[MUSIC PLAYING]

EVA SZIGETHY: I'm Eva Szigethy and I'm a child and adult psychiatrist. I have been here with the adult unit since 2006 running the visceral inflammation and pain center, the VIP center.

And I have been a psychiatrist in gastroenterology for almost 20 years. Starting at Boston Children's Hospital, moving here to the Children's Hospital here, and then Dr. Ruggiero lured me away to take care of the adolescents from the adult side. So now I take care of children and children at heart, the adults in our unit.

All right, why are you getting a state of the art talk on psycho-behavioral approaches to GI disease? Because you all practice psychosocial medicine, you just don't call it that. So my potential conflicts but none of them actually do conflict with anything I'm talking about today.

So I'm going to give you the punchlines, the brain-gut axis rules. You have patients-- whatever your subspecialty interests are in GI, your patients have psychiatric disorders and they're higher than they are in most other chronic diseases. We have evidence, not the same rigor of evidence that any other field of medicine does, but we have randomized control trial support for both the medications we used as well as the behavioral interventions. And I'm going to tell you about that.

And then I'm going to end with the new model of how do you integrate behavioral health into GI care and tell you a bit about our IBD medical home and really from the behavioral perspective. All right, so everybody here, hopefully, buys that we have constant communication between our brain and gut and that our GI tract is our most innervated organ of the body.

And in fact, it contains more neurotransmitters than the brain. So when I prescribe an SSRI, I'm thinking about its effects on the gut sometimes even more than I am for whatever I'm starting it for in the brain, both side effects and actually therapeutic effects. And in fact, our gut has been called the second brain. It actually is what kicks in first when we go into survival mode.

So any of you when you get nervous, you feel it in your gut-- butterflies in your stomach, a gut feeling. There's a reason that we have so many metaphors from the gut. It really is sort of our most primitive and accurate signal that that we should be alerted to something.

And then the growing interest in psychiatry as well as GI is the role that the microbiome plays in modulating what's happening in the brain. So a lot is going on. Really showing, for instance tryptophan metabolism, which is the precursor to serotonin and the HPA axis regulation are sort of the areas that we are focusing a lot of research effort on.

So again, reiterating that and confirming that bi-directional communication. So when we're stressed for any reason, we feel it in the brain. But through many different cascades we feel it in the gut. And this is just really to illustrate the complexity of that.

And that's both for our functional GI disorders but also for our inflammatory bowel disease, chronic pancreatitis, hepatitis. Really take your disease of choice-- celiac disease, and really everything I talk about will apply, even if I'm drawing from examples from certain GI subspecialties.

So what happens with the brain-gut communication? So the brain can amplify signals from the gut. So whether that's pain, whether that's nausea, the brain is really what determines the degree of suffering that somebody feels. Even when there's a real physiological stimulus, a real inflammation, real pressure in the gut, real abnormalities and motility, the brain is really what decides how much you take that in and then what you do with that signal.

Many different mediators of that relationship between something happening in your gut and how your brain perceives it. So luckily, we have protective factors-- spirituality, strong social support, active coping-- so people who are active problem solvers in their life actually do better even if they have a GI disorder, and general optimism.

We are increasingly finding that we have risk factors. Now, I purposely started with non-psychiatric disorders. So these are all phenomena, and we'll go through this list, that we can study. We can quantify the way that we cope, the way that we perceive.

Catastrophic thinking is the nobody, everybody, nothing, never kind of thinking-- the extreme negativity. Cognitive inflexibility is that ability to lock-on to an idea and the inability to move away from it, even if you're presented with information. Let's say your doctor says, this medication-- this biologic is really going to help you. No, it's not. And patients really get locked-in. And again, it's outside of psychiatric disorder.

Avoided coping-- so people who really shut down and run from their problems or refuse to get the care they need because they're avoidant. Self-blame-- alexithymia is the inability to recognize your emotional state. So when you ask these people how they feel, they say I don't know.

Lots of literature, especially in IBS that the degree of severity of IBS is inversely proportional to the degree that you can verbally express your emotion. So it's like these people are letting their guts express their feeling in somatic signals and they can't express it verbally.

Trauma-- so trauma now is anything that threatened your integrity that made you feel extremely afraid. So it does not have to be life threatening. It can happen any time in your life. Obviously, the earlier it happens in somebody's life the more lasting effects it has. And people who've been traumatized in any way tend to just be more hyper vigilant in general to what happens in their body, especially signals of danger and so that also goes for the gut.

And with that comes an anxious hyperarousal. Some people have that as a trait, so they're born being sort of just more easily autonomically sympathetically activated. And some people, it happens because of environmental trauma.

And then depression-- so if we take one disorder that has the biggest risk of amplifying the signal in the gut, it's actually patients who are depressed. We think it cuts off their frontal lobe ability to modulate their emotional state, their physiological state, and sort of their thinking. And that then translates to a louder GI signal, whether that's nausea or pain.

