

METHODOLOGY

Origins of baseline variance and the Law of Initial Values

GARY G. BERNTSON, BERT N. UCHINO, AND JOHN T. CACIOPPO

Department of Psychology, Ohio State University, Columbus

Abstract

The Law of Initial Values (LIV) asserts that the magnitude of a phasic psychophysiological response is dependent on the initial baseline level. Although results in accord with the LIV are often observed, exceptions are frequent, especially for between-subjects analyses. A general assumption in studies of the LIV is that a given baseline difference is equivalent regardless of its functional origin. The present study examined the relationships between basal heart period variance, arising from alternate sources, and the magnitude of the chronotropic response to a speech stressor. Results reveal that baseline differences due to orthostatic manipulations, which are known to be largely of autonomic origin, yielded larger LIV effects than did individual differences in basal heart period, which include a significant nonautonomic component. The minimal LIV-like effects of baseline differences associated with nonautonomic factors, relative to variations in autonomic control, may contribute to the inconsistent appearance of between-subjects LIV effects.

Descriptors: Autonomic nervous system, Baseline, Heart period, Heart rate, Initial levels, Law of Initial Values, Orthostatic manipulations, Speech stress, Terminal levels

The Law of Initial Values (LIV) asserts that the magnitude of a phasic psychophysiological response is dependent on the initial baseline level. Specifically, Wilder (1931, 1957, 1967) suggested that increases in baseline level may limit or constrain further incremental responses and may enhance subsequent decremental responses. Although the LIV has potentially profound implications for psychophysiology, it has experienced a rather checkered history. Historical challenges have ranged from suggestions that the LIV may (a) hold only for some organ systems (Hord, Johnson, & Lubin, 1964), (b) be state dependent (Hutt & Hutt, 1970), (c) apply only within subjects (Scher, Furedy, & Heslegrave, 1985), or (d) represent a statistical artifact (Myrtek & Foerster, 1986). Given frequent inconsistencies, Stern, Ray, and Davis (1980) prefer the designation of *principle* rather than *law* of initial values, and Furedy and Scher (1988) have argued that the LIV is more appropriately viewed as an empirical generalization rather than a methodological rule.

Especially inconsistent have been the between-subjects predictions of the LIV. Wilder (1967) maintained that the Law of Initial Values is a statistical assertion, which would be expected to hold in the aggregate but not necessarily in any given instance. Wilder was especially cautious about between-subjects manifestations of the LIV, given the wider range and greater variance of the determinants of psychophysiological response across subjects. While others have questioned on statistical grounds any

fundamental distinction in within-subjects and between-subjects LIV analyses (Myrtek & Foerster, 1986), between-subjects comparisons have been much more likely to yield inconsistent results. This has led some authors to recommend the "repeal" of the between-subjects version of the LIV (Furedy & Scher, 1988). Before such a litigious action, however, Wilder's caution concerning the wider sources of variance in between-subjects analyses may be worth further consideration.

A frequent implicit assumption in studies of the LIV is that a given baseline difference is equivalent regardless of its functional origin. As Wilder cautioned, this assumption may not always be warranted (see also Jin, 1992). The short baseline heart periods of infants, for example, are not necessarily associated with the same constraints on psychophysiological response as equally short heart periods in adults. Even for adults, baseline variance arising from distinct functional origins may have differential effects on the magnitude of subsequent phasic responses. Both autonomic and nonautonomic factors can contribute to basal heart period (Berntson, Cacioppo, Quigley, & Fabro, 1994; Jose & Taylor, 1969; Lewis, Nylander, Gad, & Areskog, 1980). Individual differences in baseline heart period have sizeable nonautonomic components, related to factors such as age and aerobic capacity, that contribute to differences in intrinsic heart period even after autonomic control has been pharmacologically blocked (Berntson et al., 1994; Jose & Taylor, 1969; Lewis et al., 1980). These nonautonomic factors may thus entail a shift of the overall operating range of autonomic control to a higher (or lower) level, without altering the dynamic range or the basal locus within that range. Consequently, non-

Address reprint requests to: Gary G. Berntson, 48 Townshend Hall, Ohio State University, Columbus, OH 43210.

autonomic contributions to individual differences in baseline state may have relatively little impact on the magnitude of phasic response.

