Autonomic Nervous System Activity in Emotion: A Review

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Review

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.

Contents

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1. Introduction

Autonomic responding in emotion has been an active research topic since, almost a century ago, Walter Cannon (1915) first studied the physiology of emotion (Brown and 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.

1.1. 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 et al., 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 et al., 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 et al., 1997, 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-specific patterning was found to be more consistent than emotion-specific patterning: negative emotions were associated with stronger autonomic responses than positive emotions (cf. 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 et al., 2008; Mauss and Robinson, 2009).

Diametrically opposed to Feldman-Barrett's (2006) position, Stemmler (2009) argued why the ANS should *not* convey specific activation patterns for emotions, if those have specific functions for human adaptation. Stemmler (2004, 2009) 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 et al., 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 (2009) 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.

1.2. 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 and 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 and McLachlan, 1992b,a; Jänig and 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 et al., 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 and 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 and Ellenberger, 1988; Poon and Siniaia, 2000; Taylor et al., 1999). Thus, comprehensive assessment of cardiovascular, electrodermal, and respiratory measures can provide complementary 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 and 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.

1.3. 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 and 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, models on different levels have complementary value, as

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 and Scherer (2003); Scherer (1984, 1987, 2001, 2009)
Specific cardiovascular appraisal hypotheses	Blascovich and Katkin (1993); Blascovich et al. (2003); Gendolla (2004); Gendolla and Wright (2005); Wright (1996, 1998); Wright and Kirby (2001)
Functioning of Associative Networks	
Bio-informational theory of emotional imagery	Lang (1979, 1993); Miltner et al. (1986); Sartory (1993)
Brain–behavioral level	
Functioning of brain–behavioral systems	
Behavioral coping	Obrist (1981); Schneiderman and McCabe (1989)
Dual-system models	Bradley and Lang (2000); Cloninger (1987); Davidson (1998); Lang and Bradley (this issue); Lang et al. (1997)
Polyvagal theory	Porges (1995); Porges et al. (1996); Porges (2001, 2007)
Reinforcement sensitivity theory	Beauchaine (2001); Corr (2008); Fowles (1980); Gray (1982, 1987); Gray and McNaughton (2000)
Functioning of behavioral modes	
Basic modes of defensive coping	Folkow (2000); Stemmler (2009)
Modes of defensive coping and environmental demands	Bandler and Shipley (1994); Bandler et al. (2000); Bernard and Bandler (1998); Keay and Bandler (2001, 2002)
Predator imminence model	Bradley and 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 et al. (2000)
Sympathetic versus parasympathetic response dominance	Gellhorn (1964, 1965, 1970); Hess (1957)
Autonomic space	Berntson et al. (1991)
Functioning of transmitter substances	
Catecholamine hypothesis	Ax (1953); Funkenstein et al. (1954)
Receptor-types hypothesis	Stemmler (2003, 2004, 2009)

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.

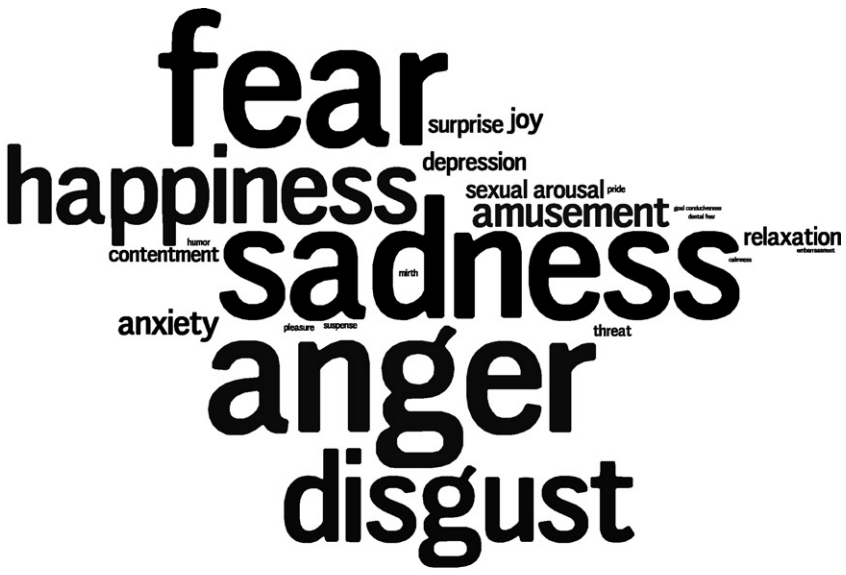
2. Empirical findings of ANS activity in emotion

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:

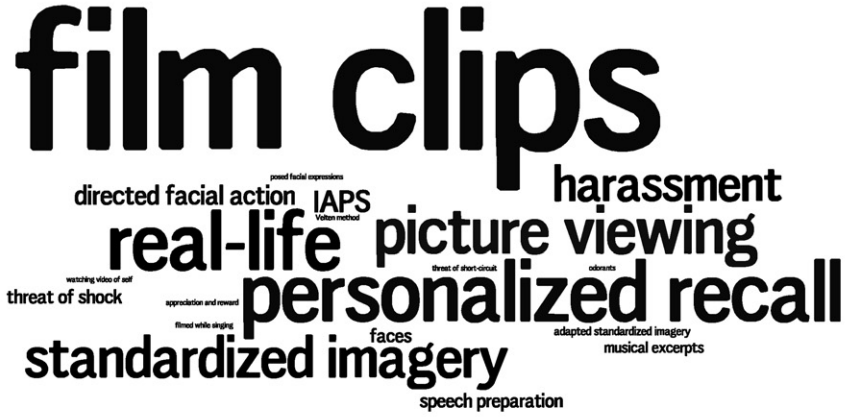
> [emotion] *and* [autonomic nervous system *or* cardiovascular *or* cardiac *or* heart *or* respiration *or* respiratory *or* electrodermal *or* skin conductance]

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 and Lang, 2000; Lang, 1994; Russell, 2003), discrete emotion theory (Ekman, 1999; Izard, 1992), as well as appraisal models of emotion (Scherer, 2001; Smith and Kirby, 2004). Emotion was thus conceptualized as a multicomponent 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.

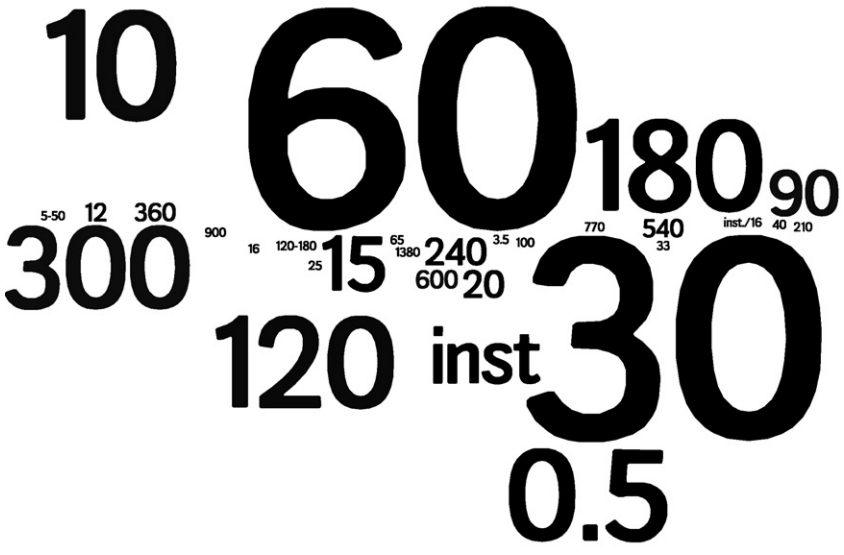
This literature search resulted in the identification of 134 publications. A detailed account of the studies included in the present review can be found in Table A.1 (Appendix). To summarize this information, tag clouds were created. A tag cloud is a visualization of word frequency in a given database as a weighted list. For the present purpose, coding labels in Table A.1 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/). Fig. 1 presents an illustration of the relative number of studies that investigated different emotions (Fig. 1a), using different kinds of emotion induction paradigms (Fig. 1b), and quantified physiological variables according to different averaging durations (Fig. 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-s intervals; other common averaging intervals are 1/2- or 10-s intervals and 120-, 180-, or 300-s intervals. It is noted that studies were coded for averaging period because it was hypothesized that this factor might influ-



(a) Investigated emotions



(b) Emotion induction method



(b) Averaging duration of physiological variables

Fig. 1. Illustration of relative frequency of investigated emotions (a), emotion induction methods (b), and averaging duration for physiological variables. (c). Figures were simplified by omitting low-frequency words.

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

Fig. 2 provides an illustration of the relative number of studies that have used different cardiovascular (Fig. 2a), respiratory (Fig. 2b), or electrodermal (Fig. 2c) measures as well as their overall usage (Fig. 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 measures, 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).

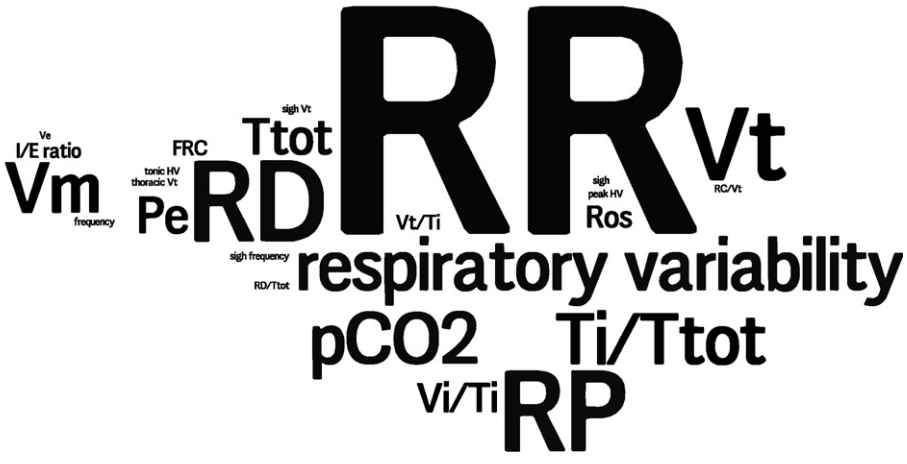
For the negative emotions, these were:

(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);

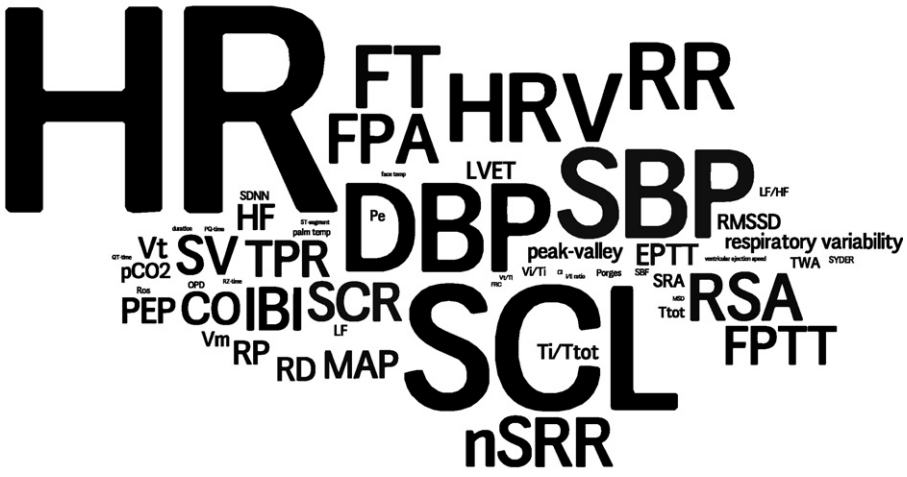
(a) Cardiovascular measures



(b) Respiratory measures

Fig. 2. Illustration of relative frequency of use of ANS measures as indicated by relative font size. Figures were simplified by omitting low-frequency words.

