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Running head: AUTONOMIC NERVOUS SYSTEM

Principles of Autonomic Nervous System Assessment for Studying Emotion in Education and Achievement Settings

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Abstract

The present chapter presents an approach to studying emotion in education and achievement settings through the noninvasive assessment of autonomic nervous system (ANS) activity. Part one outlines structure and function of the ANS, introduces three principles central to the conceptualization of ANS activity—bidirectionalism, allodynamic control, and bivariate autonomic space—and identifies the autonomic index function of cardiovascular, electrodermal, and respiratory measures. Part two illustrates application of the three principles of ANS activity in experimental paradigms relevant to the study of emotion in education and achievement settings. We highlight bidirectional ANS–brain effects in the context of test anxiety; allodynamic control of effort investment in cognitive task performance; and assessment of autonomic effects of achievement emotion according to the conceptualizatin of bivariate autonomic space. Part three outlines avenues for future research on emotion in education and achievement settings afforded by the application of the three principles of ANS activity.

Keywords: autonomic nervous system; psychophysiology; emotion; achievement motivation

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> Klara [. . .] was working on the medium difficulty problem. In the first few minutes, she experienced anxiety, which was discernible from her nonverbal expression. . . she confirmed that she indeed was anxious. . . The heart rate at about this time accelerated significantly. . . Similarly, as Klara continued working on the problem she experienced anger. This was evident in her face too. . . The heart rate measure also revealed a significantly increased heart rate. . . Klara confirmed experiencing anger. As Klara completed about 75% of the task, enjoyment surfaced. Corresponding [heart rate changes] were not significant [. . .], but the clear drop from those of anger and anxiety suggests that the associated decrease was consequential. . . She mentioned being happy as she developed confidence in executing the task. Toward the end of the task, Klara reported being anxious. . . In spite of Klara's subjective experience of anxiety, neither her nonverbal expression nor her heart rate signaled the emotion (Ahmed, van der Werf, & Minnaert, 2010, p. 146f).

Emotion can be conceptualized as a multi-component response, elicited by appraising an event as relevant to personal goals, needs, or values, with coordinated effects on subjective feeling, physiology, and motor expression (Scherer, 2009). Consistent with this definition, Ahmed et al. (2010) assessed students' emotions in the classroom through a multi-method approach: Video-stimulated recall interview was used for measuring the experience of emotional feelings. Heart rate monitoring assessed the physiological response component of emotion. Video recordings of the students' face and torso were coded for nonverbal emotional expressions. Findings of this case study, as illustrated in Figure 1, suggest that emotionally potent events during educational episodes indeed lead to concurrent changes in emotional feelings, heart rate, and nonverbal emotional expressions.

Still, much remains to be learned about the autonomic physiology of emotion in education and achievement settings. Here, we want to raise the following three questions: (1) What is the relation between emotion and autonomic nervous system (ANS) activity? In particular, how should we conceptualize the direction of influence? That is, do autonomic responses only have an outcome function, or do they also have a feedback function, influencing central activation state, cortical arousal, and attention? (2) What regulatory function do emotions play in ANS activity? Do emotions represent instances of autonomic perturbation or adaptation? What is the assumption of autonomic activation for optimal task performance—increased, decreased, or unchanged autonomic activity? And (3) what are the underlying autonomic controls, i.e., sympathetic and/or parasympathetic contribution to observed effects? That is, in order to derive a veridical reflection of ANS activity in emotion, what autonomic measures ought we assess?

To answer these questions, we first introduce concepts relevant to the prediction, measurement, and interpretation of functional ANS patterns of emotion, identifying three central principles. Then we discuss applications of these principles in laboratory-based research studies relevant to education and achievement settings. Finally, we return to Ahmed et al.'s (2010) case study to outline future research directions by applying each of these principles.

The Autonomic Nervous System

The ANS is a major output system of the brain. Functional activation patterns are hypothesized to be at the core of autonomic responding in emotion (Kreibig, 2010).

Prediction, measurement, and interpretation of functional ANS patterns of emotion, first, requires knowledge of the structure and functioning of the ANS. Second, it ought to be based on sound models of ANS reactivity in emotion. Third, it needs to be guided by appropriate selection of autonomic measures of emotional responding. We discuss each of these three points in the following.

Structure and Functioning of the Autonomic Nervous System

The ANS, as illustrated in Figure 2, consists of a system of nerves that regulates organ functioning throughout the human body, including the viscera, vasculature, glands, and other tissues, except striated muscle fibers (Langley, 1903, 1921; Jänig, 2003).[1] Both efferent and afferent pathways are involved in autonomic regulation. Efferent, or descending, pathways relay peripherally directed motor commands to visceral target organs. Afferent, or ascending, pathways return sensory input from the viscera to central processing structures.

The ANS comprises several subsystems, including the sympathetic (SNS) and parasympathetic (PNS) nervous systems. Their distinction is based on the anatomical structure of the autonomic innervation from the central nervous system to peripheral target tissues. The SNS originates in thoracolumbar regions of the spinal cord and is relayed at the paravertebral ganglia, resulting in shorter pre- and longer post-ganglionic neurons. It releases acetylcholine at preganglionic neurons and norepinephrine at postganglionic neurons, which activates adrenergic receptor cells on the peripheral target tissues. Two exceptions exist to this general structure of the SNS. First, sympathetic postganglionic neurons innervating sweat glands release acetylcholine for the activation of muscarinic receptors. Second, the adrenal medulla is directly innervated by sympathetic preganglionic neurons and releases norepinephrine and epinephrine into the blood stream.

The structure of the PNS contrasts with that of the SNS. The PNS originates in craniosacral regions of the spinal cord and is relayed at ganglia close to or embedded into the target organ, resulting in longer pre- and shorter post-ganglionic neurons. It releases acetylcholine at both pre- and postganglionic neurons that activates nicotinic receptors at postganglionic neurons and muscarinic receptors at the target organ.

Every organ is innervated by one or both of the sympathetic and parasympathetic outflows. As shown in Figure 2, the SNS innervates the heart, smooth musculature of blood vessels, erector pili muscles, pupils, lungs, evacuative organs, sweat, salivary, and digestive glands, adipose tissue, liver cells, the pineal gland, and lymphatic tissues, as well as the adrenal medulla. The PNS innervates the heart's pacemaker cells and atria, smooth muscles and glands of the airways, intraocular smooth muscles, the smooth musculature, exocrine and endocrine glands of the gastrointestinal tract, pelvic organs (lower urinary tract, hindgut, reproductive organs), epithelia and mucosa throughout body, exocrine glands of the head, as well as some intracranial, uterine, and facial blood vessels not contributing to blood pressure regulation.

Autonomic regulation is normally fast, occurring within a subsecond time range. Autonomic regulation is moreover highly differentiated. A precise neural regulation of body functions is achieved by an anatomically and physiologically distinct organization of the SNS and PNS that consists of several different, functionally distinct subsystems. Each subsystem is associated with a different type of target tissue. Very little or no cross-talk exists between these different peripheral pathways (Jänig & McLachlan, 1992b, 1992a; Jänig & Häbler, 2000). This highly differentiated organization supports adaptive responses of the body during different types of behavior, including emotional responding. Highly differentiated effects of the SNS and PNS on target organs can thus bring about a large repertoire of distinct autonomic responses (Folkow, 2000; Jänig, 2003), constituting the basis for differentiated autonomic responses in emotion.

Models of Autonomic Nervous System Reactivity in Emotion

Causal Relation of Autonomic Change and Emotion What is the relation between emotion, feelings, and autonomic change? This question is as old as the field of emotion research, but none the less still controversially discussed.

