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Electrodermal Activity as an Indicator of Emotional Processes

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Abstract The differentiation of emotions by means of psychophysiological measures has been only moderately successful so far. A major reason for this dilemma may be the lack of appropriate neurophysiological modeling for the various autonomic nervous system based measures being used in emotion research.The aim of the present article is to provide such a neurophysiological background for electrodermal activity which has been frequently used as an indicator of emotional processes. First, the literature is reviewed with respect to the usability of electrodermal measures as an indicators of emotion. Second, the neurophysilogical sources of electrodermal phenomena in general are described. Electrodermal activity has different origins in the central nervous system, a limbic-hypothalamic source that dominates during negative emotions as opposed to a premotor and basal ganglia source being predominantly active during positive emotions. Panksepp's model of four basic emotive systems is adopted for demonstrating subcortical structures and pathways possibly involved in the elicitation of both kinds of electrodermal activity in comparison with cardiovascular in dicators of emotional processes.

I. Introduction

Research into the psychophysiology of emotions dates back to the end of the 19th century. In 1884 and 1885, the American philosopher and psychologist William James and the Danish Physiologist Carl Lange independently proposed a very similar physiological theory of emotions. According to the James-Lange theory, emotion inducing sensory stimuli are first received by the cortex that triggers changes in visceral organs and skeletal muscles. The feedback from those peripheral organs travels back to the cortex where the experience of emotions is caused. The James-Lange theory has often been criticized because the autonomic responses occur too slowly to be the cause of emotional feelings. As an alternative,

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Walter Cannon and Paul Bard proposed in 1927/28 what is called the thalamic theory of emotions. According to the Cannon-Bard theory, emotional stimuli excite both the feeling of emotions in the brain and their expression via the autonomic and somatic nervous system in parallel. Their theory could have been called hypothalamic as well since they regarded the hypothalamus as the source of visceral and skeletal muscular response patterns to emotion eliciting stimuli.

A major implication of both theoretical approaches is the existence of a distinct somato-visceral response pattern for each emotional quality. However, in almost one century of research into the psychophysiology of emotions, such distinct patterns have barely been found. A possible solution for this particular problem may be not to look out for patterns of single emotions but try to distinguish a few emotional dimensions by means of their psychophysiological patterns. Determining the dimensionality of emotions has resulted in either two (Lang, 1968, Russell, 1980), three (Wundt, 1896; Schlosberg, 1954) or four dimensions (Osgood, 1966; Sokolov, 1992). There is a general agreement on two major dimensions; i. e., hedonic value and arousal. However, most of this kind of work has been performed by the application of multidimensional scaling on emotion related adjectives or facial expressions. Psychophysiological patterns, on the other hand, have been mainly related to single emotional qualities such as fear, anger and joy (Levenson, 1988).

It is far beyond the scope of the present paper to provide a general solution for the problem of finding appropriate psychophysiological patterns for different emotions or emotional dimensions. The present author is, however, deeply convinced that such a question cannot be addressed successfully without uncovering the neurophysiological background of the psychophysiological phenomena under investigation. Therefore, the major aim of this paper is to familiarize the reader with the central nervous system (CNS) mechanisms underlying a major autonomic nervous system (ANS) concomitant of emotions, i. e., the electrodermal activity (EDA), and to provide a theoretical framework for its observed indicator functions based on neurophysiological considerations.

2. Electrodermal measures as concomitants of emotional processes

Among the various psychophysiological measures that have been used in emotion research, EDA recording, mostly performed as skin conductance measurement, has played a major role (Boucsein, 1992). Several reasons may account for this:

The limbic and hypothalamic structures being responsible for the elicitation of EDA in the CNS are also regarded as major determinants of emotional behavior.

Palmar skin from which EDA is normally recorded constitutes a specialized display for emotional responses. This is also the case for plantar sites.

EDA depends on sweat gland activity which is purely innervated by the sympathetic branch of the ANS. Therefore, the amount of EDA recorded corresponds unobtrusively to the strength of an elicited emotion. There is no parasympathetic counter-regulation as in other ANS innervated organs such as the heart.

A very successful attempt to quantify the strength of emotions by the use of EDA was made by Traxel (1960). Eighty male subjects were visually presented 20 pairs of words in sequence for seven seconds each, while EDA was recorded. After each pair of stimuli the subjects had to decide which word, if any, elicited a greater amount of

emotional experience. The judgments were evaluated by the paired comparisons technique, and the obtained strength of emotion was correlated with the differences between EDR amplitudes elicited by the particular words in a specially developed a-posteriori paired comparisons technique. A linear relationship was found between the EDR amplitude and the psychophysically determined strength of emotion. After an additional square-root transformation, the correlation between subjective and electrodermal measures of emotion strength increased from an initial r = .94 to r = .99 which is just perfect.

As long as there is no clear evidence for the existence of distinct physiological patterns for different emotions, the psychophysiological investigation of emotional states cannot be performed satisfactorily by means of physiological recording only. Different emotions may elicit the same amount of general arousal, thus becoming possibly indistinguishable with respect to their ANS concomitants. Therefore, in classic psychophysiological emotion research, emotional quality is determined via subjective variables while their quantitative properties are measured by ANS parameters.

Various attempts have been made to obtain emotion specific patterns of physiological reactions, since Ax (1953) tried to differentiate the effects of anger and fear by means of ANS variables. In his experiment, a total of 43 subjects of both genders received two conditions in Counterbalanced order. In one of these, subjects were annoyed by means of an obnoxious experimenter. In the other condition they were frightened by having electric shock applied to their finger, accompanied by sparkles near the subject, and the experimenter exclaiming that this was a high voltage short-circuit. Differences between anger and fear appeared on seven of 14 physiological measures recorded, whereby anger elicited a significant increase of NS.EDR frequency (the frequency of non-stimulus specific, i. e., spontaneous changes in EDA) as compared to fear. In turn, the skin conductance level (SCL) was significantly higher under fear as compared to anger conditions. However, large individual differences were observed: for example, some of the fearful subjects evidenced higher changes in EDA than in respiration while others showed the reverse pattern. Nevertheless, Ax attributed the observed ANS reactions to certain underlying endocrine patterns as outlined in the next paragraph. For a summary of the methodological issues in this kind of peripheral physiological differentiation of emotions, see Wagner (1989), who also summarized results on anger vs. fear and on other emotions.

A systematic comparison of results taken from eight multivariate studies attempting to differentiate experimentally induced anger and/or fear by means of ANS variables, including Ax's (1953) study, was performed by Stemmler (1984). The results are presented in Table 1. In most of the studies, both emotions yielded higher cardiovascular output (increases in heart rate and blood pressure) compared to resting conditions. An increased NS.EDR frequency has been found for both emotions in two of the studies (D & E), while not differing from resting condition in another study (B). Three of the four studies with a direct comparison of anger and fear found an elevated heart rate (HR) during fear compared to anger (F, G & H). Two of the four studies found a lower diastolic blood pressure and lower tonic electromyographic (EMG) values (F & B), while three of them (F, G & H) found an increased SCL during

Table 1. Comparison of psychophysiological profiles for fear and anger from eight different studies (A-H). + means an increase, - means a decrease, 0 means no significant change of the values of the parameter in question. A bracket indicates marginal significance. Max.= HR maximum, Min.= HR minimum. Adapted from Stemmler (1984).

Study No. of subjects	Fear vs.control				Anger vs. control		Fear vs.anger			
	A	B	C	D	B	E	F	G	H	B
	35	18	33	128	18	48	43	48	20	18
Systolic blood pressure		+	+		+	0	0	0		0
Diastolic blood pressure		+	+		+	+	-	0		-
Pulse volume	+	+			0		0	(+)		0
Heart rate	+	+	+	+	+	+	Max. 0 Min. +	(+)	Max. 0 Min. +	+
Respiration rate		0			+		+	0		0
Finger temperature							0	0		
Axillary temperature		0			-					+
Forehead temperature		0			0		0	0		0
NS.EDR frequency		0		+	0	+	-		+	0
Skin conductance level		0		0	0		+	(+)	+	0
Tonic electromyogram		0	+		0		-	0		-
Phasic electromyogram		0			+		+			0

fear compared to anger. The two studies that directly compared NS.EDR frequency for both emotions (F & H) yielded opposite results. The existence of a so-called adrenaline-noradrenaline pattern under anger vs. an adrenaline pattern under anxiety as supposed by Ax could only partially be confirmed. By the way, Ax (1953) did not obtain measures of catecholamines. He simply concluded that the ANS pattern under anger resembled the expected response to adrenaline and noradrenaline injections, while the ANS pattern under fear resembled the response had adrenaline been injected.

