ELECTRODERMAL RECORDINGS DURING HUMAN ORGASM

Stelios Kerassidis*

School of Health Sciences, University of Crete, Greece

Received April 1, 2009; accepted May 3, 2009

Abstract

We tested the hypothesis that palmar sweat glands activation is expressed every time a mass sympathetic activation takes place. We performed (i) eleven palmar electrodermal recordings during sexual intercourse and orgasm of one male and one female student, (ii) 4 palmar electrodermal recordings plus heart rate during sexual intercourse and orgasm of the same couple, and (iii) 3 plantar electrodermal recordings during masturbation and ejaculation of 3 male students. High palmar electrodermal activity was recorded during sexual intercourse but small during orgasm. The higher value of heart rate was recorded at the moment of orgasm. Sizeable plantar electrodermal response was recorded during ejaculation after masturbation. We concluded that the palmar sweat glands activation cannot be considered as an indiscriminate following of sympathetic discharge.

Key words: Electrodermal activity; Orgasm; Sexual intercourse; Sympathetic nervous system

INTRODUCTION

The sweating of palms can accompany fear, anxiety, tension, discomfort, exploratory and sexual behaviour; it can be triggered by novel stimuli, pain, sudden exposition to cold, emotionally loaded words, and every kind of physical or intellectual effort (Edelberg 1973, Fowles 1986). Thus, if the electrodermal activity (EDA: changes of the palmar conductivity because of sweat glands activation) can accompany almost everything, the Fowles' (1986) question whether this function is a complex and noisy manifestation of non-specific activity must be considered justified.

If the non-specificity hypothesis is true, the palmar sweat glands' activation may follow the sympathetic discharge, since sweat glands are innervated only by sympathetic nervous system fibres. It is very possible that palmar and plantar sweating is a residue of the evolution and follows mass sympathetic activation at fight and flight reactions (Cannon's theory). Obviously, if the activation of palmar-plantar sweat glands is an indiscriminate following of some type general sympathetic discharge, it carries no specific message for the central nervous system processes and the question "why is this local sweating expressed?" become meaningless.

The following findings, coming from different fields of the research, raise doubts about the indiscriminate co-activation of palmar sweat glands with sympathetic discharges: (i) the capacity for specificity of the sympathetic nervous system, found in the former experiments of Lacey (1967) and Miller (1969a, 1969b), (ii) the autonomic manifestations among anxiety disorders (Hoehn-Saric & McLeod 1988, Ost et al. 1984) which

cannot be interpreted by a mass sympathetic activation theory. In addition, Dawson et al. (1985) found that depressed patients exhibit higher tonic heart rate levels in parallel with lower tonic skin conductance levels, (iii) EDA was eliminated during the REM phase of sleep, in contrast to all other sympathetic functions which remained at the same levels as in awakening (Johnson & Lubin 1966, Lester et al. 1967, Kushniruk et al. 1985), (iv) intraneural recordings of sympathetic nerve traffic have shown that "the view of a diffusely acting system led to the term sympathetic tone is not tenable" (Wallin 1992, Wallin & Elam 1994).

However, Schaefer (1960) found that there is no specificity in the tonic sympathetic innervation of the heart, Lazarus et al. (1963) found substantial relationships between heart rate and skin conductance, and Chrousos (1992) mentioned that to a certain threshold, stressors elicit adaptive responses specific to the nature of the stressor; however, once a certain threshold has been exceeded, a systemic reaction takes place. The study of Shih et al. (1983) gives also support to the sympathetic activation theory. In this, it is suggested that subjects hyperhidrotic in their palms have an over-functioning of the sympathetic nervous fibres passing through the 2 and 3 thoracic (T2,3) ganglia, which leads to autonomic dysfunction elsewhere.

