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PSYCHOPHYSIOLOGICAL RESEARCH ON ANGER

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Resumen: Este artículo se centra específicamente en la emoción de la ira y en el estudio de las respuestas psicofisiológicas asociadas a esta emoción básica. El presente estudio se diseñó con el objeto de comparar las respuestas cardiovasculares durante una tarea de afrontamiento activo y la emoción de ira, manipulando independiente estas dos condiciones. Para evaluar los mecanismos hemodinámicos de la reactividad cardiovascular durante la tarea de afrontamiento activo y la ira se utilizó la cardiografía de impedancia. Participaron en el estudio 63 estudiantes varones de entre 20 y 51 años. Durante el primer período de tratamiento, un grupo se dedicó a una tarea de esfuerzo de afrontamiento activo (tarea de aritmética mental) y otro grupo no (tarea de lectura). Durante el segundo período de tratamiento, ambos grupos se dividieron de dos subgrupos, la mitad de los sujetos de cada grupo fueron sometidos a provocación. La conclusión principal de los resultados del presente estudio es que la ira tiene un notable impacto sobre el funcionamiento cardiovascular. El estudio de otros parámetros cardiovasculares mediante la cardiografía de impedancia contribuyen al conocimiento de los mecanismos implicados en el aumento diferencial de las presiones sanguíneas.

Palabras Clave: Emociones, Ira, Investigación psicofisiológica y actividad cardiovascular

Abstract: In this article we will focus on the specific emotion of anger and the role of psychophysiological responses associated with this basic emotion. The present study was designed to compare cardiovascular responses during effortful active coping and the emotion of anger and to independently manipulate these two conditions. To assess the hemodynamic mechanisms of cardiovascular reactivity during active coping and anger impedance cardiography was introduced. Sixty-three male students, 20-51 years, participated in this study. During the first treatment period, one group worked on an effortful active coping task (mental arithmetic) and the other group did not (reading task). During the second treatment block, the two groups of the first treatment period were divided into two subgroups, and half of the subjects each were additionally provoked. Results of the present study show profound effect of anger on cardiovascular functioning. The additional measures of various cardiovascular parameters by means of impedance cardiography contribute to the understanding of the mechanisms involved in differential blood pressure increases.

Key words: Emotions, Anger, Psychophysiological research, Cardiovascular activity

Titulo: *Investigación psicofisiológica de la ira*

Introduction

Biobehavioral research has increasingly recognized the importance of emotions for survival and human adaptation. Emotions are characterized as being evoked by biologically relevant stimuli and by associated internal or external events. Emotions mobilize attentional and cognitive resources directed toward coping with challenging environmental circumstances (Cacioppo, Klein, Berntson & Hatfield, 1993; Plutchik, 1980). A central issue is that emotions imply an evaluation of the environment. This is connected with an evaluative categorization of stimuli into stimuli with positive or negative valence. Furthermore, evaluations involve an anticipation what situational cues might require, e.g., in one situation preparation for flight (experienced as fear), or under other circum-

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stances physical attack (experienced as anger). Such emotional structures are anchored in the somatic nervous system and have evolved on the basis of subcortical structures. Especially when they are intense or extended across time, they entail support of the autonomic nervous system (Cacioppo et al., 1993). In this article we will focus on the specific emotion of anger and the role of psychophysiological responses associated with this basic emotion.

Anger is an emotional experience arising when we feel injured by others (Averill, 1982; Hodapp & Schwenkmezger, 1993). Starting from the generally accepted view that emotions form complex sequences of reactions, several components of the emotion of anger have to be differentiated. These components involve cognitive evaluations, subjective experiences, physiological changes, and action tendencies in anticipation of responding to significant environmental challenges (Frijda, 1986; Scherer, 1984). There is an overlap between the emotion of anger and related constructs like aggression or hostility. While anger is considered to be an emotional construct, aggression generally implies destructive or punitive behavior directed to other persons or objects. Although hostility usually involves angry feelings, this concept has the meaning of a complex set of attitudes that motivate aggressive behaviors directed toward injuring other people (Spielberger et al., 1985). Unfortunately, the research literature on anger, hostility, and aggression reveals a great deal of conceptual ambiguity and confusion. Terms are defined in different and even sometimes contradictory ways. Although hostile attitudes are associated with anger and aggressive behavior, it appears to be suitable to differentiate the components of the "AHA! Syndrome" (Anger, Hostility, and Aggression; Spielberger et al., 1985) and to restrict anger to the emotional component of this syndrome.

The question if emotions are accompanied by specific autonomic nervous system activity has been the source of much controversy. The study of the physiology of anger began with Cannon who described the "flight/fight" response involving blood pressure and heart rate increases, skeletal muscle vasodilatation, visceral constriction, and biochemical changes associated with energy mobilization (Cannon, 1953). Cannon's notion of an unidimensional arousal dimension has been challenged by the observation that undifferentiated autonomic arousal during emotional states may lead to discrete emotional experiences. Fear and anger obviously differ in the phenomenology of emotional experience. Mandler (1975) and Schachter and Singer (1962) further theorized that discrete emotional experiences may derive from cognitive appraisal processes initiated by the perception of undifferentiated physiological arousal but which is attributed to different situational cues.

