Lykken, D. T., & Meehl, P. E. (1966). *Contributions to the problem of evaluating autonomic response data: I* (Report No. PR-66-2). Minneapolis: University of Minnesota, Research Laboratories of the Department of Psychiatry.

CONTRIBUTIONS TO THE PROBLEM OF EVALUATING AUTONOMIC RESPONSE DATA: I

David T. Lykken and Paul E. Meehl
University of Minnesota

A correction procedure previously described (Lykken, Rose, Luther & Maley, 1966) is designed to eliminate the effects upon autonomic measurements of individual differences in peripheral effector characteristics, which are usually irrelevant to the psychological processes in which one is actually interested. The use of this correction procedure yields an index, ϕ, which is largely free of what may be a sizeable component of error of estimate relative to the underlying variable of interest, ψ. 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. Two separate methods which could be used to specify these relationships more clearly are outlined below.

First Method. For purposes of illustration, we shall consider the problem of determining the function relating the range-corrected index of tonic skin conductance, $\overline{\phi}_{sc}$, to the underlying variable "arousal." We shall assume that the form of this function is the same 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 Fig. 1, we have plotted the inter-subjective distributions of corrected ϕ_{sc} values which might be obtained for six such stimulus situations with the latter arranged along the abscissa in order of increasing average ϕ_{sc}. These hypothetical distributions have been drawn with a positive skew to allow for the fact that some subjects will show high activation (and high ϕ) 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. 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.

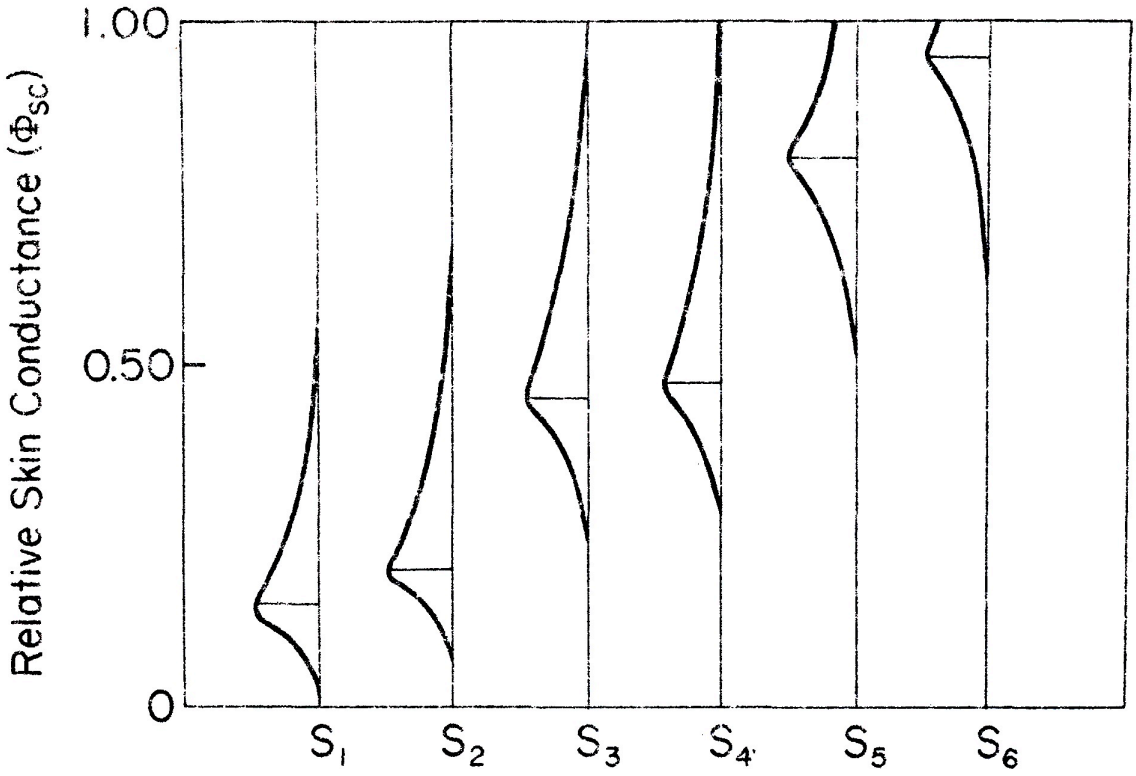

Figure 1. Hypothetical distributions (across subjects) of skin conductance scores corrected for individual differences in range. Six stimulus situations, S_1, ... S_6, are chosen to span the range from low to high average arousal value and are arranged here in order of increasing average ϕ_{sc} (and thus, ex hypothesi, in order of increasing ψ).

