

Symposium on Emotion

AUTONOMIC NERVOUS SYSTEM DIFFERENCES AMONG EMOTIONS

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Abstract—Following decades of controversy and uncertainty, there is now sufficient empirical basis for asserting the existence of a limited set of autonomic differences among emotions. Findings of autonomic distinctions among emotions derived from the work of the author and his colleagues using two methods of emotion elicitation are reviewed. For five of these autonomic distinctions, convergent findings from the work of other investigators using the same and other elicitation methods are presented.

The question of specificity, or whether different emotions are associated with different patterns of autonomic nervous system (ANS) activity, has long captivated emotion research, with the roots of the controversy tracing to James (1884), who argued for specificity, and Cannon (1927), who argued against. The state of affairs circa 1950 was captured in Woodworth and Schlosberg's (1954) experimental psychology textbook:

Ever since psychologists started the study of bodily changes during emotion, there has been the hope that some patterns would turn up that would differentiate one emotion from another. There have been a number of hopeful leads, but they have not turned out very well. . . . (p. 183)

With the advent of modern psychophysiological methods, a flurry of research in the 1950s generated evidence for specificity (e.g., Ax, 1953; Funkenstein, King, & Drollette, 1954; Schachter, 1957), but any movement toward general acceptance of this view was short-lived. In the 1960s, the zeitgeist in psychology was clearly that of undifferentiated ANS activity in emo-

tion, as reflected in several influential cognitively oriented theories of emotion (Duffy, 1962; Lazarus, Averill, & Opton, 1970; Mandler, 1962, 1975; Schachter & Singer, 1962). For these theorists, ANS differentiation in emotion most likely did not exist, and, even if it did, it was of little consequence.

It should be noted that to establish specificity does not require demonstrating that every emotion has a unique ANS signature (as proposed by Alexander, 1950), but only that some emotions differ from others in consistent ways. For specificity to transcend the merely trivial, it must consist of more than some negative emotions (e.g., fear, anger) having higher levels of "global" ANS arousal than some positive emotions (e.g., contentment, calm).

Why has establishing ANS specificity been so elusive? Studying specificity is inherently difficult. Emotions are short-lived phenomena, typically lasting only for seconds (Ekman, 1984) and occurring in complex contexts cohabited by other psychological processes such as attention/orientation, appraisal, impression management, and social interaction. Emotion-relevant ANS activity is similarly imposed on an ongoing stream of physiological activity occasioned by the ANS acting in service of its many masters, both internal (e.g., homeostasis, response to metabolic demands) and external (e.g., orientating, defense). Isolating an emotion and a segment of associated physiology for study is difficult at best.

EVIDENCE FROM OUR LABORATORY

Specificity has been a central issue in my work (conducted with Carstensen, Ekman, Heider, and Friesen), which has studied six emotions (anger, disgust, fear, happiness, sadness, and surprise) elicited in two ways: (a) the *directed facial action task*, in which subjects are instructed and coached to contract certain facial muscles to produce facial con-

figurations that are morphologically identical to prototypical emotional facial expressions, and (b) the *relived emotions task*, in which subjects recall and relive emotional memories for each emotion.

Directed Facial Action Task

Autonomic differences

Three experiments with young American subjects revealed several reliable ANS distinctions among negative emotional configurations (Levenson, Ekman, & Friesen, 1990): (a) anger, fear, and sadness produced larger heart rate acceleration than disgust; and (b) anger produced larger finger temperature increase than fear.

Differences between negative and positive emotional configurations were also found: (a) anger and fear produced larger heart rate acceleration than happiness; and (b) fear and disgust produced larger skin conductance increase than happiness. In this paper, I will focus on distinctions involving negative emotions because I consider them to be more important for demonstrating autonomic specificity and because they appear to be more robust.