At the end of the nineteenth century, William James (1884, p. 189) proposed that 'the bodily changes follow directly the *perception* of the exciting fact, and that our feeling of the same changes as they occur *is* the emotion.' This thesis equates the sensation of bodily changes to the experience of emotional feelings. A similar relation was proposed in Lange's (1885) writings. In present-day emotion research, this afferent model of autonomic emotion effects is found, among others, in the somatic marker hypothesis (Damasio, Tranel, & Damasio, 1991; Damasio, 1996; see Dunn, Dalgleish, & Lawrence, 2006, for a comprehensive discussion of peripheral feedback theories of emotion). Accordingly, afferent signals of emotion-elicited autonomic change are forwarded to higher brain regions, where such signals support decision making in complex situations. Arising "gut feelings", reflected in anticipatory electrodermal responses, may promote adaptive decision making (Bechara, Tranel, Damasio, & Damasio, 1996). Thus, central to the *afferent view* of autonomic emotion effects are the modulatory inputs of autonomic activity on the experience of feeling and the facilitation of information processing demands of the emotional situation at hand.

The *efferent view* of autonomic emotion effects dates back at least as far as to Walter Cannon's work at the turn of the century. Cannon (1927) vehemently questioned the capability of the ANS to provide the feedback signals assumed by the afferent model of autonomic emotion effects. Still, he clearly acknowledged autonomic outflows to the periphery, where these would lead to sympathetic arousal effects in emotion. Efferent effects of emotion also constitute a center piece of various present-day models of emotion.

Discrete emotion theory assumes that innate neuromotor programs produce a distinctive physiology for each basic emotion in support of emotion-specific behaviors (Ekman, 1972; Izard, 1977; Tomkins, 1962, 1963; for recent views see Russell, Rosenberg, & Lewis, 2011, and contributions). Motivational models of emotion suggest that emotion activates motivational brain circuits that in turn activate autonomic structures in preparation for defensive or appetitive actions (Lang, 1994; Lang & Bradley, 2010). Autonomic change occurring as part of emotion is also believed to protect bodily functioning (e.g., Stemmler, 2004). Thus, according to this view, emotion directly leads to autonomic change. The large majority of research on emotion has been based on this efferent conceptualization of autonomic effects of emotion.

Recent perspectives expressed within appraisal theory of emotion integrate both afferent and efferent views of autonomic emotion effects, recognizing bidirectional interactions in the control of affective and autonomic reactivity (Harrison, Kreibig, & Critchley, in press). The efferent output role of the ANS is emphasized in the conceptualization of ANS activity as a response component of emotion (Scherer, 2009). Appraisal outcomes are believed to result in feed-forward control signals that centrally orchestrate coordinated adaptive responses in physiology, motor expression, and action tendencies. The afferent input role of the ANS is emphasized in the conceptualization of the feeling component of emotion as reflecting a “multimodal integration of synchronized changes in component processes” (Scherer, 2004). Feelings are viewed as a central representation of the appraisal-driven changes occurring in emotion, assuming a feedback mechanism from the various emotional response components. This conceptualization also highlights the important distinction between emotion and feeling.

Independent of the causal direction of emotion and autonomic change, no one-to-one relation can be assumed between the two (Cacioppo & Tassinary, 1990; Kreibig, Schaefer, & Brosch, 2010). Emotion may instigate autonomic change, but so do physical activity and other non-emotional psychological factors. Autonomic afference may give rise to emotional feelings, but so do evaluative processes. Both emotion and autonomic responses then need to be viewed as multiply determined.

Regulatory Targets and Mechanisms of Autonomic Change Next, the question arises how autonomic effects of emotion can be integrated into models of autonomic regulation. The classical homeostasis model of autonomic regulation (Cannon, 1929, 1932) assumes that autonomic change is targeted at maintaining some internal set point of the *milieu intèrieur* that has been perturbed. This basic physiological principle of equilibrium, constancy, stability, or homeostasis (from the Greek "similar" and "standing still") is instantiated as a negative feedback loop. Blood pressure increases, for example, are countered by decreases in heart rate and cardiac contractility. This regulatory pattern is known as the baroreceptor reflex.

Whereas the homeostasis model assumes both a fixed set point and fixed operating characteristics of autonomic regulation, the homeodynamic model (Cannon, 1928; Dworkin, 1993) allows for variable operating characteristics (e.g., sensitivity, linearity, temporal dynamics, and stability) while maintaining the assumption of a fixed set point (Berntson & Cacioppo, 2007). Purely homeostatic processes operate compensatorily upon disturbance of a parameter. In contrast, homeodynamic regulation acknowledges modulation of responses by both anticipatory processes to maintain homeostasis as well as by stress processes that may be adaptive, but not homeostatic, such as the concurrent increase of blood pressure and heart rate during stress, suggesting a reduction in sensitivity of the baroreceptor reflex.

Alteration of the physiological set point is incorporated into models of heterostatic and allostatic control. Whereas the model of heterostatic control acknowledges the effect of external pathogens on physiological set points (Selye, 1973), the model of allostatic control also acknowledges that internal processes can adjust the set point (Sterling & Eyer, 1988). For example, a fixed set point can account for thermal cooling under hyperthermia; a variable set point can additionally account for the occurrence of fever during sickness.

The model of allodynamic regulation subsumes the wide range of regulatory processes represented by the concepts of homeostasis, homeodynamic regulation, heterostasis, and allostasis. The allodynamic model overturnes the rigid structure of closed regulatory loops by recognizing that visceral reactions may not always be rigidly regulated about a fixed set-point level, nor are operating characteristics necessarily fixed (Berntson & Cacioppo, 2007). Rather, it suggests that these can be adaptively varied to face existing or anticipated demands. Higher-order brain areas, in particular rostral structures of the hypothalamus, amygdala, and cerebral cortex—regions that give rise to emotion and other psychological phenomena—have the potential to modulate the operating characteristics of brainstem homeostatic mechanisms, such as the baroreceptor reflex. These regions can generate highly flexible patterns of autonomic outflow, which are inconsistent with a simple homeostatic model, in order to attain adequate autonomic support. The allodynamic model of autonomic control hence explicitly recognizes emotion as an adaptive regulatory mechanism.

Autonomic Determinism of the Functional State of Visceral Organs Finally, it remains to be addressed how autonomic activity can be (non-invasively) measured. Assessment of visceral target organ responses, such as heart rate or skin conductance level, for inferring ANS activity is built on the assumptions that (1) the functional state of visceral organs is governed at least in part by autonomic influences; (2) functional measures of visceral organs can provide veridical reflections of autonomic states; and (3) autonomic regulation reflects broader adaptive states of the organism (Berntson, Cacioppo, & Quigley, 1991). The model assumed to underly autonomic organization and control of visceral target organs determines what inferences can be drawn from measures of visceral target organ responses about central regulatory modes.

Visceral target organs have traditionally been viewed to be dually innervated by the two autonomic branches. These were believed to exert functionally antagonistic effects and operate in reciprocity. Thus, autonomic control was conceptualized to vary on a single dimension from sympathetic dominance at one end to parasympathetic dominance at the other end (Cannon, 1932), operating in unity throughout the entire body. Such effects are, for example, observable at the heart, which is a dually innervated organ and opposing effects—heart rate acceleration and deceleration—are brought about by sympathetic and parasympathetic stimulation, respectively. Reflexive responses of the heart, such as cardiac adaptation to orthostatic challenge or the baroreceptor reflex, are characterized by functional reciprocity, i.e., increasing activity in one branch is associated with decreasing activity in the other (Cacioppo, Berntson, & Binkley, 1994). According to this view, assessment of activity of one target organ would suffice in order to infer system-wide sympathetic or parasympathetic control dominance.

