

Blood, Sweat, and Fears

The Autonomic Architecture of Emotion

ROBERT W. LEVENSON

*Department of Psychology, University of California, Berkeley,
Berkeley, California 94720-1650, USA*

ABSTRACT: The autonomic nervous system (ANS) plays a critical role in emotion, providing metabolic support for adaptive action, generating appearance changes with high signal value for conspecifics, and producing visceral sensations that shape subjective emotional experience. In this chapter, I consider several of the most important ways that the ANS is involved in emotion, including: (a) peripheral activation of emotion; (b) autonomic influences on emotional language and the labeling of subjective emotional experience; (c) positive emotion and autonomic soothing; (d) expressive signs of autonomic origin; (e) autonomic substrates of emotional contagion and empathy; and (f) autonomic consequences of emotion regulation. For each, I describe relevant research from our laboratory and discuss implications for an evolutionary account of emotion. In these and many other ways the autonomic architecture of human emotion has evolved not only to move blood and tears in the service of fears, but also to provide us with a rich set of tools that help us communicate and signal the nature of our internal emotional experiences, understand the emotions of others, calm ourselves and others, and give us some modicum of control over harmful and unproductive emotions.

KEYWORDS: emotion; autonomic nervous system; activation of emotion; deactivation of emotion; suppression of emotion; emotion regulation

The autonomic nervous system (ANS) is the body's most critical life support system, regulating a wide range of cardiovascular, gastrointestinal, electrodermal, respiratory, endocrine, and exocrine organs. The primary regulatory functions of the ANS can be organized broadly into three processes: (a) *maintenance* of an optimal "baseline" bodily milieu (i.e., homeostasis); (b) *activation* of bodily systems to support action in response to challenge and opportunity; and (c) *deactivation* of bodily systems when action is no longer

Address for correspondence: Robert W. Levenson, Institute of Personality and Social Research, 4143 Tolman Hall #5050, University of California, Berkeley, Berkeley, California 94720-5050. Voice: 510-642-5050; fax: 510-643-9334.
boblew@socrates.berkeley.edu

Ann. N.Y. Acad. Sci. 1000: 348–366 (2003). © 2003 New York Academy of Sciences.
doi: 10.1196/annals.1280.016

needed. Emotion and the ANS are intricately intertwined—emotions are a major consumer of autonomic resources, and the ANS provides a key to understanding many of the functions of human emotions. Because integration of emotion and the ANS is so important to our survival and reproduction, refinement of the autonomic architecture of emotion and integration of this architecture with other processing and control systems was undoubtedly an important part of human evolution. Despite enormous differences between contemporary and early evolutionary times, the autonomic architecture of emotion is still a remarkably effective and efficient design, which profoundly influences our day-to-day lives. In this chapter, I will discuss five key features of this architecture:

- (1) emotional activation: autonomic reactivity and peripheral activation;
- (2) language and labeling of subjective emotional experience: the autonomic element;
- (3) positive emotion: autonomic soothing;
- (4) autonomic expression: signals from the viscera;
- (5) emotional contagion and empathy: the autonomic substrate;
- (6) emotion regulation: autonomic consequences.

I will not provide a comprehensive review of each of these topics, but rather will draw primarily on work done by my research group to help illustrate the commingling of emotional and autonomic functioning. In keeping with the theme of this volume, whenever possible I will speculate as to the evolutionary forces that may have shaped these interconnections.

EMOTIONAL ACTIVATION: AUTONOMIC REACTIVITY AND PERIPHERAL ACTIVATION

The ANS prepares the body for dealing with a range of internal and external challenges that require coordinated action. Many of these challenges are such that they require a rapid response (e.g., responding to the appearance of a predator) while others require a more gradual, sustained response (e.g., regulating blood flow to maintain core body temperature). There is often a life-and-death quality to ANS activation—failure to adjust can be fatal.

Autonomic Reactivity

Human emotions evolved to deal with situations of great significance to the individual and group in which a rapid, multisystem response is required. I have described this as follows:

Emotions are short-lived psychological-physiological phenomena that represent efficient modes of adaptation to changing environmental demands. Psychologically, emotions alter attention, shift certain behaviors upward in response

hierarchies, and activate relevant associative networks in memory. Physiologically, emotions rapidly organize the responses of disparate biological systems including facial expression, somatic muscular tonus, voice tone, autonomic nervous system activity, and endocrine activity to produce a bodily milieu that is optimal for effective response. Emotions serve to establish our position vis-à-vis our environment, pulling us toward certain people, objects, actions and ideas, and pushing us away from others. Emotions also serve as a repository for innate and learned influences, possessing certain invariant features, and others that show considerable variation across individuals, groups, and cultures.¹

I have written previously about my views on the evolution of emotion activation, referring to this as the “core system” in emotion.² This core system is thought to have evolved to solve a set of elemental problems^{1,3-5} that are fairly common to all species as they interact with their external environment, with conspecifics, and with members of other species.

The core system has all of the capabilities necessary for processing incoming information continuously and for detecting a small number of prototypical situations that have profound implications for the organism’s immediate well-being and long-term survival. Having recognized in the stream of incoming perceptual information the configuration of features that defines one of a small number of prototypical situations, the core system activates an emotion, which is comprised of a set of response tendencies that have been selected by evolution for their high probability of dealing successfully and efficiently with the problems posed by that particular situation.²

For humans, the response package spans a number of different systems including perception/attention,⁶ gross motor behavior, purposeful behavior,⁷ expressive behavior,^{8,9} gating of higher mental processes,¹⁰ and physiological support.^{11,12}

Among the physiological systems that are activated by emotion, the ANS is particularly important. The ANS must prepare the body for a set of diverse actions that include (but are not limited to) fighting, fleeing, freezing, comforting, bonding, and expelling, each of which requires somewhat different configurations of physiological support.¹³ Like a modern factory that subscribes to the “just in time” model of inventory control, the ANS not only has to deliver sufficient quantities of all of the components needed to craft an appropriate response, but also has to deliver them at precisely the right time, and then quickly remove anything that is unused. This daunting task is made all the more difficult by the fact that emotion-eliciting situations are dynamic rather than static; thus, midcourse adjustments in physiological support are the rule rather than the exception.

