

3. E. F. Evans, in *Neuronal Mechanisms of Hearing*, J. Syka and L. Aitkin, Eds. (Plenum, New York, 1981), p. 69; M. B. Sachs and E. D. Young, *J. Acoust. Soc. Am.* **66**, 470 (1979).
4. J. E. Rose, J. E. Hind, D. J. Anderson, J. F. Brugge, *J. Neurophysiol.* **34**, 685 (1974); D. H. Johnson, thesis, Massachusetts Institute of Technology (1974); M. B. Sachs and E. D. Young, *J. Acoust. Soc. Am.* **68**, 858 (1980).
5. E. Javel, *J. Acoust. Soc. Am.* **69**, 1735 (1981).
6. N. F. Viemeister, *ibid.* **56**, 1594 (1974); B. C. J. Moore and D. H. Raab, *ibid.* **57**, 400 (1975).
7. D. H. Johnson, *Biophys. J.* **22**, 413 (1978).
8. The final threshold for each subject was based on the average of at least five separate threshold estimates. The standard deviation of these estimates was typically less than 2 dB and ranged from 0.7 to 3.8 dB.
9. The cutoff frequencies were chosen such that at the highest level of the band-reject masker, the bandpass noise was approximately 10 dB above its masked threshold for all subjects. The noises were produced by multiplying a lowpass and a bandpass noise by a 10-kHz sinusoid and adding a 20-kHz highpass noise.
10. Audio Technica ATH-7 Electret headphones were used. Since no high-frequency calibration standard exists for these or any headphones, the spectrum levels reported are the 1-kHz equivalents measured with a flat plate coupler. The overall level is approximately 43 dB greater than the reported spectrum level; the highest levels used in this experiment were in excess of 100-dB SPL overall.
11. M. H. Lurie, *J. Acoust. Soc. Am.* **11**, 420 (1940); Y. Katsuki, N. Suga, Y. Kanno, *ibid.* **34**, 1396 (1962); N. Y.-S. Kiang, *Ann. Otol. Rhinol. Laryngol.* **77**, 656 (1968).
12. M. C. Liberman, *J. Acoust. Soc. Am.* **63**, 442 (1978); *Science* **216**, 1239 (1982).
13. A. R. Palmer and E. F. Evans, *Exp. Brain Res. Suppl. II* (1979), p. 19; *Hear. Res.* **7**, 305 (1982).
14. This is the number of independent, optimally

combined Poisson channels that would yield a threshold equal to that observed in the band-reject condition. For a single channel the performance measure, d'_i , is approximately $\Delta I_{dB} r'(I) [T/r(I)]^{1/2}$, where T is the interval over which events (spikes) are counted, $r(I)$ is the mean firing rate at intensity I , $r'(I)$ is the slope of the rate-intensity function at I , and ΔI_{dB} is the decibel difference between the two stimuli. For n independent, optimally combined channels with equal d'_i , the resulting overall performance is $d' = n^{1/2} d'_i$. Thus, $n = [d' / \Delta I_{dB} r'(I)]^2 r(I) / T$. [See J. P. Egan, *Signal Detection Theory and ROC Analysis* (Academic Press, New York, 1975), pp. 162-217, for an excellent treatment of Poisson observers.] At $N_0 = 50$ dB in the band-reject condition, $\Delta I_{dB} = 4.8$ for $d' = 0.77$. Assuming a fixed counting interval of 100 msec and a mean firing rate of 100 spikes per second at $N_0 = 50$ dB, then for slopes of five or two spikes per second per decibel, n is 1.03 or 6.4 fibers, respectively. These slopes are reasonable for rate-intensity functions obtained with broadband noise at lower spectrum levels [T. B. Schalk and M. B. Sachs, *J. Acoust. Soc. Am.* **67**, 903 (1980)]. The assumption of optimum combination requires, in effect, that saturated channels, those with $d'_i = 0$, be ignored. Whether the auditory system actually achieves or approximates optimum combination is not directly relevant to the question of whether, at the level of primary fibers, a rate code is even possible.

15. I thank D. A. Burkhardt, W. D. Ward, M. Ruggero, and G. Wakefield for their comments on the manuscript. I also thank H. S. Colburn and W. M. Rabinowitz for stimulating discussions on this topic. This work was supported by grant NS12125 from the National Institute of Neurological and Communicative Disorders and Stroke.

