The psychophysiology of crying

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Abstract

Two conflicting views have emerged as to why people cry when they are sad. One suggests that crying serves homeostasis by facilitating recovery; the other suggests that crying produces an aversive high-arousal state that motivates behavior aimed at ending the tears. To test hypotheses drawn from these views, we showed a short film known to elicit sadness to 150 women. During this film, 33 subjects spontaneously cried and 117 did not. Subjects who cried exhibited more expressive behavior and reported feeling more sadness and pain than did subjects who did not cry. Crying also was associated with increases in somatic and autonomic nervous system activity. The increases in autonomic activity could not be accounted for solely by the increases in somatic activity. Crying is thus associated with an aversive state, including negative emotion and a complex mixture of sympathetic, parasympathetic, and somatic activation, and we speculate about the functional implications of these findings.

Descriptors: Crying, Sadness, Tears, Grief, Recovery, Emotion

Crying when sad appears to be a uniquely human response (Frey & Langseth, 1985), and scientists since Darwin (1872) have theorized about the functions of such tears. In this paper, we review two schools of thought as to the functions crying may serve and test two hypotheses drawn from these views.

The Recovery View

Common to many theories of crying is the notion that tears of sadness help to restore homeostasis. Three mechanisms have been proposed for the homeostatic function of crying: (a) alleviating depression or related negative affects (Beck, Rush, Shaw, & Emery, 1979; Scheff, 1979); (b) releasing stress-related biochemical toxins (Frey & Langseth, 1985; Frey, DeSota-Johnson, Hoffman, & McCall, 1981); or (c) decreasing sympathetic nervous system activation (Bindra, 1972; Efran & Spangler, 1979).

Alleviating depression. Theories that link crying to the cathartic release of depression and related negative emotions are largely based on anecdotal evidence from clinicians and from criers themselves (Beck et al., 1979; Frey & Langseth, 1985; Kraemer & Hastrup, 1988; Sadoff, 1966; Scheff, 1979). Such theories hinge on the fact that phenomenologically most people report feeling better after they cry (Frey & Langseth, 1985), endorsing

the view that "having a good cry" releases pent-up negative emotions (Frijda, 1986).

These theories make the general prediction that crying should be associated with decreases in negative emotion, increases in positive emotion, or both. In one test of this prediction, Labott and Martin (1988) found that, compared with subjects who did not cry during a sad film, subjects who cried reported greater levels of stress during the film and similar levels of stress 2 hr later. Choti, Marston, Holston, and Hart (1987) reported comparable data. How, then, are we to explain the common perception that crying makes one feel better? As Kraemer and Hastrup (1988) noted, anecdotal reports about how people feel after they cry do not take into account the mere passage of time. Perhaps negative emotions dissipate over time with or without tears. One way to test whether crying brings greater relief than does time alone is to compare mood ratings obtained after crying with ratings obtained after people felt like crying but did not. Having collected such data in both laboratory and natural environments, Kraemer and Hastrup suggested that depressed affect typically diminishes in 1 or 2 hr regardless of whether people cry or inhibit crying (Kraemer & Hastrup, 1986; see also Martin & Labott, 1991).

Releasing toxins. Frey and colleagues have argued that emotion-induced crying, like other excretory processes (e.g., exhaling, urinating, perspiring, defecating), helps to restore homeostasis, in this case via the release of stress-related toxins (Frey & Langseth, 1985; Frey et al., 1981). In support of the view that tears of emotional distress serve a special function, tears shed while viewing a sad film have been found to contain higher concentrations of protein than tears shed in the presence of a freshly cut onion (Frey et al., 1981).

Although these findings provide a basis for the claim that emotional and nonemotional tears differ, the argument that the increased protein concentration in emotional tears represents a release of toxins in the service of homeostatic reequilibration requires a large inferential leap.

Decreasing sympathetic activation. Other proponents of the recovery view have pointed to the fact that the lacrimal gland, which is responsible for the production of tears, is innervated by parasympathetic fibers of the seventh cranial nerve (Carpenter & Sutin, 1983; Records, 1979; Werb, 1983). Assuming that sympathetic and parasympathetic branches of the autonomic nervous system act in opposition, some researchers have suggested that intense emotions spark sympathetic activation, which may be followed by a burst of parasympathetic activity that serves to reduce previous high levels of sympathetic activation and restore autonomic quiescence. According to this view, tears are the result of a temporary parasympathetic overcompensation for previous high levels of sympathetic activation (Bindra, 1972; Efran & Spangler, 1979; Frijda, 1986; Heilbrun, 1955). Crying is thus seen as a recovery or arousal-reduction response: "The onset of crying seems to indicate that . . . the autonomic system shifts from arousal to recovery" (Efran & Spangler, 1979, p. 71).

As was the case with alleviation of depression and removal of toxins models, this physiological model suggests that crying is associated with restoration of homeostasis. However, unlike these other mechanisms, here there is an explicit prediction of temporal sequencing within the recovery process (i.e., sympathetic activation precedes parasympathetically mediated crying). This leads to an empirically testable hypothesis, a variant of the recovery view that we will call the *physiological recovery hypothesis*, that predicts that once subjects begin to cry they should enter a lower arousal state of decreased sympathetic nervous system activation.

The Arousal View

Despite the chorus of theories consistent with the recovery view of crying, remarkably few published studies have directly tested its predictions. In fact, even the physiological recovery hypothesis has only been subjected to one empirical test. In that study, Kraemer and Hastrup (1988) hypothesized that the onset of crying would be associated with decreases in both heart rate and electrodermal responding. Data from their comparison of 16 criers with 16 noncriers revealed just the opposite: heart rate and skin conductance fluctuations each significantly increased once subjects began to cry. Contrary to the physiological recovery hypothesis, these data suggest that crying produces a state of high physiological arousal, a variant of the arousal view that we will call the *physiological arousal hypothesis*.