Peripheral Activation

In the account of emotional activation presented above, the sequence begins with an external or internal stimulus, progresses through a process of

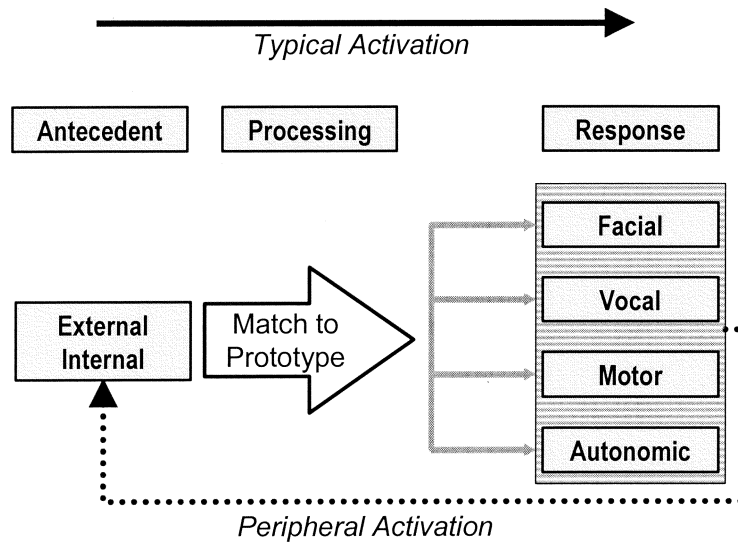


FIGURE 1. Emotional activation sequence begins with an external or internal stimulus, progresses through a process of matching percepts to prototypes, and moves on to the activation of various response systems including the autonomic nervous system; direction of activation is indicated as flowing from left to right.

matching percepts to prototypes, and moves on to the activation of various response systems including the autonomic nervous system. This sequence is depicted in FIGURE 1, with the typical direction of activation indicated as flowing from left to right. However, this is only one of many possible ways that emotion can be activated. In fact, it is becoming increasingly clear that activation can flow from right to left as well, as indicated by the dotted arrow labeled *Peripheral Activation* in the figure. Thus, any of the so-called response systems can become the initiating event for emotional activation. James advanced a particularly cogent version of this notion, stating:

...[T]he emotion both begins and ends with what we call its effects or manifestations. ...[A]ny voluntary arousal of the so-called manifestations of a special emotion ought to give us the emotion itself.¹⁴

From an evolutionary perspective, if emotion is viewed as highly critical to survival, then an activation system with multiple initiation points would have considerable adaptive advantage.

In a series of studies¹⁵⁻¹⁸ we have used the Directed Facial Action task to examine the capacity of one response system (the face) to initiate the emotion sequence. In this task, subjects are given specific muscle-by-muscle instructions (e.g., "raise your brows," "draw them together," etc.), which, if fol-

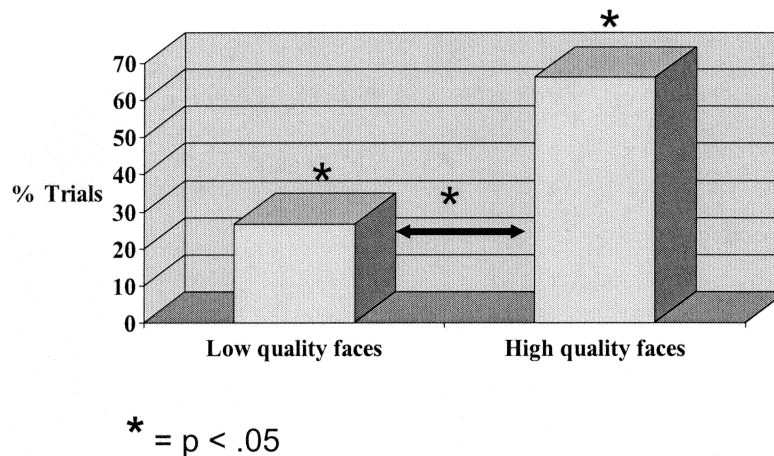


FIGURE 2. Reports of experiencing the emotions displayed on face. The more closely the faces resembled the associated prototypical expression, the greater the likelihood of experiencing the emotion.

lowed correctly, result in the production of a facial configuration that resembles a prototypical emotional expression (anger, disgust, fear, happiness, sadness, or surprise). A “coach,” viewing the subject’s face on a video monitor, provides coaching as needed to help the subject comply with the instructions (e.g., “raise your brows, but don’t bring them together”). Facial configurations are held for 10 seconds, during which time autonomic and somatic nervous system activity is measured. The experimental session is organized into trials, each consisting of a baseline rest period, instructions to produce a nonemotional facial configuration, instructions to produce an emotional facial configuration, and questions concerning thoughts, feelings, and sensations that might have occurred.

The first question to consider is whether these facial actions are sufficient for producing subjective emotional experience. Our findings are shown in FIGURE 2. Subjects reported experiencing the emotion associated with the facial configuration at significantly greater than chance levels (chance was one in six emotions or 16.6%). Importantly, when the configuration most closely matched the prototype, the subject was significantly more likely to report feeling the associated emotion than when the face did not closely match the prototype. In fact, with high-quality faces, subjects reported feeling the associated emotion on over 60% of trials. Those who conduct laboratory studies of emotion will recognize that a 60% hit rate would be very good for any procedure designed to produce particular emotions (e.g., films, slides). This finding^{19,20} provides important support for the capacity of the face to initiate subjective emotional experience.