8 February 1983; revised 24 May 1983

remedy methodological problems that have lessened the impact of previous studies: (i) A broad sample of six emotions was studied, rather than the two or three that are typical. (ii) Verification procedures were instituted to maximize the likelihood that each sample contained only the single target emotion and no other. (iii) A sufficiently broad sample of autonomic measures was obtained to enable differentiation of multiple emotions, with appropriate statistical protection against spurious findings due to multiple dependent measures. (iv) Autonomic measures were taken from the onset of emotion production continuously until it was terminated. More typical measures taken before and after production of an emotion may completely miss short-lived target emotions. (v) Multiple eliciting tasks were used with the same subjects. (vi) Professional actors ($N = 12$) and scientists who study the face ($N = 4$) served as subjects to minimize contamination of emotion samples by extraneous affect associated with frustration or embarrassment.

We studied six target emotions (surprise, disgust, sadness, anger, fear, and happiness) elicited by two tasks (directed facial action and relived emotion), with emotion ordering counterbalanced within tasks. During both tasks, facial behavior was recorded on videotape, and second-by-second averages were obtained for five physiological measures: (i) heart rate—measured with bipolar chest leads with Redux paste; (ii) left- and (iii) right-hand temperatures—measured with thermistors taped to the palmar surface of the first phalanges of the middle finger of each hand; (iv) skin resistance—measured with Ag-AgCl electrodes with Beckman paste attached to the palmar surface of the middle phalanges of the first and third fingers of the nondominant hand; and (v) forearm flexor muscle tension—measured with Ag-

Autonomic Nervous System Activity Distinguishes Among Emotions

Abstract. *Emotion-specific activity in the autonomic nervous system was generated by constructing facial prototypes of emotion muscle by muscle and by reliving past emotional experiences. The autonomic activity produced distinguished not only between positive and negative emotions, but also among negative emotions. This finding challenges emotion theories that have proposed autonomic activity to be undifferentiated or that have failed to address the implications of autonomic differentiation in emotion.*

For almost a century scientists have argued about whether or not activity in the autonomic nervous system (ANS) is emotion-specific. Some of the most influential cognitive theories of emotion (1,

2) presume undifferentiated autonomic arousal despite a number of reports of emotion-specific autonomic activity (3-5). We now report evidence of such specificity in an experiment designed to

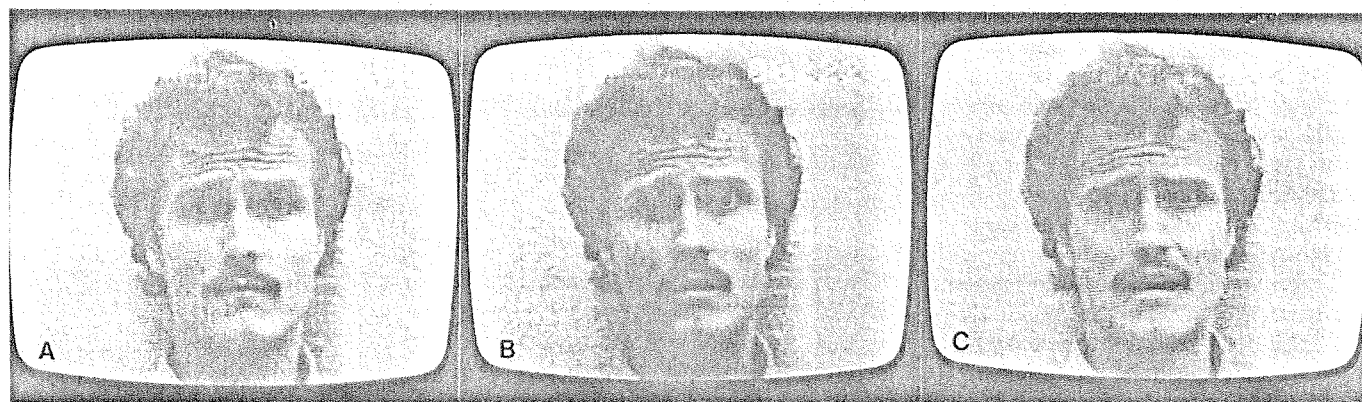


Fig. 1. Frames from the videotape of one of the actor's performance of the fear prototype instructions: (A) "raise your brows and pull them together," (B) "now raise your upper eyelids," (C) "now also stretch your lips horizontally, back toward your ears."