In order to check whether palmar sweating follows a mass sympathetic activation that takes place when no benefit of palmar wetting exists, we performed recordings of EDA during orgasm. Orgasm could be considered as a mass sympathetic discharge because (i) the neuronal firing in orgasm is initiated within the L1, L2

*Correspondence to: Stelios Kerassidis, email: kerassidis@nlg.gr

sympathetic ganglia (Chusid 1979, Guyton 1990) which innervate the low extremities, (ii) the augmentation of heart rate that takes place during orgasm, indicates that thoracic ganglia (which innervate the heart and the upper extremities) is also activated and, (iii) the augmentation of blood pressure is an indication of vasoconstrictor activity and/or of elevation of cardiac output that means sympathetic activation also. Additionally, in the moment of orgasm no benefit for the action of palmar wetting exist, because neither friction improvement (Adelman et al. 1975) nor abrasion prevention (Wilcott 1966) of the palms is demanded. This way, if palmar sweat glands activation takes place in the moment of orgasm, it may be considered as a following of the sympathetic discharge. If no, we have to conclude that palmar sweat glands activation has its own triggers and it is not an indiscriminate following of the sympathetic discharge. It must be mentioned, that the choice of orgasm was rather incumbent, in spite of the obvious difficulties that implies. The huge emotional interindividual differences, as response to a stimulus, did not permit any certainty that a chosen stimulus could cause high sympathetic activation without causing a reaction in which palmar wetting could be beneficial to the tested subjects.

MATERIALS AND METHODS

Participants

Four students participated to these recordings. Palmar EDA was recorded from 1 male and 1 female students (11 recordings, 7 from female and 4 from male) and HR plus EDA (4 recordings, 2 from each student) during sexual intercourse and orgasm. The EDA of the sole of the over mentioned male and of 2 more male students, was recorded during masturbation and ejaculation.

Materials

The first recordings performed at home with portable recording apparatuses. The chart recorder of Philip-Harris and an Hg battery ($E=1.35\text{ V}$) as an electric source were used. The applied voltage on the students' fingers was about $0.5\text{--}1\text{ V}$ and the current was less than 10 microA cm^2 . The electrodes displayed a surface of 1 cm^2 and were covered by silver-silver chloride. A cream prepared according to the published recommendations of Fowles et al. (1981) based on neutral ointment cream mixed with saline (2:1) was used as the electrolytic media. In the other recordings, a Beckman R 511A was used for single EDA recordings and for simultaneous recordings of EDA and heart rate (HR). For these EDA recordings, a stabilized power supply of 0.5 V was used.

Procedure

The first 11 recordings of the EDA of the couple of students were performed in their house, in a long period of time, at different moments of a day, by themselves.

Electrodes were placed in one's, out of the two participants, first phalanxes of the fingers of the left palm. The person with the recording electrodes, was lied in bed and held the arm with the electrodes still during the sexual intercourse; she/he removed one out of the two electrodes, as event-marker, after orgasm.

Four additional recordings of palmar EDA and HR, during the sexual intercourse of the same couple, were performed. The HR recording was used as an indicator of sympathetic activity. For these recordings, Beckman recorder, stabilized power supply, and auxiliary instruments were carried in students' house. The recording apparatuses were more precise and carefully adjusted before every recording. The signals for HR recordings were carried through electrodes attached to the left and right forearm and to the chest (ground). At preparatory sessions, it was completely confirmed that no kind of interaction between EDA and HR recordings occurred. The person with the recording electrodes and wires was lied in bed and held a passive attitude during the sexual intercourse and gave immediately after orgasm the Beckman's event-marker signal.

Three recordings of plantar EDA during masturbation and orgasm of the 3 male students were performed in the laboratory. A plantar sweat glands activation during orgasm was possible, because neuronal firing in orgasm is initiated within the L1, L2 sympathetic ganglia which innervate the low extremities. All students, before recording, placed their left sole in a vessel with water, for 5 min. This preparation aimed to wet the very thick plantar stratum corneum and reduce its high resistance which otherwise could cause significant unsensitivity of the recording (Venables & Christie 1973, Fowles 1986, Fowles et al. 1981). Electrodes were placed in the middle of the superficial layer of the left sole, in a distance of 2 cm to each other and a sock was worn over the electrodes. Care had been paid for the electrodes to not be pushed when the student stood up. After this, the student entered in a dim light and low ambient noise room with an armchair. The recording apparatuses were out of this room. The plantar EDA was recorded during the masturbation and ejaculation. The student, immediately after ejaculation, gave the Beckman's event-marker signal.