Even in terms of the mean ϕ 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 ϕ will be monotonic-increasing, as shown in Figure 2.

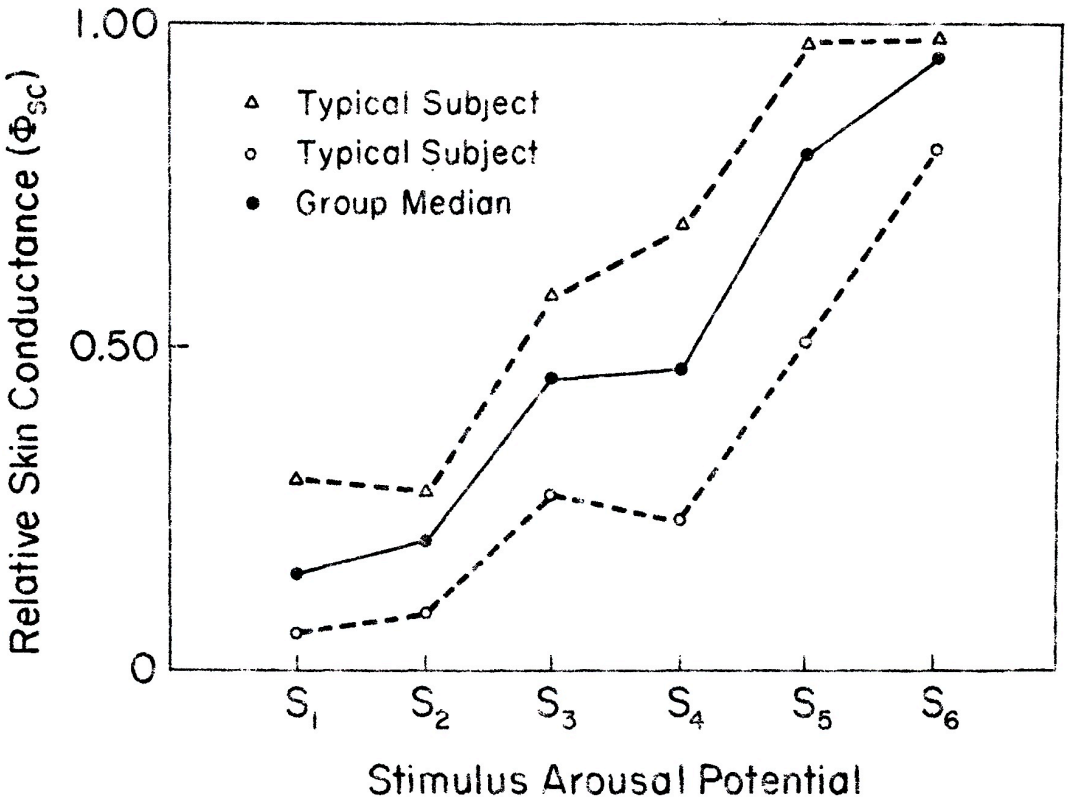

Figure 2. The medians of the six hypothetical distributions shown in Fig. l, together with the ϕ_{sc} scores of two "typical" individual subjects in each of the six stimulus situations.

The next step in the process is to alter the intervals between stimuli on the abscissa so as to make the plot of mean ϕ 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 3, the inter-stimulus intervals on the abscissa have been expanded or compressed as necessary to convert the ϕ means into a linear array. Holding this arrangement fixed, we now fit a straight line to each individual's set of ϕ values, finding the parameter, ρ_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.

