<table>
<thead>
<tr>
<th>Title</th>
<th>Effects of actual and potential stressor control on physiological and self reported stress responses</th>
</tr>
</thead>
<tbody>
<tr>
<td>Authors(s)</td>
<td>Carr, Alan; Wilde, Gerald J. S.</td>
</tr>
<tr>
<td>Publication date</td>
<td>1988</td>
</tr>
<tr>
<td>Publisher</td>
<td>Guilford Press</td>
</tr>
<tr>
<td>Link to online version</td>
<td><a href="http://pqasb.pqarchiver.com/guilford/doc/848876898.html">http://pqasb.pqarchiver.com/guilford/doc/848876898.html</a></td>
</tr>
<tr>
<td>Item record/more information</td>
<td><a href="http://hdl.handle.net/10197/5551">http://hdl.handle.net/10197/5551</a></td>
</tr>
</tbody>
</table>
VOLUME 6, NUMBER 3/4
1988

JOURNAL OF SOCIAL AND CLINICAL PSYCHOLOGY
The JOURNAL OF SOCIAL AND CLINICAL PSYCHOLOGY incorporates reports of clinical and experimental social research, invited reviews, short 'point-counterpoint' exchanges on controversial issues, book reviews, and, occasionally, autobiographical reflections regarding the seeds of important research, theory, or approaches to clinical practice.

Among the topic areas typifying the social–clinical interface addressed in the journal are close relationships; attributions, beliefs, labels, and interpersonal patterns in mental health and illness; helplessness and perceived control; emotions, nonverbal behavior, and psychopathology; social skills; stress and coping; behavioral medicine and health psychology; development of social relationships; social psychophysiology and neuropsychology; client–therapist interactions; and bias and stereotyping in social interaction and clinical practice.

EDITOR
C. R. SNYDER
University of Kansas

ASSOCIATE EDITORS
SUSAN M. ANDERSEN
New York University
MARK R. LEARY
Wake Forest University
JAMES E. MADDOX
George Mason University
TIMOTHY W. SMITH
University of Utah
THOMAS ASHBY WILLS
Albert Einstein College of Medicine

EDITORIAL BOARD
Elizabeth M. Atmaier, University of Iowa
Barbara Andersen, University of Iowa
Craig Anderson, Rice University
Robert Arkin, University of Missouri, Columbia
Robert S. Baron, University of Iowa
John Berg, University of Mississippi
Larry E. Beutler, Arizona Health Sciences Center
Paul Blaney, University of Miami
Sharon S. Brehm, University of Kansas
Steven D. Brown, Loyola University of Chicago
Thomas G. Burish, Vanderbilt University
Thomas F. Cash, Old Dominion University
June Chiiodo, Temple University
Andrew Christensen, University of California, Los Angeles
David D. Clarke, University Park, Nottingham, England
Carolyn Cutrona, University of Iowa
Valerian Derlega, Old Dominion University
Steve Duck, University of Iowa
Amerigo Farina, University of Connecticut
Guy Fielding, Dorset Institute, Dorset, England
Frank D. Fincham, University of Illinois, Urbana–Champaign
Donelson R. Forsyth, Virginia Commonwealth University
University
Frederick X. Gibbons, Iowa State University
Constance Hammen, University of California, Los Angeles
John H. Harvey, University of Iowa
Kenneth Heller, Indiana University
Clyde Hendrick, Texas Tech University
Susan S. Hendrick, Texas Tech University
William Ickes, University of Texas, Arlington

Alice M. Isen, University of Maryland, Baltimore County
Ronnie Janoff-Bulman, University of Massachusetts
Frederick H. Kanter, University of Illinois, Urbana–Champaign
Herbert M. Lefcourt, University of Waterloo
Richard Lewine, Grady Memorial Hospital, Atlanta
Richard P. McGlynn, Texas Tech University
Beth Meyerowitz, Vanderbilt University
Rowland Miller, Sam Houston State University
Louis A. Penner, University of South Florida
Richard A. Petty, Ohio State University
John Riskind, George Mason University
Daniel Russell, University of Iowa
J. Sidney Shrauger, State University of New York, Buffalo
Bonnie Spring, Texas Tech University
Melinda A. Stanley, Western Psychiatric Institute and Clinic
Cal D. Stolltenberg, University of Oklahoma
Bonnie R. Strickland, University of Massachusetts
Stanley R. Strong, Virginia Commonwealth University
William B. Swann, University of Texas, Austin
Abraham Tesser, University of Georgia
Jerzy Trzebinski, University of Warsaw
Jalie Tucker, Wayne State University
David L. Watson, University of Hawaii at Manoa
Ann Weber, University of North Carolina, Asheville
Gifford Weary, Ohio State University
Gregory L. White, University of California, Santa Cruz
Donald J. Woods, Texas A&M University
Seymour Zelien, California School of Professional Psychology