We're going to focus on three disorders today. And the list could go on and on-- there's nothing protective having a GI disorder and getting a psychiatric co-morbidity. Anxiety, depression, and PTSD are probably the ones that have been most studied and have the most, I would say, negative influence on what's happening in the gut.

So what do we do when we do a psychological psychiatric assessment? The team that you have here, by the way-- so I'm a psychiatrist. I have another psychiatrist, Bob Howland, in the VIP center. We have social workers and we have psychologists.

And everybody on my team is trained up in med-psych so they know their GI disorders. They know how to assess all of those coping strategies that we talked about. They know how to assess in a more structured way for psychiatric disorders, using the criteria that we use, the DSM.

And they're really good at assessing people for their readiness, motivation to change-- so their disease self management skills. So it's not just that you have a behavioral clinic embedded in GI but you really have specialists in GI in that clinic. Because I'm going to encourage all of you to think about referring your patients there. We accept anybody who has seen a gastroenterologist at least once a year and you all count. So they're welcome in the clinic.

So let's go through anxiety. So really, we look at patient's symptoms always from a how much it impairs their functioning lens. So you could be extremely anxious and handle it well. I might be extremely anxious. I'm not but I could be and I could be masking it right now. So it's really the--

[STUTTERING]

--if my anxiety I'm impaired, more concern. So again, mild anxiety-- and now we're really thinking about our GI populations here. So you can have age appropriate or situation appropriate worry. You could have an adjustment disorder, which is just sort of mild anxiety in the context of a stressful situation. Pretty much dissipates when the event goes away.

A lot of our patient's anxieties are picked up in their first five years. So that's when our-- all of us, our coping style's imprinted the first five years of our life. The rest of your life is practicing. So if you had parents that modeled anxiety or avoidance for you, even if you didn't have the genetic propensity for that, you're going to have a slightly higher risk of being more anxious in situations because that's sort of what you picked up as the norm for functioning.

Generalized anxiety disorder, and we'll go into little more detail what that is, is the most common disorder. The hallmark symptom is excessive worrying and worrying that leads to avoidance.

Panic attack-- so you can just have panic. And panic attacks happen spontaneously. There often is not a trigger and it becomes a disorder if that panic gets so extreme that you avoid situations that you think are linked to that panic. The classic example is elevators. So people who can't tolerate elevators because they've had a panic attack usually in that situation and then they avoid elevators.

Illness anxiety disorder-- so our somatization disorders have been reclassified into two broad classes-- somatic symptom disorder and illness anxiety disorder. Somatic symptom disorder is when you have symptoms and you're extremely anxious. Illness anxiety disorder is you don't have any somatic symptoms but you're always worried about getting sick.

And I put them here even though they're classically not anxiety disorders. In the reclassification they're somatization disorders. We deal with them the same way. It's really an anxiety link that we're trying to dampen.

Severe anxiety, obsessive compulsive disorder-- obsessive compulsive disorder needs specialized help. These are people who have thoughts that really get stuck-- they play over the same thoughts over and over and the only way they let themselves out of that obsessive thought cycle is by doing rituals. They can be counting, they can be hand-washing rituals. And those rituals take two to eight hours a day. So I have patients with OCD who's basically entire day is OCD.

Where we find it in our patients is the excessive bathroom use. So you could have realistic worry. I mean, if you have anything that causes diarrhea-- we've all had a GI flu. You want to know where the bathroom is and you're going to somewhat plan your day around it. It's when that takes on a life of its own, even when there's not necessarily a GI drive. And we have patients that spend hours a day basically with bathroom rituals. That is when we call it obsessive compulsive.

And post-traumatic stress disorder-- now, we've had a trauma. And that trauma again, revs us up in terms of a hyperarousal state. And that then basically we can have nightmares, we can have flashbacks to whatever the event is. But we have more and more recognized mood changes-- the ability, the anxiety. And we have that hyperarousal of the sympathetic nervous system that prevents us from sleeping well, that doesn't allow us to relax easily.

We basically keep ourselves up in a hyped state. And in that hyperarousal state, our immune system takes a hit. Because if you don't let your parasympathetic have a chance at the wheel in the driver's seat, you're always going to be in a high cortisol state, relatively speaking. And so when your body needs to mount a response, let's say you're having an IBD flare or some other flare, you just don't have the juice there to do it and the immune system doesn't function as well.

So post-traumatic stress disorder is again, any amount of post-traumatic stress disorder-- And really that's having that set of symptoms for longer than six months is something that we really want to pay attention to. Because all of these things-- I don't want to say they're treatable but we can dampen them all down to background noise level where they're neither impairing function or having an adverse effect on the GI tract.

These are our changes. So DSM-IV became DSM-5 about a year and a half ago. And for the purposes of a gastroenterologist, you really don't have to re-certify on your boards like I do a year from now and know all of these new names. But just in a general way, basically we did do a lot of reclassification if you start seeing it in our notes.

And that really was to try to fit the symptom clusters. Because really, we still diagnosis in psychiatry by if you meet a certain amount of symptoms and types of symptoms over a certain period of time to fit what we are learning about the biological etiology of those symptoms.

