ACTTION-APS-AAPM

Pain Taxonomy for Acute Pain

April 29, 2016

A Matter of Record

(301) 890-4188

ACTTION

Developing the Framework for a
Comprehensive and Evidence-Based ACTTION-APS-AAPM
Pain Taxonomy (AAAPT) for Acute Pain

Friday, April 29, 2016
8:03 a.m. to 3:49 p.m.

Sofitel Washington DC
Lafayette Square

C O N T E N T S

C O N T E N T S (continued)

P R O C E E D I N G S

(8:03 a.m.)

DR. TERMAN: Good morning. I'm still Greg Terman from the American Pain Society. I hope you had a pleasant evening. I'm going to be introducing the speakers this morning. I think there's a little -- I'm sorry? Right, there's housekeeping.

So if any of you have any housekeeping issues in your rooms, Dr. Turk will --

(Laughter.)

DR. TURK: Dr. Terman is available 24/7 for any of you who have any concerns. He does windows and all kinds of other -- just very quickly, so you all know the importance of signing in and that there is taxi sign-up.

So for those of you that are looking toward going to airports this afternoon, you should sign up in the sign-up sheet. Make sure also you signed in.

If any issues have come up about your hotel room or about your travel plans, talk to Valorie or

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Andrea in the front. They tell me they're available 24/7. Any time you call them, you could expect that they will respond to you at the exact same time 24 hours later.

(Laughter.)

So if you call them at 3 a.m., in the morning, you're going to get a call back at 3 a.m. But that's all.

Any housekeeping issues, any concerns?

Lunch will be in the same place it was yesterday on the -- the second floor in the Madeleine Room, I believe. Any issues, any problems, anything that's come up? Hopefully, you all had an opportunity to resolve all the questions from yesterday.

Yesterday was a fun day, and then today, you get to work. And this afternoon, we're going to make sure that we finish off the beginning draft. Obviously, you'll see it subsequent times after that. And we will go as late as it takes until you've reached a consensus.

So we booked this room until midnight, and

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we're quite available. And no one is permitted to leave until that consensus. Greg?

DR. TERMAN: All right. So there's going to be -- I think it's eight talks about different, I guess, in yesterday's terminology, events that produce acute pain. And there will be a break in between at some point.

The people asked to talk about these events were asked to try and put those situations into the 5 dimensions from the original AAPT. And unless they changed their slides a lot last night, that's what they've done.

Steve has been kind enough to redo some of the work that we did yesterday, rewrite some of the dimensions that we talked about. But obviously, it's almost a philosophical question as to whether we really want to have completely different dimensions or whether there's a way to try and stay kind of close to the original AAPT.

So there may be ways -- there may be importance of host factors in chronic pain, for instance, and they may already be embedded in the

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previous AAPT output.

So the first person is going to be talking today -- and do please think about the integration versus kind of lumpers and splitters that we talked about yesterday. The first person is going to be Chris Wu, talking about surgical and procedural pain. He's from the Johns Hopkins Medical School. And I'll just get off the stage.

Presentation – Chris Wu

DR. WU: Thank you very much, Greg.

So as Greg mentioned, this was somewhat challenging. Instead of creating a new algorithm, we're trying to fit potentially acute procedural surgical pain into the dimensions we have, and then potentially propose potential other dimensions, and then looking at some surgical procedures or other type of procedures where we might apply whatever we come up with to sort of examine the reliability, the validity -- again, the 5 dimensions that we'll be discussing.

What I did was to sort of list onto the top what the original -- and I'm not going to read the

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entire thing -- what was for the chronic pain, and then try to at least give you an idea of what we might have for the acute pain, procedural and surgical pain setting.

So again, the first, Dimension 1 for the chronic pain, signs and the diagnostic findings. For acute pain, certainly, I think the signs and symptoms, you could think of it as a more -- again, this is up for discussion -- a more homogenous than we might see for our chronic pain colleagues or patients.

I think we mentioned this yesterday. The diagnostic test findings may not be as relevant because, obviously, we have an inciting event. So this part may be a little more straightforward because, presumably, you have a discrete injury or insult. So that might be a little different.

For the second dimension, the chronic pain, this is a little lengthy in terms of common features. Again, you think about what chronic pain has to do. We have to try to identify what might be CRPS and things like that.

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So again, you're going to have different characteristics, location, temporal qualities, other non-pain features that might affiliated with these diagnoses. And then obviously, these are not components of the core diagnostic criteria.

So for acute pain, I think some of these will be important. Some of these we discussed yesterday regarding the disorder.

We certainly want location -- some of these other factors, location, temporal qualities as we mentioned, whether we mentioned the long potential transition into chronic post-surgery pain, that's up for debate or may be placed in other locations. Obviously, descriptors are very important.

Then non-pain features, the only thing with this is that, yes, you can have things like numbness and fatigue, but as you'll see in there later on, for some of us who do acute pain -- again, Deb and I were just at a conference where the acute pain is no longer -- we're not interested in just the pain severity, but there's many other factors our acute pain patients will

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experience, things that will affect social functioning, other physical functioning, sleep.

So it's a multidimensional concept. Whether we put this here or other places, we can discuss that later.

The common medical comorbidities, again, with our chronic pain patients, we have many patients who have medical and psychiatric disorders. Particularly, this was mentioned, major depression. Again, that's not uncommon for someone who has a chronic pain condition.

Now, for acute pain -- and this was brought up yesterday -- typically, we'll just focus on the pain, treating the pain. But again, we are becoming more cognizant that it's not just a physical symptom, that we have to recognize that there are psychosocial issues involved with are our patients despite the fact that it is presumably a brief insult.

I can't remember -- maybe, Dan, it was your slide where they had pain is isolating. So these are things that we have not been really adept at

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addressing in our post-operative pain patients. So it's something that we probably need to do.

Obviously, I don't think it's going to be the major depression in most cases that our chronic patients will have, but certainly that is an important part that we need to address for our acute pain patients.

Again, we need to recognize the factors that may be an important for the development of chronic post-surgical pain.

In terms of the other medical comorbidities, we mentioned opioid-tolerant patients, and there may be other medical comorbidities where the treatment may be difficult, for instance, obstructive sleep apnea and use of opioids.

The fourth for chronic pain, we talked about the neurobiological, psychosocial, and functional consequences. Again, these are things like sleep and mood disorders. And again, similarly for acute pain, we have these. We have not focused on them, frankly, as much as we should. But certainly, we need to look at how pain will affect, even if it's

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brief, their social function, their physical function, and things like sleep quality.

Again, we are transitioning -- many of us are thinking instead of just a unidimensional assessment of pain to a multifactorial assessment, which includes not only the pain level itself but these other related factors.

Finally, the 5th dimension, the putative neurobiological and psychosocial mechanism, risk factors, again, this is self-explanatory for the chronic pain.

We do certainly have this for acute pain, although we don't think of it like that for our acute pain patients. But certainly, again, this is where we might think of our risk factors for the development of chronic post-surgical pain, if that's something we want to address.

There are certainly other dimensions. Again, we'd like to just throw this out. Some of these, I haven't mentioned. We can think of a temporal aspect of it.

Obviously, the acute pain, the different

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insults will have different lengths of injury. Although this is somewhat one-dimensional, we can obviously group them by anatomic location or tissue type. Those are something that Tim mentioned yesterday.

So whatever we decide we have, once we have those, this taxonomy, we have to apply that to see if this is valid and reliable. So the things we want to look at, at least we have thought about in the conference, is we have to address procedures that are relatively common, that may be clinically important.

Certainly, we want to look at research opportunities. I'm not sure for regulatory or other issues, we certainly want to make sure that it's valid for different age groups, different ethnic groups, and then obviously genders also. So we have to think about that as we propose the procedures.

So thinking about the potential procedures, I've listed -- and again, this is up for discussion -- 1 abdominal, 1 thoracotomy, and

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2 orthopedic procedures. Obviously, there may be more.

I chose cesarean delivery, again, this is a very common procedure, obviously all female. But you do have different types of pain, any sort of visceral-musculoskeletal pain. And surprisingly -- maybe not for most of us men, I guess, but it is very painful.

There's an article in Anesthesiology 2013 that said it's the number 9 most painful procedures out of 179, and then the question of whether there's a possible link to chronic post-surgical pain. Now, that's something that is a potentially useful procedure to look at.

Another one would be a thoracotomy, again, a relatively common procedure mostly in adults. But it is a relatively painful procedure with potential transition to chronic post-surgical pain. And obviously, men and women will undergo this procedure.

The last two are orthopedic procedures, but they're really different in some ways. One is a

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more limited knee arthroscopy ACL reconstruction; again, very limited, very common, but you would think mostly a limited -- particularly with a scope procedure, a limited duration of pain.

Obviously, both men and women have it. And then we do have both adult and children. Although the children is not -- you probably would get mostly teenagers. Again, we see more and more people, our teens playing sports. I wouldn't say they're very young children, but certainly in the teens. So that's a population we can capture.

Finally, I think spine surgery is very important to look at. This is obviously a very painful procedure. You have different types of pain: bone, muscle spasm.

These typically are very difficult to treat because they're opioid-tolerant patients. And then obviously, this connection to chronic pain is present, and we do have -- it's mostly adults, but we cover both genders, and again, this issue of long-term pain.

Now again, this is procedural pain, so we

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can think of other nonsurgical things that we might be looking at. And again, if you look at something like subcutaneous injection. Obviously, it's typically very, very self-limited. But if you think of something like vaccines, it's very, very common.

Then actually, if we look at the vaccine literature, they have quite a bit on this, on sort of pain related with vaccines. It covers both genders, obviously. We both have all age ranges from infants to geriatric patients.

Less common is lumbar puncture, but again, it's self-limited. It covers both genders and all age groups. And then finally, any type of biopsy pain, again, we can think of whether it'd be a bone marrow, a breast biopsy, depending on what we're looking at, we have both genders, again, and all age groups.

So those are the things to think about. There are certainly others, but I just propose those so that those are things we might be applying our taxonomy to later on.

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Just quickly for this Anesthesiology 2013 article -- I'll just show you -- the top obstetric, orthopedic, general surgery, neurosurgery are the most painful procedures overall. Then, if you look at the worst pain since surgery, again, it's mostly the orthopedic, the myomectomy is number 4, which was somewhat surprising to me. But again, most of these are orthopedic procedures.

Again, I tried to look at using our existing dimensions and trying to fit acute procedural/surgical pain into that and see how that might fit, proposed some surgical and procedures that we might look at later on.

Again, the discussion that we had yesterday, obviously it's not going to be very easy. But the 5 dimensions do have some merit, at least to look at, before we decide to move on to maybe a totally new taxonomy.

All right. Thank you very much.

(Applause.)

DR. TERMAN: So I'm going to assume we want to hold questions until later? Hold the questions.

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So are there any specific questions on that -- and again, not the bigger questions but -- so Chris Bernard?

DR. BERNARD: Yeah. Hi. Just a comment, actually. I think under orthopedic, as an example, I think that the bunionectomy patients, an orthopedic procedure actually is surgery. So that might be listed also, Chris, on your slide and in the paper.

Then talking about other factors, I don't know if it was number 2 or 3, in the acute situation, obviously, catastrophizing, as it is for chronic pain, certainly should be considered. But also, anxiety, because it isn't a chronic pain. It is often a new pain to patients.

So the anxiety and its relationship to acute pain should be considered I think in all of these surgical conditions, especially those that deal with trauma.

DR. WU: Yeah. I think those are two good points. I think the bunionectomy, it has been well studied in the acute pain model. Again, I was just

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limited -- I had to choose where I had two orthopedic models. But certainly, that's one that could be easily replaced with, let's say, ACL. I think the spine should probably stay.

I think you're correct. As we mentioned yesterday, there are other factors, catastrophizing anxiety. Those are things that we can insert into the dimensions if we choose to use that. Those are -- yeah.

DR. BERNARD: Just a suggestion.

DR. TERMAN: Okay. Seeing no further questions, we'll move on.

Trip Buckenmaier from The Uniformed Services Hospital and University is going to come and talk about traumatic pain, including burns.

Presentation – Trip Buckenmaier

DR. BUCKENMAIER: I appreciate this opportunity. I do go by "Trip." Chester is a way too painful name to have to go through life with.

(Laughter.)

DR. BUCKENMAIER: I feel I need to start out by apologizing because I was not present yesterday,

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and I think that was pretty obvious to this august body. I really wanted to be present because I think this is so vital for the system that I spent my career.

The sad fact of my career since 9/11 when I actually finished my training, I've really done nothing since then but clinically managed or research trauma pain in our soldiers after 15 years of conflict, if you can imagine. Yes, those wars are still going on.

But the Defense Health Agency, which after retiring, I went right back in the belly of the beast as a federal employee essentially doing the same work much to the chagrin of my spouse. But it is a group of 6-year-old children, and they will quite happily burn the building down around themselves, and then bitch at you for not stopping them.

So that's what I was doing yesterday. I was trying to prevent my colleagues from burning the building down around them. And it's a full-time job, and it was just a bad nexus.

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Sadly, I could redeem myself today, but I'm not. My boss for the last 10 years is retiring, so when I finish this, I'm going to have to go to Defense Health Agency because there's no way after of decade of work with this man that I couldn't go and honor him.

But I'm really impressed with what I've heard so far. I think this is a tremendous opportunity. And that taxonomy is desperately needed if we're going to be able to build this system.

Goals. I did try to think about this in terms of the dimensions from this article and make some suggested associations. I don't think anything that I'm necessary going to say is going to be really surprising. I'm hearing a lot of consistency and so I'm encouraged that we're going to be able to find a pathway forward.

We have what we call BLUF. When you're dealing with generals, you only get one page. So you'll go ahead and put all your slides on there, but you get one slide called the Bottom Line Up

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Front. And that's usually the only slide you get through before the general start talking. They actually get paid by the word, and so you don't really get many comments.

So trauma and burn pain are defined by mechanism of injury. I really kind of focused on this organ system approach from a trauma perspective because that's how I ordered it in my mind when we were dealing with these folks.

When I was trying to develop a plan, particularly after an acute injury in Iraq or Afghanistan, I was thinking about the systems that were involved, the bones that were broken, the skin from a burn, for example, and how I would manage the various tools in my armamentarium pharmacologically, peripheral nerve blocks, central nerve blocks and the like, to deal with this specific individual that was in front of me.

But the real issue that I thought, whether it's a hospital or a military prosecuting a battle, was a system in place to manage this patient far beyond the acute setting.

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So this idea that acute pain lasts only 3 days, or 7 days, or 2 weeks is an anathema to me because that's not what I've experienced in the last 15 years of conflict, particularly with this type of trauma.

It definitely has a psychological and emotional component. I'm of the opinion that this focus on intensity as our sole metric for measuring pain for so many decades has, essentially, in the large part, is responsible for the hole that we've dug ourselves into right now with opioids.

As an anesthesiologist out in the battle field, if intensity is my goal to manage, well really, the best drug out there is opioids. I can get your pain to zero. You'll be blowing spit bubbles and useless, but I'll have your pain to zero. And it certainly isn't doing anything about your psychological or emotional component with your pain.

What was so poignant and revolutionary about the regional anesthetic was not the impact it had on pain intensity; it was the impact on the ability

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of the patient to relate to their caregivers and relate to the system that was evacuating them, so they began to work on their recovery.

I've written about various soldiers, but one in particular, I'll tell one war story since I got 20 minutes. He's a British leftenant. He had his ankle blown off, had the education to know, "Wow. My life has really changed."

I got some morphine from the British. They still use auto-injectors, wasn't touching him. He's 10 out of 10 pain, and I looked in his eyes and I said, "Don't worry. When you wake up from this operation, you'll be pain-free."

That's pretty ballsy. But I knew that with ultrasound technology at the time in 2009, we'd place these blocks and the kid would wake up and be pain-free. It was a very busy day that day; it was a bad day for coalition. And I finished with him, put the blocks in, threw him in the recovery room and had to go back and do other cases.

When I went back, yes, his pain intensity was zero. You know what was more important though?

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When I went back to look at him, he was on the phone to his mother and said along the lines of, "Mom, they took my foot but I'm okay" -- he's talking to his mother; this is less than a few hours after his operation; his foot has been blown off -- "and I'll see you in a few weeks."

Think about that. That's the goal that I think we should be establishing for ourselves with this taxonomy and why I'm pausing on this psychological and emotional component because that's where I think a lot of the meat of this actually exists.

There are genetic, and epigenetic, and psychological factors that I am aware of but certainly don't understand. And I think we as a community don't understand and have a lot of work to do. But that can't be ignored.

Then acute trauma and burn pain appears to fit with dimensional model. At the time when I was doing this, that's how I felt. I can be swayed.

(Laughter.)

DR. BUCKENMAIER: You know, core diagnostic

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criteria -- I'm going to go through this very quick -- but I was lazy. I pulled out a dictionary and I just looked, well what's "trauma" definition?

It's an injury, so there's a tissue component. It has a psychic and behavioral state also. You can traumatize your psyche and certainly, we've demonstrated that to ourselves in our soldiers. And there's an emotional component that can't be ignored.

These themes continue to repeat for me through this. So I produce these comments again. I'm not going to belabor or read the slides to you. But there is a physical and emotional component, so those categories kept coming up for me personally.

Common features. The way I've always ordered trauma in my mind is by the mechanism. If it was a burn, a mechanism was the burn, and then I would think about the organ systems involved, in this case the skin as an organ system, and then what was going to mean to me diagnostically as far as therapy and how my therapy for pain was going to interrelate with that.

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That's a key feature of being an acute pain physician, is having a taxonomy to discuss these issues with the trauma team because our language has to mesh with the actual goal of the patients surviving this trauma, which we've been very good at by the way, less than 10 percent died of wounds rate.

But in many cases, because of these pain issues, we've been snatching defeat from the jaws of victory when we have not paid attention to pain throughout the various siloed roles of care. So while we're very good at stopping the bleeding, and mending the bones, and healing the wounds, when the patients heal, they end up with chronic pain because we weren't paying attention to the chronification of that pain as they were moving through our system.

Each silo was doing the right thing for the patient, and when I was in my silo on the battlefield, I could get your pain to zero, but then you're out of sight, out of mind, and nobody was paying attention to what was happening between

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the nodes. So you'd arrive at the next node in agony, and we start the process again.

As Wolf has pointed out to us, it doesn't matter if you preempt the pain. If you allow it to reoccur anywhere along the pathway, you get the same unwanted consequences that we've been discussing.

Dimension 3, all trauma including burns is going to have a psychological and emotional component, which we've stated again. Pre-trauma, physical and emotional conditioning will significantly influence that.

I put that on there because I think of this idea of acute and chronic pain, again, as artificial. It's very important from a system's approach to have people working in the acute space and have people working in the chronic space. But I think we need to think of it as a continuum, and that actually, there are factors that the patient shows up to at the injury, at the point of injury that will influence how they're going to respond in terms of developing chronic pain or the

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chronification of the acute pain.
I thought that it's appropriate to look at the mechanism of injury and the organ system involved as two very important categories.
From a neurobiological, psychosocial, and functional consequences -- again, I'm repeating myself because this theme continued to seem to repeat for me that a psychological and emotional response to the trauma or the burn is as important as the physical aspects of the burn itself.
The pain response to a given individual in a common trauma mechanism can vary considerably and is likely influenced by genetic, epigenetic, and psychological factors. Again, this is a big deal in the military right now where we're trying to build resiliency in the soldier.
We're actually trying to pretreat this population that we're putting into an environment where they're exposed to a situation where they will have trauma. And we feel that if they're physically fit, emotionally fit, that they'll be able to respond to their recovery and

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rehabilitation far better.
To do that sort of work, you have to have far better measuring systems than I think we have today, and that's been a big focus with the DOD, and with our own pain scale, the DVPRS and PASTOR, PROMIS, which leverages the NIH PROMIS measures and biopsychosocial outcomes.
I really like this slide, and this has had a lot of penetrance with our leadership up at DHA, about the relationship between chronic pain, PTSD, and TBI.
Unless you have a decent taxonomy and understanding of how to describe pain states, you can't really deal effectively with the PTSD and the TBI, and, boy, we've learned this in spades in the last 15 years.
Dimension 5, you've already seen this slide because Dan, as usual, preempted me, but Matt Gallagher is the primary author on this, a big mentor and great friend of mine. But this is the chronification of pain cycle. And I'm of the opinion, it's hard, but to ignore that right now

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would be a huge mistake because we already have data from 15 years of conflict that states that the things that we do on the battlefield do influence weeks, months, years later on the outcome of these soldiers.
So this chronification phenomenon is not a subjective idea to me; it's a physical reality that we're actually dealing with right now. No, the data is not pristine and it probably will not be for quite some time, but that's no reason not to start developing a lexicon to deal with this reality.
Historically, pain has been seen as a symptom, and I think we need to look at it as a disease process. From a preventive medicine standpoint, disease processes have a point where you can prevent them -- I look at acute pain as a way to do that -- and they have a point where they become chronic and they need to be managed. And I see that we have a role for chronic pain providers also.
So these are some of diagnostic groups that

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I propose from this effort. And again, I applaud this group, and I appreciate this opportunity. And I really am sorry that I'm such a jackass for not being here.
(Laughter.)
(Applause.)
DR. TERMAN: Any questions, particularly about that last remark? Yes?
MALE SPEAKER: Why is he such a jackass?
(Laughter.)
(Crosstalk.)
DR. TERMAN: Let's definitely move on.
(Laughter.)
DR. TERMAN: So our next speaker is Steve Stanos from Swedish Medical Services in Seattle at the University of Washington. We call that "the competition."
(Laughter.)
DR. TERMAN: And he's going to talk about musculoskeletal pain.
Presentation – Steven Stanos
DR. STANOS: I'm sorry, Trip. I'm thinking

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about Al Franken, "You're thoughtful, and, darn it, people like you." Remember that one?

I'm Steve Stanos. It's great to be here. I moved to Seattle a year and a half ago, and whenever I would do a presentation -- I'd been in Chicago for many years before that -- I always had to get up there -- and there was always something bad in the news about Chicago. Unfortunately, I think Speaker Hastert and the unfortunate victims he had, it's been a bad week for politicians.

But I always make comments about our governors because we had like three governors in jail at one time. We got down to one governor. So the idea was in Chicago that you're safe if there was at least one governor in jail. So I think Blagojevich is still in jail. I had a great picture of Blagojevich. That was the big, thick-haired guy. He's still in jail.

But I moved to Seattle. The news is not interesting.

(Laughter.)

DR. STANOS: I mean, they're digging

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like -- Bertha, they're digging under the viaducts, and it's three years behind. That's the big news. I'm like, "Can't you guys think of something? I need corruption, something bad to happen."

So no offense, John and everyone from Washington.

I'm going to talk about acute musculoskeletal pain. I want to make this a little more interactive. I know that sometimes could be scary. I kind of think of this as more of a physiatrist than a pain specialist because a lot of what we're talking about here, with acute pain, I think we as chronic pain physicians look at like post-operative pain, hospitalized pain from trauma.

But I want to almost shift it towards what's very common, which is under this whole realm of musculoskeletal symptoms and conditions, which is itself a very heterogeneous group.

Again, in preparing for this, like many of us, I was trying to figure how do I fit these into these different criteria and dimensions. And I think over time, a lot of the stuff that we're

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thinking about with acute musculoskeletal pain actually is this continuum to chronic pain. So hopefully, we can kind of figure out a way that we can continue with some of these dimensions and integrate those.

There's been a lot, I think, just from an epidemiologic standpoint. It's actually the decade of Bone and Joint Health from 2010 to 2020. This is actually a very good review talking about just the changes in musculoskeletal conditions to be expected even by 2040 and 2050 with the aging population.

There's so many different parts of the society that are going to be affected "musculoskeletal conditions," yet our diagnostic criteria is somewhat lacking.

These numbers go over 25 million people affected. I think work days lost, cost. We're all familiar with the National Pain Strategy and the Institute of Medicine report talking about the cost of chronic pain. A good percentage of those are related to acute pain or pain conditions, or kind

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of conditions that may repeat over time.

Musculoskeletal related pain is the most common reason for self-medication and entry into the healthcare system. It affects 1 in 4 as a leading cause of serious long-term pain and physical disability.

As a chronic pain physician, I see a lot of patients with chronic low back pain, and we think of those patients. But what about the patient with repetitive trauma with extensor tendinopathy or other types of musculoskeletal conditions?

I think what's also important is besides those working people -- and I didn't realize I was going to be speaking after Trip -- even if you look at the military, musculoskeletal conditions are the leading cause of disability discharged from service for non-battle injuries. So really, across all these different areas of work, musculoskeletal conditions are prevalent.

The key would be, though, from an acute -- maybe if I look at myself as a sports medicine physician, or I'm a primary care doctor

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doing sports medicine, or seeing acute injuries, are they even thinking about chronicity or is it just a small percentage of those patients move to the right? Most of them stay to the left. How do we do a better job of assessing those patients, and does the taxonomy reflect actually what's going on?

So when I was looking through for musculoskeletal conditions, it's been described in a number of different ways and lumped and split. Commonly, you see under musculoskeletal conditions, joint conditions, osteoporosis, spinal disorders, MSK injuries, childhood disorders. I mean, that's a huge swath of patients.

Obviously, the ACTTION taxonomy for chronic pain, now the musculoskeletal pain system has broken it down into fibromyalgia, myofacial pain, and widespread pain. Many times, that's been kind of under this rubric of musculoskeletal conditions, gout, osteoarthritis, rheumatoid arthritis and spondyloathropathies.

So is this more disease-orientated; is it more syndromal; is that really going to matter? Or

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when we look at acute pain conditions, do we have to kind of shift back towards mechanisms because this chronic process hasn't happened? Or has it happened? It's just that acute injury is actually representing two or three years of degeneration of a joint, and then you develop pain?

So is it truly an acute condition, or is it just all of a sudden now hitting that failure point where patient presents with pain even though their condition started many years ago?

So musculoskeletal, I think, is an interesting term in itself. It depends on whom and where you ask. I think in the U.S., musculoskeletal includes more of sports medicine, joint pain, osteoarthritis. Europeans maybe even present it differently.

I work with Gordon Irving. He says, muscular ske-LE-tal. And as an astute American, I'm always like, "Huh, what's that?" So I think even the Europeans and other parts of the world, musculoskeletal may be more soft tissue, maybe more regional pain versus if you asked young trainees in

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the U.S., and you said you're doing musculoskeletal medicine, that's kind of the term for sports medicine or sports and spine. Those clinicians would say, "I don't even see pain patients." Yet, probably 90 percent of the patients that presented to them have a pain condition. So even the terminology, I think, gets confusing.

I decided to maybe look at three different conditions in trying to go through the dimensions to see where they fit. Acute low back pain, many of us in this room have had low back pain and hopefully won't have another episode, or we're suffering with chronic low back pain.

We'll talk about sprains and strains, which is, I think, a very heterogeneous group even though we throw these terms many times together with our diagnosis. And then we'll touch upon knee pain.

With acute low back pain, there's different approaches. I think from an anatomic standpoint -- we're all familiar with this -- is it discogenic; is it a compressive; is it soft tissue, facet, sacroiliac?

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Unfortunately, that kind of classification doesn't always help predict our treatment outcomes. The APS/ACP, a number of years ago, we put together these little back pain guidelines. The thinking back then was low back pain, you should not do MRIs. Most of these patients are primary care level. You should just kind of sit and watch those patients.

So we've developed low back pain guidelines for that group. This was based on the Quebec Criteria. We took three of those criteria. "Nonspecific" low back pain was, well, it's just axial low back pain; don't even do an x-ray. You can sit, watch those patients, reassure them they'll get better regardless of what you do.

The second group was radiculopathy, spinal stenosis, neurologic-compromised. Maybe those patients need to get an MRI; maybe they need to see a specialist.

Then there was this third group, which is, is it organ; is it a red flag; is it something else? And the thought was there, at least, if you

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could break it down into three different parts, then the primary care doctor could at least kind of classify a patient and give them a better outcome and a better treatment. And yet, that in itself is probably lacking in quality.

If we look at low back pain itself, again, nonspecific, I mean, can you think of a worse term? "Nonspecific." It's like "I don't know" diagnosis should be a better term.

So it could be disc; it could be organ; it could be facet; it could be referred. Is it kidney? Is it SI joint? Bone? Is it a stress reaction in the younger patient that has a stress fracture? Is it ligamentous?

We have a number of different pain generators in the spine, yet, we kind of classify them as nonspecific. Or is it, God forbid, muscle? Is it I can palpate their lumbar paraspinals, but their multifidi are athrophied, or they have deeper pain in muscles I can't get to, or pelvic myofacial pain. But yet, we kind of throw this term "nonspecific."

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So there are risk factors. I've shown smoking, BMI, also tumor necrosis factor; there's disc changes, genetic factors. All these different areas have been studied. How does that correlate with our taxonomy? Different polymorphism, even motor changes, all these different things we look at kind of more from a biomedical standpoint as predictors, potentially.

So is that going to be important in our taxonomy, understanding that?

What I want to touch upon just briefly is the shift in low back assessment. There's a lot in the physical therapy world in subclassifying, in risk stratifying patients with regards to treatment.

So what you do with that is you target the treatment towards the factors that are modifiable. They look at leg pain, widespread symptoms, those as prognostic factors, but also psychosocial factors. So again, I think this may be something -- I'm not going to say we're going to change our whole taxonomy, but there's been a lot

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of research in this area, and what they'd been able to show is, if you can better subclassify these patients with "nonspecific" low back pain, you get better outcomes.

So maybe our taxonomy should have that understanding that if we can subclassify patients better, that's going to be an important taxonomy and be useful.

So how they do it is they'd say classifying patients in groups based on clinical characteristics and matching these patients' subgroups to management strategies will benefit them and improve their outcome. We'll just kind of go through this briefly.

How many of you heard of McKenzie therapy? No? Okay. So McKenzie therapy, the idea is that you've got an intact nucleus pulposus, but the disc is causing pain. And maybe with certain movement patterns, it's going to change the movement of the disc, and that may change your symptoms.

So with McKenzie therapy, they do certain movements, repeated movements, to centralize the

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pain. So I want you guys to slide your chair back a little bit. Slide your back -- come on guys. Movement is memory.

So if you do flexion -- and I don't want you guys to take your clothes off and get on the ground with your --

(Laughter.)

DR. STANOS: I mean, if you want to do that, hey, what the heck. So if we do it -- our friend here is doing it in his -- I guess those are black tighty-whities. But you can do it seated.

Spread your knees out a little bit, and I want you flex forward 5 times. So you came in. You've got right leg pain going down your thigh in your right calf. How do you do those maneuvers? So the therapist does it, and your leg pain, look what happens. Oh, his underwear changed colors there.

(Laughter.)

