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target-organ response. Because these modes entail activation of one ANS division and inhibition of the other (antagonistic) division, the actions of both divisions tend to synergistically promote the same directional response in the target organ. Consequently, variations in the response of either division, although affecting response amplitude, would not alter the direction of the target-organ change. In contrast, nonreciprocal modes (co-activation or co-inhibition) yield a fundamentally variable directional response, or may yield no response at all if the changes in the two divisions were functionally equivalent. Thus, co-activation of the chronotropic controls of the heart could yield either tachycardia (sympathetic dominance) or bradycardia (vagal dominance). Like reciprocal patterns, uncoupled autonomic modes yield unidirectional responses because only one ANS division undergoes active change.

These differences can be summarized by the *Principle of Directional Stability*, which asserts that fundamental differences exist in the directional consistency of an organ response under alternate modes of autonomic control. This principle is apparent in Fig. 7.6, where movements along the activation continuum yield monotonic target organ responses for reciprocal modes and nonmonotonic functions for the nonreciprocal modes. In summary, although the reciprocal modes are characterized by high directional stability, the nonreciprocal modes yield a fundamentally unstable directional response of the target organ.

Dynamic Range. Autonomic modes of control also differ in the dynamic range of the target organ response. For antagonistic innervations, the reciprocal changes in activities of the two ANS divisions result in concordant effects on the target organ. Consequently, the reciprocal modes can yield the widest dynamic range of response. Reciprocal sympathetic activation, for example, entails an increase in the positive chronotropic effects of the sympathetic innervation, and a corresponding decrease in the negative chronotropic actions of the vagus. Both of these changes synergistically promote tachycardia. In contrast, nonreciprocal modes of control evidence a substantially restricted dynamic range, due to the opposing effects of the ANS divisions. In co-activation of the sympathetic and vagal controls of the heart, for example, the positive (sympathetic) and negative (vagal) chronotropic effects mutually oppose the functional impact of the other division. The uncoupled autonomic modes would display an intermediate dynamic range, since only one ANS division undergoes active change.

These properties of the autonomic modes, illustrated in Fig. 7.6, are captured by the *Principle of Dynamic Range*, which asserts that basic differences exist in the range of organ responses under alternate modes of control. These differences lie along a continuum from a wide dynamic range (reciprocal modes) to a restricted range (nonreciprocal modes).

Reactive Lability. Differences in the lability of the target organ response also characterize the modes of autonomic control. This is illustrated, for example, by the effects of a given increment of sympathetic activity under different autonomic modes. The slope of the target-organ responses would be expected to be greatest in the reciprocal pattern because the organ response to a given increment in sympathetic activity would be enhanced by the concurrent decrement in vagal control. In contrast, target organ lability would be expected to be minimal for nonreciprocal modes, since a given increment in sympathetic response would be countered by a concurrent increase in vagal activity. The uncoupled modes would again be expected to yield intermediate target-organ lability, since they are driven by a single ANS division.

These relationships are subsumed by the *Principle of Reactive Lability*, which maintains that functional differences exist in the response lability of an organ system under alternate modes of control. These differences lie along a continuum from a high lability under reciprocal modes, to a low lability with nonreciprocal modes. This principle is illustrated by differences in the slopes of the output functions of Fig. 7.6.

Adaptive Features of the Autonomic Modes

A high degree of functional coherence emerges among the formal properties of the autonomic modes of control. For dual, antagonistic ANS innervations (such as those of the heart), the nonreciprocal modes of co-activation and co-inhibition tend to stabilize the functional state of the target organ because both the dynamic range and the response amplitude lability are reduced. At the same time, the nonreciprocal modes evidence a fundamental instability in response direction, which may afford a degree of flexibility in the directional response to physiological or behavioral conditions. In contrast, the reciprocal modes of control feature high directional stability, a wide dynamic range, and a high response lability. These formal properties may be associated with distinct adaptive features. (The properties described here would be reversed in the case of synergistic actions of the ANS divisions.)

Reciprocal modes can yield large, directionally stable shifts in the functional state of the target organ, which may be optimal for well-defined adaptive adjustments to survival challenges. Thus, it is not surprising that critical baroreflex compensatory adjustments to perturbations in blood pressure evidence a reciprocal pattern (Spyer, 1988). Moreover, highly evocative survival challenges may precipitate a striking reciprocal sympathetic response (Cannon, 1929, 1939), with sufficient authority to inhibit or override even baroreflex-mediated facilitation of vagal controls (Bard, 1960; Lundin, Ricksten, & Thoren, 1984; Stephensen, Smith, & Scher, 1981). Consequently, stressors can lead to an increase in HR, in spite of an elevated blood

pressure that would normally serve to suppress sympathetic output and enhance vagal control. One consequence of reciprocal modes, therefore, is a shift among the ANS divisions in the tonic control of a target organ, which would be associated with a shift in its baseline functional state.

