

[Commentary] What does left-right autonomic asymmetry signify?

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Running Head: Autonomic asymmetry

What does left-right autonomic asymmetry signify?

Author Names

Hugo D Critchley^{1,2,3}

Yoko Nagai¹

Affiliation

¹Brighton and Sussex Medical School, University of Sussex, UK

²Sackler Centre for Consciousness Science, University of Sussex, UK

³Sussex Partnership NHS Foundation Trust, UK

Correspondence to:

Author Name	Hugo Critchley
Affiliation	Brighton and Sussex Medical School, , UK
Address	University of Sussex
Address	Brighton BN1 9RR
Address	East Sussex
Phone:	+44 1273 678336
Fax:	+44 1273 877548
Email:	h.critchley@bsms.ac.uk

Abstract

The situation-dependent lateralization of sympathetic electrodermal arousal during real-life stress (Picard, Fedor, & Ayzenberg, 2014) may challenge a unitary notion of arousal, and call into question on the practice of unilateral electrodermal recording, but there are broader implications. Here we consider a potential relationship between stress-induced lateralized shifts in electrodermal activity, and a theory concerning lateralized emotion-induced cardiac arrhythmia.

Keywords: arousal, brain, cardiovascular, electrodermal, emotion, stress

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Hugo D Critchley^{1,2,3}

Yoko Nagai¹

Affiliation

¹Brighton and Sussex Medical School, University of Sussex, UK

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Correspondence to:

Author Name	Hugo Critchley
Affiliation	Brighton and Sussex Medical School, , UK
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Text

Arousal is a useful term describing states of attentive wakefulness and action-readiness, linked to emotion and measurable in physiological reactivity.

Electrodermal activity is an accessible, sensitive correlate of centrally-driven sympathetic nervous activity on sweat glands, and is well-suited as an index of psychophysiological arousal (Boucsein, 2012): Unlike pupil size or heart rate, electrodermal activity does not incorporate parasympathetic drive; unlike blood pressure, there is no reflexive coupling with viscerosensory feedback; moreover, unlike most sympathetic effector synapses, sweat gland innervation is cholinergic and independent of circulating adrenaline and noradrenaline. Electrodermal recording has helped refine emotion theory and characterize perceptual, cognitive and affective salience. Moreover, electrodermal biofeedback has

therapeutic application to attenuate stress-related arousal, or to enhance the control of cerebrocortical excitability (Nagai, Goldstein, Fenwick, & Trimble, 2004). In the human brain, interacting neural centers along the neuraxis support cognitive and emotional arousal, and drive sympathetic response (Critchley, Nagai, Gray, & Mathias, 2011). In the periphery and spinal cord, pathways are lateralized, and can be tracked up ipsilaterally into hypothalamus. Nevertheless pathway crossing, and left-right interaction, can occur at multiple levels (Vetrugno, Liguori, Cortelli, & Montagna, 2003). The findings of Picard and colleagues (2014) fit with a literature associating sympathetic activity with lateralized brain activity. Stimulation, neuroimaging and lesion studies suggest sympathetic drive to the heart has a right hemisphere dominance (e.g. Oppenheimer, Kedem, & Martin 1996) and there are appealing links to purported right cerebral hemisphere dominance for negative emotional processes (e.g. Ahern & Schwartz 1985—Picard and colleagues highlight amygdala data).

Importantly, asymmetrical sympathetic discharge may have clinical significance as a mechanism for emotionally-triggered cardiac death (Lane and Schwartz 1987; Taggart, 2013). Sympathetic nerves regulate the rate of myocardial electrical repolarization for the next cardiac contraction. If regions of the heart are prematurely ready to contract, the heart becomes predisposed to potentially fatal ventricular arrhythmia. In fact, left and right sympathetic nerves differentially innervate the front and back of the heart (Yanowitz, Preston, & Abildskov, 1996). Ventricular arrhythmia may thus be triggered by left-right sympathetic imbalance,

originating from a central 'spillover' of lateralized sympathetic activity during stress (also an account for sudden unexpected death in epilepsy).

Experiments circumstantially support this brain heart-laterality hypothesis. Pro-arrhythmic cardiac changes can be indexed non-invasively, using external electrocardiography, and correlate with lateralized midbrain activity (Critchley et al., 2005). Observed asymmetry in sympathetic electrodermal responses during stress might also therefore indicate cardiac sympathetic asymmetry. Sympathetic outflow to the skin may not necessarily reflect outflow to the heart and vasculature (Donadio, Karlsson, Elam, & Wallin, 2002), but a correspondence is observed in asymmetric muscle and skin sympathetic nerve traffic during vestibular stimulation (El Sayed, Dawood, Hammam, & Macefield, 2012). The potential yoking of sympathetic 'sub-axes' predicts that stress-related autonomic asymmetry could be apparent across other organs, e.g. 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 ch. The paper of Picard and colleagues (2014)

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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