Autonomic Nervous System Activity in Emotion: A Review

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Abstract

Autonomic nervous system (ANS) activity is viewed as a major component of the emotion response in many recent theories of emotion. Positions on the degree of specificity of ANS activation in emotion, however, greatly diverge, ranging from undifferentiated arousal, over acknowledgment of strong response idiosyncrasies, to highly specific predictions of autonomic response patterns for certain emotions. A review of 134 publications that report experimental investigations of emotional effects on peripheral physiological responding in healthy individuals suggests considerable ANS response specificity in emotion when considering subtypes of distinct emotions. The importance of sound terminology of investigated affective states as well as of choice of physiological measures in assessing ANS reactivity is discussed.

Keywords: emotion, autonomic nervous system, emotional response specificity, autonomic response organization, cardiovascular system, respiratory system, electrodermal system

Autonomic responding in emotion has been an active research topic since, almost a century ago, Walter Cannon (1915) first studied the physiology of emotion (T. M. Brown & Fee, 2002; Dale, 1947). Still, there is no scientific consensus on whether there exists a relation between emotion and the organization of autonomic nervous system (ANS) activity

and, if so, in what form. The various positions, which contemporary researchers hold on this topic, are first addressed in this article, before turning to the physical components—or the hardware—of autonomic responding in emotion. Next, a brief overview of the various theories and models that have been suggested to explain and identify mechanisms of autonomic response organization in emotion is given. The center part of this article consists of a review of the empirical basis for the postulate of emotion-specific ANS activity, considering 134 experimental studies on ANS activity in emotion. The next section summarizes and discusses how empirical emotion effects relate to models of autonomic response organization, points to the importance of choosing adequate measures of autonomic activation components, and addresses the issue of emotion terminology. A final section considers boundary conditions of the definition of emotion employed in the present article and its implications for identifying emotion-specific ANS activation.

Current Positions on Autonomic Responding in Emotion

Contemporary researchers in the field of emotion hold contrary positions on the topic of ANS activation in emotion. At one extreme, Feldman-Barrett (2006, p. 41), for example, stated that "it is not possible to confidently claim that there are kinds of emotion with unique and invariant autonomic signatures," but rather that configurations follow general conditions of threat and challenge and positive versus negative affect. Feldman-Barrett named three points of critique regarding the evidence for autonomic differences between emotions: first, the high heterogeneity of effects in meta-analytical studies (e.g., Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000) is interpreted to suggest the presence of moderator variables in the relation of emotion and ANS activity; second, autonomic differences that do emerge between specific emotions are viewed to be along lines of dimensional differentiation; and third, ANS activity is said to be "mobilized in response to the metabolic demands associated with actual behavior [...] or expected behavior" (p. 41) and because different behaviors have been shown neither to be emotion-specific nor to be context-invariant (e.g., Lang, Bradley, & Cuthbert, 1990), Feldman-Barrett views emotion-specific autonomic patterns as a priori improbable.

An intermediate position is suggested by meta-analyses of physiological responding in emotion (Cacioppo, Berntson, Klein, & Poehlmann, 1997; Cacioppo et al., 2000) that report some degree of autonomic emotion specificity. Besides certain reliable differences between specific emotions, Cacioppo et al. also noted context-specific effects of ANS activity in emotion (i.e., according to different induction paradigms). Moreover, valence-

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specific patterning was found to be more consistent than emotion-specific patterning: negative emotions were associated with stronger autonomic responses than positive emotions (cf. S. E. Taylor, 1991). However, only one positive emotion, happiness, which subsumed joy, was used in the meta-analysis. This unequal representation of merely one positive as contrasted to a sample of five negative emotions may significantly bias the kind of distinction discerned. Due to a limited number of studies considered, a restricted range of physiological variables (only cardiovascular and electrodermal, but no respiratory measures), and the univariate nature of the meta-analytic approach, such results give only an imperfect answer to the question of autonomic patterning in emotion. Authors of review articles thus typically acknowledge that discrete emotions may still differ in autonomic patterns even if they do not differ in single variables (Larsen, Berntson, Poehlmann, Ito, & Cacioppo, 2008; Mauss & Robinson, 2009).

Diametrically opposed to Feldman-Barrett's (2006) position, Stemmler (in press) argued why the ANS should *not* convey specific activation patterns for emotions, if those have specific functions for human adaptation. Stemmler (2004, in press) reasoned that emotions have distinct goals and therefore require differentiated autonomic activity for body protection and behavior preparation. Autonomic activity for behavior preparation is physiological activation that occurs *before* any behavior has been initiated that itself engages the ANS according to behavioral demands. Such autonomic activity has even been reported in experimentally paralyzed animals (Bandler, Keay, Floyd, & Price, 2000), underlining that it is not merely overt behavior that causes this activity. This also resonates with Brener's (1987) notion of "preparation for energy mobilization," which contrasts to Obrist's (1981) view of ANS activity as a component of the motoric response.

Stemmler (2004) reported on a meta-analysis on autonomic responding in fear and anger—two emotions that are believed to share similar valence and arousal characteristics—in which he found considerable specificity between the two. Taking a functional approach to autonomic responding in emotion, Stemmler (2003, 2004) stressed the importance of studying autonomic regulation patterns in emotion rather than single response measures. According to the view that the central nervous system (CNS) is organized to produce integrated responses rather than single, isolated changes (Hilton, 1975), any variable which can be described or measured independently is constituent of several such patterns. Only when considering comprehensive arrays of physiological measures can such regulation patterns be discerned. Stemmler (in press) stressed that this should include variables that indicate both specific and unspecific effects of emotion. Unspecific emotion effects distinguish between control and emotion conditions, but not between emotions, whereas specific emotion effects distinguish between emotions. The pool, from which indicators of independent autonomic activation components can be drawn, is considered in the subsequent section.

Physical Components of Autonomic Responding in Emotion

Although physiologists at the beginning of the last century characterized the ANS as too slow and undifferentiated to quickly produce highly organized response patterns in emotion (Cannon, 1927), contemporary physiologists see considerable room for such organization (Bandler et al., 2000; Folkow, 2000; Jänig & Häbler, 2000; Jänig, 2003; see also Levenson, 1988). Research over the past 50 years has invalidated the view that the

sympathetic devision of the ANS functions in an 'all-or-none' fashion without distinction between different effector organs (Cannon, 1939). Rather, each organ and tissue is innervated by distinct sympathetic and parasympathetic pathways, with very little or no cross-talk between them (Jänig & McLachlan, 1992b, 1992a; Jänig & Häbler, 2000). Pools of sympathetic neurons can be selectively engaged, such that individual systemic circuits or other effector units are independently activated (Folkow, 2000).

The originally assumed functional unity of the sympatho-adreno-medullary system is now known to consist of two separately controlled system parts—a direct-nervous and an adrenomedullary hormonal one—that under most situations have different functional roles (Folkow, 2000). Whereas the former executes precise, rapid, and often highly differentiated adjustments, the latter independently modifies important metabolic functions. In some emergency situations, where massive and generalized sympatho-adrenal system activation can occur, the two parts may also mutually support each other.

The inclusion of respiratory measures under autonomic measures also deserves some comment here. Respiratory activity evidences effects of autonomic control as well as significant independent contribution of peripheral and central chemoreceptors sensitive to CO_2 (Wilhelm, Schneider, & Friedman, 2005). Measures of respiratory activity may thus yield additional information on ANS functioning in emotion to that indicated by cardiovascular and electrodermal measures. There moreover exist important interactions of the respiratory system with the cardiovascular system, as, for example, attested by the phenomenon of respiratory sinus arrhythmia (Grossman & Taylor, 2007). Here, respiratory measures are important in the interpretation of effects of ANS functioning indicated by cardiovascular measures, which are modulated by respiratory effects. Finally, the cardiorespiratory control system can be viewed as one functional unit as it pursues the common aim of providing the tissues with oxygen, nutrients, protective agents, and a means of removing waste by-products (e.g., Feldman & Ellenberger, 1988; Poon & Siniaia, 2000; E. W. Taylor, Jordan, & Coote, 1999). Thus, comprehensive assessment of cardiovascular, electrodermal, and respiratory measures can provide complimentary information on ANS functioning in emotion.

Central coordination of autonomic activity represents a cornerstone of current views of integrated nervous system functioning (cf. central autonomic network, CAN; Benarroch, 1993, 1999; see also Damasio, 1998; Thayer & Lane, 2000). Unlike the original conceptualization of the ANS as functioning independently of the rest of the nervous system (e.g., involuntary, automatic, and autonomous control), close interactions between the central and autonomic nervous systems exist in various ways. Thus, like the somatic nervous system, the ANS is integrated at all levels of nervous activity. Whereas segmental autonomic reflexes are coordinated by the spinal cord, suprasegmental integration higher in the brain stem is required for regulation of functions such as respiration, blood pressure, swallowing, and pupillary movement. More complex integrating systems in the hypothalamus influence the brain stem autonomic subsystems. Many of the activities of the hypothalamus are, in turn, governed by certain cortical areas, particularly the insular, anterior cingulate, and ventromedial prefrontal cortices as well as the central nucleus of the amygdala, that process inputs from the external environment. Thus, fundamental adjustments of the organism to its environment can only be attained by the concerted coordination and integration of somatic and autonomic activities from the highest level of

neurological activity in the cortex down to the spinal cord and peripheral nervous system. This high degree of specificity in ANS organization is needed for precise neural regulation of homeostatic and protective body functioning during different adaptive challenges in a continuously changing environment. In this context, emotions may provide quick and reliable responses to recurrent life challenges. But still, the question remains how autonomic response organization in emotion might be achieved.

Conceptual Levels of Autonomic Response Organization in Emotion

William James is often credited for originating the idea of peripheral physiological response specificity in emotion (e.g., Ellsworth, 1994; Fehr & Stern, 1970; see also Friedman, this issue, for a historical overview). James's (1884) proposal that the feeling component of emotion derives from bodily sensations, i.e., the perceived pattern of somatovisceral activation, reversed the causality of emotion and bodily responding. Acknowledging a high degree of idiosyncrasy in emotion, James stated "that the symptoms of the same emotion vary from one man to another, and yet [. . .] the emotion has them for its cause" (1894, p. 520). Even more so, James believed that the physiological responses were "almost infinitely numerous and subtle" (1884, p. 250), reflecting the infinitely nuanced nature of emotional life. Still, James recognized limits to bodily variations in emotion: "the symptoms of the angers and of the fears of different men still preserve enough *functional* resemblance, to say the very least, in the midst of their diversity to lead us to call them by identical names" (1894, p. 520, emphasis in original). James thus strongly argued for "a deductive or generative principle"(James, 1890, p. 448) that may explain physiological response specificity in emotion.

James' claims associated with his peripheral perception theory of emotion were met with differentiated reactions—they instigated critique (most prominently the five-point rebuttal by Cannon, 1927), support (e.g., Angell, 1916), as well as various propositions for general organizing principles of autonomic responding in emotion. Although a number of different models have been proposed since then, these co-exist in a rather disjunct fashion, without clear empirical rejection of one or the other. As detailed in Kreibig (in press), the various models of autonomic responding in emotion can be organized by recognizing that these models address different conceptual levels, on which an organizing principle of autonomic responding in emotion may operate. Table 1 shows how the various theories map onto different conceptual levels that span from the physiological over the behavioral to the psychological level. A first class of models is identified, which draw on a basic physiological systems level; these are models that see the organizing principle of autonomic responding in emotion in the structure and functioning of the ANS or in the functioning of transmitter substances. A second class of models is based on brain–behavior interactions and views the organizing principle of autonomic responding in emotion in the functioning of brain–behavioral systems and refined behavioral modes. A third class of models centers on psychological processes of meaning assessment and memory retrieval; these models place particular emphasis on the functioning of psychological appraisal modules and associative networks as a general organizing principle of autonomic responding in emotion. A detailed discussion of the various models on each level can be found in Kreibig (in press). It is of note that from a component-view of emotion (Scherer, 2009), models on the same conceptual level rival each other. In contrast,

Table 1 Conceptual Levels of Autonomic Response Organization in Emotion (Kreibig, in press)

Psychological level	
Functioning of Appraisal Modules	
Componential process model	Ellsworth (1994); Ellsworth & Scherer (2003); Scherer (1984, 1987, 2001, 2009)
Specific cardiovascular appraisal hypotheses	Blascovich & Katkin (1993); Blascovich, Mendes, Tomaka, Salomon, & Seery (2003); Gendolla (2004); Gendolla & Wright (2005); Wright (1996, 1998); Wright & Kirby (2001)
Functioning of Associative Networks	
Bio-informational theory of emotional imagery	Lang (1979, 1993); Miltner, Birbaumer, & Gerber (1986); Sartory (1993)
Brain–behavioral level	
Functioning of Brain–Behavioral Systems	
Behavioral coping	Obrist (1981); Schneiderman & McCabe (1989)
Dual-system models	Bradley & Lang (2000); Cloninger (1987); Davidson (1998); Lang & Bradley (this issue); Lang, Bradley, & Cuthbert (1997)
Polyvagal theory	Porges (1995); Porges, Doussard-Roosevelt, Portales, & Greenspan (1996); Porges (2001, 2007)
Reinforcement Sensitivity Theory	Beauchaine (2001); Corr (2008); Fowles (1980); Gray (1982, 1987); Gray & McNaughton (2000)
Functioning of Behavioral Modes	
Basic modes of defensive coping	Folkow (2000); Stemmler (in press)
Modes of defensive coping and environmental demands	Bandler & Shipley (1994); Bandler et al. (2000); Bernard & Bandler (1998); Keay & Bandler (2001, 2002)
Predator imminence model	Bradley & Lang (2000); Craske (1999); Fanselow (1994); Lang et al. (1997)
Peripheral physiological level	
Functioning of Autonomic Systems	
Undifferentiated sympathetic activation	Cannon (1915, 1927)
Parasympathetic activation	Kling (1933); Vingerhoets (1985); Vingerhoets, Cornelius, Van Heck, & Becht (2000)
Sympathetic versus parasympathetic response dominance	Gellhorn (1964, 1965, 1970); W. R. Hess (1957)
Autonomic space	Berntson, Cacioppo, & Quigley (1991)
Functioning of Transmitter Substances	
Catecholamine hypothesis	Ax (1953); Funkenstein, King, & Drolette (1954)
Receptor-types hypothesis	Stemmler (2003, 2004, in press)

models on different levels have complementary value, as they address different levels of response organization (cf. Mausfeld, 2003). It will be seen in the discussion section how these models fit with the empirical findings that are presented next.