DR. STANOS: His leg pain gets worse. You're peripheralizing. That's not good. Now, you can change your hair color and change your

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underwear.
Now, everyone, stand up for a second. Now, McKenzie is repeated movement, so it's mostly extension. Most patients like extension. So kind of lean back, extend about 5 or 6 times in a row. If you ever see a guy doing this in the airport, he's doing his McKenzie exercises. And that should, in patients that respond, centralize their symptoms.
So then that therapist would give you extension-based exercise; 80, 90 percent of patients that respond to this therapy, you do extension. But there's some that do side bending. There's all sorts of different ones.
But the key is, though, that they're specifying based on the exam what your treatment is going to be.
You guys just moved. Isn't that good? A little movement is better, right?
(Audience participation.)
MALE SPEAKER: Can we do that (inaudible)?
DR. STANOS: Oh, you want to go --

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(Laughter.)
DR. STANOS: Let's not go there. Okay.
So again, so that's just an idea of just that type of treatment, and it's based on how you respond to care. And they've been able to show a number of studies, with the subclassification improve outcomes. So that's just one way.
If you look at the ICD-10 codes, it kind of alludes to this because it talks, I think, a little better detail than ICD-9 about diagnosis. We have acute, subacute low back pain with mobility deficits; acute low back pain, the second one, with movement and coordination impairments; the third is with referred or lower extremity pain, referred pain being kind of -- referred like an SI joint or facet referral pattern versus -- radicular pain is more nerve root irritation, radiculopathy.
So again, this taxonomy -- I mean, would the taxonomy be able to more clearly describe what's actually going on?
This is from the ICF classification. And again, just to give us an idea of things that we

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could be looking at, if you look at the first, it says "acute low back pain with referred lower limb pain." The symptoms are low back pain associated with referred buttock and thigh, so it's like sclerotomal referral. So like an SI joint has a referral pattern; your facet joint has a referral pattern; your disc has a referral pattern.
So those patients, their impairment is for low back pain, lower extremity pain caused by -- it can be centralized. You would give them exercises. You may give them traction. You may give them repeated centralization exercises. So that patient with that referral pattern would get that type of treatment.
The other diagnosis, acute low back pain with radiating pain, that may be more radiculopathy, so they have neural tension. You guys have a disc herniation, and S1 leg symptoms on the right. I put you on the table; I raise your leg up; I can reproduce your symptoms. So we're aggravating your nerve root; we're reproducing your symptoms.

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So those patients may go to a therapist and get nerve glides where we loosen up the nerve to make it less sensitive, and we show them different exercises.
So the key is not getting stuck on what the therapist is doing here but the idea that we're doing a better job with our taxonomy and figuring out what the actual treatment is going to be.
This is just from different stories. And Fritz has done a lot of this work, but looking with treatment-based classification with mobilization, a patient has mobilization problem, they may have SI joint or lumbar pattern; if they have specific exercises they respond to, or they may have a flexion pattern or extension pattern like you guys just demonstrated; again, the point here being thinking of low back pain a different way versus this is just disc.
I think all of us suffer with our patients with reading these 7-page MRI reports with all of these abnormalities, which really don't mean anything, or maybe one or two parts of that MRI

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actually mean something.
I'm just putting this up here, the STarT Back Screening Tool -- are you familiar with STarT Back? STarT Back has been used in the physical therapy and literature. We're using it at our center to help better predict, as a screening tool, bad outcomes.
What STarT Back does is the first questions are: Do you have back pain spreading down your leg? I have pain in the shoulder and neck. So you're trying to pick up do they have pain in other areas of the body? In the last two weeks, have you dressed more slowly? And then the rest of the questions kind of deal with catastrophizing anxiety.
Then you score that. And then from that, we're able to score and give a composite of what's their risk for chronicity, and then does that also pick up patients that may need different treatments?
So if you have a higher STarT Back Screening Tool, maybe there's underlying psychological

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factors, catastrophizing. They would go in a different way of a pathway or different treatment approach; the key being those with risk factors for chronicity, those need to be considered in our taxonomy.
This is just from Swedish -- we're putting together a low back pain pathway. The patients present -- I know you can't read this. We rule out that they have radicular pain or red flags based on their STarT Back Screening. If it's a low score, maybe they need education, reassurance. They don't need a lot of imaging; they can stay at the primary care level. Those patients with higher risk maybe need to be sent to a physiatrist or a pain management specialist or need behavioral interventions early on.
So the key is, though, that within that, we still need a better kind of diagnostic taxonomy to look at these patients. We're still calling them low back with leg pain. Is there something else we can better identify those patients once they're stratified? So would a good taxonomy be able to

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pick that up?
I'll talk about sprains and strains. This is a very different group. Sprains is more ligamentous versus strains is muscular. So that's like saying sprains and strains. That could be saying I'm going to treat chest pain and shoulder pain the same because they're in the same area of the body, yet completely different.
So even within strains or muscles, is it myofacial; is it delayed onset muscle soreness? How many of you guys have ever tried to work out? You go work out, and you're sore for three days? That's a great example of hyperalgesia of your soft tissues. You rip some of those smaller muscles; you get pain; you're kind of sore walking around; you think you're a lot better, and then it goes away. Delayed onset muscle soreness has been well studied. It's a muscle hyperalgesic response, but then we recover.
Looking at just sprains itself, sprains are the supporting structures of a joint. It's a tear of the ligament or capsule versus a strain is in

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the muscle or the myotendinous unit, so very different even though they get lumped together.
Signs for a sprain can be tenderness, swelling, weight-bearing pain. Symptoms can be pain with weight bearing. A strain is more focal tender muscle tenderness. There can be hematoma if there's a significant rip or a tear depending on what part of the muscle belly is affected.
Then there's a complete rupture. Many of us see patients with biceps ruptures. They have no pain. They tear their bicep. Now, they have pain when they get their surgery done; it doesn't go well. But with a full rupture, there's actually no pain.
So common features, if we look at just sprain, there's injury to a ligament as a result of an excessive load. Sprains are more common in older adolescents, young adults, and middle-aged adults. So across different timelines, there's different characteristics.
In children, the physis is the weak link, so many times, we'll see -- that's why we have

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limitations in how many curve balls an 8-year-old can pitch because their physis hasn't closed, and they can get injuries because the muscles are pulling on the tendon and that's close to the physis.

Older adults, the bone is weak, and they can have falls. So with osteoporosis, a simple fall can cause a fracture. You can have sudden trauma inversion injuries. A lot of our ankle injuries are ligamentous tears related to sudden trauma.

The diagnosis may be negative x-rays but a positive ultrasound and maybe changes on the MRI. And what I'm seeing as a physiatrist is all these guys doing ultrasounds. I mean, most of our patients get sent to us, they don't have an MRI done; they have an ultrasound done, and you see four paragraphs of this beautifully written ultrasound report.

So we're actually seeing these and picking up on a lot of these intricacies a lot sooner. I think, in some ways, we're doing a better job of managing them. But again, I think the black box of

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these tendinous injuries are a lot better. We actually can get a better understanding of the biomedical problems or biomedical changes.

But these are the classification systems for sprains: Grade 1, 2 and 3. Unfortunately, without ultrasound, that's hard to pick up. And a lot of times from a physical exam standpoint, it may be difficult in what's the difference between a grade 1 and a 2 and are you able to pick that up on physical exam? Does this classification help us?

So if we look at common features for strains, again, that's the muscle tendon insertion many times, but it also can be in the middle of the muscle belly. It can incur with aging. And remember, there's changes in the collagen and the muscle tendon itself. So that muscle tendon junction may initially have an injury and there's inflammation.

Chronic degeneration, the myofibrils may actually line up and there's collagen that heals correctly. But you can get chronic degeneration, and that may go from a tendonitis to a

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tendinopathy. So even within this myotendinous injury, there's a whole different cascade of events.

This just shows with muscles strains, again, trying to break down from a classification standpoint, is it interstitial strain all the way up to complete rupture. And sometimes you may be able to differentiate based on the physical exam findings. You may be able to feel actually a defect in the tissue. But again, very challenging from a diagnostic standpoint.

Just from Dimension 3, if we look at sprains and strains, what are the risks? There could be high exposure to game conditions. So a lot of football players, you'll hear about strain. They're out for three weeks with a hamstring strain. These guys are very strong. They're in good shape, but those accelerated forces can cause injury; or it could be a low back pain dysfunction, poor endurance, core muscle strength. You can have reduced extensor muscle endurance, and that could be a risk factor as well.

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The last group I'm just going to talk about briefly is osteoarthritis, and this is just more from the OARSI classification, and they're actually developing an OA taxonomy, a newer taxonomy. But they discuss the importance of disease versus illness with osteoarthritis.

Early on, the disease is actually all those molecular changes in the joint itself, and then you lead to this kind of clinical point where you report pain, and is that the actual illness, even though we're calling it an acute presentation.

So if we think osteoarthritis, there's this kind of balance between the catabolic breakdown in the joint with the balance of the anabolic repair of the joint. And over time, what we see is catabolism kind of wins out.

Then we get changes in the joint itself, the synovium, the cartilage, and the bone. A lot of the genetic variability within patients with osteoarthritis is in this ability and inability to catabolize and to reform tissues. And now, within the osteoarthritis literature, they are able to

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look at phenotypes, tissue changes to kind of actually better assess those levels of changes in the joint.

What they talk about with their taxonomy is, do we have the disease elements, the molecular changes, followed by the anatomic and physiologic changes, and then you reach this tipping point where they present with pain, or dysfunction, or illness? And they've just developed a composite score that kind of includes those different factors, and is that another thing to consider?

Some of these conditions that have biomedical differences, can you develop some type of composite score within that, versus saying all of our MSK conditions, we're going to use this criteria, or are there certain MSK conditions that could use kind of a marker looking at this composite or variability from patient to patient because it's so complex.

This is just kind of looking at Dimension 4, ongoing cellular distress, again, like we talked about in the joint itself, anatomic changes, those

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could be neurobiologic consequences, sleep problems, the kinetic chain consideration.

So everyone, stand up for a second. Everyone, stand up. So we've all developed -- we're 70 years old. We develop spinal stenosis, so we have to bend forward when we walk so we can limit our neurovascular claudication, so we can get to the airport and walk well.

So instead of us walking around bent forward, what do we do? We bend our knees, so bend your knees and your hips, and we stand around like that.

Now, I'm going to do the rest of my lecture, and I want you guys to stay like that. Okay. What we're going to talk about is the kinetic chain. The kinetic chain concept is -- no, bend a little bit more. This is keeping all the orthopedic surgeons in business.

So what happens in about five years or about five minutes from standing like that? Do your knees hurt? You're starting to feel a tension in your knees, right? Okay.

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So most of our patients that have spinal stenosis also develop hip arthritis and knee arthritis. For many years, they're walking around with their knees slightly flexed and their hips flexed. So the kinetic chain idea is that you have knee pain you're presenting with, but you also have hip flexion tightness, and you also have core weakness.

So how is that assessed as part of a taxonomy? Because that could be a risk factor as well. You guys all look like you're squatting, I won't say doing what, but that's good.

So just as the concept, for acute pain, we've got to be thinking about not just the joint but the other joints around it, which is also not really considered in a lot of these criteria because that might be a bigger risk factor.

You guys can sit down. Sorry. No one developed back pain or knee pain, right?

Then the other consequences or functional consequences is lost work days, activity limitation, participation restrictions. Do we need

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to be a little more specific about their function?

I mean, I'm a physiatrist. We all love to talk about function. Function is hard to assess. So do we need to do a better job? Is it your activity limitations? Is it your participation? Do we need to do a better job of breaking that down?

Almost done. Sorry.

DR. STANOS: Finally, for Dimension 5 is the risk factor for OA. Also, what have been studied are obesity, knee trauma, meniscectomy, significant literature showing the accelerated degeneration of a joint just after a meniscectomy. Was it the meniscectomy that did it or is it just are you being marked?

I think the other area, which is important, is this whole -- the damaged cartilage, there's all these interventions now at trying to improve or decrease the catabolism of the joints. So we have physicians doing a lot of regenerative medicine, biologic therapies. How do we consider that within our taxonomy?

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We can't forget with Dimension 5, catastrophizing anxiety, depression. But on the other side, what are protective factors? Is there a strong social and work environment? So a patient presents in the ER with acute pain from a joint problem, but they've got a good working environment. They actually want to go back to their job.

We actually see the opposite with the guy that has problems, doesn't want to go back to his job. But are those potentially protective factors? Or do we go totally biomedical and say it's their joint phenotype? Maybe they've got a better chance of recovering in a joint injury than the second person you're going to see that day, just on their phenotype of their joint.

The last one here is looking at the putative under biologic, psychosocial mechanism, and risk factors. So I think what's important, too, is occupational factors. Again, is the patient a floor-layer versus a sedentary job? Are they lifting and kneeling? Are they squatting? You

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know, a lot more detail on what the actual aggravating factors are related to that, quote/unquote, "acute pain" injury.

Is there anger, perceived injustice? I always think with chronic pain patients, many times that could be the real barrier for them getting better. But they have that perceived injustice when they first got injured. Again, just to kind of throw out other psychological variables that could potentially impact patients with acute musculoskeletal pain developing chronic pain.

The last thing I'm going to say is the individual -- remember, we all know red flags. Yellow flags are those things that predict kind of bad outcomes. But within yellow flags, there's workplace conditions or black flags, which is the workplace is unsafe, I don't want to go back to that lousy job; or the workplace factors, it's too much lifting, too much bending.

Again, are those other things we need to be looking at within a taxonomy or at least within the assessment?

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Acute MSK pain taxonomy may need to shift back or in some areas, looking at the mechanisms. We're not going to ignore the biopsychosocial context in the syndromal approach, but where do we find that balance?

With low back pains, again, is the taxonomy based on treatment-based classification? What we did with extension and flexion and movement, is that something we should be assessing more to help better understand or breakdown in the taxonomy where those patients lie?

Is ICD-10 a better version of what we had before? Can we use that within our taxonomy? And again, also thinking, too, with a lot of these musculoskeletal conditions, what's the underlying disease that led to this, quote/unqote, "acute" incident? There's a lot of things going on in the joint, in the muscle, in the tendon before they presented with pain. And is that important? And remember the kinetic chain, right?

Okay. One last thing. For tendinopathy, you guys use a mouse. Just get your wrist, flex,

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extend your wrist a couple of times, radioulnar deviate. And pretend like you use your mouse all day doing that. Do you guys want to keep doing this all day? Probably not, right?

Now, I want you to put your hand on the table like you're using your mouse. Instead of moving your hand back and forth like this, use your scapula to move your arm.

That's another kind of kinetic chain concept, how do we move a limb higher up more proximally to affect what's going on in our wrists. So on our assessment, do we have to be looking at not just the wrist but higher up in the shoulder?

I'm just kind of throwing these out there, again, thinking more from a sports medicine approach with these acute conditions. That's going to be a key with the assessment.

Again, where does that lie? Or we can do a better job with the taxonomy and picking up acute localized pain, which would prevent this, and is subclassifying these patients going to do a better job to keep patients from going from left to right

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and keeping them hopefully towards the middle or to the left. I didn't mean that politically. Sorry.
(Laughter.)
DR. STANOS: Thank you. You guys all survived. Thank you very much.
(Applause.)
MALE SPEAKER: Steve, quick question. You got time? That was a very good talk. Thank you.
Could you comment or would it be a good time to comment maybe about -- you've got the screening tools and outcome psychological/psychosocial factors. Do you screen for, do you have data, and is it helpful in your field?
DR. STANOS: I'm just thinking in our own system, we're doing anxiety depression scales in questionnaires already. The STarT Back is just one that we're using for low back pain.
Within that, if they're high on their STarT Back -- I don't think STarT Back is going to cure all of our ills, but if there are high scorers on that screen, then that clues the primary care provider or even our physical therapists.

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A lot of patients see the physical therapist before the even see the physician. They're doing the screening tool, and then that clues them in. If they have a higher score, do they need to do other screens or do they need to send them to primary care or a specialist?
So I think that's just one of the tools that we're using. But most of the patients are still using other questionnaires as well, if that answers your question.
MALE SPEAKER: Do you have literature yet or is this early --
DR. STANOS: No. We're actually starting the low back pain pathway at one hospital system, and then the idea is we're going to using that across our system. But the STarT Back has been used in -- I think Intermountain has been using STarT Back for a long time within their clinical pathways and other areas in the country.
But it's interesting, a lot of this came out of the PT literature, and now it's kind of finding its way into the MSK and the kind of medical world.

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MALE SPEAKER: [Inaudible - off mic].
DR. STANOS: Yeah. Oh, yeah.
MALE SPEAKER: [Inaudible - off mic].
DR. STANOS: And what's interesting -- I mean, again, yes, we're still talking about nonspecific low back pain, but we have a subset of people that are doing a lot better work in identifying these patients, and subclassifying them, and I think getting better outcomes.
With that being said, we had a guy that tested high in his STarT Back tool, and they did it over the phone. So one of our MSK clinicians referred him to the pain clinic because that's where they're supposed to see us.
We saw the guy. He probably has somatoform disorder. He had been to the ER like 4 times in 4 months. He had eye pain from looking in the microscope; I mean, everything. But at least the screening tool alerted us to that. He was not open to what we want to do in our pain clinic, but at least we were able to identify him.
He's still causing a lot of strife, but it

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was interesting. That little STarT Back thing at least tripped our primary care -- or musculoskeletal physician to be like, wait; you need to go to the pain center. We have behavioral health in our pain center. Can they assess this guy? So it was kind of funny.
Now, is that going to help our hospital system save money? I mean, I think we've identified that this guy has a lot of psychologic and psychiatric issues. But it was just funny how the screening tool in a sense worked, but then what do you do with it?
DR. SURESH: Steve, I have question for you. Thank you for this excellent lecture.
So my patient population is all kids. The big problem that we have is there's a lot of sports injuries. About 50 percent of the patients that we see are generally sports injury patients.
The big problem that we see is there is a chronicity to these acute injuries because they are treated by their pediatrician or somebody -- the school nurse treats these guys.

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It's really difficult to draw the line between the acute and the chronicity of MSK injuries. How can you come up with this division?

DR. STANOS: Well, I think you bring up a good point because most of those -- like those patients you're seeing, the acute, they have multiple acute episodes where they're getting a recurrent kind of return of their back pain with sports.

So yeah, I do think we have to consider -- I think most of these are subacute or repeated acute conditions. I'm having flashbacks. In our chronic pain program, we had adolescents. And they all started out with the musculoskeletal condition, and then it kind of progressed.

So I think they have the same kind of sensitization, had the same factors and issues with their parents and all of the other kind of issues that kind of turned them into a chronic pain patient. But yeah, I think many of those aren't really acute injuries; they're repeated injuries that aren't really properly assessed or treated.

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DR. TERMAN: Thanks a lot.

Our next speaker will be Mike Kent from Walter Reed. He's going to talk about acute visceral pain.

Presentation – Mike Kent

DR. KENT: Good morning. I'm Mike Kent. I'm an active duty Navy anesthesiologist at Walter Reed. I trained under the good Dr. Buckenmaier.

I kind of drew the acute visceral pain straw, and what I did is, actually, I took the dimensions that we started to rework yesterday, and I had a bourbon in the bar and tried to rework everything towards those dimensions.

So full disclosure, it gets a little messy, but I use it kind of as a tool to more in the chalk talk, like can we talk about the dimensions? Let me take my topic and try to fit it into what we talked about yesterday afternoon.

Full disclosure, I think when I line the dimensions up from what we talked about yesterday, I think they're actually pretty similar to what it was before, and we'll go through that and go from

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there.

Just like some complaints like acute musculoskeletal pain, I think acute visceral pain kind of falls in a category unlike, oh, a patient has acute pain in front of me. I know what I'm looking at.

Well, that's kind of the opposite with acute visceral pain, where hundreds and hundreds of diagnoses can lead to any type of acute visceral pain depending on actually where you're talking about it.

Also, we already have numerous diagrams, and numerous surgical text, or pain text, or internal medicine text. I've actually had to approach algorithmically numerous types of visceral pain. And when I was initially trying to fit these into the original dimensions, I was really trying to uncouple in my mind classifying or diagnosing visceral right upper quadrant pain with the actual process of making the diagnosis of acute cholangitis.

The two aren't necessarily completely

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mutually exclusive, especially in the prior dimensional concept. I'll skip over this. But before we even get to kind of the dimension part, is considering what sources of acute visceral pain do we even consider in the first place.

In the chronic pain taxonomy, when we think about intracranial, do we want to consider that as a group, as a source of visceral pain? Is it deep somatic; is it visceral? I mean, we already have a huge classification system for headache, but I think whoever ends up with this working group, at least, has to pay some credence to acute events that need to be diagnosed on the spot that may present with an intracranial visceral process.

Then the thoracic, the pelvic, abdominal components, I think, are rather common, rather universally understood as having etiologies for visceral pain, but nonetheless something to think about as we move on.

This is kind of how I initially framed it with the initial dimensions that came up with the chronic pain taxonomy. And I actually picked the

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pyramidal concept on purpose where Dimension 1 had more broad strokes, key attributes narrowing down to Dimension 2, what kind of diagnostic test, physical exam, lab tests were associated with it. And right about that time, you would usually come to a diagnosis.

Then moving on from that, what are common comorbidities associated with that diagnosis, let's say myocardial ischemia, biological, psychosocial risk factors with that, and then you get down to the nitty-gritty mechanisms of whatever acute pain diagnosis you come up with, but it's exquisitely linked to the medical diagnosis at the same time.

I thought the pyramidal concept really shows, especially with acute visceral pain, it's very tough to separate the process of diagnosing myocardial ischemia with classifying acute visceral chest pain. So a lot of the processes are in parallel; they crosstalk, but a consideration with fitting these in the original dimensions.

Here, this is kind of my own summary of what we quasi came up with yesterday. And I put numbers

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by it, but I should have just put letters, X, Y, Z, A, B, C because I don't want to denote any priority in terms of 1, 2, 3, 4, 5, 6.

But I also put this table up here, kind of my own reorganization of what we came up with yesterday compared to the chronic side. And I lined them up a little bit, and I don't think they're terribly different.

Now, the fine text is going to probably be very different or a lot more contributory in terms of the acute side, but this is how I'm going to try to fit it with acute visceral pain in terms of what we came up with yesterday.

So I stole this slide from Pat, and he said I actually had better colors, which was great. But one of the things that I'm still stuck on -- and I'm moving into Dimension 1. The Dimension 1 concept we talked about yesterday is what's the event; what's the tissue injury; what's the extent; maybe considering the organ system.

I'm still struggling with "event." I still think we need to work on this classification or

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semantically how we want to handle event first before we move into the dimensions. I'll go into this in a second.

So referencing Dave and Rob in the back of the room who gave me this idea at the end of the day yesterday, for acute visceral pain, I'm just going to grossly categorize these as a medical event, a surgical event, or a traumatic event. I'm not saying that's right. I'm just throwing that out there to try to fit it in, make it dirty, make the discussion move forward.

So a medical event such as cholangitis; a surgical event just like visceral post-hysterectomy pain; traumatic event, visceral pain associated with a hemorrhagic headache, associated with some form of trauma.

Now, in the realm of acute visceral pain, though, the degree of tissue injury does not correlate with the severity of pain, as we all know. Moving on, though, delving a little deeper into this Dimension 1 of event or tissue injury, what is the event? Is cholecystitis the event or

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is gall bladder inflammation swelling the event? Yes, that is cholecystitis, but cholecystitis is the diagnosis associated with alk-phos, imaging studies, physical exam, et cetera, et cetera.

Or is it a medical event resulting in a disorder process of the hepatobiliary system triggering a nociception in the acute setting? Is that more of a broad stroke that we need to think about either in Dimension 1 or just a step before Dimension 1, specifically for acute visceral pain? Up for discussion.

So where are we? Coming back to this, is this something that we at least maybe should consider before moving on to Dimension 1, a broad stroke, before moving on to the core kind of concept of what we're starting with semantically in Dimension 1, and my topic specifically with acute visceral pain?

I made this slide when I was fitting it to the initial chronic pain dimensions, where you had differential diagnostic considerations. In a broader stroke there, we're thinking -- and I'm

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just talking about abdominal visceral pain, right upper quadrant, central visceral pain, a long list of diagnosis crossing numerous physiologic symptoms: GI, cardiopulmonary, hepatobiliary.

But coming down, if we conceptualize the event, or tissue type, or organ system in a taxonomy we want to approach acute pain with, at least for visceral pain, it makes it a little cleaner.

So if we call the event an acute urological infectious event, it makes our differential diagnosis, if we actually choose to put this in Dimension 1, a little cleaner: cystitis, prostatitis, epididymitis, all probably what we would consider medical events, but at least it's a little cleaner than a broad stroke right upper-quadrant central type of phenomenon.

So again, I come back to this comparison, moving on from what we talked about Dimension 1 yesterday and moving on to let's talk about Dimension 2. And we talked about pain quality, temporal characteristics, spatial, and I put

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characteristics there as well. Definitely, we'll have to hone that in.

I put Dimension 1/2 here because there may be some core criteria to think about for acute visceral pain but some supplemental criteria that can sift down more into a common features concept.

When you really think about visceral pain, IASP put out a good summary, and there isn't a ton on acute visceral pain that's actually very well delineated with comparisons, but think about the definition -- at least according to IASP -- of true visceral pain, it's vague pain that's in the midline, and that's about it. And the rest of it, referral patterns which we'll get to in a second, can either be lumped into this key diagnostic criteria or can be considered associative.

The pain duration and the temporal characteristics, I think, is critical in the acute visceral setting on numerous fronts. One is the duration, because this is where you get a lot of crosstalk between acute, subacute, and chronic visceral conditions.

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Are we talking about acute, subacute, subacute recurrent, chronic recurrent, chronic recurrent? And what's the line that we draw in this in terms of this taxonomy to choose those conditions to talk about and classify as well as provide diagnostic assistance?

I use the diagnosis and classification terminology there on purpose, a line that needs to be discussed. And numerous conditions can point to this.

So duration, but also the pattern of acute visceral pain, I think, in terms of a temporal nature is essential to think about. Is it something like nephrolithiasis? And we'll just take the example of a stone that's passable. Huge, horrible event, coming to the ER, the stone has passed, pain resolved. There may be some evidence of persistent hyperalgesia, depending on how long the stone was, but nonetheless an acute event that went away.

But what about GERD? Well, is GERD acute? Is GERD a subacute? If I had GERD symptoms after

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2 days, versus 4 days, versus 5 days, I go to the ER. I get some Zantac, it goes away, come back 5 days later. Okay, we're kind of in that subacute realm, but I get a PPI; then it goes away. Is that still part of our taxonomy? It's subacute, but it started as acute.

So these are some of the challenges, I think, with drawing lines with visceral pain. But this is a nice graph when you start thinking about patterns of how we describe acute visceral pain and how temporal qualities, I think, are so essential is that if you take line A, that's kind of when your aorta ruptures, huge onset, big abdominal visceral pain that needs to get taken care of right away.

Rapid onset, not as rapid as A; B, being more of a pancreatitic-type pattern; C, which kind of just kind of a slow undulating pain, more of a choledocholithiasis; or E, kind of an insidious onset pain over 1, 2, 3 days. Appendicitis usually may fit into that.

So again, all these we would probably

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consider acute, but all have very distinct -- not universal but distinctive temporal characteristics in terms of their onset. And we haven't even got to the resolution part yet.

So staying in this Dimension 2 type concept here, at least now providing a comparison to pure somatic pain and how this acute visceral pain is at least described. Well, in vague sense -- oh, I guess in visceral sense, it's described as having more of a strong effective or autonomic response. But again, this isn't universal. This is mostly seen as commentary versus actually quantified, but nonetheless quite commonly referred to.

Referral, and I think this is something that can either fit in a core diagnostic criteria or may be added in terms of a supplemental sense, where visceral pain may be referred to a neurally-linked organ without hyperalgesia, where on your exam, you cause no more increased pain on exam, or you have referred pain with hyperalgesia, very tough to differentiate from parietal information if we're talking about the abdominal sense, or if it's just

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true hyperalgesia with no somatic involvement of parietal peritoneum as an example.

So it's something that I think probably fits well into this Dimension 2; definitely can be up for debate for fitting in terms of a diagnostic core criteria depending on how we actually end up on defining event.

Associated symptoms, of course, urologic symptoms, hematuria, hematochezia. Now, these aren't really associated symptoms of the visceral -- Or they're associated with the visceral pain, but one argument, again, these parallel processes of making the diagnosis versus classifying the pain, hematuria is probably more associated with the diagnosis of, whatever, versus actually the pain. But it's in the globe; it's in the world of it.

Physical exam findings, again, there really is no pathognomonic physical exam finding where you can just walk into the ER bay, touch the patient, and then leave with their hands in the air screaming victory, and then you're done; but

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definitely many associated physical exam findings that are associated with a presentation of visceral pain and all the things we've learned in medical school and beyond, but suggestive in nature, and I don't believe necessary in a core diagnostic concept.

Observational components, again, also associated with diagnosis but associated with the presentation of the pain and speaks to maybe the magnitude of the pain in terms of an autonomic response or effective response, pallor, vital signs, rebound, things like that.

But again, going back, physical exam, in terms of specifically focusing on acute hyperalgesia, it may give you some sense of how much hyperalgesia has set in or not set in as related to your acute visceral pain event.

This comes from the original dimensions where epidemiology was considered in Dimension 2. I offer this as more of a supplemental characteristic of whatever the -- at this point, you've already come to what's causing your visceral

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pain.

You can approach it on numerous fronts such as location. You know, in a series out of the UK, it's their tenth highest reason for secondary admits; chest pain, 20 percent prevalence in certain populations.

Or you can take it based on diagnosis if you're already come to your diagnosis of visceral pain, 20 percent prevalence, or appendicitis and add some other supplemental characteristics into this epidemiologic framework, but may also serve as a modifying factor down the road such as a 95 percent incidence of pain in young populations but in elderly population, only 55 percent of people actually report of pain in the right lower quadrant; or gender concepts or age concepts, as I just mentioned.

Laboratory finding. This was also mentioned in the initial chronic pain dimensions. And again, this speaks to this parallel process of how appropriate this is in terms of bringing it in to classifying acute visceral pain versus diagnosing

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visceral pain in the setting of its underlying pathophysiological medical or surgical diagnosis.

Elevated amylase and lipase, is that associated with the acute visceral pain that's caused by pancreatitis? It's associated with it. It's associated with the diagnosis, but is it truly associated with the magnitude of the pain? I don't believe that's true.

High prevalence of negative findings, I think any ER physician will speak to this, of how many times someone's come in with abdominal visceral pain, eating Doritos, asking for Dilaudid. And they have a negative work-up even though they may have complained of classic symptoms.

But again, numerous etiologies or numerous diagnosis have very specific laboratory abnormalities associated with them that is directly linked to their acute visceral pain complaint. And again, I'm mostly focusing on thoracic, abdominal, pelvic. I think intracranial is a discussion to be had in the future.

What about Dimension 3 from what we came up

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yesterday? Risk factors, host, patient, internal. In the original set, this was comorbid diseases. Well, we talked about -- Dan mentioned "host" so I group these under "host."

So comorbid conditions are risk factors. So if we think on an organic level, we take myocardial ischemia, well, we have risk factors for that. We have comorbid conditions with that: smoking, hypertension, hyperlipidemia.

What about bowel obstruction as the etiology for visceral pain for numerous associations or risk factors for that, whether it's tumors, stricture, inflammatory conditions, ulcerative colitis, Crohn's.

Functional etiology. Every case of irritable bowel syndrome presents probably with an acute -- or sometimes form, presents to their physician's office with maybe at least some form of acute symptoms.

If you take the percentages, 20 percent of all IBS has presented with an acute infective event that has served as the onset of their symptoms, if

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you look at the epidemiologic tables. But definitely psych linkages as everyone knows in this room in terms of comorbid conditions linked with those; that part's obvious.

But in terms of this Dimension 3, risk factors, host factors, so any host factors or any incidences in terms of the experience of acute pain relating to chronicity risk, and is this appropriate here in this dimension I think is up for conversation.

But we know for certain things, the level of acute visceral pain prior to cholecystectomy is a predictor for chronic pain after cholecystectomy. So the process of how visceral pain is processed by the host may definitely serve as a characteristic to place in this dimension in terms of taxonomizing risk in the realm of acute visceral pain.

Protective factors, I kind of use the term "modulating factors" as well, and I think this can reside in the biopsychosocial milieu or biopsychosocial characterization of the host in terms of how bad the initial presentation is going

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to be and also speak to possible trajectories of resolution and risk for chronicity, so may apply in this.