(c) Electrodermal measures



(d) All measures of autonomic functioning

Fig. 2. (*Continued*).

(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 A.1 (numbers in brackets refer to the study number in Table A.1). 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 following. 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.

2.1. The negative emotions

2.1.1. 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.

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	↓	↑	(↑)	↓ (↓) deptl	↑ ↓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 ($FETCO_2$)
P_e	Post-expiratory pause time	
P_i	Post-inspiratory pause time	
RC/V_t	Percentage of rib cage contribution to V_t	
RD/T_{tot}	Amount of respiratory work (depth divided by breath cycle duration)	
R_{os}	Oscillatory resistance	
RR	Respiration rate	1/Total respiratory cycle duration (T_{tot})
SaO_2	Transcutaneous oxygen saturation	
T_e	Expiratory time	
T_i	Inspiratory time	
T_i/T_{tot}	Inspiratory duty cycle	
V_e	Expiratory volume	
V_e/T_e	Expiratory flow rate or expiratory drive	
V_i	Inspiratory volume	
V_i/T_i	Inspiratory flow rate or inspiratory drive	
V_m	Minute ventilation	
V_t	Tidal volume	Respiration depth (RD), typically uncalibrated ribcage measurements in arbitrary units
V_t/T_i	Mean inspiratory flow rate	
V_tV	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)	

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 T_i and T_e, increased P_i [15], shortened T_e and decreased I/E-ratio [80], increased [34], unchanged [75], or decreased [15] respiratory depth, and increased FRC, increased R_{os} [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) 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 and 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 and 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.

2.1.2. 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 T_i and T_e [12, 30, 84, 121], as well as decreased V_t [12, 121], increased sigh frequency and V_t variability [12] (however, higher sigh frequency during relief than tension has also been found [127]), increased R_{os} [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 T_{tot} associated with increased T_e and decreased T_i, decreased V_m, and an unspecific small increase in R_{os}. 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).

2.1.3. 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 et al. (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 et al. (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 non-differentially 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 T_i and increased or no change in T_e [14, 15, 94], that may contribute to decreased T_i/T_{tot} and V_t/T_i [15], decreased respiratory volume (e.g., decreased V_t, V_m [14, vomiting clip 15, 24, 75, 94]), and increased R_{os} [94], as well as larger variability in T_e, V_t, V_m, V_t/T_i [vomiting clip 15]. Other than decreased V_t [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.

2.1.4. 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.

2.1.5. 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 T_i and T_e [14, 120, 122], or predominantly decreased T_e and unchanged T_i, as also indicated by increased T_i/T_{tot} and I/E-ratio [33, 34, 64], and increased P_i [14]. Volumetric measures moreover indicate increased respiratory volume [34, 75] or decreased respiratory volume [14, 67, 120, 122], and increased V_m [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 V_i/T_i [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 and 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 and Roth, 1998).

2.1.6. 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 material, 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 V_t [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, T_i, T_e, and V_t, and increased P_i 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 et al. (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 R_{os} [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 T_e [33], decreased RR and RD [67], unchanged [61] or increased RR, associated with decreased T_e, T_i, and P_e [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 T_i and T_e, 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 V_t [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 R_{os}, 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, T_i, V_t, increased T_{tot}, T_e, HRV (peak-valley RSA), unchanged R_{os} 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.

2.2. The positive emotions

2.2.1. 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 T_i/T_{tot}, increased variation in T_e, and decreased variation in V_t, V_m, and V_t/T_i [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.

2.2.2. 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 R_{os} [93], decreased T_i, V_t, T_i/T_{tot}, and increased P_i and variability of T_e, V_t, V_m, and V_t/T_i [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]).

2.2.3. 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 T_i, T_e [94, 120] or unchanged T_i and T_e [122], decreased V_t [94, 122] or increased V_t [120], and increased pCO_2 [120, 122] as well as unchanged R_{os}, SCR, and V_m [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), T_i, T_e, and P_i 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.

2.2.4. 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 T_i and T_e [14, 84], decreased P_e [84], increased P_i and FRC [14], or unchanged T_i, decreased T_e, and unchanged respiratory variability [33], increased depth [75] or decreased depth [14, 67], decreased respiratory variability of period and amplitude [90], increased V_t/T_i, unchanged FRC, and increased R_{os} [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 respiratory activity (decreased RR, V_t, R_{os}, and increased T_i and T_e) 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 et al., 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 et al., 2009).

2.2.5. 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.

2.2.6. 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), T_i, T_e, decreased RR, V_t, V_m, and unchanged R_{os} [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, T_i, T_e, and V_t [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.

2.2.7. 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.

2.2.8. 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 T_i, T_e, increased V_t, and decreased V_t variability as well as decreased sigh frequency [12]; or increased V_i including sighs, unchanged V_i excluding sighs, and increased sigh frequency [127]). Notably, increased sigh frequency has also been reported for conditions of relief in animal experiments (Soltysik and Jelen, 2005). Finally, decreased electrodermal reactivity is typically reported (decreased SCR reactivity [12, 17]; decreased nSRR [12]; decreased SCL [12, 20]).

2.3. Emotions without clear valence connotation

2.3.1. 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 et al., 1975; Niepel, 2001; Qiyuan et al., 1985) and the orienting reflex (Siddle and Heron, 1976; Siddle et al., 1983; Siddle, 1985, 1991; Sokolov, 1990) may prove more conclusive.

2.3.2. 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 T_e, P_e, V_t/T_i, and variability of T_e, and increased T_i/T_{tot} [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 et al., 1968).

3. 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?

3.1. 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 et al., 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.

3.1.1. 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 and 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 et al., 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 and 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 et al., 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, 2009)—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 T_i/T_{tot}, 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). T_i and T_i/T_{tot} 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 T_i and increased T_e, 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 T_i/T_{tot}. 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 and Lang, 2000; Lang et al., 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 et al., 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 et al. (this issue).

3.1.2. 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, 1993; Stemmler et al., 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 and 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 autonomic response patterns would speak for high directional stability, a wide dynamic range, and high response lability that is characteristic of reciprocal modes of activation.

3.1.3. 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 and 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 and Granic, 2000; Tangney et al., 1996; Teroni and 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 et al., 1994; Scherer, 1982, 2001; Smith and 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.

3.2. 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.

3.2.1. 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 and 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 and Gendolla, 2007), music excerpts (e.g., Gendolla and Krüsken, 2001), autobiographic recall (e.g., Gendolla and 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 and 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.

3.2.2. 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 et al., 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).

3.2.3. 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 et al., 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 et al., 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 and 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 A. Overview of reviewed studies

Table A.1 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 A.1
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 et al.	1972	10	Sexual arousal	Film clips	HR, HRV (CVT), SBP, DBP, FT	RR	nSRR	30, 120, 240
2	Adsett et al.	1962	30	Anger, anxiety, dejection, depression	Stress-interview	HR, SBP, DBP, CO, SV, TPR			inst.
3	Alaoui-Ismaïli et al.	1997	44	Anger, disgust, fear, happiness, sadness, surprise	odorants	HR, palm temp., SBF	RR	OPD, SYDER	0.5
4	Allen et al.	1996	100	Achievement failure, social rejection	Standardized imagery	HR			30
5	Aue et al.	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, T_i/T_{tot}, RD	nSRR, SCR	6
8	Baldaro et al.	1996	30	Fear	Film clips	HR			120
9	Baldaro et al.	2001	42	Disgust	Film clips	HR, HRV (RSA (Porges))	RR		600
10	Bernat et al.	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 et al.	2006	42	Anxiety	Threat of shock	HR, TWA, HRV (RSA (HF), RSA (Porges), LF/HF, LF, VLF), FPTT, FPA	RR, T_i, T_e, P_i, P_e, T_i/T_{tot}, V_t, V_m, V_i/T_i, respiratory variability, pCO_2, sigh frequency, sigh V_t, % thoracic V_t	SRA, nSRR, SCL	300

Table A.1 (*Continued*)

No.	Authors	Year	*N*	Emotion labels	Experimental paradigm	Cardiovascular	Respiratory	Electrodermal	Averaging period (in s)
13	Bloom and Trautt	1977	64	Anxiety	Threat of shock	HR, FPA			30
14	Boiten	1996	16	Anger, disgust, fear, happiness, sadness, surprise	Directed facial action	HR	T_{tot}, T_i, T_e, P_i, V_t, V_m, FRC		10, 30
15	Boiten	1998	27	Amusement, disgust, fear, suspense, tenderness	Film clips		T_{tot}, T_i, T_e, P_i, P_e, T_i/T_{tot}, V_t, V_m, V_t/T_i, RC/V_t, respiratory variability		120
16	Bradley et al.	2001	95	Disgust	Picture viewing (IAPS)	HR		SCR	0.5
17	Bradley et al.	2008	49 (control)	Dental anxiety	Threat of shock	HR		SCR	20
18	Britton et al.	2006	40	Appetite, disgust, amusement, sadness	Film clips	HR		SCR	30, 90
19	Brown et al.	1993	16	Elation, sadness	Velten method	HR, SBP, DBP, MAP			
20	Chan and Lovibond	1996	23 22 (Control)	Threat	Threat of shock			SCL	40
21	Christie and Friedman	2004	34	Amusement, anger, contentment, disgust, fear, sadness	Film clips	IBI, HRV (MSD), SBP, DBP, MAP		SCL	60
22	Codispoti and De Cesarei	2007	50	Disgust, sexual arousal, threat	Picture viewing (IAPS)	HR		SCR	0.5
23	Codispoti et al.	2008	55	Disgust, sexual arousal	film clips	HR, HRV (RSA (Porges))		SCL	60
24	Collet et al.	1997	30	Anger, disgust, fear, happiness, sadness, surprise	Picture viewing (faces)	SBF, palm temp.	RR	OPD, SYDER, SCR, duration	0.5
25	Davidson and Schwartz	1976	20	Anger, relaxation	Personalized recall	HR			120
26	Demaree et al.	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 and 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
30	Dudley	1964	10	Anger, anxiety, depression, relaxation	Hypnosis		RR, V_m, pCO_2		
31	Eisenberg et al.	1988	82	Anxiety, sadness, sympathy	Film clips	HR			0.5, 3.5
32	Ekman et al.	1983	16	Anger, disgust, fear, sadness, surprise	Directed facial action, personalized recall	HR, FT		SCL	10, 30
33	Etzel et al.	2006	13 (18)	Fear, happiness, sadness	Musical excerpts	HR, HRV (SDNN, SDSD, RSA (peak-valley))	T_{tot}, T_i, T_e, respiratory variability		1, 5, 65
34	Feleky	1916	6	Anger, disgust, fear, hatred, laughter, pleasure, wonder	Personalized recall		(RR), I/E ratio, RD, RD/T_{tot}		
35	Fiorito and Simons	1994	31 (control)	Anger, contentment, fear, joy, sadness, sexual arousal	Standardized imagery, personalized recall	HR		nSRR	20
36	Foster et al.	1999	36	Anger	Real-life, standardized imagery, personalized recall	HR		SCL	30?
37	Foster and Webster	2001	10	Anger, mirth	personalized recall	HR		SCL	30
38	Foster et al.	2003	23	Mirth	Real-life, standardized imagery, personalized recall	HR		SCL	30?
39	Fredrickson and Levenson	1998	60 72	Fear, sadness	Film clips	HR, FPTT, EPTT, FPA			120
40	Funkenstein et al.	1954	69	Anger	Real-life (harassment)	HR, SV, CO, SBP, DBP, TPR			inst.
41	Gehricke and Fridlund	2002	20	Happiness, sadness	Standardized imagery	HR		SCL	60
42	Gilissen et al.	2008	78 92	Fear	Film clips	HRV (RMSSD)		SCL	60
43	Gilissen et al.	2007	78	Fear	Film clips	HRV (RMSSD)		SCL	60
44	Gross	1998	120	Disgust	Film clips	IBI, FPA, FT		SCL	1, 60
45	Gross et al.	1994	150	Sadness	Film clips	HR, FPA, FPTT, EPTT, FT	RP, RD	SCL	~100
46	Gross and Levenson	1993	43 42	Disgust	Film clips	HR, FPA, FPTT, EPTT, FT	RP, RD	SCL	1, 60