The arousal view suggests a very different function of crying from that of the recovery view — if crying is associated with high levels of physiological arousal, it cannot also facilitate physiological recovery (at least in the short term). Instead, as Tomkins (1963) and Averill (1968) have postulated, crying may lead to a physiologically stressful and aversive state of activation that signals that all is not well. This state may function to maintain social cohesion as both crier and proximate others strive to alleviate the aversive tears.

Unfortunately, at least two obstacles limit our ability to generalize from the Kraemer and Hastrup (1988) study and thus decide which of the two competing hypotheses regarding the functions of crying has greater merit. First, Kraemer and Hastrup (1988) used a restricted sample consisting of carefully selected high-frequency and low-frequency criers identified on the basis of a self-report measure of crying frequency. Second, their subjects were instructed either to cry or not to cry, a request that itself may have been arousing.

Empirical Strategy

To evaluate these two opposing models of crying, we presented a sadness-eliciting film to female subjects in two different experiments conducted in the same laboratory under similar conditions. We chose to restrict our study to women because our previous experience using this film with an independent sample of subjects of both sexes revealed that 24% of women but only 3% of men spontaneously cried. This finding was consistent with other reports that women cry much more frequently and easily than men (see Frey & Langseth, 1985; Labott & Martin, 1987; Williams, 1982). Because we hoped to have a sample of at least 30 episodes of crying, we estimated that we would have to show this film to 1,000 men, a prohibitively large number. Instead, we decided to study female subjects, combining data from two experiments (150 subjects in all, 33 [22%] of whom cried) to explore two competing hypotheses: (a) The physiological recovery hypothesis, which predicts that crying will lead to decreased sympathetic arousal, and (b) the physiological arousal hypothesis, which predicts that crying will lead to increased sympathetic arousal.

Method

Subjects

One hundred fifty female undergraduates enrolled in introductory psychology courses at the University of California–Berkeley served as voluntary participants in one of two experiments. Subjects received course credit in exchange for their participation.

Experiment 1. Sixty female undergraduates participated in an experiment conducted during the 1990–1991 academic year. These subjects ranged in age from 17 to 29 years ($M = 19.07$ years, $SD = 1.7$ years). Forty percent of subjects in Experiment 1 identified themselves as Asian, 7% as black, 22% as Caucasian, and 31% as Latino, which approximates the demographics of the student population at the University of California–Berkeley.

Experiment 2. Ninety female undergraduates participated in an experiment conducted during the 1991–1992 academic year. Subjects ranged in age from 18 to 26 years ($M = 19.14$ years, $SD = 1.2$ years). As in Experiment 1, sample ethnicity in Experiment 2 approximated that of the Berkeley student population: 40% Asian, 8% black, 29% Caucasian, 20% Latino, and 3% other.

Film Stimulus

Subjects viewed one of two versions of a short film clip depicting a mother at her daughter's funeral. The film clips were excerpted from the film *Steel Magnolias* (Stark & Ross, 1989) and will be referred to as the *sadness* film.

Experiment 1. Subjects viewed a version of the sadness film that starts with a neighbor coming to visit a family at their house and then shifts to a scene where the mother is quietly telling her

friends at her daughter's graveside how she felt watching her daughter die. As she talks, she begins to cry, and by the end of the film clip, tears are streaming down her face. The film clip ends with a humorous interchange between the mother and a friend. This version of the film stimulus is 320 s in length.

Experiment 2. Subjects viewed a slightly shorter version of the same film stimulus, which begins at the point at which the mother tells her friends how she felt watching her daughter die. This version continues with the mother beginning to cry and ends with tears streaming down her face (the humorous interchange was omitted). This version of the film stimulus is 205 s in length.

Procedure

Subjects participated in individual experimental sessions. Upon arrival, they were seated in a comfortable chair in a small, well-lit room. The experimenter told subjects that the study was designed to learn more about emotion, that they would be videotaped,[1] and that their reactions would be monitored using physiological sensors. Subjects signed a consent form, and physiological sensors were attached. During a 5-min adaptation period, subjects completed a demographic questionnaire.

Experiment 1. Subjects were exposed to several stimuli over the course of the 1-hr session, including other films, music, and white noise. Subjects then watched the sadness film on a 27-in. color monitor positioned at a distance of 1.75 m. Prior to the film, subjects were instructed to relax for a few minutes. The last 60 s of this prefilm rest period served as a baseline period. After the film, subjects used a self-report inventory to describe their emotional responses to the film.[2]

Experiment 2. Subjects viewed one to three emotion-eliciting or neutral films and then watched the sadness film on the same monitor, with the same instructions, prefilm baseline, and post-film emotional report as in Experiment 1. The number or kind of films viewed prior to the sadness film did not influence the probability of subjects crying during the sadness film.

Apparatus

The apparatus used in each experiment were identical, and the two experiments were conducted in the same experimental room.

Audiovisual. A remotely controlled high-resolution color video camera placed behind darkened glass behind a bookshelf was used to record subjects' facial behavior and upper body movement unobtrusively.

Self-report. Subjects indicated their emotional responses to the film using a self-report inventory consisting of 16 terms: amusement, anger, arousal, confusion, contempt, contentment,

disgust, embarrassment, fear, happiness, interest, pain, relief, sadness, surprise, and tension. Subjects were asked to rate the greatest amount of each emotion they had felt during the film, using an anchored 9-point Likert scale (0 = none, 8 = the most in my life; adapted from Ekman, Friesen, & Ancoli, 1980).