Empirical Findings of ANS Activity in Emotion

To what extent are postulated differences between emotion reflected in empirical data on ANS functioning? To address this question, a qualitative review of research findings was carried out, focusing on effects of experimentally manipulated emotions on ANS responding in healthy individuals. To cover both the psychological and medical literature, an exhaustive literature search using the databases PsycINFO, PsycARTICLES, and PubMed was conducted with the following search terms:

[emotion] *and* [autonomic nervous system *or* cardiovascular *or* cardiac *or* heart

or respiration *or* respiratory *or* electrodermal *or* skin conductance]

References of such identified publications were additionally screened for further research reports falling under the specified criteria. Because the present review aimed at surveying the extent to which autonomic effects of emotion are reported in research studies, an inclusive approach was chosen, applying only basic validity and reliability criteria to study selection. Publications were included in the final selection if data from an original experiment were reported, in which emotions were manipulated and ANS measures were assessed during emotional responding. Emotion, for this purpose, was broadly defined, covering definitions of dimensional models of emotion (Bradley & Lang, 2000; Lang, 1994; Russell, 2003), discrete emotion theory (Ekman, 1999; Izard, 1992), as well as appraisal models of emotion (Scherer, 2001; Smith & Kirby, 2004). Emotion was thus conceptualized as a multi-component response to an emotionally potent antecedent event, causing changes in subjective feeling quality, expressive behavior, and physiological activation. Terms such as *mood* or *affect* were considered synonymous with *emotion*, if the experimental manipulation targeted a stimulus- or event-related change of subjective feeling (see the concluding section for boundary conditions for such a conceptualization of emotion). Experiments involving patient groups and/or emotion regulation were excluded; control groups of these studies were, however, included (i.e., healthy individuals or unregulated responding, respectively). Publications were also excluded if no specific emotion label was provided or if no specific emotion contrasts were tested (e.g., if only reporting valence and/or arousal contrasts or only coding according to positive/negative affectivity). Publications were moreover excluded if not measuring physiological activity during the period of emotional responding, not reporting data from an original experiment, or not reporting analyses pertinent to the present review (e.g., regression or pattern classification were not considered). Articles were also excluded if, instead of individual physiological variables, a composite score was formed and only this measure was reported.

This literature search resulted in the identification of 134 publications. A detailed account of the studies included in the present review can be found in Table A1 (Appendix). To summarize this information, tag clouds were created. A tag cloud is a visualization of word frequency in a given database as a weighted list. For the present purpose, coding labels in Table A1 were used as tags (drawn from individual columns). The absolute frequency of tag occurrence is visualized with font size. Tag clouds were created with the Wordle.net web application (http://www.wordle.net/). Figure 1 presents an illustration of the relative number of studies that investigated different emotions (Figure 1a), using different kinds of emotion induction paradigms (Figure 1b), and quantified physiological variables according to different averaging durations (Figure 1c). It can be seen from these illustrations, that the emotions most often investigated are anger, fear, sadness, disgust, and happiness. Experimental manipulations most often utilize film clips for emotion induction, followed by personalized recall, real-life manipulations, picture viewing, and standardized imagery. Response measures are most often averaged over 60- or 30-second intervals; other common averaging intervals are 1/2- or 10-second intervals and 120-, 180-, or 300-second intervals. It is noted that studies were coded for averaging period because it was hypothesized that this factor might influence the reported pattern

of physiological responses. This effect was, however, not observed in the present data and is thus not further considered here.

Figure 2 provides an illustration of the relative number of studies that have used different cardiovascular (Figure 2a), respiratory (Figure 2b), or electrodermal (Figure 2c) measures as well as their overall usage (Figure 2d). These figures show that heart rate is the cardiovascular response variable most often reported; other popular cardiovascular measures include systolic and diastolic blood pressure, heart rate variability, and finger temperature. For respiratory measures, respiration rate is the most often reported index together with respiratory period and respiratory depth as well as tidal volume, duty cycle, and respiratory variability. For electrodermal measure, skin conductance level is the response variable most often reported, followed by skin conductance response rate and skin conductance response amplitude. Over all autonomic measures, heart rate is the indicator most often reported, followed by skin conductance level and other cardiovascular variables.

Reports of physiological responses in emotions were coded according to the emotion label provided by the authors and subsequently grouped together based on synonymous expressions drawn from *Merriam-Webster Online Dictionary* (2009). Thus, six negative and eight positive emotion groups, and two emotion groups without clear valence connotation were identified (labels subsequently listed in parentheses were considered synonymous).

For the negative emotions, these were:

(a) anger (approach-oriented anger, withdrawal-oriented anger, anger in defense of other, anger in self-defense, indignation);
(b) anxiety (dental anxiety, performance anxiety, agitation);
(c) disgust (disease-related disgust, food-related disgust);
(d) embarrassment (social anxiety, shame, social rejection);
(e) fear (threat);
(f) sadness (achievement failure, dejection, depression).

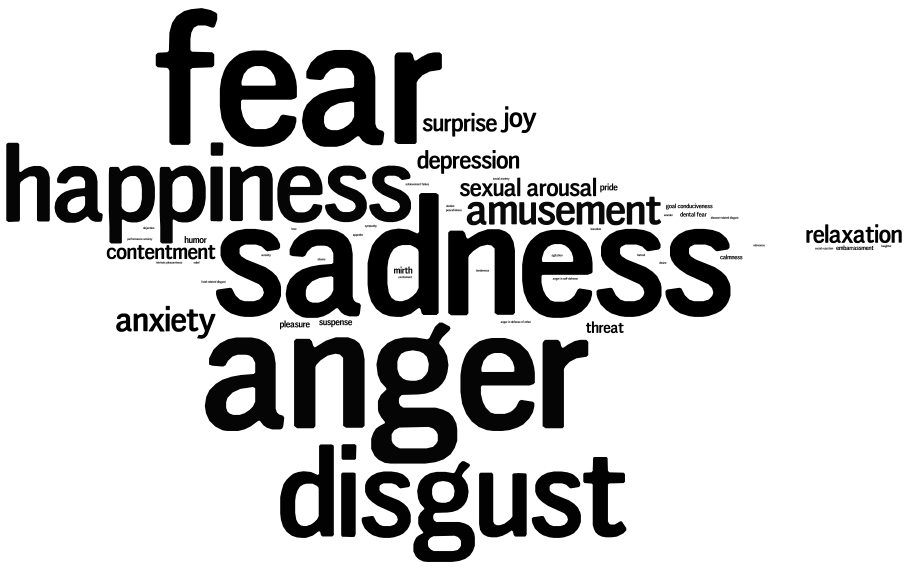
For the positive emotions, these were:

(a) affection (love, tenderness, sympathy);
(b) amusement (humor, mirth, happiness in response to slapstick comedy);
(c) contentment (pleasure, serenity, calmness, peacefulness, relaxation);
(d) happiness (except happiness in response to slapstick comedy);
(e) joy (elation);
(f) anticipatory pleasure (appetite, sexual arousal);
(g) pride;
(h) relief (safety).

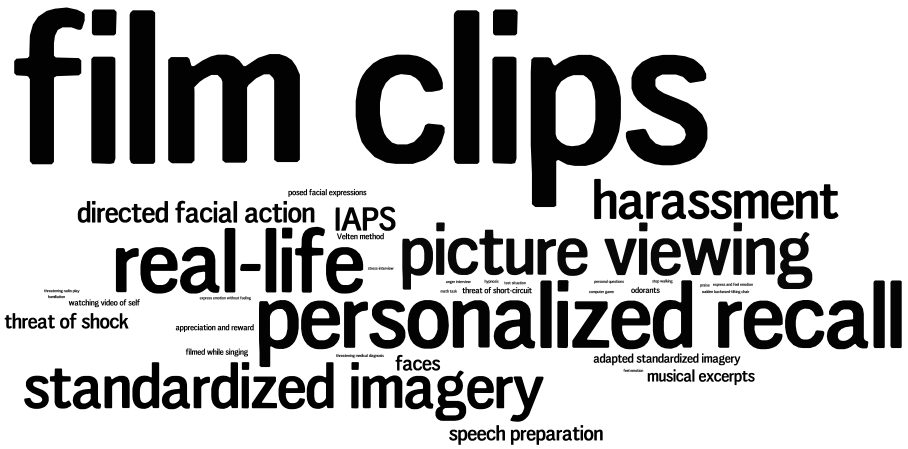
For the emotions without clear valence connotation, these were:

(a) surprise (wonder);
(b) suspense.

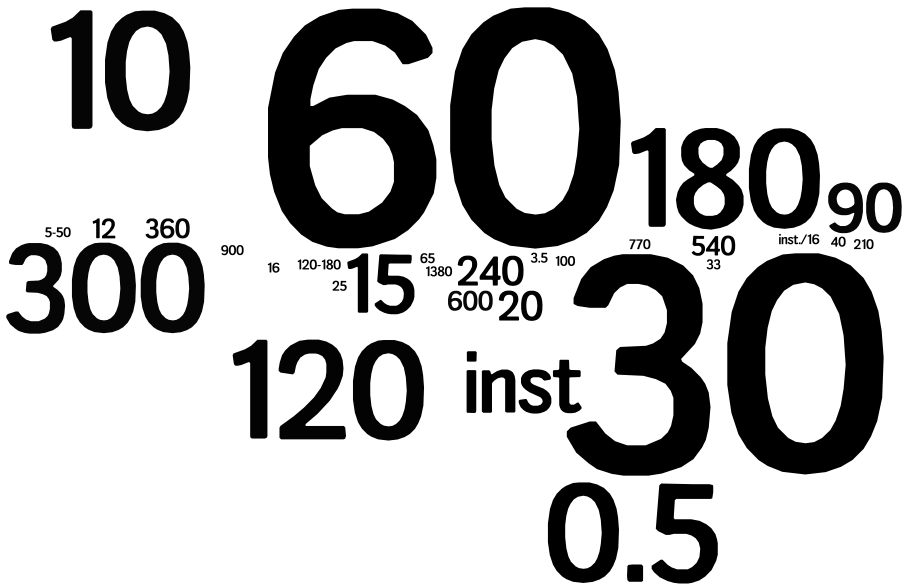
Subsequent sections present a summary of findings of autonomic emotion responses reported in studies described in Table A1 (numbers in brackets refer to the study number in Table A1). Direction of change in ANS activity was coded as change from baseline or, if present, from a neutral comparison condition. Table 3 gives abbreviations, full names, and near-synonymous expressions of autonomic measures used in the fol-



(a) Investigated emotions.



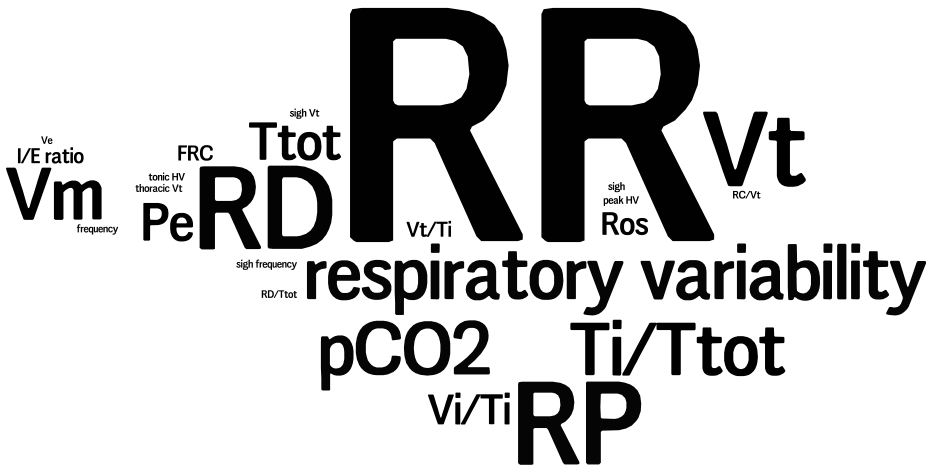
(b) Emotion induction methods.



(c) Averaging duration of physiological variables (in sec).

Figure 1 Illustration of relative frequency of investigated emotions (Figure 1a), emotion induction methods (Figure 1b), and averaging duration for physiological variables (Figure 1c).

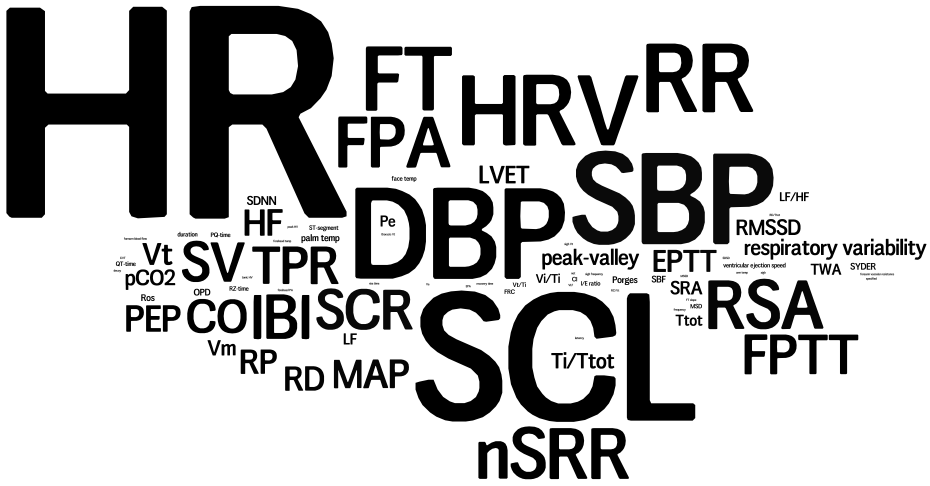
(a) Cardiovascular measures.



(b) Respiratory measures.

Figure 2 Illustration of relative frequency of use of ANS measures as indicated by relative font size.

(c) Electrodermal measures.



(d) All measures of autonomic functioning.

Figure 2 Continued.

lowing. It should be stressed that the current review is of qualitative nature; thus, the results of different studies were not integrated using a weighing procedure that considers sample size, mean, and standard deviation, and thus power of a study. Rather, to organize and integrate the different findings reported in the various studies, a modal response pattern was defined as the response direction reported by the majority of studies (unweighted), with at least three studies indicating the same response direction. Modal response patterns for each emotion are summarized in Table 2.

The Negative Emotions

Anger Physiological responding in anger-eliciting contexts of harassment or personalized recall describe a modal response pattern of reciprocal sympathetic activation and increased respiratory activity, particularly faster breathing.

In particular, the anger response is characterized by α- and β-adrenergically mediated cardiovascular effects: increased HR, increased SBP and DBP, and increased TPR, accompanied either by increased SV and CO [51, 104], decreased SV and increased CO [88, real-life 111], decreased SV and unchanged CO [83, 89], or decreased SV and CO ("anger out", i.e., anger directed outward away from the self) [40, 54]. Increased SBP, DBP, CO, and TPR, but no increase in HR and SV (stressful interview) [2] as well as increased HR, SBP, DBP, SV, CO, and unchanged TPR (personalized recall) [106] have also been reported. Other studies, that did not assess all indices, produce partial replications [7, 14, 25, 29, 35, 36, 37, 55, 63, 80, 87, 90, 96, 119, imagery-task 105, 107, 113, 123, 128, 130, 131, 134]. This response pattern is further characterized as an α- and β-adrenergically mediated response by measures indicating shortened PEP [54, 81, 83, 87, 106, 111] and LVET [81, 106, 111], lower TWA [110, 111], increased HI [81, 110, 111], and increased R–Z time [110]. Moreover, decreased FPA [29, 110, 111, 123] or unchanged FPA [75], and shortened FPTT [75, 111, 123], decreased HT [7, 104] and FT [7, 98, 107], increased HT [109, 111], or unchanged FT [89] point to vasoconstrictive effects in the periphery and local increases of circulation in the face.

