

## Shrink Rap Radio #265, June 3, 2011, The Polyvagal Theory with Stephen Porges, Ph.D.

**Dr. David Van Nuys, aka “Dr. Dave” interviews Dr. Stephen Porges** (transcribed from [www.ShrinkRapRadio.com](http://www.ShrinkRapRadio.com) by Athena Papadakos)

**Excerpt:** *“So if we’re in a state of fight flight mobilized, our ability to be friendly, to be social and even to be appropriately evaluative of others, is going to be compromised. We don’t recognize this in both our education or either in our medical, clinical world. We don’t realize that the person’s physiological state is really the window, the portal, to both learning and treatment modalities. Whether we are talking about psychotherapy, body therapies, or basic practice of medicine, we want to create physiological states that recruit neural circuits that not only support social behavior, but that support health, growth and restoration. And the beauty of the model is that the same circuits involved in social behavior, [the] social engagement system, are also the same circuits that are involved in health, growth, and restoration.”*

**Introduction:** That was the voice of my guest Dr. Stephen Porges speaking about what he has termed the Polyvagal Theory. Stephen W. Porges, Ph.D. is Professor of Psychiatry and Bioengineering and Director of the Brain Body Center at the University of Illinois at Chicago. His work on the autonomic nervous system has led to a new understanding of mechanisms involved in behavioral regulation and social engagement behaviors. He is developing new biobehavioral assessment tools to monitor individual differences in physiological regulation of behavioral states. His research has led to an innovative intervention: the Listening Project, designed to exercise the neural regulation of middle ear structures to reduce auditory hypersensitivities and to improve the ability to listen to and attend to human speech. Dr. Porges speaks throughout the world about his Polyvagal Theory and its applications to clinical and populations.

He is the author of the 2011 book [The Polyvagal Theory: Neurophysiological Foundations of Emotions, Attachment, Communication and Self Regulation](#). Now, here’s the interview.

**Dr. Dave:** Dr. Stephen Porges, welcome to Shrink Rap Radio.

**Porges:** Thank you, David. It’s a pleasure to be here.

**Dr. Dave:** Well, I’m really happy to have this opportunity to meet with you and to discuss your work. I got a kick out of discovering that we have some things in common. We both went to graduate school in Michigan: you at Michigan State, me at the University of Michigan. We both got our Ph.D.’s in the same year and interestingly, we both did research as graduate students on attention: you on attention in relation to the heart, and me in relation to altered states of consciousness.

**PORGES:** That’s very interesting. And I will say one thing: you’re probably a lot smarter than me because you’re not in the Midwest now, are you?!

**Dr. Dave:** (Laughs). Well if that's going to be the measuring stick, that's probably the only one that I would come out ahead on! You really continued with a career in research and I was just trying to get out of graduate school! I didn't follow up on my research into attention. But it's interesting to see how in the intervening years since way back when in the Dawn of Time, attention has really emerged to be a central concern of research. And that at the time that you and I were mucking about with it, there weren't any of the brain correlates, no good ways to measure attention.

**PORGES:** That's quite interesting because it really was a William James construct; that was clearly as it was defined by William James in the late 1800's, early 1900's. But maybe a good point to start with is, "How did I get from attention into Polyvagal?" Is that something you might find of interest?

**Dr. Dave:** Yeah, that's sounds great. Go ahead. Tell us that.

**PORGES:** I don't mean to lead the interview. But it really is kind of an interesting story because like many of us we were very interested in cognitive function and looking for other indicators of cognition, so if you put electrodes on a person you wouldn't have to ask them if they were attending. You could actually look at the physiological patterns and when I was doing this, I actually observed a very unique phenomenon. And that was when people start to sustain their attention, the heart rate patterns started to become more stable so the beat-to-beat changes in heart rate were reduced. And this was quite an interesting phenomenon because it was very statistically powerful and it was nondiscriminatory regarding the types of attention. So it was like reflecting an attentive state, and I got very excited about this but no one quite understood what was the mechanism of this change in heart rate variability. And then I noticed that people who had larger responses, meaning that they stabilized their heart rate more, and had faster reaction times, these were the same people who when they were in a relaxful state had more beat-to-beat heart rate variability. And that started the journey of trying to figure out what that beat-to-beat heart rate variability was all about and that's how the Polyvagal theory got started.

**Dr. Dave:** Ok well you've put the Polyvagal theory on the table and of course you've got a new book that just came out called the Polyvagal Theory, and you're widely known for this theory. I think a good place for us to start--well we've already started--would be with the word "polyvagal". "Poly" to me suggests "many", and many listeners will know there's something called the vagus nerve, but maybe not much more. So tell us, what does "polyvagal" refer to?

**PORGES:** It was actually as you've already come up with a description of what people would call "vagus" or "vagal tone" or "vagal activity", was really a non-descript term. It was too ambiguous because there were multiple vagal pathways. And it also forgot, or left out of the equation, the most important part of the whole vagal circuit, and that is the sensory or afferent component, the part that's bringing all of the information from the body up to the brain. Polyvagal, the term, was coined to appreciate the fact that when you're measuring vagal activity, it could come from different areas in the brain and it could be reflecting information coming up from the body.