The JOURNAL OF SOCIAL AND CLINICAL PSYCHOLOGY is published quarterly by Guilford Publications, Inc., 72 Spring Street, New York, N.Y. 10012. Subscription Price: Volume 6, 1988, four issues, institutions—$75 U.S., $90 Canada and Foreign (includes airmail postage), individuals—$22.50 U.S., $37.50 Canada and Foreign (includes airmail postage). Payment must be made in U.S. dollars or at the current rate of exchange. For payment not made in U.S. currency and through a U.S. bank, add $6 service charge. Change of Address: Please inform the publisher at least six weeks prior to move. Enclose present mailing label with change of address. Claims for Missing Issues: Claims cannot be honored beyond four months after mailing date. Duplicate copies cannot be sent to replace issues not delivered because of failure to notify publisher of change of address.

Authorization to photocopy material for internal or personal use under circumstances not falling within the fair use provisions of the Copyright Act is granted by Guilford Publications, Inc., to libraries and others registered with the Copyright Clearance Center Transactional Reporting Service, provided that the fee of $2 per copy is paid directly to the Copyright Clearance Center, 21 Congress Street, Salem, Mass. 01970. The identification code for the
Two experiments were conducted in which stressor controllability was varied while stressor predictability and other stressor properties were held constant. In each experiment stressor control led to a reduction in anticipatory physiological stress. These findings support the minimax hypothesis but contravene alternative theories that attribute the beneficial effects of stressor control to the predictive information furnished by controlling actions.

A growing body of literature suggests that behavioral control over stressors probably leads to a reduction in anticipatory stress responses, and possibly minimizes stress responses at impact (Averill, 1973; Miller, 1979; Thompson, 1981). Miller (1979) has argued that explanations for the stress modifying effects of control fall into two broad categories: predictability theories and controllability theories. Predictability theories recognize that behavioral control furnishes the individual with predictive information about the stressor. This information leads to stress reduction by diminishing the uncertainty or surprise associated with the stressor (Berlyne, 1960; Sokolov, 1963), by providing relevant feedback (Weiss, 1971), or by providing safety signals (Seligman, 1968). Controllability theories, on the other hand, argue that control has stress-reducing effects independent of those
deriving from predictability. Miller's (1979) minimax hypothesis is one such theory. It states that

a person who has control over an aversive event insures having a lower maximum danger than a person without control. This is because a person with control attributes the cause of relief to a stable internal source—his own response—whereas a person without control attributes relief to a less stable, more external source (p. 294).

Critical experiments to test the comparative validity of predictability versus controllability theories would hold predictability constant and vary control across experimental and comparison groups. Of course, the properties of the stressor and the amount of motor activity engaged in by subjects would also have to be equated across conditions. Miller (1979) has drawn attention to two experimental paradigms that meet these requirements—the actual control equated for predictability paradigm, and the potential control paradigm. Since (as Miller's, 1979, review highlights) few experiments that employ these paradigms have previously been conducted, these paradigms formed the basis for the experiments reported in this paper. A primary goal of these two experiments was to test the minimax hypothesis.

EXPERIMENT 1

In this experiment the actual control equated for predictability paradigm was used (Miller, 1979). In this paradigm subjects in the experimental group exercise actual control over a stressor, whereas their comparison group counterparts do not. Actual control may involve altering the duration, intensity, or other characteristics of the stressor, or the probability of the stressor's occurrence. In the experiment reported below, subjects with actual control reduced stressor duration. The amount or predictive information about the stressor available to subjects, the physical characteristics of the stressor (such as intensity or duration), and the amount of motor activity engaged in by subjects are held constant across conditions in this paradigm. Thus intergroup differences on dependent variables may be attributed to the variable "actual control" alone.

METHOD

SUBJECTS

For this experiment 44 subjects were recruited on a volunteer basis from various institutes of higher education in Kingston, Ontario; 22 were male and 22 female. Ages ranged from 18 to 36 years (M = 21.95, SD = 3.22). An equal number of males and females were randomly assigned to experimental and comparison groups. Prior to the experiment proper, all subjects completed a brief screening interview and were administered a hearing test. In no case was there evidence of hearing deficits. Subjects were paid $5 for their participation in the experiment.

