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**NEUROPSYCHOLOGY AND PSYCHOPHYSIOLOGY  
IN PERSONALITY RESEARCH**

**Part II. Psychophysiological Techniques and  
Personality Theory**

**by**

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**(References listed at the end of Part II include  
articles cited in Part I, PR-65-3)**

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## PART II. PSYCHOPHYSIOLOGICAL TECHNIQUES AND PERSONALITY RESEARCH

More and more research in the personality field qualifies as "psychophysiological" by Stern's (1964) definition, in that physiological measures are employed as dependent variables. The remainder of this chapter is devoted to a discussion of some of the more important of these experimental tools and to associated problems of methodology and interpretation.

### Biochemical Indicators

Emotional arousal is accompanied by hormonal changes which, especially if the arousal is intense or prolonged, may be detected through biochemical analysis of body fluids. Adrenal secretion is particularly important in the emergency emotions of fear and rage and methods are now available for measuring the products (or their metabolic end-products) of adrenal cortex and adrenal medulla in the blood plasma or in the urine.

#### Hormones of the Adrenal Cortex.

The pituitary hormone ACTH or corticotropin stimulates the adrenal cortex to secrete hydrocortisone which circulates in the blood plasma and metabolizes to produce at least 11 different steroids (Persky, 1962). Several of these metabolic products are referred to collectively as the hydroxycorticoids and another group constitutes the 17-kertosteroids. Elevated levels of hydrocortisone are found in the blood plasma of acutely disturbed or anxious patients and the levels can also be reliably increased by the stress of a disturbing interview or the like. Hypnotic induction of anxiety in normal subjects can also produce a marked elevation in plasma hydrocortisone (Persky et al, 1959). Stress associated with the conditioned emotional response (CER) and the Sidman avoidance procedures produce marked elevations of plasma 17-hydroxycorticoid levels

in experimental monkeys (Brady, 1964). Handlon (1962) was able to produce a significant decrease in plasma 17-hydroxycorticosteroid levels in normal subjects through relaxing hypnotic suggestion or by having his subjects watch a "bland" motion picture or engage in an absorbing but non-stressful perceptual discrimination task. Urinary excretion of hydroxycorticoids is markedly elevated in anxious psychiatric patients (Persky, 1962) and also in medical students just after taking an important final examination (Schwartz and Shields, 1956).

Fox et al (1961) attempted a longitudinal study of healthy college students combining interviews and Rorschach testing with daily measurements of urinary hydroxycorticoid and 17-ketosteroid excretion. These authors were not able to relate the manifest anxiety levels of their subjects to the biochemical indices but do report that the highest hydroxycorticoid excretors showed relatively poor control of their feelings and impulses while the lowest hydroxycorticoid excretors were characterized by emotional over-control. In a cross-sectional study with normals, Fiorica and Muehl (1962) found a correlation between score on Taylor's Manifest Anxiety Scale and the level of free 17-OH-CS in the venous blood. Long term longitudinal studies of individuals in psychotherapy have produced conflicting results; Schwartz and Shield's (1965) patient showed lower steroid excretion during periods of high tension whereas the psychoanalytic patient studied over a three year period by Fox et al (1958) showed an elevation of urinary hydroxycorticoid excretion during the first year or two of treatment followed by a decrease both in mean level and in variability during the final year as the treatment was brought to a successful conclusion. Like 17-hydroxycorticoid excretion, the level of

17-ketosteroids in the urine increases with stress or anxiety level and, in normal subjects, seems to be a predictor of anxiety proneness. Persky (1962) has demonstrated that the adrenal cortex of the anxious patient is unusually responsive to ACTH and found elevated levels of this pituitary hormone in the plasma of these patients as well. He suggests that patients, or normals under extreme stress, may show a change in their pattern of metabolizing hydrocortisone, from a primary output of hydroxycorticoids to a relative increase in the production of 17-ketosteroids.

Detailed biochemical methods for determining the levels of these substances in plasma or in urine may be found in McCarthy, et al (1964), Peterson (1963), Quesenberry and Ungar (1964), Silver (1963) and Venden-Heuvel, Creech and Horning (1962).

#### Secretions of the Adrenal Medulla.

The chemical mediator released by sympathetic nerve-endings is the catecholamine norepinephrine (NE) which is closely related to the great emergency hormone, epinephrine (E) or adrenaline, which stimulates metabolism and promotes blood flow to the skeletal muscles, preparing the organism for "fight or flight." Epinephrine is secreted into the bloodstream by the adrenal medulla. It has been estimated that as much as 20 percent of the total catecholamine content of adrenomedullar product consists of norepinephrine and it is said that stimulation of different hypothalamic areas varies the proportions of NE and E released (Goth, 1965). Although chemically very similar, the two differ greatly in many of their effects; e.g. in physiological quantities, both increase systolic blood pressure while diastolic pressure is raised by E (which increases peripheral vascular resistance) but lowered by NE; E sharply increases heart rate

while NE produces a reflex slowing of the heart secondary to vasoconstriction and increased blood pressure. Although the catecholamines are believed to be largely blocked by the blood-brain barrier, it is known that a region of the upper midbrain reticular formation is sensitive to E and may mediate the cortical arousal effects of that hormone (Jasper, 1958) and it appears that NE may play a role in hypothalamic activity (Vogt, 1954).

Funkenstein and his co-workers (1952, 1957) showed that there are wide individual differences in the pattern of blood-pressure response following the injection of mecholyl. Pre-injection of NE in normals yields a marked hypertensive reaction (brief decrease and then pronounced, overshooting elevation of blood pressure) while pre-injection of E produces an exaggerated hypotensive reaction (persisting fall in blood pressure lasting many minutes). The NE-like mecholyl response was found in psychiatric patients characterized by aggressiveness and outward expression of anger while the E-like response was associated with inward-directed anger or anxiety and fearfulness. Silverman and Cohen (1960) obtained similar findings from a more homogeneous group of seven military aviators and were also able to show that the ratio of NE to E excreted in the urine of their subjects tended to accord with expectations; i.e. the three men rated as anxious but not angry had low NE/E ratios and gave hypotensive mecholyl reactions while the three men rated to be most aggressive and least anxious gave high urinary NE/E ratios and hypertensive mecholyl reactions. In another experiment, these same authors attempted to make their aviator subjects angry during a centrifugation test for g-tolerance; the subjects who seemed to be frightened by this stress were found to have the least tolerance for rotational acceleration and showed the highest levels of

excreted E and the lowest levels of NE. Conversely, the men who reacted to the stress with overt anger and aggression had the highest g-tolerance, the lowest stress levels of E and the highest stress levels of NE.

Elmadjian, Hope and Lamson (1957) measured the urinary E and NE levels of a group of professional hockey players, before and after a typically extro-punative display of their art in a game, finding an average five-fold increase in NE and a two to three-fold increase in E after the game. The goalie, whose duties limit his aggressive opportunities, and two injured players who watched from the sidelines showed only slight NE increments but a considerable rise in E excretion. A group of amateur boxers were found to have very high NE levels both before and after their encounters in the ring. In a preliminary report of findings from a study of a group of Formosan children, Wolf and Lambert (personal communication) indicate a similar tendency for high ratings of inter-personal aggressiveness to be associated with high ratios of NE to E as excreted in the urine.

Although the mechanisms involved are not well understood, it thus seems clear that either the absolute or the relative levels, or both, of secretion or excretion of these two catecholamines must bear a close and important relationship to emotional arousal and to temperamental differences in fear or anger readiness. Although various biochemical techniques are available for assessing E and NE levels in plasma and in urine (e.g., Antom and Sayre, 1962; Crout, 1961; Fales & Pisano, 1962; Vemdsalu, 1960; vonEuler and Lishajko, 1961), normative data are not yet available which would allow one to compare the quantitative findings of different investigators. One eagerly awaits the technological improvements which may make these promising dependent variables available to the personality researcher and

the psychiatric clinician.

### The Electroencephalogram (EEG)

The electroencephalogram is a graph against time of rhythmic variations in the minute electrical potentials recorded between two electrodes on the scalp (bi-polar recording) or between a single scalp electrode and an "indifferent" or reference electrode attached to the ear lobe or to the back of the neck (mono-polar recording). These voltage waves vary in amplitude from a few microvolts (near the residual noise level of most recording systems) up to as high as 200 microvolts, and in frequency from near-DC to 50 cps or more. Experience has made it possible to divide the EEG spectrum into bands of frequencies having special significance. In his original paper in 1929, Berger described the alpha waves, which include frequencies in the range from 8 to 13 cps, and beta waves, in the range from 18 to 30 cps. Alpha waves tend to dominate EEG tracings recorded from occipital, parietal, and temporal locations on the resting subject, especially when the eyes are closed. Electrodes placed over the frontal regions commonly show more low voltage, fast activity in the beta range. Slow waves having a frequency of 3.5 cps or below and generally of large amplitude, characteristic of sleep, are known as the delta rhythm. The intermediate frequencies from 4 to 7 cps, known as theta waves, are common in the EEG of the young child. Other EEG correlates and their relation to the arousal continuum are discussed elsewhere in this chapter.

The origin of these "brain waves" is still not definitely established. The earlier view was that these rhythms were produced by spreading waves of firing of cortical neurons, i.e., that the slow EEG wave is a kind of



average envelope of many brief axon spikes. It is now much more widely believed, however, that the slower, graded activity of the apical dendrites of the pyramidal cells of the cortex may be the primary source; i.e., that the rhythmic activity of many of these units, waxing and waning in synchrony, may produce the waves that are recorded on the scalp. Actual cell firing also contributes to the recorded electrical activity as in the case of the cortical evoked potentials. Epilepsy, focal and diffuse brain damage, anesthetic agents and other pathological conditions may produce bizarre or characteristic electro-cortical activity and therefore the EEG is an important clinical tool for use in diagnosis, localizing irritative cerebral lesions, monitoring anesthesia, and the like.

#### Techniques of Recording.

EEG electrodes are most commonly small silver disks, sometimes chlorided electrolytically before use, which are attached to the scalp either by a special cap or harness or else individually cemented in place. Subcutaneous needle electrodes are also frequently used but may not be worth the discomfort which they cause to the subject. The electrode site should be lightly abraded and rubbed well with electrode paste to insure minimum electrode resistance. For clinical purposes and to insure a comprehensive coverage of all cortical areas, as many as 20 individual electrodes may be used at a time, placed over the head in a more or less standard arrangement suggested by Jasper (1958b).

The standard electroencephalograph allows for the recording of as many as eight or more channels simultaneously, each channel representing the signal developed between one scalp electrode and the reference, in the case of monopolar recording. Bipolar recording, between two scalp electrodes,

is more difficult to interpret since the result is an algebraic difference between the activities of the two sites. However, bipolar recording is especially useful in localizing tumors or lesions. Because EEG potentials are so small, EEG amplifiers must be very sensitive and this in turn presents a considerable problem of interference from electrical noise. Amplifiers must have a high capacity for rejecting in-phase signals appearing at both electrodes and the subject must be well grounded. In electrically noisy environments, a shielded room may be required.

For psychophysiological research purposes, fewer channels or even a single channel may be used. A common practise now is to record the EEG on magnetic tape, using an oscilloscope or a standard ink-writer for on-line monitoring. Once stored on tape, the record can be later reviewed by playing back to an oscilloscope; important segments can be saved by playing back to an ink-writer without the need for writing out yards of unessential record, and the tape-recorded data can also be easily subjected to various types of automatic analysis. For example, the tape can be played-back repeatedly into an electronic frequency analyzer or a bandpass filter in order to determine the amplitude of various frequency components in each channel, or other specialized electronic analyzers may be used to compare phase relationships between channels, compute autocorrelations, and the like. Cortical evoked potentials may now be obtained from EEG recordings by computer averaging techniques, providing that the eliciting stimulus can be repeated at least 50 to 100 times; this method is explained more fully elsewhere in this chapter.

Of the many useful references available on EEG method and theory, attention may be directed to the symposia edited by Hill and Parr (1963) and by Glaser (1963).

## The Electrocardiogram

The electrocardiograph (EKG or ECG) is a graph against time of the electrical activity of the heart as it is picked up between two electrodes on the surface of the body. Like all muscle fibers, those of the heart muscle are electrically polarized in the normal state; that is, the surface of each muscle fiber is some 50 millivolts positive with respect to the interior of that fiber. When stimulated into contraction, the muscle fiber depolarizes and the surface loses its positive potential and even becomes momentarily negative with respect to the interior of the muscle cell. Immediately thereafter, the cell begins to repolarize back to its normal resting condition. When a large number of such fibers contract nearly simultaneously as they do in the mass of the heart muscle, their changing surface potentials may summate in the form of a rather considerable wave of voltage.

Within the heart muscle, contraction spreads in a regular wave of excitation originating in the auricles and spreading into the more massive ventricles. These waves of excitation are initiated by a cardiac pace-maker mechanism which is situated in the sino-auricular node in the right atrium. Excitation spreads through both auricles until it reaches the bundle of His near the upper margin of the ventricular mass. The bundle of His is a two-branched system of specialized conductive tissue whose branches course downward and laterally to the walls of the right and left ventricles and guide the spreading wave of excitation smoothly through these regions. Because the ventricles comprise most of the mass of the heart, ventricular contraction generates the larger waves of electrical activity recorded in the EKG.

An electrode placed on or near the heart itself will record maximum electrical activity at the moment that the wave of excitation passes directly under the electrode site. This relatively short, large amplitude deflection will normally be followed by a second wave of smaller amplitude but longer duration representing the spread of repolarization of the muscle tissue. Repolarization will ordinarily spread in the same direction as the original depolarization and should then produce a wave of opposite polarity. The shape of the wave produced by depolarization will depend upon the placement of the electrode relative to the course of the excitatory process. In bipolar recording, where the second or reference electrode is placed upstream from the first (with respect to the wave of contraction), the first or recording electrode will appear to go positive as the wave of depolarization passes under the reference electrode and then negative again as its own region depolarizes, returning both electrodes to the same potential. Should the reference electrode be situated downstream from the recording electrode, this same spread of excitation will be recorded as a negative voltage wave rather than as a positive wave.

In electrocardiography, the recording electrodes are placed at some distance from the contracting muscle mass and separated from it by the fairly complex conducting pathway represented by the intervening tissue. Potentials at the heart surface radiate and interact so that a small electrode on the surface of the body will tend to behave rather like a much larger electrode in direct contact with the heart.

#### Electrode Placement.

Clinical electrocardiography normally employs three fixed electrodes situated on both arms and the left leg together with one or more exploratory

electrodes located in anatomically standardized positions on the chest. In the early days of electrocardiography, recording was entirely bipolar between various possible pairs of these standard electrode positions. Unipolar recording, developed during the 1940's, is a technique in which each standard electrode site is compared against a reference point obtained by tying the remaining standard electrodes together through 5,000 ohm series resistors (all standard electrodes may also be tied together in this way to form the common reference, including the lead from which one is recording). Since all electrodes or leads are thus compared against a standard and less changeable reference point, unipolar recording provides relatively simpler results in which the recorded electrical phenomena are easier to rationalize with respect to the underlying physiological events.

A standard nomenclature has evolved for describing these various electrode arrangements. In the case of bipolar recording, the comparison of the right arm electrode against the left arm electrode is known as Lead I, the right arm against the left leg as Lead II, the left arm against the left leg as Lead III, and an exploring electrode on the chest wall compared to an indiffereent electrode on the left leg is known as Lead  $CF_4$ . In the case of unipolar recording, where the three standard electrodes are tied together through resistors to a common reference terminal, the comparison against this common terminal of the right arm electrode is known as Lead VR, use of the left arm electrode gives Lead VL and use of the left leg electrode gives Lead VF. A list of standard locations for unipolar chest electrodes has also been agreed upon. Finally, there are the so called "augmented" standard unipolar leads, designated aVR, aVL. and aVF respectively in which the electrode from which one is actively recording is disconnected from the

common reference terminal. This system increases the amplitude of the recorded deflections (hence the designation "augmented") but, since the reference terminal is to some extent electrically different for each lead, this method may also lead to certain distortions of the resulting EKG.

#### EKG Waveform

One cycle of the typical EKG as might be recorded by bipolar Lead II is represented in Figure 10. There are a total of 5 waves in the cycle, designated by the letters P, Q, R, S and T respectively. The initial low amplitude P-wave represents the contraction of the auricles. Following the P-wave is the QRS complex which is the largest deflection in the EKG and represents depolarization and contraction of the ventricles. The final component in the record is the relatively small T-wave which is associated with the process of repolarization of the heart muscle.

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Figure 10  
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The actual wave shapes of the normal EKG may vary greatly from this standard pattern depending upon the leads employed and also upon the anatomical position of the heart relative to the rest of the body, which may vary considerably even among healthy subjects and thus cause variation in the pattern of electrical activity picked up at the standard lead positions. The usual clinical EKG will include recordings from the extremities, usually bipolar leads I, II, and III and the augmented unipolar leads aVR, aVL and aVF together with a set of unipolar precordial (chest) leads  $V_1$  through  $V_6$ . Most abnormalities of heart function produce more or less characteristic changes in the patterns recorded from these standard leads, changes in the

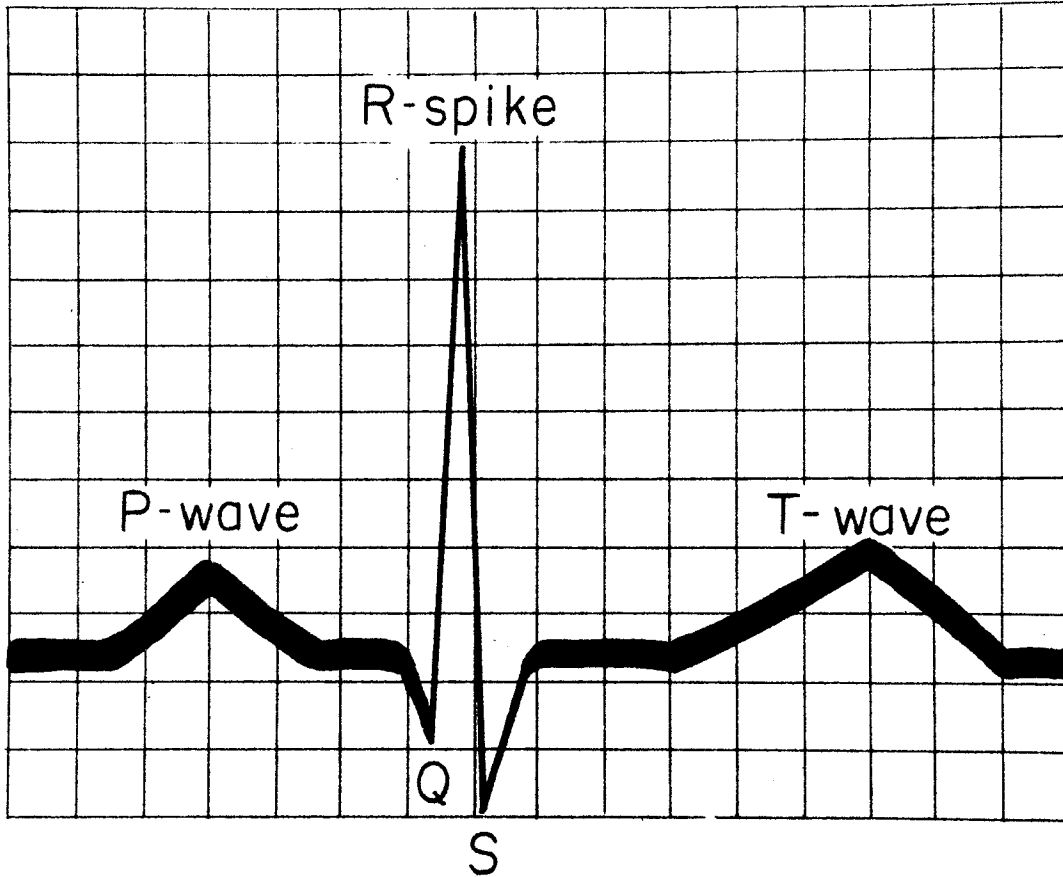


Figure 10. Typical electrocardiographic (EKG) cycle. P-wave represents contraction of auricles, QRS complex results from ventricular contraction and T-wave represents repolarization of heart muscle. Shape and relative size of EKG components may vary greatly from lead to lead or from subject to subject. Each vertical division represents 0.5 mV while each horizontal division represents 100 mSec.

amplitude, shape, duration or polarity of one or more of the five waves or changes in the timing of the intervals between waves. At the present time, electrocardiography is a semi-theoretical discipline in the sense that certain inferences about heart position, structure or function can be made from the EKG by deduction from a theoretical understanding of the anatomical, physiological and electrical principles involved. However, most clinical diagnostic usage is still largely empirical; previous clinical or laboratory investigations have shown that certain physiological abnormalities yield characteristic changes in the electrical record although the details of the mechanism involved may be somewhat obscure.

There are many competent texts on clinical electrocardiography. Two good examples are Bernreiter (1963) and Burch and Winsor (1960).

The various waves which are the phenomena of interest in the EKG may range in amplitude from a few hundredths of a millivolt to a few millivolts as measured from the skin surface. The duration of the QRS complex is characteristically less than one hundred milliseconds although the slower P and T-waves may last as long as 200 milliseconds. Since the steady-state potential difference between the recording electrodes has no significance relative to the heart, EKG recording is normally done by means of an AC recording system having good low frequency response. Because of AC recording, electrode polarization is not a serious problem and large German silver electrodes are commonly used together with a convenient, non-drying saline jelly to insure a good contact with the skin surface. Large or variable skin resistances under the several electrodes may be a problem, however, and a common practice is to include fine pumice granules in the electrode paste and to rub this paste thoroughly into the skin before applying the



electrode. Alternatively, the electrode site can be lightly abraded with fine sandpaper. Standard EKG recording is on a strip chart from 4 to 5 centimeters wide, moving at a speed of 5 centimeters per second and with the amplifier calibrated to yield a deflection of one centimeter per millivolt.

