

**Special Topics Call  
SIDS Ad Hoc Teleconference**

***Presenters:***

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**Dr. Carrie Shapiro-Mendoza**

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Good afternoon, this is John Eichwald from the CDC EHDI Team, and I welcome you to this very special ad hoc teleconference.

I want to thank our speakers for joining in, and thank all of you for joining in at this time....

[Various introductory and logistical content omitted]

Dr. Rubens, are you ready? If you could talk for about 20 minutes or so and take a couple questions, but stay on the line.

After Carrie's presentation, we'll have another session for questions.

>> DR. RUBENS: Sounds good.

I'm a pediatric anesthesiologist at Seattle Children's, and I looked at SIDS, sudden infant death syndrome, five or six years ago. I started thinking about the possibility of a relationship between inner ear and SIDS.

Basically, I looked at what we know about SIDS, and there are a number of features which we know.

Having said that, we basically do not understand the mechanism of it, which is quite different if we look at other things in medicine, such as Alzheimer's disease or multiple sclerosis or malignancy. We know what those things are, and we're doing research to find therapies and so on.

So I felt that we needed to understand something about the way that the body works which I felt we may have missed, and that understanding will then help us to figure out the syndrome of SIDS.

So to some degree, I felt we need to go out of the box, and the reason I wanted to look in the inner ear was that the inner ear contains specialized nerve cells, as we know, for hearing and for balance. The tissue is enclosed in bone, and it's right in proximity to the brain stem with pathways between it.

Nobody's ever really looked inside there, especially in relation to SIDS. It's not part of the SIDS autopsy.

So, basically that's what started me looking in that direction.

I then thought: Well, how would one look inside the inner ear?

Every study with SIDS is difficult, because the findings are at death, so it's very hard to do something before the baby dies.

But I discussed this with the audiologists at Children's Hospital, Susan Norton that some of you may know. She suggested to go to Rhode Island -- this is three or four years ago -- because of the well-kept database and the number of years that they've been doing that there.

So I talked to Betty Vaughn and Richard Tucker, Research Assistants, and we looked at the data over a number of years. We identified 60 or so SIDS babies over a 13-year period, and went back to look at their hearing findings, which in Rhode Island is the TEOAE. Once we did that, pulled the data and matched the SIDS cases as carefully as we could, there were only 31 cases with usable data.

Based on that, we then published our findings, and if you've got the PowerPoint, one graph summarizes really clearly what we found.

We found a unilateral hearing difference on the right side, which is a difference in the signal-to-noise ratios. These infants did not fail the hearing test.

What's interesting about it is that the difference is consistent across the frequencies tested on the right side. That includes even the 1500 Hertz frequencies. So they tested the 1500, the 2000, the 3000, and 4000 Hertz, and at those frequencies, we found this consistent difference of approximately minus 4 when comparing the SIDS to control.

But having said that, we're only saying the three higher frequencies are statistically significant, because the P-value is significant only at those three higher frequencies.

The 3000 hertz is especially significant with a very low P-value. We felt that the 1500 hertz, even though the finding is consistent, wasn't consistently significant, probably because of the amount of interference from background noise interference most prominently at that point.

We're only claiming significance at the three higher points.

This is a preliminary research finding.

It points to a very interesting possibility of the potential for an early identifier for SIDS by newborn hearing screening. But it is potential; we need to do more work, do larger studies.

One of the issues I'm sure many of you are aware of is the media.

It's been quite difficult -- and important -- to get this finding out, in order to take it to the next level of research and get collaboration for that. But at the same time, it's been pretty difficult, because of the way the media put in excitement, implying that there's potentially a hearing test available at this point, which isn't the case.

As many of you are aware, this does not mean that the SIDS cases failed the hearing test. This is specifically with the transient evoked otoacoustic emissions.

We are looking at doing a follow-up study, and discussing this with Karl White at the National Center for Hearing Assessment and Management, and with a number of other audiologists around the country.

