

CHAPTER EIGHT

THE ELECTRODERMAL SYSTEM

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Prologue

OVERVIEW

Electrodermal activity (EDA) has been one of the most widely used – some might add “abused” – response systems in the history of psychophysiology. A search of the PsychLit computerized database reveals that the use of electrodermal activity in research has remained high and stable over the past several decades. During this time, research involving EDA has been reported in mainstream psychology, psychiatry, and psychophysiology research journals such as the *Archives of General Psychiatry*, *Biological Psychiatry*, *Biological Psychology*, *International Journal of Psychophysiology*, *Journal of Abnormal Psychology*, *Journal of Experimental Psychology*, *Journal of Personality and Social Psychology*, and *Psychophysiology*, but it has also appeared in more surprising venues such as the *International Journal of Eating Disorders*, *Behavior Therapy*, *Crisis*, and the *European Journal of Parapsychology*. The wide range of journals in which EDA research is published is reflective of the fact that EDA measures have been applied to a wide variety of questions – ranging from basic research examining attention, information processing, and emotion to more applied clinical research examining predictors and/or correlates of normal and abnormal behavior. The application of EDA measures to a wide variety of issues is due in large part to its relative ease of measurement and quantification combined with its sensitivity to psychological states and processes.

The purpose of this chapter is to provide a tutorial overview of EDA for the interested student, researcher, or practitioner who is not a specialist in this particular system. We begin with a historical orientation and then discuss the physical, inferential, and psychosocial aspects of EDA.

HISTORICAL BACKGROUND

The Discovery of Electrodermal Activity

The empirical study of the electrical changes in human skin began over 100 years ago in the laboratory of Jean Charcot, the French neurologist famous for his work on hysteria and hypnosis. Vigouroux (1879, 1888), a collaborator of Charcot, measured tonic skin resistance levels from various patient groups as a clinical diagnostic sign. In the same laboratory, Féré (1888) found that, by passing a small electrical current across two electrodes placed on the surface of the skin, one could measure momentary decreases in skin resistance in response to a variety of stimuli (visual, auditory, gustatory, olfactory, etc.). The basic phenomenon discovered by Féré is that the skin momentarily becomes a better conductor of electricity when external stimuli are presented. Shortly thereafter, a Russian physiologist named Tarchanoff (1890) reported that one could measure changes in electrical potential between two electrodes placed on the skin without applying an external current (see Bloch 1993 for interesting details regarding these initial discoveries). Hence, Féré and Tarchanoff are said to have discovered the two basic methods of recording electrodermal activity in use today. Recording the skin resistance (or its reciprocal, skin conductance) response relies on the passage of an external current across the skin and hence is referred to as the *exosomatic* method, whereas recording the skin potential response does not involve an external current and hence is referred to as the *endosomatic* method. The present chapter will focus on the exosomatic method of recording skin resistance and conductance because this clearly is the method of choice among contemporary researchers (Fowles et al. 1981).

It is interesting and somewhat humbling to find that the very early investigators identified many of the important aspects of EDA that remain of interest today. For

example, the tonic-phasic distinction was implied in the earliest of these publications. The tonic level of skin resistance or conductance is the absolute level of resistance or conductance at a given moment in the absence of a measurable phasic response, and it is referred to as SRL (skin resistance level) or SCL (skin conductance level). Superimposed on the tonic level are phasic decreases in resistance (increases in conductance), referred to as SRRs (skin resistance responses) or SCRs (skin conductance responses). Similar distinctions are made with skin potential and are referred to as SPL and SPRs. (It should be noted that other terms have been used to refer to EDA phenomena in the history of this response system, particularly psychogalvanic reflex, PGR, and galvanic skin response, GSR.)

Even more humbling is that many of the variables and phenomena intensively studied today were already being investigated in this early research. For example, the ability of various types of sensory stimuli to elicit phasic EDA changes was clearly in evidence. The fact that stronger stimulation would elicit larger responses, as well as the fact that repetitions of the same stimulus would lead to habituation, were noted. Moreover, the effectiveness of mental images, mental effort (e.g., solving arithmetic problems), emotions, and surprise in eliciting EDA also was demonstrated. Individual differences in EDA were observed, and questions regarding the utility of this new measure in distinguishing normal from pathological groups were being raised. Clearly, these early investigators recognized the psychophysiological significance of this newly discovered phenomenon and laid the foundation for more than a century of subsequent research. A detailed historical review of these early articles is provided by Neumann and Blanton (1970); English translations of some of the classic early reports on electrodermal phenomena can be found in Porges and Coles (1976).

Issues in the History of EDA Research

Several issues identified in this early research have continued to be sources of considerable speculation and investigation throughout the history of this response system. One set of such issues concerns the mechanisms and functions of EDA. In terms of peripheral mechanisms, Vigouroux proposed what became known as the "vascular theory" of EDA (Neumann & Blanton 1970). The vascular theory associated changes in skin resistance with changes in blood flow. Tarchanoff favored a "secretory theory" that related EDA to sweat gland activity. This theory was supported later by Darrow (1927), who measured EDA and sweat secretion simultaneously and found the two measures to be closely related. However, the phasic EDR would begin about 1 sec before moisture would appear on the surface of the skin, so it was concluded that activity of the sweat glands, not sweat on the skin per se, was critical for EDA. (Other lines of evidence indicating that sweat glands are the major contributors to EDA have been reviewed by

Fowles 1986, pp. 74-5.) It was generally known at the time that palmar sweat glands are innervated by the sympathetic chain of the autonomic nervous system, so EDA was said to reflect sympathetic activation. In terms of more central physiological mechanisms, work by early investigators such as Wang and Richter indicated that EDA was complexly determined by both subcortical and cortical areas (for a review of this early research, see Darrow 1937). Darrow proposed that "the function of the secretory activity of the palms is primarily to provide a pliable adhesive surface facilitating tactful acuity and grip on objects" (1937, p. 641).

Issues surrounding the proper methods of recording and quantifying EDA also have been important in the history of this response system. Lykken and Venables noted that EDA provides useful data "in spite of being frequently abused by measurement techniques which range from the arbitrary to the positively weird" (1971, p. 656). In fact, we would date the beginning of the modern era of EDA research to the early 1970s, when Lykken and Venables (1971) proposed standardized techniques of recording skin conductance and standardized units of measurement. This was followed shortly by an edited book (Prokasy & Raskin 1973) that was devoted entirely to EDA and contained several useful review chapters, including a particularly outstanding chapter by Venables and Christie (1973). Published around the same time were several other excellent reviews (Edelberg 1972a; Fowles 1974; Grings 1974). More recently, book-length reviews of EDA have been provided by Boucsein (1992) and by Roy and colleagues (1993a); see also Fowles (1986) and Hugdahl (1995).

Another issue of central importance concerns the psychological significance of EDA. From the beginning, this response system has been closely linked with the psychological concepts of emotion, arousal, and attention. Early in this century, Carl Jung added EDA measurements to his word association experiments in order to objectively measure the emotional aspects of "hidden complexes." An American friend joined Jung in these experiments and in 1907 enthusiastically reported: "Every stimulus accompanied by an emotion produced a deviation of the galvanometer to a degree in direct proportion to the liveliness and actuality of the emotion aroused" (Peterson, cited by Neumann & Blanton 1970, p. 470). About half a century later, when the concept of emotion was less in favor, Woodworth and Schlosberg devoted most of one entire chapter of their classic textbook in experimental psychology to EDA, which they described as "perhaps the most widely used index of activation" (1954, p. 137). They supported this indexing relationship by noting that tonic SCL is generally low during sleep and high in activated states such as rage or mental work. The authors also related phasic SCRs to attention, noting that such responses are sensitive to stimulus novelty, intensity, and significance.

Many of these issues have remained important for contemporary psychophysologists and are discussed in the

remainder of this chapter. In the next section we present a summary of the contemporary perspectives regarding the basic physiological mechanisms and proper recording techniques of EDA.

Physical Context

ANATOMICAL AND PHYSIOLOGICAL BASIS

The skin is a selective barrier that serves the functions of preventing entry of foreign matter into the body and selectively facilitating passage of materials from the bloodstream to the exterior of the body. It aids in the maintenance of water balance and of constant core body temperature, functions accomplished primarily through vasoconstriction/dilation and through variation in the production of sweat. As pointed out by Edelberg (1972a), it is not surprising that an organ with such vital and dynamic functions constantly receives signals from control centers in the brain. Edelberg suggested that we "can listen in on such signals by taking advantage of the fact that their arrival at the skin is heralded by measurable electrical changes that we call electrodermal activity" (1972a, p. 368).

There are two forms of sweat glands in the human body: the eccrine, which have been of primary interest to psychophysologists, and the apocrine, which have been relatively unstudied. The distinction between these two is usually made on the basis of location and function (Robertshaw 1983). Whereas apocrine sweat glands typically open into hair follicles and are found primarily in the armpits and the genital areas, eccrine glands cover most of the body and are most dense on the palms and soles of the feet. The function of the apocrine glands is not yet well understood, and this may account for the subordinate status they currently hold within the field of psychophysiology. However, there have been some recent suggestions in the literature that apocrine glands may be more interesting than was once believed. For example, in mammals such as dogs and monkeys, apocrine glands are believed to produce a secretion that serves – when modified by bacteria on the surface of the skin – as an identifying or sexual scent hormone (pheromone). Some authors (e.g. Jakubovic & Ackerman 1985) have suggested that apocrine glands in humans may serve a similar function. It has also been noted that there is some evidence suggesting that apocrine gland secretion is induced by any emotional stress that causes sympathetic nervous system discharge (Jakubovic & Ackerman 1985). To date, the evidence still appears to be inconclusive, and the responsivity of the apocrine glands to emotional, stressful, or sexually arousing stimuli is still under debate (Shields et al. 1987).

In contrast to the apocrine gland, a great deal is known about the function of the eccrine sweat gland. For example, it is known that the primary function of most eccrine

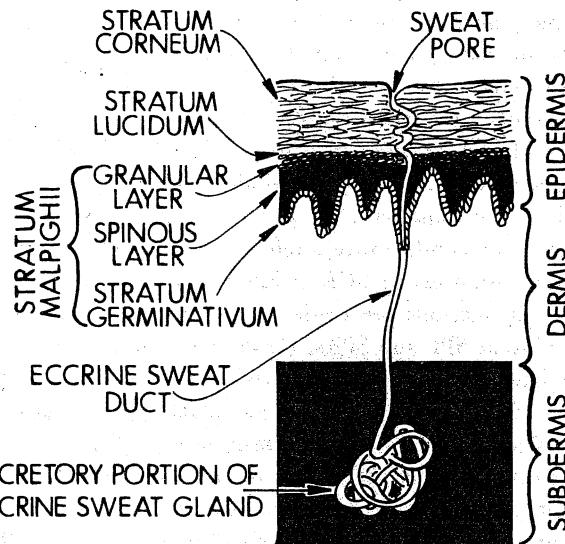


Figure 1. Anatomy of the eccrine sweat gland in various layers of skin. Adapted with permission from Hassett, *A Primer of Psychophysiology*. Copyright 1978 W. H. Freeman and Company.

sweat glands is thermoregulation. However, those located on the palmar and plantar surfaces have been thought of as being more concerned with grasping behavior than with evaporative cooling (Edelberg 1972a), and it has been suggested that they are more responsive to significant or emotional stimuli than to thermal stimuli. Although all eccrine glands are believed to be involved in emotion-evoked sweating, such sweating is usually most evident in these areas primarily because of the high gland density (Shields et al. 1987). The measurement of EDA by psychophysologists is primarily concerned with this psychologically induced sweating.

Figure 1 shows the basic peripheral mechanisms involved in the production of EDA. The extreme outer layer of the skin, the stratum corneum or horny layer, consists of a dead layer of cells that serves to protect the internal organs. Below the stratum corneum lies the stratum lucidum, and just below that is the stratum Malpighii. The stratum Malpighii actually consists of three cell layers: the granular layer; the spinous layer; and the deepest layer – the stratum germinativum – which consists of cells that are continually reproducing and replacing the dead cells on the skin's surface. The eccrine sweat gland itself consists of a coiled compact body that is the secretory portion of the gland and the sweat duct, the long tube that is the excretory portion of the gland. The sweat duct remains relatively straight in its path through the stratum Malpighii and stratum lucidum; it then spirals through the stratum corneum and opens to the surface of the skin as a small pore (Edelberg 1972a).

Many models have been suggested to explain how these peripheral mechanisms relate to the electrical activity of the skin and to the transient increases in skin conductance elicited by stimuli. The dominant view has been a

two-effector model summarized by Edelberg (1972a). According to this model, there are two peripheral mechanisms that contribute to EDA: (i) secretion of sweat from the sweat gland and the attendant filling of the sweat duct; and (ii) the activity of a selective membrane that lies somewhere in the epidermis. However, there have been lingering questions about the selective membrane, leading to the proposal of alternative models (Fowles 1986). More recently, Edelberg (1993) concluded that it is not necessary to hypothesize participation of an active membrane in EDA. Instead, one can account for the variety of electrodermal phenomena – including changes in tonic SCL and phasic SCR amplitude and recovery – with a model based on the single effector of the sweat glands (see Edelberg 1993 for details regarding the origins of the original two-effector model and the proposed single-effector model).

To understand how electrodermal activity is related to the sweat glands, it is useful to think of the sweat ducts (the long tubular portion of the gland that opens onto the skin surface) as a set of variable resistors wired in parallel. Columns of sweat will rise in the ducts in varying amounts and in varying numbers of sweat glands, depending on the degree of sympathetic activation. As sweat fills the ducts, there is a more conductive path through the relatively resistant corneum. The higher the sweat rises, the lower the resistance in that variable resistor. Any change in the level of sweat in the ducts changes the values of the variable resistors and yields observable changes in EDA.

