that are preventing the individual from having the depression diagnosed or treated appropriately.

Besides this relationship between physical disability and depression, there really is a double whammy for the disabled individual in that depression can also impair rehabilitation. In the context of acute rehabilitation after an illness or injury evidence suggests that depressed older individuals have poor functional recovery compared to nondepressed individuals regardless of the medical diagnosis. And in fact having a major depression in the context of an acute medical event leads not only to increased mortality, but also increased institutionalization.
Let’s explore this a little bit further. In the context of poststroke depression we know that that, that depression in that context leads to worse rehabilitation outcomes, that individuals with poststroke depression have decreased post-rehabilitation scores on their functional independence measure and their FIM efficiency. This is even when accounting for baseline differences in functional impairment. We know that remission of the depression leads to improved functional outcomes. And this is actually independent of treatment. Sometimes depressions spontaneously resolve on their own, it does not matter how the person’s depression got better as long as it gets better at least to better functional outcomes.

Most interesting is that remission of the poststroke depression however does not lead to rehabilitation outcomes comparable to stroke survivors who never develop depression. And this finding was based on the, a very interesting study done in Italy in which they compared individuals who went through an intensive 2 to 3 month inpatient rehabilitation stay after stroke, individuals who were either those who did not develop poststroke depression, those who developed poststroke depression but were successfully treated with Fluoxetine, or those individuals who remained depressed. By the end of their rehabilitation stay those individuals who did not develop depression did the best in terms of functional independence with ambulation. Those individuals who developed major depression after stroke and did not get better did the worst, and those individuals who had developed depression but who’s depression had responded to treatment had a middle outcome between those who did the best and those who did the worst.
Let’s move on and review the symptoms of major depression. Once again, when we make the diagnosis it’s based on clinical interview in which we are assessing for the presence of 9 possible symptoms. For a person to receive a diagnosis of major depression they must either have sadness, low mood or loss of pleasure, also known as anhedonia. We will talk in a moment about how to assess all of these symptoms. To start off with sadness, individuals sometimes will not endorse being depressed or having depressed mood even though they may be having intense feelings of sadness. For some individuals this may be because they just lack the vocabulary to label their emotional state. However for some individuals, particularly older individuals they may not have the vocabulary to discuss their emotions because of a cohort effect. What I tell my trainees is that I’ve never met a World War II veteran who has ever endorsed being depressed, but I will – but they will endorse feeling blue or down in the dumps or feeling like they just can’t pull their boots up and keep on marching. So when one is assessing for major depression there does need to be some sensitivity for where the patient has been in life, and what is expected and normal for their stage in life either due to age or due to cultural background.

However even though sadness is the symptom we most frequently associate with major depression, a person does not have to have sadness to actually meet criteria for depression because the other symptom they could have is loss of pleasure or anhedonia. And this is a pervasive loss of enjoyment and motivation, which is present most days, most of the time for at least 2 weeks. This symptom can be somewhat hard to assess for an individual who has just had an acute medical event and say has just
been admitted to an inpatient rehabilitation facility. If you ask a person who has just had a stroke with left hemiparesis what do they enjoy doing they may say gee whiz, I love to play golf but I can’t do that anymore now, can I? So the goal of this interview to, to assess this symptom is to ask the patient if they are still enjoying things in their day to day life. So the person who does not have anhedonia, even if they are physically ill should still be able to enjoy simple things such as visits with their family or maybe watching their favorite TV show. And when they lose that, that would then meet criteria for anhedonia.

As a way of illustration, I frequently tell a vignette about a woman I saw in the hospital quite a few years ago who was lucky enough to have a window bed, it was a beautiful day with blue skies, lots of sunshine and I asked her what she could still enjoy, she said no, nothing. When I asked her if she could enjoy looking out the window at the beautiful blue skies, she said she couldn’t care if it was day or night, sunny or cloudy, it just didn’t do anything for her. So once again, for major depression a person must endorse either low mood or loss of pleasure most of the day, more days that not for over a 2 week period.