Cardiac parasympathetic inhibition is indicated by decreased HRV (MSD [21]; spectral RSA [77]; RMSSD [87, 110]); others have found unchanged HRV (peak-valley and spectral RSA, RMSSD, MSD, SDNN [90]; SDNN [113]). Reports furthermore indicate increased electrodermal activity (increased SCR [29]; increased nSRR [7, 65, 87, 111]; increased SCL [7, 21, 35, 37, 77, 80, 93, 98, 107, 109, 111, 115]), additionally implicating sympathetic effects at the eccrine sweat glands, an effect which is cholinergically mediated.

For respiratory variables, findings indicate increased respiratory activity, particularly faster breathing. Specifically, unchanged [14] or increased RR [7, 34, 75, 80, 90, 93], shortened Ti and Te, increased Pi [15], shortened Te and decreased I/E-ratio [80], increased [34], unchanged [75], or decreased [15] respiratory depth, and increased FRC, increased Ros [93], and increased variability of respiratory amplitude [90] have been found.

Two exceptions to the modal response pattern of reciprocal sympathetic activation in anger are noteworthy: first, responding to material that features expressions of anger differs from responding to harassing material. Specifically, physiological responding to picture viewing of facial emotional expressions of anger diverges such that HR decelerates instead of an acceleration, SCL decreases instead of an increase, and HRV (spectral RSA)

Table 2 Overview of Modal* ANS Responses Found for Reviewed Emotions.

	Anger	Anxiety	Disgust Contamination	Disgust Mutilation	Embarrassment	Fear	Fear Imminent threat	Sadness Crying	Sadness Noncrying	Sadness Anticipatory	Sadness Acute	Affection	Amusement	Contentment	Happiness	Joy	Antic. Pleasure Visual	Antic. Pleasure Imagery	Pride	Relief	Surprise	Suspense
Cardiovascular																						
HR	↑	↑	↑--	↓	↑	↑	↓	↑	↓	↑	↓	↓	↓↑	↓	↑	↑	↓	↑	↓↑	↑--	↑	(↓)
HRV	↓	↓	↑	--	(↓)	↓	(--)	--	↓	(↓)	↑--		↑	↓↑	↓	(↑)	(↑)		(--)			
LF		(↑)				(--)									--							
LF/HF		(↑)		(↓)									(--)									
PWA						(↑)																
TWA	↓			(↑)		(↓)								(↑)								
LVET	↓		(↓)	(↓)		↓				↓↑				(↑)	(–)	(↓)						
HI	↑					(↑)																
PEP	↓		(↓)	(↓)	(↓)	↓				↓↑	↑		(↑)		(↑)	↓↑			(--)			
SV	↓↑	(--)	↓	(--)		↓				↓--					(--)	(↓)						
CO	↓↑	(↑)	(↓)	(↓)	(–)	↑				↑--			(↓)		(–)	--			(--)			
SBP	↑	↑	↑	↑	(↑)	↑				↑	(↓)		↑--	(↓)	↑	↑						
DBP	↑	↑	↑	↑	(↑)	↑				↑	↓--		↑--	(↓)	↑	--						
MAP			↑	↑		↑					↓--		↑--	(↓)	↑							
TPR	↑		↑	(--)	(↑)	↓				↑			(↑)		(↑)	(--)			(--)			
FPA	↓	↓	↓	↓		↓	(↓)	↓	↓	(↑)	↓		(↓)	(--)	↓↑					(↑)		
FPTT	↓	(↓)	↓↑	↓↑		↓	(--)			(↓)	↑				↑							
EPTT		(↓)	↓↑	↓↑		↓	(↓)				↑				↑							
FT	↓	(↓)	↓↑	↓↑		↓		↓	↓	↓↑	↓		(--)		↑		(↑)				↓↑	
HT	↓↑	(↑)	(↓)	(↓)			(↑)															
Electrodermal																						
SCR	↑	↑	↑	↑		↑	↑				↓		↑	(--)			↑			↓		
nSRR	↑	↑	↑	↑		↑		↑	↓	↑	↑--		↑		↑	↑		↑		(↓)		(↑)
SCL	↑	↑	↑	↑	(↑)	↑	↓	↑	↓	↑	↓	(↑)	↑	↓	↑--	--	↑		↑	↓	(↑)	(↑)
Respiratory																						
RR	↑	↑	↑	↑		↑		↑	↑	↓↑	↓↑		↑	↓↑	↑	(↑)	(↓)	↑		(↓)	↓--	(↑)
Ti	(↓)	↓	↓	--		↓--					(↓)		(↓)	(↑)	↓		(↑)	(↓)		(↑)		
Te	(↓)	↓	↑	--		↓					(↓)			(↑)	↓		(↑)	(↓)		(↑)		(↓)
Pi	(↑)					(↑)									(↓)						(↑)	
Pe											(↓)											(↓)
Ti/Ttot			(↓)	--		(↑)						(↑)	(↓)									(↑)
Vt	↓↑	↓	↓	(↓)		↓↑		↓	↑	↓↑	↓		↓↑	↓↑	↓↑		(↓)	(↓)		(↑)	↑--	
Vi/Ti			(↓)	--											(↑)							(↓)
V(rhyth)			(↑)	--						(↑)		(↑)	(↑)		(–)							(↓)
V(vol)	(↑)	(↑)	(↑)	--		↑						(↓)	(↑)		(↓)					(↓)		
sighing		↓↑																		↓↑		
Ros	(↑)	(↑)	(↑)	--									(↑)	(--)	(↑)		(--)					
pCO_2		↓				↓					↑			↑				(↓)				
ANS activation components																						
α-adrenergic	↑	(↑)	↑	(--)	(↑)	↑		(↑)	(↑)	(↑)			↑--	(↓)	(↑)	--						
β-adrenergic	↑	(↑)	(↑)	(↑)	(↑)	↑				↓↑	↓		(↓)	(↓)	↓	↓↑			--			
cholinergic	↑	↑	↑	↑	(↑)	↑		↑	↓	↑	↓	(↑)	↑	↓	↑--	↑--	(↑)	(↑)	(↑)	↓	(↑)	(↑)
vagal	↓	↓	↑	--	(↓)	↓		--	↓	(↓)	↑--		↑	↓↑	↓	(↑)	(↑)		(--)			
respiratory	↑	↑	↑	--		↑		↑	↑	(↑)	↑	(↑)	↑	↓	↑	(↑)	↓	↑		↓	↓	(↑)
	↑pause	↓depth	↑exp			↑insp		↓depth	↑depth		↓depth	↓↑var	↑var				(↓) deptr	↓depth		↑depth	↑pause	↓exp

Note. *Modal responses were defined as the response direction reported by the majority of studies (unweighted), with at least three studies indicating the same response direction. Arrows indicate increased (↑), decreased (↓), or no change in activation from baseline (−), or both increases and decreases between studies (↓↑). Arrows in parentheses indicate tentative response direction, based on fewer than three studies. *Abbreviations*: pause – respiratory pause time; depth – respiratory depth; exp – respiratory expiration time; insp – respiratory inspiration time; var – respiratory variability. For abbreviations of other physiological measures, see Table 3.

Table 3 Abbreviations, Full Names, and Synonymous Expressions of Autonomic Measures Used in Studies on Emotion.

Abbreviation	Full name	Near-synonymous expression
Cardiovascular measures		
CO	cardiac output	cardiac output ∗ BSA (CI)
DBP	diastolic blood pressure	
EPA	ear pulse amplitude	
EPTT	ear pulse transit time	
FPA	finger pulse amplitude	
FPTT	finger pulse transit time	
FT	finger temperature	
HI	Heather index	
HR	heart rate	1/interbeat interval (IBI)
HRV	heart rate variability	
CVT	coefficient of temporal variability	
HF	high frequency spectral HRV (RSA)	
LF	low frequency spectral HRV	
LF/HF	low frequency/high frequency ratio	
MF	mid frequency spectral HRV	
MSD	mean difference between successive RR intervals	
MSSD	mean square of successive RR interval differences	
pNN50	percentage of successive normal sinus RR intervals > 50 ms	
RMSSD	root-mean-square of successive normal sinus RR interval differences	
RSA	respiratory sinus arrhythmia	
SDNN	standard deviation of the normal-to-normal intervals	
SDSD	standard deviation of successive differences	
VLF	very low frequency spectral HRV	
HT	forehead temperature	
LVET	left ventricular ejection time	
MAP	mean arterial pressure	
PEP	preejection period	
PWA	P-wave amplitude	
SBF	skin blood flow	
SBP	systolic blood pressure	
SV	stroke volume	stroke index ∗ BSA (SI)
TPR	total peripheral resistance	
TWA	T-wave amplitude	
Respiratory measures		
FRC	functional residual capacity	
I/E ratio	inspiratory/expiratory ratio	
HV	hyperventilation	
pCO_2	end-tidal carbon dioxide partial pressure	end-tidal fractional CO_2 concentration ($FETCO_2$)
Pe	post expiratory pause time	
Pi	post inspiratory pause time	
RC/Vt	percentage of rib cage contribution to Vt	
RD/Ttot	amount of respiratory work (depth divided by breath cycle duration)	
Ros	oscillatory resistance	
RR	respiration rate	1/total respiratory cycle duration (Ttot)
SaO_2	transcutaneous oxygen saturation	
Te	expiratory time	
Ti	inspiratory time	
Ti/Ttot	inspiratory duty cycle	
Ve	expiratory volume	
Ve/Te	expiratory flow rate or expiratory drive	
Vi	inspiratory volume	
Vi/Ti	inspiratory flow rate or inspiratory drive	
Vm	minute ventilation	
Vt	tidal volume	respiration depth (RD), typically uncalibrated ribcage measurements in arbitrary units
Vt/Ti	mean inspiratory flow rate	
VtV	tidal volume variability	
Electrodermal measures		
nSRR	nonspecific skin conductance response rate	
OPD	Ohmic Perturbation Duration index	
SCL	skin conductance level	
SCR	skin conductance response (amplitude, evoked)	
SYDER	SYDER skin potential forms	
SRA	skin conductance response amplitude (spontaneous)	

increases instead of a decrease or no change [28, 59, 129]. Because emotional responses to anger expressions that signal threat have been related to fear, this response pattern may be taken as suggestive of a fear response rather than an anger response (see discussion of fear responses associated with decreased HR, below). Similarly, film viewing for anger elicitation differs in resulting in decreased HR in the presence of decreased HRV (MSD [21]), pointing to sympathetic–parasympathetic cardiac deactivation that may rather indicate passive sensory intake (Obrist, 1981; Schneiderman & McCabe, 1989). Along these lines, Stemmler and colleagues (2007) demonstrated that approach-oriented anger was characterized by unchanged HR, while withdrawal-oriented anger showed decreased HR [110]. This finding may point to the fact that motivational direction influences the heart rate response in anger.

A second deviation from the modal response pattern in anger is evident in the absence of α-adrenergic vasoconstrictive effects in several studies: directed facial action (DFA) of anger is characterized by increased, instead of decreased, FT [32, 74, 75] (although decreased FT has also been reported for anger in DFA [73]), an effect that reflects β-adrenergically mediated vasodilation in contrast to α-adrenergically mediated vasoconstriction (Cohen & Coffman, 1981; Rowell, 1986). TPR decreased in association with increased HR, LVET, SV, CO, HI, SBP, DBP, and MAP and shortened PEP in a film study of anger [81]. Similarly, a response pattern labeled "anger in" (i.e., anger directed toward the self) is characterized by increased HR, SV, and CO, unchanged SBP and DBP, and decreased TPR [2, 40]. Increased HR, SBP, DBP, SV, CO, and forearm blood flow, but decreased levels of TPR have also been reported under conditions of experimenter harassment in accompaniment of a friend [71]. Finally, increased HR and SBP, but decreased DBP and MAP was found in the context of emotional step walking [105]. These findings suggest that various subforms of anger may exist, which are differentiated by motivational direction that appears to influence the heart rate and α-adrenergic response.

Anxiety Using predominantly experimental paradigms that incorporate an anticipatory component (e.g., threat of shock [12, 13, 17, 20, 127]; speech preparation [82, 118]), anxiety has been almost unanimously characterized by sympathetic activation and vagal deactivation, a pattern of reciprocal inhibition, together with faster and shallower breathing. Apparent overlaps with the above-reviewed anger response on certain response variables will have to be addressed in future research that will need to fill gaps of measures that are either predominantly assessed in anger research (e.g., α- and β-adrenergically affected measures of sympathetic functioning, such as PEP, LVET, MAP, and TPR) or in anxiety research (e.g., respiratory measures of sighing or carbon-dioxide blood levels).

In particular, reports on anxiety indicate increased HR [2, 31, 82, 97, 118, 121], decreased HRV (spectral RSA [82]; peak-valley RSA [84]) as well as increased LF and LF/HF [82], increased SBP [2, 118], increased DBP [84, 118] or unchanged DBP and TPR [2], unchanged SV [2, 84] and increased CO [2], decreased FPA [12, 13, 118] as well as decreased FPTT and EPTT [118], decreased FT [91, 97], and increased HT [91]. Reports include moreover increased electrodermal activity (increased SCR and nSRR [12] and increased SCL [12, 20, 82, 93]). Respiratory variables indicate increased RR due to decreased Ti and Te [12, 30, 84, 121], as well as decreased Vt [12, 121], increased sigh frequency and Vt variability [12] (however, higher sigh frequency during relief than tension has also

been found [127]), increased Ros [93], decreased end-tidal pCO_2 [30, 121], and increased oxygen consumption [30].

A striking exception to this otherwise classic pattern of reciprocal sympathetic activation and parasympathetic deactivation for anxiety constitutes a study of picture viewing (e.g., pictures of a snake, shark, tornado, knife, or attack [94]): this study reports HR deceleration, accompanied by increased HRV (peak-valley RSA), and a trend of increased Ttot associated with increased Te and decreased Ti, decreased Vm, and an unspecific small increase in Ros. Thus, this study suggests a pattern of reciprocal parasympathetic activation and decreased respiratory activity for anxiety. Other exceptions that do not fully support a pattern of reciprocal sympathetic activation for anxiety include results from a threat-of-shock context, where unchanged HR [13] or decreased HR and increased SCR [17] has been reported. HR deceleration, accompanied by increased PEP and LVET, has also been found in the context of music-induced agitation [84]. All these response patterns point to response fractionation across organ systems (Lacey, 1967).

Disgust Disgust-related autonomic responding falls into two partially overlapping patterns: (a) disgust elicited in relation to contamination and pollution (e.g., pictures of dirty toilets, cockroaches, maggots on food, foul smells, facial expressions of expelling food), characterized by sympathetic–parasympathetic co-activation and faster breathing, particularly decreased inspiration (cf. physiological response associated with vomiting; Sherwood, 2008); (b) disgust elicited in relation to mutilation, injury, and blood (e.g., injections, mutilation scenes, bloody injuries), characterized by a pattern of sympathetic cardiac deactivation, increased electrodermal activity, unchanged vagal activation, and faster breathing. Increased HRV sets contamination-related disgust apart from most other negative emotions, which typically show decreased HRV. Similarly, decreased CO distinguishes disgust in general from the other negative emotions, which show increased CO, as is typical for mobilization for action (Obrist, 1981).