**DR. DAVE:** Listeners will be familiar with the sympathetic and parasympathetic systems that make up the autonomic nervous system, but I don't recall that we used to hear much about a vagal system in the past. What's the relationship between the vagal system and the autonomic system?

**PORGES:** The vagus is the primary pathway for the parasympathetic nervous system. So most of the parasympathetic activity to our viscera comes through the vagus. It's a large meandering nerve, and that's why it was called the "vagus": it's because it was vagabond, it was moving around. And actually we're going to get to another point because when we look in the brain stem, the motor fibers were thought to come out of one are called the dorsal motor nucleus of the vagus. But when people started to study comparative neuroanatomy and the technology became better over the last two or three decades, they found out that in mammals there were vagal pathways coming from another nucleus and that one is called nucleus ambiguus. So now you have a vagabond nerve that comes from an ambiguous site!

**DR. DAVE:** Great!

**PORGES:** But the beauty of this whole circuit is when you start understanding about what's coming out of this area "nucleus ambiguus" it really creates a heart-based connection. It's an area that controls many of the activities of motor-fibers regulating muscles in the face and the head. So that's why when people speak with different intonation they're telling you really about their visceral state. They're telling you whether they're calm or anxious. As clinicians, you already know that, but there's a true physiology to that.

**DR. DAVE:** That's really fascinating. Part of the story involves the myelination of the nerves, so talk to us about that and the implications for our experience.

**PORGES:** There are two parts. If we look phylogenetically and just look at the evolutionary history of where we came from, we evolved from reptiles. That's part of our history. At the transition from reptiles to mammals, there are many interesting things that are occurring that are intricately related to social and emotional regulation. One is that mammals have this unique myelinated vagus that is no longer being regulated from the dorsal motor nucleus of the vagus but now from the nucleus ambiguus. Actually during embryology, during development, the source nuclei migrate, but that's really irrelevant to what most people want to know about. The issue is that myelinated fibers are really fibers that work very very rapidly. They communicate quickly. They are tightly regulated. And what those myelinated vagal fibers are capable of doing, they are capable of inhibiting sympathetic activity and calming us down. The issue is that what we often are doing is we have what I call a "vagal brake", and this is this myelinated vagus that sits on top of the sympathetic nervous system and can calm us down and slow our heart rate up [sic] and make us calm, but we can rapidly retract the vagal brake without stimulating the sympathetic so we can have increased metabolic activity. We can stand up. We can sit down. We can shake hands. We can look at people without really triggering sympathetic activity. So an understanding of the myelinated vagal system and the vagal brake shifts our conceptualization from a sympathetic-parasympathetic balance model, meaning that if you needed more heart rate you stimulate the sympathetics, to a model that says, "This is all put together hierarchically." And that you have a system that is basically a governor or brake upon a metabolic motion system

which requires sympathetics. And when we take this brake away, we then have this opportunity to mobilize very efficiently. But when we are in safe situations, we don't want this massive engine to be fired up.

**Dr. Dave:** OK. You mentioned the face. You said that the theory has two important parts: the first is the link between the nerves of the face and the nerves that regulate heart and lungs. Let's start with that first part about the face and hearing and heart and lungs. Tell us about that.

**PORGES:** If we think about what we know in terms of our social interactions, all this becomes extraordinarily intuitive. So that when we see people whose faces are flat, meaning they're not exuberant. They're not smiling. They often are not speaking with much intonation either. Their voice tends to be monotonic. They don't use expressivity. They tend to gravitate toward lower frequencies if they're male; if they're female they tend to be high pitched, non-modulated sounds. So the face is flat, the voice is monotone and they often, if you talk to them, they often aren't really listening to you. Their eyes may roll up. They may not be processing what you're saying. The point of all that is that the nerves that regulate the striated muscles of the face and head are all linked in the brain stem to an area that controls this myelinated vagus. If we think in terms of our evolutionary history, what was this all about? It was all about conveying to conspecifics, whether we were physiologically safe to come close to. If our heart rate is calm, modulated by this vagal brake, then the vocalizations will have prosody, then we'll be able to hear what people are saying, and then we'll be expressive. If we are in a physiological state that supports defenses, fight flight behaviors, then our face looks different, our ability to extract information through the acoustic channels changes, and we are conveying to the other person, the conspecific, and if it's not a person, another mammal, that you should not be someone that you should cuddle with. So it's this face-heart connection which evolved really as a transparent social communication; it made our viscera transparent to conspecifics. And something has been lost along the way. We've given up in a sense our natural intuition about people and we started to get very cognitive and started using words and syntax. And one of the take homes of this discussion will be that it's really not what people are saying but how they say it that affects other people.