In contrast, nonreciprocal modes are more conservative, and tend to preserve the baseline functional state of the organ. Co-activation could thus allow an increase in both sympathetic and parasympathetic controls, in the absence of notable changes in baseline function. In fact, no change in basal state may be seen at all, if the two ANS divisions evidence functionally equivalent activation. This would tend to maintain or enhance the reactive ability of both ANS divisions, while sustaining the basal functional state of the organ. Functionally equivalent activation of the two divisions is improbable, however, and varying degrees of co-activation (or co-inhibition) are more likely the norm. Depending on the relative activation of the two divisions, the autonomic balance in the functional control of the organ could be shifted toward either the sympathetic or the vagal division.

One contribution of nonreciprocal controls may be in the integration or fine tuning of autonomic influences, which can arise from asymmetries in actions of the ANS divisions. Given the relatively greater inotropic action of the sympathetic division (Levy, 1984), co-activation of both sympathetic and vagal controls of the heart may maximize cardiac contractility while minimizing increases in rate. This could result in increased stroke volume (due to prolonged ventricular filling time) and increased cardiac output, a possibility that was confirmed in a study employing direct electrical activation of vagal and sympathetic cardiac nerves (Koizumi, Terui, Kollai, & Brooks, 1982). These authors found that vagal stimulation alone resulted in bradycardia and reduced cardiac output, while sympathetic stimulation yielded tachycardia and increased cardiac output. Simultaneous activation of both vagal and sympathetic nerves, however, produced an even greater increment in cardiac output, which was associated with an intermediate heart rate and a larger stroke volume.

Clearly, much work will be necessary to fully appreciate the functional significance of the various modes of autonomic control. It is apparent, however, that a comprehensive understanding of autonomic regulation is not afforded by the Doctrine of Reciprocity. The next section outlines a more complete model of the functional regulation of visceral activity.

Autonomic Space and its Functional Surface

Given a two-dimensional representation of autonomic space, it is possible to derive the expected functional state of an organ for any locus within this space. The surface illustrated in Fig. 7.7 represents the functional output from Equation 1, at all combinations of activity within the ANS divisions. The axes

Autonomic Space & Overlying Functional Surface

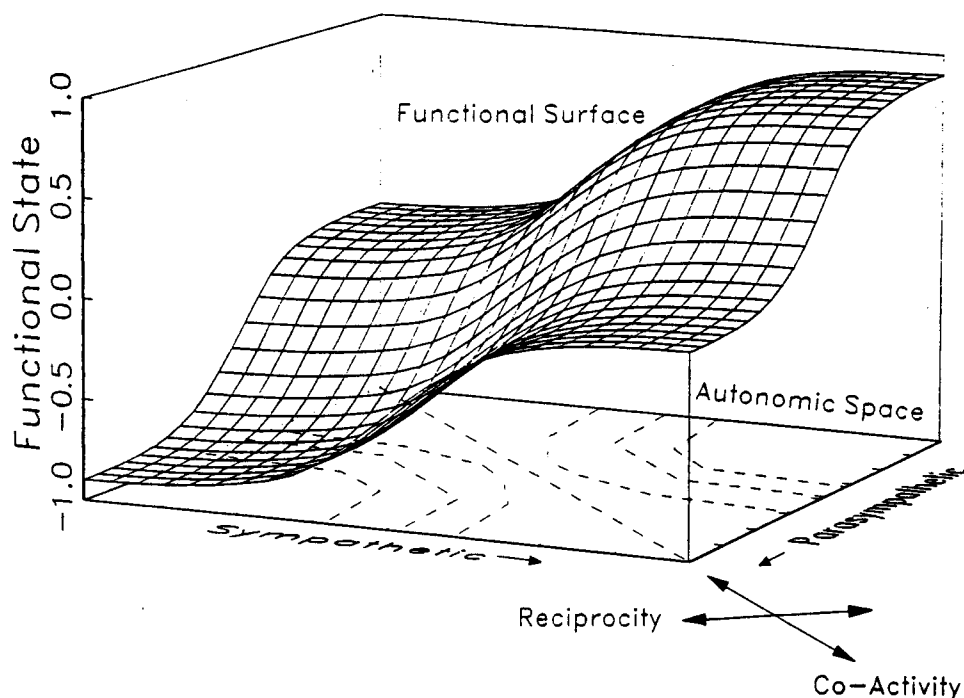


FIG. 7.7. Two-vector autonomic space and its associated functional surface. The functional surface represents the operational state of the target-organ, expressed in relative units, as derived from Equation 1. Dotted lines represent iso-functional contour lines projected on the autonomic space, illustrating points within the autonomic space that have equivalent functional outputs. (Reprinted with permission from Berntson et al., in press.)

dimensions of Fig. 7.7 are in decile units of functional activation, and the overlying surface represents the functional state of the target organ at any point in autonomic space. Again, this surface is not intended to represent a specific target organ, but rather to illustrate general features of autonomic control, which transcend specific coupling coefficients and input functions.

