CHAPTER 6

ANOTHER LITTLE PIECE OF MY HEART

Positive Emotions and the Autonomic Nervous System

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In 1884, William James proposed that the subjective feeling of emotion is rooted in “a wave of bodily disturbance” (p. 189) encompassing our instinctive visceral reactions to important stimuli. For psychologists whose theories of emotion grew directly from the Jamesian perspective, physiological responses mediated by changes in the autonomic nervous system (ANS) remain a defining feature of emotional reactivity (e.g., Cosmides & Tooby, 2000; Ekman, 1992). Theories emphasizing dynamic cognitive appraisals as the engines of emotion also identify physiological responses as important aspects of emotional responding (e.g., Scherer, 2009), and dimensional models of affective experience consider subjective arousal to be a key dimension as well (e.g., Russell, 2003). Across theoretical perspectives, questions regarding the role of autonomic responding in emotional experience continue to drive exciting research. The bulk of this work has emphasized “fight/flight” sympathetic nervous system activation associated with the experience of negative emotions, following Walter Cannon’s (1915) early description of this system’s involvement in fear and rage. By contrast, comparatively little attention has been paid to ANS aspects of positive emotion, and the available literature has sometimes suggested that positive emotions have limited ANS involvement at all (e.g., Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000).

This is beginning to change, for a variety of reasons. Research in the last decade or so has highlighted the complexity of the ANS, characterized by multiple distinct neural branches within its parasympathetic division and multiple neurotransmitter mechanisms within its sympathetic division. Technologies have become more advanced for capturing these differing mechanisms through peripheral measures, and are readily available for psychology lab use. Most important, multiple theories of autonomic involvement in positive emotion have been proposed and are generating strong empirical research (e.g., Fredrickson & Levenson, 1988; Kreibig, Gendolla, &

Scherer, 2010; Porges, 2007; Shiota, Neufeld, Yeung, Moser, & Perea, 2011). The psychophysiology of positive emotion is still a scattered field, but one that is growing rapidly as a result of these developments.

In this chapter, we provide an overview of this emerging and exciting field. First, we provide a brief foundation in the structure of ANS, addressing a few important methodological issues in conducting and interpreting emotion psychophysiology research. Second, we describe four major theories of autonomic involvement in positive emotion, illustrating each theory with key research findings. Third, we offer tentative conclusions, suggest future directions, and discuss implications with respect to the light and dark sides of positive emotion.

THE PSYCHOPHYSIOLOGY OF EMOTION: FOUNDATIONS AND METHODOLOGICAL ISSUES

The ANS is highly complex, and measuring its activity and organ-level effects involves many technical and statistical challenges. High-quality “out of the box” commercial systems for laboratory-based psychophysiology measurement are now readily available to researchers, greatly facilitating use of these methods (e.g., Biopac Systems, Inc., Goleta, CA; Mindware Technologies LTD, Gahanna, OH). Despite the ~~ease of the use~~ of these systems, several principles of data quality, study design, and interpretation of findings must be considered in doing meaningful research, ~~yet~~ are commonly misunderstood. Researchers and readers alike should use a critical eye in evaluating study designs and interpreting findings. For this reason, we precede our review of the positive emotion literature with a brief description of the ANS, and a few methodological issues to consider.

GENERAL PRINCIPLES

The ANS consists of neural pathways that receive input from the hypothalamus and brainstem, travel through the spinal cord, and terminate on a wide range of visceral organs including cardiac and smooth muscle tissue. The ANS is split into two major divisions, the sympathetic and parasympathetic divisions. Broadly speaking, increased activation of the *sympathetic* division leads to organ-level changes that support immediate physical effort. These include increasing heart rate to circulate more blood; altering vascular constriction patterns to direct more blood to the skeletal muscles and brain, and less to the digestive system and skin; increasing oxygen intake via breathing and bronchiole expansion; activating sweat glands; and inhibiting digestion, among other effects. Broadly speaking, increased activation of the *parasympathetic* division promotes “rest and digest” activities such as decreasing heart rate and constricting bronchioles; activating the digestive system; and stimulating the liver to store energy. The nature of the ANS and its activity make it important for researchers to keep several methodological principles in mind.