Dimension 4. This was initially probably Dimension 5 in the chronic pain taxonomy, but mechanisms and pathophysiology. Definitely, long treatises have been written about mechanisms of visceral acute pain both in the acute and chronic setting; diffuse receptive fields, silent nociceptors, threshold-based nociceptors, but still without a clear link of how that actually is completely processed to the external realm in terms of the pain experience.

As Dr. Brennan mentioned yesterday, I think much has been written on this, but the clear delineation from point A to point Z still has a lot of work to do. And it's very dependent on the diagnosis, what's the organ, how is the organ innervated, where is the organ. And so a lot of room for work here but apropos; apropos for a mechanistic type of dimension.

Dimension 5, impact and functional

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consequences. Again, I think this -- and I use the word "event-dependent," so it may not just be diagnosis-dependent but also event-dependent in terms of considering it in a consideration.

So I go back to nephrolithiasis, at least that one, which a stone can be passed in the acute setting, an acute event where you want to die and then it goes away, and you want to live again; or a small bowel obstruction, which may have -- just by the diagnosis itself, it's an acute event with a more of high likelihood of impaired function in the immediate setting: NPO, possibly surgery, NG tube.

That's associated with the pain, associated with visceral pain, but it's probably the diagnosis, the burden of the diagnosis in this setting, instead of the burden of the visceral pain that describes dysfunctional impairment.

Psychosocial and functional concepts, this kind of goes back to the conundrum of acute visceral pain where the magnitude of injury doesn't correlate with the magnitude of the pain felt, and a full spectrum of having a fully functional

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patient or a vegetative patient lying on the floor with gas pain is a possibility; but nonetheless, I think apropos for this dimension as we started to fit in. Again, messy but I did my best to try to fit it.

Dimension 6, my bar tab was up at this point.

(Laughter.)

DR. KENT: I didn't get a whole lot of time to think about it. This is the one I struggled with the most in terms of milieu, and I'm still working on it in my mind. I hope that we can have some good discussion about it this afternoon.

But going back to kind of lining up what we started to come up with yesterday with the chronic pain taxonomy, I thought it was very reassuring that there's a lot of crosstalk.

I think the devil is in the details in terms of, one, how are we going to define event and all the fine print of how we want to finally detail these in the acute setting versus how they were finely detailed in the chronic setting.

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So I hope it wasn't too messy for acute visceral pain. Thank you very much.

(Applause.)

DR. TERMAN: So I might take a little bit of time for maybe a few more questions if there are any here on this bourbon-fueled presentation because this is clearly -- rather than just moving on, this attempted integration is really useful.

So if there are questions?

FEMALE SPEAKER: I just wanted to say that was great, and I think that it's nice that you lined those up. And it is reassuring, and it's great that you could do that while drinking.

(Laughter.)

DR. KENT: It's my super power.

FEMALE SPEAKER: I don't think the -- I think the diagnostic and then the sort of -- you outlined this tension with the diagnostic goal, it won't be as prevalent in other types of acute pain but probably there will be the most tension in this particular category, I think.

MALE SPEAKER: Mike, great presentation.

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You went through the exercise of trying to map the concept of acute visceral pain to a couple different dimensional constructs, what we started with, with the initial 5 mappings and then a couple evolutions that we went through yesterday.

Could you talk a little bit about your experience and which ones seem more reasonable, which ones you struggled with conceptually, and any lessons you learned from that process?

DR. KENT: Sure. The acute visceral pain, it seemed to fit okay in the original 5 dimensions, had to sit down and do some thinking about it. But it was easy to kind of squeeze in, even with the tension with the diagnosis and describing the burden of the pain. But I think it fit pretty well with what we came up yesterday.

For me, for acute visceral pain, what is core, and it's already a vague topic. Between Dimensions 1 and 2 is where I had the strongest difficulty in separating what was core, what was common for something that has a such a wide array of presentations.

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Dimensions 1 or 2, I think I struggled with the most, and I guess particularly in the concept of the event. I just may have a mental block with this where I-just-got-my-knee-replaced pain, sweet. I got the event right there.

But coming into Dimension 1, coming into event, you almost have to start with the diagnosis. You have to start at the bottom of the iceberg up top. Again, I'm probably being as vague as the concept itself. But those are the two I struggled with the most.

MALE SPEAKER: There were a couple of comments that were excellent yesterday, suggestions about linking this to ICD-10. Do you think that would have been a helpful launch into the event in your schema, and would it take care of that tension between Dimensions 1 and 2?

DR. KENT: Possibly, yeah. Yes, sir?

MALE SPEAKER: Mike, just a quick comment. So coming at this from a completely different silo, I've sat with sponsors who intend to develop molecule, where a regulatory scientist will say,

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well, I'd like to see it work in an acute pain indication. Then they say, well, maybe a surgical or nonsurgical. And then they drop the bomb, well, why don't you try in visceral pain?

At that point, you want to shoot yourself because you can't diagnosis what the hell you -- who's going to go into it at the start. You prospectively have, as you said, a lot of negative diagnostic signs. And at the end of months of thought, it's like, goddam, I only got 5 patients enrolled in this study.

So this is probably one of the most vexing areas that I've seen both from a clinical presentation point of view but also in trying to deal with it. And I've had a number of colleagues suggest we should just eliminate the visceral pain category because it's just the trash bag of stuff that you can't figure out at the start.

I wonder what your comment is after having to wrestle with this concept for a couple of weeks now?

DR. KENT: Well, it's probably not worth

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throwing out just because it has such a vast amount of impact, especially I think in the ER.

Yes, that's an interesting point. I don't think you can throw it away. I think about the initial chronic pain taxonomy and that bottom category of "not otherwise specified," not that this is synonymous with that, but it falls into the vagaries of that where it's just going to be tough to do.

Going on, you know, Dr. Bruehl's studies with CRPS and stuff like that, let's just be honest, it's going to take a lot of work and decades, and decades, and decades to get to that Jaffe textbook of surgical procedures, where you look up how to do -- as a CA-1, how to do the anesthesia for acute cholecystitis, and there our taxonomy is for what the acute pain taxonomy and diagnosis for it is in terms of percentage of this pain, percentage of that pain, percentage of risk trajectory.

I'm not saying that's going to happen, but we're a long way with it. And that's one of the

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things I've realized every day. We think we have a lot of work to do in chronic pain. We have a lot of work to do in acute pain, where we think that we know what's in front of us, but when you actually start asking the numbers and look for the analysis -- I think we kind of are starting to know about total knee because that's all we really talk about anymore. But I think we have a lot of work to do.

I don't know if that answered your question.

Yes, sir?

MALE SPEAKER: I had a question. Under Dimension 4, the milieu that Dan was talking about, how do you account for, in clinical trials as well as practice, the effect that the therapeutic intervention may have on the diagnosis and also on outcomes?

By outcomes, I don't mean necessarily beneficial but adverse. It may worsen the condition as you're trying to study the therapeutic efficacy of the agent you're studying.

DR. KENT: I looked over to see if

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Bob Dworkin was there. That's a great question. I don't have an answer to that to --

MALE SPEAKER: I mean, certainly for example, with opioids, this role, effects of opioids, at least when I was taught, was somewhat controversial, and now, we use them; I understand that. But still they present complications for diagnosis and maybe in some ways confirm a diagnosis; I understand that as well. That's Dimension 1 and 2.

But I'm more concerned about the milieu that was spoken about yesterday, where there may be adverse effects of the medications we're using and that we're studying or evaluating, and how they may actually diminish any sensitivity to the therapeutic effect. And maybe that's why there's such difficulty in conducting the trials that Paul was talking about.

MALE SPEAKER: Can I just comment? I thought you put on the screen there exactly what I had been thinking, because as I've been watching these talks and have been looking at what we came

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up with yesterday, I think the dimensions map not entirely but fairly closely with what the chronic pain had.

I'd just like to mention that and get everybody thinking, because this afternoon, after we're finished with all the talk, I think that will be a very useful thing to do, is to revisit these and maybe think about how we can get it as close as possible to what the original 5 dimensions were just for consistency.

But the more I think about it, I think what we mentioned yesterday all fits pretty well, with a couple of exceptions with what we came up. I think you did a great job putting it together.

DR. SURESH: First, I want to congratulate you on taking on this really difficult topic. It's a lot of work, and I would have happily sponsored a couple more drinks for you if you could have gotten a little bit more work done.

(Laughter.)

DR. SURESH: The question I had -- and this is the slippery slope that again we get into.

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GERD, you talk about GERD. In pediatrics, we have this EE, eosinophilic esophagitis. These are recurrent patients who keep coming back to us.

Is this an acute or chronic? Like a sickle cell disease patient who then comes in with an acute crisis, how do we -- because the distinction is going to be really difficult and it forms a really good framework especially as we are submitting the ACGME paper for acute pain. There was no address of this particular issue, so I was just curious to see your thoughts.

DR. KENT: That's a good point. And I think that talking to Roger Fillingim yesterday, the keyword I would focus on, whatever we come up with, is "flexibility."

If you go by the text book, the textbooks, they talk about the current, subacute current. And they just chose a line in a sand saying that any recurrent visceral process that is -- we exclude any recurrent process that has a high probability of chronicity from what we consider to be acute visceral pain.

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I'm not saying that's what -- I'm just saying that's what the book said, or one of the books in one of the articles that I sifted through.

With things like eosinophilic esophagitis, you bring up a really good point. I don't know what to cut off. I don't think that's 7 days, but I think in other things, it could be 7 days. And that's why I think this speaks to the mammoth amount of work that needs to get to done.

I think each condition or each pain condition we talk about, it's going to have its own unique transition point to when we consider it, This needs to be managed in, and we'll call it arbitrarily a chronic pain setting.

I think that's going to be condition-specific, and I think it's an elephant in the room because that's going to take a lot of work in terms of trying to put a blanket over all acute visceral GI events, let's just call -- let's call chronic once they've occurred 3 times.

That may be useful from clinical standpoint, but I think, especially for an ACGME standpoint and

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the development of acute pain medicine physicians, I think it's even more important to make that gray to encourage that crosstalk between acute pain medicine physicians and chronic pain physicians so that there's not a delineation but a continuing conversation. And I know there will be big disagreements on that, but that's just my own personal view.

So I think it's going to be diagnostic-dependent, and it's going to just take a lot of work, and probably institutionally, your institutional capability.

At Walter Reed, we don't have block jocks. With the acute pain medicine service, we do it all. We manage all in-patient pain. We do the blocks; we do everything. We do in-patient.

Other institutions don't have that model. And that's not bad. That's just what that institution has. Maybe you have other institutions where you just have the block guys, and then chronic pain manages everything afterwards.

That may not be a wrong thing. It may work

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really great for that institution, but that institution may have defined eosinophilic esophagitis as, you know what, our chronic pain guys manage that just because these are our pathways. Our outcomes are doing great. Our patients are happy.

So just too muddy the water even more, I'll throw that out there. I'm sorry. I'm taking up too much time.

DR. TERMAN: No, no. Thank you very much. Thanks for all that work.

Just a reminder that this meeting is to develop a framework, not to necessarily get every hole filled for every event type. The idea is to develop a framework in the same way that was done with the original AAPT meeting, and then later had to be filled in by the various work groups that we heard about yesterday.

So the next talk, again, a little bit of a grab bag in terms of cancer and immune-mediated pain. Knox Todd, who until recently is from MD Anderson and is now from ABIA, a bar stool in

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Argentina; is that right?

(Laughter.)

Presentation – Knox Todd

DR. TODD: We call it a bar stool, yes. Si, de barra.

Thanks. This is a daunting task for me. When Bob and Dan asked me to do this, I said, thank you so much for this daunting task. Would you please limit the topic to cancer pain? Which I've done. So take out "immune-mediated pain."

I was thinking about the stages in my career, and there was a time at which I actually -- like all academics learn more and more about less and less. Right? That was my -- that was the career -- that was the plan.

In thinking about the visceral, the pain question, I was thinking about scopes, colonoscopy. So my brother had a diagnosis of colon cancer at a very young age; taken care of. He's in his 80s now. He's doing great.

But all the family had to get screened. So I'm in my 30s, fine. I'm at Emory. My buddy in GI

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says, come in, let's do your scope. I said, "Oh. I'd like to learn more about this. I would like absolutely no analgesia or sedation during this event." And I wondered, would this be a great visceral pain model?

It was an interesting hour. It seemed like 5 hours. It was not a bright thing to do, but I did learn more and more about less and less at the time. I've subsequently had two -- passed that, offline, with whatever, propofol or -- not there at all for that experience.

For the last few years, I'd been more of an administrator, so I've learned -- now, my job is to know a little bit about a lot of things. And I'm getting toward the point in my career where I -- there's so many things about which I know absolutely nothing. That's really success for me.

This is an area I've been working in as a manager, developing a new department of emergency medicine, MD Anderson, which is a task that we just finished up. There's MD Anderson at the bottom. It's a 55-bed emergency department, about 25,000-

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patient visits a year, all for cancer.
When I got there in 2010, this was the first article that looked at the presence of emergency department cancer visits.
It's a North Carolina study. Sam, you probably know these people. It's from the NC DETECT program, which was a surveillance system built for bioterrorism as I recall. It's been used for a number of things, but the first study that looked at cancer visits, cancer patients who visit the emergency department.
Like all visits to the emergency department, pain was the number one reason patients with cancer came to us. Oftentimes in this population, the visit to the emergency department was the index visit where cancer was diagnosed. And that had some meaning when we start thinking about how to define acute cancer pain.
Oftentimes, patients would come to the emergency department here with cancer progression, symptoms of progression, treatment issues, radiation, chemotherapy. And when we think about

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that, we had to go back and think about this event process that I've been hearing a good deal about.
I had a sad -- unfortunately, a good friend of mine was diagnosed with lung cancer just 3 weeks ago; New York, went to Mount Sinai, into the ER with symptoms. She said, "You know, this has been bothering me for a long time. I've had this funny kind of pain in my chest." And she's 50, a nonsmoker, spent a lot of time at 9/11 a number of years back.
Went in. Finally took maybe about 6 months, these sorts of symptoms; just kind of unbearable, went in, was diagnosed with a stage 4 non-small cell with leptomeningeal disease. It gradually progressed. My wife and I just went up and transported her to Florida.
But these visits to the emergency department, obviously, her life was devastated at that time, and you can imagine all her reactions: anger, pain, frustration, guilt. Nine-year-old son, my godson, just really crucible-type experiences in the emergency department with pain.

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But I was wondering, when do we think about event in that case? Was it the genetic mishap perhaps related to 9/11 exposure 10 years ago? I have no idea. Was it some build-up in the ischemia lactate in the tumor mass that caused her symptoms? I really have no idea.
But in the North Carolina study, pain is the number one reason people come to the emergency department with cancer-related problems. It looks like it's about 30 percent or so of all cancer-related visits in North Carolina.
We repeated a similar study in Houston, and we think the number of emergency department visits related to cancer is about 1 to 2 percent of all visits. For us, that would mean -- what do we have, 130 million visits a year -- a lot of visits, 1 to 3 million, something like that, a year in the United States.
If you look at the cancer literature and try to think where does pain fall out, there's not much separation between acute and chronic pain. Most of the literature assumes that all cancer pain is

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chronic. And if you look at the prevalence of pain and different cancer types, head and neck cancer is always at the top of the list, head/neck, GI, lung with others with a lower prevalence, which we might want to think about when we look at particular candidate models for studying acute cancer pain.
This is the quotation from Roselyne Rey that Dan referred to the other day. He did a great job, by memory, of that quotation. But it actually reads, "Pain is bio-psycho-social phenomenon. Nociception represents anatomy and physiology, but cultural and social factors are the foundation for the expression and treatment of pain."
I don't think there's any condition where that social and cultural meaning is so strong as in cancer pain. And if you look at temporal trends on how we approach treatment in cancer pain, look at the quote from the late 1800s, talking about the use of opioids to control terminal cancer pain, and the 180-degree pendulum swing in the mid part of the last century where in JAMA, one of our physicians noted that addiction is a bad side

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effect of opioid treatment for cancer.
Where we are today, I don't know. But I do know that cancer has a privileged place when it comes to pain. We can have lots of discussions about how cancer pain, chronic cancer pain, differs from non-chronic cancer pain and treatment approaches. We know that many patients with cancer tend to be surviving longer now. These questions are incredibly complex to tease apart.
So as a clinician -- this was asked I think yesterday -- what would we like this taxonomy to do? Certainly, when we assess cancer pain, we look at the presenting features, we try to make guesses about pathophysiology and prognosis and determine our treatment plans.
There is a long history of various assessment tools related to cancer pain. And in looking at some of the literature and thinking about this, it's really a daunting task to look at the past work in this area and think how to evolve that into another taxonomy.
Probably, the Edmonton system is one of

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the -- although developed in a single-center, perhaps not as consensus-based as others, it's descriptive and somewhat a prognostic classification scheme.
Many of these systems give a hierarchy to neuropathic pain, elevating it over other types of pain. Some of these tools are quite crude, the Pain Management Index being one of them, many limitations in terms of content validity, lots of need for validation studies.
It's wonderful that the papers are being published in two of the U.S. journals. Is that correct, Dennis? So how do our editors, how do our editorial staffs view these assessment schemes, what are the standards for using these?
This is a typical clinical approach. We look at the sensory features of the pain experience as we can collect them from our patients, look at what is the meaning of pain to them, what pain behaviors do we see, try to understand the social, culture, and effective components of that experience and try to arrive at an etiology and

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then a treatment plan.
This is the typical classifications you'll see for -- like I said, you'll note that when you see this, you always see cancer in the middle under "mixed," right? Isn't it always there? It's one line, "cancer." It's a mixed pain.
It just means that cancer is so many things. That's why I think tackling something like acute cancer pain is such a daunting task: tumor invasion, direct neuropathic pathophysiology, leptomeningeal disease. We'll talk a little about chemo neuropathy. Various somatic, direct invasion of tumors into the soft tissues, bony mets, and then visceral pain, quite common in cancer patients. And the number one cause of visceral pain in my emergency department, MD Anderson, is bowel obstruction, a very common presentation.
In general, the literature would tell us that about 75 percent of cancer pain is due to the tumor itself. About 25 percent is due to the treatments of cancer: surgery, chemo, and radiation. Then depending on your patient

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population, there's a large proportion of these patients who have non-cancer pain syndromes, and how to tease these apart is going to be really tough in this area.
There's so many things going on at the same time. It's kind of like a bad day for an air traffic controller when you have multiple planes that are unstable in the air and trying to keep them all afloat and achieve the best outcome for the largest number of passengers.
Clinically, using the dimensions that we had before coming into the meeting, we get around 1, 1 and a half, and then most clinicians will try to find a bucket to drop pain into. We may think, well, this is neuropathic syndrome under one of these categories. This is due to surgery; this is a radiation plexopathy; this is some paraneoplastic syndrome or some chemotherapy-related peripheral neuropathy, and that will help us arrive at a treatment strategy.
We'll try to go through the core diagnostic criteria -- the core dimensions as -- or the

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dimensions as they were presented to me, briefly, and I'm going to -- a lot of this is repetitive, obviously. But I'm going to highlight some questions that I had in thinking about going through this as it relates to cancer pain. And then we'll talk about a few models that might be useful for a study in here; I think there's a lot of promise. And we'll identify some candidates using the criteria on the bottom.

So core diagnostic criteria. When I see a patient with pain, the first thing I'd have to ask is, is cancer present? And that would seem to be a reasonable thing and fairly easy to figure out.

Remember, that a number of patients who present, say, to my treatment setting haven't been diagnosed. It's their index visit, so they don't have a diagnosis of cancer yet, difficult to know how to study that population.

For a clinical trial, would patients be required to have a tissue diagnosis? How extensive would that tissue diagnosis be in order to enter into a trial? I think those are questions that we

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could ask.

Then what is probably more troubling here is, is the pain that we're attempting to study really cancer-related? I mean, there are any number of comorbidities that patients with cancer have. And we might think, well, radiation for a tumor or chemotherapy and pain related to that, that's clearly cancer pain, right? Would you all agree with that?

So what about post-procedural pain? Henrik, you have a patient who has a mastectomy. Is post-mastectomy pain, is that a cancer? Is that an acute cancer pain model?

I would say, well, probably. But it kind of depends. If the post-operative course or the features of that procedure are really indistinguishable for the patient without cancer, I'm not sure how useful that model might be; anyway, empirical questions that we can answer.

The difficulty in the last question, the temporal nature, is pain acute, how do we deal with all these planes in the air, these multiple pain

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trajectories for a patient who may have pain due to tumor, predictable pain due to treatment, pain due to comorbid conditions. How do we decide what these represent? How do we put those into buckets? How do you sort of prune and sharpen this taxonomy to take care of those things?

Even breakthrough pain, if it's not due to end-of-dose failure, if it's -- you know, oftentimes, breakthrough pain is the presentation of an extension or a progression of cancer. Is that an acute pain? Probably, but difficult to tease out; or should we just decide that the only cancer pain we're allowed to study in an acute cancer pain model is that there's no prior chronic pain or pain unrelated to the new event.

Anyway, interesting questions I don't have an answer for.

We mentioned, too, it was perhaps somewhat easier -- the typical features, again, a wide variation in pain features across cancer pain syndromes. And in cancer, we see symptom clusters. Very often pain is associated with wasting, with

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depression, with fatigue, and there's multidimensional assessments addressing all of these particular symptoms we need to think about how to tease apart or perhaps lump together.

Dimension 3. Cancer is a disease of aging. So many people with cancer, it's an aging population, are going to have multiple comorbidities. How do we deal with those? Those comorbidities may influence our treatment plans, depending on the prognosis that the comorbidity would imply for someone who presents with cancer.

Certainly, comorbidities are associated with various toxicities that will limit our ability to treat pain and limit our ability to treat cancer. And there's some conflict that could occur between those two goals. We'll talk about that in a bit.

Psychiatric comorbidities associated with the cancer, you can imagine that my friend went through a number of stages as she dealt with her new cancer diagnosis.

Interestingly, with regard with substance use, coming back to the discussion, these temporal

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trends and how we think about opioids, say in cancer, there has been some assumption by many of my colleagues in emergency medicine that cancer is a vaccine against substance abuse.

We think this is crazy, but it's commonly -- cancer is a morally-positive disease to have, right? One of the things when I came to MD Anderson, I was interested in was how do we think about this, how do we study this in a place where there's perhaps more light and less heat than perhaps studying another condition.

So one of the things we did is look at -- I'll just use a simple -- so risk stratification for opioid abuse risk in cancer patients in the emergency department, and found, really, kind of a high proportion of our patients were, by that assessment tool, at risk for opioid abuse.

At the same time, we looked at prescription monitoring programs statewide to say, well, is there something that would suggest that there are problems? And indeed, there were associations

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between subscores and prescription monitoring program data.

So I went back and asked my patients in a survey, what do you do with your opioids? Because in the cancer community, we always had patients who were switching from one opioid to another one. Remember, multiple planes in the air, multiple approaches, and many opportunities for drugs to filter out of the healthcare chain.

So a decent proportion of my patients explained to me, quite patiently, that they were in dire economic strains. Cancer is an economically devastating condition. They have a valuable drug. "What do I do, Doc? Pay the rent?" So these issues are there in this community and they're often not talked about.

The consequences of acute cancer pain, as you can imagine, tremendous effects, even existential threats with a new symptom related to cancer. The patients, when they come to the MD Anderson emergency department with a new symptom, come in with incredible fear as to what

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the meaning is of this new symptom.

We spend a lot of time doing family counseling in emergency departments trying to sort out the multiple communication issues they may be having with their caregivers, the threats that this pain represents for their social roles.

One of the things about cancer, particularly those conditions that are likely to have a negative functional trajectory, if you're trying to understand the consequence of pain, we're less often attempting to return or restore a patient to previous function than to prevent or delay a progressive decline in functional status, so a bit of a difference between acute cancer pain and other acute pain syndromes.

Now, if you think about personalized precision medicine, oncology, right, is the place to be. Huge amounts of data, huge amounts of -- as Donald Trump would say "huge amounts" of computational capacities, statistical models to deal with genomic data, genome-wide association study -- incredible amount of data. And I think

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the pain community would benefit from a closer alliance with the oncology community, closer than it already is.

Pain is a brutally inflammatory condition, so it would seem that this is a population for which an acute to chronic pain transition is quite likely. It may be very sensitive a model for studying these things.

Again, with multiple cancer syndromes overlapping, how to decide on more specificity, how to prune that tree to provide more information is going to be a complex task.

We talked a little bit about addiction. One of my colleagues, Cielito Reyes-Gibby, has published a couple of papers looking at pain as an independent predictor of survival in head/neck patients. Why is that? What's the mechanism for this?

There is interesting data, somewhat epidemiologic with some biological reason to it, that perhaps mirror opioid receptor activation is associated with tumor progression,

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epithelial mesenchymal transformation in metastatic disease. I don't know if that's real or not, but there are other analgesic modalities: antioxidants, steroids even that may interact with our goal of trying to provide tumor-directed treatment. And perhaps pain treatments may on occasion inhibit our ability to provide tumor-directed treatments.

This is a grab bag of different issues. There are many classification schemes related to tumor itself, treatment, and prognosis, how do we capture those elements in a scheme like this. And maybe this is simpler than I think, just picking and choosing how you want to do this. There's so many institutional infrastructure, structural organizations that have a stake in this and how do we interact with those in attempting to develop an acute cancer pain taxonomy?

We talked a little bit about the impact of pain therapy and our ability to deliver cancer therapy. And sometimes, there must be a tradeoff there and important to study.

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Just thinking about a patient who comes to the emergency department with a spinal cord compression, and I want to give that patient steroids, I need to know that that patient doesn't have a lymphoma that might be glucocorticoid responsive before I do that; so some interesting questions in how to deal with that tradeoff between pain therapy and treating cancer.

There are a number of schemes out there from NCI that detail how we're supposed to judge toxicities of agents, how do we incorporate those into the system. And I keep going back to my friend who had this acute pain syndrome.

So what would I learn out of this is an acute pain syndrome that I can recognize in the emergency department, perhaps accompanied by a biomarker that would allow me to diagnose cancer before it was stage 3 or stage 4. And that's really where the money is in trying to prevent the consequences of cancer. Screening and preventing, is where we need to go; might there be a phenotype, a marker that could help me understand who needs a more thorough work up.

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Sean and I were talking about rapid learning systems and how we capture information from patients, from clinicians, investigators, in an efficient fashion that would allow us to incorporate that into our larger databases.

How do we think about editorial consensus about how to develop a system like this, how to get it accepted within our journals? Should that be an international effort or remain national? Are our instruments culturally robust, and will they cross the borders that exist within even in the United States?

So what models might we think about? And I just put these up as I know useful things to think about. Whether these are the right models, I really have no idea, but here's my rationale.

If you want to go for prevalence, certainly, head/neck cancer would seem to be a disease with high pain prevalence, early pain presentation, dense trigeminal innovation, and very much large functional consequences.

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So every time we eat, swallow, wet our lips, that's going to impact or cause pain, and the ability to function with pain during that period of time is very important in terms of preventing hospitalization, allowing us to tolerate chemo and radiation therapy.

There are probably ways to think about the acute to chronic pain transition that we will be able to impact. There are mouse models that we can use to study underlying mechanisms. It meets a lot of the criteria that would seem to be important here.

Bony mets, I think that's come up with the chronic pain group, but mouse models have been around for 20 years to study. Bony mets, interesting condition where the primary tumor in its location may not cause pain, but it causes pain in the metastatic site, presumably due to local circumstances. But some mets cause pain; some mets cause no pain, and it makes no sense. It's like low back pain. Imaging doesn't really correlate with symptoms in those patients. Why is that?

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Can we use phenotypes in this population to predict response to therapy? And a number of mediators are proposed here, but would seem to be a reasonable acute pain model and has synergy with the chronic pain effort.

Chemotherapy-associated pain, I think you'd all agree, is probably a reasonable thing to look at. Very common during chemotherapy, lots of important implications for whether patients can tolerate therapy, and fairly distinct acute neuropathic pain syndromes that may be easy to relate to a specific agent.

So one can tease apart taxane and platinum-based mechanisms spatially in the periphery, dorsal horn and central nervous system by mechanism. And there's some indication, given that many neuropathic pain drugs don't work well for this condition, that these mechanisms might point to a more profitable areas to explore.

Certainly, acute chemotherapy-associated pain happening over days to weeks is predictive of chronic peripheral neuropathy. So again, a nice

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tie-in between the chronic pain effort and the acute pain effort.

A bear for us in the emergency department is mucositis. Chemotherapy or radiation-related mucositis has huge functional implications. We very commonly are forced to admit patients; they will get TPN; they will end up with gastrostomy tubes because they cannot function because of their pain.

How do we prevent that? There are a number of assessment scales that we can use, a number of topical and systemic treatments that might be useful, all candidates for study, and again, nice animal models to help us make a bench-to-bedside translation.

Finally, post-mastectomy pain, the older literature had these numbers up around 50 percent. Once you do define post-mastectomy pain more precisely, it comes to be about 25 percent will have either severe or moderate pain going up to 6 months.

This is from Chris Miaskowski who identified

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various pain trajectories with a larger sample, is continuing to do work to look at phenotypic and genomic risk factors for these patients. And again, we come to this question of how does a mastectomy differ from a breast reduction surgery in terms of consequences? What's the difference there, and kind of a nice model to perhaps look at.

I'm sure you could choose any of these and come up with good reasons to think about proposing these acute cancer pain models. The list and the data -- I mean this is really a daunting, I think, task, And I can imagine this taking a long time to do, not only the intellectual concepts, but just the organizational issues in dealing with all of the cancer organizations, dealing across very established organizations with their own thoughts about how to assess these issues.

So finally, on a more positive note, the answer on the word cloud -- so I'm going to throw back a word cloud. We're just finishing the first book on oncologic emergency medicine, what I hope is a more comprehensive text written by emergency

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physicians and oncologists, coming out in a couple of months. This is the world cloud from our chapter on pain treatment.

Many promising things about working with the pain community. Again, huge data, large repositories of information, large computational capacity, complex organizational structures to work through, but a wealth of support.

Considering NCI budget is larger than NHLBI and NINDS combined. For this talk, I looked at the NIH funding categorization tool, which is kind of an art more than science perhaps. But cancer is funded at 6.3 billion, number 4 of 256 conditions; pain, 481 million, number 63.

Don't feel bad about that. Emergency medicine is number 128. We're sandwiched between inflammatory bowel disease and cannabinoid research.

Nonprofits, the interest in looking at toxicities related to cancer therapies, huge amount of philanthropic funding in the country. This was the culmination of some work over the last few

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years. NCI in the Office of Emergency Care Research, which is kind of like the Pain Consortium at NIH. It's got an office, a little bit of funding, and some logistical support.

But it announced a new consortium of emergency departments, most associated with a conference of cancer centers to collaborate on emergency department-related cancer research. And I think this would be an ideal group to take validation studies before as we think further about this.

So that's a quick run-through. Thanks.

(Applause.)

DR. TERMAN: So we're going to wait and do the acute neuropathic pain after the break. But if there are any questions briefly for --

MALE SPEAKER: So I have a comment, which ism, first, it's fabulous that you're here doing this because as you put the different Venn diagrams up for different topics, I'm thinking you're yourself at the intersection of several circles, almost uniquely, among people on the planet.

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Where I'm going with this is also, I think that it was very valuable that you called attention to the sociological factors, which could influence uptake of any taxonomy.

I think that for a number of years, for whatever reason, the concept that acute pain is worth studying, worth intervening, worth thinking about became kind of eclipsed as the intellectual center of gravity in the pain realm migrated to chronic pain and, to some degree, cancer pain.