There have been a number of criticisms of the Rhode Island study. Some of these have been addressed in a correspondence back in the journal in which the original article was published in *Early Human Development*, and those letters should appear with the criticism and responses in the next few months. Some of the criticisms have stated that we did not include various risk factors, include whether the mother smoked or the prone sleeping position.

There was a question of the criteria that we used in Rhode Island to define SIDS, and whether those were accepted criteria according to various SIDS criteria that are out there.

And also, there are a number of questions about the statistics, and also the fact that we used signal-to-noise ratios rather than the amplitude or waveform data.

I do not want to go into too much detail about that here, other than to say there is we've replied to those comments, and also these issues have been very useful, because as we plan for a larger prospective study, we will consider all of these points.

We also considered that there are a number of issues with newborn hearing screening such as the huge variability in what's used, whether devices are handheld or not, the practitioners that are implementing the hearing test, whether they're OAEs, TEOAEs or DPOAEs. Many centers are using ABRs, and this is something we're looking at for the follow-up study.

It makes things quite difficult, but not impossible, and we believe that the DPOAE will also be useful in relation to what we found in Rhode Island, but the ABRs would be a different finding. There is a possibility the ABRs would show a difference in SIDS, but it's likely to be more problematic. In addition, there are papers published in the '70s that looked at that. There was one paper that found a difference; it actually wasn't in SIDS cases, but in what they call near-miss SIDS cases.

If any of you might be interested in participating in follow-up study, and if you're using OAEs of any kind, then please contact Karl or myself. We would like that.

This also brings up the opportunity in a large-scale study of this kind, that down the track, we could look at ways of perhaps standardizing newborn hearing screening in ways that would benefit everybody.

So now I'd like to move on and talk a little bit about the theory that I'm bringing to this, which is a new perspective, and what I'm calling a "unifying theory." And I'm the first to admit that this is a theory that I need to prove, and that I've got my work cut out to do that. I've got three or four other studies in the works -- two of them are underway -- to look at all of the things that I'm talking about.

But if we go back, I mentioned in the beginning that I said I looked at SIDS, and I really looked carefully at what we do know. Some of it's very simple and obvious, but some of it we can't really explain.

Basically, this is a 2- to 4-month infant -- the peak incidence of SIDS is between 2 to 4 months of age. The baby presents dead, and it's the most terrible thing for a parent, that they just find their baby in their sleep, passed away.

After 2 to 4 months of age, the incidence drops by 6 months. You do still see SIDS up to 6 months, and then it drops off quite dramatically, and then it drops off again completely after 1 year. So there's a peak at that period, and then you get a smaller incidence, and then drops off at the end of 12 months. SIDS deaths are rare at less than 3 weeks of age or one month of age. You do see them, but extremely rare. It's quite a consistent feature that you don't see SIDS deaths in that immediate period after birth.

So a theory, anybody that has an explanation of SIDS has to be able to explain that feature. It's a unique feature of the syndrome.

The babies all die in their sleep, or they're found dead in their sleep by a caregiver. I think most everybody's aware of the "back to sleep" campaign, which basically demonstrated a 40% decrease in incidence simply by placing the baby on their back rather than on their bellies.

Smoking plays a high role in SIDS; there's a four-fold increase in SIDS with a smoking mother. There's a role of overheating.

Up to 40% of SIDS babies have a cold or upper respiratory tract infection in a number of series of studies.

The incidence of SIDS is seasonal. You see more deaths in the fall and the winter.

There are racial and socioeconomic factors. There's a much higher incidence of SIDS in low socioeconomic class and also certain ethnic groups.

An interesting feature is also that you see increased deaths at extreme latitude. There's a much higher incidence of SIDS reported at least in Scandinavia, northern Europe, Alaska, even the Pacific Northwest, and on the other extreme in Tasmania and New Zealand and down in the southern hemisphere. Some people might claim that's because reporting is better in those regions, but that doesn't explain all of it.

When you look at these findings, currently most of the prominent SIDS researchers believe that SIDS is probably a mix of disorders of congenital, metabolic, rare disorders. They believe that when we look at it, we will find over time that there will be a little bit of this, some of the deaths will be from pieces of that, and that's what SIDS will be found to be over time.

