Electrodermal activity in the 1980s: a review[1]

Margaret J Christie PhD
Applied Psychobiology Research Unit
School of Studies in Psychology, University of Bradford, West Yorkshire BD7 1DP

Aims and intentions

This paper aims to provide pathways through the forest of nomenclature and techniques in contemporary electrodermatology, to describe the recording of some electrodermal indices in areas relevant to psychopharmacology, and to look briefly at developments likely to influence electrodermal work in the 1980s.

The area is being reviewed, not by a psychopharmacologist, but by a psychophysiologist with interests in peripheral mechanisms underlying electrodermal phenomena. Measurement of electrodermal indices provides evidence of eccrine sweat gland activity and its variation with changes in psychological state: the eccrine sweat glands are innervated by sympathetic fibres (although there is cholinergic transmission at postganglionic sites) and, in normal ambient temperatures, palmar (or plantar) glands reflect responses to psychological rather than thermoregulatory stimuli.

This review is necessarily superficial but useful expansion of some of its material can be found in a section of the 'Handbook of Biological Psychiatry' (van Praag *et al.* 1980), particularly chapters by Walrath & Stern, Siddle *et a'.*, Venables, Christie *et al.*, Lader.

Terminology and techniques

Electrodermal activity may be more familiar as the GSR or the PGR, but the 1970s saw a succession of publications (Edelberg 1972, Venables & Christie 1973, Christie 1976*a*) in which the case was made for elimination of these hallowed terms, recorded electrodermal phenomena by then being too varied and complex for adequate description by the traditional labels, the use of which invited confusion and misconception. The term 'psychogalvanic reflex' (PGR) seems to have been coined by a neurologist, Veraguth, who saw a demonstration of palmar sweat gland responses to sensory stimulation – recorded as changes in potential – and in the early 1900s introduced his PGR technique to Jung. The latter welcomed this as offering scope for probing the unconscious, and the emotions associated with 'hidden complexes'. Jung's enthusiasm was contagious and for a couple of decades research reflected this 'mind reading' use of electrodermal recording, but there was less attention to investigation of physiological mechanisms and measurement techniques. By mid-century, McCleary (1950) was deploring the prevailing ignorance about fundamental mechanisms and in 1966 Tursky & O'Connell expressed disenchantment with results from electrodermal methods characterized, as they saw them, by unreliability. Lykken & Venables, however, wrote in 1971 that electrodermal recording '... continues stoutly to provide useful data in spite of being abused by measurement techniques which range from the arbitrary to the positively weird'. Their assessment was based on awareness of those reliable results which had been generated since mid-century, from fundamental research on mechanisms and from developments in measurement techniques. This blossoming of research and development resulted in a range of electrodermal phenomena being reported as recordable, and in the generation of a plethora of labels. By 1967 there were proposals for a standardized nomenclature from Brown (1967) and Venables & Martin (1967*a*); extensions of nomenclature were needed for a review chapter in the 1970s (Venables & Christie 1973) and the most recent summary of phenomena and

[1] Paper read to Section of Measurement in Medicine, 13 April 1981. Accepted 15 May 1981

0141–0768/81/080616–07/$01.00/0 © 1981 The Royal Society of Medicine

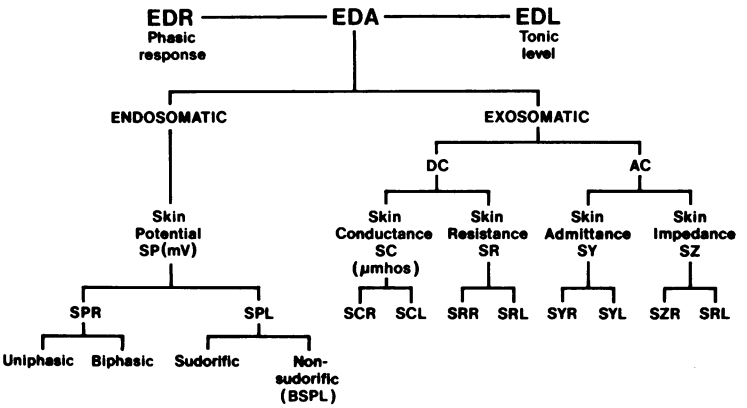

Figure 1. Contemporary labelling of electrodermal activity

labels is now available in Venables & Christie (1980). A schematic view of current phenomena and their labels is shown in Figure 1.

