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Cardiovascular, electrodermal, and respiratory response patterns to fear- and sadness-inducing films

SYLVIA D. KREIBIG,[a] FRANK H. WILHELM,[b] WALTON T. ROTH,[c,d] AND JAMES J. GROSS[e]

[a]Department of Psychology, Swiss Center for Affective Sciences, University of Geneva, Geneva, Switzerland
[b]Institute for Psychology, University of Basel, Basel, Switzerland
[c]Department of Psychiatry and Behavioral Sciences, Stanford University School of Medicine, Palo Alto, California, USA
[d]Department of Veterans Affairs Health Care System, Palo Alto, California, USA
[e]Department of Psychology, Stanford University, Stanford, California, USA

Abstract

Responses to fear- and sadness-inducing films were assessed using a broad range of cardiovascular (heart rate, T-wave amplitude, low- and high-frequency heart rate variability, stroke volume, preejection period, left-ventricular ejection time, Heather index, blood pressure, pulse amplitude and transit time, and finger temperature), electrodermal (level, response rate, and response amplitude), and respiratory (rate, tidal volume and its variability, inspiratory flow rate, duty cycle, and end-tidal pCO_2) measures. Subjective emotional experience and facial behavior (Corrugator Supercilii and Zygomaticus Major EMG) served as control measures. Results indicated robust differential physiological response patterns for fear, sadness, and neutral (mean classification accuracy 85%). Findings are discussed in terms of the fight–flight and conservation–withdrawal responses and possible limitations of a valence-arousal categorization of emotion in affective space.

Descriptors: Emotion, Film, Physiological patterns, Autonomic nervous system, Respiration

Fear and sadness are two emotions that play a crucial role in adaptation (e.g., Darwin, 1872; Ekman, 1984, 1999). Although both emotions are thought to be negative, withdrawal-related emotions (Coan & Allen, 2003), they are clearly distinguished on psychological, experiential, expressive, and behavioral grounds. Fear, experienced as apprehension, uncertainty, and feelings of danger (Izard & Buechler, 1980), arises at the prospect of physical or social threat to the self, and serves to protect the organism from harm (Ekman, 1999; Power & Dalgleish, 1997). The prototypical facial expression of fear is that of eyebrows raised and pulled together, upper eyelids raised, and lower lids tensed—combined with an open mouth (e.g., Ekman & Friesen, 1978). Fear is characterized behaviorally by an urgent avoidance tendency, preparing the individual for avoidance or withdrawal behavior with the goal of achieving one's "own inaccessibility" (Birbaumer & Schmidt, 1999; Frijda, 1986; Lang, Levin, Miller, & Kozak, 1983). The avoidance of danger can be achieved by a fearful person, for example, by running away, staying put, calling for help, or brandishing a weapon. In this context, the object of avoidance may be a physical stimulus (e.g., a knife), a perceived danger (e.g., the possibility of being stabbed), or a psychological consequence of the stimulus or danger (e.g., the pain of a wound). When fear is elicited by stimuli that are not realistically threatening, this emotional response is maladaptive (e.g., Barlow, 1991, 2002; Kring & Bachorowski, 1999; Power & Dalgleish, 1997).

In contrast to the emergency response associated with fear, sadness is commonly described as a more passive emotion. Sadness occurs when a goal can no longer be achieved, as when one is separated from a valued person or object, or when one loses a sense of control (e.g., Birbaumer & Schmidt, 1999; Ekman, 1999; Lazarus, 1991; Power & Dalgleish, 1997). Sadness, experienced as feelings of loneliness, discouragement, rejection, and dissatisfaction with oneself (Izard & Buechler, 1980), may represent a psychobiological reaction that helps maintain group attachment (Bowlby, 1980). Throughout life—and particularly so in the infant—sadness communicates a need for change and may elicit empathic responses. The prototypical facial expression of sadness is one of a raised and pulled together inner portion of the

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Address reprint requests to: Sylvia D. Kreibig, Dipl.-Psych., Swiss Center for Affective Sciences, University of Geneva—CISA, 7, Rue des Battoirs, 1205 Geneva, Switzerland. E-mail: sylvia.kreibig@cisa.unige.ch

eyebrows (forming glabellar wrinkles), narrowed eyes, raised cheeks, pulled down corners of the lips, and a pushed up chin boss, often accompanied by crying and a closed and downward looking posture formed by head and chest being bent forward (e.g., Coulson, 2004; Ekman & Friesen, 1978; Parke & Waters, 1996). Although emotions in general have been connected to urgent action tendencies (Frijda, 1986), sadness appears to be associated with a *decrease* in responsivity to environment. Depression constitutes an emotional disturbance fundamentally related to sadness (Barlow, 1991; Power & Dalgleish, 1997). In depressive disorders, sadness is exhibited in a stereotyped, context-insensitive fashion accompanied by an overall emotional numbing, instead of the emotion response being a flexible behavioral guidance system (e.g., Rottenberg & Gross, 2003; Rottenberg, Gross, & Gotlib, 2005).

Based on these clear psychological, experiential, expressive, and behavioral distinctions between fear and sadness, one might expect that physiological responses—constituting another important response component of the emotion complex (e.g., Gross, 1998; Lang, 1995; Scherer, 2001)—are similarly well distinguished. However, we still do not have a sufficient understanding of the psychophysiological differences between emotions in general (for reviews, see Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000; Cacioppo, Klein, Berntson, & Hatfield, 1993; Stemmler, 1998), and of the psychophysiological differences between fear and sadness in particular. In the present article, our focus is thus on examining physiological differences between fear and sadness.

Theoretical Predictions of Physiological Responses in Fear and Sadness

Narrative accounts in the literature on autonomic nervous system activity during states of fear and sadness characterize the former as a high arousal state with "quickened heart-beats, shallow breathing, trembling lips, weakened limbs, goose-flesh, and visceral stirrings" (James, 1884, p. 194), whereas the latter—in resemblance to grief—has been characterized by "diminished muscular tonicity, ... bloodlessness of the skin, ... and feeble activity of the muscles of expiration ... combined with long drawn sighs" (Lange, 1887, as cited in James, 1890, p. 444).

Based on theoretical considerations, these two emotions might be expected to differ physiologically, as physiological changes in emotion, in general, are hypothesized to modify a context-specific physiological pattern (e.g., Lang, 1979, 1993; Leventhal, 1984; Scherer, 2001; Stemmler, 1989, 1992) and, more specifically, to prepare the body for subsequent actions (e.g., Ekman, 1984; Levenson, 1994; Tooby & Cosmides, 1990). In particular, two contrasting biological emergency reactions have been hypothesized to be mediated by the central nervous system (Bernard & Bandler, 1998; Kaufman & Rosenblum, 1967). The first one—variously referred to as a flight–fight, defense, or active coping response (Cannon, 1929; Graham, 1979; Turpin, 1986)—involves activity, energy expenditure, and engagement with the environment to control sources of supply and avert danger; its counterpart—conservation–withdrawal or passive coping (Bohus et al., 1988; Engel, 1977; Schmale, 1958; Schneiderman & McCabe, 1989)—involves inactivity, energy conservation, raising of the stimulus barrier, and withdrawal from the environment (Buerki & Adler, 2005; Engel, 1962). Both reaction patterns are directed toward adaptation to stressful situations, aimed at self-protection and self-preservation (Kelsey, Ornduff, Reiff, & Arthur, 2002).

The first reaction may be considered the biological foundation of fear and anger, activated as long as some controllability is perceived, and the second, the foundation of sadness and depression, activated when controllability is absent and goals are no longer perceived as achievable (Engel, 1962; Valent, 1998). The first reaction, the fight–flight reaction pattern of engagement, is characterized by sympathetic activation, causing heart rate (HR) acceleration associated with decreased preejection period (PEP), increased cardiac output, regional blood flow changes in favor of vascular beds with high metabolic demands, and vagal withdrawal, causing blood pressure elevation (e.g., Bosch et al., 2001; Lovallo, 2005), accompanied by sweating, dilation of the bronchioles, and stress-induced hyperventilation (Curtis & O'Keefe, 2002; Van Diest et al., 2001; Wilhelmsen, 2000). The second reaction, the conservation–withdrawal reaction pattern of disengagement, is characterized by an enhanced parasympathetic tone causing HR deceleration and constriction of the airways and a moderate sympathetic coactivation as indicated by shortened PEP and increased systolic blood pressure (e.g., Bosch et al., 2001; Ritz, Steptoe, DeWilde, & Costa, 2000).

Empirical Results of Psychophysiological Response Patterning in Fear and Sadness

Fear and sadness are not the typical choice of emotion contrast. Based on a hybrid discrete-dimensional model of affective space (Levenson, 1988), fear is usually contrasted with anger, whereas sadness is commonly contrasted with happiness. This may be why the predictions described in the previous section have not yet been directly tested—although, coming from this line of thought, fear and sadness seem to call out for a direct comparison of their own. A review of empirical research on psychophysiological response patterning in fear (for a recent review, see Stemmler, 2004) and sadness draws a more complex picture than the one which might be expected on theoretical grounds. Fear has been investigated in a number of different experimental settings: presenting pictures displaying scenes of aggression, physical brutality, and combat (Bernat, Patrick, Benning, & Tellegen, 2006; Bradley, Codispoti, Cuthbert, & Lang, 2001) or facial expressions of fear and anger (Dimberg & Karlsson, 1997; Lerner, Gonzalez, Dahl, Hariri, & Taylor, 2005), short films depicting violent threat (e.g., Montoya, Campos, & Schandry, 2005; Palomba, Sarlo, Angrilli, Mini, & Stegagno, 2000), fear-inducing musical excerpts (Etzel, Johnsen, Dickerson, Tranel, & Adolphs, 2006; Krumhansl, 1997), fear imagery (Sinha & Parsons, 1996; Stemmler, 1989; Van Diest et al., 2001; Vrana & Rollock, 2002), preparation for a feared speech before an audience (Borkovec & O'Brien, 1977; Pauls & Stemmler, 2003; Stemmler, Heldmann, Pauls, & Scherer, 2001), and the posing of fearful expressions in the directed facial action task (Ekman, Levenson, & Friesen, 1983; Levenson, Ekman, & Friesen, 1990; Levenson, Ekman, Heider, & Friesen, 1992). Sadness has been investigated in similar paradigms: presentation of sad pictures or dysphoric imagery (e.g., Rainville, Bechara, Naqvi, & Damasio, 2006; Ritz, George, & Dahme, 2000; Ritz, Thöns, Fahrenkrug, & Dahme, 2005; Sinha, Lovallo, & Parsons, 1992), sad films (e.g., Gross & Levenson, 1997; Kunzmann & Grühn, 2005; Palomba & Stegagno, 1993; Tsai, Levenson, & Carstensen, 2000), sad musical excerpts (Etzel et al., 2006; Krumhansl, 1997; Nyklicek, Thayer, & Van Doornen, 1997), and posing sad expressions in the directed facial action task (Ekman et al., 1983; Levenson et al., 1990, 1992).

An overview of results from studies on physiological responding in fear and sadness, disregarding differences between emotion induction paradigms, is provided in Table 1. Aiming for a broad overview, this table glosses over some known differences in paradigm properties, in particular differences of tonic versus phasic response elicitation and respective primary response indices. The table shows that the predictions of the fight–flight and conservation–withdrawal responses are neither consistently confirmed nor disconfirmed, which may be because different sets of measures were assessed with different induction paradigms, rather than recording a comprehensive set of measures in one experimental context. This is an important limitation, as comparability of responses across contexts may be limited (i.e., context specific, Stemmler, 1992).