**DR. DAVE:** Of course that's near and dear to my heart as a clinician. Going back to the second part of your theory, and I think you touched on this: there's a phylogenetic hierarchy that describes the evolutionary sequence from a primitive unmyelinated vagus, related to conservation of metabolic resources, to a sympathetic adrenal system involved in mobilization strategies, to a myelinated vagus related to modulating calm bodily states and social engagement behaviors. So there's a 3-step hierarchy. Can you say a little bit more about that sequence.

**PORGES:** Absolutely. Most of us when we were in graduate school and even since then when we teach, we tend to teach a paired antagonism model of the autonomic nervous system.

**DR. DAVE:** Right. That's what I learned.

**PORGES:** And it's still taught. I lecture in medical school and they're still teaching this. And it's in part correct, but very limited. Because it tells you really about the tonic tone to the targeted organ in the viscera. It tells you nothing about how that system will react to challenges.

And that's what the clinical world is really concerned about. So the issue is when we start to understand it from a concept of reactivity to challenge, we now move into an evolutionary theory of adaptiveness. So how does the system respond, and what are the adaptive features of that response? And what we start to see phylogenetically moving up from, functionally from, actually going back to the cartilaginous fish, which are the sharks and rays, they have an unmyelinated vagus, and what that does: when the resources are minimal, so the temperature, the food aren't there, they just slow up the metabolic activity. There's no problem for their survival with that because their nervous system is not oxygen hungry. Our nervous system is oxygen hungry. We can't slow up very well. When you look at the evolutionary or phylogenetic history of mammals, you start seeing that bony fish start having a spinal sympathetic nervous system very similar to what we have, so that they can recruit neural support for the motor activity that is now in balance with the parasympathetic. However, as we follow evolution, we have to think about the order in which these circuits come in because we retain these circuits, and that order provides us with a hierarchy of how the circuits work. Newer circuits work first and then older circuits are recruited. This was a concept that was introduced in about 1900 by an English neurologist called John Hughlings Jackson and he called it dissolution, or evolution in reverse. And many of your listeners have probably read Descartes' Error and there people talk about damage to frontal areas and cortical areas and disinhibiting older brain structures. And you have to think of the autonomic nervous system as following that same dissolution model. So what he is really trying to say is that not only is the brain going through disinhibiting older circuits, that's exactly how the autonomic system works. But there are 3 major phylogenetically ordered circuits. So there's this old unmyelinated circuit, which is a shut down one, and that one basically is related to reducing metabolic activity. You can see this used as a defensive system in mammals. When a cat picks up a mouse, and I'm going to rhetorically ask you, David, what happens when a cat picks up a mouse?

**DR. DAVE:** The mouse, I think, freezes.

**PORGES:** Can you describe the physical features of that freezing behavior?

**DR. DAVE:** I'm trying to remember if I've ever seen this, but I think the mouse maybe plays dead.

**PORGES:** Right. And the word as commonly used is death feigning. But playing dead is not a cognitive decision: it's a physiological reflex. What you have illustrated in your response was the ambiguity of the word "freezing". So we can freeze without motor tone like the mouse in the jaws of a cat or we can freeze like a deer in the headlights of a car which has lots of motor tone. One is a sympathetic response when we have increased motor tone, but the death feigning, the loose motor tone is the old vagal, the unmyelinated vagus. Both are defensive systems but they're mediated by different neural mechanisms. But the issue is when you go into this death feigning immobilization response it's potentially lethal for mammals. Many of those mice that go into that death feigning mode drop dead; 25% or more will drop dead.

**DR. DAVE:** They just die not because the cat clamped down its jaw. They just die?

**PORGES:** They die because they're not supporting the oxygen needs of their brain. And they basically have now a neural mechanism that resulted in a bradycardias, slowing of the heart rate and an apnea, a reduction of breathing and this resulted in them dying. Now there are two words, apneas and bradycardias. Have you heard those floating around with another clinical population?

**DR. DAVE:** There's sleep apnea. That's something I've certainly heard about.

**PORGES:** With pre-term babies, apneas and bradycardias are the major risk factors for death. With a pre-term baby, they come into the world without the mammalian vagal system. They come in with the reptilian one. So their defensive system is apneas and bradycardias. So phylogenetically in the sympathetic schema the sympathetics turn off that vagal circuit. We use that old unmyelinated vagal circuit primarily in our body for sub-diaphragmatic regulation, meaning our digestive track. Anything below our diaphragm is primarily unmyelinated vagal function. If you have to mobilize or run, what happens to your digestion? Does it get better? Or does it turn off?