Physiological. Using a 12-channel Grass Model 7 polygraph connected to a Digital Electronics Corporation LSI-11/73 microcomputer, continuous recordings were made of nine physiological measures.

1. Heart rate. Beckman miniature electrodes with Beckman electrolyte were placed in a bipolar configuration on opposite sides of the subject's chest. The interbeat interval was calculated as the time in milliseconds between successive R waves in the electrocardiogram (EKG).

2. Skin conductance level. A Med Associates device was used to pass a small constant voltage between Beckman regular electrodes attached to the palmar surface of the middle phalanges of the first and third fingers of the nondominant hand. The electrolyte was sodium chloride in Unibase.

3. Finger temperature. A Yellow Springs Instruments thermistor taped to the palmar surface of the distal phalange of the fourth finger of the nondominant hand was connected to a Med Associates device that provided a measure of finger temperature in degrees Fahrenheit.

4. Pulse transmission time to the finger. A UFI photoplethysmograph was attached to the distal phalange of the second finger of the nondominant hand. The interval was timed between the R wave of the EKG and the upstroke of the pulse wave at the finger.

5. Finger pulse amplitude. The trough-to-peak amplitude of each finger pulse was measured to index the maximum volume of blood in the tip of the finger during each heart beat.

6. Pulse transmission time to the ear. A UFI photoplethysmograph was attached to the right ear. The interval was timed between the R wave of the EKG and the upstroke of the pulse wave at the ear.

7. Respiration period. A pneumatic bellows was stretched around the thoracic region, and the intercycle interval was measured as the time in milliseconds between successive inspirations.

8. Respiration depth. The point of maximum inspiration minus the point of maximum expiration was determined from the respiratory tracing.

9. General somatic activity. An electromechanical transducer attached to a platform under the subject's chair generated an electrical signal proportional to the amount of movement in any direction.

This set of physiological measures was selected to sample broadly from major organ systems known to be important to emotional responding (cardiac, vascular, electrodermal, respiratory, and somatic), to allow for continuous measurement, and to be as unobtrusive as possible.

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[1] The Committee for the Protection of Human Subjects at the University of California–Berkeley prefers that subjects be informed about videotaping prior to the start of experiments.

[2] A subset of subjects also provided on-line ratings of how they were feeling throughout the film. These ratings had no effect on the results presented here and are not reported.

Behavioral Data Reduction
The procedure for determining the precise onset of crying was the same for subjects in each experiment. In addition, for subjects in Experiment 2 only, a number of other aspects of expressive behavior were also coded.

Experiments 1 and 2. To determine precisely the onset of crying, two trained coders (the first and second authors) carefully reviewed the video records of subjects' behavioral responses during the sadness film. Because subjects in Experiment 1 saw a longer version of the sadness film than was used in Experiment 2, we considered only the 205 consecutive seconds of the film that were viewed by subjects in both experiments. Defining *crying* as moisture in at least one eye that represented a clear change from baseline, the coders identified 10 subjects from Experiment 1 and 23 subjects from Experiment 2 as *criers*. For these 33 criers, the coders independently identified the precise second during the film at which crying began; disagreements were resolved by discussion. Although the initial aim was to code cry offset as well, this coding was impossible because all 33 criers had at least one eye that remained visibly moist until the end of the film segment that was reviewed by coders. Subjects began crying from 20 to 183 s into the film ($M = 104$ s, $SD = 19.1$ s), and the duration of subjects' crying episodes (marked by the end of the common film stimulus) ranged from 22 s to 185 s ($M = 101$ s, $SD = 19.1$ s).

Experiment 2. For the 90 subjects in Experiment 2 only, behavioral responses during the film were independently coded by four coders (two male, two female) using a system developed in this laboratory (see Gross & Levenson, 1993a) that includes 11 codes: (a) blinks, (b) body movement, (c) crying, (d) face touching, (e) facial movement, (f) happiness behavior, (g) intensity of behavior, (h) mouth movement, (i) pleasantness of behavior, (j) sadness behavior, and (k) smiles. Two of these responses were frequency measures (blinks, smiles), which were converted to events per minute for analysis. The rest were continuous measures whose values represented an aggregate of intensity, duration, and frequency of response. Interrater reliabilities for this system are reasonably high (e.g., mean Pearson $r = .81$, range: .64 for sadness to .96 for smiling: Gross & Levenson, 1994).

Physiological Data Reduction
During the experimental sessions, laboratory software computed second-by-second averages for each of the nine physiological measures throughout baseline and film periods and stored the results on disk. For each of the physiological variables, these second-by-second values were used to compute each subject's averages for the 60-s baseline period and for a precry period and a cry period during the film. Because subjects in Experiment 1 saw a longer version of the sadness film than was used in Experiment 2, we only considered data from the 205 consecutive seconds of the film that were viewed by subjects in both experiments.

For each subject who cried, we partitioned physiological responses into two periods defined by the onset of crying (precry and crying) and calculated average physiological responses for each of the nine physiological variables for these two periods. Subjects who did not cry were randomly matched to subjects who did cry. Physiological responses of noncriers were divided into periods equivalent to those of the crier with whom they had been matched, and average physiological responses were calculated for each of the nine physiological variables for these two periods. The periods used to compare criers and noncriers are thus equivalent with respect to location and to mean (standard deviation) length.

Results

Analytic Strategy
Because of the low base rates of spontaneous crying, we combined subjects from both experiments in our analyses. This approach seemed acceptable given that (a) we used the same sadness elicitation procedure in each experiment, (b) we employed the same dependent measures in each experiment, and (c) subjects in these two experiments did not differ in age ($t[98.13] = -0.31$, n.s.) or ethnicity ($\chi^2[4, N = 150] = 4.77$, n.s.). Because there might nonetheless be important differences between the two experiments, we included experiment as a between-subjects variable in the analyses.