The Present Study

We investigated the emotional response patterning of fear and sadness in normal participants with autonomic, self-report, and behavioral measures, using a film paradigm for emotion induction. Films have a rich history of laboratory application for emotion induction (e.g., Rottenberg, Ray, & Gross, in press) and a number of standardized film sets have been developed and validated for populations of different languages and cultures (Gross & Levenson, 1995; Hagemann et al., 1999; Philippot, 1993). Concerns about a relative nonselectivity of the emotions thus elicited have been raised by a number of researchers (Gross & Levenson, 1995; Stemmler, 2002) as emotion self-reports often have shown that other emotions are elicited in addition to the target emotion. However, recent validations have demonstrated that films can be quite specific in the emotion they elicit (Hewig et al., 2005; Rottenberg et al., in press; Schaefer, Nils, Sanchez, & Philippot, 2007). Because films presumably elicit emotion relatively automatically, film paradigms do not necessitate deceiving participants about the aims of the study to control for subjects trying to meet implicit demands in the viewing situation. Recent research on the psychophysiology of emotional responses has hence relied significantly on films as a means of laboratory emotion induction (e.g., Christie & Friedman, 2004; Frazier, Strauss & Steinhauer, 2004; Fredrickson, Mancuso, Branigan, & Tugade, 2000; Gomez, Zimmermann, Guttormsen-Schär, & Danuser, 2005; Gross & Levenson, 1993; Hubert & de Jong-Meyer, 1990, 1991; Kunzmann & Grühn, 2005; Montoya et al., 2005; Palomba et al., 2000; Ritz, Steptoe, et al., 2000; Simons, Detenber, Roedema, & Reiss, 1999; Sloan, 2004; Vianna, Weinstock, Elliott, Summers, & Tranel, 2006; Wiens, Mezzacappa, & Katkin, 2000).

In the present study, our main aim was to examine whether fear and sadness differ from each other and from a neutral condition. We included some less frequently used measures of emotional activation, namely, low- and high-frequency HR variability (the latter quantifying respiratory sinus arrhythmia, RSA), tidal volume variability, and end-tidal carbon dioxide partial pressure (end-tidal pCO_2). Variables from three major peripheral physiological systems were continuously monitored: cardiovascular (including measures allowing differentiation of sympathetic [PEP] and parasympathetic [RSA] cardiac influences; Berntson, Cacioppo, & Quigley, 1991; Stemmler, 1993), electrodermal, and respiratory. To our knowledge, such a comprehensive sampling of emotionally relevant physiological response systems in an emotion elicitation study is unique and thus may help clarify the nature of the emotional response patterns associated with fear and sadness in this context. A secondary aim of the present study was an applied one, namely, to test whether the combination of physiological variables found to differ between the investigated emotion conditions could be used to successfully predict emotional state.

Method

Participants

Thirty-seven healthy student volunteers were recruited from the Stanford University student body through advertisements. Of these, 34 participants (19 women) were included in the analysis. Two participants were excluded due to technical problems, and 1 participant did not comply with experimental instructions. Mean age for men was 20.8 years ($SD = 1.26$) and for women 21.2 years ($SD = 2.70$). The ethnic background of the sample was 6% African-American, 34% Asian-American, 58% European-American (47% non-Hispanic, 11% Hispanic), and 2% other.

Participants' eligibility was determined by a phone screening in which potential participants were asked for demographic information and about their physical and mental health using an abbreviated version of the Structured Clinical Interview for DSM-IV, Axis I (SCID-I; First, Gibbon, Spitzer, & Williams, 1997). Exclusion criteria based on physical health were smoking, epilepsy, coronary, respiratory, or thyroid disease, and neurological disorders. Participants were also excluded on the basis of a reported lifetime history of brain injury and on current diagnoses or lifetime history of panic disorder, major depressive disorder, manic episodes, or symptoms of psychosis. Participants with Axis I mental disorders were excluded because such disorders alter emotional reactivity in specific ways that would make the sample more heterogeneous (e.g., Allen, Trinder, & Brennen, 1999; Dimberg, 1990a; Rottenberg, Kasch, Gross, & Gotlib, 2002). Furthermore, participants were excluded if they were alcohol or substance dependent or had a history of dependency. All participants were free of any kind of medication with effects on the cardiovascular, respiratory, or central nervous system. Participants were tested for and found to have normal hearing. Each gave written consent prior to the experimental session. They were paid US $30 for their participation in the approximately 2.5-h laboratory session.

Materials

Six film clips (two frightening, two sad, and two neutral) were selected based on criteria recommended by Gross and Levenson (1995). The neutral clips were included to provide a comparison against which the effects of the fear- and sadness-inducing films could be contrasted (Piferi, Kline, Younger, & Lawler, 2000). Two sets of clips (two clips for each target emotion) allowed us to test whether reactions were specific to the emotion domain represented by the films or to specific clips (cf. Rottenberg et al., in press).

Film clips were selected to last about 10 min each. Small variations in length of film clips were accepted to make sure the thematic content was understandable and involving. Each clip was preceded by a 30-s audio introduction laying the groundwork for the plot, because context information may amplify the emotional impact (Palomba & Stegagno, 1993).

The fear-provoking films were chosen to evoke themes of anticipation of immediate bodily injury or impending death by a pursuer and final confrontation with the source of the threat. Scenes from *I Know What You Did Last Summer* (Chaffin, Feig, Moritz, & Gillespie, 1997) and *I Still Know What You Did Last*

Table 1. *Overview of Studies on Peripheral Physiological Responding in Fear and Sadness, Disregarding Differences Between Emotion Induction Paradigms*

Dependent variable	Fear	Study	Sadness	Study
Cardiovascular system				
Heart rate	↗	Borkovec & O'Brien, 1977	↗	Ekman, Levenson, & Friesen, 1983
	↗	Eisenberg, et al., 1988	↗	Frazier, Strauss, & Steinhauer, 2004
	↗	Ekman, Levenson, & Friesen, 1983	↗	Gross & Levenson, 1997
	↗	Etzel, Johnsen, Dickerson, Tranel, & Adolphs, 2006	↗	Kunzmann & Grühn, 2005
	↗	Frazier, Strauss, & Steinhauer, 2004	↗	Levenson, Ekman, & Friesen, 1990
	↗	Lerner, Gonzalez, Dahl, Hariri, & Taylor, 2005	↗	Levenson, Ekman, Heider, & Friesen, 1992
	↗	Levenson, Ekman, & Friesen, 1990	↗	Palomba & Stegagno, 1993
	↗	Levenson, Ekman, Heider, & Friesen, 1992	↗	Ritz, George, & Dahme, 2000
	↗	Palomba, Sarlo, Angrilli, Mini, & Stegagno, 2000	↗	Sinha, Lovallo, & Parsons, 1992
	↗	Palomba & Stegagno, 1993		
	↗	Pauls & Stemmler, 2003		
	↗	Sinha & Parsons, 1996		
	↗	Stemmler, Heldmann, Pauls, & Scherer, 2001		
	↗	Tourangeau & Ellsworth, 1979		
	↗	Vrana & Rollock, 2002		
	↘	Bernat, Patrick, Benning, & Tellegen, 2006	↘	Eisenberg, et al., 1988
	↘	Bradley, Codispoti, Cuthbert, & Lang, 2001	↘	Etzel, Johnsen, Dickerson, Tranel, & Adolphs, 2006
	↘	Dimberg, 1986	↘	Krumhansl, 1997
			↘	Tsai, Levenson, & Carstensen, 2000
Pulse rate (finger)	↗	Van Diest, et al., 2001		
			↘	Van Diest, et al., 2001
Heart rate variability			↗	Rottenberg, Wilhelm, Gross, & Gotlib, 2002, 2003[a]
	↘	Frazier, Strauss, & Steinhauer, 2004[a]	↘	Frazier, Strauss, & Steinhauer, 2004[a]
	↘	Pauls & Stemmler, 2003[b]		
			↔	Etzel, Johnsen, Dickerson, Tranel, & Adolphs, 2006[c,d]
			↔	Krumhansl, 1997[e]
			↔	Rainville, Bechara, Naqvi, & Damasio, 2006[a]
			↔	Ritz, George, & Dahme, 2000[c]
			↔	Ritz, Thöns, Fahrenkrug, & Dahme, 2005[c]
T-wave amplitude	↘	Palomba, Sarlo, Angrilli, Mini, & Stegagno, 2000		
Cardiac output	↗	Pauls & Stemmler, 2003		
	↗	Stemmler, Heldmann, Pauls, & Scherer, 2001		
	↘	Montoya, Campos, & Schandry, 2005	↘	Sinha, Lovallo, & Parsons, 1992
Stroke volume	↘	Montoya, Campos, & Schandry, 2005		
	↘	Pauls & Stemmler, 2003		
	↘	Stemmler, Heldmann, Pauls, & Scherer, 2001		
Preejection period	↘	Montoya, Campos, & Schandry, 2005		
	↘	Pauls & Stemmler, 2003		
	↘	Stemmler, Heldmann, Pauls, & Scherer, 2001		
Total peripheral resistance	↗	Montoya, Campos, & Schandry, 2005	↗	Sinha, Lovallo, & Parsons, 1992
	↘	Pauls & Stemmler, 2003		
	↘	Stemmler, Heldmann, Pauls, & Scherer, 2001		
Systolic blood pressure	↗	Lerner, Gonzalez, Dahl, Hariri, & Taylor, 2005	↗	Krumhansl, 1997
	↗	Pauls & Stemmler, 2003	↗	Sinha, Lovallo, & Parsons, 1992
	↗	Stemmler, Heldmann, Pauls, & Scherer, 2001		
	↗	Vrana & Rollock, 2002		
Diastolic blood pressure	↗	Lerner, Gonzalez, Dahl, Hariri, & Taylor, 2005	↗	Krumhansl, 1997
	↗	Pauls & Stemmler, 2003	↗	Sinha, Lovallo, & Parsons, 1992
	↗	Stemmler, Heldmann, Pauls, & Scherer, 2001		
	↗	Vrana & Rollock, 2002		
	↔	Sinha & Parsons, 1996		
	↔	Sinha, Lovallo, & Parsons, 1992		
Mean arterial pressure	↗	Lerner, Gonzalez, Dahl, Hariri, & Taylor, 2005	↗	Krumhansl, 1997
	↗	Pauls & Stemmler, 2003		
	↗	Stemmler, Heldmann, Pauls, & Scherer, 2001		
	↗	Vrana & Rollock, 2002		

Table 1. (Contd.)

