TPWKY – Ep 221 – ECT 2

Leonardo V. Lopez: [00:00:00] So, this patient was actually five at the time that she was diagnosed. She presented initially with seizures, was diagnosed with epilepsy, was sent home from the hospital and then came back with unusual movements and mutism and was diagnosed by the team there with catatonia, which is a psychiatric syndrome that we see across a variety of illnesses, both psychiatric and medical. That provoked a further medical workup, including a lumbar puncture, spinal tap, and she was found to have what is known anti-NMDA receptor encephalitis. This condition is frequently characterized by the things I just mentioned: seizures, catatonia, other psychiatric symptoms, and is treated with a combination of immunotherapies to deal with the overactivity of the immune system and also sort of conventional treatments to manage seizures, catatonia, etcetera. So this patient received immunotherapy, many immunotherapies actually, and received lorazepam or Ativan, which is the standard medication that we give for catatonia to very, very high doses, and continued to receive these treatments over the course of about nine months.

And after that nine month period, she remained very catatonic. She could not speak. She could not dress herself, which she'd been able to do previously. She could not feed herself. She required a gastrostomy tube or a feeding tube for all her feeding. She required total care from her family and from the nursing staff. And so at that time, the team at the hospital where she was a patient at the time started discussing the possibility of treating her with ECT. ECT is a gold standard treatment for catatonia, has very high response rates across all causes of catatonia, both psychiatric and medical. However, it's not typical to treat ECT in patients so young, in part because it's not typical to see catatonia in patients so young or anyway, it has not been recognized in patients so young, historically. It's almost certainly been underrecognized. So it would've been unusual to treat a patient this age with ECT. But they had sort of run out of options. They had given her many, many doses of immunotherapies are also chemotherapeutic agents that have their own side effects. They had treated her with very high dose lorazepam, as I said. And so the decision was made to transfer her to our hospital, of course with the consent of her parents, to treat her with ECT. That was the youngest case that we've treated, one of the youngest cases in the literature. We began treatment of her nine months into her illness, and after about eight weeks, her catatonia had resolved entirely. She was speaking again, walking again. She was able to play games with her hands and draw and blow bubbles and all these things.

So we in fact had her well enough to eat again. So she had come in in September, and we got her eating enough just in time for Thanksgiving dinner. So she was able to eat a full Thanksgiving with her family, which was very exciting for us. So it was just a testament to the power of the treatment, particularly for catatonia. And sort of a reminder that we do have this treatment available for anyone who needs it, and we should always be thinking about it in these cases.

EW: Dr. Lopez, thank you so much for sharing that story with us. It is, it still blows my mind

EAU: I know.

EW: and there's a full episode on it and the Advances in Care podcast, so go check it out, but whoa.

EAU: Yeah, there's also a YouTube video, Erin, that you had sent me, um, that the hospital put together that's also really moving. And yeah, thank you so much for taking the time. Um, and everyone, you'll get to hear more from Dr. Lopez later during the episode.

EW: you will. Yes you will. Hi, I'm Erin Welsh

EAU: I'm Erin Allmann Updyke.

EW: This is, this podcast will kill you.

EAU: Welcome to ECT part two.

EW: ECT, part two. If you, is this, if this is your first time tuning in, check out last week's episode on the history of ECT. Helpful context. Is it necessary to understand ECT today? Yeah.

EAU: Feel like it is, definitely feel like it's okay. So go listen to it if you haven't already.

EW: Yeah.

EAU: if you haven't already, then you might not know. ECT is electroconvulsive therapy. See, there's important things you need to learn from that episode.

EW: Yeah. Pretty important. Pretty important.

EAU: Uh, but today we're gonna be talking all about what is ECT, how does it actually work, how do we use it today? Uh, and of course we're going to be interviewing an expert that is Dr. Leonardo Lopez, who is vice chair for inpatient services and associate professor of Clinical psychiatry at Weill Cornell Medical College. And he oversees a lot of inpatient psychiatric services, including ECT.

EW: Including ECT.

EAU: It's gonna be.

EW: it is really thrilling to get to like get this, this actual expert

EAU: Yes.

EW: this, this, this therapy

EAU: Yeah,

EW: so much about.

EAU: I know.

EW: that'll be later in the episode. But for now, we still have a few important things to cover,

EAU: Like quarantini [00:05:00] time.

EW: Time this week as, as with last week, we are drinking shock talk.

EAU: Shock talk.

EW: is grapefruit, ginger ale, basil, it's tasty.

EAU: Yeah.

EW: out our recipe on our website. This podcast will kill you.com, as well as on all of our social media channels. Follow us if you're not already. Our website has lots of things. If you listen to last week's episode, you know, check it out.

EAU: check it out. This podcast will kill you. Dot com. Rate review and subscribe. We're on YouTube. Hi, shall we get into this? There's a lot to cover.

EW: I would love to, let's take a quick break and, and get started.

EAU: Okay.

So last week, Erin, you walked us through the history of ECT as a practice from the pretty gruesome to the remarkably effective and kind of left off with where are we now, and kind of where do we go. So my goal for today is to make ECT feel for so many of us who maybe have never witnessed it or who have never experienced it, feel like something that is real that we have a concept of in our mind that we can visualize. Because I think for so many of us, we might be able to picture like what a surgery looks like. We might be able to picture in our mind what it might be like to have to take a medication every day. But the images that we likely have of ECT, like you talked about so much last week, Erin, they're tinged with so much sensationalism.

EW: Yes.

EAU: And it might be that one flew over the Cuckoo's Nest is our best image of what ECT is, and that's not a picture of what it actually looks like today at all. So what we're gonna talk about today is what this actually looks like as a medical procedure, what the situations are that we use it for today. And then, like I said already at the end, we're gonna be joined by Dr. Leonardo Lopez to talk about kind of the future of ECT. So. There's a lot to cover.

EW: really is.

EAU: So just a few things on the agenda.

