

**On The Methodological Profile of GSR Studies in the light of the Recent Advances  
Obtained in the Knowledge of Its Neurological Correlates**

**Rich Norman<sup>(1)</sup>, Leonardo Mendolicchio<sup>(1,2)</sup>, Cengiz Mordeniz<sup>(1,3)</sup>, Enrico  
Pieranageli<sup>(1)</sup>, Paolo Pannarale<sup>(1)</sup>, Franco Orsucci<sup>(4)</sup>, Elio Conte<sup>(1)</sup>**

<sup>(1)</sup>School of Advanced International Studies on Applied Theoretical and Non Linear Methodologies of Physics, Bari Italy

<sup>(2)</sup>Villa Miralago, Clinic for the therapy of the eating disorders, Varese, Italy;

<sup>(3)</sup>Namik Kemal Üniversitesi and Tekirdağ Laboratory of Psychophysiology, Turkey

<sup>(4)</sup>University College London. England

**Abstract:**

The primary focus of this report is to advance in depth study of the neurological correlates of GSR with the intention, on one hand, to clarify the neurological patterns produced during recording, monitoring and analysis of GSR, and on the other hand, to delineate the methodological profile that absolutely must be followed during the recording of this GSR signal.

In this context, the present relation formulates the methodological criteria that the operator must follow during the recording, monitoring, and analysis of the GSR signal. The relation defines and prioritizes GSR signal evaluation under these different conditions: the GSR should be recorded at rest and affected by different stimuli, specifically: visual stimuli such as sudden bursts of light as well as images modified to create multi-level energetic allocations, that is, soft images but also more potent images able to induce strong visual perturbation, linguistic stimuli including words and phrases across a spectrum of energetic/affective presentation (neutral, soft, strong and shocking), tactile stimuli (again soft, sudden, and of short duration as well as strong, shocking and continued [of consistent duration]), addressing different parts of the body throughout the session. Auditory stimulus and olfactory stimulus will be included in the test procedures. All the stimuli may be repeated several times in order to evaluate habituation, memory, recall and other important neurological functions. Other stimuli may be used to foster memory load and stress, as well as conflicting semantic stimuli, respectively, to be represented in resultant numerical calculations reflecting mental performance, evidenced through Stroop effects, and every other cognitive demonstration. It is important to keep in mind that the most difficult step in maintaining accurate GSR monitoring and analysis, is to insure proper subjective evaluation of the manner in which a subject responds to different levels of visual input, sounds, words, language strings (phrases) and tactile stimuli, in the context of habituation, meaning, the performance of subject response to repeated identical subsequent stimuli, or mnemonic recall of previous stimuli. For proper evaluation of subjective parameters, that is **calibration**, it is necessary to explore a great variety of stimuli differentiated as previously described. The term calibration here refers to the fact that each subject has a subjective behavioral component in their GSR. Therefore, it becomes of fundamental importance to identify and estimate the basic parameters in his/her personal context in absolute values as well as in relation to standards, eventually established, tabulated and recorded from normal subjects constituting a large GSR database obtained by monitoring a suitable population of subjects.

Consequently, the various responses to a defined stimuli have a particular, fundamental role in GSR monitoring that relates to the methodological profile since, under different conditions of stimulus, the operator may calibrate the recorded GSR subjective profile in terms of the basic parameters derived across population that characterize the phase of the GSR signal, such as Habituation, Latency Time under stimuli, Reaction Times, Peak Values subsequent to stimuli, Half Recovery Time following the peak amplitude of the phase, yielding a baseline value resultant of different stimuli, within the neurological correlates identified across a sampled population, allowing a correct, relative subjective analysis of the tone and of the phase of the GSR signal by Linear and Non Linear methodologies. Toward this end it is important to recall that GSR is an intrinsically non linear electrophysiological signal. Therefore its recording enables subsequent analysis based from one side on the fixation and estimation of basic established parameters such as latency, reaction times, peak amplitude, half recovery time, baseline compared at rest and after various stimuli as specified, and from the other, relating that baseline to aspects of the time series for the tone and phase *of each subject* subjected to analysis by non linear methodologies. In this way we may ascertain the real inner structure of the complex electrophysiological signal.

**Keywords:** protocol running of GSR monitoring, analysis of a GSR signal, Linear and non Linear Methods in GSR analysis, Neurological correlates of the GSR

## PROFILE OF THE AUTHORS

**Rich Norman**, Researcher in Neurology and Psychology at the School of Advanced International Studies on Applied Theoretical and Non Linear Methodologies of Physics, Bari, Italy

**Leonardo Mendolicchio**, psychiatrist, Director Villa Miralago, Clinic for the therapy of the eating disorders, Varese, Italy;

**CengizMordeniz**, Associated professor of anesthesiology, Namık Kemal Üniversitesi and Tekirdağ Laboratory of Psychophysiology, Turkey

**Enrico Pierangeli**, Neurosurgeon and former professor of neurosurgery University of Bari- Italy

**Paolo Pannarale**, neurologist and former Hospital Neurologist, Bari-Italy.

**Elio Conte**, Director School of Advanced International Studies on Applied Theoretical and Non Linear Methodologies of Physics, Bari, Italy

**Franco Orsucci**, psychiatrist, professor of psychiatry; University College London; Lead Consultant, CPFT – Cambridge University Health Partners; Director, Institute for Complexity Studies of Rome

corresponding author: [elio.conte@fastwebnet.it](mailto:elio.conte@fastwebnet.it)

---

## SCIENTIFIC RELATION

### **The GSR, history and interconnected affective/perceptual systemic modus operandi**

Abbreviated general historical account:

Galvanic Skin Response (GSR) is demonstration of the continuous dynamic variations of the electrical properties of the skin. A great many terms have been derived from various approaches both passive and active, such as: skin conductance, galvanic skin response (GSR), electrodermal response (EDR), psychogalvanic reflex (PGR), skin conductance

response (SCR), sympathetic skin response (SSR) and skin conductance level (SCL). Often the term **electrodermal activity (EDA)** is now ascribed to the phenomenon (although Skin Conductance and the associated Skin Conductance Response are also common in modern literature) (Critchley, 2002). For the remainder of this essay, the more traditional term **GSR** will be used in conjunction with Skin Conductance (**SC**) and Skin Conductance Response (**SCR**).