Non-emotional influences on autonomic activity. One key principle in research on the psychophysiology of emotion is that autonomic activity is influenced by many factors other than emotion. Physical movement (even slight), speech, social stimuli such as faces and experimenters, and perceptual and cognitive activity all have robust autonomic effects (e.g., Bradley, 2009; Britton, Taylor, Berridge, & Mikels, 2006; Sherwood, Allen, Fahrenberg, Kelsey, Lovallo, & van

Doornen, 1990), that can dominate or interact with those associated with emotional experience in a typical laboratory study. The ANS also responds to non-emotional features of the current environment, such as ambient temperature (e.g., Gagge, Stolwijk, & Hardy, 1967), and to disruptions of homeostasis, as in hypoglycemia (e.g., Teves, Videen, Cryer, & Powers, 2004). Good studies aim to control for these factors as much as possible, and are explicit about how this is done. Controlling for inherently non-emotional factors can be a challenging task; in some cases, the emotion-eliciting properties of stimuli require certain perceptual (e.g., complexity) and/or content (e.g., social) features that are not easily controlled. When such controls are not feasible, possible confounds and alternate explanations of autonomic effects should at least be discussed. Data should also be “cleaned” carefully to remove artifact associated with non-emotional events such as coughing, sneezing, unexpected speech or movement, among others (e.g., Sherwood et al., 1990). Precise rules for this cleaning vary to some degree across laboratories and studies, depending on the participant sample, study procedures, specific measures, and analysis software (which can occasionally help control for random noise).

Accounting for individual differences: exclusionary criteria and baseline-to-trial change.

A second key principle is that there are many trait-level individual differences in autonomic functioning and its effects that are independent of psychological state. These differences may reflect non-autonomic physiological factors (e.g., body mass; atherosclerosis) as well as individual differences in resting levels of ANS influence. Typically, studies exclude participants who have known cardiovascular problems or are taking medications that likely influence autonomic responding and/or its organ level effects (e.g., beta-blockers, many blood pressure medications, any stimulants). Studies of emotion psychophysiology should also exclude participants taking psychoactive medications (unless necessary for the research question), as these drugs often alter ANS activity as well as ~~alter~~ mood. Another implication of this principle is that establishing the autonomic properties of some psychological state requires measuring *change* in physiology, from an immediately preceding baseline through the period when a participant is presumed to be in that state. In some cases, reasonable research questions may address the relationship between resting/baseline physiological measures and some psychological trait (e.g., the relationship between resting respiratory sinus arrhythmia, an index of parasympathetic influence on the heart, and trait positive emotionality; (Oveis, Cohen, Gruber, Shiota, Haidt, & Keltner, 2009). Usually, however, the research question requires measuring change in ANS activity thought to accompany a corresponding change in psychological state. Establishing a valid baseline becomes especially tricky when doing ambulatory, real-life psychophysiology research, but is necessary nonetheless.

Multidimensionality of ANS mechanisms. A third key principle is that the ANS includes a rich variety of mechanisms across the sympathetic and parasympathetic divisions (Stemmler, Aue, & Wacker, 2007; Stemmler, Grossman, Schmid, & Foerster, 1991). Commonly used organ-level measures such as heart rate and blood pressure capture the *effects* of activity within the ANS rather than neural activity itself. Many organ-level operational measures confound the influence of multiple neural mechanisms. For example, the heart is innervated by both the sympathetic division (which speeds heart rate) and the parasympathetic division (which slows it down). An increase in heart rate might indicate an increase in sympathetic influence, a decrease in parasympathetic influence, or some combination of the two. Similarly, blood pressure is influenced by multiple neural mechanisms and feedback loops across the sympathetic and parasympathetic divisions (Guyenet, 2006). Studies limited to these and similar measures risk blurring what are actually quite distinct autonomic profiles associated with psychological

states, including emotions. Thus, operational measures that tease apart various mechanisms are preferred.

NEURAL MECHANISMS AND OPERATIONAL MEASURES

A comprehensive review of the neural mechanisms of autonomic influence and their organ-level effects is beyond the scope of this chapter. However, much cutting-edge emotion psychophysiology research distinguishes among four mechanisms that can be operationalized with fair precision via non-invasive measures. These are: (1) alpha-adrenergic noradrenaline receptor-mediated effects within the sympathetic division; (2) beta-adrenergic noradrenaline receptor-mediated effects within the sympathetic division; (3) cholinergic paths acting on muscarinic receptors within the sympathetic division; and (4) the vagal branch of the parasympathetic division, which also uses the neurotransmitter acetylcholine and muscarinic receptors. Table 6.1 offers a summary of these mechanisms.

Sympathetic division: alpha- vs. beta-adrenergic receptor mechanisms. As noted above, heart rate and blood pressure confound multiple mechanisms within the ANS. This is for a slightly different reason in each case. With heart rate, the problem is that the sinoatrial node, which regulates heart rate, is influenced by both the beta-adrenergic sympathetic and the vagal parasympathetic mechanisms listed above. With blood pressure, the problem is that pressure is determined by multiple physiological processes, including contraction of smooth muscles surrounding the arteries and the volume of blood being pumped by the heart (cardiac output), each of which is regulated by a different neural mechanism.