I think it was just a separate compartment, and acute pain operationally was defined as the services provided by the acute pain service in the hospital, meaning the services provided by the person carrying the beeper that day for the acute pain service that never got a critical mass.

So I think this is terrific, and it speaks kind of across the whole taxonomy. But we must keep in mind the sociology of the intended users or clients. Otherwise, this will just be in some box that they perceive is not related to their own box.

DR. TODD: Right. And incorporate it -- one of the ways to do that is to bring them to the table as early as possible. It's truly achieving a consensus and figuring out who those people are and how to engage them in a nonthreatening way.

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FEMALE SPEAKER: It was a really nice talk. I think your discussion of the chronic post-mastectomy pain or post-mastectomy pain sort of brings up one of the categorization problems that we may run into.

Certainly, in a chronic realm, putting post-mastectomy pain with cancer pain makes a lot of sense. But then when I think about acute pain after mastectomy, it almost seems to fit naturally into the post-surgical.

So I wonder how -- and there may be a number of different cancer-related surgeries that would fall into this dilemma. I'm just wondering about your thoughts about that.

DR. TODD: I think you're right, and I think Henrik would probably agree that this is a post-surgical model. The same issues of anesthetic technique, the same issues of surgical approaches, nerve preservation, to my mind, put it a little more in the post-surgical realm. Agree.

Henrik, right?

DR. KEHLET: Yes.

DR. TODD: Okay.

DR. TERMAN: Okay. Thanks, Knox.

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So we're going to take a break. We're running a little bit behind, but we have the room until 10 o'clock tonight. Is that right? So I'm going to keep the break at half an hour in case those of you who haven't checked out yet need to do that. So we'll be back at 10:45.

(Whereupon, at 10:17 a.m., a recess was taken.)

DR. TERMAN: Hello. We're going to start here. The next speaker is Srinivasa Raja from The Johns Hopkins University again, and he's going to be talking about acute neuropathic pain.

Presentation – Srinivasa Raja

DR. RAJA: Good morning. Thanks to Bob and Dennis for the invitation, and particularly thanks to Greg in the sense that I didn't have to compete

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with the coffee break.
(Laughter.)
DR. RAJA: I did my presentation like many of you, and I got done about a couple of days ago. I thought we had already discovered the holy grail of pain taxonomy, and Roger and Bob had already found and discovered this holy grail, and everything that we had to do was in relation to this holy grail.
But yesterday afternoon, we thought there may be some changes to this holy grail. Much like Mike's presentation, I spent some time last night trying to update the presentation, but without the bourbon. So I'm not sure how well my update is going to be compared to Mike's.
I think about a month or so ago, there was a conference call among all the presenters. And some of the discussion was what should we be presenting, what should be our goal? And some of the questions that came was, you need to look at, is this a good bucket for an acute pain condition; is the condition homogenous; give some examples of

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conditions that would be useful to include; what are some inclusion/exclusion criteria; and what is the usefulness of these conditions based on problems, clinical, or research importance; and does the condition share some common pathophysiological mechanisms? So I'll try to address some of these issues as we go along.
I knew that I was going to be speaking to a very erudite, scholarly group of individuals, and I thought I needed to be innovative and come up with something cool. However, working through this presentation, I realized that trying to reinvent the wheel is going to be kind of challenging and not very fruitful.
That's particularly because over the last decade or so, NuPSAC, a Neuropathic Pain Specialists group, has spent a lot of time coming up with taxonomies and definitions for neuropathic pain. So I'm going to take a couple of the studies that have been done by NuPSAC and use that as a foundation for some of the diagnostic criteria or the core diagnostic criteria.

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Ralph Treede had a publication in Neurology in 2008, and that was on a grading system for neuropathic pain. And just recently, Nanna Finnerup did an updated grading system, which is now in press and will become out this year in Pain.
What was found was looking between 2008 to 2015, there were 414 citations of this initial grading system, and 316 of these had used the definition that NuPSAC had come up with in 2008 for their clinical trials, and used that as a kind of concept for defining who has neuropathic pain.
So for example, the leading complaint obviously is pain. And based on history, one looks at the history of relevant neurological lesion or disease and a pain distribution in a neuroanatomically plausible distribution.
If that's not present, both those conditions, then we say it's unlikely neuropathic pain. If both those conditions, based on history, are present, then you say it's a possible neuropathic pain. Then you examine the patient,

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and if the examination comes up with pain associated with sensory signs in the same neuroanatomical distribution, then it becomes a probable neuropathic pain. Then, to get to the next stage of a definite neuropathic, confirmatory tests are needed, such as diagnostic tests will come to what that could be.
So this was kind of the grading system that has been updated. In the infinite wisdom, they don't talk about acute or chronic, so they talk about neuropathic pain as such. I think the same can be adapted for acute neuropathic pain as well.
Another study, again, sponsored by a neuropathic pain special interest group, which was published in Pain a year ago, was the attempt to phenotype patients with neuropathic pain for large-scale genetic studies.
This was based on the Delphi survey of experts like you. One of the first things was survey of a few questions, and these questions were to determine sensitivity and specificity of symptoms and signs and whether it could be done,

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whether it was feasible to be done by the patient themselves contributing or the presence of non-experts.

What the experts came up with was that if you just look at symptoms, then it's thought that patients could fill that in, and 75 percent agreed that patients could fill in their symptoms, that non-specialists could assess it based on symptoms. However, in terms of sensitivity and specificity of this approach, there was no consensus among the experts. So symptoms by itself was a poor way of diagnosing neuropathic pain.

It was felt that clinical signs are essential, and 75 percent of the experts agreed that if you add the clinical signs, that probably the diagnosis can be made with some degree of confidence.

The next round of this Delphi survey was the panel was asked what is the combination that's going to be needed to have a degree of certainty, whether it'd be possible, probable, or definite? And what the expert panel said, if the

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symptoms -- a body chart in the history, 80 percent felt that a possible diagnosis of neuropathic pain can be made. But for a probable diagnosis, you need the clinical signs, the symptoms, body chart and a history. And really, for a definite diagnosis, you needed some additional investigations.

So this is a kind of framework, which could help up develop the diagnostic criteria or the core diagnostic criteria for neuropathic pain.

As you know, there are a lot of questionnaires that have come up with neuropathic pain: the LANSS, the painDETECT, DN4, NPQ. These were discussed. While these help provide us kind of a spectrum of the symptoms and signs, it was not something we felt was essential for the diagnosis and probably not useful in the broad taxonomy context.

Again, a number of diagnostic tests such as quantitative sensory testing, somatosensory evoked potentials, laser evoked potentials, et cetera, skin biopsies. These are maybe acquired for the

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research aspect in terms of where you want a high degree of specificity, but it's probably not useful for taxonomy and a diagnosis in the clinical setting.

So if you come up with a terminology of acute neuropathic pain, it can be defined as acute pain caused by a lesion or disease of the somatosensory nervous system, as NuPSAC defined earlier. It may be the result of injury that involves any aspect of the peripheral or the central nervous system.

So we had this discussion about including organs or systems in our taxonomy, and based on that, one way would be to characterize them as acute peripheral neuropathic pain and acute central neuropathic pain.

Much like Mike, what I did was initially started with the earlier core diagnostic criteria, but then took some of the discussions that went on yesterday and said, do some of the themes fit into those core diagnostic criteria?

So let's just take acute neuropathic pain,

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and the one core diagnostic criteria was use a NuPSAC definition of neuropathic pain that is related to an injury or a disease. The injury is a clear event. The disease, maybe the event, as was discussed, may be a little bit more challenging as to when the event occurs. But clearly that is part of our core diagnostic criteria.

The one question we can discuss is, the duration between the event and the diagnosis or the neuropathic pain, should that be days, should be 7 days, should be 30 days, should be weeks? What should, be, that temporal relationship between that trauma or the injury and the event?

The common features of neuropathic pain has been very well described, the quality of the neuropathic pain, as we said, in terms of the different scales that have been used, the quality of the pains have been well described.

We know the temporal relationship or time course of events and the spatial distribution in relation to the injury. So I think these common features can be easily incorporated into the prior

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domain of dimensions of common features.
We talked about host, which was earlier common medical comorbidities. And this could be etiology-dependent such as for herpes zoster or acute zoster-associated pain; a change in the immune status such as associated with cancer or HIV; aging in terms of vascular insufficiency leading to amputation. So those are common comorbidities that we can talk about. And these are host factors, diabetes associated with diabetic neuropathy and diabetic neuropathic pain.
So all of these are really host factors, but they're also common medical comorbidities that may be associated with neuropathic pain.
In terms of the fourth criteria of dimension of neurobiological, psychosocial, and functional consequences, it's been well known that these are common in neuropathic pain states. In fact, work by Blair Smith and their group has clearly indicated that the quality of life impairment with neuropathic pain is greater compared to non-neuropathic pain states, but the functional

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consequences will vary depending on the etiology.
For example, the functional consequences of somebody having an acute zoster pain with a single dermatome will be very different from the functional consequences of somebody who has post-amputation pain.
So the functional consequences, the impact on their functions will be very depending upon the nature or the subgroup within the acute neuropathic pain.
In terms of putative mechanisms, risks and protective factors, we know a lot about the mechanisms of neuropathic pain such as ectopic activity, peripheral and central sensitization, neurogenic inflammation, and descending inhibition.
So much is known about the putative mechanisms. It may vary a little bit depending upon the etiology. But again, it fits fairly well under the dimensions that have been described earlier.
So I thought, once we have this acute peripheral neuropathic pain, we can then have

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possible etiological subgroups such as acute neuropathic pain from an infectious disease. Good examples are acute zoster, HIV, and leprosy. One of the most common neuropathic pain states is acute painful radiculopathy resulting from nerve root compressions to this disease.
Acute post-nerve injury neuropathic pain, such as causalgia or CRPS-2; acute post-amputation neuropathic pain, obviously, there is some overlap here between trauma as well as neuropathic but clearly, nobody would argue that phantom and stump pains are a neuropathic pain; acute trigeminal neuralgia.
Again, we talked about chemotherapy-induced neuropathy as a cancer pain. I think of it as an acute toxic neuropathic pain, and that it is truly neuropathic pain state resulting from a toxin such a chemotherapeutic agent. So one could argue should it come under cancer pain or should it be under an acute neuropathic pain state.
What I'll present is some examples of how some of these subgroups fit into the core

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diagnostic criteria that we've talked about. I've taken some studies in these subgroups and looked at the inclusion/exclusion criteria that have been used in these clinical trials. And thanks to Jennifer, who provided me a whole series of articles, reading material for the last month in terms of how the literatures use these different subgroups, and then some additional epidemiological studies in neuropathic pain state.
So let's take the most commonly studied neuropathic pain state, zoster-associated neuropathic pain. The pain has unique characteristics. It's unilateral in distribution; one or more spinal dermatomes, or in vivo distribution; the pain can have spontaneous or evoked characteristic.
There is a clear temporal relationship to the zoster rash except for that subgroup where there can be zoster-associated pain without the rash. So there is a small subgroup that may be an exception.
The examination of the patient is associated

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with negative signs of sensory deficits as well as positive sensory signs of allodynia and hyperalgesia. One can do quantitative sensory testing and further characterize. I don't think it's essential for the diagnosis but may be helpful in subclassifying the acute zoster-associated neuropathic pain.

When we look at the other dimensions, the epidemiology and the time course is very well known and studied. We know that there are some common comorbidities such as aging, stress, decreased immune status with HIV and cancer. We know the psychosocial and functional consequences such as effects on quality of life, social interactions. And for example, in trigeminal acute zoster, there may be vision disturbances.

We know a fair bit on putative mechanisms and certain risk factors such as age, extent of rash, and the early onset of antiviral therapies. So I think there is kind of a slightly different tick mark there because we don't know exactly how the viral activation and the inflammation of the

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dorsal root ganglion cells results in the pain state, so there may be some question on the exact mechanisms. But in most of these dimensions we know a fair bit in terms of acute zoster-associated neuropathic pain.

Let's take the other most common neuropathic pain state such as painful radiculopathy. Again, the history will be fairly clear here of pain radiating from the neck or the lower back to the extremities.

So the pattern of the pain has a certain characteristic to it. It may be in the innovation territory of one or more nerve roots. The question based on a number of the clinical trials that I've looked at is when do you call it acute and when does that transition to subacute or chronic? And clinical trials have used either 30 days or some 12 weeks or longer.

So again, we can argue when is the transition from an acute radicular pain or radiculopathy to when it becomes subacute or chronic.

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In terms of examination, the straight-leg test has been shown to be positive. In terms of its sensitivity and specificity, it varies a bit, and that varies depending on what one takes at the criteria. Studies, again, in clinical trials have taken anything from a pain associated with a 30-degree leg flexion to a 70-degree. And maybe some of the sensitivity and specificity will vary depending on what you take as your outcome measure in that examination.

Confirmatory tests could be MRI or CT, looking at encroachment of disc material. The studies have looked differential diagnosis of cauda equina syndrome, other sciatic pain secondary to tumors.

Again, because this is one of most prevalent forms of acute pain states, its common epidemiology is well known. Some of the comorbidities such as obesity has been well described, the neurobiological, psychosocial factors. And when these are variable, it depends on the occupation of the individual, it depends on the age of the

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individual.

The putative mechanisms, risk and protective factors, again, risks such as obesity, occupation, trauma are well known. However, the mechanism is still unclear. We all think that cytokines released by the disc material may be responsible, and that justifies our use of epidural steroids in these patients. But is that the true mechanisms? We don't know.

So let's move on to acute post-amputation neuropathic pain. Again, the core diagnostic criteria of distribution of pain and a missing body part, that its onset in 75 percent is within one week so it is acute, and 50 percent within 24 hours of the amputation.

The nature may vary in terms of frequency, intensity, and the nature of the pain. Again, you examine the patient. And some patients with residual limb pain such as stump pain, palpations of neuroma can cause pain and associated Tinel's sign. And there is some association in the sense that there's a close association of those who have

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stump pain or neuroma pain will also have -- a high proportion of them also have phantom pain.

We know a fair amount on the epidemiology; 40 to 80 percent of amputees will have some degree of post-amputation pain. It may vary between the site and the patient population.

Some of the comorbidities that we know are that it occurs more commonly in the elderly, particularly when it's related to peripheral vascular disease or diabetes. We know the functional consequences. In fact, post-amputation pain can result in higher risk for obesity, chronic joint and low back pain. Patients with amputation pain also have low back pain and sleep disorders.

A number of putative mechanisms have been studies and described, and the risk factors such as diabetes, peripheral vascular disease, trauma. A number of mechanisms are well known in terms of the ectopic activity from a neuroma, central changes such as cortical reorganizations, and psychosocial factors such as stress, depression, and catastrophizing.

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There are some controversy with regards to can this be prevented by pre-emptive neural blockades or by changing surgical techniques, so there are areas which we are still not clear about, so that's a tentative tick mark up there.

The other acute neuropathic pain is after a nerve injury. This is in the distribution of the innovation territory of the lesion nerve, usually distal to the site of trauma, surgery, or compression. And you examine the patient. You can demonstrate the sensory loss in the distribution of the affected nerve and the presence of allodynia, hyperalgesia, and can be confirmed with EMG or neuroconduction studies.

With regard to chemotherapy-induced neuropathy, I think it's an acute neuropathic pain. The pain distribution is distal, usually symmetrical in all extremities. And it's usually fairly close to the -- although this says within one month, there are reports that suggest that within the first week of initiation of chemotherapy, a good proportion of patients will

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have chemotherapy-induced neuropathic pain. And again, this can be confirmed on examination, where the pattern of distribution of this neuropathy or sensory deficits and the nerve conduction, EMG, may be helpful.

These were primarily peripheral neuropathic pain states. How about some central neuropathic pain states?

We all know that spinal cord injury is associated with an acute neuropathic pain, and this can occur as high as varying from 35 to 94 percent of patients depending on which study you look at.

Post-stroke patients, again, 10 to 50 percent of stroke patients can have an acute neuropathic pain. Acute traumatic central neuropathic pain can result from plexus or avulsion injuries or concussions and brain injuries, and a group of central neuropathic pain with neurological diseases such as multiple sclerosis.

We don't know what proportion of patients with multiple sclerosis may have a pain that is acute, in the sense early after their disease is

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diagnosed.

When we look at spinal cord injury, again, and look at the core diagnostic criteria, the pain distribution, it can be at or below the level of spinal cord injuries, therefore, clearly a characteristic distribution of their pain.

Onset, again, is fairly early, within weeks after the injury, days to week. And they have spontaneous or evoked dysesthesia, hyperesthesia and paresthesia. Again, their examination has these difficult findings and one can confirm the spinal cord injury with CT and MRI, so we know the level of injury.

Similarly, post-stroke acute neuropathic pain has some clear core diagnostic criteria in terms of the temporal relationship to the stroke; the distribution of the pain in terms of the limb contralateral to the lesion side, as well as in some cases the ipsilateral phase; the sensory and motor deficit; the changes in tone with exaggerated reflexes and associated bowel or bladder dysfunctions. And there are easily some

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confirmatory tests such as CT, MRI, and vascular studies.

Based on these kinds, what I suggest is that we have an acute pain state, so that is the event, which is onset or duration is related to that event. We then have an etiology or a mechanism that is injury-specific or a disease to the nerves, or the peripheral or the central nervous system.

So then we have an acute neuropathic pain, and depending on the system, where the site of the pathology is or the disease is, or the injury, we can have an acute peripheral neuropathic pain or an acute central neuropathic pain. I've given some examples of these acute peripheral as well as acute central neuropathic pain states.

There are several things that we need to clarify for this study and future studies, that is when does this acute neuropathic pain that we see with these conditions, when does it transition to becoming subacute or chronic?

What are the factors that predict this transition from this acute to chronic neuropathic

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pain states? Is the mechanism of pain in acute neuropathic pain different or similar to that of chronic neuropathic pain states?

Are the therapies that we all know, which have been well studied in chronic neuropathic pain states, are they equally effective in acute neuropathic pain?

Finally, as Steve has suggested, we need to test the validity of the diagnostic criteria that NuPSAC has suggested in terms of is that the gold standard, and the reliability of the diagnostic criteria.

So let me end by saying, we were asked to answer a few questions. So the first question was, is this a good bucket for acute neuropathic pain? I think clearly acute neuropathic pain is a good bucket to be used.

Is this condition homogenous? I think so. There is some differences based on the etiology, but overall this is a homogenous pain state.

I've given some examples about acute peripheral neuropathic pain states, as well as some

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central neuropathic pain states as examples, which can be used as subgroups within the acute neuropathic pain states.

Based on some of the clinical trials, some of the papers that Jennifer provided, I provided some examples of inclusion/exclusion criteria that have been used in these different studies.

Are they useful? I definitely think because of their prevalence, the functional consequences, that definitely this is a useful subset within the taxonomy of acute pain.

Do they share a common pathophysiological mechanisms? It's something I'm unsure of. There are some common pathophysiological mechanisms, but there are some differences between the different peripheral versus central pain states. And I thank you for your kind attention.

(Applause.)

DR. RAJA: Yes, Chad?

DR. BRUMMETT: Dr. Raja, could you expand a little bit on the group's decision not to use some of the self-report measures and why those weren't

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considered maybe -- just the application of those in an effort like this would be very attractive, right? It would make things much easier.

These could fit into different components of what we're talking about because these could be risk factors are they could be diagnoses. But you can you talk to me a little bit about -- tell us a little bit more about why that decision was made?

DR. RAJA: Yes. There was a lot of discussion on these, on the self-report measures and whether they should be used or not. I think the self-report measures are very good screening tools, and they are being validated as such in a number of countries.

Therefore, it's a good tool overall to screen the potential presence, but it was felt that the sensitivity and the specificity of those and the cumbersomeness of that, whether it should be part of diagnostic process or not. And the consensus was while it's a useful tool, it's unlikely to be -- one of the things that Roger had in his holy grail was to be simple; it should

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useable by everybody.
The tools vary quite a bit, some are more simple than others. Some require just symptoms; some require a combination of symptoms and examination, so signs. So there was enough variability between these tools, so no one tool could be picked as the ideal tool. And I think the consensus opinion was let's not use that as one of our criteria.
DR. BRUMMETT: Did the issue of the fact that there are a number of conditions that probably haven't been deemed by the group to be neuropathic pain but would have scored high, did that come up?
DR. RAJA: No, I don't think that was an issue. Yes, Henrik?
DR. KEHLET: If we talk about acute post-operative pain, how much is neuropathic? Because it's difficult to do an operation without nerve injury? Is there a given size of the nerve that leads to neuropathic pain?
DR. RAJA: That's a good question, and you saw that I didn't put post-surgical pain as a

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neuropathic pain because I left that challenging question for Chris Wu to answer because that was part of his box. I'll let him answer first, and then maybe I can add on.
Chris, what do you think? Should that be neuropathic? How much of it is neuropathic?
DR. WU: Yes. We've had other comments of, you know, there's going to be -- for instance, whether a cancer surgery, for breast surgery, is that acute procedural pain or is that cancer pain.
So there's going to be overlap; there's no question about that. My bigger concern is what are the bigger boxes and would they fit, and then I think everything else will flow after that.
So I'm more concerned -- I don't disagree that this is an issue, but I'd rather figure out what the big overall boxes, buckets are before we figure something like this out, because obviously, there's going to be overlap not only in surgical procedures but other things like symptoms.
DR. RAJA: I agree with you. There can be an incision in surgery without cutting nerve. So

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in theoretical basis, all post-operative pain can be considered as neuropathic. On the other hand, the challenge is the differential diagnosis between an inflammatory pain state and a neuropathic pain in the immediate post-operative period.
So unless you have a big enough nerve where I can do a sensory examination and say I have a loss of sensation in this area of the ilioinguinal nerve, so it's a true neuropathic pain, I think the differential diagnosis is going to be challenging in the first week after the surgery because the symptomatology between an inflammatory pain and neuropathic pain in the post-operative period can be overlapping.
MALE SPEAKER: Raj, could you comment on your working group? I wonder -- I don't know the answer, but it's a question of using a dermatologic exam to find evidence of nerve injury when there may be nerve injury that's predominantly in other tissues that we can't examine. Has that ever come for discussion?
DR. RAJA: Give me an example. Such as?

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MALE SPEAKER: Well, you said there is zoster pain without a rash, but maybe it's a deep tissue nerve injury by the zoster that didn't predominantly affect cutaneous afferents.
So we've got pain in this distribution that's deep and parietal, or muscular, or pick your tissue, that's producing neuropathic pain that doesn't have a predominantly cutaneous manifestation. So the dermatologic exam becomes limited, as it could be with any nerve injury.
DR. RAJA: I don't think the definition specifically says a dermatological; it just says in the possible distribution. It could be either dermatological or myotomal. So if you have the distribution of the appropriate myotomes, they could still be under that criteria. I don't think there's a big conflict there. But I don't remember a specific discussion of that in the discussions.
MALE SPEAKER: Do you think there's a difference between actual direct, either peripheral or more central, nerve injury and the injury that happens to the smaller nerves with any surgical

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procedure or any cutting procedure of the skin, for example?
I mean, there is a qualitative difference, certainly clinically, in how those patients present. I mean, we know that around surgical sites, there's plenty of evidence that there's neuropathic pain in the immediate post-op period from the initial event, and it happens very quickly. But there's a qualitative and I would argue a quantitative difference long-term between a simple skin incision that didn't cut any major nerve roots, or nerve branches, and an actual direct nerve injury. And do you think that's worth characterizing?
DR. RAJA: I think it's worth characterizing. I think it depends on the time frame after the initial injury that you're looking at. I think it'd be much more challenging in the immediate post-operative period.
When the initial inflammatory component subsides, it will be a little bit easier to tease those differences out. But my gut feeling, at

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least my own personal experiences, in the few days to a week after the surgery, it's very difficult to separate which component is from a nerve injury and neuropathic nature or not.
Would you say something different Henrik?
DR. KEHLET: [Inaudible - off mic].
DR. TERMAN: Let me ask a question. So can you just tell me again, when Tim talked about pathological or pathophysiological mechanisms, he talked about nociception, inflammation, neuropathic pain.
The last line there -- that you can't see under the "thank you" -- is about is neuropathy a mechanism, or in this sort of taxonomy, would it be a disease state? What would you say about that?
DR. RAJA: When I think of mechanisms, I think a little bit more in terms of the pathophysiology in terms of changes in the transection process, changes in the amplification of that transection process at multiple levels. And things like the mediators, the inflammatory component as part of that pathophysiological

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mechanisms, I don't know how I can separate these.
That's why there is, as you can see, an expression there as to there's still some uncertainty in terms of do all of these different injuries, for example, like disc lesion causing radiculopathy, is the mechanism there the same as a zoster-causing DRG, sensory loss of neurons?
So there are some differences based on the etiology in the mechanisms, and therefore it depends on whether you're a lumper or a splitter how you work on this taxonomy.
DR. TERMAN: Thank you.
DR. RAJA: Thank you.
DR. TERMAN: Thank you, Raj.
So those of you that came back from the break should have found at your spot this piece of microfiche --
(Laughter.)
DR. TERMAN: -- written by someone much younger than me. But it will be useful for this afternoon's discussion, I believe.
The next talk is going to be by

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Paul Desjardins from Tufts Dental School on acute orofacial pain.
Presentation - Paul Desjardins
DR. DESJARDINS: Following Raj reminds me of one other time in my career where I was in a position that nobody wanted to be in, where you're competing for attention with somebody who really gives a spectacular talk.
A one-minute digression, I was invited to speak to a group of pharmacologists and pharmacists in Kansas City years ago. And we had 200 pharmacists, pharmacologists, sitting in a room, not dissimilar from the set up here. And across a paper-thin wall nobody had thought about was 100-person a cappella choir that was going to start singing at the same time as my presentation.
(Laughter.)
DR. DESJARDINS: So Raj, I thank you for not bringing in the choir with you, but following you is not much easier than following the a cappella choir.
So let me tell you, two points of view.

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Number 1, I am not a diagnostic acute pain clinician who sees patients on an everyday basis. I operate in two different silos, and the first is that I teach dentists, dental hygienists, nurses, physicians about oral diagnosis and disorders.

I teach them about clinical pharmacology, and I actually perform clinical studies. That's what I have done for the last 40 years. And one of the things that we'll think a little bit about during this time is who are the other silos that touch the type of categories that we're dealing with, and certainly who are the other practitioners who deal with orofacial pain, and how does that change how we think about it?

So where are we now? In acute orofacial pain, by and large, the categorization of those pains across the board are by anatomical structure. They're associated generally with a known diagnosis.

Most of those disorders are not treated on pain services. They're treated in primary practitioners' offices; they're treated in dental

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offices; they're treated in acute pain clinic. And one of the big differentiators that I can see, as residents present cases to me, is that we think about that pain as either primary or referred.

The challenge with a box, which is very small -- and this is the box -- is that there are a lot of players in that box, both anatomically, neurologically, and in terms of what type of practices they have.

So interestingly, the same presenting symptom on any given patient can be referred to as dental pain, toothache pain, odontalgia, or pulpitis, depending on the level of specialty and sort of which discipline sees it first and where the referrals are coming from.

If we have no idea what the diagnosis, then we resort to Latin or Greek, and we give it names like osteitis dolorosa sicca. What the hell is that? That's my oral surgery colleagues who -- that's their name -- that's the $800-name for dry socket, which can be treated pretty easily.

So one of the challenges that we have is

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that orofacial pain is a great imitator. And interestingly, some of the diagnostic features of those disorders, pain is only one piece of what they see and what they are treating.

So besides the overlapping specialties that see these disorders, why even get involved in this? Why even think about it? And the hope -- and again, I'm expressing my hope in them, hope of other colleagues with whom I've discussed this -- is that a more specific taxonomy might lead to more accurate diagnosis. It might provide more effective treatments because a bucket that's got 10 different disorders that are trying to be treated with one therapy is likely going to be unsuccessful in a number.

The other piece of this is that efficacy conclusions that need to be made by groups, whether they are pharmacologists, whether they are pain docs, whether they're medical regulators at regulatory agencies, it's very difficult to generalize across disorders right now.

We've not made a lot of progress in terms of

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how much can we generalize from proof of concept studies. A major challenge we've had is drugs have been developed in those countries; how many studies are enough? Is two in each bucket, replicate studies in each bucket enough? Is one peripheral neuropathy, is one central neuropathy enough to get a broad indication for all neuropathic pain?

So those are ongoing challenges, and having a taxonomy that helps us think more rigorously, I feel, will help us around this problem.

The other thing which I had mentioned yesterday as well is from the point of an individual practitioner, knowing who are the outliers, who are the nonresponders, and why are they the nonresponders, and maybe they didn't get categorized appropriately. But who is not going to follow that predictable trajectory is certainly one of the outcomes that I hope happens.

Now, who plays in the area? Who works with all orofacial pain? Well, depending on which resident is in the emergency room on any given night, you might see an ophthalmologist; you might

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see an otolaryngologist. There are three or four different dental specialties that work in the emergency room when somebody comes in with acute facial pain.

Plastic surgeons, facial pain clinics, certainly, the surgeons, if there's a fracture involved anywhere in the face between the ophthalmologist, the ENT, and the oral surgeons, they're either going to flip a coin or they've got an allocation system as to who's going to follow up with those patients.

So the other issue, I think, that it brings up is how do we disseminate the discussion; how do we make our conversations inclusive enough so that those other disciplines participate from the start and they buy into a change in taxonomy?

So a couple of thoughts as I've listened to some of the discussions as well, that acute pain is only of the symptoms or many disorders that show you acute pain and can we come up with common diagnostic features across the board.

A real challenge, Bob, was trying to lump

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these many different disorders that have overlapping systems to try to think about how to go through them systematically.

I believe the current drivers that I see with my residents and the attendants that I work with is that their discussion of acute pain diagnosis is pretty much limited to history of present illness and how is it presented.

They will discuss the primary symptom in terms of its anatomic region. Certainly, for that particular presentation, they give us a pretty detailed temporal pattern. They know how to probe with appropriate questions or instruments.

Certainly, a level of severity or intensity, which we've sort of poo-pooed in the past, we've got too much data in that area. At least, we collect it from the right places now; it's not just in clinical trials to look at 4-point categorical scales.

Between the attenuating factors and the other presenting signs and symptoms, my general sense is that coming into the room, that first set

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of strategies that we discussed yesterday seemed to fit reasonably well. But the hard work is going to be going through disorder by disorder or coming up with at least what we think are the lead diseases to be studied.

So the next two or three slides are simply a list of disorders of various organ systems, which show up in this bucket, which present with pain, and what are some of the other major symptoms that diagnosticians, that the treating docs are looking for.

Well, certainly, in the eye, corneal abrasions, uveitis, scleritis, not unusual. Interestingly, conjunctivitis, one of the important differentiators when they first come is, do they have evidence of infection? Is there a pink eye or not? It's as simple as that. What other types of symptoms are there?

But one of the critical pieces in working with our residents as well is what are the other types of eye pain that can show up that really portend the really serious prognosis going forward,

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whether it's an ocular motor palsy or some other cranial nerve disorder that has accompanying signs and symptoms, and where are those really red flags and the warning signs that we see.

Certainly, in the ear, they're presenting otalgia as a common presenting symptom. We understand that this represents about 35 percent of pediatric presenting chief complaints in pediatricians' offices. There can be external pain, lacerations, burn, frostbite, sunburn, all of the things that fall into the other buckets that we talked about in terms of the initiating factor.

Then a critical piece in almost everything that I could see that occurs in the ear, nose, throat, or contiguous structures, is there an infection in that area? Because all of a sudden, if it's infectious in nature, that is a great big bucket of disorders that show up as acute pain.

