Physiology

Panel Discussion

RICHARD J. DAVIDSON, *Moderator*

QUESTION: I was actually interested in the visual cortex activation that you got for the moms viewing their kids faces. In the negative emotion results, you were talking about the amygdala input to the visual cortex and explaining it that way, but I'm wondering what could lead to that with these positive emotional stimuli?

RICHARD J. DAVIDSON (*University of Wisconisn—Madison, Madison, WI*): Certainly some of the extant data would lead you to propose the same mechanism—that is, that the amygdala is detecting information of salience, particularly critical information of survival value for the organism, and it will then activate other brain regions to alert those systems that additional important incoming information is available. We certainly know from David Amaral's and others' work that the anatomy is such that the amygdala has the connections to recruit those cortical regions; and data suggest that the amygdala is activated not just by negative stimuli, but by positive stimuli as well.

QUESTION: To follow that up with David, in the social interactions your results were focused specifically on fear, and yet we do know that the amygdala can respond to positive affect–inducing events. Would you say that it's positive emotion-eliciting stimuli that are highly salient? Would it be a surprise, which you'd subsequently interpret as positive? Do you have an explanation as to why we can see amygdala activation in response to positive stimuli?

DAVID AMARAL (*University of California–Davis, Davis, CA*): I don't have an explanation for that. There are data that highly positive—for example, erotic—stimuli might activate the amygdala. I think it's an open question at this point; I think more evaluations have to be done. It looks like for our monkeys the predominant response of the amygdala is to noxious or potentially threatening stimuli. For example, in appetitive reward object discrimination, amygdala lesions have no effect whatsoever. So if there is an appetitive component to amygdala function, I think it's a bit more distant or more complex.

Ann. N.Y. Acad. Sci. 1000: 367–374 (2003).
doi: 10.1196/annals.1280.017

QUESTION: Dr. Amaral, what's your explanation for the fact that the infant monkeys with amygdala lesions showed no fear in groups, but fear in the dyadic testing situation?

AMARAL: I don't have an explanation; I wish I did. Even in that context, while we do see all of the normal species-typical social behaviors, it's not to say that they're interacting exactly the same way as the other two groups. We do see a slight decrease in social interaction—we actually see more time spent on the mother—with the amygdala-lesioned animals. There's something clearly different about these animals from the other animals, and it does affect the quantity, if not quality, of their social interactions. Why they have less fear when they become habituated to the group, I don't know, but that seems to be the observation.

COMMENT: One thing that differs between the dyadic situation and the group situation is that in the group situation they can see other animals interacting with each other, and in the dyadic situation, they can't. That suggests that the experience of watching two individuals interact is important for getting you in the right emotional state, so that you can then interact with others; and it raises the possibility that if you then repeated the snake experiments with the amygdalectomized monkeys and you put them in with two or three other normal animals, all of whom were afraid of the snake, they would quickly learn to be afraid themselves.

ANSWER: That's an interesting experiment; we haven't tried it yet. I think it's possible. But again I'll just remind you that on a daily basis these animals are with their other social cohorts; they're performing essentially normally.

QUESTION: Dr. Levenson, do we have evidence from self-reports within the United States population that we do infer emotions from autonomic physiology? Is that speculation based on the differences that you got?

ROBERT W. LEVENSON (*University of California–Berkeley, Berkeley, CA*): I think the better evidence is from the large international surveys that show that across a range of Western cultures people associate the same set of physical changes with different emotions. That's one line of research. The second relevant line of research is a series of studies that were done in experimental psychophysiology looking at the extent to which people can accurately perceive things like changes in blood pressure. There is also the more casual version of that that Pennebaker did when he was writing his book on physical symptoms or sensations. The message of all of that is people are very interested in their visceral perceptions, and they associate them very clearly with different emotions; but they're not very accurate in making fine judgments. Those are the two things that we know; the rest is all speculation.

QUESTION: Would you expect to find similar patterns in cultures that are more likely to somaticize than not?

ANSWER: That would be a good prediction.