AgCl electrodes with Redux paste and electronic integration of the electromyogram.

The directed facial action task comprised six trials; in each a nonemotional expression was performed and followed by an emotion-prototypic expression, that is, an expression that theory and evidence indicate universally signals one of the target emotions (6). Subjects were not asked to produce an emotional expression but instead were told precisely which muscles to contract (Fig. 1). Their attempts to follow these instructions were aided by a mirror and coaching (by P.E.). The nonemotional expression comprised two actions not included in any of the emotional expressions to control for ANS changes associated with making any facial movement. Expressions were held for 10 seconds. This task resembles a traditional emotion posing task (in which, for example, subjects are asked to look fearful), but improves on it by precisely specifying for the subject, and for the experimenter's subsequent verification, the exact set of muscle movements that is required. Video records of facial expressions were measured (7) to ensure that autonomic data would be included in the analyses only if the instructed set of actions had been made; 86.5 percent of the data were used.

In the relived emotion task, subjects were asked to experience each of the six emotions (in counterbalanced orders) by reliving a past emotional experience for 30 seconds. This task resembles traditional imagery tasks, but more specifically focuses on reliving a past emotional experience. After each trial, subjects rated the intensity of any felt emotion on a scale from 0 to 8. Autonomic data were used only when the relived emotion was felt at the midpoint of the scale or greater and when no other emotion was reported at a similar strength; 55.8 percent of the data were used.

Change scores were computed for each emotion on each task (directed facial action: averaged data during emotional face minus that during nonemotional face; relived emotion: averaged data during relived emotion minus that during the preceding 10-second rest period). The experiment was analyzed in a 2 by 2 by 6 (actors versus scientists by task by emotion) multivariate analysis of variance. Our hypothesis that there are autonomic differences among the six emotions was supported [emotion main effect, $F(25, 317) = 2.51, P < 0.001$]. There were differences in emotion-specific autonomic patterns between the two eliciting tasks [task by emotion interaction, $F(25, 62) = 2.0, P = 0.014$].

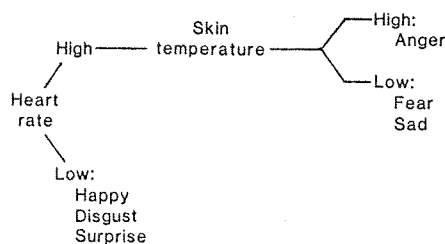


Fig. 2. Decision tree for discriminating emotions in direction facial action task.

The nature of the emotion-specific ANS activity was explored with *t*-tests within significant univariate effects. Two findings were consistent across tasks: (i) Heart rate increased more in anger (mean calculated across tasks \pm standard error, $+8.0 \pm 1.8$ beats per minute) and fear ($+8.0 \pm 1.6$ beats per minute) than in happiness ($+2.6 \pm 1.0$ beats per minute). (ii) Left and right finger temperatures increased more in anger (left, $+0.10^\circ\text{C} \pm 0.009^\circ$; right, $+0.08^\circ\text{C} \pm 0.008^\circ$) than in happiness (left, $-0.07^\circ\text{C} \pm 0.002^\circ$; right, $-0.03^\circ\text{C} \pm 0.002^\circ$).

In addition to these differences between the negative emotions of anger and fear and the positive emotion of happiness, there were important differences among negative emotions. In the

directed facial action task we were able to distinguish three subgroups of emotions (Fig. 2) on the basis of heart rate and finger temperature differences (Fig. 3). Additional differentiation in the relived emotions task enabled distinction between sadness and other negative emotions on the basis of significantly larger decreases in skin resistance in sadness [-12.6 ± 164.6 kilohm (8)] than in the others (fear, -0.37 ± 1.0 kilohm; anger, -2.1 ± 3.7 kilohm; and disgust, $+4.4 \pm 6.6$ kilohm).