Relation to emotion

We hypothesized that these particular configurations of facial muscle contractions produced these particular ANS differences because they were *emotional* configurations. Evidence supporting a relation to emotion derived from numerous sources: (a) subjects reported experiencing the emotions associated with the configurations; (b) when configurations most closely resembled the prototypical emotional expressions, emotional report was more frequent; (c) when configurations most closely resembled the prototypical emotional expressions, ANS differences were more pronounced; and (d) when the associated emotion was reported, ANS differences were more pronounced.

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We examined three alternative explanations for these findings, any of which would have undermined interpretation in terms of emotion. Neither concomitant activity of nonfacial muscles, nor number of facial muscles contracted in the configurations, nor difficulty of producing the configurations was found to account for the findings (Levenson et al., 1990).

Generality

Initially (Ekman, Levenson, & Friesen, 1983), we used actors and facial researchers as subjects. In subsequent studies, it was found that findings generalized to subjects without specialized training (Levenson et al., 1990).

To determine whether patterns of emotion-specific ANS activity were maintained in very old age, a sample of subjects aged 71 to 90 was recruited. We found the same ANS differences among negative emotions as found previously with young subjects; however, the magnitude of ANS changes was smaller among the elderly (Levenson, Carstensen, Friesen, & Ekman, 1991).

Across all experiments, with both young and elderly subjects, no important differences were found between males and females in any aspects of the findings.

To determine if the same ANS distinctions would be found in a non-Western culture, we (Levenson, 1989; Levenson, Ekman, Heider, & Friesen, 1991) traveled to West Sumatra to study the Minangkabau, a Moslem, matrilineal society with strong proscriptions against the public display of anger. Working with young male subjects who had no prior experience with scientific research, we found the same distinctions among negative emotions as had been found with American subjects.

With both American and Minangkabau subjects, distinctions among negative emotions obtained regardless of whether subjects had visual access to facial expressions (in a mirror or on the coach's face).

Relived Emotions Task

In two experiments with young subjects and one with elderly subjects, the ANS differences among negative emotions found using the directed facial action task also obtained when emotions

were elicited using the more conventional relived emotions task (Levenson, Carstensen, Friesen, & Ekman, 1991).

EVIDENCE FROM OTHER LABORATORIES

Finding that the ANS distinctions among negative emotions produced by the directed facial action task were also obtained when we used another more conventional eliciting task (i.e., relived emotions) greatly increased our confidence in the robustness of these distinctions and in their relation to emotion. Additional confidence would be obtained if these distinctions were not unique to our laboratory but were similar to those found by other investigators using the same and other methods.

To assess the strength of this convergent evidence, I will review studies from other laboratories¹ that are relevant to five aspects of ANS specificity derived from our work. Each of these five distinctions is based on findings that have been confirmed by us in multiple experiments and using both the directed facial action task (Ekman et al., 1983; Levenson, 1989; Levenson et al., 1990; Levenson, Carstensen, Friesen, & Ekman, 1991; Levenson, Ekman, Heider, & Friesen, 1991) and the relived emotions task (Levenson, Carstensen, Friesen, & Ekman, 1991).

Heart Rate Acceleration During Anger

The association of anger with accelerated heart rate may reflect anger's close association with a motor program or action tendency (Frijda, 1986) of "fight," which makes significant metabolic demands on the heart.

Consistent findings include (a) large maximum heart rate increases in anger provoked by hostile experimenter (Ax,

1953; Schachter, 1957); (b) large percentage increases in heart rate during "anger in" provoked by criticism during mental arithmetic (Funkenstein et al., 1954); (c) larger increases in heart rate during anger imagery than happy or relaxing imagery (Schwartz, Weinberger, & Singer, 1981); (d) larger increases in heart rate during anger imagery than neutral imagery (Roberts & Weerts, 1982); (e) greater heart rate acceleration during anger facial expressions than joy or interest expressions in 4.5-month infants during infant-mother play (Cohen, Izard, & Simons, 1986); (f) greater heart rate acceleration during anger situation (removal of toys, restraint) than interest and joy situations in 9- and 12-month-old infants (Provost & Gouin-Decarie, 1979); (g) heart rate acceleration from baseline during anger imagery (Waters, Bernard, & Buco, 1989); (h) greater heart rate acceleration when provoked by hostile confederate than when not provoked (Frodi, 1978); (i) greater heart rate acceleration to videotape (with sound) of angry expressive displays of President Reagan than happy displays (McHugo, Lanzetta, Sullivan, Masters, & Englis, 1985); and (j) greater heart rate acceleration when harassed while solving anagrams than when not harassed (Van Egeren, Abelson, & Thornton, 1978).