But I'm actually proposing -- and this is what brought me to look at hearing and this understanding -- that if you look at these features, they're very classic and it looks more like something that is a single syndrome with various factors that interplay because of the inability to accurately detect carbon dioxide levels. It's very likely that there are risk factors and multiple factors that bear on the process at different points.

But it really seems to me that it is a single syndrome because of these features: That you've got a peak incidence at a certain period. You don't see deaths at this time. Babies lying in the prone position have a substantially increased risk. All these things to my mind point to a single phenomenon which could cause it.

In my paper, I went into some detail, and I won't say too much about it here, but I'm proposing that there's a possibility of an injury at birth when the baby is born, and this injury is very rare, because SIDS is extremely rare.

If one considers the incidence of SIDS to be 1 in 1,200 to 1 in 2,000, depending on which census you look at for Europe or the United States, and if we presume this is 1 in 400, or three times that, then it's still extremely rare.

I'm proposing there's an injury as the baby comes out of the womb and the cord is straightened, that in some instances the placenta is full of blood and that it enters the baby's venous circulation under extremely high pressure. That injury causes an injury of the small veins in various organs, and some organs would be more prone to this injury than others, including the inner ear.

There's good reason why that would be, and I've explained in my paper why that would be. Also, certain areas of the brain stem may be involved. We're not talking about massive bleeding, where the baby would die from hemorrhage, but small bleeding in the tiny end veins of certain organs.

I'm proposing that the inner ear would be one of those organs, as well as the brain stem areas, and other areas may be variable, depending on features of this high pressure surge.

Smoking may also play a part in that. If you consider the flows and pressures and what we know of the effects smoking has on vasospasm, that might play a role.

So having said that, going back to the paper, we found a right-sided finding, but I don't believe that there's only injury on one side. I believe that the injury is bilateral.

With the TEOAE, at the level of the testing which is currently used, there's likely to be a more widespread injury on the right side, and that's why we're picking it up at those frequencies.

So I'm also proposing if we were to look at the higher frequencies, above 4000 hertz -- again that's quite difficult to do, but not impossible -- we'd find there's damage on both sides, and I believe that SIDS needs to have damage on both sides or SIDS wouldn't occur.

And it's not just the inner ear that needs to be affected, but the brain stem, as well, because if the brain stem didn't suffer from this injury, then the baby wouldn't be vulnerable to SIDS.

So I'm proposing that there's an injury at birth that this placental surge of blood, and it's not necessarily only because the placenta is so full of blood. In every baby that's born, there's a delivery of blood from the placenta into the baby being born. That's necessary because as the lungs open up, we need blood to fill it.

What I'm proposing is that in some instances that's a high-pressure injury, and that, of course, is the problem.

So the inner ear is vulnerable because the great veins, big veins, that carry the blood from the placenta through the heart up to the head can carry this kind of pressure. We know when you cough or sneeze, you have pressures up to 100 millimeters of mercury. The big veins can hold the pressure, but the small veins can potentially rupture and bleed.

I absolutely agree I have to prove this, and nobody's proven or disproven this at this point, but I have my work cut out to prove it.

For the inner ear, you have these tiny little veins or small veins that come directly off the carotid vein into the inner ear, which is soft tissue, highly specialized nerve tissue, susceptible to hypoxia with some bleeding, and then swelling responds. This would damage the cells of hearing and the vestibular hair cells and impair them.

That's fine, but why isn't there a problem in the first three weeks of life?

What I'm proposing is that damage to the cells of hearing lead to the marker, or potentially lead to the marker, of the hearing cells. The subgroup of the vestibular hair cells that damage impairs is the issue in relation to the baby being unable to detect rising levels of carbon dioxide.

I think at this point this sounds "out there" -- and nobody's ever mentioned vestibular hair cells could be involved in this, but that's what I'm proposing. I'm currently

undertaking an animal study in infant mice of approximately the same age as human infants coming from SIDS, and I'm damaging the vestibular hair cells. We're then looking at their response to carbon dioxide.