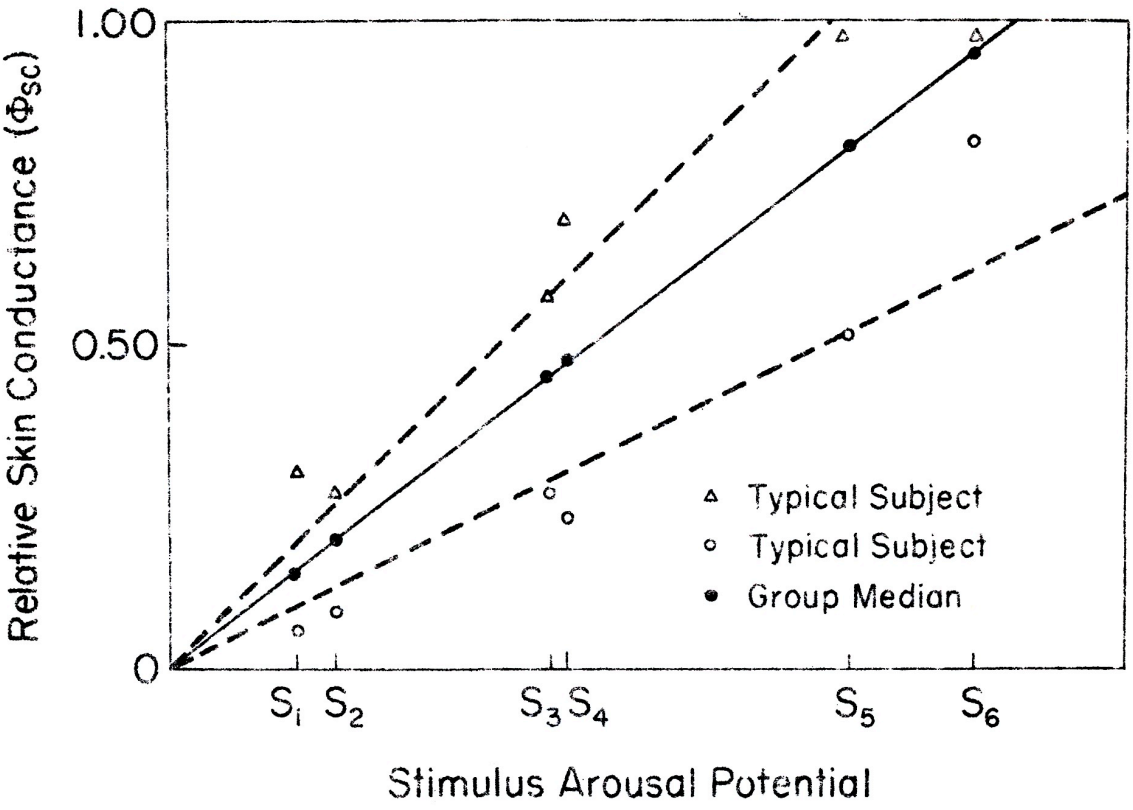

Figure 3. Same data as in Fig. 2 except that spacing of S_1, S_2, ...S_6 on the abscissa has been changed to force group medians into a linear array including the origin (0). Straight lines have been fitted to the two sets of individual scores, showing rather poor fits in both cases.

Since ϕ cannot rise above 1.00 and we are assuming that activation is maximal at that point, same 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 ϕ 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 ϕ 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 ϕ to ψ more accurately. As indicated in Figure 4, we might now change the inter-stimulus distances on the abscissa so as to force the median values of ϕ 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 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. (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. We have not attempted to work out what special