Table A.1 (*Continued*)

No.	Authors	Year	*N*	Emotion labels	Experimental paradigm	Cardiovascular	Respiratory	Electrodermal	Averaging period (in s)
47	Gross and Levenson	1997	180	Amusement, sadness	Film clips	IBI, FPA, FPTT, EPTT, FT	RP, RD	SCL	210
48	Grossberg and Wilson	1968	18 10 (control)	Fear	Adapted standardized imagery	HR		SCL	25
49	Gruber et al.	2008	54 (control)	Disgust, happiness, pride, sadness	Film clips	HR, HRV (RMSSD)		SCL	90
50	Guliani et al.	2008	16	Amusement	Film clips	HR, SV, MAP, FPA, FPTT, EPTT, FT	RR	SRA	10, 20
51	Hamer et al.	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 et al.	2000	30	Boredom, excitement, humor	Film clips	HR, PEP, SV, CO, SBP, DBP, MAP, TPR			60
54	Herrald and Tomaka	2002	109	Anger, pride, shame	Real-life (harassment, humiliation, praise)	HR, PEP, SV, CO, SBP, DBP, MAP, TPR			180
55	Hess et al.	1992	28	Anger, happiness, peacefulness, sadness	Feel emotion, express emotion without feeling, express and feel emotion	HR		SCL	30
56	Hofmann et al.	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 and de Jong-Meyer	1990	24	Amusement, suspense	Film clips	HR	RR	nSRR, SCL	30
58	Hubert and de Jong-Meyer	1991	20	Amusement, suspense	Film clips	HR		nSRR, SCL	60
59	Jönsson and Sonnby-Borgström	2003	53	Anger, happiness	picture viewing (faces)	HR, HRV (RSA (HF))			0.5, 300
60	Kaiser and Roessler	1970	20	Disgust	Film clips			SRA, nSRR	770
61	Khalfa et al.	2008	50	Happiness, sadness	Musical excerpts	HR, SBP, DBP	RR	nSRR	15
62	Klorman et al.	1977	42 (control)	Disgust, humor	Picture viewing	HR	RR, T_i, T_e	SCR, SCL, decay	1
63	Kornreich et al.	1998	14 (control)	Anger, disgust, amusement, sadness	Film clips	HR, HRV (SDNN)			300
64	Kreibig et al.	2007	34	Fear, sadness	Film clips	HR, TWA, HRV (RSA (HF), LF), PEP, LVET, HI, SV, SBP, DBP, EPA, EPTT, FT	RR, T_i/T_{tot}, V_t, V_i/T_i, pCO_2, respiratory variability	SRA, nSRR, SCL	600
65	Kring and Gordon, study 1	1998	43	Disgust, fear, amusement, sadness	Film clips			nSRR	300
	Kring and Gordon, study 2	1998	67	Anger, disgust, fear, happiness, sadness	Film clips			nSRR	240
66	Kring and 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 and Grühn, study 2	2005	96	Contentment, sadness	Film clips	IBI, FPA, FT	RP	SCL	540
69	Kunzmann et al.	2005	95	Contentment, disgust	Film clips	IBI, SBP, DBP, FPA, FPTT, EPTT, FT	RP, RD	SCL	60
70	Lang et al.	1993	64	Disgust, sexual arousal	Picture viewing (IAPS)	HR		SCR	0.5
71	Lavoie et al.	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 et al.	2005	92	Fear, anger, disgust	Real-life (harassment)	HR, SBP, DBP, MAP			1380 (inst.)

Table A.1 (*Continued*)

No.	Authors	Year	*N*	Emotion labels	Experimental paradigm	Cardiovascular	Respiratory	Electrodermal	Averaging period (in s)
73	Levenson et al.	1991	20 62 [a]	Anger, disgust, fear, happiness, sadness, surprise	Directed facial action, personalized recall	HR, FT		SCL	10, 15
74	Levenson et al., study 1	1990	16 [b]	Anger, disgust, fear, happiness, sadness, surprise	Directed facial action	HR, FT		SCL	10
	Levenson et al., study 2	1990	16	Anger, disgust, fear, happiness, sadness, surprise	Directed facial action	HR, FT		SCL	10
	Levenson et al. study 3	1990	30	Anger, disgust, fear, happiness, sadness, surprise	Directed facial action	HR, FT		SCL	10
75	Levenson et al.	1992	46 62 [a]	Anger, disgust, fear, happiness, sadness	Directed facial action	HR, FPA, FPTT, FT	RP, RD	SCL	10
76	Luminet et al.	2004	50	Sadness	Film clips	HR, SBP, DBP			60, 360
77	Marci et al.	2007	10	Anger, happiness, sadness	Personalized recall	IBI, HRV (RSA (HF))		SCL	60
78	Marsh et al.	2008	23 (control)	Sadness	Film clips	HRV (RSA (HF)), PEP		SCL	1, 30
79	McCaul et al., study 1	1982	27	Calmness, fear	Posed facial expressions	HR		SCL	16
	McCaul et al., study 2	1982	34	Calmness, fear, happiness	Posed facial expressions	HR		SCL	10
80	Miller et al.	1987	24 (12)	Fear, anger	Standardized imagery, personalized recall	HR	RP, RD, I/E ratio	SCL	30
81	Montoya et al.	2005	32	Anger, fear	film clips	HR, PEP, LVET, HI, SV, CO, SBP, DBP, MAP, TPR			900
82	Murakami and Ohira	2007	24	Anxiety	real-life (speech preparation)	HR, HRV (RSA (HF), LF, LF/HF)		SCL	300
83	Neumann and Waldstein	2001	42	Anger, joy, relaxation, sadness	Personalized recall	HR, PEP, LVET, SV, CO, SI, CI, SBP, DBP, MAP, TPR			180
84	Nyklicek et al.	1997	26	Agitation, happiness, sadness, serenity	Musical excerpts	IBI, HRV (RSA (peak-valley)), PEP, LVET, SV, CO, SBP, DBP, MAP, FPA, TPR	RR, T_i, T_e, P_i, P_e		180
85	Palomba et al.	2000	46	Contentment, disgust, fear	Film clips	HR, HRV (RSA (peak-valley)), TWA	RR	SCL	33
86	Palomba et al.	1993	15	Fear, sadness	Film clips	HR	RR		60
87	Pauls and Stemmler	2003	78	Anger, fear	Real-life (harassment, speech preparation)	HR, HRV (RMSSD), PEP, SBP, DBP, TPR		nSRR	60
88	Prkachin et al.	2001	50	Anger	Real-life (anger interview, harassment)	HR, SV, CO, SBP, DBP, TPR			300
89	Prkachin et al.	1999	31	Anger, disgust, fear, happiness, sadness	Personalized recall	HR, SV, CO, SBP, DBP, FT, TPR			300 (inst./16)
90	Rainville et al.	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 and Kagan	1996	32	Anxiety, fear, happiness, performance anxiety	Real-life (test situation, personal questions), film clips	FT			Inst.
92	Ritz et al.	2000	12	Depression, happiness	Picture viewing (IAPS), Velten method	IBI, HRV (RSA (peak-valley))	T_{tot}, T_i, T_e, P_e, T_i/T_{tot}, V_t, V_i/T_i, V_m, FRC, R_{os}		12
93	Ritz et al.	2000	24 (control)	Anxiety, anger, contentment, depression, amusement	Film clips, math task, picture viewing	HR, SBP, DBP	RR, V_t, V_m, R_{os}	SCL	180
94	Ritz et al.	2005	30 (control)	Anxiety, contentment, depression, disgust, happiness, sexual arousal	Picture viewing (IAPS)	IBI, HRV (RSA (peak-valley))	T_{tot}, T_i, T_e, P_e, T_i/T_{tot}, V_t, V_t/T_i, V_m, R_{os}	SCR	1, 15
95	Ritz et al.	2005	14 (control)	Disgust	Film clips	HR, SBP, DBP	pCO_2, peak HV, tonic HV		0.5, 10, 240
96	Roberts and Weerts	1982	16	Anger, fear	Adapted standardized imagery	HR, SBP, DBP			inst., 30
97	Robin et al.	1998	44	Dental anxiety	Odorants	SBF, palm temp.	RR	OPD, SYDER, SCR, duration	0.5

Table A.1 (*Continued*)

No.	Authors	Year	*N*	Emotion labels	Experimental paradigm	Cardiovascular	Respiratory	Electrodermal	Averaging period (in s)
98	Rochman and Diamond	2008	27 36	Anger, sadness	Personalized recall	HRV (RSA (HF)), FT		SCL	120
99	Rohrmann and 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 et al.	2005	26 (control)	Happiness, sadness	Film clips, standardized imagery, personalized recall	HR, FPTT	RR	nSRR	180
101	Rottenberg et al.	2002	33 (control)	Sadness	Film clips	HR, FT	RR	nSRR, SCL	180
102	Rottenberg et al.	2002	33 (control)	Amusement, fear, sadness	Film clips	HR		nSRR	180
103	Rottenberg et al.	2003	31	Sadness	Film clips	IBI, HRV (RSA (HF))	RR, V_t		180
104	Schachter	1957	15 (48)	Anger, fear	Real-life (harassment, threat of short-circuit)	HR, SI, CI, SBP, DBP, TPR, FT	T_i/T_{tot}	SCR	6
105	Schwartz et al.	1981	32	Anger, fear, happiness, relaxation, sadness	Personalized recall, step walking	HR, SBP, DBP			15
106	Sinha et al.	1992	27	Anger, fear, joy, sadness	Personalized recall	HR, PEP, LVET, HI, SV, CO, SBP, DBP, MAP, TPR			30
107	Sinha and 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 et al.	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
111	Stemmler et al.	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 and Schmidt	2006	20 (control)	Anger, fear, happiness, sadness	Film clips	HR, HRV (SDNN)			60
114	Tourangeau and Ellsworth	1979	123	Fear, sadness	Film clips	HR	RR	nSRR, SCL	5
115	Tsai et al.	2002	98	Anger, disgust, happiness, love, pride, sadness	Personalized recall			SCL	120
116	Tsai et al.	2000	96	Amusement, sadness	Film clips	IBI, FPA, FPTT, EPTT, FT	RP	SCL	120
117	Tsai et al.	2003	10 (control)	Amusement, sadness	Film clips	IBI, FPA, FPTT, FT		SCL	180
118	Tugade and 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 et al.	2001	40	Depression, fear, pleasure, relaxation	Standardized imagery		T_i, T_e, V_t, pCO_2		60

Table A.1 (*Continued*)