Historically, both the sympathetic and parasympathetic divisions of the autonomic nervous system (ANS) were considered as possible mediators of EDA. This is partially due to the fact that the neurotransmitter involved in the mediation of eccrine sweat gland activity is acetylcholine, which is generally a parasympathetic neurotransmitter, rather than noradrenaline, the neurotransmitter typically associated with peripheral sympathetic activation (Venables & Christie 1980). Now, however, it is generally agreed that (i) human sweat glands have predominately sympathetic cholinergic innervation from sudomotor fibers originating in the sympathetic chain but that (ii) some adrenergic fibers also exist in close proximity (Shields et al. 1987). Convincing evidence for the sympathetic control of EDA has been provided by studies measuring sympathetic action potentials in peripheral nerves while simultaneously recording EDA. The results have shown that, within normal ranges of ambient room temperature and subject thermoregula-

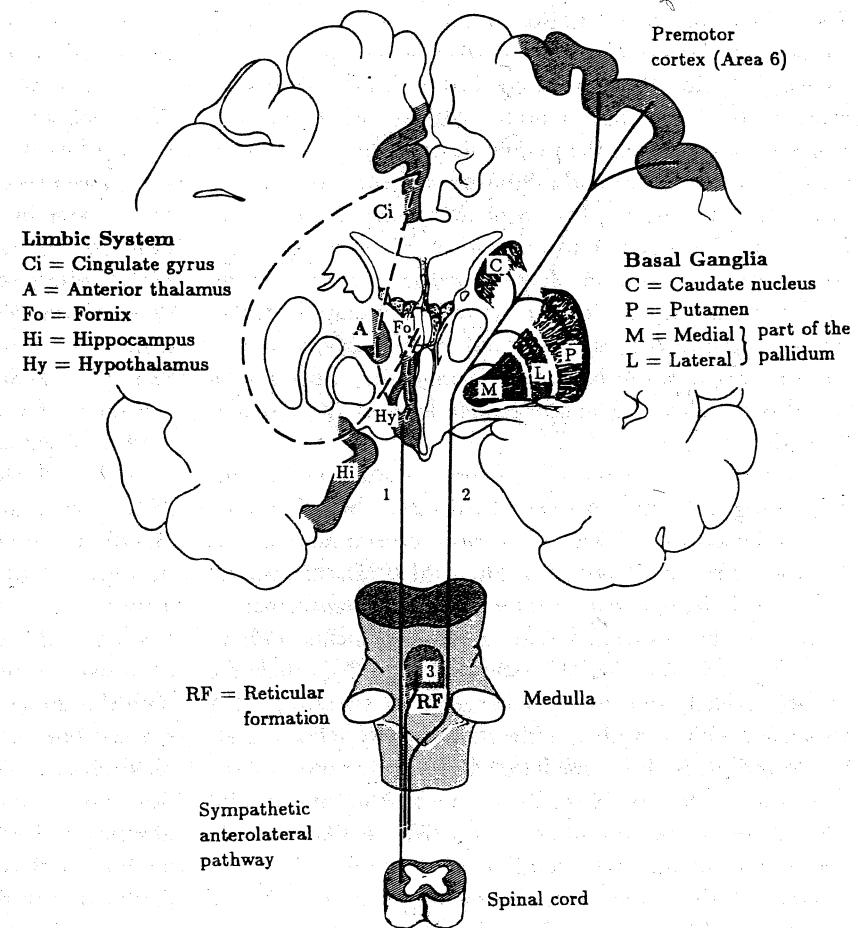


Figure 2. Central nervous system determiners of EDA in humans. See text for discussion of the three pathways shown. Reprinted with permission from Boucsein, *Electrodermal Activity*. Copyright 1992 Plenum Press.

tory states, there is a high correlation between bursts of sympathetic nerve activity and SCRs (Wallin 1981).

Excitatory and inhibitory influences on the sympathetic nervous system are distributed in various parts of the brain, so the neural mechanisms and pathways involved in the central control of EDA are numerous and complex. These mechanisms have been reviewed by Edelberg (1972a) and Venables and Christie (1973) and, more recently, by Boucsein (1992), Hugdahl (1995), Raine and Lencz (1993), Roy, Sequeira, and Delerm (1993b), and Sequeira and Roy (1993).

Boucsein (1992, pp. 30–6) followed the suggestions of Edelberg (1972a) in describing at least two and possibly three relatively independent pathways that lead to the production of SCRs (see Figure 2). The first level of EDA control involves ipsilateral influences from the hypothalamus and limbic system (Sequeira & Roy 1993). There is considerable evidence of an excitatory hypothalamic descending control of EDA. Limbic influences are complicated, but there is evidence of excitatory influences from the amygdala and inhibitory effects originating from the hippocampus.

The *second* and highest level of central EDA control involves contralateral cortical and basal ganglion influences (Sequeira & Roy 1993). One cortical pathway involves excitatory control by the premotor cortex (Brodmann area 6) descending through the pyramidal tract, and another involves both excitatory and inhibitory influences originating in the frontal cortex. The *third* and lowest-level mechanism is in the reticular formation in the brainstem (Roy et al. 1993b). Activation of the reticular formation by direct electrical stimulation or sensory stimulation evokes skin potential responses in cats and (presumably) skin conductance responses in humans. An inhibitory EDA system has also been located in the bulbar level of the reticular formation.

Given the roles of the various brain centers just identified, different functional roles of EDA have been hypothesized to be associated with the various central mechanisms (Boucsein 1992; Edelberg 1973; Hugdahl 1995). Electrodermal activity elicited by activation of the reticular formation is likely to be associated with gross movements and increased muscle tone; EDA associated with hypothalamic activity is likely due to thermoregulatory sweating; EDA associated with amygdala activation is likely reflecting affective processes; EDA mediated by the premotor cortex may occur in situations requiring fine motor control; and EDA elicited by prefrontal cortical activity is likely associated with orienting and attention.

Most of the evidence regarding the central pathways that control EDA is derived from animal studies, usually cats (see e.g. Roy et al. 1993b; Wang 1964). More recently, neural mechanisms of human EDA have been studied with neuroimaging techniques (Raine & Lencz 1993), in patients with focal cerebral lesions (Tranel & Damasio 1994), and in patients with direct electrical stimulation of brain structures (Mangina & Beuzeron-Mangina 1996). These lines of research are just beginning and much remains to be done. However, the results are likely to be complex given the multiple central determiners of EDA. As useful as EDA is for indexing psychological processes, it may be more difficult to identify specific brain centers and pathways given its diffuse levels of control.

PHYSICAL RECORDING BASIS

As briefly described earlier, EDA is measured by passing a small current through a pair of electrodes placed on the surface of the skin. The principle invoked in the measurement of skin resistance or conductance is that of Ohm's law, which states that skin resistance (R) is equal to the voltage (V) applied between two electrodes placed on the skin surface divided by the current (I) being passed through the skin; that is, $R = V/I$. If the current is held constant then one can measure the voltage between the electrodes, which will vary directly with skin *resistance*. Alternatively, if the voltage is held constant then one can

measure the current flow, which will vary directly with the reciprocal of skin resistance, skin *conductance*.

Lykken and Venables (1971) argued strongly for the direct measurement of skin conductance with a constant-voltage system rather than measuring skin resistance with a constant-current system. This argument was based in part on the fact that skin conductance had been shown to be more linearly related to the number of active sweat glands and their rate of secretion. This is so because the individual sweat glands function as resistors in parallel, and the conductance of a parallel circuit is simply the sum of all of the conductances in parallel. On the other hand, the overall resistance of a parallel circuit is a complex function of each of the individual resistances. Thus, unlike the relationship of SRR and SRL, the SCR is potentially independent of SCL, since a given increment in the number of active sweat glands will produce the same increment in the total conductance of the pathway regardless of the level of basal activity. Boucsein (1992, pp. 208–16) stated that the discussion of this issue often confounds the choice of method of measurement (constant voltage versus constant current) with the choice of units of measurement (conductance versus resistance). He raised thoughtful questions about the bioelectrical assumptions underlying the parallel conduction model. Nevertheless, Boucsein agreed on pragmatic grounds with the recommendation to use constant-voltage methods and skin conductance units (for the sake of standardization in the field).

A description of constant-voltage circuits that allow the direct measurement of skin conductance can be found in Lykken and Venables (1971) as well as in Fowles et al. (1981), and most of the physiological recording systems currently on the market include constant-voltage couplers for the direct recording of skin conductance.

EDA Recording Systems

The decision of whether to record skin conductance or skin resistance and the availability of a constant-voltage coupler for direct recording of skin conductance are important factors that should be considered when choosing an EDA recording system. In addition, serious consideration should be given to the issue of paper-based versus paperless systems.

Paper-based systems provide a continuous on-line hard-copy record of the EDA recording over the experimental session. The advantages of paper-based systems are: (1) they can provide a continuous record of an entire experimental session; (2) they provide an easy means for the experimenter to "flag" important events such as participant movement that may produce artifact in the recording (the experimenter simply writes on the polygraph chart "participant moved here"); and (3) they are not dependent on expensive computer interfaces or complex software, since the EDA can be quantified by "hand scoring" directly from the paper record. However, there are three disadvantages

of paper-based systems that have led to the search for alternatives: (1) the cost of polygraph paper, ink, and pens; (2) the potential for malfunction and mess that is invariably associated with a mechanical ink-on-paper system; and, most importantly, (3) the time required for (and potential unreliability of) quantification from the paper record. To deal with the latter issue, most researchers using paper-based systems also collect EDA on a computer using an analog-to-digital converter and specialized computer programs for quantification (see the quantification section later in this chapter). However, this computer sampling is generally carried out on an event-by-event basis rather than as a continuous recording.

In contrast to a paper-based system, a paperless system involves the digitization and storage of EDA by a computer with no on-line hard-copy record. In most paperless systems, a researcher must select the time points at which the computer will sample the EDA. This sampling window has traditionally been event-related, such as following each presentation of an experimental stimulus. In these cases, EDA at all other time points is lost. Fortunately, with expanding computing capability, it is now possible for a paperless system to sample EDA continuously, to allow an experimenter to flag critical events with a keypress, and to provide a continuous printout of an experimental session. Therefore, in choosing an EDA recording system, one must consider its output capabilities (paper only, computer only, or paper and computer) and also its computing capabilities and software issues as well. For example, some manufacturers offer software packages for the acquisition of EDA, some offer software for the quantification of EDA, and some offer both.

In addition to selecting an EDA recording system, special consideration must be given to the choice of recording electrodes, electrode paste, and electrode placement. Silver-silver chloride cup electrodes are the type most typically used in skin conductance recording because they minimize the development of bias potentials and polarization. These electrodes can be easily attached to the recording site through the use of double-sided adhesive collars, which also serve the purpose of helping to control the size of the skin area that comes in contact with the electrode paste. This is an important parameter because it is the contact area, not the size of the electrode, that affects the conductance values.

The electrode paste is the conductive medium between electrode and skin. Probably the most important concern in choosing an electrode paste is that it preserve the electrical properties of the bioelectrical signals of the response system of interest. Since the measurement of EDA involves a small current passed through the skin, the electrode paste interacts with the tissue over which it is placed. For this reason, the use of a paste that closely resembles sweat in its salinity is recommended (Venables & Christie 1980). Instructions for making such paste are given in Fowles et al.

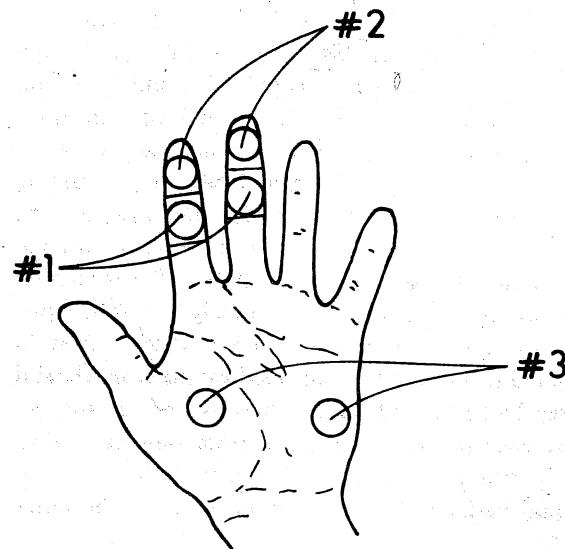


Figure 3. Three electrode placements for recording electrodermal activity. Placement #1 involves volar surfaces on medial phalanges, placement #2 involves volar surfaces of distal phalanges, and placement #3 involves thenar and hypothenar eminences of palms.

(1981, p. 235) and Grey and Smith (1984, p. 553). Commercial ECG or EEG gels should not be used because they usually contain near-saturation levels of sodium chloride and have been shown to significantly inflate measures of skin conductance level (Grey & Smith 1984).

Skin conductance is recorded with both electrodes on active sites (bipolar recording); hence it does not matter in which direction the current flows between the two electrodes. Skin conductance recordings are typically taken from locations on the palms of the hands, with several acceptable placements. The most common electrode placements are the thenar eminences of the palms and the volar surface of the medial or distal phalanges of the fingers (see Figure 3). It should be noted that, although electrodermal activity can be measured from any of these sites, the values obtained are not necessarily comparable. Scerbo and colleagues (1992) made a direct comparison of EDA recorded from the distal and medial phalange sites simultaneously and found that both the elicited SCR amplitude and SCL were significantly higher from the distal recording site. Moreover, the greater level of reactivity at the distal site was found to be directly related to a larger number of active sweat glands at that location (Freedman et al. 1994). Therefore, the distal phalange site is recommended unless there are specific reasons for not using the distal site (e.g., recording from children whose fingertips may be too small for stable electrode attachment, presence of cuts or heavy calluses on the fingertips, etc.).

Another recording issue concerns the hand from which to record. Many laboratories use the nondominant hand for EDA measurements because it is less likely to have cuts or calluses and because it leaves the dominant hand free to perform a manual task. However, this begs the question

of whether there are significant laterality differences in EDA. Although differences between left- and right-hand EDA recordings have been reported, the differences reported by different studies are often in opposite directions and the interpretations have been ambiguous (see a review of early literature by Hugdahl 1984). It is tempting to speculate that the prior conflicting findings may be due to the lack of clear distinctions between emotional and nonemotional tasks (Hugdahl 1995). Electrodermal activity in emotional tasks is presumably controlled primarily by the ipsilateral limbic system, whereas EDA in nonemotional tasks may be controlled by the contralateral system (see Figure 2). However, there is not yet any definitive evidence that EDA recorded from one hand gives consistently different results with respect to the effects of experimental variables than that recorded from the other hand.

Because it is critical in EDA recording that the electrical properties of the response system be preserved, the electrode sites for bipolar recording should not receive any special preparation such as cleaning with alcohol or abrading the skin, which might reduce the natural resistive/conductive properties of the skin. However, since a fall in conductance has been noted following the use of soap and water (Venables & Christie 1973) and since the length of time since the last wash will be variable across subjects when they arrive at the laboratory, these authors recommended that subjects be asked to wash their hands with a nonabrasive soap prior to having the electrodes attached. It is recommended that the electrodes be kept on the same hand to avoid ECG (electroencephalographic) artifact and that the placement sites be clean and dry (Venables & Christie 1973).

Inferential Context

QUANTIFICATION PROCEDURES

Figure 4 shows tracings of two hypothetical skin conductance recordings during a 20-sec rest period and then during three repetitions of a simple discrete stimulus (e.g., a mild tone). Several important aspects of EDA can be seen in the figure. First, it can be seen that tonic SCL begins at $10 \mu\text{S}$ (microsiemens) in the upper tracing and at $5 \mu\text{S}$ in the lower tracing. Although tonic SCL can vary widely between different subjects and within the same subject in different psychological states, the typical range is between $2 \mu\text{S}$ and $20 \mu\text{S}$ with the types of apparatus and procedures described here. Computing the log of SCL can significantly reduce skew and kurtosis in the SCL data and is recommended by Venables and Christie (1980).