The traditional view holds that increased sweat gland activity which is a function of the sympathetic branch of the autonomic nervous system, increases skin conduction, and allows measurement of said conduction to function as a measure of systemic arousal. Du Bois-Reymond in the mid 1800s first observed skin conductivity through emersion of limbs in zinc sulfate solution, and observation of muscular response affected by current. In 1878, Hermann and Luchsinger demonstrated the involvement of the sweat glands, and Hermann later derived increased conductance effects from the palms and hands supporting the role of perspiration in the process. Correlations between affect and GSR were found in the late 1870s and 1880s, by Vigouroux and Féré respectively. In 1889 IvaneTarkhnishvili developed a functional meter to demonstrate the effects as they unfolded in time. Modern scientific study of the phenomenon beginning in the early 1900s is partly credited to Jung, and specific reference can be found in his seminal work: *Studies in Word Analysis*, published in 1906. [Information condensed from: [https://en.wikipedia.org/wiki/Electrodermal\\_activity](https://en.wikipedia.org/wiki/Electrodermal_activity)]

Emotional arousal in a nonspecific sense is tied to increased sympathetic system activity. Electrodermal resistance, and also (passive) electrodermal potential (associated with perspiration and blood flow), in their combination create the particular dynamic result. The response of skin and muscle to internal and external stimuli, can be measured as variations in the range of microsiemens ( $\mu\text{S}$ ) conductance discrepancies over time. The esteemed Hugo D. Critchley has said: "EDA is a sensitive psychophysiological index of changes in autonomic sympathetic arousal that are integrated with emotional and cognitive states." (Critchley, 2002p. 132).GSR is still used extensively today (Ogorevc et al. 2013).

**Means and locus of specific applicability and basic signal parameters:** [condensed information retrieved from: [http://www.psychlab.com/SC\\_explained.html](http://www.psychlab.com/SC_explained.html)]

GSR/Skin-Conductance (SC) is typically measured with silver or silver chloride electrodes placed on the medial phalanx of the index and middle fingers secured by double sided electrode collars in conjunction with non-saline jell. The responses are a measure of subject (autonomic/sympathetic) arousal. The medial phalange of the fingers or palm are typical electrode application points, or in rare cases the heel of the foot is used.

To determine SC, low voltage ( $\sim 0.5\text{V}$ ) is sent across properly positioned electrodes to

measure conductance.

Within general GSR indications of sympathetic systemic arousal then, we have detailed response components, onset, rise time, peak, and exponential decay. Although the relative level of conductance alteration itself provides only general information concerning systemic arousal, the component dynamics may be interactively interpreted as to both their linear and nonlinear attributes to ascertain specific information about detailed affective response (Wang, Liu and Yang, 2014; Karthikeyan, Murugappan, and Yaacob, 2013).

### **Neuroanatomical circuit pathways and structures affecting GSR in particular component stimulus responses to internal and external perception:**

Jung referred to GSR as a sort of “looking glass” into the unconscious (Brown, 1977). As every psychologist is aware, that statement implies GSR contains much more affective information than just general arousal, in the context of fight or flight, as is the reductionist view of sympathetic arousal. Indeed, the full plethora of hidden (unconscious) physiological affective instantiations which create the valence of perceived reality must be involved. Indeed, this is so.

Let us first delineate the relation between GSR and sympathetic connectivity so as to determine if GSR is indeed an accurate measure of autonomic sympathetic systemic response. The sweat glands *are exclusively innervated by the sympathetic nervous system*, and are activated via postganglionic sudomotor fibers (Benedek and Kaernbach 2010). A collective temporal grouping of single fiber triggering spikes is called a nerve burst, which corresponds to a single SCR. SCR amplitude then, may be taken as a measure of sympathetic activity. The SC time series demonstrates: a. slowly changing tonic activity ie. skin conductance level, and b. rapidly changing phasic dynamics ie. SCRs. The train of SCRs present as a series of superpositions, the slowly declining portion of the last SCR overlapping the next SCR. Clearly, *GSR is a highly exclusive window into the activational state of the sympathetic nervous system*, although at this point in it is not exactly straight forward as to how an indistinct phasic component might best be extracted from the superposition (Boucsein,1992). Suitable methods of phasic analysis will be presented below.

Next, we must observe the relationship to sensory processes and input. The prolific innervations of the sympathetic nervous system, extend to nearly every organ and bodily structure.

### **Visual stimulus:**

The lateral geniculate nucleus of the thalamus receives information from the retina and

distributes it to area V1.

In the most general sense, from area V1: the superior longitudinal fasciculus includes axons terminating in the posterior parietal cortex, where object location ("where" information) is derived, and, the inferior longitudinal fasciculus contains axons terminating in the inferotemporal cortex, a region implying object identification ("what" information) (Gazzaniga et al., 2009, p. 209). Initial lesioning experiments with monkeys implied this reasoning (Pohl, 1973).