#### Measurement of Heart Rate (HR).

Heart rate can be obtained from the EKG record by measuring the average time between corresponding waves in successive heart cycles (the average interbeat interval) in seconds and dividing this value into 60 to get heart rate in beats per minute. Because it is normally the largest and most sharply peaked of the five waves, the R-wave of the QRS complex is the usual reference point for this measurement. Measurement of heart rate is done automatically by an instrument called the cardiotachometer in which the amplified EKG signal is filtered in such a way as to produce a single pulse corresponding to each successive R-wave. These pulses are then electrically molded into a standard shape and amplitude and fed into an integrating circuit whose output is a voltage inversely proportional to the last one or several inter-beat intervals, i.e., directly proportional to the momentary rate. Less expensive instruments for clinical use integrate over a number of successive beats and display the average heart rate either on a meter or on a moving chart recorder. More sensitive (and expensive) instruments may integrate over only one cycle at a time, reporting out each successive inter-beat interval to a strip chart recorder or to a high speed digital printer. Since all cardiotachometers identify each heart cycle by the relatively large spike of the R-wave, it is essential that the other waves in the EKG signal shall be sufficiently small relative to the R-wave so that the filtering

circuits in the in the cardiometer can differentiate between them. This implies that, although only one pair of bipolar leads are required for heart rate measurement, these leads should be so located as to produce a relatively large R-spike. A chest-to-leg connection normally will be the best in this respect followed by the right arm-left leg (Lead II) and the right arm-left arm (Lead I) connections, in that order. Failure to provide an adequate R-spike or the presence of spikey electrical noise in the record may cause the cardiometer to miss beats or to count each beat more than once, producing over- or under-estimation of the true heart rate.

#### Blood Pressure

Arterial blood pressure (BP) is a complex function of heart rate, the volume of blood pumped at each stroke, the force of the stroke and the resistance of the circulatory system. Arterial pressure is maximum during the systole or contraction of the heart muscle and minimum during the diastole, the period in which the heart relaxes and refills with blood. Accurate continuous measures of arterial BP are obtained in animal research by cutting a vessel and inserting a strain gage pressure transducer. With humans, periodic measurements of BP are made by wrapping a pneumatic pressure cuff (a sphygmomanometer) about the upper arm while monitoring flow sounds in the brachial artery just below the cuff with a stethoscope. The cuff is inflated to a pressure which occludes flow and then the pressure is slowly bled off until the sharp pulse of the systole is first heard; the pressure reading of the sphygmomanometer at this point is the systolic blood pressure. As more pressure is released, a point is reached at which the characteristic sound of the diastole is heard; this is the diastolic blood pressure.

No wholly satisfactory method of obtaining accurate continuous measures of systolic (or diastolic) pressure without cannulation have been developed. Conventional "lie detectors" measure a somewhat ambiguous quantity known as "relative BP" by applying a cuff pressure midway between systolic and diastolic and then recording changes in cuff pressure (the pen is commonly driven by direct pneumatic connection with the cuff). Rather elaborate systems have been described (Darrow, 1937; Davis et al, 1954) which automatically inflate and deflate the cuff at intervals, using the appearance and disappearance of the peripheral pulse to identify the systolic level. At least one such system is available commercially. Davis (1957) describes another method of applying a strain-sensitive transducer above the radial artery in such a manner as to record pressure variations without occlusion of the vessel at any time.

#### Blood Volume.

The plethysmograph is a device for measuring changes in the volume of a part of the body (e.g. a finger) as an index of the volume of blood in the peripheral small vessels. Mechanical plethysmographs contain the member in a volume of air or water while changes in the size of the member are measured as changes in the volume of air or water displaced under constant pressure. The impedance plethysmograph measures variations in the impedance of a portion of the body to moderately high frequency alternating current; under proper conditions, impedance variations will reflect variations in local blood volume. A typical impedance plethysmograph is described by Scheer and Kroeger (1961). Brown, Giddon and Dean (1965) provide a comprehensive discussion of plethysmographic techniques, including the promising new method of optical plethysmography.

## Electromyography

Electromyography is concerned with the recording and interpretation of the electrical activity involved in the contraction of muscle fibers. Each motor unit, consisting of a single motor neuron and all the individual muscle fibers innervated by that neuron, generates a motor unit potential immediately before fiber contraction, which typically consists of a two- or three-phase voltage wave, several hundred microvolts in amplitude and lasting some few milliseconds. In clinical electromyography, the behavior of individual motor units is studied, by means of needle electrodes inserted into the muscle, as an aid in the diagnosis and treatment of neurogenic and myogenic disease. The amplitude and wave form of the single motor unit response changes in characteristic ways in various nervous and muscular disorders and clinical electromyography can also be used to measure changes in motor nerve conduction velocity, to accurately specify the effects of motor nerve lesions, and the like.

The psychophysicologist will employ electromyography as a means for measuring tonic muscle tension and sometimes also to detect insipient or inhibited motor movements. For these purposes, surface electrodes similar to those used in EEG recording are usually employed, localized on the skin over the "belly" of some large muscle mass. Surface electrodes pick up simultaneously from large numbers of individual motor units and typically produce a complex, "spiky" record in which the chief interest is its average amplitude, indicating the tonic level of tension in that muscle, and any marked changes in amplitude which may indicate phasic muscle activity.

EMG recording commonly employs standard EEG equipment since the

sensitivity and frequency characteristics required are very similar in the two cases. For psychophysiological purposes, an electronic integrating system is often used, the output of which provides a continuous, smoothed average of EMG amplitude. As is true in EEG work also, the electromyographer must frequently cope with serious problems of electrical noise, consisting mainly of 60-cycle AC interference in his records. By avoiding the proximity of fluorescent lights, electrical motors, diathermy machines, and the like, and by using modern differential-input amplifiers with an active and a reference electrode plus a separate ground electrode on the patient, these noise problems can usually be overcome. In some environments, a special shielded room may be required for EEG and EMG recordings.

A good discussion of clinical applications of electromyography may be found in the handbook by Pearson (1961) while a standard reference for psychophysiological work is the manual by J. F. Davis (1959).

#### Eye Movements and Pupil Size

A number of methods have been described for measuring movements and position of the eye. One approach involves mounting a tiny mirror on a contact lens and projecting a light beam onto the mirror from a stationary source while recording the position or movements of the reflected beam on a target (Ditchburn et al, 1959). A similar technique employs a tiny light source on the contact lens (Byford, 1960). A commercial system is available which uses goggles on which an infra-red light source and a photo-cell are mounted. Due to the lower reflectance of the pupil, the amount of red light reflected to the photo-cell varies with the position of the eye, generating a relatively large electrical signal unencumbered by most of the usual artifacts. Another method employs corneal reflection without

goggles and can be used to track the area of the stimulus target being viewed by the subject (Mackworth and Mackworth, 1958). One of the simplest methods, although prey to various artifacts and problems, is electro-oculography. Each eyeball functions as a small battery, maintaining a constant potential of many millivolts from front to back, the pupil being positive. If two electrodes are attached to the skin above and below the eye or, to record lateral movements, one on either side of the eyes, any deviation of the eyeball from the position of direct forward gaze will produce a voltage difference between the electrodes, the one toward which the pupil turns going positive. A system of two pairs of electrodes, one in vertical and the other horizontal alignment, will make it possible to determine eye position with considerable exactitude. The signals produced are in the range of from 10 to 40 microvolts per degree of eye movement. The technique of electro-oculography is described in admirable detail by Shackel (1961).

Pupil size is most commonly measured semi-continuously by photographic methods (Hess & Polt, 1960; Hess, 1965). An automatic camera operating at about two frames per second gives adequate temporal resolution for most purposes and the system can be arranged so as not to interfere with the subject's vision of stimulus material, using mirrors and infrared illumination of the eye. A highly sophisticated (and expensive) instrument is available commercially which provides accurate and continuous measures of pupil size by means of an infrared scanning system.

#### ELECTRODERMAL PHENOMENA

The skin of the palms and soles (i.e., the volar regions) has the curious property of altering its electrical characteristics in concert with

changes in the psychological status of the subject. Other skin regions, especially the backs of the hands and areas about the face and chest, may also participate in these psychophysiological reactions, particularly if the subject is highly aroused or if the ambient temperature is elevated. The palmar and plantar regions, however, are consistently reactive across subjects and conditions. Since, as we shall see, the sweat glands appear to be the principal effector organs for these electrodermal phenomena, it is significant that the volar sweat glands are unique in that they do not participate in thermo-regulation but respond to psychological activation or excitation while glands in non-volar areas participate in such "emotional" sweating only under conditions of high temperature or high arousal. Darrow (1936) provides a convincing argument that volar sweating serves the function of preparing the organism for action by moistening the relatively thick epithelium of the palms and soles, thereby increasing the adhesiveness and the tactual sensitivity of these manipulative surfaces.

#### Skin Resistance Phenomena

If two suitable electrodes are affixed to the skin surface and a battery is used to drive a weak "exogenous" electric current between them, then one can calculate the apparent electrical resistance of the tissue from the ratio of the applied voltage to the current passed; i.e., by Ohm's Law:  $R = E/I$ . If the current (I) is held constant, then the voltage (E), measured between the two electrodes, will vary linearly with the apparent resistance of the tissue through which the current flows. Most of this resistance is contained within the two transverse sections of epidermis which are in contact with the two electrodes; piercing the skin under one electrode so as to make direct electrical contact with the moist and

highly conductive sub-dermal tissue reduces the apparent resistance nearly in half, no matter whether the electrodes are located on opposite hands or on the same hand. If, instead, the voltage applied across the electrodes is held constant, then the current flowing in the circuit will vary linearly with the reciprocal of the apparent skin resistance, the skin conductance (SC). The unit of electrical conductance is the mho; a skin resistance (SR) of 100,000 ohms is equivalent to a skin conductance (SC) of 10 micromhos.

The tonic SC of the volar regions displays marked diurnal variations. It is lowest during deep sleep, somewhat higher during restless sleep or dreaming and rises sharply when the subject is awakened. Tonic SC tends to increase gradually during the morning hours and to decrease again toward evening, falling somewhat faster as the subject goes to sleep. In the waking subject, SC will be lowest during quiet relaxation, higher during attentive listening or active work and higher yet during excitement. Thus, there is considerable support for the view that tonic SC varies in proportion to some dimension of psychological arousal (Darrow, 1936; Freeman & Griffin, 1939; Malmö, 1958; Richter, 1926; Rose 1964; Woodworth & Schlosberg, 1961).

Superimposed upon these relatively slow, tide-like changes of the tonic SC may be seen the wave-like or phasic changes known as the galvanic skin reflex or GSR. If a subject is stimulated by a brief shock, a sudden noise or signal, or by an internal stimulus such as a cough or an itch or an idea, then following a latency of about two seconds from the onset of the stimulus will be seen a sharp increase in conductance which reaches a peak in from two to five seconds and falls off again, roughly exponentially, toward the original SC value during the ensuing five to ten seconds. If



a series of stimuli of different subjective intensities are administered, the amplitude of the GSRs produced will tend to vary in proportion to these intensities (e.g. Hoveland & Rieson, 1940; Kimmel, 1964; McCurdy, 1950; Plutchik, 1963). However, since the subjective intensity of a stimulus appears to depend in part on the subject's expectations (Lykken, 1959), a novel or unexpected stimulus will commonly produce an unusually large GSR or "orienting reflex" (Sokolov, 1960). Many subjects under conditions of excitement will show a fairly high frequency of non-specific GSRs presumably elicited by internal stimuli. There is some evidence (Burch & Greiner, 1960; Cohen, Silverman & Burch, 1956) that the rate of such spontaneous responding, like tonic SC level, varies with psychological arousal.

#### Skin Potential Phenomena.

If an electrode on the palm and a reference electrode on an inactive region are connected to a sensitive high-impedance voltmeter, one will normally observe an endogenous potential difference with the palm being from about 5 to 50 millivolts (mV) negative with respect to the reference. Like tonic SC, the tonic level of skin potential (SP) also shows wave-like, phasic changes which resemble and are related to the more familiar conductance GSR. These phasic changes in SP are often designated by one of the various labels applied to phasic SC changes--GSR, "psychogalvanic response" or pgr, "electrodermal response" or EDR, etc.--but the two phenomena are not identical and this practice is to be discouraged. Alternative locutions such as "skin potential GSR" or "endogenous GSR" seem awkward. In spite of a reluctance to add further neologisms to an already over-burdened glossary, we shall employ the expression "skin potential reflex" or SPR in the following discussion.

The SPR is frequently a biphasic response consisting of an initial alpha wave of increased negativity at the active site, followed by a beta wave during which the potential swings in the positive (i.e. less negative direction) (Forbes, 1936; Forbes & Bolles, 1936). A more relaxed subject or one who has become better adapted to the stimulus or the experimental setting may show only the negative-going alpha wave while a rare individual will produce uniphasic positive or beta-wave SPRs (Wilcott, 1958a). Thus, the presence of the beta component, other things equal, will normally suggest either a more excited subject or a stronger eliciting stimulus. However, the exact significance of these variations in the wave-form of the SPR, although much debated, are still something of a mystery. It is possible that they are secondary to changes in the tonic level of SP. Montagu (1958) observed that SPRs tend to be positive when the tonic SP is high-negative, negative when SP is relatively positive (i.e. low-negative), and biphasic for intermediate SP levels. Lloyd (1961) reports similar findings in recording from the foot-pad of the cat, although the positive deflections he describes are much slower than the usual beta wave and there is some doubt whether biphasic SPRs occur in infra-human species at all [Wang (1957, p. 300) denies their existence in the cat and Takagi and Nakayama (1959) obtained only alpha waves in the monkey]. Wilcott (1964) actually manipulated SP level by passing an external current between the recording electrodes, finding that all subjects could be induced to yield uniphasic-negative SPRs when the active electrode was driven positive or uniphasic-positive SPRs when the active electrode was driven negative.

Since it is quite well established that the beta wave appears as the

subject becomes more excited and at least fairly well established that diphasic or uniphasic-positive SPRs are associated with high-negative SP levels, then it should follow that tonic (negative) SP must increase with increasing arousal. Venables and Sayer (1963) report moderate R-type correlations (i.e. across subjects) -- +.68 and +.51 for two samples of schizophrenics -- between tonic SP and palmar SC, which we know does increase with arousal (vide supra). In another study, however, Venables (1963) found that SP correlates negatively with an independent indicant of arousal, the two-flash threshold (TFT) -- high-negative SPs being associated with large TFTs which indicate low arousal -- while Rose (1964), in four samples including both normals and psychiatric patients, found that high values of palmar SC were consistently associated with small TFTs. Taken together, the Rose and Venables studies imply that, at least for normal subjects, high SP values should be associated with low SC and high TFT, i.e. with low arousal. This inference also accords with Lloyd's (1961) observations, based on controlling the sudomotor activity of the cat footpad by severing the sudomotor nerve and electrically stimulating the distal stump. Lloyd reports that the resting, denervated footpad shows a low conductance and a high potential; sudomotor stimulation produces pad sweating together with an increase in SC and a decrease in SP. However, Wilcott (1964) finds that inactivating human sweat glands by atropinization lowers SP, implying that palmar sweating produced by arousal should result in high-negative SPs. In short, the psychophysiological significance of tonic SP levels remains somewhat obscure.

#### The Biophysics of Electrodermal Phenomena

The electrical activity of skin is concentrated in (a) the sweat glands and (b) the cutaneous "barrier membrane". As shown schematically in Figure 11, the epidermis consists of an outer, "horny" layer, a dry, porous cornified epithelium which is relatively thick (0.5 to 1.5 mm) in volar

skin and an inner mucus layer which is moist and electrically conductive, the two being separated by one or two thin layers of densely packed cells, the granular layer and--at least in volar skin--the stratum lucidum, only a few microns thick. Somewhere at the base of the horny layer, probably coextensive with the stratum lucidum, is a membrane (consisting perhaps of the semi-continuous membranes of the cells of this layer) which has primary responsibility for limiting the transfer of water and other matter through the skin and for resisting the invasion of the body by harmful chemicals, bacteria, ultraviolet radiation, and the like (Griesemer, 1959; Rothman & Lorenze, 1963).

-----  
Figure 11  
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In human skin, this barrier membrane is penetrated at intervals by the excretory ducts of the eccrine sweat glands which spiral downward some 3 mm or more to the coiled, secretory portion of the gland, located in the moist regions of the dermal-subdermal boundary. A cross-section of the upper or excretory portion of the tubule shows a lumen of some 15 microns diameter with a two-layer cell wall surrounded by a "basement" or limiting membrane. The secretory portion has a somewhat larger lumen, a single layer of secretory cells and, between this and the basement membrane, a single layer of muscle cells, disposed longitudinally, the myoepithelium (Weiner & Hellmann, 1960). The myoepithelial lining is rhythmically activated, by some as yet unknown mechanism, in a manner which narrows the lumen of the lower portion so that the level of sweat in the tubule periodically rises and falls (e.g. Kuno, 1956). The density of

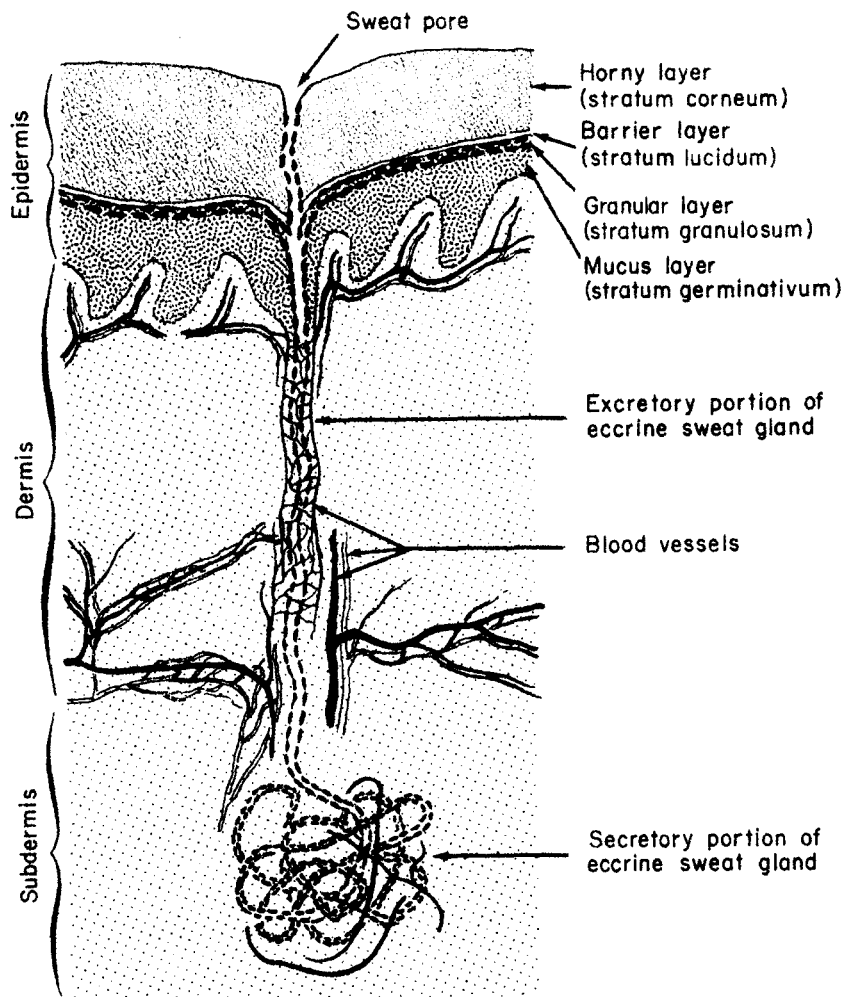


Figure 11. Semi-schematic representation of section of palmar epidermis showing eccrine sweat gland.

eccrine sweat gland distribution in man varies from about  $100/\text{cm}^2$  on the skin of the trunk to as high as  $2000/\text{cm}^2$  on the palms and soles (Weiner & Hellmann, 1960). On the palm, the density of active glands is greater on the protuberant grasping surfaces, especially the volar surface of the distal phalange (Kuno, 1956). If the average diameter of the sweat duct is about 15 microns in the epidermis (Kuno, *op. cit.*), then these openings through the barrier membrane constitute as much as one percent of its area on portions of the palms and fingertips.

Under normal conditions, there is a slight but continuous loss of water outward through the cutaneous membrane ("insensible perspiration"), amounting to perhaps 0.5 kgm or more per day (Kuno, *op. cit.*). Active sweating, in contrast, releases some 3 kgm per day (up to as high as 15 kgm/day in extreme conditions), part of which may first diffuse from the sweat pores into the dry surface epithelium, thus providing a larger area for evaporation. Sweat contains a variety of salts and organic compounds in small or trace amounts but its principal constituents include chloride, sodium and potassium ions, urea and lactic acid. The concentrations of chloride and especially sodium are lower than in plasma while potassium and lactic acid are somewhat more concentrated in sweat than in blood (Kuno, *op. cit.*). Sweat also contains an enzyme which, upon exposure to protein materials in skin tissue, synthesizes the active polypeptide, bradykinin, which apparently is responsible for active dilatation of cutaneous blood vessels (Fox & Hilton, 1958) and which, since it is a potent smooth muscle stimulant, may also be implicated in sweat gland myoepithelial activity.

