So you have a patient who has been on monotherapy, Levothyroxine, for a few months now and the patients are still asymptomatic. And you identify that maybe they're not getting enough penetrance or they don't have enough circulating levels of T3 that's not being adequately monitored in the lab type identify those patients. So you want to do that you want to add T3. So how do you do it?

Well, there's several ways, and the conventional way that you'll see is-- certainly in the thyroid guidelines that are out there for anybody just to Google is that you can add a synthetic T3, they're generally short acting, and liothyronine, which is Cytomel and you can add that dose to your levothyroxine. I don't recommend that, by the way, in primary care. It's a little dicey. You have to cut back, usually cut back your LT4 dose by 25 micrograms and then you can start this other pill, Cytomel two point five to seven point five milligrams. Usually requires multiple split doses during the day, kind of awkward and not a steady state. It's just awkward to use.

The other option is a synthetic combination, which is T4 and T3 in combination. It's synthetic, so you have Levothyroxine and almost like liothyronine together, it's combined and that's Liotrix. And that's another option that's out there and I've got a number of patients who've been on that.

But there's one option that I would like to posit that's been around for a long time that is also a combination thyroid T4/T3 medication that you may be familiar with-- certainly have heard about it and it's been around a long time. I'll tell you one thing, a lot of your patients have heard about it. In fact, there's been big names in the political crowd who, back about four years ago, made news having been placed on a dessicated thyroid extract. So people are talking about it, your patients are talking about it, they're going to ask you about it. So I want to spend some time chatting about DTE. Now I've been in this business a long time and-- a long time and I've used DTE in practice deliver it to patients.

But you need to understand that dessicated thyroid extract has been around for about 130 years, probably longer than that if you include China. It was first, I believe was first utilized in a patient 1891. Dessicated thyroid extract, it probably came from sheep back then-- they used different animals. But then that patient was on it for 52 years. 52 years treated with dessicated thyroid extract.

Now, that's all we had that was all available. Thank goodness we had that to help manage thyroid hormone replacement in the hypothyroid placement, otherwise a lot of people-- it saved a lot of people from death and horrible morbidities. So that was used for a long time. Understanding the way drugs and therapy was in those days, the way that the drug was processed, it wasn't consistent in the quality of the pill, there wasn't consistent doses of T4 and T3 early on for a long, long time. And people knew that was a problem, but they had. And I'm going to tell you, this helps us understand a little bit of the biases and the preferences-- biases pro and against use of DTEs. And this is one of the biases against because there's people still remembering that you didn't have consistency of dosing in these products. It's not the case now.

So, synthetics. So we knew there was a problem with this and so the synthetics came out at about 1962 ish around, I think, is when they came out with the first synthetic, Levothyroxine. Didn't make a splash. People, providers were comfortable using DTEs. I'm going to tell you, there was a big push in the 70s-- I'm just showing you how this is an amazing journey and why we are and how we look and evaluate the use of DTEs now. But in the 70s a big push-- I remember headlines, because I was working back then, to get rid of all DTEs, go to synthetic. That was the news and the headlines, but there was still a hesitancy in actually using that by providers and also by the patients.

Then something happened in the 80s that changed it all. And that is with the radioassay of TSH, finally there was a lab that was able to give you an indirect identification of the adequacy of circulating thyroid hormone. And with that and also measuring thyroid T4 and looking at the TSH, wow. That became really cool because understand, before that, what we use with measuring basal metabolic rate and peptide binding iodides, nothing was good. TSH was groundbreaking so then we can actually assay circulating levels of T4, or indirectly. So you can't diagnose, again, with TSH but you can certainly get a good idea of how those thyroid levels were managing.

But that wasn't enough to push us over. The other thing was-- and this is key-- and colleagues, this is what changed my mind and got me to prescribing synthetic at that point is because we knew that patients need T4 and T3. We know that this combined. And T3 is the metabolically active hormone and dessicated thyroid hormone-- it's important you understand, this is the combination T4/T3.

We found out in the 80s that the majority of T3 comes from T4. And that changed everything. So then we thought, well. oh you don't need to give somebody T3 and T4, you can give them T4 and a lot of it will be, in the needs of the body through feedback mechanisms, you're going to have production of T4. Now I mention that and I take time to mention that, but this was huge. So you have the TSH, which became now the gold standard, for better or worse, for 40 years and then we looked at you could just use monotherapy. That was the start of it, that's what we thought about and let the body's own innate divine design work. Let T4 predominantly come from-- T3 come from T4. And then you can activate it, inactivate it and do what we can with the inactive forms of T3. So that's what changed.

And finally what got the consumers on board: safety, safety, safety. The drug was safe Levothyroxine was safe. It was consistent blood levels of T4 hormones. It was long shelf life-- that was a problem early on with DTEs, shelf life. Now that's not the problem anymore, but that was a problem back then. And the big thing was cost. Boy, have things changed then. So costs came down, consumers bought and that became, colleagues, why we're looking at the age of LT4 monotherapy.

Now we're finding, in the last 10 years, people are willing to talk about, not all patients are being treated, being relieved the symptoms. The labs are not as accurate for looking at-- we can't see, there's no biomarkers for tissue penetration of T3. We know that symptoms aren't relieved in everybody. Levothyroxine as monotherapy is not the Holy Grail for all patients. And remember, we need to treat symptoms and we have to alleviate and minimize comorbidities.