So generalized anxiety disorder-- excessive anxiety and worry at least 50% of days in at least two life domains and that's been sustained for three months. And that worry is associated with any of these three-- some restlessness or feeling keyed up or on edge, being easily fatigue, difficulty concentrating or just your mind going blank, irritability, muscle tension, and sleep disturbance.

So all of us have had days where we're described like this. Possibly, under really stressful periods, we've all had even a month that we're like this. So the key here is that these symptoms lead to marked avoidance or marked impairment in functioning. But I think it's so important to see that it's not so hard to reach this diagnosis and it can be reached either when you're having a disease flare or when your IBD or your GI disease is in remission.

The somatization disorders I covered. So we've clumped a bunch of them in to basically the two main ones-- somatic symptom and illness anxiety disorder. Conversion disorder is still a conversion disorder just renamed to functional neurological symptom disorder. Because most of conversion symptoms, like pseudoseizures and motoric kinds of hypermotoric symptoms that patients have, are in that neurological realm.

And factitious disorder is another disorder where basically people's minds are playing tricks on them and they really believe they have a disease when they don't. And really the rationale, for all of this including the somatoform disorders-- was the rationale to change from DSM-IV to DSM-5 was really to get a better ideological grip here when we're talk about medically unexplained symptoms-- what we're talking about here.

Again, we talked about somatic symptom disorder. You have symptoms and it's disproportionate, persistent, or excessive worry about those symptoms or an excessive behavioral response. Often attention seeking in medical settings or an emotional response. And again, typically this is going on for at least six months.

Now somatization disorder has been studied in patients who have irritable bowel syndrome or really any type of functional GI. So that can be nausea, that can be pain in 30% to 42%. So high rates of somatization. Again, I always think of this as somebody whose brain sent their anxiety response straight to their gut and they they're expressing that angst in the gut. And that's what we treat.

And very high rates of post-traumatic stress disorder and somatization disorder in our patients. Now something to be aware of is even as a psychiatrist, you don't really get an honest trauma history from most patients until they really have a trusting relationship with you. You often, even when we ask in our intake, we get a no, nothing traumatic happened. And then it comes out three, four months later when we get to know them.

So just because you don't see it, doesn't mean it's not there. And if things aren't getting better, either the IBS or for us, anxiety, depression with the common things that we do-- and in fact, the harder we try, the worse people get. We usually are bringing that trauma differential up in our differential diagnosis very quickly. And then really working in ways in eliciting that so that we can treat it.

PTSD-- so you have to have an exposure to a trauma; one or more intrusive symptoms-- persistent avoidance of any kind of trauma associated stimuli. The change in DSM-5 is you also now meet criteria for negative changes in your thinking. So negative thinking and negative mood states and the arousal and reactivity. So you're constantly in fight flight mode.

Why do some people get PTSD and others do not? And I think the biggest risk of PTSD in our population is your GI procedures. You, someday, maybe will do so many colonoscopies that it doesn't even occur to you sort of how invasive, how traumatic that is. The state of being put under-- and again for any kind of scope and somebody's, when I'm out of control, is going in there and looking around inside my body.

So people who've had any kind of trauma that can be, again, very reactivating. But the procedures itself-- it's amazing when you talk to people and-- remember, it's not what really happens. It's the perception to that patient what happens. So maybe nothing happened but if that patient thinks something happens and then that's what they're replaying, they're going to start physiologically, emotionally, cognitively responding as if something happened.

So three things go into why some people get PTSD for the same event and others do not-- personal factors, the nature of the traumatic event, and the recovery environment. So personal factors are the history. There's certain demographic features-- female gender, younger age, and minority status have put you at higher risk for post-traumatic stress.

And while there is no PTSD gene, it does tend to run in families. And so people are looking for a PTSD gene. But we don't do as well in psychiatry with our genotype phenotype associations. It seems a little more complex. Traumatic events could be deliberate. So combat, assault, abuse, or unintentional.

And again, I think the major reclassification here in DSM is medical events or even having a really bad course of your disease. So I have patients who have PTSD from having perforations. So near life experience-- so when our patients come in and are hospitalized for their GI disorders and are deathly ill, any of our patients that go to ICU-- it's a huge set up to have post-traumatic symptoms afterwards.

And I think it's something that we're not sensitized enough to, all of us, that the event doesn't have to be sort of classically something that threatens your life in the moment in a deliberate way. The recovery environment-- so if you have low social support, if you have other stressors going on when this happened, or if it's a new trauma, again, these are things that will toggle whether you get PTSD or not.

All right, depressive disorder psychopathology-- so we can have some age appropriate mood lability. So adolescents are moody. That is OK as long as they are not functionally impaired. Actually, their moodiness helps them individuate from parents.

It's amazing. I mean, I always laugh when college students are going off to college. They usually get more angry and annoyed at their parents because it's so much easier to leave somebody when you're angry at them than if you have that same kind of loving type bond that you did all the time. So we can have adjustment disorders that are more depressive.

