Hello, friends and colleagues, and welcome to episode 169 of Dermasphere, the podcast by dermatologists, for dermatologists, and for the dermatologically curious. I am one of your hosts. My name is Luke Johnson. I am a pediatric dermatologist and general dermatologist with the University of Utah. And joining me, of course, is-- This is Michelle Tarvox. I'm an associate professor of dermatology and dermatopathology at Texas Tech University Health Sciences Center in beautiful sunny Livoc, Texas. But right now, I am recording in beautiful sunny Las Vegas because I'm speaking at the fall clinical meeting. We look forward to hearing all about it after you get back. And of course, the third member of the cast is the pimping bell, which exists to highlight especially question-worthy content. So if you are in academics, for example, pay attention, because those might be questions worth asking or answering. Also, friends, speaking of being in academics and learning stuff, I wanted to let you know that I am participating, running a CME experience through a platform called Gathered, or GatherEd, G-A-T-H-E-R hyphen E-D. I've done this a few times through the platform and find that it's quite valuable. They do a good job. You do some self-study modules that you go at your own pace a lot. And then there's also a few live group discussions where we can all learn from our colleagues. This particular experience is on chronic spontaneous urticaria. And the experience itself is a bit more chronic than normal because we do what has become normal for these gathered platforms. You do some modules. We meet as a group once or twice. But then we also stay together as a group to meet two or three additional times throughout the year to discuss updates in the world of chronic spontaneous urticaria from various dermatology or allergy meetings. So you'll certainly feel real up to date on the latest CSU information if you stick with it for the full six to nine months. We'll put a link in the show notes. And you can find it on our social media, et cetera, as well. Love to see you all there. And speaking of learning stuff, today we have a guest to help us learn even more stuff than usual. This is Dr. Joseph Lam. Joseph, thanks so much for joining us. Thanks. I'm super, super excited to be here. We're super excited to have you. And unfortunately today, we are not a video cast. Or the guests can all see Joseph wearing a bow tie and suspenders and me wearing my pajamas in my closet. Michelle's sort of in the middle. Look like you're ready to go talk at the Las Vegas conference. Joseph, do you want to start just by introducing yourself? Is there something about you? Sure. Yeah. I'm Joseph Lam. I'm a clinical professor of pediatrics and dermatology and skin sciences at UBC here in Vancouver, BC, Canada. We're America's hat. And while Michelle is in sunny Las Vegas, we have liquid sunshine here in Vancouver. That's what makes us green. And it makes the trees grow, which is absolutely lovely. Exactly. Well, really happy to have you here, Joseph. You have published on a number of issues. And in particular, stuff that's kind of like benign normal baby stuff, but it's not well reported in the medical literature. But the lay public have words for this stuff. Exactly. Like a sugar bug. Should I be worried that I'm going to be bitten by sugar bugs? No, you don't have to worry about being bitten by sugar bugs. But it's something if you Google your baby having this blue line across the bridge of the nose, you're going to find this thing that's been described for quite a while. And they call it the sugar bug vein. I have no idea why it's called sugar bug vein. It has nothing to do with sugar. But people know that it's there and know that it's benign. But like a lot of things in medicine, if you don't have a name for it, oftentimes if you see that, you look at that medical literature, you say, I don't know what this is. And people have referred to this blue line in the base of the nose for venous malformations or something else that they're worried about or thinning of the skin. And the funny thing is that in medicine, we recognize patterns. And sometimes it's not just medical people that recognize patterns, but people who see a lot of babies. So in baby blogs, they've had this name called the sugar bug vein. And you know it's nothing bad. We hope it's nothing bad. Or they think it's nothing bad. Someone has put this association with the MTE, the folate-- Oh, MTHFR? Yeah, which has nothing to do with it really. So here, it's kind of

funny. I have a good colleague in Spain, Antonio Torello. And I often ask him what's going on if I don't know what's going on with my patients. And he knows just about everything. And he sent me this picture of a patient with this blue line across the nose. And he said, what is this? And I said, oh, yeah, we see a lot of this. We just tell the families it's nothing. But he said, what's it called? We're like, I have no name for it. But if you look in blogs, they have a name for it. And he said, well, we should really give it a name. So I think he talked to a colleague in Mexico who looked at, I think, 700 kids across different ages and found that up to half of them had this. And it's much more noticeable in patients who have lighter skin. And I think as kids get older, the skin gets thicker, or they get more color in the skin, you don't notice it. It's one of those funny things where I think if people have been in practice for a long time, they recognize it. And they say, oh, yeah, this is nothing. I've seen this a lot of times. I don't know what the name for this is, but it's fine. But then you have someone early in practice, they take a look at it. And I think in medicine, if you see something you don't know, you should try to figure out what it is, because it could be something benign, or it could be something serious. So we thought, oh, we should really give it a name. So this colleague in Mexico looked at all these kids, and this is published as a transverse nasal root vein. Just so now we have a name. That's a great name. It clarifies that it has nothing to do with sugar or bugs, which is very important, because babies are sweet. But when you say sugar problem, to most people, they're going to worry about diabetes or something like this. And I don't know about you, but bugs on a baby, that creeps me out. We don't want bugs in your baby. You don't think calling your baby your little sugar bug isn't cute? I mean, that is cute. That is cute. So you guys published this in Pediatric Dermatology in 2024. The title of the article is "Frequency of the Prominent Transverse Nasal Root Vain in Children." And the colleague in Mexico is Igor Lopez Carrera. And like you say, 701 patients. About half of them had this finding, though according to the text of the article, a lot of the parents weren't even aware until the clinicians were like, hey, baby's got this thing. And then they were like, oh, yeah, I guess you're right. You also shared with us one of these baby blog articles. I was actually shocked at how quality it was. It was from the What to Expect website, I guess. And it looks like they do a good job sourcing their material. And there's actually an MD who reviews the article for medical accuracy. So I presume that this represents a rather normal but high quality post online about the sugar bug vein for the lay public. And that post suggested that they would kind of go away by age one year. But in the study you guys published, still about 50% of