Chapter 7
Somatovisceral Activation During Anger

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Abstract What is the physiological signature of anger? If not anger, which emotion would be more destined to turn a "cold" object perception into a Jamesian "hot" emotional encounter? Indeed, reports of bodily anger sensations are descriptions of heat and tension. However, the message from studies reporting physiological anger responses is more difficult to reconcile. The chapter discusses landmark studies on the differentiation between anger and fear. It is emphasized that their methodological characteristics are decisive for demonstrating or failing to show physiological anger specificity. A meta-analysis shows that anger provocation elicits strong changes in systolic and diastolic blood pressure, heart rate, number of skin conductance responses, and muscle activity. The pattern resembles the combined action of adrenaline and noradrenaline, accompanied by strong vagal withdrawal. It is argued that these coordinated changes have a functional value for the pursuit and finally the attainment of the goal of anger: to motivate individuals to avoid failure and pain by averting subordination under physically or socially caused harm and to gain superiority.

Anger, like all powerful emotions, has11 a marked immediacy and salience in our experience. Almost instantaneously, we sense changes in our body. Something has a grip on us that we cannot control easily – then we fly into a passion (from *lat.* pati, to suffer). This common experience of people in all cultures and ages led, e.g., Aristotle to believe that mental phenomena were linked to organismic matter in a stepwise way: This link was presumed to be strong for emotion, less strong for thinking, and absent for the active intellect, which was thus considered "free." Throughout Western thinking, a guiding idea has been that emotions entail irrationality, loss of free will, and animalistic drives; the core of this idea was that emotions are embodied ("psychophysiological symbolism," Averill, 1974; Chapter 9 by J. Green et al., this book) and shaped by nature ("natural kinds," Barrett, 2006).

One might think that it is quite easy to show that anger is associated with certain changes in our bodily state, since we so obviously experience anger's actions in our body. But science is about questioning the obvious. We could ask, for example, what causes the reports about our sensed bodily changes during anger or what do these bodily changes look like? I start with the first question and in the main part of the chapter proceed to the second one.

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7.1 Sensed Bodily Changes

William James is considered the founder of the psychophysiological tradition in emotion research (Plutchik, 1980). He affirmed the layperson's belief that an emotion is its feeling. Somatovisceral responses follow directly upon the perception and interpretation of an emotion-eliciting stimulus. The sensation of these somatovisceral changes is a necessary condition for the formation of a feeling (for an extended Discussion, see Ellsworth, 1994; James, 1884); sensed bodily changes render the "cold" object perception into a "hot" emotional encounter. Consequently, there should be more or less strong associations between bodily responses to the emotion-arousing stimulus and their experienced sensation. Indeed, studies have shown the impact of afferent signals stemming from somatovisceral activity on brain systems and feelings (Berntson, Sarter, & Cacioppo, 2003; Critchley, Wiens, Rotshtein, Ohman, & Dolan, 2004; Pollatos, Gramann, & Schandry, 2007).

7.1.1 Reports of Bodily Sensations During Anger

Adults in various cultures report that during anger, the face turns red, the body tenses and feels hot, and breathing accelerates (see also Chapter 10 by Z. Kövesces, this volume). Janke (2002) showed that this knowledge is present by the age of 10. For example, reported increases in "feeling hot" were higher in anger than in fear, joy, and sadness; the other bodily changes sensed in anger (tension, fast breathing, rapid heart rate) were as high in fear (Rimé, Philippot, & Cisamolo, 1990). Similarly, across 2,921 respondents from 37 countries on five continents, Scherer and Wallbott (1994) found that anger was characterized by rapid heart rate (49.5% of respondents endorsed this sensation), tension (42.7%), fast breathing (37.1%), and feeling hot (31.7%). Compared to fear, anger ranked second in these bodily sensations, except for "feeling hot," which, however, was endorsed even more strongly in shame.

Three conclusions can be drawn from these results. First, with some degree of universality, people can differentiate the bodily sensations of anger from most other emotions. Second, the differentiation between sensed bodily changes during anger and fear depends on a *pattern* of changes and is difficult to make. Finally, the question remains how valid these reports of bodily sensations are when compared to actual somatovisceral changes. I will pursue this question in a later section after describing the physiological anger responses.

7.2 Physiological Anger Responses

There are hundreds of studies on the psychophysiological anger response. At the time of this writing, PsycINFO lists 603 entries for "anger and cardiovascular," 510 for "anger and blood pressure," 494 for "anger and (biology or biological)," 322 for "anger and (psychophysiology or psychophysiological)," and 1,029 for "anger and (physiology or physiological)." Many of these studies report associations of anger with clinical or subclinical states (e.g., high blood pressure, treatment programs), effects of anger on just one physiological response, or effects of just one emotion, namely anger. Such studies do not allow the drawing of conclusions about the specificity of physiological anger responses. That is why the two published meta-analyses on physiological emotion specificity identified only few qualified studies (Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000, 18 studies comparing anger vs. other emotions published up to 1997; Stemmler, 2004, 15 studies

comparing anger vs. fear published up to 2001). Results from these meta-analyses will be presented after some landmark studies are discussed in the following section.

7.2.1 Responses in Anger Versus Control Conditions

The studies described here differ in many aspects: The kind of emotion induction, sample sizes, the timing of recording periods, baseline assessment, the assessment of components of emotion other than the physiological (e.g., emotion self-report, facial expression, voice), the statistical analysis, and whether and how the situational context was controlled for. Situational context control is necessary because every emotion induction works through a situational mediator (e.g., a film, autobiographical recall, interpersonal interaction) which leaves its own traces in physiological activity. To be sure, emotion responses should be captured free from such situational context effects. Otherwise we find in separate studies different "anger" physiologies only because the induction contexts differed.

7.2.1.1 Ax (1953)

This is *the* classical study on the psychophysiology of fear and anger; it claimed physiological specificity for these emotions. A partly identical experiment with an overlapping set of subjects was published by Schachter (1957), who gave a more complete description of the experiment than Ax did. Subjects were 43 unemployed women and men. They were asked to lie down on a bed and rest before the emotions were induced by "real-life" situations presented in a balanced order. Anger was induced by an incompetent, arrogant, and previously fired technician, who helped out "just today" (his successor was supposedly ill). Without permission of the experimenter, the technician checked the wiring, abruptly turned off the calming music played in a rest period, handled the subjects roughly and painfully, and criticized them without reason. Fear was induced by a putative life-threatening electrical short-circuit in the recording apparatus.