Brief sickness behavior occurring during any kind of GI flare-- so any time you're metabolically impaired, so that's whether your liver enzymes are elevated or you're having elevated C-reactive protein in your system, the brain is going to feel that. And we get basically serum sickness and that looks like the somatic symptoms of depression.

And if they're severe, even if we think they're totally somatic, the cool thing is we have studies now to show that a course of cognitive behavioral therapy can help those somatic symptoms that are directly related to a biological event in the body-- get better faster if we help people cope during that sickness and give them the tools to cope.

Major depression is having a cluster of symptoms that is for at least two weeks leading to functional impairment. Dysthymia is milder depression where you sort of have low grade but still impairing symptoms for over a year. And chronic sickness behavior is when those symptoms of depression persist even when the markers in the body are coming down-- that the metabolic or the inflammatory event is over.

When major depression has suicidality associated with it, that's something that we pay attention to. And actually, you can be suicidal and have mild symptoms. So the severity of the depression is not always what determines if somebody is suicidal or not. Even severe sleep deprivation-- when they're getting astronauts-- this is some of the work that was going on at Harvard when I was there. They're getting astronauts ready to go to Mars. And so I got to be involved in doing psychological testing.

They were trying to determine, with Provigil, how long can the human brain stay awake and be functional. It's 16 hours. After 16 hours your cognitive ability just falls apart. And after 24 hours-- and I mean, you have to pass pretty rigorous tests to be even trained as an astronaut, they get suicidal. 24 hours of sleep deprivation. And these are like literally, they're poked-- they're not allowed to sleep a wink. And then, every hour they're challenged with tasks.

So nobody is absolutely protected. It's just that some people who have again, medical illness or genetic propensity for these things will have these events happen in an easier way or a more serious way. Any time we have co-morbid anxiety disorders, so more than one disorder, we want to think about this as being more severe. And than anybody under any kind of extreme life stress.

Remember, having a newborn at home-- extreme life stress. So that life stress can be positive stress but anything that disrupts normal functioning again, in the context of symptoms, we want to pay more attention. So we have somatic symptoms of depression, cognitive symptoms of depression.

You have to have either a depressed mood or anhedonia, so a lack of pleasure in things, and then you need five of either the rest of the somatic symptoms or cognitive symptoms. And recurrent thoughts of death count as a symptom so you don't have to be suicidal. If you just wished you were dead and you're wishing you were dead more days than not, that actually counts as a depressive symptom.

We have a lot of specifiers. So this is where we, in the behavioral health field-- major depressive disorder can be with a seasonal component. There can be mood congruent or incongruent psychosis. It can be an anxious depression. Mixed features is having actually some mania, so high irritability and impulsivity. Again, they're not meeting criteria for bipolar disorder. You can be catatonic, it can be postpartum, it can be with melancholic features.

Those patients are just heavy, leaden. They feel like their whole body's-- just they can't move. They become immobile And atypical features is actually when you eat more, you sleep more. So everything is in the more instead of the less direction. And why this is important is each of these specifiers-- we do something differently with what medication we might prescribe and what we do with these patients in their psychotherapy for their coping skills.

All right so what are the rates of psychiatric disorders across GI disorders? Now, this is heavily drawn from IBD, IBS because that's where it's been most studied. There is a growing interest in chronic pancreatitis-- that we have to do a better job, especially with the depression.

But again, 20% to 40% of the patients-- and again, think of the IBS group as anything functional. So if your patients have, with any disorder, also a functional component, a gastroparesis has been studied, high rates of anxiety, high rates of depression, and the post-traumatic stress disorder that we can detect, 2% to 10%. So again, not a benign number.

When we're thinking about chronic pain, so chronic GI pain, we think about anxiety, depression. Eventually, if you have chronic pain for long enough, you're going to have some kind of mood change because the change will happen in the brain. So the brain doesn't stay static. Chronic pain, unfortunately, reorganizes the brain in wrong directions. And we have a functional imaging that is showing us that more and more.

Some cognitive traits-- neuroticism, perfectionism, high degrees of that negative catastrophizing. Social situation-- so isolation, people going through a divorce or any kind of breakup are at very high risk for having exacerbation of their pain. And then trauma-- PTSD and again, extreme sustained stress.

We talked about it with PTSD and IBS-- so about half of women who are referred to a GI clinic and had IBS have sexual physical abuse on their history. And that's really associated with many more somatic symptoms but including pain and abdominal pain is really up there. Seems to be greater in IBS than IBD but I think it's been studied more in IBS versus IBD. So you always have to think about the context of the study design.

Now we don't have time to cover this because I wanted to spend the second half of your talk on what we do about these disorders. But eating disorders can, in GI, masquerade as I don't eat because I'm nauseous. I don't eat because I vomit or I'm afraid of vomiting. I don't eat because I have abdominal pain.