Processing, is refined via the progressive signal chain of visual areas, each with more detailed functionality. the ventral intraparietal sulcus (VIP) is where visual and somatosensory information are integrated. Then, in Brodmann Area 20 (*Inferior temporal, Fusiform and Parahippocampal gyri*) and other areas, the processing is recombined into integrated perceptual wholes (Gerlach 2002). The medial temporal lobe, along with limbic connectivities may well provide integrative information and directly aid in perceptual processing apart from the commonly acknowledged role as an exclusive memory system (Lee and Rudebeck 2010; Murray and Mishkin 1998). Object recognition is dependent on the Rhinal cortex (which is part of the medial temporal lobe) (Murray 2000).

In vision, the sympathetic nervous system dilates the pupil and has other known connectivities.

- a. mydriasis- contract pupillary dilator muscle (alpha 1 receptor)
- b. contract superior tarsal muscle to hold eyelid open (alpha 1 receptor)
- c. Relax ciliary muscle for distant vision ( $\beta_2$  receptors)
- d. Enhance aqueous humor formation ( $\beta_2$  receptors)
- e. Inhibit aqueous humor formation (alpha 2 receptors)

[Information Retrieved from: <http://www.uruq.com/download/3159.html>]

### **Tactile Stimulus:**

“There are different types of skin receptors that respond to and transmit stimuli. Pacinian corpuscles and free nerve endings are found in both hairless and hairy skin. The Pacinian corpuscles are skin receptors that receive stimuli associated with high frequency vibrations, while free nerve endings receive pain stimuli. Meanwhile, the Meissner’s corpuscles are exclusive in hair skin and respond to low frequency vibrations and pressure stimuli. Other touch receptors include Merkel’s disks (pressure) and Ruffini’s corpuscles (low frequency vibrations).

The sensory information from the receptors is transmitted through either one of the three systems: (1) dorsal-column-medial lemniscal system (touch and proprioception), (2) anterolateral system (pain and temperature), or (3) spinocerebellar system (proprioception) towards the dorsal columns. From there, the input is transferred to the thalamus, which then relays the information to the primary somatosensory cortex for processing.”

[Retrieved from: <https://explorable.com/neural-pathways-of-smell-taste-and-touch>]

The sympathetic nervous system deeply affects skin receptors and smooth muscle structures and is intimately tied to the sense of touch, as well as exclusively innervating sweat glands as mentioned above (Efes, 1992).

### **Sensory signaling in Olfaction**

"Information is conducted from the olfactory bulbs by the **lateral olfactory tract** to the **primary olfactory cortex**. From there, it goes to the **thalamus (mediodorsal nucleus)** and on to the **orbito-frontal cortex** where conscious smell perception occurs. Primates also have a pathway that runs from the thalamus to the **amygdala** which is part of the **limbic system**, and then on to the **hypothalamus**. The limbic system is involved in the perception of emotions and is responsible for the "affective" component of smell. This may explain why scents can engender strong emotions and/or take us back to previous experiences." [Retrieved from: <http://www.ucalgary.ca/pip369/mod8/smell/pathways>]

Sympathetic nervous system in olfaction: "The olfactory epithelium is extensively innervated by sympathetic nerve endings, which release norepinephrine, and parasympathetic nerve endings, which release acetylcholine. Because olfactory sensory neurons have adrenergic and muscarinic receptors in addition to odorant receptors, autonomic stimulation can modulate the responses of olfactory sensory neurons to odorants." (Hall, 2011).

### **The sense of taste:**

"The tongue contains small bumps called papillae, within or near which taste buds are situated. In the tongue's taste buds, the taste receptors receive sensory input via two important mechanisms – depolarization and neurotransmitter release. Intake of salty foods leads more sodium ions to enter the receptor, causing the said mechanisms. The same is true with intake of sour foods (hydrogen ions) and sweet foods (sugar molecules), both of which result to the closing of K<sup>+</sup> channels upon their entry.

From the axons of the taste receptors, the sensory information is transferred to the three taste pathways via the branches of cranial nerves VII, IX and X. The chorda tympani of CN VII (facial nerve) carries the taste sensory input from the tongue's anterior two-thirds. Then, the rest of the taste sensations from the throat, palate and posterior tongue are transmitted by the branches of CN IX (glossopharyngeal nerve) and CN X (vagus nerve). From these cranial nerves, taste sensory input travels through the nerve fiber synapses to the solitary tract, the ventral posteromedial thalamic nuclei, and the thalamus. In these three locations, there are clustered neurons which respond to the same taste (sweet, sour, salty or bitter). The thalamus relays the information to the primary gustatory cortex located in the

somatosensory cortex. The primary gustatory cortex is where the perception of a particular taste is processed." [Retrieved from: <https://explorable.com/neural-pathways-of-smell-taste-and-touch>]

Sympathetic nervous system and taste: sympathetic responses delineate between healthy and unhealthy food choices and quantities (Rousmans, 2000).