APPARATUS

Stimulus materials. A Revox two-channel tape recorder (type A77) was used for both recording and presenting the stimulus materials. The beginning of the orientation trial and each of the six experimental trials were signaled by 5-second, 74 dB, 500-Hz tones. In each case this was followed by 3 minutes and 10 seconds of silence. Depending on the experimental conditions, a trial could end with either a 10-second or a 3-second burst of 100 dB white noise. On track one of the stimulus tape, all trials ended with a 10-second burst of noise. A 3-second burst of noise concluded each of the trials on track two of the tape. In addition to these stimulus materials, stimuli for two demonstration trials were also recorded. Each of these trials began with a 5-second, 70 dB, 500-Hz tone. This was followed by a 30-second period of silence. Each of these trials ended with an 86 dB, 10-second burst of white noise on track one, and an 86 dB, 3-second burst of white noise on track two.

All stimulus materials were presented over a loudspeaker mounted 1 meter in front of the subject in the experimental chamber. Sound levels of stimuli were set according to sound level meter readings taken from a position directly in front of the loudspeaker at a distance of 1 meter. A subject in the experimental condition could change the tape recorder output from track one to track two by pressing an illuminated red button. This button was mounted beside each subject's dominant hand on the table top in front of him or her. A wire connected the button to an electrical interfacing panel in front of the subject. Thus it was obvious to all subjects that the button was connected to some equipment in the control room. When they pressed this button, the light inside it extinguished. In this way they were given feedback that their stressor controlling actions were effective. Red and green illuminated buttons were mounted on top of the tape recorder in a room adjacent to the experimental chamber. When a subject depressed the track changing button, the red light on top of the tape recorder extinguished and the green light came on. This allowed the experimenter to monitor subjects' button-pressing behavior. Furthermore, at the beginning of each trial, the experimenter changed the stimulus tape to track one by depressing the red button on
top of the tape recorder. This also caused the button mounted on the table in front of the subject to light up.

In the comparison condition a button, identical in appearance to that employed in the experimental condition, was mounted on the table top in front of the subject beside his or her dominant hand. However, this button, unlike that in the experimental condition, was very obviously not connected to the tape recorder in the adjoining room. No wire connected this button to the electrical interfacing panel in front of the subject. A battery inside the button’s mounting box powered a bulb that could be illuminated or extinguished by pressing the button.

**Physiological recording apparatus.** Throughout the experiment, electrodermal and cardiovascular activity were monitored on a Beckman Dynograph recorder (type R611). Beckman silver-silver chloride electrodes, Beckman electrode collars, and Beckman electrode paste were used for all physiological recordings. Effective electrode sites using this equipment were circular and of 1 cm diameter. Electrodes were placed on the ventral surfaces of the middle phalanges of the first and second fingers of the nondominant hand to monitor skin resistance. For heart rate, electrodes were placed one on each side of the neck, posterior to the sternomastoid muscle, just below the ear. The third heart rate electrode was placed on the midforearm ventral surface of the nondominant limb. The electrode on the nondominant forearm and that on the dominant side of the neck served as the active leads. The remaining electrode served as the reference lead.

Electrodermal activity was recorded as skin resistance on two channels using a 9892A coupler connected to an AC 9806A coupler. This permitted a highly sensitive and a moderately sensitive record of electrodermal activity. Thus small spontaneous skin resistance responses during the anticipation period, and large skin resistance responses to the noise stimuli could accurately be measured. Cardiac activity was measured as heart beats using an AC 9806A coupler. This was connected to a 9857 cardiotachometer coupler. Output from this coupler was used to assess heart rate variability (see below).

**Self-report instruments.** The Desirability of Control Scale (Burger & Cooper, 1979) and the Eysenck Personality Inventory (Eysenck & Eysenck, 1965) were administered to all subjects. The Desirability of Control Scale is a 20-item instrument that assesses the extent to which individuals seek to control events in their day-to-day lives.