But they don't eat, they don't seem that concerned about not eating, they don't want to be re-fed, and they really are cognitively very inflexible. So that's the anorexic group. And then, I know in IBD we are seeing increasing obesity.

And a lot of emotional binge eating and patients who can't eat when they're really sick, then they get the steroids, then they overeat and then that overeating pattern gets stamped in. And so we're looking for eating disorders in anybody basically who's having extreme or sustained fluctuations in their weight in either direction.

Alcohol dependence and substance abuse-- opioids. And it is unfortunate that medical marijuana is legalized and that Crohn's disease is out there and nausea is out there as some of the possible indications for this. Because so far in all of gastroenterology the rigorous studies showing true efficacy are very weak but the cognitive effects and some of them lasting cognitive effects of marijuana are actually very well known.

And as you know, cannabis binds to fat. And so, even with just a couple time a week use of it, you can really have sustained cognitive symptoms. Some people paranoia-- it can actually change your mood, you become irritable, you have a dysthymic kind of depression. And so again, these aren't benign substances to the brain.

So what do we do with our interventions? Well, we want to break up as many different negative cycles as we can. So we want to break up the relationship between the environment and the physiology. We want to treat the symptoms directly. If there are early life events-- genetics, we can't do anything about yet. But certainly childhood adversity is very treatable with certain techniques, again.

And so we're really thinking about the whole patient and all the different ways we can disrupt those cycles that are having the emotional disorders drive even worse psychiatric disorders. Or as the evidence is showing in your field, if you're depressed, whatever you're given for treatment of the biological condition, those patients don't do as well. They don't have as robust response to your GI medications that you're giving.

So anxiety disorders, depressive disorders, somatization disorders, we really try to use behavioral interventions first. So we really don't try to trigger those antidepressants for any of these unless we really feel that somebody hasn't responded. Because 70% of patients have a significant response 70% have 70% response improvement in their symptoms after three to six months of behavioral treatment.

Of those behavioral treatments, cognitive behavioral therapy is most studied, hypnosis, mindfulness meditation. And I'm going to go and just give you a few slides on each of those just because when they come to us, the success that we can have and the quickness of our success depends on how well all of you can sell these as validated approaches. And that as much as you all can talk about the brain-gut connection being real and that you really are treating your gut by taking care of your brain in these ways.

So five core principles of behavioral therapy. So reciprocal inhibition-- two incompatible states cannot co-occur. You can't be both relaxed and stressed at the same time, try it. Really physiologically impossible to have certain states at the same time. You can feel joy-- truly feel joy in the moment and be severely depressed at the same time. So we really use these principles a lot in what we do.

Conditioned fear anxiety can be subjected to counter conditioning. So if you are conditioned to be a certain way, good news, we can decondition that in the other direction. It involves shifting somatic attention and a mastery of self-regulation. Exposure to fear is often necessary.

And it's just amazing how much people fear their GI tracts-- their symptoms, the what's going to happen to me if I have an accident of some kind-- a bowel accident. The fear of what people go through. I mean, for you guys you write the order for bowel preps. For a lot of patients, there's a lot of emotional draining that happens. And compassion toward one's uniqueness-- acceptance is key.

So we really are always building on patients' strengths in behavioral therapy. So what kind of general techniques are we using for your GI patients? So strategies that directly reduce autonomic nervous system arousal and we're really talking to patients about the brain-gut axis-- heartburn, IBS, nausea, cyclic vomiting.

I mean, cyclic vomiting has a huge psychic component. And actually one of our most successful treatments for cyclic vomiting in our clinic is medical hypnosis. So these patients often their bodies reject medications with the same oomph that they're rejecting whatever else they're rejecting psychically. And just literally throwing it up and getting it out.

So you could have some gastroparesis. You can have some physiological changes. But to really get to the core of cyclic vomiting, it does take the reduction of that autonomic nervous system cycle that has become activated and then helping them to understand what else-- what emotions are literally coming up and are they trying to push out?

Diaphragmatic breathing is a wonderful vagal nerve stimulator. And it's a right kind of vagal nerve simulators. So it's the best way to bring on parasympathetic response. Gut directed hypnosis has empirical randomized control trial evidence for IBS, heartburn, functional dyspepsia, and IBD-- keeping ulcerative colitis in longer remission.

Guided imagery is something where you just basically ask people to use their imaginations to take them somewhere pleasant. So if I asked all of you to think about your favorite vacation spot or that place you go when you just want to completely unwind, where is it-- and I ask you to put yourself there with all your senses, we've done guided imagery.

So it's a technique that really helps distract people from that somatic focus. Muscle relaxation can do that. And then cognitive behavioral therapy, we use that a lot. So let's talk about these three in a little bit more detail.

So what's cognitive behavioral therapy? It's built on the premise that our feelings, thoughts, behaviors, and our body reactions are all connected. If you're feeling upset, your thoughts are going to be more negative, you're going to have more usually negative body reactions or be aware-- you're going to be queued in to body reactions if you have that propensity.