It can also be seen in the lower tracing of Figure 4 that the SCL drifts downward from $5 \mu\text{S}$ to nearly $4 \mu\text{S}$ during the rest period. It is common for SCL to gradually decrease while subjects are at rest, then rapidly increase

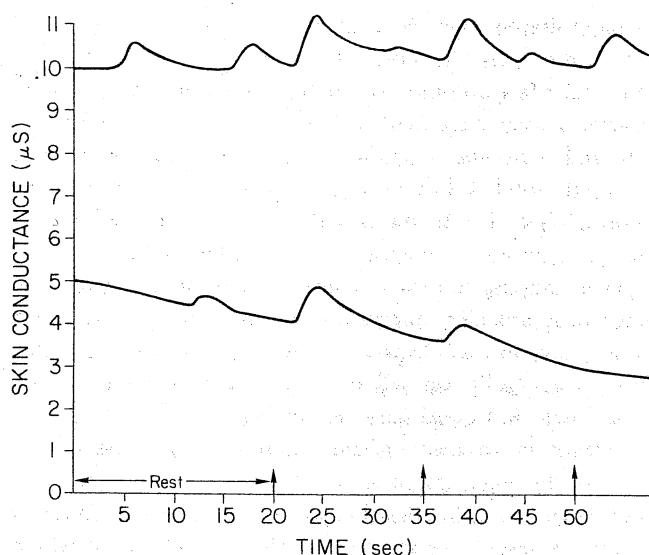


Figure 4. Two hypothetical skin conductance recordings during 20 sec of rest followed by three repetitions of a simple discrete stimulus. Arrows represent the presentation of a stimulus. Reprinted from Dawson & Nuechterlein (1984), "Psychophysiological dysfunctions in the development course of schizophrenic disorders," *Schizophrenia Bulletin*, vol. 10, pp. 204-32.

when novel stimulation is introduced, and finally gradually decrease again when the stimulus is repeated.

Phasic SCRs are only a small fraction of the SCL and have been likened to small waves superimposed on the tidal drifts in SCL (Lykken & Venables 1971). If the SCR occurs in the absence of an identifiable stimulus, as shown in the rest phase of Figure 4, it is referred to as a "spontaneous" or "nonspecific" SCR (NS-SCR). The most widely used measure of NS-SCR activity is its rate per minute – typically between 1 and 3 while the subject is at rest. However, responses can be elicited by sighs, deep breaths, and bodily movements, so unless these also are recorded it is impossible to say which responses truly are NS-SCRs.

Presentation of a novel, unexpected, significant, or aversive stimulus will likely elicit what is known as a "specific" SCR. With the exception of responses elicited by aversive stimuli, these SCRs are generally considered to be components of the orienting response. As is also the case with NS-SCRs, one must decide upon a minimum amplitude change in conductance to count as an elicited SCR. A common minimum response amplitude is $0.05 \mu\text{S}$, which is a largely arbitrary value that happens to be the minimum change that can be reliably detected through visual inspection of most polygraph paper recordings. This arbitrary minimum is decreasing as computer scoring becomes more popular, because the computer can reliably detect much smaller changes. Another decision regarding scoring of SCRs concerns the latency window of time during which time a response will be assumed to be elicited by the stimulus. Based on frequency distributions of response latencies to simple stimuli, it is common to use a 1-3-sec

TABLE 1. Electrodermal Measures, Definitions, and Typical Values

Measure	Definition	Typical Values
Skin conductance level (SCL)	Tonic level of electrical conductivity of skin	2–20 μ S
Change in SCL	Gradual changes in SCL measured at two or more points in time	1–3 μ S
Frequency of NS-SCRs	Number of SCRs in absence of identifiable eliciting stimulus	1–3 per min
ER-SCR amplitude	Phasic increase in conductance shortly following stimulus onset	0.2–1.0 μ S
ER-SCR latency	Temporal interval between stimulus onset and SCR initiation	1–3 sec
ER-SCR rise time	Temporal interval between SCR initiation and SCR peak	1–3 sec
ER-SCR half recovery time	Temporal interval between SCR peak and point of 50% recovery of SCR amplitude	2–10 sec
ER-SCR habituation (trials to habituation)	Number of stimulus presentations before two or three trials with no response	2–8 stimulus presentations
ER-SCR habituation (slope)	Rate of change of ER-SCR amplitude	0.01–0.05 μ S per trial

Key: ER, event-related; NS, nonspecific; SCR, skin conductance response.

or 1–4-sec latency window. Hence, any SCR that begins between one and three (or four) seconds following stimulus onset is considered to be elicited by that stimulus. It is important to select reasonably short latency windows, perhaps even shorter than 1–3 sec, so as to not have the NS-SCR rate contaminate the measurement of elicited SCRs (Levinson, Edelberg, & Bridger 1984).

An important advance in EDA research has been the development of computerized scoring programs. Scoring software is available from the manufacturers of several EDA recording systems, and customized software or shareware is frequently used as well. One example of shareware is SCRGauge by Peter Kohlisch, available in Boucsein (1992). Another shareware with a long history is SCORIT 1980 (Strayer & Williams 1982), which is a revision of SCORIT (Prokasy 1974). Interested readers may contact Dr. William C. Williams (BWilliams@EWU.edu) for an updated version of SCORIT 1980. Computer scoring is significantly less time-consuming than hand scoring and has the advantage of ensuring perfect reliability (the computer will score the same response the same way every time and will apply the same rules to every response). Another advantage of computerized scoring is the availability of sophisticated algorithms such as that proposed by Lim and associates (1997) for discerning individual components of skin conductance responses. As mentioned before, another advantage of computerized acquisition and scoring is that a computer can detect responses that are too small to be scored reliably by hand. Depending on the range of the analog-to-digital converter and the sensitivity of the skin conductance coupler, responses as small as 0.008 μ S may be detected and scored. However, whether this is a psychologically or physiologically meaningful change in conductance remains to be determined.

Having decided on a minimum response amplitude and a latency window in which a response will be considered as an "event-related" SCR, one can measure several aspects of the elicited SCR besides its mere occurrence and frequency. Definitions and typical values of the major EDA component measures are given in Table 1 and are shown graphically in Figure 5. For example, size of the SCR is quantified as the amount of increase in conductance measured from the onset of the response to its peak. The size of an elicited SCR typically ranges between 0.2 μ S and 1.0 μ S.

When a stimulus is repeated several times and an average size of the SCR is to be calculated, one may choose to compute either mean SCR amplitude or mean SCR magnitude. *Magnitude* refers to the mean value computed across all stimulus presentations including those without a measurable response, whereas *amplitude* is the mean value computed across only those trials in which a measurable (nonzero) response occurred (Humphreys 1943). Prokasy and Kumpfer (1973) argued strongly against use of the magnitude measure in large part because it confounds frequency and amplitude, which do not always covary. A magnitude measure can create the impression that the

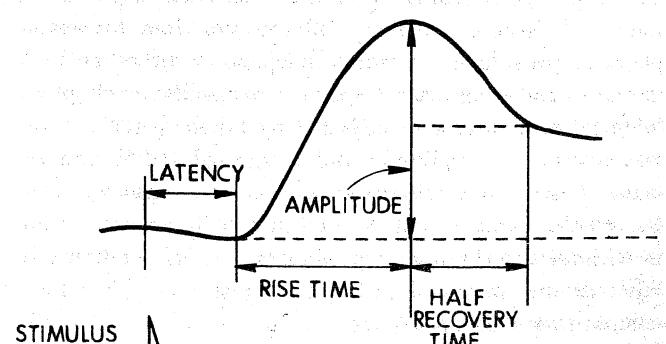


Figure 5. Graphical representation of principal EDA components.

response size is changing when in fact it is the response frequency that is changing. Hence, these authors recommend separate assessments of frequency and amplitude rather than magnitude. However, it is important to note that a complication with the amplitude measure is that the N used in computing average response size can vary depending on how many measurable responses a subject gives, and the data of subjects without any measurable response must be eliminated. Thus, a subject who responds on each of ten stimulus presentations with a response of $0.50 \mu\text{S}$ will have the same mean SCR amplitude as a subject who responds on only the first stimulus presentation with a response of $.50 \mu\text{S}$ but does not respond thereafter. We concur with Venables and Christie (1980) that there are arguments for and against both amplitude and magnitude; although no absolute resolution is possible, it is important to keep the difference between the two measures clearly in mind. In fact, we recommend that in most situations it is reasonable to compute and compare results obtained with SCR frequency, amplitude, and magnitude.

Like SCL, SCR amplitude and magnitude are frequently found to be positively skewed and also leptokurtotic, so a logarithmic transformation is often used to remedy these problems. If measurements are being made of SCR magnitude, so that zero responses are included, then $\log(\text{SCR} + 1.0)$ may be calculated, since the logarithm of zero is not defined (Venables & Christie 1980). Another common practice is to use a square root transformation, $\sqrt{\text{SCR}}$, to normalize response amplitude data; this does not require the addition of a constant (Edelberg 1972a). In some cases the choice of the square-root or logarithmic transformation should be guided by considerations of achieving or maintaining the homogeneity of variance across several groups (Winer, Brown, & Michels 1991, pp. 356–8). If skew, kurtosis, or homogeneity of variance problems do not exist in a particular set of data, transformations need not be performed.

In addition to response size, one can also measure temporal characteristics of the SCR including onset latency, rise time, and half-recovery time. These temporal characteristics of the SCR waveform are not as commonly quantified as amplitude, and their relationship to psychophysiological processes is not as well understood at this time. The issue of whether SCR recovery time, for example, can provide information independent of other EDA measures and is uniquely responsive to specific psychophysiological processes was suggested by Edelberg (1972b) but was questioned by Bundy and Fitzgerald (1975) and remains unsettled (Edelberg 1993, pp. 14–15; Fowles 1986, pp. 84–7). This is not to say that SCR recovery time is without discriminating power; rather, its qualitatively different informational properties relative to other EDA components is an open issue.

The usual constellation of EDA components is for high SCL, frequent NS-SCRs, large SCR amplitude, short la-

tency, short rise time, and short recovery time to cluster together. However, the correlations among the EDA components generally are not very high, usually less than 0.60 (Lockhart & Lieberman 1979; Venables & Christie 1980). The size and consistency of these relationships is compatible with the hypothesis that many of the EDA components may represent partially independent sources of information, although – as indicated with regard to SCR recovery time – this is a controversial hypothesis. The one exception to the weak relationships among EDA components is the consistently high correlation between SCR rise time and recovery time. Based on this relation, Venables and Christie (1980) suggested that SCR rise time and half-recovery time may be essentially redundant measures and that – since recovery time is not always as available as rise time (owing to subsequent NS-SCRs) – rise time may be the preferred measure.

A problem with quantifying the SCR components occurs when the response to be scored is elicited before an immediately preceding response has had time to recover. It is customary to measure the amplitude of each response from its own individual deflection point (Edelberg 1967; Grings & Lockhart 1965). However, the amplitude as well as the temporal characteristics of the second response are distorted by being superimposed on the recovery of the first response. For example, the measurable amplitude of the second response will be smaller given its occurrence following the first response. The amount of distortion of the second response is a function of the size of the first response and the time since the first response (Grings & Schell 1969). Although there is no good solution to the response interference effect when hand scoring EDA, it can be pointed out that response frequency may be the least distorted component of the response in this situation. In addition, as mentioned earlier, one advantage of computerized scoring of EDA is the availability of more sophisticated scoring algorithms. In this regard, Lim et al. (1997) conducted an investigation in which they applied a multiparameter curve-fitting algorithm to the scoring of overlapping skin conductance responses; they were able to decompose the overall response complex into meaningful components of the separate responses.

Another problem with quantifying the EDA components concerns the existence of large variability due to extraneous individual differences. Thus, whether an SCL of $8 \mu\text{S}$ is considered high, moderate, or low will depend upon that specific subject's range of SCLs. For example, one can see in Figure 4 that an SCL of $8 \mu\text{S}$ would be relatively low for the subject depicted in the upper tracing but would be relatively high for the subject depicted in the lower tracing. Similarly, an SCR of $0.5 \mu\text{S}$ may be relatively large for one person but relatively small for another. Lykken and colleagues (1966) proposed an interesting method to correct for this interindividual variance. The correction procedure involves computing a range for each individual subject and

then expressing the subject's momentary value as a proportion of this range. For example, one may compute a subject's minimum SCL during a rest period and a maximum SCL while the subject blows a balloon to bursting; the subject's present SCL can then be expressed as a proportion of his or her individualized range according to the following formula: $(SCL - SCL_{min}) / (SCL_{max} - SCL_{min})$.

For SCR data, the minimum SCR can be assumed to be zero and the maximum can be estimated by the presentation of some strong or startling stimulus. Each SCR can then be corrected for individual differences in range simply by dividing each SCR by that subject's maximum SCR. The rationale underlying these procedures is that an individual's maximum and minimum SCL and SCR are due mainly to physiological differences (e.g., thickness of the corneum) that are unrelated to psychological processes. It is the variation within these physiological limits that is normally of psychological interest (Lykken & Venables 1971).

Although the range correction procedure can reduce error variance and increase the power of statistical tests in some data sets, it also can be problematic in others. For example, range correction would be inappropriate in a situation where two groups being compared had different ranges (Lykken & Venables 1971). Also, the range correction procedure relies upon adequate and reliable estimates of maximum and minimum values, yet estimates of these individual extreme values can be unreliable. For this reason, Ben-Shakhar (1985) recommended using within-subject standardized scores to adjust for individual differences because this transformation relies upon the mean, a more stable and reliable statistic than the maximum response. For further discussion of these and other transformation procedures, see Boucsein (1992, pp. 150–7).

Another important aspect of elicited SCRs is their decline in amplitude and eventual disappearance with repetition of the eliciting stimulus (SCR habituation). Habituation is a ubiquitous and adaptive phenomenon whereby subjects become less responsive to familiar and nonsignificant stimuli. There are several methods of quantifying habituation of the SCR (Siddle, Stephenson, & Spinks 1983). One simple method involves counting the number of stimulus repetitions required to reach some predetermined level of habituation (e.g., two or three consecutive trials without measurable SCRs). This "trials-to-habituation" measure is useful and has been widely employed since its use by Sokolov (1963), but it is subject to considerable distortion by the occurrence of a single response. For example, whether an isolated SCR occurs on trial 3 can make the difference between a trials-to-habituation score of "0" (indicative of an atypical nonresponder) and a "3" (indicative of a typical rate of habituation).

Another common measure of habituation is based on the rate of decline of SCR magnitude across trials as assessed by a "trials" main effect or interaction effect within an analysis of variance. However, this measure does not

provide information about habituation in individual subjects and moreover can be distorted by differences in initial levels of responding.

A third measure of habituation is based on the regression of SCR magnitude on the log trial number (Lader & Wing 1969; Montague 1963). The regression approach provides a slope and an intercept score (the latter reflecting initial response amplitude), which usually are highly correlated with each other. Covariance procedures have been used to remove the dependency of slope on intercept, providing what Montague (1963) called an "absolute rate of habituation." However, this technique rests on the assumptions that (i) slope and intercept reflect different underlying processes and (ii) the treatment effects under investigation do not significantly affect the intercepts (Siddle et al. 1983). Use of the slope measure also assumes that subjects respond on a sufficient number of trials to compute a meaningful slope measure, which may not be the case for some types of subjects with mild innocuous stimuli. Nevertheless, to the extent that these assumptions can be justified, the slope measure is often preferable because: (1) unlike the analysis-of-variance approach, individual habituation scores can be derived; (2) unlike the trials-to-habituation measure, isolated SCRs have less of a contaminating effect; (3) unlike the trials-to-habituation measure, the slope measure makes fuller use of the magnitude data; and (4) unlike the trials-to-habituation measure, the slope measure can discriminate between subjects who show varying degrees of habituation but who fail to completely stop responding for two or three consecutive trials.