RESULTS

The first 11 recordings of sexual intercourse showed that during orgasm, of both participants, the EDA was small or negligible, the skin conductance level (SCL) of the palm was decreased, and in the case that a SCR occurred it was small in relation to the great SCRs during sexual intercourse. Huge SCRs were recorded especially at moments of discomfort due to real life problems. The values of conductivity and its alterations cannot be precisely estimated, because they related with the occasional zero adjustments, which were performed by the

no recorded student, analogously to the SCL of his/her recorded partner, before their intercourse.

The finding that EDA during orgasm was small in relation to EDA of other moments during sexual intercourse was very important. However, it could not undoubtedly be interpreted as a demonstration that EDA does not indiscriminately follow mass sympathetic activation. It could not be excluded that the higher SCRs recorded in discomfort during sexual intercourse masked the EDA during orgasm.

The simultaneous recording of EDA and HR, of the same couple during sexual intercourse and orgasm, without real life problems, confirmed the previous findings that the EDA during orgasm was small or negligible. However, the high value of HR recorded at the moment of orgasm. In the recordings of male student, the HR was elevated to 125 pulses min^{-1} during orgasm (both times) but his EDA was negligible (during both orgasm and sexual intercourse). In the recordings of the female student the HR was elevated to about 100 pulses min^{-1} during orgasm (both times) and only a SCR of 0,2 microS was recorded the first time (Fig. 2). During the time of sexual intercourse the HR of both students was varied (about 60-83 pulses min^{-1} for the female, and 60-100 pulses min^{-1} for the male), but was always under that of the moment of orgasm. High EDA was recorded during sexual intercourse for the female student. Many SCRs, up to 0,6 microS, were recorded.

A sizeable sweat glands activation of the soles of all the male students was recorded at the onset of ejaculation which followed the masturbation. SCRs of 1.66, 0.60 and 0.46 microS occurred at 20, 15 and 35 sec respectively for each one of the students, before the end of ejaculation and these plantar SCRs were the biggest, or one of the biggest recorded during the masturbation of every student.

DISCUSSION

From the first recordings during sexual intercourse and orgasm it was found that the palmar sweat glands activation was weak during orgasm, for both male and female students, but it was strong, especially at moments of discomfort, during sexual intercourse. The recordings of EDA plus HR during sexual intercourse and orgasm confirmed that the sweat glands activation was weak during orgasm and showed that the recorded HR was maximal at this time. The male plantar EDA recordings reveal that at the onset of ejaculation a considerable plantar EDR occurred.

After the end of the first recordings, we realized that their performance in a house and not in a laboratory gave a significant advantage. The students faced real life problems which would had been excluded in a laboratory procedure. This way it was revealed that palmar sweat glands activation was enormous at moments of discomfort but not during orgasm. This finding answered

the posed main question of this work: the mass sympathetic activation which occurred during orgasm does not activate the sweat glands of the palm. However there was some doubt yet. The EDA during orgasm could had been masked by the EDA which was caused during sexual intercourse, in the face of real life problems. In addition, the information about the physiology of human orgasm, and the sympathetic activation during this, is not complete, because of the limited number of the studies on this issue. In other words, the activation of the thoracic ganglia (T2,3) which innervate the sweat glands of the palms may be weak during orgasm. The recording of HR, simultaneously with EDA, was used as an estimator of the degree of activation of the thoracic sympathetic ganglia.

In these recordings (EDA plus HR) the EDA during sexual intercourse was reduced, probably because of the exclusion of real life's problems and the taking of a more passive attitude of the recorded student. The recorded EDA during orgasm remained weak, although any masking effect was excluded in these recordings and the HR reached its maximum value. These findings can take only one explanation: the mass sympathetic activation which indeed takes place at the moment of orgasm does not activate the sweat glands of the palm. This explanation is not bizarre if the sympathetic nervous system is capable to express specialized activation. As Wallin (1992) mentioned "... the degree of sympathetic differentiation is even greater than previously known; it occurs not only between different tissues but may also occur between different regions of the same tissue. Probably such differences may originate both at spinal and supraspinal levels." In the palmar sweat glands activation, structures of all the levels of the central nervous system are involved (Ladpli & Wang 1960, Isamat 1961, Wilcott 1969, Wilcott & Bradley 1970, Venables & Christie 1973, Edelberg 1973, Delerm et al. 1982, Fowles 1986, Sato et al. 1989, Weitkunat et al. 1990, Venables 1991, Turkstra 1995). Probably, highest centres control the palmar sweat glands activation unless otherwise prevented (Venables 1991) and the whole expression of the function is more complicate than the simple scheme of the general sympathetic arousal view implies.