Specifically, contamination-related disgust is associated with HR acceleration [3, 14, 49, 73, 128] or no change from baseline [32, 74, 75, 99]. HR acceleration has also been reported in the context of personalized recall [73, 89] or films [63] where disgust-type remained unspecified. This response pattern is furthermore characterized by increased HRV (SDNN [63], RMSSD [99], peak-valley RSA [94]), increased TPR, and decreased SV [89, 99], suggesting sympathetic–parasympathetic coactivation. As a notable exception, unchanged or even decreased skin conductance has been reported in response to contamination pictures [22] and no change in nSRR has been reported in response to film clips depicting contamination-related material [66].

Mutilation-related disgust, on the other hand, was characterized by HR deceleration [9, 18, 21, 23, 44, 46, 62, 85, 99, 108, 133] or a depressed phasic HR response [70]. Palomba, Sarlo, Angrilli, Mini, and Stegagno (2000) note that HR reduction occurred between the first and the last interval of a 132-s film, indicating a slow late deceleration [85]. Similarly, in response to picture viewing, Winton, Putnam, and Krauss (1984) describe a triphasic response pattern of HR change that was characterized by an early deceleration, a brief and dampened acceleration, followed by an early onset of a second deceleration [133]. This response pattern is furthermore characterized by no change in HRV (RMSSD [99]; peak-valley RSA [85]; Porges' RSA [9]; however, increased HRV (spectral RSA) and

decreased LF/HF has also been reported [108]). Increased TWA [85] and no change in SV and TPR [99] have also been found for mutilation-related disgust, suggesting decreased cardiac and increased electrodermal sympathetic control together with unchanged vagal influence (increased SCR for mutilation- versus contamination-related disgust has also been reported [16, 22]). Still, one study [22] reported non-differential HR deceleration for both contamination and mutilation pictures that was largest compared to all other affective categories.

Both response patterns, i.e., mutilation- and contamination-related disgust, were non-differentially accompanied by increased SBP, DBP, and MAP [21, 69, 89] or no change in blood pressure [99], decreased PEP, LVET, CO [99], or no effect on CO and FT [89], increased FT [32, 74, 75], decreased FT [24, 44, 46, 73], decreased FPA [44, 46, 69, 75], increased FPTT [46, 69], and decreased FPTT [75], no change in EPTT [46], and decreased facial blood flow and velocity [108]. Responses in these variables do not seem to fall into a coherent pattern.

Across paradigms (e.g., picture viewing, film clips, DFA, and personalized recall), disgust is consistently reported to be nondifferentially associated with increased electrodermal activity, as indicated by increased SCR [18, 60, 62, 70, 133], increased nSRR [60, 65, 108], and increased SCL [21, 23, 26, 32, 44, 46, 49, 69, 74, 75, 85, 99, 108, 115, 126]. Electrodermal activity is furthermore characterized by long-duration SCR [3] in response to disgust-eliciting odorants, whereas picture viewing of disgust-expressing faces has been reported to elicit relatively short OPD, small SCR, positive skin potentials of rapid increase and slow decrease [24] or a delayed SCR of medium response size and slow rise time [132].

There is a general effect of increased RR in disgust [15, 24, 34, 46, 69, 75, 85], although increased respiratory duration [94] or no change [108] have also been reported. Notably, contamination-related disgust has been characterized by decreased Ti and increased or no change in Te [14, 15, 94], that may contribute to decreased Ti/Ttot and Vt/Ti [15], decreased respiratory volume (e.g., decreased Vt, Vm [14, vomiting clip 15, 24, 75, 94]), and increased Ros [94], as well as larger variability in Te, Vt, Vm, Vt/Ti [vomiting clip 15]. Other than decreased Vt [69] for mutilation-related disgust, generally no change in respiratory timing [9, torture clip 15] or volume parameters [46] is reported (see also [34]). In summary, the distinction between contamination versus injury disgust appears to be important in determining the specific type of disgust response and will need to be more systematically investigated in future research.

Embarrassment Inducing embarrassment by experimenter humiliation, watching a video of oneself singing, or imagery, studies consistently indicate broad sympathetic activation and vagal withdrawal, a pattern of reciprocal inhibition. Whereas this response pattern largely overlaps with those of anger and anxiety reviewed above, the relatively small number of studies as well as the limited number of response variables assessed highlights the importance for future research to test specific physiological differences between negative emotions, such as facial blushing in embarrassment.

Studies inducing embarrassment in particular report increased HR [4, 52, 54, 56], accompanied by decreased PEP, no effect on CO, and increased TPR [54], increased SBP and DBP [52], decreased HRV (peak-valley RSA), and increased SCL [56]. Harris (2001)

reports that HR rose significantly during the first minute of watching an embarrassing film of oneself singing, but returned to baseline levels during the second minute, a pattern that replicated in a second study [52]. As the empirical basis for the physiological response pattern of embarrassment is scant, much remains to be done in future research.

Fear Laboratory fear inductions typically use presentation of threatening pictures, film clips, or music, standardized imagery or personalized recall, and real-life manipulations (e.g., imminent threat of electric short circuit). One of the earliest attempts to induce fear in the laboratory, used a sudden backward-tilting chair [11]. Due to the nature of the manipulation, it is, however, not clear whether in fact fear, or rather surprise, was induced. Moreover, because confounds caused by the change in body posture complicate interpretation of results, this study is not considered here.

Overall, studies on fear point to broad sympathetic activation, including cardiac acceleration, increased myocardial contractility, vasoconstriction, and increased electrodermal activity. In distinction to the physiological response to anger, peripheral resistance typically decreased in fear, whereas it increased in anger. This response is accompanied by decreased cardiac vagal influence and increased respiratory activity, particularly faster breathing based on decreased expiratory time, resulting in decreased carbon dioxide blood levels.

Various of the studies investigating fear report increased HR [5, 8] or increased electrodermal activity in single measures (increased SCR [24]; increased nSRR [65]; increased SCL [132]) or in co-assessment (nSRR [35, 114]; SCL [48, 74, 79, 80, 114]; although increased HR and unchanged SCL [124] and unchanged nSRR [66] have also been reported), indicating a general arousal response.

More complete patterned responses are derived from studies that assessed combinations of cardiovascular and/or cardiorespiratory parameters. A number of studies report increased HR together with indicators of increased vasoconstriction: decreased FT [32, 64, 73, 89, 107, 109] (see, however, [74] for a report of increased FT); decreased FPA [67, 75, 109, 110, 111]; decreased FPTT [75, 111]; and decreased EPTT [64] (see, however, [67] for a report of increased FPTT and EPTT). Increased HR and increased blood pressure have also been variously reported: increased SBP and DBP [7, 64, 81, 87, 89, 96, 104, 107, 111], as well as increased MAP [21, 67, 72, imagery 105, 130]; some have reported unchanged DBP [exercise 105, 106, 119] and decreased MAP [exercise 105]. Reports on vascular resistance indicate either increased TPR [81, 89] or, more often, decreased TPR [87, 104, 106, 111]. Furthermore, HR increase co-occurs with increased myocardial contractility: increased ejection speed [111], shortened PEP [64, 81, 87, 106, 110, 111], decreased [106, 110, 111] or unchanged LVET [81], and increased HI [110, 111] (however, see [81] for a report of decreased HI). These are associated with consequent changes in cardiac pump function: increased [7, 104] or decreased SV [64, 81, 89, 106, 110, 111], and increased [104, 106, 111], unchanged [89], or decreased CO [81] have been reported. Increased sympathetic cardiac control is furthermore indicated by increased PWA and decreased TWA [85, 110, real-life 111]. Vagal withdrawal is evidenced in decreased HRV (MSD [21]; MSSD [111]; RMSSD [42, 43, 87, 110]; peak-valley RSA [90]; spectral RSA [90, 126]), although some report unchanged HRV (peak-valley RSA [67, 85]; spectral RSA [64]) and unchanged LF [126].

Further studies report HR increases together with increased respiratory activity, including measures of breathing rhythm: increased RR [7, 34, 64, 67, 75, 80, 85, 86, 90, 111, 122], and either both decreased Ti and Te [14, 120, 122], or predominantly decreased Te and unchanged Ti, as also indicated by increased Ti/Ttot and I/E-ratio [33, 34, 64], and increased Pi [14]. Volumetric measures moreover indicate increased respiratory volume [34, 75] or decreased respiratory volume [14, 67, 120, 122], and increased Vm [64]. Gas exchange analysis indicates decreased pCO_2 [64, 120, 122]. Furthermore, increased variability of respiratory parameters has been noted, such as increased variability of respiratory amplitude [90] or increased variability in pCO_2 and Vi/Ti [121].

The already above-mentioned increase in electrodermal activity was also found in numerous of these multi-measure studies (increased SCR [7, 111]; increased nSRR [87, 111, 119]; increased SCL [21, 42, 43, 64, 80, 85, 107, 111, 126, 130]).

Only a few studies report HR deceleration in the context of laboratory fear elicitation: decreased HR along with signs of increased vasoconstriction (decreased FPA and EPTT, unchanged FPTT) has been found in response to a film clip eliciting fear of falling [39]; decreased HR and unchanged HRV (SDNN) has been reported in children watching a film clip that portrayed Snow White running through a dark haunted forest [113]; decreased HR and increased SCR was reported in response to picture presentation of snakes and spiders [27] or other threatening material (e.g., angry face, aimed gun, attack [10, 22]); decreased HR, decreased SCL, and increased HT has been found in a real-life induction context (radio play, announcement of uncontrollable event, and sudden outage of light [109]); decreased SCL has been similarly found for fear induced by music excerpts [67]. It is possible that these latter fear paradigms elicited a stronger degree of self-involvement, leading to higher imminence of threat (Bradley & Lang, 2000; Craske, 1999; Fanselow, 1994; Lang et al., 1997), such that participants were further along the "fear continuum," characterized by immobilization rather than an active coping response that leads to sympathetic inhibition (see also the above discussion of outliers for anger and anxiety). However, such findings will need to be contrasted with such intense fear responses as found, for example, in phobias, which constitute a good model to study the type of fear with high immediate threat characteristics (e.g., Wilhelm & Roth, 1998).

Sadness Inspecting the activation components reported for sadness reveals a heterogeneous pattern of sympathetic–parasympathetic coactivation. Only a few studies considered mediating variables, such as cry-status [45, 102, 103]. These studies associate uncoupled sympathetic activation with crying sadness, whereas sympathetic–parasympathetic withdrawal appears to be characteristic of non-crying sadness.

Parsing reports of physiological response patterns of sadness that were not analyzed according to cry-status suggests two broad classes of physiological activity in sadness—an activating response and a deactivating response. The activating sadness response, which partially overlaps with the physiological response of crying sadness, is characterized by increased cardiovascular sympathetic control and changed respiratory activity, predominantly reported in studies using DFA, personalized recall, and some studies using film material. On the other hand, the deactivating sadness response, which partially overlaps with the physiological response of non-crying sadness, is characterized by sympathetic withdrawal, reported in the majority of studies using film mate-

rial, as well as music excerpts, and standardized imagery. A distinct characteristic of deactivating/non-crying sadness to all other negative emotions is the decrease in electrodermal activity. In contrast, the activating/crying sadness response largely overlaps with that of, for example, anxiety—a point that will be returned to below.

Specifically, for participants who cried in response to a sadness-inducing film clip, studies unanimously report increased HR, associated with increased SCL, decreased FPA, FT, smaller increases in RR, and non-differentially increased RD [45], increased nSRR and unchanged SCL [101], or increased RR, unchanged HRV (spectral RSA) and Vt [103]. In contrast, sad participants who did not cry while watching the film clip, exhibited decreased HR, associated with decreased electrodermal activity (decreased SCL and smaller nSRR [45, 101]), increased respiratory activity (increased RR and RD [45, 103]), increased [103] or decreased respiratory depth [45], decreased HRV (spectral RSA [103]), and decreased FPA and FT [45].

With respect to the activating response in sadness, which partially overlaps with the physiological response of crying sadness, DFA has been found to consistently prompt increased HR [14, 32, 73, 74, 75]. In some studies, shortened FPTT and increased FPA [75], increased SCL [74], increased [73], unchanged [74], or decreased FT [32], and increased RR and respiratory depth [75] or decreased RR, Ti, Te, and Vt, and increased Pi and FRC [14] is reported. Similarly, sadness elicited by personalized recall is characterized by increased HR associated with increased [32, 98, 115] or unchanged [77] SCL as well as increased SBP, DBP, and TPR [51, 83, 89, 106], unchanged [51, 89] or decreased SV [83, 106], increased [51] or unchanged CO [83, 89, 106], and increased [83] or decreased PEP and LVET [106]. FT has been reported to remain unchanged [89] or to decrease [98]. For HRV, decreases (MSD, SDNN [90]; spectral RSA [77]), no change (spectral RSA [98]; a correlation of increased HRV with increased sadness intensity is, however, also reported), or increases (peak-valley RSA [90]) were found. Respiration was characterized by increased respiration period and increased variability in respiration period [90]. Only small increases in HR and SBP and unchanged DBP have also been reported [105]. Allen, de L. Horne, and Trinder (1996), examining social rejection and achievement failure, characterize the emotion they investigated as high-arousal sadness and report increased HR [4]. Some studies using films for sadness induction report increased HR [63, 68, 76], increased electrodermal activity (nSRR [65, 100]; SCL [68, 93, 117, 126]; although no effect on nSRR has also been reported [66]), and increased RR [68, 100], associated with decreased FPA, FPTT, FT [68] or unchanged HR and FPTT [100], decreased HRV (spectral RSA) and unchanged LF [126] or increased HRV (SDNN [63]), unchanged SBP, DBP [76], and increased Ros [93].

The activating response contrasts with a deactivating sadness response, which partially overlaps with the physiological response to non-crying sadness. This response pattern is found in the large majority of studies using film clips for sadness induction, which report a pattern of decreased cardiac activation and decreased electrodermal activity: decreased HR [6, 18, 21, 31, 47, 49, 64, 86, 113, 114, 116] (although see [39] for report of unchanged HR), longer PEP [64, 78], increased HRV (MSD [21]; spectral RSA [78]) or unchanged HRV (SDNN [113]; RMSSD [49]), unchanged [64] or decreased DBP and MAP [21], increased EPTT and FPTT, associated with decreased EPA, FPA, and FT [39, 64], decreased electrodermal activity (SCR [18]; SCL [21, 47, 78, 112, 116]; however,

see [114] for increased SCL and unchanged nSRR, and [64] for increased nSRR). Some studies report decreased respiratory activity [47], as indicated by decreased RR and increased pCO_2 [64], while others report increased RR [86, 116]. Averill (1969) also reported decreased HR and SCL, however, together with increased SBP, DBP, FPA, unchanged FT, increased nSRR, and unchanged RR and respiratory irregularity, as elicited by a film clip on the aftermath of the assassination of John F. Kennedy [6], showing the funeral and burial of the US President—material that might have elicited nostalgia or mixed emotions of both sadness and anger.

Such cardiovascular deactivation has also been found in an exercise paradigm for emotion induction [exercise 105], in which sadness was the only emotion that evidenced decreases in HR, SBP, DBP, and MAP compared to a neutral comparison condition. Music-induced sadness is similarly reported to be characterized by decreased HR associated with decreased RR and increased Te [33], decreased RR and RD [67], unchanged [61] or increased RR, associated with decreased Te, Ti, and Pe [84].