The autonomic space depicted in Fig. 7.7 exhaustively represents the variations in sympathetic and parasympathetic activation, and the overlying surface exhaustively depicts the functional state of the target organ at all locations within autonomic space. The neural input functions (see Fig. 7.6) are apparent at the edges of the functional surface, where one ANS division varies and the other remains constant.

Iso-Functional Contours. An important feature of the surface depicted in Fig. 7.7, is that a given functional state of the organ may be ambiguous with

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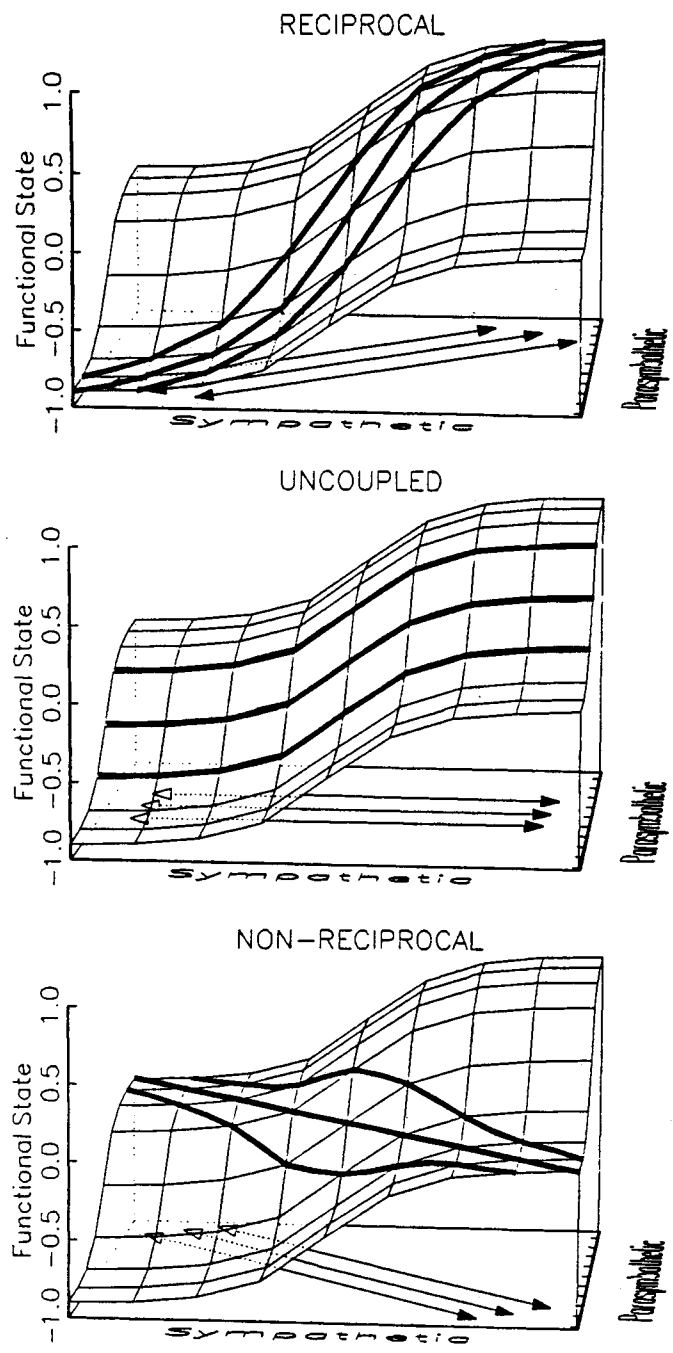
regard to its autonomic origins. This is evident in the *iso-functional contour lines* projected onto the autonomic plane of this figure. These contours illustrate the multiple loci in autonomic space (combinations of sympathetic and parasympathetic activities) that yield a common functional output state of the organ. Thus, a given psychophysiological output may arise from distinct autonomic states. As a consequence, additional information, beyond a simple measure of the functional state of the target organ, may be necessary to disambiguate autonomic origins.

Baseline States. An important feature of the functional surface of Fig. 7.7 is that the baseline state of an organ may arise from differing levels of *tonic* activity in the two ANS divisions. Thus, basal functional states represent a tonic counterpart to phasic responses, and can be characterized in terms of the autonomic modes of control. In Fig. 7.7, the diagonal extending from the back to the front represents the continuum of co-activity in tonic controls, the diagonal from the left to the right represents the continuum of reciprocity, and the sympathetic and parasympathetic axes depict uncoupled variations in tonic control.