### **The sense of hearing**

“The inner ear consists of the cochlea and vestibular apparatus. The cochlea is a component of osseous labyrinth that contains perilymph and the cochlear duct. The cochlear duct is a component of membranous labyrinth and contains endolymph. The cochlea makes 3.25 turns in the dog (2.5 in man) around a core of bone (called the modiolus) through which the cochlear nerve passes. The entire complex resembles a snail’s shell (whence the term cochlea is derived). Within the cochlea, the cochlear duct (scala media) separates two perilymph chambers: the scalavestibuli, which contacts the oval window membrane, and the scala tympani, which contacts the round window membrane. Perilymph can flow from one scala to the other through an opening (helicotrema) at the apex of the cochlea. The helicotrema is non-functional with respect to the physiology of hearing, it merely precludes perilymph stagnation.” [Retrieved from: <http://vanat.cvm.umn.edu/NeuroLectPDFs/LectAuditorySys.pdf>]

Sympathetic nervous system in hearing: The cochlea is innervated by the sympathetic nerve fibers. Sympathetic functioning levels appear to mediate trauma (Wada, 1999).

And of music: specific features of music (e.g., its beat, tempo, or pitch level) trigger neurophysiological, psychophysiological, emotional, and behavioral responses. . . . continued work within these different paradigms may reveal a common finding: that the ANS serves as the final common pathway by which music exerts a therapeutic effect on health and disease (Ellis and Thayer, 2010).

### **Language: word valence, phrases and affective strength and GSR:**

Response to words is a “unidirectional” measure, meaning it reflects only the strength of an attitude. Smith in 1922 discovered that GSR deflections are associated with affectively laden topics. Mc Curdy in 1950 disclosed after reviewing the literature that a very significant +.75 correlation was found between the size of an electrodermal response and affective vividness. Affective directionality was not indicated, only response strength independent of positive or negative valence (Petty et al., 2014).

In (Barry, 1980) we read that the response of subjects to words is deeply related to the

personality structure of the subject being examined. Affectively inhibited subjects, display less response.

Is there more depth to be had in ascertaining affective response and internal state? Is there a better approach with more information gained? We believe there is.

### **A new model:**

Next we may consider a specific model of sympathetic and parasympathetic interactivity, "A topological model of biofeedback based on catecholamine interactions" (Basak et al. 2005). A model is presented in which the subject's condition may be assessed within the context of biofeedback as represented in mathematical analysis of the component response structures comprising GSR, which may be seen as dissipative or conservative, allowing internal subject states to be quantified. Dissipative response structure evidences changing dynamics by way of diminishment within the *transduction phase*, which will be evidenced as a declining exponential function. A conservative system response by contrast is characterized by rising phases, which are hypothesized to be due to sustained levels of catecholamines. Through the mutual innervation of sympathetic and parasympathetic systems within the hypothalamus, an effect is advanced where sympathetic activity is mediated by parasympathetic interactivity under the particular moniker of *parasympathetic stimulation*. Transduction phase analysis is proposed to derive correlations with pathogenic conditions such as migraine and psychosomatic digestive disorders. Negative feedback curtails excessive response unless a pathological condition is in evidence. Phasic components have a residual factor, also. Residual homeostatic output level,  $\Delta V$ , is correlated with GSR. This correlation is understood in the context of long lasting residual homeostatic response associated with "sustained catecholamine action." The familiar balanced interdependence of sympathetic and parasympathetic branches of autonomic functioning in the context of adrenergic and cholinergic mediation is detailed and described in this paper. We see how "noradrenergic enhancement is diminished as cholinergic neurotransmission becomes established."

So through analysis of conservation in systemic expression (conservative dynamics increasing transduction effects) vs. dissipative dynamism (reduction in transduction effects) as understood in the context of adrenergic and cholinergic receptor activity, a mathematical assessment of pathological internal state associated dynamics is possible. Systemic input may be auditory, visual or tactile. It is demonstrated how subject response to stimuli within the context of biofeedback may thus provide a graded pathologic systemic metric.

**This proposed theoretic methodology may then be generally applied as follows:** A recording of the tone and phase of the GSR signal is compared with the subject at rest and under external stimulation. Sensory modalities include those of sight (utilizing various strengths and visual time durations), tactile stimulus (of various durations, somatic targets, distributions and strengths), phrases and words (of various affective presentations from delicate to shocking) and sounds. Each stimulus type and specifically mediated presentation then, can be assessed and attributed to specific neurological dynamics

inferred from the revealed GSR pattern analysis. To determine the responses properly in their subjective specificity, a clear reference value must be first established. This will yield the patterning to be analyzed, once framed in a proper referential context. This is accomplished through tripartite analysis of: a. latency time, of b. peak amplitude and of c. half-recovery time, which are of course essential. Recording, monitoring and analysis of the habituation factor is also required. However, a complete analysis will not stop short of the *deeper* facts revealed through direct use of non linear-chaotic-deterministic tools. Those may allow the realization, of Jung's vision. We must now progress toward that end, and gain a mid-level anatomical analysis to go with what we have derived, before completing the picture.

### **Anatomical activations associated with GSR:**

Now that we have articulated a suitable framework within which GSR signal interpretation may be instituted, and then advanced a general theoretical approach to experimental construction within said framework, it remains for us to demonstrate the proposed patterned anatomical specificity activated in association with GSR. Can active brain states across time and their corresponding anatomical structures be inferred from GSR to a deeper level of connectivity, allowing the autonomic/sympathetic measure of GSR a window into more complex organizational levels of cognition?

In (Critchley, 2002) we find the next level of connectivity into the deeper system is indeed available to articulate. Here, the seemingly peripheral sympathetic system, which we now know informs us also by way of its intra-connected balance within the autonomic whole of homeostatic functioning, also contains demonstrable correlations to deeper levels of affective and attentional anatomical specificity, just as our general experimental outline would require (please see the original article for inter-text citations embedded within the following quotation):

“Within the hypothalamus and brainstem, there exists a discrete set of brain regions involved in homeostatic control of sympathetic arousal that controls peripheral EDA via ipsilateral descending connections to the spinal cord. The autoregulatory functions of these brain regions are dynamically modulated to adapt bodily arousal to meet the demands of behavior. It is this second-order modulation, manifest in discrete peaks of electrodermal activity (SCR, GSR), that has been the basis of the application of EDA to psychophysiological research.