**DR. DAVE:** It turns off.

**PORGES:** This is an example of that phylogenetic hierarchy. If you now need the sympathetics, you're going to inhibit that old parasympathetic. Now, if you are in a safe room with walls and friendly people and intonation of voice and people feel good, then that face-heart connection will dampen the sympathetic nervous system. So you now have this hierarchy. But when you dampen that sympathetic nervous system you pay a price. You pay a price because you give up many of the features that you and I learned in grad school that were associated with vigilance. We turn off our social engagement system when we're vigilant. Our social engagement system is the myelinated vagus and its linkage with the striated muscles of the face and head. What that does, some of the striated muscles go to an area, or are in an area called the middle ear. And what they do is they control the tension of the little bones, the ossicles, between the ear drum and the inner ear, and they change the tension on the ear drum. It's like tuning up a kettle drum. And when that tension is tight, you have difficulty hearing low frequency sounds. And when it's loose, you can hear low frequency sounds extraordinarily well. But the higher frequency sounds of the human voice disappear. So you can start gleaning the cost-benefit ratio curve.

**DR. DAVE:** I was really surprised to learn that we have the inner ear muscles.

**PORGES:** No, I've got to correct that: middle ear muscles. We have inner ear hair cells, but we have middle ear muscles. Don't get confused on that part.

**DR. DAVE:** That's even further back then, right?

**PORGES:** It's closer to the surface. When children have severe ear infections, otitis media, inflammation of the middle ear, what happens is when it's inflamed and when the infection goes away, often the neural regulation of the muscles is compromised. So many children who have otitis media often have difficulties in language skills. It's not causal, but it's related because



many individuals will rehabilitate spontaneously. The point I really want to make here is the middle ear muscles control what is called the transfer function of what sounds get into the inner ear and then into our brain. So if the middle ear muscles are not tense, pulling those bones tight together, it's like a rubber band. It's real loose, and only low frequencies can come through. If the muscles are tense, it tightens the ossicle chain, and now higher frequencies come in. But the higher frequencies come in at the expense of dampening low frequencies. Now I'll work with this and I'm going to ask you a rhetorical question. What frequencies are predators associated with?

**DR. DAVE:** You said it's rhetorical so that means I don't have to answer it! (Both laugh)

**PORGES:** It's a quiz, David! Really, low frequency sounds are phylogenetically wired into our nervous system to reflect predator.

**DR. DAVE:** Like the growling of a bear.

**PORGES:** Growling or earthquakes or thunder, or for autistic kids it will be vacuum cleaners, elevators, ventilation systems in buildings, low frequencies, airplanes. Those low frequency sounds trigger a defensive state. And when we are safe and if we can then trigger the middle ear muscles to work, we can filter out those low frequency sounds. Now I'm going to ask you another question.

**DR. DAVE:** Oh, no! I thought I had graduated!

**PORGES:** Oh, no, you didn't just graduate, you're on the couch! (Both laugh). Do you have children?

**DR. DAVE:** Yes, I do.

**PORGES:** Do you have any sons?

**DR. DAVE:** Three of them. I have four kids: three sons and a daughter.

**PORGES:** When they were teenagers, did your sons do anything to your car radio?

**DR. DAVE:** Well they would change the settings which would mess up my preprogrammed settings.

**PORGES:** No, I mean, what did they do with the bass and treble? Did they do anything with that?

**DR. DAVE:** Yeah, I think they probably liked to kick up the bass.

**PORGES:** Absolutely, because they were young guys with good middle ear muscles and they couldn't hear the bass if they were listening to human voices. So they were performing a

psychophysics experiment in front of your eyes. They were cranking up the bass so they could hear it. But to you when then they cranked up the bass, you couldn't hear the human voice.

**DR. DAVE:** You know, I suffer from tinnitus. Do you have anything to say about that in terms of what's going on?

**PORGES:** Well, not today. When we're finished with this and we're not being taped we'll talk about that! But the bottom line is that there is an adaptive function. If you're in safe environments and you're face looks animated, your middle ear muscles tend to be tense and you have difficulty hearing low frequency sounds because you're now able to pick up the higher frequencies of human voice. Those harmonics are conveying meaning. The problem with a lot of people is they can hear if people are speaking, but they can't hear the ends of the words. And it's those phonemes with the consonants that tell us what people are really saying. That is reserved for safe environments because when we're in dangerous environments we want to hear predators. So there's this dialectic occurring in terms of what is safe and what is not safe and how our nervous system responds to it. So the hierarchy in terms of polyvagal theory is phylogenetic, only with mammals that you have this face-heart connection that involves facial motor activity with the myelinated vagus. And that is inhibitory over the sympathetics because we really want to turn off defensive systems if we want to cuddle. Then the sympathetics will turn off digestive and subdiaphragmatic if we need to mobilize. So now you have the autonomic nervous system hierarchically organized for a very important adaptive functioning.

**DR. DAVE:** I want to take a step back to the mouse that freezes. I don't know if this has ever happened to you, but I've been in situations in the past where I was in relationship with a woman, and for some reason she was really tearing into me, just tearing me down. And I would freeze and just couldn't think of anything to defend myself and just really kind of go into that frozen mouse mode.