Phasic Responses. The functional surface of Fig. 7.7 can be viewed as a static representation of tonic states of a target organ at different autonomic loci. Phasic movements within the autonomic space, however, are translated into a response trajectory across the functional surface. This is illustrated in Fig. 7.8, which maps the input functions of Fig. 7.6 onto the two-dimensional autonomic space, and depicts the translation of these inputs into corresponding response functions on the overlying functional surface. If these inputs were time-varying signals, this surface depiction would provide a representation of phasic responses in the spatial domain. Any change in the location in autonomic space would translate into a corresponding movement on the functional surface. Given the *iso-functional contours* on the overlying surface, however, this may or may not be expressed as a change in the functional state of the organ. Thus, time varying changes in the functional state of the target organ provide an ambiguous account of autonomic response, whereas movements within autonomic space are definitive.

The Laws of Autonomic Constraint

As discussed, any point on the functional surface of Fig. 7.7 could represent a baseline state in a given context. Importantly, variations in the baseline location in autonomic space set fundamental constraints on the potential phasic modes of autonomic control. The *Laws of Autonomic Constraint* capture these fundamental constraints on autonomic response.



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FIG. 7.8. Patterns of movement within autonomic space and their translation onto the functional surface. Arrows on the autonomic space depict the movement vectors associated with the input functions of Fig. 7. The translation of these autonomic vectors to functional changes in the target organ are depicted on the functional surfaces. (Reprinted with permission from Bernston et al., in press.)

The Law of Dynamic Range. Because autonomic space is bounded by the dynamic range of the ANS divisions, the extent to which each division can vary is constrained by proximity to its physiological boundaries. The effect of baseline state on psychophysiological reactivity has been long recognized (Lacey & Lacey, 1962; Wilder, 1931, 1967). The Law of Initial Values (Wilder, 1931) asserts that variations in basal state can exert directionally specific constraints on reactive changes. The Law of Initial Values is illustrated by the (reciprocal) diagonal which extends from the left to the right axes intersections of Fig. 7.7. Changes in baseline state along this diagonal serve to directionally constrain the potential reactivity of the target organ. At the far left corner of the functional surface, reactive decreases in functional state are precluded because the system is already at its dynamic limit. Conversely, at the extreme right, reactive increases in functional state are precluded by the upper limit of the dynamic range.

The Law of Initial Values is empirically based, arising from measures of the functional state of the end-organ. It can be derived, however, from the broader Law of Dynamic Range, in terms of the boundaries of the underlying autonomic space. At the extreme left of the reciprocal axis in Fig. 7.7, neither of the nonreciprocal modes (co-activation or co-inhibition) are possible. Similarly, reciprocal parasympathetic activation is precluded, as are uncoupled modes of sympathetic withdrawal and parasympathetic activation. Only three reactive modes of control remain, those being reciprocal sympathetic activation, uncoupled sympathetic activation, and uncoupled parasympathetic withdrawal. Each of these modes yields an increase in the functional state of the organ. Conversely, at the right extent of the reciprocal diagonal, only uncoupled sympathetic withdrawal, uncoupled parasympathetic activation, or a reciprocal parasympathetic mode could be manifest. Each of these modes yields a decrease in the functional state of the organ. This is the essence of the Law of Initial Values, but here is expressed in terms of autonomic space, rather than the functional surface (i.e., target-organ baseline). The underlying neural mechanism for the Law of Initial Values is thus informed by the Law of Dynamic Range.

As is apparent in Fig. 7.7, autonomic locations along the reciprocal diagonal are associated with different functional states of the organ. Consequently, autonomic constraints along the reciprocal diagonal could be couched either in terms of the location within autonomic space or of the basal functional state (i.e., the Law of Initial Values). A fundamental difference in these two approaches relates to the fact that a point in autonomic space is unique, being defined by two cartesian coordinates. In contrast, the functional state of the organ (baseline) does not specify a unique location on the functional surface, given the existence of iso-functional contours. Thus, for example, the baseline functional state may be invariant along the co-activity diagonal (see Fig. 7.7). At the same time, different locations along this diagonal are associated with widely differing constraints on reactive change of the two ANS divisions. At

the extreme front of this diagonal, neither reciprocal modes, uncoupled activation modes, nor co-activation are possible. In contrast, at the far end of this continuum, neither reciprocal modes, uncoupled withdrawal modes, nor co-inhibition may be evidenced. The Law of Initial Values is blind to varying constraints on reactivity which lie along this, or any other iso-functional contour, since the functional state of the organ does not change. Consequently, the broader set of autonomic constraints can not be derived from baseline measures. In short, the Law of Initial Values can be subsumed as a special case of the more general Law of Dynamic Range.