In his own experiment, Stemmler (1989) induced three emotions (fear, anger, and pleasure) to 42 female subjects in a repeated measures design. Fear was induced by tape presentation of a fear-evoking short story being accompanied by an unannounced darkening of the room. Anger was induced by presenting a series of anagrams, most of which were not solvable, which, however, could not be detected by the subjects during the short presentation. Pleasure was induced by positive reinforcement and the announcement of increased payment at the end of the study. Various peripheral physiological measures including

palmar skin conductance were obtained. To obtain an additional objective measure of the forehead anxiety sweat , another EDA recording was taken from the forehead. Recordings were continuously taken during the induction of emotions and during interspersed resting phases as well. In addition, several standardized ratings of emotional states were applied. These subjective measures yielded their most pronounced results in the appropriate situational context. However, their sensitivity was less during fear and anger conditions as compared to the pleasure condition. Out of the 34 parameters that were extracted from the physiological recordings, the 14 which significantly differentiated between the reference phases for the three emotions were included in a discriminant analysis, which resulted in different profiles for fear and anger. However, no significant differences between the profiles for pleasure and the other emotions emerged. Multivariate comparisons yielded low EMG values together with peripheral vasoconstriction, low skin temperature, and a low SCL taken from the palms, but an increase of forehead SCL during the fear state. During anger, the forearm EMG and the vasodilatation at the hand and the forehead were increased, and an increase of forehead SCL appeared, too. The NS.SCR frequency did not belong to the variables discriminating between fear and anger. Stemmler's results did not sufficiently fit those of Ax (1953) with respect to EDA, although the palmar SCL was lowest under fear in both studies.

Recently, Boucsein and Baltissen (1998) performed a series of studies in order to simultaneously differentiate both qualitative and quantitative aspects of emotions by means of psychophysiological measures. First, 23 slides from the Lang, Oehman and Vaitl (1988) series were judged by 120 subjects (30 male, 90 female) with respect to their properties to elicit different emotional qualities. In a second study, 24 subjects (12 of each gender) were presented two slides from each of the following categories: Erotic happiness, nurturant happiness, fear, sadness, disgust and emotionally neutral. The slides were presented twice in randomized order. During the first presentation, HR, EDA and three facial EMGs (taken from zygomatic major, corrugator supercilii and lateral frontalis muscles) were recorded. The second presentation was performed to obtain subjective ratings of the emotions elicited. In general, the physiological measures used in this study sufficiently differentiated between positive, negative and neutral emotional slides, except for fear. Therefore, in a third study with another 24 subjects (14 male, 10 female), the same slides were presented under two different arousing conditions for each half of the subjects in order to vary the quantitative (arousal) aspect of emotionality. High arousal was induced by 80 dB white noise, while 50 dB noise served as a low-arousal inducing control condition. The same physiological measures as in the second study were recorded, except for the frontalis EMG which was replaced by the finger pulse volume. Instead of generally enhancing the psychophysiological responses, 80 dB noise evoked rather different patterns for the different emotional qualities. Interestingly, the differentiation by means of EDA measures was better under the low-arousal condition but HR differentiated better between the emotion-inducing slides under the high arousal condition. This parallels the findings in the clinical domain that EDA recordings provide highly sensitive parameters for minimal changes such as in general anxiety states, while HR has its

major indicator function in high-arousal states of anxiety such as phobias (Boucsein, 1992). In general, fear and disgust inducing slides elicited the most pronounced initial EDR amplitudes. This is in accordance with EDA being regarded as the psychophysiological system with a specific reactivity to negative emotions. However, if an evaluation period as long as 10 seconds was taken into account, presenting erotic happiness-inducing slides resulted in greater EDA as compared to negative emotion-inducing slides. This effect was attenuated by noise.

There are several consequences to be drawn from the hitherto performed research into the use of EDA as an indicator of emotion. First, the level of arousal, i. e., the quantitative aspect of emotionality, is critical to its indicator function. Second, the evaluation interval can be critical as well. Initial response strength may be different from the overall response. Third, a single measure or a single psychophysiological system cannot account for all changes elicited by the different emotional qualities. Therefore, multivariate research is needed in this area and the sensitivity of different measures within each system ought to be determined as, for example, in stress-strain research (see Boucsein & Backs, 1999). The problem of how to parallel qualitatively different states of emotion with respect to their quantitative properties remains central to this kind of research. In addition, particular conditions of the experimental design such as counterbalance of the sequence of different emotions may have a strong influence on the results. Various kinds of response specificity as already observed by Ax (1953) and systematically treated by Engel (1972) and Fahrenberg (1988) further complicate multivariate research of emotional states. However, there is no alternative in sight.

Another potential differential indicator function for EDA in emotional states stems from research on emotional expression, which does not only play a role in normal subjects but also in psychopathology, especially in schizophrenia (Turpin, Tarrier & Sturgeon, 1988). The hypothesis of facial expression being not only a concomitant of emotion but also having a role in regulating the emotional experience itself, dates back to Darwin, and was taken up again by Gellhorn (1964) and Izard (1971). These authors suggested that the facial actions trigger central nervous circuits that elicit ANS changes as well as the emotional experience. Phasic electrodermal parameters have gained an important role in the research into the so-called facial feedback hypothesis of emotions (Vaughan & Lanzetta, 1981).

Lanzetta, Cartwright-Smith, and Kleck (1976) investigated the influence of an instruction to manipulate one's facial expression on EDRs during the anticipation and application of electric shocks of different intensities. They performed three subsequent experiments, the third being the most carefully controlled one. In this study, 10 subjects of each gender received 2 seconds electric shocks of 33 %, 66 %, and 99 % of a previously established individual tolerance limit. They were announced 8 seconds prior to their presentation displaying the numbers 1, 2, and 3, respectively. During the 10 seconds following each of the 15 baseline trials, the subjects rated their discomfort caused by the shock on a scale from zero to 100. The session was videotaped, and the discomfort experienced was rated afterwards by six judges (four males, two females) who neither knew the shock intensity nor the subject's rating. The anticipatory EDR was computed as an increase from the average SCL 2 minutes prior to the beginning of

shock application to the average SCL from 2 minutes prior to the beginning of the slide projection. The latter SCL was used as a reference for the EDR to shock application, thus being subtracted from the average SCL 6 and 8 minutes after its application. The anticipatory EDR as well as the EDR following the shock itself increased monotonous with shock intensity; as did the self-rating of experienced discomfort as well as the judges' rating of observed discomfort. During another 26 trials with counteralanced intensities, subjects had either to hide or to amplify their experienced discomfort, dependent on different colors of the slides, by means of their facial expression. Those manipulations were not only successful with respect to influencing the judges appropriately; in addition, the EDR amplitude, especially the one following the shock, was significantly lower when subjects tried to hide their discomfort as compared to extensive facial pain expression.

Monotonious relationships between the degree of mimic expression of experienced pain and EDR strength were confirmed in other studies of the Lanzetta group (Kleck, Vaughan, Cartwright-Smith, Vaughan, Colby & Lanzetta, 1976; Colby, Lanzetta & Kleck, 1977; Orr & Lanzetta, 1980). In addition, Vaughan and Lanzetta (1981) investigated the effects of facial expression on vicarious emotional arousal. They exposed three groups of 20 subjects each to a videotaped model, giving them either instructions to amplify or to inhibit their own facial muscles when the model appeared to be shocked, while the third group received no facial instructions. The fact that instructions were effective could be shown by means of EMG recordings from three facial muscles (orbicularis oculi, masseter, and medial frontalis). The model appeared to undergo a word-shock classical conditioning paradigm with four practice, 10 acquisition, and 10 extinction trials, during which the subject's EDRs were recorded. All groups showed greater electrodermal responsivity on CS+ trials (i.e., the trials where a conditioned stimulus was presented with an unconditioned stimulus) as compared to CS-trials (i.e., trials without the presentation of an unconditioned stimulus). However, this difference was more pronounced for the group having received the "amplify" instruction as compared to either "inhibit" or uninstructed subjects. Additionally, in the extinction phase a tendency appeared for the amplify subjects to show a greater EDR to CS+ than to CS-, while the other groups did not. Those results gave support to the facial feedback hypothesis with respect to autonomic reactivity in general, since HR recordings showed a similar pattern. This is rather close to the James-Lane view, stating that different emotions are caused by muscular reafferents to the CNS (McFarland, 1981, p. 289). However, the above results do not unambiguously show whether EDRs in fact serve as indicators for emotion, or only as an ANS correlate of increased facial muscle activity, regarded as artifactual.