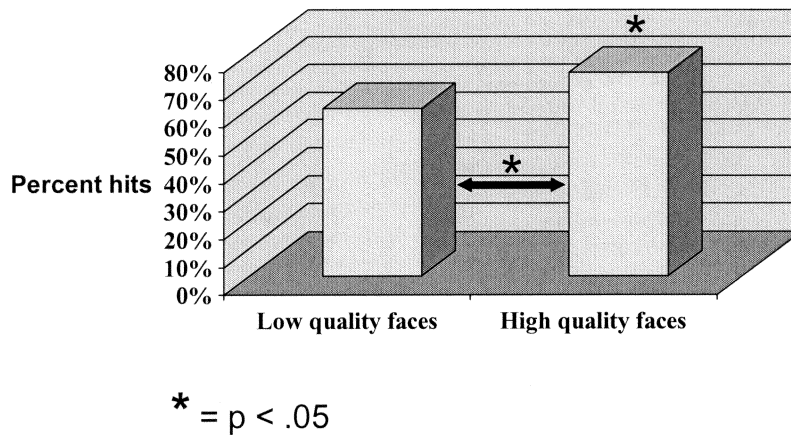


FIGURE 3. The extent to which four common autonomic differences among negative emotions (i.e., faster heart rate in anger versus disgust, fear versus disgust, sadness versus disgust; warmer skin temperature in anger versus fear) are found when emotions are activated using the Directed Facial Action task. As was the case with self-reported emotional experience data described earlier, the more closely faces resembled the associated prototypical expression, the greater the degree of autonomic differentiation among emotions.

Of course, subjective emotional experience is only one part of the picture (a part that is notoriously susceptible to demand characteristics and the like). Does the face have the capacity to activate the autonomic nervous system component as well? Our research¹⁷ suggests that the answer to this question is also yes. FIGURE 3 depicts the findings for the extent to which four common autonomic differences among negative emotions (i.e., faster heart rate in anger versus disgust, fear versus disgust, sadness versus disgust; warmer skin temperature in anger versus fear) are found when emotions are activated using the Directed Facial Action task. As was the case with self-reported emotional experience data described earlier, as faces more closely resembled the associated prototypical expression, the degree of autonomic differentiation among emotions increased. Putting this latter finding in perspective, we found that the degree of autonomic differentiation produced by voluntary facial action was essentially equivalent to that produced by initiating emotion in a more “conventional” way via recalling emotional memories.²¹

These findings that voluntary facial expression can lead to self-reported emotional experience and differentiated autonomic activity provide support for the viability of peripheral activation by the face. We expect that similar activation occurs for involuntary facial expression (e.g., with automatic empathic mimicry of the facial expressions of others). Although it is possible that the face occupies a privileged position among peripheral responses in

terms of being able to activate emotion, we think that James¹⁴ is correct and that this capacity is shared with the other peripheral response systems as well.

LANGUAGE AND LABELING OF SUBJECTIVE EMOTIONAL EXPERIENCE: THE AUTONOMIC ELEMENT

We know precious little about subjective emotional experience—what it is and where it comes from. Unlike other emotion response systems (autonomic, motor, vocal, expression), which have been studied in multiple species, subjective emotional experience has been studied almost exclusively in humans. Whether subjective emotional experience exists in other species remains an interesting question (e.g., can dogs feel shame?). Currently, however, human language is the best tool available to us for studying these subjective states. Not surprisingly, the ease of collecting self-report data and their potential richness has made them the most popular measure in research on human emotions. Thus, much of what we know about human emotion rests on a foundation of self-report data. This, however, is clearly a mixed blessing. We know that self-reports of any kind are vulnerable to all sorts of biases (e.g., demand characteristics, social desirability biases). The fragility of emotion self-reports was illustrated quite dramatically four decades ago when Schachter and Singer²² showed that manipulating the cues in the environment could radically change what participants said they were feeling. Their model of emotion, which underscored the malleability of self-reported subjective experience, is captured in the following quotation:

...[G]iven a state of physiological arousal for which an individual has no explanation, he will label this state in terms of the cognitions available to him. ... [B]y manipulating the cognitions, ... we can manipulate his feelings in diverse directions.²²

Schachter and Singer were primarily concerned with understanding the ways that things *outside* of the person influence the labeling of emotional states. Internal sources of emotional experience were seen primarily as sources of “confusion” (i.e., perceptions of unexplained physiological arousal). But we know that it is not *necessary* to observe clowning or threatening others to feel and label emotions such as happiness or anger. Thus, the question remains: What is the stuff of which feelings are made? Based on a number of sources of evidence, it appears that the primary ingredients of our subjective emotional experience are visceral and somatic. In this regard, the phenomenologists’ challenge of describing what an emotion “feels” like without reference to bodily changes is instructive. What can we say about the feeling of fear once we eliminate the changes in temperature, tension, breathing, and heart-beating? James illustrates this point very nicely:

If we fancy some strong emotion, and then try to abstract from our consciousness of it all the feelings of its characteristic bodily symptoms, we find we have nothing left behind...¹⁴

If our subjective emotional experience is based primarily on our perceptions of autonomic and somatic response systems, then the accuracy of our self-reports of emotion is going to be linked in part to the accuracy of our visceral perceptions. Research on visceral perceptions clearly indicates that we are not very accurate at such tasks as tracking our heart beats or estimating our current autonomic levels.^{23,24} But our limited accuracy in these demanding tasks does not mean that there is no connection between physiological activity and verbal report. Rather, the very language we use to talk about our emotions is replete with metaphors that translate the emotional into the physiological.²⁵