An alternative position stems from evidence supporting the view of a physiological differentiation in emotion. This tradition goes back to William James's (1884) famous article where he asserted that emotional feelings are consequences rather than antecedents of peripheral physiological changes elicited by external stimuli. There is no place to extensively discuss implications of James's theory and attempts to link James's position with contemporary concepts (see Lang, 1994). However, several studies demonstrated qualitative differences in autonomic and somatic responding in different emotional states supporting theories which hold that discrete emotional experiences stem from distinct somatovisceral patterns (e.g., Levenson, Ekman & Friesen, 1990).

One of the first studies conducted in this area was the study of Ax (1953). He induced the emotions of fear and anger and

compared the physiological patterns associated with these emotions. Fear was induced by simulating dangerous manipulations with the polygraph. In the condition of anger a technical assistant provoked the subjects claiming their non-cooperative behavior. In seven of fourteen physiological parameters significant differences between the conditions could be observed. In the anger condition stronger increases in diastolic blood pressure, the number of galvanic skin responses, and muscle tension were shown. Furthermore, during anger there were marked decreases in heart rate. In the condition of anxiety, the level of skin conductance, the number of muscle tension peaks, and respiratory frequency was increased compared to the anger condition.

Similar results were received by Schachter (1957) who added pain produced by the cold pressure test to the design. The most markant differences were found in the cardiovascular system. Similarly to Ax, Schachter explained the different cardiovascular reactions by different proportions of the catecholamines epinephrine and norepinephrine. Epinephrine generates a general arousal reaction with an increase in systolic blood pressure and stroke volume accompanied by vasodilatation in the skeletal muscular system. On the contrary, norepinephrine leads to vasoconstriction in the skeletal muscles associated with an increase in diastolic blood pressure.

While in the classical studies on the question of autonomic specificity in emotion mostly only differences between fear and anger have been investigated, and emotions were induced by situational manipulations, other authors expanded the range of emotions and examined cardiovascular patterns following affective imagery. Humans have the capacity to visualize affective arousing situations and they respond to their internal images as an emotionally significant event. Conceiving emotions as integrated "perceptual-motor sets" (Lang, 1979), this method appears to be especially suitable to reliably generate various emotional states. Schwartz, Weinberger and Singer (1981) asked their subjects to visualize an affective arousing situation. The study provided evidence that emotional responses of happiness, sadness, anger, and fear each evoked specific, biologically meaningful, patterns of cardiovascular adjustment. It is interesting to see that anger, the typical "fight" response, led to the greatest overall increases in cardiovascular measures and was distinctly opposite from relaxation. Anger had a more profound effect on cardiovascular functioning than any other emotion. In comparison to fear, anger produced significantly greater increases in diastolic and mean arterial pressure which was replicated several times (Ax, 1953; Ekman, Levenson & Friesen, 1983; Funkenstein, King & Drolette, 1954; Hodapp, Bongard, Heinrichs & Oltmanns, 1993; Roberts & Weerts, 1982; Schachter, 1957). Anger was also associated with strong increases in systolic pressure although systolic pressure was not different during fear and anger. Regarding a relatively low heart rate, the authors consistent with previous studies emphasize the role of peripheral vasoconstriction in the pressor response in anger (Schachter, 1957). There is evidence from several studies (cf., Schwartz et al., 1981) that vasoconstriction and increased diastolic blood pressure are associated with increased isometric muscle strength, vigilant sensory intake, and protection against the acute effects of hemorrhage – a pattern whose biological function resembles demands during physical fight.

While the study of Schwartz et al. (1981) used only systolic and diastolic blood pressure and heart rate as parameters of cardiovascular functioning, more recent investigations derived cardiovascular response patterns non-invasively by imped-

ance cardiography (Sherwood, 1993). A major advantage of this non-invasive and unobtrusive technique for measuring cardiac function is to assess the hemodynamic mechanisms underlying differential blood pressure changes. Sinha, Lovallo, and Parsons (1992) examined these mechanisms governing pressor responses during various emotions. Heart rate, blood pressure, stroke volume, peripheral vascular resistance, cardiac output and indices of myocardial contractility were measured during fear, anger, joy, sadness, physical activity, and neutral imagery conditions. Again, anger imagery, rather than fear, was accompanied by the largest effects on the cardiovascular system. Increased diastolic blood pressure was associated with maintained levels of peripheral vascular resistance and increased cardiac output and heart rate. Thus, the underlying pattern of cardiovascular activation appears to differ between the major classes of emotions. The question arises if these results of Sinha et al. (1992) may be extended to a real-life anger provoking situation, and which relationships between the cardiovascular response patterns activated during anger and other emotional or motivational states do exist?

Among situational conditions that produce excessive activation of the sympathetic nervous system, effortful active coping is one of the most prominent issue. Active coping tasks (e.g., reaction time tasks, mental arithmetic) produce cardiovascular responses characterized by activation effects on heart rate, systolic blood pressure, and contractility parameters with smaller effects on diastolic blood pressure and vascular resistance. Although reviews (e.g., Fredrikson & Matthews, 1990) came to the conclusion that excessive activity of the sympathetic nervous system during active behavioural stress contributes to pathophysiological development and to chronic elevated blood pressure, it also may be argued that enhanced sympathetic activation during effortful active coping may be quite adaptive because it allows for an efficient and speedy distribution of resources throughout the body necessary, e.g., to improving task performance (Dienstbier, 1989; Tomaka, Blascovich, Kelsey & Leitten, 1993). Some authors argued that enhanced cardiovascular activation is of no etiological relevance as long as the enhanced blood flow can be compensated for by a decrease in vascular resistance. Only cardiac activation that is accompanied by simultaneous vasoconstriction should be of risk to the organism (Saab & Schneiderman, 1993).