For statistical analysis, emotion effects were captured as difference scores between induction and prestimulus phases. During anger, heart rate, stroke volume, systolic and diastolic blood pressure, respiration rate, skin conductance level, and number of responses as well as the electromyogram (EMG) of the *m. frontalis* increased (reported are the maxima during the induction period!), whereas finger and face skin temperature fell (the minima were reported). Seven of the 14 physiological variables differentiated between the anger and fear inductions. Specific responses for anger were larger increases in diastolic blood pressure, muscle tension, and number of skin conductance responses, as well as larger decreases in heart rate minima than for fear. Ax integrated the observed physiological profile differences in the hypothesis that the physiological anger pattern resembles the effects of a mixed adrenergic–noradrenergic secretion, and the fear pattern, an adrenergic secretion.[1]

[1] Adrenaline stimulates alpha-adrenergic, beta1- and beta2-adrenergic receptors. Adrenergic responses are defined as reductions in finger temperature (probably because blood is redistributed from the skin to skeletal muscles), diastolic blood pressure, and total peripheral resistance and as increases in heart rate, systolic blood pressure, stroke volume, left-ventricular contractility, cardiac output, number of skin conductance responses, and respiration rate. Noradrenaline stimulates alpha- and beta1-adrenergic receptors. Noradrenergic responses are characterized by increases in systolic and diastolic blood pressure, left-ventricular contractility, number of skin conductance responses, and total peripheral resistance, as well as reductions in heart rate and finger temperature (Chessick et al., 1966; Löllgen, Meuret, Just, & Wiemers, 1985; Wenger et al., 1960). Compared to adrenaline, noradrenaline produces a lower heart and respiration rate, lower stroke volume and cardiac output, but a higher diastolic blood pressure and total peripheral resistance and a higher finger temperature.

Commendable features of Ax' experiment are the credibility, "vividness," and probably the intensity of the emotion inductions, the multivariate physiology, and the use of a non-student sample. But there are also some important limitations. First, the lack of data from other emotion components (e.g., subjective ratings of emotion) prohibits an unambiguous conclusion about the validity of the emotion inductions. In particular, it is not clear if pure anger or a fear–anger blend was induced. Second, subjects spoke and moved during the recording periods, activities that may strongly affect physiological data. Third, recording periods were overly long (7 min); increasing the likelihood of non-emotional contributions to the physiological profile. Fourth, physiological minima and maxima in long recording periods are not representative of the whole period and are less reliable than means or medians. Fifth, the calculated difference score contains both emotion and context effects. Generalizability of results can be achieved only if the effects of the non-emotional context are markedly reduced or eliminated. Finally, the statistical analysis reported was incomplete, only post hoc calculations produced the estimates of the physiological anger response noted above.

7.2.1.2 Funkenstein, King, and Drolette (1954)

This study also advanced the idea that anger is associated with a mixed adrenergic–noradrenergic physiological response (more on this below), but the unique feature of this study is the differential perspective taken. Not all subjects respond alike to the same emotion stimulus (see Chapter 15 by T. Wranik & K.R. Scherer, this volume). Consequently, subjects could be partitioned into subgroups of similar affective experience before their physiological patterns are calculated.

The authors studied 69 subjects in a frustrating number task. Subjects had to repeat six 10-digit numbers forward and backward. Then they had to solve difficult mental arithmetic tasks. Subjects were criticized when they made errors, and finally their complete failure was stated condescendingly. After that physiological data recording began. In an ensuing interview, the subjects' feelings were assessed for the mixture of anger and anxiety (anger out, anger in, anger equally out and in, equal anger and anxiety, anxiety, no emotion, miscellaneous). The anger out group ($N = 21$) showed significant percent increases from baseline to the post-provocation period in heart rate, systolic and diastolic blood pressure, and, from a probably quite unreliable ballistocardiographic recording, an increase in total peripheral resistance as well as a reduction in stroke volume and no change in cardiac output. Compared to the anxiety group ($N = 9$), the anger out group had a lower heart rate, systolic blood pressure, stroke volume, and cardiac output response, a larger response in total peripheral resistance, and an equal diastolic blood pressure response.

The strengths of this study are (1) the assessment of feeling states, (2) the use of this information to partition the sample (more on post-experimental subject selection, see Stemmler, 2003) for a potentially more concise assessment of the physiological anger response, and (3) a separation of task and recording period. Again the major problem of this study is the lack of a control condition that would permit the emotion and non-emotion effects of the induction procedure to be disentangled. A non-emotion effect in this study is the mental effort exerted during the task (for an experimental proof of such effects in a public speaking task, see Erdmann & Baumann, 1996) limiting the generalizability of the anger response data.

7.2.1.3 Levenson, Ekman, and Friesen (1990)

In this publication, the authors introduced a new emotion induction technique in a series of three experiments: The Directed Facial Action Task. Subjects were instructed to contract and hold several facial muscles. This procedure is based on the assumption ("facial feedback hypothesis") that the expression of emotions, especially in the face, would enable the corresponding affect program in the

brain and thus also its specific somatovisceral activations. Subjects followed sequential instructions to voluntarily contract sets of universally recognized facial muscle configurations of anger, disgust, fear, happiness, sadness, and surprise. Before each trial, subjects produced a standard control face that served as a baseline for the subsequent emotion face. In the first experiment, the authors varied the situational context and presented the subjects with both the Directed Facial Action Task and an imagery task, which yielded different autonomic patterns of emotion.

Levenson et al. (1990) reported means in four physiological variables across the three experiments. Compared to the control face, the anger face produced a higher heart rate, finger skin temperature, and skin conductance level. No differences were found for the muscle activity at the forearm flexor. A difference between the anger and the fear face was seen only in the higher finger skin temperature of anger. The authors interpreted the results as a clear evidence for emotion-specific autonomic activity.

The experiments of this study demonstrate the methodological advancement across the 40 years since the Ax or Funkenstein studies. There was good context control because the baseline was a control face and not just a resting period. Another positive point was the induction of no less than six "primary" emotions and the extensive use of subject information to subdivide the sample with separate specificity analyses.