Dependent variable	Fear	Study	Sadness	Study
Finger temperature	↘	Krumhansl, 1997	↘	Gross & Levenson, 1997
	↘	Rimm-Kaufman & Kagan, 1996	↘	Krumhansl, 1997
	↘	Sinha & Parsons, 1996	↘	Kunzmann & Grühn, 2005
	↘	Stemmler, 1989		
	↔	Rimm-Kaufman & Kagan, 1996		
Finger pulse transit time	↗	Krumhansl, 1997		
	↘	Levenson, Ekman, Heider, & Friesen, 1992	↘	Gross & Levenson, 1997
	↘	Stemmler, 1989	↘	Krumhansl, 1997
	↘	Stemmler, Heldmann, Pauls, & Scherer, 2001	↘	Kunzmann & Grühn, 2005
			↘	Ekman, Levenson, & Friesen, 1983
			↘	Levenson, Ekman, Heider, & Friesen, 1992
Finger pulse volume amplitude			↗	Ekman, Levenson, & Friesen, 1983
			↗	Levenson, Ekman, Heider, & Friesen, 1992
	↘	Krumhansl, 1997	↘	Gross & Levenson, 1997
	↘	Levenson, Ekman, Heider, & Friesen, 1992	↘	Krumhansl, 1997
	↘	Stemmler, 1989	↘	Kunzmann & Grühn, 2005
	↘	Stemmler, Heldmann, Pauls, & Scherer, 2001		
Electrodermal system				
Skin conductance level	↗	Ax, 1953	↗	Christie & Friedman, 2004
	↗	Borkovec & O'Brien, 1977	↗	Ekman, Levenson, & Friesen, 1983
	↗	Folkins, Lawson, Opton, & Lazarus, 1968	↗	Levenson, Ekman, & Friesen, 1990
	↗	Levenson, Ekman, & Friesen, 1990		
	↗	Palomba, Sarlo, Angrilli, Mini, & Stegagno, 2000		
	↗	Stemmler, Heldmann, Pauls, & Scherer, 2001		
	↗	Vrana & Rollock, 2002		
			↘	Gross & Levenson, 1997
			↘	Hess, Kappas, McHugo, Lanzetta, & Kleck, 1992
			↘	Krumhansl, 1997
			↘	Kunzmann & Grühn, 2005
	↔	Stemmler, 2004	↔	Tourangeau & Ellsworth, 1979
	↔	Tourangeau & Ellsworth, 1979		
Skin conductance response rate	↗	Frazier, Strauss, & Steinhauer, 2004	↗	Frazier, Strauss, & Steinhauer, 2004
	↗	Pauls & Stemmler, 2003		
	↗	Stemmler, 2004		
	↗	Stemmler, Heldmann, Pauls, & Scherer, 2001		
	↗	Tourangeau & Ellsworth, 1979		
	↔	Stemmler, 1989		
Skin conductance response magnitude	↗	Bernat, Patrick, Benning, & Tellegen, 2006		
	↗	Bradley, Codispoti, Cuthbert, & Lang, 2001		
	↗	Dimberg, 1986		
Respiratory system				
Respiration rate	↗	Ax, 1953	↗	Levenson, Ekman, Heider, & Friesen, 1992
	↗	Bloch, Lemeignan, & Aguilera, 1991		
	↗	Boiten, Frijda, & Wientjes, 1994		
	↗	Etzel, Johnsen, Dickerson, Tranel, & Adolphs, 2006		
	↗	Krumhansl, 1997		
	↗	Levenson, Ekman, Heider, & Friesen, 1992		
	↗	Palomba, Sarlo, Angrilli, Mini, & Stegagno, 2000		
	↗	Palomba & Stegagno, 1993		
	↗	Rainville, Bechara, Naqvi, & Damasio, 2006		
	↗	Stemmler, 2004		
	↗	Stemmler, Heldmann, Pauls, & Scherer, 2001		
	↗	Van Diest, et al., 2001		
	↗	Wilhelm & Roth, 1998		
	↘	Ritz, Thöns, Fahrenkrug, & Dahme, 2005	↘	Boiten, Frijda, & Wientjes, 1994
			↘	Etzel, Johnsen, Dickerson, Tranel, & Adolphs, 2006
			↘	Gross & Levenson, 1997
			↘	Kunzmann & Grühn, 2005
			↘	Palomba & Stegagno, 1993
			↘	Ritz, Thöns, Fahrenkrug, & Dahme, 2005
	↔	Boiten, 1998	↔	Boiten, 1998
	↔	Stemmler, 1989		

Table 1. (Contd.)

Dependent variable	Fear	Study	Sadness	Study
Tidal volume/ respiratory depth	↗	Boiten, Frijda, & Wientjes, 1994	↗	Boiten, Frijda, & Wientjes, 1994
			↗	Etzel, Johnsen, Dickerson, Tranel, & Adolphs, 2006
			↗	Gross & Levenson, 1997
			↗	Kunzmann & Grühn, 2005
	↘	Boiten, Frijda, & Wientjes, 1994	↘	Ritz, Thöns, Fahrenkrug, & Dahme, 2005
	↘	Bloch, Lemeignan, & Aguilera, 1991	↘	Van Diest, et al., 2001
	↘	Ritz, Thöns, Fahrenkrug, & Dahme, 2005		
	↘	Van Diest, et al., 2001		
End-expiratory pCO_2	↘	Alpers, Wilhelm, & Roth, 2005		
	↘	Boiten, Frijda, & Wientjes, 1994		
	↘	Ritz, Wilhelm, Gerlach, Kullowatz, & Roth, 2005		
	↘	Van Diest, et al., 2001		
	↘	Wientjes, 1992		
Expiration pause			↘	Nyklicek, Thayer, & Van Doornen, 1997
Variability in respiratory period	↗	Bloch, Lemeignan, & Aguilera, 1991	↗	Rainville, Bechara, Naqvi, & Damasio, 2006
	↗	Boiten, Frijda, & Wientjes, 1994		
Tidal volume variability	↗	Wilhelm, Gerlach, & Roth, 2001		
	↗	Wilhelm, Trabert, & Roth, 2001		
	↗	Wilhelm, et al., 2005		
Total respiratory resistance			↗	Ritz, George, & Dahme, 2000
Oscillatory resistance	↗	Ritz, Steptoe, DeWilde, & Costa, 2000	↗	Ritz, Steptoe, DeWilde, & Costa, 2000
Facial expressive system				
Corrugator Supercilii	↗	Bernat, Patrick, Benning, & Tellegen, 2006	↗	Allen, Horne, Trinder, 1996
	↗	Bradley, Codispoti, Cuthbert, & Lang, 2001	↗	Fillingim, Roth, & Cook III, 1992
	↗	Dimberg, 1986	↗	Hess, Kappas, McHugo, Lanzetta, & Kleck, 1992
	↗	Dimberg & Karlsson, 1997		
	↗	Sinha & Parsons, 1996		
	↗	Stemmler, Heldmann, Pauls, & Scherer, 2001		
	↗	Vrana & Rollock, 2002		
	↘	Pauls & Stemmler, 2003		
			↔	Ritz, George, & Dahme, 2000
Zygomaticus Major	↗	Pauls & Stemmler, 2003		
	↗	Stemmler, Heldmann, Pauls, & Scherer, 2001		
	↘	Dimberg, 1986		

Note: [a]spectral analysis RSA estimate; [b]root of the mean square of successive heart period differences (RMSSD); [c]peak-valley RSA estimate; [d]standard deviation of the RR interval series (SDNN) and standard deviation of successive differences (SDSD) heart rate variability; [e]RSA estimate not specified.

Summer (Beasley, Chaffin, Feig, Moritz, & Cannon, 1998), 639 and 674 s in length, respectively, were used as fear clips. Sad films were chosen to address themes of unjust suffering, loss, and grief in the context of families. Scenes for sadness-inducing clips were taken from *Steel Magnolias* (Ross, 1989) and *John Q*. (Burg, Koules, & Cassavetes, 2002), 713 and 761 s long, respectively. The neutral films depicted nature scenes, wild animals, and visitors to a National Park. Both neutral clips were excerpted from *Alaska's Wild Denali* (Rohlfing, 1997), 599 and 601 s in length.[1] Published reports (e.g., Gross & Levenson, 1997; Harris et al., 2000; Rottenberg et al., in press) and a pilot study assured that these films selectively elicit the targeted emotions.

To avoid possible order effects on emotion elicitation, six different orders of film presentation were created: three different orders of emotion, following a Latin square design, by two different orders of set.

[1]Detailed editing instructions are available from the authors upon request.

Equipment

Film clips were presented on a 20-in. diagonal (50.8 cm) television monitor at a viewing distance of 1.75 m in a room with low ambient light. The room was equipped with two remotely controlled high-sensitivity video cameras positioned behind darkened glass in opposing bookshelves, used to record both participants' facial behaviors and upper body movements, as well as the correct timing and quality of the film presentation during the experiment.

Procedure

Participants were randomly assigned to one of the six film presentation conditions. Data recording was conducted individually for each participant. Participants were seated upright in a chair. Their nondominant forearm rested on an armrest at heart level.

Following an orientation period, physiological sensors were attached. After that, participants were asked to find a comfortable sitting position and reminded to avoid any unnecessary movements and speech during the procedure. Moreover, they were to stay alert, keep their eyes open, and to breathe through their nose with their mouth closed so that end-tidal pCO_2 could be accurately assessed. Participants were provided with a pen, the experimental questionnaires, and instructions for experimental tasks, which were kept on their arm rest on the side of their dominant hand.

Physiological channels were continuously sampled during the film presentations and interspersed rest baseline periods to permit an exact assessment of changes that accompany the emotions studied. During the experimental session, the experimenter sat in an adjacent room from which the assessment room could be viewed by the two adjustable video cameras. Communication was possible via an intercom.

First, the amplifiers were calibrated and an additional respiration calibration task was carried out using fixed volume bags (Morel, Forster, & Suter, 1983). Participants then engaged in a startle habituation procedure of 2 min length.[2] After this, a 3-min quiet sitting period followed, which served as a first resting baseline measurement.

Then, participants started viewing the series of six film clips. Films were separated by 3-min resting baseline periods, during which participants were instructed to sit quietly. All films were preceded by instructions to watch the film carefully. However, participants could look away or shut their eyes if they found the films too distressing. Immediately after each film, participants' retrospective self-reported emotional responses to the film clip were assessed. Self-report of how participants felt before starting the viewing of the next clip after the resting baseline was also obtained. Each report was made on a separate page, which participants turned when they finished.

After viewing the sequence of six films, participants completed a paced breathing calibration task for vagal assessment (Wilhelm, Grossman, & Coyle, 2004) that was followed by a second respiration calibration to adjust for within-session linear trends and variation due to shifts in position. Finally, participants were disconnected from monitoring devices, debriefed, reimbursed, and dismissed.

Emotion Experience Measures

To verify that the films elicited the targeted emotional states, participants' self-reported emotional experience to the film was assessed using a comprehensive questionnaire. Among other constructs, we assessed fear (afraid), sadness (sad), feelings of arousal (activated, alert, and stimulated), and valence (negative/positive) using Likert-type scales, each comprising a line with equally spaced numbered tick marks labeled 0 to 10.[3] Participants were asked to judge their emotions by marking the spot on the line with a cross (possibly also between numbers) that best represented the strength of their emotion. Composite scores for arousal were computed for each participant by summing their ratings of the terms activated, alert, and stimulated (Cronbach's alpha = .83). Before the film clips (i.e., after the prior rest baseline), ratings were obtained for how the participant "feels right now." After the film clips, ratings were obtained for how the participant "generally felt during the last film clip." Additionally, participants were asked if they had seen the film before and whether they closed their eyes or looked away during the film (as suggested by Gross & Levenson, 1995).

Emotion-Expressive Behavior Measures

As facial emotional expressions are less prone to demand characteristics than self-reported emotional experience, surface EMG activity of facial muscles was additionally recorded to verify successful induction of targeted emotions. As an index of facial smiling and frowning behavior, activity of the Corrugator Supercilii and Zygomaticus Major muscles was measured on the right side of the face at a sampling rate of 1000 Hz using the multichannel amplifier described below for physiological measures. Prior to application of the electrodes, the designated sites on the skin surface were cleaned with distilled water and cotton pads and abraded using fine emery paper. EMG recordings were obtained with 4-mm miniature Beckman Ag/AgCl electrode pairs filled with Oxford Instruments (Hawthorne, NY) Teca Gel. In a bipolar configuration, electrodes were placed above the right eyebrow for assessment of Corrugator Supercilii muscle activity and on the right cheek in the middle of the mouth-to-ear tip line for Zygomaticus Major activity. Sensor placement followed recommendations by Fridlund and Cacioppo (1986). Interelectrode distance (center to center) was 1 cm for each recording site. While the sensors were applied, participants were instructed to completely relax their face. After placement, the resistance of the EMG electrodes was checked with a multimeter. If resistance exceeded 10 KΩ the skin preparation procedure was repeated and the electrodes reapplied in order to achieve lower impedance. The raw EMG signal was subjected to a 500-Hz anti-aliasing hardware filter and a 28–500-Hz digital band-pass filter (van Boxtel, 2001), rectified, and smoothed using a 10-ms time constant. Activity was quantified as percent of baseline level of the mean EMG level during the film condition immediately following.