To evaluate the association between physiology and language, we²⁶ devised a procedure to assess the connection between autonomic nervous system activity and emotional language. We created verbatim transcripts of 143 unrehearsed 15-minute conversations between spouses about a problem area in their marriage.²⁷ These conversations are known to be rich sources of emotion.¹⁶ Working from the transcripts, we located all metaphors that suggested heat and pressure (e.g., "I'm really burning up," "I'm going to blow my top"). Across the 15-minute conversations, couples used between 0 and 5 of these metaphors. We correlated the number of metaphors with ANS levels averaged over the 15-minute conversation. Results revealed a striking relationship between cardiovascular levels and the use of these heat and pressure metaphors: the greater the amount of blood in the fingers, the faster the blood moved from the heart to the fingers, and the warmer the hand temperature, the more heat and pressure metaphors the couple used. Thus, there was a clear association between particular kinds of autonomic (cardiovascular) activity and the use of metaphors that were suggestive of that activity. Because of the correlational nature of this study, the direction of causality cannot be determined. Still these findings support the notion that our subjective experience of emotion (as reflected in the metaphors we use to describe our emotions) reflects our underlying state of autonomic activation. Similar studies looking at emotional language in relation to motor, vocal, and facial activity would be very useful in exploring the breadth and depth of the relationship between the language of emotion and the internal physiological milieu.

AUTONOMIC EXPRESSION: SIGNALS FROM THE VISCERA

The study of expressive behavior in emotion has focused primarily on the face and voice. This research has been facilitated greatly by the availability of tools for precisely quantifying facial expression²⁸ and for measuring the acoustic properties of vocalizations.²⁹ Both of these expressive systems

TABLE 1. Changes in appearance and ANS activity associated with specific emotions

Type	Change	ANS-Mediated Basis	Emotion
Coloration	reddening	vasodilation, increased contractility	anger
	blushing	vasodilation	embarrassment
	blanching	vasoconstriction	fear
Moisture and secretions	sweating, clamminess	sweat glands	fear
	salivating, drooling	salivary glands	disgust
	foaming	salivary glands	anger
	tearing, crying	lacrimal glands	sadness
	lubricating	mucus membranes	sexual arousal
Protrusions	piloerection	muscle fibers at base of hair follicles	fear, anger
	genital erection	vasodilation	sexual arousal
	blood vessels bulging	vasodilation	anger
Appearance of eyes	constriction	pupils	anger
	dilation	pupils	fear
	bulging	eyelid muscles	anger, fear
	drooping lids	eyelid muscles	sexual arousal
	twinkling	lacrimal glands plus contraction of orbicularis oculi	happiness

clearly have high signal value for conspecifics. Although the primary role of the ANS in emotion is usually thought to be providing physiological support for action, many of these autonomic adjustments create appearance changes that have strong signal value. Most prominent are those that produce visible changes in color, moisture, protrusion, and in the appearance of the eyes. In TABLE 1, I have listed some of these appearance changes along with the likely underlying ANS activity and the emotions they typically signal.

This list, which is intended to be illustrative rather than exhaustive, is indicative of the large number of visible signs of ANS changes in emotion that have developed signal value for conspecifics. To the extent that these visible signs are involuntary and merely reflect the activity of bodily systems, their signal value best fits Darwin's³⁰ third principal of expression (i.e., direct action of the nervous system). That humans make decisions, plan strategies, and

regulate their behavior in response to these signs of underlying autonomic activity in others underscores the utility and value of these signs as indicators of emotional states.

EMOTIONAL CONTAGION AND EMPATHY: THE AUTONOMIC SUBSTRATE

For social species, there are enormous advantages to having a mechanism by which emotions can be transferred quickly and efficiently across individuals. In humans, emotion contagion serves a number of functions including: (a) alerting, (b) calming, and (c) empathy. In the case of alerting, the emotion induced in an individual who has directly experienced a dangerous situation can be transmitted quickly to others who have not experienced the danger directly. When this works well, the group can be quickly mobilized to attack, defend, or flee. However, if the group response is out of proportion to the original threat, it can lead to inappropriate group behavior such as panic or violence. In the case of calming, signs of positive affect (e.g., smiles, laughter) in the face of potentially dangerous situations can defuse group activation.

Empathic contagion usually operates on a smaller, more intimate scale, with one person coming to know or feel the emotions of another person. Empathy is critical to social bonding and attachment, and, under appropriate conditions, can lead to prosocial, helping behaviors. Because empathy often occurs in dyads, it is quite amenable to laboratory study. For this reason, much of what we know about the role that the ANS plays in emotional contagion comes from studies of empathy. In the typical study, ANS activation in a person who is observing another person's distress is interpreted as indicating that emotion contagion has occurred.^{31,32}

In our work on emotional contagion, we have proposed that when the ANS physiology of two people shows "linkage" (i.e., one person's patterns of activation and deactivation across ANS measures mirror those of another person), there is emotional involvement. In our work studying the interactions of married couples, we found that high levels of physiological synchrony between spouses when discussing marital problems were most likely to be found in the most unhappily married couples.³³ We hypothesized that physiological linkage in this instance was an indicator of the high level of contagion and exchange of negative emotions that occur in unhappy marriages. This ebb and flow of negative emotion produces concomitant parallel patterns of ANS activation and deactivation in the spouses.

In later studies, we turned more explicitly to empathic accuracy (i.e., the extent to which one person can know the emotions of another) in strangers. Here we found that higher accuracy in detecting the negative emotions of an-

other person was associated with higher degrees of physiological linkage between the person detecting the emotions and the person whose emotions were being detected.³⁴ We speculated that this physiological linkage results in part from a process of emotional contagion through which the observers who rate the emotions of others most accurately have emotions that are similar in type and timing to those experienced by the person being observed. A subsequent study suggested that those individuals who are most accurate at rating the emotions of others do in fact show the most facial evidence of emotion.³⁵ However, questions of whether these signs of emotional contagion are similar in type and timing to those of the person being observed and whether they are the basis of the observed physiological linkage remain untested. In the meantime, the possibility that ANS linkage between individuals is a nonverbal marker of emotional contagion and empathy remains an intriguing possibility that could provide a very useful tool for research in humans and other species.