A technique called *impedance cardiography* has proved extremely useful in teasing apart these sources of influence, allowing more precise estimates of ANS activity per se. Impedance cardiography involves passing an electrical current through the chest wall and measuring

Table 6.1: Major Neural Mechanisms of Autonomic Influence in Emotion
Psychophysiology Research

ANS Division		Sympathetic		Parasympathetic
Common Term for Mechanism	α -adrenergic	β -adrenergic	Cholinergic	Vagal
Neurotransmitter	Noradrenaline/ norepinephrine	Noradrenaline/ norepinephrine	Acetylcholine	Acetylcholine
Receptor Subtype	α -adrenergic	β -adrenergic	Muscarinic	Muscarinic
Key Peripheral Measure	Total Peripheral Resistance (TPR)	Cardiac Pre-Ejection Period (PEP)	Electrodermal Activity (GSR, SCL)	Respiratory Sinus Arrhythmia (RSA)
Also Influences:	Systolic Blood Pressure Diastolic Blood Pressure Piloerection	Heart Rate/Cardiac Interbeat Interval (IBI) Systolic Blood Pressure		Heart Rate/ Cardiac Interbeat Interval (IBI) Systolic Blood Pressure

change over time in the delay, or impedance, of that current. If sensors are properly placed, a major source of variability in impedance, the expulsion of blood from the heart through the aorta, can be accounted for. Researchers can thereby identify the timing of aortic valve opening with precision and also estimate the volume of blood being pumped by the heart with each beat.

These two pieces of information, combined with information from an electrocardiogram (EKG) and a blood pressure monitor, offer a window into alpha- versus beta-adrenergic mechanisms of sympathetic influence. Beta-adrenergic receptors for noradrenaline (a.k.a. norepinephrine—the terms are interchangeable and used in different parts of the world) have exclusive control over the contractility of ventricular cardiac muscle, and thus how quickly the ventricles build up enough pressure to pop open the aortic valve. The point at which ventricles begin to contract can be identified on the EKG; the point at which the aortic valve opens can be identified from the impedance signal. The difference between the two, or *cardiac pre-ejection period*, thus provides a pure index of beta-adrenergic receptor-mediated sympathetic influence on the heart.

The approach to assessing alpha-adrenergic receptor-mediated activity is not quite as precise, but still useful. Constriction of smooth muscle layers within the arterial walls (vasoconstriction) is regulated largely, though not exclusively, by alpha-adrenergic noradrenaline receptors. Blood pressure offers a somewhat blurry index of peripheral vasoconstriction, as it is strongly influenced by cardiac output (the volume of blood pumped by the heart per minute) as well. The extent of vasoconstriction per se, commonly referred to as *total peripheral resistance*, can be calculated from blood pressure and cardiac output as derived from the impedance signal. Total peripheral resistance is currently the best single measure of alpha-adrenergic receptor-mediated sympathetic influence. However, arrector pili muscles at the base of body hairs, which contract to cause piloerection or “goosebumps,” are activated solely by alpha-adrenergic receptors. High-fidelity laboratory measures of piloerection would thus be another excellent measure of the alpha-adrenergic mechanism.

Sympathetic division: cholinergic mechanism. Measures of sweat gland activity are commonly used to assess the third mechanism of sympathetic nervous system influence. Although sweat gland activity is one of the most commonly measured physiological changes in psychology research, it reflects something of an anomaly within the ANS. The sweat glands are the only organs innervated by cholinergic mechanisms within the sympathetic division; cholinergic mechanisms are more characteristic of the parasympathetic division as a whole. All measures of sweat gland activity take advantage of the principle that the skin conducts electricity, and sweaty skin conducts electricity faster than dry skin. As in impedance cardiography, a sensor is used to introduce a small electrical signal at one location, and the time elapsed before it reaches a sensor at another location (both are typically on the hands) is measured. Even minute bursts of sweat gland activity result in striking changes in conductance across the skin between the sensors, generally referred to as *electrodermal activity*.

Electrodermal activity can be quantified in a number of ways. One approach counts the number and/or calculates the magnitude of brief increases in conductance, or *galvanic skin responses*, that occur in response to some stimulus or task. Another approach averages conductance over a longer period of time to produce a mean *skin conductance level*. Measures produced by these two approaches are often highly correlated. However, a particular measure may be more appropriate for certain research questions. For example, studies investigating responses to a large number of briefly presented stimuli will typically examine galvanic skin responses, whereas studies using longer-duration emotional tasks may emphasize mean skin conductance

level. In either case, the sweat gland activity captured by the measure can be attributed solely to sympathetic influence by way of cholinergic mechanisms.