“Higher” brain regions that influence EDA include the ventromedialprefrontalcortex, anteriorcingulate, parietal lobe, insula, amygdala, and dorsolateralprefrontal cortex. There are distinct anatomical contributions to the contextual control of EDA: The ventromedial prefrontal cortex and amygdala are associated with EDA responses

during motivational behavior, but they differ in their specific roles. Thus, the ventromedial prefrontal cortex is involved in anticipatory EDA responses, whereas the amygdala is implicated in EDA responses to learned associations between stimuli and reinforcement (e.g., during fear conditioning). There is also evidence suggesting that a primary role for the anterior cingulate cortex is to integrate autonomic bodily states with behavior (Critchley and others 2000a, 2001a, 2001b). Thus, anterior cingulate activity varies with EDA responses to emotive stimuli (Fredrikson and others 1998), and anticipatory EDA in the context of risk (Critchley and others 2001a), and is also associated with volitional modulation of EDA responses (Critchley and others 2001b). The interaction between EDA-indexed arousal and attention is perhaps of more general importance. A critical area for visual attention is the right parietal cortex. Lesions here not only impair attention but also diminish EDA responses (Tranel and Damasio 1994; Zahn and others 1999; Tranel 2000), and right parietal cortex activity covaries with EDA (Critchley and others 2000b). These findings suggest commonality in the neuroanatomy supporting both attention and bodily arousal, consistent with the use of EDA as an index of attention and the observation that attention is directed toward stimuli that evoke arousal (e.g., Lane and others 1999).”(Critchley, 2002).

Now that basic attentional, affective and anatomical connectivities represented in GSR have been presented, we need but go one step deeper to ascertain the complete picture, so as to determine the full measure of systemic information which might be derived, before suggesting the mathematics to accomplish these ends. We conclude: systemic imbalance at all levels of psychological and neuroanatomical depth may be ascertained through further development of this model of analysis. *To perturb the autonomic system with specific modes of stimulation will reveal in linear vs. nonlinear, and, conservative vs. dissipative transduction phase analysis: causal underlying psychological states, dynamics and pathology.* Observation of manifest autonomic sympathetic functioning, reflects a copious and rich measure of psychophysical state specificity, if properly analyzed within the context of associated phase transduction, chaotic-nonlinear and linear aspects. That analysis extends past behaviorist inferences, into the very depths of affective assignment.

To accomplish this we must ask: How does the basic affective repressive regulatory circuitry and mnemonic affective processing interact with cortical and limbic expression, as related to GSR?

**Limbic/OFC circuits and related structures, their dynamics and GSR—the hidden**

### **mind measured—Jung’s looking glass:**

Now that we have drawn out a general picture of the basic connectivities of the sympathetic branch of the autonomic system (and balanced parasympathetic dynamics), we need complete the picture and add the increased complexity of the related mnemonic, cortical and limbic affective structures as they intersect, integrate and cross-modulate affective expression in internal ideation and external stimulus processing. Neural activity occurs across brain structures, and must be assessed as such (Norman, 2016). Then the limit, abundance of information and analytic potential can be fully appreciated. A circuit analysis set in a primary developmental context related to object quality and repression is required. Are the structures involved correlated with GSR? How deeply might GSR be able to peer into the hidden human questions of health and illness?

### **Affective Regulatory Circuitry analysis:**

Schore has discovered two circuits which are primary in development, and function in opposition to each other: the dopaminergically modulated sympathetic ventral tegmental limbic circuit, and the noradrenergically modulated lateral parasympathetic tegmental limbic circuit [Schore as cited in (Kaplan-Solms&Solms, 2002 p. 234-235)]. The sympathetic circuit, which we propose underlies intersubjective Alpha Function (Norman, 2013, 2014; Brown, 2011) is formed, much as Bion had supposed, as a function of the dyadic exchange between infant and mother of glance and gaze, and we will add an inference which is quite obvious and easily supported (Panksepp, 1998 p. 272; Keveren, 1989; Montagu, 1978) as infants engaged in the exchange of maternal glances are usually being held, that *maternal touch* and the subsequent addition of neuropeptides/endorphins also have a part to play in creating the result:

"It is hypothesized that maternal regulated high intensity socioaffective stimulation provided in the ontogenetic niche, specifically occurring in dyadic psychobiologically attuned, arousal amplifying, face to face reciprocal gaze transactions, generates and sustains positive affect in the dyad. These transactions induce particular neuroendocrine changes which facilitate the expansive innervation of deep sights in orbitofrontal areas, especially in the early maturing visuospatial right hemisphere, of ascending subcortical axons of a neurochemical circuit of the limbic system—the sympathetic ventral tegmental limbic circuit." [Schore as cited in (Kaplan-Solms & Solms, 2002p. 234)]

The sympathetic tegmental limbic circuit is dopaminergically modulated, and can rightly be thought of as a primary manifestation of libidinal excitation and discharge (Kaplan-Solms & Solms, 2002 p. 237). It should be noted that the dopaminergic and opioid systems and circuitry which respond to create the good feelings which reinforce socially mediated behavior, both involve many of the same areas, such as the ventral tegmental area, where the A-10 mesolimbic dopamine cells are located (Panksepp, 1998 p. 118). Neuropeptides such as the endogenous opioids including beta-endorphin which is triggered by social cues and touch, have a primary role in creating social bonds, quelling pain, both physical and mental, are key in alleviating separation distress, creating sexual reward, and addictive reinforcement (Panksepp, 1998 p. 255, 264). So we can see here, in

the formation of the sympathetic ventral limbic circuit triggered by maternal exchanges of glance, sight and touch, a source of libido, an energetic dopaminergic circuit which up-mediate arousal and shapes behavior, formed presumably by way of allocating both endorphins, and those neuroendocrine functions involved with encouraging the substantial innervations of dopaminergic projections into orbitofrontal areas. Here, in the activity of the completed circuit, along with the peptide systems, dopamine and opioids serve their reward and motivational functions as social and energetic contributors.

