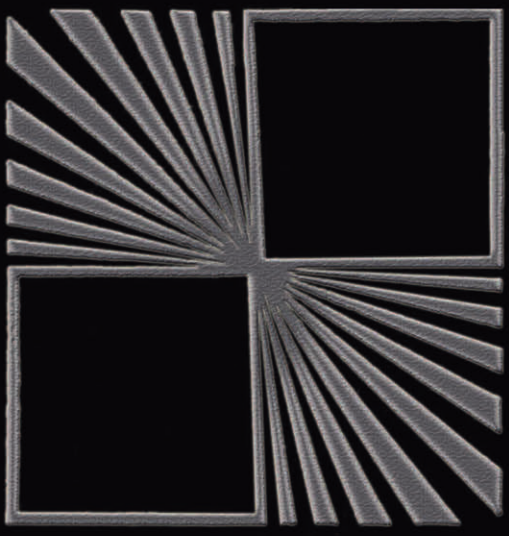
The Plenum Series in Behavioral Psychophysiology and Medicine
Series Editor: William J. Ray

Electrodermal Activity



Wolfram Boucsein

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THE PLENUM SERIES IN BEHAVIORAL PSYCHOPHYSIOLOGY AND MEDICINE

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Electrodermal Activity

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Foreword

Electrodermal activity was observed for the first time more than 150 years ago in Germany where, a quarter-century later, the scientific study of psychology also originated. Well into the 20th century, English-speaking psychologists all read German and, if they could, made pilgrimages to Leipzig and Heidelberg and other seats of German scholarship. Then gradually the focus of psychological research, including the new field of psychophysiology, shifted to the United States and Britain. Studies of electrodermal activity in particular originated mainly in North America. As a student in the early 1950s, I learned about what we then called the Galvanic Skin Response or GSR by reading C.W. Darrow, G.L. Freeman, R.A. Haggard, R.A. McCleary, and H.G. McCurdy, all in American English.

The current renaissance of German science has made it necessary for psychologists, once again, to attend to and learn from the work of their Teutonic colleagues. Fortunately for us monolingual Americans, English has become the lingua franca of our field; German scholars speak our language fluently when they visit the United States and understand us when we go to them. The return of German scholarship to what I shall loftily call the high table of psychophysiology is exemplified by this fine book, the most comprehensive treatise on the electrodermal system to appear in any language and now available in English.

In 1971, in the eighth volume of the journal *Psychophysiology,* Lykken and Venables commented, "Of all psychophysiological variables, the GSR can lay reasonable claim to being the most popular in current use. In spite of years of searching study, we are still surprisingly uncertain about the function, not to say the mechanism of this phenomenon. . . . Nevertheless, the GSR seems to be a robust sort of variable since, in hundreds of experiments, it continues stoutly to provide useful data in spite of being frequently abused by measurement techniques which range from the arbitrary to the positively weird." Now, more than twenty years later, the findings collected and integrated by Professor Boucsein should make it possible for future investigators to address this "robust sort of variable" with standardized technique and the respect that it deserves.

Wolf Boucsein was educated at the University of Giessen and is now Professor of Physiological Psychology at the University of Wuppertal. He has published extensively in the areas of psychophysiology and differential psychology. In the present volume he

has provided what should become the standard reference on the topic of electrodermal activity.

David T. Lykken

University of Minnesota

Preface

Since the discovery of the galvanic skin response over one hundred years ago, recording of electrodermal phenomena has become one of the most widely used methods of measurement in various fields of psychophysiology. This book provides, for the first time, a comprehensive summary of perspectives and histories from different scientific disciplines as well as a complete outline of methodological issues, and a review of results from the different areas of electrodermal research.

The book is divided into three parts. Part 1 (Chapters 1.1–1.5) focuses on the anatomical, physiological, and biophysical origins of electrodermal phenomena. Peripheral and central nervous system mechanisms are discussed, and fundamental biophysical principles are provided together with an extensive discussion of the current electrical models of electrodermal activity.

Part 2 (Chapters 2.1–2.6) outlines principles and methods of electrodermal recording, scoring techniques, and the action of internal and external influences on the signal, and describes statistical properties of the different electrodermal parameters. It ends with a summary of recent discussions of the advantages and disadvantages of the different methods.

Part 3 (Chapters 3.1–3.6) reviews applications of electrodermal recording techniques within psychophysiology, personality research, clinical and applied psychology, and medical disciplines, for example, dermatology and neurology. Areas such as orienting and habituation, classical and instrumental conditioning, information processing and storage, multidimensional arousal, sleep, and stress research are considered with respect to the theoretical modelling of vegetative concomitants of central nervous system phenomena. Aspects of specific validity of electrodermal measures are discussed within the framework of neurophysiological and psychophysiological systems.

The present volume is conceptualized as a handbook. A reader who is not especially interested in the signal's origins may start with Part 2, after having read the introductory Section 1.1.1 and the summary in Chapter 1.5. Readers having fundamental knowledge in electrophysics may skip Section 1.4.1, and also Sections 2.1.1 and 2.1.2. Since the book contains numerous cross-references to the different sections, starting from any point is possible without loss of content. Several chapters and sections end with summaries that provide the appropriate highlights (Chapters 1.5 and 3.6, and Sections 2.1.6, 2.2.7, and 2.3.5).

Appreciation for adding to the book's content is given to my co-workers Rüdiger Baltissen, Jörn Grabke, Peter Kirsch, and Florian Schaefer as well as to Mike Dawson, Bob Edelberg, and John Furedy. I would like also to thank Ulrike Hillmann, Marlies Knodel, Brigitte Kapanke, and Boris Damke for doing the text editing, and Sebastian Boucsein, Katrin Boucsein, Martina Promeuschel, and Timothy Skellett for helping with figures, references, and language editing. In addition, I would like to thank Cecilia Secor, Judith Ray and especially Alex Vincent, who helped tremendously to improve my English, the latter one also for making several proposals that added to the content. Finally, appreciation is given to the series editor, Bill Ray, who performed a great job getting the present volume published.

Wolfram Boucsein

Wuppertal, Germany, August 1992

Contents

1 Principles

Part 1: Principles

Since the 1880s, when psychological factors in relation to electrodermal phenomena were first observed, electrodermal recording has become one of the most frequently used biosignals in psychophysiology. The main reason for this popularity is the ease of obtaining a distinct electrodermal response, the intensity of which seems apparently related to stimulus intensity and/or its psychological significance. This is even possible with rather inexpensive equipment, not only in the laboratory but also under less controlled field conditions.

In spite of the widespread use of electrodermal recording in research and application, electrodermal phenomena are not completely understood. Stemming from neurology and physiology, electrodermal recording has become a domain of psychophysiology, and only in the last three or four decades has basic research in mechanisms underlying electrodermal phenomena intensified. However, a tradition of joint research is lacking in the related disciplines of anatomy, physiology, physics, and psychology. Moreover, scientific articles and summaries concerning electrodermal recording are spread over a wide variety of journals and books, and a comprehensive handbook on electrodermal activity has not been available until now.

After a general introduction, the first part of the present book combines anatomical, physiological, and biophysical aspects of electrodermal research that allows users with different backgrounds to understand all aspects of electrodermal phenomena without troublesome study of the large number of appropriate original texts.

1.1 Introduction

This chapter outlines terminology and gives basic definitions of the different electrodermal phenomena (Sect. 1.1.1). An introduction to electrodermal methodology and research is given in the mainly historically oriented Section 1.1.2, and finally a brief overview of more recent basic electrodermal research is given in Section 1.1.3.

1.1.1 Definitions and terminology

Electrodermal activity (EDA) was first introduced by Johnson and Lubin (1966) as a common term for all electrical phenomena in skin,[1] including all active as well as passive electrical properties which can be traced back to the skin and its appendages. One year later, a proposal for standardization made by a terminology commission of the Society of Psychophysiological Research had been published (Brown, 1967), and

[1] Dermal stems from Latin: *derma* = true skin, see Table 2, Sect. 1.2.1.1.

Table 1. Methods of electrodermal recording, units of measurement, and abbreviations in the corresponding classes of units.

Methods of recording	endo-somatic	exosomatic			
Applied current		direct current		alternating current	
Units	skin potential	skin resistance	skin conductance	skin impedance	skin admittance
Abbreviations:					
in general	SP	SR	SC	SZ	SY
tonic (level)	SPL	SRL	SCL	SZL	SYL
phasic (response)	SPR	SRR	SCR	SZR	SYR
Supplementary abbreviations:					
nonspecific reaction	NS.SPR	NS.SRR	NS.SCR	NS.SZR	NS.SYR
frequency	SPR freq.	SRR freq.	SCR freq.	SZR freq.	SYR freq.
amplitude	SPR amp.	SRR amp.	SCR amp.	SZR amp.	SYR amp.
latency	SPR lat.	SRR lat.	SCR lat.	SZR lat.	SYR lat.
rise time	SPR ris.t.	SRR ris.t.	SCR ris.t.	SZR ris.t.	SYR ris.t.
recovery time:					
63 % recovery	SPR rec.tc	SRR rec.tc	SCR rec.tc	SZR rec.tc	SYR rec.tc
50 % recovery	SPR rec.t/2	SRR rec.t/2	SCR rec.t/2	SZR rec.t/2	SYR rec.t/2

is now generally accepted (Table 1).[2] Electrodermal recordings which do not use an external current are called endosomatic, since only the potential differences originating in the skin itself are recorded. Methods of exosomatic recording use either direct current (DC) or alternating current (AC) applied to the skin. In DC measurement, if

[2]Abbreviations are determined by the first letter of the words: Skin, Potential, Resistance, and Conductance. Unfortunately, the commission overlooked that the abbreviation C is already reserved in physics for capacitance, and G is used for conductance instead, according to SI units (Sect. 1.4.1.1). Terminology used in AC methodology is somewhat more complicated. Edelberg (1972a) proposed A for admittance, and Z – the last letter in the alphabet – for the reciprocal unit, impedance. While the latter abbreviation was kept, admittance is abbreviated today by Y, the penultimate letter of the alphabet.

voltage is kept constant, EDA is recorded directly in skin conductance (SC) units, while skin resistance (SR) units are obtained when current is kept constant (Sect. 2.1.1). Accordingly, if effective voltage is kept constant in AC measurement, EDA is recorded directly as skin admittance (SY), while the appliance of constant effective current results in skin impedance (SZ) recordings (Sect. 2.1.5). The 3rd letters in electrodermal units refer to either level (L) or response (R). Accordingly, electrodermal activity is divided into tonic (EDL = electrodermal level) and phasic phenomena (EDR = electrodermal response or reaction). Typical forms of phasic EDRs are shown in Figures 34 and 35 (Sect. 2.3.1.2). Tonic electrodermal measures are obtained either as EDLs in reaction-free recording intervals, or as the number of non-stimulus-specific EDRs in a given time window (Sect. 2.3.2).

The use of the term "reaction" for phasic electrodermal phenomena suggests that there is a distinct relationship to a stimulus producing an EDR. However, there are often phasic parts of EDA which cannot be traced to any specific stimulation. Hence, they are called "spontaneous" or "nonspecific" EDRs (Sect. 2.3.2.2), which are characterized by the prefix "NS" (e.g., NS.SCR is used as an abbreviation for nonspecific skin conductance reaction).

In addition, various suffixes are added to the abbreviations of electrodermal reactions as shown in the lower part of Table 1, indicating the parameter which is obtained from the phasic component: frequency (e.g., SCR freq.), which means number of EDRs in a given time window; amplitude (e.g., SCR amp.), the height of a single response; latency (e.g., SCR lat.), the time from stimulus onset to reaction onset in case of a specific EDR; rise time (e.g., SCR ris.t.), the time from the onset of a reaction to its maximum; and recovery time, indicating the time that is needed to recover either 50% (e.g., SCR rec.t/2) or 63% (e.g., SCR rec.tc) of the amplitude. All those parameters are described in detail in Section 2.3.1.

An older notation persisting in the literature is "galvanic skin reaction" or "galvanic skin reflex" (GSR). It is recommended this term not be used for several reasons. Firstly, it suggests that skin can be regarded as a galvanic element, which does not correspond to the multiplicity and complexity of electrodermal phenomena (Sect. 1.4.2 & 1.4.3). Secondly, it points to EDRs as being elicited as a kind of reflex, which is the case neither in spontaneous EDRs nor in psychologically elicited electrodermal changes. Finally, the term GSR has been used to cover not only phasic electrodermal responses but also electrodermal phenomena in general, including tonic EDA, which gives rise to ambiguity.

There are also some recent tendencies in neighboring disciplines to use other terms and abbreviations, for example, the introduction of "peripheral autonomic surface potential" (PASP) in neurology (Knezevic & Bajada, 1985) instead of skin potential (SP). For the sake of interdisciplinary clarity, the sole use of terms and abbreviations as given in Table 1 is strongly recommended.

1.1.2 Early history of electrodermal research

The history of research on electrodermal activity, which has been thoroughly reviewed by Neumann and Blanton (1970), dates back to experiments performed in 1849 by DuBois-Reymond in Germany. He had his subjects put either both hands or both feet into a zinc sulphate solution, and observed an electrical current going from the limb at rest to the one that was voluntarily contracted (Veraguth, 1909). However, in accordance with the opinion shared by most workers at that time, DuBois-Reymond considered the observed phenomenon as being due to muscle action potentials.

Hermann, working in Switzerland, tried to explain electrical activity recorded from the skin as being caused by sweat gland reactions instead. The first experiment that showed a connection between sweat gland activity and current flow in skin was performed by Hermann and Luchsinger (1878), who observed that an electrical stimulation of the sciatic nerve in the curarized cat resulted in sweat secretion as well as an electric current in the footpad on the same side. Injections of atropine sulfate increased the latency of the current, decreased its intensity, and finally stopped both current and secretion. Three years later, Hermann repeated the voluntary movement experiment with humans as performed by DuBois-Reymond, and found that areas with stronger sweating such as palms and fingers showed greater skin current than other body sites such as the wrist or elbow regions, pointing to the importance of human sweat glands in electrodermal phenomena (Neumann & Blanton, 1970).

The observation which first related psychological factors to electrodermal activity is attributed to Vigouroux (1879), an electrotherapist working in France. He measured SR changes that paralleled changes in the amounts of anesthesia in hysterical patients, and supposed that both phenomena were dependent upon central processes. However, he could not believe that the sudden changes in SR he observed could be produced by local processes in the skin itself. Instead, he presumed a change in vascular conductivity, which was in line with the developing research on autonomic nervous control of blood flow at that time.

The essential discovery of electrodermal phenomena is, however, attributed to two researchers who might not have been aware of each other, the French neurologist Féré (1888), and the Russian physiologist Tarchanoff (1889). Féré used an external direct current, and observed a decrease in SR following sensory or emotional stimulation in hysterical patients. Since his paper on SR was a brief and informal report, it was not cited during the rest of the 1800s, and it is possible that Tarchanoff did not notice the work of Féré before publishing his own results on EDA in the same French journal (Veraguth, 1909). However, Neumann and Blanton (1970) suggested that international tensions between France and Germany, where Tarchanoff published his results again in 1890, were the cause of each ignoring the other's results. Tarchanoff himself did not use the exosomatic method as Féré did. Instead, he used endosomatic, or SP, recording in his observations of electrodermal changes following sensory stimulation, imagination, mental arithmetic, expectation, and voluntary muscle contractions.

Tarchanoff (1890), in his German paper, clearly presumed the electrodermal phenomena which he had observed to result from sweat gland activity and to the appropriate action of the secretory nerves, which were not well known at that time. He observed a current flow, even at rest, from areas rich in sweat glands to those poor in them. This result seems to be in line with the Swiss/German tradition mentioned above. On the contrary, Féré – more in the French tradition – presumed the decrease of SR following stimulation to be due to a decrease of skin blood flow caused by partial displacement of the peripheral resistance of blood by the lower resistance of interstitial fluidity. The interpretation of the SRR as a vasomotor phenomenon, which had been defended for the last time in 1933 by McDowall (Edelberg, 1972a), is no longer pursued seriously, because the EDR showed independence from plethysmographic changes, and relationships between EDA and skin blood flow remain more or less contradictory (Sect. 2.4.2.1). Another hypothesis stated in 1902 by Sommer, suggesting the EDR was a result of involuntary muscle activity, could also not be proven, since a correlation between EDR and finger tremor is lacking (Venables & Christie, 1973).

In 1904, the engineer Müller demonstrated the electrodermal phenomenon to the Swiss neurologist Veraguth, claiming to have discovered it independently (Neumann & Blanton, 1970). Veraguth's monograph entitled "Das psychogalvanische Reflexphänomen" *(The psychogalvanic reflex phenomenon)*, which was published in 1909, for the first time gave rise to a broader interest from psychiatrists and psychologists, because it focused on the suggested psychophysiological origin of electrodermal phenomena. Since then, the number of articles on basic electrodermal research as well as on various applications has increased.

In spite of the insufficient methods for biosignal recording and amplification at that time, physiologists pushed forward with the investigation of the origins of electrodermal activity. In 1921, Ebbecke found a local EDR which could be elicited by rubbing or pressing skin sites, even several hours post-mortem (Keller, 1963), and these results directed attention to the polarizational properties of skin (Sect. 1.4.2.3). In 1923, Gildemeister applied high-frequency alternating currents to skin, and found very small, or even an absence of, EDRs. His conclusion that SR was possibly only an impedance phenomenon resulting from membrane polarizations (Sect. 1.4.2) is no longer regarded as valid (Edelberg, 1971). Additionally, AC measurement, the use of which is quite common in investigations of electrical properties of tissues, was not used very much in electrodermal recordings.

In 1928 and 1929, Gildemeister and Rein made a decisive contribution to the investigation of the origins of endosomatic EDA. For the first time they restricted the locus of SP origin to only one of two recording sites by injuring the skin below the other electrode, where no SP of its own could develop (Keller, 1963). In 1929, Richter was the first to state the hypothesis of a causal mechanism for EDA including both epidermal and sweat gland mechanisms, which is still regarded as valid (Edelberg, 1972a; Fowles, 1986a). Thus, by the end of the twenties, the early phase of electrodermal research was completed. Rather extensive reviews of early electrodermal methodology and its sig-

nificance in physiological research on arousal and emotion (Sect. 3.2.1 & 3.2.2) were provided by Wechsler (1925) as well as by Woodworth and Schlosberg (1954) in their book on experimental psychology.

1.1.3 Recent developments in electrodermal research

Improvements in equipment for physiological and psychophysiological research, such as the invention of the oscilloscope, the polygraph, and today's highly integrated amplifier technique, contributed to the increase not only in applications but also in the amount of basic electrodermal research during the last three or four decades.[3] Thus Bloch (1952), with human subjects, and Ladpli and Wang (1960), with cats, were equipped to take polygraphic recordings simultaneously from different limbs, and Wang (1964) added much to the knowledge of central mechanisms eliciting EDA by using appropriate methods with cats, performing lesion experiments at different levels in the CNS (Sect. 1.3.4.1). Although these animal results are not fully generalizable to humans (Footnote 9, Sect. 1.2.3), much of our knowledge concerning the central origin of EDA is based on research with cats, since investigations of EDA in nonhuman primates are sparse (e.g., Kimble, Bagshaw, & Pribram, 1965).

Basic electrodermal research with human subjects as performed in the previous decades concentrates on the peripheral mechanisms (Chapter 1.5) as well as on the influence of different methods of measurement on recordings (Chapter 2.6). As a result, Darrow (1964) as well as Lykken and Venables (1971) strongly supported skin conductance units as being adequate with respect to physiological models of the peripheral EDA mechanisms (Sect. 2.6.5). Edelberg (1971), after having performed electrodermal research for more than a decade, proposed an electrical model of the skin which takes into account the presence of polarization capacitances (Sect. 1.4.3.2). Using this background, Edelberg (1972a) for the first time established the different psychophysiological aspects of various EDA components in detail, including parameters which were recently focused on, for example, rise times and recoveries of EDRs (Sect. 2.3.1.3). Additional basic research was performed on the influence of peripherally acting drugs on EDA (e.g., anticholinergics like atropine). Recently, basic research also centered on AC measurement of EDA (Sect. 1.4.3.3).

With a few exceptions, EDA methodology is nowadays regarded as being well established (see Part 2). Earlier comprehensive reviews of EDA methodology were given by Edelberg (1967) in Brown's book on psychophysiological methods, and – enriched with more empirical results – by Edelberg (1972a) in the handbook of psychophysiology edited by Greenfield and Sternbach. In the only book solely concerned with EDA that has appeared up to now – a reader edited by Prokasy and Raskin (1973) – the methodological section was written by Venables and Christie, who also wrote the

[3] In the thirties, Darrow (e.g., Darrow, 1933, 1937a, b) worked as a pioneer in various fields of electrodermal research.

EDA chapter in the Martin and Venables (1980) reader on techniques in psychophysiology. In that chapter, Venables also reported the results of his extensive cross-sectional survey on EDA performed in Mauritius as a follow-up study during the seventies.

During the beginning of the 1980s, the development of EDA methodology came to a standstill. As a consequence, Fowles's (1986a) chapter on EDA in a handbook of psychophysiology contains only an appendix concerning methodology, and focuses instead on recent research into basic electrodermal mechanisms and the psychological significance of electrodermal parameters. The widespread use of EDA as a tool in basic as well as in applied psychophysiological research has resulted in a great variety of publications on EDA. Edelberg (1972a) estimated the number of these publications as exceeding 1,500 in 1972, and a first attempt to summarize EDA research in the different fields occurred in the above-mentioned reader (Prokasy & Raskin, 1973). Since then, many additional studies have appeared (see Part 3), giving evidence that EDA measurement is nowadays widely regarded as a most useful method in many psychophysiological contexts.

However, according to the present author's opinion, there is much work to be done concerning basic electrodermal phenomena, since EDA turns out to be a rather complex phenomenon with respect to its central and peripheral causal mechanisms. Future basic electrodermal research should be encouraged by the integrative view of anatomical, physiological, and physical principles of EDA, as outlined in the following chapters. Electrodermal research also requires an improvement of interdisciplinary collaboration in basic research as well as in various applications.

1.2 Anatomy

It is far beyond the scope of this chapter to give an exhaustive description of the skin's complex features. Only those parts of the skin and its appendages that are necessary to understand the mechanisms of EDA – the epidermis and the sweat glands – are discussed in detail. The reader who is particularly interested in this topic is referred to the textbook by Millington and Wilkinson (1983), to the multivolume series edited by Jarrett (e.g., 1973a, 1980), or to the handbook of dermatology and venerology which is in part German and in part English (e.g., the volumes edited by Marchionini & Spier, 1963; or by Schwarz, Spier, & Stüttgen, 1979).

The skin functions both as protection against and as contact with the environment. It protects the body against chemical, mechanical, and thermal assault, certain types of radiation, and numerous infections. As a contact surface for sensory stimuli, the skin contains mechanoreceptors, thermoreceptors, and nociceptors (pain receptors). The skin's role in the regulation of perspiration is twofold. On the one hand, the skin prevents the body from drying out; on the other hand, with the aid of the sweat glands, the skin enables a controlled emission of body fluid.

1.2.1 Vertical structure of the skin

The skin is composed of different layers which are easily distinguishable from one another by means of a light microscope. These layers show characteristic differences at different body sites. Thus, the layers described in Figure 1 and Table 2 do not appear in the same way and are not clearly recognizable at all sites. Figure 1 shows a typical cross-section of glabrous (hairless) skin, as would appear on the palms of the hands (palmar) and the soles of the feet (plantar or volar). On these strongly mechanically stressed surfaces, which are especially significant for EDA measurements because of their specificity for emotional sweating (Sect. 1.3.2.4), the epidermis has an unusual thickness of ca. 1 mm; ordinarily it is only 50–200 μm.

The skin (cutis) is composed of two markedly differentiable layers, the dermis and the epidermis.[4] The epidermis lies on the skin surface and consists of epithelial tissue, which becomes progressively more horny nearer the surface. The deeper-lying dermis consists of taut, fibrous connective tissue. The epidermis is relatively thin in comparison with the dermis.

The subcutis is composed of loose connective tissue which forms the transitional layer between the skin and the deeper-lying tissue. It contains the secretory part of the sweat glands, appearing as a glomerulus (Fig. 1), as well as fatty tissue, and the larger vessels which supply the body surface.

1.2.1.1 The epidermis

The most common horizontal division of skin is into five different layers (Jarrett, 1973a; Klaschka, 1979).[5] The lowest layer, in which the epidermal cells are built up, is the *stratum germinativum* (from germinate); it lies on top of the basal lamina (which is included in the dermis) and is sometimes named after this lamina as the basal layer. This layer produces mainly *keratinocytes*, which are cells that can store keratin and later become horny; it also produces *melanocytes*, which supply the skin pigment melanin, as well as Langerhans and Merkel cells. Within a period of around 30 days, the keratinocytes reach the skin surface, and are exfoliated there in the form of horny (keratinized) plates. The characteristic form changes which these cells undergo as they migrate have been used in part to characterize the corresponding epidermal layers.

Figure 1 shows the basal cells in the stratum germinativum, which are at first columnar and later rounded. During the course of their migration they shrink, which enlarges the intercellular spaces. Since they, with their cytoplasmic extensions, come to appear as having spines, sometimes the term *stratum spinosum*, or prickle cell layer, is applied as a special name for this particular stage. The stratum germinativum and the stratum

[4]Note that the dermis is also known as the *corium* (Latin for tauter skin) and that sometimes the term *cutis* is used only for the dermal part of skin (cutis vera, see Table 2).

[5]Some authors (e.g., Orfanos, 1972) proposed a division into three layers, which may have been dependent upon microscopic technology.

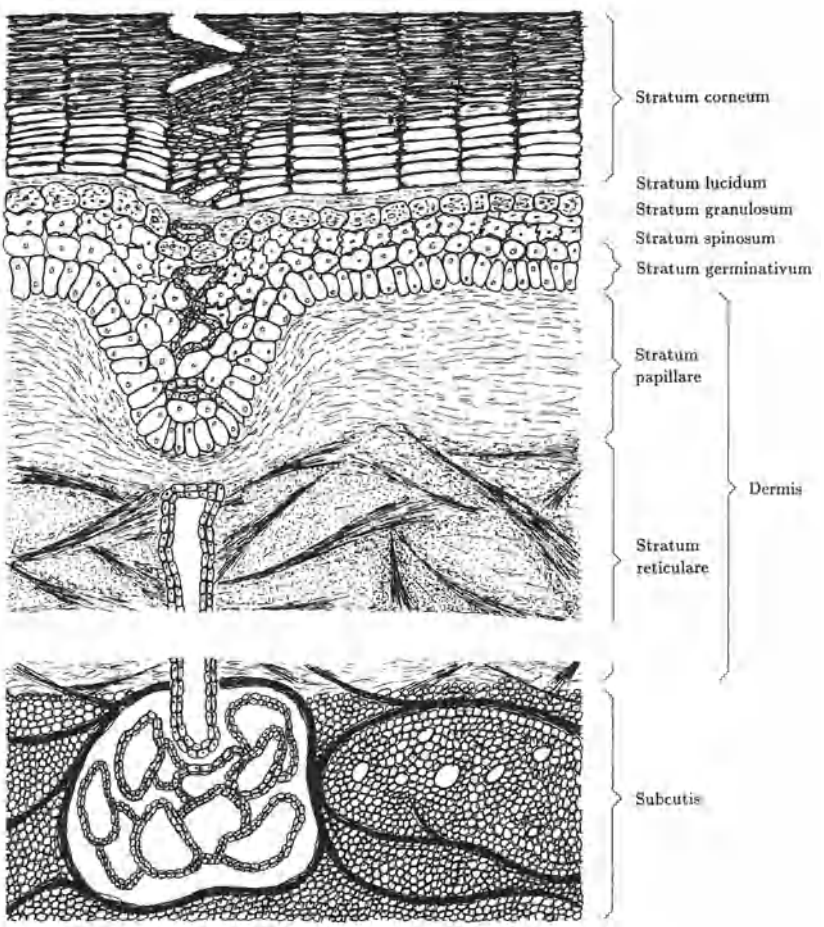

Figure 1. Layered construction of the glabrous human skin. An eccrine sweat gland, in its glomerulus, together with its straight dermal and irregularly coiled epidermal duct (acrosyringium) is shown in cross-section. A part of the reticular layer has been omitted due to its size in relation to the rest.

Table 2. The layers of the skin. The zonal layering is not so distinct in every region of the skin. The stratum lucidum is only clearly recognizable on the palmar and plantar skin areas.

Cutis (skin)	Epidermis	Stratum corneum	upper zone middle zone lower zone
		Stratum lucidum Stratum granulosum (granular layer)	Stratum intermedium
		Stratum spinosum (prickle cell layer) Stratum germinativum (basal layer)	Stratum Malpighii
	Dermis (cutis vera = true skin)	Stratum papillare (papillary layer) Stratum reticulare (reticular layer)	
Subcutis (hypodermis)			

spinosum are labelled together as the Malpighian layer (*stratum Malpighii*) as shown in Table 2.

The *stratum intermedium* represents a transitional zone[6] between the cells of the stratum Malpighii, which are not horny, and the horny cells of the outer epidermal layer, the stratum corneum (Table 2).

In the lower stratum intermedium the keratinocytes contain keratohyaline granules, thus this layer is named the granular layer (*stratum granulosum*). During their upward migration, the cells may be soaked with an oily substance called eleidin. Due to their ability to strongly refract light, they appear as a homogenous layer, named the *stratum lucidum*. The stratum lucidum, however, is visible only on some body sites, especially on the palms and the soles, upon successful removal of the whole horny layer (Sect. 2.2.1.2).

The outer epidermal layer is called the *stratum corneum* (from Latin *cornu* for horn) or, since its cells are fully keratinized,[7] the keratin-layer zone. In accordance with both

[6]This transitional layer, with an overall thickness of around 1 μm, is unusually thin in comparison with the underlying Malpighian layer and the overlying part of the epidermis. Orfanos (1972) has hence suggested that this layer should not be subdivided, but on the contrary should be named on the whole as the stratum intermedium (i.e., intermediate). However, sometimes only the granular layer is distinguished as the transitional zone (Jarrett, 1973a).

[7]The process of keratinization begins during mitosis via forming of so-called *tonofilaments*, which are thin intraplasmatic fibers. In the upper layers, they are transformed into bundles of *tonofibrillas* having a greater density. With the keratohyaline generated in the lower stratum intermedium, these fibrillas merge to form complexes, which are converted in the upper stratum intermedium from one cell layer to another into epidermal keratin through changes in the cellular milieu. The tonofilaments are approximatively

the form of its cells and the width of its intercellular spaces, the horny layer can be subdivided into a lower, a middle, and an upper part (Table 2). However, this subdivision is not as clear in other sites as it is in the palmar and plantar ones. Therefore, the corneum layers can sometimes only be distinguished as a taut layer called the *stratum compactum*, or *stratum conjunctum*, and the *stratum disjunctum*, a loose surface layer. According to Tregear (1966), the stratum disjunctum forms the epidermal part which cannot be removed by the stripping technique (Sect. 1.3.4.2.1).

In the corneum, the fully keratinized cell acts at first as a direct barrier between the body and its environment. After a while, the horny cell desquamates like a withered leaf, curling up from its margin. An adult's skin surface area, which is approximately 1.7 m^2, loses about .5–1 g of such horny material every day. At the same time, an appropriate amount of keratinocytes is replaced through cell proliferation (mitosis) of the basal epidermal cells in a kind of continuous molting.

In the stratum germinativum, cells are meshed together like a zipper. Sometimes a total melting of cell membranes appears, together with a reduction of intercellular spaces. Some of these contact areas of adjacent membranes remain visible in the stratum corneum as membrane-like lines. They probably work as electrical contact areas for transmission of action potentials from cell to cell (Sect. 1.3.4.2.2). The keratinized epidermal cells are kept in their formation by desmosomes (Footnote 7) until they fall away from the skin surface.

As shown in Figure 1, the keratinocytes undergo a typical metamorphosis of their form and position while moving from the basal layer to the corneum, probably caused by the growing pressure of the cells pushing from inside out. The basal cells change their form from upright spheric or elliptic cell bodies, to flat, keratinized cells lying parallel to the skin's surface. In the formation of the horny layer, each cell forms a hexagonal sheet approximately 30–50 μm in diameter, interlocking precisely into ramifications of adjacent cells above and beneath like a zipper. If the epidermis is not influenced by irritation, single cell layers may be visible, stacking up from the basal to the upper horny layer like boxes.

The epidermis, the layering of which is of great importance to EDA, consists of a regularly arranged cell formation, which becomes dryer toward its outside layer as the cells become less tightly packed and look more like slide-shaped parallel structures. The completely horny outer layer, the stratum corneum, is especially thick on palmar and plantar sites, which are preferred for electrodermal recording (Sect. 2.2.1.1). The stratum corneum's role in producing changes of the skin's resistance is discussed in Section 1.4.2.1.

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parallel to the surfaces of the flattened cells, but are not in contact with the *desmosomes*, which are the intercellular contact zones of the keratinocytes. In the spinous layer, the number of desmosomes decreases, and the intercellular spaces enlarge. This enables other cells, e.g., the melanocytes, to change their positions through movements.

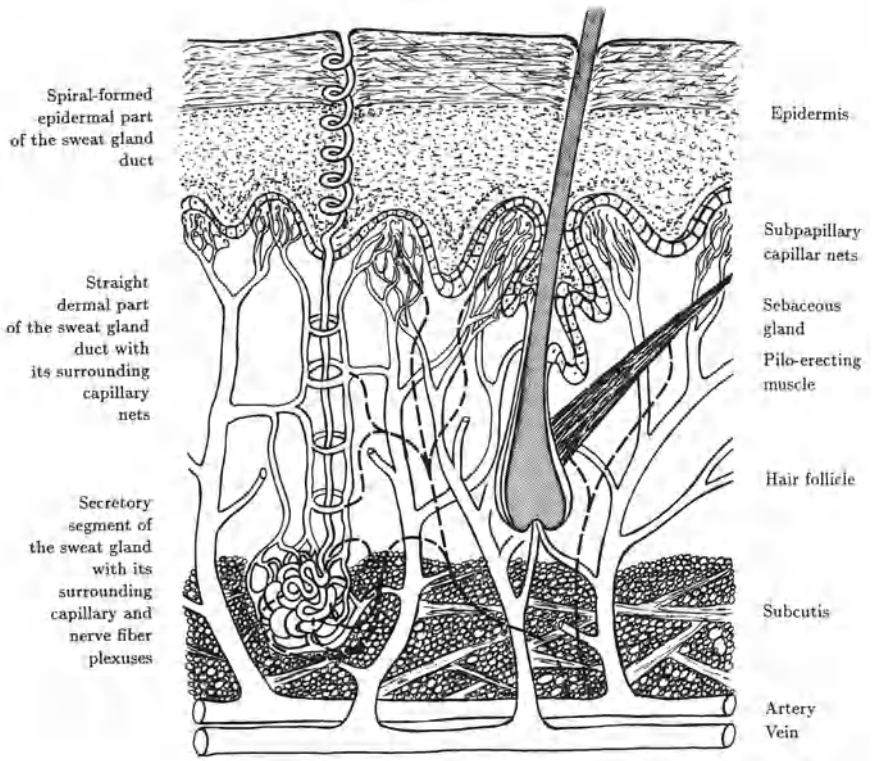

Figure 2. Schematic vertical section of the skin that artificially combines a sweat gland in ridged skin (left) together with a hair and a sebacous gland in polygonal skin (right). Besides the supply with blood vessels, the efferent sympathetic innervation is indicated by dashed lines (Sect.1.3.2).

1.2.1.2 Dermis and subcutis

The dermis, also labelled the *corium* (which means leather, since leather is the tanned corium of animals), is much thicker than the epidermis. However, it consists of only two dermal layers (Table 2), distinguishable according to their density and the arrangement of their collagen fibers (Fig. 1).

The dermal layer next to the epidermis is called the *papillary stratum.* It is named after fingerlike projections, the *dermal papillae*, which fit into cavities in the underside of the epidermis. Thus, the two main parts of the cutis are intimately intermeshed at the epidermal dermal junction. Apart from the possible adhesive effects of this gearing, there is also a great increase of the basal-layer area, and hence an enlargement of

the area for producing new epidermal cells (Montagna & Parakkal, 1974). The epidermal dermal boundary is formed by a so-called basal-membrane zone, which is an adhesive layer in which the epidermal basal cells are inserted with projections. In the papillar stratum, arterial and venous blood vessels end in a capillary net (Fig. 2), and receptor organs, melanocytes, and free collagen cells are included in this layer. The inner dermal layer is called the *reticular stratum* because of its texture, made by strong collagenous fibers, giving the skin a high resistance against rupture. Thus, the reticular stratum, sometimes called the *fibrous stratum*, forms the leathery skin in its true sense.

The subcutis, which is also labelled the *hypodermis*, consists of loose connective tissue. It connects the skin with the connective tissue covering the muscles, and it allows for good horizontal mobility of the skin across its surface. The subcutis has the ability to store fat, thus working as a thermal as well as a mechanical insulation layer. It contains the nerves and vessels that supply the skin, also the hair follicles and glands, (e.g., the secretory part of the sweat gland; Fig. 1). According to some authors (e.g., Millington & Wilkinson, 1983), the secretory parts of some sweat glands are located in the dermis and not in the subcutis. These sweat glands then are surrounded by fatty tissue instead of collagen fiber bundles (Fig. 2).

1.2.1.3 Vascular system of the skin

As pointed out in the preceding section, the bigger vessels supplying the body surface are located in the subcutis. From there, smaller ramifications supply the sweat glands, the hair follicles, and the subpapillary capillary nets. The arterioles located there are surrounded by small muscles which are innervated adrenergically and regulate the cutaneous blood flow. Arteriovenous anastomoses, which form bypasses of the capillary nets (Stüttgen & Forssmann, 1981), and also glomeruli (similar to those located in the kidney) in some body parts which jut outwards, such as fingertips, are also controlled by these adrenergic muscle fibers.

The lymphatics form nets in the dermis and in the subcutis. The main portion of the lymph flows away via subcutaneous lymph vessels. As well as the blood and the interstititial fluid (Sect. 1.4.2.1), the lymph contributes to the relatively high electrical conductivity of the inner skin layers.

1.2.2 Horizontal structure of the skin

There are regional differences not only in the skin's vertical layering, as outlined in Section 1.2.1.1, but also in its horizontal structure. In the early stages of embryonal development, patterns are formed by either ridge formation or folding (Millington & Wilkinson, 1983), and thus are referred to here as ridged skin and polygonal skin.

Ridged skin is seen only on the palms and soles, including the flexor side of fingers and toes. At these sites, the skin surface is covered with ridges and furrows, the pattern of which is genetically fixed and corresponds to the pattern of the papillary layer

(Sect. 1.2.1.2). Two papillar ridges project into each epidermal ridge. The sweat gland ducts usually enter the epidermis at the nadir of the ridges. The ridged skin is glabrous (hairless) and has no sebaceous nor scent glands.

The rest of the body is covered by polygonal skin patterns. *Polygonal skin* is divided by thin channels into numerous polygons, the pattern of which also corresponds to the pattern of the papillary layer. Both the number of polygons and the depth of channels are dependent on the degree of elasticity necessary, for example, for body movements. At some sites the channels may even disappear. Unlike ridged skin, the ducts of sweat and scent glands enter the epidermis at the tops of the ridges. Hairs and sebaceous glands, however, are located in the channels of the polygonal skin.

1.2.3 Distribution and structure of sweat glands

The sweat glands are considered to be exocrine glands because they secrete directly onto the skin's surface. The human body has about three million sweat glands, the greatest density being found on the palms, soles, and forehead, and the least density on the arms, legs, and trunk (Kuno, 1956). They are totally missing on the lips and in the inner ear channel (Pinkus, 1971), on the glans penis and clitoris, on the labia minora, and on the inner surface of the prepuce (Montagna & Parakkal, 1974). The following mean numbers of sweat gland per cm^2 of the adult's skin are taken from Millington and Wilkinson (1983): 233 on the palms, 620 on the soles, 360 on the forehead, and only 120 on the thighs. However, not all of these glands are active. Children may have much greater sweat gland densities, since their number decreases from fetal stage (3,000 per cm^2 in the 24th week of pregnancy) to adulthood (Sect. 2.4.3.1). This is because the total number of sweat glands is fixed at birth, yet the total body surface area increases by a factor of 7 from birth to adulthood (Montagna & Parakkal, 1974). There are also some racial differences in the distribution and activity of sweat glands (Sect. 2.4.3.3).

The majority of human sweat glands are regarded as *eccrine*, which means that their secretions do not contain noticeable amounts of cytoplasm from the glandular cells. In addition, a major number of the large sweat glands, mainly found in the areola region of the breast, the axillary, the circumanal, and the genital regions, are *apocrine* (Sato, 1977; Millington & Wilkinson, 1983).[8] This means that the secretion is formed when the apical (distal) part of the glandular cell is tied off, and the cytoplasm which is lost via secretion has to be replaced. Apocrine glands open into hair follicles, and their secretory functions do not begin until puberty (Sato, 1977). However, apocrine sweat glands play only a negligible role with respect to the total amount of sweating (Herrmann, Ippen, Schaefer, & Stüttgen, 1973).[9]

[8]Some authors (e.g., Thiele, 1981a) use the term "atrichial" (from Greek: τριχσς = hair) for the eccrine glands, which means these glands are not associated with hair follicles; most apocrine glands are (Venables & Christie, 1973, p. 19).

[9]Sweat glands are found in the footpads of many species (Edelberg, 1972a, p. 378). Wang (1964) presumes that the cat's sweat glands are apocrine instead of eccrine, but this view remains controversial

The eccrine sweat gland can be subdivided into the secretory segment and the duct. The secretory segment is located in the subcutis or in the dermis (Sect. 1.2.1.2). It consists of a tube which is irregulary coiled into a rounded mass approximately .4 mm in diameter (Fig. 2). From it comes the duct, following an undulating course through the dermis (called, however, the straight duct) and then a spiral course through the epidermis (Ellis, 1968). The dermal part of the duct also contains secretory cells (Odland, 1983).

Both the secretory as well as the ductal segments are formed by double or triple cell layers surrounding a lumen of 5–10 μm in diameter. The cells of the outer layer are called basal cells, those of the inner layer luminal cells. The wall of the epidermal part of the duct, the acrosyringium, has no cells of its own in the part which passes through the stratum corneum (Fig. 1). The duct opens onto the skin surface through a little pore.

According to Hashimoto (1978), a part of the duct between the secretory segment and the beginning of the undulating dermal duct has walls composed of only single cell layers, and is not surrounded by myoepithelial cells (Sect. 1.3.2). On the other hand, a cross-section of the secretory segment shows that it is surrounded by a thin sheet, the basal lamina, or basement membrane. Above it, a layer of myoepithelial cells, the form of which resembles that of smooth muscle cells, lines the outermost portion of the secretory tubule.

The secretory cells are subdivided into either clear, serous cells, or dark, mucous cells (Sect. 1.3.3.1). The clear cells are noteworthy for their glycogen content and abundant mitochondria (Sato, 1977), which is suggestive of the high rate of metabolic activity necessary for active sweat secretion by these serous cells. The other secretory cells show a dark color under the light microscope, resulting from abundant mucous granules and ribosomes which are responsible for the secretion of mucous substances (mucopolysaccharides).

There are two kinds of intercellular spaces in the secretory segment of the sweat gland: the intercellular channels, which open into the basal interface, and the intercellular canaliculi, which may be regarded as extensions of the lumen (Sato, 1977). While the intercellular channels allow absorption of material from the interstitium, the canaliculi permit the secretion of sweat into the lumen.

While the coiled part of the dermal duct, which cannot be clearly distinguished visually from the secretory part of the sweat gland, shows very tight junctions of its luminal cells, the cells in the so-called straight dermal duct show interspaces. According to Hashimoto (1978), these interspaces may function to increase the surface area available for reabsorption of sodium from the precursor sweat (Sect.1.3.3.1). The acrosy-

(Edelberg, 1972a). Sato (1977) found it unlikely that the cat's or the rat's sweat glands had the capability to reabsorb ductal NaCl (Sect. 1.3.3.1), while those from the monkey's paw closely resembled human eccrine sweat glands. Thus, generalization from primate results to human species is more tenable than from cats or rats. Types and distributions of sweat glands in different species are summed up in a description by Fowles (1986a, p. 54); more details are given by Weiner and Hellmann (1960).

ringial luminal cells (Fig. 1) also show those interspaces, which – more prominent in the human embryo than in the adult – seem to contain lysosomes. Hashimoto assumes that materials and fluid absorbed by the epidermal duct must be digested within these lysosomes. It is assumed that the acrosyringium produces its own keratinocytes (Sect. 1.2.1.1), and that its keratinization is more advanced than that in the surrounding epidermis. Thus the sweat gland pore opening onto the skin surface is of the same type as epidermal cells, making its wall indistinguishable from its surrounding, and allowing the sweat to pour out easily onto the stratum corneum.

1.2.4 Other effector and sensor organs in the skin

Besides sweat glands, the dermis contains other glands: the scent glands, which are present only at some sites (the axillae, the genital and perianal area, as well as in the external ear canal); the sebaceous glands, which are – with a few exceptions – linked to the hair follicles (Fig. 2); and the mammary glands. The hairs are likely to have efferent and afferent functions as well. They mount diagonally to the body surface out of their roots, which are infundibular insertions into the skin into which the sebaceous glands discharge. Below the sebaceous gland pore, originating from the side to which the hair is inclined, lies a small bundle of smooth muscle cells, the pilo-erecting muscle, which runs diagonally up to beneath the epidermis. The muscle can erect the hair and can also retract the skin (forming goose flesh), whereby the sebaceous glands are compressed between the muscle and the hair-root sheath. Hair is found only on polygonal skin and not in the regions used for EDA measurement, the palmar and plantar areas (Sect. 1.2.2).

The hair roots reach into the upper subcutis. Each is surrounded by a dense nerve net which possibly serves a perceptual role. Other receptors of skin sensory organs appear in all layers of the subcutis and cutis in large numbers. There are free-ending sensitive nerves; that is, they do not end in recognizable specific receptor structures. The endings can, however, also be encapsulated in connective tissue (e.g., Pacinian corpuscles).

Some of the sensory nerves reach into the epidermis, such as nerve endings found in the stratum germinativum, and these possibly serve as tactile receptors (Sect. 1.3.5). There is also an indication of an efferent system which sensitizes such peripheral receptors (Edelberg, 1971).

1.3 Physiology

As in the preceding chapter concerning the anatomical aspects, this chapter will outline only those physiological mechanisms required for understanding electrodermal mechanisms. However, the appropriate restrictions cannot be made as easily as in anatomy, because the innervations of skin and sweat glands are placed in the context of

the autonomic nervous system (ANS), which is highly complex. Its thermoregulatory aspects, in which skin and sweat glands are also involved, are especially influenced by various physiological mechanisms. Thus, the description of mechanisms which have more indirect influences on EDA (e.g., peripheral vascularization) is restricted to some peripheral parts localized in the skin.

1.3.1 Efferent innervation of the skin

Human skin has numerous efferent vegetative fibers, including sympathetic fibers for innervation of the eccrine sweat gland's secretory segment[10] and of the pilo-erecting muscles (Fig. 2), as well as vasoconstrictive efferences for the blood vessels. As they are intermeshed, it is not possible by means of a light microscope to differentiate nerve fibers going to the sweat glands from those supplying the blood vessels. Whether there is an additional parasympathetic innervation of the skin's blood vessels, which could be analogous to the regulation of the blood flow in skeletal muscles, or whether the dilatatory reactions of the skin's blood flow are only due to a central inhibition of the sympathetic vasoconstrictive fibers, is still debatable (Jänig, Sundlöf, & Wallin, 1983).[11]

The postganglionic sympathetic fibers leave the sympathetic trunk via the gray communicating ramus, being included in the so-called mixed spinal nerve, which also contains all motor as well as sensory fibers travelling into and from the periphery (Fig. 3). At its peripheral end, the sympathetic fibers run intimately with the somatosensory fibers for surface sensibility, forming their characteristic plexuses afterward (Fig. 2).

Separation of human sudorisecretory from vasoconstrictive fibers is only possible in the periphery, namely, via stimulation and blocking procedures, but not at the spinal cord level, where sudorisecretory efferents cannot be differentiated from other sympathetic fibers (Schliack & Schiffter, 1979). The spinal sympathetic nerves, which descend in the anterolateral part of the cord near the pyramidal tract (Sect. 1.3.2.2), are switched over in the lateral horn, and leave the cord via its ventral root together with the motoric fibers, travelling via the white communicating ramus to the sympathetic trunk (Fig. 3). Here the neuronal activity is distributed by numerous collaterals to different levels of the sympathetic trunk, so that one preganglionic fiber may reach up to 16 postganglionic neurons. The collaterals of fibers originating in the upper thoracic part are mainly cranial oriented, whereas those from fibers originating in the lower thoracic, as well as in the lumbar part of the cord, are mainly caudal oriented (Schliack & Schiffter, 1979). In spite of the fact that neuronal activity is widely distributed, being typical for the sympathetic as compared to the parasympathetic system, the organization within the sympathetic nervous system is mainly segment oriented. This can be shown with

[10] Only few human apocrine glands possess an ANS innervation (e.g., those in the axilla); they may be partly or even mainly under the control of circulating adrenaline (Weiner & Hellmann, 1960).

[11] The search for origins of the skin's vegetative activity is further complicated by the fact that transmitters such as noradrenaline, which acts vasoconstrictorily on the peripheral blood vessels (Sect. 1.2.1.3), are also circulating freely in the blood.

segmental reflexes elicited by electrical stimulation of the ventral root, which are most pronounced when the same segment is stimulated (Jänig, 1985).

However, skin reflexes can also be elicited without inclusion of the spinal cord, as can be shown with so-called axon reflexes. These reflexes are dependent on the peripheral organization of the vegetative fibers in a certain region of the skin, which all stem from a distribution point within the inner half of the dermis. If one of the efferent collaterals originating there is mechanically stimulated, an impulse is transmitted backwards to that distribution point, which is acting like a ganglion, sending efferent "sympathetic" signals via the other collaterals into the periphery, thereby causing a local sweat secretion (Schliack & Schiffter, 1979). Another kind of axon reflex is elicited by nociceptive afferents, which cause vasodilatation in the corresponding skin area via a hitherto unknown pathway.

1.3.2 Innervation of sweat glands

Of all vegetative efferent innervations, those of the sweat glands have been investigated most thoroughly. The secretory part of the sweat gland is supplied by widely ramified sympathetic nerve fibers, some of which also reach the dermal part of the duct (Sinclair, 1973). Though postganglionic sympathetic transmission is normally adrenergic, using noradrenaline as a transmitter, sudorisecretory transmission is cholinergic, which means that acetylcholine acts as a synaptic transmitter. This has given rise to discussions of a possible parasympathetic innervation of sweat glands (Tharp, 1983), in contrast to the more generally adopted view of sympathetic sweat gland innervation. There has always been some question concerning reasons for the cholinergic transmission in the postganglionic sympathetic system. With respect to this, Fowles (1986a) pointed to the fact that the sweat glands have exocrine functions, and that cholinergic innervation is common in exocrine glands.

Nevertheless, there are also adrenergic fibers that travel to sweat glands via the peripheral nerve supplying the skin. Previously, the additional adrenergic supply had been thought to be related to the specific reactivity of sweat glands on palmar and plantar sites to emotional changes (Sect. 1.3.2.4). However, because an adrenergic innervation has also been found in eccrine sweat glands within other regions (Shields, MacDowell, Fairchild, & Campbell, 1987), this is no longer thought to be true.

Weiner and Hellmann (1960) showed adrenaline acting on apocrine glands by bringing about myoepithelial contractions. Thus, there had been some speculation that these fibers also supply the myoepithelial cells surrounding the secretory segment as well as the dermal part of the eccrine sweat gland, the function of which is described in Section 1.3.3.1. However, Sato (1977) showed that those myoepithelial cells react to cholinergic stimulation only.

There is some evidence for adrenergic supply of the apocrine sweat glands, as mentioned in Section 1.2.3 (Millington & Wilkinson, 1983). Since a general neural innervation of human apocrine sweat glands is lacking (Footnote 10, Sect. 1.3.1), Grice and

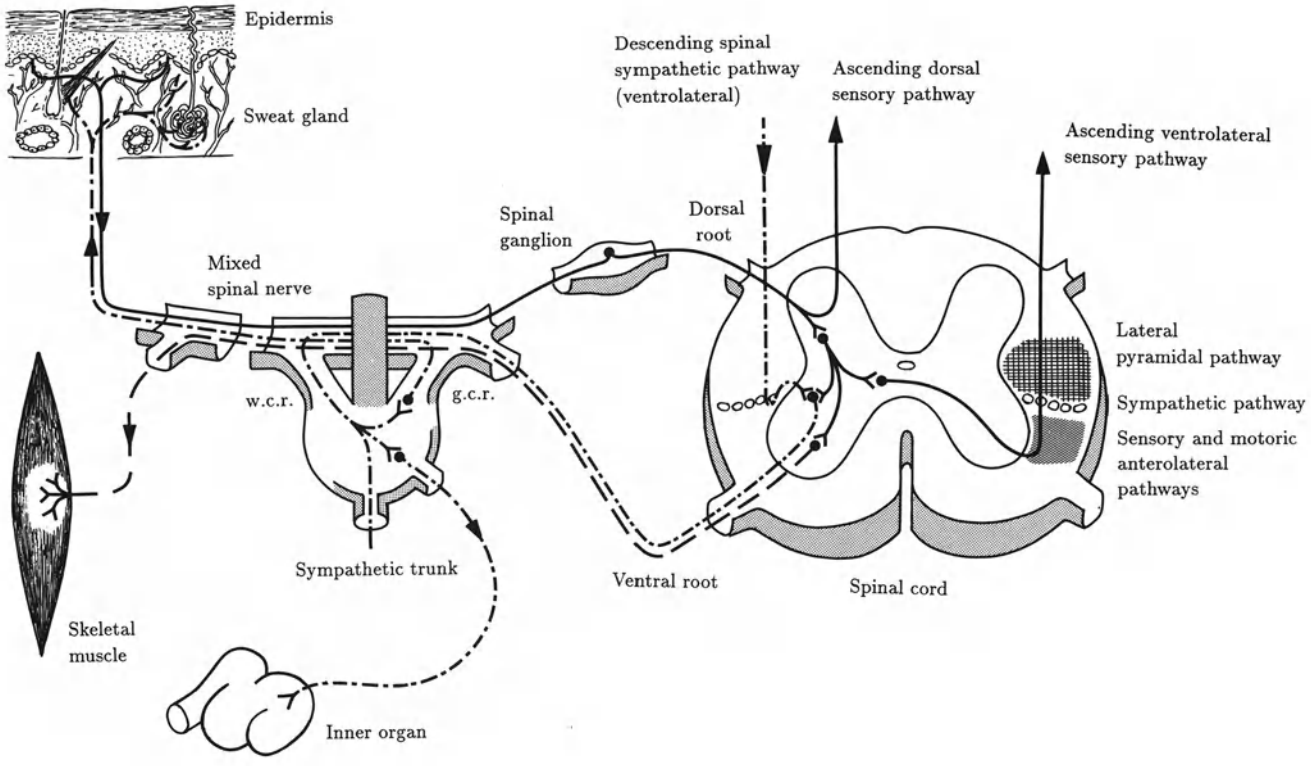

Figure 3. Travelling of skin afferents and efferents at spinal cord level, and their connections with ascending and descending pathway. – – –: motoric pathway to the skeletal muscle, – · –: sympathetic efferents, ——: skin afferents. w.c.r. = grey communicating ramus.

Verbov (1977) suggested that the adrenergic influence on these glands may be exerted via sympathetic nerve fibers ending near the blood vessels, and/or via freely circulating noradrenaline stemming from the adrenergic medulla. As a whole, the contribution of adrenergic stimulation to sweat secretion is poorly understood. In any case, its action on eccrine sweat glands plays a minor role as compared to the role of cholinergic innervation (Millington & Wilkinson, 1983; Sato, 1983).

1.3.2.1 Peripheral aspects of sweat gland innervation

As already mentioned, the preganglionic sudorisecretory neurons travel into the periphery, together with the other sympathetic nerve fibers, going from the lateral horn of the spinal cord ipsilaterally via the sympathetic trunk, in which they are switched to the postganglionic neurons. According to Jänig et al. (1983), their transmission velocity is 1.2–1.4 m/sec.

The cell bodies of the preganglionic sympathetic neurons are not present in all segments of the spinal cord, only from C8–L2. The boundaries must constrict for the origins of sudorisecretory efferents in the cord, since no important sudorisecretory fibers leave rostral to T3. Thus the sudorisecretory innervation of dermatomes is deviant from their sensory innervation (Table 3).

As can be inferred from Table 3, it is not always possible to establish an unambiguous correspondence between the sudorisecretory cell bodies within specific segments of the cord and certain dermatomes, which is also due to the distribution of neuronal activity by collaterals to the different levels of the sympathetic trunk (Sect. 1.3.1).

However, even the correspondences outlined in Table 3 have to be regarded with caution. They are mainly based on observations made in patients with lesions of the sympathetic trunk (Sect. 3.5.2.2). It is not easy to describe or produce precisely localized lesions in sympathetic pathways, because often collaterals often are not completely degenerated and therefore remain able to transmit neuronal activity. Additionally, it is presumed that microscopically small single cells and cell units in the neighborhood of autonomic nerve fibers, located between the sympathetic trunk and the periphery, may serve as relay stations of the sympathetic system. They may even function as ganglia, which would serve as an explanation of the sometimes surprising residual sweat gland activity after sympathectomy (Schliack & Schiffter, 1979). More recently established microneurographical methods (Wallin, 1981) could be used to prove a close relationship between the discharge amplitude of the sympathetic part of the median nerve, which innervates part of the palms, and the appropriate SRR amp. in human subjects (Sect. 1.3.2.2).

The secretory part of the sweat gland is surrounded by a very dense plexus of sympathetic fibers which allows a wide distribution of ANS activity. It is not yet fully clear how the cholinergic transmission of nerve impulses to the sweat gland cells is made. It has not been possible to establish real synaptic clefts, nor have nerve endings been observed to penetrate into the secretory cells. Ellis (1968) suggested these cells were

Table 3. Correspondence of the sudorisecretory fibers, leaving the ventral roots of the spinal cord, to sensory dermatomes, according to Klaschka (1979). C = cervical, Th = thoracic, L = lumbar, and S = sacral segments.

Anterior root of spinal nerve	Dermatomes of skin which are influenced by the appropriate sudorisecretory neurons
Th 3 – 4	Trigeminus region und C 2 – C 4
Th 5 – 7	C 5 – Th 9
Th 8	Th 5 – L 11
Th 9	Th 6 – L 1
Th 10	Th 7 – L 5
Th 11	Th 9 – S 5
Th 12	Th 10 – S 5
L 1	Th 11 – S 5
L 2	Th 12 – S 5

being stimulated via neurohumoral substances poured out by nerve endings nearby. Presumably it is the transmitter itself which empties from sympathetic nerve endings into the immediate neighborhood of the secretory cells. Edelberg (1967) pointed to the dependency of acetylcholine's transportation velocity upon body temperature together with the temperature dependency of the EDR lat. (Sect. 2.4.2.1), stating that 25 to 50% of the latency is due to the mechanism of acetylcholine transportation.

1.3.2.2 Central aspects of sweat gland innervation

In the sympathetic pathways, forming a smooth bundle between the lateral pyramidal tract and the anterolateral tract (Fig. 3), the sudorisecretory fibers, which end at the preganglionic sudorisecretory neurons, are in close proximity with other sympathetic fibers (e.g., those for vasomotor or pupilomotor efferences). Schliack and Schiffter (1979) suggested that there is no compact pathway for sweat gland activity. Instead, the sudorisecretory fibers are diffusely mixed with the surrounding sympathetic fibers.

At the spinal cord level, the sudorisecretory fibers are in close contact with afferent pathways, located in the anterolateral tract and belonging to the nonspecific (extralemniscal) somatosensory system. These are regarded as classical pathways for the perception of temperature as well as pain, contributing to the affective tone of perception, to the degree of consciousness, and to the elicitation of the defensive response (Sect. 3.1.1.2). Though there are no direct reports of synaptic connections of thermosensitive afferents with sudorisecretory efferents in the spinal cord, influences of somatosensory afferents on vegetative efferents at the spinal level are generally well known.

The representation of the sympathetic system is widespread and manifold throughout the brain. However, the hypothalamus is regarded as the controlling center of all

vegetative functions, including those for vasomotor activity and sweat secretion. Electrical stimulation in the "sympathetic" (ergotropic) hypothalamic area, especially in the paraventricular and posterior nuclei, is always followed by sympathetic reactions such as vasoconstriction, piloerection (Sect. 1.2.4), and sweat secretion. Since knowledge of the neuronal organization in this phylogenetically primordial structure is still incomplete, the functioning of hypothalamic elicitation of sweat gland activity cannot be pursued in detail.

Figure 4 shows the origin and course of the most important descending sympathetic pathway from the hypothalamus to the spinal cord. It originates in the weakly myelinated part of the hypothalamus (shaded in Fig. 4) and, according to results available up to now, goes via the tegmentum and ventrolateral reticular formation (RF) to the lateral spinal sympathetic tract (mentioned above). For a long time there has been controversy as to whether the course of this hypothalamic-reticular-spinal sympathetic pathway is ipsilateral or, partly or wholly, contralateral. However, Schliack and Schiffter (1979) were convinced that its course is mainly ipsilateral (Fig. 6, Sect. 1.3.4.1).

Hypothalamic sympathetic activity can be elicited or modified by higher-level cerebral structures. Various influences on the thermoregulatory hypothalamic functions are controlled from the limbic system, especially from the amygdala and the hippocampus (Edelberg, 1973a). There is also close proximity of the so-called Papez circuit (Papez, 1937) with the nuclei in which the hypothalamic-reticular-spinal sympathetic pathway originates (Fig. 4). Additionally, other structures like the ventro-oral internal thalamic nucleus, extrapyramidal nuclei, and various cortical areas have connections to the hypothalamus. As a result of stimulation and lesion experiments, the basal ganglia (striatum and pallidum), the thalamus, and the Brodmann area 6 of the cortical temporal lobe, which is adjacent to the precentral motor area, can be regarded as taking part in eliciting sweat gland activity (Schliack & Schiffter, 1979). The pathways stemming from these structures, however, cross in the medulla to the contralateral side before they reach the spinal sympathetic pathways (Fig. 6).

All existing findings concerning the central innervation of sweat gland activity point to several centers of sweat origin, which are at different levels of the CNS, and are partly independent of one another. Sympathetic activity can be elicited from the cortex, the basal ganglia, diencephalic structures like the thalamus and the hypothalamus, the limbic system, and brain stem areas. Accordingly, there are not only specific cerebro-efferent pathways to the sudorisecretory neurons, which reach the spinal cord either directly or after synaptic transmission, but also nerve fibers connecting the various areas with one another taking part in the elicitation of sweat (Fig. 5).

In spite of the existence of other cerebro-efferent pathways to the sudorisecretory neurons in the spinal cord, the hypothalamus should be regarded as the main region regulating sweat secretion (Schiffter & Pohl, 1972; Schliack & Schiffter, 1979). The hypothalamus is also the main thermoregulatory center. Therefore, an older view provided by Kuno (1956) restricted hypothalamic elicitation of sweat secretion to thermoregulatory sweating, which appears on the whole body surface except for the palms

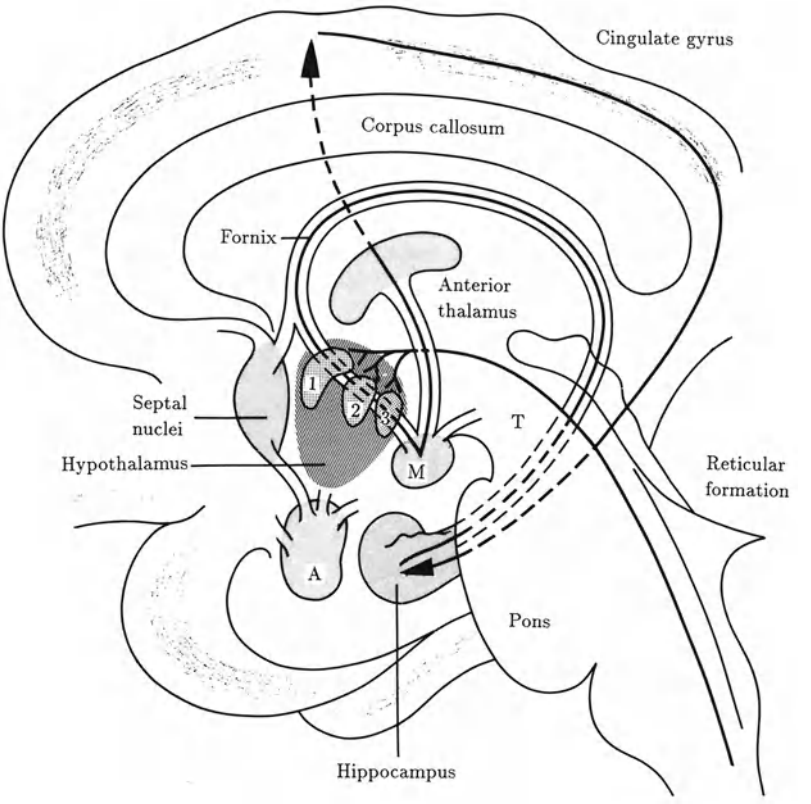

Figure 4. The limbic system in medial section. The hypothalamic-reticular-spinal sympathetic pathway stems from the paraventricular (1), posterior (2), and supramammillary (3) nuclei of the hypothalamus (weakly myelinated part). The Papez circuit (which is partly dashed because of its spatial course) goes from the hippocampus via the fornix to the mammillary body (M), to the anterior thalamus, to the cingulate gyrus, and back to the hippocampus. T: Tegmental midbrain area; A: Amygdala. Adapted from H. Schliack and R. Schiffter (1979), Neurophysiologie und Pathophysiologie der Schweißsekretion, Fig. 4d. In E. Schwarz, H. W. Spier, and G. Stüttgen (Eds.), *Handbuch der Haut- und Geschlechtskrankheiten*, Vol. 1/4A. Copyright ©1979 by Springer. Used by permission of the publisher and the first author.

and the soles (Sect. 1.3.2.4). In contrast, so-called emotional sweating (Sect. 1.3.3.3) which appears on palmar, plantar, and axillary areas, had been regarded by Kuno as being mostly under cortical control. Critical to this position is the influence of limbic structures on hypothalamic sweating as outlined by Schliack and Schiffter (1979) which probably also contributes to emotional sweating. The possibility of different CNS influences on sweat gland activity which are in part independent from one another will be also discussed with respect to CNS elicitation of electrodermal phenomena in Section 1.3.4.1.

1.3.2.3 Questions of double innervation and resting activity in sweat glands

Present understanding leads to the conclusion that sweat glands receive only excitatory sympathetic nerve impulses. As outlined by Wang (1964), inhibitory effects on EDA are supposedly interconnected with excitatory effects already present at the CNS level. In the past, an additional peripherally inhibitory parasympathetic innervation of sweat glands had been discussed (Braus & Elze, 1960). In particular, the active reabsorption of sweat in the ducts (Sect. 1.3.3.1) was assumed to be controlled via parasympathetic fibers that reach the peripheral cutaneous nerve from the spinal cord's dorsal root. This, along with the existence of vasodilatatory neuronal influences on the skin's blood vessels (Sect. 1.3.1), could never be proved. There is no need to assume the existence of a sweat-inhibiting innervation, since in the absence of sudorisecretory impulses the sweat normally vaporizes so quickly that additional sweat inhibition would not reduce the amount of sweat significantly.

Whether the stimulation of the myoepithelia around the ductal walls is mediated by freely circulating transmitters, or whether it requires a separate orthosympathetic innervation stemming from the sympathetic trunk, is yet to be resolved. Adrenergic transmission to the myoepithelia, formerly assumed to be responsible for the sweat secretion stimulated by adrenaline or noradrenaline, is no longer believed likely (Sect. 1.3.2). However, a possible cholinergic parasympathetic innervation of the ductal walls or, alternatively, the possible action of freely circulating acetylcholine is discussed (Sect. 1.3.4.2).

The problem of the sweat gland's resting activity still remains unresolved. Schliack and Schiffter (1979) regarded spontaneous sweating as an expression of an appropriate resting tonus (Sect. 1.3.3.3), whereas Jänig et al. (1983) could not find spontaneous sweat gland activity, at least at temperatures below the thermoregulatory neutral zone (Sect. 1.3.3.2).

1.3.2.4 Specific innervations of sweat glands in different regions of the skin

Regional specificities in the innervation of sweat glands are found especially in the face as well as at palmar and plantar sites, the latter sites also being morphologically different from the rest of the body's skin (Sect. 1.2.1 & 1.2.2).

The sweat gland innervation in the face, where measurement of EDA is uncommon, has been thoroughly investigated. In contrast to all other regions of the skin, there is a dual path of distal sympathetic efferent fibers to the region innervated by the trigeminal nerve. This is indicated by the remission of sweat gland activity in this region after an irreversible trigeminal lesion.[12] Since the trigeminus is a cranial nerve and not a spinal nerve, the sudorisecretory fibers originating in the cranial sympathetic trunk leading to the trigeminal nerve (Sect. 1.3.2.1) first have to follow the common carotid artery. Subsequently, the main part of sudorisecretory neurons follows the internal cartoid to the trigeminal branches, while the rest directly follows the external cartoid to the facial sweat glands.

Facial sweat secretion is especially active on the upper and lower lips, the bridge of the nose, the nasolabial folds, and the forehead, where it is most pronounced on the frontal bald area. Above the radix of the nose there is sometimes a small circular region in the medial line which is totally anhydrous. There has been much discussion of trigeminal fibers having the ability to inhibit sweat. These fibers are supposedly independent of the sympathetic trunk. In addition, a second possible parasympathetic "bulbar" pathway within the facial nerve has been discussed as acting either excitatory or inhibitory on sweat secretion. However, both of these possibilities lack convincing evidence (Sect. 1.3.2.2).

Most interesting with respect to electrodermal recording is the innervation of sweat glands at palmar and plantar sites, which probably differs from innervation of sweat glands on the rest of the body. It is controversial whether the sweat glands at palmar and plantar sites take part in thermoregulatory perspiration at all (Kuno, 1956). Schliack and Schiffter (1979) reported that palms and soles remained dry while the rest of the body was vigorously sweating, for example, when they were held out of a hot bath. On the other hand, Jänig et al. (1983) reported that palms and soles take part in thermoregulatory sweating, however, only at high ambient temperatures. Wilcott (1963) observed palmar and plantar sweat production following thermal stimulation. In the case of psychological strain, palmar and plantar sweating may be observed together with vasoconstriction, which is paradoxical with respect to thermoregulation. This provides evidence that these sites are linked to emotional rather than thermoregulatory sweat gland activity, though other parts of the body also take part in emotional sweating (Sect. 1.3.3.3). However, Conklin (1951), in a study described in Section 2.4.1.1, could not find differential temperature dependency of SCL at the palms as compared with the wrist and the forehead.

There is also some evidence for at least two different kinds of sweat gland receptors at palmar sites, since emotional sweating is completely abolished by atropine blockade, while both adrenaline-induced and spontaneous palmar sweating remain unaffected (Millington & Wilkinson, 1983). The specialization of sweat gland activity at

[12] An appropriate double innervation may exist in the region of the mouth floor which corresponds to dermatome C3 (Table 3, Sect. 1.3.2.1).

palmar and plantar sites, together with the possibility of unigue innervation, has given rise to speculations concerning the biological significance of palmar and plantar sweat glands (Sect. 1.3.5) and stimulated psychophysiological investigations of the so-called palmar/dorsal effect (Sect. 3.1.1.2 & 3.4.1.1). Some authors discuss an intermediate position of palmar and plantar sweat glands between apocrine glands and the phylogenetically younger eccrine glands. The apocrine sweat glands (Sect. 1.2.3) play no role in EDA measurement, so idiosyncrasies in their innervation can be disregarded at this point.

1.3.3 Functions of sweat gland activity

While the preceding section focused on sweat gland innervation, the following sections will describe the functional aspects of sweat gland activity. These are the mechanisms of sweat production and the embedding of sweating in thermoregulatory activity as well as in other functional relationships. An extensive review of the regulation of sweat secretion and the mechanism of sweat production have been provided by Fowles (1986a, pp. 62–72).

1.3.3.1 Mechanism of sweat secretion and contents of sweat

The secretory segment of the sweat gland, which is surrounded by a layer of myoepithelial cells, consists of clear and dark secretory cells (Sect. 1.2.3). The clear cells produce the liquid part of secretion, while the mucin produced by the dark cells may have protecting functions within the lumen. According to Ellis (1968), the serous cells act like a filter through which water and specific ions pass from plasma into the lumen. Since the so-called precursor sweat in the secretory segment of the sweat gland is slightly more hypertonic than blood, Fowles (1974) suggested an active transport mechanism for sodium chloride from the interstitial fluid into the lumen, producing an osmotic gradient which the water follows.

The human precursor sweat contains approximately 147–151 mM Na, 123–124 mM Cl, 5 mM K, probably 10–15 mM bicarbonate, and 15–20 mM lactic anion (Sato, 1977, p. 103). Sweat also contains small amounts of other ions and urea, as well as traces of biogenic amines and vitamins. The precursor sweat is modified as it passes through the ductal part of the sweat gland. In surface sweat, Na varies from 10–104 mM, paralleled by the Cl concentration ranging from 10–30 mM (Sato, 1977). These lower concentrations gave rise to the hypothesis of an active NaCl reabsorption mechanism in the ductal wall, comparable to the one in the renal tubulies, which is regarded as being well established (Fowles, 1986a). The NaCl concentration at the skin surface, which falls within the range of .015–.06 M (Rothman, 1954), becomes higher with an increased rate of perspiration, which presumably reflects a limited reabsorption capacity of the duct. Since the concentration has been reduced by the time the sweat reaches the epidermal duct, most of the reabsorption is likely to have taken place in the dermal

duct (Schulz, Ullrich, Frömter, Holzgreve, Frick, & Hegel, 1965); however, there is possibly an additional NaCl absorption mechanism in the epidermal ductal walls (Sect. 1.4.2.3, Fig. 14). The reabsorption as a whole may prevent the body from excessive NaCl loss through sweating in high ambient temperatures (Fowles, 1974).

NaCl can be regarded as playing a mediator function in sweat production: it is actively transported into the lumen within the secretory segment of the sweat gland to produce an osmotic gradient, which draws water into the lumen, and is subsequently actively reabsorbed within the duct.[13]

Sweat does not flow continuously through the duct to the skin surface, but rather in a pulsatile manner with pulsations of 12–21 Hz, as Nicolaidis and Sivadjian (1972) showed by applying a fast-moving humidity sensitive film. Rhythmic contractions of the myoepithelia, surrounding not only the secretory but also the ductal part of the sweat gland like a helix, are regarded as the source of the pulse. An adrenergic influence on these myoepithelia has been discussed. However, they might be innervated cholinergically as well, since adrenergic receptors play a minor role, if any, in eccrine sweat secretion (Sect. 1.3.2).[14] According to Sato (1977), sweat can be observed rising and falling in a micropipette brought into the lumen at a frequency of .5–2 Hz during low sweating rate. He followed Kuno (1956) in arguing that this phenomenon should be due to neural stimulation rather than to myoepithelial contraction. However, the nature of an appropriate innervation remains unclear (Sect. 1.3.2.3).

1.3.3.2 Thermoregulatory function of sweating and skin blood flow

Vaporization from the skin is mainly considered with respect to its thermoregulatory function. It is divided into visible, or so-called sensible perspiration, and invisible, or so-called insensible perspiration.

The thermoregulatory functions of vaporization from the skin and of blood flow in the skin are closely related to each other. With respect to thermoregulation in the so-called neutral zone (28–30^{0}C, 50% relative humidity and calm wind, for an unclothed resting adult), heat loss can be controlled solely by vasomotor activities. Below this zone, water vaporizes through insensible perspiration (Thews, Mutschler, & Vaupel, 1985). Visible thermoregulatory sweating appears above the neutral zone, and water loss through sweating reaches a significant amount above 34^{0}C (Thiele, 1981a).

One-half to two-thirds of the total insensible water loss is through the skin, while the rest is through the lungs, but the skin's portion is not all conducted by sweat glands,

[13]It is also important for cardiac functions related to blood pressure. The metabolic processes that take place during sweat secretion, including the possible role of Ca as a second messenger in cholinergic sweating, are discussed in detail by Sato (1977), and summed up by Fowles (1986a).

[14]Sato (1977) generally questions the hypothesis of expulsion of already-existing sweat by adrenergic stimulation. In his in vitro studies with isolated monkey sweat glands, he found so little preformed sweat in the lumen that an initial myoepithelial contraction could not expel an appreciable amount of sweat. Furthermore, Nicolaidis and Sivadjian (1972) used forehead sites for recording, so their observations may not be generalized to palmar sweat secretion.

since anhydrotics lose as much water as normals (Tregear, 1966). However, atropinization, which blocks the cholinergic sweat gland innervation reduces the amount of insensible perspiration to 50% (Herrmann et al., 1973).[15] Thus, a considerable part of insensible perspiration is controlled by sweat gland activity, using at least the whole peripheral apparatus including the efferents from the sympathetic ganglia (Schliack & Schiffter, 1979).

The regulation of skin blood flow depends on two different mechanisms that are represented regionally. The skin sites further from the trunk, such as palms, soles, and ears, are rich in sympathetic adrenergic fibers acting in a vasoconstrictory manner. They show strong tonic activity even under temperature-indifferent resting conditions. Peripheral vasodilatation is a consequence of inhibiting their activity. At the proximal parts of extremities, as well as at the trunk, the resting activity of those sympathetic adrenergic fibers is low, and its increase leads to vasoconstriction. In these areas, vasodilatation is a direct consequence of sweat gland activity, since bradykinin, which is released when sudorisecretory fibers are stimulated, has a strong vasodilatatory action upon the skin's capillaries. With regard to these mechanisms, Edelberg (1972a) considered the possibility of reflex sweating being nothing more than a hand-maiden of the cardiovascular system.

The efferent CNS control of the skin's thermoregulatory function does not only include the sympathetic part of the ANS; because voluntary as well as involuntary muscle activity takes part in thermal balance, the somatic sensory system is also included. The afferent limb is formed by peripheral thermoreceptors, mainly by receptors for cold and heat in the skin, and by a centrally located receptor which belongs anatomically to the medial hypothalamus, the firing rate of which is primarily determined by the internal body temperature (Kupfermann, 1985). Temperature control, including thermoregulatory sweating, is modulated by hypothalamic structures (Sect. 1.3.2.2).

1.3.3.3 Other functions and more specific properties of perspiration

Besides the thermoregulatory sweating described in the previous section, Schliack and Schiffter (1979) gave evidence for an additional five kinds of sweating which are classified according to the stimuli eliciting them.[16] Every type uses the postganglionic sympathetic neuron, which has its origin in the sympathetic trunk and goes via the

[15] In deviation from the classical dermatological hypothesis, Tregear (1966) presumed that the sweat gland ducts, as well as the hair follicles, were of no importance for the amount of the body's water loss. He referred to observations that the palmar skin, having three times the density of sweat glands compared to the rest of the body, is not very permeable to fluids, and that persons without any sweat glands show as much insensible perspiration in a cool environment as normal persons.

[16] The specific meaning of these different kinds of sweating for EDA is as yet unexplored, except those of the so-called emotional-sweating type. They are reported here for the sake of completion and with respect to possible future electrodermal research.

peripheral cutaneous nerve to the sweat gland as a final common terminal path (Sect. 1.3.2.1). However, their mechanisms of CNS elicitation differ in part.

(1) "Emotional sweating" means increased sweat gland activity as a concomitant of psychological and, especially, emotional states which appear, for example, in high activation or under stress (Sect. 3.2.1). It is likely to be elicited via hypothalamic-limbic connections as described in Section 1.3.2.2. Emotional sweating is observed mainly on palmar and plantar sites (Sect. 1.3.2.4), but also in the axillary and genital regions (Millington & Wilkinson, 1983) as well as on the forehead (Schliack & Schiffter, 1979). However, Allen, Armstrong, and Roddie (1973) observed increased sweating at other body sites during emotional strain as induced by arithmetic exercises. They observed the amount of sweating as being directly proportional to the number of sweat glands per region, thus indicating no regional differences in emotional sweating. Shields et al. (1987) also considered the possibility of the specific reactivity of palmar and plantar sweating to psychological stimulation as being dependent on the greater sweat gland density on these sites. Thus the specifity of emotional sweating remains to be considered further.

(2) Gustatory sweating appears when food is consumed which is especially sour, highly salted, or spicy. There are marked interindividual differences with respect to the sites included. Gustatory sweating mainly appears on the face (e.g., on the forehead and the upper lip; Schliack & Schiffter, 1979), and on the wings or the top of the nose. Its intensity can be irritating without being pathological. However, pathological gustatory sweating may appear after sympathetic nerve lesions. It can also be elicited in the absence of gustatory stimuli – it does not require intact gustatory sensation – via chewing and olfactory as well as psychological stimulation. It is probably elicited via an irritation or partial blocking of sudorisecretory pathways from the sympathetic trunk or its peripheral ramifications, which lead to a local disinhibition of an otherwise subliminal physiological reflex (Schiffter & Schliack, 1968).

(3) Ubiquitous spontaneous sweating can be observed even by mere magnified observation on palmar and plantar sites. It may be regarded as an expression of a resting tonus, comparable to the resting muscle tonus of motor units. However, the existence of such a resting tonus in sweat glands remains debatable (Sect. 1.3.2.3).

(4) Reflex sweating is an expression that describes sweat gland activity at sites which are innervated from spinal cord segments (see Table 3, Sect. 1.3.2.1) distal to the locus of a certain damage (e.g., a paraplegia). The expression is also used for a confined, local sweating following stimulation of an area with radiation, heat, needle punctures, or electricity. It is assumed to be mediated through so-

called axon reflexes (Sect. 1.3.1) instead of possible spinal connections between sympathico-efferents and pain afferents.

(5) Pharmacologically produced sweating is a local sweat secretion elicited through either subcutaneous or intracutaneous injection, as well as through iontophoresis with cholinergic substances (e.g., nicotine or pilocarpine).

There is also a special mechanism of sweating which underlies the so-called cold sweat. Startle reaction (Sect. 3.1.1.2) and other strong emotionally tinted responses, as well as deep breathing and coughs – all situations associated with a sudden elicitation of adrenaline – also lead to sweat secretion. Free-circulating adrenaline, however, acts in a vasoconstrictory manner similar to a cold stimulus. At the same time, sweat secretion may be activated via hypothalamic centers. Both reactions result in cold sweat, which seems to be paradoxical with respect to thermoregulation. However, Ebbecke (1951) was convinced that expulsion of so-called cold sweat is not a result of the secretory part of the sweat gland's secretory stimulation. Instead, he adopted an adrenergic stimulation of the myoepithelia around the duct as a source of expulsion of sweat already produced in this case, a hypothesis also held by Kuno (1956) but which is no longer regarded as valid (Sect. 1.3.2.& 1.3.3.1).

1.3.4 Specific physiological mechanisms underlying electrodermal activity

The following two sections investigate CNS mechanisms (Sect. 1.3.4.1) and epidermal as well as sweat gland features (Sect. 1.3.4.2) with respect to their contributions to electrodermal phenomena. These descriptions must be in part speculative, since the central origin of sweat gland activity especially is not understood in detail (Sect. 1.3.2.2). An additional problem is that most findings are taken from animal preparetions, and generalization to humans cannot be easily made (see Footnote 9, Sect. 1.2.3).

1.3.4.1 Central origins of electrodermal activity

Electrodermal phenomena are not only influenced by parts of the CNS that are involved in the classical sympathetic elicitation of sweat secretion (Sect. 1.3.2.2); various subcortical and cortical regions are involved as well, forming a complex system, the role of which, however, is far from being fully understood. A thorough review of the evidence with regard to different brain levels has been provided by Venables and Christie (1973). The present author will add some recent results and provide an integrative view on the origin of both tonic and phasic electrodermal phenomena in the CNS.

The hypothalamic areas that exert thermoregulatory control can also be considered to play a major role in the elicitation of EDA (Fig. 4, Sect. 1.3.2.2). The finding that EDRs could be elicited in some animal preparations by stimulation of anterior hypothalamic regions (for summaries see Edelberg, 1972a; Venables & Christie, 1973), which

are regarded as belonging to the trophotropic (parasympathetic) system, gave rise to the hypothesis that at least part of CNS elicitation of EDA could be under parasympathetic control (Venables & Christie, 1973, p. 30). However, as Bard (1960) pointed out, no mechanism related to the parasympathetic system exists in the hypothalamus or in any other part of the CNS that parallels the representation of the sympathetic system. Furthermore, evidence for influences of anterior hypothalamic regions on sweat gland activity in humans is lacking (Sect. 1.3.2.2).

Much of what is known about the central origins of electrodermal phenomena stems from studying the effects of lesions and stimulations in the cat's brain, while evidence from humans and from nonhuman primates is sparse. Figure 5 outlines the knowledge as of 1964 about the excitatory and inhibitory sweat centers from which the cat's electrodermal activity originates as described by Wang (1964), summarizing results of various studies with anesthetized and unanesthetized animals. Excitatory centers for the origination of EDA are located in the cat's anterior hypothalamus, in the dorsal thalamic region, and in anterior limbic and infralimbic areas (Isamat, 1961) as well as in sensorimotor cortical areas (see arrows in the left part of Fig. 5). The midbrain does not seem to play an important role with respect to excitatory control of EDA, although its descending sudorisecretory pathways are regarded as being well established in the cat. The ventromedial part of the RF at the rhombencephalic level is regarded as the most powerful inhibitory center for EDA in the cat's brain. The roof nuclei in the anterior lobe of the cerebellum only inhibit autonomic functions during strong muscular movement. Accordingly, removal of the cerebellum has no observable effect on EDRs of cats. At the striatal level, an important inhibitory center for EDA seems to be located in the caudate nucleus. Additionally, inhibitory but also excitatory areas with respect to EDA were found in the frontal cerebral cortex (e.g., Wang & Lu, 1930; Wilcott, 1969; Wilcott & Bradley, 1970; see also Langworthy & Richter, 1930). The abovementioned possible inhibitory center for EDA in the hippocampus was omitted from the figure by Wang (1964), because he could not form any simple notion concerning its descending pathway. Since synchronization of spontaneous SPRs in the cat's four footpads was present in the striatal but not in the hypothalamic cat, Wang assumed a regulatory center for EDA located in some part of the pallidum (indicated in Fig. 5 by interconnections of this region with all origins above the rhombencephalic level).

The limbic system, which is regarded as the neurophysiological basis for emotional phenomena and partly for motivational phenomena (Sect. 3.2.1.2 & 3.2.2.1), can be regarded as directly influencing sudorisecretory hypothalamic areas. These influences mainly stem from the hippocampus (which is included in the Papez circuit) and from the amygdala (Edelberg, 1972a; see also Sect. 3.4.2). Some animal research points to antagonistic actions of these structures on EDA (Yokota, Sato, & Fujimori, 1963; Yokota & Fujimori, 1964), that is, excitatory influences from the amygdala and inhibitory influences from the hippocampus, the latter being supposed by Wang (1964) to stem from the mammillary body via the fornix. Stimulation of the basolateral amygdala can also evoke a single EDR with its typical recovery in lightly anesthetized cats (Lang, Tuovi-

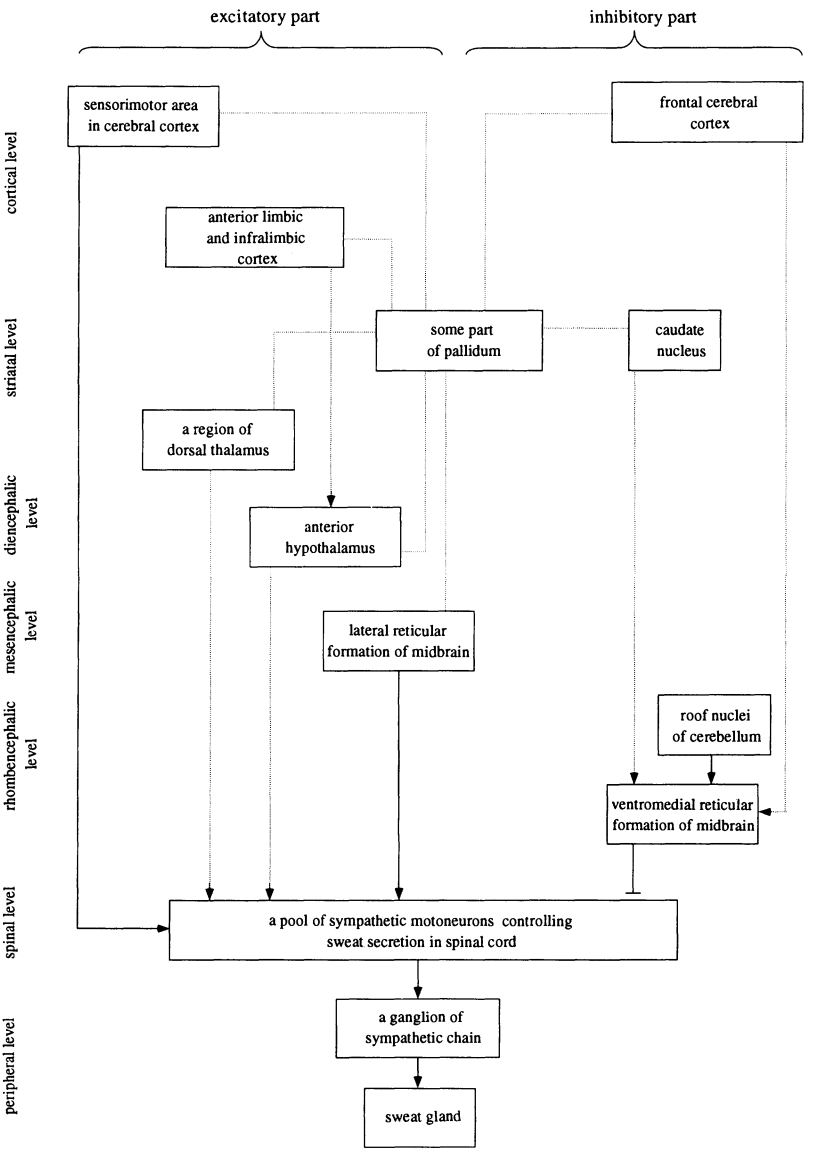

Figure 5. Block diagram showing sweat centers and their efferent pathways to sweat glands in the cat. ————: Tract known; ·····: Tract unknown. Adapted from G. H. Wang (1964), *The neural control of sweating*, Fig. 8.1. Copyright ©1964 by the Regents of the University of Wisconsin. Used by permission of the publisher.

nen, & Valleala, 1964), and amygdalectomy in monkeys produced a marked impairment of EDRs following acoustic stimulation (Bagshaw, Kimble, & Pribram, 1965), which gave rise to the hypothesis that the amygdala is responsible for the elicitation of the electrodermal orienting response (Sect. 3.1.1.1).[17] On the other hand, if amygdaloid after-discharge occurs following this stimulation, the EDR maintains a peak for a considerable amount of time consisting of a series of small flutterlike waves, as shown by Lang et al. (1964) with cats.

Cortical areas that may interact with the limbic system were found to influence EDA in humans and in nonhuman primates as well. Skin conductance responses were diminished in monkeys with lateral frontal lesions (Bagshaw et al., 1965), and were completely absent in monkeys with bilateral removal of the dorsolateral cortex (Grueninger, Kimble, Grueninger, & Levine, 1965). Luria and Homskaya (1970) observed reduced electrodermal orienting responses in patients with frontal lesions as compared to those with lesions in other cortical areas. Recently, Raine, Reynolds and Sheard (1991) used a magnetic resonance brain imaging technique relating individual differences in the size of selected brain areas to electrodermal phenomena in normal human subjects. They found a strong relationship between spontaneous and elicited EDA and the prefrontal area measure. They also found for the left temporal area (including the amygdala) – and not for the right temporal area – significant correlations to electrodermal orienting responses (Sect. 3.1.3.1). No relationships were observed between EDA and nonfrontal cortical areas. At the brain stem level, the pons area was related to electrodermal orienting while the cerebellum was not.

There is also strong evidence for cerebral influences on EDA from regions outside the limbic system and related cortical areas. As mentioned already, Wang (1964), in his lesion experiments with nonanesthetized cats, found that the synchronization of spontaneous SPRs in all four paws was controlled by some part of the pallidum which belongs to the basal ganglia, and not by hypothalamic structures. Earlier, Langworthy and Richter (1930) as well as Spiegel and Hunsicker (1936) emphasized the role of premotor cortical regions (Brodmann area 6, see Fig. 6) in eliciting EDA, since a close connection between the pyramidal fibers for the transmission of skeletal muscle impulses and sudorisecretory fibers has been found in degeneration studies. As Darrow (1937a) pointed out, those pathways cannot be identical, because pyramidal stimulations did not elicit reactions in the skin.[18] He suggested that the sudorisecretory fibers were corticopontine, rather than corticospinal like the pyramidal fibers. Thus, the neurophysiological basis for electrodermal changes which accompany changes in posture should be influenced from tegmental or pontine areas in which the premotoric fibers end. Since it is now widely accepted that subcortical structures such as the basal

[17]This had been recently questioned by Tranel and Damasio (1989) who obtained regular electrodermal orienting responses in a patient whose entire amygdaloid complex had been destroyed bilaterally.

[18]However, Langworthy and Richter (1930) could elicit EDRs and other autonomic responses by stimulating the pyramidal tract in cats. Roy et. al. (1984) suggested this was due to the collaterals from the pyramidal tract reaching the RF, by which reticular elicitation of EDA has been mediated.

ganglia participate in motoric integration or programming (Marsden, 1982), the combined striatal and premotor cortical origins of EDA can be viewed together as a single premotor electrodermal component (Fig. 6).

When premotor cortical areas are electrically stimulated, or are removed, excessive sweating is observed. This might be explained by postulating excitatory as well as inhibitory cortical sudorisecretory influences. In their animal studies Wang & Brown (1956) were able to demonstrate inhibitory influences from large frontal cortical areas acting on excitatory cortical sweating centers of the sensorimotor and the anterior limbic cortex.[19] The role of EDRs as concomitants of motor reactions is well established in human subjects (Edelberg & Wright, 1964; Pugh, Oldroyd, Ray, & Clark, 1966). Bilateral electrodermal recordings at palmar sites following strong acoustic stimuli sometimes showed noticeable lateral differences, which, however, never exceeded a ratio of 1 : 1.5 (Fisher, 1958; Obrist, 1963). However, if subjects were asked to move one foot as a reaction to the acoustic stimulation, the lateralization increased in favor of the EDR amp. at the ipsilateral hand (Culp & Edelberg, 1966).

Taking all these human as well as animal results together, two main different cerebral sources of EDA may exist. First, there are ipsilateral hypothalamic influences on sweat secretion that are controlled by limbic structures (Fig. 4) with facilitatory influences stemming mainly from the amygdala (e.g., in the case of orienting and defensive reactions) and inhibitory influences stemming mainly from the hippocampus (e.g., in the case of behavioral inhibition, see Sect. 3.2.1.2). The basal ganglia together with premotor cortical areas form a second system with separate and mainly contralateral influences on sweat secretion and hence on EDA (Sect. 1.3.2.2). These influences were not only found in animal lesion studies, but also in stimulation and lesion studies in humans (Schliack & Schiffter, 1979). The limbic-hypothalamic electrodermal source is labelled here as "EDA 1," and the premotor source as "EDA 2" (sources 1 & 2 in Fig. 6; see also Fig. 48, Sect. 3.2.1.2).

However, the laterality of EDA may disappear at the reticular level and/or below, since unilateral cortical and pyramidal stimulation in the cat elicited bilateral SPRs with comparable amplitudes (Sequeira-Martinho, Roy, & Ba-M'hamed, 1986) and reticular stimulation in the cat was always followed by bilateral responses (Ba-M'hamed-Bennis, Sequeira-Martinho, Freixa i Baqué, & Roy, 1985). The RF itself can have eliciting as well as modulating influences on EDA (Roy, Sequeira-Martinho, & Brochard, 1984). Bloch (1965) pointed to EDA as a reflection of central activation which is controlled by excitatory as well as inhibitory areas in the RF. These areas are regarded as mainly influenced by inhibitory corticofugal neurons. An inhibitory reticular center is located in the ventromedial RF (Wang & Brown, 1956; Roy, Delerm, & Granger, 1974), whereas the lateral portion of the midbrain RF and portions of the diencephalic RF have excitatory effects on EDA (Bloch, 1965; Edelberg, 1972a). Venables and Christie (1973)

[19] In general, the question of ipsi- or contralaterality of inhibitory vs. excitatory influences on electrodermal phenomena remains unresolved (Sect. 3.1.3.4).

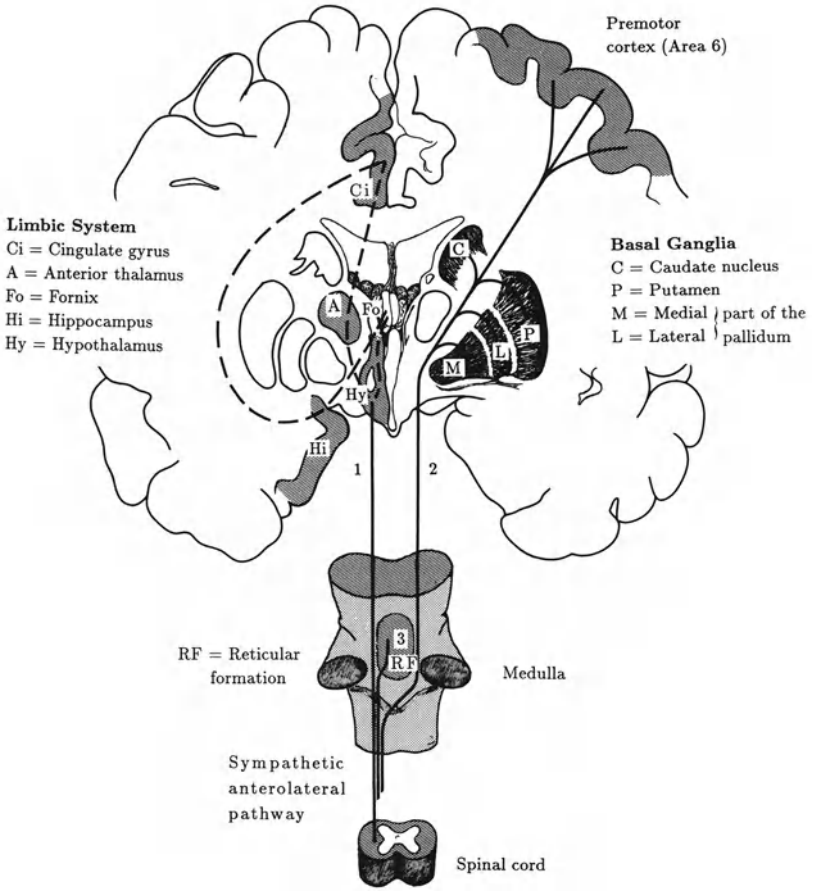

Figure 6. Central elicitation of EDA in humans. 1: Ipsilateral influences from the limbic system via hypothalamic thermoregulatory areas (EDA 1); 2: Contralateral influences from premotor cortical and basal ganglia areas (EDA 2); 3: Reticular influences. Dashed: Connections within the limbic system (Fig. 4, Sect. 1.3.2.2). Adapted from H. Schliack and R. Schiffter (1979), Neurophysiologie und Pathophysiologie der Schweißsekretion, Fig. 4c. In E. Schwarz, H. W. Spier, and G. Stüttgen (Eds.), *Handbuch der Haut- und Geschlechtskrankheiten*, Vol. 1/4A. Copyright ©1979 by Springer. Used by permission of the publisher and the first author.

provided evidence that stimulation of these mesencephalic excitatory regions facilitates motor activity via the action of the RAS (Sect. 3.2.1.1). Because the RF is connected with the striopallidum as well as the cerebellum, and is known to strongly influence skeletal muscle tone as well as muscle contractions via the gamma-efferent system, there is likely to be a close connection between the reticular modulation of sweat gland activity and skeletal muscle activity (Roberts & Young, 1971). Thus, while influences on EDA stemming from EDA 2 have to be regarded as concomitants of preparing to activate distinct motor untits, reticular influences on EDA (source 3 in Fig. 6) are more likely to be connected with a general increased muscular tone due to an increased general arousal (Footnote 192, Sect. 3.2.1.1). Reticular-mediated EDRs should likely be concomitants of locomotive changes, which may appear in emergency situations, and not of distinct or even fine manipulative motor actions which require a stronger cortical participation. Whether electrodermal changes that appear as concomitants of inhalation, and which are mainly regarded as artifacts in electrodermal recording (Sect. 2.2.5.2), are more cortically or more reticularly influenced remains unanswered.

In summary, the experimental as well as clinical evidence concerning the CNS elicitation of EDA points to the existence of two different origins above reticular level, which were already suggested by Edelberg (1972a): a limbic-hypothalamic source labelled EDA 1, being thermoregulatory and also emotionally influenced, and a premotor-basal ganglia source labelled EDA 2, eliciting electrodermal concomitants of the preparation of specific motor actions. In addition, there may be a reticular modulating system which mediates EDA changes that appear with variations of general arousal (source 3 in Fig. 6). The reticular modulating system is also likely to be responsible for inhibitory influences on EDA (Fig. 5), which may be either ipsi- or contralateral. However, under conditions of diffuse sweat gland activation with generalized EDA, the specifity of those neuronal systems may at least partly disappear.[20]

1.3.4.2 Properties of skin and sweat glands influencing electrodermal activity

While the dermis and also the subcutis are well supplied with blood and interstitial fluid, at least the upper epidermal layers consist of relatively dry and horny cell structures which are not necessarily surrounded by much fluid. Hence, both kinds of skin structure will have different electrical properties, which is of great importance for EDA. The intact skin shows little permeability for water and soluble agents, which is also seen in epidermal tissue taken apart from skin. On the other hand, skin from which the epidermis has been removed shows very high permeability (Tregear, 1966). Therefore, an epidermal barrier layer is assumed, which is penetrated by skin appendages (Sect. 1.3.4.2.2). These structures show not only resistive but also membrane-dependent polarization properties.

[20]Wilcott (1963) showed that nonpalmar areas of skin that are normally regarded as thermoregulatory also took part in emotional sweating during a stressful situation (Sect. 3.2.2.2).

1.3.4.2.1 The role of the epidermal barrier layer. There has been much discussion concerning the localization of an epidermal diffusional barrier, which is reviewed in detail by Fowles (1986a). Though it is not possible to give an exact localization of this barrier (Thiele, 1981b), there are some researchers who suggest a gradient of increasing resistance from the outer to the inner parts of the horny layer (e.g., Montagna & Parakkal, 1974), pointing to the inner stratum corneum as the main portion of the barrier layer (Table 2, Sect. 1.2.1.1). However, most findings provide evidence that the entire stratum corneum forms the barrier, with the exception of its desquamating surface cells (Jarrett, 1980).

Attempts to localize this barrier layer were mainly performed using the so-called stripping technique. With this technique, epidermal layers down to the stratum lucidum are successively pulled off by means of Pliofilm, which makes 4–40 successive trials necessary, depending on the method used (Klaschka, 1979). With the stripping technique, only the fully keratinized dry epidermal layers can be removed, because the Pliofilm does not adhere to the inner humid layers such as the stratum intermedium. It has been shown that after complete removal of the corneum, there remains only a low diffusional resistance (Tregear, 1966) and also a low electrical resistance in the epidermis (Lykken, 1968). However, since stripping also results in erythema (inflammation stemming from hyperemia), these results cannot exclude the existence of another barrier layer loacated in the deeper layers of the intact corneum (Tregear, 1966).

Since the stratum corneum consists of dead cell material, the nature of the epidermal barrier is likely to be that of a passive membrane. This was shown by in vitro experiments using epidermal preparations, which yielded the same estimates for permeability to water, electrolytes, nonelectrolytes of low molecular weight (such as alcohols), and steroids (Fowles, 1986a). The barrier properties stem from lipids and essential fatty acids like linoleic acid, and not from the keratin in the cells of the corneum. This is evident because strongly keratinized structures like nails are permeable to water, but delipidization of the corneum or diets deficient in essential fatty acids causes a marked increase in transepidermal diffusion of water.

A factor influencing epidermal barrier function under normal physiological conditions is skin temperature. The water permeability of skin increases exponentially with rising temperature. As Fowles (1986a) pointed out, the permeability for water doubles with an increase in skin temperature of 7–8 ^{0}C within the range of 25–39 ^{0}C. Since palmar skin temperature variations fall within this range, temperature's effects on water permeability may significantly influence EDA (Sect. 2.4.2.1).

In spite of this diffusional barrier, and independent of sweat gland activity, there is a continuous transmission of water, from the dermis via the epidermis, out of the body by insensible perspiration (Sect. 1.3.3.2). Thus, under physiological conditions the corneum, which is extremely hydrophilic, is always partially hydrated. Its hydration is, however, dependent on external as well as internal factors. There is a linear relationship between the increase of environmental relative humidity (Sect. 2.4.1.2) and corneal hydration up to humidities of about 60–70%. At higher levels, the increase of hydration is

exponential up to a relative humidity of 95% (Fowles, 1986a). Thiele (1981b) showed that the thickness of the corneum is halved when the air's relative humidity is halved.

Sweat gland activity in which the ducts are filled up to the epidermis, results also in corneal hydration. Since the acrosyringium has no wall cells of its own (Sect. 1.2.3), sweat penetrates unobstructed into the corneum as a result of high pressure in the duct and/or diffusion. If sweating rate increases, sweat pouring out onto the surface additionally increases corneal humidity from the outside. Using cats, the footpads of which were dried out, Adams (1966) showed that repeated stimulation of plantar nerves resulted in an outpouring of sweat after a certain temporal delay, which varied inversely with stimulation frequency. This shows that the corneum becomes hydrated first, before sweat reaches the surface. If the stratum corneum is already hydrated, an adequate nerve stimulation results in the appearance of visible sweat immediately.

Since sweat contains numerous ions (Sect. 1.3.3.1), the electrical conductance of the corneum increases when being soaked with sweat, thus being dependent on sweat gland activity (Sect. 1.4.2.1). However, as compared to the ducts filled with sweat, the stratum corneum provides a relatively weak conducting path. In their in vitro studies, Campbell, Kraning, Schibli, and Momii (1977) found an unequivocal relationship between the hydration of the plantar stratum corneum and its electrical resistance. According to Tregear (1966), it is uncertain which part of the corneum adds more to its electrical conductivity: the hydrated keratinocytes themselves or the fluid within the intercellular spaces. However, Fowles (1986a) argues that water-soluble molecules follow a transcellular route as a pathway through the barrier.

1.3.4.2.2 The role of skin components and of membranic processes. It is unlikely that the outlets of the sebaceous glands form shunt conductances through the stratum corneum as do the sweat gland ducts, since lipids act as electrical isolators. The same is true for hair, since their sites are always connected to sebacous glands (Sect. 1.4.2).

On the other hand, there is only little doubt (Footnote 15, Sect. 1.3.3.2) that the sweat glands form quantitatively important as pathways for water loss ("diffusional shunts") under some conditions and at some body sites (Fowles, 1986a). Scheuplein (1978) has produced a systematic theoretical treatment of this topic. Under steady state conditions, the greater cross-sectional area of the stratum corneum compared to the area covered by sweat glands (by a factor of 1,000–100,000) more than balances its lower diffusivity for water, so that the possible contribution of the diffusional shunt function of sweat glands may become insignificant. However, prior to attaining a steady state, their role as shunts may be quite important. This is because water loss occurs via the sweat glands sooner than it does through the corneum, and the time lag is more pronounced with an increasing thickness of the corneum (Scheuplein, 1978). Thus, at palmar and plantar sites, where the stratum corneum is especially thick (Sect. 1.2.1.1), water loss through the sweat glands should make a significant contribution to the total water loss for a much longer time than at other sides on the body (Fowles, 1986a). However, this effect may in part be canceled by the fact that the coefficient of diffusion (i.e., the rate

of penetration of a given solute through the tissue) is much greater on palms and soles than on other body sites (Scheuplein, 1978).

Unfortunately, appropriate comparisons were made only for various solutions but not for electrolytes by Scheuplein (1978), who stated that the permeability of the intact stratum corneum for electrolytes is extremely low. Edelberg (1971), on the other hand, provided some evidence that corneum, which is at least moderately hydrated, will allow ions to diffuse through it. He also suggested that the electrical current employed through skin during exosomatic electrodermal recording may facilitate epidermal diffusion.

Though the relative contributions of the epidermis and sweat gland ducts to total diffusion of electrolytes are unclear, the sweat gland ducts on palms and soles may play a more important role as electrical shunts than those on the rest of the body. So the possibility of site-specific time courses of the electrodermal signal on palmar and plantar sites, which are preferred for electrodermal recordings (Sect. 1.2.1.1), has to be considered.

With respect to its electrical resistance, the skin has an inner humid, conductive layer, formed by the dermis together with the nonhorny epidermal layers (Sect. 1.2.1), and an outer less humid layer which contains a barrier for water and ions and is therefore less conductive (Campbell et al., 1977). This barrier is broken by sweat gland ducts, which act as diffusional as well as electrical shunts.

Besides these purely resistive properties, living tissue also has capacitative or polarization features which stem from its active membranes (see Chapter 1.4). Living cell membranes, as in the noncorneal part of the skin, can build up polarization capacities (Sect. 1.4.2) via an active ion transportation mechanism through their semipermeable structures. These active membranes are also present in the wall cells of the sweat gland, and in the myoepithelial cells surrounding them. It is not likely that active membrane processes are present in the stratum corneum, since fully keratinized cells behave electrophysiologically like plant cells. Perhaps the membrane-like lines between corneal cells (Section 1.2.1.1) still act as contact zones for the transmission of action potentials (Klaschka, 1979), but they do not have any capability to form polarization capacities.

While tissue conductivity as discussed above is responsible for tonic EDA and perhaps contributes to phasic electrodermal phenomena with slow recovery, active membrane processes following a nerve impulse will be more likely to elicit EDRs with fast recovery. Polarization features of membranes may also be influenced by humoral factors. Various hormones like adrenaline, noradrenaline, and bradykinin act directly on biological membranes. Detailed descriptions of pharmacological actions of those substances on sweat glands in vitro are given by Sato (1977, 1983).

1.3.5 Suggested biological relevance of electrodermal phenomena

Apart from the psychophysiological relevance of EDA, which is the subject of the third part of this book, there have been several hypotheses concerning its biological significance, especially that of palmar and plantar EDA, as stressed by Edelberg (1972a, 1973a). Though there is some evidence that those sites do not take part in thermoregulatory sweating (Sect. 1.3.2.4), Edelberg (1972a) includes the palms and soles in the adaptive mechanism of thermoregulatory functioning of sweat secretion as well.

Thermoregulatory responses to environmental stimuli that elicit emotional reactions may be biologically adaptive in different ways. Heat loss due to peripheral vasodilatation together with increased "emotional" sweating may constitute preparatory adaptations to the increase of body core temperature as a result of enhanced metabolic activity in states of high arousal or stress (Sect. 3.2.1.1 & 3.2.2.2).

Another hypothesis concerning the biological significance of the EDA focuses on the role of the sweat glands in regulating the hydration of palmar and plantar corneum. Darrow (1933) has argued that hydration provides optimal frictional contact with objects being manipulated, and increases tactile sensitivity. However, it is questionable whether tactile sensitivity is really dependent on corneal hydration, or whether EDA is to be regarded as a concomitant of CNS activity leading to a sensitization of cutaneous receptors (Sect. 1.2.4). Edelberg (1971), who investigated the relationship between the degree of ANS activity on the one hand, as measured by SRR and finger pulse volume, and the tactile sensitivity using 250-Hz vibratory stimuli on the other hand, found evidence for a central activation process, providing an explanation of the high correlation between ANS and sensitivity threshold shift. During rest, the correlations between spontaneous EDRs and tactile sensitivity were low, whereas these correlations increased rapidly in registration periods following a startling stimulus. The lowering of sensation thresholds was paralleled by an increase in central activation, which was in turn already regarded by Edelberg (1961) as a source for autonomic activity as well as for the sensitivity threshold shift.

The relationship between EDA and tactile sensitivity is further enlightened by pharmacological evidence. Arthur and Shelley (1959) and Fitzgerald (1961) suggested that free nerve endings that extend into the epidermis (Sect. 1.2.4) serve as sensory afferents. However, those fibers may also serve as autonomic efferents which may take part in the origin of SP (Niebauer, 1957). Additionally, there is evidence for a direct influence of a cholinergic agent on cutaneous sensitivity. Bing and Skouby (1950) showed that the number of active cold receptors at the volar surface of the lower arm increased following injections of acetylcholine, mecholyl, or prostigmine. Injections of atropine had the opposite effect. Wilcott (1966) found that intracutaneous mecholyl injections into the forearm resulted in changes of sensory thresholds. He also showed that lowering of pain threshold following a needle prick was associated with a negative SP wave, whereas a rise of this threshold was associated with a positive SP wave (Sect. 1.4.2.3). In another experiment reported in the same article, Wilcott (1966) investigated

the relationship between palmar SP and changes in pain threshold elicited by electrical stimulation. He found a lowering of thresholds that was accompanied by either positive or negative SPRs. These results may suggest that changes in sensitivity thresholds are influenced by cholinergic agents, which also produce the EDA changes being observed as concomitants of threshold shifts. Earlier, Löwenstein (1956) found that the stimulation of sympathetic fibers travelling to the frog's skin resulted in lowered tactile receptor thresholds and a delay of their adaptation. However, sympathetic influences in the frog are transmitted adrenergically.

The possible association between EDA and improvement of frictional contact may be illustrated by everyday behavior, which also optimizes the wetness of palmar epidermal sites, such as moistening the finger with the lips before turning pages, or rubbing one's palms before grasping a tennis racket. In the latter case, there is an inverted U-shaped relationship between the degree of moistening of the skin and the frictional contact with the rugged synthetic surface of the racket (Adams & Hunter, 1969). However, the frictional properties of skin reach their maximum at an intermediate level of surface moisture. Thus, Edelberg (1972a) suggests a control mechanism to safeguard against excessive moistening. Edelberg (1973a) suggests that this is due to the absorption reflex which is connected with the positive SP wave. Hence, the positive SP component could be interpreted as an indication of task-oriented, finely coordinated motor activity (Edelberg, 1972a).

Additional evidence came from Edelberg (1967), who showed that EDRs could be recorded on those sites of the soles – the heel and the ball of the foot – that are in direct contact with the ground. Another site which shows considerable EDA is on the inner side of the foot between the big toe and the ankle (Sect. 2.2.1.1). This region is especially stressed in tree-climbing primates. Edelberg (1967) also found that the amplitudes of the negative SPRs on palmar and dorsal surfaces of the fingers were nearly identical, however, lower by far than those at thenar and hypothenar sites or at the foot. On the other hand, the positive SPRs were particularly high on palmar sites of the fingers and the hand. Hence, positive SPRs are prominent on those sites which are needed for tactile manipulation, while sites that are included in gross body movements, such as on the feet, show predominantly negative SPRs. However, it remains open as to what degree the different thicknesses of stratum corneum at different sites contributed to the results.

Besides the biological significances of EDA already discussed, moisturizing of skin following subsequent EDRs may also have protective properties in cases of injury, since the resistance of the corneum against cutting or rubbing is increased (Adams & Hunter, 1969). Wilcott (1966) observed that skin treated with atropine, which abolishes sweat gland activity, can be more easily abraded with a dental drill than untreated skin or skin soaked with distilled water. Having a defensive orientation, this kind of adaptation could serve as an explanation for the observation that threatening situations are strong eliciting stimuli for EDRs.

A somewhat risky interpretation of the "emotional" sweating occurring at palmar and plantar sites (Sect. 1.3.3.3) is given by Edelberg (1972a): because sweat is not simply a solution of electrolytes, but additionally contains organic substances (Sect. 1.3.3.1), sweating at those sites could serve as a tracking aid in certain species. It is possible that the olfactory action of organic agents in sweat would help, for example, a child to identify his/her mother's scent and, in addition, act as a signal for a threatening situation.

1.4 Biophysics

Electrodermal phenomena are spontaneous as well as elicited changes of a complex system with elements showing different electrophysical properties. The various electrodermal models outlined in Section 1.4.3 are all built of fixed as well as variable resistors and capacitors. Some of them contain voltage sources localized in the skin or the sweat glands, representing polarized membranes. However, these can be regarded as capacitors which are already charged. So the following introduction mainly focuses on resistances and conductors and their physical properties.

From a system-theoretical point of view, the methods of electrodermal recording can be assigned to the following three groups:

(1) Endosomatic recording (Sect. 2.2.3.1). With this method, only those properties of the electrodermal system which result from active changes of the system are considered. The electrical energy is presumed to stem from the polarized membranes in the skin as mentioned above.

(2) Exosomatic recording with direct current (Sect. 2.1.1, 2.1.2, & 2.2.3.2). The electrodermal system is supported with electrical energy from outside. This is performed using either constant voltage or constant current. These methods mainly focus on passive properties of a system, in which capacitors are charged, and changes in the signal are mainly due to resistive changes.

(3) Exosomatic recording using alternating current (Sect. 2.1.5 & 2.2.3.3). This method is used infrequently. In addition to the system properties mentioned under (2), responses of the electrodermal system to oscillatory signals are investigated, which also include changes in capacitors or charged membranes in the skin.

Prior to the discription of electrophysical properties of the skin and the sweat glands some fundamental principles of electrophysics and systems theory will be discussed.

1.4.1 Resistor- and capacitor-based systems

In this section some fundamentals are described in an illustrative and understandable manner, as necessary for the comprehension of electrodermal phenomena and the

corresponding models. Readers who already have knowledge of electrophysics will especially find Section 1.4.1.1 and the beginning of Section 1.4.1.2 rather elementary. However, such prior knowledge cannot be presupposed for all researchers applying EDA measurment.

1.4.1.1 Some fundamental electrical dimensions

Between two bodies with electrical charges Q of different sizes (e.g., between the two poles of a battery) there exists a potential difference, which is described as the voltage U and is measured in volts (V). When the two bodies are connected by a conductor, an electrical current will flow through the conductor until the potential difference is equalized and voltage becomes zero; this current I is measured in amperes (A), 1 A being defined as the amount of current that flows with a charge of 1 coulomb for one second.

In the simplest case, voltage and current are proportional; that is, the quotient of voltage and current is constant. This constant is defined as the electrical resistance R, the relationship between the three dimensions being set out in the following equation:

$$U = RI \tag{1}$$

This equation is known as Ohm's law. Electrical conductors which obey this law are known as ohmic resistances. Their strength is given in ohms (Ω) and is defined as follows: when, by a voltage of 1 V, a current of 1 A flows, there exists a resistance of 1 Ω.

Equation (1) states that the proportionality between applied voltage and flowing current is dependent upon the resistance R. It also illustrates the reversed proportionality between resistance and current flow with constant voltage; the greater the resistance is, the less current can flow. This dependence can also be formulated using the reciprocal of resistance (i.e., conductance G) as follows:

$$G = \frac{1}{R} \tag{2a}$$

G (Footnote 2, Sect. 1.1.1) is measured in Siemens (S).[21] The reverse is also true:

$$R = \frac{1}{G} \tag{2b}$$

When the reciprocal conductance value from Equation (2b) is inserted in place of R in Equation (1), it follows:

[21] In Anglo-American usage until recently the unit "mho," the mirror image of "ohm," was used instead of the unit S. Meanwhile, S was applied as the SI-unit for conductance. Venables and Christie (1980) argued for the continued usage of mho, but in the last ten years most researchers have used the unit S for conductance.

$$U = \frac{I}{G} \tag{3}$$

Hence, the strength of the current I flowing through the resistance is directly proportional to the conductance G if the voltage U is constant.

In biological processes, resistances appear commonly in the region of several thousand ohms, the unit for resistance most used is the kiloohm (kΩ). Correspondingly, it is usual to use microsiemens (μS), whereby the following calculation mode for resistance and conductance is used:

$$G[\mu S] = \frac{1000}{R[k\Omega]} \tag{4a}$$

and

$$R[k\Omega] = \frac{1000}{G[\mu S]} \tag{4b}$$

In the recording of electrodermal activity, not only are the values of resistance and conductance taken for a specific point in time, but also, and above all, the changes in these values (ΔR and ΔG) are recorded and used. In this case the simple relationships of the Equations (4a) and (4b) do not apply anymore. When $\Delta R = R_2 - R_1$ and $\Delta G = G_2 - G_1$, then the following equation applies:

$$\Delta G = \frac{1}{R_2} - \frac{1}{R_1} = \frac{R_1}{R_1 R_2} - \frac{R_2}{R_1 R_2} = -\frac{\Delta R}{R_1 R_2} \tag{5a}$$

The minus sign makes it clear that an increase in resistance leads to a decrease in conductance. Correspondingly, the reverse, following Equation (2b), also applys:

$$\Delta R = \frac{1}{G_2} - \frac{1}{G_1} = \frac{G_1}{G_1 G_2} - \frac{G_2}{G_1 G_2} = -\frac{\Delta G}{G_1 G_2} \tag{5b}$$

In the calculation of resistance changes from conductance changes and vice versa, levels for conductance and resistance must be taken into account; that is, they must also be recorded. In practice, instead of the product of R_1 and R_2, $R_1{}^2$ is normally used in the denominator of Equation (5a), because the error is small when ΔR is relatively small in comparison to R (Sect. 2.3.3.2). The corresponding is true for Equation (5b) when ΔG is small in comparison to G_1.

1.4.1.2 Changes in RC circuits when DC is applied

An RC circuit is an electrical circuit in which a capacitor (C) is charged and discharged through a resistor (R).

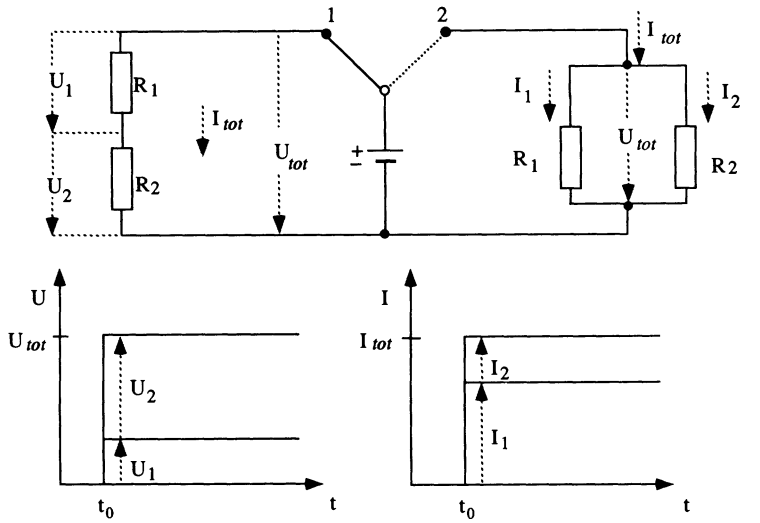

Figure 7. Resistors connected serially (left, circuit position 1) and in parallel (right, circuit position 2) and the resulting subdivisions of the total voltage U_{tot} and total current I_{tot}.

An ohmic resistor to which direct current is applied will transform electrical energy into heat. Voltage is said to "drop" across the resistor. Basically there are two ways to connect resistors in a circuit: either in series with each other or in parallel.[22]

Figure 7 shows, on the left-hand side, two serially connected resistors. Over each resistor the voltage U_{tot} that was applied to the circuit as a whole drops; the partial voltages U_1 and U_2 add up to the original U_{tot}. The current I_{tot} is the same in both resistors. Serially connected resistors are treated additively.

When the resistors are not connected serially, but in parallel, another effect results: while the same voltage U_{tot} lies on each resistor independent of its size, since they are all directly connected to the full voltage, the current is subdivided according to the size of each resistor, whereby Ohm's law must be applied to each resistor. Figure 7 shows, on the right-hand side, such a parallel circuit. The currents I_1 and I_2 add to equal the current I_{tot}, which flows through the circuit.