Exceptions to this model are, however, quite common (Berntson et al., 1991): Single rather than dual-system innervation is the case for most target tissues. Antagonistic effects of the SNS and the PNS are—in contrast to common conception—rather rare (Koizumi, Terui, & Kollai, 1983). And unlike reciprocal operation, the systems typically work either synergistically or under separate functional or temporal conditions. These deviations are to be expected, since distinction of the ANS into SNS and PNS were originally based on purely anatomical rather than functional considerations, as pointed out above. The doctrine of functional reciprocity has consequently been superseded by the view that sympathetic and parasympathetic activity is organized within a two-dimensional space, as formalized in the model of autonomic space (Berntson et al., 1991). This model assumes that activity in the two ANS divisions may operate either coupled or uncoupled. Coupled modes include the traditional view of reciprocal functioning as well as modes of co-activation and co-inhibition. In uncoupled mode, the two autonomic divisions function independently.

Berntson, Sarter, and Cacioppo (1998) suggested that descending influences particularly from rostral brain systems of the hypothalamus, amygdala, and cerebral cortex—as pointed out above, regions that give rise to emotion and other psychological phenomena—yield nonreciprocal modes of autonomic activation in behavioral contexts. Consistent with this, recruitment of flexible autonomic response modes has been documented in the context of various psychological challenges (Berntson et al., 1994).

Autonomic Measures of Emotional Responding

Because modes of autonomic control may be more closely related to emotional states and processes than simple measures of end organ states, it is of interest to infer the specific mode of autonomic control from activation measures of visceral target organs (Berntson et al., 1998). Table 1 presents an overview of the response measures discussed in the following.

The bivariate model of autonomic space (Berntson et al., 1991) has important implications for the selection of autonomic measures. Given that the heart is a dually innervated organ, both autonomic branches can evoke heart rate changes. This means that heart rate increases can result from increased sympathetic influence, decreased parasympathetic influence, or both. Thus, heart rate is a relatively unspecific index of autonomic functioning.

Trying to disambiguate the heart rate response through measures of the electrodermal system (e.g., skin conductance level, response rate, and amplitude) would remain inconclusive. First, skin conductance measurements can only provide information about sympathetic activation, given that the electrodermal system is solely innervated by the sympathetic nervous system. Second, generalization of sympathetic activation states based on skin conductance measurement to other organs is limited by the fact that its influence is mediated by cholinergic rather than adrenergic transmitters, as is the case for other sympathetic effector organs (Schütz et al., 2008; Shields, MacDowell, Fairchild, & Campbell, 1987). Distinct autonomic communication pathways as well as distinct effector organs may give rise to what has been termed directional fractionation (Lacey, 1959), where autonomic measures indicating sympathetic activation at one organ and sympathetic deactivation at another.

Specific indices of sympathetic and parasympathetic effects on dually innervated organs are thus of key interest. At the heart, for example, pre-ejection period and respiratory sinus arrhythmia can distinguish sympathetic and parasympathetic activation components. Pre-ejection period is the time interval from the beginning of electrical stimulation of the ventricles to the opening of the aortic valve (i.e., electrical systole). This measure of myocardial contractility is the best indicator of sympathetic β-adrenergic impact on the heart. Respiratory sinus arrhythmia is the high-frequency heart rate variability associated with spontaneous breathing (i.e., respiration-mediated): During inspiration, vagal activity is attenuated and heart rate accelerates, whereas during expiration vagal activity is reinstated and heart rate slows. Respiratory sinus arrhythmia thus constitutes a relatively pure index of parasympathetic vagal effects on the heart. Recent evidence suggests that the low-frequency component of heart rate variability may reflect baroreflex-mediated vagal activity (Goldstein, Bentho, Park, & Sharabi, 2011), whereas earlier conceptualizations suggested a combination of vagal and sympathetic activity (Malliani, Pagani, Lombardi, & Ceruti, 1991; Martinmäki, Rusko, Kooistra, Kettunen, & Saalasti, 2006).

Cardiac processes may be additionally influenced by vascular changes, i.e., vasodilation and -constriction. Hence, measures of arterial functioning, such as diastolic blood pressure, total peripheral resistance, pulse amplitude, and peripheral surface temperature are useful to supplement. In particular measures of peripheral resistance can help differentiate cardiac effects that are caused either by changes in cardiac contractility, hence indicating sympathetic β-adrenergic effects on the heart, or by change in preload or afterload. Whereas preload is affected by venous blood pressure and the rate of venous return, afterload effects are a consequence of primarily aortic pressure.

Finally, respiratory activity can interact with activity of other response organs in significant ways. As mentioned above, heart rate changes as a function of the respiratory cycle. Given concerns of confounding effects of concurrent changes in respiratory rate and volume on estimates of respiratory sinus arrhythmia (Grossman, Karemaker, & Wieling, 1991; Wilhelm et al., 1998), respiratory correction or, at least, co-assessment is generally suggested (Allen, Chambers, & Towers, 2007; Grossman & Kollai, 1993; Ritz, 2009). Respiration also affects electrodermal activity. For example, deep breaths can trigger skin conductance responses. Likewise, voluntary changes in respiratory rate can influence occurrence and cycle time of rhythmical components of electrodermal activity (Rittweger, Lambertz, & Langhorst, 1997). Respiratory co-assessment can help identify respiration-generated effects on electrodermal activity.

Given that breathing is a phenomenon that falls under both conscious and unconscious control, it is often treated as a separate response system. Respiratory phenomena include changes in respiratory cycle times, such as respiratory rate or its subcomponents of inspiratory, expiratory, and respiratory pause times, as well as volumetric changes of respiratory depth, as assessed by tidal volume. The relation of these respiratory response measures to each other allows inferences about the regulation of the breathing cycle (Gautier, 1980; Wientjes, 1992). On one hand, mean inspiratory flow rate, i.e., the ratio of inspiratory tidal volume to inspiratory time, indexes the intensity of a central inspiratory drive mechanism, which determines the intensity of the inspiratory impulse. On the other hand, inspiratory duty cycle, i.e., the ratio of inspiratory to total breath time, indexes a rhythm generator with variable periodicity, which cyclically switches the central drive mechanism on and off, and may hence be viewed as a respiratory control mechanism. The rhythm generator is one of the main determinants of the duration of the inspiratory phase of respiration. It can also introduce marked breath-by-breath variations in respiration rate and tidal volume.

Taken together, cardiac, vascular, electrodermal, and respiratory organ systems interact in complex ways. Whereas assessment of single measures can only provide a limited window onto changes in target organ activity, multi-measure assessment allows a more complete picture of autonomic control modes. Table 1 summarizes cardiovascular, electrodermal, and respiratory response measures and their autonomic index function. To interpret physiological data it is important to select the assessed ANS parameters on the basis of hypotheses about the psycho-physiological meaning with regard to activation and deactivation involved in different emotions. That is, the selection of assessed ANS parameters should be hypothesis- and theory-based rather than guided by exploration.

Autonomic Responding in Emotion in Education and Achievement Settings

Autonomic responding is an integral component of emotion. The three above discussed principles of bidirectionalism, allodynamic control, and bivariate space of autonomic activation can each be applied to research topics on emotion in the context of education and achievement settings. To illustrate this, we briefly present representative examples of paradigms assessing ANS reactivity relevant to each principle.

The Principle of Bidirectionalism in the Context of Test Anxiety

The tenet of bidirectional autonomic influences holds that signals transmitted via both efferent and afferent autonomic pathways have functional significance. Whereas autonomic efferences generate an autonomic activation state that is believed to protect bodily functioning and support action preparation, autonomic afferences transmit feedback signals that feed into the experience of emotional feelings and modulate central information processing.