The cutaneous barrier membrane is negatively charged (Rothman, 1954); amphoteric proteins in the membrane adsorb a layer of negative ions and

thereafter repel other anions, so that the skin tends to be selectively permeable to cations. This property is maintained only at normal pH; acidifying the skin will discharge the membrane or even reverse its charge. The permeability of skin has not been well studied and it is quite possible that, as is true for other biological membranes, skin permeability to particular ionic species may be independently variable, e.g. that certain chemical or hormonal influences may alter permeability to  $\text{Na}^+$  without affecting permeability to  $\text{Cl}^-$  or  $\text{K}^+$ . Larger ions generally are blocked by the membrane so that  $\text{SO}_4^{--}$  and large organic ions are generally unable to pass the barrier in appreciable quantities. An interesting possibility is that, like cell membranes, the cutaneous barrier or the sweat glands themselves possess a "sodium pump"--a metabolically energized mechanism which actively transports sodium ions inward against the existing concentration gradient. Such a mechanism is well known in frog skin (e.g. Whitfield, 1964) where it is required to prevent the loss of vital sodium by outward diffusion into the fresh water in which this amphibian normally lives. Since man is capable of a considerable sodium loss through his 1 to 4 million sweat glands (Weiner & Hellmann, 1960), active inward sodium "pumping" would seem to be adaptively appropriate and such a mechanism might well explain the fact that the skin surface is normally some tens of millivolts negative with respect to the interior.

The static electrical charge across the wall of the sweat gland tubule--between the lumen and the interior body tissue--will normally depend upon the relative concentration of ions in the two regions. If sweat in the duct has the same characteristics as have been measured in sweat collected on the skin surface, the duct should normally be negative with respect to

the interior due to the sweat being more hypotonic with respect to the positive sodium ion than with respect to the negative chloride ion. Activation of the secretory cells should increase their permeability, allowing freer passage of all ions; this depolarization of either the secretory cells or the myoepithelium following sudomotor innervation is thought to be responsible for the phasic increase of conductance measured as the GSR. Lloyd (1961) has suggested that the negative-going alpha wave of the SPR may represent the secretory action-potential; since both the secretory and the adjacent muscle cells are presumably, like other cells, many millivolts negative in their interior, depolarization of their cell membranes might yield a wave of electronegativity at the skin surface. In the inactive cat footpad, low conductance is coincident with high surface negativity, as might be expected when the membranes are polarized and the surface  $\text{Na}^+$  concentration is hypotonic to plasma. Sudomotor innervation increases conductance (due to depolarization together with duct filling--see below) and reduces negativity; i.e., the surface does change in the positive direction but slowly and tonically, with the same time constants as the conductance change, rather than quickly and phasically as in the human beta wave (Lloyd, 1961). These effects could be understood if (a) sweat as initially secreted is not in fact hypotonic--or at least not relatively deficient in sodium ion--and (b) we assume that there is inward sodium pumping within the sweat gland tubule. Then a sharp increase in sweating would increase the  $\text{Na}^+$  concentration at the surface, lowering surface negativity until the sodium pump has time to "catch up".

As mentioned earlier, there is conflicting evidence as to whether human



volar SP becomes less negative with increasing SC and increasing arousal -- as is true in the cat -- or more negative, as assumed by Venables (1963), based upon the finding of positive correlations between (negative) SP and concurrent measures of SC in two samples of schizophrenics (Venables & Sayer, 1963).

The weight of evidence appears to favor the latter conclusion, particularly Wilcott's (1964) data showing that palmar SP becomes less negative when sweat gland activity is blocked by iontophoretic introduction of atropine into the skin, a procedure which concurrently lowers SC. I have recently confirmed this finding and also observed tonic SP to become more negative while tonic SC rises as normal subjects respond to activating stimulation.

Venables' (1963) exciting finding that SP and the two-flash threshold (TFT) correlate about .70 in two samples of normals and about equally strongly but in the opposite direction in two separate groups of schizophrenics is complicated by the fact that these two putative indicants of arousal were negatively related in his normal samples (i.e. positively in the schizophrenic group) which seems unlikely on the face of it and conflicts with Rose's (1964) finding using SC in place of SP. Preliminary work in my own laboratory indicates that high-negative SP does indeed accompany low two-flash thresholds and high SC values in normal subjects, contradicting Venables. We hope soon to study this relationship in schizophrenics, hoping at least to replicate Venables' finding of a difference, although the direction of that difference, if it exists, will have to be opposite in our data to that which he reported.

It has been suggested that the phasic beta-wave of the SPR may be secondary to increased surface negativity, e.g., the result of a partial rupture of a membrane over-stressed by too strong a surface-negative

potential (Trehub, et al, 1962). Wilcott's (1964) ability to produce apparent beta-waves by driving the skin surface negative seems to lead toward a similar conclusion. However, Wilcott's data can easily be explained in conductivity terms; his "beta waves" were most probably merely GSRs -- decreases in skin resistance--- which, in a constant-current circuit like his, will always appear as decreases in the voltage across the skin and hence, under the negative electrode, as decreases in negative potential. Moreover, the beta response disappears with, e.g., exsanguination (Wilcott, 1958b) which does not systematically decrease negative SP nor eliminate the negative-going alpha wave.

A more plausible, although still wholly speculative, way of accounting for the mysterious beta-wave derives at once from the notion that the skin surface is normally more negative than the sweat gland interior, due to sodium pumping across the cutaneous membrane. Then any sudomotor response strong enough to spill the contents of a number of sweat ducts out onto the skin surface should momentarily increase total relative  $\text{Na}^+$  concentration on the surface and produce a wave of decreased (negative) potential. As in the cat, only those sudomotor responses which are strong enough to produce actual secretion and a spilling over of some ducts onto the surface would produce such a positive potential, following the initial action-potential or alpha wave. The stronger sodium pumping action in the cutaneous membrane of human volar skin -- if present as hypothesized -- might account for the relatively large positive deflections and more rapid return to the original negative SP levels which are characteristic of the human beta wave.

The fact is that the local origins of neither the alpha nor beta components of the SPR can as yet be confidently identified. The alpha wave is

almost certainly a result of some pre-secretory sweat gland activity; it is consistently recorded from a micro-electrode placed inside a single sweat pore (Takagi & Nakayama, 1959), it can be produced by stimulation of the cut end of the sudomotor nerve (Lloyd, 1961) and eliminated by local application of atropine which obstructs sudomotor innervation (Wilcott, 1964), but it is not eliminated by exsanguination which prevents actual sweat secretion (Wilcott, 1958b). If the alpha wave is an "action potential", as Lloyd (1961) suggests, it is not clear whether it reflects secretory cell activity or myoepithelial contraction -- Ebbecke (1951) believed that the myoepithelium responds to weaker innervation than the secretory cells, but there are at least equal grounds for thinking that the myoepithelial response is a secondary effect, produced either by mechanical pressure of sweat in the duct or by excitation from the bradykinin in the sweat. In any case, it is not clear why depolarization of either cell layer from the outside should produce a negative potential at the inside of the tubule. This may not be a serious difficulty, however, since these cells are small and depolarization should spread rapidly to the entire cell membrane. Thus, the entire periphery of the tubule would become rapidly negative relative to a distant reference electrode.

The beta wave, similarly, is almost certainly a secondary effect of actual sweat secretion: it is seen only when moisture can also be detected at the skin surface (Darrow, et al, 1957), it is eliminated or reduced coincidentally with the sweating response, by exsanguination (Wilcott, 1958b) or by lowering site temperature (Yokota et al, 1959) but it is not observed in SPRs recorded from a single sweat pore (Takagi & Nakayama, 1959). The true, phasic beta wave is not observed in recording

from the footpad of the cat (Wang, 1957) nor in the monkey (maccaca fusucata) (Takagi & Nakayama, 1959) and, indeed, appears to be associated with the profuse distribution of eccrine sweat glands over the entire body surface which is, in turn, uniquely human. The presence in human eccrine sweat of the bradykinin-forming enzyme would seem to be significant. Sweat-produced bradykinin has been shown to be responsible for the cutaneous vasodilatation which produces the "blushing" response on the face and neck (Fox, Goldsmith & Kidd, 1962) and it seems not unlikely that the beta wave may be an electrical by-product of some similar effect of this potent substance on tissue adjacent to the sweat pore, perhaps an effect which is related to the sodium pumping of the cutaneous barrier membrane as hypothesized above.

#### Biophysics of Skin Resistance.

Since the flow of electric current through tissue is almost entirely by means of ionic movement, the apparent electrical resistance of tissue is determined mainly by (a) the availability of charge carriers (ions), (b) frictional or inertial factors (e.g., large ions move more slowly, porous membranes may prevent large ions from passing at all and impede a proportion of the smaller ones as well), and (c) electrostatic forces (e.g., a negatively charged membrane repels anions and hence limits current flow to cationic charge carriers). Apparent resistance is very low for moist tissue, body fluids and the column of sweat in the duct of a sweat gland. The external, cornified epithelium is porous but is permeated by a lipid film (Griesemer, 1959) and may have a fairly high apparent resistance, especially when dry. The chief barriers to the flow of unidirectional or low-frequency alternating current through the skin are the cutaneous barrier membrane and the cell layers or membranes which line the sweat gland tubules. These principal current pathways are diagramed in Figure 12.

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Figure 12  
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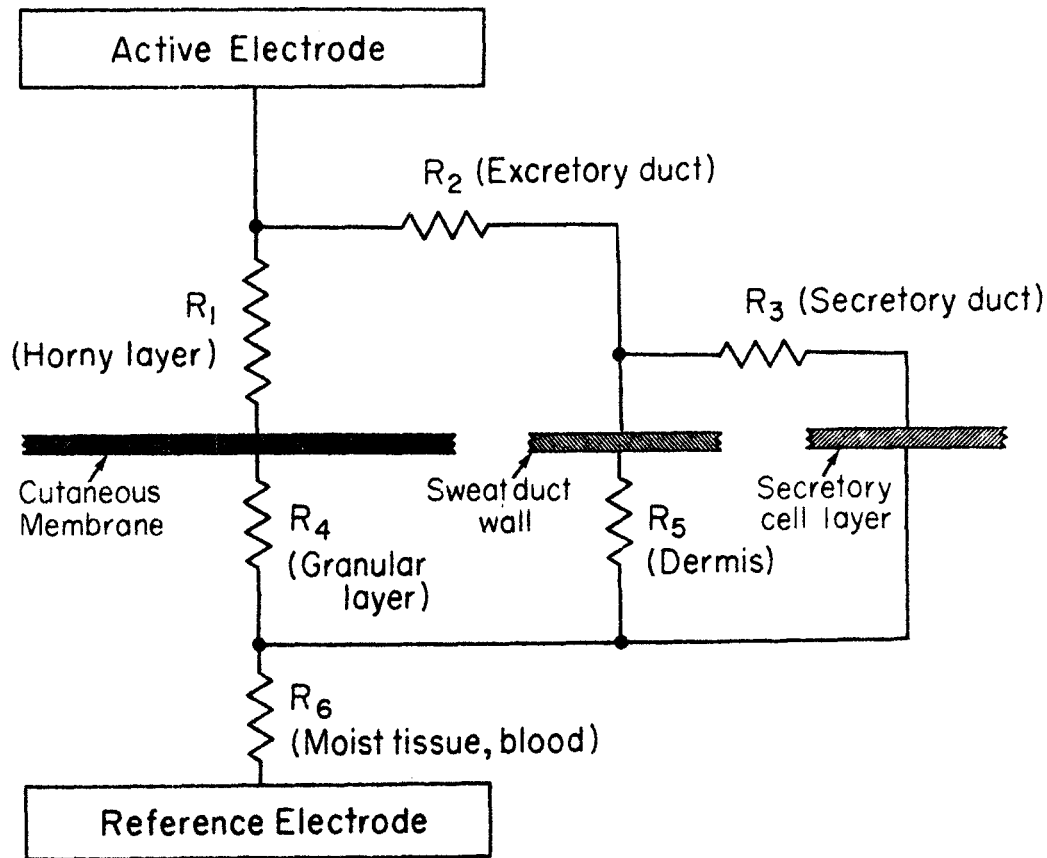


Figure 12. Schematic diagram of principal current pathways through the skin. It is assumed that the reference electrode is in direct electrical contact with subdermal tissues.  $R_1$ , the resistance of the dry horny layer, will normally be very high while the resistance of the lower, more moist layers will be lower ( $R_4$ ,  $R_5$  and especially  $R_6$ ). The resistance of the current path down the excretory duct of an eccrine sweat gland ( $R_2$  and  $R_3$ ) will be very low when the duct is filled with saline sweat but may be high when the duct is empty. All tissue layers but especially the compact cutaneous membrane and the walls of the sweat gland may generate potentials in opposition to the flow of current.

If a brief (say, 20 mSec) square wave of constant voltage (say, 0.5 volts) is applied across the skin while the current flow is observed on an oscilloscope, the current will be found to decrease exponentially during the first 0.2 mSec or so to a fraction of its instantaneous initial value, e.g., from 50 microamps/cm<sup>2</sup> to about 10 microamps/cm<sup>2</sup>. At the end of the applied voltage pulse, one can observe briefly a voltage of opposite polarity across the skin, nearly as large as the original pulse, which decays exponentially to zero within 0.1 mSec or so. These observations indicate a polarization phenomenon, a rapid build-up of a voltage or "counter emf" across the membrane which acts in opposition to the applied voltage in such a way as to reduce the flow of current. Rein's experiments (quoted in Rothman, 1954) showed that skin membranes (either the cutaneous barrier membrane or those lining the sweat glands or both) are negatively charged; this is interpreted to mean that the membrane adsorbs a number of anions which become fixed negative charges in or near the pores of the membrane (Sollner, 1955). The "counter ions" of these fixed wall charges, small cations held in proximity to the fixed anions by electrostatic attraction, have some freedom of movement through the pores of the membrane in either direction. A possible explanation of the membrane polarization phenomenon is that the application of an external voltage across the membrane causes these counter ions to move through the pores in the direction of the negative pole until the backward attraction of the fixed wall charges prevents further movement. This results in the formation of an ionic double-layer with the fixed anions on the side toward the positive pole of the applied voltage gradient and the counter-cations forming a layer facing the negative pole of the applied gradient. Such a double-layer would act as a battery in

series-opposition to the applied voltage. The initial surge of current would then represent the conductivity of the membrane as limited only by the size of its pores, its resistance to anionic flow and the like. The 100 microseconds or so required for this initial surge to fall nearly to its steady state value would be interpreted as the time required for the counter-cations to move into the orientation of the double-layer.

This polarization hypothesis [which, in one form or another, goes back to Gildemeister (cf. Forbes & Landis, 1935)] helps to account for the failure of skin resistance to obey Ohm's Law at higher current densities. At low current levels, the counter emf of polarization might be expected to increase as a constant fraction of the applied voltage, yielding a correspondingly linear increase in current. But the polarization capacity of any membrane is limited; when all available counter-cations are optimally arrayed in the ionic double-layer, the counter emf can increase no further so that additional increase in applied voltage must be accompanied by a sharp increment in current flow. Moreover, it is likely that high external voltages would actually discharge the membrane, stripping away the adsorbed anions and thus reducing its polarization capacity until normal metabolic processes are able to restore the initial conditions. It is for this reason, perhaps, that apparent skin resistance decreases with increasing current density above a level of about 10 microamperes/cm<sup>2</sup> (Edleberg, Greiner & Burch, 1960).

If, while observing current flow through palmar skin using the experimental arrangements described above, one then stimulates the subject so as to produce a GSR, it will be found that this phasic increase in apparent conductance is observed only as a transitory increase in the asymptotic level

to which current falls after polarization; the GSR affects neither the maximum amplitude of the initial current pulse nor the rate at which the current falls during the ensuing 100 microseconds or so. This would seem to indicate that the local effect of GSR activity is one of momentarily decreasing the polarization capacity of the skin membrane. A similar conclusion was drawn by McClendon and Hemingway in 1930 and again by Forbes and Landis in 1935 as a result of observing that the GSR, in the form of a decrease in apparent impedance to a sinusoidal applied voltage, seemed to disappear when the frequency of the applied waveform was increased beyond about 10,000 cps. Their observations, however, might be accounted for merely in terms of capacitative shunting. The thin, relatively non-conductive membrane functions as a capacitor to alternating current, shunting an increasing proportion of the applied current as frequency increases. Even if the GSR were some sort of decrease in the ohmic component of apparent impedance (e.g. an increase in the level of sweat in the ducts or an increased porosity of the membrane), this increase in parallel conductance would be of less and less importance--i.e. less observable--at high frequencies due to the great decrease in capacitative reactance of the membrane. The square wave analysis described above indicates, however, that there is no decrease in parallel impedance during the GSR but rather only a decrease in apparent polarization capacity.

The locus of this variable polarization capacity in skin is probably in the membranes of the sweat gland secretory cells or myoepithelium. However, at higher rates of sweating, GSR activity (i.e. a phasic increase in sudomotor innervation) may quickly increase sweat concentration in the horny layer, which may in turn affect the polarization capacity of the



cutaneous membrane. Thus, the conductance GSR accompanying a typical biphasic SPR may consist, first, of a depolarization of secretory cells, providing additional high-conductance pathways for current flow through the skin, followed by a partial depolarization of the cutaneous barrier, which would increase the conductivity of the direct pathway. After exsanguination of the limb, sudomotor innervation continues to produce secretory cell depolarization accompanied by the alpha wave; however, the lack of sweat prevents any effect upon the cutaneous membrane and hence prevents the beta wave and greatly reduces conductance change. When the residual sweat in the ducts has all been reabsorbed, depolarization at the base of the sweat gland no longer provides a low resistance shunt through the skin and the conductance GSR may be then entirely eliminated.

#### Conclusions

This relatively brief review of the voluminous and controversial literature concerned with electrodermal phenomena will have illustrated that this technology has not yet stabilized; nearly every investigator uses some unique combination of electrodes, electrode placement, signal conditioner and recording method, current level, electrolyte constituents and concentration, and so on--and we have seen that each such variation can influence, sometimes drastically, the measurements obtained. In spite of these complexities and although many investigators have employed such poor techniques as to render their findings difficult to interpret, electrodermal phenomena continue to be widely used by psychologists in psychophysiological researches, in studies of classical conditioning, in studies of emotional reactivity, arousal, reactivity to pain, habituation, in the study of perceptual defense and in a host of practical applications.

The fact that this dependent variable has provided so many provocative results in such a diversity of applications, in spite of the lack of a standard technology and in the face of so many examples of clearly inadequate technique, implies that these phenomenon must be unusually rich in psychological significance to have survived so much careless handling so well. The mass of findings available supports quite firmly the general conclusion that skin conductance (and presumably also skin admittance, positive palmar potentials, and frequency of non-specific GSRs) varies monotonically with some basic dimension of psychological activation or arousal. Similarly, it seems clear that the phasic conductance change or GSR (and presumably the negative-going skin potential change or SPR) varies in amplitude directly with what can best be called the subjective intensity of the eliciting stimulus or, alternatively, the attention-value of that stimulus.

The problems of electrodermal methodology have been gone into here in some detail (although a really adequate discussion of this extensive literature would have required a volume to itself) for two reasons. First, these phenomena appear to comprise the most important psychophysiological variables currently available for use in general psychological research (the electrical activity of the brain--the EEG, evoked potentials, and the like--are no doubt the most important in an absolute sense but not as they are currently accessible to and interpretable by the average psychologist). Secondly, it seems necessary to remind some psychologists, who find themselves in increasing numbers turning away from paper-and-pencil techniques to the more promising tools of the experimental laboratory, that these procedures are complex and cannot be correctly used merely by

following cookbook formulas. The psychological investigator, like his colleagues in the other experimental sciences have long since done before him, must adapt himself to the need for mastering an increasingly elaborate research technology. At the other end of the continuum of scientific evolution, nuclear physics has produced a division of labor between the experimenter on the one hand the the theoretician on the other whose laboratory is his blackboard; one wonders whether present day psychology can afford that luxury.

#### A Method for Direct Measurement of Apparent Skin Conductance

Although the partial account of the biophysics of electrodermal phenomena given above seems plausible on the existing evidence, it should be re-emphasized that it is largely speculative and that most of these issues are still quite unsettled. Considerable additional research will be required to determine which, if any, of these speculations may be true and to answer the remaining questions before one can expect workers in this field to settle upon a standardized technology. Meanwhile, the non-specialist might be wise to avoid the major mysteries of the skin potential methods. Alternatively, the direct measurement of apparent skin conductance recommends itself as relatively easy to instrument, quite simple to use and reasonably immune to major artifacts.

The usual practise of measuring apparent skin resistance and then converting the resistance values into units of conductance is both onerous and subject to considerable error under certain conditions (Lykken & Roth, 1961). A better approach is to measure SC directly by applying a small constant voltage to the skin and measuring variations in current. Since this tissue obeys Ohm's Law to a reasonable approximation, as long as

current densities are maintained below about 10 microamps/cm<sup>2</sup>, current in this range will vary linearly with conductance in such a constant-voltage circuit. A suitable signal conditioner for this purpose is diagramed in Figure 13. This circuit is designed for use with a single active

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Figure 13  
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finger electrode and a typical high-gain DC pre-amplifier of the chopper-stabilized type (vide infra). The amplifier should be equipped with a zero-suppression control (with which one can subtract a calibrated amount from the input voltage) and the usual step-type input attenuator switch (which adjusts the amplification of the net input signal to various calibrated fractions of the maximum gain of the amplifier). The voltage across the subject is set by adjusting P<sub>1</sub> to a value which will insure that the current density will never exceed the limit of 10 microamps/cm<sup>2</sup> for the particular electrodes used. With a single 1 cm<sup>2</sup> finger electrode and the skin punctured at the reference site, minimum skin resistance to be expected is about 50 K-ohms; hence, a proper subject voltage would be about

$$E_s = (50 \times 10^3) \text{ ohms} \times (10 \times 10^{-6}) \text{ amperes} = 0.5 \text{ volts.}$$

Because the output signal is the voltage dropped across the small series resistance, R<sub>s</sub>, rather than across the subject, the pre-amplifier is not required to have a high-input impedance. Moreover, this low resistance in series with the subject essentially "short circuits" endogenous skin potentials, preventing SPR activity from affecting the conductance measurements even at very low current levels.

