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ReachMD
www.reachmd.com
info@reachmd.com
(866) 423-7849

The Fifth Pillar? Closing the Gap in HFrEF

Announcer:
Welcome to CE on ReachMD. This activity, titled “The Fifth Pillar? Closing the Gap in heart failure with reduced ejection fraction” is provided by Medcon International.

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Dr. Lam:
There have been some very interesting developments in the world of heart failure recently, with data presented at the European Society of Cardiology and Heart Failure Society of America meetings. Today, we are going to discuss some of these exciting new findings and, very importantly, talk about the clinical implementation and the clinical implications of these findings in our patients with heart failure, in particular, heart failure with reduced ejection fraction.

This is CE on PACE-CME and ReachMD, and I'm Dr. Carolyn Lam.

Dr. Butler:
And I'm Dr. Javed Butler.

Dr. Greene:
And I'm Dr. Stephen Greene.

Dr. Lam:
So let's get started. Dr. Greene, you've written a lot about guideline-directed medical therapies for heart failure with reduced ejection fraction, or HFrEF. Do we really need more? I mean, you've got the quadruple. What more? Tell us about it.

Dr. Greene:
Yeah. So first and foremost, I'll say we definitely do need more, to be quite frank. I mean, yes, quadruple medical therapy has enormous benefits. I mean, when you put the 4 therapies together—we're talking about ARNI, beta-blocker, MRA, SGLT2 inhibitor—projected more than 70% relative risk reduction in all-cause death. Huge absolute risk reduction, number needed to treat of less than 4 to save a life over 2 years.

Another way to phrase it is that if you take a 55-year-old man and you have them on double therapy but you upgrade them to quadruple medical therapy, you could extend survival by 6 years for that patient's life. So, yes, quadruple medical therapy does work and it works well, but we also need to have humility for what we're dealing with here.

And then I would argue that HFrEF, whether it's worsening HFrEF or even outpatient HFrEF, is really an extreme-risk condition. We look at absolute event rates, much higher than what we see in ASCVD. And even though quad therapy makes it lower, it does not make it low risk in terms of absolute risk, and there's no such thing as a low-risk HFrEF patient.

Dr. Lam:

So how about put it into numbers for us? So even with quad therapy, is that risk, in terms of the ASCVD risk, how does that compare? Is it still higher?

Dr. Greene:
Absolutely, still higher. I mean, just to give you some more context, too, you can look at secondary analyses of clinical trials or EMPEROR-Reduced, for example, which is a trial of SGLT2 inhibitor versus placebo. I think it's really an example of what kind of residual risk we're talking about, even with quad therapy. In EMPEROR-Reduced, you have the empagliflozin arm; 100% got SGLT2 inhibitor. But you look at the background therapy, 90+% are getting beta-blocker; 90% are getting the ACE/ARB/ARNI; more than 70% are getting MRA. And even with that level of quad therapy, much higher, unfortunately, than we ever see in real-world practice, we're still talking about an absolute event rate for CV death or heart failure hospitalization of over 15 events per 100 patient-years.

Just to put that in context, when we talk about ASCVD and the people that even in the lipid guidelines would meet criteria for being very high risk, those patients are generally like 5% to 8% pre-risk of MI or stroke. So, again, I'm not saying those aren't high risk or very high risk, but if we're going to compare apples to apples, our absolute event rates really are, in my opinion, the extreme risk range, even with quad therapy.

Dr. Butler:
It's interesting that, I've heard this quite a lot, that there are 4 drugs already. Like, do we really need more therapy? And then the question is, how do you answer that question? And the answer simply cannot be numerical, right? You cannot say that 2 drugs are too few and 4 drugs are too many, so 3 is the right number. And the problem here is that the heart failure event rate was this high, and on 4 drugs, it has come down to this level, which is great. Except that the age-sex match people who don't have heart failure, their outcome is here. And that's the gap that we need to fill.

Dr. Lam:
Yeah, that is so true. And we keep talking about relative risk reduction. I mean, that was like 70% relative risk. But relative of a very high absolute risk still leaves a lot, right?

Dr. Greene:
Yes, absolutely.

Dr. Lam:
And I think this is what we're really referring to, that residual risk. Talk about high absolute risk. We, the 3 of us actually, we embarked on the VICTORIA trial, didn't we? With one of the highest absolute risks I've ever seen in an exclusive HFrEF outcomes trial. Do you want to tell us about it? Maybe Dr. Greene first, and then.