THREE TYPES OF PARADIGMS IN WHICH EDA IS OFTEN USED

Now that the principal EDA components have been identified and defined, we can describe basic experimental paradigms used to study the relationships of these components to psychological states and processes. In this section we will identify three general types of paradigms: those that involve (1) the presentation of discrete stimuli, (2) the presentation of chronic stimuli, and (3) the measurement of individual differences in EDA. In the next section we discuss specific applications and theoretical issues involved in these paradigmatic studies of EDA.

The SCR is elicited by almost any novel, unexpected, potentially important, discrete stimulus in the environment, as well as by the omission of an expected stimulus (Siddle 1991). One of the most widely used paradigms in psychophysiology involves measuring the elicitation and habituation of various indices of the orienting response, of which the SCR is a reliable and easily measurable component. This "orienting response habituation" paradigm typically consists of the repetitive presentation of a simple discrete innocuous stimulus (commonly a 1-sec tone of approximately 75 dB) with interstimulus intervals varying

between 20 sec and 60 sec. The initial elicitation and subsequent habituation of the SCR can be measured in this paradigm as a component of the orienting response. The typical finding with this paradigm is that the SCR rapidly declines in amplitude with stimulus repetition and eventually disappears completely. The rate of the decline and disappearance of the SCR varies with such factors as stimulus significance, stimulus intensity, and the length of the interval between stimuli (see review by Siddle et al. 1983). The shape of the response also changes with stimulus repetition as the latency, rise time, and half-recovery time become longer (Lockhart & Lieberman 1979). Moreover, the background tonic SCL generally declines and the frequency of NS-SCRs becomes less. All in all, the picture that emerges is one of a less active and less reactive response system with stimulus repetition.

Next we turn to a paradigm that involves the presentation of a continuous, chronic stimulus or situation such as that involved in performing an ongoing task. Bohlin (1976), for example, required one group of subjects to perform a series of arithmetic problems; a second group of subjects was threatened with delivery of electric shock for poor performance on the arithmetic task. Both groups then were presented a series of discrete innocuous tones during the task so that the elicitation and habituation of SCRs could be measured. It was found that the ongoing task (and threat) (1) increased SCL, (2) reversed the usual decline over time in SCL, (3) increased the overall frequency of NS-SCRs, (4) increased the frequency and magnitude of elicited SCRs, and (5) retarded the rate of elicited SCR habituation (at least with the trials-to-habituation measure). These results demonstrated the sensitivity of these EDA components to simple but powerful manipulations in the ongoing stimulus situation.

The EDA "individual difference" paradigm is fundamentally different from the preceding two paradigms. Here, EDA is assumed to be a relatively stable subject trait related to behavioral and psychological individual differences. Consistent with this assumption is the fact that EDA components exhibit moderate test-retest stability. The test-retest reliability coefficients generally range between 0.50 and 0.70 when measured over a range encompassing a few days to a year or longer (see Freixa i Baqué 1982 for a review of early studies; for more recent findings see Fredrikson et al. 1993 or Schell, Dawson, & Filion 1988). Another line of evidence consistent with the assumptions of the individual difference paradigm is that many of the EDA components have a partial genetic influence (Lykken et al. 1988).

Advantages and Disadvantages of the Use of EDA

When one is considering the use of EDA as an indicator of some psychological state or process of interest, it is well to

remember that, in the great majority of situations, changes in electrodermal activity do not occur in isolation. Rather, they occur as part of a complex of responses mediated by the autonomic nervous system.

Experimental operations such as those just described that have the effect of increasing SCL and/or NS-SCR rate (e.g. Bohlin 1976) also would generally be expected to increase heart rate level and blood pressure and to decrease finger pulse volume, to mention a few of the more commonly measured ANS responses (Engel 1960; see also Grings & Dawson 1978). Stimuli that elicit an SCR would also be expected to elicit certain components of heart rate response (Bohlin & Kjellberg 1979; Graham & Clifton 1966) and a peripheral vasoconstriction (Uno & Grings 1965), and these response components would ordinarily be increased in magnitude by the same operations that would increase SCR magnitude. The response or responses chosen for monitoring by a particular investigator should reflect such considerations as those discussed next.

Although it is true that operations which alter EDA also typically alter other ANS and nonautonomic response measures, so that the electrodermal response occurs as part of a response complex, it is also true that different components of that response complex may correlate poorly with each other across individuals. The poor intercorrelation across subjects of the different components of the global ANS response pattern reflects in large part what is termed *individual response stereotypy* (Engel 1960; Lacey & Lacey 1958; Wenger et al. 1961). That is, individuals tend (some more strongly than others) to produce the same pattern of relative change across physiological systems, and this pattern differs across individuals. Thus, the decision about which particular physiological response to monitor is an important one.

Before considering examples of the three types of paradigms in which EDA is frequently employed, the reader should consider that, as with any response system, the measurement of EDA has both advantages and disadvantages. It is well to keep these in mind while reviewing specific examples.

For some researchers, EDA may be the response system of choice because, unlike most ANS responses, it provides a direct and undiluted representation of sympathetic activity. As we have pointed out, the neural control of the eccrine sweat glands is entirely under sympathetic control. Therefore, if SCL is observed to increase in a situation or if the SCR is enhanced, then this can be due only to increased tonic or phasic sympathetic activation. If heart rate is observed to slow, on the other hand, this could be due to decreased sympathetic activation of the heart via the cardiac nerves, increased parasympathetic input via the vagus nerve, or some combination of the two. With heart rate, as with most ANS functions (pupil diameter, gastric motility, blood pressure), a change in activity in response to stimuli or situations of psychological significance

cannot be unambiguously laid to either sympathetic or parasympathetic activity; it may be due to either one or to a combination of both. Thus, the researcher who wishes an unalloyed measure of sympathetic activity may prefer to monitor EDA, whereas the experimenter who wishes a broader picture of both sympathetic and parasympathetic activity may prefer heart rate (assuming that instrumentation constraints allow only one measure to be recorded). Similarly, if for some reason (perhaps the use of medication with side effects on cholinergic or adrenergic systems) one wishes to monitor a response which is predominately cholinergically mediated at the periphery but which is also influenced by sympathetic activity, then EDA would be the choice.

Another advantage of using SCR is that its occurrence is generally quite discriminable. Thus, on a single presentation of a stimulus, one can determine by quick inspection whether or not an SCR has occurred. In contrast, the presence of a heart rate response to a single stimulus presentation may be difficult to distinguish from ongoing changes in heart rate (HR) that reflect changes in muscle tonus or respiratory sinus arrhythmia. For instance, the triphasic deceleratory-acceleratory-deceleratory HR response that appears in long interstimulus interval classical conditioning or during a long (8.0-sec) reaction time foreperiod (Bohlin & Kjellberg 1979) is often discriminable only when one averages second-by-second HR changes following stimulus presentation over a number of trials, just as one must average over a number of stimulus presentations in order to discriminate the cortical event-related potential (ERP) recorded from the scalp from background electroencephalographic (EEG) activity. This may be disadvantageous for the researcher who has a strong interest in activity occurring on a few individual trials.

In addition to decisions made on the basis of neuroanatomical control and basic response characteristics, an investigator may prefer EDA to other response systems because of the nature of the situation in which the subjects are assessed. Fowles (1988) argued convincingly that HR is influenced primarily by activation of a neurophysiological behavioral *activation* system that is involved in responding during appetitive reward seeking, to conditioned stimuli associated with reward, and during active avoidance. On the other hand, EDA is influenced primarily by activation of a neurophysiological behavioral *inhibition* system that is involved in responding to punishment, to passive avoidance, or to frustrative nonreward. This latter system is viewed as an anxiety system. Thus, if an investigator is studying the reaction of subjects to a situation or to discrete stimuli that elicit anxiety – but in which or to which no active avoidance response can be made – then the electrodermal system should be the physiological system that is most responsive.

For many investigators, an additional advantage of the use of EDA relative to other response systems is that, of all

forms of ANS activity, individual differences in EDA are most reliably associated with psychopathological states. In the next section we will discuss in detail the correlates of some of these stable EDA differences between individuals.

Finally, one should bear in mind that, in comparison with many other psychophysiological measures, EDA is relatively inexpensive to record. After initial purchase of the recording system, expenses for each subject are trivial, involving electrode collars and paste and the occasional replacement of electrodes. Electrical shielding of the room or booth in which the subject sits – which is generally needed for noise-free recording of EEG or event-related potentials – is unnecessary, and the costs of using EDA as a response measure are minuscule compared to those of hemodynamic techniques such as PET scans or functional MRI. Furthermore, the techniques used to record EDA are completely harmless and risk-free, and thus they can be used with young children and in research designs that require repeated testing at short intervals of time.

There are also potential disadvantages to the use of EDA as a dependent measure. First, EDA is a relatively slow-moving response system. As mentioned previously, the latency of the elicited SCR is about 1–3 sec, and tonic shifts in SCL produced by changes in arousal, alertness, and so forth require approximately the same time to occur. Thus, an investigator who is interested in tracking very rapidly occurring processes, or stages within a complex process, will not find EDA useful. Such research questions require response measures (e.g., cortical-evoked potentials or prepulse inhibition of the startle blink) that can discriminate among events or processes occurring at intervals of 100 msec or less. Yet even though the SCR cannot index such rapidly occurring processes as sensory gating or stages of stimulus analysis on a real-time basis, the SCR has been found to be correlated with real-time measures of these processes. For example, Lyytinen, Blomberg, and Näätänen (1992) observed that the parietal P3a was larger when an SCR was elicited by a novel tone than when it was not elicited. Interestingly, the latter investigators found that the mismatch negativity component of the ERP was not related to elicitation of SCRs, unlike the P3a. This pattern of results suggests that the SCR orienting response is elicited by a controlled cognitive process (indexed by P3a) that is preceded by an early automatic discrimination process (indexed by mismatch negativity).

Electrodermal activity has sometimes been criticized as a measure because it is multiply caused: the elicited SCR is not specific to a single type of event or situation (as, for instance, the N400 ERP component appears to be specifically influenced by semantic expectancy; Kutas 1997). However, the multiple influences on EDA may actually be as much an advantage as a disadvantage. As described throughout this chapter, EDA can be used to index a number of processes, including activation, attention, and the task significance or affective intensity of a stimulus. In using EDA

as a response measure, one must simply take care to control experimental conditions – that is, to be sure that one is varying only a single process that may influence EDA at a time. Such experimental control is essential for all attempts to draw clear inferences from results, whether one is recording EDA or, for instance, an electrocortical or hemodynamic measure, given the number of processes that may influence these measures as well.

Thus, like any single response system, EDA has distinct advantages and disadvantages as a response system to monitor. The ideal situation, of course, is one in which the researcher can record more than one response measure. When ANS activity is of primary interest, EDA and heart rate are probably the two most common choices: EDA for its neuroanatomical simplicity, trial-by-trial visibility, and utility as a general arousal/attention indicator; and heart rate for its potential differentiation of more subtle psychological states of interest to the researcher.

Psychosocial Context

In this section, we review in more detail the psychosocial factors that influence EDA results occurring in the three types of paradigms identified earlier: (1) those that involve the presentation of discrete stimuli, (2) those that involve the presentation of chronic stimuli, and (3) those that involve examining the correlates of individual differences in EDA.

EFFECTS OF DISCRETE STIMULI

The stimuli to which the SCR is sensitive are wide and varied; they include stimulus novelty, surprisingness, intensity, emotional content, and significance. It might be argued that, because EDA is sensitive to such a wide variety of stimuli, it is not a clearly interpretable measure of any particular psychological process (Landis 1930). This view is certainly correct in the sense that it is impossible to identify an isolated SCR as an "anxiety" response, or an "anger" response, or an "attentional" response. However, the psychological meaning of an SCR becomes interpretable by taking into account the stimulus condition or experimental paradigm in which the SCR occurred. The better-controlled the experimental paradigm, the more conclusive the interpretation. That is, by having only one aspect of the stimulus change across conditions (e.g., task significance) while eliminating other differences (e.g., stimulus novelty, intensity), one can more accurately infer the psychological process mediating the resultant SCR. Thus, as we will illustrate in the following discussion, the inference of a specific psychophysiological process requires knowledge of both a well-controlled stimulus situation and a carefully measured response.

One discrete stimulus paradigm that relies on the SCR's sensitivity to stimulus significance is the so-called guilty

knowledge test (GKT), a test for detecting deception ("lie detection"). The GKT involves recording SCRs (as well as other physiological responses) while presenting subjects with a series of multiple-choice questions (Lykken, 1959). For example, a suspect in a burglary case might be asked to answer "no" to each of the alternatives given for a question concerning details about the burglary. For each question, the correct alternative would be intermixed among other plausible alternatives. The theory behind the technique is that the correct answer to each question will be more psychologically significant to a guilty subject than will the other alternatives, whereas for the innocent subject all of the alternatives would be of equal significance. Therefore, the guilty subject is expected to respond more consistently to the correct alternatives, whereas the innocent subject is expected to respond randomly (Lykken 1959). Lykken (1981) suggested that guilty subjects can be detected nearly 90% of the time and innocent subjects can be correctly classified nearly 100% of the time with a properly constructed GKT. For a discussion of the differing views regarding the accuracy of various techniques for detecting deception, see Jennings, Ackles, and Coles (1991; see also Chapter 28 of this volume).

Tranel, Fowles, and Damasio (1985) developed another type of discrete stimulus paradigm with which to study the effects of significant facial stimuli on SCRs. In their initial investigations, SCRs were recorded from normal college students while being presented a set of slides depicting faces of famous people (e.g., Ronald Reagan, Bob Hope) and faces of unfamiliar people. A total of 55 slides were presented, including 5 initial buffer items, 42 presentations of the nonsignificant stimuli (unfamiliar faces), and 8 intermixed presentations of the significant stimuli (famous faces). Subjects were instructed simply to sit quietly and look at each slide. The results revealed that the average SCR was much larger to slides of significant faces ($X = 1.26 \mu\text{S}$) than to the nonsignificant faces ($X = 0.19 \mu\text{S}$).

Tranel and Damasio (1985) also employed this paradigm with two prosopagnosic patients. Patients with prosopagnosia lose the ability to consciously recognize faces. One patient had a pervasive syndrome and showed a complete failure to consciously recognize any of the faces of famous people (as well as faces of family members, close friends, or even herself). Despite this syndrome, this patient exhibited more frequent as well as larger SCRs to the significant faces than the nonsignificant faces. Bauer (1984) used a paradigm patterned more closely after the GKT with another prosopagnosic patient and also observed SCR discrimination without verbal discrimination.

Although the GKT of Lykken (1959) appears to be quite adequate to detect concealed information (and hence the guilty person) and the paradigm of Tranel et al. (1985) appears adequate to test for recognition of famous faces, one may question whether either paradigm is sufficient to demonstrate the effect of stimulus significance on SCR.

It may be argued that both paradigms confounded relative novelty with relative stimulus significance. If guilty subjects dichotomize items into relevant and irrelevant categories in the GKT (Ben-Shakhar 1977), then the relevant category is presented less often than the irrelevant category and this relative novelty may contribute to the differential SCRs. Likewise, in the studies using slides of famous faces, the significant category of stimuli was presented less often than the nonsignificant category, and this difference in relative novelty may have contributed to the differential SCRs. The number of presentations of significant stimuli should have been equal to that of nonsignificant stimuli in order to unambiguously demonstrate the effect of stimulus significance on SCRs. As mentioned earlier in this section, close control over stimulus properties (e.g., novelty or significance) is necessary in order to infer the psychological processes eliciting the SCR.

