**Episode 14: Puerperal Fever – Part 3**

By Dr. Joe Chappelle

Hello everyone, and welcome back. I’m Joe Chappelle and you’re listening to Episode 14 of the OB/GYN Podcast. Today, we’ll be finishing our discussion on puerperal fever by admittedly going a little bit outside the margins. Group B Strep and its association with neonatal sepsis and the ideology behind out attempts to prevent it will be the topics of discussion for today. And I know this is not technically a puerperal infection, but it is caused by passing bacteria from mother to baby during the birth process, and so I think it counts. But, honestly, I just really wanted an excuse to cover it because I think it’s interesting.

But before we dive into today’s topic, I want to thank everyone again who is on the Slack. I think it is going to be a really great resource for all of us. And remember that if you want to get it on it, just send me an email at feedback@obgyn.fm and I’ll shoot you an invite. You can also use the Slack for topic suggestions, general feedback or just connecting with other Ob/Gyns around the world. I love hearing from all of you, so please keep it coming. Anyway, let’s get started with Episode 14: Puerperal Fever – Part 3.

Going back through history, it’s difficult to tease out how much an effect neonatal sepsis had on the overall mortality rate, because infection wasn’t something that we even recognized as its own entity until the 1800s. But judging by its prevalence in modern times, it was probably a decent number. But today, we aren’t really going to talk about neonatal sepsis as a whole, but rather we are going to focus on the neonatal sepsis caused by group B Strep.

Group B Strep is a commensal G.I. and G.U. bacteria that is generally considered harmless. The primary member of this group is Streptococcus agalactiae, which is a Gram-positive facultative anaerobe that is present in about 30% of adults. Group B Strep, or GBS, gets its name from the Lancefield classification schema, which is based on the carbohydrate composition of the cell walls. Over time, this classification has become effectively useless, and it’s almost never used anymore, but the name has stuck with us, and so today we still talk about group A Strep, group D Strep and for today, group B Strep. The bacteria itself was first discovered by Edmond Nocard in the late 1800s. He worked with Louis Pasteur at the Louis Pasteur Institute before setting up his own productive laboratory in rural France. While investigating the cause of bovine mastitis, he found a new bacteria, which he cultured and called Streptococcus agalactiae. He named it because it caused dairy cows to stop producing milk, hence a-galactiae. Outside the world of dairy cows, it was not thought much about until 1938, when R.M. Fry, working in London, published a case series of several women who died from what appeared to be GBS sepsis. This was a shocking finding. Apparently, this bacteria, which everyone thought was harmless in humans, was causing death in women and babies. His results were verified by further case reports, and along with the rising field of pediatrics and neonatology, led to the understanding that GBS caused neonatal sepsis. And by the late 1940s, it was well established as the leading cause.

When we talk about neonatal sepsis, I want to make sure that we’re talking about the same thing. So here, definitions matter. There is early onset sepsis, which occurs less than 7 days after birth and which has a very high mortality rate. And then there is late onset, which occurs 7 to 90 days after birth and is generally not as severe. In early onset sepsis, GBS is the most common bacteria responsible, and makes up over 50% of the cases. E. coli, Staph, H flu and other Gram-negative anaerobes each account for about 10 to 15%. When we look at late onset, the causes are a little more diverse, with about 30% coming from E. coli, 20% from Staph and 13% from Enterococci with almost no GBS cases. Therefore, today we’re going to be focusing almost exclusively on early onset sepsis, as it is the one most commonly associated with GBS.

Early onset GBS sepsis is almost universally associated with maternal colonization with GBS. And at any given time, roughly 20% of pregnant women are colonized with group B Strep. This number varies in the literature between 3 and 35% and has to do with the type of culture performed and how the sample is collected. But about 20% does seem to be the consensus, and so we’re going to work with that today. Group B Strep colonization can come and go throughout pregnancies, and in studies where they tested women and 28 weeks and again at 36 weeks, there was a different cohort of positive women at each time point. This becomes very important when we talk about the timing of screening. Additionally, there are no great ways of predicting women who will have GBS, whether we use race, age, marital status, education or other socioeconomic factors. To highlight this, interestingly in one study, they found that Hispanic women had the highest rate of GBS colonization if they were living in the Northeast, but the lowest if they were living in the Pacific Northwest, even when controlling for all other factors. This demonstrates that a lot of the useful risk factors that we talk about don’t work when we’re trying to predict who will have group B Strep.