For the behavioral and self-report variables (which were available for the whole film period only), we compared criers' responses to the sadness film with those of noncriers. For the physiological variables, we computed averages for precry and crying periods based on the actual time of crying for criers and matched periods for noncriers as described above. To assess whether crying was associated with recovery or arousal, we computed change scores (from the baseline period preceding the sadness film) for each period and then compared physiological responses during precry and crying periods. In this way, data from noncriers provided a control for the influence of the passage of time.

Competing Hypotheses
The physiological recovery hypothesis and the physiological arousal hypothesis make diametrically opposing predictions concerning the changes that should occur in the physiological variables when they are examined prior to and following the onset of tears. More specifically, relative to noncrying controls, the physiological recovery hypothesis predicts that criers will show a decrease in sympathetic nervous system activation during the crying period relative to the precry period, whereas the physiological arousal hypothesis predicts that criers will show an increase in sympathetic nervous system activation across the two periods.

Before examining the data relevant to these two hypotheses, we assessed several matters relevant to the efficacy of the experimental procedure and to the behavioral and subjective concomitants of crying.

Effectiveness of the Elicitation Procedure
As a manipulation check, we determined whether the sadness film had indeed elicited an emotional response and whether both subjects and observers would describe this response as sadness by examining subjects' emotion self-reports and observers' ratings of expressive behavior. Both sources of data suggest that our elicitation procedure was successful. Subjects reported significantly greater sadness than any other emotion (Table 1). Regarding expressive behavior, subjects in Experiment 2 (the only subjects for whom behavioral ratings were available) were coded as showing significantly elevated levels of sadness expressive behavior ($M = 2.04$, 95% confidence interval = 1.64–2.44, which does not include 0.00).

Table 1. *Mean Values (Standard Errors) for Emotion Self-Reports*

Emotion	Self-report
Amusement	2.78 (0.19)
Anger	2.05 (0.17)
Arousal	1.85 (0.16)
Confusion	1.47 (0.15)
Contempt	1.33 (0.16)
Contentment	1.59 (0.16)
Disgust	0.81 (0.12)
Embarrassment	0.41 (0.09)
Fear	1.00 (0.12)
Happiness	1.78 (0.18)
Interest	4.17 (0.15)
Pain	2.37 (0.19)
Relief	1.67 (0.18)
Sadness	4.57 (0.15)
Surprise	1.87 (0.17)
Tension	2.51 (0.16)

Note: All emotion ratings differ from sadness ratings at $p < .05$.

Thus, the elicitation procedure was effective in producing both self-report and behavior consistent with the emotion of sadness (see Gross & Levenson, 1993b, for a discussion of the importance of assessing behavior and self-report data when eliciting emotion with film stimuli).

Behavioral Concomitants of Crying

To understand the larger context within which crying occurred, we examined the behavioral concomitants of crying. Because behavioral data were available only for subjects in Experiment 2 (23 criers, 67 noncriers), we first conducted a two-level multivariate analysis of variance (MANOVA) (cry [yes, no]) with all of the behavioral variables except crying (which was used to determine group membership). This analysis revealed a multivariate main effect for cry, $F(10,79) = 13.46$, $p < .001$.

Follow-up univariate analyses for each behavioral code indicated that, compared with noncriers, subjects who cried also showed more sadness behavior (criers = 4.48, noncriers = 1.21, $t[30.18] = -9.61$, $p < .001$), more emotional intensity (criers = 4.17, noncriers = 1.93, $t[88] = -7.18$, $p < .001$), more face touching (criers = 2.61, noncriers = 1.15, $t[30.16] = -2.82$, $p = .008$), more facial movement (criers = 3.26, noncriers = 1.69, $t[88] = -5.38$, $p < .001$), more mouth movement (criers = 3.04, noncriers = 2.24, $t[88] = -2.31$, $p = .023$), and less pleasantness (criers = 0.87, noncriers = 1.88, $t[88] = 6.40$, $p < .001$). There were no differences between criers and noncriers in happiness behavior, smiling, blinks, or body movement.

In summary, crying was associated with increased sadness behavior, more intense expressive displays, increased face touching (possibly to wipe away tears), increased facial movement, increased mouth movement, and decreased pleasant expressive behavior.

Subjective Concomitants of Crying

To assess the subjective correlates of crying, we conducted a 2×2 MANOVA (cry [yes, no], experiment [first, second]) with all 16 emotion self-report variables. This MANOVA yielded a multivariate main effect for cry, $F(16,126) = 4.68$, $p < .001$, and

for experiment,[3] $F(16,126) = 12.61$, $p < .001$, and a Cry $\times$ Experiment interaction $F(16,126) = 3.32$, $p < .001$.

Follow-up univariate tests for the multivariate cry effect revealed effects for embarrassment, sadness, and pain, which indicated that subjects who cried during the sadness film reported greater embarrassment (criers = 1.00, noncriers = 0.24, $t[32.55] = -2.14$, $p = .04$), greater sadness (criers = 5.71, noncriers = 4.27, $t[146] = -4.06$, $p < .001$), and greater pain (criers = 3.44, noncriers = 2.09, $t[147] = -2.92$, $p = .004$) than subjects who did not cry.

Follow-up univariate tests for the multivariate Cry $\times$ Experiment interaction revealed interactions for amusement, $F(1,145) = 4.39$, $p = .038$, and embarrassment, $F(1,144) = 29.08$, $p < .001$. The t tests comparing criers and noncriers in each experiment revealed that criers reported more embarrassment than noncriers in Experiment 1 (criers = 2.67, noncriers = 0.30, $t[8.14] = -2.42$, $p = .041$) but not in Experiment 2 (criers = 0.35, noncriers = 0.19, $t[27.56] = -0.75$, n.s.). There were no differences in amusement self-reports between criers and noncriers for either Experiment 1 or Experiment 2.

