TRACK 3 32:35

8 total speakers here. Known: **Alice Cronin-Golomb of BU, Robert Nebess from U Pitts, and Larry Bell.** This must be at APA Tribute to Sperry in California. If I recall correctly, Larry Bell and Nebess spoke in one of the tracks I transcribed in Spring.

Speaker 1 (??):

Okay. That was the... I guess that's pretty much it. I'm done on time!

Speaker 2 Presenter:

I'm going to hold all the questions to the end of this session. You can [INAUDIBLE 00:00:37]. Thank you very much [INAUDIBLE 00:00:41]. Our next speaker is Alice Cronin-Golomb of Boston University. Her topic is the impact of visual dysfunction on cognition and Alzheimer's disease. Alice. Will somebody... You can operate the machine from here out.

Speaker 3 (Alice Cronin-Golomb of BU; worked with Sperry as grad student '79):

[INAUDIBLE 00:01:21] really screw up and go over. I wasn't sure how a talk on Alzheimer's disease was going to sit in a Sperry symposium. I'm now relieved to realize that I'm in the good company of people like Bob Nebes, Evelyn Tang, Larry Johnson, other people who are working in dementia [INAUDIBLE 00:01:39] college research. When I was with the Sperry Lab as a graduate student in 1979 to '84, one of the things I was interested in was differential perception, perceptual ability of the two hemispheres and how that might impact on [INAUDIBLE 00:01:54] vision. And I'm still very interested in that, only now in the context of Alzheimer's disease. So, let me just tell you that, first of all, for some patients with Alzheimer's or AMD, some call it, there are visual dysfunctions that are very obvious. They can be among the earliest and most striking symptoms. But in most patients with AMD, [INAUDIBLE 00:02:19] come into the clinic complaining of color vision deficits, depth perception deficits, that sort of thing. So, the visual status in these clinically asymptomatic cases was something we felt we needed to investigate. We've conducted several studies of vision in AD, with two goals in mind. First, to describe a wide variety of visual behaviors in AD, and secondly, to try to determine the cognitive [INAUDIBLE 00:02:43] of any vision deficits that we might see. My principal collaborators on these studies were Suzanne Corkin, John [INAUDIBLE 00:02:51], and Joe [INAUDIBLE 00:02:52]. Can I have the first slide, please. You just have to turn... Push the button all the way to the right.

Speaker 2 Presenter:

It says "power".

Speaker 3 Alice Cronin-Golomb of BU:

Thanks. So, the patients I studied were... Can I get the lights down a little bit, please. There were 53 patients diagnosed with probable Alzheimer's disease according to standard criteria. 35 elderly control subjects, [INAUDIBLE 00:03:23], and 11 young control subjects, [INAUDIBLE 00:03:25]. The two elderly groups were matched for age and all three groups were matched for education level. We had a wide variety...a wide range of dementia severity represented in the [INAUDIBLE 00:03:37] as shown by the [INAUDIBLE 00:03:39] dementia scale. I want to emphasize that none of these patients were chosen on the basis of visual symptomatology. The only criteria for inclusion in the study was consecutive appearances at the clinic. Thank you. 25 of the Alzheimer's patients from this group were studied in some detail at the Massachusetts Eye and Ear Infirmary, where they received neuro-ophthalmological and electrophysiological testing. The result of this testing showed that there was nothing in particular that was abnormal about the group. We considered them normal as far as these tests go. The tests I'm going to be describing are as follows. Central acuity revealed, say, that they were normal. Being new patients, they had the same range of acuity scores that the control subjects had. This is corrected acuity. And I'll be describing the results with these other tests.

Okay. First, I want to mention that on a couple of the tests, the absolute level of performance for the Alzheimer patients was worse than in either control group, and one of these tests was stereoacuity. In

stereoacuity, the goal is to indicate which of the three circles you see there appears to stand out in-depth when you're looking through polarizing lenses, which are in that lower left corner, but you can't really pick up that those are supposed to be glasses. What it's measuring inside the mark is the binocular disparity needed to perceive depth. And these are the results of that. The Alzheimer group is on the right. You can see that the threshold for stereoacuity is significantly higher than for the other two groups, which don't differ from each other. A bar is one standard deviation, so you can also see that the variance in the Alzheimer performance is much greater than in the groups, but stabilizing the variance didn't change the results, so they simply are worse at depth perception across the board.