In contrast to the results of the Lanzetta group, Buck and his colleagues found inverse relationships between EDA and facial reactions to emotion inducing slides (summarized by Buck, 1980). Buck and Miller (1974) ran 32 subjects of each gender in randomly chosen sender-observer pairs, while their facial reactions to 25 slides were videotaped. There were five slides of each of five categories (sexual, pleasant landscape, pleasant people, unpleasant injuries, and unusual photographic effects) presented in randomized order. The observer rated the sender's mimic on a

nine-point scale from pleasant to unpleasant, and the sender performed the same rating while watching the slides. Communication accuracy, measured by the correspondence between the sender's and observer's ratings, correlated significantly negative with the sender's EDA, however only in case of male sender (r = .74). As already found in a previous study (Buck, Savin, Miller & Caul, 1972), the correlation of communication accuracy with HR was insignificant (r = .27 for male senders). However, the subjects reacted more with their cardiovascular than with their electrodermal system during a post-experimental verbal description of the slide contents. Buck and Miller (1974) interpreted this as being consistent with other findings (e.g. Campos & Johnson, 1967) which showed that emotional arousing visual stimuli affect EDA more than HR responding, while a requirement to make overt responses (including verbal ones) is more likely to elicit HR acceleration than EDRs. This also parallels the results of Fowles (1980) with respect to the specific sensitivity of HR and EDA.

Buck et al. (1974) offer a conditioning explanation for the negative correlation between facial expressiveness and electrodermal responding to stimuli, assuming a socially learned inhibition of overt affective responses. Inhibitory responses may become CSs that elicit similar autonomic responses as the former unconditioned stimulus (UCS). Thus, together with a masked facial expression, a large ANS response will appear, which is more likely an electrodermal than a cardiovascular one because no action (including facial muscles) is elicited. This social learning should have appeared early during life, since Buck (1977) found comparable negative correlations between EDR amplitudes and communication accuracy even in preschoolers, giving nonverbal messages via spontaneous facial expressions and gestures to their mothers. Following this line, Buck (1980, p. 821) concluded that facial expression is more likely to serve as a readout device than as a feedback device, and that our facial emotional expression reflects central processes, not the reverse.

Winton, Putnam and Krauss (1984) tried to explain the opposite findings of the Buck group and the Lanzetta group by their different experimental manipulations of the two major dimensions of affective experience, i. e., the intensity (arousing) and the evaluative (pleasant vs. unpleasant) aspect of emotions. Lanzetta and his colleagues used expressiveness ratings as measures of shock painfulness, thus reflecting the intensity dimension of the affective response, which is positively correlated with the EDR amplitude. In the Buck studies, however, the slide stimuli presumably evoked affective changes not only on the intensity but also on the evaluative dimension, while the subject's as well as the judge's ratings may have specifically concerned the evaluative dimension. As suggested by the Laceys (e.g. Lacey & Lacey, 1970), phasic HR would be the more appropriate measure for this dimension than phasic EDRs.

To further test this hypothesis, Winton et al. (1984) performed an experiment with 24 male subjects viewing a series of 25 emotionally evocative slides in a paradigm similar to that of Buck et al. (1972, 1974). Facial expressions were covertly videotaped and were later shown to 90 judges of both genders. HR and EDA were continuously recorded, and subjects rated pleasantness of slides 10 seconds after slide onset on a seven-point Likert scale. While HR increased monotonous with increasing subjec- tive pleasantness of the slides

shown. EDR amplitude (between 1 and 5 seconds following stimulus onset) yielded a U-shaped course, being larger in highly pleasant and in unpleasant stimuli as compared to neutral ones. The HR results paralleled the judges' ratings of facial pleasantness, while ratings of facial intensity showed the same U-shaped relationship to slide pleasantness as the EDR amplitudes. These results could be replicated in two subsequent slide rating studies of Putnam and co-workers (cf. Winton et al., 1984).

The observed curvilinear relation of the EDR amplitude with pleasantness could have been interpreted as supporting Schachter and Singer's (1962) theory of emotion. This cognitively oriented theoretical view regarded physiological arousal as a necessary condition for the elicitation of an emotional state, the nature of which, however, is determined by situational cues. EDR amplitude being high in extreme self-report categories and low in moderate categories, as observed by Winton et al. (1984), would have aligned along those predictions, if one would have regarded EDA as an index of general arousal. Instead, when considering HR together with EDR results, the conclusion has to be that different emotional states correspond to different patterns of autonomic activity.

Similar relationships between emotional valence and HR as well as between EDA and emotional arousal obtained by Winton et al. (1984) were found by Greenwald, Cook and Lang (1989). Forty-eight subjects of both genders were presented 21 slides for six seconds each, while facial EMG (zygomatic major and corrugator supercilii), HR and EDA were continuously recorded. Based on results from a previous validation study, valence and arousal dimensions of the slides were regarded as relatively independent ($r = .01$ for males, and $r = .24$ for females). Subjective ratings were recorded by a computerized self-assessment technique. Larger changes in EDA were significantly related to increased arousal ratings, the effect being pronounced solely for males. Changes in EDA were not related to valence ratings as HR changes were. Thus, EDA appeared as a sensitive and specific measure of arousal, while phasic HR acceleration proved to be a sensitive and specific measure of emotional valence.

In three subsequent experiments performed with a total of 62 subjects (27 males and 35 females: actors, emotional facial expressions researchers, students, and non-student adults), Levenson, Ekman and Friesen (1990) studied subjective and autonomic concomitants of voluntary facial configurations for the negative emotions anger, fear, sadness and disgust, and for happiness and surprise as positive emotions. EDA was measured together with HR, finger temperature and forearm flexor muscle tension. EDA clearly differentiated between positive and negative emotions, being higher in the negative ones, while HR was lower in the disgust condition as compared to the other negative emotions, not clearly reflecting the emotional valence as in the Winton et al. (1984) study.

An attempt to determine an individual's most reactive ANS channel (electrodermal or cardiovascular) was made by Levis and Smith (1987), using the balloon-burst test to pre-classify their subjects as high electrodermal responders, high HR responders, high responders in both channels, or low responders in both channels. In a subsequent presentation of a fear-eliciting slide (a man who died in an accident), those subjects defined as high responders on a given channel showed greater reactivity on that particular channel as compared

to low responders. Given the background of the apparent differential indicator functions of electrodermal and cardiovascular variables (Fowles, 1980; Boucsein, 1992), the use of multivariate methodology in psychophysiological research into emotional states is strongly advocated, despite some inconsistencies between the appropriate studies as described at the beginning of this section. Within this frame, EDA may also not be regarded as a unitary ANS variable. Instead, different parameters could have different validities with respect to various emotional states, as observed by Stemmler (1989). However, a theoretical framework for the use of different tonic EDA measures together with phasic measures of EDR amplitude and shape to explain different components of variance in various emotional contexts is still lacking.

3. Central origins of electrodermal activity

Electrodermal phenomena are primarily influenced by parts of the CNS that are involved in the classical sympathetic elicitation of sweat secretion. However, various subcortical and cortical regions contribute to the complex system that regulates EDA. A thorough review of the evidence with regard to different brain levels has been provided by Venables and Christie (1973). The present author will add more recent results and provide an integrative view on the origin of both tonic and phasic electrodermal phenomena in the CNS.

The hypothalamic areas that exert thermoregulatory control can be considered to play a major role in the elicitation of EDA. These areas are influenced by the limbic system, which is regarded as the neurophysiological basis for emotional phenomena. Limbic influences on sweat eliciting hypothalamic areas mainly stem from the hippocampus (which is included in the so-called Papez circuit), from the amygdala and from anterior limbic and infralimbic cortical areas (for an overview, see Boucsein, 1992). Evidence from animal research points to antagonistic actions of these structures on EDA (Yokota, Sato & Fujimori, 1963; Yokota & Fujimori, 1964; Wang, 1964). Those are excitatory influences from the amygdala and inhibitory influences from the hippocampus, the latter being supposed by Wang (1964) to stem from the mamillary body via the fornix. However, CNS origins of EDA include other subcortical and also some cortical regions as well.

Figure 1 summarizes the present knowledge about the excitatory and inhibitory sweat centers, integrating various degeneration and stimulation studies with anesthetized and unanesthetized animals and evidence from neurological patients. Cats have been used as preferred animals for research into the CNS elicitation of EDA, since thermoregulatory sweating can be disregarded in cats (Jaenig, Sundloef & Wallin, 1983). Therefore, electrodermal phenomena to be recorded from the cat's foodpad are analogous to what is called emotional sweating in humans (Roy, Sequeira & Delerm, 1993). On the other hand, cats are not a suitable species for studying peripheral mechanisms of EDA since there is no sodium reabsorption in the cat's sweat gland duct (Sato, 1977) and their sweat glands have been suggested being apocrine instead of eccrine (Wang, 1964) which, however, remains debatable (Edelberg, 1972).

Excitatory centers for the origination of EDA are located in the cat's anterior hypothalamus, in a dorsal thalamic region, and in anterior limbic and infralimbic as well as in sensorimotor neocortical areas.