Sadness elicited in the context of standardized imagery is similarly reported to be characterized by decreased HR [41, 122] or only small HR increases [124], unchanged SCL [41, 124], increased Ti and Te, resulting in decreased RR, and increased pCO_2 [120, 122]. Another study [30] also reports of decreased ventilation, decreased oxygen consumption, and increased pCO_2 in the context of hypnosis, as well as decreased [120] or unchanged Vt [122]. Increased HR and decreased nSRR has also been reported [35]. Similarly, in an emotion self-generation task, unchanged HR and decreased SCL for sadness has been reported [55].

Picture viewing for sadness induction has been reported to lead to increased HR and Ros, unchanged HRV (peak-valley RSA) and ventilation (depressing picture content, such as hospital patients, scenes of catastrophe, soldiers in action, or dead animals [92]), decreased HR, Ti, Vt, increased Ttot, Te, HRV (peak-valley RSA), unchanged Ros and SCR (depressing picture content, such as cemetery, plane crash, war victim, or a duck in oil [94]), or moderately increased RR, decreased FT, smallest SCR, and positive SP (pictures of sad facial expressions [24]).

Contrasting contents related to the activating and deactivating sadness responses suggests a differentiation according to imminence of loss, with the activating pattern occurring predominantly in response to film clips that depict scenes related to impending loss, such as individuals coping with cancer or Alzheimer's, a husband waiting for the result of his wife's operation, or a man talking to his dying sister (cf. helplessness; Seligman, 1975). On the other hand, the deactivating pattern occurs predominantly in response to film clips that depict scenes related to a loss that *has* occurred, such as a mother at her daughter's funeral, a young boy crying over his father's death, or the death of Bambi's mother. It may be that such distinctions as anticipatory sadness (i.e., worry or anticipation of loss) as contrasted to acute sadness in the experience of loss or grieving in the aftermath of a loss play a role in addition to cry-status in differentiating physiological responses in sadness (Barr-Zisowitz, 2000; Kreibig, 2004). To allow a clearer picture of the type of autonomic activation associated with sadness, it will be important for future research to consider cry-status in analyzing physiological responses. Moreover, care should be taken to distinguish between anticipatory and acute sadness.

The Positive Emotions

Affection Love, tenderness, or sympathy evoked by film clips [15, 31] or personalized recall [115], have been reported to be associated with decreased HR (similar to sadness [31]), an unspecific increase in SCL [115], and increased Ti/Ttot, increased variation in Te, and decreased variation in Vt, Vm, and Vt/Ti [15]. Because of the few studies that have investigated physiological responding in affection-related emotions, no conclusive statement on the type of response pattern can be made.

Amusement Laboratory elicitation of amusement has almost exclusively employed film clips; only two studies used alternative paradigms (picture viewing [62] or personalized recall [37]; see also [38]). Although all film clips depicted comedic material, several response components emerge. Overall, response variables point to increased cardiac vagal control, vascular α-adrenergic, respiratory, and electrodermal activity, together with sympathetic cardiac β-adrenergic deactivation in amusement.

HR is the most variable response component, with reports of deceleration [18, 21, 26, 58, 62, 112], no change [47, 50, 53, 57], or acceleration [6, 37, 63, 116]. More consistently, increased HRV (SDNN [63]; MSD [21]; spectral RSA [26]), unchanged LF/HF [26], and increased PEP and decreased CO [53] are reported. Blood pressure remains unchanged (SBP [6, 53]; DBP [6]; MAP [50]) or increases (SBP, DBP, MAP [21]). Increased vasoconstriction is indicated by decreased FPA, FPTT, EPTT, and FT [47, 50]; increased TPR [53], and decreased FPA and unchanged FT [6] have also been reported. Respiratory activity is increased, as evidenced in increased RR [6, 47, 57, 93, 116], increased RD [47], increased respiratory irregularity [6], increased Ros [93], decreased Ti, Vt, Ti/Ttot, and increased Pi and variability of Te, Vt, Vm, and Vt/Ti [15]. Increased electrodermal activity is shown in increased SCR [18], increased nSRR [6, 57, 65], and increased SCL [37, 47, 57, 62, 93, 116, 117]; still, some have reported unchanged SRA [50] and nSRR [66] or even decreased electrodermal activity (SCL and nSRR [6, 58, 112]).

Contentment Studies on psychophysiological effects of contentment or pleasure have particularly relied on film clips displaying nature scenes [21, 85, 94], standardized imagery (e.g., wood fire, book reading, soft music [83, 120, 122, 128]) or personalized recall [25, 105]. Taken together, decreased cardiovascular, respiratory, and electrodermal activation is suggestive of decreased α-, β-adrenergically, and cholinergically mediated sympathetic activation and mild cardiac vagal activation. Compared to the physiological response to amusement, the physiological response to contentment appears to have a stronger sympathetically deactivating component, whereas both share cardiac vagal activation. Further studies are, however, needed to clarify the exact nature of autonomic and respiratory activity in contentment.

Studies on the physiological response of contentment indicate HR deceleration [21, 55, 84, 85, 94, 105, 122] or unchanged HR [25, 79], increased TWA, unchanged HRV (peak-valley RSA), and increased RR [85], or decreased HRV (MSD [21]), decreased SBP, DBP, MAP [21, 105], and decreased SCL [21, 55, 85] or unchanged SCL [79]. Decreased RR has been reported together with increased HRV (peak-valley RSA [94]), increased Ti, Te [94, 120] or unchanged Ti and Te [122], decreased Vt [94, 122] or increased Vt [120], and

increased pCO_2 [120, 122] as well as unchanged Ros, SCR, and Vm [94]. Unchanged I/E ratio and moderately increased respiratory work, depth, and rate has also been reported [34]. Using music excerpts for emotion induction [84], increased LVET and unchanged FPA, together with increased RR, and decreased HRV (peak-valley RSA), Ti, Te, and Pi has been found. Moderate increases in HR, SBP, DBP, PEP, TPR, unchanged CO, and decreased SV has been reported for relaxation imagery [83]. As this overview shows, the physiological response pattern of contentment is similar to a relaxation response. Still, inconsistencies of the response pattern noted by various studies will have to be addressed in future research.

Happiness Happiness has been induced with various emotion elicitation paradigms, including DFA [14, 73, 74, 75], personalized recall [77, 89, 90, 105, 115], standardized imagery [41], film clips [100, 113, 126], music [33, 61, 67, 84], or pictures [59]. The autonomic response pattern of happiness is characterized by increased cardiac activity due to vagal withdrawal, vasodilation, increased electrodermal activity, and increased respiratory activity. This response pattern points to a differentiated sympathetic activation state of decreased α- and β-adrenergically mediated influences, while at the same time cholinergically-mediated effects are increased. Happiness shares with various negative emotions a central cardiac activation component due to vagal withdrawal, whereas it is distinguished from these by peripheral vasodilation.

In particular, the physiological response to happiness includes increased HR [14, 41, 55, 59, 61, 74, 75, 77, 79, 89, 90, 92, 105, 113] or unchanged HR [33, 84, recall visualizing 131] (although decreased HR has been reported in [67]), unchanged HRV (SDNN [113]; peak-valley RSA [92]) or decreased HRV (spectral RSA [59, 77, 90, 126]; peak-valley [67, 84, 90]), and unchanged LF [126]. Furthermore, reports indicate increased blood pressure (increased SBP, DBP, MAP [67, imagery 105]; increased SBP, DBP [61, 89, recall visualizing 131]; increased SBP, decreased DBP, MAP [exercise 105]; unchanged SBP and DBP [84]). Increased PEP and unchanged LVET and SV has been furthermore found [84]. Increased TPR, decreased SV, and unchanged CO have also been reported [89]. Vasodilation is moreover reported, including increased FT [74, 75, 109] (however, unchanged or decreased FT have been reported in [89] and [67], respectively), increased [109], unchanged [75], or decreased FPA [67, 84], and lesser shortening [75] or increase of FPTT and EPTT [67, 84]. Increased electrodermal activity is shown in increased SCL [74, 109, 115, 126] and increased nSRR [61, 100]. Some studies also reported unchanged SCL [41, 55, 75, 77] or decreased SCL [67]. Increased respiratory activity is evidenced in increased RR [14, 33, 61, 67, 75, 84, 90, 100] or unchanged RR [59], decreased Ti and Te [14, 84], decreased Pe [84], increased Pi and FRC [14], or unchanged Ti, decreased Te, and unchanged respiratory variability [33], increased depth [75] or decreased depth [14, 67], decreased respiratory variability of period and amplitude [90], increased Vt/Ti, unchanged FRC, and increased Ros [92].

A few exceptions are of note that occurred in happiness induction with visual material, such as pictures [28, 94] or film clips [49, 131]: instead of the typical increase in HR, these studies report decreased or unchanged HR. Decreased HR and increased SCL have been found in response to pictures of happy faces [28]. Decreased cardiac activity (decreased HR and slightly increased HRV, i.e., peak-valley RSA) and decreased respi-

ratory activity (decreased RR, Vt, Ros, and increased Ti and Te) have been reported in [94] for happiness elicited with pictures from the International Affective Picture System (e.g., family, sky divers, happy teens, roller coaster, water slide; Lang, Bradley, & Cuthbert, 2005). Decreased HR and SCL have been found in children in response to a happy scene in the film Bambi [112]. Decreased HR has also been found in response to a film depicting a figure skater winning an Olympic gold medal [49]. Decreased cardiovascular activity as expressed in decreased HR and unchanged SBP and DBP have been reported in response to a film clip depicting a joyful mother–daughter interaction [131]. This variance may point to the fact that a relatively wide range of positive emotions is commonly subsumed under the umbrella term 'happiness.' For certain of the above cases, a label such as admiration, contentment, excitement, joy, or pride may be a more appropriate descriptor. Certain emotional stimuli may also derive special meaning from the context in which they occur, such as pictures of smiling faces in the event of winning or losing a game (Vrticka, Andersson, Sander, & Vuilleumier, 2009).

Joy Laboratory joy elicitation has particularly relied on standardized imagery [35, 124, 128, 130, 134] and personalized recall [83, 106] for emotion induction. Some studies have also used picture viewing (e.g., faces [129]), real-life manipulations (e.g., expression of appreciation and reward by experimenter [119]), or the Velten method [19]. Taken together, an autonomic response pattern of increased cardiac vagal control, decreased α-adrenergic, increased β-adrenergic, and increased cholinergically mediated sympathetic influence as well as increased respiratory activity may be concluded, however, awaiting confirmation by further investigations. Whereas all other positive emotions are characterized by decreased β-adrenergic sympathetic influence, joy appears to be characterized by increased β-adrenergic sympathetic activation, an autonomic response component that has been associated with increased motivational engagement (Wright, 1996), co-occurring with increased vagal activation in the response pattern of joy.

Specifically, the physiological response pattern of joy was generally characterized by increased HR, accompanied by reports of either unchanged SCL [124, 128, 130] or increased SCL [129] as well as increased nSRR [35, 119]. The physiological response pattern of joy was further characterized by increased HRV (SDNN [63]), decreased PEP and LVET, and unchanged CO and TPR [106], or increased PEP and TPR, decreased SV, and unchanged CO [83], as well as increased SBP, DBP, and MAP [83, 134], or increased SBP and unchanged DBP or MAP [106, 119, 130]. Effects on respiratory activity show increased RR [119]. Using the Velten method for joy induction [19], no change in HR, SBP, DBP, and MAP has been reported. For an emotion amalgam of joy and pride elicited in the context of a computer game [125], mildly increased SCR, decreased HR in anticipation of the event, and increased HR after onset of the event, an initial deceleration, followed by an increase, and a second decrease in FPTT, as well as faster rise in FT at low difficulty levels, as contrasted to stronger decrease in FT at high difficulty levels has been reported.

Pleasure, anticipatory The emotion complex "anticipatory pleasure" here considers both appetite [18] and sexual arousal [1, 23, 35, 70, 94, 120, 122, 133]. Physiological responses of anticipatory pleasure appear to be grouped according to type of task, indicating physiological deactivation when emotionally evocative material is visually presented

(e.g., picture viewing [18, 70, 94] or film clips [1, 23]) and physiological activation when emotionally evocative material is imagined (e.g., standardized imagery [35, 120, 122]). Overall, these studies suggest that visual material that relates to anticipatory pleasure elicits increased cardiac vagal control, increased electrodermal activity, and respiratory deactivation. On the other hand, imagined material that relates to anticipatory pleasure elicits increased cardiac activation (either via increased sympathetic or decreased parasympathetic influence) and increased respiratory activity.

Looking at material that relates to anticipatory pleasure is associated with decreased HR [10, 18, 22, 23, 94] and increased SCR [10, 22, 70] (although small or unchanged SCR have also been reported [18, 94]) and increased SCL [23] together with increased FT [18] and increased HRV (peak-valley RSA), Ti, Te, decreased RR, Vt, Vm, and unchanged Ros [94]. Imagining material that relates to anticipatory pleasure, in contrast, is associated with increased HR [35, 122], increased nSRR [35], and increased RR together with decreased pCO_2, Ti, Te, and Vt [120, 122]. As an exception, increased HR and increased SCR has been reported in the context of presenting erotic pictures [133] and increased HR, HRV, SBP, DBP, SCR, SCL, decreased FT, and unchanged HT, RR, and respiratory variability has been reported in the context of presenting an erotic film clip [1]—notably, both studies included only male participants.

Pride Laboratory induction of pride has used film clips [49], personalized recall [115], or real-life manipulations of experimenter praise [54]. These studies report decreased HR and unchanged HRV (RMSSD [49]), increased SCL [49, 115], and a small increase in HR together with unchanged PEP, CO, and TPR [54]. These results may suggest an activation pattern of decreased β-adrenergic cardiovascular activity, increased cholinergic sympathetic influence, and unchanged vagal control in pride. However, due to the small number of studies that investigated pride, further research is strongly needed.

Relief Conceptualizing the absence of danger in a threat-of-shock paradigm as relief (e.g., Vlemincx et al., 2009), such studies characterize the physiological response to relief by decreased sympathetic vascular and electrodermal influence and decreased respiratory activity. As is true for the largest part of physiological responding in positive emotion, only further research will allow firm conclusions. Similar to sadness, the physiological response to relief shows decreased electrodermal and respiratory activation, which is a distinguishing characteristic of relief to all other positive emotions.

In particular, the physiological response to relief is marked by moderate cardiovascular changes (mild HR acceleration [17]; or unchanged HR [13]; and increased FPA [12, 13]). There is moreover a decrease in respiratory activity (decreased RR, associated with increased Ti, Te, increased Vt, and decreased Vt variability as well as decreased sigh frequency [12]; or increased Vi including sighs, unchanged Vi excluding sighs, and increased sigh frequency [127]). Notably, increased sigh frequency has also been reported for conditions of relief in animal experiments (Soltysik & Jelen, 2005). Finally, decreased electrodermal reactivity is typically reported (decreased SCR reactivity [12, 17]; decreased nSRR [12]; decreased SCL [12, 20]).