And all of that can lead to shut down or it can lead to agitation but it can lead to maladaptive coping disorders. And then whatevers happening in the environment, if those environmental factors that reinforce that in the wrong direction, you're a real mess. So what we're doing with cognitive behavioral therapy is we're targeting the thoughts, the behaviors, and the physical symptoms to reverse things that are going in the wrong direction spirals and get people feeling better.

So these are the kinds of thoughts and this is related to now having chronic pain, unhelpful beliefs about best ways to cope with pain, that negative hypervigilance. There's often low self-esteem or self punishment themes in patients who have a lot of persistent physical pain, feelings of depression, anger, anxiety, worry, pain related behaviors, reduced activity, social isolation. And then often with chronic pain we get poor sleep, we get sort of tension or even pain in other parts of the body, a lot of lethargy and fatigue.

So all of these are what we target in cognitive behavioral therapy. And we're really teaching coping skills. We're taking those renos and we're giving people skills and then asking them to practice it. Chronic abdominal pain is where it's been most studied. And there have been many randomized controlled trials and has a medium to large effect size for pain and a medium effect size for comorbid depression and anxiety. And really superior to other active and inactive treatments.

For PTSD, we don't have a magical pill for PTSD. So we do a lot of education about the trauma and the body's reaction. We do a lot of autonomic nervous system retraining with hypnosis. We have fancy, hypnotic techniques. Hypnosis is just putting people in a trance, eye movement desensitization, and reprocessing. We're desensitizing them to their internal cues that set off the cycle. We do stress inoculation training, so some prolonged exposures.

We really listen to their stories and help them change their narrative so that they can have mastery of their trauma reminders. So this takes a lot of work. This is not fast work. But usually within a year, if I can get somebody engaged, I can make the PTSD sort of just basically another annoyance in their life and not sort of the driver of so much physical and emotional pain. And it works, these techniques work in PTSD.

IBD-- and this is some of the work that I've done through the decades. But there's been some small trials that had mixed results for CBT for anxiety and depression. Recently, both in children and adults, we are showing a sustained effect of CBT for anxiety depression and also for effects on IBD activity.

One of the trials that I did, I compared cognitive behavioral therapy to supportive therapy for depression in adolescents with IBD and we were able to see a significant reduction, not only in their depressive severity but 65% of the sample went into remission. So both types of psychotherapy worked. Interestingly, when we looked at the degree of IBD activity, it was more robustly reduced with CBT than with supportive therapy.

I wanted to understand this more so I looked at patients with active and inactive Crohn's disease. And when he had active Crohn's disease, you've got the serum sickness, more inflammation so you have a higher clustering of depressive symptoms. And what was fascinating is when you had mild to moderate inflammation and depression, that group had a more robust response to learning coping skills to retraining the autonomic nervous system than the group that had inactive IBD.

So we hypothesize that the way that cognitive behavioral therapy-- it's resetting your autonomic nervous system tone, it's resetting sort of the way your brain is receiving those cues, and that has a positive feedback to dampening the inflammation itself. Those kids in that group had a faster-- so the slope of their improvement in their IBD symptoms was also greater. And when we looked at the probability of flare in Crohn's disease, I'm looking out 18 months, they also had a more sustained-- a longer remission period.

All right, lots of things going on in the brain with inflammation and depression. And one of the hypotheses is that high levels of TNF-alpha actually take that tryptophan to serotonin pathway. And remember, serotonin is key to fight against anxiety and depression. And it turns it into a neurotoxic pathway, kynurenine.

And in fact, even in people who don't have a systemic inflammatory response, we're starting to look at kids who are depressed and show that this pathway occurs. There is something that's activating their brain pathways that are turning tryptophan into kynurenine. And so the neurotoxic effects of that is what it prolongs and sustains the depression.

We actually started looking at this in adolescents and found that same pattern-- that the more depressed you were, you had higher kynurenine and your ratio of kynurenine to tryptophan was higher than the patients who didn't have depressive-- especially if there was a strong component of anhedonia.

So we're actually putting in a Crohn's and colitis foundation grant right now, due in two weeks, to look at this in a little more detail. So that's the other nice thing about our whole behavioral set up is we're heavily in to research. So that's your goal research plug. Those of you who like translational neuroscience, there's a place for you if you ever want to join any of our projects.

All right, medical hypnosis-- so hypnosis is a trance state. How many of you, when you're on your iPhone, can totally zone? Like you're drawn in, you're da, da da, da da, and you're relatively oblivious. Like somebody could be calling your name and you don't really hear it because your mind is here. OK. If you do that, you're hypnotizable. In fact, you are in a self-trance.

So basically what we do with hypnosis is we take advantage of the human's mind ability to dissociate and we use it for even greater good. We're mobilizing attention inward but away from the area of distress, or the symptom. And we're taking people to pleasant places, we're helping the brain ignore the body.

And then sometimes we're actually doing, so you could have a cognitive behavioral lens with hypnosis, or you could have a psychodynamic lens. So you could have different lenses. And again, we use what makes sense given that patients history and pathology.