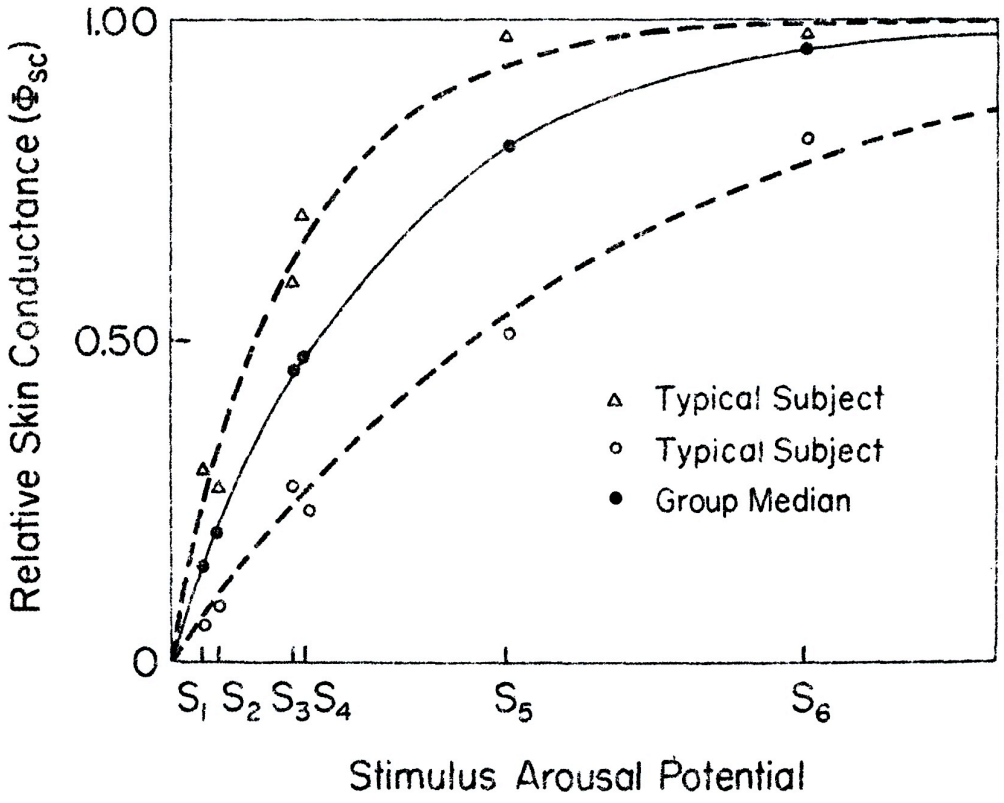

Figure 4. Same data again but with spacing of S_1, S_2, ...S_6 on the abscissa altered to force group medians to fall on a growth curve from the origin (0). Similar functions have been fitted to the two sets of individual scores, showing rather good fits in both cases.
[Note: Images for figures 4 & 5 were interchanged in original publication.—LJY]

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, ϕ, and the hypothetical underlying variable, ψ.

Thus far we have been plotting on the abscissa, not ψ, 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 4, for example, a stimulus having the arousal value of S_5 produces a ϕ 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 5. From this curve, we can infer that S_5 produced about 23 percent of maximum arousal in our first subject (whose ϕ was 0.50), about 99 percent of maximum arousal in the second subject (for whom ϕ was 0.95) and about 54 percent of maximum ψ on the average in the group as a whole.

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 ψ 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., $\phi_j = \log \alpha_i \psi_j$, 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 $\alpha = -\log_e .05 \cong 3$. Thus, the general curve we plot in Figure 5 is $\phi_{ij} = 1 - e^{-3\psi_j}$.

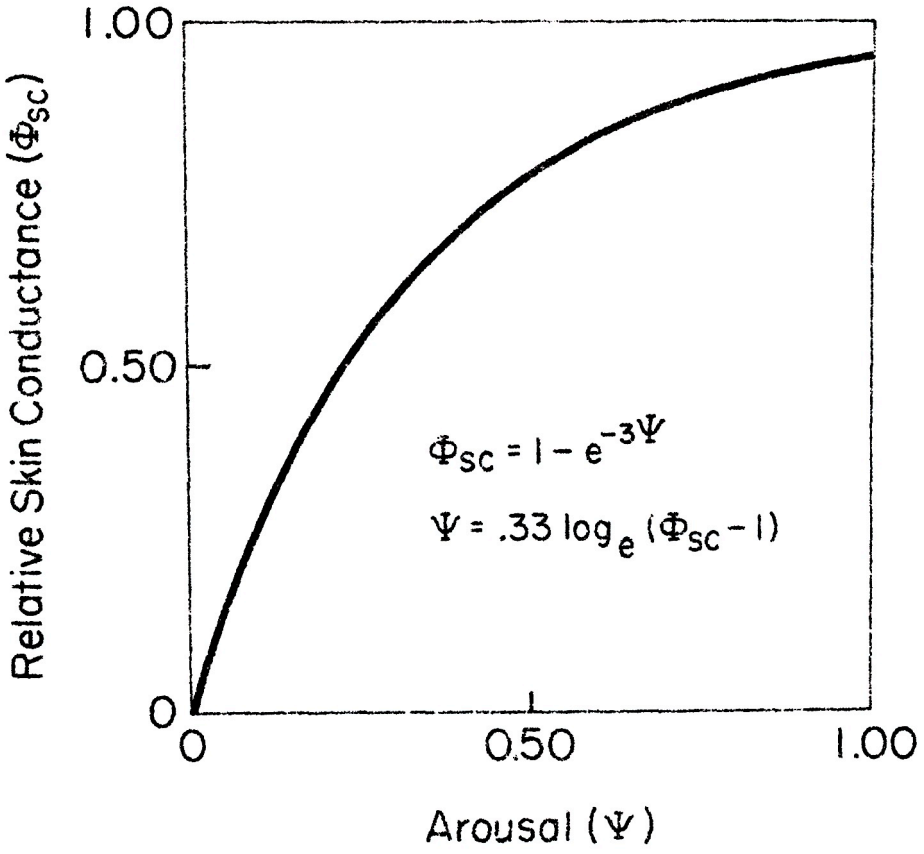

Figure 5. Growth function relating ϕ_{sc} to ψ. This function might be derived from the hypothetical data of Fig. 4 by "relativizing the abscissa" (see text). The relationship of ϕ_{sc} to ψ illustrated by this curve is tabulated in Table 1.
[Note: Images for figures 4 & 5 were interchanged in original publication.—LJY]

This curve (Figure 5) 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.