At the very top of last week's episode, Erin, you defined ECT for us. Electroconvulsive therapy is basically delivering electricity to our brain with the goal of provoking a seizure to treat a variety, a really kind of wider variety than you might think of what we call affective disorders. So those are things like treatment resistant depression, which might mean either unipolar depression, so that's things like major depressive disorder or bipolar depression. We also use it for psychotic disorders, including psychotic depression, schizoaffective disorder, and schizophrenia, and catatonia and related disorders, which we're gonna talk about in a lot more detail and can present in both more neurologic and more kind of psychiatric conditions.

EW: Hmm.

EAU: But what does it actually look like?

So if you or I were going to go get ECT, if we had a need for this procedure, this is kind of the steps that we would go through. First, there would be a very lengthy discussion of the risks and the benefits of this procedure. The person who's performing it would be talking with us. They would be talking with our family or our friends and other like surrogate decision makers if needed. There is a really robust informed consent process that has to be gone through. Usually there's also a general physical exam that has to be done either by a primary care or hospitalist physician, or sometimes by a cardiologist to make sure that our heart and our body is overall healthy enough to withstand this procedure, especially the anesthesia.

We would then have to not eat anything for an entire night, and then the next morning we'd arrive to a room, either in a hospital or an outpatient clinic, depending on the situation. We would be wearing a hospital gown like we always do for any of these kinds of procedures, and we'd probably be greeted by either a nurse or a technician and an anesthetist. We'd get an IV put in our arm. We'd have a blood pressure cuff. They'd take our vitals. Actually, they'd put two blood pressure cuffs on, one on our arm, and one on our right leg. We'll talk about why in a second, and then while they're chatting with you, they'll start to hook up a whole bunch of wires that they're gonna use for monitoring us. That'd be an EKG on our chest to monitor our heart. During the procedure, there'd be an EEG. On our head to monitor the seizure in our brain, and there would be an EMG or an electro myelogram that would be placed on our right foot. I'll tell you again why in a little bit.

EW: right foot.

EAU: And that's to monitor the seizure in the muscle itself. We'd probably get a little bit of extra oxygen via a mask. There might be a little thing put up our nose to monitor our CO2 levels, one of those clippies on our finger to monitor our blood oxygen, and depending on what type of electrodes that are going to be used for this procedure, they might put those on our head right now. Uh, if they're the sticky kind or they might use the kind of more old school kind that are handheld. In that case, you, you might not see those yet. [00:10:00] And then the anesthetist is gonna give us a medication that will put us to sleep. There's a few different medicines that they use. They're pretty standard anesthetics like Propofol, orate. No one cares about these names. Okay. Once you're unconscious, then that second blood pressure cuff, the one around our

ankle, our right leg is inflated. The reason for this is that that's going to reduce the amount of blood flow to our right foot. Not cut it off completely, but reduce the amount of blood flow to that foot, and then they're going to administer another medication after we're unconscious. And this one is a muscle relaxant. Usually it's something called succinylcholine. This kicks in pretty quickly and you can tell when it's kicked in because as you're administering it, it actually causes kind of these twitchings, these little tremors in your muscles, and then those tremors will stop. Once those tremors stop, it means that the muscles are completely relaxed. Okay. And then they're going to put a bite block in your mouth, and that's to protect your teeth and your tongue. And then they're going to administer a very brief pulse of electricity via those electrodes that either they stuck already onto your head or they're holding onto your head. And by a very brief pulse of electricity, I literally mean on the order of seconds, or sometimes even a half of a second of electricity.

EW: Okay.

EAU: The amount of electricity specifically that they're going to deliver depends on what your seizure threshold is, but the goal is that it's enough electricity to cause a generalized seizure. Now this seizure is not going to be visible in your muscles the way that a grand mal or a generalized tonic-clonic seizure would be because all of your muscles are relaxed. But what you will be able to see is the seizure on EEG, which we're monitoring and on EMG attached to the right foot, which has less blood flow, so has not been affected by the muscle relaxant. Succinylcholine. That seizure is only going to last between 25 and 70 seconds. That's how long the whole seizure itself should last, and then that's it. The procedure is over. You're disconnected from all of your wires. The anesthetic will wear off within a matter of minutes, maybe a half an hour, maybe an hour, and then that's the end of the procedure. And you'll be wheeled to the recovery room to wake up.

EW: That was so helpful.

EAU: Thank you.

EW: That was really great.

EAU: I worked actually really hard on that, so thank you.

EW: No, it really, it really was like a helpful contrast to like, okay, what do we see on movies when, when we hear this in our head, what, what do we, what do we picture?

EAU: Yeah.

EW: And it is often a far cry. And so I

EAU: Yeah.

EW: really, really great.

EAU: Exactly. I know you have a million questions.

EW: Okay.

EAU: Give them to me

EW: Okay. Right foot.

EAU: right foot. Why the right foot? Oh, that's such a great question. Um, okay. There's a few reasons for this. In part, it's, it's not completely random. To answer that question. I wanna actually answer first where we put the electrodes,

EW: Okay.

EAU: Then I'll be able to kind of like long way get to that question. Okay. Okay. So the electrodes are placed, uh, there's a few different ways that you can place the electrodes. You, in most of the, like, media representations, the electrodes are placed on the temples on both of them. So kind of like if you draw an imaginary line from the corner of your eyes back to your ears, it's kind of midway along that line, just above it in right on your temples. And that is kind of old school standard placement still used quite often, um, but not necessarily the first line. Treatment option for a lot of people in the US today, but that's a perfectly decent option. Um, sometimes instead of being on the temporal, people will put them bi frontal, so like on the forehead, kind of like outer corners of the eyebrows.

EW: Okay.