No.	Authors	Year	*N*	Emotion labels	Experimental paradigm	Cardiovascular	Respiratory	Electrodermal	Averaging period (in s)
121	Van Diest et al.	2006	98 [c]	Anxiety, fear	Standardized imagery		Respiratory variability: T_i, T_e, V_i, V_i/T_i, pCO_2		30, 90
122	Van Diest et al.	2001	40	Depression, desire, fear, relaxation	Standardized imagery	HR	RR, T_i, T_e, V_t, pCO_2		90
123	Van Egeren et al.	1978	28	Anger	Real-life (harassment)	HR, SBP, DBP, FPA, FPTT			6
124	Van Oyen Witvliet and Vrana	1995	48	Fear, joy, relaxation, sadness	standardized imagery	HR		SCL	8
125	Van Reekum et al.	2004	33	Goal conduciveness, intrinsic pleasantness	Computer game	IBI, FPTT, FT slope		SCR	5, 3
126	Vianna and Tranel	2006	16	Disgust, fear, happiness, sadness	Film clips	HRV (RSA (HF), LF)		SCL	120
127	Vlemincx et al.	2009	36	Threat, relief, relaxation	Threat of shock		RR, V_i, V_e, sigh frequency		10
			42						10
			37						5–50
128	Vrana	1993	50	Anger, disgust, joy, pleasure	Standardized imagery	HR		SCL	4
129	Vrana and Gross	2004	9 (control)	Anger, joy	Picture viewing (faces)	HR		SCL	8
130	Vrana and Rollock	2002	112	Anger, fear, joy	Standardized imagery	HR, SBP, DBP, MAP		SCL	30
131	Waldstein et al.	2000	30	Anger, happiness	Film clips, personalized recall	HR, SBP, DBP			Inst., 120–180
132	Williams et al.	2005	13	Anger, disgust, fear	Picture viewing (faces)			nSRR, SCR, latency, rise time, recovery time	0.5
133	Winton et al.	1984	20	Disgust, sexual arousal	Picture viewing	HR		SCR	1, 12
134	Yogo et al.	1995	24	Anger, joy	Standardized imagery	SBP, DBP, MAP			30

[a] Comparison group based on pooled sample from Levenson et al. (1990).
[b] Same sample as Ekman et al. (1983).
[c] Includes participants from Van Diest et al. (2001).

References

Adamson, J.D., Romano, K.R., Burdick, J.A., Corman, C.L., Chebib, F.S., 1972. Physiological responses to sexual and unpleasant film stimuli. Journal of Psychosomatic Research 16, 153–162.

Adsett, C.A., Schottsteadt, W.W., Wolf, S.G., 1962. Changes in coronary blood flow and other hemodynamic indicators induced by stressful interviews. Psychosomatic Medicine 24, 331–336.

Alaoui-Ismaïli, O., Robin, O., Rada, H., Dittmar, A., Vernet-Maury, E., 1997. Basic emotions evoked by odorants: comparison between autonomic responses and self-evaluation. Physiology and Behavior 62, 713–720.

Allen, N.B., de, L., Horne, D.J., Trinder, J., 1996. Sociotropy, autonomy, and dysphoric emotional responses to specific classes of stress: a psychophysiological evaluation. Journal of Abnormal Psychology 105 (1), 25–33.

Angell, J.R., 1916. A reconsideration of James's theory of emotion in the light of recent criticism. Psychological Review 23, 251–261.

Aristotle, Peters, F.H., 1893. The Nichomachean Ethics, 5th ed. Kegan Paul, Trench & Co., London.

Averill, J.R., 1968. Grief: its nature and significance. Psychological Bulletin 70, 721–748.

Aue, T., Flykt, A., Scherer, K.R., 2007. First evidence for differential and sequential efferent effects of stimulus relevance and goal conduciveness appraisal. Biological Psychology 74, 347–357.

Averill, J.R., 1969. Autonomic response patterns during sadness and mirth. Psychophysiology 5, 399–414.

Ax, A.F., 1953. The physiological differentiation between fear and anger in humans. Psychosomatic Medicine 15, 433–442.

Baldaro, B., Battachi, M.W., Codispoti, M., Tuozzi, G., Trombini, G., Bolzani, R., Palomba, D., 1996. Modifications of electrogastrographic activity during the viewing of brief film sequences. Perceptual and Motor Skills 82, 1243–1250.

Baldaro, B., Mazzetti, M., Codispoti, M., Tuozzi, G., Bolzani, R., Trombini, G., 2001. Autonomic reactivity during viewing of an unpleasant film. Perceptual and Motor Skills 93, 797–805.

Bandler, R., Keay, K.A., Floyd, N., Price, J., 2000. Central circuits mediating patterned autonomic activity during active vs. passive emotional coping. Brain Research Bulletin 53 (1), 95–104.

Bandler, R., Shipley, M.T., 1994. Columnar organization in the midbrain periaqueductal gray: modules for emotional expression? Trends in Neurosciences 17, 379–389.

Barlow, D.H., 1991. Disorders of emotion. Psychological Inquiry 2, 58–71.

Barr-Zisowitz, C., 2000. "Sadness"—is there such a thing? In: Lewis, R., Haviland-Jones, J.M. (Eds.), Handbook of Emotions, 2nd ed. Guilford Press, New York, pp. 607–622.

Beauchaine, T., 2001. Vagal tone, development, and Gray's motivational theory: toward an integrated model of autonomic nervous system functioning in psychopathology. Development and Psychopathology 13, 183–214.

Benarroch, E.E., 1993. The central autonomic network: functional organization, dysfunction, and perspective. Mayo Clinic Proceedings 68, 988–1001.

Benarroch, E.E., 1999. Functional anatomy of the central autonomic nervous system. In: Appenzeller, O. (Ed.), Handbook of Clinical Neurology: vol. 74, The Autonomic Nervous System. Part 1. Normal Functions. Elsevier Science, Amsterdam, The Netherlands, pp. 53–86.

Bernard, J.F., Bandler, R., 1998. Parallel circuits for emotional coping behaviour: new pieces in the puzzle. The Journal of Comparative Neurology 401, 429–436.

Bernat, E., Patrick, C.J., Benning, S.D., Tellegen, A., 2006. Effects of picture content and intensity on affective physiological response. Psychophysiology 43, 93–103.

Berntson, G.G., Cacioppo, J.T., Quigley, K.S., 1991. Autonomic determinism: the modes of autonomic control, the doctrine of autonomic space, and the laws of autonomic constraint. Psychological Review 98, 459–487.

Berntson, G.G., Cacioppo, J.T., Quigley, K.S., 1993. Cardiac psychophysiology and autonomic space in humans: empirical perspectives and conceptual implications. Psychological Bulletin 114 (2), 296–322.

Berridge, K.C., 1999. Pleasure, pain, desire, and dread: hidden core processes of emotion. In: Kahneman, D., Diener, E., Schwarz, N. (Eds.), Well-being: The Foundations of Hedonic Psychology. Russell Sage Foundation, New York, pp. 527–559.

Blascovich, J., Katkin, E.S. (Eds.), 1993. Cardiovascular Reactivity to Psychological Stress and Disease. American Psychological Association, Washington, DC.

Blascovich, J., Mendes, W.B., Tomaka, J., Salomon, K., Seery, M., 2003. The robust nature of the biopsychosocial model challenge and threat: a reply to Wrigth and Kirby. Personality and Social Psychology Review 7, 234–243.

Blatz, W.E., 1925. The cardiac, respiratory, and electrical phenomena involved in the emotion of fear. Journal of Experimental Psychology 8 (2), 109–132.

Blechert, J., Lajtman, M., Michael, T., Margraf, J., Wilhelm, F.H., 2006. Identifying anxiety states using broad sampling and advanced processing of peripheral physiological information. Biomedical Sciences Instrumentation 42, 136–141.

Bloom, L.J., Trautt, G.M., 1977. Finger pulse volume as a measure of anxiety: further evaluation. Psychophysiology 14, 541–544.

Boiten, F.A., 1996. Autonomic response patterns during voluntary facial action. Psychophysiology 33, 123–131.