Parasympathetic division: vagal influence on the heart. Although emotion research has traditionally focused on the “fight/flight” sympathetic division of the ANS, the last decade has seen an increase in research on the parasympathetic division as well. This is especially the case for positive emotion, due to theory specifically linking parasympathetic activity to emotion in pleasant social interaction (discussed in detail below). However, activity within the parasympathetic division is difficult to capture. Most organs innervated by the parasympathetic division are also innervated by the sympathetic division, making it hard to tease out unique effects. The few effects that can be attributed solely to parasympathetic activity, such as gastric acid secretion, cannot be measured easily in a psychology lab setting.

The most common measure, by far, indexes influence of the vagal branch of the parasympathetic division on the heart. Increased activity along this pathway slows heart rate. However, vagal parasympathetic influence on the heart is briefly inhibited during inspiration, or breathing in (Hirsch & Bishop, 1981). The result is faster heart rate during inspiration and slower heart rate during expiration (breathing out), a pattern that may increase the efficiency of oxygen transfer (Grossman & Taylor, 2007; Yasuma & Hayano, 2004). Variability in heart rate that can be attributed to breathing may therefore be used as a measure of the extent of vagal parasympathetic influence on the heart.

Quantifying respiration-linked heart rate variability, or *respiratory sinus arrhythmia* (RSA), is complicated and controversial, because RSA is only one of several factors in overall heart rate variability (HRV). A common approach to measuring RSA uses spectral analysis, a technique in which the waveform of inter-beat intervals over some window of time is broken down into specific sine wave frequencies, and the extent to which frequencies typical of breathing account for overall variability is quantified (Berntson, Bigger, Eckberg, Grossman, Kaufmann, Malik et al., 1997). The root mean square successive difference (RMSSD) across a series of heartbeats may be used to index RSA as well, but this measure can be contaminated by sympathetically-mediated effects on heart rate variability (Berntson, Lozano, & Chen, 2005).

There is also debate over whether to control for baseline-to-trial changes in respiration rate and depth in examining corresponding changes in RSA (Grossman & Taylor, 2007; Porges, 2007). A full discussion of this controversy is well beyond the scope of this chapter. In brief, the issue is that respiration can have effects on measured RSA that are independent of vagal influence, by altering the pattern of windows in which heart rate speeds or slows (Grossman & Taylor, 2007). This is especially problematic when measuring individual differences in resting RSA rather than within-subject change in RSA across multiple conditions. However, some effects of respiration involve feedback loops that travel through vagal mechanisms, and vagal activity and respiration rate are regulated by some of the same neural pathways, so controlling for respiration rate may overcorrect and remove variability that should actually be attributed to vagal influence (Porges, 2007; Grossman & Taylor, 2007). We echo Porges' (2007) call for more research addressing the specific conditions in which corrections for breathing are appropriate in studying RSA. In the meantime, task-related change in RSA should be reported both with and without within-subjects control for change in respiration rate, and possibly tidal volume as well.

Beyond the distinction among these four mechanisms lie even more fine-grained subtleties. There are several subtypes of alpha- and beta-adrenergic receptors that are influenced by specific neuropeptides as well as noradrenaline, and are located on different effector organs (Folkow, 2000; Jänig & Häbler, 2000). Feedback loops are pervasive within the ANS and its organ-level

effects as well, further complicating the picture. However, the four mechanisms discussed above offer a useful foundation for interpreting the research on autonomic responding in positive emotion, to which we now turn.

AUTONOMIC RESPONDING IN POSITIVE EMOTION: THEORETICAL PERSPECTIVES

Initial studies of physiological reactivity associated with “happiness” (elicited through muscle-by-muscle instructions to form a Duchenne smile, or reliving of a happy personal experience) as compared with negative emotions such as anger and fear found little evidence of autonomic responding (e.g., Ekman, Levenson, & Friesen, 1983; Levenson, Ekman, Heider, & Friesen, 1992). Evidence across studies since then has been inconsistent. A meta-analysis of available studies in 2000 concluded that positive emotion might involve relatively little autonomic responding (Cacioppo et al., 2000). However, this analysis aggregated data on states labeled “happiness” or “joy” by the authors of the original studies, and it is possible that different varieties of positive emotion were subsumed under this generic term (Shiota et al., 2011). Also, the meta-analysis excluded studies of states given other labels, such as amusement and love (Kreibig, 2010). Thus, the possibility remains that positive emotions do involve reliable ANS effects that were undetected by the meta-analysis.

One useful way to organize research on the autonomic aspects of positive emotion is by theoretical perspective. Researchers have articulated several clear theories of the nature and function of positive emotion, and these theories have offered a strong basis for hypotheses about ANS responding. As we shall see, each theory has nudged research in a particular direction, and each has received empirical support.

THE “UNDOING” EFFECT

In an early suggestion regarding the function of positive emotion, Tomkins (1962) proposed that joy is associated with relief from the tension that accompanies emotional distress. A possible role of positive emotion in coping was further articulated by Lazarus and Folkman, who posited that positive emotions offer a “psychological break or respite” from stress (e.g., Lazarus, Kanner, & Folkman, 1980). Taking this a step further, Levenson (1988) proposed that one effect of positive emotion might be to “undo” the autonomically-mediated arousal caused by negative emotion.