Then, what are the other comorbidities that go on? And interestingly enough, virtually any of the cranial nerves that innervate this area can have cranial nerve pathology that's associated with

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it.

The last quote on this slide was one made by my oral surgery colleague who teaches diagnosis in the oral surgery, "God bless those other signs." They allow you to start separating out what you're dealing with because the patient who shows up with a maxillary toothache, allegedly, may very well have a middle ear infection; they may have a sinus infection. So the differential diagnosis is pretty wide until you actually look in the ear and see what's going on.

In the nose, interestingly, rhinosinusitis, not infrequent. It sort of crosses over, Steven, to your area. How often the kids develop an upper respiratory tract infection? If you got a two or three-year-old son or grandson, chances are this year, they're going to have six infections at that age.

Again, another common presenting symptom, but they don't show up in pain clinics. They show up in pediatricians' office, they show up emergency rooms, and they show up in ENT offices.

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One of the interesting areas that pharyngitis, again, common-presenting symptom, one of the things that patients self-treat very frequently, one of the few disorders in this area that actually has a pain model associated with it. And that's where Bernie has worked, in this area for probably the last 10 or 15 years, that we actually have data on how severe is it.

It's not only how severe is it, but some of the characteristics he's put into the pain evaluation system is how much does this impair your ability to swallow. That discussion of function has been incorporated in, a large part, the assessment systems that Bernie set up.

I'm going to not discuss discussions of trauma because they pretty much fall into the discussions of post-operative -- they are the same issues in terms of post-operative pain and how that's managed.

The biggest challenge for many of those patients is that facial trauma usually is only one part of many of the other traumas that they have.

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My first year working in Newark, New Jersey, we had 365 jaw fractures on our service over a period of a year. I thought we had a busy service in Rochester when we had 30 in a year. So 350 -- last year, they had 45 in Newark, New Jersey.

Why do you think that is? The violence dropped? They don't survive their other injuries. If you've got five bullet holes in you, a jaw fracture is the least of your problems. There are some reasons a few of us sort of take gun violence seriously.

Disorders that present in the mouth, and teeth, and jaws have a pretty wide list of issues. There is pulpitis, which interestingly enough, the question I ask is, does that really fit into the acute visceral pain type of bucket? Because it is a compression type of injury in a very confined space, and it can be -- among all the dental pains I've seen, that's the most excruciating, have people sort of on their knees, and all of a sudden, you give them a local anesthetic, it's like you've

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hit them with a magical serum.

Periodontal abscesses, caries, impacted teeth, and pericoronitis, just a little touch, and I'll bring one of my other pet biases in. Oral surgery, post-oral surgery pain of the third molar, we've learned more about acute post-operative pain and how to standardize it than any other model. We took the learning from what happened when you extract patients' third molars, and we have extended that to post-operative pain. And bunionectomy pain model was spawned by dental impaction pain model.

Acute TMJ pain is interesting because now we're dealing with a musculoskeletal problem. And we're dealing with patients that may have excruciating pain for just a short period of time, but it's amazing how muscle exercises and cold and resolve those types of issues.

Atypical odontalgia are the type of things from chronic pain clinics that I have seen and would not believe, where patients will have a trigger point which is a millimeter or two wide,

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where patients are having severe pain. You can put a local anesthetic on it, and their pain resolves, immediately, immediately.

What the hell is going on there? A very focal neuropathic type of disorder? I don't know. But there's somebody that has to treat them, and they don't come into the pain clinics most of the time.

Certainly, one of the concerns -- and we see them frequently -- are patients who have myocardial ischemia that show up as jaw pain or tooth pain. So there certainly are other comorbidities that sit out there.

I think my next couple of slides on the dimensions are a bit like -- sort of a repetition of what's been discussed by the other speakers, so I'm just going to go through them very quickly, that they generally seem to fit. Those dimensions fit reasonably well. But the hard work, I think, is going to have to be done by groups that we put together, which are going to involve individuals that treat those disorders and who understand the

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underlying pathology and similar diagnostic criteria.

Dimensions 2 and 3, one of the critical areas in the facial pain literature has to do with, is this an infectious etiology; is it an inflammatory etiology; or is it referred pain from adjacent structures?

So how best to put that into our taxonomy, I think, was part of what we were wrestling with yesterday about where to put them. And it took me a day to digest the discussion of yesterday afternoon because it wasn't easy for me to think about to think about 15 disorders and how to bucket them easily.

The psychological and emotional component, perhaps in many of these, they're sort of short-duration disorders that I've discussed with you, medically-managed by and large. And while there can be emotional impact, it's frequently not critical to the initial diagnosis that I've seen.

But how do I know that there are the psychological impacts? We've had people we've

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brought to the university to talk about acupuncture. And all of a sudden, I've had a room full of docs, a room full of nurses sitting there and they show craniotomies being down with acupuncture. And all the docs are like, hmm, that's interesting. Really? You can do that?

Then we'll show a delivery, a cesarean section with acupuncture anesthesia. That's interesting. Then they say, now, we're going to show you a tooth extraction? The whole audience goes, "Oh, my God. How can you show that?" Well, you think maybe they're carrying a little bit of their previous traumas into the room?

The functional consequences, I think, are a rich area of research, which has by and large been ignored. Interestingly, a few models have actually included a few of the systematic evaluations in acute pain. So a throat pain, dental impaction pain, they've collected information on function. But how were they considered by regulators today?

Secondary measures, we're not primarily interested in that. We're not going to approve

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your drug based on whether you feel better or walk better. It's do you eliminate the pain?

So that's an ongoing conversation. And why do you think pain intensity sits in those clinical trials as the primary measures of efficacy? Because it's a negotiation with regulatory agencies as to which outcome is most important.

The conversation, which has existed for at least the last 32 years, that I'm aware of, is pain and pain intensity has been reproducible enough, and we know how other drugs -- it comes down to creating a level playing field for new drug developers or device developers. But unless this group can also convince and wrap their arms around the regulators and the type of data they're asking for, we're going to have more of the same when we get together five years from now.

One of the key questions -- and again, this has been an ongoing question. I don't know how much this effort is going to be able to contribute to this. But can we, in different categories, come up with one or two generalizable models that could

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be developed for a given bucket?
I can't tell you how many times I've spoken to groups of clinicians. They say, what do you mean there's really not many clinical trials dealing with this particular type of orofacial pain, whether it's TMD, whether it's -- I don't know -- dry mouth or burning mouth, how could there not be clinical trials in that area? Because it's so difficult. It's hard to find enough patients and agree on the measures and do it.
So my hope is that, over time, that the learning from other models from this group that's talking about the taxonomy will actually help us in generalizing to other similar conditions. And I raise the question before, is dental pain -- is pulpitis, inflammation of a dental pulp, what are the common characteristics between that and other visceral pains? Because, quite honestly, we don't see into the tooth any better than you see into the belly, and probably less so.
What are some of the things that I think would help in improving acute pain study designs?

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Baseline pain determinants, the natural course of the history, we've talked about the trajectory, what's normal?
We turn to Henrik to sort of talk to us about what are the type of interventions that help, but what is the standard by which we compare the future interventions?
What about those surgical characteristics? It's interesting. I have spent hours in talks debunking myths; oh, dental pain, that's just the pain of inflammation. So wait a minute. We remove bone on every single case. We do a skin incision in every single case. In order to get those tooth fragments out, we cause a crushing injury to the periodontal ligament, and then we leave the hole in the jaw open.
So you tell me, is that soft tissue pain? Is that bone pain? Is that neuropathic pain? Yeah, we cause nerve injuries; 2 or 3 percent of patients have paresthesias that happen after surgery. But fortunately, they resolve in the next month. What about the 1 percent of those cases

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that go on to have permanent paresthesias and post-operative pain?
Among the other issues, it's interesting that I talk to many anesthesiologists, and they talk about multimodal forms of anesthesia. How do pharmacologists look at this and how do regulators look at it? Concomitant therapy. They confuse the decision about whether this drug works. Yet, when I speak to my oral surgery colleagues, what do we know beyond a shadow of a doubt?
In third-molar surgery, if you give somebody a long-acting local anesthetic block with carbocaine and you can keep it numb for 5 to 8 hours, and you give them a pre-operative corticosteroid, their pain course is smooth, smooth. I mean, those patients are absolutely comfortable. You don't get calls on the weekend and at night. But if you go back and look for the data, you've really spent a lot of time finding studies that really document how effective are they. So we resort to meta-analyses that say, yeah, it seems to work.

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Ongoing issues in terms of drug -- so where would you like to be? Where would I like to be? Interestingly, we had several opportunities where we studied essentially the same drugs with identical outcomes, looking at pain intensity over 8 hours, where we could generate an effect size.
Now, for those of you who don't do biostatistics frequently, the effect size was just the size of the -- the mean effect of the drug, subtract out the placebo. So that gives you the signal. And you divide it by the pool standard deviation, sort of a signal-to-noise ratio.
Interestingly, we hypothesized early on in four studies that we did, bunionectomy pain for the same dose of drug with ibuprofen, used in both models, that you'd get about 65 percent of the bang out of the buck when you did foot surgery and gave it to them post-operatively.
Well, lo and behold, when we went back and looked at other nonsteroidal drugs and Cox 2 inhibitors, and you looked at the magnitude of effect that were in reported studies after

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bunionectomy surgery, after orthopedic surgery where they knee and hip replacements, well, lo and behold, that same effect size is what you saw across other pain models.

Wouldn't it be nice to be able to do the same thing with other acute pain models with what you're dealing now?

So last thought, and then I realize I'm standing between you and one speaker and lunch, I ask myself, as we go through this multiple times, are we just a hostage to our own specialties and disciplines?

How are we going to diffuse the information across the boxes that deal with this area? How do we best help tomorrow's clinicians and investigators understand the problem?

I honestly think that answers that have as much detail and insight as some of the presentations I heard today are going to lead to that. Can it give us more accurate, more simple -- I don't know about simple, how likely. More predictable recovery is the hope?

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The last slide reminds me that although we've been talking about orofacial pain for the last 20 minutes, the face is an area that gives and receives so much pleasure.

Okay. Questions?

(Applause.)

DR. DESJARDINS: Good. Any questions? No? Raj?

DR. RAJA: Quick question. Should pain from -- ophthalmological pain, would that come under orofacial? It's still part of the face.

DR. DESJARDINS: Yeah. I think as we think about it, my sense is if that pain is mediated through the fifth cranial nerve, yeah, I think about it as orofacial pain.

Those individuals are not usually sitting in most of our meetings, but absolutely, eye, ear, nose, throat, mouth, I think they're commonly related and they have commonly referred pain between them.

Fortunately, for many of the ones that I show, they're diagnostically pretty easy to pick up

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as where the problem is. But I think eye pain is another one that sort of fits into this bucket very easily.

Okay? Good. Thank you.

DR. TERMAN: So the last speaker will be Steve Weisman from the Medical College of Wisconsin. Talk about a far ranging topic he was assigned;, pediatric, geriatric and special populations on acute pain.

Presentation – Steven Weisman

DR. WEISMAN: Twenty minutes, huh? Actually, I'll get done ever more quickly. But I'd like to start with something special.

(Music plays.)

DR. WEISMAN: Who is that?

MALE SPEAKER: The 5th Dimension.

DR. WEISMAN: Right, the 5th Dimension.

(Laughter.)

DR. WEISMAN: I can't get that out of my head ever since we started talking about this.

(Laughter.)

DR. WEISMAN: I looked. There isn't a 1st

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to a 4th dimension in the music world, but there is a 5th Dimension. Let me get my technology organized.

MALE SPEAKER: Just start singing, Steve.

DR. WEISMAN: No, no, no. We'll go to lunch if I start singing.

I took a different approach to this, a very simple one because the topic is much too broad. And I thought that, really, what our main purpose these two days is in fact to wax philosophical in terms of coming up with our overriding schema.

So instead of focusing in on, for example, the unique pain in a baby having a thoracotomy for a TEF repair, or the uniqueness of a newborn getting repetitive heel sticks and what that does, I just want us to think in more generalities about things.

Since Greg strong-armed me to be the head of the Ethics Committee on APS, these are my disclosures. God forbid we ever give a talk without it. And just to remind you, many of you didn't.

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What I'd like to do is first start out and get us to think about what are the special populations that we ought to be thinking about, and what are some of the differences that members of these populations bring to the table when we're thinking about creating a taxonomy? And then I did try to frame it in my current version of what the fifth dimension, the 5 dimensions ought to be.

So certainly, neonates and infants are a population unto themselves. They are physiologically very different. Their nervous system is different. How they respond to pain is different.

Children and adolescents become more mainstream, if you will. Seven to eight percent of the patients taken care of in my hospital or Suresh's hospital are developmentally delayed and by definition have had normal nervous systems. And where do they fit in the acute pain spectrum?

Geriatric patients clearly begin the decline, and their nervous systems are very different. How they respond to acute pain is often

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very different. Many of them actually, I might argue, are maybe somewhat protected in a different way than we use to think infants and neonates were protected from acute pain.

I almost never give a talk about acute pain without acknowledging the trauma that I caused in babies and children. You know, it's sort of like revealing that you are a Nazi, which is that when I learned to put my first chest tube into a baby for a pneumothorax in the newborn period, we did it without anything except the trocar. And that was standard practice, really, in pediatrics until the '80s and '90s; really unbelievable stuff.

I tell this story, too, actually. I was a second-year pediatric resident, and the revered Dr. C. Everett Koop was dropping off a baby in the newborn intensive care unit at Children's Hospital of Philadelphia, to the erstwhile second-year pediatric resident.

I looked at him and I said, "So Dr. Koop, by the way, what kind of anesthesia do these babies get?" And he looked at me and he said,

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"Pancuronium, oxygen, and a darn good surgeon." and he turned around and he walked away. And he was a very scary guy.

(Laughter.)

DR. WEISMAN: And I turned to his fellow and I said, you know, "Seriously, Victor? Is he shitting me?" And he said, "No."

That was like my first eye bubble, my first like, Oh, my God. Seriously? The baby had a lateral thoracotomy and only was paralyzed. And at any rate, there, I've gotten my guilt off the table.

We shouldn't leave out pregnancy and breastfeeding. There are issues related to how the pregnant woman or the becoming ex-pregnant woman deals with acute pain issues that are worth thinking about when we develop a taxonomy.

I don't know that -- in terms of studies, there are lots of pharmacologic issues related to studying potentially breastfeeding women, which are very challenging. But I don't know that that really needs to be part of a taxonomy.

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Critically-ill patients, I think, need to be thought about as a group. And as we develop a taxonomy, how do we define the acute pain disorders that we can apply to these patients, who much like the smallest of our patients or some of our oldest patients are unable to do self-report?

So do we need a very strict, rigid taxonomy that defines a lot of the acute pain disorders that remove self-report from the taxonomy?

Substance-abusing patients in the acute pain world are a challenge; we all know that. We heard a little bit about -- well, indirectly -- about the war-injured patients and the meaning of their injuries and how they manage acute pain.

It's certainly staid by now and old, but I used to use the example of Forrest Gump in the pain clinic. Do people remember what happened to Forrest Gump in the movie that would be important for acute pain?

MALE SPEAKER: At which point?

DR. WEISMAN: Pardon?

MALE SPEAKER: At which point?

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DR. WEISMAN: Well, when he was a young adult.
MALE SPEAKER: He got shot.
DR. WEISMAN: He got shot. And does everyone remember what he did when he got shot? He rescued Bubba. He carried like a dude bigger than me to safety. Then when he got to safety, one of my favorite lines in the movie was, "Oh, my goodness. I've been shot in the but-tocks."
(Laughter.)
DR. WEISMAN: And he, as a warrior, as a soldier, managed his acute pain very differently. We know this, that a lot of the immediate acute pain responses of the war vets are very different than a lot of the other patients that we deal with. Do we need to consider that somehow in our taxonomy?
Then last, we have an obligation, I think, for the other linguistic barrier, in addition to development or decline in development, our immigrants and non-native speaking patients who may not necessarily be able to communicate with us

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about the taxonomy of their pain problem and how do we account for that when we're thinking about developing this taxonomy.
So these were the proposed dimensions last evening, so I just ran off of them in terms of some my other thoughts. As I've already said, many of the populations that I just went over cannot rely on -- we cannot rely on self-report to define their pain problem.
So should we think about a taxonomy that really is more event or etiology-defined as the first level of looking at the taxonomy?
Then, there's the challenge of -- we could say things about needle pain. So as another example, if you look at the burden of cancer treatment in children -- and this is very old psychological stuff, if you will, that was studied in the '70s and '80s. And you query young survivors of cancer about the burden of cancer, what they will tell you is the single most burdensome thing about their treatment were all the needles they got, whereas many of us in the room

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will accept needle puncture and give it a very low priority in terms of the acute trauma, if you will, that it's causing. And that's an important distinction.
When you look at needle trauma in babies, for example, there are good data that show that those preemies who get repetitive heel sticks develop a neuropathic pain syndrome with hyperalgesia and allodynia, if you will, in their foot in a wide distribution from getting repetitive blood draws done as part of their care.
So the significance of some of the simplest procedures that we think about with procedure pain are very different. When we think about spine surgery in our patient population, being the pediatric population, what, again, Suresh and I do is we don't do single disc fusions. We take care of patients who have spine fusions from T2 to L4, and have an incision that's literally that big with incredible degrees of bone destruction and an enormous amount of muscle and fascial tissue damage. Is that different from a single level

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spine fusion?
In a patient who comes in to the operating room without any pain before, as opposed to the single-level fusion patient who's probably had significant debilitating radicular pain for a long period of time, they're very different patients; they really are. And their acute post-operative recovery is remarkably different, and how do we think about that when we develop our taxonomy?
If you think about -- at least, this is how I was starting to think about things. As we look, if we focus in on Dimension 1, we could develop a taxonomy in some form like this.
I mean I'm digressing a little bit from the special populations, but one in which we look at acute illnesses, we look at trauma, we look at surgery, we look at procedures, diseases. Although as nice as Knox's talk was about cancer, when I think about it, I don't know what cancer pain is.
I mean cancer pain is trauma to a nerve. Cancer pain maybe is treatment-related. In fact, again, if you look at children, the real burden

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besides their needle pain is treatment-related pain that comes from mucositis, or neuropathy, or the local destructive effects of radiation, et cetera.

So really, I think we are pandering, if you will, the pain community by repetitively talking about specifically cancer pain as if it's something different.

Then we've avoided ischemic pain, but I still think that that's an important topic. When I think about sickle cell pain, most of it is ischemic pain from small blood vessels being blocked by the abnormal cells leading to local ischemic reactivity in that area, much the same way sometimes compartment syndromes are, much the same way maybe that some cardiac pain is, and do we need to think about that as well?

That was, at least, my first shot at coming up with one way to maybe to start to look at the taxonomy and then put disease categories within each of those potential groups.

So again, pain assessment, which is always an integral part, if you will, of defining acute

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pain or chronic pain is a challenge in almost all these populations. And fortunately, with our younger patients especially, we have good validated tools that we can use and apply. So I think this can be incorporated into a taxonomy, if we think it's important.

But the issue of perceptions and then communication of that is impossible. You cannot get, with any reliability, a 6-, 7-, 8-, 9-, 10-year-old to distinguish some of the features of neuropathic pain when you're interviewing them in the course of trying to define what their pain cause might be.

The extremes of age, again, have individual responses to pain that are different, and I don't know how we're going to take that into account. And finally, the cultural differences, much like the soldier's response to an acute injury, can be very different than a civilian who sustains a similar injury. There are a lot of cultural implications that we really haven't touched on at all but I think are worth thinking about.

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In terms of Dimension 2, host and risk factors, some of these patients may have different responses. You don't see the same cardiovascular, for example, variability in neonates and preemies with significant acute pain stimuli. You just don't see it, so you can't rely on that.

We certainly have to factor in all comorbidities in the elderly, prior pain experience and then the multitude of underlying diseases. And I don't know how in our taxonomy, we'll actually incorporate the neuropsychobiology of this stuff.

We've spent, actually, a lot of time talking about that, and I think it's important to build it into the schema that we come up with because I do believe that it's just as important in acute pain as it is in chronic pain.

Pain quality, it's interesting that when a different group of folks just like those assembled today met to go through the original pediatric IMMPACT meeting, we didn't consider pain quality as one of the domains to be measured because of the challenge of the unreliability of coming up with

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any meaningful data in that category.

Again, I've already alluded to in these special populations, language, age, being in the ICU, being very young is going to prevent us from categorizing and characterizing a lot of the disorders that we'd like to.

Environmental context, again, I touched on that. The perception of acute pain in labor is very different when you produce a young healthy infant or your expectation is to produce a young healthy infant. It's very different than other types of acute pelvic pain.

Even the meaning of pain in the immigrant/refugee or even war scenario, an injury gets you away from hell and what's the significance and the meaning of that, I think that's going to be very hard to factor in.

Pathophysiology may be largely similar across all these populations. But having said that, we know the physiological impact of some acute pain, diseases or causes, are likely very different in these different populations.

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We know from the original work of Sunny Anand and Paul Hickey that poorly managed pain in babies undergoing cardiovascular surgery doesn't just lead to a poor Picker rating when they leave the hospital; it leads to death. And the morbidity in babies undergoing heart surgery, who back in the dark Dark Ages were managed with non-opioid-based analgesic regiments, resulted in higher rates of mortality in the hospital.

So it isn't just a quality issue. And we know the implication from lots of work done on elderly with hip fractures that a good pain management and addressing that makes a really big difference in outcomes, really big difference.

In terms of function, this is also going to be another challenge because a lot of the populations I mentioned begin at various levels of functionality, ranging from the very elderly, who may be debilitated by some of their comorbidities, to the common patients, again, I might take care of who have spastic quadriplegia from a major devastating brain injury.

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When you look at simple things, if you're thinking even in the acute arena of potential outcome measures, babies have very different sleep cycles. The elderly have very different sleep cycles than the rest of the population.

Remember that -- this is always hard -- 22 percent of the American population is under the age of 18; 17 percent of the American population is somewhere between 65, 75, and older. So that's almost half of the American population even though there's just a small cohort of us here representing them. Forty percent of the folks we need to work with are in these special population groups, so they're an insignificant group to think about.

When I thought about the cognitively-different populations, again, Dimension 1 is going to be very difficult to define if we rely as it traditionally was done in chronic pain and if we move forward with this in terms of descriptors et cetera. The epidemiology and obviously specific tissue injury element should be pretty easy to characterize.

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Dimension 3 may be near impossible in terms of we never can get a qualitative assessment from a lot of these patients, and we almost never get temporal features at all. And if we do get them, secondarily they're from family caregivers who are supposing that the pain problem started a certain time ago.

The other dimensions I think are relatively similar. And again, functionality will be a difficult domain to define.

I think that we shouldn't let this get in the way of us defining this taxonomy. But as long as we can do our best as we think through it to pay attention to these factors, I think we'll end up with a useful taxonomy that we can apply to acute pain. Thanks.

(Applause.)

FEMALE SPEAKER: Thanks for taking on this huge group. I was just wondering when we were talking about the different dimensions, one of them was host and sort of risk and protective factors. I wonder if a certain slice of this sort of special

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population that you described could be absorbed into that.

So for example, like an adolescent having surgery may not be quite as different from an adult having surgery, so they could be absorbed into the post-surgical acute pain with the caveat that their host -- that they have certain risk factors or social characteristics, so that your slice of the pie is not as big; and similarly on the older age, if you know what I'm saying.

DR. WEISMAN: No, I do. I think the overriding goal, I still see us coming up with that structure. Then, we can create whatever caveats are essential for some of these special populations, for example. I think that's really the only way to approach this.

Okay, lunch, I guess.

DR. TERMAN: All right. Well, hopefully, some discussion will continue over lunch, and we'll see you back here at 1:30.

(Whereupon, at 12:27 p.m., a lunch recess was taken.)

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A F T E R N O O N S E S S I O N
(1:33 p.m.)
DR. DWORKIN: Mike, and Patrick, and Steve, please come up to the podium.
(Pause.)
DR. DWORKIN: We're just going to wait one more second for Pat.
(Pause.)
DR. DWORKIN: So before we start, everyone should have in front of them -- because it's what we're going to be discussing this afternoon -- this one-page, very small font flowchart prepared by Mike.
If you don't have that, try and find because it's really critical for the next 15 minutes to 3 hours, depending on how long it takes us.
(Laughter.)
DR. DWORKIN: We're waiting for Pat because he's obviously a critical person here. If he doesn't show up in 30 seconds, we'll start without him.
DR. COHEN: Would this be a time to ask a

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question?
DR. DWORKIN: Sure. Go ahead, Bob.
Pat just arrived so --
DR. COHEN: Can I ask a question --
DR. DWORKIN: Yes, you can ask the question you were about to ask now or later?
Now that everybody is here, I promise that we will end by no later than 4 o'clock, but we can end sooner. It depends whether we achieve what we need to achieve in this final session.
The way, I think those who've been involved in setting up this meeting think about this final session, in a kind of very straightforward, simple-minded way, is making sure that Pat and Mike have the raw material that they need to draft the manuscript that will be a kind of acute pain version of the manuscript that you've all carefully read, the lead author of which was Roger for the chronic pain.
So really, our criterion for success this afternoon is have we agreed on -- consensus -- on the raw material that Patrick and Michael used to

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draft the manuscript.
So that's the objective for the rest of this afternoon. You're all going to be receiving that manuscript. Some of you know this from other meetings that we've had. You'll be receiving a draft of that manuscript. They'll be comments on it, revisions, and it doesn't get submitted for publication until everybody thinks it looks great. Being author on it is optional.
If you decide you don't want to be an author, for some reason, on the manuscript, that's entirely up to you. But everyone who's at this meeting is offered authorship.
Steve wanted to lead with some opening comments, and then we'll kind of throw the meeting open to all of you to discuss that flowchart that you hopefully have in front of you and really a discussion of, as I view it, does the flowchart in front of you look like essentially the scaffolding of a reasonable manuscript that we would all co-author and submit for publication in Journal of Pain and Pain Medicine.

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So Steve, why don't you take it away?
Discussion and Approaches
DR. BRUEHL: Thanks, Bob.
All right. What I wanted to do is to talk about what we did yesterday and show some transformation that had happened, which hopefully will be clear that we haven't really changed anything at all.
Then, we're finished with that, I want to bring up the AAAPT chronic pain criteria and just kind of show you what similarities and differences there are.
So first thing I'm going to do -- so what I took was yesterday being kind of settled on this model that included the following elements: risk factors, event, host, environment, pathophysiology and impact. That was from the epidemiological model. That was what we had in there. We used that to organize some of the specific things that were brought up before. All I've done is collapsed categories and renamed them, but the essence of the construct is the same.

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So here, in our list of things we wanted to make sure were covered included event -- which I will check off as we go through this. We got the event done; degree and extent of tissue damage, that's here; organs, tissues and symptoms. So that was considered to be very important. So that's there. That's here.

Now, we had temporal trajectory and that is now under this Dimension 2, whatever we call it. But you got temporal pattern, typical trajectory. So we've got both those pieces of information there.

MALE SPEAKER: Can I ask a question? [Inaudible - off mic].

DR. BRUEHL: Yes.

MALE SPEAKER: You know, I'm looking on this as you're going, so --

DR. BRUEHL: I'm sorry that -- the small thing -- there's a handout. Everything on here is in the box.

MALE SPEAKER: Right. What you're pointing at and what we're looking at here are intended to

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be the same.

DR. BRUEHL: Similar.

MALE SPEAKER: The same boxes?

DR. BRUEHL: Yes, very similar, yes. This was done a little before we made a couple of revisions.

MALE SPEAKER: Yes.

DR. BRUEHL: But it should be pretty similar.

So we've got the temporal trajectory, the trajectory questions that had to do with chronic pain risk -- it's like how risky is this person to develop chronic pain -- are now going to be under the modulating factors, which will be factors affecting chronic pain risk in that acute pain context.

Pain qualities, this is now under Dimension 2, the common features or associated features. Very specifically, it includes temporal patterns, spatial and pain qualities themselves.

Now, we have something called modulating conditions; and context, the psychosocial, legal,

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work-related factors, what Dan ended up referring to as the milieu, right?

So all of that -- did I say that well? Good.

FEMALE SPEAKER: Is that what we call host?

DR. BRUEHL: What? That was what we -- yes. That's what we were calling host before, which was like person-centered things that now are collapsed under this modulating factors.

Bob had said "moderating." Somebody pointed out that "modulating" may get -- that "moderating" in one sense can mean to reduce, to reduce something. "Modulating" may be a more appropriate word. But that includes psychosocial factors, which we get at the catastrophizing, anxiety, depression, trauma.

MALE SPEAKER: Over lunch, I thought about -- I think I kind of propose a different heading for 3 because "modulating" also suggests diminishing. How about "relevant host factors" for 3?

DR. BRUEHL: I like that in a sense, but I

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am also concerned that people who weren't in this room to hear the context of that when they see "host" are just going to drop their jaw and go, "Huh?"

MALE SPEAKER: Think about the -- this is the "risk protective," and the only thing about risk obviously is we're also talking about things that if it modulates after it happens, not necessarily just cause it to be there or not [inaudible – off mic]. So modulating or risk or protective.

DR. BRUEHL: Yes, I put --

MALE SPEAKER: In some way communicate to people who are just seeing this the first time, what causes pain to be worse or better --

DR. BRUEHL: Exactly. Exactly. Or progress, yeah.

MALE SPEAKER: How do we just convey that --

DR. BRUEHL: I don't know what the -- yes.

DR. SURESH: So instead of calling it "modulating," why don't you call it "modifiers"?

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DR. BRUEHL: Modifying factors?
DR. SURESH: Modifiers.
FEMALE SPEAKER: Modifiers.
DR. SURESH: Then it becomes risk --
DR. BRUEHL: Okay. I'll write it down as an option. These are all things we could probably resolve easier in an email to let everybody vote on it officially.
DR. DWORKIN: The most straightforward way of doing it is that Mike and Pat make a decision and put it in the manuscript, and we all endorse it or object to it. So we can leave some of these terminology challenges up to the two of them to decide during the drafting process. You guys are up to it, right?
DR. TIGHE: Yes.
DR. DWORKIN: Good.
DR. TIGHE: And I also would like to say I think there's certainly room for discussion on the terminology used. But the fundamental concepts on each of these dimensions that Steve has shown is there is general consensus that these concepts are

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the important domains we'd like to cover. Or do we think that we need to step back before we get to the terminology and look at a reconsideration of the domains as headers?
FEMALE SPEAKER: I think it's helpful to go through -- just as we are now -- and decide what it is that we're talking about before we can totally answer that, if that makes sense.
DR. BRUEHL: Well, at the end of this, let's ask that question again.
(Laughter.)
FEMALE SPEAKER: Yes.
DR. BRUEHL: So we got -- now calling modulating, modifying, whatever that is, but we're talking about, yes, those things that increase risk, increase severity or reduce risk or severity. That could be physiological; it could be psychological. That would include, in my mind, things even like family factors and that kind of thing.
MALE SPEAKER: Social meaning but [inaudible - off mic] --

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DR. BRUEHL: Yeah, any context issues, yeah, like --
DR. WEISMAN: I know the synonym would be adaptive.
DR. BRUEHL: Well, not necessarily. It's also dysfunctional. I mean, it depends on the direction we're talking about. But it would include adaptive factors for sure.
Context is subsumed under that Dimension 3 we just talked about. That would also include things like I-hate-my-work; that is a context factor that would be under there. Having litigation potentially could go under there. So it's a nice placeholder for all these other not direct disease-related factors that may make it better or worse.
Previous pain experiences also would go -- because that was something that was brought up yesterday. Taking high dose opioids at the time that the injury occurs can affect pain afterwards. All of that could go under this particular category because they are all modifying factors.