The other test on which they were just worse overall was backward masking. Now, in this test there are eight letter stimuli that appear for durations ranging from 17 to 300 milliseconds. The letter then disappears and is immediately replaced by a mask for 100 milliseconds. The mask disappears and the subject has to identify the letter. In half the trials, the mask is homogenous, which is the central picture you see there, and in half the trials it's a pattern mask, the lines of which correspond exactly to all the contours of the letters. The pattern masking condition is much more difficult. Both of these are thought to assess central visual processes. Now, the threshold here, the y axis, is the letter duration in 17 millisecond units. So, it's showing that if you have a higher threshold, you need to have a letter on for a longer duration in order to escape the effect of the mask and correctly identify the letter. So, again, you can see that the thresholds for the Alzheimer patients are significantly higher than for the other two groups, which don't differ from each other. And, again, stabilizing the variance did not change the results. The pattern masking condition is just harder for everyone, and you can see that in the group on the right as opposed to the left.

So, those are the two tests [INAUDIBLE 00:07:04]. They just seemed to have much more difficulty than the other groups. Now, there are some tests on which the pattern of performance as well as the absolute level is different for the groups, and the first one I'm going to talk about is contrast sensitivity function. Stimuli that vary in contrast level are extremely difficult to reproduce photographically, and as I'm sure you'll agree by looking at this chart, but I wanted to give you an idea of the task conditions. So, this is the chart that they would be looking at. The task is to indicate the line orientation — either vertical, slanted to the left, or slanted to the right. There are five spatial frequencies represented going from top to bottom — the low spatial frequencies to the high frequencies, so the lowest being 1.5 and the highest [INAUDIBLE 00:07:51]. Contrast varies from left to right across rows, so each patient has five scores. One for each spatial frequency. And showing the final circle of the final contrast level at which they could reliably indicate the line orientation. And this produces what we call a contrast sensitivity curve. The first thing that should strike you here is that the shape of the curves is very similar for all three groups. Everyone is more sensitive to the middle frequencies than to the lower or higher frequencies. But you should also notice, if you look carefully, that there are differences among the groups. If you look at the top line, which is the young control subjects, and contrast that with the middle line, the elderly control subjects, what you should notice is that on your left, the lower frequencies, they're performing the same. But as you go up toward the higher frequencies, you should see a divergence, and that's a normal aging effect — that as we age, we get less sensitive to higher frequencies. Now, contrasting that with the bottom line, the Alzheimer patients, what you should see is that they are impaired at all frequencies. In fact, more aging patients had trouble with the lower than the higher frequencies, so this is not just an exaggeration of the normal aging patterns.

The other test on which we saw a pattern difference is color discrimination. This is the City University color vision test. There is one correct answer; that is, you point to the circle on the outside that is closest in hue to the central circle. None of them are identical. There's one correct answer and, of the three possible errors, they correspond to the three kinds of color vision deficits. If you have normal color vision, [INAUDIBLE 00:09:38], you should point to the one on the right. They're both green and they're close in hue. If you have what we call a protan deficit, a red-range deficit, you might point to the one on the left. If you have a deutan, green-type deficit, you would point to the bottom one. That's the usual congenital color vision deficit. And the one on the top is called a tritan or a blue-hue deficit, which is relatively rare.

20 of the 37 Alzheimer patients we tested showed deficits on this test [INAUDIBLE 00:10:13] error on the test. And, as you can see here, the errors did not distribute evenly. They had significantly more tritan or blue-hue errors than either protan or deutan, which did not differ from each other. The control groups made very few errors on the test, and those errors distributed across the three possible [INAUDIBLE

00:10:33] types. Now, we were extremely surprised by this result. Nobody had ever recorded that Alzheimer patients have a blue-hue deficit, but many clinicians were using tests like the Ishihara, which just look at red and green color vision deficits [INAUDIBLE 00:10:49] congenitally. So, they weren't looking for blue. But we still thought it was strange and maybe [INAUDIBLE 00:10:54] of this particular test, but we've since replicated these findings with two other color vision tests, the Farnsworth [INAUDIBLE 00:11:01], so we think... And another group have already replicated this, so we think it's real.