them were there around age six. Most adults don't seem to have them, so they must go away sometime. But perhaps they persist longer than the lay public generally thinks. Yeah, I think what happens is that if you have something that's benign, you see it every day, then you just don't notice it anymore. So I think patients often present early, especially if it's the first baby saying, what's this? And after a while, if it doesn't do anything, I think they probably don't present anymore, which is why physicians probably don't see it. But I think it's good to recognize, because especially for a first time parent-- I tell first time parents, it's normal to worry about everything because you don't know anything about your kid. It's kind of funny where parenting is one of the jobs, one of the most important jobs out there, but one of the few jobs where you don't get screening and training for, you kind of dive right in. But the flip side is you become a good parent by just observing. And I think observing, oh, this is kind of funny. Is it good or bad? You go to your doctor. But after a while, you stop observing things that are benign. I think that's what happens for this. And as adults, I think what happens is the skin gets thicker, so you don't even see it, even though it's still there. And those of us who have kids also know that as the months and years go by, we just get beaten down. And so we just don't see as much stuff that's going on in our kids, especially if you have more than one. It was more common in patients with lighter phototypes. So phototype 2, which is the lightest phototype down there in Mexico that they had enough data to

report on, was 71% of the patients. 71%. And phototype 4 was 40%. So there might be something to this lighter skin issue. You also mentioned a few of the differential diagnoses. So I often tell my rotating residents, part of our job, especially as subspecialists, is to make sure we're thinking about the scary things and making sure it's not those things. So you mentioned various vascular malformations or anomalies, like a venous malformation or an early infantile hemangioma could potentially look like this, potentially something right in the midline there. You would want to think about dermoid cysts or other midline abnormalities. So those are the things that should at least be crossing through your mind as a dermatology clinician. And if you're like, no, no way it's any of those things, then reassurance seems like a great plan. Exactly. In the same vein is another-- Oh, Luke, that was-- Another condition known in the lay press as the milky tongue. You want to tell us about that one, Joseph? That was kind of neat, because I think each of you have probably seen this. You've seen a lot of babies where they have the white on their tongue. And the things we usually think about is retained milk or thrush. And we've seen a lot of these kids with this buildup, this residue that's there. But it doesn't fit with milk. It's not easy to take off. And it doesn't look like thrush. Oftentimes, with thrush, you can get it on the buccal mucosa and other areas. And if you swab it, you often don't see any candida that's there. So again, if you look at the article, it's Antonio Torello, who's the senior author. Because he said, hey, what's going on with these kids' tongues? And I was like, yeah, I don't think it's candida. I don't think it's milk. It's the stuff that's there. And it's almost like a similar vein to the first case, where you see these blogs talking about the stuff called milky tongue-- not milk tongue, but milky tongue. And there's even a YouTube video of this guy who's cutting the straw so you have a sharp edge, scraping off his baby's tongue. I do not recommend that. But it looks like this debris that's there. So for a bunch of our kids, we took-- not all of them, but some of them we took scrapings from the skin-- from the tongue. And we found that it's just epithelial cells that are there. So I think it just retained skin cells on the tongue. And this tends to disappear as the kids get older. But it doesn't fit with milk. It doesn't fit with candida. And we looked in the literature. There was a series of kids who they thought were candida, but in a subset of them, they couldn't find any candida at all. We think, oh, this has probably been there for a while. However, like a lot of things, it can be noticed by the parents first. And you have a lot of blogs talking about this milky tongue phenomenon. So we collected a bunch of cases. It's kind of funny. Me and Antonio were talking. And a friend of ours, Agnes in Switzerland, I think she's at, she put together a case series as well. We're like, hey, you know what? Let's pull this all together. And so we did. And we tried to get a fancy name for this. So we call it transient infantile lingual leukoplakia. So it actually forms a name, too. Oh, that's cute. I like this. We couldn't find one that fit with the mouth or the tongue that well. So another publication from 2024 in pediatric dermatology by you and Antonio Tirelli and Agnes Schwieger-Brielle and Tram Nguyen. It's called Transient Infantile Lingual Leukoplakia, an Under-recognized Cause of White Tongues in Infancy. Congrats on the international collaboration. This was Canada, Germany, Switzerland, and Spain. That's pretty cool. And it sounds like you found 22 of these kiddos. And one of the distinctive features is that it spares the tip of the tongue. And it's just on the tongue. And it doesn't scrape off easily like retained milkwood. It doesn't spread across other mucosal surfaces like thrush wood. And then it's just gone usually by a year of age. So you have a couple hypotheses that you propose about why this might be a thing. You want to run those by us? Yeah. At the end of the day, we don't know. But when you see something, you try to explain what happens with the clues that we have. And here we think that it's probably friction that rubs off all the retained skin cells that are there. So probably the tip of the tongue, it can rub against the alveolar ridge of the mouth. And later on, we probably don't see it because kids start eating solids. And it rubs this off the surface of the tongue, we think. We don't know for sure. But it's probably the

case. And another good table with differential diagnoses. So we mentioned oral candidiasis and retained milk. A couple others that are worth remembering, especially for those of us deep in the weeds of pediatric dermatology, white sponge nevus. Remember that one, listeners? So that one can also involve the buccal surfaces. And that one's inherited in an autosomal dominant fashion. A couple of our genodermatoses can also present with white stuff in the mouth, like pachyonicia congenita, which can be painful leukoceratosis, again, on the tongue and buccal surfaces. And then they also have some other features of the disorder, like painful PPK, hypertrophic nail dystrophy. And then another genodermatosis, dyskeratosis congenita. Their oral findings usually present a little bit later in life, so you wouldn't necessarily expect it in an infant. But again, they have it on the tongue and other surfaces in the mouth. And then they also have nail dystrophy, bone marrow failure, and lacy reticulate pigmentation of the upper chest and neck. So I appreciated the nice table in there about the differential stuff that, again, we should make sure that it is not. Before we tell the parents, everything is fine. I love that. Joseph, really appreciate you having you