The sizeable sweat glands activation of the sole of males, during orgasm at the ejaculation, probably shows that the neuronal firing of the L1, L2 sympathetic ganglia caused a plantar sweat glands activation. This finding indicate that the plantar sweat glands activation during orgasm was different of that of the palm. This difference could be attributed to the factors: (i) the male students, in order to ejaculate after masturbation, had to raise from the armchair. This raising could cause an elevation of sympathetic tone in the low extremities, including the plantar sweat glands activation, (ii) the ejaculation after masturbation in a laboratory room may

cause discomfort which could then cause the recording SCR, (iii) the ability for specificity of sympathetic nervous system may be higher in upper than in low extremities. It means that excitatory pulses from thoracic sympathetic ganglia may be directed to the heart and vasoconstrictor muscles and not to the sweat glands of palms, but the lumbar ganglia firing may cause a more indiscreetly activation of the organs and tissues which receive innervation from that.

Undoubtedly, the sympathetic nervous system may "acts as a unit: faster heart rate, vasoconstriction, increased blood sugar, discharge of adrenaline and erection of the hairs", in "fight" and "flight" reactions (Canon's theory). Besides, the anatomical unity of sympathetic nervous system must serves the functional purpose of mass activation of the innervated tissues and organs by that. But the findings show that the sympathetic nervous system has also the capability of specificity (probably, a later fabrication of evolution).

The main finding of this work is that the mass and high sympathetic activation which takes place in orgasm can exclude palmar sweat glands. In a previous work we have found that palmar and plantar sweating do not directly participate to thermoregulation (Kerassidis 1994). In the present work, we conclude that palmar EDA cannot be considered as an indiscriminate following of sympathetic discharge.

REFERENCES

Adelman, S., Taylor, C.R. & Meglung, N.C. (1975). Sweating on paws and palms: what is its function? *The American journal of psychology* 229, 1400-1402.

Chusid, J.G. (1979). The Autonomic Nervous System. In: *Correlative neuroanatomy & functional neurology*. LANGE Medical Publications, Los Altos, California.

Chrousos, G.P. (1992). Regulation and dysregulation of the hypothalamic-pituitary-adrenal axis. *Endocrinology and metabolism clinics of North America*, 21, 833-858.

Delerm, B., Delsaut, M. & Roy, J.C. (1982). Mesencephalic and bulbar reticular control of skin potential responses in kittens. *Experimental brain research*, 46, 209-214.

Dawson, M.E., Schell, A.M., Bratten, J.R. & Catania, J.J. (1985). Diagnostic utility of autonomic measures for major depressive disorders. *Psychiatry Research* 15, 261-270.

Edelberg, R. (1973). Mechanisms of electrodermal adaptations for locomotion, manipulation, or defence. In: E. Stellar & J. M. Sprague (eds) *Progress in physiological psychology*, 5, 155-209. Academic Press, New York.

Fowles, D.C. (1986). The eccrine system and electrodermal activity. In: M. G. H. Coles, E. Donchin & S. W. Porges (eds) *Psychophysiology: Systems, Processes and Applications*, 51-96. The Guilford Press, New York.

Fowles, D.C., Christie, M.J., Edelberg, R., Grings, W.W., Lykken, D.T. & Venables, P.H. (1981). Publication recommendations for electrodermal measurements. *Psychophysiology* 18, 232-239.

Guyton, A.C. (1990). Reproductive functions of man. In: *Human Physiology and Mechanisms of Disease*, 716-727. Litsas Medical Publications, Athens.

Hoehn-Saric, R., & McLeod, D.R. (1988). The peripheral sympathetic nervous system. Its role in normal and pathological anxiety. *The Psychiatric clinics of North America*, 11 (2), 375-386.

Isamat, F. (1961). Galvanic skin response from stimulation of limbic cortex. *Journal of neurophysiology*, 24, 176-181.

Johnson, L.C., & Lubin, A. (1966). Spontaneous electrodermal activity during waking and sleeping. *Psychophysiology*, 3, 8-17.

Kerassidis, S. (1994). Is palmar and plantar sweating thermoregulatory? *Acta physiologica Scandinavica*, 152, 259-263.

Kushniruk, A., Rustenburg, J. & Ogilvie, R. (1985). Psychological correlates of electrodermal activity during REM sleep. *Sleep*, 8, 146-154.

Lacey, J.I. (1967). Somatic response patterning and stress: Some revisions of activation theory. In: M.H. Appley & R. Trumbull (eds) *Psychological Stress (Issues in research)*, 15-42. Appleton-Century-Crofts, New York.

Ladpli, R. & Wang, G.H. (1960). Spontaneous variations of skin potentials in footpads of normal, striatal and spinal cats. *Journal of neurophysiology*, 23, 448-452.

Lazarus, R., Speisman, J.C. & Mordkoff, A.M. (1963). The relationship between autonomic indicators of psychological stress: heart rate and skin conductance. *Psychosomatic medicine*, 25, 19-30.

Lester, B.K., Burch, N.R. & Dosett, R.C. (1967). Nocturnal EEG-GSR profiles: the influence of presleep states. *Psychophysiology* 3:238-248.

Miller, N.E. (1969a). Learning of visceral and glandular responses. *Science* 163, 434-445.

Miller, N.E. (1969b). Psychosomatic effects of specific types of training. *Annals of the New York Academy of Sciences* 159, 1025-1040.

Ost, L., Sterner, U. & Lindahl, I. (1984). Physiological responses in blood phobics. *Behaviour research and therapy*, 22, 109-117.

Sato, K.T., Kang, W.H., Saga, K. & Sato, K.T. (1989). Biology of sweat glands and their disorders. 1. Normal sweat gland function. *Journal of the American Academy of Dermatology*, 20, 537-563.

Schaefer, H. (1960). Central control of cardiac function. *Physiological reviews*, 4, 213-231.

Shih, C.J., Wu, J.J. & Lin, M.T. (1983). Autonomic dysfunction in palmar hyperhidrosis. *Journal of the autonomic nervous system* 8, 33-43.

Turkstra, L.S. (1995). Electrodermal response and outcome from severe brain injury. *Brain injury*, 9, 61-80.

Venables, P.H. (1991). Autonomic activity. *Annals of the New York Academy of Sciences*, 620, 191-207.

Venables, P.H., & Christie, M.J. (1973). Mechanisms, Instructions, Recording Techniques, and Quantification of Responses. In: W.F. Prokasy & D.C. Raskin (eds) *Electrodermal activity in psychological research*, 1-123. The Academic Press, New York.

Wallin, B.G. (1992). Intraneural recordings of normal and abnormal sympathetic activity in man. In: R. Bannister & C.J. Mathias (eds) *Autonomic Failure. A textbook of clinical disorders of the autonomic nervous system*, 359-377. Oxford Medical Publications, Oxford.

- Wallin, B.G. & Elam, M. (1994). Insights from intraneural recordings of sympathetic nerve traffic in humans. *News in Physiological Sciences*, 9, 203-207.
- Weitkunat, R., Buhner, M. & Sparrer, B. (1990). Cortical initiation of phasic electrodermal activity. *International journal of psychophysiology*, 9, 303-314.
- Wilcott, R.C. (1966). Adaptive value of arousal sweating and the epidermal mechanism related to skin potential and skin resistance. *Psychophysiology* 2, 249-262.
- Wilcott, R.C. (1969). Electrical stimulation of the anterior cortex and skin- potential responses in the cat. *Journal of comparative and physiological psychology*, 69, 465-472.
- Wilcott, R.C. & Bradley, H.H. (1970). Low-frequency electrical stimulation of the cat's anterior cortex and inhibition of skin potential responses. *Journal of comparative and physiological psychology*, 72, 351-355.