In an initial study of the undoing hypothesis, Fredrickson and Levenson (1998) asked participants to watch a frightening film clip (a man standing on the ledge of a tall building), followed by a second clip intended to elicit contentment, amusement, neutral affect, or sadness. The frightening clip caused significant reduction in heart rate, reduction in pulse transit time to the ear, and reduction in finger pulse amplitude. The first of these effects is consistent with increased parasympathetic influence on the heart and/or beta-adrenergic sympathetic withdrawal, and is also consistent with the well-established orienting response to new stimuli (Bradley, 2009). The latter two effects are consistent with increased vascular constriction, likely mediated by increased sympathetic alpha-adrenergic receptor activation.

Recovery from this physiological state was operationalized in terms of the mean number of seconds required for each measure to return to a pre-fear-film baseline confidence interval

for five of six consecutive seconds, averaged across the three measures for each participant. Consistent with the undoing hypothesis, mean recovery times for those in the contentment and amusement second-film conditions were approximately half the duration seen in the neutral control condition, and a third of the duration seen with the sad film. These effects were not accounted for by self-reports of interest in the second film, so it is unlikely that the positive films were simply more distracting than the neutral and sad films.

In a follow-up study, Fredrickson and Levenson (1998) showed participants a sad film, followed by a period with no visual stimuli during which participants' facial expressions of emotion were recorded. Unlike the frightening film used in Study 1, the sad film elicited an *increase* in ear pulse transit time, and increase in finger pulse transit time, and a decrease in finger pulse amplitude. This response is not easy to characterize in terms of simple increase in sympathetic responding, as the measures are influenced by both alpha- and beta-adrenergic receptor mechanisms, and some measures suggest a withdrawal of sympathetic influence. Participants were classified as "smilers" or "non-smilers" based on whether they smiled spontaneously during the three minutes after the end of the film. Using the same index of recovery time as in Study 1, smilers recovered more than 20 seconds faster than non-smilers, again consistent with the undoing hypothesis.

The undoing effect has been replicated several times using a variety of negative stimuli and stress tasks. Some studies used a public speech task to elicit initial stress and cardiovascular arousal, finding that experimentally elicited (Fredrickson, Mancuso, Branigan, & Tugade, 2000) and spontaneous (Tugade & Fredrickson, 2004) positive emotions were associated with shorter recovery times. In a daily diary study, the experience of positive affect was found to reduce the effect of negative affect on blood pressure (Ong & Allaire, 2005). Experimentally elicited positive emotion has also been found to help buffer against cardiovascular reactivity during a subsequent stress task (e.g., Ong & Isen, 2010). Taking the undoing effect from an individual-level to a dyad-level phenomenon, one study found that epochs of physiological soothing observed in either spouse during married couples' conflict discussions were accompanied by greater positive emotional behavior (in a ratio with negative emotional behavior) than epochs without soothing (Yuan, McCarthy, Holley, & Levenson, 2010).

Importantly, the positive emotion stimuli used in some of this research did not appear to evoke reduction in cardiovascular arousal (broadly speaking) unless they were preceded by negative stimuli (e.g., Fredrickson et al., 2000). This suggests that the positive emotions were actually undoing the physiological effects of distress, rather than replacing them with independent physiological effects of positive emotion (Fredrickson et al., 2000). The mechanisms of the undoing effect have not yet been investigated closely. One possibility is that positive emotions do offer a potent respite from focusing on stress/distress, and that it is this break that explains physiological undoing rather than positive emotions per se (e.g., Folkman, 1997; Lazarus et al., 1980). Another is that positive emotion does instigate independent autonomic effects that were not captured by these particular studies. This latter possibility has been advocated by Porges, in particular, in his polyvagal theory.

POLYVAGAL THEORY AND PARASYMPATHETIC ACTIVATION

The vagal branch of the mammalian parasympathetic nervous system has two pathways that may have different phylogenetic histories (Morris & Nilsson, 1994; Porges, 1995). An older, dorsal,

unmyelinated pathway is thought to facilitate freezing or “playing dead” under extreme threat. Polyvagal theory (Porges, 1995, 2007) proposes that a ventral, myelinated pathway evolved more recently as a result of mammals’ new social milieu and the associated selection pressures; that it is intertwined with mechanisms for emotional communication (the motor tract of the vagus nerve controls muscles of the face and neck, regulating facial and vocal expression of emotion); and that, under the appropriate circumstances, it inhibits “fight/flight” sympathetic nervous system activation in order to facilitate affiliation, including cooperative/affiliative responses to stress. This theory offers a tentative mechanism by which positive emotions might tend to undo or buffer against the arousal associated with negative emotion, while not showing clear independent effects in the physiological measures that were used in undoing research. If positive emotion involves an increase in vagal parasympathetic activation, this might inhibit stress-related influence on the heart, yet fail to produce independent effects on measures of pulse transit time and amplitude (which are not strongly affected by parasympathetic influence).