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FEMALE SPEAKER: Age? What's that? How about age?
DR. BRUEHL: Yes, okay. So age would be a modifying factor also, yes. It sure would. Gender, in some cases, maybe. I don't know.
Roger? Functional interference and impact, that --
MALE SPEAKER: Would genetics go there?
DR. BRUEHL: Yes, I would say so. Sure.
MALE SPEAKER: [Inaudible – off mic]
DR. BRUEHL: Yes, I think that would go under Dimension 3 also, genetics, because that would clearly be something that would modify the person's response.
MALE SPEAKER: Did you mean environmental or did you mean [inaudible – off mic]?
DR. BRUEHL: Or did I -- oh, that was just -- yes, no, I don't -- we'll worry about that later. But yes, it was not intended to refer literally to the --
MALE SPEAKER: I just want to make sure --
DR. BRUEHL: -- pollution or anything. Yes,

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social context maybe. Okay.
Functional interference and impact is its own dimension here, so it's Dimension 4. Let's see. We've got all this already taken care of.
Now, response to initial pain treatment, I'm thinking that is something that probably would fit under modifying factor also, because as soon that the person has already been injured, has acute pain when you see them, and you're making a diagnosis, and you find out that they've had high-dose opioids and have not had any response to that; just as an example, that would be something you might list there.
Roger?
DR. FILLINGIM: You know, in some ways, you could think of that as a method, not whatever the proper term in the ontology is. And that might be a method to discern putative pain mechanisms rather than modifying factors.
DR. BRUEHL: Yes. That's certainly a consideration. I'll add that to here because that was brought up yesterday, too, so the treatment

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response or lack thereof --
MALE SPEAKER: [Inaudible - off mic]. I think it wouldn't be a mechanism but just a way of determining -- [inaudible – off mic]. So I think Roger is saying it's separate from that.
DR. TIGHE: So when we look at things you can do to pain, we generally diagnose and treat in general. And don't we usually traditionally have thought of diagnostic tests? So we do imaging, biochemical assays, et cetera.
Those treatments, just like our diagnoses, can serve as treatment, as diagnostic tool. So that would actually fit into multiple headings across multiple domains.
DR. BRUEHL: It could. The one thing I'm thinking of is by putting it here explicitly in Dimension 5, one thing that does -- which I think is probably appropriate -- is we don't know enough about what drives treatment responsiveness or non-responsiveness to use that in Dimension 1 as a core criterion.
So I think putting it here as a way of

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inferring something about mechanisms would make a lot of sense.
DR. TIGHE: But wouldn't this manuscript -- would it be fair to say that it's one of the more direct linkages that we would have towards mechanisms, because I know there's been a lot of --
There was some concern about making that revolutionary step in the first draft but this would be an example where we'd say we did a treatment; we saw a response. We may not know the finite mechanism, but we have some insight into it, whatever it is, as far as the patient is concern, and their response was.
MALE SPEAKER: I just -- to me, that box seems like what causes the acute pain disorder, what is the pathobiology, what is the physiology. And I just don't understand why treatment would be in there if it's a way to discern the mechanism or biology but it's not actually part of the biology itself, the physiology itself.
Like I might ask someone about the meaning

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of their illness as a way of determining something about box 3, but I wouldn't say that that is part of box 3.
DR. BRUEHL: No. And that may be very specific to the types of the treatment --
MALE SPEAKER: So does it capture your point if the phrase "response to therapeutic trial" would be in there somewhere? Is that the point you were getting at?
DR. BRUEHL: With the distinction being drugs were involved as opposed to just an interview?
MALE SPEAKER: Well, just the first draft is just response to therapeutic trial, if that captures the concept that's being raised.
DR. DWORKIN: So we certainly didn't include anything about treatment response for the chronic pain diagnostic criteria.
So I'd love to hear an example of how treatment response really illuminates the patient's diagnosis, because as was just said, I think there's very imperfect association between the

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existing drugs we have and underlying pathophysiologic mechanisms.
So I just can't think of a good example of where treatment response really illuminates these issues.
MALE SPEAKER: Can I suggest from Paul's --
MALE SPEAKER: What?
MALE SPEAKER: Just fresh in mind is Paul's talk where there's a tiny area that triggers paresthesias if touched just --
DR. DWORKIN: Well, is that treatment response or is that just physical exam?
MALE SPEAKER: No, I think that was treatment -- that was the treatment --
MALE SPEAKER: That was the --
MALE SPEAKER: He talked about infiltrating.
MALE SPEAKER: But it's still at the means of determining mechanism. It's not a mechanism itself.
MALE SPEAKER: Right. Right.
MALE SPEAKER: Like for that conceptual box to be the mechanism box, why would we put a

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treatment trial in that box?
DR. BRUEHL: The keyword is --
MALE SPEAKER: I just wonder what -- it's the physiology of the disorder; it's not about how I determine that physiology, just like a question about meaning is not about --
DR. BRUEHL: So it's methods -- you're talking about is it a method or is it a concept? Yes.
DR. FILLINGIM: Yes. And I think what Mike and Patrick can do when they're -- so Sam's right. That is not a putative pain mechanism. It is a method, a potential test that one might use to provide information about putative mechanisms. And Mike and Patrick can describe that, however.
But central sensitization or excessive activation of this channel or nerve damage, those are mechanisms. I don't think we want to list methods that are used to assess the mechanisms in the actual dimensions. You can provide information about that as you write the text.
DR. BRUEHL: I will use a different example

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there. So the concept we're interested in, that's actually the mechanism, the central sensitization, what we might do is a temporal summation about pain protocol. That is the test for it.
So we wouldn't put temporal summation protocol as Dimension 5. We would say that is how you would assess it.
I think in the drug trial, what would be relevant would be, for example, you have a particular condition you get that you think is inflammatory. You give anti-TNF drug, and they don't respond. Can you infer from that, then, that certain inflammatory pathways may not be active in that particular patient? That would be one way I might think about using that as a method.
DR. TIGHE: Because treatments and diagnoses and any other methodologies have not been considered before, would it be worth rolling that into a stage 2 project so that could be done in conjunction with the chronic pain development, so that we'd be developing an approach to methods --
DR. BRUEHL: Yes.

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DR. TIGHE: -- in conjunction with others rather than have it --
DR. BRUEHL: Yes.
DR. TIGHE: Would that be reasonable?
MALE SPEAKER: That makes sense.
DR. DWORKIN: My recollection is that Rob Edwards' paper on phenotyping for chronic pain trials, which is now in press in Pain has pharmacologic challenge as an example of one approach to phenotyping. And so this would be consistent with that.
DR. BRUEHL: Right.
Back to where we were, just to finish this up, so we did previous experience with the same condition or treatment. That was brought up I think under the Dimension 3 when we're talking about medical or history factors that are relevant, not only with the opioids but prior pain conditions. And I suppose in that context, you could say something, if it was relevant, about prior treatment or prior response. But there is a place, at least, to put that on there.

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Now, I will say what we don't really have covered, explicitly that is, on the original AAPT, are conditions and comorbidities, which is a separate dimension in the chronic pain taxonomy.

Now, my argument for why that might be appropriate is that in a chronic pain setting, it was ongoing for a very long time. There's more opportunity for all these comorbidities to be present that may or may not directly impact on the condition.

What we've done differently in Dimension 1, compared to the chronic pain -- because, in many cases, really, the only sign or symptom you have is the person reports pain, and there may not be much else to tie it to.

To distinguish between conditions, we are having to rely on a description of the location, the organ system, the tissue, and the disease process if there is one. So we've kind of already talked about the most relevant comorbidities like here in Dimension 1, explicitly as part of the diagnosis because those are the ones that are

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really relevant to how that person presents right then.

I like leaving it that way if everybody's okay with it, just because it keeps this at 5 dimensions, which is kind of paralleling what we've got with the other one. The more you've got, the more unwieldy it gets. And we still are capturing relevant, the most relevant comorbidities and conditions in Dimension 1. That is maybe open for debate, but I think that makes some sense.

MALE SPEAKER: Can it go in Dimension 3 then?

DR. BRUEHL: Yes, I mean, I think you could squeeze it in other places like that.

MALE SPEAKER: Dimension 2, like at common features, it could be -- like for my world of -- let's say a rape survivor with acute pain, a common feature would be acute stress disorder symptoms or something like that. I mean, you could put it in that box.

DR. BRUEHL: Yes. In that context, I would probably do it in Dimension 3 actually, but it's

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debatable, I guess.

Okay. So if you look, this was the list we had yesterday. Everything that we've talked about needing to go into that is covered somewhere on this. They're all fairly broad dimensions, which was what the intent was so that we could have a place to put other things that might come up.

I think we succeeded in doing that, and I will kind of, I guess, leave that where -- oh, can you bring up the one slide? I wanted to just do a real fast comparison here just so you can see.

So this is the chronic pain criteria. Core diagnostic criteria, that's exactly what we've got here. The main difference is that we have the event listed and some of the more physiological tissue issues, and that kind of thing that's relevant. But it's basically the same thing.

Dimension 2, we called it -- Roger, I didn't realize you -- these are your slides, right?

DR. CARR: Mine.

DR. BRUEHL: Oh, yours, Dan? So okay, that explains why "frequent" is up there.

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MALE SPEAKER: This is a little wordsmithing. When I first read it, if I see common features, I'm expecting to see another thing that has some aspects of it that are in common with the first thing. But I thought the intent was really to use the word "frequent" or, as you said, "associated."

DR. BRUEHL: Yes.

MALE SPEAKER: So that's a tweak to capture more precisely, what I thought was the intent of that dimension.

DR. BRUEHL: Okay. So you guys that are writing this, so we have the -- "common" is what the original chronic pain taxonomy said. "Associated features" is what I had written here originally.

Dan is suggesting we could use "frequent" also.

MALE SPEAKER: [Inaudible - off mic] or "associated," that – [inaudible].

DR. BRUEHL: Yes. So whatever we call that, though, that dimension is actually very similar to

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what we had up here where it was the non-pain features, as well as many aspects of the pain itself that were qualitative. That's where those went in the chronic pain taxonomy.

Yeah?

FEMALE SPEAKER: Just to clarify then -- because on this handout, signs, symptoms and quality fell under common features, under Dimension 2, so then we moved them back up to 1 or no?

DR. BRUEHL: No. So -- yeah, the best way to explain that is a -- remember yesterday in the talk on CRPS, I said if you went to the literature, you'd see this long list of signs and symptoms that have been associated with it.

When we did the diagnostic criteria, they only incorporated a certain portion of those. That didn't make those other ones not exist anymore. It just went they weren't used for diagnosis.

So what we actually have are diagnostic signs and symptoms that are crucial to diagnosis in Dimension 1. And other signs and symptoms that

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aren't actually used to make the diagnosis per se but are kind of characterizing the disorder, those go in Dimension 2.

That's an important point because this is what I discovered in doing this for the chronic pain. When we're getting things back from the working groups with proposed criteria, some of them felt like -- it was very clear, they felt they had an obligation to list every single sign and symptom that had ever been reported in the literature as part of their diagnostic criteria. And in one case, I think there were at least 12 or 15 criteria.

That is not necessary because many of these things only occurred in small portions of patients and were things that maybe didn't differentiate very well from other conditions, so they wouldn't be very useful.

Henrik?

DR. KEHLET: Where do we have the pain assessment? Rest pain versus evoked pain, which is crucial.

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DR. BRUEHL: That's a methodology and that -- okay, but what that would do is Dimension -- in my mind, Dimension 2 would be common features. So the common feature listed might be pain exacerbated with movement or pain only on movement.

What you just said, like having them do some activity to evaluate that, to assess that, that is the methodology you would use to carry out Dimension 2.

MALE SPEAKER: If it was part of the core diagnosis, it could be in core criteria.

DR. BRUEHL: It could be.

MALE SPEAKER: If a particular pain disorder -- if mechanical allodynia was a key feature, that could be --

DR. BRUEHL: And that's absolutely right. You guys in the working groups would be the ones to decide what's important enough to put as a core diagnostic criterion. And it might very well be that pain with movement is pathognomonic for that particular condition, so you'd put it up there.

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DR. TIGHE: To address Henrik's question, we have trajectory, at least, to list under the common features or associated features. We considered expanding that as somewhat to temporal, which included trajectories of ascending, descending, and flat stability for random cycles, or versus stabled, ranges and then inducibilities as well.

So we took that single term and we expanded upon it a little bit. But I agree, this is something that will have to be better defined in the working groups outside of this discussion.

DR. BRUEHL: And I think the graphic that was shown earlier that had an X-and Y-axis and then five different lines, different slopes or different oscillations, that is what's captured in Dimension 2. That's what part of what would go there.

MALE SPEAKER: While the idea is in the air, how about just saying "other features"? Because if something is a feature, that means it's featured, so there's redundancy. Just saying "other features."

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DR. BRUEHL: You guys can write that down under 5.
We have lots of options. But I think that's -- I mean all these are okay to consider.
Greg?
DR. TERMAN: So again, I really like the idea of trying to stay near the chronic pain taxonomy as possible. But my real point is that the temporal trajectory may end up in the first dimension to distinguish it from chronic pain conditions.
DR. BRUEHL: Can you explain that a little bit? What do you mean by that, like an example?
DR. TERMAN: Well, I think Raj's example is the best one. We talked about it -- Dr. Kehlet talked about, you know, certain --
If you're talking about post-surgical pain, for instance, well, you're probably talking about it within a certain time epic. So in order to have it be a core criteria of post-surgical pain, you need to put that epic, even if it's arbitrary. You're not going to call it post-surgical pain if

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it's 3 months out.
DR. BRUEHL: So pain that occurs within one week of having undergoing X surgery --
DR. TERMAN: I have no idea what time frame is appropriate.
DR. BRUEHL: I think that --
DR. TERMAN: But it seems like, for all of these acute sorts of conditions or events, we're going to be talking -- we're going to need, as a core criteria, when is it no longer acute.
DR. BRUEHL: So event and the temporal association with the event. Right?
DR. TERMAN: Yeah.
DR. BRUEHL: I guess we didn't make that explicit.
DR. KENT: So another way to possibly look at it, too, on the microfiche is -- we have type of event listed in core criteria and in common, other or associated features, temporal.
So one concept would be to lump it into core criteria or to split it in terms of having time from event, whatever that may be for whatever that

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working group is. But in the same sense of core versus common, having that graph of undulation and everything being more of the descriptive richness of the temporal nature in common. But at least that core concept of time from event be in the core set.
DR. BRUEHL: I will tell you in writing actual criteria to be used in clinical practice, when you have a situation where you're having to arbitrarily pick a time period, like 7 days or 14 days, in some ways, that is something that may be better to leave it up to the clinician and leave it as something to say, yeah, you got pain that occurs following X surgery, and the pain is temporally associated with the surgery.
You don't say what "temporally associated" means. You leave it up to their judgment, and that avoids the issue of having to be arbitrary in doing that.
DR. DWORKIN: But, Steve, you couldn't do it that way if you wanted to do a clinical trial. If I'm doing a clinical trial of acute

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post-thoracotomy pain, I've got to define my time window.
So I agree with Greg that I think in -- in a sense, I agree with Mike also, that for many of these conditions, maybe all of them, one aspect of the core diagnostic criteria is going to be the time window used to define acute pain.
DR. TIGHE: So one of the implicit constructs here under core criteria with time from event is at the time from event is also directly associated with the length diagnosis, so post-thoracotomy, from thoracotomy, but also the tissues involved with Dr. Brennan's talk, so we know exactly what features we're talking about in that, and then the location to give us a little bit more of a representation to other clinical context that already exist.
So we've tied those together into a single entity. We really can no longer talk about time from event without also considering the diagnosis, just like we can't talk about the diagnosis without considering how far out we are from the injury.

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DR. BRUEHL: And I'm getting a little confused here. So are you arguing that the time window, whatever that may be, is part of the Dimension 1; it's going to have to be?
DR. TIGHE: I believe so.
DR. BRUEHL: I do, too. I just want to make sure we're agreeing on that.
DR. TIGHE: But now, the fluctuations and other sub-characterizations, I think, are common features because they don't necessary drive the core criteria.
DR. BRUEHL: Okay. Perfect.
DR. TIGHE: And you're not always going to know them either depending on --
DR. WEISMAN: Yes, I disagree. In terms of developing this as a hierarchy, you don't need time in this first box or this first category. And to address Bob's concern, that's easy. That falls into Dimension 2, where you might be doing an acute trial for a burn population and the clinical trial defines it as having to be within the first 24 hours of the burn.

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I mean, that would be easy. You don't need that temporal feature in the definition of the acute pain condition. And I think that's going to cloud Dimension 1.
I think Dimension 1 should literally define what the acute pain types are, and it should be irrespective of time because time is a common feature, modifying feature. It might even be modifying, or modulating, or adapting in Dimension 3. That's where time comes in.
DR. BRUEHL: Just pragmatically, so if you're diagnosing a patient -- a hypothetical example here, so our criteria say it has to have pain onset within 3 days of X surgery, right? And you've got a patient who has had nothing else happen who, day 4, starts complaining about pain. Then you got another patient where 2 weeks after surgery is complaining about pain.
Now, which of those would fall into our diagnostic category? So if the person is one day beyond our window, would you still, as a clinician, think that they had that diagnosis that pain is

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linked to the surgery? And if it's 2 weeks out, would you say that --
MALE SPEAKER: Well, no, it's like -- it seems like, you know, with the DSM-III example, that DSM-III says, oh, of course, in clinician practice, there may be people that you call depression that just fall one side of the line of the other. But we still need standard criteria so that when you study depression and I study depression, we know that we're both studying something that we call "depression."
It seems like these time periods would be similar if I do something for acute pain, something, and I want an indication for that, that it would be sort of a cutoff knowing that they're going to be in clinician practice.
DR. BRUEHL: And I think that makes perfect sense logically to have something like that specified.
I'm sorry, behind you? Yes?
DR. POLOMANO: I'm still grappling with early acute pain, which I do believe has to be

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addressed. And early acute pain is going to differ for every pain condition.
With zoster, it's not subacute pain 3 weeks later. They're still having acute pain. With joint replacements, they're still having significant pain when they're entering rehab.
DR. BRUEHL: And we're not specifying how long the pain lasts.
DR. POLOMANO: Right. But we talked about that subacute, which I think, again, subacute infers that it's -- if you have subacute care, you get less care; it's less intensive.
So if we could do early and persistent as terms and focus on the early, and not define it by a time course but a way that it would characterize the initial acute pain presentation --
DR. DWORKIN: Rosemary, I want to ask you a question. So I agree with you that, personally, I would consider pain 3 weeks after rash onset in a patient with zoster to be acute pain. But what about 5 weeks? And I think it's up to us or the working groups to set those windows.

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DR. POLOMANO: Yes. So it's all different but it would be different for every condition.
DR. DWORKIN: Absolutely.
DR. POLOMANO: Right. So if we could just define that the later kind of pain that might be -- just trying to align with the chronic pain because you have the subacute -- you had the chronic and we have IASP with this subacute category.
So I'm trying to line the trajectory up to chronic pain for this particular -- so if there was a way to do early and persistent and not give any time because it's all relevant to the pain condition.
DR. BRUEHL: It is specific to each pain condition. And don't mistake the fact that this is standardized language up here to mean that what's in these boxes for any given condition are being specified. That will be totally up to the people in the working group who can factor in that in this condition, 3 weeks is considered abnormal and very long; in this condition, that's totally normal.

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DR. POLOMANO: Can I make one more comment? I feel like the source of this whole debate is that for some conditions, different, like temporal character, should be in the core criteria, whereas others, it maybe is more -- it's better to be in common features because it's less important.
It seems like Dimension 1 has been like, you know, diagnostic criteria, and then 2 is like the less important features; is that what's happening here?
DR. BRUEHL: Kind of. Kind of.
DR. POLOMANO: So I mean, it may be different per type, what is in 1 and what is in 2.
DR. BRUEHL: I think it is probably true. Correct me if I'm wrong. In any acute pain condition that we're talking about here that you can think of, is there any of those where you would not find that the pain has a clear association in time with whatever the triggering event was?
Basically, I'm talking about do we have an acute pain condition that has no triggering event, that just has no way to say that it is linked in

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time with any --
DR. STANOS: So is that like some person who presents with knee pain for the first time, and they say, oh, my knee pain started Friday? They didn't have an injury, but they just noticed it.
DR. BRUEHL: Now, we're going on the boundaries of diagnostic criteria.
DR. STANOS: But like what happened before that that led to that time on Friday, they noticed knee pain. I'm just throwing that out there. I mean, it's a little different when you look at these acute conditions.
MALE SPEAKER: It's probably true that there's no acute pain condition that wouldn't have some sort of time limit, if that's what you --
DR. BRUEHL: Time -- no, I was actually saying that -- because we're talking about two different issues. One is that the onset of the pain is temporally associated with some event, and that's what I was just referring to. But we've also got the issue of once you got that pain, how long does it persist, how does it change over time,

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those kinds of issues.
I think for a core criteria, it makes perfect sense to say that the pain is temporally related to some identifiable event that will leave out those people that just have spontaneous pain where they can't recall any injury. But maybe you do some exam and you discover they've got a ligament injury from something that they did that they didn't really remember.
So we're really talking about two different aspects of that here.
Yes, Roger?
DR. FILLINGIM: I mean, I think it's safe to say that there's something temporal that belongs in Dimension 1. There are plenty of acute pain conditions for which no event will be identified, so you can't link it in time to an event because there is no discernable event.
DR. BRUEHL: Which wouldn't rule out the diagnosis, then, in those cases.
DR. FILLINGIM: No, no, no. But there's always a time dimension, right? So if I've had jaw

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pain for 17 years, that's almost certainly not acute TMD. But if I've had jaw pain for a couple of days due to nothing whatsoever --

So there is something temporal there. There are other temporal things in Dimension 2, and how those apply to the conditions, as you've stated, will depend on what the condition is. And the working groups can figure that out, I think.

DR. RAJA: Steve, I'm just thinking of an example. A good example is phantom pain. Epidemiological studies show that while the majority of them come very early on, there have been descriptions of a proportion of patients who have, months to years after their amputation, the onset of an acute phantom pain. So does that rule out an acute pain? I don't know.

DR. BRUEHL: That is a gray area, yeah. And you got to think about those odd cases when you're coming up the diagnostic criteria. Do you want them to apply to somebody who would fall under that situation? And if you do, you have to word it so that the criteria would capture that person.

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MALE SPEAKER: So --

MALE SPEAKER: Steve --

DR. POLOMANO: So --

DR. BRUEHL: We got two -- you guys fight it out. Sorry.

FEMALE SPEAKER: I'm just thinking of that story that Steve gave about somebody who's had degenerative knee disease for many years but the pain starts spontaneously suddenly. So that's an abrupt onset of pain.

But I don't know that I would categorize that as acute pain because that's going to continue. That's going to be a chronic pain condition because of the degenerated joint even though the onset --

DR. STANOS: Maybe it's not the knee. They just have a short --

FEMALE SPEAKER: Right. You can argue, is that acute pain or chronic pain?

DR. STANOS: Yes, I guess both ways. They have an acute pain problem, but then they get better on their own. I don't know. I think it can go either way.

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DR. BRUEHL: That is probably for the individual working group.

DR. SURESH: Steve, I think we do run the risk of overlapping too much on chronic pain if we don't define a time period and if we don't define a clear cut demarcation between this and chronic pain.

I'm really concerned that the mode of the discussion is leading towards what happens a month from now. This is acute pain. Let's deal with acute pain. And that's where we need to start focusing because it's extrapolating into this huge, what am I going to be doing a month from now? We'll deal with this as chronic pain later or subacute pain.

DR. BRUEHL: Do you want to put up your graphic and let's --

DR. COHEN: When Steve Stanos was talking about muscle tissue and the type of pain associated and the transition, that frequently involves a time course that's longer than we might consider as

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acute pain.

It brought to mind this article that Patrick wrote in Pain, Volume 16 and I just wanted to read about 600 words from page 1813. But if everybody agrees we could just add it to the transcript later.

DR. BRUEHL: What is the gist, very briefly?

DR. COHEN: It's transition from acute to chronic pain.

DR. BRUEHL: Okay.

DR. COHEN: And it includes 12 potential mechanisms that could be involved.

(Dr. Cohen's insert.)

"Transition from Acute to Chronic Pain, an essential component to include in the AAAPT report. (Excerpted from page 1813) in Tighe, P., Buckenmaier, C. C., Boezaart, A. P., Carr, D. B., Clark, L. L., Herring, A. A., Kent, M., Mackey, S., Mariano, E. R., Polomano, R. C. and Reisfield, G. M. (2015), Acute Pain Medicine in the United States: A Status Report. Pain Medicine, 16: 1806-1826. doi: 10.1111/pme.12760

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"Transition from Acute to Chronic Pain
Nearly, all cases of chronic pain begin as acute pain. Many models suggest that prolonged exposure to acute pain leads to structural changes within the central nervous system that transform this condition into a chronic pain syndrome [104–107]. Depending on the type of surgery, as many as 50% to 70% of patients may experience surgical-site pain at least 6 months after surgery, with approximately 10% rating their pain as severe in intensity [108,109]. Established risk factors for the transition of acute to chronic pain in the surgical setting include younger age, female gender, catastrophizing, low socioeconomic status, preoperative pain, impaired diffuse noxious inhibitory control, type and duration of surgery, injury to specific nerves, severity of acute pain, and, possibly, prior exposure to radiation therapy and chemotherapy [21,110]. Notably, the focus of research to date has been on acute-to-chronic pain transitions in the perioperative setting; investigations on the acute-to- chronic transition

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in nonoperative patients have lagged.
"The association between acute pain severity and the risk of chronic pain deserves special attention. It is important to note the paucity of evidence demonstrating within-subject effects of intervening on acute pain to definitively reduce the incidence of chronic pain. Thus, it remains unclear whether the association of acute pain intensity with chronic pain incidence is predicated upon high nociceptive loads, poor analgesic effectiveness, high pain sensitivity, poor coping, all of the above, or perhaps none of the above. Further, given the importance of timing in such transitions, it remains unclear whether there are certain temporal or even spatial thresholds of acute pain that increase the probability of developing chronic pain. It also remains possible, if unlikely, that observations of acute and chronic pain are conditionally independent of one another. Parsing the independent effects of the above factors is difficult in experimental—let alone clinical—settings, given that the observed state of

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a patient in pain reflects the interaction of the above factors. It is even unclear whether this represents multiple facets of a single disease (e.g., chest pain and arm numbness from a ST segment elevation myocardial infarction [STEMI]) or separate disease states with distinct mechanisms that present with a similar set of symptoms (e.g., gastroesophageal reflux disease and STEMI both produce chest discomfort yet have radically distinct mechanisms).
"A limited number of studies have identified predictors for the development of chronic postsurgical pain (CPSP) syndromes, strongly boosting the rationale for advocating for more comprehensive perioperative assessment of pain and related outcomes and aggressive pain prevention management [82,111–114]. It is estimated that 10% to 50% of patients undergoing common procedures such as thoracotomy, breast surgery, inguinal hernia repair, leg amputation, and coronary artery bypass experience chronic pain following surgery [108]. Interestingly, a more extensive body of

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evidence for CPSP exists for patients having surgery for cancer [115]. A number of studies have uncovered high rates of CPSP among patients having general surgery, joint replacements, and prostatectomies [116–119]. Simanski et al. conducted a follow-up evaluation (mean 29 months postsurgery; N 5 911) and found that CPSP, defined by pain intensity 2' 3 of 10, was experienced by 83 patients (14.8%) [116]. When analyzed by surgical discipline, 28% were general surgery patients, 15% vascular, and 57% trauma/orthopedic, and CPSP was observed in patients having major or minor procedures. Chronic pain prevalence was as high as 44% following total knee replacement and 27% following total hip replacement [117]. Overall, estimates of chronic pain prevalence range from 10% to 34% following total knee arthroplasty and from 7% to 23% following total hip arthroplasty [118]. A relatively smaller proportion of patients, about 14% at 3 months and 1% at 6 months, experience CPSP after prostatectomy [119]."
(End of insert.)

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DR. COHEN: With that regard, I would like everybody to also channel Steve. Take a deep breath, and then I would like people to take responsibility for providing foster care for the orphan that doesn't have parents.

Subacute pain is not part of chronic pain; it's not part of acute pain. Chronic pain medicine is never going to address that. I would suggest that in our system, we create a space that we call "subacute pain," and we leave it empty to be filled later, but that would allow us to take care of patients who may have persistent pain or may be at risk for chronification.

So all I'm suggesting is that -- I don't know if my pointer points -- is that right there, I don't care what we call it, just an empty space that could be on a line that everybody agrees for a particular condition is beyond the acute period that we could add stuff to later.

I wouldn't debate how long the period is because it might be the separation is different for different conditions.

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DR. DWORKIN: So Bob, the way I think about this -- and I just want to make sure whether it is or is not consistent with what you're saying -- is that for Dimension 1, it seems like we've all agreed that there's going to be some time window from event when there's an event or just as an absolute value if there's no event.

To me, this kind of acute transitioning to subacute is in Dimension 2 as the temporal trajectory. So for whatever condition, we've already given a window for the diagnosis, but I think it would be a very interesting paragraph in Dimension 2 to talk about the trajectory beyond that window.

So to go back to my favorite example of shingles, if let's say with Rosemary, we define acute zoster pain is within 3 weeks of rash onset, I think a paragraph, a very interesting paragraph is what about 3 weeks and beyond? Does that capture what --

DR. COHEN: Exactly. And then we can -- if we can own the period of time that goes between

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what whatever everybody agrees is acute pain and what everybody agrees is chronic pain, if we can take responsibility for that portion of pain disease, pain conditions to be worked on later, I think we will be making an important step in the right direction to helping a lot of patients who might be in that box but we don't know a lot about it.

MALE SPEAKER: We used to call this the course of an acute illness. I mean that's what we're talking about. And without new words, we could just say what doctors know already.

DR. BRUEHL: Right. We're calling it "trajectory," but it is --

MALE SPEAKER: I know. But it sounds like we're forcing new words on what is, what, hundreds of years old.

DR. COHEN: Does anybody have a problem with the idea that our schema could include stuff that went more than a week?

DR. BRUEHL: No, I don't have a problem, and I think the relevant issue is the AAPT chronic

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pain, in the discussions when we were planning that out, exactly this issue was brought up, was like what is the dividing line for saying somebody has chronic pain?

The best we could come up with was what's used in the epidemiological studies, which is 3 months or 6 months of persistent daily or near daily pain. And I don't know yet what's going to happen with all the working groups, but I know for several of them, they're just totally skipping that issue because they don't want to have to try to define that.

I think that at the very least, we do need a discussion of this because it could very well -- if they're in their heads in the chronic pain, thinking 6 months, then there are clearly going to be a pretty large number of people that will be excluded from any taxonomy, kind of in a gray zone, if we don't at least say maybe this applies to them, too. So I think it's absolutely worth noting.

But I will also say we were weasels when we

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did the chronic pain when we weren't intentionally not talking about it because we don't know what the right answer is.

DR. COHEN: We'll take responsibility, though.

DR. BRUEHL: Okay.

MALE SPEAKER: But no one is saying that the chronic nosology is permanent.

DR. BRUEHL: No.

MALE SPEAKER: That's right.

DR. BRUEHL: They will be modified also.

MALE SPEAKER: So there may be some features here, or words, that may affect the wording there, too. Eventually, they're going to agree, I have a feeling.

DR. BRUEHL: I suspect so.

Yes. Henrik?

DR. KEHLET: Well, if the definition of chronic pain was lasting 3 months or more, why isn't acute pain lasting from zero to 3 months?