7.2.1.4 Sinha, Lovallo, and Parsons (1992)

This study is an excellent example for the thoughtful application of the induction of anger via imagination. Subjects came to four separate sessions. The first session served to screen subjects (according to quality of mental imagery, alexithymia, and depression/state anxiety) and obtain personalized emotion scripts. Two sets of scripts for each of the emotions of anger, fear, joy, and sadness and for a physical action and a neutral state scene were developed by each subject, then rated for emotional content and intensity by both the subject and two independent raters. The training session allowed subjects to train imagery effective for enhanced physiological responsivity. The two experimental sessions presented each of the six scripts in randomized order, interspersed with recovery periods that lasted until a stable physiological baseline was achieved. Imagery effects were captured as imagination minus prescript differences, context control was established by defining emotion effects as emotion imagery effect minus neutral imagery effect.

The anger imagery effect comprised increases in heart rate, systolic and diastolic blood pressure, cardiac output, and total peripheral resistance, whereas left-ventricular ejection time and stroke volume decreased. Compared to fear, anger was characterized by larger increases in diastolic blood pressure and total vascular resistance.

This was a very carefully designed study. Subjects were selected to be good imaginers and they were trained. The scenes represented rather pure emotions. A representative sample of cardiovascular variables was recorded. In addition, imagination procedures have quite low non-emotional effects on somatovisceral activation, and whatever the imagination context elicited was probably effectively controlled by the neutral script.

7.2.1.5 Stemmler, Heldmann, Pauls, and Scherer (2001)

This study performed emotion inductions of anger and fear each with two different techniques (real life and imagery). An extensive set of 29 somatovisceral variables were recorded from a sample of 158 female subjects in two experimental sessions. Real-life anger was induced in three consecutive periods with intermittent recordings of physiological activation and emotion self-report. First, subjects were presented with a difficult test of general knowledge. If they did not know the answer, they

had to say loudly "I don't know!" After the second, almost unsolvable item, subjects were asked to speak louder. Midway through, the experimenter angrily interrupted the subject saying he could not understand her. After the task subjects were informed that they had solved only one-third of the items correctly. Second, subjects performed a mental arithmetic task silently and as quickly as possible. They were interrupted twice and asked to tell the result at the moment. The experimenter commented on the poor performance and had the subject start all over again grumbling at her. Third, subjects had to solve a difficult anagram task. After 6 of 12 words, the experimenter angrily argued with the subject for moving around too much, and at the end, he accused her of noncompliance. Physiological profiles in the three induction periods were homogeneous which allowed us to use the more reliable mean of the three periods for specificity analysis. Imagery induction of anger was performed 1 week later. Subjects were asked to recall each induction period of the real-life session as vividly as possible.

Context control was instantiated by a separate control group. Control subjects were told at the beginning of the experiment that the real objective of the study was to induce anger (or fear in the respective group). To strengthen the trustworthiness of this information, the prerecorded harassments used during the later anger induction were played. Then exactly the same experiment was performed as in the anger treatment group. That is, the control subjects were exposed to exactly the same stimuli; only their interpretation of the stimuli, and the resulting emotional responses, differed. Thus, the difference between treatment and control groups should capture the emotion effects proper. All analyses were based on these differences.

The anger real-life effect comprised changes in 19 of 29 somatovisceral variables, including increases in heart rate, systolic and diastolic blood pressure, cardiac output, skin conductance level, skin temperature at the forehead, and extensor digitorum muscle activity. Compared to fear, specific responses to anger were larger total vascular resistance, skin temperature at the forehead, and extensor digitorum muscle activity. The anger imagery effect comprised 8 of 29 somatovisceral variables, among them increases in heart rate, systolic and diastolic blood pressure, number of skin conductance responses, and skin temperature at the forehead. Differences to fear imagery were seen in diastolic blood pressure.

In sum, this study pushed methodological standards even further, with the introduction of discriminant (anger vs. fear) and convergent validity (real-life vs. imagery) tests, with multiple induction periods, and an effective though costly context control strategy. Multivariate somatovisceral recordings and multivariate statistical analysis complemented each other.

The next section moves from particular landmark studies to overviews of the field.

7.2.2 Meta-analyses of Anger Effects

Psychophysiological research on emotion specificity has been analyzed in the meta-analysis of Cacioppo et al. (2000). The methodological quality of the included experimental studies was not weighted. The authors found a higher heart rate increase for anger, fear, and sadness compared to disgust and of anger compared to happiness. Compared to fear, anger responses were higher in diastolic blood pressure, number of skin conductance responses, total peripheral resistance, facial skin temperature, and finger pulse volume. Heart rate, stroke volume, and cardiac output increases were smaller in anger than in fear. The authors' conclusion was: "In sum, the meta-analyses indicated that even a limited set of discrete emotions such as happy, sad, fear, anger, and disgust cannot be fully differentiated by visceral activity alone . . ." (p. 184). The authors reasoned that instead ". . . the negative emotions in this literature are associated with stronger ANS [autonomic nervous system] responses than are the positive emotions" (p. 184). Their Table 11.2 reveals that 10 out of 22 somatovisceral variables significantly differentiated between negative and positive emotions; this result was

based on all of the studies reviewed. The comparison between anger and fear, however, yielded eight significant variables; and these derived from only a subset of all of the studies. Thus, contrary to the authors' conclusions, it seems that their own data reveal quite a sizable amount of differentiation between anger and fear and thus make a case at least for anger and fear's somatovisceral specificity.

A meta-analysis of somatovisceral anger and fear effects was conducted by Stemmler (2004). It was based on 15 studies which reported anger and fear contrasts in at least two somatovisceral responses. Only those response variables were considered which had been used in at least three studies. Results are shown in Table 7.1.

Table 7.1 Physiological anger and fear response

Variable	*k*	*d* Anger vs. Control	*d* Fear vs. Control	*d* Anger vs. Fear
Systolic blood pressure	11	1.81**	1.67**	−0.06
Diastolic blood pressure	11	1.58**	0.93**	0.43**
Heart rate	14	1.39**	1.32**	−0.16
Number of skin conductance responses	4	1.06**	1.15**	−0.02
Muscle activity	4	1.04**	0.32*	0.37**
Skin temperature face	4	0.68**	−0.02	0.45**
Stroke volume	4	−0.63**	−0.43*	−0.12
Skin conductance level	5	0.49**	0.12	0.18
Respiration rate	7	0.47**	0.87**	−0.41**
Cardiac output	4	0.43**	0.85**	−0.41**
Skin temperature finger	8	−0.32**	−0.68**	0.18
Total peripheral resistance	3	0.32	−0.58**	0.43**

Meta-analytic results from Stemmler (2004). k = Number of independent studies. d = Effect size d (positive values denote higher means for anger in column 3, for fear in column 4, and for anger in column 5). By convention, an effect size of 0.20 denotes a "small," of 0.50 a "medium," and of 0.80 a "large" effect (Cohen & Cohen, 1983).
*$p \leq 0.05$; **$p \leq 0.01$.