Just very briefly, to explain why the deaths wouldn't occur, or why that might not be an issue in the first three weeks of life, there is evidence from animal studies in lambs that shows that the control of breathing in those first few weeks (in fact, up to 8 weeks of life) is still under a separate fetal control of breathing. This is anatomically different from the pre-Botzinger, which controls the respiratory pacemaker in the born human as opposed to the fetus. So there's the remaining fetal control.

There are many other issues, as well, that explain why SIDS doesn't occur in three weeks, but a key issue is the fact that this fetal pacemaker is intact and operates up to 8 weeks of life, and it's used to high levels of carbon dioxide, because that's the case in the fetus. So its pacemaker keeps the SIDS baby breathing regularly at a rate and a tidal volume, the excursions of the lung, which are great enough to allow the baby to expire carbon dioxide.

After three weeks or so, that ability drops off. Then creeps in the normal or the post-birth response which we all depend on, which is the peripheral chemoreceptors, the carotid bodies. That system does depend on the ability to respond to rising levels of carbon dioxide.

And I'm proposing with the inner ear damage, as well as the brain stem damage, prevent that SIDS baby from detecting the rising carbon dioxide levels. It's not detecting that because that information is transmitted from the peripheral receptors via the inner ear to the brain stem. Of course, that's a theory that I need to prove, and I'm hopefully in the process of doing that.

So the transmission is cut off, and the SIDS baby, as the carbon dioxide levels rise, is not detecting that, and the child falls into a fatal cascade.

I don't really have time to talk in more detail, but another feature is that anybody with a theory about this has to be able to explain what's happening to a SIDS baby 20 minutes before they die, 10 minutes before they die. I'm hoping that with this animal study that I'm doing, we will be able to have a clearer picture of that, and then look at ways of preventing it.

>> JOHN EICHWALD: At this point, we'll take one or two questions, and then we'll have our next speaker.

>> DR. PICKARD: Can I ask a question? This is Dr. Pickard in Miami.

>> JOHN EICHWALD: Please, go ahead.

>> DR. PICKARD: Just to clarify something you mentioned in the first sentence or two that you said, Dr. Rubens, those 31 babies that you studied, they're babies who died from SIDS, whatever that is.

And they passed or they failed the infant hearing screening?

>> DR. RUBENS: They did not fail a hearing screening test.

>> DR. PICKARD: So in other words, to extrapolate backwards, which is what the media amplifies, was that somehow we could make a jump from this data, which is a statistical study of the omission studies, versus one ear and the other?

That's basically it; is that correct?

>> DR. RUBENS: Yes, but at this point in time, and I made it as clear as possible, we are not in a position to do that.

It doesn't mean that with time and with looking at this in more detail that there may be a way of doing that.

But at this point, there absolutely is not a hearing test available.

>> DR. PICKARD: Thank you.

>> DR. SININGER: This is Yvonne Sininger at UCLA.

I wonder if I can just make a comment.

We did the study that showed the asymmetry in newborns' OAEs, that, in fact, in a large group of babies, you see a bigger transient evoked otoacoustic emission in the right ear than you do the left.

And I think rather than looking perhaps that the hair cells may have been damaged, that it might be worth considering that the mechanism imparts that asymmetry which we think is the olivocochlear efferent system may not be functioning properly in those babies.

So if you don't have this efferent system in the brain stem that is somehow creating an asymmetry, then you get what you would have gotten, which is symmetrical responses from the left and the right ear.

And that to me makes more sense than the fact that there's a slightly reduced OAE doesn't really imply that there's any damage to the hair cells, and you've made that point.

Could it not be that perhaps in a normally functioning baby, you see the efferent system somehow suppressing the left ear transient emission, and/or augmenting the right ear, but you're not seeing that in these infants, and that the brain stem may be more the mechanism?

>> DR. RUBENS: I take your point.

And keep in mind that the asymmetry for the SIDS babies is in the other direction on the right side. We made that point in the paper.