DAVIDSON: Bob, is anyone doing new work with pharmacological blockers to evaluate more experimentally your suggestion that the autonomic feedback is really playing a critical role in the subjective experience? It seems to me that a beautiful way to begin to dissect that is to block that autonomic change. You would certainly have a strong prediction that there should be a corresponding subjective change.

LEVENSON: I'm not aware of anybody who's doing that. It is the obvious study. People have been going the other way, looking at the extent to which the prominence of certain visceral information is associated with the stronger experience of emotion. For example, Katkin's group was looking at a pretty good measure of cardiac contractile force. You probably know that when you become aware of your heart beating, it's really the rate and pressure that you're aware of. We can measure that more directly now. This supports the idea that the stronger, the greater the contractility, everything else held equal, the stronger the report.

DAVIDSON: Although obviously, as you well know, that can all be produced by central changes.

LEVENSON Sure.

DAVIDSON: So it doesn't speak directly to the issue of whether it's the automatic feedback that's driving the subjective experience.

COMMENT: I don't think Bob's taking the position that the only source of subjective experience is feedback from the autonomic nervous system. I'm sure he would agree that there are lots of determinants of expectations, ideas of what social norms are, situations.

AMARAL: In the old Papez_circuit, the notion that Papez came up with is that you didn't get conscious appreciation of an emotion until you got up through the anterior cingulate, or anterior thalamus to the cingulate cortex; it was in the cingulate cortex that you were aware of the emotion. You were implying that the decision that an emotion was going to be generated had taken place in the amygdala, although it may not have led to conscious appreciation, and then a whole series of steps took place. Perhaps then there was some pathway where conscious appreciation of emotion was taking place. I wonder how you think about the amygdala in terms of subconscious versus conscious emotion.

DAVIDSON: I think the amygdala can respond and begin to generate autonomic activity prior to any conscious experience of emotion, and I think the conscious experience is probably something that takes place further in the processing stream. I think consciousness is kind of slow and sloppy, so there's a lot of processing that occurs prior to any subjective awareness.

LISA A. PARR (*Emory University, Atlanta, GA)*: Since Darwin's third principle deals with direct action of the nervous system and with these physiological changes and feedback that you have all have talked about, I'm just wondering if you can talk about the process of contagion from an evolutionary perspective. It seems to be a really important process whereby animals can unconsciously come to learn about the emotional states of others. Why is this so controversial?

ANSWER: It seems to me there's a lot of value in knowing what conspecifics feel, in being in tune with both their positive and negative feelings. In fact, it's hard to think of a situation in which it wouldn't be enormously helpful for you to have some sense of an emotion that a conspecific is having. Does that have to be conscious? Probably not. So you get into this interesting question of whether or not it's really knowledge about what the other person is feeling. The other person's emotions could become stimuli that could set up either learned or hard-wired responses. I think we're very far away from any critical experiments, certain in humans, that could begin to dissect that.

QUESTION: I think this would probably be a very fruitful way to begin to think about how animals might become emphatic or the evolution of empathy: if you see a stimulus that may produce a hard-wired response and then you become aware of those feelings yourself, the next time you're in that situation you may be more likely to feel that way. Then these stimuli come to have meanings and representations without your actually having to go through the situation or knowing exactly what the consequences are that led up to the event. It seems like a really interesting process. But when you talked about contagion, you said it was somewhat controversial or a little bit dubious that this process actually takes place. Is an autonomic response really necessary for the subjective feeling of emotion?

ANSWER: I think in humans there are other paths to emotional or empathic enlightenment. By one path you can feel what the other person is feeling. I think that's reflected in a kind of autonomic coactivation. By another path we're quite capable of figuring out what another person is feeling; we can be quite analytic. We have a finding from a recently completed dissertation that suggests that in very long–term married couples, husbands over time begin to use a much more cognitive strategy to figure out what their wives are feeling; they don't actually show evidence that they're feeling what their wives are feeling. Their wives continue to have the coactivation pattern.