Boiten, F.A., 1998. The effects of emotional behaviour on components of the respiratory cycle. Biological Psychology 48, 29–51.
Bradley, M.M., Codispoti, M., Cuthbert, B.N., Lang, P.J., 2001. Emotion and motivation I. Defensive and appetitive reactions in picture processing. Emotion 1, 276–298.
Bradley, M.M., Lang, P.J., 2000. Measuring emotion: behavior, feeling and physiology. In: Lane, R., Nadel, L. (Eds.), Cognitive Neuroscience of Emotion. Oxford University Press, New York, pp. 242–276.
Bradley, M.M., Silakowski, T., Lang, P.J., 2008. Fear of pain and defensive activation. Pain 137, 156–163.
Brehm, J.W., 1999. The intensity of emotion. Personality and Social Psychology Review 3, 2–22.
Brener, J., 1987. Behavioural energetics: some effects of uncertainty on the mobilization and distribution of energy. Psychophysiology 24, 499–512.
Britton, J.C., Taylor, S.F., Berridge, K.C., Mikels, J.A., Liberzon, I., 2006. Differential subjective and psychophysiological responses to socially and nonsocially generated emotional stimuli. Emotion 6, 150–155.
Brown, T.M., Fee, E., 2002. Walter Bradford Cannon: pioneer physiologist of huamn emotions. American Journal of Public Health 92, 1594–1595.
Brown, W.A., Sirota, A.D., Niaura, R., Engebretson, T.O., 1993. Endocrine correlates of sadness and elation. Psychosomatic Medicine 55, 458–467.
Cacioppo, J.T., Berntson, G.G., Klein, D.J., Poehlmann, K.M., 1997. The psychophysiology of emotion across the lifespan. Annual Review of Gerontology and Geriatrics 17, 27–74.
Cacioppo, J.T., Berntson, G.G., Larsen, J.T., Poehlmann, K.M., Ito, T.A., 2000. The psychophysiology of emotion. In: Lewis, R., Haviland-Jones, J.M. (Eds.), The Handbook of Emotion, 2nd ed. Guilford Press, New York, pp. 173–191.
Cannon, W.B., 1915. Bodily Changes in Pain, Hunger, Fear, and Rage. Appleton & Company, New York.
Cannon, W.B., 1927. The James-Lange theory of emotions: a critical examination and an alternative theory. American Journal of Psychology 39, 106–124.
Cannon, W.B., 1939. The Wisdom of the Body. Norton, New York.
Carver, C.S., 2001. Affect and the functional bases of behavior: on the dimensional structure of affective experience. Personality and Social Psychology Review 5, 345–356.
Chan, K.Y.C., Lovibond, P.F., 1996. Expectancy bias in trait anxiety. Journal of Abnormal Psychology 105, 637–647.
Christie, I., Friedman, B., 2004. Autonomic specificity of discrete emotion and dimensions of affective space: a multivariate approach. International Journal of Psychophysiology 51, 143–153.
Cloninger, C., 1987. A systematic method for clinical depression and classification of personality variants. Archives of General Psychiatry 44, 573–588.
Codispoti, M., De Cesarei, A., 2007. Arousal and attention: picture size and emotional reactions. Psychophysiology 44, 680–686.
Codispoti, M., Surcinelli, P., Baldaro, B., 2008. Watching emotional movies: affective reactions and gender differences. International Journal of Psychophysiology 69, 90–95.
Cohen, R.A., Coffman, J.D., 1981. Beta-adrenergic vasodilator mechanism in the finger. Circulation Research 49, 1196–1201.
Collet, C., Vernet-Maury, E., Delhomme, G., Dittmar, A., 1997. Autonomic nervous system response patterns specificity to basic emotions. Journal of Autonomic Nervous System 62, 45–57.
Contrada, R., 1992. T-wave amplitude: on the meaning of a psychophysiological index. Biological Psychology 33, 249–258.
Corr, P.J. (Ed.), 2008. The Reinforcement Sensitivity Theory of Personality. Cambridge University Press, Cambridge.
Craske, M.G., 1999. Anxiety Disorders: Psychological Approaches to Theory and Treatment. Westview Press, Boulder, CO.
Dale, H.H., 1947. Walter Bradford Cannon. 1871–1945. Obituary Notices of Fellows of the Royal Society 5, 407–423.
Damasio, A.R., 1998. Emotion in the perspective of an integrated nervous system. Brain Research Reviews 26, 83–86.
Damasio, A.R., 1999. The Feeling of What Happens: Body, Emotion and the Making of Consciousness. Harcourt Brace, Orlando, FL.
Davidson, R.J., 1998. Affective style and affective disorders: perspectives from affective neuroscience. Cognition and Emotion 12, 307–330.
Davidson, R.J., Scherer, K.R., Goldsmith, H.H. (Eds.), 2003. Handbook of Affective Sciences. Oxford University Press, New York.
Davidson, R.J., Schwartz, G.E., 1976. Patterns of cerebral lateralization during cardiac biofeedback versus the self-regulation of emotion: sex differences. Psychophysiology 13, 62–68.
Demaree, H., Schmeichel, B., Robinson, J., Everhart, D.E., 2004. Behavioural, affective, and physiological effects of negative and positive emotional exaggeration. Cognition and Emotion 18, 1079–1097.
Dimberg, U., 1986. Facial reactions to fear-relevant and fear-irrelevant stimuli. Biological Psychology 23, 153–161.
Dimberg, U., Thunberg, M., 2007. Speech anxiety and rapid emotional reactions to angry and happy facial expressions. Scandinavian Journal of Psychology 48, 321–328.
Drummond, P.D., 1999. Facial flushing during provocation in women. Psychophysiology 36, 325–332.
Dudley, D.L., Martin, C.J., Holmes, T.H., 1964. Psychophysiological studies of pulmonary ventilation. Psychosomatic Medicine 26, 645–660.
Eisenberg, N., Fabes, R.A., Bustamante, D., Mathy, R.M., Miller, P.A., Lindholm, E., 1988. Differentiation of vicariously induced emotional reactions in children. Developmental Psychology 24, 237–246.
Ekman, P., 1984. Expression and the nature of emotion. In: Scherer, K.R., Ekman, P. (Eds.), Approaches to emotion. Erlbaum, Hillsdale, NJ, pp. 319–343.
Ekman, P., 1994. All emotions are basic. In: Ekman, P., Davidson, R.J. (Eds.), The Nature of Emotion: Fundamental Questions. Oxford University Press, New York and Oxford, pp. 15–19.
Ekman, P., 1997. Emotion families. In: Rauch, I., Carr, G.F. (Eds.), Semiotics Around the World: Synthesis in Diversity. Mouton de Gruyter, Berlin/New York, pp. 191–193.
Ekman, P., 1999. Basic emotions. In: Dalgleish, T., Power, M.J. (Eds.), The Handbook of Cognition and Emotion. John Wiley & Sons, Ltd., Sussex, UK, pp. 45–60.
Ekman, P., Levenson, R.W., Friesen, W.V., 1983. Autonomic nervous system activity distinguishes among emotions. Science 221, 1208–1210.
Ellsworth, P.C., 1994. William James and emotion: is a century of fame worth a century of misunderstanding? Psychological Review 101, 222–229.
Ellsworth, P.C., Scherer, K.R., 2003. Appraisal processes in emotion. In: Davidson, R.J., Goldsmith, H., Scherer, K.R. (Eds.), Handbook of Affective Sciences. Oxford University Press, New York, pp. 572–595.
Epstein, S., Boudreau, L., Kling, S., 1975. Magnitude of the heart rate and electrodermal response as a function of stimulus input, motor output, and their interaction. Psychophysiology 12 (1), 15–24.
Etzel, J.A., Johnsen, E.L., Dickerson, J.A., Tranel, D., Adolphs, R., 2006. Cardiovascular and respiratory responses during musical mood induction. International Journal of Psychophysiology 61, 57–69.
Fanselow, M.S., 1994. Neural organization of the defensive behavior system responsible for fear. Psychonomic Bulletin and Review 1, 429–438.
Fehr, F.S., Stern, J.A., 1970. Peripheral physiological variables and emotion: the James–Lange theory revisited. Psychological Bulletin 74, 411–424.
Feldman, J.L., Ellenberger, H.H., 1988. Central coordination of respiratory and cardiovascular control in mammals. Annual Review of Physiology 50, 593–606.
Feldman-Barrett, L., 2006. Are emotions natural kinds? Perspectives on Psychological Science 1, 28–58.
Feleky, A., 1916. The influence of the emotions on respiration. Journal of Experimental Psychology 1 (3), 218–241.
Fiorito, E.R., Simons, R.F., 1994. Emotional imagery and physical anhedonia. Psychophysiology 31, 513–521.
Flykt, A., Esteves, F., Öhman, A., 2007. Skin conductance responses to masked conditioned stimuli: phylogenetic/ontogenetic factors versus direction of threat? Biological Psychology 74, 328–336.
Folkow, B., 2000. Perspectives on the integrative functions of the 'sympatho-adrenomedullary system'. Autonomic Neuroscience: Basic and Clinical 83, 101–115.
Foster, P.S., Smith, E.W.L., Webster, D.G., 1999. The psychophysiological differentiation of actual, imagined, and recollected anger. Imagination, Cognition and Personality 18, 189–203.
Foster, P.S., Webster, D.G., 2001. Emotional memories: the relationship between age of memory and the corresponding psychophysiological responses. International Journal of Psychophysiology 41, 11–18.
Foster, P.S., Webster, D.G., Williamson, J., 2003. The psychophysiological differentiation of actual, imagined, and recollected mirth. Imagination, Cognition and Personality 22, 163–180.
Fowles, D.C., 1980. The three arousal model: implications of Gray's two-factor learning theory for heart rate, electrodermal activity, and psychopathy. Psychophysiology 17 (2), 87–104.
Fredrickson, B.L., Levenson, R.W., 1998. Positive emotions speed recovery from the cardiovascular sequelae of negative emotions. Cognition and Emotion 12, 191–220.
Fredrikson, M., Furmark, T., Olsson, M.T., Fischer, H., Långström, J.A.B., 1998. Functional neuroanatomical correlates of electrodermal activity: a positron emission tomographic study. Psychophysiology 35, 179–185.
Friedman, B.H., this issue. Feelings and the body: the Jamesian perspective on autonomic specificity of emotion.
Frijda, N.H., 1986. The Emotions. Cambridge University Press, Cambridge.
Frijda, N.H., 1993. Moods, emotion episodes, and emotions. In: Lewis, M., Haviland, J.M. (Eds.), Handbook of Emotions. Guilford Press, New York, pp. 381–403.
Funkenstein, D.R., King, S.R., Drolette, M., 1954. The direction of anger during a laboratory stress-inducing situation. Psychosomatic Medicine 16, 404–413.
Furedy, J.J., Heslegrave, R.J., Scher, H., 1992. T-wave amplitude utility revisited: some physiological and psychophysiological considerations. Biological Psychology 33, 241–248.
Gehricke, J.-G., Fridlund, A.J., 2002. Smiling, frowning, and autonomic activity in mildly depressed and nondepressed men in response to emotional imagery of social contexts. Perceptual and Motor Skills 94, 141–151.
Gellhorn, E., 1964. Motion and emotion: the role of proprioception in the physiology and pathology of the emotions. Psychological Review 71, 457–472.
Gellhorn, E., 1965. The neurophysiological basis of anxiety: a hypothesis. Perspectives in Biology and Medicine 8, 488–515.
Gellhorn, E., 1970. The emotions and the ergotropic and trophotropic systems. Psychologische Forschung 34, 48–94.
Gendolla, G.H.E., 2000. On the impact of mood on behavior: an integrative theory and a review. Review of General Psychology 4, 378–408.
Gendolla, G.H.E., 2003. Mood effects on effort mobilization in learning: theory and experimental evidence. In: Mayring, P., von Rhöneck, C. (Eds.), Learning Emotions: The Influence of Affective Factors on Classroom Learning. Verlag Peter Lang, Bern, Switzerland, pp. 29–46.
Gendolla, G.H.E., 2004. The intensity of motivation when the self is involved: an application of Brehm's theory of motivation to effort-related cardiovascular