So you have a pain signal. So often when you have pain, automatically you're going to be keyed into that pain. Pain equals danger. You're anticipating punishment. The pain itself is punishing. You have a lot of anxiety, depression. You tend to be withdrawn. And remember, you can have two reciprocal states at the same time.

So relatively speaking, those circuits in your brain that anticipate reward, that maybe can counter that hurt doesn't always have to equal harm, have feelings of joy optimism or have goal directed behavior. Relatively speaking, those circuits become inactive because all of the action is happening in the behavioral inhibition system.

So basically this actually happens automatically. So this isn't something that you're trying to do this is how the brain protects itself, it derails. Depression has a very self-preservation. I mean, if you are that shut down, the world can't hurt me. So it has some survival value, to a point.

So basically we're facilitating with hypnosis, really with cognitive behavioral therapy. But we're just using it with different kind of levers. We are activating those positive circuits and then, by definition of the reciprocal inhibition, the negative circuits get less.

And this has all been very eloquently shown with functional neuroimaging studies that sort of have people while they're in the brain imager do certain tasks that call out these circuits. And so this is really what we are trying to do with patient. Probably hypnosis has the strongest evidence of all of our behavior techniques in improving pain and reducing anxiety.

And with IBS, if you have a successful response, so 12 sessions of hypnosis every other week, you can have a complete or at least stay only mild IBS for-- I think some of the longitudinal studies are going out 12 years. So we can really make these changes permanently if patients are willing to be open to these techniques and practice.

What's mindfulness? So mindfulness is paying attention on purpose to the present moment and accepting it. So it's all about non-judgmental awareness of the moment. And then that buffers us from all that negativity that you saw in those cycles. Sounds lovely-- very hard to do.

And the one thing about the mindfulness meditation work-- so it does have some evidence in IBS and IBD. If you don't practice, you don't get the effect. And here you really have to have a very high degree of practice to keep the effects.

So the Tibetan monks actually meditate for 10 hours a day. And interestingly, they have very low rates, almost no rates, of GI disorders. Well, except as they're getting more introduced to the Western diet and now they're getting obese. They're actually starting to. But relatively speaking to the rest of the population but I mean, that's a lot of work. A lot of people don't have 10 hours a day to meditate.

But again, some of these techniques work for some people, some for others and we offer them to everybody. There's some really cool apps out there. You want to give your patients something to do if you think that your patients need to treat anxiety or chill or stress reduction. Headspace is out there. Now all of these have a period of a free trial and then they have to pay some nominal amount for it.

But Headspace is self-guided meditation. Good for all of you too, by the way, because we're not immune from burnout and just something you have on your phone. And it's a cool Australian accent, for the girls. I mean, the guys might not be into it. But he's a very easy voice to listen to and really just draws you in to a trance state.

All right, so in the eight minutes we have left let's go through psychotropic medications a bit. So for major depression these are the meds and that's why you have these on your handout. So we still use the SSRIs, they still have been the most studied. Followed by the SNRIs.

And then in our populations, bupropion SR does have some mild anti-TNF activity. And we are careful with it in our patients who have vomiting because more so than any of the other agents, if you're vomiting and you have metabolic imbalances, the potential for seizures with bupropion is a little bit higher than the others. We use Remeron for depression and nausea. And then we use trazadone to sleep.

And I think, the important thing to know about these medications-- if we take a critical look in our literature, all the randomized trials for antidepressants for major depression, there's only a 12% response rate, if you're truly looking at remission for depression as your outcome.

So when you're giving these medications, I always tell patients OK, we're going to dampen down some of your symptoms with these medications but these meds aren't going to teach you how to cope with what's driving this and the other kinds of factors. It's the therapy that's going to do that.

The tricyclic antidepressants we use at very low doses. Again, they're wonderful for RGI symptoms for pain. And nortriptyline and desipramine have been most studied. And/or they're also helpful for our patients who have diarrhea, have short gut syndrome, we can use their GI motility effects to our advantage.

The monoamine oxidase inhibitors, we don't use them a lot. We now have some patch forms of selegilline. But it's because of the diet and the crisis that people can get into hypertensive crisis, higher rates of serotonergic syndrome. They are wonderfully effective. I use them but I would not advise any of you to ever prescribe an MAOI inhibitor because you really have to monitor much more carefully than any other class.

We use a lot of augmenting strategies. So if you see some of your depressed patients on lithium, it doesn't mean they have bipolar disorder we might just be augmenting a response. Lithium and Lamictal actually are the only two drugs in trials that showed protecting patients against suicidality, all caused suicidality. So we sometimes use them for that purpose. Buspar, atypical anti-psychotics-- we use the Seroquel and the Abilify.

And what's hot in the pipeline? Probably out in the next six months is a new anti-depressant class that's a direct IL-6 antagonist that is showing very good response for depression in patients with cardiovascular disease.

All right, anxiety disorder-- hey, guess what? We use the same drugs-- SSRIs, SNRIs, the tricyclics especially if it's IBS, mirtazapine, buspar. And again, you often see a faster response for anxiety with these meds than you do for depression.