DAVIDSON: Lots of work has been done on imitation learning of an emotional sort. Learning via observational learning of emotional responses, the situation that Frans mentioned earlier with the snake fear, is a good example of that. There are lots of other examples, both in humans and in an animals of that kind of observational learning. I think one suggestion implied in Bob's work is that some of that learning may be mediated through autonomic reci-

procity or coherence, which I think would be very interesting to explore. If you somehow disrupted the autonomic response, you may actually interfere with that kind of emotional, observational learning.

QUESTION: Bob, because of time you really gave very short shrift to your work on empathy, which I think is really fascinating and has so much relevance to our understanding of how people understand each other. I was interested in whether or not you have tested your nonempathic people separately, to verify that they don't do as well as other people on reading either the face or the voice. Also, have you seen whether or not the absence of empathy might possibly be interaction or situation specific—that is, if you present to them a series of people, both males and females in the couples, do they do badly on all of them? In other words, is this really a trait that you were seeing? Last, can we use this as a kind of predictor of who will be good therapists, good interviewers?

LEVENSON: The only thing we really know is that it is trait-like in the sense that it seems to carry over. I think the most we've assessed is four different targets in one person, and it is consistent in that way. We don't know what the consequences of this kind of empathy are.

QUESTION: You were noting that when you assume a certain posture with your face, the emotion tends to follow. I'd like to bring up the converse of that. How about patients with pathological laughter or crying? I have a patient who laughts every time I take his blood pressure. He doesn't just giggle; he laughs a full-throated, full-blown laugh, which makes everyone in the room laugh as well. I ask him, "How are you feeling right now?" He says, "Embarrassed." There's nothing in his autonomic system or physiology that suggests the embarrassed state. He laughs like crazy. How do you describe the mismatch between the physiology going one way and the internal subjective experience going exactly the opposite way?

ANSWER: I suppose the most provocative possibility is that an emotion is designed with redundancies across these different systems—the physiology, the expression, and what not; and when everything is working according to plan and you're seeing an emotion uninhibited, unaffected by the demands of a situation, uninfluenced by pathology, there's a lot of coherence and agreement across these systems. Discoherences reflect an intentional interruption in the flow of emotion, as when you try to cover up what you're feeling. Maybe the essence of emotional pathology is that these disorders—whether they are something as dramatic as pseudobulbar palsy, where you see explosions like you're talking about and they seem to be removed from any contingent stimulating event; or findings like in the schizophrenia literature, where you see patients with blunted facial affect but high levels of internal autonomic response and subjective emotion—are signs that the system has broken down for some reason. What you've described in a patient is a great example of that.

QUESTION: Dr. Levenson, it makes sense that in a more collectivist culture, people would deemphasize their own facial expressions when gauging their emotions. I was wondering if you got cross-cultural differences for disgust. If you did, how you would explain it?

LEVENSON: Yes, in this particular instance where Paul and I did this work together, we had two ways of studying disgust. One was with the facial activation paradigm, and the other was by showing people surgical films. Interestingly enough, we didn't see this dissociation between physiology and self-report for the films. Looking at a surgical procedure produced the same level of report of disgust in this culture as it did in our comparison Western control group. I'm not sure exactly how this is going to play out with your underlying question, but I do think it suggests that the self-reported emotion may be not only culture bound; it may be that within a culture there are situation-related variations. For example, if you see someone in pain undergoing a kind of a gross surgical procedure, whether you say you feel sadness or disgust may be just as cultural bound as whether or not, when your face is looking disgusted but there's no one else in the context, you say that it's disgust. I think there's a real opportunity here for quite situation-specific feeling rules within cultures. This is not a new idea: Hochschild wrote about it a number of years ago, and I think it's pretty well accepted in anthropology and sociology.

QUESTION: As a psychiatrist I was told that two things are actually going on: sympathy, when you have an autonomic response and feel exactly like your patient; and empathy, where you put your yourself in your patient's shoes. Can you please comment on this distinction?