This tenet is readily illustrated in research on bidirectional interactions in stress and anxiety. The cardiovascular afference model of cognitive effects of anxiety (Berntson et al., 1998) postulates a basal forebrain cholinergic link that relays peripheral afferences to generate broad cortical stimulation. This model centrally assumes that the efficacy of cortical information processing can be altered by anxiety-elicited changes in cardiovascular reactivity. Bidirectional interactions of central and autonomic nervous system effects of anxiety are conceptualized in terms of descending and ascending components. The descending system mediates the expression of anxiety and initiates autonomic responses (e.g., Kreibig, Wilhelm, Roth, & Gross, 2007). The ascending system of cardiovascular afferents, in turn, is hypothesized to enhance cortical evaluative processing of anxiety-related stimuli and contexts through activation of basal forebrain cholinergic neurons. Specifically, functions relating to the detection and selection of relevant stimuli as well as their extended processing, i.e., attentional processes, are suggested to be affected. Because attentional processes also play a prime role in learning and memory (Sarter, Bruno, & Gives, 2003), student anxiety would be expected to directly impact these processes via the outlined link.

Concurrent assessment of measures of autonomic reactivity, such as heart rate, heart rate variability, and pre-ejection period, together with either behavioral measures of performance outcome or measures of cortical activation, such as regional cerebral blood flow or electroencephalography, allow to test proposed interactions within analytical frameworks that predict cerebral activation or performance outcomes based on autonomic reactivity.

While performance impairments of test anxiety – the most intensively studied emotion in educational settings (Pekrun, 2005) - have been documented in numerous studies (e.g., Cassady, 2004; Hancock, 2001), there is paucity of research investigating interactions between central and autonomic nervous system processes in the context of test anxiety. Still, indirect links suggest anxiety-specific impairments of attentional processes (Shackman et al., 2006) as well as modulation of sensory discrimination and attentional performance by autonomic nervous system states (Basile et al., 2012; Hatfield, Landers, & Ray, 1987; Saxon & Dahle, 1971). This example illustrates the potential that bidirectional conceptualizations of brain–ANS interactions offer for the explanation of emotional phenomena in education and achievement settings.

The Allodynamic Principle in the Conceptualization of Mental Effort

The tenet of allodynamic regulation of autonomic activity holds that the ANS has variable operating characteristics that are jointly determined by homeostatic and higher-order controls. Adjustment of set points or gains may occur at any level.

This tenet is readily illustrated in research on effort mobilization in cognitive tasks with a clearly defined performance standard. Motivational intensity theory (Brehm & Self, 1989) postulates that effort, i.e., motivational intensity, is directly related to perceived task difficulty. A maximum level of potential motivation, however, places an upper limit on effort investment. This upper level is determined by various motivational variables, including the importance of the consequences associated with a successful outcome of the task. Beyond this upper level, success is either viewed as impossible or to require more effort than is warranted by its importance and hence, all effort is withdrawn (Figure 3a).

How the specific relation is expressed may be qualified by (1) an amplification of the response function by increased (or decreased) self-involvement in performance settings where instrumental behavior has implications for an individual's self-evaluation, social evaluation, or personal interests and values (Gendolla & Richter, 2010, Figure 3b); and (2) a shift of the response function on the abscissa by high or low appraised ability or task capability, given that performers judge tasks to be increasingly difficult, the less capable they view themselves within the relevant performance domain (Wright, 1998, Figure 3c). Effects corresponding to those of low vs. high ability were found for sad vs. happy moods and weak vs. strong depressive symptoms (see Gendolla, Brinkmann, & Silvestrini, 2012).

Integration of motivational intensity theory with the active coping approach to cardiovascular arousal (Obrist, 1981) by Wright (1996) specifies how motivation-related appraisals influence autonomic reactivity. Specifically, within this framework effort is conceptualized as sympathetic β-adrenergic cardiac reactivity. Whereas pre-ejection period is the best noninvasive indicator of sympathetic β-adrenergic impact on the heart, systolic and diastolic blood pressure as well as heart rate can also reflect its influence, albeit less clearly.

Typical results demonstrate shortened pre-ejection period and increased systolic blood pressure, and—with less consistency—increased diastolic blood pressure and heart rate with increasing level of task difficulty as long as success is possible and justified: Low cardiovascular reactivity is observed for tasks that are easy, impossible, or not worth investing the necessary effort; high cardiovascular reactivity is observed in conditions of high or unspecified difficulty, interpreted to index importance of success (e.g., Richter & Gendolla, 2009; Richter, Friedrich, & Gendolla, 2008). Conditions of high self-involvement as contrasted to low self-involvement have been demonstrated to increase cardiovascular reactivity for difficult and unfixed tasks, indicating an increase of importance of success (see Gendolla & Richter, 2010). Research has moreover demonstrated higher cardiovascular reactivity for participants with low perceived ability than for participants with high perceived ability at low levels of task difficulty, whereas the reverse pattern is found at high levels of task difficulty, i.e., higher cardiovascular reactivity for participants with high perceived ability than for participants with low perceived ability (see Wright, 1998; Wright & Kirby, 2001 for overviews). These results are typically interpreted as indicating higher perceived demand in low-ability participants than in high-ability participants, leading to higher activation at lower levels of difficulty and earlier disengagement from goal striving in low- than in high-ability participants at higher levels of task difficulty. This example illustrates the importance of the assumed underlying model of autonomic regulation—with fixed relations and tunable parameters—for the prediction and interpretation of autonomic effects as well as for the choice of dependent autonomic measures.

The Principle of Bivariate Autonomic Space in the Assessment of Emotional Effects

The tenet of a bivariate autonomic space holds that sympathetic and parasympathetic branches of the ANS may operate in coupled or uncoupled modes. Consequently, independent assessment of sympathetic and parasympathetic influences on dually innervated effector organs is required to elucidate autonomic activation components.

This tenet is readily illustrated in a series of studies on achievement emotions in response to performance feedback that varied in outcome relevance (i.e., relevant or irrelevant) and outcome valence (i.e., success or failure; Kreibig, Gendolla, & Scherer, 2010, 2012). It was predicted that relevant success feedback would elicit positive achievement emotions, as expressed in the experience of joy and pride and autonomic co-activation. Relevant failure feedback was predicted to elicit negative achievement emotions, as expressed in the experience of disappointment and autonomic co-inhibition. In contrast, irrelevant feedback was expected to not lead to an emotional response, given that goal relevance is often assumed a prerequisite for the elicitation of emotion (Scherer, 2009).

Pre-ejection period was assessed as an index of sympathetic β-adrenergic cardiac activity. Heart rate variability was assessed in order to quantify respiratory sinus arrhythmia as an index of parasympathetic cardiac (vagal) activity. Additionally, indices of cardiac autonomic balance (considering the predominance of sympathetic or parasympathetic controls via the difference between normalized PEP and normalized RSA) and cardiac autonomic control (considering the joint influence of sympathetic and parasympathetic controls by summing over normalized PEP and RSA) were calculated (Berntson, Norman, Hawkley, & Cacioppo, 2008). This central set of measures was supplemented with measures of heart rate, given traditional assessment of this index, mean arterial blood pressure and total peripheral resistance, for probing vascular effects, and skin conductance level and response amplitude, for indexing electrodermal activity.

Central to the present discussion, the derived index of cardiac autonomic regulation indicated autonomic co-activation during relevant success feedback and autonomic co-inhibition during relevant failure feedback, whereas irrelevant success and failure feedback conditions showed no effects. In contrast, more traditional measures of heart rate and skin conductance showed less differentiated effects. This example illustrates the importance of assessing independent indicators of sympathetic and parasympathetic effects for sensitively quantifying autonomic emotion effects.