In summary, the level of self-reported emotional experience and the pattern of changes associated with crying was the same in each experiment (as indicated by the relative absence of Cry $\times$ Experiment interactions), with the exception that in Experiment 1 only, crying was associated with increased embarrassment.[4] In each experiment, crying was associated with increased sadness and pain.

Physiological Concomitants of Crying

To assess the physiological effects of crying, we conducted an overall $2 \times 2 \times 2$ MANOVA (cry [yes, no], experiment [first, second], episode [precry, cry], with episode as a within-subjects variable) with all nine physiological variables. This overall

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[3] The version of the sadness film used in Experiment 1 was longer and more emotionally complex than the version used in Experiment 2 insofar as the former had two additional segments (a visit by a neighbor at the start and a humorous interchange between the neighbor and the mother at the end). For this reason, we expected to find some differences in the emotional reports elicited by the two versions. Clearly, sadness was the emotion of greatest interest to us in this study of crying, and the two versions did not differ in how much sadness they produced. However, the excerpt used in Experiment 1 produced greater amounts of a number of other emotions, including amusement, anger, confusion, contempt, contentment, disgust, embarrassment, happiness, interest, relief, and surprise. For these differences, univariate experiment effects and means were as follows: amusement (Experiment 1 = 4.81, Experiment 2 = 1.42, $t[147] = 12.20$, $p < .001$), anger (Experiment 1 = 2.54, Experiment 2 = 1.71, $t[147] = 2.48$, $p = .014$), confusion (Experiment 1 = 1.81, Experiment 2 = 1.22, $t[147] = 1.99$, $p = .049$), contempt (Experiment 1 = 1.93, Experiment 2 = 0.92, $t[98.68] = 3.11$, $p = .002$), contentment (Experiment 1 = 2.78, Experiment 2 = 0.80, $t[86.45] = 6.52$, $p < .001$), disgust (Experiment 1 = 1.27, Experiment 2 = 0.50, $t[85.49] = 2.98$, $p = .004$), embarrassment (Experiment 1 = 0.66, Experiment 2 = 0.23, $t[73.32] = 2.05$, $p = .044$), happiness (Experiment 1 = 3.54, Experiment 2 = 0.62, $t[81.58] = 9.14$, $p < .001$), interest (Experiment 1 = 5.17, Experiment 2 = 3.51, $t[147] = 5.89$, $p < .001$), relief (Experiment 1 = 3.57, Experiment 2 = 0.43, $t[71.98] = 10.30$, $p < .001$), and surprise (Experiment 1 = 2.71, Experiment 2 = 1.29, $t[94.97] = 3.97$, $p < .001$).

[4] In Experiment 1, subjects who cried reported feeling more embarrassed than did noncriers. They also reported greater embarrassment than either criers or noncriers in Experiment 2. Given that the version of the sadness film used in Experiment 1 had a humorous ending, it may be that crying in the context of something amusing was what was embarrassing for these subjects.

Table 2. *Mean Change (Standard Errors) from Prefilm Rest Period for Physiological Variables*

	Criers		Noncriers	
Variable[a]	Precry	Crying	First episode	Second episode
IBI	31.98 (9.09)	−18.20 (12.20)	30.11 (4.37)	17.22 (4.38)
SCL	0.19 (0.13)	0.09 (0.19)	−0.14 (0.06)	−0.36 (0.08)
FPA	−2.46 (0.46)	−3.00 (0.46)	−1.80 (0.28)	−2.39 (0.34)
TEM	−0.72 (0.14)	−1.55 (0.24)	−0.47 (0.08)	−0.91 (0.15)
RP	−141.79 (151.27)	−53.30 (190.52)	−404.12 (74.33)	−576.41 (75.38)
RD	−21.32 (22.23)	3.58 (24.11)	−38.24 (8.01)	−26.21 (8.43)
ACT	−0.05 (0.02)	−0.02 (0.02)	−0.07 (0.01)	−0.07 (0.01)
PTTF	9.49 (2.26)	10.62 (2.53)	6.43 (1.17)	7.79 (1.35)
PTTE	−1.18 (1.11)	−0.02 (1.83)	−0.44 (0.54)	0.12 (0.67)

[a]IBI = interbeat interval; SCL = skin conductance level; FPA = finger pulse amplitude; TEM = finger temperature; RP = respiratory period; RD = respiratory depth; ACT = general somatic activity; PTTF = pulse transmission time to the finger; PTTE = pulse transmission time to the ear.

MANOVA revealed effects of cry, $F(9,136) = 3.16$, $p = .002$, experiment, $F(9,136) = 2.18$, $p = .027$, and episode, $F(9,136) = 18.20$, $p < .001$ and an interaction of Cry × Episode, $F(9,136) = 5.54$, $p < .001$.

These multivariate effects indicated that subjects' physiological responses varied as a function of whether or not they were crying. Importantly, the absence of either Cry × Experiment or Cry × Experiment × Episode effects suggests that the overall effects of crying did not vary as a function of experimental context. Table 2 presents the means and standard errors for all physiological variables for both criers and noncriers (broken down by episode).

In the sections that follow, we present the results of the follow-up univariate tests for the significant multivariate cry, episode, and Cry × Episode effects.[5] For each of the nine physiological variables, we consider whether the results support the physiological recovery or the physiological arousal hypotheses.