on today. Anything else you'd like to tell our listeners or more places where they can find you or your work? No, just except that this is a great podcast. You should definitely hit the Subscribe button. There's great stuff here. Click on Subscribe button. Do we even have those in podcasts? I'm not sure. I think you do an iPod in the podcast player on Apple. I'm going to check. I think you do. Yes, mash it. Yes, exactly. Exactly. Just boop. All right, Joseph, thanks so much for joining us. We'll have to have you back. I like your insights and the curiosity. I love that. Sometimes I feel like we've lost that as adults and as doctors who've been through this for a while. But I love that you guys are still looking at stuff and saying, what is this? We should name it. And why does it happen? That's cool. Fabulous. Thanks so much for having me. Thank you for coming on the show. So in addition to learning about normal stuff about babies, another way we learn stuff is through CertLink. At least, many of us who are board-certified dermatologists in the United States use this CertLink program for the American Board of Dermatology to maintain our certification. And so every-- I don't even know-- six to 12 months or something, you look through and you pick some articles that you think look good. So I was looking through them. And I saw this article on social determinants of health. And I started to glance right past it, as I do for most of the articles. No offense, but CertLink does find some good stuff. They've got a lot of-- you know, DermPath or cosmetic Derm or whatever. And then I paused for a moment. And I thought to myself, I said, Luke, I am not sure you are very good at social determinants of health. What are they? Are you bad at recognizing them? And Luke, are you cynical about your ability to do anything to impact them? And then I thought, Luke, you're right, you handsome devil. Maybe I could read this article after all. And so I did. And then I thought I would share it with you guys. So the title is "The Social Determinants of Health and Their Impact on Dermatologic Health, Part 2-- Taking Action to Address the Social Determinants of Health." Your answers include Eileen Chang and Sacha Ritha Bowers from UCSF and Southern Illinois University. And this was published in an article called "Dermatology Clinics-- Social Determinants of Health." So these are non-medical factors that influence a person's health and well-being and include primarily the conditions in which people are born, grow, live, learn, work, play, and age. They contribute to health outcomes and health disparities much more than access to health care does. So I think most of us can appreciate that these are kind of a big deal. But perhaps, listeners, you are also kind of like me and that you're cynical in our ability to impact any of them in the dermatology clinic. And you might think to yourself, it's a good thing we have social workers so they can work on this stuff and I can work on putting try-em-sin-alone on their skin. But that might be an overly cynical way to look at this. A few other important background bits. So apparently, the most common social needs that patients report in clinical settings are related to the following issues-- housing-- so they are unhoused or their housing

situation is unstable or low quality-- food, which I think is especially embarrassing in a wealthy country. Come on, people. We've got enough resources to feed our populace. And we should be feeding them healthy food-- hot take. They are concerned about child care. Certainly, part of me can relate to this one, despite all of the advantages that I have enjoyed. And then financial strain, including the cost of health care as well as more general financial concerns. Some of these concerns are referred to as health-related social needs. So that dovetails with the social determinants of health. You can have health-related social needs that are kind of in the same line. And apparently, patients who are high utilizers of health care often have one or more of these health-related social needs versus having zero. And having multiple such needs is related to having additional emergency room visits. It's negatively associated with the number of wellness visits you attend. And the more social needs you have, the more you utilize health care, despite the fact that you are younger and have fewer medical comorbidities. And the authors also quote, "There is growing data to support social needs interventions as a means to reduce health care utilization." So if our goal is to reduce the amount of money we spend on health care, one potential way to do it is to increase the interventions to address some of these social needs. OK, I can appreciate all of that, but I'm still not sure how much I can do to influence it. The authors also say that because, quote, "social determinants of health are heavily influenced by society's social and economic policies, individuals from the same neighborhood or community often have similar health-related social needs." We can probably understand that. And if you are an optimist, like I am, maybe this is a feature rather than a bug. So if there's this neighborhood that's got some problem, like doesn't have access to good food or all the housing and it sucks, if you make some impact on the neighborhood, you're impacting a bunch of people with perhaps a single intervention. So maybe that's better than having our social needs distributed randomly throughout society. So what can we do about this? Well, it's tough to say, but they do say that there are things that we can do. So there are five activities that health care providers can engage in, they say, which are the five A's, roughly in order of which one should come first and how localized it is to you and your clinic versus out there in society. So the first A is awareness. Makes some sense. You've got to be aware of these social needs before you can maybe do anything about them. Next is adjustment. So they say, "alter clinical care to accommodate social barriers." So I actually kind of think I do this and probably most of our listeners do as well. If you recognize that your patient might have some financial hardship, you can use GoodRx cards or the $4 generic lists. These authors also cite Costco and Amazon pharmacies as places where you can actually look up the price of medicines online before going there. I didn't actually know you could do that. Oftentimes, I'll ask patients how easy it is for you to come back here. We have a huge catchment area as you do too, Michelle. People are driving for hours to get to you sometimes. And I try not to say anymore, where do you live? Because if they have to take three buses, then that still doesn't really matter that they might live in the same city. It's still a huge pain to get there. Or if they come in with a bunch of little kids, these are all reasons to let telehealth shine. So it's pros and cons, of course. We all are aware of them. But I think they can do a lot of good. It can save people hours of driving. It can save people having to look for child care, et cetera. And there have been a strange set of new constrictions placed on telehealth, which is puzzling to me because they've basically restricted it to only rural areas, regardless of how far a patient has to travel for care and regardless also of how challenging it is for a patient to get to the care provider, like you just said. So I'm hopeful that that is a change that will be reversed with some thoughtful policy. Yeah, honestly, I guess I don't know what you're talking about. It hasn't seemed to impact my practice. I don't know if it's a Texas thing or it has to do with their insurance or what. It's a Texas thing. The University of Utah just says, whatever, do what you need. We'll figure it out on the back end because we can basically see

