

03| Neuroimaging and Neuropsychology, Friends or Foes? (Part 1) – With Dr. Steve Correia

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Speakers: Steve Correia, Ryan Van Patten, John Bellone



Intro Music 00:00



Ryan Van Patten 00:17

Welcome, everyone, to Navigating Neuropsychology: A voyage into the depths of the brain and behavior. I'm Ryan Van Patten...

John Bellone 00:24



...and I'm John Bellone. We interviewed Dr. Steven Correia who is a board certified neuropsychologist with extensive experience using various imaging techniques, both in research and in incorporating imaging into his clinical work. Steve is an Associate Professor in the Department of Psychiatry and Human Behavior in the Alpert Medical School of Brown University. He is the Neuropsychology Section Chief at the Providence VA Medical Center, and is the Director of Brown's Clinical Neuropsychology Specialty Program for postdoctoral training. Steve's research focuses on using diffusion tensor imaging to examine the cognitive and behavioral correlates of white matter integrity in aging, dementia, and other conditions.

Ryan Van Patten 01:14

Just for transparency, we'll let everyone know Steve was a supervisor to each of us. So we're both very familiar with both the breadth and the depth of his knowledge in neuroimaging, which is really what drove us to ask him to be a guest on our podcast in the first place. We're both thrilled that he accepted.



One more disclaimer: we recorded this episode in one shot, but we ended up breaking it up into two parts. We might do this on occasion if it feels like there's a clear separation within the episode. For example, this first half is really an overview of neuroimaging basics, some important terminology, and a general overview of the most common types of imaging technologies. Whereas, on the other hand, the second part is more of a broad discussion on interdisciplinary practice, training in neuroimaging, and daily clinical practice. So now we give you Stephen Correia.



Transition Music 02:06



Ryan Van Patten 02:16

All right, we are here with Steve Correia. Welcome to the podcast, Steve.



Steve Correia 02:20

Thank you for having me. Honored to be here, actually,



Ryan Van Patten 02:22

Yeah, we appreciate it. So we're just going to go ahead and launch off. Steve, tell us a bit about your background, your training in neuroimaging, and how you got the knowledge and skills you have.

Steve Correia 02:32

Sure. Before I dive into that, though, let me just say that, again, thank you for having me here. I feel fortunate to be here. There are lots of people with lots of imaging experience and so I just feel like I was in the right place at the right time. I appreciate you guys inviting me to do this.



So, how did I get started in imaging? I was thinking about this. You know, I think the first time that I really got enamored of imaging was after college. I worked as a mental health worker at a psychiatric hospital and we had this one particularly young man who was very behaviorally dysregulated after a severe traumatic brain injury. I used to shadow the behavioral neurologist that we had and I was fascinated the day he brought in the films of the guy's brain injury and talked me through how the frontal injuries that he had mapped onto this patient's behavior, which was pretty difficult to manage. And so that really got me going on this whole thing. I had already had some interest in the brain. I'd gone through a psychology training as an undergrad - my favorite course was physio - and I had read some popular psychology books about the brain and so forth. So that really triggered me. Then I took a career diversion and kind of came back into psychology and, again, fascinated by the brain anatomy courses that I took. Fortunately, when I started my practicum training with Dr. Malloy at Butler hospital and at the time at the Providence VA, Dr. Malloy had expertise in imaging and would walk me through the scans of patients when they were available. Then later on in my postdoctoral fellowship, again with Paul Malloy and also with Dr. Salloway at the Memory and Aging Clinic, walking through scans and became really interested. Going to radiology rounds and those sorts of things. Then I decided to take the step into doing a little bit of imaging research and that's when the learning curve really went steep.



Ryan Van Patten 04:31

Yeah.



Steve Correia 04:32

Yeah.



John Bellone 04:32

Before we go into the weeds of imaging, which we'll do, let's take a big picture perspective - neuroimaging for neuropsychologists. So what are common scenarios where clinical neuropsychologists encounter neuroimaging?