The size of an insertable resistance R_{tot} through which the same current flows by the same voltages as by the parallel resistances R_1 and R_2 can be calculated by Ohm's law (Equation (1)):

[22]By such considerations, the inner resistance of the voltage source should always be negligible on the grounds of simplification.

$$R_{tot} = \frac{U}{I_{tot}} = \frac{U}{I_1 + I_2} \tag{6a}$$

Again following Ohm's law:

$$I_{tot} = \frac{U}{R_{tot}} \quad \text{and} \quad I_1 = \frac{U}{R_1} \quad \text{and} \quad I_2 = \frac{U}{R_2} \tag{6b}$$

By dividing the right and left side of Equation (6a) by U, inverting them and inserting of the values of I_1 and I_2 from Equation (6b), the following results:

$$\frac{U}{R_{tot}} = \frac{U}{R_1} + \frac{U}{R_2} \tag{6c}$$

When both sides of Equation (6c) are divided by U, the following results:

$$\frac{1}{R_{tot}} = \frac{1}{R_1} + \frac{1}{R_2} = \frac{R_1 + R_2}{R_1 R_2} \tag{6d}$$

From that directly follows:

$$R_{tot} = \frac{R_1 R_2}{R_1 + R_2} \tag{6e}$$

From Equation (6e), it follows, as can be seen using numerical examples, that the replacement resistance for a parallel circuit is smaller than the sum of single resistances.

In contrast to resistors which consume electrical energy, capacitors store it. Technical capacitors consist of two parallel, electrically conductive plates separated by an isolating so-called dielectric. When a voltage is applied to these plates, they are charged and build up an electrical field. When the plates are fully charged, no more charging current will flow. When the voltage source is removed the full voltage remains between the plates until they are short circuited through a load. Via discharge, a current, in the opposite direction to the charging current, flows until the voltage between the plates reaches zero.

A capacitor's capacitance indicates its ability to store electrical load. The bigger it is, the more load can be stored, given a fixed voltage. The relationship between the load Q, the capacitance C, and the voltage U is linear, as shown in Equation (7):

$$Q = CU \tag{7}$$

The capacity of a capacitor is expressed in farads (F) and is defined by the following: a capacitor in which the voltage reaches 1 V in 1 second by a charge current of 1 A has the capacity of 1 F. In practice, as in the case of conductance (Sect. 1.4.1.1), the usual values are much smaller. Therefore capacitance is given in μF, nF, or pF.

Figure 8 shows the temporal relationship between voltage and current from charging and discharging a capacitor. In circuit position 1 the capacitor is charged. Voltage U_C, measured on the capacitor C, rises exponentially until the value U is reached, while the

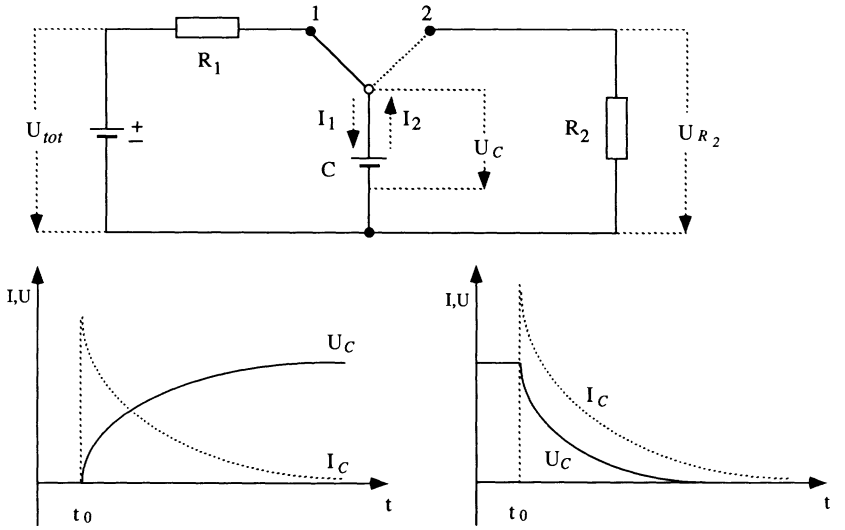

Figure 8. The charging (left, circuit position 1) and discharging (right, circuit position 2) of a capacitor in an RC circuit with the corresponding current and voltage graphs.

charge current I drops exponentially to zero, dependent upon the serially connected resistor R_1 and the capacitor C. When the fully charged capacitor is connected as in position 2, the capacitor discharges through R_2, (i.e., it is short-circuited). Thereby, voltage and current drop exponentially to zero, whereby the strength of the discharge current I_2 as a temporal alteration of the capacitor charge is defined by:

$$I_2 = \frac{dQ}{dt} \tag{8}$$

The falling voltage U_{R2} on the resistor R_2 of the short-circuited system is in the opposite polarization to the voltage U_C and shows the same temporal course as I_2, so that:

$$U_C + U_{R2} = 0 \tag{9a}$$

Following Ohm's law, $U_{R2} = R_2 I_2$, and transformation of Equation (7) results in $U_C = Q / C$. Therefore:

$$(R_2 I_2) + \frac{Q}{C} = 0 \tag{9b}$$

Dividing by R_2 and insertion from Equation (8) gives:

$$\frac{dQ}{dt} = -\frac{Q}{CR_2} \tag{9c}$$

When the variation of a value is in proportion to that value, an exponential course of that value in time is indicated, as seen in the differential Equation (9c) for the charge Q of the capacitor. It is fulfilled, as can be shown through insertion, by the following equation:

$$Q = Q_0 e^{-\frac{t}{RC}} \tag{10a}$$

whereby Q_0 is the initial charge value and Q is the charge value at a particular time point t. The product of resistance and capacitance, RC, is labelled time constant τ and indicates how fast the exponential curve declines:

$$\tau = CR \tag{10b}$$

Thereby, capacitance in F and resistance in Ω must be given. A capacitor, when being charged, reaches 63% of its full charge at τ sec. If being discharged, after τ sec 37% of its charge is left (Sect. 2.3.1.3.2). A raising of the capacitance n times leads, as does a raising of the resistance n times, to the time constant of $n\tau$.

When several capacitors are connected in series and are fully charged, the charge is the same in each capacitor and corresponds to the total charge Q_{tot}. In the case of two serially connected capacitors this means:

$$Q_1 = Q_2 = Q_{tot} \tag{11a}$$

When Equation (7) is solved for U for each capacitor, Q_1 and Q_2 can be replaced by Q_{tot} following Equation (11a):

$$U_1 = \frac{Q_{tot}}{C_1} \quad \text{and} \quad U_2 = \frac{Q_{tot}}{C_2} \tag{11b}$$

As on the left-hand side of Figure 7, the voltages U_1 and U_2 add up to the voltage U_{tot}, the same goes for U_{tot} as for the single voltages (Equation (11b)), and the following results:

$$\frac{Q_{tot}}{C_{tot}} = \frac{Q_{tot}}{C_1} + \frac{Q_{tot}}{C_2} \tag{11c}$$

When both sides of Equation (11c) are divided by Q_{tot}, the following results:

$$\frac{1}{C_{tot}} = \frac{1}{C_1} + \frac{1}{C_2} \tag{11d}$$

In the case of serially connected capacitors, the reciprocal value of the replacement capacitor is determined by addition of the reciprocal values of the single capacitors, in opposition to serially connected resistors which are added to each other.

When two capacitors are connected in parallel, the full voltage U_{tot} lies across both simultaneously. The charges of the capacitors are calculated using Equation (7) as follows:

$$Q_1 = C_1 U_{tot} \tag{12a}$$

and

$$Q_2 = C_2 U_{tot} \tag{12b}$$

As the adjacent plates of the single capacitors can be regarded as one big capacitative plate, the total charge is calculated as follows:

$$Q_{tot} = Q_1 + Q_2 = C_1 U_{tot} + C_2 U_{tot} \tag{13a}$$

Isolating U_{tot} and dividing both sides of Equation (13a) by it gives:

$$\frac{Q_{tot}}{U_{tot}} = C_1 + C_2 \tag{13b}$$

Following a corresponding transformation of Equation (7) it can be seen that the left side of Equation (13b) equals C_{tot}. Therefore:

$$C_{tot} = C_1 + C_2 \tag{13c}$$

Capacitors connected in parallel, therefore, behave as if added to each other, in contrast to the replacement resistance for parallel resistors, which is smaller than the sum of the single resistors.

Figure 8 shows an RC circuit in which a resistor and a capacitor are connected in series (i.e., the capacitor C is charged through the resistor R_1 and discharged through R_2). Networks of resistors and capacitors connected in parallel may also be built. The charging and discharging processes are similar to those shown in Figure 8, however, the voltage rise will be delayed in time.

1.4.1.3 Changes in RC circuits when AC is applied

Once the capacitor in a RC circuit is fully charged following the application of DC, only the resistive properties of the circuit are measurable. Should the capacitative properties of RC circuits also continually be determined, as in the course of possible variations in polarization capacities during the EDR (Sect. 2.1.5 & 2.2.3.3), either the direct current must be continually switched off and on (pulsed DC, Sect. 1.4.1.4) or, for example, a sinusoidal alternating voltage must be applied for the measurement of the system's electrical properties.

Alternating voltages are defined by the fact that their strength and direction change periodically. The most commonly used alternating voltage is sinusoidal; in this case, the voltage amplitude is calculated by the sinus of the angle of a circle whose radius

is the maximum amplitude and which is passed once during a period, as shown in the following equation:

$$U(t) = U_0 sin(2\pi f t) \tag{14}$$

where f is the frequency of the alternating voltage, $U(t)$ the amplitude at time t, and U_0 is the maximum amplitude of the voltage. In an AC circuit when only ohmic resistances are involved, the voltage drops as in a DC circuit. Furthermore, the effect of serially connected resistors as voltage dividers, and of parallel-connected resistors as current dividers, is the same as in a DC circuit (Fig. 7). Current and voltage in the presence of purely ohmic resistances are always "in phase."

This is no longer the case when a capacitor is put into an AC circuit. In DC, following the finish of charging, no more current can flow through the capacitor branch and the full voltage is measurable across the capacitor (Fig. 8), but the electrically conductive plates in an AC circuit charge up alternatingly positive and negative, so that a standing alternating charge and discharge current flows.

In a circuit with only one capacitor, an AC is measurable the strength of which varies with the rise and fall of the alternating voltage. When the voltage and current of a capacitor are measured, the phase of the current will lag behind that of the voltage. The reason for this is the following: before a voltage can be built up on the capacitor's plates, a current must flow. This current is at its maximum when the voltage is zero, and is itself at zero when the full voltage is reached (Fig. 8, lower left). This is true for the positive and for the negative phase (i.e., the current's maximum is reached a quarter period before the maximum of the positive and negative voltage amplitude). This phase displacement is described by the phase angle φ through which the current flow precedes the voltage course.

The AC resistance of a capacitor is frequency dependent. With a lower frequency the capacitor will be charged and discharged less often during a certain period; the average strength of the current is therefore smaller with a lower than with a higher frequency, by which the capacitor would be charged and discharged more often. The rising current indicates a higher transmission factor for AC, which means that the AC resistance of the capacitor decreases with rising frequency.

This can be seen mathematically from Equations (7), (8), and (14). The transformation and differentiation of Equation (7) with t gives:

$$\frac{dQ}{dt} = C\frac{dU}{dt} \tag{15a}$$

For sinusoidal alternating voltage, differentiation of Equation (14) with t gives:

$$\frac{dU}{dt} = (2\pi f U_0)\cos(2\pi f t) \tag{15b}$$

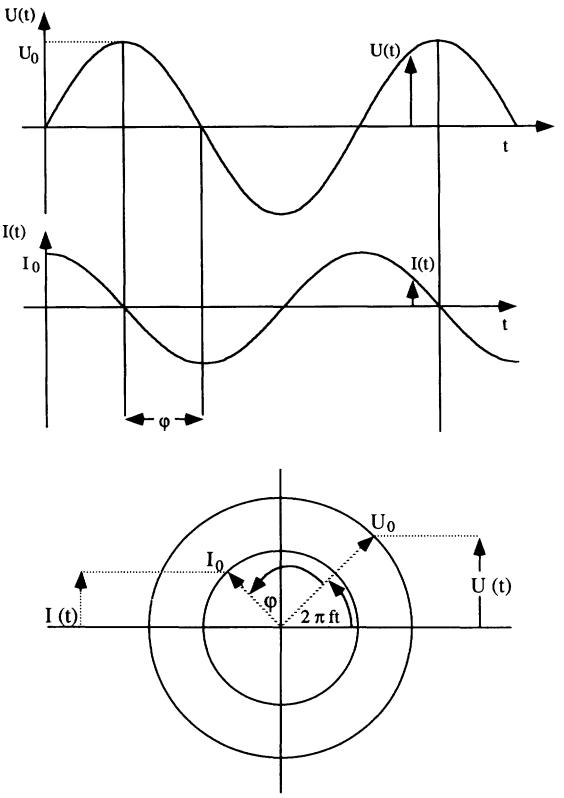

Figure 9. Phase displacement of voltage and current by application of alternating voltage to a capacitor (upper part) together with the corresponding vector diagram (lower part). (See text for explanations.)

When Equation (15b) is inserted in Equation (15a), and it is noted that from Equation (8) that $I = dQ/dt$, then:

$$I(t) = (2\pi C f U_0)\cos(2\pi f t) \tag{16a}$$

The product $2\pi C f U_0$ is a constant and gives the maximal value of the current I_0 by a certain frequency f:

$$I(t) = I_0\cos(2\pi f t) \tag{16b}$$

Figure 9 correspondens to a system composed of only one capacitor, where the inner resistance of the voltage source is negligible. In this case, the phase displacement φ will be 90^0 as shown in the vector diagram in the lower part of Figure 9. In this diagram, the current I, whose strength is calculated by Equation (16b), has a value of:

$$|I| = 2\pi C f U_0 \tag{16c}$$

The value of the impedance $Z\ (f)$ for the frequency f is given as the quotient of the values of U and I from Equations (14) and (16c):

$$|Z(f)| = \frac{|U|}{|I|} = \frac{U_0}{2\pi C f U_0} = \frac{1}{2\pi f C} \tag{17}$$

It can be seen from Equation (17) that the impedance Z behaves as the reciprocal of the frequency f, given a constant capacity C (i.e., by increasing frequency, the AC resistance decreases).

Since an ohmic resistance transforms electrical energy into heat (Sect. 1.4.1.2) it is described as an active resistance. By contrast, a capacitor in a circuit does not transform electrical energy but stores it. Despite this the capacitor limits the current dependent upon the frequency f of the applied alternating voltage. This effect is described as blind resistance, or as reactance X:

$$X(f) = Z(f)\sin\varphi(f) \tag{18a}$$

Since the phase angle φ is 90^0 in a system composed of only one capacitor as shown above (which can, however, be only theoretically true in case of a so-called ideal capacitor), $X(f) = Z(f)$ in that case (i.e., reactance equals impedance).

Through incorporation of an active (ohmic) resistance in such a circuit, the phase angle φ is changed, in relation to the frequency f of the alternating voltage, between 0^0 and 90^0. From the impedance $Z(f)$ and the phase angle $\varphi\ (f)$, the reactance (blind resistance) $X(f)$ can be calculated from Equation (18a). The ohmic resistance $R(f)$ is calculated as follows:

$$R(f) = Z(f)\cos\varphi(f) \tag{18b}$$

When $R(f)$ as the abscissa and $X(f)$ as the ordinate are plotted on a graph, a curve for $Z(f)$ results (Fig. 10). This curve, a so-called locus, fully describes the transfere function of the RC system and can be used for its characterization.[23]

In Figure 10, three differing loci are plotted, through which the responses of different systems to the applied alternating voltage can be described. The curves shown in the diagram are for a system with a parallel circuit of a resistor and a capacitor.

[23]These connections can also be elucidated through a depiction with complex numbers. There $R(f)$ is taken as the real part and $X(f)$ as the imaginary part of a complex function. This depiction, though preferred in electrophysics and in systems theory, not be developed here, due to the need for simplification.

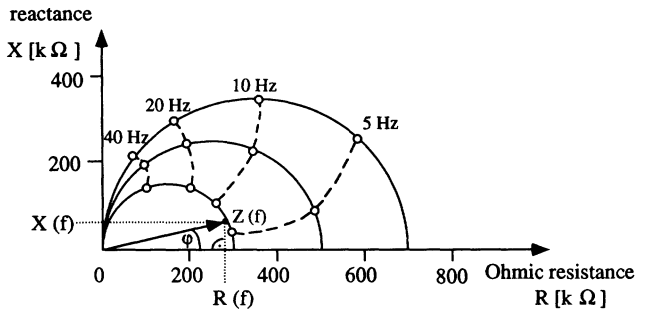

Figure 10. Three different loci. The innermost curve results from an impedance vector $Z(f)$ of around $f = 7$ Hz with its projections $X(f)$ and $R(f)$ drawn to the respective axes.

Such a locus results as follows: when the frequency f of the alternating voltage has the value 0 the system is on, practically, direct voltage. In this case the resistance of the capacitor C would be infinite (Sect. 1.4.1.2) and the impedance Z would be determined alone by the Ohmic resistance; therefore $Z(0)$ would equal $R(0)$. The vector Z would therefore lie on the R-axis by $f = 0$. When f is raised by an applied alternating voltage, then C would, so to speak, allow current flow through it (i.e., the Z vector becomes shorter as the total impedance of the system decreases). With increasing f, the angle of the vector Z to the X axis increases, that is, the blind resistance part of the impedance increases. The total impedance decreases continously with rising frequency (i.e., the Z vector becomes continously shorter), until it achieves 0 by $f \rightarrow \infty$, as then the capacitor practically short-circuits the resistor.

From the projection of the impedance vector $Z(f)$ to the R and X axes the relationship between Z, R, and X can clearly be shown. Following Pythagoras's theorem, Equation (19a) holds for each frequency, and therefore being independent from the phase angle:

$$Z(f) = \sqrt{R(f)^2 + X(f)^2} \tag{19a}$$

The AC conductance, which corresponds to the AC resistance (i.e., impedance $Z(f)$), is named the admittance and symbolized by Y (Table 1, Sect. 1.1.1):

$$Y(f) = \frac{1}{Z(f)} \tag{19b}$$

It can be subdivided into the real part, the conductance $G(f)$, and the imaginary part, the susceptance $B(f)$ (Footnote 23). With application of Equations (19a) and (19b),

the susceptance B can be calculated as follows from the reactance X and the ohmic resistance R:

$$B(f) = \frac{X(f)}{X(f)^2 + R(f)^2} \tag{20a}$$

The conductance G is calculated as follows:

$$G(f) = \frac{R(f)}{R(f)^2 + X(f)^2} \tag{20b}$$

B and G can be determined thereby from the impedance Z and the phase angle φ with the help of Equations (18a) and (18b). The locus determination of conductance and susceptance ensues correspondingly from Figure 10, and the equivalent of Equation (19a) also applies to the relationship between $Y(f)$, $G(f)$, and $B(f)$. Examples of loci in the conductance-susceptance graph are given in Section 1.4.3.3.

The processes which occur by application of alternating voltage to biological tissues with an ability to build up polarization capacities (Sect. 1.4.2.2) are comparable to technical capacitors. However, they are complicated by the fact that tissues must be, electrophysically, regarded as circuits of higher complexity than the simple RC circuits built up here, partly as containing more resistors in series and in parallel, and also with more capacitors (Sect. 1.4.3.2). Through these types of additional elements, the current and voltage processes through time are further affected. In principal, however, the resistive and capacitative properties of such complex systems can be simulated by relatively simple substitute circuits (Sect. 1.4.3.3).

The various measurement procedures for determining the phase angle φ, the impedance Z, and the admittance Y are described in Section 2.1.5.

1.4.1.4 Determining system properties of unknown RC systems

The consequences of application of sinusoidal alternating voltage to circuits composed of resistors and capacitors, as described in the foregoing section, can be regarded in systems theory as the deformation of a defined input signal by a system. In systems theory such processes are used to research the properties of unknown systems.

A graphic example of so-called Lissajous figures, also the quantitative determination needs only the appropriate depiction of phase displacement and amplitude relationship between the input and output signal with the help of an oscilloscope by which the time basis is interchangeable with the amplifier insert. Figure 11 shows how the combination of an input signal with the maximal amplitude E_0 and an output signal with the maximal amplitude A_0 creates an elliptical figure whose main axical incline is dependent upon the A_0/E_0 relationship. The length of the short axis is dependent upon the phase displacement; it reaches its maximum by $\varphi = 90^0$ and disappears by $\varphi = 0^0$.

The system properties of an RC circuit with known resistors and capacitors in series and in parallel can be determined through the application of a single alternating voltage

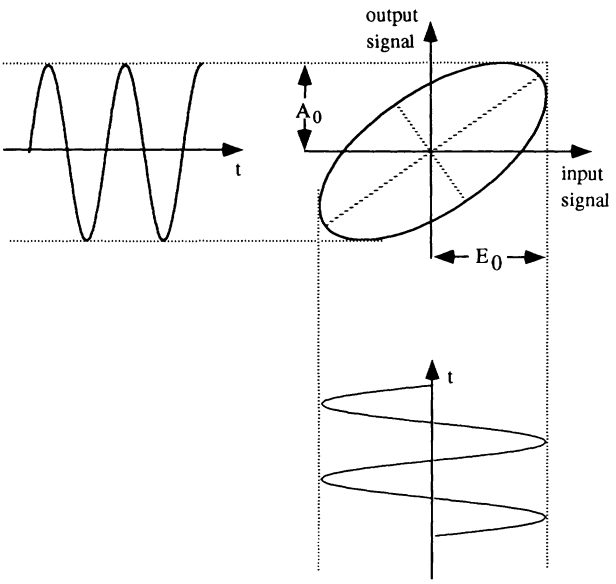

Figure 11. Lissajous figure. E_0: Maximal amplitude of the input signal. A_0: Maximal amplitude of the output signal. *t*: Time axis. Dotted: The axes of the ellipse.

frequency. To investigate unknown systems such as the skin, however, recording must be repeated with a number of different frequencies. The disadvantage of such a process is the long time necessary for measurement, especially with the inclusion of low frequencies; in the low-frequency area the system becomes stabilized only after around five full periods. Therefore, techniques using successive impulses with differing alternating voltage frequencies have been developed to date only for the recording of the tonic parts of EDA and not for those of a phasic EDR (Sect. 2.1.5).

However, it is theoretically possible to stimulate a system such as the skin with all frequencies of a defined spectrum at the same time (known technically as "noise"). The system's response is divided into its spectral components with the help of Fourier analysis, and phase and amplitude spectrums are obtained from which the system's response to the differing frequencies of the given input noise can be derived. This process requires a very high temporal resolution and – depending on the narrow phasic variations in comparison to the possible tonic values of the EDA signal (Sect. 2.1.3) – necessitates, not only in the temporal but also in the amplitudal area, highly resolutive analog/digital (A/D) converters and laboratory computers which make a correspondingly fast data transfer possible.

To record the system's response to all frequencies of a spectrum simultaneously, a further possibility is offered by the so-called pulse spectrum analysis. The responses to pulse-formed signals, which begin at zero, return to zero, and remain there till the next signal, are described in systems theory as "transients." The unknown system is stimulated by a sequence of periodic DC impulses (so-called pulsed DC). Each sequence of square wave impulses can be conceived as a result of overlapping sine waves of different frequencies. Figure 12 shows how a certain spectrum of sine waves is summarized, forming needle impulses (a process that can be regarded as opposed to Fourier analysis). The resulting value y at a given time t is calculated as the sum of the amplitudes from n overlapping sine waves according to Equation (21), where $\pi/2$ is brought in to obtain a maximum at the beginning:

$$y(t) = \sum_{t=1}^{n} \sin\left(2\pi f i t + \frac{\pi}{2}\right) \tag{21}$$

In the upper part of Figure 12, due to the need for lucidity, only $n = 3$ overlapping sine waves are displayed. It can be seen that at particular points in time (see arrow), when all single sine waves are in phase, constructive interference occurs. In the middle part of Figure 12, the summation curve of three sine waves, as determined by Equation (21), is shown. Here, an enlargement of the resulting amplitude at all time points at which the single waves lie in phase can be seen. From the summation of sine waves by $n = 60$ frequencies, peaked square wave impulses will result, as is shown in the lower part of Figure 12. When the number of such overlapping frquencies is very large, spikes are created, the so-called Dirac impulses or delta surges, which are preferred in systems theory applications because of their ideal properties (theoretically infinitely peaked and containing all frequencies). Dependent on the system's recovery time, they can be applied in very fast sequences, thus making a continuous recording of the system's properties possible which is only limited by the scanning rate and the repetition rate of the impulses. The system's response to the impulse of a delta surge, which consists of all stimulation frequencies, can be calculated via Fourier analysis, which again requires fast data handling.

Theoretically, the electrical properties of the unknown skin-sweat-gland system may be continously analyzed by using transient analysis, for example, with Dirac impulses (see also Footnote 31, Sect. 2.1.5) or by using noise as a probe signal, instead of using various successive frequencies. However, these techniques will create specific problems in electrodermal recording that will be discussed in Section 2.1.5.

1.4.2 Electrophysical properties of skin and sweat glands

When an external current is applied to biological tissues, such as skin, they act like electrical networks built of resistors and capacitors. Electrical modelling of the skin using the elements described in Section 1.4.3 does not require the skin to be built of elements having discrete resistive or capacitative properties. However, there are parts

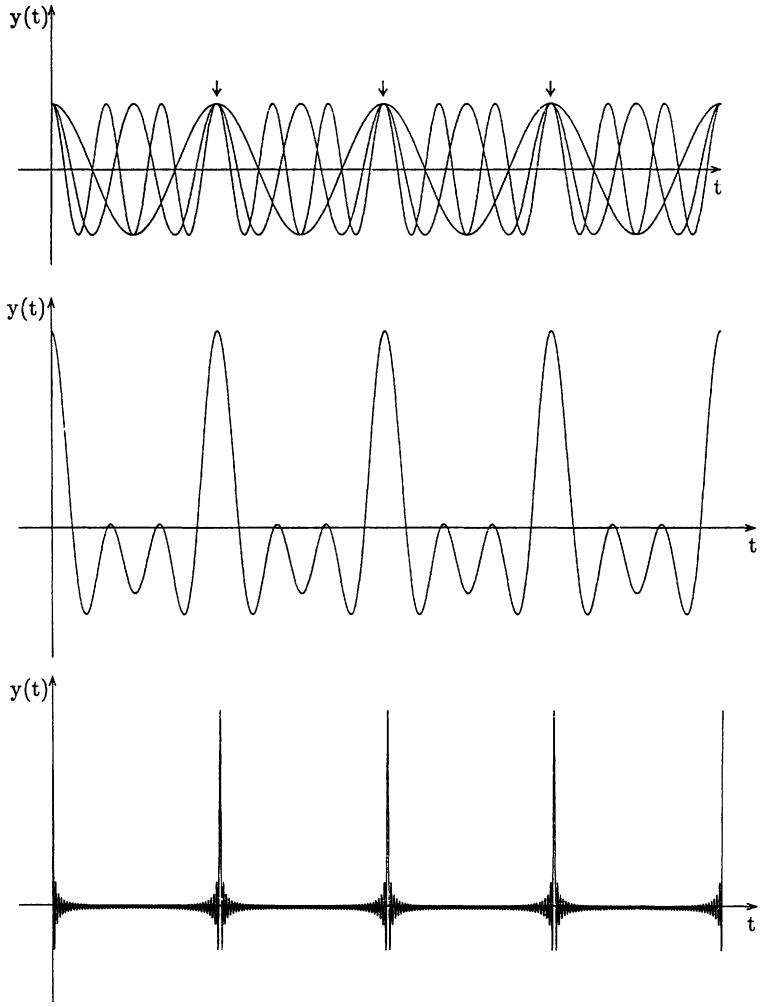

Figure 12. Overlapping of three sine curves (above), their summation curve (middle), and a corresponding summation curve of 60 basic sine frequencies. The arrows show at which temporal points all waves are in phase.

of the skin and sweat glands that are likely to act electrophysically more or less like the resistive or capacitative elements included in the appropriate models.

Blood, ductal sweat, and interstitial fluid show different conductivities, which vary with their ionic concentration. Therefore, they act as variable resistors (Sect. 1.4.2.1). In contrast, the cellular boundaries formed by membranes show more capacitor-like characteristics (Sect. 1.4.2.2), since their selective permeability forms an obstacle for the ions involved in the current flow. As a result, storage of ions at these boundaries is followed by the buildup of a potential difference, the direction of which is opposite to the applied voltage, and hence called the "back electromotive force" (back e.m.f.).

Those membranes have the ability to store electrical energy like capacitors, which gives them the property to act as a polarization capacity (Fricke, 1932). They become sources of potentials, which are included in some models of EDA (Sect. 1.4.3.2). Membranes which have polarization capacity and hence capacitor-like or potential-like properties, are presumed to be located at the sweat gland membranes, at the dermal-epidermal boundary membrane, and in the epidermis (Sect. 1.3.4.2.2). All these properties together form the active sources for electrodermal phenomena (Sect. 1.4.2.3).

1.4.2.1 Resistive properties of skin and sweat glands

The dermis and the subcutis, which are well supplied with blood and interstitial fluids (Sect. 1.2.1.3), show good electrical conductivity, which may vary to some extent, depending on changes in blood flow. In addition, the epidermal Malpighian layer as well as the stratum intermedium (Table 2, Sect. 1.2.1.1) may be regarded as relatively conductive structures, thus not adding much to the skin's resistance. Therefore, the lower corneal zone, which is relatively impermeable to water and solutions, is thought to be mainly responsible for the skin's resistance (Fowles, 1974). However, as outlined in Section 1.3.4.2.1, an exact localization of such an epidermal barrier is not possible, and the whole stratum corneum is regarded as being a variable resistor, depending on its degree of hydration.

The stratum corneum with its keratinized cells does not contain living membranes, which maintain a diffusional balance between the inner and outer cellular milieu. Instead, the whole corneum acts as a sponge, taking up water and solutions from inside as well as from outside the body, which are depleted when the corneum dries out. Under normal physiological conditions the corneum is always partially hydrated, and the degree of its hydration is dependent on environmental relative humidity. With an increase in sweating, corneal hydration also increases, leading to tonic and/or slow phasic changes in skin resistance. If the corneum becomes dry, for example, as a result of aging (Sect. 2.4.3.1), and probably by spontaneous reabsorption of water into the underlying dermis (Edelberg, 1973a), tonic skin resistance increases.

However, it is more likely that the conductivity of the stratum corneum depends on its electrolyte content than on its humidity (Sect. 1.3.4.2.1). As outlined by Fowles (1986a), corneal permeability for the electrolytes is much less thoroughly investigated

than that for water. Edelberg (1971), in discussing some more or less contradictory results, states that most ions will be able to penetrate the main part of the stratum corneum, where lots of intercellular spaces are present, at least to the barrier layer as mentioned above. He also presumes that the corneal permeability for the electrolytes is the same as water, since an active ionic transport seems to be improbable in fully keratinized cells (Sect. 1.3.4.2.2). Thus, moistening of the corneum by sweat through the acrosyringium and/or via the skin surface will add more to its conductance than insensible perspiration, which penetrates the epidermal barrier layer (Sect. 1.3.3.2).

As already mentioned in Section 1.3.4.2.2, sweat gland ducts act as electrical shunts through the stratum corneum. This is especially important with respect to palmar and plantar sites which are preferred for electrodermal recording, because of their great sweat gland density (Sect. 1.2.3). It is generally supposed that skin conductance increases with the height of the column of ductal sweat (e.g., Edelberg, 1968). Accordingly, the slow decline in SCL which appears in the absence of EDRs may reflect a gradual dissipation of sweat in the ducts, possibly attributable to the reabsorption mechanism mentioned in Section 1.3.3.1 (e.g., Rothman, 1954). However, electrical models of skin that focus on its resistive properties (e.g., Montagu & Coles, 1966) regard each sweat gland as a single resistor with a more or less fixed value that can be switched on or off (Fig. 15, Sect. 1.4.3.1). This kind of modelling will be adequate in the case of a fast rise and fall of ductal sweat, leaving the gradual changes of conductance owing to the corneal moistening.

To sum up, resistive properties of the skin sweat gland system may be described as several serially and parallel-connected resistors, as illustrated in Figure 13:

(1) A variable resistor formed by the stratum corneum.

(2) A fixed resistor formed by the epidermal barrier as mentioned in Section 1.3.4.2.1.

(3) Resistances of sweat gland ducts that are switched either into or out of the circuit.

(4) A fixed but relatively low resistance of the lower epidermis, the dermis, and probably the subcutis.

In addition to these resistive pathways which are vertical to the skin surface, various horizontal resistances can be assumed in all layers of skin, especially in the lower epidermis and in the dermis, depending on the tissue's conductivity. However, the majority of electrodermal models do not consider those resistances, except for the model given by Fowles (1974), which is shown in Figure 17 (Sect. 1.4.3.2).

1.4.2.2 Capacitative properties of skin and sweat glands

When an external current is applied to the skin, its cell membranes exhibit their polarization capacities, storing electric potentials like technical capacitors (Sect. 1.4.1.3). However, the selective permeability for ions, which is the basis of these capacitative

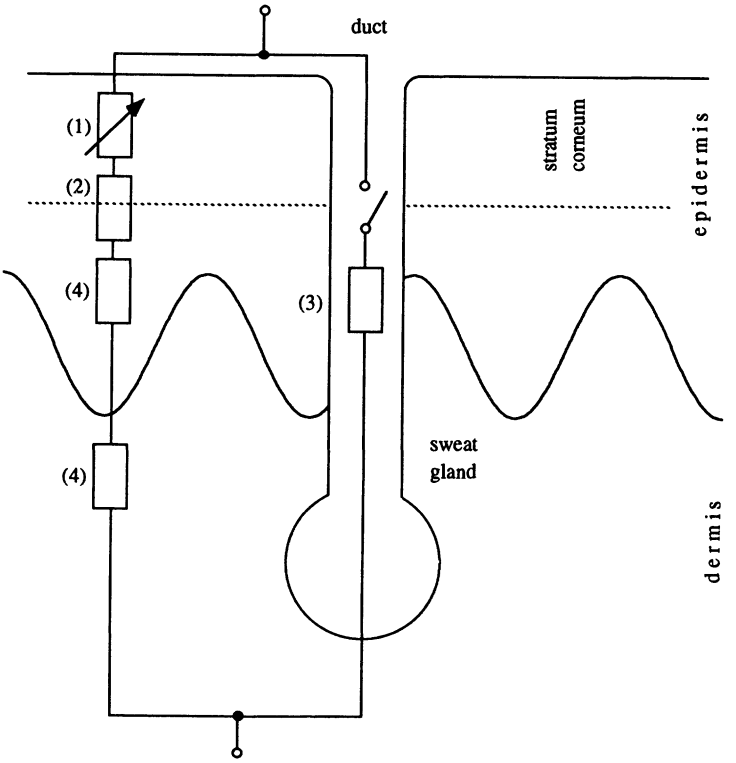

Figure 13. Schematic illustration of resistive pathways through the skin and the sweat gland. An explanation of the numbering is given in the text.

properties, is not only linked to single membranes (Sect. 1.3.4.2.1). In addition, according to Edelberg (1971), whole cell assemblages such as epidermal layers may act selectiveley to some degree on the influx of ions of different sizes because of the cell structures extending into the intercellular spaces (e.g., in the stratum spinosum; Sect. 1.2.1.1). Thus, the whole epidermis will react to an external current like a network built from RC links connected in parallel and in series.

As already outlined in Section 1.3.4.2.2, the membrane-like properties of the keratinized epidermal layers have to be regarded as passive compared to those of living tissue. However, skin also contains active membranes (e.g., those of nerve, muscle, and glandular cells).

These membranes have a resting charge which becomes reversed when stimulated. In addition, they also show capacitative properties when an external current is applied. Active membranes that act as capacitors with respect to electrodermal activity are located mainly in the secretory part of the sweat gland. Edelberg (1972a) hypothesizes an active epidermal membrane with a fixed negative charge, making it selectively permeable to cations.[24] He postulates a phasic increase in permeability, which can be detected with surface electrodes as an EDR. It is probably located either in the stratum granulosum, at the dermal-epidermal boundary, or in the epidermal wall of the sweat gland duct.[25] Other capacitative properties may stem from membrane polarizations and depolarizations in the blood capillaries, the pilo-erecting muscles (Sect. 1.2.4), and the myoepithelia surrounding the sweat glands (Sect. 1.2.3).

Edelberg (1971) regards any contribution by myoepithelial potentials to the endosomatic EDA as unlikely, because potentials arising there would be shunted by the freely conducting dermal tissue. According to his view, this cannot be generalized to the influence of capacitative properties of the sweat gland as well, since potential changes at its secretory membrane are transmitted immediately to the skin surface when sweat gland ducts are filled. There is also a possibility of epidermal reabsorption processes in the ductal walls, found up to the stratum germinativum (Sect. 1.3.3.1), to form membrane-like capacitors having an influence on EDA. However, it is not certain that these charges are big enough to be measurable with relatively large electrodes at the skin surface.

The capacitative properties of skin and sweat glands have been much less investigated than the resistive ones. Those investigations require measurement with AC, which is far less common than DC measurement (Sect. 1.4.3.3).

1.4.2.3 Origins of active electrical properties in the skin and sweat glands

While the previous two sections focused on passive electrical properties of the skin/sweat gland system, in this section the active electrodermal phenomena stemming from the active membranes already mentioned will be discussed. Those properties are mainly investigated using endosomatic measurement of EDA (without an external current), the result of which is skin potential recording.

Exosomatic EDRs have a simpler form than endosomatic ones, since they are always unidirectional. On the contrary, SPRs can appear as monophasic negative responses, as biphasic responses, where an initial negative component is followed by a

[24]Edelberg (1971) reported microelectrode recordings which provide evidence for the existence of an electrical barrier layer in the deeper layers of the epidermis. The SRL measured via a microelectrode, which had been slowly pushed into the epidermis, showed a slow continuous decrease at the beginning. If a certain point had been passed at which the subject first reported weak pain, SRL suddenly decreased until only the electrode resistance itself was present. The depth of the appropriate layer is 350 μm at the palm and 50 μm at the forearm.

[25]Edelberg (1971) first suggested a second barrier membrane at the dermal-epidermal boundary. According to Fowles (1974), he later preferred the ductal wall at the height of the stratum germinativum as the locus of this second membrane.

positive one, or as triphasic responses, where the positive limb of the biphasic response achieves a greater negativity than the initial negative wave (Sect. 2.3.1.2.1). Under certain circumstances it is also possible that only a positive SPR is recorded showing either no initial negativity or an extremely small one (Fowles, 1986a). This variety of responses has generated various explanations that have combined active membrane properties together with resistive properties of corneal hydration and duct filling, as discussed in Section 1.4.2.1. A major portion of the appropriate research was performed with the cat's footpads,[26] and based on these results hypotheses were formed by Lloyd (1961), Darrow (1964), and Adams (1966), which were comprehensively reviewed by Edelberg (1972a).

Lloyd (1961) found that each single sympathetic nerve stimulation was followed by a negative SPR, which he called presecretory. Repeated stimulation resulted in a very slow positive SPR wave of several minutes duration, which was accompanied by duct filling, and thus labelled secretory potential. When ducts were already filled, further stimulation led to presecretory potentials with an increase in amplitude. Hence, the rise of sweat in the ducts is likely to enable better electrical contact to the generator of the presecretory SPR.

Darrow (1964) as well as Darrow and Gullickson (1970) regarded the sweat gland as the source of changes in SP, both negative and positive. They further assumed that neural impulses may cause increases in permeability of the epidermis, including the corneum. They regarded the intraluminal potential of the sweat gland as being positive with respect to the surrounding tissue, leading to negative SPRs on the surface resulting from the extraluminal tissue when ducts are empty, and to positive lumen-generated surface potentials with filled ducts. However, positive SPRs could not be obtained from the cat's footpad, even when the ducts were full (Wilcott, 1965; see also Footnote 26). Additionally, with direct microelectrode measurements, Schulz et al. (1965) found that the lumen of the human sweat gland duct is highly negative with respect to the surrounding tissue.

Therefore, Edelberg (1968, 1971), in his model outlined in Section 1.4.3.2, presupposes a negative intraluminal potential, together with a relatively steady tonic sweat gland activity which results in the sweat column normally reaching up to the Malpighian layer. Outpouring of sweat onto the surface may result from either increased sweat gland activity or a contraction of the myoepithelial tissue surrounding the duct (Sect. 1.3.3.1). This causes an increase in surface negativity (a negative SPR with long rise time and slow recovery) which is due to the sweat reabsorption in the ductal walls.

However, Edelberg (1972a) points to the fact that this duct-filling component neither explains the mostly short SPR recovery times, nor the observation that EDRs appear with heavy perspirers whose ducts should always be completely filled. Therefore, he suggests a short-lasting increase of the permeability for cations in the active epi-

[26]The SPRs in cats differ from those seen in humans in that they show only a monophasic negative SPR (Edelberg, 1973a), which reaches its peak amplitude very quickly (for differences between species, see also Footnote 9, Sect. 1.2.3).

dermal membrane, already mentioned in Section 1.4.2.2, as the appropriate source for EDRs with fast rise times and quick recoveries. He assumes the appropriate mechanism is connected with the control of evaporation (Sect. 1.3.3.2), either with the control of corneal moistening, or with the reabsorption of sweat in the ductal walls. The independence of such a reabsorption component from sweat gland secretion, as assumed by Edelberg, is, however, questioned by Bundy and Fitzgerald (1975), who found a dependency of the EDR recovery on the previous phasic electrodermal activity (Sect. 2.5.2.5).

Edelberg explains in his model that the biphasic and triphasic SPRs (Fig. 35, Sect. 2.3.1.2.1) are composed of a positive membrane component with short recovery time and a negative duct-filling component with long recovery. Whether the SPR begins with a negative or positive component is, according to Edelberg (1971), dependent on the degree of duct filling at the onset of the reaction. If ducts are relatively empty, the rise of sweat will establish a connection between skin surface and the negative lumen potential, thus leading to an initial negative SP wave. On the other hand, if ducts are already filled, an additional sweat secretion will result in corneal hydration, thus producing the epidermal potential, which is less negative than the ductal one (Fig. 16, Sect. 1.4.3.2) and which is observable at the surface as a positive SP shift. Because hydration would occur too slowly to explain fast, positive SPRs, Edelberg (1972a) assumed an epidermal or ductal membrane response as the source of the positive SPR with fast recovery.

Empirical evidence is available for both an active sweat gland and an active epidermal component of SP. Fowles and Johnson (1973) as well as Fowles and Rosenberry (1973) showed that the amplitudes of positive and negative SPRs markedly decrease when the stratum corneum becomes moistened. They assume this is caused by a mechanical closure of sweat pores, and additionally take these observations as evidence that positive as well as negative SPRs are due to changes in sweat gland potentials. Experiments with parallel recordings from the fingertip and the nailbed, which does not contain sweat glands, performed by Edelberg (1973b) and repeated by Burbank and Webster (1978), gave evidence for additional sources of skin potential, probably in the epidermis.

Figure 14 schematically depicts the localization of all hitherto discussed active electrodermal components. The depicted potential sources also act as capacitors in case of an applied external current (Sect. 1.4.2.2). They mainly correspond to the potential sources as assumed in the Fowles model (Fig. 17, Sect. 1.4.3.2): E_1 is located in the secretory part of sweat gland, E_2 corresponds to the potential source in the epidermal duct at the level of the stratum germinativum, and E_3 is the membrane potential stemming from the inner corneal zones. E_2 and E_4 correspond to the membrane potentials mentioned in Section 1.4.2.2, which relate to the sodium reabsorption mechanism in the dermal and possibly also in the epidermal, part of the duct (Sect. 1.3.3.1). E_5 stems from the myoepithelia and is probably cholinergically supported (Sect. 1.3.2).

It remains questionable whether an active epidermal membrane E_3 should be included, since evidence for its innervation is lacking. Fowles (1974) points to authors

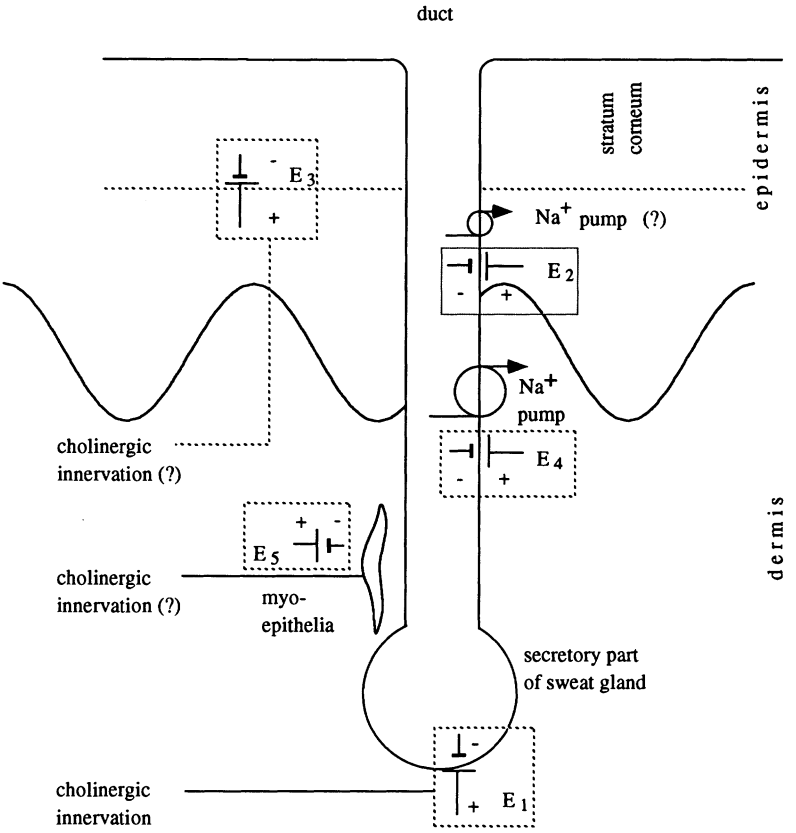

Figure 14. Schematic illustration of the localization of active electrical properties in the skin and the sweat gland. E_1, E_2, and E_3: See text explanations for Figure 17 (Sect. 1.4.3.2); E_4 and E_5: See text for explanations. Adapted from F. H. Muthny (1984), *Elektrodermale Aktivität und palmare Schwitzaktivität als Biosignale der Haut in der psychophysiologischen Grundlagenforschung*, Fig. 17.4. Copyright ©1984 by the author. Used by permission.

such as Lykken (1968), who – contrary to Edelberg's view – locate the suggested active membrane responsible for fast SPR components not in the epidermis but in the secretory part of the sweat gland. Thus, those electrodermal components could also be regarded as a result of an increased permeability of the secretory cells during secretion. Like all active membranes, secretory cell membranes have a high polarization capacity at rest, which is diminished during depolarization. However, Fowles (1974) objects to this hypothesis, stating that this active membrane is easily reached by solutions on the skin surface, which change its properties. It is not clear whether the active membrane, which is probably responsible for a main portion of the EDR, is really cholinergically innervated. Muthny (1984), in his experiments described in Section 2.4.2.2, could not abolish all palmar EDRs after an intradermal application of atropine, a finding contrary to all previous research. So the nature and localization of this active membrane component remain unclear (Edelberg, 1983).

1.4.3 Models of the electrodermal system

To assist in the depiction of the electrical properties of the skin and sweat glands and their interactions, a succession of electrical equivalent circuits of varying complexity, which simulate the electrodermal system, are sketched out below. Discussion of such models is found in Edelberg (1971) and Fowles (1974), as well as in Millington and Wilkinson (1983).

As our knowledge of the electrical properties of the skin is still very limited, all attempts to develop electrical equivalent circuits for the represented structures have been tentative (Venables & Christie, 1980). It must also be pointed out that although the skin may display the same systematic properties as an electrophysical model, it still cannot be said that the skin is built in the same manner as the model. The following section describes the most important electrical circuits, together with perspectives for the future research of the electrodermal system, and for further modelling by means of the application of AC technology.

1.4.3.1 Models based exclusively on resistive properties

Although there is no doubt that the electrodermal system also contains capacitative properties, models built solely of resistive elements have at least a heuristic value for DC measurements. In this approach, capacitors play a role for only a short period, that is, after switching current on or off (Sect. 1.4.1.2). Such a model has been presented by Montagu and Coles (1966).

The left-hand panel of Figure 15 shows the Montagu-Coles model, which displays, but does not further discuss, an additional capacitative element C. Resistor R_1 represents a series resistance located in the dermis and body core. R_2 represents the resistive value of the stratum corneum, which is in parallel to resistors r_1, ..., r_n of the sweat gland ducts. These single ductal resistors can be switched either into or out of the

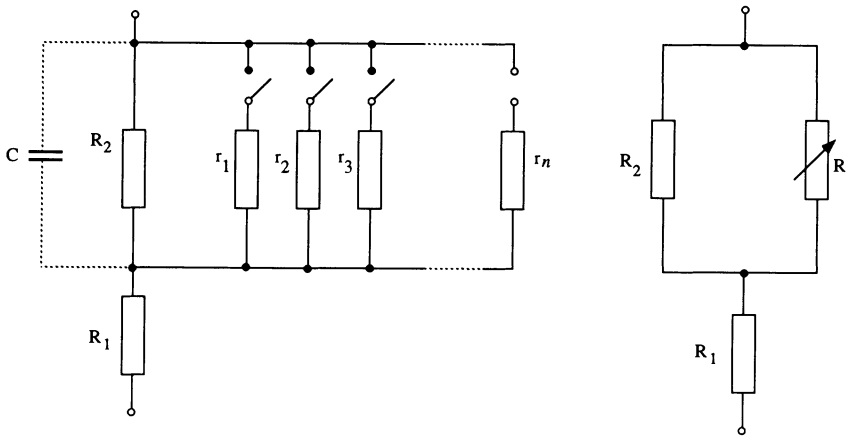

Figure 15. Left-hand panel: Electrical equivalent circuit for the skin, according to Montagu and Coles (1966). R_1: Resistance of the dermis and the body core. R_2: Resistance of the stratum corneum. r_1, ..., r_n: Connectable resistances of the sweat gland ducts. C: Capacitative element. Right-hand panel: Simplified Montagu-Coles model. R: Variable resistance resulting from sweat gland ducts. Left-hand panel from J. D. Montagu and E. M. Coles (1966), Mechanism and measurement of the galvanic skin response. *Psychological Bulletin, 65*, Fig. 1, p. 262. Copyright ©1966 by the American Psychological Association. Reprinted by permission of the publisher and the second author.

circuit, depending on the sweat gland activity, thereby altering the parallel resistance (Sect. 1.4.1.2).

The right-hand panel of Figure 15 shows a simplified model set up by Boucsein, Baltissen, and Euler (1984a), who have formally substituted a variable resistor R in place of these single parallel resistances of the sweat glands, and who also left out the capacitor C, which was not considered further. The assigning of a constant value for the resistance of the corneum by Montagu and Coles (1966) is a simplification (Sect. 1.4.2.1). However, it may be taken as an initial approximation that R_2 is relatively constant as opposed to R, since the keratinized layer has a considerably narrower range of resistive changes than do the sweat gland ducts. The total resistance of the equivalent circuit on the right-hand panel of Figure 15 is calculated as follows (Sect. 1.4.1.2, Equation 6e):

$$R_{tot} = R_1 + \frac{R_2 R}{R_2 + R} \tag{22}$$

Fluctuations of the total resistance, which depend upon small variations of the resistance R, are calculated according to the following differential equation: [27]

$$dR_{tot} = \frac{{R_2}^2}{(R_2 + R)^2} dR \tag{23}$$

It can be inferred from Equation (23) that, in the case where the keratinized layer resistance (R_2) is not considered constant, differentiation using a second variable is then necessitated, which would considerably complicate the equation system. However, the more serious limitation of the Montagu-Coles model stems from its being a solely resistive model, which can only take into account changes in resistance. Therefore, the application of this model is limited to DC measurements of EDA. Since, on the one hand, the greatest number of EDA investigations have used external DC, and because, on the other hand, the introduction of capacitative elements significantly complicates the mathematical formulation for models, the heuristic value of this simple model remains undiminished.

1.4.3.2 Models additionally including capacitative properties

While the model proposed by Montagu and Coles (1966), described in the preceding section, focuses mainly on the resistive properties of the skin, the following models from Edelberg (1971) and Fowles (1974) additionally take into account potential sources. These are regarded as origins of endosomatic EDA. In the case of exosomatic EDA measurement, these potential sources primarily display capacitative properties of the electrodermal system.

To further the construction of the model for the active electrical processes which are the basis for the origins of skin potentials, Edelberg (1971) has introduced the depiction of inner potential current into his model, which is presented in Figure 16. From his results, obtained by means of microelectrode measurements, it follows that the epidermis, as well as the lumen of the sweat gland duct, displays a negative potential in reference to the body core, and furthermore that the lumen displays a greater negative potential (Sect. 1.4.2.3). Edelberg views the current in the skin as being a major factor determining the potential difference at the surface, which depends on strongly differing polarization capacities (Sect. 1.4.2) in the epidermis and the sweat glands (Fig. 16); the current I flows from the less negative pole P_E (epidermis) to the more negative pole P_S (sweat glands). The potential, measured with the voltmeter (VM) on the surface of the skin, is additionally a function of the resistance of the epidermis, R_E, and of the sweat gland duct, R_S.

This model from Edelberg does not depict the processes involved in exosomatic EDA measurements using DC, as no connecting, resistive pathway between the elec-

[27] Taking into consideration that R_1 = const. and with application of the quotient rule for differentiation. The corresponding conductance equation is given in Boucsein et al. (1984a).

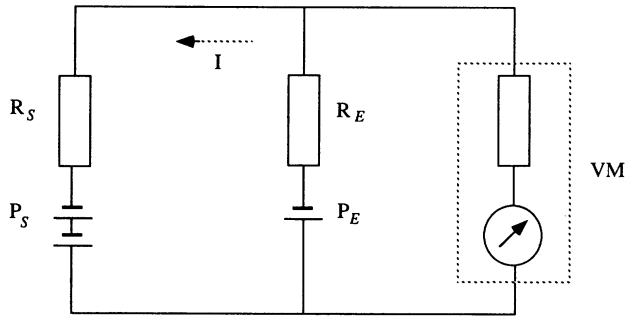

Figure 16. Equivalent circuit for the generation of the inner potential compensative current I in the skin. R_S: Sweat gland resistance and the inner resistance of the generator of the sweat gland potential P_S. R_E: The epidermal resistance and the inner resistance of the generator of the epidermal potential P_E. VM: Voltmeter, with its internal resistance. From R. Edelberg (1971), Electrical properties of skin. In R. Elden (Ed.), *A treatise of the skin: Vol 1. Biophysical properties of the skin*, Fig. 15.3. Copyright ©1971 by the New York Academy of Sciences. Reprinted by permission of the publisher and the author.

trodes is taken into account, and therefore once the capacitative elements are fully charged, no further current can flow through the system (Sect. 1.4.1.2).

Figure 17 shows the electrical model of the skin as proposed by Fowles (1974), in which three potential sources from Figure 14 (Sect. 1.4.2.3) are combined with three main conductance paths, as follows:

(1) The lumen negative potential E_1, which originates in the ductal wall in the dermis, and is determined primarily by the sodium concentration in the lumen, together with the possible variable resistance R_4 of the dermal duct wall, and the variable resistance R_2 of the dermal section of the duct which depends upon duct filling (E_1 and R_4 correspond in part to P_S and R_S in Figure 16).

(2) The potential E_2, which is also a lumen negative potential, is generated across the epidermal duct wall at the stratum germinativum level. It is dependent on the concentration of sodium and chloride ions in the lumen. Due to the capacitative properties of the ductal wall (Sect. 1.4.2.2), the membrane in this part of the duct is less selective. Hence, E_2 is likely to be smaller than E_1 during sweat gland activity. R_3 represents the variable resistor of the duct wall, and R_1 is the resistive value of the epidermal part of the duct depending on the duct filling.

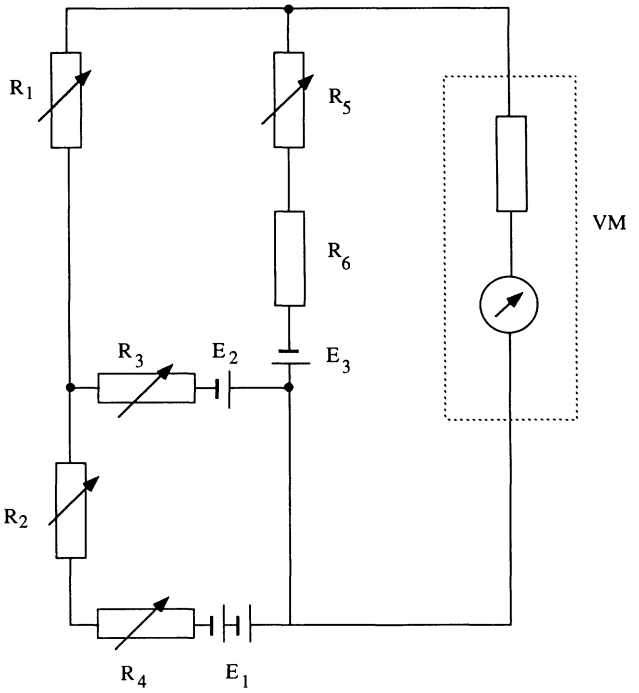

Figure 17. Equivalent circuit for the skin. VM: Voltmeter with its internal resistance. See text for further explanations. From D. C. Fowles (1974), Mechanisms of electrodermal activity. In R. F. Thompson and M. M. Patterson (Eds.), *Methods in physiological psychology: Vol.1. Bioelectric recording techniques, Part C: Receptor and effector process*, Fig. 9/5. Copyright ©1974 by Academic Press. Reprinted by permission of the publisher and the author.

(3) Following Fowles's suggestions, E_3 should be located according to the localized membrane potential, in the lower zone of the stratum corneum (Table 2, Sect. 1.2.1), and is a function of the potassium concentration in the interstitial fluid as well as of the applied electrolyte in the electrode paste (Sect. 2.2.2.5). It will be surface negative as long as the outer potassium concentration is greater than that of the interstitial fluid. Resistive values in this pathway are the relatively constant resistance R_6 of the compact keratinized-layer zone as well as the variable resistance R_5 of the upper layers of the corneum, varying with their hydration (E_3 and R_6 correspond in part to P_E and R_E in Figure 16).

For the sake of simplification, Fowles omitted a fourth pathway in which the current flows into the duct from the corneum and then along the two sweat gland pathways.

Under complete resting conditions, reabsorption predominates over secretion, whereby the resistances R_1, R_2, and R_3 maintain high values, while the potentials E_1 and E_2 are minimal. The potential E_3 is then the most important factor for measurement of the potential at rest and reflects the potassium concentration in the interstitial fluid. A small or moderate sweat secretion lowers the resistance of R_2 and probably of R_1 as well, producing a slow-recovery SCR. At the same time, the sodium concentration of the lumen rises, thereby increasing E_1. The increase of this potential together with the decrease of the resistance in the duct leads to a slow-recovery negative SPR.

These reactions cause an increase in both the SCL and the SPL. Larger sweat gland responses or those occurring in ducts that are partially full will further decrease R_1 and R_2. If the hydrostatic pressure (or the sodium concentration) is high enough to depolarize the epidermal duct membrane, a response in the epidermal duct occurs whereby R_3 is decreased and a small lumen negative potential originates at E_2. This membrane response produces an SCR with a short recovery time, and at the same time a positive SPR appears, because of a shunting effect on E_1. However, in most cases there will be a small initial negative component of the SPR since the negative wave begins earlier than the increase in permeability. Once the ducts are maximally filled, further sweat gland secretions will produce only membrane responses.

In this model, most of the presumed origins of electrodermal phenomena are taken into account (Sect. 1.4.2.3). According to Edelberg (1971), negative SPRs and SCRs with longer recovery times appear as a result of the filling of the ducts, while the membrane responses cause SCRs with shorter recovery times, positive SPRs, and possibly the low-risetime negative SPR, which appear either alone or as the initial portion of many biphasic SPRs. The modification of Edelberg's model by Fowles (1974) is that positive SPRs are not regarded originating in a positive potential of the epidermal duct's wall. Instead, their origin is supposed to be a breakthrough of the potential from the dermal part of the duct. Furthermore, it is presumed that the membrane reaction is triggered through hydrostatic pressure and not through a cholinergically transmitted neuronal reaction. Edelberg's model is further revised by relating the duct potentials to the sodium transport mechanism, and attributing the epidermal membrane potential, which is independent of sweat gland reactions, to the potassium concentration in the interstitial fluid and in the applied electrolyte.

Contrary to the purely resistive model in Section 1.4.3.1, the models presented here also allow the depiction of active electrical properties of the skin and sweat glands, which go beyond simple variations in the ohmic resistances. However, in a strict sense the models of Fowles and Edelberg are limited to the explanation of skin potentials, because both include no resistor pathway between the electrodes which is not in series with a capacitative element. However, biological membrane potentials are usually designated as "leaky" capacitors; that is, the capacitor is in parallel with a resistor. In any case, the heuristic value of such complicated systems remains questionable, as clearly

defined anatomical and physiological structures of the skin have not yet been successfully categorized with respect to their postulated electrical elements. Consequently, the Japanese team of Yamamoto and Yamamoto used, in their investigations based on AC measurements (Sect. 1.4.3.3), a simplified model (Fig. 18) which corresponds more closely to the Montagu-Coles model (Fig. 15) than to the Fowles model depicted in Figure 17.

1.4.3.3 Specific advantages of AC methods in model building

The models portrayed in the two previous sections are based on the preponderance of DC measurement and potential measurement of EDA. Although investigations of the system, using AC technology or transients, were already proposed in single cases (summarized by Edelberg, 1971), systematic sequences of appropriate studies are still missing, especially on the intact human skin (Sect. 2.5.3 & 2.6.3).

Results obtained by AC measurements on such a complex system as the skin are not easily interpretable (Millington & Wilkinson, 1983). Therefore, simpler substitute circuits are applied to modelling with AC measurements, as compared to the Fowles model presented in the preceding section (Fig. 17). In the simplest case, such depictions represent a fixed polarization capacitaty being connected in parallel with a resistor, together with a resistor in series with both of them (e.g., Edelberg, 1971). The serial resistance is necessary for the description of AC properties of the skin, since the impedance of the system decreases by using very high frequencies, due to the decrease of the resistive properties of the capacitor (Sect. 1.4.1.3), but does not fully disappear. From the value of this residual impedance the purely ohmic components of the skin resistance, which are not connected in parallel with the capacitor, can be determined.

The same holds for the Montagu-Coles model (Fig. 15), in which a variable resistance R (replaceable respectively by the single resistors r_1, ..., r_n) is connected in parallel with the fixed resistor R_2. Yamamoto, Yamamoto, Ohta, Uehara, Tahara, and Ishizuka (1978) based the interpretations of their results using AC measurement upon such a model which closely resembles the one Tregear (1966) proposed, adding a capacitor in parallel (Fig. 18). Yamamoto et al. (1978) regard the resistance R_1 as negligible, thus only specifying values for R_2, R, and C (Sect. 2.5.3.1). The model from Lykken (1971), based upon pulsed DC measurements, unites R and R_2 into a single variable resistor and thereby presents the simplest depiction of a circuit composed of a capacitor and resistors connected in series and in parallel. The capacitor and the parallel resistors are localized in the stratum corneum, while the serial resistor represents the deeper dermal and epidermal skin layers, including the stratum granulosum.

With the help of AC measurements, the quantification of variations in the different elements of this simple electrical equivalent circuit will now be explained by means of their loci (Sect. 1.4.3.1). Following the presently dominating preference of conductance units over resistance units (Sect. 2.6.5), conductance (C) and susceptance (B) are used here instead of ohmic resistance (R) and reactance (X), respectively, and ad-

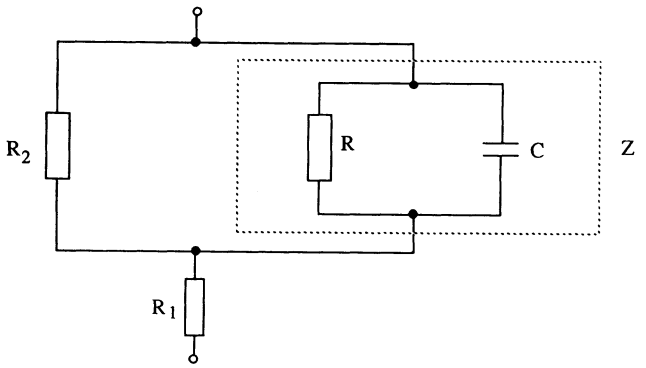

Figure 18. Equivalent circuit for the skin, according to Yamamoto et al. (1978). R_1: Resistance of the dermis. R_2: Constant resistance of the epidermis. Z: Variable impedance, composed of the ohmic section R and the capacitative section C. Adapted from Y. Yamamoto, T. Yamamoto, S. Ohta, T. Uehara, S. Tahara, and Y. Ishizuka (1978), The measurement principle for evaluating the performance of drugs and cosmetics by skin impedance. *Medical and Biological Engineering and Computing, 16,* Fig. 1, p. 623. Copyright ©1978 by Peter Peregrinus. Used by permission of the publisher.

mittance instead of impedance as well. Therefore, opposed to what is shown in Figure 10 (Sect. 1.4.1.3), the vector moves to the right on the locus with increasing AC frequency. Figure 19 shows the change of the admittance vector $Y(f)$ as a function of the frequency of the applied measurement voltage as used in the electrical equivalent circuit of Figure 18, where, for the sake of simplicity, R and R_2 are combined as a single resistor, R_2 (Lykken, 1971), as both circuits are electrically equivalent.

When $f = 0$ Hz (i.e., by DC) the admittance is determined by the conductance of the ohmic resistors R_1 and R_2 alone, since no more current flows once the capacitor C is fully charged (Sect. 1.4.1.2), the phase displacement is zero, and the vector Y lies on the real axis (Footnote 23, Sect. 1.4.1.3). The length of the vector $Y(0)$ (i.e., the conductance of the entire system by $f = 0$) corresponds to the conductance G_{tot} for DC, and is calculated, as derived from Equation (2a), by:

$$Y(0) = G_{tot} = \frac{1}{R_{tot}} = \frac{1}{R_1 + R_2} \tag{24a}$$

When the values for R_1 and R_2, in the form of $1/G_1$ and $1/G_2$, are inserted into Equation (24a) according to Equation (2b), a common denominator G_1G_2 is formed in the denominator by which Equation (24a) is expanded, and the following results:

$$Y(0) = \frac{G_1G_2}{G_1 + G_2} \tag{24b}$$

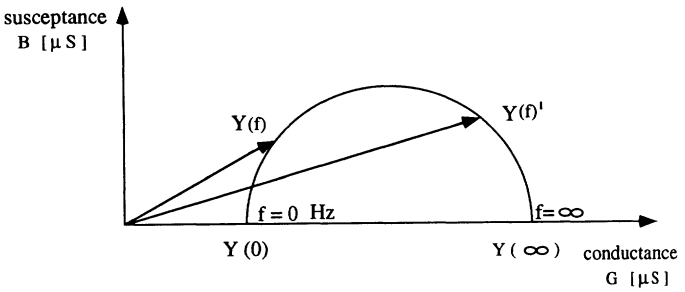

Figure 19. Alteration of the admittance vector $Y(f)$ in dependence upon the frequency of the applied measurement voltage.

The admittance $Y(0)$, in the case of DC voltage in the circuit shown in Figure 18, corresponds to the harmonic mean of conductances of both the series (R_1) and parallel (R_2, including R) resistors.

When f is increased, the capacitor begins to conduct (Sect. 1.4.1.3 for apparent conductance) and increasingly short-circuits the parallel resistor R_2. This lengthens the admittance vector, which describes a circle in the conductance/susceptance plane (compare vectors in Figure 19) forming the locus (see Fig. 10, Sect. 1.4.1.3). At infinitely high values of f on the end of the curve, the apparent conductance becomes so high that ultimately admittance is determined by the series resistor R_1 alone, and is composed of only the real conductance component G_1; that means $Y(\infty) = G_1$.

The course of such a locus depends upon the construction of the circuit. Empirical loci for skin admittance (Yamamoto & Yamamoto, 1976, 1981) show good congruity with a semicircle; therefore, the underlying electrical model of the skin (Fig. 18), can be regarded as quite adequate.

Quantitative fluctuations of the resistive (or conductive) and capacitative values of single elements of this model can be quantitatively described through alterations of the locus. As shown in Figure 20, an increase in the parallel resistance R_2 will displace to the right the starting point of the locus along the G-axis, thus diminishing the radius, since the end point remains stationary on the G-axis. For the admittance at a specified frequency f, an increase occurs only in the conductance, but not in the susceptance (compare the displacement between $Y(f)$ and $Y(f)'$ in Figure 20).

In contrast, if the serial resistance R_1 is increased, there will be only a small displacement of the curve's starting point, since R_1 is very small in comparison to R_2 (Sect. 2.5.3.1). Therefore the end point of the semicircle on the G-axis is displaced to the right, thus increasing the radius (Fig. 21). The manner in which these changes act upon the components of the admittance at a specific frequency f is therefore dependent

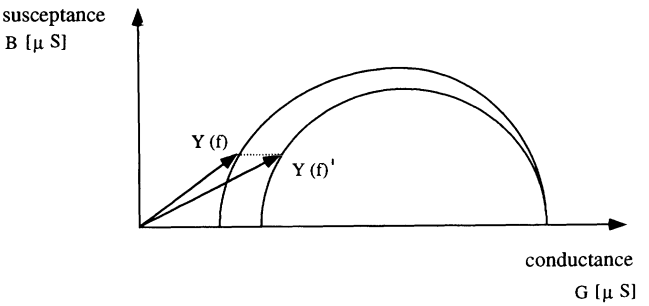

Figure 20. Alteration of the locus and the admittance vector $Y(f)$ by an increase of the parallel resistance R_2.

upon which point on the locus is regarded. However, an alteration of R_1 affects, above all, the susceptance (compare the displacement between $Y(f)$ and $Y(f)'$ in Figure 21).

An isolated fluctuation of the capacitator C at a specified frequency f will have an effect in the same manner as a frequency fluctuation at a fixed value of C (and the other elements); the admittance vector proceeds along the locus. With small and high values of f, an alteration of C will result mainly in an increase or decrease, respectively, of the susceptance, while average values of f will cause an alteration in the conductance. It is therefore necessary to determine, within a range of measurements with the widest possible frequency spectrum, the form and position of the locus, so as to be able to correctly interpret results obtained from a single measurement frequency.

Although the loci and parameters of the single elements from the electrical equivalent circuits resulting from AC measurements can be used for representing the frequency dependent system properties (Sect. 3.5.2.1), it must be borne in mind that these are not ideal RC circuits that are being considered but, on the contrary, real and very complex systems. While AC measurements in these systems give results that are compatible with the properties of such models, the same measurements can, however, result from different physical processes (Millington & Wilkinson, 1983). To date the dependence of these systems upon the current density, as upon possible nonlinear elements, has been considered only in an extremely small number of cases (e.g., Yamamoto et al., 1978), as is the case with implications of system properties of electrical equivalent circuits that include a variable capacitor C (e.g., Tregear, 1966).

Models which try to simulate the anatomical features of the skin in different ways are hardly testable with conventional EDA measurement techniques. Tregear (1966) describes the stratum corneum alone as a system of around 12 parallel-connected pairs of a resistor and a capacitor each, a model that can only be empirically approximated through the aid of the stripping technique (Sect. 1.3.4.2.1).

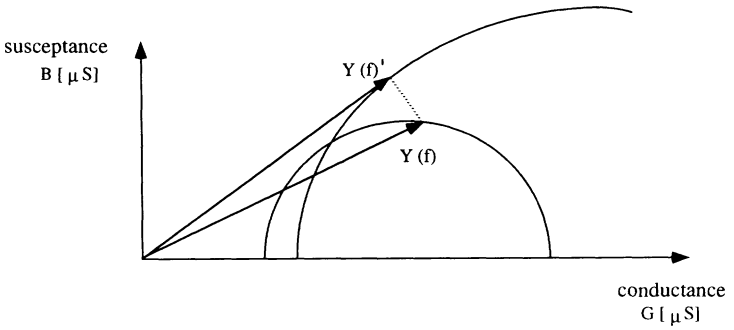

Figure 21. Alteration of the locus and the admittance vector $Y(f)$ by an increase of the serial resistance R_1.

Yamamoto and Yamamoto (1976) have determined the dielectrical and resistive values of five places in the corneum with this technique (Sect. 3.5.2.1). Thiele (1981a) states in his modelling considerations that the electrodermal system is composed of several closely spaced interconnected single systems. Lykken (1971, Fig. 6) has also already suggested a model comprising several sequential, parallel-connected RC networks lying in series with additional potential generators. As a result of his measurements with pulsed DC (Sect. 1.4.1.4), he doubts that polarization capacity in the sense of a frequency-dependent capacitance is present (Sect. 1.4.2.2). In any case, it is possible to use, as simulations, parallel polarized capacitors, connected in series with resistors, such as RC networks used by Lykken (1971). Salter (1979) has shown that for real physiological systems, polarized capacitances cannot be the only elements in electrical equivalent circuits. Additional elements have to be taken into account, such as ideal capacitors being in series and/or parallel in material that is mainly build up dielectrically, or ideal resistors in parallel and in series in material that is composed of conductors and semi-conductors, respectively. As an alternative, the AC properties of the skin (with the inclusion of the sweat glands) can be depicted through nonlinear electronic components, such as Zener diodes and capacitative diodes (Thiele, 1981a) or transistors (Salter, 1981). It remains to be seen whether or not the dynamics of these nonlinear systems will better fit the signal than those of the RC models.

In summary, investigating the AC properties of the skin and sweat glands is more complex than investigating the DC properties. However, due to the capacitative properties of the electrodermal system, such AC investigations are indispensable for modelling. Through investigations using a wide spectrum of AC frequencies and/or the use of transients (Sect. 1.4.1.4), it should be possible to quantitatively comprehend the electrical properties of single components of the electrodermal system, to sketch out

electrical equivalent circuits, and to describe the tonic as well as the phasic components of EDA in connection with their origins and interaction.

Especially in regard to the phasic EDA components, pioneer research is still needed, as the temporal relationship of the single components of skin impedance and admittance (Sect. 1.4.1.3) during electrodermal reactions has hardly been investigated. In a pilot study aided by a specially developed phase voltmeter (Sect. 2.2.3.3), Boucsein, Schaefer, and Neijenhuisen (1989) showed that the phasic variations during an EDR are found in the parallel resistors R and/or R_2 (as shown in the simple models in Figs. 18 and 15), not in the serial resistor R_1 or the capacitative part C, or only to an extremely small degree (Sect. 2.5.3.1).

1.5 Summary of mechanisms

Electrodermal activity results from an interaction of sympathetic nervous system activity and local processes in the skin. There are at least two different CNS sources of sudorisecretory activity leading to electrodermal changes (Fig. 6, Sect. 1.3.4.1). However, they use the same peripheral sudorisecretory efferents to the sweat glands as a common final pathway (Sect. 1.3.2.1). Since specific central sudorisecretory pathways are not really well known (Sect. 1.3.2.2), and hence the central elicitation or inhibition of electrodermal phenomena remains somewhat ambiguous (Sect. 1.3.4.1), much research on the central causation of EDA remains to be performed.

In contrast to the above, local processes in the skin underlying EDA are much better known. It is now generally supposed that sweat gland innervation is cholinergic, an exception within the postganglionic sympathetic system (Sect. 1.3.2). Sometimes, additional adrenergic influences on myoepithelia surrounding the sweat gland (Sect. 1.2.3), which have the capacity to squeeze preformed sweat out of the duct, are discussed. However, these play a minor role, if any (Sect. 1.3.3.1). Another possible cholinergic innervation, which could influence the permeability of a suggested epidermal membrane, remains uncertain, as does the existence of such an active membrane in general (Sect. 1.4.2.3).

There is ample empirical evidence that sweat gland activity in conjunction with epidermal membrane processes plays a major role in the causation of electrodermal phenomena. When sweat gland activity is abolished in humans, either as a result of congenital absence, by sympathectomy, by peripheral sudorisecretory nerve discharge, or by pharmacological blocking, SCRs and SPRs are normally eliminated and SCL is reduced (Fowles, 1986a). Martin and Venables (1966) found that SCR freq. is greatest in skin areas where sweat glands are densest. Thomas and Korr (1957) reported a median intrasubject product moment coefficient of $r = .91$ (with a range from .44 to .96) between counts of active sweat glands and the SCL measured with dry electrodes, held only a few seconds to the skin, so that the corneum beneath the electrode remained dry (Sect. 2.6.5). Since EDA is normally recorded using electrolytes and thus with a

moistened corneum, these high correlations cannot be generalized to EDA recording as a whole. Thus, Edelberg (1971), using his microelectrode technique, found that sweat glands are only responsible for less than 50% of the SCL with moistened corneum.

Sweat secretion does not only lead to duct filling but also to moistening of the relatively dry upper epidermal layer, the stratum corneum (Sect. 1.3.4.2). Both processes cause changes in skin conductance: the ducts form electrical shunts through the epidermal barrier, thus connecting the skin surface with the highly conductive dermal tissue, and moistening of the corneum with the salty sweat generally increases skin conductivity (Sect. 1.4.2.1). Those purely resistive properties of the electrodermal system can be depicted in relatively simple electrical models (Fig. 15, Sect. 1.4.3.1).

In addition to these rather slow-going processes, there are also active electrical properties of the skin and the sweat glands with shorter rise times and recoveries taken into account, which act as potential sources in endosomatic recording (Sect. 1.4.2.3), or as capacitors in exosomatic recording (Sect. 1.4.2.2). These electrical properties are formed by the secretory activity of the sweat gland, the sodium reabsorption mechanism in the dermal and perhaps also in the epidermal part of the duct (Sect. 1.3.3.1), by a suggested epidermal barrier membrane, and by the electrical activity of the myoepithelia surrounding the duct.

Sources of electrical potentials taking part in the occurrence of an EDR could be shown not only for endosomatic but also for exosomatic recordings. It is widely accepted that the effects of electrical potentials on the form of the EDR depend on both duct filling and corneal hydration, which are in turn not independent from each other (Sect. 1.4.2.1). Edelberg (1971) as well as Fowles (1974) argued that the duct filling and the hydration components themselves, together with the ductal reabsorption, represent relatively slow processes, and hence can only influence recovery time but not rise time, which is shorter (Sect. 2.5.2.3 & 2.5.2.4). The fast EDR components were presumed to be caused by membrane polarizations and depolarizations, as described in Section 1.4.2.3, which take place in one of the potential sources mentioned above (e.g., Edelberg, 1972a).

However, electrical models additionally taking into account those various sources of electrical potentials remain of questionable heuristic value, at least for modelling exosomatic EDA, since there are no appropriate methods available to experimentally investigate the behavior of their elements (Sect. 1.4.3.2). Consequently, for an explanation of exosomatic electrodermal changes, Edelberg (1983, Fig. 1) comes back to a resistive model of EDA, which resembles the simplified form of the Montagu-Coles model as depicted in the right-hand part of Figure 15 (Sect. 1.4.3.1). He regards the corneum and the sweat duct as resistors in parallel, connected in series with a resistor which includes some corneal structures and all subcorneal structures, except the sweat gland lumen. Because of its hitherto uncertain role in contributing to conductance changes (see Boucsein et al., 1989), Edelberg also leaves out the frequently discussed active epidermal membrane (Sect. 1.4.2.3), which in cases of exosomatic recording would act as a capacitor in parallel to the resistors of the corneum and the sweat duct.

The recent view of Edelberg (1983) on modelling of EDA takes away a lot of the rather complicated interrelationships of various peripheral elements in causal EDA mechanisms: If the corneum is very dry – which is normally not the case in EDA recordings using electrolytes – SRL increase should rely upon the sweat duct filling. However, the hydrated corneum will act as a shunt around the sweat duct, thus reducing the ductal contribution to the SRL. In his experiments using cats, in which the central sympathetic component was interrupted, Edelberg (1983) found that sympathetic nerve stimulations at approximately 30 sec intervals produce EDRs of much higher amplitudes in preparations with dry corneum than in those with moistened corneum. So there is ample evidence that under a solely resistive perspective, corneal hydration is mostly responsible for EDL, while the exosomatic EDR relies on a secretory membrane component together with a duct-filling component.

For most applications of electrodermal recording, a simple resistive model will be sufficient to explain the phenomena observed. However, capacitative elements certainly play an important role in tonic EDA (e.g., in dermatological applications; Sect. 3.5.2.1). Their possible role in electrodermal reactions is unknown, and should therefore be investigated further. An appropriate electrical modelling of EDA has to take into account, at minimum, one capacitor in parallel to the resistors of the corneum and the sweat duct (Fig. 18, Sect. 1.4.3.3), and EDA recording has to be performed using high-resolution AC instead of DC methods of measurement (Sect. 2.2.3.3).

The utilization of these models and recording techniques is valuable, therefore, for research into the causal mechanism of EDA and is able to supplement and correct some speculative conceptions which were developed on the basis of DC and potential measurements. Strengthened teamwork by physicists, engineers, dermatologists, and psychophysiologists, with the aim of improving measurement techniques, is necessary to overcome the specific problems of measurement using AC.

Part 2: Methods

The second part of the book discusses the different methods used for electrodermal recording. As mentioned in the introduction to Part 1, the observation of electrodermal phenomena is possible with relatively simple equipment, resulting in a variety of methodologies.

During the last two decades, there have been several attempts to standardize techniques of electrodermal recording (e.g., Lykken & Venables, 1971; Fowles, Christie, Edelberg, Grings, Lykken, & Venables, 1981). However, because of their narrow empirical basis, these recommendations do not provide sufficient information on what will result as a consequence of their violation. Therefore, Chapter 2.6 comprehensively reviews the recent state of discussions concerning the use of various concepts in recording and evaluation of EDA.

As a supplement to the electrophysical and system-theoretical fundamentals given in Section 1.4.1, Chapter 2.1 outlines the basic principles of measurement techniques for electrodermal recording. Even for those experienced in the measurement of biosignals, specific problems may arise in EDA recording, and these are discussed in the Sections 2.1.3 and 2.1.4.

Measurement techniques, including recording techniques and analysis of data, are outlined in Chapter 2.2, followed by methods of parametrization in Chapter 2.3. Chapter 2.4 discusses the various influences of physical as well as nonelectrodermal physiological influences, including age, sex, race, and heredity. Data concerning distributions and reliabilities of, and interrelationships between, the different electrodermal parameters are given separately for the different measurement techniques in Chapter 2.5.

The reader who only wants basic knowledge of the most important principles of measurement and evaluation techniques is referred to the final sections of Chapters 2.1 to 2.3, which contain short summaries of the appropriate standards together with cross-references for more details.

2.1 Basic issues

The fundamentals of measurement techniques reported here are restricted to the problems that appear in circuitry which measures EDA. Since endosomatic EDA measurement does not require specific circuitry (Sect. 2.2.3.1), except for obtaining phasic reactions with higher resolution (Sect. 2.1.3), the following descriptions refer mainly to exosomatic EDA recording techniques.

The electrical current necessary to perform exosomatic EDA recording can be applied on skin by either DC or AC. Because the latter is not often used in electrodermal recording, Sections 2.1.1 to 2.1.3 discuss DC measurement only, and Section 2.1.5 discuss AC recording separately. This section includes techniques for pulse spectrum or transient analyses (Sect. 1.4.1.4), which can be regarded as a special case of AC mea-

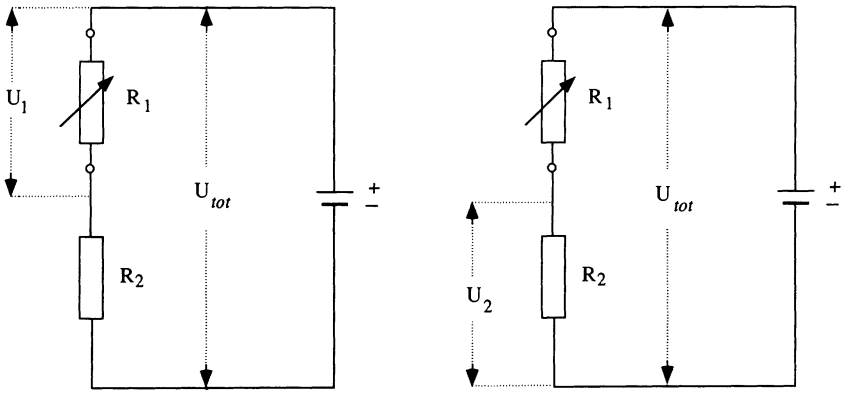

Figure 22. The quasi-constant current method (left) and the quasi-constant voltage method (right) of exosomatic EDA measurement. U_{tot} is the source voltage as applied to the system, and U_1 and U_2 are the measured partial voltages in the measurement of the variation of the resistance R_1 of the skin. R_2 is a fixed reference resistor.

surement, since the current does not flow continuously in one direction but switches on and off. Section 2.1.4 outlines specific problems of coupling, amplification, and filtering in electrodermal recording devices. Disregarding these features is likely to produce considerable inaccuracy of measurement and adulteration of the signal.

The measurement techniques given in this chapter should enable users to form their own opinions concerning the quality of various EDA recording devices and to localize possible sources of measurement error in their own device, as well as to eliminate those errors. Since not every polygraph system provides EDA recording facilities, it will sometimes be necessary to construct the appropriate circuitry. In this case, even an experienced engineer should be aware of the specific problems, discussed in Sections 2.1.3 and 2.1.4, that arise from the wide range of the electrodermal signal. An electric circuitry for an EDA coupler to be used with a high-quality biosignal amplifier is given in Figure 33 (Sect. 2.2.3.2).

2.1.1 Principles of measurement

In this form of exosomatic measurement, two electrodes attached to the subject's skin and a system reference resistor are connected in series, as shown in Figure 22. A voltage source is applied, supplying a constant voltage U_{tot}. The fluctuations in the electrodermal system can be read through the variations of the partial voltages (Sect.

1.4.1.2). With the aid of such a voltage divider, two different methods of measurement can be applied:

(1) The quasi-constant current method (see beginning of Sect. 2.1.2): The voltage is measured on R_1, the resistance of the skin (left-hand panel of Figure 22). The measured voltage U_1 is in the same proportion to U_{tot} as R_1 is to R_{tot}, R_{tot} being $R_1 + R_2$ combined:

$$\frac{U_1}{U_{tot}} = \frac{R_1}{R_1 + R_2} \tag{25a}$$

Multiplying both sides by U_{tot} gives:

$$U_1 = U_{tot}\frac{R_1}{R_1 + R_2} \tag{25b}$$

Following Ohm's law, and with regard to $R_{tot} = R_1 + R_2$, gives:

$$I_{tot} = \frac{U_{tot}}{R_{tot}} = \frac{U_{tot}}{R_1 + R_2} \tag{25c}$$

When the system is calibrated so that the fixed reference resistor R_2 is much larger than the variable skin resistance R_1, I_{tot} can be regarded as largely determined through R_2. Fluctuations of the skin resistance R_1 hardly affect the strength of the flowing current I_{tot}. Therefore, a "constant current system" will result. When R_2 is much higher than R_1, the denominator of Equation (25c), can be regarded as being almost constant. The voltage U_2, taken from the skin, is then almost totally proportional to variations of the skin resistance R_1.

(2) The quasi-constant voltage method: The voltage is measured on R_2, the fixed resistor (right-hand panel of Figure 22). The measured voltage U_2 is in the same proportion to the applied total voltage U_{tot} as R_2 is to R_{tot}, R_{tot} being $R_1 + R_2$ combined:

$$\frac{U_2}{U_{tot}} = \frac{R_2}{R_1 + R_2} \tag{26a}$$

Multiplying both sides by U_{tot} gives:

$$U_2 = U_{tot}\frac{R_2}{R_1 + R_2} \tag{26b}$$

When a system is calibrated so that the fixed reference resistor R_2 is much smaller than the variable skin resistance R_1, then the current I_{tot} flowing through the

system, according to Equation (25c), is no longer constant: as R_2 is negligible in comparison to R_1, the current increases as the skin resistance R_1 decreases and vice versa. As the voltage dropping on R_2 is negligible in comparison to that on R_1, practically the whole voltage U_{tot} is applied to the skin. Therefore, a "constant voltage system" will result. When R_1 is much larger than R_2, the numerator of Equation (26b) can be regarded as negligibly small in comparison to the denominator. The voltage U_2 measured on the reference resistor is then almost wholly proportional to variations in the reciprocal conductance value G_1 of the skin resistance R_1.

A higher signal amplification is needed for the quasi-constant voltage method than for the quasi-constant current method, which can be explained with the following example. If the skin resistance decreases from 100 kΩ to 90 kΩ, then, under the quasi-constant current method following Equation (25b), with a reference resistor of R_2 = 10 MΩ and an applied voltage of U_{tot} = .5 V, the recorded voltage U_1 changes from 4.950 mV to 4.459 mV (i.e., a change of around 491 μV). Under the quasi-constant voltage method with U_{tot} = .5 V and a reference resistor of R_2 = 100 Ω, following Equation (26b), the recorded voltage U_2 changes from .499 mV to .555 mV. This is a change of 56 μV, which is about a tenth of the difference obtained by the quasi-constant current method.

Every voltage measurement in such a circuit, whether by voltmeter, oscilloscope, or with another amplifier system, causes a change of the phenomenon under investigation. This change is dependent upon the properties of the measuring instrument. A decisive influence is caused by the internal resistance of the measurement instrument and the input resistance of the amplifier. When measuring voltages, this resistance and the input impedance should be as high as possible, commonly known as "high-impedance" amplification.

The necessity of using a high-impedance voltmeter when measuring voltage can be illustrated with the help of the right-hand panel of Figure 7 (Sect. 1.4.1.2). When R_2 is regarded as the internal resistance of the meter, being used to measure the voltage that drops over R_1, the total resistance of this parallel circuit is calculated according to Equation (6e). As a result, the total resistance of the circuit and voltmeter system will be smaller than the resistance of the circuit alone, as represented through the resistor R_1 in the right-hand panel of Figure 7. Hence, the voltage which drops over the entire system and is shown on the meter is smaller than the voltage which drops over R_1 alone. As can be inferred from Equation (6e), the measurement error produced by the use of the voltmeter is smaller the higher R_2 (the internal resistance of the meter) is in comparison to R_1.

Even with the use of a high-input impedance of, for example, R_2 = 10 MΩ, a clearly perceptible error of measurement remains. If, for example, skin resistance drops 10 kΩ from 100 kΩ to 90 kΩ, the insertion of 100 kΩ for R_1 in Equation (6e) results in a measurement of 99.0 kΩ for R_{tot}; the insertion of 90 kΩ for R_1 results in 89.2 kΩ. The

measured difference is 9.8 kΩ, which in comparison to the actual change of 10 kΩ is an error of 2%.

2.1.2 Measuring with operational amplifiers

The methods of measurement described in Section 2.1.1 using voltage dividers have been referred to as quasi-constant current and quasi-constant voltage methods. For example, the voltage U_1 applied to the skin when using the quasi-constant current method (not shown for the sake of simplicity in the right-hand panel of Figure 22) is not fully constant, but is a result of the difference between the voltage U_{tot} from the voltage source and the voltage U_2 used for measurement, and varies in accordance with this difference. The greater the fluctuations in EDA, the more the voltage applied to the skin electrodes oscillates. The respective measurement error of both methods is also determined by the dependence upon the relationship between the resistance values of R_1 and R_2.

In order to avoid the possible measurement errors caused by voltage divider based circuits, Lowry (1977) suggested an active circuitry for measurement of EDA (as opposed to the use of a passive voltage divider) based upon an operational amplifier as shown in Figure 23.

The amplification factor k of an operational amplifier is determined through the relationship of its input impedance R_i to the feedback resistor R_f being necessary for stabilization, as follows:

$$k = \frac{R_f}{R_i} \tag{27}$$

The output voltage U_0 results from the product of the voltage U_{tot} on R_i and the amplification factor k, whereby U_0 is inverted in comparison to U_{tot}, as shown with a minus sign in Equation (28):

$$U_0 = -kU_{tot} = -\frac{R_f}{R_i}U_{tot} \tag{28}$$

Since the internal resistance of today's operational amplifiers is in the range of GΩs, the voltage U_i lying on the active input of the operational amplifier is practically determined by the relationship of R_i and R_f of the so-formed voltage divider. As the current is the same through both these resistors, Ohm's law can be applied so that:

$$U_{tot} - U_i = R_i I \tag{29a}$$

and

$$U_i - U_0 = R_f I \tag{29b}$$

Solving Equations (29a) and (29b) for I results in:

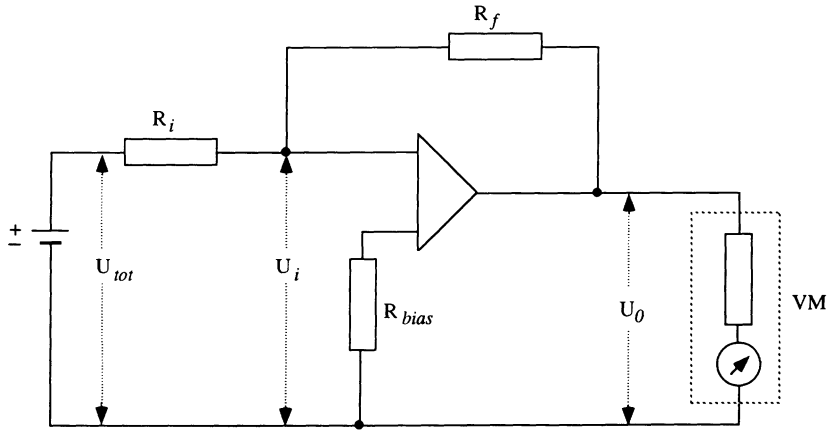

Figure 23. Operational amplifier for measuring electrodermal activity. U_i: Input voltage, U_0: Output voltage. R_i: Input impedance. R_f: Feedback resistor. R_{bias}: Reference resistor. VM: Voltmeter with its internal resistance. From R. Lowry (1977), Active circuits for direct linear measurement of skin resistance and conductance. *Psychophysiology, 14*, Fig. 2, p. 330. Copyright ©1977 by the Society for Psychophysiological Research. Reprinted by permission of the publisher and the author.

$$\frac{U_{tot} - U_i}{R_i} = \frac{U_i - U_0}{R_f} \tag{30a}$$

Multiplying out Equation (30a) gives:

$$(R_f U_{tot}) - (R_f U_i) = (R_i U_i) - (R_i U_0) \tag{30b}$$

Inserting of U_0 according to Equation (28) and solving for U_i gives:

$$U_i = \frac{(R_f U_{tot}) - (R_f U_{tot})}{R_f + R_i} = \frac{0}{R_f + R_i} = 0 \tag{31}$$

The voltage U_i lying on the operational amplifier input is therefore always set to zero by the amplifier. This enables a genuine constancy of current and/or voltage. U_{tot} is thereby taken from a constant voltage source (a stabilized power supply). Two different methods of measurement are possible with the use of this circuit:

(1) The constant current method: The skin is used as the feedback resistor R_f in the system. The current which flows through R_i and the skin is determined following Ohm's law as the quotient of the voltage drop $U_{tot} - U_0$ over both resistors and the sum of both resistors:

$$I = \frac{U_{tot} - U_0}{R_i + R_f} \tag{32a}$$

Inserting U_0 as taken from Equation (28) gives:

$$I = \frac{U_{tot} + \left(U_{tot}\frac{R_f}{R_i}\right)}{R_i + R_f} \tag{32b}$$

U_{tot} is bracketed in the numerator and $R_i + R_f$ can be shortened as follows:

$$I = \frac{U_{tot}\left(\frac{R_i + R_f}{R_i}\right)}{R_i + R_f} = \frac{U_{tot}}{R_i} \tag{32c}$$

Since R_i is a fixed value and U_{tot} is stabilized, the current I that flows through the skin will be constant. The current flow can be determined by the choice of appropriate values for U_{tot} and R_i. The voltage U_0, measurable on the output of the operational amplifier, is proportional to the skin resistance R_f following Equation (28), but with inverted polarity.

(2) The constant voltage method: The subject's skin is used as the input impedance R_i of the system. The voltage lying on the skin electrodes results from the difference between U_{tot} and U_i following Figure 23. The input voltage U_i is, however, always set to zero following Equation (31), so that a constant, because well stabilized, voltage U_{tot} lies on the skin. The voltage U_0, measurable on the output of the operational amplifier, is proportional to the reciprocal of the skin resistance R_i (i.e., the conductance value) but with inverted polarity.

The measurement of the voltage on the output of the operational amplifier can be done at a relatively low impedance level, as the measurement errors that result from the insertion of a meter in a voltage divider, and the consequent parallel connection of the meter's internal resistance and the system's resistance, do not occur here (Sect. 2.1.1). The reference resistor R_{bias} (Fig. 23) is introduced to set measurement error caused by bias currents at minimum.

Today's physiological equipment commonly employs differential amplifiers. These amplifiers work in the way shown in Figure 23, but they do not amplify the potential variations of a single input signal with respect to the ground common to output and input. On the contrary, they amplify the difference between two input voltages lying on two respective inputs of the operational amplifier. This voltage difference is independent of a reference point. Therefore, the problematic endosomatic contamination of exosomatic measurement values (according to Edelberg, 1967, p. 27f.) no longer exists.

2.1.3 Separating electrodermal reactions from levels

Electrodermal reactions normally display small variations compared to the total range of measurement (i.e., the possible tonic range). If the measurement system is set up in a way that level variations in their fullest possible range can be recorded without altering the amplification, EDRs to single stimuli and spontaneous fluctuations can only be recorded with a very small resolution and are therefore subject to a high measurement error (Sect. 2.1.4).

A simple compensation for the EDL is offered by the Wheatstone bridge circuit, composed of two voltage dividers connected in parallel shown in Figure 24. The potential difference between the central points of both voltage dividers is measured by a voltmeter, or over both inputs of an operational amplifier. One of the voltage dividers uses the subject's skin as resistor R_1 and a fixed resistor R_2; the other uses a variable resistor (a potentiometer) R_3 and a fixed resistor R_4. At the beginning of measurement the bridge is calibrated. In place of the unknown skin resistance R_1, a defined resistor must be inserted. R_3 is adjusted so that the potential difference between the two voltage dividers is zero. When an EDR occurs, this balance is disturbed and the potential difference can be read on the meter. As the corresponding variations are small compared to the possible range of level values, a significantly greater amplification can be used, as for the EDL, which leads to greater resolution of the EDR. Since as a rule the EDL will drift during the course of a measurement, it is necessary to adjust R_3 from time to time to prevent the recorded potential difference from exceeding the range of measurement.

Another method of suppressing the baseline component of EDA in recording is through the use of an AC amplifier (AC-coupled amplifier). Here a capacitor is inserted ahead of the input resistor R_i in the operational amplifier circuit (Fig. 23). The voltage time curve on the operational amplifier output runs in a similar manner to that in Figure 8 (Sect. 1.4.1.2); thus, the measurement signal shows only the variations in voltage. The amplifier reacts to a variation in the input voltage, as evoked through an EDR, resulting in a variation in the output signal. The new baseline level is not being transmitted but instead drops back down to zero after a certain amount of time. The transmission properties of the operational amplifier are characterized by its time constant (see Equation (10b)). Here, also, the range of measurement is small in comparison to the possible range of level variations, thereby enabling a greater amplification and a greater resolution of the EDR. The special problems of amplification that apply here are discussed in Section 2.1.4. It should also be noted that the amplitude and the course of an EDR signal as recorded by an AC-coupled amplifier differ from the corresponding parameters of the original EDR.

The EDR can be regarded electrophysically as the alternating voltage component of the EDA signal, the EDL being regarded as the direct voltage component. As the AC resistance of a capacitor is frequency dependent, as shown in Section 1.4.1.3, the EDR signal is changed in shape at the output of the operational amplifier. As this becomes

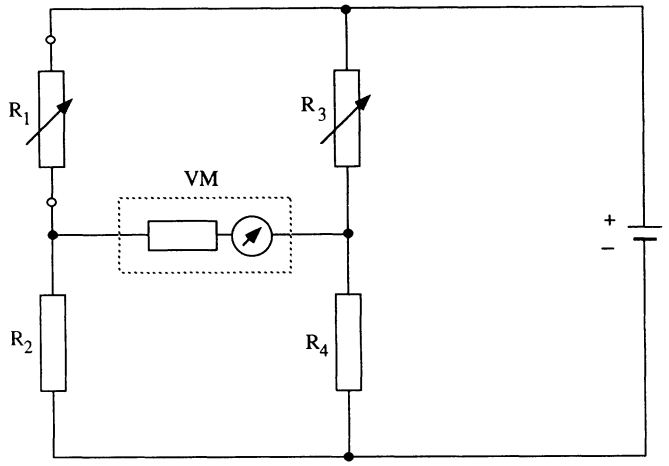

Figure 24. Wheatstone bridge circuit. R_1: Resistance of skin. R_3: Variable resistor for alignment of the level value, R_2 and R_4: Fixed reference resistors. VM: Voltmeter with its internal resistance.

critical only when the phase duration of the EDR signal approaches the time constant τ of the amplifier, measurement error can be minimized through an appropriate choice of τ. The time constant of the AC amplifier plays a significant role in the evaluation of the rise and recovery times of the EDR (Sect. 2.3.1.3). It should be at least 3 sec in the case of exosomatic measurement. Fowles et al. (1981) recommend time constants of over 6 sec to avoid distortion of the EDR amp. Following Edelberg (1967), time constants of 15 sec and above should be chosen for endosomatic measurements.

Elimination of the level component of the EDA is also enabled by "backing-off" circuits. Here, the direct voltage component is actively suppressed, similar to the adjustment of the input voltage U_i to zero in the operational amplifier (Fig. 23) however, not by the input amplifier but at a later time. An appropriate circuit is depicted in Figure 31 (Sect. 2.2.3.2). With such a system, to avoid loss of data, it is necessary that the experimenter observe the recording curve and change the backing-off voltage before the curve proceeds out of the measurement range. Therefore, additional circuits are in use which automatically adjust and record the backing-off voltage (Fig. 32, Sect. 2.2.3.2).

Special problems of amplification that occur with AC-coupled amplifiers as well as "backing-off" circuits are discussed in Section 2.1.4. With large enough time constants, the AC coupling leads only to a small change in the EDR; despite that, some authors recommend that this change be eliminated by transforming to a very large time constant, which can be done during computer analysis (Sect. 2.2.4.2; Thom, 1988). It must

be noted that, in the use of all decoupling methods for electrodermal reaction values described in this section, a new type of measurement scale is introduced for the EDA signal during the process.[28]

When resistance values are to be transformed into conductance values (Sect. 2.3.3.2), and this cannot be performed prior to the stage of data collection, tonic values will be required, as transformations with change values alone are not calculable without knowledge of the base levels (Sect. 2.3.3.2). It is therefore sometimes recommended to record the EDL on a parallel channel to the EDR, though with correspondingly smaller amplification.[29] Because in polygraphic recording often a very limited number of channels are available, special measurement procedures for the simultaneous recording of the EDL and the highly resolved EDR on a single channel have been developed. The EDA coupler of such a polygraph system enables the recording of the EDL as a sequence of impulses which overlie the AC signal of the EDR and whose spacing is proportional to the EDL. These impulses must in any case undergo a special treatment through subsequent automated processing of the EDR (Sect. 2.2.4.2).

2.1.4 Specific electrodermal recording problems

Even with the technology available nowadays, a relatively distortion-free high amplification of biosignals existing as voltages in the range of μV to mV can be obtained, some specific problems have to be discussed. Those problems appear in coupling, amplification, and filtering of the EDA signal.

In contrast to the couplers used for other biosignals, where the input amplifiers are connected together through the electric ground, the input section of an EDA coupler for exosomatic measurement must be fully electrically separated from all other amplifier inputs. Otherwise, the application of a ground electrode for another signal (e.g., EKG, EEG, etc.) will short-circuit the EDA measurement voltage applied to the skin and will thereby significantly reduce the EDA signal. The smaller the resistance between the ground electrode and skin in comparison to that of the EDA electrodes, the greater this reduction will be. In the case of simultaneous application of several EDA couplers to one subject, the input sections of the different EDA couplers must be fully electrically separated; otherwise, cross currents will appear.

Problems for the amplification of the EDA signal mainly result from its wide range, since the many possible inter- and intraindividual differences in EDLs result in a large recording range. In comparison, the fluctuations appearing as EDRs are relatively

[28] Although the respective EDL is built up of physical units with ratio scales, it is sometimes recommended that the EDL signal – like most psychophysiological variables – be treated as based only on interval scales (Levey, 1980; Stemmler, 1984), which would naturally also affect the performance of transformation (Sect. 2.3.3). However, EDR amplitudes obtained by AC-coupled amplification cannot be treated as based on a ratio scale anyhow.

[29] The reading off and manual recording of the placement of the potentiometer R_3 in Figure 24 cannot be recommended because of its error proneness.

small. When, for example, an SC-recording device covers a range from 0 to 100 μS, and fluctuations with amplitudes of .05 μS should be scored as SCRs (Sect. 2.3.1.2.3), the resolution in the analysis must be better than .0005. This, however, is not obtainable with paper analysis (Sect. 2.2.4.1); when 25-cm recording width is available for the EDA channel, with the whole 100-μS bandwidth being taken for a basis, SCR amplitudes of .125 mm must be recognized as such, which would also no longer be possible with a magnifying glass. With a computer analysis (Sect. 2.2.4.2) using A/D conversion with 12-bit accuracy, such resolution would only just be achieved; thereby, possible 4,096 digital scores would lead to a resolution of .025 μS/bit, and the minimal amplitude of .05 μS would be converted into 2 bits.

In order to achieve a sufficiently high resolution of the SCRs, it is possible with most EDA measurement systems to uncouple the EDR component of the signal from the measured EDL and to amplify it with a higher gain (Sect. 2.1.3). When this uncoupling ensues during recording by electrical compensation of the baseline component using a Wheatstone bridge (Fig. 24), the entire amplification range is available for the EDR. However, the SCL is lost during recording.

If an additional recording of the EDL is also attempted, then the entire EDL range must be covered through preamplification, and the EDR component must be uncoupled through a second amplification with the help of an AC amplifier, as described in Section 2.1.3. This leads to a problem of amplification, since only a very small part of the entire recorded EDL is of interest and, therefore, will be amplified. In the previous example, the minimal SCR of 0.5 μS takes up only .05% of the entire amplification range, if the first amplifier is set for an SCL range from zero to 100 μS. This requires an excellent signal-to-noise ratio.[30]

Another problem may appear in the filtering process in connection with the EDA signal amplification. As shown in Section 2.1.1, the amplitude of the EDR signal in exosomatic measurement can be in the μV range. Therefore, it is in the same range as noise which the skin, electrodes, and electrode cables pick up (Sect. 2.2.5.1). It is not possible to totally eliminate all such noise from the input signal. Therefore, in general, filtering is combined with amplification. As in the case of the EDR signal and

[30]Every amplifier produces noise. Good amplifiers have only .01% noise on average. The signal-to-noise ratio is measured in dB = 20 log (signal voltage/noise voltage). A noise ratio of .01% corresponds therefore to 20 log (100/.01) dB = 20 log 10,000 dB = 80 dB. If as in the previous example the signal takes up only .05% of the amplifier's working range, the signal-to-noise ratio is only 5:1 = .2. This corresponds to a signal-to-noise ratio level of 20 log 5 dB = 14 dB. In this case, the signal-to-noise ratio of an 80 dB-amplifier may drop to 14 dB. Furthermore, the above taken .01% noise ratio of the amplifier relates to the average noise amplitude; at particular times the voltage peaks, varying at random, can reach higher noise amplitudes, so that the noise component may sporadically appear in the range of the EDR signal. Since the high-frequency noise components are eliminated through the normally used low-pass filtering, only a noise component in the frequency range of the EDA signal remains and thus can simulate small EDRs. It is therefore necessary for the preamplification of the EDA signal to use an amplifier with a signal-to-noise ratio of at least 80 dB.

especially the EDL signal, when slow fluctuations are in question, low-pass filters are inserted in the EDA amplifiers.

The use of a low-pass filter can be explained with the help of Figure 23 in Section 2.1.2. When a capacitor C is connected in parallel with the feedback resistor R_f and the direct voltage is replaced by alternating voltage with a variable frequency f, the amplification factor, as calculated following Equation (27), changes in dependence upon f. This is because the AC resistance of the capacitor C decreases with a rise in frequency (Sect. 1.4.1.3). As the resistance of this parallel circuit becomes smaller with the resistance of C (Sect. 1.4.1.2), the feedback resistance of the operational amplifier becomes smaller with increasing frequency f. When the input resistor R_i remains constant, the amplification factor is directly proportional to the feedback resistance (i.e., it decreases when R_f decreases).

Through an appropriate selection of the values for R_f and C, the low-pass filter can be constructed with differing limiting frequencies f_{lim}, following the Equation $f_{lim} = 1/(2\pi\ R_f\ C)$, which, according to the construction of each filter, more or less steeply cuts off frequencies over the limit. Often, instead of the limiting frequencies, the time constant is given, $\tau = R_f\ C$ (see Equation (10b) in Section 1.4.1.2). Accordingly, time constants of .25, .5, 1 and 2 sec correspond to limiting frequencies of .64, .32, .16 and .08 Hz. Such low-pass filters eliminate a large part of possible noise in EDA measurement. By contrast, the EDRs with small time constants (i.e., with short recovery times) may be changed with respect to amplitudes as well as parameters of reaction shape (Sect. 2.3.1.3). How much such an RC circuit may distort an input signal is shown in Figure 9 (Sect. 1.4.1.3).

Though these filtering problems are more easily controlled in exosomatic DC measurement than in AC measurement, significant errors of measurement can arise in apparently simple EDA measurements with DC when the above problems are ignored. These errors are not normally noticed by the experimenter, because a characteristic-looking EDA signal can be obtained with below-standard experiment. Therefore, the standards described in this section should be especially noted when procuring an EDA measurement apparatus.

2.1.5 Measuring electrodermal activity with AC

In the simplest case, arrangements of measurement of the skin impedance and admittance (Sect. 1.4.1.3) differ from DC measurement in that an alternating voltage source is used instead of a direct voltage one. AC measurement devices can be constructed with a voltage divider (Sect. 2.1.1, Fig. 22) or with the help of an operational amplifier (Sect. 2.1.2, Fig. 23). The measured voltage is rectified and amplified; it can also be used to separate EDR and EDL components with a backing-off circuit (Edelberg, 1967; Sect. 2.1.3). Exosomatic AC measurement provides values for admittance or impedance but does not provide information concerning the phase angle φ, from which the capacitative properties of the skin could be determined.

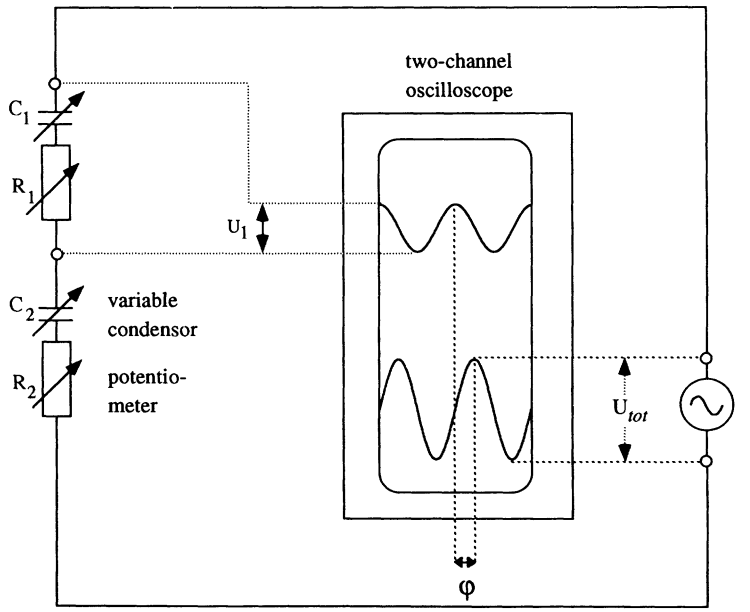

Figure 25. Device for the AC measurement of EDA. C_1 and R_1 serve as a simplified simulation of the skin. C_2 and R_2 are variable components of the measurement circuit. φ is the angle of the phase displacement between U_{tot} and U_1.

However, the most significant gain by using AC instead of DC measurement in electrodermal recording is the possibility to split impedance into its ohmic part R and the reactance X, or to split conductance into its ohmic part G and susceptance B, respectively. This can be performed when recording the phase angle φ as well (Sect. 1.4.1.3). In the present section, as already discussed in Section 1.4.1.3 and later in Section 2.2.3.3, the principle of impedance measurement is focussed on, because at present, skin admittance measurement with AC is rarely used. In addition, Salter (1979) states a preference for the constant current method, and, thereby, impedance measurement, in biomedical applications of AC measurement, because with the constant voltage method uncontrolled fluctuations in the current density can lead to non linearity in transmission behavior (Sect. 2.6.2). The equivalent admittance values are calculable, in turn, through the respective transformation (Sect. 1.4.1.3, 1.4.3.3, & 2.3.1.2.4).

One method given by Tregear (1966) to simultaneously determine X and R is shown in Figure 25. The figure shows a modification of the measurement principle

shown on the left-hand panel of Figure 22 (Sect. 2.1.1), where the direct voltage source is replaced with an alternating voltage source, the resistor R_1 is complemented by a capacitor C_1, and a variable capacitor and a potentiometer are inserted in place of R_2. The temporal course of the input voltage U_{tot} and the measured voltage U_1 are displayed together on an oscilloscope. Then the values of the variable resistor R_2 and the serially connected capacitor are changed until the voltage course of U_1 is in phase with U_{tot} and has half the amplitude. Then not only is the skin impedance the same as the impedances of the equivalent RC circuit, but it is also composed of the ohmic resistance and the reactance in the same manner. The ohmic component R is directly readable from the placement of the potentiometer, while the reactance X is calculable from the set value of the variable capacitor and the frequency of the AC according to the following formula:

$$X(f) = \frac{1}{2(\pi f C)} \tag{33}$$

Such an arrangement for measurement is only conditionally suitable for continuous recording of EDA. This is because every change in the weighting circuit requires a certain amount of time, as variations in the AC circuit resistance and capacitance influence each other (Sect. 1.4.1.3), so that the courses of short-period changes either in impedance and phase angle or in reactance and the ohmic component of the resistance cannot be reliably recorded with this method.

Moreover, when a more complicated electrical model of the skin is used (Sect. 1.4.3.2), further resistors and/or capacitors must be included in the compensatory branch of the voltage divider, which considerably lengthens the time necessary for trimming. Moreover, the accuracy of all these methods is dependent upon the accuracy of the manual trimming (Edelberg, 1967).

More recently, methods using analog computer components have been applied to the continuous recording of EDA with AC. With these, reactance and the ohmic resistance, with the use of a given frequency, can be directly calculated and constantly recorded from a comparison between an alternating voltage applied to the skin and the output voltage of the EDA measurement system.

Figure 26 illustrates the principle of such a circuit as described by Yamamoto and Yamamoto (1979, Fig. 2). An operational amplifier is fed by the output voltage $U(f)$, which corresponds to the output voltage U_0 according to Figure 23 (Sect. 2.1.2), with an alternating voltage source oscillating with the frequency f used instead of a direct voltage source. The output of a sine wave oscillator, which is in the same phase with the frequency f, is both directly and through a differentiator (which shifts the phase by 90^0, thus calculating $\cos 2\pi f$) multiplied by the output signal of an operational amplifier. On the first output $X(f) = Z(f) \sin \varphi(f)$ can be measured, as can $R(f) = Z(f) \cos \varphi(f)$ on the second output. With these two results, the AC characteristics of RC systems with a single given frequency f can be described (Sect. 1.4.1.3). In contrast to the method of photographic recording of Lissajous figures (Fig.11, Sect. 1.4.1.4) every

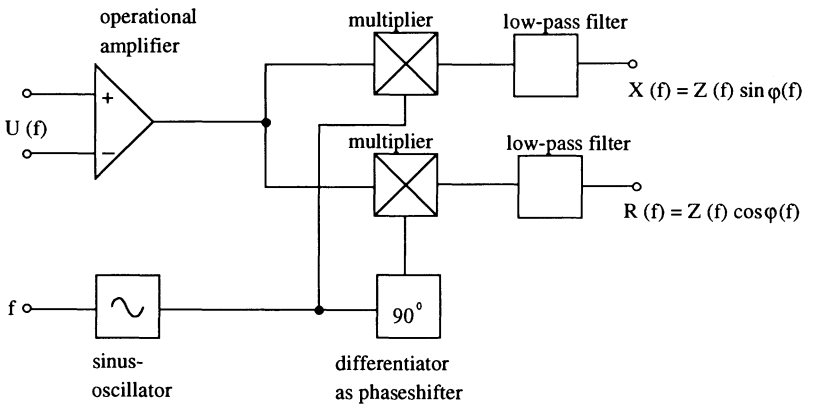

Figure 26. Circuit for the continuous recording of the reactance $X(f)$ and the ohmic resistance $R(f)$. (See text for explanations.)

.8 sec used by Yokota and Fujimori (1962, Fig. 5), such a continuous recording of $R(f)$ and $X(f)$ has the advantage of better quantification and control of the parameters of the electrodermal system. The phase voltmeter for EDA measurement described in Section 2.2.3.3 operates according to the principle shown in Figure 26. Through the appropriate transformations (Sect. 1.4.1.3), the susceptance $B(f)$ and conductance $G(f)$ can be continually calculated in place of the reactance and resistance.

For successive stimulation of the skin with varying alternating voltage frequencies (Sect. 1.4.1.4), it is necessary to go through a certain frequency range of the input voltage $U(f)$ using a voltage-dependent oscillator's directing voltage, which is recorded and calculated as well. Particularly suitable here is a "lock-in amplifier," with which the single frequencies of a certain range are given in succession in very small bandwidths to the system under investigation, and the responses are selectively amplified. With the use of a single lock-in amplifier, EDR courses can be recorded only when the measurement is repeated with all frequencies in question – several times during an EDR – which is hardly feasible with lower frequencies due to the attenuative behavior of the electrodermal system itself and the low-pass filter used (Sect. 1.4.1.4). Therefore, Morkrid and Quiao (1988, Fig. 2) used two lock-in amplifiers in parallel, together with a three-electrode arrangement, to measure skin admittance with two frequencies simultaneously.

Spectral analysis methods also allow continuous monitoring of the responses of the electrodermal system to stimulation by several alternating voltage frequencies. Here the direct voltage circuit shown in Figure 23 can be used to illustrate the principle (Sect.

2.1.2), in which case, according to Faber (1980), the constant voltage method should be preferred because of its faster regulability. Instead of continuously flowing DC, a test signal composed from a base frequency and its harmonics (Sect. 1.4.1.4) is applied to the skin (i.e., a square wave signal or sequences of Dirac impulses)[31]. Noise, which is composed of all frequencies, could also be used. The amplitude and phase angle can be determined for the entire spectrum from the output signal by means of Fourier analysis. This avoids problems with time requirements created by the lock-in technique, as the temporal resolution is only limited by the lowest frequency component used in the spectrum. In any case, a very high temporal resolution of the output signal is necessary with digitalization. Experience with such methods of EDA analysis is lacking.

Special technological problems that also exist for the recording of EDRs by means of AC are due to the highly selective amplification of the EDR, which is necessary because of the signal range (Sect. 2.1.4). In order to separate electrodermal level and reaction values, Wheatstone bridges can be used for single frequencies – either that given by Edelberg (1967, p. 33), which corresponds to the one shown in Figure 24 (Sect. 2.1.3) with a variable capacitor connected in parallel with variable resistor R_3, or that given by Schwan (1963, p. 367), in which the resistors R_2 and R_4 are supplemented by capacitors connected in parallel. Once the bridge is trimmed by the proper setting of the variable resistors and capacitors, fluctuations of X and R can be displayed in high resolution as deviations from the respective baselines.

When backing-off circuits are used to separate reaction from baseline levels with AC measurements, the resistive and capacitative components of the signal must be actively suppressed (Sect. 2.2.3.3). A technical solution for a continuous adjustment of backing-off circuits during an EDR has not yet been developed.

A basic problem of AC measurement of EDA appears during amplification through the filtering techniques usually used for noise reduction (Sect. 2.1.4). As the skin's response to applied alternating voltage or impulses can also lie in the noise range, a simple filtering of the EDA signal leads to a suppression of the relevant components of the output signal. Therefore, special care must be taken to thoroughly avoid noise from foreign alternating voltage sources, such as hum produced by power lines (Sect. 2.2.5.1), while using as little filtering as possible.

The development of methods for recording phasic electrodermal phenomena by means of AC measurement is still in its infancy (Boucsein et al., 1989). In contrast, AC measurement of tonic EDA in some applied areas such as dermatology (Sect. 3.5.2.1) is well developed and realizable at little technical expense (Sect. 2.2.3.3).

[31] Dirac impulses only exist in theory; in practice, a very narrow bandwidth square wave impulse with high amplitude is used. There may be danger of pain and burning.

2.1.6 Summary of recording principles

EDA can be measured either without externally applied voltage (i.e., the endosomatic method) or with application of DC or AC (i.e., the exosomatic method). By far the most commonly used method is exosomatic DC recording.

With the application of direct voltage, skin resistance measurements will result when current is kept constant, while skin conductance measurements will result when voltage is kept constant. Correspondingly, in the application of alternating voltage, if constant effective current is used, the result measured is the impedance, whereas the use of constant effective voltage will result in the admittance. Advantages and disadvantages of constant voltage and constant current methods are discussed together in Section 2.6.2.

Older principles of measurement based on the voltage divider (Sect. 2.1.1) are supplemented today by those based upon operational and differential amplifiers (Sect. 2.1.2), which not only guarantee genuinely constant voltage or current, but also enable minimization of measurement errors, owing to the avoidance of problems that stem from the internal resistance of the measurement apparatus.

A problematic feature of EDA measurement, as compared to other biosignals, is the relationship between the total width of the measurement range and the usually small variation of the signal during an EDR. To cover the total range of the EDA signal, EDRs may be separated from the EDL with the help of a Wheatstone bridge, backing-off circuits, or AC coupling (Sect. 2.1.3). Other specific recording problems resulting from amplification, especially from the amplifier's signal-to-noise ratio and from the danger of signal distortion through filtering, have been discussed in Section 2.1.3.

In summary, if the factors mentioned in this chapter are considered, adequate and disturbance-free EDA measurement systems can be built with the measurement and amplification technology available today. Significant new developments are needed in the area of AC measurement (Sect. 2.1.5), especially to record appropriate phasic fluctuations. Specifications for exosomatic EDA measurement with DC and AC are provided in Section 2.2.3.1.

2.2 Recording

Following the principles of electrodermal recording as described in Chapter 2.1, the following chapter gives detailed information on how to measure EDA. As far as possible, the standard methodology proposed by Venables and Christie (1980) and recommendations from a commission of the Society for Psychophysiological Research (Fowles et al., 1981) are used.

However, both articles recommend the sole use of constant voltage methodology for exosomatic EDA recording. As can be inferred from Part 3 of this book, these recommendations are not followed by all researchers, since recent publications point out the use of various methodologies. In addition, the above-mentioned recommendations

are to be questioned because both the constant voltage and constant current methods have advantages and disadvantages (Sect. 2.6.2). Therefore, as far as exosomatic EDA measurement is concerned, the following chapter focuses on the proposed standard methodology but includes constant current recording as well.

2.2.1 Recording sites

Though there are some special techniques using up to four electrodes (Sect. 2.2.6.4), electrodermal recording is mostly performed with two electrodes. As a rule, exosomatic techniques use two active sites, while endosomatic recording requires an active as well as an inactive site. There have been several proposals concerning the choice and a possible pretreatment of the appropriate sites. However, even with respect to those rather simple aspects of EDA methodology, there is no generally accepted standardization up to now.

2.2.1.1 Choice of sites

Most researchers make use of the palms or the volar surfaces of the fingers as active sites for electrodermal recording. The following reasons for this are given by Venables and Christe (1980):

(1) Electrodes can be fixed easily, and those sites are not susceptible to disturbance by movement.

(2) The size of available area is sufficient.

(3) Those sites are relatively free from scarring.

(4) Palmar sites show distinguished electrodermal activity.

Figure 27 shows the preferred palmar recording areas for exosomatic and endosomatic EDA measurement. Following Edelberg (1967), Venables and Christie (1980) recommend the medial phalanges of the index and middle fingers for bipolar recordings (sites A and B). The medial phalanges are less prone to scarring and to movement effects than the proximal ones, and the distal phalanges as well as the other fingers provide smaller areas for electrode fixing. Staying within the same dermatome with both electrodes may avoid EDA asynchrony, obtained in some individuals by Christie and Venables (1972). However, the importance of differences between adjacent dermatomes remains questionable, given the yet unexplained organization of the sudorisecretory cell bodies in certain segments of the spinal cord (Sect. 1.3.2.1).

