Response to Commentaries on "Multiple Arousal Theory and Daily-Life Electrodermal Activity Asymmetry"

Rosalind W. Picard, Szymon Fedor, and, Yadid Ayzenberg,

MIT Media Laboratory

Cambridge, MA 02139

picard@media.mit.edu, szymon.fedor@gmail.com, yadid@mit.edu

corresponding author: Rosalind Picard, 617.253.0611

Response to commentaries on "Multiple Arousal Theory and Daily-Life Electrodermal Activity Asymmetry"

Abstract

We respond to the commentaries of Critchley & Nagai, Mendes, Norman, Sabatinelli, and Richter. We agree that a theory needs to make predictions and we elaborate on the predictions we made so far. We do not agree that arousal has to have a precise definition in order to present theory about it; however, we do provide concrete answers to questions raised about Multiple Arousal Theory.

Keywords: Emotional arousal, sympathetic nervous system (SNS), electrodermal activity (EDA)

Arousal is like an elephant in the famous cartoon where blind scientists are touching it, one feeling the tail and saying, "It's a snake" and one feeling the leg, "It's a tree trunk." With arousal in emotion studies we hear, "It's a brain state", "It's a self-reported feeling", "It's a state of autonomic activation," and more. Arousal is harder to characterize than an elephant because it does not have clearly defined physical boundaries: In emotion theory, as a "dimension," arousal is more eigenvector than animal. While arousal has been operationalized as physiological changes, self-report items, brain activity measures, and more, none of these fully defines it. As Mendes points out, it has a long history of poor definition. Fortunately, a lack of precision in definition does not mean we cannot make systematic explanations (or "theory") rooted in factual observations. Consider, as example, that one can state facts, make predictions, and provide explanations about Mount Everest, and summit it, without defining which rocks at its base are and are not a part of it.

Theories should enable predictions, and Multiple Arousal Theory does lead to new predictions. Here is one that we wrote in our article: "With our theory we can make predictions such as, all other factors constant, larger right amygdala activation would contribute to larger right EDA in a right-hander experiencing significant anxiety or depression." We further predict that studies that measure only left EDA might miss this effect. In short, if an emotional state activates the right amygdala more than the left, and if there are no other strong sources contributing to left electrodermal arousal (e.g., no task demands for left motor activity), then in a right-hander[1] we would expect to measure greater EDA on the right than on the left palm.

[1] An estimated 80% of right-handers are left-brained, so the prediction should be qualified for people who have been strongly right-handed since birth, and exclude those who were forced to use their right hand.

It was the Multiple Arousal Theory that led us to predict that EDA asymmetry should be present in short-term lab data (we had originally taken the asymmetry seriously only when we saw it consistently in long-term measurement). We predicted more cases of right-dominant EDA in the challenging counting-backwards task since participants were viewed by a critical judge pushing an obnoxious buzzer when they erred. This situation could trigger social threat, which we'd expect to increase right amygdala activation more than left (in a right-hander). Thus, we would expect many participants to have their right EDA go higher than their left. Indeed, the data matched the theory.

Our theory also predicts that it won't always hold for every participant: Some people care what the experimenter thinks of their performance while others may not care at all. Some may also have equally high left activation from other influences on parts of the brain giving rise to left EDA. Emotional states and task demands contribute to activating the multiple arousals.

Norman properly reminds that if we associate arousal with only SNS activation, there are still many endpoints to measure – cardiac, vascular, pupil, EDA. While our theory does not restrict to SNS arousal, our article focused on EDA because it is purely innervated by the SNS (Boucsein, 2011). Also, EDA is widely known and conveniently and comfortably measured 24/7 with a wearable sensor. That said, a measurable change in EDA is not necessary for arousal (see below).

Critchley and Nagai's commentary is fascinating, telling of how asymmetric sympathetic arousal from the right side of the brain is associated with cardiac arrhythmia. Our theory can also make predictions in this case: if the sympathetic activation is strongly right-sided to the heart, then all other sources held constant, we would expect to see EDA also be higher on the right wrist than

on the left. We may also predict that emotional states that activate the right (or triggering) side are potentially more dangerous in an at-risk patient than states that do not activate this side. As Mendes points out, it may be valuable to examine the connections between our findings and those of others who have differentiated benign from malignant arousal.