Electrodermal activity is commonly measured using a discrete stimulus paradigm known as "discrimination classical conditioning," which highlights the influence of stimulus significance while controlling for stimulus novelty (Grings & Dawson 1973). For example, Dawson and Biferno (1973) employed a discrimination classical conditioning paradigm in which college student subjects were asked to rate their expectancy of the UCS (unconditioned stimulus; here, a brief electric shock) after each presentation of a CS+ and a CS-; tones of 800 Hz and 1,200 Hz were presented equally often and served (respectively) as the reinforced CS+ and the nonreinforced CS-, counterbalanced across subjects. Thus, for each conditioning trial, the subject's expectancy of shock and the associated SCR was recorded. The results, shown in Figure 6, revealed that subjects tended to respond equally to the reinforced CS+ and to the nonreinforced CS- until they became aware of the contingency between the conditioned stimuli and the shock. There was no evidence of SCR discrimination conditioning prior to the development of awareness; however, once the subject became aware, the CS+ became more significant than the CS- and there was an abrupt increase in the magnitude of the SCRs elicited by the CS+.

The SCR discrimination conditioning data suggest that subjects must be consciously aware of the differential stimulus significance before differential SCRs are elicited, whereas the earlier reviewed findings with prosopagnosic patients suggest that conscious awareness is not necessary for SCR discrimination. This apparent contradiction could perhaps be resolved by the hypothesis that conscious awareness is necessary for the initial learning of stimulus significance but not for the later evocation of SCRs to previously learned significant stimuli (Dawson & Schell 1985). However, we should note that the results obtained from one of the prosopagnosic patients studied by Tranel and Damasio (1985) do not appear consistent with this hypothesis. This patient suffered only from anterograde prosopagnosia in that she failed to consciously

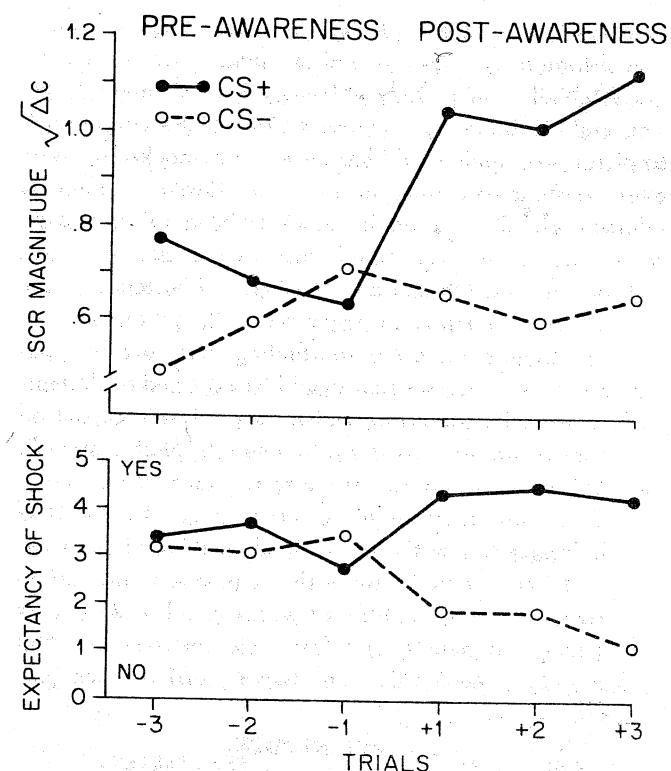


Figure 6. Mean SCR magnitude (top) and mean expectancy of shock (bottom) to the reinforced conditioned stimulus (CS+) and the nonreinforced conditioned stimulus (CS-) on three pre-aware and three post-aware trials. Reprinted with permission from Dawson & Biferno, "Concurrent measurement of awareness and electrodermal classical conditioning," *Journal of Experimental Psychology*, vol. 101, pp. 55-62. Copyright © 1973 by the American Psychological Association.

recognize only new faces experienced since the onset of her prosopagnosia. When this patient was exposed to slides of faces of persons with whom she had contact only since the onset of her illness (physicians, psychologists, etc.), she was unable consciously to recognize these faces yet she still gave more frequent and larger SCRs to these significant faces compared to nonsignificant faces. This provocative finding has essentially been replicated in another patient (Tranel & Damasio 1993), further suggesting that affective associative learning with faces as conditioned stimuli may occur without apparent conscious awareness.

In a related vein, the SCR has been used in the discrimination classical conditioning paradigm to investigate an interesting series of questions concerning "preparedness" and conditioning in normal subjects. The basic idea of preparedness (see Seligman 1970) is that some associations (such as the association between taste and nausea, or perhaps faces and affect) are — because of their survival value for the organism — much more easily and strongly established than others (such as that between an arbitrary tone and a shock). Öhman and his colleagues extended Seligman's concept to human autonomic conditioning, using what have been termed "biologically

prepared," "potentially phobic," or "fear-relevant" conditioned stimuli – pictures of spiders, snakes, and angry faces (see McNally 1987; Öhman 1986, 1992). Öhman, Eriksson, and Olofsson (1975) reported that SCR_s conditioned to pictures of spiders and snakes using a shock UCS were more resistant to extinction (i.e., were slower to diminish following the absence of the shock UCS) than were SCR_s conditioned to neutral stimuli such as pictures of flowers and mushrooms; Öhman and Dimberg (1978) reported the same effect for pictures of angry versus happy faces.

Of particular interest is the finding that cognitive manipulations or processes that would be expected to dramatically reduce the conditioned SCR have a lesser impact on responses conditioned to these potentially phobic stimuli. Such SCR_s, for instance, are more resistant to extinction instructions (information given to the subject that the UCS will no longer follow the CS – Hugdahl 1978; Hugdahl & Öhman 1977). Figure 7 shows the acquisition and extinction trials of groups conditioned with fear-relevant (spiders and snakes; left panels) and fear-irrelevant (triangles and circles; right panels) CSs. The lower panel of each pair

shows the effects of extinction instructions on the SCR. The top two panels exhibit the generally greater resistance to extinction of SCR_s conditioned to fear-relevant CSs. The lower right panel shows the immediate abolition of the conditioned SCR by the extinction instructions when fear-irrelevant CSs are used. The lower left panel, on the other hand, shows the resistance to the extinction instructions of the SCR conditioned to the fear-relevant CSs. Skin conductance responses conditioned to such stimuli are also retained past the point of cognitive extinction (no greater expectancy of the UCS after the CS+ than after the CS-) when cognitive extinction has been created by the presentation of many nonreinforced trials (Schell, Dawson, & Marinkovic 1991), whether extinction trials are given immediately following acquisition or after a delay of several months. Investigators continue to explore the properties of the SCR_s acquired by these stimuli as well as the implications that the unique properties of these stimuli – and the responses conditioned to them – have for theories of emotional learning and clinical phenomena (see e.g. Hugdahl & Johnsen 1993; Öhman et al. 1993).

As mentioned earlier, SCR_s elicited by discrete nonaversive stimuli are generally considered to be part of the orienting response (OR) to novel or significant stimuli. We believe that the data reviewed in this section can be interpreted within this theoretical setting. The task of subjects exposed to the GKT is to deceive or conceal knowledge, and the correct item is more relevant to this task than are incorrect alternative items. Thus, subjects orient more to the task-significant items than to the task-nonsignificant items. Likewise, faces of famous people may be perceived as more significant and attention-demanding than the faces of unfamiliar people, and the signal of an impending shock (CS+) is more significant than the signal of no shock (CS-). Thus, the results observed here are consistent with the notion that the SCR is highly sensitive to stimulus significance, although the reader is reminded of the caveat regarding the confounding of stimulus frequency with stimulus significance in some of these paradigms.

There have been several models proposed to account for the elicitation of autonomic ORs such as the SCR (see Siddle et al. 1983 for a review). For example, an influential information processing model was proposed by Öhman (1979). This model distinguishes between automatic preattentive processing and controlled capacity-limited processing. Autonomic orienting is elicited when the preattentive mechanisms call for additional controlled processing. According to this model, there are two conditions under which this call is made. First, the call is made and the OR is elicited when the preattentive mechanisms fail to identify the incoming stimulus because there is no matching representation in short-term memory. Thus, the OR is sensitive to stimulus novelty. Second, the call is made and the OR is elicited when the preattentive mechanisms recognize the stimulus as significant. Thus, the OR represents

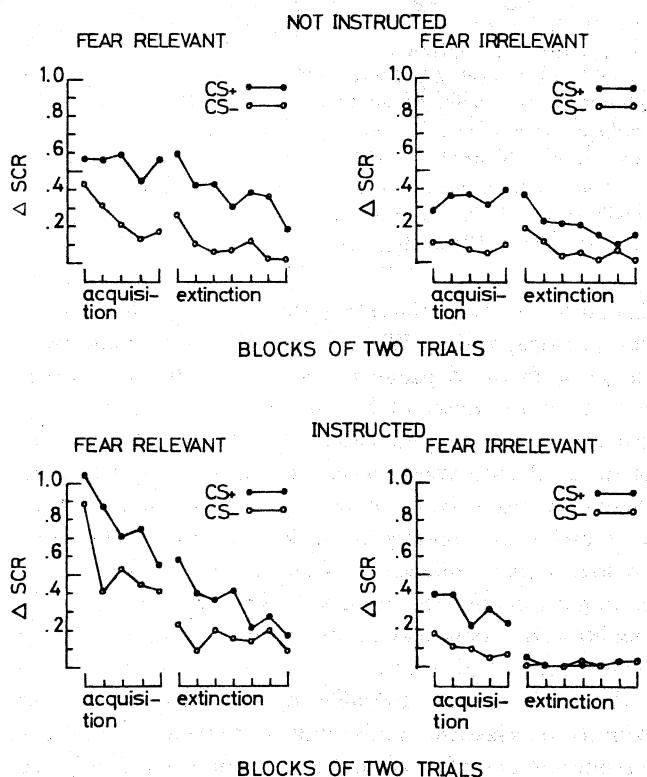


Figure 7. Mean SCR magnitude to the reinforced conditioned stimulus (CS+) and the nonreinforced conditioned stimulus (CS-) during acquisition and extinction for groups conditioned with fear-relevant and fear-irrelevant CSs, groups that either were or were not instructed about the omission of the shock UCS prior to extinction. Reprinted with permission from Hugdahl & Öhman, "Effects of instruction on acquisition and extinction of electrodermal responses to fear-relevant stimuli," *Journal of Experimental Psychology: Human Learning and Memory*, vol. 3, pp. 608-18. Copyright © 1977 by the American Psychological Association.

a transition from automatic to controlled processing based on preliminary preattentive analysis of stimulus novelty and stimulus significance. Others, however, have suggested that the OR occurs when controlled processing resources are actually allocated to the processing of the stimulus, at least where fear-irrelevant stimuli are concerned (Dawson, Filion, & Schell 1989; Öhman 1992).

In conclusion, in this section we have described some of the discrete stimulus paradigms in which EDA is most often measured and has proven to be most useful. Within this description we have emphasized that determining the psychological meaning of any particular SCR is dependent on a well-controlled stimulus situation. In addition, we have described a theoretical model that may be used to account for the SCRs elicited in the paradigms described. Finally, these areas of research examining the SCR to discrete stimuli underscore the point made previously that one advantage of the SCR is that the response can easily be measured on individual presentations of a stimulus. Thus, one may determine whether the response to a "guilty" stimulus in a group of stimuli is greater than to "innocent" stimuli, whether the SCR elicited by a CS+ is greater on the first trial after awareness of the CS-UCS relationship occurs than on the last trial before that awareness occurs, and whether the SCR elicited by a fear-relevant CS+ is greater than the SCR elicited by a CS- on the first trial pair following extinction instructions.

EFFECTS OF CHRONIC STIMULI

We turn now to an examination of the effects of more chronic, long-lasting stimuli or situations as opposed to the brief, discrete stimuli just reviewed. Chronic stimuli might best be thought of as modulating increases and decreases in tonic arousal. Hence, the most useful electrodermal measures in the context of chronic stimuli are SCL and frequency of NS-SCRs, because they can be measured on an ongoing basis over relatively long periods of time.

One type of chronic stimulus situation that will reliably produce increases in electrodermal activity involves the necessity of performing a task. The anticipation and performance of practically any task will increase both SCL and the frequency of NS-SCRs, at least initially. For example, Lacey and colleagues (1963) recorded palmar SCL during rest and during the anticipation and performance of eight different tasks. The tasks ranged from those requiring close attention to external stimuli, such as listening to an irregularly fluctuating loud white noise, to those requiring close attention to internal information processing and rejection of external stimuli, such as solving mental arithmetic problems. The impressive finding for present purposes was that SCL increased in each and every one of the task situations. Typically, SCL increased about $1 \mu\text{S}$ above resting level during anticipation and then increased another 1 or $2 \mu\text{S}$ during performance of the task.

Munro and associates (1987) observed that large increases in SCL and NS-SCR frequency were induced by a different task-significant situation. In this case, college student subjects were tested during a 5-min rest period and then during performance of a continuous performance vigilance task. The task stimuli consisted of a series of digits presented visually at a rapid rate of 1/sec with an exposure duration of 48 msec; the subject's task was to press a button whenever the digit "0" was presented. Both the number of NS-SCRs and SCL increased sharply from the resting levels during this demanding task and then gradually declined as the task continued.

The finding that electrodermal activity is reliably elevated during task performance suggests that tonic EDA can be a useful index of a process related to energy regulation or mobilization. An attentional or information processing interpretation of this finding might be that tasks require an effortful allocation of attentional resources and that this is associated with heightened autonomic activation (Jennings 1986). A different (but not necessarily mutually exclusive) explanation would invoke the concepts of stress and affect rather than attention and effortful allocation of resources. According to this view, laboratory tasks are challenging stressors, and a reliable physiological response to stressors is increased sympathetic activation – particularly EDA arousal.

Social stimulation constitutes another class of chronic stimuli that generally produce increases in EDA arousal. Social situations are ones in which the concepts of stress and affect are most often invoked. For example, early research related the EDA recorded during psychotherapeutic interviews to concepts such as "tension" and "anxiety" on the part of both patient and therapist (Boyd & DiMaschio 1954; Dittes 1957). In one such study, Dittes measured the frequency of NS-SCRs of a patient during 42 hours of psychotherapy. The results of this study indicated that the frequency of NS-SCRs was inversely related to the judged permissiveness of the therapist, and Dittes concluded that EDA reflects "the anxiety of the patient, or his 'mobilization' against any cue threatening punishment by the therapist" (1957, p. 303).