everybody via telehealth. Though I will say I suspect that the reimbursement is less for those visits these days. OK, so we had awareness. We have adjustment. Next is assistance, which is connecting patients with resources. So this is probably about where my ability to feel like I can do stuff kind of falls off. There probably are some social workers and who work with the Children's Hospital. Every so often, I've connected patients with those. I have uninsured patients and we tell them about the free clinics. You think you have a skin cancer on your face, but rather than us taking care of it here at the university, here's some information about the 4th Street Clinic, et cetera. So awareness, adjustment, assistance then is alignment. They say, understand existing social care assets in the community, organize them to facilitate synergies, and invest in and deploy them to positively affect health outcomes. The jargon sounds a little businessy, facilitate synergies. But if you understand what assets are out there, then perhaps we can work to utilize them best in our practices/societies. And then finally, advocacy-- sorry, go ahead. I have a good example of alignment. So as many people who know me know, I direct dermatology services at our free clinic in Lubbock, Texas that's a student-led free clinic for our working poor and homeless in our community, unhoused, working poor and unhoused people in our community. And the synergy that's provided is that the center has social work is there. There's food assistance. There's a clothes closet. There's career training. And all of it's provided in the same location. And there's also support for travel. And the free clinic has a pharmacy that helps us to get medication to patients. And we also have arrangements with local pharmacies to provide low-cost medications to patients from the free clinic. So putting all of those resources in one place so the patient only has to get to one location helps to get them access to services that they might otherwise have to travel five or six different places to achieve. I love it. And the final A is advocacy. I think a lot of us would understand what that is. So awareness, adjustment, assistance, alignment, and advocacy. That awareness piece is so important. When we teach the dermatology, medical literacy, and advocacy undergraduate elective at my institution, we started that about two years ago. We're going to our third year in the springtime. We actually teach about the social determinants of health. We talk about the impact it has on people's ability to access the health care system and adequately treat their conditions. And I think that teaching this early on in medical training is very important to ensure that people are literate in these things that impact our patient's health that are sometimes outside of both their and our control. But I think that the points that you brought forward are good ways to think about it systemically and try to address it in that way. A couple other actionable items are to make the patient's next clinic visit before they leave the office, especially if they're using an interpreter, for example. And if you are wondering about their health literacy or their ability to understand based on the language barrier, you can have them use the teachback method. I will admit that I kind of find it difficult. It always seems patronizing to be like, OK, now can you tell me what I told you so I can make sure you understand it. But there's probably a better way to put it. The authors close by saying, "advancing equity is possible, but only with awareness followed by intentional action." And this review aims to be a step in that direction. I love it, Luke. Social determinants of health and getting patients the assistance they need to live their healthiest life is very important. And you use a lot of language to help us understand those important points. And I want to make the point that language is important and classification is important for many different things. So I'm going to review now an article from the journal Itch, which is the international forum for the study of itch. And this is a United States expert panel consensus on uniform nomenclature and diagnosis for neuropathic parietus by author Sean Quatra, who's a friend of the podcast. We stan Sean Quatra. And also, Julia-- What's up, Sean? What up, Sean? And he's actually going to be at the fall clinical meeting, so I shall have to say hello and get a photograph. And then also, the corresponding author, Julia Supovich. And this study was done through many different

prestigious institutions-- Johns Hopkins, Mass General, Emory Penn, UCSF, Duke, and University of Miami. And the challenge that was posed was that there is no distinct ICD-10 code for neuropathic parietus. We actually have lumped that in with other parietus as L29.8. So that's not a very specific diagnosis. There's scarce data on incidence and prevalence, because we haven't named or classified it, so we haven't collected that data. It's often mistaken for other conditions, and there's a lack of consensus in the nomenclature. There are FDA-approved therapies for many conditions that affect patients' quality of life and the way that chronic itching can, such as atopic dermatitis. But therapeutic options for neuropathic parietus are somewhat unclear and usually reliant on older repurposed therapeutics. And it can be a debilitating condition. And because it lacks standardized diagnostic methods, it also is not a target for drug development or treatment pathways. So that causes an issue for taking care of this risingly more common issue, especially as we have more of an aging population. And we also have more of a modified environment for our ancient physiology to respond to. So their expert panel was 10 expert participants that they were leading dermatologists specializing in parietus and inflammatory skin disease. It was a 50-50 gender balance. So there was five men and five women on the panel out of seven academic centers. And they achieved 100% consensus with full agreement on all definitions and recommendations. And this was done in November of 2021 via the Zoom platform. So one of the good things that came out of probably the modifications to society from the COVID pandemic. So first, we need to describe something if we're going to study it. There is a philosopher, Ludwig Wittenstein, that would say, "The limits of my language means the limits of my world." Meaning we can only study, conceptualize, or perceive what we have language for. We actually see this over the course of history with colors. So all ancient societies evolved words, language to describe, colors kind of in a similar order. Black and white would be first, so light and dark. Then red would come in. Blue comes in much, much later. So there's a lot of old writings that talk about the wine dark sea and calling the sea wine colored. Which I don't think usually the sea is that color. But when the color blue started to be described and used, then you saw that show up more in people's utilization. So how we describe a condition can also influence how we study it. So there was a condition called hysteria. I don't know if