Physiological Measures

An SA Instruments (San Diego, CA) 12-channel bioamplifier was interfaced with a Data Translation (Marlborough, MA) DT3001 PCI 12-bit 16-channel A/D conversion board to a computer that sampled physiological channels at 400 Hz, except for impedance cardiography, which was sampled at 1200 Hz. Channel data were simultaneously streamed to disk. Recorded signals were analyzed and averaged for each film period using an integrated set of biosignal analysis programs written in MATLAB (Mathworks, Inc., Natick, MA) (Wilhelm, Grossman, & Roth, 1999, a shareware version of which is available at the software repository of the Society for Psychophysiological Research; www.sprweb.org; Wilhelm & Peyk, 2005).

Cardiovascular system. For these measurements, alcohol pads were used to clean the skin sites and cotton pads to dry them. Electrocardiography was recorded using three disposable pregelled Ag/AgCl spot electrodes positioned in a three-lead unipolar modified chest configuration: The two active electrodes were placed on the right collar bone and the lowest rib on the left side in line with the left nipple, and the ground electrode was sited

[2]During the experiment, startle probes were administered about every minute. Number of startle probes presented and that elicited valid responses during each film and rest period was constant between conditions. Results for startle are discussed in a separate report (Kreibig, Wilhelm, Roth, & Gross, 2007; see also Kreibig, Wilhelm, Roth, & Gross, 2006).

[3]Besides these four constructs, this 31-item measure assessed perceived bodily sensations, cognitive states, reality distortion, emotion regulation, sleepiness, and embarrassment.

on the left collar bone. *Heart rate* (HR, in beats per minute) was analyzed by a program that detects R-waves in the ECG and calculates consecutive R–R intervals. Beat-by-beat values were edited for outliers from artifacts or ectopic myocardial activity, linearly interpolated, and converted into continuous R–R interval estimates in clock time updated every 250 ms using weighted-average interpolation. The resulting R–R interval time series was "instantaneous," as required for assessment of RSA.

T-wave amplitude (TWA, in microvolts) was determined by first locating the T-wave as the maximum value from 50 to 300 ms after an R-wave. The difference between this maximum and the isoelectric P–Q interval (mean of the last 50 ms before the occurrence of the present R-wave) is taken as TWA (Rau, 1991). The validity of TWA as a measure of sympathetic influence on myocardial performance (largely β-adrenergically driven) has been established pharmacologically and by physical and behavioral manipulations (e.g., Rau, 1991; Russell & Dart, 1986).

Heart rate variability parameters were quantified using customized software based on fast Fourier transform and Welch's averaged periodogram method (Welch, 1967). In short, R–R interval time series, resampled at 4 Hz, are first partitioned into 60-s segments, overlapping by 30 s. Each segment is then linearly detrended, Hanning windowed, and zero padded to 64 s (256 data points per segment). The segments are subjected to fast Fourier transform for frequency decomposition. The resulting power spectral density functions are adjusted to account for attenuation produced by the Hanning window. Successive power spectral density functions are then ensemble averaged across segments to obtain reliable estimates. Frequency band selection for high- and low-frequency power were based on recommendations by the Task Force Report (1996) and by Berntson et al. (1997) (cf. Allen & Chambers, 2007, for a recent review of the topic of cardiac vagal control). *Respiratory sinus arrhythmia* (RSA, in milliseconds squared) was thus estimated as the high-frequency power of heart period variability, reflecting parasympathetic (or vagal) influences on the heart. It was computed as the summed power spectral density of R–R interval fluctuations between 0.15 and 0.40 Hz, the frequency band typically associated with respiration. The primary index reported here is corrected RSA (RSA_c) with tidal volume and respiration rate partialed out (within individual) to disambiguate the influence of respiratory activity on this parameter (Grossman, Karemaker, & Wieling, 1991). Calibration data for each individual from a paced breathing procedure was used (paces were 9, 11.5, and 15 cpm, 60 s each; for details, see Wilhelm et al., 2004). The conventional, uncorrected RSA measure (RSA_{uc}) is also reported, because this is the index presently most commonly used. Similarly, spectral density of R–R interval fluctuations was summed over the *low-frequency band* (LF, 0.05–0.15 Hz, in milliseconds squared), reflecting both cardiac sympathetic and parasympathetic influences as well as blood pressure regulation (Appelhans & Luecken, 2006). Both measurements were normalized by natural logarithmic transformation.

Impedance cardiography was recorded with a second-generation Minnesota type impedance cardiograph (HIC-2000, Instrumentation for Medicine, Inc., Old Greenwich, CT). The impedance cardiograph measures electrical impedance changes in the thoracic cavity using a 4-mA constant current source with 100 kHz oscillator frequency, creating a current field along the thorax. A four-spot electrode configuration over the neck and thorax suggested by Qu, Zhang, Webster, and Tompkins (1986; as reported in Sherwood et al., 1990; cf. Sherwood, Royal, Hutcheson, & Turner, 1992) employing pregelled adhesive electrodes (No. 2238, 3M Red Dot Soft Cloth Electrodes, 6 cm diameter) was used. The distance between the two front electrodes was measured and noted. The impedance cardiogram (ICG) dZ/dt signal was ensemble averaged in synchrony with the ECG R-wave, and the derived measures were averaged over analyzed film segments. Characteristic points (B, E, X) of the inverted dZ/dt signal of ensemble averaged beats were identified automatically after exclusion of abnormal beats and edited when necessary. ECG Q-waves were detected for quantification of *preejection period* (PEP). PEP (in milliseconds) was calculated as the interval from the ECG Q-point to the ICG B-point. PEP is inversely related to left-ventricular contractility and β-adrenergic sympathetic influences on the myocardium. *Stroke volume* (SV, in milliliters) of the left ventricle was estimated using the Kubicek formula (Kubicek, Krnegis, Patterson, Witsoe, & Mattson, 1966). *Left-ventricular ejection time* (LVET, in milliseconds) was calculated as the interval from B- to X-point in the ICG, estimating the time interval from the opening to the closing of the aortic valve (mechanical systole), that is, how long blood is pumped out of the left ventricle from the heart. The *Heather index* (HI, in ohms per second squared) was defined as the ratio of dZ/dt_{max} to Q–E interval (i.e., the electromechanical time interval). This index has been shown to be especially sensitive to changes in cardiac contractility.

Systolic and diastolic arterial pressure (SBP and DBP, respectively, in millimeters of mercury) was measured intermittently with an automatic blood pressure monitor (Dinamap 1846SX, Critikon & GE Healthcare, Chalfont St. Giles, UK) through an arm cuff at the participant's nondominant upper arm. Inflation was initiated at minutes 1, 5, and 9 of the film conditions and at minute 2 of the rest baselines and lasted about 50 s.[4] We decided to not use continuous blood pressure measurement (i.e., the Finapres) because this method may involve an unsystematic drift over time, adding substantial error variance in this long protocol (Ristuccia, Grossman, Watkins, & Lown, 1997).

Pulse wave amplitude at the ear (PA, in arbitrary units), the difference between peak and valley of the pulse waveform, was measured as an index of peripheral vasoconstriction by a plethysmograph transducer (UFI, Morro Bay, CA) attached to the participant's left ear. Customized software was used to calculate beat-to-beat mean amplitude. *Pulse wave transit time to the ear* (PTT, in milliseconds) was indexed by time elapsed between the closest previous ECG R-wave and the steepest upstroke of the peripheral pulse at the ear.

Finger temperature (FT, in degrees Fahrenheit), measured by a thermistor attached to the palmar surface of the distal phalanx of the fifth finger, was averaged over time.

Electrodermal system. A constant-voltage device maintained 0.5 V between 10-mm Beckman Ag/AgCl electrodes filled with isotonic Med Associates (St. Albans, VT) Electrode Paste TD-246 attached to the palmar surface of the middle phalanges of the first and second fingers of the nondominant hand. Before electrode application, participants washed their hand with Ivory soap. The skin conductance channel was analyzed as mean level (*skin conductance level*, SCL, in microsiemens) after movement and electrode contact artifacts had been edited out. The *rate of*

[4]For 3 participants, SBP and DBP were calculated beat to beat from the continuous arterial pressure waveform (Finapres 2300, Ohmeda, Madison, WI).

nonspecific skin conductance responses (SRR, in number of responses per minute) was quantified as the number of skin conductance increases from a zero-slope baseline exceeding 0.025 μS, per minute. *Skin conductance response amplitude* (SRA, in microsiemens) was quantified as the difference between the zero-slope onset and peak of a skin conductance increase exceeding 0.025 μS, applied to all nonspecific fluctuations.

Respiratory system. Two channels of respiration were measured using a plethysmography device (James Long Co., Caroga Lake, NY) connected to pneumographic bellows placed around the abdomen and chest. The top respiration band (thoracic respiration) was placed high on the chest, above the breasts; the bottom respiration band (abdominal respiration) was placed below the naval and above the pants/belt. First, raw signals were converted to calibrated lung volume change using data from the fixed volume bag calibration procedure (Chadha et al., 1982). A least squares fit multiple regression procedure was used to establish weighting coefficients to best predict bag volume from output of the two bands (Morel et al., 1983). *Respiratory rate* (RR, in cycles per minute) and *tidal volume* (Vt, in milliliters) were calculated breath by breath using customized programs (Wilhelm & Roth, 1998). The square root of the mean of squared successive differences (MSSD) values of breath-by-breath tidal volume were subsequently calculated to obtain a measure of *tidal volume variability* (VtV, in milliliters). *Minute ventilation* (Vm, in liters) was computed breath by breath as the inverse of total breath time multiplied by tidal volume. This value was then assigned to the whole duration of that breath in epochs of half seconds. If the onset of the breath did not fall exactly on the onset of the half second epoch, the value for this breath and the following breath were weighted and combined. *Duty cycle* (Ti/Ttot) was calculated as the ratio of inspiratory to total breath time and *inspiratory flow rate* (Vi/Ti, in milliliters per second) was calculated as the ratio of inspiratory tidal volume to inspiratory time.

Expiratory pCO_2 was measured continuously by a calibrated infrared capnograph (N-1000, Nellcor, Hayward, CA) into which air was drawn with a flow rate of 150 mL/min through a 1.2-mm-diameter plastic tube ending in dual prongs placed in the nostrils. End-tidal pCO_2 (pCO_2, in millimeters of mecury), which is close to alveolar and arterial values, was determined as the level at which pCO_2 stopped rising at the end of expirations (cf. Wilhelm, Alpers, Meuret, & Roth, 2001). Only breaths in which pCO_2 waveforms reached a distinct plateau were considered. The criterion for a plateau is that in the last 0.25 s of expiration, values cannot be more than 3 mmHg less than the final maximum. Plateaus were required to be present in 50% of breaths in a measuring epoch for it to be considered.

Scoring of Physiological Data

Raw scores were defined as the arithmetic mean of the physiological data within each experimental condition (10 min for the film presentations, 3 min for the rest baselines). This approach likely leads to a conservative estimate of emotional response specificity, as subjects are unlikely to manifest a strong emotional response at a consistent magnitude at the very beginning and throughout the film period.