Schwartz and Shapiro (1973) reviewed electrodermal findings in several areas of social psychophysiology up to 1970. The areas most relevant here are those in which EDA was measured during social interactions. These are situations in which there may occur intense cognitive and affective reaction and therefore large changes in EDA and other physiological responses. For example, in one series of social psychophysiological studies conducted since the Schwartz and Shapiro review, EDA was recorded during marital social interactions (Levenson & Gottman 1983, 1985). The researchers measured SCL (in addition to heart rate, pulse transmission time, and somatic activity) in married couples who were discussing conflict-laden problem areas. It was found that couples from distressed

marriages had high "physiological linkage"; that is, there were greater correlations between husbands' and wives' physiological reactions in distressed marriages than those in satisfying marriages during the discussions of problem areas. Moreover, greater physiological arousal, including higher SCL, during the interactions and during baselines was associated with a decline in marital satisfaction over the ensuing three years.

Another series of studies related the effects of stressful social interactions on EDA to the study of relapse among schizophrenia patients. It has been well documented that the emotional attitudes expressed by a family member toward a patient with schizophrenia can be a powerful predictor of later relapse of the patient. That is, patients are at increased risk for relapse if their relatives are critical, hostile, or emotionally over-involved with them at the time of their illness (Brown, Birley, & Wing 1972; Vaughn & Leff 1976; Vaughn et al. 1984). The term "expressed emotion" (EE) is used to designate this continuum of affective attitudes ranging from low EE (less critical) to high EE (more critical) on the part of the relative.

It has been hypothesized that heightened autonomic arousal may be a mediating factor between the continued exposure to a high-EE relative and the increased risk of relapse (Turpin 1983). According to this notion, living with a high-EE family member produces excessive stress and autonomic hyperarousal. Autonomic hyperarousal has been characterized as one of several transient intermediate states that can produce deterioration in the patient's behavior, which in turn can negatively affect people around the patient. Hence, a vicious cycle can be created whereby the increased arousal causes changes in the patient's behavior that have an aggravating effect on the social environment, which serves to increase further autonomic arousal. Unless such a cycle is broken (e.g., by removal from that social environment), it can lead to the return of schizophrenia symptoms and a clinical relapse (Dawson, Nuechterlein, & Liberman 1983; Nuechterlein & Dawson 1984).

One prediction derived from this model is that patients exposed to high-EE relatives should show heightened sympathetic arousal compared to patients exposed to low-EE relatives. The first study to test this prediction obtained rather clear confirmatory results (Tarrier et al. 1979). These investigators measured the EDA of remitted patients living in the community whose relatives' levels of EE had already been determined by Vaughn and Leff (1976). Patients were tested in their homes for 15 minutes without the key relative and for 15 minutes with the key relative present. The frequency of NS-SCR activity of the patients with high-EE relatives and low-EE relatives did not differ when the relative was absent from the testing room, but if the key relative was present then patients with high-EE relatives exhibited higher rates of NS-SCRs than did patients with low-EE relatives. These results indicate that the presence of high-EE and low-EE relatives have differential

effects on EDA, which is consistent with the hypothesis that differential autonomic arousal plays a mediating role in the differential relapse rates of the two patient groups.

In a subsequent investigation, Sturgeon and associates (1984) employed similar testing procedures with acutely ill hospitalized patients. In this case, it was found that patients with high-EE relatives exhibited more NS-SCRs than patients with low-EE relatives, whether or not the key relative was present in the testing room. Sturgeon et al. speculated that this pattern of results may indicate that patients from high-EE homes undergo a more sustained elevation of sympathetic arousal than patients from low-EE homes when they experience an exacerbation of schizophrenic symptoms. Another possibility is that the patients knew that the high-EE relatives were at the hospital and anticipated that the relatives would join them. In either event, these studies demonstrate the powerful effects of social stimulation on EDA and the potential importance and applicability of these effects in the area of psychopathology. More complete reviews of these studies and their implications can be found in Turpin, Tarrier, and Sturgeon (1988).

INDIVIDUAL DIFFERENCES IN EDA

We have discussed the utility of EDA as a dependent variable reflecting situational levels of arousal or activation by and attentiveness or responsiveness to individual stimuli. In this section we consider the EDA as a stable trait of the individual – as an individual difference variable. Individual differences in EDA are reliably associated with behavioral differences and psychopathological states of some importance, and we will examine some of these.

Individual differences in the rate of NS-SCRs and the rate of SCR habituation have been used to define a trait called "electrodermal lability" (Crider 1993; Lacey & Lacey 1958; Mundy-Castle & McKiever 1953). Electrodermal *labiles* are those subjects who show high rates of NS-SCRs and/or slow SCR habituation, whereas electrodermal *stabiles* are those who show few NS-SCRs and/or fast SCR habituation. Electrodermal lability is an individual trait that has been found to be relatively reliable over time, and labiles differ from stabiles with respect to a number of psychophysiological variables, including measures of both electrodermal and cardiovascular responsiveness (Kelsey 1991; Schell et al. 1988). In this section we present the behavioral and psychological differences associated with this individual difference in both the normal and abnormal populations.

Electrodermal lability is a trait that has been of interest in psychological research in part because many investigators have reported that labiles outperform stabiles on tasks that require sustained vigilance. When individuals perform a signal detection task that is sustained over time, deterioration across time in the accurate detection of

targets is frequently observed, a phenomenon referred to as "vigilance decrement" (Davies & Parasuraman 1982). Numerous experimenters have reported that, when vigilance decrement occurs, it is more pronounced among electrodermal stabiles than among labiles. As time on the task goes by, labiles are apparently better able to keep attention focused on the task and to avoid a decline in performance (Crider & Augenbraun 1975; Hastrup 1979; Munro et al. 1987; Vossel & Rossman 1984). For instance, Munro et al. reported that – with a difficult attentional capacity-demanding detection task – stabiles showed a significant decrement over time in the signal detection measure d' , which reflects perceptual sensitivity, whereas labiles did not. Furthermore, the degree of task-induced sympathetic arousal as measured by increases in NS-SCR rate was negatively correlated across subjects with d' decrement.

Researchers investigating such behavioral differences have concluded that electrodermal lability reflects the ability to allocate information processing capacity to target stimuli (Katkin 1975; Lacey & Lacey 1958; Schell et al. 1988). As Katkin (1975, p. 172) concluded, "electrodermal activity is a personality variable that reflects individual differences in higher central processes involved in attending to and processing information." Viewing electrodermal lability in this way suggests that labiles should differ from stabiles in a variety of information processing tasks. Consistent with this view, EDA labile children have been found to generally outperform stabiles on a variety of tasks that require perceptual speed and vigilance (Sakai, Baker, & Dawson 1992).

In addition to the differences between stabiles and labiles in the normal population, reliable abnormalities in electrodermal lability are associated with diagnosable psychopathology. The most common electrodermal abnormality in psychopathological groups is extreme stability in the form of very rapid habituation (or complete absence) of the SCR orienting response, although extreme lability has also been reported in some groups. In the following paragraphs we will summarize EDA abnormalities reported in schizophrenia and depression. A more general discussion of psychophysiological abnormalities in these and other psychopathologies can be found in Chapter 26 of this volume as well as in Zahn (1986).

The most commonly reported EDA abnormality in schizophrenia has been SCR nonresponding and hyporesponding to innocuous tones (see reviews by Bernstein et al. 1982; Dawson & Nuechterlein 1984; Iacono, Ficken, & Beiser 1993; Öhman 1981). In a pathbreaking study on this topic, Gruzelier and Venables (1972) presented a series of nonsignal tones to a large group of schizophrenia patients and to normal controls. Whereas all of the normal controls initially responded to the stimuli and then showed habituation of the SCR, the schizophrenia group showed a bimodal distribution at both extremes of habituation: 54% of the schizophrenia patients failed to give even one SCR, while 42% not only responded but failed to meet

the habituation criterion. Gruzelier and Venables (1972) referred to the stable group as "nonresponders" and to the labile group as "responders."

The high proportion of electrodermal nonresponders in schizophrenia is a very reliable finding. For example, Bernstein et al. (1982) examined a series of fourteen related studies in which samples of American, British, and German schizophrenia patients and normal controls were studied using a common methodology and response scoring criteria. Their consistent finding was that approximately 50% of schizophrenia patients were nonresponders, compared with only 5%–10% of controls. (More recent data, reported and reviewed by Venables & Mitchell 1996, suggest the percentage of SCR nonresponders among normal controls may be close to 25%.) A minority of the studies also reported the existence of a responder subgroup of patients showing slower than normal habituation, but the majority finding is normal rates of habituation in the responder subgroup. The responder–nonresponder distinction is a potentially important one because it may be useful in identifying useful subgroups within the heterogeneous disorder(s) of schizophrenia. For example, nonresponders and responders have been reported to show different symptomatology, with responders generally displaying more symptoms such as excitement, anxiety, manic behavior, and belligerence; nonresponders tend to show more emotional withdrawal and conceptual disorganization (Bernstein et al. 1982; Straube 1979). Furthermore, SCR hyporesponsivity has been related to a more severe form of illness (Katsanis & Iacono 1994), lower overall and regionally specific brain metabolism (Hazlett et al. 1993), and poor premorbid adjustment (Öhman et al. 1989), although SCR hyperactivity also has been associated with higher levels of symptomatology in a group of recent-onset schizophrenia outpatients (Dawson et al. 1992b).

Findings in this area may become clearer by taking into consideration the symptomatic state of the patients at the time of testing. Higher than normal SCL and rates of NS-SCRs have been found in patients while in a psychotic state but not in the same patients while in a symptomatically remitted state (Dawson et al. 1994); see Figure 8. Phasic SCR hyporesponsiveness, on the other hand, was observed in both the psychotic and the remitted state when responsiveness was corrected for overall tonic activation levels. These findings suggest that tonic electrodermal hyperarousal is a state-sensitive symptomatic episode indicator, whereas SCR hyporesponsivity may be an enduring traitlike indicator when corrected for existing arousal level.

Tonic electrodermal hyperactivity may be not only an episode indicator but also an early precursor of symptomatic exacerbation or relapse in schizophrenia. Hazlett and associates (1997b) found elevated EDA in four of five symptomatically completely remitted patients in the week or so prior to a psychotic exacerbation or relapse compared with the levels shown by these same patients in non-prerelapse

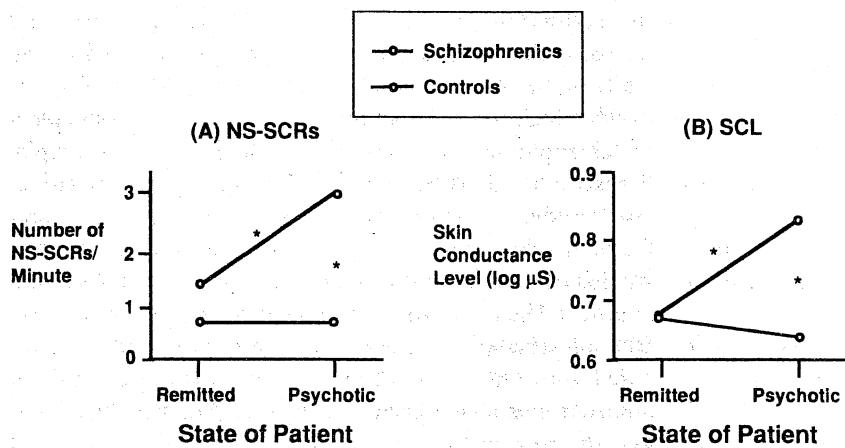


Figure 8. Mean number of nonspecific SCRs per minute (left) and mean SCL (right) obtained from normal controls and patients with schizophrenia when the patients were in remitted and psychotic states. Reprinted with permission from Dawson, Nuechterlein, Schell, Gitlin, & Ventura, "Autonomic abnormalities in schizophrenia: State or trait indicators?" *Archives of General Psychiatry*, vol. 51, pp. 813-24. Copyrighted 1994, American Medical Association.

periods. These findings are consistent with a theoretical model which hypothesizes that sympathetic activation is associated with a "transient intermediate state" that precedes psychotic episodes in vulnerable individuals (Nuechterlein & Dawson 1984). According to this theoretical model, not all such intermediate states will be followed by psychotic exacerbation or relapse. Rather, these states constitute periods of heightened vulnerability and an increased risk of relapse, with the actual occurrence of relapses or exacerbation being influenced by environmental stressors.

Phasic and tonic electrodermal hypoactivity also have been reported for depressed patients (Sponheim, Allen, & Iacono 1995; see also Chapter 26). For example, Mirkin and Coppen (1980) found a higher than normal incidence of SCR nonresponding (67% vs. 13%) in unmedicated depressive inpatients than in controls. Numerous other investigators found EDA hypoactivity in the form of low levels of SCL and/or small SCRs to different classes of stimuli (Dawson, Schell, & Catania 1977; Iacono et al. 1983; Lader & Noble 1975; Ward, Doerr, & Storrie 1983). Although SCR nonresponders have not been treated as a distinct subgroup in depression (as they have in schizophrenia), it should be noted that EDA hypoactivity in terms of both tonic SCL and phasic SCR responsivity has been reported to be more prominent in psychotic than neurotic depression (Byrne 1975); endogenous than nonendogenous depression (Mirkin & Coppen 1980); and retarded than agitated depression (Lader & Wing 1969). Such hypoactivity has also been associated with overall greater depressive symptomatology (Dawson et al. 1977).

The phasic SCR orienting response deficit observed in both schizophrenia and depression suggests the possibility of a common information processing deficit. Con-

sistent with this possibility, Hazlett and colleagues (1997a) found secondary reaction time abnormalities during a test of SCR orienting in recent-onset schizophrenia patients. The secondary reaction time measure indicated a delay in the allocation of attentional resources in the patients. This abnormality was particularly pronounced in the SCR hyporesponders, consistent with the notion that the SCR deficit is related to an underlying attentional dysfunction in schizophrenia.

However, several lines of evidence suggest that the SCR impairments observed in schizophrenia and depression may reflect different underlying dysfunctions. *First*, Bernstein and his associates (1988, 1995) demonstrated that, although SCR nonresponding is more frequent in both schizophrenia patients and depressive patients than in normal controls, only schizophrenia patients displayed excessive nonresponding with the finger pulse amplitude component of the orienting response. These findings suggest that schizophrenia is associated with a central orienting deficiency, whereas depression may involve a deficit more specific to electrodermal activity (e.g., peripheral cholinergic hypoactivity). *Second*, Bernstein et al. (1988) found that increasing the stimulus significance of the "innocuous" tones served to normalize the elicited SCR on initial trials in schizophrenia but not depression. Although signal stimuli do not always normalize elicited SCRs in schizophrenia (Dawson et al. 1992a; Iacono et al. 1993), these results do suggest that patients with schizophrenia have an orienting dysfunction that can be normalized (at least temporarily), whereas depressed patients have chronic peripheral (cholinergic?) deficits in EDA. *Third*, Lencz, Rainey, and Sheard (1996) utilized cluster analysis of MRI and SCR data in patients with schizophrenia, affective disorders (mainly major depression), and controls. They found that SCR hypoactivity in schizophrenia was heterogeneous but tended to cluster with reduced prefrontal area and normal lateral ventricles, whereas SCR hypoactivity in affective disorder patients clustered more with normal prefrontal area and enlarged ventricles. Thus, schizophrenia patients and depressed patients may be SCR hypoactive for different reasons.