The Law of Reactive Lability. Apart from the limits of dynamic range, additional constraints on autonomic lability are imposed by the basal location in autonomic space. Movements in autonomic space along the reciprocal diagonal are associated with notable changes in the functional state of the target organ, while similar movements along the co-activity diagonal (or any other iso-functional contour) are not associated with changes in organ state. Consequently, variations in target organ lability, to a directional displacement in autonomic space, are apparent even at loci remote from dynamic range boundaries. The Law of Reactive Lability is illustrated in Fig. 7.9, which shows the variations in the lability of a target organ at different points, and with different directions of movement, within autonomic space. The surfaces in this figure depict the directional magnitudes of the gradient of the functional surface of Fig. 7.7, which corresponds to the instantaneous rate of target organ change with specific directional movements through autonomic space (modes of control). As is apparent, minimal lability is seen at the four corners for all modes of control, since autonomic influences plateau at those points. The modes differ considerably, however, in the level of reactivity at other points. Thus, reciprocal modes evidence a peak reactive lability in the center of autonomic space, whereas nonreciprocal modes show minimal lability at this point. The Law of Reactive Lability maintains that a given displacement in autonomic space can yield widely differing changes in the target organ response. As illustrated in Fig. 7.9, these differences are related not only to the starting location within autonomic space, but the specific direction of movement (mode of control).

The Law of Directional Stability. A final set of constraints on phasic responses is related to variations in the directional coherence of target organ change with movements through autonomic space. As illustrated in Fig. 7.8, movements along any vector parallel to the reciprocal diagonal yield similar directional responses in the target organ (although they may vary in amplitude). In contrast, movements along the co-activity diagonal yield no organ response, whereas movements along parallel vectors may yield target responses of opposite direction. Moreover, the direction of the target organ

FIG. 7.9 (Tum)