In order to continuously monitor subjects’ subjective states of tension or relaxation during the anticipation periods of each trial, the Self-Reported Tension Scale was administered to all subjects. When this scale was administered subjects were required to spontaneously report any changes in subjective state along the relaxation-tension dimension for the duration of each anticipation period. After each noise burst subject were asked to give a rating, relative to their anticipation period rating, to describe their subjective state during the noise. The Self-Reported Tension Scale was adapted from an instrument designed to measure fluctuations in perceived risk in automobile drivers (Browning & Wilk 1977; Moran, 1982).

After each trial all subjects were asked to complete two copies of the Stress Arousal Checklist. These instruments offered a further assessment of subjects’ subjective states during the anticipation and impact periods of the trial. The Stress Arousal Checklist is a 34-item adjective checklist that yields separate stress and arousal scores (Mackay et al. 1978).

**Laboratory setting.** Throughout the experiment subjects were seated in an electrically shielded room. In order to minimize movement artifacts in the physiological records, subjects sat in a comfortable but rigid chair. An adjustable footrest was secured to the legs of the chair, and a shaped table top was attached to the arms of the chair after each subject was seated. The illuminated buttons were mounted on this table top.

The tape recorder from which the stimulus materials were presented and the physiological recording apparatus were housed in a room adjacent to the experimental chamber. An intercom system between these two rooms allowed the experimenter to monitor the subjects’ verbal reports of tension during the experimental trials. These reports were written directly onto the polygraph paper record.

**PROCEDURE**

After the screening interview, hearing test, and attachment of the electrodes, subjects were seated in the experimental chamber. Here the completed the Desirability of Control Scale and the Eysenck Personality Inventory while the experimenter adjusted the physiological recording apparatus in the adjoining room.

The orientation trial instructions were then read to each subject. These instructions informed subjects that following a tone that signaled the beginning of the trial, they were to rate their experienced level of tension or relaxation using the Self-Reported Tension Scale until the occurrence of a 10-second burst of loud white noise. After the trial subject were asked to rate their subjective state during the noise burst on the Self-Reported Tension Scale. They were also asked to complete two copies of the Stress Arousal Checklist to furnish an assessment of their states during the anticipation and impact periods.
The experimental instructions proper were then read. Subjects in the experimental condition were told that pressing the illuminated button after the onset of the noise would shorten the duration of the noise burst from 10 seconds to 3 seconds. They were also informed that when they pressed the button, the light inside it would extinguish. This, they were told, was to be interpreted as feedback concerning the effectiveness of their response. They were asked to exercise this form of stressor control in each of six trials. However, before beginning these trials they were given two brief trials during which they could choose to press or not press the button. In this way the controlling power of the button was demonstrated to them.

Subjects in the comparison condition were told that they would have no control over the 3-second burst of noise to which they would be exposed at the end of each of six trials. They were asked, however, to turn off the light inside an illuminated button by pressing it. This button was clearly not connected to the source of the noise. They were told that this activity might serve as a distraction from the noise burst, but that such distraction was unlikely to be effective in reducing their response to the noise. Before the six experimental trials these subjects were given two brief trials during which they could choose to press or not to press the button. In this way the independence of the button from the source of the noise was demonstrated.

Subjects then underwent six experimental trials. The procedure for completing the self-report instruments during each experimental trial was the same as that for the orientation trial.

After the sixth experimental trial two minutes of baseline data were collected. During this period subjects were required to relax.

Thereafter the electrodes were removed, and a postexperimental interview was conducted. A brief explanation of the aims and design of the study was then presented.

DATA REDUCTION

In coding continuously monitored physiological and self-report variables, the initial 10 seconds of each trial (which followed the signal tone) were disregarded. Consequently, responses to the tone that signaled trial onset were not included in anticipation period scores. Separate scores were derived for each of the remaining 3 minutes on each of the continuously monitored variables. This data coding system permitted the exploration of anticipation curves as a function of the stressor control.

STRESSOR CONTROL

Data from the orientation trial and six experimental trials were coded in a similar manner. Data from the baseline period were coded in the same way as data from the anticipation periods.

Spontaneous skin resistance responses were defined as decreases in skin resistance equal to or greater than 400 ohms occurring during the anticipation period. The number of such responses in each of the 3 minutes of each anticipation period was determined and coded. The amplitude of skin conductance responses to the noise bursts were calculated by subtracting the immediate prestimulus levels from the post-stimulus peak values after they had been converted to units of conductance.

Heart rate variability was measured using a simplified version of Kalsbeek's (1971) technique. Changes in heart rate, on the cardiotachometer record, of more than six beats per minute were given a score of one. By summing these scores, a heart rate variability score was derived for each of the 3 minutes of the anticipation period in each trial. Phasic heart rate responses of the noise were calculated by subtracting the number of heart beats in the 10-second period preceding the onset of the noise from the number of heart beats in the 10-second period following the noise onset.