Compared to control, the anger provocation elicited strong changes in systolic and diastolic blood pressure, heart rate, number of skin conductance responses, and muscle activity. Facial skin temperature rose, as did skin conductance level, respiration rate, cardiac output, and total peripheral resistance, whereas stroke volume and finger temperature dropped. This pattern resembles the combined action of adrenaline and noradrenaline, accompanied by strong vagal withdrawal (see Footnote 1). Compared to fear, anger was characterized by a larger response in facial temperature, diastolic blood pressure, total peripheral resistance, and muscle tension. During fear, respiration rate and cardiac output were larger than during anger. Thus, 6 out of 15 variables indicated specific responses when anger was compared to fear. But there were strong responses to anger which overlapped considerably with responses to fear, such as heart rate or systolic blood pressure. I will return to this point later.

The meta-analysis also revealed that the studies were significantly heterogeneous with respect to the effect sizes just noted. Three potential moderator variables were examined for their ability to reduce this heterogeneity and to gather additional information about the conditions under which the specificity of the physiological anger response was largest. The analysis of moderator variables was restricted to data sets with at least 10 studies. This criterion left only heart rate and systolic and diastolic blood pressure as outcome variables.

The first moderator variable was the induction context (imagination; real life). Somatovisceral anger versus control effect sizes did not depend on the induction context; compared to fear, however, anger specificity in diastolic blood pressure was very large, when imagination (Cohen's effect size $d = 1.06$) was the induction method, but it was low during real-life inductions ($d = 0.18$). The second moderator variable was the design of the emotion effect (within-subjects, i.e., repeated emotion

inductions within individuals; or between-subjects, i.e., only one emotion induction per individual). In diastolic blood pressure, anger versus control effect sizes were larger when a within-subjects ($d = 1.85$) rather than a between-subjects design of the emotion effect ($d = 1.06$) was used. A within-subjects design ($d = 0.72$) was also preferable when anger specificity was probed (between-subjects design: $d = 0.14$). The third moderator was the design of the control strategy used (within-subjects comparison of emotion inductions with a rest, prestimulus, or poststimulus period; within-subjects comparison with a control condition that controls for context effects). Again in diastolic blood pressure, context control produced larger specificity effects ($d = 1.06$) than a simple rest period ($d = 0.47$). In sum, at least for diastolic blood pressure, these analyses suggest that the optimal study to demonstrate anger versus fear specificity uses imagination as the induction method, a repeated measures design to induce anger and fear in the same subject, and an effective control imagery.

7.3 Bodily Sensations and Actual Somatovisceral Responses

Earlier in this chapter I reviewed studies on bodily sensations during anger compared to fear. The result was that anger was characterized in particular by feeling hot. The meta-analysis presented above and in Table 7.1 also suggested that face temperature is a distinguishing feature of the physiological anger response. Figure 7.1 shows the standardized endorsement rates of bodily sensations for anger and fear from Scherer and Wallbott (1994) together with the standardized physiological effect sizes of Table 7.1's columns "*d* Anger vs. Control" and "*d* Fear vs. Control." Six variables matched between sensation and physiological data sets, "breathing change" – respiration rate change, "feeling cold/shivering" – finger temperature change (reversed), "feeling hot/cheeks burning" – face

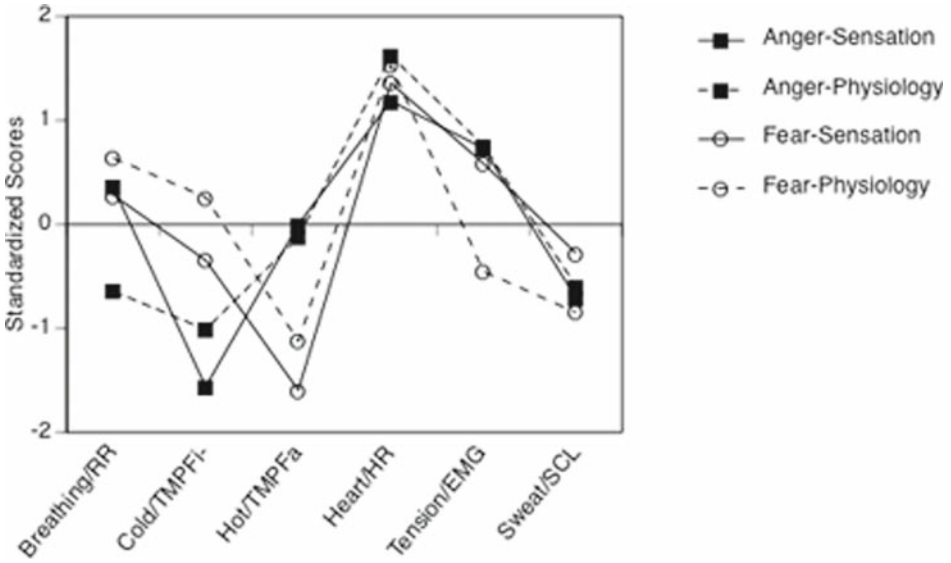

Fig. 7.1 Profiles of anger sensation, anger physiology, fear sensation, and fear physiology in matching sensation and physiology variables (*z*-standardized). Data for bodily sensations in anger and fear are from Scherer and Wallbott (1994), Table 8; data for physiological responses are from Stemmler's (2004) meta-analysis, see also Table 7.1 this chapter. See text for details

temperature change, "heart beating faster" – heart rate change, "muscles tensing/trembling" – muscle activity change, and "perspiring/moist hands" – skin conductance level change.