Heart Rate Acceleration During Fear

The association of fear with accelerated heart rate may reflect fear's close association with the motor program of "flight,"² which makes significant metabolic demands on the heart.

Consistent findings include (a) large maximum heart rate increases in fear provoked by mild electric shock and incompetent experimenter (Ax, 1953; replicated by Schachter, 1957); (b) large percentage increases in heart rate during anxiety provoked by criticism during mental arithmetic (Funkenstein et al., 1954); (c) larger increases in heart rate during fear imagery than happy or relax-

1. Differences among studies in conceptualization, methodology, and completeness of reporting required some subjective judgment in characterizing findings. Omitted from this review are studies of (a) ANS concomitants of emotional dimensions such as "pleasantness"; (b) nonspecific affective constructs such as "stress" and "tension"; (c) facial expressions used to modulate ANS responses to external stimuli; and (d) self-reports of perceived physiological changes during emotion.

2. Animal studies suggest sharply accelerated heart rate also characterizes "freezing," another motor program often associated with fear (e.g., "motionless posture" in Duncan & Filshie, 1979; Jones, Duncan, & Hughes, 1981; "tonic immobility" in Gentle, Jones, & Woolley, 1989).

ing imagery (Schwartz et al., 1981); (d) greater heart rate increases during fear imagery than neutral imagery (Bauer & Craighead, 1979; Hirota & Hirai, 1986; Roberts & Weerts, 1982); (e) greater heart rate acceleration during posed fear expressions than calm or neutral expressions (McCaul, Holmes, & Solomon, 1982); (f) heart rate acceleration to mutilation slides in subjects high in fear of mutilation (Klorman & Ryan, 1980; Klorman, Weissberg, & Wiesenfeld, 1977; Klorman, Wiesenfeld, & Austin, 1975); (g) heart rate acceleration to slides of spiders in subjects high in fear of spiders (Hare, 1973); (h) greater heart rate increases during imagery of snakes and of social performance than neutral imagery (Hirota & Hirai, 1990); (i) heart rate acceleration from baseline during fear imagery (Waters et al., 1989); (j) greater heart rate acceleration to slides of phobic object than to neutral slides in animal and blood phobics (Fredrikson, Danielssons, Iremark, & Sundin, 1987); (k) heart rate increase from baseline during anxiety-eliciting film (Thyer, Papsdorf, Davis, & Vallecorsa, 1984); (l) greater heart rate acceleration during high-fear film than low-fear film (Mewborn & Rogers, 1979); (m) greater heart rate acceleration during personalized anxiety imagery than neutral imagery (Boudewyns & Levis, 1975); (n) greater heart rate acceleration during fear of impending interaction with uncontrollable child than with controllable child (Bugental & Cortez, 1988); (o) greater heart rate acceleration to videotape (with sound) of fear-expressive displays of President Reagan than happy displays (McHugo et al., 1985); (p) linear relation between magnitude of subjective fear and heart rate acceleration to real insect or animal (Sartory, Rachman, & Grey, 1977); (q) increasingly faster heart rate as stranger gets increasingly closer to 6- and 11-month-old infants (Skarin, 1977); (r) greater heart rate acceleration to snake imagery for snake phobics than nonphobics (May, 1977); (s) greater heart rate acceleration to public-speaking imagery for public-speaking phobics than nonphobics (Giesen & McGlynn, 1977); and (t) phobic fear associated with heart rate acceleration, which becomes less pronounced as phobias are desensitized (Lang, Melamed, & Hart, 1970).

Heart Rate Acceleration During Sadness

The association of heart rate acceleration with sadness suggests that sadness is characterized by high cardiovascular arousal, despite not being associated with high-activity motor programs such as fight or flight. This high arousal may function to make sadness particularly stressful, thus strongly reinforcing soothing social contacts that reduce this arousal (see Averill, 1969, for a similar analysis of grief).

Consistent findings include (a) greater heart rate increase during sad imagery than neutral imagery (Schwartz et al., 1981); (b) greater heart rate acceleration during sad facial expressions than anger, joy, or interest expressions in 4.5-month infants during infant-mother play (Cohen et al., 1986); (c) greater heart rate acceleration during distress situation (mother leaves room) than interest and joy situations in 9- and 12-month-old infants (Provost & Gouin-Decarie, 1979); and (d) heart rate acceleration from baseline during sad imagery (Waters et al., 1989).

Heart Rate Deceleration During Disgust

The association of heart rate deceleration (or lack of acceleration) with disgust suggests a quite different cardiovascular state than for the negative emotions of anger, fear, and sadness. Considerable parasympathetic activation likely occurs in disgust (as would be indicated by increased salivation and increased gastrointestinal activation), which could produce a vagally mediated slowing of heart rate. Unfortunately, disgust has rarely been included in studies of ANS specificity.

Consistent findings include heart rate deceleration to mutilation slides in subjects without mutilation phobias (Hare, 1972; Hare, Wood, Britain, & Frazelle, 1971; Hare, Wood, Britain, & Shadman, 1971; Klorman & Ryan, 1980; Klorman et al., 1975).

Peripheral Vascular Differences Between Fear and Anger

Compared with anger, fear appears to be associated with lower diastolic blood

pressure and with cooler surface temperatures, greater vasoconstriction, and lesser blood flow in the periphery. This pattern of physiological changes is consistent with a functional view that fear is associated with a "flight" motor program in which blood flow is redirected away from the periphery and toward large muscles of locomotion.

Consistent evidence contrasting fear and anger includes (a) smaller maximum diastolic blood pressure increase during fear induced by mild electric shock and incompetent experimenter than during anger induced by hostile experimenter (Ax, 1953); (b) smaller increase in diastolic blood pressure during high-intensity fear imagery than high-intensity anger imagery (Roberts & Weerts, 1982); (c) smaller increase in diastolic blood pressure during fear imagery than anger imagery (Schwartz et al., 1981); (d) larger decrease in hand temperature in fear provoked by hostile experimenter and by mild electric shock than in anger provoked by incompetent experimenter (Schachter, 1957); (e) lower hand temperature in fearful attitude of being on guard than in angerlike attitude of being mistreated (Graham, 1962); (f) greater decrease in head temperature during fear elicited by dramatic reading, music, and sudden darkness than during anger elicited by criticized anagram task (Stemmler, 1989); and (g) facial pallor during fearful states in daily life and facial blushing during angerlike states (Wolf & Wolff, 1947).

Studies investigating fear and anger separately also provide evidence consistent with these vascular differences: (a) cephalic vasoconstriction to fear slides (spiders) in spider phobics (Hare, 1973); (b) greater finger temperature decrease during fear imagery (snakes and social performance) than neutral imagery (Hirota & Hirai, 1990); (c) finger temperature decrease from baseline during anxiety-eliciting film (Thyer et al., 1984); (d) greater finger temperature decrease during fear of impending interaction with uncontrollable child than with controllable child (Bugental & Cortez, 1988); (e) greater finger temperature decrease to fearful imagery than neutral imagery (Hirota & Hirai, 1986); (f) finger temperature decrease to threat of shock and shock (Boudewyns, 1976); (g) greater diastolic blood pressure increase during

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anger (provocation by hostile confederate) than in no-provocation condition (Frodi, 1978); and (h) anger release (retaliation against abusive confederate) associated with lowering of diastolic blood pressure (Van Egeren et al., 1978).