The hypothetical data on which Figure 5 is based are listed in Table 1 and were calculated on the assumption that the present approach might indicate that ϕ is a growth function of ψ, $\phi = 1 - e^{-3\psi}$. 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 of measurement. Then each raw conductance level, SC_{ij}, obtained from the ith 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 1 from which the associated estimates of true activation level could be read.

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

Second Method. Another, and independent, method of determining the relationship of ϕ to ψ may be employed when one has measures of two (or more) variables, ϕ_x and ψ_y, both of which are believed to be related to the same underlying variable, ψ. For example, if ϕ_{sc} is some function of activation, $f(\psi)$, and if volar skin potential (sp) similarity corrected, (ϕ_{sp}), 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 of arousal at the time of measurement and obtain

from them measures of ϕ_{sc} and ϕ_{sp} (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 $\psi = (1/\alpha)\log_e(\phi_{sc} - 1)$; the inverse of $\phi_{sp} = \beta/\psi$ is $\psi = \beta/\phi_{sp}$. If these two functions, respec–tively, provide a good approximation of the true relationship between ϕ_{sc} and ψ and between ϕ_{sp} and ψ, then the correlation between $[(1/\alpha)\log_e(\phi_{sc} - 1)]$ and $[\beta/\phi_{sp}]$ 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 1 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 ϕ alone. In analyzing an experiment in which tonic SC level figures as a dependent variable, one might reasonably use ϕ_{sc} first and then to repeat the analysis using estimates of ψ from Table 1 $[\psi = .33 \log_e(\phi - 1)]$ to see whether this transformation did not help to clarify the 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 ϕ_{sc}, $\Delta\phi_{sc} = \phi_1 - \phi_O$, resulting from an increment in sudomotor innervation, $\Delta\psi = \psi_1 - \psi_O$. If the relation of ϕ_{sc} to ψ is linear, then $\Delta\phi_{sc}$ will be a linear function of $\Delta\psi$ and uncorrelated with the pre-stimulus level ϕ_O (except insofar as $\Delta\psi$ is correlated with ψ). But, if $\phi_{sc} = f(\psi) = 1 - e^{-\alpha\psi}$, then:

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

Therefore,

(2) $$\Delta\phi = e^{-\alpha\psi_O}\ (1 - e^{-\alpha(\Delta\psi)})$$

or

(3) $$\Delta\phi = (1 - \phi_O)(1 - e^{-\alpha(\Delta\psi)}).$$

Thus, if ϕ is a growth function of ψ, then $\Delta\phi$ will be a growth function of $\Delta\psi$ (with ψ_O or ϕ_O held constant) and an inverse linear function of ϕ_O (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 3 for $\Delta\psi$),

(4) $$\widehat{\Delta\psi} = (1/\alpha)[\log_e(1 - \phi_O) - \log_e(1 - \phi_O - \Delta\phi)]$$

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

(5) $$\widehat{\Delta\psi} = (1/\alpha)[\log_e(SC_{mx} - SC_o) - \log_e(SC_{mx} - SC_1)],$$

where SC_{mx} is the upper limit of that individual's conductance range, SC_O is the pre-stimulus tonic level, SC_1 is the peak post-stimulus conductance ($SC_1 = SC_O + \Delta SC$), and α is the parameter of the function $\phi_{sc} = 1 - e^{-\alpha\psi}$. That is to say that, <u>if</u> the relationship of the corrected tonic level, ϕ, to the underlying process, ψ, 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 proceeding 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 <u>is</u> independent of pre-stimulus level (e.g., by using equation (5) 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, Φ_O)

However, since the relation of Φ to ψ 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 $\Delta\phi = f(\phi_O)$. 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 ϕ_O will be ambiguous since it will confound the effects of a non-linear relation of ϕ to ψ (which effects one will want to remove) with the effects of any possible relation of $\Delta\psi$ to ψ_o (which <u>are</u> 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, ψ_o, 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

$$\Delta\Phi_{ij} = \Delta SC_{ij}/[SC_{i(max)} - SC_{i(min)}] \quad (6)$$

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 ΔSC_{ij} alone. Pending determination of the true relation of ϕ to ψ, one must keep in mind that an unknown portion of any observed correlation between $\Delta\phi$ and ϕ 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 5 may be used.

References

1. Estes, W. K. (1956). The problem of inference from curves based on group data. *Psychol. Bull*, *53*, 134-140.
2. Lykken, D. T., Rose, R., Luther, B., & Maley, M. (1966, submitted). Correcting psychophysiological measures for individual differences in range. [Submitted to *Science*, January, 1966.]
3. Sharpless, S., & Jasper, H. H. (1956). Habituation of the arousal reaction. *J. Brain*, *79*, 655-680.