Overall, the profiles for anger and fear were quite parallel, with the notable exceptions of cold (peak for fear) and hot temperature (peak for anger) responses. Anger sensation and anger physiological profiles were highly correlated, $r = 0.85$; fear sensation and fear physiological profiles, $r = 0.78$. But between fear and anger profiles there were numerically high correlations suggesting the need to partial the bivariate correlations with respect to the remaining profiles. For example, the high correlation between anger sensation and anger physiology profiles to a marked extent could be due to the *indirect* influence of fear sensation and/or fear physiology. Partialing these two influences from the anger sensation and anger physiology correlation would provide a clearer picture of their *direct* association. Interestingly, anger sensation and anger physiology still correlated $r = 0.76$ as did fear sensation and fear physiology, $r = 0.74$, whereas all other correlations practically vanished. In particular, anger sensation correlated with fear physiology $r_{partial} = –0.02$; fear sensation correlated with anger physiology $r_{partial} = 0.29$. These purely descriptive data suggest that anger profiles of sensation and physiology have a common and specific core once the influence of fear is partialed out, and the same is true for fear. That is, on top of rather general, valenced bodily feelings (this feels good or bad) sensations seem to correspond quite well to the actual physiological changes. While the present analysis can only be suggestive, not conclusive, it is nonetheless consistent with the pan-cultural linguistic metaphors for the experience of anger based on somatovisceral physiology (Chapter 10 by A. Kövesces, this book) and justification for the inclusion of physiological imagery in anger-control psychotherapy that is the antithesis of anger-associated changes (e.g., cooling sensations in the body, Chapter 28 by E. Fernandez, this book.)

The functional relationship between physiological responses and self-reports of feeling has a long history (Cannon, 1927; James, 1884, 1894) that cannot be covered here in any detail. The debate was centered around the question whether physiological changes in emotion are *necessary* for feelings of emotion. Behind that question lurked the distinction between peripheralist versus centralist (Fehr & Stern, 1970) and – enlarged today – between biological versus cognitive explanations of emotion (Barrett, 2006). Early on, James advocated the study of spinal cord injured patients to solve this question. But the evidence from such studies is still equivocal: Somatovisceral activation seems to have at least some importance for the experience and action tendencies following emotions (Wiens, 2005). Various processes such as central sensitization, affective vulnerability, autonomic control, and autonomic representations in the brain all seem to be involved which have diverging effects on emotion outcome variables (Cobos, Sanchez, Garcia, Vera, & Vila, 2002; Nicotra, Critchley, Mathias, & Dolan, 2006).

Researchers have speculated how discrepancies between perceived/reported and objectively measured physiological changes might be explained. It could be the case that the attribution of bodily sensations to emotions is not based on the sensations per se, but on emotion schemata which might arise from individual history and general cultural influences (Rimé et al., 1990). Since cultural influences and learned stereotypes are expected to be similar for members of one culture, the ratings of study participants could be quite consistent. This was exactly what Rimé et al. (1990) found: Reports about bodily sensations during actual emotion episodes were not different from ratings of the stereotyped picture of such sensations. But, conversely, the source of these "stereotypes" could be just the common experience of prototypical bodily changes during strong emotions. Then the ratings of the stereotype would be better called "common experience" or "valid knowledge."

Cacioppo, Berntson, and Klein (1992) introduced another theoretical account for non-veridical reports, the Somatovisceral Afference Model of Emotion (SAME), which proposes three routes along which somatovisceral activity might shape bodily sensations. According to the SAME model, an emotional stimulus is subjected to a rapid but incomplete appraisal, which can evoke bodily

responses. These responses could be emotion specific, only partially differentiated, or completely undifferentiated. The pattern of this somatovisceral activation is fed back to the brain and evaluated, just as is the emotion-eliciting stimulus.

The goal of the cognitive evaluation is to arrive at an unequivocal statement about one's emotional state; this is best achieved with the label for a discrete feeling. In the case of an emotion-specific somatovisceral pattern, a discrete feeling would derive from accurate pattern recognition. In the case of a completely undifferentiated somatovisceral afference, "one labels, interprets and identifies this state in terms of the characteristics of the precipitating situation and of one's apperceptive mass" (Schachter, 1975, p. 530; Schachter & Singer, 1962), and thus arrives at a discrete feeling. If somatovisceral activation is only partially differentiated, emotional schemata would be prompted and would lead to emotional percepts with a high degree of definition.

7.4 The Issue of Specificity

The preceding paragraphs make it obvious that somatovisceral responses during anger – even in the laboratory and under such "non-provocative" conditions as imagery – can be quite strong. But there is also some degree of overlap in the responses to anger and other emotions, especially fear, leading some authors to the conclusion that physiological emotion specificity is at best unproven and probably a myth (Barrett, 2006; Ortony & Turner, 1990). In this section, I will argue for the case of specificity, at least for a small number of basic emotions including anger.

7.4.1 Physiological Considerations

7.4.1.1 The Autonomic Nervous System (ANS)

Up to the present day, claims for physiological emotion specificity are countered with the argument that the sympathetic nervous system generates only an undifferentiated and diffuse innervation of its target organs. However, physiological research contradicts this notion (Jänig, 2003, 2006).

The main task of the ANS is the distribution of specific signals of the central nervous system (CNS) to the end organs in order to achieve an optimal state of homeostasis. Interactions with the external world are carried out by motor systems. The endocrine system and the ANS support the motor systems by establishing an optimal internal milieu under changing conditions and demands. Both motor and endocrine as well as autonomic homeostatic regulations are coordinated under the control of the forebrain and they are integrated with representations of the perceptual world. Various efferent signals transmitted through pre- and postganglionic neurons are functionally separate from one another and therefore allow a very precise CNS control of the target regions. The CNS signals can be modified within the autonomic ganglia, which allows for self-regulation at various system levels.

7.4.1.2 Spinal and Supraspinal Control

The spinal cord contains many autonomic reflex centers. Supraspinal centers in the lower brain stem organize the homeostatic control of the cardiovascular, respiratory, and enteric system (e.g., blood pressure control through baroreceptor reflexes). Supraspinal centers in the upper brainstem, hypothalamus, and limbic system elicit distinct autonomic response patterns which coordinate organismic adjustments across somatomotor, autonomic, and endocrine response systems (Smith, DeVito, &

Astley, 1990). Jänig (2006) describes eight response patterns which are reliably induced during specific behaviors, among them exercise, vigilance, confrontation, or flight. Still higher centers are the anterior cingulate cortex, the insula, or the orbito- and ventromedial prefrontal cortex. These and other brain regions that exert an effect on autonomic response patterns are collectively called the "Central Autonomic Network" (Thayer & Brosschot, 2005).

The conclusion from this brief review is that the brain has the capacity to elicit specific and integrated autonomic responses. Because anger – as one of the basic emotions – is a psychobiological state, it functions to

- provide perceptual, cognitive, and organismic resources for the attainment of the emotion anger's goal (see below),
- signal conspecifics about one's own emotional state and an increased likelihood of aggressive responding,
- enhance intraorganismic information exchange, homeostasis and coordination, and
- protect the body against adverse consequences like injury when goal attainment becomes difficult or fails.