The final test I want to describe here is critical flicker fusion, which is defined as the frequency at which flickering light is first perceived to be a steady light. So, if I were to flash the lights on and off here very quickly, if I did it fast enough, it would appear to you to be a steady light. And at that point where it changes from flickering to steady, that would be your threshold. A higher threshold is better performance. Now, unlike the other tests, you'll see here that the Alzheimer group is really quite the same as the elderly control group. The thresholds are the same. The variance is the same. They're not performing at ceiling level because [INAUDIBLE 00:11:44] are impaired relative to the young control subjects, but they're just normal on this, and this is rather reassuring to us, that we found a test that they were normal on, that's not just a general cognitive deficit that's driving these results. Dementia severity correlated with performance on some tests but not others. So, for backward masking and the two low frequency contrast sensitivity, those correlated with dementia severity. The three color vision tests, performance on those tests—on stereoacuity, on critical flicker fusion, and on the high frequency contrast sensitivity—there was no correlation, again suggesting that it's not a general cognitive deficit that's driving the results.

The conclusions so far are that the patients with AD showed significant selective deficits in visual functions assessed by these tests. They showed normal results on the neuro-ophthalmological and electrophysiological examinations, and we tentatively conclude—or infer, I should say—that the observed deficits are not attributable to [INAUDIBLE 00:12:51] defects, but rather we would be inferring that it's the visual cortex that might be the problem since we know that [INAUDIBLE 00:12:58].

The second part here, very quickly, is: What's the effect on cognition for these kinds of visual deficits? The same patients received multiple tests of cognitive function. I'm not sure how well you can read this here, but you'll see that there are multiple cognitive tests on the left, and then the best vision predictor of those...performance on those tests. And what I want you to notice [INAUDIBLE 00:13:27] is that pattern masking and low frequency contrast sensitivity predicted performance on a number of cognitive tests. The color or the tritan hue deficit predicted performance on the Stroop color subtest. So, if any of you are using that test to look at frontal lobe function of Alzheimer's patients, you should know that they confuse the blue and green ink colors, so that affects your results.

Okay. How well do they predict performance? On these tests here, over 25% of the variance, up to 57% of the variance, on the cognitive test was accounted for by visual performance. On these tests, less than 25%, as low as 7%, of variance was accounted for by visual performance. Do you know any of these tests at all? If you don't, I can tell you about them later. You might notice that in this group are included tests that are sensitive...or associated with the dorsal visual processing stream — things like map reading, mental rotation, though possibly involving posterior parietal cortex on one hand. And going back one, these are mostly tests of object identification, object recognition, which are associated with the ventral visual processing stream, including the inferior temporal cortex, on the other hand. So, our tentative interpretation of this is simple. We know that there's extrastriate damage Alzheimer's disease that's showing itself in the visual deficits that I've described here, and that we believe that those kinds of deficits are impacting more on the cognitive capacities of the ventral than the dorsal visual processing stream. Thank you.

[Applause]

Speaker 2 Presenter:

I'm sure you have a lot of questions, and we'll get back to it as soon as we have our next speaker. That was very interesting.

Alice Cronin-Golomb of BU::

Thank you.

Speaker 2 Presenter:

Yeah. Our next speaker—we're running slightly behind—is Robert Nebes from the University of Pittsburgh. His topic is [INAUDIBLE 00:15:41] in Alzheimer's disease and geriatric depression.

Speaker 4 [Robert Nebess from U Pittsburgh]:

Could you put the other tray back in?

Speaker 5 [?]:

Side tray?

Speaker 4:**Robert Nebess from U Pittsburgh:**

No, the tray that [INAUDIBLE 00:15:53]. I'm probably going to leave the lights on. Like a lot of the other speakers in this session, I have wandered very far from the path of the callosum that I took when I was in Sperry's Lab back in the late 1960s. However, Jay Meyers pointed out yesterday that all of us picked up a number of things from working with Sperry, one of which is we never went into his office and said, "Gee, I just read about this task. I wonder what would happen if we gave it to an Alzheimer patient...the [INAUDIBLE 00:16:32] patients," because he'd immediately say, "What's the question? What are you going to learn? Who cares? Convince me that you're going to answer an important question." So, you learned very quickly you didn't go in with a technique; you went in with an issue, and then you came out with a technique [INAUDIBLE 00:16:48] interesting answer of the question that you wanted to answer. And so we didn't [INAUDIBLE 00:16:53].