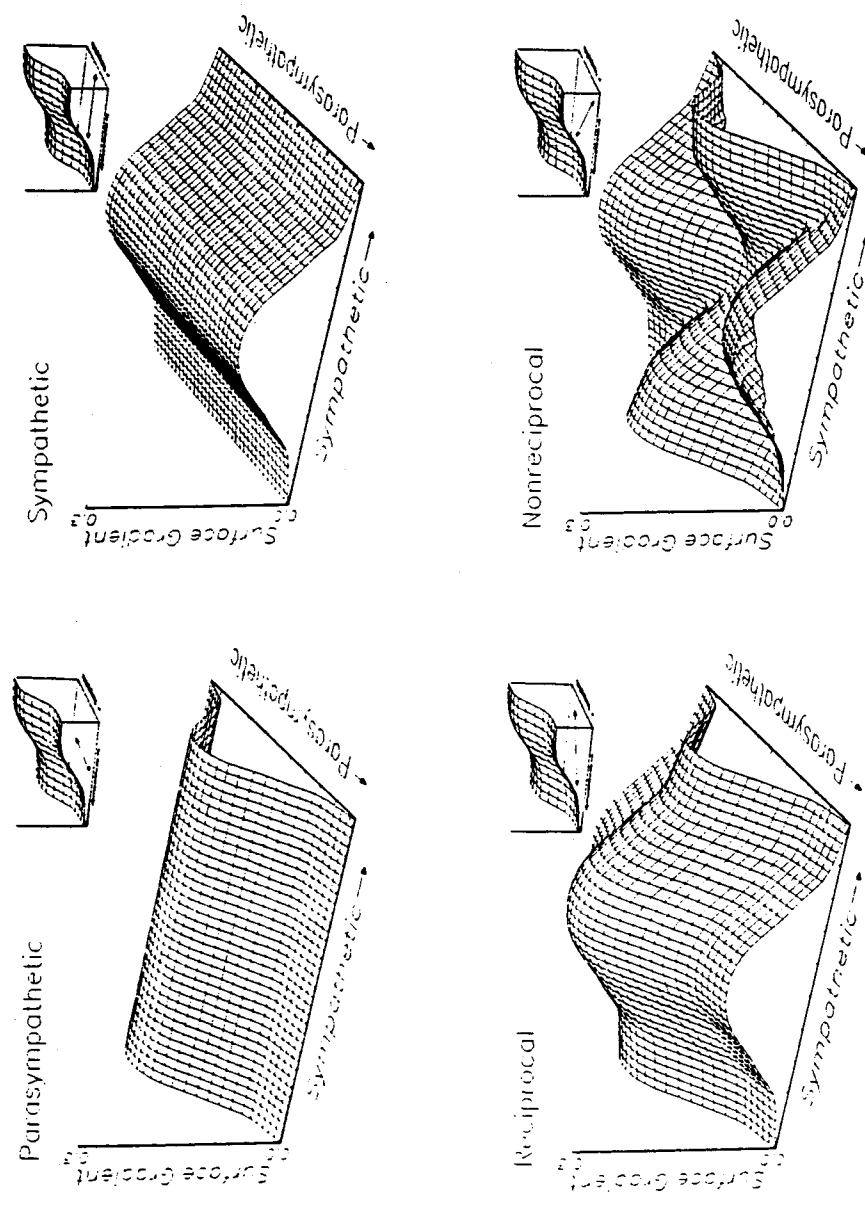


FIG. 7.9. Surface plots of reactive lability as a function of the direction of movement (mode of control) within autonomic space. Small insets show the functional surface of Fig. 7.7, and the arrows on the autonomic plane depict the direction of movement. The larger plots depict the gradients (sum of the partial derivatives) of the functional surface across autonomic space. Variations in the surface amplitude in these figures illustrate the instantaneous changes in organ state associated with the indicated movement from any point in autonomic space. (Reprinted with permission from Bertson et al., in press.)

response is not uniform even for movements along a given vector parallel to the co-activity diagonal. Rather, the target-organ response changes sign as the vector crosses the reciprocal diagonal (see Fig. 7.8).

The Law of Directional Stability asserts that differing modes of autonomic control are associated with variations in the directional coherence of target organ responses. This law cautions against a rigid interpretation of a specific directional response in a visceral organ, in the absence of knowledge of the origin and trajectory of the movement in autonomic space.

Summary of the Laws of Autonomic Constraint. The Laws of Autonomic Constraint impose fundamental restrictions on visceral responses. In some cases, these constraints become intuitively apparent, given an adequate understanding of the structure of autonomic space. Although conceptually straightforward, The Law of Dynamic Range reveals the basis for a perplexing, 60-year-old observation on the relationship between basal state and reactivity (Wilder, 1931). In other instances, constraints are far from obvious. The Law of Directional Stability, for example, asserts that a specific directional movement within autonomic space can yield diametrically opposite organ responses, depending on the locus from which the movement begins. These nuances of autonomic control can contribute substantial variance to psychophysiological measures. If understood, this variance may translate into important experimental effects. If not, it simply appears as error variance.

Applications to the Autonomic Control of the Heart

The functional surface of Fig. 7.7 represents a generalized model of the translation from autonomic space into target-organ state. Variations in the terms of Equation 1, from organ to organ, may alter the topographic features of the functional surface. For the chronotropic control of the heart, the coefficients of coupling for the two ANS divisions are different (Berger et al., 1989; Levy & Zieske, 1969). Moreover, the coefficient of the interaction term is not zero because sympathetic influences are progressively inhibited at higher levels of vagal activity (Levy, 1984; Levy & Zieske, 1969). These features of chronotropic control yield a functional surface that would be canted downward at the front of the co-activity diagonal of Fig. 7.7. Importantly, however, these variations: (a) do not alter the relative features of the autonomic modes, (b) do not eliminate iso-functional contours, and (c) do not embarrass the Laws of Autonomic Constraint (Berntson et al., in press).

IMPLICATIONS FOR THE AUTONOMIC OR

Autonomic responses offer considerable advantages for studies of orienting and attention. The literature reveals a notable consistency in the cardiac

features of the orienting response through both ontogeny and phylogeny. This general consistency, however, belies the complexity of the origins and meaningful interpretation of autonomic responses. The two divisions of the ANS, for example, may exert differential organ-specific contributions to autonomic ORs at different target sites. The autonomic components of the OR include (a) cardiac deceleration, arising in part from activation of the vagal control of the heart; (b) pupillo-dilation, attributable in part to withdrawal of parasympathetic control of the pupillary muscles; and (c) electrodermal responses associated with sympathetic activation of eccrine sweat glands (Beatty, 1986; Lynn, 1966; Siddle et al., 1983; van der Molen, Boosma, Jennings, & Nieuwboer, 1989). Further complexities arise from the multiple modes of autonomic control within a given target organ.

The deceleratory cardiac OR has generally been considered to arise from an increase in vagal influences on the heart. From the vantage of a reciprocal model of autonomic control, this may constitute an adequate characterization of its autonomic origins. It is not adequate, however, under nonreciprocal modes of control, which may be operative in the orienting context. A two-dimensional model of autonomic space provides a more comprehensive account of autonomic control and consequence than does a single-vector model, and offers a powerful quantitative approach to investigations of psychophysiological relationships. Due to the many-to-one mapping between loci within autonomic space and functional state (Cacioppo & Tassinari, 1990), as reflected in the iso-functional contour lines of Fig. 7.7, the response of the target organ per se may be less closely linked to cognitive, emotional, or behavioral processes than the specific patterns of movements within autonomic space. Although potentially yielding comparable bradycardia, a vagally dominant autonomic co-activation may have functional origins, adaptive consequences, and response features that are quite distinct from those of a reciprocal vagal pattern.