Emotions without Clear Valence Connotation

Surprise Surprise has been reported to be associated with short-duration SCR [3] of medium response size and characterized by rapid increase and rapid return [24], increased SCL [74], increased HR [14, 32, 74], decreased [32] or increased FT [24, 74], unchanged respiratory timing and volume parameters [14], or decreased RR and increased respiratory depth [34]. Feleky (1916, p. 230) pointed out a "decided inspiratory pause" of the characteristic breathing curve of wonder, that—albeit its overall similarity to that of fear—makes it distinct. No uniform response pattern can be derived due to the limited number of studies investigating surprise. Including the literature on unexpected stimulus presentation (Epstein, Boudreau, & Kling, 1975; Niepel, 2001; Qiyuan, Richer, Wagoner, & Beatty, 1985) and the orienting reflex (Siddle & Heron, 1976; Siddle, Stephenson, & Spinks, 1983; Siddle, 1985, 1991; Sokolov, 1990) may prove more conclusive.

Suspense Suspense, induced in the context of film clips, has been found to be associated with decreased HR, increased nSRR and SCL [57, 58] as well as increased RR, decreased Te, Pe, Vt/Ti, and variability of Te, and increased Ti/Ttot [15]. While the physiological response to suspense clearly differs from that to surprise by cardiorespiratory measures, further research will have to address whether suspense constitutes a separate emotion class or whether it may be subsumed under anxiety (see Nomikos, Opton, Averill, & Lazarus, 1968).

Discussion

ANS activity is viewed as a major component of the emotion response in many recent theories of emotion (see Table 1). Different levels, on which an organizing principle of autonomic responding in emotion might be located, were identified in the introduction and the complementary nature of these approaches was pointed out. The empirical review compiled a large database that can be drawn on to evaluate such statements. What is the empirical evidence for positions of various degrees of ANS specificity in emotion?

Autonomic Responding in Emotion

With the chosen approach, both specificity and similarity of autonomic activity in emotion was shown. Table 2 presents a summary of the modal response pattern found for each emotion. The large scope of this review necessitated a considerable degree of abstraction; thus, only direction, but not magnitude of response, was coded (cf. Folkow, 2000). This choice was made because quantification of response magnitude ultimately depends on the type of baseline or comparison condition used, operationalization of which varied greatly across studies (see Kreibig, Wilhelm, Gross, & Roth, 2005; Levenson, 1988, for issues of physiological response quantification in emotion in relation to baseline choice). Also, a number of assumptions had to be made in order to code and classify the large variety of studies. Moreover, numerous conclusions remain tentative at best, as the number of studies reporting effects on certain parameters remains limited. In that way, Table 2 may serve as an instructive guide for future research of specific emotion contrasts and autonomic parameters that demand further empirical study.

Summary of empirical emotion effects and their relation to models of autonomic response organization A number of notable differences between emotions emerged: HR was increased for negative (anger, anxiety, contamination-related disgust, embarrassment, fear, crying sadness) and positive emotions (imagined anticipatory pleasure, happiness, joy) as well as for surprise. HR decreased in mutilation-related disgust, imminent-threat fear, non-crying sadness, acute sadness, affection, contentment, visual anticipatory pleasure, and suspense—emotions that all involve an element of passivity, and may be taken to suggest vagal mediation (cf. Porges, 1995, 2001; Vingerhoets, 1985). Contamination-related disgust was, however, the only negative emotion with conclusive data on increased cardiac vagal influence, as indicated by increased HRV (see also predictions of PNS involvement in disgust, Woody & Teachman, 2000). Acute sadness may be characterized by increased cardiac vagal influence as well, an assumption that remains to be clarified in future research. For positive emotions, increased HRV was present in amusement and joy, whereas HRV was decreased in happiness and visual anticipatory pleasure. This pattern of results supports previous statements that PNS activity may play a role in both pleasant and unpleasant emotions (e.g., Gellhorn, 1970; Kling, 1933).

TWA, an index of sympathetic influence on the heart (Furedy, Heslegrave, & Scher, 1992; but see Contrada, 1992), was found to be decreased in both anger and fear, whereas it was increased for mutilation-related disgust and contentment, indicating decreased cardiac sympathetic influence in the latter. Decreased HR in mutilation-related disgust and contentment may thus be caused by sympathetic withdrawal rather than parasympathetic influences (see also decreased LF/HF in mutilation disgust). In line with this, contentment was the only emotion that evidenced increased LVET, pointing to decreased left ventricular contractility that indicates decreased β-adrenergic sympathetic activation. Likewise, decreases in cardiac contractility were present in acute sadness, amusement, and happiness, as indicated by increased PEP. Notably, these emotions have all been related to approach motivation—with either successful (amusement, happiness) or unsuccessful outcome (acute sadness)—whereas emotions that are related to increased cardiac contractility (anger, disgust, embarrassment, and fear) may be summarized as an active coping response to aversive situations (Obrist, 1981; Schneiderman & McCabe, 1989) or be located on a dimension of avoidance, with the exception of anger that has been suggested to be associated with approach motivation (Carver, 2001; Harmon-Jones, Gable, & Peterson, this issue; but see the distinction of 'moving against' and 'moving toward'; Roseman, 2001). Effects of decreased β-adrenergic activation in certain approach-related emotions are also evident in peripheral cardiovascular measures. Decreased activation was found for acute sadness, with decreased blood pressure (SBP, DBP, MAP) and increased pulse transit time. Decreased blood pressure moreover occurred in contentment, and lengthening of pulse transit time in happiness. Larger pulse amplitude was present for anticipated sadness as well as for relief, although skin temperature generally decreased for different types of sadness.

Fear and anger were similar in a number of parameters, though differed—as predicted by the catecholamine hypothesis (Ax, 1953; Funkenstein et al., 1954; Stemmler, 2003, in press)—regarding TPR, which increased in anger, whereas it decreased in fear. Remarkably, fear was the only emotion in the present review that evidenced a decrease in TPR. All other emotions were characterized either by increased (anger, contamination-

related disgust, embarrassment, anticipatory sadness, amusement, happiness) or unchanged TPR (mutilation-related disgust, joy, pride).

Emotional activation was moreover shown to be related to notable differences in respiratory activity. For contamination-related disgust, respiratory timing parameters indicated faster breathing with increased expiratory and decreased inspiratory duration. This expiratory shift is also indicated in decreased Ti/Ttot, and may function to expel foul smell and related agents that the organism might have inhaled, as would be postulated by a basic coping strategies approach (compare to the physiological response pattern of vomiting; Sherwood, 2008). Ti and Ti/Ttot were also decreased in amusement, possibly reflecting effects of laughing on respiration, which notably occurs during the expiratory part of breathing. Of note, whereas amusement and contamination-related disgust were remarkably similar with respect to changes indicated by respiratory variables as well as vagal indicators, the two differed on β-adrenergic cardiac activation, with decreased PEP in contamination-related disgust, and increased PEP in amusement. In contrast, both increased Ti and increased Te, resulting in a general slowing of breathing, occurred in contentment, visual anticipatory pleasure, and relief. A marked inspiratory pause was present in anger, fear, and surprise, together with increased breathing frequency and increased Ti/Ttot. Fast deep breathing has been found for non-crying sadness that may function as an expressive emotion regulation strategy to actively suppress crying—a hypothesis that needs to be addressed in future research. Slow deep breathing has been found for relief, whereas shallow breathing occurs in anxiety, disgust, certain types of sadness, as well as anticipatory pleasure. Decreased pCO_2, indicating hyperventilation, was moreover reported for anxiety, fear, and imagined anticipatory pleasure, whereas increased pCO_2 was reported for acute sadness and contentment. These constellations may suggest variations according to basic motivational features such as valence and arousal (Bradley & Lang, 2000; Lang, Bradley, & Cuthbert, 1993) or shared core processes (see Berridge, 1999, for a discussion of commonalities between anxiety, fear, and anticipatory pleasure, viz. desire).

Decreases in electrodermal activity were present but in a few emotions, namely non-crying sadness, acute sadness, contentment, and relief. All other emotions were accompanied by increased electrodermal activity, which has been proposed to reflect cognitively- or emotionally-mediated motor preparation (Fredrikson, Furmark, Olsson, Fischer, & Långström, 1998), consistent with the notion of emotion causing an increase in action tendency (Brehm, 1999; Frijda, 1986). The decrease in electrodermal activity may in turn be taken as indicative of a decrease of motor preparation in the former emotions: sadness is typically experienced under conditions when a loss has occurred that cannot be undone, relief is experienced after a threat has passed, and contentment is experienced when one has attained a satisfactory outcome. As Brehm (1999, p. 7) pointed out, "the outcome has already occurred and there is nothing more to be done about it." Hence, neither emotion is characterized by an urge for action; rather, passivity is the shared motivational state.

Across response systems, psychophysiological responses in sadness-inducing contexts were characterized by decreased FPA, increased pulse transit time, and decreased electrodermal activity. As an exception, anticipatory sadness showed a reversed response pattern that was remarkably similar to that of anxiety in a number of measures. This may

point to a shared dimension of anticipation of harm or loss, as discussed in more detail below. Differential association of sadness or grief with either predominant SNS (Averill, 1968) or PNS activation (Gellhorn, 1964, 1970) might have been the result of having such different types of sadness as crying versus noncrying sadness or anticipatory versus acute sadness in mind.

It may be asked whether such positive emotions as amusement, happiness, and joy differ physiologically. The present review suggests that, whereas in amusement and joy HRV increases, it decreases in happiness. Amusement and happiness share a lengthening of PEP that is less clear in joy. All three emotions are characterized by increased electrodermal activity and faster breathing, which is deeper in amusement, but shallower in happiness. Similarly nuanced physiological response differences between interest, joy, pride, and surprise have been reported by Kreibig, Gendolla, and Scherer (this issue).

Measures of autonomic activation components Scientific investigation should not stop at the question of whether emotions differ physiologically, but rather ask whether and in which way emotions differ in terms of activation components of the ANS (e.g., Berntson et al., 1991; Berntson, Cacioppo, & Quigley, 1993; Stemmler, Grossman, Schmid, & Foerster, 1991; Stemmler, 1993). Investigations of ANS responding in emotion have long been impeded by the exclusive use of "convenience measures," such as HR and electrodermal activity, as sole indicators of the activation state of the organism (notably 23 of the publications included in the present review). However, as far back as William James (1884, 1894), complex emotion syndromes of highly specific and regionally organized regulation patterns have been described that include various quantifiable cardiovascular, eccrine, and respiratory responses. Because the heart is dually innervated by the SNS and PNS that speed or slow HR either in coupled (reciprocal, coactivated, or coinhibited) or uncoupled modes, HR is not informative of the respective branch's influence upon cardiac functioning (Berntson et al., 1991, 1993). Measures such as PEP and RSA that have been shown to be indicative of β-adrenergic sympathetic and vagal influence on the heart, respectively, are more informative and should thus be preferred. Moreover, skin conductance cannot function as the sole indicator of sympathetic activity since directional fractionation between response systems, such as the cardiovascular and electrodermal, is known to exist (Lacey, 1967). In addition, Berntson et al. (1991, p. 483) pointed out that "even chronotropic and inotropic influences on the heart . . . are mediated by separate efferent pathways that may be subject to differential central control. Consequently, indices should optimally be derived from the same functional dimension of the target organ." Thus, as the physiological adjustments that are elicited by emotion consist of an integrated pattern of responses, it is important to judiciously select a sufficient number of response measures to allow for the response pattern and its variations to be identified (Hilton, 1975; Schneiderman & McCabe, 1989; Stemmler, 2004).

Current models of autonomic control may moreover serve as a guide for interpreting findings of autonomic measures, in particular within replication studies of emotions (Berntson et al., 1991). Low replicability of autonomic response patterns of certain emotions may indicate low directional stability (i.e., nonmonotonic response functions), a restricted dynamic range, and low response lability (i.e., small rate of change) that is characteristic of nonreciprocal modes of activation. In contrast, high replicability of au-

tonomic response patterns would speak for high directional stability, a wide dynamic range, and high response lability that is characteristic of reciprocal modes of activation.

Emotion terminology In measuring autonomic responding in emotion, it is moreover important to place expected or observed effects on a sound conceptual basis. In this context, the importance of a clear and generally agreed upon terminology for labeling emotions cannot be stressed enough. Part of noted inconsistencies can be attributed to a lax and indistinct use of emotion labels for describing investigated emotions. For example, it is important to distinguish between such emotions as fear and anxiety, although they are both related to threat appraisals, but differ on the dimension of threat imminence (Barlow, 1991; Craske, 1999) or may be altogether based on two distinct behavioral systems (e.g., Gray, 1982; Gray & McNaughton, 2000). Similarly, amusement and happiness are both emotions related to a pleasurable experience. Amusement, however, refers to appealing to the sense of humor and should be reserved to such emotion inductions as those using slapstick comedy, whereas happiness refers to feelings of well-being or a pleasurable or satisfying experience, often caused by a deed of good fortune external to one's proper control (Aristotle, 1893; Veenhoven, 1991). Another important differentiation that could not be given due account in the above review of research findings is the distinction of shame and embarrassment (Lewis & Granic, 2000; Tangney, Miller, Flicker, & Barlow, 1996; Teroni & Deonna, 2008). Whereas shame is typically instigated by personal failure, embarrassment is more related to social exposure. On the other hand, the low-arousal positive emotions, here subsumed under the label of contentment, appear under a number of different names, such as pleasure, serenity, calmness, peacefulness, and relaxation. Moreover, anticipatory states of fear (anxiety) and sadness (anticipatory sadness), that were here distinguished from other forms of fear and sadness, respectively, might be regrouped into a category of worry or mental distress resulting from concern for an impending or anticipated painful experience of harm or loss, cutting across linguistically-defined boundaries (cf. Barr-Zisowitz, 2000, for a discussion of types of sadness). Both share an uncertainty about the kind of harm and what can be done to prevent a fatal outcome (cf. helplessness; Seligman, 1975). Appraisal models that present prescriptive appraisal–emotion mappings (e.g., Roseman, 1984; Roseman, Wiest, & Swartz, 1994; Scherer, 1982, 2001; Smith & Ellsworth, 1985) may serve as a general guide of how to label different experimental emotion conditions.

Apparent inconsistencies previously noted regarding autonomic activity in emotion (e.g., Feldman-Barrett, 2006) may thus be accounted for by conceptualizing "modal emotions" (Scherer, 1994, 2001) or "emotion families" (Ekman, 1997, 1999) as umbrella terms, under which different subtypes of that emotion exist, related to small but important differences in appraisal outcomes. In that sense, emotions might be grouped together in functional complexes under an abstract theme (cf. core relational themes; Lazarus, 1991) with its various specific, i.e., condition-sensitive, implementations.

Boundary Conditions

The present review focused on the relation between emotion and ANS activity. Emotion was defined as a multi-component response to an emotionally potent antecedent

event, causing changes in subjective feeling quality, expressive behavior, and physiological activation. However, there is no one-to-one relationship between emotion and changes in autonomic activation: feeling changes may occur without concomitant autonomic changes, just as autonomic changes may occur without concomitant feeling changes. Moreover, the present review assumed that study participants can faithfully report on their emotional state. However, decoupling of subsystems may occur, such as in emotion elicitation by subliminal stimulus presentation, unconscious emotions (presence of physiological effects, but absence of conscious feelings), or low response system coherence due to some intervening process, such as emotion regulation. To conclude, boundary conditions of the relation between emotion and autonomic activity and their implications for our understanding of emotion, feeling, and autonomic changes are discussed.