If data within conditions were missing, the interpolation method described in Stemmler (1989) was applied. Percentage of missing data for single physiological variables ranged between 1 and 24 of 408 conditions (6 films, 6 baselines, 34 subjects) or 0.25%–5.9% of total conditions recorded.

Reactivity for each variable was defined as follows: First, difference scores were calculated by subtracting from the film raw score the prior baseline score, which was the average of the quiet sitting period immediately preceding each film clip. Second, individual differences from the individual's overall mean were excluded by centering (separately for each variable) the individual's array of scores by subtraction of the individual mean. Third, all centered difference scores of a variable were subjected to the McCall normalization transformation (Krus & Kennedy, 1977).[5] In a fourth step, variables were mapped onto a T scale with a mean of 50 and a standard deviation of 10. A standardized difference score of 60 thus represents an increase of one standard deviation from baseline of the respective measure in response to the emotion manipulation.

Statistical Analysis

Preliminary analyses were performed to test for the absence of differences between the two sets of film clips. Because there was no significant effect of set,[6] the data were collapsed over the set factor.

Manipulation check. Measures of emotional experience and emotional expressive behavior were subjected to planned contrasts (Rosenthal & Rosnow, 1985). To test differences a posteriori, we calculated Tukey honestly significant difference (HSD) post hoc tests (selected for their property of being located midway between conservative and progressive post hoc tests).

Analysis of physiological parameters. All analyses were calculated on standardized difference scores. Physiological variables were first tested for univariate significant differences between emotion conditions (fear, sadness, neutral) by repeated-measures analysis of variance (ANOVA) with Greenhouse–Geisser correction and Tukey HSD post hoc tests for each physiological measure when the ANOVA was significant. Additionally, effect

[5]Results based on the McCall normalization transformed data did not differ from those based on the linear *z*-transformation.

[6]Film sets did not differ significantly with respect to self-reported emotional experience, $F(1,31) = 0.28$, 1.35, 0.12, and 1.17, all n.s. for fear, sadness, valence, and arousal, respectively. A 2 (set) × 3 (emotion) × 23 (parameter) MANOVA showed that the main effect of the set factor was nonsignificant, $\lambda = 1.00$, $F(1,27) = 0.00$, $p < .99$, as were the interaction effects for emotion, $\lambda = 0.84$, $F(2,26) = 2.54$, $p < .10$, and parameter, $\lambda = 0.36$, $F(22,6) = 0.49$, $p < .90$. Analyses of each film set separately gave very similar results and are hence not reported here. Similarly, analyses that included additional control variables showed no significant influence. Gender did not affect physiological responding to the two film sets, $\lambda = 0.99$, $F(1,26) = 0.16$, $p < .69$, n.s., physiological measures, $\lambda = 0.12$, $F(22,5) = 1.66$, $p < .30$, n.s., or responding to emotion, $\lambda = 0.81$, $F(2,25) = 2.89$, $p < .07$, n.s. Whether the participants had seen the film before showed no significant interaction with physiological responding in a covariance analysis, $F(22,5) = 0.57$ and 0.43 for fear-inducing films of sets A and B, respectively, 0.40 and 0.39 for sad films of sets A and B, respectively, and 2.19 for the neutral film of set A (all nonsignificant). No variance was present in the covariant for the neutral film of set B—all participants had never seen the film before; therefore, no covariance analysis could be calculated for this case. Whether participants closed their eyes or looked away similarly showed no significant interaction with physiological responding in a covariance analysis, $F(22,5) = 0.77$ and 1.55 for fear-inducing films of sets A and B, respectively, 0.38 and 5.68 for sad films of sets A and B, respectively, and 1.81 and 0.49 for neutral films of sets A and B, respectively. All were nonsignificant except the sad film of set B, which was also found to be nonsignificant in post hoc testing, that is, the significant covariance effect of "closing eyes" on physiological parameters could not be traced to any single physiological measure that it affected.

sizes were calculated for differentiation of fear versus neutral, sadness versus neutral, and fear versus sadness conditions.

In a next step, we analyzed whether univariate differences found between conditions represent differences in profile elevation or in profile nonparallelism by calculating multivariate analyses of variance (MANOVA). The MANOVA main effect for Emotion tests whether profile *levels* (i.e., intensity) are equal between conditions, whereas the MANOVA interaction effect of Emotion × Parameter tests whether profiles are *parallel* (i.e., will be significant if there exist differences in profile scatter and shape; cf. Stemmler, 1988; Tabachnick & Fidell, 2007). The main analysis of physiological measures was thus based on three separate Emotion × Parameter MANOVAs, with the respective univariate significant variables separating fear versus neutral, sadness versus neutral, and fear versus sadness.

Finally, we tested whether variables identified to differ significantly between experimental conditions could also be successfully used to predict the respective emotion condition. For this, a predictive discriminant analysis (PDA) was calculated. This analysis focuses on predicting group membership by classifying units. PDA derives a set of emotion classification functions from a *practice sample*, which can be reapplied to the same sample in order to derive an ''index of internal structure'' (internal hit rate) or to a *validation sample* in order to derive an ''index of generalizability'' (i.e., external hit rate). The latter was calculated using a jackknifed classification approach that computes the classification leaving out one case at a time and applying the thus derived classification rule to the left-out case, performed for all possible combinations. It must be emphasized that the resultant internal index of structure does not represent a true hit rate, because it derives from a fitted model; true hit rates are estimated by results of external classification. For PDA, the linear classification rule assuming normally distributed data and equal prior probabilities of group membership was used. A casewise deletion criterion instead of the more liberal means-substitution criterion was used to handle missing cases, which resulted in a final data set of 28 participants because values for 6 participants were missing in the electrodermal channels.

Results

Manipulation Check

Participants' emotional experience was successfully manipulated. The three film types (fearful, sad, and neutral) were rated as significantly different by participants on the fear scale of the emotion questionnaire, fear versus sad conditions: $F(1,33) = 29.36$, $p < .001$; fear versus neutral conditions: $F(1,33) = 72.15$, $p < .001$ (post hoc Tukey HSD test of sad vs. neutral conditions: $p < .003$), and the sadness scale of the emotion questionnaire, sad versus fear conditions: $F(1,33) = 163.33$, $p < .001$; sad versus neutral conditions: $F(1,33) = 299.90$, $p < .001$ (post hoc Tukey HSD test of fear vs. neutral conditions: $p < .06$, n.s.), with highest fear ratings for the fear condition and highest sadness ratings for the sad condition. Dimensional emotion ratings also gave the expected results: Fear and sadness conditions were rated more negatively than the neutral condition, fear versus neutral: $F(1,33) = 32.13$, $p < .001$, sad versus neutral: $F(1,33) = 34.81$, $p < .001$, whereas there was no difference in valence between the two emotion conditions, fear versus sad: $F(1,33) = 0.42$, $p < .52$, n.s. Similarly, for self-reports of experienced arousal, fear and sadness conditions were rated more arousing than the neutral condition, fear versus neutral: $F(1,33) = 41.41$, $p < .001$; sad versus neutral: $F(1,33) = 33.41$, $p < .001$; whereas there was no difference in arousal level between the two emotion conditions, fear versus sad: $F(1,33) = 2.62$, $p < .12$, n.s. See Table 2, top panel, for means and standard deviations of emotional self-report.

Results of the analysis of facial emotion expressions as indexed by EMG also support successful induction of target emotions—although these data reflect a somewhat different pattern than the one described above for self-reported emotion experience: The Corrugator Supercilii was significantly more activated in the fearful than in the sad condition, $F(1,33) = 9.29$, $p < .01$, and significantly more activated in the sad than in the neutral condition, $F(1,33) = 11.84$, $p < .01$; similarly, higher activation of the Zygomaticus Major was present in the neutral than in the sad condition, $F(1,31) = 7.83$, $p < .01$, and higher activation was present in the sad than in the fearful condition, $F(1,31) = 4.64$, $p < .05$. Table 2, bottom panel, reports means and standard deviations of facial behavior.

Univariate Analysis of Emotion Effects

Physiological measures were analyzed by univariate repeated-measures ANOVAs, and—if significant—post hoc Tukey HSD tests. Table 3, right side, reports univariate analyses of the emotion effects. Results of the repeated-measures ANOVAs on single physiological variables showed that significant emotion effects were present in the 13 variables HR, SV, PEP, SBP, DBP, PTT, FT, SCL, SRR, RR, Vm, Ti/Ttot, and pCO_2 (with PA reaching marginal significance, $p < .063$). Post hoc Tukey HSD tests indicated significant differences between fearful and neutral conditions for SV, PEP, SBP, DBP, PTT, FT, SCL, SR, RR, Vm, and Ti/Ttot. Significant differences between sad and neutral conditions were present in FT and SRR (with DBP and PA reaching marginal significance, $ps < .052$). Fearful and sad conditions were significantly different with respect to HR, PEP, DBP, PTT, RR, and pCO_2. The activation patterns of physiological measures in conditions of fear, sadness, and a neutral emotional state are depicted in Figure 1.

Table 3, left side, reports the effect sizes for fear versus neutral, sadness versus neutral, and fear versus sadness contrasts. Following Cohen's (1992) taxonomy, large effect sizes were present for the fear versus neutral contrast in DBP, FT, and SRR, as well as for the sadness versus neutral contrast in SRR. Medium-size effects for the fear versus neutral contrast were present in PEP, SBP, PTT, SCL, RR, Vm, and Ti/Ttot. Medium-size effects were

Table 2. *Means and Standard Deviation of Emotion Self-Report and Facial Behavior*

	Fear films		Sadness films		Neutral films	
	M	*SD*	*M*	*SD*	*M*	*SD*
Emotion self-report						
Afraid	3.86	2.54	1.55	1.62	0.25	0.32
Sad	1.09	1.20	6.14	2.13	0.35	0.59
Valence	− 0.80	1.38	− 0.96	1.38	1.36	1.58
Arousal	5.49	2.05	5.14	1.90	3.37	1.67
Facial behavior						
Corrugator Supercilii	268.3	123.7	215.3	73.4	170.1	63.4
Zygomaticus Major	109.6	23.5	125.9	51.9	146.2	78.5

Note. Emotion self-report was on a scale from 0 to 10. Facial behavior was percent of mean baseline EMG during the immediately following film condition.