Each subjects' status on the Self-Reported Tension Scale was sampled at 10-second intervals. Thus for each minute in each anticipation period, six readings were obtained. The average of these six readings was coded as the self-reported tension score for that minute. Self-reported tension during the noise was coded as the single score on the Self-Reported Tension Scale obtained directly from the subject following the noise burst at the end of each trial.

Self-reported stress and arousal scores for the anticipation and impact periods were derived from subjects' responses to the Stress Arousal Checklist.

After they had been coded and verified, all of the variables outlined above were transformed using Rose's (1964) range correction procedure, or Lykken's (1972) modification of this transformation. Skin conductance and heart responses at impact were transformed using Lykken's formula. Rose's original procedure was used to transform the remaining variables. When the data had been range-corrected, all values were multiplied by 100. Thus all variables (with the exception of heart rate variability) were now on scales with a range of 100, where 1 represented a state of low arousal, stress, or tension and 100 represented an elevated state on these variables.

A decrease in heart rate variability represents a state of increased arousal and an increase in heart rate variability reflects a state of relaxation (Kalsbeek, 1971). To change the direction of heart rate variability scores
to that of the other variables, the range-corrected heart rate variability scores that had been multiplied by 100 were subtracted from 100.

To take account of the phenomena of situational stereotypy (Lacey, 1967) and response specificity (Engle, 1972), composite physiological stress scores were derived from the cardiovascular and electrodermal activity data for both the anticipation and impact periods. (These composite scores were similar to Thayer's, 1970, index A.)

The anticipatory physiological stress variable was derived by choosing for each subject, for each minute in each trial, that physiological variable with the greatest range-corrected value. For example, if a subject in minute 1 of trial 1 obtained a heart rate variability score of 30, and a score of 60 on the frequency of spontaneous skin resistance responses variable, his or her anticipatory physiological stress score would be 60. The physiological stress at impact variable was derived by choosing for each subject, for each impact period in each trial, that physiological variable with the greatest value. For example, if a subject in trial 1 obtained a skin conductance response amplitude score of 50 and a heart rate response score of 67, he or she would obtain a physiological stress at impact score of 67 for that impact period. In summary, the data reduction procedure yielded the following eight dependent variables:

1. Anticipatory physiological stress
2. Anticipatory self-reported tension
3. Anticipatory self-reported stress
4. Anticipatory self-reported arousal
5. Physiological stress during the impact period
6. Self-reported tension during the impact period
7. Self-reported stress during the impact period
8. Self-reported arousal during the impact period

RESULTS

An initial analysis was conducted to check that the stimuli used in the experiment were stress inducing. The self-report data contained in Table 1 shows that subjects experienced anticipation periods as less stressful than impact periods. Significance levels for each of the three self-report variables contained in the table are based on t-tests for correlated samples. (Since physiological stress was assessed in different ways during the anticipation and impact periods, and since data were range-corrected separately for each period, meaningful comparisons of anticipation and impact physiological data could not be made.)

Postexperimental interviews revealed that the independent variable was effectively manipulated. All subjects in the experimental group reported having experienced actual control over stressor duration, whereas their comparison group counterparts did not.

Reliability analyses were conducted on the anticipatory physiological stress variable, the physiological stress at impact variable, and the Self-Reported Tension Scale. For these analyses each minute of the anticipatory periods and/or each burst of noise during the impact periods were viewed conceptually as items (albeit items with different difficulties) in a scale. Cronbach's alpha reliability coefficients calculated on this basis, therefore, reflect the consistency of subjects' scores on the variable in question across time periods. Coefficients of .94 and .81, which indicate an acceptable level of reliability, were obtained for the Self-Reported Tension Scale and the anticipatory physiological stress variable, respectively. For the physiological stress at impact variable, an alpha value of .40 was obtained. The poor reliability of this measure argues for a cautious interpretation of the ANOVA results for this variable, since the likelihood of type I error is increased. Real differences may go undetected.

T-tests indicated that the experimental and comparison groups did not differ reliably (p > .1) on a number of organismic variables—age (t (42) = 0.3), desirability of control (t (42) = 0.2), neuroticism (t (42) = 0.8), and extraversion (t (42) = 1.5).