Feeling changes without concomitant autonomic changes A large body of literature reports on feeling changes in the absence of effects on autonomic responding. Typically, the type of affect manipulated within the context of such studies is labeled 'mood,' referring to a diffuse and long-lasting affective state that is not object-related, i.e., not experienced in simultaneous awareness of its causes (Frijda, 1993; Gendolla, 2000; Schwarz & Clore, 1988; however, see also the concept of the 'as-if body loop,' Damasio, 1999). Unlike emotions that are associated with specific motivational functions, e.g., motivating to remove the object of anger or to escape from the object of fear, moods do not have specific and stable motivational functions, but only informational function. Although moods have thus no direct impact on behavior, they do influence effort investment in subsequent behavior, such as performing a task.

Thus, whereas moods have immediate effects on subjective feeling state and facial expression, autonomic effects are typically absent during mood induction. No change from baseline activation of systolic and diastolic blood pressure, heart rate, and skin conductance level or spontaneous response rate has been found in the context of disguised mood manipulations, ranging between eight and ten minutes, with film excerpts (e.g., Silvestrini & Gendolla, 2007), music excerpts (e.g., Gendolla & Krüsken, 2001), autobiographic recall (e.g., Gendolla & Krüsken, 2002), or odors (Kiecolt-Glaser et al., 2008). Still, autonomic activation in subsequent task performance is moderated by mood, with the direction of effect depending on perceived difficulty level of the task (Gendolla, 2003; Gendolla & Brinkmann, 2005). When addressing affective effects on ANS activity, it is therefore of utmost importance to distinguish mood from emotion in order to know when to expect autonomic effects and when not.

Autonomic changes without concomitant feeling changes Reviewed results of effects of emotion on autonomic activity necessarily underly a specific measurement model. The ANS is not exclusively servant to emotion. Non-emotional physical, behavioral, and psychological factors affect physiological activation before, during, and after emotion, producing a complex amalgam of effects on physiological activity. Emotions are typically assumed to influence the ANS during a relatively brief period of time in the range of seconds to only a few minutes (Ekman, 1984, 1994). Once a behavioral reaction has been initiated, the physiological activity is in the service of that behavior and no longer reflects predominantly effects of emotion (Levenson, 2003; Stemmler, 2004).

To disentangle the potential confounding context effects from emotional effects on physiological activation, three major factors have been recognized that influence physiological responding (Stemmler, Heldmann, Pauls, & Scherer, 2001; Stemmler, 2004): (a) effects of the non-emotional context include posture, ambient temperature, ongoing motor activity, or cognitive demands, that are not in the service of emotion, constraining the physiological effects that the other components may exert; (b) effects of the emotional context include organismic, behavioral, and mental demands of enacting the emotion, given the specific momentary situational allowances and constraints on the emotional behavioral response, representing context-dependent effects of emotion that may be variable across situations; (c) effects of the emotion proper reflect specific physiological adaptations with the function to protect the organism through autonomic reflexes and to prepare the organism for consequent behavior, representing context-independent effects of emotion, which are expected to be stable across situations. Only the third component of the model, the emotion signature proper, is expected to allow statistical identification of specific, non-overlapping emotion responses (Stemmler et al., 2001).

Decoupling of subsystems in emotion To demarcate emotion from other physical and psychological influences on ANS activity, subsystem synchronization has been proposed as a distinctive feature of emotion (Scherer, 2001). Coherence constraints between response systems of emotion have, however, been noted in some studies (e.g., Mauss, Levenson, McCater, Wilhelm, & Gross, 2005; Reisenzein, 2000; Ruch, 1995). Such dissociation among different measures of emotion may be relatively normal rather than reflecting aberrant functioning. Emotion regulation, as one prominent process in this regard, may influence subsystem coherence in various ways, such as with respect to awareness of emotional responses (Koole, 2009). Emotions can, moreover, be elicited by subliminally presented stimuli that do not enter conscious awareness (e.g., Flykt, Esteves, & Öhman, 2007; Öhman, Carlsson, Lundqvist, & Ingvar, 2007; Wiens, Peira, Golkar, & Öhman, 2008). Thus, although feelings are often and typically conscious, conditions may arise, under which people do not report and/or are not aware of an emotional experience, although other subsystems, such as facial expression, physiological activation, and behavioral tendency indicate occurrence of emotion (cf. unconscious emotions; Wilson, 2002; Winkielman & Berridge, 2003, 2004).

Collecting valid data on autonomic responding in emotion has been and remains to be a challenge to emotion research (e.g., Levenson, 1988; Stemmler, 2003). For progress in the understanding of the functional organization of ANS activity in emotion, future researchers will have to closely scrutinize and, if possible, verify the specific type of emotion elicited as well as individual variations when analyzing autonomic parameters that need to be selected such that they allow differentiation of the various activation components of the ANS. Only if the hypothesis of autonomic response organization is properly tested, can valid inferences be drawn. It is hoped that this will pave the road to arriving at James' (1890) call for a generative principle that can summarize and account for the varieties of emotion.

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Appendix
Overview of reviewed studies

Table A1 provides an overview of the studies considered in the present review. Emotions were coded according to the emotion labels provided by the authors. The table moreover indicates the type of emotion induction method as well as assessed physiological measures (grouped into cardiovascular, respiratory, and electrodermal). Averaging period is the time segment over which averages for physiological variables were calculated; in case of different averages for different physiological variables, more than one number

is indicated; in case of varying averaging periods due to different stimulus presentation lengths, the mean averaging duration rounded to the next full minute is indicated.

This table can be downloaded as a text file from http://www.stanford.edu/∼skreibig. Data presented in this table were also used to generate the tag clouds.

Table A1 Overview of Studies on Effects of Emotion on Autonomic Nervous System Activity.

No.	Authors	Year	N	Emotion Labels	Experimental Paradigm	Cardiovascular	Respiratory	Electrodermal	Averaging Period (in s)
1	Adamson, Romano, Burdick, Corman, & Chebib	1972	10	sexual arousal	film clips	HR, HRV (CVT), SBP, DBP, FT	RR	nSRR	30, 120, 240
2	Adsett, Schottsteadt, & Wolf	1962	30	anger, anxiety, dejection, depression	stress-interview	HR, SBP, DBP, CO, SV, TPR			inst.
3	Alaoui-Ismaïli, Robin, Rada, Dittmar, & Vernet-Maury	1997	44	anger, disgust, fear, happiness, sadness, surprise	odorants	HR, palm temp., SBF	RR	OPD, SYDER	0.5
4	Allen, de L. Horne, & Trinder	1996	100	achievement failure, social rejection	standardized imagery	HR			30
5	Aue, Flykt, & Scherer	2007	42	goal conduciveness, relevance, threat	picture viewing (IAPS)	HR, FT, arm temp.			1, 5
6	Averill	1969	54	mirth, sadness	film clips	HR, SBP, DBP, FPA, FT, face temp.	RR, respiratory variability	nSRR	15, 360
7	Ax	1953	43	anger, fear	real-life (harassment, threat of short-circuit)	HR, SV, SBP, DBP, FT, face temp.	RR, Ti/Ttot, RD	nSRR, SCR	6
8	Baldaro, Battachi, Codispoti, Tuozzi, Trombini, Bolzani, et al.	1996	30	fear	film clips	HR			120
9	Baldaro, Mazzetti, Codispoti, Tuozzi, Bolzani, & Trombini	2001	42	disgust	film clips	HR, HRV (RSA (Porges))	RR		600
10	Bernat, Patrick, Benning, & Tellegen	2006	48	sexual arousal, threat	picture viewing (IAPS)	HR		SCR	6
11	Blatz	1925	18	fear	real-life (sudden backward-tilting chair)	HR	RR		
12	Blechert, Lajtman, Michael, Margraf, & Wilhelm	2006	42	anxiety	threat of shock	HR, TWA, HRV (RSA (HF), RSA (Porges), LF/HF, LF, VLF), FPTT, FPA	RR, Ti, Te, Pi, Pe, Ti/Ttot, Vt, Vm, Vi/Ti, respiratory variability, pCO2, sigh frequency, sigh Vt, % thoracic Vt	SRA, nSRR, SCL	300
13	Bloom & Trautt	1977	64	anxiety	threat of shock	HR, FPA			30
14	Boiten	1996	16	anger, disgust, fear, happiness, sadness, surprise	directed facial action	HR	Ttot, Ti, Te, Pi, Vt, Vm, FRC		10, 30
15	Boiten	1998	27	amusement, disgust, fear, suspense, tenderness	film clips		Ttot, Ti, Te, Pi, Pe, Ti/Ttot, Vt, Vm, Vt/Ti, RC/Vt, respiratory variability		120
16	Bradley, Codispoti, Cuthbert, & Lang	2001	95	disgust	picture viewing (IAPS)	HR		SCR	0.5
17	Bradley, Silakowski, & Lang	2008	49 (control)	dental anxiety	threat of shock	HR		SCR	20
18	Britton, Taylor, Berridge, Mikels, & Liberzon	2006	40	appetite, disgust, amusement, sadness	film clips	HR		SCR	30, 90
19	Brown, Sirota, Niaura, & Engebretson	1993	16	elation, sadness	Velten method	HR, SBP, DBP, MAP			
20	Chan & Lovibond	1996	23 22 (control)	threat	threat of shock			SCL	40
21	Christie & Friedman	2004	34	amusement, anger, contentment, disgust, fear, sadness	film clips	IBI, HRV (MSD), SBP, DBP, MAP		SCL	60
22	Codispoti & De Cesarei	2007	50	disgust, sexual arousal, threat	picture viewing (IAPS)	HR		SCR	0.5
23	Codispoti, Surcinelli, & Baldaro	2008	55	disgust, sexual arousal	film clips	HR, HRV (RSA (Porges))		SCL	60
24	Collet, Vernet-Maury, Delhomme, & Dittmar	1997	30	anger, disgust, fear, happiness, sadness, surprise	picture viewing (faces)	SBF, palm temp.	RR	OPD, SYDER, SCR, duration	0.5
25	Davidson & Schwartz	1976	20	anger, relaxation	personalized recall	HR			120
26	Demaree, Schmeichel, Robinson, & Everhart	2004	26 (control)	amusement, disgust	film clips	IBI, HRV (RSA (HF), LF/HF)		SCL	120
27	Dimberg	1986	28	fear	picture viewing	HR		SCR	1
28	Dimberg & Thunberg	2007	28 (control)	anger, happiness	picture viewing (faces)	HR		SCR	1, 5
29	Drummond	1999	19 (control)	anger	real-life (harassment)	IBI, SBP, DBP, FPA, forehead PA		SCR	15

Table A1 (Continued)

No.	Authors	Year		Emotion Labels	Experimental Paradigm	Cardiovascular	Respiratory	Electrodermal	Averaging Period (in s)
30	Dudley	1964	10	anger, anxiety, depression, relaxation	hypnosis		RR, Vm, pCO2		
31	Eisenberg, Fabes, Bustamante, Mathy, Miller, & Lindholm	1988	82	anxiety, sadness, sympathy	film clips	HR			0.5, 3.5
32	Ekman, Levenson, & Friesen	1983	16	anger, disgust, fear, sadness, surprise	directed facial action, personalized recall	HR, FT		SCL	10, 30
33	Etzel, Johnsen, Dickerson, Tranel, & Adolphs	2006	13 (18)	fear, happiness, sadness	musical excerpts	HR, HRV (SDNN, SDSD, RSA (peak-valley))	Ttot, Ti, Te, respiratory variability		1, 5, 65
34	Feleky	1916	6	anger, disgust, fear, hatred, laughter, pleasure, wonder	personalized recall		(RR), I/E ratio, RD, RD/Ttot		
35	Fiorito & Simons	1994	31 (control)	anger, contentment, fear, joy, sadness, sexual arousal	standardized imagery, personalized recall	HR		nSRR	20
36	Foster, Smith, & Webster	1999	36	anger	real-life, standardized imagery, personalized recall	HR		SCL	30?
37	Foster & Webster	2001	10	anger, mirth	personalized recall	HR		SCL	30
38	Foster, Webster, & Williamson	2003	23	mirth	real-life, standardized imagery, personalized recall	HR		SCL	30?
39	Fredrickson & Levenson	1998	60 72	fear, sadness	film clips	HR, FPTT, EPTT, FPA			120
40	Funkenstein, King, & Drolette	1954	69	anger	real-life (harassment)	HR, SV, CO, SBP, DBP, TPR			inst.
41	Gehricke & Fridlund	2002	20	happiness, sadness	standardized imagery	HR		SCL	60
42	Gilissen, Bakermans-Kraneburg, van Ijzendoorn, & van der Veer	2008	78 92	fear	film clips	HRV (RMSSD)		SCL	60
43	Gilissen, Koolstra, van Ijzendoorn, Bakermans-Kraneburg, & van der Veer	2007	78	fear	film clips	HRV (RMSSD)		SCL	60
44	Gross	1998	120	disgust	film clips	IBI, FPA, FT		SCL	1, 60
45	Gross, Fredrickson, & Levenson	1994	150	sadness	film clips	HR, FPA, FPTT, EPTT, FT	RP, RD	SCL	~100
46	Gross & Levenson	1993	43 42	disgust	film clips	HR, FPA, FPTT, EPTT, FT	RP, RD	SCL	1, 60
47	Gross & Levenson	1997	180	amusement, sadness	film clips	IBI, FPA, FPTT, EPTT, FT	RP, RD	SCL	210
48	Grossberg & Wilson	1968	18 10 (control)	fear	adapted standardized imagery	HR		SCL	25
49	Gruber, Johnson, Oveis, & Keltner	2008	54 (control)	disgust, happiness, pride, sadness	film clips	HR, HRV (RMSSD)		SCL	90
50	Guliani, McRae, & Gross	2008	16	amusement	film clips	HR, SV, MAP, FPA, FPTT, EPTT, FT	RR	SRA	10, 20
51	Hamer, Tanaka, Okamura, Tsuda, & Steptoe	2007	55	anger, depression	personalized recall	HR, SBP, DBP, TPR, SV, CO			300
52	Harris	2001	34 36	embarrassment	real-life (filmed while singing, watching video of self)	HR, SBP, DBP			60
53	Harrison, Carroll, Burns, Corkill, Harrison, Ring, et al.	2000	30	boredom, excitement, humor	film clips	HR, PEP, SV, CO, SBP, DBP, MAP, TPR			60
54	Herrald & Tomaka	2002	109	anger, pride, shame	real-life (harassment, humiliation, praise)	HR, PEP, SV, CO, SBP, DBP, MAP, TPR			180
55	Hess, Kappas, McHugo, Lanzetta, & Kleck	1992	28	anger, happiness, peacefulness, sadness	feel emotion, express emotion without feeling, express and feel emotion	HR		SCL	30
56	Hofmann, Moscovitch, & Kim	2006	32	embarrassment, social anxiety	real-life (speech preparation, filmed while singing, watching video of self)	HR, HRV (RSA (peak-valley))		SCL	30
57	Hubert & de Jong-Meyer	1990	24	amusement, suspense	film clips	HR	RR	nSRR, SCL	30
58	Hubert & de Jong-Meyer	1991	20	amusement, suspense	film clips	HR		nSRR, SCL	60
59	Jönsson & Sonnby-Borgström	2003	53	anger, happiness	picture viewing (faces)	HR, HRV (RSA (HF))			0.5, 300