EAU: But there's also another option, and that is right, unilateral placement. And so this means that you'll have one electrode on the right side in that same like temporal position, and then the other one just to the right of the very top of your skull, right at the apex there. A lot of times we start with this right lobe only, right unilateral placement because it's associated with a lower risk of side

effects. However, it tends to also be slightly less effective than bilateral placement. The reason that they chose initially to do right rather than left is because for most people, like 98% of right-handed people and 70 to 90% of left-handed people, our language function is all on the left temporal lobe. So the thought was that. Maybe there would be less side effects, especially with language. Um, if we avoid direct current to that left temporal lobe.

EW: Huh.

EAU: But so the right foot, uh, regardless of the position of those electrodes, is a place that you can easily isolate and is going to still have motor activity regardless of where, I mean, really you should have motor activity anywhere. So I think right foot is just like, that has become the standard of where you do it. It could be the left foot. Sometimes people will use the arm if [00:15:00] they have to. Let's say you don't have a right foot or something like that, you can use something else. Um.

EW: Okay.

EAU: Okay, I guess I didn't really need to answer the placement question, but

EW: was helpful. It

EAU: thank you.

EW: context. Interesting. Um, the, uh, seizure threshold,

EAU: Seizure threshold. I knew that you were gonna ask that question. So the seizure threshold means. The lowest amount of electricity that's going to give you a seizure.

EW: Yeah.

EAU: How we determine what that seizure threshold is, kind of depends on where you're going to be getting ECT and how they're going to do it. There are like age-based guesstimates that we can use. The other thing that you can do to try and estimate it is to start with a really low charge, and then go up in increments until you get to a seizure and then, you know, okay, the threshold was somewhere in between. The most lowest that didn't do it, and then this one that did. But the key actually is that we're not just hitting that seizure threshold. The goal is actually to give an amount of electricity that's between two and a half to five times that minimum seizure threshold. That based on all the data

that we have is what seems to be the most effective for ECT. Two and a half if you're doing bilateral, so two and a half times threshold for bilateral and about five times if you're doing only unilateral. So you need a higher charge if you're doing unilateral placement.

EW: Okay.

EAU: So.

EW: This is, this is, I think maybe getting into like a, a, a bigger question or like something that you will address,

EAU: Okay. Maybe.

EW: you've mentioned placement when

EAU: Yep.

EW: efficacy

EAU: Yeah.

EW: and you've mentioned the actual like stimulation provided.

EAU: Yeah.

EW: measure effectiveness? What does that mean?

EAU: Oh, that's such a great question. Effectiveness is measured by resolution of symptoms

EW: Okay.

EAU: So that's gonna depend on what the thing is that you are treating. But there's usually, like, let's say that it's depression, unipolar or bipolar depression, you've got a bunch of questionnaires that people are being given on how bad their depressive symptoms are prior to ECT, and then you're gonna be giving those same questionnaires after ECT, and that's how you're going to judge how effective the ECT has been. You're also going to be. Estimating side effects, right? So then you're always going to be doing kind of a cost benefit analysis. Did we start with bilateral and we're having too many side effects? Well then

maybe we wanna switch to unilateral. Did we start with unilateral and maybe we're not having as great of an effect as we want? Then maybe we switch to bilateral. Isn't that interesting? There's really, it's an, it is definitely one of these places where we talk about this a lot. There is both art and science in medicine, and that is definitely true for ECT because there is not like one standard protocol that everyone is going to respond to or that everyone is going to get.

EW: Okay, so you also mentioned that the seizure that is induced can last, you know, up to 75 seconds, whatever. It's,

EAU: Yep. It's like a minute. 30 seconds to a minute. Yep.

EW: What happens if it doesn't stop?

EAU: Yeah, if it doesn't stop, then you give medications the same way you would with any other seizure that's lasting too long, um, to stop that seizure. And that does happen sometimes. It also can happen where somebody maybe doesn't have a seizure, and so then you might have to give a second dose or decide if you're going to increase, um, the amount of electricity to try and induce a seizure. Or if someone had what's considered an inadequate, so less than 25 seconds of a seizure.

EW: How fascinating. Okay.

EAU: And then each, oh, can I, one thing that I'm guessing you might ask, um. I said that this was this after that, you know, 60 seconds or so of the seizure, that's it. The procedure is over, but that is not the end of ECT because ECT is generally something that is done multiple times. So usually it starts about three times a week and can last anywhere from like six to 18 sessions or so. And for some people it might even be that they have continuation or maintenance therapy where they kind of taper it down. So it's no longer three days a week, maybe it's twice a week, and then once a week, maybe it's once a month. Um, but in general it's quite rapid acting, especially compared to so many other therapies that we have. We usually see substantial improvement in symptoms within the first few sessions, so within the first couple of weeks.

EW: Okay. Yeah, I, I mean, I guess I don't, the, the next questions I have are, is like, how this works.

EAU: How does it work? Yeah.

EW: Yeah.

EAU: Yes. Okay. Let's get into it. Um, of course, the short answer to how does this work is that we still don't really know. We don't really know, and that's in part because we don't know the biological mechanisms of so many of these disorders that we can treat with ECT,

EW: Right.

EAU: But that doesn't mean we don't have hypotheses. There are a few main hypotheses as to how ECT works in our brains. The first is that we see substantial increases and changes in a bunch of our neurotransmitters. These are friends that. We know well from other episodes like dopamine, serotonin, et cetera. We also see big changes in gaba, which is an inhibitory neurotransmitter. And what's interesting [00:20:00] about GABA is that we think, well, maybe this is just like a neurotransmitter effect, but also GABA is involved in seizures because it's like an inhibitory neurotransmitter. If GABA levels are low, then your seizure threshold is lower, and we know that ECT actually raises the seizure threshold. So during a course of ECT, uh, seizures tend to get shorter. And sometimes we have to go up on that electricity dose in order to have an actual seizure because our threshold is rising with increasing treatments. So there's some thought that maybe it's actually a GABA related anti-convulsant effect that somehow is, is how ECT is working.

EW: Like,

EAU: We dunno.