Steve Correia 04:49



Right, well, I can only really speak from my own experience in what I see around me and my colleagues. I think most of the time when we encounter imaging, we get a referral question and discover that there's imaging in the record. The imaging may or may not be related to the purpose for which you were consulted. I think there's a fair amount of times when the imaging was done some time ago for some other reason and we sort of get it as an adjunct to our referral questions. Again, depending on the setting. I think if you're working in a setting - for example, an epilepsy center where the imaging is part of the workup for the condition for which you're seeing the person. In a general setting, that might not be the case as much. So I do think it relates to the setting. But my experience is that, a lot of the time, I've seen images that were acquired for a reason peripheral to or maybe not directly related to the reason I'm seeing the patient.

Ryan Van Patten 05:53



And, of course, a lot of neuropsych populations can benefit from neuroimaging, right? It's not just restricted to a few. We tend to think of stroke or TBI, but there are a broad range of different populations that can benefit. Which populations both adult and pediatric do you think seem to benefit the most or to be most frequently referred for imaging?

Steve Correia 06:19



Well, I'm an adult neuropsychologist, I don't know what the base rate is for pediatrics. I think the question really is, is there a clinical need for the imaging irrespective of the person's age? If there's a need for imaging, then usually some clinician will have ordered it. Or perhaps you as a neuropsychologist might, after having seen the patient, might think that neuroimaging would be helpful in your differential diagnosis. So I don't know if there's a difference by age; others might disagree with me. In fairness, I think as we age we have more opportunities to acquire insults to the brain. And so I think there might be more reasons for an older person to have imaging than a child or an infant. But there are plenty of reasons for them to get imaging as well.

John Bellone 07:15



And similar to neuropsych testing, some patients are going to have just one scan, while others might have serial scans across years. I realize that neuropsychologists typically don't directly order these scans, but what are some important factors to consider when thinking about repeated neuroimaging?

Steve Correia 07:36



Sometimes you're fortunate to have seen repeated neuroimaging that was done to monitor some disease process - for example, somebody with an arteriovenous malformation that neurology is following. They want serial imaging. I think you're talking more about the situation where you're thinking you have imaging from some time in the past, and you've seen the person and now you want an update on that imaging. I think that's what you're asking me. Is that? Have I got that right?

John Bellone 08:10



Yeah, just important factors to consider when you're thinking about imaging.

Steve Correia 08:13



I think important factors to consider would be: Is there a clinical need? Will the information that you obtain from imaging help you in the differential diagnosis or formulating treatment recommendations? I mean, after all, it's patient care that we're after, right? We think of imaging as having very low risk, and that's true, but it's not no risk for some people. I think exposing or recommending neuroimaging because you have an intellectual curiosity that doesn't bear on the referral question, I wouldn't recommend it. [laughs] Some might. I think it's useful in a situation where you think it would help you with a differential diagnosis. In my experience at the Providence VA, often I'll see that a patient is referred for memory loss and there's a CT image from 8 years ago or 10 years ago when the person fell and hit their head and had a very minor concussion. They came in and got a CT scan in the context of an acute head injury. They have a period of good recovery and now there's a decline. And the question is, why is there decline in a case like that? Imaging might be helpful, depending on what you find.

John Bellone 08:38



We'll talk a little bit more about the reasons to order the testing or to request that it's ordered in a little bit. But, that's perfect.

Steve Correia 09:52



Does that answer your question?

John Bellone 09:53



Yeah, absolutely.



Steve Correia 09:54

Okay. Great.

Ryan Van Patten 09:55



So we wanted to ask a few broad introductory questions at the start. If we've gotten through some of those, I think before we move forward into more conceptual questions, it might be helpful for listeners to get a brief background of core foundation and some terminology frequently used in imaging. Of course, some people will be familiar with these terms and some won't, but we'll cover them pretty quickly.



Steve Correia 10:18

I'll do my best. [laughs]



Ryan Van Patten 10:19

So to start, if you can just run through the basics in the differences between CT, structural and functional MRI, DTI, and PET.



Steve Correia 10:31

Okay. You guys recognize the futility of talking about images in an audio podcast, right? [laughs]



John Bellone 10:39

[laughs]



Ryan Van Patten 10:39

Yes. [laughs]



Steve Correia 10:39

You're explaining a picture. Like, this is a CT scan, this is an MRI. They look different. [laughs]



Ryan Van Patten 10:44

[laughs]

Steve Correia 10:45

We can try to put those into words a little bit. Basically, at the foundation, I think the source of the signal that generates the image is different. In CT, the source of the signal is X-ray. So you're bombarding the tissue of X-ray and the image is rendered based on the radiodensity of the tissue that the X-rays are passing through.