POSITIVE EMOTION: AUTONOMIC SOOTHING

Evolutionary accounts of emotion often start (and end) with the threat of predators and the survival advantages associated with rapid activation in support of combat or escape. Thus, it is not surprising that “fight” and “flight” have been the dominant metaphors in evolutionary accounts of human emotion. Accordingly, we know a great deal more about the activating aspects of human emotion than we do about its deactivating aspects. This imbalance is quite unfortunate. While human evolution no doubt looked favorably on the ability to activate when danger was present, it also likely favored the ability to calm down when safety was restored. Human emotions are intimately involved in coping with both danger and safety. Analogously, we would expect that emotions would also be involved both in rapidly activating and in rapidly deactivating physiological systems.

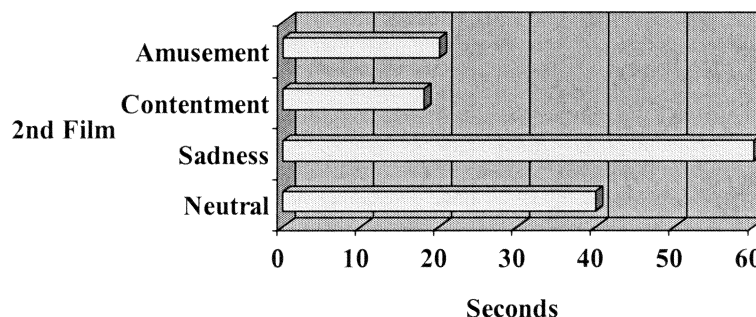


FIGURE 4. Cardiovascular response: time to return to baseline.

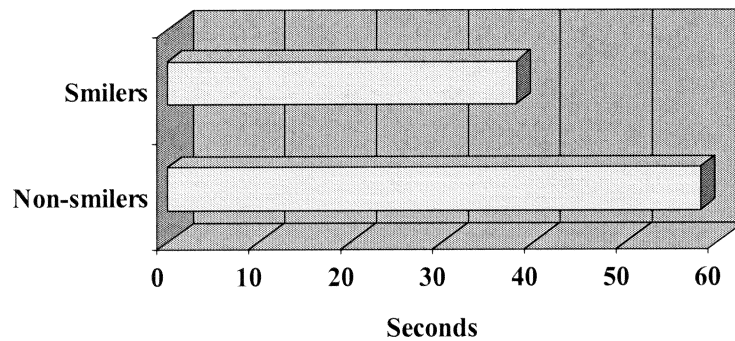


FIGURE 5. Cardiovascular response: time to return to baseline—smilers versus non-smilers.

In thinking about the role that emotions play in efficiently activating physiological systems, “negative” emotions such as fear, anger, and disgust come to mind. These emotions seem to have the capacity to activate physiological systems in ways that provide optimal support for actions such as fleeing, fighting, and expelling.^{2,13} In contrast, when considering the role that emotions play in efficiently deactivating physiological systems, “positive” emotions such as amusement, contentment, and happiness come to mind.³⁶

To test the role that positive emotions play in deactivating or “undoing” physiological arousal, we³⁷ conducted two studies, one experimental and one correlational. In the first study, subjects watched a fear-inducing film that was followed immediately by one of four other films (which either induced sadness, amusement, or contentment, or was emotionally neutral). Our dependent measure was the time it took participants to calm down from the cardiovascular arousal caused by the fear-inducing film. The results can be seen in FIGURE 4, which shows that return to the pre-fear film baseline levels was twice as fast when the second film induced positive emotion (either amusement or contentment) as when the second film was emotionally neutral. Moreover, this return to baseline associated with positive emotional films was three times as fast as that associated with a second film that induced a negative emotion (sadness).

In our second study, we explored a situation that we often see in the laboratory and in life—people showing positive affect in situations where we would expect negative emotion. A classic example of this is the laughter that often follows the most shocking moments in a horror film. In accordance with our thinking about positive emotions, these intrusions of positive emotion may be quite functional, taking advantage of their capacity to calm physiology and reduce tension. In our study, we showed participants a very sad film and coded their facial expressions to detect participants who smiled during

the film. Dividing the sample into those who smiled and those who did not, we compared the two groups in terms of how long it took for their cardiovascular system to calm down (i.e., return to prefilm baseline levels) once the film had ended. As can be seen in FIGURE 5, those who smiled during the sad film calmed down cardiovascularly almost twice as fast as those who did not smile.

These two studies support the notion that certain positive emotions play an important role in rapidly restoring physiological calm. In these studies, this effect was found for positive emotions such as amusement and contentment and for smiles. I expect that this may hold true for some positive emotions,³⁸ but not for others (e.g., exhilaration). It is also important to acknowledge that this calming effect is only one of several functions of positive emotions that likely played an important role in their evolution. Positive emotions also are critical in building interpersonal attachments and in shaping and reinforcing the behavior of others. Moreover, they play an important role in creating conditions conducive to expansive thinking and creativity.^{39,40}

EMOTION REGULATION: AUTONOMIC CONSEQUENCES

In moments of extraordinary danger where there is scant time for pondering options and planning strategies, we are well served by having a simple emotional system that does a few things exceedingly well, automatically choosing precisely those actions that have the highest likelihood of success, and quickly adjusting bodily systems to create the optimal support for the se-