EW: your brain to produce more gaba, and that's having other.

EAU: That's an anticonvulsive effect, and then why is that also affecting all these other things? There's some involvement there that we don't know. We also know that ECT has induces really big fluctuations in a whole bunch of hormones that our brain releases. So inside of our brain, there's these areas called the hypothalamus and the pituitary gland. We've talked about these in other episodes, and these produce hormones that go throughout our body and interact with a whole bunch of other organs like our thyroid. Our ovaries, our testes, our adrenal glands, and what we see after ECT is increases in a lot of these hormones that are produced by the hypothalamus and the pituitary, things like prolactin, uh, A CTH, which stimulates the release of cortisol. We also see increases in TSH and just a bunch of changes that happen in our hormones. How does this then directly cause improvements in symptoms? We don't know, but we see these changes from ECT. And finally there's also evidence, and this one is so interesting, Erin, ECT induces what's called neurogenesis, which really

means like structural brain changes in various parts, in various different cell types. This we think is mostly mediated by this really interesting compound called BDNF or brain derived neurotrophic factor. And this actually induces neurogenesis. And so we think maybe it's down to that, maybe it's some combination of all of these different things that are all happening as a result of ECT. And we don't necessarily have like a one to one-to-one mechanism of like, this is the thing that helps with depression. This is the thing that helps with suicidality. This is the thing, you know, we don't, we don't necessarily know.

EW: Well, and I feel like this wide range of effects explains why it has helped in so many different things that we don't, I mean, I guess we don't necessarily know if there's the same physiological

EAU: Right.

EW: underpinning them

EAU: Yeah.

EW: Yeah.

EAU: And there probably isn't, right? Like the things that cause psychosis in schizophrenia are probably not exactly the same things that are causing treatment resistant depression. Right? That doesn't make sense that those are the same exact thing, but we can treat both of them with something like ECT. So it is, it's really interesting.

EW: interesting.

EAU: Yeah,

EW: interesting.

EAU: and, and really, like we talked about last week, Erin, ECT works.

EW: Yeah.

EAU: Is remarkably effective. One of the main indications for ECT, and one of the main reasons that we use it, especially here in the States and in a lot of you know, European countries and Australia and New Zealand, is treatment resistant depression. And that can be depression with or without psychosis, and with or

without suicidal ideation. It can be unipolar or bipolar depression, but most of the studies on remission and response rates are. Remarkable. We can see anywhere from like 50 to 60% or more remission, meaning you have essentially no depressive symptoms like you have gone into quote unquote remission and response rates, meaning improvement in your depressive symptoms can be anywhere from 60 to 90% in some studies. And when we compare this to SSRIs, which we talked about in a prior episode, they have a remission rate at best of around 40%, right?

EW: Wow. Yeah.

EAU: There's also data that ECT can cut readmission rates in the first 30 days in half. Um, it's not perfect. There is certainly still the risk of remission in the first six to 12 months especially, or the risk of relapse essentially. Um, and the recommendations on maintenance therapy are not clear cut by any means.

EW: Yeah.

EAU: And of course there are definite side effects to ECT.

EW: Yes, yes.

EAU: There are minor side effects that often don't even get mentioned because they're considered so minor. But these might be things like headache or muscle aches, especially actually from the succinylcholine because it causes those twitchings at first. So you might actually get muscle aches from that, which is so interesting.

EW: Hmm.

EAU: Uh, nausea, which is usually from the general anesthetic more than anything. These are all typically very easily controlled with things like antiemetics and like acetaminophen over the counter medications, and that's why I think no one really talks about [00:25:00] those, but those are definitely post-procedural side effects. But it's the cognitive effects and specifically the memory effects that are the main complication of ECT. And these can happen actually on a few different timescales. Immediately after the procedure, people are going to be a little bit confused. That's from the seizure itself, and that's also from coming out of anesthesia. But this is something that usually only lasts a few minutes, maybe like 30 minutes or so. Some people might have a harder time coming out of anesthesia. They might even get a little bit agitated, but this is pretty rare, and it's usually just the first 30 minutes where people are quite

confused about what just happened. The first couple of weeks, some people might have problems with attention or things like executive functioning, like being able to do the kind of high level tasks that you might have to do to say, run a household or something like that. But what's really interesting is that most of the data on most aspects of cognitive function are that they actually improve after ECT.

EW: In the long term. Okay.

EAU: the long term. Exactly. And we don't know why that is, except that maybe it's because we've improved the depressive symptoms especially. Right. And that depression has a huge impact on cognitive functioning, but memory is a different story.

EW: Memory is a different story.

EAU: And there's two types of memory loss that can happen with ECT. There's something called anterial grade amnesia, and that's difficulty retaining new information after the procedure. So like you do the procedure, you go home and you're like, I don't know where I put my keys today. I can't remember what's on the schedule for today. What did you tell me this morning? Did we have a conversation? Those kinds of things.

EW: Forming memories.

EAU: New memories. And this can actually get worse over the course of ECT since this is gonna be, you know, anywhere from six to 18 or more sessions. Um, and that is why often during ECT therapy, even if people are going home in between each session and are not hospitalized, they might have restrictions on things like driving. They might be, um, advised not to make big life decisions. They probably aren't able to work and things like that. But this type of anterograde amnesia, the difficulty forming new memories. It almost always resolves within a few weeks of completing the course of therapy.

EW: Okay.

EAU: So people are usually able to start forming new memories again after they have finished a course of ECT. The bigger concern for most people is retrograde amnesia. This is the loss of memories of things that happened before the ECT procedure. It's usually cited as more recent events, so like the last few weeks or months tend to be the most affected. Do you remember, Erin? 'cause I

know you just watched Mad Men recently, but there was, um, Peter Campbell was having an affair with Alexis Bledel.