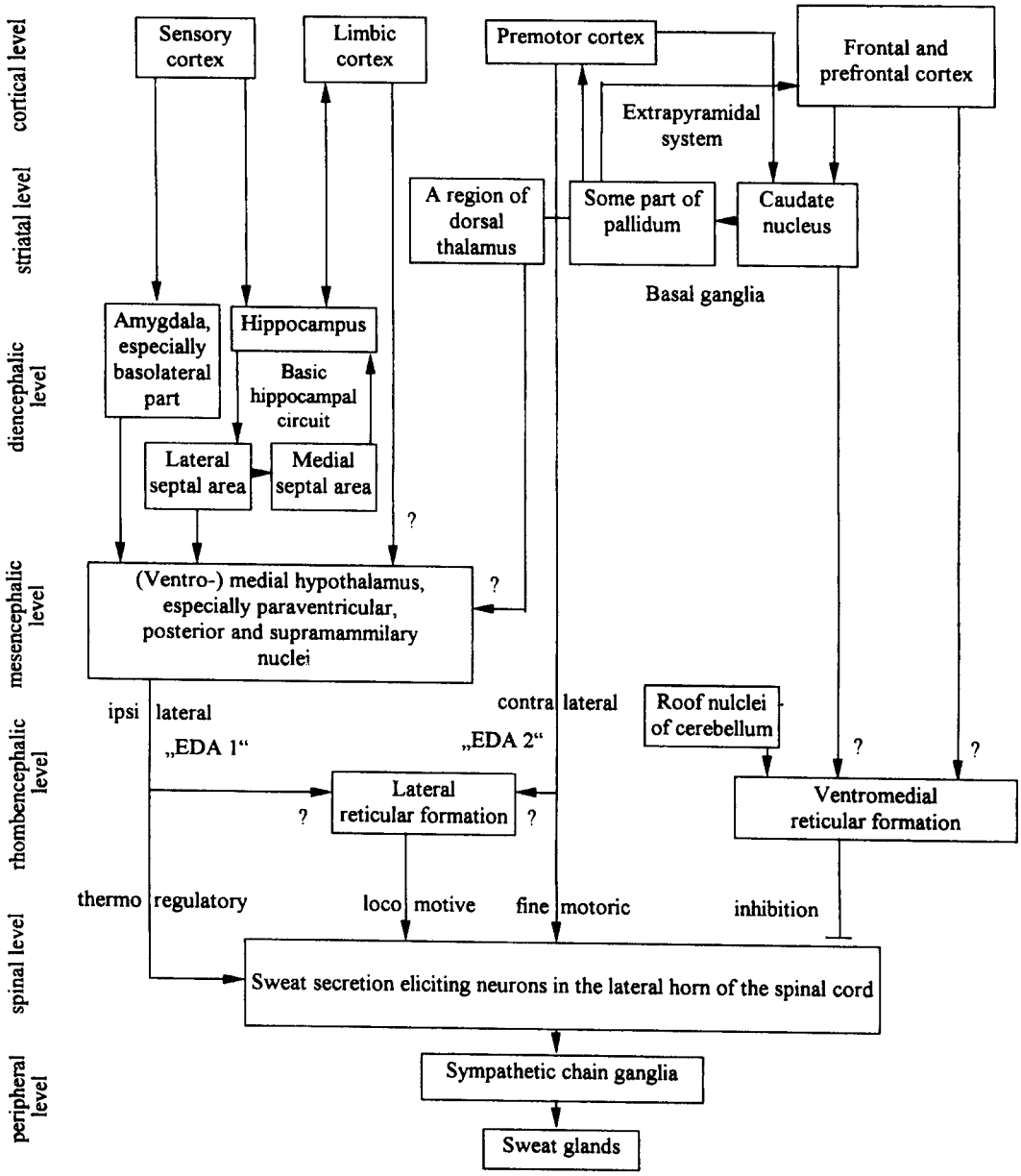

Figure 1. Block diagram showing sweat centers and their efferent pathways to sweat glands in the cat after Wang (1964), combined with knowledge from human neurophysiology. Question marks indicate suggested pathways for which Whang could not locate a specific tract.

The midbrain does not seem to play an important role with respect to excitatory control of EDA, though its descending sweat eliciting pathways are regarded as being well established in the cat. In the cat's brain, the ventromedial part of the reticular formation (RF) at the rhombencephalic level is regarded as the most powerful inhibitory center for EDA, whereas the roof nuclei in the anterior lobe of the cerebellum only inhibit autonomic functions during strong muscular movement. Removal of the cerebellum has no observable effect on EDRs in cats. At the striatal level, an important inhibitory center for EDA seems to be located in the caudate nucleus. Additionally, inhibitory but also excitatory areas with respect to EDA were found in the frontal cerebral cortex (e.g., Wang & Lu, 1930; Langworthy & Richter, 1930; Wilcott, 1969; Wilcott & Bradley, 1970). The above mentioned possible inhibitory function of the hippocampus was not included in Figure 1 because Wang (1964) could not form any simple notion concerning its descending pathway. At the spinal level and below, only excitatory control of the cat's EDA is seen. Since the pallidum was demonstrated being necessary for synchronization of spontaneous SPRs in the cat's four footpads, Wang assumed a regulatory center for EDA located in some part of the pallidum.

The finding that EDRs may be elicited in animal preparations by stimulation of anterior hypothalamic regions (for summaries see Edelberg, 1972; Venables & Christie, 1973) belonging to the parasympathetic system, gave rise to the hypothesis that at least part of CNS elicitation of EDA could be under parasympathetic control. However, evidence for influences of anterior hypothalamic regions on sweat gland activity in humans is lacking. Instead, paraventricular, posterior and supramammilary nuclei in the weakly myelinated part of the hypothalamus form the major source of sweat-eliciting nerve fibers in the human brain. Those are in close proximity of the so-called Papez circuit in the limbic system (Boucsein, 1992, Fig. 4), thus providing neuroanatomical evidence for the particular role of EDA as an indicator of emotional processes.

The amygdala plays an important role in phasic EDA. Stimulation of the basolateral amygdala can evoke a single EDR with its typical form of recovery in cats (Lang, Tuovinen & Valleala, 1964), while amygdalectomy in monkeys produced a marked impairment of EDRs following acoustic stimulation (Bagshaw, Kimble & Pribram, 1965) which gave rise to the hypothesis that the amygdala is responsible for the elicitation of the electrodermal orienting response. This has been questioned by Tranel and Damasio (1989) who obtained regular electrodermal orienting responses in a patient whose entire amygdaloid complex had been destroyed bilaterally. On the other hand, if amygdaloid after-discharge occurs following this stimulation, the EDR maintains its peak for a considerable amount of time consisting of a series of small flutter-like waves, as shown by Lang et al. (1964) with cats. The amygdala exerts its influence on EDA through the sweat eliciting areas in the hypothalamus (Figure 1).

There is also evidence for cerebral influences on EDA being independent from hypothalamic pathways. Wang (1964), in his lesion experiments with nonanesthetized cats, found that the synchronization of spontaneous SPRs in all four paws was controlled by some part of the pallidum which belongs to the basal ganglia, but not by hypothalamic structures. Langworthy

and Richter (1930) as well as Spiegel and Hunsicker (1936) emphasized the role of premotor cortical regions (Brodmann area 6, see Boucsein, 1992, Fig. 6) in eliciting EDA, since a close connection between the pyramidal fibers for the transmission of skeletal muscle impulses and sweat eliciting fibers has been found in degeneration studies. As Darrow (1937) pointed out, those pathways cannot be identical, since pyramidal stimulations were not followed by responses in the skin. However, Langworthy and Richter (1930) could elicit EDRs and other autonomic responses by stimulating the pyramidal tract in cats. Roy, Sequeira-Martinho and Brochard (1984) suggested this being due to the collaterals from the pyramidal tract reaching the RF, by which reticular elicitation of EDA has been mediated. They suggested that the sweat eliciting fibers were corticopontine rather than corticospinal like the pyramidal fibers. Thus, the neurophysiological basis for electrodermal changes that accompany changes in posture should be influenced from tegmental or pontine areas where the premotor fibers terminate. Since it is now widely accepted that subcortical structures such as the basal ganglia participate in motoric integration or programming (Marsden, 1982), the combined striatal and premotor cortical origins of EDA can be viewed together as being responsible for the premotor electrodermal component.

Excessive sweating is observed if premotor cortical areas are electrically stimulated or removed. This might be explained by both excitatory and inhibitory cortical influences on sweat secretion. In their animal studies, Wang and Brown (1956) were able to demonstrate inhibitory influences from large frontal cortical areas acting on excitatory cortical sweating centers of the sensorimotor and the anterior limbic cortex. Although the question of ipsi- or contralaterality of inhibitory vs. excitatory influences on electrodermal phenomena is still unresolved, the role of EDRs as concomitants of motor reactions is well established in human subjects (Edelberg & Wright, 1964; Pugh, Oldroyd, Ray & Clark, 1966). Bilateral electrodermal recordings at palmar sites following strong acoustic stimuli sometimes showed noticeable lateral differences, which, however, never exceeded a ratio of 1 : 1.5 (Fisher, 1958; Obrist, 1963). However, if subjects were asked to move one foot as a reaction to the acoustic stimulation, the lateralization increased in favor of the EDR amplitude at the ipsilateral hand (Culp & Edelberg, 1966). Taking all these human as well as animal results together, there are at least two different cortical sources of influence on EDA. Besides ipsilateral hypothalamic influences on sweat secretion that are controlled by limbic structures (see Boucsein, 1992, Fig. 4), the basal ganglia together with premotor cortical areas form a second system that exerts separate and contralateral influences on sweat secretion and therefore on EDA. Those influences were not only found in animal lesion studies, but also in stimulation and lesion studies in humans (Schliack & Schiffter, 1979). The limbic-hypothalamic electrodermal source is labeled "EDA 1" in Figure 1, while the premotor and basal ganglia source is labeled "EDA 2" (cf., sources 1 and 2 in Boucsein, 1992, Fig. 6).