Dr. Greene:
Sure. So, I mean, just to refresh everyone's memory, the VICTORIA trial was the pivotal registration trial that got vericiguat FDA-approved. But just to set the context for this, so on one hand, it had many criteria very similar to a large phase 3 HFrEF trial. Patients had to be on good background medical therapy at the time, had to be symptomatic, reduced ejection fraction, in this case, less than 45%. But the unique thing around the VICTORIA patient population was that every single patient had to have a history of a recent prior worsening heart failure event. So it really enriched the baseline risks that we're talking about in this patient population to a level, like you said, Carolyn, that really hasn't been seen in our standard HFrEF trials before.

And just to cut to the chase with the results, this is vericiguat versus placebo, titrated to a target dose of 10 mg, and VICTORIA was a positive clinical trial. There was a 10% relative risk reduction for the primary endpoint of CV death or heart failure hospitalization. But again, because this was such a high underlying baseline risk, that 10% relative risk reduction translated to a very large absolute risk reduction of over 4 events per 100 patient-years, low number needed to treat. So, again, because of these data, as well as vericiguat being safe and well tolerated, really no different than placebo, vericiguat was approved for patients with recent worsening heart failure.

Dr. Lam:
And then, Dr. Butler, I remember that you published after that, sort of really highlighting the absolute risk reduction in VICTORIA and comparing it with the other big, famous outcomes trial. Tell us a little bit more about that and then why we went further after VICTORIA.

Dr. Butler:
Yeah. So let me highlight something that Dr. Greene just mentioned. VICTORIA trial was not a trial of hypotensive patients. VICTORIA trial was not a trial of patients in acute renal failure. It was not a trial of patients with decompensated heart failure in the ICU, balloon pump. These patients, granted that they had worsening heart failure, but the definition was hospitalization within the past 6 months, or not even requiring hospitalization, just required outpatient IV diuretic within the past 3 months, right? 89% of the patients were enrolled

in the outpatient. In other words, they were treated by the doctors and the nurses the best you can, and they were 2, 3, 4, 5, 6 months post discharge.

The annualized event rate was in excess of 35%. 35%. Imagine if you're a 60-year-old person with heart failure and you have more than 1 in 3 chances of dying. So this is the whole problem that we are trying to tackle. So because of that high risk, a 10% relative risk reduction translated, as Dr. Greene mentioned, about 4.2% absolute risk reduction.

Now because, first of all, it's an unfair comparison unless and until you do head-to-head comparison, you cannot compare. But just for our audiences who don't look at the absolute risk reduction on an ongoing basis, just to give an idea, this is sort of right in the middle. So if you look at, for instance, DAPA-HF, it was 4% absolute risk reduction. If you look at EMPEROR-Reduced, it was 5.2%. Here, it's 4.2%. So it's also sort of in the same ballpark.

But the other very interesting thing that we saw is that, remember, there comes a point where people are too sick and medical therapy doesn't work. Now you're sort of in the device, transplant. And in these patients who were sort of 3, 4, 5, 6 months post-discharge, many of these people were hanging around with an NT-proBNP, not the decompensated NT-proBNP, but the refractory NT-proBNP of 8-, 9, 10,000 and those patients seemed to not benefit as much. So we got this really good interaction that the NT-proBNP in the lower 3 quartiles, even in this relatively short-term trial, there was a mortality benefit of an excess of 20% relative risk reduction. Now, it's a subgroup analysis, and the absolute risk reduction was in excess of 7%. So really good, good data. So those were all the things that led to the VICTOR trial.

So the 2 things that we wanted to achieve in the VICTOR trial: One, was to now take the population to have the results of the vericiguat and heart failure across the spectrum. So these were the patients who did not have worsening heart failure. So no hospitalization within the past 6 months and no need for outpatient IV diuretic within the past 3 months. But also, with this whole lesson of NT-proBNP, it restricted the patients with NT-proBNP of less than 6,000 in the VICTOR trial, which by the way, in the real world, is the vast, vast, vast majority of patients. But we didn't want to get that end-stage advanced heart failure patient.

Dr. Lam:
Yeah. I think if I could phrase it a different way, so VICTOR plugged the evidence gaps in terms of the population of heart failure, and it made sure that we were treating patients with modifiable risk.

Dr. Butler:
Yes.