you've ever watched any historical films about this. There's actually a movie called Hysteria that's about this. And it was a catch-all diagnosis for a range of psychological and sometimes somatic symptoms that we would now call anxiety disorders, depression, conversion disorder, things like that. But they were blamed on the wandering womb. So if you were trying to study hysteria, you would probably not hit the target of the patient has an anxiety disorder. Instead, you would blame the wandering womb for the woes that this person was experiencing. We've seen this also with HIV. When it first started to be recognized as a problem, it wasn't called HIV. It was actually called GRID, which was Gay-related Immunodeficiency. And it was blamed actually on something called-- they were inhaled amyl nitrates that are sometimes called poppers. That's one of the explanations that came out early for the developing problem that was HIV. And so the people who were trying to study it were looking in the wrong direction. They weren't looking at it as an infectious disease. So how we describe things can influence how we study it. So until recently, patients who have things like neuroinflammation or pruragonogularis were recognized as having something called neurotic excoriations, which is actually a moralizing and stigmatizing term. And as we've come to understand the disease state better, we understand that there's neuroinflammation in cytokine-driven processes. So we used to sort of blame patients. Oh, if you just would stop itching, or if you would just stop scratching, you would stop itching. But we do need to have accurate language to describe things. So medicine can advance through discovery, but also through definition. And having ICD-10 codes for diagnoses helps to capture data, allow for data collection, and then eventually allow, hopefully, for adequate

and appropriate therapies. So the core clinical features of neuropathic parietis are that the patient has normal skin or only secondary changes from scratching. So they don't have any primary skin lesions. The distribution can be localized, can be dermatomal, or may be generalized. And there's several subtypes we'll talk about. Associated symptoms can include dysesthesias, pain, stinging, burning, or tingling sensations. And treatment response is usually suboptimal if you're aiming at TH2 polarization markers. And standard therapies are often not going to be adequate for neuropathic parietis. So the authors kind of break down the great age categories of neuropathic itch as brachioradial parietis, nontalgeparasthetica, multilevel symmetric parietis, which I'm going to define in a minute, scalp parietis, post-herpetic neuralgia, which I think is pretty well understood by dermatologists, small fiber neuropathy, and then parietis secondary to syndromes affecting central nerves, like post-stroke parietis, brain tumors, Crutchfield-Yakov disease, trigeminal trophic syndrome, multiple sclerosis, things like this. And then finally, peripheral nerve damage with mixed etiology, like post-surgical parietis or scar-related parietis. So brachioradial parietis, we all learn about in our early careers as dermatologists. It's proximal lateral forearm itching, usually in middle-aged women. And it's connected to UV radiation and cervical spinal issues. There's a good test for brachioradial parietis, where you actually can place a little ice pack onto the patient's skin where they're experiencing that discomfort. And if they achieve relief from that, it can be a positive indicator that that's indeed what you are dealing with. Nontalgeparasthetica, also many of us are aware of this condition. Usually unilateral upper back, sometimes both sides, often associated with some cutaneous nerve impingement from upper spinal, either postural or kyphotic changes. Scalp parietis can also be a very troublesome condition, localized parietis on the scalp, often due to cervicalgia or impingement of cervical nerves in the posterior cervical plexus. And you can also have burning scalp syndrome as a subtype of this. Multi-level symmetric parietis is, I think, the one that people are going to be the least familiar with. But as I describe it, you're going to be like, I have seen this patient. So multi-level symmetric parietis occurs with symmetric, well-delineated dermatitis of the extremities, trunk, or back, often associated with moderate to severe degenerative disc disease. And again, this is probably nerve compression. So when I'm explaining this to patients, I tell them, when our nerves are in trouble, they don't have a whole lot of ways to tell us that they're in trouble. They can either hurt, or they can itch, or they can have a shooting electrical sensation. And all of these are very noxious stimuli. So the symmetric presentation of the multi-level symmetric parietis is usually indicative, potentially, of that severe degenerative disc disease, moderate to severe. Post-herpetic neuralgia is areas where you've previously had Zoster. I think we all understand that well. Small fiber neuropathy is a little bit more of a niche diagnosis. Can be associated with chronic itch or chronic pain. It is challenging to diagnose. There are specialized skin biopsies and special labs that can process the tissue in a way that allows them to assess for small fiber neuropathy. And then central nerve damage, like post-stroke parietis, brain tumors, Krushfeld-Jakob, trigeminal trophic, multiple sclerosis, and then finally peripheral nerve damage, usually post-surgical or related to trauma. So to diagnose localized parietis, you might consider targeted imaging, like an MRI or CT for brachioradial parietis, nortalgia peristhetica, or that multi-level symmetric parietis to identify areas of spinal degeneration, nerve impingement or herniation. The ice pack test works for brachioradial parietis, causes improvement after the ice pack to help confirm the diagnosis. Sometimes skin biopsy may play a role if you need to rule out alternative diagnoses, or if you're looking for small fiber neuropathy to quantify intraepidermal nerve fiber density. Other patients that might benefit from skin biopsy might be those with non-bullus pemphigoid or prodromal pemphigoid before they really develop a bull-A. And some patients with bullus pemphigoid don't have a whole lot of what appears to be primary skin lesions. So this can be important. For diffuse parietis, you can do systemic screening,