Table 3. *Statistical Summary for Physiological Measures*

Physiological variable	Abbreviation	Effect sizes			ANOVA		Tukey HSD test		
		d(f-n)	d(s-n)	d(f-s)	*F*	ε	f vs. n	s vs. n	f vs. s
Cardiovascular									
Heart rate	HR	0.30	− 0.35	0.62	$6.52^{a,**}$	.74	n.s.	n.s.	.0019
T-wave amplitude	TWA	0.27	0.24	0.07	1.56^{a}	.95	—	—	—
Respiratory sinus arrhythmia (corrected)	RSA_c	− 0.14	0.18	− 0.26	1.42^{a}	.78	—	—	—
Low-frequency heart rate variability	LF	− 0.37	− 0.14	− 0.19	2.02^{a}	.93	—	—	—
Stroke volume	SV	− 0.48	− 0.29	− 0.39	$5.86^{b,**}$	.77	.0035	n.s.	n.s.
Preejection period	PEP	− 0.64	− 0.17	− 0.41	$6.34^{b,**}$	.98	.0030	n.s.	.039
Left-ventricular ejection time	LVET	− 0.12	0.26	− 0.33	2.09^{b}	.93	—	—	—
Heather index	HI	− 0.10	− 0.26	0.08	0.65^{b}	.85	—	—	—
Systolic blood pressure	SBP	0.54	0.18	0.42	$5.88^{a,**}$	.98	.0039	n.s.	n.s.
Diastolic blood pressure	DBP	0.80	0.42	0.41	$11.49^{a,***}$	1.00	.00014	n.s.	.048
Ear pulse amplitude	PA	− 0.27	− 0.39	0.13	3.10^{a}	.82	—	—	—
Ear pulse transit time	PTT	− 0.67	− 0.19	− 0.58	$9.92^{a,***}$	.96	.00029	n.s.	.0052
Finger skin temperature	FT	− 0.85	− 0.71	− 0.37	$16.71^{a,***}$	.90	.00011	.0015	n.s.
Electrodermal							—	—	—
Skin conductance level	SCL	0.65	0.50	0.41	$9.33^{c,***}$	.82	.00029	n.s.	n.s.
Nonspecific skin conductance response rate	SRR	1.23	1.01	0.57	$32.55^{d,***}$	.81	.00012	.00012	n.s.
Skin conductance response amplitude	SRA	0.27	0.27	0.01	1.51^{d}	.96	—	—	—
Respiratory									
Respiration rate	RR	0.66	− 0.10	0.73	$13.49^{a,***}$	.85	.00030	n.s.	.00015
Tidal volume	Vt	− 0.13	0.07	− 0.26	0.81^{a}	.89	—	—	—
Tidal volume variability	VtV	− 0.10	− 0.23	0.12	0.89^{a}	.98	—	—	—
Minute ventilation	Vm	0.55	0.14	0.32	$4.25^{a,*}$	.95	.016	n.s.	n.s.
Inspiratory flow rate	Vi/Ti	− 0.19	− 0.20	− 0.03	0.94^{a}	.93	—	—	—
Duty cycle	Ti/Ttot	0.52	0.38	0.27	$5.94^{a,**}$	.90	.0030	n.s.	n.s.
End-tidal pCO_2	pCO_2	− 0.27	0.45	− 0.50	$6.32^{a,**}$	.79	n.s.	n.s.	.0022

Notes. f: fear films, s: sadness films, n: neutral films, ANOVA: Greenhouse–Geisser-corrected repeated-measures ANOVA, $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$, $^{a}df = 2/66$, $^{b}df = 2/64$, $^{c}df = 2/58$, $^{d}df = 2/56$.

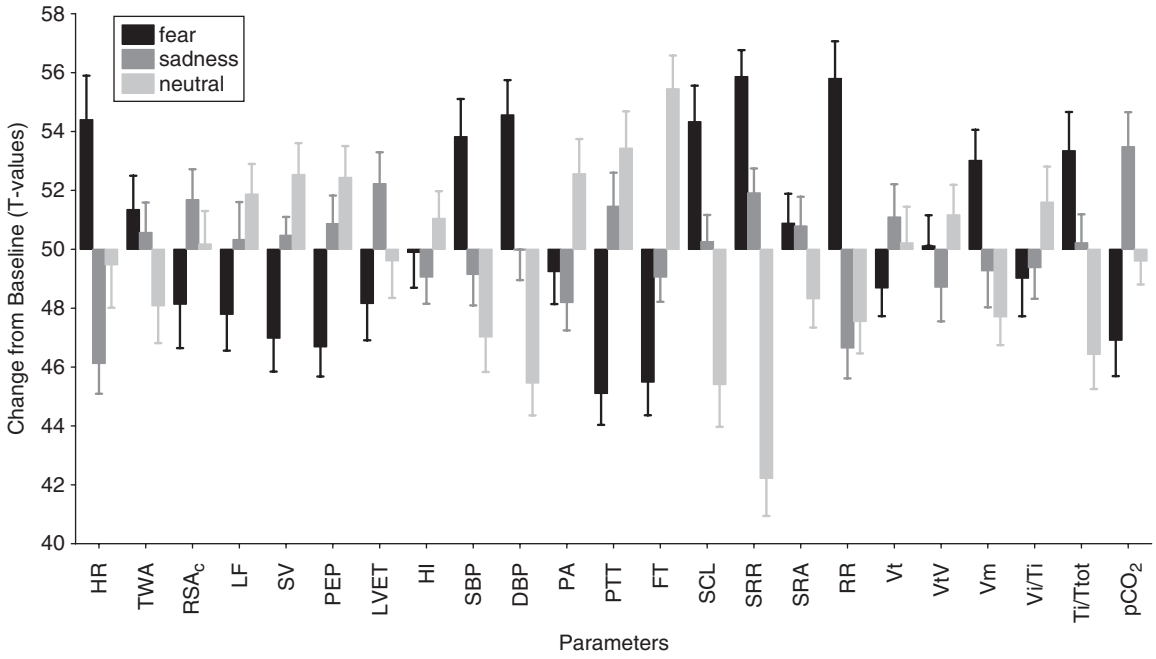

Figure 1. Means and standard errors of autonomic and respiratory parameters for emotion conditions. Whiskers indicate 1 standard error of the mean. HR: heart rate; TWA: T-wave amplitude; RSA_c: respiratory sinus arrhythmia (corrected); LF: low-frequency heart rate variability; SV: stroke volume; PEP: preejection period; LVET: left-ventricular ejection time; HI: Heather index; SBP: systolic blood pressure; DBP: diastolic blood pressure; PA: ear pulse amplitude; PTT: ear pulse transit time; FT: finger skin temperature; SCL: skin conductance level; SRR; nonspecific skin conductance response rate; SRA: skin conductance response amplitude; RR; respiration rate; Vt: tidal volume; VtV: tidal volume variability; Vm: minute ventilation; Vi/Ti: inspiratory flow rate; Ti/Ttot: duty cycle; pCO_2: end-tidal carbon dioxide partial pressure (end-tidal pCO_2).

found for differences between sadness versus neutral conditions with respect to FT and SCL. For the fear versus sadness contrast, there were medium-size effects in HR, SRR, RR, and pCO_2.[7]

Multivariate Analysis of Emotion Effects

To test whether the univariate differences found represent differences in profile elevation or in profile parallelism of the physiological response profile, three separate MANOVAs were calculated for the univariate significant variables separating (1) fear and neutral, (2) sadness and neutral, and (3) fear and sadness.

The MANOVA for the fear versus neutral comparison—2 (emotion: fear, neutral) × 11 (parameter: SV, PEP, SBP, DBP, PTT, FT, SCL, SRR, RR, Vm, Ti/Ttot)—demonstrated both a significant Emotion main effect, $\lambda = 0.67$, $F(1,27) = 13.00$, $p \leq .001$, and Parameter × Emotion interaction effect, $\lambda = 0.22$, $F(10,18) = 6.43$, $p < .001$. This indicates that the observed univariate differences for the fear versus neutral comparison were a result of both intensity as well as shape and scatter differences of the physiological profile between the fearful and neutral conditions.

The MANOVA for the sadness versus neutral comparison, including the two marginally significant variables PA and DBP—2 (emotion: sadness, neutral) × 4 (parameter: DBP, PA, FT, SRR)—resulted in a significant Emotion × Parameter interaction effect, $\lambda = 0.37$, $F(3,26) = 14.54$, $p < .001$, and no significant Emotion main effect, $\lambda = 0.98$, $F(1,28) = 0.49$, $p < .49$, n.s.[8] This points to the fact that univariate differences in the physiological profile between the sad and neutral conditions represented differences in shape and scatter rather than intensity of the physiological profiles.

The MANOVA for fear versus sadness—2 (emotion: fear, sadness) × 6 (parameter: HR, PEP, DBP, PTT, RR, and pCO_2)—yielded a significant Parameter × Emotion interaction effect, $\lambda = 0.49$, $F(5,28) = 5.87$, $p < .001$, and no significant Emotion main effect, $\lambda = 0.94$, $F(1,32) = 2.19$, $p < .15$, n.s., indicating that the univariate differences found for the fear versus sadness contrast again represented differences in shape and scatter rather than intensity.

Pattern Classification Analysis

Predictive discriminant analysis was based on the univariate significant variables, including the marginally significant variable PA, thus consisting of the following 14 variables: HR, SV, PEP, SBP, DBP, PTT, PA, FT, SCL, SRR, RR, Vm, Ti/Ttot, and pCO_2.

Classification rates for internal and external classification are presented as both number and percentage (in parenthesis) of observations in Table 4. Classification hits (i.e., when the predicted emotion condition matched the actual emotion condition) form the diagonal of the classification matrix. An *I* statistic was calculated that is equivalent to an effect size index, indicating that $100 \times I\%$ fewer classification errors resulted using the classification rule than would be expected by chance (Huberty, 1994). Results of the classification analysis performed for physiological variables were each significant at $p < .001$. For the internal classification analysis, good discriminatory power was shown among fearful, sad, and neutral conditions with classification hit rates

Table 4. *Predictive Discriminant Analysis for Internal and External Classification (Jackknifed Analysis)*

Internal classification

	Predicted emotion condition				Relative condition frequency
	Fear	Sad	Neutral	Total	
Actual emotion condition					
Fear	**22 (78.6)**	6 (21.4)	0 (0.0)	28	1/3
Sad	2 (7.1)	**25 (89.3)**	1 (3.6)	28	1/3
Neutral	0 (0.0)	4 (14.3)	**24 (85.7)**	28	1/3
Total	24	35	25	84 = *N*	

Total percent correct classifications: 84.5

Emotion condition	*N*	Observed	Expected	*z*	*p*	Lower bound for 99% interval
Fear	28	22	9.33	5.078	.000	16.198
Sad	28	25	9.33	6.281	.000	19.198
Neutral	28	24	9.33	5.880	.000	18.198
Total	84	71	28	9.953	.000	60.951

$H_o = 0.845$ $H_e = 0.333$ $I = 0.768$

External classification

	Predicted emotion condition				Relative condition frequency
	Fear	Sad	Neutral	Total	
Actual emotion condition					
Fear	**19 (67.9)**	8 (28.6)	1 (3.6)	28	1/3
Sad	6 (21.4)	**18 (64.3)**	4 (14.3)	28	1/3
Neutral	0 (0.0)	7 (25.0)	**21 (75.0)**	28	1/3
Total	25	33	26	84 = *N*	

Total percent correct classifications: 69.0

Emotion condition	*N*	Observed	Expected	*z*	*p*	Lower bound for 99% interval
Fear	28	19	9.33	3.875	.000	13.198
Sad	28	18	9.33	3.474	.000	12.198
Neutral	28	21	9.33	4.677	.000	15.198
Total	84	58	28	6.944	.000	47.951

$H_o = 0.690$ $H_e = 0.333$ $I = 0.536$

Notes. Based on HR, SV, PEP, SBP, DBP, PTT, PA, FT, SCL, SRR, RR, Vm, Ti/Ttot, pCO_2. Classification rates are presented as both number and percentage (in parenthesis) of observations. Classification hits (i.e., when the predicted emotion condition matched the actual emotion condition) are marked in bold face. H_o: observed hit rate; H_e: expected hit rate; *I*: improvement over chance classification. For other abbreviations see Table 3 and Figure 1.

[7]We also analyzed RSA_{uc} and found that $M_{fear} = 47.12$ (1.31), $M_{sad} = 52.37$ (1.06), and $M_{neutral} = 50.51$ (1.17) (numbers in parentheses indicate standard error of the mean). The Greenhouse–Geisser-corrected repeated-measures ANOVA was significant, $F(2,66) = 3.39$, $\varepsilon = .91$, $p < .05$, and post hoc Tukey HSD tests indicated a significant difference for fear versus sadness ($p < .05$). Effect sizes were -0.26 for fear versus neutral, 0.19 for sadness versus neutral, and -0.43 for fear versus sadness.