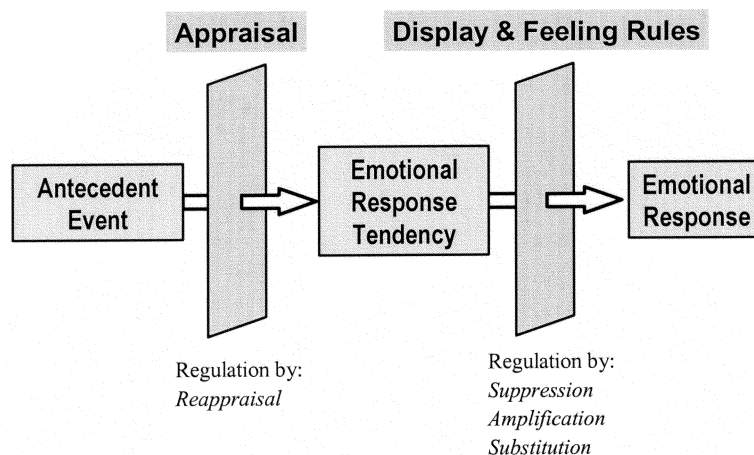


FIGURE 6. Model of emotion and the regulation of emotion. (Reprinted from Levenson² with permission.)

lected action.² During the human evolutionary period, selection pressures likely worked in favor of this kind of system. As long as our major threats were of the “predators jumping out of trees” variety, this kind of emotion system served us well. In more contemporary times, however, such an automatic system is often not advantageous. Many of the situations that threaten us today are more nuanced, more psychological, and more chronic; and, more often than not, we have to try to deflect our powerful initial emotional tendencies in the service of our own welfare and that of others.

Humans spend a great deal of energy in the service of learning to control their emotions. Training in these skills begins in early childhood; acquired skills are sorely tested by adolescence; and, arguably, we may achieve true expertise in emotion regulation only late in life.⁴¹ Although there are myriad specific strategies for emotional control,⁴² most of the time we seem to control our emotions in one of two primary ways: (a) changing the ways we appraise incoming information, and (b) altering the natural transition between *tendencies* to respond in a given way and the *actual* responses we produce. FIGURE 6 portrays a simplified schematic of a model of emotion and emotion regulation that I have presented elsewhere.² In this model, the two primary forms of emotional regulation are shown as vertical planes that: (a) early in the process, interrupt the flow between antecedent events and the activation of emotional response tendencies, and (b) later in the process interrupt the flow between emotional response tendencies and the actual emotional response.

Because the ANS response is such an important aspect of “unregulated” emotion, the question is raised as to how various regulation strategies impact this system. In a series of studies, we^{43,44} examined the effect of emotional suppression, a variant of the second kind of regulation strategy that involves attempting to reduce the visible signs of emotion. In these studies subjects were shown emotion-eliciting films and asked to “behave so that somebody

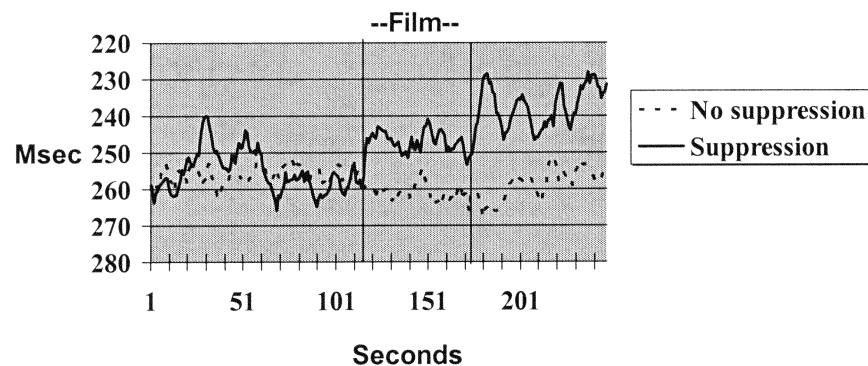


FIGURE 7. Finger pulse transmission time.

watching you would not know that you were feeling anything.” We found that for several different emotions participants could dramatically reduce the visible signs of emotion, and that this reduction had little effect on the strength of their self-reported emotional experience. However, this voluntary suppression of emotion produced large increases in the amount of cardiovascular activation compared to controls who merely watched the films. FIGURE 7 illustrates this finding by depicting the aggregated cardiovascular responses of participants who either watched a disgust-eliciting film or watched it and tried to suppress their emotional responses.⁴³ In this figure, greater cardiovascular activation is indicated by changes in the upward direction. As can readily be seen, the group that suppressed the behavioral signs of emotion had greater cardiovascular activation during the film, and this continued even after the film was over.

We interpreted these cardiovascular changes as indicating that stopping the behavioral manifestations of an emotion once the emotion has been set into motion requires significant levels of effort and this exacts a substantial metabolic cost. But are the costs and benefits of all kinds of emotion regulation the same?

Gross⁴⁵ compared the effects of variants of the two primary kinds of emotion regulation when he compared suppression with reappraisal (by asking subjects to “adopt a detached and unemotional attitude ... such ... that you don’t feel anything at all”). He found that reappraisal reduces the behavioral manifestations of emotion (but not as completely as suppression did) and reduces the level of subjective emotional experience without exacting the large physiological cost that suppression engenders. FIGURE 8 summarizes the findings from this entire series of studies.

Although reappraisal is not as effective as suppression in eliminating the behavioral signs of emotion, its relatively low metabolic cost underscores its value as a means of controlling emotion. Moreover, reappraisal also has the advantage over suppression in reducing subjective emotional experience

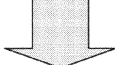
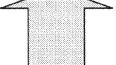
	Face	Subjective experience	Autonomic
Suppression 	Large reduction 	No change 	Large increase 
Reappraisal 	Reduction 	Reduction 	No change 

FIGURE 8. Summary of studies of suppression and reappraisal of emotion.