Electrodermal activity (EDA) is a general term for the electrical activity originating from eccrine sweat glands (sudorific) and their associated dermal and epidermal tissues (non-sudorific). The terms 'phasic' and 'tonic' differentiate between phenomena with shorter and longer time courses, the former being electrodermal responses (EDR) to eliciting stimuli, or of unknown origin and labelled as nonspecific responses (NS). Tonic phenomena are reflected in electrodermal levels (EDL): these change more slowly and EDR, changes which are measured in seconds, are superimposed on a more slowly changing EDL.

Two aspects of EDA have been reported since the latter part of the nineteenth century: Hermann in Zurich demonstrated during the 1870s and 1880s that the secretory processes of eccrine gland function were associated with generation of a potential, and then Tarchanoff (1889) recorded potential differences between a palmar and a reference site, changes in which were associated with the presentation of sensory stimuli or, for example, exploration of subjects' memories. Both workers were recording endosomatic EDA, in contrast to Charcot, Vigouroux & Féré who all recorded the galvanic skin response of a change in resistance to current, or exosomatic EDA (for review *see* Neumann & Blanton 1970).

Exosomatic EDA is recorded with either direct or alternating current. In the former case it is preferable to record skin conductance, by a constant voltage method (Lykken & Venables 1971), or to convert resistance, recorded by a constant current method, to its reciprocal

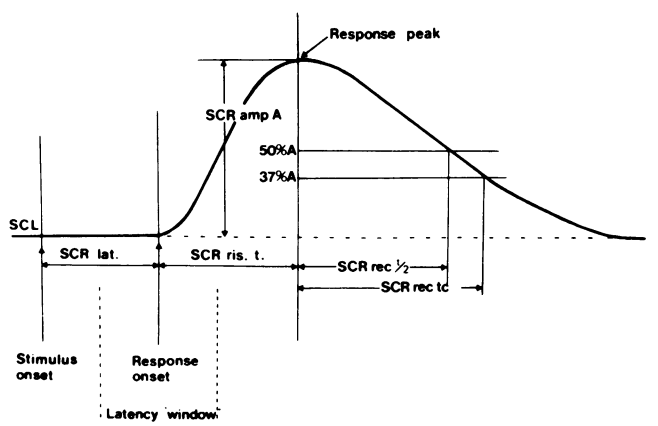

Figure 2. Schematic skin conductance response. (Reproduced from Venables & Christie 1980, with kind permission)

conductance value. Tonic and phasic aspects of skin resistance (SR) are shown in Figure 1, but will be largely neglected in this review, as will the AC recorded measures of skin admittance (SY) and skin impedance (SZ): subsequent description is restricted largely to skin conductance (SC) and skin potential (SP) methods.

Skin conductance levels (SCL) and responses (SCR) provide a range of information: data from the phasic changes of the skin conductance responses are seen in Figure 2, which shows a schematic skin conductance response to a stimulus. Here is seen the measurement of amplitude (as the maximum displacement from the tonic level) together with three temporal measures of latency (between stimulus onset and response onset), rise time (between response onset and response peak) and recovery time (between response peak and return to pre-stimulus level, but usually reported as half-time (t/2) or sometimes in terms of the time constant). If a mean value for a number of response heights is reported, this may be calculated for all occasions where a response is given, and expressed as amplitude; or calculated for all occasions where a response might be given (i.e. all stimulus occasions) and expressed as magnitude. Venables and his co-workers have recently recommended, on the basis of findings from some 2500 subjects, log conversion of SCL and SCR amplitude to produce normally distributed data (Venables & Christie 1980).

Figure 3 shows an SCR together with one form of skin potential response (SPR). Endosomatic recording provides assessment of SPR and of skin potential levels (SPL). The former may be biphasic, as seen in the figure, or uniphasic, or even triphasic, thus presenting problems of measurement. SPL has been shown to have two distinct aspects: SPL, recorded when there is evidence of any sudorific activity, is an index of autonomic activity, which can be correlated with heart rate and SC indices (Christie & Venables 1971*a, b*); but with eccrine sweat gland quiescence the non-sudorific 'basal' skin potential level (BSPL) appears to reflect the generation of an electrochemical potential (Christie & Venables 1971*c*, Christie 1976*b*).

Turning from nomenclature to measurement techniques, there has been recent activity in the American Society for Psychophysiological Research to provide guidelines and foster standardization. A selection of fundamental points from the report of a working party (Fowles *et al.* 1981) is offered here.