Dr. Lam:
I mean, honestly, right? NT-proBNP in the tens of thousands? We all know that's, like, go look for something else. That’s like something really, you’ve got to look for some strange esoteric causes or start thinking about VADs and end stage. So modifiable risk and in—I don't want to use the word stable, and we'll tell everybody, because it's not. This is like you would never call cancer stable.

Dr. Greene:
Lower risk, but not low.

Dr. Lam:
Yeah.

Dr. Butler:
Symptomatic stable, not stable heart failure, something like that.

Dr. Lam:
For those just tuning in, you're listening to CE on PACE-CME and ReachMD. I'm Dr. Carolyn Lam, and here with me today are Dr. Javed Butler and Dr. Stephen Greene. We’re discussing data that were recently presented, including at the 2025 European Society of Cardiology and Heart Failure Society of America meetings. We are giving our perspectives on the clinical implementation and implication of these very exciting findings.

Right, right. So then we embarked on VICTOR. Okay, we can come back to why we did it this way, but first, can you tell us the results of VICTOR?

Dr. Butler:
Yeah, so the VICTOR trial was a large outcomes trial. 6,000 patients or so enrolled, NT-proBNPs less than 6,000. This group of patients we have never studied in a heart failure trial before. One, because we did not want to take any patient with worsening heart failure. We really moved the sort of the whole right pursuit really, really, again, I don't know how to avoid the word stable. But really that

group of patients who were sort of outpatient, doing sort of okay. So just as a comparison, again, if you look at the recent trials with other agents in the HFrEF space, they have about 60%, 70% NYHA Class II patients. Here, we had 80% NYHA Class II patients. General baseline GFR 58, 60. Here, the GFR was over 70. Here, the hospitalization, although we excluded patients with hospitalization within the past 6 months, prior hospitalization outside of 6 months were allowed. But only 15% of the patients were hospitalized between 6 and 12 months. So over 85% of the patients were either never hospitalized, about 47%, and then the rest had remote hospitalization 2 years, 3 years out or something like that.

Now, not only is that sort of getting a group of patients that we have never studied, the baseline medical therapy in this trial, we have never seen before. 95% with RAS inhibitor use, 95% beta-blocker use, and of that 95% RAS inhibitor use, close to 60% was ARNI, and close to 60% was SGLT2 inhibitor, and close to 80% MRA, and a third had ICD implanted as well. So it's a very different sort of population that we studied.

The primary endpoint was cardiovascular death, heart failure hospitalization. There was a 7% reduction directionally, going in the right direction, but did not reach statistical significance for the primary endpoint.

But there were so many firsts about this trial: the population that had never been hospitalized, the baseline medical therapy. But here's the new first, and that is that we have never seen a heart failure medical therapy trial before where we did not see a statistically significant benefit for heart failure hospitalization, but there was a statistically significant benefit for cardiovascular mortality. Now remember, this trial was designed to address the issue of cardiovascular mortality. It was powered for cardiovascular mortality. We had over 600 mortality events. And the consistency, there was a statistically significant reduction in cardiovascular death, in all-cause death, in heart failure-related death, and in sudden cardiac death. So that's where we are now.

Dr. Lam:
Wow. Gosh, this is incredible, right? So taking a step back, what we just talked about is VICTORIA. High risk, positive trial, but mainly driven by reduction in heart failure hospitalizations and correct direction, but neutral for cardiovascular death. Then we have VICTOR. Neutral overall, but a strong signal for survival benefit, like death. Like you couldn't get more objective than that.

How are we going to put this all together is the thing, right? If I may now share. I mean, we did a pooled analysis, and this was prespecified. That was very important, of course, right? Putting the 2 together in what now becomes the largest heart failure reduced ejection fraction program ever of more than 11,000 patients, right? And it was designed such that these were admitted or hospitalized 6 months prior; these were not.

So it kind of fits nicely in there, right? And if I may summarize the overall results of the pooled analysis, there was a positive overall primary outcome, where the primary outcome was cardiovascular death or first heart failure hospitalization reduction. With, talking about absolute risk reduction, that was very impressive. I think it was 2.7 per 100 patient-years, and that was a number needed to treat of only 37.

So this overall program was highly successful. And then, of course, now if you look at the components in it, there was a reduction, statistically significant, of both the heart failure hospitalization and the cardiovascular deaths. And then, let's not forget that this is a compound that's really easy to give, once a day, oral, and there were no new safety signals.