EW: From Gilmore

EAU: From Gilmore Girls and she like went to him and she was like, I just wanted to talk to you because I'm not going to remember who you are after this because I'm going to go in and have ECT. And I thought that was so interesting. And then sure enough, she didn't remember him after. He went in and she was like, oh, who are you? Thanks for coming to visit. And she was much better.

EW: remember that now.

EAU: Yeah. And so that's not uncommon that you might have a loss. It's usually not a complete loss of like all of your memory, but there can be kind of spotty memory loss. Usually we say the first few weeks or months, but it can go back years.

EW: Okay.

EAU: For most people, these memories will come back or this memory loss will resolve over time. It doesn't always, and for some people, this memory loss can be permanent. It's much more common with bilateral treatment rather than unilateral treatment. and what's really interesting, especially when you read through some of the papers, people have a lot of really strong opinions about ECT. I know that you encountered this as well, Erin.

EW: Yes.

EAU: Um, and you can tell as soon as you start reading a paper, if this is someone who's like super pro ECT or actually anti ECT. And I read a paper by someone, it was actually a commentary, um, but it was in a peer reviewed journal, but it was by someone who has done ECT research for decades, right? So is very, you know, pro ECT, but was, he was railing against people who were trying to claim that we don't have good data, that this permanent memory loss is a real thing.

EW: I think I came across that commentary.

EAU: It was a really interesting commentary because I think that what's so important about this conversation when it comes to memory loss is that this kind of memory loss, even if it's not like your whole, it's not like your whole life

is being erased, right? It's usually spotty memories, but it can be memories of times or places or events that were really personally important to you. You might not remember your wedding. You might not remember specific events that happened, or vacations that you took, things that you used to remember. And that may or may not be really distressing to you, and we don't know for sure if it's going to happen or if it's going to come back.

EW: I think that's sort of what is, you know, when, when you were [00:30:00] talking about the procedure and you were talking about, uh, unilateral versus bilateral application and, and, you know, language, uh,

EAU: Right.

EW: that, is that we don't fully understand why it causes memory loss

EAU: exactly.

EW: fully understand why it happens to some people and not

EAU: Yep.

EW: to then sort of say, well what's, what's the risk of this happening to

EAU: Exactly.

EW: I was wondering, could do you have numbers for this?

EAU: A good question. I I didn't see great numbers on like, how many people do we expect to have permanent versus temporary memory loss. Most of them just say that most people, it's going to come back. How much is most?

EW: Right.

EAU: Yeah.

EW: And I think it's, I mean, it, it is all part of the, the cost benefit,

EAU: It is

EW: and saying like, if you can't function or if you feel like there's no hope left and and it seems like pretty likely that most people will experience some benefit. It's not guaranteed, but

EAU: Yep.

EW: we don't know. It's like much harder to articulate.

EAU: Right to like put exact numbers on the chances that you're going to have. Worse side effects, really.

EW: Yeah.

EAU: And it is one of those situations where we have to, like we said in last episode, hold a lot of truths that are conflicting all at once, right? ECT is one of the most effective treatments that we have for severe affective disorders among other things. And we've come so far in how this procedure is done, how we do informed consent for it. The safety of this procedure, it's incredibly safe. And it has the potential for substantial side effects.

EW: Yeah,

EAU: All all of that is true all at the same time.

EW: of these things are true.

EAU: Yeah.

EW: Yeah.

EAU: We also don't use it as much as we could. Um, for a lot of reasons, cost, especially in the US access, especially in the us. Um, you're much more likely to get access to ECT as an option if you have private insurance or Medicare than if you're on Medicaid or Medi-Cal.

EW: Which is just all, all medical things in

EAU: I, it's so many things. Yep. Um, we don't have really great numbers on like, how many people really get ECT worldwide every year, but in a lot of papers it'll be like maybe one, one and a half, 2% of people with severe depression who actually got ECT, which I think is really interesting.

EW: Hmm.

EAU: Yeah, so it's pretty low percentage wise. It's interesting 'cause you mentioned Erin, that in it was the 1950s that it became standard to do ECT under anesthesia and with muscle relaxants. But there was a paper from 2012 that noted that in some parts of the world, ECT is still done, uh, what's called unmodified. So without anesthesia and without muscle relaxers, which I think is. Really wild because those are totally available.

EW: Yeah.

EAU: it probably comes down to the lack of availability of anesthesiology.

EW: I

EAU: Is what it probably comes down to. And we also use it, I've talked a lot about severe or treatment resistant depression, um, but we use ECT for a lot of other things as well. In bipolar disorder, it's most often used for a depressive episode, but it can also sometimes be used for manic episodes as well. Um, but less commonly. And it started out like you mentioned, as a treatment for schizophrenia. Globally, this is probably still the number one indication that ECT is used for, but in the US it's much less common to use it for schizophrenia or schizoaffective disorder than it is for depressive disorders, but except,

EW: Okay.

EAU: For something called catatonia. So I wanna take a second to talk about catatonia. We should do an episode on catatonia, Erin.

EW: Yeah,

EAU: It has a lot. There's a lot there.

EW: There's a lot.

EAU: Um, but briefly, catatonia is considered a psychomotor condition, meaning there's psychological symptoms and then there's actually these motor symptoms that we see. It used to be considered just a subset of schizophrenia, but it is not by any means a subset of schizophrenia. It can happen in a pretty wide variety of psychological and neurological conditions. But what we see in catatonia is this profound unresponsiveness where people are awake, their eyes are open, but they are not responsive to the world around them. They're often,

uh, they don't talk at all, so they have what's considered mutism, or sometimes they'll have something called echolalia, which is this like repetitive vocalizations, either repeating specific words or maybe repeating a word that you said. Their body is very rigid. There's something called catalepsy, which is where, uh, and also this thing they call waxy flexibility, where you, like, let's say that I moved someone's arm into this upright position and then I look, took my hand away. It would just stay there. Um, they wouldn't move back down. And so people are very, very immobile in this kind of rigid state. And because of how immobile and because of how unresponsive people are [00:35:00] in catatonia, this is a pretty life-threatening condition. People generally cannot eat. They're going to have substantial weight loss and malnutrition. They're not moving, so they're at risk of developing things like pressure ulcers, which of course can get infected and cause major problems. And depending on what position they're in and how rigid they are, they also can start to have like muscle breakdown in things, right? You imagine that your muscle is in this contracted position for a really long time. You can end up with contractures in the long term.