What is the evidence that positive emotion does involve vagal parasympathetic activation? At the trait level, there is considerable evidence that individuals who report higher levels of dispositional positive affect also show higher resting levels of RSA (e.g., Eisenberg et al., 1995; Oveis, Cohen, Gruber, Shiota, Haidt, & Keltner, 2009), and chronically elevated vagal tone is characteristic of people at risk for mania (Gruber, Johnson, Oveis, & Keltner, 2008). Some studies have also observed higher resting RSA among those who showed greater increases in self-reported positive emotion during laboratory tasks (e.g., Kettunen, Ravaja, Naatanen, & Keltikangas-Jarvinen, 2000); although other studies have failed to detect this effect (e.g., Frazier, Strauss, & Steinhauer, 2004; Oveis et al., 2009). Another study found that higher resting RSA prospectively predicted increase in daily positive emotion over the next two months (Kok & Fredrickson, 2010). However, the link between trait-level resting vagal tone and positive emotionality could be mediated by strong emotion regulation skills, which have also been linked to high resting RSA (e.g., Appelhans & Luecken, 2006; Demaree, Robinson, Everhart, & Scmeichel, 2004; Fabes & Eisenberg, 1997; Fabes, Eisenberg, & Eisenbud, 1993).

At the state level, there is remarkably little evidence that transient experience of positive emotion is associated with increased RSA. For example, Shiota and colleagues (2011) did not observe any change in RSA among participants viewing photographs eliciting five positive emotions that could not be accounted for by changes in respiration rate, and several positive emotions led to *decreases* in RSA prior to this control. Several other studies have also reported decreases in RSA associated with positive emotion, as well as in negative emotions (e.g., Rainville, Bechara, Naqvi, & Damasio, 2006; Vianna & Tranel, 2006). However, some preliminary evidence suggests that positive social emotions, such as love and compassion, may be characterized by increased vagal parasympathetic activation. McCraty and colleagues (1995) found that the sympathovagal balance of influence on the heart shifted toward vagal influence while participants vividly experienced appreciation for another person. More recently, Matsunaga and colleagues (2009) found that RSA increased while participants viewed film clips showing a marriage ceremony or a confession of love, but not during a neutral control clip (although changes in respiration were not measured or controlled in this study).

Such an effect could help provide a buffer against stress. One study found that high trait compassion predicted greater increases in RSA during a stressful speech task in the presence of supportive evaluators, but not in the presence of neutral evaluators (Cosley, McCoy, Saslow, & Epel, 2010). Notably, one of the positive emotion videos used in the undoing research discussed above presented a playful puppy, a highly effective nurturant love stimulus. A specific link

between vagal parasympathetic activation and positive social emotions is consistent with poly-vagal theory (Porges, 2007). However, few studies have addressed the ANS aspects of these kinds of positive emotion, so much more work is needed to investigate this possibility (Kreibig, 2010).

APPROACH MOTIVATION AND SYMPATHETIC ACTIVATION

A third theoretical perspective links positive emotion to approach motivation, with quite different implications for autonomic responding (Harmon-Jones, 2003). Not all positive emotions are high on approach motivation, and not all approach-motivation emotions are positive (e.g., anger; Carver & Harmon-Jones, 2009; Harmon-Jones, Gable, & Peterson, 2010; Gable & Harmon-Jones, 2008). However, the possibility that some positive emotions are characterized by strong goal-attainment or approach motivation leads to the prediction that they will be accompanied by increases in sympathetically-mediated arousal (Kreibig, Gendolla, & Scherer, 2010).

Considerable evidence that positive emotions may involve increased sympathetic activation has accumulated in the last decade (Kreibig, 2010). For example, Kreibig and colleagues (2010) observed shortened pre-ejection period, increased cardiac output, and increased skin conductance among participants who had just received positive feedback on a challenging task. Vrana and Rollock (2002) and Rainville and colleagues (2006) each reported increases in heart rate during vividly imagined joy or happiness that differed significantly from a neutral control condition, and were comparable to those seen in negative emotions. Tsai and colleagues (2002) observed skin conductance increases while participants relived happiness and pride that were comparable to those while reliving anger, disgust, and sadness. Vianna and Tranel (2006) also obtained skin conductance increases using film clips showing happy couples as stimuli.