Now remember with the SSRIs, activation is common in 10% to 15% of our patients. So they get hyped up. They can feel a little bit manic but the true bipolar switch is very uncommon. And it usually means you misdiagnosed a patient, they had bipolar disorder and you didn't catch it.

The GI side effects happen early on but often people accommodate to them. Remember that the SSRIs can cause bruising and increase bleed times. For a different mechanism, Wellbutrin can dry out the nasal passages. And so people have more bloody noses.

And then all of these have been associated with suicidal activation, actually any psychotropic med has a black box warning. When they carefully studied these effects however, they were associated with worse disease that wasn't getting better. And actually we have never been causally linked to these agents.

But because those Brits got that study out quickly without really looking up their data, it translated quickly into the black box warning. So we still have to talk about it with our patients but it's really rare that these medications truly cause it.

So I think that I look at the IBD patients here a lot because I live in that world. And our SSRIs, SNRIs, and bupropion are sort of the favorite mix that we use followed by low-dose trycyclists. Again, there's no magic pills for PTSD. We do use some mood stabilizers for irritability. We do use some beta blockers and some alpha adrenergic agonists for that to dampen the hyperarousal.

And prazosin actually has very strong support to dampen nightmares. So if you see your patients on prazosin from us, they probably have PTSD. Everything that we use for visceral pain in our world is off label. So we use the antidepressants, we use the mood stabilizers. We don't really fully understand how they work, we have a lot of hypotheses but they can work. And rarely again are they the magical cure without behavioral interventions.

Whatever we use, we take one medication a time, we titrate very slowly to the lowest dose possible and we do a lot of expectancy training. Expect side effects before effects because if somebody's expectancy is off, they're going to reject the med.

All right, so the last couple of slides is the IBD medical home. So we provide these treatments in two models-- the VIP center is across the street in the medical arts building, our behavioral specialists, including me, have clinic there. And you can refer a patient there.

And then, we see them and then we coordinate with their GI team. Both by having our notes right in Epic so hopefully, you read our note. If nothing else, read the assessment and plan. Unless you're interested, you don't really have to get to know your patients that well, though it doesn't hurt. But we have a pretty succinct assessment and plan. And it's really good for you to reinforce that.

And then we have our IBD medical home. Right now, it's for health plan patients but likely to expand. We're basically three afternoons a week. This is for IBD but we're thinking about it for chronic pancreatitis as well. Where we're basically doing the behavioral care right in the clinic and we're screening.

We actually have telepsychiatry available to these patients in the home. So they don't have to come see us for their behavioral care, we can provide it to them in the comfort of their home. And we also are testing some cool, digital apps.

One of the apps we're testing is Lantern. It's basically an app on your phone that teaches cognitive behavioral therapy for depression anxiety. And it comes with a real life coach that texts you and does it sort of the motivation and teaches our patients the CBT-- unloads it from us so that the patients are using this and are getting better. Within two months, anxiety and depression is significantly improving in some of our early trial results.

And this is just some of our early trial results that we just presented at DW. The nice thing about our medical home is we have our cohort of first, patients who have been in the home for at least a year. We're not just seeing significant improvement in their clinical symptoms but their unplanned care and their medical hospitalizations are being significantly reduced. So that's exciting.

And it's a collaborative care model. It's interdisciplinary but it's also transdisciplinary. So the gastroenterologist, Dr. Ruggiero, the nurse practitioner, the nurses, the dietician are all trained up in cognitive behavioral therapy, motivational interviewing, hypnosis, and know how to diagnose.

So this way, we are all constantly reinforcing each other's messages. And we are doing it not just again, as a team. But to the patient we present as a team and so we have higher adherence to our GI treatments.

So this is the VIP center. And again, we accept all insurances. And our most common referrals are chronic pain, actually nausea, vomiting, anxiety, depression, sleep fatigue. So we do sleep evaluations. We do extreme fatigue evaluations, substance abuse.

And our diagnoses I mean, I'm the one driving the IBD diagnoses but we also see a lot of functional GI disorders, chronic pancreatitis, cyclic vomiting, some celiac disease. But I think the important thing is we take anybody with any GI disorder and we do the full evaluation and then, comprehensive treatment.

We try. And so when you message this to the patients, we want to be a coping clinic. So we want to get them in acutely for 8 to 20 sessions again, depending on the severity, acuity, of their situation. And then, we want to switch them to maintenance.

So acute is weekly to every other week and maintenance is monthly to every three months check-in. So we also are not trying to take them in and keep them intense. But again, put it on them to learn disease self-management; learn coping skills for their anxiety, depression, and pain; and then get back into their lives.

And that's Greg Thorkelson. So he went to private practice and now we have Bob Howland as our other psychiatrist there. And Helen Sysko, in the middle, is our psychologist. And Emily Weaver, whose picture isn't here, is our social worker. And so that's sort of our core team. So, thank you.