EW: Hmm.

EAU: Catatonia from any cause can have a mortality rate of up to 20% if it's untreated.

EW: Whoa.

EAU: And there's also a kind of subset of catatonia called malignant catatonia, and another condition that's life-threatening called NMS or Neuroleptic malignant syndrome. That is very similar to catatonia, but caused by antipsychotic medications or other drugs. And so similarly, you have this rigidity, you have this unresponsiveness, but you also see fever, tremors, muscle cramps, um, and it's usually a pretty sudden onset. So malignant catatonia as well as NMS and Catatonia, regardless of the cause, is another reason that we might use ECT and it's incredibly effective for catatonia.

EW: Hmm.

EAU: Yep.

EW: How effective.

EAU: Oh, I don't have a number on that.

EW: Um, and,

EAU: we can ask.

EW: fully know why.

EAU: We don't fully know. And it's usually not like first line therapy for catatonia. It's usually if we've tried other things. Usually benzodiazepines,

EW: Okay.

EAU: are a first line therapy. So that's what we would use to, like, what's interesting is we use it to stop a seizure. But there's a lot of things that can cause catatonia. One of the things that can cause catatonia quite frequently. Is NMDA receptor encephalitis. Many of you might think that you've never heard of that, 'cause it sounds like a fancy medical word, but if you've ever read the book or watched the movie Brain on Fire,

EW: It's great.

EAU: That is an example of NMDA receptor encephalitis and that like 30% of cases can end up with catatonia. And while it's first line treated with immunotherapy, in some cases ECT has been used effectively to treat catatonia in those cases as well. So it kind of just like really bolsters the use of ECT in all forms of catatonia, which is really interesting.

EW: Maybe this is a bigger question, but ECT never seems like a standalone therapy

EAU: Yes.

EW: one condition. It's usually used in like a a with a, along with a suite of other

EAU: In combination.

EW: and, but are there situations where it does not last resort?

EAU: If it's an emergency.

EW: Okay.

EAU: So something like a, a catatonia or, or a neuroleptic malignant syndrome. I mean, and honestly, even then, you're gonna start with other therapies first, but if they're not improving, if someone has really severe treatment resistant depression and they come in and they've had a lot of weight loss, they're not eating, um, they're really severely suicidal or something like that, then it might be, but it's, it's kind of hard to say that that's really first line right, because those often, those individuals Exactly. They've already gone through so much to get to that point. But in 2001, the. American Psychiatric Association, I'm gonna get that, that acronym wrong, but they basically came out and they were like, ECT should not be last line therapy. ECT should not be the thing that we think of when everything else has tried and failed so many times. And yet still, that's what it often is, which is unfortunate.

EW: It is. I mean, I think it's where like timeline, the, the, the timeline, the urgency, the severity all are kind of weighed within this. Catatonia is interesting because the person is non-responsive

EAU: Yeah.

EW: still an informed consent process. Can you tell me what that's like?

EAU: Yeah, it's, it is an interesting. For all of the cases that we are going to use ECT, I think that the informed consent process is a really interesting and incredibly important part of that process because even if someone is not so severe that they have catatonia where they're not responding at all, but even if they have really severe depression, their sickness, their illness is causing them to not care in many cases about getting better. Right. And not always. That's not, it's a generalization, but that, like, that is part of the nature of so many of these disorders. So the informed consent process is, is quite long and detailed. If someone is not able to make decisions for themselves, and this is actually true for all procedures, not just ECT, um, but then we have what's called a surrogate decision maker. And so you have to identify who that person is. Is it their spouse, is it their parent? Um, is it someone that they have designated as their healthcare power of attorney, which is so important that everyone has a healthcare power of attorney to make your decisions if you can't make decisions for yourself. Um, but that's really extra important. And family and or friends or whoever those people are, are usually always involved [00:40:00] in the ECT process. Even if someone you know is able to respond, it's usually recommended that it's kind of, everyone is on board with this. But of course, in the case of catatonia or when someone is unresponsive, then yes, it's someone else who is going to be going through that informed consent process for them.

EW: Okay. Okay.

EAU: Yeah. So yeah, Erin, that is what we know about ECT and how it works, and I ended with talking about ECT in catatonia and NMDA receptor encephalitis because now. We get to pause and be joined by Dr. Leonardo Lopez, who is an expert in ECT, including in the use of ECT for catatonia, including in cases of NMDA receptor encephalitis, and I am really excited to be joined by him. So let's go.

EW: Let's go.

Dr. Lopez, thank you so much for joining us today.

EAU: Yeah, we're really excited.

Leonardo V. Lopez: My pleasure.

EW: Could you give us a sense of how you use ECT in your practice? Some of the, the diverse applications that you use ECT for?

Leonardo V. Lopez: Sure. Absolutely. So, um, we use, uh, ECT for a variety of illnesses. Largely illnesses that have traditionally been considered psychiatric, although there are other conditions, um, that are sort of. In a more liminal space that we also use ECT for the most. Uh, most common indication is for treatment resistant depression, patients who have depression, and have received medication for depression therapy, uh, and have, uh, not responded and, and remain ill. And then there are a variety of others. So we use it for, um, bipolar disorder, both in, uh, the depressed phase and in the manic phase. we use it for what's called psychotic depression, which is a form of depression where patients, uh, also have delusions, uh, and or hallucinations. Um, use it in schizophrenia, particularly in patients who have not responded to treatment with antipsychotics or, uh, who cannot tolerate them. Then we use it in catatonia, which is a neuropsychiatric condition characterized by changes in speech and motor behavior that sort of spans, uh, a number of diagnosis. So it can occur with schizophrenia, depression, bipolar disorder, but can also occur. Um, in a variety of medical illnesses, uh, including autoimmune diseases like lupus, autoimmune encephalitis. And a variety of other, uh, neuropsychiatric conditions. Those are the most common indications. There are other things that we do use it for outside of the psychiatric realm for, um, for example, it can be used to treat, super refractory status epilepticus when patients, are seizing continuously and are on high doses of antiepileptic drugs, and yet their seizures

have not, uh, been brought under control. But it's most common usage is, is in what's now considered the psychiatric realm.