Now, we put all of that together, what are the clinical implications of these data? I mean, Dr. Greene, what do you think?

Dr. Greene:
Yeah, I mean, well, the first thing, I think, to get through to the clinical community—and people will look at VICTOR and they say, well, it's a neutral trial for the primary endpoint and they suddenly lose attention—but I think we have to try to convey here, just taking a step back. Yes, from a pure statistical perspective, you could argue once the primary endpoint is neutral, everything else is exploratory, hypothesis generating, and you need another study. But just, kind of using my, quote/unquote, maybe commonsense hat and just taking a step back with how to think about VICTOR, I mean, the mortality signal in VICTOR didn't come out of thin air, right? This was generated based on the hypothesis that Dr. Butler kind of elaborated on, where in VICTORIA, in the bottom 3 quartiles of NT-proBNP, yeah, you saw a magnified benefit for the primary endpoint, the composite inventory, but you also saw a CV mortality benefit. You also saw all-cause mortality benefit.

So then, as a good scientist, you would say, well, now let's validate that. Let's do the second trial. And lo and behold, VICTOR validates that, you could argue. So I think that's one piece of evidence. The other thing, as Dr. Butler said, this trial was powered for CV mortality. So we have a lot of precision and, again, I would say liability. If you power a trial for CV mortality and you lower CV mortality, you think it's probably the truth. And then you also highlight the meta-analysis when you put everything together. Although in VICTORIA and VICTOR, people might say, oh, it's totally different results, I would argue the meta-analysis says these trials are much more similar than

different. Really, the treatment effect was consistent, statistically speaking. And again, when you put the trials together, replicating what an essentially normal heart failure trial design looks like, when you have a mix of patients that have recently hospitalized, people that are never hospitalized, and put them all together, you get a positive trial.

So I think from a clinical community, I would say to my mind, the first thing to understand is that I think the mortality benefit is real. Although again, statistically people are going to maybe argue against me, but I think that's the first thing to communicate.

Dr. Lam:
That's a very strong point.

Dr. Butler, let's just talk about the elephant in the room. Why was VICTOR's primary result neutral? Why can you lower mortality without lowering heart failure hospitalizations? And maybe go into a little bit about what we're learning about. There's a lot more heart failure hospitalizations and deaths in VICTORIA and a lot less heart failure hospitalizations and deaths in VICTOR than VICTORIA. I mean, what's going on here?

Dr. Butler:
Yeah. So a couple of things. So first of all, there is a narrative out there that VICTOR and VICTORIA have the opposite results. In VICTORIA, we had hospitalization but no mortality. In VICTOR, mortality and no hospitalization. And I think that's a little bit of a false narrative. At least the way I am looking at the results, the results are exactly the same across the 2 studies. In VICTORIA, there was hospitalization benefit, and the mortality benefit signal was very strong if you had a little bit of a longer follow-up in those advanced heart failure patients that were sort of diluting that out.

Now, when we come to VICTOR, just think about it. If you have a patient in the clinic or a group of patients in the clinic, they have never been hospitalized—by the way, I forgot to say, 30% of these patients were not even on a loop diuretic, right? So, again, that just tells you that this is a group of patients, so excellently well-treated patients, really less symptoms, 80% NYHA Class II, not on loop diuretic, never been hospitalized.

Now, they come to you, and I have a little bit of lower extremity edema. I'm getting a little bit short of breath. I'm not feeling well. What do you do? Well, you start them on oral diuretics. If they're on oral diuretics, you go ahead and increase their dose. If they're on high doses, you go ahead and add a second diuretic, like a thiazide diuretic or something like that.

So we have learned a lot, both in observational studies and in clinical trial secondary analyses from other trials, that this outpatient manipulation of oral diuretic is the first sign of worsening heart failure and is associated with prognosis. So we said, well, let's look in the VICTOR trial and see what happened. Oral manipulation, oral diuretic manipulation in outpatient setting increased the risk of mortality by over 60%. The hazard ratio was 1.69. So we validated it.

So then we said, well, let's look at the totality of worsening heart failure. Did that get better or not? And the answer was absolutely yes. So at least my interpretation is that when you get these patients who are really well treated, never hospitalized, not on diuretic, we are seeing worsening heart failure signal, but that signal is not the hospitalization signal. So to me, the issue is not the intervention, but the way we do trials is that it's just a norm to do cardiovascular death, heart failure hospitalization. But I think in this modern era, we have to start thinking a little bit differently.