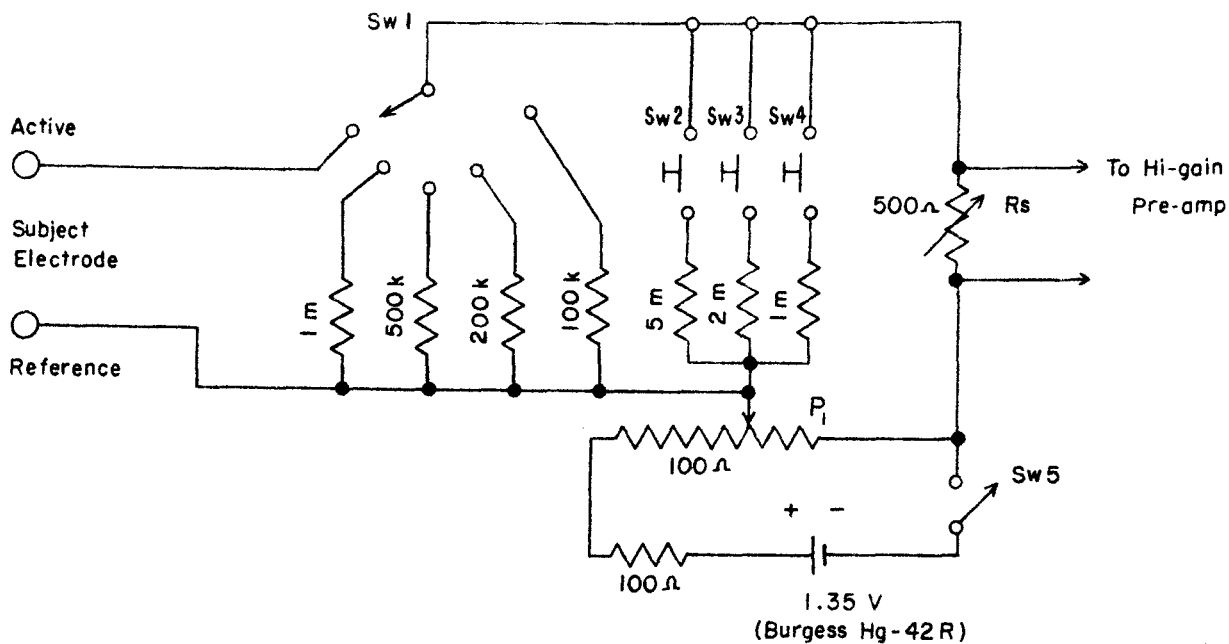


Figure 13. Sensor or signal conditioner for use in the direct measurement of skin conductance (SC). Voltage across subject, determined by setting of  $P_1$ , remains essentially constant so that current through subject, producing output voltage across  $R_s$ , will vary linearly with subject's apparent conductance. Details of operation are given in text.

Calibration of the system is accomplished in two steps. First, a known conductance is substituted for the subject by appropriate setting of  $Sw_1$  and then the size of the output signal, determined by the setting of  $R_s$ , is adjusted so that the zero-suppression control on the pre-amplifier (usually a 10-turn potentiometer calibrated to read from 0.00 to 10.00) can be read directly in micromhos or in even-multiples thereof. For example, with  $E_s = 0.5$  V and  $Sw_1$  set at 200 K-ohms (5 micromhos), current flowing through  $R_s$  will equal 2.5 microamps; thus, the voltage across  $R_s$  can be varied from zero to  $(500)(2.5 \times 10^{-6}) = 1.25$  mV. If the zero-suppression on the pre-amplifier can be set to suppress 1 mV at the maximum setting of 100.00, then--to make this control read directly in micromhos-- $R_s$  should be adjusted downward to give an output of 0.5 mV for this 5 micromhos conductance (which will then be just cancelled by a zero-suppression setting of 05.00).

Secondly, the gain of the pre-amplifier must be adjusted so that a given change in conductance will produce a known deflection of the recording pen. Amplifier input-attenuator switches are typically calibrated "X1, X2, X5, X10, ..., X1000"; set at X1, the gain is maximum, while set at X10, the gain is one-tenth maximum, and so on. Therefore, it is convenient to adjust the gain control of the pre-amplifier so that a conductance change (e.g. a GSR) of 1 micromho produces full-scale pen deflection at X1, half-scale at X2, and so on. This may be done using the push-button switches  $Sw_2$ ,  $Sw_3$ , and  $Sw_4$  on the signal conditioner. Thus, with the amplifier attenuator at X1, pressing  $Sw_3$  adds a conductance of 0.5 micromhos to that already in the circuit and should produce half-scale deflection of the recorder once the amplifier gain control has been properly adjusted.

In use, the subject is inserted in the circuit by means of  $Sw_1$  and the

zero-suppression control is adjusted so as to bring the recorder pen to some convenient position on the chart. One then writes down the setting of this control (e.g. "03.64 micromhos") together with the setting of the attenuator (e.g. "X2") directly on the chart paper. From this information, one can determine with precision the subject's exact momentary SC value for any position of the pen on the chart. Another important advantage of recording SC directly is that the variations in the subject's tonic conductance during the experimental session tend to be much smaller than his corresponding variations in tonic resistance, relative to the amplifier sensitivity required for measuring phasic changes. This means that the pen is not continually drifting off the chart, requiring range-changing or manual resetting of the zero-suppression.

#### Electrodes for Bio-electric Measurement

Measurement of most bio-electric phenomena requires that a connection be made between the measurement apparatus and the tissue at two or more junctions which are known as electrodes. At the electrode, the manner in which current is transported changes from electronic in the wires to principally ionic conduction in the tissue. This transition necessarily implies that chemical processes occur at both electrodes, resulting in the production of electrode potentials or resistances which may seriously distort the measurements one is attempting to make. From the standpoint of the psychophysicologist, such problems are of greatest importance in relation to electrodermal measurement although poor electrode technique can also create difficulties in EEG and EMG recording as well as in the administration of electrical stimulation or shock.

## Electrode Potentials.

When any metal is immersed in an electrolyte, the metal tends to discharge cations into the solution, this tendency being stronger for the more active metals. At the same time, metal cations in the solution tend to transmit their positive charge to the metal electrode in proportion to their concentration. If the "dissolving pressure" of the metal is greater than the osmotic pressure of the cations in solution, the metal shows a net loss of positive charges to the solution and this becomes relatively negative; if the osmotic pressure predominates, the electrode potential will be positive. Although it is not possible to eliminate such electrode potentials entirely, two identical metals immersed in identical electrolytes will show no voltage difference with respect to one another. However, any difference in concentration in the ions in solution at the two electrodes will produce a net "concentration potential" between them which can be calculated from the equation

$$(1) \quad E_c = \frac{RT}{nF} \left[ \log_e \frac{c_1}{c_2} \right]$$

where R is the gas constant, T the absolute temperature, n the ionic valency, F is the faraday, and  $c_1$  and  $c_2$  are the respective concentrations of the relevant ion in the two solutions. Converting to Briggsian logarithms and simplifying:

$$(2) \quad E_c = 60.6\text{mV} \left( \log \frac{c_1}{c_2} \right) \text{ at } 30^\circ \text{ C.}$$

Thus, for example, if the electrode paste used at the active electrode on the skin contains 0.1N NaCl while the electrolyte at the reference electrode contains 0.01N NaCl, then the active electrode (having the more concentrated solution) will prove to be some 60 mV positive with respect to the



reference due to concentration potential alone, even if the tissue is electrically inert.

### Liquid-junction potentials.

At the junction between any two liquids containing salts in different concentrations, ions will tend to diffuse in the direction of lower concentration, the more mobile ions diffusing more rapidly. As a rule, the diffusion of either positive or negative ions will be faster, resulting in an imbalance of electric charge across the liquid-junction. If a solution of 0.1N NaCl is connected to a solution 0.01N NaCl by a "salt-bridge" made of cotton wick soaked in the latter solution, one can measure a voltage between the two solutions which can be computed from the formula:

$$(3) \quad E_d = \frac{u-r}{u+v} E_c = \frac{u-r}{u+v} (60.6 \text{ mV}) \log \frac{C_1}{C_2} \text{ at } 30^\circ \text{ C}_3$$

where  $v$  and  $u$  are the mobilities of the negative and positive ions respectively. The mobility of  $\text{Na}^+$  ( $u$ ) is about 4.5 while the mobility of  $\text{Cl}^-$  ( $v$ ) is 6.8; therefore,

$$E_d = \frac{-2.3}{11.3} [60.6 \log \frac{0.1}{0.01}] = -12.4 \text{ mV};$$

(i.e., the more dilute solution negative).

Since the mobilities of the potassium and chloride ions are so nearly equal, salt-bridges saturated with a solution of KCl are commonly used as a means of avoiding liquid-junction potentials. If these same NaCl solutions have been connected by a saturated KCl bridge, the diffusion of  $\text{K}^+$  and  $\text{Cl}^-$  into solution at both ends of the bridge would have occurred at about the same rate leading to a negligible net junction potential on the order of 1 mV.

## Membrane Potentials.

When two solutions are separated by a semi-permeable membrane, differences in ionic concentration will produce a "diffusion" potential across the membrane as at any liquid junction. However, the membrane may greatly alter the effective mobilities of the various ionic species involved. Thus, a membrane with small pores may be far less permeable to the  $\text{Na}^+$  ion, which is hydrated and large, than to the  $\text{K}^+$  or  $\text{Cl}^-$  ions, so that different dilutions of NaCl will yield a much higher potential across such a membrane than the same dilutions of KCl. A negatively charged membrane such as skin is relatively impermeable to anions regardless of their size. Therefore, only cations will diffuse across the membrane and the potential will depend only on cationic concentrations; since, in equation (3),  $v = 0$ , we get:

$$(4) \quad E_m = \frac{u}{u} [60.6 \log \frac{c_1}{c_2}] \text{ mV} = 60.6 [\log \frac{c_1}{c_2}] \text{ mV at } 30^\circ \text{ C.}$$

If the membrane separates salts having different cations, the membrane potential can be calculated from the somewhat more general formula:

$$(5) \quad E_m = 60.6 [\log \frac{u_1 c_1}{u_2 c_2}] \text{ mV at } 30^\circ \text{ C,}$$

where  $u_1$  and  $u_2$  are the mobilities in the membrane of the two cations and  $c_1$  and  $c_2$  are their respective concentrations. In the negatively-charged collodion membrane, the mobility of  $\text{K}^+$  is some 7.5 times the mobility of Na (Bures, et al, 1960), so that such a membrane separating solutions of NaCl and KCl in equal concentrations will produce a membrane potential of about  $60.6 [\log 7.5] \text{ mV} = 53 \text{ mV at } 30^\circ \text{ C}$ . The relative mobilities of various cations in skin have not been determined; the possibility of active sodium suggests that these mobilities may not be the same in both directions and

it must also be expected that the mobility of (i.e. permeability for) different cations in skin may vary considerably with local conditions. The basic point to remember is that any difference in ionic (especially cationic) concentration between the local body fluids inside the skin and electrode fluid used on the skin surface will in general produce a membrane potential which may be on the order of tens of millivolts.

#### Types of Electrodes.

Even with the same metals and identical electrolyte concentrations, it proves to be quite difficult in practice to produce pairs of electrodes whose potential difference remains low (e.g. less than a few millivolts) and stable over time. Minute impurities in the metal or in the electrolyte, producing complex and cumulative chemical reactions, are probably responsible for these problems. When current is passed between any electrode pair, as it must be even in the measurement of potentials, the difficulties are compounded. The flow of current sets up diffusion potentials within the electrolyte, due to local variations in ionic concentration. Most important, chemical reactions occur at both metal-electrolyte junctions which may bring about marked and cumulative changes in the apparent resistance of the electrodes due, e.g. to progressive changes in electrolyte composition or the deposition (plating) of insoluble metallic salts upon the surface of the electrode metal. So-called "non-polarizing" electrodes have the property that such reactions are completely reversible when the direction of current flow is reversed; i.e. no reaction products are lost through precipitation or the evasion of gas. Most non-polarizing electrodes consist of two types; (1) a metal plate separated from the tissue by a solution containing a salt of that metal (e.g. a zinc plate used with a zinc-sulphate electrolyte)

or (2) a metal plate coated with an insoluble salt of that metal and separated from the tissue by a solution containing the same anion (e.g. a silver electrode coated with silver chloride and used with a KCl or NaCl electrolyte).

However, not all metal-salt combinations yield suitable electrodes; the processes involved are surprisingly complex in detail so that electrode design is based to a considerable extent upon trial and error. Measuring electrodermal potentials probably presents the most difficult problem, requiring electrodes having stable bias potentials of about a millivolt or less. EEG, EKG and EMG potentials are essentially AC phenomena so that fairly large electrode potentials can be tolerated; the main desiderata here are convenience, secure contact with the tissue to prevent movement artifacts, and low electrode resistance. The latter is accomplished by breaking or discharging the cutaneous membrane; i.e. by puncturing the skin or by rubbing in a strongly hypertonic electrode paste (one commercial EKG paste contains pumice grit which seems to facilitate this process). Skin conductance measurement requires stable electrodes with relatively low bias potentials and the ability to pass a small unidirectional current without excessive build-up of apparent electrode resistance. The use of the so-called "dry electrode", in which the electrode metal is placed directly against the skin, depends upon the tissue fluids to supply the necessary electrolyte, with its composition, concentration and amount being outside the control of the experimenter. This practice is naive and to be avoided in any kind of careful work. Most of the electrode systems which have proven useful in psychophysiological applications are described briefly below.

The Calomel Electrode. This is a high quality non-polarizing electrode widely used by neurophysiologists for recording nerve action-potentials and the like. Distilled mercury is covered by a layer of calomel ( $\text{Hg}_2\text{Cl}_2$ ) which is covered in turn by a solution of KCl. Contact with the tissue is commonly made by means of a wick soaked in an isotonic NaCl solution. Detailed instructions for constructing the calomel electrode may be found in Bures et al (1960) or in Whitfield (1964) and they are also available commercially.

The Silver-Silver Chloride Electrode. Perhaps the best quality electrode suitable for direct attachment to the skin, the Ag-AgCl electrode is made by electrolytically depositing a layer of silver chloride on a pure silver base and is employed with an isotonic (or slightly hypotonic) NaCl electrolyte. In one version, a helix of platinum wire is coated with silver-oxide which is then reduced by baking, leaving a porous "sponge" of silver having a very large surface area. After plating with silver-chloride, such electrodes may show extremely low bias potentials and excellent stability. Detailed instructions can be found in Feder (1963) and in O'Connell and Tursky (1960) and commercial versions are also available (e.g. from Beckman or Lexington Instruments).

A disadvantage of the silver-sponge type electrode is that it is somewhat fragile and difficult to clean and re-chloride in the event of drying or contamination. Although their bias potentials may be on the average somewhat higher, chlorided silver discs have the advantage of being easier to make in the first place and easier to repair when damaged. Venebles and Sayer (1963) describe a technique in which a pure silver disc, set in a rubber grommet, is chlorided by being made the anode in an electrolytic bath of 0.5% KCl through which a current of 0.5 Ma is passed for one hour.

These electrodes commonly show a bias potential of less than 0.1 mV but the chloride coat is rather fragile and must be protected from injury or drying. A more durable version, developed by Ralph Miller, may be made by mounting the silver disc permanently in a plastic housing prior to plating. The finished electrode is then inserted into a collar of soft plastic tubing which projects slightly beyond the chloride surface thus affording some protection to it. This assembly, having an o.d. of 3/8 inch, is then filled with electrode paste and inserted into the opening of a felt corn pad mounted on the skin. The whole assembly can then be secured with surgical tape. If damaged, the electrode is easily disassembled, sanded clean and re-plated. These electrodes should be stored in small groups, short-circuited in .07N NaCl. Before using, it is best to check the bias potentials of various pairs, selecting that pair showing the lowest potential difference. Still another disc-type Ag-AgCl electrode, used in the NASA Mercury program for EKG recording, is described in detail by Day and Lippett (1964).

The Zinc-Zinc Sulphate Electrode. This electrode is most commonly made in the form of a pure zinc disc sealed in plastic, sanded bright and clean before each use, and coated with a paste containing zinc sulphate. For electrodermal recording, the zinc sulphate electrolyte has the disadvantage that the zinc cation tends to depolarize the tissue, decreasing apparent skin resistance and affecting skin potential as well. One way to minimize this difficulty is to separate the zinc sulphate from the skin by a layer of KCl or NaCl paste or by a sponge soaked in a dilute solution of one of these chlorides. Another method is to use only one active electrode which is connected to the negative pole of the external current source; the skin

being highly impermeable to the sulphate anion, the only ionic movement at the active site will be migration of cations outward from the body fluids while depolarization at the reference site can be accomplished at the outset by puncturing the skin. In the case of skin potential recording, the active or palmar electrode is normally negative anyway and the zinc sulphate electrode should be adequate. This type of electrode typically has a somewhat higher electrode potential and electrode resistance than the best examples of the silver chloride electrode, but is considerably easier to make and to use. As a general practice one should have a number of pairs of such electrodes available and use a pair having a minimum electrode potential when immersed in physiological saline.

The Lead Electrode. A very simple electrode for electrodermal recording and one which is also useful for EKG and EMG work can be made from a disc of high purity lead. As with the other disc electrodes mentioned above, the metal disc with the lead wire soldered to its back side should be cemented tightly into some type of plastic housing so that only the single metal surface is exposed, surrounded by a collar of plastic. The exposed lead surface should be sanded bright before each use and may contact the skin surface through a standard KCl or NaCl electrode paste. One can usually find pairs of such simple electrodes which will maintain bias potentials of less than three millivolts in saline over a period of several hours. In electrodermal work, the single element lead electrode should be considered only for potential recordings, where the flow of current is negligible; even a few microamperes of current will soon build up a substantial amount of polarization on these electrodes, giving high electrode resistances and potentials. Whitfield (1964) describes a non-polarizing lead-lead chloride

electrode which may also have applications in electrodermal measurement.

The Two-Element Electrode. Another type of electrode has been described (Lykken, 1959) for use in measuring skin resistance, consisting of a small metal disc surrounded by, but electrically isolated from, an annular ring element made from the same metal. After separate lead wires have been attached, the disc and ring elements are cemented flush into a plastic housing, the face of which exposes the central metal disc, a ring of plastic, the metal ring element and then a final outer ring of plastic. As used for skin resistance measurements, the ring elements of a pair of these electrodes are connected to the external source of current; the center disc elements are connected to a separate, high impedance voltage measuring instrument (e.g., a DC amplifier and associated strip-chart recorder). Since all the significant current flow in this arrangement is between the ring elements, electrode polarization effects are largely limited to the surfaces of these rings and to the immediately adjacent portion of the electrode paste. The potential registered between the relatively uncontaminated central discs is therefore a very accurate representation of the voltage (IR) drop through the skin. With such electrodes made of high-purity lead, and used with an ordinary saline electrolyte, one can measure skin resistance accurately but at the expense of providing an electrically isolated constant-current supply.

Other Electrode Considerations. Since contact with the skin is made through the electrolyte rather than by the electrode metal proper, the effective area of an electrode is determined not by the size of the metal disc but by the area of skin surface wet with electrolyte. Since apparent electrical conductance of the skin varies directly with effective area, this



parameter must be held constant in SC measurement. Some electrodes are enclosed in a cup-like plastic housing, the lips of which press against the skin to contain the spread of the electrolyte. This arrangement is subject to artifacts from movement and pressure variation, however; a better method is to demarcate the effective skin area with a piece of wide surgical adhesive tape having a hole punched in its center. Then an ordinary corn pad is used between the skin and the electrode proper as described by Lykken (1959). (I have found that the adhesive supplied on one side of the corn pads is not really sticky enough to assure a good seal to the skin and so have resorted to using the surgical tape in addition). It would be a help if investigators would follow the practice of reporting the exact location of their active electrodes in SC measurement and specifying the apparent skin conductance per square centimeter of effective skin area. In measuring skin potentials, as well as in EKG work and the like, controlling electrode area is of much less importance, except where the skin surface might become saturated with saline sweat and this "short circuited" over a broad expanse.

The best electrolyte for most electrodermal work will be a chemically inert, non-drying paste or cream, somewhat thinner in consistency than ordinary cold cream, and containing a 0.07 molar solution of NaCl, which will be approximately isotonic with the principal ionic constituents of surface sweat. Such a paste can be made by boiling the salt solution with a small quantity of agar (about 4%), stirring the mix as it cools. A still easier method is to add sufficient water and salt to a neutral ointment base (e.g. Parke-Davis' "unibase") to produce a mixture of suitable consistency and salt concentration. For use with zinc electrodes, zinc-sulphate may be

substituted for the NaCl or, better, the electrode metal can be coated with a stronger  $ZnSO_4$  paste (say, 0.5 N) while the skin is coated the usual isotonic NaCl paste. For EEG, EKG and EMG work, minimum skin resistance is desirable so that a strong, depolarizing electrolyte is appropriate. An agar or Unibase paste containing a 3.0 N-KCl solution should be a good choice (see also Day & Lippitt, 1964).