There were also three negative findings of note. No significant differences were found between emotions on the forearm flexor measure, thus indicating that heart rate effects were not artifacts of fist clenching or other related muscle activity. No statistically significant differences were found between actors and scientists studying facial expression, indicating that the findings generalized to both of these populations. Finally, when the major analyses were rerun including all ANS data without regard to whether verification criteria were met, only the negative versus positive emotional distinctions remained; all distinctions among negative emotions were lost. We interpret this finding as supporting the importance of verification of emotional

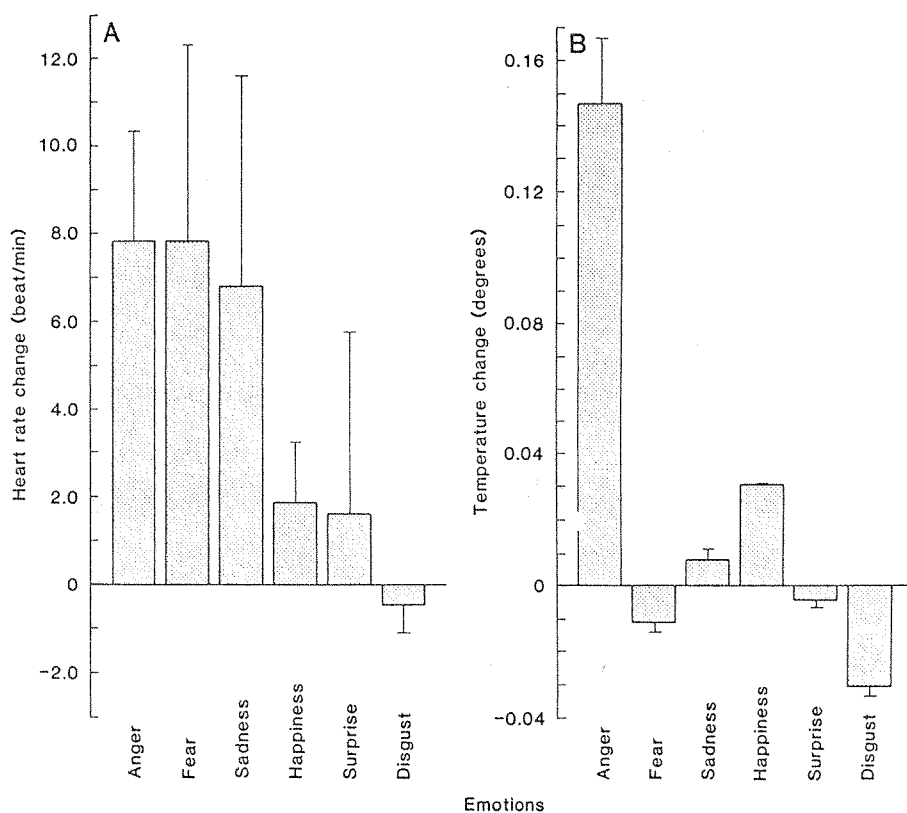


Fig. 3. Changes in (A) heart rate and (B) right finger temperature during the directed facial action task. Values are means \pm standard errors. For heart rate, the changes associated with anger, fear, and sadness were all significantly greater ($P < 0.05$) than those for happiness, surprise, and disgust. For finger temperature, the change associated with anger was significantly different ($P < 0.05$) from that for all other emotions.

state and as indicating one reason previous studies that failed to include verification procedures have been unable to distinguish so many negative emotions.

Combining the results from the two tasks, this experiment provides the first evidence (to our knowledge) of autonomic differences among four negative emotions (disgust and anger distinguished from each other and from fear or sadness in the directed facial action task; sadness distinguished from disgust, anger, or fear in the relived emotion task) as well as showing general distinctions between positive and negative emotions in both tasks. In addition to this new evidence, we replicated with the directed facial action task the single most reliable finding from past studies: anger and fear show similar heart rate increases but differ in peripheral vascular function (indicated by our finding of colder fingers in fear than in anger). The magnitude of these heart rate increases, both mean (Fig. 3) and maximum (fear, +21.7; anger, +25.3 beats per minute) are comparable to other such findings (9).