Sabatinelli provides some careful methodological recommendations to strengthen the Multiple Arousal Theory. We agree with his suggestions, and in fact one has been followed since this paper was accepted: Akane Sano at MIT measured synchronized EDA on the left and right fingers (with traditional placements and gel) and on forearms (with dry electrodes) running the classic counting-backwards study with a new group of participants. As predicted, the EDA on the right fingers was usually more responsive than on the left fingers (and on the right forearms more than on the left). While this replicated our earlier findings, to our surprise there were also some individuals for whom the finger asymmetry and the forearm asymmetry were not the same; some of these differences may be accounted for in the time it takes for the forearm/dry electrodes to become as responsive as the finger/gelled electrodes, while other differences may be more complex (e.g. there are many dermatomes that run through the wrist and palm.) Sano's study will be reported in more detail soon.

Richter asks, "If two situations lead to differences in EDA at two body sites does this indicate the activity of two different arousal systems?" Our answer is "This is not sufficient." There are many situations that can elevate EDA (what if one wrist is held over hot steam?) or that can reduce it (what if one side has neuropathy?) and alternative sources must be considered before

evidence is credited to multiple arousals. We have particularly emphasized certain regions of the brain that a protocol might try to elicit, e.g. amygdala activation with its ipsilateral influence or motor cortex with its contralateral, but one must also consider environmental effects, differences in sweat gland distributions, mediating factors for the sudomotor system, and more.

One of Richter's challenges is "Do these differences [in EDA measures] need to be accompanied by differences in subjective experience […] ?" We do not think subjective reported experience should be necessary. A challenge when testing predictions that involve asking people what they feel, is that many people are poor at interpreting or reporting what they feel: Many are alexithymic. In one of our pilot studies on the perception of arousal and its relationship to EDA, we gave ten healthy MIT graduate students (average age 30.8, SD=4.2) a twenty-item Toronto Alexithymia Scale (TAS-20) (Bagby, Parker, & Taylor, 1994). Six scored with values of 14 or lower, while four scored in the high range 19-24 on the "difficulty identifying emotions" subscale. We predicted that the four highest-scoring participants would have the lowest correlations between their self-reported arousal and overall EDA activation. Indeed, two of them had the only negative correlations measured between self-reported arousal and the wrist-based EDA measure of all the ten. However, the other two who scored worst on identifying emotions had the two highest positive correlations, which initially puzzled us. When we asked them how hard it had been to label their daily arousal (without getting to look at their EDA), they said it was very easy because their arousal states were extreme and obvious. When we went back and checked their EDA data, it matched what they said: Their skin conductance (area under the curve) had both the highest overall average

of the ten participants, and the highest day-to-day variance. Their self-reported arousal agreed with their very high or very low EDA measures (taken on the right wrist). While that was just a ten-person pilot study so we do not want to overgeneralize, it does fit common sense: we can expect some people's self-reported arousal to match their physiology, even if they are alexithymic, if their physiology has huge swings; however, others who are alexithymic or nearly so, with normal varying physiology, can be expected to give uncorrelated or negatively correlated self-reports.

Richter asked, "What if a researcher observes differences in brain activity and subjective experience but no difference in EDA – does this provide sufficient evidence for multiple arousal systems?" The first thing to note is that there are people who have essentially no measurable EDA (sometimes from medications or from conditions we do not understand) and thus we are careful to not require electrodermal response as a necessary component of arousal. These people may still have other aspects of an arousal experience (e.g. heart rate accelerations) and report feelings such as calm or excitement. If they have differential experiences of arousal that map consistently to differential brain activation patterns then, yes, we would be inclined to credit those as evidence for multiple sources of arousal.

Some open questions are indeed up to definition, but we underscore that slight variations on the definition do not undermine our findings or proposed theory. For example, Richter asks, "Does any kind of stimulation that activates a specific brain region and that leads to a change in EDA provide evidence for a change in arousal?" We could decide that distinct mappings should be called

distinct kinds of arousal – or not. We prefer to wait and get a lot more data first to figure out which distinctions are most meaningful for diagnostics and for better understanding of human experience.

Despite these and many other new questions, one thing that we do know now is that operationalizing arousal with the traditional EDA one-sided measurement, and assuming it will increase with high arousal and decrease with low arousal, is not the whole story. The scientists figuring out arousal still are largely blinded; however, allowing for multiple sources of arousal and measuring their distinct output patterns will help us figure out this elephant.

References

Bagby, R. M., Parker, J. D. A., & Taylor, G. J. (1994). The twenty-item Toronto Alexithymia scale—I. Item selection and cross-validation of the factor structure. *Journal of Psychosomatic Research*, *38*(1), 23–32.

Boucsein, W. (2011). *Electrodermal activity*. *Techniques in psychophysiology* (2nd Editio.). Springer-Verlag New York.