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Emotion's Response Patterns: The Brain and the Autonomic Nervous System

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Abstract

The article considers patterns of reactivity in organ systems mediated by the autonomic nervous system as they relate to central neural circuits activated by affectively arousing cues. The relationship of these data to the concept of discrete emotion and their relevance for the autonomic feedback hypothesis are discussed. Research both with animal and human participants is considered and implications drawn for new directions in emotion science. It is suggested that the proposed brain-based view has a greater potential for scientific advance than the traditional model that emphasizes specific states of mind as mediators or reflectors of visceral action.

Keywords

autonomic nervous system, brain circuits, emotion, motivation

Question 1a: How specific versus general are emotional patterns seen in the autonomic nervous system? How might patterns best be conceptualized?

There is a considerable experimental literature supporting the view that both animals and humans show specific, autonomic reflex patterns in reaction to cues humans describe as putatively "emotional." The conceptual foundation for this work posits motivational circuits evolved in the mammalian brain that promoted the survival of individuals and their progeny. In animals, these circuits mediate reflex responses that are on the one hand, defensive, coping reactions to external hazards that imperil life, and on the other, appetitive, reactions to obtain rewards/needs that are life sustaining. The behavioral output consequent on circuit activation is highly varied in complex organisms. However, two features characterize the primary reflex reactions to both threatening and appetitive stimuli: (a) increased sensory processing, and (b) mobilization of the organism for action. In this brain-based model, the overlapping defense and appetitive circuitry are held to be the motivational foundation for the varied reports of emotional states. That is, these primitive brain circuits are activated by cues that humans are likely to describe as evoking an unpleasant/aversive or pleasant/desirable state.

Extensive research with animals (e.g., Davis, 2000; Fanselow & Poulos, 2005; Kapp, Supple, & Whalen, 1994; LeDoux, 2003, 2012) has defined a set of neural structures that makeup the brain's survival network (for an overview, and its relationship to research with human participants, see Davis & Lang, 2003; Lang & Davis, 2006). The bilateral amygdala—two small, almond-shaped bundles of nuclei in the temporal lobe—plays a central role: Motivationally relevant cues projected from cortex and thalamus (sensory) or hippocampus (memory) activate the amygdala's central and lateral nuclei and its extension in the bed nucleus of the stria terminalis. Subsequent projections from these amygdaloid structures engage a variety of other brain regions that prompt orienting, sustain vigilance, enhance perceptual (information) processing, and activate efferent-projecting structures that mediate defensive or appetitive reflex actions. Of particular importance for the associated autonomic responses are the nucleus ambiguous and the dorsal motor nucleus of the vagus (slowing the pulse) and structures of the hypothalamus, notably the lateral hypothalamus (prompting tachycardia, skin conductance increase, pupil dilation, and various vascular changes) and the paraventricular nucleus (corticosteroid release).

Motivationally significant cues engage behavior patterns that are remarkably similar across a wide range of species (e.g.,

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Campbell, Wood, & McBride, 1997; Timberlake, 1993). Thus, for example, when a prey animal espies a predator at a distance its first reaction is to "freeze," immobile in a vigilant posture. This somatic reflex is mediated by the previously described survival circuit (via projections from the amygdala to the ventral periacqueductal (central) gray; Fanselow, 1991). The circuit also engages the autonomic nervous system (ANS), prompting a widening of the pupil, multiple exocrine and endocrine glandular responses, and robust heart rate deceleration, frequently referred to as "fear bradycardia."[1]

The covariation of amygdala activation and threat-evoked bradycardia has been clearly shown in research by Kapp and colleagues (e.g., Kapp, Gallagher, Underwood, McNall, & Whitehorn, 1982; Pascoe & Kapp, 1985). Studying rabbits, electrodes were implanted in cells of the central nucleus of the amygdala, such that neural unit firing could be recorded. Two different tone stimuli (CS+ and CS−) were presented in a classical conditioning paradigm with aversive electric shock serving as the unconditioned stimulus. When confronted by the threatening cue (CS+), a close relationship was obtained between cell spikes at the amygdala site and the extent of cardiac deceleration, increasing over exposure trials. In contrast, these same cells were essentially unresponsive to the control stimulus (CS−).