(*a*) Electrodes should show minimal bias potential and not polarize on the passage of current. Silver-silver chloride (Ag/AgCl) laboratory-made (Venables & Sayer 1963) or commercially available electrodes are widely used: zinc-zinc sulphate electrodes (Lykken 1959) provide an alternative. Considerable care in preparation, maintenance, monitoring and use of electrodes is essential, particularly for endosomatic (SP) recording.

(*b*) Commercially produced electrolytes are usually unsuitable for electrodermal recording: NaCl or KCl in physiological concentrations and appropriate media must be selected. Inappropriate electrolyte alters epidermal characteristics and influences the electrochemical potential (BSPL).

(*c*) Palmar sites for electrode placement are shown in Figure 4. SC is measured with a bipolar placement on midphalangeal sites of fingers 1 and 2 (or 3 and 4). SP needs a unipolar

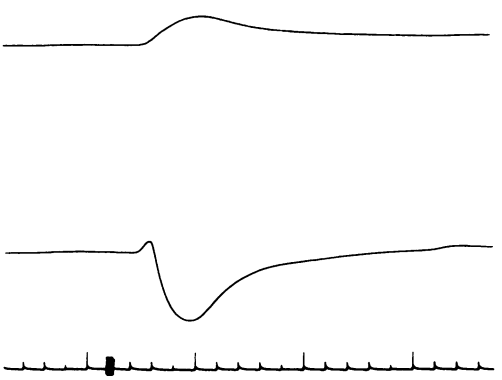

Figure 3. Conventional polygraph write out of skin conductance response (upper) and biphasic skin potential response (lower). (Reproduced from Venables & Christie 1980, with kind permission)

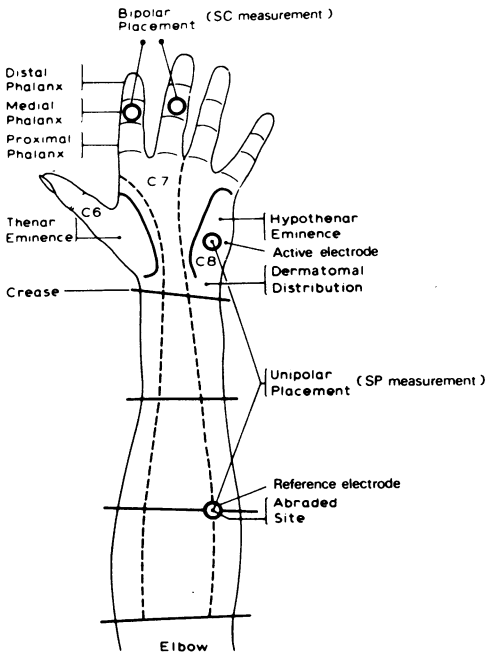

Figure 4. Suggested palmar electrode sites for measurement of skin conductance and skin potential activity. (Reproduced from Venables & Christie 1980, with kind permission)

placement with the active electrode on a finger site (as for SC) or on the thenar or hypothenar eminence. The reference electrode is placed over an abraded site on the volar forearm surface.

Commercially produced polygraph systems (e.g. Grass) provide appropriate high impedance pre-amplifiers, suitable for SP recording, but it may be necessary to construct equipment for SC measurement. Further, if SC response amplitude (or magnitude) is the index being measured, this can be recorded at greater gain from a zero baseline by suppression of the output proportional to the SCL. An automatic voltage suppressor suitable for use with computer analysis of SC data is described in Venables & Christie (1980). EDA remains, however, an index which can be recorded with relative ease and handscored without excessive difficulty, hence its enduring popularity.

Use of electrodermal indices in areas relevant to psychopharmacology

This section looks at some fundamental research on peripheral mechanisms underlying EDA, assessment of EDA in relation to subclassification of depressive state, and assessment of phasic changes reflecting treatment effects in (a) anxiety and (b) schizophrenia with propranolol, and with maintenance phenothiazines in home environments.

Mechanisms underlying EDA

Lader (1975) has reviewed psychobiological aspects of psychopharmacology under two headings, the first of which is 'use of drugs to evaluate peripheral psychophysiological mechanisms'. He reports his work with Montagu (Lader & Montagu 1962) in which the palmar SCR was abolished by iontophoretic introduction of atropine without affecting vasomotor activity, while bretylium, iontophoresed into a palmar site, abolished vasomotor activity but left SCR unaffected. This excluded a role for vasomotor generation of SCR and established that SCR was subserved by a cholinergic mechanism: as palmar eccrine sweat glands were the only known structures with cholinergic innervation at this site the dependence of SCR on sweat gland function was established.