Recently, Scerbo, Freedman, Raine, and Dawson (1992) found SCR amplitudes recorded from the distal phalanges being 3.5 times larger than those from medial sites, and SCLs being 2.08 times larger at distal sites as well. Furthermore, the distal sites

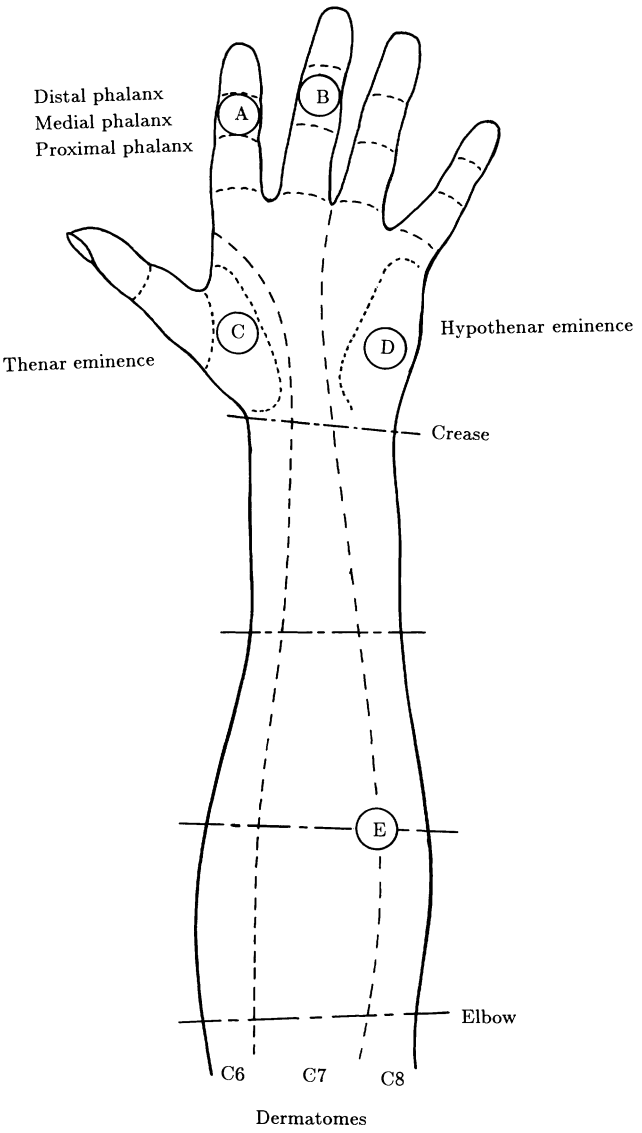

Figure 27. Preferred palmar or volar electrode sites (A–D), recommended position for the inactive electrode (E) used in endosomatic recording, and their relationship to dermatomes C6–C8. From P. H. Venables and M. J. Christie (1980), Electrodermal activity. In I. Martin and P. H. Venables (Eds.), *Techniques in psychophysiology*, Fig. 1.7. Copyright ©1980 by John Wiley & Sons, Ltd. Reprinted by permission of the publisher.

were more sensitive to habituation in a series of 10 orienting and defensive stimuli (Sect. 3.1.1.2 & 3.1.1.3). Therefore, the autors recommend using distal instead of medial finger sites in electrodermal recording.

Sometimes, however, difficulties may appear with fixing the electrodes at the fingers (e.g., if fingers are too slim or large surfaced electrodes are used; Sect. 2.2.2.1). In these cases, bipolar recordings may be taken from thenar and hypothenar eminences instead (sites C and D). According to Edelberg (1967), the SCL and the SCRs are even slightly higher at those sites as compared to the fingers. The two electrodes can also both be fixed on either the thenar or hypothenar eminence, as long as no direct electrical contact is established between them by a possible outpouring of electrode paste. The center of the palm is not recommended because of the difficulty of fixing electrodes firmly and its proneness to movements as the hand flexes (Venables & Christie, 1980). Also, the nondominant hand should be used, as it tends to be less calloused, the probability of the appearance of movement artifacts is lower (Sect. 2.2.5.2), and the dominant hand will be free for writing or other activity.

Systematic investigations comparing the SCL on various body sites have found that the scalp possesses almost four and a half times as much conductance as the inner sides of the fingers (Edelberg, 1967), presumably due to the numerous hair follicles. Corresponding comparisons of SRRs (Rickels & Day, 1968) have found, however, that all nonpalmar sites, except the feet, display long periods of electrodermal inactivity at the same time that spontaneous and evoked palmar EDRs can be obtained. The special suitability of the palmar and plantar skin surfaces for EDA recording is probably due to the irregular innervation of the respective sweat glands and their special role in "emotional" sweating (Sect. 1.3.2.4).

Therefore, in the case where both hands are needed for manipulation during the time of measurement, the next best recording sites are the plantar ones (i.e., the soles of the feet). Edelberg (1967) recommends instead a medial site on the side of the foot, over the abductor hallucis muscle (the extensor of the big toe's base joint) adjacent to the foot sole, and midway between the proximal phalanx of the big toe and a point directly beneath the ankle (Fig. 28).

This site displays, according to Edelberg (1967), the greatest SCRs in the foot area, and almost as great an SCL as the plantar surface. Rickles and Day (1968) also found that evoked SRRs recorded from below the ankle joint appeared in parallel to those taken from plantar sites. The sites shown in Figure 28 have an advantage over the soles of the feet because the socks do not need to be removed to place the electrodes but only pulled down. In addition, both rest and movement with the attached electrodes are possible, since the sites are not directly subjected to pressure during standing or walking. However, these measurements could be disturbed by muscular tension and pressure artifacts, a problem that does not arise with sitting subjects.

The inactive electrode necessitated for endosomatic measurement (Sect. 2.2.3.1) should be placed on a site that has the smallest potential difference between the skin surface and the body core and that is essentially inactive in terms of SPRs. According

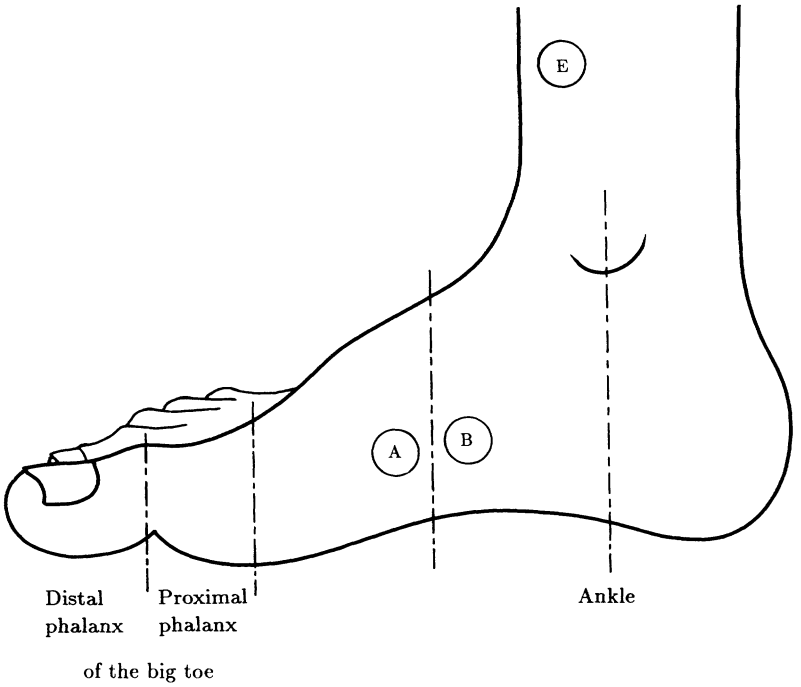

Figure 28. Medial side of the right foot with the recommended recording sites A and B for exosomatic recording, and position E of the inactive electrode for endosomatic measurement.

to Edelberg (1967), the inner aspect of the ear lobe is the most inactive reference site, but its use may introduce EKG artifacts when recording SP from the hands or the feet (Sect. 2.2.5.2). In addition, when physiological variables other than skin potentials are to be recorded, the subject is usually grounded on one site only, in which case the ground electrode should be placed as near as possible to the recording site for the biosignal showing the lowest voltage. Therefore, one has no choice but to place the ground electrode on the head when, for example, both EEG and SP are being measured. In this case an amplifier uncoupled from the common ground is necessary for the SP measurement (Venables & Christie, 1980) because, otherwise, scattering from EKG would appear in the SP recordings (Sect. 2.1.4).

According to Venables and Christie (1980), in palmar SP measurements, the inactive electrode should be placed on a slightly abraded site (Sect. 2.2.1.2) on the volar surface of the forearm (site E in Fig. 27) about two-thirds of the distance from the

wrist to the elbow. This site is even more likely to be electrically inactive than the site recommended by Edelberg (1967) on the ulnar bone about 4 cm underneath the elbow. In the case of SP measurements on the feet, Edelberg (1967) suggests a site on the skin about 3 cm above the ankle (site E in Fig. 28), which should be prepared in the same way as the forearm site.

2.2.1.2 Pretreatment of sites

Various recommendations have been made concerning pretreatment of the active electrodermal recording sites. Both the degree of hydration and the electrolyte concentration on the skin surface doubtlessly influence EDA. However, if the electrode paste is allowed to penetrate the skin for a certain time following the fixation of the electrodes (see end of Sect. 2.2.2.5), effects such as the decrease of NaCl concentration following washing with soap – as recommended for standardization by Venables and Christie (1980) – are neutralized. Washing with soap may cause a swelling of the epidermis and therefore a lowering of the SCL. Walschburger (1976) recommended washing with lukewarm water without soap and a final cleaning with a 70% solution of ethanol. In the author's experience, there is normally no need for a pretreatment of sites used for exosomatic measurement. In cases of extremely oily skin, cleaning of skin surface with alcohol may be necessary in order to attach the adhesive tape for the electrodes (Sect. 2.2.2.1).

In contrast, the site used for the inactive electrode in endosomatic measurement must be pretreated. The stratum corneum has to be removed to lessen the difference in potential between the site and the body core underneath the electrode. This can be done by light rubbing with fine sandpaper until a small, shiny pit can be seen on the skin by transverse illumination (Venables & Christie, 1980), which demonstrates that the stratum lucidum has been reached (Sect. 1.2.1.1).

In no case should abrasion be carried so far as to produce a wound (Venables & Christie, 1980). Great caution and some preliminary practice is required; therefore, the researcher should practice on his or her own skin first. Pain can result from contact of the electrode paste with the pretreated site, and following removal of the electrode a rash and/or light swelling may result. Therefore, one must weigh a lesser pretreatment, possibly lowering SPLs and SPR amplitudes, against possible unpleasantness for the subject.[32]

Some authors recommend the use of a type of dentist's drill to remove the stratum corneum (e.g., Shackel, 1959). Pasquali and Roveri (1971) describe a method (later automated by Zipp, 1983) by which the decrease of SRL can be measured during skin drilling, thereby avoiding an unnecessarily deep abrasion of the skin. Instead of the sur-

[32]According to Venables and Christie (1980), no differences of potentials between abraded and non-abraded forearm sites were observable with children, so when children are used as subjects, pretreatment can be omitted.

face excision, Burbank and Webster (1978) use a micropuncture technique for lowering the resistance of the stratum corneum. The skin- stripping technique (Sect. 1.4.2.1) is also principally suitable for removing the upper layers of the corneum.

2.2.2 Electrodes and electrolytes

In contrast to the localization and pretreatment of recording sites, a tendency toward standardization is already observable in the choice of electrodes and electrolytes in EDA measurement. Problems with the described arrangements can, however, arise with measurements that take longer than a couple of hours. These are described in detail in Section 2.2.6.1. Electrodes and electrolytes for further special purposes are described in Sections 2.2.6.3 and 2.2.6.4.

2.2.2.1 Forms of electrodes and their attachment

The EDA electrodes used today are disc electrodes that have the electrode surface on the bottom of a cylindrical plastic chamber that produces a space between the electrode and the bottom of the ring which is filled with the electrode paste containing the electrolyte (Sect. 2.2.2.5). Such an electrode is shown in cross section in Figure 29. It consists of a round silver plate about 6 mm in diameter on which a sintered silver/silver chloride (Ag/AgCl) layer has been deposited (Sect. 2.2.2.3).

The electrodes are usually attached to the skin with appropriate-sized double-sided adhesive collars. The adhesive surface of the collar is fitted to the electrode rim; then the chamber is held upside down and filled with electrode paste, avoiding air bubbles, and the surplus cream is removed with a spatula, while the protective cover still remains in place on the outer side of the adhesive ring. The cover is then removed and the electrode fixed on the skin, thus providing a precisely defined area of contact between the skin and the electrolyte, necessary, for example, for the constant current method (Sect. 2.2.3.2). However, a precise contact area will be lost if electrode paste gets between the adhesive ring and the skin,[33] which can easily happen, especially with attachment of electrodes to the fingers (Sect. 2.2.1.1). Venables and Christie (1980) recommend an alternative way of attaching electrodes by fixing the adhesive ring on the skin first and then fastening the paste-filled electrode to the other side of the ring. The electrode rim must not come into contact with the paste, as this can lead to detachment of the electrode. Therefore, some preliminary practice is indicated for this method. Contact of the electrode rim with the paste can be avoided through the sacrifice of a second adhesive ring. The electrode is prepared with the adhesive ring attached, which is then removed along with any excess paste; the filled electrode is then fastened to a second adhesive ring already fixed on the skin.

[33]The relative error due to seepage is dependent on electrode diameter. 1 mm of seepage increases the contact area to 2.25 times its original size with 4 mm diameter miniature electrodes, but to only 1.13 times with the use of 1 cm diameter electrodes (Venables & Christie, 1980).

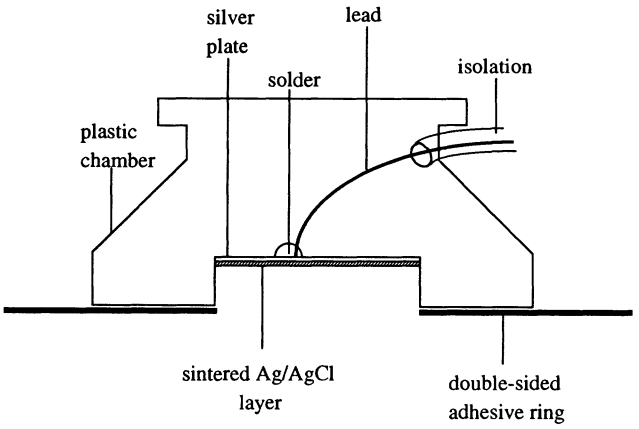

Figure 29. Cross section of Ag/AgCl electrode. The electrode surface consists of a silver plate which is covered by a sintered Ag/AgCl layer. It is soldered to an isolated lead and enclosed by a cylindrical plastic chamber which is attached to the skin with a double sided adhesive ring.

When using this method, it may happen that the surface area of the paste-filled electrode chamber does not exactly match the hole in the adhesive ring. But if the skin-electrolyte contact surface and the electrolyte/electrode surface (as shown in Fig. 30) remain constant, no distortion of measurement results is expected. The same holds for smaller air bubbles, which can result from incompletely filled electrode chambers, since the conductance of the electrode cream in the remaining paste bridges is so high that a somewhat reduced cross-sectional area in comparison to the total area does not matter (Fig. 30). The adhesive ring fixing the electrode can detach quickly with a strongly sweating subject. An additional wrapping of the fingers and electrodes with adhesive tape when hand movement is expected, as recommended, for example, by Venables and Christie (1980), leads to the danger of mechanical pressure upon the skin. Edelberg (1967) points out that changes in pressure may act as local stimuli producing Ebbecke waves, which must be regarded as artifacts (Sect. 1.4.2.3 & 2.2.5.2). In addition, through pressure on the electrode, variations in local circulation may be elicited and thereby cause reduction of the EDRs, especially of the positive component of the SPR; consequently, the pressure the electrode exerts on the skin should be as small as possible. Alternatively, the electrodes can be attached with histoacryl glue and later removed with acetone.[34]

[34]Such glue must be refrigerated. Warning: careless use can result in eye damage! Also, acetone will attack the rim of the plastic chamber of the electrode, which will roughen it and eventually damage the surface of the plastic chamber.

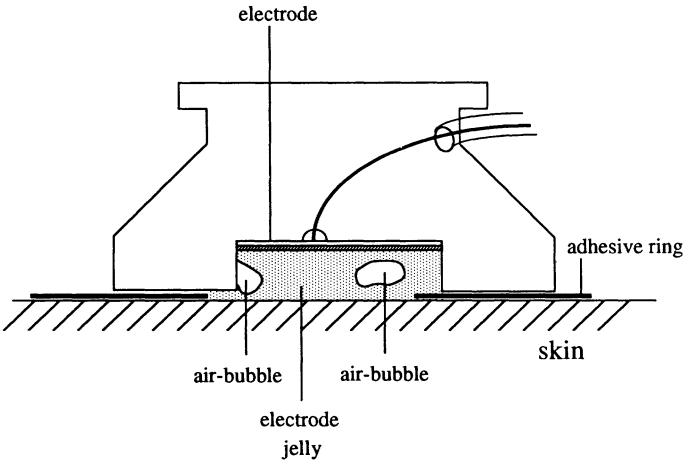

Figure 30. The effects of displacement of the electrode chamber on the adhesive ring attached to the skin, and of air bubbles in the electrode paste. (See text for explanations.)

If this method is used, an adhesive ring should be attached to the electrode first, then the electrode filled with cream and the overflow removed together with the ring, after which the electrode is fixed onto the skin using the histoacryl glue. An alternative technique is to fix the empty electrode to the skin with the glue first, then to fill the electrode with cream using a syringe. To apply this technique, two holes must be drilled through the electrode's plastic chamber on opposite sides, roughly 1–1.5 mm in diameter, one for the syringe and the other to let the air escape. The syringe must be completely filled with cream to completely rid the chamber of air (Andresen, 1987). In the case of long-term measurements (Sect. 2.2.6.1) collodium has also been successfully used for electrode attachment (Turpin, Shine, & Lader, 1983).

In order to avoid strain on the electrode, the electrode lead should be fixed to the skin at a distance of 2–3 cm from the electrode, using a strip of adhesive tape. Lasting contact of the electrode to the skin with constant contact area between the skin and the electrolyte must be ensured. The hand with the electrodes should be placed in a lightly bent, resting position, with the palm either up or down; a soft, thermally conductive underlay on which to rest the palm may be used (Walschburger, 1975).

2.2.2.2 Bias potentials and polarization of electrodes

Edelberg (1967) and Fowles et al. (1981) pointed out the following requirements for EDA electrodes:

(1) They should show a minimal bias potential (between pairs of electrodes).

(2) They should display little tendency toward polarization upon the passage of current, even when large current densities are applied.

Bias potentials are the differences of potentials that exist between two electrodes in the same electrolyte without application of voltage. The development of a bias potential is often incorrectly labelled polarization (Edelberg, 1967, p. 6). The bias potential can be measured by joining two paste-filled electrodes together in full surface contact. For endosomatic EDA measurement, the bias potential should be smaller than 1 mV (Venables & Christie, 1980; Fowles et al., 1981). Normally electrodes with smaller bias potentials also display a smaller drift over a period of time (Sect. 2.3.4.3). Fowles et al. (1981) recommended checking the bias potentials every two or three days, for which an amplifier system that can reliably measure between 100 μV and 10 mV is necessary.

Bias potentials influence exosomatic as well as endosomatic EDA measurements, although they play a significantly greater role in the latter, in that they can easily amount to an error of 100% in SPL readings (Edelberg, 1967). Electrodes which are used for endosomatic measurement should be checked every 3 days; if they display bias potential of more than 3 mV they should be replaced (Fowles et al., 1981). For exosomatic measurements, bias potentials between 3 and 5 mV can be tolerated.

Polarization refers to the development of a counter electromotive force (or back e.m.f.; Sect. 1.4.2) in biological membranes and at the interface between the electrode and electrolyte, if an external voltage is applied (Fowles et al., 1981). It is a result of either energy barriers on the electrode surface created by oxidation-reduction interaction or of ion transport over these borders being limited by the ion-diffusion rate (Venables & Christie, 1980). Polarization voltages influence exosomatic measurements by counteracting the applied voltage. According to the results from Barry's (1981) investigation, polarizable electrodes hardly influence the SRR, but a possible influence on the SRL cannot be ruled out.

Polarization effects can be checked in vitro or in vivo. An in vitro method of measuring polarizability of electrodes has been described by Edelberg (1967) as follows: two electrodes, each with a surface area of 1 cm^2, are placed 1 cm apart in a .1 n NaCl solution[35] whose specific resistance is around 100 Ω per cm. The voltage that appears between both electrodes should be 1 mV at most; higher differences in potential are due to polarization effects. Fowles et al. (1981) recommended a method which can be used in vivo during EDA measurement with DC: the polarity of the electrodes is reversed, and the time necessary to reach the original SCL or SRL is noted. After some practice, the shape of the corresponding transients can be judged as being due either to polarization of the electrodes or to normal polarization of skin membranes. However, quantifiable results are not obtainable with this method.

[35]This corresponds to a .1 molar solution of the monovalent NaCl (i.e., .58 g NaCl dissolved in 100 ml water).

A systematic reversal of polarity during measurement additionally ensures a better comparability of the electrodes over the long term, since if DC is used together with Cl containing electrolytes, one electrode will become chlorinated and the other dechlorinated during EDA recording. Though there is some probability that simply the effect of random placement of the electrodes over the long term will prevent systematic polarization effects, the reversal of polarity recommended here will be safer.

2.2.2.3 Choice of electrodes and set up

Today sintered silver/silver chloride (Ag/AgCl) electrodes are practically the only standard EDA electrodes in use. These are the so-called reversible electrodes which are made from a metal in contact with a solution of its own ions (Fowles et al., 1981). Such electrodes display the smallest bias potentials and are practically unpolarizable (Sect. 2.2.2.2).[36] According to Edelberg (1967) compared to the zinc/zinc chloride and zinc/zinc sulphate electrodes, Ag/AgCl electrodes require only a NaCl electrolyte solution that is tolerable to the skin (Sect. 2.2.2.5). Fowles et al. (1981, footnote 3) report that zinc/zinc sulphate electrodes are also unpolarizable. While these are not commercially available, they are simple to construct. However, because of problems with the exact composition for an adequate electrode cream as well as with maintenance, these electrodes are not recommended by Fowles et al. (1981).

The contact surface area of the electrolyte with the skin should be 1 cm^2 according to Fowles et al. (1981), as long as the recording site permits this, because proportional error increases with possible seepage of the electrode paste (Footnote 33, Sect. 2.2.2.1), and problems of linearity may appear with the use of the constant current method (Sect. 2.6.2). However, the commercially available standard electrodes[37] have a surface area of only around .6 cm^2.

The effect of variation in contact surface area is still a matter of discussion. Mitchell and Venables (1980) provided evidence from their systematic investigations of the relationship between electrode contact areas of .017 to .786 cm^2 and SCL as well as SCR amp. They find that the electrode-skin contact area has a minimal effect, at least within the range tested. They recommend that for finger recording the electrode-skin contact area should be around .8 cm in diameter (ca. .503 cm^2). They state, however, that the influence of the size of the area upon measurement is so small that electrodes of other sizes could also be used (Sect. 2.2.3.2). More recent results reported by Mahon and Iacono (1987), however, indicate a marked linear dependence of the SCL and

[36]The Beckman biopotential electrodes (see next footnote) show bias potentials of less than 250 μV and polarization potentials of less than 5 μV (Venables & Christie, 1980). Ag/AgCl electrodes can also be homemade, albeit unsintered (cf. Venables & Christie, 1973, p. 107), and good results are obtainable, but the process is very expensive, pure silver (99.99%) being necessary. Usually commercially available Ag/AgCl electrodes are preferred.

[37]The standard-biopotential-electrodes from Beckman Instruments have a contact area of .636 cm^2. In-Vivo-Metric-Systems electrodes, which are also provided alternatively together with a plug connection between the electrode and the cable, are of approximately the same size.

the SCR amp. upon the electrolyte-skin contact area (Sect. 2.3.3.1), so that a standardization of the electrode area appears desirable. In the case of endosomatic EDA measurement, the electrode size plays a subordinate role (Sect. 2.2.3.1).

2.2.2.4 Cleaning, maintenance, and storage of electrodes

In order to prevent damage on the Ag/AgCl layer, the electrodes have to be cleaned very carefully. EDA electrodes must never be mechanically cleaned or dried under any circumstances. The electrodes must be fully washed under flowing water immediately after use; a water-pik can be used. In order to avoid calcium deposits from tap water, electrodes should be rinsed additionally with distilled water. Air drying should follow, possibly with the help of a fan. The recent recommendations for electrode storage proposed by Tassinary, Geen, Cacioppo, and Edelberg (1990) state that sintermetallic Ag/AgCl electrodes should be stored in a mild NaCl solution with the leads shorted via a carbon rod also partially immersed in the solution.[38]

If electrodes are not continuously used, they should be stored in dry conditions. Venables and Christie (1980) recommend that before EDA measurement electrodes should be short-circuited and soaked for at least 24 hours in a solution of electrolyte of the type and concentration which is used in the electrolyte paste, allowing local reactions to take place before measurement.[39]

After some time, despite the greatest care, a black deposit of AgCl may appear on top of the grey Ag/AgCl layer as a result of chlorinization (Sect. 2.2.2.2). As long as bias potentials and polarizational tendencies do not increase too much, functioning of the electrode will remain unimpaired. Under no circumstances should mechanical removal of deposits be attempted.[40] If a layer of bright silver appears, the electrode can be rechlorinated (Sect. 2.2.2.3), but then the electrode coverage is no longer completely composed of a sintered Ag/AgCl layer.

2.2.2.5 Electrolytes and electrolyte media

Because they are hypertonic, electrode gels used for other biosignals such as the EKG, the EEG, and the EMG are not suitable for EDA measurement. Hypertonic gels have a higher conductivity than the epidermis, which is necessary for some biosignals in

[38]The evidence presented is not very compelling since the air-dry method was best overall (see Tassinary et al., 1990, Fig. 2, p. 239). Another problem with the proposed storage method is one from the present author's own experience. Long-term immersion in an NaCl solution led to a separation of the wire from the chamber at the chamber-wire junction. This problem with wire separation and the trouble and expense of the proposed method still leaves the air-dry method as most recommendable.

[39]The present author does not support this recommendation, since in his own experience the fluid may penetrate into the electrode chamber, thus exerting negative influences (e.g., corrosion) on the contact between electrode and lead.

[40]Sensor Medics recommends removing the deposit with dilute ammonium hydroxide. A five to one dilution with distilled water has been used with success (personal communication, A. Vincent, July 1990).

order to transmit potentials created in the body core underneath the skin to the electrodes with minimal loss of voltage.

In contrast, in EDA measurement the electrolyte-skin system must be disturbed as little as possible, as an interaction between skin and electrolytes can have a marked effect on variations in EDA. Barry (1981, footnote 1) showed that additional lowering of the skin resistance with hypertonic electrode gel invalidated the transformation of resistance into conductance units (Sect. 2.3.3.2). Edelberg, Greiner, and Burch (1960) also found that many gels contain multivalent ions such as calcium, zinc, and aluminium which may potentiate the skin conductance and lower the skin potential. Since both NaCl and KCl appear as salts with monovalent ions in the stratum corneum (Sect. 1.3.4.2.1), these two are suitable to use for electrolytes in EDA measurement. Since NaCl ions are preponderant in sweat, the use of a NaCl-based electrolyte can be expected to disturb the electrodermal system least.

As the NaCl concentration in sweat varies between .015 and .06 molar dependent upon the amount of sweating (Sect. 1.3.3.1), it is not possible to determine precisely the optimal electrolytic concentration in the electrode paste. Fowles et al. (1981) were convinced that if the NaCl concentration of the electrolyte is between .05 and .075 molar it is unlikely that NaCl will transfer out of the sweat into the paste, and thereby alter the electrolytic concentration significantly. Edelberg (1967) recommended a .05 molar concentration, which can be made by dissolving .29 g of pure NaCl in 100 ml of distilled water. Hygge and Hugdahl (1985) found no difference in size among SCRs with four electrode pastes differing in NaCl concentration, showing that small deviations from the recommended NaCl concentration can be tolerated.

Some authors use the monovalent KCl instead of NaCl (cf. Fowles & Schneider, 1978). Venables and Christie (1980) recommended, on the basis of their own laboratory experiments, .05 molar NaCl pastes for exosomatic and .067 molar KCl pastes for endosomatic EDA measurements. Schneider and Fowles (1978) suggested KCl as electrolyte for SP measurement (see the end of this section). Fowles et al. (1981) pointed out that KCl, above all, should be used for SPL measurement so that results published in the literature are comparable, especially with those from the Venables group, while NaCl is adequate for SPR measurement. Since the contents of commercially available gels – including the so-called isotonic ones – are not provided by the manufacturers,[41] investigators should follow the suggestion made by Fowles et al. (1981) to manufacture their own mixture.

As a base for the electrolytes, a number of hydrophilic and widely ion-free media are suitable (Edelberg, 1967), for example, agar (a type of gelatine made from algae),

[41]This is also true of the electrode cream "Synapse" made by Beckman Instruments which has been frequently used in EDA measurements. An analysis of this and other gels was made by Zipp, Hennemann, Grunwald, and Rohmert (1980), who found in "Synapse" a significant quantity of K and Cl ions in addition to Na ions. Grey and Smith (1984) reported that the Beckman paste has a NaCl concentration of 4.1 mol/l, and contains glycerol, gum tragacanth, and .5% benzyl alcohol (as preservative). An isotonic cream from Hellige, Freiburg (see Footnote 1 in Boucsein and Hoffmann, 1979) is no longer available.

starch, or methyl cellulose (the base of wallpaper glue but also used in food).[42] However, these media may cause problems, since their manufacture and storage require a controlled temperature. Furthermore, when agar is used, the SCL and SCR decrease with time, which is probably due to hydration of the corneum following blocking of the sweat gland ducts by the agar (Fowles & Schneider, 1974). Those types of electrode pastes are also very susceptible to bacterial degradation. Glycol, used by Edelberg, escapes from under the adhesive tape with which the electrode is fixed (Venables & Christie, 1980) and thereby builds up conductive bridges on the skin.

Unibase has become the standard medium for EDA electrolytes in North America (Fowles et al., 1981). Unibase is an ethylene polymer which requires an emulgator due to its hydrophobic properties. This white, smooth ointment base can take up to 30% of its own weight in water. Fowles et al. (1981) advise that to manufacture an EDA paste, 1 pound (i.e., 453.6 g) of Unibase should be mixed with 230 ml of physiological NaCl solution. This is .15 molar, or .9%, NaCl solution, which can be bought commercially or made by immersing 2.0 g of chemically pure NaCl in 230 ml of distilled water. Unibase and NaCl solution have to be well mixed using an electric mixer (to remove lumps) and allowed to stand for 24 hours. The resulting electrode paste has an approximately .05 molar NaCl concentration.[43] The paste should be kept cool (in the refrigerator), otherwise it degrades and can leak from the tube.

Despite all efforts to match electrolytes to the skin electrolyte concentration, and control of other factors such as the paste's consistency, the electrolytes can still affect the skin over time. Therefore, the time lag between electrode placement and recording should be considered as a possible source of error in EDA measurement. Since destabilizing effects such as drift can be expected at the initial phase of measurement, the electrodes should be placed at least 10 min, preferably 15–20 min, before the beginning of recording, so that the skin-electrolyte interface can stabilize. According to results from Campbell et al. (1977), who used microelectrode measurement in vitro (Sect. 2.2.6.4), the water concentration of the stratum corneum had matched that of the electrolytes and had stabilized after approximately 16 min.

Problems that arise with long-term measurements, where the electrodes stay attached to the skin for several hours, are discussed in Section 2.2.6.1. Here it appears that electrode creams based on polyethylene-glycol have an advantage over the stan-

[42]Grey and Smith (1984) used a homemade gel of .05 molar NaCl solution in methyl cellulose. However, they did not provide the correct formula of the ingredients they used (Clements, 1989).

[43]Unibase is produced in the U.S. by Warner Chilcott Laboratories, Morris Plain, NJ. Grey and Smith (1984) have published the following ingredients of Unibase: cetyl and stearyl alcohols, soft paraffin, glycerol, and, as preservatives, .0015% propyl hydroxy-benzoate, sodium citrate, and sodium lauryl sulphate. The relative quantities are not provided. The water content is 63,4%. According to Grey and Smith, Unibase contains .028 mol/l Na ions. The author has had this recipe chemically analyzed; the results were .07 mol/l Na and .045 mol/l Cl, the increased sodium being due to the Unibase itself (see previous footnote). The cream is free from K and Ca ions (less than .01 g/kg) and has a nearly neutral pH value of 6.5. The analysis was performed by B. Neidhart, Institute for Industrial Physiology at the University of Dortmund, Germany .

dard ointment-based creams, due to their lesser propensity to hydrate the epidermis. Schneider and Fowles (1978) recommend a mixture of 100 ml polyethylene-glycol and 100 g Unibase with .76 g KCl also added for SP measurements.

2.2.3 Measurement devices

The following three sections provide details for the methodology used in measuring EDA with the three different methods outlined in Section 1.4: Endosomatic measurement (Sect. 2.2.3.1), exosomatic measurement with DC (Sect. 2.2.3.2), and exosomatic measurement with AC (Sect. 2.2.3.3). Since the reader probably already has access to equipment for EDA measurement, the following sections will give examples of appropriate devices, and their features can be compared with the reader's own equipment.

2.2.3.1 Endosomatic recording

In contrast to the exosomatic recording techniques, endosomatic EDA measurement does not require an external voltage applied to the skin, since only potential differences are recorded. However, it is recommended that one active and one inactive site be used, as already mentioned in Section 2.2.1.1. If two active sites are used, SPL will be rather low, and SPRs will look similar at both sites; that is, there will be no marked shift in potential differences between them. The appropriate inactive sites for SP recordings at the palms of the hands and on the feet are marked by E in Figures 27 and 28. Though those sites are relatively inactive, an interindividual and intraindividual comparability of SP recordings is given only if the corneum is extensively abraded (Edelberg, 1967). Thus, skin-drilling methods are recommended to prepare an inactive site as described in Section 2.2.1.2, which will reduce skin resistance from about 1 MΩ to 100 kΩ (Venables & Christie, 1980).

Endosomatic recording of EDA requires an amplifier with at minimum 1 MΩ input impedance; Venables and Sayer (1963) recommended 5 MΩ. The necessity for a high-impedance amplifier can be shown using the left-hand panel of Figure 7 (Sect. 1.4.1.2). If the skin is regarded as a voltage source with its internal resistance R_2, and R_1 is regarded as the amplifier's input impedance over which the voltage changes that are produced by the skin are read off, R_1 and R_2 form a voltage divider (Sect. 2.1.1). Hence, R_1 should be as big as possible compared to R_2, to ensure that the greatest proportion of the voltage measured descends over R_1, thus enabling it to be available for measurement and amplifying. Today, polygraph equipment with very high input impedances are available, so there is no need to give a specific circuit diagram for an SP amplifier.

However, some additional problems may arise in using standard amplifier techniques for SP recording. First, there may be no way to lower the amplifier gain enough for recording SPL, which may require a range down to 500 mV/division. Thus, an attenuation of amplification may become necessary. Second, considering reasons men-

tioned in Section 2.2.1.1, an SP amplifier is required whose ground is separated from the ground of amplifiers recording other low-voltage biopotentials (e.g., EEG). If the subject is not grounded otherwise (e.g., by the use of simultaneous EKG or EEG recordings), the subject must be additionally grounded to prevent power line noise (Sect. 2.1.4 & 2.2.5.1). Third, because SPRs are of small size compared to the whole bandwidth of the SP signal, a backing-off control is required to measure SPRs with satisfyingly high resolution (Sect. 2.1.3). Bridge circuits can not be used because an external voltage is not available. Therefore, backing off has to be performed by adding a potential of equal value but of opposite sign to the SPL, so that SPR is amplified at a higher gain around an arbitrary zero point. Another effect of this backing-off procedure is to reduce the current flow in the circuit and hence lessen the proportion of voltage which is dropped across R_2, which will drive the apparent input impedance of the amplifier up to 10 MΩ and reduce measurement error to 1% (Venables & Christie, 1980). A backing-off circuit which allows compensation as well as simultaneous recording of SCL is recommended by Venables and Martin (1967a). If SCL is not being recorded, an AC-coupled amplifier may be used, which must, however, have a rather high time constant of 30 sec minimum, to prevent deformation of the signal. Because no external voltage is applied, there is no danger of polarization effects on the boundary between electrode and electrolyte (Sect. 2.2.2.2). However, due to its small bandwidth of 0–3 Hz, the signal is superimposed inseparably by possible electrode drifts (Sect. 2.2.2.5). Therefore, for SP recording, the use of electrodes free from bias potential is strongly recommended (Sect. 2.2.2.2).

The electrode diameter as well as the delimitation of the contact area between skin and electrolyte play a subordinate role in SP recording. By contrast, differences in skin temperature between sites may result in error potentials up to 2 mV (Venables & Christie, 1980), because they exclusively influence the active site (Venables & Sayer, 1973). Temperature differences are likely to occur between sites at great distance from each other, for example, the distance between the palm and the upper forearm. Additionally, an increase of electrode temperature may result in error potentials up to 450 $\mu V/^0C$. Venables and Sayer (1973) report an electric circuit for the compensation of temperature effects in SP recording.

Problems with bias potentials can be minimized with careful selection and maintenance of electrodes for SP recording (Sect. 2.2.2.2). The possibility of using KCl paste instead of NaCl paste, especially in SPL recording, has already been mentioned in Section 2.2.2.5.

2.2.3.2 Exosomatic recording with DC

DC measurement of EDA is the most frequently used method for electrodermal recording. Its physical principles, as well as the advantages and disadvantages of the various recording techniques, were described in Chapter 2.1. A description of a skin conductance coupler that uses appropriate methodology – with the exception of filter

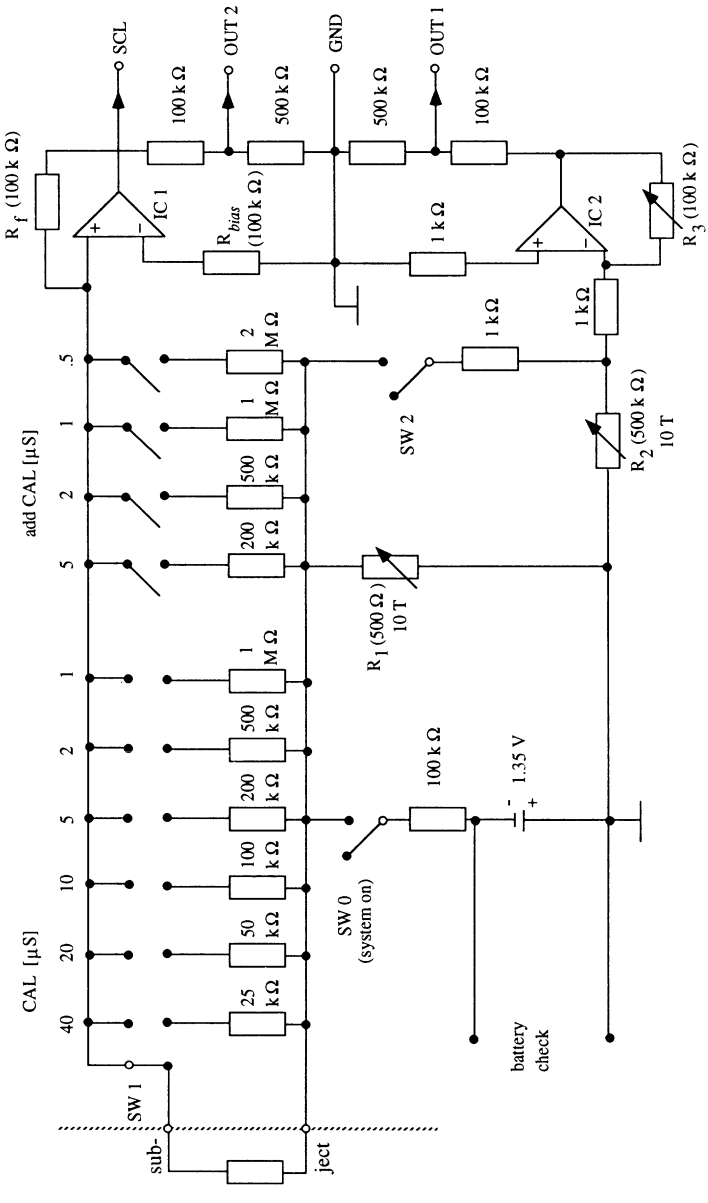

Figure 31. Active skin conductance coupler. (See text for explanations.) Reprinted from P. H. Venables, and M. J. Christie (1980), Electrodermal activity. In I. Martin, and P. H. Venables (Eds.), Techniques in psychophysiology, Fig. 1.13. Copyright ©1980 by John Wiley & Sons, Ltd. Reprinted by permission of the publisher.

characteristics (Sect. 2.1.4) – is given by Venables and Christie (1980; Fig. 1.13). The circuit for this active coupler, depicted in Figure 31, is derived from one presented by Lowry (1977), incorporating some of the operating features of the passive circuit described by Venables and Christie (1973).

The circuitry shown in Figure 31 enables continuous DC recording of SCL as well as SCR with high resolution, using a constant voltage source.[44] SW0 connects a 1.35 V mercury cell battery to the circuitry (system on), whereas SW1 selects calibration (CAL) or subject to be operated. CAL allows dummy loads of 1, 2, 5, 10, 20, and 40 μS to be connected in place of an actual subject. Additionally, pressbutton switches allow conductance values of .5, 1, 2, and 5 μS to be added during the course of recording (add CAL). The resistors providing those dummy load conductances or the actual subject are equivalent to R_i in Figure 23 (Sect. 2.1.2). The operation amplifier IC1 is fed back by R_f, and referenced by R_{bias} to ground. The differential amplifier of the recording system for SCL is connected to outputs SCL and GND.

The lower-right part of the circuitry shown in Figure 31 provides additional simultaneous recording of SCR with higher resolution between OUT1 and OUT2. The ten-turn potentiometers R_2 and R_3, together with the network around the amplifier IC2, provide a manually operated backing-off system, which is switched on by SW2. Instead of manually operated suppression, an automatic high-resolution evaluation of SCR may be obtained with the use of an AC-coupled amplifier or a digital operating autosuppression device described later (Fig. 32), which is connected to outputs SCL and GND.

The 1.35 V mercury cell together with the 100 Ω resistor and the ten-turn potentiometer R_1 provide the voltage appearing across the subject to be set at .5 V. The use of a constant .5 V stems from a recommendation made by Edelberg (1967, p. 19), who observed the voltage/current curves as being linear below an impressed voltage of .8 V across a single recording site. Since in exosomatic DC measurement of EDA, bipolar recording is predominant, the applied voltage is halved according to the principle of the voltage divider (Sect. 2.2.1). Hence, across each single recording site, only half of the applied voltage is dropped, given that the resistances of the sites are approximately equal. In spite of this, a total voltage of .5 V has been introduced as a standard for constant voltage DC measurement (Lykken & Venables, 1971). Fowles et al. (1981) state that in this case both sites will show .25 V potential difference, given the same SRL at the sites.[45]

The calibration procedure of the coupler depicted in Figure 31 is as follows (cf. Venables & Christie, 1980, p. 58):

(1) Set sensitivity of recording instrument (e.g., polygraph) to 5 mV/cm.

[44] Since Lykken and Venables (1971) recommended skin conductance as being the appropriate unit for EDA measurement, constant current recording went out of use in most psychophysiological laboratories. However, because of the lower amplifier gain required, constant current methods are frequently preferred in field applications (Sect. 2.1.1 & 2.6.2).

[45] Though Venables and Christie (1980, p. 40) argue for the application of .5 V across each active site, following a suggestion by Edelberg (1967), their circuitry depicted in Figure 31 provides a total of .5 V.

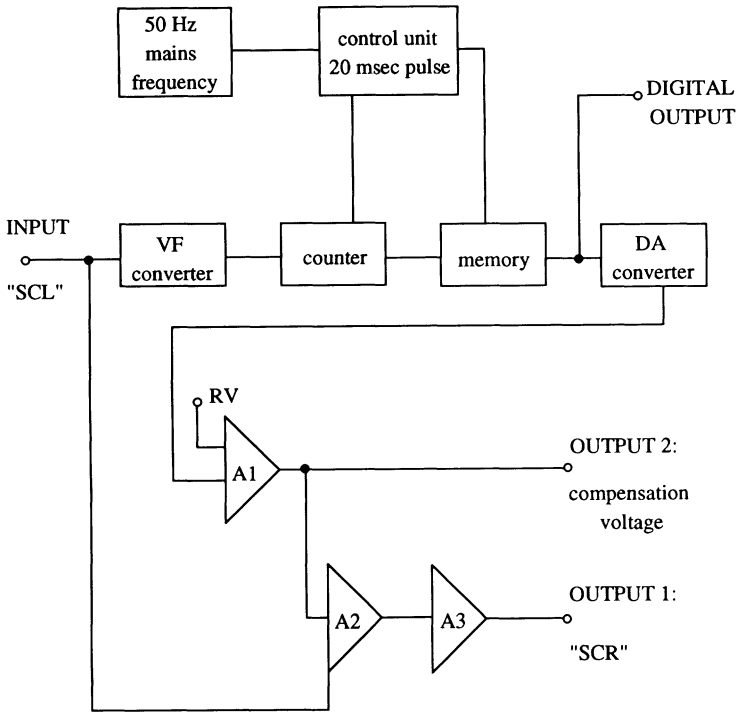

Figure 32. Block diagram of the automatic voltage suppressor. Adapted from W. R. Simon and R. W. G. Homoth (1978), An automatic voltage suppressor for the measurement of electrodermal activity. *Psychophysiology, 15*, Fig. 1, p. 503. Copyright ©1978 by the Society for Psychophysiological Research, Inc. Used with permission of the publisher and the first author.

(2) Select dummy load value (CAL) of 20 μS with SW1.

(3) Switch on coupler with SW0 and adjust R_1 to achieve a pen deflection of 1 cm. The voltage across the dummy load is then .5 V, and the current is 10 μA.

(4) Set dummy load to 40 μS, switch on suppression circuit with SW2, turn the zero suppression R_3 potentiometer to its maximum. Return pen to zero deflection by adjusting the suppression calibration control potentiometer R_2.

(5) Select subject with SW1, with an unconnected subject (i.e., with an open circuit). Return zero suppression potentiometer R_3 to zero and check that the pen gives zero deflection.

Steps (4) and (5) calibrate the zero suppression control R_3 to give 4 μS per turn of the potentiometer. If other values are introduced in step (4), the potentiometer R_3 may be calibrated with other values (e.g., if the dummy load is set to 10 μS R_3 calibration would be in terms of 1 μS per turn). The polygraph indicates 20 μS/cm with the sensitivity of 5 mV/cm selected in step (1). If the polygraph amplifier gain is increased to, for example, .05 mV/cm, an increase of sensitivity to .2 μS/cm results. Thus, following from the restricted width of the recording channel (normally 4–5 cm), the SCR output with zero suppression and higher resolution (Fig. 31) has to be used for recording with higher polygraph amplifier gain (Sect. 2.1.3).

In addition to the coupler circuitry depicted in Figure 31, Venables and Christie (1980, Fig. 1.11) provide a block diagram of a complete SC recording system with automatic suppression of SCL, the elicitation of temperature (Sect. 2.4.2.1), and calibration pulses for computer analysis (Sect. 2.2.6.2). In their diagram, the output SCL is fed into the automatic SCL suppression system developed by Simon and Homoth (1978), schematically depicted in Figure 32.

As mentioned above, the automatic voltage suppressor in Figure 32 is used together with the active skin conductance coupler depicted in Figure 31. The voltage that corresponds to the total SC (which appears between SCL and GND on the output side of the SC coupler depicted in Figure 31) is fed into a voltage-to-frequency converter (VF converter) with a dynamic range from .01–10 V. It is thereby transformed into a pulse rate which is fed into the counter during a time interval of 20 msec. Those 20 msec pulses are deduced from the 50 Hz AC power frequency, the rise of the pulses trigger the storage in memory, and the contents are converted by a digital/analog (D/A) converter to analog voltage, which is available until the next pulse from the control unit arrives. Additionally, a digital output of SC is provided.

The output of the D/A converter is reduced by an adjustable reserve voltage (RV) in the amplifier A1 and is subtratced from the input signal by the amplifier A2. The resulting voltage is amplified in A3 by a factor of 100 and thus gives a high resolution SCR in output 1. The compensation voltage which comes from A1 is led to a separate output 2 and can be recorded and processed separately. After adjustment of the RV, the compensation voltage varies in discrete and reproducible steps. Thus the original signal of the SC can be exactly reproduced by adding output 2 to output 1.[46]

A simple coupler for exosomatic DC recording can be used in combination with a high quality biosignal amplifier (e.g., for EEG measurement). The device depicted in Figure 33 uses a special integrated circuit (LM 10) to obtain a highly constant voltage U_{ref} = .5 V. By means of an operational amplifier circuitry OP 1 (see Fig. 23, Sect. 2.1.2), the current through the subject is converted to a negative voltage $-U_{SCL}$, which is proportional to the SCL, showing a sensitivity of .5 V/μS. This output voltage is further amplified and inverted using another operational amplifier OP 2, resulting in

[46]Simon and Homoth (1978) state an error of .4% of the D/A converter's output voltage. A circuit diagram of their voltage suppressor is given in their Figure 2, together with a list of components in their Table 1.

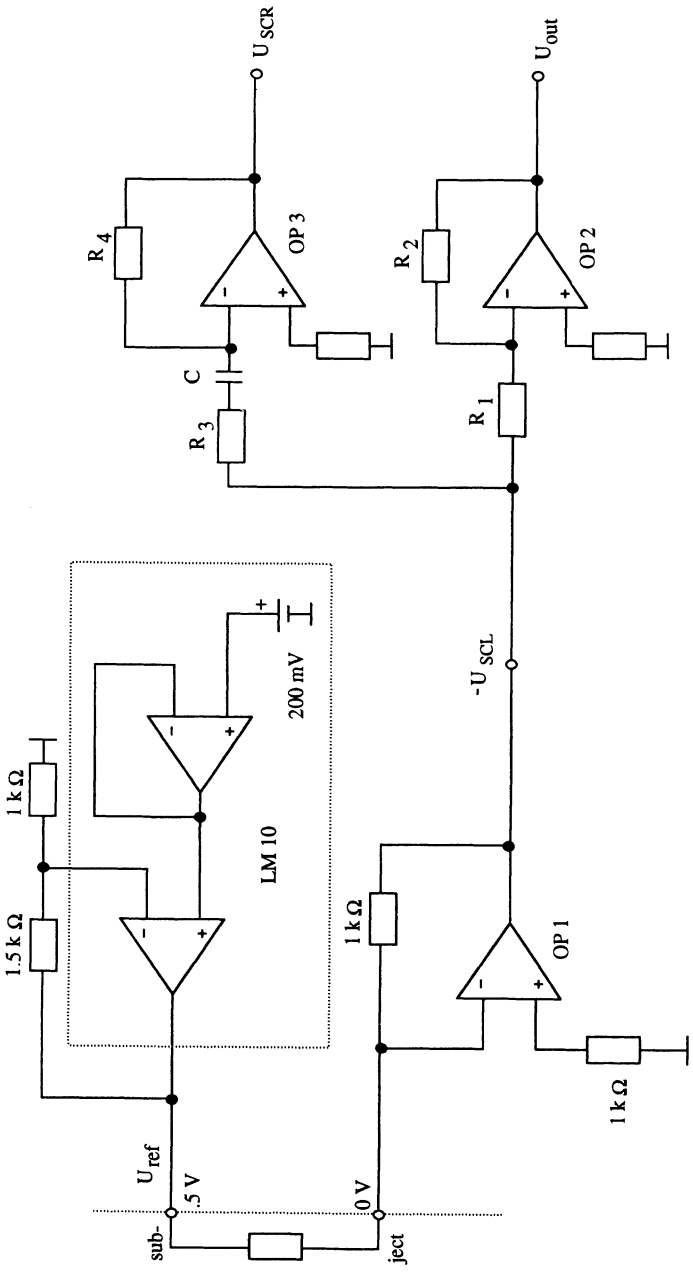

Figure 33. Coupler for DC recording of SCL and SCR, to be used with a high-quality amplifier system (designed by Jörn Grabke, University of Wuppertal, Germany).

$U_{out} = VU_{SCL}$, where $V = R_2/R_1$. If the biosignal amplifier's time constant τ is not large enough (e.g., 10 sec), OP 3 can be used together with the amplifier's DC mode output voltage U_{SCR}, which has the sensitivity $S = (R_4/R_3)$ (.5 V/μS) and the time constant $\tau = R_3C$. A maximum of security is guaranteed if the device is battery driven, and if the bioamplifier's input is galvanically separated.

Since constant voltage recording is predominantly used and strongly recommended (Venables & Christie, 1980; Fowles et al., 1981; Fowles, 1986a), there is no additional need to depict and describe circuitry for constant current recording here. However, some remarks on this method should be added. According to Edelberg et al. (1960) and Edelberg (1967), the current density should not exceed 10 μA/cm^2, since above this value SRL as well as SRRs markedly decrease, time parameters of SRRs are changed, and sweat gland damage may result in extreme cases (Sect. 2.6.2). Hence, Venables and Martin (1967a) as well as Edelberg (1972a) recommend the use of a 8 μA/cm^2 current density for constant current recording. Because the electrode's contact area forms a circle, the current I that has to be applied with a given electrode diameter d is: $I = 2\,\pi\,d^2$ (Venables & Martin, 1967a).

Since the standard electrodes described in Section 2.2.2.3 have a contact area of .6 cm^2, an appropriate current density between 4.8 and 6 μA is to be applied, leading to current density values between 8 and 10 μA/cm^2. Because those values must be kept constant, the limited contact area between skin and electrolyte must be maintained, which, however, is not necessary if constant voltage recording is used instead (Sect. 2.2.2.1). If results obtained with constant current recording are reported – despite being given in SR or SC units – the size of the electrode area should additionally be reported. This will enable the reader to calculate the specific resistance to use in making subsequent comparisons with other results (Sect. 2.3.3.1). However, Mitchell and Venables (1980) regard – contrary to the older hypothesis – the influence of the contact area as being negligible (Sect. 2.2.2.3).

2.2.3.3 Exosomatic recording with AC

The hitherto nearly exclusive use of DC methods in exosomatic EDA recording prevented the development of a widely accepted technique of AC measurement until now. Some recommendations exist concerning the appropriate frequencies. Montagu and Coles (1968) found that the lead electrodes they used showed the least polarization if 5 Hz was applied. Edelberg (1967) pointed to the impossibility of obtaining EDRs with sufficient reliability using high AC frequencies, because the polarization capacities of skin become too low. According to Brown (1972), the biological membrane loses its changeability for polarization between 5 and 10 kHz, and its capacitative properties become negligible above 20 kHz. Faber (1980), who continuously varied the AC frequency between 5 Hz and 10 kHz, showed the greatest differences in frequency locus with the lower frequencies when measuring EDA in three subjects during differ-

ent tasks. Thus, according to the present state of research, the use of AC frequencies between 5 and 100 Hz is recommended.

One possible application of AC recording in EDA measurement is to prevent polarization effects (Sect. 1.4.2.2). Instead of DC, an appropriate AC may be used as an exosomatic electricity source, together with a subsequent rectification of the signal. However, the phase angle must also be determined if capacitative properties of the skin are under investigation (Sect. 2.1.5). Devices for measuring the frequency locus are available for cardiac surgery, but the specific problems of filtering as well as separating phasic and tonic parts of EDA (Sect. 2.1.3 & 2.1.4) require the construction of specific instruments for EDA recording. As used by Boucsein et al. (1989), a device for AC recording of EDA is described here, which is a further development of a phase voltmeter constructed by Neijenhuisen and de Jongh (1981) for use in dermatology. Although constant voltage is generally preferred for DC recording of EDA (Sect. 2.2.3.2), measurements were taken with a constant current source, since Salter (1979) pointed out that this method is preferred for medical applications to prevent nonlinearities that may result from uncontrolled current densities when using a constant effective voltage source. Impedance and phase angle are obtained as analog output signals; a digital laboratory computer is used to transform those values into reactance and resistance as well as susceptance and conductance.

A block diagram of the phase voltmeter for AC measurement is given in Figure 34. A sine wave voltage from a 1 Hz to 1 kHz continuously adjustable oscillator is converted by a voltage-to-current converter into a constant current, adjustable between 0 and 10 μA peak value, which is delivered to the subject's skin. The terminal voltage from the skin site is preamplified and processed in two ways:

(1) To evaluate impedance, the voltage is submitted to a voltage amplifier, which is adjustable in sensitivity between 1 mV and 10 V in steps of decades, and the output signal is rectified and low-pass filtered with either .1 Hz or 1 Hz. After subtracting a manually adjusted offset, the signal is delivered to output 1 as well as to a digital display.

(2) For measuring phase angle, the preamplified signal is multiplied in a phase sensitive detector with the oscillator signal – which has been phase shifted with the possibility of adjusting the phase angle continuously – acting as a zero offset for the phase signal. The output signal of the phase sensitive detector is also rectified, low-pass filtered with the same frequency limit as the impedance signal, and delivered to output 2 as well as to a second digital display.

Outputs 1 and 2 are digitized using two channels of a 12 bit A/D converter with 16 Hz. Resolution for impedance is about 10 Ω/bit, for phase angle $.008^0$/bit. $X(f)$ as well as $R(f)$ are calculated from impedance $Z(f)$ and $\varphi(f)$ at the given AC frequency f, according to Equations (18a) and (18b), through the use of a digital computer. In addition, susceptance $B(f)$ and conductance $G(f)$ can be calculated at every sampled data point.

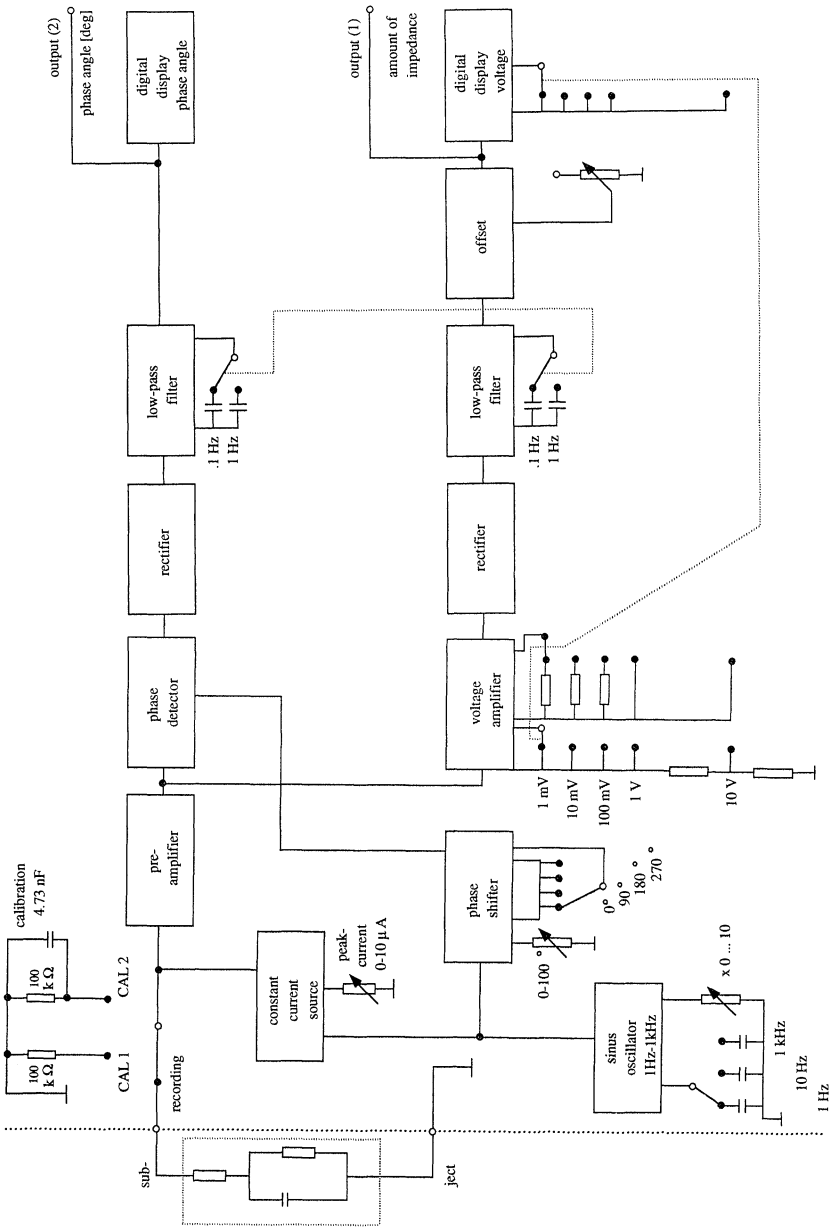

Figure 34. Block diagram of a phase voltmeter for simultaneous recording of impedance and phase angle. (See text for explanations.) From W. Boucsein, F. Schaefer, and W. Neijenhuisen (1989), Fig. 3. Copyright ©by the Society for Psychophysiological Research. Redrawn with permission of the publisher.

Calibration is enabled in two positions. CAL 1 for impedance uses a 100 kΩ resistor as a substitute. CAL 2 for the phase angle adds an additional 4.73 nF capacitance in parallel. The input impedance is 10 MΩ; the signal-to-noise ratio for impedance as well as for the phase angle is better than 98 dB.

Another attempt to continuously measure reactance as well as the ohmic part of impedance was performed by Almasi and Schmitt (1974); using Lissajous figures for signal control (Fig. 11, Sect. 1.4.1.4). Those authors presumed that only three periods of the applied AC current would be sufficient for determining EDA with a single frequency, which enabled them to obtain several measuring points even with low frequencies within relatively short periods (e.g., with the use of f = 1 Hz every 3 sec). Nevertheless, they stated that an experienced experimenter would need approximately 1 min to determine the impedance locus from three discrete frequencies between 500 Hz and 1 kHz.

Salter (1979) reported the development of a continuous AC measurement technique based on a 16 bit microprocessor. In his concept, the sine wave AC frequency used for exosomatic EDA recording was created in the central processor and given, via a D/A converter, to the analog part of the measuring device. The advantage was that the whole calculating procedure could be performed on a digital basis, using the originally generated digital sine wave signal. This also made all manual changes during recording (e.g., frequency changes) superfluous because all adjustments could be performed by software. Unfortunately, Salter did not pursue this concept further, and details about it are difficult to obtain.

Lykken (1971) used pulsed DC, instead of the usual sine wave AC method, for EDA recording, but only with one subject (Sect. 2.5.3.2). The use of pulsed DC current may be regarded as equivalent to sine wave AC recording, since Faber (1980) found an intraindividual correlation of r = .93 between SCLs obtained by a 10 Hz sinusoidal recording and a pulsed DC recording with a 10 msec pulse and an interval of 250 msec duration. Thus, in AC measurement of exosomatic EDA, further development and validation studies need to be performed.

2.2.4 Methods of storage and evaluation of the electrodermal signal

2.2.4.1 Paper recording and evaluation by hand

Several laboratories still use the conventional method of recording EDA on polygraph paper and evaluating the recording by hand. Since the changes in the EDA signal which form a single EDR are relatively small as compared to the whole bandwidth of the signal (Sect. 2.1.3), polygraph recording is frequently performed as a continuous registration of both the EDL and EDR with higher resolution (Sect. 2.1.3). This can be done by using either two polygraph channels or by superimposing impulses on the EDR signal, the distance of the impulses indicating the current EDL, thus requiring only one recording channel.

The extremely wide range of the EDA signal is likely to cause problems with recording and evaluation. If the normal polygraph recording width of 40–50 mm is used, and the AC-coupled EDR signal is recorded with a high amplifier gain, unexpectedly large EDRs will exceed the boundaries of the registration channel, and data will be lost (Fig. 43, Sect. 2.3.4.1). By contrast, if amplification is reduced, small EDRs will not be identifiable. Therefore, for paper recording of EDA, compensation recorders which allow 20 or 25 cm bandwidths for each channel are recommended (Table 4, Sect. 2.3.1.2.3).

For the evaluation of parameters like EDR amp. or NS.EDR freq., a paper speed of 5–10 mm/sec will be sufficient. However, if electrodermal time parameters, like latencies, rise times, or recovery times, are to be obtained from the recording chart (Sect. 2.3.1.1 & 2.3.1.3), paper speeds below 10 mm/sec will lead to unreliable scoring. The curvilinear pen deflections of some polygraphs may also cause marked distortions of rise times and recovery times, especially in EDRs with high amplitudes. However, the attenuation of frequency caused by technical features of the galvanometer system plays practically no role in the recording of the relatively low-frequency EDA signal.

The experimenter has to note the paper speed as well as the amplifier gain and every change made during the recording on the registration paper. This includes the possible changes in range as well as calibration marks, which should be applied in phases of relative EDR inactivity. Those calibration marks, which can be obtained using the "addCAL" switches in Figure 31 (Sect. 2.2.3.2), will be helpful for the subsequent evaluation, and will add clarity even when the recording has been calibrated in absolute values.

As a convention for write-outs, Venables and Christie (1980) recommend taking over the neurophysiologist's tradition of recording "negative up." This makes sense especially in SPL recording, where an increase of negativity is consonant with increasing general arousal (Sect. 3.2.1.1), and the initial SPR component, which is also likely to be negative in most cases (Sect. 1.4.3.2), will also show upward deflection. Using exosomatic methods, "negativity" makes no direct sense. Hence, the convention is to have increasing skin conductance values and thus an SCR, which indicates an increase of arousal, recorded as upward deflection. In turn, its reciprocal, SRR, should also be recorded as going upward deflection because a decrease of skin resistance also indicates an increase of arousal. Thus, in tonic exosomatic recording, an increase of SCL as well as a decrease in SRL would deflect upward on the paper output.

Evaluation of the EDA signal requires marking of the appropriate time windows, which belong either to a single stimulus or which indicate the period to which the NS.EDR freq. refers (Sect. 2.3.2.2). Before the single EDR amplitudes are evaluated, an amplitude criterion should be defined (Sect. 2.3.1.2.3). Using a ruler and perhaps a magnifying glass, amplitudes are obtained in millimeters or even in fractions of millimeters, and may be either listed in millimeters or transformed into appropriate units (mV, μS, or kΩ).

To obtain EDR ris.t., EDR rec.t/2, or EDR rec.tc it will be helpful to mark the previous level from which the EDR deflected with a horizontal line, using pencil and ruler, and to draw in a vertical line at the maximum peak as well as another horizontal line at the height of the desired portion of amplitude (Fig. 36, Sect. 2.3.1.2.2). However, these pencil lines will prevent an independent additional evaluation which could be made for testing reliability. Additionally, curve-matching techniques using transparent templates may be applied (Sect. 2.3.1.3.2).

2.2.4.2 Off-line computer analysis

Because psychophysiological laboratories are normally equipped with microcomputers, the evaluation of EDA is often performed by computer analysis. Since the EDA signal requires careful inspection when being analyzed, it is normally preferable to store the signal during recording and to perform off-line computer evaluation later. A flow diagram for a computer analysis system is given by Venables and Christie (1980, Fig. 1.16), and programs are now available in BASIC (e.g., Spinks, Dow, & Chiu, 1983) in FORTRAN (e.g., Foerster, 1984; Thom, 1988) and in C.[47] Since the automatic treatment of artifacts (Sect. 2.2.3.3) is not fully performed by those programs, the complete EDA signal should be recorded in a form that allows its reconstruction for subsequent control. Parallel recording can be performed in several ways:

(1) A parallel hand copy (Sect. 2.2.4.1), which additionally requires a temporal synchronization of the polygraph with the recording computer.

(2) An analog recording of the frequency-modulated EDA signal on a magnetic tape recorder, which in turn allows a later inspection of more interesting parts of the recording on an oscilloscope or on a paper write out.

(3) A digital storage of the AD-converted original EDA signal on tape, floppy, or hard disk with a sampling rate of approximately 20 Hz.[48] Afterwards, following a DA conversion, the data may be inspected as outlined in (2).

If EDA analysis with high-resolution A/D converters is applied, a new problem appears: it is now possible to quantify very small changes that look like EDRs which could, however, never be seen in paper recording. If the conversion is made with 12 bits, this results in 4,096 steps (Sect. 2.1.4). If for the AC-coupled SCR, a bandwidth of 20 μS is chosen, the conversion will result in .01 μS being represented as two steps of digital information stored in the computer, which can be reliably determined by the

[47] A program for the detection and parametrization of SCRs has been written by Peter Kohlisch in the present author's laboratory. It has been printed out as an appendix of this book, and will be made available as shareware.

[48] The conversion rate depends on the A/D converter used in the computer system. Thus Venables and Christie (1980) recommend 20 Hz, while Foerster (1984) used 16 Hz, which makes no difference in accuracy with respect to the phasic changes that occur in the electrodermal signal.

program. However, this resolution is far beyond the normally accepted amplitude criterion of .05μS (Sect. 2.3.1.2.3). Since researchers are not yet experienced with such highly exact EDR evaluation, they must use additional methods to separate EDRs from possible artifacts (e.g., from amplifier characteristics; see Sect. 2.1.4). As a possible solution, Foerster (1984) as well as Thom (1988) use a minimal ascent of .08 μS/sec as an additional criterion for the identification of an EDR, together with an amplitude criterion of .01 μS.

Because of the above-mentioned high resolution performed by A/D conversion, it is possible to dispense with a separate recording of the AC-coupled EDR in addition to the EDL. Given 12 bit A/D conversion and 50 μS bandwidth, which would cover all SCLs that appear under normal circumstances (Sect. 2.5.2.1.2), a change of .05 μS would result in four steps of digital information stored and could be reliably detected. However, for the sake of visual inspection, and since various EDA couplers provide AC-coupling of the signal as an ordinary method to record EDRs, uncoupling of EDR from EDL as described in Section 2.1.3 (e.g., by backing-off circuitry) is often maintained. A subsequent reconstruction of the EDL from the AC-coupled signal stored in the computer requires the following steps:

(1) The short time constant used during recording may be replaced by transforming it to a longer one (e.g., 10 sec to 100 sec), which eliminates the deformation of the signal that resulted from AC-coupling (Foerster, 1984; Thom, 1988).

(2) The appropriate EDL values are added to the retransformed EDR curve. If EDL is not recorded as a continuous curve, but as a sequence of pulses (Sect. 2.2.4.1), a higher conversion rate of, for example, 250 Hz must be applied to obtain the exact pulses. The EDL curve may then be restored from these pulses (Foerster, 1984).

Since those calculation procedures may result in some inaccuracies, Foerster (1984) prefers the AC-coupled EDR signal for the parametrization procedure. However, if transformations are to be performed later – which requires level information (Sect. 2.3.3.2)– a reconstruction of the EDL curve will be necessary.

For thorough control of artifacts (Sect. 2.2.3.3), an interactive computer evaluation of EDA has been proposed by Thom (1988). During the computer analysis of the EDR signal, as many "EDRs" as possible are detected which fulfill the above-mentioned amplitude and minimal ascent criteria, and those being artifact prone are shown on a high-resolution video display. In these critical cases, the experimenter's decision is required whether or not this is an EDR (following a recommendation by Venables and Christie, 1980), and artifact-correcting procedures are offered by the program. To avoid this time-consuming procedure, the computer program in the appendix of this book specifies various types of EDRs in its output, thus allowing the experimenter to identify and exclude artifact-prone reactions without interaction during the computer evaluation procedure.

Ambulatory monitoring of EDA with portable electronic storage devices during field studies requires specific features such as frequency modulation which is normally not available in portable magnetic tape recorders. A device for ambulatory measurement of SC based on a portable instrumentation cassette recorder has been described by Simpson and Turpin (1983). White and Charles (1983) described a telemetric system for SC recording to be used with a computer under both amulatory and laboratory conditions. Until recency, microcomputer hardcore mass storage of EDA has been seriously limited, since no data reduction at this point of recording can be performed, thus requiring storage of the full 16 or 20 Hz digitized signal. The development of low-volume mass storage in the range of megabits will – together with the availability of 12 bit instead of 8 bit portable A/D converters – help to improve the development of portable field recording devices for EDA measurement.

2.2.4.3 On-line computer analysis

An on-line computer analysis of all information the EDA signal contains requires a great amount of run time for data collection, analysis, and storage of this single signal. Therefore, in most applications, a subsequent off-line analysis, as described in the previous section, is preferred. Additionally, an interactive evaluation of artifacts may hardly be performed on-line since it requires the complete attention of an experimenter.

However, in some applications (e.g., biofeedback; Sect. 3.1.2.2), immediate signal evaluation must be done. If the information for feedback should go beyond the simple EDL, which shows only slow changes, an on-line evaluation of each EDR has to be performed. This requires the intermediate storage of a certain time window in a data buffer, thus leading to temporal delays in feedback. Also, a quick decision of whether or not a recorded change is due to an artifact is required.

Fried (1982) used time series analysis as proposed by Lathrop (1964) for an on-line analysis of the EDA. He was able to show a very good correlation between the parameters obtained and the EDR amp. An on-line procedure for the evaluation of skin impedance developed by Almasi and Schmitt (1974) was described in Section 2.2.3.3.

A further development of these methods of on-line computer analysis should take into account the considerations concerning the *Gestalt* of an EDR. This was discussed in Section 2.3.1.3.2 with respect to modelling the recovery limb. The minimal ascent criterion mentioned in the previous section is not sufficient to make the on-line computer evaluation of the EDR similar to eyeball detection, which is normally performed by the experimenter.

2.2.5 Sources of artifacts

Though the previous sections have already mentioned several sources of artifacts, as well as methods to avoid them, a thorough discussion of artifactual problems in EDA measurement is needed. Artifacts are changes in the recorded biosignal which do not

stem from the signal source in question. Instead, they may result from the measurement procedure (Sect. 2.2.5.1) or come from physiological reactions in systems other than the electrodermal one (Sect. 2.2.5.2).

2.2.5.1 Artifacts stemming from recording

As in every biosignal which is directly recorded as electrical activity from the body surface, a main source for artifacts is the AC frequency input, which is – depending on the country one is in – either 50 or 60 Hz. The frequency noise may be reduced to a great extent by means of shielding and/or grounding. Sometimes, twisting of electrode leads is recommended to prevent their differential antenna effects. Another way to reduce higher-frequency noise is low-pass filtering prior to amplification, which may be performed with time constants (e.g., between .25 and 2 sec; equivalent to frequency limits between .64 and .08 Hz, respectively). However, this may lead to a visible deformation of fast-recovering EDRs, as described in Section 2.1.4. Some amplifiers also provide "notch" filters that selectively block a narrow frequency band around 50 or 60 Hz, which frees electrodermal recording from most power line noise.

Since the danger of noise increases with amplification, constant voltage recordings, which require greater amplification, are more artifact prone than to constant current recordings (Sect. 2.1.1 & 2.6.2). Special problems with filtering may also arise in the AC measurement of EDA (Sect. 2.1.5).

In endosomatic EDA recording (Sect. 2.2.3.1), additional noise may result from insufficient grounding /or from increased transient resistance between skin and reference electrode (Sect. 2.2.1.2). This normally requires additional treatment of the electrode site and reattachment of the electrode. Similar artifacts may result when electrodes detach from the skin or when there are problems stemming from wiring contacts.

Additional sources of artifacts, which are also most likely in endosomatic EDA recording, are drifts caused by bias potentials (Sect. 2.2.2.2), which inseparably superimpose themselves on the SP signal. Drifts, which may result from polarization of electrodes, can be eliminated by changing polarity during recording (Sect. 2.2.2.2 & 2.2.3.2). Section 2.2.6.1 outlines how drifts that arise during long-term recordings can be prevented.

2.2.5.2 Physiologically produced artifacts

The most important physiological source of artifacts in EDA recording is movement. This not only includes skin movements beneath the electrodes, but also muscular activity at body sites that are not directly included in electrodermal recording.

Therefore, to provide an optimal artifact-free EDA measurement, gross body movements should be avoided during recording. It is best to tell the subject to sit or lie quietly, to relax, and to try to avoid movements, at least those of the limbs from which EDA

is recorded. Thus, artifacts which arise from pressure or stretching of the skin at the recording site and from changes in skin blood flow can be prevented (Sect. 2.4.2.1).

Edelberg (1967, p. 38) points to the following four main sources of movement artifacts:

(1) Disturbance of the electrolyte concentration near the solid-liquid electrode interface.

(2) Change in the intimacy of contact between electrode and skin.

(3) Pressure-induced local changes in SR (Ebbecke waves, Sect. 1.1.2).

(4) Movement of the appendage across an electromagnetic field.

Quantitative relationships between skin stretching at the volar side of the forearm and the elicited EDA artifact have been established by Burbank and Webster (1978). While skin tension reached a plateau during stretching, SP continued to increase with an increase of stretching. Skin impedance, which had been simultaneously measured with 10 Hz AC, showed no change with stretching. Ödman (1981), using a similar arrangement, investigated the immediate consequence of a reduction of skin stretching in one subject. He also found different courses of SP and SZ, with SP being more prone to stretching artifacts than SZ. Hence, endosomatic recordings are likely to be more influenced by stretching than exosomatic measurements.

Millington and Wilkinson (1983) find a reduction of both sweat rate and salt loss (both sodium and potassium) when pressure is applied to the skin, which may be due to an increased ductal reabsorption while sweat gland activity continues. It is likely that these processes also influence EDA (Sect. 1.3.4.2).

Apart from peripherally elicited artifacts, body movements may directly lead to EDRs via the premotor cortical and basal ganglia central nervous pathway (Sect. 1.3.4.1). Thus, an increase of the NS.EDR freq. is to be expected during work periods (Sect. 3.5.1.1) and also during speech activity. Tongue biting, which is used as a method to "beat the test" in lie detection (Sect. 3.5.1.2), elicits an EDR. Additionally, subjects may elicit voluntary EDRs by deep breathing and subsequent holding of their breath (e.g., Hygge & Hugdahl, 1985). Stern and Anschel (1968) investigated the action of different respiration patterns on the SRR amp. and on cardiovascular variables in 20 subjects. They found increasing electrodermal changes with more frequent and/or deep respiration. Therefore, the respiration curve should be recorded in addition to EDA recording patterns, to enable a later elimination of artifacts caused by irregular breathing patterns (Fig. 44, Sect. 2.3.4.1).

Whether EDRs as concomitants of motor actions and respiration have to be regarded as artifacts, or whether these physiological variables and electrodermal changes can be interpreted as covarying indicators of the psychological changes under investigation, depends mainly on the question being investigated. The latter may be the case, for example, in orienting and defensive reactions (Sect. 3.1.1.2), so it must be determined

whether or not to eliminate certain EDRs when emotional aspects are being investigated (Sect. 3.2.2.1). However, if possible, subjects should be instructed to avoid irregular respiration as well as speaking, in order to obtain artifact-free electrodermal recording.

Another source of physiological artifacts is the influence of temperature on EDA recording, discussed in Section 2.4.2.1. Interference from EKG may appear in SP recordings where electrodes are separated by a relatively large distance (Sect. 2.2.1.1).

2.2.6 Techniques of electrodermal recording in specific contexts

The EDA recording methods described in Sections 2.2.1–2.2.3.3 are standard techniques commonly used today. However, some relatively seldom-treated questions require specific EDA recording techniques. These techniques are described in the following sections.

2.2.6.1 Long-term runs

Some psychophysiological investigations – for example, studies of extended sensory isolation, circadian rhythms including sleep (Sect. 3.2.5), and night and shift work (Sect. 3.5.1.1.2) – require that EDA electrodes be left in place undisturbed for an extended period, up to several days. Edelberg (1967) points out the following problems in such applications:

(1) If a closed electrode/electrolyte system is used as described in Section 2.2.2, a gradual buildup of osmotic pressure results from the use of hypertonic electrode paste. The use of an unsealed system leads to a progressive drying out of the electrode paste.

(2) Inflammations may occur, especially along the edge of the electrode.

(3) Discomfort will result if electrodes are fixed by rubber bands or adhesive tape to the limbs, because of pressure or blood constriction.[49]

(4) Maceration of skin may occur after long exposure to an aqueous electrode paste.

(5) In long-term exosomatic DC recording, a progressive de-anodization of the cathodal electrode and a progressive depression of SR has been observed.

Edelberg (1967, p. 40) describes a recording method which allows EDA electrodes to be left in place for more than 10 days and avoids the problems mentioned above. He uses silver cloth electrodes forming a partially closed system, held in place with a nonadhesive elastic bandage. The cloth is soaked with a solution of 78% glycerol having a total concentration of .6% NaCl (which is .1 molar) or one of 90% polyethylene

[49] Additional fixing should be avoided at all if possible because of the artifacts that may result (Sect. 2.2.2.1 & 2.2.5.2).

glucol-400 with the same NaCl concentration. That electrolyte has been found to be in vapor equilibrium with ambient air at 65% relative humidity.

Using this method with seven subjects continuously for 12–14 days with replenishment of paste, Edelberg (1967) found problems with inflammation and minor skin eruptions along the edge of the electrode in only 2 of the 16 test sites. The EDR amp. values initially averaged 96%, and at the end of the long-term run 74% of control values. In another group of five subjects, in which replenishment of the paste was not possible, constant current methodology appeared to be superior to constant voltage recording (Sect. 2.6.2). When 9- to 11-day old sites that showed only slight activity in the constant voltage recording were changed over to a constant current system, the response rose from 15% of the cntrol site to 60%. Therefore, in cases where daily or 48-hour replenishment of the electrolyte is not possible, constant current should be used.

Zipp et al. (1980) investigated the effect of different NaCl concentrations in long-term AC measurements on the back of 12 subjects. An increase of NaCl content led to a faster stabilization of the system (after 30 min), as compared to a decrease of SZL which lasts hours when using lower NaCl concentrations. However, they observed more severe skin irritations with greater NaCl concentrations.

According to Venables and Christie (1973), skin macerations will be a more severe problem in endosomatic than exosomatic recording, since in the latter case the different effects of corneal hydration may neutralize each other; on one hand, SCL will increase because of increased humidity, while on the other hand, a decrease in SCL will result through mechanical pore closure (Sect. 1.4.2.3). Using 5% KCl paste with SP recordings in five subjects, Venables and Christie (1973, p. 58) found a marked reduction in SPR amp. during recordings of less than one hour duration compared to freshly prepared control sites. The low-basal skin potential level (Sect. 2.3.2.1), however, appeared relatively uninfluenced by hydration. As a consequence, Schneider and Fowles (1978) recommended using a less hydrating mixture made from Unibase and glycol for endosomatic recording, and Unibase without glycol for exosomatic measurement (Sect. 2.2.2.5). A mixture of Unibase and glycol may also be suitable for long-term runs.

Turpin et al. (1983) continuously recorded SC from 12 subjects for seven hours, evaluating three periods of rest and reaction-time task performance of 10 min duration each. Whith a week's distance, they compared two electrolytes in permuted order: A nonhydrating polyethylen-glycol paste and a hydrating methyl-cellulose paste. As a control for daytime effects, fresh electrodes were attached to two fingers of the same hand which were not used for long-term runs, and differences between these and the probe sites were formed. The hydrating paste yieled significant reductions in NS.SCR freq. and in SCR amp. as compared to the nonhydrating polyethylen-glycol.

Whether or not a problem of polarization will appear in exosomatic DC recording depends on the actual recording time. Thus, intermittent recording may be provided, even when electrodes are left in place during the entire course of an experiment. However, if continuous recording is requested (e.g., during sleep studies), either AC

recording should be used (Sect. 2.2.3.3), or the polarization of DC should be changed regularly during recording (Sect. 2.2.2.2).

During sleep, a marked increase in SRL is likely (Sect. 3.2.1.3). This may lead to additional problems if constant current is used because relatively high voltages will be necessary to maintain the intended current density, possibly leading to tissue damage (Sect. 2.6.2). Edelberg (1967, p. 42) proposed a solution using the endosomatic potential produced by the skin to measure its own resistance. If two SP electrodes, between which an initial potential U_0 is measured, are suddenly shunted with an external resistor R_S, the potential U_0 recorded with a high-impedance amplifier will drop to U_S. According to Equation (34a), the internal resistance R_0 of the SP generator in skin can be calculated:

$$R_0 = \frac{U_0 - U_S}{U_S} R_S \tag{34a}$$

However, considerable switching artifacts may arise if SP measurement is performed in conjunction with EEG recordings. Therefore Edelberg (1967) describes an alternative which couples SP with SY measurement. Because of its more general usability, the appropriate method is described separately in the next section.

It should be pointed out that procedures for correction of drifts which occur during long-term runs require the registration of control values using additional freshly prepared control sites (Sect. 2.3.4.3).

2.2.6.2 Recording simultaneously with different techniques

Some scientific questions (e.g., direct comparisons of different EDA recording techniques; Sect. 2.6.1–2.6.3) may require simultaneous recording of EDA using different methods. In these cases, the inputs of the amplifiers used must be separated galvanically to avoid cross currents (Sect. 2.1.4).

A method of simultaneous recording of skin potential and skin admittance was described by Edelberg (1967, p. 42). Between one of two SP electrodes and the amplifier, a low-impedance source of low-level and low-frequency AC (e.g., 10 mV at 20 Hz) is interposed. This voltage will divide itself between the subject and the amplifier in proportion to their impedances (Sect. 2.1.1). If an AC-coupled amplifier whose output is rectified is used with a time constant of .05 sec (Sect. 2.1.3), the SPL part of the signal will be blocked, and the SCL part can be calculated according to Equation (34b):

$$SCL = \frac{Y_S}{Y_T - Y_S} C_A \tag{34b}$$

where C_A is the amplifier input conductance, Y_T is the deflection produced by the AC source when the subject leads are short-circuited, and Y_S is the deflection with the subject in series.

According to the present author's experience, it is not easy to obtain artifact-free recordings using AC and DC measurement techniques simultaneously, since the AC

signal which is applied to the skin at one site superimposes the DC-recorded signal, even contralaterally. One way to overcome this problem is to switch between these recording methods continuously, which also allows the same site to be used for both methods. The disadvantage is that switching skin to an EDA coupler always results in an adaptation of gain process, following the filter characteristics of the amplifier system, which is time consuming. Parallel recordings of SP and exosomatic DC measurement contralaterally are possible without such problems.

To ensure that different measurement techniques do not influence each other, they should be used together with a substitute circuitry formed either by resistors alone (e.g., Boucsein & Hoffmann, 1979), or by resistors together with capacitors, according to the electrical model of skin as depicted in Figure 18 (Sect. 1.4.3.3). Also, possible influences stemming from other physiological variables recorded at the same time should be carefully controlled.

2.2.6.3 Measuring with dry electrodes or liquid electrolytes

Today's standard technique of electrodermal recording using electrodes together with humid electrolytes as described in Section 2.2.2 may cause the following problems, as pointed out by Muthny (1984):

(1) The moistening of skin by the electrolyte paste leads to an EDL drift over time, and additionally makes the system less sensitive to EDRs (Sect. 2.2.6.1).

(2) Polarization which appears at the boundaries of the electrode-skin system may be reduced to some extent when appropriate electrodes and electrolytes are used (Sect. 2.2.2.2). However, since they can never be totally eliminated, an oscillation of the electrode-skin system may appear and may last for hours.

(3) There may be uncontrolled and as yet unexplored interactions between electrodes, electrolytes, and skin, which may influence the measurement in an uncontrolled manner.

Fowles (1974) suggests the swelling of the stratum corneum being the main reason for the effects seen with skin moistening, as outlined under (1). On one hand, moinstening leads to an increase in SCL, while on the other hand ductal pores are mechanically closed, causing a decrease in EDR amp., since the ducts are no longer in use as electrical shunts between skin surface and sweat gland membranes (Sect. 1.4.2.3).

To prevent changes in EDA that may arise from moistening of the skin, dry electrodes have been used by some authors. However, Millington and Wilkinson (1983) point to the existence of mechanisms for ionic transport in dry corneum (Sect. 1.4.2.1). Additionally, sweat will act as an electrolyte between dry electrodes and the skin surface, which makes recording conditions even more uncontrollable (Muthny, 1984). Thomas and Korr (1957) artificially dried out the skin with heat to prevent this effect.

Dry electrodes have to be used in parallel recordings of EDA and skin vaporization. Thus in an appropriate investigation described in Section 2.4.1.1, Rutenfranz and Wenzel (1958) used dry electrodes made from V2A-steel nets, which appeared, however,to be polarizable. Hence, Zipp and Faber (1979) developed a dry electrode made from platinum/platinum-Mohr, which has a low polarization proneness, similar to that of Ag/AgCl electrodes. The electrode is provided in a ventilated chamber and attached with a constant pressure of .5 kPa to the skin. In measurements performed with one subject the authors found no marked differences in the amplitudes of the oscillations over time of the SYL between their dry electrode and a conventional, humid Ag/AgCl electrode. However, their comparisons of EDRs obtained with the different methods were not stringent, so the asserted advantage of their dry electrode method remains somewhat doubtful.

EDA recording measured with liquid electrolytes is used, for example, in the measurement of the influence of locally acting drugs or cosmetics on the peripheral mechanism of EDA (Sect. 3.5.2.1). Edelberg (1967, p. 12) described a method, used in a similar manner by Lykken and Rose (1959) for measuring EDA in rats, which he combined with a special masking technique. First, the recording site on the finger is covered with a disc of pressure-sensitive tape. Second, the entire finger, including the nail, is covered with a rubber paper cement, two thin coatings being more effective than a single thick one. Third, when the cement is almost dry, the covering of the recording site is removed. Two fingers prepared like this are immersed in separate baths, each of which is connected via an agar-KCl salt bridge (Sect. 2.2.2.5) to a chamber with an Ag/AgCl electrode in 1 molar KCl solution. The salt bridge's end dips into a perforated plastic tube forming a barrier, thus preventing contamination of the contact electrolyte and the KCl.

Another method to measure EDA using liquid electrolytes has been used on the forearm by the Yamamoto group (cf. Yamamoto et al., 1978, Fig. 5). They vertically aplied to the skin a plastic tube with two open ends, filled with an electrolyte made of 91.6% polyethylene glycol, .9% NaCl, and 7.5% water (by weight), in which an Ag/AgCl electrode was immersed. Because of the negative effects on EDA elicited by impeded blood circulation, the tube should not be affixed with the use of adhesive tape or rubber bands, but instead through the use of a histoacrylic glue (Sect. 2.2.2.1).

2.2.6.4 Other specific electrodes and site arrangements

This section outlines some infrequently employed recording techniques using more than two electrodes or unusual types of electrodes. In addition to the construction of Ag/AgCl chamber electrodes, Venables and Martin (1967a) give a description of how to construct sponge electrodes, which should be less prone to error potentials and to electrode drift (Sect. 2.2.2.2 & 2.2.5.1). Therefore, their use is appropriate for endosomatic rather than exosomatic EDA recording (Grings, 1974).

A two-element electrode has been discribed by Lykken (1959a), consisting of an inner circle surrounded by a concentric ring, both mad from zinc, and insulated against each other. Two of these electrodes were used, and the measurement current is brought to the skin via the outer rings, while the inner circles are used for recording. Because the current flow through the inner circles is rather low, no polarization occurs. Edelberg (1967), Montagu and Coles (1968), as well as Grings (1974) advocate that the principle of the two element electrode not be transferred to constant current measurement which however, might be performed by the use of comparator circuitry.

A similar principle of measurement is used by the four-line microelectrode, proposed by Campbell et al. (1977), which has a total width of .11 mm. The two outer electrode lines are connected with the voltage source, while recording is performed via the two inner electrode lines (cf. also Millington & Wilkinson, 1983, p. 129). These microelectrodes allow recording from a very small single site and may be used to investigate resistivity through the stratum corneum (Footnote 24, Sect. 1.4.2.2).

More than two electrodes have been used for AC recording of EDA with higher frequencies. Edelberg (1971) and Yamamoto et al. (1978) used a three-electrode technique, while Salter (1979) and Thiele (1981a) used four electrodes in AC recording. Thorough discussions of the technical implications for measurement are given by Salter (1979, p. 36ff.) and by Schwan (1963).

2.2.7 Summary of recording techniques

EDA recording is normally taken from palmar sites, with the use of an inactive reference electrode on the upper forearm in endosomatic recording (Fig. 27, Sect. 2.2.1.1). While the reference site has to be pretreated to reduce the electrical resistance between surface and inner tissue, no such treatment is necessary for the active recording sites, both in endosomatic and exosomatic measurement, although some authors recommend this procedure (Sect. 2.2.1.2).

Typically, Ag/AgCl chamber electrodes with .5–1 cm^2 area are used (Sect. 2.2.2.3). These are filled with an isotonic NaCl gel, made from a neutral medium like Unibase (Sect. 2.2.2.5), and fixed with the aid of double-sided adhesive collars (Sect. 2.2.2.1). After being used, electrodes are rinsed carefully with distilled water to avoid damage of the Ag/AgCl layer. When not being used, they may be short-circuited and stored in NaCl solution (Sect. 2.2.2.4).

There are specific features of the EDA signal to consider in order to obtain a reliable and artifact-free record (see Chapter 2.1). Readers should be aware of these problems, and should compare the features of their own equipment with those of the apparatus described in Section 2.2.3.1. While there is no standard for exosomatic recording using AC (Sect. 2.2.3.3), it is recommended that DC recording use a constant voltage of .5 V (Sect. 2.6.2) or a constant current not exceeding 10 $\mu A/cm^2$ (Sect. 2.2.3.2). These recording techniques are referred to as "standard methodology, " a term that will be

used when reporting results in Chapters 2.4 and 2.5, as well as in Part 3 of this book (see Footnote 63, introductory part of Chapter 2.4).

Recording methods, which may use either paper or electronic storage, must consider the wide range of the electrodermal signal (Sect. 2.2.4.1). The recording of EDRs requires a high resolution, and care must be taken not to lose data by exceeding the boundaries of the registration channel. Extensive use can be made of computerized systems for recording and for data evaluation (Sect. 2.2.4.2 & 2.2.4.3).

In the typical exosomatic DC recording of EDA, interference is less problematic than for other biosignals (Sect. 2.2.5.1). Physiologically produced artifacts have to be considered, especially when movement and respiration artifacts play a major role (Sect. 2.2.5.2). Artifact-correcting procedures in computer evaluation of EDA are time consuming (Sect. 2.2.4.2). Thus, artifact-free recordings should be attempted.

2.3 Scoring

As with every biosignal, parameters have to be extracted from electrodermal recordings prior to statistical evaluation. As compared to higher-frequency signals like EMG, EEG, or even EKG, EDA is a relatively slow-changing biosignal. The evaluation of phasic changes mainly focuses on irregularly appearing single events rather than on patterns that may be characterized by changes in frequency and/or amplitude. Hence, common procedures like power spectrum or Fourier analyses cannot be used in obtaining parameters from electrodermal recordings.

The first stage of parametrization is the extraction of phasic and tonic values from the recorded signal. Because the evaluation of one kind of tonic parameter, the NS.EDR freq., presumes knowledge of how to obtain phasic parameters, phasic parameters are treated first (Sect. 2.3.1) and the tonic parameters later (Sect. 2.3.2). Two subsequent sections in the present chapter outline further possibilities of data treatment prior to statistical evaluation (Sect. 2.3.3 & Sect. 2.3.4).

2.3.1 Parameters of phasic electrodermal activity

The phasic portion of EDA is always called a reaction, although there is not always a distinct relationship between a stimulus and an EDR (Sect. 1.1.1). However, most phasic changes of EDA show a rather characteristic course or *Gestalt* (Sect. 2.3.1.3.2), which enables the experimenter to separate them from artifacts with sufficient reliability. Unfortunately, algorithms for the detection of an EDR *Gestalt* are not yet available for computer analysis (Sect. 2.2.4.3), and therefore it has to be obtained with the visual aid of an experimenter (Sect. 2.2.4.2).

2.3.1.1 Latency times and windows

Electrodermal reactions have relatively long latencies compared to other biosignals such as event-related potentials (ERPs) or changes in heart rate (HR). Latencies of exosomatic EDRs are normally between 1 and 2 sec, but may be prolonged up to 5 sec in cases of skin cooling (Edelberg, 1967).

The latency of the first SPR component is about 300 msec shorter than the SCR lat. (Venables & Christie, 1980). There are many arguments about the appropriate time window for an EDR following a distinct stimulus and hence for the possible range of latencies. Edelberg (1972a) gives a window between 1.2 and 4 sec, regarding 1.8 sec as a characteristic value for comfortable ambient temperature. However, Venables and Christie (1980) propose that an EDR lat. exceeding 4 sec is too high, and state that a window between 1 and 3 sec would be rather conservative but suitable in most cases (Fig. 42, Sect. 2.3.2.2).

Levinson and Edelberg (1985, Table 4) report a synopsis of all EDR latencies published in the journal *Psychophysiology* between 1977 and 1982. According to this synopsis, windows between 1 and 4 sec and between 1 and 5 sec are the most frequently used. These authors recommend the calculation of a specific window for each experiment from the range of the EDR lat. of all subjects to the first stimulus applied. They report windows between 1.0 and 2.4 sec from their own laboratory.

Stern and Walrath (1977) propose an individual standardization of the time window, using the individual modal value, and limiting the window to $\pm$ .5 sec of this value. Venables and Christie (1980) also recommend this kind of standardization, however, only in cases of atypical EDA, for example, those obtained from older subjects (Sect. 2.4.3.1) or from patients with psychopathology (Chapter 3.4).

In some subjects, it is difficult to obtain stimulus-dependent EDRs and hence EDR latencies at all, since they show a high frequency of nonspecific phasic changes in EDA (Sect. 2.3.2.2). Also, the lack of exact criteria for appropriate windows or their inadequate application may have led to a misinterpretation of nonspecific EDRs as specific EDRs in many cases (Levinson & Edelberg, 1985).

Some authors (e.g., Thom, 1988) use the point of the EDR maximum to calculate the EDR lat. instead of the EDR onset. In this case, the rise time (Sect. 2.3.1.3.1) must be subtracted to obtain values comparable to ordinary latencies.

Another problem that arises in the evaluation of EDR lat. is the often indistinct onset of an EDR.In this case, finding the first derivative may be helpful (Sect. 2.3.1.3.1), which, however, presupposes electronically recorded data.If paper recording is used, latencies can only be obtained in a reliable manner if paper speed is at minimum of 10 mm/sec.

Additionally, skin temperature should be recorded if EDR latencies from different investigations are to be compared (Sect. 2.4.2.1 & 2.5.2.3), since 20-50% of the EDR lat. is dependent on the acetylcholine transport in the periphery, which varies with temperature (Sect. 1.3.2.1).

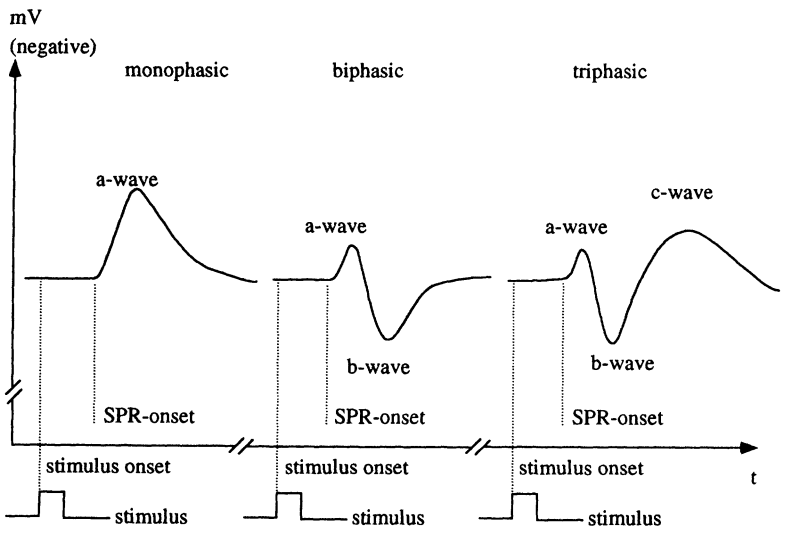

Figure 35. Different types of SPRs: mono- , bi- , and triphasic forms. Higher values mean greater negativity of the active with respect to the passive site (Sect. 2.2.1.1).

2.3.1.2 Amplitudes

The amplitude is the most frequently used parameter to describe a single EDR. Evaluation of amplitudes must use certain criteria for a minimum value (Sect. 2.3.1.2.3) as well as for the correct treatment of superimposed EDRs (Fig. 37, Sect. 2.3.1.2.2).

A further complication arises from the inconsistent use of the term "EDR magnitude." Unfortunately, some authors, like Venables and Christie (1980), recommend the use of the term *magnitude* instead of *amplitude*. However, for the sake of clarity, "magnitude" should be restricted to a kind of missing data treatment, described in Section 2.3.4.2, which takes into account zero reaction to stimuli and includes these zero reactions in averaging, thus leading to a *mean magnitude* which is different from the mean of all observed EDR amplitudes.

2.3.1.2.1 Amplitudes of endosomatic reactions. While the exosomatic EDR is always monophasic, as pointed out in the next section, endosomatic EDRs may be mono-, bi-, or even triphasic, for reasons which were discussed in Section 1.4.2.3. Figure 35 shows examples of different kinds of SPRs. As proposed by Forbes (1964), the first negative deflection is labelled a-wave, the positive deflection b-wave, and the second

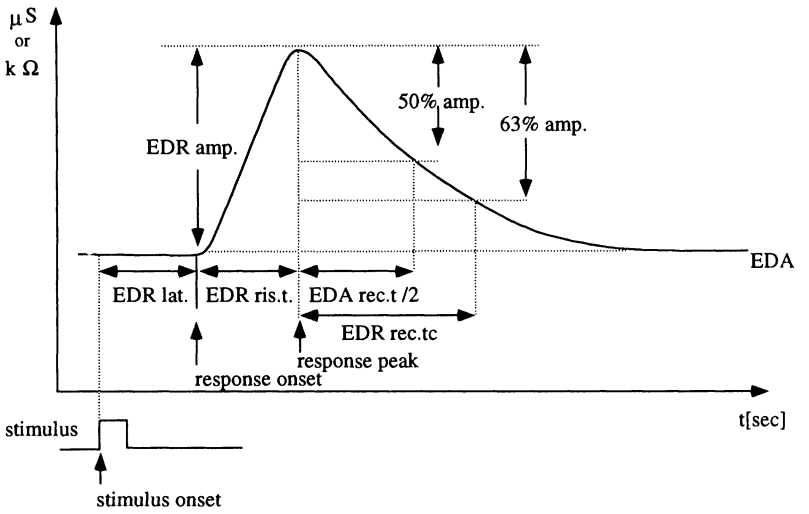

Figure 36. An ideal type-1 DC recorded exosomatic EDR and the parameters to be obtained from it. (See text for explanations.)

negative deflection c-wave or γ-wave.[50] Monophasic SPRs may also be positive instead of negative. Because the observed SPR is always composed of two underlying processes, the evaluation of SPR amplitudes remains problematic (Venables & Christie, 1980; for a further discussion see Edelberg, 1967, p. 48). In biphasic SPRs, some authors prefer an amplitude evaluation from the negative to the positive peak instead of having a negative and a positive deflection from the prestimulus level; however, there is not enough evidence that this is an appropriate evaluation (Venables & Christie, 1973).

SPR amp. is recorded in mV. Results from several experiments that made use of SPR amp. evaluations are given in Section 2.5.1.1. The advantage that endosomatic recording incurs from being free from current applied to the system is outweighed by the ambiguity of the EDA amplitude evaluation.

2.3.1.2.2 Amplitudes of DC-recorded electrodermal reactions. Exosomatic EDRs always show a monophasic course, as shown in Figure 36. A given stimulus will – after a certain latency (Sect. 2.3.1.1) – lead to a deflection which is an increase in SC or a decrease in SR, depending on the recording technique (Sect. 2.1.1 & 2.1.2). The

[50]The convention for write-out is, as in the neurophysiological tradition of EEG recording, "negative up" (Venables & Christie, 1980).

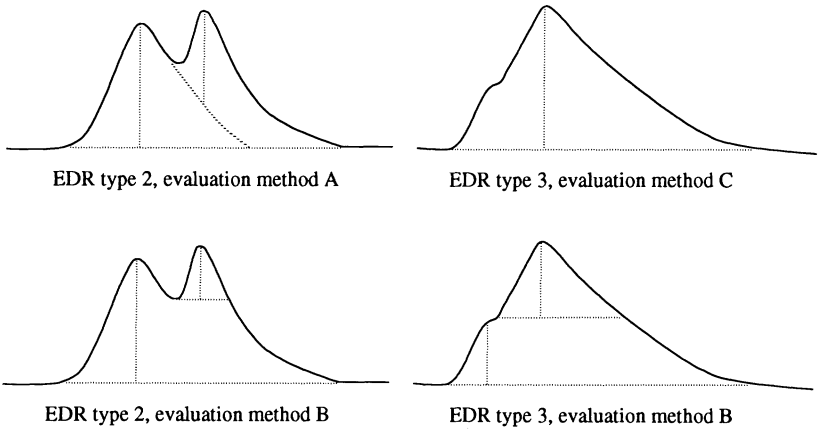

Figure 37. Examples for overlapping exosomatic EDRs of type 2 and 3, and methods of evaluating the appropriate amplitudes. (See text for explanations.)

SCR amp. is measured in μS, while the SRR amp. is measured in kΩ (Sect. 1.4.1.1). The form of an exosomatic EDR has a relatively steep onset and a flat recovery; that is, EDR ris.t. is shorter than recovery time (Sect. 2.3.1.3.2).

Evaluation of single EDR amplitudes may become problematic when overlapping EDRs appear, as in states of high arousal (Sect. 3.2.1.1), with subjects high in electrodermal lability (Sect. 3.3.2.2), or during conditioning (Sect. 3.1.3.1). Figure 37 shows two examples of superimposed EDRs. They are labelled – in opposition to the ideal "type 1" in Figure 36 – as "type 2" and "type 3." In cases of overlapping reactions, where there is evidence for an incomplete response, evaluation method "A" in Figure 36 can extrapolate the first EDR. The amplitude of the second EDR is then obtained by measuring the length of the vertical from its peak to the extrapolated recovery line of the first reaction (Hagfors, 1964).

However, Edelberg (1967, p. 46), using electrical stimulations of the distal stump of the cat's plantar nerve, showed that evaluation method "B" in Figure 37 will lead to sufficiently exact results, in most cases. Because method B is easier to perform, it is regarded as standard.

While in type-2 recordings there are always two distinct EDRs to be detected, this is not the case in type-3 overlaps, especially if there is no return subsequent to the first

peak of the curve, but instead another ascent. To avoid bias, one has to fix appropriate criteria before starting the evaluation,that is, whether such an EDR course is to be regarded as a single EDR (evaluation method "C" in Fig. 37) or as two superimposed EDRs (evaluation method "B"), as recommended by Edelberg (1967, Fig. 1.16 d). The computer program described in the appendix provides an even more detailed typology of EDRs, taking into consideration additionally the course of EDL immediately before reaction onset. For statistical computations, it is possible to evaluate the three types of EDRs separately (Thom, 1988).

Foerster (1985), in his computer evaluation program (Sect. 2.2.4.2), uses a criterion of distance to the secant line to distinguish between a hump that is formed by superimposed EDRs and another one that may be due to artificial deflections. A formalized hump detection procedure is used by the computer program in the appendix to indentify superimposed EDRs.

2.3.1.2.3 Choice of amplitude criteria. Before starting with the evaluation of the electrodermal signal, a criterion should be fixed concerning the minimum deflection in μS or kΩ, which has to appear to register an EDR. That criterion is largely dependent on the resolution of the recording, which in turn depends on the signal's expected range and its amplification. Table 4 gives examples of the dependency of the amplification on the amplitude criterion. Given that 1 mm is the minimum deflection reliably detectable by hand evaluation and the normal polygraph recording channel width is 40 mm, a range for registration of SC between 10 and 30 μS will lead to an amplitude criterion not smaller than .5μS. If, however, electrodermal reactivity is so low that the range may be restricted to values between 10 and 20 μS, without danger of SCRs exceeding this range (Sect. 2.2.4.1), an increase of amplification can occur and, in turn, the amplitude criterion can be lowered to .25 μS. If a compensation recorder with 20 cm channel width is in use instead of the above mentioned polygraph, the resolution will become 5 times as high, which with a 20 μS range will lead to a minimum amplitude criterion of .1 μS, and with a 10 μS range to a .05 μS criterion. An example for SR recording is given in the lower part of Table 4.

As pointed out in Section 2.2.4.2, A/D conversion with subsequent computer parametrization of the electrodermal signal may allow much higher resolution than paper recording and hand evaluation. Therefore, to make those different evaluations comparable, the same amplitude criterion has to be chosen for both. This may lead to the disregarding of very small EDRs, which today can be evaluated in computer parametrization in place of hand scoring.

In some scientific contexts, as in research on schizophrenia, electrodermal nonreactivity has to be defined, which requires a fixed amplitude criterion (Sect. 3.4.2.2). As can be inferred from Table 4, values recommended in these contexts (e.g., .05 μS, Gruzelier & Venables, 1972; or .4 kΩ, Zahn, 1976) require a relatively high amplification, and hence cannot be reached by every electrodermal recording device.

Table 4. Dependency of the minimal amplitude criterion on amplification and channel width in paper recording.

Method	Expected values		Resolution	
	Upper and lower limit	Range	Corresponding value to 1mm deflection, given a channel width of:	
			40 mm	200 mm
SC	10 – 30 μS	20 μS	.5 μS	.1 μS
	10 – 20 μS	10 μS	.25 μS	.05 μS
SR	100 – 500 kΩ	400 kΩ	10 kΩ	2 kΩ
	20 – 100 kΩ	80 kΩ	2 kΩ	.4 kΩ

Moreover, the question of whether or not the use of a specific amplitude criterion makes sense, cannot be estimated without knowledge and inclusion of the signal-to-noise ratio of the recording system.[51] As pointed out in Section 2.1.4, the frequently used decoupling of EDR with higher amplification may lead to effective signal-to-noise ratios below 20 dB. Given a total range of 20 μS, as in line 1 of Table 4, and a signal-to-noise ratio of 20 dB, it is possible that changes produced by noise reach 2 μS. This can be calculated using a transformation of the equation provided in Footnote 30 (Sect. 2.1.5). Thus, it would not make sense in this case to lower the amplitude criterion below this value.

Because of the possible level dependency of the EDR (Sect. 2.5.4.2), Edelberg (1972a) proposed a relative amplitude criterion of .1% of the initial SRL to count SRRs. He also recommended resetting the amplitude criterion again, if changes in SRL exceed 10%. However, it has become more common to prefer an amplitude criterion which remains constant throughout the whole range of measurement. In this case, Edelberg (1972a) recommended .1 μS for SCRs. Venables and Christie (1980) choose the above-mentioned criteria of .05 μS for SCRs and .4 kΩ for SRRs.

While the SCR is independent from the contact area between skin and electrode, this area plays an important role in SRR because of its influence on current density (Sect. 2.2.3.2). Several authors give their SR results related to the electrode area as specific resistances in k$\Omega \times$ cm^2 (Sect. 2.3.3.1).

[51] A declaration of the signal-to-noise ratio, which is obvious in audio devices, is often lacking in descriptions of polygraph amplifiers.

Edelberg (1972a) points to a method of EDR evaluation which can be used if a signal cannot be unambiguously defined, using the difference between a prestimulus and a poststimulus EDL. Those EDLs are obtained either as mean or as minimum-to-maximum values of a period, for example, 15 sec before and after stimulus onset.

2.3.1.2.4 Amplitudes of AC-recorded electrodermal reactions. Since the output signal of AC-recorded electrodermal activity is rectified (Sect. 2.1.5), the appropriate EDRs are evaluated in the same manner as shown for DC-recorded EDRs in Figure 36 (Sect. 2.3.1.2.2). Due to the recording technique and/or subsequent transformations used, evaluations are performed in kΩ in case of a SZR or in μS in case of a SYR. If the phase angle φ is recorded continuously in addition to impedance or admittance, it is possible to analyze the phase angle's course in time in a similar manner to the courses of R and X or B and G, respectively, which are computed according to the appropriate equations given in Section 1.4.1.3.

An example for evaluating an AC-recorded EDR is given by Boucsein et al. (1989, Fig. 4) using the measurement device described in Figure 34 (Sect. 2.2.3.3). The authors found very similar, though mirror-imaged, courses of Z and φ. This similarity remained after a transformation into values of R and X. However, if a transformation into values of G and B was performed, EDR-like changes of the signal could only be observed in conductance but not in susceptance, even with high resolution. This provides evidence for main changes during an EDR taking part in the parallel resistance R_2 (Fig. 18, Sect. 1.4.3.3), as can be inferred from Figure 20. If a continuous recording of EDA using more than one AC frequency is attempted, as described in Section 2.1.5, the depiction of the course of an EDR could be plotted in the form of loci (Fig. 10, Sect. 1.4.1.3), and extended to incorporate the time dimension, as used in three-dimensional EEG recordings.

2.3.1.3 Reaction shape

The main electrodermal parameters which describe the shape of an EDR were depicted in Figure 36 (Sect. 2.3.1.2.2). The following sections describe how to obtain those as well as additional parameters of ascent (Sect. 2.3.1.3.1) and descent (Sect. 2.3.1.3.2) of an EDR.

2.3.1.3.1 Parameters of ascent. To obtain reliable values for EDR rise times, it is necessary to define its onset as well as its peak unambiguously. This may become problematic if an EDR is less ideally formed than that depicted in Figure 36.[52] Therefore, Edelberg (1967) recommended the use of a first derivate, which passes the zero

[52]To avoid this problem, rise time is defined in electrodermal measurement techniques as the time the curve passes between the 10% and the 90% mark for the amplitude. In electrodermal research, however, EDR ris.t. is defined as the time span between response onset and response peak (e.g., Venables & Christie, 1980).

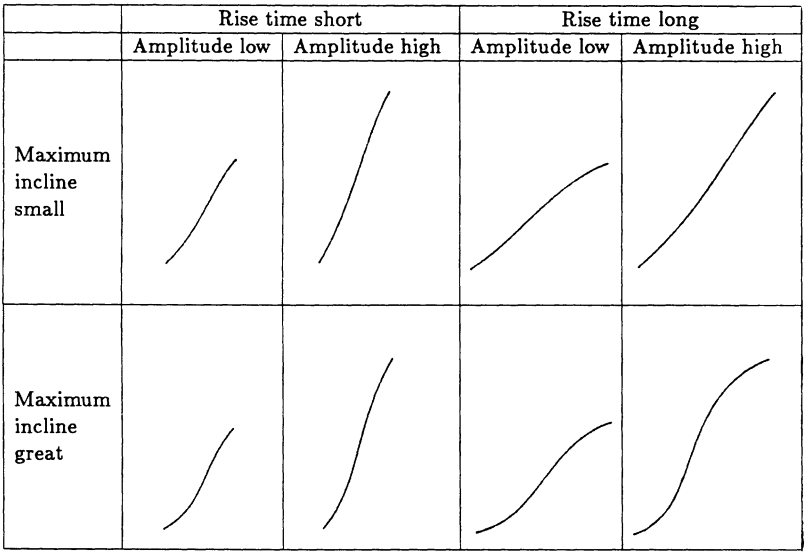

Figure 38. Examples of different forms of an EDR ascent, which have different rise times and maxima of their incline depending on the EDR amplitude.

line if the curve shows maxima or minima. However, this method only provides unambiguous values for the peak of an EDR, since its onset is normally not a minimum in the EDA curve, but a deflection point from a more or less steady line. Hence, Foerster (1984), in his computer program for EDA analysis (Sect. 2.2.4.2), detects the turning point of the ascent as a first step, going back to the point where the incline goes under 1% of its maximum value, and defining this point as EDR onset.[53] Instead, the computer program described in the appendix of this book uses the gradient of incline to decide when to start an EDR evaluation.

When doing hand evaluation of paper-recorded EDA, response onset and peak are obtained with the aid of graphic methods (Sect. 2.2.4.1). As with the evaluation of latency times, the accuracy of EDR ris.t. is dependent on recording paper speed (Sect. 2.3.1.1). The point of response onset, which separates EDR lat. from EDR ris.t., is much less reliably obtained than the point of an EDR peak, especially if the EDR begins to ascend as a flat curve.

An additional parameter of ascent, which can, however, be obtained with sufficient reliability only by computer evaluation, is the EDR's maximum incline (e.g., Foerster,

[53]Thom (1988) uses a criterion of 10% instead, because the application of the 1% criterion is difficult if numerous electrodermal fluctuations appear.

1984; Thom, 1988). Figure 38 shows hypothetical forms of an EDR ascent, combining characteristics of amplitude, rise time, and maximum incline. The maximum incline can be used to describe the *Gestalt* of an EDR ascent, which is more S-shaped the greater its maximum incline is. Figure 38 also shows that – given the same amplitude – a negative correlation between rise time and maximum incline of an EDR ascent is to be expected.

2.3.1.3.2 Recovery parameters. In most cases it will not be possible to determine the exact point in time when an EDR is terminated. As indicated in Figure 36 (Sect. 2.3.1.2.2), the decline ends more or less asymptotically. Additionally, due to EDL shifts that may occur during an EDR, the end point may not reach the starting level of the EDR in cases of DC coupling or when time constants in AC-coupled systems are long (Sect. 2.1.3).

Nevertheless, to obtain parameters for EDR recovery, Darrow (1937b) take the half-life concept applied to radioactive matter. Half-life indicates the time span after which one half of the available amount of a radioactive substances decays. Transferring this concept to an EDR, its amplitude is regarded as the "total amount," and its half-life, or EDR rec.t/2, is the time from response peak to the point where the curve falls below one-half of the EDR amp. (Fig. 36).

In the following equation, the EDR amplitude's height is indicated by A,[54] and the velocity of an EDR recovery can be calculated – according to the appropriate function in a radioactive matter – as follows:

$$\frac{dA}{dt} = -\tau A \tag{35a}$$

where τ is the time constant, and the minus sign indicates that the EDR is recovering. If a quantity is proportional to its own change, as shown by A in Equation (35a), this always indicates an exponential course of the quantity with respect to time (Sect. 1.4.1.2). Such a time course can be described in terms of electrophysiology as a capacitor's discharge within an RC circuit (Equation (10a)):

$$A = A_0 e^{-\frac{t}{\tau}} \tag{35b}$$

A_0 is the initial value of A (i.e., the EDA at the response peak) which can be shown when substituting $t = 0$, because $e_0 = 1$.

To obtain the time constant τ in seconds, t must be substituted by τ in Equation (35b). This leads to an exponent of $= -1$, and because $e^{-1} = 1/e$, it follows:

$$A = \frac{A_0}{e} = \frac{A_0}{2.7182...} = .3678...A_0 \tag{35c}$$

[54] A is measured in mV for SP, in μS for SC and SY, and in kΩ for SR and SZ.

The time, which corresponds to the time constant τ is reached, when the EDR has descended to about .37 of its maximum value A_0 (i.e., the EDR has recovered by about 63%). Thus, calculating the EDR rec.tc leads directly to the time constant for EDR recovery (Sect. 1.4.1.2).

The half recovery time, EDR rec.t/2, in the following indicated by λ, is calculated using Equation (35b), in which t is substituted by λ and A by $A_0/2$ (i.e., half of the maximum amplitude A_0):

$$\frac{A_0}{2} = A_0 e^{-\frac{\lambda}{\tau}} \tag{35d}$$

Dividing both sides of Equation (35d) by A_0 and forming the reciprocal values leads to:

$$e^{\frac{\lambda}{\tau}} = 2 \tag{36}$$

At both sides of Equation (36), natural logarithms are formed:

$$\frac{\lambda}{\tau} = \ln 2 \tag{37}$$

Multiplying both sides by τ gives:

$$\lambda = \ln 2\tau = (.6931....\tau) \tag{38}$$

Venables and Christie (1973, p. 96) give .7 as an approximate value in their equation for calculating EDR rec.t/2 out of the time constant τ, corresponding to Equation (38): $\lambda = .7\tau$.

However, the form of the decline of an EDR curve, as depicted in Figure 36 (Sect. 2.3.1.2.2), cannot sufficiently be approximated through a simple e-function as in Equation (35b). This has been shown by Stephens (1963) who performed a comparison of empirically determined SRR decline curves with theoretical courses. He also came to the conclusion that especially with high initial values a simple exponential curve does not correspond to but only approximates the SRR decline curve. Instead, a superposition of several e-functions with different time constants will be necessary, as exemplified in Figure 39.[55]

The determination of the time constants for the three e-functions used in Figure 39 is made empirically from several SC and SP curves of an experimental subject. The displayed curve shows the response to a square pulse of 1.4 sec duration (Sect. 1.4.1.4), whereby a combination of two e-functions with time constants of .2 and 6.0 sec was used for the decline, and an e-function with τ = .1 sec was used for the incline. The simulated SCR curve in Figure 39 gives a SCR ris.t. of 1.8 sec and a SCR rec.t/2 of

[55]The curve was kindly made available by F. Foerster, University of Freiburg, Germany.

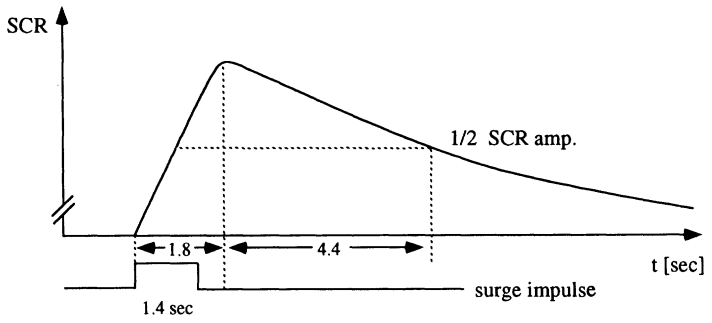

Figure 39. Simulated SCR curve which results from the summation of three *e*-functions. (See text for explanations.)

4.4 sec, which is a good approximation of a typical EDR course such as that shown in Figure 36.[56]

The incomplete approximation of EDR decline curves through a single *e*-function with a negative exponent questions the value of the approximation of the inexactly determined recovery time through the parameter EDR rec.tc. Furthermore, the relation between the so-calculated recovery time and the EDR rec.t/2 remains questionable, as the necessary preconditions are only approximative. This problem is also present in Edelberg's (1970) proposed use of graphic matching methods, so-called curve matching, as an alternative to the EDR rec.tc calculation. Through the insertion of resistors of differing resistances in a simple RC circuit, a group of comparison curves can be produced, with which the steepest point on the measured EDR decline curve should be compared. The time constant of the RC circuit wich most closely approximates the EDR decline curve should then serve as an estimation for the EDR rec.tc. The comparison may be performed using transparent templates. This method can also produce a decline form parameter in those cases where the measured EDA curve only attains 20–30% recovery. Edelberg (1971) gives a range from 1–15 sec, with typical values between 4 and 6 sec, for the time constants of the EDR decline curve (Sect. 2.5.2.4).

[56]Refined mathematical modelling of EDR curves has been performed by Hunt (1977), who developed an equation based on overlapping Gaussian distributions to fit the course of SRRs, and by Schneider (1987). Schneider fitted a three-compartment model to the recorded SC curve (personal communication) which includes the physical properties of the duct filling, the active membrane response in the duct walls, and the corneal hydration (Sect. 1.4.2). Schneider could show that a typical SCR can be modelled by assuming a roughly triangular input signal and choosing as an impulse response a sum of two exponentials with time constants of approximately 2 and 20 sec, respectively.

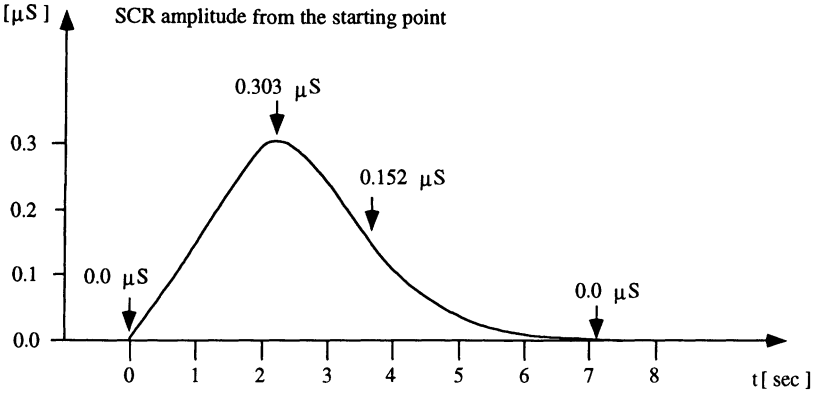

Figure 40. Simulated SCR curve, as obtained by the interpolation between four empirically determined points of an SCR with the help of cubical splines. (See text for explanations.)