Dr. Lam:
Very interesting. So what you're saying is that the oral diuretic intensification could be like a surrogate of hospitalization in a modern era, in a relatively, here we go again, stable population of HFrEF. And so if we counted those in VICTOR, it would be a positive effect of vericiguat.

Dr. Butler:
Yes.

Dr. Lam:
Could I say it a different way? So maybe VICTORIA was underpowered for CV death and VICTOR is underpowered for heart failure hospitalizations because of the underlying risk of these basic populations.

Dr. Greene:
I mean, the way I think of it too is that VICTOR, essentially, was de-enriched for downstream heart failure hospitalization. Normally, some trials, they require enrichment of a prior heart failure hospitalization to get in, to get the event rate up. But here, we're having an endpoint that includes a component of heart failure hospitalization, yet our characteristics de-enriching for recent heart failure hospitalization. So we've never really done that before as an experiment, and this is what we get.

But anyway, I do think, to Javed's point, I think it's absolutely correct to think about totality of worsening heart failure when you include outpatient hospitalization. There's clearly a signal in VICTOR.

Dr. Lam:
That's so cool. And if I could share what we shared in Heart Failure Society of America, was a regional look at the VICTOR results. And very interestingly, in the countries where there is a greater heart failure hospitalization practice, and that is North America and Asia-Pacific, where the heart failure hospitalization-to-death ratios are higher, you see a stronger impact and signal with vericiguat versus areas where there are low hospitalization-to-cardiovascular death. That was Western Europe, for example. The signal is not so strong, but that is the area that the outpatient diuretic intensification increased the number of events. The greatest was Western Europe. And then you shift the signal of benefit. It's very obvious. So again, very, internally at least, consistent results that what we're seeing is real.

Dr. Greene:
Yes.

Dr. Butler:
And I'll make a couple of points, right? So one is that we have to be conservative in our assessment of results, right? So at the end of the day, whenever you do subgroup analysis, you'll always find some subgroup, right? So there are, you know you have to control for multiplicity, and we have to be conservative and that's good. But the question here is that, first, nobody was claiming mortality benefit in VICTORIA, although we saw that signal, right? But you generated a hypothesis, and then you did the hypothesis, and you proved it in the VICTOR trial.

Now, we all know that the choice of primary endpoint is a subjective choice. We could have made cardiovascular mortality the primary endpoint and the combined endpoint the secondary. But the way the endpoints are chosen is what is happening in the environment today, and that's how you sort of choose it. So now, the question is cardiovascular mortality. If your primary endpoint is neutral and you find a signal of improvement, either by accident that you are not looking for—which is not the case here, remember, we powered and looked for CV mortality—or you found a signal for an outcome like patient felt better or their biomarker got better, well, maybe you need to retest it or generate hypothesis.

But let me ask you a question. If you were to do a study on arthritis, a rash, something like that and you did a trial and it was gloriously positive, a really positive trial, but there was a higher risk of mortality, would you ignore mortality?

See, that's the problem. That mortality signal cannot be ignored. And now, if you put it in the perspective that here the mortality signal was not only consistent, strong, but also was hypothesis-driven and powered for, that's the point.

Dr. Lam:
Yes. Yes, it truly is. And if I may extend that, why would you not give it? Let's talk about the safety data.

Dr. Greene:
Yeah.

Dr. Lam:
What's the price?

Dr. Greene:
I know. From safety and tolerability, I mean, it's right up there as far as any of our GDMTs in terms of safety and tolerability. No difference versus placebo, really, in terms of the people getting to the target doses, side effects, adverse events. You know, even things that we care a lot about. Things like symptomatic hypotension. I mean, so again, the safety and tolerability make it kind of like an easy button when you think about our GDMTs in terms of use. So, again, another reason to err on the side of saying yes to this instead of giving it a hard time and really criticizing, I think.

Dr. Lam:
Yeah. One more thing: Is the VICTORIA and VICTOR patient really different? I mean, we have a patient—I can admit it, I'm a VICTORIA patient. And then I stay out of hospital more than 6 months, now I'm a VICTOR patient. Now, I mean, it's actually transitions through the patient's journey. And as we have consistent benefit, right?