Heart rate. For heart rate, there was an effect of episode, $F(1,148) = 76.22$, $p < .001$, as well as a Cry × Episode interaction, $F(1,148) = 26.65$, $p < .001$. Follow-up t tests indicated that, although criers did not differ from noncriers during the precry period (mean change in interbeat interval: criers = 31.98, noncriers = 30.11, $t[148] = 0.19$, n.s.), criers did show greater decreases in interbeat interval (indicating faster heart rates) than did noncriers during the cry period (mean change in interbeat interval: criers = −18.20, noncriers = 17.22, $t[148] = -3.54$, $p < .001$). These heart rate data favor the physiological arousal hypothesis over the physiological recovery hypothesis.

Skin conductance level. For skin conductance, there was an effect of cry, $F(1,148) = 6.71$, $p = .011$, and an effect of episode, $F(1,148) = 8.09$, $p = .005$. Follow-up t tests indicated that across all subjects skin conductance level generally declined from

the first half to the second half of the film (mean change in skin conductance: first episode = −0.07, second episode = −0.26, $t[149] = 4.11$, $p < .001$). Over the same period, however, criers showed greater increases in skin conductance than did noncriers (mean change in skin conductance: criers = 0.14, noncriers = −0.25, $t[148] = -2.59$, $p = .011$). Although these data on electrodermal activity do not reveal the expected Cry × Episode interaction, they nonetheless favor the physiological arousal hypothesis, suggesting that subjects who cried during the film had greater electrodermal activation throughout the film relative to subjects who did not cry.

Finger pulse amplitude. For finger pulse amplitude, there was an effect of episode, $F(1,148) = 17.31$, $p < .001$. Follow-up t tests indicated a general decrease in finger pulse amplitude (indicating greater vasoconstriction) from the first half of the film to the second half of the film across all subjects (mean change in finger pulse amplitude: first episode = −1.95, second episode = −2.52, $t[149] = 5.16$, $p < .001$). Because changes in vasoconstriction did not discriminate criers from noncriers, these data do not help us decide between the physiological arousal and physiological recovery hypotheses.

Finger temperature. For finger temperature, there were effects of cry, $F(1,148) = 3.92$, $p = .049$, and episode, $F(1,148) = 50.28$, $p < .001$ and an interaction of Cry × Episode, $F(1,148) = 4.86$, $p = .029$. Follow-up t tests indicated that although criers did not differ from noncriers during the precry period (mean change in finger temperature: criers = −0.72, noncriers = −0.47 $t[148] = -1.03$, n.s.), criers did show greater decreases in finger temperature than did noncriers during the cry period (mean change in finger temperature: criers = −1.55, noncriers = −0.91, $t[148] = -2.65$, $p = .009$). Assuming that decreases in skin temperature are indicative of higher arousal, these data favor the physiological arousal hypothesis over the physiological recovery hypothesis.

Respiratory period. For respiratory period, there was an effect of cry, $F(1,148) = 5.78$, $p = .017$, and an interaction of Cry × Episode, $F(1,148) = 11.38$, $p = .001$. Follow-up t tests revealed that although criers did not differ from noncriers dur-

[5] Follow-up univariate tests for the multivariate experiment effect revealed an effect for finger pulse amplitude, $F(1,148) = 4.41$, $p = .038$. This effect appears to have been due to Experiment 1 subjects having lesser decreases in finger pulse amplitude during the film than Experiment 2 subjects (mean change in finger pulse amplitude: Experiment 1 = −1.59, Experiment 2 = −2.67, $t[147.52] = 2.25$, $p = .026$).

ing the precry episode (mean change in respiratory period: criers = −141.79, noncriers = −404.12, $t[148] = 1.56$, n.s.), criers did show lesser decreases in respiratory period than did noncriers during the cry episode (mean change in respiratory period: criers = −53.30, noncriers = −576.41, $t[148] = 3.12$, $p = .003$). Assuming that faster rates of breathing indicate greater arousal, these data, unlike the data for heart rate, skin conductance, and finger temperature, support the physiological recovery hypothesis over the physiological arousal hypothesis.

Respiratory depth. For respiratory depth, there was an effect of episode, $F(1,148) = 8.24$, $p = .005$. Follow-up t tests indicated that there was a general increase in respiratory depth from the first half of the film to the second half of the film (mean change in respiratory depth: first episode = −34.52, second episode = −19.65, $t[149] = −2.79$, $p = .006$). Because changes in respiratory depth did not discriminate criers from noncriers, these data do not help us decide between physiological arousal and physiological recovery hypotheses.

Somatic activity. For somatic activity, there was an effect of episode, $F(1,146) = 9.72$, $p = .002$, and an interaction of Cry × Episode, $F(1,146) = 9.19$, $p = .003$. Follow-up t tests indicated that although criers did not differ from noncriers during the precry episode (mean change in somatic activity: criers = −0.05, noncriers = −0.07, $t[146] = 0.76$, n.s.), criers did show lesser decreases in somatic activity than did noncriers during the cry episode (mean change in somatic activity: criers = −0.02, noncriers = −0.07, $t[146] = 2.40$, $p = .017$). These data suggest that criers were more active than noncriers, lending support to the physiological arousal hypothesis over the physiological recovery hypothesis.

Summary. Five of the nine psychophysiological variables that we measured distinguished those who cried during a sad film from those who remained dry-eyed: (a) heart rate, (b) skin conductance level, (c) finger temperature, (d) respiratory period, and (e) somatic activity. Of these five, the patterns of change evident in four variables were supportive of the physiological arousal hypothesis insofar as crying was associated with increased arousal. Only changes in the fifth variable (respiratory period) were consistent with the recovery view.