Whereas, with MRI, the signal that generates the image are hydrogen spins. Hydrogen have protons at a positive charge, they spin, they create a little magnet, and they interact with a magnetic field in the scanner, and you get an image. That's a gross oversimplification. [laughs] The physicists out there would have my head for that one. But, yeah, so one is X-ray and one is looking at protons spins with no radiation exposure. What else did you ask about? Did you ask about DTI?

Ryan Van Patten 11:47



DTI and PET.

Steve Correia 11:52

Well, DTI is an MRI technique. It's an MRI technique that looks at, characterizes the speed and direction of water diffusion in tissue. It's great in the brain because we have white matter and water flows along white matter axons, at least myelinated white matter axons, more quickly than it flows across them. We can use that as an indirect marker of white matter integrity. All tissues have diffusion, too, and DTI is not strictly limited to the brain although I think that's where it gets a large application. PET is different. PET is molecular imaging where there's an IV administration of a molecule, that radioactive tag. And then the signal that produces the brain image is the radioactive decay in the brain as it passes through.



Ryan Van Patten 12:57

Okay. Yeah, that was exactly what I was looking for. With CT and MRI, I'm familiar with a few pros and cons from a practical perspective. I'd like to run through those and then you tell me if and where I'm wrong. [laughs] So, CT tends to be cheaper and more readily available. MRI tends to be more sensitive to soft tissues. CT tends to be a little bit better if there is a skull fracture. Anything else?



Steve Correia 13:26

I think that's correct. CT tends to be a little bit more available. CT scanners aren't as expensive or hard to site as MRI scanners are because MRI scanners are heavy and as there are siting issues and shielding issues. With CTs as well. But, yeah. I think the CT is X-ray sensitive to bone density and MR doesn't show bone very well, because bone doesn't have a lot of hydrogen.





Ryan Van Patten 13:54

Right. Makes sense.



Steve Correia 13:56

That's basically it. And CT is often used as a quick screen. It's a little less expensive. I mean, CT is fine for a lot. Advanced CT imaging is really quite good.



Ryan Van Patten 14:10

Right.



John Bellone 14:11

Maybe we can dive a little bit further into MRI. Can you talk a little bit about the similarities and differences between common sequences like T1, T2, FLAIR?



Steve Correia 14:22

Also difficult without pictures. [laughs]



John Bellone 14:24

[laughs]



Steve Correia 14:25

So I'll do my best. You'll hear people talk about the T1-weighted images or the T2-weighted images. So what does that mean? Gosh, it's a little tricky to describe. In MRI, as I mentioned, the signal comes from protons spins and there's two sources of that signal. One is the T1 signal which is a signal that's given off when you perturb the spins of the protons so that you have flipped their alignment against the magnetic field and then wait for them to kind of come back and give off energy. And that energy is captured as a signal, as a T1 time. There's a second source of signal that comes from dephasing of the spin. So protons interacting with one another and giving off their energy that way. That's, again, a gross oversimplification. It's hard to describe this without pictures and graphs. So those two terms, T1 and T2 have to do with the different sources of the signal. That said, what do they look like, right? So, in general, for the T1-weighted images in the brain, the ventricles are going to be dark, CSF is going to be dark. And on the T2-weighted images, the CSF is going to be bright. That's about the most dramatic difference. The gray matter and white matter appear inverted across those two images. But, yeah, it's a little tricky to describe, again, without a picture.



John Bellone 16:06

Yeah. And FLAIR? FLAIR is another one that we hear often.

Steve Correia 16:09

Yeah, FLAIR is a common acquisition in brain imaging. So FLAIR is an acronym. It stands for fluid attenuated inversion recovery, and I'm not going to unpack that for you. [laughs] I'm again oversimplifying, and the physicists in the world are going to have my head. But I think it's helpful to think of FLAIR as sort of a combination between T1 and T2. It's really a T2 sequence in which there's a physics trick done in the magnet so that the water signal from free flowing CSF is suppressed. So it tends to look dark, as it does on a T1 image. Whereas water that's embedded in tissue from edema or something shows up as bright so that your eye can see the lesions a little bit better. See those white matter hyperintensities a little bit better than you could if everything is bright.