**PORGES:** That's really interesting, because now we start moving into trauma where the mammal, or let's say the human, retains some features of death feigning and some other features. So often we may dissociate, we may not be able to utilize in a sense certain parts of our nervous system if we're not safe. Actually, what you were describing, you didn't lose motor tone. You just probably tightened up physically and started to get defensive. You didn't start to pass out, right? If we take this whole concept of defensive systems, we have two basic categories of defense. And it's not fight or flight. It's fight-flight because they recruit the same autonomic components. We have a fight-flight system or mobilization system but we also have this immobilization system. You were in more of a fight-flight even though you weren't running. You might have felt you couldn't run. Your sympathetic nervous system probably got triggered. And when you got into that state, the feedback from your viscera to the brain stem basically didn't enable certain cortical areas to be recruited.

**DR. DAVE:** Yeah, I felt dumb!

**PORGES:** You got it! Yes, it's like what happens when we yell at kids and say, "Don't you know that!" And the child just can't recruit anything because what we've done is exactly what Hughlings Jackson talked about. We have a dissolution. But he was talking of brain damage



and brain disease. And what we're talking about is functional adaptive circuits that basically slide up and down that phylogenetic tree. So it's not good behavior or bad behavior. It's adaptive in certain settings. And you perceived that it was now a dangerous setting so your nervous system was basically reading the cues probably very accurately saying, "This is uncomfortable. I don't really want to be here." So you're not into a dialog of friendly chatter. You're into a defensive mode.

**DR. DAVE:** Yes. The beauty of your theory is that it provides insights into such seemingly disparate conditions as PTSD, Post Traumatic Stress Disorder, autism and borderline personality. And you've started talking a bit about trauma. And I want us to touch each of those three areas. In one place you refer to PTSD as "preparation to be killed".

**PORGES:** First of all, let's back off a little bit from clear cut diagnostic categories because diagnostic categories are totally resultant of a consensus of a series of behavioral and psychological features; so basically, a fever-type model. What I'm going to talk a lot about are common cores that seem to transcend diagnostic categories. And in the scientific world, let me give you a picture. There tends to be little resources available for people who want to talk about common cores across diagnostic categories. Where the money is is specificity and disease diagnosis. If there's a specific feature unique to autism or PTSD then that becomes of interest. But there are many core features that change or appear to be common cores/causes of many mental health disorders. One would be state regulation issues, behavioral and physiological state. The other one would be the facial affect that's going to be different. You're going to see features even of auditory hypersensitivities across many of them. You get the auditory hypersensitivities when those middle ear muscles aren't working right. You get the flat affect when the neural tone to the facial muscles isn't working right. You get state regulation issues when the vagal influence is not inhibiting the sympathetics. And I clustered all those systems together as a social engagement system. There's an organizing principle that if you understand the brain regulation of the face and the heart, it all comes together. And people learn some of this when they learn neuroanatomy and they learn about a set of pathways called special visceral afferents and those are the pathways that control the striated muscles of the face and the head. They're linked in the brain stem to the regulation of the myelinated vagus. Let's go back to really what you were asking. You want to talk about trauma.

**DR. DAVE:** Yes.

**PORGES:** I'll give you an example and then we're going to work off of that. I was at a conference and I was watching CNN before I had to go give a talk. And on the CNN show, it was the news part of their show. They were showing on the screen an airplane that was having great difficulty in landing. The wingtips were flipping side to side--it did not look pleasant. They then went down to interview the people on the airplane with the expectation that the people would be describing that they were really scared; they couldn't wait to get out of the plane, that they were jumping out of their skin. The reporter goes to a woman and says, "Will you please describe your experience on the plane?" And she says, "Experience? I passed out." So what is she saying?! She's like the mouse in the jaws of a cat. Under that setting the cues in the environment triggered this very old unmyelinated vagus immobilization defensive system. Did that happen to everyone in the plane with the same principle stimuli? No. Some people were of

course screaming, couldn't wait to get out, were highly mobilized. So what I've conceptualized, I actually coined the term "neuroception". And "neuroception" is really our nervous system's constant evaluation of risk in the environment. And it is through neuroceptive processes that we shift from these different circuits. The difficulty with neuroception in our cognitive centric world that we live in, is that this is not perception, it's not an aware response. Our nervous system is doing this for very profound adaptive reasons.

But we can't predict on a person to person basis, at least at this point, who's going to shut down or who's going to go into a panic state. And I will actually share my own personal experience with this. I went to get an MRI, and of course, in my profession a lot of people do research in MRI. So I was really kind of thrilled, "This is going to be a lot of fun!" "I got down there. I got on the table and they started to move me into the magnet, to hold my arms over, it's a small bore--not that much room! I got up to my forehead and I said, "Can we wait a moment? Can I get up and get a glass of water?" And I took the glass of water, got back on, and I got up to my nose and I said, "I'm out of here!" I had no idea that the features in the environment would trigger in my body a defense behavior of mobilization. I basically went into a state, as a clinician you'd call it panic, but it wasn't real panic. I knew I couldn't be there so I said, "Get me out of here." I took control. So if I have to take an MRI I get a little assist from certain types of medication. But the point I'm making is we don't even know--even those of us who study these things--we don't know what will trigger these responses. This becomes critical in the area of PTSD because people are functionally victimized when they go into states of shutdown. Because the clinical world had no construct of a defensive shutdown response they thought all defense was fight or flight. Even in naming PTSD as post-traumatic stress disorder people assumed they would have high cortisol; they would be high sympathetic tone, all these features of mobilization. What they found out is that many of them don't. They have low cortisol, low autonomic tone, so it was totally confusing to the clinical world because they didn't have the concept of a defensive system that was related to immobilization.