In studies of emotional picture processing in humans, we have observed a similar cardiac deceleration, significantly greater than for neutral or pleasant cues, when participants view arousing unpleasant images—particularly strong if the picture represents a threat towards the viewer, as an aimed gun (e.g., Bradley, Codispoti, Cuthbert, & Lang, 2001). These same unpleasant pictures are also associated with increased amygdala activation and a subsequent increased activation in visual cortex (e.g., Sabatinelli, Bradley, Fitzsimmons, & Lang, 2005; Sabatinelli, Lang, Bradely, Costa, & Keil, 2009). In effect, the parasympathetic, vagal-mediated deceleration and the often-accompanying somatic "freezing" are components in a whole-body attentional and information gathering process, consistent with the posture observed in nonhuman animals facing a distant threat. Interestingly, a strong skin conductance response (e.g., Bradley et al., 2001; Lang, Greenwald, Bradley, & Hamm, 1993) and widening of the pupil (e.g., Bradley, Miccoli, Escrig, & Lang, 2008) are also prompted by these same arousing stimuli, indicating coactivation of the sympathetic branch of the ANS in this primitive stance of attention and action preparation.

ANS branch coactivation was also observed in a recent fMRI study by Hermans, Henckens, Roelofs, and Fernández (2013). These researchers assessed brain defense circuit activation and concurrently measured heart rate and pupil dilation while human participants viewed pictures with either neutral or unpleasant/aversive content. A parallel strong activation of both amygdala and the periaqueductal gray was observed uniquely during aversive images, as in animal conditioning studies of the "freezing" defense response. Furthermore, these same unpleasant scenes prompted greater pupil dilation (sympathetically mediated) and marked bradycardia (mediated by the parasympathetic branch of the ANS). Interestingly, activation of the central gray was significantly correlated trial-by-trial with heart rate deceleration, consistent with the hypothesis that motor inhibition and threat-evoked bradycardia are, as in other mammals, coordinate components of an early stage defense response that facilitates observation and information gathering.

In studies of arousing pleasant pictures (e.g., erotica, pictures of loved ones; Lang et al., 1993; Vico, Guerra, Robles, Vila, & Anllo-Vento, 2010) autonomic engagement is also enhanced relative to neutral cues (e.g., other human faces, objects, domestic animals)—with many reflex responses similar to those prompted by arousing aversive pictures. Thus, greater skin conductance responses and widening of the pupil indicated enhanced sympathetic activation (Bradley et al., 2008). However, the coactive parasympathetic effect, sustained bradycardia, is not observed. That is, arousing pleasant pictures during free-viewing prompted only a brief decelerative orienting (as for neutral scenes) followed by significant cardiac acceleration—vagal release that is coordinate in this context with a general sympathetic reaction to highly engaging pleasant cues.[2]

As previously noted, brain circuit activation in response to appetitive cues includes many of the same neural structures activated in defensive arousal (e.g., amygdala), consistent with the broad similarity in ANS response. Nevertheless, both animal and human research have implicated additional neural structures specific to appetitive stimulation—the ventral striatum (e.g., nucleus accumbens) and ventral medial prefrontal cortex (e.g., Peciña, 2008; Smith & Berridge, 2007). In an fMRI study of picture perception (Sabatinelli, Bradley, Lang, Costa, & Versace, 2007), for example, we found marked activation in these striatal and cortical sites when participants viewed pleasant scenes (erotic or romantic). Although the amygdala response was similarly robust for both pleasant and unpleasant scenes, the latter pictures (depicting physical threat/abuse, mutilated bodies) failed to activate ventral medial prefrontal cortex or the nucleus accumbens. We followed up this research with a similar investigation (Costa, Lang, Sabatinelli, Versace, & Bradley, 2010) in which participants processed texts describing pleasant, neutral, and unpleasant events. Again, the same appetitive circuit was activated—amygdala, accumbens, and medial prefrontal cortex—only by arousing pleasant cues. How these subcircuit differences directly relate to the different ANS responses found for pleasant and unpleasant stimuli is an important task for future research.

Question 1b: What is the relation of observed patterns to what have been proposed as discrete emotions?

Propelled by the seminal James–Lange theory of emotion (James, 1884, 1890, 1894), the 20th century saw a host of experiments testing the hypothesis that reports of discrete emotions (e.g., fear, anger, joy) are associated with discrete patterns of autonomic reactivity. In aggregate, the research has

not supported the hypothesis (see Lang, 1994, for a full discussion of these issues). That is, although such a relationship has sometimes been claimed, a generally accepted list of discrete emotions clearly associated with specific ANS patterns has not surfaced.