DR. BRUEHL: Because we couldn't decide on 3 months or 6 months or any other specific number.

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Although, we could agree that that's most commonly used to define it.

MALE SPEAKER: Winner takes all, might as well?

DR. BRUEHL: Yes, what we could do is we could say generally less than 6 months, I mean, if you wanted to do it that way. But I don't think we can get very specific about it.

FEMALE SPEAKER: So I think the thing that's missing that you see in other definitions is beyond a reasonable period of tissue healing, too, which brings back that issue of procedures versus, you know, an amputation --

DR. BRUEHL: Which brings back to Bob's issue of how do you employ that in a clinical trial to be specific because it is clinician judgment, right, whether it's beyond the normal healing or not?

DR. STANOS: I'm just going back to knee. I mean, if you have an ACL tear and then you have pain related to that, that's very different from -- like Deb saying, just a prepatellar

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bursitis. But if you're looking at these acute pain conditions, there's got to be some flexibility in deciding that.

DR. BRUEHL: I'm in favor of having some wording that makes it a little bit flexible. But I think Bob raises a good point. For clinical trials, that can lead to problems, potentially.

MALE SPEAKER: Why not have something like an asterisk that says "relevant to" a particular condition can be changed, but give an outlier 3 or 6 months, whichever it is. Some conditions, it may be 6 months. Probably, in most, it's 3, but we can discuss that. And then say "subject to the particular condition and its usual course."

DR. TIGHE: I'd like to congratulate our group and our team here for having the discussion. I'm brought back to the p-value, 5 percent, which kind of got stuck in the literature without much discussion, and here we are much later.

But one of the implicit lessons, the reason it has gone out, it's just little probably too conservative, but not so much that it's been

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egregious in how it's been used throughout the past several decades.

The numbers that have been thrown out so far has been 3, 7, 14 and 30 days. I do love the idea of an asterisk. I think we could probably make a general stance where we'd say -- we'd pick one of these numbers and say, in general, we expect acute pain would last in this region. However, it's conditioned upon other factors, and so exceptions are common and expected.

So we leave ourselves out. It's not a complete firm, concrete stance, but at least we're not abrogating our responsibility to address the elephant in the room there.

DR. BRUEHL: I think you should write that, and then let everybody see how it looks in writing when they read that, does it sound legitimate or not.

Yes, Sean?

DR. MACKEY: To add a mild variation to that, on the NIH chronic low back pain task force, the definition for chronic low back pain was back

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pain at least half the time for 6 months. And the idea was that during that, you would have accumulated at least 3 months of chronic pain; so just for the sake of parallelism and other efforts that are ongoing.

DR. BRUEHL: It might be good to mention that as an example in there.

DR. KENT: I think the example with the asterisks and throwing out something general is appropriate. This isn't the only version that's ever going to be put out there, that to say in our current state with our best discussion that we had in the current state of the literature, we'll generally consider a time frame of this, completely dependent on the condition, with full rights to be revised as evidence accumulates and more consensus is reached.

DR. TURK: And you can give one or two examples to illustrate that it's 3 days in this circumstance; it will be 12 days in this circumstance. And that gives you the idea that it's clause specific to whatever case you're

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talking about or disorder you're talking about.

But I think if you give an example, it'll drive the point that for some circumstances, it's 3 days; for others, it may be 3 weeks.

MALE SPEAKER: Exactly. I mean, we don't want to sound clinically ridiculous.

(Laughter.)

MALE SPEAKER: I don't know about that --

DR. BRUEHL: Speak for yourself.

(Laughter.)

MALE SPEAKER: You left out more.

DR. BRUEHL: What? Oh.

(Laughter.)

DR. BRUEHL: All right. It took me a second to get that one, John. Thank you.

(Laughter.)

DR. BRUEHL: All right. What else do we got? Anything else with this? Okay. Bob?

DR. DWORKIN: Question. Is there a broad consensus on these 5 dimensions as a framework for a multidimensional approach to the diagnosis of acute pain? Does anyone have a problem with the

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5 dimensions that Steve just summarized?

DR. SURESH: I think it's a start, but I think once we evolve into something on paper and as we continue to edit, I think we're going to make a change. I think it's very hard because we're spinning here. And in many ways, I think we all need to just sit down and look at this.

DR. DWORKIN: Suresh, thank you. You're exactly right. The consensus-building doesn't end until the colleagues to my right press "send" or "submit" on the journal websites. So these dialogues will continue as the manuscript that they draft is revised and finalized.

But does anyone have any kind of comments that need to be considered right now about this 5-dimension, multidimensional framework?

Roger?

DR. FILLINGIM: So maybe just one question I would raise. I don't have any particular opinion about this. But is everybody happy with the order in which the dimensions are listed? Because no matter how many times we say the order is

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arbitrary, people interpret them in order of decreasing priority or something like that.

So if you accept that people are going to stop somewhere before they get to Dimension 5, which -- yes.

DR. BRUEHL: I think they're perfect as is.

MALE SPEAKER: I don't.

(Laughter.)

MALE SPEAKER: I agree, and I think somebody actually used the word "secondary" before, and that's just what we don't want. I mean, each one of these five has importance.

The way I envision it, again, just to use Venn diagrams -- or I'm sure you have a better way, Steve -- is just to maybe, centrally, you have the core criteria in the center, and then you have four circles around or boxes, whichever they are for each of the other four, and they all interrelate, not only with one but also the one another if that's feasible. And I'll leave that up to our scribes to pictorialize.

FEMALE SPEAKER: Originally, I think I

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raised this concern that Dimension 3 or things that are host factors or that are psychosocial and stuff like that would get lost.

As someone said, oh, hey, people just look at Dimension 1, whatever is at the top of the list, and then they don't look at the rest and they don't use the rest. And I think that would be doing a disservice to our mission.

DR. DWORKIN: On behalf of ACTTION, I will offer the services of a graphic designer if Patrick and Mike need such help in preparing a more visual way of presenting these 5 dimensions that doesn't suggest a hierarchy. It sounds like we all think that would be beneficial if it were possible.

DR. STANOS: Could we consider just that, instead of saying "5 dimensions" like something in front of that? Like, I'm looking at core, but "5 principle dimensions" or "5 core dimensions" to make that a little stronger, that the dimensions are -- would that help?

DR. DWORKIN: Actually, I don't think they're equal because I think dimension -- but it's

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not Dimension 1. The core diagnostic criteria is really in a separate status.

Those are the inclusion/exclusion criteria for clinical research, clinical trials, epidemiologic studies. But certainly that is a core with the other 4 dimensions enriching it. Those make a lot of sense if it can be written about and depicted in some way that people understand.

I learned at lunch that Steve Bruehl's wife is an artist, so I think he was just volunteering her services to Patrick and Mike. So we may be taken care of completely in this regard.

Yes, Mark?

DR. SCHUMACHER: Yes. Hi. Just a point of clarification in the same theme, the domain, NBPF consequences, was that constructed in any hierarchical way? Again functional consequences landing on the bottom of this sub-box, that was the only thing that really caught my attention since the functional consequences are so critical.

DR. TIGHE: I think it's probably worth

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discussing what should be subsumed under the functional consequences.

DR. SCHUMACHER: In the sequencing here, I was just wondering if -- they're not in alphabetical order, so I didn't know whether they were put in a certain priority order.

DR. TIGHE: It was based on -- we listed it based on the initial chronic pain listing, if I recall correctly.

The ontologic representation has an example that we started to show for -- under this, it's called risk or protective factors, that box. You can see that there can be further ontologic development on this microfiche. And I apologize again for the small text size.

But we can break out biological factors quite extensively. Psychological comorbidities, more factors can be segued into -- for instance the example given here is mood disorders, personality disorders, and substance abuse. And obviously, mood disorders is still quite a broad category and can be expanded on further.

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So each of these can influence the underlying dimensions as broadly or narrowly as we like. But the framework, I would like to lock down so that we at least have a structure with which to grow.

MALE SPEAKER: But there's no hierarchy.

DR. SCHUMACHER: No, I understand that perfectly.

DR. TIGHE: There's no hierarchy. We cannot --

DR. SCHUMACHER: That's not the question. The question is the sequence in which they appear --

DR. TIGHE: We could just say biopsychosocial consequences.

DR. SCHUMACHER: Sorry. The common reader would be that that function is on the bottom.

DR. TIGHE: If we just say five --

DR. BRUEHL: Are you talking about the 5 dimensions, the ordering of the 5 dimensions or within each one?

DR. SCHUMACHER: No, no, no. No, within the

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dimension box.
DR. BRUEHL: Maybe we should specify that these are not listed in order of importance --
DR. SCHUMACHER: Yes, I would just throw that out there as a detail, that's all.
MALE SPEAKER: Also, on the first round, just a procedural note, I would appreciate it if both of you would look at the original notes that Steve had and make sure that everything is written down when you circulate it.
Let's not assume that everybody knows that one term implies another, so that we all see it. And you have it all written down. It's just a matter of looking at again.
MALE SPEAKER: [Inaudible - off mic].
MALE SPEAKER: Yeah, I know. I saw you. That's why -- okay. Good.
DR. TIGHE: Hopefully, we'll have them encoded in yet another format to circulate as well, because, yeah, we're depending on this quite heavily.
DR. DWORKIN: Rosemary?

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DR. POLOMANO: Just a couple things, [inaudible - off mic]. I was just keeping track of all the issues that came up. So we're agreeing that it would be first and center -- or maybe we should agree on some terminology that's going to be used throughout that we are all in agreement in terms of this.
One of the other issues that came up that we didn't discuss was the integration of patient-reported outcomes. They came up in almost everyone's talk.
So how is -- I mean, I am fine with these domains, and I think they really align with the chronic pain ones. But we really didn't come to any kind of consensus about patient-reported, except for the fact that I think we were in agreement that some are reliable and valid and should be used, and there are far too many to put in this type of a framework for disease-specific, or condition-specific, or event-specific diseases.
But I think one of the things that we should do is, if at all possible in this taxonomy, are

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there going to be any opportunities to make recommendations because I think one of the things that we should do for patient-reported outcomes is fit these [inaudible – off mic].
DR. DWORKIN: It's a question of how the components of these 5 dimensions are assessed is probably beyond the scope of this paper. Because, of course, we're only so far talking about the first half of this paper. We haven't gotten yet to the taxonomy of acute pain conditions.
So what we did with the chronic pain initiative is to have -- and you've heard talk about this. There's a supplement in press in Journal of Pain with 8 articles. About 3, maybe 4 of those articles discuss assessment.
So it remains to be determined whether we're going to go down the same path for this effort of preparing a supplement that we talk about assessing function, and sleep, and et cetera in patients with acute pain. But I think it's probably beyond the scope of this paper other than to just mention it.
DR. POLOMANO: Yes, I think it should be

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mentioned. Yes, I think there are far too many outcomes to --
DR. TIGHE: So already, we have a list of things we are proposing to address later down the line, and that includes a definition of methods, further exemplar characterizations of explicit acute pain states, what is currently underway for the chronic pain system.
I would consider the pros as yet another approach. Hopefully in another two or three years, there will be a much more robust acute pain pro pool. I know that's actively being investigated currently.
I think throwing out four to catch later, is that reasonable? And are there other domains that folks would like to us to mention that we would consider in the future? Education, connotations, or others? I think this would be a good point, just trying to kind of plant a flag for looking forward.
DR. TERMAN: I'm not sure this is related to that question, but it may be. I personally hate to

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lose "comorbidity," the word "comorbidity" out of Dimension 3 there, the title that is.

I realize that "comorbidity" is too narrow for all the things that are in there because you're also talking about things that protect against longer-term pain. But I do think that somebody who's on a bunch of high-dose opioids, that that is a comorbidity. And I'm kind of tired of surgeons calling and saying, the only reason they're here in the hospital is because of their pain. Well, yes, actually, the only reason they're here is because of their surgery.

(Laughter.)

DR. TERMAN: The point is that there's this big comorbidity of the 180 milligrams of OxyContin 3 times a day that was completely unrecognized when the decision to have surgery was made, so I hate to lose that.

DR. DWORKIN: So Greg, how about making it -- and I think Steve is going to do it right now. The proposal would be making it "modulating factors including medical and psychiatric

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comorbidities"?

DR. TERMAN: Again, that's just a personal input because I felt like that's what you were asking for us to do.

DR. DWORKIN: And it also emphasizes the continuity with the chronic pain effort. It's a little different, but it's certainly continuous.

DR. BRUEHL: How about "modulating factors and relative comorbidities" or something like that with short -- is that okay?

MALE SPEAKER: That's really [inaudible – off mic].

DR. TERMAN: Yes?

FEMALE SPEAKER: I wanted to just add to that. I wonder if -- you know, the language that's being used a lot are complex pain patients coming in for acute pain. It's that mixed pain syndrome, which I think that's a little different than comorbidities, which is Greg is getting at to some degree.

So I don't know if it's just another word within this category or -- do you know what I mean?

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They're complex pain because they've got chronic pain; they're on opioids; they've got a pump with stimulator; they've got a chronic pain syndrome. You're right.

DR. TERMAN: So again, the reason I chose comorbidity is because I'm a lumper and because that's in Dimension 3 of the chronic pain thing. But I think it does add something. You could use another word instead, but I'd hate to see it fall out completely because I think it adds something.

DR. TIGHE: Just to make sure I understand it, without exception, would there be a role in the vignette you offered also for including into Dimension 4 as a functional consequence? Because it's heavily not just modulating the pain experience; it's heavily modulating what happens with the acute pain event itself.

We run to this issue daily, if not hourly, so I certainly agree with the importance of emphasizing it. I just want to make sure we emphasize it with its due attention.

DR. TERMAN: You're calling out opiate

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tolerance in particular? Well, I mean it also may be related to Dimension 5. This is just one example. And again, each group is likely to want to work on those examples maybe quite different for other acute things besides surgical.

But I guess my point is that if you think of comorbidities as other medical conditions that are affecting this acute pain process -- I mean, a lot of what we've written there are comorbidities.

DR. BRUEHL: Greg, is part of the issue with this, trying to increase the parallelism a little bit, too, by using the same wording that we used before?

DR. TERMAN: I won't deny that.

DR. BRUEHL: Okay.

(Laughter.)

DR. TERMAN: It's easier to teach when it's not something completely different.

DR. BRUEHL: Then we can pay lip service to that and add a word.

Raj?

DR. RAJA: I just wanted to add that in

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thinking of conceptualizing this paper, while it's important to develop these dimensions, I would not lose the opportunity to present some future considerations, both in terms of research and other perspectives, as part of our discussion.

DR. DWORKIN: We are halfway to the time I promised you that was going to be the adjournment to this meeting, and we're only halfway through, I think, this manuscript because we haven't yet talked at all about the acute pain buckets, silos, whatever we call it, that we heard about this morning.

As you can see from the page in front of you, once you drop below the box of the 5 dimensions, there's a proposal for these acute pain silos.

So we have about an hour and 15 minutes to talk about what -- so the way we viewed it for chronic pain was that the dimensions were a framework for the diagnostic criteria, that the categorization of chronic pain conditions was a taxonomy.

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So what we're now doing is moving from the framework, the multidimensional framework, to an acute pain taxonomy, that depending on how Patrick and Mike do it, is either table 1 or table 2. But these are really the two key tables in the manuscript.

So unless there are specific questions from Patrick and Mike, what do people think about an acute pain taxonomy?

I don't think we have a slide, but everyone has it in front of them

DR. COHEN: While I'm considering the slide, I wonder if part of the process could include faithful mapping between what AAPT has done and what AAAPT is doing.

I would include that within the taxonomy as well as the dimensions to make it easier for folks who want to make that transition from RSD and causalgia to CRPS-2 and 1.

We should at least be keeping track of where the alignments are. Is there a mechanism for that that doesn't make a lot of work for the people who

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are doing this?

DR. DWORKIN: So if I'm understanding you, just one possibility is to align as closely as possible the acute pain taxonomy to what we've already done for chronic pain.

That would make it look substantially different than what we have at the bottom of this page, where there's a hierarchy that begins with three classes of events, kind of disease trial, mass surgical.

We didn't use anything like that for chronic pain. And I think you saw it earlier on yesterday, peripheral and central neuropathic, spine pain, arthritides and arthropathies, fibromyalgia and chronic widespread pain, cancer and sickle cell pain, so that's a taxonomy. It's very different than what's at the bottom of the page here.

FEMALE SPEAKER: I actually think what's at the bottom of the -- I like it in that it kind of reflects what we've been talking about the last couple of days and sort of captures that. I think in some ways -- I mean, although we want to keep

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parallel, acute pain is fundamentally different.

FEMALE SPEAKER: So are you looking for suggestions for the kingdom boxes? Is this the level for the kingdom to -- because I think -- so was procedure -- didn't we say procedural was going to be at the same level of trauma and surgical because procedure wouldn't fall in at that level?

DR. TIGHE: So I'll preface this by saying I don't know if this is the best way to proceed. I am certainly not wedded to this idea. It was just an initial framework.

The other issue to address is if you look at the example given for a fracture, it has ties both to surgery and trauma. The idea is that each of these elements may overlap.

Now, the inheritance pattern suggests that if it's surgical condition, we'd have to consider the ICD procedure code, so the specific event. But you'd also have to consider the anesthetic type and potentially other issues germane to the surgical class.

You'd also have to consider details germane

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to the trauma class, which might include an injury severity score or an Apache score or such. So fracture would then contain all of the elements from those areas.

There are certainly other ways of organizing this aside from surgical trauma and medical. This seemed to be a reasonable approach of an 80 percent solution for acute pain issues. Whether we want it -- and we can put a second layer underneath this layer, for instance, look at neuropathic pain, musculoskeletal, and then have a merging.

I've mapped some of that out. I think it got even more confusing. So I don't know if that's helpful or hurtful, to be honest with you.

FEMALE SPEAKER: Well, I think it's fine that what you have. The only thing is that I would add the box for procedural as an event, aligned with trauma and surgical. So I would just add "procedural" at the same level.

Then to capture that "medical" box, I would put -- instead of medical service, I would just make that box "health conditions." I don't know

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that "illness" is the right term, but they would be all of the health problems, diseases, the conditions, and you illustrate it with pancreatitis.

So I think it's really good. I would just make those --

MALE SPEAKER: Let me just say briefly that I hate the buckets because I think we have no idea what causes so many of these disorders. We're going way beyond what we know, and we're just creating things that'll be --

I understand we could say that while we're doing this based on current knowledge, a lot of this is going to be wrong, but why do it? Why not just come up with a taxonomy and a definition for each acute pain disorder and not try to call this a relationship with them?

Because if you ask me what causes pain in rape survivors, I will say, well, we have no idea what causes pain in rape survivors. Or if you say, what causes pain in people who've just had motor vehicle collision -- I think that people think very

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often that because that trauma is associated with the pain, the trauma caused the pain, and so much evidence suggests that's not the case.

So I just I don't know why we're -- we're getting into this big mess where a million things overlap.

DR. TIGHE: It's certainly a point well taken and, frankly, if I had my druthers, I'd like to keep that approach. The problem where I run into practically is with any of our outcome studies, where somebody says, I'd like to look at all the patients who had surgery, or I want to separate the surgical and medical patients. We might say, well, actually, all of our surgical patients, all of their pain was not related to their surgery whatsoever. It was for a toenail clipping or something.

But there's almost a mandate in developing this for a research domain for a clinical enterprise that we have the ability to roll up and roll down. I'll be the first to admit that those --

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MALE SPEAKER: You could still do that without creating a binning. Could you just say, I want to look at acute pain syndromes that occur after surgery? And then it's maybe in a core criteria of, oh, currently, there is 25 defined acute pain disorders that occur after surgery. Here, they are. But you don't have to do that by creating a relationship because, again, we have no idea.

DR. TIGHE: Taken to the nth example, and I readily acknowledge this is sort of the extreme, but if we classify them, left total knee replacements versus right total knee replacements, and I'd say I'd like all them knee replacements surgeries, I don't know if I've captured them all.

Revisions versus primaries. Are total knees and total hips really similar? I'm not sure. But at some level in a clinical decision-making, we've grouped those together as total joint arthroplasties, even though we know that a total shoulder is probably going to be quite different than a knee, than a hip.

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This speaks also, I think, to the difference between revolutionary and evolutionary approaches. With a revolutionary approach, I agree; I'd actually like to go to an analytic report where we look at the pure mechanisms. We only list what we know and keep it at that, and I think there's a reasonable argument to do so.

The evolutionary stage would say, we have this long trail of things where we've done something similar in the past, ether implicitly or explicitly. And to ignore that would mean we'd have trouble basically pairing the current or proposed schema back to anything historical.

But I'm certainly open to other solutions. And these bins that we are proposed, I think they're a step forward, but they're not, again, the optimal by any guarantee.

DR. COHEN: Patrick, what comes under these? For example, I could also see these working as a 6th dimension, which would allow you to have it very high up in terms of the hierarchy for investigational purpose but wouldn't require

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everything to -- it's unclear to me how things are going to align under this.

Are all of those categories that are on this slide just simply going to filter under these three?

DR. TIGHE: This is a completely separate --

DR. TURK: This is my slide. Let me justify it. The only reason I put it up there was whatever the bin -- I call it "categories" -- are going to be, part of the functional reason for doing that is to have working groups who are going to spend time on this.

Within those, there may be multiple specific diagnoses, and they're not going to try and cover, as I said earlier, every one of the possible post-surgical conditions under there, but to pick two or three of either prototypic, or very common, or very costly or research-intense, whatever the working group decides, but they will then describe and go into the nuances and the details that we're talking about.

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But if we don't come up with some kind of binning, then how are with going to identify a group that's going to work on pulling these things together? Each of which will come to all the issues you're talking about, the nuances, about the rape example.

Well, if you have a trauma working group and they chose to pick that as a diagnostic group they want to look at, they could discuss that particular issue. But I don't think you could have this as totally unbinned because how would we divvy up who's going to work on what? What's the focus?

So just think of it as for functional purposes at this point. It may be, down the line, we decide we don't like those functional categories -- sorry -- those title categories. But it's the only way that I can see to help us work through with the nuances that you're not going to be able to capture it in the next hour and a half.

DR. BRUEHL: The other issue related to what Dennis just said is that for publication purposes, so with the AAPT, publications are coming out by

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working groups. So each working group will have a single publication describing their 3 to 5 syndromes. It's not feasible to have 40 different papers, each one covering one syndrome.

So while it doesn't have to, in the big scheme of things, be a perfect fit, it has to have some logical coherence, some reason why a reader would go, well, that makes sense that they've got these three conditions together. So it's a pretty low threshold for why we would be grouping those at this point.

DR. SURESH: Patrick, you had alluded to using ICD-10 codes, but can you just briefly explain how you plan on using that for this?

DR. TIGHE: That's a very good question. I'll start the response by saying I'm not exactly sure. The potential first element, actually, that I think would contain the most utility would be to include it as the first the item -- and in this case, I think it would or would be important -- of your core criteria.

Because if we have an event that's specified

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as an ICD-10 diagnosis, the ICD-10 hierarchy is quite extensive, the turtle's falling on your head, but it also allows us to roll up and zoom down. I think that would allow us to first linkage.

How the ICD-10s would map on to this category structure, or the three-category structure or any, I'm not exactly sure. In the post-op or surgical, we could include an ICD-10 procedure code.

Rosemary also proposed a procedure bin, which would also be immediately linked to the procedure code rather than the diagnostic code. That is one approach, but I think it certainly deserves further discussion.

DR. RAJA: At the expense of being labeled as a splitter, I don't understand the rationale behind these three groups. There should be some mechanistic rationale.

Surgery I think is a form of trauma. So separating surgery and trauma is artificially creating two separate groups. And then if Steve's job of lumping pediatrics, geriatrics, and other

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groups into one was a difficult lecture, having all medical acute pain into one lump is an enormous task for any working group. I don't think any working group can come to anything that's rational.

So I think we need to split this somehow to make it a little more workable and rational.

DR. KENT: And you have a good point. The intent wasn't that these would be the three buckets, that they didn't need to be split further. I mean, these would be massive national conference working group, take up a ball room. So yeah, totally, indeed.

I disagree a little bit with the trauma and the surgical part. There's obviously true trauma with surgeries, so I think there is some credence to that but overlap, but definitely a gray zone there. But I think bringing up discussion, we do not propose this to be completely sold to it. And I completely agree, there has to be some level of splitting below, especially in the medical component. I mean, surgery, we can pair that down. But yeah, and I think if the discussion can give us

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some ideas, that'd be great.

DR. RAJA: But as you've always shown in your -- when they come below the next level, you're seeing there's a lot of commonality between surgery and trauma. Your boxes, there's a fair amount of overlapping areas there. I mean, I agree it's not exactly the same, but in terms of either the tissue injury, the mechanisms, there's a fair amount of commonality there.

DR. KENT: True. True. True.

DR. TIGHE: Well, one important difference I do want to highlight -- and the reason I do is -- this was an independently constructed one that also maintained the post-operative phase -- is that in the surgical population, we often know exactly when the insult initiated, and we're able to intervene and analyze before that.

When we get to trauma or many of the medical conditions, we are constrained to the post-event time period, both for analyses and treatments. So I think that's a pretty important temporal distinction to make in some approach.

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The surgical trauma medical could also be rephrased as we know exactly when it started. We knew when it started, but we couldn't control it, and we often aren't exactly sure when the tissue trauma initiated. So these could also be reformulated as more temporal distinctions very broadly.

You make a great point. I certainly wouldn't want to be writing that paper to do any of those three work groups.

DR. DWORKIN: Deb?

DR. GORDON: Yes, I've been struggling for the last two days about tissue type versus mechanism. When I look at Dennis', it's kind of helped me clarify why I struggle.

DR. DWORKIN: I just want to clarify. I took me a moment too. This is not Dennis' anything. This is this morning's agenda.

DR. GORDON: Okay.

DR. DWORKIN: I think Steve's talk, so this responds to the seven talks we had this morning. We can give Dennis credit for it.

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DR. TURK: I did the slide. I didn't create the time -- I made the slide.
DR. GORDON: But if you take colorectal surgery,. where people have a referred pain to their shoulder from distention and air put in, they might have, several days later, some inflammatory infectious leak process. Is that post-op pain or is that inflammatory pain?
I like the division between medicine and surgery, but I remember Dan yesterday started out by saying anatomy matters when it comes to acute pain.
So I'm looking at table 2 of Roger's paper on the chronic that's got the taxonomy of chronic pain disorders. And I think some of this fits under medicine and surgery by anatomy type, right, musculoskeletal pain, visceral pain, orofacial pain.
So I think we could probably use some of this division in table 2 under medicine and surgery.
I don't know about trauma because you're

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talking about both physical and psychological trauma with a rape victim, right? I mean, I'm in a trauma hospital, and people are often coming in and they have -- just like Trip was saying -- all of that stuff. They've got multi-organ trauma, and they've got a lot of psychological trauma.
So I think trauma becomes a modifying -- it's like the blast injury, versus the gun shot, versus the surgery. It's a modifying factor. I don't think it's a category in it of itself.
DR. TIGHE: Deb, one question I have for you. Are you okay with overlapping constructs? So the person who comes in with the trauma and has a fractured ankle would fit under two different categories here, two different categories in the two-part schema.
Are you okay with pulling from multiple categories, or do we think we need to stick with -- force everything to a single category?
DR. GORDON: Well, if you're going to do a clinical study, I mean how would your put your

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exclusion/inclusion criteria? I don't know.
To me, you'd be like, do you want to include people that have been in a trauma, or do you want people who had a planned operation?
DR. TIGHE: We do this actually for rib fracture studies. We find people that have rib fractures, and they have lots of other trauma. There's heterogeneity in the rib fracture. I know you all have a lot of experience with that particular population.
DR. TURK: I think you want one, and a way we did this in the chronic pain -- and then, Bob, you can speak to this about the chemotherapy-induced neuropathy -- is that under a cancer or is that under neuropathic pain? And we made a decision it was going to go in one, and the other group simply said to refer to the other group to get that information.
So I don't think you want something that occurs in two different places. There has to be some agreement among whoever is doing it as to where are they going to fall. And it's totally

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appropriate to refer to, see the other one that's relevant to this particular problem. We're not covering it under this particular grouping.
So I don't think you want to have two, but I think it's totally okay to cross-reference. In fact, that's part of the beauty -- if I can say that word -- of using the same dimensions, whatever they are, is that, in fact, you can refer back and forth to these, and they'll make sense as you'll hear about them.
MALE SPEAKER: I think also we have to take into consideration that we -- a lot of us are using electronic medical records, and it becomes very difficult to separate some of this when we extract the data or we do retrospective data.
I've done this. It's easy to separate surgical from non-surgical patients when you extract the data. But trauma patients, there is no easy way to figure out from the rest of your patients.
I honestly think that we can say, surgical or non-surgical, trauma can fall into both groups,

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non-surgical trauma and surgical trauma, because not every trauma goes to surgery.

So we can subdivide after that to whatever we want and make it a bit easier for electronic medical records to go back and extract the data.

DR. WEISMAN: It's always going to be very confusing. I was sitting here thinking about actually a real person who was shot, and it took out the greater part of this sciatic nerve as it entered his abdomen and blew out a lot of stuff in his abdomen.

When he first hit the ER, he had acute projectile injury pain. Then as he was bleeding to death, he was taken to the operating room, and as you can imagine, had a zipper created in his front.

Then as he was recovering from that, after he had been resuscitated, on day 2, developed acute neuropathic pain because his sciatic nerve was taken out by the injury.

So I would argue it's okay to have categories like this. And even though I'm sort of going back on myself a little bit, the time in that

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individual dictated, if you will, what category he belonged in as he went along this insane acute pain trajectory. Right?

So we're going to have a hard time simply dividing all post-op patients into post-op. Many of them come in with acute pain from whatever, either their illnesses or their trauma is et cetera. And I think it's okay still to have a taxonomy which looks at these different buckets, these different categories. And at different points in time, an individual may fit into multiple buckets. That's totally okay.

DR. DWORKIN: They're multiple diagnoses.

DR. WEISMAN: Sure, absolutely. And they would be in the electronic record that way as well. Their ICD-10 codes, for that individual I described, he'd have all of them in there.

DR. DWORKIN: Bob?

DR. COHEN: Just to make clear, I didn't understand the children before, but now, I do. So under medical, the children are everything else that was presented, and they could be specifically

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listed as acute musculoskeletal, acute visceral, other acute disease associated.

I'm looking at your handout that says surgical trauma, medical. And as long as medical then has these other boxes to take care of the other topics that were discussed this morning, it would seem that, although they might be at slightly different levels in your tree diagram, they all would lend themselves to working group, to be a good size for a working group.

DR. KENT: Just for the sake of throwing out an idea that can get shot down, if we take the surgical and non-surgical -- I'm just throwing this out as a proposal. You have the surgical and non-surgical. Under surgical, you're going to have all surgery, but it'll be subclassified for whatever framework that is.

FEMALE SPEAKER: Often hard to --

DR. KENT: And then you take non-surgical. Other than post-op, just put all that stuff under non-surgical. I'm not saying that's a perfect listing, but at least just make -- you know, like

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chalk talk, just moving something up there, like it, not like it. But all surgery, subclassified in whatever we decide it to be, ortho, neuro, whatever.

Non-surgical acute pain, everything there minus post-op, and maybe it's a place to start, moving forward to see if we have acceptable buckets.

DR. TIGHE: So then you could also take -- if you had surgery for a specific type such as neuro, you could also pull the elements over from neuro. We found there are certain elements that were very particular for back pain. You just make sure they were included in the post-operative back pain domain. So you could still have splitting of certain elements in those areas.