>> DR. SININGER: But they do look somewhat symmetric.

>> DR. RUBENS: Right, but the SIDS hearing is negative on the right side. It's minus 4.

>> DR. SININGER: Right, but the left and the right ear are both passing, and they're both --

>> DR. RUBENS: Correct.

So it's a subtle finding, but consistent, and I'm not claiming anything different at this point. Time will tell.

We need to repeat this in a larger study, but also I think it would be really useful to look at the higher frequencies for that reason.

And also by looking at the animal study I'm doing, looking at a connection between vestibular hair cell function and the respiratory response to elevated levels of carbon dioxide.

So that will be interesting, and we'll have to see what that shows, but I take your point with that.

>> JUDY HARRISON: Hello, this is Judy Harrison from A.G. Bell.

I'm curious if there's any correlation or lack of correlation with babies who were born through cesarean section and did not go through labor?

>> DR. RUBENS: That's a very good question.

There's been some SIDS studies that have looked at cesarean section, but they're not -- a lot of things actually about SIDS, one study finds something, and then another doesn't find that finding, so it's not clear.

What I would propose -- and again, it's all based on this theory -- is that a baby born by a cesarean section would still be at risk of SIDS, because it's not just the mother pushing, or it's not just the placenta filling with blood that's the issue, although that would obviously play a big part in it.

But what would be really interesting would be to look at elective cesarean sections where the mother has no contractions whatsoever to see if there's a -- according to what I'm suggesting you would find, there shouldn't be SIDS in mothers that have an elective cesarean section.

Now, that's not something that I go claiming, obviously, now, because there's good reasons to have a cesarean section.

It wouldn't be appropriate to say if you had an elective cesarean section, you wouldn't have SIDS, but that definitely would be data that would be useful to look at.

And we have considered it, but it's actually not quite as simple as you'd think to simply collect that data, because you'd have to be really sure that it's an elective cesarean section, meaning the mother is not having any contractions, but you really have to be sure she wasn't having contractions to make that a true study, and that would be a good study to do.

DR. SHAPIRO-MENDOZA: I'm Carrie Shapiro-Mendoza, an Epidemiologist in the Division of Reproductive Health at the National Center for Chronic Disease and Public Health Prevention at the Centers for Disease Control.

My research interests include SIDS and infant mortality, as well as health outcomes of infants born late pre-term. I've been at CDC about four years. In the past four years, I've initially co-lead and am now leading the Sudden, Unexpected Infant Death

Initiative which has advanced both research and programs to better understand and prevent SIDS and other sudden unexpected infant deaths (SUIDs).

So in today's presentation I will give you some background about SIDS and classification of other sudden, unexpected infant deaths. I'll talk about the research we've done at CDC, tell you about our SUIDI, or Sudden, Unexpected Infant Death Initiative, and then tell you a little bit about the accomplishments to date related to our initiative, and some of our future plans.

Basically, the definition we go by here for SIDS, or sudden infant death syndrome is that SIDS is the sudden death of an infant under one year of age which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history.

You can't have SIDS unless you've got a really good death scene investigation and an autopsy, and have reviewed the clinical history of the deceased infant. This definition was published in 1991 by a national workgroup up individuals representing both federal and non-federal agencies.

You have heard me use the term sudden unexpected infant death or SUID. These are a category deaths that also include SIDS. It is often very difficult if not impossible to differentiate SIDS from other SUID without a thorough death scene investigation.

Other examples of SUID include:

Suffocation, such as that caused by being asphyxiated by soft bedding or a pillow, or perhaps a water bed mattress.

Overlaying: When a parent or caregiver or sibling rolls over on top of or against a baby while sleeping.

Wedging. That could be the baby gets stuck or caught between the mattress and a wall, or a mattress and a bed frame and her airway is restricted.

Strangulation between the bed railings, or in between the side of the bed and the wall.

When you're considering a diagnosis of SIDS versus another SUID diagnosis, you need to also consider homicide, hypo- or hyperthermia, metabolic disorders, or poisoning as other potential causes of death.