Because a person must have one or the other of the symptoms asking about those two symptoms is a good way to screen for the presence of depression. The PHQ2 is actually a way to – is actually a way to screen for the presence of major depression. In the example on the slide these questions are worded to ask about the patient’s entire lifetime. But these questions have been validated in the stroke population and when both questions are asked the sensitivity for detecting major depression is
100%; however the specificity is approximately 77%. But once again, if the clinician’s goal is to identify those individuals with major depression using the PHQ2 screening questions would be appropriate.

Moving on, the next set of symptoms we look at when making the diagnosis of major depression are the neurovegetative symptoms. Impaired sleep is one of the symptoms we assess and it can either be insomnia, not getting enough sleep, or hypersomnia, getting too much sleep. And when looking at insomnia we look at both difficulty falling asleep, the ability to stay asleep during the night and the time at which the person wakes up in the morning. Typically we say a person should fall asleep in 20 to 30 minutes after going to bed. An individual who is taking longer than ½ hour to fall asleep upon going to bed qualifies for having initial insomnia. Middle insomnia is defined when a person has multiple awakenings at night and has difficulty returning back to sleep. Early morning awakening or late insomnia is defined as waking up 2 or more hours earlier than anticipated and being unable to fall back asleep.

Impaired sleep sometimes is difficult to judge, especially if somebody is in an inpatient setting, because we know hospital units can be noisy, nurses, phlebotomists need to come in to draw blood, check vital signs during various times during the night. However even if a person has a likely explanation for why they are having trouble say falling asleep, we still count that towards the diagnosis of major depression. In general for these neurovegetative symptoms, if a person endorses a symptom, even if we suspect that it may be due to another medical or environmental cause we still
count that towards the diagnosis of major depression because in the context of endorsing sadness or loss of pleasure those neurovegetative symptoms will improve as the depression picture in total improves.

Another neurovegetative symptom we look for is impaired appetite. For most individuals this is loss of appetite with associated weight loss, although it can be increase in appetite as well. Individuals can complain of low energy and feeling fatigued. And then individuals can have either psychomotor agitation or retardation. Some individuals with depression feel very restless, they have difficulty sitting still and may actually get up and pace. On the other hand, individuals with major depression may feel very slowed down, that they are thinking slower, that they are moving slower and it may actually be observable to the interviewer as well.

The occurrence of psychomotor retardation in major depression can actually be quite dramatic. I think every psychiatrist has a story of having admitted a person to an inpatient unit, usually an older person with severe depression who had recently been diagnosed with Parkinson’s disease, and that – maybe even started on medication, moving very slowly. And then after the depression is successfully treated the person’s motor symptoms resolve, they no longer need the medication for Parkinson’s disease, and they are walking very well again.

Another symptom people report in major depression is poor concentration. This is also sometimes seen as difficulty making decisions. The patient with major depression may report that they have a
hard time focusing on what they are doing. For example, somebody may love reading but have such poor concentration that they can’t even read a short newspaper article anymore. This symptom may also be expressed as inability to make decisions. And this is a subjective sense that the patient has.

Another symptom of major depression we look for are feelings of worthlessness or guilt. Individuals when they have major depression frequently lose their sense of self esteem, they feel worthless, they feel that they have failed their family and friends, or they may actually start feeling guilty about either current or past events, things that they did or things that they should have done. Once again there is a cultural effect that one needs to be aware of when assessing this symptom. In the older individual they did not grow up in a time when people were freely talking about self esteem. So as I tell my trainees, asking a World War II veteran how their sense of self esteem is, is probably not going to get you too far because the person really doesn’t understand the concept of self esteem. But if they are asked about how they feel about themselves, do they still feel they can be still be a good husband, father and grandfather, that would be another way to get around this – get around any cultural barriers to assessing this symptom.

In the medical setting where people may have numerous medical issues going on that may make it difficult to assess the neurovegetative symptoms, this is a very important system – symptom to be sure to assess. As a way of illustration, I once was asked to assess a gentleman on an inpatient rehabilitation unit after stroke, the staff was wondering if he was developing depression. He wasn’t and in particular when I asked him about his sense of self esteem or sense of worthlessness he
admitted to feeling concerned that he was going to be a burden on his wife, but he still felt that he would be able to find some way to contribute to the family.