So, about 20% of women are colonized. Our next question is, how often is that bacteria passed on to the neonate during a vaginal delivery? That number seems to be about 50%. And so, given that we started at 20%, that means that about 10% of babies born will be colonized with group B Strep. How many of those babies will get sick? To answer that question, I’m going to talk about a great longitudinal dataset from the Yale New Haven Hospital, where they’ve been collecting information about every baby with sepsis since 1928. Over that time period, the rate of sepsis has ranged from about 1% to 3%, and includes neonates of all gestational ages, which is important, because we’ll see in a minute that prematurity is a huge risk factor for sepsis. The most recent data before the beginning of antibiotic prophylaxis was about 2%, of which about half or 1% would be from GBS, so we’re going to use that 1% for our purposes. So, if 10% of babies are colonized with GBS and about 1% of total deliveries become septic from GBS, that means that about 10% of babies that are colonized with group B Strep will become septic from it. These numbers are going to become important when we discuss the preventative strategies, so bear with me as we walk through all these numbers.

Going back to risk factors, let’s talk about which ones are most associated with group B Strep sepsis. First off, and I guess this is pretty obvious, but maternal colonization is the highest risk factor, with an odds ratio of over 200. But we can break this down a little bit more by talking about the density of colonization. In a couple of studies where they looked at how much GBS women had as opposed to there just being a positive or a negative result, they found that the denser the colonization, the higher the risk. The light colonization gave an odds ratio of 97, while the heavy colonization had an odds ratio of 247. One note here is that about 1% of all cases of group B Strep sepsis are in babies who tested negative for group B Strep, which is probably a false negative swab, but it’s still worth keeping in mind.

As we talked about prematurity, and therefore the corollary low birth weight, both are associated with increased odds. Being born at less than 28 weeks carries an odd of 22, while weight under 2,500 g. has an odds ratio of 7.4. Keep in mind this is in all cases, as most women who would deliver this early don’t know whether they’re colonized with group B Strep.

The last few are important because they’re going to make up the criteria for the alternative approach to GBS sepsis prevention. They are any fever in labor, which gives an odds ratio of 4, chorioamnionitis, with an odds ratio of 6.4 and rupture of membranes greater than 18 hours with an odds ratio of 7.3. Additionally, GBS bacteriuria and having a previous neonate affected by group B Strep, have also been listed as high-risk factors. There is not much data on either one of these points, but in small studies, GBS bacteriuria seems to be associated with about a 10 times elevation in risk of GBS sepsis when compared against no GBS in the urine. For the previous affected neonate, there’s even less data, but most schema still retain it. But there is one study that I found that quotes that women who have a GBS affected neonate in the previous delivery, will have GBS at the delivery of their next child about 40% of the time, which is about double the risk.

All this information comes from a meta-analysis published in the Journal of Pediatrics in 1999. Two points here. One, is that most of the studies included were done before the onset of antibiotic prophylaxis, which means that we can use it to compare with data after we started using penicillin and ampicillin in labor. And secondly, I just want to note this is one of the most well-written, easy to read, clear and concise meta-analysis that I’ve ever read, and I recommend all of you click on that link in the show notes and look it over. If you’re at all interested in the art of scientific literature, it is well worth your time.

Anyway, knowing that about 1 to 3% of neonates born will have GBS sepsis, with a mortality rate of about 15 to 30%, people began to look at ways to prevent these infections. This started in the late ‘80s and continued into the early ‘90s. They took advantage of the fact that streptococcus is almost universally sensitive to penicillin or its derivatives and they began to test giving penicillin or ampicillin during labor to see if they could prevent that sepsis. One of the seminal papers was published in the New England Journal of Medicine in 1986 by Gotoff and Boyer. In their study, they randomized women deemed high risk due to either premature labor, prolonged rupture or intrapartum fever, to either get ampicillin in labor or no treatment at all. 40 of the 79 neonates in the control group were found to be colonized as compared to 8 of the 85 in the ampicillin group. And therefore, unsurprisingly, the rate of GBS related neonatal sepsis was much lower in the treatment group, with 0 of 85 being affected in the treatment group versus 5 of 79 in the no treatment group.