Kreibig (2010) recently assessed a massive catalogue of experiments on ANS activation and emotion. These many investigations targeted a host of different candidate affects, using a great diversity of experimental paradigms, timing periods and measures, and generating a complexity of results that challenges meaningful summarization and interpretation. As the author concluded, "Collecting valid data on autonomic responding in emotion has been and remains to be a challenge to emotion research," and further, "future researchers will have to closely scrutinize and, if possible, verify the specific type of emotion elicited" and thus answer "James' (1890) call for a generative principle that can … account for the varieties of emotion" (p. 411).

Following James, most investigators have proposed that an individual's "feelings," his/her affective conscious experience is the signature of a discrete emotion, and furthermore, that the evaluative reports of participants can provide verifying evidence that the emotional experience has occurred. At midcentury, Mandler and colleagues tested this view in an impressive series of experiments (Mandler & Kremen, 1958; Mandler, Mandler, Kremen, & Sholiton, 1961; Mandler, Mandler, & Uviller, 1958), assessing the covariation of reported affect and autonomic activity. His final conclusion after years of research: "None of the evidence suggested that different kinds of emotional responses were associated with different patterns of visceral response" (Mandler, 1975, p. 130). Our own research has been consistent with this finding. For example, when participants (N = 64; Lang et al., 1993) viewed a range of images varying in reported intensity of emotional arousal, the *group data* showed an expected covariation with increasing skin conductance responses. However, when Pearson correlations were calculated for individuals (over the picture series) between these simple ratings of experienced emotional arousal and their conductance responses, the statistic was significantly different from zero (r = .37) in less than a third of the participants.[3]

There is, furthermore, a long history of research with patients—anxiety and mood disorders—consistently showing low correlations between report of fear and physiological reactivity (e.g., Cook, Melamed, Cuthbert, McNeil, & Lang, 1988; Lang, 1964, 1968, 1978; Marks & Huson, 1973; McTeague & Lang, 2012). This lack of concordance is also found in the context of treatment: Rachman and Hodgson (1974) examined assessed verbal reports of fear, fear behaviors, and physiological reactivity during therapeutic intervention, and found highly desynchronous patterns of change with treatment. These results undermine the Jamesean hypothesis. How can reports of feelings be a perception of the physiology, if the two events are not reliably coincident?

A further problem for the view that discrete emotions have a specific autonomic signature concerns the marked response differences that are primarily attributable to the source of the evoking stimulus—external or internal. For example, when participants respond to a perceived event in the surrounding environment (e.g., viewing a picture with threatening content, or hearing a tone that signals an imminent electric shock), many human participants report "fear" and, as already noted, their response is predominantly heart rate deceleration, parasympathetically mediated. However, if a participant is retrieving the memory of a reportedly frightening event, the reflex response is sympathetically driven heart rate acceleration (e.g., Bradley & Lang, 2007; Lang, Levin, Miller, & Kozak, 1983; McTeague, Lang, Wangelin, Laplante, & Bradley, 2012). It is noteworthy that for both external and internal cues, the primary neural structures of the brain's motivational circuitry (e.g., amygdala) appear to be similarly activated (Lang & Bradley, 2010), yet clearly mediate different ANS effects downstream.[4]

Research suggests (Bradley & Lang, 2007; Lang, Bradley, & Cuthbert, 1997), furthermore, that the autonomic reaction to an emotional event is often a moving target—changing with an altering context (but not in its affect label). For example, we studied ANS responses to increasingly proximal cues that threatened a loss, or alternatively, signaled an emotionally positive event—the chance to receive a valued reward (Löw, Lang, Smith, & Bradley, 2008). When the appetitive reward cue was very close, a key press was required that if fast enough, resulted in a monetary gain; when the unpleasant/threatening cue was similarly proximal, a slow response resulted in a financial loss. Autonomic reactions to these cues varied dramatically with the cue's imminence. For both appetitive/pleasant and aversive/unpleasant cues, skin conductance increased monotonically with proximity, sharply ascending just prior to the anticipated response. Conversely, after a brief orienting response, heart rate increased modestly to both threat and reward cues when they were still at a distance; however, with cue approach, a progressively more marked deceleration began, most profound just before the most proximal approach. Although this cardiac pattern was similar both in anticipating a possible loss or a hoped for reward, the deceleration component was significantly greater for loss/threat cues—as in so called "fear bradycardia." Penultimately for both cue types, immediately prior to the response cue, heart rapidly accelerated for both aversive and appetitive stimuli.