It is not, however, a precondition for the calculation of recovery parameters of EDA, as for the EDR rec.t/2, that the decline form a base for a recovery process which can be described with one or more *e*-functions. Such characteristic decline values can be built for steadily falling curves as long as the curve declines. For example, a good approximation of the SCR curve can be obtained by an interpolation from the point of EDR onset, the maximum and half-way decline points, and the extrapolated total decline point, with the use of cubical splines (Ahlberg, Nilson, & Walsh, 1967). The problem here is the definition of the starting point, because the interpolation method cannot simulate naturally occurring steep slopes. Using cubical spline interpolation would lead to a subzero deflection following the start of the response, which was calculated, but not depicted, for the curve displayed in Figure 40.

The simulated SCR curve in Figure 40 is based upon the empirical values of an experimental subject, determined with the help of computer analysis (Sect. 2.2.4.2; see also Thom, 1988). The SCR amp. = .303 μS, the distance between the point of onset and the calculated peak point = 2.125 sec, and the SCR rec.t/2 = 3.602 sec. The time point for the theoretical termination of the SCR is defined as approximately the tripling of the SCR rec.t/2. The curve shown in Figure 40 is an empirical interpolation by means of a third-grade function group in relation to the decline.

As long as systematic comparisons of the different methods of determining the EDR decline parameters and more exact mathematical functions for the observed EDR decline forms are lacking, the evaluation can be supported by considerations of practicality. Undoubtedly, for the evaluation on paper the determination of EDR rec.t/2 is

simpler than that of the EDR rec.tc. Furthermore, the half point is more easily attained than the 63% recovery point. Since the decay of radioactive matter which in fact follows an *e*-function is nevertheless expressed in half life time, the present author does not see a convincing reason for using EDR rec.tc instead of EDR rec.t/2 (see also the computer program in the appendix).

In the case of curvilinear writing, as used in some polygraph systems, it must be noted that the drawing itself distorts the form parameter especially when the drawing goes over the middle third of the writing channel (Edelberg, 1970).

The incompleteness of matching negatively accelerated *e*-functions to observed EDR decline curves leads to implications with respect to discussions in later sections. It is partly the independence of the time constant τ from the initial value of such a decay process that provides the basis for the assumption of independence between EDR rec.tc and EDR amp. (Sect. 2.5.2.5). The arguments used by Sagberg (1980) for the differing recovery of SC and SR values (Sect. 2.6.2) are based on an assumption that the decline forms of SCR and SRR are well enough approximated through a single *e*-function.

Frequently neither EDR rec.tc nor EDR rec.t/2 can be determined because before a corresponding recovery point is reached another EDR has already started (Fig. 37 in Section 2.3.1.2.2, EDR type 2). In that case, Fletcher, Venables, and Mitchell (1982) recommend calculating the EDR rec.t/4 instead. Using samples of more than 1,000 subjects in total, correlations between SCR rec.t/2 estimated from the log SCR rec.t/4, and the actually measured log SCR rec.t/2 were around $r = .90$. Using SCR rec.t/4 instead of SCR rec.t/2, they were able to obtain a 23% increase in the number of SCRs for which decline parameters are calculable. Waid (1974) used the SRR rec.t/3 and also significantly increased the number of SRRs with calculable recovery values.

When the EDR rec.t/2 was not obtainable, Foerster (1984) determined the tangent in the turning point of the decline of the curve. The intersection point of this tangent with the parallels of the time axis at half amplitude is usable for the extrapolation of the fall times for the turning point, which however must be evaluated separately from the other recovery times (see also the computer program in the appendix).

It is doubtful whether the evaluation of the recovery of relatively rapid overlapping EDRs provides homogeneous, reliable, and valid information. It should at least be attempted to ensure that the forms of the calculated EDRs are in fair concordance with that of an individual normative EDR obtained within a recording period without any overlays. As ascent and descent times show a clear correlative connection (Sect. 2.5.2.4), following Venables and Christie (1980), the replacement of the EDR recovery by the EDR ris.t. as an adapted parameter of reaction shape may be used (Sect. 2.3.1.3.1).

Some authors prefer the use of *recovery rate* instead of recovery time (see Sect. 3.4.2.1). Recovery rate is expressed as ohms gained per sec during EDR rec.t/2 (Mednick & Schulsinger, 1968).

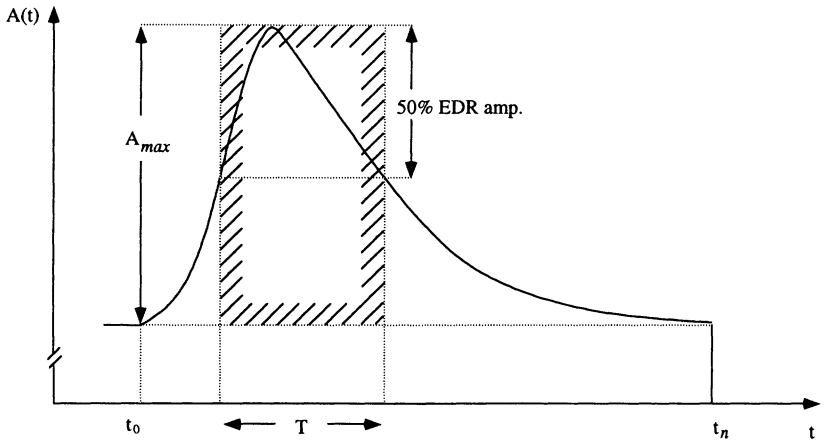

Figure 41. Approximated area (shaded) under the EDR curve. (See text for explanations.)

2.3.1.4 Area measurements

Traxel (1957) has suggested using an extended concept of electrodermal recovery for a further description of the EDR. He assumed that not only the peak amplitude but also the EDA recorded at every point during the course of an EDR may be regarded as a value for the "strength of affect" (Sect. 3.2.3.2). Consequently, the calculation of the particular integral which corresponds to the area under the curve between the starting and end points of the EDR serves as the value for the entire "quantity of affect" (Traxel, 1957) as follows:

$$F = \int_{t_0}^{t_n} A(t)dt \tag{39}$$

whereby F represents the area under the curve between t_0 (starting point) and t_n (end point), and $A(t)$ represents the EDR amp. to the time point t (Fig. 41).

As it is practically impossible to determine t_n exactly (Sect. 2.3.1.3.2), Traxel recommended approximating the area under the curve by multiplying the EDR amp. by the time T the curve exceeds half of the amplitude (see the shaded rectangle in Fig. 41):

$$\hat{F} = A_{max}T \tag{40}$$

where $\hat{F}$ is the approximate area and A_{max} the EDR amp. According to Traxel (1957), the correlation between this approximation and the area measured with a planimeter

was .91 for 50 SRR scores. Schönpflug, Deusinger, and Nitsch (1966) pointed to the dependency between amplitudes and time parameters of the EDR (Sect. 2.5.2.5), therefore generally questioning the validity of area measures. In contrast, Lüer and Neufeldt (1967, 1968) showed that a moderate correlation between the EDR amp. and the half-life period[57] does not lead to a decrease in the validity of the area measurement obtained from these parameters, but may have more validity than each of them alone. Up to now, area measurements are not in use in the international psychophysiological literature.

2.3.2 Parameters of tonic electrodermal activity

The tonic EDA values are discussed after the phasic parameters for two reasons. First, the tonic values discussed in Section 2.3.2.2 are derived from phasic values, and therefore the phasic parameters must be known beforehand. Second, the level values discussed in Section 2.3.2.1 are, at least for the most widely used exosomatic measurement, of less practical significance than the specific and nonspecific EDRs, as they prove to be less reactive to variations of experimental conditions. Therefore, in many investigations a special evaluation of the EDL is not performed.

2.3.2.1 Electrodermal level

The determination of real electrodermal level values is not as easy as it might appear at first glance: although at any given time point an EDL score can be recorded, a true level score can, however, only be obtained when it is not in the EDR range. While the usual time constants for the ascent of the EDR are around .5 sec and for the descent of the EDR around 4–6 sec (Sect. 2.3.1.3.2), for EDL displacements, time constants from 10–30 sec must be considered. If the chosen evaluation point falls within the range of the EDR, the time point can be shifted without significantly affecting the reliability of measurement. Such a displacement is easy to perform during inspection of paper recording (Sect. 2.2.4.1). However, if computer analysis does not include interactive work on the video display screen (Sect. 2.2.4.2), EDL scores distorted by an actual EDR can only be avoided with the use of averaging techniques. This is done by forming the average EDL of all artifact-free scanning points with an adequate interval (e.g., 10 sec). However, the so-formed EDL is likely to be overestimated in states of high arousal (Sect. 3.2.1.1), due to the greater number of EDRs.[58] To avoid this overestimation of the EDL, use could be made of the EDL minimum during a certain time interval in an automatic, nonoptically controlled computer analysis. This is not fully recommended,

[57]The half-life period is defined by these authors – in contrast to the nomenclature used in Section 2.3.1.3.2 – as the time the curve remains over the half amplitude (T) in Fig. 41). It therefore includes the time characteristics of the ascent as well.

[58]The same is true for the EDL values which are calculated from the decoupled AC curve of overlying impulses (Sect. 2.1.3 & 2.2.4.2).

however, since false minima can appear in the evaluation because of movement artifacts (Sect. 2.2.5.2).

More exact values are provided by the averaging of all respective EDL scores measured at the beginning of an EDR. These are available from the calculation of the EDR amp. (Sect. 2.3.1.2.2; Fig. 36), whereby the EDLs at the beginning of superpositioned EDRs (Fig. 37) must be excluded. Sufficiently reliable data can be expected from such a process only when enough EDRs appear during the interval in question.

Another EDL score is available in endosomatic EDA evaluation, the BSPL (low-basal skin potential level), proposed by Christie and Venables (1971). Lykken, Rose, Luther, and Maley (1966) still found interindividual differences in minimum SPL in fully relaxed subjects; Venables and Christie (1980) conjectured, on the basis of theoretical considerations and investigations from their research group, that the BSPL corresponds to the membrane potential E_3 in the Fowles model (Fig. 17 in Sect. 1.4.3.2). This BSPL, quickly obtainable after a long resting period from fully habituated subjects, can be regarded as the individual minimum score of the SPL and can be used for setting the time points in the measurement of the minimum SCL for range correction (Sect. 2.3.3.4.2).

2.3.2.2 Tonic parameters derived from phasic changes

As already mentioned in Section 1.1.1, electrodermal recording yields phasic variations which are not traceable to specific stimuli and therefore are known as "electrodermal spontaneous fluctuations" or "nonspecific EDRs." Since internal or external stimuli cannot be demonstrated, these EDRs are regarded as being an expression of tonic EDA. Thus, the frequency of the nonspecific EDRs (NS.EDR freq.) with respect to a fixed time interval, usually *1* min, leads to another tonic EDA measure.

It supposedly can be concluded that the EDL (Sect. 2.3.2.1) and the NS.EDR freq. are autonomic parameters of tonic EDA. Venables and Christie (1980) summarize the investigations to date comparing the SRL and NS.SCR freq. and conclude that while these two tonic measures are correlated, each can still have differential validity. This aspect is more thoroughly discussed in Section 3.2.1.1.

If NS.EDRs are to be determined during a measurement phase where defined stimuli also appear, EDRs which are traceable to specific stimuli must not be considered. As a conservative rule, EDRs which appear up to 5 sec after the start of an intentional or unintentional stimulus should not be regarded as NS.EDRs. Additionally, the end of a stimulus can be regarded as the trigger of a specific EDR as well, to avoid the inclusion of false positives, only EDRs which begin later than 5 sec after the end of a stimulus should be evaluated as nonspecific (Fig. 42).

Another method of determining NS.EDR measurements which are free of specific EDRs and artifacts is described by O'Gorman and Horneman (1979): the EDA recording is divided into 10 sec segments; then all segments in which movement and breathing artifacts are recorded (Sect. 2.2.5.2) together with all segments which directly follow

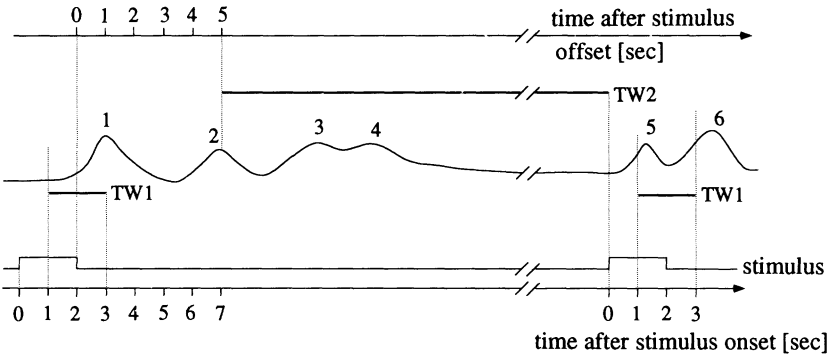

Figure 42. Separation of specific and nonspecific EDRs. TW1 = time window for specific EDRs, TW2 = time window for the NS.EDRs. 1 and 6 are specific EDRs following prior stimulation, 3 and 4 are NS.EDRs in the interval between both stimuli, 2 and 5 could not be evaluated as either specific or nonspecific EDRs.

these are excluded from further evaluation. The remaining artifact-free 10 sec segments are divided into those in which "large" NS.EDRs (amplitudes greater than 1% of the EDL) appear, and those in which "small" NS.EDRs (amplitudes smaller than 1% of the EDL) appear. The number of both types of segments is contrasted to the total number of artifact-free intervals. However, the possibility of discovering small NS.EDRs is highly dependent upon amplification (see Sect. 2.3.1.2.3). For the determination of the NS.SPR freq., problems may possibly arise for biphasic and triphasic SPRs (Sect.2.3.1.2.1), since SPRs quickly following each other under high activation cannot be separated from each other with certainty (Venables & Christie, 1980).

Besides this frequency measure, means and standard deviations of the amplitudes of all NS.EDRs can be computed as additional parameters of tonic EDA. One measure that principally falls into this category is the so-called EDA magnitude, which due to the inclusion of zero reactions is discussed in connection with the missing data handling in Section 2.3.4.2. Edelberg (1967) proposed using the frequency of changes in the EDL within a certain time span instead of the NS.EDR freq. as an indicator of arousal, a method which has not yet been investigated. Recently, Besthorn, Schellberg, Pfleger, and Gasser (1989) used the variance of the SCR amp. over the whole stimulation time as a tonic EDA measure in continuous audiovisual stimulations lasting from 1.5–3 min.

2.3.3 Transformation of electrodermal parameters

In general, the usefulness of data transformation has been treated controversial in the literature on statistics and methodology. Levey (1980), who gives a comprehensive discussion of this topic, states that ideally, transformations would be necessitated by known or assumed properties of the system under investigation. However, the transformations that are relatively commonly used in EDA research are seldom based on physiological or systems-theoretical considerations. Usually it is statistical considerations that lead to the transformations of EDA raw data (e.g., Edelberg, 1972a; Venables & Christie, 1980).

Transformational procedures must be judged by the analysis of the derived measure, wether or not the procedure leads to an improvement of the validity of the EDA measure with respect to the representation of the underlying psychophysiological processes. Since corresponding generalizations on the respective specific experimental contexts have not been fully possible to date (Levey, 1980), the question of whether statistical analysis should be carried out with transformed data or with raw data still cannot be answered in general.

2.3.3.1 Taking the electrode area into account

A transformation that is especially useful in the application of the constant current technique is the calculation of scores related to the electrode area, called *specific* EDA scores (Sect. 2.2.3.2). As a result of the reciprocal relationship between resistance and conductance, the specific resistance is expressed in $k\Omega \times cm^2$, while the specific conductance is expressed in $\mu S/cm^2$ (Edelberg, 1967). Correspondingly, the impedance and admittance values can be related to the electrode area. Such a transformation is not common in endosomatic measurement. On the basis of the differing principles of measurement as outlined in Section 2.1.1, it can be shown that the calculation of specific resistance is of greater importance than calculation of specific conductance. Underlying a model of parallel resistances/conductances, an increased electrode area in the constant current method means that the current is divided by more pathways (Sect. 1.4.3.1). Although the current is limited in total, the current per pathway decreases (since the effect of the applied constant current is dependent upon the current density and thereby upon the electrode area). In the case of the constant voltage method, by contrast, the electrode area plays no role, since with an increase in the number of parallel pathways the applied voltage on any pathway remains constant (Sect. 2.6.2).

In spite of this, Lykken and Venables (1971) have supported reporting SC scores as specific conductances as well, because these authors have empirically found a linear relationship between the electrode area and the SC. However, Venables and Christie (1980), relying on data reported by Mitchell and Venables (1980), conclude that no such linearity exists between the SCL or SCR amp. and the electrode area, because an increase above .8 cm^2 of the electrode area does not appear to increase conductance

(Sect. 2.2.2.3). Therefore, they do not endorse a weighting of the SC scores to the electrode area. Instead, for purposes of comparison, the size of the electrodes should be given, not the specific conductance. In contrast, Mahon and Iacono (1987) found a linear relationship between electrolyte-skin contact areas, varying in size in six steps from .131 cm^2 to .786 cm^2, and the SCL/SCR amp. Thus, they also support reporting the specific conductance. There are no data available concerning a possible dependency of the form parameters of EDA upon the electrode area.

2.3.3.2 Transforming resistance into conductance units

After Lykken and Venables (1971) had strongly recommended the use of the constant voltage method for exosomatic EDA measurement, many authors who continued to use the constant current method transformed the obtained SR units into SC units before further statistical evaluation. If computerized EDA evaluation is available, (Sect. 2.2.4.2 & 2.2.4.3), this can be done by transforming the SRL into the SCL for every point sampled with Equation (4a) in Section 1.4.1.1. For this purpose Foerster (1984) provided a FORTRAN program using a sampling rate of 12.5 Hz. If the parametrization has already been performed by means of the SR recording curve, transformation into conductance is more complicated. According to Equation (5a) in Section 1.4.1.1, the SRL scores at the onset of the respective reactions must be known in order to convert the SRR amp. into the SCR amp. For simplification, the square of the SRL score at the onset of the reaction is used as the denominator instead of the product from the SRL scores of the reaction's onset and its maximum. This is possible because the difference between both SRL scores is usually small compared to the SRL scores themselves. The conversion follows Equation (42a) in Section 2.3.3.4.1.

Hagfors (1964) points out that through the transformation of the SRR amp. into the SCR amp., variations in the ranking of the amplitudes can be given if the corresponding EDRs result from differing EDLs. Thereby, the conversion of SR into SC units can also lead to other results with respect to the EDR amp. However, within the range of stimulation usually applied in psychophysiological experiments, SCR amplitudes as calculated from SRR amplitudes obtained by constant current recording can be expected to yield results comparable to SCR amplitudes measured directly by using constant voltage. [59]

On the other hand, form parameters may not show the same kind of invariance to resistance-conductance transformations. Sagberg (1980) has empirically and theoretically demonstrated that the SCR rec.tc must be shorter than the corresponding SRR rec.tc. However, he used a single e function for modelling EDR recovery which is questionable (Sect. 2.3.1.3.2.).

[59] This has been demonstrated by Boucsein et al. (1984a) using both recording methods in parallel during the application of 2-sec white noise stimuli with intensities between 60 and 110 dB.

2.3.3.3 Improving distributional characteristics of EDA

In order to improve the distributional characteristics of EDA data, especially with respect to skewness, log transformations are primarily used. Edelberg (1972a) and Venables and Christie (1973) regarded logarithmic transformations of SC scores as unnecessary because of their relatively normal distributions. Later, Venables and Christie (1980) advocated a different point of view based on voluminous data sets from three large-sample investigations. They found clear improvements in both skewness and slope through the log transformations of the SCL scores. These improvements were also true of SCR amplitudes if the log transformations were done following the determination of the amplitudes, but not if the amplitudes were already determined by means of log transformed raw scores (i.e., as the difference between the log SCL on the peak response and the log SCL at the response onset).

Venables and Christie (1980) also investigated the unusual log tranformations of latency, rise time and recovery measures of the SCR. They found clear improvements in the distributional characteristics through these transformations for the SCR rec.t/2, though not for the SCR lat. and the SCR ris.t. Why a log transformation leads straight to a normalization of the EDA data cannot be statistically determined (Levey, 1980).

Two mathematical considerations on the problem of using log transformations should be noted:

(1) If EDA is evaluated as stimulus dependent and, as in experiments on habituation (Sect. 3.1.1.3), zero reactions are expected, the log SCR amp. is mathematically undefined. Venables and Christie (1980) suggest that in this case 1 should be added to all SCR amp. scores before the log transformation is performed.

(2) To transform SR scores which have already been logarithmically transformed into SC scores, the following transformation should be used (Edelberg, 1967): $\log G = -\log R$, which follows from $G = 1/R$. As G is expressed in μS and R in kΩ (Sect. 1.4.1.1) and $\log 1000 = 3$, then according to Equation (4a):

$$\log G \quad [\mu S] = 3 - \log R \quad [k\Omega] \tag{41}$$

Another transformation widely used for the improvement of distributional characteristics is the square root transformation (Grings, 1974). This transformation is suited to the normalization of a Poisson distribution for rare events such as those observed in many physiological processes (Levey, 1980). In EDA evaluation, it has been mostly applied to SCR amp. scores.

Both square root and log transformations are sometimes used in addition to other transformations (e.g., weighting with respect to the electrode area) (Sect. 2.3.3.1). A distributional normalization of the EDR amp. can also be attained through a standardization to z scores (Sect. 2.3.3.4.3).

Some authors subject the EDR recovery times to a reciprocal transformation with the intention of obtaining the recovery speed. This so-called *recovery rate* leads to a deviation – stronger than the recovery – from the normal distribution, so that a reciprocal transformation of the recovery times cannot be recommended from the point of view of distribution (Sect. 2.5.2.4).

2.3.3.4 Reduction of interindividual variance

Several transformations, which can be completed before the further statistical treatment of the EDR data from various subjects, lead to a reduction of the interindividual variance. Among the reasons for their use are the supposed baseline dependence of the EDR data (Sect. 2.5.4.2), the aim of expressing the respective EDR in terms of the individual reaction range, and the matching of the distribution of the EDRs from various subjects to each other in order to achieve better preconditions for a group analysis.

The methodological and statistical implications of these transformations are discussed here only insofar as they can be regarded as being EDA specific; for further discussion the respective literature can be referred to.

2.3.3.4.1 Evaluating EDR with respect to EDL. A simple way of building transformed EDR scores, with the respective EDL taken into account, is the forming of a quotient from the EDR amp. and the EDL immediately before the start of the appropriate EDR. This quotient can also be expressed as a percentage: EDR amp./EDL × 100% (Traxel, 1957). Edelberg (1967, p. 47) purports that such a transformation is unnecessary in the case of SC measurements, because according to Equation (5a) in Section 1.4.1.1, ΔG (i.e., the SCR amp.) already takes note of the resistance level in the denominator. However, Edelberg's claim only makes sense in the case where SC is regarded as the sole adequate measurement unit (Sect. 2.6.5), because, correspondingly for ΔR, the basic conductance is already represented in the denominator, as can be seen in Equation (5b).

A further relationship can be derived from Equation (5a), as done by Edelberg (1967): as the difference between R_1 and R_2 is relatively small in comparison to R_1 and R_2 themselves, ${R_2}^2$ can be used as an approximation instead of the product of both values in the denominator of Equation (5a) in Section 1.4.1.1. That is, if R_2 is replaced by R for the sake of simplicity:

$$\Delta G = -\frac{\Delta R}{R^2} \tag{42a}$$

Both sides of Equation (42a) are multiplied by R, and on the left-hand side R is replaced by $1/G$, according to Equation (2b):

$$\frac{\Delta G}{G} = -\frac{\Delta R}{R} \tag{42b}$$

Equation (42b) shows that the relative variations of the conductance and resistance are approximately equal in their absolute values. The corresponding transformations therefore lead to the same data independent of whether the raw scores are conductance or resistance values.

2.3.3.4.2 Range corrections. Transformations that reduce interindividual variance so that every value is set in proportion to the intraindividual range are known as range correction procedures. The "correction" is based on the assumption that the part of interindividual physiological differences which does not relate directly to the psychophysiological processes can be eliminated (Levey, 1980). A precondition for this procedure is the determination of the intraindividual range of maximal reactivity. Paintal (1951) determined the maximum possible EDR amp. by applying a strong electrical stimulus and dividing each EDR amp. by this value (Paintal index, cf. Edelberg, 1972a). Lykken and Venables (1971) recommended inflating a balloon until it exploded in order to determine the maximal SCL. It remains, however, questionable whether these two techniques approximate the ideal of recording the physiological capacity free from psychological influences. Lykken et al. (1966) give the following formula for range correction:

$$SCL_i' = \frac{SCL_i - SCL_{min}}{SCL_{max} - SCL_{min}} \tag{43a}$$

where SCL_i is the uncorrected level value and SCL_i' the range corrected level value at the time point i, while SCL_{max} is the highest possible value and SCL_{min} the lowest possible value for the particular subject. The determination of the minimum value is much more difficult than that of the maximum value. Venables and Christie (1973) suggest using the SCL obtained during the appearance of the BSPL (Sect. 2.3.2.1), thus requiring simultaneous recordings with both exosomatic and endosomatic techniques (Sect. 2.2.6.2).

The determination of a minimum value appears not to be a problem, at least in theory, for the EDR amp., as the minimum EDR can be taken as a nonexistent EDR. Accordingly, Lykken and Venables (1971) derive the range correction for the SCR amp. from Equation (43a), in which $SCR_{min} = 0$ has been inserted, as follows:

$$SCR_i' = \frac{SCR_i}{SCR_{max}} \tag{43b}$$

Correspondingly, in Equation (43b), SCR_i' is the corrected and SCR_i the uncorrected skin conductance reaction, while SCR_{max} is the maximum possible SCR amp.. Following Venables and Christie's (1973) method, the maximum SCR_{amp}. can be determined during the experiment with the application of loud tones or electrical stimuli, or through deep breathing. The orienting response to the first stimulation of an habituation paradigm will also usually display the maximum EDR of the experiment (Sect. 3.1.1.3).

Additional problems may appear in the use of range correction. Grings (1974) has shown that the range, especially in small samples, is usually an unreliable value which is highly dependent upon the situational conditions of the respective experiment. Sagberg (1980) has shown that range correction is not invariant throughout transformations of SR into SC values and vice versa. Ben-Shakhar (1985) cautions that the greatly extended rest period necessary for the determination of the minimum EDL, together with manipulations for the determination of the maximum EDR amp. and/or the highest EDL, may invalidate the whole experiment.

2.3.3.4.3 Transformation to standard values. The problem of determining individual EDR maxima that appear in range correction as described in the last section can be bypassed if standardization is based upon the individual mean and standard deviation of EDRs instead of their range. The raw scores can then be turned into standard values with the appropriate transformation.

For a transformation of the EDR amp. into z scores, undertaken, for example, by Ben-Shakhar, Lieblich, and Kugelmass (1975), means and standard deviations of the recorded EDRs are calculated for each particular subject. Then a standard value is calculated for each EDR amp. as follows:

$$z_{ik} = \frac{X_{ik} - \bar{x}_i}{s_i} \tag{44a}$$

where X_{ik} is the raw score and z_{ik} the standard value of the subject i for the EDRk; together with the mean $\bar{x}$ and the standard deviation s_i of all EDRs of the subject i.

The z scores are normally distributed with an average of 0 and a standard deviation of 1. It is mostly usual to transform z scores into T scores as follows:

$$T_{ik} = 50 + 10z_{ik} \tag{44b}$$

The T scores are normally distributed with a mean of 50 and a standard deviation of 10; therefore minus signs drop out.

In 147 subjects, Ben-Shakhar (1985) performed systematic comparisons between SCRs in raw score form calculated from SRRs and range-corrected as well as z-transformed electrodermal reaction scores. He found that the reaction scores obtained through standard transformations differentiated most clearly between meaningful and neutral stimulus conditions, a finding which he ascribed to the z-transformed average individual reactivity being more representative than the rather unreliable maximum individual reactivity which was used for calculating range-corrected scores. However,in a simulated data study, Stemmler (1987) showed that the design used by Ben-Shakhar, together with the z-score calculation and the inferential statistics used for the determination of the differences between conditions, resulted in a positive bias for the method of standard transformation. Therefore, the generalizability of Ben-Shakhar's argument remains in question.

2.3.3.4.4 Using autonomic lability scores. Lacey (1956) suggested a method of standardization for psychophysiological reaction scores which considers the respective baseline values (Sect. 2.3.3.4.1) as well as undertakes a standard transformation (Sect. 2.3.3.4.3). These so-called autonomic lability scores (ALS scores) form reaction scores, where the component which is predictable by means of linear regression from the level score is ruled out. Thus, they can be regarded in principle as covariance analytical adjusted scores (Grings, 1974).

In order to calculate the ALS scores, all n EDRs obtained from a subject i in a sequence $k = 1, ..., n$ are collected together, as in the standard transformation described in the previous section. However, instead of using the EDR amp., for each EDR the EDL at the reaction onset, as X_{ik}, and the EDL at its maximum, as Y_{ik}, are determined (i.e., $Y_{ik} - X_{ik}$ = EDR amp.$_{ik}$). According to Lacey, the score sequence X_{ik} builds up the baseline values, while the sequence Y_{ik} builds up the reaction scores. Between both score sequences the correlation$(r_{xy})_i$ is calculated for each subject i. The values X_{ik} and Y_{ik} are transformed into standard values $(z_x)_{ik}$ and $(z_y)_{ik}$ using their respective means, $\bar{x}_i$ and $\bar{y}_i$, and their standard deviations $(s_x)_i$ and $(s_y)_i$, according to Equation (44a). Then an ALS score is calculated for each EDR k of a subject i as follows:

$$ALS_{ik} = 50 + \left(10\left[\frac{(z_y)_{ik} - (r_{xy})_i(z_x)_{ik}}{\sqrt{1 - (r_{xy})_i^2}}\right]\right) \tag{45}$$

These ALS scores are, like the T scores in Equation (44b), normally distributed with a mean of 50 and a standard deviation of 10, and are linearly independent from the baseline values of the respective subject.

The calculation of ALS scores presupposes a large number of EDLs per subject, as a correlation between baseline and reaction scores must be determined for each subject.[60] In addition, Johnson and Lubin (1972) pointed out that ALS scores only have an advantage over raw scores with respect to reliability and validity if the so-called law of initial values holds true (for further discussion, see Sect. 2.5.4.1).

Levey's (1980, p. 621) opinion of the application of ALS scores to SCRs may serve as an example of the problems of transformations in general (see the introductory remarks in Section 2.3.3) and of the problems of ALS transformations in particular. He interpreted the results of an investigation published by Germana (1968) showing that ALS and log EDR amp. transformations differ from each other in nearly the same way as both differ from the EDR amp. raw scores. Levey then argued against the ALS correction, as it only corrected the baseline effects, and argued for log transformation as derivable from theoretical considerations and modelling.

[60] ALS scores can be standardized not only intraindividually over the different EDRs, but also interindividually for each reaction over all subjects. In this case both score sequences X and Y are calculated by means of the EDRs of all subjects to the same stimulus. In any case, this process does not lead to a reduction of interindividual variance.

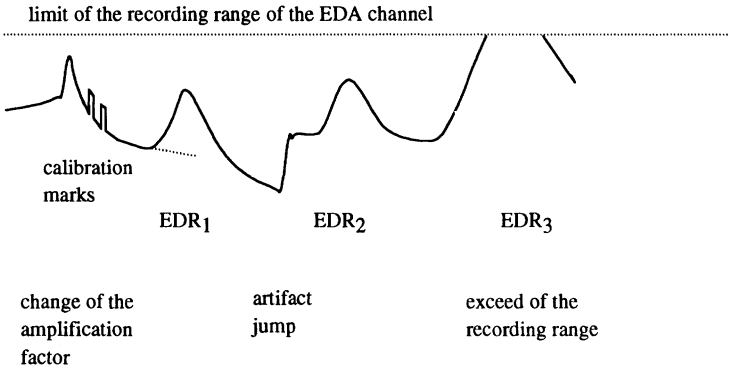

Figure 43. Examples of artifacts in an EDA recording sequence. (See text for explanations).

2.3.4 Removing artifacts and treatment of missing data

In spite of exact control of measurement techniques, the behavior of the subject and/or the environmental conditions during EDA measurement can lead to the appearance of artifacts (Sect. 2.2.5) which must be eliminated before further statistical treatment of the EDA data. Therefore, identification of artifacts, as described in Section 2.3.4.1, plays a significant role after the parametrization of the EDA signal, because the evaluator must choose between correcting or discarding a data value artifactually created.

The gaps created by missing data (e.g., if the electrodes are detached; Sect. 2.2.2.1 & 2.2.5.1) necessitate corresponding treatment (Sect. 2.3.4.2). A formal missing data treatment may also be necessary in the EDA evaluation if the stretch of data in question does not need to be deleted, as when, for example, expected EDRs fail to appear due to genuine zero reactions, as during advanced habituation (Sect. 3.1.1.3), in extinction (Sect. 3.1.3.1), or with electrodermal nonresponders (Sect. 3.4.2.2).

2.3.4.1 Identification of artifacts during measurement

The detection of artifacts in the EDA signal necessitates a visual inspection of the data sequence by the experimenter, even if an automatic parametrization is performed by means of laboratory computers (Sect. 2.2.4.2). To facilitate this procedure, all artifacts created by changes of amplification factors and/or calibration as well as movement (insofar as they can be identified from the record) should be noted during the recording.

Figure 43 shows some typical artifacts in an EDA record segment. A change of the amplification factor causes a transient response which appears as a jump in the record-

ing, followed by an exponential adaptation to a new baseline. This process might not be terminated until the occurrence of the following EDR, thus obscuring its course (as indicated by the dashed line in Fig. 43). Jumps in the EDA curve can also result from movements of the electrodes or the area to which they are attached (Sect. 2.2.5.2), shown as an artifact jump in Figure 43. Since these usually do not result from an exponential adjustment, the EDR_2 in Figure 43 can be unequivocally evaluated, in contrast to the EDR_1. Computer programs can automatically recognize and correct such jumps (Foerster, 1984) or enable correction through interactive work with the video screen display (Thom, 1988). Andresen (1987), having used an interactive artifact correction with a big data series, advocates its use in spite of being time consuming. The present author prefers automatically removing artifacts as performed by the computer program in the appendix of this book. Calibration marks can influence evaluability, especially if they are produced during the rise time or the reaction maximum of an EDR. For automatic evaluation, they must be eliminated.

EDRs which exceed the recording range, such as the EDR_3 in Figure 43, have to be excluded from evaluation. They can appear, for example, as part of an orienting or defensive response to an unexpected, strong stimulus (Sect. 3.1.1.2). Although in this particular case they are not artifacts with respect to the goal of the investigation (Sect. 2.2.5.2), the questionable recording sequence must be excluded from parametrization. Such EDRs also appear subsequent to strong respiratory activity (e.g., sighing) or gross physical movement. Escapes from the recording range can be avoided either by choosing a larger bandwidth (e.g., through less amplification) or by using automatic reset procedures (e.g., Thom, 1988).

An EDA artifact can be performed by visually inspecting both EDA and respiratory curves (Fig. 44). No connection with inhalation can be seen with the EDR_1 in Figure 44. But the EDR_2, which follows a sufficiently long latency period in the time window after an excessively deep breath, has to be treated most probably as a respiratory artifact. Problems of interpretation arise if the inhalation as well as the EDR in a corresponding time window can be regarded as covariant indicators of an orienting or defensive reaction. In such case the EDR is not merely an artifact; however, it is also not independent from breathing. Artifacts resulting from the subject's speech activity cannot be easily determined, since the relationship of speech activity to single EDRs is less clearly recognizable.

2.3.4.2 Missing data treatment and EDR magnitude

Missing data in EDA parametrization can be handled in the same way as it is in other biosignals. Since many statistical packages do not have missing data procedures, intended supplements of the data sets should be implemented directly following parametrization.

For missing data treatment, a time window grid could be laid over the data so that the smallest window is defined for the statistical evaluation. Depending on the problem

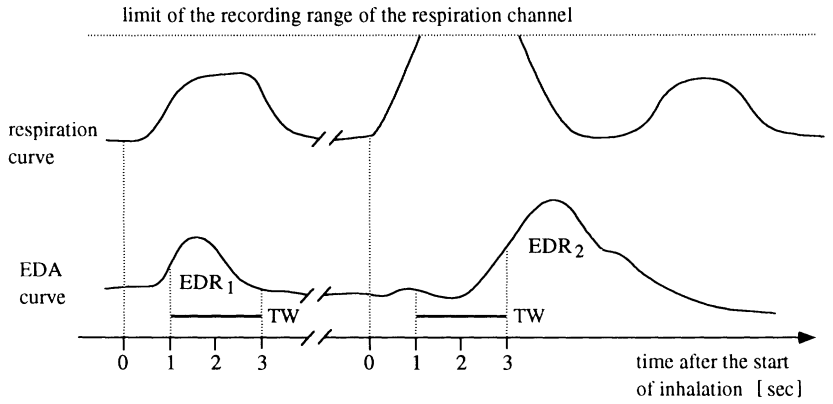

Figure 44. EDA artifact identification with the help of the respiration curve. TW: Time window of a respiratory artifact, suggested 1–3 sec after the start of inhalation. As the EDR_1 does not begin within the time-window, it does not result from a respiratory artifact, while the EDR_2 does.

in question, this window can be between 5 sec and 1 min. For the NS.EDR freq., EDRs in each interval are counted (Sect. 2.3.2.2). If no EDRs appear within a certain window, it is normally a case of zero reactions (see below), not of missing data. If only a part of the window under evaluation is so error prone that it is impossible to parametrize it, the weighted mean may be calculated, taking the ratio of an estimable time span to the total window into consideration. If a considerable part of the window has to be excluded from evaluation, the score of either the preceding or the subsequent window may be inserted, as long as no clear change of the situational condition has occurred in the meantime.

While such corrections as a rule can be usefully made in the case of lost EDL scores, the case of possible lost NS.EDRs is more questionable because the appearance of spontaneous EDRs is a nonpredictable event. Therefore, it is not possible to proceed from an approximately equal distribution of EDRs in neighboring same-length time windows. Thus records of a particular subject containing large portions of missing data should be excluded from the statistical evaluation of NS.EDRs. Missing data corrections for stimulus-dependent EDRs cannot be recommended, neither intraindividually, due to the possible appearance of habituation (Sect. 3.1.1.3), nor through group analysis, due to the large interindividual differences.

A parameter which is often calculated for the description of electrodermal habituation processes, which takes into account expected but nonappearing stimulus-dependent

EDRs, is the EDR magnitude.[61] The inclusion of such "zero reactions" presupposes that it is possible to clearly define when EDRs are to be expected within the course of an experiment, which is only true with exactly defined and recorded stimuli. EDR magnitude is normally used in an interindividual averaging procedure (e.g., to determine the average reaction strength to a certain stimulus in an habituation series), but it might be used as an intraindividual measure as well (e.g., to obtain the total reactivity of an individual measure as well (e.g., to obtain the total reactivity of an individual to a series of stimuli).

Which measure, mean amplitude or mean magnitude, is the more adequate one for determining the average strength of EDRs, remains debatable. Arguments for EDR magnitude are the difficulty of defining zero reactions (e.g., see Sect. 3.4.2.2) and having the same sample size for each cell in further statistical analysis (Venables & Christie, 1980). On the other hand, the magnitude measure confounds frequency of response and response strength which do not necessarily co-vary (Prokasy & Kumpfer, 1973). Both methods of missing data treatment may produce considerably differing results. For example, if habituators and nonhabituators are combined, different habituation courses will be obtained when using the interindividual mean EDR amplitude instead of the more commonly used mean magnitude; the mean magnitude will yield an overall habituation, while calculating the mean amplitude will result in an habituation followed by an increase of reaction strength in later trials. [62]

2.3.4.3 Correction for EDL drift

The supplementary correction of drift in the EDA signal is only possible if control scores are registered during data recording. For example, this is possible with the regular setting of calibration marks (Sect. 2.2.4.1); if the signal curve alters with the same value resistances and constant amplification, a drift has appeared. All drift correction is costly in terms of calculation and prone to high uncertainty. However, in the case of long-term runs (Sect. 2.2.6.1), such corrections are usually indispensable.

Drift resulting from electrode polarization in exosomatic direct voltage measurement can be checked by variation of the polarization during long-term measurement phases. Drift correction is not possible in endosomatic measurement (Sect. 2.2.5.1).

Body temperature as well as ambient temperature can be used for EDL drift correction. Here Grings's (1979) empirical value of 3% SRR elevation per ^{0}C decrease can be used. As long as the temperature drift results from the device used, the amplifier can be allowed to run free of an input signal for the same length of time as the experiment's

[61] Some authors use the term "EDR magnitude" instead of "EDR amplitude" (Sect. 2.3.1.2); therefore, care should be taken in ascertaining just which method of evaluation is used in the respective publications.

[62] Mathematically, this is a type of missing data treatment, as nonappearing EDRs, or EDRs which remain below an amplitude criterion (Sect. 2.3.1.2.3), are taken into account in evaluation. The EDR magnitude (or, really, the mean EDA magnitude) is calculated by dividing the sum of the evaluated EDR amp. by the number occasions on which EDRs might have been expected.

measurement time, and the resulting recorded SCL "signal" is used to correct drift in the remaining recorded data.

A pragmatic reduction of drift due to any cause can be made using the trend validation methods proposed by Stemmler (1984). Here, EDL differences in reference phases of identical structure (e.g., simple resting periods), which shortly precede or follow the experimental conditions under investigation, are calculated, and a linear interpolation in real time is calculated.

2.3.5 Summary of scoring techniques

Depending on the problem under investigation, different parameters of phasic as well as tonic electrodermal activity can be extracted from the recorded EDA signal. The most commonly used phasic measure is the EDR amplitude (Sect. 2.3.1.2), where amplitude criteria must be defined, because of possible individual differences in the amplification factors used (Sect. 2.3.1.2.3). The next most commonly extracted phasic measure is the form parameter of the recovery, such as the half recovery time (Sect. 2.3.1.3.2); ascent parameters are less used (Sect. 2.3.1.3.1). If the EDRs follow defined stimuli, latency times can be determined, as long as temporal resolution is high enough (Sect. 2.3.1.1). As a tonic measure, the frequency of non-stimulus-specific EDRs (Sect. 2.3.2.2) is used, next to recordings of the EDL (Sect. 2.3.2.1). Evaluating NS.EDR freq. requires a separation of nonspecific from specific EDRs by using the corresponding time windows (Fig. 42).

With respect to all possible transformations, the transformation of resistance into conductance units is used overwhelmingly, following the suggestion for standardization made by Fowles et al. (1981); the range correction recommended by Lykken and Venables (1971) is frequently used for the reduction of interindividual variance. In all cases it should be determined whether the intended transformations are really necessary with respect to possible improvement of reliability and validity.

Much care should be taken with the identification and elimination of artifacts and drift, whereby records of other biosignals can be helpful (Sect. 2.3.4.1 & 2.3.4.3). If missing data problems or genuine zero reactions are present, the EDR magnitude can be calculated instead of the mean amplitude (Sect. 2.3.4.2). Unfortunately, some authors use the two labels interchangeably.

In summary, a number of EDA parameters can be extracted, and their number may be increased through possible transformations. Suggestions for the choice of parameters in each context are given in Chapter 2.5 and the corresponding passages in Part 3. In most cases only a small range of parameters are used in applied EDA measurement; large ranges are only used in research.

2.4 External and internal influences

This chapter deals with factors that are possible sources of variance in EDA measurement. These factors include environmental conditions (Sect. 2.4.1) and direct influences from other physiological factors (Sect. 2.4.2), including age, gender, and racial differences (Sect. 2.4.3).

A problem that arises in the following two chapters as well as in Part 3 of this book is the variety of recording techniques used in the investigations. These are reported in footnotes, except for studies using the standard methodology as summarized in Section 2.2.7.[63] This will allow the readers to make their own judgements on the generalizability of results from the studies reported.

2.4.1 Climatic conditions

Investigations of the influence of climatic factors on EDA are mostly concerned with air temperature; measurements of other climatic factors (e.g., humidity) are sparce.

One feels comfortable in what is known as the thermoneutral zone, in which neither shivering nor evaporative heat loss through sweating occurs (Thews et al., 1985). The thermoneutral zone for or a sitting, lightly clad person is between 25 and 26^{0}C, if the wall and air temperatures are equal, with a relative humidity of 50%. The comfortable temperature for a person doing office work is 22^{0}C. Venables and Christie (1973) allowed a range of laboratory temperature between 20 and 30^{0}C, within which the vasomotor control of body temperature functions fully. However, they recommended not to use low temperatures with subjects taking part in psychophysiological experiments because these subjects are mostly inactive and cool more quickly than active subjects, causing a reduction in electrodermal reactivity (Sect. 2.4.1.1). Heat loss may be prevented by covering the subject with a light sheet even in summer.[64]

2.4.1.1 Ambient temperature

The investigations of EDA dependence upon ambient temperature can be divided into those in which the laboratory temperature is measured and those in which the outside temperature is measured. Influences of the ambient temperature on EDA were investigated by Venables and Christie (1980) in their Mauritian study with 640 subjects aged 5 to 25. Positive correlations between .20 and .40 were displayed between temperature and SCL, as well as negative correlations in the same range between temperature,

[63]Standard methodology means recording with sintered Ag/AgCl electrodes having .5 – 1 cm^2 contact area and filled with isotonic NaCl paste made from Unibase or a similar neutral ointment. The electrodes are fixed with double-sided adhesive collars to the skin, which is neither pretreated nor prewashed. Smaller deviations from this standard methodology will be mentioned in the text.

[64]This is understandable if one notes that Venables and Christie give 21^{0}C as the correct temperature for a European laboratory, while in the present author's experience this is somewhat too low. Instead, the author in his own laboratory maintains a constant temperature of 23 ^{0}C and 50% relative humidity.

on the one hand, and SCR amp., SCR lat., and SCR rec.t/2, on the other.[65] However, those connections appeared only in subjects older than five, and no such correlations were found in an investigation performed simultaneously with 1,800 three-year-old children.

Some older studies reviewed by Rutenfranz and Wenzel (1958) and Venables and Christie (1973) point to clear differences between EDA in summer and in winter months. Venables and Christie suggest that the cause of this yearly swing is a hormonal change in reaction to heat effects, as pituitary adrenocortical hormones influence both eccrine sweating and the electrolytic structure of epidermal tissue.

An interaction between seasonal and room temperature effects on EDA is shown in the results from Neumann (1968). In three experiments with 11 adults and 26 children (aged between 6 and 11), using 10 active sites on the hand and forearm, Neumann investigated the connection between log SRL and room temperatures of 18.3–40 ^{0}C at different times throughout the year. She found differing SRL patterns from the recording sites in summer and in winter, as well as during a heat wave, and the children showed less variation in their SRL patterns than the adults. Heating and cooling led only partly to the expected increases and decreases of the SRL, which differed according to the recording site and the season. The SCL was lowest when the temperature, independent of all other conditions, was 35^{0}C.

In a repeated-measures design with seven subjects, Conklin (1951) investigated the relationship between three different room temperatures (21.9, 26.9, and 29.5^{0}C) and the SCL measured at three different sites (wrist, forehead, and palm).[66] He found that the SCL decreased with temperature, and differences between the recording sites were not significant.

Rutenfranz and Wenzel (1958) also reported a clear connection between temperature increase and SRL decrease. They studied five subjects for using a treadmill in a climate chamber in which the relative humidity was 60–65 %, the wind speed was a constant .5 m/sec, and the air temperature and the radiation temperature of the walls varied simultaneously between 15 and 36^{0}C. Through their AC recording of EDA they found that the SZL increased with lowering temperature, while the skin capacitance decreased, interindividual differences being more pronounced at lower temperatures than at higher ones.

Significant correlations between the SCL measured with dry electrodes and the effective room temperature were also found by Venables (1955), however, only with a sample of neurotic subjects (N = 52), but not with the healthy controls (N = 210), and only with temperatures exceeding 20^{0}C. The direction of the correlations changed from –.48 during motor exercise to .51 during a resting period.

Summarizing three investigations, Wenger and Cullen (1962) reported correlation coefficients between the palmar log SCL and the room temperature of –.09 and –.15

[65]The authors reported that they had to raise the laboratory temperature to an unusually high 30^{0}C, since at lower temperatures the Mauritians displayed hardly any EDRs.

[66]This study was performed with electrodes that were pressed to the skin.

for male subjects and of .22 for female subjects. They took this as an indication of a gender-specific temperature effect. However, corresponding readings from the forearm gave the same positive correlations for male and female subjects between SCL and temperature.

Wilcott (1963) found an SRL decrease with an increase of room temperature up to 65.5^{0}C in 21 subjects. Additional variations in amplitude and shape of the SRRs he observed were not conclusive, as his stimulus conditions under different temperatures were not comparable. Grings (1974) reported an SRL decrease of 3%/^{0}C, a value which is also reported for skin temperature variations by Edelberg (1972a). While the SRR amp. can rise with decreasing temperature, it clearly falls when lower temperatures (e.g., 20^{0}C) are present over longer periods of time.

Temperature effects on the SPL differ according to type of electrode and electrolyte concentration (Grings, 1974). The positive and negative components of the SPR are influenced in different ways by room temperature. The positive SPR decreases together with the temperature, while the negative SPR component can be seen more clearly.

In an habituation experiment with 96 subjects, Fisher and Winkel (1979) found significant correlations between the outside temperature and the SCR amp. as well as the NS.SCR freq., but did not correct their data because they judged the influence of temperature as small. Waters, Koresko, Rossie, and Hackley (1979), who were more interested in the middle-term and long-term connections between EDA and meteorological variables (Sect. 2.4.1.2), showed a significant connection in 336 subjects only between temperature and SCL over long-term observations (one week or a month), and not with phasic EDA measures. In the study described in Section 2.2.6.1, Turpin et al. (1983) recorded SC in 12 subjects and room temperature during seven-hour work days, and determined at hourly intervals inter- and intraindividual correlations. They found a significant correlation of .61 between room temperature and SCR freq., and almost as high a correlation of .53 between temperature and SCL, but only as interindividual correlations. The corresponding between-subjects correlations were between .81 and –.50, and, on average, not significant. The authors traced this to differing climatic conditions on different days of the investigation.

Since there is an overall clear dependency of EDA upon the room temperature and the seasonal temperature, these variables should also be recorded, and the ambient temperature should be held as close as possible to a constant 23^{0}C. Keeping EDA investigations within seasonal limits is also definitely recommended.

2.4.1.2 Other environmental conditions

Venables and Martin (1967a), and Grings (1974) have reported negative correlations of both SCL and SCR to relative humidity. In the study from Venables (1955) mentioned in the previous section, negative correlations to the SCL were found when

relative humidity was between 54% and 66%. Higher and lower relative humidities produced positive correlations.[67]

Results from Wenger and Cullen's (1962) study point to gender-specific differences in the correlations between humidity and SCL (as with temperature; see previous section), which were in any case relatively small, as were the correlations ($r = -.11$ for male and $r = -.23$ for female subjects).

Fisher and Winkel (1979) could not determine any significant connection between EDA and humidity. Waters et al. (1979) found significant connections between humidity and SCL, in their short- and middle-term study, and between humidity and the square root of the SCR amp. as well as an habituation index, in the middle- and long-term study (details for both studies, see previous section).

Venables and Christie (1980) obtained some positive correlations between relative humidity and SC variables with the 5- to 20-year-olds (from $r = .20$ to $r = .40$) in their Mauritius study (Sect. 2.4.1.1). No such correlations appeared with the older adults. However, these correlations were found in different age groups in different variables (SCL, SCR amp., SCR lat., SCR rec.t/2). The same applies to correlations found in studies of the connection between air pressure and skin conductance parameters (cf. Venables & Christie, 1980, Table 1.7). All in all, air pressure does not appear to influence EDA significantly. Only Wenger and Cullen (1962) found significant correlations of .27 between air pressure and SCL for their male subjects, and Waters et al. (1979) found a corresponding effect in their long term-study. Fisher and Winkel (1979) could not confirm correlations between EDA and air pressure.

Waters et al. (1979) calculated multiple correlations between their EDA parameters and three meteorological variables: outside temperature, air humidity, and pressure. They concluded that EDA was influenced by these variables over short-, middle-, and long-term periods. With one-half of their subjects ($N = 169$) they obtained predictors of EDA parameters by means of meteorological variables, which positively correlated within the other half of the subjects in middle- and long-term observation throughout with the measured SCL scores and the square-root transformed SCR amp. scores. However, the percentage of explained variance was only 6–9%.

In summary, the influence of meteorological variables is difficult to demonstrate as the variables cannot be easily experimentally manipulated. If clear climatic changes are expected during the course of an experiment, meteorological variables should be recorded as controls.

2.4.2 Physiological variables

In the following sections, physiological variables that are intimately related to sweat gland activity and that may have an influence on EDA are described. These are physiological factors connected with thermoregulation, whose contribution to elicitation of

[67] The dry electrodes used in this study may have contributed to this inconsistency.

the EDA has already been discussed in Chapter 1.3. A systematic treatment of relationships between EDA and various other physiological measures is beyond the scope of this volume. Where it is of theoretical interest, the connections are reported in the appropriate passages of Part 3.[68]

EDA, like all vegetatively controlled processes, is subject to a circadian influence (Rutenfranz, 1955). For example, the relationship between EDA and temperature parameters may be obscured by circadian factors. Venables and Christie (1973) have shown that the daily courses of SC and body core temperature progress similarly, while the circadian rhythm of the finger temperature is a mirror image to that of the body core temperature. Using both DC and AC measurements over 24 and 29 days, Rutenfranz (1958) found two SC minima in two subjects, occurring at 10 a.m. and 7 p.m.

Infradian rhythms may also influence EDA, such as body temperature fluctuations associated with the menstrual cycle. With ovulation, the basal body temperature (measured in the morning under basal metabolic rate conditions) suddenly rises by about .5^{0}C and remains at this level until next menstruation as an effect of progesterone on temperature regulation (Thews et al., 1985). Hot flushes which appear as the most widely reported menopausal women symptom in Western culture may also be at least partly dependent on changes in the production of sex hormones and their action upon skin (Sect. 2.4.3.1).

2.4.2.1 Skin temperature and skin blood flow

EDA lat. as well as EDR ris.t. increase as skin temperature decreases (Maulsby & Edelberg, 1960), which can be explained by the temperature dependency of acetylcholine transport (Sect. 1.3.2.1). Venables and Christie (1980, Table 1.8) also found negative correlations between skin temperature and latency, rise time, and recovery parameters of SCRs in 260 eleven-year-old children. However, these correlations where small: from –.18 to –.30. While neither SCL nor SCR amp. was correlated with skin temperature in this study, Maulsby and Edelberg (1960) found a 3% increase of SRL for each degree decrease in finger temperature between 40 and 20^{0}C (see Edelberg, 1972a; and Sect. 2.4.1.1). In addition, the relationship between log SRL and skin temperature was found to be linear. With another sample of seven subjects, Maulsby and Edelberg found an inverse relationship between SRR amp. and skin temperature averaging 5 %/^{0}C as a result of abrupt temperature changes. The changes in electrodermal reactivity recovered within 2–8 min after the temperature change, and, thereafter, in some subjects actually displayed a reverse trend. While recommending a correction of

[68]One interesting result should be mentioned here: Christie and Venables (1971) found a correlation between the BSPL (Sect. 2.3.2.1) and the T-wave amplitude (TWA) in the EKG, with 21 male subjects lying down ($r = -.70$) as well as with 15 subjects in a sitting position ($r = -.61$). The authors suggest the extracellular potassium ion concentration as being the cause for both the TWA and the negativity of the BSPL. A more recent paper by Furedy and Heslegrave (1983) suggests that the TWA is an index of excitatory sympathetic activity. Since it is known that the EDA is a valid index of sympathetic excitation, a high correlation between TWA and EDA is expected.

the SRL according to skin temperature, Maulsby and Edelberg (1960) advise against a corresponding correction of SRR parameters due to the heterogeneity and instability of the effects found.

Lobstein and Cort (1978) investigated the effects of variation in skin temperature over the entire body on SCR. They enveloped each of their 14 subjects in a plastic suit, except for the face and one hand, and warmed the air inside. The average skin temperature taken at the six experimental sites increased from 26.0 to 37.1 ^{0}C. In each temperature condition, SCR parameters in response to three tones with signal characteristics were measured and averaged. They found a significant decrease of the SCR lat. with warming, but no influence on the SCR rec.t/2. The results, which were obtained under better-controlled conditions than those of Maulsby and Edelberg (1960), indicate that a correction of the EDR amp. in respect to the skin temperature is necessary even for small temperature variations.

The possible significance of skin temperature for SP measurement was also stressed by Venables and Sayer (1963). Due to the distance between recording sites (Sect. 2.2.1.1), the skin areas for the active and inactive electrodes may display different temperatures, leading, theoretically, to deviations of 1 mV per 5 ^{0}C in the range from 20 to 35^{0}C. However, the authors could not statistically demonstrate such a relation in an empirical study.

To date only a few systematic studies on the relationship between EDA and skin temperature exist, but clear increases of the SRL, the latency time, and possibly other temporal EDR parameters must be reckoned with when a decrease in skin temperature occurs.

Only a very small number of investigations of the influence of skin blood flow on EDA have been performed. Muthny (1984) summary of these investigations states that, while in a few cases, excessively high or low blood flow to specific areas affect endosomatic and exosomatic EDA, in most other investigations no dependence of EDA upon skin blood flow could be found. Thus, vasomotor activity is no longer regarded as a significant factor of influence for EDA today (Sect. 1.1.2). Possibly the sometimes-observed connection between the skin blood flow and EDA can be traced to the vasomotor activity and sweat gland activity in thermoregulation (Sect. 1.3.3.2 & 1.3.5). Although the connection between skin blood flow and electrodermal phenomena appears to be rather unsystematic, disturbances of the blood flow should be avoided in all cases; therefore, wrapping of the electrode and the fingers with adhesive tape in order to fasten the electrode should be avoided (Sect. 2.2.2.1).

2.4.2.2 Evaporative water loss and skin moisture

Though sweat gland activity is one of the most important factors in the origination of EDA (Sect. 1.4.2.3), there is no perfect correlation between EDA and sweating, so they must be treated as two separate biosignals.

Edelberg (1972, Table 9.2) summarized the results taken from eight older studies that measured sweat secretion as well as EDA using various methods. While as a rule between-subject correlations were below .50, within-subject correlations exceeded .85. However, in a study done by Edelberg (1964) with 12 subjects, intraindividual correlations between SRR and SCR amplitudes and skin vaporization as measured by resistance hygrometry (directing a stream of dry air over a skin area of 1 cm^2), showed median correlational values as low as .24 and .30.

The independence of EDA from sweat gland activity was also reported by Wilcott (1964), as a general result from three studies with a total of 26 subjects. During mental strain, the steam content of a dry nitrogen current led across the skin was recorded, together with alternating SP and SR readings. In addition, atropine (an anticholinergic agent) was applied iontophoretically to the skin of five subjects. The differences in recovery to this procedure, between both exo- and endosomatic EDA on one hand and skin evaporation on the other hand, indicated some independence of electrodermal and sweat gland activity measures.

A series of three studies of the covariation between EDA and sweat gland activity has been performed by Muthny (1984) with a total of 70 subjects. Blowing a dry air current of a constant 2 ml/sec across a 5 cm^2 palmar skin area, he measured steam release with an evaporimeter. From adjacent skin sites, SP and SC recordings were taken simultaneously. He examined habituation tasks and stress tasks such as cold pressor, arithmetic performance under noise, anticipation of giving a speech, and taking a blood sample. Also investigated were the direct effects of locally applied (either injected or iontophoretically applied) atropine as well as neostigmine (a parasympathomimetic agent, facilitating cholinergic transmission) on EDA and sweat gland activity, as measured by skin steam release. The latency of the phasic sweat gland activity was on average 1.1 sec longer than the SCR lat. This result demonstrates that no outpouring of sweat on the skin surface is necessary for eliciting an SCR (Sect. 1.4.2.3). Consistent with previous reports (Edelberg, 1972a), within-subject correlations were much higher (median $r = .88$) than those between subjects ($r = .23$ to $r = .64$), indicating marked differences of interindividual variances in both biosignals (Muthny, 1984).

Measuring sweat secretion by quantification of skin moisture is provided by gravimetric methods, where a humidity sensitive film is attached to the skin, or by colorimetric techniques (e.g., fingerprints). With the aid of a magnifying glass, the number of active (i.e., completely filled) sweat glands per skin area can be obtained with the latter method (Malmo, 1965). Thomas and Korr (1957), studying six subjects, found within-subject correlations ranging from .44 to .96 between sweat gland counts, which were recorded photographically, and the SCL.[69] Johnson and Landon (1965), in their study described in Section 2.4.3.3, used a technique developed by Sutarman and Thomson (1952), in which a plastic ink impression of the skin is transferred to Scotch tape

[69]EDA was measured as SRL with a 2.54 cm^2 dry silver disk electrode and transformed into SCLs.

and studied microscopically with a magnification of 25.[70] Their intraindividual correlations between SCL and the number of active glands were between .29 and .79, thus, not as high on average as those reported by Thomas and Korr (1957). Köhler, Vögele, and Weber (1989) used a fixation solution containing polyvinyl formaldehyde, removed with a Scotch tape strip from the finger and used to count the active sweat glands microscopically. With 20 subjects, they found a correlation between the occurrence of palmar sweating and the SCL change during a stress film of .71. All other correlations were not significant.

There is some evidence that tonic measures of skin moisture such as sweat gland counts show a sufficient relationship to tonic EDA measures. However, the phasic sweat gland activity cannot be reliably obtained with the simple method of measuring skin moisture. For this reason, refined methods to quantify evaporative water loss are necessary. These are very expensive, and only moderate correlations to EDR parameters are expected when simultaneously recorded from heated skin.

2.4.3 Demographic characteristics

From the demographically determined individual differences which can contribute to differing behavior of the electrodermal system, age and gender differences are the ones most carefully investigated (Sect. 2.4.3.1 & 2.4.3.2), while possible influences of racial and ethnic factors as well as heredity on EDA are rarely studied (Sect. 2.4.3.3).

2.4.3.1 Age differences

The first clear age-related changes in the skin in adult humans appear between the third and fourth decades of life. In the fourth decade there occurs a relatively sudden decrease in skin thickness and elasticity. Insensible perspiration first clearly decreases after the 60th year of life, presumably due to decreasing skin blood flow (Leveque, Corcuff, de Rigal, & Agache, 1984). With age, the binding of the epidermis and dermis (Sect. 1.2.1.2) loosens, the epidermis flattens, and the epidermal barrier function is reduced; decreases occur in the number of active eccrine sweat glands and the sweat quantity per gland, as well as the salt content of the sweat (Pollack, 1985). The epidermal ridges on the palmar and plantar surfaces of ridged skin (Sect. 1.2.2) are partly lost, and the basic surface for the epidermal mitosa in relationship to the horny cell layer decreases (Sect. 1.2.1.1), and more mitosa per cm^2 are necessary to compensate for the loss of keratinocytes (Steigleder, 1983). Morimoto (1978b) reported a decreased amount of sweating per gland with increased age for males but not for females, while Silver, Montagna, and Karacan (1965), found a decrease in the number of active glands as well as in the output per gland with aging for both genders. Millington and Wilkinson (1983) point to two possible changes in old age that may influence sweat gland

[70] EDA was measured as SR through Ag/AgCl sponge electrodes of 1 cm diameter with an "inert" electrolyte using 40 μA current and a Wheatstone bridge, being transformed to SC values.

activity; the deterioration of an intrinsic glandular condition, resulting in a limitation of responses to all kinds of stimuli, and an extrinsic factor affecting sensitivity to cholinergic stimulation in general.

Old age commonly brings a decrease of the SCL and an increase of the SRL, but the causes are not well understood. Edelberg (1972a) holds that the epidermal changes in aged skin are too small to explain the observed increase in skin resistance. However, considerable changes in relationships between EDA and related properties of skin appear during aging. With 12 young subjects of both genders (mean age 25.3 years), Catania, Thompson, Michaelewski, and Bowman (1980) found a correlation of .74 between the number of active sweat glands, as measured by means of fingerprints (Sect. 2.4.2.2), and the SCL during rest, recorded with standard methodology and Beckman paste from the preferred hand. By contrast, a group of 12 older subjects (mean age 69.5 years) showed a correlation of only .22.

To see the effects of varying corneal hydration, Garwood, Engel, and Quilter (1979) measured SC and SP in 12 young and 12 old subjects (mean ages 30.8 and 75.5 years) with KCl electrolytes with varying moisture. Varying corneal hydration did not affect the SCL in either the young or old subjects but did affect the SPL. While a monotonic relationship between SPL and hydration existed in the young subjects, so that the most negative SP appeared with the smallest hydration, old subjects displayed an increase of negativity of SP with increasing hydration. The authors attribute these results to an increase in the epidermal potential component and a decrease in sweat gland potential in old age as sequential to decay of the sweat gland ducts. In a further investigation by Garwood, Engel, and Kusterer (1981) with 25 young and 37 old male subjects, these findings were confirmed.

Surwillo (1969), recording SP in 58 young (23–53 years) and 64 old (54–85 years) males during 15 min of a time-keeping task, found that mean SPLs were in the same range in both groups, but the older males fit the normal distribution better than the young males. However, Surwillo (1965) reported a low but significant correlation of –.23 between age and SPL from the same set of data. A significant decrease of NS.SPR freq.[71] with age has been found by Surwillo and Quilter (1965) in 132 males between the ages of 22 and 85.

Investigations of changes of phasic EDA in old age have shown somewhat more confusing results. This may be partly due to pronounced age-related changes in the hypothalamus (Andrew & Winston-Sale, 1966), leading to additional differences in the central triggering of vegetative reactions which may interact with peripheral physiological differences of old as compared to young subjects (Edelberg, 1971). Botwinick and Kornetsky (1960), as well as Shmavonian, Yarmat, and Cohen (1965) and Shmavonian, Miller, and Cohen (1968), found a decrease of electrodermal reactivity during classical conditioning with elderly subjects. Zelinski, Walsh, and Thompson (1978) also observed a decrease of the SCR amp. in a group of very old subjects, in contrast

[71] Recorded with pure silver spiral electrodes chlorided electrolytically.

to young and old subjects, during a memory test. On the other hand, Furchtgott and Busemeyer (1979) found no differences in the change of SC[72] during mathematical exercises and memory tests with 67 male subjects (23–87 years) divided into four age groups. Eisdorfer (1978) summarized several studies performed by his group, reporting more electrodermal reactivity in older subjects when they were emotionally charged, but not during learning tasks or under relatively nonthreatening conditions. Garwood et al. (1979) also found no influence of age upon the SCR in various reaction time (RT) tasks in their above-mentioned investigation.

Plouffe and Stelmack (1984) compared electrodermal orienting responses to pictures of simple objects with either familiar or unfamiliar word names of 30 young (17–24 years) and 30 old (60–88 years) women. The young group showed a higher SCL during a baseline period as well as larger SCRs (recorded with standard methodology, however, using KY-gel) as compared with the old group. Furthermore, the older women displayed larger SCR amplitudes to items they recalled in a subsequent recognition memory test, particularly to recalled unfamiliar-named items. This result points to an unexplored differential sensitivity of the SCR to information processing (Sect. 3.1.4.1) in young and old people (i.e., being more CNS or more peripherally determined).

No differences between young and old age groups of the electrodermal orienting response on the first two of a series of 1 kHz tones, as well as in the trial-to-habituation criterion (Sect. 3.1.1.3), were found by Catania et. al (1980). These authors claim that the method of recording is the cause of the failure to obtain age differences in phasic EDA, since studies that used constant current (e.g., Botwinick & Kornetsky, 1960; Shmavonian et al., 1965, 1968) found differences, while those that used constant voltage (e.g., Eisdorfer, 1978; Catania et al., 1980) did not. This difference has been interpreted as being due to the sensitivity of the constant current method to the decrease of the number of active sweat glands (Sect. 2.6.2).

In addition, the appearance of differences in reactivity between young and old subjects may be dependent upon the experimental context and the character of the stimuli, such as their emotional meaningfulness. Silvermann, Cohen, and Shmavonian (1958) as well as Shmavonian and Busse (1963) showed that old subjects reacted with a clear increase of the EDR amp. to words that had specific emotional significance to them, in comparison to neutral stimuli. Baltissen (1983), using standard methodology, also found that his 20 male subjects, aged 65 to 75 years, showed no less electrodermal reactivity than 20 young male control subjects aged 25 to 35 years, to non-age-specific emotion-inducing material. In contrast, the elderly subjects showed even higher SRR amp. and NS.SPR freq. during presentation of pictures of children varying in emotional quality and intensity.

[72]Recorded as SR with 46 μA from palm vs. forearm with 1cm^2 Ag/AgCL electrodes held in place by an elastic band, using cellulose sponge holders soaked with saline as electrolyte.

Results concerning EDA in infants and children are somewhat inconsistent (Edelberg, 1972a), though an increased SRL was found in a study by Corah and Stern (1963), described in Section 2.5.2.2.2. Kaye (1964) also found a clear increase of the SCL[73] on palmar and plantar skin surfaces in 112 neonates during their first four days, and ascribed this result to their increasing sweat gland activity. Spontaneous and evoked EDRs have been observed in babies only a few days old (for further references see Edelberg, 1972a, p. 408).

Curzi-Dascalova, Pajot, and Dreyfus-Brisac (1973) investigated the appearance of spontaneous SPRs in 29 normal children during sleep. The sample consisted of both full-term and premature infants, their age being calculated as between 23 and 41 weeks from the estimated date of conception. SPRs[74] first appeared in the premature infants at the 28th week of life. After this time the triggering mechanism of phasic electrodermal activity appeared to be fully functional. The authors found an increased appearence of SPRs during REM sleep, while, in contrast to adults, the NS.EDR freq. was higher in slow-wave sleep than in REM sleep (Sect. 3.2.5), and the frequency of nonspecific EDRs during sleep was less in total for children than for adults.

Hot flushes – a symptom appearing frequently in menopause women – were shown by Swartzman, Edelberg and Kemmann (1990) being paralleled by marked increases in palmar and sternal SCL (2.05 and 1.34 μS, respectively). The correlation between sternal SC changes during flushes and subjective flush severity ratings was .592. Influences of hormonal changes on peripheral and central thermoregulatory processes may contribute to EDA being a suitable objective indicator of hot flushes as a prominent age-related symptom in females.

In summary, age-related physiological and psychological changes have to be considered as possible causes for both decreases of SCL as well as amp. in older subjects. In addition, the method of recording may interact with peripheral changes appearing in older subjects. The electrodermal behavior of infants and children is also different from that of adults. Characteristic values for EDA parameters for some age groups are reported in Sections 2.5.2.1 and 2.5.2.2.

2.4.3.2 Gender differences

Differences between women and men have been studied for both sweating and EDA. Women have a greater sweat gland density than men, but they display more delayed and, in total, less sweating (Morimoto, 1978b; Edelberg, 1971).

The observed gender-related differences in both sweating and EDA can presumably be ascribed to endocrine influences (Venables & Christie, 1973), that is, to women hav-

[73]Recorded with silver electrodes covered with an AgCl layer attached by means of flexible wires to palmar, plantar, and calf regions, with a constant voltage of 1.35 V.

[74]Unipolar measurements taken from the palm and the sole with Ag/AgCl electrodes of 7 mm diameter, filled with Beckman electrode paste and mixed with additional salt. The inactive electrode was attached to the dorsum of the respective hand and foot, and another one was attached on the forearm as control.

ing slightly but not significantly more active sweat glands but men showing a greater gland flow (Fowles, 1986a). The menstrual cycle may contribute to gender differences as well (Edelberg, 1972a). A number of studies have also found gender-based differences in electrodermal activity and reactivity. Kimmel and Kimmel (1965) obtained a significantly greater mean SCR amp.[75] with eight male subjects than with eight female subjects in reaction to the presentation of simple visual stimuli. Purohit (1966) showed that the SRR amp.[76] was significantly higher in 64 male subjects than in 64 female subjects in both an acquisition phase and an extinction phase of a light/tone conditioning paradigm.

In contrast, higher electrodermal activity has been determined for female subjects in the presence of threatening stimuli. Kopacz and Smith (1971) found a lowered SRL[77] and a heightened NS.SRR freq. among 30 female subjects in comparison to 30 male subjects during anticipation of an electric shock, where a faster rise of the NS.SRR freq. was especially noticeable among the female subjects in the first of several anticipation phases.

Ketterer and Smith (1977), using standard methodology, studied 32 female and 27 male subjects under different experimental conditions and found a significant interaction between gender and condition. The NS.SCR freq. was highest among the female subjects under verbal stimulation and under resting conditions, and highest among the male subjects with presentation of music. By contrast, Hare, Wood, Britain, and Frazelle (1971) found greater tonic EDA and greater electrodermal reactivity[78] in 25 male subjects than in 25 female subjects. In the initial 10 min resting phase, the male subjects displayed a significantly higher SCL and, following announcement of the stimuli presentation, a significantly greater number of NS.SCRs per min than the female subjects. In response to the 30 slides presented, including neutral, sexual, and forensic material, the men displayed a significantly higher SCR amp. at the beginning and, in total, a slower SCR recovery with the sexual material than the women.

An investigation by Neufeld and Davidson (1974) also yielded significantly higher averaged SCL maxima[79] for 30 male subjects than for 30 female subjects during presentation of accident and control slides. Maltzman, Gould, Barnett, Raskin, and Wolff (1979a) also found greater electrodermal reactivity among male subjects in comparison to females in two of their experimental studies involving 440 subjects of both genders. The studies included initial habituation with verbal stimuli as well as classical conditioning and extinction. Especially among subjects with smaller orienting reactions at

[75]Recorded by zinc-zinc sulphate saline electrodes from palmar sites, transformed to log values.

[76]Method not properly reported, presumably unipolar.

[77]With zinc electrodes and zinc sulphate electrode paste from palmar sites.

[78]Recorded as SR with standard methodology, using Beckman paste, and transformed to SC. Differences appeared in both SCL and NS.SCR freq.

[79]Maximum SCL reached during the presentation of 5 slides, recorded with KY-gel, the type of electrodes not being mentioned.

the beginning and toward the end of the conditioning and extinction phases was the SRR amp. more marked for the male subjects than for the female ones.

In contrast, Eisdorfer, Doerr, and Follette (1980), using a valsalva maneuver,[80] observed a significantly higher specific SCR, with standard methodology, among 20 female subjects (20–29 years old) in comparison to male subjects. By contrast, in the 40–49 and 65–75 age groups, no gender differences were displayed in reactivity. However, before starting the experiment, the SCLs of the male subjects of all the age groups were higher than those of the females.

Román, García-Sánchez, Martínez-Selva, Gómez-Amor, and Carrillo (1989), presenting a verbal and spatial performance task to 22 subjects of each gender, could not show gender differences in SCR amp. and NS.SCR freq.[81] after subjects were grouped according to their preferred side of reaction (Sect. 3.1.4.2). This invalidated previous findings of the same group when disregarding lateralization.

With respect to skin potential, gender-based differences have also been observed. Edelberg (1972a) reports opposing SP variations among male and female subjects in his laboratory studies. Gaviria, Coyne, and Thetford (1969) found marked differences between 20 male and 20 female subjects in the correlations between SPL and SRL scores (Sect. 2.6.1) just before presentation of acoustic (especially verbal) stimuli. The tonic endosomatic and exosomatic EDA of the male subjects did not significantly correlate, while those of the female subjects did, between –.48 and –.59. Corresponding gender-based differences did not appear in the correlations between the phasic measures. Possibly the differences between the correlations may partly stem from the greater variance of SPL and SRL scores among the female subjects than among the males.

In summary, in many cases female subjects display a higher tonic EDA, while male subjects tend to show a greater electrodermal reactivity under conditions of stimulation. However, this conclusion cannot be generalized with respect to clinical applications. In their studies (described in Sect. 3.4.1.3) Ward, Doerr, and Storrie (1983) and Ward and Doerr (1986) found significantly lower SCLs in depressed female patients than in depressed male patients, and therefore they applied different diagnostic criteria along gender lines. Also, smaller electrodermal lateralizational effects (Sect. 3.1.4.2) are observed in women than in men. Additional gender differences are observed in correlations between EDA and temperature (Sect. 2.4.1.1) as well as relative humidity (Sect. 2.4.1.2), and also with respect to age effects on EDA as described in the previous section.

2.4.3.3 Ethnic differences and heritability

Possible ethnic differences rarely appear in the literature on EDA. Despite the difference in the number of active sweat glands between dark- and light-skinned subjects

[80]Increasing thoracic pressure through pressing and swallowing following deep breathing.

[81]Recorded with 7 mm diameter Ag/AgCl electrodes, .068 molar NaCl paste, and two .2 V constant voltage amplifiers on both hands simultaneously.

(cf. Millington & Wilkinson, 1983, Table 3), which is presumably attributable to an increase in sweat gland density along a temperature gradient (Morimoto, 1978; Muthny, 1984), not much regard has been given to related differences in EDA. Thompson (1954, Table 3) gave norm values for Europeans and Africans for the regional distribution of sweat glands and outputs per gland, but unfortunately only for dorsal areas of hands and feet. Venables and Christie (1973) report that Japanese subjects have more eccrine sweat glands on their extremities than Europeans. Fowles (1986a) points to the difficulty of studying different ethnic groups under precisely the same conditions, and to the minimal differences in sweating between Caucasians, Blacks, and Japanese obtained in the few investigations fulfilling those requirements.[82] Consequently, Venables and Christie (1980) reported normative values for EDA without further consideration, though these were obtained from Mauritian subjects (Sect. 2.5.2.1).

Some studies compared EDA of Caucasian and black subjects living in North America under comparable environmental conditions. Johnson and Corah (1963) found reliably higher skin resistance base levels in blacks than in Caucasians, and Bernstein (1965) reported the same for skin impedance levels,[83] both in normal and schizophrenic subjects. An increased SRL in blacks was also confirmed by Fisher and Kotses (1973).[84] No differences in NS.SRR freq.[85] were seen during a resting phase, but subjects examined by experimenters with opposite ethnic affiliation showed a significantly higher NS.SRR freq. during the presentation of the first half of 14 white noise stimuli of 75 dB and 5 sec duration as compared to the second half, which the authors explained as novelty effects.

Using sweat gland fingerprints in addition to SR measurement in 31 black and 32 Caucasian males during resting, along with responses to 10 tones (1 kHz, 55 dB, 5 sec), Johnson and Landon (1965)[86] could not confirm the hypothesis that blacks have a lower SCL because of their smaller number of active sweat glands. They found that Caucasians were more reactive during the first 4 tones, which seems contradictory to common concepts of level dependency (Sect. 2.5.4.2). In addition, rank-order correlations between both variables were consistently smaller in Caucasians than in blacks. Sweat gland counts and SR measures[87] were also recorded in parallel by Juniper and Dykman

[82]It is sometimes said that Chineses do not have sweat glands. This erroneous statement may result from sweat gland activity being normally lower in Asiatic people, due to their sweat glands being smaller as compared to those of Caucasians.

[83]Recorded with stainless steel disc electrodes of 9.5 mm diameter, filled with so called Cambridge paste, by means of a tissue resistance monitor providing 8 Hz square wave and a constant current of 20 μA.

[84]For discussion of experimenter's ethnic group on subject's physiological reactions see Venables and Christie (1973).

[85]Measured palmar/dorsal at the dominant hand by Ag/AgCl electrodes and Beckman electrode paste, using "a constant direct current of 20 μV" (which should be presumably μA).

[86]Method of EDA measurement as used by the Johnson group, see Footnote 202 (Sect. 3.2.1.3.)

[87]Obtained with a so-called Fels Dermohmeter and Zn-electrodes from the palms, and in one group from the plantar arch.

(1967) from different clinical groups of both genders. They confirmed lower SRLs and lower sweat gland counts in blacks than in Caucasians for males and females. Using varying numbers of subjects (from 2 to 27 in each group), they also found an increase of SRL and a decrease of active sweat glands with increasing age (Sect. 2.4.3.1). In addition, they reported that black females aged 20 to 39 years had fewer active sweat glands than Caucasian women in the same age range.

Korol and Kane (1978) compared SRLs[88] during a rest period and SRRs to a 1 kHz 60 dB tone of 10 sec duration taken from 26 Caucasians, 25 blacks, and 25 Indians, the latter being anthropologically more Caucasian but in skin color more akin to Afro-Americans. They observed a significant correlation of –.34 between skin color as measured with a pigmentometer and the resting SRL in the total sample, the SRL of Indians being intermediate to that obtained in black and Caucasian subjects. However, they found no differences in SRRs. Their conclusion is that skin color may have a greater influence on SRL than ethnic affiliation itself, which had not been clear from the data of a previous investigation by Korol, Bergfeld, and McLaughlin, (1975) using a very similar procedure with 25 whites and 25 blacks.

Lieblich, Kugelmass, and Ben-Shakhar (1973) obtained SCLs[89] under baseline conditions from groups in Israel of differing ethnic and cultural origin and compared their data with those from other groups in previous studies. As a confirmation of other studies, Caucasians showed significantly higher SCLs than black subjects. In addition, Caucasian bedouins yielded the highest SCLs, while the SCLs of the black bedouins were lowest, interpreted by the authors as showing the importance of ethnic origin in influencing SC in subjects of the same cultural and geographical environment.[90]

Janes, Hesselbrock, and Stern (1978) investigated the influence of ethnic group as well as parental psychopathology on SP in a total of 206 black and Caucasian children with a mean age of 9.6 years (measured with standard methodology, however, with Beckman electrode paste). A factor analysis of 18 SP and two movement-artifact variables obtained from an habituation series to 10 cool-air stimuli and a conditioning series (20 trials with cool air as CS and warm air as UCS) yielded five factors. In none of these did children of schizophrenic, manic depressive, physically ill, or normal parents differ from each other. However, children of psychotics showed more movements during the experimental session than control children, showing genator artifact proneness (Sect. 2.2.5.2). Caucasian children showed significantly more nonspecific EDA as compared to Afro-Americans, but most ethnic differences remained nonsignificant. In an earlier study using the same methodology, Janes, Worland, and Stern (1976) also found an increased SP reactivity in 42 Caucasians as compared to 64 black children, while va-

[88]Measured with standard methodology, however, using Beckman electrode paste.

[89]Measured with standard methodology (using Beckman paste) as skin resistance (20 μA constant current), transformed to SC.

[90]These results are highly questionable, since the Caucasian bedouin sample subjects were partly gathered by the police and were being moved, both actions may raise SCL markedly.

somotor responsiveness as measured by fingerpulse volume was greater in black than in Caucasian children.

Fredrikson (1986) investigated 21 Caucasian and 15 black subjects of both genders during rest as well as during a stress task.[91] Resting SCLs as measured with standard methodology were greater in Caucasian than in black hypertensives. However, the difference was not significant in normotensive subjects and also disappeared during the stress task. NS.SCR freq. showed a very similar pattern. Cardiovascular activity showed an opposite pattern, yielding no ethnic differences under resting conditions and a greater increase in Caucasian as compared to black subjects under stress. SCR amp. to the stimuli were also higher in Caucasian than in black subjects.

Sternbach and Tursky (1965) studied ethnic differences in SPRs[92] to 29 repetitive electrical stimulations of 1 sec duration each applied to the left forearm of 15 housewifes from the following groups: Old Americans (whose parents and grandparents were born in the U.S.), Jewish, Italian, and Irish immigrants. The Old Americans showed a faster and more complete habituation of the SPR than all other groups, which had an attitudinal correlate in their more matter-of-fact orientation towards pain as verbalized in an interview.

Little data has been given for any possible genetic determination of EDA to date. Lobstein and Cort (1978, Table 4), in a small sample of fraternal and identical twins, found heightened correlations between the rating of genetic fitness and different EDA parameters. Raine and Venables (1984) reported in summary that the majority of existing studies show no genetic influence of the orienting response on the SCR amp. However, Lykken (1982) provided data for 63 pairs of monozygotic twins and 18 pairs of dizygotic twins being presented with 17 tones (110 dB, .5 sec). The mean SCR amp. over the first four trials yielded an intra-class correlation of .55 for monozygotic twins and – .13 for dizygotic twins. After applying a range correction, correlations increased to .65 for monozygotic and to .37 for dizygotic twins. Lykken (1982) believed these results were due to the removal of interaction effects between genetically determined ANS responses to the stimuli and peripheral factors, like density and reactivity of the sweat glands, by means of range correction (Sect. 2.3.3.4.2).

In sum, blacks tend to have higher SRLs or lower SCLs than Caucasians during resting conditions. This is presumably due to the decreasing number of active sweat glands with increasing darkness of skin. Additionally, differences in sweat electrolyte concentrations of blacks and Caucasians are hypothesized to have a role in SRL and SCL differences (Johnson & Landon, 1965). Caucasians are also more electrodermally reactive during presentation of tones or noises. Thus, ethnic differences have to be carefully controlled for in EDA studies. Hereditary influences on EDA have not been adequately investigated up to now to estimate their importance for electrodermal recording.

[91]Presentation of 16 combinations of a tone (1 kHz, 68 dB, 35 sec) with a 110 dB white noise of .5 sec duration, the length of which could be shortened by pressing a button.

[92]Measured by Ag/AgCl sponge electrodes from the right palm.

2.5 Statistical properties

This chapter provides characteristic values of EDA parameters of endosomatic measurements (Sect. 2.5.1), of exosomatic recording with direct current (Sect. 2.5.2) – where SC and SR measures are treated separately – and exosomatic recording with alternating current (Sect. 2.5.3). In addition, Section 2.5.4 presents the level dependence of the different EDA parameters.

In trying to cope with the vast number of publications on EDA (Sect. 1.1.3), only studies that were methodologically oriented are included. Only in cases where such studies or appropriate review articles were not available are results from other studies reported. The present author prefers reporting results within the body of the text rather than providing tables because of the wide variety of methods used to obtain those results (see the introductory remarks of Chapter 2.4). In addition to means and standard deviations, reliabilities for, and intercorrelations between, various EDA parameters are provided.[93] The aspects of validity of electrodermal recordings are treated in Part 3.

2.5.1 Characteristics of endosomatic measurements

Skin potential measurement poses problems for both recording and evaluation (Sect. 2.6.1). Therefore, only a relatively small number of studies have made use of endosomatic methods, especially among those that have extracted phasic parameters (Sect. 2.5.1.1). In a number of studies, connections between endosomatic and exosomatic EDA parameters have been investigated, and these are reported in Section 2.5.1.3.

2.5.1.1 Skin potential reactions

The problem of evaluating endosomatic reactions has already been discussed in Section 2.3.1.2.1.[94] Since the SPR may not only be monophasic, but also biphasic or triphasic, a measure of the total amplitude is always questionable. The range of observed SPRs, yielding usually only a few mV, is between .1 and – 20 mV (Venables & Christie, 1980). Relationships between the stimulus strength and the size of the SPR are hardly predictable, since even a markedly uniphasic reaction can be altered and thereby weakened by a latent polarity which counteracts the reaction. Disregarding this, several studies used the measure of the difference between the negative and positive SPR maxima, for example, Gaviria et al. (1989) in their correlation study (Sect. 2.5.1.3).

The separate evaluation of single components of the SPR also poses problems for parametrization. Thetford, Klemme, and Spohn (1968) determined both the negative amplitude, measured from the prestimulus level to the negative maximum, and the positive amplitude, which was calculated either, in the case of a single positive SPR, from

[93] A review of reliabilities of different EDA parameters, including various investigations, is given by Freixa i Baqué (1982).

[94] Since phasic SP measures are so dependent upon experimental as well as recording conditions, reporting typical distribution parameters and reliabilities will not be discussed.

the prestimulus level or, in the case of a biphasic SPR, from the preceding negative maximum. In addition, they calculated the number of biphasic reactions. While a marked habituation was displayed with the positive SPRs, an inconsistent process appeared over the 20 trials with the negative SPRs.

Using 30 subjects, Knezevic and Bajada (1985) calculated the average amplitude of the biphasic SPR from the negative to the positive maximum, following electrical stimulation of the median nerve in the wrist.[95] The mean palmar measured SPR amp. was 479 μV, with SD = 105 μV, while the mean for the plantar surface was 101 μV, with SD = 40 μV. The palmar mean latency time was 1.52 sec, with SD = .13 sec. The plantar mean latency time was 2.07 sec, with SD = .16 sec. Here it should be noted that, as expected, the latency times of the first SPR waves are 300 msec shorter on average than the SCR latencies (Sect. 2.3.1.1).

The expected reductions of both the negative and the positive SPR amp. caused by an increasing hydration of the recording sites were quantified by Fowles and Rosenberry (1973), using different sites on 12 subjects. In the beginning, the negative SPR amp. at the hydrated sites was around 14 mV less than that at the nonhydrated sites; after 20 min the difference decreased to 8 mV. The positive SPR amp. almost fully disappeared at the hydrated sites.

Francini, Zoppi, Maresca, and Procacci (1979) measured SPL and SPR during repeated electrical stimulation in 32 subjects of both genders. At the beginning of the procedure, the SPL was positive and monophasic. During the procedure, the SPL became more negative, and the SPR adopted a biphasic-negative shape with an increasing negative and decreasing positive component. This result again shows that SPR amp. evaluation is complicated by level dependence.

2.5.1.2 Skin potential levels

The SPL can lie between 10 mV and –70 mV[96] (Venables & Christie, 1980); thus, the skin surface normally displays a negative potential in contrast to the body core, being greatest on the palmar and plantar surfaces. The average transcutaneous potential on the palms is –39.9 mV, and the corresponding value for the forearm is only –15.2 mV (Edelberg, 1971). The potential on the right hand has been found to be around 5–7 mV more negative than the one on the left hand, and this is true for both right- and left-handed people (Sect. 3.1.3.4). Edelberg (1971) suggested a connection between this difference and the higher conductance of the right hand. Positive SPL values represent an exception (Venables & Christie, 1980). Like the SPR, the SPL is dependent upon the degree of hydration. Fowles and Rosenberry (1973) found, in their study discussed in the previous section, a decrease of the SPL of around 25–30 mV through hydration of the recording sites.

[95] They used tin electrodes with a contact surface area of .72 cm^2, probably without electrolytes.

[96] The method of determining the BSPL as the minimal obtainable SPL was discussed in Section 2.3.2.1.

Shapiro and Leiderman (1954) investigated connections and distributional characteristics of various SP measures, which they obtained for two resting pauses and one simple reaction task with 53 student nurses as subjects. They found that the average SPL, lying between 0 and –55 mV, was approximately normally distributed, while the variance and the average quadratic successive differences (a time series statistic) displayed a positive skew. The mean SPL correlated at the same numerical value with both other measures (r= .32), and the correlation of the variance with the time sequence statistics was .78. The rank correlations between the two resting pauses, being 1 min apart, pointed to differing reliabilities of each measure used (r= .71 for the mean SPL; r= .47 for the SPL variance; and r= .63 for the time sequence statistics). Surwillo (1969), in his study described in Section 2.4.3.1, found palmar SPLs between –12.3 and – 56.8 mV during attentive conditions, the mean SPLs being normally distributed solely in the old-age group.

With 17 subjects of each gender, Foulds and Barker (1983) determined the SPL on numerous sites around the entire body in contrast to a reference electrode in electrical contact with the forearm dermis.[97] They found a mean SPL of –23 mV, with SD = 9 mV. Significantly higher negativity appeared on the palmar and plantar surfaces, and relationships to dermatomes were not displayed (Table 3, Sect. 1.3.2.1).

Use of the NS.SPR freq. as a tonic parameter (Sect. 2.3.2.2) can lead to ambiguous results because of the multiphasic structure of the SPLs (see previous section). However, Fowles et al. (1981) were convinced that a particularly sensitive parameter is in question here (Sect. 2.6.1). In their study described in Section 3.3.2.2, Crider and Lunn (1971) found a mean of 6.36 and SD = 5.42 for the NS.SPR freq. during 4 min of 72 dB white noise, with a reliability of .70 after seven days.

In summary, tonic SP measures, as well as the phasic SP measures described in the previous section, are dependent to a considerable extent upon recording and other environmental conditions. Both the SPL and the NS.SPR freq. also show relatively small reliabilities.

2.5.1.3 Relationships between endosomatic and exosomatic measurements

Burstein, Fenz, Bergeron, and Epstein (1965) investigated SR and SP measures concurrently during a word association test and found that the correlations between the amplitude of the SP c-wave (Fig. 35, Sect. 2.3.1.2.1) and the SRR increased from .63 to .79 together with stimulus intensity. The a-wave yielded a significant correlation to the SRR only with stimuli of high emotional importance (r= .62).

Lykken, Miller, and Strahan (1968) studied simultaneous SC and SP measurements taken from 19 subjects during a stress period and a following rest period, with differing combinations of two active and two inactivated (by skin drilling) palmar sites on the fingers. The mean intraindividual correlations between the SCR amp. and SPR amp. lay between –.18 and .96, with an average of .69. The largest connections appeared if

[97] They used liquid electrolytes (KCl/agar) and calomel (mercury chloride) electrodes.

the observed SPR could be regarded as the result of a superposition of an a-wave on a b-wave; whereby the positive b-wave, which reduces the SPR, first appears with high arousal (i.e., with a high SCL).

Wilcott (1958) measured SPR and SRR amp. alternatingly using the same sites (palmar against forearm) from 25 subjects performing word association and mental arithmetic tests. He found significant correlations between the SPR and the SRR amplitudes. The interindividual correlations were higher (r= .75 to .97, average r= .90) for monophasic, negative, and positive SPRs than for biphasic SPRs (r= .51 to .95, average r= .62).

Gaviria et al. (1969) studied simultaneous SP and SR measurements from 20 male and 20 female subjects during two sessions with a 2–9 day interval, each with five different acoustic stimuli, and found very high intraindividual correlations between the amplitudes of the SRR and those of the SPR.[98] The SPR amplitudes were obtained as the differences between the positions of the negative and positive maxima, an evaluation which is controversial (Sect. 2.5.1.1).

Venables and Sayer (1963) reported results from two studies in which SP and SR were taken in parallel from 93 schizophrenic subjects (Sect. 3.4.2). A curvilinear relationship between the SPL and the SRL was displayed. If the SRL scores were transformed into SCL scores, a linear relationship of these transformed scores to the SPL scores resulted, together with correlations between these two measures of .60 and .51.

2.5.2 Characteristics of exosomatic DC measurements

Exosomatic direct voltage measurements are performed with either constant voltage or constant current methods (Sect. 2.2.3.2), resulting in different units of measurement (i.e., conductance or resistance units). Many authors transform values of resistance into values of conductance before statistical data processing (Sect. 2.3.3.2). The majority of published results obtained with exosomatic DC measurements are expressed in terms of conductance. As conductance values transformed from resistance values obtained through the constant current method are equivalent to conductance values obtained through the constant voltage method (Sect. 2.6.2), values of skin conductance obtained with either method are therefore given in Section 2.5.2.1. In Section 2.5.2.2, the untransformed results of skin resistance measurements are given. The results for the form parameters are displayed together, specifically with respect to the latencies and the ascent parameters in Section 2.5.2.3, and with respect to the recovery times in Section 2.5.2.4. In Section 2.5.2.5, the mutual independence of electrodermal recovery and amplitude is discussed.

[98] They used the earlobe as an inactive site for the SP measurements, and dry silver electrodes with 3.8 cm^2 surfaces for the SR measurements.

2.5.2.1 Results of skin conductance measurements

Venables and Christie (1980) summarize the most important aspects of the distributional characteristics of skin conductance values. The authors note especially, however, that both the SCL and the SCR amp. can vary with the concentration of electrolytes and with the size of electrodes.

2.5.2.1.1 Skin conductance reactions. Venables and Christie (1980) report that the maxima of the SCR amp. are between 2 and 3 μS if standard methodology is used (Sect. 2.2.7). If these values are logarithmized, maximum SCRs lie between .30 and .47 log μS. Corresponding values for minima cannot be given, because they are dependent upon the amplification and the definition of the amplitude criterion (Sect. 2.3.1.2.3).

Venables and Christie (1980, Table 1.1) report the distributional data from a Mauritian sample (N = 539) divided into five age groups (5 – 25 years in steps of five).[99] The mean of the SCR amp. was .518 μS; the values climbed from .430 to .668 μS with increasing age of the subjects. The standard deviation was .576 μS and increased with age from .475 to .734 μS. Distributions of the amplitudes were significantly positively skewed and leptokurtic as compared to the normal distribution. Through logarithmization of the SCR amp., the distributions could be normalized. After normalization the total mean was –.496 log μS, the variance .200 log μS. Since the logarithmization had been performed with the SCR amp. values, no differences appeared between logarithmized SCL values. Corresponding improvement of the distributional characteristics through log transformation was obtained with a sample of 1,761 three-year-old children (Venables & Christie, 1980, Table 1.3). In a further sample of 65 subjects between 18 and 75 years of age, the distribution of the raw scores of the SCR amp. was similarly positively skewed, but markedly less leptokurtic, while both kurtosis and skewness deviated significantly from the normal distribution. This could possibly be due to the low intensity of tones at only 75 dB, while earlier reported results were obtained with tones of 90 dB (Venables & Christie, 1980, Table 1.4).

Fahrenberg, Foerster, Schneider, Müller, and Myrtek (1984) measured SCRs with 58 subjects during active and resting conditions in the laboratory, using standard methodology. They found an average SCR amp. between .46 and .89 μS with a standard deviation between .30 and .70 μS. The values, with the exception of one of the mental arithmetic exercises, were significantly positive skewed and leptokurtic. The short-term reliabilities were high only during activity (r= .72), while in the resting condition values less than r= .20 were found.[100]

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[99] Venables's team used KCl based electrode cream. Although sweat contains by far more NaCl than KCl, the difference between those monovalent ions is given little importance in the literature on methodology (Sect. 2.2.2.5).

[100] Iacono, Lykken, Haroian, Peloquin, Valentine, and Tuason (1984a), in their study described in Section 3.4.1.3, found a one-year retest reliability of the maximum SCR amp. during series of tones of r= .68 in 23 normal subjects.

Much higher average SCR amp. were found in an additional evaluation of data in one of the studies performed by the present author (Boucsein & Hoffmann, 1979), in which the SC and the SR on the middle phalanges of the left hand were measured in parallel using standard methodology. Thirty stimuli were presented, consisting of 2 sec each of white noise between 60 and 110 dB. The grand mean of all subjects and stimuli for the SCR amp. was 1.152 μS with SD= 1.021 μS. The distribution was significantly positively skewed and leptokurtic in comparison to the normal distribution. Following a log transformation which eliminated positive skewness, the mean SCR amp. was 1.033 log μS with SD= .535 log μS. The reliability, estimated according to Hoyt (1941), was .971 for the raw scores.

In total, it is expected that the SCR amp. will be clearly positively skewed and leptokurtic, so log transformations are recommended. The reliabilities are high in arousal conditions and low in rest conditions.

2.5.2.1.2 Tonic skin conductance measures. In addition to the SCL scores, the frequencies of the nonspecific EDRs are used as tonic measures (Sect. 2.3.2.2). In a summary of the different studies in the respective literature, Venables and Christie (1980) came to the conclusion that these measures cannot be simply regarded as interchangeable parameters of tonic EDA. Silverman, Cohen, and Shmavonian (1959) showed in their study discussed in Section 3.2.1.1 that the number of NS.SCRs can increase due to a decrease of SRL. Kimmel and Hill (1961) found that while the SCL could be used as a stress indicator, the NS.SCR freq. could not. Katkin (1965), as well as Miller and Shmavonian (1965), also found that both measures diverge as indicators of tonic arousal. Martin and Rust (1976) found only narrow correlations between tonic measures for interindividual (r= .27) and for pooled intraindividual (r= .15) correlations. However, Fahrenberg and Foerster (1982) found, in their study described below, markedly higher coefficients between the SCL and the NS.SCR freq., which were .55 for interindividual and .50 for pooled intraindividual correlations.

Minima and maxima of the SCL can hardly be reported, as they are dependent upon the electrode size. Venables and Christie (1980) have argued against a weighting of the SCL to the electrode area (Sect. 2.3.3.1) as they found a nonlinear relationship between conductance and electrode area. For recordings with two active electrodes, they report a SCL range of 1–40 μS, and of 0–1.6 log μS.

In the table from Venables and Christie (1980) already cited in the previous section, the corresponding values for the SCLs and the log SCLs are given.[101] There, the average SCL of the total Mauritian sample was found to be 3.040 μS, whereby the aging process was not linear; the mean for the five-year-olds was 3.597 μS, while the values from the ten-year-olds to the twenty-five-year-olds climbed from 2.613 to 3.223 μS. Corresponding differences were also found in the standard deviations: 2.467 for

[101] Since the SCL scores are also dependent upon the type and concentration of the electrolytes, the distribution data from the study of Venables's team cannot easily be generalized, as they used a KCl paste that is no longer used for SC measurements.

the five-year-olds and thereafter a rise from 1.901 to 2.539 μS with an average SD = 2.238 μS. The distributions of the SCL scores were only slightly positively skewed but significantly more leptokurtic than the normal distribution, while not as pronounced as the SCR amp. scores. It was also possible to eliminate the deviation from the normal distribution to a large extent through a log transformation. The results were based upon measurements from 635 subjects. The SCL mean (2.383) of the three-year-olds (N = 1,145) was markedly lower than that of the five-year-olds of the Mauritian sample; the standard deviation was 1.564 μS. In respect to skewness and kurtosis, the distribution was more or less the same as the Mauritian sample, but a log transformation was not fully able to eliminate the leptokurtic property of the distribution. In the sample of 18- to 75-year-old adults (N = 45), the average SCL was 3.612 μS with SD = 2.470 μS. The distribution was not skewed but significantly more leptokurtic than the normal distribution, which was eliminated through log transformation.

In the study by Fahrenberg et al. (1984), mentioned in the previous section, average SCLs of between 9.1 and 16.58 μS were found, and the standard deviation lay between 8.88 and 13.60 μS. The distribution deviated significantly from the normal distribution in skewness and kurtosis.

Walschburger (1976) found reliability coefficients of .95 and .98 for SCL over differing rest pauses of a laboratory experiment determined with standard methodology from 67 subjects. This extreme stability was traceable to the very small intraindividual variations in comparison to the interindividual variance. Fahrenberg and Foerster (1982) also report a short-term stability of .96 for 125 subjects, using standard methodology for the SCL measurement.

Jones and Ayres (1966) determined the SCL of 15 former addicts during therapeutic sessions over five weeks. Each session lasted 25 min, began with an injection of a placebo, and contained 12–15 electrical stimuli. The reliabilities in the first 3 weeks lay between .81 and .94, but decreased thereafter (1–5 weeks) to .60. Iacono et al. (1984a) found a one-year retest reliability of .66 for the mean SCL in 23 subjects (see Footnote 100).

Boucsein and Hoffmann's study, described in the previous section, reported that the mean of the SCL before application of stimuli was 8.263 μS with SD = 4.646 μS, with the distribution being significantly positive skewed and leptokurtic. Following the log transformation, which only eliminated the positive skewness, the mean SCL was 2.139 log μS with SD = .214 log μS. The reliability of the raw data, calculated as per Hoyt (1941), was .998.

Hardly any normative data have been published to date for the NS.SCR freq. Fahrenberg et al. (1983) found, in the above-mentioned study of 58 subjects under resting conditions, mean values between 3.0 and 3.5 SCRs per min with standard deviations of between 4.0 and 5.0 SCRs per min. Under activity conditions, they found mean values of between 13 and 13.5 SCRs per min with standard deviations of between 5.0 and 5.5 SCRs per min. Under resting conditions the distribution was significantly positively skewed and leptokurtic, while under activity conditions the normal distribution hypoth-

esis could be retained. Walschburger (1976) gives values for stability coefficients of .80 to .90 for the NS.SCR freq., determined in his above-mentioned experiment with 67 subjects in different resting phases. Fahrenberg and Foerster (1982) determined a short-term stability of .81 for the NS.SCR freq., and Iacono et al. (1984a) yielded a one-year retest reliability of .62.

The number of observed nonspecific EDRs is dependent upon the amplification factor and the amplitude criterion used (Sect. 2.5.1.1). In this author's experience, they can lie between zero and 10 SCRs per min in periods of relative quiet, while in activated periods values of around 20 SCRs per min are not unusual. However, in this case, overlying reactions frequently appear, and the number of SCRs will depend upon additional criteria of the evaluation (Sect. 2.3.1.2.2).

In summary SCL scores, compared to the normal distribution, tend to be rather positively skewed and leptokurtic, which can be eliminated through log transformations. Their reliability is very high but markedly decreases over periods of several weeks between measurements. No safe assertions for the NS.SCR freq. values can yet be made, as not enough data have been presented. The intercorrelations of both tonic SC parameters are middling high to low.

2.5.2.2 Results of skin resistance measurements

Today, skin conductance measurements predominate, and since many authors who use the constant current method transform their results into conductance units (Sect. 2.3.3.2), relatively little data exist for resistance values. When comparing results from different studies, it must be noted that both the SRL and the SRR amp. can vary, depending on electrode size (Sect. 2.2.3.2).

2.5.2.2.1 Skin resistance reactions. Venables and Christie (1980) do not report any statistics for SRR data as they chose to use skin conductance measures in general. Instead, in their Table 1.5 they give only a typical range for the SRR amp., which is .1–16.6 kΩ, with an assumed SRL of 100 kΩ, and a range from .02 to 4.54 kΩ with a base resistance of 50 kΩ.

Kaelbling, King, Achenbach, Branson, and Pasamanick (1960) recorded the SRR values from 12 subjects in response to acoustic, electric, and verbal stimuli, and obtained mean values between 3.0 and 16.3 kΩ with ranges up to 76.9 kΩ. The reliability was .76 after two days. Bull and Gale (1973, Table 1) presented 1 kHz tones of 90 dB to 12 subjects and measured SRR values at four time intervals.[102] They found values for SRR amp. of between 1.5 and 33.5 kΩ for the first trial and between 0 and 11.5 kΩ for the fourth trial. The reliability, calculated as an intraclass correlation, was, however, not significant (r= .42).

[102]Three weeks between the 1st and 2nd measurements and between the 3rd and 4th measurements, and 6 weeks between the 2nd and 3rd measurements.

Unpublished data taken from a study of Boucsein and Hoffmann (1979) with 60 subjects (Sect. 2.5.2.1.1) yielded norm values of 21.01 kΩ for the mean SRR amp. With SD = 24.30 kΩ, the distribution deviated significantly in positive skewness and kurtosis from normality. After log transformation with a resulting mean of 1.057 log kΩ and SD = .522 log kΩ, the distribution was no longer skewed but was slightly leptokurtic. The reliability of the raw data, estimated according to Hoyt's (1941) formula, was .975.

In summary, as with the SCR amp., a positively skewed and leptokurtic distribution is to be expected with the SRR amp., and log transformation can lead to improvement of skewness. The reliabilities are high for short-term periods but markedly decrease with time intervals, in the range of weeks.

2.5.2.2.2 Tonic skin resistance measures. As with the phasic values, Venables and Christie (1980, Table 1.5) give a possible range for SRLs of between 25 and 1,000 kΩ as being equivalent to SCLs from 40 down to 1 μS. Edelberg (1967) reports values of between 10 and 500 k$\Omega \times cm^2$ for the specific resistances (Sect. 2.3.3.1).

An evaluation of the unpublished SCL data from Boucsein and Hoffmann's (1979) study with 60 subjects (Sect. 2.5.2.1.1) resulted in mean SRLs of 167.2 kΩ, with SD = 74.88 kΩ. Following log transformation, the mean was 2.174 log kΩ, with SD = .205 log kΩ. Both the raw scores and the transformed values significantly deviated in kurtosis but not in skewness from the normal distribution. The reliability of the raw data, calculated according to Hoyt's (1941) formula, was .997.

Retest reliabilities reported for SRLs were, on the other hand, much lower. Wieland and Mefferd (1970) recorded the SRL from three subjects during two resting periods within a stimulus series over 120 days and obtained high reliabilities of the intraindividual differences, between r= .95 and .97. However, Galbrecht, Dykman, Reese, and Suzuki (1965), in 20 subjects, found concordance coefficients of only .67 for SRLs measured a day apart under stimulation by 60 dB tones.

Arena, Blanchard, Andrasik, Cotch, and Myers (1983) reported even lower reliabilities in an investigation with 15 subjects, where the SRL was measured with palmar and dorsal electrodes under different resting and stress conditions. The measurements were made on the 1st, 2nd, 8th and 28th day. Only the correlations between the 8th and the 28th day (average r= .72 for the resting conditions, and r= .453 to r= .556 for the stress conditions) and the 2nd and 8th day (r= .482 for resting conditions) were significantly different from zero. All other reliability coefficients were nonsignificant, which led to the conclusion of the authors that the SRL is an unreliable measure.

The frequencies of NS.SRRs, as another tonic measure (Sect. 2.3.2.2), could on average outnumber those of the NS.SCRs. This is because less amplification is necessary when using the constant current method than when using the constant voltage method (Sect. 2.1.1). EDRs are therefore more easily discovered, and the amplitude criterion is able to be set correspondingly lower (Sect. 2.3.1.2.3).

O'Gorman and Horneman (1979), in their study with 48 subjects described in Section 2.3.2.2, investigated the stability of measures for "small" and "large" NS.EDRs,

whose number was determined under three experimental conditions two weeks apart.[103] The number of "large" NS.EDRs significantly decreased after two weeks, while a corresponding increase was observable with the "small" NS.EDRs. Unfortunately, reliability coefficients were not calculated.

Docter and Friedman (1966) investigated the reliability of NS.SRRs[104] under 80 dB white noise, using 23 subjects. The average reliabilities computed from several single coefficients were .54 after 5 days, and .30 after 30 days. The intercorrelation of both tonic measures, the SRL and the NS.SRRs, was in the direction predicted ($r = -.34$), though not significant. On different days, the medians of the NS.SRRs were between 10 and 15 within a 15 min measurement period, while the scores ranged from 0 to 90.

Using a total of 24 seven- to eight-year-old children, Corah and Stern (1963) found, during a 2-min resting period, average SRL values between 194.1 and 275.3 kΩ, with standard deviations between 74.8 and 97.4 kΩ. Mean NS.SRRs per min were between 7.3 and 13.8, with standard deviations between 5.0 and 7.3. The intercorrelation of both tonic measures was between –.33 and –.64. The average reliability for measurements a day apart was .86 for the SRL and .61 for the NS.SRR freq.

With 48 pilots, Johnson (1963) obtained a reliability of the NS.SRR freq. measured a day apart of $r = .69$. Hustmyer and Burdick (1965) determined a reliability of .75 for the NS.SRR freq. measured during 15 min resting periods from 14 subjects 2–4 months apart. Bull and Gale (1973) reported, in their study mentioned in Section 2.5.2.2.1, an intraclass reliability of .91 for the NS.SRR freq. taken four times. The reliability of the mean amplitude of the NS.SRRs was .75.

In summary, it can be seen that the SCL values are strongly positively skewed and more leptokurtic in comparison to the normal distribution, while log transformation may at least improve the skewness. The reliabilities for short periods are fairly high but, as expected, decrease over intervals of several weeks. In contrast to the NS.SCR freq., enough data exist for the reliability of the NS.SRR freq. to say that while it appears rather lower than that of the SRL values over short periods, it is still comparably high over long periods of weeks to months. While the intercorrelations of both tonic SR measures are also relatively low, they are somewhat higher than those of the corresponding tonic SC measures (Sect. 2.5.2.1.2).

2.5.2.3 Latency and rise time parameters

A range for the latency times cannot be given since they are based on a time window which is limited a priori (Sect. 2.3.1.1). In seven subjects, Maulsby and Edelberg (1960) found an SRR lat. (evoked by sneezing) having a mean of 1.5 sec with a room temperature of 30 °C, rising to 4 sec when room temperature was lowered by 5–10 °C.

[103] The authors used standard methodology, with electrodes 12 mm in diameter, but transformed the SR scores into SC units before calculation of nonspecific responses.

[104] In this study, an unusually high current of 70 μA was used.

Edelberg (1972a, p. 370) points out that the latency is dependent upon both the temperature (Sect. 2.4.2.1) and the recording site.

Venables and Christie (1980, Table 1.2) give distributional data for the latency times of SCRs in response to 4 sec white noise of 90 dB with 559 subjects in their Mauritian sample. The lowest mean (1.472 sec) was among the 5-year-olds, and the highest was among the 15-year-olds (1.822 sec). Standard deviations were lowest with the 5-year-olds (SD = .373 sec), while the largest ones appeared among the 10-year-olds (SD= .418 sec). The deviations from the normal distribution were small in comparison to those of the SCL scores and the SCR amp. Hence, the benefit from log transformation is correspondingly lowered.

In addition, Venables and Christie (1980) calculated reciprocal values for the latency times, in addition to other temporal measures of the EDR, since these had the extra advantage of being proportional to the reaction speed. Here, as with log transformation, small improvements of distributions toward normality were made (Sect. 2.3.3.3).

Under the same stimuli as in their above-mentioned Mauritian study, Venables and Christie (1980) found that latency times for 1,161 three-year-olds were on average 1.488 sec, with SD = .714 sec, and were significantly positively skewed as well as leptokurtic in appearance. Among 45 adults between 18 and 75 years of age the latencies of the SCRs in response to 75 dB tones of 1 kHz and 1 sec duration yielded a mean of 1.896 sec and SD = .349 sec, with nonsignificant deviation from the normal distribution.

With 18 subjects, Rachman (1960) found average latency times of 2.94 sec, with SD = .71 sec, when evaluating EDRs in response to 35 loud buzzer tones of 2 sec duration each. The retest reliability over 6–8 weeks was .96. Lockhart (1972) found, in a sample made up of five experiments with 129 students, a mean latency time of 2.11 sec with SD = .56 sec.[105]

Levinson and Edelberg (1985, Table 5) reported means and standard deviations for the SCR lat. in response to differingly strong acoustic stimuli for the first and subsequent stimulus presentations within habituation experiments performed with various groups of schizophrenics and control subjects (Sect. 3.4.2.2). No differences resulted between the first and subsequent presentations; in one data set the SCR lat. was, however, shorter (1.44 sec) in response to white noise of presumably over 100 dB used as UCS than in response to tones of 78 dB (1.92 sec).

With 42 subjects, Surwillo (1967) found highly significant differences in the SPR lat. between conditions of simple and disjunctive acoustic stimuli. In the first case, the SPR lat. had a mean of 1.73 sec with SD = .2 sec, while in the second case a mean of 1.65 sec with SD = .224 sec was obtained.

The EDR rise time is a relatively seldom studied variable. Grings (1974) gives the range as being between .5 and 5 sec. Venables and Christie (1980) only determined the

[105]He used zinc electrodes with a contact surface area of .32 cm^2, zinc sulphate as electrolyte, and a constant current method, with 3.0 μA; results were transformed into conductance units, and the SCR amp. underwent a square root transformation (Sect. 2.3.3.3.).

SCR ris.t. values for their sample of sixty-five 18- to 75-year-old subjects, and found it was 2.184 sec on average, with a standard deviation of .643 sec, the distribution being slightly positively skewed and platykurtic, but far from a significant deviation from normality. In his study cited above, Lockhart (1972) found a mean SCR ris.t. of 2.8 sec with SD = 1.54 sec.

Venables and Christie (1980), in Tables 1.9–1.11 of their review, give interindividual and intraindividual correlations between SCR lat., SCR ris.t. and other temporal, level, and amplitude measures of the skin conductance from their own and other investigations. From these results, the relative independence of the latency time from other temporal measures as well as the SCR amp. and the SCL could be shown. The correlations were mostly negative and smaller than –.21. By contrast, the correlations to the logarithmized SCL and SCR amplitude scores were higher, whereby a marked connection between the SCR lat. and the logarithmized amplitudes was displayed (r= –.31 to –.58). That means, if SCR amp. are logarithmized, a connection exists in that following shorter latency times, larger amplitudes appear. The correlations between SCR lat. and SCR ris.t. were between .17 and .30 (i.e., following greater latency times, shorter rise times tend to appear).

Using intraindividual correlations within 13 subjects, Bull and Gale (1971) also found links between a low SRR amp. on the one hand, and long latency times and short rise times on the other. However, in most cases significance was not found. In a further investigation with 12 subjects, Bull and Gale (1973, Table 3) found significant interindividual rank correlations between the SRR lat. and several other EDA parameters ranging from –.44 to –.64. The reliabilities, calculated as intraclass correlations over four measurement periods (Footnote 102 in Sect. 2.5.2.2.1) were .84 for the SRR lat., and .67 for the SRR ris.t.

Venables, Gartshore, and O'Riordan (1980) obtained (N = 65) a greater correlation between the SCR ris.t. and measures of attentiveness toward the environment derived from the ECG, than between the SCR rec.t/2 and the ECG measures in question.

To date, few results are reported on the EDR's maximum incline (Sect. 2.3.1.3.1). Fahrenberg, Walschburger, Foerster, Myrtek, and Müller (1979) calculated the respective mean of the maximum incline of NS.SCRs during a 2 min resting period and during a 2 min mental arithmetic exercise under noise with 125 subjects. The scores, measured in .01 μS/sec units, displayed a mean of 103.5 with SD = 60.69 during rest, and a mean of 133.6 with SD = 67.25 under the stress condition. The distributions under both conditions were significantly positive skewed and platykurtic. The maximum incline was practically uncorrelated with the SCR ris.t. (r= .03), and was slightly negatively correlated with the SCR rec.t/2 (r= –.29). By contrast, a positive correlation existed with the SCR amp. (r= .66) and also – though being somewhat lower – with the NS.SCR freq. (r= .25). During the mental arithmetic condition, the SCR amplitudes were higher overall than in the resting period, the SCRs being of shorter duration and displaying a greater maximum incline.

In summary, only little distributional data exist for the parameters discussed in this section. Thereby, latency and rise times appear to satisfy the criteria of a normal distribution well (except with children), while the maximum incline shows a rather positively skewed flatter distribution. The reliability of the latency tends to be somewhat higher than that of the rise time. The latency appears to be a relatively independent parameter with respect to the other temporal measures, but it can display correlations with both the rise time and the EDR amp. The maximum incline appears to be an autonomic parameter of reaction shape, which, however, can be positively correlated with EDR amplitude.

2.5.2.4 Measures of recovery

Venables and Christie (1980) report recovery scores as SCR rec.t/2 values. For the 220 subjects from the Mauritian sample, there was a mean of 4.144 sec (3.252 sec for the 5-year-olds and 4.851 sec for the 25-year-olds) with a standard deviation of 2.466 sec (2.197 sec for the 5-year-olds and 2.725 sec for the 25-year-olds), the distributions being slightly positively skewed and leptokurtic. These deviations markedly increased if reciprocal scores of the recovery were calculated, while a logarithmic transformation only increased the kurtosis. The SCR rec.t/2 mean was 4.113 sec with SD = 3.217 sec for 678 three-year-olds. The insignificant deviation in skewness from the normal distribution became highly significant after a reciprocal transformation. The SCR rec.t/2 mean was 3.971 sec with SD = 5.012 sec for 42 subjects from the adult sample. By means of the above-mentioned transformations, the positive skewness became insignificant, but the kurtosis still significantly deviated from the normal distribution. Therefore, Venables and Christie (1980) recommend against using reciprocal transformation of the rec.t/2 measure and also point out that the logarithmic transformation will not improve any distributional problems.

In their study described in Section 3.4.1.2, Levander, Schalling, Lidberg, Bartfai, and Lidberg (1980) found a significant negatively skewed distribution of the SCR rec.t/2 in an habituation paradigm using 25 male delinquents between 18 and 30 years of age. Through logarithmic transformation the distribution was normalized. In a two day habituation experiment with 71 hospitalized male subjects, Hinton, O'Neill, Dishman, and Webster (1979) found a SRR rec.t/2 reliability of .63.[106] As for the rise time (see previous section), Bull and Gale (1973) found only an insignificant (r= .18) reliability for their recovery time.[107]

Correlations with other SC measures, collected from various studies, were also published for the SRC rec.t/2 by Venables and Christie (1980) in their Tables 1.9–1.11. The data show high consistency over differing stimulus conditions, gender, and clini-

[106]Recording was performed with constant current using concentric electrodes with internal diameters of 5 mm and external diameters of .6–1 cm, using .05 molar KCl cream on an agar base, from the index and middle fingers of the left hand.

[107]Recovery measured in percent of amplitude decrease 2 sec after the point of maximal deflection.

cal groups. The correlations of the SCR rec.t/2 with the other SC parameters were low throughout, although values of around .40 also appeared. Correlations between SCR rec.t/2 and rise time, on the other hand, lay between .54 and .80. The half-time recovery therefore appears to be relatively independent of other components of the SCR, but a marked relationship to the rise time exists. These authors conclude that if recovery is difficult to measure (e.g., if a NS.SCR appears before half-time recovery is reached; Sect. 2.3.1.3.2), the less difficult and determinable rise time can be used as a form parameter instead.

In his 5 experiments with a total of 129 students (Sect. 2.5.2.3), Lockard (1972) also found a positive correlation of .62 between the rise time and the SCR rec.t/2. The correlations that he found between the amplitude and the three temporal measures – latency, rise time, and recovery – were rather small and insignificant (r= –.11, .04, and –.06). These correlations were somewhat higher for the rise time and recovery (r= .39 and .44) if the unexpected appearence of an electrical stimulus as UCS was evaluated separately. Lockhart attributed this condition-dependent intercorrelation between the EDR amp. and the EDR rec.t/2 to the action of a homeostatic mechanism under certain conditions, while under most other conditions the two measures remain independent of each other. He obtained a mean of 4.8 sec with SD = 2.92 sec for the SCR rec.t/2 .

Becker-Carus and Schwarz (1981) studied 30 male soldiers during a series of short-term memory tasks and correlated the SRR amp. with the "half-life period," as defined by Lüer and Neufeldt (1968), which contained both the rise time and recovery characteristics (see Footnote 57 in Section 2.3.1.4). The correlations were positive and mostly significant, ranging from .19 to .63. In their above-mentioned study, Levander et al. (1980) found significant negative correlations in an habituation series between the mean SCR rec.t/2, on the one hand, and the mean SCL (r= –.55), as well as the mean NS.SCR freq. (r= –.65), on the other hand. The correlation to the mean SCR amp. remained insignificant (r= –.14).

In summary, the frequently used EDR recovery parameters, which also often cannot be objectively evaluated (Sect. 2.3.1.3.2), yield measures of rather questionable reliability. Their possible dependency upon other EDA parameters is also partly unexplained (Sect. 2.5.2.5). Transformations of recovery times should be avoided, as they tend to worsen the distributional characteristics.

2.5.2.5 Relationship between measures of amplitude and shape

While little attention has been paid to, and little data gathered on, the relationship between EDR amp. and rise time (Sect. 2.5.2.3), the possible autonomy of the recovery time has been the subject of numerous studies and controversy.

The starting point for the controversy was Edelberg's (1972a) hypothesis that sweating and sweat reabsorption have separate nervous controls and that the SCR rec.t/2 is the best measure for the reabsorption processes (Sect. 1.4.2.3). The view of EDR recovery as having an independent indicator function (in contrast to other EDA parameters)

is supported by the studies of Edelberg (1972b) and Janes (1982) described in Section 3.1.3.1. Recovery time could be used to differentiate between rest and stress conditions, and also between task performances of varying complexity, and stimulus significance may affect recovery to an even greater degree than amplitude. In addition, the EDR recovery has been shown to be an especially valid predictor in some risk studies on schizophrenia (Sect. 3.4.2.1). The hypothesis of an autonomy of the recovery time has been supported by Venables and Christie (1973). They argue that on the basis of an exponential drop of the EDA the time constant must be mathematically independent from the amplitude per se. However, as shown in Section 2.3.1.3.2, the exponential function is only one possible description of the recovery process of the EDR.

A decisive empirical objection to the recovery's autonomy was made by Bundy and Fitzgerald (1975), who found that the time of descent was dependent upon the number and intensity of preceding SCRs. Bundy and Fitzgerald proposed a measure "X," in which the amplitudes of the two spontaneous SCRs immediately preceding a stimulus-dependent SCR were divided by the respective times (t_1 and t_2) between them and the stimulus-dependent SCR, the results then being added as per Equation 46. This measure "X" displayed intraindividual correlations between –.51 and –.91, with the half-life of the stimulus-dependent SCR obtained from five subjects.

$$X = \frac{SCR\,amp._1}{t_1} + \frac{SCR\,amp._2}{t_2} \tag{46}$$

This measure was also used by Venables and Fletcher (1981), who studied the dependence of the SCR rec.t/2 upon the SCR amp. of preceding EDRs among 65 subjects of both genders, using the same techniques of measurement as in the Mauritian study with three-year-old children (Sect. 2.5.2.1.1). The subjects received 20 stimuli of 75 dB and 1 kHz, except for the sixth stimulus, which was 1,311 Hz. Intraindividual correlations were calculated only from those 10 subjects with two spontaneous SCRs before the stimulus-elicited SCR in a minimum of 5 out of 20 trials. Apart from two positive correlations, which were .47 and .84, all others between the SCR rec.t/2 and the measure "X" were negative (from $r = -.15$ to $r = -.79$), as would be expected according to Bundy and Fitzgerald (1975). However, those coefficients were significant only in two cases, which may partly be due to the low number of scores per subject (between 5 and 12).