Which, interestingly, fits our previous information that about 10% of babies with GBS colonization will get sick. In this study, there were 8 in the treatment group colonized and no cases of sepsis, and in the control group there were 40 cases of colonization and 5 sepsis. Both fall roughly in that 10% range. I like it when things make sense and so this one made me happy. Importantly, it also tells me that maybe it’s not the antibiotics per se that is preventing sepsis but rather the ability of the antibiotics to decrease colonization after birth. Looking at the two groups here, we’ve seen that ampicillin decreased the colonization rate by about 75%, and so we should expect about the same reduction in GBS sepsis cases. Larger studies came along and confirmed these findings, with a consensus of about 65% reduction rate. And in 1996, the Centers for Disease Control, the American College of Obstetricians and Gynecologists and the American Academy of Pediatrics put out guidelines that recommended universal screening for group B Strep in the last trimester of pregnancy. Then, intrapartum penicillin if tested positive.

Let’s take a minute now and let’s talk about that screening. First off, where do we obtain a specimen from? Group B Strep tends to colonize the lower third of the vagina and also the perirectal area, and so current recommendations are for a rectovaginal swab as culture of the vagina alone will miss about 50% of women who are colonized. With that settled, we next need to know when to culture. Let me take a step back here and lay out what our goal is. We want to know who is going to have group B Strep colonization at birth. I know that sounds simplistic but bear with me a minute because I want to walk us through this.

Studies have found that about 2.5% of women who were positive a week ago will be negative if tested again, and that about 0.7% of women will acquire GBS colonization in any given week. Taking this in mind, again Gotoff did a study looking at the predictive value of perineal cultures at 28 weeks and 36 weeks. They found that positive predictive value of a culture performed at 28 weeks for GBS colonization at delivery was about 48.7% versus 58.6% if that culture was performed at 36 weeks. The negative predictive value also differed. At 28 weeks it was 95.3% and at 36 weeks it was 98.2%. You might be asking, it seems like later is better, so why not do it as late as possible? Well, simply enough, because we don’t know when women are going to deliver, and we don’t want to miss anyone. So, we choose a date, like 36 weeks, because we know that most women will not deliver before then. This also means, however, that if we did do a swab before 36 weeks for something like preterm labor, then we need to repeat that if she’s still pregnant at 36 weeks to ensure that we have the correct result for delivery.

So, that was a lot of numbers, and I know they’re a little abstract, so I want to bring them into a clinical sense. If the culture is negative at 36 weeks, that means that less than 2% of women will test positive at delivery, and for practical reasons we can assume that for all women who test negative at 36 weeks, that they’re negative at birth and don’t need treatment. We can assume this because, remember, only 50% will pass on GBS and only 10% of those babies will get sick, which gives us a rate of about 0.1% of negative women who will have a baby with GBS sepsis, which is quite low, and the number needed to treat in the swab-negative women would be about 1,000 to prevent 1 case of sepsis.

Moving on to the positive results. With a positive predictive value of only roughly 60%, it means that 40% of the women that we treat for GBS at delivery will not be colonized. Doing the math out in this one tells us that about 30% of the babies born to mothers who test positive for GBS will be colonized, leading to about 3% of babies with sepsis if left untreated. Treatment lowers this to almost 0, and so we can say that we need to treat 33 women to prevent 1 case of GBS related neonatal sepsis, and 100 women to prevent 1 death. Looking at these numbers, we can see that although the positive predictive value of only 60% doesn’t look too good, when we instead look at the number needed to treat, that value proposition behind screening and treatment starts to look a lot better. Treating 33 women to prevent 1 case of GBS related neonatal sepsis seems like a win to me and a no-brainer.

Now, let’s take a step further back and I want to look at cost, because what we’re talking about here really is public health, so we really want to think about the cost of treatment as well. The cost of a GBS swab is about $25, and the cost of penicillin G is about $8 a dose. Let’s a assume an average of 3 doses, and of course the first dose is a double dose, and so the cost of the total treatment is about $32 for the antibiotics. Now, we do need to add some costs for medical supplies and nursing time, so let’s be generous and call it about $100 per patient total, including the group B Strep test. We need to test 166 women to find the 33 that will be GBS positive and then treat those 33 to prevent 1 case of neonatal sepsis. In dollars, that amounts to about $16,600 per prevented case of group B Strep. The average cost of NICU care is about $3,500 to $4,000 a day, and the length of stay for a baby with GBS sepsis can often be measured in weeks. So, even assuming that we don’t prevent every case even if we screen and test, we will still spend much less on prevention than we will on treatment.