Further research is needed to choose between two alternative explanations of the differences in the results we obtained with the two eliciting tasks: (i) the tasks involve different neural substrates, which generate different patterns of emotion-specific autonomic activity; or (ii) the tasks differ in the extent of emotion blending they produce. Further work is also needed to demonstrate that emotion-specific autonomic activity is not unique to actors and scientists, although the possibility that training in either profession would have such a profound effect on autonomic patterning in emotion seems unlikely.

Our finding of emotion-differentiated autonomic activity, albeit important in

its own right, begets the question of how that activity was generated. Particularly intriguing is our discovery that producing the emotion-prototypic patterns of facial muscle action resulted in autonomic changes of large magnitude that were more clear-cut than those produced by reliving emotions (a more naturalistic process). With this experiment we cannot rule out the possibility that knowledge of the emotion labels derived from the facial movement instructions or seeing one's own or the coach's face was directly or indirectly responsible for the effect. We find this unlikely since it would indicate either (i) that just viewing an emotional face directly produced autonomic patterning or (ii) that subjects inferred the "correct" set of autonomic changes from the label and then somehow produced these complex patterns. The biofeedback literature (10) suggests that people cannot voluntarily produce such complex patterns of autonomic activity.

We propose instead that it was contracting the facial muscles into the universal emotion signals which brought forth the emotion-specific autonomic activity. This might occur either through peripheral feedback from making the facial movements, or by a direct connection between the motor cortex and hypothalamus that translates between emotion-prototypic expression in the face and emotion-specific patterning in the ANS. Although further studies are needed to verify this hypothesis and to determine the pathways involved, the fact that emotion-specific autonomic activity occurred is of fundamental theoretical importance, no matter what the underlying mechanisms may turn out to be. It raises the question of how such complex patterns of autonomic activity relate to

changes in the central nervous system, cognitive processes, motor behaviors, and the subjective experience of emotion; it also underscores the centrality of the face in emotion as Darwin (11) and Tomkins (12) suggested.

PAUL EKMAN
ROBERT W. LEVENSON*
WALLACE V. FRIESEN

Department of Psychiatry,
University of California School of
Medicine, San Francisco 94143

References and Notes

1. S. Schachter and J. E. Singer, *Psychol. Rev.* **69**, 379 (1962).
2. G. Mandler, *Mind and Emotion* (Wiley, New York, 1975).
3. A. F. Ax, *Psychosom. Med.* **15**, 433 (1953).
4. D. H. Funkenstein, S. H. King, M. Drolette, *ibid.* **16**, 404 (1954); R. A. Sternbach, *J. Psychosom. Res.* **6**, 87 (1962).
5. R. J. Roberts and T. C. Weerts, *Psychol. Rep.* **50**, 219 (1982).
6. P. Ekman and H. Oster, *Annu. Rev. Psychol.* **30**, 527 (1979).
7. P. Ekman and W. V. Friesen, *The Facial Action Coding System* (Consulting Psychologists Press, Palo Alto, Calif., 1978). To avoid directly suggesting emotion labels, we did not obtain emotion ratings, relying instead on facial measurement for verification in this task.
8. This large value reflects the influence of one outlying subject.
9. Distinctions between fear and anger were based on changes in heart rate and diastolic blood pressure and on the magnitude of heart rate change (3, 5). Increases were also comparable to heart rate change in Schachter and Singer's (1) anger-epinephrine condition.
10. G. E. Schwartz [*Science* **175**, 90 (1972)] reported one of the few instances in which biofeedback produced complex patterns of two ANS functions—heart rate and blood pressure. There are a few reports of different patterns of one autonomic and one somatic function (for example, heart rate and respiration rate) [D. Newlin and R. Levenson, *Biol. Psychol.* **7**, 277 (1978)].
11. C. Darwin, *The Expression of the Emotions in Man and Animals* (Univ. of Chicago Press, Chicago, 1965).
12. S. S. Tomkins, *Affect, Imagery, and Consciousness* (Springer, New York, 1962).
13. We thank E. Callaway, R. Lazarus, P. Tannenbaum, and S. Tomkins for their comments on this report; the John D. and Catherine T. MacArthur Foundation for research funding; and L. Temoshok, J. Kamiya, and L. Zegans for the use of equipment.

* Present address: Department of Psychology, Indiana University, Bloomington 47405.

3 May 1983