response. In: Wright, R.A., Greenberg, S.S. (Eds.), Motivational Analyses of Social Behavior. Erlbaum, Mahwah, NJ, pp. 205–244.
Gendolla, G.H.E., Brinkmann, K., 2005. The role of mood states in self-regulation: effects on action preferences and resource mobilization. European Psychologist 10, 187–198.
Gendolla, G.H.E., Krüsken, J., 2001. The joint impact of mood state and task difficulty on cardiovascular and electrodermal reactivity in active coping. Psychophysiology 38, 548–556.
Gendolla, G.H.E., Krüsken, J., 2002. Mood state, task demand, and effort-related cardiovascular response. Cognition and Emotion 16 (5), 577–603.
Gendolla, G.H.E., Wright, R.A., 2005. Motivation in social settings: studies of effort-related cardiovascular arousal. In: Forgas, J.P., Williams, K., von Hippel, W. (Eds.), Social Motivation. Cambridge University Press, New York, pp. 71–90.
Gilissen, R., Bakermans-Kraneburg, M.J., van Ijzendoorn, M.H., van der Veer, R., 2008. Parent–child relationship, temperament, and physiological reactions to fear-inducing film clips: further evidence for differential susceptibility. Journal of Experimental Child Psychology 99, 182–195.
Gilissen, R., Koolstra, C.M., van Ijzendoorn, M.H., Bakermans-Kraneburg, M.J., van der Veer, R., 2007. Physiological reactions of preschoolers to fear-inducing film clips: effects of temperamental fearfulness and quality of the parent–child relationship. Developmental Psychobiology 49, 187–195.
Gray, J.A., 1982. The Neuropsychology of Anxiety. Oxford University Press, New York.
Gray, J.A., 1987. The Psychology of Fear and Stress. Cambridge University Press, New York.
Gray, J.A., McNaughton, N., 2000. The Neuropsychology of Anxiety: An Enquiry Into the Functions of the Septo-hippocampal System. Oxford University Press, Oxford.
Gross, J.J., 1998. Antecedent- and response-focused emotion regulation: divergent consequences for experience, expression, and physiology. Journal of Personality and Social Psychology 74, 224–237.
Gross, J.J., Fredrickson, B.L., Levenson, R.W., 1994. The psychophysiology of crying. Psychophysiology 31, 460–468.
Gross, J.J., Levenson, R.W., 1993. Emotional suppression: physiology, self-report, and expressive behavior. Journal of Personality and Social Psychology 64, 970–986.
Gross, J.J., Levenson, R.W., 1997. Hiding feelings: the acute effects of inhibiting negative and positive emotion. Journal of Abnormal Psychology 106, 95–103.
Grossberg, J.M., Wilson, H.K., 1968. Physiological changes accompanying the visualization of fearful and neutral situations. Journal of Personality and Social Psychology 10, 124–133.
Grossman, P., Taylor, E.W., 2007. Toward understanding respiratory sinus arrhythmia: relations to cardiac vagal tone, evolution, and biobehavioral functions. Biological Psychology 74, 263–285.
Gruber, J., Johnson, S.L., Oveis, C., Keltner, D., 2008. Risk for mania and positive emotional responding: too much of a good thing? Emotion 8 (1), 23–33.
Guliani, N.R., McRae, K., Gross, J.J., 2008. The up- and down-regulation of amusement: experiential, behavioral, and autonomic consequences. Emotion 8, 714–719.
Hamer, M., Tanaka, G., Okamura, H., Tsuda, A., Steptoe, A., 2007. The effects of depressive symptoms on cardiovascular and catecholamine responses to the induction of depressive mood. Biological Psychology 74, 20–25.
Harmon-Jones, E., Gable, P.A., Peterson, C.K., this issue. The role of asymmetric frontal cortical activity in emotion-related phenomena: a review and update.
Harris, C.R., 2001. Cardiovascular responses of embarrassment and effects of emotional suppression in a social setting. Journal of Personality and Social Psychology 81, 886–897.
Harrison, L., Carroll, D., Burns, V., Corkill, A., Harrison, C., Ring, C., Drayson, M., 2000. Cardiovascular and secretory immunoglobulin A reactions to humorous, exciting, and didactic film presentations. Biological Psychology 52, 113–126.
Herrald, M.M., Tomaka, J., 2002. Patterns of emotion-specific appraisal, coping, and cardiovascular reactivity during an ongoing emotional episode. Journal of Personality and Social Psychology 83 (2), 434–450.
Hess, U., Kappas, A., McHugo, G.J., Lanzetta, J.T., Kleck, R.E., 1992. The facilitative effect of facial expression on the self-generation of emotion. International Journal of Psychophysiology 12, 251–265.
Hess, W.R., 1957. Functional Organization of the Diencephalon. Grune & Stratton, New York.
Hilton, S.M., 1975. Ways of viewing the central nervous control of the circulation: old and new. Brain Research 87, 213–219.
Hofmann, S.G., Moscovitch, D.A., Kim, H.J., 2006. Autonomic correlates of social anxiety and embarrassment in shy and non-shy individuals. International Journal of Psychophysiology 61, 134–142.
Hubert, W., de Jong-Meyer, R., 1990. Psychophysiological response patterns to positive and negative film stimuli. Biological Psychology 31, 73–93.
Hubert, W., de Jong-Meyer, R., 1991. Autonomic, neuroendocrine, and subjective responses to emotion-inducing film stimuli. International Journal of Psychophysiology 11, 131–140.
Izard, C.E., 1992. Basic emotions, relations among emotions, and emotion–cognition relations. Psychological Review 99, 561–565.
James, W., 1884. What is an emotion? Mind 9, 188–205.
James, W., 1890. What is an emotion? In: James, W. (Ed.), The Principles of Psychology. Dover, New York, pp. 442–485.
James, W., 1894. Discussion: The Physical Basis of Emotion. Psychological Review 1, 516–529.
Jänig, W., 2003. The autonomic nervous system and its coordination by the brain. In: Davidson, R.J., Scherer, K.R., Goldsmith, H.H. (Eds.), Handbook of Affective Sciences. Oxford University Press, New York, pp. 135–186.
Jänig, W., Häbler, H.-J., 2000. Specificity in the organization of the autonomic nervous system: a basis for precise neural regulation of homeostatic and protective body functions. Progress in Brain Research 122, 351–367.
Jänig, W., McLachlan, E.M., 1992a. Characteristics of function-specific pathways in the sympathetic nervous system. Trends in Neurosciences 15, 475–481.
Jänig, W., McLachlan, E.M., 1992b. Specialized functional pathways are the building blocks of the autonomic nervous system. Journal of the Autonomic Nervous System 41, 3–13.
Jönsson, P., Sonnby-Borgström, M., 2003. The effects of pictures of emotional faces on tonic and phasic autonomic cardiac control in women and men. Biological Psychology 62 (2), 157–173.
Kaiser, C., Roessler, R., 1970. Galvanic skin responses to motion pictures. Perceptual and Motor Skills 30, 371–374.
Keay, K.A., Bandler, R., 2001. Parallel circuits mediating distinct emotional coping reactions to different types of stress. Neuroscience and Biobehavioral Review 25, 669–678.
Keay, K.A., Bandler, R., 2002. Distinct central representations of inescapable and escapable pain: observations and speculation. Experimental Physiology 87.2, 275–279.
Khalfa, S., Roy, M., Rainville, P., Bella, S.D., Peretz, I., 2008. Role of tempo entrainment in psychophysiological differentiation of happy and sad music? International Journal of Psychophysiology 68 (1), 17–26.
Kiecolt-Glaser, J.K., Graham, J.E., Malarkey, W.B., Porter, K., Lemeshow, S., Glaser, R., 2008. Olfactory influences on mood and autonomic, endocrine, and immune function. Psychoneuroendocrinology 33, 328–339.
Kling, C., 1933. The role of the parasympathetics in emotions. Psychological Review 40, 368–380.
Klorman, R., Weissberg, R.P., Wiesenfeld, A.R., 1977. Individual differences in fear and autonomic reactions to affective stimulation. Psychophysiology 14, 45–51.
Koole, S., 2009. The psychology of emotion regulation: an integrative review. Cognition and Emotion 23, 4–41.
Kornreich, C., Philippot, P., Verpoorten, C., Dan, B., Baert, I., Le Bon, O., Verbanck, P., Pelc, I., 1998. Alcoholism and emotional reactivity: more heterogeneous film-induced emotional response in newly detoxified alcoholics compared to controls—a preliminary study. Addictive Behaviors 23, 413–418.
Kreibig, S.D., 2004. Situational and individual response specificity to emotional films: effects on experiential, cardiovascular, electrodermal, respiratory and muscular responses. BUMTh, University of Kiel, Kiel, Germany. Available from http://www.affective-sciences.org/node/819.
Kreibig, S.D., in press. Emotion, motivation, and cardiovascular response. In: Wright, R.A., Gendolla, G.H.E. (Eds.), Motivation Perspectives on Cardiovascular Response, American Psychological Association, Washington, DC.
Kreibig, S.D., Gendolla, G.H.E., Scherer, K.R., this issue. Psychophysiological effects of emotional responding to goal attainment.
Kreibig, S.D., Wilhelm, F.H., Gross, J.J., Roth, W.T., 2005. Specific emotional responses as deviations from the experimental context. Psychophysiology 42 (s1), S77.
Kreibig, S.D., Wilhelm, F.H., Roth, W.T., Gross, J.J., 2007. Cardiovascular, electrodermal, and respiratory response patterns to fear and sadness-inducing films. Psychophysiology 44 (5), 787–806.
Kring, A.M., Gordon, A.H., 1998. Sex differences in emotion: expression, experience, and physiology. Journal of Personality and Social Psychology 74, 686–703.
Kring, A.M., Neale, J.M., 1996. Do schizophrenic patients show a disjunctive relationship among expressive, experiential, and psychophysiological components of emotion? Journal of Abnormal Psychology 105, 249–257.
Krumhansl, C.L., 1997. An exploratory study of musical emotions and psychophysiology. Canadian Journal of Experimental Psychology 51 (4), 336–353.
Kunzmann, U., Grühn, D., 2005. Age differences in emotional reactivity: the sample case of sadness. Psychology and Aging 20, 47–59.
Kunzmann, U., Kupperbusch, C.S., Levenson, R.W., 2005. Behavioral inhibition and amplification during emotional arousal: a comparison of two age groups. Psychology and Aging 20, 144–158.
Lacey, J.I., 1967. Somatic response patterning and stress: some revisions of activation theory. In: Appley, M.H., Trumbell, R. (Eds.), Psychological Stress. Appleton-Century-Crofts, New York, pp. 14–37.
Lang, P.J., 1979. A bio-informational theory of emotional imagery. Psychophysiology 16, 495–512.
Lang, P.J., 1993. The three system approach to emotion. In: Birbaumer, N., Öhman, A. (Eds.), The Structure of Emotion. Hogrefe & Huber Publishers, Seattle, pp. 18–30.
Lang, P.J., 1994. The motivational organization of emotion: affect-reflex connections. In: VanGoozen, S., Van de Poll, N.E., Sergeant, J.A. (Eds.), Emotions: Essays on Emotion Theory. Erlbaum, Hillsdale, NJ, pp. 61–93.
Lang, P.J., Bradley, M.M., this issue. Emotion and the motivational brain.
Lang, P.J., Bradley, M.M., Cuthbert, B.N., 1990. Emotion, attention, and the startle reflex. Psychological Review 97 (3), 377–395.
Lang, P.J., Bradley, M.M., Cuthbert, B.N., 1993. Emotion, arousal, valence, and the startle reflex. In: Birbaumer, N., Öhman, A. (Eds.), The Structure of Emotion: Psychophysiological, Cognitive and Clinical Aspects. Hogrefe & Huber Publishers, Seattle, pp. 243–251.
Lang, P.J., Bradley, M.M., Cuthbert, B.N., 1997. Motivated attention: affect, activation, and action. In: Lang, P.J., Simons, R., Balaban, M.T. (Eds.), Attention and Orienting: Sensory and Motivational Processes. Lawrence Erlbaum Associates, Mahwah, NJ, pp. 97–136.
Lang, P.J., Bradley, M.M., Cuthbert, B.N., 2005. Internatinal affective picture system (IAPS): instruction manual and affective ratings (Technical Report A-6). University of Florida.