Reference electrodes should always be located in electrically inactive regions and should show minimum apparent resistance. For electrodermal recording, both inactivity and low resistance can be insured by puncturing the skin under the reference electrode using the skin-drilling technique described by Shackle (1959). If the barrier region cannot be broken by puncturing or sanding, its apparent resistance can be reduced by soaking in saturated KCl or a weak acid (e.g. vinegar) and using a large effective electrode area. Generally the best location for the active electrode in electrodermal work is on the palmar surface of the distal phalange of the fingers, where sweat gland distribution is most dense and GSR activity is maximal. (The second phalange, a site recommended by Edelberg & Burch (1962), has the advantage that this skin is less likely to show cuts or other damage but it also contains many fewer sweat glands). Alternatively, the palms, the soles or even the forehead or chest might be employed, but since individual differences in sweat gland distribution and GSR activity are so great, the finger location recommends itself wherever possible.

The proposed site should be examined, preferably under low-power magnification, for tiny cuts or abrasions which might provide a short-circuit pathway for current through the epidermis. The skin may be rubbed lightly with alcohol or ether to remove surface oils before application of

the electrodes (recent soaking in strong detergents may sharply alter SC and GSR findings). One difficulty with the finger location is the necessary restriction imposed upon electrode area. Other things being equal, a larger electrode area will contact a larger and more stable population of sweat glands, minimize errors resulting from small variations in area, and also allow for a reduction in average current density when measuring SC directly (see above). One way to obtain larger areas with finger electrodes is to use separate electrodes on two or more fingers. Each finger can then be tested separately against the reference as a check on possible artifacts (each active electrode should show about the same apparent SC) and then all of them can be tied together electrically to serve jointly as the active electrode.

Electrode technique is particularly critical in skin potential measurement. The electrodes themselves must of course have negligible (and stable) bias potentials. Moreover, as implied by the foregoing discussion of concentration and membrane potentials, the potential actually measured may be strongly affected by the salt used in the electrolyte, its concentration, and the relative permeability of the skin at the active and reference sites.

#### Electrodes for Shock Stimulation.

The administration of electric shock stimulation through skin electrodes has been greatly illuminated by the recent work of Tursky and Watson (1964). These authors have shown that variations in apparent skin resistance, including those produced by the passage of the shock current itself, produce changes in the subjective intensity of the stimulus no matter what sort of control is applied to the physical stimulus (e.g. maintaining the shock current or voltage constant). Their solution is to employ a concentric disc electrode

applied to a skin area previously treated by rubbing with a depolarizing electrode paste, which reduces skin resistance to a low and stable level. Thereafter, either constant-voltage or constant-current stimulation will produce stable subjective stimulus intensities. The Tursky and Watson method can be recommended as a standard procedure and the above reference should be consulted for details.

### Instrumentation for Psychophysiological Research

Sensors or Transducers. The kymograph, with its smoked drum and its styli driven by mechanical or pneumatic linkages to the preparation, has gone to an honorable retirement along with the string galvanometer and those many giants of physiological research who somehow managed to discover so much with such primitive tools. Modern electronics has made it possible to amplify, record and analyze electrical signals with extraordinary ease and fidelity. The first step (and frequently the weak link) in almost all current physiological measurement is to convert or transduce the phenomenon of interest into an electrical signal having corresponding amplitude or temporal characteristics. A thermister can be used as a temperature transducer because its electrical resistance varies with temperature over a wide range. A strain gage is a carbon or semi-conductor element whose resistance changes as a function of mechanical strain and, suitably mounted, can be used to convert pressures, positions and movements into electrical signals. A pH meter uses a conductivity cell to transduce the acidity (hydrogen ion concentration) of a test solution into an electrical potential. (Technically, recorders of all kinds are transducers also, converting the amplified electrical signal back into readable or storeable form, but convention reserves the term for transducers whose output is electrical.)

The term "sensor" includes transducers and applies also to the measurement of phenomena which are intrinsically electrical in nature, including the electrodes and signal conditioners which may intervene between the preparation and the amplifier-recorder.

#### Amplifiers.

The power amplifier is designed to drive whatever recording equipment is to be used--oscilloscope, tape recorder head, recording galvanometer--and normally has a high-impedance input of some 1 to 10 volts sensitivity. The pre-amplifier drives the power amplifier and is designed with input and amplification characteristics to match appropriate transducers. AC-coupled pre-amplifiers are relatively inexpensive and can combine high sensitivity with good stability but cannot handle DC levels or very low frequency signals. Until recently, stable DC pre-amplifiers of high (e.g. 1 mV) sensitivity were difficult to obtain. A common solution to the problem of designing stable DC amplifiers is to use chopper-stabilization; the DC input signal is "chopped" by an electrical or mechanical switch and the resulting "AC" signal is fed to the primary of an input transformer from whence it passes to a conventional AC-coupled amplifier. The amplified signal is then converted back again to DC at the output. Chopper-type amplifiers have the limitation that their input impedance is normally too low for some applications (e.g. from about 5 K-ohms). Unless the input impedance of the amplifier is high relative to the impedance of the signal source, an appreciable fraction of the signal voltage will be dropped across the source impedance and, hence, not recorded. Where the signal source consists of a pair of skin electrodes (as in SC or SP recording), the pre-amp input impedance must be at least one megohm to prevent degradation of the signal.

Electrometer amplifiers are especially designed to have the very high input impedance ( $10^9$  ohms and above) required for use with high-impedance micro-electrodes.

An important property of physiological amplifiers is the ability to reject in-phase signals or noise. Electrostatic fields in the surround commonly induce radio-frequency voltages at the electrodes or in the connecting cables, voltages which may be large in relation to the small signals one wishes to record. An analogous problem arises in attempting bioelectric recording from a subject who is also being electrically stimulated or shocked. Frequently these "noise" potentials, although large with respect to ground, may be instantaneously identical or nearly so at the active and the reference electrodes, providing neither is grounded. If neither amplifier input is grounded (i.e. "floating" inputs) or if the amplifier is of the differential or "push-pull" input type, such in-phase noise can be rejected although many times greater in amplitude than the desired signal. Useful discussions of methods of eliminating stimulus artifact and noise may be found in Becker et al, (1961) and in Guld (1961).

### Recorders

Because it paints its picture with an almost inertia-less electron beam, the oscilloscope provides the most accurate representation of high-frequency electrical activity. Long persistence cathode-ray tubes can be used to retain the image for several seconds or the image can be photographed for permanent storage. Recording galvanometers using light-sensitive paper and mirror galvanometers which reflect an intense light beam upon the moving paper can accurately record signals up to 10,000 cps. Many channels of information can be recorded simultaneously on a single 10-inch chart and a

useful characteristic of the light-beam recorders is that one or more channels can be interlaced--i.e. one or more of the galvanometers can be allowed to write over the full chart width rather than remaining in their respective narrow tracks. Most oscillographs or "direct writers" draw their graphs of voltage against time by means of galvanometer-driven pens or heated styli writing on conventional or heat-sensitive paper which is driven at a constant speed beneath them. Typical oscillographs of the type used for EEG, EKG and GSR recording may be obtained with from one to 12 pens, a frequency range of from DC to about 100 cps, and a suitable range of chart speeds. Older oscillographs have the disadvantage that the written waveform is distorted as a result of the pen's being mounted on a stationary pivot and moving in an arc. Recti-linear coordinates are provided by systems using a heated stylus which wipes the paper as it is pulled over a straight-edge and also by modern pen-writers employing special linkages between the pens and the pen-motors. The simplest oscillographs are recording milliameters in which the pen-motor is simply a robust one-milliampere meter movement; these instruments commonly have a fairly wide chart and a low frequency capability but are quite adequate for, say, GSR recording.

The potentiometric recorder employs a servo-mechanism which automatically adjusts an internal potential to match the applied signal until zero input current flows. A pen is simultaneously driven across the recording paper to a position corresponding to the setting of this internal voltage. Like the recording milliammeter, the potentiometric recorder is a relatively low-speed device, requiring from about 0.1 to as much as 2 seconds for full-scale excursion of the pen. However, the potentiometric recorder has very high

input impedance (nearly infinite at balance), compared to about 1000 ohms for the recording milliammeter, and a high enough sensitivity (often as high as 1 mV full-scale) to be used without additional amplification. The X-Y recorder uses a single rectangle of stationary chart paper over which the pen is moved both vertically and horizontally by two independent servo-systems. This instrument is frequently used as a computer-output device.

An important recent improvement in psychophysiological instrumentation is the use of magnetic-tape data recording. Most commonly, the input signal--which may vary in frequency from DC to several thousand cps--is amplified and then used to frequency-modulate a carrier signal which is recorded on the tape. During playback, this FM signal is de-modulated to reproduce the original input exactly. As many as seven or more separate channels may be recorded on a single tape, usually with an audio channel also provided, at a recorder cost of about \$1000 per channel complete with input and output electronics. Thus equipped to store and reproduce the "raw data" in its original form, the experimenter is able after the experiment to review the data at his leisure, using e.g. an oscilloscope; to make written records of only those aspects which prove to be of interest by feeding the tape output to an oscillograph; to make convenient use of electronic methods of data analysis; and to recapture aspects of the data which might have been lost with more limited methods of on-line recording. When the recorder is equipped with both low and high tape speeds, high-frequency signals can be slowed down for oscillograph recording--by recording at high speed and played back at low speed--or, conversely, slow or intermittent phenomena can be speeded up.

A resumé of the more important transducer and amplifier



requirements for some common psychophysiological dependent variables provided in Table 2.

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Table 2  
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More detailed treatment of psychophysiological instrumentation can be found in Brown and Saucer (1958, Bures et al (1960), and Donaldson (1958). An excellent primer of basic electrical theory and circuits is Cornsweet (1963). More advanced general texts include Malmstadt and Enke (1962) and Prenskey (1963).

Table 2. Instrumentation Requirements for Recording Various Autonomic Phenomena

Phenomenon	Transducer or Signal Conditioner	Amplifier Requirements		
		Frequency Range	Input Impedance	Full-Scale Sensitivity
EEG	Electrodes on scalp	DC to 250 cps	100 K or higher	10 $\mu$ V to 100 $\mu$ V
EMG	Electrodes on or inserted into skin	10 cps to 5 K cps	1 Megohm or higher	5 $\mu$ V to 100 $\mu$ V
Averaged Evoked Potentials	as for EEG	as for EEG		
EKG	Electrodes on arms, leg and chest	.05 to 100 cps	1 Megohm or higher	10 $\mu$ V to 10 mV
Heart Rate (HR)	as for EKG	as for EKG		
GSR, Skin Resistance (SR)	Palmar electrodes with external constant-current source. Calibrated zero suppression	DC to 100 cps although DC to 10 cps adequate for most purposes	1 Megohm or higher	50 mV to 10 V
GSR, Skin Conductance (SC)	Palmar electrodes with external constant-voltage source. Calibrated zero suppression		5 K ohm	250 $\mu$ V to 5 mV
SPR, Skin Potential (SP)	Palmar electrode with reference on arm. Electrode temperature compensation. Calibrated zero suppression		1 Megohm or higher	1 mV to 100 mV
Blood Pressure (BP): Continuous absolute BP Intermittant absolute BP Continuous relative BP	Pressure transducer with: - vein cannula; - automatically inflating arm cuff; - arm cuff at below systolic pressure	DC to 100 cps	Carrier-type pre-amplifier for pressure gage	
Skin or Core Temp.	thermister probe with associated bridge circuitry	Amplifier generally not required		
<u>Eye Movements</u> Electro-oculography	EEG electrodes either side of orbit	As for EEG		
Infra-red Photocell	Commercial goggles with light source and Pre-amplifier	As for EKG		
Pupil Size	Cinematography Infra-red reflection	None Commercial Instrument (expensive)		

## INTERPRETING PSYCHOPHYSIOLOGICAL RESPONSE DATA

### The Problem

The most common--although not the best--method for monitoring GSR activity is to make continuous recordings of skin resistance (SR). Wave-like decreases in the SR curve are the skin-resistance GSRs. But we could also choose to measure skin conductance (SC) instead, either directly or by taking the reciprocals of SR measurements, and we could express the GSR in units of conductance change. Since this transformation is nonlinear, one choice of unit may give us different results than another. Suppose, for example, that a drug supposed to produce increased arousal yields tonic SC values of 1, 1, 10 and 10 micromhos in four experimental subjects while a placebo yields SC values of 5, 5, 5 and 5 micromhos in four control subjects. The mean SC for the drug group (5.5 micromhos) is higher than for the controls, suggesting that the drug has indeed produced a stimulating effect. But expressed in SR units, these results are reversed; the experimental group shows SRs of  $10^6$ ,  $10^6$ ,  $10^5$  and  $10^5$  ohms, i.e., a mean of 550,000 ohms, while the control group has a mean SR of 200,000 ohms. Although conductance and resistance are reciprocals of one another, the experimental group has both a higher mean SR and a higher mean SC!

This particular problem will arise only if the groups differ for some reason in their variances but it is clear that non-linear transformations of scale can play havoc with group comparisons and summary statistics of many kinds; the product-moment correlation, for example, may be considerably changed if one variable is transformed to its reciprocal. Note too that reciprocal units are naturally available for other response systems as well; one can express the rhythm of the heart in terms of beats-per-minute or,

equally logically, in terms of the period or average inter-beat interval.

A different kind of problem has to do with the relationship of the post-stimulus change in some response measure to the tonic or pre-stimulus value. Smith and Jones show resistance GSRs of 10 K-ohms and 1 K-ohm, respectively, to the same shock stimulus; which subject has been more disturbed by the shock, which has shown the larger psychological reaction? With no other evidence available, we should have to say "Smith", but suppose that his pre-stimulus SR was 1,000 K-ohms while Jones' was only 100 K-ohms and suppose also that we know from other evidence that resistance GSRs have a high positive correlation with pre-stimulus SRs (as they almost always do). Then, clearly, we must assume that Smith's response would have been smaller had the stimulus been administered when his SR was as low as Jones'. One obvious solution might be to use the known regression of GSR on SR to correct Smith's GSR value; that is, we could partial-out the effect of SR so that the resulting transformed GSRs could be compared in the same way that we could compare the raw scores if all subjects happened to show that same SR at the moment of stimulation. This, in fact, is the principle of Lacey's autonomic liability score (Lacey, 1956) which will be discussed below.

Note, however, that it is not at all clear that one should always want to remove all correlation between pre-stimulus values and change or response scores. It may be, for example, that Smith, with his high SR, was relaxed and rather sleepy when the shock was administered while Jones was alert and "ready for it;" it seems likely that in this case Smith might really have been more disturbed by the sudden shock, that it actually felt stronger to him than it did to Jones and that Smith's GSR was therefore validly

larger than Jones'. If the pre-stimulus value has some meaning concerning the physiological and psychological state of the subject at that moment (and we do usually interpret it so), then some valid relationship between this value and the subsequent response is to be expected; this is after all just another way of stating the usual formula that behavior is some joint junction of the stimulus and the state of the organism at that time. If one rushes to transform away all correlation between pre-stimulus and change scores, assuming it to be some sort of statistical artifact, one may distort the proper psychological interpretation of one's data.

#### Some Relevant Considerations

One can distinguish some four stages in the causal sequence underlying an observed psychophysiological response: (A) An initial central event or psychological process (e.g., the perception and analysis of the stimulus, identifying it as intense or threatening or painful), followed by (B) the central initiation of a response to the stimulus (subsequent to the perceptual process itself, e.g., reticular arousal, central sympathetic discharge, initiation of activity in the sudomotor pathways to the palms); this central physiological reaction leads to (C) a peripheral physiological change at the site of our electrodes or other transducer (e.g., increased heart rate, desynchronization of cortical dendritic potentials, increased palmar sweating), some aspect of which produces (D) the observed change in the physical variable which we are actually recording (changing electrical potentials between arm and arm or between two points on the scalp, a change in current flowing through the palmar skin). As we shall see, there are times when one might wish to talk about either A or B or C--e.g., about how strong the subject perceived the stimulus to be, about how disturbed or

aroused he was by it, or about how much faster it caused his heart to beat--but in any case one has only the observed value, D, upon which to base one's inference about A, B and C.

Now the relationship between D, the observed datum, and C, the local physiological change, in this example will depend on the characteristics of the transducer and recording system used. Thus, if C is "a ten percent increase in the number of active sweat glands per unit area of palmar skin," then D might be a change in conductance of from 1.0 to 1.1 or 0.1 micromhos. But if our measuring circuit gives us readings of resistance instead, then D would in this case be a resistance change of from 1000 to 909 or 91 K-ohms. Now there is fairly good reason to think that the main effect of sudomotor innervation really is to increase the density of active sweat glands which would amount to adding more conduction paths in parallel to those already present. In this event, conductance would be the logical unit to employ since it would be linearly related to the density of active glands. Moreover, if this model of the peripheral process, C, is approximately correct, increments in SC (the conductance GSR) would not in general be correlated with pre-stimulus values (since adding more conductors in parallel produces the same increase in conductance no matter how many are already present) whereas in this same situation resistance GSRs would be strongly correlated with the pre-stimulus SR! (For example, the same 0.1--micromho conductance GSR would give resistance GSRs of 91, 24, 4 and 1 K-ohms, respectively, as the pre-stimulus SR varies from 1000 to 500 to 200 to 100 K-ohms.) Here would be an example of a correlation between pre-stimulus and change scores which is entirely an artifact of the choice of unit; given resistance units, the relation

of transducer to peripheral physiology forces such a correlation quite apart from any valid relation there may be between the state of the subject and the way in which he responds to stimulation. This kind of correlation is entirely spurious for the psychologist's (or physiologist's) purposes and should (and can) be eliminated by, in this case, using the proper units in the first place.

The relationship between C, the local change, and B, the central physiological response, in this example will depend upon the current state of the local effector "organ" or mechanism and, hence, upon its recent history or pre-stimulus activity. Floor or ceiling effects may enter in here; e.g., if most of the sweat glands are already active, a given sudomotor impulse will not yield as great a change in C (or in D) as it otherwise would. Homeostatic or negative feedback influences may also complicate the picture. The electrodermal mechanism seems to be unique in its lack of any apparent peripheral homeostatic control; there seems to be no mechanism to monitor the rate of palmar sweating and adjust sudomotor outflow so as to maintain some stable intermediate rate. But the heart is of course under strong homeostatic restraint so that, as Lacey (1956) has argued, the same cardio-accelerating stimulation will be opposed by greater vagal inhibition when heart rate and blood pressure are already high and so will yield a smaller acceleration than it would had the previous heart rate been slower.

The local response, C, may also vary with respect to its source of excitation, B, due to fatigue of the effector mechanism or changes in its reactivity related to nutritional factors and the like. In the case of the EEG, a given increment in reticular arousal, B, might produce an increase in alpha amplitude, C, if the subject is drowsy, or a decrease in

alpha amplitude if the subject is already wide awake. Thus, even if our observed variable, D, is a faithful, linear measure of the local process, C, we cannot be guaranteed that D will measure B with equal fidelity. Sometimes our interest is in C specifically; we want to know the heart rate, the amount of palmar sweating, the alpha amplitude. But in other cases we are concerned with B instead and we require that our observations tell us something about the "degree of sympathetic arousal" or the "amount of reticular activation"; if this is the kind of question one chooses to ask, then one must be concerned not only about the relation of D to C but also about the relation of C to B. That is, the further back in the causal sequence is the variable we wish to assess, then the more nuisance factors there may be to complicate and distort the relationship between that variable and that end effect which we can actually observe.

Finally, the relation between the perceptual process, A, and the central response, B, which it initiates must be considered. For present purposes, I have included under A the entire process of evaluation of the stimulus situation; after A has been completed, the subject has assigned whatever meaning he can to the stimulus and commands have been initiated for appropriate changes in arousal, emotional reactions and the like. But again we know that there can be both inter-subjective and intra-subjective variability in the response to such "commands". An exhausted subject may identify a new stimulus as "an emergency", calling for increased activation--just as he would at any other time--and yet be unable to muster the degree of central arousal that he normally would show. We know that stimuli can be identified as "painful" and even ordered for degree of painfulness by subjects, who, for some reason, are not experiencing the usual "distress"



concomitant of pain; in such cases one would speak of a disruption of the expected relation of B to A. In an experiment concerned with GSR conditioning in the psychopathic personality (Lykken, 1957), a few psychopaths who seemed to have pathologically little susceptibility to normal anxiety gave negligible GSRs to signals which had been repeatedly paired with a painful electric shock. It was assumed that these subjects were perfectly aware of the CS-shock contingency but that they "didn't much care"; perceiving the CS and realizing that it meant shock was to follow, (A), failed in these individuals to initiate the expected central and peripheral pattern of emotional arousal, (B). A few other, apparently neurotic, subjects gave almost as little GSR to the CS and other evidence was found which gave some support for the hypothesis that these individuals might actually not have been fully "aware" of the CS-shock contingency. Their tonic conductance levels were uniquely depressed, suggesting that they might have reacted to the general stress of the situation by a kind of defensive "turning out the lights" so that the rather similar response observation, D, was in their case tentatively attributed to a peculiarity at the level of perception, state A, rather than to an abnormality in the relation of B to A.