EAU: How much at the hospitals or hospital that you work in, uh, patient volumes, are you seeing, is it sort of an everyday practice for you or kind of, how common is it that you're, that you're using ECT.

Leonardo V. Lopez: Uh, it's very common where, where we work. It's an everyday practice for us. Um. We, we are scheduled three days a week, although, uh, I oversee services at, at two different hospitals. And, um, the days are different. So it ends up being a five day, a week, uh, practice. I can tell you that, you know, the average patient will receive 10 to 12 treatments, some more, some fewer, and a given year we perform. Uh, in excess of 6,000 treatments, between the two hospitals, that I oversee. So, um, quite frequent. on a given day, we might treat, you know, anywhere between 25 and 30 patients at one campus, depending, um, that includes both patients who are hospitalized for psychiatric reasons and patients who are coming in from home, and receiving either maintenance treatment, meaning they responded to the treatment previously and they're receiving it to prevent relapse. or they're ill but not enough to be, uh, in the hospital and so they, they're brought in from home for treatment there. Occasionally includes patients who are hospitalized on medical floors rather than psychiatric floors in the hospital, and occasionally patients who are in the intensive care unit.

EW: In the firsthand account that you shared of, of that young child with anti NMDA receptor encephalitis. I mean, there was clearly such a profound impact and throughout the episodes we have discussed how effective ECT has been and shown to be in treating a multitude of conditions, yet there is so much resistance. Of course, there's like, from the, the general public side of things, the fear and the stigma and misinformation, but even among medical fields, there seems to be. of a divide or a reluctance. How do you think the perception of ECT across different fields of medicine contributes to [00:45:00] Its under utilization.

Leonardo V. Lopez: I think that, insofar as there's reluctance in other medical fields to think about or use ECT, which I think there is, it largely stems from a lack of exposure. If people have not seen, patients treated with ECT, both just witnessed the, procedure with their own eyes and seen the progression of the patient when they're treated, um, the only exposure they have to it is, you know, in a textbook in medical school or the many, many depict. Uh, that they see in the media and all the, their medical professionals, and they know those, those, uh, depictions are often inaccurate. Um, still they leave a, a mark, right? Um,

they, they leave an impression. I think what we find is that when we have a colleague in another discipline, with, whom we're collaborating and treating their patient, and they see the procedure and see how, uh, unremarkable it appears, and, uh, also see the, the, the development, then, um, they become. Converted is, uh, if you will, to the utility of the treatment very quickly. So most of this is about exposure. I think, the more that, we are able to. Show our colleagues in other fields the value of this treatment, the more they support it. I think that's been a big reason why we have tried, at my hospital to sort of be very forward facing with other departments with, as I mentioned before, with the ICUs, um, with the pediatrics department who took care of this patient with the Medicine department, and try to get them involved and show them, um, you know, what it is we can do, um, because we need as much support. as we can get, to make this treatment available to all the people who could benefit from it.

EAU: Kind of along those lines, you know, whether it's sort of availability or, or the procedure itself. What do you see as maybe some of the, the biggest limitations that are facing ECT today?

Leonardo V. Lopez: I think the biggest limitation is access to treatment. Without question, obviously we've, uh, maybe that sounds contradictory given the numbers I gave you earlier. I think we've worked very hard at our institution to establish access to treatment because we really believe in this, in this therapy. But nationally it is not, easy to find, ECT providers. It's not easy, uh, if you can find one. To find one who can do it, at the sort of large scale that's sometimes required given the number of patients that can benefit from it. and then there are a variety of sort of specialty areas. For example, we may get to down the road, there are more and more patients, in the pediatric population with autism, with autoimmune encephalitis being identified as catatonic. And there's much less experience even in the ECT community treating those patients than there is treating adults. So, um, so finding, uh, someone who can, who can provide this treatment is very difficult. I think. Um, the patient with autoimmune encephalitis you may have mentioned, came from out of state and we've had numerous patients come, uh, particularly pediatric patients come to us, uh, although not exclusively, also adult patients, uh, come to us from out of state, simply because they could not find the treatment readily available in their area or they could not find treaters with experience, in their area. So to me, that's the biggest, that's the biggest barrier. and there, there are a variety of reasons why that's the case, but we need to overcome that first.

EW: So looking to the future now, I'm, I'm, I'm guessing that one of the things you hope to see is, is increased availability. Increased access. What are some of

your other hopes for the future of ECT say in the next, you know, 5, 10, 15 years?

Leonardo V. Lopez: Um, oh, I have so many. So, um, yeah, so cer certainly, uh, access is, is a big one. And, um, you know, we take great pride, in being able to bring patients in from out of state and, and treat them. Um, but of course it would be better if they didn't have to travel, um, for treatment and they could just, uh, receive treatment at home. So I think that's the biggest one I would like to see more acceptance and engagement around the use of ECT outside of the psychiatric community, the, the, um, medical, pediatric, um, critical care communities often take care of catatonic patients because they can't be taken care of in a psychiatric setting or because their catatonia is not due to a psychiatric illness. and, and those are patients that, often even if they're in a place where e CT is accessible, do not receive it simply because there's not the knowledge or comfort on the part of their, primary teams taking care of them. So I'd like to see, you know, more, More spreading of knowledge to those communities and also more spreading of knowledge by those communities, more advocacy, um, from those communities, which I think we have, uh, started to see particularly in pediatrics in the last few years. I'd of course like to see a greater understanding of mechanism. I think we, we have a much better understanding of, um, what [00:50:00] ECT does to the brain than we did, uh, 10 or 20 years ago at, at this point. but still we can't say with, uh, great certainty, why it works. And in fact, we can't say. um, the reason it works for depression is the same reason that it works for catatonia, or the same reason it works for psychosis. it's a global treatment, so it may have different effects on different illnesses, and we don't understand that. So, um, I'd certainly like us to make progress, uh, in that area.