However, the laterality of EDA may disappear at the reticular level and/or below, since unilateral cortical and pyramidal stimulation in the cat elicited bilateral EDRs with comparable amplitudes (Sequeira-Martinho, Roy, & Ba-M'hamed, 1986) and reticular stimulation in the cat was always followed by bilateral EDRs

(Ba-M'hamed-Bennis, Sequeira-Martinho, Freixa i Baque & Roy, 1985). The RF itself may exert generating as well as modulating influences on EDA (Roy et al., 1984, 1993). Bloch (1965) pointed to EDA as a reflection of CNS arousal that is controlled by excitatory as well as inhibitory areas in the RF. These areas are regarded as mainly influenced by inhibitory corticofugal neurons. An inhibitory reticular center for sweating is located in the ventromedial RF (Wang & Brown, 1956; Roy, Delerm & Granger, 1974), whereas the lateral portion of the midbrain RF and portions of the diencephalic RF have excitatory effects on EDA (Bloch, 1965; Edelberg, 1972). Venables and Christie (1973) provided evidence that stimulation of these mesencephalic excitatory regions facilitates motor activity via the action of the reticular activation system. Because the RF is connected with the striopallidum as well as the cerebellum and is further known to strongly influence skeletal muscle tone as well as muscle contractions via the gamma-efferent system, a close connection between the reticular modulation of sweat gland activity and skeletal muscle activity can be suggested (Roberts & Young, 1971). Thus, while influences on EDA stemming from EDA 2 have to be regarded as concomitants of preparing the activation of distinct motor units, reticular influences on EDA (cf., source 3 in Boucsein, 1992, Fig. 6) are more likely to be connected with a general increased muscular tone due to an increased general arousal. Reticular mediated EDRs should likely be concomitants of locomotive changes, that may appear in emergency situations, and not of distinct or fine manipulative motor actions which require a stronger cortical participation. Whether electrodermal changes that appear as concomitants of inspiration (which are mainly regarded as artifacts in electrodermal recording, cf., Boucsein, 1992, p. 125) are more cortically or more reticularly influenced remains unanswered.

In summary, experimental as well as clinical evidences concerning the CNS elicitation of EDA point to the existence of two different origins above the reticular level, as first suggested by Edelberg (1972): A limbic-hypothalamic source labeled "EDA 1" here, being thermoregulatory and also emotionally influenced.

A premotor and basal ganglia source labeled "EDA 2" here, eliciting electrodermal concomitants of the preparation of certain motor actions.

In addition, there may be a reticular modulating system which mediates EDA changes that appear with variations of general arousal. This reticular modulating system is also likely to be responsible for inhibitory influences on EDA (see lower right part of Fig. 1) that may be either ipsi- or contralateral. However, under conditions of diffuse sweat gland activation with generalized EDA, the specificity of those neuronal systems may at least partly disappear. For example, Wilcott (1963) showed that non-palmar areas of skin that are normally regarded as thermoregulatory also took part in emotional sweating during a highly stressful situation. Therefore, the specificity of the different CNS sources of EDA may get lost during states of very high arousal which has implications for using EDA as an indicator of emotions. Low and high intensity emotional states may elicit rather different electrodermal patterns as demonstrated in section 2.

4. Central nervous elicitation of emotioninduced electrodermal activity

As already stated in section 2, attempts to empirically determine psychophysiological response patterns that are specific for

particular emotions have been only moderately successful so far. Part of this dilemma may be due to an insufficient backup by neurophysiological theories. In psychophysiological research into emotions, the non-theoretical multivariate shot-gun approach is still prevailing. The following section provides an attempt to close the gap between knowledge about the brain's emotional functioning and its peripheral concomitants.

During the last two decades, attempts have been made to replace the notorious one-dimensional arousal theories (e. g., Duffy, 1951: Malmo, 1959) by multiple-arousal systems such as Routtenberg (1968, 1971) or Pribram and McGuinness (1975). The distinction between different kinds of arousal can be used for modeling neurophysiological origins of emotion-related psychophysiological patterns as well. Interestingly, the so-called Papez circuit of emotions (Papez, 1937) was assigned a more generalized role as an information processing system in Gray's (1982) neurophysiological model of anxiety. Fowles (1980), providing evidences from both experimental and clinical studies, came to the conclusion that the prevalence of Gray's so-called Behavioral Inhibition System (BIS) which dominates during anxiety goes with increased tonic EDA. On the other hand, if Gray's so called Behavioral Activation System (BAS) dominates, a tonic increase of HR is likely to be observed. A neurophysiological model for backing up such a differential sensitivity of the electrodermal and the cardiovascular system has been provided by Boucsein (1992, Fig. 48), integrating the three-arousal models of Pribram and Mc Guinness (1975) and Fowles (1980), the two-arousal model of Routtenberg (1968) as well as several other theoretical considerations of subcortical-cortical processes of arousal, emotion and information processing. Since anxiety constitutes the only emotion that has been deliberately modeled in this arousal-focused system, an additional neurophysiological model of emotional functioning which refers to four classes of emotions will be introduced here. Based on emotional behavior in the rat, Panksepp (1982, 1986) proposed four kinds of what he called emotive systems. Those have the properties to elicit fairly organized behavioral sequences of the following classes:

The "expectancy" command system constitutes a sensory-motor system for the mediation of exploratory behavior, approach or investigation (mainly with respect to foraging). This system coincides with neural circuits mediating electrical self stimulation of the lateral hypothalamus. Taken together all evidences from Panksepp's review, that particular system appears to be congruent with Gray's (1982) BAS and the reward system of Olds and Olds (1965) in the medial forebrain bundle. Panksepp clearly identifies this system with the transmitter dopamine, since noradrenaline which had been discussed as another candidate is no longer regarded as important for self stimulation

The "rage" command system, being the first hypothalamic emotive system that was discovered in the 1940ies by Eckhard Hess (see Kolb & Whishaw, 1996, p. 418) has some anatomical overlapping with the expectancy system. However, it seems to be clearly a cholinergic system, since applying cholinergic drugs into the hypothalamus and the amygdala can provoke rage responses (Panksepp, 1982). On the other hand, cholinergic drugs also decrease the rate of self stimulation. In turn, self stimulation is increased by anticholinergics, probably by inhibiting dopamine cells through GABA interneurons. Therefore, the

expectancy and the rage system can be sufficiently distinguished on a neurophysiological basis.

The "fear" command system described by Panksepp slightly differs from Gray's (1982) BIS. For fear, Panksepp considers response topographies of species-typical flight responses, as opposed to fight responses in the case of rage. Gray differentiates between flight/fight responses on one hand and his BIS on the other hand. Stimulation of the hypothalamus in rats and humans evokes anxiety, while the site that provokes flight is surrounding this hypothalamic area. There appears to be substantial anatomical overlap and functional interaction between these systems since concurrent stimulation of both parts can yield synergistic effects, with either threat or flight being intensified. Although both systems are not so clearly separated at the hypothalamic level, they can be well separated in the amygdala (see Figure 2).

The "panic" command system has not been so deliberately worked out by Panksepp. He suggests the anterior basal hypothalamus as being responsible for eliciting its behavioral patterns, and the cingulate gyrus together with the interstitial nucleus of the stria terminalis as being probably in control of those. The system is mainly attuned by social loss. Brain opiods may be involved in its action as well.

Panksepp suggests these systems being genetically determined. Therefore, certain emotional challenging situations have the capability to elicit certain kinds of emotions without being conditioned. These are:

1. positive incentives for the expectancy command system,
2. pain and life threat for the fear command system,
3. frustration for the rage command system, and
5. social loss for the panic command system.

Each system is suggested to elicit certain somato-motor, autonomic and endocrine response patterns that have been figured out being advantageous during evolution. Unfortunately, no suggestions for psychophysiological concomitants have been made by Panksepp. He restricted himself to behavioral outcomes, leaving research about the hypothalamic exits of his systems to a more psychobiological approach, as he called it in 1986.