What I'd like to do is start off first with the question why anybody would be interested in looking at response time and geriatric depression in Alzheimer's disease. Well, first of all, if you're interested in Alzheimer's disease, which has become a big topic lately because with my generation, [INAUDIBLE 00:17:10] security system. There's going to be a lot of trouble because of the big [INAUDIBLE 00:17:16] of people moving into old age fairly rapidly. So, there's going to be a lot of Alzheimer's disease, and so there's been a lot of work in it. But if you start looking and are interested in the cognitive deficits associated with Alzheimer's disease, you soon find out that especially in the early stages, there's a lot of other conditions that give symptoms that are very similar to early Alzheimer's, including depression. Depressed geriatric patients often have trouble with their memory. They may have trouble solving problems. They have attentional difficulties. And so, you know, there's this sort of similarity of symptoms. There's always been an underlying view among researchers that, well, the symptoms may look the same, but the mechanism is different. There are different causes. It isn't just that the same brain systems are dysfunctional. In geriatric depression, they are...being reversible with treatment. But there's something different, and one area that I was looking at, which is behavioral [INAUDIBLE 00:18:17], which is certainly a common symptom of Alzheimer's and depression, there has been some suggestion that the underlying causes are different, that Alzheimer patients are slow to respond. If they do the task, you'll find that they're usually very slow not because they just are slow to make motor responses, but because they are thinking slower. They actually have a slowing [INAUDIBLE 00:18:41] information processing, whereas the geriatric depression subjects are slow not because they're thinking slowly, but just because they're slow to get the response out. It's motor retardation. And since all you're measuring is the time between the onset of the stimulus and the onset of their response, you're going to come up with them being slow.

Now, one set of data that supports this came from an earlier study from our lab, which used an approach in which subjects are asked to repeat the same mental operation multiple times. Now, by looking at how reaction time changes as a result of number repetitions of [INAUDIBLE 00:19:22], we can get an idea of the rate at which they can carry out this operation. Now, the best example of this for those of you who do this sort of work, this is Sternberg memory set task in which the subject is given two, three, four items to remember, and then they're given a single item and they have to say yes or no, whether it was a member of the memory set. So, five, seven, nine. Six? Well, no. But if you did a short memory set, you'd get a faster response than if you did a longer memory set [INAUDIBLE 00:19:51]. That is the long set. Okay. Let me just go through these rapidly. Okay. Great. And what you usually get with this sort of task is a linear slope memory set response time. [INAUDIBLE 00:20:14] is a linear function of the number of items in the memory set, and so you get two measures, one of which is the slope and the other of which is intercept, and it's

usually assumed that by looking at the slope, you're getting an idea of the rate at which an individual can do a single memory comparison, whereas the intercept reflects other processes, including the time it takes them to actually carry out a response that you can record. Now, this is a very nice task and able to separate out motor and cognitive operation rates. But the problem is that if you try giving this to Alzheimer patients [INAUDIBLE 00:20:52] a mildly impaired subject, there's a lot of trouble with this. I mean, they just can't remember three or four items, and so they make a lot of errors.

So, what we did in an earlier study was to look at a much simpler task in which instead of using a memory task, we asked the subject simply to say how many items are present [INAUDIBLE 00:21:12] — one, two, three, four items. So, all they had to do was say one, three, et cetera. As has been shown in normals on this task, the response time increases linearly with the [INAUDIBLE 00:21:23]. So, what happens when we give this to our depressed and our Alzheimer patients? What we found is, yes, they all show very nice linear increase with [INAUDIBLE 00:21:34]. The young were...showed a linear increase, so did the normal old, so did the geriatric old. But the slope was the same in all three groups, almost identical, whereas the Alzheimer patients had a significantly increased slope. For every increase in one [INAUDIBLE 00:21:52], it took them a lot longer to come up with the answer than it did for the geriatric depressed subjects, normal old subjects, or the young. Well, that was very nice, but there were a number of problems with this task in terms of data and the simplicity of it. I mean, it would've been nice to have something a little bit more complex here, but also have a little bit more variety in terms of [INAUDIBLE 00:22:17] tested.

So, what I'm going to describe today is another task that does make some demand on memory. Now, again, if you work with Alzheimer patients, you've got to be very careful of how much memory you have and what sorts of memory. So, [INAUDIBLE 00:22:33] looking at here was the time that it took an individual to access [INAUDIBLE 00:22:38] information — the sound of a word. If you present a word on a screen, people can very rapidly—even Alzheimer patients—come up with the sound of it. They know what it sounds like. They can pronounce it. That's one of the things they do fairly well. And it's known that in normals, the time it takes to [INAUDIBLE 00:22:56] a word either externally or internally to yourself varies with the number of syllables in the word. "Constitution" takes longer to come up with than does "oust". And that you can detect the rate by... What you do is you give a person a sound. For example, you could say, you know, "Does the word that you're about to see rhyme with "off"? And you can give a person a one-syllable word, a two-syllable word, a three-syllable word, and all they've had to do is say yes or no, so the response is always the same, and what you find out is that the time it takes them to make that decision varies as a function of a number of syllables in the words. Otherwise, [INAUDIBLE 00:23:36] localization, you could mentally represent the sound of the word varies from the number of syllables in the word.