Ryan Van Patten 17:11

T1 is better for anatomy, and T2 is better for pathology. Correct?

Steve Correia 17:15

In general, that's how they're often thought of. Although pathology shows up on both types of scans, right? So the T1 image is nice for, again, for pathology, that sort of traditional - I mean, excuse me, for anatomy. The reason that T2 is useful, or at least my understanding of the reason why T2 is often useful for identifying pathology, is that most pathologies in the brain have increased water content in them. So they'll show up as bright on the T2 image. But, again, because all the fluid is bright, it's sometimes hard to see those images, especially if you're looking at the white matter. An area of high signal in the white matter that's right up against the ventricles might be hard for you to tell where that boundary is on a T2 image. Whereas in a FLAIR image, because the ventricles are going to look dark, you can see the boundary a little bit better between what is the normal CSF and water that's in the tissue. I don't know if that helps.



Ryan Van Patten 18:13

It does.



John Bellone 18:14

Yeah. And just for any students who are listening right now, I want it to maybe back up just a second. So CT is computed tomography, and MRI is magnetic resonance imaging.



Steve Correia 18:24

Oh, yes. Yes.



John Bellone 18:25

DTI is diffusion tensor imaging. And then PET is positron emission tomography.



Steve Correia 18:30

Yes.



John Bellone 18:31

Then, in terms of the anatomical plane, there are three. Axial, you can think of as top to bottom. Sagittal is side to side. And coronal is front to back. These are usually presented in 2D, so slices, but some software does allow you to see them in 3D as well.



Steve Correia 18:50

Yes. Well, it depends. Yes, there's software that allows you to visualize, kind of render the brain in three dimensions. So it appears like a 3D object on your two dimensional computer screen. Although, at Brown, we had people who were doing things in, like, holographic space, which was kind of neat at one point.



John Bellone 19:12

What is holographic space?



Steve Correia 19:13

Well, just sort of, like, in suspended 3D space. You could walk around the image.



Ryan Van Patten 19:20

We have those clinically?

Steve Correia 19:21



[laughs] I don't know if that facility exists anymore at Brown. But there are probably other sites that have that technology. When you talk about rendering in 3D like that, to do that, you really need to have acquired your data in 3D, basically. So that the image voxels are cubes and not bricks, if that makes sense. Because then you can reformat it in any direction. If you have brick so that the in-plane resolution is different than the through-plane resolution, then it's harder to render those in 3D.

Ryan Van Patten 19:37



In terms of the orientation of the scan we're looking at, radiologic convention is flipped, correct? So on my left is the right hemisphere, and vice versa.

Steve Correia 20:19



Right, as you look at the scan on the screen. If you're thinking of looking at an axial section, for example, so you're looking at a computer screen, the eyeballs are in the top half of the computer screen, the back of the heads of the bottom of the computer screen. What appears to you on your right as the audience, is the patient's left.

Ryan Van Patten 20:46



Right.

Steve Correia 20:46



There's lots of ways to remember that, but.

John Bellone 20:49



One way I think of it is, it's as if the person is lying down and their feet are closer to you. You're kind of looking through the bottom of their chin almost into the brain.

Steve Correia 21:02



That's one way to look at it. You can also think of it as, like, audience right and stage right. They're swapped. [laughs]

Ryan Van Patten 21:08



Yeah. There's also a neurologic convention, right? Which would be the opposite of radiologic convention.



Steve Correia 21:15

In neurologic convention, left is left and right is right.



Ryan Van Patten 21:20

So how do we know? If we're looking at a scan, how do I know if it's radiologic or neurologic convention?



Steve Correia 21:26

That's a good question. Clinical images, by convention, are displayed in radiologic convention so that your right is the patient's left and vice versa. The neurologic convention is used more commonly in research settings. It may be used in some clinical settings, but by and large, you're going to see radiologic convention. And there should be some clue somewhere on the image. And often you'll look at the image and it'll tell you some information about the image - how it was acquired, technical information like echo time, TE time, and TR time, and so forth. It may tell you right on the scan. There may be a little L someplace or a little A someplace so you know what's up and down and left and right.