The experiment was intended to assess the predator–prey context in human participants. The results are consistent with the animal data: An alerting response occurs when predator–or prey–is at distance (*postencounter*; Fanselow, 1994), i.e., "freezing," orienting, and heart rate deceleration that increases with proximity; subsequently in the *circa-strike zone* where action is most imminent, the sympathetic system dominates, prompting both a rapidly increasing sweat gland response and rapid heart rate acceleration. This cascade of changing responses (and I have not mentioned the pattern of associated somatic reflexes) is readily characterized as emotionally engaging. There is, however, nowhere the stability implied by the concept of discrete emotions with unique autonomic signatures.[5]

Question 2: What role does activity in the autonomic nervous system serve further downstream? What, for example, is the status of feedback hypotheses according to which subjective emotional experience is influenced by activity in the autonomic nervous system?

In Thomas Kuhn's famous view (1962/2012), investigation in any field of scientific study begins with a prolonged period of highly diverse experimentation prompted by many different theories and ideas. This scattered preparadigm stage, gives way to "normal science" when investigators coalesce around a common, and broadly applicable conceptual framework. The field then has a "dominant paradigm," and from this point on, researchers are generally assessing aspects of this common view, testing predictions, elaborating the model, refining measurement technologies, and addressing the paradigm's inevitable puzzles, discovered inconsistencies, and/or surprising new directions for investigation.

Kuhn's paradigmatic examples were mainly from physics and astronomy. However, it seems reasonable to consider scientific psychology as having been in this preparadigm stage in the latter half of the 19th century. In its last decade, however, a theoretical view emerged that arguably became the dominant paradigm in the psychophysiology of emotion for the subsequent hundred-plus years. I have elsewhere written at length on the emergent James–Lange theory, on its scientific significance, and its century-long reign over much psychophysiological research (Lang, 1994). It is the continuing aura of this theory, of course, that yet necessitates the question: "is … subjective emotional experience … influenced by activity in the autonomic nervous system?" For indeed, we continue to grapple with the Jamesean hypothesis—that emotional experience is an apperception of physiological changes, autonomic and somatic, activated by a prior emotionally arousing cue (the *angry* bear).

In the late 19th century, psychology was consensually defined as the study of consciousness, and the specific intention of James's theory was to explain the phenomena of conscious emotional experience. His view was radical only in the temporal primacy given to the physiology of emotion, that is, the act preceding the thought. In either case, a true test of their relationship requires that we have objective measures of both phenomena—on the one hand, physiological reactivity and on the other, conscious "feelings" (Lang, 1994). Physiological measurement is of course practical and highly developed in scope and precision. However, what has come to be called "the hard problem of consciousness" (e.g., Chalmers, 1995) in philosophy has not been solved and nothing augurs that it will be resolved anytime soon. Indeed, there is doubt that consciousness ever could be elucidated in the physical terms of natural science. Emotional "feelings" remain the private experience of individuals and opaque to direct scientific inquiry, and as such, are a poor place to begin a study of the manifest information processing behavioral functions and dysfunctions grouped under the lay term, emotion.

As has been noted (e.g., Lang, 1988), there are three measurement domains available to a science of emotion: affective language (evaluative and expressive), overt behavior, and physiological reactivity. The dominant theory of their relationship hypothesizes an organizing, inner affective experience. We have already considered here the long pursued efforts to use evaluative language reports as a stand-in for conscious experience, and the result of a century's effort—a lack of reliable association with physiological reactivity. Have sufficient anomalies not yet accrued? Has no alternative view emerged that might better guide emotion science?

In recent decades there have been dramatic advances in study of the brain, and importantly, in understanding the neural structures and circuits that are active in threat and reward and mediate visceral and behavioral responses in animal subjects. Furthermore, the development of brain imaging technology has opened the way to parallel investigations of human brain function. Methods such as functional magnetic resonance imaging (fMRI) are, of course, indirect and compared to the invasive animal research, blurred views of the brain's complexities. However, both the science and the supporting technology have already made impressive progress. In this regard, I suggest that we may be at the beginning of what Kuhn called a "paradigm shift," in which subjective experience is not the central integrating concept, nor the immediate thing to be explained. Taking its place may be a developing brain model. In this paradigm *emotion is what emotion does*. In contexts we call "emotional" the brain acts, on the one hand, to increase attention, perceptual processing, and mediate a medley of instrumental actions; or on the other, given dysfunction in the brain's evolved motivational systems, it may prompt cognitive disarray, helplessness, and ineffectual coping behavior.