The two-dimensional model of autonomic space also raises an important issue as to the fundamental nature of psychophysiological adjustments in orienting and other behavioral contexts. At least three possibilities arise. In some instances, psychophysiological responses may be best characterized by a directional vector through autonomic space. These *vector-specific* (or *mode-specific*) adjustments may be associated with a wide variance in the functional response of the organ, depending on the basal starting point in autonomic space (see Fig. 7.8). Vector-specific responses may show commonalities only in the direction and magnitude of the movement vectors through autonomic space (i.e., in the autonomic mode of control). Alternatively, the specific directional vector may be less germane than the ultimate end-point of movement within autonomic space. For these *locus-targeted* adjustments, both the optimal mode of control, as well as the related functional changes in the organ, may vary depending on the starting point within autonomic space.

The common feature of these responses would be the end locus of the autonomic trajectory. Finally, a psychophysiological response may have as an end-point a specific functional state of the organ, or a specific change in that state. In these cases, the autonomic mode or response vector could vary with the autonomic starting point, and the autonomic end-point could vary along any iso-functional contour line. It is only for these *output-targeted* responses, that the common feature could be discerned from the magnitude or functional end-point of a visceral response. For present considerations, it is of relevance that output-targeted adjustments are probably the least represented in the autonomic control of the viscera because they necessitate an explicit monitoring of the functional state of the organ. Although this may be the case for blood pressure (via baroreceptors), there is no comparable neural mechanism for directly monitoring the chronotropic state of the heart (or most other visceral organs). Consequently, it becomes increasingly important to specify psychophysiological relationships within the dimensions of autonomic space.

In view of these considerations, the availability of specific indexes of sympathetic and vagal control would permit a detailed probing of autonomic space, which could improve the resolution of autonomic measures and enhance their applicability to behavioral studies. The cardiac OR to a non-signal novel stimulus may evidence the same general form as that to an adaptively meaningful stimulus. The evoked behavioral dispositions, however, although perhaps sharing common attentional features, may differ among these classes of stimuli. Given the close integration of autonomic, somatic, and cognitive reactions, it is also likely that differences may exist in the modes of ANS control associated with differing action components.

Clearly, independent measures of the relative activities of the two ANS divisions are desirable to permit specification of the autonomic state, or its reactive change, in terms of autonomic space. Unfortunately, quantification of the specific activities of the ANS divisions can be problematic. While pharmacological blockade represents one approach to documentation, it is not without limitations, and may not always be pragmatically feasible (Berntson et al., 1990). Alternate possibilities include noninvasive indices of sympathetic or vagal action derived indirectly from measures of the functional state of the organ. Given the highly specific patterns of autonomic activity that can be seen across organs, measures of the two ANS divisions ideally should be derived from the same target organ. Moreover, since even chronotropic and inotropic influences on the heart are mediated by separate efferent pathways (Billman et al., 1989; Randall & Ardell, 1985), indices would optimally be derived from the same functional dimension of the target organ.

A number of indices of sympathetic and vagal controls of the heart have been proposed (T-wave amplitude, carotid dp/dt, heart period variance,

systolic and diastolic time intervals, respiratory sinus arrhythmia). Many of these measures, however, show only a relative specificity among the ANS divisions, or derive sympathetic and vagal activities from manifestations in different functional dimensions. Alternative measures are desirable in which the relative sympathetic and parasympathetic influences on a target organ can be derived from the specific functional dimension of interest, such as chronotropism. Developments in the analysis of frequency components of heart period variance hold promise in this regard (Porges, 1986; Porges & Bohrer, 1990; Porges et al., 1982). Although these measures are also not without limitations (Grossman, Karemaker, & Wieling, in press; Quigley, Cacioppo, & Berntson, 1990), they may provide frequency-specific metrics of activities in both vagal (Akselrod et al., 1981; Grossman, Stemmler, & Meinhardt, in press; Katona & Jih, 1975; Porges, 1986; Porges & Bohrer, 1990; Porges et al., 1982) and sympathetic divisions (Akselrod et al., 1981; Pagani et al., 1986; Saul, Rea, Eckberg, Berga, & Cohen, 1990; Shin, Tapp, Reisman, & Natelson, 1989).

A recognition of the multiple determinants of autonomic state and the multiple modes of autonomic control underlying HR responses would not only increase the power and specificity of cardiac measures, but may resolve apparent discrepancies in the literature. These include the apparent paradoxes posed by the Amsel group and the Campbell group (see Exceptions to Autonomic Parallelism), to which we now return.