John Bellone 22:20

That's how I know. [laughs]



Steve Correia 22:21

That's how you know, right? In the old days, you had films and you had to know. [laughs]



Ryan Van Patten 22:26

Right, right.



Steve Correia 22:27

Yeah.



John Bellone 22:28

And the other thing is, sometimes there's contrast. So sometimes you'll see a scan and it'll be, you know, CT with and without contrast. Can you tell us a little bit about what that means and why contrast is used?

Steve Correia 22:41



So contrast is used to show the vasculature in the brain. That's basically what it is. By and large, for most clinical applications in CT, the contrast agent is iodine and it's administered intravenously. The MRI techs can do that, they're trained to do that. CT techs, and same for MRI techs, in general medical centers, they're trained to administer the contrast agent. The contrast agent in MR is gadolinium which is in the periodic table, just like iodine. Don't eat raw gadolinium. [laughs]



Ryan Van Patten 23:18

Tip of the day. [laughs] A few basic...



Steve Correia 23:23

But the gadolinium that is administered is chelated.



Ryan Van Patten 23:27

Okay.



John Bellone 23:28

So we can if we want... [laughs]



Ryan Van Patten 23:30

We could consume it, if we wanted to. [laughs]



Steve Correia 23:32

Ohhh, I wouldn't. I wouldn't. [laughs]



Ryan Van Patten 23:34

Fine, okay. [laughs] Some basic brain pathology. How do you define atrophy?

Steve Correia 23:41



Atrophy is the lesion of absence, I guess. It's the, you know, the absence of tissue where there ought to be tissue. But not - one wouldn't look at a brain scan in somebody who had a significant head injury or stroke where there's encephalomalacia. There's liquid active necrosis because of an injury like that. That's different from atrophy. In atrophy, I think we're talking about just gradual tissue volume loss that can occur with aging and neurodegenerative disorders.



Ryan Van Patten 24:19

You mentioned both of the other terms I'd like to ask you about. So one in particular that is used very commonly, but I found it to be hard to nail down exactly what it means, is lesion. How would you give us an operational definition of a lesion?



Steve Correia 24:36

Well, I think it's a general term that pretty much refers broadly to tissue that's been damaged from trauma or disease. I think what you're asking is, you know, when we look at MR images and we see something there that we know in normal anatomy ought not to be there or there are signal characteristics. As you look at images, you start to develop a sense of what's normal and what's not. You see signal characteristics that are not normal, not what you expect to see. We might call that a lesion. So something that you see that doesn't belong.



Ryan Van Patten 25:19

Yeah.



Steve Correia 25:20

There are certainly artifacts that can masquerade as lesions or things like that.



John Bellone 25:25

The artifact just means something that you see, but it's not necessarily pathology.



Steve Correia 25:31

It's not physiological necessarily. It can arise from the interaction between the physiology and the scanner. But, usually, it's some sort of noise. It's caused by - there's a long list of things that cause artifact - movement, radio waves getting into the MRI scanner. Yeah, lots of things.



Ryan Van Patten 25:53

You mentioned encephalomalacia. My understanding is that the term means "soft brain" and was used more by neuropathologists performing autopsies in the past, when you actually were touching and there was a tactile softness to the brain. But now it's moved into imaging, is that correct?



Steve Correia 26:11

That's correct. Yeah. That's basically correct. Softness or loss of tissue, again, caused by trauma, vascular abnormality, infection.



Ryan Van Patten 26:21

Okay.



John Bellone 26:22

You also said necrosis, which is just the loss of cells. The death of neurons.



Steve Correia 26:27

Yeah. That's what I mean by that. A pathologist might have a more specific term, but that's what I mean.



John Bellone 26:32

[laughs] Just broad strokes for our listeners is perfect. So I've seen a lot of terms that seem to describe white matter pathology - leukoaraiosis, small vessel ischemic disease, subcortical ischemic vascular disease, white matter changes, white matter lesions, white matter hyperintensities. Are these all the same? Or is there a way to differentiate them? Or should we even go into that?