Two × 6 × 3 (control × trials × minutes) one between factor, two within factors ANOVAs were run on the anticipatory physiological stress and anticipatory self-reported tension variables. Other 2 × 6 (control × trials) one between factor, one within factor ANOVAs were run on the remaining six dependent variables. The only reliable intergroup difference
occurred on the anticipatory physiological stress variable, $F(1, 42) = 7.74, p = .008)$. The group with actual control over the stressor displayed a lower mean level of anticipatory physiological stress than the comparison group (see Table 1).

Statistically significant trials, minutes, and trials by minutes effects were present in the analyses. These effects reflect habituation across trials, change in the slope of the anticipatory responses curves, and the interaction between these two phenomena. Since these effects are not pertinent to the hypotheses being tested, they are not considered further. No significant interactions between actual control and either trials or minutes were observed.

**EXPERIMENT 2**

Experiment 1 was concerned with the effects of actual (or exercised) control. In Experiment 2 the effects of potential (or unexercised) control were examined. Miller (1979) has described a paradigm for determining the effects of potential control. Subjects in the experimental group are exposed to a stressor but are made aware that a controlling action is available that can lessen the impact of the stressor (should it become intolerable). However, social pressure is brought to bear on these subjects so that they do not execute the controlling action. Their comparison group counterparts have no potentially controlling action available. Stressor characteristics are held constant across conditions. In experiments that employ this paradigm, intergroup differences on dependent variables may be attributed to potential control alone, since the amount of predictive information available to subjects and stressor characteristics are constant across conditions while potential control is varied.

**METHOD**

**SUBJECTS**

For this experiment 44 subjects were recruited on a volunteer basis from various institutes of higher education in Kingston, Ontario; 22 were male and 22 female. Subjects' ages ranged from 16 to 31 years ($M = 21.05, SD = 2.59$). An equal number of males and females were randomly assigned to experimental and comparison groups. Prior to the experiment proper, all subjects completed the screening interview and hearing test noted previously. In no case was there evidence of hearing deficits. Each subject was paid $5 dollars for participating in the experiment.

**STRESSOR CONTROL**

**APPARATUS**

With the exception of the modifications outlined below, the apparatus for this experiment was the same as that used in Experiment 1.

In the experimental condition a red panic button was mounted beside each subject's dominant hand on the table in front of him or her. The button was connected to the tape recorder from which the stimulus materials were presented. Pressing the button caused the tape recorder to stop.

In the comparison condition no buttons or other instruments were available to subjects.

In both experimental and comparison conditions, only track one of the stimulus tape was used, that is, the duration of the noise bursts at the end of each trial in both conditions was invariably 3 seconds.

**PROCEDURE**

With the exception of the experimental instructions proper, all aspects of the procedure in this experiment were the same as those in Experiment 1.

In the experimental condition subjects were informed that there would be six experimental trials, and that each trial would end with a 3-second burst of loud white noise. They were told that if the noise became unbearable, they could press the red panic button and the noise would stop. However, they were asked to endure as much noise as they could. (In fact no subject ever used the panic button during the experimental trials.) Before the experimental trials these subjects were given two brief demonstration trials during which they could test the effectiveness of the panic button in stopping the noise. In this way the controlling power of the button was demonstrated.

In the comparison condition subjects were informed that they would have no control over the burst of noise that would occur at the end of each of six experimental trials. Before these trials, subjects were administered two brief demonstration trials. They were informed that these would familiarize them with the procedure.

**DATA REDUCTION**

The same procedures for data reduction and analysis were employed in this experiment as were used in Experiment 1.
RESULTS

The analyses for the potential control data took the same form as those for the actual control data, outlined above.

The self-report data set out in Table 2 show that the impact periods were experienced as more stressful than the anticipation periods.

Postexperimental interviews indicated that the independent variable was effectively manipulated.

As in Experiment 1, reliability analyses were conducted. Cronbach’s alphas of .95 and .83 were obtained for the Self-Reported Tension Scale, and the anticipatory physiological stress variable, respectively, indicating acceptable levels of reliability for these measures. The obtained physiological stress at impact reliability coefficient of .22 was low enough to argue for a cautious interpretation of ANOVA results for this variable because of the increased probability of type 11 error.

On the following organismic variables, no reliable ($p > .1$) intergroup differences were found: age ($t (42) = 0.2$), desirability of control ($t (42) = 0.5$), neuroticism ($t (42) = 0.5$), and extraversion ($t (42) = .7$).