The contrary circuit, the parasympathetic lateral limbic circuit, is to be thought of as a balance, a cut off, a competing inhibitory system to counter the rewarding energetic expression of the sympathetic circuit (Kaplan-Solms & Solms 2002 p. 237). This circuit functions to stop our energetic libidinal expression: functional, conditional, affect regulation in response to social cues (ibid. pp. 234-238) and so, can best be understood as the physiological structure triggered by social disapproval: *by shame and guilt*. Both of these circuits are innervated into the orbitofrontal areas, which mediate social cues and functioning, just as one would expect.

These two circuits provide in the resultant homeostatic balance, the basic emotional tone and underlying affective regulation in man, and may well be the true foundation of *Empathy*, of which mirror neurons are but a small imitative component subset (Norman 2013, 2014, 2016b).

### **Connection between GSR and human affective repression and expression**

Is there a relation between GSR, and the activity of these primary dopaminergic and noradrenergically modulated sympathetic and parasympathetic limbic/OFC circuits? Is GSR able to assess sympathetic activity between the associated cortical areas such as the OFC/ventromedial prefrontal cortex and its connectivity to the limbic areas, circuitry so primary to the deep complexity of human affective states, and their hidden causes? Can GSR and linear/nonlinear analysis determine the essence of unconscious human affect? Yes, and as you will see, this is exactly as it should be-primary.

In (Nagai et al., 2004) we read the following: “We examined neural activity related to modulation of skin conductance level (SCL), an index of sympathetic tone, using functional magnetic resonance imaging (fMRI) while subjects performed biofeedback arousal and relaxation tasks. Neural activity within the ventromedial prefrontal cortex (VMPFC) and the orbitofrontal cortex (OFC) covaried with skin conductance level (SCL), irrespective of task. Activity within striate and extrastriate cortices, anterior cingulate and insular cortices, thalamus, hypothalamus and lateral regions of prefrontal cortex reflected the rate of change in electrodermal activity, highlighting areas supporting transient skin conductance responses (SCRs). Successful performance of either biofeedback task (where SCL changed in the intended direction) was associated with enhanced activity in mid-OFC.” (p. 234.)

Next, please recall that the primary affective regulatory and energetic expressive circuitry associated with the fundamentals of affective repression and release, is the core limbic/OFC circuitry: the sympathetic ventral tegmental limbic circuit [Schoore as cited in (Kaplan-Solms & Solms, 2002 p. 234)], and the parasympathetic lateral limbic circuit. The connections between limbic structures and the OFC are primary (Norman, 2013, 2014). If GSR is to probe the true depths of human experience, it MUST demonstrate correspondence in its measurements, to these circuits which span the limbic system and OFC. For here we find the most primary regulatory/energetic mechanism which dispenses interest in the world (please think of Panksepp's SEEKING system (Panksepp, 1998), elation, shame and guilt (Kaplan-Solms & Solms, 2002). The OFC contains affective coding which spans population, and the limbic system is of course known as the affective mainspring (Chikazoe et al., 2014). These circuits are a primary basis of manifest human emotion. Is there reason to believe GSR can provide information about them? If so, it is clear, the unconscious itself in all of its nuanced complexity as the mediator of human conscious affect may be measured in its effects with GSR.

Indeed this is just the case. In (Frey, Zlatkina and Petrides, 2009) we read the following: "The results demonstrate that the right rostral orbitofrontal cortex is involved in the active encoding of novel tactile information, while a more caudal region of the orbitofrontal cortex, which is more closely connected with limbic and autonomic regions of the brain, was activated when subjects explored novel aversive tactile stimuli. *These results suggest that the orbitofrontal cortex, through its connections with the limbic areas of the medial temporal lobe, influences the processing of incoming information and thus contributes to its encoding.*" (p. 650.) [emphasis added]

Also this:

Baseline GSR rates were recorded in microsiemens (IS) and were first obtained by presenting the subjects with familiar control stimuli (Control). Novel: GSRs to non-aversive tactile stimuli used in the PET novel tactile encoding condition. Aversive: GSRs to aversive stimuli used in the PET aversive tactile condition. Significant differences were observed between the aversive and the non-aversive tactile stimuli (paired t-test,  $P < 0.05$ ). (caption p. 652)

And from p. 654:

"After an initial baseline GSR trace was established, all subjects exhibited deviations from this baseline state that are characteristic of typical traces observed from sympathetic electrodermal activity (see Fig. 1). Although the responses varied across stimuli and subjects, significant statistical differences were found between all of the aversive tactile stimuli and five of the selected stimuli from the nonaversive tactile condition (paired t-test,  $t = 1.92$ ,  $df = 5$ ,  $P < 0.05$ ). These findings imply that the aversive tactile stimuli led to elevated skin conductance levels and were

likely more emotionally charged in comparison to the novel tactile stimuli. In the psychophysiological experiment, performance in the recognition memory test for the nonaversive tactile stimuli was 71.5% correct, sd 5 19.9, and for the aversive tactile stimuli was 88.5% correct, sd 5 6.4 (paired t-test,  $t = 2.46$ ,  $df = 7$ ,  $P < 0.05$ ).\" (p. 654 *ibid.*)