Venables & Martin (1967*b*) extended this work, demonstrating that anticholinergic iontophoresis was associated with major effects on SCR, SCL and SPR, but that only 25% of recorded SPL was reduced by this blocking of sudorific function. Thus a significant portion of

SPL was, apparently, generated by a non-sudorific mechanism. Christie & Venables, in their series of studies during the early 1970s, showed that the value of this BSPL could be expressed as a function of the potassium (K^+) concentration gradient between the KCl in the Ag/AgCl recording electrodes and a source of K^+ in the skin (Christie 1976*b*).

Recorded EDA and diagnosis of depressive state

Psychophysiological contributions toward the understanding, subclassification and treatment of depressive states have recently been reviewed by Christie *et al.* (1980). Attempts to classify depressive states appear still unable to yield clear evidence of their possibly diverse aetiology: Angst (1974) commented, in relation to depression, that it 'has become urgent in doing therapeutic research to deal with diagnostically correct entities', and it may be that measurement of peripheral indices such as EDA can contribute to adequate diagnosis. Selecting from psychophysiological contributions some studies of EDA and subclassification of depressive state: Lader & Wing (1969) reported that a group of agitated depressives failed to show habituation of SCRs, while most of their retarded depressives showed so little reactivity that habituation rates could not be estimated. Noble & Lader (1971, 1972) subsequently showed that severity of depressive symptomology tended to be associated with low SCLs and reduced nonspecific SCRs, endogenous depressives being particularly likely to show these characteristics. Byrne (1975) compared neurotic and psychotic depressives: the latter had lower SCLs and few nonspecific SCRs while the former showed high SCLs and more spontaneous fluctuations.

Myslobodsky & Horesh (1978), however, reported evidence of reduced EDA in reactive depressives during a cognitive task: thus, while there is evidence of the potentially useful role for EDA recording, data are obviously not wholly unequivocal in the area of subcategorization of depressive state.

Phasic EDRs reflecting psychopharmacological treatment effects

(*a*) *In anxiety*

Lader & Wing (1966) reported on the value of EDA measurement for assessment of the comparative effectiveness of barbiturate drugs and chlorodiazepoxide in the treatment of pathological anxiety. Tonic levels were measured, together with enumeration of nonspecific responses and with investigation of habituation. They used SCR amplitude measurement in conjunction with presentation of a series of identical auditory stimuli. Compared with control subjects, patients showed increases in nonspecific responding, with delayed habituation; both indices were affected by drug treatment, EDRs being reduced and habituation accelerated. Habituation and the complementary phenomenon of orienting are also central to the discussion of schizophrenia and propranolol which follows.

(*b*) *In schizophrenia*

Propranolol: Several lines of evidence support the view that schizophrenic pathology reflects limbic system dysfunction. Given the role of limbic structures in orienting and habituation, and the EDA abnormalities of these functions seen in monkeys with lesions of the amygdala and hippocampus, it became appropriate to focus attention on these structures and their possible dysfunction, as this might be reflected in the EDA of schizophrenics. This earlier work was reviewed by Venables (1975), and reports of Gruzelier (e.g. Gruzelier & Yorkston 1978, Gruzelier 1978) describe restoration of more normal orienting and habituation in schizophrenics treated with propranolol.

Gruzelier (1978) describes the phenomenon of EDR habituation, when amplitudes of successive SCRs diminish with repeated presentation of a moderately intense pure tone. Amygdalectomized monkeys do not show orienting and then habituation: they either fail to exhibit any response or fail to habituate with repeated presentation, while hippocampal monkeys fail to habituate. Schizophrenics have been shown to display disorders of orienting and habituation similar to those of amygdalectomized monkeys – approximately 50% being non-responders and 50% being responders with deficits of habituation to meaningless sound stimuli. Reports of the antipsychotic potential of propranolol for the treatment of

schizophrenia include accounts of clinical, metabolic and pharmacological findings (e.g. Hanssen *et al.* 1980) and summaries from several groups of research workers active through the 1970s. Gruzelier and co-workers have reported on psychophysiological findings, particularly on restoration of more normal EDR habituation in propranolol-treated schizophrenics. Further, Gruzelier *et al.* (1979) report the examination of dextro-propranolol (which has significantly less hypotensive action) and its therapeutic effects in schizophrenia, seen as rated clinical improvement and facilitated habituation of EDRs. These workers attribute the effect to a central not a peripheral action of propranolol and note the lack of restored habituation with chlorpromazine.