The experimental and comparison groups differed reliably only on one dependent variable—anticipatory physiological stress, $F (1, 42) = 5.78$, $p = .02$. Subjects with potential control displayed lower scores than subjects without such control on this variable.

As in Experiment 1 a number of significant trials, minutes, and trials by minutes effects were observed that have no bearing on the hypotheses being tested, and so are not discussed further.

### ANCILLARY ANALYSES

A statistical issue deserving comment is the use of composite indices of physiological stress during the anticipation and impact periods. These indices take account of both situational stereotypy and response specificity with a minimum of assumptions. There are two other major alternatives to handling these sort of data so as to take account of these phenomena. The first is to assess each subject’s most reactive physiological response channel before the experiment proper, and then base his or her physiological stress scores within the experiment on this variable alone. This procedure effectively takes account of individual response specificity, but assumes that such specificity outweighs situational stereotypy effects. (Although rarely used in laboratory experiments on groups of subjects, this approach is commonly used by clinicians with individual clients.)

A second alternative is to assume that response specificity has been controlled for through random assignment of subjects to groups, and then to conduct separate analyses on data from each physiological response channel. Information on situational stereotypy may be derived by identifying those physiological variables on which groups in different experimental conditions (or situations) reliably differ. A problem with this approach is that in small group research ($n < 100$) it may be inappropriate to assume that random assignment controls for response specificity. Thus observed intergroup differences on physiological variables may be interpreted in at least two ways: (1) they may be taken to mean that particular types of situations lead to changes on particular physiological variables, or (2) that, despite random assignment, the subjects in the two groups differ in the types of physiological response patterns they typically display. Although this approach has its shortcomings, it is a common way for dealing with data from laboratory experiments in this field.

So that the data from Experiments 1 and 2 could be compared with similar studies in the literature, separate analyses were conducted on the following untransformed and range-corrected psychophysiological variables: anticipatory heart rate variability, the frequency of spontaneous skin resistance responses during the anticipation period, heart rate change at impact, and the amplitude of the skin resistance response at impact. The analyses took the same form as those detailed in the Results sections. Means and standard deviations derived from the range-corrected data for Experiments 1 and 2 are presented in Table 3.

In both experiments Cronbach’s alphas in excess of .80, indicating acceptable levels of reliability, were obtained for all anticipatory period variables. Impact period variables yielded reliability coefficients between .35 and .55. In both studies, therefore, these measures displayed poor reliability.

### TABLE 2

<table>
<thead>
<tr>
<th></th>
<th>POTENTIAL CONTROL</th>
<th>NO CONTROL</th>
<th>IMPACT PERIOD</th>
<th>POTENTIAL CONTROL</th>
<th>NO CONTROL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physiological stress</td>
<td>M 64.0*</td>
<td>71.7*</td>
<td>71.4</td>
<td>73.5</td>
<td></td>
</tr>
<tr>
<td>SD 21.4</td>
<td>23.1</td>
<td></td>
<td>27.1</td>
<td>28.7</td>
<td></td>
</tr>
<tr>
<td>Self-reported tension</td>
<td>M 30.0*</td>
<td>36.3*</td>
<td>75.1*</td>
<td>77.0*</td>
<td></td>
</tr>
<tr>
<td>SD 21.2</td>
<td>24.3</td>
<td></td>
<td>22.0</td>
<td>21.5</td>
<td></td>
</tr>
<tr>
<td>Self-reported stress</td>
<td>M 44.1*</td>
<td>51.8*</td>
<td>71.5*</td>
<td>80.6*</td>
<td></td>
</tr>
<tr>
<td>SD 28.6</td>
<td>30.1</td>
<td></td>
<td>24.1</td>
<td>21.2</td>
<td></td>
</tr>
<tr>
<td>Self-reported arousal</td>
<td>M 40.2*</td>
<td>45.3*</td>
<td>80.9*</td>
<td>84.2*</td>
<td></td>
</tr>
<tr>
<td>SD 26.2</td>
<td>26.6</td>
<td></td>
<td>17.4</td>
<td>17.0</td>
<td></td>
</tr>
</tbody>
</table>

*Note. In each row scores marked by * differ reliably ($p < .05$) from scores marked by *.
TABLE 3
Means and Standard Deviations of Range-Corrected Electrodermal and Cardiovascular Variables during Anticipation and Impact Periods in Experiments 1 and 2