**DR. DAVE:** One of the things that I was interested to read is that you teach them to reframe their experience, to actually celebrate their bodies' successful survival strategy.

**PORGES:** So if we think about what much of clinical practice is about, and also what much of education is about, it's about evaluation. And whenever we tell someone that they are not doing something right, they should do something different, we're functionally evaluating them. And what are the neural-receptive responses to someone evaluating you? It's defensive. So how can we recruit this wonderful mammalian social engagement system if we're triggering defenses? Do you follow the paradox?

**DR. DAVE:** Yeah. And I think that you've said that some of the approaches to treating PTSD, are actually retraumatizing people.

**PORGES:** Well, they're basically keeping people in very defensive states and potentially retraumatizing. So if we say to a person, "Your body has done great things for you. It saved you, just like the mouse in the jaws of the cat by death feigning or by dissociating or by passing out. You were able to live through very very difficult unpleasant situations. The problem is that when you went into that state it was a little bit difficult to get out of. So it's as though people

can't move out of that very easily. But let's say, let's start off by saying, "Let's feel good about what your body did for you." I was giving a few workshops and I started to incorporate this type of dialog in the workshops. I started to get literally floods of emails back from clinicians who were working with trauma patients. And they were telling me the power of telling their patients this, their clients this. We're now in a sense seeing the heroic component of themselves as opposed to the victim component. So the issue is we're so locked into the cognition. We start understanding how we respond to certain subtle things like, "You did a great job. Your body did a wonderful thing for you," as opposed to, "I really feel for you because you can't engage with other people." There's a difference in the intonation and in the dialog about either evaluating the limitations of the behavior versus sharing the joy and pride of surviving as a starting point.

**DR. DAVE:** Yes, I can really relate to that. Do you have any thoughts about current approaches to treating PTSD which typically involves some sort of reexposure to the traumatic event?

**PORGES:** (Sighs) The issue...you can notice the gasp here!

**DR. DAVE:** I don't mean to cause you any pain here! (Both laugh.)

**PORGES:** I'm evaluating the costs that I will pay for making comments on your show! I would basically structure what I think would be the goal of a good therapy. A goal of a good therapy would be to be able to treat the traumatic event as if it were like an event and it had no visceral component to it. Probably with repeated exposure...and this goes back to our old training in classical conditioning where classical conditioning really was visceral conditioning. There were even concepts back in the days when we were in grad school. They were called one trial learning. And trauma has to be conceptualized functionally as a one trial learning because it's such a profound visceral shift with certain association. So what needs to be done is not to work on the stimulus, which is really what exposure therapy is all about, but to get people into different visceral states. If you can recruit the social engagement system and get people in a sense more regulated through this myelinated vagus with the striated muscles of the face and head, they're functioning using newer circuits and able now to recruit different levels of the brain. And those higher levels of the brain are inhibitory on those lower levels where some of this trauma resides. So the strategy is not exposure, but modulation of the visceral state, getting the person to be safe. Then when they are safe they have much better or much more plasticity or flexibility to deal with a variety of stimuli.

**DR. DAVE:** I would imagine that a behavior therapist would argue that in fact that's exactly what they're trying to do: get a person into a relaxed state and through successive approximations approach that scary experience, backing off as soon as they lose the relaxation.

**PORGES:** Perhaps. What I would actually say is that a lot of people think that therapy could be delivered through what they call manualization. "It's not the therapist that's critical; it's the delivery of the strategy." What I'm really saying is the social engagement system is the therapist. So in a sense the therapist's ability to modulate voice, to be appropriately attentive and then not to be too...to respect a person's space, whether that person is a behavioral therapist or any other form of therapy, the school of therapy is almost irrelevant. It's whether or not the

person has the natural skills and intuition to in a sense perform a neural, or be part of a neural, exercise with the client of this back and forth interaction of social engagement.

**DR. DAVE:** I totally agree with you. There is a lot of research to support that point of view, that it's the person of the therapist that is really important. Let's move on to talk a bit about autism because your theory has quite a bit to say about that as well.