Table A1 (Continued)

No.	Authors	Year		Emotion Labels	Experimental Paradigm	Cardiovascular	Respiratory	Electrodermal	Averaging Period (in s)
60	Kaiser & Roessler	1970	20	disgust	film clips			SRA, nSRR	770
61	Khalfa, Roy, Rainville, Bella, & Peretz	2008	50	happiness, sadness	musical excerpts	HR, SBP, DBP	RR	nSRR	15
62	Klorman, Weissberg, & Wiesenfeld	1977	42 (control)	disgust, humor	picture viewing	HR	RR, Ti, Te	SCR, SCL, decay	1
63	Kornreich, Philippot, Verpoorten, Dan, Baert, Le Bon, et al.	1998	14 (control)	anger, disgust, amusement, sadness	film clips	HR, HRV (SDNN)			300
64	Kreibig, Wilhelm, Roth, & Gross	2007	34	fear, sadness	film clips	HR, TWA, HRV (RSA (HF), LF), PEP, LVET, HI, SV, SBP, DBP, EPA, EPTT, FT	RR, Ti/Ttot, Vt, Vi/Ti, pCO2, respiratory variability	SRA, nSRR, SCL	600
65	Kring & Gordon, study 1	1998	43	disgust, fear, amusement, sadness	film clips			nSRR	300
	Kring & Gordon, study 2	1998	67	anger, disgust, fear, happiness, sadness	film clips			nSRR	240
66	Kring & Neale	1996	20	fear, disgust, happiness, sadness	film clips			nSRR	300
67	Krumhansl	1997	38	fear, happiness, sadness	musical excerpts	IBI, HRV (RSA (not specified)), SBP, DBP, MAP, FPA, FPTT, EPTT, FT	RP, RD	SCL	1, 180
68	Kunzmann & Grühn, study 2	2005	96	contentment, sadness	film clips	IBI, FPA, FT	RP	SCL	540
69	Kunzmann, Kupperbusch, & Levenson	2005	95	contentment, disgust	film clips	IBI, SBP, DBP, FPA, FPTT, EPTT, FT	RP, RD	SCL	60
70	Lang, Greenwald, Bradley, & Hamm	1993	64	disgust, sexual arousal	picture viewing (IAPS)	HR		SCR	0.5
71	Lavoie, Miller, Conway, & Fleet	2001	42	anger in defense of other, anger in self-defense	real-life (harassment)	HR, CO, SV, SBP, DBP, TPR, forearm blood flow, forearm vascular resistance			540
72	Lerner, Gonzalez, Dahl, Hariri, & Taylor	2005	92	fear, anger, disgust	real-life (harassment)	HR, SBP, DBP, MAP			1380 (inst.)
73	Levenson, Carstensen, Friesen, & Ekman	1991	20	anger, disgust, fear, happiness, sadness, surprise	directed facial action, personalized recall	HR, FT		SCL	10, 15
74	Levenson, Ekman, & Friesen, study 1	1990	62[a] 16[b]	anger, disgust, fear, happiness, sadness, surprise	directed facial action	HR, FT		SCL	10
	Levenson, Ekman, & Friesen, study 2	1990	16	anger, disgust, fear, happiness, sadness, surprise	directed facial action	HR, FT		SCL	10
	Levenson, Ekman, & Friesen, study 3	1990	30	anger, disgust, fear, happiness, sadness, surprise	directed facial action	HR, FT		SCL	10
75	Levenson, Ekman, Heider, & Friesen	1992	46 62[a]	anger, disgust, fear, happiness, sadness	directed facial action	HR, FPA, FPTT, FT	RP, RD	SCL	10
76	Luminet, Rimé, Bagby, & Taylor	2004	50	sadness	film clips	HR, SBP, DBP			60, 360
77	Marci, Glick, Loh, & Dougherty	2007	10	anger, happiness, sadness	personalized recall	IBI, HRV (RSA (HF))		SCL	60
78	Marsh, Beauchaine, & Williams	2008	23 (control)	sadness	film clips	HRV (RSA (HF)), PEP		SCL	1, 30
79	McCaul, Holmes, & Solomon, study 1	1982	27	calmness, fear	posed facial expressions	HR		SCL	16
	McCaul, Holmes, & Solomon, study 2	1982	34	calmness, fear, happiness	posed facial expressions	HR		SCL	10
80	Miller, Levin, Kozak, Cook, McLean, & Lang	1987	24 (12)	fear, anger	standardized imagery, personalized recall	HR	RP, RD, I/E ratio	SCL	30
81	Montoya, Campos, & Schandry	2005	32	anger, fear	film clips	HR, PEP, LVET, HI, SV, CO, SBP, DBP, MAP, TPR			900
82	Murakami & Ohira	2007	24	anxiety	real-life (speech preparation)	HR, HRV (RSA (HF), LF, LF/HF)		SCL	300
83	Neumann & Waldstein	2001	42	anger, joy, relaxation, sadness	personalized recall	HR, PEP, LVET, SV, CO, SI, CI, SBP, DBP, MAP, TPR			180
84	Nyklicek, Thayer, & Van Doornen	1997	26	agitation, happiness, sadness, serenity	musical excerpts	IBI, HRV (RSA (peak-valley)), PEP, LVET, SV, CO, SBP, DBP, MAP, FPA, TPR	RR, Ti, Te, Pi, Pe		180

[a] *comparison group based on pooled sample from Levenson et al. (1990)*

[b] *same sample as Ekman et al. (1983)*

Table A1 (Continued)

No.	Authors	Year		Emotion Labels	Experimental Paradigm	Cardiovascular	Respiratory	Electrodermal	Averaging Period (in s)
85	Palomba, Sarlo, Angrilli, Mini, & Stegagno	2000	46	contentment, disgust, fear	film clips	HR, HRV (RSA (peak-valley)), TWA	RR	SCL	33
86	Palomba & Stegagno	1993	15	fear, sadness	film clips	HR	RR		60
87	Pauls & Stemmler,	2003	78	anger, fear	real-life (harassment, speech preparation)	HR, HRV (RMSSD), PEP, SBP, DBP, TPR		nSRR	60
88	Prkachin, Mills, Zwaal, & Husted	2001	50	anger	real-life (anger interview, harassment)	HR, SV, CO, SBP, DBP, TPR			300
89	Prkachin, Williams-Avery, Zwaal, & Mills	1999	31	anger, disgust, fear, happiness, sadness	personalized recall	HR, SV, CO, SBP, DBP, FT, TPR			300 (inst./16)
90	Rainville, Bechara, Naqvi, & Damasio,	2006	43	anger, fear, happiness, sadness	personalized recall	IBI, HRV (RSA (peak-valley, HF), RMSSD, SDNN, MSD)	RP, RD, respiratory variability		90
91	Rimm-Kaufman & Kagan	1996	32	anxiety, fear, happiness, performance anxiety	real-life (test situation, personal questions), film clips	FT			inst.
92	Ritz, George, & Dahme	2000	12	depression, happiness	picture viewing (IAPS), Velten method	IBI, HRV (RSA (peak-valley))	Ttot, Ti, Te, Pe, Ti/Ttot, Vt, Vi/Ti, Vm, FRC, Ros		12
93	Ritz, Steptoe, Wilde, & Costa	2000	24 (control)	anxiety, anger, contentment, depression, amusement	film clips, math task, picture viewing	HR, SBP, DBP	RR, Vt, Vm, Ros	SCL	180
94	Ritz, Thöns, Fahrenkrug, & Dahme	2005	30 (control)	anxiety, contentment, depression, disgust, happiness, sexual arousal	picture viewing (IAPS)	IBI, HRV (RSA (peak-valley))	Ttot, Ti, Te, Pe, Ti/Ttot, Vt, Vt/Ti, Vm, Ros	SCR	1, 15
95	Ritz, Wilhelm, Gerlach, Kullowatz, & Roth	2005	14 (control)	disgust	film clips	HR, SBP, DBP	pCO2, peak HV, tonic HV		0.5, 10, 240
96	Roberts & Weerts	1982	16	anger, fear	adapted standardized imagery	HR, SBP, DBP			inst., 30
97	Robin, Alaoui-Ismaïli, Dittmar, & Vernet-Maury	1998	44	dental anxiety	odorants	SBF, palm temp.	RR	OPD, SYDER, SCR, duration	0.5
98	Rochman & Diamond	2008	27 36	anger, sadness	personalized recall	HRV (RSA (HF)), FT		SCL	120
99	Rohrmann & Hopp	2008	83–89	disease-related disgust, food-related disgust	film clips	HR, HRV (RMSSD), PEP, LVET, SV, CO, SBP, DBP, TPR		SCL	60
100	Rottenberg, Gross, & Gotlib	2005	26 (control)	happiness, sadness	film clips, standardized imagery, personalized recall	HR, FPTT	RR	nSRR	180
101	Rottenberg, Gross, Wilhelm, Najmi, & Gotlib	2002	33 (control)	sadness	film clips	HR, FT	RR	nSRR, SCL	180
102	Rottenberg, Kasch, Gross, & Gotlib	2002	33 (control)	amusement, fear, sadness	film clips	HR		nSRR	180
103	Rottenberg, Wilhelm, Gross, & Gotlib	2003	31	sadness	film clips	IBI, HRV (RSA (HF))	RR, Vt		180
104	Schachter	1957	15 (48)	anger, fear	real-life (harassment, threat of short-circuit)	HR, SI, CI, SBP, DBP, TPR, FT	Ti/Ttot	SCR	6
105	Schwartz, Weinberger, & Singer	1981	32	anger, fear, happiness, relaxation, sadness	personalized recall, step walking	HR, SBP, DBP			15
106	Sinha, Lovallo, & Parsons	1992	27	anger, fear, joy, sadness	personalized recall	HR, PEP, LVET, HI, SV, CO, SBP, DBP, MAP, TPR			30
107	Sinha & Parsons	1996	27	anger, fear	personalized recall	HR, SBP, DBP, FT		SCL	30
108	Sokhadze	2007	29	disgust	picture viewing (IAPS)	HR, HRV (RSA (HF), LF, LF/HF)	RR, RD	SRA, nSRR,SCL	60
109	Stemmler	1989	42	anger, fear, happiness	real-life (threatening radio play, harassment, appreciation and reward, personalized recall)	IBI, FPA, FPTT, FT	RP	nSRR, SCL	60
110	Stemmler, Aue, & Wacker	2007	118	anger, fear	standardized imagery	IBI, TWA, PQ-time, QT-time, ST-segment, HRV (RMSSD), PEP, LVET, HI, SV, CO, ventricular ejection speed, RZ-time, SBP, DBP, TPR, FPA, FPTT	RR		60

Table A1 (Continued)

No.	Authors	Year		Emotion Labels	Experimental Paradigm	Cardiovascular	Respiratory	Electrodermal	Averaging Period (in s)
111	Stemmler, Heldmann, Pauls, & Scherer	2001	158	anger, fear	real-life (harassment, speech preparation), adapted standardized imagery	IBI, TWA, PQ-time, QT-time, ST-segment, HRV (MSSD), LVET, PEP, SV, CO, ventricular ejection speed, HI, RZ-time, SBP, DBP, TPR, FPA, FPTT, FT, forehead temp.	RR	SRA, nSRR, SCL	60
112	Sternbach	1962	10	fear, happiness, humor, sadness	film clips	HR, FPA	RR	SCL	30, 60
113	Theall-Honey & Schmidt	2006	20 (control)	anger, fear, happiness, sadness	film clips	HR, HRV (SDNN)			60
114	Tourangeau & Ellsworth	1979	123	fear, sadness	film clips	HR	RR	nSRR, SCL	5
115	Tsai, Chentsova-Dutton, Freire-Bebeau, & Przymus	2002	98	anger, disgust, happiness, love, pride, sadness	personalized recall			SCL	120
116	Tsai, Levenson, & Carstensen	2000	96	amusement, sadness	film clips	IBI, FPA, FPTT, EPTT, FT	RP	SCL	120
117	Tsai, Pole, Levenson, & Munoz	2003	10 (control)	amusement, sadness	film clips	IBI, FPA, FPTT, FT		SCL	180
118	Tugade & Fredrickson	2004	57 57	anxiety	real-life (speech preparation)	HR, SBP, DBP, FPA, FPTT, EPTT			60
119	Uchiyama	1992	6	anger, fear, joy	real-life (threatening medical diagnosis, harassment, appreciation and reward)	HR, SBP, DBP	RR	nSRR	30
120	Van Diest, Proot, Van De Woestijne, Han, Devriese, Winters, et al.	2001	40	depression, fear, pleasure, relaxation	standardized imagery		Ti, Te, Vt, pCO2		60
121	Van Diest, Thayer, Vandeputte, Van de Woestijne, & Van den Bergh	2006	98[c]	anxiety, fear	standardized imagery		respiratory variability: Ti, Te, Vi, Vi/Ti, pCO2		30, 90
122	Van Diest, Winters, Devriese, Vercamst, Han, Woestijne, Van de Woestijne, et al.	2001	40	depression, desire, fear, relaxation	standardized imagery	HR	RR, Ti, Te, Vt, pCO2		90
123	Van Egeren, Abelson, & Thornton	1978	28	anger	real-life (harassment)	HR, SBP, DBP, FPA, FPTT			6
124	Van Oyen Witvliet & Vrana	1995	48	fear, joy, relaxation, sadness	standardized imagery	HR		SCL	8
125	Van Reekum, Johnstone, Banse, Etter, Wehrle, & Scherer	2004	33	goal conduciveness, intrinsic pleasantness	computer game	IBI, FPTT, FT slope		SCR	5, 3
126	Vianna & Tranel	2006	16	disgust, fear, happiness, sadness	film clips	HRV (RSA (HF), LF)		SCL	120
127	Vlemincx, Van Diest, De Peuter, Bresseleers, Bogaerts, Fannes, et al.	2009	36 42 37	threat, relief, relaxation	threat of shock		RR, Vi, Ve, sigh frequency		10 10 5-50
128	Vrana	1993	50	anger, disgust, joy, pleasure	standardized imagery	HR		SCL	4
129	Vrana & Gross	2004	9 (control)	anger, joy	picture viewing (faces)	HR		SCL	8
130	Vrana & Rollock	2002	112	anger, fear, joy	standardized imagery	HR, SBP, DBP, MAP		SCL	30
131	Waldstein, Kop, Schmidt, Haufler, Krantz, & Fox	2000	30	anger, happiness	film clips, personalized recall	HR, SBP, DBP			inst., 120-180
132	Williams, Das, Liddell, Olivieri, Peduto, Brammer, et al.	2005	13	anger, disgust, fear	picture viewing (faces)			nSRR, SCR, latency, rise time, recovery time	0.5
133	Winton, Putnam, & Krauss	1984	20	disgust, sexual arousal	picture viewing	HR		SCR	1, 12
134	Yogo, Hama, Yogo, & Matsuyama	1995	24	anger, joy	standardized imagery	SBP, DBP, MAP			30

[c] *includes participants from Van Diest, Winters, et al. (2001)*

Note. For abbreviations of physiological measures, see Table 3.