<table>
<thead>
<tr>
<th>EXPERTMENT 1</th>
<th>EXPERTMENT 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACTUAL CONTROL</td>
<td>NO CONTROL</td>
</tr>
</tbody>
</table>

**ANTICIPATORY PERIOD**

- Frequency
  - Mean: M (Experiment 1) 32.90\(^a\), M (Experiment 2) 43.51\(^b\)
  - SD: 25.53, 26.18, 23.35, 28.75
- Heart rate variability
  - Mean: M (Experiment 1) 57.69\(^a\), M (Experiment 2) 68.26\(^b\)
  - SD: 26.05, 24.46, 25.48, 27.97

**IMPACT PERIOD**

- Amplitude of SCR
  - Mean: M (Experiment 1) 57.23, M (Experiment 2) 55.45
  - SD: 2.86, 3.08, 2.64, 3.63
- Change in heart rate
  - Mean: M (Experiment 1) 43.16\(^a\), M (Experiment 2) 58.21\(^b\)
  - SD: 38.40, 58.64, 51.91

**Note.** For each experiment in each row scores marked by \(^a\) differ reliably (p < .05) from scores marked by \(^b\). For anticipatory period variables, mean scores per minute are given.

**DISCUSSION**

Main effects for stressor control in the ANOVAs on anticipatory heart rate variability, F (1, 42) = 7.32, p < .01, the frequency of skin resistance responses during the anticipatory period, F (1, 42) = 6.07, p < .05, and heart rate change at impact, F (1, 42) = 7.58, p < .01 were observed in Experiment 1. In Experiment 2 anticipatory heart rate variability was the only main effect for stressor control that approached significance, F (1, 42) = 3.69, p < .10. These four findings support the view that stressor control leads to a reduction in stress responses and are therefore in accord with the results of the main analyses.

The statistics based on untransformed data presented in Table 4 convey the magnitude of the initial raw scores for anticipatory and impact physiological variables. When ANOVAs were conducted on these data (which have not been transformed so as to reduce variance due to individual differences in response range), only one significant result emerged. In Experiment 1 the group with actual control displayed more heart rate variability (indicating less stress; Kalsbeek, 1971) than their comparison group counterparts, F (1, 42) = 9.19, p < .01.
high intersubject variability in the patterning of responses across trials. That is, the habituation curves (representing scores on physiological variables at impact across six trials) varied greatly from subject to subject. This was not the case for physiological measurements taken during the anticipatory period.

The results of the main analyses of the two experiments may be summarized as follows. In Experiment 1 it was found that actual control led to a reduction in anticipatory physiological stress. In Experiment 2 potential control was shown to have a similar effect. Neither form of control appears to have reduced physiological stress at impact (although the reliability of this index was too low to allow firm conclusions to be drawn). Self-report indices of tension, stress, and arousal during both anticipation and impact periods were unaffected by the presence of either actual or potential control.

The single positive finding in each experiment supports the minimax hypothesis (Miller, 1979), which entails the view that stressor control leads to a reduction in anticipatory stress, but fails to support the predictability theories (Berlyne, 1960; Seligman, 1968; Sokolov, 1963; Weiss, 1971).

The information-seeking theory is the only position that entails a hypothesis concerning stress responses at impact (Berlyne, 1960; Miller, 1979; Sokolov, 1963). It argues that if the amount of predictive information is the same across experimental and comparison groups, then no intergroup differences in stress at impact will be observed. This position was supported by the physiological and self-report results from both experiments. Miller (1979) has suggested that the minimax hypothesis might be extended to cover the stressor impact situation by arguing that when subjects have control they experience less anticipatory stress, and against this background of relative relaxation aversive stimuli lead to less stress at impact. The impact results from Experiments 1 and 2 do not support this extended version of the minimax hypothesis.

None of the theories tested by these experiments can account for the desynchrony (Hugdahl, 1981) between anticipatory, physiological, and self-report data. A number of possible hypotheses may be offered to account for this observed desynchrony (Hodgson & Rachman, 1974). It may be that stressor control specifically modifies physiological responses. It is also possible that the desynchrony may be a function of the quality, frequency, intensity, duration, or other characteristic of the stressor, or the duration of the anticipation period. The clarification of this issue is a worthwhile goal for future research in this area. Ultimately, theories of stress and control, such as the minimax hypothesis, must more precisely articulate predictions about dependent variables that fall under the general rubric of "stress."

---

**REFERENCES**