The psychologist will be interested in events at level A, of course, frequently in events at level B and much less frequently in events at level C (he may have an intrinsic interest in the rate of heartbeat, for example, because the proprioceptive feedback from a pounding heart may have important psychological stimulus effects, but qua psychologist his usual concern is with the central events for which he is using heart rate as an indicant). There is interest for the physiologist in events at levels B and C and, perhaps to some extent at level A as well. The relation of D to C represents

mainly a measurement problem, of little general scientific interest but having great methodological significance. These problems are sometimes negligible as in the case of heartrate again where the "local" effector mechanism is clearly specifiabile and where modern techniques permit us to be confident that, e.g., Smith's and Jones' hearts are in fact both beating at 70 per minute if the cardiometer reads "70" in both instances. At the other extreme, the local mechanism for electrodermal phenomena is still not well understood and it is certain that a variety of extraneous factors can exert considerable effect upon our observations, D. Thus, the observed value of SC is affected by the chemistry and concentration of the electrolyte, the current density, the location of the electrode, the thickness and hydration of the skin, racial differences in skin characteristics, hormonal effects relating to the menstrual cycle, and so on and all such factors would normally be regarded as "noise" in one's measurements.

#### Evaluating Measures of Tonic Level

Most of the response systems presently under consideration are alike in that one can distinguish a tonic level of activity, determined by the subject's general physiological status, his current level of arousal, the average excitatory value of the environmental situation and the like, on which may be superimposed phasic fluctuations elicited by specific stimuli. Both kinds of measures involve special problems of interpretation. In considering these problems in their turn, we shall use skin conductance--the tonic SC level and the phasic GSR--as a fairly typical response system for purposes of illustration.

#### The Choice of the Best Physical Unit of Measurement.

The first step is to choose among alternative physical units in which to

express the observations to be made. There is adequate evidence that skin conductance increases with increasing sudomotor activity over at least most of the range. Although we do not yet know the form of this increasing monotonic function, we can at least be confident that it will be somewhat simpler than that obtained by expressing our measurements in resistance units. For similar reasons we would choose heart rate in preference to heart period on the grounds that the former will be more simply related to cardioaccelerator excitation.

#### Correction for Individual Differences in Range.

We cannot expect the effector organ to exhibit the same absolute level of activity in all individuals for the same level of central activity. In their study of cardiac activity in infants, Bridger and Reiser (1950) observed "that babies in the same activity state--from profound sleep to violent crying--may have different heart rate levels, and that babies with the same heart rate levels may be in different states of excitability." (p. 274). Individual differences in the range of SC values are probably still greater. Where one subject's SC ranges from one micromho during sleep to five micromhos in high excitement, we may find another subject showing SC values of 5 and 20 micromhos under the same conditions of measurement.

Even with no sudomotor innervation, the skin displays a certain minimum conductance,  $SC_{i(\min)}$ , which will vary among individuals in relation to local anatomical and physiological peculiarities. Similarly, the maximum conductance  $SC_{i(\max)}$ , produced under the condition of maximal central activation, will vary from person to person due to characteristics of local effector reactivity. More generally, it seems reasonable to say that neither the minimum nor the maximum absolute level of an autonomic

response system (measured under conditions of zero or maximum activation, respectively, of the central process which controls this system) will normally be correlated with the properties of this central process nor with any other variable of direct psychological interest.

Therefore, we can say that the tonic level,  $TL_{ij}$ , observed in individual  $i$  under stimulus condition  $j$  is the sum of that individual's minimum tonic level,  $TL_{i(\min)}$ , plus a component of increased activity,  $\hat{TL}_{ij}$ , attributable to the stimulus situation:

$$(1) \quad TL_{ij} = \hat{TL}_{ij} + TL_{i(\min)}$$

Now this component of increased activity may be expressed as the product of the "activity potential,"  $\rho_i$ , of the local effector, times some function,  $f_i(\psi_j)$ , of the central process,  $\psi_j$ , evoked by the stimulus condition  $j$  and in the magnitude of which we are principally interested;

$$(2) \quad \hat{TL}_{ij} = \rho_i f_i(\psi_j).$$

If we arbitrarily let  $\phi_{i(\min)} = f_i(\psi_{\min}) = 0$ , and  $\phi_{i(\max)} = f_i(\psi_{\max}) = 1$ , then the coefficient  $\rho_i$  represents the maximum increase in activity which can be produced in this  $i$ th individual's local effector when the central process is at maximum arousal;

$$(3) \quad \hat{TL}_{i(\max)} = \rho_i f_i(\psi_{\max}) = \rho_i = TL_{i(\max)} - TL_{i(\min)}.$$

Therefore since

$$(4) \quad TL_{ij} = \rho_i f_i(\psi_j) + TL_{i(\min)},$$

we can write

$$(5) \quad \phi_{ij} = f_i(\psi_j) = \frac{TL_{ij} - TL_{i(\min)}}{TL_{i(\max)} - TL_{i(\min)}}$$

This suggests that, after choosing what seems to be the most rational physical unit in which to express the tonic level of a response system, the next step should be to convert one's absolute measurement  $TL_{ij}$ , into the corrected index,  $\phi_{ij}$ , from which the influence of individual minima and maxima has been removed, i.e., into a unit representing one's best available estimate of  $f_i(\psi_j)$ . This index,  $\phi_{ij}$ , which varies between zero and +1.00, expresses the tonic level produced in person  $i$  by stimulus situation  $j$  as a proportion of the maximum increment over his minimum tonic level of which this individual is capable.

To illustrate, suppose that Jones and Smith show tonic SC values of 10 and 7 micromhos, respectively, in an experimental situation, X, and assume that we know Jones' minimum and maximum SCs to be 5 and 20 micromhos compared to 1 and 10 micromhos for Smith. We compute corrected indices according to the formula

$$(6) \quad \phi_{ix} = \frac{SC_{ix} - SC_{i(\min)}}{SC_{i(\max)} - SC_{i(\min)}}$$

For Smith,  $\phi_{sx} = (7 - 1) / (10 - 1) = 0.67$ .

For Jones,  $\phi_{jx} = (10 - 5) / (20 - 5) = 0.33$ . Thus, although Jones' absolute conductance level was higher than Smith's, we must conclude that in fact our stimulus condition produced a relatively larger tonic level in Smith.

Knowing that the central process,  $\psi$ , which controls intra-individual variation in SC is some component of psychological arousal or of CNS activation, we can further conclude that Smith's arousal or activation in our situation X was greater than Jones'.

It should be possible to devise relatively simple and feasible means for estimating the limits of the tonic range. One or two inhalations of

CO<sub>2</sub> may elevate heart rate to a near approximation of the individual's normal upper limit, a brief standardized exercise might do the same for blood pressure. In the case of skin conductance, local sweat gland activity can be eliminated by iontophoretic application of atropine and maximized by application of pilocarpine. It is possible, but requires experimental proof, that the low and high SC values thus produced might be fair estimates of the individual's normal range of variation.

Even much cruder estimates can be an improvement over the use of uncorrected absolute values. Rose (1964) correlated the two-flash threshold, which varies inversely with arousal, with tonic SC in four separate samples. He also "corrected" his SC values by the method described above using as his estimate of minimum SC simply the lowest SC value shown by the subject in the session while as estimates of the maximum SC he used the highest value reached while the subject was blowing up a balloon to bursting. As shown in Table 3, these "corrected" SC values correlated more highly with the two-flash threshold than did the raw SC scores in all four samples. If the two variables are indeed both measures of arousal, then their un-shared variance must be largely due to their combined errors of measurement; correcting the SC scores even by means of such crude estimates of the individual SC ranges decreased the total un-shared or "error" variance by some 26 percent on the average (and increased their common variance by an average of 76 percent).

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Table 3  
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Determining the Relationship Between the Corrected Index of Tonic Level,  $\phi$ , and the Underlying Variable of Interest,  $\psi$ .

The above procedure for deriving the corrected index,  $\phi$ , from the raw

Sample	N	Correlation between TFT and:		Reduction in "Error" Variance
		SC	$\phi_{sc}$	
1. (normal males)	22	-.54	-.76	.29 (41%)
2. (normals, both sexes)	36	-.41	-.53	.11 (13%)
3. (psychiatric patients, males)	20	-.61	-.70	.12 (19%)
4. (psychiatric patients, females)	20	-.50	-.72	.27 (36%)

Table 3. Improvement obtained in the correlation between two putative measures of arousal, tonic skin conductance (SC) and the two-flash threshold (TFT), when one variable (SC) is corrected for individual differences in range (yielding the index  $\phi_{sc}$ ) by the method described in the text. (Data from Rose, 1964)

measurements of tonic level is designed to eliminate the effects of individual differences in peripheral effector characteristics (which are usually irrelevant) and thus to eliminate what may be a sizeable component of error of measurement in the estimation of the underlying variable of interest,  $\psi$ . Uncertainty will still remain, however, as to the exact form of the relationship between this derived index and the underlying variable, i.e., the form of the function  $\phi = f(\psi)$  remains unknown. In the case of skin conductance, a considerable accumulation of imperfect (and imperfectly analyzed!) data provides reasonable empirical support for the conclusion that the function  $\phi_{sc} = f(\psi)$ , where  $\psi$  represents some aspect of arousal or CNS activation, is at least monotonic-increasing and probably negatively accelerated. Two separate methods which could be used to specify these relationships more exactly, both of which would require a somewhat complex analysis of a large quantity of carefully collected data, are outlined below.

First Method. One ingenious method by which the true relationship between  $\phi$  and  $\psi$  could be approximated was suggested to me by P. E. Meehl. We shall assume that  $\phi$  bears the same functional relationship to  $\psi$  for all individuals. Let us expose a large and representative sample of persons,  $P_i$ , to each of a number of stimulus situations,  $S_j$ , which differ widely among themselves in "arousal value." In Figure 14 we have plotted the inter-subjective distributions of corrected  $\phi$  values for six such stimulus situations with the latter arranged along the abscissa in order of increasing average  $\phi$ . These hypothetical distributions have been drawn with a positive skew to allow for the fact that some subjects will show high activation (and high  $\phi$ ) even in the less arousing situations, either



because they are already aroused for idiosyncratic reasons or because they are unusually excitable individuals whose level of arousal will reach its asymptote in situations which might be only moderately exciting for the average subject.

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Figure 14  
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Moreover, the distributions have been given considerable variance and overlap between stimuli since it must be expected that the ordering of the arousal value for these stimuli for particular individuals will not always parallel the order of the group means. Thus, "making a parachute jump" will be more activating than "giving a book report in class" for most individuals (and well above asymptote for some) but not for everyone.

Even in terms of the mean  $\phi$  values for the group, we must not assume that the increments in arousal value between successive stimuli in this ordinal series will be equal. The stimuli have been chosen and arranged to insure only that the plot of mean  $\phi$  will be monotonic-increasing, as shown in Figure 15.

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Figure 15  
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The next step in the process is to alter the intervals between stimuli on the abscissa so as to make the plot of mean  $\phi$  values take the form of the first function whose fit to the data we desire to test. We might reasonably begin by inquiring whether  $\phi = f(\psi)$  is a straight line. In Figure 16, the inter-stimulus intervals on the abscissa have been expanded or compressed as necessary to convert the  $\phi$  means into a linear array. Holding this arrangement fixed, we now fit a straight line to each individual's set of  $\phi$  values,

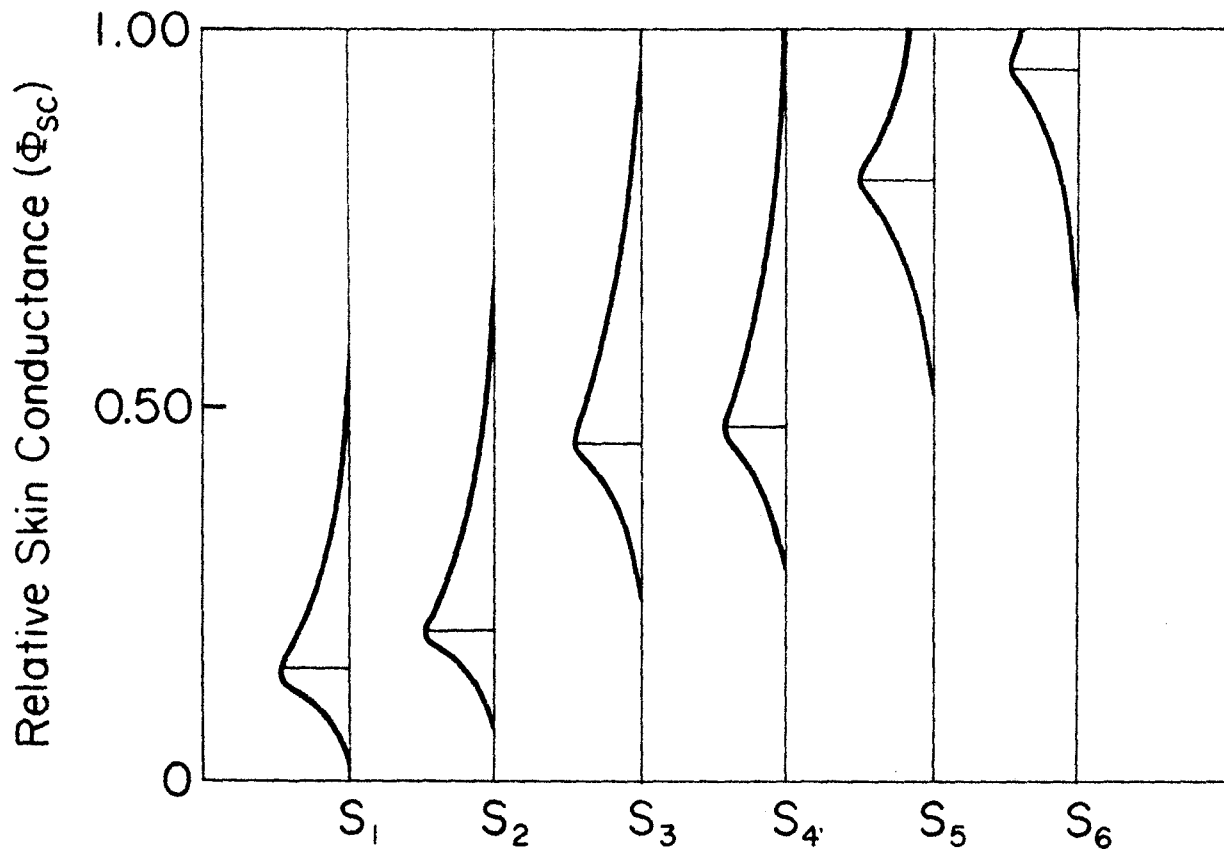


Figure 14. Hypothetical distributions (across subjects) of skin conductance scores corrected for individual differences in range. Six stimulus situations,  $S_1, \dots, S_6$ , are chosen to span the range from low to high average arousal value and are arranged here in order of increasing average  $\phi_{sc}$  (and thus, ex hypothesi, in order of increasing  $\psi$ ).

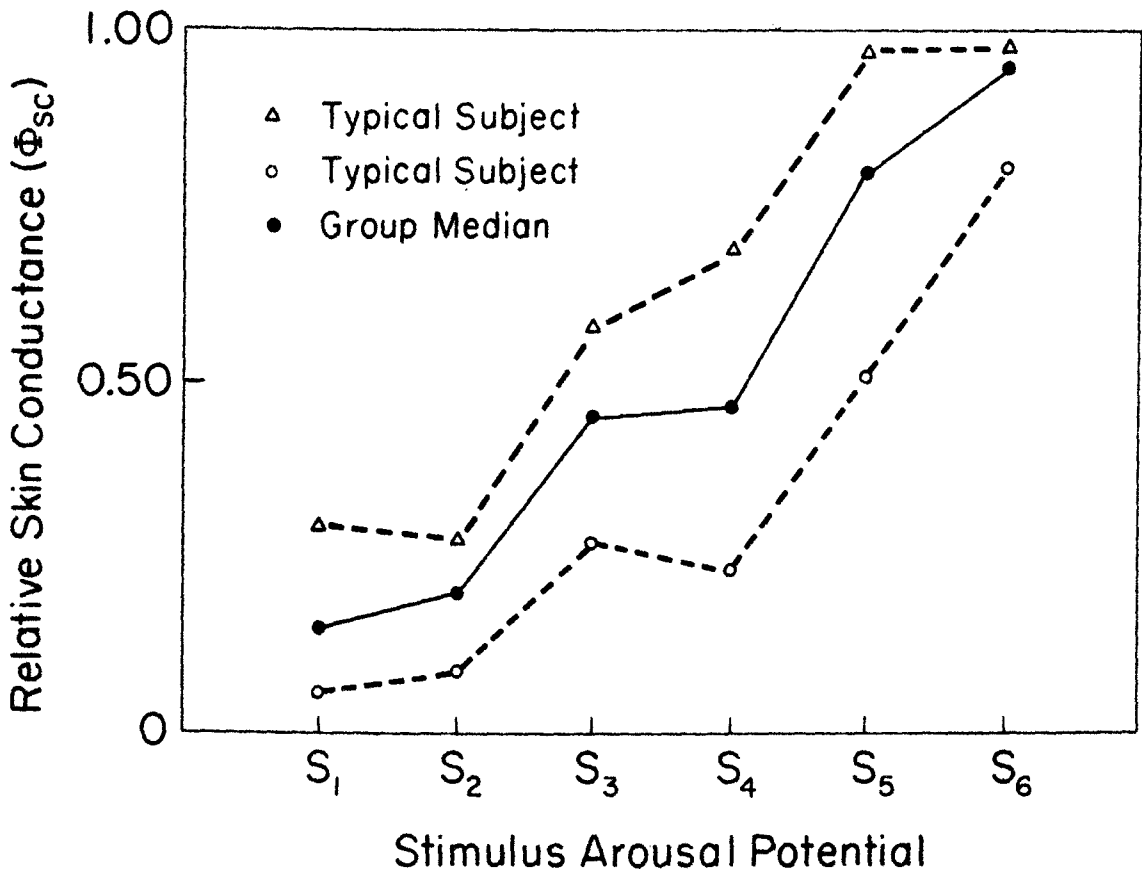


Figure 15. The medians of the six hypothetical distributions shown in Fig. 14, together with the  $\phi_{sc}$  scores of two "typical" individual subjects in each of the six stimulus situations.

finding the parameter,  $\rho_i$ , which gives the best fit for him (n.b. all individual lines must meet the abscissa at zero). Then we compute for each individual the residual sum of squares about this best-fitting line and, from this, a statistic representing the over-all average goodness-of-fit of the individual linear functions.

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Figure 16

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Since  $\phi$  cannot rise above 1.00 and we are assuming that activation is maximal at that point, some subjects may reach this asymptote even for some of the less arousing stimuli in our series, e.g.,  $S_3$ ,  $S_4$  or  $S_5$ . In this event, the locus of the group means will not faithfully trace the curve relating  $\phi$  to activation; e.g., even if this actual function is linear, the group mean curve will become negatively accelerated as more and more subjects reach the  $\phi$  asymptote. One way to avoid this problem would be employ medians in place of the group means.

We must now repeat the process for some other function which we expect may describe the relation of  $\phi$  to  $\psi$  more accurately. As indicated in Figure 17, we might now change the inter-stimulus distances on the abscissa so as to force the median values of  $\phi$  to fall upon a positive-growth curve. As before, we then fix this abscissa and proceed to find the best-fitting function  $\phi = 1 - e^{-\rho_i \psi_j}$  for each individual. From the

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Figure 17

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residual variability of each subject's data points about these best-fitting growth functions we then compute our measure of average goodness-of-fit and compare it with the value previously obtained with the straight line.

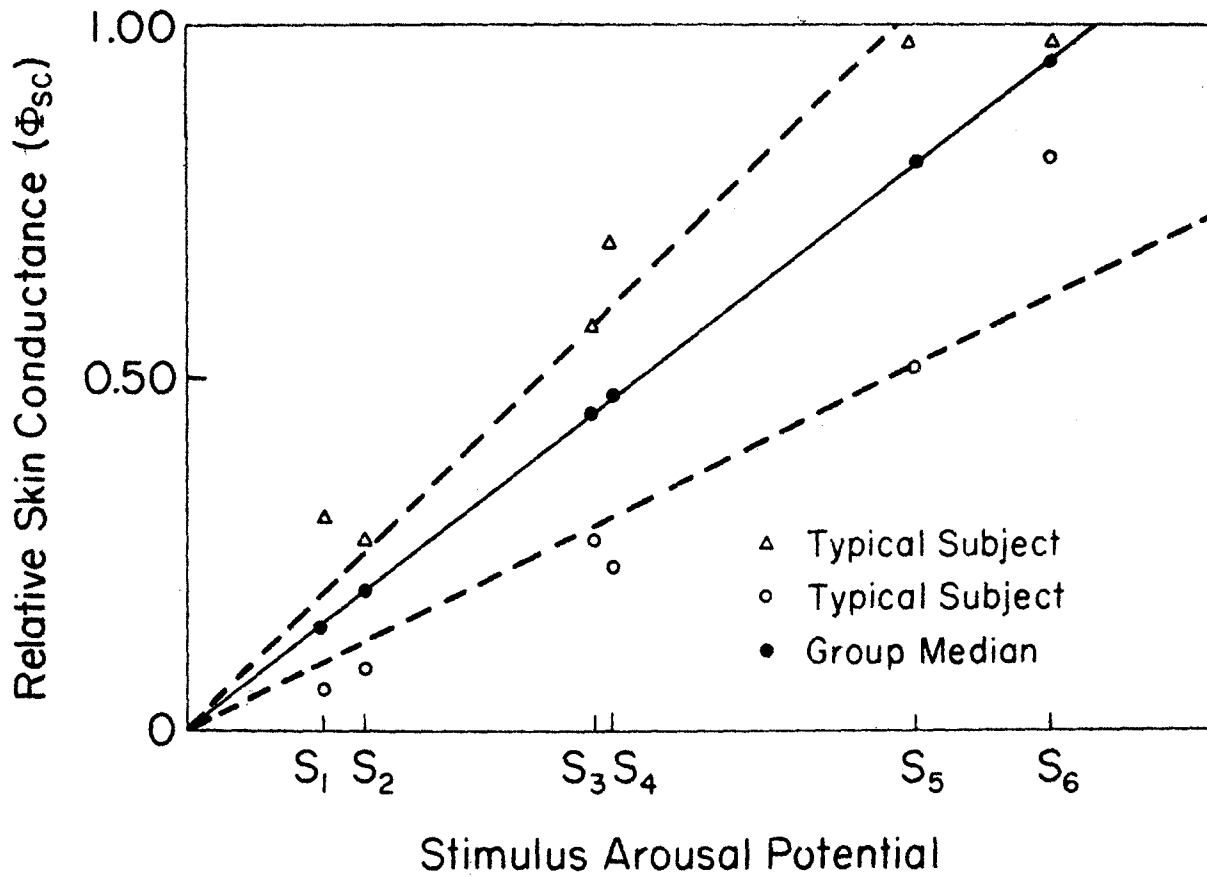


Figure 16. Same data as in Fig. 15 except that spacing of  $S_1, S_2, \dots, S_6$  on the abscissa has been changed to force group medians into a linear array including the origin (0). Straight lines have been fitted to the two sets of individual scores, showing rather poor fits in both cases.