Bundy and Fitzgerald also took analysis of data from the Mauritian study into regard, but found only 11 cases out of the almost 1,800 children in the study who showed two anticipatory SCRs (FIR and SIR, Sect. 3.1.2.1) in the CS-UCS interval of a conditioning paradigm over more than half the trials. The intraindividual correlations between the SCR amp. of the UCS and the measure "X" displayed the total range of possible positive and negative scores (cf. Venables & Fletcher, 1981, Table 3). This study is also an example of the data base being too narrow to either support or reject Bundy and Fitzgerald's (1975) hypothesis, owing to the small minority of investigated cases with the necessary data.

Large individual differences with respect to the connection between recovery time and the amplitudes of preceding EDRs were also found by Edelberg and Muller (1981). In an experiment with 20 subjects, the authors correlated the SCR rec.t/2 (using standard technique with KY-gel) and the "X" score during word association and reaction time tests. The "X" scores could only predict 14% of the recovery variance; the individual scores were between 0% and 70%. A reanalysis of the data from Edelberg (1972b), with "X" as a covariate, resulted in no significant alteration of the differential indicator function of the recovery time. However, if the number of NS.SCRs – appearing in the last 15 sec before the SCR whose recovery was evaluated – was used as covariate, the differences in SCR recovery originally found (as described at the beginning of this section) were no longer significant.

In a study exploring differentiated motor reactions to varying acoustic stimuli using 55 subjects of different races and both genders, Janes, Strock, Weeks, and Worland (1985) showed that the SCR rec.t/2 (using standard technique with KCl electrolyte) was independent from both the "X" score, according to Bundy and Fitzgerald (1975), and from the NS.SCR freq. before the specific SCR, determined according to Edelberg and Muller (1981). Significant intraindividual correlations with an average score of .61 appeared in 16 cases.

In summary, the question of a possible autonomy of EDR recovery measures cannot be totally settled on the basis of existing data. However, there is some evidence that the possibility of recovery times being not independent from preceding spontaneous EDA should be taken into account. Alternatively, this problem could be considered as a question of dependency of phasic upon tonic EDA, which is, however, discussed in the context of links between the EDR amp. and the directly preceding EDL (Sect. 2.5.4.2), and does not necessarily imply a dependency of recovery on tonic measures as well. As long as the mechanism of electrodermal recovery is not fully understood (Sect. 1.4.2.3), there is no need to dismiss recovery as a relatively independent measure of a single reaction (Venables & Fletcher, 1981), given also the low intercorrelations between recovery and amplitude as reported in the previous section. That the time measures are independent from electrode size and electrolyte type, like the amplitude measures, implies a relative independence from time and amplitude measures of the EDR (Venables & Christie, 1980). Cort, Hayworth, Little, Lobstein, McBrearty, Reszetniak, and Rowland (1978) combined intraindividual correlations between the SCR amp. and SCR rec.t/2 from five different studies with a total of 140 subjects and found differing dependencies of both measures; in the experiments using habituation of simple orienting responses (Sect. 3.1.1.1), significant links between amplitude and recovery appeared with almost all the subjects. However, if motivational and emotional situative components were added, the number of significant correlations decreased to less than 50%.

2.5.3 Characteristics of exosomatic AC measures

Since alternating current EDA recordings have been used to date nearly exclusively to investigate the system properties of the skin (Sect. 1.4.3.3), data from only a small number of studies with AC measurements are available. Furthermore, results from those studies, most of them also having used only a small number of subjects, are hardly comparable with each other, owing to different concepts of measurement used. In the next two sections, on sinusoidal AC and pulsed DC, respectively, a separation between level and reaction scores can be omitted because, for the most part, only the tonic measures are recorded. With respect to results from older studies the reader is referred to reviews given by Tregear (1966) and Edelberg (1971).

2.5.3.1 Recordings with sinusoidal current

In an experiment performed with 104 subjects, Lawler, Davis, and Griffith (1960) used a Wheatstone bridge (Sect. 2.1.5) with an oscilloscope; two variable capacitors were wired in parallel with the potentiometer. The authors used alternating voltage of 2 V and .1 mA with frequencies of 1, 4, 10, and 20 kHz.[108] The necessary R and C values for the bridge were used to calculate the phase angle and the impedance. As expected, the impedance decreased with rising frequency, the mean impedance being 6.487 kΩ (SD = 1.733 kΩ) at 1 kHz and .507 kΩ (SD = .111 kΩ) at 20 kHz. The authors then decided to use 4 kHz for further measurements, which yielded an average impedance of 1.882 kΩ (s = 0.468 kΩ) with approximately normally distributed scores. The phase angle decreased between 1 kHz and 20 kHz with means of 75^0 (SD = 5.0^0) and 57^0 (SD = 5.9^0), respectively. A higher impedance and lower capacitance were observed; however, quantitative values were not reported. In addition, the authors removed the stratum corneum and stratum intermedium on certain sites with 23 of the subjects by the skin stripping technique (Sect. 1.2.1.1), after which the impedance fell and the phase angle increased as the frequency rose, in contrast to the intact skin sites.

Plutchik and Hirsch (1963) performed AC measurements of 14–61 μA with 1, 10, 50, 100, and 1,000 Hz using two subjects. The authors used dry silver electrodes of 1 cm diameter fastened to the palmar side of a finger. As the frequency rose, the impedance fell from 130 to 30 kΩ and the phase angle increased from -2^0 to -58^0. Both measures showed themselves to be invariant with respect to the applied current density. The interindividual differences were smaller with the phase angle than with the impedance. Faber (1977) observed – without giving exact descriptions of the measurement technique or the number of subjects – a decrease of impedance from 152.6 kΩ to 14.6 kΩ with frequencies of between 10 Hz and 1 kHz, which somewhat confirms the results from Plutchick and Hirsch.

[108] They used stainless steel electrodes with diameters of 2 cm placed 2 cm apart on the volar middle of the underarm, fastened with rubber bands to filter paper impregnated with a NaCl solution.

Burton, David, Portnoy, and Akers (1974) studied the palmar skin response in six subjects to AC with .1– .3 V effective voltage and 13 or 3 different frequencies of between 10 Hz and 100 kHz, using a frequency analysis suitable for passive electrical systems, and Ag/AgCl electrodes of 2 cm^2 area each with isotonic cream. The results were very differentiated; most importantly, they confirmed the decrease in impedance and the increase in the phase angle with rising frequency. The means over all frequencies for each subject were determined for the single parameters of the Montagu-Coles model (Fig. 15, left-hand panel, Sect. 1.4.3.1). Burton et al. observed (with respect to 1 cm^2 area of skin) scores of between 470 Ω and 2.0 kΩ for the serial resistance R_1, between 159 and 212 kΩ for the parallel resistance R_2, and between .0075 and .013 μF for the capacitative element C, the phase angle being between -8^0 and 63^0.

Yamamoto et al. (1978) determined the single parameters of their equivalent circuit (Fig. 18 in Sect. 1.4.3.3), but they set aside the resistance R_1 representing the deeper skin layers because in an earlier study (Yamamoto & Yamamoto, 1976) they found that in using the skin stripping technique the skin impedance mainly derived from the resistive properties of the keratinized layers of the epidermis, while the deeper layers (including the stratum granulosum) contributed less than 500 Ω per cm^2 of skin to the overall resistance. Recordings were made three times within 6 hours on both forearms, using Ag/AgCl electrodes with liquid electrolytes (Sect. 2.2.6.3), having a skin contact area of 3.14 cm^2. A constant current of 10 μA was applied at frequencies of 10 Hz to 1 kHz. The value found for the conductance component corresponding to the resistance R_2 was between 1.84 and 4.17 μS, the conductance component corresponding to the resistance R was between .029 and .793 μS, and values for C were between .143 and .155 μF. In total, the variances were small and the distributions approximately normal, as described by Yamamoto and Yamamoto (1978), who covered the same data set.

DeJongh (1981) studied SZ, using an alternating voltage at 25 Hz and 32 μA with 263 subjects. He used platinum electrodes of 1 cm^2 in area and a liquid electrolyte (.015 molar NaCl) with a contact surface of 6.2 cm^2. The average impedance from three equidistant sites on the volar side of the right underarm was over all subjects 51.211 kΩ (SD = 13.234 kΩ); the corresponding logarithmic mean was 1.692 log kΩ (SD = .117 log kΩ). Both raw scores and logarithmized scores were normally distributed. Using frequencies between 7 Hz and 1 kHz at recording sites on the backs of 14 subjects, Zipp et al. (1980) found a markedly higher decrease of skin impedance after 30 min in the lower-frequency range as opposed to higher frequencies.

As previously noted in Section 1.4.3.3, hardly any data exist for the behavior of single components of skin impedance and admittance during the EDR. McClendon and Hemingway (1930) found both a variation in the impedance and a marked capacitative change during the EDR with a single subject. However, neither the underlying skin model nor the method used to calculate C were specified by the autors; the temporal trends also point to tonic rather than phasic variations. Forbes and Landis (1935) found capacitative changes during the EDR, which, however, only constituted .5–1% of the tonic values.

These results of the capacitative component of EDRs could not be confirmed by Boucsein et al. (1989), who used 100 Hz alternating voltage with considerably better equipment (Sect. 2.2.3.3) and three subjects; the EDR appeared to be due primarily to changes in the parallel resistance R. With a similar arrangement of measurement based on a lock-in amplifier, Grimnes (1982) found marked capacitative changes at 20, 90, 500, and 1,000 Hz with EDRs provoked by movement together with the holding of the breath. However, since he used dry electrodes covered with AgCl, and since the EDRs had unusually long rise and recovery times, it is presumable that he struck a polarization capacitance build-up effect in the epidermis, rather than a moisturizing effect on the sweat gland ductal walls due to the rise of the sweat column during the EDR, the explanation proposed by Grimnes. He also found that the latency time was 2 sec or more longer for the susceptance than for the conductance, a finding that also does not support his view.

Thus, Edelberg's (1971) conclusion, that the electrical processes during an EDR are fully attributable to resistance variations in the parallel branch of the electrodermal skin model, owing to the .5–1% (at most) capacitative changes, could not be conclusively disproved until recently. To a large degree, the results of corresponding studies, like those using constant voltage measurement, show dependence upon marginal conditions such as the type of electrode, electrolyte, and, in addition, the AC frequency used. Therefore, in order to solve the question of the part that capacitative structures play in the EDR, more systematic investigations must be performed.

2.5.3.2 Recordings with square wave current

Yokota and Fujimori (1962) studied the changes in the skin's system properties during the EDR in one subject, using square wave pulses of 50 msec in length, 10 μsec in rise slope, with 20–100 mV, and a repetitive frequency of 3–5 Hz. The authors used a unipolar recording with an active palmar Ag/AgCl electrode (with a physiological NaCl solution as electrolyte) and an inactive electrode on the forearm. In concordance with the model shown in Figure 18 (Sect. 1.4.3.3), the values of the serial resistance R_1, the parallel resistance R_2 (including R), and the capacitance C were determined both before an EDR and during its maximum. During an EDR, the change in the serial resistance R_1 was smaller than .1 kΩ, and the change in the capacitance was smaller than .001 μF, while the change in impedance fully loaded the parallel resistance R_2, which decreased by between 15% and 49%. The resting value for R_1 was between 300 and 800 Ω, that for R_2 between 34 and 168 kΩ, and that for C between .12 and .29 μF.

Kryspin (1965) used pulses of 4 sec duration with a current density of between .1 and 90 μA/cm^2. He found the average palmar impedance from 5 of his 14 subjects to be 406 kΩ, using Ag/AgCl electrodes on both palmar and dorsal hand sites, as well as dorsal foot sites.

Lykken (1971) performed a study using pulsed DC with a unipolar recording from an active palmar electrode of 10 cm^2 together with a reference electrode on a forearm

site previously prepared by skin drilling. The bipolar pulse sequence was 50 msec positive and 50 msec negative, with 50 msec pause at the active electrode, at between .2 V and 10 V. Recordings from the six subjects of the study were not reported individually. It was found that the serial resistance R_1 remained constant, which also held true for the parallel resistance R_2, until the 2 V level was reached. Then R_2 decreased by 24% at 5 V and by 35% at 10 V. If an active recording site was previously prepared by skin drilling, the charging and discharging behavior of the skin changed so that it appeared as if a parallel circuit, composed of a number of very small resistors with a capacitor, had been replaced by a solely capacitative circuit with a small serial resistance.

In his investigation of the linearity of the current voltage curves of the skin, Stephens (1963) used pulsed DC of between 3 and 300 msec duration at 60 μA to 1 mA during 1 min rest pauses, with a unipolar recording with a 7 cm^2 liquid electrode on the underarm. He found (although with only one probe) linear behavior between −1V and +1V, and a skin impedance of 13 kΩ, which decreased to 4 kΩ at 400 μA. The voltage built up in the skin in the first 4 msec approximated an e-function until 300 μA, but deviated markedly from such functions at higher current (Sect. 2.3.1.3.2). The decrease in voltage following termination of the current was approximately exponential at .6 V, and significantly steeper at 1.4 V and 4 V. On that basis, the author decided that the behavior of the skin can be modelled by a nonlinear resistance together with a parallel capacitance in circuit.

Van Boxtel (1977) used DC pulses of 1 msec duration at different frequencies and at 1–10 mA with both constant current and constant voltage. He used Ni/Ag electrodes of 3.53 cm^2 contact area and an isotonic NaCl electrolyte cream on bipolar recording sites on the lateral and medial gastrocnemius muscles. The parallel resistance R_2 displayed a marked dependence on the current, showing changes both over time and following stimulation; by contrast, R_1 changed little. With these results, van Boxtel confirmed the findings from Lykken (1971), including the effects of skin drilling.

To date, pulsed DC has hardly been used in recording of the EDR. In using this method with an oscilloscope, Lykken (1971) showed that, during an EDR, although variations attributable to a capacitative influence appeared, a similar picture could be made to appear on the oscilloscope through proper changes in the parallel resistance R_2. As for measurement with sine wave AC, too few results exist for safe assertions regarding the effects of variations of single components of an underlying skin model during the EDR.

2.5.4 Level dependence

Level dependencies of psychophysiological data are usually discussed with reference to the so-called Law of Initial Values (LIV) formulated by Wilder (1931), which states that reaction amplitudes in ANS-controlled physiological systems are expected to be reciprocal to their respective baselines. The LIV is physiologically based upon the antagonism between the sympathetic and parasympathetic branch of the ANS, which

brings about homeostatic functioning by preventing preponderance of either branch. Since EDA can be regarded as being only influenced by the sympathetic branch of the ANS (Sect. 1.3.2.3 & 1.3.4.1), the LIV is not directly applicable to EDA.

Hord, Johnson, and Lubin (1964, p. 86) considered EDA as belonging to the "slow equilibrium variables," and owing to the missing parasympathetic counter regulation, the LIV is not valid. Nevertheless, a large number of studies investigating the validity of the LIV have used EDA. This use may be due to a misunderstanding. While for most other physiological variables the necessary baseline scores can only be determined with specific sampling methods taking into account their respective functional fluctuations (see, for example, Malmstrom, 1968), the EDA baseline value appears to be easily determined as the EDL measured immediately prior to each single EDR.

Instead, from a psychophysiological conceptual point of view, investigations into the validity of the LIV as regards EDA, on the one hand, and investigations into the dependence of the EDR upon the preceding EDL, on the other, should be clearly separated from each other. The main reason is that, most probably, differing physiological mechanisms are responsible for the EDR and the EDL (Section 1.4.2.3), but LIV investigations make the same class of parameters the basis for baseline values and reaction scores. The differentiation of EDR and EDL has also been suggested by Levey (1980, p. 619) who separates the following two tonic EDA scores:

(1) The resting EDL before commencing each stimulation and/or before introducing any experimental condition. This would correspond to the baseline as required by the LIV.

(2) The EDL in the intervals between single EDRs during subsequent stimulation, for example, during the interstimulus intervals of an habituation study (Sect. 3.1.2). This tonic value is the respective EDL for investigating tonic phasic relationships.

The two following sections deal separately with the possible dependence of the EDR upon the resting EDL (Sect. 2.5.4.1) and the possible relationship of EDR to the directly preceding EDL (Sect. 2.5.4.2).

2.5.4.1 Dependence of treatment recordings on baseline recordings

The problem of psychophysiological reaction scores, obtained under treatment conditions, being dependent on baseline recordings had been discussed during the 1950s in a number of publications; however, the discussions were based mainly on insufficient empirical data bases without careful separation of physiological from statistical concepts. A thorough depiction together with the theoretical basis and an empirical scrutiny of the LIV, with extensive data, is to be found in Myrtek, Foerster, and Wittmann (1977), who systematically studied the statistically important a(a – b) effect[109] with regard to

[109]When a baseline score (a) and a reaction score (b) are uncorrelated, the correlation of the reactivity measure (a – b) and the baseline score (a) can not equal zero because they have a common term (Myrtek

baseline dependence. An intraindividual scrutiny of the LIV with 20 subjects over 16 trials, as well as two interindividual studies of baseline dependence (N = 107 and 67), displayed only a few negative correlations between baseline and reaction scores which would confirm the LIV. Instead, twice as many positive correlations appeared, which were contrary to the LIV. Thus, owing to the lack of convincing data, the LIV cannot be regarded as an overall valid law, as Wilder (1931) and many later studies tried to show. Instead, the LIV must be regarded more as a rare exception to the rule than the rule itself (Myrtek & Foerster, 1986).

There are few studies on level dependence of EDA that do not refer to the dependency of the EDR on the preceding EDL. This kind of level dependence will be discussed in the next section. In addition, the question of baseline dependence of EDA (as with level dependency) is too quickly coupled with the problem of the choice between SC and SR units (Sect. 2.6.5), and the necessity of checking the a(a – b) effect remains mostly unrecognized.

Hord et al. (1964) recorded the SRL with 105 subjects just before presentation of a 500 Hz, 73 dB tone and recorded the lowest SRL[110] in the 5 sec following the tone. The authors transformed the resistance units into conductance units, and partly found high positive correlations (between .35 and .77) between the SCL prior to the stimulus and the rise of the SCL afterwards, therefore invalidating the LIV. Instead, the authors point out that owing to the reciprocal relationship between conductance and resistance, the LIV must be valid for resistance. Benjamin (1967) performed a so-called Monte Carlo study with a large number of random number correlations. He could show that while a reciprocal transformation may change the polarity of the correlations from prestimulus to difference scores, this is not necessarily the case. Thus, the author concluded that the above-mentioned considerations from Hord et al. (1964) on the validity of the LIV for SR data as opposed to SC data were based on false premises.

Myrtek et al. (1977) recorded both the baseline and the mean SCL during a 5 min measurement phase during which their 67 subjects did mental arithmetic exercises under the stress of noise. The authors found a nonsignificant correlation of –.19 between the baseline and the SCL increase during the mental arithmetic. In addition, a specific coefficient for the determination of the "true" baseline dependence (avoiding the a(a – b) effect) was also nonsignificant.

Shapiro and Leiderman (1954) could not confirm the validity of the LIV in an experiment measuring SP with 53 student nurses (Sect. 2.5.1.2). They found only a correlation of –.09 between the average SPL baseline and its rise during an easy reaction task. Gaviria et al. (1969) studied the baseline dependence of endosomatic and exosomatic EDA by simultaneously recording both SP and SR from 20 married couples (for the methodology, see Sect. 2.5.1.3). The correlations between the baseline scores

& Foerster, 1986). With many physiological variables (a) and (b) are not totally independent from each other, which leads to differingly high correlations between (a) and (b), and therefore to a differingly large a(a – b) effect.

[110]Palmar recording with zinc sulfate cream, at 40 μA.

and variations of the EDL during five different acoustic stimulus presentations were between .47 and –.47, being significant for the SR for only one male and one female subject; that is, in most cases the LIV appeared to be invalid. Because of the overwhelmingly negative results, Venables and Christie (1980) conclude that no consensus on the validity of the LIV in regard to EDA exists, and it is doubtful if the question could be answered in general anyway.

However, an examination of possible baseline dependencies cannot be dispensed with when reaction scores must be calculated in the form of differences to baseline scores. When the LIV applies in such a case, evaluations of the individual reactivity using the differences to the baseline are subject to a systematic error as significantly entailed by each baseline score (Fahrenberg & Myrtek, 1967). The same holds for statistically significant positive correlations between reaction and baseline scores which contradict the LIV.

In order to avoid the problem by calculating bias-free reactivity scores, many authors make use of ALS scores (Sect. 2.3.3.4.4) as suggested by Lacey (1956). These are practically based upon a covariance analytical correction of the reaction scores which, after correction, are independent of baseline influence by definition. However, assumptions such as the linearity of regression of the reaction and baseline scores, and the bivariate normal distribution or even the homoscedasticity, are often neither checked nor given (Fahrenberg & Myrtek, 1967; Lykken & Venables, 1971). Also, often no care is taken to make sure that the samples are not too small for the necessary standardization when calculating the ALS scores.

Fahrenberg et al. (1979), using a large data set, performed a systematic analysis of different baseline correction methods, ranging from simple difference scores to reaction scores based on main component analysis. Their conclusion was that no particular method can be recommended. Instead, in each single case the question of baseline dependence should be checked using bivariate distribution of resting and reaction scores, together with their intercorrelation; then the decision can be made as to whether to make a correction or not. Whether statistical corrections of reaction scores in relation to baseline scores make sense psychophysiologically or not, or if baseline dependency should only be recorded through particular parameters as a system-specific reaction component and therefore additionally be reported, cannot yet be answered. Furthermore, the answer to this question depends on which source of variance the experimenter will be interested in.

A baseline dependency that appears in extreme ranges of tonic EDA is formed by so-called ceiling and bottom effects. When the SCL is already very high in the rest phase at the beginning of the experiment, because of physiological considerations, SCL can only increase to a limited degree when activated by experimental conditions (a ceiling effect), in contrast to a more average SCL baseline. The same but opposite effect is true for an extremely low initial SCL baseline, which cannot be lowered much further by deactivating conditions, thus yielding a bottom effect. This baseline dependency

simulates the idea of the LIV, but it does not correspond to the original concept, as it is not a consequence of a homeostatic regulation (Lykken & Venables, 1971).

A similar effect occurs in comparing groups which possibly display differing EDLs, as for example with anxious and nonanxious subjects (Edelberg, 1972a; see also Sect. 3.3.1.2). Therefore, baseline scores should be considered on an individual basis when necessary for the evaluation of electrodermal reactivity.

2.5.4.2 Dependence of phasic EDA on tonic values

As already outlined in the previous section, the majority of investigations into the baseline dependency of EDRs take the directly preceding EDL as a reference. There is not much point in referring to the existing literature on phasic-tonic correlations, as the results of the level dependency studies are extraordinarily inconsistent, resulting mostly from differing experimental settings. Different level dependencies may result from situational characteristics, for example, when an experimental group is activated and thus the SCL increases steadily, while a control group shows deactivation instead, with a decrease in SCL. If, under those circumstances, SCRs from a repeated single stimulation are considered, which are expected to habituate over a number of trials (Sect. 3.1.2), a negative tonic-phasic correlation would result under the experimental condition and a positive correlation between SCL and SCR amp. would be more likely under the control condition.

On the other hand, differing level dependencies appear in general, depending on whether interindividual or intraindividual correlations are calculated. Thus Martin and Rust (1976) found that the relationship between the SCR amp. and the SCL depended on the correlational methods used. In an habituation experiment with 84 twins as subjects, each presented with 21 tones of 1 kHz at 95 dB, Martin and Rust found that the interindividual correlation of the mean scores for all stimuli was .619, while the pooled intraindividual correlation yielded only .081. Their interindividual correlation was in line with a result reported by Venables and Christie (1980), who reported a correlation of .62 between the SCR amp. following a single acoustic stimulus and the preceding SCL, in a study with 123 subjects.

In 60 subjects, Boucsein et al. (1984a) also found positive mean interindividual correlations between the SCR amp. and the SCL, which however decreased with rising intensity of the applied stimuli (from $r = .613$ at 60 dBA to $r = .315$ at 110 dBA white noise). The corresponding intraindividual correlations, calculated over all 30 stimuli including all levels of intensity, had a mean of only .06 and a large range. In an experiment with 18 subjects who were presented with 32 electrical stimuli of four intensities, Block and Bridger (1962) found that the form of the interindividually determined regression of the SRR amp. to the preceding SRL was not predictable from the regression among the single subjects over the trials. Thus, in addition to taking the level dependencies calculated from group statistics into consideration, intraindividual

correlations between EDRs and the EDL should also be calculated and included in the interpretation.

Many corrections of the EDR using the EDL are made with the aim of eliminating phasic dependencies upon tonic EDA. As with the baseline problem discussed in the previous section, the general use of corrections with respect to a possible level dependency cannot be recommended. In using such corrections without a careful scrutiny of individual data structures, important parametric properties may be lost and erroneous interpretations of the reactivity of the electrodermal system may be made. According to Grings (1974), two cases should be distinguished in the matching of the EDR as based upon the EDL:

(1) Both scores can be regarded as correlating indicators of the phenomenon under investigation, both of them explain specific variance components. In this case, no baseline correction should be made; instead, the information from both scores should be combined. An example is canonical correlations between "electrodermal behavior" on one hand, and personality dimensions on the other (Chapter 3.3).

(2) The investigation focuses on EDRs, whereby the influence of differing EDLs is regarded as erroneous and thus should be eliminated. An example is the use of the EDR as an indicator for an orienting (Sect. 3.1.1) or for a conditioned reaction (Sect. 3.1.3). In this case, corrections considering varying EDLs may be applied (Sect. 2.3.3.4). However, it should be carefully ensured that the differing EDLs do not themselves appear as a consequence of the experimental manipulation (Edelberg, 1972a).[111]

It is both theoretically and practically important that the level dependence of the EDR is noninvariant during a transformation of SR into SC data and vice versa (Johnson & Lubin, 1972; see also Sect. 2.3.3.2). In their above-mentioned study, Boucsein et al. (1984a) showed that the correlations between SRR and SRL increased together with stimulus intensity, while the correlations between the simultaneously measured SCR and SCL behaved inversely. Therefore, the intraindividual tonic-phasic correlations for the SR, as determined across the different stimulus intensities, were significantly higher than those for SC, and a high standard deviation of the correlations pointed to large interindividual variations in the level dependence of the EDR.

One possible explanation of this differential level dependence with the use of either SC or SR can be given using the simplified Montagu-Coles model (see the right-hand panel of Fig. 15 in Sect. 1.4.3.1). Here, R_1 and R_2 (the resistances of the dermis and of the stratum corneum) are regarded as mostly responsible for the EDL, and a variable resistance x (which can be traced back to sweat gland activity) is regarded as being responsible for the EDR. The total resistance R of the system can be calculated

[111] A thorough discussion of such corrections which are based either on the use of transformations or on regression techniques is found in Levey (1980, p. 619ff.).

by using the rule for adding two resistors in parallel for the addition of R_2 and x, and adding R_1 according to the more simple rule for resistors in series[112]:

$$R = R_1 + \frac{R_2 x}{R_2 + x} \tag{47}$$

The conductance values G (corresponding to R), G_1 (corresponding to R_1), G_2 (corresponding to R_2), and y (corresponding to x) are given according to Equation (2a) in Sect. 1.4.1.1 as follows:

$$\frac{1}{G} = \frac{1}{G_1} + \frac{\frac{1}{G_2}\frac{1}{y}}{\frac{1}{G_2} + \frac{1}{y}} \tag{48a}$$

When the sums in the denominator of the right-hand fraction of Equation (48a) are added, $1/y\ G_2$ can be shortened, producing:

$$\frac{1}{G} = \frac{1}{G_1} + \frac{1}{G_2 + y} \tag{48b}$$

Further addition leads to:

$$G = \frac{G_1 + G_2 + y}{G_1(G_2 + y)} \tag{48c}$$

When Equation (48c) is inverted, an equation for the total conductance equivalent to Equation (47) is given as follows:

$$G = \frac{G_1(G_2 + y)}{G_1 + G_2 + y} \tag{48d}$$

In order to calculate the variation dR of the total resistance due to small variations dx, Equation (47) is differentiated, whereby R_1 disappears because it is a constant:

$$dR = d\frac{R_2 x}{R_2 + x} \tag{49a}$$

Because during an SRR, R_2 can also be regarded as constant, following the rules for differentiation of constants produces $d(R_2\ x) = R_2\ dx$ and $d(\mathrm{R}_2 + x) = dx$; according to the quotient rule, Equation (49a) then produces:

$$dR = \frac{(R_2 + x)R_2 dx - R_2 x dx}{(R_2 + x)^2} \tag{49b}$$

which once multiplied out gives:

$$dR = \frac{{R_2}^2}{(R_2 + x)^2} dx \tag{49c}$$

[112]See Equation (6e) in Sect. 1.4.1.2. To avoid confusion, the parameter x is used instead of R in Equation (22) in Sect. 1.4.3.1.

Correspondingly, differentiation of dG as for dy from Equation (48d), when taking G_1 and G_2 as constants, produces:

$$dG = d\frac{G_1(G_2 + y)}{G_1 + G_2 + y} \tag{50a}$$

Multiplying out the numerator in Equation (50a) gives: $G_1\,(G_2 + y) = (G_1\,G_2) + (\,G_1\,y\,)$. When this is differentiated, since $G_1\,G_2$ is constant, the following results: $d((G_1\,G_2) + (G_1\,y)) = G_1\,dy$. Differentiation of the denominator in Equation (50a) produces: $d(G_1 + G_2 + y) = dy$. Following the quotient rule, this results in:

$$dG = \frac{(G_1 + G_2 + y)G_1 dy - ((G_1 G_2) + (G_1 y))dy}{(G_1 + G_2 + y)^2} \tag{50b}$$

Multiplying this out gives:

$$dG = \frac{{G_1}^2}{(G_1 + G_2 + y)^2} dy \tag{50c}$$

As can be inferred from the equivalent Equations (49c) and (50c), the SRR and the SCR are not only dependent upon the "true" resistance and conductance variations dx and dy but are also influenced in different ways through the various branches of the base resistances or conductances of the model which result in a nonlinear dependence of reaction on level (Boucsein et al. 1984a).[113] Thus, dependencies of phasic EDA upon tonic EDA, as found in many studies, can be shown also theoretically in the model. Additionally, a differential level dependence appears when regarding SC and SR data, which – in addition to the above-mentioned individual influences – can lead to differing and partly contradictory results when SC or SR methods are used.

How cautiously one should be with a pure statistical treatment of tonic-phasic dependencies can be shown with the following two examples. Lykken and Venables (1971, p. 669), using fictitious data, show that uncorrelated tonic and phasic SC scores may display an almost totally positive correlation after being transformed into SR units. Their example is repeated in the left-hand panel of Table 5. In the right-hand panel, uncorrelated SRL and SRR scores are produced in the same manner and are transformed into SC units, which then display almost the same positive correlation owing to the reciprocal relationship between SC and SR. Thus, it cannot be inferred from that particular example that SC units are to be preferred, owing to the nonexistent dependence of the EDR upon the EDL.

Another example is taken from Bull and Gale (1974). Their aim was to avoid habituation effects while recording the EDR in response to only a single stimulus on 10 different days; this worked with only 7 of their 15 subjects. Use of the standard constant current method (but without the necessary adaptation time for the electrode-skin

[113] When the resistance R_2 (or the conductance G_2) of the epidermis is also regarded as variable, Equations (49c) and (50c) become more complicated, as they must be differentiated for a second variable. However, in this case, different level dependencies will also result.

Table 5. Fictitious examples of generating correlative dependencies between formerly independent EDRs and EDLs after transformation from SC into SR and vice versa. Example A: from Lykken and Venables (1971, p. 669). Example B: Contrary example for SR. Equations (5a) and (5b) were used in the transformation (Sect. 1.4.1.1).

Example A:					Example B:				
Trial	SCL	SCR	SRL	SRR	Trial	SRL	SRR	SCL	SCR
	μS	μS	kΩ	kΩ		kΩ	kΩ	μS	μS
1	10	1	100	9.09	1	100	10	10	.11
2	11	1	91	7.57	2	90	10	11	.13
3	12	1	83	6.41	3	80	10	13	.18
4	13	1	77	5.49	4	70	10	14	.24
5	14	1	71	4.76	5	60	10	17	.33
6	15	1	67	4.17	6	50	10	20	.50
7	16	1	62	3.68	7	40	10	25	.83
Correlation EDR/EDL	.0		.998			.0		.985	

system) displayed a clear inverse relationship between the SRR and the immediately preceding SRL. This was interpreted by the authors as an unwanted action of the LIV (Sect. 2.5.4.1) when using SR scores. The tonic-phasic connection got lost after a transformation into SC scores, and a contradictory trend appeared. However, this can be attributed to the transformation used and to the narrow data base provided, and should not be used as a general argument for the SC being the more adequate unit of measurement.

In summary, an empirical explanation of the relationship between tonic and phasic EDA has not yet been reached, and the application of baseline corrections of the EDR using the EDL is problematical. Furthermore, the connection of questions concerning level dependence to those concerning an adequate unit of measurement for exosomatic EDA is not justified on the basis of the existing data.

2.6 Summary of conceptual discussions

Out of the three basic approaches to EDA – (1) endosomatic, (2) exosomatic DC, and (3) exosomatic AC measurements (see introductory remarks to Chapters 1.4 & 2.5) – the second method is used in the majority of investigations. Although endosomatic measurements have acknowledged advantages owing to the lack of an applied current, exosomatic recordings can be obtained and interpreted more easily, as described more fully in Section 2.6.1. AC measurement is more complicated in recording and evaluation but may provide more information than DC measurement. However, it is used to date only by a small number of authors. Section 2.6.3 compares AC with DC methods.

Additionally, the controversy regarding the problem of constant current vs. constant voltage methods with regard to exosomatic DC measurement is discussed in Section 2.6.2. In spite of Lykken and Venables's clear recommendation in the early seventies for the use of constant voltage methods, both methods are still in use.

AC uncoupling of the EDR from the EDA signal, described in Section 2.1.3, was discussed in terms of its advantages and disadvantages in Section 2.6.4. Another problem of recording which is less central, that of the choice between moist or dry electrodes, was discussed in Section 2.2.6.3.

The comparisons made in this chapter between the differing methodological concepts will provide readers with the ability to plan for optimal EDA measurement in their own investigations. In addition, the discussions in the following sections will show that – despite all attempts for standardization – the various approaches toward recording of EDA often maintain methodological values of their own.

2.6.1 Endosomatic versus exosomatic recording

Although the relative worth of the use of an external current in measurement has been thoroughly discussed in the literature (e.g., Grings, 1974, p. 277), the majority of EDA studies have been performed using exosomatic methods. One of the main reasons for this use presumably is the problem of parametrizing and interpreting the SPR amplitude (Sect. 2.3.1.2.1 & 2.5.1.1). Thus Fowles et al., (1981) agree with the recommendation made by Lykken and Venables (1971) that one should give preference to skin conductance over skin potential measurement, unless one has a definite interest in comparing one's work with the relevant literature on SP. Nevertheless, either technique has its own advantages and disadvantages as summarized below. *Exosomatic* EDA measurement has the following technical advantages over endosomatic measurement (Edelberg, 1967):

(1) Exosomatic measures are always unidirectional and therefore easier to analyze. This is not the case for biphasic or triphasic potential reactions which are inseparably composed of negative and positive waves (Sect. 2.3.1.2.1).

(2) Exosomatic recording is less affected by electrode artifacts such as bias potentials or drift (Sect. 2.2.2.2).

(3) When constant current is used in exosomatic recording, considerably less amplifier gain is required as compared to endosomatic recording (Sect. 2.1.1).

(4) No inactive reference electrode is required at an abraded site (Sect. 2.2.3.1). Abrasion may cause pain and the danger of infection (Sect. 2.2.1.1).

Fowles et al. (1981) listed two further advantages of exosomatic measurement:

(5) The sensitivity of SPL to hydrational effects is probably greater than that of SCL.

(6) Much more is known about the psychological correlates of exosomatic EDA measurements, since the majority of studies have used this method.

By contrast, according to Edelberg (1967) *endosomatic* methods have the following advantages over exosomatic ones:

(1) Endosomatic recording is regarded as being more "physiological," since the skin system is not influenced by application of an external current. This is especially an advantage in long-term runs (Sect. 2.2.6.1).

(2) Electrode polarization is prevented as no external current is applied.

(3) No special circuits are needed, as endosomatic EDA measurement can be made with sufficiently sensitive high-ohmic amplifiers (except when a separation of baselines from reaction scores is undertaken; Sect. 2.1.3). Therefore, no additional EDA coupler is needed when a good biosignal amplification system is used.

Fowles et al. (1981) added two further advantages of endosomatic methods:

(4) The simple counting of NS.EDRs (Sect. 2.3.2.2) without regard to amplitude may be more sensitive in SP measurement than in SC measurement, according to a personal note of Edelberg to the above authors.

(5) Endosomatic recordings are not affected by variations in contact area, as long as skin areas with different potentials are not connected together.

Hardly any systematic methodological comparison studies with large samples using simultaneous endosomatic and exosomatic EDA measurement (e.g., at different sites) have been made to date. Burstein et al. (1965) found a high correspondence between the appearances of SRRs and SPRs in reaction to emotionally significant stimuli with 20 subjects (Sect. 2.5.1.3). Lykken et al. (1968) attempted to find a method to estimate SC parameters from SP scores, using parallel measurements from 19 subjects. Montagu (1958) compared SP scores with SZ scores from 24 subjects and found differing baseline dependencies for both. Gaviria et al. (1969) found (with 20 male and 20 female subjects) large interindividual and gender-based differences in simultaneously measured SP and SR raw scores (Sect. 2.4.3.2), but they also obtained high correlations between SP and SR change scores throughout. Venables and Martin (1967b) studied the effects of denervation and pharmacologically blocking the sweat glands upon SP and SC, however, only with a very few subjects. The differentiated connections between SPR and SRR amplitudes found by Wilcott (1958) with 25 subjects were reported in Section 2.5.1.3.

The special importance of the SPL for the determination of the individual minimum state of activation (BSPL) was discussed in Section 2.3.2.1. Endosomatic EDA measurement remains of basic interest for research; while the interpretation of the single

positive and negative components of an exosomatic EDR appearing in response to a stimulus gives rise to additional questions of interpretation, it is possible that they may have a different psychological meaning (Edelberg, 1967).

2.6.2 Constant current versus constant voltage recordings

The quality of the discussion on the pros and cons of constant current vs. constant voltage methods has suffered from the indistinct separation between the questions of methods of measurement and units of measurement (Sagberg, 1980; Boucsein et al., 1984a). As derived in Section 2.1.1, the results from constant current methods are proportional to SR, while those from constant voltage methods are proportional to SC. As long as the corresponding EDL has also been recorded (Sect. 2.1.3), SR and SC scores can be transformed into each other (Sect. 2.3.3.2). Such transformed results obtained from either constant current or constant voltage measurements can be regarded as being equivalent (Boucsein & Hoffmann, 1979; Sagberg, 1980), since all apparent differences can be traced back to the unit of measurement (conductance vs. resistance) and not to the method of measurement (constant voltage vs. constant current; see Boucsein et al., 1984a). The argument that conductance measurement is more suitable to the physiological model of the skin (e.g., Lykken & Venables, 1971) is misleading, since it relates to the method and not to the unit of measurement, and is so discussed below. Discussion of the correct unit will follow in Section 2.6.5.

The first thorough comparison of constant current vs. constant voltage systems was made by Edelberg (1967). According to his view, the use of the constant current method had several disadvantages. Practically all models of EDA regard the sweat gland ducts being conductance paths through the epidermis. They alter their resistance or conductance according to the degree by which they are filled with sweat; single paths can therefore drop to practically zero in conductance (Sect. 1.4.2.1). When the resistance of the epidermis increases, these conductors must carry larger currents when the total current remains constant. Since the electrode area is precisely limited (Sect. 2.2.2.3), the current density therefore remaining constant, the current flowing through a single duct is in inverse proportion to the number of sweat-filled ducts. Edelberg then assumed the extreme case where most ducts no longer carry current, so that the total current is divided by a few ducts. This situation can lead to nonlinearities in the current-voltage curve.[114] Such nonlinearities appearing in dependence upon the EDL had actually been observed by Edelberg (1967) with the use of constant current methods. Subjects with low skin resistances could tolerate current densities up to 75 $\mu A/cm^2$ without the current voltage curve displaying nonlinearities, which otherwise appeared at 4 $\mu A/cm^2$ in subjects with high SRLs. Owing to these and other similar observations, the recommendation was made that with constant current measurements the current density

[114]This discussion has been used by Catania et al. (1980) to explain the dependency of age-related differences in electrodermal reactivity upon the method of measurement (Sect. 2.4.3.1).

should be limited to 10 $\mu A/cm^2$ (Sect. 2.2.3.2) and electrodes with the greatest possible area should be used (Sect. 2.2.2.1).

The above-mentioned extreme case, which may lead to damage or destruction of the affected sweat gland, cannot happen when constant voltage methods are used, since the same voltage drops over each duct and the current alters in accordance with Ohm's law in proportion to the resistance of each duct. However, with the constant voltage method, the total current is dependent upon the number of "switched on" ducts (this is not discussed by Edelberg). In an extreme case, with a high epidermal resistance and only a few filled ducts, only a very low current can flow through the whole system, and as the SR approaches the value of the inner resistance of the voltmeter, there is a greater probability for errors of measurement (Sect. 2.1.1).

The constant voltage method also has its problems according to Edelberg (1967), since the relevant models of EDA include a resistance in series with the variable resistances of the epidermis and ducts (Sect. 1.4.3.1). With a dry stratum corneum of the epidermis (which does not occur with the use of electrode paste), the EDR can be traced for the most part back to the membrane component of sweat gland activity (Sect. 1.4.3.2). Here the ohmic or impedance parts (Sect. 2.1.4) of the epidermis and the dermal structures form a voltage divider, over which practically the total voltage drops through the epidermis. With the use of constant voltage methods as compared to the use of constant current methods, changes in the membrane component of dry skin appear in the range of measurement errors. On the other hand, the absolute fluctuations of the resistance in dependent upon the membrane components remain independent from the size of the serially connected epidermal resistance (Edelberg, 1967, p. 24). In addition, very high currents (up to 100 $\mu A/cm^2$) can also flow with low SRLs, even when voltages as low as .5 V are used. However, a dry corneum will not appear when electrode paste is used.

The following comparison of the advantages of each measurement method had been given by Edelberg (1967).[115] *Constant current* techniques have the following advantages:

(1) They need less amplification by about a factor 10 than constant voltage techniques do (Sect. 2.1.1 and 2.2.4). This is especially useful in field polygraphy.

(2) The density of the current flowing through the electrodes is limited and therefore the danger of electrode polarization is reduced (Sect. 2.2.2.2).

(3) An unknown series resistance (e.g., a dry corneum) has a less serious effect in a constant current as compared to a constant voltage system.

By contrast, *constant voltage* methods have the following advantages:

[115]The points raised by Edelberg (1967, p. 25f.) on technical control of the current density with constant voltage systems are not discussed further here since they are taken care of in modern equipment (Sect. 2.2.4).

(1) High voltages over single sweat glands resulting from current concentration on a few ducts are avoided, eliminating the danger of sweat gland damage.

(2) In a certain sense, the system is self-correcting with respect to the peripheral influence of the EDL upon the EDR amp.

(3) The reference resistance, over which the amplifier measures the voltage, is low and constant (Sect. 2.1.1 and Figure 22), which leads to a good, constant relationship of the system and input impedances of the amplifier.

(4) The currents through both electrodes are independent from each other.

(5) When electrodes of differing sizes are used (which is not usual), no matching of the current density to the electrode area must be made.

An apparent advantage of constant voltage methods is that EDA measurement results are immediately expressed in conductance units and do not have to be transformed if one prefers conductance values.

What was decisive for the preference for the constant voltage technique (Lykken & Venables, 1971; Venables & Christie, 1980; Fowles et al., 1981) was the above-mentioned observation made by Edelberg (1967) concerning a possible nonlinearity when using constant current, which, however, has only been substantiated through a few trials with a small number of subjects. The concentration of the current on only a few paths of conductance may hardly ever appear in normal application with electrodes of .6 cm^2 (Sect. 2.2.2.1) and with a number of more than 200 sweat glands per cm^2 (Sect. 1.2.3). While the recommendation to use constant voltage is to be welcomed as a proposal for standardization, it is not convincing in a technical sense.

Furthermore, in electronic engineering the constant current technique is generally preferred, because with the equipment now available constant current sources are more easily stabilized and display much smaller tolerances than constant voltage sources. The recommendation here is to choose either technique according to the device available, but generally to prefer the constant voltage method on behalf of standardization. One area in which the constant current method is overwhelmingly preferred is the application of EDA measurement in so-called lie detection (Sect. 3.5.1.2).

Direct empirical comparisons of EDA measurements with constant current and constant voltage methods have seldom been made. Wilcott and Hammond (1965) performed 1 min measurements alternately with either technique using 66 subjects, where the applied voltage was varied.[116] SRL scores were calculated from the results of both methods; these were in good accordance at low voltage levels, but differed ever more as the voltage was increased. Wilcott and Hammond concluded from this that – at least with SRL scores in a mean range – both methods are equivalent. However, they recommend the use of constant voltage methods with high SRL scores.

[116]Zinc electrodes of 21 mm diameter together with zinc sulfate as electrode paste were used.

Boucsein and Hoffmann (1979) made a direct comparison study of EDRs obtained from both methods with 60 subjects. Using standard methodology, they measured EDA from both hands of each subject simultaneously with constant current and constant voltage methods.[117] The data from this study were reanalysed by Boucsein et al. (1984a); the EDR scores, obtained with constant current, were transformed into SCRs, while the results from constant voltage measurements were transformed into SRRs (Sect. 2.6.5). Reactions to stimuli with intensities exeeding 90 dB, of both the SR and SC, were found to differ from each other, regardless of whichever method of measurement – constant current or constant voltage – was used.

Barry (1981)[118] also showed that such differences do not appear if stimuli of lower intensities, both acoustic and visual, are used. In the context of an orienting reaction and the consequent habituation, the results of the EDA measurements showed themselves to be very robust no matter which method of measurement was used. The conclusion here is that the unit of measurement is more important for results than the method with which results are obtained (Sect. 2.6.5).

2.6.3 Using direct versus alternating current

As already mentioned at the beginning of Chapter 2.6, exosomatic EDA measurements are performed mainly using direct current. Even the simpler use of the alternating current measurement method (i.e., without recording information from the phase angle; Sect. 2.1.5) has only been realized to date in a small number of studies.

Both advantages and disadvantages exist in using either method. With AC recording, electrode polarization and error potentials are ruled out (Sect. 2.2.2.2), which play a subordinate role nowadays in any case, with the practically unpolarizable electrodes used (Sect. 2.2.2.3). Difficulties can arise when using AC with amplification and filtering (Sect. 2.1.4 & 2.1.5); that is, when the EDA signal itself contains higher frequency AC components, the elimination of possible noise during measurement and amplification is more complicated since the noise may be within the same frequency range as the desired signal.

It has already been shown that, at least for theoretical purposes, studies of interest in the electrical properties of the skin with inclusion of the phase angle are still in their infancy (Sect. 2.1.5 & 2.2.3.3). Newer systematic investigations have been to a large extent performed only by the Yamamoto group since the mid-1970s. These authors estimated values for the single resistances and capacitances in a simple model

[117] With .6 cm^2 Beckman Ag/AgCl electrodes, isotonic Hellige electrode cream (see Footnote 41 in Section 2.2.2.5), .5 V constant voltage, and 10 $\mu A/cm^2$ constant current. The subjects received 30 acoustic stimuli at intensities varying between 60 and 110 dB.

[118] In a study where 20 subjects were presented with 10 tones of 50 dB each and additionally, white quadrangles as stimuli. With the constant current measurements, Barry (1981) used polarizable electrodes and nonisotonic cream, while the constant voltage measurements were made using standard methodology.

of the skin (Fig. 18, Sect. 1.4.3.3) using mathematical derivations (Sect. 2.5.3.1 & 3.5.2.1; see also Millington and Wilkinson, 1983, p. 135f.). Other studies performed by Salter (1979), Faber (1980), Thiele (1981a) and Boucsein et al. (1989) are regarded, in contrast, more as paradigmatic methodological investigations.

A series of older results with AC recording were summarized by Tregear (1966). Additionally, but only published in German, studies into the area of work physiology were performed by Rutenfranz (1955) and Rutenfranz and Wenzel (1958, Sect. 2.4.1.1), during which both impedance and capacitance of the skin at frequencies from 500 Hz to 10 kHz were measured.

Common to most of these studies is that they cannot be directly compared with direct current studies because they do not use standard methodology. So, for example, Yamamoto et al. (1978) did not record from the palmar or plantar areas, using instead the forearm skin with electrodes of 3.14 cm^2 at 10 μA effective current. This method of measurement is presumably developed from the work examining changes in skin impedance under the influences of cosmetics done by these authors (Sect. 3.5.2.1). As to whether the information gained from recording the phase displacement provides valid parameters for the application of EDA measurement in the psychophysiological area is therefore still an open question and requires systematic investigations. As the necessary technology for measurement and evaluation is relatively expensive (Sect. 2.1.5), a widespread application of AC technique is not to be expected in the near future.

Thus, EDA measurement using AC represents to date a very interesting and promising theoretical area, but its practical application has not been investigated enough. Systematic comparisons between AC and DC current measurements are especially lacking, such as those that have been done in DC with constant current vs. constant voltage (Sect. 2.6.2). Systematic investigations of the theoretically interesting frequency dependency of the phasic components (i.e., SZR and SYR) have also not been performed. Examples for the time courses of single scores of R, X, G, and B calculated from the impedance and phase angle at a frequency of 100 Hz were given by Boucsein et al. (1989, Fig. 4).

2.6.4 DC versus AC coupling

When using an AC-coupled amplifier (Sect. 2.1.3), it must be kept in mind that the raw signal undergoes a mathematical differentiation; that is, the recorded EDA signal corresponds to the first derivation of the original signal as the value of the applied time constant becomes smaller (Edelberg, 1967). In an extreme case, therefore, a biphasic EDR could be created from a monophasic one by the use of AC coupling.

Obscuring the form of the EDR by differentiation is especially noticeable in rise times and recovery times (Sect. 2.3.1.3), but may also lead to narrowing of the amplitude. Edelberg (1967, Fig. 1.13) showed that a linear relationship between the original EDR amplitudes and those of the AC-amplified EDR exists even when intercorrelations are as low as .05. AC coupling has the following *advantages*:

(1) A much lower resolution is necessitated for EDR recognition as compared to DC coupling.

(2) Therefore, the necessary amplification factors and/or the recording ranges are smaller (Table 4 in Sect. 2.3.1.2.3).

(3) Owing to the self-regulating artificial zero line, continuous regulation by the experimenter is not needed, except when using a Wheatstone bridge circuit. This is especially advantageous with long-term studies.

By contrast, AC-coupling has the following *disadvantages*:

(1) The data have only interval scale levels (Sect. 2.1.3).

(2) The form parameters (Sect. 2.3.1.3) can only be evaluated with the help of special back calculation methods (Thom, 1988).

(3) When the EDL is not recorded additionally, no transformations of SR into SC units, and vice versa, are possible (Sect. 2.3.3.2 & 2.6.5).

(4) Special amplification problems can appear with performing a high amplification of a small section of the original signal (Sect. 2.1.4).

Barry (1981) pointed out that to date no systematic studies into the influence of AC coupling upon stimulus reaction relationships under differing amplification conditions have been made.

2.6.5 Resistance versus conductance units

The controversy on an adequate unit of measurement for exosomatic EDA dates back to the 1950s and is still going on. It was pointed out in Section 2.6.2 that unfortunately this discussion often remains confounded with the question of the choice of the method of measurement, that is, constant current or constant voltage.

According to Grings (1974), the question of the unit of measurement should be focused on mainly for three reasons:

(1) An attempt on theoretical considerations to find the most appropriate electrical unit, especially with regard to concepts of the electrical model of the skin.

(2) The statistically grounded choice of units with respect to desirable distributional characteristics for further treatment of the data (e.g., normal distribution) and independence of means and variances from each other.

(3) The search for relatively baseline independent EDR units, that is, such units where the EDR (as a measure of change) is not dependent upon the directly preceding EDL (Sect. 2.5.4.2).

It appears that the question of the adequate unit of measurement cannot be separately discussed from the underlying model concepts. This discussion started with simple resistance models, as shown in Figure 15 (Sect. 1.4.3.1) with the Montagu-Coles model, using parallel resistors connected in series with another resistor. The parallel resistors represent, in essence, single sweat gland ducts, which can be turned on or off according to the activity of the sweat glands. If a voltage is applied to such parallel-connected resistors, the total resistance cannot simply be calculated by adding the sum of the individual resistors (see Equation 6e). The reciprocal values of the single resistors (i.e., their conductance values) are added according to Equation (6d), resulting in the reciprocal value of the total resistance (Sect. 1.4.1.2). The total conductance is therefore calculable from the sum of the conductance values of the single elements. The change of the total conductance with an EDR would be in this case proportional to the sum of conductances of the elements, which is not true for the change in resistance.

The results from a study by Thomas and Korr (1957) are often used as evidence for a linear relationship between the conductance and the number of active sweat glands (Sect. 1.5). However, the experiment in question was performed with dry electrodes and heated skin, so that the upper layers of the stratum corneum had been dried out, and no contact existed between completely filled sweat gland ducts and the electrode. Therefore, no plain use can be made of these results for usual EDA measurements where the skin is not artificially dried and, in addition, moist electrode cream is used with the electrode. Blank and Finesinger (1946) showed that the sweat glands display graded reactions to neural impulses of differing frequencies. When the normal case is taken into regard, where the skin surface and thereby the electrode come into electrical contact with less filled sweat gland ducts (e.g., through the fully moisturized corneum), the simple supposition of the all-or-nothing principle of connected parallel resistances no longer helds. Instead, the resistance resulting from sweat gland activity depends much more upon the degree of duct filling. Since in this case the relationship between the decrease of the resistance and the height of the duct filling is linear, resistance and not conductance would be the adequate measure.

As discussed in Section 1.4.2.3, the secretional processes of the sweat glands are far too slow to be the only cause of the relatively quickly changing EDR signal. Therefore, Edelberg (1971) presumed that only the slow fluctuations of EDA may be attributed to duct filling, while EDRs with shorter recovery times should be attributed to changes in permeability of the sweat gland membranes. Hence, the above discussion on resistance is not of basic importance to the EDR. However, for parallel-connected membranes which behave like capacitors, a simple addition of the single capacitances is valid (Sect. 1.4.1.2), and thus conductance is an adequate measure for the EDR. Hence, contrary demands exist for the measurement unit of choice for the slow and fast components of EDRs. In addition, the differential dependence from the respective EDL must be noted for comparison between SCR and SRR (Sect. 2.5.4.1).

To date, modelling of electrodermal phenomena does not allow a theoretical answer to the question of adequate units of measurement. When the argument that con-

ductance is more "physiological" (which in fact relates to the method and not to the unit of measurement; Sect. 2.6.2) is not considered, the question of the adequate unit of measurement can only be answered empirically. Distributional characteristics and level dependencies can serve here as criteria, and the validity of the different units of the obtained parameters should also be considered.

Although a conversion of resistance units into conductance units, and vice versa, is easily done when EDL scores are available (Sect. 2.3.3.2), only a few empirical studies comparing different units of measurement for EDA have been done to date. Lader (1970) blocked the activity of the sweat glands on the fingers with iontophoretic application of atropine and subsequently recorded SRRs in response to a series of stimuli. It took up to 40 min before the effect of the atropine had advanced so far that no more SRRs could be observed. When the SRRs were transformed into SCRs, an exponential decrease in the amplitudes was observable. By contrast, the increasing atropinization effect showed an irregular influence on the nontransformed SRR amp. This study is often cited as empirical proof for the preponderance of conductance units instead of resistance units.

Using SC data they had obtained from 28 subjects, Hölzl, Wilhelm, Lutzenberger, and Schandry (1975) showed that a transformation of the SCR scores and the SCL scores into units of resistance (i.e., into SRR and SRL units) led to a better matching of normal distribution than with the original SC data. According to these results, transformations of resistance scores to conductance scores for normalization (Sect. 2.3.3.3) did not need therefore to be done.

Boucsein et al. (1984a) made a systematic comparison of SCR and SRR amplitudes which were obtained from 60 subjects through parallel constant voltage and constant current measurements (Sect. 2.6.2); these scores were additionally transformed into each other while taking into account the respective EDLs. It was found that with high levels of stimulus intensity (i.e., with white noise exceeding 90 dB) the SRR and SCR amplitudes were clearly differentiated in their habituation, whereby the direction of the results (habituation or sensitization, Sect. 3.1.2) was dependent upon the respective unit of measurement and not on the method of measurement originally applied. Through the proper transformation of the amplitudes of both units with the use of the mean level changes throughout the experiment, the differing courses could be uncovered. Then the equivalence of both methods of measurement could be shown; however, a recommendation for one or the other method could not be made, owing to a missing validity criterion.

Bitterman and Holtzman (1952) reported finding a differential level dependency of SRR and SCR amplitudes similar to that found by Boucsein et al. (1984a); the above two authors recorded the EDA from 40 subjects with a constant current technique and investigated the level dependency of raw data and of data transformed to conductance units. For the first stimulus of the extinction phase, they found a significant negative correlation of the SCL with conductance units, while a corresponding level dependency

for resistance units did not appear. Therefore, the authors decided to report their results in resistance units.

The question of choice between units of conductance or resistance for exosomatic EDA appears to be mostly an academic one. On one side, the use of conductance units provides a standardization, while on the other side, not enough empirical proof yet exists to fulfill the above-mentioned three criteria as outlined by Grings (1974) and to decide the issue. Although the whole matter can be at best pragmatically solved in favor of conductance units as an attempt for standardization, theoretical as well as empirical questions remain to be resolved with respect to the adequate unit of electrodermal measurement.

Part 3: Applications

The third part of this book is dedicated to various applications of EDA measurement. The aim is to provide a theoretical framework for the use of the different EDA parameters described in Part 2 as psychophysiological indicators in the appropriate fields. Since there are thousands of articles reporting EDA results (Sect. 1.1.3), their comprehensive description would go far beyond the limits of the present volume. Instead, the focus will be on giving more detailed information especially for studies which enlighten either methodological issues or provide support for interpretation of results in light of psychophysiological theories related to EDA. The scope of applications will be mainly restricted to those areas where considerable developments in the use of EDA measurement have taken place during the last two decades.[119]

As already mentioned in Chapters 2.4 and 2.5, the term "standard methodology" will be used for EDA measurements in accordance with the standards as outlined in Section 2.2.7 (see also Footnote 63, Chapter 2.4). Methodology seems a most crucial point in fields of application outside laboratory psychophysiology. It is the author's hope that this book will stimulate the use of these standards in different fields of applied research within as well as outside psychology.

3.1 Stimulus-related psychophysiological paradigms

With respect to the preponderance of either phasic or tonic parameters, the scope of psychophysiology may be divided into parts, focusing either on responses to distinct stimuli or on physiological parameters as indicators of changes for more general states. While the second kind of paradigm is dealt with in Chapter 3.2, the present chapter's focus is on electrodermal concomitants that appear during stimulation and information processing.

As a consequence of the widespread use of EDA parameters in orienting, habituation, and conditioning research, the respective results have been summed up by different authors (cf. the appropriate contributions in the readers of Prokasy and Raskin, 1973; Kimmel, van Olst, and Orlebeke, 1979; Siddle, 1983; and Gale and Edwards 1983). Therefore, Sections 3.1.1 and 3.1.2 will be restricted to the extraction and use of appropriate EDA parameters, while reporting some studies as typical examples for parametrization in the different contexts. Specific regard is given to more recent views on the role of EDA in information processing, as outlined in Section 3.1.3.

[119]For summaries of older results see Prokasy and Raskin (1973) and Edelberg (1972a). More recent summaries are referred to within the specific sections.

3.1.1 Electrodermal indices of orienting and habituation

The concepts of orienting and habituation are widespread in psychophysiology. A short outline of the most important theories, for example, Sokolov's (1963) "neuronal model," the two-process theory formulated by Groves and Thompson (1970), and Wagner's (1976) so-called priming theory, is given by Stephenson and Siddle (1983).

The orienting response (OR) was first discovered by Pavlov (1927) as a reflex elicited by environmental change, the biological aim of which is to let the organism turn toward the source of stimulation in order to analyze its content or meaning. The components of an OR are lowering of sensory thresholds, pupillary dilation, eye and gross body movements, blockade in the EEG, as well as various vegetative changes, the most prominent of which is an EDR, together with a deceleration-acceleration pattern in heart rate (HR), cephalic vasoconstriction, and finger vasodilation.

Since the OR is unspecific, it is elicited by both an increase and a decrease of stimulus intensity, hence, not only following stimulus onset but also the end of stimulation. Different kinds of ORs may be distinguished:

(1) Generalized vs. localized OR (Sokolov, 1960; Lynn, 1966). A generalized OR is characterized by a general activation of the sensory cortex as well as by an increase of sensitivity in various sensory systems. By contrast, a localized OR is restricted to the stimulation of a specific system, and therefore does not fully meet the criterion of nonspecifity typical of the OR. Despite this lacuna, it is classified as an OR by Sokolov (1963) because of its nonspecificity with respect to the direction of change. There are also noticeable differences in habituation speed; the generalized OR typically habituates within 2–5 trials, while the localized OR needs 20 or more trials to habituate (Sect. 3.1.1.3). Examples for localized ORs are long-lasting EDRs following tactile stimuli and the occipital blockade due to visual stimulation.

(2) Phasic vs. tonic OR (Sokolov, 1963, 1966). A phasic OR can be traced back to a transient increase of sensitivity of the different receptor systems. The term *receptor system* is used here as an equivalent to Sokolov's (1963) "analyzer," which is an integrating system of peripheral as well as central mechanisms fundamental for transmission and processing of stimulus properties. Within the electrodermal system, the stimulus-related EDR is regarded as the phasic OR component. By contrast, the tonic OR consists of a kind of level-shift in the background sensitivity of the receptor systems which may even continue after the end of an habituation series. Whether a phasic or a tonic OR appears is presumed to be dependent on the overall cortical arousal level (Sect. 3.2.1.1). A novel stimulus may cause a tonic OR lasting up to an hour in a drowsy subject while eliciting only a phasic OR in an awake person. According to Sokolov, the increase of tonic EDA following stimulation is a typical indicator for a tonic OR, which may be an increase in SCL and/or in NS.EDR freq. (Sect. 2.3.2).

A further differentiation between involuntary and voluntary ORs has been made by Maltzman (1979a); the latter are cognitively mediated (Sect. 3.1.1.1) while involuntary ORs are solely elicited by stimulus characteristics, thus habituating faster. Barry and O'Gorman (1987) pointed to EDR lat. as being significantly longer with voluntary ORs (e.g., to stimulus omission) than with involuntary ORs.

After having habituated, an OR reinstatement follows changes in stimulus intensity, modality, duration, frequency, sequence (i.e., duration and variability of the interstimulus intervals), complexity, information content, or stimulus significance which may be acquired during classical conditioning (Sokolov, 1960; see Sect. 3.1.2.1). OR reinstatement appears during below-zero habituation[120] and also during dishabituation. Dishabituation means an increase in EDR amp. following a stimulus, within the current habituation series, which has been preceded by a novel stimulus not belonging to the series. Thompson and Spencer (1966) argued for high intensities as a requirement for dishabituation stimuli. However, dishabituation may also be elicited by changes in stimulus modality, by frequency changes of acoustic stimuli, and by stimulus omission (Magliero, Gatchel, & Lojewski, 1981; McCubbin & Katkin, 1971; Edwards & Siddle, 1976; Martin & Rust, 1976; Siddle, Remington, Kuiack, & Haines, 1983c).

Another stimulus modality, the dishabituating effect of which has been stressed by Berlyne (1961), is conflict, or the possibility of choice between several reactions. In addition, during the 1970s there was a controversy concerning the role of stimulus significance between Bernstein (1979) and Maltzman (1979a), on one side, and O'Gorman (1979), on the other. They discussed the role of the subject's personality characteristics, individual experience, mood, motivation, state of activation, as well as various other contextual conditions. However, the main issue was the importance of cognitive factors in explaining individual differences in OR. These factors have been especially stressed by Bernstein, who regarded the OR mainly as a consequence of the stimulus-input's cortical evaluation, including the subject's above-mentioned individual characteristics. By contrast, O'Gorman regarded individual OR differences as being due mainly to different degrees of readiness in the peripheral physiological systems, giving as a typical example the dependence of the electrodermal OR upon the spontaneous EDA (Sect. 2.5.4.2).

3.1.1.1 EDR as an indicator of orienting responses

As compared to other physiological indicators of an OR, the phasic EDA emerges as a most suitable correlate of stimulus intensity. For example, a linear relationship between SCR amp. and the intensity of 2 sec white noise stimuli between 70 and 100 dB[121] has been found in 12 subjects by Uno and Grings (1965).

[120] Below-zero habituation represents a continuation of stimulus presentation after reaching an individual habituation criterion, e.g., two subsequent "zero" reactions (Sect. 3.1.1.3).

[121] In steps of 10 dB, each stimulus applied five times in a balanced design, SR recording with 50 μA constant current using 2 cm^2 Ag electrodes from two fingers of the right hand, transformed to SC.

Jackson (1974) also found a monotonous increase of the SCR amp., averaged over the first 4 as well as over all 10 trials, during an increase in intensity of a 1 kHz tone,[122] while the phasic HR showed an increase-decrease-increase change with increasing stimulus intensity. Barry (1975), varying the intensity of a 1 kHz tone between 20 and 50 dB in 10 dB steps, which were presented in permuted order to 24 subjects, also found an approximately linear increase of the SRR amp. with an increase of stimulus intensity.[123] In a study using the same experimental conditions, EEG power remained nearly constant up to an intensity of 40 dB, only showing an increase under the 50 dB condition (Barry, 1976). Turpin and Siddle (1979), as well as Boucsein and Hoffmann (1979) in their studies described below and in Section 2.5.2.1.1, also demonstrated a positive linear relationship between stimulus intensity and the EDR amp.

A great number of investigations have been performed with respect to the influence of the stimulus significance or "salience" on the electrodermal OR's strength and course of habituation. It can be regarded as a generally accepted result that the OR following a certain stimulus or a class of stimuli can be enlarged if those stimuli are given signal value for a cognitive or a motor reaction (Maltzman & Langdon, 1982; Spinks & Siddle, 1983)[124]. Maltzman and Langdon (1982) attempted to separate the influences of novelty and significance on the electrodermal OR. One hundred and twelve subjects of both genders were presented .5 sec 1 kHz 70 dB tones. The interstimulus intervals (ISIs) between the tones was 12 sec during the presentation of the initial 16 training stimuli, and varied between 5.5 and 26.0 sec in logarithmic steps during subsequent test series. Half the subjects served as the experimental group and were instructed to lift their foot as fast as possible from a pedal, thus imposing significance on the stimuli, while the control group was instructed to sit quietly and listen. The experimental group showed significantly greater EDR amp.,[125] while the control group showed faster habituation. The control group reacted only to the 26 sec interval with a significant increase of the EDR amp. with respect to the training phase. On the other hand, in the experimental group which was more reactive overall, only the difference between the 5.5 sec ISI and the training interval approached significance. The authors concluded that stimulus significance is not a necessary condition for the occurrence of an OR. Instead, it may predetermine the effects of novelty to some degree. Without referring to a specific theoretical model, the authors offered global concepts like "dominant focus" or "cortical set," as already stated by Maltzman (1979a), for an explanation of their results; however, they did not offer predictions for further research in this area.

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[122]In steps of 20 dB; SCR recorded with standard electrodes and voltage, however, unipolar thenar against a neutral forearm side and with KY-gel (Experiment 3).

[123]Recorded AC-coupled (Sect. 2.1.3) with a 5 sec time constant volar from the left hand's fingers.

[124]Rotenberg and Vedenyapin (1985) in a study with 15 subjects who were presented a series of tones, a subset of whom had to react, found some evidence of SPR amp. to these tones being more dependent on decision making than on motor preparation.

[125]Recorded as SRR palmar, .5–5.0 sec after stimulus onset, and subjected to a logarithmic SCR transformation.

The same question was investigated by Barry (1982) in two experiments; he introduced stimulus significance by cognitive demands instead of requiring motor reactions. In the first experiment, two groups of 10 subjects (male and female) were given a series of seven stimulus pairs (capital letters A and B in random order). The experimental group had to count the Bs, thus giving them significance, while the controls counted every letter. There was a significantly enhanced mean SCR amp.[126] in the experimental group. In the second experiment, the procedure was repeated with the same number of subjects, followed by another seven stimuli with different relations of As to Bs to induce novelty. Unfortunately, these were different in both groups, which confounded task difficulty and group effects, thus invalidating, in part, the interpretation of the observed significant increase of SRR amp. during the eighth trial as a consequence of novelty. Altogether, the EDR amp. appeared to be influenced by both stimulus characteristics and cognitive processes. However, among all psychophysiological variables recorded, only EDA reflected manipulations of significance.

Recently, Ben-Shakhar, Asher, Poznansky-Levy, Asherowitz, and Lieblich (1989) reported results from three experiments investigating the effects of stimulus novelty and significance. In the first experiment, with a total of 108 subjects of both genders, nonsignificant test stimuli were introduced at different positions within a complex eight-stimulus series.[127] The test stimuli did not produce any enhanced SCR (measured with standard methodology) under either condition. In the second experiment performed with a total of 128 subjects, a sequence of simple standard stimuli were introduced at the same positions, yielding greater SCRs, contrasting with Sokolov's neuronal model (Sokolov, 1963). The goal of the third experiment using 128 subjects was to directly compare the response to a nonsignificant stimulus change with that to a similar stimulus change including a significant element. Therefore, prior to its presentation, the test stimulus was made relevant in one condition by the use of the so-called Guilty Knowledge Technique (Sect. 3.5.1.2). However, the attempted effect of significance had probably been obscured by the nature of the test stimulus,[128], thus, effects of novelty and significance could not be clearly separated. Both factors were regarded by the authors as nonadditive, and instead of viewing significance as a necessary condition for an OR, it was suggested that stimulus significance is only necessary when the contrast between test and standard stimuli is small (i.e., when the test stimulus follows a complex sequence of stimuli).

[126]Measured with stainless steel 2 by 3 cm electrodes from the volar surface of the left hand's fingers with Biocom Inc. Biogel as contact medium; 1–5 sec following stimulus onset was the time window used.

[127]Stimuli were either one-word (name of an occupation) or two-word stimuli (occupation name combined with name of a hobby), both kinds used as standard and as test stimuli as well. The subjects had to recall as many words as possible. For evaluation, Receiver Operating Characteristic (ROC) curves were generated by comparing the distributions of standardized responses to test stimuli vs. standard stimuli.

[128]A name of a hobby instead of a certain number, as used in an earlier experiment (Ben-Shakar & Lieblich, 1982) having had a clear common component shared with the standard stimuli (which were also numbers).

Janes (1982) reported some evidence that electrodermal recovery is a valid indicator of stimulus significance, showing an additional effect with respect to EDR amp. as suggested by Edelberg (1970). Ten subjects of both gender were delivered 76 tones (800 Hz, 75 dB) after 16 habituation trials in a within-subjects design. Combinations of delivering the tone to the right or left ear in a distinct sequence were used to induce stimulus meaning (they either prepared to press a foot pedal, or did not, at the onset of the next tone) with identical stimulation. SCR amp.[129] was greater and SCR rec.t/2 was longer when stimulus meaning increased, both measures being significantly correlated in 9 out of 10 subjects.[130] However, the recovery time increase held up under two types of amplitude correction applied both as an individual regression procedure and as an inspection of amplitude-matched trial pairs. Therefore, Janes (1982) concluded that stimulus significance may affect EDR recovery to an even greater degree than its amplitude, and both peripheral as well as central mechanisms are involved in determining both parameters (Sect. 1.4.2.3).

A differentiated view concerning the role of EDR as an indicator of OR is provided within the multiprocess OR model proposed by Barry (1987). This model assumes different registers or systems interacting during an OR, which are represented in various physiological measures. These are: a stimulus register, as indicated by HR deceleration and cerebral pulse volume; an intensity register, the indicators of which are EDR and peripheral pulse volume; a novelty register, being indicated by EDR, respiration break, and EEG-α; and the response system, the activation of which is accompanied by HR acceleration.

3.1.1.2 EDR in differentiating orienting from defensive reactions

The OR is hypothesized to exert an important survival function, since it shifts the organism to a state in which its resources are mobilized for an adequate reaction to changed or new stimulation. If stimuli being non-threatening or even unimportant to the organism are repeated, OR amplitude decreases as a sign of an adaptation process, which shows up as habituation of the OR. However, highly intense or even aversive stimulation requires a different reaction pattern, which indicates a continuous readiness for reaction.

Pavlov (1927) already distinguished between the OR and the so-called defensive reaction (DR), the biological aim of which is to protect the organism against harmful stimulus properties. As a consequence, the DR does not show considerable habituation. In addition, a DR never appears at the end of stimulation as may the OR. According to Sokolov (1963), OR and DR can be distinguished by their vasomotor components: peripheral vasoconstriction and cephalic (forehead) vasodilatation in cases of an OR,

[129]Recorded bilaterally from the hypothenar eminence with standard methodology, using Beckman-miniature electrodes, logarithmically transformed.

[130]Bundy's "X" (Sect. 2.5.2.5, Equation 46) and SCR rec.t/2 were significantly correlated for 3 of the 10 subjects.

versus constriction at both sites during a DR. Lacey (1967) described differentiated HR patterns: a biphasic deceleration-acceleration pattern typical of an OR versus a monophasic acceleration pattern indicating a DR.

Another distinction has been made by Dykman, Reese, Galbrecht, and Thomasson (1959) between OR and startle reaction based on their concomitant body movements; they are directed in the OR but not in the startle case. Also, the latency time is shorter for the startle reaction. However, EDR latencies cannot fall below a threshold of .5 sec (Sect. 2.3.1.1). According to Graham (1979), the startle reaction habituates quickly, as opposed to the DR. On the other hand, Turpin (1986) claimed that the startle reaction was an early component of a DR, elicited by stimuli with short rise times.

Several EDA parameters have also been used to distinguish between OR and DR. As Uno and Grings (1965) showed in their study mentioned in the previous section, the number of biphasic SPRs[131] increased when stimulus intensity approached 100 dB. In addition, the amplitudes as well as the rise times of the SPRs and the SCRs recorded in parallel increased with stimulus intensity. Using a broader range of stimulus intensities,[132] Raskin, Kotses, and Bever (1969), found only small differences in SPR and SCR amp. when comparing the different intensities. However, they found the positive SPR component markedly increased with stimulus intensity, thus interpreting the positive SPR component as an indicator for DR and the negative SPR component as an indicator for OR. Edelberg (1970) questioned their conclusion, since he found a connection between the positive SPR component and a prolonged SCR rec. t/2, which he later proposed as an index of aversive stimulation (Edelberg, 1972a).

Another attempt to differentiate OR from DR by means of EDA assessed the difference between palmar and dorsal recording. Older studies performed by Darrow (1933) and by the Edelberg group (for a review see Edelberg, 1972a) pointed to a differential reactivity of these sites, the dorsal being more likely to reflect OR, while the palmar EDA seemed to be more closely linked with DR or anxiety reactions (Sect. 3.4.1.1). Possible sources of the so-called palmar/dorsal effect were to be seen in the differences in palmar and dorsal surfaces with respect to sweating (Sect. 1.3.2.4), as well as the greater sweat gland density in the palmar ridged skin as compared to the dorsal polygonous skin (Sect. 1.2.2 & 1.2.3). Based on his two-component model of EDA (Sect. 1.4.2.3), Edelberg (1973) suggested that the predominance of the sweat gland component over the epidermal component is responsible for the greater SCR amp. and the prolonged SCR rec.t/2 at palmar as compared to dorsal sites during aversive stimulation. This is because only the membrane component, which has a short recovery time, plays a considerable role in the dorsal EDR (Sect. 1.4.3.2).

[131]Recorded between an active palmar and an inactive wrist site with Ag electrodes 2.6 cm in diameter.

[132]Between 40 and 120 dB in steps of 20 dB, with independent groups of 25 subjects each. Endosomatic EDA was recorded from left thenar against treated forearm sites, while exosomatic measures were taken as SR from the right hand palmar vs. dorsal, with 40 μA, being transformed to SC values, using Beckman Ag/AgCl electrodes with NaCl paste for both measures.

Some evidence for the suggested greater complexity of the palmar as compared to the dorsal EDR comes from a study by Sorgatz (1978), with 80 subjects performing a monotonous task with relatively low requirement for muscular activity. The cross-product matrix of the SZRs[133] from all 24 trials was factor analyzed. Of the dorsal SZR variance, 80% could be explained by one component, while two components were necessary to explain the same portion of the palmar SZR variance. Using the same recording technique, Sorgatz and Pufe (1978) recorded palmar and dorsal SZR from 36 subjects during aversive and neutral stimulation; electric shocks as well as slides displaying skin diseases were the aversive stimuli, while light flashes as well as cartoon slides were neutral stimuli. The stimuli were presented in permuted sequence with ISIs being either 10 or 20 sec. Under the electric shock condition, as compared to light flashes, anticipatory palmar SZR amplitudes were increased, while dorsal amplitudes were higher in reaction to the shocks. However, inverse palmar/dorsal relationships appeared under the slide conditions, thus rendering a simple interpretation in terms of OR vs. DR untenable.

A possible differential indicator function of SCR recovery may be its prolongation in cases of DR as compared to OR, which could be inferred from Edelberg's (1973a) considerations (independently from palmar vs. dorsal sites). However, this has not yet been demonstrated. Using 1 kHz tones of five different intensities between 45 and 105 dB[134] presented to 15 subjects each, Turpin and Siddle (1979) showed a linear increase of SCR amp. following the first tone with increasing intensity. However, there was no unequivocal relationship between the inverse of SCR rec.t/2 following the first tone and its intensity. It was only in the 7th and 9th trial of the 15-trial habituation series that the recovery time following the 105 dB stimuli was markedly longer than those following the lower-intensity stimuli (cf. Turpin & Siddle, 1979, Fig. 1). There was also no systematic relationship between SCR ris.t. and stimulus intensity. One reason for the absence of differentiation between DR and OR by means of EDR parameters in this study may be that the stimulus rise times used could have been too long to elicit a DR. In his study described in Section 3.4.1.2, Hare (1978a) found SCR rec.t/2 increasing with stimulus intensity only with 10 msec but not with 25 msec stimulus rise times,[135] especially when using 100 dB and above. Since he obtained this relationship mainly in left-hand recordings, the question of a possible lateralization of electrodermal time parameters with respect to DR is raised (Sect. 3.1.3.4). Boucsein and Hoffmann (1979), in their study described in Section 2.5.1.1, reported significant main effects of stimulus intensities in logarithmized SCR rec.t/2 as well as SRR rec.t/2, the recovery times being markedly prolonged following 100 and 110 dB white noise stimulation of

[133]Recorded with 32 Hz and 8 μA via 1 cm^2 Ag electrodes, using paper soaked with .5% NaCl as an electrolyte, dorsal from the 1st and 3rd finger, and palmar from the 2nd and 4th finger of the nondominant hand.

[134]In 15 dB steps, 30 msec stimulus rise time, and 2 sec duration; EDA recorded with standard methodology, using KY-gel.

[135]1 kHz tones between 80 and 120 dB in steps of 10 dB.

2 sec duration with instantaneous rise time. In the same study, ORs and DRs appeared to be differentiated by their course of habituation, which will be discussed in the following section.

3.1.1.3 Electrodermal indices of habituation

According to the classical definition given by Humphrey (1933) and Harris (1943), habituation is characterized by decreasing reaction intensity with repeated stimulation. More recently, habituation has also been regarded as the most elementary form of learning (e.g., Thorpe, 1969; Petrinovich, 1973) which may be reflected in different ways in various variables and/or parameters (Siddle, Stephenson, & Spinks, 1983b). With respect to this view, a conceptual distinction must be made between extinction (Sect. 3.1.2.1) and habituation, since the OR which habituates cannot be regarded as an outcome of learning (Sokolov, 1963).

Among the psychophysiological indicators of habituation in human subjects, the EDR is the one most frequently used.[136] This may be due to the fact that a stimulus-dependent EDR is easily detectable and decrease of its amplitude over trials can be followed even with visual inspection. At first glance, the time point at which the reaction is completely habituated also seems to be simply determined by the disappearance of the EDR. However, criteria for "zero reactions" depend on properties of the measuring device (e.g., the amplifier's signal-to-noise ratio; see Sect. 2.1.4) and on the resolution chosen for registration, thus requiring the definition of an appropriate minimal amplitude criterion (Sect. 2.3.1.2.3).[137]

As already pointed out in the previous section, a repeated presentation of stimuli eliciting a DR should not lead to habituation. Groves and Thompson (1970) pointed to a hypothetical sensitization process underlying the phenomenon of a delayed or missing decrease of reaction strength with repeated stimulus presentation. In their two-process theory of habituation, these authors regarded the course of reaction amplitudes over trials as a result of interacting habituation and sensitization processes, which may also lead to an increase over trials with overwhelming sensitization. However, Siddle et al. (1983b) preferred the term "dishabituation" instead of "sensitization," since it does not require assumptions concerning unknown underlying neuronal processes.[138] Factors

[136]For example, the studies being conducted to develop the theory of the "neuronal model" (Sect. 3.1.1) used EDR as the main physiological indicator (e.g., Sokolov, 1963).

[137]Providing such an amplitude criterion is also of fundamental significance for investigations into the so-called below-zero habituation (Thompson & Spencer, 1966), which also requires consideration of the sensitivity of the physiological system in question (Stephenson & Siddle, 1976). However, there is only weak evidence from human studies for an influence of the duration of below-zero habituation on the spontaneous recovery of an OR after being habituated (i.e., OR reinstatement; Siddle et al., 1983b).

[138]There are only few data from human studies supporting the Groves-Thompson theory, which has been developed by research on decerebrated cats. Hölzl et al. (1975, Fig. 12), in their study described in Section 2.6.5, observed three types of SCR courses in their 28 subjects. Most of them showed an

which cause or modify the dishabituation phenomena have not been sufficiently studied (Siddle & Hirschhorn, 1986).

The fact that the electrodermal DR component does not show habituation still remains debatable. There is some evidence from the study of Boucsein et al. (1984a, Fig. 3) in which data taken from the Boucsein and Hoffmann (1979) experiment (Sect. 2.5.2.1.1) was reanalyzed, that SCR amp., as well as level-dependency-corrected SRR amp. yield a marked sensitization effect to 110 dB white noise stimuli.[139] Sensitization effects could be clearly shown with a group that received a series of 110 dB white noise stimuli of 2 sec duration without warning signals in the Baltissen and Boucsein (1986) study which will be described in detail in Section 3.1.2.1. Turpin and Siddle (1979), in their study described in the previous section, also observed an initial delay of habituation to their 105 dB white noise stimuli. However, the SCR amp. habituated during the 15-trial series. Therefore, the recommendation given by Walrath and Stern (1980) should be followed to quantitatively differentiate between the course of habituation for OR and DR, instead of making the initial distinction between habituating and nonhabituating reactions. However, quantification of EDR habituation is less unequivocal as it seems at first glance. Figure 45 shows how the use of different habituation indices may lead to divergent results.

As pointed out in the beginning of this section, the two concepts of habituation have implications for an appropriate quantification (Schandry, 1978):

(1) If habituation is regarded as change (i.e., decrease in reaction over time), the parameter of interest is the course of habituation, which is, for example, quantified by the steepness of decrease.

(2) If, on the other hand, habituation is regarded as "learning not to react," one will be interested in determining the end of habituation process, and the time (or number of trials) is determined, which is needed before no reactions are observed.

Figure 45 illustrates how the forms of quantification may lead to contrasting results though using the same data. In the following, the most frequently used EDA parameters for determining either course or end of the habituation process are provided. Further electrodermal habituation indices are described by Ben-Shakhar (1980) as well as by Hiroshige and Iwahara (1978).

There are three different types of indices with which the process of habituation can be described:

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exponential decrease over trials, a few "sensitizers" showed a slight increase, and some other "initial sensitizers" showed an increase followed by a considerable decrease.

[139]This result gives rise to the hitherto unanswered question of the adequate unit of measurement in EDR habituation studies (Sect. 2.6.5). However, the habituation effects in this study may have been obscured by the fact that a permuted presentation of different stimulus intensities was used. This is different from standard habituation series, and may have contributed to the marked dishabituation effects observed with the high-intensity stimuli.

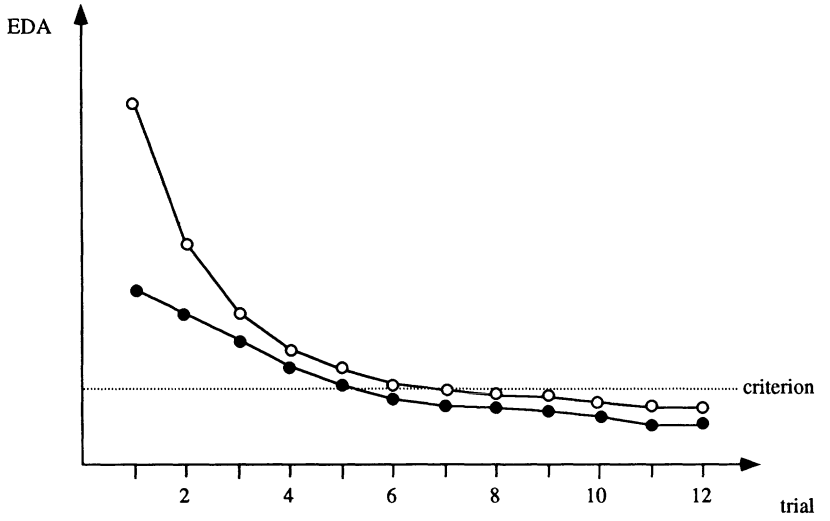

Figure 45. Results of the use of different habituation indices. Parameters of the habituation course yield a fast habituation for the empty dots, and a slow one for the solid dots. Parameters which determine the end of the habituation process by the use of a minimal amplitude criterion would yield a contrasting result. From R. Schandry (1978), *Habituation psychophysiologischer Größen in Abhängigkeit von der Reizintensität*, Fig. 3. Copyright ©1978 by the author. Reprinted by permission.

(1) Regression indices using the slope of a curve that fits the empirical data, which is either a straight line or an exponential of habituation rate, the most well known of which is Lader and Wing's (1966) H score (see Equation 51).

(2) Amplitude scores using the EDR amp. at a given point of the habituation curve for an ANOVA procedure (Siddle et al., 1983b). Alternatively, mean amplitudes or magnitudes (Sect. 2.3.4.2) of the whole habituation series are used as amplitude scores (O'Gorman, 1977).

(3) Difference scores describing differences between the EDR amp. at two or more points within an habituation series, for example, the difference between the mean EDR amp. of the first and the last trial block[140] (Koriat, Averill, & Malmstrom, 1973). Frequently, those difference scores are formed by using interaction terms from ANOVA (O'Gorman, 1977).

[140] Many authors combine two or more trials forming a trial block when reporting habituation courses.

Since the course of habituation can be normally described by a negative exponential function with respect to trial number (Thompson & Spencer, 1966), a decadic log transformation of the abscissa should be performed before forming regression indices as mentioned under (1), to obtain a linear relationship between number of trials and EDR amp. Afterwards, a regression of the EDR amp. on the log trial number is calculated starting from trial number two. There are two indices to be formed from this regression equation (Montagu, 1963); the slope of the regression line b, and the the y-intercept a. Typically high negative correlations are obtained between a and b; subjects having larger EDRs during the first trials usually show a higher absolute value of the slope b, which is used as an habituation index.

To obtain an habituation index independently of the initial EDR, the slope b has to be corrected with respect to the intercept a, which again is performed by the use of a regression equation (51), the result of which is labelled as b' by Montagu (1963) and as H by Lader and Wing (1966):

$$H = b - c\,(a - \bar{a}) \tag{51}$$

where b is the individual regression slope of the reaction strength plotted against trial numbers, a is the individual intercept, $\bar{a}$ is the mean of the intercepts across all subjects, and c is the regression of the slope b on the values of a. The index H is an estimation of the absolute rate of habituation, assuming that the y-intercept is a constant (e.g., comparable to a sample mean). The appropriate calculation therefore corresponds to the use of covariance analytic techniques.

Koriat et al. (1973) pointed out that the calculation of b' values is only justified if different processes can be assumed underlying a and b. Hence, their intercorrelation can be regarded as due to an artifact. In addition, when using experimental groups differing a priori in the y-intercept a, the covariate, the regression measure according to Equation (51) may be obscured. Overall and Woodward (1977) deliberately suspended applying analysis of covariance procedures in cases where a significant correlation between an experimental condition (or an organismic variable like age) and the covariate (the y-intercept a) is to be expected.[141]

If the course of habituation is described using amplitude scores as mentioned under (2), differences in habituation speed or in habituation rate are determined by means of the interaction between experimental condition and trial number. Therefore, individual measures of habituation speed cannot be obtained by this type of index. Instead, it

[141]For example, if significantly smaller values for a as well as for b are observed in a group with older subjects as compared to a younger group, and if the age effects on the ordinate value a and on the gradient b are independent from each other as well as from the correlation between a and b, the correction of the gradient b according to Equation (51), taking regard of the ordinate value a, will lead to a reduction of the "independent" age effect on gradient b. In general, it must be regarded as difficult to determine the relative influence of experimental conditions or organismic variables like age on values of a and b. Therefore, if those conditions have a significant influence on a, corrections according to Equation (51) should be avoided.

enables the comparison of effects for different experimental conditions on the course of EDR amp. over trials. However, that particular technique becomes insensitive with an increasing number of trials, since all experimental groups reach stable low EDR amp. with a large number of trials. Hence, this problem may be circumvented by including only the first part of trials in ANOVA, which, however, requires an a priori formulation of reasons for that particular choice.

An amplitude measure sometimes used to quantify the amount of habituation is the mean EDR amp. or – if "zero reactions" are included – the mean EDR magnitude (Sect. 2.3.4.2), averaged over all trials (Siddle & Heron, 1976; Vossel & Roßmann, 1982). Such a measure does not provide any specific information concerning the change of EDR amp. over time. It is simply assumed that reactions showing a smaller EDR amp. or "zero-reactions" during late trials contribute to an overall smaller mean amplitude or magnitude. However, subjects showing rather low EDR amp. during the first trials will also yield a strong habituation index, even if they showed an increase in EDR amp. over trials. Therefore, an overall mean amplitude or magnitude has to be questioned seriously as a particular index of habituation. This is also pointed out by Siddle et al.(1983b), who state that the average-magnitude measure confounds individual differences in amplitudes and habituation course.

Calculating mean amplitude scores is further questioned by results of several studies. Siddle and Heron (1976) obtained only relatively low 3–5 month reliabilities (between .26 and .66) of those scores, and correlations to a criterion measure of habituation were as low as –.06 and –.23, those to the H score being not higher than .08 to .47.[142] In turn, high positive correlations between .63 and .76 were obtained with the ordinate value a of the regression curve. Vossel and Roßmann (1982) also reported a correlation of .86 between a and the mean EDR magnitude. As opposed to Siddle and Heron (1976), they obtained a positive correlation of .32 to an habituation criterion measure; however, they also obtained a similar correlation of .46 to the H score. The relatively high correlations between the mean EDR magnitude and the EDR amp. following the first stimulus as obtained by these authors point to the mean amplitudes being considerably determined by the initial OR. Thus, neither adding valuable information nor really describing the course of habituation properly mean EDR amp. or magnitude measures are not recommended.

Difference scores of type (3) describing the habituation process are also dependent on the value of the initial OR, especially if the amplitudes of the first and last trial are included. Subjects or groups showing a high initial EDR amp. would yield a higher habituation rate, even if their reactions to the other trials show no differences to subjects having lower initial ORs.

According to O'Gorman (1977) difference scores are mainly used to compare groups of subjects. In that sense, the interaction term as mentioned above under (2), taken from

[142]SCR measured with standard methodology during the presentation of 1 kHz tones (70 or 90 dB). The signs from Siddle and Heron's (1976) Table 2 were inverted to make the directions of correlations comparable to each other.

ANOVA using repeated measures over trials, is comparable to the interaction term in an ANCOVA using difference scores (Huck & McLean, 1975).

Difference indices can also be obtained as an individual characteristic (Koriat et al., 1973; Lader, 1964; Vossel & Roßmann, 1982). Correlations between H scores and these difference scores are negative (between –.21 and –.51). Difference scores correlate negatively with the regression slope b (r = –.90 to –.94) and positively with the ordinate values a (r = .80 to .90). This leads to the conclusion that difference scores do not provide essential information beyond the regression indices as mentioned under (1), but in turn also may be used as an alternative to those measures of habituation process.

A special difference index of an individual has been proposed by Gruzelier (1973). He used only the first section of the habituation series, in which the decrease in amplitude approximates an exponential curve, and dropped the second part, in which a visual inspection yields a more random individual course. The first section was divided in two halves and the mean EDR amp. of each one was calculated, the difference between both being used as an index of individual habituation course. Gruzelier (1973) as well as Gruzelier and Venables (1972) used that index to obtain differential habituation in schizophrenics and normal controls (Sect. 3.4.2.2) as well as in EDR lateralization studies (Sect. 3.2.2.2).

Instead of giving a description of its course, habituation is more frequently measured in terms of its duration. Determining the end of the process requires a criterion of "zero-reaction" (Fig. 44). Two different habituation measures can then be formed:

(1) One method is the use of criterion measures. The number of trials necessary, or the time elapsed, to reach the end of the habituation process (O'Gorman, 1977) is taken as a measure. Usually the criterion consists of two or three consecutive trials eliciting zero-reactions (Siddle et al., 1983b).

(2) A second method is to form a frequency index. Here, the number of trials which exceed the zero-reaction is used as a criterion. Using this method, OR reinstatement which occurs after several trials of zero-reaction is accounted for, which is not done in the criterion method. Thompson, Groves, Teyler, and Roemer (1973) claim this procedure provides a more precise analysis of the underlying habituation process. They claim it is superior to the method of inspecting the amplitude changes over trials (however, see O'Gorman ,1977, for a contrary position on the matter).

The overall high correlations between criterion and frequency measures of habituation seem to point to the fact that both may be describing a similar aspect of the habituation phenomenon (i.e., trials to habituation). For example, Coles, Gale, and Kline (1971) presented 60 subjects with a series of 20 stimuli[143] and obtained a corre-

[143] 1KHz, 65 db, 5 sec duration tone. EDA was recorded as SR with 1 cm^2 Ag/AgCl electrodes and NaCl paste, using 11 μA current, transformed into log SC units.

lation of .92 between both types of measures. A similar correlation of .94 was reported by Vossel and Roßmann (1982). However, in some instances criterion and frequency measures may yield different results, as shown by Zahn, Carpenter, and McGlashan (1981a) in schizophrenic samples (Sect. 3.4.2.2).

Both measures seem to be influenced by individual differences in tonic EDA, since several studies report significant positive correlations to NS.EDR freq. between .44 and .75 (e.g., Coles et al., 1971; Crider & Lunn, 1971; Siddle & Heron, 1976; Martin & Rust, 1976; Vossel & Roßmann, 1982). Therefore, Crider and Lunn (1971) claimed that speed of habituation (as indicated by either measure) and spontaneous fluctuation of EDA are interchangeable, which is, however, not justified since the mean common variance explained over studies is approximately 36%.

In addition, the criterion measure is likely not to be independent from the initial amplitude in the series, which is apparent since the decrease of a large response requires more trials than a small initial response. For example, Nebylitsyn (1973) reported a correlation of .68 between the EDR amp. within the first trial and the number of trials to reach the habituation criterion. Furthermore, Coles et al. (1971), in their above-mentioned study, found inverse relationships between SCR lat. following the first stimulus and the criterion measure ($r = -.58$) as well as the frequency measure of habituation ($r = -.67$).

Reliabilities of the frequently-used criterion measures are moderately high. O'Gorman (1974) reported coefficients between .55 and .75 from several studies that used intervals between 1 week and 3 months. Similar coefficients were obtained by Siddle and Heron (1976) over 3–5 months from 37 subjects, where the criterion index[144] yielded retest reliabilities between .47 and .56.

Electrodermal habituation indices of end of process seem to be relatively independent from those of habituation course, thus indicating different aspects of the phenomenon. This was shown by Spinks (1977, after Siddle et al., 1983b) in a factor-analytic study with 45 subjects, where both types of parameters yielded their highest loadings in different factors. Hence, there is need for theories that allow precise selection of the appropriate measure (Koriat et al., 1973). Current theories do not explain the apparent dependency of EDA habituation on the initial EDR amp. (Siddle et al., 1983b). Therefore, range correction of EDRs in an habituation series should be taken into consideration using the initial OR as the maximum reaction (Sect. 2.3.3.4.2).

Furthermore, Levinson and Edelberg (1985) showed that the use of large time windows may yield spontaneous EDRs that are misclassified as being stimulus dependent (Sect. 2.3.2.2), thus invalidating habituation indices. Those authors therefore proposed the use of relatively small windows (1.0–2.4 sec after stimulus onset), probably using the latencies to the first stimulus taken from all subjects to determine the window's upper limit. They also recommended the use of not more than two subsequent zero-reactions as an habituation criterion.

[144]Three subsequent SCRs recorded with standard methodology below .02 μS.

A similar conclusion was made by Barry (1990) in his investigation of time window with relation to end of habituation. Comparing EDR magnitudes evaluated during time windows of 1–3 sec and 1–5 sec after stimulus onset, he found only small differences in the course of the habituation process. Additional comparisons using criteria of two vs. three subsequent zero-reactions yielded the most pronounced habituation when combining the narrow time window with the low-number zero-reaction criterion.

Evaluation of electrodermal habituation should consider significant correlations of various habituation indices to the NS.SCR freq. For criterion as well as frequency measures, those correlations are between .52 and .67 (Martin & Rust, 1976; Siddle & Heron, 1976; Vossel & Roßmann, 1982); for the index of mean amplitude (Bull & Gale, 1973; Martin & Rust, 1976) they are between .41 and .56, and for the H-score they are between .47 and .77 (Lader, 1964; Lader & Wing, 1966; Siddle & Heron, 1976). Correlations between the regression coefficient b and NS.SCR freq. are considerably lower (r = .19 to .32; Martin & Rust, 1976; Siddle & Heron, 1976), and the y-intercept is uncorrelated with spontaneous EDA (Siddle & Heron, 1976).

Because of the relatively close interrelations between spontaneous EDA and various habituation parameters, Crider and Lunn (1971) suggested habituation speed (as obtained by criterion measures) and NS.SCR freq. as indices of a common dimension of "electrodermal lability" (Sect. 3.3.2.2). However, the mean common variance is not higher than 55% (Vossel & Roßmann, 1982). By contrast, Martin and Rust (1976), as a result of a factor-analytic study that used various EDA parameters including several habituation indices, pointed to the possible existence of a common factor "general reactivity."

When taking into consideration concepts like "general reactivity" or "electrodermal lability" in the discussion of habituation processes, there is an apparent missing link based on possible structural, physiological, and biochemical factors. Therefore, correlations may be simply determined by peripheral factors the influence of which has been discussed in detail by Lykken et al. (1966) as well as Lykken and Venables (1971). These authors recommended a range correction, as mentioned above, the advantages and disadvantages of which in habituation studies are discussed by Siddle, Turpin, Spinks, and Stephenson (1980). In general, theories that try to explain the habituation phenomenon do not specifically address the idiosyncrasies of the electrodermal system. Some authors attempt to model the OR's habituation along more cognitive lines (Sect. 3.1.3.2).

3.1.2 Conditioning of electrodermal reactions

Though integrated in most learning instances, classical and instrumental (or "operant") conditioning are presented separately for theoretical and experimental purposes. With respect to EDA, that distinction parallels the one between stimulus-specific EDRs (Sect. 2.3.1) and NS.EDRs, which cannot be easily separated in every case (Fig. 41,

Sect. 2.3.2.2). Hence, separating EDR components with respect to this distinction is stressed within the following sections.

3.1.2.1. Classical conditioning of electrodermal reactions

Classical EDR conditioning has already been described in detail by Grings and Dawson (1973) and by Prokasy and Kumpfer (1973). Therefore, only the most important EDR parameters and some recent approaches will be described below. Within human Pavlovian autonomic conditioning, EDA is the most frequently used psychophysiological variable.

Before performing an EDR conditioning procedure, it must be ascertained that the conditioned stimulus (CS), which should be neutral, does not provoke an EDR. This is normally performed by exposing the subject to a CS series prior to the conditioning trials, to allow the OR following the CS to habituate. However, this preexposure to the CS is also a condition that provokes a latent inhibition to the CS: a CS presented several times without being followed by the unconditioned stimulus (UCS) gains properties of a CS– (i.e., a neutral stimulus indicating that no UCS will follow). In this sense a CS– cannot be associated with a UCS as easily as a nonpresented CS could be. Though this inhibition could be demonstrated in several animal studies (e.g., Wagner, 1969), it has not been unambiguously seen in human classical conditioning studies (for a review see Siddle & Remington, 1987).

In order to demonstrate that the conditioned response (CR) is really dependent on the CS-UCS coupling, two different approaches may be used. The "between-subjects" design compares two groups of subjects, one of which receives contingent CS-UCS pairings, while the other group is exposed to the same stimuli in random order. In a "within-subject" design, each subject serves as his or her own control, insofar as reactions to the solely presented neutral stimuli (CS–) are compared to reactions to conditioned stimuli (CS+) which were followed by a UCS. Conditioning has appeared only if the CR following the CS+ is significantly higher than the CR to the CS– during the extinction period.[145]

Figure 46 shows a typical development of the EDR in the course of CS-UCS pairings during the first CS-UCS presentation (left-hand panel) and after several trials (right-hand panel). To identify the EDR components, the ISI between CS onset and UCS onset has to be at least 4 sec (Bitterman & Holtzman, 1952; Rodnick, 1937). Nevertheless, most EDRs show an overlapping form in the CS-UCS interval, as depicted in the right-hand panel of Figure 46 (Grings, 1969). In the analysis and interpretation of those EDRs the question arises to what extent these single components may be separated (Sect. 2.3.1.2.2). This separation is necessary in order to analyze how those components vary with experimental conditions. In addition there might be anticipatory reactions to an expected UCS especially during long ISIs. To differentiate those

[145]The problems with this design are discussed in detail by Grings, Givens, and Carey (1979), Rescorla (1967), and Seligman (1969).

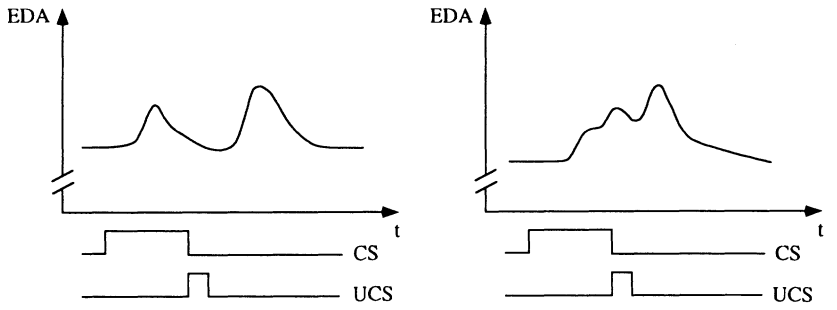

Figure 46. Typical course of an EDR during classical conditioning in the first trial (left-hand panel) and after several CS-UCS pairings (right-hand panel). From W. W. Grings (1969), Anticipatory and preparatory electrodermal behavior in paired stimulation situation. *Psychophysiology, 5*, Fig. 1, p. 599. Copyright ©1969 by The Society for Psychophysiological Research. Reprinted by permission of the publisher and the author.

components, Prokasy and Ebel (1967) ran a conditioning experiment with 121 subjects. They presented an eight-sec 1 kHz tone CS with an intensity of 75 or 100 dB, and a .2-sec electric shock as UCS. They were able to distinguish three different kinds of EDRs[146] with respect to their latencies:

(1) A first-interval anticipatory response (FIR), starting within of 1.35 to 4.95 sec after CS onset.

(2) A second-interval anticipatory response (SIR)[147], with a latency between 4.95 and 9.53 sec after CS onset.

(3) A third-interval unconditioned response (TUR), which should be unambiguously elicited by the UCS, starting between 9.53 and 14.55 sec after CS onset.

Since the authors did not find correlations between FIR and SIR, they concluded that both were independent reactions rather than two components of a single reaction. In addition, Prokasy and Kumpfer (1973) introduced another term for EDRs appearing when the CS was presented without UCS (i.e., in the extinction phase):

(4) A third-interval omission response (TOR), which appears in the same time window as the TUR to the omitted UCS.

[146]EDA recording has been described as being taken from the left index finger and the right palm with a Fels dermohmeter.

[147]Formerly abbreviated as FAR and SAR, respectively.

Since the FIR appears within the time window for an EDR following the CS (Sect. 2.3.1.1), it is likely to be interpreted as an OR to the CS. This view is supported by the FIR amplitude often showing an exponential decrease over trials, which indicates habituation (Graham, 1973). However, sometimes an increase during the first trials can be observed, the FIR amplitude reaching its maximum when the subject becomes aware of the CS-UCS contingency. According to Zeiner (1970), this phenomenon may be traced back to the CS habituation series preceding the CS-UCS presentation as described above. Since normally only one or two stimuli are used as CS during classical conditioning, the CS-UCS contingency is recognized immediately, which explains the rapid decrease of FIR amplitudes during the early trials. If the trials are preceded by a CS habituation series, a delay in contingency recognition is likely to appear, and an initial change in expectation (CS alone vs. CS-UCS coupling) leads to an increase of FIR amplitudes (Maltzman, Raskin, & Wolff, 1979b).

Öhman (1971) showed that the FIR also fulfills another important criterion of an OR; it is susceptible to novelty. In an aversive conditioning study, all 40 subjects received a 70 dB tone with a frequency of 3 kHz during a training period. During the test period, tones with 200, 500, and 1,200 Hz were presented as CSs. In both the conditioning and the control groups (without CS-UCS coupling), a direct association appeared between the FIR (measured as SCR using standard methodology) following the novel stimulus and the difference between frequencies of training and test stimuli.

As opposed to the FIR, the SIR shows only a small amplitude at the beginning of the conditioning (Dengerink & Taylor, 1971). The SIR also remains uninfluenced by CS properties (Orlebeke & van Olst, 1968) or by training trials prior to conditioning (Surwit & Poser, 1974). Instead, the SIR depends on the UCS quality and on the probability of its occurrence. Many results indicate that SIR is more frequently conditioned if electric shocks are used as UCS instead of acoustic stimuli (Dengerink & Taylor, 1971). Thus, the SIR may be regarded as a preparatory reaction to expecting the UCS.

The appearance of a TOR instead of a TUR in cases of UCS omission after subsequent CS-UCS coupling can be regarded as an OR to a stimulus change (Öhman, 1983). In terms of cognitive psychology, the TOR can be traced back to the discrepancy between an expected and a real stimulus situation. After the subjects have learned to expect a specific UCS, changes in UCS appearance will be followed by a marked EDR. These perceptual disparity responses (Grings, 1960) presuppose a learned expectation of the UCS following the CS, which implies that an associative learning process is necessary for the TOR to occur. However, the authors who regard the TOR as being due to OR reinstatement (Sect. 3.1.1) deny that associative processes contribute to its occurrence (Furedy & Poulos, 1977). In any case, the TOR amplitude parallels the amount of change in UCS properties (Kimmel, 1960).

In his information processing model of OR, Öhman (1979) provides a theoretical interpretation of the origin of different components (Fig. 47). The so-called expectancy loop points to the cognitive presentation CS-UCS contingency which is represented in the short-term memory (STM), where qualitative and quantitative properties of UCS

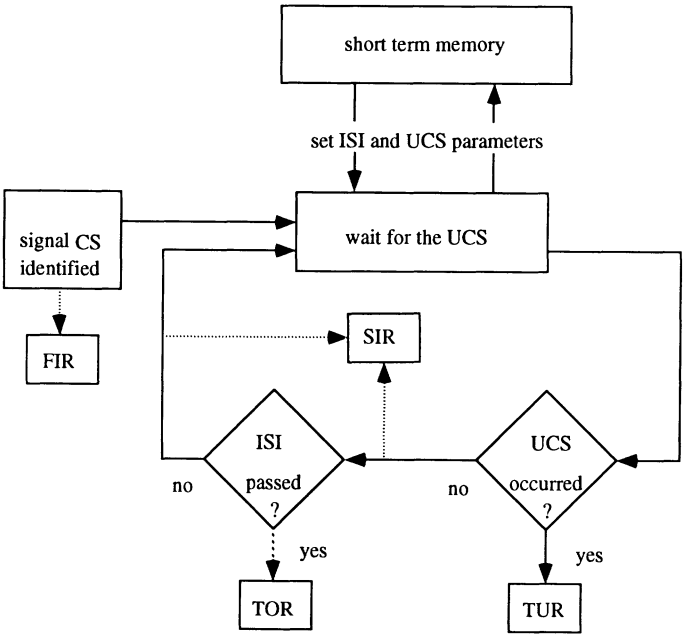

Figure 47. Information processing model after A. Öhman (1979). The orienting response, attention, and learning: An information-processing perspective, Fig. 27.3. In H. D. Kimmel, E. H. van Olst, and J. F. Orlebeke (Eds.), *The orienting reflex in humans.* Copyright ©1979 by the author. Used by permission.

as well as temporal relations between CS and UCS are stored (i.e., those of ISI duration and variability). With respect to ISI properties, the UCS occurrence is checked continuously, which is manifested by the SIR occurrence. If the UCS does not occur in the expected time window (i.e., the learned ISI plus a psychophysically determined difference time threshold), a TOR is elicited. If a UCS occurs, a TUR is elicited, while the FIR is regarded as an OR following the CS.

The fact that conscious cognitive processes are probably included in classical conditioning was shown by Dawson, Catania, Schell, and Grings (1979) in a study with 64 subjects, who were presented with different light and tone stimuli simultaneously, and a certain stimulus combination served as CS for the subsequent shock-UCS pairings. All subjects received information about the light-shock contingency, while half of them had additional information concerning the tone-shock combination.[148] Contrary to the

[148]EDRs were recorded as SRRs using standard methodology, with KY-gel.

light-tone informed, the tone-only informed group showed CRs neither subsequent to the tones alone nor to the light-tone combinations. The authors concluded that classical conditioning is due to complex information processing comprising different stages. Among those stages enumerated were: CS perception and recognition, and memory of the UCS occurrence and encoding, along with the storage of this information at the end of each trial. The autonomic reactions being observed in the classical conditioning paradigm can be regarded as peripheral correlates of the outlined central cognitive processes.

In a subsequent experiment, Dawson, Schell, Beers, and Kelly (1982) demonstrated the importance of cognitive processes in classical conditioning, by using an RT task within the CS-UCS interval as an indicator of allocation of attentional resources. The CS was followed in 300–750 msec by a signal to which the subjects had to respond. RT was significantly longer to the CS+ (signalling an electric shock) as compared to the CS–, which the authors interpreted as due to the higher amount of attentional capacity used for CS+ processing (Sect. 3.1.3.2).

The hypothesis that cognitive processes are involved in classical conditioning (summarized by Dawson & Schell, 1987) was questioned by Furedy and colleagues (Furedy & Schiffman, 1971, 1973; Furedy, Poulos, & Sciffman, 1975; for an overview see Furedy & Riley, 1987). Stimulated by the control group discussion within classical conditioning being elicited by Rescorla (1967), these authors investigated the connection between EDR and subjective awareness of CS-UCS contingency. They showed a positive correlation between EDR amp. and the amount of awareness of the CS+ UCS contingency, which is in accordance to the hypothesis of Maltzman et al. (1979b). However, significant differences between a randomly paired CS condition and a contingent nonreinforced (CS–) condition appeared exclusively in the expectation of the subjects (as measured by movement of a lever). In addition, there was a zero correlation between EDR and subjective contingency. Despite this, Furedy and Riley (1987) concluded from a large number of experiments that EDR conditioning is at least partly due to noncognitive response learning, even though knowledge of the CS-UCS contingency seems to be necessary for EDR conditioning (Dawson & Furedy, 1976).

Another phenomenon which appears regularly in electrodermal conditioning is the so-called UCR diminution during repeated CS-UCS combination. Some authors believe this is due to the buildup of conditioned inhibition, whereby the CS gains inhibitory properties (e.g., Kimmel, 1966). An alternative view has been provided by Lykken (1968) in his so-called preception hypothesis which states that temporal predictability of a UCS reduces its aversiveness (Sect. 3.2.2.2). This is performed by a phasic and selective inhibition process that reduces the arousal reaction to the UCS, which is cognitively mediated by the warning-signal quality of the CS. However, this interpretation of UCR diminution remains questionable. On the one hand, the physiological (especially the electrodermal) reactions to such a UCS paired with a warning CS decrease over trials as predicted (e.g., Grings, 1960; Lykken, Macindoe, & Tellegen, 1972; Furedy, 1970, 1975). On the other hand, most studies did not yield a decrease

of subjective reactions to the UCS over trials (Baltissen & Boucsein, 1986). Lykken et al. (1972) continuously recorded HR and SCR with standard methodology in 48 male subjects that received 12 consecutive shocks under each of four conditions that were permuted in order. They were combined with the presence of a warning signal vs. no warning and predictable vs. nonpredictable locus of shock (on which of the four limbs the next shock would occur). Range-corrected SCR amp. were consistently reduced when shock was preceded by a warning signal, while the effect of predictability of locus was weak. Neither aspect of predictability influenced shock magnitude estimates made by half of the subjects.

Therefore, in order to explain UCR decline, additional concepts were considered, referring to habituation of the OR as well as to reaction interference. The observation that an EDR amp. following an unpredictable electric shock is greater than that following a predictable one has been interpreted by Grings(1969) as well as by Furedy and Klajner(1974) as OR reinstatement, because the less predictable a stimulus, the greater its novelty (Sect. 3.1.1.1). Lykken and Tellegen (1974) proposed a so-called first-signal system, which causes habituation without attentional or conscious processes being involved. An additional second-signal system which is responsible for the "preception" mechanism should have the ability to "short-circuit" the sluggish habituation process. This may lead to a weakening of the OR during the first trials before the normal habituation process reduces the reaction strength. This hypothesis, which states an interesting connection between habituation and classical conditioning, is, however, not yet supported by empirical evidence (Baltissen & Boucsein, 1986).

Another possible explanation for UCR decrease during classical conditioning is supported by data from Grings and Schell (1969) using a within-subjects design ($N = 27$). They showed that the SCR amp., following a constant UCS, changed reciprocally to the intensity and proportionally to the duration of a preceding CS.[149] This allowed for the interpretation of UCR decrease as being dependent on CR-UCR interference. However, the interference hypothesis is contradicted by results showing that a decline in the UCR elicited by a CS has the quality of a warning signal, even if possible interference effects are controlled for. This had been found by Peeke and Grings (1968) using 20 subjects in each group with constant (5 sec) versus variable ISIs (.6–11 sec) between a CS and an electric shock as UCS. They found smaller SCR amp.[150] to shocks preceded by constant ISIs, comparing only those trials in which the ISIs for the variable condition were the same as for the constant condition.

Katz (1984) showed that the preception hypothesis can be confirmed if interference effects are controlled, if stimulus conditions are sufficiently aversive, and if the subjective impact is adequately assessed. Using 80 female subjects, he found a decrease

[149]Recorded from the fingers as SR with 2 cm^2 Ag electrodes, NaCl paste, and 45 μA current strength, changed to SC values and square-root transformed. 2-sec white noise of 100 dB served as UCS, while the CS was 80, 90, or 98 dB, varying between 2 and 10 sec, in 2 sec increments.

[150]Recorded from the fingers as SR with 2.5 cm^2 Ag electrodes, using 45 μA current, changed to SC and square-root transformed.

of the electrodermal UCR[151] following a warning stimulus (Sect. 3.2.2.2). However, he used a series of lights which were illuminated sequentially during the ISI instead of a classical delayed conditioning paradigm, thus facilitating time estimation, which can be assumed to be a critical factor with respect to preception (Furedy, 1975).

To test differential predictions based on an habituation versus preception position, Baltissen and Boucsein (1986) presented two groups of 20 subjects each with a series of thirty 110 dB, 2 sec white noise UCSs with or without a CS (dimming the ambient light 5 sec prior to UCS). A third group received 70 dB white noise stimuli without warning. SCR amp. (recorded with standard methodology) following warned UCSs habituated within 6–8 trials; however, they did not reach as low a level as the 70 dB control stimuli. The SCRs following the unsignalled 110 dB UCSs showed a marked tendency toward sensitization instead (Sect. 3.1.1.2). However, the subjective aversiveness,[152] which slightly decreased under all conditions, did not differentiate warned and unwarned 110 dB noise, being only slightly lower following 70 dB. Therefore, the data seemed to be in line with an OR reinstatement interpretation.

A subsequent study performed by Baltissen and Weimann (1989) varied aversiveness (60 vs. 100 dB white noise) and predictability (6 sec constant vs. 2–12 sec variable ISIs) in a factorial design with 15 subjects in each group. In the total of 30 trials, only those 11 having a 6 sec ISI under all conditions were evaluated. A significant interaction between aversiveness and predictability, which could have confirmed the preception hypothesis, appeared only as a tendency toward significance in the SCR magnitudes (Sect. 2.3.4.2) recorded with standard methodology, but not in subjective aversiveness. Possible reaction interferences between FIR and/or SIR, on one hand, and TUR on the other, cannot serve as an alternative explanation in this instance, since ISIs were comparable between groups differing with respect to predictability. Thus, for classical conditioning research in EDA, a sufficient number of ISIs having equal length in each group will be necessary to control for possible interference effects.

3.1.2.2 Instrumental or operant conditioning of the EDR

According to the dated view of Miller and Konorski (1928), classical conditioning should be restricted to ANS variables, while motoric reactions should be influenced only by instrumental conditioning. Meanwhile, several ANS functions including EDA could be successfully modified by instrumental conditioning techniques as well. Kimmel (1967) and Miller and co-workers (e.g., DiCara & Miller, 1968; Miller, 1969, 1972) challenged the view that visceral functions could only be classically, not instrumentally, conditioned. In their experiments conducted with curarized rats, these authors showed conditioning of ANS functions (e.g., HR, peripheral vasomotor tonus, or gastrointestinal activity) as a result of stimulating "reward" areas in the brain (Sect. 3.2.1.2).

[151] Recorded as SR from the medial phalanges using standard methodology. SRRs exceeding an amplitude criterion of .05 kΩ were transformed to SCRs and additionally square-root transformed.

[152] Recorded after each UCS presentation by means of a seven-point Likert scale via micro-keys.

The question of whether instrumental conditioning of autonomic functions could also occur in humans had been determined to be equivocal, since it could not be ruled out that instrumental visceral conditioning effects might be mediated by skeletal responses (Beatty, 1983). Possible muscular mediation does not play an important role in discussing the elicitation of electrodermal phenomena, except for some supportive actions of duct-surrounding myoepithelia in squeezing sweat out of the pores (Sect. 1.3.3.1). However, the possible cognitive mediation of instrumentally conditioned EDA has been a central issue. Hence, as opposed to classical conditioning of EDA, where the main problem is to distinguish various EDR components, the focus within instrumental conditioning is on the elicitation of the "instrumental" EDR.

This view is exemplified by a study performed by Martin and Dean (1970) consisting of two experiments. In the first, 33 female subjects were presented with a light as a discriminative stimulus. An EDR[153] during the presentation of a red light switched the light off and elicited an electric shock of one sec duration. A spontaneous EDR appearing during a blue light prevented the shock. Subjects in the instrumental group were informed of this contingency and were told to elicit EDRs only through internal emotional processes and not by movement or respiration. Subjects of a yoked control group received the same instructions, but the shocks were given according to the reactions of their partner in the instrumental group. Another yoked control group received only the instruction that both lights were followed by an electric shock in an irregular manner. As compared to the others, the latter group showed smaller EDRs following the discriminative stimulus which announced the shock. The spontaneous EDR amp. in this group was higher than that in the other groups during presentation of the light signalling the EDR allowing for the prevention of shock. This result points to a marked influence of instruction on the instrumental modification of EDA. In their second experiment, they showed that a reaction-contingent presentation of shock does not have an instrumental effect. It seems that the different reactions to the discriminative stimuli observed in the first experiment had been cognitively mediated. It might be that these mediating cognitive processes are even more important for instrumental conditioning than the stimulus presentation's contingency upon the elicited EDR, because even with noncontingent shock application, as for the second group of the first experiment, both stimuli elicited EDRs of different strength.

Several studies performed during the 1960s and 1970s showed successful instrumental conditioning of various EDA parameters. Helmer and Furedy (1968) elicited significantly higher EDR amp. in a group of 40 subjects who were reinforced with money for an amplitude increase[154] than in a control group of equal size with noncontingent reinforcement. With a total of 36 male subjects, Kotses, Rapaport, and Glaus

[153]Recorded from the 1st and 2nd left-hand fingers with constant current.

[154]Measured as SR with a Fels dermohmeter, using 70 μA current, with Zn/MnO_2 electrodes from the left palm and upper arm. Contact was made with a 5% saline-soaked cotton ball.

(1978) found an SRL[155] increase induced by reinforcement, while SRL changes in a yoked-control group varied unsystematically.

EDA not only can be elicited but can also be suppressed by instrumental conditioning. This was demonstrated by Johnson and Schwartz (1967) using an aversive loud tone (700 Hz, 3 sec, 95 dB), following spontaneous SRRs.[156] With noncontingent presentation of the aversive stimulus, the experimental group ($N = 16$) showed in the acquisition as well as in the extinction phase a significantly lower NS.SRR freq. as compared to a control group of equal size. Using parallel measures of EMG, the authors found no evidence supporting a possible skeletal muscle mediation explanation of their results with SRRs (for further results see Kimmel, 1973, p. 265).

Experiments like the ones reported above do not allow for the unambiguous determination of whether the results are solely mediated by instrumental conditioning. They may be due to uncontrolled classical conditioning or to cognitive processes, and they might even be elicited by voluntary muscular contractions (contrary to instructions) leading to an artifactual EDR (Sect. 2.2.5.2). To avoid this, Katkin and Murray (1968) proposed the use of curare which paralyzes the skeletal muscles without influencing functions of the brain and of inner organs. Birk, Crider, Shapiro, and Tursky (1966) performed a study using only one subject. They showed a tendency towards conditioning, despite a decrease of EDR rate during application of curare.[157]

By recording respiration as well as EMG in addition to EDA, van Twyer and Kimmel (1966), in a study with 42 subjects, tried to control muscle artifacts without the application of curare or of muscle relaxants.[158] A light was presented when EDRs deflected 1% or more from the EDL. Recordings of EMG and respiration did not yield significant differences between the contingent and the noncontingent reinforced groups. In further analyses, only those EDRs were taken that were not accompanied by changes in respiration or muscle activity. In the contingently reinforced group, as compared to the noncontingent one, EDR frequency increased during acquisition and during extinction, thus pointing to a conditioning effect.

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[155]Recorded with Ag/AgCl electrodes and Beckman paste from ethanol-cleaned volar finger sites using 20 μA constant current. Red and green lights were used to signal that either an increase or a decrease of SRL would be reinforced by a flashing white light.

[156]Two SR channels with different sensitivity were used to avoid loss of SRR data in the high-sensitivity channel (Sect. 2.1.3) which allowed the detection of 500-Ω changes (amplitude criterion). Eight-mm diameter zinc electrodes together with a zinc-sulfate paste were placed on the fingers of the subjects. Spontaneous EDRs were defined as not occurring within 6 sec following any observable event which could give rise to an EDR (see Fig. 42, Sect. 2.3.2.2.).

[157]Roberts, Lacroix, and Wright (1974) could not observe instrumental conditioning of spontaneous SPRs in curarized rats. They used an electric shock as reinforcer, which was applied when SPRs appeared exceeding either 10%, 35%, 60%, or 75% of the greatest reaction during the baseline. As compared to a yoked control group, no differences in SPR frequency appeared. Neither variation of shock intensity, or of curare dosage, had an effect.

[158]Recorded palmar versus dorsal with 2 cm^2 zinc electrodes filled with NaCl paste, transformed to log units.

In his comprehensive review of studies using different forms of instrumental EDR conditioning (i.e., positive and negative reinforcement as well as avoidance conditioning), Kimmel (1973) concluded that enough evidence was available to support the existence of the phenomenon under investigation. However, the main problem is the lack of a generally accepted theoretical framework. Additionally, experimental paradigms proposed to separate operantly conditioned from cognitively mediated ANS reactions did not yield unequivocal results in electrodermal research.