Possible Confounding by Somatic Activity
The association between crying and greater somatic activity is not surprising (e.g., the behavioral coding revealed greater face touching for criers than for noncriers). Even so, this association raises the possibility that the increased autonomic arousal, and especially the increased cardiovascular arousal, associated with crying has more to do with bodily activity than with crying per se (Obrist, 1981). To test whether the pattern of physiological findings we obtained could be explained by changes in somatic activity alone, we repeated each of the analyses reported above, using somatic activity as a covariate. These analyses of covariance yielded a pattern of results identical to those reported above. Most notably, each result consistent with the physiological arousal hypothesis remained intact: Cry × Episode interactions for heart rate ($F[1,145] = 16.75$, $p < .001$) and finger temperature ($F[1,145] = 4.58$, $p = .034$), along with the cry main effect for skin conductance level ($F[1,145] = 5.49$, $p = .02$). These findings indicate that the autonomic arousal associated

with crying cannot be attributed solely to the increased body movement that accompanied crying.

Discussion

To understand the psychophysiology of crying and to evaluate two opposing models of crying, we examined behavioral, self-report, and physiological data collected while subjects were watching a sad film.

What Happens When People Cry?
Subjects who cried during the film exhibited more expressive behavior and reported higher levels of sadness and pain than did subjects who did not cry. Also, crying was associated with increased somatic and autonomic activation.

Taken as a whole, these results favor the physiological arousal hypothesis over the physiological recovery hypothesis. Even the physiological arousal hypothesis, however, may not do justice to the full complexity of the physiological changes associated with crying. There was increased sympathetic activation during crying (e.g., decreases in finger temperature, increases in skin conductance) and increased somatic activity, decreased respiration rates, and increased parasympathetic activity (as indicated by activation of the lacrimal gland). It may be precisely this coordinated activation of parasympathetic and sympathetic branches of the peripheral nervous system—activation that goes beyond that demanded solely by changes in somatic activity—that makes crying such a potent physiological state (see Tomkins, 1963). This physiological activation, which exceeds current metabolic demands, may contribute to the experience of crying as a powerful and distressing subjective state.

Why Do People Cry?
Failure to support the homeostatic function of crying. Three mechanisms have been proposed by which crying when sad may restore homeostatic balance: (a) alleviating negative emotions, (b) ridding the body of toxins, and (c) decreasing high levels of sympathetic activation. Although our data clearly do not speak to the biochemistry of emotional tears, our findings, and those of Kraemer and Hastrup (1988), argue against the homeostatic view of crying insofar as we found crying associated with an increase in negative emotion and an increase in autonomic and somatic arousal. If crying does not immediately facilitate recovery, what functions might it serve?

One possibility is that crying really does restore homeostasis, but only over time, after a protracted period of increased arousal. Because we did not continue to measure physiological activation for long periods following the crying episode, this possibility could not be tested using data from the present study. Although our results do rule out the idea that crying is coincident with autonomic recovery, it is conceivable that crying when sad functions in a manner similar to confronting personally relevant emotional issues. In this view, short-term pain might bring long-term gain (Kenemy, 1993; Pennebaker, 1989, 1990; Pennebaker, Kiecolt-Glaser, & Glaser, 1988).

Without precluding the possibility that additional research will reveal that crying facilitates autonomic recovery in the long run, findings from our own work studying the nature of recovery from autonomic arousal over longer time periods argue against this possibility. Testing the hypothesis that a primary function of the positive emotions is to efficiently undo the arousal produced by negative emotions (Levenson, 1988), we

recently presented evidence that amusement and contentment can speed recovery from the autonomic arousal occasioned by a fear-inducing film (Fredrickson & Levenson, 1993). Although we did not measure crying, in contrast to the positive-affect films, we found that a very potent sadness-inducing film (in which a young boy is shown crying over the death of his father) did not hasten autonomic recovery.

An alternative socioemotional view of the function of crying. It seems quite possible that crying does not restore homeostasis but rather serves other important functions. Tomkins (1963) was one of the first to theorize that the biological changes associated with crying serve to communicate to both self and others that all is not well and to motivate action to alleviate crying, suffering, or both. In a similar vein, Averill (1968) proposed that the grief reaction (which may include but does not require tears) is a physiologically stressful state of activation that functions to maintain and promote group cohesion because it is aversive both to the person who cries and to others nearby. More recently, Cornelius (1984, 1988) has extended this line of reasoning by emphasizing crying's role in modifying ongoing social interactions. These theoretical accounts suggest that (a) crying is associated with high levels of autonomic activation, (b) the crier perceives this activation as subjectively painful, (c) others can perceive the crier's distress, and (d) crying is aversive to those who witness it.

The data obtained in this study directly test the first two of these assertions, and indirectly address the third and fourth.

Concerning the first two points, we found that crying is indeed associated with increased levels of autonomic (both sympathetic and parasympathetic) and somatic responding and that crying is subjectively perceived as painful. Concerning the third point, our behavioral ratings of subjects in Experiment 2 revealed that crying is associated with other visible facial behaviors in addition to tear production. This finding suggests that conspecifics have redundant sources of visible information available to enable them to perceive the crier's distress. Concerning the fourth point, our finding that viewing a film in which a woman was crying provoked our subjects to report high levels of sadness and caused a number of them to cry is supportive of the aversive quality of crying to others. This confluence of physiological and subjective changes in both the crier and the observer may well motivate conspecifics to empathic responses, including providing relief or comfort to the person who cries.

Summary. Our findings fail to lend support to the physiological recovery hypothesis, which posits that crying when sad serves to restore physiological homeostasis. Rather, our findings are consistent with the alternative physiological arousal model in which crying is associated with high levels of autonomic, somatic, and behavioral activation and is a highly negative subjective emotional experience. This aversive state has strong signal value to others and can be contagious, and we speculate that through the principle of negative reinforcement, crying may motivate proximate others to do something to end the tears, thus serving to increase the cohesion of social groups.