**PORGES:** With autism, what I want to focus on is a lot of its symptoms. I don't want to focus on causes. Again, autism is a major problem not because there are so many individuals getting the diagnosis, but because treatments are so limited in improving the quality of life for the individual and their family. There are certain things that we've been working on. We've been working on the reduction of auditory hypersensitivities and then what happens when you reduce those. The idea in my model with dealing with autism is if you look at the features of autism you have a compromised social engagement system. You have lack of facial affect, you have poor eye gaze, you have auditory hypersensitivities, you have language delays often, you have lack of prosody in voice and you also have autonomic state regulation issues. You'll often see the individual sticking their fingers in their ears and it's not because they don't want to hear you. It's because the sounds they are hearing can be painful. And if you don't have middle ear muscles working you're being bombarded with a whole different acoustic array. What we've been working on is building a model of, "Can you engage the neural regulation of the middle ear muscles to dampen out or at least reduce or improve the severity of auditory hypersensitivities?" When you do that, what happens to the rest of the social engagement system? And this is a project I call the Listening Project and we've been doing this as a research project for about a decade with over 200 individuals. And what we're seeing is that if an individual has auditory hypersensitivity, about 60 percent will no longer have them after about five 45 minute sessions listening to acoustic stimulation that has been computer altered with the objective of trying to engage a neuroregulation of middle ear muscles. And what that really means is that the prosodic features of the vocal music that we're using has been computer altered to magnify the modulations. It's as if we have a hyperprosodic voice. And our nervous system, in a sense, goes towards prosody.

**DR. DAVE:** Can you imitate that to give us a sense of what it would sound like?

**PORGES:** My voice is very dry right now! (Both laugh!) I can't. What it would sound like, it would sound extraordinarily melodic, but then it would sound very thin and then it would start sounding fuller. The voice would appear to disappear and then come back. It becomes very engaging. When you're listening to music or someone singing, and it becomes very narrow, then becomes very full, we get exuberant when it gets fuller. We move towards it. Composers have always understood this in terms of how they pull in other instruments, make the frequency bands richer or narrower, softer and louder.

**DR. DAVE:** Dare I admit that I'm a big fan of American Idol! And when the contestants go for that sort of big emotional push it often brings tears to my eyes. It's very moving.

**PORGES:** Our nervous system evolved to listen for prosodic features. And so if you have a dog and you talk to a dog like that, the dog will literally go down to the ground, "Did I do

something wrong?” But if you say, “Come here, little girl, come here!” this melodic voice conveys to all mammals that they’re in friendly hands, friendly places. Our nervous system knows this. This is a given. And what I’m saying is if we can define the type of acoustic stimulation that we’re providing to people who have auditory hypersensitivities, flat faces, lack of prosody, you can start triggering these circuits to work. Of the individuals whose auditory hypersensitivities were improved with the intervention, 85% of them start to show more spontaneous social behavior. It doesn’t mean that they’re not autistic, but what we’re dealing with is we do think that [for some we can improve] some of the primary symptoms of autism to make the child’s life better and create portals of both social communication, language development, and education.

**DR. DAVE:** You talk about the importance of soothing as an intervention for some psychological disorders and that set me to wondering if you see meditation as fitting into your theory as an intervention.

**PORGES:** Actually, I gave a talk a few weeks ago and talk was called, “Safety as a Transformative State: Mindfulness from a Polyvagal Perspective.” What the talk was really trying to say was the features of meditation, or let’s use the term mindfulness meditation exercise, if we can move to that, deal with breath, deal with listening, and some may also deal with vocalizations. These are neural exercises of the myelinated vagal system, or the social engagement system. I’ll elaborate a little bit. If you exhale slowly, you are functionally enhancing the access of that myelinated vagus. So if we inhale rapidly and exhale slowly, the calmingness that we feel with that type of breathing pattern is very predictable because it’s increasing the amount of functional, or neural influence, of that myelinated vagus; it’s calming. If we reverse the inspiration expiration ratio we can make people anxious or what people used to know, put them into panic. So hyperventilation used to be viewed as a cue for panic. So the mindfulness breathing can be very important. The idea of listening, whether we listen to our own bodies or we listen to others is also this neural engagement of middle ear muscle structures. But we can’t listen and we can’t exhale slowly unless we are in safe places.

**DR. DAVE:** Well that certainly makes sense. Now before we run out of time here, I do want to touch on what the Polyvagal theory has to say about borderline personality and its treatment. I’m no huge fan of the DSM either, I might add. But still, it is something you’ve written about.

**PORGES:** Yes, what we’ve written about is the fact that we’ve actually studied them. We studied them and we actually found something that we weren’t looking for. We thought if we showed borderlines videos of different emotional scenarios they would respond differently than control subjects. That’s not what the study showed. The study showed that over time in an experimental setting, the borderline individuals start to lose their myelinated vagal control of the heart. And their heart rate started to go very fast: in a sense physiologically ready to jump out of their skin. While the controls, in exactly the same environment, over time became calmer and calmer and calmer. More and more vagal activity, slower heart rate. And when they walked into the study they were at the same place. What that started to tell me is that they are taking the same physical cues in the environment and, if we use the term “neuroception”, they are processing it differently. And what I realized was that we had the experimenter in the room with the client, the subject, and for the borderline, a strange person in the room was not calming. But



for a typical person, another person in the room sent a signal to the nervous system that this was safe. And so we see that two different people, one with a clinical diagnosis and the other group without any diagnosis, respond to the same physical features in two different ways. One is going into defensive strategy and one is going into a physiological state that supports social engagement. And I want to summarize this point and basically emphasize that those physiological states, whether we're talking about shutting down an unmyelinated vagal state or a sympathetic mobilization state or this myelinated vagal social engagement state, these are functionally neural platforms upon which a range of behaviors can occur or coexist.