These actions and displays initiated by anger depend on a differentiated and well-functioning ANS.

7.4.2 Physiological Maps

For more than 50 years, multichannel physiological recordings have demonstrated that somatovisceral responses are strongly influenced by situations, individual differences, and individual-specific states (from, e.g., Lacey, Bateman, & van Lehn, 1953 to Foerster, 1985). The ANS is obviously able to produce specific patterns (Fahrenberg, 1987; Stemmler & Fahrenberg, 1989). Furthermore, somatovisceral patterns are both distinct and high stable.

Figure 7.2 presents an example of situational response specificity. Each label stands for the somatovisceral profile of one experimental situation. The axes are discriminant functions representing the plane that maximizes physiological differences between situations relative to variance among subjects within situations. The data are from Stemmler (1989). The anger induction was embedded in an anagram task, similar to the one described above. The fear induction consisted of a dramatic recitation of parts of E. A. Poe's "The fall of the house of Usher" dubbed with anxiety-provoking music (Prokoviev's second symphony) and ended with an unanticipated sudden darkness. The physiological anger patterns were located in the lower right quadrant, and the fear patterns, in the upper left quadrant. The patterns displayed clear distinctness and stability, as did patterns during repeated tasks, such as a simple numbers task (labels N1–N4), periods before a speech (B1–B3), or periods during a speech (D1–D3).

7.4.3 The Component Model of Somatovisceral Response Organization in Anger and Fear

It does not come as a surprise that at any given moment of time several factors may contribute to the physiological response pattern. Being angrily aroused when hit by a shopping cart while standing in line will result in different somatovisceral responses than being angrily aroused while imagining the same event in a comfortable armchair. Thus, the physiological pattern at any given moment does

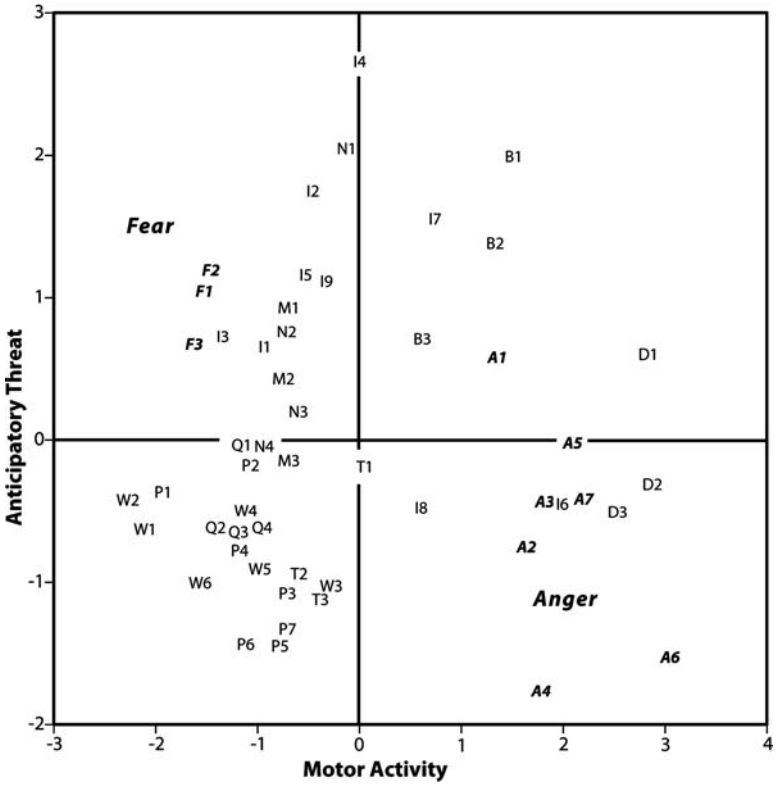

Fig. 7.2 Physiological map of 52 situations on the basis of 34 somatovisceral variables and 42 subjects (Stemmler, 1989). Bold labels denote somatovisceral patterns during induction periods for anger (A) and fear (F). A1 = Anagrams 1–15. A2 = 1st Interrupt. A3 = Anagram 16. A4 = 2nd Interrupt. A5 = Anagram 17. A6 = 3rd Interrupt. A7 = Anagrams 17–25. I6 = Instruction to wait after anger induction. F1 = Radio Play. F2 = Sudden darkness. F3 = Lights on. I = Instruction periods. M = Instruction for imagination task. P, Q = Pre- and poststimulus periods. W = Waiting periods. N = Number tasks. B, D, T = Before, during, after speech

not easily reveal which factors produced it. It could be that the differences between a physiological anger pattern and that of another emotion reflect just differences between the contexts in which the emotions were induced, and not differences between the emotions proper. Maybe physiological emotion differences do not exist after all, and all we see in the physiological recordings are emotion-unspecific effects? That is what Lang, Bradley, and Cuthbert (1990) implied when they wrote: "... that such physiological specificity in emotion may be tactical. That is, although specific action dispositions may be implicit in the conception of particular emotions (e.g., avoidance behavior with fear states, inhibition with sadness), they are also heavily modified by the demands of any specific context of expression" (p. 388).

The "contamination" of measures is quite a common problem in science. We may differentiate a theoretical and a pragmatic level to deal with it. Theoretically, I have proposed four different models of physiological emotion specificity, which are based on different assumptions about this confound (Stemmler, 1984, 1992b, 2003). For example, context-deviation specificity views emotion specificity as a conditional concept. An emotion "stimulus" is assumed to modify a preexisting context-bound physiological pattern. The pragmatic level, then, is to find ways to pull apart this confound. I have described experimental designs and validation strategies which constitute both necessary and sufficient conditions for claiming physiological emotion specificity (Stemmler, 1992b, 2003).

As postulated in the context-deviation model of physiological emotion specificity, various influences may impinge upon the activity of physiological variables. Just how such multiple stimuli combine in autonomic response amplitudes has been studied with two and three simultaneously acting stimuli in comparison with each stimulus administered separately (Foerster, Myrtek, & Stemmler, 1993; Myrtek & Spital, 1986). The results showed that in the majority of the physiological responses the effects of the stimuli combined synergistically and not just in an independent additive way.