Summary and Future Directions

We opened up the current chapter with a case description of a student's emotions occurring while solving actual mathematical problems in her regular classroom (Ahmed et al., 2010). Considering questions regarding the relation between emotion and ANS activity, the role of emotions in regulating autonomic reactivity, and the autonomic controls of emotion effects led us to explore the structure and functioning of the ANS. Three central principles were identified—bidirectionalism, allodynamic regulation, and a bivariate autonomic space. Applications of each principle were next illustrated, outlining the relevance of bidirectional autonomic effects in the context of test anxiety, allodynamic regulation in the investment of mental effort, and the implications of a bivariate conceptualization of autonomic space in the assessment of autonomic effects of achievement emotions. We conclude by returning to Ahmed et al.'s (2010) case study, which documented the richness of interactions of emotion, appraisal, and potential autonomic states in educational and achievement settings. Application of the three central principles of bidirectionalism, allodynamic regulation, and a bivariate autonomic space to the research context of emotion in education and achievement settings opens up new directions for future reseach.

First, consideration of bidirectional effects allows to address both effects of visceral efferences in emotion, for example, as an outcome of specific events during test taking, as well as effects of visceral afferences in emotion leading to specific psychological states or performance outcomes. Of course, efferent and afferent effects of emotion need not be limited to states of anxiety that may lead to sympathetic arousal and attentional impairments, but should also consider beneficial effects of both positive and negative emotion. As Panel C in Figure 1 suggests, experience of positive affect may sooth autonomic arousal and facilitate task performance (e.g., Gendolla & Krüsken, 2001).

Second, conceptualization of autonomic effects of mental effort from the vantage point of allodynamic regulation, as formalized in motivational intensity theory, allows to derive specific predictions for effects of task difficulty, task value, and student ability. This promises to clarify relations between appraisals, subjective feelings, and autonomic variables in the context of academic task performance.

Third, assumption of a bivariate model of autonomic space calls for differential sympathetic and parasympathetic indices of cardiac activity, such as pre-ejection period and heart rate variability. The specific assessment of autonomic activation components—and thus of autonomic modes—promises to allow identification of specific effects of students' emotions of anxiety, anger, joy, and so on. Addition of vascular, electrodermal, and respiratory response measures allows for a comprehensive multi-system assessment and more specific inferences regarding multi-system differentiation of autonomic emotion effects. That way, monitoring ANS responses can provide important information about the experience of emotions in educational settings.

References

Ahmed, W., van der Werf, G., & Minnaert, A. (2010). Emotional experiences of students in the classroom: A multimethod qualitative study. *European Psychologist*, *15*, 142–151.

Allen, J. J. B., Chambers, A. S., & Towers, D. N. (2007). The many metrics of cardiac chronotropy: A pragmatic primer and a brief comparison of metrics. *Biological Psychology*, *74*, 243–262.

Basile, B., Bassi, A., Calcagnini, G., Strano, S., Caltagirone, C., Macaluso, E., et al. (2012). Direct stimulation of the autonomic nervous system modulates activity of the brain at rest and when engaged in a cognitive task. *Human Brain Mapping*.

Bechara, A., Tranel, D., Damasio, H., & Damasio, A. R. (1996). Failure to respond autonomically to anticipated future outcomes following damage to prefrontal cortex. *Cerebral Cortex*, *6*, 215–225.

Berntson, G. G., & Cacioppo, J. T. (2007). Integrative physiology: Homeostasis, allostasis, and the orchestration of systemic physiology. In J. T. Cacioppo, L. G. Tassinary, & G. G. Berntson (Eds.), *Handbook of psychophysiology* (pp. 433–452). Cambridge, UK: Cambridge University Press.

Berntson, G. G., Cacioppo, J. T., Binkley, P. F., Uchino, B. N., Quigley, S., K, & Fieldstone, A. (1994). Autonomic cardiac control. III. Psychological stress and cardiac response in autonomic space as revealed by pharmacological blockades. *Psychophysiology*, *31*(6), 599–608.

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

Berntson, G. G., Norman, G. J., Hawkley, L. C., & Cacioppo, J. T. (2008). Cardiac autonomic balance versus cardiac regulatory capacity. *Psychophysiology*, *45*(4), 643–652.

Berntson, G. G., Sarter, M., & Cacioppo, J. T. (1998). Anxiety and cardiovascular reactivity: The basal forebrain cholinergic link. *Behavioural Brain Research*, *94*, 225–248.

Brehm, J. W., & Self, E. A. (1989). The intensity of motivation. *Annual Review of Psychology*, *40*, 109–131.

Cacioppo, J. T., Berntson, G. G., & Binkley, P. F. (1994). Autonomic cardiac control. II. Noninvasive indices and basal response as revealed by autonomic blockades. *Psychophysiology*, *31*, 586–598.

Cacioppo, J. T., & Tassinary, L. G. (1990). Inferring psychological significance from physiological signals. *American Psychologist*, *45*, 16–28.

Cacioppo, J. T., Tassinary, L. G., & Berntson, G. G. (Eds.) (2007). Handbook of Psychophysiology (3rd ed.). New York: Cambridge University Press.

Cannon, W. B. (1927). The James-Lange theory of emotions: A critical examination and an alternative theory. *American Journal of Psychology*, *39*, 106–124.

Cannon, W. B. (1928). The mechanisms of emotional disturbance of bodily functions. *New England Journal of Medicine*, *198*, 877–884.

Cannon, W. B. (1929). *Bodily changes in pain, hunger, fear, and rage* (2nd ed.). New York and London: D. Appleton & Co.

Cannon, W. B. (1932). *The wisdom of the body*. New York: Norton.

Cassady, J. C. (2004). The influence of cognitive test anxiety across the learning–testing cycle. *Learning and Instruction*, *14*, 569–592.

Damasio, A. R. (1996). The somatic marker hypothesis and the possible functions of the prefrontal cortex. *Philosophical Transactions of the Royal Society London B*, *351*, 1413–1420.

Damasio, A. R., Tranel, D., & Damasio, H. C. (1991). Somatic markers and the guidance of behavior: Theory and preliminary testing. In H. S. Levin, H. M. Eisenberg, & A. L. Berntson (Eds.), *Frontal lobe function and dysfunction* (pp. 217–229). New York: Oxford University Press. Available from `http://books.google.com/books?id=kB19aJKJLoEC`

Dunn, B. D., Dalgleish, T., & Lawrence, A. D. (2006). The somatic marker hypothesis: A critical evaluation. *Neuroscience and Biobehavioral Reviews* , *30*, 239–271.

Dworkin, B. R. (1993). *Learning and physiological regulation*. Chicago: University of Chicago Press. Available from `http://books.google.com/books?id=fDE-teUjYRoC`

Ekman, P. (1972). Universal and cultural differences in facial expression of emotion. In J. K. Cole (Ed.), *The Nebraska Symposium on Motivation, 1971* (pp. 207–283). Lincoln: University of Nebraska Press.

Folkow, B. (2000). Perspectives on the integrative functions of the 'sympatho-adrenomedullary system'. *Autonomic Neuroscience: Basic and Clinical* , *83*, 101–115.

Gautier, H. (1980). Control of pattern of breathing. *Clinical Science* , *58*, 343–348.

Gendolla, G. H. E., Brinkmann, K., & Silvestrini, N. (2012). Gloomy and lazy? On the impact of mood and depressive symptoms on effort-related cardiovascular response. In R. A. Wright & G. H. E. Gendolla (Eds.), *How motivation affects cardiovascular response: Mechanisms and applications* (pp. 139-155). Washington DC: APA Press.

Gendolla, G. H. E., & Krüsken, J. (2001b). The joint impact of mood state and task difficulty on cardiovascular and electrodermal reactivity in active coping. *Psychophysiology, 38,* 539-548.

Gendolla, G.H.E., & Richter, M. (2010). Effort mobilization when the self is involved: Some lessons from the cardiovascular system. *Review of General Psychology, 14,* 212-226.