Another conceptual problem is that instrumental (or operant) EDR conditioning is often connected with electrodermal biofeedback. For example, the above-mentioned study by van Twyer and Kimmel (1966) used EDR biofeedback. Hence, studies of instrumental EDR conditioning were largely integrated in research on biofeedback during the 1970s (see Obrist, Black, Brener, & DiCara, 1974; Shapiro, 1977; Beatty & Legewie, 1977). The term *biofeedback* refers to the use of a wide variety of experimental procedures that present parameters obtained from biosignals to an organism as exteroceptive feedback, with the aim of modifying the underlying physiological processes (Beatty & Legewie, 1977). In terms of learning theory, the operant response is the occurrence of the ANS change, and the information about the response serves as reinforcer (Shapiro, 1977).

Therefore, EDA biofeedback may use the occurrence of spontaneous EDRs (i.e., NS.EDRs) as an appropriate parameter, as most studies on instrumental conditioning of EDA did. However, in order to maintain a close temporal relationship between the underlying physiological processes and the presented signal, the latter has to be evaluated on-line without noticeable delay. This requires a small time window for signal evaluation. As compared to HR, where the interbeat interval, or an integration across a few of those intervals, may be used for feedback, an analogue of phasic changes in EDA cannot be obtained as easily. First, spontaneous EDRs appear without regularity (see Fig. 42, Sect. 2.3.2.2), and a time window of 3–5 sec is required to obtain at least one EDR, since even in states of high arousal, NS.EDR freq. is not expected to exceed 20 per min (Sect. 2.5.2.1.2). Second, there exist various problems with on-line detection of EDRs, as discussed in Section 2.2.4.4. Therefore, EDA biofeedback has mostly been performed using the EDL signal, which has the disadvantage of showing only slow changes.

EDA biofeedback may be principally used to change autonomic arousal in both directions. However, the main aim has been its therapeutic use (e.g., in the reduction of clinical anxiety; Sect. 3.4.1.1), where a decrease in arousal is attempted. In summarizing results from earlier studies, Holmes, Frost, Bennett, Nielsen, and Lutz (1981) found that EDA biofeedback was effective for increasing arousal in all of the five studies where an increase in arousal had been attempted. Instead, two of three experiments failed to show the effectiveness of biofeedback for decreasing arousal. In their own study, Holmes et al. (1981) performed two experiments on the effect of instructions

to increase as well as to decrease SRL[159] with or without the aid of biofeedback in nonstressful and stressful conditions.

In the first experiment with 48 subjects of both genders, Holmes et al. (1981) found that during six 3-min nonstressful recording periods, biofeedback generally increased SRL (i.e., decreased arousal), but biofeedback only aided those subjects following the instruction to increase their arousal, and not those who tried to decrease it. Furthermore, subjects who were not instructed to change their SR and not given feedback showed the lowest level of arousal. Following Shapiro's (1977) suggestion that biofeedback training may be more effective when conducted in stressful situations in which subjects are more aroused, Holmes et al. (1981) performed a second experiment with 52 subjects, using five conditions: stress induced by threat of electric shock alone; together with instructions to relax; with relax plus biofeedback; with relax plus placebo; and a nonstress condition as control. SRL was decreased by stress, and instructions to relax increased SRL (decreased arousal), but neither SR biofeedback nor placebo aided the subjects in reducing their arousal.

Apart from the question of the therapeutic viability of biofeedback, the study of Holmes et al. (1981) clearly shows the general control group problem in this research area. As discussed above for instrumental conditioning of EDA, various factors have to be controlled carefully, including cognitive processes as well as artifactual elicitation of EDRs (e.g., by muscular contractions, or irregularities in respiration), which may influence EDL in an uncontrolled manner. The question of how biofeedback is mediated, and whether the control gained over the particular signal is selective or concerns general changes in the ANS, as thoroughly discussed by Shapiro (1977), remains debatable.

An attempt to investigate the specificity issue was made by Roberts (1977), reporting data from an experiment that compared SCL and HR biofeedback, under either increase or decrease instruction, in four independent groups with eight subjects each. Control over EDA and HR was affected differently by various training variables. Simple instructions to change SCL in either direction without exteroceptive feedback resulted in substantial control over HR but not over SCL. By contrast, with the aid of biofeedback it was possible to gain electrodermal control[160]. Roberts offered a neurophysiological explanation of these differences between both systems. While HR may be voluntarily changed by the use of different afferent sources (e.g., pressure receptors, auditory sensations, or mechanoreceptor stimulation by vascular changes), interoception that arises directly as a consequence of sudomotor effector action is questionable (Kuno, 1956), though tactile or thermal changes following sweat gland activity are

[159]These authors measured skin resistance with constant voltage (which normally gives conductance values) from the first and second fingers of the subject's nondominant hands, using 20 by 25 mm electrodes.

[160]Stern (1972) showed that SRR biofeedback training had only a small effect on the subject's ability to detect whether or not they reacted electrodermally to a low buzz. Furthermore, prior EDA biofeedback training was more effective for the detection of large EDRs than small ones.

known (Edelberg, 1961). Thus, some specifity of the EDA biofeedback may be given, but the overall effect seems to be rather weak (Roberts, 1977).

In general, the usability of EDA biofeedback in its most advocated clinical-therapeu use remains ambiguous. However, therapeutic applications with greater proximity tc the signal in question might be more promising in the future, such as the use in anhidrosis or hyperhidrosis and other dermatological disorders (Sect. 3.5.2.1).

3.1.3 Electrodermal indices of information processing

In addition to the electrodermal concomitants of simple orienting and conditioning as outlined in the previous sections, there is also theoretical as well as empirical evidence for close connections between higher stages of information processing and certain EDA parameters. Aside from today's mainstream focus on EEG variables (e.g., components of event-related potentials) as suitable correlates of information uptake, decision, and storage processes, a small but effective psychophysiological research area is maintained using ANS parameters as indicators of different stages of cognitive processing (Sect. 3.1.3.1 – 3.1.3.3). Another field in which EDA has been related to cognitive functioning is hemispheric lateralization, reviewed in Sect. 3.1.3.4.

Since the focus of the present chapter is on phasic EDA parameters, results which consistently show an improvement of performance (e.g., in vigilance and RT tasks) with higher levels of tonic EDA are not reported here.[161] These are due to a higher level of general arousal which causes increased attention and motor readiness as well as higher tonic EDA values (Sect. 3.2.1.1).

3.1.3.1 Neurophysiological considerations on EDA and information processing

As already demonstrated with respect to Sokolov's (1963) model of the OR and its habituation (Sect. 3.1.1), subcortical structures are generally accepted as exerting an important influence on higher levels of information processing. Further theoretical support is given by a model proposed by Pribram and McGuinness (1975), based on their extensive review of studies of neuropsychological and psychophysiological research on attention performed with monkeys as well as with human subjects. They concluded that there exist three neuronally distinct and separate attentional subcortical systems operating upon the information processing mechanism, which closely resembles Posner's (1975) taxonomy for attentive processes, and also includes the main aspects of the two-arousal hypothesis as proposed by Routtenberg (1968):

(1) An "Affect Arousal System" which centers on the amygdala as part of facilitatory and inhibitory serotonergic pathways. It regulates specialized "arousal" neurons and is organized around a "stop" or balancing mechanism. It also regulates focusing of attention, or selective attention in Posner's sense, elicits phasic

[161] The reader is referred to reviews by Raskin (1973), Spinks and Siddle (1983), or Koelega (1990), as well as to Section 3.3.2.2.

physiological concomitants to sensory input, and is closely connected to the kind of arousal covered by Routtenberg's Arousal System I (Fig. 48, Sect. 3.2.1.2).

(2) A "Preparatory Activation System" centered around the basal ganglia and therefore being apparently dopaminergic in nature. It exerts control over the "go" mechanism in brain systems which elicit tonic physiological concomitants of increased perceptual and motor readiness, corresponding to alertness in Posner's terms, and more closely resembling Routtenberg's Arousal System II.

(3) An "Effort System" which comprises the hippocampus and the Papez circuit. It exerts control over the relationship between amygdala "arousal" and basal ganglia "activation," leading to changes in central representation which may be conceived as changes of state, set, or "attitude." This process entails "effort," which is related to the degree of consciousness according to Posner (1975). The effort system has the ability to decouple both of the other systems and thus coordinate tonic and phasic components of physiological reactions.

After having reviewed studies on problem-solving taken from the psychophysiological literature, Kahneman (1973) suggested that the above-mentioned "arousal" component was an indicator of modification in the allocation of resources from a limited capacity attentional system to mental activities. While Kahneman did not clearly distinguish between "arousal," "capacity," "effort," and "attention," Pribram and McGuinness (1975) restricted the amygdala-guided component of information processing to viscero-autonomic, mainly sympathetic "arousal," which often mirrors stimulus parameters such as novelty and complexity. Therefore, McGuinness and Pribram (1980) closely associated the "affect arousal" to the OR (Sect. 3.1.1). Basal ganglia guided "activation," on the other hand, was mostly identified by those authors with somatomotor readiness, and thus they regarded cardiovascular processes as the most adequate indicator of preparatory activation. Hippocampal "effort," which is necessary to overcome established connections between stimulus and response characteristics, is indicated by its theta rhythm (not directly recordable from the intact human brain) and may also show up in some aspects of the contingent negative variation in the EEG.

However, all those CNS structures mentioned above presumably also influence EDA. As Bagshaw et al. (1965) demonstrated, the electrodermal component of the OR and its habituation is markedly impaired in amygdalectomized monkeys. The basal ganglia are involved in the origin of a premotor (locomotor) electrodermal component labelled EDA 2 (Sect. 1.3.4.1). The hippocampus has easy access to the origin of hypothalamically elicited electrodermal activity (EDA 1) via its connections within the limbic system (see Fig. 4, Sect. 1.3.2.2), but it also has inhibiting properties on EDA as shown in experiments with primates (Pribram & McGuinness, 1976).

Further evidence for a tight connection between hippocampal information processing and EDA is provided by the psychophysiological extension of Gray's (1973, 1982) septo-hippocampal system suggested by Fowles (1980), as outlined in Section 3.2.1.2.

Based on this theoretical framework, an increased tonic EDA may be regarded as a concomitant of behavioral inhibition with increased selective attention and a thorough information content analysis. Combining those models with the one provided by Pribram and McGuinness, different components of EDA can be hypothetically related to the different stages of information processing:

(1) A phasic electrodermal concomitant of sensory input, indicating an OR or a DR (Sect. 3.1.1.2), which is a result of an interaction between hippocampus and amygdala.

(2) Another phasic or perhaps tonic electrodermal component[162] indicating expectation or preparation, which is a result of an interaction between hippocampal and basal ganglia structures.

(3) A tonic electrodermal concomitant of increased attention and arousal, which is an indicator of hippocampal information processing.

Using the labels for the different origins of EDA as introduced in Section 1.3.4.1, the EDA 1 pathway will be used for the above type (1) and (3) components, while the EDA 2 pathway will be used solely for the type (2) component.

Evidence for an opposite action of the amygdala and hippocampal structures on electrodermal concomitants of information processing can be derived from considerations relating EDR recovery time to the range of attention made by Venables (1975), based on animal as well as on human experimental and clinical research. As Bagshaw et al. (1965) suggested from their research with hippocampal- and amygdala-lesioned monkeys, the duration of an EDR may be regarded as an index of the registration process for stimulus characteristics in a Sokolovian neuronal model (Sect. 3.1.1). Thus, a faster recovery indicates a shorter registration process and brings about a slower build up of the neuronal model, and hence a greater probability for reorientation together with a slower habituation. This view closely resembles Kahneman's (1973) report on pupillary as well as electrodermal concomitants of resource allocation policy. Venables (1975) combined this view with his own reinterpretation of results obtained by Edelberg (1972b), who recorded EDA[163] from 16 subjects during a resting and a stress condition (cold pressor test), as well as during four tasks differing in complexity (counting forward and backward, reading aloud, and mirror-drawing). EDR rec.tc was longest

[162]Despite this component being labelled as "tonic" by Pribram and McGuinness (1975), the preparatory EDA is often clearly phasic in nature, as in the case of the SIR (Sect. 3.1.2.1). Additionally, the CNV, which is regarded by those authors as the appropriate paradigm for testing preparatory activation, is mostly tested within S1-S2, with intervals of less than 6 sec. "Tonic" cannot be used here in the sense of longer-lasting shifts of arousal level, therefore "phasic" is more suitable. Since the sudorisecretory pathway used here is clearly connected to CNS structures preparing distinct motor actions (see Fig. 6, Sect. 1.3.4.1), the label "phasic" for this component is preferred here.

[163]Measured as SR unipolar between a 2 cm^2 electrode at the middle finger and a 75 cm^2 electrode on the upper arm with starch-paste and 8 $\mu A/cm^2$ constant current, transformed into SC scores.

during rest and under stress, and decreased with increasing task complexity. In another 14 subjects, prolonged EDR recoveries were found under threat of electric shock while the subjects were performing an RT task. Edelberg's conclusion was that acceleration of recovery indicates a mobilization for goal-directed behavior, and that slow recovery may be an indicator of a DR.

Instead, Venables (1975) offered the hypothesis of EDR recovery time as an indicator of readiness for information uptake. According to this theory, short recovery times indicate an "open attentional gate" (i.e., a wide range of attention), for example, during complex cognitive tasks. Instead, long recovery times are likely to occur under conditions of a "closed attentional gate," which appear during resting and under stress conditions as well. By using a tone-shock conditioning paradigm with 28 subjects, [164] Furedy (1972) provided additional evidence for a prolonged electrodermal recovery under increased anticipatory stress. In this study a significant increase of electrodermal FIR rec.t/2 (Sect. 3.1.2.1) was found with an increase of the UCS intensity.

Clinical evidence for Venables's (1975) suggestion that EDR recovery is related to attention and information uptake stems from research with psychopaths and schizophrenic patients. Psychopaths are known to show fast electrodermal habituation (Sect. 3.4.1.2), and they also show poor passive avoidance conditioning. They are assumed to constantly focus their attention, and their long electrodermal recovery times may indicate a "closed gate" state of attention. Thus, psychopaths may have deficits in their septo-hippocampal system, preventing them from good conditioning to punishment, nonreward, and also to novel stimuli. As will be pointed out in Section 3.2.1.2, these conditions form the input of the septo-hippocampal Behavioral Inhibition System according to Gray (1982).

Schizophrenics, by contrast, exert slow electrodermal habituation and short electrodermal recovery times (Sect. 3.4.2.1).[165] These are signs for an "open gate" state of information processing, which may be due to a predominance of subcortical activity from the hippocampal area over neural impulses from the amygdala. Table 6 combines all this evidence for two different subcortical systems on the elicitation of EDA during information processing including habituation and conditioning.

Differential influences on attention of the subcortical structures mentioned in Table 6 were suggested by Douglas and Pribram (1966) as well as by Pribram and McGuinness (1975). According to these authors, the amygdala's influence is predominantly on focusing of attention, while hippocampal influences determine the direction of attention. However, the connection between EDR recovery and cognitive processes outlined in Table 6 must be treated as a working hypothesis, since most of the evidence comes from research with schizophrenics and is controversial in some respects, which is also the case for the differences in habituation speed (Sect. 3.4.2.2). Furthermore,

[164] Performed as a reanalysis, in terms of electrodermal recovery, of data reported by Furedy and Klajner (1972) from a study with high vs. low intensity and signalled vs. unsignalled UCS.

[165] This can be stated only for schizophrenic responders, since habituation cannot be measured in nonresponders (Sect. 3.4.2.2).

Table 6. Suggested relationships between subcortical activity, EDR recovery, habituation speed, attentional processes, and psychopathological groups. (See text for explanations.)

	Predominance of subcortical activity from	
	Amygdala	Hippocampus
Buildup of the neuronal model	fast	slow
Electrodermal habituation	fast	slow
Electrodermal recovery time	long	short
Range of attention	narrow (focused)	over–wide (distributed)
Attentional gate	closed	open
Clinical groups	Psychopaths	Schizophrenics

the possibility that recovery is dependent on EDR amp. has to be considered, though Janes (1982), in her study described below, as well as Janes et al. (1985), in their study reported in Section 2.5.2.5, found ample evidence that electrodermal recovery was independent of EDR amp. as well as of prior EDA. By contrast, results with respect to the role of EDR amp. as an indicator of cognitive processes seem confusing because of the wide variety in theoretical backgrounds and experimental paradigms used. Extensive reviews of the appropriate literature have been provided by Spinks and Siddle (1983) with respect to cognitive influences on the OR, and by Dawson and Schell (1985), focusing on cognitive control in autonomic classical conditioning.

3.1.3.2 EDR and processing capacity

One of the most widely discussed theoretical views of the autonomic reaction's role in cognitive processing is the Öhman (1979) model (see Fig. 46, Sect. 3.1.2.1), which suggests that the elicited OR indicates a call for information processing resources. In this model, an OR is elicited when an incoming stimulus fails to find a matching repre-

sentation in STM.[166] This initiates a call for resources in a central processing channel with limited capacity, or initiates an unspecific response mobilization (Öhman, Dimberg, & Esteves, 1989). An opposing view has been advocated by Pribram and McGuinness (1975), stating that the OR is a passive reflection of the amount of information being registered in the CNS. In a later article, McGuinness and Pribram (1980) associated the OR with their "affect arousal system," which elicits phasic physiological concomitants of sensory input (Sect. 3.1.3.1). In order to test those different views of the OR's significance, Spinks, Blowers, and Shek (1985) presented 76 subjects of both genders a complex series of two-stimulus anticipation tasks. The imperative stimulus consisted of a slide with either one or six letters presented for either 100 or 1,000 msec, the content of which had to be reported not earlier than 8 sec following stimulus offset. It was preceded 8 sec earlier by a warning stimulus with three possible stages of information concerning the imperative stimulus (no, partial, or full information). Similar to the FIR-SIR differentiation being used in S1-S2 paradigms (Sect. 3.1.2.1), the authors used a time window of 1–5 sec following the onset of the warning stimulus for the stimulus-registration component, and a window of 5–9 sec for the preparatory component of the EDR.[167] The amplitudes of both EDR components turned out to be more dependent on the anticipated amount of information within the imperative stimulus than on the information content of the warning stimulus. Therefore, the authors concluded that the registration of information is neither a necessary determinant of the electrodermal OR's amplitude nor a parsimoniously useful part of OR theory. Instead, the authors suggested that registration of information be used to index an anticipatory activating process; this was also suggested by Sokolov (1966).

Cognitive processes may, however, not only influence electrodermal ORs to external stimulation. Internal cognitive processes (e.g., thoughts or expectations) may themselves elicit EDRs, which has recently been shown by Nikula (1991). These were labelled as "voluntary" ORs by Maltzman (1979a) (Sect. 3.1.1.1). They may also be regarded as a possible source of spontaneous EDA, thus influencing NS.EDR freq., which is taken as a tonic EDA measure (Sect. 2.3.2.2). The TOR which follows stimulus omission (Sect. 3.1.2.1) can be regarded as another example of a nonstimulus elicited EDR. This was shown by Siddle and Packer (1987), who attempted to test the models of Wagner (1978) and Öhman (1979). With the use of an S1-S2 paradigm in several experiments, they demonstrated a reliably higher mean SCR amp. to S2 omission together with a dishabituation to re-presentation of S2 in the subsequent trial. [168]

[166]The autonomic OR component is regarded as being elicited by "preattentive" mechanisms in Neisser's (1967) sense.

[167]Recorded as SR with standard methodology, using an additional abraded forearm site for grounding, changes in SCRs, range-corrected with respect to the EDR following a 1 sec, 100 dB white noise stimulus at the end of the experiment (Sect. 2.3.3.4.2), and square root transformed.

[168]Experiment 1 with two independent groups (12 subjects each of both genders) with and without omission in the 16th trial, either light-tone (1 kHz, 75 dB) or tone-light pairing as S1-S2, SCR recorded

When using reaction time to auditory and visual probes in a similar experiment,[169] there was also some evidence of electrodermal omission responding and dishabituation, which was accompanied by a slowing of RT. The authors concluded that longer RTs were due to the fact that both omission and representation of an expected stimulus demand information processing resources.

Similar results were obtained by Packer and Siddle (1989) in studying the effects of stimulus miscuing on EDR. In their first experiment with 24 subjects of both genders, the control groups received 33 S1-S2 pairings intermixed with 33 presentations of a different stimulus S3.[170] The experimental group received 29 S1-S2 pairings intermixed with 29 S3-alone presentations and four S3-S2 pairings. Miscuing in the experimental group increased SCR amp. as compared to appropriate trials in the control group. However, SCR amp. decreased across miscued trials, showing that miscuing became less surprising over time. Significant enhancement of SCR amp. appeared only in the first re-presentation trial. Both results were paralleled by those of continuously recorded S2 expectancy, which the authors interpreted as consistent with Wagner's (1978) priming theory of STM. In the second experiment performed with another 24 subjects of the same population, a RT stimulus probe (after the 300 msec S2 onset) was added, which yielded slower RTs following miscued and re-presented S2 trials as compared to ordinary S1-S2 pairings within the control group. Thus, an enlarged EDR following unexpected omission or signalling of stimuli can be regarded as due to incongruencies in expectancy that appear during information processing within the central channel, with the aid of information stored in the STM (Fig. 47, Sect. 3.1.2.1).

The EDR has been used as an indicator of automatic processing of salient stimuli which the subjects are instructed to ignore while taking part in experiments on selective attention.[171] Those studies use competing information, methods of distraction, shadowing, or variations of attention via instructions. A frequently used paradigm is "dichotic shadowing," where the attended ear is presented information given relevance, while the other (unattended) ear is provided (masked) with irrelevant information.

Dawson and Schell (1982) confirmed results obtained earlier by Corteen and Wood (1972) as well as Corteen and Dunn (1974) showing that even unnoticed information is processed to a level enabling semantic analysis. In addition, they found influences of shifts of attention as well as of laterality (Sect. 3.1.3.4). Significant stimuli scattered within irrelevant material were followed by an EDR only if they were presented to the left ear. During the first phase of the experiment, all 60 subjects were subjected

with standard methodology. Results were subjected to a range-correction using a 100 dB stimulus at the end of the experiment to elicit SCR max, and a square root transformation.

[169]Experiment 3 with 48 subjects, presenting light circles or tones as RT probes 1,300 msec following S2 omission and S2 representation.

[170]Similar stimulation and the same recording and evaluation techniques were applied as by Siddle and Packer (1987). A vibratory stimulus was used in addition to tone and light stimuli, all of them occurring equally often as S1, S2, and S3 in a latin square design.

[171]The various filter theories and those of channel capacity are summarized elsewhere (e.g., Broadbent, 1971; Massaro, 1975).

to a differential conditioning procedure in which animal words were associated with shock while anatomical control words were not. In the test phase, all words were repeated without shock. The critical words were presented to 20 subjects embedded in the attended message, and to 40 subjects in the unattended one. Half of the latter group had to press a key when they perceived a critical word. The attended message was tape recorded by a male, while the nonattended one was recorded by a female. Both consisted of single-syllable high-frequency words (every 750 msec). The presentation ear was counterbalanced over subjects. EDR amp.[172] were higher following shock-conditioned words as compared with neutral ones in the irrelevant material. However, this was true only in trials where subjects temporarily shifted their attention to the nonattended ear, which was demonstrated by masking errors, through postexperimental interviews, and by key-presses in the appropriate group. Thus, a short-term attentional shift was a prerequisite for detecting significant stimuli within unattended material being accompanied by an ANS reaction. This finding casts doubt on the earlier hypothesis of cognitive processing with unperceived sensory input, as stated by Davies (1983).

Spinks and Siddle (1983, p. 259), in summarizing older studies performed by their own group, also concluded that electrodermal ORs to irrelevant stimuli redirect attention from the attended channel to the unattended one. A larger EDR amp. presumably reflects a greater redistribution of attention, while rapid habituation is an indicator of the speed of development of processes that inhibit analysis of the distracting stimulus.

By contrast, Frith and Allen (1983) came to the conclusion that the EDR amp. following irrelevant stimuli probably reflects the level of attention rather than its direction, which shows up in the habituation rate. In their first experiment, 41 subjects performed different computer tasks (reaction time, vigilance, and arithmetic) while hearing irrelevant tones which were also presented during pauses (1 kHz, 70 dB). The SCR amp.[173] following irrelevant stimuli were significantly higher during task performance than during the pauses. In the second experiment with 39 outpatients suffering from minor neuroses, 16 subjects heard the irrelevant tones while performing a forewarned reaction time task (as in the first experiment), while 23 subjects were presented the same sequence of tones, but had no task to perform. Habituation speed was significantly greater in subjects given the tones during task performance. The authors suggested that a higher attentional level during the task was responsible for the greater OR to irrelevant stimuli as well as for the faster habituation.

One method to determine whether or not limited processing resources are shifted involves the use of a dual-task paradigm. With this method, slowing of reaction time to

[172]Recorded as SR (without reporting current density) from the fingertips of the left hand with Beckman Ag/AgCl electrodes and KY-gel, transformed to square-root conductance values. SCR amplitudes were evaluated quantitatively within 1–3 sec following each critical word, in contrast to the Corteen group, which used an all-or-none amplitude criterion of 1 kΩ for an EDR appearing within a 13 sec window.

[173]Recorded from the left-hand's fingers with 1 cm^2 Ag/AgCl electrodes and KY-gel, within 1–4 sec following stimulus onset. Two subsequent intervals without an SCR exceeding .02 μS were used as the habituation criterion (Sect. 3.1.1.3).

secondary task stimuli indicates the degree to which resources are allocated to a primary task. This method was used by Dawson, Filion, and Shell (1989a) in two experiments on the relation between the electrodermal OR magnitude and resource allocation. In the first experiment, 75 subjects of both genders performed a primary binaural auditory orienting task consisting of 48 trials. They were instructed to count the number of longer tones (7 sec instead of 5 sec duration of 70 dB 1 kHz tones) presented to one ear (task-relevant tones) and simply to ignore the same kinds of tones presented to the other ear (task-irrelevant tones). The last trial included an unexpected 1.5 kHz tone presented binaurally. The secondary task consisted of 248 visual reaction time probes that were interspersed within the primary task in a way that could not directly affect the OR. SCR amp. obtained with standard methodology[174] from 12 clear presentations of the orienting stimuli were greater to significant stimuli, which have been of longer duration as compared to nonsignificant stimuli. There was also a significant correlation ($r = .40$) between OR magnitude and the amount of resources allocated to the significant orienting stimuli, as determined by the reaction time in the secondary task. However, this was only true for reaction time probes presented either 300 or 600 msec following stimulus onset, while at the 150 msec probe position the insignificant orienting stimuli elicited the greatest resource allocation.

To further enlighten this unexpected result of a directional dissociation between the OR magnitude and resource allocation, Dawson et al. (1989a) modified their methodology and performed a second experiment with 86 subjects drawn from the same population. They replicated the findings of the first experiment. However, manipulation of predictability and discriminability of significant versus insignificant orienting stimuli affected the observed dissociation. Increase in predictability made the dissociation disappear because of the generally lowered requirement of resource allocation; an increase of difficulty of discrimination also reduced the dissociation because there was large and equal resource allocation to both kinds of stimuli. The authors concluded that the relationship between the ANS orienting response and resource allocation is a complex one. However, they also claimed that the theoretical framework provided by Kahneman (1973) and Öhman (1979) is consistent with their results, and may be used in the planning of further research.

The role of awareness in classical electrodermal conditioning (Sect. 3.1.2.1) is discussed extensively by Dawson and Schell (1985, p. 107f.). They compared the technique used in various studies of their own group and by other researchers – embedding CS-UCS pairings within a "masking task" – with the method of CNS ablation studies, since the subject's attention and concern is effectively directed away from the learning task. During the 1970s, the Dawson group published seven separate experiments involving more than 300 subjects using the same masking task (reporting colors of lights and position of tones). They all showed that CS-UCS pairings were not sufficient to

[174] Including a square-root transformation, using a time window within 1.05–3.05 sec following stimulus onset, and an amplitude criterion of .008 μS.

establish differential autonomic conditioning unless subjects were aware of the contingency, the most suitable measure of which was a short recognition postconditioning questionnaire. Their conclusion was that controlled cognitive processes are necessary for human autonomic discrimination in classical conditioning.

3.1.3.3 EDR and information storage

Besides their role as concomitants for cognitive processes during information uptake, processing, and establishing learned relationships, EDRs are also discussed as possible indicators of memory storage and retrieval. However, appropriate results are not unequivocal. Studies reviewed by Raskin (1973) point to stimuli which were accompanied by a larger EDR amp. having a greater probability of being transferred to long-term memory (LTM), but the same is not true for STM. This was also confirmed by a study performed by Corteen (1969), who divided 60 subjects into three groups. The first group had to recall 21 or 15 words immediately, the second group after 20 min, and the third group after two weeks. The point-biserial correlation between the log SCR amp.[175] following the stimulus presentation during acquisition and the recall criterion increased from .13 to a range of from .23 to .40 with the interval between learning and recall. This result was interpreted as showing the establishment of LTM traces for highly activating stimuli, while autonomic activation does not influence STM, where stimuli differing with respect to this activation are recalled with about the same probability.

The information processing model provided by Öhman (1979) which tries to integrate the concepts of OR, learning, and attention allows the derivation of a specific indicator function of the EDR for processing in STM and LTM. As already mentioned in Section 3.1.2.1, the EDR components that appear during classical conditioning can be regarded as concomitants of specific cognitive processes within this model. A new and/or unexpected stimulus being not represented in the STM will elicit an OR, and initiate concurrently analyzing and processing of the properties of the stimulus. This processing includes search and finally storage in LTM, which is connected with "cognitive effort" (Kahneman, 1973). Therefore, Stelmack, Plouffe, and Winogron (1983a) concluded that the high recall rate of unusual stimuli is due to an increased amount of energy needed during their storage, which had been available because of the strong OR following those stimuli.

This hypothesis was tested by Stelmack et al. (1983a) in three experiments on the role of the OR in recognition memory for pictures and words, presented for 3 sec on slides. The stimuli had been dichotomized with respect to their frequency of recall. In the first experiment, 60 subjects of both genders were assigned to one of four groups of stimuli. After 10 trials a test stimulus of the alternate representational form was

[175]Recorded as SR by the use of a Wheatstone bridge, presumably using AC, transformed into log SCR. Ag/AgCl electrodes, saturated with 3% NaCl solution, were screwed into the armrest of the subject's chair, fitting to the palm of the right hand.

introduced. The SCR amp.[176] was significantly higher in the group which received high-recognition memory pictures than in the other groups. Thus, the connection between the electrodermal OR and the recall probability could only be shown for figures. In both subsequent experiments, 56 subjects received 12 repetitions of a picture or word stimulus which they either recognized or failed to recognize. The initial SCR amp. was higher for the notrecognized than for the recognized stimuli. This finding was replicated in a third experiment with 40 subjects. Stelmack et al. (1983a) interpreted these results as being due to a "priming" effect, indicated by a relative SCR decrement to stimuli which had been recognized.

An attempt to combine emotional properties of stimuli with the role of EDA during information processing was made by Öhman et al. (1989). They exposed two groups of 20 subjects each to pictures of angry and happy faces taken from the Ekman and Friesen set (Ekman, Friesen, & Ellsworth, 1972) while SCRs[177] were measured. Subjects were given the opportunity to habituate to unmasked as well as masked versions of these faces. An acquisition phase followed in which one group was shock conditioned to angry faces and the other one to happy faces, which had a clear effect on the subsequent test trials. When the conditioned stimuli were masked with neutral faces, at least a portion of the differential responding survived backward masking in angry faces but not in happy ones. The authors concluded that responses conditioned to visual stimuli can be elicited very early in the informational chain of events, even if its access to awareness is blocked through backward masking. However, this effect appeared to be specific to biologically fear-relevant stimuli like angry faces, since it did not show up in happy faces despite an equal amount of conditioning for these pictures. Thus, emotional stimuli may be capable of evoking physiological responses after a very quick stimulus analysis, and even if the stimuli are blocked from entering consciousness. Investigating the specific role of emotional and cognitive factors in eliciting electrodermal concomitants to stimulation requires highly controlled experimental research, paralleled by neurophysiological modelling, an example of which will be given in Section 3.2.1.2.

3.1.3.4 Hemispheric asymmetry and electrodermal lateralization

Despite early reports on EDA asymmetry appearing in the 1920s, appropriate observations did not elicit much interest until the 1970s, since EDA was mainly considered as a measure of nonspecific arousal under reticular control (Freixa i Baqué, Catteau, Miossec, & Roy, 1984). Together with recent changes in arousal theory (Sect. 3.2.1.2) and more refined knowledge of CNS origins of EDA (Sect. 1.3.4.1), a growing interest in hemispheric specialization stimulated various studies of electrodermal lateralization. In accordance with this development, topics changed from researching simple

[176]Recorded with standard methodology using Beckman miniature electrodes and KY-gel, within 1–5 sec following stimulus onset, using an amplitude criterion of .024 μS, and squareroot transformed.

[177]Method of recording not reported. A range-correction was performed.

left-hand/right-hand differences in EDA (e.g., Fisher, 1958; Obrist, 1963) to measuring EDA during typical hemisphere-specific tasks, either in the intact brain (Gross & Stern, 1980; Hugdahl, Broman, & Franzon, 1983; Lacroix & Comper, 1979; O'Gorman & Siddle, 1981) or in patients with unilateral brain lesions (Sect. 3.5.2.2). In addition, specific interest was given to bilateral EDA recordings in affective disorders and in schizophrenics (Sect. 3.4.2.3). A thorough review has been provided by Hugdahl (1984), while Miossec, Catteau, Freixa i Baqué, and Roy (1985) have extensively discussed the methodological problems involved in EDA lateralization research.

Most of the recent studies in this area used the hypothesis of cognitive specialization of both hemispheres as a theoretical background. According to the traditional view, verbal stimuli should be mainly processed in the left hemisphere, while visual-spatial stimuli are thought to be typically processed in the right hemisphere. However, this traditional verbal/nonverbal dichotomy has been questioned in several reviews (e.g., Dimond & Beaumont, 1974; Bradshaw & Nettleton, 1981). According to these views, there seems to be more a quantitative rather than qualitative difference between hemispheres, with a high degree of duplicated function, and with hemispheric specialization as an additional feature.

Lacroix and Comper (1979) examined patterns of bilateral differences in SCR amp.[178] as a function of verbal versus spatial tasks in three experiments, performed with a total of 40 female subjects. The first two experiments used right-handed subjects, and left-handed subjects were used in the third experiment. Despite some differences in experimental techniques, EDA lateralization effects of both types of tasks were consistently found with dextral subjects. However, when sinistral females or tasks activating both hemispheres (mental arithmetic or music) were used, laterality effects disappeared.[179] Since in dextral subjects these authors observed smaller SCR amp. in recording contralateral to the activated hemisphere, they reported that the neurophysiological mechanism responsible for bilateral EDA differences was contralaterally inhibitory in nature.

This hypothesis is supported by ablation studies with clinical populations (Darrow, 1937a; Holloway & Parsons, 1969) as well as by animal experiments using stimulation techniques (Wilcott, 1969; Wilcott & Bradley, 1970). In addition, more recent studies performed by Boyd and Maltzman (1983), Ketterer and Smith (1982), and Smith, Ketterer, and Concannon (1981) report EDA bilateral differences in subjects solving hemisphere-specific tasks. However, different lateralization effects appeared in tonic and phasic EDA parameters. While recording EDA bilaterally,[180] Smith et al. (1981) presented 64 right- and left-handed subjects of both genders a counterbalanced series of 31 slides depicting objects spatially and 31 slides describing the same objects verbally. Stimulus-specific EDRs (within 1–4 sec following stimulus onset) showed no laterality differences in amplitudes. Instead, the hand contralateral to the activated hemisphere

[178]Measured between the distal phalanges of two fingers from both hands using standard methodology.

[179]In sinistrals, lateralization is not as prominent as in dextrals (Dean, 1981).

[180]Measured from medial phalanges of both hands as SR with standard methodology, transformed to SC and square-root transformed.

yielded lower amplitudes of NS.EDRs, which appeared outside the time window. Supposedly the contralateral inhibition of EDA acts as a contrasting phenomenon. When tonic EDA is reduced on the body side opposite to the activated hemisphere, phasic EDRs (probably as concomitants of motor preparation, Sect. 1.3.4.1) yield higher amplitudes, thus displaying a true level dependency (Sect. 2.5.4.2).

Gruzelier, Eves, and Connolly (1981a) investigated lateralization effects on electrodermal habituation in three experiments with a total of 109 subjects (minor surgery patients, medical students, and hospital staff) of both genders, 9 of which were sinistrals. At the beginning of a series of 1 kHz tones (70 or 90 dB with varying ISIs), SCR amp. recorded from the left hand (with standard methodology using KCl paste) were higher and habituated faster than those taken from the right hand, while a lower NS.SCR freq. appeared during this process. By contrast, slower habituation appeared together with a higher spontaneous EDA in the right hand. Therefore, inhibitory influences from the left hemisphere could have evoked both a lower NS.SCR freq. and a faster habituation ipsilaterally, as well as the reduction of SCR amp. on the contralateral body side. Thus, the hemispheres may differ in the polarity of their influences on EDA, the left being predominantly inhibitory while the right acts excitatorily.

Besides the frequently assumed contralateral inhibition, an additional ipsilateral facilitation of EDA is under investigation. Myslobodsky and Rattok (1977) measured bilateral tonic and phasic EDA[181] during four different tasks from 14 subjects (12 dextral and 2 sinistral). The authors formed a kind of range-corrected (Sect. 2.3.3.4.2) asymmetry index (EDA right – EDA left)/EDA max, which significantly differentiated between reactions to the visual and to the verbal tasks in dextral subjects.

In contrast to the results obtained by Lacroix and Comper (1979), Myslobodsky and Rattok (1977) found increases of SCR amp. contralaterally to the stimulated hemisphere. However, they did not discuss their results on the basis of a contralaterally excitatory control (as might have been expected). Instead, they claimed an ipsilateral control mechanism with respect to the close association of EDA and OR (Sect. 3.1.1.1). This implies that the less informed (and perhaps competent) a system is, the higher the electrodermal OR generated by a stimulus should be. Thus, higher EDR amp. should be generated by the ipsilateral hemisphere which is not familiar with the kind of stimuli presented. However, this interpretation could only be true if information processing is strictly separated within the hemispheres, which is highly improbable, as outlined at the beginning of this section. Overall, the empirical support for the hypothesis of contralateral excitation is less than that for contralateral inhibition (Hugdahl, 1984, p. 389).

[181] Recorded as SR using standard methodology (except a current density as high as 20 $\mu A/cm^2$) from the distal phalanges of two fingers of each hand, transformed into SC values. Unfortunately the EDA values reported in the results section remain ambiguous with respect to their unity and magnitude. The tasks were: visual-imagery (15 slides, 9 of them with sexual content, with subsequent imaging); verbal-analytic (series of words, from which numbers had to be selected for calculation); auditory; and light stimulation.

As an alternative to bilateral presentations of hemisphere-specific cognitive tasks, the so-called visual half field (VHF) technique has been used to study asymmetries in the intact human brain (Kimura, 1973; Springer, 1977; Beaumont, 1982). This technique makes use of the anatomically crossed visual pathways of the CNS, with the nasal part of the retinal image projected to the contralateral visual cortex, and the temporal part to the ipsilateral visual cortex. Therefore, stimuli flashed briefly either to the left or to the right of a central fixation point are transmitted only to the contralateral hemisphere, even though the corpus callosum is intact. However, stimuli must be flashed briefly, before saccadic eye movements can interfere.

To establish relationships between electrodermal OR habituation and hemispheric asymmetry, Hugdahl et al. (1983) used the VHF technique, presenting 15 verbal and 15 spatial stimuli in randomized order to the left and right VHF of 20 right-handed subjects of each gender. They recorded SCL as well as SCRs with standard methodology from the medial phalanges of both hands. Results showed significantly larger mean SCR amp. (and slower habituation) to the verbal stimuli than to the spatial ones when stimuli were presented in the left VHF. This result was reversed when stimuli were presented in the right VHF. However, this was only true when stimuli were presented in a 6.0^0 angle of projection, and not in an additional 2.5^0 condition. There were no differences between left- and right-hand recording, and the SCL yielded no significant effects except a linear decrease over time. Thus, the electrodermal OR system was influenced differentially by CNS laterality of processing stimuli. However, there was no direct connection with electrodermal lateralization.

Despite a great number of positive and even partially replicated results, Hugdahl (1984), in his review of the literature, concluded that bilateral electrodermal recordings cannot be unambiguously related to the phenomenon of hemispheric asymmetry. Bilateral differences in EDA are small and easily distorted, thus requiring optimal experimental conditions (Hugdahl, 1984, p. 389). Therefore, the different techniques used, as outlined above (e.g., unilateral vs. bilateral presentation of stimuli), may have been counterproductive in supporting a generalized hypothesis of hemispheric influences on EDA. The most important factors influencing EDA lateralization can be summarized as follows:

(1) Stimulus complexity. Most studies used spatial vs. verbal stimuli without assessing their possible differences with respect to complexity. The higher the information content of a stimulus, the greater the probability for the processing to occur in both hemispheres. Therefore, the use of abstract words (as, e.g., used by Prior, Cumming, & Hendy, 1984, in a dichotic listening paradigm) could be advocated, and spatial stimuli should be reduced to their geometric elements.[182]

[182]It is, however, questionable whether geometric figures are really typical for right-hemisphere processing, since cognitive representation of abstract material may be more prone for left-hemisphere processing.

(2) Emotional significance of stimuli. There has been a lot of debate concerning lateralization of processing emotions within the CNS. In general, there is a high probability that the right hemisphere plays an important role in mediating emotional processes (summarized by Gainotti, 1979). A more differentiated view has been advocated by Dimond, Farrington, and Johnson (1976), who state that each hemisphere has its own distinct emotional vision of the world, the right hemisphere being more unpleasant and horrific than the left one. However, this typical right-hemisphere world view is usually suppressed. This idea is only partly in accordance with Tucker's (1981) view that the left hemisphere is inhibitory or at least regulates the right hemisphere, which is in turn regarded as being primarily involved in processing emotional material. Thus, emotional content of stimuli is considered to be an important variable.

(3) Stimulus duration. It is generally accepted that transfer time between both hemispheres is below 1 sec (summarized by McKeever & Gill, 1972). Nevertheless, some authors used presentations of 6 sec (e.g., Williams, Parsons, & Strayer, 1981), 10 sec (Myslobodsky & Rattok, 1977), 15–25 sec (Smith et al., 1981; Meyers & Smith, 1986), 1 min (Smith, Gatchel, Korman, & Satter, 1979), or even 5 min (Ketterer & Smith, 1977). These stimulus durations clearly facilitate processing within both hemispheres, thus obscuring CNS laterality interpretations of EDA results.

(4) Gender of subjects. Kimmel and Kimmel (1965), Ketterer and Smith (1982), and Boyd and Maltzman (1983) reported smaller lateralization effects from males than female subjects. Kimura (1969), Rizzolatti and Buchtel (1977), and Bryden (1979) yielded smaller effects with the VHF technique in female than in male subjects, especially with nonverbal stimuli (Sect. 2.4.3.2). Based on these results, differential gender-specific cognitive mechanisms were thought to exist (e.g., female subjects are more prone to use verbal strategies even when processing spatial stimuli). However, Román et al. (1989) showed that gender differences in EDA lateralization disappeared when subjects were grouped into right-handed and left-handed responders. Since there had been a higher percentage of left-handed responders among males in two previous studies of Román et al., results on gender differences in EDA lateralization may have been obscured by a gender-specific asymmetry in electrodermal reactivity.

(5) Handedness of subjects. Sinistral as compared with dextral persons show either an opposite lateralization or no left/right differences when given hemisphere-specific tasks (Bryden, 1965; Springer & Deutsch, 1981; Annett, 1982). Hécaen and Sauguet (1971) pointed out that only sinistrals having a familial history of left-handedness differ from dextral subjects in lateralization experiments. Appropriate differences due to familial handedness were also shown by Smith et al. (1981) in their study described above.

(6) Lateralization of skin thickness and sweat gland activity. Since the skin of the dominant hand is more frequently physically stressed than the contralateral one, its stratum corneum is likely to be thicker, thus increasing the SRL. This may have consequences for a differential level dependency of both hands (Sect. 2.5.4.2). Furthermore, dextral subjects show more sweat gland activity on their right arm than on their left one, which is, however, possibly a concomitant of its greater muscular activity (Sect. 1.3.4.1), as supposed by Ogawa (1984). Such a lateral dominance of sweat gland activity could not be found with sinistrals, which may partly explain the weak relationship between handedness and electrodermal laterality (Miossec et al., 1985).

The above-mentioned dependence of electrodermal lateralization on emotional stimulus qualities has been investigated by Meyers and Smith (1986). Twenty-eight right-handed subjects of each gender (all having two right-handed biological parents) were presented two positive (a woman laughing and a baby cooing) and two negative (a woman crying and a woman screaming) emotional acoustic stimuli of 24 sec duration each. The same stimuli integrated and modified by means of a 1 kHz tone served as neutral control stimuli that were presented prior to the test phase. Critical stimuli were given twice in randomized order: under an "affective" instruction (focus on the feelings), and under a "cognitive" instruction (think of how to react to the sound). A significant interaction between body side and instruction was observed in the analysis of the largest SCR amp.[183] response, yielding a greater relative activity in the "affective" condition on the right side and greater relative activity in the "cognitive" condition on the left side, regardless of stimulus kind and gender of subject. These results point to the possibility that the request of an affective stimulus processing may be more important for EDA lateralization than the emotional content of the stimulus material itself, a finding which is at variance with Myslobodsky and Rattok's (1977) interpretation reported above.

In addition, electrodermal lateralization may be influenced by the state of general arousal (Sect. 3.2.1.1). Obrist (1963) suggested that low levels of arousal facilitate EDA asymmetry, whereas alerting or stressing the subject decreases the size of unilateral differences in EDA.[184] Freixa i Baqué and de Bonis (1983) found extremely high values of electrodermal asymmetry during sleep in four male subjects[185] as compared

[183]Recorded as SR (9.66 μA/cm^2) with Ag/AgCl cup electrodes and .05 molar NaCl paste from the pretreated (rubbing with isopropyl alcohol and drying) volar middle phalanges of the first and middle finger of both hands, transformed to SC.

[184]Recorded from five male subjects with two Fels dermohmeters using a constant current of 70 μA each, with zinc/zinc sulphate electrodes and electrode jelly (unspecified). Electrode positions were the center of each palm and the midline of the chest. Separate measures of SRL and mean NS.SRR amp. were taken during rest and during a serial learning task (as an appreciable stressor) on each of 24 or 36 days.

[185]Recorded during three consecutive nights as NS.SPRs with standard methodology and Beckman paste, with an unusually low time constant of .6 sec (Sect. 2.1.4). Monophasic and diphasic SPRs (Sect.

to results from waking states obtained in other studies. To quantify asymmetry, they used a laterality coefficient (LC) proposed by Birkett (1977):

$$LC = \frac{EDR_{right} - EDR_{left}}{EDR_{max}} \tag{52}$$

To obtain this coefficient, right/left differences for each pair of EDR amp. are categorized as either higher on the right than on the left side (R > L) or vice versa (L > R), excluding differences which do not exceed a specific criterion.[186] The overall LC during sleep was 80% as compared to 9% during stress, and 18% during nonstressful situations (de Bonis & Freixa i Baqué, 1980).

Various electrodermal lateralization effects appear in psychiatric disorders. Studies of EDA lateralization related with hemispheric dysfunctions in schizophrenia will be reported in Section 3.4.2.3. There are results pointing to endogenous depressives (Sect. 3.4.1.3) showing an inverse EDA lateralization pattern due to a hyperactivity of their right hemisphere (Freixa i Baqué et al., 1984). Psychopaths (Sect. 3.4.1.2) and even patients suffering from cardiovascular disorders (Sect. 3.5.2.3) have also yielded specific electrodermal lateralization effects. Therefore, further research in this field will not only provide techniques for investigating hemispheric specialization in healthy subjects, but also for testing neuropsychological hypotheses in the area of psychopathological research.

3.2 Generalized psychophysiological states

In contrast to Chapter 3.1, in which the use of phasic EDA parameters was discussed as event-related concomitants in various psychophysiological paradigms, the present chapter is concerned with those paradigms in which EDA is an indicator of more general psychophysiological states such as general and motivational arousal, including sleep (Sect. 3.2.1), as well as states of emotion and stress (Sect. 3.2.2). The use of EDA in some of these fields has been previously reviewed in the readers of Prokasy and Raskin (1973) and Gale and Edwards (1983). Therefore, the present chapter's focus is on the theoretical background, including a comprehensive neurophysiological modelling of different kinds of EDA (Fig. 48, Sect. 3.2.1.2), and on providing some typical and more recent studies as examples of the use of EDA in different fields.

2.2.3.1) exceeding .2 mV in at least one of the two hands were evaluated; differences less than .1 mV between both hands were not considered.

[186]It is recommended that the same values as for the amplitude criterion (Sect. 2.3.1.2.3) be used.

3.2.1 Electrodermal indices of arousal

3.2.1.1 EDA as an indicator of general arousal

Although unidimensional arousal concepts that dominated the 1950s have been supplemented or replaced by more differentiated neurophysiological views of arousal processes (Sect. 3.2.1.2), tonic EDA parameters are still used as indicators of general arousal.[187] Unidimensional concepts in arousal theory (e.g., Lindsley, Schreiner, Knowles, & Magoun, 1950) proceed from the RF and its sensoric inflow as well as its projections into cortical, hypothalamic, and thalamic areas; these are labelled the Reticular Activating System (RAS), the neuroanatomical substrate of nonspecific arousal. Stimulating the RAS through sensory input or by direct electrical stimulation of the RF leads to an arousal with clearly observable EEG changes. Thus, the so-called EEG α-blockade or increase of fast EEG components (β-activity) is regarded as the classic marker variable for general arousal processes. Closely related to this concept was the observation of an inverse U-shaped relationship between arousal and performance or behavioral efficiency (Malmo, 1959), which gave rise to the hypothesis that task specific, optimal arousal was of moderate level within the assumed arousal continuum.

Following Duffy's (1951) energetic view of general arousal as an organic overall excitation, it should be possible to quantify arousal processes not only by means of CNS indicators but also by using parameters of the ANS and the endocrine system. With respect to ANS variables, EDA has for a long time been the most frequently used indicator of arousal in psychophysiological research (Duffy, 1972). Thus, the focus of the following discussion will be on the indicator functions of the different EDA parameters with respect to general arousal.

In several experiments on the effects of physical strain on physiological and performance variables elicited by rotation, Silverman et al. (1959) showed that in a certain range of the presumed arousal continuum tonic and phasic EDA parameters can show differential indicator functions.[188] In a first gravitational experiment, five subjects were given accelerations of 2.5 g-force and 4 g-force, as well as .4 g-force prior to the loss of consciousness. The average EDA variations showed increasing arousal under higher acceleration; the stimulation of the subjects with 2.5 g-force led to an increase of both the mean SRR amp. and the NS.SRR freq. Under 4 g-force the mean SRR amp. was slowly reduced, while the NS.SRR freq. kept increasing. This progress was more clear under a gravitational condition of .4 g-force prior to the loss of con-

[187] Older results in the area of EDA and arousal can be found in Duffy (1972) as well as in Raskin (1973). More recent descriptions of activational, attentional, and cognitive phenomena with respect to their physiological concomitants can be found in the second volume from Gale and Edwards (1983). A methodologically oriented, strongly generalized integrative presentation of psychophysiological paradigms is given by Fahrenberg (1988).

[188] In these experiments, EDA was taken as SR from the soles of the feet with 2 × 4 cm lead electrodes and KY-gel on acetone cleaned sites.

sciousness, where the SRR amp. strongly decreased, while the NS.SRR freq. showed a further increase. In another experiment, 15 subjects were given a tracking task under 2 g-force and 4 g-force. In accordance with the above-mentioned hypothesis of an inverse U-shaped-relationship between arousal and performance, an improvement in psychomotor performance was displayed in states of medium arousal as defined by means of EDA parameters, while an increase in arousal above this level led to a performance decrement.

The results from Silverman et al. (1959) were largely confirmed by Burch and Greiner (1960), who systematically varied arousal with pharmacological substances (Sect. 3.4.3). As a result, an S-shaped relationship was displayed between the NS.SRR freq.[189] and the arousal level, while SRRs in response to an electrical stimulus followed an inverse-U function. The administration of sedatives led, in a dose- dependent manner, to a decrease in the NS.SRR freq. and to a lowering of the amplitudes of stimulus dependent SRRs. Simultaneously, a reduction of the fast EEG components was observed. The increasing arousal of the subject following injection of stimulants led to an increase of both the NS.SRR freq. and the stimulus-dependent SRR amplitudes. An increase of the dose or a chronic administration of the stimulant resulted in a dissociation of both EDA parameters, since the NS.SRR freq. increased further, while the amplitudes of the specific SRRs decreased. In the highest-arousal state, the subject displayed very few reactions to the standard stimulus.

Both Silverman et al. (1959) and Burch and Greiner (1960) deduced from their results different courses of the indicator functions for tonic and phasic EDA parameters. The number of NS.EDRs was assumed to show an approximately linear relationship to the CNS arousal state, while, by contrast, the observed curve of SRR amp. has found to correspond to the inverse-U function which also appears in the relationship between arousal and performance. The lowering of the EDR amp. in the excited states (cf. Malmo, 1959, Footnote 3) may reflect a breakdown in the adaptive, goal-oriented behavior as a result of a deficit in selective processing of environmental stimuli at the upper end of the assumed continuum. As an appropriate electrodermal indicator for arriving at the other extreme of the arousal continuum, Christie and Venables (1971) suggested the BSPL, which is the SPL reached after a long resting period (Sect. 2.3.2.1).

Empirical evidence for different tonic EDA parameters yielding some kind of differential validity with respect to arousal processes is given by Walschburger (1976) in his study already described in Section 2.5.2.1.2. He found that the SCL, being originally tonic in nature, shows only a slight increase as a result of arousal processes. Furthermore, a clear relationship between SCL and arousal can be seen only in the moderate range of arousal, with a tendency towards ceiling effects (Sect. 2.5.4.1). On the other hand, the NS.SCR freq. which is derived from phasic electrodermal phenom-

[189]Recorded with lead electrodes without electrode paste from the inner sides of the fingers against the forearm. A bipolar parieto-occipital EEG was recorded in parallel to the SR measurement. This study, though often cited, was performed with only one subject.

ena shows a steady rise from rest values of practically zero, over a wide range of the arousal continuum.

An attempt to take into account general as well as localized aspects of arousal (Lindsley, 1951), with respect to psychophysiological measurement, was made by Haider (1969, 1970). In his hierarchical arousal model, different physiological parameters taken from the CNS and ANS were assumed as indicatoring different levels of generality of arousal phenomena. For example, EDRs were regarded as indicators of localized phasic arousal processes, while tonic electrodermal parameters were supposed to be used as a measure of generalized arousal. This model provides an appropriate rationale for deriving tonic EDA parameters from phasic electrodermal changes (Sect. 2.3.2.2).[190] A predicted appearance of EDRs indicates the existence of numerous phasic arousing processes which underlie a heightened, general tonic arousal.[191]

Above all, the discussion of physiological variables as indicators of arousal suffers from a general lack of clarity of the concept itself. Furthermore, one of the most consistent findings in arousal research seems to be the dissociation of various psychophysiological parameters. Because of various methodological problems such as the specificity and covariation of peripheral physiological (and also subjective) arousal indicators, psychophysiological research shifted. It moved away from a study type based on the assumption of a generalized unidimensional arousal concept to investigations of marginal conditions that are supposed to elicit certain arousal states and alterations as well as to research in differential psychophysiology (summarized by Fahrenberg, 1988). Despite some progress in multivariate arousal research, no satisfactory predictions can yet be made with respect to the dependence of the differential strength and direction of arousal from possible predictors such as stable interindividual differences, so that instead of global arousal concepts the development of psychophysiological microtheories of activating processes should be attempted (Fahrenberg et al., 1983).

There is, however, one hypothesis, referred to in the next section, for which at least some support is given from psychophysiological arousal research. Generally, electrodermal and cardiovascular variables seem to have different domains of validity with respect to their indicator function in different parts of an assumed arousal continuum. EDA is regarded as a sensitive and valid indicator for the lower arousal range, reflecting small, mostly cognitively conditioned variations in arousal. By contrast, HR is more suited as an indicator for the higher arousal range and for pronounced and often somat-

[190]However, Haider (1969) did not use the NS.EDR freq. but slow SP changes as an example of tonic EDA.

[191]Furthermore, considerations of a differentiated view of the role of the RF in the elicitation of EDA were made by Sharpless and Jasper (1956), which could complement the neurophysiological concepts of EDA origins described in Section 1.3.4.1, if confirmed empirically. Those authors regarded the caudal (deeper) structures as the neurophysiological correlates of tonic EDA, while the rostral (higher) components of the RF were regarded as mainly contributing to phasic EDA phenomena, which indicate orienting or attentional processes (Sect. 3.1.1.1 and 3.1.3).

ically determined arousal processes (Epstein, Boudreau, & Kling, 1975; Miezejeski, 1978; Walschburger, 1986).

The possibly differentiated areas of validity of those different systems within the ANS do not have to be directly traced back to differences in their central control. Instead, the differing dampening and regulative behavior of both systems can also play a role. So it is possible that the above-mentioned lower sensitivity of tonic electrodermal parameters in the upper range of arousal is brought about by the increasing moisturization of the stratum corneum following frequently appearing EDRs (Sect. 1.4.2.3). This would be supported by the decrease of the SRR amp. as found by Silverman et al. (1959), while the HR can depict rapid arousing and de-arousing processes also with high arousal and motor activity. In contrast, the HR can easily become insensitive in a low overall arousal range dependent on system compensatory regulative processes, while the electrodermal system responds to each psychological variation with a clear-cut EDR, no matter how small the variation.[192] Thus, concepts of differential indicator functions can be provided not solely for various physiological systems; additionally, specific indicator functions may be found for different parameters obtainable within one system. This seems to be at least a reasonable line of study to follow, with respect to further arousal research.

3.2.1.2 EDA during states of motivational arousal

During the last two decades, there have been several attempts to replace the above-mentioned unidimensional arousal theories by more complex systems with regard to different sources and kinds of arousal. Routtenberg (1968, 1971) formulated his two-arousal hypothesis, taking into account the arousing properties of the limbic midbrain reward structures as found by Olds and co-workers (e.g., Olds & Olds, 1965), in addition to the popular RAS (Sect. 3.2.1.1).

Though he could not give an unequivocal anatomical description of the two systems proposed, Routtenberg (1968) related his "Arousal System I" to the RAS as responsible for drive-related response energy. On the other hand, his "Arousal System II" has been related to the medial forebrain bundle (MFB) that runs through hypothalamic and telencephalic structures, influencing positive incentive or reward-related behavior.[193] The

[192]Fundamentally different neuronal trigger mechanisms of cardiovascular and electrodermal reactions were also found in rat experiments of Roberts and Young (summarized by Roberts, 1974). In a series of investigations into the effect of aversive stimuli upon approach behavior, consistent connections between the heart rate and physical movement of rats were found, which decreased over the trials, while both the SC and the negative SP component showed an ascending progress during the course of the trials. Roberts (1974) excluded all possibilities of a somatic coupling of EDA, as in, for example, overall muscular tension or breathing, and he therefore presumed motivational and/or attentional processes (Sect. 3.1.3) as causative factors in the increase of electrodermal activity.

[193]In his 1971 revision of his theory, Routtenberg was more careful with respect to the neuroanatomical structures that may underlie "System II," which was, in turn, more related to motor components of behavior.

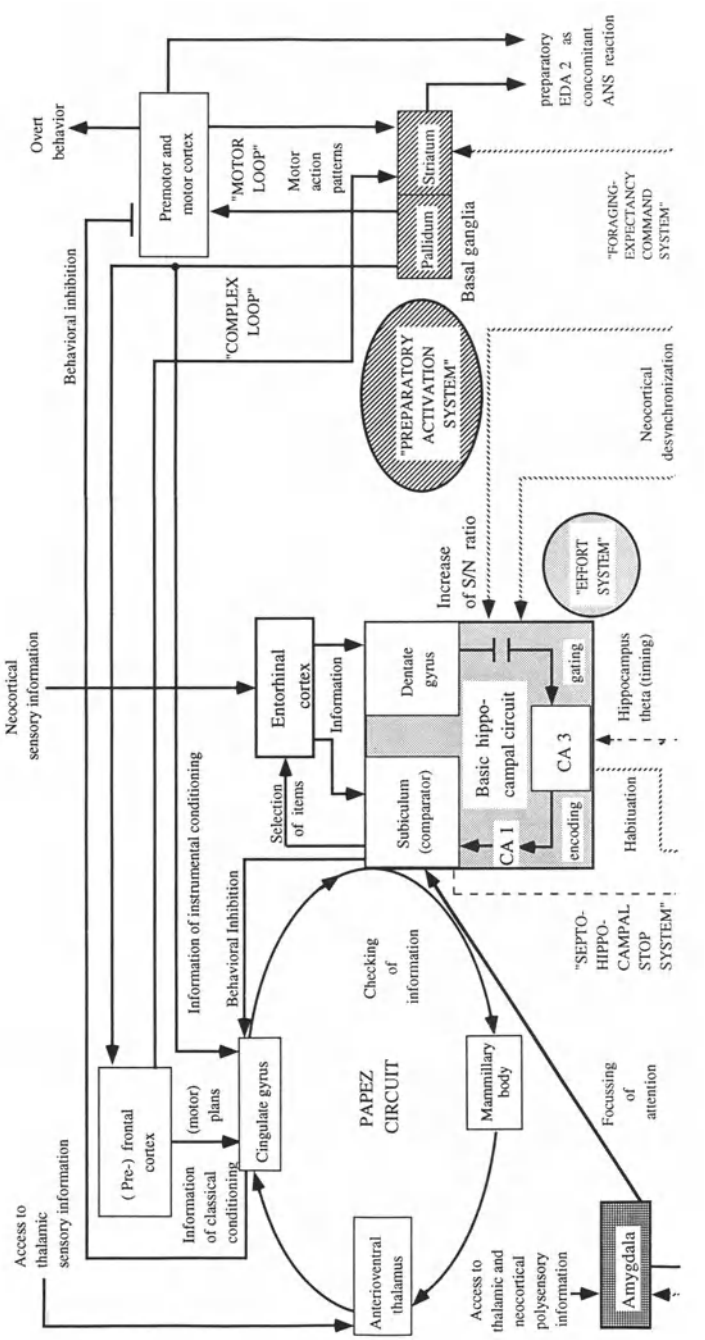
lower-right part of Figure 48 depicts the two-arousal system as proposed by Routtenberg (1968, Figure 2), indicated by the dashed connections. "Arousal I," which is more general in nature, inhibits – via negative incentive structures in the dorsal midbrain – the limbic system and thus "Arousal II," which is more motivationally determined. In turn, System II inhibits System I via septal-hippocampal pathways. Both systems have the property to suppress each other, while posterolateral hypothalamic stimulation can facilitate both systems. Since both systems can elicit EEG desynchronization, the only possibility of differentiating Arousal I and II electrophysiologically may be via the hippocampal theta as a concomitant of the action of System II. Thus, Routtenberg's formulation has not had much impact on psychophysiology, especially with respect to ANS variables. As already outlined in Section 3.1.4, both arousal systems may be connected to Pribram and McGuinness's (1975) model of information processing. "Arousal System I" in some respects resembles their "Affect Arousal" and/or "Effort" systems, while "Arousal System II" is tied to their "Preparatory Activation" system. According to McGuinness and Pribram (1980), the former is a phasic, short-lived, and more reflexlike response to input, while the latter is long-lasting in preparation to respond.

Recent research into the function of brain neurotransmitters enlightened the nature and role of this motivationally determined "Arousal II." Both noradrenaline and dopamine facilitate self-stimulation at various sites in the MFB, and also in the lateral hypothalamus, but the dopamine effect is better established (Panksepp, 1982, p. 418). By contrast, the nigrostriatal dopamine fibers running through the MFB and lateral hypothalamus also supply the basal ganglia, thus facilitating motor preparation via reciprocal connections to the premotor and motor cortex, as well as enhancing associational learning via a "complex loop" (DeLong et al., 1983) connecting basal ganglia with (pre-)frontal cortical areas. The outcomes of this motivational activation system are directed cortical-driven motor actions, on the one hand, accompanied by the preparatory EDA 2 (Fig. 6, Sect. 1.3.4.1) as a concomitant ANS reaction; and hypothalamic action patterns which typically occur during exploration and self-stimulation, on the other hand.

Figure 48 depicts the nigrostriatal dopaminergic pathway from the substantia nigra to the striatum (caudate nucleus and putamen), which also plays a role in certain neurological disorders of movement (Sect. 3.5.2.2). This system may well be regarded as responsible not only for reward and self stimulation in animals (Olds & Olds, 1965) but also as the neurochemical basis of Panksepp's (1982) "foraging expectancy command system." It also provides a more differentiated view of Routtenberg's "Arousal System II."[194]

Acetylcholine is another neurotransmitter that plays an important role in CNS activation. Since there is strong evidence for EEG arousal being controlled by cholinergic

[194]The close connections of the nigrostriatal dopaminergic fibers to motor behavior via the basal ganglia matches well with the slightly changed view of Routtenberg (1971) concerning his "System II" (see Footnote 193).


Overt behavior
Premotor and motor cortex
"MOTOR LOOP"
Motor action patterns
preparatory EDA 2 as concomitant ANS reaction
Behavioral inhibition
Striatum
Pallidum
Basal ganglia
"FORAGING-EXPECTANCY COMMAND SYSTEM"
"COMPLEX LOOP"
"PREPARATORY ACTIVATION SYSTEM"
Neocortical desynchronization
Increase of S/N ratio
"EFFORT SYSTEM"
Neocortical sensory information
Entorhinal cortex
Information
Dentate gyrus
Basic hippo-campal circuit
gating
CA 3
Hippocampus theta (timing)
Selection of items
Subiculum (comparator)
CA 1
encoding
Habituation
Information of instrumental conditioning
Behavioral Inhibition
"SEPTO-HIPPO-CAMPAL STOP SYSTEM"
Checking of information
PAPEZ CIRCUIT
Mammillary body
Focussing of attention
(Pre-) frontal cortex
(motor) plans
Information of classical conditioning
Cingulate gyrus
Access to thalamic sensory information
Anterioventral thalamus
Access to thalamic and neocortical polysensory information
Amygdala

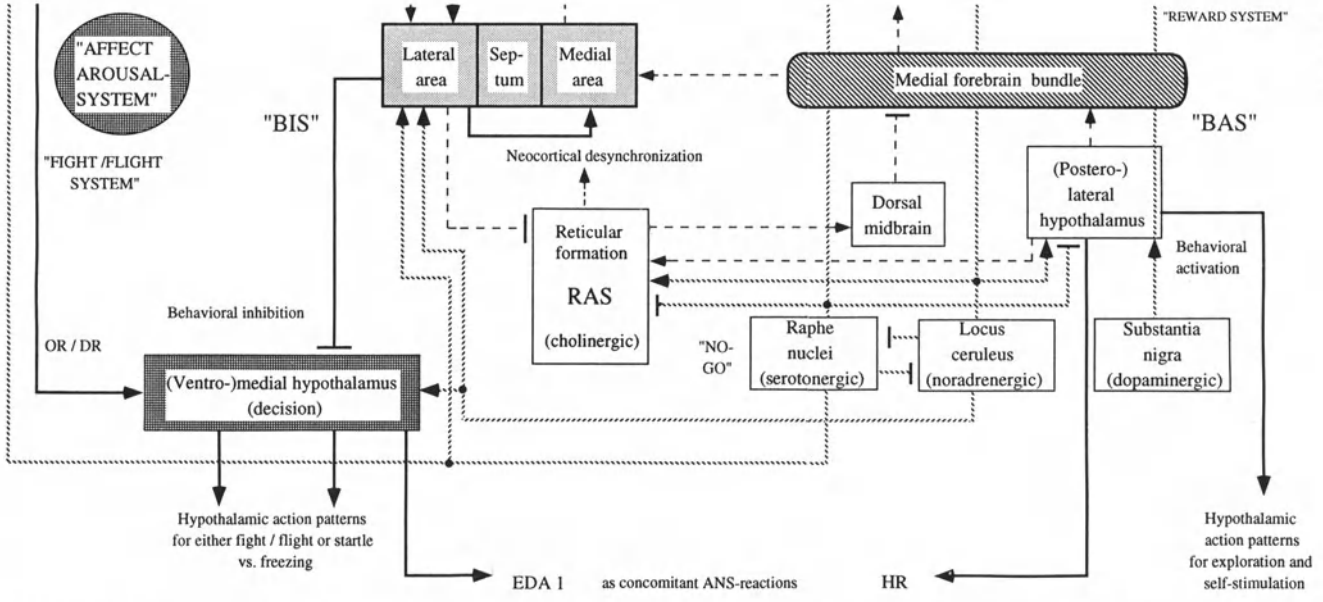


Figure 48. Integration of multidimensional arousal systems, together with their electrodermal and cardiovascular concomitans. (See text for explanations).

pathways from the RF (Warburton, 1983), its main neurotransmission function is regarded here as being cholinergic. Furthermore, the noradrenergic system facilitates RF activity, thus indirectly increasing neocortical desynchronization and hence the efficiency of cortical information processing. In turn, the serotonergic system inhibits not only RF activity but also the (postero-)lateral hypothalamus, thus reducing cortical processing and diminishing the probability of behavioral output as well. The general idea of brain noradrenaline acting excitatorily in a nonspecific way, and serotonin acting in a typically inhibitory way on motivated behaviors, is generally consistent with existing evidence (Panksepp, 1982, p. 419). Thus, the noradrenergic system which acts on cholinergic transmission may be regarded as the neurochemical basis for Routtenberg's "Arousal I."

As Figure 48 further shows, serotonergic fibers from raphe nuclei stimulate the amygdala, which is regarded as an "Affect Arousal" system by Pribram and McGuinness (1975). The amygdala receives thalamic as well as neocortical polysensory information, which may facilitate the elicitation of OR and DR together with their ventromedial-hypothalamic mediated behavioral action patterns typical for fight/flight, orienting, or startle (Sect. 3.1.1). Therefore, the amygdala-hypothalamic connection may be labelled the "flight/fight" system. The amygdala does not only increase subcortical "Affect Arousal" but also the focusing of attention by acting on the subiculum (Sect. 3.1.4.1). With respect to Routtenberg's two-arousal hypothesis, this arousing mechanism is covered in "System I."[195]

However, besides the reticular structures that have general arousing properties, and the limbic reward structures whose activation facilitates motivated behavior, Gray and Smith (1969) and later Gray (1973, 1982) proposed another arousal-related system which inhibits overt behavior and increases attention by means of mainly subcortical connections. This "septo-hippocampal stop-system" or "Behavioral Inhibition System" (BIS) has signals of nonreward or punishment as well as novel and fear stimuli as inputs (Gray, 1982). Gray (1973, Figure 6) first regarded the observed behavioral inhibition as a concomitant of the inhibiting influence of the medial septal area via the hippocampal formation on the RAS, which had also been described by Routtenberg (1968). However, in his later formulation, Gray (1982) proposed a highly complex system including the Papez circuit of the limbic system (Fig. 4, Sect. 1.3.2.2), the medial as well as lateral septal areas, and highly differentiated hippocampal substructures, which can be identified as Pribram and McGuinness's "Effort System" (Sect. 3.1.4.1), as also depicted in Figure 48.

In Gray's view, neocortical sensory information is forwarded via the entorhinal cortex to the subicular area directly, as well as indirectly via the dentate gyrus, CA3, and

[195]This is in accordance with Sokolov's (1960) view of arousal within the OR (Sect. 3.1.1), insofar as its concomitants are an RF-mediated EEG desynchronization, together with hypothalamic ANS action patterns.

CA1.[196] This basic hippocampal circuit selects particular information which has to be compared with the past (stored regularities) as well as with the future (plans). The subicular area is regarded as the comparator, and the checking of information is performed by the Papez circuit, which has direct access to sensory information via the anterioventral thalamus, as well as to stored information via the cingulate gyrus. The latter receives information concerning classical conditioning as well as planning of motor behavior from the prefrontal cortex, and information of instrumental conditioning from the basal ganglia.

According to Gray (1982), this system is continuously monitoring what should occur. If an event can be predicted, the BIS has no control over behavior. On the contrary, if the event cannot be predicted, it stops behavior via projections from the lateral septum to the hypothalamus, and also increases attention with respect to items entering the system via the entorhinal cortex. If the dentate gyrus-CA3 gate is closed as a consequence of increasing familiarity and unimportance of the stimulus, habituation is induced by CA3 to the lateral septum. As a consequence, the OR is likely to be influenced by the BIS, insofar as habituation is mediated via inhibition of the ventromedial hypothalamus by lateral septal activity (Gray, 1987). The BIS has two main outcomes:

(1) Inhibition of behavior, including instrumentally or classically conditioned ones, and even innate behavioral patterns. This is labelled "freezing" at the bottom of Figure 48, which is opposed to fight/flight or startle patterns. However, behavioral inhibition is not only induced by preventing the elicitation of hypothalamic action patterns, but also by inhibiting cortically released motor activity via the subiculum-cingulate gyrus-motor cortex (see top of Fig. 48).

(2) Facilitation of a comprehensive and accurate analysis of environmental stimuli, especially those showing characteristics of novelty. The hippocampal system switches to an active mode, so that the subicular comparator acts upon the entorhinal cortex deliberately to select items to be processed within the basic hippocampal circuit.

As shown in Figure 48, ascending noradrenergic fibers from the locus ceruleus stimulate those septo-hippocampal and hypothalamic decision centers, and serotonergic fibers from the raphe nuclei do the same with the hippocampus. Here, both monoamines open the gate between the dentate gyrus and CA3 for specific sensory information to be checked by the subiculum (Gray, 1982). They also stimulate the lateral septal area.

[196]From Latin *cornu ammonis*, which is another name for hippocampus. The hippocampus theta which is generated in the medial septal area, as already outlined by Routtenberg (1968), is transmitted to the CA3 field in the hippocampus for the sake of quantifying time within the system. Gray (1982) suggests a feedback loop from the subiculum to the medial septal area, the anatomical confirmation of which is, however, lacking. Therefore it is not included in Figure 48.

Those monoaminergic pathways are stimulated in generalized states of stress and anxiety (Sect. 3.2.2.2 and 3.4.1.1), and are inhibited by the action of "antianxiety drugs" (Sect. 3.4.3).[197] The dopaminergic pathway from the ventral tegmental area to the prefrontal cortex, in which activity is also increased during those states, is regarded as secondary by Gray, since the ventral tegmental area itself receives a noradrenergic input from the locus ceruleus (not depicted in Fig. 48).

The initial model as proposed by Gray and Smith (1969) had been directed towards explaining approach versus avoidance behavior–with an appropriate decision-making mechanism presumably located in the medial hypothalamic area–as preceded by a reward and a punishment system inhibiting each other and both facilitating general arousal. While Gray (1973, 1982) expanded the punishment system to the above-mentioned BIS, he did not pursue an opposed Behavioral Activation System (BAS) formulated earlier (Gray, 1970). However, he identified the BAS later on with the reward structures described by Olds and Olds (1965) without giving as detailed a description of its action as he did for the BIS (Gray, 1973).

Consequently, Gray (1987, p. 225) distinguished three separate mechanisms in his conceptual nervous system: a reward system which responds to signals of reward or nonpunishment by initiating approach behavior; the BIS, which responds to signals of punishment or nonreward by suppressing overt behavior; and a fight/flight system, which responds to unconditioned punishment or nonreward by activating escape or aggressive behavior.[198] BIS activity inhibits not only the activity of the reward system (which could be performed by serotonergic inhibitory action on the lateral hypothalamus as depicted in Figure 48) but also the activity of the fight/flight system via serotonergic inhibition of an output from central-gray located fight/flight neurons.[199]. Thus the role of serotonin as inhibiting all forms of activation is well established.

Gray (1982), focusing on observations of animal behavior, has always been cautious concerning psychophysiological concomitants of BIS or BAS activity. An attempt to develop systematic hypotheses concerning psychophysiological concomitants of motivation-directed arousal processes was made by Fowles (1980, 1986b). Starting from the model of Gray and Smith (1969), Fowles (1980) explicitly formulated a "three-arousal model," in which two mutually antagonistic systems determine the outcome of either behavioral activation or inhibition, while another system, which receives input from both of them, presumably increases behavioral vigor or intensity. While the latter

[197]Gray (1982) explains the anxiolytic properties of tranquilizers as well as hypnotics via their facilitating properties on the inhibitory action of GABA (gamma-aminobutyric acid) on noradrenergic and serotonergic synaptic transmission. Tranquilizers of the benzodiazepine type act directly via a specific postsynaptic receptor, while hypnotics like barbiturates and alcohol act indirectly through a blockade of picrotoxine-receptors, a substance which inhibits GABA.

[198]Gray (1987, p. 226) pointed to the difference between his own view and Panksepp's (1982) concerning fear and anxiety (Sect. 3.4.1.1), Panksepp identifying them largely with the activity of the fight/flight system, whiie Gray identified them with BAS activity.

[199]Those neurons, which are not depicted in Figure 48, receive afferent impulses from the medial hypothalamic decision center (Gray, 1987, p. 265).

system is identified with the RAS (Sect. 3.2.1.1), the Behavioral Activation System, or BAS, corresponds to the "Arousal System II" as per Routtenberg (1968), and the third type of arousal, which mediates the effects of aversive stimuli, has its neurophysiological substrate in the BIS as formulated by Gray (1973, 1982).

While Fowles (1980) did not explicitly deal with the problem of psychophysiological indicators for general arousal or "Arousal System I" – for which EEG activity may still serve as a main variable (Sect. 3.2.1) – he integrated various animal as well as human psychophysiological studies with respect to presumed autonomic nervous system correlates of BIS and BAS activity. His conclusion was that HR can be regarded as an even more accurate index of BAS activity than overt behavior and, hence, indicate positive emotional and motivational aspects of arousal. On the other hand, EDA appears to respond more to situations typically connected to BIS activity, such as those characterized by the presence of fear or punishment-relevant stimuli. Since the appropriate neural impulses for EDA are most likely to stem from hypothalamic areas, they should be labelled as EDA 1 (Fig. 6, Sect. 1.3.4.1).

The elicitation of EDA 1 is indicated at the bottom of Figure 48, where the model of a medial hypothalamic decision structure as proposed by Gray and Smith (1969) is extended according to the results from hypothalamic lesion and stimulation studies on motivated behavior (for a comprehensive summary see Stellar and Stellar, 1985). Another extension of the model concerns the possibility that the ventromedial hypothalamus is the output region for behavioral inhibition, and the (postero-)lateral hypothalamus is more likely the output region for behavioral activation. Though there is no direct evidence for a corresponding selective action of these structures on EDA and HR, the appropriate hypothetical notion for these variables is that they may be concomitants of BIS and BAS activity according to Fowles (1980). Therefore, they are added in Figure 48 as well.

The specific indicator function of EDA, with respect to BIS activity as worked out by Fowles (1980), relies on research in the field of conditioning and stress. It is mainly focused on the appearance of stimulus-dependent as well as spontaneous EDRs, but not on the EDL, which is regarded as being influenced more by peripheral physiological states (e.g., corneal hydration, Sect. 1.3.4.2.1) than by fear-evoking stimuli. Evidence for the suggested coupling of BIS and EDA is drawn indirectly from observations which has been interpreted by Fowles as directional fractionation[200] between EDA and HR during situations of nonreward (frustration) or punishment. The occurrence of directional fractionation is suggested to be dependent on response contingency. That means, in cases when the subject expects his responding not to result in avoidance or escape but in an increased probability of punishment, or, if the experimental demands are strong enough for the subject to reject active avoidance as an alternative, only EDA, but not HR, will demonstrate an acceleration.

[200] "Directional fractionation" is a term employed by Lacey (1967) with respect to the observation that different parts of the autonomous nervous system may show opposite reactions with respect to general arousal, which is a strong argument against an unidimensional arousal theory (Sect. 3.2.1.1).

As opposed to the coupling of BAS and HR, which was shown in a series of subsequent experiments (Fowles et al., 1982; Tranel et al., 1982; Tranel, 1983), only the study performed by Tranel (1983) explicitly investigated the supposed BIS-EDA coupling. Forty-eight subjects of both genders underwent six trials of a choice-RT task. During trials 2–4 they received feedback tones indicating monetary reward for success, which had been set to 100% success. During the last two trials, no tones were given. Half of the subjects were informed that this would happen, the remaining subjects were not informed. The latter, frustrated group showed a significant increase in NS.SCR freq. compared to the expected no-feedback group, which only showed the further decline of EDA that normally occurs during subsequent trials. This was interpreted as conforming with Fowles's (1980) hypothesis of EDA indicating BIS activity during frustrating nonreward, while HR was not influenced by the change of experimental conditions. However, it remains possible that the increase of NS.EDR freq. could simply be attributed to uncertainty or an increase of orienting activity that appeared as a consequence of the unannounced situational change.[201]

Although convincing evidence from experimental data is not yet available, Fowles's (1980) concept remains an interesting psychophysiological approach with respect to the differential indicator function of EDA. As opposed to HR, which responds to positive hedonic motivational states but not to aversive stimuli, NS.EDR freq. does not correspond to appetitive motivational activation (Fowles, 1988). Instead, nonspecific EDA is increased in arousal states accompanied by negative emotions, such as those elicited by aversive stimuli. However, up to now, results of multivariate psychophysiological research have not proven the existence of particular ANS variables exerting such a differential and specific indicator function (Sect. 3.2.2.1). Parameters of different systems tend to covary or dissociate dependent on stimulus properties and/or individual differences (Fahrenberg, 1988). Nevertheless, developing psychophysiological hypotheses on the basis of a more differentiated neurophysiological model of activation process as performed by Fowles (1980, 1986b) may help to enlighten the hitherto unexplained relationships between arousal-related changes in frequently measured variables like HR and EDA.

[201]Fowles (1988) reported data from a doctoral dissertation performed by Fisher in 1985, examining the effects of 10, 50, and 100% success using 20 subjects per group. In contrast to a feedback-only condition, a monetary incentive condition, holding the amount of money earned constant across the different success groups, yielded significantly greater HRs regardless of the amount of success. On the other hand, NS.SCR freq. was significantly heightened in the 10% group as compared to the other ones, regardless of monetary incentive, which supports EDA not being influenced by appetitive motivational states during task performance. Sosnowski, Nurzynska, and Polec (1991) could not find an influence of manipulating monetary reinforcement on both HR and SCR amp. (measured with standard methodology) in 60 student women randomly assigned to reward, frustation, and control groups performing a problem solving task. Subjects were run in pairs; the active subject solved the problem, being observed by the passive one. During the task, SCR amplitudes decreased significantly in active subjects while being markedly increased in passive subjects. The authors interpreted this result as being in accordance with Fowles's EDA-BIS hypothesis since EDA was increased in what they called passive coping.

3.2.1.3 EDA during sleep stages

Until the early sixties it was generally accepted that spontaneous EDA practically disappears during sleep, especially in stages of slow wave sleep (Kleitman, 1963). Johnson and Lubin (1966), however, found the highest NS.EDR freq. in the deepest sleeping stages (III and IV). They recorded endosomatic and exosomatic EDA[202] during three consecutive nights from 6 epileptic patients of each gender and from 17 healthy male subjects. The analyses were collapsed over groups since no differences in EDA were found between patients and controls. While they recorded less than one SRR or two SPRs per min on average in sleep stage I and during REM sleep, an average of 2.2 NS.SRRs and 7.2 NS.SPRs per min was observed in stages III and IV. There were marked intraindividual differences between the nights of the study. However, similar patterns appeared in comparable sleep stages within a given night.

The above authors interpreted their observed differences between frequencies of spontaneous SRRs and SPRs as demonstrating a dissociation between exosomatic and endosomatic EDA during sleep, but Edelberg (1972a) pointed to potential confounding caused by local differences of recording sites. This view was supported by Broughton, Poire, and Tassinari (1965). By recording SPR[203] at six to eight sites on 25 subjects during sleep, they found an increase of spontaneous EDA during stages of deep sleep, but in addition a marked increase of SPR lat. in caudal and distal directions on the body surface.

The appearance of a maximum in spontaneous EDA during stage IV, as well as its decrease during REM phases, is regarded as a well-established result in sleep research (Jovanović, 1971; Edelberg, 1972a). This had been shown for example by Freixa i Baqué et al. (1983, Table 1), who reviewed 12 studies published between 1962 and 1976. In addition, they reported results of their own, obtained from eight male subjects during three nights (following an habituation night). Within all nights, differences in NS.SPR freq.[204] between the first and three consecutive sleep cycles were observed in a consistent manner. While total EDA (independent from sleep stages) as well as EDA during stage II were significantly lower in the first than in the following cycles, there was a significantly higher NS.SPR freq. during the REM phase in the last cycle than in the first three sleep cycles.

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[202]Recorded with Ag/AgCl sponge electrodes from the fingers of the right hand with an "inert" electrolyte and 21 $\mu A/cm^2$ for SR, and from the left middle finger to the scrubbed left forearm with Redux paste, and 1 sec time constant, for SP. Amplitude criteria: 50 Ω and 100 μV (positive or negative), respectively.

[203]Recorded with silver cup electrodes and EEG paste from acetone-cleaned sites, each pair having inter-electrode distances of 5–6 cm. They were placed palmar/dorsal; at the dorsal forearm; and over the frontal, trapezius, deltoid, biceps, extensor digiti, or other muscles, approximately along the neuraxis and peripheral nerves.

[204]Recorded with Beckman electrodes, time constant .6 sec, low-pass filtered with 15 Hz, amplitude criterion 200 μV. Results were log transformed.

Despite frequently observed differences in EDA between sleep stages, attempts to differentiate these stages by means of EDA parameters remain unsatisfactory. Koumans, Tursky, and Solomon (1968) recorded SP and SR[205] from nine male subjects during two nights of normal sleep, together with EEG and EOG. SPL and SRL did not differentiate the sleep stages, and only in SPL was there a significant change from the waking state to sleep (an increasing negativity). Instead, in both EDA recordings there were higher rates of NS.EDR freq. during stages III and IV, and lower rates during REM sleep than during the waking state. Though the authors were cautious with respect to identifying sleep stages by spontaneous EDA, they inferred from its decrease, which occured consistently about 6 min before the onset of each REM period, that it should be possible to use NS.EDRs to detect REM-phase onset (e.g., for the sake of REM deprivation).

Johns, Cornell, and Masterton (1969) found similar results in a study with 12 students and 19 convalescent surgical patients of both genders, recording SR[206] together with EEG and EOG during sleep. By means of tonic EDA parameters (SRL as well as NS.SRR freq.), they determined the time of sleep onset, disturbances during the night, and waking, with a resolution of 1–2 min. Furthermore, they obtained a rough estimate of the amount of slow wave sleep.

In a study performed with 20 male subjects, Hori (1982) showed that palmar but not dorsal SP[207] could be used as an indicator for the onset of sleep, since only the palmar SPL decreased between 3.5 min before and 1.5 min after stage I onset. Similar anticipatory changes were found in palmar SPRs and slow eye movements, suggesting the existence of a common underlying CNS mechanism.

Some results indicate that nocturnal tonic EDA is an indicator of stress-induced changes of sleep quality. Lester, Burch, and Dossett (1967) investigated the aftereffect of different stressors on EEG and SR measures.[208] After two habituation nights, 5 student subjects were studied in the following sequence: three consecutive nights before, during, and after a time-interval anticipation test used as a mild stressor; three other consecutive nights clustered around a one-day medical exam as a strong stressor; and a minimum of two nights after the semester ended. During the initial nights, the overall mean NS.SRR freq. decreased, increased in nights after stressful experiences, and was highest the night following exams. This effect was most prominent in stages III and IV and lowest during REM sleep. A maximum of nonspecific EDA was usually noted during the first and/or second period of slow wave sleep, and an average of 10–12 EDRs/min was not unusual, leading the authors to the use of the term "storm"

[205]Recorded from comparable unipolar right and left sites (thenar against forearm) with Ag/AgCl sponge electrodes; amplitude criteria 200 μV and 100 Ω, respectively.

[206]SRL and SRRs measured from different washed volar sites at two fingers, with Ag electrodes, 5 mm in diameter contact area. An AC-coupling circuit was used that resulted in biphasic SRRs.

[207]DC-recorded thenar and dorsal against a skin-drilled forearm site with Ag/AgCl electrodes and .05 molar NaCl agar-agar paste. Amplitude criterion for SPRs (negative, diphasic, and positive waves) was 250 μV.

[208]Recorded from the left middle finger by Ag/AgCl electrodes, NaCl paste, and 10 μA/cm^2 constant current. Amplitude criterion for NS.SRRs: 50 Ω.

to describe this unique electrodermal activity. However, nocturnal EDA was markedly reduced when following unexpected severe life stresses which appeared during the investigation, thus showing that a direct relationship between severeness of stress and nocturnal EDA is equivocal.

In a series of three studies on spontaneous EDRs ("storming") in stages III–IV sleep, McDonald, Shallenberger, Koresko, and Kinzy (1976) investigated the influence of various factors on nocturnal EDA. In all studies, EEG, EOG, HR, finger plethysmogram, respiration rate, and exosomatic as well as endosomatic EDA[209] were continuously recorded. In their first study done with 46 subjects, they found significant correlations between spontaneous EDA during the 5 min period of waking prior to sleep onset and "storming" rates in stage IV sleep (r = .41 for NS.SRR freq. and r = .37 for NS.SPR freq.). Twenty-one subjects were instructed to remain awake as long as possible, while the others were told to go to sleep normally. The group who remained awake showed in their subsequent sleep a highly significant greater NS.SPR freq. than did the others. In their second study, 21 male subjects performed two nights of laboratory sleep. There was a significant decrease of NS.SPR freq. as recorded during stage IV from the first to the second night. McDonald et al. (1976) interpreted this as an habituation effect, though correlations with different indices of sleep quality were not found (e.g., amount of movement). In addition, subjects were divided into subgroups of "stormers" and "nonstormers" (showing either more or less than one SPR per min in stage III–IV). "Stormers" showed significantly higher values in anxiety and egostrength measures of the MMPI as compared with "nonstormers." During the third experiment, 23 male subjects who slept two consecutive nights in the laboratory were awakened 3–7 times each night and asked if they had been dreaming. Affirmative reports of dreaming were given in 87.5% of the REM awakenings, and in 34.7% of the non-REM awakenings. However, dream reports from "storm" awakenings were reported 54.0% of the time but reported only 15.4% of the time in "nonstorm" awakenings. There were no differences in dream contents in phases with different amounts of EDA. HR and finger pulse differentiated only between REM and the other sleep stages and showed no relationships to the various sleep variables as did EDA.

McDonald et al. (1976) interpreted their results and those of Lester et al. (1967) reported above as showing an increase in tonic EDA during stages III–IV sleep that is not a by-product of deep sleep. Instead, EDA "storming" seems to be highly related to some presleep activities or events. Since EDRs also appear as concomitants of information processing during waking (Sect. 3.1.3.2), these authors suggested that "storming" during sleep may indicate releasing of information stored during the day. These processes disturbed and shortened phases of deep sleeping, as shown in the reported negative correlation between stage IV duration and EDA during sleep. A more

[209]Recorded with Beckman Ag/AgCl electrodes as SR, from the fingers of the right hand using 40 μA, and as SP between left index finger and forearm with .24 sec time constant. A time window of 1–3 sec following stimulus onset was used to determine EDR; SRP amp. was measured as the total biphasic amplitude (Sect. 2.3.1.2.1); SRR amp. was transformed to log SCR amp.

careful interpretation of the observed connection of EDA during sleep and activities during the day has been provided by Lester et al. (1967). They suggested that EDA during slow wave sleep is a continuation of EDA during wakefulness, the reason for which could be cessation of cortical inhibition during those sleep stages.

Several studies on EDA during sleep focused on orienting and habituation (Sect. 3.1.1) instead of spontaneous activity. Johnson and Lubin (1967) reported additional data obtained from 12 healthy subjects in their 1966 study described at the beginning of this section. Those subjects received a series of tones[210] starting before they went to sleep and continuing throughout the night. During sleep, the previously habituated OR following the tones appeared again. This was interpreted by these authors as indicating resistance to habituation of autonomic response not only at the upper but also at the lower end of the arousal curve (Sect. 3.2.1.1). The decrease in SPR amp. observed after 3–4 hours of stimulation (and after 380 or more stimuli) was interpreted as being more likely associated with diurnal patterns of physiological activity rather than due to true habituation.

In contrast, Firth (1973) doubted the suggested incompatibility of sleep and habituation. He pointed to the Johnson and Lubin's method of averaging responses over hourly periods as well as to the fact that long, irregular ISIs may have prevented the observation of short-term habituation. Therefore, in Firth's own study with three subjects, he compared the effect of six ISI conditions on the SPR[211] in a within-subjects design. The SPR amp. showed significant habituation in all sleep stages (II, IV, or REM) and under all ISI conditions, which also appeared in the HR response (except in REM sleep).

In an attempt to confirm the lack of habituation during sleep as found in their earlier research, Johnson, Townsend, and Wilson (1975) recorded SRRs to a series of 20 stimuli[212] during stage II and REM sleep. Data from nine subjects with complete recordings did not yield clear stimulus-dependent SRRs in either sleep stage– due to the infrequency of responses during REM– and either no SRRs or too much spontaneous EDA during stage II. Therefore, an estimate of electrodermal habituation effects was infeasible. In accordance with Firth's (1973) results, HR response habituation occurred during stage II but not in REM sleep.

McDonald and Carpenter (1975) did not find habituation of exosomatic or endosomatic EDRs in 46 subjects during the presentation of 33 tones (500 Hz, 40 dB, ISIs at random 10, 15, or 20 sec) during stage IV and REM sleep. By contrast, HR and finger

[210] 1 kHz, 30 dB tones presented over a loudspeaker for 45 sec. Amplitude criteria: 125 Ω for SRRs and 100 mV for SPRs, within a time window of 1–5 sec after stimulus onset.

[211] Recorded between scrubbed volar forearm and thenar sites with a .3 sec time constant. A 1 kHz, 1 sec tone was presented over loudspeakers with 70 dB at the subject's head, using three regular (10, 20, and 30 sec) and three randomized irregular ISI conditions (8, 10, or 12; 16, 20, or 24; and 24, 30, or 36 sec).

[212] 800 Hz, 75 dB, 1 or 2 sec duration, at 30, 45, and 60 sec ISIs. SRRs were obtained by dividing the prestimulus SRL by the maximum poststimulus resistance change occurring within 7 sec after stimulus onset, log transformed.

plethysmographic responses showed marked habituation, and also dishabituation to a tone of different frequency.

In summary, EDA during sleep does not sufficiently contribute to the differentiation of sleep stages. However, an increased spontaneous EDA, especially during deep sleep phases, can be used to indicate continuing processing of information taken up during the day. This is confirmed by observations of a larger amount of EDA during those phases following stressful days.[213]. On the other hand, results concerning electrodermal orienting and habituation during sleep remain somewhat contradictory, which makes the role of EDA as an indicator of information processing during sleep ambiguous. In sleep studies, the circadian periodicity of tonic EDA has to be considered, because it may show several maxima and minima (Sect. 2.4.2.1) as well as big individual differences (Rutenfranz, 1958). Furthermore, problems of long-term recording must be dealt with as discussed in Section 2.2.6.1.

3.2.2 Electrodermal indices of emotion and stress

3.2.2.1 EDA in emotional states

The history of ANS variables in research on emotions is a long and controversial one, starting with James and Lange, who published their psychophysiological theories of emotion at the end of the last century. The basic theoretical issues discussed in this field since then are summarized by Levenson (1988).[214] Among these are the specificity question of ANS patterns, as well as the dimensionality issue, both of which will be referred to below. Attempts to include knowledge about the role of CNS structures in elicitating autonomic responses have so far not gone beyond theories of general arousal (Sect. 3.2.1.1). Recently, a closer theoretical connection was established between neurophysiological mechanisms of information processing and motivational arousal (Sect. 3.1.3.1 & 3.2.1.2), on the one hand, and the neurophysiologically oriented emotion theory of Papez, on the other. These views have special implications for the use of EDA and thus will be briefly outlined below.

According to Papez (1937), emotional excitement is maintained via circulation of neuronal activity in the limbic circle which carries his name – the Papez circuit (Fig. 4, Sect. 1.3.2.2). Here, emotions not only can be checked against sensory information

[213] A similar result was reported by Ottmann, Rutenfranz, Neidhart, and Boucsein (1987), in their study described in Section 3.5.1.1. They recorded EDA intermittently, with standard methodology during five consecutive days and nights in the laboratory, from 24 subjects performing vigilance tasks with additional STM strain. Half of the subjects who received 80 dB white noise during work showed a significant increase of NS.SCR freq. during the subsequent nights as compared to a control group with 50 dB noise. Unfortunately, no EEG recordings were made so that results differentiating sleep states are not available.

[214] With respect to the theories of the psychobiology of emotions in general, the reader is referred to appropriate comprehensive reviews (e.g., Grings & Dawson, 1978; Plutchik, 1980; Panksepp, 1986; Schwartz, 1986).

within thalamic areas, but can also have access to ANS programs which are stored in the hypothalamus, thereby eliciting autonomic concomitants of various emotional states. Gray (1982), in his neurophysiological model of anxiety, again used the Papez circuit for processing emotionally tinged information (Fig. 48, Sect. 3.2.1.2). Gray extended the ideas of Papez with respect to the central role of the subiculum (a part of the hippocampus), which uses the information check within the Papez circuit to decide whether or not behavior should be inhibited via the septo-hippocampal stop system or the BIS. As outlined in Section 3.2.1.2, Fowles (1980) suggested EDA has specific validity with respect to BIS activity, thus providing a theoretically founded, but not yet empirically proven, background for the use of EDA as a specific indicator of anxiety states (Sect. 3.4.1.1).

As opposed to general arousal research, the psychophysiological investigation of emotional states requires measures not only at the physiological but at the subjective level as well. Therefore, even a neurophysiologically oriented inquiry into emotional processes must start with a taxonomy of emotional experience in humans (Panksepp, 1982). As already pointed out by Wundt (1896), the subjective experience of emotions has to be regarded as multidimensional. Different emotions may elicit the same amount of general arousal, thus possibly becoming indistinguishable with respect to their ANS concomitants. Therefore, in classic psychophysiological emotion research, emotional quality is determined via subjective variables, while their quantitative properties are measured by ANS parameters.

An attempt to quantify the strength of emotions with the use of EDA was made by Traxel (1960). Eighty male subjects were visually presented 20 pairs of words, for seven sec each, in sequence while recording SZ.[215] After each pair, subjects had to decide which word, if any, elicited a greater amount of emotional experience. The judgments were evaluated by paired comparisons, and the obtained strength of emotion was correlated with the differences between SZR amp. elicited by the particular words in a specially developed a posteriori paired-comparison technique. A linear relationship appeared between log SZR amp. and the psychophysically determined strength of emotion. After an additional square-root transformation (Sect. 2.3.3.3), the correlation between subjective and electrodermal measures of emotion strength increased slightly from .94 to .99.

Several attempts have been made to obtain emotion-specific patterns of physiological reactions, since Ax (1953) tried to differentiate the effects of anger and fear by means of ANS variables. In his experiment, a total of 43 subjects of both genders received two conditions in permuted order. In one of these, subjects were annoyed by an obnoxious experimenter. In the other condition, after having electric shock applied to their finger, they were frightened by accompanying sparks and the experimenter exclaiming that this was a high-voltage short-circuit. Differences between anger and fear

[215]Recorded with 600 Hz, 1 V, using a RC bridge (Sect. 2.1.5) and metal electrodes of 1 cm diameter. SZR amp. were subjected to log transformation with respect to a suggested validity of Fechner's law for EDR.

appeared on 7 of 14 physiological measures recorded, whereby anger elicited a significant increase of NS.EDR freq.[216] as compared to fear. In turn, EDL was significantly higher under fear conditions than under anger conditions. However, large individual differences were observed; for example, some of the fearful subjects evidenced higher changes in EDA than in respiration, while others showed the reverse pattern. Nevertheless, Ax attributed the observed ANS reactions to specific underlying endocrine patterns as outlined in the next paragraph.

Methodological issues in this kind of peripheral physiological differentiation of emotions have been recently discussed by Wagner (1989), who also summarized results on anger versus fear and on other emotions. A systematic comparison of results taken from eight multivariate studies attempting to differentiate experimentally induced anger and/or fear by means of ANS variables, including Ax's (1953) study, was performed by Stemmler (1984, 1989). In most of the studies, both emotions yielded higher cardiovascular output (increases in HR and blood pressure) than resting conditions. An elevated NS.EDR freq., which was found for both emotions in one study, did not differ from resting condition in another study. Four studies with a direct comparison of anger and fear yielded an elevated HR, two of them a decrease in diastolic blood pressure, lower tonic EMG values, and an increased SCL during fear. The two studies that directly compared NS.EDR freq. yielded opposite results. In summary, the so-called adrenaline-noradrenaline pattern under anger versus adrenaline pattern under anxiety, as supposed by Ax, could be only partially confirmed.[217]

In his own experiment, Stemmler (1989) induced three emotions (fear, anger, and pleasure) to 42 female subjects in a repeated-measures design.[218] Various peripheral physiological measures including skin conductance[219] were taken continuously during the induction of emotions and during interspersed resting phases as well. In addition, several standardized ratings of emotional states were applied. These subjective measures yielded their most pronounced results in the appropriate situational context. However, their specificity was less during fear and anger conditions than during pleasure condition. Out of the 34 parameters that were extracted from the physiological

[216]Recorded with 60 Hz AC and a bridge from volar finger surfaces, evaluated as SY, using 1 μS as amplitude criterion.

[217]Ax (1953) did not obtain measures of catecholamines but concluded that the ANS pattern under anger resembled the expected response to adrenaline and noradrenaline injections, while the ANS pattern under fear resembled the response had adrenaline been injected.

[218]Fear was induced by tape presentation of a fear-evoking short story accompanied by an unannounced darkening of the room; anger was induced by presenting a series of anagrams developed by Boucsein and Frye (1974), most of which were unsolvable; the insoluble nature of the anagrams was not detected by the subjects during their presentation; pleasure was induced by positive reinforcement and the announcement of increased payment at the end of the study.

[219]Recorded with standard methodology from the left-hand's fingers. To obtain an additional objective measure of the "forehead anxiety sweat," another EDA recording was taken from the forehead. Electrodes were fixed by means of histoacryl (Sect. 2.2.2.1); the evaluation of EDA followed Thom's (1988) method (Sect. 2.2.4.2).

recordings, the 14 which significantly differentiated between the reference phases for the three emotions were included in a discriminant analysis, which resulted in a highly significant rejection of the null hypothesis of profiles for equality of the three emotions. Multivariate comparisons yielded low EMG values together with peripheral vasoconstriction, low skin temperature, and a low SCL taken from the hand but an increase of forehead SCL during the fear state. During anger, the forearm EMG and the vasodilatation at the hand and the forehead were increased, and an increase of forehead SCL appeared, too. The NS.SCR freq. did not belong to the variables discriminating fear and anger. Though SCL as measured from the hand was lower under fear and showed some parallel with Ax's findings, Stemmler's results did not totally fit those of Ax (1953) with respect to EDA. The pleasure condition yielded an increase of the hand's skin temperature and a decrease of forehead SCL.

To obtain valuable information concerning the specific role of electrodermal parameters in further multivariate emotion research, experimental conditions have to be chosen which enable a direct comparison of different emotions. The problem of how to equate qualitatively different states of emotion with respect to their quantitative properties is central. In addition, marginal conditions of the experimental design, such as permutation of the sequence of different emotions, may have a strong influence on results. Various response specificities, observed by Ax (1953) and systematically treated by Engel (1972) and Fahrenberg (1988), further complicate multivariate research of emotional states.

Another potential differential indicator function for EDA in emotional states stems from research on emotional expression, which not only plays a role in normal psychology, but also in psychopathology, especially in schizophrenia (Sect. 3.4.2.3).[220] The hypothesis that facial expression is not only a concomitant of emotion but also has a role in regulating the emotional experience itself dates back to Darwin, and was taken up again by Gellhorn (1964) and Izard (1971). These authors suggested that the facial actions trigger central nervous circuits that elicit ANS changes as well as the emotional experience. Though Pribram (1980) did not indicate that EDA has an emotion-specific indicator function, in his biologically oriented view of psychophysiological correlates of emotion, phasic electrodermal parameters have gained an important role in the research into this "facial feedback" hypothesis.

Lanzetta, Cartwright-Smith, and Kleck (1976) investigated the influence of instructions to manipulate one's facial expression on EDRs during the anticipation and application of electric shocks of different intensities. They performed three experiments, the third being the most carefully controlled one. In this study, 10 subjects of each gender received 2 sec electric shocks of 33%, 66%, and 99% of a previously established individual tolerance limit. The shocks were announced 8 sec prior to their presentation by displaying the numbers 1, 2, and 3, respectively. During the 10 sec following each

[220] A further domain is animal research, about which Panksepp (1982, p. 410) states that brain research in this area seems to regard the study of emotional expression as the only credible scientific approach.

of the 15 baseline trials, the subjects rated their discomfort caused by the shock on a scale from zero to 100. The session was videotaped, and the discomfort experienced was rated afterwards by six judges (four males, two females), who neither knew the shock intensity nor the subject's rating. The anticipatory EDR[221] as well as the EDR to the shock itself increased monotonically with shock intensity, as did the self-rating of experienced discomfort and the ratings of observed discomfort by the judges. During another 26 trials with permuted intensities, subjects had either to hide or to amplify their experienced discomfort by means of their facial expressions. Those manipulations were successful in influencing the judges appropriately; in addition, the EDR amp., especially the one following the shock, was significantly lower when subjects tried to hide their discomfort than when they tried to amplify pain expression.

Monotonic relationships between the degree of mimic expression of experienced pain and EDR strength were confirmed mainly in other studies of the Lanzetta group (Kleck, Vaughan, Cartwright-Smith, Vaughan, Colby, & Lanzetta, 1976; Colby, Lanzetta, & Kleck, 1977; Orr & Lanzetta, 1980). In addition, Vaughan and Lanzetta (1981) investigated the effects of facial expression on vicarious emotional arousal. They exposed three groups of 20 subjects each to a videotaped model, giving them either instructions to amplify or to inhibit their own facial muscles when the model appeared to be shocked, while the third group received no facial instructions. The fact that instructions were effective was shown by means of EMG recordings from three facial muscles (orbicularis oculi, masseter, and medial frontalis). The model appeared to undergo a word-shock classical conditioning paradigm with 4 practice, 10 acquisition, and 10 extinction trials, while the subject's electrodermal FIR, SIR, and TUR were recorded during the appropriate time windows (Sect. 3.1.2.1).[222] All groups showed greater electrodermal responsivity on CS+ trials as compared to CS– trials. However, this difference was more pronounced for the group having received the "amplify" instruction than for either the"inhibit" group or the uninstructed subjects. Additionally, in the extinction phase a tendency appeared for the "amplify" subjects to show a greater FIR to CS+ than to CS–, while the other groups did not. Those results gave support to the facial feedback hypothesis with respect to autonomic reactivity in general, since HR recordings showed a similar pattern. This view is rather close to James's ideas incorporated within the so-called James-Lange theory, stating that different emotions are caused by muscular reafferents to the CNS (McFarland, 1981, p. 289). However, the results above do not unambiguously show whether EDRs in fact serve as indicators for

[221] As inferred from another article of the Lanzetta group (Kleck et al., 1976), EDA was probably recorded with 3.14 cm^2 zinc electrodes using a zinc sulphate electrode paste and a Fels dermohmeter with 70 μA constant current. SRL values recorded from palmar sites were transformed to SCL values. The anticipatory EDR was computed as an increase from the average SCL 2 min before and at the beginning of shock application to the average SCL from 2 min before and at the beginning of the slide projection. The latter SCL was used as a reference for the EDR to shock application, thus being subtracted from the average SCL 6 and 8 min after its application.

[222] EDA was measured as SR via palmar Ag/AgCl electrodes, transformed into SC, and individually standardized.

emotion, or only as an ANS correlate of increased facial muscle activity, regarded as artifactual (Sect. 2.2.5.2).

In opposition to the results of the Lanzetta group, Buck and his colleagues found inverse relationships between EDA and facial reactions to emotion-inducing slides (summarized by Buck, 1980). Buck and Miller (1974) ran 32 subjects of each gender in randomly chosen sender-observer pairs, while their facial reactions to 25 slides were videotaped. There were five slides of each of five categories (sexual, pleasant landscape, pleasant people, unpleasant injuries, and unusual photographic effects), presented in randomized order. The observer rated the sender's mimic on a nine-point scale from pleasant to unpleasant, and the sender performed the same rating while watching the slides. Communication accuracy, measured by the correspondence between the sender's and observer's ratings, correlated significantly negatively with the sender's EDA,[223] however, only with male senders ($r = -.74$). As found in a previous study (Buck, Savin, Miller, & Caul, 1972), the correlation of communication accuracy with HR remained insignificant ($r = -.27$ for male senders). However, the subjects reacted more with their cardiovascular than with their electrodermal system during a postexperimental verbal description of the slide contents. Buck and Miller (1974) interpreted this as being consistent with other findings (e.g., Campos & Johnson, 1967) which showed that emotionally arousing visual stimuli affect EDA more than HR, while a requirement to make overt responses (including verbal ones) elicits HR acceleration more than EDRs. This also parallells the results of the Fowles group with respect to differential validity of these ANS variables (Sect. 3.2.1.2).

Buck et al. (1974) offer a conditioning explanation for the negative correlation between facial expressiveness and electrodermal responding to stimuli, assuming a socially learned inhibition of overt affective responses. Inhibitory responses may become CSs which elicit similar autonomic responses as the former UCSs. Thus, together with a masked facial expression, a large ANS response will appear which is more likely electrodermal than cardiovascular in nature because no action (including facial muscles) is elicited.[224]. Following this line, Buck (1980, p. 821) concluded that facial expression is more likely to serve as a readout device than as a feedback device, and that our facial emotional expression reflects central processes, not the reverse.

Winton, Putnam, and Krauss (1984) tried to explain the contrasting findings of the Buck and Lanzetta groups in light of their having performed different experimental manipulations of two dimensions of affective experience, consistent with those proposed by Wundt (1896): the intensity (arousing) and the evaluative (pleasant vs. unpleasant) aspect of emotion. Lanzetta and his colleagues used expressiveness ratings as measures of shock painfulness, thus reflecting the intensity dimension of affective

[223]Unipolar recordings with zinc electrodes and a zinc sulphate electrode paste from palmar against forearm sites, using a "low" constant current, transformed into SC values.

[224]This social learning should have appeared early in life, since Buck (1977) found comparable negative correlations between SCR and communication accuracy even in preschoolers, giving nonverbal messages via spontaneous facial expressions and gestures to their mothers.

response, which is positively correlated with EDR. In the Buck studies, however, the slide stimuli presumably evoked affective changes not only on the intensity but also on the evaluative dimension, while subject's as well as judge's ratings may have specifically concerned the evaluative dimension. As suggested by the Laceys (e.g., Lacey & Lacey, 1970), phasic HR is the more appropriate measure for this dimension than phasic electrodermal responses.

To further test this hypothesis, Winton et al. (1984) performed an experiment with 24 male subjects viewing a series of 25 emotionally evocative slides in a paradigm similar to that of Buck et al. (1972, 1974). Facial expressions were covertly videotaped and were later shown to 90 judges of both genders. HR and SCR (using standard methodology with KY-gel) was continuously recorded, and subjects rated pleasantness of slides 10 sec after slide onset on a seven-point Likert scale. While HR increased monotonically with increasing subjective pleasantness of the slides shown, SCR amp. (between 1 and 5 sec following stimulus onset) showed a U-shaped course, being higher in pleasant and in unpleasant stimuli than in neutral ones. The HR results paralleled the ratings of facial pleasantness of the judges, while ratings of facial intensity showed the same U-shaped relationship to slide pleasantness as the SCR amp. These results were replicated in two subsequent slide rating studies of Putnam and co-workers (cf. Winton et al., 1984).

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

Similar relationships between emotional valence and HR as well as between SCR and emotional arousal obtained by Winton et al. (1984) were found by Greenwald, Cook, and Lang (1989). Forty-eight subjects of both genders were presented 21 slides for 6 sec each, while facial EMG (zygomatic and corrugator), HR, and SC[225] were continuously recorded. Based on results from a previous validation study, valence and arousal dimensions of the slides were regarded as relatively independent ($r = -.01$ for males and $r = -.24$ for females). Subjective ratings were recorded by a computerized self-assessment technique. Larger SC changes were significantly related to increased arousal ratings, the effect being pronounced solely for males. SC change was not related to valence ratings, as HR was. Thus, EDA appeared as a sensitive and specific measure

[225]With standard methodology, using KY-gel. Mean SC change was calculated by subtracting the 1 sec prestimulus average from the average between 2 to 7 sec after stimulus onset.

of arousal, while phasic HR acceleration proved to be a sensitive and specific measure of emotional valence.

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

Given the background of the apparent differential indicator functions of electrodermal and cardiovascular variables,[226] the use of multivariate methodology in psychophysiological research into emotional states has to be strongly advocated, despite some inconsistencies between the appropriate studies as described at the beginning of this section. Within this frame, EDA may also not be regarded as a unitary ANS variable. Instead, different parameters could have different validities with respect to various emotional states, as observed by Stemmler (1989).[227] However, still lacking is a theoretical framework for using different tonic EDA measures (Sect. 2.3.2), together with phasic measures of EDR amp. and shape (Sect. 2.3.1), to explain different components of variance in various emotional contexts.

3.2.2.2 EDA as an indicator of stress

The use of the term *stress* in psychology covers a wide range of phenomena, from simple over- or understimulation, via fustrative experience, to life-challenging situations. Therefore, it is not easy to treat stress as a clearly unitary concept, especially with respect to activation or emotional experience. Most researchers would accept that stress results in distressing experience of high intensity (Lazarus, 1966), since only a minority of studies have been concerned with the concept of eustress (i.e., experience of stress in a positive emotional context). Despite these problems of delineation, stress

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

[227] Seligman (1975), in an explorative study with six subjects, obtained differential effects of pleasant and released vs. unpleasant and inhibited feelings (as reported on a Mood Adjective Check List) on negative vs. positive SPR waves respectively, during 10 counselling sessions of 50 min duration each. However, Edelberg (1972a) had already pointed to results with respect to the emotional valence of different SP wave forms being equivocal in general.

can be generally defined as a state of high general activation and negatively tuned but unspecific emotion, which appears as a consequence of stressors acting upon the subject. They are subjective and/or objective challenges exceeding a critical level with respect to intensity and/or duration.

Stress reactions are regarded as having properties to reestablish homeostasis by using physiological and psychological levels as well. If this goal cannot be attained by the subject, fundamental psychophysiological changes are expected. However, it remains debatable whether long-lasting neuroendocrine changes (as focused on in the stress concept of Selye, 1976) develop as a consequence of continuous short-lasting psychophysiological and endocrine reactions which can be elicited in laboratory stress situations. Experimental evidence for this comes from animal research (as in the development of ulcers, cf. McFarland, 1981) but cannot be performed with human subjects for ethical reasons.

Modelling stress in laboratory settings, however, serves as a tool to observe the course of corresponding psychophysiological processes under experimentally controlled conditions. Therefore, the investigation of the characteristic course of psychophysiological parameters over time can be regarded as a major aim in this area of research (McGrath, 1982, p. 36). Thus, continuously observing this time course may form a specific paradigm with respect to comparisons of simple group means in research on general activational or emotional states, as well as to stimulus-dependent short-lasting phasic changes in expressed emotion research (Sect. 3.2.2.1). Tonic electrodermal parameters, such as the EDL or the NS.EDR freq. (Sect. 2.3.2), may be regarded as the most suitable measures to continuously monitor ANS activity-elicited by stress, since EDA is solely determined by the activity of the sympathetic branch of the ANS which is predominant in stress states.

Early research with continuous recording of EDA (together with HR) during stress and coping processes was performed by the Lazarus group (summarized by Lazarus, 1966; Lazarus & Opton, 1966). In several experiments they showed a marked SCL increase in subjects watching stressful scenes of films. By presenting those films with coping-inducing sound tracks, the SCL could be markedly reduced. Following the concept of threat and appraisal as developed by Lazarus (1966), the subsequent laboratory experiments of his group focused on the role of anticipation of harmful events.

In one study performed by Nomikos, Opton, Averill, and Lazarus (1968), two groups with 26 subjects of both genders viewed two versions of an industrial safety film portraying three wood-mill accidents. The group which viewed the short version, in which most of the anticipatory scenes preceding the accidents were edited out, yielded a lower increase of SCL[228] than the group viewing the original version. In addition, for both treatment groups, most of the buildup of electrodermal stress reaction occurred during the anticipation period immediately before the accident. Thus, view-

[228]Recorded with a Fels-dermohmeter and 70 μA current, by means of zinc/zinc sulphate electrodes (paste not mentioned), transformed into SC units.

ing the accident as opposed to its anticipation, added comparatively little to the rise in ANS activity. For HR, group differences were in the same direction as those for SC, but did not reach significance. The self-report measures also did not differentiate the treatment conditions. The general finding that anticipating a stressful event could elicit electrodermal changes comparable to, or even greater than, those following the event itself initiated various systematic studies on conditions that influence anticipatory stress. They are: the duration of the anticipation interval, temporal and event uncertainty (i.e., predictability of time and probability of the appearance of the aversive event), using settings with and without temporal feedback, and controllability (i.e., having control over the aversive event by, e.g., terminating it by pressing a button). Most of these studies used tonic EDA parameters as measures of stress.

Folkins (1970) threatened 60 male subjects (in independent groups of 10 subjects each) with electric shock at the beginning of six different anticipation intervals (5 sec, 30 sec, 1 min, 3 min, 5 min, and 20 min). Time cues were provided by a large clock; however, no shocks were delivered. The subjects were reassigned on a separate random schedule to previously applied control conditions using the same intervals which were, however, terminated by a neutral stimulus (a lamp). During the anticipation intervals, a 1 min prewarning baseline and a 1 min poststimulus period, SCL[229] and HR were recorded every 10 sec (except for the 5 sec conditions, where only one reading was performed). Each of the subjects was interviewed post hoc, and 30 additional subjects were run but were interrupted for on-the-spot reports on cognitive functioning during anticipation of stress. The latter subjects were assigned to the following three independent groups: interruption of a 1-min interval after 30 sec, of a 20-min interval after 30 sec, and of a 20-min interval after 3 min. Subjective stress was also assessed by three self-report measures. SCL as well as HR steeply increased immediately after the shock announcement, with a continuing increase during anticipation periods up to 1 min, while there were no changes in control conditions.

Within the 3-min and 5-min intervals, a plateau appeared in SCL with another rise immediately before the end of the interval period. By contrast, during 20-min intervals a decrease to base levels appeared after 2 min, and another rise of SCL began during the 16th min. The greatest amount of subjective stress was reported with the 1-min anticipation interval. The interviews performed after interruptions showed that coping mechanisms started after 1 min. The presence of these mechanisms was used by the authors to explain the plateau or even the decrease observed in SCL course. As opposed to SCL, HR showed inconsistent courses, and could not differentiate experimental from control groups, in the intervals exceeding 1 min.

By contrast, Monat, Averill, and Lazarus (1972) found a relatively good concordance between EDA, HR, and subjective measures in two experiments concerning the effects of temporal uncertainty on anticipatory stress reactions during a 3-min inter-

[229] Bilateral thenar recording as SR with Beckman electrodes of 1 cm diameter, 10 μA constant current, transformed to SC values.

val. The first study was performed with 80 male subjects that were assigned to independent groups receiving three trials with either temporal or event uncertainty, with temporal and event certainty (100% shock at time known), and two additional groups with event uncertainty (50% probable shock receiving either no or 100% shock). The second experiment with 40 male subjects used a within-subjects design with the first three conditions as in the first study, but with an additional 5% event uncertainty condition. In both experiments, the mean SCR amp.[230] continuously decreased during the condition of temporal uncertainty (with 100% event certainty), while the different conditions (100%, 50%, and 5%) with temporal certainty (and temporal feedback) showed the same course of initial decrease of EDRs and again a steep increase during the last 30 sec. The courses of HR and of the self-reported tension (during first, middle, and last thirds of trial) showed similar results but turned out to be more susceptible to the repeated-measurement design effects than did EDA. These led to a diminution of the difference between temporal certainty and uncertainty in those measures at the end of the anticipation interval, which were probably due to learning effects.

Different courses of SCL[231] and HR in dependence on temporal feedback (by means of a clock) given or not during a 6-min period of anticipating an electric shock were found by Gaebelein, Taylor, and Borden (1974) with a total of 20 male subjects. In the feedback group, HR showed a slight decrease followed by a steep increase at the end of the interval, and a continuous HR decrease appeared in the group without temporal feedback. Instead, the SCL markedly increased following instruction in the no-feedback group and remained at this level, while under the condition of temporal feedback there was a decrease in SCL following an initial increase, and another increase during the last min. The authors attributed the steep HR increase during the anticipation period to a covert preparation for shock receipt via increased muscular tension. Thus, HR was interpreted as reflecting somatic activity, while the SCL course was more likely reflecting the course of psychophysiological stress reactions themselves. This interpretation remains, however, ambiguous, since EMG recordings were not taken.

Niemelä (1975) investigated the preparatory effect of long-lasting anticipatory periods on EDRs to a film of wood-shop accident scenes. Thirty male subjects expected to view the film three days after being presented with a subincision[232] film, followed by a detailed description of the film scenes that were to be expected. However, they were shown the stress film either immediately or after one or two days. Subjects from another study performed in the same laboratory viewing the film at the time announced

[230]Recorded with 10 μA constant current from thenar/hypothenar sites, using Ag/AgCl electrodes of 1 cm diameter with KY-gel, amplitude criterion 80 Ω. Responses in 10-sec intervals were averaged and transformed to log SC.

[231]Measured with Ag/AgCl sponge electrodes (other details of recording missing) as SRL values, averaged for every 10 sec and transformed to SCL values.

[232]Crude surgical operations on the male genitals of a primitive native culture in Australia. EDA has been recorded from both hands as SR with zinc electrodes of 2 cm diameter, using an agar zinc electrode paste, and a 40 μA constant current. SR values were transformed to log SC changes (differences between pre- and postaccident SCLs).

(from zero to 3 days) served as controls. For each scene, the SCR was computed and subjects reported their expectations afterwards. The SCRs to the accident scenes were lower the shorter the interval was and different from the results in the control group that viewed the film at the time announced. This was interpreted by the author as a consequence of the subject's suppression of the stressor material, which increases with the distance from the expected stressful event.

Bankart and Elliot (1974) performed three experiments using shock anticipation with varying probabilities of occurrence (event uncertainty). In their first study which confounded shock probability and number of trials, all subjects (male) received eight electric shocks, 10 of the subjects under each of the following conditions: within 8, 11, 16, or 32 trials (i.e., 100%, 73%, 50% or 25% probability). The intertrial intervals were 30 sec with a verbal countdown from 10 to zero. A rise of SCL[233] occurred during the session as well as during the anticipation periods, the latter one increasing in steepness with event certainty. In a second experiment an equal number of subjects were given either 5, 10, 15, or 20 shocks in 20 trials. This confounded probability with the number of shocks. SCL did not discriminate among the groups since it increased in all groups both within and across trials. Since no habituation of EDA could be observed in the second study, a third experiment was performed using the same 25% and 100% probability conditions, however, with markedly reduced shock intensities. HR differences which had appeared as a direct function of number and probability of shocks in the second experiment disappeared when shock intensity was reduced, thus being dependent on the amount of threat. Again, no group differences, but a slightly overall drift downward in SCL, appeared. The authors concluded that variations in event uncertainty do not exert influence on the anticipatory proportion of the SCL. However, they admit that other EDA parameters like the NS.EDR freq. could serve as more appropriate indicators of anticipatory stress than the EDL.

A possible differential indicator function of these two kinds of tonic EDA measures has been found by Katkin (1965). He compared the course of SRL and of NS.SRR freq.[234] during the 10-min anticipation of electric shock (which was not actually given) with a control condition ("the experimenter will be back at the end of time span"), using 26 male students in each condition. NS.SRR freq. was significantly increased in the experimental as compared to the control group, while the SRL decreased in both groups during a postexperimental interview. The clear effect of stress on spontaneous EDA was replicated in several subsequent studies performed by Katkin and co-workers (summarized by Katkin, 1975). Katkin's (1965) interpretation of EDL assumes it is more influenced by cognitive demands, while NS.EDR freq. is more prone to anticipatory stress. These hypotheses were supported by a study of Kilpatrick (1972) with 32

[233]Recorded as SR with dry 2 cm^2 lead electrodes from acetone-cleaned finger sites, transformed to values of $\mu S/cm^2$.