Maintenance phenothiazines in home environments: Leff & Tarrier (1981) have reviewed work from the British Medical Research Council's Social Psychiatry Unit which has examined interactions between the emotional quality of a schizophrenic's home environment, the possibility of a patient being able to maintain social distance from a key relative, phenothiazine medication and the extent of relapse in the period following discharge from hospital. Relapse during a 9-month period at home was predictable from an index of Expressed Emotion (EE): this assessed over-involvement of, with hostility and critical comments from, the key relative during interview on the patient's admission to hospital. Patients in high EE homes, having minimal social distance and not taking drugs, showed a relapse rate of 92%; if they did distance themselves by spending less than 35 hours per week with the key relative, relapse rate dropped to 42% and if they also took medication it was down to 15%. Investigation of EDA – SCL, SCR and nonspecific responses – was undertaken by Tarrier *et al.* (1978*a*) who recorded these data in home and then in the hospital laboratory and reported that EDA in patients from high EE and from low EE homes could not be distinguished when recorded in the hospital environment; however, when tested at home (Tarrier *et al.* 1978*b*) high EE patients showed more EDA than did low EE subjects. Tarrier *et al.* (1978*a*) emphasize '... the limitations of laboratory testing in investigating a disorder such as schizophrenia whose course may be markedly influenced by environmental and social factors'. This observation leads to the final section, which looks briefly at developments likely to influence electrodermal recording in the 1980s.

Into the 1980s

This review noted the growth of interest in peripheral mechanisms throughout the 1960s and 1970s, but there has not been a parallel increase in knowledge about central structures relevant to EDA. Venables & Christie (1973) noted the problems associated with interpretation of data from anaesthetized animal preparations, but it may be that the 1980s may perhaps offer possibilities of work with techniques such as positron emission tomography.

There has been effort toward standardization of nomenclature, evident in the 1960s and 1970s, followed by the recommendations for standardized procedure made recently: if standardization does follow, this should allow much greater comparability between laboratories than is the case at present. Another relevant aspect of standardization relates to criteria adopted for computer analysis of data: increasingly the use of computers makes possible the handling of much larger quantities of EDA data – for example Venables' 2500 subjects recorded in the Mauritian studies (Venables & Christie 1980).

Finally, one would hope that the next stage of development will produce adequate equipment for monitoring ambulant subjects, stored data from which can then be analysed by computer. The comments of Tarrier *et al.* (1978*a*) about the problems of the laboratory for psychophysiological assessment were noted; the general area of experimenter-subject-situational interaction in psychophysiology was reviewed by Christie & Todd in 1975, and by Gale & Baker in 1981; a report of an ongoing study was given to the Section of Measurement in Medicine at the Royal Society of Medicine in 1980 (Christie, unpublished). Increasingly it is being realized that there is a need to move out from the laboratory, but commercially available equipment for ambulant monitoring does still present some problems and there is obvious scope for technical development in the 1980s.

References

Angst J (1974) In: Factors in Depression. Ed. N S Kline. Raven, New York; pp 1–19

Brown C C (1967) *Psychophysiology* **4,** 260–264

Byrne D G (1975) *Australian and New Zealand Journal of Psychiatry* **9,** 181–185

Christie M J (1976*a*) In: Modern Trends in Psychosomatic Medicine. Ed. O W Hill. Butterworth, London; pp 66–85

Christie M J (1976*b*) *Medical Hypotheses* **2,** 227–229

Christie M J, Little B C & Gordon A M (1980) In: Handbook of Biological Psychiatry, vol 1, Part II. Ed. H M van Praag *et al.* Marcel Decker, New York; pp 145–182

Christie M J & Todd J L (1975) In: Research in Psychophysiology. Ed. P H Venables & M J Christie. Wiley, London; pp 50–68

Christie M J & Venables P H (1971*a*) *Psychophysiology* **8,** 525–532

Christie M J & Venables P H (1971*b*) *Psychophysiology* **8,** 779–786

Christie M J & Venables P H (1971*c*) *Japanese Journal of Physiology* **21,** 659–668

Edelberg R (1972) In: Handbook of Psychophysiology. Ed. M S Greenfield & R A Sternbach. Holt, New York; pp 367–418

Fowles D C, Christie M J, Edelberg R, Grings W W, Lykken D T & Venables P H (1981) *Psychophysiology* (in press)