ANSWER: I'm going to take your two and raise you one: I think there are three processes at work here. The first is now called *empathic accuracy*; simply speaking, it's knowing what someone else is feeling. I think it's really useful for therapists to know what their patients are feeling. The second is what we've been talking about: feeling what someone else is feeling. It's debatable whether it's good for nurses, therapists, or even actors to feel what the character is feeling; it depends on what you think. The third thing is being helpful: you know someone's in pain and you're acting in a proactive, helpful way. I think that's probably also pretty useful. Therapists do it, nurses do it. I think the real controversy concerns living somebody else's emotions—whether that does you or the other person any good.

QUESTION: Back in 1993, there was a paper published in the *American Scientist* devoted to defects in the pleasure center using PET scan studies and relating it to things like attention deficit disorder, alcoholic addiction, gambling addiction, and probably drug addiction. One of the questions I have is why they used PET scans when functional MRI's and MRI's were also available and probably in some cases better. Further, with autistic children, the latest

controversy is multiple vaccination: how would that affect the amygdala in producing autism?

ANSWER: We're anticipating using PET, we haven't done it yet. We're going to use microPET. The reason we're doing that as opposed to functional imaging is, first, doing functional imaging with a monkey is extremely difficult. There are a couple of places in the world that are trying to do it. It takes a specialized fMRI that has a vertical board, because the animals don't like to lie on their backs. What we're really looking for is the integrated brain activity that takes place while the animal is engaging in a social interaction, so obviously the animal has to be behaving while you're imaging their brain. To do that, you can use PET because you can inject the glucose tracer into the animal before it actually engages in the behavior. Those brain areas that are activated take more of this tracer. You can image that 20 or 30 minutes later, after the animal has been finished the behavior. It really is the only technique for evaluating the functional activation of the monkeys brain when you have them running around doing something. The beauty of microPET is that the resolution is about 1–2 mm. We'll be doing those studies next year.

I'm intrigued by all of the potential causes for autism, and parents are very concerned about the possibility that combined vaccines might be causing autism. My institute has some studies ongoing to see whether there is a link between vaccinations and autism. Interestingly, there was a paper published just last week in the *New England Journal of Medicine* from a longitudinal study done in Denmark, where, as I understand, the medical records of the whole population are much better. There's a large database of all the citizenry. The bottom line is that they looked at kids over the last 20 years who have and have not received the measles, mumps, and rubella combined vaccine. They found that if you received the vaccine, the risk for autism actually was slightly lower than if you didn't. So essentially they found no relationship between the vaccine and autism. Now, of course, there are other vaccines; other research groups are examining these. We don't have a leading hypothesis at the moment. While it's a genetic disorder in part, there's concern that it's a matter of both genetics and some other factor, such as environmental contamination. The number of kids with autism is, I think it's clear, going up. The strategy at my institute is still to cast a pretty broad net on trying to find causes. From this study in Denmark and a couple of others, it's looking like the MMR vaccine cannot be the strongest candidate. It's still conceivable that a subset of kids with autism that has so-called regressive autism might actually have some link to MMR, but I think the evidence at the moment is against a strong link between MMR and autism.

QUESTION: I'm interested in the contagion of emotion between mothers and infants. I don't think anyone has come up with emotional mirror neurons yet, but I was wondering if you, for instance, trained a mother to be afraid of

a sound and then played the sound to her infant, would the infant's brain have a contagion of activation along with the mother? Has that been studied?

ANSWER: There has been precious little work that I'm aware of where both the mother and the infant had been recorded simultaneously in interaction. There are pragmatic constraints against doing that, although it's certainly not impossible. But behavioral data is available that suggests that the kind of emotional contagion you're suggesting can occur. I think it's the principal vehicle through which affective skills can be conveyed during sensitive periods of development; and we know from research at the animal level that environmental input during these sensitive periods can set up brain systems in ways that are quite enduring and, in certain cases, persist for life. At the animal level, there is evidence to suggest that these kinds of environmental inputs can actually change gene expression in systems that we've talked about. So I think that it's a wide open area; there's just very little research at the human level that has specifically addressed this issue with regard to neural systems.