Figure 2 combines the models of Panksepp and Gray together with several other models such as the three systems of Pribram and McGuinness (1975), the defense systems of Blanchard and Blanchard (1988), the reward/punishment systems of Olds and Olds (1965), features of the central nervous system described by Hess in the 1940ies and a complex cognitive loop between basal ganglia and frontal cortex described by DeLong, Georgopoulos and Crutcher (1983). In Figure 2, the four basic emotions are arranged in columns. Cortical and higher subcortical levels are depicted on top of hypothalamic and brain stem levels, in order to ensure comparability with Figure 1.

Although obtained by means of different experimental paradigms, the models of Gray and Panksepp complement each other, especially on the neuro-humoral level. Gray focuses on noradrenergic and serotonergic fibers and their action on his septo-hippocampal system, while Panksepp identifies his expectancy system with dopamine and his rage system with acetylcholine. According to Panksepp (1986), noradrenaline enhances and serotonine reduces emotional behavior as a

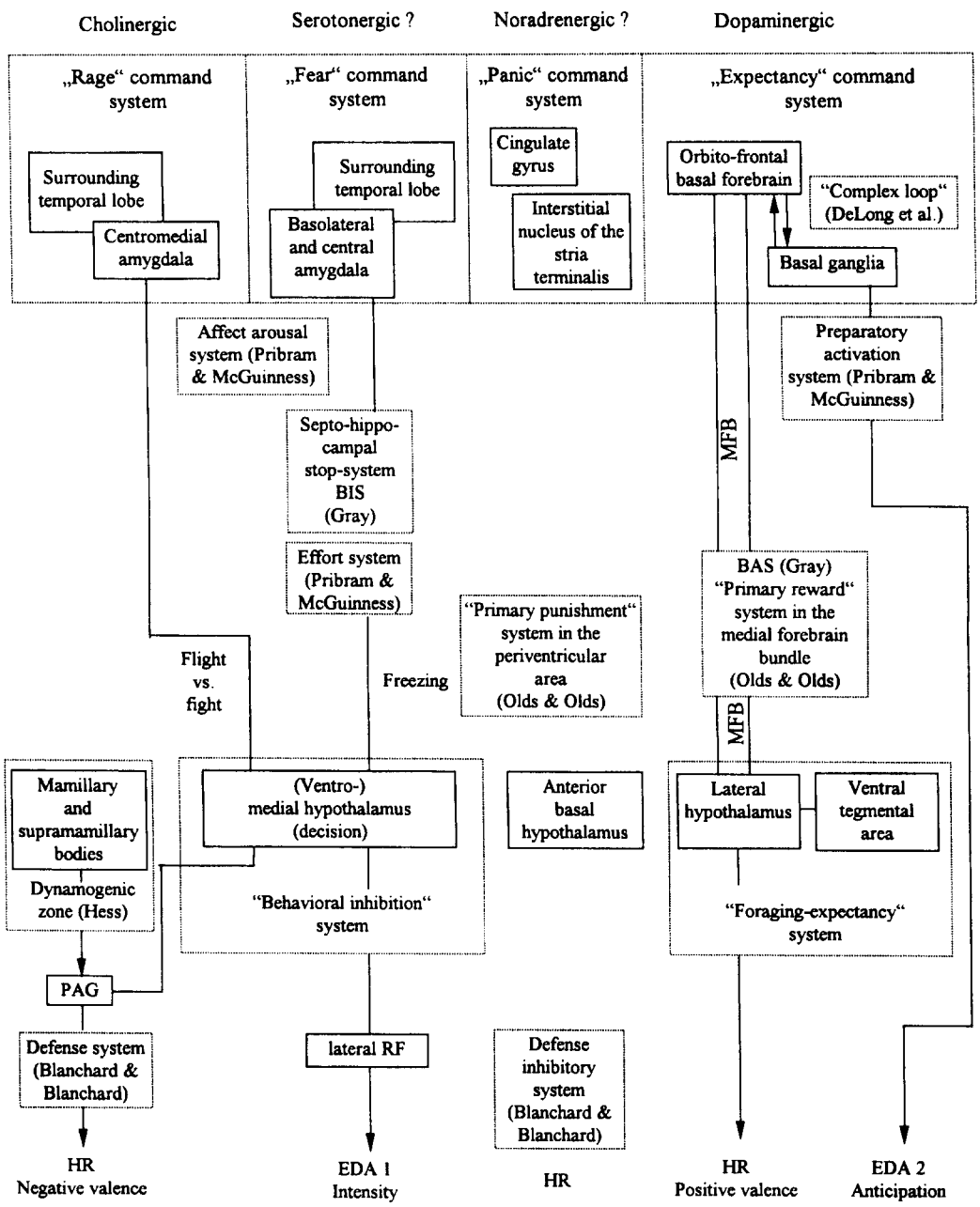

Figure. 2 An integrative model for the CNS elicitation of Panksepp's (1982) four basic emotive systems. Solid boxes = anatomical structures; dashed boxes = proposed systems. MFB = medial forebrain bundle; PAG = periaquaeductal gray; RF =

whole. Therefore, the fear system has been preliminary labeled as mainly serotonergic and the panic system as possibly noradrenergic in Figure 2.

In the lower part of Figure 2, possible outcomes in the electrodermal and cardiovascular systems are shown. The model depicted here allows to generate testable hypotheses for psychophysiological experimentation that generally overcome the simple one-dimensional arousal notion as well as the oversimplified sympathetic/parasympathetic dichotomy in ANS variables. In addition, it is also shown that a dichotomy in which EDA stands for negative and HR for positive hedonic states, as could have been drawn from the work of Fowles (1980), cannot really be made. Both kinds of autonomic measures may well indicate positive and negative emotive aspects. For example, a tonic increase in HR together with a certain aspect of EDA, labeled EDA 2 in the lower right part of Figure 2 (see also Figure 1), may indicate a positively tuned anticipation, while an increase in HR without EDA being affected may indicate the rage or fight-like component of negative emotions (lower left part of Figure 2). On the other hand, an increase of tonic EDA without changes in HR may indicate the fear or anxiety component in a negative emotional state (labeled EDA 1 in Figure 2, see also Figure 1).

A comparison of Figure 2 with Figure 1 yields relatively good coincidence of the CNS structures involved in certain emotive systems and those involved in the generation of EDA. The hypothalamus-generated so-called EDA 1 is connected to the amygdala with respect to its stimulation dependent properties, presumably to the amygdala's basolateral and central parts. Its major emotive determinant is fear. The appropriate information processing is performed within septo-hippocampal and limbic pathways which include the Papez circuit. Electrodermal action patterns are generated in the ventral parts of the hypothalamus and in rather medial than in lateral areas (see also Figure 1). They travel through the descending pathways that are used for the initiation of thermoregulatory sweating as well. Emotional intensity is most appropriately represented by the NS.EDR frequency (Boucsein, 1992). Presumably, central parts of the amygdala together with the hippocampus form a not yet fully understood complex system which exerts both excitatory and inhibitory control on a generalized electrodermal reactivity labeled EDA 1 here.

Electrodermal phenomena are also clearly connected to Panksepp's expectancy command system. The basal ganglia generated so-called EDA 2 serves as an indicator for anticipatory behavior of the brain, which is under the control of premotor and frontal cortical areas that interact with the basal ganglia. The appropriate EDA patterns travel together with the motor fibers in the internal capsule to the spinal cord. The amount of anticipatory EDA is most appropriately represented by the EDR amplitude (Boucsein, 1992).

The possible role of EDA for both the rage and the panic command systems is less clear. The mamillary bodies which are viewed as part of the rage system (lower left part of Figure 2) have been demonstrated to inhibit EDA in animal studies (see section 3). In turn, they may accelerate HR as a concomitant of defensive behavior, the final pathway of which may be the periaquaeductal gray (Bandler, 1988). This efferent pathway can be modified by influences from the centromedial amygdala on the hypotha-

lamus but also by means of the amount of testosterone in the brain (Blanchard & Blanchard, 1988). The panic command system is generally not well understood which is the reason for the missing arrows in Figure 2. With respect to neuroanatomy, this system widely corresponds to the defense inhibitory system of Blanchard and Blanchard (1988) that shows great coincidence within various mammalian species (Albert & Walsh, 1984). Although this system gets input from the lateral septum, an area which exerts a major inhibiting function in Gray's (1982) BIS, it is rather unlikely to be involved in the generation of freezing behavior (Ursin, Blanchard, Blanchard & Ursin, 1981). As a counterpart to the primary reward system in the medial forebrain bundle, the primary punishment system located in the periventricular area by Olds and Olds (1965) may be subsumized under Panksepp's panic command system. The appropriate psychophysiological concomitant for panic is a vigorous HR increase (Markgraf, Taylor, Ehlers, Roth & Agras, 1987).