FEMALE SPEAKER: Can I just ask a really simple, practical question? How many working groups are there going to be?

MALE SPEAKER: One million.

DR. BRUEHL: It depends on how many clusters we end up with.

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(Laughter.)
FEMALE SPEAKER: Do we have like just a ballpark figure? I mean, just so we can sort of wrap our --
DR. DWORKIN: We have like 8 or 9 for chronic pain, and that's worked really well. So I imagine the same neighborhood would work equally well for acute pain. Two or three is too small because it means the working groups are responsible for too many conditions, and 20 is clearly unwieldy.
FEMALE SPEAKER: Right. Because it seemed like the presentation sort of followed these kind of natural predestined things. And one thing that struck me was that, although, obviously, there's going to be lots of boundary issues, there are certain boundary issues that it might be helpful for us to discuss as a group that are more important than other smaller-boundary issues.
Like it would be good to get to that today if we could find like, okay, here's what we think the buckets are going to be --

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DR. TURK: What we did in the chronic pain group was we came up with the categories because we had people who were willing to work on those areas, which partially defined this. But then when there became boundary issues, it went to a steering committee, which was essentially who we were, the five people organizing this.
We would say, well, there's an overlap between the chemotherapy-induced pain for neuropathy, both in the cancer -- which group -- and then we would negotiate with the groups to see who preferred to pick this up, and then we let them cross-reference.
So you had somewhat of an adjudicating group, if you will, to oversee it. But the reason we came up with the number of working groups is because we had people -- and I'm emphasizing this because when you asked the question how many working groups, that's interesting. How many people in this room are willing to be involved in a working group? And if it turns out that only two of you, we'd have two working groups.

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(Laughter.)
DR. TURK: That's going to be partially reality.
DR. DWORKIN: To go back to the taxonomy, I just want to make sure I know what's on the table -- because it sounds to me like there's a fair amount of agreement -- that we could have a superordinate structure that would be very simple. And what's been proposed is either what's on the piece of paper, which is medical, surgical, and trauma, or I might just mention combining surgical and non-surgical.
So there's two different superordinate proposals on the table. But it sounds to me -- separate and apart from what we decide about that superordinate structure, it sounds like pretty much every agrees that the agenda for this morning, which is on Dennis' slide, is a reasonable breakdown for seven or eight working groups.
Is that correct or am I being too optimistic?
DR. RAJA: I'd like to tell Mike that I like

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that new organizational structure that you've indicated because from what we heard yesterday that -- and we have as it core criteria that the organ tissue systems make a difference. And we heard from all the presentations today that there are some differences between the nature of the pain, between these different systems.
So surgical, non-surgical, and then listing some of these other categories makes a lot of sense to me.
DR. DWORKIN: Greg?
DR. TERMAN: I guess another way to ask it is whether there is anybody's favorite or even non-favorite acute pain event that is not included in those categories up there.
FEMALE SPEAKER: Ischemia.
DR. RAJA: Vascular ischemia is not really, though, the same thing.
DR. SURESH: There was also another area that Rosemary alluded to, and that is procedural pain; so as a separate category because there's a lot of procedural stuff that is done, cancer

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patients and other patients.
DR. GORDON: So I wonder if that fits with burn, though, as a skin, as an organ? Because it's cutaneous, it's percutaneous needle sticks for a lot of these procedures. Right?
DR. KENT: Can that be grouped -- it's not a surgery, but can it be grouped under surgery?
DR. GORDON: No. I'm just asking, is it grouped under procedures or is it skin? Because where does burn fit up there?
DR. KENT: Oh, burn? I thought you were talking about IV sticks.
DR. TIGHE: So I think the intention is for procedure, it fits, and we know when it's going to occur. So it could fit under -- because we've identified timing is so important, it seems like it would fit well under the surgery category that Mike proposed.
DR. DWORKIN: And that's what we had this morning with Chris' talk.
DR. TIGHE: Would that be reasonable, Dr. Suresh?

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DR. SURESH: So there are things like a --
DR. TIGHE: It would be a subheading.
DR. SURESH: Okay. No. My only concern is there's a lot of procedural stuff that's done. Particularly in the pediatric population, there's tons of procedural stuff that's done and carries a completely different implication.
These are repeated stuff, too. Like a lumber puncture could be done 10 times in a week; that's a lot of procedural pain.
DR. TIGHE: One other advantage to addressing the procedural pain has allowed us to make a distinction between intra-procedural nociception versus procedural pain, versus post-operative pain.
I think we can create that. If we have its own heading, as Mike proposed for surgery, it would allow us to both differentiate those elements under the surgical heading but also give them some important special attention.
DR. SURESH: So what happens to the patient in the emergency department then, who's not

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actually having surgery yet but could have a fracture?
DR. TIGHE: If they're having a procedure, the pain attributed to that procedure would be under the surgical heading, or maybe we'd generalize it to a more procedural anticipated.
DR. BRUEHL: One thing I wanted to mention just to complicate the issue a little bit, I'm being bothered as I'm listening to this and looking at this up here. And Patrick indicated he's confused too, which is a problem is he's the organizer of everything.
We got a little bit of confounding, I think, between what the trigger is and what the presumed mechanisms are. You can see that, so we're talking about surgical, non-surgical and trauma. But conceivably within all three of those, you've got neuropathic pain, you've got visceral pain conditions, you've musculoskeletal conditions.
So if we're coming down to a taxonomy where we've got a defined number of, let's say, 3 to 5 conditions within each working group, do we end

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up with like -- do we have 9 different diagnostic criteria, each one specific to trauma, non-surgery or surgery, or do we have three conditions, like a neuropathic, visceral and musculoskeletal that has some modifier that says whether it's associated with surgical, non-surgical or trauma?
I'm just wondering, in practice, how does that get laid out?
DR. TIGHE: So I haven't been able to figure how to rectify that with having subordinate classes. One solution would be -- when we talked about the putative pain mechanisms as one of the dimensions -- is that we make sure that every pain object up here includes details on the mechanism.
So we have subtypes of orofacial pain that are predominantly neuropathic or -- visceral is not the right term. But you would just emphasize those in the dimensional characterization, rather than have certain subtypes.
I don't know how that overlaps with what was done with the chronic pain approach because it seemed to be that those were taken out as

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mechanisms and instead put as separate objects.
Are we going to be implicitly describing them according to those mechanisms, according to the dimensional structure for everything?
MALE SPEAKER: It seems to me like that might be having our cake and eating it in a good way and that we're not putting our money down on a specific mechanism when the mechanism is very dirty and overlapping.
No one can argue that the surgical pain is surgery-related. I mean, in most cases, if you have an incision-related -- for example, or a trauma-related. So at least that's general category that's not so artificial.
DR. BRUEHL: So just to make sure I understand what you're saying there, so the diagnosis would be post-surgical pain, something like that. And then within that, for a given individual, you would actually select out the modifier and -- I don't know exactly what it would it be. I guess it would be reflected to some extent in the -- pain qualities and things like

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that might differ between these 3 subgroups.
It still implies though that post-surgical pain is not a distinct diagnostic category because you're going to have a great deal of variability within that.
For example, if we try to say all post-surgical pain is one diagnostic category, when we get to trying to list the signs and symptoms that you'd use to make the diagnosis, we would have to include features that are characteristic of neuropathic pain, and visceral pain, and musculoskeletal.
MALE SPEAKER: The surgical is just a bucket, and then the individual disorders might be, you know --
DR. BRUEHL: That's what I'm asking.
MALE SPEAKER: -- your laparoscopic cholecystectomy, or pain after -- you know, much more specific.
DR. BRUEHL: Okay. So it doesn't use the neuropathic, visceral, musculoskeletal at all, but it implies those mechanisms were relevant in these

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other dimensions.
MALE SPEAKER: The individual diagnoses or individual acute pain disorders could be under that, and then within each -- a pain disorder, as we've said, one of the dimensions relates to the mechanisms, the putative mechanisms.
DR. TURK: Think about the function. How is this going to be used? If someone is interested in post-surgical pain issues, they're going to go into this and go to post -- they're not going to look across by mechanism; they're going to try and identify where do I start to use this?
So if you don't put them in some kind of buckets, how do you guide the user making decision where do they go?
DR. BRUEHL: So in the surgical bucket, we might pick the top five surgeries that are associated with problematic acute pain, right, and those would be your five diagnostic categories. Within there under non-surgical acute pain, you might have sprains and strains as a category. I'm not sure what else would fall in there.

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So the working groups, it would make sense to organize by event, in a sense, surgical, non-surgical trauma, orofacial or disease, right?
DR. KENT: I was going to throw that out there. I was wondering if for non-surgical pain, what the group felt of the agenda today. The names of each of the talks today, do you think that's sufficient to fill non-surgical pain, non-surgical musculoskeletal pain, non-surgical visceral pain, et cetera, et cetera?
I'm throwing that out there to --
DR. BRUEHL: I think the one problem child with that is the cancer pain. And I think it was mentioned earlier that you could look at cancer pain as, in some context, a surgical -- it could be a surgical pain in some cases. It could be procedural. It could fall into other categories.
DR. KENT: But if we don't -- let's not go back to the -- my thought would be let's not go back to the mechanism-based because then we just run into the same problem we've been trying to avoid if we, within those buckets, name it by the

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type of -- I mean, you can call it orofacial if you want to, but if we use bins that are descriptive but not mechanistic, not impugning a specific mechanism --

DR. BRUEHL: We have "disease-related," was kind of a category with cancer being one. Are there other prototypes for that are major -- that would fall under acute pain?

Sickle cell, by the way, is under the chronic pain. Do we want to have -- here's an issue. I would say for something like that where the features may be similar, do we want to just co-opt what has been done for the chronic pain and modify it?

DR. FILLINGIM: I don't think the chronic group with sickle cell is doing vaso-occlusive crisis pain.

MALE SPEAKER: I agree because they're not chronic.

DR. BRUEHL: Okay.

DR. FILLINGIM: So like cancer pain, they will be both acute and chronic disease associated.

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DR. DWORKIN: So the other thing, I think, for this taxonomy, we don't have to select at this point the 4 or 5 specific conditions that would be in each of these bins. That would be up to the working groups.

The way we did it for the chronic pain effort is that the early paper, Roger's paper, just gave some examples of each of these buckets that aren't necessarily what was moved forward into the actual activities of the working group.

It sounds like we certainly have a sufficient number of examples for each of these for this step of the process. And then the working groups would decide, are those examples the most prevalent, clinically, and research-relevant conditions, or are they different conditions that should be focused on?

DR. BRUEHL: I think I've written up here the buckets that may be appropriate would be surgical, non-surgical, trauma, orofacial, disease and neuro. Is that not correct?

FEMALE SPEAKER: Non-surgical contains

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trauma and -- I think we were talking about two. We have medicine and surgical. That's what I was -- that's what I'm -- what's the word? Promoting now. I don't think trauma is a separate bucket.

MALE SPEAKER: Or disease-related.

DR. BRUEHL: That's fine. I'm just --

DR. FILLINGIM: Steve, I think you're mixing superordinate categories with subcategories.

DR. BRUEHL: That's what I'm trying to get is what is the --

DR. FILLINGIM: There are two proposals on the table for the superordinate categories: surgical/procedural and non-surgical. That's one.

DR. BRUEHL: Okay.

DR. FILLINGIM: And the other is what's written here, and I don't know if Patrick and Mike are going to decide or if we're all going to decide. And then under that, I think the current proposal is everything from trauma to the right goes under non-surgical.

MALE SPEAKER: Should we also include an

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ischemic pain category?

MALE SPEAKER: Is that a mechanism or --

MALE SPEAKER: We actually treat it distinctly. And interestingly, we see certain treatments will not work for ischemic pain.

DR. DWORKIN: So does that fit as other disease-associated acute pain?

DR. COHEN: We've also left out -- where would obstetrical pain fit in? Because being pregnant isn't a disease.

MALE SPEAKER: And I don't understand how neuro is separate from post-surgical.

DR. DWORKIN: It's non-post-surgical neuropathic pain, John, because obviously --

MALE SPEAKER: That means I don't have to worry; I can't create any pain by doing surgery?

DR. DWORKIN: No, no, no. Certainly, some acute post-surgical pain is neuropathic. It's just for the sake of moving the effort forward, we have to create some arbitrary artificial distinctions, and that's a reasonable one.

In other words, in Raja's talk this morning,

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he didn't talk about, as I recall, neuropathic pain following surgery because that was already covered by Chris. But I completely agree with your point; that's arbitrary.

We could put post-surgical pain in the neuropathic bin if we all believe it's mostly neuropathic. There's going to be these arbitrary placing conditions in bin because it's not [indiscernible].

MALE SPEAKER: Similarly, orofacial, so much of the orofacial pain is due to a surgical procedure on a tooth. Now, what is that going to be? Is that going to orofacial or is it going to be post-surgical?

DR. DWORKIN: It's equally arbitrary.

MALE SPEAKER: Okay.

DR. TIGHE: One comment about the ischemic issue, we have unpublished data. We looked at 600,000 abstracts on PubMed and looked to see to what they're actually talking about using natural image processing. The first thing we -- all of the abstracts contain the term "pain."

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Far and away, the number one thing everybody was talking about was cardiac, and it was all pertaining to myocardial ischemia, chest pain, et cetera, et cetera. We had to do some fancy footwork to avoid getting into the pure cardiac domain for the first 30 concepts we looked at.

So whatever we look at as our perspective for how we would rank different categories, I think it's worth at least acknowledging that the broader health community is going to say cardiac is pretty important to some extent. And I don't know what other domains we may be missing in that area as well.

DR. GORDON: So I like the idea of surgical/procedural medicine, and then kind of again like table 2 from the chronic pain, there is a condition-associated pain not classified elsewhere or special, like labor and delivery, acute ischemic events like MI or, you know, you've struck your toe, and now you've got a dead toe, or something else somebody mentioned.

Maybe sickle cell, maybe acute sickle cell

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is called out as a special condition that's not really classified anywhere.

DR. TURK: I keep wanting to remind you -- and I'm sorry to sound like a broken record -- we're not assuming that this covers every possible condition in that category -- some group may come along and say we'd like to take on that particular issue. As long as they follow whatever template we agree on, that's fine, but it's not as if this covered -- there's no --

In the IASP classification, I believe there's 317 different chronic pain diagnoses. We're not trying to cover every one of those 317 diagnoses. We're trying to say, here is a template; here are examples within these different buckets that we've chosen to use.

There are many things that were not covered. There are some overlap, and we will refer back and forth to each other.

But I think we're getting caught up that we're going to have the -- the total taxonomy is all going to be handled by this group, at least not

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in the next year and a half. That's for sure.

DR. DWORKIN: Sean?

DR. MACKEY: Yes. Steve, you may have already covered this. One could frame this as either traumatic or not traumatic and have surgery as a form of trauma.

I mean, when you think about it, surgery is nothing more than a controlled induced trauma. The patient just happens to be unconscious, paralyzed, and amnestic. But the mechanisms, the things that you're doing to the patient is no different than if they walked out and were struck by a car. It's exactly the same mechanism. It's just clean.

DR. BRUEHL: You could look at it that way.

DR. MACKEY: So you've got trauma, which you've got surgical trauma; you've got non-surgical, and then you've got your other bucket, which is non-traumatic.

MALE SPEAKER: That's a non-surgical way of looking at it.

(Laughter.)

MALE SPEAKER: The only thing I would say is

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I think that whether to have the trauma with the surgery underneath, or surgery with trauma underneath, that having the surgical and non-surgical is perhaps more context-sensitive because undergoing a particular type of surgery is --

MALE SPEAKER: So on that theme, one of the things that's fascinates me is this group has basically assumed that acute pain starts when the patient hits the recovery room.

Why not start when the patient starts to have a surgical procedure? Why are we overlooking what may or may not be done during the operation that plays a role in this?

DR. BRUEHL: Surgical trauma.

MALE SPEAKER: Like when it says post-op, it doesn't say "surgery."

DR. TIGHE: We could easily revise, as Dr. Suresh also pointed out, procedural pain. And so if we looked at the surgical category and looked at procedural and classified it as from the time of the onset of tissue injury, whether it's an IV

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placement or it's a cystectomy, but also continue that general superordinate classification to include surgery and post-operative issues as well, and then the non-surgical superordinate category would have the remainder.

MALE SPEAKER: Particularly since you're looking later on here at risk factors and preventative things, I think we need to start when the scalpel comes out, not when the stitches go in.

DR. DWORKIN: So I've heard relatively broad agreement on minor modification on what's on the slide. I'm not sure I know -- and this goes back to a question Roger raised a moment ago. Are we thinking that the superordinate categories are 2 or 3? And if it's 2, which are the two?

DR. BRUEHL: Yes. Can you tell me what they are? I'm trying to write this down. Since you can't see this -- how it stands now, we have surgical/procedural with no categories under it, separate.

We have non-surgical that's neuro, disease, visceral, musculoskeletal, and trauma, and maybe

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ischemic, and those are the only two. So we have all these subcategories under non-surgical and none under surgical at this point.

Is that correct?

DR. TIGHE: Correct. But I would actually put "procedural" and "post-operative" under the surgical category, and then under the post --

DR. BRUEHL: Oh, okay. Yes. So "surgical," then under that you've got procedural and post-operative.

DR. TIGHE: Correct. And then we can distinguish that by type of surgery or other hallmarks.

FEMALE SPEAKER: That's like major surgery versus procedure. Now, you're going to have a post-procedure period, too.

DR. TIGHE: The intention of the procedural aspect is to look at the nociceptive and/or pain response that occurred during the active procedure or surgery, and the post-operative would be things that occur after the official end of the surgery.

DR. TURK: So Patrick, as the resident

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ontologist in the room, can I ask, what added value do we have of having this superordinate distinction of surgical/procedural versus non-surgical, as opposed to kind of the more level playing field on the slide?

DR. TIGHE: The two general attractions would be if we had certain attributes or constraints that we would like to apply only to the surgical setting or to the non-surgical setting that allows us to distinguish the two.

So we have our core dimensions. If there are other features we would like to consider for all surgical patients but not all medical patients, the split allows us to consider those because of the inheritance patterns.

The second is when we're doing a roll-up. So I would need to all the patients' pain scores in the hospital, and I need to make some distinctions. Having a vertical hierarchy allows me to track up or down that tree as much as I like.

Now, those are arguing for it. Obviously, against it are that it adds some certain complexity

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to this, and that is not a trivial amount of complexity. But our current experience with ontologies in EMRs is that we generally end up getting in trouble by not addressing these assumptions early enough, that we end up having to go back and do some fancy footwork to address post-op rules. And it's sometimes easier to buy a little complexity early.

DR. DWORKIN: So you think the superordinate dichotomy of surgical/procedural versus non adds value in terms of doing studies of electronic medical records?

(Crosstalk.)

DR. TIGHE: My personal belief, yes, but I don't want to minimize that there is a counterargument that's certainly [indiscernible].

DR. DWORKIN: Raj and then Steve.

DR. RAJA: I think one other aspect of this dichotomy is that except for the emergency surgery, in most other surgeries, we know when the temporal event is going to happen, while in diseases and other conditions, we're not sure when that time of

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event is.

DR. TIGHE: I think that's an excellent point. You had changed the term "surgical" to "anticipated." We know exactly when it's going to occur. Whereas, as those that are non-surgical, we're usually playing catch-up to some extent, not always but --

MALE SPEAKER: That then poses the problem of lumping trauma under surgical, which is one of the thought processes that I had when I was sort of suggesting having trauma as a third branch, is because of the predictability and a lot of the psychological and other factors, risk factors that go into trauma versus predictable or elective surgery.

DR. DWORKIN: Steve, you were --

DR. STANOS: Yes. I'm just thinking for musculoskeletal pain, for low back pain, patient has acute low back, right leg pain, radiculopathy, has a laminectomy, where do we put that in? I mean is it still musculoskeletal or is it going to be under non-surgical? I mean it's kind of --

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DR. DWORKIN: Before the laminectomy, it's musculoskeletal. At the point at which they entered John's OR, it becomes surgical, not post-surgical.

DR. STANOS: Okay, I'm just --

MALE SPEAKER: And then six months later failed back surgery [indiscernible].

(Laughter.)

MALE SPEAKER: If you could just book that, you just predict that right up front.

(Laughter.)

DR. COHEN: What is the disadvantage of having this level one be an additional dimension? Is there a disadvantage that this would be a dimension so that it could be applied to every patient?

DR. TIGHE: You lose some of the inheritance patterns in that regard.

DR. COHEN: Okay.

DR. COHEN: I like it because, again, it stresses that these are defined by context and not -- it's mechanistically bound as one might

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think looking across that list.

DR. BRUEHL: So at this point, we are not creating trauma as a separate category. It goes under medical/non-surgical; is that correct?

DR. DWORKIN: Paul?

DR. DESJARDINS: The one gap that strikes me is that multiple speakers have talked about post-orthopedic as being some of the most recalcitrant pain to treat. But yet, that doesn't explicitly seem to land in this category.

It may well be that orthopedic and musculoskeletal are close enough that we'll consider it there. But it just seems to me that if it really is a major problem, a set of disorders are difficult to treat, they don't follow as predictable a trajectory, you need more of whatever therapy you need. My question was maybe we need to pump up orthopedic a little bit.

I don't have a dog in the fight. I'm not an orthopedist but --

DR. TIGHE: So Paul, to support that, I think when we get to our work groups for looking at

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the exemplar classes for post-operative, I think many of our orthopedic surgeries will probably flow to the top as one of the first to address in a subwork group. And likely, that may occur on the non-surgical side as well. Because it's so common, it may be one of the first to addressed.

DR. DESJARDINS: Okay. I think that's a reasonable way to target it.

DR. DWORKIN: Greg?

DR. TERMAN: So Dennis may tell me that this isn't important, but this morning when we heard about trauma, we heard about burn as well. And although you'd put trauma underneath surgery maybe, I'm not sure you'd want to put burn underneath.

DR. BRUEHL: Yeah. We don't have burn up here. What does that go under?

(Crosstalk.)

MALE SPEAKER: You're missing an organ. It should go under an organ called "skin," which after all has lots of nociceptors in it.

DR. TIGHE: And it could easily be put in under trauma.

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MALE SPEAKER: Why don't you take this?

FEMALE SPEAKER: "Trauma/burn"?

MALE SPEAKER: And "burn" if you want.

(Crosstalk.)

DR. TIGHE: Would burn fall under trauma?

MALE SPEAKER: Why not?

MALE SPEAKER: At first, it was a trauma. And then subsequently, there typically would be one or more surgical procedures.

(Crosstalk.)

FEMALE SPEAKER: Fracture would be the same thing, right?

DR. TURK: Could you use your microphones so the transcriber --

(Crosstalk.)

FEMALE SPEAKER: What were the other ones besides labor and burn?

DR. GORDON: So it seems to me that there's just a couple of conditions that really stick out that fit under there that can't be classified any other way. It's probably labor pain, it's burn, and it's acute sickle cell crisis.

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DR. BRUEHL: Sickle cell --

DR. GORDON: Because labor pain is visceral medical medicine, but you can't really lump it in with all these other things, can you?

DR. TURK: Labor pain could be special populations.

DR. GORDON: Right. That's what I'm saying, burn, I think, is too.

DR. BRUEHL: Now, are special populations a modifier of the other categories or is it a separate category?

DR. TURK: It was special this morning.

DR. BRUEHL: Thank you. Very helpful.

DR. SCHREIBER: Like let's say people want to work on a particular topic. I think it'd be great to sort of flesh out these individual buckets and say what's going to go where, like is post-surgical going to -- like what's all going to include, and what is neuro going to include. You know what I'm saying? Can we do that?

DR. BRUEHL: I don't think that -- I think that was the one we were waiting for next time to

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do.

DR. DWORKIN: Right.

DR. SCHREIBER: Okay.

DR. DWORKIN: The way we did it with chronic pain is that the working groups made those decisions because they're not straightforward decisions about what are the most prevalent and/or most clinically relevant conditions to include within, for example, musculoskeletal pain or acute neuropathic pain.

So I think it would be taking some autonomy away from the working groups that will ultimately be constituted for us to do that here. By the way, we don't have the time.

DR. SCHREIBER: Okay. But looking at it another way, to define the working groups, to decide who's going to go on what working group, it may --

DR. DWORKIN: Kristin, I think we've defined the working groups, to my satisfaction, that's --

DR. SCHREIBER: No. But I mean like who's on what working group.

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DR. DWORKIN: You will be hearing from us. The answer to that question is, wait and see.
DR. SURESH: So I do have a little bit of concern about the special groups because I think we do run the risk of missing out on certain things that you would have clumped under the others.
So I think my personal bias would be to include these special groups as an addendum to each one of these subcategories. Otherwise, you know, what are you going to do, rewrite the entire thing for these special categories? It won't make sense.
DR. BRUEHL: That's actually what we did with the chronic pain group. We had a chapter on special populations that gave all the details and general things that apply across disorders.
DR. SURESH: Right.
DR. BRUEHL: And in the individual working group chapters for specific diagnoses, we are referring to that group paper unless there's something very specific to that condition with regards to kids or older people. And in context to the diagnostic description, we would put that

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information. So it's kind of like an addendum.
Now, one thing we didn't do -- and this is up for debate, I guess -- once you get in a working group is whether to have alternate criteria that were more appropriate for kids. I think in the adult chronic pain literature, the conclusion was, from the experts, that we didn't know enough to do that yet at this point. So we left it by default.
DR. SURESH: But there's a lot of data on acute pain, though, in children.
DR. BRUEHL: And maybe that is something that needs to be done differently.
Bob, I don't know if you have other things to add to that.
MALE SPEAKER: What about infectious pain? Where is that going to fit?
FEMALE SPEAKER: Medical.
DR. BRUEHL: Disease, under disease, I guess.
MALE SPEAKER: So the pulpitis, which is the most common type of dental pain, which is really an infection, that's where it would go? And otitis

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media was mentioned also as common.
DR. BRUEHL: Orofacial, is that on here?
MALE SPEAKER: Orofacial is not there anymore.
DR. BRUEHL: Oh, that got dropped off. Sorry.
MALE SPEAKER: Okay. So that's how you get there? Okay.
DR. BRUEHL: Yeah.
DR. DWORKIN: So is there any disagreement with the superordinate dichotomy of surgical procedural versus essentially other, and then a subordinate set of, I guess, it's 6 or 7 categories that you saw on Dennis' more content-filled slide, the one before this one?
Or do we have broad agreement in the room with that superordinate and subordinate structure? Because we don't have the time, and I think it's really beyond our purview to fill out the very specific set of 20-30 acute pain conditions that will be included in these seven bins.
But is there any disagreement with the

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structure that we've just finalized over the last hour or so?
MALE SPEAKER: Just a question. What happens if the hypothesis that created the dichotomy is inaccurate? We've started by lumping, but we're eventually going to be having 7 or 8 buckets, let's call them.
Maybe we should do the buckets, which are there already. And if he's right, because I think you mentioned, too, there's a differentiating factor between the two genuses, let's call them. And if you're right, then we then lump into the two.
DR. DWORKIN: Maybe I should let Patrick answer, but if one of the key drivers of the superordinate dichotomy is use of electronic medical records being facilitated, it's hard to imagine that anything we're going to do is going to invalidate that.
DR. TIGHE: Well, it also brings up a good point, though, the 1 in 12 rule. I think because we have in other, and we've already thrown a couple

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other new areas out that were not directly addressed, we're starting to get to the upper bound of how many different classes we would like. And that suggests that we need to figure out some way to split that, not necessarily critically for today but to promote further growth without having to reorganize the structure.

So by having the superordinate piece also allows us to grow without having to redo this framework.

MALE SPEAKER: I thought the main reason -- I thought Dennis said it quite well. We're going to have working groups. We're not going to have 2; we're going to have 7 or 8. So in the end, we're going to have 7 or 8 working groups.

I'm just a little concerned that we're assuming that the medical record system, the electronic medical records can't find these 7 without this dichotomy. I don't know if that's true. I've heard some people, who know more about it than I do, say that's rubbish; we're still going -- you can still locate them without the two

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genuses. That's all.

I don't think it matters at this point. I just hate to see anything published that has the two overriding genuses, and then later we abandon them because we realize they weren't necessary. That's all.

DR. TIGHE: There's no reason we can't remove them, and there's nothing keeping us. This is not an overriding constraint. We could keep it as well.

MALE SPEAKER: I'd just like to see the readership -- and there are multiple audiences we talked about that some are pedagogic, some are clinical and a lot of them are research and regulatory -- have a great response and acceptance of this and familiarity with the terminology, as well as the organization.

I don't want to turn people off by saying, oh, I don't think there should be that false -- to them, false; I'm not saying it is. But I understand the rationale for it. I just don't know if that's true. But I know later -- I mean I was

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always trained to be a lumper later.

So that's my bias. And I know from what Dennis said, his experience with Roger and Bob before, is that it's really to create the working groups, which we're going to do anyhow.

MALE SPEAKER: Yes, but if it's presented later, and people walk up, and they see those, and they see their condition underneath it, and they go, that's not visceral, that's this -- I think having the ordinate groups serve these utilities, but it also helps people understand that it's sort of a functional working group and we're not going mechanism-based, though.

DR. DWORKIN: I think at a moment like this, there are two options. We could vote on whether the superordinate structure appears to all of us to add value. The other option is we just let Patrick and Mike draft the manuscript, and then we get to see how it all looks.

I can't imagine that very many words are going to be devoted to this kind of superordinate dichotomy. And I think if during the drafting and

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revising process, we all kind of end up agreeing with Bernie, that it'll be easy enough to take out if they decide to keep it in. Does that seem sensible rather than voting on it now? It just seems like it's not something we should be voting on at this point.

Dennis is making faces, meaning he doesn't want to vote on it. So let's defer to Dennis' wisdom to defer this --

DR. TURK: Always. Always.

DR. DWORKIN: We're going to defer to Dennis' wisdom to defer this decision to the review of the manuscript that we will all be involved in.

I love ending meetings at least a few minutes early. Any other comments about the multidimensional framework, the superordinates, subordinate taxonomy that's on the slide?

MALE SPEAKER: Who's going to come up with an acronym? That was very important.

Next Steps

DR. DWORKIN: Well, I think we have the acronym.

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Okay. So just a couple of final comments. As you've heard, you're all going to be seeing a draft manuscript from Mike and Patrick. We will also, at some point in the next several months, be getting in touch with the individuals who gave talks this morning to begin to think in more detail than we had time to do today about working groups, populating working groups, next steps for working groups to develop diagnostic criteria. So that is also in the horizon in addition to in tandem with the manuscript.

I've been asked to emphasize to all of you who gave presentations to make sure to get up-to-date slides to Valorie and Andrea because if you've revised your presentation, we want your latest version of your presentation to go on the ACTTION AAAPT website rather than the version you prepared a week ago.

I'd like to thank all of the speakers on behalf of the AAAPT steering committee, ACTTION, APS, AAPM, for really a wonderfully provocative, interesting, stimulating series of talks.

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Personally, I think the talks we've had at this two-day meeting have really been better than just about any of the previous meetings in terms of the quality and the care and thoughtfulness that went into the talks.

I'd like to thank all of you for your patience and hanging in here until 4 o'clock on a Friday afternoon. And particularly, they're not in the room, but again to thank Valorie and Andrea for making sure this has been a seamless, and easy, and straightforward meeting.

So you'll be hearing from us. Email us if you have any concerns, any questions, thoughts. And safe flights home, everybody.

Finally, thank you, Patrick and Mike.

(Applause.)

DR. DWORKIN: If it weren't for the two of them, Dennis would have to be writing this manuscript, and you wouldn't see it until 2027.

(Laughter.)

(Whereupon, at 3:49 p.m., the meeting was adjourned.)

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