Empirical study

The present study was designed to compare cardiovascular responses during effortful active coping and the emotion of anger and to independently manipulate these two conditions. Bongard, Pfeiffer, Al'Absi, Hodapp & Linnenkemper (1997) showed that adding the negative emotion of anger to an active coping task increased the load on the cardiovascular system. This study was conducted with female subjects. The present study involves male subjects. Furthermore, to assess the hemodynamic mechanisms of cardiovascular reactivity during active coping and anger impedance cardiography was introduced. Only results with respect to anger will be discussed in more detail in the present article.

Method

Participants

Sixty-three male students, 20-51 years (M = 28.06 years, SD = 5.28 years), participated in this study. One subject was excluded because of critical values in blood pressure during baseline. Participants were recruited by advertisement at the university and by personal contact. Students of psychology were excluded from the sample.

Subjects received a small present for participating in this study.

Design

The total sample was divided into two groups. During the first treatment period, one group worked on an effortful active coping task (mental arithmetic) and the other group did not (reading task). During the second treatment block, the two groups of the first treatment period were divided into two subgroups, and half of the subjects each were additionally provoked.

Provocation manipulation

The provocation condition was derived from manipulations used by Everson, McKey, and Lovallo (1995) and Suarez and Williams (1989). Provocation began at the end of a rest period which served to assess physiological baseline measures and continued 10 min between the two treatment periods. At that point, the first researcher entered the room and told the subject that he remembered an important appointment and that he should look for someone else to proceed with the experiment. He left the room, 2 min later a second experimenter entered the room and explained that the first experimenter had forgotten an appointment and had to leave.

In an unfriendly manner, the new researcher asked the subject if he had read the instruction and then asked him to summarize the instruction. Immediately after the subject had started to repeat the instruction, a phone in the adjacent control room was activated. The experimenter left the room without any apology, keeping the door open, and simulated a 3-min telephone conversation. The experimenter ostensibly spoke with a friend loudly enough for the subject to follow the conversation and realize it was a social call.

While the subjects were working on the task (mental arithmetic vs. reading), those in the anger condition were provoked three times through the intercom. In a serious and harsh tone of voice, the experimenter asked the subject to sit calmly because otherwise the physiological recordings would be useless. Under the no provocation condition, subjects were treated in a friendly manner throughout the experiment and no provocative utterances ensued. At the end, subjects were debriefed about the purpose of the study.

Measurements

For assessing the emotional states experienced during the experimental conditions, the state version of the Positive and Negative Affect Schedule (PANAS; Watson, Clark & Tellegen, 1988) in the German adaptation (Krohne, Egloff, Kohlmann & Tausch, 1996) was incorporated. The PANAS state items were given at the end of the baseline periods and after the treatment periods. To assess specifically the anger emotion, three items covering anger responses (‘irritable’, ‘upset’, ‘hostile’) were summed up.

Cardiovascular parameters were assessed using an automated computerized impedance cardiography system (cardioscreen, medis GmbH, Illmenau, Germany). The system further consists of a PC with data processing software and a transmitting unit with four pairs of spot electrodes for the analysis of the thoracic impedance field. Configuration of electrodes allowed registration of an electrocardiogram (ECG). Using an interface, the impedance cardiographic unit was connected with an oscillometric blood pressure monitor (elmed ASM 2000). The blood pressure measures were automatically transferred to the computer and used for calculating total peripheral resistance. The impedance cardiographic device provided various cardio-

vascular parameters and hemodynamic indices. The following variables were measured: heart rate (HR, measured in beats per minute, bpm), systolic blood pressure (SBP, in mmHg), diastolic blood pressure (DBP, in mmHg), mean arterial pressure (MAP, in mmHg), stroke volume (SV, in ml), cardiac output (CO, in liter/min), contractility measures like pre-ejection period (PEP, in msec) and left ventricular ejection time (LVET, in msec), and finally total peripheral resistance (TPR, in dynes-s cm^{-5}).

Results

Experience of anger

Figure 1 delineates the results for the subjective anger measure during treatment periods I and II. As in the first treatment period no anger manipulation occurred, groups did not show any differences. However, during treatment period II a very clear difference revealed. Subjectively experienced anger dramatically increases for the provocation group, and remains stable for the no provocation group. Provocation had distinct effects on the subjective experience of anger.