So if we're in a state of fight flight mobilized, our ability to be friendly, to be social and even to be appropriately evaluative of others, is going to be compromised. We don't recognize this in both our education or either in our medical, clinical world. We don't realize that the person's physiological state is really the window, the portal, to both learning and treatment modalities. Whether we are talking about psychotherapy, body therapies, or basic practice of medicine, we want to create physiological states that recruit neural circuits that not only support social behavior, but that support health, growth and restoration. And the beauty of the model is that the same circuits involved in social behavior, social engagement system, are also the same circuits that are involved in health, growth, and restoration.

**DR. DAVE:** One of the things we haven't talked about explicitly, but that I think is implied in what you just said is, I seem to recall from my interview with Dacher Keltner is that the vagus system and oxytocin are implicated in affairs of the heart. So what about love?

**PORGES:** Let me give the appropriate credit before I start talking. My wife is Carol Sue Carter, or C. Sue Carter. Sue is the one who discovered the relationship between oxytocin and social behavior.

**DR. DAVE:** I recall seeing her name now.

**PORGES:** Yes, she's the prairie vole person.

**DR. DAVE:** Oh, yes!

**PORGES:** She developed that model. The prairie vole has something unique, of course, high oxytocin levels, but it also has high myelinated vagal tone, unlike most small rodents. It has tremendous vagal activity. And when they're with their litter mates, they have high vagal activity. And if you put them into isolation, they lose the vagal activity in a hurry: now they are tachycardic very fast and like mice. And when they're with their partners it's very slow and very rhythmic. But the interesting part--and this is the part we are working on together--because the area of the unmyelinated vagus, the dorsal motor nucleus, the vagus, is a hot spot for oxytocin. And this is critical to understand. So the area of the immobilization circuit, which I talked about only as defense, is really oxytocin sensitive. And what that means is oxytocin enables the recruitment of this old defensive immobilization circuit for reproductive and other behaviors. So that we can be next to another person and immobilized as opposed to being defensive, required phylogenetically or through evolutionary selection, oxytocin or some peptide to start re-upregulating the mobilization circuit. Let me make it clearer. For mammals, immobilization is



dangerous. How do we get a mammal to immobilize? This is where oxytocin is involved. It enables the system to immobilize. This is critical for nursing, for parturition, and for reproductive behaviors.

**Dr. Dave:** OK, shifting ground just a bit here: you're a pioneer in the emerging field of interpersonal neurobiology. Perhaps you can give us a sense of what's implied by that term "interpersonal neurobiology". I had the privilege of interviewing Dr. Dan Siegel not long ago and do I recall correctly that he's also part of this interpersonal neurobiology movement?

**PORGES:** I would say that Dan is really the person who spearheaded this whole idea of trying to bring into psychotherapy and clinical, both psychiatry and clinical psychology, an understanding of the nervous system to help clinicians deal with their clients. What I bring into the story is the notion of the viscera, the actual underlying part of our body and how that influences higher brain structures. And then how higher brain structures influence our body. So what I'm really working on is expanding what I call an "interpersonal neurobiology narrative" that would then be helpful in both demystifying mental health disorders and then also developing strategies that would reduce symptoms and help improve outcomes.

**DR. DAVE:** OK, well, we should probably wrap it up here. I wonder if there is any final thought that you'd like to leave our listeners with?

**PORGES:** Well, first of all, thank you very much for inviting me. The final thought would be that the hierarchy that's described by the Polyvagal Theory is extraordinarily intuitive and provides a very useful framework to understand many aspects of human behavior. We have to also realize that those circuits that were used for defense, the one that I was talking about, sympathetic mobilization and the immobilization unmyelinated vagus, are actually coopted for very prosocial events or processes in mammals. So I just talked about how the immobilization circuit is coopted and is now used for reproduction, nursing and parturition, but the mobilization circuit is coopted away from defense and we call it "play". How do we do that? If we watch people playing, it's always mobilization and face to face. So using the social engagement system to basically make sure that the mobilization circuit is not interpreted as fighting or danger. So when people play and they accidentally hit each other, if they don't rapidly make face-to-face and diffuse it, repair the violation, you have fights.

**DR. DAVE:** Dr. Stephen Porges, it's been my privilege to be socially engaged with you today! And I want to thank you for being my guest on Shrink Rap Radio.

**PORGES:** Thank you very much, Dave.