Lang, P.J., Greenwald, M.K., Bradley, M.M., Hamm, A.O., 1993. Looking at pictures: affective, facial, visceral, and behavioral reactions. Psychophysiology 30, 261–273.
Larsen, J.T., Berntson, G.G., Poehlmann, K.M., Ito, T.A., Cacioppo, J.T., 2008. The psychophysiology of emotion. In: Lewis, R., Haviland-Jones, J.M., Feldman-Barrett, L. (Eds.), The Handbook of Emotions, 3rd ed. Guilford, New York.
Lavoie, K.L., Miller, S.B., Conway, M., Fleet, R.P., 2001. Anger, negative emotions, and cardiovascular reactivity during interpersonal conflict in women. Journal of Psychosomatic Research 51, 503–512.
Lazarus, R.S., 1991. Progress on a cognitive–motivational–relational theory of emotion. American Psychologist 46 (8), 819–834.
Lerner, J.S., Gonzalez, R.M., Dahl, R.E., Hariri, A.R., Taylor, S.E., 2005. Facial expressions of emotion reveal neuroendocrine and cardiovascular stress responses. Biological Psychiatry 58, 743–750.
Levenson, R.W., 1988. Emotion and the autonomic nervous system: a prospectus for research on autonomic specificity. In: Wagner, H.L. (Ed.), Social Psychophysiology and Emotion: Theory and Clinical Applications. Wiley, Chichester, pp. 17–42.
Levenson, R.W., 2003. Autonomic specificity and emotion. In: Davidson, R.J., Scherer, K.R., Goldsmith, H.H. (Eds.), Handbook of Affective Sciences. Oxford University Press, New York, pp. 212–224.
Levenson, R.W., Carstensen, L.L., Friesen, W.V., Ekman, P., 1991. Emotion, physiology, and expression in old age. Psychology and Aging 6, 28–35.
Levenson, R.W., Ekman, P., Friesen, W.V., 1990. Voluntary facial action generates emotion-specific autonomic nervous system activity. Psychophysiology 27, 363–384.
Levenson, R.W., Ekman, P., Heider, K., Friesen, W.V., 1992. Emotion and autonomic nervous system activity in the Minangkabau of West Sumatra. Journal of Personality and Social Psychology 62, 972–988.
Lewis, M.D., Granic, I. (Eds.), 2000. Emotion, Development, and Self-organization. Cambridge University Press, New York.
Luminet, O., Rimé, B., Bagby, R.M., Taylor, G.J., 2004. A multimodal investigation of emotional responding in alexithymia. Cognition and Emotion 18, 741–766.
Marci, C.D., Glick, D.M., Loh, R., Dougherty, D.D., 2007. Autonomic and prefrontal cortex responses to autobiographical recall of emotions. Cognitive, Affective, and Behavioral Neuroscience 7 (3), 243–250.
Marsh, P., Beauchaine, T.P., Williams, B., 2008. Dissociation of sad facial expressions and autonomic nervous system responding in boys with disruptive behavior disorders. Psychophysiology 45, 100–110.
Mausfeld, R., 2003. No psychology in—no psychology out. Commentary on "Biological Psychology 2010—Visions of the future of the field in psychology". Psychologische Rundschau 54, 185–191.
Mauss, I.B., Levenson, R.W., McCater, L., Wilhelm, F.H., Gross, J.J., 2005. The tie that binds? Coherence among emotion experience, behavior, and physiology. Emotion 5, 175–190.
Mauss, I.B., Robinson, M.D., 2009. Measures of emotion: a review. Cognition and Emotion 23, 209–237.
McCaul, K.D., Holmes, D.S., Solomon, S., 1982. Voluntary expressive changes and emotion. Journal of Personality and Social Psychology 42, 145–152.
Merriam-Webster Online Dictionary, 2009. Springfield, MA: Merriam-Webster Inc. http://www.merriam-webster.com/dictionary (accessed 16.05.09).
Miller, G., Levin, D., Kozak, M., Cook, E., McLean, A., Lang, P.J., 1987. Individual differences in imagery and the psychophysiology of emotion. Cognition and Emotion 1, 367–390.
Miltner, W., Birbaumer, N., Gerber, W.-D., 1986. Verhaltensmedizin [Behavioral Medicine]. Springer, Berlin.
Montoya, P., Campos, J.J., Schandry, R., 2005. See red? Turn pale? Unveiling emotions through cardiovascular and hemodynamic changes. The Spanish Journal of Psychology 8, 79–85.
Murakami, H., Ohira, H., 2007. Influence of attention manipulation on emotion and autonomic responses. Perceptual and Motor Skills 105, 299–308.
Neumann, S., Waldstein, S.R., 2001. Similar patterns of cardiovascular response during emotional activation as a function of affective valence and arousal and gender. Journal of Psychosomatic Research 50, 245–253.
Niepel, M., 2001. Independent manipulation of stimulus change and unexpectedness dissociates indices of the orienting response. Psychophysiology 38 (1), 84–91.
Nomikos, M.S., Opton, E., Averill, J.R., Lazarus Jr., R.S., 1968. Surprise versus suspense in the production of stress reaction. Journal of Personality and Social Psychology 8, 204–208.
Nyklicek, I., Thayer, J.F., Van Doornen, L.J.P., 1997. Cardiorespiratory differentiation of musically-induced emotions. Journal of Psychophysiology 11, 304–321.
Obrist, P.A., 1981. Cardiovascular Psychophysiology. Plenum, New York.
Öhman, A., Carlsson, K., Lundqvist, D., Ingvar, M., 2007. On the unconscious subcortical origin of human fear. Physiology and Behavior 92, 180–185.
Palomba, D., Sarlo, M., Angrilli, A., Mini, A., Stegagno, L., 2000. Cardiac responses associated with affective processing of unpleasant film stimuli. International Journal of Psychophysiology 36, 45–57.
Palomba, D., Stegagno, L., 1993. Physiology, perceived emotion, and memory: responding to film sequences. In: Birbaumer, N., Öhman, A. (Eds.), The Structure of Emotion. Hogrefe & Huber Publishers, Seattle, pp. 156–167.
Pauls, C.A., Stemmler, G., 2003. Repressive and defensive coping during fear and anger. Emotion 3, 284–302.
Poon, C.-S., Siniaia, M.S., 2000. Plasticity of cardiorespiratory neural processing: classification and computational functions. Respiration Physiology 122, 83–109.
Porges, S.W., 1995. Orienting in a defensive world: mammalian modifications of our evolutionary heritage. A Polyvagal Theory, Psychophysiology 32, 301–318.
Porges, S.W., 2001. The polyvagal theory: phylogenetic substrates of a social nervous system. International Journal of Psychophysiology 42, 123–146.
Porges, S.W., 2007. The polyvagal perspective. Biological Psychology 74, 116–143.
Porges, S.W., Doussard-Roosevelt, J.A., Portales, A.L., Greenspan, S., 1996. Infant regulation of the vagal "brake" predicts child behavior problems: a psychobiological model of social behavior. Developmental Psychobiology 29, 697–712.
Prkachin, K., Mills, D., Zwaal, C., Husted, J., 2001. Comparison of hemodynamic responses to social and nonsocial stress: evaluation of an anger interview. Psychophysiology 38, 879–885.
Prkachin, K., Williams-Avery, R., Zwaal, C., Mills, D., 1999. Cardiovascular changes during induced emotion: an application of Lang's theory of emotional imagery. Journal of Psychosomatic Research 47, 255–267.
Qiyuan, J., Richer, F., Wagoner, B.L., Beatty, J., 1985. The pupil and stimulus probability. Psychophysiology 22 (5), 530–534.
Rainville, P., Bechara, A., Naqvi, N., Damasio, A.R., 2006. Basic emotions are associated with distinct patterns of cardiorespiratory activity. International Journal of Psychophysiology 61, 5–18.
Reisenzein, R., 2000. Exploring the strength of association between the components of emotion syndromes: the case of surprise. Cognition and Emotion 14, 1–38.
Rimm-Kaufman, S.E., Kagan, J., 1996. The psychological significance of changes in skin temperature. Motivation and Emotion 20, 63–78.
Ritz, T., George, C., Dahme, B., 2000. Respiratory resistance during emotional stimulation: evidence for a nonspecific effect of emotional arousal? Biological Psychology 52, 143–160.
Ritz, T., Steptoe, A., Wilde, S.D., Costa, M., 2000. Emotions and stress increase respiratory resistance in asthma. Psychosomatic Medicine 62, 401–412.
Ritz, T., Thöns, M., Fahrenkrug, S., Dahme, B., 2005. Airways, respiration, and respiratory sinus arrhythmia during picture viewing. Psychophysiology 42, 568–578.
Ritz, T., Wilhelm, F.H., Gerlach, A.L., Kullowatz, A., Roth, W.T., 2005. End-tidal pCO_2 in blood phobics during viewing of emotion- and disease-related films. Psychosomatic Medicine 67 (4), 661–668.
Roberts, R.J., Weerts, T.C., 1982. Cardiovascular responding during anger and fear imagery. Psychological Report 50, 219–230.
Robin, O., Alaoui-Ismaïli, O., Dittmar, A., Vernet-Maury, E., 1998. Emotional responses evoked by dental odors: an evaluation from autonomic parameters. Journal of Dental Research 77, 1638–1646.
Rochman, D., Diamond, G.M., 2008. From unresolved anger to sadness: identifying physiological correlates. Journal of Counseling Psychology 55, 96–105.
Rohrmann, S., Hopp, H., 2008. Cardiovascular indicators of disgust. International Journal of Psychophysiology 68, 201–208.
Roseman, I.J., 1984. Cognitive determinants of emotion—a structural theory. In: Shaver, P. (Ed.), Emotions, Relationships, and Health, vol. 5. Sage, Beverly Hills, CA, pp. 11–36.
Roseman, I.J., 2001. A model of appraisal in the emotion system: Integrating theory, research, and applications. In: Scherer, K.R., Schorr, A., Johnstone, T. (Eds.), Appraisal Processes in Emotion: Theory, Methods, Research. Oxford University Press, New York, pp. 68–91.
Roseman, I.J., Wiest, C., Swartz, T.S., 1994. Phenomenology, behaviors, and goals differentiate discrete emotions. Journal of Personality and Social Psychology 67, 206–221.
Rottenberg, J., Gross, J.J., Wilhelm, F.H., Najmi, S., Gotlib, I.H., 2002. Crying threshold and intensity in major depressive disorder. Journal of Abnormal Psychology 111, 302–312.
Rottenberg, J., Kasch, K.L., Gross, J.J., Gotlib, I.H., 2002. Sadness and amusement reactivity differentially predict concurrent and prospective functioning in major depressive disorder. Emotion 2, 135–146.
Rottenberg, J., Salomon, K., Gross, J.J., Gotlib, I.H., 2005. Vagal withdrawal to sad film predicts subsequent recovery from depression. Psychophysiology 42, 277–281.
Rottenberg, J., Wilhelm, F.H., Gross, J.J., Gotlib, I.H., 2003. Vagal rebound during resolution of tearful crying among depressed and nondepressed individuals. Psychophysiology 40, 1–6.
Rowell, L.B., 1986. Human Circulation Regulation During Physical Stress. Oxford University Press, New York.
Ruch, W., 1995. Will the real relationship between facial expression and affective experience please stand up: the case of exhileration. Cognition and Emotion 9, 33–58.
Russell, J.A., 2003. Core affect and the psychological construction of emotion. Psychological Review 110, 145–172.
Sartory, G., 1993. The associative network of fear: how does it come about? In: Birbaumer, N., Öhman, A. (Eds.), The Organisation of Emotion: Psychophysiological Approaches to the Structure of Emotion. Hogrefe, Toronto, pp. 193–204.
Schachter, J., 1957. Pain, fear, and anger in hypertensives and normotensives: a psychophysiological study. Psychosomatic Medicine 19, 17–29.
Scherer, K.R., 1982. Emotion as a process: function, origin, and regulation. Social Science Information 21, 555–570.
Scherer, K.R., 1984. On the nature and function of emotion: a component process approach. In: Scherer, K.R., Ekman, P. (Eds.), Approaches to Emotion. Erlbaum, Hillsdale, NJ, pp. 293–317.
Scherer, K.R., 1987. Toward a dynamic theory of emotion: the component process model of affective states. Geneva Studies in Emotion and Communication 1, 1–98.
Scherer, K.R., 1994. Toward a concept of "modal emotions". In: Ekman, P., Davidson, R.J. (Eds.), The Nature of Emotion: Fundamental Questions. Oxford University Press, New York and Oxford, pp. 25–31.
Scherer, K.R., 2001. Appraisal considered as a process of multi-level sequential checking. In: Scherer, K.R., Schorr, A., Johnstone, T. (Eds.), Appraisal Processes

in Emotion: Theory, Methods, Research. Oxford University Press, New York and Oxford, pp. 92–120.
Scherer, K.R., 2009. The dynamic architecture of emotion: evidence for the component process model. Cognition and Emotion 23, 1307–1351.
Schneiderman, N., McCabe, P.M., 1989. Psychophysiologic strategies in laboratory research. In: Schneiderman, N., Weiss, S.M., Kaufman, P.G. (Eds.), Handbook of Research Methods in Cardiovascular Behavioral Medicine. Plenum Press, New York, pp. 349–364.
Schwartz, G.E., Weinberger, D.A., Singer, J.A., 1981. Cardiovascular differentiation of happiness, sadness, anger, and fear following imagery and exercise. Psychosomatic Medicine 43, 343–364.
Schwarz, N., Clore, G.L., 1988. How do I feel about it? The informative function of affective states. In: Fiedler, K., Forgas, J.P. (Eds.), Affect, Cognition, and Social Behavior. Hogrefe, Göttingen, Germany, pp. 44–62.
Seligman, M.E.P., 1975. Helplessness: On Depression, Development, and Death. W.H. Freeman, San Francisco.
Sherwood, L., 2008. Human Physiology: From Cells to Systems, 7th revised ed. Cengage Learning, Belmont, CA.
Siddle, D.A.T., 1985. Effects of stimulus omission and stimulus change on dishabituation of the skin conductance response. Journal of Experimental Psychology: Learning, Memory, and Cognition 11, 206–216.
Siddle, D.A.T., 1991. Orienting, habituation, and resource allocation: an associative analysis. Psychophysiology 28, 245–259.
Siddle, D.A.T., Heron, P.A., 1976. Effects of length of training and amount of tone frequency change on amplitude of autonomic components of the orienting response. Psychophysiology 13, 281–287.
Siddle, D.A.T., Stephenson, D., Spinks, J.A., 1983. Elicitation and habituation of the orienting response. In: Siddle, D.A. (Ed.), Orienting and Habituation: Perspectives in Human Research. John Wiley & Sons Ltd., New York, pp. 109–182.
Silvestrini, N., Gendolla, G.H.E., 2007. Mood effects on autonomic activity in mood regulation. Psychophysiology 44 (4), 650–659.
Sinha, R., Lovallo, W.R., Parsons, O.A., 1992. Cardiovascular differentiation of emotion. Psychosomatic Medicine 54, 422–435.
Sinha, R., Parsons, O.A., 1996. Multivariate response patterning of fear and anger. Cognition and Emotion 10, 173–198.
Smith, C.A., Ellsworth, P.C., 1985. Patterns of cognitive appraisal in emotion. Journal of Personality and Social Psychology 48, 813–838.
Smith, C.A., Kirby, L.D., 2004. Appraisal as a pervasive determinant of anger. Emotion 4, 133–138.
Sokhadze, E.M., 2007. Effects of music on the recovery of autonomic and electrocortical activity after stress induced by aversive visual stimuli. Applied Psychophysiology, and Biofeedback 32, 31–50.
Sokolov, E.N., 1990. The orienting response, and future directions of its development. The Pavlovian Journal of Biological Science 25, 142–150.
Soltysik, S., Jelen, P., 2005. In rats, sighs correlate with relief. Physiology and Behavior 85, 598–602.
Stemmler, G., 1993. Receptor antagonists as tools for structural measurement in psychophysiology. Neuropsychobiology 28, 47–53.
Stemmler, G., 2003. Methodological considerations in the psychophysiological study of emotion. In: Davidson, R.J., Scherer, K.R., Goldsmith, H. (Eds.), Handbook of Affective Sciences. Oxford University Press, New York, pp. 225–255.
Stemmler, G., 2004. Physiological processes during emotion. In: Philippot, P., Feldman, R.S. (Eds.), The Regulation of Emotion. Erlbaum, Mahwah, NJ, pp. 33–70.
Stemmler, G. (2009). Somatoviszerale Aktivierung [Somatovisceral activation], In: Niels, B., Dieter, F., Julius, K., Stemmler, G. (Eds.), Psychologie der Emotion, Enzyklopädie der Psychologie [Psychology of emotion, encyclopedia of psychology]. Hogrefe, Göttingen, Germany, pp. 314–361 (Chapter 8).
Stemmler, G., Aue, T., Wacker, J., 2007. Anger and fear: separable effects of emotion and motivational direction on somatovisceral responses. International Journal of Psychophysiology 66, 141–153.
Stemmler, G., Fahrenberg, J., 1989. Psychophysiological assessment: Conceptual, psychometric, and statistical issues. In: Turpin, G. (Ed.), Handbook of Clinical Psychophysiology. Wiley, Chichester, pp. 71–104.
Stemmler, G., Grossman, P., Schmid, H., Foerster, F., 1991. A model of cardiovascular activation components for studies using autonomic receptor antagonists. Psychophysiology 28 (4), 367–382.
Stemmler, G., Heldmann, M., Pauls, C.A., Scherer, T., 2001. Constraints for emotion specificity in fear and anger: the context counts. Psychophysiology 38, 275–291.
Sternbach, R.A., 1962. Assessing differential autonomic patterns in emotion. Journal of Psychosomatic Research 6, 53–68.
Tangney, J.P., Miller, R.S., Flicker, L., Barlow, D.H., 1996. Are shame, guilt, and embarrassment distinct emotions? Journal of Personality and Social Psychology 70, 1256–1269.
Taylor, E.W., Jordan, D., Coote, J.H., 1999. Central control of the cardiovascular and respiratory systems and their interactions in vertebrates. Physiological Review 79, 855–916.
Taylor, S.E., 1991. Asymmetrical effects of positive and negative events: the mobilization-minimization hypothesis. Psychological Bulletin 110, 67–85.
Teroni, F., Deonna, J.A., 2008. Differentiating shame from guilt. Consciousness and Cognition 17, 725–740.
Thayer, J.F., Lane, R.D., 2000. A model of neurovisceral integration in emotion regulation and dysregulation. Journal of Affective Disorders 61, 201–216.
Theall-Honey, L.A., Schmidt, L.A., 2006. Do temperamentally shy children process emotion differently than nonshy children? Behavioral, psychophysiological, and gender differences in reticent preschoolers. Developmental Psychobiology 48, 187–196.
Tourangeau, R., Ellsworth, P., 1979. The role of facial response in the experience of emotion. Journal of Personnality and Social Psychology 37, 1519–1531.
Tsai, J.L., Chentsova-Dutton, Y., Freire-Bebeau, L., Przymus, D.E., 2002. Emotional expression and physiology in European Americans and Hmong Americans. Emotion 2 (4), 380–397.
Tsai, J.L., Levenson, R.W., Carstensen, L.L., 2000. Autonomic, subjective and expressive responses to emotional films in older and younger Chinese Americans and European Americans. Cultural Diversityand Ethnic Minority Psychology 15, 684–693.
Tsai, J.L., Pole, N., Levenson, R.W., Munoz, R.F., 2003. The effects of depression on the emotional responses of Spanish-speaking Latinas. Cultural Diversity and Ethnic Minority Psychology 9, 49–63.
Tugade, M.M., Fredrickson, B.L., 2004. Resilient individuals use positive emotions to bounce back from negative emotional experiences. Journal of Personality and Social Psychology 86, 320–333.
Uchiyama, I., 1992. Differentiation of fear, anger, and joy. Perceptual and Motor Skills 74, 663–667.
Van Diest, I., Proot, P., Van De Woestijne, K.P., Han, J.N., Devriese, S., Winters, W., Van Den Bergh, O., 2001. Critical conditions for hyperventilation responses. Behavior Modification 25, 621–639.
Van Diest, I., Thayer, J.F., Vandeputte, B., Van de Woestijne, K.P., Van den Bergh, O., 2006. Anxiety and respiratory variability. Physiology and Behavior 89, 189–195.
Van Diest, I., Winters, W., Devriese, S., Vercamst, E., Han, J.N., Van de Woestijne, K.P., Van den Bergh, O., 2001. Hyperventilation beyond fight/flight: respiratory responses during emotional imagery. Psychophysiology 38, 961–968.
Van Egeren, L.F., Abelson, J.L., Thornton, D.W., 1978. Cardiovascular consequences of expressing anger in a mutually-dependent relationship. Journal of Psychosomatic Research 22, 537–548.
Van Oyen Witvliet, C., Vrana, S.R., 1995. Psychophysiological responses as indices of affective dimensions. Psychophysiology 32, 436–443.
Van Reekum, C.M., Johnstone, T., Banse, R., Etter, A., Wehrle, T., Scherer, K.R., 2004. Psychophysiological responses to appraisal dimensions in a computer game. Cognition and Emotion 18, 663–688.
Veenhoven, R., 1991. Is happiness relative? Social Indicators Research 24, 1–34.
Vianna, E.P.M., Tranel, D., 2006. Gastric myoelectrical activity as an index of emotional arousal. International Journal of Psychophysiology 61, 70–76.
Vingerhoets, A.J., 1985. The role of the parasympathetic division of the autonomic nervous system in stress and the emotions. International Journal of Psychosomatics 32, 28–34.
Vingerhoets, A.J., Cornelius, R.R., Van Heck, G.L., Becht, M.C., 2000. Adult crying: a model and review of the literature. Review of General Psychology 4, 354–377.
Vlemincx, E., Van Diest, I., De Peuter, S., Bresseleers, J., Bogaerts, K., Fannes, S., Li, W., Van den Bergh, O., 2009. Why do you sigh: sigh frequency during induced stress and relief. Psychophysiology 46, 1005–1013.
Vrana, S.R., 1993. The psychophysiology of disgust: differentiating negative emotional context with facial EMG. Psychophysiology 30, 279–286.
Vrana, S.R., Gross, D., 2004. Reactions to facial expressions: effects of social context and speech anxiety on responses to neutral, anger, and joy expressions. Biological Psychology 66, 63–78.
Vrana, S.R., Rollock, D., 2002. The role of ethnicity, gender, emotional content and contextual differences in physiological, expressive, and self-reported emotional responses to imagery. Cognition and Emotion 16, 165–192.
Vrticka, P., Andersson, F., Sander, D., Vuilleumier, P., 2009. Memory for friends or foes: the social context of past encounters with faces modulates their subsequent neural traces in the brain. PLoS ONE 3, e2868.
Waldstein, S.R., Kop, W.J., Schmidt, L.A., Haufler, A.J., Krantz, D.S., Fox, N.A., 2000. Frontal electrocortical and cardiovascular reactivity during happiness and anger. Biological Psychology 55, 3–23.
Wiens, S., Peira, N., Golkar, A., Öhman, A., 2008. Recognizing masked threat: fear betrays, but disgust you can trust. Emotion 8, 810–819.
Wilhelm, F.H., Roth, W.T., 1998. Taking the laboratory to the skies: ambulatory assessment of self-report, autonomic and respiratory responses in flying phobia. Psychophysiology 35, 596–606.
Wilhelm, F.H., Schneider, S., Friedman, B.H., 2005. Psychophysiological assessment. In: Hersen, M. (Ed.), Comprehensive Handbook of Behavioral Assessment (vol. II: Child Assessment). Wiley, New York, pp. 201–231.
Williams, L.A., Das, P., Liddell, B., Olivieri, G., Peduto, A., Brammer, M., Gordon, E., 2005. BOLD, sweat and fears: fMRI and skin conductance distinguish facial fear signals. NeuroReport 16, 49–52.
Wilson, T.D., 2002. Strangers to Ourselves: Discovering the Adaptive Unconscious. Harvard University Press, Cambridge, MA.
Winkielman, P., Berridge, K.C., 2003. What is an unconscious emotion? (The case of unconscious "liking"). Cognition and Emotion 17, 181–211.
Winkielman, P., Berridge, K.C., 2004. Unconscious emotion. Current Directions in Psychological Science 13, 120–123.
Winton, W.M., Putnam, L.E., Krauss, R.M., 1984. Facial and autonomic manifestations of the dimensional structure of emotion. Journal of Experimental Social Psychology 20, 195–216.
Woody, S.R., Teachman, B.A., 2000. Intersection of disgust and fear: normative and pathological views. Clinical Psychology: Science and Practice 7, 291–311.
Wright, R.A., 1996. Brehm's theory of motivation as a model of effort and cardiovascular response. In: Gollwitzer, P.M., Bargh, J.A. (Eds.), The Psychology

of Action: Linking Cognition and Motivation to Behavior. Guilford, New York, pp. 424–453.
Wright, R.A., 1998. Ability perception and cardiovascular response to behavioral challenge. In: Kofta, M., Weary, G., Sedek, G. (Eds.), Control in Action: Cognitive and Motivational Mechanisms. Plenum, New York, pp. 197–232.
Wright, R.A., Kirby, L.D., 2001. Effort determination of cardiovascular response: an integrative analysis with applications in social psychology. In: Zanna, M. (Ed.), Advances in Experimental Social Psychology, vol. 33. Academic Press, San Diego, CA, pp. 255–307.
Yogo, Y., Hama, H., Yogo, M., Matsuyama, Y., 1995. A study of physiological response during emotional imaging. Perceptual and Motor Skills 81, 43–49.