Steve Correia 26:59

Right. These terms, typically, are used in aging populations, when we're looking at images of folks who are aging. And they're somewhat interchangeable. I think there's subtle differences between them. The term leukoaraiosis derives out of CT imaging, actually. Vladimir Hachinski was the one who coined the term to describe white matter rarefaction, which shows up as dark or hypodense on CT scan. The other term, white matter hyperintensities, would refer to MR. And notice that it's hyperintensities. So, in CT, we talk about the radiodensity of tissues so things are hypo (darker) or hyper (brighter) dense - hyperdense, hypodense - relative to the surrounding tissues. In MR, hypo or hyper intense, because you're working with signal intensity.

In any rate, going back to your question about are these terms changeable. So leukoaraiosis and white matter hyperintensities mean similar things. The white matter hyperintensities - when I'm talking about this in any kind of presentation that I'm doing, a poster or a paper or a verbal presentation, I'm talking about those changes that we've seen in the white matter of older folks. We have high signal in a FLAIR or T2 image. White matter hyperintensities, to me, seems to be the most precise radiographic term because it doesn't imply what the underlying pathology is. The other terms, in my view, have some implication of the underlying pathology. So, you know, white matter lesions, subcortical ischemic vascular disease - one would

think that that's the underlying pathology that gives rise to the white matter hyperintensity.



John Bellone 27:07

And when we talk about white matter, we're referring to the myelinated axons because the lipids that make up the myelin turn up white on imaging.



Steve Correia 29:15

Yeah. Right. So on a T1-weighted image, the white matter appears a little brighter than the gray matter. And it's inverted on T2, the other way around.



Ryan Van Patten 29:33

Lacunar infarct? How is that different from the terms we've just defined?



Steve Correia 29:38

Okay, so the terms that you're defining, referred to - If I can just back up just a little bit because I don't know. If I get too technical, stop me. [laughs] So, we're talking about - the terms that you mentioned leukoaraiosis and white matter hyperintensity mean similar things. The white matter hyperintensities, whiter matter lesions, and so on. I think you know that those terms are often used to describe signal abnormalities, either on CT or MRI. The ones that apply to MR in the white matter, are usually the supratentorial but also it could be in the brainstem or the cerebellum as well - areas that there are signal abnormality. On CT, those tend to appear darker or hypodense. And on MR, hyperintense or brighter than the surrounding tissues, at least on a FLAIR image. So what causes the signal abnormalities? Well, again, it's water in the tissue. Usually caused by edema, usually caused [unintelligible] from hypoperfusion, due to vascular abnormalities and so forth. A lacune, on the other hand - my understanding and the way I've been trained is that a lacune is a small completed infarction. And it's the lake, the small lake in the brain, really. It's usually under 20 millimeters in size. Often found in the deep white matter or in the area of the basal ganglia in an elderly person.



John Bellone 31:18

Okay, so the underlying mechanism, it sounds like, at least to cause most of the white matter hyperintensities that we see on imaging - if I could just take a stab at it, you said a bit about it. But something like hypertension, right, high blood pressure could cause a hardening of the blood vessels and that leads to the reduced blood brain barrier permeability that leads to the hypoperfusion and the tissue isn't getting

the oxygen and nutrients that it needs. And so that leads to damage and inflammation, edema, which is the water buildup. Is that right?

Steve Correia 31:53



That's right. I mean, is it reduced permeability? Or is it increased permeability of the blood brain barrier? But, anyway, it's abnormalities in the vessels that the downstream effects are causing edema in tissue. I think that that's what that's causing. And if I could just back up for a second, so you're referring to the white matter hyperintensities that occur in aging from vascular pathology, right? But, again, white matter hyperintensities is somewhat of a nonspecific term. MS lesions appear bright on T2- and FLAIR-weighted images and they'll appear dark on T1, they'll appear hypointense. So I just want to kind of, for the listener, to know we're talking about the kinds of white matter lesions we're seeing in older folks without other known white matter pathologies.

John Bellone 31:53



Yeah, that's a really good distinction to make. Right. So there's vascular, and there's other...

Steve Correia 32:21



Right. And one can have multiple sclerosis, and one can have both.

John Bellone 32:58



Do other cerebrovascular risk factors like diabetes, hyperlipidemia - do those also have a similar mechanism to hypertension?