In emotion research, which kinds of influences on physiological variables can be distinguished? The component model of somatovisceral response organization in anger and fear (Stemmler et al., 2001) postulates three classes of influences:

- The first component is characterized by the "non-emotional" context of an emotion induction, such as posture, ambient temperature, ongoing motor activity, or cognitive demands, which are not in the service of an emotion.
- The second component reflects a fixed (specific?) somatovisceral adaptation, which has at least two important functions: (1) the protection of the organism through autonomic reflexes and (2) the preparation of the organism for prototypical behaviors in the service of an emotion's goal attainment. These somatovisceral emotion signatures are recognizable probably only during a rather short temporal window during and after the arousal of an emotion and before actual behavior has started. It could be that the supraspinal autonomic response generators described above produce these signatures. In the case of anger this would be the generator for the confrontation pattern.
- The third component embraces contextual resources. These are effects of organismic, behavioral, and mental demands that are necessitated by the momentary situation in the pursuit of an emotion goal. This component allows for a flexible organization of bodily resources given the momentary situational circumstances. For example, where an opportunity for "fight or flight" arises, the defense reflex is likely to be activated because it prepares the organism "to cope with an emergency and specifically to perform the extreme muscular exertion of flight or attack" (Hilton, 1982, p. 159). Depending on the context as it is physically laid out and as the individual perceives and understands it, responses elicited by this third component may produce a marked overlap of physiological responses across emotions.

What functional value can be ascribed to the somatovisceral signature of anger? Anger is a neurobehavioral system which motivates individuals to avoid failure and pain by averting subordination under physically or socially caused harm and to gain superiority. Plutchik (1980) called the prototypical adaptation pattern of anger "destruction." Attack is a common behavioral response that requires a strong activation of sympathetic systems for its support. The behavioral response demands a persistent, isometric muscular tension. Circulatory responses under such conditions comprise an increased diastolic blood pressure and vascular resistance, which functions in opposition to the reduced effective perfusion pressures in the regions of intense muscular contraction (Buell, Alpert, & McCrory, 1986; Shanks, 1984). This means that the cardiovascular system operates to force blood to muscles whose contraction has squeezed and reduced their vascular supply. This autonomic pattern is seen already in anticipation of the handgrip task, which provokes this circulatory response (Mäntysaari, Antila, & Peltonen, 1988). The face turns red and hot, signaling an opponent the state of preparedness for attack.

Other important functional outcomes arise from interactions between the cardiovascular system and the brain. First, it is well known that rising blood pressure excites baroreceptors in the aortic arch and carotid sinus, which in turn have an inhibitory influence on pain thresholds (determined by pain ratings, see Ring, Edwards, & Kavussanu, 2008). In dangerous situations, this mechanism is certainly

advantageous. Second, behavioral and electrophysiological research shows a positive association between increased blood pressure and phasic and tonic cortical alertness, level of vigilance, and preparedness to react (Duschek, Meinhardt, & Schandry, 2006). Again, this psychophysiological mechanism is extremely important for quick and decisive action. One can imagine that when hyperaroused, this mechanism can also lead to a functional uncoupling of inhibitory, prefrontal executive control processes. Blind rage could be the consequence.

The direction of the association between blood pressure and brain activity is still unknown. On the one hand, the Central Autonomic Network exerts a steering function on the ANS, resetting, for example, set points of blood pressure regulation. On the other hand, catecholamines, especially noradrenaline, play an important role in the regulation of alertness and cortical activity. In addition, afferent sympathetic and parasympathetic fibers project into the prefrontal cortex, the insula, and the anterior cingulate (Critchley et al., 2003). Thus, there is a rich interconnection of the central and the autonomic nervous system, details of which will become more clear in the years to come.

7.4.4 The Adrenaline–Noradrenaline Hypothesis

Ax (1953) and Funkenstein, King, and Drolette (1954) suggested that the autonomic pattern during anger corresponds to the effects of a mixture of noradrenaline and adrenaline (see Footnote 1). In contrast, fear would be characterized by an autonomic pattern resembling the effects of adrenaline alone. This hypothesis was bolstered by interspecies comparisons of the relative concentrations of adrenaline and noradrenaline in the adrenal medulla. Baboons, rabbits, guinea pigs, rats, and humans possess a larger proportion of adrenaline, whereas lions, small sharks, and whales have a larger proportion of noradrenaline (Funkenstein, 1956). The observation that the noradrenaline level of ice hockey players rose markedly more than their adrenaline level, whereas the opposite was true for the coach and the substitutes, also seemed to fit the hypothesis well (Elmadjian, Hope, & Freeman, 1957). However, vigorous movement instead of anger aggression might have been responsible for these effects (see also Glass et al., 1980; Ziegler, Lake, & Kopin, 1977).

Wagner's (1989) discussion of studies on hormone secretion during emotions found them to mostly support the adrenaline–noradrenaline hypothesis. Other studies were not compatible with this interpretation, however. For example, Chessick, Bassan, and Shattan (1966) induced anger and fear, also infused their subjects with adrenaline and noradrenaline, then compared the ensuing autonomic response profiles. There were no marked similarities between emotion and catecholaminergic infusion profiles. In a review of many studies performed in her laboratory, Frankenhaeuser (1979) disputed the notion of a selective secretion of adrenaline and noradrenaline in emotional states.

The meta-analytically derived somatovisceral differences between anger and fear in Table 7.1 agree with the expected differential effects of the catecholamines in the following responses: diastolic blood pressure and total peripheral resistance (higher in anger than fear) as well as cardiac output and respiration rate (higher in fear than anger). With regard to heart rate and finger skin temperature, the direction of the empirical results does conform to expectations, but the results do not reach significance. In sum, the adrenaline–noradrenaline hypothesis of anger and fear turns out to be a good but not an exhaustive hypothesis to account for the characteristics of the respective autonomic responses.

7.4.5 Anger and Alpha-Adrenergic Activation

Another mechanism-based model of anger specificity could refer to major receptor types in the ANS. For example, alpha-adrenergic, beta-adrenergic, and cholinergic cardiovascular tone might be described as distal mechanisms and diastolic blood pressure as a proximal variable. Blockade studies suggested that diastolic blood pressure rises both with alpha-adrenergic tone (Nelson, Silke,

Hussain, Verma, & Taylor, 1984) and with loss of vagal tone (Knoebel, McHenry, Phillips, & Widlansky, 1974; Levine & Leenen, 1989). In contrast, diastolic blood pressure is not controlled by beta-adrenergic tone under resting conditions (Silke, Nelson, Ahuja, Okoli, & Taylor, 1983). Thus, diastolic blood pressure will rise both during an alpha-adrenergic state and during a state of vagal withdrawal.