Of course it has long been known that proper functionality in biology can be fruitfully determined by nonlinear analysis (Panksepp, 1998, pp. 93-94; Kleick, 1987; Elbert et al., 1994; Freeman 1991, 1995; Lipsitz and Goldberger, 1992 p. 1808). It now makes good sense to read that positive affect such as happiness, has a distinct nonlinear signature derivable with GSR, for it is *sympathetic OFC/limbic circuitry* which distributes happiness/elation (Wang, Liu and Yang, 2014; Kaplan-Solms & Solms, 2002). Ergo: nonlinear sympathetic autonomic systems analysis is appropriate to define the state. We may conclude that GSR contains encoding endemic to the deepest hidden affective responses in the human animal, and clearly understand, only a proper method of linear/nonlinear mathematical analysis is needed to extract this vital information.

We retain, in accordance with (Basak et al., 2005; Wang, Liu and Yang, 2014) that a primary analytical emphasis on GSR further contextualized as to its linear/nonlinear attribution via inclusion of secondary variables such as EEG and ECG, within a primary dissipative/conservative transduction phase analysis, allows assessment of basic peripheral sympathetic responses in GSR to controlled multi-level stimulus including-tactile, olfactory, general auditory (sounds), linguistic (words and phrases), and visual stimuli-so as to reveal internal affective states and pathological categorization, with accuracy never before achieved. Next we will detail the specific mathematics used to assess human affect, within this promising and important new analytic framework as has been developed and researched in our laboratory of electrophysiology in Bari at the S.T.M.P.

## References:

Barry, R. J. (1980) Electrodermal responses to emotive and non-emotive words as a function of personality differences in affect level. *Biological Psychology* Volume 11, Issues 3–4, November–December, Pages 161–168

Basak, T. K., Halder, S., Kumar, M., Sharma, R., Midya, B. (2005)  
A topological model of biofeedback based on catecholamine interactions.  
*Theoretical Biology and Medical Modelling*, 2:11 doi:10.1186/1742-4682-2-11

Benedek, M. and Kaernbach, C. (2010) A continuous measure of phasic electrodermal activity. *J Neurosci Methods*. 190(1-5): 80–91.  
doi: 10.1016/j.jneumeth.2010.04.028 PMID: PMC2892750  
<http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2892750/>

Boucsein, W. (1992) *Electrodermal activity*. Plenum University Press; New York.

Brown, B. (November 9, 1977). "Skin Talks -- And It May Not Be Saying What You Want To." *Idaho State Journal*. p. 32. Pocatello, Idaho: Field Enterprises, Inc. <https://www.newspapers.com/newspage/22744127/>

Brown, L. (2011) *Intersubjective Processes and the Unconscious*. Routledge, London.

Chikazoe, J., Lee, D. H., Kriegeskorte, N. & Anderson, A. K. (2014)  
Population coding of affect across stimuli, modalities and individuals.  
*Nature Neuroscience* 17, 1114–1122 doi:10.1038/nn.3749  
<http://www.nature.com/neuro/journal/v17/n8/full/nn.3749.html>

Critchley, H. D. (2002) "Book Review: Electrodermal Responses: What Happens in the Brain." *The Neuroscientist* 8 (2): 132–142. doi:10.1177/107385840200800209. PMID 11954558.

Efes, E. D. (1992) Mechanism of effect of sympathetic nervous system on skin receptors. *Neurophysiology*. Volume 24, Issue 5, pp. 358-363

Elbert, T., Ray, W. J., Kowlaik Z. J., Skinner, J. E., Fraf, K. E., & Birdbaumer, N. (1994). Chaos and physiology: Deterministic chaos in excitable cell assemblies. *Physiol. Rev.* 74: 1-47.

Ellis, R. J. and Thayer, J. F. (2010) Music and Autonomic Nervous System (Dys)function. *Music Percept.* (4): 317–326. doi: 10.1525/mp.2010.27.4.317  
PMCID: PMC3011183

Freeman, W. J. (1991).  
The physiology of perception. *Sci Am.* 264 (Feb.): 78-85

Freeman, W. J. (1995).

*Societies of the brain: a study in the neuroscience of love and hate.*  
Hillsdale, NK.: Lawrence Erlbaum.

Frey, S., Zlatkina, V., and Petrides, M. (2009) Encoding touch and the orbitofrontal cortex.  
*Human Brain Mapping.* 30(2):650-9.DOI: 10.1002/hbm.20532

Gazzaniga, M., Ivry, R. and Mangun, G. (2009) *Cognitive Neuroscience: The Biology of the Mind.* Norton Press, London.

Gerlach, C., Aaside, C.T., Humphreys, G.W., Gade, A., Paulson, O.B., Law, I. (2002)  
Brain activity related to integrative processes in visual object recognition: bottom-up  
integration and the modulatory influence of stored knowledge. *Neuropsychologia.* 40(8):1254-67.  
<http://www.ncbi.nlm.nih.gov/pubmed/11931928>

Hall, R.A. (2011) Autonomic modulation of olfactory signaling. *Sci Signal.* 4(155)  
doi: 10.1126/scisignal.2001672.  
<http://www.ncbi.nlm.nih.gov/pubmed/21224443>

Kaplan-Solms, K., & Solms, M. (2002) *Clinical Studies in Neuropsychanalysis: Introduction to a Depth Neuropsychology.* London: Karnac Press.