There are important lessons for all EDA researchers in this series of studies on psychopathology. The presence or absence of an SCR cannot be interpreted in isolation. In order to reasonably interpret the meaning of the presence or absence of an SCR, one needs to know the stimulus conditions eliciting the response (e.g., signal or nonsignal stimuli), and it is often useful to obtain other behavioral responses (e.g., secondary reaction time), psychophysiological responses (e.g., finger pulse volume), or brain imaging measures (e.g., MRI) in conjunction with the SCR.

Epilogue

Electrodermal activity has proven to be a useful psychophysiological tool with wide applicability. Social and behavioral scientists have found (i) that tonic EDA is useful for investigating general states of arousal and alertness and (ii) that the phasic SCR is useful for studying the multifaceted attentional process and stimulus significance, as well as individual differences that may be related to behavioral differences or psychopathological states. We believe that future research will continue to use EDA in a variety of situations and stimulus conditions to test these theoretical concepts.

Another important direction for future research involves sharpening the "inferential tool" characteristics of EDA itself. That is, basic research is needed to address the specific conditions under which specific EDA components reflect specific psychological and physiological processes and mechanisms. For example, under what stimulus conditions does the SCR amplitude component of the orienting response reflect automatic preattentive cognitive processes versus controlled cognitive processes? Likewise, under what test situations do the tonic and phasic EDA components reflect the different brain systems shown in Figure 2? We certainly hope that the expanding cognitive paradigms and neuroimaging techniques will be applied to elucidate these issues in both normal and abnormal populations, making EDA an even more useful psychophysiological tool.

NOTE

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REFERENCES

- Bauer, R. M. (1984). Autonomic recognition of names and faces in prosopagnosia: A neuropsychological application of the guilty knowledge test. *Neuropsychologia*, 22, 457-69.
- Ben-Shakhar, G. (1977). A further study of the dichotomization theory of detection of information. *Psychophysiology*, 14, 408-13.
- Ben-Shakhar, G. (1985). Standardization within individuals: Simple method to neutralize individual differences in skin conductance. *Psychophysiology*, 22, 292-9.
- Bernstein, A., Frith, C., Gruzelier, J., Patterson, T., Straube, E., Venables, P., & Zahn, T. (1982). An analysis of the skin conductance orienting response in samples of American, British, and German schizophrenics. *Biological Psychology*, 14, 155-211.
- Bernstein, A. S., Riedel, J. A., Graae, F., Seidman, D., Steele, H., Connolly, J., & Lubowsky, J. (1988). Schizophrenia is associated with altered orienting activity; depression with electrodermal (cholinergic?) deficit and normal orienting response. *Journal of Abnormal Psychology*, 97, 3-12.
- Bernstein, A. S., Schnur, D. B., Bernstein, P., Yeager, A., Wrable, J., & Smith, S. (1995). Differing patterns of electrodermal and finger pulse responsivity in schizophrenia and depression. *Psychological Medicine*, 25, 51-62.
- Bernstein, A. S., Taylor, K. W., Starkey, P., Juni, S., Lubowsky, J., & Paley, H. (1981). Bilateral skin conductance, finger pulse volume, and EEG orienting response to tones of differing intensities in chronic schizophrenics and controls. *Journal of Nervous and Mental Disease*, 169, 513-28.
- Bloch, V. (1993). On the centennial of the discovery of electrodermal activity. In Roy et al. (1993a), pp. 1-6.
- Bohlin, G. (1976). Delayed habituation of the electrodermal orienting response as a function of increased level of arousal. *Psychophysiology*, 13, 345-51.
- Bohlin, G., & Kjellberg, A. (1979). Orienting activity: two-stimulus paradigms as reflected in heart rate. In H. D. Kimmel, E. H. van Olst, & J. F. Orlebeke (Eds.), *The Orienting Reflex in Humans*, pp. 169-98. Hillsdale, NJ: Erlbaum.
- Boucsein, W. (1992). *Electrodermal Activity*. New York: Plenum.
- Boyd, R. W., & DiMascio, A. (1954). Social behavior and autonomic physiology (A sociophysiological study). *Journal of Nervous and Mental Disease*, 120, 207-12.
- Brown, G., Birley, J. L. T., & Wing, J. K. (1972). Influence of family life on the course of schizophrenia. *British Journal of Psychiatry*, 121, 241-8.
- Bundy, R. S., & Fitzgerald, H. E. (1975). Stimulus specificity of electrodermal recovery time: An examination and reinterpretation of the evidence. *Psychophysiology*, 12, 406-11.
- Byrne, D. G. (1975). A psychophysiological distinction between types of depressive states. *Australian and New Zealand Journal of Psychiatry*, 9, 181-5.
- Crider, A. (1993). Electrodermal response lability-stability: Individual difference correlates. In Roy et al. (1993a), pp. 173-86.
- Crider, A., & Augenbraun, C. (1975). Auditory vigilance correlates of electrodermal response habituation speed. *Psychophysiology*, 12, 36-40.
- Darrow, C. W. (1927). Sensory, secretory, and electrical changes in the skin following bodily excitation. *Journal of Experimental Psychology*, 10, 197-226.
- Darrow, C. W. (1937). Neural mechanisms controlling the palmar galvanic skin reflex and palmar sweating. *Archives of Neurology and Psychiatry*, 37, 641-63.
- Davies, D. R., & Parasuraman, R. (1982). *The Psychology of Vigilance*. London: Academic Press.
- Dawson, M. E., & Biferno, M. A. (1973). Concurrent measurement of awareness and electrodermal classical conditioning. *Journal of Experimental Psychology*, 101, 55-62.
- Dawson, M. E., Filion, D. L., & Schell, A. M. (1989). Is elicitation of the autonomic orienting response associated with allocation of processing resources? *Psychophysiology*, 26, 560-72.
- Dawson, M. E., & Nuechterlein, K. H. (1984). Psychophysiological dysfunctions in the developmental course of schizophrenic disorders. *Schizophrenia Bulletin*, 10, 204-32.
- Dawson, M. E., Nuechterlein, K. H., & Liberman, R. P. (1983). Relapse in schizophrenic disorders: Possible contributing factors and implications for behavior therapy. In M. Rosenbaum, C. M. Franks, & Y. Jaffe (Eds.), *Perspectives on Behavior Therapy in the Eighties*, pp. 265-86. New York: Springer.

- Dawson, M. E., Nuechterlein, K. H., & Schell, A. M. (1992a). Electrodermal anomalies in recent-onset schizophrenia: Relationships to symptoms and prognosis. *Schizophrenia Bulletin*, 18, 295-311.
- Dawson, M. E., Nuechterlein, K. H., Schell, A. M., Gitlin, M., & Ventura, J. (1994). Autonomic abnormalities in schizophrenia: State or trait indicators? *Archives of General Psychiatry*, 51, 813-24.
- Dawson, M. E., Nuechterlein, K. H., Schell, A. M., & Mintz, J. (1992b). Concurrent and predictive electrodermal correlates of symptomatology in recent-onset schizophrenic patients. *Journal of Abnormal Psychology*, 101, 153-64.
- Dawson, M. E., & Schell, A. M. (1985). Information processing and human autonomic classical conditioning. In P. K. Ackles, J. R. Jennings, & M. G. H. Coles (Eds.), *Advances in Psychophysiology*, vol. 1, pp. 89-165. Greenwich, CT: JAI.
- Dawson, M. E., Schell, A. M., & Catania, J. J. (1977). Autonomic correlates of depression and clinical improvement following electroconvulsive shock therapy. *Psychophysiology*, 14, 569-78.
- Dittes, J. E. (1957). Galvanic skin response as a measure of patient's reaction to therapist's permissiveness. *Journal of Abnormal and Social Psychology*, 55, 295-303.
- Edelberg, R. (1967). Electrical properties of the skin. In C. C. Brown (Ed.), *Methods in Psychophysiology*, pp. 1-53. Baltimore: Williams & Wilkins.
- Edelberg, R. (1972a). Electrical activity of the skin: Its measurement and uses in psychophysiology. In N. S. Greenfield & R. A. Sternbach (Eds.), *Handbook of Psychophysiology*, pp. 367-418. New York: Holt.
- Edelberg, R. (1972b). Electrodermal recovery rate, goal-orientation, and aversion. *Psychophysiology*, 9, 512-20.
- Edelberg, R. (1973). Mechanisms of electrodermal adaptations for locomotion, manipulation, or defense. *Progress in Physiological Psychology*, 5, 155-209.
- Edelberg, R. (1993). Electrodermal mechanisms: A critique of the two-effector hypothesis and a proposed replacement. In Roy et al. (1993a), pp. 7-29.
- Engel, B. T. (1960). Stimulus-response and individual-response specificity. *Archives of General Psychiatry*, 2, 305-13.
- Féré, C. (1888). Note on changes in electrical resistance under the effect of sensory stimulation and emotion. *Comptes Rendus des Séances de la Société de Biologie* (Ser. 9), 5, 217-19.
- Fowles, D. C. (1974). Mechanisms of electrodermal activity. In R. F. Thompson & M. M. Patterson (Eds.), *Methods in Physiological Psychology, Part C: Receptor and Effector Processes*, pp. 231-71. New York: Academic Press.
- Fowles, D. C. (1986). The eccrine system and electrodermal activity. In M. G. H. Coles, E. Donchin, & S. W. Porges (Eds.), *Psychophysiology: Systems, Processes, and Applications*, pp. 51-96. New York: Guilford.
- Fowles, D. C. (1988). Psychophysiology and psychopathology: A motivational approach. *Psychophysiology*, 25, 373-91.
- Fowles, D., Christie, M. J., Edelberg, R., Grings, W. W., Lykken, D. T., & Venables, P. H. (1981). Publication recommendations for electrodermal measurements. *Psychophysiology*, 18, 232-9.
- Fredrikson, M., Annas, P., Georgiades, A., Hursi, T., & Tesman, Z. (1993). Internal consistency and temporal stability of classically conditioned skin conductance responses. *Biological Psychology*, 35, 153-63.
- Freedman, L. W., Scerbo, A. S., Dawson, M. E., Raine, A., McClure, W. O., & Venables, P. H. (1994). The relationship of sweat gland count to electrodermal activity. *Psychophysiology*, 31, 196-200.
- Freixa i Baqué, E. (1982). Reliability of electrodermal measures: A compilation. *Biological Psychology*, 14, 219-29.
- Graham, F. K., & Clifton, R. K. (1966). Heart rate change as a component of the orienting response. *Psychological Bulletin*, 65, 305-20.
- Grey, S. J., & Smith, B. L. (1984). A comparison between commercially available electrode gels and purpose-made gel, in the measurement of electrodermal activity. *Psychophysiology*, 21, 551-7.
- Grings, W. W. (1974). Recording of electrodermal phenomena. In R. F. Thompson & M. M. Patterson (Eds.), *Bioelectric Recording Technique, Part C: Receptor and Effector Processes*, pp. 273-96. New York: Academic Press.
- Grings, W. W., & Dawson, M. E. (1973). Complex variables in conditioning. In Prokasy & Raskin, pp. 203-54.
- Grings, W. W., & Dawson, M. E. (1978). *Emotions and Bodily Responses: A Psychophysiological Approach*. New York: Academic Press.
- Grings, W. W., & Lockhart, R. A. (1965). Problems of magnitude measurement with multiple GSRs. *Psychological Reports*, 17, 979-82.
- Grings, W. W., & Schell, A. M. (1969). Magnitude of electrodermal response to a standard stimulus as a function of intensity and proximity of a prior stimulus. *Journal of Comparative and Physiological Psychology*, 67, 77-82.
- Gruzelier, J. H., & Venables, P. H. (1972). Skin conductance orienting activity in a heterogeneous sample of schizophrenics: Possible evidence of limbic dysfunction. *Journal of Nervous and Mental Disease*, 155, 277-87.
- Hassett, J. (1978). *A Primer of Psychophysiology*. San Francisco: Freeman.
- Hastrup, J. L. (1979). Effects of electrodermal lability and introversion on vigilance decrement. *Psychophysiology*, 16, 302-10.
- Hazlett, E. A., Dawson, M. E., Buchsbaum, M. S., & Nuechterlein, K. H. (1993). Reduced regional brain glucose metabolism assessed by PET in electrodermal nonresponder schizophrenics: A pilot study. *Journal of Abnormal Psychology*, 102, 39-46.
- Hazlett, E. A., Dawson, M. E., Filion, D. L., Schell, A. M., & Nuechterlein, K. H. (1997a). Autonomic orienting and the allocation of processing resources in schizophrenia patients and putatively at-risk individuals. *Journal of Abnormal Psychology*, 106, 171-81.
- Hazlett, H., Dawson, M. E., Schell, A. M., & Nuechterlein, K. H. (1997b). Electrodermal activity as a prodromal sign in schizophrenia. *Biological Psychiatry*, 41, 111-13.
- Hugdahl, K. (1978). Electrodermal conditioning to potentially phobic stimuli: Effects of instructed extinction. *Behavior Research and Therapy*, 16, 315-21.
- Hugdahl, K. (1984). Hemispheric asymmetry and bilateral electrodermal recordings: A review of the evidence. *Psychophysiology*, 21, 371-93.
- Hugdahl, K. (1995). *Psychophysiology: The Mind-Body Perspective*. Cambridge, MA: Harvard University Press.
- Hugdahl, K., & Johnsen, B. H. (1993). Brain asymmetry and autonomic conditioning: Skin conductance responses. In Roy et al. (1993a), pp. 271-87.