There is another method of screening that can be used and is used in some places, and it forgoes swabbing women for GBS and instead relies on risk factors to determine treatment. The high-risk factors often used are GBS bacteriuria at any point in the pregnancy, a history of a previously affected pregnancy by GBS sepsis, rupture of membranes greater than 18 hours or a fever during labor. Going back to our discussion of risk factors for GBS sepsis, we can see that although all of these do infer increased risk, they are really small potatoes when compared against GBS colonization. To remind you, all of those risk factors had an odds ratio of 5 to 10, while being colonized alone gives an odds of over 200. Therefore, to me it is not surprising that this method does not work as well at decreasing the risk of GBS sepsis as compared to screening and treating.

Surprisingly to me, there are still several developed nations, including the United Kingdom, the Netherlands and New Zealand that use this method rather than universal screening. In the United Kingdom, this policy has been examined thoroughly, and multiple studies have shown that the cost of treating neonatal sepsis far outweighs the cost of screening and prophylaxis. One study from 2007 determined that England alone could save $75 million a year just by ditching the high-risk method and going to screening and treating like we do here in the States. And they also found that the risk-based strategy doesn’t work as well. This is based on research by Schrag et al. from 2002, where they looked at 5,000 women who delivered in the United States who used one of the two strategies. In hospitals that used universal screening, about 90% of the women received antibiotic prophylaxis if it was indicated, while in the risk-based group only 79% received the appropriate antibiotics. Now, 11% difference may not seem too high, but push that out over the entire U.S. population and that 11% starts to mean a lot of sick babies. This difference makes sense to me because there is much less room for error in the screening group. If you have a positive result in front of you, it is far easier to remember to give the antibiotics. However, if you are using a risk-based model, then you need to get an accurate history from the mother, put it through your mental algorithm to determine who is high risk and who is not, and then remember to start the antibiotics. So, I am not surprised here that universal screening works better.

So far, I’ve been making a pretty solid case for universal screening and intrapartum prophylaxis. But let me turn the page and give you the other side of the argument. It is important to know why the other side does what they do. In many cases, it is just that they’re valuing different things or making different trade-offs. To do that, I’m going to quote from the Royal College of Obstetricians and Gynecologist Green-top Guideline on group B Strep. In regards to universal screening, they state the following: “Until it is clear that antenatal screening for GBS carriage does more good than harm, and that the benefits are cost effective, the National Screening Committee does not recommend routine screening in the United Kingdom. Initiating national swab-based screening for antenatal GBS carriage would have a substantial impact on the provision of antenatal care within the United Kingdom. Major organizational changes and new funding would be required to ensure an equitable and quality-assured service.”

Okay, so let’s break this statement down. First, I would say that we can pretty safely state that there is a cost savings with the universal screening and treatment based on the studies, some of which were even done in England. Second, let’s focus on the harm of the treatment, because this is really the crux of their argument. The argument for harm from treatment is based on a trial called ORACLE that was performed in the late ‘90s in London and looked at antibiotics for PPROM in preterm labor. One study of women who received amoxicillin and erythromycin for preterm labor showed that their children were at higher risk of cerebral palsy than women who did not receive the antibiotics. However, a different paper from the same group looked at women with PPROM and did not show this effect at all. This seems to be the harm that they’re talking about when it comes to prophylaxing women in preterm labor, which by the way the American College of Ob/Gyns does recommend.

The Royal College states the following: “Women presenting in uncomplicated spontaneous preterm labor with intact membranes are the same group of women as those recruited to the ORACLE trial, where there was evidence of harm in terms of adverse neurodevelopmental outcome, including cerebral palsy in their infants at 7 years of age in the absence of any demonstratable benefit in the short term. Although the antibiotics used in ORACLE are different from those used for intrapartum prophylaxis for GBS, there is no evidence from long-term follow up studies that other antibiotics, including penicillin, are safe. As the risk for early onset GBS infection in this group of infants is still low, prompt management of early onset sepsis, if it occurs, is preferable to intrapartum prophylaxis for large numbers of women.”