In contrast, I was once asked to consult on a lady who had had a relatively minor stroke, she had not required inpatient rehabilitation and she was having difficulty returning to the job, a job as a teacher and when I asked her about this symptom she completely became very distraught and said that even though she had – even though she realized she only had mild residual clumsiness in her left hand as a result of the stroke she felt that she could never teach to the same level she had before and felt that she was worthless and therefore it wasn’t worthwhile trying to go back to the job.

And finally there is, one has to talk about thoughts of death and suicidal thoughts in the context of major depression. When we are assessing the symptom we are not just assessing for suicidal thinking, but we are also assessing for if a person has been thinking more about death in general or has become obsessed by death. Just having more thoughts about death in general would quality a person for meeting this symptom criteria, a person may be spending more time thinking about their own death, death of loved ones, funerals they may have attended or they may actually have progressed to having suicidal thoughts.

I’m also often asked how does one judge this symptom if somebody is elderly and perhaps only has a few years left to live, or if somebody has severe medical illness. This symptom really focuses on premorbid thoughts of death, it is totally understandable if a person in their 80s or 90s or who has
severe medical illness has thought about death, but a person should be still focused on living even if
they are aware that death may be soon for them. As an example I was once asked to see a delightful
lady in her 90s in the nursing home who had a prior history of depression. When I was seeing her she
was not currently depressed. When I asked her this question she told me that she acknowledged the
fact that she could die at any moment, she was at peace with the fact that she may die but she was
really looking forward to bingo next week. And she hoped she would make it to bingo, but if she
died that night she would be okay.

A few words about suicide, suicide is unfortunately the eleventh leading cause of death for all
Americans, despite what you hear on the news most Americans are more likely to die from suicide
than from homicide. It is estimated for every 2 homicides in this country there are 3 suicides. Suicide
is a very complex behavior, but almost all individuals who kill themselves have a diagnosable
psychiatric illness or a substance abuse illness or both. And of those individuals who kill themselves
having a depressive diagnosis is the most likely psychiatric diagnosis.

A few words of who is most likely to actually die by suicide in this country. And I ask you to look at
this slide and make a decision of which age group you think is most likely to die by suicide. Is it
males from the age of 18 to 25 who may be involved in substance use and risky behaviors? Is it girls
age 14 to 21 who may be having a lot of difficulties with image, with self image? Is it males over the
age of 75 who may be experiencing significant medical illness or is it women age 60 to 75 who may
be experiencing widowhood and other life transitions? Unfortunately it’s actually adult men age 75
and older who have the highest rate of successful suicide in this country with a rate of almost 38 deaths per 100,000 population; now obviously this is not the number one cause of death in older men, but it’s very striking nevertheless.

Interestingly adult white males have the highest rate, their cause of death is usually by firearms. And that is why they are among older individuals there is usually one completed suicide for every 4 suicide attempts. In the younger age population they actually have a lot – significantly more attempts per one completed suicide. White older men have the highest suicide rate, while older women also have a high suicide rate. African-American older women have the lowest suicide rate. Persons of Hispanic or Native American origin, their suicide rate is unfortunately increasing and coming close to that of the Caucasian suicide rate.

Traumatic brain injury appears to be a risk factor for suicide. In a study that interviewed individuals with chronic brain injury almost 25% endorsed suicidal thoughts and approximately 18% endorsed having had a suicide attempt since their traumatic brain injury. The risk factor most likely associated with having suicidal thoughts was the development of new psychiatric or behavioral problem after the traumatic brain injury. Or put in another way, having had a psychiatric or behavioral problem prior to the traumatic brain injury did not appear to be a risk factor for suicide thoughts.

When assessing a person for depression it’s always important to fully assess their suicide risk, individuals who go on to attempt suicide just didn’t wake up one morning deciding to kill themselves,
instead they likely went through this continuum of thoughts starting off with having a preoccupation with death, that once again we commonly see in depression. They may have gone on to evolve a passive death wish in which they were hoping they would die or maybe even praying to God to take them soon. They may have developed thoughts then of harming themselves and these thoughts may have been either thinking about an active plan of hurting themselves, such as shooting themselves or jumping. On the other hand, they may have thoughts of harming themselves by discontinuing medication or stopping eating. Individuals then go through a phase in which they plan their suicide, which could include getting their affairs in order. Therefore when I see a patient with depression I want to know have they recently written a will, have they started giving away their valuables, etc. And then, only then will a person actually proceed to actually make an attempt.