Dr. Greene:
Yeah, and it was so great to see the NT-proBNP hypothesis, which is, again, a biologic marker of risk, become validated in VICTOR. So now we're, again, seeing it twice. And again, mortality benefits in both the post hoc from VICTORIA, now validated in VICTOR. So, again, that's that consistent biology that I think you're getting at. So, again, there's really remarkable consistency there.

Dr. Lam:
Wow. You know, this has been an incredible discussion. I'm going to really put you guys on the spot now because I stuck my neck out. I did. And I will say that at the ESC, I did say I think we have a fifth pillar in guideline-directed medical therapy for HFrEF. And if we question if we have a fifth pillar, we need to understand, is this mechanism of action different from the others? Dr. Greene?

Dr. Greene:
Yeah, so it's a great question. I mean, we have our 4 pillars that we talk about and they work through very, I would say, well-established pathways for the most part. We're talking about sympathetic nervous system with beta-blockers, the renin-angiotensin-aldosterone system, neprilysin inhibition. SGLT2 inhibitors work through a whole host of cardio and kidney mechanisms, potentially.

What we're talking about here with vericiguat is that it's a soluble guanylate cyclase stimulator, so it kind of interacts in this other pathway. This is the nitric oxide-soluble guanylate cyclase, cyclic GMP pathway. And what happens in HFrEF is that we have reduced bioavailability of nitric oxide. This kind of shuts down this pathway and leads to a downstream cyclic GMP deficiency.

And you say, well, what's cyclic GMP good for? Well, it's good for a whole host of both cardiac and vascular benefits, endothelial function and the like. So what vericiguat does is it acts at that middle step in this pathway, directly stimulates soluble guanylate cyclase and kind of corrects this cyclic GMP deficiency.

Dr. Lam:
Yeah. Interesting, right? It's actually stimulating the good stuff.

Dr. Greene:
That's right.

Dr. Lam:
Whereas everything else shuts down the bad stuff.

I would love to know your thoughts. Should we be considering vericiguat in the clinical implications? Again, we know that the guidelines have not changed yet. We know that you need to prescribe it as allowed by regulatory approvals in each of our countries, yes. But just looking forward, and maybe Dr. Butler first and Dr. Greene.

Dr. Butler:
I would say that considering the safety, tolerability, one thing where vericiguat stands out is that vericiguat and SGLT2 inhibitors are the easiest drugs to use and best tolerated. Our patients are at a high risk and that mortality simply, in my mind, cannot be ignored. And there are really nice data that show that, you know, we talk about the vulnerable phase, that vulnerability is primarily for hospitalization. So just before hospitalization, hospitalization, just outside of hospitalization, you see sort of the hospitalization rates vary. But there's a consistent risk for mortality, consistent risk for sudden cardiac death. You know the first time there was a statistically significant benefit for sudden cardiac death in the VICTOR trial was 5 months? So these things do occur for these patients that I would find a really hard time to not give vericiguat. How about that?

Dr. Lam:
I heard yes. Okay.

Dr. Greene:
All right. So my take. I mean, again, the word “pillar,” we can debate what that means, but just here's the facts that I think. I mean, we have 5 mortality-reducing drugs in HFrEF, the way I think. And historically, we've labeled the 4 pillars as unique because they all had all-cause mortality benefits. And I would argue now, based on totality evidence, I think vericiguat offers a CV death and all-cause death benefit.

Now, again, not going to take anything away from the 4 pillars. We're not trying to replace 4 pillars, not saying anything bad about that. But again, I just say the phrase, “why not”? Again, you have a prognosis comparable to a cancer in terms of death and morbidity. You have now a fifth therapy that not only reduced mortality but is safe and well tolerated. Why would you not want to give a therapy that you truly believe reduces CV death, all-cause death to a patient population that needs it more than almost anything in medicine to reduce death?

And what we're talking about here is, scientifically, a patient population that I argue needs all the help it can get. And now you have a therapy that we feel pretty confident that reduces risk of death and is safe, well tolerated, and pretty simple to use. So, again, I think I start with the phrase “why not,” from a scientific perspective.

Dr. Lam:

Yeah. A therapy that's safe and saves lives.

I'm afraid that's all the time we have for you today. So thank you. Thank you, audience, for joining us. Thank you so much, Dr. Butler, Dr. Greene, for sharing your thoughts and insights with us. It was great speaking with everyone today.

Thank you.

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