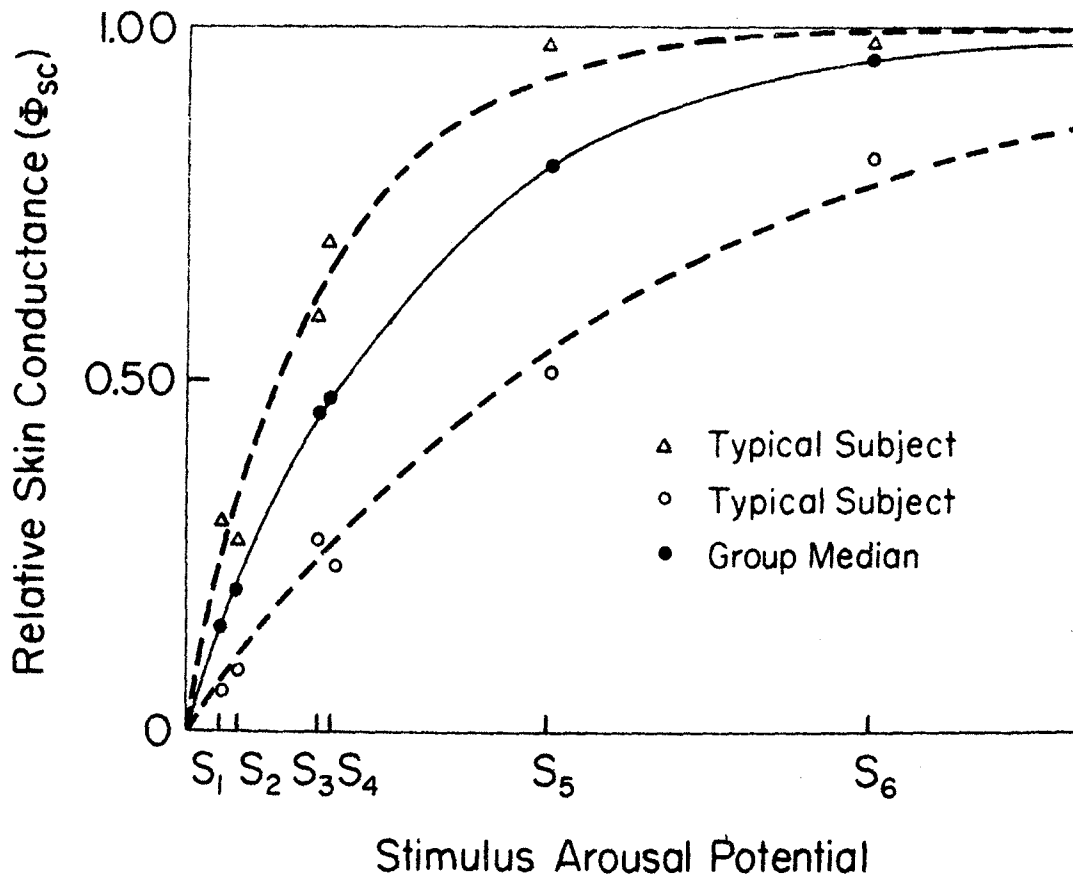


Figure 17. Same data again but with spacing of  $S_1, S_2, \dots, S_6$  on the abscissa altered to force group medians to fall on a growth curve from the origin (0). Similar functions have been fitted to the two sets of individual scores, showing rather good fits in both cases.

(Note that the testing of an exponential model raises special difficulties since, as Estes (1956) has shown, the relation between individual curves and that for the group mean is ambiguous in the case of exponential functions. I have not attempted to work out what special procedures or facilitating assumptions might be required to meet this difficulty in the present application.) By means of this procedure (which would be feasible only through the use of high-speed computer facilities) one might hope to identify which of the families of functions tested best describes the relation of the corrected measurements of the dependent variable,  $\phi$ , and the hypothetical underlying variable,  $\psi$ .

Thus far we have been plotting on the abscissa, not  $\psi$ , but rather the average arousal value of the stimulus. That is, when the inter-stimulus distances have been adjusted so that the medians of the vertical arrays lie on that curve which subsequent analysis shows to be the "best," then we may assume that activation increases linearly along the abscissa for each individual up to that point at which the individual's curve reaches its asymptote. However, the increase in activation represented by a given increment along the abscissa will vary from subject to subject. In Figure 17, for example, a stimulus having the arousal value of  $S_5$  produces a  $\phi$  value of about 0.50 for one individual, about 0.95 for the other, and a median of about 0.80 for the group as a whole. If we now relativize the abscissa so that its units represent equal increments in individual activation, we get the single curve shown in Figure 18. From this curve, we can infer that  $S_5$  produced about 23 percent of maximum arousal in our first subject (whose  $\phi$  was 0.50), about 99 percent of maximum arousal in the second

subject (for whom  $\phi$  was 0.95) and about 54 percent of maximum  $\psi$  on the average in the group as a whole.

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 Figure 18  
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To illustrate the method of "relativizing the abscissa" suppose first that  $\phi_{ij} = \log_{\alpha_i} \psi_j$ . We now want to re-draw the individual curves with  $\psi$  represented on the abscissa, remembering the convention that  $0 \leq \psi_{ij} \leq 1.00$ . All individual curves on these coordinates must begin at the point  $\phi = 0, \psi = 0$  and must terminate at the point  $\phi = 1, \psi = 1$ . Since all individual curves must have the same form, e.g.,  $\phi_j = \log_{\alpha} \psi_j$ , in this case, therefore all individual curves coincide on this figure as the curve  $\phi_j = \log_{10} \psi_j$  [since  $\log_{\alpha}(1) = 1, \alpha = 10$ ]. If  $\phi_{ij} = 1 - e^{-\alpha_i \psi_j}$ , there is the special problem that this growth function never actually reaches  $\phi = 1$ . But we can arbitrarily stipulate, e.g., that  $\phi_{ij} = .95$  when  $\psi_j = 1.00$ . Then  $1 - e^{-\alpha} = .95$  and  $\alpha = -\log_e .05 \approx 3$ . Thus, the general curve we plot in Figure 18 is  $\phi_{ij} = 1 - e^{-3\psi_{ij}}$ .

This curve (Figure 18) represents the real fruit of our labors. It is a hypothetical curve because the necessary data collection and analysis have not yet been done. The task is rather formidable and would not be worth the doing except on a sufficiently large scale and with enough care to permit confident generalization of the findings. For reasons of economy, data should be collected at the same time on concurrent heart rate, blood pressure levels and the like to permit scaling of such other important physiological response systems according to the same general method.



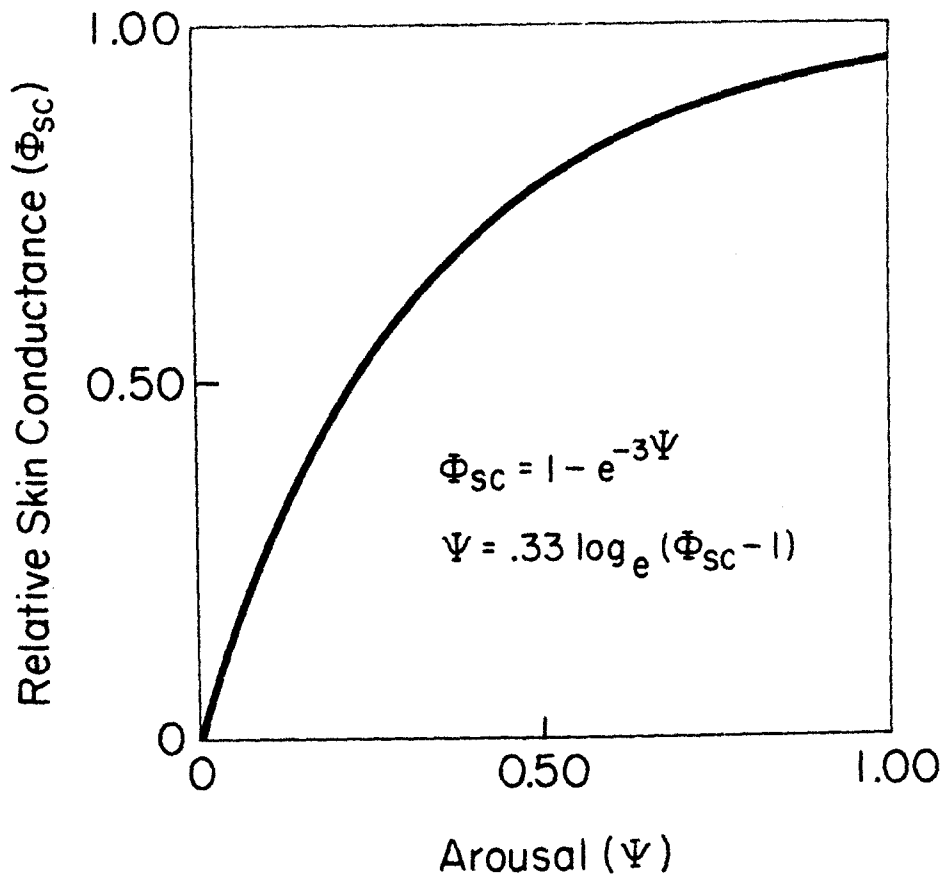


Figure 18. Growth function relating  $\phi_{sc}$  to  $\psi$ . This function might be derived from the hypothetical data of Fig. 17 by "relativizing the abscissa" (see text). The relationship of  $\phi_{sc}$  to  $\psi$  illustrated by this curve is tabulated in Table 4.

The hypothetical data on which Figure 18 is based are listed in Table 4 and were calculated on the assumption that the present approach might indicate that  $\phi$  is a growth function of  $\psi$ ,  $\phi = 1 - e^{-3\psi}$ . The practical significance of such findings would be as follows. On all occasions in which tonic SC levels are to be employed as indicants of central activation, one should provide some means of estimating each individual subject's minimum and maximum SC levels under the conditions

[Table 4]

of measurement. Then each raw conductance level,  $SC_{ij}$ , obtained from the  $i$ th subject in stimulus condition  $j$ , should be corrected by the formula  $\phi_{ij} = (SC_{ij} - SC_{i(\min)}) / (SC_{i(\max)} - SC_{i(\min)})$ . Finally, these corrected conductance levels could be used to enter Table 4 from which the associated estimates of true activation level could be read.

Second Method. Another, and independent, method of determining the relationship of  $\phi$  to  $\psi$  may be employed when one has measures of two (or more) variables,  $\phi_x$  and  $\psi_y$ , both of which are believed to be related to the same underlying variable,  $\psi$ . For example, if  $\phi_{SC}$  is some function of activation,  $f(\psi)$ , and if the two-flash threshold, similarly corrected, ( $\phi_{tft}$ ), is some other function of activation,  $g(\psi)$ , then the linear correlation between the two inverse functions,  $f^{-1}(\psi)$  and  $g^{-1}(\psi)$ , will approach unity, limited only by errors of measurement of the two variables. Although both functions (with their inverses) are unknown, one could specify a small number of families of functions which would be likely to include both  $f(\psi)$  and  $g(\psi)$  to a reasonable approximation: e.g.  $\alpha\psi$ ,  $\log\alpha\psi$ , etc. Assume that we stipulate six such functions for trial. We then obtain a representative sample of, say, 1000 people who can be assumed to show a reasonable range

$\phi_{sc} = 1 - e^{-3\psi}$	$\psi$ (Arousal)
0	0
0.1	0.035
0.2	0.078
0.3	0.119
0.4	0.170
0.5	0.231
0.6	0.305
0.7	0.401
0.8	0.536
0.9	0.768
0.95	0.999
1.00	

Table 4. Hypothetical values of activation ( $\psi$ ) corresponding to various corrected SC levels, ( $\phi_{sc}$ ), on the assumption that the curve-fitting procedure described in the text might show that  $\phi_{sc}$  is a growth function of  $\psi$ ,  $\phi_{sc} = 1 - e^{-\alpha\psi}$ , with  $\alpha = 3$ .

of arousal at the time of measurement and obtain from them measures of  $\phi_{sc}$  and  $\phi_{tft}$  (by using corrected values of both variables we are then entitled to study their relationship across individuals in a R-type rather than a P-type design). We now require our computer to obtain the inverse of each of the six functions of both variables and to compute all 36 correlations. For example, the inverse of  $\phi_{sc} = 1 - e^{-\alpha\psi}$  is  $\psi_{sc} = \frac{1}{\alpha} \log_e (\phi_{sc} - 1)$ ; the inverse of  $\phi_{tft} = \beta/\psi$  is  $\psi_{tft} = \beta/\phi_{tft}$ . If these two functions, respectively, provide a good approximation of the true relationship between  $\phi_{sc}$  and  $\psi$  and between  $\phi_{tft}$  and  $\psi$ , then the correlation between  $[\frac{1}{\alpha} \log_e (\phi_{sc} - 1)]$  and  $[\beta/\phi_{tft}]$  should be larger than that between the inverses of any other pair of functions that we test.

Until some such method of analysis is actually applied to real data, we shall have no adequate empirical grounds for specifying the function relating the corrected measures of tonic level to the underlying variable of interest. However, the simple step of correcting the raw measures for individual differences in range will generally provide a large improvement in the accuracy and utility of measures of tonic level (and, one might add, this step alone is a considerable improvement over most current practice). Moreover, there is some evidence and considerable rational justification for assuming that the function relating tonic level to central state must be at least negatively accelerated and therefore likely to be better approximated by a logarithmic or a growth function than by a linear one. Indeed, although the data of Table 4 are wholly hypothetical, it would be surprising if they do not give a better approximation to the real state of affairs than the linear function implicitly assumed when one

uses  $\phi$  alone. In analyzing an experiment in which tonic SC level figures as a dependent variable, I should be inclined to use  $\phi_{SC}$  first and then to repeat the analysis using estimates of  $\psi$  from Table 4 [ $\psi = .33 \log_e (\phi - 1)$ ] to see whether this transformation did not help to clarify my findings.

#### Evaluating Measures of Phasic Change

One must first of all be careful to distinguish phasic changes, those wave-like fluctuations in the output of a response system which are generally elicited by specific stimuli, from more lasting changes in tonic level, such as may occur when the overall stimulus situation changes. Thus, the start of an experimental task may elicit a GSR or phasic increase in SC that returns within seconds to a relatively stable level which itself may be higher than the previous or resting level, reflecting the higher level of arousal necessitated by the demands of the task. Similarly, the termination of that task period might elicit another GSR, again a phasic increase in SC which, however, returns this time to a level lower than before; i.e., here the phasic increase is accompanied or followed by a decrease in the tonic level. Increments in tonic SC have a relatively long latency, persist far longer and seem to represent a generalized response of the organism to the total situation, while the GSR has a shorter latency, a much faster rate of recovery and is typically identifiable as a differentiated response to a specific stimulus; these contrasts are reminiscent of the differences Sharpless and Jasper (1956) observed between cortical arousal effects of midbrain reticular stimulation and the more phasic and specific effects of stimulation of the rostral thalamic reticular regions. However, although the significance of these two kinds of peripheral change seems to be different, in the sense that they appear

to be related to somewhat different central events, it is likely that the efferent linkage and effector mechanism is the same for both--that both response systems share the same final common path.

On this assumption, the GSR is an increase in  $\phi_{SC}$ ,  $\Delta\phi_{SC} = \phi_1 - \phi_0$ , resulting from an increment in sudomotor innervation,  $\Delta\psi = \psi_1 - \psi_0$ . If the relation of  $\phi_{SC}$  to  $\psi$  is linear, then  $\Delta\phi_{SC}$  will be a linear function of  $\Delta\psi$  and uncorrelated with the pre-stimulus level  $\phi_0$  (except insofar as  $\Delta\psi$  is correlated with  $\psi$ ). But, if  $\phi_{SC} = f(\psi) = 1 - e^{-\alpha\psi}$ , then:

$$(7) \quad \Delta\phi = (1 - e^{-\alpha\psi_1}) - (1 - e^{-\alpha\psi_0}) = e^{-\alpha\psi_0} - e^{-\alpha\psi_1}.$$

Therefore,

$$(8) \quad \Delta\phi = e^{-\alpha\psi_0} (1 - e^{-\alpha(\Delta\psi)})$$

or

$$(9) \quad \Delta\phi = (1 - \phi_0)(1 - e^{-\alpha(\Delta\psi)}).$$

Thus, if  $\phi$  is a growth function of  $\psi$ , then  $\Delta\phi$  will be a growth function of  $\Delta\psi$  (with  $\psi_0$  or  $\phi_0$  held constant) and an inverse linear function of  $\phi_0$  (with the increment,  $\Delta\psi$ , held constant). In this event, one's best estimate of underlying change,  $\Delta\psi$ , would be the index (obtained by solving equation 9 for  $\Delta\psi$ ),

$$(10) \quad \hat{\Delta\psi} = \frac{1}{\alpha} [\log_e (1 - \phi_0) - \log_e (1 - \phi_0 - \Delta\phi)],$$

which would be uncorrelated with pre-stimulus level,  $\phi_0$ . This index could be computed more easily directly from raw conductance measurements thus:

$$(11) \quad \hat{\Delta\psi} = \frac{1}{\alpha} [\log_e (SC_{mx} - SC_0) - \log_e (SC_{mx} - SC_1)],$$

where  $SC_{mx}$  is the upper limit of that individual's conductance range,  $SC_0$  is the pre-stimulus tonic level,  $SC_1$  is the peak post-stimulus conductance ( $SC_1 = SC_0 + \Delta SC$ ), and  $\alpha$  is the parameter of the function  $\phi_{SC} = 1 - e^{-\alpha\psi}$ .

That is to say that, if the relationship of the corrected tonic level,  $\phi$ , to the underlying process,  $\psi$ , is nonlinear (e.g. a growth function), then the change in the tonic level brought about by a given change in the underlying process will necessarily be dependent upon the level preceeding the change. A given increment,  $\Delta\phi$ , from a high pre-stimulus level will not indicate the same change in the underlying process that is indicated by that same increment from a low pre-stimulus level. If one knows the function  $\phi = f(\psi)$ , then one can compute from  $\Delta\phi$  an index which will provide an estimate of the underlying process change,  $\Delta\psi$ , which is independent of pre-stimulus level (e.g., by using equation (11) in the case of a growth function, provided one has not only a measure of the increment,  $\Delta\phi$ , but also a measure of the pre-stimulus value,  $\phi_0$ ).

However, since the relation of  $\phi$  to  $\psi$  has yet to be determined empirically, it would be academic to develop such possible correction methods any further here. Although there are many published studies reporting correlations between, e.g., GSRs and pre-stimulus levels, it should be noted that such findings do not provide the data we require. The correlation across subjects between raw measures of tonic level and GSR (or change in tonic level), uncorrected for individual differences in range, is so contaminated by various extraneous influences as to be largely meaningless as a basis for inferring  $\Delta\phi = f(\phi_0)$ . Indeed, the reported values vary greatly from one experimental situation to another, as the present analysis would lead one to expect. Even if the range correction is employed, the observed relationship between  $\Delta\phi$  and  $\phi_0$  will be ambiguous since it will confound the effects of a non-linear relation of  $\phi$  to  $\psi$  which effects one will want to remove-- with the effects of any possible relation of  $\Delta\psi$  to  $\psi_0$  --which are psychologically relevant and the elimination of which would not ordinarily be desirable

That is, the same stimulus may be perceived differently or produce a different central reaction at a low level of pre-stimulus arousal,  $\psi_0$ , than at a high level and one would normally want one's peripheral change measure,  $\Delta\phi$ , to reflect that real and psychologically meaningful difference.

For immediate practical purposes, the present analysis indicates that raw conductance GSRs should be expressed as changes in the individual's corrected index of tonic SC level, which can be done by means of the formula

$$(12) \quad \Delta\phi_{ij} = \frac{\Delta SC_{ij}}{SC_{i(\max)} - SC_{i(\min)}}$$

Since it is free of the "noisy" influence of individual differences in range of SC, this index must provide a more accurate estimate of  $\Delta\psi$  than any algebraic function of  $\Delta SC_{ij}$  alone. Pending determination of the true relation of  $\phi$  to  $\psi$ , one must keep in mind that an unknown portion of any observed correlation between  $\Delta\phi$  and  $\phi$  may be due to nonlinearity in that relationship and hence artifactual. If one is willing to gamble that  $\phi = 1 - e^{-3\psi}$  is not far from correct, then the index given in Equation 11 may be used.