It’s important to assess this entire continuum. A person if asked – who is being assessed for depression, if they are asked if they are planning to hurt themselves could honestly say no, but they may already be at the passive death wish stage where they are praying to God at night to take them. To truly know where a person is on the continuum and truly understand their risk for suicide it’s important to ask about all steps in the continuum.

So once again major depression is a clinical diagnosis based on eliciting 5 out of 9 possible symptoms. A person must have depressed mood or anhedonia to meet criteria, and they must be bothered by the symptoms most of the day, more days than not for at least 2 weeks with associated emotional or functional impairment.
How does one obtain this information if dealing with a patient who either has a language impairment or other cognitive impairment? Well there is a couple of techniques we can use. First and foremost is relying on informants, including family member and clinical staff. Especially family members, they know this person the best and they should be able to observe and report whether there is any outward change in their mood. Other information that informants can give us though can really let us know about those neurovegetative signs of depression. Nursing staff can tell us whether a person is eating, whether they are sleeping, a rehabilitation staff can tell us whether or not a person is able to concentrate on activities, whether they give up easily and also whether – do they seem to enjoy some of the rehabilitation activities and camaraderie.

It’s also important to observe nonverbal behavior. Just an example I was once asked to see a lady for possible depression in the hospital, and when I came into her room there she was in the middle of the day sitting in a dark room with the shades drawn, TV off, staring at the floor. With her nonverbal behavior I think it was a big indication she may be experiencing depressed mood.

For the person who is having trouble communicating or maybe understanding spoken language we can try to ask about the questions of depression using simple questions that could be answered with either a yes or a no. Or we could use categorical responses, we could use cards with key phrases to help prompt the person to – with the question or to help them respond to the question.
There is also visual analog mood scales. A visual analog scale is usually a 100 millimeter line which is anchored on one end as the worst possible score and the other end as the best possible score and a person is asked to put a hatch mark on the line to indicate what their mood is like. For example, in stroke a scale very similar to this has been validated in which that 100 millimeter line is anchored by best mood and worst mood and the presence of a smiley face and the presence of a face that’s frowning. And again, the person is asked to put a mark to indicate how close they are to the best point or the worst point their mood has been.

Interestingly the scale has also been validated for watching change in mood. By using a ruler a person could actually measure how many millimeters up from the worst the person had indicated their mood was and then over time assess how far that person’s mark is moving, hopefully in the direction of feeling better. Sometimes it’s hard on the clinical unit to have a ruler available, and at times I have used a system similar to this where I’ve just taken a piece of paper and drawn a smiley face, a neutral face and a face with a frown and asked the person to indicate which symbol they feel their mood is, is closest to.

One of the big differentials to major depression in neurological disorder is – disorders is pathological laughing and crying, and this syndrome is diagnosed as emotional expression that is characterized by the sudden onset of crying or laughing that the person feels is not under their control, and which is not an expression of depression or grief. So for example in pathological crying, which is the more common circumstance a person may start crying uncontrollably any time they feel a strong emotion,
independent of whether that emotion was a sad or happy or neutral emotion. And of course this is diagnosed in the absence of a major depression or a grief reaction that would explain the excessive crying.

Pathological laughing is a less common phenomenon but it’s very similar in terms of a person having a sudden outburst of laughter that they feel is not under their control which occurs in unusual situations, whether they are confronted by a happy or a sad emotion. The key issue here is that it’s unusual – that this pattern of emotional expression is unusual for the person and they feel like they have no control over it.