Some of the modifiable risk factors for SIDS that are amenable to change include smoking and substance abuse, especially prenatal maternal smoking, post-natal exposure to cigarette smoke, being face down on a sleep surface, prone or side sleeping position, , sleeping or placement on soft surfaces and loose bedding, overheating, as well as bed sharing, especially with an intoxicated individual, a caregiver who smokes, or somebody other than the main caregiver such as a sibling or babysitter.

Rates of SIDS have declined in recent years.

If you look at the handout that I gave you, you see a graph beginning with 1980 going through 2004. You see rates have declined, the biggest decline occurring in the early 1990s, when it went from about 130 SIDS deaths per 100,000 live births down to 2005, we're at 55 per 100,000 live births.

During the years between about 1992 to 1996 when the "Back to Sleep" campaign happened, and when there was a lot of encouragement for parents to put their babies on their back to sleep. You see the biggest decline there.

Why should we be concerned about SIDS and other sudden, unexpected infant deaths?

SIDS is the leading cause of postneonatal mortality. In other words it's the leading cause of death in children from one month to one year of age, the third leading cause when you look at overall infant mortality, and only deaths caused due to birth defects and pre-term birth are higher as far as leading causes of mortality among all infants.

Infant death rates attributed to accidental suffocation and strangulation in bed have tripled in the last decade. Suffocation deaths have gone from 4.1 per 100,000 live births in 1994 to 12.5 deaths for 2004.

And if you were to combine all these SIDS and other SUID rates together, we get rates that are very comparable to birth defects mortality rate. There are about 4,600 SUID deaths per year. And 2,500 of these, the majority, are SIDS deaths.

Some of the research we've done here were published in the "American Journal of Epi" in 2006.

What we found was that in recent years, from about 1999 to 2001, the decline in SIDS is being offset by increasing rates of two other causes: Cause unknown or those unspecified, and deaths attributed to suffocation, wedging, and overlaying in a sleep environment.

We think this change in classification is likely explained by how investigations are conducted, and how these causes of death are reported and classified, or how these diagnoses are made.

In the next slide, you see some of the slides from the published in the article I mentioned in the "American Journal of Epi."

You see the time period 1989-2001. You see the rate per 100,000 live births. When you look at the top line, that's the SIDS rate, and the bottom line is a combination of other SUID deaths plus unknown/unspecified causes.

You can see for SIDS that there was a decline, especially during the years prior to '92 and after '96. There is no data between '92 and '96 because I used birth and death certificates that were linked, and data wasn't available during those years.

So you see SIDS declining, when you look at the bottom line, that's the rate of the SUID and the cause unknown, you see it stayed pretty stable up until about 1980 -- 1998. Then you see it beginning to rise.

When you look at the next slide, I've added a line on top, and that combines the two bottom lines, the SIDS plus the sudden, unexpected infant death and unknown. When you combine them, you see the top line looks pretty much like the SIDS line up until about 1998. Then you see it beginning to flatten.

So what that's telling me is that the decline in SIDS during those later years from 1998 to 2001 is being offset by the increase in the other SUID and unknown cause.

Based on a more recent assessment, I can tell you this trend has pretty much been continuing through 2004, the latest year we have data.

But why should we be concerned about this change in SUID reporting?

First, you should know that many SUID deaths are not investigated. When they are investigated, there's not always standard practices of data collection that are followed to investigate these deaths.

Then, also, the other problem is there's inconsistent classification and reporting of SUID deaths. These deaths may be classified by Medical Examiners or Coroners. Every state has different systems for investigating deaths, and some states have only Medical Examiners who are Board Certified pathologists. Other states have elected officials who may be Coroners that don't have any medical training at all. They may be the Funeral Director, they may have a GED. They're filling out death certificates and establishing cause of death.

So if we want to reduce these infant deaths, we need to really improve the data that we have. We need more data that is valid and reliable that will support any future research and prevention efforts. So we need better death scene investigations. Better death scene investigations are also helpful to the pathologist when he's conducting an autopsy. He needs that data to better inform his diagnosis.