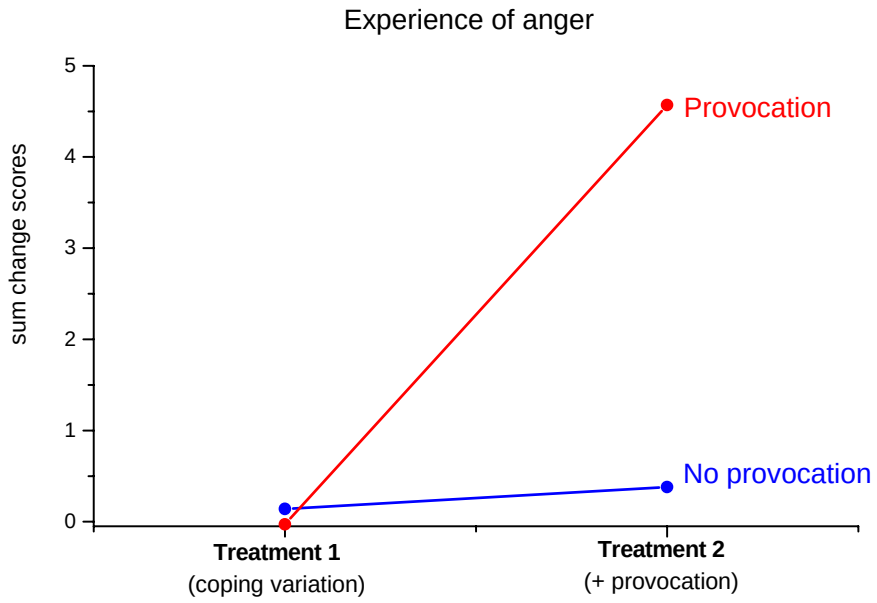

Figure 1. Anger experience during treatment periods 1 and 2.

Cardiovascular responses

The values for the cardiovascular parameters during the treatment periods are graphically depicted in Figures 2 to 5. Concerning the variables of heart rate and blood pressure a result which often has been demonstrated in the literature could be replicated. While during coping variation no group differences in HR arose, during provocation very strong increases in heart rate could be observed (Figure 2). An increase of about 12 beats per minute from baseline to the provoking situation may be regarded as considerable. Naturally, for

treatment I increased heart rate was expected as coping variation implies one group with the mental arithmetic task, i.e., effortful active coping which is also associated with an activation of the cardiovascular system.

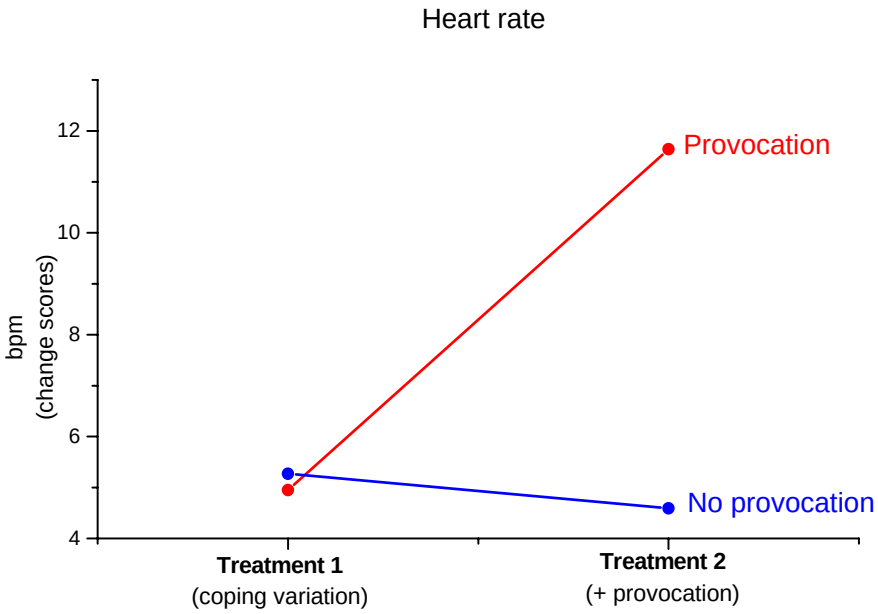

Figure 2. Heart rate changes during treatment periods 1 and 2.

The typical pattern of cardiovascular activation for anger is demonstrated in Figure 3. There is an increase in systolic as well as diastolic blood pressure responses from treatment I to treatment II. While for DBP very similar values for the provocation and no-provocation group in treatment I period exist, SBP differences during treatment period I could not be expected referring to some heterogeneities in these groups although provocation was not manipulated in this period of the experiment. Nevertheless, while increases in SBP may be observed for any aversive emotions, the heightened DBP is specifically an effect seen in the evocation of the anger emotion.

A special advantage of the methodology of impedance cardiography is that mechanisms leading to differential blood pressure responses can be detected. Based on the results of former studies, we expected decreases in stroke volume and in parameters of cardiac contractility. Decreases in parameters of cardiac contractility mean increases in contractile forces of the heart. For both parameters, stroke volume and left ventricular ejection time, the expected pattern could be found (see Figure 4). For treatment I values of groups are very similar whereas for treatment II provocation was associated with decreased values in these cardiovascular parameters.