In contrast, baseline variance associated with changes in autonomic outflow can shift the basal locus within the dynamic range of autonomic control. Orthostatic manipulations, for example, are known to alter baseline chronotropic levels largely through autonomic mechanisms (Head & McCarty, 1987; Robinson, Epstein, Beiser, & Braunwald, 1966; Spyer, 1981). Because the dynamic range of autonomic control is limited (Berntson, Cacioppo, & Quigley, 1991; Berntson et al., 1994), baseline differences related to variations in autonomic control would likely be associated with altered constraints on subsequent phasic responses. Thus, basal differences in heart period due to postural manipulations may yield sizeable LIV effects, even for between-subjects analyses.

In the present paper, we describe an explicit experimental test of both the within- and between-subjects predictions of the LIV for the chronotropic response to stress. We further directly compare the between-subjects effects of basal heart period variance related to individual differences, which include a sizeable non-autonomic component, and to orthostatic manipulations, which are largely autonomically mediated. Results reveal that orthostatic manipulations of baseline heart period yield consistent within- and between-subjects effects on phasic heart period response, in accord with the LIV. In contrast, comparable baseline variance associated with individual differences in heart period is largely without effect on the magnitude of response. Results document the differential implications of baseline differences arising from distinct functional origins.

Method

Subjects

Subjects were 68 healthy undergraduate women, aged 17–30 years ($M = 18.76$). Subjects received either experimental credit or \$5.00 for approximately 2 hr of participation in a study of individual differences in the psychophysiological response to stress. Results of the broader study will be reported separately. The inclusion criteria were that subjects (a) be in good health, (b) be within 20% of their ideal body weight, (c) not be well-conditioned athletes, (d) not have experienced any recent traumatic negative life event (e.g., death in the family), (e) have no class exam on the day of the test, (f) have no self-reported problem with math, speeches, or needles, (g) have no history of psychological disorder or chronic illness, (h) not be on any chronic medication or use recreational drugs or tobacco products, and (i) not consume more than 10 alcoholic beverages per week. Subjects were asked to refrain from ingesting anti-inflammatory agents, antihistamines, or alcohol during the 24 hr preceding the test day.

Apparatus

A Minnesota Impedance Cardiograph (Model 304B) was used to measure electrocardiograms (ECG), basal thoracic impedance (Z_0), and the first derivative of the impedance signal (dZ/dt). Disposable ECG spot electrodes were placed in a tetrapolar configuration as proposed by Qu, Zhang, Webster, and Tompkins (1986). The two outer (current) electrodes were placed over the fourth cervical and the ninth thoracic vertebrae, and the two inner (recording) electrodes were placed 4 cm above the clavicle and over the sternum at the level of the fourth rib. A 4-mA

ac current at 100 kHz was passed through the two outer electrodes, while the Z_0 and dZ/dt signals were recorded from the two inner electrodes. The ECG signal was digitized, and inter-beat intervals were derived from a custom software package. Outliers in the heart period records were identified and corrected, with artifact resolution confirmed by the artifact detection algorithm of Berntson, Quigley, Jang, and Boysen (1990). Only the heart period data will be reported here.

Procedure

Following informed consent, subjects spent approximately 20 min in the laboratory, during which time they filled out several psychological surveys and questionnaires (not reported here). Electrode sites were then cleaned with alcohol and the electrodes were attached. Shortly thereafter, cardiovascular measures were obtained over 2-min baseline periods while the subjects were seated and while standing. The order of postural testing was counterbalanced across subjects, and 30 s were allowed after assumption of a given posture before baseline measures were begun.

After baseline testing, a speech stress manipulation was imposed. Subjects were asked to imagine they had been accused of shoplifting by a department-store security guard. The subject was instructed to prepare a 4-min speech to (a) tell their side of the story to the store manager, (b) explain what the security guard did wrong and why the guard may have suspected them of shoplifting, (c) show how they can prove they did not steal the item, (d) specify what should happen to the security guard for this mistake, and (e) summarize their points. Subjects were instructed to give intelligent, prepared answers because their speech would be recorded and compared with the speeches of others.

Subjects were given 4 min to prepare and 4 min to present their speeches. Half (2 min) of the speech was delivered while seated and half while standing, with the order of postural testing counterbalanced across subjects.