So, what would happen in our geriatric and our Alzheimer patients in this situation? Would they show the same pattern? Well, yes, they do. Both normal...normal young and depressed patients showed the same sort of pattern of results. Now, the depressed patients here are depressed about that they're about to be given drugs. They're hospitalized. They're about to be given medications for their depression. They're moderately depressed. For those of you who know the Hamilton Depression Scale, they have a score of around 20, which is significant depression. But when they were doing this task, none of these individuals were medicated. Neither the depressed had been medicated, nor any of the normals — at least I hope our normals [INAUDIBLE 00:24:26]. But you see... And that's sort of something you have to worry about when you deal with geriatrics because there are so many medications that affect cognition, especially reaction time. But none of these people were on medications, yet our Alzheimer patients showed a dramatically increased slope. For every additional item or syllable, it took them about 112 milliseconds versus about 63, 64 for the normal young, the normal young, and the depressed elderly. So, again, we get the same pattern—an increased slope—for the geriatric Alzheimer patients. Though the depressed patients were slower than normal old, but all the slowing appears [INAUDIBLE 00:25:05] in the intercept. So, it doesn't matter if it's one or two or three syllables; they're equally slow with comparison to the normal old. So, if anything, it would support this notion that both geriatric depression and Alzheimer's individuals are slowed in psychological testing, but the underlying cause of that slowing is probably very different. The slowing in the Alzheimer patients results from a cognitive...or a slowing of the rate at which they carry out the task, a sort of information processing slowing, whereas in the geriatric depression, it appears to be more likely to be [INAUDIBLE 00:25:43] sort of motor retardation. [INAUDIBLE 00:25:46] a very simple task, but it takes them a long time to get the response out, perhaps because of motivational problems, attentional problems, whatever. I mean, there are a number of suggestions about this. But there are number of other people who

have just looked at geriatric depression or depression and showed the same sort of situation. They can do the Sternberg memory search and they showed the same slope as do the normal old individuals. So, this is where I'd like to sort of end up here, saying that in this sort of situation, similarity of symptoms may mask a very different underlying mechanism in these two populations.

Speaker 2 Presenter:
Thank you very much. Can we have the lights?

[Applause]

Speaker 2 Presenter:
May we have the lights, please? Will someone turn up the house lights? Thank you very much. We have time for some discussion. Actually, we can stay here until the next group of questions forces us out. I want to point out that two interesting things have emerged in the last two papers. One, when I went to school, we learned that colorblindness was a [INAUDIBLE 00:26:56] phenomenon. Alice Cronin-Golomb is now suggesting that at least one form of colorblindness becomes a cortical phenomenon, and that brings it into the area of psychology. That's kind of a revolutionary notion. That means you can now start thinking about color vision without getting into visual perception. That's very interesting, and I find that very challenging. But Dr. Nebes, I'd like to make one comment, and also to Alice. Probably the last time I'm going to have the chance to say anything to psychologists, so let me say: Alzheimer's disease has depression. It's not defined in terms of the things that you measure; it's defined in terms of the plaques and tangles which are found postmortem. So, if you're not following your patients through to postmortem examination and you can't get it straight that they have plaques and tangles, why don't you just say dementia, possibly Alzheimer's? Well, it's very clear...very important that psychologists begin to get that level of sophistication. There are many types of dementia and they all look alike, but you never know whether it's Alzheimer-defined disease in terms of pathology, not in terms of behavioral [INAUDIBLE 00:28:11], and I'd like to see us [INAUDIBLE 00:28:13].

Speaker 4 **Robert Nebess from U Pittsburgh:**
There is a set of criteria that have been set forth by the NINCDS-ADRDA criteria, which have been shown a pathologic exam if you do it in an Alzheimer's center, carried out by, usually, the neurologists and psychiatrists that test these people, being 90, 95% accurate. They say it's probable Alzheimer's if you look at the pathology, 95%.

Speaker 2 Presenter:
That's a good term.

Speaker 4 **Robert Nebess from U Pittsburgh:**
That's the common terminology. It's probable Alzheimer's, possible Alzheimer's. So, all of the ones, I'm sure, [INAUDIBLE 00:28:47] were in the probable Alzheimer category.