kidney and liver tests, thyroid tests, iron panel, fasting glucose, and hemoglobin A1C. You may also do additional testing if indicated by history, such as B12, HIV, or hepatitis B and C. And of course, age-appropriate cancer screening, because sometimes diffuse parietis can be the presenting sign of a malignancy, especially a hematologic one. A CBC with eosinophils and a normal serum IgE is typical of neuropathic parietis. So it's a non-immunological origin of the itch. And a neurologic evaluation can help rule out central causes, like multiple sclerosis, neurodegenerative disorders. And you may do quantitative sensory testing in select patients. So if you were going to do a skin biopsy, you would, of course, biopsy where the patient is itchy. If you're looking for length-dependent neuropathies, you would go to the distal extremities. When you are looking for small fiber neuropathy, you have to also obtain control biopsies from unaffected regions. And then the lab actually has to do 50-micrometer sections with PGP 9.5 stains, which can stain those nerve fibers. And they do quantification of the intraepidermal nerve fiber density compared to an unaffected site. So that's very specialized testing that can be done at reference labs. Wait a sec. What's this length-dependent thing? So length-dependent neuropathy. This can happen in conjunction with spinal disease. But it's one of the causes of neuropathy of the lower extremities that isn't related to diabetes. So it's not something that, as I think dermatologists, we deal with as much. But there's an interesting thing that happens when people get too tall, also, I feel like. So Mr. Dr. Tarbox is very tall compared to me. And there's actually some studies that show that very tall people actually get more skin cancer than shorter people. And is it because they're closer to the sun? I don't know. It's very interesting. But length-dependent neuropathy, it's like peripheral nerve damage in which the longest nerves are affected the most severely. So it usually starts in the feet and then progresses upward like a stocking distribution, and then eventually gets to the upper body. And the longer the electrical cable is, the more vulnerable it is to injury from metabolic toxic or ischemic insults. So that kind of long fiber neuropathy is something that can disrupt the axonal mechanism. And you end up with the farthest ends of the longest axons. So that's what we call the axon-like degenerating firsts. So they kind of die back, if that makes sense. And that can be caused by many different things. Toxic neuropathy happens from chemotherapies with axonal degeneration and typically affects the feet first. Metabolic, of course, which is often related to diabetes mellitus, can affect the feet as well. Charcot-Marie-Tooth disease is one to think about. There's also idiopathic long fiber neuropathy, which is up to 30% of cases. If that makes sense. Gotcha. So for treatments, the first thing you want to do is try to treat the underlying etiology. So that's why you might do some of those studies to figure out, is a vitamin deficiency, is it a physical issue where you have a herniated disc compressing a nerve? Is it something that can be addressed in a way that targets the primary underlying etiology? Because that is going to be the most likely to have success and have lower side effects. Topical therapies can be used for localized conditions, systemic ones for more refractory cases, and the procedural options can be reserved for more severe conditions. So patients will sometimes benefit from topical anesthetics like lidocaine or promoxine. You do have to be thoughtful about the potential for contact dermatitis, especially if legs are involved, especially if there's stasis dermatitis. Capsaicin, that's sort of like lighting all of the fireworks in the fireworks booth at the same time, because then they can't put on any show for the rest of the day because you've exhausted their supplies. So when you put on capsaicin, you tend to elaborate quite a lot of substance B all at once, and it's sort of like having a little like firework explosion and then the rest of the day less sensitive to pain or itch. Coolants can be helpful. Menthol and camphor can provide a cooling sensation or ice packs for brachioradial parietus because our small, unmyelated C fibers that carry pain and itch can really kind of only do one thing at a time, and they also feel heat and cold. So if you make a nerve that is feeling itchy feel cold, it can't also feel itchy at the same time. And the temperature takes predominance.

Compounded medications sometimes help. I have treated patients with compounded topical amitriptyline, even ketamine, lidocaine, or doxepin. And the topical treatments are going to be most helpful for things that are localized, so brachioradial parietus, scalp dysesthesia, notalgia parasitica. When it gets to systemic therapies, you can look into things like gabapentinoids. I think many of us use those off label to manage pruritic neuropathies. They are usually going to start low dose and then titrate up. Usually, again, we use this for notalgia parasitica. Now, if you are going to start patients on gabapentinoids, you do need to understand how to help patients get off of those medicines if they are not giving them the therapeutic efficacy they need or if they can't tolerate the side effects. Because there's actually been case reports of documented withdrawal symptoms in patients taking as little as 400 milligrams for at least three weeks. So if the patient is on a moderate dose, like 900 to 1,200 milligrams per day, for more than three to four weeks, you need to taper. Anything high dose, like over 1,800 milligrams a day, regardless of indication for greater than two weeks, has to be tapered. Because you can actually potentially trigger seizures if you withdraw patients too quickly from a gabapentinoid. And if the patient is like a seizure disorder, a history of alcohol use, CNS depressive use, or benzodiazepine, concomitantly with gabapentinoids, a longer taper may be needed. If you abruptly discontinue gabapentin, patients can have anxiety, insomnia, nausea, sweatness, and pain rebound or itch rebound, and rarely seizures, which is why you don't want to do that. So if you have a patient that has been on a higher dose for a long period of time, you go down by about 300 milligrams every five to seven days. Slower is safer when you're tapering patients off of gabapentinoids. And if patients have renal impairment, they may need proportionately smaller steps, because they may have been accumulating drug. So you taper-- - Hey Michelle, what's a dose that you often start patients with for gabapentin? - So most of my patients that have neuropathic paritis are older patients that have other medical comorbidities. So I do tend to start very low. I start with a nighttime dose, and then we see the patient back, and then we add some progressively daytime doses and midday doses if they need those. Some patients are very profoundly affected by the sedating effects of gabapentinoid. Some patients don't have as much of an impact, but I don't want somebody to have an increased risk for falling or have difficulty conducting their activities of daily living, because I go too hard in the paint with the gabapentinoids. - So what dose? - So I usually start in a patient that's very medically fragile. I'll even start as a test the water with 100 milligrams at bedtime. I know that's not gonna do very much, but I need to show them that they can tolerate it. And then I'll increase to 300 at bedtime. And if they're tolerating that well, we might add a 100 in the morning. We might then add, go up to a 300 in the morning, and then a midday dose if they still need further control and they're not too sedated. You do have to always balance the risk of sedating medication, especially in a medically frail or older patient. And our patients, as they get older and become more medically frail, are more likely to have neuropathic paritis. So just to kind of finish up, 'cause I know I'm getting a little long on this article. Systemic therapies like antidepressants can be helpful. So SSRIs, SNRIs, tricyclic antidepressants, and doxepin are sometimes used. Opioid modulators like naltrexone. Remember that mu is boo. So mu is the opioid receptor that is connected with itch. Kappa opioids, when you agonize those, or when you stimulate those receptors for Kappa opioid receptors, patients have better control of paritis. There's actually difilikephalin, which is a medication we've spoken about on the podcast before, that's approved for renal paritis for that indication for stimulating those Kappa receptors. Kinabinoids can be helpful in some patients. Drenabinol is available for some patients in some states. And you can also kind of modify the protocols based off of what the patient needs. You can also do procedural interventions. I've written a manuscript about physical therapy and its benefit in treating N. peristetica. And this can be very helpful for musculoskeletal contributors, like brachioradal parietis