Steve Correia 33:07



There are all sorts of bad juju for the blood vessels. [laughs]

Ryan Van Patten 33:10



[laughs]

Steve Correia 33:13



Yeah. When we think about subcortical ischemic changes, we're talking about changes in arterioles, basically. Those of you in the audience and us who are used to looking at these images in older folks often see them in the regions of the lateral ventricles, either periventricular or the deep white matter - areas of the basal ganglia, maybe the pons, maybe the cerebellar white matter. But thinking about the

ones that are sort of periventricular and deep white matter, it's because those are end arterioles. They are often arterioles that don't connect well. They're sort of dead ends or they're in watershed regions. And so they're vulnerable to changes in the vasculature. What I know and what I've learned is that after age, the most powerful risk factor for those is hypertension. I think diabetes and hyperlipidemia are vascular risk factors. Certainly hyperlipidemia is a risk factor for large vessel stroke.

Ryan Van Patten 34:23



Okay. So, I want to take this in a slightly different direction. Earlier, we talked a little bit about DTI, diffusion tensor imaging, which I think in our field is becoming much more well known. A lot of people are very interested in research on DTI and I hear the term a lot. But DTI is an extension of diffusion weighted imaging. Correct?

Steve Correia 34:46



Correct.

Ryan Van Patten 34:46



And I've heard much less about DWI. So I think it may be helpful if you sort of walk us and our listeners through the basics of DWI. Is it ever still used? Is it just replaced by DTI? If so, what's it used for? Why don't we hear about it relative to DTI?

Steve Correia 35:04



Yeah, so, diffusion weighted imaging. You've seen diffusion weighted imaging. [laughs] It's often done routinely at medical centers in adults just to make sure that there's no ischemic stroke going on. It's quite sensitive to that at a certain point in time after the ischemia starts. And then after a period of time a lot of these lesions will evolve so the signal characteristics change with time past the injury. So, diffusion weighted imaging is common. It's a common clinical sequence used to rule out ischemic stroke. So you've seen it. And diffusion tensor imaging is diffusion weighted imaging in many directions. Diffusion weighted imaging is usually three directions, often three orthogonal directions. When I say directions, I'm talking about the image that DTI captures - the speed and magnitude of water diffusion. Those can be measured in different planes, in different directions through the anatomy. So diffusion weighted imaging is measuring the speed and magnitude of water diffusion in three orthogonal directions. Whereas, in DTI, you need at least six directions to do DTI. Nobody does six anymore. They do way more than that. I don't know if that helps or confuses you or the listeners. [laughs]



Ryan Van Patten 35:19

No, it does. I was hoping to get you to talk a little bit about DWI in case we have listeners who haven't really heard the term even though it's commonly used.



Steve Correia 36:45

Yeah, yeah.



Ryan Van Patten 36:47

A follow up on DTI - this is something I've thought about and I'm curious about your thoughts. So it seems to be very popular in neuropsychology and related fields. It's a great way to measure the integrity of white matter, right? It seems to be becoming a very sexy topic, in terms of research - people doing posters and such on it. And that's not necessarily a bad thing. I don't think that takes away from the validity of the technique at all. But I've wondered, are we getting mesmerized by tractography and these beautiful pictures at the expense of truly understanding the technique and what it is? Of why it's truly useful in its core?



Steve Correia 37:31

And what its limits are.



Ryan Van Patten 37:31

And what its limits are. Do you think that's the case? Or no?



Steve Correia 37:36

Well, they sure do produce pretty pictures. [laughs]



Ryan Van Patten 37:40

[laughs]



Steve Correia 37:40

A few years ago it was on the cover of National Geographic.



Ryan Van Patten 37:42

Yeah.