(e.g., when reducing the experience of negative emotions is deemed desirable). Unfortunately, emotion-eliciting events do not always lend themselves to this kind of intervention early in the course of activation—in many situations, the later-acting suppression strategy, with its greater metabolic costs, may be the only option.

THE AUTONOMIC ARCHITECTURE OF EMOTION: FINAL THOUGHTS

In traditional accounts of emotion the ANS does a lot of heavy lifting, creating the physiological milieu necessary to support behavioral action. In this kind of model, if emotion is the stimulus, then ANS activity is the response. In this chapter I have tried to expand this simple model by demonstrating that the role the ANS plays in emotion is far more elaborate and pervasive.

The ANS is intimately involved in our subjective emotional experience, helping us know that we are feeling something, what it is that we are feeling, and how to label the feeling. Growing out of this important contribution to the phenomenology of emotion, autonomic percepts influence the way we describe and talk about our emotions to others. The metaphors we use to enliven and vivify our emotional language are predominately autonomic in origin. In addition to language, we communicate our emotions to others via a universally recognized set of signs and signals. Although facial expressions and vocalizations garner most of the attention in this research area, there is a rich and arguably equally universal set of appearance changes of autonomic origin. These changes give notice to others of what we are feeling in ways that can profoundly influence their actions and feelings.

Continuing in this social/interpersonal vein, emotions are also contagious. We become emotional in the presence of others who are emotional, sometimes as part of group processes of alerting and calming and sometimes in the more intimate realms of interpersonal empathy. In empathy, the ANS plays an important role as a marker of states of shared and interconnected emotion. No doubt this is another area where appearance changes of autonomic origin play an important role in communicating to others that we are feeling things in response to their feelings. Moreover, this autonomic substrate of empathy has the promise of providing us with a nonverbal window onto this elusive phenomenon that could be extremely useful in emotion research.

The ANS is profoundly intertwined in processes of emotional soothing. Certain positive emotions have the capacity to hasten the restoration of ANS equilibrium in the aftermath of ANS activation produced by negative emotions. This kind of tension reduction is enormously reinforcing and no doubt plays an important role in building attachment bonds when relationships are experienced as soothing and emotionally positive.

The ANS provides a way of understanding the impact and costs of the various ways we attempt to regulate our emotions. Strategies that attempt to shut down the behavioral manifestations of emotions after they have gained a full head of steam are very costly in terms of additional ANS activation. In contrast, more preemptive strategies that involve viewing the world in a different, less threatening way may have much the same result without engendering the physiological costs. Historically, emotions have been seen as having profound implications for health and wellness. Although a life replete with chronic, sustained activation of negative emotions will be autonomically costly, it may be even more detrimental to live a life in which negative emotions are chronically suppressed. At the risk of sounding like an evangelist for new-age religion, applying our rational powers in the service of viewing the world in different, less threatening ways; trying to look at things from a broader perspective; considering contextual factors; and being more accepting of the shortcoming of others may have tangible health benefits when they are part of reappraisal strategies of emotion regulation.

In these and many other ways the autonomic architecture of human emotion has evolved not only to move blood and tears in the service of fears, but also to provide us with a rich set of tools that help us communicate and signal the nature of our internal emotional experiences, understand the emotions of others, calm ourselves and others, and give us some modicum of control over harmful and unproductive emotions.

ACKNOWLEDGMENTS

Preparation of this chapter was supported by grants from the National Institute on Aging (AG17766 and AG19724) and the National Institute of Mental Health (MH50841).

REFERENCES

1. LEVENSON, R.W. 1994. Human emotion: a functional view. *In* *The Nature of Emotion: Fundamental Questions*. P. Ekman & R.J. Davidson, Eds.: 123–126. Oxford. New York.
2. LEVENSON, R.W. 1999. The intrapersonal functions of emotion. *Cogn. Emotion* **13**: 481–504.
3. EKMAN, P. 1992. An argument for basic emotions. *Cogn. Emotion* **6**: 169–200.
4. LAZARUS, R.S. 1991. *Emotion and Adaptation*. Oxford University Press. New York.
5. TOOBY, J. & L. COSMIDES. 1990. The past explains the present: emotional adaptations and the structure of ancestral environments. *Ethol. Sociobiol.* **11**: 375–424.
6. MATHEWS, A. & B.P. BRADLEY. 1983. Mood and the self-reference bias in recall. *Behav. Res. Ther.* **21**: 233–239.