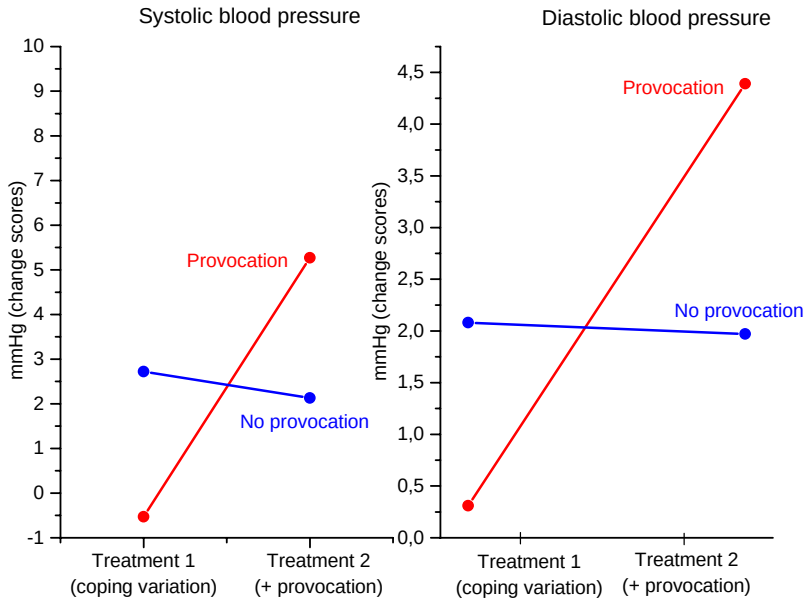

Figure 3. Blood pressure responses for treatment periods 1 and 2.

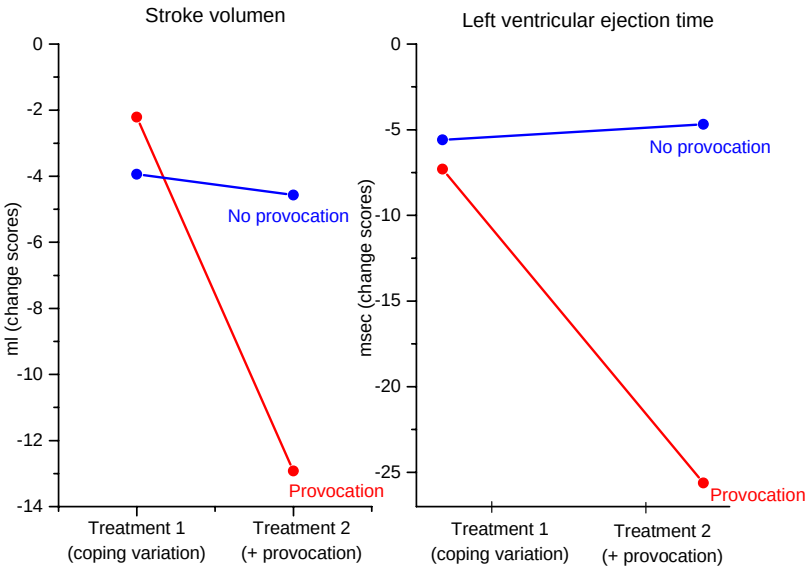

Figure 4. Impedance cardiographic variables of stroke volume and left ventricular ejection

Finally, to interpret diastolic blood pressure increases during anger total peripheral resistance has to be considered. Figure 5 presents the values of TPR during treatment periods I and II. As it easily can be seen, differences during treatment I are rather small whereas group differences under provocation demonstrate remarkable increases for vascular resistance when subjects were provoked. Especially this result provides evidence for autonomic specificity for the anger emotion and explains the observed diastolic blood pressure responses by increases in resistance in the vascular system. All effects graphically shown are significant. A more detailed presentation and discussion of the results of this study is in preparation (Hodapp & Bongard, in preparation).

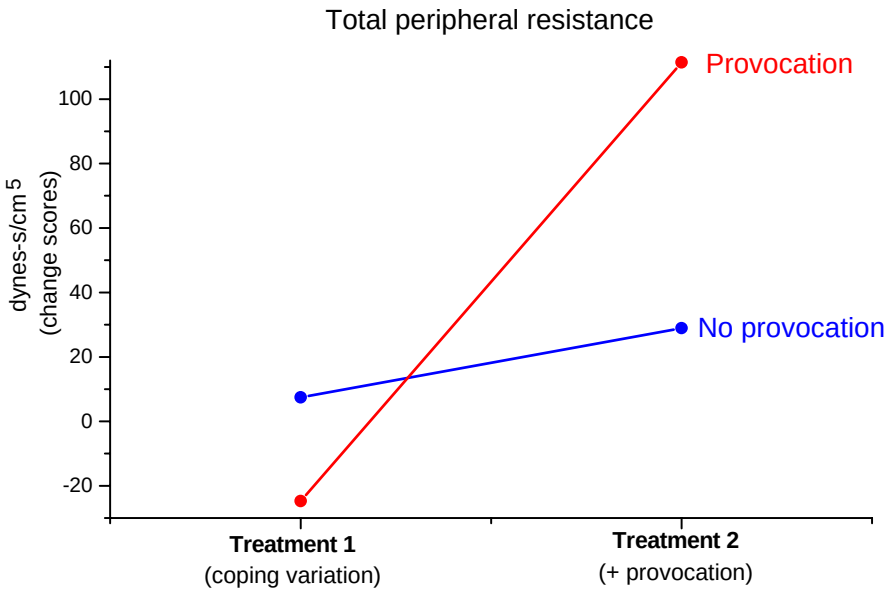

Figure 5. Total peripheral resistance for treatment periods 1 and 2.

Applications and conclusions

A major conclusion from the results of the present study is the profound effect of anger on cardiovascular functioning. More than any other emotion anger is associated with an activation of the cardiovascular system. Consistent with earlier results there are remarkable changes in heart rate and systolic and diastolic blood pressure during anger. Essentially, diastolic blood pressure increases during anger but not in fear (Hodapp et al., 1993; Roberts & Weerts, 1982; Schwartz et al., 1981; Sinha et al., 1992).

The additional measures of various cardiovascular parameters by means of im-

pedance cardiography contribute to the understanding of the mechanisms involved in differential blood pressure increases. Along with increases in diastolic blood pressure, decreases in stroke volume, left ventricular ejection time, and increases in the total peripheral resistance were observed. Pre-ejection period did not change significantly while cardiac output decreased. Comparing our results with the results of Sinha et al. (1992), the cardiovascular response patterns described in both studies appear to be very similar. Decreases in stroke volume and left ventricular ejection time were commonly observed. While in the study of Sinha et al. also decreases in pre-ejection time occurred, this measure remained constant in our study. Cardiac output increased in the study of Sinha et al., while in our study cardiac output showed slight decreases. Total peripheral resistance decreased minimally from baseline during anger in the imagery study, while in the present study total peripheral resistance significantly increased. Despite some differences in changes in single parameters, the results provide evidence that maintenance or increase of vasoconstriction along with increases of contractile forces (indicated by shortening of contractility parameters PEP and LVET) account for the increases in both systolic and diastolic blood pressure during anger. The cardiovascular pattern during anger resembles the norepinephrine-like response observed by Schachter (1957) where heart rate and systolic blood pressure indicate that the typical anger response may reveal as mixed epinephrine-like and norepinephrine-like response.

When discussing evidence for the autonomic differentiation of emotions it should be taken into account that the method of emotion induction could play an important role in the physiological pattern associated with the elicited emotion. Schwartz et al. (1981) and Sinha et al. (1992) used an imagery induction technique requiring subjects to identify specific emotional situations from their own lives and to recreate as vividly as possible feelings and sensations associated with the scene or script. We applied a real-life manipulation which involved provocations derived from the phenomenology of anger which has been characterized as an emotion originating from external attribution of injury perceived as arbitrary and hostile. Especially Stemmler (1989) and Stemmler, Heldmann, Pauls, and Scherer (2001) report that emotional autonomic patterning may be specific to the context of emotion induction. We suggest that some differences in the cardiovascular response patterns observed in our study and the imagery studies of Schwartz et al. (1981) and Sinha et al. (1992) may depend on the specific methods of emotion induction. Nevertheless, the distinct cardiovascular activation during anger which is very similar in the extent of response indicates how important it is to induce emotions in the laboratory to study the mechanisms underlying emotional processes.

An interesting application of research on the psychophysiology of anger refers to hypotheses about the potential role of anger and hostility in the development of hypertension and cardiovascular diseases. Intense emotions play an important role in the onset of acute coronary events such as myocardial infarction and sudden cardiac death (Krantz, Kop, Santiago & Gottdiener, 1996; Saab & Schneiderman, 1993). The massive effects of anger on cardiovascular responses nourished the supposition that cardiovascular activation due to intense and continually existing psychological stress may lead to chronic elevations in blood pressure or damaging processes in the vascular system (“cardiovascular reactivity hypothesis”; Rozanski, Blumenthal & Kaplan, 1999). Suarez and Williams (1989) and Weidner, Friend, Ficarotto, and

Mendell (1989) found greater cardiovascular responses during anger provoking situations in person characterized by high trait hostility. Epidemiological studies (e.g., Barefoot, Dodge, Peterson, Dahlstrom & Williams, 1989; Dembroski, MacDougall, Costa & Grandits, 1989; Shekelle, Gale, Ostfeld & Oglesby, 1983) found relationships between the personality trait of hostility and the incidence of coronary heart disease and general morbidity or mortality rates. Hostility, anger, and the expression of anger are major variables which emphasize the potential role of emotions for psychological and physical health. Psychophysiological research may contribute to a better understanding how emotions affect health, in an adaptive, health preserving as well as in a maladaptive, illness-provoking sense (Miguel-Tobal, 2001). The present study could demonstrate the profound effect of anger on cardiovascular functioning and the interplay of emotional and physiological processes in human behavior.

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Resumen en castellano

Este artículo se centra específicamente en la emoción de la ira y en el estudio de las respuestas psicofisiológicas asociadas a esta emoción básica.

La ira es una experiencia emocional que aparece asociada a situaciones donde nos sentimos heridos por los otros (Averill, 1982; Hodapp y Scwenkmezger, 1993). La emoción de ira es compleja y deben diferenciarse varios componentes que incluyen, al igual que en otras emociones, la valoración cognitiva, la experiencia subjetiva, los cambios fisiológicos asociados y las conductas que se llevan a cabo como respuesta a los retos significativos del ambiente (Frijda, 1986; Scherer, 1984).

Así mismo, existe un cierto solapamiento entre los constructos relacionados de ira, agresión y hostilidad. Mientras que la ira debe considerarse como un constructo emocional, la agresión generalmente implica conductas punitivas o destructivas dirigidas hacia otras personas u objetos del entorno. La hostilidad, aunque implica sentimientos de ira, debe entenderse más como un conjunto complejo de actitudes que motivan la aparición de conductas agresivas hacia otras personas (Spielberger et al., 1985). Desgraciadamente, la literatura sobre la investigación de los constructos de ira, hostilidad y agresión refleja un alto grado de confusión y ambigüedad conceptual. Aunque la hostilidad está relacionada con la ira y las conductas agresivas, parece pertinente diferenciarlas tal y como se propone en el Síndrome IHA (Ira-Hostilidad-Agresión; Spielberger et al., 1985)), restringiendo la ira al componente emocional del síndrome.

La cuestión de si las emociones conllevan una actividad específica del Sistema Nervioso Autónomo o no ha sido fuente de grandes controversias. El estudio de la fisiología de la ira comenzó con Cannon al describir que la respuesta de lucha-huida implicaba incrementos de la presión sanguínea y la tasa cardiaca, vasodilatación en la musculatura esquelética y vasoconstricción visceral, y cambios bioquímicos asociados a la movilización de la energía (Cannon, 1953). La concepción unidimensional del arousal de Cannon se puso en duda por la observación de que la activación autonómica indiferenciada durante los estados emocionales conllevaba experiencias emocionales discretas. El miedo y la ira difieren en la fenomenología de la experiencia emocional. Mandler (1975) y Schachter y Singer (1962) plantearon que las experiencias emocionales discretas derivarían de procesos cognitivos de valoración iniciados por la percepción indiferenciada de la activación fisiológica que se atribuía a diferentes claves situacionales.

Una posición alternativa apoya la idea de la diferenciación fisiológica de las emociones. Esta tradición se remonta al famoso artículo de William James de 1884, donde afirmaba que los sentimientos emocionales son consecuentes más que antecedentes de los cambios fisiológicos periféricos elicitados por la estimulación externa. Diferentes estudios han demostrado la existencia de diferencias cualitativas en la activación autonómica y somática en diversos estados emocionales, apoyando la idea de que las experiencias emocionales discretas se basan en patrones somatoviscerales diferenciados (Levenson, Ekman y Friesen, 1990).

Estudio empírico

El presente estudio se diseñó con el objeto de comparar las respuestas cardiovasculares durante una tarea de afrontamiento activo y la emoción de ira, manipulando independiente

estas dos condiciones. Bongard, Pfeiffer, Al'Absi, Hodapp y Linnenkemper (1997) demostraron que añadir la emoción negativa de ira a una tarea de afrontamiento activo incrementaba la carga sobre el sistema cardiovascular. Dicho estudio se llevó a cabo con sujetos mujeres; el presente trabajo se realiza solamente con varones. Para evaluar los mecanismos hemodinámicos de la reactividad cardiovascular durante la tarea de afrontamiento activo y la ira se utilizó la cardiografía de impedancia. En este trabajo se presentan en detalle únicamente los resultados relacionados con la ira.

Muestra

Participaron en el estudio 63 estudiantes varones de entre 20 y 51 años (Media = 28.06 años; Sx = 5.28 años). Un sujeto fue excluido al mostrar valores críticos en la presión sanguínea durante la toma de Línea Base. No se incluyeron en el estudio estudiantes de Psicología. Los sujetos recibieron un pequeño regalo por su participación en el estudio.

Diseño

La muestra total se dividió en dos grupos. Durante el primer período de tratamiento, un grupo se dedicó a una tarea de esfuerzo de afrontamiento activo (tarea de aritmética mental) y otro grupo no (tarea de lectura). Durante el segundo período de tratamiento, ambos grupos se dividieron de dos subgrupos, la mitad de los sujetos de cada grupo fueron sometidos a provocación.

Manipulación de la condición de provocación

La condición de provocación se basó en las instrucciones utilizadas por Everson, McKey y Lovallo (1995) y Suarez y Williams (1989). La provocación empezaba al final del período de descanso que servía para monitorizar la actividad fisiológica en condiciones de línea base y continuó durante 10 minutos entre los dos períodos de tratamiento. En este momento, el primer investigador entraba en la cabina de experimentación y le decía al sujeto que se había acordado que tenía una cita importante, ineludible y debía dejar el experimento, pero que buscaría a alguien más para que continuase con el mismo. El primer experimentador salía de la cabina de experimentación y, dos minutos después, entraba un segundo experimentador que explicaba que el primero había olvidado una cita importante y debía irse.

De forma poco amistosa el nuevo experimentador preguntaba al sujeto si había leído correctamente las instrucciones y le instaba a que hiciera un resumen de las mismas. Inmediatamente después de que el sujeto hubiera comenzado a repetir las instrucciones a petición del experimentador, se activaba el sonido de un teléfono en la habitación continua a la cabina de experimentación y el experimentador salía a atender el teléfono sin disculparse previamente, dejando la puerta abierta y simulando una conversación telefónica durante tres minutos. El experimentador hablaba en voz suficientemente alta para que el sujeto pudiera escuchar la conversación y darse cuenta de que estaba teniendo una charla social con un amigo, que nada tenía que ver con el experimento.

Mientras que los sujetos trabajaban en la tarea (aritmética mental versus lectura), los asignados a la condición de ira fueron provocados tres veces a través de un intercomunicador. Con un tono de voz serio y violento, el experimentador pedía al sujeto que se mantuviera sentado y sin moverse porque de lo contrario el registro fisiológico quedaría inva-

lidado. En la condición de no-provocación, los sujetos fueron tratados de forma amistosa a lo largo de todo el experimento, sin provocación alguna. Al final del experimento se les explicó a los sujetos el propósito del mismo y la razón de haber sido tratados de esa forma.

Medidas

Para evaluar los estados emocionales experimentados durante el experimento se utilizó la versión estado del Positive and Negative Affect Schedule – PANAS (Watson, Clark y Tellegen, 1988), usando la adaptación alemana de Krohne, Egloff, Kohlmann y Tausch (1996). El PANAS se administró el final de los períodos de línea base y tras los períodos de tratamiento. Para evaluar específicamente la emoción de ira se sumaron los tres ítems que hacen referencia a las respuestas de ira ("irritable", "enfadado" y "hostil").

Los parámetros cardiovasculares se evaluaron mediante un sistema de cardiografía de impedancia computerizado. El equipo de cardiografía de impedancia permite el registro de diversos parámetros cardiovasculares e índices hemodinámicos. Se registraron las siguientes medidas: tasa cardiaca, presión sanguínea sistólica y diastólica, presión arterial media, volumen de fuerza cardiaca, salida cardiaca, contractabilidad miocardial, tiempo de eyección del ventrículo izquierdo y resistencia periférica total.

Resultados

Experiencia de ira.

La figura 1 muestra los resultados de las medidas subjetivas de ira durante los períodos de tratamiento I y II. Al no manipularse la ira en el primer período, no existieron diferencias significativas entre ambos grupos. Sin embargo, en el segundo período se obtuvieron diferencias claras. El grupo de provocación experimentó incrementos drásticos en la experiencia de ira mientras que en el grupo de no-provocación los niveles se mantuvieron estables. La provocación tiene efectos significativos en la experiencia subjetiva de ira.

Medidas cardiovasculares.

Los valores obtenidos en los parámetros cardiovasculares se muestran gráficamente representados en las figuras 2 a 5. Respecto a la tasa cardiaca y la presión arterial, los resultados están acordes con la literatura experimental existente. Durante el período de tarea no hubo diferencias en tasa cardiaca en ambos grupos, pero durante el segundo período, el grupo de provocación experimentó fuertes incrementos en tasa cardiaca (Figura 2), alrededor de 12 latidos/minuto respecto a la línea base, que deben ser tomados como considerables.

La figura 3 muestra un patrón típico de activación cardiovascular debido a la ira. Se produce un incremento en la presión arterial sistólica y diastólica en el segundo período respecto al primero. Mientras que los incrementos de la presión sistólica se pueden observar en cualquier emoción aversiva, el incremento notable de la presión diastólica es un efecto característico de la evocación de la emoción de ira.

Esperábamos descensos en el volumen de fuerza cardiaca en los parámetros de la contractibilidad cardiaca. La disminución de la contractibilidad cardiaca incrementa la fuerza de contracción del corazón. Ambos parámetros, volumen en la fuerza cardiaca y tiempo de eyección del ventrículo izquierdo, mostraron los patrones esperados (Figura 4), descenciendo sus valores en el segundo período respecto del primero.

Finalmente, la figura 5 muestra los valores de la resistencia periférica total en los períodos de tratamiento I y II. Las diferencias entre grupos en la condición de provocación muestran incrementos notables de la resistencia vascular cuando los sujetos eran provocados. Este resultado aporta evidencia de la especificidad autonómica para la emoción de la ira y explica los resultados de la presión diastólica debido al incremento de la resistencia del sistema vascular.

La conclusión principal de los resultados del presente estudio es que la ira tiene un notable impacto sobre el funcionamiento cardiovascular. Más que ninguna otra emoción, la ira está relacionada con la activación del sistema cardiovascular. Consistentemente los resultados aportados por otros estudios anteriores, hemos encontrado cambios notables en la tasa cardiaca y la presión sistólica y diastólica durante la experiencia de ira. Básicamente, ña presión diastólica aumenta durante la ira pero no durante el miedo (Hodapp et al., 1993; Roberts y Weerts, 1982; Schwartz et al., 1981; Sinha et al., 1992).

El estudio de otros parámetros cardiovasculares mediante la cardiografía de impedancia contribuyen al conocimiento de los mecanismos implicados en el aumento diferencial de las presiones sanguíneas. Hemos observado que junto al incremento de la presión diastólica se producían descensos en el volumen de fuerza cardiaca y el tiempo de eyección del ventrículo izquierdo, aumentando la resistencia periférica total.

Nuestros resultados aportan evidencia que el mantenimiento o incremento de la vasoconstricción y la fuerza de contracción del corazón están muy relacionadas con el aumento de la presión sistólica y diastólica durante la ira.

Una aplicación interesante derivada de la investigación psicofisiológica de la ira se refiere a la hipótesis sobre el papel potencial de la ira y la hostilidad en el desarrollo de la hipertensión y los trastornos cardiovasculares. La hostilidad, la ira y la expresión de la ira son variables que enfatizan el potencial papel de las emociones en la salud física y mental. La investigación psicofisiológica puede contribuir a un mejor conocimiento de cómo las emociones afectan a la salud, preservándola, si son adaptativas, y provocando enfermedades, si son desadaptativas (Miguel-Tobal, 2001). El presente estudio podría demostrar el notable efecto de la ira en el funcionamiento cardiovascular y las relaciones entre procesos emocionales y fisiológicos en la conducta humana.