Wow. I’m not going to argue with the ORACLE trial, although you can find many people who have. But I do want to compare the risk of cerebral palsy with the risk of neonatal sepsis and its subsequent risk of morbidity and mortality. In the ORACLE trial, the risk of getting cerebral palsy was extremely low, and as I told you before, one of the highest risk factors for neonatal sepsis is prematurity. And so, this seems to me that they’re throwing the baby out with the bath water. Literally. The last argument against screening and treating has to do with E. coli sepsis. As mentioned, behind GBS, E. coli is the next most common cause of early onset neonatal sepsis. In early studies of the effect of GBS prophylaxis, several groups reported an increase in E. coli sepsis after starting the prophylactic antibiotics. In one study published by Schtomm et al. in 1999, they found that GBS sepsis fell from 1.7 per 1,000 deliveries to 0 per 1,000 but that E. coli related sepsis rose from 0.3 per 1,000 to 1.3 per 1,000, which resulted in no net change in the overall sepsis rates. This and other studies cause some concerns. However, larger studies and meta-analysis were performed to study this further. In a large study published in 2001 in Pediatrics from 19 Connecticut hospitals, they failed to confirm this rise in E. coli, but did show a large increase in the resistance of E. coli to ampicillin. Several other studies followed suit, and the fear of rampant E. coli thankfully dissipated. However, the resistance issue did cause a change in treatment recommendations to penicillin, because it has a much narrower spectrum of coverage and does not result in the same levels of resistance.

This all leads me to today and the current recommendations from the American College of Obstetricians and Gynecologists. These guides are similar to almost all other developed countries. They recommend universal screening at 36 weeks pregnant, and if positive, to treat with penicillin for at least 4 hours before delivery if possible. As a little side note here, that 4-hour recommendation is essentially made up, and there is almost no data to support any of it. There is one study I could find on the topic, and they did find that 1 to 2 hours is probably sufficient, so take this time recommendation with a grain of salt. Anyway, if GBS positive women are allergic to penicillin, then ACOG recommends testing the GBS for resistance to both clindamycin and erythromycin, as there’s often cross resistance between them. If it is sensitive to both, then they recommend giving clindamycin. If resistant to one or both, then they recommend vancomycin. You might ask, why not use a cephalosporin in women with a penicillin allergy? Well, one study did look at that and they found it to be effective and safe with very few allergic reactions to the cephalosporin. Using a cephalosporin in these cases is unfortunately not in the guidelines, but it is something to consider as opposed to giving vancomycin. They also recommend treatment, regardless of testing, for women with GBS bacteriuria and for women who had a previously affected neonate. As I mentioned, there is not a lot of data here, so it’s hard to say yes or no on this one, so I think erring on the side of caution is not wrong. For women in preterm labor they recommend treatment if their GBS status is unknown or positive, but state that treatment can be differed if they have a recent GBS-negative swab. Lastly, they recommend that we fall back on the list of high-risk factors for treatment if a woman is term but the GBS status is unknown.

Let’s contrast this to the guidelines of the Royal College in England. As I mentioned, they do not recommend universal screening but do recommend treatment if there GBS bacteria in the urine or a history of a previously affected neonate. They also do not recommend treatment in cases of preterm labor with intact membranes because, and I quote, “although the risk of early onset GBS infection is higher in preterm than in term infants, there are reasons to be cautious about widespread prescriptions of antibiotics for those women, because approximately 50% of all women thought to be in preterm labor will not deliver preterm.” Then they go on of course to quote the ORACLE study again. For treatment, they do recommend penicillin and then state that given the resistance rate of only 10% to clindamycin in the United Kingdom, there is no need for antibiotic testing like we do here in the States.

When I start these projects, I often have no idea what I’m going to find. More often than not I find the data that our guidelines are based on is poor or represents expert opinion. Therefore, it was nice in this case to find so much data to back up our guidelines, and a little disheartening to find that there are some places in the developed world that don’t follow them. Interestingly, as women become more educated about these topics, they’re starting to demand GBS testing, as can be seen by the numerous labs in England that offer private screening with a self-administered swab. There is an ongoing review of the GBS policy in England, and I think that they will soon join the rest of us in universal screening. This is not to say that we should not keep looking at this, and if a group is not already doing this, I hope that someone will look at the effects of penicillin on neurodevelopment so that we can answer the question of its safety.