Better death scene investigation is also important for child death review committees to identify areas in need of prevention. Better death scene investigation is also important for SUID cases that are suspected to be homicide-related that could be tried in court.

And when we do have a complete death scene investigation, we'll have improved classification and reporting of SUID, and then with this more valid and reliable data, we can monitor trends in SUID, we can conduct more valid research to identify risk factors for sudden infant death syndrome, we'll be able to design interventions to prevent these deaths, and have better data to inform evaluation programs aimed at prevention.

So a little bit about the initiative and the activities.

First of all, we had four goals.

The first goal was to standardize and improve data collected at the death scene, again based on the research that we conducted.

Second, to promote the consistent diagnosis and classification of cause of death.

Third, we wanted to improve national reporting of SUID so we could have better estimates when monitoring rates over time.

And the final goal is to prevent SUID by using this improved data to identify those at highest risk.

We also had several activities that were part of our SUID initiative. The main activities included a revision of a 1996 death scene investigation form. It was known as

the SUIDIRF. The SUIDIRF was released in 1996 through an MMWR and feedback was it wasn't being used to a great extent. It wasn't user friendly. I'll go on to some of this in more detail in a minute.

Another activity of the initiative was to develop a training curriculum and materials.

A third activity was to disseminate and promote the use of the SUIDI Reporting Form that was revised, as well as promoting and disseminating the training curriculum and materials.

And finally, another activity was to pilot test a SUID case registry.

So the first activity was revision of the 1996 form. We had a big national work group that included medical examiners, coroners, law enforcement, researchers, parents, SIDS organizations, everybody interested in SIDS prevention. They all got together over several days over a period of a year, and we revised the SUIDI Reporting Form. Our aim was to make the form more user-friendly and get people to use it. We pilot tested the form in Washington State and in Georgia, and feedback was pretty positive. And then after all the pilot training, we released the newly, revised form now known as the SUIDI Reporting Form in March of 2006. This form is not mandated. We can only encourage its use.

The next thing we did was to develop a training curriculum and materials. Again we formed a national steering committee and a work group. These groups were made up mostly of people who taught law enforcement or death scene investigator or medical examiners about how to conduct a death scene investigation or information that would be needed to conduct investigation or an autopsy.

We tried to put together a curriculum that would teach people who conduct death scene investigations on how to conduct a comprehensive death scene investigation, what are the elements that there are, give them a step by step demonstration on how to do a scene reenactment and how to interview grieving families and caregivers.

In the case of a lot of these law enforcement people, when they go to interview a family, it's more about interrogation. That's what they're used to doing. We tried to get them to understand these SIDS deaths were likely not homicides. The great majority of them will not be homicides, and that an interview and not an "interrogation" is needed with a family if you wanted to get good information.

Finally, our aim in the training was to help differentiate SIDS from suffocation and other sudden infant deaths by carefully examination the death scene.

Our next thing was to disseminate and promote the use of the SUIDI materials. We hired a contractor, Steve Clark, and we have a National Training Coordinator, Terry Davis, and we conducted -- we're in the process of conducting -- 5 regional train the trainer academies. The training academies train multidisciplinary teams of 5 individuals from every state. The 5 individuals included a Medical Examiner or a Coroner, a death scene investigator, somebody from law enforcement, somebody who teaches at a university or college to this group of people, and a child advocate, usually somebody from a child death review team.

The goal was to train 250 individuals, to have 50 teams of 5 members, and they would go on and train 5 people. So the goal was to get 1250 people trained nationally. To date, we've had 4 national training academies. We have one more to go in Seattle, Washington, in 2008. We've trained 237 faculty, staff, and state team people as train the trainers. And they've actually gone on, and only about a quarter of them have reported to us that they've gone on to train up to 10,000 other participants.

So our goal was only 1,250. We've got more than 10,000 people trained and still have one more training academy to go.

Also, in Tennessee -- I don't think this was included in the 10,000 -- they've trained 871 law enforcement, 1136 EMS, and 983 fire personnel in the last 18 months since they've attended the training.