and N. peristetica, and sometimes the cervical spinal disease that can cause scalp parietis. TENS units, so transcutaneous epidermal nerve stimulation or acupuncture may be helpful. Botulinum toxin can be used sometimes in localized refractory cases and sometimes CT guided nerve root injections, which obviously we wouldn't do, can be helpful for specific nerve involvement as can dorsal root ganglion stimulation, which is going through exploratory trials for severe neuropathic paritis. Now, any of us that treat patients that suffer from this condition know there is a significant quality of life burden for this condition. It can impact sleep, it can distract patients' attention, and it can be debilitating and often misunderstood. And again, we add on top of that, blaming the patient for the condition with things like neurotic excoriations as a diagnosis. And then there can be central sensitization where patients have heightened neurotransmitter release and a hyper excitable set of spinal neurons with generalized parietis coming from originally localized damage. Patients can get itchier and itchier if they go untreated. So again, our key ways to differentiate this from inflammatory dermatosis, normal skin with only secondary lesions, so no primary inflammatory lesions, normal eosinophils, IgE levels, it's not a type two inflammation signature, it doesn't respond well to standard anti-inflammatory therapies, and the origin tends to be neurologic rather than immunologic. Now, how many FDA approved therapies do you think there are for neuropathic parietis, Luke? - None. - There are zero, that is correct. And there is only a single ICD-10 code, which is not specific, which is other parietis, L29.8. So there are no FDA approved therapies, there is not a distinct diagnostic code. So we do need to standardize approaches and help dedicate research funding for this. So we have now a uniform diagnostic name and diagnostic criteria, workup guidelines and subtype classification with the eight great kinds of neuropathic parietis. We talked through the treatment framework for topical, systemic, and procedural options, and highlighted the importance for research moving forward. So that shared nomenclature and hopefully eventual ICD-10 unique code for this condition will allow for study to happen on these conditions, will allow for gathering of data, allow for potentially evidence-based outcomes research, hopefully randomized controlled trials, and eventually novel therapeutic development to help improve patients' quality of life. So this shared nomenclature, I think is a very important first step. - Sean Quatra himself has been on the podcast a few times. So listeners, if you'd like to go back and hear him talk about itch and his research, you can find him on episodes 73, 88, and 140. And if you would like to hear us talk about congenital syphilis, stick around, 'cause that's what we're gonna do next. So I guess syphilis has been increasing in the United States over the past 10 years. And so we in the world of dermatology and pediatric dermatology might see more congenital syphilis. There were about 2000 cases of it reported in the US in 2020. I can't really remember what congenital syphilis looks like though. So we have this article, again, a Cirqlink article. This one was published in pediatric dermatology. It's called "Mucocutaneous Manifestations of Congenital Syphilis in the Neonate, a Review of a Surging Disease." Authors include Jasmine Newton and Bernard Cohen from the University of South Dakota, from Tufts, from Massachusetts General Hospital, and from Johns Hopkins. So the rates of syphilis have been increasing, including in the prenatal world, due to multiple factors. Some of these might sound similar to the structural societal determinants of health. Geez, I can't even remember what the S stands for now. That we talked about a few minutes ago. So these authors cite structural barriers to prenatal care, such as poverty, substance use, citizenship status, healthcare coverage, low sexual health literacy, and gender inequality. And perhaps for similar reasons, there are certain minority groups that have higher rates of sexually transmitted infections with causative factors, including reduced access to care, distrust of the healthcare system, lower education levels, lower health literacy, and quote, "Other social determinants of health." Hey, they're social determinants of health. "Commonly present in underserved communities." Because of these conditions, presumably, the

highest rates of syphilis are found in a couple specific minority groups, American Indians and Alaskan Natives. You probably remember that syphilis is caused by a spirochete, which is a type of bacteria, called treponema pallidum. Pregnant women are generally screened for syphilis, assuming they do get prenatal care. "Untreated maternal syphilis increases the risk of a bunch of stuff, such as premature delivery, low birth weight, intrauterine growth restriction, spontaneous abortion, stillbirth, non-immune hydrox." Michelle, you remember what hydrox is? - Yeah, hydrox fatalis. Like, that's kind of like a swollen baby that's not making red blood cells terribly well. And that's one that can be-- - Swollen baby. - Yeah, can't that also be associated with infection with parvovirus? - Makes sense to me, it sounds right. So yes, hydrox is like accumulation of fluid in numerous tissues in the fetus. So non-immune hydrox is hydrox that's not caused by immune activity, I guess. - Yeah, immune hydrox is 'cause of like the RH incompatibility situation. - Gotcha, look at you, you know so much. And perinatal death. And of course, untreated maternal syphilis increases the risk of congenital syphilis. Sorry, Michelle, I cut you off, what'd you say? - Oh, it's okay, it can also be associated with other infections. So like parvovirus is one of them, syphilis, definitely one of them, toxoplasmosis, and cytomegalovirus. And in those settings, it tends to be involved also with anemia and hepatic dysfunction. - If you get an ultrasound during your pregnancy, it could detect some signs of congenital syphilis, including syphilis of the placenta, placental syphilis. So this can cause anemia in the fetus, which they could manifest as hepatomegaly, splenomegaly, and/or hydrox, so that's something the ultrasound could find, as well as ascites, polyhydramnios, and placentomegaly. Two thirds of infants with congenital syphilis are asymptomatic at birth. But the third who are symptomatic might have the following symptoms. Rash, nasal discharge. There's all this syphilis stuff that has fancy names. - That's the one called snuffles. (laughing) - It's kind of a cute name for what you're probably bringing up. - I know, it's a cute name for not a great condition, yeah. - Jaundice, hepatomegaly, splenomegaly, anemia, thrombocytopenia, bone lesions, and CNS syphilis. And congenital syphilis can be divided into early and late versions. So the early manifestations occur before age two, and the late manifestations are