Gendolla, G. H. E., Richter, M., & Silvia, P. J. (2008). Self-focus and task difficulty effects on effort-related cardiovascular reactivity. *Psychophysiology* , *45* , 653–662.

Goldstein, D. S., Bentho, O., Park, M.-Y., & Sharabi, Y. (2011). Low-frequency power of heart rate variability is not a measure of cardiac sympathetic tone but may be a measure of modulation of cardiac autonomic outflows by baroreflexes. *Experimental Physiology* , *96* , 1255–1261.

Grossman, P., Karemaker, J., & Wieling, W. (1991). Prediction of tonic parasympathetic cardiac control using respiratory sinus arrhythmia: The need for respiratory control. *Psychophysiology* , *28* , 201–216.

Grossman, P., & Kollai, M. (1993). Respiratory sinus arrhythmia, cardiac vagal tone, and respiration: Within- and between-individual relations. *Psychophysiology* , *30* , 486–449.

Hancock, D. R. (2001). Effects of test anxiety and evaluative threat on students' achievement and motivation. *The Journal of Educational Research* , *94* , 284–290.

Harrison, N. A., Kreibig, S. D., & Critchley, H. D. (in press). A two-way road: Efferent and afferent pathways of autonomic activity in emotion. In J. L. Armony & P. Vuilleumier (Eds.), *Handbook of human affective neuroscience.* Cambridge, England: Cambridge University Press.

Hatfield, B. D., Landers, D. M., & Ray, W. J. (1987). Cardiovascular–CNS interactions during a self-paced, intentional attentive state: Elite marksmanship performance. *Psychophysiology* , *24* , 542–549.

Izard, C. E. (1977). *Human emotions*. New York: Plenum Press. Available from `http://books.google.com/books?id=A8K1G9FXMsEC`

James, W. (1884). What is an emotion? *Mind* , *9* , 188–205.

Jänig, W. (2003). The autonomic nervous system and its coordination by the brain. In R. J. Davidson, K. R. Scherer, & H. H. Goldsmith (Eds.), *Handbook of affective sciences* (pp. 135–186). New York: Oxford University Press.

Jänig, W., & Häbler, H.-J. (1999). Organization of the autonomic nervous system: Structure and function. In P. J. Vinken, G. W. Bruyn, & O. Appenzeller (Eds.), *Handbook of clinical neurology: Vol. 74. The autonomic nervous system: Part 1. Normal functions* (pp. 1–52). Amsterdam: Elsevier. Available from `http://books.google.com/books?id=ZpaBSzZQOQsC`

Jänig, W., & Häbler, H.-J. (2000). Specificity in the organization of the autonomic nervous system: A basis for precise neural regulation of homeostatic and protective body functions. *Progress in Brain Research*, *122*, 351–367.

Jänig, W., & McLachlan, E. M. (1992a). Characteristics of function-specific pathways in the sympathetic nervous system. *Trends in Neurosciences*, *15*, 475–481.

Jänig, W., & McLachlan, E. M. (1992b). Specialized functional pathways are the building blocks of the autonomic nervous system. *Journal of the Autonomic Nervous System*, *41*, 3–13.

Koizumi, K., Terui, N., & Kollai, M. (1983). Neural control of the heart: Significance of double innervation re-examined. *Journal of the Autonomic Nervous System*, *7*, 279–294.

Kreibig, S. D. (2010). Autonomic nervous system activity in emotion: A review. *Biological Psychology*, *84*, 394–421.

Kreibig, S. D., Gendolla, G. H. E., & Scherer, K. R. (2010). Psychophysiological effects of emotional responding to goal attainment. *Biological Psychology*, *84*, 474–487.

Kreibig, S. D., Gendolla, G. H. E., & Scherer, K. R. (2012). Goal relevance and goal conduciveness appraisals lead to differential autonomic reactivity in emotional responding to performance feedback. *Manuscript under review*.

Kreibig, S. D., Schaefer, G., & Brosch, T. (2010). Psychophysiological response patterning in emotion: Implications for affective computing. In K. R. Scherer, T. Baenziger, & E. Roesch (Eds.), *Blueprint for affective computing: A sourcebook* (pp. 105–130). Oxford, England: Oxford University Press.

Kreibig, S. D., Wilhelm, F. H., Roth, W. T., & Gross, J. J. (2007). Cardiovascular, electrodermal, and respiratory response patterns to fear- and sadness-inducing films. *Psychophysiology*, *44*(5), 787–806.

Lacey, J. I. (1959). Psychophysiological approaches to the evaluation of psychotherapeutic process and outcome. In E. A. Rubenstein & M. B. Parloff (Eds.), *Research in psychotherapy* (Vol. 1, pp. 160–208). Washington, DC: American Psychological Association.

Lang, P. J. (1994). The motivational organization of emotion: Affect–reflex connections. In S. VanGoozen, N. E. Van de Poll, & J. A. Sergeant (Eds.), *Emotions: Essays on emotion theory* (pp. 61–93). Hillsdale, NJ: Erlbaum.

Lang, P. J., & Bradley, M. M. (2010). Emotion and the motivational brain. *Biological Psychology*, *84*, 437–450.

Lange, C. (1885). The mechanism of the emotions. In E. Dunlap (Ed.), *The emotions* (pp. 33–92). Baltimore, Maryland: Williams & Wilkins.

Langley, J. N. (1903). Das sympathische und verwandte nervöse Systeme der Wirbeltiere (autonomes nervöses System). *Ergebnisse der Physiologie*, *27/II*, 818–827.

Langley, J. N. (1921). *The autonomic nervous system (Pt. 1).* Cambridge, England: Heffer.

Malliani, A., Pagani, M., Lombardi, F., & Ceruti, S. (1991). Cardiovascular neural regulation explored in the frequency domain. *Circulation*, *84*, 482–492.

Martinmäki, K., Rusko, H., Kooistra, L., Kettunen, J., & Saalasti, S. (2006). Intraindividual validation of heart rate variability indexes to measure vagal effects on hearts. *American Journal of Physiology – Heart and Circulatory Physiology*, *290*, H640–H647.

Obrist, P. A. (1981). *Cardiovascular psychophysiology: A perspective.* New York: Plenum Press.

Pekrun, R. (2005). Progress and open problems in educational emotion research. *Learning and Instruction, 15,* 497-506.

Richter, M., Friedrich, A., & Gendolla, G. H. E. (2008). Task difficulty effects on cardiac activity. *Psychophysiology*, *45*, 869–875.

Richter, M., & Gendolla, G. H. E. (2009). The heart contracts to reward: Monetary incentives and preejection period. *Psychophysiology* , *46* , 451–457.

Rittweger, J., Lambertz, M., & Langhorst, P. (1997). Influences of mandatory breathing on rhythmical components of electrodermal activity. *Clinical Physiology*, *17*, 609–618.

Ritz, T. (2009). Studying noninvasive indices of vagal control: The need for respiratory control and the problem of target specificity. *Biological Psychology*, *80*, 158–168.

Russell, J. A., Rosenberg, E. L., & Lewis, M. D. (2011). Introduction to a special section on basic emotion theory. *Emotion Review*, *3*, 363.

Sarter, M., Bruno, J. P., & Gives, B. (2003). Attentional functions of cortical cholinergic inputs: What does it mean for learning and memory? *Neurobiology of Learning and Memory*, *80*, 245–256.

Saxon, S. A., & Dahle, A. J. (1971). Auditory threshold variations during periods of induced high and low heart rates. *Psychophysiology*, *8*, 23–29.

Scherer, K. R. (2004). Feelings integrate the central representation of appraisal-driven response organization in emotion. In A. S. R. Manstead, N. H. Frijda, & A. H. Fischer (Eds.), *Feelings and emotions: The Amsterdam Symposium* (pp. 136–157). Cambridge: Cambridge University Press.