If cardiovascular activity is governed to an important degree by the action of alpha-adrenergic, beta-adrenergic, and cholinergic influences, or "activation components," then anger with its strong vascular component could be associated with an alpha-adrenergic state, probably accompanied by a marked beta-adrenergic state. In order to test this notion, Stemmler (1992a) induced anger during the third of four sessions 1 week apart and each time under a different combination of partial dual receptor blockades.[2] The sessions were identical with the exception of the anger induction right before a sentence completion task. Subjects were harassed for moving too much:

> "What's going on here! We've had it! The whole recording is totally messed up – we'll have to junk it. Why do you think we keep asking you to sit still?! You'd think we could expect just a little more cooperation from a med-student. Now we have to go through the whole procedure again! You'll have to come back one more time – but you know we can't pay extra for it. – Now let's see if at least the next task will work. – But try to control yourself a little bit this time – sit still and keep your arms as relaxed as possible and don't talk!" (Stemmler, 1992a, p. 182)

Compared to the control group angered subjects under Placebo showed a significant increase in the activation of the beta-adrenergic component (see Fig. 7.3). Unexpectedly, the anger group under Placebo did not demonstrate an alpha-adrenergic activation (Panel a). In the "alpha-free" group,

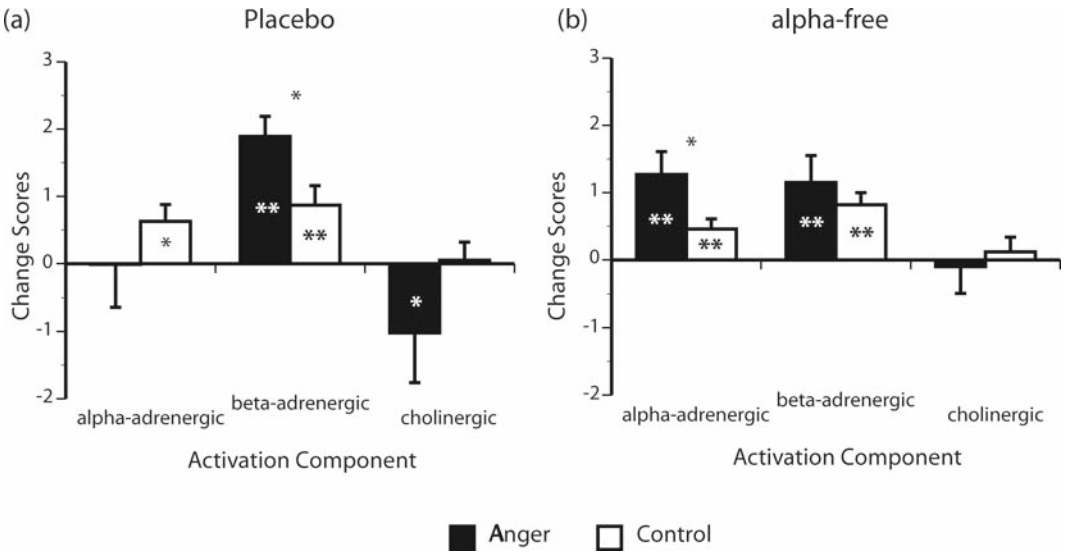

Fig. 7.3 Effects of an anger induction on three cardiovascular activation components in comparison with a control condition with identical context (Stemmler, 1992a). Panel (a) shows data of subjects under Placebo, Panel (b), under partial beta-adrenergic (60 mg Propranolol) and cholinergic blockade (1 mg Atropine sulfate; "alpha-free"). Each bar represents the mean of 12 subjects (+/– SEM). Stars inside/outside of bars denote significant differences from zero/between anger and control groups. Change scores are task minus prestimulus scores

[2]In a crossed carry-over design and randomized across sessions, subjects were given either Placebo, partial beta-adrenergic and cholinergic blockade ("alpha-free"), partial alpha-adrenergic and cholinergic blockade ("beta-free"), or partial alpha- and beta-adrenergic blockade ("chol-free").

however, the beta-adrenergic activation was reduced to the level of the control group, but an alpha-adrenergic activation became apparent (Panel b). Thus, the partial beta-adrenergic receptor blockade *unmasked* the alpha-adrenergic activation, a phenomenon not unknown in the literature (Martin et al., 1974). These results suggest that anger is indeed characterized by a strong alpha-adrenergic influence, which especially in real life, but probably less so in imagery induction contexts, is masked by a strong beta-adrenergic activation.

7.4.6 The Problem of Unspecific Somatovisceral Responses

How tenable is the request that a *complete* differentiation by autonomic activity of discrete emotions is necessary to demonstrate their biological distinctness? Such a demand is not well founded. Fact is that physiological emotion responses are distinct in only a subset of recordable variables (*incomplete* differentiation). Nevertheless, the biological function of physiological emotion responses emerges only when all variables, specific and unspecific ones, are considered. For example, a heart rate increase without a strong blood pressure boost may indicate vagal withdrawal, whereas a heart rate increase together with a strong blood pressure boost may indicate a combined alpha- and beta-adrenergic activation. Without consideration of the unspecific heart rate increase the former case would no longer be interpreted as vagal withdrawal, but as a non-response.

Unspecific responses can discriminate between control and emotion induction conditions, but not between inductions of different emotions. The meta-analysis of anger and fear noted unspecific responses for heart rate, systolic blood pressure, number of unspecific skin conductance responses, and finger skin temperature. These responses can very well be a genuine part of the physiology of these emotions, for example, since they prepare for action (*fight and flight*). In order to understand the physiological "signature" and the functional meaning of an emotion, both specific and unspecific responses need scrutiny.

7.5 Conclusions

As I have tried to show, anger has a distinct somatovisceral physiology which is also sensed quite well by children and adults. At its core is an alpha-adrenergic activation, which enables continued isometric exertion of skeletal muscles. In addition, the rise in blood pressure has effects on the brain, for example, elevated pain thresholds. There are also positive associations between blood pressure and EEG arousal, which mark an increase in sustained alertness, vigilance, and preparedness to react. The prefrontal cortex, the insula, and the anterior cingulate could be the brain regions where cardiovascular arousal, regulation of pain, and cortical activation interact. These coordinated changes have a functional value for the pursuit and finally the attainment of the goal of anger: To motivate individuals to avoid failure and pain by averting subordination under physically or socially caused harm and to gain superiority. For survival and social organization of men and mice this is so important a goal that it is deeply embedded in the mammalian brains (Panksepp, 2007).

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