after age two. Two years, not two months. So the ones we just mentioned with snuffles, et cetera, are of course early manifestations that could be present at birth. And mucocutaneous findings, though, usually appear one to six weeks after birth. And these findings that dermatologists might be interested in appear in about 70% of neonates with congenital syphilis. The most common is a symmetric copper red morbilliform rash. (bell dings) You can get this somewhat pathognomonic finding by getting these little scaly macules and plaques with little colorettes on the plantar surfaces and the palmar surfaces. - Yes, the colorette is a very good sign. Really, like, you'll catch syphilis, like, you won't catch syphilis. You'll properly identify, you do not want to catch syphilis, you want to properly identify syphilis. But if you look up for the colorette, you will not miss it. - Colorette has a fancy name. - Biets colorette, yes it does. - Yes, indeed. Less common findings include desquamation, so perhaps like super biets 'cause it's usually on the acral surfaces. - I like that. - You can get various vesiculobolis lesions. Again, this has a fancy name. - Say again. - I'm looking at you, Michelle, 'cause you know all this stuff. - Is it the coronavineris? Is that the one you're talking about? - This one is Pemphigus syphiliticus. - Ah, cute. I think the coronavineris is the one that's around the hairline usually. And it's like the crown of Venus. - Yeah, that's not the crown you want from Venus. - Not that one, no. - You can get mucus patches on the palate or perineum. - I had that as an unknown from my residents this week. - Oh, you had it. - No, I presented it. - Gotta watch our words, we can talk about that all day. - I presented it as an unknown from a properly identified source in the dermatologic literature, yes. - Excellent. The TTI, you can get stuff that looks like erythema multiforme. You can get lichenoid lesions. So remember, syphilis is one of our great mimickers. Perhaps this is one reason why, as you like to remind our listeners, we all

started as syphilologists. - Yes, we were syphilologists first. - And then there's another interesting appearance. A satellite papules surrounding a central hyperpigmented lesion. This is called the carimbos. - Ah ha ha. - Yeah, look at you. - Thank you. - Carimbos pattern. What does carimbos mean? - I am a syphilologist. So it has to do with the way like a cornflower is shaped, I think, is like what carimbos means. Yeah, yeah. - You can get moth eaten alopecia. - Of course. And scaly, it's scaly alopecia too. So it has all of the clinical, like a dermoscopic and histologic features of alopecia areata. But it's also scaly, which can help you distinguish the two. - Thank you. You can get fissures, perioral, perinasal, or perianal fissures. - Regadies. - Fissures are our first orifices. That's later. - Okay. - According to these authors. But we'll get there in just a sec. And condylomalata, remember condylomalcuminata is warts, whereas condylomalata is syphilis. And so late manifestations include, pronounce it for us. - Ragadies. - Ragadies, cracks and fissures at the corners of the mouth. I guess those are different than the perioral fissures you get early, at least according to these authors. Saddle-nose and then Hutchinson's triad. Anyone remember Hutchinson's triad? - Is it the sabershins and the mulberry molars? And then like, hold on, that's the other thing. - Is that what it is? - So the blunted upper incisors, which are probably the mulberry teeth you mentioned, and then interstitial keratitis. - The keratitis, I always forget the keratitis. And then it's the hearing loss. - That's right, eighth cranial nerve deafness. So the sabershins are not part of this perioral triad. - They are not, but they are fun to talk about. - If you do think one of your patients has congenital syphilis, do some x-rays, 'cause they get these bony changes as well. Testing for syphilis, ugh, remember this from the textbooks? There's like, paragraphs on it for some reason. - And so the short story is you need a screening test and then a confirmatory test. So the screening tests are sensitive but not specific. So they're good at finding something that might be syphilis, but not so good at saying for sure it is syphilis. These are called non-treponemal antibody tests, and they are either the VDRL or the RPR. Because they're sensitive but not specific, we need a confirmatory test. There's a bunch of confirmatory tests. They include PCR, it's probably the most common these days, I would guess, as well as fluorescent antibody testing or enzyme immunoassays. And do recall that patients with SLE, especially if they have antiphospholipid antibodies, can have false positive syphilis testing results. - That is correct. - For both types of tests, but mostly the initial screening tests. You treat syphilis with penicillin. If you have a high suspicion, even if they don't have testing or supportive clinical features, just give them penicillin. The authors say with adequate treatment, quote, "Clinical features should resolve within three months "and serological markers should resolve within six months." - And you do wanna use the same lab because if you're gonna be monitoring for treatment response, every lab will have different results for the same patient. So if you can use the same lab, that is preferred. - So there's congenital syphilis, and there's our show for today. So thanks for joining us and learning with us today, friends. And thanks again to Joseph Lam for joining us. So from Joseph, we learned about the sugar bug vein, also called the prominent nasal root vein. And we learned about milky tongue, also called transient infantile lingual leukoplakia. And then we learned about social determinants of health. We learned about neuropathic paritis, and we learned about congenital syphilis. Thanks to our institutions, of course. Thanks to the University of Utah for supporting the podcast. Thanks to Texas Tech for lending us Michelle. And thanks to all of the members of Team Dermasphere, a dedicated group of medical and pre-medical students who keep things moving along behind the scenes. Because of them, you can find us on social media. We are present on X, Facebook, and Instagram. You can also find us on our website, https://linkprotect.cudasvc.com/url?a=https%3a%2f%2fdermaspherepodcast.com&c=E,1,yxzKcyh_6FrBmY9tvoZLsmn4yI_gIZ5M3GUv_ddIVMvqMjyfaK2g1VPrcMGxO953G0E9E2eby_Zkk4CqXnfKtqLK8U2t77hwiVL3etH-rTmAVNNJyI8,&typo=1, which is also thanks to Team Dermasphere. Team

Dermasphere does include Jordan Easterling, Austin Callister, Justin Lyon, Hiral Patel, Karen Makul, Mary Tidwell, Alithan, (mumbles) It's been a long, long show, I guess. Alison Roker and Sahi Talisila. Thanks for everything that you guys do, including a lot of other stuff behind the scenes like sound editing and distributing the podcast and so on. Friends, if you would like to hang out with us some more, hopefully you're at Fall Clinical right now and can go say hi to Michelle in the next 20 minutes. Otherwise, I'd love to see you at the CME Chronic Spontaneous Erticaria Experience. You don't really have to do anything if you don't feel like it. If you sign up and realize you don't have time, no harm, no foul, but it does have a lot of good content and you get to hang out with me via Zoom several times. Again, we'll put a link in the show notes and it's also gonna be on our social media. And that is all for today, friends. We'll see you in two weeks.