EW: It is such an incredible. Therapy to to think about. This is one of our oldest

EAU: Yeah.

EW: therapies in psychiatry. It's still around today and there's so much misinformation surrounding it, and so I think that it has been a real eye-opening experience to kind of get to research these two episodes, to get to chat with you and hear about all of the different applications of ECT.

EAU: Right.

EW: yeah, thank you so much for taking the time.

EAU: Yeah. Thank you so much.

Leonardo V. Lopez: It's my pleasure. I, I think, uh, I appreciate it more than, than you guys do because, um, we need, we need, every outlet we can to, to, to give, real positive and, you know, fact-based information to the public. Um, because there is so much information to given to the public that's, uh, not reliable. and sometimes that drowns out our voices. So, uh, I'm very grateful to you guys for having me on.

EW: That was, that was so great. Thank you so much Dr.

EAU: Thank you so much.

EW: us. So informative,

EAU: all of your expertise, it's, it really makes a huge difference to have someone who like really knows we try our best, but let's be honest.

EW: We try, we try, but it, it really does

EAU: Yeah.

EW: Um, that, that was fascinating. Uh, you know, Erin, I had like one last quick question

EAU: course you do.

EW: ECT is often mentioned alongside other therapies that are similar

EAU: Yeah,

EW: like TMSC do, can you like, do you have like a, a brief

EAU: little brief one.

EW: about him?

EAU: I do, Erin, 'cause you know, I know what questions you're gonna ask.

EW: It's

EAU: I know your brain. So yes, TMS, uh, or transcranial magnetic stimulation. Uh, is the other like thing that people often talk about when they're

talking about ECT for especially affective disorders like depression. Um, technically it's RTMS 'cause it's repetitive TMS, but that's neither here nor there. Basically, this uses electromagnetic stimulation, so using a really strong magnet to generate a magnet. Magnetic field that's able to then induce an electric field in our brains and depolarize our neurons in the, in some way. That's kind of the same thing that we're doing with ECT, just using a magnet,

EW: Yeah.

EAU: Which means that the electric field that it's generating is not nearly as strong as it is in ECT, which means it's does not stimulate a seizure. Okay. What's interesting is they usually do it, um, they stimulate muscle contraction of the thumb, and that's how they know that they're in the right spot and doing the right thing. Isn't that interesting? One of the big benefits though of TMS compared to ECT is that the risk of cognitive effects, like memory loss is substantially lower. We basically don't see it, we don't see the same types of amnesia that we see with ECT. However, most of the data does suggest that while it is probably effective and more effective than placebo, it is still not as effective as ECT in terms of remission and response. Um, especially for depressive disorders, which is mostly what we're using TMS for.

EW: Okay.

EAU: There's also, sometimes people will talk about other things like DBS or Deep Brain Stimulation or VNS, which is vagal nerve stimulation. These are things that we mostly use for either like Parkinson's in the case of DBS or refractory epilepsy in the case of VNS. But these like require the implantation of devices, and so those are kind of different.

EW: yeah.

EAU: And then there's also interest in actually using magnetic stimulation to induce seizures

EW: Okay.

EAU: If that somehow by using magnets to induce the electric field and then also get the seizure, which we know is the main thing that's giving us benefit in ECT. Does that have less memory effects? Um, but as far as I could tell, that's still just in the research realm. So we don't have like availability of that yet.

EW: Okay.

EAU: It's a great question though. And do you know what? People might have so many more questions after this and I can tell you where to learn more.

EW: us, tell us.

EAU: A book that I actually loved so much, which was by Kelner from 2012. It was called Brain Stimulation in Psychiatry, E-C-T-D-B-S-T-M-S and other Modalities. It was a very comprehensive book. Um, really loved it. There was also, uh, honestly, there was so many papers, one by Merkel at all from 2009 called Antidepressant Electroconvulsive Therapy, mechanism of Action, recent Advances and Limitations. Um, another one by Critzer et al from 2023, so much more recent. An electroconvulsive therapy mechanism of action, clinical considerations, and future directions. Honestly, I I have a very, very, very long list,

EW: sure that you do.

EAU: so I'm not gonna read 'em all, but you can find them all on our website. This podcast will kill you.com, where you can find the list of sources from this episode and everyone that we've ever done.

EW: [00:55:00] Yes, a big thank you again to Dr. Leonardo Lopez. That was so helpful, so wonderful. And check out the Advances in Care episode if

EAU: Yes.

EW: more about that story of the NMDA receptor encephalitis

EAU: Yeah, thank you to

EW: Blood

EAU: Mobile for providing the music for this episode in all of our episodes.

EW: Yes, thank you. Thank you to Lianna and Mark and Jess and Pete and everyone at Exactly right. Who helps make this podcast happen. We really appreciate

EAU: We couldn't do it without you, and we also couldn't do it without you, listeners and watchers. Uh, so thank you. Thank you. Literally, thank you so much.

EW: Truly, really, truly, it's, yeah, you

EAU: Yeah.

EW: this, we'll, keep making it as long as we can.

EAU: yes.

EW: thank you to our wonderful, generous patrons. We truly appreciate your support. It means everything.

EAU: Does.

EW: Uh, well until next time, wash your hands.

EAU: filthy animals.