Washington State has gotten real excited, and they haven't had the training yet, but they've developed plastic-coated message cards based on the training curriculum. The cards help remind investigators about the procedures to use for sudden, unexpected infant death investigations.

We've also had a lot of international interest. England, United Kingdom and Australia attended our Boston training, and several other countries have either adapted our forms or have expressed interest.

Terry Davis has done an awesome job of getting endorsement of the SUIDI training by major organizations who this is important to, such as NAME, the National Association of Medical Examiners, the National Sheriffs Association, International Association of Chiefs of Police.

You have the list there, and you can see that for yourselves, but this is really an important part, since we can only encourage and not mandate the use of the form or the other training materials. If we have support and endorsement from these groups, then maybe their members will be encouraged to use this form and other CDC resources.

The Indiana Council on Infant Health and Survival recommends the CDC Protocols and Reporting Form.

>> DR. BARNES-JOSIAH: Dr. Mendoza?

This is Debbie Barnes-Josiah from the Nebraska Department of Health. My question was for Dr. Rubens.\*

Based on Dr. Shapiro's presentation, based on the number of injury deaths that are suffocation deaths that are mixed into SIDS deaths, you don't do a very thorough examination.

What impact do you think that has on your findings from Rhode Island? And also the impact for using this idea further?

>> DR. SHAPIRO-MENDOZA:

Let me just finish and say we also have developed a case registry where we want to learn more about the circumstances of infant deaths, so we've tried to pilot this by collecting information we have from the death scene investigation and death certificates, and doing this at a state level.

Now if you have any specific questions for me, please feel free to ask.

>> DR. BARNES-JOSIAH: This is Debbie Barnes-Josiah again.

How do you think these mixed data would impact this possible finding on hearing?

Which is very exciting.

>> DR. SHAPIRO-MENDOZA: I think it would be very important to consider how those cases in Dr. Rubens' study were identified.

If I recall, they were based on death certificates, and the deaths occurred over a period of time when there was a lot of change. The study period included times before and after the 1996 SUIDIRF version was released.

I'd want to know more about how in Rhode Island they conduct death scene investigations, especially over this long time span to know I truly had SIDS cases and not suffocation cases or anything else in my case population I was using in my study.

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[ \* For technical reasons, Dr. Rubens was unable to reply at the time the question was asked. He subsequently offered the CDC EHDI program the following response to the original inquiry:

The criteria for the diagnoses of SIDS made in the Rhode Island Medical Examiner's office were based on the following definition developed by an NICHD Expert Panel and published by Willinger et al<sup>1</sup>: "the sudden death of an infant under one year of age which remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of the clinical history" In addition, bacteriological cultures were obtained for all cases as was consultation in neuropathology and cardiac pathology.

1. Willinger, Marian, L. Stanley James, and Charlotte Catz. "Defining the Sudden Infant Death Syndrome (SIDS): Deliberations of an Expert Panel Convened by the National Institute of Child Health and Human Development." *Pediatric Pathology* 11 (1991):677-684.]

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CALLER>> I have a question for Dr. Mendoza.

I'm calling from Michigan State Pediatrics Residency Program. I'm a Resident.

All the discussions you had about the education and training for someone like me, is there any way for me to find out if there are opportunities in my state with any organizations or agencies that may be involved with that?

>> DR. SHAPIRO-MENDOZA: Sure, we have already trained a team of 5 from Michigan and those individuals should be available to provide additional training. You could either write me, and I think I gave my e-mail at the end of the handout. Or contact Teri Covington. She's the Director of the Child Death Review Program in Michigan at the Public Health Institute.

My guess is -- Teri's been really involved in promoting our training materials and curriculum, and she could tell you who the team was that was trained, and where you could go to get this training from the train the trainers.

If she's not helpful, you could always just e-mail me and ask me, and I'll refer you to Dr. Terry Davis. She could help you out.

Anybody else have any questions?

Okay, if there are no more questions, I want to thank you all for your time and attention to this what for me is an important topic.

[End of Call ]

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