Increases in arousal have also been reported in several studies of amusement. For example, Tsai and colleagues (2000) observed heart rate increases in young adults watching an amusing film clip, although this effect was not observed in older adults. Another study found baseline-to-trial changes in a composite of cardiovascular measures (formed from finger and ear pulse transit time, finger pulse amplitude, and finger temperature) consistent with increased sympathetic activation among participants viewing an amusing film, an effect that differed significantly from a neutral film (Giuliani, McRae, & Gross, 2008). Christie and Friedman (2004) reported skin conductance increases during amusement films that were greater than those during neutral, sad, and anger-inducing films.

There is still a great deal of inconsistency in these findings, and effects observed in one study often fail to replicate in another, especially at the level of individual variables. However, two trends are worth noting. First, there is little evidence of increased alpha-adrenergic receptor-mediated sympathetic activity in positive emotion (Kreibig, 2010). While evidence of beta-adrenergic (cardiac) and cholinergic (skin conductance) effects is fairly robust, few studies report increases in total peripheral resistance or blood pressure associated with experimentally elicited positive emotion. Second, increases in arousal may be linked specifically to high-approach-motivation positive states. While labeling of positive emotion states should always be taken with a grain of salt, arousal increases are much more common in states labeled "happiness," "joy," and "amusement" than in "contentment" (Kreibig, 2010). In one important study, Richter and Gendolla (2009) found that the magnitude of PEP shortening (reflecting increased beta-adrenergic sympathetic influence on the heart) during a pleasant task with a monetary incentive increased with

the size of the possible reward—a dose-response-like relationship between approach motivation and beta-adrenergic sympathetic activation. But what about positive emotions characterized by a lesser degree of approach motivation?

AUTONOMIC SPECIFICITY AMONG MULTIPLE POSITIVE EMOTIONS

As noted at the beginning of the chapter, the field of emotion psychophysiology has its roots in a proposal by William James (1884) that the subjective feeling of emotion is grounded in perception of changes in the body, now recognized as mediated by the ANS. In that same publication, James (1884) suggested that: “no shade of emotion, however slight, should be without a bodily reverberation as unique, when taken in its totality, as is the mental mood itself” (p. 192). This suggestion developed into the *autonomic specificity hypothesis*, the proposal that different functionally and phylogenetically “basic” or “discrete” emotions are characterized by qualitatively distinct profiles of ANS activation, and the corresponding patterns of organ-level effects (Levenson, 1988). Many studies have investigated the autonomic specificity of negative emotions, especially fear, anger, sadness, and disgust (for thorough reviews see Cacioppo et al., 2000; Kreibig, 2010). Yet few have explicitly considered the possibility of autonomic specificity among multiple varieties of positive emotion.

Unfortunately, few studies have included multiple positive emotion states as well as multiple physiological measures, and explicitly tested differences among the positive conditions. Christie and Friedman (2004) used a pattern classification approach to categorize emotion trials on the basis of profiles across heart rate, skin conductance, blood pressure (systolic, diastolic, and mean arterial), and mean successive differences in heart period (an alternative measure of vagal parasympathetic influence on the heart). Within-subjects emotion conditions, all manipulated by short film clips, included amusement and contentment as well as several negative-valence emotions and a neutral control. Approximately one-third of amusement trials and two-thirds of contentment trials were classified correctly; although this indicates a non-trivial failure rate, especially for amusement, both proportions are well above chance. In a later follow-up study using a similar design and much wider range of physiological measures, 47 percent of amusement trials and 51 percent of contentment trials were correctly classified (Stephens, Christie, & Friedman, 2010).

In a recent study, Shiota and colleagues (2011) presented participants with sets of visual images evoking anticipatory enthusiasm, attachment love, nurturant love, amusement, and awe, as well as a set of affectively neutral images. Physiological measures included cardiac inter-beat interval (IBI, the inverse of heart rate, such that lower values equate with higher heart rate), pre-ejection period, number of galvanic skin responses, respiration rate, mean arterial pressure, and RSA (analyzed both with and without correction for change in respiration rate). Repeated measures analyses detected a significant emotion condition $\times$ physiological measure interaction, indicating that the positive emotion conditions were associated with qualitatively distinct profiles of responding across the various measures. Enthusiasm was associated with a global increase in sympathetic activation indicated by shortened IBI, greater number of skin conductance responses, increased mean arterial pressure, increased respiration rate, and a non-significant directional reduction in pre-ejection period (all effects are relative to neutral control). Attachment and nurturant love were both characterized by shortened IBI and

increased respiration rate that might have been due to increased beta-adrenergic sympathetic activation or parasympathetic withdrawal, but with no signs of alpha-adrenergic or cholinergic effects (e.g., blood pressure or galvanic skin response). Amusement was associated only with an increase in respiration rate. Awe was associated with a distinctive lengthening of PEP, indicating withdrawal of beta-adrenergic receptor-mediated sympathetic influence on the heart, as well as an increase in respiration rate. Enthusiasm, attachment love, nurturant love, and awe all led to decreases in RSA relative to neutral control, but these effects were no longer significant after controlling for the increases in respiration rate.