- Hugdahl, K., & Öhman, A. (1977). Effects of instruction on acquisition and extinction of electrodermal responses to fear-relevant stimuli. *Journal of Experimental Psychology: Human Learning and Memory*, 3, 608-18.
- Humphreys, L. G. (1943). Measures of strength of conditioned eyelid responses. *Journal of General Psychology*, 29, 101-11.
- Iacono, W. G., Ficken, J. W., & Beiser, M. (1993). Electrodermal nonresponding in first-episode psychosis as a function of stimulus significance. In Roy et al. (1993a), pp. 239-56.
- Iacono, W. G., Lykken, D. T., Peloquin, L. T., Lumry, A. E., Valentine, R. H., & Tuoson, V. B. (1983). Electrodermal activity in euthymic unipolar and bipolar affective disorders. *Archives of General Psychiatry*, 40, 557-65.
- Jakubovic, H. R., & Ackerman, A. B. (1985). Structure and function of the skin, Section I: Development, morphology, and physiology. In S. L. Moschella & H. J. Hurley (Eds.), *Dermatology*, vol. 1, pp. 1-74. Philadelphia: Saunders.
- Jennings, J. R. (1986). Bodily changes during attending. In M. G. H. Coles, E. Donchin, & S. W. Porges (Eds.), *Psychophysiology: Systems, Processes, and Applications*, pp. 268-89. New York: Guilford.
- Jennings, J. R., Ackles, P. K., & Coles, M. G. K. (1991). *Advances in Psychophysiology*, vol. 4. London: Jessica Kingsley.
- Katkin, E. S. (1975). Electrodermal lability: A psychophysiological analysis of individual differences in response to stress. In I. G. Sarason & C. D. Spielberger (Eds.), *Stress and Anxiety*, vol. 2, pp. 141-76. Washington, DC: Aldine.
- Katsanis, J., & Iacono, W. G. (1994). Electrodermal activity and clinical status in chronic schizophrenia. *Journal of Abnormal Psychology*, 103, 777-83.
- Kelsey, R. M. (1991). Electrodermal lability and myocardial reactivity to stress. *Psychophysiology*, 28, 619-31.
- Kutas, M. (1997). Views on how the electrical activity that the brain generates reflects the functions of different language structures. *Psychophysiology*, 34, 383-98.
- Lacey, J. I., Kagan, J., Lacey, B. C., & Moss, H. A. (1963). The visceral level: Situational determinants and behavioral correlates of autonomic response patterns. In P. H. Knapp (Ed.), *Expression of the Emotions in Man*, pp. 161-96. New York: International Universities Press.
- Lacey, J. I., & Lacey, B. C. (1958). Verification and extension of the principle of autonomic response-stereotypy. *American Journal of Psychology*, 71, 50-73.
- Lader, M., & Noble, P. (1975). The affective disorders. In P. H. Venables and M. J. Christie (Eds.), *Research in Psychophysiology*, pp. 258-81. New York: Wiley.
- Lader, M. H., & Wing, L. (1969). Physiological measures in agitated and retarded depressed patients. *Journal of Psychiatric Research*, 7, 89-100.
- Landis, C. (1930). Psychology of the psychogalvanic reflex. *Psychological Review*, 37, 381-98.
- Lencz, T., Raine, A., & Sheard, C. (1996). Neuroanatomical bases of electrodermal hypo-responding: A cluster analytic study. *International Journal of Psychophysiology*, 22, 141-53.
- Levenson, R. W., & Gottman, J. M. (1983). Marital interaction: Physiological linkage and affective exchange. *Journal of Personality and Social Psychology*, 45, 587-97.
- Levenson, R. W., & Gottman, J. M. (1985). Physiological and affective predictors of change in relationships satisfaction. *Journal of Personality and Social Psychology*, 49, 85-94.
- Levinson, D. F., Edelberg, R., & Bridger, W. H. (1984). The orienting response in schizophrenia: Proposed resolution of a controversy. *Biological Psychiatry*, 19, 489-507.
- Lim, C. L., Rennie, C., Barry, R. J., Bahramali, H., Lazzaro, I., Manor, B., & Gordon, E. (1997). Decomposing skin conductance into tonic phasic components. *International Journal of Psychophysiology*, 25, 97-109.
- Lockhart, R. A., & Lieberman, W. (1979). Information content of the electrodermal orienting response. In H. D. Kimmel, E. H. van Olst, & J. F. Orlebeke (Eds.), *The Orienting Reflex in Humans*, pp. 685-700. Hillsdale, NJ: Erlbaum.
- Lykken, D. T. (1959). The GSR in the detection of guilt. *Journal of Applied Psychology*, 43, 383-8.
- Lykken, D. T. (1981). *A Tremor in the Blood*. New York: McGraw-Hill.
- Lykken, D. T., Iacono, W. G., Haroian, K., McGue, M., & Bouchar, T. J. (1988). Habituation of the skin conductance response to strong stimuli: A twin study. *Psychophysiology*, 25, 4-15.
- Lykken, D. T., Rose, R. J., Luther, B., & Maley, M. (1966). Correcting psychophysiological measures for individual differences in range. *Psychological Bulletin*, 66, 481-4.
- Lykken, D. T., & Venables, P. H. (1971). Direct measurement of skin conductance: A proposal for standardization. *Psychophysiology*, 8, 656-72.
- Lyytinen, H., Blomberg, A., & Näätänen, R. (1992). Event-related potentials and autonomic responses to a change in unattended auditory stimuli. *Psychophysiology*, 29, 523-34.
- Mangina, C. A., & Beuzeron-Mangina, J. H. (1996). Direct electrical stimulation of specific human brain structures and bilateral electrodermal activity. *International Journal of Psychophysiology*, 22, 1-8.
- McNally, R. J. (1987). Preparedness and phobias: A review. *Psychological Bulletin*, 23, 283-303.
- Mirkin, A. M., & Coppen, A. (1980). Electrodermal activity in depression: Clinical and biochemical correlates. *British Journal of Psychiatry*, 137, 93-7.
- Montague, J. D. (1963). Habituation of the psycho-galvanic reflex during serial tests. *Journal of Psychosomatic Research*, 7, 199-214.
- Mundy-Castle, A. C., & McKiever, B. L. (1953). The psychophysiological significance of the galvanic skin response. *Journal of Experimental Psychology*, 46, 15-24.
- Munro, L. L., Dawson, M. E., Schell, A. M., & Sakai, L. M. (1987). Electrodermal lability and rapid performance decrement in a degraded stimulus continuous performance task. *Journal of Psychophysiology*, 1, 249-57.
- Nuechterlein, K. H., & Dawson, M. E. (1984). A heuristic vulnerability/stress model of schizophrenic episodes. *Schizophrenia Bulletin*, 10, 300-12.
- Neumann, E., & Blanton, R. (1970). The early history of electrodermal research. *Psychophysiology*, 6, 453-75.
- Öhman, A. (1979). The orienting response, attention and learning: An information processing perspective. In H. D. Kimmel, E. H. Van Olst, & J. F. Orlebeke (Eds.), *The Orienting Reflex in Humans*. Hillsdale, NJ: Erlbaum.
- Öhman, A. (1981). Electrodermal activity and vulnerability to schizophrenia: A review. *Biological Psychology*, 12, 87-145.
- Öhman, A. (1986). Face the beast and fear the face: Animal and social fears as prototypes for evolutionary analyses of emotion. *Psychophysiology*, 23, 123-45.

- Öhman, A. (1992). Orienting and attention: Preferred preattentive processing of potentially phobic stimuli. In B. A. Campbell, H. Hayne, & R. Richardson (Eds.), *Attention and Information Processing in Infants and Adults: Perspectives from Human and Animal Research*, pp. 263-95. Hillsdale, NJ: Erlbaum.
- Öhman, A., & Dimberg, U. (1978). Facial expressions as conditioned stimuli for electrodermal responses: A case of "preparedness"? *Journal of Personality and Social Psychology*, 36, 1251-8.
- Öhman, A., Eriksson, A., & Olofsson, C. (1975). One-trial learning and superior resistance to extinction of autonomic responses conditioned to potentially phobic stimuli. *Journal of Comparative and Physiological Psychology*, 88, 619-27.
- Öhman, A., Esteves, F., Flykt, A., & Soares, J. F. (1993). Gateways to consciousness: Emotion, attention, and electrodermal activity. In Roy et al. (1993a), pp. 137-57.
- Öhman, A., Öhlund, L. S., Alm, T., Wieselgren, I. M., Istm, K., & Lindstrom, L. H. (1989). Electrodermal nonresponding, premorbid adjustment, and symptomatology as predictors of long-term social functioning in schizophrenics. *Journal of Abnormal Psychology*, 98, 426-35.
- Porges, S. W., & Coles, M. G. H. (Eds.) (1976). *Psychophysiology*. Stroudsburg, PA: Dowden, Hutchinson, & Ross.
- Prokasy, W. F. (1974). SCORIT: A computer subroutine for scoring electrodermal responses. *Behavior Research Methods and Instrumentation*, 7, 49-52.
- Prokasy, W. F., & Kumpfer, K. L. (1973). Classical conditioning. In Prokasy & Raskin, pp. 157-202.
- Prokasy, W. F., & Raskin, D. C. (Eds.). (1973). *Electrodermal Activity in Psychological Research*. New York: Academic Press.
- Raine, A., & Lencz, T. (1993). Brain imaging research on electrodermal activity in humans. In Roy et al. (1993a), pp. 115-35.
- Robertshaw, D. (1983). Apocrine sweat glands. In L. A. Goldsmith (Ed.), *Biochemistry and Physiology of the Skin*, pp. 642-53. New York: Oxford University Press.
- Roy, J. C., Boucsein, W., Fowles, D. C., & Gruzelier, J. H. (Eds.) (1993a). *Progress in Electrodermal Research*. New York: Plenum.
- Roy, J. C., Sequeira, H., & Delerm, B. (1993b). Neural control of electrodermal activity: Spinal and reticular mechanisms. In Roy et al. (1993a), pp. 73-92.
- Sakai, M. L., Baker, L. A., & Dawson, M. E. (1992). Electrodermal lability: Individual differences affecting perceptual speed and vigilance performance in 9 to 16 year-old children. *Psychophysiology*, 29, 207-17.
- Scerbo, A., Freedman, L. W., Raine, A., Dawson, M. E., & Venables, P. H. (1992). A major effect of recording site on measurement of electrodermal activity. *Psychophysiology*, 29, 241-6.
- Schell, A. M., Dawson, M. E., & Filion, D. L. (1988). Psychophysiology correlates of electrodermal lability. *Psychophysiology*, 25, 619-32.
- Schell, A. M., Dawson, M. E., & Marinkovic, K. (1991). Effects of potentially phobic conditioned stimuli on retention, reconditioning, and extinction of the conditioned skin conductance response. *Psychophysiology*, 28, 140-53.
- Schwartz, G. E., & Shapiro, D. (1973). Social psychophysiology. In Prokasy & Raskin, pp. 377-416.
- Seligman, M. E. P. (1970). On the generality of the laws of learning. *Psychological Review*, 77, 307-21.
- Sequeira, H., & Roy, J. C. (1993). Cortical and hypothalamo-limbic control of electrodermal responses. In Roy et al. (1993a), pp. 93-114.
- Shields, S. A., MacDowell, K. A., Fairchild, S. B., & Campbell, M. L. (1987). Is mediation of sweating cholinergic, adrenergic, or both? A comment on the literature. *Psychophysiology*, 24, 312-19.
- Siddle, D. (1991). Orienting, habituation, and resource allocation: An associative analysis. *Psychophysiology*, 28, 245-59.
- Siddle, D., Stephenson, D., & Spinks, J. A. (1983). Elicitation and habituation of the orienting response. In D. Siddle (Ed.), *Orienting and Habituation: Perspectives in Human Research*, pp. 109-82. Chichester, U.K.: Wiley.
- Sokolov, E. N. (1963). *Perception and the Conditioned Reflex*. New York: Macmillan.
- Sponheim, S. R., Allen, J. J., & Iacono, W. G. (1995). Selected psychophysiological measures in depression: The significance of electrodermal activity, electroencephalographic asymmetries, and contingent negative variation to behavioral and neurobiological aspects of depression. In G. A. Miller (Ed.), *The Behavioral High-Risk Paradigm in Psychopathology*, pp. 222-49. New York: Springer.
- Straube, E. R. (1979). On the meaning of electrodermal nonresponding in schizophrenia. *Journal of Nervous and Mental Disease*, 167, 601-11.
- Strayer, D. L., & Williams, W. C. (1982). SCORIT 1980. Paper presented at the annual meeting of the Society for Psychophysiological Research (Washington, DC).
- Sturgeon, D., Turpin, G., Kuipers, L., Berkowitz, R., & Leff, J. (1984). Psychophysiological responses of schizophrenic patients to high and low expressed emotion relatives: A follow-up study. *British Journal of Psychiatry*, 145, 62-9.
- Tarchanoff, J. (1890). Galvanic phenomena in the human skin during stimulation of the sensory organs and during various forms of mental activity. *Pflügers Archiv für die gesamte Physiologie des Menschen und der Tiere*, 46, 46-55.
- Tarrier, N., Vaughn, C., Lader, M. H., & Leff, J. P. (1979). Bodily reactions to people and events in schizophrenics. *Archives of General Psychiatry*, 36, 311-15.
- Tranel, D., & Damasio, A. R. (1985). Knowledge without awareness: An autonomic index of facial recognition by prosopagnosics. *Science*, 228, 1453-4.
- Tranel, D., & Damasio, A. R. (1993). The covert learning of affective valence does not require structures in hippocampal system or amygdala. *Journal of Cognitive Neuroscience*, 5, 79-88.
- Tranel, D., & Damasio, H. (1994). Neuroanatomical correlates of electrodermal skin conductance responses. *Psychophysiology*, 31, 427-38.
- Tranel, D., Fowles, D. C., & Damasio, A. R. (1985). Electrodermal discrimination of familiar and unfamiliar faces: A methodology. *Psychophysiology*, 22, 403-8.
- Turpin, G. (1983). Psychophysiology, psychopathology, and the social environment. In A. Gale & J. A. Edwards (Eds.), *Physiological Correlates of Human Behavior*, pp. 265-80. New York: Academic Press.
- Turpin, G., Tarrier, N., & Sturgeon, D. (1988). Social psychophysiology and the study of biopsychosocial models of schizophrenia. In H. Wagner (Ed.), *Social Psychophysiology*. Chichester, U.K.: Wiley.

- Uno, T., & Grings, W. W. (1965). Autonomic components of orienting behavior. *Psychophysiology*, 1, 311-21.
- Vaughn, C., & Leff, J. P. (1976). The influence of family and social factors on the course of psychiatric illness. *British Journal of Psychiatry*, 129, 125-37.
- Vaughn, C. E., Snyder, K. S., Jones, S., Freeman, W. B., & Falloon, I. R. H. (1984). Family factors in schizophrenic relapse: A California replication of the British research on expressed emotion. *Archives of General Psychiatry*, 41, 1169-77.
- Venables, P. H., & Christie, M. J. (1973). Mechanisms, instrumentation, recording techniques, and quantification of responses. In Prokasy & Raskin, pp. 1-124.
- Venables, P. H., & Christie, M. J. (1980). Electrodermal activity. In I. Martin & P. H. Venables (Eds.), *Techniques in Psychophysiology*, pp. 3-67. Chichester, U.K.: Wiley.
- Venables, P. H., & Mitchell, D. A. (1996). The effects of age, sex and time of testing on skin conductance activity. *Biological Psychology*, 43, 87-101.
- Vigouroux, R. (1879). Sur le rôle de la résistance électrique des tissus dans l'électro-diagnostic. *Comptes Rendus Société de Biologie*, 31, 336-9.
- Vigouroux, R. (1888). The electrical resistance considered as a clinical sign. *Progrès Médical*, 3, 87-9.
- Vossel, G., & Rossman, R. (1984). Electrodermal habituation speed and visual monitoring performance. *Psychophysiology*, 21, 97-100.
- Wallin, B. G. (1981). Sympathetic nerve activity underlying electrodermal and cardiovascular reactions in man. *Psychophysiology*, 18, 470-6.
- Wang, G. H. (1964). *The Neural Control of Sweating*. Madison: University of Wisconsin Press.
- Ward, N. G., Doerr, H. O., & Storrie, M. C. (1983). Skin conductance: A potentially sensitive test for depression. *Psychiatry Research*, 10, 295-302.
- Wenger, M. A., Clemens, T. L., Coleman, M. A., Cullen, T. D., & Engel, B. T. (1961). Autonomic response specificity. *Psychosomatic Medicine*, 23, 185-93.
- Winer, B. J., Brown, D. R., & Michels, K. M. (1991). *Statistical Principles in Experimental Design*. New York: McGraw-Hill.
- Woodworth, R. S., & Schlosberg, H. (1954). *Experimental Psychology*, rev. ed. New York: Holt.
- Zahn, T. P. (1986). Psychophysiological approaches to psychopathology. In M. G. H. Coles, E. Donchin, & S. W. Porges (Eds.), *Psychophysiology: Systems, Processes, and Applications*, pp. 508-610. New York: Guilford.