Before I finish up today, I want to talk about one more aspect of this topic, and that is ways of eradicating GBS before delivery. This is a growing topic, at least here in the U.S., and I think it deserves some attention. This idea, on the surface, is a great one. If we could test for GBS and then offer positive women a way to eliminate the colonization prior to delivery, that would help eliminate the need for antibiotics. Whether we use a natural method or a more medicinal method, it could be a cost effective and safe way of dealing with this GBS risk. Natural methods usually revolve around probiotics given prenatally.

There is very little data here, but one pilot study from Safdar et al. from 2014 took 20 women and gave half of them daily Florajen3 probiotic, which is mostly lactobacillus. The idea behind this is that the lactobacilli outcompete group B Strep and over time, this will decrease colonization. The study found 2 women in each group were colonized, but, interestingly, the women in the probiotic group were less densely colonized. First off, it’s nice to see that 20% number come up again, which fits our previous studies. And secondly, since we know the risk of sepsis is correlated with density of colonization, maybe there’s a beneficial effect to these probiotics. The group states that they’re going to perform a larger study to investigate further, and I am eagerly awaiting their findings. For now, though, I would say that women can certainly take probiotics during pregnancy, and that they may decrease the rate and density of GBS colonization. But until we have more evidence and more data, it should not replace the usual screening and treatment that we have today.

The other method of reducing colonization is to perform a lavage of the perineum, anus and vagina with either chlorhexidine or iodine. Again, there is little data here. What there is studies the effects of the lavage at time of delivery and not antenatally. In one study, they gave 31 positive women a vaginal and perineal lavage with chlorhexidine, and then took a swab 4 days later. They then compared those results against 74 women who had no treatment. About 15% of the women in the control group were GBS negative 4 days after delivery, while 32% of the women who received the chlorhexidine were GBS negative. This does show that there is an effect and may influence the number of GBS-affected neonates even with penicillin treatment. But that given that it only showed an effect it about a third of women means that it cannot replace screening.

Another larger study from South Africa looked at using chlorhexidine wipes for the vagina prior to birth, and then again wiping the neonate after birth. However, they found no decrease in newborn colonization or early onset sepsis. Maybe this is due to the fact that they used wipes and not a lavage, which I could imagine might have a different effect. I would be interested to see what effect a lavage in GBS positive women at 36 weeks has. In my mind, I can see a schema wherein we test women at 35 to 36 weeks for group B Strep. The women who test positive take a probiotic and do the lavage. And then we test them one to two weeks later, and if negative, then they won’t need treatment at delivery. If even moderately successful, it would eliminate the need for antibiotics for a large number of women, for the cost of the lavage and a re-swab. I think it’s something to think about, and honestly, I feel a little new research project coming on. Now I just have to find a resident who’s interested in group B Strep.

In both cases, though, we are still waiting for good data, and I have to withhold judgement until we have it. But I do think that each of them holds promise and warrants further research. Although I probably will be more likely to get on board with my patients taking a probiotic as opposed to using the chlorhexidine, because I’m not sure what the effects of chlorhexidine are on mucous membranes like the vagina, and that makes me a little nervous.

In any case, I think that’s a good point and I’m going to wrap it up here. We talked about the natural state of early onset sepsis and the risk to babies. We covered the risk factors and two of the most common screening algorithms. We went in depth into the data behind universal screening and treatment and the risks and benefits that go with it. We also discussed the risk-based method and the pitfalls that I think are associated with it. And lastly, we talked about some new ideas in prevention or treatment of GBS colonization that I find really intriguing. Like I said earlier, one of the things that drove me to do this podcast is my desire to learn about the why behind what we do. Digging into this topic was fascinating for me, and I hope that you all enjoyed hearing about it as much as I enjoyed researching it. When we understand the data behind what we do, we are better able to apply it in our clinical practice and better able to educate our patients. In the case of GBS sepsis it was nice to see that what we are doing in clinical practice is what the data supports.

So, thank you all for listening. As you know, you can find the show notes at [www.obgyn.fm](http://www.obgyn.fm) and you can follow me on Twitter @JChappelleMD or on Facebook at the OB/GYN Podcast page. For those of you on Slack, I can’t wait to discuss this episode, and for those of you who want to get in on it, please just send me that email to feedback@obgyn.fm and I’ll get you an invite. This episode concludes our puerperal fever discussion and we’ll be moving on to some more bread and butter OB stuff in the next few episodes before we turn the ship back to GYN. With luck, the next episode will be a little different and with a new voice, so you can all look forward to that. But until then, thanks for listening.