#### The Interpretation of Changes in Tonic Level.

Consider the following experimental problem which is typical of many possible applications of psychophysiological techniques in the broad domain of personality research. There is some evidence that children with specific reading problems perform less well than do normal children in discriminating letter-like forms. Some authorities will infer from this that the reading disability is a consequence of a primary defect in perception--that these children actually see the figures differently or fail to perceive those attributes which are essential for discriminating similar forms from one



another. An alternative view is that the reading disability is secondary to a conditioned anxiety reaction which has been developed in the child by unfortunate or inept handling and which manifests itself in all reading and test-like situations. This anxiety interferes with both learning and performance in school and, by easy generalization, to the school-like circumstances of the laboratory. The latter hypothesis suggests that children with reading problems should be more anxious--and hence more aroused or excited--in the laboratory than control children, matched for age, sex, IQ, and the like, both in a task involving discrimination of letter-like forms and a fortiori in a task involving discrimination of actual letters. The perceptual-defect hypothesis, on the other hand, suggests that, while the problem-readers may indeed show higher autonomic arousal while working with letters (by this time many of them will have become emotional about letters, whatever the etiology of their problem) any increase in arousal, due to generalization, when they are working with the novel letter-like forms will not be great enough to explain their discrimination defect. (This is not a very good experiment since the most probable outcome, slightly greater arousal in the problem-reader group, will not permit a clear conclusion. It is not, however, in this respect atypical of contemporary psychological research!)

Suppose, then, that the average reading-disabled child and his normal control mate displayed corrected tonic SC levels as shown in Table 5. As the anxiety-psychogenesis hypothesis predicted, the problem readers show substantially higher arousal than the normals during the letter-discrimination task and somewhat higher arousal during the task

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Table 5  
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	Corrected Tonic Levels, $\phi$			Change in Tonic Level, $\Delta\phi$		
	Pre-stress	Letters	Forms	Letters	Forms	Forms
	$\phi_0$	$\phi_1$	$\phi_2$	$\phi_1 - \phi_0$	$\phi_2 - \phi_0$	$\phi_2 - \phi_1$
Reading-disabled	.70(.40)	.80(.54)	.60(.31)	.10(.14)	-.10(-.09)	-.20(-.23)
Normals	.40(.17)	.50(.23)	.50(.23)	.10(.06)	.10(.06)	.00
Differences	.30(.23)	.30(.21)	.10(.08)	0(.08)	-.20(-.15)	-.20(-.23)

Table 5. Results of hypothetical experiment described in text. Reading-disabled children show much higher tonic SC levels than normals in discriminating task with letters, somewhat higher in task involving letter-like forms. But in terms of the increment in tonic level over pre-stress levels, the two groups were identical on the letter task and the problem readers actually showed less relative arousal than the normals on the forms task. (The values in parentheses are estimates of arousal level,  $\psi$ , based on the assumption that  $\phi = 1 - e^{-3\psi}$ .)

involving forms. However, we note that they were also much more aroused during the "rest" or pre-stress period at the start of the experiment. "Therefore," argues the exponent of the perceptual-defect view, "one surely cannot attribute all of the arousal shown by these children during the letter task to the effects of that task; the increment in arousal due to the letter task itself, estimated by the difference between the pre-stress level and the level during the task, is actually the same for both groups. Therefore, one must conclude that problem-readers are no more frightened by letter tasks than normal readers."

"The fact remains," rejoins the defender of the anxiety hypothesis, "that the problem-readers were much more aroused--and the circumstances here permit us to attribute this arousal to anxiety--than were the normals. The pre-stress differences indicate either that they are generally more anxiety reactive than normals or else that the laboratory situation was 'school-like' enough to trigger their specific phobias by generalization."

We shall have to concur with this latter interpretation and endorse the use of tonic levels rather than increments in tonic level in this case. Obviously the fact that the problem-readers had higher tonic levels in all three experimental conditions does not prove that their reading problem is psychogenic or anxiety based--as we acknowledged at the onset, the design of this experiment is inadequate, albeit not unrepresentative. But we can at least deny with some confidence the claim that these results disprove the anxiety hypothesis on the grounds that the specific letter task does not show a larger increment in arousal in the problem-readers.

Autonomic level scores are very commonly analyzed in this way using as the dependent variable the difference between post-stress and pre-stress

values, i.e., changes in tonic level are treated like phasic changes elicited by fleeting stimuli. But by definition a change in tonic level is a relatively enduring change brought about by a persisting alternation in the stimulus situation. The subject has time to take stock of the new situation and there is opportunity for his psychophysiological mechanisms to adjust themselves accordingly. Whereas phasic reactions to novel stimuli are usually transient increases in activity, changes in tonic level may be either positive or negative. Thus, an apprehensive subject may discover, after the start of the experimental task, that its demands are not as great as he had expected so that the post-stress level may actually be lower than the pre-stress level for him. The problem readers in our hypothetical experiment had a tonic level of .70 at the start of the experiment, which decreased to .60 during the task of discriminating letter-like forms; under the same conditions, the control children showed an increase from .40 to .50. Clearly one must conclude that this task was more stressful for the problem readers even though it produced a decrease in their tonic level, indicating, perhaps, that it was not as stressful as they had anticipated.

The argument in favor of using increments in tonic level, rather than the actual "post-stress" levels themselves, is based on the notion that part of the arousal during the stress situation must be attributable to factors other than the stimuli being manipulated in the experiment; the subject with a high pre-stress level is already aroused for extraneous reasons, hence it is only the increment to the post-stress level that can rightfully be attributed to the effects of the stress. But surely this conception is too great an over-simplification. The introduction of the task or stressor does not merely add a stimulus to the stimuli already present; rather it

changes the total situation. Normally, the pre-stress or resting situation is relatively unstructured. The subject has little to occupy him and is free to worry apprehensively or to think pleasant thoughts, as his fancy and his temperament may predispose him. The experimental task or stressor, in contrast, is typically engrossing, so that the subject's average tonic level during the experimental manipulation can usually be taken as a fair index of his reaction to that controlled, external situation.

#### Some Illustrative Examples.

The reader will already have become convinced that interpreting physiological response data can sometimes be a rather tricky business. One must first acquire an adequate grasp of the specific technological problems involved in measuring the response systems one wishes to employ as dependent variables. The next step is to very carefully formulate the questions one wishes to answer. Some of the more common types of experimental questions are illustrated in the following examples.

##### (1) Individual Differences in Autonomic Reactivity.

Example: Jones shows a more elevated heart rate than does Smith while giving a speech before a class. We know that Jones is more susceptible to "stage fright" than is Smith and we hypothesize that this may be partially explained by the fact that Jones has the more labile autonomic nervous system; his stronger responses to the "same" stress produce more insistent proprioceptive feed-back which disrupts his concentration on which he is trying to say. Our experimental question is therefore a physiological one. To prove that Jones' heart rate is more labile requires us to show that his tonic heart rate is higher under a wide variety of stressors (allowing for the possibility that Smith's heart rate may be higher in certain situations

which, for some reason, have special significance for him). The appropriate unit of measurement is the raw physical unit, rate, since our question is concerned with the absolute activity of the effector organ--which will determine the level of visceral afferent feedback--rather than with the psychological state which that level implies for the individual. The same demonstration for a number of autonomic response systems would allow us to speak of individual differences in general autonomic reactivity.

As a rule, we may expect to find that Jones is more labile in some autonomic response systems while Smith is more labile in others. This generalization is what Lacey (Lacey, Bateman and Van Lehn, 1953) has called "the principle of autonomic response specificity." We might still ask whether Jones does not show greater lability at least in those systems which most easily disrupt performance in the public speaking situation: e.g., hyper- or hypo-secretion of the salivary glands, greater hand tremor, and the like. Note that if one is asking a psychological question--e.g., which subject is more aroused in this situation?--autonomic response specificity need not pose a problem of interpretation provided one employs the unit of measurement corrected for individual differences in range as discussed above.

## (2) Individual Differences in the Character of the Psychological Response.

Example: Remaining in the stage fright context, it has been argued that serious stage fright may result when an individual is for some reason angry at his audience; such anger is said to lead by the mechanism of projection to the conviction that the audience is angry with him, which in turn makes him afraid. To test this psychological hypothesis, we might employ autonomic measurement to determine whether Jones but not Smith betrays the peripheral accompaniments of anger at the start of his

performance. Thus, we might expect to find in Jones' record evidence of the autonomic pattern which Ax (1953) has shown to be characteristic of anger. A difficulty here is that Ax's results were given in terms of absolute physical units; e.g., his subjects averaged more spontaneous GSRs, higher diastolic blood pressure and a smaller increase in respiration rate during anger than during fear. Now suppose that Jones shows 12 GSRs per unit time, a diastolic blood pressure increase of 18 millimeters of mercury, and a respiration rate increase of less than two per minute--these values are about equal to the means of Ax's subjects under the anger condition. Can we therefore conclude that Jones is in fact angry?

One suspects that Ax himself would be uneasy about such an application of his findings. Suppose, for example, that Jones tends to be unusually labile with respect to both number of spontaneous GSRs and diastolic blood pressure and unusually stable with respect to respiration rate; the "principle of autonomic response specificity" tells us that such might well be the case. Jones might then tend to show an "anger-like" pattern in response to nearly any stimulus and, in our particular experiment, to show an anger pattern when he is in fact afraid.

Clearly, this problem devolves from a lack of intra-subjective standardization of units. Suppose Ax had expressed each individual's levels in both emotional conditions in relation to the maximum and minimum levels of which that individual was capable in each autonomic response system--i.e., in terms of the range-corrected units advocated herein. Then his results might have read something like this: "average diastolic blood pressure was .50 under anger and .30 under fear, the relative index of spontaneous GSRs was .60 under anger and .30 under fear, and relative

respiration .10 under anger and .40 under fear." Now suppose that Jones' relativized scores on these three variables are .60, .65 and .20, respectively. Noticing first of all that these scores are somewhat higher than the means for Ax's anger group, can we attribute this difference merely to the possibility that Jones has a generally higher autonomic reactivity? Probably not, since these are relativized scores from which differences in physiological reactivity have been already partialled out. We can conclude that Jones reacted psychologically more intensely to the situation we are using than did Ax's average subject to his anger stimulus, either because our situation was effectively stronger or because Jones is psychologically more reactive than the average person. Secondly, we note that the pattern of Jones' response is similar to the pattern of the mean anger response of Ax's subjects. Can this be again an artifact resulting from Jones' being hyper-active in the first two channels and hypo-reactive in the third? Probably not, again because these relativized scores are independent of mean reactivity differences, channel by channel.

Suppose that we had expressed Jones' responses as standard scores relative to the inter-subjective norms provided by the (absolute) responses of Ax's subjects, giving him scores of, say, +2.0, +1.5, and -1.5 respectively. This would give us somewhat more precise information as to how Jones' absolute compared with those of Ax's subjects; for example, we might now be able to say that Jones' diastolic blood pressure was 0.5 sigma greater than the mean of Ax's subjects under the anger condition and 2.5 sigma greater than their mean under the fear condition. However, the advantages of this inter-subjective relativization are more apparent than real; upon reflection, one can see that these data will still be very nearly as ambiguous with respect



to our hypothesis as they were in their original raw score form. The question we are asking is a psychological one--having to do with the character or quality of the subject's psychological response to the situation--and for this we require that our data be expressed in units relativized with respect to intra-subjective variability.

(3) Individual Differences in Tonic Psychophysiological Level.

Example: Judging from the autonomic response record, was the stage fright situation more psychologically disturbing for Jones than for Smith? This typical problem calls for the use of range-corrected units of tonic level. We are not concerned with whose skin conductance or heart rate is higher in absolute terms but rather we wish to evaluate the relative intensities of the underlying psychophysiological states. Nor do we measure the increment in corrected tonic level when the subject walks to the front of the room to give his speech because our interest is in his degree of arousal while speaking. We realize that his arousal level just before he is called on to perform may depend upon a variety of uncontrolled factors--whether he was expecting to be called on just then, how effective his psychological defense mechanisms may be, and the like--so that including even a range-corrected measure of "pre-stress" level in the process of computing increments will probably add only error to our assessment.

In some response systems which are under strong homeostatic control, the function relating tonic level with the underlying variable of interest may actually be non-monotonic so that increases in the latter above some critical value,  $\psi_p$ , may actually produce a decrease in the former,  $\phi$  [Wilder's (1962) "paradoxical response"]. This phenomenon was apparently regularly observed in Bridger and Reiser's (1959) study of heart rate in

neonates (although one would like to have seen independent evidence that the infants showing HR decrease were actually more aroused after stimulation than before). However, it is much less commonly seen in work with older subjects when one is careful to identify those cases where the subject was for some reason actually more aroused under the "pre-stress" condition so that his negative "pre-post" stress increment is actually a proper indication of his change in state rather than a "paradoxical" response. When  $\phi = f(\psi)$  is actually non-monotonic--i.e., not single-valued--no simple range-correction or other transformation can solve the problem and one's only recourse is the plotting of individual regressions of  $\Delta\phi$  on  $\phi_0$  as recommended by Bridger and Reiser (op. cit.)

Suppose we find that Jones' tonic SC was higher than Smith's but his tonic blood pressure was lower, even when both are expressed in range-corrected units? If we were using absolute physical units, such a result might be attributed to "autonomic response specificity." A common practice in such cases is to convert both scores into inter-subjective standard scores. Thus, relative to measures obtained for the total sample, Jones' SC level might be +2.0 sigma and his BP level +1.0 sigma while corresponding scores for Smith might be -1.0 and +1.5. Then such scores are averaged--Jones' mean being +1.5 and Smith's being +0.25--leading to the conclusion here that Jones was more disturbed by the situation than was Smith. However, finding such differences among autonomic response channels when levels are expressed in individual range-corrected units implies simply (and reasonably) that these two dependent variables are not measuring the same thing--that the psychophysiological process underlying variation in (corrected) skin conductance is not identical to the process governing

variations in (corrected) measures of blood pressure. Extrapolating from the interesting findings of Lacey et al (1963), we might speculate that, while Jones' higher SC indicates that he experienced greater general CNS arousal, Smith's higher BP shows that he was more actively inhibiting exteroceptive input so as to better concentrate on his speech.

(4) Individual Differences in Phasic Response to Specific Stimuli.

Finally, let us suppose that we have arranged for a series of disturbances to occur at intervals while each subject is delivering his speech; e.g, after 3 minutes, a voice from the back calls, "Speak louder", after 5 minutes a bell rings; two minutes later, a member of the audience gets up and leaves; etc. We measure the phasic autonomic response--e.g., the GSR--to each of these "standard" disturbing stimuli. We wish to know which subject is more distracted by such stimuli on the average as indicated by a greater phasic response. We assume that a subject may react differently to, e.g., a bell while giving a speech than he might under other circumstances and therefore we must expect some sort of relationship between his phasic response to the bell and his tonic level under the stage fright condition. We wish and expect this relationship to display itself in the final results of the experiment, from which we intend to draw our inferences about the psychological processes which were at work. But we realize that these phasic and tonic output variables may also be related in some manner for other, extraneous reasons--such as homeostatic restraint or the biophysical peculiarities of the peripheral effector organ--and we do not wish to retain the influence of this sort of relationship in the data as finally analyzed. In particular, if the function relating the measure of tonic level to the underlying variable of interest is non-linear, then we know that measures of phasic response will necessarily display a spurious relationship to

pre-stimulus level. If this function,  $\phi = f(\psi)$ , is known, then one can compute from  $\Delta\phi$  an index which estimates  $\Delta\psi$  independently of any spurious correlation with initial level,  $\phi_0$ , as illustrated for the case of the GSR above in equations 11 and 12.

### Other Approaches

#### Normalizing Transformations.

An approach to the problem of finding the "correct unit" in which to express psychophysiological data which was popular some years ago involved trying various algebraic transformations on the raw measures, searching for one which would yield a normal distribution of the sample values. For example, since GSR distributions are often positively skewed, logarithmic or square-root transformations were frequently applied. Such scalar changes may be useful if one plans to employ statistical methods which involve distributional assumptions but it is clear that the data should not be transformed for this purpose unless and until it has first been adjusted to provide the best possible index of the underlying variable of interest, along the lines suggested above. GSR data, for example, should first be expressed in units of conductance-change,  $\Delta SC$ , then adjusted for individual differences in SC range [by applying the formula  $\Delta\phi = \Delta SC / (SC_{\max} - SC_{\min})$ ], and then finally adjusted for non-linearity in the relation of  $\phi$  to  $\psi$  so as to provide a linear estimate of  $\Delta\psi$  [e.g., by application of equation 11 or 12, if  $\phi = f(\psi)$  is a growth function]. If now the sample distribution of  $\hat{\Delta\psi}$  is skewed and one wishes to employ ANOVA or some other normality-assuming procedure, an appropriate transformation of scale may be applied to the  $\hat{\Delta\psi}$  values.

In some of the earlier literature it is implied that if the transformation  $Y = g(X)$  normalizes the sample distribution of the raw autonomic measures,  $X$ , then the transformed variable,  $Y$ , must be a better estimate of the true underlying variable,  $\psi$ . This particular non sequitur has tended to infect psychometric thinking since the days of Quetelet and is to be sedulously avoided. The so-called Normal Law of Variation which Quetelet introduced was never ratified by the Almighty and one has no a priori assurance whatever that the population distribution of any individual difference variable has any particular form. Similarly, the notion that a transformation of some measure of autonomic change, which eliminates any correlation between the adjusted change score and the initial tonic level, must therefore be a better measure of the true change in the underlying variable of interest is also specious though seductive. We shall repeat one last time that, if the initial tonic level has some significance concerning the state of the organism at the time of stimulation, then clearly it is usually to be expected that a measure of the organism's response to stimulation at that time will be in some way related to the tonic level; a "blind" algebraic procedure which is designed specifically to eliminate any vestige of correlation between change and initial level must in general serve to distort the data and to increase the error variance.

Lacey's Autonomic Lability Score. In a thoughtful and widely quoted analysis of the problems of autonomic measurement, Lacey (1956) proposed what was intended to be a general method of evaluating measures of autonomic change, both phasic changes and changes in tonic level. Lacey is specifically concerned with the difficulties imposed by the action of the

so-called "Law of Initial Values" (Wilder, 1962) which asserts that an excitatory stimulus evokes smaller increments when pre-stimulus activity already is high while an inhibitory stimulus produces greater decrements from elevated tonic levels than when pre-stimulus activity is already low. In the terms of the present analysis, this alleged "Law" is contained in the somewhat more general proposition that the function  $\phi = f(\psi)$  is non-linear. For example, if  $\phi = f(\psi)$  is a growth function, then the LIV will hold as stated; a given increment in  $\psi$  will yield a smaller increment in  $\phi$  as the initial tonic level,  $\phi_0$ , increases. That is, the increment,  $\Delta\phi$ , produced by a given change in the underlying process,  $\Delta\psi$ , will be a function of the pre-stimulus level,  $\phi_0$ , viz.  $\Delta\phi = (1-\phi_0)g(\Delta\psi)$ , and the correlation between  $\Delta\phi$  and  $\phi_0$  thus produced will be spurious in the sense that it will detract from the accuracy of  $\Delta\phi$  as an index of  $\Delta\psi$  when initial level,  $\phi_0$ , is allowed to vary.

Now the actual relation of  $\phi$  to  $\psi$  has yet to be determined for any psychophysiological response system. There are adequate grounds for confidence that the nature of this function will vary from one system to another. We have suggested above methods by which the form of this relationship might be determined empirically from appropriate measures of activity in a given system. Lacey's Autonomic Lability Score was designed to deal with this problem in a very general way by means of regression analysis. Specifically, the ALS is the difference between the observed post-stimulus level,  $TL_1$ , and the value predicted from the pre-stimulus level,  $TL_0$ , on the basis of the observed regression of  $TL_1$  on  $TL_0$ , this difference being expressed in T-score units (with mean = 50 and SD = 10).

To illustrate this method, we shall return to the earlier example of

assessing individual differences in "stage fright" from measures of SC obtained while the subject is giving a speech. To compute the ALS, one would measure each subject's  $SC_0$  before he gets up to perform and his  $SC_1$  while performing and correlate these two values across the sample. One would then compute the usual linear regression equation for estimating  $SC_1$  from  $SC_0$ , i.e.

$$\hat{SC}_{1(i)} = (\overline{SC}_1 - r_{01} \frac{s_1}{s_0} \overline{SC}_0) + (r_{01} \frac{s_1}{s_2}) SC_{0(i)}.$$

From this, one computes the ALS,

$$ALS_{(i)} = 10 \frac{SC_{1(i)} - \hat{SC}_{1(i)}}{s_1 \sqrt{1 - r_{01}^2}} + 50.$$

Thus, by converting the "post-stress"  $SC_1$  values to Autonomic Liability Scores, we can be assured that these scores will be uncorrelated (across subjects, R-type correlation) with their  $SC_0$ s obtained prior to performing. But one can argue that the arousal (or anxiety) shown by a subject while performing should be correlated with his level of arousal while waiting to perform; we have repeatedly inveighed against "blind" statistical procedures designed to eliminate such correlations from the data. In fact, the ALS procedure eliminates any R-type correlation between  $SC_0$  and  $SC_1$  but does not eliminate the correlation between the better measures of pre- and post-stress level,  $\phi_0$  and  $\phi_1$ ; the ALS assumes that the regression across subjects of  $SC_1$  and  $SC_0$  is a close estimate of the regression of post-stress on pre-stress conductance which one would observe in repeated measures of the same subject and this assumption does not generally hold due to individual variation in the range parameters. Without attempting a detailed critique here, these considerations alone may be sufficient to suggest that the ALS has outlived its usefulness.

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