REFERENCES

Averill, J. R. (1968). Grief: Its nature and significance. *Psychological Bulletin, 70*, 721–748.

Beck, A. T., Rush, A. J., Shaw, B. F., & Emery, G. (1979). *Cognitive therapy of depression.* New York: Guilford.

Bindra, D. (1972). Weeping: A problem of many facets. *Bulletin of the British Psychological Society, 25*, 281–284.

Carpenter, M. B., & Sutin, J. (1983). *Human neuroanatomy* (8th ed.). Baltimore, MD: Williams & Wilkins.

Choti, S. E., Marston, A. R., Holston, S. G., & Hart, J. T. (1987). Gender and personality variables in film-induced sadness and crying. *Journal of Social and Clinical Psychology, 5*, 535–544.

Cornelius, R. R. (1984). A rule model of adult emotional expression. In C. Z. Malatesta & C. E. Izard (Eds.), *Emotion in adult development* (pp. 213–233). Beverly Hills, CA: Sage.

Cornelius, R. R. (1988, April). *Toward an ecological theory of crying and weeping.* Paper presented at the meeting of the Eastern Psychological Association, Buffalo, NY.

Darwin, C. (1872). *The expression of the emotions in man and animals.* London: Murray.

Efran, J. S., & Spangler, T. J. (1979). Why grown-ups cry: A two factor theory and evidence from *The Miracle Worker. Motivation and Emotion, 3*, 63–72.

Ekman, P., Friesen, W. V., & Ancoli, S. (1980). Facial signs of emotional experience. *Journal of Personality and Social Psychology, 39*, 1124–1134.

Fredrickson, B. L., & Levenson, R. W. (1993). *Positive emotions speed recovery from negative emotional arousal.* Manuscript submitted for publication.

Frey, W. H., II, DeSota-Johnson, D., Hoffman, C., & McCall, J. T. (1981). Effect of stimulus on the chemical composition of human tears. *American Journal of Ophthalmology, 92*, 559–567.

Frey, W. H., II, & Langseth, M. (1985). *Crying: The mystery of tears.* Minneapolis, MN: Winston.

Frijda, N. H. (1986). *The emotions.* Cambridge, England: Cambridge University Press.

Gross, J. J., & Levenson, R. W. (1993a). Emotional suppression: Physiology, self-report, and expressive behavior. *Journal of Personality and Social Psychology, 64*, 970–986.

Gross, J. J., & Levenson, R. W. (1993b). *Emotion elicitation using films.* Manuscript submitted for publication.

Gross, J. J., & Levenson, R. W. (1994). *The acute effects of emotional suppression.* Manuscript in preparation.

Heilbrun, G. (1955). On weeping. *Psychoanalytic Quarterly, 27*, 245–255.

Kenemy, M. (1993). Emotion and the immune system. In B. Moyers (Ed.), *Healing and the mind.* New York: Doubleday.

Kraemer, D. L., & Hastrup, J. L. (1986). Crying in natural settings: Global estimates, self-monitored frequencies, depression and sex differences in an undergraduate population. *Behaviour Research and Therapy, 24*, 371–373.

Kraemer, D. L., & Hastrup, J. L. (1988). Crying in adults: Self-control and autonomic correlates. *Journal of Social and Clinical Psychology, 6*, 53–68.

Labott, S. M., & Martin, R. B. (1987). The stress-moderating effects of weeping and humor. *Journal of Human Stress, 13*, 159–164.

Labott, S. M., & Martin, R. B. (1988). Weeping: Evidence for a cognitive theory. *Motivation and Emotion, 12*, 205–216.

Levenson, R. W. (1988). Emotion and the autonomic nervous system: A prospectus for research on autonomic specificity. In H. L. Wagner (Ed.), *Social psychophysiology and emotion: Theory and clinical applications* (pp. 17–42). London: Wiley.

Martin, R. B., & Labott, S. M. (1991). Mood following emotional crying: Effects of the situation. *Journal of Research in Personality, 25*, 218–244.

Obrist, P. A. (1981). *Cardiovascular psychophysiology: A perspective.* New York: Plenum.

Pennebaker, J. W. (1989). Confession, inhibition, and disease. In L. Berkowitz (Ed.), *Advances in experimental social psychology* (Vol. 22, pp. 211–244). New York: Academic.

Pennebaker, J. (1990). *Opening up: The healing powers of confiding in others.* New York: William Morrow.

Pennebaker, J., Kiecolt-Glaser, J. K., & Glaser, R. (1988). Disclosure

of traumas and immune function: Health implications for psychotherapy. *Journal of Consulting and Clinical Psychology*, *56*, 239–245.

Records, R. E. (1979). *Physiology of the human eye and visual system*. Hagerstown, MD: Harper & Row.

Sadoff, R. L. (1966). On the nature of crying and weeping. *Psychiatric Quarterly*, *40*, 490–503.

Scheff, T. (1979). *Catharsis in healing, ritual and drama*. Berkeley: University of California Press.

Stark, R. (Producer), & Ross, H. (Director). (1989). *Steel magnolias* [Film]. Burbank, CA: Columbia/Tri-Star.

Tomkins, S. S. (1963). *Affect, imagery, consciousness: Vol. 2. The negative affects*. New York: Springer.

Werb, A. (1983). The anatomy of the lacrimal system. In B. Milder (Ed.), *The lacrimal system* (pp. 23–32). Norwalk, CT: Appleton-Century-Crofts.

Williams, D. G. (1982). Weeping by adults: Personality correlates and sex differences. *The Journal of Psychology*, *110*, 217–226.