Autonomic Nervous System Activity Distinguishes among Emotions

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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 marker, the bandpass noise was approximately 10 dB above its masked threshold for all subjects. The noise was 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.


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, $d_1'$ is approximately $M_d(0,T)/\sqrt{r(T)}$, where $T$ is the interval over which events (spikes) are counted, $r(T)$ is the mean firing rate at intensity $I$, and $M_d$ is the slope of the rate-intensity function at $I$. $d_1'$ is adjusted to $M_d$. The difference between the two stimuli. For $n$ independent, optimally combined channels with equal $d_1'$, the resulting overall performance is $d_1' = \sqrt{n} d_1'$. Thus, $n = (\lambda/M_d(0,T))^2/\lambda T$.

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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](image-url)  
**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 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, $F(25, 62) = 2.0$, $P = 0.014$].

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 ± standard error, +8.0 ± 1.8 beats per minute) and fear (+8.0 ± 1.6 beats per minute) than in happiness (+2.6 ± 1.0 beats per minute). (ii) Left and right finger temperatures increased more in anger (left, +0.10°C ± 0.009°C; right, +0.08°C ± 0.008°C) than in happiness (left, −0.07°C ± 0.002°C; right, −0.03°C ± 0.002°C).

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 ± 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
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.

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References and Notes
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 (3.51 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)).
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