[234]Measured unipolar from left middle finger against forearm sites with electrodes (metal not specified) of .32 cm^2 and 3 x 4 inches, respectively, starch-paste, and 20 μA constant current. 100 Ω were used as amplitude criterion.

male subjects, half of which were given stressful instructions (anticipating an intelligence test), the other half control instructions. NS.SRR freq. but not SRL[235] differentiated between conditions. However, the SRL markedly decreased during a subsequent performance test.

Boucsein and Wendt-Suhl (1976) used a 20-min anticipation period to test differential cardiovascular and electrodermal reactivity to announcements of different shock intensities. Thirty male subjects in two groups were told they would receive a shock either two or five times stronger than a previously experienced one rated as unpleasant. The control group expected only a questionnaire. The NS.SRR freq. recorded with standard methodology (using Beckman paste) decreased during the first 5 min and significantly increased in the last 2 min of the anticipation interval in both stress groups as compared to the control group. However, the increase in spontaneous EDA, which was paralleled in subjective finger-span rating of emotional arousal, was not different in the groups that received different shock strength announcements. HR did not parallel EDA or subjective measures, since only in the group anticipating a shock five times as strong did a significant increase in HR appear during the last min.

A direct comparison of electrodermal and cardiovascular changes during 6 sec anticipation of an aversive stimulus (100 dB, 1 sec auditory signal) was performed by Sosnowski (1988). The stimuli were delivered in the first and the fourth of four trials, and the announcement was made by presenting two slides with circles each time. Eighteen subjects were run under either condition: an unambiguous clear announcement, and an ambiguous signal given by the circles. Changes in SC[236] were greater in the latter condition, showing a similar course as during the unambiguous condition, with an increment during presentation of the message, a plateau, and another increase after delivery of the aversive stimulus. By contrast, HR changes recorded were relatively small, except for a rise after message presentation, and the ambiguity factor did not significantly influence HR. However, the author's conclusion that EDA was a more suitable indicator of anticipatory stress, while HR is more likely to reflect cognitive elaboration of the message and/or coping with the stressor, remains equivocal with respect to the narrow data base.

Controllability of a stressor also influences EDA. Gatchel, McKinney, and Koebernick (1977), in a study on "learned helplessness," used 12 subjects of both genders in each of three groups. The experimental subjects could terminate unsignalled tones (1 kHz, 95 dB) by pressing a microswitch four times, while the yoked control group could not, and the third group (also yoked) was instructed to passively listen to the tones. They had to press the switch when the tone came on, which ruled out confounding controllability and motor activity. The tones were presented in five blocks of seven

[235]Recorded unipolar thenar vs. forearm with 5 cm^2 and 58 cm^2 Ag/AgCl electrodes, respectively, filled with .5 molar NaCl (probably liquid), using 10 $\mu A/cm^2$. Amplitude criterion for NS.SRRs was 100 Ω.

[236]Measured with Ag/AgCl electrodes of 4 mm diameter, KCl paste (.67 molar) in agar, and .5 V, evaluated as log SC change from pretreatment level.

trials, during which the SCR amplitudes[237] to tones habituated. They were significantly higher in experimental subjects and habituated more slowly when compared to the other groups.

Even in situations where subjects did not really exert control over a stressor but believed they could, a reduction of EDA could be found. Geer, Davison, and Gatchel (1970) subjected 40 male subjects to a RT task, during the first 10 trials of which they were told to react to the onset of a 6-sec shock. For the next 10 trials, half of the subjects were made to believe that decreasing their RT would reduce shock duration, while the remaining subjects were simply informed that shock duration would be reduced. Nonveridical control over the shock significantly reduced the NS.SCR freq. (obtained with standard methodology using Beckman paste), and log SCR amplitudes following shock onset showed a faster habituation as compared to the first 10 trials as well as to the control group.

To avoid confounding the effects of controllability and predictability, Geer and Maisel (1972) performed a study with 20 subjects in each of the following three groups that were presented 10 slides showing victims of violent death. In the controllability condition the subjects were able to terminate the presentation by pressing a button; the predictability group was given the mean duration for a given yoked subject in the first group, the value of which they were told; both groups were warned 10 sec prior to the slide by a 1 kHz, 60 dB tone, while the third group received random presentations of slides and tones of the same length as the second group. Predictability increased EDR amplitudes,[238] though a marked habituation occurred over trials. EDR amplitudes following the aversive stimuli were markedly increased in both the predictability as well as in the group without any control, as compared to the group that exerted control over stimulus duration. These results point to a relative independence of the action of controllability and predictability of aversive events on the ANS.[239] However, the number of NS.EDRs occurring between stimulus presentations did not differentiate between the two experimental groups, being significantly higher in both of them as compared to the group without any control. Thus, nonspecific EDA seemed to serve as an indicator of generally increased cognitive activity (as already suggested by Katkin, 1965, and Kilpatrick, 1972), while stimulus qualities (e.g., being a warning cue or aversive in nature) are reflected in specific phasic EDA components.

Studies testing the "preception" hypothesis can also be regarded as having an aspect of predictability of stressful events. Katz and Wykes (1985), in a further evaluation of

[237]Recorded as SR with 10 μ constant current, Beckman Ag/AgCl electrodes, and KY-gel palmar/dorsal. Separate channels were used for SRL and SRRs (sensitivity 50 Ω). SRL values were transformed to SCL, and SCRs were computed as differences between logarithmized SCLs before and the maximum within 6 sec after stimulus onset.

[238]Recorded as SR between palm and forearm with Beckman electrodes and paste, transformed to square-root SCR. Time window for SCRs: .5–3 sec after stimulus onset; amplitude criterion for NS.SRR freq. : 200 Ω.

[239]This was shown by Overmier (1985) with animals, using completely different experimental conditions, and plasma cortisol as a stress indicator.

the Katz (1984) study described in Section 3.1.2.1, investigated the effect of temporal certainty in 80 females anticipating six predictable and six unpredictable electric shocks in a within-subjects design with permuted order. During the anticipatory intervals (of 9, 12, or 15 sec duration), the NS.SRR freq. was significantly higher under the condition of temporal uncertainty compared with knowledge of the duration of the anticipation period.

Phillips, Evans, and Fearn (1986), in a study with 12 subjects of each gender, varied shock predictability by varying probabilities for warning signals (5%, 20%, or 50%) in three trials of 3 min duration each. During each trial subjects could continuously choose between monitoring or receiving distracting information by pressing appropriate keys. Subjects were further assigned to three different controllability conditions (0%, 50%, or 100%) which allowed the termination of the shock by means of another key. While SCL[240] steadily increased during the experiment, NS.SCR freq. significantly increased with predictability (i.e., the probability of warning signals). In addition, there was a significant correlation between NS.SCR freq. and the average time spent with monitoring (r =.37). Again, NS.EDR freq. appeared to be a better indicator of anticipatory stress reactions than did EDL.

Elevated nonspecific EDA was also observed during the anticipation of public speaking, which proved to be a considerable stressor in laboratory experiments. Erdmann, Janke, and Bisping (1984a) applied four different stress conditions of 10 min duration each to 24 male subjects in permuted order. They were: white noise (95 dB) presented discontinuously; anticipation of a painful electric shock; anticipation of public speaking; and a Charlie Chaplin film (as an "eustress" condition). The NS.SRR freq. (recorded with standard methodology, using an amplitude criterion of 300 Ω) was significantly higher during the anticipation of a public speech as compared to all other situations. The same was true for HR, blood pressure, and a subjective rating of emotional tension taken twice during stress periods. Spontaneous EDA reached its peak during the first third of the anticipation period, except in the condition with anticipation of pain (where the time course could be monitored via a clock) which led to a biphasic course with another peak in the final third of the anticipation period. However, the subjectively rated emotional tension did not parallel the course of NS.SR freq. in the anticipation of speech condition, since it increased throughout.

Differences in time courses between electrodermal and subjective measures of stress were also obtained from nonlaboratory research with parachutists (for a summary, see Epstein, 1972). Whether or not psychophysiological dissociations during the course of anticipating stressful events increase with the complexity of the stress situation remains to be tested. This requires combined laboratory/field studies in the area of stress research, which are also necessary to establish possible relationships between short-

[240]Recorded at palmar sites with Ag/AgCl electrodes and 10 μA constant current, transformed to SC. No information was provided concerning electrode size, paste, and amplitude criterion for SCRs.

lasting psychophysiological stress effects and long-lasting changes as discussed at the beginning of this section.

3.3 Personality and individual differences

The psychophysiological research on individual differences has been comprehensively reviewed recently by Gale and Edwards (1986). Their general conclusion was that research in this area is still fragmented and lacks integration. One aspect of their criticism on research in this field, which has specific implications for the use of ANS variables like EDA, concerns the discrepancy between the focus on behavior in personality theories and the fact that most studies examined subjects in a merely passive nonbehaving state with only weak external stimulation. Furthermore, the author's impression is that only a few personality theories offer testable predictions for psychophysiological reactivity. The following chapter will focus on broad as well as narrow personality dimensions that have specific relevance to electrodermal reactivity.

Since the beginning of this century, the various attempts to establish relationships between personality dimensions and electrodermal responses have adopted an implicit arousal model in their interpretation (Edelberg, 1972a). This was the case both for broad personality traits like extraversion/introversion or emotional lability (Sect. 3.3.1) and for more specific traits like repression/sensitization or sensation seeking (Sect. 3.3.2). Among these is electrodermal lability, a personality characteristic based on EDA measurement itself (Sect. 3.3.2.2).

The concept of electrodermal reactivity as a relatively stable individual characteristic is also related to the problem of idiosyncratic ANS responses or individual specificity (Engel, 1972). An individual who consistently tends to overreact in some physiological modality is commonly regarded as being prone to develop psychophysiological (psychosomatic) diseases related to that particular system (Sect. 3.5.2.3).

3.3.1 General traits

Psychophysiological correlates of general personality traits (i.e., those on the factor analytic C level) have been most thoroughly investigated for extraversion/introversion and emotional lability ("neuroticism"). Eysenck (1967) proposed a neurophysiological basis for these two major, independent second-order factors which caused a great number of empirical studies in this field. In general, Eysenck identified introversion with the existence of lower thresholds in the various parts of the RAS (Sect. 3.2.1.1) and emotional lability with lowered thresholds in the limbic system. According to this, emotionally labile subjects should be more ANS-reactive than stable ones, and extraverts were suggested to have a tendency toward a lower resting arousal level than

introverts (Eysenck, 1983). Since the appropriate literature has been thoroughly reviewed by Stelmack (1981), the following two sections can be restricted to conceptual and methodological considerations, providing results only from several representative studies.

3.3.1.1 EDA and extraversion-introversion

According to Eysenck (1967), introverts are characterized by their easier conditionability, because activity of their RAS-cortical loop is increased, facilitating consolidation of learned material. Extraverts are thought to elicit cortical inhibition faster, providing a kind of protection against strong stimulation. This brings extraverts close to being sensation seekers (Eysenck & Zuckerman, 1978; Sect. 3.3.2.1). Eysenck's conclusion is that, given the same objective stimulation, introverts should be more aroused than extraverts within an average intensity range, whereas the opposite is true for the upper intensity range, due to the increase of cortical inhibition in extraverts.[241]

The greater reactivity of introverts to stimuli of moderate intensities, which has been shown in various studies of the Eysenck group, were found most consistently in the electrodermal system (Stelmack, 1981; Eysenck & Eysenck, 1985). In addition, tonic EDA is higher in introverts than in extraverts, though not frequently noted as appropriate differences in phasic EDA (Eysenck, 1983). The following three different approaches may be used to establish relationships between EDA and personality characteristics like extraversion/introversion:

(1) An approach which correlates personality questionnaire data and EDA obtained under conditions of resting and stimulation.

(2) ANOVA approach using one or more traits as organismic factors. Groups are formed either by the use of median split or by selecting extreme groups. This allows an investigation of the main effects of personality characteristics as well as interactions of experimental conditions with personality factors.

(3) A psychopharmacological approach (Sect. 3.4.3) which has been especially favored by Eysenck (1967), in which the cortical excitation/inhibition balance is shifted by the use of specific drugs, thus providing an experimental manipulation of the neurophysiological correlate of extraversion/introversion.

[241] Eysenck discussed his inhibition concept with respect to Pavlov's "transmarginal inhibition" or "protective inhibition," the neurophysiology of which he regarded as unrealistic, though the phenomenon itself (which is also in accordance with the so-called Yerkes-Dodson law, and the inverse U-shaped relationship between arousal and performance; Sect. 3.2.1.1) has been observed many times (Eysenck, 1983, p. 18). However, Eysenck's attempt to include various aspects of inhibition into a unitary CNS inhibition concept may have contributed to various differences between theoretical concepts and experimental results (Nebylitsyn, 1972, p. 21; Strelau, 1983, p. 145), thus being in part responsible for pitfalls in establishing reliable psychophysiological correlates of extraversion/introversion.

For each of these three approaches, a sample investigation into the differences in EDA between extraverts and introverts will be summarized below.

Rajamanickam and Gnanaguru (1981) performed a study following approach (1) with 23 male subjects, using the change in SRL[242] before and after the application of an electric shock. Significant correlations to extraversion ($r = -.62$) as well as to emotional lability ($r = .52$) were obtained, interpreted by the authors as due to an increase in ANS reactivity in introverts as well as in emotionally labile subjects.

A more refined analysis with respect to extraversion was performed by Fowles, Roberts, and Nagel (1977) following approach (2), in a series of four experiments. They also investigated emotional lability ("neuroticism") as a personality trait besides extraversion/introversion. However, they formed extreme groups by the use of lower and upper 30% of the scale, and they measured changes in SCL[243] in series of twenty 1 kHz tones of different intensities. In addition, general arousal level was manipulated via solvable and unsolvable tasks performed prior to stimulation.

In their first experiment with 80 male subjects (half introverts and half extraverts), as well as in their second study (a replication with 20 subjects of each group), they reported a greater increase of SCL in extraverts with high tone intensities (103 dB) as compared to a control condition (83 dB), independent of arousal manipulations (by varying task difficulty). A corresponding difference appeared, however, in introverts only when easily solvable tasks had been presented. By contrast, in case of a previous increase of arousal induced by difficult tasks, the second study yielded smaller changes in SCL to high intensity tones, supporting Eysenck's hypothesis of a protective cortical inhibition. This was more clearly shown in the third experiment with 80 female subjects (half introverted and half extraverted), as well as in the fourth study with 10 female subjects in each of four groups formed by the combination of extreme groups of extraversion/introversion and emotional lability. Since both experiments were performed without previous manipulation of arousal by means of performance tasks, base-level arousal was assumed to be relatively low in all subjects. Under these conditions, introverts showed a marked SCL decrease with increasing stimulus intensity, whereas the SCL in extraverts increased or remained unchanged with increasingly intense stimuli. Emotional lability did not yield an effect on SCL changes.

Another study performed by Smith, Wilson, and Jones (1983) followed approach (3). Forty-eight extraverts and 48 introverts, half males and half females, were selected as extreme groups from a larger sample. To induce different levels of cortical arousal, they were randomly assigned to three caffeine doses (1.5, 3.0, and 4.5 mg/kg body weight) and a placebo condition. After 45 min, the subjects received two series of six 1.5 kHz tones with intensities varying between 60 and 110 dB, either with or without

[242]Measured by the use of zinc electrodes of 25 mm diameter with 1% zinc sulphate paste, current density not reported.

[243]Recorded with 2 cm^2 Ag/AgCl electrodes, .5 % KCl in Unibase, and 1.0 V constant current.

a prewarning signal, in randomized order. Introverts showed a higher SCL[244] than extraverts. Furthermore, a significant interaction between stimulus intensity and personality trait appeared, introverts reacting to tones of lower intensities (up to 80 dB) with larger SCR amplitudes as compared to extraverts, while no such difference appeared with tones of high intensities. The second-order interaction between personality, caffeine doses, and presence or absence of the warning signal also reached significance. There was a steady increase of SCR amp. in extraverts with increasing doses of caffeine regardless of warning condition. On the contrary, introverts showed a steady decrease of SCR amp. with increased doses when warning signals were absent, but a decrease from placebo to the lowest caffeine condition and a further increase with increasing doses under the warning condition.

The results of Smith et al. (1983) can be regarded as supporting some of the hypotheses proposed by Eysenck (1957). First, a higher general activation of introverts under conditions of low stimulation was found throughout the study. Second, the introversion-inducing effect of stimulants, which is part of Eysenck's "drug postulate," was shown, since an increasing arousal in extraverts with increasing caffeine dose is in accordance with the hypothesis of a protective cortical inhibition mechanism in this group when activation is increased.

On the other hand, anticipatory processes induced by the warning signal could have reduced the effects of increased stimulation level, thus dropping the cortical arousal beyond the threshold for the elicitation of a protective inhibition. This points to an important role of attention as a moderator variable within Eysenck's postulated excitation-inhibition continuum. Indeed, introverts in general yielded a better performance in vigilance tasks than extraverts (Krupski, Raskin, & Bakan, 1971).

However, in general the empirical data on activation of introverts versus extraverts were equivocal. Therefore, Gray (1970, 1973) proposed a modification of Eysenck's original theory, starting from the existence of relatively independent reward and punishment systems in the brain (Sect. 3.2.1.2). According to this, introverts were no longer regarded as more highly conditionable. Instead, Gray (1970) suggested they were more susceptible to punishment and frustrative nonreward. On the other hand, extraverts were regarded as being more susceptible to positive reward. Furthermore, according to Gray's view, the extraversion-introversion dimension is not orthogonal to emotionality or "neuroticism" in Eysenck's sense. Emotionality is treated as the degree of sensitivity to both reward and punishment. This is in accordance with repeatedly confirmed negative correlations between extraversion and emotional lability with questionnaire data (Boucsein, 1973) and is depicted by the additional axis in Figure 49. Later, Gray (1981) identified susceptibility to positive reinforcement with the personality dimension "impulsivity", while sensitivity to negative reinforcement had

[244]Recorded with Ag/AgCl electrodes of 1 cm diameter, .05 molar NaCl paste, and 9.55 $\mu A/cm^2$ current density. SR values transformed into SC values, SCRs were obtained, square-root transformed, and range-corrected.

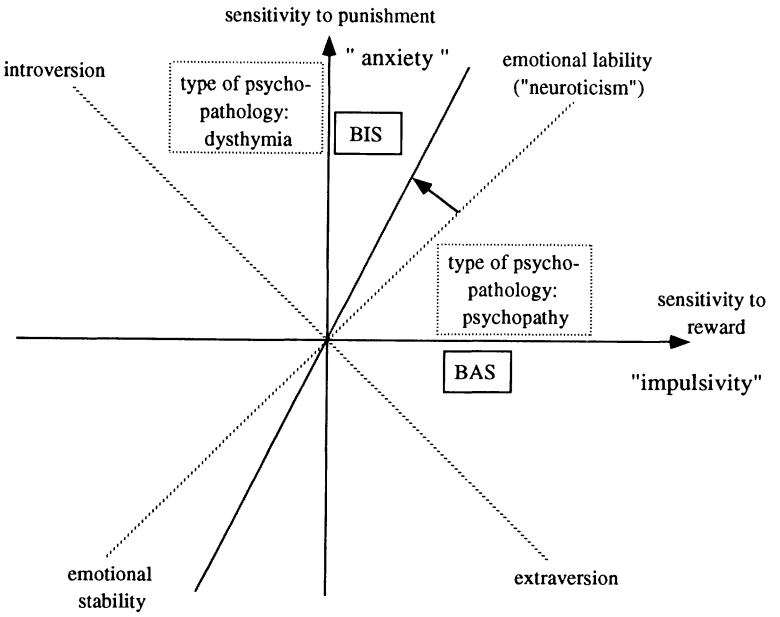

Figure 49. Eysenck's (1967) C-level personality dimensions (dotted lines) extraversion/introversion and "neuroticism" (emotional lability versus stability) as bisectors of angles in Gray's (1970) modified dimensional system (solid lines) formed by impulsivity and anxiety, together with their suggested behavioral (sensitivity to reward versus punishment) as well as CNS correlates (BAS versus BIS), and related types of psychopathology. The correlation between Eysenck's dimensions as found in questionnaire data is indicated by an additional axis which is rotated against the original lability/stability axis (see arrow).

been operationalized by the Taylor (1953) manifest anxiety factor, both regarded as orthogonal.

As Figure 49 shows, Eysenck's extraversion/introversion dimension is located as a bisector of the angles between impulsivity and anxiety. The aim of this axis rotation was the establishment of a one-to-one relationship between C-level personality factors and their suggested behavioral and psychobiological sources. Increasing degrees in Gray's anxiety factor were regarded as being a correlate of an increasing sensitivity to signals of nonreward and novelty. On the other hand, an increase in impulsivity should be correlated with an increasing susceptibility to signals of reward and absence of punishment.

In addition, Gray (1970, 1982, 1987) suggested that anxiety reflects individual differences in the BIS (i.e., the septo-hippocampal stop system), while individual differ-

ences in the approach system (the limbic reward system or BAS; Sect. 3.2.1.2) should form the neurophysiological basis for impulsivity. The latter part of that hypothesis is in accordance with results concerning electrodermal and cardiovascular activity in psychopaths. These were regarded by Eysenck (1967) as neurotic extraverts, as opposed to dysthymics who are neurotic introverts (Figure 49). Hence, as Fowles (1980) stated, psychopaths should be prone to high BAS activity and a weak BIS, which is reflected in an increased HR and a decreased EDA in that particular clinical group (Sect. 3.4.1.2).

From this point of view, a differential validity of the electrodermal and the cardiovascular system for these two broad personality dimensions can be suggested as follows. An increase in EDA may be regarded as a specific indicator of anxiety (or introverted emotional lability), while an HR increase should specifically reflect impulsivity (or extraverted emotional lability). However, the connection between EDA and BIS as suggested by Fowles (1980) has not yet been sufficiently established (Sect. 3.2.1.2). Furthermore, most evidence for a direct connection between anxiety and BIS as formulated by Gray (1982) stems from animal studies. In a multivariate study with 66 female subjects on personality and psychophysiological reactions to various emotion-inducing situations (frightening acoustic and optic stimulation, preparing a free speech, etc.), Andresen (1987) reported that NS.SCR freq. (measured with standard methodology, using .01 μS as an amplitude criterion) indicated more anxious activation than behavioral inhibition. In addition, EDA showed a connection with decreased sensation seeking (Sect. 3.3.2.1). The question remains unanswered as to whether tonic EDA really reflects differences in second-order personality factors, or will be more tightly connected to specific traits or even states which may both be correlated with general personality dimensions.

3.3.1.2 EDA and emotional lability

According to Eysenck (1967), individuals scoring high on the personality dimension of emotional lability should exhibit a higher tonic level as well as hyperreactivity of the ANS, especially under conditions of stress. Indeed, a high correlation between this trait and EDA was regarded as one of the most established results in psychophysiological personality research (Stern & Janes, 1973) until being questioned more recently (Katkin, 1975; Stelmack, 1981). In addition, Eysenck's identification of emotional lability with "neuroticism" does not provide a clear-cut separation of anxiousness in normal subjects from neurotic anxiety, which is regarded as psychopathological (Sect. 3.4.1.1). Furthermore, the localization of that particular personality dimension in the second-order factor space remains unclear. Gray's (1981) identification of Taylor's (1953) manifest anxiety scale (MAS) with his own anxiety dimension, which he proposed as being a bisector in Eysenck's introversion/"neuroticism" quadrant, had been criticized by Eysenck (1982). Eysenck pointed to the higher correlation between MAS scores and his "neuroticism" dimension (r = .70) as compared to the correlation of MAS scores with introversion (r = .30). This fits well to the shift of the emotional la-

bility axis as indicated by the arrow in Figure 49, which is, however, only one possible solution to this dilemma.

Of the three approaches outlined in the previous section, approach (2) has been mostly used in research on EDA and emotional lability. As an example, a study performed by Rappaport and Katkin (1972) is reported here. These authors used a short form of the MAS to form 24 high- and 24 low-anxious subjects by selecting the upper and lower 20% of a larger male student sample. Sixteen subjects of each group were instructed after a rest period to report their own emotional reactions by pressing a foot pedal, which had been previously shown to produce no movement artifacts in EDA (Sect. 2.2.5.2). The subjects were told that electrodermal recording could be used to validate their subjective report. The additional eight subjects in each group served as a control group that underwent another rest period.

High- and low-anxious subjects did not differ significantly in their NS.SRR freq.[245] during the initial resting condition. High-anxious subjects reacted to self-reporting their emotionality with a marked increase, and during the control condition with a decrease, of NS.SRR freq. as compared to low-anxious subjects. Cognitive processes could be ruled out as a possible explanation, since there were no differences in self-reported reactions to the test situation between groups. On the other hand, the appearance of differences in EDA between high- and low-anxious subjects seems to be connected to situations inducing moderate stress and including ego-involvement, like the one used in that particular study. This is supported by the lack of correlations between trait anxiety and tonic EDA under rest (Stern & Janes, 1973), as well as under strong stress conditions like receiving electric shocks (Katkin, 1975).

Attempts to investigate the influence of emotional lability on EDA according to approach (3) have be made by using tranquilizers as a tool to produce anxiolytic effects. These will be reported in detail in Section 3.4.3.1. However, influences of anxiolytic drugs were predominantly on state anxiety, which is not markedly influenced by the personality factor emotional stability versus lability (e.g., Boucsein & Wendt-Suhl, 1982).

In summarizing various studies, Stelmack (1981) came to the conclusion that relationships between emotional lability ("neuroticism" in Eysenck's sense) and EDA have not been demonstrated with sufficient consistency to infer the proposed ANS basis for this broad personality dimension. Instead, as already discussed at the end of the previous section, NS.EDR freq. may be used as an effective index of emotional response to anxiety and stress states (Sect. 3.2.2.2), a conclusion which is also in accordance with the findings of Katkin (1975).

As Fahrenberg (1987) stated, it is doubtful that a single psychophysiological measure may be used as a valid indicator for a specific personality trait. Instead, he advocated a multivariate approach leading to individual specific reaction patterns. These may show closer relationships to subfactors of the broad C-level personality dimen-

[245]Recorded with standard Beckman Ag/AgCl electrodes and Beckman paste from the left palm, using 20 $\mu A/cm^2$ as current density and 100 Ω as an amplitude criterion.

sions than to general traits themselves. When following this line of study, approach (1) may be of only limited value, because relationships between personality and psychophysiological patterns lack transsituational invariance. Instead, approach (2) should be preferred, since it allows for the study of main effects of situational factors and their interactions with the personality factors under investigation as well. Approach (3), which would be a rather tough testing of the suggested CNS-ANS relationships underlying personality dimensions, requires drugs of highly specific and selective action within these systems which are, however, not yet available for human psychopharmacological study (Sect. 3.4.3.1).

3.3.2 Specific traits

3.3.2.1 Traits based on questionnaire data

Attempts to establish long-lasting individual differences in electrodermal reactivity were also attempted through the use of questionnaires focusing on traits below the second-order factorial level. The *repression/sensitization* scale constructed by Byrne (1961) from the MMPI was used to measure defensive styles in six studies of film stress performed by the Lazarus group (Sect. 3.2.2.2). These studies were reanalyzed by Weinstein, Averill, Opton, and Lazarus (1968), who showed that the discrepancy between self-report and psychophysiological responses to stress was greater in repressors than in sensitizers.[246]. Their conclusion was that both groups were equally physiologically aroused, but repressors consistently claimed to be less distressed than sensitizers.

In order to test this hypothesis, Boucsein and Frye (1974) classified 58 male subjects as repressors, nonspecific defenders, and sensitizers using the upper, lower, and middle thirds of Byrne's scale. Each of these groups was partitioned into a stress group who was blamed for failing to solve anagrams, most of which were unsolvable, and a control group that was told they would not have to solve the task completely because of the experimenter's interest in reaction differences to difficult versus easy anagrams. Skin resistance was recorded continuously (with standard methodology using Beckman paste), and a subjective mood scale was applied twice during the anagram-solving task. ALS scores (Sect. 2.3.3.4.4) were obtained for the mean NS.SRR amp. (Sect. 2.3.2.2) as well as for the subjective scales. The discrepancy scores that were formed as differences between standardized subjective and EDA scores were subjected to ANOVA. The only personality-stress interaction that reached statistical significance showed a greater subjective than physiological reactivity of repressors, and a correlation of .78 was ob-

[246]Individual ANS reactivity was obtained using the higher of either HR or SC standardized scores. However, since SC, not HR, was used in all six studies, individual reactivity might have been mainly expressed within the electrodermal system.

tained between the MAS and the Byrne scale. Therefore, the authors concluded that repression/sensitization may not be, either as a construct or with respect to psychophysiological reactivity, clearly separated from the second-order factor trait "anxiety".

Weinberger, Schwartz, and Davidson (1979) also obtained a very high correlation (r = .94) between the MAS and the Byrne scale in their 40 male subjects. In order to separate "true repressors" from purely low-anxious subjects, they used an ad hoc scale to measure subjectively reported repressive defensiveness. Fifteen low-anxious, 11 high-anxious, and 14 "true repressors" were selected from a total of 200 male subjects. After an adaptation period they were subjected to a phrase association procedure as a mild stress condition. The "true repressors" showed a significantly higher NS.SRR freq.[247] than the other two groups under stress, but not under resting conditions. Since their RT was also significantly longer, Weinberger et al. (1979) concluded that "true repressors" not only have a higher trait anxiety than they claim with questionnaire data, but they also show ineffective coping with psychosocial stressors.

Another specific personality construct with a close relationship to psychophysiological reactivity was the *type A versus type B* concept, measured by questionnaire as well as behavioral data, which has been related to risk for coronary heart disease (Rosenman, Friedman, Straus, Wurm, Jenkins, & Messinger, 1966). The coronary-prone type A has been characterized by time urgency, excessive activity, competitiveness, impatience, aggressiveness, and hostility, all of which are easily evoked under environmental challenges due to an increased lability in the sympathetic-parasympathetic balance (Dembroski, MacDougall, & Shields, 1977). Despite the fact that various studies using cardiovascular measures yielded a greater psychophysiological arousal in type A's, mainly in interpersonal threatening situations (summarized by Houston, 1983), most investigations that used EDA as a typical measure for the suggested higher sympathetic reactivity of type A's did not yield supporting results (Krantz, Glass, & Snyder, 1974; Dembroski et al., 1977, 1978a; Steptoe & Ross, 1981; Holmes, McGilley, & Houston, 1984; Steptoe, Melville, & Ross, 1984), with the exception of one study performed by Lovallo and Pishkin (1980). They divided 80 male subjects into equal numbers of type A's and B's by rating their behavior pattern through interviews. All subjects underwent three tasks (serial pattern learning, anagram solving, and concept identification) while SC was measured with standard methodology. Under all three tasks, type A subjects showed higher SCLs and higher NS.SCR frequencies (with .1 μS as an amplitude criterion), and also reacted with a higher SCL to success in the pattern-learning task, while SCL decreased under that condition in type B's.

In a multivariate study performed with 144 postinfarction patients, Langosch, Brodner, and Foerster (1983) analyzed 20 EDA parameters during 11 different task periods. In addition, cardiovascular, respiratory, performance, and subjective measures were used for stepwise multiple prediction of six diagnostic and prognostic criteria. They

[247]Recorded with 16 mm Ag/AgCl electrodes and .05 molar NaCl in Unibase from the palms. Amplitude criterion: 100 Ω.

came to the overall conclusion that EDA was of minor importance as compared to the other predictors investigated. Thus, EDA does not appear to be useful for selecting subjects with coronary proneness, the connection of which with type A has also to be generally questioned with respect to recent research (Myrtek, 1984).

Another personality construct that has come into focus in differential psychophysiology during the last two decades is *sensation seeking*. The concept was originally extracted from a questionnaire developed by Zuckerman, Kolin, Price, and Zoob (1964) to measure long-lasting individual differences in the optimal arousal level (Sect. 3.2.1.1). Correlations could be established between the Sensation Seeking Scale (SSS) and, for example, variety of sexual partners, experience with drugs, as well as preference for risky sports and more complex tasks (Zuckerman, 1983). As Feij (1984) pointed out, psychophysiological studies found close relationships of SSS to the "strength of the nervous system," which is the first basic property of higher nervous activity in Pavlov's biologically oriented personality concept (Nebylitsyn, 1972). According to this, subjects scoring high on the SSS possess a stronger nervous system than those scoring low, which should result in elevated sensory thresholds as well as in lower reactivity. Indeed, Ridgeway and Hare (1981) demonstrated that sensation seekers reacted with cardiovascular OR patterns to 60 dB 1 kHz tones, while subjects scoring low on the SSS yielded patterns similar to defensive reactions (Sect. 3.1.1.2). No supportive results were found with EDA.

In several other studies, higher amplitudes of electrodermal ORs were observed in subjects scoring high on the SSS. Neary and Zuckerman (1976) performed two experiments with extreme groups formed by the SSS (i.e., approach (2) in Sect. 3.3.1.1). Their first study used 14 male and female subjects from the upper and lower 15% of the SSS distribution. They measured the electrodermal OR[248] to 10 presentations of a white rectangle followed by 10 repeated presentations of a complex colored design. Sensation seekers showed significantly higher EDRs on the first stimulus of both series but did not differ in the habituation course from subjects scoring low on the SSS. In their second experiment, 20 subjects of each SSS extreme group were chosen, half of which were additionally classified as high or low anxious by means of the MAS (Sect. 3.3.1.2). After presenting 10 white rectangles followed by one presentation of a complex colored design, 10 tones (1 kHz with approximately 70 dB) were applied, and a 200 Hz tone ended the series of stimuli. Sensation seekers showed an overall higher level of EDRs to the visual but not to the acoustic stimuli but a similar reaction to novel stimuli at the end of both series as subjects scoring low on the SSS. No significant trait anxiety effects were observed.

Smith, Perlstein, Davidson, and Michael (1986) reported relationships between sensation seeking and electrodermal reactivity to relevant, novel stimuli (tones, words, slides, and videotaped scenes), in 36 subjects who belonged to extreme groups of ei-

[248]The authors usually performed SR recordings with an active palmar and an inactive forearm electrode, Beckman standard electrodes and paste, and 20 μA constant current. SR scores were transformed to SC, and SCR amplitudes were square-root transformed.

ther high or low scorers on the SSS. Sensation seekers showed larger initial EDRs under all types of stimulation and also showed higher SCLs for words.

By contrast, Stelmack, Plouffe, and Falkenberg (1983b) found only weak relationships between SSS scores and electrodermal ORs. Since there is a positive correlation between sensation seeking and the C-level personality dimension extraversion, while predictions concerning electrodermal reactivity of sensation seekers and extraverts were opposite (Sect. 3.3.1.1), they applied Eysenck's personality questionnaire in addition to the SSS. The correlation between extraversion and the total SSS score was .60 in their total sample of 91 male and 93 female subjects, which is considerably higher than the usual correlation of .40 (Andresen, 1987). Out of the total sample, 118 subjects received 10 repeated presentations of a geometric figure, while 66 subjects received 10 verbal stimuli instead. Each series was followed by a novel stimulus. At the beginning of the verbal series, introverts showed higher SCR amplitudes (measured with standard methodology using KY-gel) than extraverts, and subjects scoring higher in two of the four SSS subscales yielded greater SCR amplitudes than low sensation seekers. However, no appropriate effect could be found for the total SSS score, and no personality effects were obtained for visual stimulation.

In another study with visual stimulation (presenting photographic slides), Plouffe and Stelmack (1986) also did not find a relationship between SSS and SCRs to the stimuli. Instead, they found an age dependency of the correlation between SSS scores and SRLs. While a positive correlation appeared in 26 younger females (aged 17–24 years), no correlation was observed in 25 older females (aged 60–78 years).

Overall, differences in OR between subjects scoring high and low on the SSS were more prominent in HR and in EEG-evoked potentials than in EDA measures (Zuckerman, 1983; Feij, 1984). Furthermore, the various personality traits discussed in this section did not show a closer relationship to EDA than did general traits (Sect. 3.3.1). It seems questionable whether psychophysiological variables like EDA parameters may show some validity as universal indicators of personality dimensions obtained by questionnaire data. It seems more promising to focus on measures of personality as being closer to psychophysiological constructs in order to perform research in differential psychophysiology, an example of which is given in the next section.

3.3.2.2 Electrodermal lability as a trait

The rationale of using EDA to determine long-lasting individual differences in ANS reactivity dates back to the 1950s, when Lacey and Lacey (1958) used the term "electrodermal labiles" for subjects showing a high NS.EDR freq. at rest and/or slow habituation to repeated stimulation, whereas subjects who produced few NS.EDRs or habituated rapidly were labelled "electrodermal stabiles." In several studies performed during the 1960s, spontaneous EDA under resting conditions has been found to be consistently related to electrodermal reactivity and to the course of habituation (Wilson & Dykman, 1960; Johnson, 1963; Koepke & Pribram, 1966).

Katkin and McCubbin (1969), who originally intended to establish differential habituation courses as a function of anxiety as measured by the MAS (Sect. 3.3.1.2), performed an additional classification of their subjects into electrodermal labiles and stabiles using the median of NS.EDR freq. during a 10 min rest period prior to stimulation. Two groups of 24 median-separated high- and low-anxious male subjects did not differ in their habituation course of the log SCR[249] to 15 presentations of 1 kHz tones of either low or moderate intensity. However, when spontaneous resting EDA was used to establish individual differences, the 25 electrodermal stabiles showed a steeper habituation rate in the moderate intensity series than the 25 electrodermal labiles. Though the difference appeared to be related to the higher initial EDR amp. of the stabiles (although not significant), these authors preferred to interpret their results in terms of habituation lacking in the labile group, thus showing a DR instead of an OR (Sect. 3.1.1.2).

Crider and Lunn (1971) found that spontaneous electrodermal fluctuation rate as well as habituation speed were stable individual difference characteristics. Since both measures were also highly intercorrelated, these authors suggested both variables indicate a common underlying dimension of electrodermal lability. NS.SPR freq.[250] was recorded twice, seven days apart, from 22 male students during a 5 min rest period, and during 20 trials of 90 dB, 1,300 Hz tones of 2 sec duration masked by 72 dB white noise. Retest reliabilities were .54 for the NS.SPR freq., and .70 for the habituation speed, which was determined as number of trials needed to fall below the habituation criterion of .1 mV within 3 sec after the end of a stimulus (Sect. 3.1.1.3). The intercorrelations between both measures were .51 in the first and .73 in the second session. Both of them showed zero correlations with neuroticism, while correlations with extraversion as well as with different subfactors of impulsivity ranged from –.24 to – .46 for NS.SPR freq., and from –.40 to –.57 for habituation speed. Thus, reliability as well as validity aspects yielded a slight preponderance of habituation speed as a measure of electrodermal lability.

Katkin (1975) used electrodermal lability instead of anxiety, recorded by means of questionnaire data, as a personality correlate, to predict reactivity to stressors of medium intensity levels. Based on a reanalysis of earlier experiments performed by his group, he developed the hypothesis that a high rate of spontaneous electrodermal fluctuations is not only an indicator of a defensive or anxious hyperreactivity to environmental stimuli. In addition, it should be regarded as a reliable indicator for state anxiety, in which attention is the critical mediating variable that leads to an increase of EDA (Sect. 3.1.3.1). Therefore, it has been concluded that electrodermal lability may

[249]Recorded as SR with Ag/AgCl electrodes and NaCl paste from Beckman, unipolar palmar against forearm, using 20 μA/cm^2 current, performing transformation to SC and logarithmization. The amplitude criterion for NS.EDRs during rest was 100 Ω.

[250]Measured with nonpolarizing Ag/AgCl sponge electrodes between the palm and an alcohol-cleaned forearm site, AC-coupling (Sect. 2.1.3) with .45 sec time constant, amplitude criterion: .1 mV. Several fluctuations within a 6 sec window were regarded as a single SPR.

reflect a subject's ability to allocate information processing capacity to stimuli that are to be attended to (Lacey & Lacey, 1958; Katkin, 1975; Schell, Dawson, & Filion, 1988; Wilson & Graham, 1989; Dawson, Schell, & Filion, 1990).

In accordance with this view is the well-established result that electrodermal labiles are likely to show a better ability at keeping their attention focused on an ongoing task, thus avoiding decrements in vigilance and performance (Coles & Gale, 1971; Siddle, 1972; Crider & Augenbraun, 1975; Hastrup, 1979; Vossel & Roßmann, 1984; Munro, Dawson, Schell, & Sakai, 1987). In turn, this strengthens the close relationship of electrodermal lability and introversion, found not only by Crider and Lunn (1971) but also by Managan and O'Gorman (1969), by Nielsen and Petersen (1976), as well as by Coles et al. (1971), since a better performance in vigilance tasks was found in introverts (Krupski et al., 1971). However, Sostek (1978), measuring the course of electrodermal habituation to 75 dB tones as well as the NS.SRR freq.[251]in 66 subjects, found only insignificant correlations between electrodermal lability and various personality dimensions like introversion, emotional lability, or the SSS (Sect. 3.3.2.1). "Habituation lability" was a better predictor of vigilance task performance than "spontaneous lability." Electrodermal labiles as identified by either measure showed the same reaction differences in a risky versus cautious payoff instruction, but electrodermal stabiles did not. Thus, Sostek (1978) suggested that electrodermal labiles may be more sensitive to environmental contingencies than stabiles, which could be due to the higher attentional capacity of the former group (cf. also Hastrup, 1979).

Further support for the treatment of electrodermal lability as a personality dimension of its own came from a study performed by Hastrup and Katkin (1976) with 120 male students. They correlated a pool of 478 self-report items taken from various personality questionnaires with the NS.SRR freq. during a 15 min rest period and the habituation speed taken from a series of 20 tones (440 Hz, 93 dB, 2 sec.). Several correlational and discriminant function analyses did not yield any close relationships between electrodermal lability and traditional self-descriptive psychometrics. In order to test Katkin's (1975, p. 173) hypothesis that electrodermal lability reflects differences in cognitive efficacy (i.e., a selective enhancement of effective central processes) rather than merely a generalized arousal mechanism, Solanto and Katkin (1979) performed a study on differential classical light-shock conditioning (Sect. 3.1.2.1), using the same kind of SR recordings transformed to log SC as Hastrup and Katkin (1976). Twenty electrodermal labiles and 21 stabiles were selected from 63 male students, according to their scoring above or below the medians of both the distribution of spontaneous EDRs during 10 min rest and of habituation scores. The latter were obtained as trials to habituation (three subsequent SRRs below 1 kΩ) in a series of 20 tones with 60 dB intensity. The electrodermal labiles showed an overall greater SIR amp. However, the data pro-

[251]SR recorded palmar against a forearm site with 2 cm^2 Ag/AgCl electrodes, Beckman paste, and 10 $\mu A/cm^2$ constant current. Amplitude criterion: 100 Ω.

vided no support for possible differences in EDR conditioning between electrodermal labiles and stabiles, as had been already suggested by Öhman and Bohlin (1973).

Siddle, O'Gorman, and Wood (1979) investigated the differential influence of stimulus significance on ORs (Sect. 3.1.1.1) in electrodermal labiles and stabiles. They selected 28 subjects of each group from a total of 230 male students for their first study, using the lower and upper 40% of the NS.SCR distribution, EDA being recorded with standard methodology and an amplitude criterion of .02 μS during a 5 min period of no stimulation. The subjects were presented with a series of 12 tones (1 kHz, 70 dB, 5 sec), followed by a single presentation of a 500 Hz tone. Half of each group had to respond as quickly as possible to each tone offset while the other half did not. Electrodermal labiles were more responsive than stabiles, and RT groups displayed larger SCRs to the stimuli than non-RT groups, but the interaction was not significant. Furthermore, group differences were not higher than the increase in SCR amp. produced by stimulus change alone. In a second experiment, 20 subjects of either group were presented 12 slides with female names, and on trial 13, half of the subjects in each group received their own name, while the others received a neutral male name. Again, electrodermal lability and stimulus significance displayed larger SCRs. No interaction effects appeared, except electrodermal labiles showed a considerably higher SCR amplitudes on presentation of their own names as compared to all other conditions.

Waid and Orne (1980) also confirmed that electrodermal reactivity to emotionally significant stimuli is higher in electrodermal labiles than in stabiles, with two experiments on the detection of deception (Sect. 3.5.1.2). In summary, electrodermal lability as obtained during stimulus-free recordings, or as differential habituation speed to relatively neutral stimuli, can be successfully used to predict reliable individual differences in EDRs under various conditions of stimulation. However, this is presumably part of a general autonomous reactivity instead of an idiosyncrasy of the electrodermal system, since electrodermal labiles and stabiles show differences with respect to various psychophysiological variables, including HR responsiveness (e.g., O'Gorman & Lloyd, 1988; Schell et al., 1988; Kelsey, 1991). Furthermore, it is still unknown how electrodermal lability can be embedded in a frame of personality dimensions as obtained by questionnaire data, which is of theoretical interest within the framework of differential psychophysiology.

3.4 Psychopathology

One of the most important areas in the field of applied psychophysiology is the use of physiological measures in diagnosis and therapy of psychopathological disorders. Accordingly, a great deal of electrodermal research has been performed in the area of clinical psychophysiology. However, browsing through even recent summaries in this field leaves one with the impression that only few aspects have been added to hypotheses and results known since the 1950s and 1960s.

Therefore, with respect to the majority of applications of EDA in psychopathology, the reader is referred to Stern and Janes (1973), supplemented by the third volume of the reader of Gale and Edwards (1983). Hence, the present section of this book will be restricted to clinical areas in which the use of EDA is based upon certain aspects of electrodermal mechanism and/or hypotheses concerning the differential validity of EDA parameters. These are disorders of anxiety, psychopathy, and depression (Sect. 3.4.1), schizophrenia (Sect. 3.4.2), as well as psychopharmacological treatment of anxiety disorders (Sect. 3.4.3).[252]

3.4.1 EDA in the assessment of anxiety, psychopathy, and depression

3.4.1.1 EDA in patients with generalized anxiety and phobias

Symptoms of *anxiety* can be found in most psychiatric disorders. According to DSM III-R (American Psychiatric Association, 1987), generalized anxiety appearing in neurotics is accompanied by symptoms of motor tension, autonomic hyperactivity, apprehensive expectations, as well as excessive vigilance and scanning. Clinical anxiety may be permanently present or appear in the form of attacks.

Anxiety is also found in schizophrenia, attributed at the beginning of the illness to the subjective experience of strange personality changes, and in later stages to delusional contents. In depressive disorders, anxious and depressed mood states are often not clearly distinguishable with respect to their phenomenology as well as to their therapeutic aspects (Foulds & Bedford, 1976). This is shown by the action of antidepressive and antianxiety drugs (Sect. 3.4.3), which belong to different psychopharmacological classes, but which act upon both depressive as well as anxious states (Derogatis, Klerman, & Lipman, 1972).

Besides various subjective scales that were constructed for the diagnosis of general anxiety (e.g., Taylor's MAS; Sect. 3.3.1.2), various attempts were made to objectively measure the actual anxiety level by recordings of its autonomic concomitants (e.g., using cardiovascular, electrodermal, or electromyographical variables). However, correlations between indicators of clinical anxiety stemming from the subjective and physiological level were, in general, rather low (Lang, 1970; Hodges, 1976), as is the case in any emotional state (Sect. 3.2.2.1).

The studies done by Lader and Wing (1964, 1966) are the most frequently cited attempts to differentiate anxious patients from healthy controls by means of EDA.[253]

[252]Further uses of EDA in therapeutic contexts are in biofeedback (Sect. 3.1.2.2), as well as in systematic densitization (summarized by Katkin & Deitz, 1973).

[253]Lader and Wing's research has been continued by Chattopadhyay et al. (1975, 1980, 1981, 1983).

Lader and Wing (1964) found the SCL[254] as well as the NS.SCR freq. to be significantly higher in 20 patients suffering from anxiety states (17 of them showing "free floating" anxiety all the time) than in 20 matched normal controls. An elevated level of tonic EDA during rest in anxiety patients, mostly confirmed by later studies (e.g. Raskin, 1975; Chattopadhyay & Biswas, 1983), was interpreted as being due to a general "overarousal" in these patients, as postulated by Malmo (1957).

In addition, Lader and Wing (1964) presented their subjects with 20 identical tones (1 kHz, 100 dB, 1 sec duration). While the SCL showed a continuous decrease in normal subjects during rest as well as during the tone series (after an initial increase), anxiety patients showed a slight but continuous increase of SCL all the time. By contrast, the number of spontaneous EDRs showed a steady decline, except for a small increase in response to the first few stimuli, being very similar in both groups. Lader and Wing concluded that although SCL and NS.SCR freq. can be regarded as two equivalent measures of tonic EDA in normals, this is not the case in patients suffering from anxiety, where both measures may reflect different aspects of arousal or vigilance. An habituation regression analysis performed with log SCR amplitudes yielded significantly higher initial ORs and a steeper habituation gradient in the normal controls. Lader and Wing (1964) as well as Lader (1975) interpreted the lower reactivity of patients as due to ceiling effects or to functioning of the law of initial values (Sect. 2.5.4.2).

This means that stimulation may have had little effect in addition to the already-existing autonomic overarousal in patients. However, the lower initial reactivity did not determine the slowing down of habituation rate as well, since there was also a significant difference in H scores between patients and normals (an habituation index independent of the size of the initial reaction; Sect. 3.1.1.3). Slowed or even missing habituation in anxiety patients is a consistent result not only for SCRs (Lader & Wing, 1964, 1966; Lader 1967, 1975) but also for cardiovascular measures (Malmo & Smith, 1951; McGuinness, 1973), for EMG (Davis, Malmo, & Shagass, 1954), and for EEG parameters (Ellingson, 1954; Bond, James, & Lader, 1974).

Hart (1974) provided an alternative interpretation of the anxious patient's failure to habituate as rapidly as normal subjects. The 100 dB tones used by Lader and Wing could have produced a DR in patients that was more resistant to habituation than the OR which appeared in controls. The suggestion was that anxious subjects have a lower threshold for exhibiting a DR than nonanxious subjects. In his own study, Hart attempted to differentiate ORs from DRs in response to three intensities of signal and nonsignal tones in 18 psychiatric anxiety patients and 18 normal controls. He used an initial HR deceleration as an objective measure for an OR, and an HR acceleration as an indicator for DR (Sect. 3.1.1.2). The EDA parameters log SCL, SCR amp., and NS.SCR freq. were recorded with standard methodology as resistance measures and

[254]Recorded with 10 μA constant current using lead electrodes filled with .05 molar NaCl paste, from the distal phalanx of the right thumb against an inactive (rubbed) site on the lateral aspect of the arm above the elbow. SR was converted to log SC, and a SCR of .003 log μS was used as an amplitude criterion.

transformed to conductance units. The nonsignal stimuli consisted of 30 tones (1 kHz, 2 sec) with an intensity of 50, 75, or 100 dB, presented in series of triads, with each triad containing one tone of each intensity. Under signal conditions, tones were presented in pairs, 2 sec apart, 12 trials at each level of stimulus intensity. The second tone of each pair was of a different frequency in eight trials of each series, and the task of the subjects was to judge whether the tones were the same or different in pitch.

In contrast to the results of Lader and Wing (1964), Hart (1974) did not find significant differences between anxious and nonanxious subjects in SCR amplitudes following the first stimuli, and SCL also did not differentiate the groups. In addition, the results of HR analyses were inconsistent with the hypothesis that anxious patients fail to habituate the OR as rapidly as nonanxious subjects. Instead, it seemed that, relative to normals, anxious subjects showed a deficit in orienting behavior, being more prone to respond with a DR pattern even when stimuli of low intensity were presented. There was also no significant difference in the slope of electrodermal habituation between the two groups. This was interpreted by Hart as being due to 50% nonhabituators in his control group, while Lader and Wing reported that their normal subjects were all habituators. In both studies, there was a comparable percentage of habituators in the anxious group.

The contrasting results in the studies concerning the slope of habituation in anxious versus nonanxious subjects may have been due to Hart's (1974) variations in signal values of the stimuli, and also to the stimulus intensity changes used in his design. Both conditions may have reduced monotony in stimulus presentation, which, in turn, prevented habituation. Another difference between the two studies was in spontaneous EDA. Anxious patients in Hart's study showed only less than twice as much NS.SCRs in a 3 min period preceding the first nonsignal tone as compared to normal controls, while Lader and Wing's patients showed more than three times as much spontaneous EDRs during rest than did the normals used in their studies.[255]

Differences in EDA between anxious and nonanxious subjects can also be influenced by the method of anxiety diagnosis. Neary and Zuckerman (1976) reported a negative correlation between the SCR amp. following 70 dB tones and anxiety scores as obtained by the state-anxiety scale of the Multiple Affect Adjective Checklist (Zuckerman & Lubin, 1965). An additional classification of their subjects according to trait-anxiety scores of the MAS (Taylor, 1953), however, did not yield any connection between EDA and anxiety level. State anxiety is consistently a better predictor of initial SCR amplitudes in stimulus series than trait anxiety, while it remains vague whether and under which circumstances the OR may be altered by the presence of anxiety (Sartory, 1983). According to Neary and Zuckerman (1976), a generalized increase in reactivity caused by elevated levels of anxiety may reduce discriminability and hence the initial reactions in stimulus series. However, there is strong evidence for an undoubt-

[255]Possibly the small difference in amplitude criteria – .002 log μS used by Hart (1974) versus .003 log μS used by Lader and Wing (1964) – may have also contributed to that difference.

edly higher tonic EDA in anxious subjects facilitating ceiling effects during specific stimulation as mentioned above.

As opposed to patients with generalized anxiety, *phobic* patients are characterized by overreaction to specific stimuli (or stimulus classes) that are neutral or of mild threat for normal persons. In summarizing several studies on autonomic reactions to phobic stimulation, Sartory (1983) arrived at the conclusion that the cardiovascular system shows a clear DR pattern in subjects with particular phobias, while EDR amp. are elevated as long as stimulus duration is under 5 sec. By contrast, longer stimulus presentations fail to differentiate between phobic and nonphobic reactions, which may be due to habituation effects.

Several studies on phobic reactions use the induction of phobia in normal subjects by means of classical conditioning (Sect. 3.1.2.1). According to Seligman's (1971) "preparedness" hypothesis, those stimuli should be more prone to being established as CSs for phobic reactions which gained fear-inducing properties during phylogenetic development. In this case, even a one-trial learning should evoke phobic reactions to a previously neutral but "prepared" stimulus.

In order to test this hypothesis, Öhman, Erixon, Löfberg (1975) presented 64 subjects of both genders 10 colored slides (snakes, houses, and faces) for 8 sec each during an acquisition and an extinction phase. Half of their subjects were conditioned by an uncomfortable electric shock to the potentially phobic stimuli (snakes), while in the other half the UCS was paired with the other, supposedly neutral (unprepared) CSs. EDA was recorded with standard methodology from the left hand's first and second fingers, and evaluated as logarithmized FIRs, SIRs, and TORs in slightly changed intervals (1–4 sec, 4–9 sec, and 9–13 sec after CS onset; Sect. 3.1.2.1). FIR amplitudes were significantly higher, and TORs showed more resistance to extinction in phobic stimuli than in neutral stimuli. As an additional experimental manipulation, half of the subjects in each group were informed before the extinction phase that no more shocks would be given, while the other half were not. Surprisingly, the informed subjects in the group that received the potentially phobic CSs showed more resistance to extinction as compared to the uninformed subjects. This pointed to some resistance of potentially phobic stimuli to plain cognitive manipulations. No such effects of information were found in the group being conditioned to the neutral CSs. Seligman's (1971) hypothesis was supported insofar as potentially phobic stimuli were more easily electrodermally conditioned, and it may be used to develop a laboratory analogue of phobic fears that cannot be easily removed by instructions.

Frederikson (1981) used the supposed indicator value of a greater SCR amp. at palmar as compared to dorsal sites (Sect. 3.1.1.2) to distinguish between electrodermal DR and OR with respect to phobic versus nonphobic reactions. Twenty-four spider- or snake-phobic women were compared with 24 female student controls who were conditioned to pictures of either snakes or spiders by means of electric shocks. Slides of flowers and mushrooms served as neutral stimuli, and HR was recorded as an additional dependent variable. During the first session, which served for conditioning in the con-

trols, the phobic group received eight phobic and eight neutral stimuli, and adjusted an electric shock level until it was experienced as uncomfortable, without further application of shocks. After the same procedure of adjusting the shock level, the control group was conditioned to either 12 snake slides or 12 spider slides (the other class of slides served as CS–) and also received eight neutral stimuli. During the second session on another day, half of each group received either fear-relevant or neutral stimuli during the extinction phase. Palmar SCRs were recorded from the middle phalanges of the left first and second fingers of the subjects, while dorsal SCRs were taken from the middle phalanges of the left third and fourth fingers, using standard methodology together with range correction (Sect. 2.3.3.4.2).

In the conditioning group, palmar FIRs during acquisition tended to be greater in dorsal than in palmar responding to CS+, while the reverse was true for CS–. This palmar/dorsal effect disappeared through extinction. Phobics reacted with a palmar/dorsal pattern reflecting a DR when confronted with their feared objects, while nonfeared and neutral stimuli only elicited an OR pattern. In addition, verbal-cognitive components of fear were positively correlated with palmar but not with dorsal SCRs, thus supporting Edelberg's (1973a) theory that palmar EDRs reflect aversiveness (Sect. 3.1.1.2). HR was accelerated following phobic material and decelerated following neutral material. A similar tendency was found during acquisition in the conditioning group.

Thus, besides the well-proved HR accelaration, a palmar/dorsal SCR amp. difference may serve as a sensitive indicator for fear reactions in phobics. The increase in HR can be interpreted as due to an active mechanism of coping with fear-relevant stimuli (Obrist, 1976), which is also in accordance with HR acceleration being an indicator of BAS activity (Sect. 3.2.1.2) as advocated by Fowles (1980). Similar response patterns can be elicited during acquisition of phobic reactions in healthy subjects, especially when using "prepared" stimuli as CS+, while during extinction the DR pattern switches to an OR pattern in both HR and EDA. As opposed to patients with generalized anxiety, phobic patients do not show a generally increased EDA but only overreact to their specific fear-relevant stimuli.

3.4.1.2 EDA in psychopathic or antisocial disorders

The term "psychopathy" is used for a variety of nonpsychotic personality disorders showing affective and social disturbances (Checkley, 1964). The main characteristic of psychopaths is an increased appearance of social conflicts (Hare, 1975), which has been traced by Eysenck (1967) to their general lack of conditionability. Thus, psychopaths are located in the extravert-neurotic quadrant of Eysenck's personality dimension system (Fig. 49, Sect. 3.3.1.1). In addition, psychopaths are supposed to have specific deficits in passive avoidance learning, discussed within the framework of Mowrer's (1960) theory by Lykken (1957) and Trasler (1973). Accordingly, the contingency between the appearance of a socially unwanted behavior and its negative consequences cannot be learned, since the reduced ANS reactivity of psychopaths prevent them from

developing an unpleasant state of arousal following punishment. As a consequence, psychopaths are sometimes labelled as antisocials or sociopaths, and are regarded to be prone to developing criminal behavior.

Hare (1978b) summarized several studies, mainly performed by his own group, that compared EDA in psychopaths and control subjects under various experimental conditions. There was a general tendency for tonic EDA to be lower in psychopaths, an effect that appeared more clearly for the EDL than for the NS.EDR freq. However, under conditions of overstimulation (e.g., in situations with aversive elements) or understimulation (e.g., boring or monotonous situations), the difference between groups became more prominent, due to a further decrease of tonic EDA in psychopaths, while nonpsychopathic subjects showed an increase or no change in tonic EDA. Hare's conclusion that differences between both groups were more consistent under conditions of stimulation than under rest conditions was also confirmed by Siddle (1977).

Borkovec (1970) found an insignificantly lower SCL [256] during rest and stimulation periods in 19 psychopathic than in 21 neurotic and 26 subcultural normal juvenile delinquents. The SCR amp. following the first stimulus of a 21 tone (1 kHz, with "moderate intensity") series was significantly lowered in psychopaths. However, no group differences were found throughout the rest of the habituation series, pointing to lowered initial autonomic reactivity rather than quick adaptation in psychopaths.

Though the hypothesis of an initial electrodermal hyporeactivity of psychopaths to unsignalled auditory stimuli was not confirmed in 9 of 10 additional studies (summarized by Raine & Venables, 1984), there is some evidence that these results were at least partly dependent on the method of parameter extraction. Using results from an earlier study of his own, Hare (1975) compared two different procedures of range correction (Sect. 2.3.3.4.2). While the application of Equation (43a) did not yield significant results, Equation (43b), which uses the maximum response given through the experiment, led to significantly smaller range-corrected SCR amplitudes in psychopaths, both to the first tone and to a dishabituation tone in a series of 80 dB stimuli.

In another study performed with 24 psychopathic and 40 nonpsychopathic delinquents, Hare (1978a) showed that electrodermal hyporeactivity in psychopaths only appeared during highly intense, fast rise-time stimulation, and only in the psychopathic subgroup scoring low in the socialization scale of the California Psychological Inventory (CPI; Gough, 1969). Six 1 kHz, 1 sec tones of each intensity (80 to 120 dB, in 10 dB steps) were presented in permuted order, three with a fast rise time (10 μsec) and three with a slow rise time (25 msec) (SC was recorded bilaterally with standard methodology, using hypertonic paste of 5% NaCl). For EDA recorded from the right hand, the interaction of intensity with socialization was significant, in contrast to its interaction with psychopathy, indicating that socialization has at least as important an influence on EDA hyporeactivity to aversive stimulation as psychopathy itself.

[256]Recorded with gold-plated electrodes and Beckman paste, taped over soaped, dried, and alcohol-washed volar finger sites.

Electrodermal hyporeactivity in psychopaths also appeared in several studies using conditioning to electric shocks, as summarized by Hare (1975). Psychopaths do not readily develop conditioned EDRs, especially when the UCS is aversive. The question of whether the reduced conditionability of psychopaths, mostly shown with delinquent subjects, causes or follows the development of antisocial behavior was investigated in a prospective study performed by Loeb and Mednick (1977). Sixty male and 44 female subjects were conditioned prior to any delinquent behavior, using a 4.5 sec 96 dB irritating noise as UCS that appeared .5 sec after CS onset (a 1 kHz, 54 dB tone). An habituation phase with eight presentations of the CS was followed by 14 partial reinforcement trials (9 CS-UCS parings and 5 CS alone), and a generalization-testing with two tones of different frequencies ended the session. Ten years later, seven of the male subjects had become delinquent. They were paralleled by seven nondelinquent subjects from the original sample. A comparsion of both groups yielded lower SCR amplitudes[257] in the later delinquents during all phases of the testing procedure performed 10 years earlier, especially in the initial UCR, and delinquents yielded a reduced amount of conditioning. In addition, only one of the later delinquents showed stimulus generalization. Therefore, the authors concluded that lowered autonomic reactivity, as measured by EDA, may have contributed to the development of an antisocial personality in their delinquents.

A study performed by Raine and Venables (1981) with 101 fifteen-year-old male children pointed to the dependence of poor electrodermal conditionability on socialization. Besides teacher's ratings of refractory school behavior, several self-report measures of socialization, including the appropriate CPI scale, Eysenck's psychoticism scale, and the disinhibition scale of the SSS (Sect. 3.3.2.1), were taken from each subject in order to obtain a factor-analytic socialization score. In a classical conditioning paradigm with partial reinforcement, 15 CSs (65 dB tones, 1 kHz, 10 sec) were paired with 10 UCSs (105 dB tones, 1 kHz, 1 sec) while SC was recorded.[258] For every presentation of CS without UCS, the mean magnitudes of FIR, SIR, and TOR were correlated with the teacher's rating and the socialization score. Intercorrelations were generally low, and a relationship between undersocialization and poor conditionability appeared only in the high-class children, while this relationship was reversed in lower-class subjects. However, the highest correlation obtained was –.27 (between TOR and socialization score), which to some extent obscured the discussion of differential effects of the different EDR components on socialization.

Raine and Venables (1984) reported correlations from the same sample between the first electrodermal OR obtained in a habituation series to nine of the above-mentioned CSs and their socialization score, all of them being also small and negative (up to r = –.41) but statistically significant. An additional analysis using categories of responding-

[257]Recorded with 7-mm diameter zinc electrodes and zinc sulphate electrolyte, using a Wheatstone bridge. The polarity of the 1.5 V reference reversed every 1.2 sec.

[258]Bilateral measurements with Ag/AgCl electrodes of 4.5 mm diameter, filled with .5% KCl in agar-agar paste, using .5 V constant voltage. Amplitude criterion: .05 μS.

nonresponding yielded 81% of the electrodermal nonresponders as antisocials, while 80% of the nonhabituators were prosocials. A second analysis used the original SSS subscales and measures of "schizoid" tendency as personality measures, together with EDA results and an antisocial index, to form groups of prosocial nonresponders, antisocial responders, and antisocial nonresponders. This analysis indicated that the latter group showed significantly more "schizoid" tendencies but not more sensation seeking. Therefore, Raine and Venables hypothesized that electrodermal nonresponding may be a biologically predispositional factor in antisocial personality as it is supposed to be in schizophrenic disorders (Sect. 3.4.2.2).

Raine, Venables, and Williams (1990a) reported data from a 15-year follow-up with the sample of Raine and Venables (1981). By this time, 17 of the 101 subjects were found to possess a criminal record, differing from the noncriminals in school status and residential characteristics but not in occupations, and only slightly in age. Fifteen years before, the criminals-to-be had shown a significantly lower resting HR, lower NS.SCR freq. taken from both hands, and more slow-frequency EEG activity than noncriminals. The findings for the SCL were in the same direction but nonsignificant. These results provided clear evidence for the role of both ANS and CNS underarousal in the development of criminal behavior. Raine, Venables, and Williams (1990b) presented appropriate data from a nine-year follow up of the ANS reactivity data reported by Raine and Venables (1984). As compared to the 84 noncriminals, the 17 subjects who later became criminals showed significantly smaller SCRs and smaller HR acceleratory and deceleratory responses nine years before, thus providing evidence for ANS hyporeactivity, in addition to hypoactivity, as a marker for the development of criminal (antisocial) behavior.

The electrodermal hyporeactivity found in psychopathic or antisocial subjects is also in accordance with Fowles's (1980) EDA-BIS hypothesis (Sect. 3.2.1.2). As Fowles (1988) stated, a weak BIS should result in an inability to inhibit responses which may be followed by punishment, and Checkley's (1964) characterization of psychopaths fits very well into the pattern one would expect from a weak aversive motivational system with appetitive motivation being quite normal. The role of motivation in poorer conditionability of psychopaths was also stressed by Hare (1978b), who pointed to the possible influence of cognitive factors on conditioning (Sect. 3.1.2.1 & 3.1.3.1). Accordingly, psychopaths may show lack of motivation to follow the learning experiment's intention, thus showing a deficit in awareness of the particular contingencies. Indeed, psychopaths especially show problems with differential electrodermal conditioning (i.e., learning to react with a higher EDR amp. to a CS+ as compared to a CS–). A lack of general activation may also play a role, since cardiovascular and electrodermal ORs as well as habituation rates are reduced in psychopaths, appearing together with an increased amount of EEG-theta activity, as observed during drowsiness. Furthermore, conditionability of psychopaths may be increased by the introduction of nonspecific stimulation (Hare, 1978b).

Electrodermal reactivity in psychopaths appears to be influenced to a great extent by motivational and activational states. In comparison to control subjects, psychopaths show lower EDA amplitudes during experimental procedures that are either monotonous or threatening (i.e., inducing either under- or overarousal), while situations being moderately exciting (i.e., inducing medium arousal levels) do not produce such differences (Hare, 1978b). For example, Jutai and Hare (1983) could not find differences in SCL (recorded with standard methodology) between 11 psychopaths and 10 controls during video games. It is likely that psychopaths tend to be drowsiness-prone in boring situations, thus showing electrodermal hypoactivity as well as hyporeactivity, while their EDA is indistinguishable from normals during motivating conditions producing optimal arousal (Sect. 3.2.1.1). Under threatening conditions with high arousal, EDRs of psychopaths decrease again. This may be due to their lower susceptibility to punishment. Another interpretation could be appearance of "sensory rejection" (Lacey & Lacey, 1974) as characterized by an increase in HR together with cortical deactivation. Indeed, cardiovascular activity is markedly increased in psychopaths anticipating aversive stimuli, while the anticipatory EDR is reduced (Hare, 1978a; Blackburn, 1983). This is also in accordance with the hypothetical BIS/BAS antagonism and its EDA/HR correlates (Sect. 3.2.1.2).

Besides electrodermal hyporeactivity, as shown by reduced EDR amplitudes, a prolonged EDR recovery time in psychopaths is a consistent experimental result (Siddle, 1977; Hare, 1978b), also found in the prospective study performed by Loeb and Mednick (1977) described above. However, Hare (1978a), in his above-mentioned study using stimuli between 80 and 120 dB, could only find an increased SCR rec.t/2 following the 120 dB tones with short rise times, which can be characterized as aversive stimuli producing a startle response (Sect. 3.1.1.2). Two different explanations may be given for a prolonged EDR recovery in psychopaths (Hare, 1978b):

(1) It indicates a "tuning out" or attenuating of sensory input with aversive qualities (Hare, 1978a). This is in accordance with Edelberg (1970, 1972b), who suggested that slow recovery is indicative of a DR. Furthermore, Venables (1975) argued that a long SCR rec.t/2 is related to a "closed gate" state of attention, due to a predominance of the amygdala's excitatory influence on subcortical information processing (Table 6, Sect. 3.1.3.1). This in turn may point to psychopaths having deficits in their septo-hippocampal system, preventing them from good conditioning to punishment, and from showing an appropriate electrodermal OR to novel stimuli.

(2) It is indicative of a delay in passive avoidance learning (Mednick, 1974). According to the two-process theory of Mowrer (1960), a reduction of anticipatory fear could act as a reinforcer for an inhibition of antisocial behavior. Thus, a quick diminishing of anticipatory fear enables effective avoidance learning. Since long SCR recoveries could be regarded as an indicator for a slow diminishing of antic-

ipatory fear reactions, they may also indicate a slow diminishing of anticipatory fear reactions and thus a less successful learning to avoid antisocial behavior.

According to Hare (1978b), his observation that prolonged SCR rec.t/2 in psychopaths only appears with really aversive stimulation, is in accordance with the Mednick hypothesis formulated under (2). However, he questions that the recovery rate to simple stimuli is predictive of the recovery rate during avoidance learning.

Since psychopaths also show lower EDR amp. than normal controls, the relationships found by Bundy and Fitzgerald (1975) between EDR rec.t/2 and amplitudes of preceding responses should be taken into account when discussing EDR recovery times in conditioning studies (Sect. 2.5.2.5). This was done by Levander et al. (1980), who studied the electrodermal habituation of 24 imprisoned delinquents with a series of 21 tones (1 kHz, 93 dB).[259] There was a significant correlation ($r = .47$) between inverted scores of the CPI socialization scale and the mean recovery time of trials 2–20. Since this recovery time showed a significant negative relationship to the mean NS.SCR amp. ($r = -.65$), the Bundy-effect was adopted by these authors as a possible alternative explanation to the DR interpretation formulated under (1).

Neurophysiological support for the relationships between psychopathy or antisocial behavior and EDA comes from different sources. Hippocampal deficits, which may be caused by heredity, prenatal or perinatal factors (Mednick & Schulsinger, 1973), possibly account for a prolonged EDR recovery time (Table 6, Sect. 3.1.3.1) and a reduced NS.EDR freq. as well. The latter can be regarded as being due to a malfunction of the septo-hippocampal BIS (Fig. 48, Sect. 3.2.1.2). According to Gray (1982), the BIS not only inhibits motor behavior, but it also increases attention as well as cognitive activity. Therefore, the supposed hippocampal dysfunction in psychopaths fits well into Eysenck's classification of that group into the extravert-neurotic quadrant (Fig. 49, Sect. 3.3.1.1), since extraverts are regarded by Eysenck as showing a lowered cortical activation. A reduced cognitive analysis of possible consequences of antisocial behavior may be cause as well as consequence of a BIS dysfunction.

According to Fowles's (1980) BIS/BAS model, psychopaths should have an increased BAS activity, the psychophysiological concomitant of which is HR (Sect. 3.2.1.2). Indeed, when an unavoidable aversive stimulation was present, psychopaths showed not only electrodermal hyporeactivity but, in addition, an increase in HR, while nonpsychopaths were more likely to react with an increase of EDA and with only small HR changes (Hare, 1978b; Fowles, 1980).

[259] EDA was recorded with Ag/AgCl electrodes of 9 mm diameter with .9% NaCl paste from ethanol (75%)/acetone (25%) cleaned sites of the left hand's second and third fingers. The authors used a combined current density (9 $\mu A/cm^2$)/voltage (2.7 V) limiting system. If necessary, the SCR rec.t/2 values were extrapolated by the method of curve matching (Sect. 2.3.1.3.2). Since the mean recovery time of the EDR to the first tone was conspicuously longer than those following the other tones, the latter ones were averaged, and analyses were made separately for the first recovery and the mean of the others. Amplitude criterion for NS.SCR: .0043 logμS.

Furthermore, in several studies on EDA in psychopaths, laterality effects were obtained. The prolonged SCR rec.t/2 found by Hare (1978a) appeared only at right-hand sites. This was interpreted by Hare (1978b) as indicating a dysfunction of the temporal-frontal limbic system of the left (dominant) hemisphere. However, this interpretation requires an ipsilateral control of EDA, evidence for which has not been found (Sect. 3.1.3.4).

Despite psychopathy being an interesting research field with respect to EDA and psychopathology, results from different studies often cannot be easily compared because of problems existing with the diagnosis of psychopathy (Siddle, 1977; Hare, 1978b). Most investigators use questionnaires or rating scales, while several others rely on the appearance of delinquent and/or socially aggressive behavior, sometimes combined with imprisoning or hospitalization, to classify subjects as psychopaths, sociopaths, antisocials, etc. Additionally, some authors differentiate primary from secondary psychopaths, with feelings of guilt or shame being present in the latter ones (Blackburn, 1983), which may further obscure the validity of results obtained with this clinical group.

3.4.1.3 EDA in depressive patients

The clinical picture of major depression, as described by the DSM III-R (American Psychiatric Association, 1987), is characterized by psychological symptoms (e.g., dysphoric mood, feelings of worthlessness or guilt, and thoughts of death, including suicidal ideation), major psychomotoric changes (agitation or retardation), as well as ANS disturbances (e.g., changes in sleep, weight, loss of energy, and various vegetative compiaints). Major or unipolar depression is characterized by the absence of manic episodes, which appear in bipolar affective disorders. The few older studies that used EDA to differentiate depressed patients from normal controls, or subgroups of depressives, were summarized by Stern and Janes (1973). According to these studies, spontaneous EDA as well as electrodermal responsivity may be reduced in depressed patients, except those who are agitated.

Electrodermal hypoactivity and hyporeactivity in depressed patients has been confirmed by several recent studies (Carney, Hong, Kulkarni, & Kapila, 1981; Donat & McCullough, 1983; Lenhart, 1985; Williams, Iacono, & Remick, 1985). According to Iacono et al. (1983,1984a), reductions of SCL, SCR amp., and NS.SCR freq. can be regarded as reliable signs of depressive disorders, since the appropriate differences between normals and depressive patients persist even after the patients have received clinical treatment. Skin conductance was recorded bilaterally with standard methodology from 26 unipolar (20 male, 6 female), and 24 bipolar (16 male, 8 female) depressives, and from 46 normal controls (38 male, 8 female), during a series of 17 tones (1 kHz, 86 dB) including a dishabituation tone of 500 Hz at trial 16, together with an ignore instruction (Iacono & Lykken, 1979). No lateral asymmetries were found in any group (Sect. 3.1.3.4). Unipolar and bipolar depressives had 58% and 54% nonresponders,

respectively, compared to 24% in the normal group. In both patient groups, habituation rate (as assessed by the number of trials to three consecutive nonresponses) was faster, the maximum individual SCR amp. was lower, and the dishabituation responses were smaller as compared to the control groups. Furthermore, average SCLs preceding the tones as well as resting SCLs were significantly lower in patients. Using 6 μS as a cutoff, 96% and 79% of the patients with unipolar and bipolar disorders, respectively, fell below that level, compared with 54% of the normal control group. Twenty-nine of the depressives and 23 of the controls participated in a study one year later, being exposed to eight 86 dB tones, twelve 105 dB tones, eight familiar sounds that reached a peak intensity of 105 dB, and a ballon-burst test, while EDA was recorded as in the year before. The electrodermal hypoacitivity and hyporeactivity of the patients was fully confirmed, showing statistically significant reliabilities between .45 and .69 (Sect. 2.5.2.1).

No influence of treatment on EDA was demonstrated by Dawson, Schell, and Catania (1977) in 20 hospitalized depressed patients (16 females and 4 males) exposed to a series of electroconvulsive shocks. Compared to an age- and gender-matched group of nondepressed controls, the patients showed lower SCLs and smaller SCRs with longer latencies[260] before and after therapy. Unexpectedly, electroconvulsive treatment significantly lowered SCL in depressives. Changes in neither EDA nor HR were related to differences in clinical improvement following therapy. Psychopharmacological treatment with antidepressants or antipsychotic medication also did not influence SCL and SCR amp., which were recorded by Storrie, Doerr, and Johnson (1981) with standard methodology during rest and during performance of three valsalva maneuvers. Both EDA measures yielded lower values in 13 male depressed patients than in 10 healthy male controls, irrespective of treatment.

Two systematic investigations into the usefulness of SCL as a sensitive marker for depression were performed by Ward et al. (1983) with 21 male and 12 female patients, and by Ward and Doerr (1986) with 15 male and 22 female patients, both groups meeting various criteria for unipolar depression. The control group in the 1983 study consisted of 38 male and 33 female subjects, whereas in the 1986 study, 201 males and 204 females served as controls. SCL was recorded bilaterally with standard methodology, and related to electrode area (Sect. 2.3.3.1). The mean log SCL during the 15th and 16th min of the baseline resting period served as EDA measures. Since no significant laterality effects appeared, data was solely reported from the nondominant hand.

In both studies, SCLs in patients were significantly lower than in controls. In addition, there was a significant main effect of gender in the 1983 study, where women displayed lower SCLs than men did (which is quite unusual; Sect. 2.4.3.2); this difference, however, has been found in the 1986 study for patiens only. Therefore, Ward and Doerr (1986) proposed the use of separate cutoff scores for classifying men (< 4.8

[260]Recorded as SR with a constant current of 6.4 $\mu A/cm^2$, using Beckman Ag/AgCl electrodes filled with KY-gel. Measurements were performed during 5 min rest, a free word-association test, stimulation with moderate intense tones and bells, a differential classical conditioning, and an RT task. EDA was expressed in terms of SC parameters.

$\mu S/cm^2$) and women (< 3.0 $\mu S/cm^2$) as depressives. Using these, 90% of patients and controls could be classified correctly. While in the 1983 study the patient/nonpatient dichotomy had been confounded with age, the 1986 study yielded neither main effects of, nor interactions with, age, so that results on EDA and depression cannot generally be attributed to age effects on EDA (Sect. 2.4.3.1). The additionally lowered SCL in patients who demonstrated repeated depressive episodes that was found in the 1983 study must be treated with caution, since the distribution of gender in that particular group was different as compared to patients that were seen for the first time. In accordance with earlier results, the 1986 study did not yield significant differences in mean SCLs between depressive subgroups (e.g., patients with or without dexamethasone suppression, being classified as endogenous or nonendogenous, and being medicated or unmedicated).

However, marked differences between subgroups of depressed patients was found earlier by Lader and Wing (1969), who compared 17 agitated (7 male, 10 female) and 13 retarded depressives (4 male, 9 female) with 35 normal controls (matched to depressed subjects of the same gender). Compared to normals, retarded depressives showed a lower SCL and fewer NS.SCRs,[261] while agitated depressives yielded higher scores in both tonic EDA measures. Furthermore, the habituation rate was much faster in agitated depressives than in normals, as obtained from a series of 20 tones (100 dB, 1 kHz, 1 sec). Slopes for retarded depressives were not calculated, since they gave so few responses. In contrast to Lader and Wing, Dawson et al. (1977), in their study described above, could not find differences in SCLs between six predominantly agitated and six retarded subjects from their depressive patient sample. As Ward and Doerr (1986) pointed out, the results of Lader and Wing that were not confirmed in subsequent reports may be partly due to the diagnosis of depression being more broadly defined in Britain than it is in DSM III-R.

Electrodermal differences between subtypes of depressive patients were investigated by Williams et al. (1985). Thirty-six patients were classified as unipolar (7 male, 20 female) or bipolar (4 male, 5 female) depressives using DSM III-R criteria. No differences between these groups were found in SCL and in SCRs during presentation of soft and loud series of stimuli.[262] This result was in accordance with Iacono et al. (1983, 1984a), who observed that remitted unipolar depressives did not differ in EDA from bipolar patients. However, further classification of patients according to their psychomotor activity and dexamethasone suppression revealed that psychomotor normal (nonretarded, nonagitated) depressives had significantly higher SCLs than psychomotor retarded depressives. Thus, diminished tonic EDA in retarded depressive patients,

[261] Recorded with 9.5 mm diameter double-element lead electrodes unipolarly (abraded arm site above the elbow vs. thumb), using a .05 molar NaCl electrolyte, and 14 $\mu A/cm^2$ constant current.

[262] 1 kHz, 1 sec tones of either 85 or 105 dB; 10 and 12 stimuli, respectively; ISI 20 – 40 sec. SC was recorded bilaterally with standard methodology; SCL measured immediately before each tone and averaged; SCR was obtained within 1 – 3 sec after stimulus onset, with an amplitude criterion of .05 μS.

also found by Lapierre and Butter (1980), can be regarded as a reliable psychophysiological result with respect to subtypes of depression.

Furthermore, EDA may also be useful in determining vulnerability to depressive states in hitherto normal subjects. Lenhart (1985) selected 20 subjects with risk and 20 subjects without risk for depression, as obtained by questionnaire data, from a total of 278 undergraduates, an equal number of each gender. SCLs during rest and SCRs following 20 presentations of 75 dB tones (1 kHz, 2 sec) were recorded with standard methodology. No significant group differences could be obtained in any tonic or phasic EDA parameters. However, when SCR magnitudes were obtained by averaging every four trials, thus reducing the effect of no-response trials (Sect. 2.3.4.2), control subjects showed a significantly greater mean response than did the subsyndromal high-risk depressive group.

In Germany, Heimann (1969, 1978, 1979, 1980), in a series of studies, attempted to differentiate depressed from neurotic patients and healthy subjects by means of their electrodermal reaction type. Based on an activation concept proposed by Claridge (1967), Heimann regarded the EDL as an indicator for tonic activation, while EDR amp. and frequencies were supposed to be dependent on the action of an arousal modulation system (Sect. 3.2.1.1). According to Heimann, agitated depressives can be characterized by an increased tonic EDA (EDL as well as NS.EDR freq.), by reacting more regularly to external stimulation, by showing delayed habituation and easier conditionability, in contrast to retarded depressives, as already found by Lader and Wing (1969). However, as Heimann (1969) showed in a sample of 100 depressed patients, agitated and retarded syndromes are not completely independent, since patients classified as agitated may also express symptoms characteristic of retarded depressives. Heimann (1979) suggested that the report of subjective anxiety and psychomotor restlessness appearing in agitated depressives does not form a part of the original depressive syndrome but instead is a reaction to the patient's general depressive state.

Heimann (1978) used factor analysis to obtain the above-mentioned group-specific EDA patterns. As EDA parameters, SRL and NS.SRR freq. during rest and during different active and passive test situations (e.g., inhalation, habituation to ten 80 dB, 1 kHz tones, tone-noise conditioning, flash discrimination, RT task, word association) were obtained from a total of 277 depressed and depressed-anxious patients, 55 of which belonged to the category of primary affective disorders, and from 73 healthy age-matched controls. Factor analyses for each of these groups yielded a factor structure similar to the two above-mentioned activation factors explaining 70% of the total variance. A stepwise discriminant analysis yielded 84% correct separation of the normals from 73 matched subjects out of the patient group, when a set of 10 variables including HR and respiration rate during rest was used. In another study, habituation to ten 80 dB tones was used by Heimann (1979) to compare depressives with 24 nondepressed neurotics and 32 normals. Frequency distributions of a criterion measure for the end of habituation process (Sect. 3.1.1.3) were similar in the two latter groups, since most subjects did

not show complete habituation after 10 stimulus presentations, while the 297 depressed patients showed a completely different J-shaped distribution.

It seems questionable whether reduced electrodermal reactivity together with faster habituation can be regarded as being specific to depression since subgroups of schizophrenics also show characteristic electrodermal hyporeactivity (Sect. 3.4.2.2). [263] However, Heimann (1980) pointed to the general diagnostic value of these psychophysiological measures, not only for differentiating between subtypes of the illness under investigation, but also for possible specificity of their neurophysiological disorders. With respect to this, it is suggested that inhibition of electrodermal reactivity in depressed patients as well as in schizophrenic nonresponders is due to a similar inhibiting mechanism in CNS information processing (Sect. 3.1.3.1). This was also shown by a reduction of the pre- and postimperative negative variation in the depressive patient's EEG (Giedke & Bolz, 1980). According to Akiskal and McKinney (1975), decreased arousability of depressives following external stimulation, which shows up in a reduction or even in a lack of electrodermal ORs and/or in an increase of habituation rate, can be neurophysiologically attributed to a dysfunction of the "reward" system or to an imbalance between "reward" and "punishment" systems in favor of the latter (Sect. 3.2.1.2). Instead, schizophrenic nonresponding may reflect a secondary adaptation of the organism to psychotic flooding from stimulation (Heimann, 1979). However, it would hardly be possible to test this kind of hypothesis by the sole use of psychophysiological measures in standard paradigms applied to patient samples.

3.4.2 Electrodermal indices in schizophrenia research

The term *schizophrenia* is used for a heterogeneous group of psychotic disorders having some typical symptoms in common (e.g., delusions, hallucinations, as well as disturbances of affect, cognition, and behavior). Despite the use of EDA in schizophrenia research starting as early as the end of the last century, results were rather inconsistent and inconclusive until the early 1970s (Stern & Janes, 1973).

However, since then, studies of electrodermal behavior in schizophrenics have yielded more unequivocal results, due to the use of standardized EDA recording procedures, on the one hand, and to the introduction of subgroups such as electrodermal responders versus nonresponders, on the other (Sect. 3.4.2.2). The appropriate literature has been comprehensively discussed by Öhman (1981). Furthermore, Spohn and Patterson (1979) summarized the results on EDA in their literature review of psychophysiology in schizophrenia. A thorough discussion of methodological problems in this area of research has been provided by Venables (1983).

[263] An interesting hypothesis on the difference between nonresponding in depressives and schizophrenics has been developed by Bernstein, Riedel, Graae, Seidman, Steele, Connolly, and Lubowsky (1988) who found similar patterns in SCR nonresponding but differences in finger pulse volume between both groups. Since depressives showed intact ORs in the finger pulse measure, their deficit in electrodermal responsivity may be due to its cholinergic mediation, thus having a different origin as in schizophranics.

Several neurophysiological hypotheses have been offered to explain the well-replicated psychophysiological abnormality of schizophrenics. For example, Epstein and Coleman (1976) suggested an inadequately modulating inhibition system in schizophrenics, causing them either to over- or underreact. The following two hypotheses formed by Venables are more specifically related to abnormalities in electrodermal behavior:

(1) There is some evidence for disturbances in limbic structures (especially in the hippocampus and in the amygdala) or limbic transmitter systems (dopaminergic and cholinergic fibers) in schizophrenics (Venables, 1983). These structures are closely connected to CNS sources of EDA (Sect. 1.3.4.1).

(2) As a specific indicator of cognitive and attentional processes (Sect. 3.1.3.1), EDA is especially sensitive to disturbances of these functions, mainly indicated by changes in the electrodermal OR (Venables, 1975).

Furthermore, psychophysiological views of schizophrenic etiology, like the vulnerability models proposed by Zubin and Spring (1977) or Nuechterlein and Dawson (1984), consider the EDA to be an especially suitable indicator of the autonomic arousability of these patients (Nuechterlein, 1987). Since comprehensive reviews have been given elsewhere (e.g., Zahn, 1986), the following sections are restricted to some typical topics in schizophrenia research, in which EDA parameters play a predominant role mainly under methodological aspects.

3.4.2.1 Electrodermal recovery and vulnerability for schizophrenia

Various attempts have been made during the last three decades to find risk factors for schizophrenia, including anomalies of the electrodermal system (for a summary, see Watt, Anthony, Wynne, & Rolf, 1984). One of the most serious methodological problems in this field is that psychophysiological differences between clinical groups and normal control groups are often obscured by factors arising from the circumstances of illness. This is especially the case in schizophrenics, where long-lasting hospitalization and heavy medication are likely to be responsible for severe social, intellectual, as well as affective deficits. To avoid this kind of bias, Mednick and McNeil (1968) advocated the use of prospective studies with groups of individuals having an increased risk for schizophrenia instead of comparisons between schizophrenics and nonschizophrenics.

Consequently, the Mednick group performed a longitudinal study (the so-called Copenhagen study), starting 1962-1963 with 207 children (mean age of 15 years) who had chronically and severely schizophrenic mothers (the high-risk group), and 104 controls as a low-risk group, with a follow-up after 10 years, and further reanalyses until recently. Besides other psychophysiological variables, SCRs[264] were recorded during

[264]Recorded with 7 mm diameter zinc electrodes, sponges saturated in zinc sulfate solution used as electrolyte, sites washed and pretreated with alcohol.

a series of eight 1 kHz tones, as well as during a tone-noise (96 dB, 4.5 sec noise as UCS) conditioning procedure (Mednick, 1967). Mednick and Schulsinger (1968) reported that those 20 individuals who suffered from a serious psychiatric breakdown during the first five years of the study showed substantially greater SCR recovery rates (see end of Sect. 2.3.1.3.2) as well as shorter SCR lat. than a matched group of other high-risk subjects without psychiatric problems as well as 20 subjects from the low-risk group. EDR recovery, out of all psychophysiological variables, showed up as the best predictor for a later psychiatric illness (Mednick, 1974).[265]

The predictive value of SCR recovery rate for the appearance of schizophrenia in high-risk subjects was again demonstrated by Mednick (1978) reporting data from the 10-year follow-up of the Copenhagen study. Thirty-four high-risk subjects had become schizophrenic by that time. Ten years before, the SCR recovery rate of that group had been significantly faster than that of stratified groups of high-risk subjects who did not develop an illness and of low-risk subjects. Furthermore, electrodermal recovery rate could predict particular symptoms of hallucinations and delusions ($r = .49$). In extending their previous view on the role of ANS factors in the development of schizophrenia, Mednick, Schulsinger, Teasdale, Schulsinger, Venables, and Rock (1978) suggested an "ANS construct" formed by the product of EDR recovery rate and electrodermal responsiveness. According to this, only in combination with high responsiveness, fast recovery could indicate a predisposition towards the development of schizophrenia which enables the learned evasion of life in those patients.

Erlenmeyer-Kimling (1975) tried to replicate the Mednick results in a prospective study performed with children aged 7–12, 44 of which had schizophrenic mothers, 23 of which had schizophrenic fathers, and 13 of which had both parents diagnosed as schizophrenic, together with 100 control children from normal parents, and 25 children with parents having other psychiatric illnesses. From these data (the so-called New York study), obtained with a similar testing procedure as in the Copenhagen study, Erlenmeyer-Kimling, Cornblatt, and Fleiss (1979) reported generally slower recovery rates in offspring of schizophrenic parents. The only exception was a slightly faster electrodermal recovery in the subgroup with schizophrenic mothers as compared to controls, which did not reach statistical significance. In addition, children of high schizophrenic risk showed longer SCR lat. than the control subjects. In total, with respect to EDA, the results of the New York study were completely contradictory to those of the Copenhagen study (Erlenmeyer-Kimling, Marcuse, Cornblatt, Friedman, Rainer, and Rutschmann, 1984, Table 3). A possibly important difference to the Mednick investigation was that no distinction had been made between subjects with later psychiatric illness and those who stayed healthy. Other differences between the two studies may

[265] Venables (1983) mentioned some critical points in the investigations of the Mednick group. Firstly the ISIs in their classical conditioning paradigm may have been too short to allow separation of the different kinds of EDRs with respect to their latencies (Sect. 3.1.2.1). Secondly, the differences found in SCR lat. may have been confounded with different absolute auditory thresholds for different frequency ranges, as can be observed in schizophrenics.

have been in the diagnostic criteria used, family histories,[266] and in motivational concomitants of taking part in the investigations, in addition to the apparent differences in the age of the subjects studied (Venables, 1983).

Another study that attempted to replicate the Mednick findings was performed by Salzman and Klein (1978) with 12 ten-year old children having one schizophrenic parent, compared to 30 controls. Twenty habituation trials (1 kHz, 2 sec tones, 75 dB) were followed by a tone-noise conditioning similar to the procedure used by Mednick (1967). These authors could only confirm higher SCR amp. to the UCS appearing in the high-risk group, while no differences in SCR lat. and SCR recovery appeared. Janes and Stern (1976), Janes et al. (1978), as well as Prentky, Salzman, and Klein (1981), also could not confirm Mednick's report of faster EDR recovery in children at risk for schizophrenia.[267]

The general conclusion of Mednick and Schulsinger (1974) that EDR recovery may be part of the genetic pattern transmitted from schizophrenic parents to their children has been seriously questioned by the failure of later studies to obtain clear-cut results. Furthermore, a much tougher test of possible genetic influences was performed by van Dyke, Rosenthal, and Rasmussen (1974), using the paradigm of Mednick and Schulsinger (1968). The results did not yield differences in electrodermal recovery between 47 subjects (mean age 33 years) having been adopted-away offspring of schizophrenic parents, and 45 control subjects. In order to gain additional predispositional factors besides the suggested genetic risk, Mednick and Schulsinger (1974) evaluated pregnancy and delivery complications as well as early separation from parents as additional determinants of EDA in their high-risk group. Response amplitudes were most heavily influenced by these complications and by separation, and the latter was correlated with short EDR lat. in the high-risk group. On recovery rate, prenatal and delivery complications showed an effect which was additive to the influence of being an offspring of schizophrenic mothers.

The hypothesis that a short EDA recovery time is possibly a highly specific prognostic indicator for vulnerability of schizophrenia can be supported by connections between an "open gate" state of attentional processes and fast EDR recovery, as outlined in Sect. 3.1.3.1. According to this view, a reciprocal relationship between the duration of electrodermal recovery and the time needed to build up a neuronal model in a Sokolovian sense (Sect. 3.1.1) is suggested. The "open attentional gate" of schizophrenics, which may be due to a predominance of hippocampal over amygdala activity (Table 6,

[266]Mednick (1978) pointed to different selection procedures: the New York study excluded subjects from nonintact families, while the Copenhagen study did not.

[267]Patterson (1976) found among 31 male chronic schizophrenics 11 nonresponders (Sect. 3.4.2.2). The remaining 20 subjects showed a bimodal distribution with respect to SCR rec.t/2, and the fast recovery subjects showed significantly slower pupillary constriction in the light/dark reflex as compared to the slow recovery subjects, which was discussed by the author as possibly due to a greater adrenergic outflow in the first group. In addition, these results also question the generality of the results that shorter EDR recoveries appear in schizophrenics.

Sect. 3.1.3.1), will cause a permanent readiness for reorientation, which also impedes habituation. As Zahn, Rosenthal, and Lawlor (1968) suggested, the slower habituation of this group to specific stimuli is partly due to constant yet partial dishabituation, which is a result of attentional shifts to novel, nonspecific stimuli. Accordingly, the rate of NS.EDRs is frequently increased in schizophrenics (Depue & Fowles, 1973), and children at high risk for schizophrenia show a generally increased tonic EDA (Öhman, 1981). Zahn et al. (1981a) also found an increase of NS.SCR freq. together with faster SCR rec.t/2[268] in a sample of 46 acute schizophrenics as compared to 118 control subjects. Thus, as Mednick et al. (1978) suggested, a combination of electrodermal hyperresponsiveness and fast recovery may be more promising for use in early detection of populations at risk for schizophrenia.

Altogether, these considerations are in accordance with the hypothesis of hippocampal dysfunctions in persons at schizophrenic risk, since they may reflect a suppression of inhibitory influences of the hippocampus on EDA (Sect. 1.3.4.1), which is connected with typical attentional deficits appearing in schizophrenic patients. Predominance of influences from the amygdala over hippocampal influences on attention and information processing as a possible factor in the vulnerability towards schizophrenia may be due to genetic influences as well as to prenatal and perinatal complications. The latter causation is supported by Mednick (1970), who reported delivery complications in 70% of children that became schizophrenic.[269]

However, the role of electrodermal recovery in patients already suffering from schizophrenia remains unclear. Mednick and Schulsinger (1968), Ax and Bamford (1970), as well as Gruzelier and Venables (1972) found higher recovery rates in schizophrenics than in controls. These results could not be confirmed with drug-free schizophrenics. Maricq and Edelberg (1975) found increased electrodermal recovery times in 28 hospitalized schizophrenics free of medication as compared to 27 controls under nonaversive conditions (i.e., rest, mild stimulation, or simple tasks), and no significant differences during an aversive cold pressor test. Furthermore, Gruzelier, Eves, Connolly, and Hirsch (1981b) did not obtain differences between unmedicated schizophrenics and controls in EDR recovery following 70 dB as well as 90 dB stimuli. Gruzelier and Hammond (1978) found faster electrodermal recoveries after applying a 12 sec, subjectively loud and unpleasant noise to 18 schizophrenics when the patients were under chlorpromazine medication as compared to drug-free intervals. Chlorpromazine, a phenotiazine which is frequently applied in neuroleptic treatment of schizophrenia, has marked sympathicolytic properties, and presumably exerts its antipsychotic action

[268] As can be inferred from other publications of the Zahn group, zinc/zinc sulphate electrodes with .79 cm^2 area were attached to palmar sites.

[269] It could be further speculated that different EDA parameters are related to different pathogenetic factors. Cannon, Fuhrmann, Mednick, Machon, Parnas, and Schulsinger (1988), using a subsample from the Copenhagen study, found that subjects with enlarged third ventricles (which may point to hypothalamic and/or amygdala deficits) showed significant overall reductions in EDR amp. and in percentage of EDRs in OR and conditioning trials.

by influencing monoaminergic pathways in the limbic system (Gruzelier and Connolly, 1979), where it may have a direct influence on the elicitation of electrodermal phenomena (Sect. 3.4.3.2). The action of chlorpromazine on EDR recovery in normal subjects was shown by Kugler and Gruzelier (1980), who showed that a single dose of this neuroleptic drug markedly reduced SCR rec.t/2 to moderately intense stimuli. The problem of medication influencing EDA in schizophrenics again supports the need for prospective studies, as stressed at the beginning of this section.

In summary, the results on electrodermal recovery and risk for schizophrenia remain equivocal, since they are not consistent across samples and setting. Nevertheless, there is some neurophysiological plausibility for the specific indicator function of parameters describing electrodermal reaction shape for possible genetic and prenatal or perinatal damages that could be related to the cause of schizophrenia. However, more recent approaches to vulnerability for schizophrenia prefer parameters like electrodermal responsitivity in general, rather than reaction shape. These models will be addressed in the next section.

3.4.2.2 Electrodermal nonresponding in schizophrenics

As Dawson (1990) pointed out, there exists a consensus in results stemming from various research groups, that a large subgroup of schizophrenic patients (between 40 % and 50 %) is electrodermally nonresponsive to innocuous stimulation, whereas the remaining patients show normal responsivity or even hyperresponsivity (for summaries, see Öhman, 1981; Bernstein, Frith, Gruzelier, Patterson, Straube, Venables, & Zahn, 1982; Dawson & Nuechterlein, 1984). Nonresponsivity concerns the failure to elicit any OR or – if an initial OR occurs – an unusually quick habituation to repeated stimulation (Sect. 3.1.1.1 & 3.1.1.3). In addition, these groups normally show differences in tonic EDA, since nonresponders also exert less NS.EDRs, while responders display an increased rate of nonspecific EDRs as compared to normals, despite their neuroleptic medication (Öhman, 1981). While Zahn et al. (1981a), in their study mentioned in the previous section, also found a considerable increase of NS.SCR freq. in their schizophrenic sample during rest, SCL yielded a significant difference in the opposite direction. Accordingly, unidimensional concepts of ANS hyperactivity and hyperreactivity as existing in schizophrenic subsamples must be treated with caution (see also the discussion of phasic/tonic relationships in Sect. 2.5.4.2).

Since, as a rule, studies on electrodermal nonresponding were performed with subjects that had already developed schizophrenia, several factors possibly having influenced the published results have to be considered: the duration and severity of the illness, often confounded with the patient's age; the type and dosage of medication; the patient's compliance as well as his/her ability to understand and follow instructions, the latter being largely dependent on the particular schizophrenic state. Furthermore, differences in intensity and frequency characteristics of the stimuli used, and an induction or prevention of directing attention towards stimulation (as influenced by instructions)

that may be controlled via subjective reactions (Venables, 1983), as well as interactions among all these factors, possibly exert influences on nonresponding.

The first authors who showed electrodermal hypo- and hyperresponsiveness in schizophrenics were Gruzelier and Venables (1972). Out of a total sample of 80 schizophrenic patients, both noninstitutionalized and institutionalized, 43 showed no EDRs to a series of 15 tones (1 kHz, 85 dB; EDA recorded with standard methodology, with the use of KCl paste.). From the other 37 patients, only three reached the habituation criterion of three consecutive SCR amplitudes lower than .05 μS (Sect. 3.1.1.3). A group of 20 nonpsychotic patients as well as a normal control group with 20 subjects showed normal EDRs and a normal habituation course. The result of a bimodal distribution of electrodermal reactivity in schizophrenics was later confirmed in several investigations, but contradictory results were also obtained (Bernstein et al., 1982).

Öhman (1981, Table 1) summarized the results from more than 30 independent samples comprising altogether nearly 1,000 schizophrenics taken from various studies, including patients in different stages of illness as well as during remission, the proportion of nonresponders in the different samples ranging from zero to 69%, with a median, close to 40%. However, these values have to be viewed in the context of a base rate of 5–10 % electrodermal nonresponders in normal and nonschizophrenic psychiatric samples (Venables, 1978; Straube, 1979). Öhman (1981) outlined the following factors that influence the appearance of nonresponding:

(1) Stimulus intensity and quality. A clear decrease in electrodermal nonresponding has been found with higher intensities, though a substantial number of schizophrenics failed to respond even to the most intense stimuli. Several failures of schizophrenics to respond may have been due to the use of tone frequencies below 1 kHz (e.g., Zahn, 1976), where schizophrenics show lower absolute thresholds than normals (Gruzelier & Hammond, 1976). In addition, stimulus rise time may have an important effect on EDR habituation in schizophrenics, as Bernstein et al. (1982, p. 192) showed, presenting a series of 15 tones (1 kHz, 90 dB) with rise times of 25 msec. The introduction of slow stimulus rise times reduced the proportion of nonhabituators within schizophrenics from 32% to zero, and within the controls from 72% to 10%. This points to the possibility that schizophrenics are hyporeactive to OR-eliciting stimuli, while their defense reflexes (DR or startle reflex; Sect. 3.1.1.2) may remain intact (Dimitriev, Belyakova, Bondarenko, & Nikolaev, 1968).

(2) Stimulus significance. Nonresponding mainly appears during the presentation of nonsignalling stimuli. Unfortunately, due to the cognitive disturbances present in schizophrenics, not much convincing data are available. Venables (1975) suggested that schizophrenics show an extreme readiness for an uptake of irrelevant stimulation, while their shortened EDR recovery time following relevant stimulation (Sect. 3.4.2.1) may indicate their slow buildup of a neuronal model in a

Sokolovian sense (Table 6, Sect. 3.1.3.1). Bernstein, Schneider, Juni, Pope, and Starkey (1980) found that schizophrenics display electrodermal hyporesponsiveness essentially to verbal stimuli that were not given explicit attentional significance. Iacono and Lykken (1979) pointed to the possibility that interindividual differences in the significance patients attach to stimuli which are facilitated by vague and uninformative instructions. For habituation experiments with schizophrenics, they recommended providing subjects with an absorbing alternative task after being instructed to ignore the habituation stimuli.

(3) Techniques of recording and scoring. There is a general tendency towards lower proportions of nonresponders in studies that used the constant current method as compared to studies using constant voltage (cf. Öhman, 1981, Table 1). This can be explained by the difference between both methods in amplification requirements (Sect. 2.6.2). In addition, the detection of EDRs is dependent on the time window used (Sect. 2.3.1.1). That means the probability for including a spontaneous EDR in the evaluation of specific EDRs within a window of 1–5 sec after stimulus onset (e.g., Gruzelier & Venables, 1972) is greater than when using a window of 1–2.4 sec (e.g., Levinson & Edelberg, 1985).

(4) Medication. Though responders and nonresponders in general did not differ in type or dose of neuroleptic medication (e.g., Gruzelier & Venables, 1972), subsamples of schizophrenics yielded differential electrodermal effects when given drugs (e.g., Stern, Surphlis, & Koff, 1965). Especially in medication-free intervals, as a rule, was there less nonresponding. However, in most instances the reported intraindividual differences were too small to infer a marked influence of medication on electrodermal responsivity. It can be assumed that neuroleptic treatment reduces EDL as well as NS.EDR freq. (Venables, 1975; see also Sect. 3.4.3.1). Results concerning the influence of these drugs on phasic EDA are less clear. Spohn, Thetford, and Cancro (1971) did not find an impact of phenothiazines on the SCR amp., while Magaro (1973) found that SCR amp. was considerably reduced under phenotiazine medication in schizophrenics and other hospitalized patients. However, SCR amp. were enhanced by drug influences in a schizophrenic subgroup having good prognostic values according to their premorbid adjustment. Whether EDR under medication may be used for prognosis of treatment outcome has yet to be investigated. In any case, the phenomenon of electrodermal nonresponding can be observed in a considerable proportion of schizophrenics, including those not on medication (Öhman, 1981; Zahn et al., 1981a).

With respect to the conceptual/methodological orientation of the present book, the possible influences of measurement techniques as well as medication on the nonresponder phenomenon will be discussed in more detail here.

During the mid-1950s, there was a striking difference in the proportion of schizophrenic nonresponders found in studies performed in Britian (ca. 49%, as summarized by Venables, 1977) as compared to those performed in the United States (at most 14.8%, as summarized by Zahn, 1976). O'Gorman (1978) hypothesized this difference to be due to the use of the constant voltage method in Britain, with an amplitude criterion of less than .05 μS for the detection of EDRs, while the U.S. studies preferred the constant current method, and an amplitude criterion of .4 kΩ (Sect. 2.3.1.2.3). However, Zahn (1978), in reanalyzing his data by eliminating EDRs with amplitudes of less than .05 μS as in the British studies, found only a small increase in the proportion of nonresponders: from 13.5 to 15.3% in the schizophrenics, and from zero to 5% in normal subjects.[270] Instead of differences in measurement, Zahn suggested that medication effects produced the difference between results obtained in both countries, since he used acute and yet unmedicated patients, while the subjects in the British sample were all taking neuroleptic drugs.

Criteria that determine the occurrence of habituation, and hence the proportion of nonhabituators among patients, also influence results on electrodermal responsiveness. Frith, Stevens, Johnstone, and Crow (1982) used the habituation criterion of three consecutive trials with SCRs below .02 μS in a series of 14 tones (1 kHz, 85 dB) for the detection of habituators among their 41 acute schizophrenics before treatment. Therapeutic outcome was markedly better in 15 habituators as compared to 22 nonhabituators (four patients were nonresponders). Comparing these schizophrenics with samples of 34 depressed and 51 anxious patients, nonhabituation was most frequent in the anxious patients and least frequent in the depressed ones, while the schizophrenics were intermediate. However, using form parameters of the habituation curve as a criterion (Sect. 3.1.1.3), schizophrenics showed significantly faster habituation than any other patient group. A reclassification of subjects that considered the amplitude of spontaneous fluctuations confirmed this result. Subjects being classified as habituators had to give two successive responses with amplitudes less than the average NS.SCR amp. Using this criterion instead of zero responses for determining habituators, schizophrenics showed the lowest frequency of nonhabituation of all groups.

Levinson, Edelberg, and Bridger (1984, Table 1) summarized results from 19 schizophrenic samples comprising more than 700 subjects with respect to the time windows used by the different authors. They came to the conclusion that only studies using a long scoring window (4–5 sec after stimulus onset) reported a considerable proportion of nonhabituators among patients. Since NS.EDRs are to be expected within 5 sec after stimulus onset (see Fig. 42, Sect. 2.3.2.2), a distinction between stimulus-elicited and spontaneous EDRs cannot be made unambiguously. In their own study, Levinson et al. (1984) presented 36 male schizophrenic inpatients and 11 male controls two series of 1 kHz tones (1 sec duration; 13 tones with 70 dB, and 12 tones with 90 dB), while

[270]Note that for purposes of transforming SCR amp. into SRR amp. and vice versa, SCLs and SRLs are required (Sect. 2.3.3.2).

recording SCRs with standard methodology, KCl serving as electrolyte. Using a response window between .8 and 5 sec, 56% of patients were classified as nonresponders and 19% as slow habituators. When restricting the window to 1.0–4.2 sec after stimulus onset, 75% of patients were scored as nonresponders, and the remainder as faster habituators than normals. Since the more narrow window led to a clear-cut subgrouping of schizophrenics, and also avoided the danger of spontaneous and OR-elicited EDA, these authors recommended its further use instead of the broader window which had been advocated by the Gruzelier group (e.g., Gruzelier et al., 1981b).

As discussed above under (4), the general question arises whether or not electrodermal nonresponding in schizophrenics is dependent on medication. Since several neuroleptic drugs (e.g., the frequently applied chlorpromazine) have anticholinergic properties, it cannot be ruled out that they directly influence central as well as peripheral elicitation of electrodermal phenomena (Sect. 1.3.4). However, the electrodermal OR blocking effect of 50 mg chlorpromazine, as found by Patterson and Venables (1981) in their study with healthy subjects described in Section 3.4.3.2, was considerably weaker than after the application of 1 mg scopolamine, a potent anticholinergic drug. Furthermore, several studies yielded relative independence of the responder/nonresponder dichotomy from medication (e.g., Gruzelier & Venables, 1972; Gruzelier & Hammond, 1978; Gruzelier, Eves, Connolly, Eves, Hirsch, Zaki, Weller, & Yorkston, 1981c; see Section 3.4.3.2). Straube (1979) also could not find differences in electrodermal reactivity between 21 drug-free schizophrenics and 29 patients tested under neuroleptic medication (including derivatives of butyrophenone, phenothazine, and other tricyclic neuroleptics). Though medication effects on electrodermal nonresponding cannot be ruled out, the general conclusion is that medication does not appear to account for nonresponding (Öhman, 1981; Zahn, 1986).

In order to test the robustness of the electrodermal nonresponder phenomenon, Bernstein et al. (1982) performed a common evaluation of 14 studies drawn from six laboratories in the United States, Britain, and Germany. These studies included chronic and acute schizophrenics of both genders with and without drugs, and normal as well as neurotic subjects as controls. Data from habituation series with both auditory and visual stimuli of different intensities and rise time properties, obtained under differing instructions and conditions, were included.

Electrodermal recordings were taken from palmar sites of either or both hands, with Ag/AgCl or zinc/zinc sulfate electrodes. The amplitude criteria were – according to the measurement technique used – either between 400 and 700 Ω or between .05 and 1 μS; time windows for ORs were either 1–3, 1–4, or .8–5 sec after stimulus onset. Frequency distributions of the number of nonresponsive subjects in each sample were obtained. From responsive subjects, the number of trials to habituation was evaluated, using a criterion of three consecutive trials without an OR.[271] Three different categories were formed:

[271] Data for the two-trial habituation criterion were also analyzed, yielding similar results.

(1) Nonresponders, defined as those subjects who failed to elicit an OR on the first three trials.

(2) Slow habituators, defined as those subjects who failed to habituate before trial number 10.

(3) Percent at both extremes, combining both nonresponders and slow habituators, to test the bimodal hypothesis that schizophrenics are more likely to be on either extreme of the trials-to-habituation distribution.

Statistical tests of the distributions showed that schizophrenics consistently displayed an abnormally high incidence of electrodermal nonresponsiveness (nearly 50% of the patient samples on average). In addition, schizophrenic responders were shown to be faster habituators than nonschizophrenic responders in most studies, though conflicting evidence existed in a minority of studies.[272] Only few of the studies analyzed by Bernstein et al. (1982) provided evidence for the bimodality hypothesis, as proposed by Gruzelier and Venables (1973) or by Rubens and Lapidus (1978).

In general, the distinction between responders and nonresponders in schizophrenics appeared to be reliable across studies and also of clinical utility, showing relationships to the distinction between positive versus negative symptomatology (Dawson, Nuechterlein, & Adams, 1989b). For example, Straube (1979) found that acute schizophrenic nonresponders as compared to responders made more errors of omission in a dichotic listening and verbal shadowing task, suggesting an impaired selective attention capability in electrodermal responders (Sect. 3.1.3.1). Furthermore, that particular subgroup showed more symptoms of emotional withdrawal, conceptual disorganization, and lower spontaneous activity. Similar connections between electrodermal nonresponding and ratings of psychiatric symptoms were found by Bernstein, Taylor, Starkey, Juni, Lubowski, and Paley (1981) in chronic schizophrenics. Gruzelier (1976) reported that slow-habituating schizophrenic responders were rated as more manic, anxious, and attention-demanding than nonresponders.

A correspondence between extreme forms of electrodermal reactivity and the development of positive versus negative symptomatology in schizophrenia has also been found by Cannon, Mednick, and Parnas (1990) in a recent reanalysis of the Copenhagen high-risk study (Sect. 3.4.2.1). Using a decision-tree model of etiology in 138 schizophrenics, they found an increase from 35% to 86% in the rate of developing schizophrenia with predominantly negative symptoms, if these individuals had been ANS nonresponders 20 years before. All seven of the schizophrenics with predominantly

[272]Results on habituation speed are largely dependent on the method used (Sect. 3.1.1.3). The trials-to-habituation criterion disregards initial amplitude differences, thus bearing the danger of misclassifying subjects showing a high amplitude to the first stimulus with subsequent borderline but not below-criterion EDRs as slow habituators. Zahn et al. (1968, 1981a), who defined habituation in terms of the EDR amp. decline relative to the trial block with the largest mean EDR amp., found schizophrenics to be slower habituators than normals.

negative symptoms had suffered severe delivery complications and were ANS nonresponders. This supports the hypothesis that perinatal complications could be a causative factor in third-ventricle enlargement (see Footnote 269, Section 3.4.2.1), which may contribute to electrodermal hyporesponsiveness (Cannon et al., 1988),[273] and is also a typical computed tomographic abnormality in schizophrenics (Cannon et al., 1989). By contrast, in another subsample of 160 schizophrenics from the Copenhagen study, six of the eight patients with predominantly positive symptoms had suffered severely unstable rearing environments and evidenced high levels of ANS responsiveness.

Electrodermal habituation speed has also some predictive value for short-term therapeutic outcome in schizophrenics. Frith, Stevens, Johnstone, and Crow (1979) found that habituation of SCRs to a series of 14 tones (1 kHz, 85 dB), as measured with standard methodology using KY-gel, was a better predictor for improvement than treatment with neuroleptics. These results are consistent with Zahn, Carpenter, and McGlashan's (1981b) finding that only schizophrenic patients who showed EDRs to an RT task similar to those of normals had a marked decrease in their symptomatology.

To answer the question of whether electrodermal nonresponsivity is purely a secondary effect of medication and other treatments or may reflect a long-term trait characteristic possibly associated with vulnerability to schizophrenia, several studies with acute versus remitted patients were carried out. Iacono (1982), in his study described in the next section, found that even in remitted schizophrenic outpatients, 46% were nonresponsive to 86 dB 1 kHz tones, while the remainder showed abnormally high tonic EDA. As a longitudinal follow-up of schizophrenics studied in their early phase by Nuechterlein, Edell, Norris, and Dawson (1986), Dawson (1990) reported data from 22 patients and 22 matched normal controls. He found that the number of trials to habituation in a series of 12 tones (1 kHz, 78 dB) significantly increased in schizophrenics from a state of remission to a state of relapse. Olbrich (1990) did not find statistically significant differences in various EDA parameters, including tonic measures, initial OR amplitudes, and trials to habituation,[274] comparing data from 11 schizophrenics in an acute unmedicated state and after remission, also without medication.

Though the conclusion is only tentative, nonresponding (and possibly also hyperresponding) is likely to be the best candidate as a reliable electrodermal indicator for long-lasting diagnostic and prognostic characteristics in schizophrenics (Bernstein et al., 1982; Levinson et al., 1984; Zahn, 1986), while nonhabituating, probably together with an increased tonic EDA, may have properties of an "episode indicator" in these patients (Dawson, 1990). However, it has to be kept in mind that electrodermal nonresponding is not specific for schizophrenia, since a large number of patients with unipolar

[273] At variance with these results are those of Schnur, Bernstein, Mukherjee, Loh, Degreef, and Reidel (1989), who found significantly wider third ventricles (by means of computed tomography) in 9 schizophrenic responders as compared to 15 nonresponders (SCRs and finger pulse reactions to three 60 dB, 1 kHz tones).

[274] Recorded in a series of 15 tones (1 kHz, 70 dB) with standard methodology, using a time window of .5–4 sec after stimulus onset (Olbrich & Mussgay, 1987).

and bipolar affective disorders (Sect. 3.4.1.3) also show this characteristic, raising the possibility that electrodermal nonresponding reflects genetic liability for several forms of psychopathology (Iacono, 1985).

3.4.2.3 Other issues in schizophrenia research related to EDA

The aim of this section is to discuss additional results of psychophysiological research in schizophrenia directly related to issues of CNS elicitation of electrodermal phenomena. Based on neurological as well as psychological evidence for left-hemisphere dysfunction in schizophrenics, a series of studies on bilateral *EDA asymmetry* (Sect. 3.1.3.4) in these patients have been conducted (for summaries, see Öhman, 1981; Zahn, 1986). In general, electrodermally responsive schizophrenics show higher right-hand than left-hand phasic as well as tonic EDA, while the tonic levels were elevated on the left hand of nonresponders.[275] This was found by Gruzelier (1973) in a study with 60 male schizophrenics and 15 healthy male controls, with an equal number of responders and nonresponders among the patients. Skin conductance was measured bilaterally with standard methodology, using KCl as electrolyte, during the presentation of 15 tones (1 kHz, 85 dB). Since phasic EDA is mainly contralaterally elicited (Sect. 1.3.4.1), failure of contralateral inhibition in schizophrenics due to the left-hemisphere dysfunction of their limbic system had been suggested as a cause of the bilateral differences (Gruzelier, 1979). However, contralateral cortical inhibition of EDA cannot be distinguished clearly from ipsilateral excitation (Venables, 1983; Sect. 3.1.3.4).

An increase of tonic EDA in the left hand, which contrasts with the results of Gruzelier (1983), was found by Bartfai, Edman, Levander, Schalling, and Sedvall (1984) in their study of 13 recently admitted unmedicated schizophrenics. The patients as well as a group of age- and gender-matched controls were presented a series of 21 tones (1 kHz, 85 dB) while SC was measured bilaterally with standard methodology, using hypertonic (.58 molar) NaCl paste. During rest as well as during stimulation, the schizophrenics displayed significantly more NS.SCRs at their left hand than at their right hand, while the controls did not. Furthermore, Öhman (1981) questioned the specificity of elevated right-hand EDLs in schizophrenics, since normal controls may show even greater relative right-hand dominance than patients.

On the other hand, the right-hand superiority of phasic EDA in schizophrenics, which is regarded as a more confirmed finding, possibly also lacks specificity for that group of patients (Zahn, 1986). Gruzelier and Venables (1974) bilaterally recorded SC during an habituation series (15 tones, 1 kHz, 75 dB) and during a discrimination task (1 kHz vs. 2 kHz in a 24 tones series), using the methodology of their 1972 study (Sect. 3.4.2.2). Ten subjects were each tested in subgroups of responders and nonresponders from hospitalized and noninstitutionalized schizophrenics under medication, from a

[275] It has been noted that depressive patients (Sect. 3.4.1.3) tend to show the reverse pattern (Venables, 1983). However, Iacono and Tuason (1983) could not find consistent bilateral asymmetries in EDA in a one-year follow up with 26 unipolar and 24 bipolar depressives.

group of unipolar depressives, and from a group with mixed personality disorders (including psychopathy, drug abuse etc.). Right-hand SCL was elevated in schizophrenics but also in the group with personality disorders, mostly during the discrimination task (i.e., under highly arousing conditions), while the depressives showed left-hand SCL dominance. The same lateralization effects as for SCL were obtained with SCR amp. In addition to the lack of specificity of laterality effects for schizophrenics, their direction was labile and related to arousal levels.

Neuroleptic medication has also to be regarded as an important factor influencing electrodermal laterality in schizophrenics. Gruzelier and Hammond (1977) recorded bilateral SC in 19 schizophrenic patients at the end of subsequent four week periods while they were on chlorpromazine, on placebo, and again on chlorpromazine medication. The right-hand EDR dominance during an OR tone sequence (1 kHz, 80 dB) as found in earlier studies was confirmed. Electrodermal asymmetry was markedly reduced during medication, which was interpreted by these authors as indicating the drug's therapeutic efficiacy. In a further evaluation, Gruzelier and Hammond (1978) reported that chlorpromazine did not consistently influence bilateral differences in the number of EDRs to loud and unpleasant noise stimuli (75 dB, 12 sec) serving as UCSs. However, there was a significant shift from right-hand dominance under placebo to higher left-hand EDR amp. under chlorpromazine. This data provided support for the existence of differential drug effects on signal and nonsignal stimuli, thus demonstrating the complex effects of anticholinergic neuroleptics, as will be outlined further at the end of Section 3.4.3.2.

Whether or not bilateral electrodermal asymmetry is related to diagnostic subgroups of schizophrenia and/or to therapeutic outcome cannot be answered sufficiently. Based on an analysis of EDA recordings[276] during the presentation of 15 tones (1 kHz, 90 dB) to a total sample of 44 undrugged hospital admissions, Gruzelier and Manchanda (1982) as well as Gruzelier (1983) reported evidence that larger left-hand EDRs were associated with "positive" symptoms (e.g., delusions and hallucinations), while higher right-hand responding appeared together with "negative" symptoms (e.g., emotional withdrawal; see also Sect. 3.4.2.2). Gruzelier (1979) suggested the prognosis was worse in schizophrenics showing right-hand EDR dominance than in those not showing that dominance.

No bilateral electrodermal asymmetry could be found by Iacono (1982) in 24 remitted schizophrenics who were compared to 22 medical outpatient controls during exposure to 17 tones (1 kHz, 86 dB; including a dishabituation trial 16 with an unusually long 500 Hz tone). None of the EDA parameters, measured with standard methodology (NS.SCR freq., SCL, number of SCRs to tones, SCR amp. to the first and to the dishabituation tone), yielded differences between hands in patients or in controls. This result is in accordance with Tarrier, Cooke, and Lader (1978), who found no evidence

[276]Measured as SCRs occurring between .8 and 5 sec after stimulus onset, exceeding a 1 mm criterion with a maximum gain setting of .02 μS/cm.

for bilateral EDA asymmetry in 18 partly remitted schizophrenics during 15 tone presentations (800 Hz, 85 dB). Considering other conflicting results obtained with acute and chronic schizophrenic patients (summarized by Zahn, 1986), right-hand dominance of EDA cannot be regarded as a stable trait in schizophrenia.

Another interesting line of research with respect to specificity of EDA parameters for schizophrenia focuses on differences in NS.EDR freq. during the presence and absence of relatives rated as high or low in "*expressed emotion*" (for a summary, see Turpin et al., 1988). Tarrier, Vaughn, Lader, and Leff (1979) reported data from 21 schizophrenic outpatients and 21 age- and gender-matched normal subjects that were tested with Tarrier et al.'s (1978) method in the laboratory. Additionally, electrodermal recordings were taken from the patients at their homes for 15 min under both conditions: with and without the presence of their key relative, who had been rated as either high or low on expressed emotion (EE) two years before. When compared to the controls, schizophrenics showed elevated levels of NS.SCR freq. during the presence of the experimenter alone. While the spontaneous EDA of subjects with relatives low on EE declined after the entry of their key relative, patients with high-EE relatives continued to show higher rates of nonspecific EDA than did normals.