Steve Correia 37:45

So, yeah, the images are great. But I think the underlying information that DTI - I mean, I'm biased. My area of research is in DTI, so I'm biased. But I think it's useful. It is useful for characterizing the integrity of the underlying white matter. I think we, as neuropsychologists and other brain researchers, understand that the brain works in networks and understanding the cortical location of functions and the cortical pattern of a function is useful. But it's also necessary to know what are the underlying connections between those and I think DTI allows us to do that. So I think we grew up thinking about disconnection syndromes and so forth. So I think it's quite appealing to us to use DTI to look at those. And, yes, the tractography pictures when you're looking at the white matter fibers are useful. But I think it's important to keep in mind that those are computer graphic renderings of the MR, of the signal that's underneath there. They are not renderings of axons, which are on the order of 20 microns or less. Typically, for most diffusion tensor sequences, the resolution is one millimeter, sometimes sub-one millimeter. So I think we have to bear in mind what it is that we're looking at. There are also limitations. And there's a lot of technology and software development going into overcoming these limitations of diffusion tensor imaging. But, by and large, somewhat oversimplified, I think it's fair to say that DTI doesn't do very well in areas where there are crossing or kissing fibers. It has trouble - at least tractography - has trouble rendering in those areas. The tractography makes a nice picture, but so what? I think what's helpful is to know what the underlying metrics are that are driving that. That's probably a little bit more than we need. We can spend a lot of time talking about this. [laughs] I don't know if we want to do that in the context of this podcast.



Ryan Van Patten 39:57

Well, you answered my question. That's helpful.



John Bellone 40:00

So now, Steve, let's get to the provocative title of the episode. [laughs]



Steve Correia 40:05

What is the title of this episode? "Grill Steve on MRI". [laughs]



John Bellone 40:10

[laughs]



Ryan Van Patten 40:10

[laughs] That's our goal. But the title is "Neuropsychology and Neuroimaging, Friends or Foes?"



Steve Correia 40:16

Oh, my gosh.



Ryan Van Patten 40:17

Are we together or against?

John Bellone 40:20



So is neuroimaging a threat to neuropsychology in the 21st century? Could further advancements in neuroimaging make neuropsychology obsolete? I think it's interesting that the very first line in Lezak's Neuropsych Assessment text is the quote by Mortimer Mishkin that, "Imaging is not enough". She wouldn't have included that if people weren't concerned that imaging techniques might be the death of our field. So can you assure Ryan and me that we didn't just waste many years of our lives in training? [laughs]



Steve Correia 40:57

Well, "assure" is a strong word. [laughs]



Ryan Van Patten 41:00

Uh oh. [laughs]

Steve Correia 41:03



No, I think, and this is just my opinion, I can only speak from my opinion. You know, questions like this can be controversial, people might disagree, but I think we're not at a place and, particularly far from a place, where imaging is going to replace what we do. I mean, if you think about the historical context of neuropsychology - lesion, location, localization - why bother with that when you can just get a nice image and show you where the lesion is, right? So I think that the images help inform what we do, but the images don't capture a person's functioning. They don't capture the person's personality, they don't capture their cognitive function. Images are images, at least at this point in time. And, yes, there's functional imaging. In which case, you put somebody in the scanner, you give them a task, and while they're doing the task, you acquire images and you look at areas of the brain that have more activation than others. But those are discrete tasks. Or you do resting state

[functional imaging] and look at just the general areas of the brain that are active at rest. But, again, I don't think you can extrapolate from those sorts of information to how someone is going to perform in their daily lives. We have a hard enough time doing that with our neuropsych tests, right? [laughs] So can you imagine, "Oh, this image says that you're going to do X in such-and-such a situation." So I think we're reasonably far from that.

I do envision a possible time when, as imaging and quantitative techniques and the software for analyzing the images become more advanced, we could develop imaging norms. So you have an idea of like, "Well, how big should the superior temporal gyrus be in a 75-year old?" And, "What's the correlates of this degree of atrophy on some memory test or some function?" I think we can get to that point, but I think we're far from that. And, even if we did, there's a lot of individual variability. I mean, there's this whole issue of cognitive reserve. So yeah. That's my long winded opinion about your question.



John Bellone 43:37

Good. So for the foreseeable future, we're safe.



Steve Correia 43:39

I think you're safe.



John Bellone 43:40

We add incremental validity, you know, above and beyond what neuroimaging can show us.



Steve Correia 43:46

Right, but I did a fine job of trimming my shrubs yesterday. And, so, just in case, I could become a gardener. [laughs] I got a backup plan.



John Bellone 43:59

Well, that's it for Part 1. Don't forget about Part 2 coming out in just two weeks. Thanks again for listening. Join us next time as we continue to navigate the brain and behavior.



Exit Music 44:09

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