These three studies, combined with a comprehensive review of the available literature (Kreibig, 2010), offer preliminary support for autonomic specificity among positive emotions. However, much more work is needed to replicate these tentative effects, and to include other physiological measures and other positive states. One important possibility to consider is that emotions, as phenomena that develop over time, may also have physiological features that develop over time rather than having a single autonomic profile (Kreibig, *in press*). For example, the anticipation of and approach toward a loved one might be characterized by increased beta-adrenergic sympathetic activation, whereas emotional intimacy and physical contact might involve a vagal parasympathetic response. Future research should address explicitly the ways in which positive emotions and their autonomic profiles can unfold over time.

CONCLUSIONS, FUTURE DIRECTIONS, AND LIGHT AND DARK SIDE IMPLICATIONS

Despite early findings suggesting limited involvement of ANS activity in positive emotion, a growing body of work points to the richness of positive emotions' effects on the body. Multiple theories of the functions and autonomic aspects of positive emotion have been proposed, and each has received tentative and circumscribed empirical support. At this point, we conclude that the possibility of autonomic specificity among multiple positive emotions is alive and well, and consistent with the overall body of research. However, far more work is needed to address this hypothesis explicitly. Strong future studies of autonomic specificity would include multiple positive emotion states, either alone or in addition to negative emotions; use several measures of ANS responding that help tease apart the neural mechanisms listed above, and possibly others; include a neutral control condition as a reference; and explicitly compare the autonomic profiles of the positive emotions against each other and the neutral condition, as well as examining effects at the level of individual variables. Whether or not the strong version of the autonomic specificity hypothesis is ultimately supported by such research, the resulting data will allow a more sophisticated understanding of how positive emotion plays out in the body, and what the implications might be for important psychosocial and health outcomes as well as subjective experience.

As others have noted, the potential light side of autonomic involvement in positive emotion psychophysiology is great. Several theorists have proposed that positive emotions are important buffers against stress, not only alleviating subjective distress, but also serving to protect against the health consequences of chronic stress-related sympathetic and hypothalamic-pituitary-adrenal (HPA) axis activation (e.g., Folkman, 1997; Fredrickson & Levenson, 1998; Lazarus et al., 1980). Polyvagal theory, while controversial, suggests one specific mechanism by which this might

take place (Porges, 2009). Evidence is growing that positive emotion may alter stress-related cardiovascular activity, and have downstream health protective effects (e.g., Cosley et al, 2010; Pressman & Cohen, 2005; Steptoe, Wardle, & Marmot, 2005). Investigating the pathways underlying these effects will be extremely important, especially as so much of the research on long-term health outcomes uses correlational designs. Health benefits of positive emotion may be mediated at least in part by autonomic responding and other peripheral physiological mechanisms (e.g., hormones), as well as by health behaviors (Pressman & Cohen, 2005). Establishing a link between short-term physiological effects of emotion in the laboratory and long-term health consequences in the real world will be challenging, but the attempt is certainly worthwhile.

There is a potential dark side to positive emotion physiology as well. Shiota and colleagues (2011) found that enthusiasm, the prototypical high-approach-motivation positive emotion, showed a sympathetically-driven autonomic profile that looked a great deal like fear. Other laboratory studies have also detected immune, cardiovascular, and pulmonary effects of intense positive emotion that could be harmful if experienced chronically (Pressman & Cohen, 2005). As has been observed with the psychological and behavioral implications of reward-focused positive emotion, it may be that too much of a good thing can become a serious problem (Gruber, 2011). Whether or not unregulated reward-focused positive emotion can have chronic health implications that are independent from their behavioral implications is a tricky but interesting question.

In a more distal, yet promising, future direction, measures of autonomic responding in positive emotion may be useful as additional indicators of clinical disorder, illuminating our understanding of both the light and dark sides of positive emotion. Psychophysiological measures can be used to help uncover and then assess mechanisms underlying psychopathology, as in disruptions of emotional responding in psychopathy and other externalizing disorders (e.g., Patrick, 2008; Patrick & Bernat, 2009). Some research already investigates possible autonomic markers of disturbances in positive emotion, as in the association of chronically elevated vagal tone with mania (e.g., Gruber et al., 2008). The theoretical analysis offered above suggests further possibilities, linking individual differences in enthusiasm-related autonomic responding to depression, for example, or linking disruption of vagal parasympathetic influence on the heart to attachment disorders. A great deal more basic research on autonomic aspects of positive emotion is needed to provide a strong context for this kind of research. In the long run, however, an understanding of the autonomic aspects of positive emotion could have considerable translational significance.

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