Karthikeyan, P., Murugappan, M., Yaacob, S. (2013) Multiple Physiological  
Signal-Based Human Stress Identification Using Non-Linear Classifiers.  
*ELEKTRONIKAI RELEKTROTECHNIKA*, ISSN 1392-1215 , VOL . 19 , NO . 7  
<http://dx.doi.org/10.5755/j01.eee.19.7.2232>

Keveren, E. B., Martensz, N., & Tuite, B. (1989)  
Beta-endorphin Concentrations in CSF of Monkeys are Influenced by Grooming Relationships.  
*Psychoneuroendocrinol.* 14: 155-1661.

Kleick, J. 1987. *Chaos: Making a new science.* NY.: Penguin.

Lee, A. C. H., and Rudebeck, S. R. (2010)  
Human Medial Temporal Lobe Damage Can Disrupt the Perception of Single Objects.  
*The Journal of Neuroscience*, 30(19): 6588-6594;  
doi: [http:// dx. doi. org/ 10. 1523/ JNEUROSCI. 0116-10. 2010](http://dx.doi.org/10.1523/JNEUROSCI.0116-10.2010)  
<http://www.jneurosci.org/content/30/19/6588.full>

Lipsitz, L. A., Goldberger, A. L. (1992). Loss of 'Complexity' and Aging  
Potential Applications of Fractals and Chaos Theory to Senescence.  
*JAMA.* 267:1806-1809.

Montagu, A. (1978) *Touching: The Human Significance of the Skin.*  
New York: Harper and Row.

- Murray, E. A. and Mishkin M. (1998). Object Recognition and Location Memory in Monkeys with Excitotoxic Lesions of the Amygdala and Hippocampus. *The Journal of Neuroscience*, 18(16): 6568-6582  
<http://www.jneurosci.org/content/18/16/6568.short>
- Murray, E. (2000). Memory for objects in nonhuman primates. In M. S. Gazzaniga, Ed. *The New Cognitive Neurosciences, 2nd ed.* London: MIT Press, pp. 753-763
- Nagai, Y., Critchley, H.D., Featherstone, E., Trimble, M.R., Dolan, R.J. (2004) Activity in ventromedial prefrontal cortex covaries with sympathetic skin conductance level: a physiological account of a "default mode" of brain function. *NeuroImage*, 22 (1) pp. 243 - 251. [10.1016/j.neuroimage.2004.01.019](https://doi.org/10.1016/j.neuroimage.2004.01.019).
- Norman, R. L. (2013) Who Fired Prometheus? The Historical Genesis and Ontology of Super-ego and the Castration Complex: The Deconstructuralization and Repair of Modern Personality—An Essay in Five Parts. *The Journal of Unconscious Psychology and Self-Psychoanalysis*.  
[www.thejournalofunconsciouspsychology.com](http://www.thejournalofunconsciouspsychology.com)
- Norman, R. L. (2014) Limbic Connectivity and Sympathetic Neural Balance: The Primary Psycho-physiological Locus of Affect. *Mind magazine*.  
<http://www.mindmagazine.net/#!/new-ideas/czpl>
- Norman, R. L. (2016) The Quantitative Unconscious: A Psychoanalytic Perturbation-Theoretic Approach to the Complexity of Neuronal Systems in the Neuroses, *Neuroquantology*, Vol. 14 issue 2 [10.14704/nq.2016.14.2.949](https://doi.org/10.14704/nq.2016.14.2.949) **356-368**
- Norman, R.L. (2016b) Homeostatic Conductance and Parasympathetic Basis Alteration: Two Alternative Approaches to Deep Brain Stimulation in Parkinson's, Obsessive Compulsive Disorder and Depression. *World Journal of Neuroscience*, 6, 52-61. <http://dx.doi.org/10.4236/wjns.2016.61007>
- Ogorevc, J., Geršak, G., Novak, D., Drnovšek, J. (2013) "Metrological evaluation of skin conductance measurements." *Measurement* **46** (9): 2993–3001.  
[doi:10.1016/j.measurement.2013.06.024](https://doi.org/10.1016/j.measurement.2013.06.024).
- Panksepp, J. (1998) *Affective Neuroscience: The Foundations of Human and Animal Emotions*. New York, NY.: Oxford Press.
- Petty, R., Ostrom, T. M., Brock, T. C. (Eds.) (2014) *Cognitive Responses in Persuasion*. Psychology Press, Routledge group, NY.
- Pohl, W. (1973). Dissociations of spatial discrimination deficits following frontal and parietal lesions in monkeys. *J. Comp. Physiol. Psych.* **82**:227–239.

Rousmans, S., Robin, O., Dittmar, A. and Vernet-Maury, E. (2000) Autonomic Nervous System Responses Associated with Primary Tastes. *Chemical Senses* Volume 25, Issue 6 pp. 709-718. <http://chemse.oxfordjournals.org/content/25/6/709.full>

Wada, T., Takahashi, K., Ito, Z., Hara, A., Takahashi, H., Kasakari, J. (1999)  
The protective effect of the sympathetic nervous system against acoustic trauma.  
*AurisNasus Larynx*; 26(4):375-82

WANG, L., LIU, G., YANG, Z. (2014) The Emotion Recognition for Grief Based on Nonlinear Features of GSR. *Journal of Computational Information Systems* 10: 4 1639–1649 DOI: 10.12733/jcis9466