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Mendes, Wendy Berry

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traffic during vestibular stimulation (El Sayed, Dawood, Hammam, & Macefield, 2012). The potential yoking of sympathetic “subaxes” predicts that stress-related autonomic asymmetry could be apparent across other organs, for example, as a “functional” Horner’s syndrome (grief-related anisocoria is indeed reported; Inman, 1922); Picard and colleagues liken electrodermal asymmetry to a crooked smile; this simile may be insightful.

The premise of autonomic “spill-over” implies a balancing process within descending autonomic brain pathways, yet Picard and colleagues note times when, for the same individual, sympathetic arousal is expressed symmetrically on one occasion and asymmetrically on another. Clearly, the underlying mechanisms need detailed characterization. The article of Picard et al. (2015) highlights a need to fine-tune our mechanistic understanding of psychophysiological arousal, not least from perspectives of efferent–afferent interaction, pathways of efferent sympathetic outflow, and neurochemical control. Such knowledge will help manage anxieties that might arise with an increasing use of self-monitoring devices. Moreover, electrodermal biofeedback training might assist in mitigating pathological asymmetrical autonomic stress responses.

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Comment: Looking for Affective Meaning in “Multiple Arousal” Theory: A Comment to Picard, Fedor, and Ayzenberg

Wendy Berry Mendes

Department of Psychiatry, University of California, San Francisco, USA

Picard et al. (2016) provide case studies, laboratory data, and a description of a theory they term “multiple arousal theory” detailing asymmetric electrodermal responses co-occurring with intense, and, possibly, threatening experiences. There is potential value in two key messages in Picard and colleagues’ article: (a) the extended use of dry electrodermal activity (EDA) sensors for long-term wear that could be integrated with experience sampling to examine dynamic changes in physiological responses along with affective states, and (b) the novel observation that asymmetric EDA might be a concomitant with

meaningful affective states. There is little question that the former finding would be of great value to emotion researchers, as long as the latter observation proves valid and reliable. I focus here on the EDA asymmetry as a possible marker of a meaningful affective state and the subsequent steps that include looking forward—replicating the effects, specifying context, boundary conditions that might modify interpretations including age, disease states, and cultural differences, and identifying the full range of affective states that might co-occur with asymmetric EDA responses—and looking backwards—situating these

findings in the psychophysiological and affective literatures to contextualize intersecting areas possibly related to asymmetric EDA responses. My commentary provides some thoughts on understanding this work in the context of affective psychophysiology starting with contextualizing these findings in the broader literature.

Picard and colleagues describe an accidental discovery of asymmetric EDA that initially shows within person reliability and situation-specific contexts. The search for physiological responses that co-occur with specific mental states, especially affective ones, is a recurring theme in psychological science with varying degrees of success and disagreements regarding whether the search is futile or useful and successful or not (see Lang, 2014; Levenson, 2014; Norman, Berntson, & Cacioppo, 2014, for recent discussions). Whether there exists specific physiological responses that share a one-to-one correspondence with an affective state, or more general physiological changes that are related to core affective features, which are highly contextualized, may still be a matter of debate, but Picard and colleagues offer a novel observation regarding lateralization of EDA, which, at face value, is an intriguing physiological pattern.

Multiple arousal theory is consistent with much previous theory and research that a single dimension of *arousal* is problematic leading to the observation that arousal is a “fuzzy” construct (e.g., Blascovich, 1992). The fuzziness is due, in part, to its ubiquity in core processes in emotion, motivation, and stress coupled with the lack of precision regarding the neurobiological underpinnings; thus, arousal means many things, yet nothing specific. To highlight an enduring example from the motivational literature consider the Yerkes–Dodson inverted U of “arousal and performance.” As most introductory psychology textbooks describe, Yerkes–Dodson’s theory argues there exists an optimal “arousal” state such that too little or too much arousal will be associated with poorer performance. Several problems stem from this perspective, the most notable, how is arousal defined? It is implied that arousal is some physiological parameter, like heart rate increases, which is problematic because of the dual influences of sympathetic and parasympathetic nervous systems which can independently and additively influence heart rate changes (Berntson, Cacioppo, Quigley, & Fabro, 1994). What if arousal is more precisely defined as sympathetic nervous system (SNS) activation like pre-ejection period (time from ventricle contraction to aortic valve opening) or EDA (as measured in the target article) or catecholamines (e.g., epinephrine), but even these measures, all indexing SNS, are far from perfect proxies for each other. What if arousal is defined as HPA activation, which compared to SNS has a more consistent inverted U relation with cognitive processes like memory. In any event, arousal has a long history of poor definition and, hence, inconsistently relates to “performance” as Yerkes–Dodson implies (see Dienstbier, 1989; Mendes & Jamieson, 2013).