Scherer, K. R. (2009). The dynamic architecture of emotion: Evidence for the component process model. *Cognition and Emotion*, *23*, 1307–1351.

Schütz, B., Engelhardt, J. von, Gördes, M., Schäfer, M. K.-H., Eiden, L. E., Monyer, H., et al. (2008). Sweat gland innervation is pioneered by sympathetic neurons expressing a cholinergic/noradrenergic co-phenotype in the mouse. *Neuroscience*, *156*, 310–318.

Selye, H. (1973). Homeostasis and heterostasis. *Perspectives in Biology and Medicine*, *16*, 441–445.

Shackman, A. J., Sarinopoulos, I., Maxwell, J. S., Pizzagalli, D. A., Lavric, A., & Davidson, R. J. (2006). Anxiety selectively disrupts visuospatial working memory. *Emotion*, *6*, 40–61.

Shields, S. A., MacDowell, K. A., Fairchild, S. B., & Campbell, M. L. (1987). Is mediation of sweating cholinergic, adrenergic, or both? A comment on the literature. *Psychophysiology*, *24*, 312–319.

Stemmler, G. (2004). Physiological processes during emotion. In P. Philippot & R. S. Feldman (Eds.), *The regulation of emotion* (pp. 33–70). Mahwah, NJ: Erlbaum.

Sterling, P., & Eyer, J. (1988). Allostasis: A new paradigm to explain arousal pathology. In S. Fisher & J. Reason (Eds.), *Handbook of life stress, cognition and health* (pp. 629–649). New York: John Wiley & Sons.

Tomkins, S. S. (1962). *Affect, imagery, consciousness: Vol. 1. The positive affects*. New York: Springer.

Tomkins, S. S. (1963). *Affect, imagery, consciousness: Vol. 2. The negative affects*. New York: Springer.

Wientjes, C. J. E. (1992). Respiration in psychophysiology: Measurement issues and applications. *Biological Psychology*, *34*, 179–203.

Wilhelm, F. H., Berkowitz, J., Hansen, M., Grossman, P., Roth, W. T., & Gross, J. J. (1998). RSA estimation during spontaneous breathing using a paced breathing calibration. *Psychophysiology*, *35*, S88.

Wright, R. A. (1996). Brehm's theory of motivation as a model of effort and cardiovascular response. In P. M. Gollwitzer & J. A. Bargh (Eds.), *The psychology of action: Linking cognition and motivation to behavior* (pp. 424–453). New York: Guilford.

Wright, R. A. (1998). Ability perception and cardiovascular response to behavioral challenge. In M. Kofta, G. Weary, & G. Sedek (Eds.), *Control in action: Cognitive and motivational mechanisms* (pp. 197–232). New York: Plenum.

Wright, R. A., & Kirby, L. D. (2001). Effort determination of cardiovascular response: An integrative analysis with applications in social psychology. *Advances in Experimental Social Psychology, 33,* 255-307.

Footnote

[1] This chapter deals with ANS assessment rather then with all physiological correlates of emotions. Therefore several other physiological measures, like central nervous system activity or facial muscular responses are not discussed here. The interested reader might want to consult other sources for these measures, as, for example, the Handbook of Psychophysiology (Cacioppo, Tassinary, & Berntson, 2007).

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Table 1 Overview of Indicator Functions of Primary Autonomic Response Measures according to Autonomic Branch, Effector Organ, Receptor Type, and Response Direction.

Autonomic Branch	Effector Organ	Receptor Type	Response Measure	Abbreviation	Response Direction
SNS	heart	β-adrenergic	heart rate†	HR	+
			pre-ejection period	PEP	−
			stroke volume	SV	+
			cardiac output	CO	+
			systolic blood pressure	SBP	+
			pulse transit time	PTT	−
	lungs‡		respiratory rate†	RR	+
			tidal volume†	V_t	+
			mean inspiratory flow rate	V_t/T_i	+
	vasculature	α-adrenergic	diastolic blood pressure	DBP	+
			total peripheral resistance	TPR	+
			finger pulse amplitude	FPA	−
			finger temperature (surface)	FT	−
	eccrine sweat glands/electrodermal	cholinergic	skin conductance level	SCL	+
			skin conductance response amplitude	SCR	+
			skin conductance response rate	nsSCRR	+
PNS	heart/respiration-mediated	cholinergic	heart rate†	HR	−
			root mean squared successive differences	RMSSD	+
			peak–valley respiratory sinus arrhythmia	p–v RSA	+
			high-frequency heart rate variability	HF-HRV	+
	heart/baroreflex-mediated		low-frequency heart rate variability	LF-HRV	+
	lungs‡		respiratory rate† tidal	RR V_t	−
			volume† inspiratory	T_i/T_{tot}	−
			duty cycle		−

Note. SNS: sympathetic nervous system; PNS: parasympathetic nervous system; *†*: unspecific index due to dual control through SNS and PNS; *‡*: conjointly influenced by autonomic, cortical, and behavioral influences; +: increase with increasing autonomic influence; −: decrease with increasing autonomic influence.

Figure Captions

Figure 1. A student's emotional responses during a math exam: feeling self-report from stimulated recall interview, nonverbal expressions, and corresponding heart rate change. From Ahmed et al. (2010). Permission required from Hogrefe Publishing.

Figure 2. The autonomic nervous system; illustration of the sympathetic and parasympathetic branches. The enteric devision, not shown separately here, consists of grops of cell bodies and nerve fibers embedded in the walls of the esophagus, stomach, and intestines. Redrawn from Jänig & Häbler (1999); Kreibig, Schaefer, & Brosch (2010). Permission required from Elsevier.

Figure 3. Illustration of relations between task difficulty and motivational intensity proposed by Motivational Intensity Theory.

Figure 1.

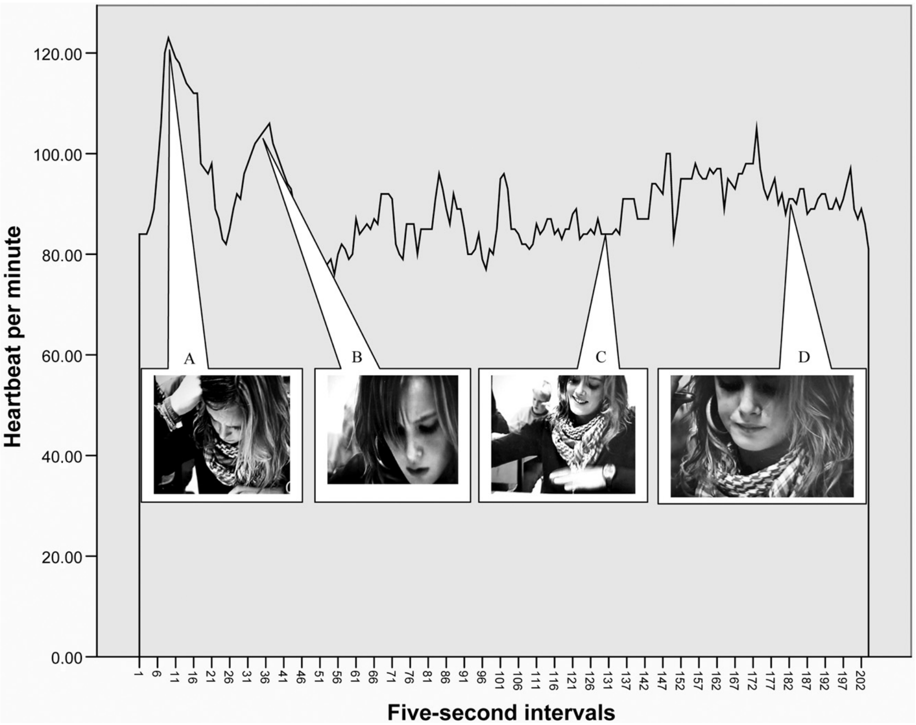


Figure 1. Nonverbal express
corresponding HRC.