[8]The same pattern of results emerged when including only the two significant variables FT and SRR in the analysis.

between 78.6% and 89.3% and $I = 0.768$, indicating substantial differences between these three response patterns and good classification into emotion conditions based on the selected set of variables. Similarly, for the external classification analysis (jackknifed classification analysis), classification hit rates ranged between 64.3% and 75.0% with $I = 0.536$, suggesting that even for an unknown sample the derived classification rule would generalize successfully.[9]

Discussion

The present study examined psychophysiological differences between fear and sadness. Physiological responding, as reflected in various measures across cardiovascular, electrodermal, and respiratory systems, was investigated in the context of film clips eliciting fear, sadness, and a neutral emotional state. Our goal was to address two questions: We first asked whether fear and sadness differ both from a neutral condition and from each other based on conventional as well as on newer, seldom-used measures of psychophysiological activation in emotional responding. Second, we asked whether physiological variables can be used to reliably predict the type of emotional state.

Emotional Experience and Facial Expressive Behavior

Measures of self-reported emotional experience confirmed successful manipulation of participants' emotional states in the present study with the target emotion rated highest for the respective emotion condition. The sadness condition also evidenced significantly higher ratings of fear than the neutral condition, whereas there was no evidence of coactivation of sadness in the fear condition (cf. Smith & Lazarus, 1990, for a discussion of the co-occurrence of fear and sadness based on shared appraisal dimensions). This coactivation of fear in the sadness condition did, however, not seem to have a significant influence on physiological response patterns, which differed considerably between conditions. Moreover, dimensional ratings of affective experience did *not* indicate differences between emotion conditions with respect to valence and arousal ratings.

When we considered facial expressions, however, a more complex picture emerged, because EMG recordings suggested a linear increase of Corrugator Supercilii muscle activity from the neutral emotional condition over sadness to fear and a similar linear decrease of Zygomaticus Major muscle activity from the neutral emotional condition over sadness to fear. This divergence between measures of emotion experience, particularly valence ratings, and facial behavior is inconsistent with results from several prior studies using picture presentation (e.g., Bradley et al., 2001; Dimberg, 1986), but has been observed in more complex emotion induction paradigms (e.g., Reisenzein, Bördgen, Holtbernd, & Matz, 2006). Our result points both to the significance of multisystem assessments of emotional responses and to potential differences between emotion-related response patterns from phasic, short-term (e.g., picture presentation) versus tonic, long-term emotion induction (e.g., presentation of film clips). We will return to this distinction and substantiate this interpretation in our discussion on physiological response profiles below.

[9]Adding facial expression EMG data of the Corrugator Supercilii and the Zygomaticus Major did not improve classification accuracy.

Psychophysiological Response Patterns during Fear, Sadness, and Neutral Films

Using univariate and multivariate analyses of variance, differential response patterning of peripheral physiological responding for fear, sadness, and a neutral emotional state was clearly supported: first, by the univariate significant results as well as the various large- and medium-sized effects for a number of physiological variables, which showed differentiated physiological responses in a number of response systems for both the emotion versus neutral contrasts and for the within-emotions contrast, and second, by the subsequent test of differential physiological response patterning based on a multivariate combination of univariate significant variables, which consistently indicated pattern differences. In combination, these results support the hypothesis that fear and sadness are distinguishable from neutral control conditions (rest baseline and film viewing) in a number of parameters. Moreover, response patterns differed *between* the two emotion conditions, not merely because the profiles differed in intensity but because their patterns were different. This indicates that physiological response patterns under fear and sadness were generated by different kinds of activation of the autonomic nervous system.

Responses to fear-eliciting films. The univariate analysis showed that the psychophysiological response pattern to fear-inducing film clips compared to a neutral emotional state was characterized by increased sympathetic cardiovascular activation: SV and PEP were decreased, reflecting β-adrenergic sympathetic influence leading to increased myocardial contractility; SBP and DBP were elevated and PTT and FT were decreased, indicating additional α-adrenergic vascular changes (Stemmler, 2002; Wacker & Stemmler, 2006). Furthermore, the electrodermal response indices evidenced cholinergic sympathetic changes, indicated by increased SCL and SRR. Finally, several physiological parameters demonstrated the involvement of respiratory changes in the fear response, with fast breathing and increased ventilation (increased RR and Vm) and an increase in Ti/Ttot marking decreased expiratory time (cf. Wilhelm & Roth, 1998). Subsequent multivariate analysis of variance showed that univariate significant differences between the fear and neutral condition were due to both intensity and pattern differences. Thus, physiological response patterns in fear as compared to a neutral condition were based on both increased arousal and differentiated autonomic functioning.

Our results are in accord with the majority of findings of investigations of fear in different experimental contexts as reviewed in Table 1. Particularly the cumulative work of Stemmler and colleagues showed that fear entails decreases in SV and PEP, elevations in both SBP and DBP, and decreases in PTT and FT (Pauls & Stemmler, 2003; Stemmler, 1989, 2004; Stemmler et al., 2001). However, our response patterns to fear-inducing film clips are inconsistent with the following: DBP reported by Sinha and Parsons (1996) and Sinha et al. (1992), who induced targeted emotions by imagery; FT reported by Rimm-Kaufman and Kagan (1996), who found a decrease in FT to threatening personal questions, but no change in response to fear eliciting film clips; PTT reported by Krumhansl (1997), where finger rather than ear pulse transit time was analyzed in emotions induced by music; and RR reported by Boiten (1998) from an emotion film study and by Ritz, Thöns, et al. (2005) based on results from a picture-viewing emotion study. For these measures, either no significant effect or an effect in the opposite direction from ours was

observed. Such discrepancies between results suggest situational variation due to induction context, for example, imagery and film (cf. Stemmler et al., 2001). However, other factors may have varied between studies, the impacts of which await evaluation.

Responses to sadness-eliciting films. The univariate analysis demonstrated that the response to sadness-inducing film clips as compared to a neutral emotional state was characterized by changes in both cardiovascular and electrodermal responding: In sadness DBP was elevated (but unchanged from rest baseline) whereas both PA and FT were decreased, indicating peripheral α-sympathetically mediated vascular constriction and decreased central hemodynamic activity (Awad, Ghobashy, Stout, Silverman, & Shelley, 2001; Awad et al., 2006; Obrist, 1981). In addition, electrodermal activation was heightened, as evidenced by increased SRR, a result of increased cholinergic sympathetic arousal (Stemmler, 2002; Wacker & Stemmler, 2006). The multivariate analysis further showed that univariate differences derive from differences in profile shape: The physiological response pattern of sadness was not simply the result of an overall intensity change from a neutral emotional state, but was a distinct autonomic activation pattern.

Observed differences between film-induced sadness and a neutral emotional state largely corroborate the results summarized in Table 1 for sadness: increased DBP (Krumhansl, 1997; Sinha et al., 1992), decreased PA and FT (Gross & Levenson, 1997; Krumhansl, 1997; Kunzmann & Grühn, 2005), and increased SRR (Frazier et al., 2004). Still, results of increased PA during sadness reported by Ekman et al. (1983) and Levenson et al. (1992) do not accord with the physiological patterns observed in the present study. However, those studies differed from ours in two important ways: First, they used finger rather than ear pulse amplitude. The former is more responsive to peripheral vasoconstriction than ear pulse, which may be more responsive to central hemodynamic changes (Awad et al., 2001, 2006). Second, they induced emotion by directed facial action and the reliving of past emotions rather than by film viewing. As for the context of fear, the effects of different induction methods on physiological response patterns remain to be systematically compared.

Responses to fear- versus sadness-eliciting films. Our univariate analyses revealed a number of differences between the two emotional states in both cardiovascular and respiratory response parameters: HR accelerated in fear and decelerated in sadness, PEP was decreased in fear and increased in sadness, DBP was elevated in fear and did not deviate from rest baseline in sadness, and PTT was decreased in fear and increased in sadness. The decrease of PEP and PTT during fear accompanied by the increase of HR and DBP suggests myocardial β-sympathetic excitation (Obrist, Light, McCubbin, Hutcheson, & Hoffer, 1979; Weiss, Del Bo, Reichek, Engelman, 1980), but Contrada, Del Bo, Levy, and Weiss (1995) have interpreted this pattern as reduced parasympathetic inhibition of ventricular myocardial activity. The observed decrease in HR and moderate increase in PTT during sadness points to decreased β-sympathetic cardiac tone (Weiss et al., 1980). In addition, RR was increased in fear and decreased in sadness whereas pCO_2 was decreased in fear and elevated in sadness, signifying fast and shallow breathing during fear and slow and deepened breathing during sadness (for similar findings, cf. Boiten, Frijda, & Wientjes, 1994; Philippot, Chapelle, & Blairy, 2002).

Our multivariate differences documented a nonparallelism of profiles between fear and sadness, again showing that the emotional differences were not just intensity differences, but differences in autonomic patterning or "response specificity." Comparing our findings for fear and sadness induced by films to the response patterns in Table 1 leads to the conclusion that our findings (a) are consistent with the majority of prior studies investigating fear and sadness in a variety of contexts, (b) are inconsistent with some results, and (c) add for consideration a number of response patterns that have not to our knowledge been previously documented. Our findings of increased HR, DBP, and RR and decreased PEP and pCO_2 for fear are consistent with the majority of other studies on psychophysiological response patterns of laboratory-induced fear. However, as seen in Table 1, decreased HR, no change in DBP, and both decreased RR or no change in RR have been reported for fear in some studies. HR decrease seems to occur particularly in the context of fear induction by picture viewing, as was the case in Bernat et al. (2006), Bradley et al. (2001), and Dimberg (1986), whereas other fear-inducing contexts prompted HR acceleration (including real-life, directed facial action, films, music, imagery, and reliving/recall). The observation that affective picture viewing entails different response patterns than other emotion induction paradigms could also be an explanation for the difference between the findings of Ritz, Thöns, et al. (2005), who reported decreased RR for fear, and those of our study as well as the large majority of other studies, which consistently found RR increase as a response to fear induction. Moreover, although our finding of decreased PTT parallels findings within a real-life emotion induction paradigm (Stemmler et al., 2001) and the directed facial action task (Levenson et al., 1992), it is at odds with the report of Krumhansl (1997) for fear induction by musical stimuli. Perhaps emotions induced by arts such as music are qualitatively different from everyday-life emotions that serve everyday adaptive behavioral functions (Zentner, Grandjean, & Scherer, 2007).

For sadness, the observed decrease in HR accords with only 4 of the 13 studies reporting on HR changes during sadness: The other studies found sadness to increase HR. This divergence cannot solely be ascribed to differences in emotion elicitation, because both HR acceleration and deceleration have been reported with film-induced sadness. The observation of decreased RR during sadness is in accord with the majority of studies in the literature, except those of Boiten (1998), who reported no significant change in respiratory patterns during film-induced sadness, and of Levenson et al. (1992), who found increased RR in the directed facial action task. This latter result, however, may have been because sadness is a relatively difficult facial emotional expression to produce, possibly entailing effort-related increases in RR (Boiten, 1996). Divergent findings for DBP during sadness were mentioned above. Particularly interesting in the present context are the findings of increased PEP, PTT, and pCO_2 during the sad condition, which, as far as we know, have never been reported before.

Conclusions. Taken together, our results generally confirm the presence of the fight or flight response during fear (Cannon, 1929; Graham, 1979; Turpin, 1986) and in one response measure confirm the presence of the conservation–withdrawal response during sadness (Bohus et al., 1988; Engel, 1977; Schmale, 1958; Schneiderman & McCabe, 1989). We found the predicted HR acceleration, shortened PEP, elevated blood pressure, increased SCL, faster RR, and decreased end-tidal pCO_2 during fear (e.g.,

Bosch et al., 2001; Lovallo, 2005). We could not confirm the expected increase in cardiac output but found evidence to the contrary: The product of HR and SV indicated a reduction rather than an increase in cardiac output while viewing the frightening films. With our measures we could not test whether the bronchioles dilated with fear. Thus, in summary, fear elicited by presentation of films produced a defense reaction (Berntson, Boysen, & Cacioppo, 1991; Campbell, Wood, & McBride, 1997; Dimberg, 1990b) with predominant sympathetic activation and parasympathetic deactivation, respiratory excitation, and heightened Corrugator Supercilii muscle activity.

In sadness, we were only able to observe the hypothesized HR deceleration (e.g., Bosch et al., 2001; Ritz, Steptoe et al., 2000) and not the predicted shortened PEP or elevated SBP. We could not investigate vagal respiratory effects; to assess these we would have needed to measure oscillatory resistance. In summary, sadness induced by films in our study produced a state of decreased β-sympathetic myocardial activation (deactivation in central cardiac measures), increased α-adrenergic and cholinergic sympathetic activation (increased activation in peripheral vascular and electrodermal measures), deep and slow breathing, and increased Corrugator Supercilii and Zygomaticus Major muscle activity.

The pattern for viewing of neutral clips as compared to a non-film-viewing baseline can be summarized as a relaxation response (Wallace & Benson, 1971), because SCL markedly decreased, and vascular resistance and RR decreased without change in end-tidal pCO_2 (cf. Jevning, Wallace, & Beidebach, 1992). Similar decreases in sympathetic and respiratory activation with no effects on parasympathetic measures were reported for moderate relaxation by Schleifer and Ley (1994). However, a design with randomized order of the rest and neutral film conditions is needed to confirm this interpretation, because many activation measures decrease over time in the absence of stimulation.

Implications for Assessing Fear and Sadness

Clearly, our body reacts with emotions, but which reactions are the best indicators of these changes? The present study included a number of less commonly used measures of cardiovascular, electrodermal, and respiratory nervous system activation in order to evaluate their discriminatory power in differentiating fear and sadness.

By effect size, SRR was the single most sensitive variable for differences between fear and neutral as well as sadness and neutral conditions of film viewing. These contrasts were also sensitively differentiated by FT and SCL. In addition, PEP, SBP, DBP, PTT, RR, Vm, and Ti/Ttot were important for discriminating between the fear and neutral films, underscoring an important role of β-adrenergic cardiovascular measures in this contrast (Berntson et al., 1994; Cacioppo, Uchino, & Berntson, 1994; Lewis, Leighton, Forester, & Weissler, 1974). RR was a very sensitive measure for distinguishing between states of fear and sadness in the context of film viewing, followed by HR, PTT, SRR, and pCO_2.

Thus, discriminating an emotional condition (fear, sadness) from a neutral emotional condition primarily involved electrodermal and vascular measures, whereas discriminating fear from neutral additionally drew from cardiac and respiratory parameters. The discrimination between fear and sadness was based on multisystem parameters, including cardiovascular, electrodermal, and respiratory measures. Although these measures have established themselves in physiological emotion research, typically only a few measures have been represented in a single study, in spite of pleas to the contrary (e.g., al'Absi et al., 1997; Hodes, Cook, & Lang, 1985; Rottenberg, Kasch, et al., 2002). One measure that we found useful, end-tidal pCO_2, has to our knowledge been reported only once before in film studies of emotion (Ritz, Wilhelm, Gerlach, Kullowatz, & Roth, 2005), without which the interpretation of RR and Vt changes would have been incomplete. In summary, our results confirm the importance of the classical measures of electrodermal activity, skin temperature, and heart rate (cf. Cacioppo et al., 1993), but also point to the importance of including respiratory indices as well as more specific cardiovascular measures that can differentiate to some extent α- and β-adrenergic sympathetic activation (e.g., Contrada et al., 1995).

Surprisingly, SV and TWA, both primarily reflecting β-adrenergic sympathetic activation, were important neither in the discrimination between fearful and sad nor between fearful and neutral conditions of film viewing. For similar results on discriminatory lack of SV, see Nyklicek et al. (1997). Reduced TWA during fear, as reported, for example, by Stemmler et al. (2001), was not replicated by our results.

Moreover, contrary to expectations, RSA, an index primarily reflecting vagal cardiac control, was no longer an important discriminator for the fear versus sadness contrast after being corrected for respiratory influences. If we had not made this correction, we would have incorrectly parsed the relative activation of the sympathetic and parasympathetic systems, as modeled by Berntson, Cacioppo, et al. (1991) (see footnote 7): RSA_{uc}, a measure of vagal control confounded with respiratory changes (e.g., Grossman et al., 1991), and PEP, an inverse measure of sympathetic control, were both decreased in the fear condition, indicating vagal withdrawal and sympathetic activation, a pattern of reciprocal control. In the sad condition, in contrast, HR deceleration went along with no change in PEP and increased RSA_{uc}, a pattern reflecting uncoupled parasympathetic activation (Berntson, Cacioppo, et al., 1991). However, as our results indicated, the experimental conditions differed markedly on respiratory indices, specifically so for RR, Vm, Ti/Ttot, and pCO_2. Because it has been previously shown that respiratory influences on RSA may moderate the relationship between RSA and cardiac vagal tone (Grossman et al., 1991), we employed a paced breathing calibration procedure to improve estimation of cardiac vagal tone (Wilhelm et al., 2004; see also Ritz & Dahme, 2006). The breathing-corrected RSA index no longer differed significantly between experimental conditions. It remains to be answered by future research whether the fact that the breathing-corrected RSA index no longer indicated a significant change in vagal influence in the present study was a result of strong confounding respiratory changes or of the threefold error variance that enters into the corrected measure of RSA (see also the recent debate on this issue by Denver, Reed, & Porges, 2007, and Grossman & Taylor, 2007).

Implications for Emotion Models

The complex pattern of cardiovascular, electrodermal, and respiratory activation present in our data is likely best captured by an empirically derived multi-response-system model (Fahrenberg & Foerster, 1982; Fowles, 1980) than by a unitary construct of activation or arousal. The latter model appears to be inadequate for explaining our results, especially when considering that the film clips were reported to be essentially equivalent on the valence and arousal dimensions of affective space (see, e.g.,

Bradley & Lang, 2000), yet autonomic responses differed considerably in pattern, not just in intensity.

Bradley and Lang's model proposes a common emotional system consisting of a bimotivational structure with subsystems of appetitive and defensive motivation, each of which varies on a dimension of intensity or arousal. Reports of valence index which motivational system is active, whereas reports of arousal index magnitude of activation or engagement within each system. If one were to apply the affective space model to autonomic responding, skin conductance and heart rate reactions would be expected to be similar for the two films (because they are on a similar place in the affective space spanned by the valence and arousal dimensions), which clearly was not the case in the present study. Perhaps the affective space model applies only to short-term emotion elicitation of a few seconds and initial appraisal processes, but not to longer term emotion elicitation, the kind more common in naturalistic settings and more compatible with cognitive accounts (Johnson & Multhaup 1992; Leventhal & Scherer 1987; Power & Dalgleish 1997; Scherer, 2001; Teasdale & Barnard 1993). This interpretation is also consistent with the observation that emotional response patterns from the picture viewing paradigm are in some cases (e.g., HR, RR) notably different from other, often longer term emotion manipulations such as film viewing, listening to music, or real-life emotion induction.

Implications for Classifying Physiological Responses to Emotional States

The second aim of the present study was to determine whether the combination of physiological variables found to reliably differ both univariately and multivariately between the investigated emotion conditions could be used to successfully predict emotional state. This question is particularly relevant for the applied domain of affective computing, one aim of which is to enable the computer to detect human emotions and adapt its "behavior" to the discerned emotional state of the user (cf. Picard, 1997; Picard, Vyzas, & Healey, 2001). Based on a classification function over the univariate significant variables, we were able to derive a classification function that mapped observations of physiological response patterns to the corresponding emotional condition with average hit rates of 84.5% and 76.8% fewer classification errors than would be expected by chance when reapplying the classification rule to the training sample. Rates were still high when applying the classification rule to an unknown test sample: Average hit rate was 69.0% and improvement over chance classification was 53.6%. Compared to chance classification of 33.3%, this analysis produced a high percentage of correct classification between experimental conditions. Furthermore, classification errors of fear were primarily misclassifications as sadness, not as the neutral emotional state. Similarly, sadness was mainly misclassified as fear, rather than as neutral. Interestingly, however, confusion of neutral observations occurred primarily with sadness, not fear.

These results indicate that fear, sadness, and the neutral emotional state can be identified successfully by relying solely on the complex emotional activation space spanned by physiological variables. Particularly, because fear has been discussed as a rather difficult emotion to differentiate from a neutral control condition (e.g., Gross & Levenson, 1995; Philippot et al., 2002), this outcome seems rather promising. These results are comparable or even superior to classification rates reported by similar studies, ranging from 52.9% to 90% for fear and 20% to 87.5% for sadness (Christie & Friedman, 2004; Lisetti & Nasoz, 2004; Nasoz, Alvarez, Lisetti, & Finkelstein, 2004; Nasoz, Ozyer, Lisetti, & Finkelstein, 2002; Nyklicek et al., 1997; Sinha & Parsons, 1996).[10] Particularly, when considering that classification accuracy remained consistently high even under the jackknifed resampling analysis, our results may be taken as a robust estimate.

Limitations and Future Directions

One limitation of the present study was its relatively small sample size. Moreover, our participants were drawn from a narrow age range. Our stimulus materials, varying by emotion, additionally differed in ways that we did not control, such as brightness and thematic complexity. Tearful crying might have had special effects that we could not evaluate because too few participants cried to allow for a separate analysis. A further limitation to generalizability was our use of startle probes throughout the films. However, if anything, presenting startle probes would have decreased our chance for finding differences across films, providing yet another way in which our findings may, in fact, be a conservative estimate of true emotion-related differences.

Moreover, it is unlikely that participants began to experience strong emotional feelings right from the beginning of the film clip or maintained those feelings at a constant level to the end, so averaging over the entire 10 min probably led to underestimations of emotional effects. Analyses of shorter time intervals of the data would be useful if "emotion markers" could be defined. A new method for accounting for the waxing and waning of an emotion has recently been validated (Mauss, Levenson, Wilhelm, McCarter, & Gross, 2005).

Results of the pattern classification analysis may have been influenced by our choice and number of physiological parameters. Classification accuracy calculated by PDA may decrease if variables entered into the analysis do not add to discrimination between groups: Models with fewer predictors may be less biased and more precise (Hora & Wilcox, 1982). Had other physiological parameters been added or substituted, the variance overlap that might have occurred could have changed the roles of some physiological parameters from discriminative variables to moderator or suppressor variables, thus affecting the patterns of correlation structure (Fridlund, Schwartz, & Fowler, 1984). Although we demonstrated good generalizability of our results employing a statistical resampling technique, the persistence of similar patterns of physiological parameters across several studies is the ultimate evidence for representativeness and replicability of the calculated patterns.

The present experimental design was further limited by the fact that different models of emotional response specificity (Stemmler, 1989, 1992) could not be tested against each other. We can only conclude that our results clearly reject emotional nonspecificity, as most prominently voiced by Cannon (1929). However, we cannot draw any conclusion as to whether our data favor absolute, context-deviation, or prototypical-behavior emotional specificity, because we used only one way of eliciting emotion. Other methods (slides, relived emotion, imagery, producing facial expressions) may have other, specific physiological profiles. The direct comparison of these methods in a comprehensive design is a reasonable future goal for research.

[10]Reported studies used a comprehensive range of physiological measures and a representative subject sample. Although this article focuses on linear discriminant function, other classification approaches are possible, such as *k*-nearest neighbor or multilayer perceptron.

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