Speaker 2 Presenter:
Thank you.

Speaker 3 **Alice Cronin-Golomb of BU**:
[INAUDIBLE 00:28:52] I agree with you entirely. We have to have [INAUDIBLE 00:28:55]. We do have [INAUDIBLE 00:28:57] that I described, so that we can do the clinical-pathological relation [INAUDIBLE 00:29:03]. It's just a matter of getting a neuropathologist [INAUDIBLE 00:29:07]. But without—

Speaker 2 Presenter: [INAUDIBLE 00:29:07].

Speaker 3 Alice Cronin-Golomb of BU:
Most of the Alzheimer's centers [INAUDIBLE 00:29:10 - 00:29:14], specifically **[INAUDIBLE 00:29:14].**

Speaker 2 Presenter:

I know you have one. What was Shirley [INAUDIBLE 00:29:17] response to your data?

Speaker 3 Alice Cronin-Golomb of BU:

I don't know. I never talked to her. I know who she is.

Speaker 6 Unknown:

...the central efficiency of the two columns...two columns are quite peculiar, and it might be a surprising result in Alzheimer's disease [INAUDIBLE 00:29:38]. [INAUDIBLE 00:29:41 - 00:29:47] in Rochester has this [INAUDIBLE 00:29:48] study [INAUDIBLE 00:29:51].

Speaker 3 Alice Cronin-Golomb of BU:

Yeah. There's certainly...There's certainly an inference. We saw the neuro-ophthalmological and electrophysiological testing try to rule that out, and of course you can't completely rule that out. But it's the same [INAUDIBLE 00:30:02] you've got fewer, short wave length sensory neurons [INAUDIBLE 00:30:07] retina. So, either way, it could be right, but we haven't found anything [INAUDIBLE 00:30:11].

Speaker 2 Presenter:

You may find that you [INAUDIBLE 00:30:14].

Speaker 3 Alice Cronin-Golomb of BU:

Well, actually, there are recent studies... There are a few cases with central [INAUDIBLE 00:30:22 - 00:30:25] frontal cortex, where they usually lose all color, so there are some cases where they lose blue, and I've never seen a case where they've lost red and green. So, there are about seven cases like this. [INAUDIBLE 00:30:36 - 00:30:41].

Speaker 3 Alice Cronin-Golomb of BU:: In any case, it's very interesting.

Speaker 7 Unknown:

Well, my main question is it's very striking [INAUDIBLE 00:30:47 - 00:30:51] dementia, so it can happen also [INAUDIBLE 00:30:53].

Speaker 3 Alice Cronin-Golomb of BU:

Well, it's the extrastriate. It's the extrastriate, rather than [INAUDIBLE 00:30:59] temporal lobe.

Speaker 7 Unknown:

Right, but you have to [INAUDIBLE 00:30:59] presentation of Alzheimer's disease [INAUDIBLE 00:31:04 - 00:31:08].

Speaker 3 Alice Cronin-Golomb of BU:

[INAUDIBLE 00:31:08] Alzheimer's [INAUDIBLE 00:31:10] and, you know, it's a [INAUDIBLE 00:31:14] function in a much smaller area of the cortex [INAUDIBLE 00:31:16] cognitive [INAUDIBLE 00:31:19]. So, my guess is that, in some patients, [INAUDIBLE 00:31:22] color areas by chance [INAUDIBLE 00:31:24] area as opposed to some of these other ones [INAUDIBLE 00:31:29 - 00:31:33] cortex [INAUDIBLE 00:31:34].

Speaker 7 Unknown:

But then you imagined that the [INAUDIBLE 00:31:38 - 00:31:46].

Speaker 3 Alice:

[INAUDIBLE 00:31:46].

Speaker 7 Unknown:

[INAUDIBLE 00:31:48].

Speaker 2 Presenter:
Any other comments, questions? Sir. What's your name?

Speaker 8 Larry Bell:
Larry Bell.

Speaker 2 Presenter:
Hi, Larry. You can start.

Speaker 8 Larry Bell:
[INAUDIBLE 00:31:58 - 00:32:01].

Speaker 4 **Robert Nebes U Pitts**: No.

Speaker 8: [INAUDIBLE 00:32:03 - 00:32:09].

Speaker 4 Robert Nebes:
And that is [INAUDIBLE 00:32:11 - 00:32:15] that normal people also normalize [INAUDIBLE 00:32:18 - 00:32:23]. So, the [INAUDIBLE 00:32:24 - 00:32:34].