Panel A

Interviewer: Can you tell me how you felt when you raised your eyebrows and drew them together like this (attempting to express the behavior)?

Klara: Yeah, I was really anxious. I was not at ease when I learned that I would not be able to do the task. This made me a bit insecure.

Panel B

Interviewer: We just saw in a single instance, your lips were pressed and we could see wrinkles on your forehead. Did you notice that yourself?

Klara: Yes I did.

Interviewer: What do they mean?

Klara: I was a bit annoyed. I understood the question but I was not sure if my answers were correct. It was annoying.

Panel C

Interviewer: You told me that you did not feel confident in doing the task earlier but now I see you laughing. How come?

Klara: Well, I was a bit in to it. I had the feeling that I can do it! However, not feeling like its too easy. I was a bit happy then.

Interviewer: Did that help you to do the task?

Klara: Sure, it did. It helped me to stay focused.

Panel D

Interviewer: Now, we are on the last segment of the video. Can you tell me how you felt?

Klara: Well, more and more students finished the task. Femke (a classmate) was sitting next to me. When I saw that she was finished, I became nervous. I said to myself Oh no, I did not finish on time. Therefore, I started working faster.

Interviewer: So, was that fear of not finishing on time?

Klara: Yes, a kind of. I was anxious but also glad that the task was not so difficult. I knew I could make.

Interviewer: You told me that you did not feel confident in doing the task earlier but now I see you laughing. How come?

Klara: Well, I was a bit in to it. I had the feeling that I can do it! However, not feeling like "it's too easy." I was a bit happy then.

Interviewer: Did that help you to do the task?

Klara: Sure, it did. It helped me to stay focused.

Toward the end of the task, Klara reported being anxious in her interview.

Interviewer: Now, we are on the last segment of the video. Can you tell me how you felt?

Klara: Well, more and more students finished the task. Femke (a classmate) was sitting next to me. When I saw that she was finished, I became nervous. I said to myself "Oh no, I did not finish on time." Therefore, I started working faster.

Interviewer: So, was that fear of not finishing on time?

Klara: Yes, a kind of. I was anxious but also glad that the task was not so difficult. I knew I could make.

In spite of Klara's subjective experience of anxiety, neither her nonverbal expression nor her heart rate signaled the emotion. As can be seen from Picture D in Figure 1, Klara's facial expression does not appear to tell us any kind of emotion. It appears to be neutral. Similarly, the heart rate graph does not show any significant change (HR = 88, M = 86, and SD = 5.8) attributable to anxiety. In general, although the emotional responses appear to converge in some of the instances, there were several instances where such convergence was not evident. As in Figure 1, there were several peaks and valleys in the heart rate graph of all the partici-

How do Appraisals and Emotions Rel

The participants' appraisals and their most f
reported emotions for each task are reported in
The relationship between appraisals and emotion
to be complex. It appears that high perceived co
and high perceived value may result in enjoyme
or anxiety. In the high competence-high value
enjoyment was reported five times, whereas anger
iety were reported three and two times, respec
appears that a student who feels highly compe
who attaches high value to a task may enjoy doing
but she or he may also end up being angry or anx
fact that appraisals were measured at the beginni
task does not however warrant such conclusion. H
petence and low value perceptions seem to lead to
rience of boredom. If a student has a high co
feeling, but finds a task uninteresting, boredom i
occur. It also appears that these appraisal com
may result in anxiety, as is the case with Dirk on
task. The low competence and low value appra
appear to consistently lead to anger or anxiety. T
appraisals differ from each other in significant way
quently, the emotions they experience also diffe
Given the depth of information about the individ
appraisal-emotion links can be assumed viable.

Does Task Difficulty Matter for the T

and Frequency of the Emotions

Figure 2

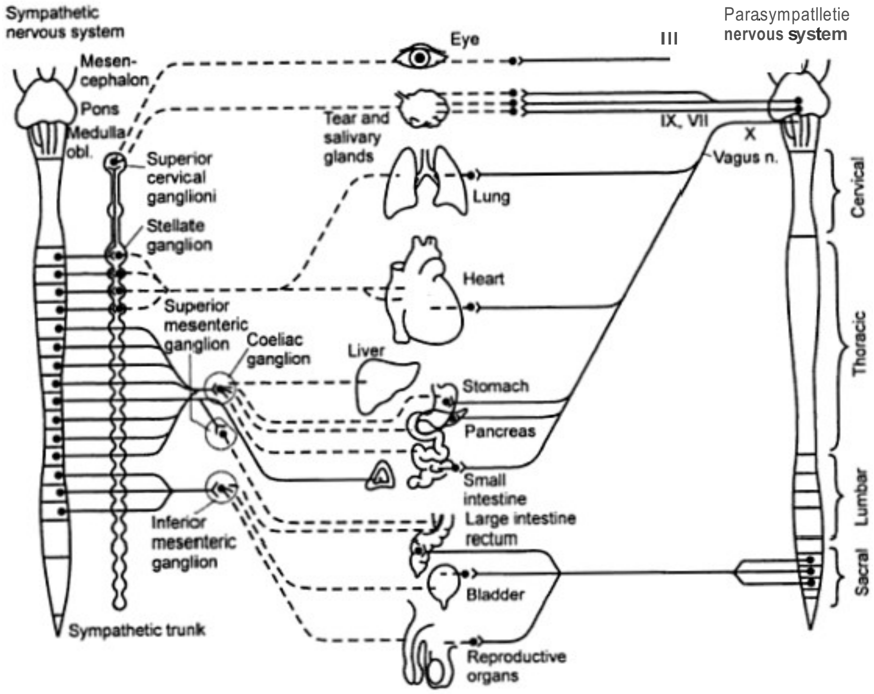
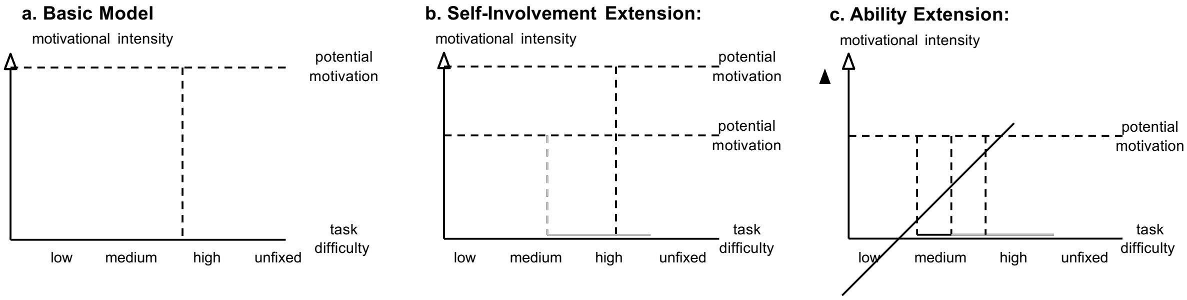

Sympathetic nervous system
Mesen-cephalon
Pons
Medulla obl.
Superior cervical ganglioni
Stellate ganglion
Superior mesenteric ganglion
Coeliac ganglion
Inferior mesenteric ganglion
Sympathetic trunk
Eye
Tear and salivary glands
Lung
Heart
Liver
Stomach
Pancreas
Small intestine
Large intestine rectum
Bladder
Reproductive organs
III
IX, VII
X
Vagus n.
Parasympathetic nervous system
Cervical
Thoracic
Lumbar
Sacral

Figure 3



a. Basic Model

b. Self-Involvement Extension:

c. Ability Extension: