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HANDBOOK OF EMOTIONS

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The Psychophysiology of Emotion

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The numerous chapters that constitute this handbook testify to the breadth and complexity of the topic of emotion. Human emotion represents psychological phenomena that encompass cognitions (e.g., feelings, memories, appraisals); visceral, humoral, and immunological reactions; gestures, vocalizations, and expressive displays; postural orientations and overt behaviors; or varying combinations of these (e.g., Fridja, 1986; Izard, 1977; Leventhal & Mosbach, 1983). Emotions have been further characterized as being evoked by biologically relevant stimuli and by associated internal or external events, as mobilizing limited attentional and cognitive resources directed toward present or future coping, and as modulating internalized and externalized actions that foster a generally adaptive coping response (Malmö, 1975; Plutchik, 1980). Emotions involve an explicit evaluative categorization of a stimulus into positive and/or negative valence classes, as well as the activation of behavioral dispositions that entail bivalent tendencies toward (e.g., approach, acquisition or consumption, affection) or away from (e.g., avoidance, escape or rejection, withdrawal, repulsion) the stimulus (Berntson, Boysen, & Cacioppo, in press). These dispositions are manifested in the somatic nervous system and, particularly when intense or extended across time, entail the logistical support of the autonomic nervous system. Although cognitive,

social, and developmental factors influence human emotions (e.g., Izard & Malatesta, 1987; Lazarus, 1966), we focus in this chapter on the psychophysiological responses associated with emotions and their likely role in emotional experience.

The embers of scientific interest in the somatovisceral substrates of the emotions (e.g., Darwin, 1872/1873) were fanned by William James's (1884) influential article, "What Is an Emotion?" James's provocative answer to this question was that emotional feelings are consequences rather than antecedents of peripheral physiological changes brought about by some stimulus. James's theory has stimulated debate and research for more than a century. Research on the influence of cognitive appraisals in emotion (e.g., Smith & Ellsworth, 1987; Valins, 1966) and on emotions in the spinal-cord-injured (e.g., Chwalisz, Diener, & Gallagher, 1988) suggests that afferent information from peripheral activity is not a *necessary* condition for emotional experience. James (1884), however, viewed emotions as being multiply determined. For instance, individuals may recall earlier emotional episodes, including their feelings, and in so doing they may re-experience the emotion. If the remembered emotion was weak originally (e.g., it involved little or no somatovisceral activation), re-experiencing the emotion may occur in the absence of significant peripheral bodily disturbances. James

(1984) therefore stated at the outset that "the only emotions I propose expressly to consider here are those that have a distinct bodily expression" (p. 189). James maintained that within this class of emotional phenomena, discrete emotional experiences can be identified with unique patterns of bodily changes, and that the perception of one of these specific patterns of peripheral physiological changes is the emotional experience (see Cacioppo, Bernston, & Klein, 1992).

Numerous theories of emotion have been proposed since James (1884), but those dealing with emotions accompanied by significant peripheral physiological changes are bracketed by (1) theories holding that discrete emotional experiences stem from distinct somatovisceral patterns (e.g., Ekman, Levenson, & Friesen, 1983; Levenson, 1988; Levenson, Ekman, & Friesen, 1990), and (2) theories holding that discrete emotional experiences derive from cognitive appraisals initiated by the perception of undifferentiated physiological arousal (e.g., Mandler, 1975; Schachter & Singer, 1962). Because of the centrality accorded to autonomic nervous system (ANS) activity in these theories and to the resurgence of research and debate on emotion-specific ANS activity (e.g., Ekman, 1992; Levenson, 1992; Zajonc & McIntosh, 1992), we begin by examining in some detail the literature on the autonomic differentiation of emotions. We then summarize representative studies and issues in electromyographic (EMG) and electrocortical studies of the emotions. We conclude by outlining a heuristic formulation depicting the possible roles of reafference in emotions that are accompanied by significant somatovisceral changes.

AUTONOMIC ACTIVITY AS A FUNCTION OF EMOTION

James (1884, 1890/1950) suggested that emotions are differentiated by somatovisceral responses, but he did not specify what these patterns should be or for what reason particular somatovisceral patterns should be linked to specific emotions. Without theoretical guidance regarding what autonomic responses to measure and why, investigations are reduced to descriptive explorations. Systematic empirical investigations of the autonomic differentiation of the emotions were therefore stimulated

when Arnold (1945) proposed that fear and anger differ autonomically because of their differential involvement of the neurotransmitters epinephrine (fear) and norepinephrine (anger). Wolf and Wolff (1951) subsequently described a patient with a gastric fistula whose stomach could be observed visually. These authors reported that feelings of anxiety were associated with reductions in stomach acidity, blood flow, and motility, whereas feelings of anger were associated with increases in stomach acidity, blood flow, and motility. The epinephrine-norepinephrine hypothesis received additional support when autonomic assessments during realistic manipulations of anger and fear indicated that anger was associated with higher peak diastolic blood pressures (Ax, 1953) and lower heart rate (Schachter, 1957) than fear.¹ Research over the next several decades produced inconsistent results (Funkenstein, King, & Drollette, 1954; see review by Wagner, 1989), and the epinephrine-norepinephrine hypothesis is no longer viewed as plausible.

Conceptual Issues

Most contemporary research on the autonomic differentiation of emotions has been guided by three general hypotheses. The program of research by Ekman, Levenson, and their colleagues has been guided by a variant of James's hypothesis that there is emotion-specific ANS activity:

Emotion provides a mechanism by which behavior, facial expression, and the appropriate ANS support can be quickly matched to the immediate environmental demands. The capacity of the

¹Among the bodily responses following moderate doses of epinephrine are increased heart rate and myocardial contractility (and hence increased cardiac output), vasoconstriction in the cutaneous vascular bed, vasodilation in the vascular beds of the skeletal muscles, and elevated systolic blood pressure. Although moderate doses of norepinephrine have some of the same effects (e.g., vasoconstriction in the cutaneous vascular beds, increased myocardial contractility), vasoconstriction rather than vasodilation is more common in the skeletal muscular vascular beds. Consequently, systemic blood pressure increases and acts to slow heart rate via the baroreceptor reflex.

The data analyzed by Schachter (1957) were a subset of those described by Ax (1953). Nevertheless, the results were not entirely consistent across these investigations. For instance, Schachter (1957) found no differences in diastolic blood pressure between anger and fear.

ANS for supporting a limited number of primary emotional/behavioral pairings is the centerpiece of its evolutionary value in emotion. (Levenson, 1988, p. 40)

The most frequently considered alternative hypotheses have been that (1) discrete emotions are associated with the perception or evocation of increased autonomic activity (e.g., Schachter & Singer, 1962; Mandler, 1975); and (2) the anticipated or realized action requirements of the emotional challenge, rather than emotion per se, determine physiological responses to the stimulus (see, e.g., the discussion by Frijda, 1986; Lang, Bradley, & Cuthbert, 1990). Both of these alternative hypotheses predict that ANS activity is not a function of the emotion per se. For instance, anger is more likely to lead to aggression, and fear to withdrawal. Yet the somatic involvement may be overlapping or equivalent for these emotions. Running may be involved, whether it is to locomote toward a fleeing antagonist to attack in anger, or away from the antagonist in fearful retreat. Measured at the level of the effectors, these emotions may be indistinguishable, even though they differ in terms of goals and outcomes.

Crucial tests among these hypotheses have been hindered by the failure of these hypotheses to make specific empirical predictions and by their not being mutually exclusive. For instance, the important descriptive study by Ekman et al. (1983) renewed interest in the question of emotion-specific ANS activity. Ekman et al. characterized happiness, disgust, and surprise as being associated with lower heart rate in their facial action task than anger, fear, and sadness, and they further discriminated between anger and the latter two emotions in terms of digital skin temperature. However, heart rate and finger temperature did not discriminate between any of these emotions when Ekman et al. (1983) used imagery (i.e., "relived emotion task") to elicit discrete emotions; instead, subsets of the emotions were differentiated by skin resistance level (which did not discriminate between the emotions in the facial action task). Whether or not these data support the hypothesis of emotion-specific ANS activity is unclear, because the mechanism by which discrete emotions are linked to particular ANS changes—or even what set of ANS changes uniquely defines each emotion—has yet to be specified.

The nonexclusivity of the hypotheses derives from research demonstrating that changes in autonomic activity (e.g., heart rate) are influenced by nonemotional factors such as individual differences (e.g., see Cacioppo, Uchino, et al., 1989; Stemmler, 1992) and by anticipated or actual somatic activity (Obrist, Webb, Sutterer, & Howard, 1970), respiration (see Bernston, Cacioppo, & Quigley, 1993), and attention (e.g., orienting; Graham & Clifton, 1966; Lacey, Kagan, Lacey, & Moss, 1963). The influence of specific emotions on ANS activity, therefore, may be difficult to discern, particularly if this influence is weak (Levenson, 1988) or if its effects on the functional output of the ANS are not strictly additive (see Bernston, Cacioppo, & Quigley, 1991).

Finally, whether the intensity and duration of an emotion moderate somatovisceral patterning is a question that warrants more careful investigation (cf. Roberts & Weerts, 1982). For instance, the thresholds and dynamic ranges of somatovisceral responses can vary across effectors (e.g., eccrine sweat glands vs. the heart), making it possible for the same emotion to be associated with additional somatovisceral changes as the intensity of the emotion increases. Furthermore, the somatovisceral and subjective responses to motivational challenges, such as food deprivation or declines in body temperature, are influenced by the magnitude and duration of the challenge. Thus, it may not be sufficient to equate emotions for signatures if the manifestation of these signatures varies as a function of intensity.

Methodological Issues

In an effort to isolate emotion-specific ANS changes, greater attention over the past decade has been paid to methodological issues (e.g., see Ekman, et al., 1983; Davidson, Ekman, Saron, Senulis, & Friesen, 1990). The following methodological issues are important to consider and are equally applicable to autonomic, somatic, or electrocortical studies of emotion (Davidson et al., 1990):

1. At least two emotions should be compared. Furthermore, because the changes in ANS activity that accompany specific emotions are of interest, it is often important to include baseline measures, nonemotional comparison conditions, and/or manipulations of emotional intensity.

2. Epochs of discrete emotions must be separable. More than one emotion may be elicited by a stimulus, either simultaneously or in close temporal proximity, and this can hinder identification of the physiological substrates of discrete emotions.

3. The epochs of the emotions should be comparable in length and concordant with the time constants on the dependent measures. Ekman (1984) has suggested that most episodes of emotions last less than 4 seconds. Although this view has been disputed (e.g., Fridja, Mesquita, Sonnemans, & Van Goozen, 1991), there is a clear need to ensure that the measurement interval allows the confluence of physiological responses.

4. Independent evidence should be provided that the intended emotion was produced during the epoch.³ Only if evidence other than the physiological measures of interest is provided can much confidence be placed in any interpretation of the resulting physiological outcomes.

5. Independent evidence should be provided that unintended emotions or confounding variations in motoric and cognitive activity were not present during this epoch.

6. Independent evidence should be provided that the intensity of the elicited emotion was matched across the epochs that were compared. Emotional intensity has long been recognized as a determinant of somatovisceral responses, and differences across emotions in their intensity can mask or masquerade as bona fide physiological differences.

7. The collection of the independent evidence and the physiological measures should be appropriately synchronized to the elicited emotion.

8. The epochs should be of sufficient duration, or the number of epochs within subjects over which aggregation occurs should be sufficient, to produce measures with satisfactory psychometric properties. Developments in psychophysiological instrumentation now make it possible to collect physiological measure-

³A key limitation in the study of emotion is the absence of a generally accepted index of the emotions. Self-reports of emotional experience are sometimes used, but emotions may be more subtle and may vacillate or pass more quickly than self-reports can typically detect. Self-reports are also subject to distortions resulting from social incentives and self-presentational concerns. Prototypical facial expressions of emotions have also been used, but these too can be insensitive. Converging operations, therefore, are often best.

ments continuously, with consequent improvements in their reliabilities. The benefit of continuous measurements derives from their aggregation within or across epochs. The low reliabilities of physiological measures based on one-shot assessments (e.g., postinduction blood pressure readings; the maximum increase, decrease, or change in a physiological variable) can contribute to measurement variance.

9. Appropriate statistical procedures should be used to examine emotion-specific configurations among the dependent measures, and to protect against spurious findings resulting from multiple dependent measures and comparisons. Procedures such as the Bonferroni correction to protect against Type I errors can be overly conservative when many physiological variables are recorded. In such studies, it may be preferable to include an internal replication (e.g., cross-validation sample) to ensure the reproducibility of significant effects (Cacioppo, Bernsten, & Andersen, 1991).

10. Multiple operationalizations (e.g., eliciting tasks) should be used across, if not within, studies to ensure that results are emotion-specific rather than task-specific.

Table 9.1 lists, in chronological order, published research that has contrasted the effects of at least two discrete emotions on two or more autonomic measures in humans. Included in the far right column is our best determination of which of the preceding 10 methodological desiderata were incorporated.⁴ As can be seen in Table 9.1, the emotions of happiness, sadness, anger, fear, disgust, and surprise have been investigated. Note, too, that only the studies by Ekman, Levenson, and their colleagues have compared the effects of more than four emotions on multiple physiological measures. Considerable variation also exists in the procedures used to elicit specific emotions and in the recording epochs used (see Table 9.1). Finally, and as would be expected, methodological and statistical procedures tend to be better in recent than in early studies.

⁴Hubert and de Jong-Meyer (1991) studied the emotions of suspense and happiness, each evoked by a film clip. Because it is unclear which emotion to equate with suspense, this study was not included. It should be noted, however, that the inclusion of this study (classifying suspense as fear) increases neither the appearance of consistency in physiological results nor the evidence for the autonomic differentiation of emotions.

THE PSYCHOPHYSIOLOGY OF EMOTION

TABLE 9.1. Studies Comparing Two or More Physiological Measures as a Function of Two or More Emotions

Study ¹	Manipulations	Dependent variables	Method Features ²
1. Ax, 1953 43 Ss (6 were not affected by one or more of the manipulations and thus excluded from the analyses)	Real-life induction: Fear—shout circuit in SCL apparatus/intermittent shock Anger—abusive polygraph operator	Manipulation period (7-minute epoch)—5 during manipulation and 2 after) minus baseline in max rises and falls of SBP, DBP, SV, HR, FCT, FT, SCL, EMC, RESP Emotion presence judged by experimenter from interview	1, 3, 4
2. Funkenstein, King, & Drollette, 1954 69 Ss (53 used in analyses)	"Problems situation" manipulation; emotions categorized post hoc as: Anger: In (21 Ss), Anger Out (22 Ss), Fear (Anxiety; 9 Ss)	Single measurement at end of problem situation minus baseline of HR (pulse), SBP, DBP, SV, CO, PR Emotion presence and direction judged by raters from taped interview	1, 3, 4, 7, 8
3. Schachter, 1957 48 Ss in three groups: hypertensives, potential hypertensives, and normotensives (the same 15 normotensives as in Ax, 1953)	Real-life manipulation: Fear, Anger—same as Ax, 1953 Pain—cold pressor	Max change during manipulation minus baseline in HR, SBP, DBP, FCT, FT, SCL, EMC, RESP, SV, PR Emotion presence and intensity assessed from judges' ratings of taped verbal, facial, and behavioral responses during manipulations, experimenter notes, and interview after manipulations	1, 3, 4
4. Averill, 1969 54 Ss (males only)	Film manipulation: Sad—Kennedy's assassination Happy (Mirth)—an adaptation of a silent comedy Control—ichthyologists Each preceded by neutral baseline film	Stimulus period (6-minute epoch) minus baseline of max rise, mean, and fall in HR, SBP, DBP, FCT, FT, SCL, FPV, NNSCRs (mean only), RESP (max increase, rate, and irregularity)—mean only Vocalization Emotional intensity	1, 3, 4, 9
5. Tourangeau & Ellsworth, 1979 Film effects ^a 128 Ss	Film manipulation: Fear—industrial accidents Sad—boy in orphanage Neutral—flower show Crossed with directed facial action (DFA): Fear, Sad, Neutral, Undirected	Stimulus (2-minute epoch) minus baseline of HR (max rise and fall), SRL (max fall), NNSCRs Emotional intensity Face expression	1, 3, 4, 5, 7
6. Schwartz, Weinberger, & Singer, 1981 ^b 32 Ss (with acting experience)	Imagery manipulation ^a of: Happy, Sad, Anger, Fear, Relax, Control	Single measurement following imagery minus baseline of HR, SBP, DBP Emotional intensity self-reported by subject and estimated by experimenter	1, 2, 3, 4, 9
7. Roberts & Weerts, 1982 16 Ss chosen from 35 ^b	Imagery manipulation of low and high intensity of: Anger, Fear, and two low-intensity Neutral	Single measurement during imagery minus baseline of HR, SBP, DBP Emotional intensity	1, 2, 3, 4, 5, 6, 7, 9
8. Ekman, Levenson, & Friesen, 1983 8a. Best faces ^{a,10} 8b. Best imagery ^{a,10} 16 Ss (actors and scientists who study the faces)	DFA and Imagery manipulations of: Fear, Anger, Happy, Sad, Surprise, Disgust	DFA (10-second epoch) or Imagery (30-second epoch) minus baseline of HR, FT (right and left), SRL, EMC Emotional intensity Facial expression	1, 2, 3, 4, 5, 7, 6, 9, 10
9. Stemmler, 1989 9a. Real life 9b. Imagery 42 Ss (females only)	Real-life manipulations of: Fear—scary radio play and music, unexpected darkness Anger—abuse during argument, induced loud speaking	Standardized reactivity scores ¹¹ of HR, FT, SCL, EMC, MVT (finger and hand acceleration), RESP, FTT, FBV, BV Emotional intensity	1, 3, 4, 9, 10

(continued)

TABLE 9.1. (Continued)

Study ^a	Manipulations	Dependent variables	Method features ^b
10. Tassinari, Cacioppo, & Green, 1989 15 Ss (females only)	Happy—nice experimenter, extra monetary bonus, shorter experiment Imagery manipulation also DFA manipulation of: Anger, Happy, Control	Stimulus (4-second epoch) minus baseline of SCL, HR Emotional intensity DFA (10-second epoch) minus baseline of HR, FT, SCL, EMG/MVT ^c	1, 2, 4, 5, 9
11. Levenson, Ekman, & Friesen, 1980 11a. All faces 11b. Best faces	DFA manipulation ^d of: Fear, Anger, Happy, Sad, Surprise, Disgust	Emotional intensity Facial expression Task difficulty (in Experiment 2)	1, 2, 3, 4, 5, 7, 8, 9
Reports combined results from three experiments: 1. Ekman et al., 1983 2. 16 Ss chosen from 100 ^e 3. 30 Ss chosen from 109			
12. Levenson, Carstensen, Friesen, & Ekman, 1991 ^h Best faces/best imagery 20 Ss chosen from 35 (older than 70; 4 on beta blockers)	DFA and imagery manipulation of: Fear, Anger, Happy, Sad, Surprise, Disgust	DFA (10-second epoch) or imagery (15-second epoch)—began when Ss indicated that they felt the emotion) minus baseline of HR, FT, SCL, MVT Emotional intensity Task difficulty	1, 2, 3, 4, 5, 7, 8, 9, 10
13. Levenson, Ekman, Heider, & Friesen, 1992 Best faces 46 Ss (males only, from Minnanghabau)	DFA and imagery manipulation of: Fear, Anger, Happy, Sad, Disgust	DFA (10-second epoch) minus baseline of HR, FT, SCL, PTT, FPV, RESP (rate and depth) Emotional intensity Task difficulty Facial expression	1, 2, 3, 8, 9

Note. Variable abbreviations: HR, heart rate; SCL, skin conductance level; SBL, skin conductance level; NNSCLR, number of nonspecific skin conductance responses; FT, finger temperature; PCT, foot temperature; EMG, muscle activity; MVT, movement; SBF, systolic blood pressure; DBP, diastolic blood pressure; SV, stroke volume; CO, cardiac output; FPV, finger pulse volume; RESP, respiration; PTT, pulse transit time; BV, blood volume.
^aBecause this table includes only studies that compared physiological reactions as a function of two or more emotions, we have not included Starbuck (1983). In this study he did measure physiological responses (SCL, HR, SV, CO, PTT, RESP, HR, systolic rate, and FPV) of children as a function of a number of emotions—sad, fearful, happy (real) and angry (imagery)—elicited by a film stimulus (Bambi), however, he compared the physiological responses of the emotions to the prestimulus level and did not perform comparisons between emotions. The only differences reported a significant increase in heart rate and skin conductance level during the sad period, in which he found an increase in skin resistance (a decrease in SCL) and a decrease in systolic and diastolic blood pressure. The only differences in facial expressions were found for differences in decrease of consistency in direction of automatic responses rather "suppression" (p. 90).
^bThe numbers correspond to the methodological features discussed in the text, representing our best judgment of those features that were processed by the corresponding study.
^cStimulus was verbal description/provocation from the experimenter while the subject repeated list of 6-10 digits in reverse order and solved word problems. Subjects' responses were also categorized as follows: (a) Anger; (b) No Emotion; (c) Expect Anger and Anxiety; and (d) Miscellaneous. These categories were not included in the statistical analyses because the frequency in each was judged to be too low.
^dImagery irregularity—(a) inspiration that was twice the average of the adjacent inspirations; (b) inspiration interrupted by inspiration; (c) inspiration departing below the baseline of surrounding breaths.
^eNo significant effects were found for differences in facial expressions during the film. Thus the differences reported in Tables 9.2 and 9.3 are different due to the emotional effects of the film.
^fSubjects also exercised and measures were taken following exercise, but those analyses are not included in this table, as they are beyond the scope of this chapter.
^gSubjects imagined a scene in which they felt the appropriate emotion as they were (in their imagination) exercising on a one-step inventory and scored near the mean on other tests than interviewed to certify that they could produce the appropriate emotional imagery. The 10 final subjects were those who went to the interview and could perform the emotional imagery task.
^hBest faces' denotes the subset of trials during which subjects' facial expressions were rated as being close to prototypic emotion faces.
ⁱThe term imagery denotes the subset of trials during which subjects reported feeling the target emotion most strongly.
^jFacial activity scores were derived by (a) obtaining difference scores of the raw scores and the current baseline (derived through linear interpolation subtracting scores to a McCall normalizing transformation). These scores were then standardized.
^kSubjects were processed for ability to exhibit good voluntary facial muscle control.
^lThe report combined 1983 frowns/faces EMG data and 1980 movement (of the subject's chair) sensor data. The subset of the authors was to measure neutral muscle activity.
^mMeasure of PTT (hr, and s finger), FFA, and REST (rate) were also collected, but were not reported.
ⁿBest faces constituted 46% and best imagery constituted 80% of the corresponding trials.

Empirical Outcomes

The results of the investigations identified in Table 9.1 are summarized in Tables 9.2 and 9.3. Given the variation across studies, one might expect to find only moderate consistency in their results. However, a few recent studies, and most notably those by Levenson, Ekman, and their colleagues, have used a common set of procedures and measures, making it possible to examine the reproducibility of emotion-specific ANS activity across studies (albeit within one laboratory). Where reproducible effects are found, questions of generalizability can be considered by looking across methods of eliciting the emotion and across laboratories. Table 9.2 follows the format introduced by Zajonc and McIntosh (1992). Differences among emotions on each dependent measure are indicated as in contrast tests. As Zajonc and McIntosh (1992) have noted, perfect distinctiveness among the emotions on a particular measure would result in a different letter within a row, and all entries within a column would be the same (indicating the replicability and generalizability of the result). It is possible that two or more emotions do not differ on a particular measure, but that they do differ in the sets of physiological responses they evoke. Therefore, Table 9.3 summarizes comparisons of the sets of physiological differences between various pairs of emotions.

Several conclusions can be drawn from the cumulative literature on the autonomic differentiation of emotion. First, imagery is not an emotional elicitation procedure that has produced reliably differentiated ANS activity, even though subjects have reported differential emotional experiences (Stemmler, 1989; Zajonc & McIntosh, 1992). It is unclear why this would be the case, unless ANS activity is responsive to the metabolic requirements of the anticipated or realized response to the emotional challenge—a requirement that would tend to be uniformly low in emotional imagery.

Second, there is little evidence for replicable autonomic differences in pairwise comparisons of the emotions on the measures of bodily tension, systolic blood pressure, facial temperature, respiration, skin conductance level, and cardiac stroke volume. For instance, Ekman et al. (1983) reported that skin resistance level decreased (i.e., skin conductance increased) more during sadness than during fear, anger, and disgust. This differentiation was found only when imagery was used to elicit the emo-

tions, however, and this particular pattern has not been replicated. Too few data exist on several other measures (e.g., skin conductance responses, peripheral resistance, cardiac output, finger pulse volume, pulse transit time, body movement) to permit us to draw strong conclusions, leaving only heart rate, diastolic blood pressure, and finger temperature to perse more closely.

Of 13 pairwise comparisons between discrete emotions, 6 were significant when diastolic blood pressure served as the dependent measure. The strongest result was the tendency for anger to be characterized by high diastolic blood pressure (see Table 9.3), but 4 of 7 comparisons failed to find this effect. The results for finger temperature also provide only tentative evidence for emotion-specific ANS activity. For instance, Table 9.2 summarizes 95 pairwise comparisons involving finger temperature, only 14 (15%) of which were significant. Of these significant comparisons, the most reliable differentiation was found between anger and fear, with anger associated with higher finger temperature than fear in 4 of 11 comparisons (36.4%).

Heart rate has been the best discriminator of the emotions, but it too is far from discriminating consistently or fully among the emotions. Unusually robust findings are evident in the differentiation by heart rate of sadness from disgust (significant in 5 of 6 or 83.3% of the comparisons), anger from disgust (83.3%), and fear from disgust (66.7%). Table 9.2 depicts modest replication rates in the differentiation by heart rate of happiness from anger (5 of 10 comparisons, or 50%), were significant), happiness from fear (44.4%), sadness from surprise (60%), and fear from surprise (60%). The remaining pairwise comparisons show little evidence of reliable differentiation by heart rate.

As modest as most of these replication rates are, there are three reasons to be concerned that these are overestimates. First, the data analyzed by Schachter (1957) were from a subset of the subjects in Ax (1953), and data from Ekman et al.'s (1983) facial action test were part of the data set analyzed by Levenson et al. (1990). Furthermore, studies occasionally involved more physiological measures than those for which results were reported, and double-blind procedures are atypical in this area of research.

Second, the elicitation procedure producing the greatest differentiation of emotions appears

TABLE 9.2. Pairwise Comparisons of Physiological Responses as a Function of Emotion

Study	Heart rate									
	Hap	Sad	Ang	Fear	Surp	Disg	Pain	Rck	Cntl	
Ax, 1953										
Max rise	A	A	A	A	A					
Max fall	A	B	B	B	B					
Funkenstein et al., 1954	AB	B	B	B	B					
Schachter, 1957 ¹										
Max change	B	A	A	A	A					C
Averill, 1969										
Mean	A	A	A	A	A					A
Max rise	A	B	B	B	B					AB
Max fall	A	A	A	A	A					A
Tourangeau & Ellsworth, 1979										
Max rise	B	AB	A	A	A					B
Max fall	B	AB	A	A	A					A
Schwartz et al., 1981										
Roberts & Weerts, 1982										C
High intensity										
Low intensity										
Ekman et al., 1983										
Best faces	B	A	A	A	A	B	B			
Best imagery	A	A	A	A	A	A	A			
Stemmler, 1989										
Real life	A	A	A	A	A					
Imagery	A	A	A	A	A					
Tassinari et al., 1989										
Levenson et al., 1990										B
All faces	BC	AB	A	A	D	CD				
Best faces	B	A	A	A	B	B				
Levenson et al., 1991										
Best faces/best imagery	AB	A	A	AB	AB	B				
Levenson et al., 1992	BC	AB	A	AB	AB	C				

Study	Finger temperature									
	Hap	Sad	Ang	Fear	Surp	Disg	Pain	Rck	Cntl	
Ax, 1953										
Max rise	A	A	A	A	A					
Max fall	A	A	A	A	A					
Schachter, 1957 ¹										
Max change	A	A	A	B	B					C
Averill, 1969										
Ekman et al., 1983										
Best faces ^a	B	B	A	A	B	B	B			
Best imagery	A	A	A	A	A	A	A			
Stemmler, 1989										
Real life	A	AB	B	B	B					
Imagery	A	A	A	A	A					
Levenson et al., 1990										
All faces	AB	AB	A	B	AB	AB				A
Best faces	B	B	A	B	B	B				
Levenson et al., 1991 ²										
Best imagery/best faces	AB	B	AB	AB	AB	A				
Levenson et al., 1992										
Best faces	A	A	A	A	A	A				

(continued)

TABLE 9.2. (Continued)

Study	Skin conductance level									
	Hap	Sad	Ang	Fear	Surp	Disg	Pain	Rck	Cntl	
Ax, 1953										
Max rise	B	A	A	A	A					
Max fall	A	A	A	A	A					
Schachter, 1957 ^{1,4}										
Max change	B	A	A	A	A					C
Averill, 1969										
Tourangeau & Ellsworth, 1979										
Max rise ^a	A	A	A	A	A					B
Ekman et al., 1983										
Best faces	A	A	A	A	A	A	A			
Best imagery	AB	A	B	B	AB	B				
Stemmler, 1989										
Real life	A	A	A	A	A					
Imagery	A	A	A	A	A					
Tassinari et al., 1989										
Levenson et al., 1990										
All faces	B	A	AB	A	B	A	A			
Best faces	B	AB	AB	A	B	A	A			
Levenson et al., 1991										
Best faces/best imagery	A	A	A	A	A	A	A			
Levenson et al., 1992										
Best faces	A	A	A	A	A	A	A			

Study	EMG									
	Hap	Sad	Ang	Fear	Surp	Disg	Pain	Rck	Cntl	
Ax, 1953 (frontalis)										
Number of peaks	B	A	A	A	A					
Max rise	A	A	A	A	A					
Max fall	A	A	A	A	A					
Schachter, 1957 ¹ (frontalis)										
Ekman et al., 1983										
Best faces	A	A	A	A	A	A	A			
Best imagery	A	A	A	A	A	A	A			
Stemmler, 1989										
Extensor digitorum										
Real life	B	A	A	B	B					B
Imagery	A	A	A	A	A	A	A			A
Trapezius										
Real life	A	A	A	A	A	A	A			A
Imagery	A	A	A	A	A	A	A			A

Study	Face temperature									
	Hap	Sad	Ang	Fear	Surp	Disg	Pain	Rck	Cntl	
Ax, 1953										
Max rise	A	A	A	A	A					
Max fall	A	A	A	A	A					
Schachter, 1957 ¹										
Max change	A	A	A	A	A					A
Averill, 1969										
Stemmler, 1989										
Real life	B	A	A	C	A					
Imagery	A	A	A	A	A					

(continued)

TABLE 9.2. (Continued)

Study	Systolic blood pressure									
	Hap	Sad	Ang	Fear	Surp	Disg	Pain	Relx	Cntl	
Ax, 1953										
Max rise	A	A	A	A	A					
Max fall	A	A	A	A	A					
Funkenstein et al., 1954	A ^a	B								
Schachter, 1957 ¹										
Max change	A	A	A	A	A					
Averill, 1969	B	A	A	A	A					
Schwartz et al., 1981	A	A	A	A	A					B
Roberts & Weerts, 1982	A	A	A	A	A					B
High intensity	A	A	A	A	A					
Low intensity	A	A	A	A	A					

Study	Diastolic blood pressure									
	Hap	Sad	Ang	Fear	Surp	Disg	Pain	Relx	Cntl	
Ax, 1953										
Max rise	A	B								
Max fall	A	A								
Funkenstein et al., 1954	A	A								
Schachter, 1957 ¹										
Max change	A	A								
Averill, 1969	B	A								
Schwartz et al., 1981	B	BC	A	B						B
Roberts & Weerts, 1982	A	B								BC
High intensity	A	B								
Low intensity	A	A								

Study	Respiration									
	Hap	Sad	Ang	Fear	Surp	Disg	Pain	Relx	Cntl	
Ax, 1953										
Max rise	B	A								
Inspiration	A	A								
Respiration amplitude	A	A								
Schachter, 1957 ¹										
Max change	B	A								
Rate	A	A								
Inspiration index ⁷	A	A								
Averill, 1969	A	A								
Mean	A	A								A
Max rise	A	AB								B
Irregular respiration	A	B								B
Levenson et al., 1992										
Best faces	B	AB	AB	A						A
Rate	A	AB	AB	AB						B
Depth	A	AB	AB	AB						

Study	Stroke volume									
	Hap	Sad	Ang	Fear	Surp	Disg	Pain	Relx	Cntl	
Ax, 1953										
Max rise	A	A								
Max fall	A	A								

(continued)

TABLE 9.2. (Continued)

Study	Stroke volume									
	Hap	Sad	Ang	Fear	Surp	Disg	Pain	Relx	Cntl	
Funkenstein et al., 1954										
Max rise	B ^a	A								
Schachter, 1957 ¹										
Max change	B	A								C

Study	Cardiac output									
	Hap	Sad	Ang	Fear	Surp	Disg	Pain	Relx	Cntl	
Funkenstein et al., 1954										
Schachter, 1957 ¹										
Max change	B ^a	A								
	B	A								C

Study	Finger pulse volume									
	Hap	Sad	Ang	Fear	Surp	Disg	Pain	Relx	Cntl	
Averill, 1969										
Mean	A	A								A
Max rise	AB	A								B
Max fall	A	A								A
Stemmler, 1989										
Real life	AB	A								B
Imagery	A	A								A
Levenson et al., 1992	AB	A								B
	A	B								A

Study	NNSCRTs									
	Hap	Sad	Ang	Fear	Surp	Disg	Pain	Relx	Cntl	
Ax, 1953										
Averill, 1969	A	A								B
Tourangeau & Ellsworth, 1979	A	A								A

Study	Blood volume									
	Hap	Sad	Ang	Fear	Surp	Disg	Pain	Relx	Cntl	
Stemmler, 1989										
Finger	A	A								A
Real life	A	A								A
Imagery	A	A								A
Head	A	A								A
Real life	A	A								A
Imagery	A	A								A

Study	Pulse transit time									
	Hap	Sad	Ang	Fear	Surp	Disg	Pain	Relx	Cntl	
Stemmler, 1989										
Real life	A	A								A
Imagery	A	A								A
Levenson et al., 1992	A	B								A
	AB	AB								A

Study	Body movement									
	Hap	Sad	Ang	Fear	Surp	Disg	Pain	Relx	Cntl	
Stemmler, 1989										
Finger	A	A								A
Real life	A	A								A
Imagery	A	A								A
Levenson et al., 1992	A	B								A
	AB	AB								A

(continued)

TABLE 9.3. (Continued)

	Happiness	Sadness	Anger	Fear	Surprise
Surprise	HR < 11a - 8ab, 11b, 12 - 8b, 12 FT < 8a, 11b, 12 - 8b, 11ab, 12 SCL < 11a EMG < 11a, 11b, 12 MVT < 11ab, 12	HR < 8a, 11ab - 8b, 12 FT < 8a, 11b, 12 - 8b, 11ab, 12 SCL < 11a EMG < 8a, 11b, 12 MVT < 11ab, 12	HR < 8a, 11ab - 8b, 12 FT < 8a, 11b, 12 - 8b, 11ab, 12 SCL < 11a EMG < 8a, 11b, 12 MVT < 11ab, 12	HR < 8a, 11ab, 12 - 8b, 12 FT < 8a, 11b, 12 - 8b, 11ab, 12 SCL < 11a EMG < 8a, 11b, 12 MVT < 11ab, 12	HR < 8a, 11ab, 12 - 8b, 12 FT < 8a, 11b, 12 - 8b, 11ab, 12 SCL < 11a EMG < 8a, 11b, 12 MVT < 11ab, 12
Disgust	HR < 8a, 11ab, 12, 13 - 8b, 12 FT < 8a, 11ab, 12, 13 - 8b, 12 SCL < 8a, 11ab, 12, 13 - 8b, 12 EMG < 8a, 11ab, 12, 13 MVT < 8a, 11ab, 12, 13 FPV < 13 PTT < 13 RESP > 13 ^a	HR < 8a, 11ab, 12, 13 - 8b, 12 FT < 8a, 11ab, 12, 13 - 8b, 12 SCL < 8a, 11ab, 12, 13 - 8b, 12 EMG < 8a, 11ab, 12, 13 MVT < 8a, 11ab, 12, 13 FPV < 13 PTT < 13 RESP > 13 ^a	HR < 8a, 11ab, 12, 13 - 8b, 12 FT < 8a, 11ab, 12, 13 - 8b, 12 SCL < 8a, 11ab, 12, 13 - 8b, 12 EMG < 8a, 11ab, 12, 13 MVT < 8a, 11ab, 12, 13 FPV < 13 PTT < 13 RESP > 13 ^a	HR < 8a, 11ab, 12, 13 - 8b, 12 FT < 8a, 11ab, 12, 13 - 8b, 12 SCL < 8a, 11ab, 12, 13 - 8b, 12 EMG < 8a, 11ab, 12, 13 MVT < 8a, 11ab, 12, 13 FPV < 13 PTT < 13 RESP > 13 ^a	HR < 8a, 11ab, 12, 13 - 8b, 12 FT < 8a, 11ab, 12, 13 - 8b, 12 SCL < 8a, 11ab, 12, 13 - 8b, 12 EMG < 8a, 11ab, 12, 13 MVT < 8a, 11ab, 12, 13 FPV < 13 PTT < 13 RESP > 13 ^a

Note: Each sign represents the direction of the relationship comparing the emotion on the row to the emotion on the column. The numbers indicate the study (see Table 9.1) that reported the finding. See Table 9.1 for an explanation of the abbreviations.

^aMax rise.
^bMax fall and mean.
^cIrregular respiration was different, mean regular respiration and max rise were the same.
^dEstimate digitum.
^eHigh acceleration in the real-life condition.
^fHigh acceleration for both the real-life and imagery conditions, and head axial.
^gMax fall and mean.
^hMax fall.
ⁱMax rise.
^jMax fall was different, max rise was not different.
^kMax fall.
^lMax rise.
^mMax fall was different, max rise was not different.
ⁿMax fall.
^oMax rise.

to determine whether or which physiological responses during an emotion (or directed facial action) differed from baseline (or non-emotional comparison) conditions. This information may be important in determining the mechanism underlying the autonomic differences that are observed. Levenson, Ekman, Heider, and Friesen (1992), who did report sufficient information to examine this issue, found that heart rate was higher in Minangkabau subjects during directed facial expressions of fear, sadness, and anger than during the directed facial expression of disgust, but the mean change score for disgust was not greater than zero. This result raises interesting questions about the reasons for heart rate's not changing during the expression of disgust.

Given these considerations, comparisons that demonstrated the absence of any differentiation may be especially informative about avenues that do not warrant further research. For instance, heart rate failed to differentiate happiness from surprise in 4 of 5 comparisons, happiness from disgust in 5 of 6

comparisons, sadness from anger in 7 of 7 comparisons, sadness from fear in 8 of 9 comparisons, anger from fear in 13 of 15 comparisons, and surprise from disgust in 5 of 5 comparisons.

Summary

The research on the autonomic differentiation of emotions is provocative, but the cumulative evidence for emotion-specific autonomic patterns remains inconclusive (Wagner, 1989). Of course, all of the potential elements and patterns of autonomic activity have yet to be examined. More importantly, potential patterns may not be describable by gross measures of end-organ response (e.g., heart rate). A major obstacle in identifying autonomic patterning as a function of emotion, particularly for dually and antagonistically innervated organs such as the heart, is that of the many-to-one mappings that may obtain between underlying neural changes and organ response. Emotional stimuli do not invariably evoke reciprocal activation of the sympathetic and parasympathetic branches of the

ANS. For instance, the presentation of an aversive conditioned stimulus can produce coactivation of the sympathetic and parasympathetic nervous system, with the consequent heart rate response being acceleratory, deceleratory, or unchanged from prestimulus levels, depending upon which activation input was greater (see Berntson et al., 1991). Berntson et al. (1991) recently proposed a theory of autonomic control and modes of autonomic activation that resolves the loss of fidelity in the translation between changes in sympathetic and parasympathetic activation and organ responses. It is possible that emotions (e.g., disgust), or components of emotions (e.g., attention), could be differentiated if the focus were on indices of the sympathetic and the parasympathetic innervation of the viscera, rather than on visceral responses per se. For instance, Quigley and Berntson (1990) found that the deceleratory heart rate response to a low-intensity nonsignal ("orienting") stimulus was small because both parasympathetic and sympathetic activity increased. The acceleratory heart rate response to a high-intensity nonsignal ("defense") stimulus, on the other hand, was larger—not because sympathetic activation was greater than that shown to the low-intensity stimulus, but because parasympathetic activity was unchanged or decreased slightly in response to the high-intensity stimulus.

Whether or not the conditions for and the elements of emotion-specific autonomic patterns of activity can be identified, what does seem clear from this research is that discrete emotional percepts can occur even when the autonomic changes do not fully discriminate the emotions that are experienced. Evidence consistent with this conclusion was provided by Ekman et al. (1983) in the imagery task, where skin resistance level provided only gross distinctions among groups of emotions, despite a careful parsing of epochs of discrete emotional experiences. Recent research on the emotional percepts of patients with lesions at differing points along their spinal cords further suggest that quite different patterns of autonomic afference can be associated with the same emotional experience (e.g., Bermond, Nieuwenhuysse, Fasotti, & Schuurman, 1991; Chwalisz et al., 1988).³

³The different "patterns" of afference derive from differences in the sites of the lesions. Lower lesions allow afference from more viscera than higher lesions. It should

If discrete emotional percepts can occur even when the autonomic changes do not fully discriminate the emotions that are experienced, does it necessarily follow that somatovisceral afference plays no role in defining these discrete emotional percepts? Cannon's (1927) answer to this question was yes; in his view, autonomic events are too slow, too insensitive, and too undifferentiated to contribute to emotions.⁴ Schachter and Singer (1962) revolutionized thinking about emotions when they suggested that undifferentiated autonomic activity can subserve discrete emotions. The mechanism by which this was accomplished, according to Schachter and Singer (1962; Schachter, 1964; see also Mandler, 1975; Reizenstein, 1983), is the arousal of an "evaluative need," which motivates the individual to understand and label cognitively his or her bodily feelings. The consequent attributional processes were thought to produce specific emotional states and influence emotional behavior. There is yet another distinct way in which peripheral bodily reactions may contrib-

ute to the emotion. Research demonstrating that the emotions experienced by individuals with spinal cord lesions are similar to those experienced by normal individuals does not logically imply that afference plays no role in the generation or shaping of emotional experience. If we let E denote a discrete emotion and we let Φ denote autonomic afference, we can represent the finding that individuals with spinal cord lesions can experience discrete emotions as the conditional probability $P(E/\text{not } \Phi) > 0$. To infer from this result that $P(E/\text{not } \Phi) - P(E/\Phi)$ is speculative, however, because (1) emotions are most likely multiply determined, in which case physiological afference may not be a necessary determinant, while still being a sufficient or contributory determinant; (2) the situations and challenges that individuals with and without spinal cord injuries encounter may differ, and (3) the perspective used by individuals with spinal cord injuries when expressing their feelings in words or along rating scales may change as they adapt to their injuries (e.g., what is meant by the label "intense arousal" or "intense anger" may change).

There is now considerable evidence that is inconsistent with the notion that autonomic activity increases in a general and diffuse manner during emotion. In addition to the data summarized in Tables 9.2 and 9.3, reliable individual and situational stereotypes have been documented (e.g., see reviews by Cecchiopoli, Uchino, et al., 1992; Lang et al., 1980). Moreover, neural changes within the sympathetic nervous system can be highly fractionated (see Johnson & Andersson, 1980), and different patterns of sympathetic and parasympathetic activity can underlie similar, appearing autonomic responses (Berntson et al., 1991). Hence, by an "undifferentiated" pattern of autonomic response, we mean only that the autonomic responses (from which interoceptive feedback is derived) do not differentiate specific emotions such as fear and anger.

ute to emotional experience—an active perceptual process by which an ambiguous pattern of somatosceral afference is disambiguated to produce an immediate, spontaneous, and indubitable emotional percept (Cacioppo, Bernston, & Klein, 1992). We briefly discuss this perceptual-emotional process in the final section, but first we review illustrative studies of somatic and electrocortical activity as a function of emotion.

FACIAL ELECTROMYOGRAPHIC AND ELECTROCORTICAL ACTIVITY AS A FUNCTION OF EMOTION

Facial Actions

Contemporary developments in facial expression as a marker of emotion can be traced to Tomkins's (1962) ascription of an instrumental role to facial movement and feedback in the experience of emotion, and to his suggestion that high-speed filming be used to perform microscopic analyses of facial expressions and emotion. These proposals led to important methodological advances in facial coding (e.g., Ekman & Friesen, 1978; Izard, 1971, 1977). Based on research that identified a small set of emotions accompanied by unique configurations of facial actions and labeled reliably across cultures, Ekman (e.g., 1973, 1992b) and Izard (e.g., 1977, 1992) have proposed that there is a small number of *basic* emotions. These basic emotions are hypothesized to be associated with distinctive innate response patterns and neural substrates. Happiness (or joy), sadness, anger, fear, and disgust are generally considered to be basic emotions, but a different five emotions (Fischer, Shaver, & Carnochan, 1990), fewer than five emotions (e.g., Panksepp, 1982), and more than five emotions (e.g., Frijda, 1986; Oatley & Johnson-Laird, 1987) have also been suggested. Recently, Ortony and Turner (1990) have challenged the very premise that there are basic emotions, and they have proposed instead that emotions are constructed from valent and nonemotional component processes. This is an important and continuing debate that will probably have implications for which emotions might be expected to have distinctive psychophysiological substrates (see Ekman, 1992a, 1992b; Izard, 1992; Panksepp, 1992; Turner & Ortony, 1992). That there are a small number

of emotions with panculturally identifiable facial expressions is less controversial, however (e.g., see Ekman, 1989; Izard, 1977).

Not all emotional and affect-laden information processes are accompanied by visually perceptible facial actions, of course, and this has limited the utility of analyses of facial actions in emotions. Approximately 15 years ago, facial EMG began to be used to investigate emotions. As Rinn (1984) noted, overt facial expressions are the result of varied and specific movements of the facial skin and connective tissue caused by the contraction of facial muscles. These movements create folds, lines, and wrinkles in the skin and the movement of facial landmarks, such as the brows and corners of the mouth. Although muscle activation must occur if these facial actions are to be achieved, muscle action potentials in the face can occur in the absence of any overt facial action if the activation of the muscle(s) is weak or very transient, or if the overt response is aborted sufficiently early in the facial action. Facial EMG activity has therefore been especially useful in studies of emotions or emotional processes that are so weak that facial action coding is insensitive (Cacioppo, Tassinari, & Fridlund, 1980).

Representative studies of facial EMG activity as a function of discrete emotions are summarized in Table 9.4. Note that most of the research has been conducted by Schwartz and his colleagues, and that emotional imagery has been the dominant method used to study facial EMG responses as a function of discrete emotions. Two distinct conceptualizations of covert facial elerence and emotion can be identified in this literature. The first is the "microexpression hypothesis," which posits that emotion-specific covert facial expressions exist even when emotions are of such weak intensity that overt expressions of emotion are absent. According to this hypothesis, the prototypical configuration of facial muscle activity associated with an emotion emerges as visibly distinct expressions of emotion as emotional intensity increases (e.g., Schwartz, Fair, Salt, Mandel, & Klerman, 1976). The "motor recruitment hypothesis," in contrast, posits that facial elerence varies only as a function of emotional valence at weak levels of emotional intensity, and that greater emotion-specific differentiation is achieved across the facial muscles at higher levels of emotional intensity (Cacioppo, Petty, & Tassinari, 1989).

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TABLE 9.4. Facial EMG Activity as a Function of Discrete Emotions

Study	Emotional imagery	Muscle region			DAO
		CS	ZM	M	
Schwartz, Fair, Salt, Mandel, & Klerman, 1976	Happy	-	0	+	+
	Sad	+	0	0	+
	Angry	+	0	0	+
Schwartz, Alern, & Brown, 1979 ¹	Happy	0	+	+	+
	Sad	+	0	0	+
	Fearful	+	0	0	+
Brown & Schwartz, 1960	Happy	0	+	0	0
	Sad	+	0	0	0
	Angry	+	0	0	0
Smith, McHugo, & Lanzetta, 1986	Fearful	+	+	0	0
	Happy	0	+	+	+
	Sad	+	0	0	+
	Angry	+	0	0	0

Note. CS denotes the corrugator supercilii muscle region; ZM denotes the zygomaticus major muscle region; LF denotes the lateral frontal muscle region; M denotes the masseter muscle region; and DAO denotes the depressor anguli oris muscle region. Because EMG activity can vary across muscle regions, comparisons are made across emotions within muscle regions. When only an omnibus *F* ratio was reported, entries in the table were based to the extent possible on the original authors' description of the results. Entries marked by "+" denote an increase in EMG activity; entries marked by "-" denote a decrease in EMG activity; and entries marked by "0" denote nonsignificant or nominal changes in EMG activity. Where all entries in a column for a study are marked by "0," the omnibus *F* test was nonsignificant. ¹Emotions were evoked using reflective questions. ²*p* < .06.

Perusal of Table 9.4 reveals that the most reliable findings are an increase in EMG activity over the corrugator supercilii muscle region during negative emotions, and an increase in EMG activity over the zygomaticus major muscle region during positive and some negative emotions.¹ In especially comprehensive study of facial EMG activity in discrete emotions, Brown and Schwartz (1980) paced 60 subjects through 48 imagery conditions designed to elicit happiness, sadness, fear, and anger at three levels of intensity, while EMG activity was recorded over the corrugator supercilii, zygomaticus major, masseter, and lateral frontalis muscle regions. Results revealed that fearful, angry, and sad imagery were associated with lighter EMG activity over the corrugator supercilii muscle regions than was happy imagery. EMG activity over the zygomaticus major region was highest during

happy imagery, but was also elevated during fearful imagery and to a lesser extent during angry imagery. Whether these latter elevations reflect some subjects' engaging in miserable or distress smiling (Ekman, Friesen, & Ancoli, 1980), the pulling of the mouth corners up and back in a sort of silent scream, or "cross-talk" from other muscles of the middle and lower facial regions is unclear. Increasing emotional intensity led to increased EMG activity, particularly over the corrugator supercilii muscle regions during sad, angry, and fearful imagery, and over the zygomaticus major muscle region during happy imagery. Again, EMG activity over the masseter and lateral frontalis muscle regions did not vary significantly. Presumably, if the emotional intensity had been sufficient, distinctive overt facial expressions would have differentiated the negative emotions. Recent evidence further suggests that the activation functions in emotion do indeed differ for different facial muscle regions. For instance, Greenwald, Cook, and Lang (1989) plotted EMG activity along the ordinate and ranked reactions to stimuli along the abscissa such that they ranged from very negative to very positive. They found that EMG activity over the corrugator supercilii

¹A psychometric study of surface EMG recordings over the depressor anguli oris muscle region, summarized in Cacioppo, Tassinari, and Fridlund (1980), revealed that elevated EMG activity over this region could be attributable to the activation of several different muscles, and that test-retest reliabilities were modest. Therefore, EMG activity over the depressor anguli oris muscle region should be interpreted with caution.

varied linearly and negatively, whereas EMG activity over the zygomaticus major varied in a J-shaped function.

In sum, despite early suggestions that facial EMG patterns are microexpressions of emotion, the evidence for covert emotion-specific facial expressions is less compelling than is the evidence for emotion-specific overt expressions (Ekman, 1980, 1989). Interestingly, developmental studies of overt emotional expressions reveal a pattern similar to that found for weak emotional states. Positive or negative hedonic reactions to olfactory and gustatory stimuli are detectable in neonates (Steiner, 1979), but the identification of distinct patterns of fear, anger, and sadness cannot be coded reliably until the end of the first year (Camras, Malatesta, & Izard, 1991). Fox (1991) further notes that the differentiation of emotions during the first year occurs through the process of addition and integration of new motor patterns associated with approach or withdrawal. Thus, facial EMG activity during low-intensity emotions may reflect a rudimentary bivalent evaluative disposition or motivational tendency rather than discrete emotions.

Anterior Electroencephalographic Asymmetry

Davidson reviews research on electroencephalographic (EEG) activity as a function of emotion in Chapter 10 (this volume). We focus here on a subset of this research indicating that anterior EEG asymmetry differentiates evaluative dispositions rather than discrete emotions per se.

Following Kinsbourne (1978), Davidson (1984) and Fox (1991) have proposed that subcortical and cortical regions provide an important substrate for approach and withdrawal, with the right anterior region subserving a withdrawal system and the left anterior region subserving an approach system. Davidson (1984; Davidson et al., 1990) has further suggested that to the degree that approach and withdrawal are components of different emotions, such emotions should differentially activate the anterior regions of the two cerebral hemispheres. Consistent with this reasoning, studies of discrete emotions and research on individual differences have found that EEG power recorded over the right anterior (frontal) region of the scalp is higher in negative than in positive emotions. In an illus-

trative study, Davidson et al. (1990) exposed subjects to silent, emotionally evocative film clips; recorded EEG activity from anterior and posterior regions of the scalp; and coded the facial actions evinced during the films. Subsequently, facial expressions of happiness and disgust were synchronized to the EEG recordings, and hemispheric asymmetry was calculated. Results revealed that expressions of disgust were associated with greater relative left-hemispheric activation than expressions of happiness. Furthermore, this patterning was evident over the anterior but not over the posterior recording sites. Differential anterior EEG asymmetry has not been found as a function of emotions that involve a comparable withdrawal (or approach) component (e.g., Fox & Davidson, 1988; see reviews by Fox, 1991, and Davidson, 1992).

In sum, research indicates that both facial EMG responses and EEG asymmetry vary more strongly as a function of evaluative (positive-negative, approach-withdrawal) disposition than as a function of discrete emotions. The intensity of the emotion may be an important moderating factor, however, at least in the facial EMG research. This is due to evidence that the generation of strong, discrete emotions produces discriminable overt facial expressions (see Camras, Holland, & Patterson, Chapter 14, this volume). Facial EMG and EEG research, therefore, offers more than an avenue for investigating weak emotional states. It also enables investigators to examine the emergence of somatovisceral supports as the intensity of an emotion increases.

AN ORGANIZING FRAMEWORK

Given the multiple determinants of emotions and of physiological activity, it is little surprise that the literature on the psychophysiology of emotion is complex. For instance, emotion-specific ANS activity was thought by James (1884) to underlie the percept of a discrete emotional state. As a result of the causal nature of this relationship, the following conditions are implied, at least idographically (see James, 1890/1950, pp. 447-449): (1) Emotion-specific somatovisceral patterns exist that generate emotional experiences; (2) a somatovisceral pattern begins before the experience of the corresponding emotion; and (3) the somatovisceral pattern is always followed by

the experience of the corresponding emotion. Importantly, to the extent that emotional experiences are multiply determined, the experience of a discrete emotion can occur in the absence of the "corresponding" somatovisceral pattern even if somatovisceral afference is an antecedent of the emotion (i.e., $P(E/\Phi) = 1.0$; Cacioppo & Tassinary, 1990). An important implication of this reasoning is that it is more informative to ask under what conditions and for what emotions is differential physiological activity observed than to search for an invariant relationship between emotional experience (or expressions) and physiological response.

Somatovisceral Afference Model of Emotion

These considerations have led to the development of a general framework within which to view the various mechanisms by which somatovisceral afference may influence emotional experience (see Figure 9.1). The somatovisceral afference model of emotion (SAME) specifies psychophysiological conditions under which (1) the same pattern of somatovisceral afference leads to discrete emotional experi-

ences, and (2) quite different patterns of somatovisceral afference lead to the same emotional experience (Cacioppo, Bernston, & Klein, 1992). A stimulus is depicted in Figure 9.1 as initially undergoing a rudimentary evaluation. This rudimentary evaluation is represented in a central state that determines the initial motivational (e.g., approach-withdrawal) tendency and generates peripheral and central changes. The anterior hemispheric asymmetries found to differentiate motivational dispositions toward the stimulus are consistent with this feature of the model.

Somatovisceral changes are not depicted as being involved in this initial appraisal, but may nevertheless play an important role in the arousal of discrete emotional states. Specifically, the model considers the possibility that somatovisceral activity may range from emotion-specific patterns of activation to completely undifferentiated activation, with ambiguous somatovisceral activation (i.e., partially differentiated activation patterns specific to multiple emotions) falling between these two endpoints along a continuum of somatovisceral patterning (see Figure 9.1, "Somatovisceral Response" column). The nodes along this continuum represent important transitions in the

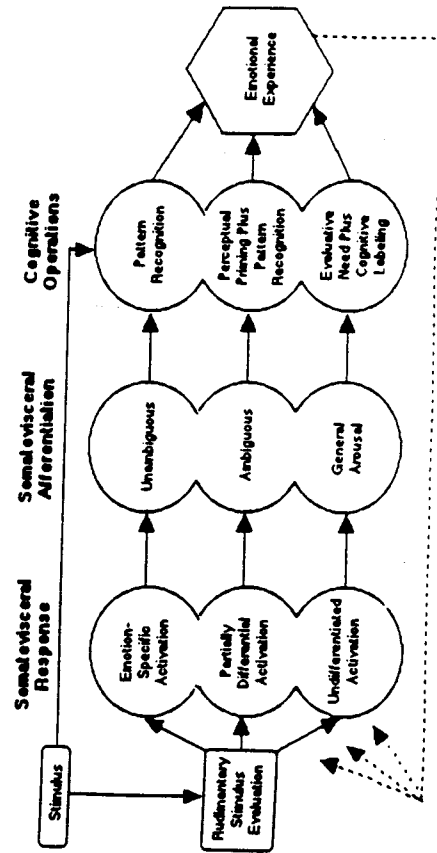


FIGURE 9.1. The somatovisceral afference model of emotion (SAME). The same pattern of somatovisceral activity has been associated with surprisingly different emotions, and the same emotion has been associated with quite different patterns of somatovisceral activity. These results have been viewed as evidence against the importance of somatovisceral afference in emotion. The SAME, depicted here and described in the text, encompasses both of these findings while emphasizing the instrumental role of somatovisceral afference and cognitive-perceptual processes in producing each. Reprinted by permission from Cacioppo, Bernston, and Klein (1992, p. 87).

constitution of the autonomic response, but the openings between these nodes underscores the continuous nature of this dimension. The pattern of somatovisceral activation produces a parallel continuum of somatovisceral sensory input to the brain. The arrows between nodes denote the major pathways for information flow.

In addition to these peripheral events, the emotional significance of the stimulus and the somatovisceral afference undergo more extensive cognitive evaluation in normal adults. Thus, Figure 9.1 also depicts the cognitive operations performed on the somatovisceral afference required to produce discrete emotional states. The extent of the cognitive elaboration of the somatovisceral afference required to produce an emotional experience ranges from simple informational analyses such as pattern recognition (e.g., James's theory of emotion as the perception of discrete patterns of somatovisceral afference) to much more complex attributional analyses and hypothesis testing (e.g., Mandler's theory of emotion), with simple cognitive appraisals of the stimulus and perceptual priming of an emotion schema falling between these two endpoints. The more extensive these cognitive operations, the longer it requires for them to be completed, and consequently the longer it takes for the somatovisceral afference to affect emotional experience. Thus, simple pattern recognition can produce an emotional experience relatively quickly, whereas detailed cognitive appraisals, attributional analyses, and systematic hypothesis testing can take longer.⁶ Note that quite different patterns of somatovisceral afference (see Figure 9.1, "Somatovisceral Response" column) can lead to the same emotional experience via three very different psychophysiological mechanisms (see Figure 9.1, "Cognitive Operations" column), whereas the same pattern of somatovisceral afference can lead to discrete emotional experiences by two distinct psychophysiological mechanisms: (1) somatovisceral "illusions" when the afference is ambiguous and an emotion schema has been primed (see below); and (2) cognitive labeling when the perception of the afference is

⁶Feature detection and discriminative processing, of course, occur during complex cognitive appraisals, too, but the proximal cognitive operations that combine with the somatovisceral afference to produce the discrete emotional states are the matters of interest here.

undifferentiated with respect to an emotion and there is an evaluative need. The former of these mechanisms warrants further comment.

Emotional Percepts as Somatovisceral Illusions

The essential feature of the proposition that discrete emotions can result from "somatovisceral illusions" can be illustrated by analogy. Figure 9.2 (see Cacioppo, Bernston, & Klein, 1992, for a more complete description of the model). Even though there is only one set of visual contours and features in Figure 9.2, top-down processes make it possible for a person looking at this picture to see or experience two very different perceptual images: the face of an Egyptian woman who is located behind a

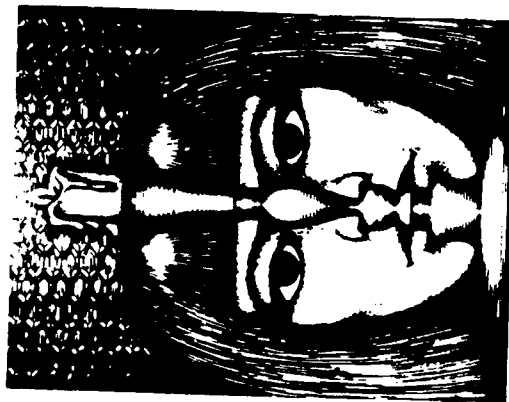


FIGURE 9.2. An ambiguous figure constructed from overlapping unambiguous elements. The picture depicts (1) the face of an Egyptian woman who is located behind a candlestick, and (2) the right and left profiles, respectively, of identical twins looking at each other. These discrete images are derived from the same sensory information, and although one can switch rapidly between these images, one cannot perceive both images simultaneously. Reprinted by permission from Shepard (1990, p. 58).

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candlestick, and the right and left profiles of identical twins looking at each other. Once these images have been identified, the viewer will find that he or she can alternate quickly between seeing these discrete images, but they cannot both be seen at once. That is, the same visual afference can lead to two different, discrete, and indistinguishable perceptual experiences.

Ambiguous visual figures such as the one depicted in Figure 9.2 are constructed using elements from two (or more) unambiguous images in such a way that the figure created by overlapping or slightly modifying the elements of the unambiguous images can be interpreted in multiple discrete ways (Sekular & Blake, 1985). Because the same sensory information in an ambiguous figure can produce such strikingly different, immediately obvious, and unambiguous perceptions, Leeper (1935) referred to ambiguous figures as "reversible illusions."

The middle nodes of Figure 9.1 denote the proposal that the active perceptual processes underlying reversible visual illusions are not limited to visual information processing, but can also operate on interoceptive (e.g., visceral) and proprioceptive (e.g., postural, facial, vocal) input.⁷ For instance, the architecture of the somatovisceral apparatus is more likely to yield ambiguous afference than is the visual system (Heed, Harver, & Katkin, 1990), and it seems likely that events as important and commonplace as the emotions have cognitive representations that include somatovisceral attributes. Thus, two important features required for the production of somatovisceral illusions are plausibly in place. A unique im-

plication of somatovisceral illusions is that discrete emotions can result from the perception of the same somatovisceral input when this input contains somatovisceral attributes of two or more discrete emotions. A second important implication is that these discrete emotional percepts are "reversible" (but can not be blended) as different emotional schemas are serially activated. Thus, just as top-down processes make it possible for people looking at Figure 9.2 to alternate quickly between seeing the face of an Egyptian woman who is located behind a candlestick and the right and left profiles of identical twins looking at each other, they may also make it possible for the person on a ride at an amusement park to alternate rapidly between the states of happy excitement and near-panic fear.

Finally, inspection of Figure 9.1 indicates some of the boundary conditions of these theories. For instance, James's (1884) theory focused on the mechanism outlined in the nodes at the top of the continua, and he did not consider the direct effects of the evaluation of the evocative stimulus on the emotional state. Cannon's (1927) theory of emotion was limited to the direct effects of the evaluation of the evocative stimulus on the rudimentary evaluative processing circuit and on the resulting activation of the viscera. Cognitive labeling theories such as Mandler's theory of emotion have focused more on the mechanism represented in the nodes at the bottom of the continua in Figure 9.1. And the processes underlying discrete emotions as somatovisceral "illusions" are represented by the middle nodes in these continua.

The framework outlined in Figure 9.1 is only heuristic at this point. It remains important to determine what are the moderating variables governing whether discrete, ambiguous, or undifferentiated somatovisceral responses are evoked by an emotional stimulus. Once this is achieved, it may be possible to specify the mechanism by which discrete emotions are linked to particular ANS changes (although the SAME does allow for the possibility that the discriminating features of somatovisceral afferences may be largely somatic in origin). Nevertheless, the model may prove helpful in designing and interpreting data from psychophysiological investigations of emotion, and in identifying moderator variables in emotion, by its explicit recognition of the different forms of somatovisceral activation and potential roles

⁷There are limitations to the usefulness of the analogy to visual processes, too. For instance, in the perception of ambiguous visual figures, the stimulus is a visual array outside the body. However, the central nervous system serves to create and interpret both the stimulus and the response to somatovisceral information. In this regard, visual processes are somewhat more like somatic instrumental processes than like visceral processes. Both differ from visceral perception, for instance, in the distinctiveness of the reafference. In the somatic case, the accuracy of response is readily ascertainable and correctable by somatosensory and visual feedback. In the visceral domain, there is no "intended" outcome in the conscious sense (although there are target outcomes in an autonomic or homeostatic sense). Hence, visceral perception differs from somatic and visual perception in that there is no discrete criterion for "correct" perception for which an individual is consciously looking. For this reason, visceral afference may be particularly prone to misperceptions and "illusions."

of somatovisceral afference in emotion. At the very least, it should diminish the tendency to view the psychophysiological mechanisms underlying emotion in terms of a simple central-peripheral dichotomy.

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