From a new neuroscience perspective, the issue of autonomic nervous system (ANS) feedback and its effect on conscious experience is not a practical question. Determining the relationship between brain function and ongoing ANS activity under conditions of threat, stress, or reward is, however, a highly significant and attainable goal. In general, organ systems have both efferent and afferent connections to the central nervous system (CNS), and their interaction is essential in regulating visceral muscle or gland. The view, for example, that afferent feedback from the cardiovascular system modulates brain activity has long been studied (e.g., via blood pressure changes at the baroreceptor neurons; Lacey & Lacey, 1978). Highlighting the potential range of such interactions, a fascinating recent study suggests that microbiota in the gut are important for healthy brain function, that pathogenic bacteria in the gastro-intestinal tract activate neural pathways to the central nervous system and may be contributory in report of anxiety and depression (Foster & Neufeld, 2013).

Importantly, greater attention should be paid in human imagining studies to the downstream, reflex consequences of circuit activation. Establishing these efferent connections is the basic first step, after which ANS afferents from the same organ system could be evaluated for their effects on brain function. In animal studies, analysis of the brain circuit is inevitably tied to a

behavioral or ANS-dependent variable. Thus, as already noted, Fanselow's (1991) related "freezing" behavior to activation of the central grey, and Kapp (e.g., Pascoe & Kapp, 1985) showed a significant covariation between amygdala activation and ANS-mediated cardiac activity. More studies with human participants are needed like the Hermans et al. study (2013), in which brain activation patterns were directly related to a concurrently recorded psychophysiological measure (pupil dilation, heart rate). Recent studies by Mobbs and colleagues provide another example. Skin conductance was recorded in the scanner while participants performed a virtual predator–prey task, under risk of electric shock. Electrodermal reactivity increased from low threat (*pre-encounter*) to high shock threat (circa-strike), coordinate with increasing activation of the periaqueductal central gray and defense circuit connectivity (Mobbs et al., 2009; see also, Mobbs et al., 2007).

Question 3: Given the available research and the methods used, what is the appropriate level of confidence in conclusions drawn in this field? What obstacles might we seek to overcome so that the level of confidence can be increased?

The field of emotion research continues to be a highly diverse terrain of different theories and methods that have so far not yielded consensus conclusions. I have proposed here (see also, Lang, 2010), however, that the study of autonomic patterns in emotion can be most confidently addressed within the parameters of the developing brain model. In this paradigm, reported emotions reflect, however imperfectly, the activation of neural circuits that evolved in the mammalian species because they facilitated the survival of individuals and their progeny. While the circuitry is highly overlapping in its individual neural structures and their connectivity, the animal findings suggest two basic subcircuits: an appetitive circuit that mediates behaviors sustaining the organism and promoting progeny (e.g., sustenance, sex, nurturance); and a defense circuit addressing the many threats to life that may be encountered. These circuits vary in intensity of activation, mediating a gradient of engagement and bodily mobilization that is finely tuned to the significance/imminence of reward and threat. More broadly, the circuitry evolved as the brain's basic motivational system in humans, and its modulated activation (or alternatively, its dysfunction) determines the efficiency of goal-oriented behavior and is a significant influence on evaluative reports.

Presuming the proposed brain model, it is not surprising that factor analytic studies of evaluative language (e.g., Bradley & Lang, 1994; Osgood, Suci, & Tannenbaum, 1957; Russel & Mehrabian, 1977) have consistently found two main integrating factors that account for the majority of the variance, and seem to parallel the broad functions of the brain's survival system—affective valence (pleasant/unpleasant) and emotional arousal. That is, these factors can be said to reflect the two evolved motivational circuits, appetitive (pleasant) and defensive (unpleasant) and an intensity gradient of circuit activation (arousal). Furthermore, as already described, group ratings of these factors show substantial correlations with reflex reactions to emotional stimuli—a finding obtained repeatedly with different samples of participants.

These semantic analyses suggest that the folk psychology of emotional language may indeed have its origins—however remote—in the motivational brain and its reactive physiology. However, as already noted, within individuals the relationship between physiology and even these general factors is highly variable, with many participants showing little or no covariation. This should not surprise, as the use of evaluative emotional language is subject to independent modification unrelated to circuit activation, varying with the individual's unique reactional biography, idiographic patterns of reinforcement, and learned instrumental use in social communication.