5. Summary and conclusions

The present article provided both empirical and neurophysiological evidence for the important role of EDA in emotional processes. The medial hypothalamic areas, including some ventral parts, that are known to be the source of thermoregulatory sweating as well, elicit one kind of EDA ("EDA 1") that is a valid indicator of the intensity of predominantly negative emotions such as fear. Especially in the low-intensity range, the NS.EDR frequency constitutes a much more sensitive indicator as the HR and is a more appropriate measure of emotion strength than the EDL (see Boucsein, 1992, pp. 261 ff). In turn, the domain of the HR as an indicator of negative emotionality is in the upper intensity range, e. g., during rage or panic. In the positive emotions domain, EDA does not seem to be as important an indicator as HR. However, a non-hypothalamic source of EDA may play a critic important role in certain kinds of positive emotional behavior. Fibers having the property to stimulate neurons in the spinal cord that are responsible for sweat secretion originate in premotor cortical areas and in the basal ganglia as well (see Figure 1). This alternative source of EDA ("EDA 2") shows up in the EDR amplitude as an anticipatory response. It constitutes another psychophysiological indicator of positive emotions, in addition to the moderate increase of tonic HR. As stated in section 2, HR is a typical ANS concomitant of overt emotional responses, while the amount of EDA is more likely to reflect the internal strength of emotions. In case a single EDR amplitude does not constitute an appropriate measure, the averaged amplitudes of NS.EDRs over a certain period of time may serve as such (see Boucsein, 1992, pp. 148 f.).

The neuroanatomical and neurophysiological considerations given in sections 3 and 4 of the present article at least partly explain the different aspects of sensitivity for the different electrodermal parameters found in the empirical studies described in section 2. Much empirical work is still to be done in this area to clarify their specific role as indicators of emotional processes. In general, there is no sound alternative to the combination of experimental psychophysiology with neurophysiological modeling as recommended in the present article.

References

Albert, D. J., & Walsh, M. L. (1984). Neural systems and the inhibitory

modulation of agonistic behavior: A comparison of mammalian species. *Neuroscience and Biobehavioral Reviews, 8*, 5-24.

Ax, A. F. (1953). The physiological differentiation between fear and anger in humans. Psychosomatic Medicine, 15, 433-442.

Bagshaw, M. H., Kimble, D. P., & Pribram, K. H. (1965). The GSR of monkeys during orienting and habituation and after ablation of the amygdala, hippocampus and inferotemporal cortex. Neuropsychologia, 3, 111-119.

Ba-M' Hamed - Bennis, S., Sequeira-Martinho, H., Freixa i Baque, E., & Roy, J.-C. (1985). Skin potential responses elicited by reticular stimulation are not lateralized in the cat. Biological Psychology, 21, 250-251.

Bandler, R. (1988). Brain mechanisms of aggression as revealed by electrical and chemical stimulation: Suggestion of a central role for the midbrain periaqueductal grey region. In A. N. Epstein, & R. Morrison (Eds.), Progress in Psychobiology an Physiological Psychology (Vol. 13, pp. 67-154). New York: Academic Press.

Blanchard, D. C., & Blanchard, R. J. (1988). Ethnoexperimental approaches to the biology of emotion. Annual Review of Psychology, 39, 43-68.

Bloch, V. (1965). Le controle central de l'activit lectrodermale. Journal de Physiologie, 57, 1-132.

Boucsein, W. (1992). Electrodermal activity. New York: Plenum Press.

Boucsein, W., & Backs, R. W. (1999). Engineering psychophysiology as a discipline: Historical and theoretical aspects. In: R. W. Backs, & W. Boucsein (Eds.). Engineering Psychophysiology. Mahwah, N. J.: Lawrence Erlbaum Associate (in print).

Boucsein, W., & Baltissen, R. (1998). Autonomic indicators of emotions elicited by positive and negative stimulus properties. In: Development of synthetic physiology signal measuring and analyzing system (KRISS-98-094-IR).pp.154-212. Taejon: Korean Institute of Standards and Science.

Buck, R. (1977). Nonverbal communication of affect in preschool children: Relationships with personality and skin conductance. Journal of Personality and Social Psychology, 35, 225-236.

Buck, R. (1980). Nonverbal behavior and the theory of emotion: The facial feedback hypothesis. Journal of Personality and Social Psychology, 38, 811-824.

Buck, R., & Miller, R. E. (1974). Sex, personality, and physiological variables in the communication of affect via facial expression. Journal of Personality and Social Psychology, 23, 362-371.

Buck, R., Savin, V. J., Miller, R. E., & Caul, W. F. (1972). Communication of affect through facial expressions in humans. Journal of Personality and Social Psychology, 23, 362-371.

Campos, J. J., & Johnson, H. J. (1967). The effect of affect and verbalization instructions of directional fractionation of autonomic response. Psychophysiology, 3, 245-290.

Colby, C. Z., Lanzetta, J. T., & Kleck, R. E. (1977). Effects of the expression

of pain on autonomic and pain tolerance responses to subject-controlled pain. Psychophysiology, 14, 537- 540.

Culp, W. C., & Edelberg, R. (1966). Regional response specifity in the electrodermal reflex. Perceptual and Motor Skills, 23, 623-627.

Darrow, C. W. (1937). Neural mechanisms controlling the palmar galvanic skin reflex and palmar sweating. Archives of Neurology and Psychiatry, 37, 641-663.

DeLong, M. R., Georgopoulos, A. P., & Crutcher, M. D. (1983). Cortico-basal ganglia relations and coding of motor performance. Experimental Brain Research, Suppl. 7, 49, 30-40.

Duffy, E. (1951). The concept of energy mobilization. Psychological Review, 58, 30-40.

Edelberg, R. (1972). Electrical activity of the skin. In N. S. Greenfield, & R. A. Sternbach (Eds.), Handbook of Psychophysiology (pp. 367-418). New York: Holt.

Edelberg, R., & Wright, D. J. (1964). Two galvanic skin response effector organs and their stimulus specificity. Psychophysiology, 1, 39-47.

Engel, B. T. (1972). Response specificity. In N. S. Greenfield, & R. A. Sternbach (Eds.), Handbook of Psychophysiology (pp. 571-576). New York: Holt.

Fahrenberg, J. (1988). Psychophysiological processes. In J. R. Nesselroade, & R. B. Cattell (Eds.), Handbook of multivariate experimental psychology (pp. 867-914). New York: Plenum Press.

Fisher, S. (1958). Body image and asymmetry of body reactivity. Journal of Abnormal and Social Psychology, 57, 292-298.

Fowles, D. C. (1980). The three arousal model: Implications of Gray's two-factor learning theory for heart rate, electrodermal activity, and psychopathy. Psychophysiology, 17, 87-104.

Gellhorn, E. (1964). Motion and emotion: The role of proprioception in the physiology and pathology of the emotions. Psychological Review, 71, 457-472.

Gray, J. A. (1982). The neuropsychology of anxiety: An inquiry into the functions of the septo-hippocampal system. Oxford: Clarendon Press.

Greenwald, M. K., Cook, E. W., & Lang, P. J. (1989). Affective judgement and psychophysiological response: Dimensional covariation in the evaluation of pictorial stimuli. Journal of Psychophysiology, 3, 51-64.

Izard, C. E. (1971). Face of emotion. New York: Appleton.

Jae nig, W., Sundloef, G., & Wallin, B. G. (1983). Discharge patterns of sympathetic neurons supplying skeletal muscle and skin in man and rat. Journal of the Autonomic Nervous System, 7, 239-256.

Kleck, R. E., Vaughan, R. C., Cartwright-Smith, J., Vaughan, K. B., Colby, C. Z., & Lanzetta, J. T. (1976). Effects of being observed on expressive, subjective, and physiological responses to painful stimuli. Journal of Personality and Social Psychology, 34, 1211-1218.

Kolb, B., & Whishaw, I. Q. (1996). Fundamentals of human neuropsychology(4th edition). New York: Freeman & Co..

Lacey, J. I., & Lacey, B. c. (1970). Some autonomic-cental nervous system

interrelationships. In P. Black (Ed.), Physiological correlates of emotion (pp. 205-227). New York: Academic Press.

Lang, H., Tuovinen, T., & Valleala, P. (1964). Amygdaloid afterdischarge and galvanic skin response. Electroencephalography and Clinical Neurophysiology, 16, 366-374.

Lang, P. J. (1968). Fear reduction and fear behavior: Problems in treating a construct. In J. Schlien (Ed.), Reseach in Psychotherapy (Vol. 3, pp. 90-103). Washington, D. C.: American Psychological Association.

Lang, P. J., Oehmann, A., & Vaitl, D. (1988). The International Affective Picture System. Gainesville: Center for Research in Psychophysiology, University of Florida.

Langworthy, O. R., & Richter, C. P. (1930). The influence of efferent cerebral pathways upon the sympathetic nervous system. Brain, 53, 178-193.

Lanzetta, J. T., Cartwright-Smith, J., & Kleck, R. E. (1976). Effects of nonverbal dissimulation on emotional experience and autonomic arousal. Journal of Personality and Social Psychology, 33, 354-370.