EVIDENCE AGAINST ANS SPECIFICITY IN EMOTION

While there certainly have been studies that failed to find support for one or more of the five distinctions reviewed here, the weight of the evidence is overwhelmingly supportive. Interestingly, the single study most often cited in support of undifferentiated ANS activity in emotion is Schachter and Singer (1962), in which ANS activity was an *independent* variable (manipulated by injection of epinephrine in five of seven conditions). Clearly, for a study to speak to the extent of ANS differentiation in emotion, ANS activity must be a *dependent* variable.

Another basis for skepticism regarding specificity undoubtedly resides with the viewpoint, clearly articulated by Cannon (1927) and rooted in 19th-century ANS physiology (Bernard, 1878; Langley, 1901), that the ANS is inherently undifferentiated. Contemporary studies, however, suggest a much greater capacity for differentiated action in the form of multiple receptor subtypes in both the sympathetic (Ahlquist, 1948) and parasympathetic (Mitchelson, 1988) branches of the ANS. The recent dramatic increase of research on ANS neurotransmitters and neuropeptides (including their cotransmission and comodulation) and on central nervous system control of autonomic functions, as well as recent demonstrations of nonuniform distributions of receptors for neurotransmitters and neuropeptides throughout the ANS, provides a more than adequate basis for ANS specificity in emotion.

IMPLICATIONS FOR EMOTION THEORY

The existence of more than one pattern of ANS activation in emotion does not refute any particular theory of emotion, because few have been constructed so as to *require* an undifferentiated ANS in emotion. However, theories that have

assumed an undifferentiated ANS (e.g., Mandler, 1975) or that have been silent on the issue might need more explicit articulation of their position as to the function, significance, and implications of ANS differentiation in emotion.

These findings also have implications for traditional models of emotion elicitation in which changes in the environment, having been perceived by the organism and appraised at least at a simple "automatic" level, lead to the occurrence of an emotional state, which recruits and organizes subordinate response systems. While it is generally accepted that memories can serve as initiating stimuli for emotion, it is less accepted that other systems typically viewed as being "responses" (e.g., facial expression, the ANS, striate muscles, vocalization) can initiate emotion. Our work with the directed facial action task and that of others with voluntary facial action (Laird, 1974; McCaul et al., 1982) suggests that facial actions can also play a role in initiating emotion. I expect it will eventually be established that any component of emotion can assume this initiating role.³ Such plasticity in elicitation would clearly not be predicted by most current theories of emotion (an exception being Camras's [in press] extension of dynamical systems theory [Kugler, Kelso, & Turvey, 1980, 1982]).

CONCLUSION

I believe there is now sufficient empirical support for asserting the existence of a set of ANS distinctions among emotions. Beyond the five distinctions reviewed here, I anticipate that additional distinctions among negative emotions will be found, and that the evidence for previously found distinctions be-

3. Preliminary support for initiation of emotion by the ANS can be found in reports that ANS activation, absent other stimuli, produces negative affective experience (e.g., Marshall & Zimbardo, 1979). Anecdotal reports that emotion can be initiated by striate muscular action (as suggested by James, 1884) and by vocalization are found for several varieties of psychotherapy in which postures and vocalizations are purportedly used to access repressed emotions (e.g., Bioenergetics, Gestalt therapy).

tween negative and positive emotions will be strengthened. Nonetheless, the final tally of distinctions is likely to be small (Levenson, 1988).

It is surely proper to ask whether ANS distinctions among emotions, even if they do exist, are of any real consequence. Are they merely vestigial remnants of an earlier time when most stressors were well defined and punctate, and the most effective adaptive responses involved rapid deployment of highly stereotyped motor and autonomic reactions? With all due respect to changing environmental demands, I would propose that ANS changes in emotion continue to have major significance, standing in readiness for dealing with those situations in which lengthy and considered appraisal would threaten survival, influencing the way emotions "feel" to us subjectively, motivating behavior aimed at modulating their levels (e.g., soothing, thrill-seeking), coloring the language we use to talk about our feelings (Kovacs, 1989; Lakoff, 1987), serving as contextual cues that bind together networks of association and memory, and playing an important role in the processes of mental and physical health and disease.

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