In concluding, it is timely to consider the clinical implications of the present discussion—how advances in understanding brain circuits and their mediated visceral physiology in emotion might facilitate improved prevention, diagnosis, and treatment of emotional pathology. Currently, the high prevalence of affective disorders—fear, anxiety, and mood—is a major public health concern (e.g., Lépine, 2001, 2002). Indeed, the consequences extend beyond reported mental pain, and include social dysfunction, reduced productivity, work hours lost, economic distress, and high risk for cardiovascular disease and other conditions of autonomic nervous system illness (e.g., Barger & Sydeman, 2005).

As for discrete emotions, the different categories of emotional disorder (e.g., specific phobia, panic disorder, dysthymia, major depression: DSM-IV) are mainly based on assessment of the patient's evaluative report of their emotions—his/her fears, feelings of sadness, worries, depressive thoughts, and associated inner experience. These conditions differ from most other illnesses in that there are no ancillary objective measures to supplement symptom report (i.e., consider treating heart disease with only the patient's symptom report to go on—without an electrocardiogram (EKG), cardiac imaging, vascular measures, and blood analysis). The diagnostic reliability of emotional disorders is an enormous problem, multiple comorbidities are ubiquitous, and a principal diagnosis rarely defines a distinct category of disease with an associated specific treatment. Furthermore, analytic studies of comorbidity patterns (e.g., Bienvenu et al., 2001) and emerging genetic findings (e.g., Hettema, Neale, Myers, Prescott, & Kendler, 2006) often suggest a very different organization of these disorders, more dimensional than categorical.

In response to these issues, the National Institute of Mental Health (NIMH) recently proposed a new focus for clinical research, the Research Domain Criteria (RDoC) initiative, Strategy 1.4 of the current NIMH Strategic Plan, encouraging the development of biomarkers that might better define emotional disorders: "If we assume that the clinical syndromes based on subjective symptoms are unique and unitary disorders, we undercut the power of biology to identify illnesses linked to pathophysiology and we limit the development of more specific treatments" (NIMH, 2010). In effect, basic and clinical researchers in emotion are invited to "develop for research purposes, new ways of classifying disorders based on dimensions of observable behavior and neurobiological

measures" (see Insel et al., 2010). It is an invitation to conceive emotion and its pathologies not as internal states of mind, but as functional, or dysfunctional, physiology and behavior—brain and its efferent effects on somatic and visceral systems.

A paradigm shift for the study of emotion?

Notes

1 Researchers studying animal subjects have often used the word "fear" in describing threat-related behavior or its mediating circuitry. Recently, Joseph LeDoux (2013) argued that its use should be discontinued. He points out that it is a confusing, "loaded" term with multiple meanings in the "common language," and proposed "threat-elicited defense responses" as a more accurate descriptor. This view is consistent with our use of the term *defense circuit* to describe brain activation to threat (Lang, 2010).

2 It is of interest that threat-evoked bradycardia is found in both mammals and reptiles; however, reptiles do not show deceleration to appetitive/neutral events. That is, cardiac orienting to any novel cue appears to be a later development of the mammalian phylum (Campbell et al., 1997). This broadening of a response—with an apparently more specific prior evolutionary function—is not atypical, and is reflected in the considerable pattern overlap found in human reflex reactions to both appetitive and aversive cues.

3 Interestingly, when facial muscle action is correlated with reported displeasure/distress, more than half of the participants show a significant covariation (Lang et al., 1993)—suggesting a closer relationship between these two modalities of social communication than is found for emotional language and patterns in the autonomic nervous system.

4 John Lacey (1959) described this general phenomenon in reports of his research. He found reliably different relationships between skin conductance and heart rate responses depending on the processing task (e.g., "mental" or perceptual) and called it "directional fractionation." Lacey also studied the reliability of autonomic patterns (blood pressure, heart rate, skin conductance) in response to repeated presentations of the same stressor, as well as the consistency of patterns across different stressors. Wide individual differences were found in both analyses, with some subjects showing highly reliable patterns, some with similar patterns, but also many with little consistency within or across stressors.

5 As this discussion suggests, it is difficult to evaluate the role of the ANS in expressed emotion without also considering co-incident or anticipated motor reactions. The somatic and autonomic systems are synergistic partners in laboratory tasks and in response to affective events, as was long emphasized by Paul Obrist (1981).

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