Similar results were obtained by Sturgeon, Kuipers, Berkowitz, Turpin, and Leff (1981) with 20 acute schizophrenic patients during an interview conducted with the patient's key relative, whose EE had been measured earlier. Spontaneous SCRs (recorded with standard methodology using KY-gel; amplitude criterion = .02 μS) were significantly reduced after the relative's entry for patients with low-EE relatives but not for patients with high-EE relatives, as measured by individual regression slopes. The authors suggested that high-EE relatives may support the maintenance of a chronic state of high arousal in schizophrenics by stressful social interactions, while low-EE relatives help the patients to adapt to stress by their supportive attitudes. Thus, the probability of relapse should be greater in the former group of patients as compared to the latter.

In order to test this hypothesis, Sturgeon, Turpin, Kuipers, Berkowitz, and Leff (1984) performed a follow-up study with 19 schizophrenics that were at high risk for relapse (i.e., their relatives were high EE), testing them with the methods used by Sturgeon et al. (1981) during acute illness and nine months after discharge. During the initial testing, patients with high-EE relatives showed a significantly higher NS.SCR freq. than a control group of 11 patients with low-EE relatives, regardless of whether their key relative had been present or not. Patients with high-EE relatives were divided into a group that was offered a number of social interventions in order to reduce the relative's EE and/or contact with the patient and another group that received no such training. This kind of intervention was highly successful in reducing relapse rates. However, its effects were not directly mediated via influences on spontaneous EDA, since patients whose relative changed during intervention from high to low EE did not reduce their rates of NS.SCRs to those of the original patient group with low-EE relatives. Nevertheless, EDA seemed to be another independent indicator of the susceptibility to schizophrenic relapse, since NS.SCR freq. during initial testing was

significantly higher for those patients who subsequently relapsed than for those patients that remained well.

It remains to be investigated in what respect and to what extent emotional expression as a social communication factor, which has shown close relationships to phasic electrodermal parameters (Sect. 3.2.2.1), and the rate of spontaneous EDA as a possible vulnerability indicator for schizophrenic relapse (Sect. 3.4.2.2) depend on each other during the course of schizophrenic illness. As Olbrich (1989) pointed out, the different theoretical positions of a purely emotional arousal indicating the property of spontaneous EDA versus its possible dependency on the schizophrenic's excessively allocating central processing capacity to irrelevant stimuli (Sect. 3.1.3.1) should be focused on in future research.

3.4.3 EDA as an indicator in psychopharmacological treatment of anxiety

Among the various kinds of drugs that have been used in the treatment of acute as well as chronic anxiety, the so-called minor tranquilizers are regarded as having the highest degree of specificity with respect to influencing anxiety. Despite the minor tranquilizers having contributed considerably to the progress in anxiety treatment since the mid-1950s, and even labelled as "antianxiety drugs" (Solomon & Hart, 1978; Rickels, 1978), their specificity remains a matter of debate (Janke & Netter, 1986). This is due to both conceptual as well as methodological deficits in human antianxiety drug research. However, during the last decade there has been considerable progress in evaluating the neuropsychological background of anxiety and in linking psychophysiological variables to anxiety. EDA has been demonstrated as being a most sensitive indicator of anxiety trait and states.

Anxiety is not only influenced by minor tranquilizers but, in addition, by other kinds of drugs such as hypnotics (e.g., barbiturates or alcohol), small doses of neuroleptics (e.g., phenothiazines or butyrophenones), which are labelled "major tranquilizers," and by other drugs with a different main action on the CNS, but anxiolytic side-effects (e.g., MAO-inhibitors, opiates, or tricyclic antidepressive agents like imipramine), peripheral-acting agents like beta-blockers (e.g., propanolol), or muscle-relaxating drugs (Rick, 1978; Lader & Petursson, 1983; Janke & Netter, 1986).

Practically no therapeutic use can be made of opiates, barbiturates, or alcohol because of their addictive properties. MAO-inhibitors and tricyclic antidepressants show considerable antianxiety effects especially during phobic anxiety and panic attacks (Klein & Rabkin, 1981), but do not generally influence anxiety states. Therefore, conceptual research on antianxiety drugs has to focus on the differentiation between minor tranquilizers as specific and neuroleptics or major tranquilizers as nonspecific anxiety-reducing agents. In addition, these drugs acting centrally on anxiety should be concep-

tually separated from drugs like beta blockers, the primary action of which is on the adrenergic receptors in the periphery.

In a first approach, pharmacological effects on anxiety may be divided into primary and secondary ones. Drugs that show primary action on anxiety-evoking neurophysiological structures are benzodiazepines and hypnotics. During the late seventies, specific benzodiazepine receptors were discovered at limbic and thalamic sites both in vitro (Squires & Braestrup, 1977) and in vivo (Williamson, Paul, & Skolnick, 1978). These receptors are linked to GABA receptors, thus increasing the inhibitory action of GABA via a specific postsynaptic receptor (Guidotti, Baraldi, Schwartz, & Costa, 1979). According to Gray (1982), benzodiazepines increase the GABA-ergic inhibition of noradrenergic activity stemming from the locus ceruleus as well as serotonergic fibers from raphe nuclei, because of the benzodiazepine receptors being located adjacent to the GABA receptors. Both monoamines are regarded as acting together in improving the analysis of stimuli within the basal hippocampal-stop circuit while influencing the gating process between the dentate gyrus and the hippocampal area CA3. The resulting more thorough analysis of the whole stimulus situation is accompanied by an increased activity of the septo-hippocampal stop system or BIS (Sect. 3.2.1.2), which causes inhibition of behavior in the rat during anxiety. Thus, the antianxiety action of benzodiazepines can be labelled as primary, since they directly influence limbic neurotransmitter systems involved in the origin of anxiety.[277]

The antianxiety mechanism in the action of the so-called major tranquilizers is even less known. It can be assumed that they do not specifically influence anxiety but reduce general arousal via their sedating properties (Lader, 1979), together with a general reduction of muscular and emotional tension (Janke & Netter, 1986). Hence, their anxiety-reducing mechanism is labelled as a secondary one.

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[277]The presumed specifity of benzodiazepine receptors for the pharmacopsychological influence on anxiety has been questioned by Janke and Netter (1986). Firstly, those receptors also appear frequently in various systems that are not directly tied to anxiety (e.g., in motor systems or in the spinal cord), which gives rise to the conclusion that they are involved in anticonvulsive rather than in antianxiety actions of benzodiazepines. Secondly, specific endogenous substances that are produced to counteract anxiety states (similar to endogenous opiates during pain), could not be detected until now. In addition, the development of experimental benzodiazepine antagonists like the beta-carbolines did not yield systematic results with respect to evoking anxiety (Rommelspacher, 1981).

Despite the specificity of the benzodiazepine-GABA link as a mechanism underlying anxiolytic effects of those drugs, it needs further empirical support, from both human as well as animal studies. The theoretical approach provided by Gray (1982) may be included as one element of a general framework of tranquilizer-action upon anxiety influencing information processing. Hypnotics like barbiturates as well as alcohol are also primary-acting antianxiety agents since they also enhance GABA-ergic inhibition of monoaminergic fibers that facilitate anxiety reactions in the limbic system. They do not directly increase the production of GABA, being instead indirectly acting GABA-mimetics, because of their property to bind to the picrotoxine receptors, thus preventing picrotoxine from acting as a GABA-antagonist. So GABA seems to be the common transmitter in the CNS for all primary-acting antianxiety agents. However, this could not be directly proved since the application of GABA did not show anxiolytic effects in animal studies (Koella, 1986).

Anxiety reduction is not only subjectively experienced but can also be measured objectively by means of physiological and behavioral parameters, the latter being predominantly used in animal research. Anxiety states are characterized by a markedly increased general activation (Sect. 3.2.1.1). Hence, antianxiety agents are expected to show at least some deactivating properties, both subjectively reported and objectively measured by various psychophysiological variables. However, inducing deactivation is common in several classes of CNS drugs. Therefore, physiological variables to be used as indicators in the psychopharmacological treatment of anxiety should show at least some specificity with respect to the phenomenon in question.

The specific indicator function of tonic EDA with respect to BIS activity as worked out by Fowles (1980) relies on research in the field of conditioning and stress, mainly focusing on the appearance of stimulus-dependent as well as spontaneous EDRs, and not on the EDL, which is generally regarded as being influenced more by peripheral physiological states (e.g., corneal hydration; Sect. 1.3.4.2) than by fear-evoking stimuli. Therefore, the following section mainly focuses on the NS.EDR freq. as a specific indicator for anxiety reduction by classical antianxiety drugs of the benzodiazepine type.

There are two different approaches in the psychophysiological investigation of psychopharmacologic actions:

(1) The clinical approach, which compares a group of patients, mostly neurotics or persons with generalized anxiety syndrome, with a similar control group which, however, shows no symptoms of anxiety. Using patients as subjects frequently yields the disadvantage of having no placebo control, because often placebo cannot be used for ethical reasons.

(2) The experimental approach, which uses only subjects without anxiety symptoms, who are assigned at random to a group either with or without experimental induction of anxiety. Despite using normal subjects, personality traits like neuroticism or trait anxiety should be controlled. The problem here is the validity of results with respect to the more severe forms of anxiety in patients.

Since both approaches show specific advantages as well as disadvantages, they have been used as complementary research strategies in psychopharmacological research.

3.4.3.1 Studies with benzodiazepines

Unfortunately, clinical studies of the above-mentioned type (1) using physiological measurements are rare, as most of them use global criteria of improvement following drug application obtained by the doctor's ratings or self-ratings (Giedke & Coenen, 1986). However, when physiological recordings were obtained, specific drug actions with respect to anxiety reduction were more likely to be observed in EDA parameters as compared to other variables.

In a study using 30 neurotics of both genders who reported anxiety as their main symptom, Lapierre (1975) found acute, subacute, as well as chronic effects of diazepam in electrodermal but not in cardiovascular or respiratory measures. Following a one-week wash-out period, subgroups of 10 patients each received three times a day either 5 mg diazepam, 7.5 mg chlorazepate (a benzodiazepine under investigation), or placebo following a double-blind schedule. Acute drug actions were recorded 3h after the first application, the subacute ones 14 days later and the chronic ones 28 days later. As compared to placebo, diazepam yielded a significant reduction of the NS.EDR freq. under resting conditions as well as an increase of EDR lat. under stimulating conditions on all three occasions. Chlorazepate effects were present only during the 3h post application measurement period: a decrease in NS.EDR freq. together with an increase of the number of EDRs elicited by a nonspecific stimulation, and no subacute and chronic effects on EDA. No significant verum-placebo differences were found in HR, blood pressure, or respiration frequency.

The studies reported below assigned neurotic outpatients at random to four drug conditions. The first one started with 5 mg diazepam twice a day, followed by the same dosis three times a day together with another 5 mg during the night. The second one started with 25 mg amitriptyline (an anti-depressant) twice a day, followed by the same dosis three times a day together with another 50 mg, and later on 75 mg during the night. A combination of both drugs not to be reported here served as a third and a placebo as a fourth condition. In the first study (Johnstone et al., 1981), 181 patients of both genders, who showed either strong depressive or anxiety symptoms and were not previously drug treated, were subjected to an habituation procedure using 14 tones of 85 dB. As compared to the baseline recordings, the NS.SCR freq. as well as the SCL were reduced under all conditions. However, the NS.SCR freq. decreased to the greatest extent under diazepam, showing again its validity as a measure of anxiety reduction. The SCR rec.t/2, which was increased under placebo, decreased under diazepam, and remained unchanged under amitriptyline. The plasma concentration of diazepam, as measured by a benzodiazepine receptor-binding technique, yielded a highly significant correlation with the SCR recovery, while all other correlations of drug plasma concentrations with EDA measures remained nonsignificant. Johnstone et al. (1981) interpreted these results as pointing to a specific indicator function of EDR recovery for a drug-induced anxiety reduction. This finding is in accordance with the hypothesis discussed in the previous section; that is, EDR recovery is shorter during states of great readiness for information uptake, together with distributed attention, which appear when hippocampal activity exceeds activity of the amygdala (see Table 6; Sect. 3.1.3.1).

The second study (Frith, Stevens, Johnstone, & Owens, 1984) applied the same drug conditions as Johnstone et al. (1981) to 91 patients who received an habituation series of 17 tones (1 kHz, 85 dB) together with a 100 dB, 2 kHz tone interspersed after the 15th tone, which they should regard as irrelevant, and also to 71 patients who underwent 21 simple reaction time tasks using the same tones preceded by a warning signal (ISI between 2 and 8 sec). While amitriptyline showed a small reduction of tonic and pha-

sic skin conductance under each experimental condition, there was a reduction of the SCR amp. following the irrelevant tones. Differential effects appeared during the reaction time paradigm. Subjects who at the beginning showed more than optimal arousal yielded more errors together with higher SCR amp. under placebo, while patients with an increased error rate showed the lowest SCR amp. under diazepam. According to these authors, this result indicates an influence of diazepam on the general arousal level as well as on attentional processes, as indicated by electrodermal measures.

Further evidence for specific influences of benzodiazepines on EDA parameters stems from experimental psychopharmacological studies of the above-mentioned type (2). Boucsein and Wendt-Suhl (1976) investigated the action of 20 mg chlordiazepoxide as compared to placebo upon two different anxiety-evoking conditions and a control condition, using 90 healthy male paid volunteers in a double-blind schedule. Fifty minutes after application, the subjects were instructed to expect an electric shock 2 times as strong under a weak-stress condition and 5 times as strong under a strong-stress condition in relation to a shock they had previously found unpleasant. During the anticipatory interval of 20 min duration, the course of which the subjects pursued on a digital clock, NS.SRR freq., HR, respiration rate, and a rating of subjective emotional arousal were continuously recorded. In both electrodermal as well as subjective variables, significant main effects of the stress factor were only observed within the last 2 min of the anticipation interval (Sect. 3.2.2.2). However, interactions between drug and stress conditions reached significance for several minutes in an earlier phase of anticipation. In the middle of the interval, chlordiazepoxide reduced the NS.SRR freq., but only during the strong-stress condition and not during the weak-stress one. In the control group without stress, so-called paradoxical effects of chlordiazepoxide appeared, yielding a higher NS.SRR freq. than the placebo. These results were in parallel to the subjective ratings of emotional arousal, while the other physiological variables yielded no significant drug effects, except a general HR decrease under chlordiazepoxide previous to the stress-inducing instructions.

To compare the anxiety-reducing effects of 5 mg diazepam and 3 mg cloxazolam (a benzodiazepine under investigation), Boucsein and Wendt-Suhl (1982), in a placebo-controlled double-blind study with 144 male paid volunteers, used three different stress conditions in a permuted order, which were paralleled by appropriate neutral conditions in the control groups. The 30 min stress period, during which skin resistance, HR, and spontaneous EEG activity were continuously recorded, started 170 min after application of the drugs. One stress condition consisted of two subsequent 3-min anticipation periods of an unpleasant electric shock, the second of which had been announced as being twice as strong, which, however, was not the case. They were separated by a 2-min interval and pursued by the subjects on a digital clock. Another stress condition used a speech anxiety paradigm (Boucsein & Wendt-Suhl, 1980), and the third stressor was the presentation of 12 anagrams, 10 of which were unsolvable in the experimental group but not in the control group (Boucsein & Frye, 1974). While cloxazolam showed results which were heterogeneous and atypical for tranquilizers, diazepam yielded dif-

ferential effects indicating its specificity to influence anxiety as well as the specific indicator function of EDA with respect to anxiety reduction, since a decrease in the mean range-corrected amplitudes of NS.SRRs (Lykken et al., 1966) under diazepam as compared to placebo could only be observed when anxiety-evoking conditions like anticipation of electric shocks or public speaking were applied, but not with an unspecific stress-evoking condition, such as trying to solve unsolvable anagrams. No significant differences between diazepam and placebo appeared in HR.

In summary, EDA can be used as a sensitive and valid indicator of anxiety-reducing properties of benzodiazepines (and of other so-called minor tranquilizers as well). However, anxiety-evoking conditions must be carefully selected for use in this type of research. Especially when testing new drugs it is necessary to maintain the induced anxiety states over a certain period of time. This may be achieved by the use of different anxiety-evoking situations.

3.4.3.2 Studies with beta-blockers and neuroleptics

During the 1980s, there was an increase in the use of beta-blockers as antianxiety agents (Netter, 1986). Their influence on anxiety may be mediated peripherally as well as centrally, since they prevent HR increase, bronchial dilatation, and glycogenolysis in the liver by blocking adrenergic beta-receptors, thus reducing typical autonomic and humoral concomitants of anxiety and stress states. However, since beta-blockers normally pass the blood-brain barrier easily, at least a part of their antianxiety property can be presumed to stem from their influences on beta-adrenergic synaptic transmissions within the CNS. As Gruzelier and Connolly (1979) pointed out, there is a direct influence of beta-blockers on the limbic system, bringing about actions which are characteristic of a preponderance of amygdala activity. They showed that propanolol induced a normal course of electrodermal habituation in schizophrenic slow-habituators. The particular deficit of habituation in these patients is supposedly due to a dysfunction of the amygdala, leading to hippocampal predominance (see Table 6; Sect. 3.1.3.1).

Besides central influences of beta-blockers on the electrodermal reaction, these agents may also influence EDA peripherally in different ways. Though sweat gland innervation is now assumed as being primarily cholinergic, there have always been discussions concerning their additional adrenergic supply (Sect. 1.3.3.1). Therefore, in addition to the widely used parasympathicolytics and parasympaticomimetics in the study of peripheral mechanisms of EDA (Muthny, 1984), direct actions of sympathicolytic and mimetic substances on EDA in the periphery have also been discussed (Edelberg, 1972b). In addition, beta-blockers may exert indirect influences on EDA via their vasomotor effects (i.e., via reduction of skin blood flow), the influence and contribution of which on EDA remains equivocal.

Experimentally controlled studies of the above-mentioned type (2) comparing the antianxiety effects of beta-blockers with those of benzodiazepines using EDA are sparse, since most studies restrict themselves to cardiovascular variables. One study was per-

formed by Farhoumand, Harrison, Pare, Turner, and Wynn (1979), who compared 480 mg oxprenolol with 2 mg lorazepam and placebo in a balanced within-subject design using six male subjects. Though given in a considerably high dosage, the beta-blocker yielded less effects on EDA than the benzodiazepine during stress produced by physical exercise.

Erdmann, Janke, Köchers, and Terschlüsen (1984b), in a 3 by 3 factorial design with independent groups, assigned 108 male subjects to either 40 mg oxprenolol, 5 mg diazepam, or placebo, following a double-blind design. Under each drug condition, two speech anxiety groups and one control group were formed. Subjects with an induction of high speech anxiety had 5 min to prepare a talk to be given in front of experts on TV, while the low speech-anxiety group had to give the speech on tape for later analysis purposes. The control group had to answer a questionnaire covering the same topic after the anticipatory period. Using measures from a 10 min baseline as covariates, NS.SRR freq., HR, and blood pressure were recorded during 10 min following the 5 min preparation phase. Drug main effects were only significant for HR, due to the direct influence of the beta-blocker. Instead, all physiological variables depicted the graded anxiety effects as attempted by the experimental design. Interactions between these and the drug effects reached significance for the NS.SRR freq., which was increased under oxprenolol in the neutral condition, showing, in turn, a tendency to be reduced under diazepam in the high speech-anxiety condition.

Since both studies did not yield antianxiety effects of beta-blockers that are typically observed under benzodiazepines, a model of two classes of drugs influencing anxiety in a different manner can be proposed: the centrally acting benzodiazepines which mainly influence EDA, and the beta-blockers, the action of which can be recorded most appropriately by HR, thus influencing only peripheral concomitants of anxiety. As Netter (1986) stated, subjective anxiety can be most effectively reduced by beta-blockers if one of its main causes is the irritating effect of an elevated HR.

Several studies compared the beta-blocker propanolol, which passes the blood-brain barrier more easily than oxprenolol, with different phenothiazines (i.e., neuroleptics). Gruzelier et al. (1981c) reported results from three habituation studies of the above-mentioned type (1) on the action of propanolol as compared with phenothiazine or chlorpromazine, performed with schizophrenics and normal controls. Besides the normalization of the habituation in one subgroup of patients, reported by Gruzelier and Connolly (1979) using the same data set (Sect. 3.4.2.1), an OR reinstatement in schizophrenic electrodermal nonresponders was observed under propanolol. Since these effects appeared independently of tonic electrodermal changes in either SCL or NS.SCR freq., the authors concluded that there was a specific influence of beta-blockers on the OR and its habituation without inducing changes in general arousal, as appeared under the influence of phenothiazines. By contrast, the phenothiazines did not influence the patient's abnormal habituation when stimuli of a medium intensity (70 dB) were used. Only in the third study, where 90 dB stimuli had been applied to 12 schizophrenics, did an average dose of 320 mg chlorpromazine bring about a general reduction in SCR

amp. Whether or not beta-blockers influence the OR, while phenothiazines act upon defensive reactions, as Gruzelier et al. (1981c) concluded, remains a testable hypothesis in psychopharmacological research.

However, as with the one of beta-blockers, the influence of neuroleptics on EDA as an indicator of anxiety reduction must be discussed with care, since these drugs also show clearly anticholinergic properties. In addition, another central mechanism acting directly on the elicitation of EDA may be involved when using neuroleptics.

Evidence comes from a study performed by Patterson and Venables (1981) with 12 healthy male subjects. These authors used two different types of neuroleptics – 3 mg haldol, which blocks the dopaminergic fibers, and 50 mg chlorpromazine, which has both anti-catecholaminergic and anticholinergic properties, together with 1 mg of scopolamine (an anticholinergic which easily passes the blood-brain barrier) – and placebo in a within-subjects design. After 15 min of rest, the electrodermal OR following a 1 kHz 75 dB tone of 1 sec duration and a short rise time of 15 msec was recorded under each drug condition. Four subjects did not show any OR; they were classified as nonresponders. In the remaining subjects, the SCR completely disappeared under scopolamine, while chlorpromazine reduced the SCR amp. and shortened the rise time as well as the SCR recovery time significantly. By contrast, halidol increased the SCR amp. together with a shortening of the SCR recovery time. This result shows that the influence of anticholinergic neuroleptics such as chlorpromazine is not mediated via a general deactivation, since halidol increased the amplitude of the electrodermal OR. Neuroleptics having anticholinergic properties exert their influence on EDA presumably via a depletion of central and possibly also peripheral acetylcholine stores, thus invalidating the specific function of EDA as an indicator of anxiety reduction via a central neurophysiological mechanism, which occurs with benzodiazepines.

In summary, though electrodermal measures show a considerable specificity for indicating a successful anxiety treatment with antianxiety drugs of the benzodiazepine type, the same cannot be stated for the anxiety reduction due to the pharmacological action of other types of drugs used in anxiety treatment such as beta-blockers or neuroleptics. Beta-blockers exert their antianxiety property mainly via a reduction of typical adrenergic-innervated concomitants of anxiety. Therefore, their effects on HR will be more prominent and probably more specific than effects on EDA. Neuroleptics are likely to influence EDA mostly via their anticholinergic properties, and they do not act directly on the neurophysiological structures in which anxiety is likely to originate.

3.5 Miscellaneous applications of EDA

Besides their clinical use, methods of electrodermal recording are present in several fields of applied psychology. In addition to these applications, the present chapter discusses the use of EDA in medicine. The main aim of this part is to stimulate interdisciplinary cooperation in the development and application of electrodermal methodology, for example, between psychophysiologists and dermatologists (Sect. 3.5.2.1).

3.5.1 EDA in various fields of applied psychology

Because EDA is one of the most sensitive psychophysiological indicators of stress (Sect. 3.2.2.2), its application should be important in stress-strain research in the work place (Sect. 3.5.1.1). However, since the best sites for EDA recordings are on the palms and soles (Sect. 1.3.3.3 & 2.2.1.1), measuring EDA during actual work may impede performance, and the results are likely to show an increased artifact proneness as well (Sect. 2.2.5.1). Therefore, EDA as a stress indicator in this field has been less used than HR, which is easier to record.

A widespread though controversial use of EDA in applied psychology is in the detection of deception (Sect. 3.5.1.2). Here, EDA as a sensitive and easy-to-evaluate method of detecting even small differences in emotional reactions to stimuli is superior to various other psychophysiological indicators.

Other possible fields of application where EDA has not been frequently used are developmental and social psychology. With respect to the former, the reader is referred to Section 2.4.3.1 as well as to a summary by Porges and Fox (1986). The use of EDA in social psychology has been summarized by Schwartz and Shapiro (1973) and more recently by Cacioppo and Petty (1986). Additional information on EDA and interpersonal communication was provided in Section 3.2.3 of the present book.

3.5.1.1 EDA in engineering psychology

The main use of EDA in the field of engineering psychology is as an indicator function for increased levels of activation and stress. One domain where EDA recording has been systematically applied is *traffic research.* Michaels (1960) conducted SR recordings from the fingers of 10 urban drivers using streets differing in traffic volume. Spontaneous SRRs were detected, and a time window between 5 sec before and 1 sec after their onset was evaluated, with an event in traffic serving as trigger. Sixty percent of SRRs could be related to unpredictable events like vehicles exiting their lane or crossing the road. In addition, mean SRR frequency was 40% lower on the individually preferred road than on an alternative route.

In another study, Michaels (1962) used EDA to differentiate among the stress-inducing characteristics of different expressway designs combined with traffic volume. Six male subjects drove an 8–10 mile section of each highway four to eight times at

off-peak hours, while events causing a speed or placement change were recorded. The relation between NS.SRR freq. and traffic volume was statistical significant, showing a linear increase up to 1,400 vehicles per lane per hour. For greater volumes, nonspecific EDA rose exponentially up to the maximum of 1,800 vehicles per lane per hour. When data were corrected for volume, significant differences between types of routes were demonstrated, dependent on the frequency of occurrence of conflicts with merging and exiting vehicles. These results indicate that NS.SRR freq. may be used as a valid measure of driver stress, which is directly related to frequency and predictability of interferences with driving. Though this result parallells those obtained in laboratory stress research (Sect. 3.2.4), it should be treated with more caution because of uncontrolled environmental influences on EDA.

The effect of rural intersection illumination on stress in four male drivers was investigated by Cleveland (1961). Each driver performed 12 runs through the intersection, twice on each of the six paths, once with illumination and once without, while EDA was continuously recorded.[278] NS.EDR freq. as well as mean EDR amp. were 20% less when the intersection was illuminated than when it was without illumination.

Taylor (1964) reported data on EDA, driving behavior, and accident rate of drivers from two studies performed with 12 subjects (7 male, 5 female) and 8 subjects (4 male, 4 female). NS.EDR freq.[279] was recorded while subjects drove standard routes during different times of day and night (which meant differences in traffic density and illumination) in permuted order. No covariation of EDA with traffic density and illumination was found. NS.EDR freq. showed a positive correlation to the number of turns per mile (r = .67), while correlating negatively with the average driving speed (r= –.75). Furthermore, in the first experiment, a positive correlation (r= .61) was obtained between NS.EDR freq. and the average number of personal-injury accidents (taken from police records) per estimated vehicle mile (by using calculations of traffic flow). Taylor interpreted his correlational data as being possibly dependent on the number of turns per mile as the determining variable for an increased number of EDRs, for more accidents, as well as for lower speed. Another result was that the mean number of NS.EDRs per min exponentially decreased with driving experience, which, however, was confounded with age (Sect. 2.4.3.1).

To investigate possible connections between EDA and accident rate, Preston (1969), working in Britain, used insurance classifications of her subjects (reflecting increasing premiums with number of accidents). In two studies, 17 and 21 subjects of both genders drove over routes including narrow country roads as well as town roads. SRR amp.,[280] summed up per km, served as dependent variable. No general effects of age, gender, or

[278]Measured with AC from the left hand's fingers, using a Wheatstone bridge.

[279]Recorded with AC (65 Hz), using 10 μA/cm^2 average current density, from the subject's fingers, data transformed into conductance changes.

[280]Recorded volar/dorsal from the left foot with 1 cm diameter electrodes filled with a paste from bentonite, glycerin, and Ringer's salt solution, using constant current of 5 μA/cm^2 and a Wheatstone bridge.

insurance classifications appeared. However, separate evaluations for different types of roads yielded a significantly higher rate and intensity of EDA in subjects with higher accident rates while driving on the country roads but not in town. Preston's interpretation was that driving in town is more limited, while driving in the country allows subjects to show risky behavior. Here EDA is a very sensitive measure of the emotional strain exhibited when drivers are not absolutely certain of exerting full control.

In Sweden, Helander (1974) continuously recorded SR[281] from 60 subjects with a wide range of age and experience in driving, on four different routes. In addition, HR, EMG from two muscle groups of the right leg (indicating release of accelerator and lifting of the leg for braking), as well as various vehicle variables (velocity, acceleration in three directions, steering wheel angle, and brake pressure) were recorded, and up to 25 traffic events were encoded by the experimenter. Measurement values for all variables, being formed for each 10 meters, were rank correlated. While Helander could not interpret a correlation of .56 between SRL and steering wheel angle, he found that EDRs were largely dependent on braking activity, since the covariation between the latter and SRR amp. was as high as .89. Since no such correlation was obtained during pressing the brake in the unmoving car, the EDR while braking was more likely to be psychologically determined than a motoric artifact.

In a reanalysis of these data, Helander (1978) excluded situations of passing or being passed by another car, and then obtained a rank correlation of .95 between SRR amp. and brake pressure. A detailed evaluation using cross-correlations of EDR and EMG data showed that the EDR was not a concomitant or consequence but an antecedent of braking. A comparison of EDRs and HR changes during steering yielded a much tighter covariation of the electrodermal system with the preparation of steering than for the cardiovascular system. Therefore, Helander concluded that EDR is a very sensitive variable with which to measure increases in task demand during driving.

In a study performed in Germany, Zeier (1979) continuously recorded SYR[282] together with frontalis EMG, HR, and information from brakes as well as from gear lever (or selector lever) from 12 male subjects driving a manual-gear or automatic car in permuted order with one week distance. Urine catecholamines, which were additionally recorded, were higher for driving a manual-gear car, as was NS.EDR freq. and HR, while no effects were found in EDL. Hence, in this study the frequency of nonspecific EDRs served as an indicator of general arousal (Sect. 3.2.1.1) rather than of specific emotional strain. Additionally, EDA and HR were also elevated in passengers (who

[281] Beckman Ag/AgCl electrodes and .1 N chloride concentration were used to record from the dorsal side of the hand with 12 $\mu A/cm^2$ constant current. For evaluation, psychophysiological responses were moved one sec earlier, in order to compensate for their time delay.

[282] AC recordings with a 5.25 Hz constant voltage (1 V) source between two Ag/AgCl electrodes filled with Hellige isotonic paste, attached according to Fig. 28 (Sect. 2.2.1.1) to the medial side of the left foot. EDA was recorded on tape with PCM electronics (Sect. 2.2.4.2). EDRs were classified into four groups: more than 10%; 8–10%; 5–7%; and 2–4% change with respect to EDL, forming arbitrary units that were averaged per min.

also took part as subjects in the study) while sitting in the manual-gear as compared to the automatic car, showing that even passive participation in the driving situation may cause considerable arousal.

As compared with its use in vehicle driving research, EDA has not been used much with *aircraft pilots*. Lindholm and Cheatham (1983) continuously recorded SCR amp.[283] and HR from six air force reserve officers while they performed a landing task at a simulator 10 times. Towards the end of the landing procedure, both measures showed a marked increase, which during practice became less steep in HR but not in EDA. Despite that result, which showed some superiority of electrodermal over cardiovascular variables with respect to persistent emotional strain, these authors decided not to record EDA in their subsequent studies with pilots.[284]

Kahabka, Oppelt, Rohmert, and Müller (1986) performed a field study with three subjects, an inexperienced and experienced pilot as well as an inexperienced passenger in a small plane. Monitoring of SCL,[285] HR, and EMG during five flight phases yielded differential effects on electrodermal and cardiovascular parameters: while HR and SCL were correlated in the inexperienced pilot, they were not in the experienced one, where HR reflected the difficulties of flight, thus indicating mental strain. SCL, which was also uncorrelated with HR, showed a steep decline in the passenger during flight, which was interpreted by the authors as being due to the specific indicator function of EDA for emotional strain.

While the use of EDA in traffic research has been in field studies, only a few studies that used EDA as an indicator of *job-related stress* have been performed at industrial work places. Rutenfranz and Wenzel (1958) investigated the dependence of skin impedance[286] on the load of physical strain at simulated work places. Data from three female subjects showed a marked decrease of SZL and an increase of the capacitative component (Sect. 2.1.5) after 15 min work at a punching machine as compared to an initial resting period. To further test the dependence of skin admittance on physical strain, Rutenfranz and Wenzel recorded EDA from one male subject repeatedly performing a bicycle ergometer task with different work load (see Footnote 286). In conditions of 10 mkp/sec performance and above, skin capacity steadily increased, starting 2.5 min after task onset, the gradient becoming steeper with increasing amount of strain, with no marked decreases during subsequent 10 min rest periods. The increase of skin capacitance was paralleled by a decrease of SZL, the gradients of which, however, showed no such clear-cut relationships to work load.

[283]Measured with Beckman Ag/AgCl electrodes and Beckman paste from palmar/dorsal sites with .5 V constant voltage. The highest SCR amp. within every 5-min section was evaluated.

[284]In fact, the visibility of EDA electrodes raises problems of compliance in pilots, which may explain some of its infrequent use in that field.

[285]Recorded with AC from the ankle, using the methodology of Faber (1983), see Footnote 288.

[286]Measured with 500 Hz, 1 kHz, and 10 kHz at constant voltage (1 V) (SYL transformed to SZL) using 3 by 4 cm V2A nets as electrodes at the backside of the lower legs while working at the punching machine, and at the inside lower arm during bicycle ergometer task (performed 10 times each with zero, 5, 10, 15, and 20 mkp/sec, and 6 times with 25 mkp/sec, with 7.5 min duration).

Schönpflug (1965) used an ergometric task to determine the dependence of SRL[287] on velocity and resistance of the ergometer in 40 male subjects. Using a counterbalanced within-subjects design with 32.0, 42.7, 64.0, and 96.0 m/min, combined with 2.85, 5.70, 8.60, and 11.45 kp resistance, a linear increase of SCL was found with an increasing subjective velocity rating, and a positively accelerated increase of SCL with an increase of subjective resistance ratings.

In an exploratory study performed with three female workers belonging to an assembly group in the electronics industry, Faber (1983) recorded SYL[288] and HR during several different subtasks. While HR increased with physical strain, an increase of SYL was found with increasing mental strain (resting pause – packing – soldering – assembling).

Rakov and Fadeev (1986) also got results showing that EDA may be used as an indicator for nonphysical work load. They measured SP[289] in 20 female workers in an electronics factory during rest and different production output as well as during testing phases of work. As compared to rest, only small increases in NS.SPR freq. appeared during manufacturing. However, a marked increase in spontaneous EDA was found during testing phases, which were reported as being especially high in emotional strain.

Strong support for differential influences of different kinds of strain (physical, mental, and emotional) on electrodermal and cardiovascular parameters comes from investigations at real and simulated office work places (Boucsein, 1991). Peters (1974) monitored HR, blood pressure, respiratory rate, frontalis EMG, skin temperature, and SR telemetrically in 11 female phonotypists. For each psychophysiological parameter, the different tasks to be performed by the subjects during their work were ranked with respect to the amount of changes induced by that particular task. While HR increase was highest during tasks with predominantly physical strain (e.g., changing paper), and lowest during the most automated task of typewriting, electrodermal changes mainly appeared during mental tasks (e.g., thinking or reading).[290] Thus, HR appeared more sensitive to physical strain, while mental (and/or emotional) strain was mainly reflected in EDA.

The present author's group performed several studies on psychophysiological stress reactions on system response times in human-computer interaction at simulated work places, using electrodermal and cardiovascular measures as dependent variables (for an overview, see Boucsein, 1987, 1989). In a pilot study, Schaefer, Kuhmann, Boucsein, and Alexander (1986) continuously recorded HR and SC, measured with standard

[287]Recorded as means of 30 sec intervals, and transformed exponentially with respect to resting values.

[288]Measured with a 10 Hz and .5 V constant voltage system, using dry electrodes made from silver-plated nylon tissue (3.2 cm^2 area), taken from palmar finger sites, and monitored telemetrically.

[289]Recorded with nonpolarizing electrodes palmar/dorsal from the left hand, amplified by an EEG coupler. The number of SPRs was individually related to productivity, in order to reduce interindividual differences.

[290]The highest amount of EDA was, however, recorded during speaking, which must be regarded as being mainly due to an artifact (Sect. 2.2.5.2).

methodology, during several hours of VDT work as well as during the interspersed resting pauses. 20 subjects (4 male, 16 female) performed five blocks of 50 error detection tasks each, while being subjected to intertask intervals of 2 or 8 sec on average, which were either constant or variable in length. Short system response times (i.e., higher work density) emerged as an increase in systolic blood pressure, as measured during the subsequent working pauses. This was interpreted by the authors as reflecting the greater amount of mental (and/or physical) strain. By contrast, NS.SCR freq. showed a tendency to be higher under the condition of long system response times.

Kuhmann, Boucsein, Schaefer, and Alexander (1987) performed another experiment with 68 subjects (46 male, 22 female) using a similar design. However, differing from the first study, trial blocks were of equal length, which implicated different number of tasks per block when varying intertask intervals (1,248 tasks in the 2 sec, and 624 in the 8 sec condition). Despite these differences in design, Kuhmann et al. (1987) replicated and extended the main results of Schaefer et al. (1986). Higher systolic blood pressure levels under short system response times and a significantly increased tonic EDA (NS.SCR freq. as well as SCL) under long system response times were found. The increase in EDA could not be attributed to artifacts (e.g., movements, or an increased number of tasks per time), since it appeared under the condition of lower work density. Therefore, these authors interpreted it as reflecting emotional strain caused by involuntary working pauses.

EDA reflecting emotional strain was directly shown in a third study performed by Kuhmann (1989), where 48 subjects (38 male, 10 female) performed the same kind of tasks with either 2, 4, 6, or 8 sec system response times, during three training trials and five working trials of 20 min each. Though no general effects of the physiological variables recorded reached significance, an averaging procedure of phasic EDA across tasks, using the end of the intertask interval as a trigger point, found that EDA (in arbitrary units) within the 2 sec interval was solely determined by EDA during the previous task, while a complex pattern appeared in the course of the 8 sec interval trials. Under this condition, during training as well as during the first working trial, EDA was higher during task performance than during the waiting interval for the next task. The reverse pattern appeared during the rest of the working trials, showing the development of an emotional tension when being interrupted in task performance by artificial temporal delays, for which EDA emerged as a sensitive psychophysiological measure.

The development of this kind of emotional strain as reflected by an increase of EDA could not be shown in short-term VDT work without time pressure (which was present in the studies reported above). Kuhmann, Schaefer, and Boucsein (1990) used system response times of 2 sec and 8 sec on average, being either constant or variable in length, in a within-subjects design with 24 subjects (18 male, 6 female) who performed error-detection tasks in four trial blocks of 10 min length each. While an increased cardiovascular activity within the 2 sec condition again showed up as an elevated HR, a variable pattern of NS.SRR freq., measured with standard methodology, appeared:

spontaneous EDA was lowest under the 2-sec-constant system response times at the beginning, but even lower under the 8-sec-variable condition at the end of the session. In summary, as suggested by Boucsein, Greif, and Wittekamp (1984b), it may be possible to use electrodermal parameters to get information on emotional strain at the work place that goes beyond the largely metabolically determined physical (and/or mental) strain as measured by the frequently used cardiovascular parameters.[291]

There have been also some attempts to use EDA in *shift-work* research. Fickova (1983) took SRL[292] recordings, in addition to HR and oral temperature, from 21 operators at the beginning, in the middle, and at the end of morning, afternoon, and night shifts. The highest SRLs appeared during the morning shift, while the lowest SRLs were observed during the late afternoon, which agrees with the results of Rutenfranz (1955). Intra-shift correlational analysis yielded significant covariations between HR and SRL in the afternoon and night shifts, while HR was correlated with body temperature in the morning shift.

Ottmann, Rutenfranz, Neidhart, and Boucsein (1987) recorded SC during five consecutive days and nights in the laboratory from 24 male subjects. These subjects performed 10 hour tasks daily at a simulated computer work place (vigilance tasks with additional STM strain). EDA was recorded intermittently (for technical reasons) with standard methodology, during work and leisure time as well as during sleep. Half of the subjects worked under either 80 dB or 50 dB white noise. EDA results with respect to shift paralleled those obtained with urine catecholamines, being higher when working during the day as compared to night work. Differential effects of noise emerged for both kinds of variables. Noise of 80 dB (as compared to the 50 dB control condition) led to an increase of adrenaline excretion for night workers, while a decrease of its excretion was observed under noise in day workers. No direct noise effects were obtained during work on EDA. However, aftereffects of noise on EDA appeared during the subsequent sleep period (Footnote 213, Sect. 3.2.1.3), yielding a significant increase in NS.SCR freq. after working under noise, together with increased excretion rates of noradrenaline. Thus, the use of EDA as a possibly sensitive indicator of long-lasting emotional strain in the field of human engineering can be highly recommended.

3.5.1.2 EDA in detection of deception

Detection of deception, which is more popularly known as "lie detection" or the "polygraph test," is an emotional topic, both in scientific and in practical use (Furedy, 1986). As Lykken (1981) reported, by the 1980s there were between one and four million American citizens being confronted with a lie detector test every year. By contrast,

[291] Goldstein and Shapiro (1988) have found that cardiovascular parameters are more sensitive to postural changes during laboratory performance tests (mental arithmetic and isometric handgrip) than EDA. However, there was also a marked increase in SCL as a transient reaction during standing up.

[292] Measured five times successively at 2-sec intervals with 10 mA constant current, from the first and second finger of the left hand, using 1 cm^2 aluminium electrodes.

in Europe there is little use of the technique, and in Germany, the supreme court decided several times not to accept the polygraph test as evidence, though acceptance was moderately advocated by several German psychologists (Wegner, 1981; Undeutsch, 1983, Steller, 1987).[293] Besides their use in forensic contexts, polygraph techniques are very commonly employed in the U.S. by federal and private security agencies (Lykken, 1981; Iacono & Patrick, 1988). Again, these applications for security checking or employment settings are rather dubiously regarded in Europe (Gudjonsson, 1986), though not much is known about the frequency of their use (Thornton, 1988).

The rationale behind using psychophysiological measures in detection of deception is that ANS reactivity on orienting and emotional stimuli is higher than on nonorienting and nonemotional stimulation (Sect. 3.1.1.1 & 3.2.2.1). Within this field, two different techniques are employed:

(1) The Control Question Test (CQT), where questions related to the relevant issue (e.g., a crime) are mixed with irrelevant ones (outside issues, but some of them also with a personal reference, e.g., the subject's first name or birth place). These control questions are individually constructed during a pretest interview (Reid & Inbau, 1977). Theoretically, an innocent subject should be more concerned about the control questions, whereas a guilty subject should react more strongly to the relevant questions (Raskin & Podlesny, 1979). The main problem with the CQT is that it is particularly prone to false-positive errors (Lykken, 1981; Gudjonsson, 1986).[294]

(2) The Guilty Knowledge Test (GKT), also labelled the "concealed information test", was developed by Lykken (1959b). The rationale of the GKT is that a guilty subject should react more strongly to certain facts that only he/she would recognize as relevant (e.g., details of a crime). These facts are embedded in a set of, for example, five alternatives that would seem equally plausible to an innocent subject. The subject has simply to answer "no" to each item, or repeat it. Sometimes, verbalization is not requested at all, which is the most suitable method with respect to avoiding artifacts from speech (Sect. 2.2.5.2). False positives can be practically excluded when using this method, since it is highly improbable that a subject consistently shows the highest reaction in the critical alternative within several subsequent items (i.e., normally in about 10 questions).[295]

[293] A comparison of the estimated numbers of polygraph examiners is provided by Barland (1988, Table 7.1).

[294] In pre- or postemployment screening situations, a slightly different question format is employed, labelled the "relevant control test" by Lykken (1981).

[295] Despite this superiority of the GKT over the CQT, the GKT requires details of the crime that are normally kept secret by the police; this is regarded as a major reason for its infrequent use by current polygraphers (Furedy & Heslegrave, 1988).

The advantages and disadvantages of both techniques were discussed in detail by Gudjonsson (1986) and Furedy (1986), and a recent international state-of-the-art summary has been provided by Ben-Shakar and Furedy (1990). According to Gudjonsson (1986), interrater as well as retest reliabilities of polygraph tests are rather high (from $r = .71$ to $r = .96$), while aspects of validity, obtained from laboratory and field studies, are less convincing: 68%–86% hits for guilty subjects, and 49%–76% for innocent subjects.[296] Ben-Shakhar, Lieblich, and Bar-Hillel (1982) critically reanalyzed seven field studies performed with the most widespread CQT by using parameters from the signal detection theory (estimates of d', the area under the ROC curve, and the payoff ratio). At first glance, polygraphers employing the CQT discriminated successfully between guilty and innocent suspects, since the smallest d' found was .77 standard deviations. However, a closer examination of the data yielded different criteria in that the polygraphers tend to value the detection of guilty suspects highly, even in the presence of a high false-positive risk. The authors therefore recommended using direct techniques only for screening out innocent suspects instead of detecting deceptive subjects.

Among the various physiological parameters used in the detection of deception, EDA[297] is regarded as the most sensitive, followed by the vasomotor response (Thackray & Orne, 1968; Barland & Raskin, 1973; Waid & Orne, 1981; Furedy & Heslegrave, 1988). One reason for their sensitivity is that clear-cut short-term changes are elicited in these variables by any change in stimulation, which increase as a function of stimulus significance (Sect. 3.1.1.1).[298] Furthermore, the EDR amp. is easy to evaluate in its quantitative aspects by visual inspection, not only by the experimenter but also by the subject (Fig. 50). This is often used to convince guilty subjects (and, unfortunately, at

[296]Taken from an U.S. Congress Office of Technology Assessment Report in 1983. Steller (1987) summarized results from laboratory studies, 11 performed with the CQT, and 7 with the GKT. According to these, the CQT correctly classified 15.8%–90% of the innocent subjects, with false positives ranging from 4.2%–31.6 %. On the other hand, five of the GKT studies made 100% correct classifications, the remaining two studies yielding 88% correct innocents and 12 % false positives. With respect to correct classifications of guilty subjects, both methods did not differ considerably (between 60% and 100% correct classifications).

[297]Raskin (1979, p. 597) recommended a time window for EDR evaluation between .5 sec after the beginning of the question and 5 sec after the subject's answer, and to express the amplitude in terms of mm of chart deflection, which had been regarded by him as producing more reliable results as compared to increases in μS.

[298]In addition to various proofs that EDA is the most sensitive variable for detection of deception in comparison with other measures (as found for example by Dawson, 1980, in his study described below comparing EDA with cardiovascular and respiratory variables), a comparison of an univariate with a multivariate statistical evaluation of CQT data performed by Kircher and Raskin (1988) also yielded superiority of SCR amp. over blood pressure, respiratory, and vasomotor responses in both methods of evaluation.

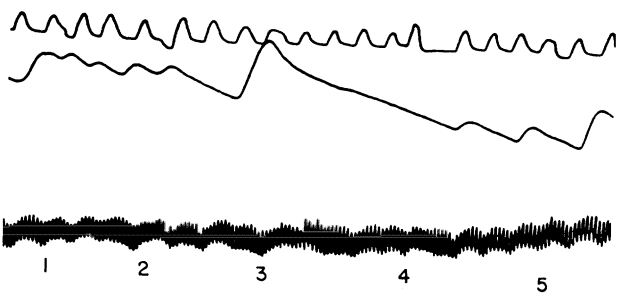

Figure 50. Polygraph recording during CQT performance. The third of five questions was the critical one. From all three parameters, respiration (upper), EDA (middle), and EKG (lower channel), EDR amp. showed the most clear-cut differentiation. From J. E. Reid & F. E. Inbau (1977), *Truth and deception: The polygraph ("lie detector") technique*, Fig. 287. Copyright ©1977 by the second author. Reprinted by permission.

times to convince false-positive innocents), leading to a breakdown and confession in about 25% of examinees (Lykken, 1981).[299]

In field polygraphy, in contrast to the recommendations given by Fowles et al. (1981) for EDA recording, there is a persisting predominance of constant current measurement. The main reason for this may be its lower amplifying requirements, reduced by a factor of 10 in comparison to the constant voltage technique (Sect. 2.6.2).

One of the most tricky problems in the use of ANS measures in the detection of deception is the possibility of so-called countermeasures, used by some subjects to appear nondeceptive during a polygraph examination (i.e., to "beat the polygraph"). These can be divided into three broad categories: mental, physical, and chemical/pharmaceutical (Gudjonsson, 1986). The most effective method is to voluntarily produce ANS reactions to the irrelevant items in order to reduce the discriminative power of the relevant ones. Attempts to augment one's reaction to the irrelevant stimuli can be made by eliciting somatomotor responses which produce artifacts in EDA and other polygraph variables, like deep inhalations, covert muscle contractions (e.g., thumbtacks in one's sock), or tonguebiting (Sect. 2.2.5.2). Lykken (1981) pointed to the fact that an innocent suspect cannot systematically self-stimulate on the control items of the GKT, since he/she does not know which alternatives are relevant. Thus, the small likelihood that

[299]The ease of detecting psychophysiological correlates of emotion-relevant thoughts by phasic EDA is also used in obscure religious practice, where so-called E-meters (simple SR monitoring devices) are used as a "scientific" tool to uncover hidden information in examinees.

false positives could be a major advantage of the GKT is not affected by countermeasures.

For the purpose of testing the lie detector's susceptibility to the use of countermeasures, the so-called mock crime paradigm has been used in laboratory experiments. In this kind of study, the experimental group receives the instruction to imagine having committed a specific crime, the details of which are given to them, while the control group receives instructions to imagine having been a witness to that particular crime. An additional payment is promised the subjects if they manage to "beat the polygraph" to provide an incentive that may increase the validity for real-life polygraphy.[300]

This technique has been used by Honts, Raskin, and Kircher (1987) to test the effect of physical countermeasures on the outcome of the CQT. Fifteen male and 15 female subjects were divided into three groups: two guilty groups that received tape-recorded instructions to enact a mock crime, and an innocent group that listened to a tape describing the crime but was instructed not to enact it. One of the guilty groups received training in the use of tongue biting and pressing the toes to the floor during the control questions. All subjects were given a field polygraph test by an experienced field examiner, and they were offered money if they could produce an innocent outcome. SC was recorded with standard methodology from the left hand, and amplitudes as well as duration of SCRs that started within a time window between .5 sec following the question and 5 sec after the subject's answer were evaluated. In addition, respiratory and vasomotor activity as well as blood pressure were taken as variables for polygraphy, and EMG recordings from gastrocnemius and temporalis muscles were recorded to detect physical countermeasures. As in standard field practice, each pair of control and relevant questions was assigned a score between –3 and +3 for each of the physiological systems, according to the largeness of the difference in the corresponding reactions; positive scores indicated that responses to control questions were stronger. Total summed scores exceeding +5 were considered innocent, those being lower than –5 were considered deceptive, the rest being regarded as inconclusive. In the innocent group, two false positives and one inconclusive subject appeared, while no false negatives and two inconclusives appeared in the guilty group without countermeasures. However, countermeasures completely distorted detection of deception, leading to seven false negatives (guilty subjects classified as innocent) and three remaining inconclusives. Comparison of these results with an earlier study performed by the Raskin group suggested that the countermeasures were dependent on the presence of high motivation, which is present in real-life polygraphy. Though EMG recordings proved to be useful as countermeasure detectors in 90% of the trained users, their effectiveness in the field has been regarded by the autors as possibly limited.

Bradley and Warfield (1984) used the mock crime paradigm to test the robustness of the GKT against innocent subjects having crime-relevant information. Forty sub-

[300]On the other hand, validity criteria of being guilty or innocent are not easy to obtain in field studies, since in the U.S. justice practice, false admissions of guilt are common to reduce the amount of penalty.

jects of both genders were randomly assigned to five groups: a guilty group, a control group which had no crime-relevant information, and three groups of innocent subjects that were given the same crime-relevant information with different instructions. These were either witness a murder, be an innocent suspect, or carry out innocent activities involving crime-relevant information. All groups except the last one were promised monetory reward if they were judged innocent. All subjects had to answer "no" to all items. SRRs, recorded with standard methodology (however, using 50 μA current) were obtained in mm deflection for each item of 10 sets of the GKT within 10 sec following the beginning of the question. The first item of each set served as a buffer item for ORs, and the other four items were scored as follows: if the response to the critical item was largest it received 2, if second largest 1, and zero for any other response magnitude. Guilty subjects scored significantly more guilty than members of every other group, showing that guilty knowledge is necessary but not sufficient for detection.

In another study with 40 male subjects, Bradley and Ainsworth (1984) tested the effects of alcohol taken during the commiting of a mock crime and during the polygraph test on the detection of deception. Eight subjects were innocent, while 32 subjects were told to be guilty of a mock crime and given appropriate information. Half of the guilty subjects were intoxicated with alcohol and half remained sober when committing the mock crime, and half of each group was intoxicated with alcohol for the polygraph test performed on the subsequent day, while the other half was not. All subjects were given both the CQT and the GKT. SRRs were recorded and scored for the GKT as by Bradley and Warfield (1984), while scores of +1, 0, or –1 were given in the CQT, depending on whether the SRR amp. to the control question was larger than, the same as, or smaller than the response to the crime-relevant question. The same kind of scoring was also performed for HR and respiration cycle time, and a composite score for all three dependent variables was formed. Using this score, 28 subjects were correctly classified in total by the CQT with 7 judgments remaining incorrect and 5 inconclusive. With the GKT, 38 subjects were correctly classified and 2 were incorrectly classified. In both tests, significantly better than chance classifications were obtained with EDA and HR but not with respiration. Intoxication with alcohol during commiting of the mock crime markedly reduced detectability by means of the composite scores as well as by the SRR in both tests, regardless of alcohol consumption during test state. The intoxicated subjects were more likely to be classified incorrectly as innocent or inconclusive in the GKT, and also appeared more innocent in the CQT. A simple explanation following an impairment of learning by alcohol intoxication did not hold, since an additional memory test did not yield appropriate differences. Unexpectedly, if the mock crime had been committed under intoxication, alcohol did not influence the EDA but it did influence HR deceleration.

Waid, Orne, Cook, and Orne (1981a) showed the effectiveness of a tranquilizer (400 mg meprobamate) in reducing detection of deception given 30 min prior to the GKT. Eleven male subjects took part in each of the following four groups: innocent; "guilty" with no medication; "guilty" with placebo; and "guilty" with meprobamate.

The latter two groups were told they would receive a tranquilizer that would help them to avoid detection. The question list consisted of 24 words, four in each of six semantic categories, one of the four being a word the guilty subjects had memorized before. While all innocent subjects, nine of the no-drug subjects, and eight subjects under placebo, were correctly classified by their SRR amp.,[301] under meprobamate, only three "guilty" subjects were classified correctly, leaving eight false negatives. The cardiovascular and respiratory measures did not discriminate between "guilty" and innocent subjects at all. Since the EDA results were not due to lack of electrodermal responsiveness among drug subjects – there were no differences among groups in the mean number of critical words that evoked a measureable EDR – the authors attributed their results as being due to the anxiety-reducing properties of meprobamate, for which EDA is a sensitive indicator (Sect. 3.4.3.1).

By contrast, Iacono, Boisvenu, and Fleming (1984b) could not find significant influences of either a tranquilizer (10 mg diazepam) or a stimulant (20 mg methylphenidate) on the validity of the GKT. Sixty male subjects were randomly assigned to three guilty groups receiving either one of these drugs or placebo, or to an innocent no-drug group. Subjects of the guilty groups watched a videotaped burglary of an apartment through the eyes of the thief, while the control group viewed the interior of another apartment. Drugs were applied prior to the 12 or 10 min videotaping. However, the drugs reached their peak action during the subsequently performed 10-item GKT. Subjects were promised a reward if they appeared innocent. SC was measured bilaterally with standard methodology during the test. SCR amp. were scored as the difference of the subject's maximum response SCL and the SCL prior to the item, and ranked within each question (except that the first alternative to each question served as a buffer item). If the SCR amp. following the critical alternative was the highest of the four responses, a score of 2 was assigned, while a 1 was scored if it was the second highest. An amplitude criterion (Sect. 2.3.1.2.3) of .03 μS was used, which had to be exceeded by the reaction to at least one alternative (other than the buffer item) to consider a question scorable. The actual guilt score was formed by summing up all 20 individual rank scores (10 from each hand's record), and by dividing this total by the number of scorable questions. Six subjects had to be excluded from the analysis because they did not meet the criterion of 10 or more scorable responses out of 20. Subjects scoring below 1 were classified innocent, and those with 1 or above were labelled guilty. The hit rates were 100% in the innocent group and 88% in the guilty groups, not considerably differing between drug conditions. However, the ability to remember critical facts recorded by a questionnaire correlated significantly ($r = .53$) with the likelihood of being found guilty, showing the susceptibility of the GKT to memory influences.

A more fundamental issue in detection of deception concerns the suggested psychophysiological mechanisms underlying the observed increase in EDR amp. follow-

[301] Recorded with Beckman Ag/AgCl electrodes thenar/hypothenar from the right hand with 3.8 $\mu A/cm^2$, using AC coupling with .3 sec time constant.

ing relevant as compared to irrelevant items. Several hypotheses have been discussed here: an increased arousal due to greater emotional involvement (Sect. 3.2.1.1 & 3.2.2.1); an acquisition of a conditioned EDR to details of the crime, which were connected with unconditioned fear stimuli (Sect. 3.1.2.1); and various cognitive influences on the EDR as outlined in Section 3.1.3 (Waid & Orne, 1981). Raskin (1979) discussed these differences in terms of OR and information processing (Sect. 3.1.1.1). He suggested that the introduction of irrelevant stimuli leads to a general habituation effect on EDRs to that particular kind of stimuli, and that only the crime-relevant stimuli have a special signal value for guilty subjects that elicit marked ORs in them. Comparing direct and indirect techniques in detection of deception, Raskin found some support in HR reaction patterns for his hypothesis that ORs would be predominantly in the CQT, while the GKT would be more likely to produce DRs. He assumed that a generally higher level of emotional arousal produced by indirect techniques was responsible for this, because of the overall more threatening and personal nature of the GKT questions.

However, the discussion of EDRs in response to CQT items within the concepts of OR and DR is obscured by the necessity to immediately respond verbally to the question (Sect. 3.1.1.2). To allow a separate measurement of EDRs to questions and answers, Dawson (1980) performed an experiment with two versions of the CQT: one in which subjects verbally responded immediately and another in which they waited 8 sec before giving their verbal answer. Twenty-four student actors of both genders, trained in using personal memories of sensory experiences in order to recreate emotional states, were randomly assigned to a "guilty" and an "innocent" group; all of them were promised doubling of their payment if they managed to appear innocent in the polygraph test. After receiving the instructions (to imagine having stolen money versus to merely wait for the assistant to return), subjects were given each of the two forms of the GKT twice in counterbalanced order. Quantitative analyses of the mean SRR magnitudes (Sect. 2.3.4.2)[302] yielded significantly larger EDRs to the relevant questions than to the control questions for the "guilty" group, while the reverse was true for the "innocent" group for both the response in the immediate-answer CQT and the OR to the question in the delayed-answer CQT. No such interaction could be found for the SRR following the verbal response in the delayed-answer CQT. Thus, the SRR magnitudes were more likely to be indicators of differential ORs to relevant versus irrelevant stimuli than influenced by verbal activities of the subjects.

Going beyond an interpretation of mere differential ORs (or DRs), Pennebaker and Chew (1985) hypothesized the suggested connection between EDA and BIS (Sect. 3.2.1.2) as a neurophysiological basis for the electrodermal detection of deception.

[302]Recorded with 2.5 x 2.5 cm stainless steel electrodes from the palmar fingertips of the left hand, using 10 μA constant current. For the CQT form with immediate response, the largest decrease in SR which occurred between 1 sec following the question onset and 5 sec following the verbal answer was measured. For the delayed-response CQT form, a time window between 1 sec following the question onset and 1 sec following completion of the question was used for the question SRR, while the largest deflection within 1 and 5 sec following the verbal answer was taken as the answer SRR.

Thirty predominantly female subjects were instructed to deceive the experimenter in a GKT on two separate occasions. They selected one of five code words printed on cards but had to answer "no" if asked for any word during the test. In the second GKT run, subjects were assigned either to a group ($N = 20$) being observed by the experimenter, who tried to find out by viewing the subject's behavior what card she/he had selected, or to a control group ($N = 10$). Mean SCL[303] did not differentiate truth from lie words in the first GKT, while there was a significantly higher SCL when subjects were observed, in addition to a marked increase of SCL from 2 to 4 sec after deception (as compared to truth items) under both experimental conditions. For the 20 subjects in the observe condition, changes in eye movement and facial expression (Sect. 3.2.2.1) were continuously coded during the second GKT, and were summed during 2-sec intervals following the questions. During seconds 2–4 and 4–6, the mean number of behavioral measures showed the reverse pattern than SCL, indicating a behavior decrease following deceptive responses where the increase of SCL was most pronounced. Thus, an increase in EDA during deception may be at least partly due to its specific indicator function for behavioral inhibition as suggested by Fowles (1980), a theoretical line which requires further research.

Possible individual differences in electrodermal lability (Sect. 3.3.2.2) should also be considered when using EDA as a dependent measure in the detection of deception. Waid and Orne (1980) reported appropriate results from two experiments, using the GKT in the first one with 28 male subjects, and the CQT in the second one with 30 subjects. In both studies, the deception of critical code words was used, while SR was recorded in the first, and SC was measured in the second (both times with standard methodology with the use of KY-gel). For each subject, NS.EDR freq. was scored in the ISIs from 4.5 sec following each word until the end of the ISI. Both studies confirmed a less frequent detection of deception by subjects showing lower spontaneous rates of EDRs (i.e., electrodermal stabiles) as compared to electrodermal labiles. Furthermore, among truthful subjects, those being more electrodermally labile were falsely detected as deceptive on more questions than the stables.

In another study, Waid, Wilson, and Orne (1981b) confirmed the effect of electrodermal lability on the detection of deception not only for electrodermal but also for cardiovascular and respiratory measures. Seventy-four male subjects were classified, using the median of their NS.SCR freq.[304] during a 3-min baseline period, into labiles and stabiles, and randomly assigned to the "guilty" or "innocent" condition of a code word deception test. They were subjected to both a GKT and a professional CQT one week later, while measuring EDRs, HR, blood pressure, and respiratory changes.

[303]Recorded as SRL with 10 mA constant current using Beckman Ag/AgCl electrodes from palmar finger sites, converted into SC units. SCL data was reported from 2 sec after the question (where the subject answered "no") to 14 sec after the question.

[304]Recorded with standard methodology, however, using KY-gel and .74 V constant voltage. Amplitude criterion = .05 μS. For detection of deception, amplitude criterion was lowered to .025 μS. The time windows began at 1.5 sec, ending at 9 sec for the CQT and at 5 sec for the GKT, after stimulus onset.

Again, deception by electrodermal stabile subjects was detected less frequently than was deception by labile subjects, and the rate of false positives among truthful subjects was greater for electrodermal labiles. Although accuracy of detection was highest with the EDR, the effects of electrodermal lability on detection of deception were similar for the other ANS measures. Whether these individual differences should be interpreted in terms of (emotional) hyperreactivity, or as being due to differences in attention or conditionability, remains to be investigated (Sect. 3.3.2.2).

In summary, though the use of EDA as a major variable in the detection of deception is well established, various methodological problems with the technique in general remain unresolved. The following critique of "lie detection" has been provided by Furedy (1986, 1987) from a standpoint of scientific psychophysiology:

(1) They are highly susceptible to examiner's influence (e.g., expectations or various examiner-examinee interactions), since blind procedures have been used only in a few research projects but are not part of practical polygraphy.

(2) The method of scoring is subjective, at least to some degree. The differences between responses to relevant and irrelevant stimuli are normally not specified in physical units (such as Ω for SRR amp.), but in qualitative categories (e.g., scoring 1, 2, or 3 for slight, clear, and marked differences, respectively, or using a magnitude ranging from +3 to –3 for each response channel).

(3) The term "control" as used in the CQT does not meet its standard, scientific sense. Control questions could only be accepted as an experimental control if they were comparable to the critical questions in all respects except for the process of deception that is under investigation.

(4) Even the more sophisticated GKT appears not to detect deception itself. Rather, there will be a differentially greater OR associated with the identification of the relevant alternative, because of its greater significance. Indeed, a stimulus might gain significance simply by experimental manipulations (Sect. 3.1.1.1).

However, Furedy, Davis, and Gurevich (1988) could unequivocally demonstrate the psychophysiological deception phenomenon in a carefully controlled experimental setting. Sixteen subjects of each gender were given two lists of 10 questions, to one of which they were to respond honestly, while to the other, deceptive but plausible answers were requested. For interrogation, the items of both lists were randomly allocated to a 20-question list, the first and second 10 items of which were presented under immediate and delayed (10 sec waiting) response conditions, respectively. The major finding was that SCRs[305] in the interval immediately following the question were

[305]Recorded as the highest deflection from prestimulus level with standard methodology (using Beckman NaCl paste) from the palmar finger sites of the left hand (previously cleaned with soap and water), within 1–5 sec following question onset. SCR amp. were expressed in mm (using .5 mm as amplitude criterion) and converted to μS response magnitudes.

significantly larger when the question was answered deceptively, which could not be attributed to other factors than to the difference between the deceptive and honest conditions. Though this is an encouraging experimental proof of the possibility of using EDA in the detection of deception as a psychological process, further theoretical and methodological clarifications of this phenomenon are needed, and attempts have to be made to avoid misuses of the so-called lie detection in practice.

3.5.2 EDA in medicine

The clinical use of EDA is not restricted to psychopathology (Chapter 3.4). In addition, several medical disciplines such as dermatology (Sect. 3.5.2.1) and neurology (Sect. 3.5.2.2) make specific use of electrodermal parameters for diagnostic purposes as well as in therapy evaluation. Furthermore, in various illnesses that are often classified as psychosomatic disorders, EDA has been used not only in diagnostics and therapy but also to establish causal models for the psychophysiological nature of these disorders (Sect. 3.5.2.3).

3.5.2.1 EDA in dermatology

Diseases of the skin should be a natural field of application for recording of electrodermal phenomena. Although standard readings in dermatology discuss EDA (e.g., Keller, 1963; Jarrett, 1978; Schliack & Schiffter, 1979; Thiele, 1981a, b), clinical dermatology continues to prefer the use of more qualitative measurements of sweat rather than quantitative EDA recordings (Sect. 2.4.2.2).

Pathological changes in the skin not only influence its resistive or conductive properties but also skin capacity. This was shown by Gougerot (1947) using AC measurement of EDA.[306] In subjects with normal skin, SZLs always exceeded 200 Ω, and phase angles were greater than 45^0, while SZLs in patients with active *eczema* were as low as 90 Ω, and phase angles decreased to 26^0. Patients with psoriasis showed lower SZLs at affected dry sites, while SZLs at unaffected skin sites were abnormally elevated. A possible explanation for the latter result could be an increased rate of mitosis in the stratum germinativum (Sect. 1.2.1.1) as found by Wright (1983) in psoriatic patients. Edelberg (1971), summarizing several studies on EDA and skin pathology, pointed to the specific value of AC measurements for differential diagnoses of epidermal changes, though the findings are also dependent on electrode size and frequency of the applied current, which both influence capacitative properties of the skin (Sect.1.4.3.2).

Salter (1979) also recommended AC measurement of EDA in order to quantify the amount of healing in dermatological illnesses, reflecting mainly the degree of corneal hydration (Sect. 1.4.2.3) which may be a critical factor in various diseases. As compared to chemical measurement procedures, recording of EDA has the advantage of being truly noninvasive, quick and painlessly applied, and quantitatively evaluated. Be-

[306] Recorded with 4 kHz AC using lead electrodes.

sides the hitherto predominantly used tonic measures in dermatology, phasic changes in capacitive properties of skin, which seem to be of minor importance in normal subjects, (Sect. 2.3.1.2.4) could be of considerable value in deliberately following up epidermal changes during the course of skin diseases.

Cambrai, Clar, Grosshans, and Altermatt (1979) used skin impedance recordings[307] to quantitatively compare the healing process in *psoriasis* in patients treated with dioxyanthranol, difluprednate, and photochemotherapy associated with 8-methoxypsoralen. Each of these treatments was applied to four subjects over 13 days. As compared to healthy skin sites, SZL and phi were markedly reduced at affected sites up to measurement frequencies of 10 kHz, while these differences disappeared with higher frequencies. Values returned to normal within 1–2 days with difluprednate, and within 5–10 days with dioxythranol, but took considerably longer with photochemotherapy. Changes in skin impedance were in good accordance with clinical data, and exemplified the usefulness of electrodermal AC recording in quantifying pharmacocinetic effects during treatment of skin diseases.

An application of EDA that is in close relationship to the phenomenon itself is its use in the quantitative diagnosis and therapy of *hyperhidrosis*. Apart from excessive sweat secretion being a diagnostic indicator of specific neurological damage (Sect. 3.5.2.2), hyperhidrosis as a systemic illness may appear mainly on palms. Persons who react under emotional strain with an increased sweat secretion notice visible palmar sweating. The unpleasant feelings, produced by this, in turn, elicits emotional excitement, thus forming a positive feedback loop for further sweating.

Since medical treatment of hyperhidrosis remains unsatisfactory (e.g., application of anticholinergics, bathing with salt or metal ingredients, or electrophysical therapy), the use of EDA biofeedback training should be regarded as more promising (Sect. 3.1.2.2). This was successfully used by Miller and Coger (1979) in 33 patients (14 male, 19 female, ranging in age from 15 to 61 years) with dishydrotic eczema. This particular disease is characterized by increased epidermal hydration (i.e., intercellular and/or intracellular edema) Twenty-two of the subjects were trained for decreased SYL, while 11 were trained to increase their SYL by means of optical EDA biofeedback.[308] Subjects trained for 15 min twice a day for a period of two weeks were seen in the laboratory before and after their training for a 10 min rest during which EDA was recorded. Subjects that were trained to decrease their SYL showed significantly lower SYLs during their second laboratory recording, while the increase group showed no change. These results were paralleled by a significant decrease of state anxiety in the SYL-decrease group and

[307]Measured with liquid electrolytes made from polyethylene glycol with .9 % NaCl (Sect. 2.2.6.3), the active electrode made from platinum, and the reference electrode made from Ag/AgCl. A frequency range of 5 Hz to 500 kHz was used.

[308]EDA was recorded in the laboratory with 100 Hz constant current less than 8 μA, using .5 cm^2 gold cup electrodes with standard NaCl Unibase paste, from alcohol-scrubbed left-hand sites. In the field part of the study, finger tip electrodes, which consisted of silver fiber applied to velcro pads, were used for EDA biofeedback. SZLs were transformed to log SYLs for evaluation.

by no change of state anxiety in the SYL-increase group, respectively. Furthermore, the SYL decrease-trained group showed overall improvement in their disease, while the others became slightly worse.[309]

EDA has also proved to be a valid indicator for the severeness of *epidermal damage* produced by using stripping techniques (Sect. 1.4.2.1) or skin drilling (Sect. 2.2.1.2). Edelberg (1971), in summarizing human as well as animal results, reported that puncturing the epidermis reduced its resistance to 10–20% of its original value. A similar decrease was caused by skin drilling in rats, taking three days for half recovery, and five days to recover entirely. Sanding human skin to the point of bleeding caused the surface negative SP to drop from –10 to –40 mV, requiring an average of 43 hours to recover. Lykken (1971) found only very little recovery of SP during the first 100 hours after performing 30 strips with Scotch tape at the lateral surface of the upper arm in two subjects. SP then returned rapidly between the fourth and the sixth days, while the SR in the parallel branch as well as polarization capacity (see Fig. 18, Sect. 1.4.3.3), measured with pulsed DC (Sect. 2.5.3.2), recovered more slowly. Pinkus (1952) found numerous epidermal keratinocytes in all stages of mitosis (Sect. 1.2.1.1) 72 hours after skin stripping.[310] Therefore, it can be suggested that removing the stratum corneum removes an electrical barrier, thus shunting the sweat gland potential (Sect. 1.4.2.3). This is in agreement with results of Takagi and Nakayama (1959), who investigated the effect of removal of epidermis by inducing a blistering agent on the left little finger, after which the epidermis was taken off by scissors, leaving only the stratum Malpighii (Table 2, Sect. 1.2.1.1). In doing this, the negative SPR component (Sect. 1.4.3.2) totally disappeared but recovered after two days. Thus, the measurement of SP may be used to quantitatively determine the process of wound healing (Foulds & Barker, 1983).

Woodrough, Canti, and Watson (1975) used the recording of SP[311] to quantify differences between affected and normal skin sites on the face in 36 patients with basal cell *carcinoma*, and in 19 subjects with inflammatory lesions serving as a control group. Using the contralateral side of the face as reference (which is not fully justified; see Section 3.1.4.2), they found a significant average increase of 14.4 mV in carcinoma-affected as compared to healthy skin, while the appropriate difference for inflammatory lesions failed to reach significance. However, the standard deviations were too large to allow differential diagnoses of skin diseases with the single use of SP recordings.

[309] A single case study of successful EDA biofeedback was published by Moan (1979), who trained a 28-year-old female urticaria patient for eight weeks with acoustic/optic EDA biofeedback combined with a relaxation training. Her mean SCL could be reduced from 12 to 7 μS (during the relaxation even to 4 μS), and the skin disease disappeared. An eight month follow-up yielded no recidivism.

[310] Klaschka (1979) reported an increase in the mitotic index by a factor of 20 two days after 30 using Scotch tape strips, returning slowly to baseline from the third day on.

[311] Measured with a liquid electrode made from a syringe, combining Ag/AgCl with physiological saline in sodium methylcellulose; the inactive electrode placed in the mouth.

Kiss, Horvath, and Hajdu (1975), used SZL[312] for determining differences between affected sites in 92 patients having malignant epidermoids and unaffected sites of these patients. They took recordings from 254 patients with benign skin deformations as an additional control, finding that SZLs in 86.9% of the malignant tumor sites were lower as compared to control sites, thus proposing the measurement of skin impedance as a diagnostic tool for skin tumors.

The validity of EDA for preliminary *cystic fibrosis* screening has been demonstrated by Williamson et al. (1985), who compared 37 established cystic fibrosis patients with 45 asthmatic patients and 10 normal controls. SP as well as SC were recorded with standard methodology[313] from palmar finger sites during voluntarily produced fast deep breaths, which served as physiological stimuli for elicitating EDRs (Sect. 2.2.5.2). The deep breaths were requested until five clear positive SPRs were obtained. Twelve parameters for endosomatic and exosomatic EDA were obtained, including baseline values and the response to a balloon test. All these electrodermal measures were highly significant in distinguishing between the cystic fibrosis patients and the normal or asthmatic control groups. Discriminant analysis using the two best EDA measures (the mean of five preresponse SPLs, and the mean of five preresponse SCLs for each subject) for assignment of experimental group membership yielded 92.7% correct classification of actual group membership. However, both SP and SC recordings were necessary to obtain such accuracy, since subject reclassifications by discriminant analysis using only the six potential or the six conductance measures lowered the percentage of correct classifications to 86% for SP and 77% for SC.

Skin damage caused by buffered and unbuffered solutions of alkali and sodium phosphate were investigated by means of skin impedance by Malten and Thiele (1973). They found that SZL could be abolished almost completely by a 15–30 min exposure to NaOH (pH 12) solution, whereas exposure up to 60 min to NaOH (pH 10) solution hardly influenced SZL. Similar effects were found with appropriate concentrations of sodium phosphate solutions. Furthermore, the changes produced by the application of pH 10 solutions on five subsequent days decreased, quantitatively showing adaptation of skin to less severe chemical damage, in contrast to severe damage.

Kiss (1979) quantitatively investigated the relationship between the concentration of NaOH, applied to the skin on filter paper for 30 min, and the SZL.[314] Up to concentrations of 1/40-n NaOH, there was only a slight decrease in SZL, which became rapid when the concentration was increased to 1/10-n NaOH (from 19 to 8 kΩ). With a further increase of NaOH concentration, the SZL showed no considerable changes,

[312]Measured with 1.6 kHz, using 3 mm steel electrodes together with 4 mm diameter filter paper, soaked with .9 % NaCl solution.

[313]From isopropyl alcohol cleaned sites. The electrolyte for SP consisted of .05 molar NaCl in a mixture of 50% Unibase and 50% polyethylene glycol, to keep the stratum corneum minimally hydrated (Sect. 2.2.6.1).

[314]Recorded with 1.6 kHz and 1 mA current density, using metal electrodes of 6 mm diameter, and a 5 mm diameter filter paper soaked with .9% NaCl solution, from the inner aspect of the forearm.

which may have been due to a severe damage of the epidermal barrier at that time so that ions could pass without impedance (Sect. 1.3.4.2). With another 10 subjects, Kiss continuously registered SZL during the application of 1/40-n NaOH. He found an immediate decrease of SZL, reaching its new level about 3 min after application. Hence, AC measurements of EDA may be of great value for quantifying chemical damage of the skin.

The use of EDA in dermatology also includes the investigation of effects of *cosmetics* on the skin. In this field of study, the Yamamoto group in Japan developed standardized paradigms using AC measurement (for description of methodology, see Sect. 2.5.3.1). Using the generalized electrical model of skin as depicted in Figure 18 (Sect. 1.4.3.3), these authors quantitatively determined the conductance values G_2 and G, being equivalent to the parallel resistances R_2 and R, as well as the capacity C, while neglecting the series resistance R_1. By matrix transformation (cf. Yamamoto et al., 1978. p. 625f.), three parameters were obtained: $\bar{C}_N$ as a quantity related to the dielectric constant due to polarization; $\bar{g}_N$ as the part of conductivity due to polarization; and $\bar{G}_N$ as the part of conductivity due to ionic conduction. $\bar{G}_N$ decreases if the ion conduction in the epidermal stratum corneum decreases, while an increase of $\bar{C}_N$ reflects an increase in the dielectric constant in the corneum. The changes in these parameters were depicted in a plane coordinate system with angles of 120^0 between each of the three axes, visualized by triangles (Fig. 51).

Figure 51 depicts the mean changes in these parameters obtained by Yamamoto et al. (1978) at forearm sites of three subjects under controlled conditions (with the subject's forearm in a climate chamber), after the application of a 50% solution of sodium-pyrrolidine-carboxylic acid (a natural moisturizing factor in the skin), which is used in cosmetics to increase skin moisture. Thirty min after its application, the corneal ion conductivity $\bar{G}_N$ was markedly increased, with a further increase during the next 2.5 hours. The polarization conductivity $\bar{g}_N$ as well as the dielectric constant $\bar{C}_N$ were also increased after 30 min, showing a decrease in the subsequent recording period. On the other hand, applications of emulsions of the type oil-in-water, or water-in-oil, yielded only small changes in these parameters. Though the mathematical rationale of their measurement technique looks complicated, the Yamamoto group provided a method of using electrodermal AC recording in dermatology that goes far beyond the usual conductance-plus-phase-angle measures, by showing a close relationship to moisture-related bioelectric changes in skin.

3.5.2.2 EDA and neurological disorders

Electrodermal as well as sweat secretion measures are primarily used in neurology to draw inferences about the type and extent of damage in the central and peripheral nerve systems. Their particular usefulness is in the slight differences in sensory and sudorisecretory dermatomes that provides additional information (see Table 3, Sect. 1.3.2.1); they also have the advantage of greater objectivity, since sweat secretion can

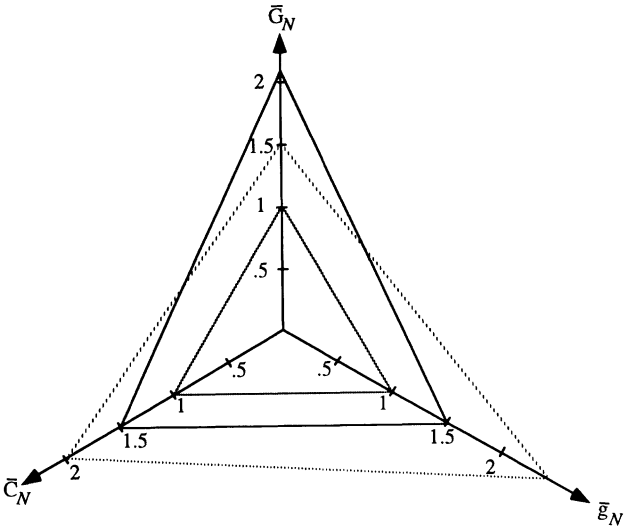

Figure 51. Changes in the dielectric constant $\bar{C}_N$ the ionic conductivity $\bar{G}_N$, and the conductivity of the polarized material $\bar{g}_N$ from baseline (inner triangle) over 30 min (solid line) until 3 hours (dotted line) after application of 50% sodium-pyrrolidine-carbonate solution. Redrawn from Y. Yamamoto, T. Yamamoto, S. Ohta, T. Uehara, S. Tahara, & Y. Ishizuka (1978), The measurement principle for evaluating the performance of drugs and cosmetic by skin impedance. *Medical and Biological Engineering and Computing, 16,* Fig. 7, p. 629. Copyright ©1978 by Peter Peregrinus. Used by permission of the publisher.

hardly be voluntarily influenced (Schliack & Schiffter, 1979). Typical fields of application are the diagnosis of lesions of spinal nerve roots, of the sympathetic trunk, or of peripheral nerves.

Riley and Richter (1975) mapped SRL[315] for 20 patients who had neck or upper extremity *pain* without clinical or radiographic evidence of spinal cord or nerve root irritation. They showed that areas of low SRL corresponded well to areas of subjective pain, thus providing some evidence for an unknown degenerative lesion process within the CNS underlying the reported pain in these patients.

Cronin and Kirsner (1982) recorded SP[316] from healthy skin areas in comparison to areas in which the sympathetic innervation blockade was well diagnosed in 60 patients.

[315]Recorded with an active roller electrode without paste, and an EKG electrode with hypertonic paste as reference at the leg or arm, by use of a dermohmeter.

[316]Using pre-gelled Ag/AgCl electrodes, the indifferent electrode attached to the wrist or ankle.

Their measure was a quotient of SPR integrals over 12 sec following a "supramaximal" stimulation, taken from normal as compared to abnormal innervated skin sites. They reported that this measure was in agreement with case histories and an adequate sympathetic block.[317]

Egyed, Eory, Veres, and Manninger (1980) compared the sensory map, the sweating map (by ninhydrine test), and the SRL map[318] in 47 patients with *injuries* of the median nerve, in 33 patients with ulnaris nerve injuries, and in 19 patients having both nerves damaged. SRL was found to be significantly higher in the areas of sensory loss than in corresponding normal skin surfaces, yielding the advantage of better quantifiability as compared to the coulorimetric ninhydrine test. Wilson (1985) used measures of SRL[319] for preoperative assessment of hand injuries and to monitor the recovery of nerve function in several cases, finding a good concordance between measures of tactile sensitivity and SRL during the healing process.

The usability of SPRs for differential diagnosis in the peripheral nervous system has been demonstrated by Shahani, Halperin, Boulu, and Cohen (1984), who recorded skin potentials[320] from 33 patients with peripheral neuropathies (aged 24–79 years), as well as from 30 normal controls (aged 13–62 years) during deep breaths and electrical stimulation. In 16 of the patients, no SPRs could be obtained. Correlations with clinical, pathological, and EMG observations showed that the SPR was usually absent in axonal neuropathies but present in demyelinating disorders.

Recording of EDA was performed in several studies by Schuri and von Cramon (1979, 1980, 1981, 1982) to establish an objective measure of vigilance loss in patients with CNS damage. In their 1979 study, an attempt was made to measure differences in reactions to meaningful versus meaningless acoustic stimulation in eight severely poisoned patients in a *coma*. The patients exhibited slightly though not significantly more SRRs[321] to their first names repeatedly being spoken forward with 90 dB intensity as compared to their names spoken backwards. In 27 patients with different levels of coma due to drug overdose, Schuri and von Cramon (1980) recorded changes in HR, finger

[317]The usability of EDA for the diagnosis of peripheral sympathetic nerve functioning was also demonstrated by Lidberg and Wallin (1981), who recorded sympathetic cutaneous nerve action potentials in the median nerve of five healthy subjects, while palmar SRRs were measured in parallel, following sudden unexpected loud noises. SP was recorded with Beckman Ag/AgCl electrodes with a contact area of 5 mm^2, using Cambridge Medical Instruments gel, 5 μA current density, and .3 sec time constant. One electrode was placed as close as possible to the median nerve innervation zone, the other one 3 cm away. The mean correlation between the amplitude of the sympathetic nerve burst and the SRR amp. was .68 (with a range from .47 to .90).

[318]Recorded with 3 mm^2 electrodes from skin washed with soap and water, followed by rubbing with alcohol.

[319]Recorded with lead coated stainless steel electrodes by the use of a digital ohmmeter.

[320]Recorded with 10 mm diameter stainless steel electrodes and commercial electrode paste from palmar vs. dorsal hand as well as foot sites, from anterior vs. posterior surfaces of the upper arm, and from patella vs. popliteal fossa sites.

[321]Recorded with standard methodology, using Hellige paste; time window 1–5 sec after stimulus onset; amplitude criterion = 500 Ω.

pulse amplitude, and SRRs measured as in the 1979 study (except using an amplitude criterion of 1 kΩ) following optic, acoustic, tactile, and electrical stimulation. HR was closely related to behavioral changes only in the more severe cases, while finger pulse invariably accompanied behavioral reactions at all coma levels. Sensitivity to stimulation and correlation with behavioral reactivity was lowest for SRRs (evaluated with a dichotomous criterion response versus no response).

In their 1981 study, Schuri and von Cramon again used speaking of the name forward and backward to differentiate SCRs (recorded with standard methodology) to meaningful versus meaningless stimulation in 16 neurological patients (8 with cerebral vascular diseases, and 8 comatose patients), compared with a control group of 16 healthy persons. Only in the most severely impaired patients, no differences were found between the two stimulus conditions, as had been the case in the 1979 study. In less severe cases, significantly larger SCR amp. to the actual names of the patients were found as compared to their names spoken backwards; however, this occurred only during the first two of ten trials. In normal controls, the differences persisted throughout the trials. Habituation appeared as an overall effect in all groups.

The dependence of appropriate results on the amplitude criterion used (Sect. 2.3.1. 2.3) was demonstrated by Schuri and von Cramon (1982) in a study with 18 neurological patients with severely disturbed vigilance and 18 healthy controls using a similar design. When SCR amp. below .025 μS were excluded, patients showed significantly less SCRs than controls, and five patients were complete nonresponders (Sect. 3.4.2.2). Lowering the amplitude criterion to .005 μS abolished the significance of the group difference, with only one patient remaining nonresponsive.

The usability of spontaneous and/or elicited SRRs as prognostic indicators for survivial in comatose patients has been shown by Bjornaes, Smith-Meyer, Valen, Kristiansen, and Ursin (1977). From a total of 40 patients, electrodermal recording was possible with 22, but only 15 met the criterion of showing any SRR[322] either spontaneously or following an electric shock. Out of these patients, all seven below 50 years of age survived the next 1.5 years; from the above-50 group, five survived, while all patients that had not shown EDA, except one, died during that period. However, Bjornaes et al. (1977) failed to establish electrodermal conditioning using a tone-shock pairing.

EDA has also been used as a correlate of emotional changes in patients with CNS lesions. Zoccolotti, Scabini, and Violani (1982) observed significant differences between SCR amp.[323] following emotional (e.g., sexual stimulation) and those following neutral (e.g., landscape) slides in 16 patients with left unilateral *brain damage*. These differences did not appear in 16 right unilateral brain-damaged patients, thus confirming

[322]Recorded with 9 mm diameter Ag/AgCl electrodes filled with commercial electrode paste from palmar/dorsal sites previously washed with acetone. When laterality of the damage was known, recording was performed at the ipsilateral hand.

[323]Recorded with 10 mm2 gold-plated electrodes from palmar finger sites. SCR amp. was calculated as the square root of the difference between previous SCL and the maximum SCL within 5 sec following stimulus onset.

results of Morrow, Vrtunski, Kim, and Boller (1981) that were obtained with a similar setting using 14 patients with damage in either hemisphere. Zoccolotti et al. (1982) interpreted these results as being in accordance with the right hemisphere's role in the organization of adequate emotional behavior (Sect. 3.1.4.2), and claimed their experimental setting to be useful to objectively state emotional indifference in right brain damaged neurological patients.[324]

Oscar-Berman and Gade (1979) observed the course of habituation of the electrodermal OR to 20 buzzer tones (100 dB, 1 sec) in 10 aphasic patients, 8 Korsakoff patients, 15 parkinsonians, 7 Huntington's chorea patients, and 18 normal controls. SCR amp.[325] following the first stimulus, a regression measure of habituation rate (Sect. 3.1.2.1), and the NS.SCR freq. as well as the SCL during an initial 10 min rest period were evaluated. There were no significant differences in measures of EDL, but patients with Korsakoff's and Huntington's disease were significantly less responsive than normals and aphasics on the first stimulus presentation, and patients with Huntington's disease were also less responsive than Korsakoff patients. These results, which were paralleled by those obtained from the habituation rate, supported the view of Stern and Janes (1973) that no generalized changes in ANS reactivity are to be expected following brain lesions. Instead, differential effects are likely to appear to be related to the specificity of the particular damage.

Such a group of neurological patients with specific relation to central origins of EDA are the *parkinsonians*. That particular neurological disorder is characterized by the degeneration of dopaminergic neurons connecting the compact part of the substantia nigra to the striatal part of the basal ganglia, causing a dopamine deficit in the latter. Besides the well-known motor symptoms (rigor, tremor, and hypokinesia), cognitive deficits were also observed in those patients (Canavan, Passingham, Marsden, Quinn, Wyke, and Polkey, 1989), which may be due to the basal ganglia's role in providing information on operant conditioning for frontal cortical areas (see Fig. 48; Sect. 3.2.1.2). Since the basal ganglia are also suggested to be the source of preparatory EDRs (i.e., "EDA 2," see Fig. 6; Sect. 1.3.4.1), this kind of EDA should be impaired together with cognitive deficits in parkinsonians, while electrodermal OR and habituation should not. The latter ones are more likely to be controlled by amygdaloid and hippocampal structures (i.e., "EDA 1," see Fig. 6).

An attempt to show this hypothetical connection between cognitive deficits in parkinsonians and specific electrodermal parameters has been performed in the present author's laboratory (Valentin, 1990). Fifteen parkinsonians were contrasted with 15 age-matched controls and 15 young, healthy subjects. As predicted, SCR amp. as recorded with standard methodology during an habituation series of 20 tones (1 kHz, 60 dB)

[324]Based on the results of an experiment with 20 healthy subjects, Tranel et al. (1985) advocated the usability of EDRs in the detection of the ability to discriminate between familiar and unfamiliar faces of prosopagnosic patients, who have lost the ability to recognize faces.

[325]Recorded as SR with 10 μA constant current unipolarly (thumb-upper arm), using 15 mm diameter Ag/AgCl electrodes, transformed to log SC values. Amplitude criterion for NS.SCRs = .003 log μS

yielded no differences between patients and their matched controls. In a subsequent classical conditioning paradigm (light-noise pairings), both parkinsonians and age-matched controls failed to elicit considerable SIRs or TORs (Sect. 3.1.2.1), which the younger controls did, supposedly dependent on the generally lower electrodermal reactivity of old-aged subjects (Sect. 2.4.3.1). Thus, in this investigation, age-related electrodermal effects may have simply bypassed possible neurological damage related effects. Therefore, further research in this area should attempt to strengthen the autonomic reactivity of old-aged groups such as parkinsonians and their controls (e.g., by increasing stimulus intensities).

Huntington's chorea may also be related to the CNS elicitation of electrodermal phenomena, since cell damage in the striatal part of the basal ganglia precedes cortical atrophy in the development of that particular illness. Establishing electrodermal markers for choreatic risk, as has been done for schizophrenia (Sect. 3.4.2.1), would be of high value for eugenic counselling in the offspring of Huntington's patients, who carry a 50% risk for developing the disease during midlife because of its autosomal dominant heritability. The marked deficit in electrodermal reactivity observed by Oscar-Berman and Gade (1979) in these patients, as described above, stimulated Lawson (1981) to compare electrodermal ORs [326] to 24 sounds ranging from 75–90 dB (including pure tones, white noise, and synthetic speech phonemes) taken from 52 symptom-free patients suffering from Huntington's chorea with those taken from 26 control subjects, tested at their homes. Seventeen subjects in the risk group turned out to be nonresponders, while only two control subjects showed electrodermal nonresponding, the difference being statistically significant.

In addition to these deficits in OR, Leonard, Podoll, Weiler, and Lange (1984) showed an increased habituation rate in 27 patients with Huntington's chorea and in 32 persons-at-risk for the disease (symptom-free offspring of patients) as compared to 26 normal controls. Habituation of SRR amp.[327] to 16 stimuli serving as UCSs in a classical conditioning paradigm (100 dB white noise) was terminated within the first eight presentations in 44% of the Huntington patients and in 16% of the persons- at-risk, but in only one of the control subjects.

By contrast, Iacono, Roshi, and Lacoste (1987) did not find significant differences in SCRs [328] between seven patients in the early stages of Huntington's disease and 29 offspring of these patients, on the one hand, and age- and gender-matched normal control samples on the other. Neither showed the at-risk group electrodermal abnormalities, nor could the patients themselves be differentiated from their controls by means of EDR.

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[326]Recorded bilaterally as SCRs with standard methodology, using .5 KCl agar paste. Time window: 3 sec after stimulus onset; amplitude criterion = .05 μS.

[327]Recorded with standard methodology using hypertonic paste. Time window: 1–5 sec after stimulus onset. Habituation criterion: three consecutive SRRs below 500 Ω (Sect. 3.1.1.3).

[328]Recorded bilaterally with standard methodology during three series of acoustic stimuli (eight 85 dB tones, twelve 105 dB tones, and two familiar sounds).

Further empirical work is needed to test assumptions concerning the specific indicator function of EDA for various neurological diseases in the CNS. Appropriate hypotheses can be derived from models of brain functioning connecting CNS structures to the elicitation of electrodermal concomitants of arousal and information processing mechanisms, as outlined in Sections 3.1.3.1 and 3.2.1.2.

3.5.2.3 EDA in other medical disciplines

Despite their specific use in dermatology and neurology, as described in the previous sections, electrodermal measures can be useful in various medical contexts where either the skin itself or the sympathetic branch of the ANS are involved. In this section, examples will be given predominantly of EDA as an indicator for internistic and psychosomatic disorders.

Lawler et al. (1960) summarized results of several studies on SZ and *dysfunctions* of the *thyroid* performed during the thirties, reporting that skin impedance and basic metabolic rates were correlated in hyperthyroidism but showed no relation to each other in hypothyroidism.

Knezevic and Bajada (1985) investigated the biphasic SPR on the electrical stimulation of the median nerve in 10 *diabetes* patients (see Sect. 2.5.1.1 for methodological details). SPR amp. were significantly lower than those taken from 30 controls, the differences being 300 μV at palmar, and 84 μV at plantar sites, while mean SPR lat. did not differ at either site.

Stocksmeier and Langosch (1973) recorded SR twice over a period of three weeks during olfactory, acoustic, and optical stimulation, from 57 male *rheumatic* patients and 24 controls, using dry silver electrodes. As compared to the controls, the patients showed significantly lower SRLs and SRR amp. during the first session, while after three weeks SRLs were higher in the rheumatics, and the SRR amp. did not differ between the two groups. The amplitude results were paralleled by an area measure (Sect. 2.3.1.4). Since during that short period no specific effect of antirheumatic treatment could be expected, the authors attributed the increase in tonic and phasic EDA to the general arousing effect of the treatment. No differences in SP or SC between asthmatic patients and normal controls were found by Williamson et al. (1985) in their study described in Sect. 3.5.2.1.

Doerr, Follette, Scribner, and Eisdorfer (1980) recorded SC with standard methodology during three valsalva maneuvers separated by 4 min periods. Both the SCL and the highest SCR amp. during each maneuver were significantly lower in 15 *dialysis* patients (9 males, 16 females) as compared to an age- and gender-matched control group of equal size. Furthermore, they obtained a significant correlation of .52 between the SCL and the residual creatine clearance. Since there were also positive correlations between EDA and nerve conduction velocity (r= .43 for SCL, and r= .41 for SCR amp.), the most suitable interpretation for the relationship between EDA and the amount of

residual kidney function is a reduction in ANS nerve conduction velocity in these patients (Muthny, 1984).

Attempts to establish differences in EDA between patients with tension headache or migraine and normal controls failed. Chattopadhayay, Mazumdar, and Basu (1982) as well as Thompson and Adams (1984) did not find appropriate electrodermal differences between these groups, either in habituation to a series of light flashes or during the inducing of stressful imageries.

Kopp (1984) did EDA comparisons of two other groups of *psychosomatic* patients (50 male hypertensives and 47 male duodenal ulcer patients, both groups in the early, mild, and reversible stages of the disease) with 65 male control subjects, during a sequence of different kinds of stimuli,[329] three tones (1 kHz, 93 dB), and 8 color stimuli. EDA was recorded as SR with standard methodology.). Both patient groups showed opposite electrodermal behavior in comparison with controls: NS.SRR freq. and EDR amp. were significantly reduced in hypertensives but increased in ulcer patients (where, however, the SRL was also increased). In the latter patients, a significant prolongation of SRR rec.t/2 following the emotionally disturbing words was obtained, interpreted by the authors as being due to a DR (Sect. 3.1.1.2), and not to a more intense information processing (Sect. 3.1.3.1); an interpretation which they used for the prolonged EDR recovery in the normal controls following unexpected stimulation.

Some specificity in EDA of ulcer patients during emotionally evoking situations was also found by Koller, Zidek, and Haider (1986). They recorded NS.SCR freq. as well as mean SCR amp. in 30 patients recovering from a recent ulcer and compared them to 30 patients recovering from their first myocardial infarction, matched for age and job status, in an experimental setting with different stressors (white intermittent noise, 90–92 dB; arithmetic with false-negative feedback; written anonymous personality reports; recall of negative live events). Maximum SCR amp. did not differ between the groups, and only in the emotionally evoking situation with personality reports, did the ulcer patients show a significantly higher NS.SCR freq. as compared to the myocardial infarction patients.

Frederikson, Dimberg, and Frisk-Holmberg (1980) compared cardiovascular and electrodermal activity in 14 patients (9 male, 5 female) suffering from essential hypertension and in 14 normotensive controls (10 male, 4 female), during two different tasks (letter identification, and mental arithmetic). During both conditions, NS.SCR freq.[330] tended to correlate negatively with systolic and diastolic blood pressure in hypertensives ($r = -.32$ to $-.48$,) while the reverse was observed in normotensives ($r = .23$ to $.52$). This result was not confirmed in a second study (Fredrikson, Dimberg, Frisk-Holmberg, & Ström, 1982) with another 9 male and 5 female hypertensives as compared to 15 age- and gender-matched normotensives, using the same kind of tasks. No significant cor-

[329]The stimulus conditions were: listening to classic music; a word association task (5 neutral words and 5 with a high emotional content).

[330]Recorded with 8 mm diameter Ag/AgCl electrodes filled with isotonic KCl paste from palmar finger sites. Amplitude criterion = .05 μS.

relation between NS.SCR freq. and systolic blood pressure was obtained, though the negative correlations for the hypertensives were of the same magnitude as those in the 1980 study, and the tendency towards positive correlations also appeared in the normotensives. Though this could only be observed during the arithmetic task (counting backwards silently from 1,070 in steps of seven), which was regarded as a typical case for "sensory rejection" in Lacey and Lacey's (1974) sense, the data did not support the hypothesis that differential electrodermal and cardiovascular reactivity is typical of hypertensives during the performance of rejection-type tasks.

Van Doornen, Orlebeke, and Somsen (1980) used a variation of the predictability of an aversive stimulus as a method to compare cardiovascular and electrodermal reactivity in 30 male infarction patients, 16 controls with high, and 18 controls with low risk for infarction. SCRs and HR were recorded following 10 tones (1 kHz, 115 dB), half of which were announced by a 60 dB 1 kHz warning tone given 12 sec before, the warning interval being monitored by a clock (Sect. 3.2.2.2), given in random order. A "preception" index (Sect. 3.1.2.1), being defined as percentage of the mean SCR amp. following warned in comparsion with unwarned aversive tones, yielded a significantly higher benefit of prewarning for the low-risk group as compared to both other groups. This difference was paralleled by a differential accelerative HR response component within 2–5 sec after warning tone onset, to which no significant group differences in SCR amp. appeared. The authors interpreted the HR acceleration following the warning stimulus as reflecting anticipation of an effective attenuating preception process (Lykken & Tellegen, 1974), leading to the attenuation of the EDR following the aversive stimulus, which was not present in infarction patients, or in coronary prone subjects.

Gruzelier, Nixon, Liddiard, Pugh, and Baxter (1986) used bilateral recordings of EDA patients with mixed cardiovascular disorders (e.g., chest pain, angina, hypertension, ischemia, and infarction) compared to age- and gender-matched controls (30 male, 10 female) to establish abnormalities in CNS control of ANS functions in this group of patients. SC, recorded with standard methodology using KCl paste, was chosen instead of cardiovascular parameters to measure habituation in a series of 13 tones (1 kHz, 70 dB) because of its pure mediation by the sympathetic branch of the ANS, unconfounded by peripheral parasympathetic influences. Using an habituation criterion of three subsequent trials without eliciting a SCR amp. greater than .02 μS, the patients showed a significantly slower habituation as compared to controls. The authors interpreted this result as suggesting an involvement of the limbic system in the development of cardiovascular disorders, since a slow habituation process indicates an over-wide range of attention, increased effort, and overresponsiveness, leading to information overload (Sect. 3.1.3.1), which may serve as a causative factor in the development of cardiovascular illness. This is in accordance with the observation that the SRR amp. to the first stimulus was significantly higher in patients than in controls. NS.SCR freq. during the ISIs and time parameters of EDRs did not differentiate between the groups. In addition, electrodermal lateralization effects appeared, with the

patients showing significantly higher right-hand SCLs (75% of patients as compared to 33% of controls), and ORs were also larger in the right hands of the patients, which was statistically supported by computing the laterality index (Equation (52), Sect. 3.1.3.4). These laterality effects were interpreted by the authors as reflecting the loss of contralateral inhibition of EDA from the left hemisphere, possibly due to the left hemisphere's vulnerability to fatigue in cardiovascular patients.

3.6 Summary and outlook

Given the background of various applications of EDA described in Part 3, the question arises as to whether or not the widespread use of EDA is justified by both theoretical and practical standpoints. Therefore, the different aspects of the validity of electrodermal measures will be summarized here.

Without question, the use of phasic EDA has its domain in stimulus-related basic psychophysiological research (Chapter 3.1). The early use of EDR recording in this field mainly focused on its indicator function in orienting and defensive reactions and their habituation (Sect. 3.1.1). EDA was also one of the most frequently used indicators in autonomic conditioning of the classical and instrumental kinds (Sect. 3.1.2).

More recently, the recording of various EDR parameters, including recovery behavior, has been used to deliberately analyze autonomic concomitants of information processing, such as uptake, comparison, and storage (Sect. 3.1.3). In this context the electrodermal system's high sensitivity for small changes in the external world as well as in CNS functions determined its specific value for this kind of basic research. As a consequence, several theoretical concepts in this field have been developed based on, or with specific respect to, electrodermal recording. Some precautions should be considered when using a peripheral indicator, the effector organs of which are far from the central processes under investigation. Properties of the peripheral system itself may obscure results, for example, when reaction interferences appear in cases of high stimulus frequencies with high EDR amp. and/or long EDR recovery times. Furthermore, dependencies of different EDA parameters on one another must be considered, such as amplitude/shape or phasic/tonic relationships (Sect. 2.5.2.5 & 2.5.4.2).

By contrast, the different parameters that can be obtained from EDRs may reflect specific aspects of the underlying psychophysiological processes. With respect to this, the size of the EDR amp. clearly increases with increasing stimulus intensity, while an increase in EDR recovery time reflects the focusing of attention (Sect. 3.1.3.1). Another example is the use of electrodermal lateralization effects in the investigation of hemispheric specialization, where different excitatory and inhibitory processes of phasic and tonic EDA have to be considered (Sect. 3.1.3.4). Furthermore, electrodermal variables play an important role in the general context of a multivariate psychophysiological approach, which can be seen in various attempts to determine the physiological concomitants of generalized psychological states like levels of arousal, motivation,

emotion, and stress (Chapter 3.2). Electrodermal parameters have been especially useful to obtain fine-grained analyses of lower activational states, where cardiovascular variables, also of widespread use in this kind of research, no longer show differentiation.

The suggested areas of validity for electrodermal and cardiovascular measures, presented in Part 3, should be seen in light of the theoretical background given by the neurophysiological modelling outlined in Section 3.2.1.2 (see Fig. 48). However, the present models should be regarded as preliminary, since they are not yet fully supported by empirical evidence. Nevertheless, the available evidence is encouraging to further research. EDA measures are highly applicable to emotions and stress research. This is because EDA is mediated solely by the sympathetic branch of the ANS and is therefore not subjected to peripheral parasympathetic influences as most other autonomic variables are. The NS.EDR freq. can especially be regarded as a valid indicator for the strength of emotion, for observing the course of emotional stress, and for objectively determining coping efficacy (Sect. 3.2.2.2). In addition, consideration should be given to the use of different EDA parameters as markers for the various emotional states, as for example in research on emotional expression, where EDR amp. is correlated with the inner emotional involvement, while HR is more likely to reflect overt emotions.

Besides these areas of basic research, the main application field of EDA is clinical psychophysiology (Chapter 3.4). Since EDA is a valid indicator in emotions and stress research, its recording for the purpose of assessment and treatment evaluation in anxiety-related psychopathological states is of special value (Sect. 3.4.1). Again, EDA is a highly sensitive indicator for minimal changes in generalized anxiety states, while cardiovascular measures find their strength in high-arousal states of anxiety such as phobias (Sect. 3.4.1.1). EDA has also shown some properties of being a marker for predicting the development of psychopathic and antisocial behavior in persons at risk (Sect. 3.4.1.2).

Prospective studies of persons at risk for schizophrenic illness (Sect. 3.4.2.1) is the main area of research where electrodermal parameters have been successfully used as predictors for the development of psychopathological behavior. Here, recovery characteristics of the EDR are a specific marker for schizophrenia. Theoretical support comes from suggested deficits in subcortical information processing appearing in schizophrenics, being just opposite to deficits assumed in psychopaths (see Table 6, Sect. 3.1.3.1). The most widespread use of EDA in schizophrenia research is, however, in trying to establish causes and consequences of electrodermal nonresponding in a large fraction of that clinical group (Sect. 3.4.2.2). In this field, transcultural conjoint research has achieved a high standard, yielding differential diagnostic and prognostic values of both electrodermal nonresponding and nonhabituating. Furthermore, it will be of theoretical and practical interest to consider hemispheric dominance and electrodermal hyporeactivity together with fast habituation in schizophrenics (Sect. 3.4.1.3).

In accordance with its specific validity in objectively determining even small changes in anxiety levels, EDA has been used as a major dependent variable in the psy-

chopharmacological treatment of anxiety (Sect. 3.4.3). However, in this field precaution has to be taken with respect to possible central and peripheral influences of drugs on the elicitation of electrodermal phenomena itself, as is especially the case when using neuroleptics with anticholinergic properties (Sect.3.4.3.2). The therapeutic use of EDA biofeedback, which was frequently advocated during the seventies, has been dropped because of inconsistent results (Sect. 3.1.2.2).

Another field of application where EDA was used more frequently in the past than in the last one or two decades is in personality and individual differences research (Chapter 3.3). Though some theoretical attempts have been made to relate general personality trait characteristics to psychophysiological indicators, including EDA parameters, the postulated individual differences cannot be demonstrated clearly (Sect. 3.3.1), since expenditive multitrait-multimethod studies in this field are lacking. With respect to electrodermal lability as a stable individual characteristic, it remains to be seen how it can be embedded in a framework of personality dimensions which are mostly obtained by questionnaire data (Sect. 3.3.2.2).

In contrast to their widespread use in clinical applications, electrodermal measures have frequently not been considered as research tools in other fields of applied psychology, except in the detection of deception (Sect. 3.5.1.2), an application that remains controversial. The use of EDA in engineering psychology, including applications in traffic, industrial, as well as in office research, is of especially high practical value, because of the differential validity of EDA as an indicator for emotional and/or mental strain as opposed to physical strain, which in turn is more adequately reflected by cardiovascular changes (Sect. 3.5.1.1).

In addition to various applications of EDA in psychology, there are several fields in medicine for which electrodermal variables are of specific value for diagnostic and therapeutic evaluation purposes (Sect. 3.5.2). Among these, dermatology as well as neurology can make use of the exact quantification of skin processes as well as of changes in the central and peripheral nervous systems by means of EDA, as opposed to the more qualitative evaluation of sweating rate. At the intersection of medicine and clinical psychology, assessment and therapy in psychosomatic disorders can be regarded as another theoretically guided area of application for electrodermal recordings.

In summary, the present book did not aim at uncritically recommending the use of electrodermal methods in all research areas. Instead, the focus was on critically discussing specific aspects of validity with respect to related methodological issues. To avoid misinterpretations or even misuses of EDA, precaution has been recommended with respect to a possible unsophisticated use of easy-to-obtain electrodermal signals as well as easy-to-detect electrodermal reactions. When using electrodermal recording, methodological aspects should be carefully considered, including recommendations for standardization, as outlined in Part 2. Furthermore, basic as well as applied electrodermal research could be improved much by forming a theoretical background, combining the psychophysiological approaches (outlined in Part 3) with knowledge about the neurophysiological origins of electrodermal activity (described in Part 1).

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Subject Index

Appendix

SCRGAUGE - A computer program for the detection and quantification of SCRs

by Peter Kohlisch, Wuppertal, Germany

SCRGAUGE is user supported software (shareware). It is written in C and available for MS-DOS on IBM AT or compatible computers. A version for TOS on Atari ST computers can be made available as well. You may license the executable program for non-commercial use on one machine by sending a cheque for 20 US Dollars to: Dr. Wolfram Boucsein, Max-Horkheimer-Strasse 20, W-5600 Wuppertal 1, Germany. All other rights are reserved. All licensees will recieve prompt notification of program revisions and will be able to purchase updates for a nominal fee. All warranties are disclaimed, including damage to your hardware and/or software from use of this program. You are welcome to share your experiences in using the program and/or your proposals for changing the program with the author at the above address. Please specify the size and density of your floppy drive (e.g., 3.5" HD). The program SCRGAUGE.EXE for IBM AT requires DOS 4.0 or a higher version and an 80286 processor. An 80287 numeric co-processor is supported. The program SCRGAUGE.TTP for Atari ST requires TOS 1.4 or a higher version and a 68000 CPU. An SFP004 compatible FPU extension is supported.

1. Pretreatment of data

Raw data are treated in three steps.

(1) Recording artifacts such as sharp pulses are removed from integer-based data.

(2) Data are converted to real numbers and filtered by discrete Fourier transformation, using a decimation-in-time FFT (cf. Oppenheim & Schafer, 1975, Sect. 6.2.1). This procedure has been chosen because it does not cause signal deformations or time shifts.

(3) An interpolation with the use of a curve fitting function is performed. For this procedure, the sampling rate is reduced from the original sampling rate (e.g., 16 or 20 Hz) to 2 Hz in order to accelerate the evaluation and to obtain an additional smoothing of data. The function applied is cubic procedure whereby two adjacent data points are connected with a third-order polynomial, and continuity of the first and second derivatives are required at all data points (cf., Ahlberg et al., 1967, Sect. 2.3.1.3.2).

2. Recognition and quantification of SCRs

Detection and evaluation of SCRs is performed in four steps:

(1) SCRs are detected by evaluating the gradient of incline at the data points. If the incline exceeds a given value, the quantification will be started.

(2) Detection of the point of SCR onset is accomplished by moving backward in the curve to the point of maximal curvature.

(3) Detection of the SCR maximum is accomplished by moving forward until the incline becomes negative. The exact point for the maximum is given by the 1st derivative approaching zero. Humps are detected when more than one point of inflection appears before the SCR maximum is reached. If humps appear, they are investigated for a maximum. In this case, the response is identified as "double SCR".

(4) Calculation of SCR rec.t/2 is performed. The algorithm determines the values of the curve until the curve falls below 1/2 SCR amplitude. To obtain the point of time in the interval left of it where half recovery is reached, Newton's method is used instead of a time consuming solution of cubic equations. If the incline becomes positive before the curve falls below 1/2 SCR amp., SCR rec.t/2 will be estimated by the tangent at the point of maximal decline.

3. Parameters of SCR being evaluated

The program module SCRFIND calculates the following values and writes them to global values:

(1) Time point of SCR onset

(2) SC at the point of SCR onset

(3) SC at the point of SCR onset

(4) Time point of the maximal SCR amp.

(5) SC at the point of maximal SCR amp.

(6) Time point for SCR rec.t/2

(7) Number of humps found in the ascent

(8) Type of SCR (see below)

Calculation of SCR ris.t., SCR amp., and SCR rec.t/2 is performed by the external output routine writeparms.

The type of SCR contains information on the point of SCR onset and on recovery time. Three classes of types are distinguished by the first decimal:

(1) Blank (Types 1 - 5): SCL decays below 1/2 SCR amp.

(2) 1 (Types 11 - 15): SCL does not decay below 1/2 SCR amp.; SCR rec. t/2 has been extrapolated.

(3) 2 (Types 21 - 25): SCR rec.t/2 cannot be calculated (has the value 0.0) or its value obtained by extrapolation has to be regarded as doubtful, thus requiring inspection of the data by the experimenter.

The differentiation by the second decimal characterizes the course of SCL immediately before SCR onset:

(1) 1 (Types 1, 11, or 21): The course of the SCL left of SCR onset is approximately parallel to the zero line. Criterion: A zero of the 1st derivative is found left from the point of maximal deflection.

(2) 2 (Types 2, 12, or 22): The SCR onset falls into a period of decreasing SCL. In most cases, this points to a superposition of SCRs, which is always the case if the preceding SCL has a type number of 11 or higher. Criterion: A zero of the 1st derivative is found forward to the point of maximal deflection.

(2) 3 (Types 3, 13, or 23): The SCR onset falls into a period of decreasing gradient. Criterion: A minimum of the 1st derivative is found before the point of maximal curvature.

(4) 4 (Types 4, 14, or 24): The SCR onset falls into a period where the SCL incline is nearly linear, and cannot be determined by a criterion of its 1st derivative. In this case, the lower limit of the interval before the point of maximal curvature is regarded as SCR onset.

(5) 5 (Types 5, 15, or 25): The SCR starts within a rather short period after the maximum of the previous SCR. These types are only found during the investigation of humps.

The program will be printed on the following pages, and an example list of parameters will be provided at its end.

```
/*************************************************************************\
*                                                                         *
*      SCRGAUGE - A program for detection and parametrization of SCR      *
*                                                                         *
*                     created in 1992 by Peter Kohlisch                   *
*                                                                         *
*                                                                         *
*  module "SCRFIND.C" : detection and parametrization                     *
*                                                                         *
\*************************************************************************/


#include <math.h>

#define elsif else if
#define polyval( p, t ) ((p).alfa + (t) * \
                         ((p).beta + (t) * ((p).gamma + (t) * (p).delta)))
#define polydlv( p, t ) ((p).beta + (t) * \
                         (2. * (p).gamma + (t) * 3. * (p).delta))
#define EPSILON 1e-3


typedef struct { double alfa, beta, gamma, delta; } spline_t;


/*  Functions for input data request.  */

int getpoly( spline_t *spl, long index );
int getbeta( double *val, long index );
int getalfa( double *val, long index );


/*  Output function and data  */

void writeparms( void );

double basetime, baseval, maxtime, maxval, desctime;
int scrtype, nhump;


double mingrad, halfval;
```

```
static int zeroderiv( double *zero, spline_t *s, int nzeros ) {

  /*  Returns up to nzeros real roots of the first derivate of a        */
  /*  spline_t polynomial s on the half-open interval [0,1[. The roots  */
  /*  are returned in a double array passed via zero. If there is any   */
  /*  root, zero[0] contains the one nearest to 1. The value of         */
  /*  zeroderiv() is the number of roots found.                         */

  double p, q, d, t;
  int cntl = 0;

  q = 1. / ( 3. * s->delta );
  p = -s->gamma * q;
  q *= s->beta;
  if ( ( d = p * p - q ) < 0. ) return 0;
  d = sqrt( d );
  t = p + d;
  if ( t >= 0. && t < 1. ) {
    *zero++ = t;
    cntl++;
    nzeros--;
    }
  t = p - d;
  if ( nzeros > 0 && t >= 0. && t < 1. ) {
    *zero = t;
    cntl++;
    }
  return cntl;
  }


static void findbase( long index ) {

  /*  Steps back to a point of maximal curvature. The basepoint of the  */
  /*  SCR lies in either of the intervals left and right to this point. */
  /*  The basepoint is a point of zero gradient or minimal gradient     */
  /*  (i.e. a zero of the 2nd derivate). If none of these conditions    */
  /*  is satisfied the basepoint is assumed to be the lower bound of    */
  /*  the left hand interval.                                           */

  double val;
  spline_t s[ 2 ], *s0, *s1, *sh;
```

```
  s0 = s; s1 = s + 1;
  getpoly( s0, index );
  do {
    sh = s0; s0 = s1; s1 = sh;
    if ( !getpoly( s0, --index ) ) {
      basetime = 0.;
      baseval = s1->alfa;
      scrtype = 0;
      return;
      }
    } while ( s0->gamma >= s1->gamma || s1->beta >= 2. * s1->gamma );
  if ( zeroderiv( &val, s0, 1 ) ) {
    basetime = index + val;
    scrtype = 1;
    }
  elsif ( zeroderiv( &val, s1, 1 ) ) {
    basetime = +( index + 1 ) + val;
    s0 = s1;
    scrtype = 2;
    }
  elsif ( ( val = -s0->gamma / ( 3. * s0->delta ) ) >= 0. && val <= 1. ) {
      basetime = index + val;
      scrtype = 3;
      }
  else {
    basetime = index;
    val = 0.;
    scrtype = 4;
    }
  baseval = polyval( *s0, val );
  }


static void inspecthump( long index ) {

  /*  Steps back to an interval containing a zero t of the 2nd derivate */
  /*  which indicates a point of minimal gradient. If s(t) > 0 holds,   */
  /*  merely nhump has to be incremented. If not, s(t) has two zeros    */
  /*  in the interval under investigation. This means two, seperate     */
  /*  SCRs have to be evaluated. The left zero is the point of maximal  */
  /*  conductance of the first SCR, and the right one is the onset of   */
  /*  the second SCR.                                                   */
```

```
  double t, mingrad, zeros[2];
  spline_t s;

  do getpoly( &s, --index ); while ( s.gamma > 0. );
  t = -s.gamma / ( 3. * s.delta );
  if ( ( mingrad = polydlv( s, t ) ) > 0. ) nhump++;
  else {
    zeroderiv( zeros, &s, 2 );
    t = zeros[ 1 ];
    desctime = maxtime = index + t;
    maxval = polyval( s, t );
    scrtype += 20;
    writeparms();
    nhump = 0;
    t = zeros[ 0 ];
    basetime = index + t;
    baseval = polyval( s, t );
    scrtype = 5;
    }
  }


static void findmax( long *index ) {

  /*  Steps forward to a point with gradient <= 0. If the gradient is   */
  /*  equal to zero the maximum is already found. If not, there is one  */
  /*  zero of the gradient in the interval left to this point,          */
  /*  indicating the maximum of the SCR. If the gradient increases      */
  /*  before the maximum is found the inspecthump will be called.       */
  /*  Afterwards findmax steps forward until the gradient decreases,    */
  /*  and starts again.                                                 */

  double grad, lgrad, val;
  long k;
  spline_t s;

  nhump = 0;
  for ( k = *index , getbeta( &grad, k ) ; grad > 0. ; ) {
    lgrad = grad;
    if ( !getbeta( &grad, ++k ) ) {
      maxtime = basetime;
      return;
      }
```

```
    if ( lgrad <= grad ) {
      inspecthump( k );
      do {
        lgrad = grad;
        if ( !getbeta( &grad, ++k ) ) {
          maxtime = basetime;
          return;
          }
        } while ( grad > lgrad );
      }
    }
  *index = k;
  if ( grad < 0. ) k--;
  getpoly( &s, k );
  zeroderiv( &val, &s, 1 );
  maxtime = k + val;
  maxval = polyval( s, val );
  }


static void extrapol( long index ) {

  /*  Steps back to a point where the 2nd derivate is <= 0. In the      */
  /*  left hand interval is a point of maximal descent indicated by a   */
  /*  zero of the 2nd derivate. From this point the SCR curve is        */
  /*  linearly extrapolated to estimate the recovery time.              */

  spline_t s;
  double maxdesc, t_maxdesc;

  scrtype += 10;
  do getpoly( &s, --index ); while( s.gamma > 0. );
  if ( s.gamma == 0. ) t_maxdesc = 0.;
  else t_maxdesc = ( -s.gamma / ( 3. * s.delta ) );
  maxdesc = polydlv( s, t_maxdesc );
  if ( polyval( s, t_maxdesc ) > .85 * ( maxval - baseval ) + baseval )
    scrtype += 10;
  if ( maxdesc == 0. ) desctime = basetime;
  else {
    halfval -= polyval( s, t_maxdesc );
    desctime = index + t_maxdesc + halfval / maxdesc;
    }
  }
```

```
static void finddesc( long *index ) {

  /*  Steps forward until the SCL has decreased below the half of the   */
  /*  SCR amplitude (halfval). Now a t wich satisfies the equation      */
  /*  s(t) = halfval is approximated by Newton's method. If the         */
  /*  derivate becomes >= 0 and the SCL is greater than halfval, the    */
  /*  recovery time is extrapolated.                                    */

  long k;
  double val, desc, desc0, t;
  spline_t s;

  halfval = .5 * ( maxval + baseval );
  k = *index;
  do {
    k++;
    if ( !getbeta( &desc, k ) ) {
      desctime = maxtime;
      scrtype += 20;
      *index = k;
      return;
      }
    if ( desc >= 0. ) {
      extrapol( k );
      *index = k;
      return;
      }
    getalfa( &val, k );
    } while ( val > halfval );
  if ( val == halfval ) {
    desctime = k;
    }
  else {
    k--;
    getbeta( &desc0, k );
    if ( desc0 < desc ) {
      getalfa( &val, k );
      t = ( halfval - val ) / desc0;
      }
    else t = 1. + ( halfval - val ) / desc;
    getpoly( &s, k );
```

```
    do {
      desctime = t;
      t += ( halfval - polyval( s, t ) ) / polydlv( s, t );
      } while ( fabs( desctime - t ) > EPSILON );
    desctime = t + k;
    }
  *index = k;
  }


void findscr( void ) {

  /*  The main function. Searches for an interval where the gradient    */
  /*  is >= mingrad. Then findscr steps forward until the gradient de-  */
  /*  creases. Now the analysis of the SCR will be started.             */

  long index;
  double grad, lgrad;
  int more;

  for ( index = 0 ; ; ) {
    while ( ( more = getbeta( &grad, index ) ) != 0 && grad < mingrad )
      index++;
    if ( !more ) return;
    do {
      lgrad = grad;
      index++;
      } while ( ( more = getbeta( &grad, index ) ) != 0 && grad > lgrad );
    if ( !more ) return;
    findbase( index - 1L );
    findmax( &index );
    finddesc( &index );
    writeparms();
    }
  }
```

A parameter list is written on a computer file for every SCR as follows:

No	type	SCR onset	SCR ris.t	amplitude	SCR rec.t/2	humps in ascent
1	4	7.00	2.05	0.223	1.70	0
2	3	12.69	2.20	0.060	0.85	0
3	11	19.31	3.71	0.540	3.06	1
4	2	35.04	1.36	0.052	0.97	0
5	11	44.66	4.96	0.934	2.20	2
6	2	51.68	0.67	0.024	0.36	0
7	21	60.27	3.25	0.505	1.58	0
8	2	64.59	1.10	0.079	0.46	0
9	2	67.18	1.35	0.170	0.67	0
10	1	71.38	1.53	0.219	1.13	0
11	1	76.14	1.36	0.099	0.99	0
12	24	81.50	1.73	0.250	0.00	0
13	5	83.38	1.00	0.035	0.36	0
14	2	90.52	1.40	0.056	1.14	0
15	4	103.00	1.85	0.081	1.61	0