Levenson, R. W. (1988). Emotion and the autonomic nervous system: A prospectus for research on autonomic specificity. In H. L. Wagner (Ed.), Social Psychophysiology and Emotion: Theory and Clinical Applications (pp. 17-42). New York: John Wiley & Sons.

Levenson, R. W., Ekman, P., & Friesen, W. V. (1990). Voluntary facial action generates emontion-specific autonomic nervous system activity. Psychophysiology, 27, 363-384.

Levis, D. J., & Smith, J. E. (1987). Getting individual differences in autonomic reactivity to work for instead of against you: Determining the dominant "psychological" stress channel on the basis of a "biological" stress test. Psychophysiology, 24, 346-352.

Malmo, R. B. (1959). Activation: A neuropsychological dimension. Psychological Review, 66, 367-386.

Markgraf, J., Taylor, C. B., Ehlers, A., Roth, W. T., & Agras, W. S. (1987). Panic attacks in the natural environment. Journal of Nervous and Mental Disease, 175, 558-565.

Marsden, C. D. (1982). The mysterious motor function of the basal ganglia: The Robert Wartenberg lecture. Neurology, 32, 514-539.

McFarland, R. A. (1981). Physiological psychology: The biology of human behavior. Palo Alto: Mayfield.

Obrist, P. A. (1963). Skin resistance levels and galvanic skin response: Unilateral differences. Science, 139, 227-228.

Olds, J., & Olds, M. E. (1965). Drives, reward, and the brain. In T. M. Newcomb (Ed.), New directions in psychology II (pp. 327-410). New York: Holt.

Orr, S. P., & Lanzetta, J. T. (1980). Facial expressions of emotion as conditioned stimuli for human autonomic responses. Journal of Personality and Social Psychology, 38, 278-282.

Osgood, C. E. (1966). Dimensionality of the semantic space for communication via facial expressions. Scandinavian Journal of Psychology, 7, 1-30.

Panksepp, J. (1982). Toward a general psychobiological theory of emo-

tions. The Behavioral and Brain Sciences, 5, 407-467.

Panksepp, J. (1986). The anatomy of emotions. In R. Plutchik, & H. Kellermann (Eds.), Emotions: Theory, Research, and Experience (Vol. 3, pp. 91-124). New York: Academic Press.

Papez, J. W. (1937). A proposed mechanism of emotion. Archives of Neurology and Psychiatry, 38, 725-743.

Pribram, K. H., & McGuinness, D. (1975). Arousal, activation, and effort in the control of attention. Psychological Review, 82, 116-149.

Pugh, L. A., Oldroyd, C. A., Ray, T. S., & Clark, M. L. (1966). Muscular effort and electrodermal responses. Journal of Experimental Psychology, 71, 241-248.

Roberts, L. E., & Young, R. (1971). Electrodermal responses are independent of movement during aversive conditioning in rats, but heart rate is not. Journal of Comparative and Physiological Psychology, 77, 495-512.

Routtenberg, A. (1968). The two-arousal hypothesis: Reticular formation and limbic system. Psychological Review, 75, 51-80.

Routtenberg, A. (1971). Stimulus processing and response execution: A neurobehavioral theory. Physiology and Behavior, 6, 589-596.

Roy, J. C., Delerm, B., & Granger, L. (1974). L'inhibition bulbaire de l'activit electrodermale chez le chat. Electroencephalography and Clinical Neurophysiology, 37, 621-632.

Roy, J. C., Sequeira-Martinho, A. H., & Brochard, J. (1984). Pyramidal control of skin potential responses in the cat. Experimental Brain Research, 54, 283-288.

Roy, J. C., Sequeira, H., & Delerm, B. (1993). Neural control of electrodermal activity: Spinal and reticular mechanisms. In: J. C. Roy, W. Boucsein, D. C. Fowles, & J. H. Gruzelier, Progress in electrodermal research (pp. 73-92). New York: Plenum Press.

Russell, J. A. (1980). A circumplex model of affect. Journal of Personality and Social Psychology, 39, 1161-1178.

Sato, K. (1977). The physiology, pharmacology, and bochemistry of the eccrine sweat gland. Reviews of Physiology, Biochemistry and Pharmacology, 79, 51-131.

Schachter, S., & Singer, J. E. (1962). Cognitive, social and physiological determinants of emotional state. Psychological Review, 69, 379-399.

Schliack, H., & Schiffter, R. (1979). Neurophysiologie und Pathophysiologie der Schweiss sekretion. In E. Schwarz, H. W. Spier, & G. St ttgen (Eds.), Handbuch der Haut- und Geschlechtskrankheiten, Bd. 1/4 A: Normale und pathologische Physiologie (pp. 349-458). Berlin: Springer.

Schlosberg, H. (1954). Three dimensions of emotions. Psychological Review, 61, 81-88.

Sequeira-Martinho, H., Roy, J.-C., & Ba-M'Hamed, S. (1986). Cortical and pyramidal stimulation elict nonlateralized skin potential responses in the cat. Biological Psychology, 23, 81.

Sokolov, E. N. (1992). Detector mechanisms of perceptions and emotions. In D. G. Forgays, T. Sosnowski, & K. Wrzesniewski (Eds.), Anxiety: Recents Developments in Cognitive, Psychophysiological, and

Health Research (pp. 153-165). Washington: Hemisphere Publishing Corp.

Spiegel, E. A., & Hunsicker, W. C. (1936). The conduction of cortical impulses to the autonomic system. Journal of Nervous and Mental Disease, 83, 252-274.

Stemmler, G. (1984). Psychophysiologische Emotionsmuster. Frankfurt: Peter Lang.

Stemmler, G. (1989). The autonomic differentiation of emotions revisited: Convergent and discriminant validation. Psychophysiology, 26, 617-632.

Tranel, D., & Damasio, H. (1989). Intact electrodermal skin conductance responses after bilateral amygdala damage. Neuropsychologia, 27, 381-390.

Traxel, W. (1960). Die M glichkeit einer objektiven Messung der St rke von Gef hlen. Psychologische Forschung, 26, 75-90.

Turpin, G., Tarrier, N., & Sturgeon, D. (1988). Social psychophysiology and the study of biopsychosocial models of schizophrenia. In H. L. Wagner (Ed.), Social Psychophysiology and Emotion: Theory and Clinical Applications (pp. 251-272). Chichester: Wiley & Sons.

Ursin, H., Blanchard, D. C., Blanchard, R. J., & Ursin, R. (1981). Flight and defense in cats with septal lesions. Bulletin of the Psychonomic Society, 17, 206-208.

Vaughan, K. B., & Lanzetta, J. T. (1981). The effect of modification of expressive displays on vicarious emotional arousal. Journal of Experimental Social Psychology, 17, 16-30.

Venables, P. H., & Christie, M. J. (1973). Mechanisms, instrumentation, recording techniques, and quantification of response. In W. F. Prokasy & D. C. Raskin (Eds.), Electrodermal activity in psychological research (pp. 1-124). New York: Academic Press.

Wagner, H. (1989). The peripheral physiological differentiation of emotions. In H. Wagner, & A. Manstead (Eds.), Handbook of Social Psychophysiology (pp. 77-98). John Wiley & Sons Ltd.

Wang, C. (1964). Neural Control of Sweating. Madison: University of Wisconsin Press.

Wang, G. H., & Brown, V. W. (1956). Suprasegmental inhibition of an autonomic reflex. Journal of Neurophysiology, 19, 564-572.

Wang, G. H., & Lu, T. W. (1930). Galvanic skin reflex induced in the cat by stimulation of the motor area of the cerebral cortex. Chinese Journal of Physiology, 4, 303-326.

Wilcott, R. C. (1963). Effects of high environmental temperature on sweating and skin resistance. Journal of Comparative and Physiological Psychology, 56, 778-782.

Wilcott, R. C. (1969). Electrical stimulation of the anterior cortex and skin potential responses in the cat. Journal of Comparative and Physiological Psychology, 69, 465-472.

Wilcott, R. C., & Bradley, H. H. (1970). Low-frequency electrical stimulation of the cat's anterior cortex and inhibition of skin potential responses. Journal of Comparative and Physiological Psychology, 72, 351-355.

Winton, W. M., Putnam, L. E., & Krauss,

R. M. (1984). Facial and autonomic manifestations of the dimensional structure of emotion. Journal of Experimental Social Psychology, 20, 195-216.

Wundt, W. (1896). Grundriss der Psychologie. Leipzig: Engelmann.

Yokota, T., & Fujimori, B. (1964). Effects of brain-stem stimulation upon hippocampal electrical activity, somatomotor reflexes and autonomic functions. Electroencephalography and Clinical Neurophysiology, 16, 375-382.

Yokota, T., Sato, A., & Fujimori, B. (1963). Inhibition of sympathetic activity by stimulation of limbic systems. Japanes Journal of Physiology, 13, 138-154.