Picard and colleagues (2016) argue that their data show “two sources of arousal” that they suggest might be related to some basic valence mapping—the “best investor meeting had the most

left over right” and the “worst ... had the most right over left”—though they responsibly note that early data cannot “prove a simple valence mapping.” The idea that arousal states might be further differentiated into positive/negative, approach/avoid, challenge/threat is not a new idea and several frameworks are consistent with this idea. Early stress researcher Hans Selye noted that both *eustress* and *distress* were characterized by acute changes in underlying stress systems with *eustress* relating to the perceived utility or desire of the event (1974). Most explicitly, Dienstbier (1989) reviewed animal and human research to make the argument that “arousal” states could be differentiated into benign, adaptive “physiologically tough” patterns dominated by SNS responses, whereas a more malignant, maladaptive pattern of arousal consisted of SNS and HPA activation and differentiated arousal related to myriad consequences including physical health and psychopathology. Other theories including physiological threat versus thriving identified distinct catabolic versus anabolic neuroendocrine profiles (Epel, McEwen, & Ickovics, 1998), asymmetric frontal cortical activation indexing general approach compared to avoidance motivation (Harmon-Jones & Allen, 1998), and challenge and threat theory, which argued that arousal states could be further differentiated with cardiovascular reactivity (Blascovich & Mendes, 2010) followed this basic distinction. Though each theory differed in its mental state labeling as well as physiological responses all share the general approach of differentiating “good” from “bad” arousal. Taken together these theories help situate the Picard et al. (2016) findings and suggest other physiological parameters that have differentiated benign from malignant arousal such as cardiovascular indicators, adrenal or anabolic hormones, and shifts in frontal cortical symmetry.

Moving to the question of how we might optimize on Picard and colleagues’ (2016) findings to understand and extend how affective states related to underlying physiological responses, we can ask several questions. For example, how does asymmetric EDA relate to other physiological systems, particularly ones previously identified to differentiate “positive” compared to “negative” arousal? Following from the autonomic space model, is right-dominated EDA more closely correlated with SNS responding? Or SNS and peripheral nervous system (PNS) coactivation? Is there a link between asymmetric EDA and frontal-cortical asymmetry? Is EDA asymmetry related to discrete emotions, like fear, core affect, or motivational states, like threat? Are these effects consistent over the life-span? Or does peripheral neuropathy in older ages interfere or potentiate asymmetric EDA? Should we consider EDA asymmetry closer to an individual difference or a situationally evoked response that occurs in most people? These questions are a small sampling of the questions that might guide future work on asymmetric EDA that would be of tremendous interest to affective scientists.

Picard et al. (2016) outline a novel response profile of asymmetric EDA responses that might be linked to negative affective states. Existing psychophysiological theory helps situate this work in the larger literature and points to possible overlapping physiological systems that might co-occur with right-dominated EDA responses. Picard and colleagues’ work also shows the

tremendous value in discovery by accident and the reciprocal process of deductive and inductive reasoning in emotion and psychophysiological theory. I have identified just a small sample of questions that one could pursue with “multiple arousal” theory and measurement of bilateral EDA responding. Pursuing these and other questions might shed light on underlying physiological processes of emotion.

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Comment: Emotional and Autonomic Arousal Constructs in Psychophysiological Research: Where Do We Go From Here?

Greg J. Norman

Department of Psychology, University of Chicago, USA

Abstract

Picard, Fedor, and Ayzenberg (2016) provide a review of the existing literature on the relationship between electrodermal activity (EDA) and affective processes and present data from a number of studies suggesting strong lateralization in EDA reactivity to emotion. As the authors note, their manuscript extends previous work suggesting the concept of arousal is more complex than previously thought, and they provide a framework for interpreting such complexities within the context of a multiple arousal theory.

Keywords

Arousal, autonomic, electrodermal, emotion, psychophysiology

Comment

Picard et al. (2016) review an extensive literature on the application of electrodermal activity (EDA) in psychophysiology and the theoretical foundations for its utilization in emotion research as a measure of arousal. Additionally, the authors

report results from a number of empirical studies that suggest clear left versus right asymmetries in EDA responses and their correlation with affective states across multiple recording sites. Such findings are of particular interest in that they may provide novel insights into the neurobiological organization underlying the relationship between emotion and autonomic processes. Moreover, the results from their studies have potentially important methodological implications for the use of EDA as a measure of physiological or emotional arousal as the majority of previous studies have recorded EDA from the non-dominant hand only and assumed relatively consistent bilateral responses.

In addition to providing evidence for emotion related lateralization in EDA responses, the authors’ discussion of the more theoretical aspects of the arousal construct and its relationship to neurobiological and psychophysiological mechanisms extends previous work suggesting that viewing arousal as a unidimensional construct, capable of being reliably quantified by a single physiological measure (e.g., EDA), is no longer tenable.

Corresponding author: Greg J. Norman, Department of Psychology, University of Chicago, 5848 South University Avenue, Chicago, IL 60637, USA. Email: gnorman@uchicago.edu