7. FRIJDA, N.H. 1986. *The Emotions*. Cambridge University Press. Cambridge.
8. EKMAN, P. 1984. Expression and the nature of emotion. *In Approaches to Emotion*. K.R. Scherer & P. Ekman, Eds.: 319–343. Erlbaum. Hillsdale, NJ.
9. IZARD, C.E. 1971. *The Face of Emotion*. Appleton-Century-Crofts. New York.
10. BOWER, G.H. 1981. Mood and memory. *Am. Psychol.* **36**: 129–148.
11. DAVIDSON, R.J., P. EKMAN, C.D. SARON & J.A. SENULIS. 1990. Approach-withdrawal and cerebral asymmetry: emotional expression and brain physiology I. *J. Pers. Soc. Psychol.* **58**: 330–341.
12. LEVENSON, R.W. 1992. Autonomic nervous system differences among emotions. *Psychol. Sci.* **3**: 23–27.
13. LEVENSON, R.W. 2003. Autonomic specificity and emotion. *In Handbook of Affective Sciences*. R.J. Davidson, K.R. Scherer & H.H. Goldsmith, Eds.: 212–224. Oxford University Press. New York.
14. JAMES, W. 1884. What is an emotion? *Mind* **9**: 188–205.
15. EKMAN, P., R.W. LEVENSON & W.V. FRIESEN. 1983. Autonomic nervous system activity distinguishes among emotions. *Science* **221**: 1208–1210.
16. CARSTENSEN, L.L., J.M. GOTTMAN & R.W. LEVENSON. 1995. Emotional behavior in long-term marriage. *Psychol. Aging* **10**: 140–149.
17. LEVENSON, R.W., P. EKMAN & W.V. FRIESEN. 1990. Voluntary facial action generates emotion-specific autonomic nervous system activity. *Psychophysiology* **27**: 363–384.
18. LEVENSON, R.W., P. EKMAN, K. HEIDER & W.V. FRIESEN. 1992. Emotion and autonomic nervous system activity in the Minangkabau of West Sumatra. *J. Pers. Soc. Psychol.* **62**: 972–988.
19. DUCLOS, S.E., J.D. LAIRD, E. SCHNEIDER, *et al.* 1989. Emotion-specific effects of facial expressions and postures on emotional experience. *J. Pers. Soc. Psychol.* **57**: 100–108.
20. LAIRD, J.D. 1974. Self-attribution of emotion: the effects of expressive behavior on the quality of emotional experience. *J. Pers. Soc. Psychol.* **29**: 475–486.
21. LEVENSON, R.W., L.L. CARSTENSEN, W.V. FRIESEN & P. EKMAN. 1991. Emotion, physiology, and expression in old age. *Psychol. Aging* **6**: 28–35.
22. SCHACHTER, S. & J.E. SINGER. 1962. Cognitive, social, and physiological determinants of emotional state. *Psychol. Rev.* **69**: 379–399.
23. KATKIN, E.S., J. BLASCOVICH & S. GOLDBAND. 1981. Empirical assessment of visceral self-perception: individual and sex differences in the acquisition of heart beat discrimination. *J. Pers. Soc. Psychol.* **40**: 1095–1101.
24. PENNEBAKER, J.W. 1982. *The Psychology of Physical Symptoms*. Springer-Verlag. New York.
25. LAKOFF, G. 1987. *Women, Fire, and Dangerous Things*. University of Chicago Press. Chicago.
26. MARCHITELLI, L. & R.W. LEVENSON. 1992. When couples converse: the language and physiology of emotion. Paper presented at the Society for Psychophysiological Research, San Diego, CA.
27. LEVENSON, R.W., L.L. CARSTENSEN & J.M. GOTTMAN. 1994. Influence of age and gender on affect, physiology, and their interrelations: a study of long-term marriages. *J. Pers. Soc. Psychol.* **67**: 56–68.
28. EKMAN, P. & W.V. FRIESEN. 1978. *Facial Action Coding System*. Consulting Psychologists Press. Palo Alto, CA.
29. SCHERER, K.R. 1989. *Vocal Measurement of Emotion*. Academic Press, Inc. San Diego, CA.

30. DARWIN, C. 1872. *The Expression of the Emotions in Man and Animals*. Murray, London.
31. VAUGHAN, K.B. & J.T. LANZETTA. 1980. Vicarious instigation and conditioning of facial expressive and autonomic responses to a model's expressive display of pain. *J. Pers. Soc. Psychol.* **38**: 909–923.
32. WIESENFELD, A.R., P.B. WHITMAN & C.Z. MALATESTA. 1984. Individual differences among adult women in sensitivity to infants: evidence in support of an empathy concept. *J. Pers. Soc. Psychol.* **46**: 118–124.
33. LEVENSON, R.W. & J.M. GOTTMAN. 1983. Marital interaction: physiological linkage and affective exchange. *J. Pers. Soc. Psychol.* **45**: 587–597.
34. LEVENSON, R.W. & A.M. RUEF. 1992. Empathy: a physiological substrate. *J. Pers. Soc. Psychol.* **63**: 234–246.
35. SOTO, J., N. POLE, L. MCCARTER & R.W. LEVENSON. 1998. Knowing feelings and feeling feelings: are they connected? Paper presented at the Society for Psychophysiological Research, Denver, CO.
36. LEVENSON, R.W. 1988. Emotion and the autonomic nervous system: a prospectus for research on autonomic specificity. In *Social Psychophysiology and Emotion: Theory and Clinical Applications*. H.L. Wagner, Ed.: 17–42. John Wiley & Sons, Chichester, England.
37. FREDRICKSON, B.L. & R.W. LEVENSON. 1998. Positive emotions speed recovery from the cardiovascular sequelae of negative emotions. *Cogn. Emotion* **12**: 191–220.
38. KELTNER, D. & J. HAIDT. 2003. Approaching awe, a moral, spiritual, and aesthetic emotion. *Cogn. Emotion* **17**: 297–314.
39. FREDRICKSON, B.L. 1998. What good are positive emotions? *Rev. Gen. Psychol.* **2**: 300–319.
40. ISEN, A.M. 1999. *Positive Affect*. John Wiley, New York.
41. GROSS, J.J. & R.W. LEVENSON. 1997. Hiding feelings: the acute effects of inhibiting negative and positive emotion. *J. Abnorm. Psychol.* **106**: 95–103.
42. GROSS, J.J. 1998. Antecedent- and response-focused emotion regulation: divergent consequences for experience, expression, and physiology. *J. Pers. Soc. Psychol.* **74**: 224–237.
43. GROSS, J.J. & R.W. LEVENSON. 1993. Emotional suppression: physiology, self-report, and expressive behavior. *J. Pers. Soc. Psychol.* **64**: 970–986.
44. GROSS, J.J., L.L. CARSTENSEN, M. PASUPATHI, *et al.* 1997. Emotion and aging: experience, expression, and control. *Psychol. Aging* **12**: 590–599.
45. GROSS, J.J. 1998. The emerging field of emotion regulation: an integrative review. *Rev. Gen. Psychol.* **2**: 271–299.