In stroke we estimate about 20 to 25% of individuals have some element of pathological laughing or crying in the immediate poststroke period, some people will have spontaneous recovery but even by 12 months after stroke approximately 10% of individuals will still report some element of pathological emotional expression. While pathological crying, as an example, is not major depression it is – this phenomenon is responsive to low dose antidepressive therapy. Studies have indicated that both selected Serotonin reuptake inhibitors and Tricyclic antidepressants are useful in suppressing the emotional outbursts and giving the person a sense that they have control over their emotional expression. And interestingly, while antidepressants usually take 2 to 4 weeks to start working in the context of major depression, in the context of pathological laughing and crying the onset can really be in 24 to 48 hours.
Another major differential we need to look at when considering major depression in neurological diseases is apathy. And apathy has been defined as a lack of motivation that is not attributable to intellectual impairment, emotional distress of diminished level of consciousness. And this lack of motivation is – has to be relative to the patient’s previous level of functioning or standards for their age and culture. And evidence for lack of motivation would include lack of goal directed overt behavior, lack of goal directed cognition and lack of emotional concomitance of goal directed behavior.

So for example the apathetic person may not actively participate in their rehabilitation program, they may not think ahead in terms of what they need to do for example to get to their rehabilitation program that day and they don’t act – they don’t have a sense of positive anticipation. So one of the questions I use, especially as we are getting close to the holidays at the end of the year is I ask people are they looking forward to the holidays and getting together with family. The truly apathetic person would say that no, they are not looking forward to, to any of that and not looking forward in either a positive or a negative way. Again, apathy is not better – cannot be better accounted for by the presence of a depression or a psychiatric disorder. And the person who is apathetic does not have emotional distress about their absence of goal directed behavior. Usually it’s their families and friends who are very distressed that the person seems disinterested. Treatment of apathy unfortunately we do not have any good treatments. Certainly when faced with a patient who has significant apathy a clinician may actually try to aggressively treat them for depression as these two syndromes do tend to
co-occur and the hope in that case is that any depression will resolve and the person’s level of motivation will actually improve.

Going on to the treatment of major depression, listed on the slides are the most commonly used antidepressants currently used to treat major depression. Most people are familiar with Prozac or Fluoxetine, which is a member of the SSRIs or Selective Serotonin Reuptake Inhibitors. The SNIRs, or Serotonin Neurogenic Reuptake Inhibitors were, were developed after the SSRIs and are commonly used at this point. Two other commonly used antidepressants are Remeron, which also affects the serotonergic system and Wellbutrin Bupropion which affects several neurotransmitters including dopamine.

A few words about pharmacological treatment of depression, most antidepressants are considered to be equally efficacious, so when choosing an agent most clinicians will base it on the safety and tolerability of that drug for the specific patient, patient’s preference or past experience with antidepressants and cost. In terms of neurological disease there has been some concern about whether or not medications work for major depression in the context of neurological disease, most evidence suggests yes. There have been several major studies in poststroke major depression that indicate antidepressants from several different classes are efficacious. It is unclear whether - the evidence is unclear whether or not medication is beneficial to individuals with traumatic brain injury associated depression, however the studies in this population are very limited and as a result it’s hard to draw any firm conclusions. So once again, when treating depression in the context of a
neurological illness, use – use of medications is appropriate, they are all equally efficacious. The agent is chosen based on tolerability. However all medications take a while to start working. We normally say it takes about 2 to 4 weeks to start seeing a benefit from an antidepressant and the full benefit may not be seen for 8 to 12 weeks. However side effects usually start early. So it’s very important to educate patients about potential side effects to ensure compliance.

When treating a patient for major depression frequent contact is needed, we need to constantly reassess for suicide risk because it takes 2 to 4 weeks for these medications to start working. During that time the person’s depression may actually worsen. We need to monitor for side effects and we also need to monitor and encourage compliance. Generally speaking if we do not see moderate improvement by 4 to 8 weeks of treatment we will consider a medication adjustment. And it’s important to aggressively treat until all symptoms of depression have resolved. Evidence indicates that even having low level residual symptoms puts a person at risk for having the major depressive episode come back.