Results

Baseline Heart Period

The postural manipulation yielded the expected effect on heart period. Baseline data were analyzed by a mixed analysis of variance (ANOVA), with posture (sitting vs. standing) as a within-subjects variable and order of testing (sitting–standing vs. standing–sitting) as a between-subjects variable. The orthostatic challenge associated with upright posture yielded a significant shortening of heart period (baseline heart period while seated, $M = 824 \pm 15$ SEM; while standing, $M = 699 \pm 11$; $F[1,66] = 228.0$, $p < .001$). The order of postural testing did not significantly influence baseline heart period values, for either the sitting or the standing condition, as evidenced by a lack of a main effect or interaction on this term.

The stability of baseline heart period was evaluated by Cronbach's alpha across the first and second minutes of the 2-min baseline periods. This statistic revealed a high reliability of the baseline measures for both the sitting ($\alpha = .98$) and standing ($\alpha = .98$) conditions, indicating a low baseline measurement error.

Group Analyses of LIV Effects

Baseline heart period was experimentally manipulated in the present study by postural alterations. The effects of this manip-

ulation on heart period response to the speech stressor were evaluated both within and between subjects through analysis of variance. These analyses provide a direct test of the effects of experimental manipulations of basal heart period, which has a distinct advantage over strictly correlational procedures.

Within-subjects analyses. All subjects were tested while both seated and standing, with the order of testing counterbalanced across subjects. Each subject was thus tested under all conditions, and an overall analysis provides a within-subjects evaluation of the experimental manipulations. As illustrated in the left-hand functions of Figure 1, speech stress resulted in a significant shortening of heart period under both sitting and standing conditions, although the tachycardiac response to stress was considerably larger when the subjects were seated. Our primary interest was in the magnitude of the heart period response to the stress manipulation, rather than the absolute heart period levels achieved. Consequently, the magnitude of the heart period change was analyzed by a mixed ANOVA with posture (sitting vs. standing) as a within-subjects variable and testing order (sit-stand vs. stand-sit) as a between-subjects variable. A significant main effect of posture reflected the larger heart period response to stress under the sitting condition ($F[1,66] = 110.6, p < .001$). No significant main effect of order emerged, indicating that the smaller response under the standing condition was not attributable to the order of the test postures. A significant Posture \times Order interaction, however, was apparent ($F[1,66] = 64.0, p < .001$). This reflected an overall decline in the magnitude of stress response in the second stress test irrespective of posture (response on first stress test, $M = -142$ ms; on the second stress test, $M = -76$ ms).

These results are in accord with the LIV and reveal an overall within-subjects attenuation of heart period decrement to

stress when baseline heart period levels are experimentally decreased by postural manipulations.

Between-subjects analyses. For the above analyses, each subject was tested under both postural conditions, and the condition means were evaluated by a repeated-measures ANOVA for within-subjects effects. In the first 2-min period of speech stress, half of the subjects ($n = 34$) were tested while seated and half ($n = 34$) while standing. To evaluate the between-subjects predictions of the LIV, the results of the first stress test were submitted to an ANOVA on heart period response as a function of posture. As depicted in the center functions of Figure 1, the standing group displayed a considerably smaller response to the speech stress than did the seated group ($F[1,66] = 38.2, p < .001$). Postural conditions were reversed for the second 2-min stress period. As illustrated in the right-hand functions of Figure 1, a similar pattern of results was apparent for the second period. While the overall magnitude of the stress response was reduced in the second stress period, responses for the standing group were again significantly smaller than for the seated group ($F[1,66] = 14.7, p < .001$).

These findings are again in accord with the LIV and reveal a consistent and significant between-subjects attenuation of heart period decreases to a stressor when baseline heart period is experimentally controlled by a manipulation that alters autonomic tone.

Regression Analyses

A standard representation of the LIV is the regression of the mean heart period under reactive challenge against the mean heart period under baseline conditions (Bridger & Reiser, 1959; Hord et al., 1964; Surwillo & Arenberg, 1965). Results of regression analyses of the present data are illustrated in Figure 2. For these representations, a regression function with a slope of 1.0 would indicate the absence of a baseline dependency for the response to the challenge stimulus. If no response was observed to the stimulus, the regression function would lie along the depicted diagonal in Figure 2. If a response was evoked by the challenge condition but was uninfluenced by baseline levels, the regression function would lie parallel to, but above (bradycardiac response) or below (tachycardiac response), the diagonal.