Gale A & Baker S (1981) In: Foundations of Psychosomatics. Ed. M J Christie & P G Mellet. Wiley, Chichester; pp 363–384

Gruzelier J H (1978) In: Propranolol and Schizophrenia. Ed. E Roberts & P Amacher. Alan R Liss, New York; pp 99–118

Gruzelier J H, Hirsch S R, Weller M & Murphy C (1979) *Acta Psychiatrica Scandinavica* **60,** 241–248

Gruzelier J H & Yorkston N J (1978) In: The Biological Basis of Schizophrenia. Ed. W A Hemmings & G Hemmings. MTP Press, Lancaster; pp127–146

Hanssen T, Heydon T, Sundberg I, Alfredsson G, Nyback H & Wetterberg L (1980) *Archives of General Psychiatry* **37,** 685–690

Lader M (1975) In: Neuropsychopharmacology. Ed. J R Boissier *et al.* Excerpta Medica, Amsterdam; pp 168–175

Lader M (1980) In: Handbook of Biological Psychiatry, vol 1, Part II. Ed. H M van Praag *et al.* Marcel Decker, New York; pp 225–247

Lader M & Montagu J D (1962) *Journal of Neurology, Neurosurgery and Psychiatry* **25,** 126–133

Lader M H & Wing L (1966) Physiological Measures, Sedative Drugs, and Morbid Anxiety. Oxford University Press, London

Lader M H & Wing L (1969) *Journal of Psychiatric Research* **7,** 89–100

Leff J & Tarrier N (1981) In: Foundations of Psychosomatics. Ed. M J Christie & P G Mellett. Wiley, Chichester; pp 407–420

Lykken D T (1959) *Journal of Comparative and Physiological Psychology* **52,** 629–634

Lykken D T & Venables P H (1971) *Psychophysiology* **8,** 656–672

McCleary R A (1950) *Psychological Bulletin* **47,** 97–117

Myslobodsky M & Horesh N (1978) *Biological Psychology* **6,** part 2, 111–120

Neumann E & Blanton R (1970) *Psychophysiology* **6,** 453–475

Noble P J & Lader M H (1971) *Journal of Psychiatric Research* **9,** 61–69

Noble P J & Lader M H (1972) *British Journal of Psychiatry* **120,** 541–542

Siddle D A T, Turpin G, Spinks J A & Stephenson D (1980) In: Handbook of Biological Psychiatry, vol 1, Part II. Ed. H M van Praag *et al.* Marcel Decker, New York; pp 45–78

Tarchanoff J (1889) *Comptes Rendus Sociétié de Biologie* (Ser. 9) **41,** 447–451

Tarrier N, Cooke E C & Lader M H (1978*a*) *Acta Psychiatrica Scandinavica* **57,** 369–376

Tarrier N, Vaughan C, Lader M & Leff J (1978*b*) *Archives of General Psychiatry* **6,** part 3, 311–315

Tursky B & O'Connell D N (1966) *Psychophysiology* **2,** 237–240

van Praag H M, Lader M H, Rafaelsen O F, Sachar E J *ed.* (1980) Handbook of Biological Psychiatry, vol 1, Part II: Brain Mechanisms and Abnormal Behaviour. Marcel Decker, New York

Venables P H (1975) In: Clinical Applications of Psychophysiology. Ed. D C Fowles. Columbia University Press, New York; pp 106–138

Venables P H (1980) In: Handbook of Biological Psychiatry, vol 1, Part II. Ed. H M van Praag *et al.* Marcel Decker, New York; pp 79–95

Venables P H & Christie M J (1973) In: Electrodermal Activity in Psychological Research. Ed. W F Prokasy & D C Raskin. Academic Press, New York; pp 1–124

Venables P H & Christie M J (1980) In: Techniques in Psychophysiology. Ed. I Martin & P H Venables. Wiley, Chichester; pp 3–67

Venables P H & Martin I (1967*a*) In: A Manual of Psychophysiological Methods. Ed. P H Venables & I Martin. North-Holland, Amsterdam; pp 53–102

Venables P H & Martin I (1967*b*) *Psychophysiology* **3,** 302–311

Venables P H & Sayer E (1963) *British Journal of Psychology* **54,** 251–260

Walrath L C & Stern J A (1980) In: Handbook of Biological Psychiatry, vol 1, Part I. Ed. H M van Praag *et al.* Marcel Decker, New York; pp 1–43