One question I’m frequently asked is how long does a person need to stay on medication when using pharmacotherapy to treat major depression. The answer is a little unclear. It depends what population we are looking at. For adults the recommendation from the American Psychiatric Association are, are based on the fact that for many people major depression is a recurrent illness. So then after having a third or more lifetime episode, a person has a greater than 80% risk of having an additional episode. So for individuals who have had 3 or more lifetime episodes the recommendation
is that once a person gets well using pharmacotherapy the person should stay on a full dose of that antidepressant long term to prevent the emergence of another episode of depression. If it is a person’s first lifetime episode of depression and it was uncomplicated, meaning there was not a suicide attempt or – and that the episode was not associated with significant functional impairment such as losing one’s job, it’s reasonable to consider tapering and discontinuing the antidepressant after a period of time. However an individual who plans to get off an antidepressant after their first lifetime episode needs to stay on the medication for after – for at least 6 months after they have been symptom free in order to decrease the risk of having a second episode. For a person who has had 2 episodes, there is no clear guidelines of what to do; however, if ever a person has an episode that was associated with a significant suicidality or significant functional impairment consideration should be given to long term antidepressant treatment.

For older individuals there are no specific published guidelines, but evidence suggests that older individuals should remain on antidepressant treatment. In a study done her at the University of Pittsburgh by Chip Reynolds, he demonstrated that older individuals with either a first ever or a recurrent episode of major depression those who received study medication, excuse me, those who received antidepressant for 2 years were much less likely to have a recurrence of their major depression than those individuals who received a placebo. And in fact, to prevent one episode of recurrent depression in one patient, only 4 patients needed to receive long term antidepressant treatment. It is unclear what should – what we should do after that 2 year period. In addition, in this study subjects who had the greatest medical burden actually received less benefit from staying on long
term antidepressant treatment. And then what do we do for patients with brain injury who have been successfully treated for major depression? Unfortunately we really don’t know. There is no evidence, no research studies that have been completed that guides us in that decision making process.

When talking about treatment of depression of course medications are an option, but so is talk therapy. Psychotherapy is actually appropriate as a monotherapy for mild depressions and is very appropriate as an adjunct to medication in individuals who have moderate to severe major depression.

In this day and age most psychotherapies are structured and brief, lasting only 12 to 16 weeks. All the – these structured brief psychotherapies which I will discuss in a moment are manualized, and walk a patient through various stages of recovery and these therapies tend to be reimbursed by insurance carriers. There is also evidence suggesting that these structured brief psychotherapies can still be very effective, effective even in individuals with acquired brain injury.

The main types of brief structured psychotherapies that are currently being used include cognitive behavior therapy. In this brief therapy patients are learned to identify and correct core beliefs that have led to or have reinforced their depression. For example, a person may have a negative belief about themselves and this therapy challenges that negative usually automatic belief.

There is problem solving therapy. Frequently in depression we see that individuals have trouble with problem solving. They fail to take into account all aspects both positive and negative with evaluating a situation and therefore they limit themselves to the possible alternatives to solve any problem they
are facing. In problem solving therapy the patient is taught the process of solving problems and as they get better at problem solving their learned helplessness is also reduced.

Interpersonal therapy is another one of the brief structured psychotherapies, it is specifically targeting alleviating depressive symptoms by focusing on one of four areas, role transitions, role disputes, grief and interpersonal death since that may lead to social isolation. Interpersonal therapy can be particularly appropriate for the individual who has just had a major medical event and now has disability as they are likely dealing with issues of role transition, for example they may not be able to return to work, they may be having role disputes at home with their spouse and clearly they may also have grief related to their losses.

Depression really can be an illness that affects the whole family, even though one individual may be identified as the patient, especially in the setting where we are dealing with a person with disability and depression who has an identified caregiver, particularly a spouse I think it’s very important to address any depression the spouse may be dealing with. In addition, it’s important to address any frustration the spouse or caregiver may have, they may be feeling particularly hopeless as they feel that their loved one who they wish to help may be less motivated or less engaged in their rehabilitation process as a result of depression. So educating the caregiver about depression is very important.
Even for the non-psychiatrist or non-psychologist clinician has an important role to play in treating depression in our patients who are undergoing rehabilitation. Frequently the depressed patient cannot feel any hope, they only see gloom and doom, they cannot see the light at the end of the tunnel, and one of the important roles that the clinician can play is providing the patient hope or being the hope proxy for the patient to encourage them to stick with treatment, treatment not only for their depression but also to engage in their rehabilitation from their illness so that the patient can keep on going while we wait for some of these depression treatments to work.

At this time I will end the presentation. Thank you very much.