Statistical regression to the mean, in the absence of change in mean level, could contribute to an apparent LIV effect. In view of the high reliability of the replicate heart period values within the baseline condition (Cronbach's $\alpha_s = .98$), regression to the mean from quasi-random fluctuations in heart period would be expected to minimally confound the present analyses. Surwillo and Arenberg (1965) suggested that a regression of two repeated baseline measures could provide an index of the potential contamination by this type of measurement error. Figure 2 (left panel) illustrates this analysis for the first and the second minutes of the baseline measures. As is apparent, the regression function lies closely along the diagonal, indicating that regression to the mean produced only negligible LIV-like effects under baseline conditions.

Results of the first stress test are illustrated in the middle panel of Figure 2, which depicts the between-subjects regression of mean heart period during stress against the corresponding baseline values. Half the subjects were tested while seated and half while standing. The slope of the regression function ($b_{y,x} = .53, p < .001$) is significantly less than 1.0 ($t[df = 66] = 6.71, p < .001$), suggesting a dependency of response magnitude

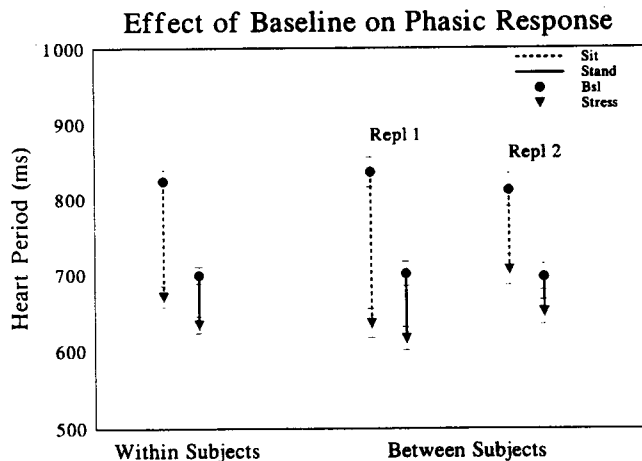


Figure 1. Heart period response to stress as a function of experimentally imposed alterations in baseline heart period. The functions to the left illustrate the within-subjects means and standard errors of heart period during baseline (solid circles) and during stress (inverted triangles), as a function of posture. The lengths of the arrows depict the magnitude of the heart period response to the speech stressor. Corresponding between-subjects data for the first stress period are illustrated by the functions in the middle, and results for the second stress period are depicted in the functions to the right. In accord with the LIV, the tachycardiac response to stress in each case was significantly smaller during the shorter basal heart periods associated with standing.

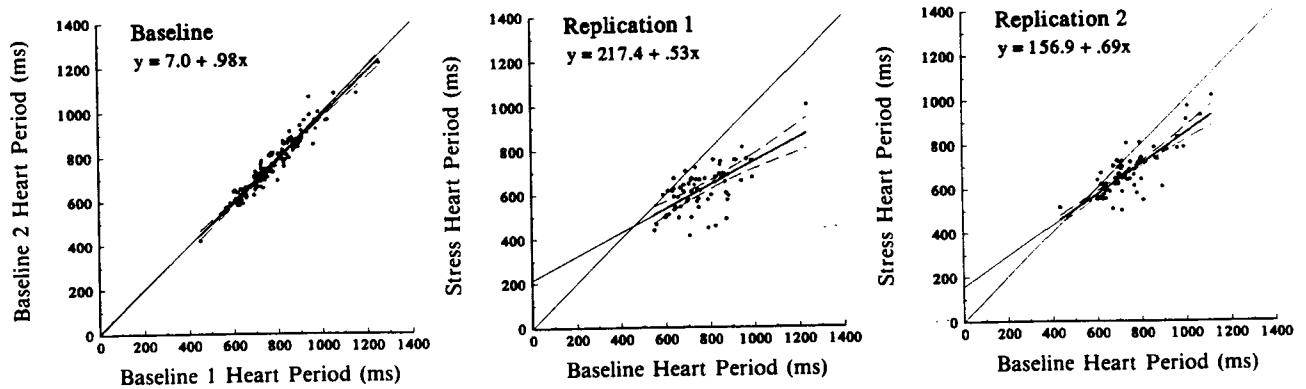


Figure 2. Regression analyses of LIV manifestations. The left panel illustrates the regression of heart period over the two 1-min periods of the baseline epoch, which reveals minimal LIV-like effects from regression to the mean. The middle panel illustrates the between-subjects regression of heart period under stress on baseline heart period. The regression slope is significantly less than 1.0, in accord with the LIV. The right panel depicts the corresponding between-subjects regression for the second stress period. The slope is again significantly less than 1.0. The thin dashed lines illustrate the 95%-confidence intervals of the slopes.

on baseline level. In accord with the LIV, decreases in baseline heart period were associated with a progressive attenuation of the decremental heart period response to stress. Myrtek and Foerster (1986) and Jin (1992) questioned the use of the regression slope in assessing LIV effects and recommended an alternate slope index (β), as proposed by Kendall and Stuart (1967). As illustrated in the summary data of Table 1, the β estimate for the present data yielded a higher slope than regression analysis ($\beta = .72$ vs. $b_{y \cdot x} = .53$). In accord with the LIV, however, the statistical test recommended by Geenen and van de Vijver (1993) continues to reveal a significant deviation of the slope from unity ($t[df = 66] = 2.50, p < .02$).

As illustrated in the right panel of Figure 2, a similar pattern was observed for the second stress test, in which postural condition was reversed for the two groups. The slope of the regression function was again significantly less than 1.0 ($b_{y \cdot x} = .69, t[df = 66] = 5.27, p < .001$), although it was steeper than that for the first stress test (.69 vs. .53), likely reflecting habituation of the stress response over replications. The slope estimate β was higher than that of the regression function but again was significantly lower than 1.0 ($\beta = .81, t[df = 66] = 2.50, p < .02$).

Table 1. Summary Statistics

Data set	n	Baseline		Stress		$r_{x \cdot y}$	$b_{y \cdot x}$	β^a	t_{GV}^b
		M	SD	M	SD				
Replication 1	68	768	125	626	100	.67	.53	.72	2.50*
Sit	34	836	119	636	112	.74	.70	.92	0.74
Stand	34	701	92	616	87	.71	.67	.93	0.61
Replication 2	68	754	128	679	107	.82	.69	.81	2.50*
Sit	34	812	129	706	114	.82	.72	.86	1.75
Stand	34	697	99	652	95	.83	.80	.95	0.65

^aError corrected estimate of slope, as recommended by Myrtek and Foerster (1986), Jin (1992), and Geenen and van de Vijver (1993).

^bt test for significant deviation of slope from 1.0, as recommended by Geenen and van de Vijver (1993).

* $p < .02$, two-tailed.

The LIV and the Functional Origins of Basal Differences

Heart period changes from orthostatic manipulations are known to arise largely from alterations in autonomic outflows (Berntson et al., 1991, 1994; Head & McCarty, 1987; Robinson et al., 1966; Spyer, 1981). In contrast, individual differences in baseline heart period have a sizeable nonautonomic component related to factors such as age and aerobic condition (Berntson et al., 1994; Jose & Taylor, 1969; Lewis et al., 1980). In view of these considerations, the present data were further analyzed to examine the relative LIV effects of baseline differences related to orthostatic manipulations and to individual differences. To avoid confusion from the apparent habituation over the stress periods, this analysis was limited to the first stress period. These between-subjects analyses were thus run on a consistent data set from 34 subjects tested under seated conditions and 34 tested while standing.¹

Between-group effects of orthostatic manipulations on both baseline and stress heart periods are illustrated by the solid line in Figure 3. As documented earlier, orthostatic manipulations had a significant effect on baseline heart period. Moreover, in accord with the LIV, these baseline differences yielded large and significant effects on the magnitude of the heart period response to stress.

A second source of between-subjects variance in these data is within-group individual differences in mean heart period. The LIV manifestations of these individual differences were derived from separate regression functions calculated for the two subject groups. These functions are illustrated by the dashed lines in Figure 3. These regression functions are approximately parallel, having similar slopes but distinct intercepts. The slopes of these functions did not statistically differ from each other, but both were significantly greater than the orthostatic function ($t_s[df = 32] > 4.33, p_s < .001$). Indeed, while both (within posture) regression slopes were less than 1.0, a statistical test of β revealed no significant LIV effects for these between-subjects

¹A similar pattern of results was obtained in parallel analyses on the second stress period.

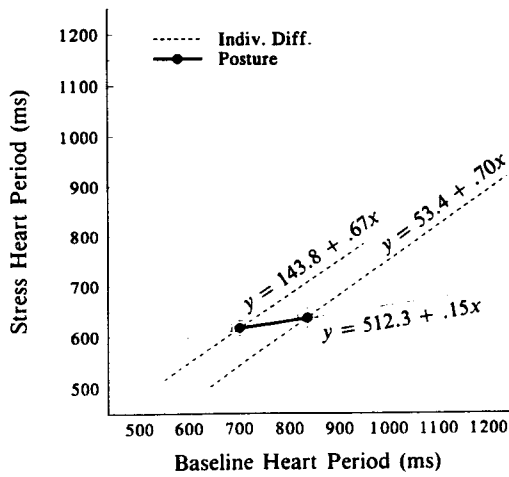


Figure 3. Comparison of between-subjects LIV manifestations of baseline variations associated with individual differences and with orthostatic manipulations. The solid line illustrates the between-subjects LIV effect of orthostatic manipulations for the first stress period. The data point on the left depicts the mean heart period data for the standing group, and the point on the right illustrates the corresponding values for the sitting group. The solid line illustrates the slope of the LIV effect for these data points. The parallel dashed lines illustrate the between-subjects regressions, within each postural group, for individual differences in basal heart period.

(within posture) analyses (see Table 1). Thus, between-subjects analyses yielded significant LIV effects when the basal differences were due to orthostatic manipulations, but not when basal variance was due to individual differences. Importantly, this was not attributable to a limited range of individual baseline differences. Indeed, the range of individual differences in basal heart period was larger than that induced by postural manipulations.

The distinction between individual differences and orthostatic variance is further indicated by the intercept differences between the two individual difference functions (which have common slopes). If baseline differences related to orthostatic manipulations were similar to those arising from individual differences, the individual difference functions of the two groups would lie along a common line. Conversely, if postural manipulations yield similar LIV effects as individual differences, the postural manipulation should have resulted merely in a translation along a common individual difference function.

The differences in slope arising from individual differences and orthostatic state did not appear to arise as a statistical artifact of heart period distribution functions. Chi-square tests revealed that none of the distributions significantly deviated from normality (all $\chi^2 < 3$, $ps > .15$).

Discussion

The present results reveal that variations in baseline heart period may have divergent effects on the magnitude of phasic response, depending on the functional origins of the basal variance. This possibility was recognized by Wilder (1967), Block and Bridger (1962), and, more recently, Jin (1992). Specifically, the present results suggest that basal differences attributable to nonautonomic factors may yield a considerably reduced LIV effect. Individual differences in nonautonomic determinants of basal heart

period are sizeable (Sutton, Cole, Gunning, Hickie, & Seldon, 1967) and stable (Jose, Stitt, & Collison, 1970). In a study of a homogeneous group ($n = 70$) of healthy young men, Sutton et al. (1967) estimated the standard deviation of intrinsic heart period is approximately 42 ms. This would account for over a third of the baseline variance observed between subjects of the present study. These (nonautonomic) individual differences in intrinsic heart period may contribute to the poorer between-subjects predictive validity of the LIV.

If individual differences in intrinsic heart period reflect merely a shift in the chronotropic level around which autonomic control operates (solid vs. dashed functions in Figure 4), they would have little effect on the magnitude of phasic response. This is illustrated in Figure 4, which models chronotropic state as a sigmoidal function of activation, as typically observed in physiological systems. An individual difference in intrinsic heart period is depicted as a vertical translation of the activation sigmoid, in which autonomic control is displaced to a higher chronotropic operating level. Although this individual difference would be associated with differences in baseline heart period (B_1 vs. B_2), these basal differences would not impact on the magnitude of response (arrows b_1 vs. b_2) to a unit increase in activation. In contrast, baseline differences related to either between or within subjects increases in basal autonomic activation (B_1 vs. B'_1) would significantly constrain further incremental responses. This is illustrated by the arrows b'_1 and b_1 in Figure 4.

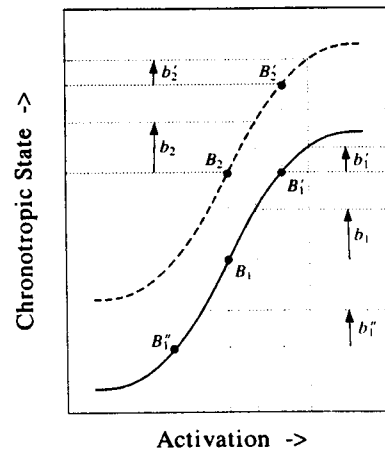


Figure 4. Alternate models of basal effects on phasic response. The solid line depicts a typical physiological sigmoidal function, relating autonomic activation to chronotropic state. Basal state is indicated by the center dot (B_1), and the vertical arrow b_1 illustrates the chronotropic response to a phasic increment of one activational unit. The dashed line depicts a similar (but vertically translated) function for another subject, representing an individual difference in intrinsic (nonautonomic) heart period. Despite the differing basal states (B_1 and B_2), a unit increment in activation would yield an equivalent response magnitude for the two subjects (arrows b_1 and b_2). In contrast, a similar basal difference (B_1 to B'_1) arising from an upward baseline shift would yield a substantial decrease in the magnitude of response to a subsequent unit activation (arrows b_1 vs. b'_1). The latter is in accord with predictions of the LIV. Finally, an equivalent decrease in baseline activation (from B_1 to B''_1) would also yield a substantial decrease in the magnitude of response to a subsequent unit activation (arrows b_1 vs. b''_1). The latter is opposite to the prediction of the LIV.

In the present study, baseline changes arising from autonomic adjustments yielded larger LIV effects than basal variance related to individual differences. The generality of the present findings, however, is not that autonomic factors invariably yield larger LIV effects. As illustrated in Figure 4, decreases in autonomic activation (B_1 to B_1'') may in fact yield smaller phasic incremental responses (b_1'' vs. b_1), as the basal locus moves progressively along the decreasing slope of the lower sigmoidal plateau. Rather, the likely generality of the present findings resides in the fact that a given basal difference may have differential LIV manifestations, depending on its functional origin. These findings underscore the need for a refined formulation of the LIV that is more closely tied to underlying mechanisms, and that appropriately distinguishes among alternate sources of basal variance.

A related contribution of the present study is the demonstration of potent between- as well as within-subjects LIV effects on phasic heart period response, when baseline differences are experimentally controlled by orthostatic manipulations. These results are consistent with a previous heart rate study where baseline levels were manipulated by alterations in autonomic tone associated with mild physical exertion (Goldwater, 1978). Many of the studies cited in Wilder's original formulation of the LIV also entailed experimental manipulations of baseline. Surprisingly, there has been relatively little recent experimental study of the LIV, the majority of studies being correlational in nature. The present results suggest that studies of the LIV may benefit from a more experimental, hypothesis driven approach. An additional advantage of an experimental approach is that tests of mean differences are not subject to many of the limita-

tions of regression methods. Of particular relevance in this experimental effort would be manipulations that yield well-documented effects on both autonomic and nonautonomic cardiac control.

We have focused here on the differential implications of autonomic and nonautonomic sources of baseline variance for the magnitude of phasic response. In fact, basal variance arising from distinct autonomic origins also may have differential impact on response magnitudes. While both autonomic branches generally contribute to baseline chronotropic state, the sympathetic and vagal divisions may contribute differentially to phasic psychophysiological responses (Berntson et al., 1991, 1994). Because each autonomic branch has its own dynamic range, activation function, and chronotropic limits, sympathetic and vagal contributions to phasic response may be subject to separate constraints. A decrease in basal heart period associated with an increase in sympathetic tone, for example, may have differential effects on subsequent decremental heart period responses mediated by sympathetic activation versus vagal withdrawal.

The LIV can account for a portion of the variance in psychophysiological studies. As a strictly empirical generalization, however, the LIV may have relatively little utility. What is clearly needed at this point is a more comprehensive model of autonomic constraints, derived not from empirical induction, but from an understanding of fundamental underlying mechanisms. Such a model could subsume the veridical aspects of the LIV and provide a more useful conception of autonomic constraints.

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