In interpreting HR change in behavioral contexts, it is imperative to consider the influence of basal location in autonomic space and the distinction between phasic response and tonic baseline change. Dailey, Wigal, and Amsel (1986) reported that the onset of a bright light yielded cardiac deceleration in infant and juvenile rats (5–21 days). This bradycardia was subject to habituation over trials, consistent with the elicitation of an OR. The offset of a bright light, however, which would also be expected to evoke an orienting response, consistently produced tachycardia in rats at all ages tested. Further, this tachycardia was not subject to habituation over 60 trials. These observations appear at variance with the elicitation of the OR, and are more consistent with the evocation of a DR. The paradox arises from the fact that an OR, not a DR should be triggered by a decrease in stimulus energy. The probable resolution of this paradox lies in the basal location and tonic adjustments within autonomic space.

In the Dailey et al. study the animals tested with light offset were, by necessity, in an illuminated condition during baseline periods (30-sec light-offset periods, 90-sec variable light-onset ITI), whereas those tested with light-onset were in the dark during the baseline condition. Effects of ambient illumination on rats are well known—rats are nocturnal and relatively inactive in the light, and would be expected to demonstrate a lower basal heart rate under illumination. Unfortunately, Dailey et al. express HR responses

during the 30-sec trials only as a change from baseline, so that prestimulus baseline heart rate can not be determined. The matched control groups (constant illumination or constant darkness), however, show consistent and dramatic differences in basal HR, with animals in the light displaying rates about 20–50 bpm (depending on age) lower than those in the dark. Because the control groups were otherwise matched, light offset in the illuminated control group would be expected to yield an increase in baseline HR of about 20–50 bpm. This baseline adjustment would entail a tonic shift in basal location within autonomic space, rather than the phasic evocation of a DR. The cardiac acceleration to light-offset observed by Dailey et al. likely reflected such a baseline shift. This is consistent with the fact that HR generally increased progressively throughout the 30-sec light-offset periods. Although a phasic deceleratory OR might be expected immediately after light offset, this could not be evaluated because the Dailey et al. data were collapsed across 5-sec bins, which may have obscured a transient response due to the tonic acceleratory shift in baseline. The presence of a transient deceleratory OR was suggested, however, by the fact that the acceleratory changes to light offset were negligible or minimal in the first 5-sec block. In contrast to the light-offset condition, the expected tonic deceleratory shift in heart rate during light-onset would not obscure but would tend to amplify the phasic deceleratory OR. Consequently, the OR was apparent in the latter condition.

The importance of considering autonomic origins and the modes of autonomic control is also illustrated by the study of Richardson et al. (1988). These authors report that the transfer of rats to an unfamiliar testing environment inhibited the HR component of the orienting response (OR) to a pulsating tone. Sayers et al. (1990) replicated these observations in preweaning rats, and also observed similar inhibitory effects of a prior shock. Because the stimulus change was identical across contexts, these authors emphasize the incongruity of their experimental findings with past and current conceptions of the orienting response. Before this apparent incongruity can be accepted, however, alternate explanations must be considered in the context of autonomic space.

Sayers et al.'s (1990) conclusion that new or stressful environments could inhibit the OR was based largely on their observation of attenuation of the heart rate deceleration to a novel tone. As we have noted, HR is influenced by both sympathetic and parasympathetic inputs, and these influences are antagonistic. If one assumes a reciprocal mode of ANS control throughout, then the inference by Sayers et al. is appropriate. Based on the pharmacological study of Quigley and Berntson (1990) as described above, however, it appears that autonomic co-activation may be manifest in orienting contexts. Moreover, novel or challenging environments—environments in which optimal or adaptive behavioral responses are unclear—may be more likely to

evoke and/or promote coactivation of both the sympathetic and parasympathetic divisions than are familiar environments. Hence, the attenuated cardiac orienting responses reported by Richardson et al. (1988) and Sayers et al. (1990) could reflect greater *conjoint* parasympathetic and sympathetic activation (i.e., co-activation), rather than attenuation of the vagal response associated with the OR (Quigley & Berntson, 1990). Examination of the second \times second HR changes in the Sayers et al. study (their Fig. 6) support this interpretation. The sympathetic system is known to have a longer latency than vagal influences on the heart. Although vagal influences on heart rate can be apparent within the first beat of a triggering event, sympathetic manifestations are typically delayed by 2-3 sec (Karemaker, 1985; Warner & Russell, 1969). In the Sayers et al. study, the experimental manipulations (shock or context change) did not alter the heart rate response during the first second after the stimulus, rather experimental curves progressively diverged from the control condition over the subsequent 2 to 3 sec. This is consistent with a concurrent (longer latency) sympathetic activation, which would obscure the vagal response. Autonomic co-activation is also consistent with the absence of baseline increases in heart rate in the Sayers et al. study, even after repeated shocks. If research bears out this interpretation, then measures of the orienting response derived from organs that are not dually and antagonistically innervated (e.g., eccrine glands in humans) may be valuable under these conditions. Alternatively, independent estimates of sympathetic and vagal control of the heart could be used to address this issue.

Although this discussion has not resolved the autonomic origins of the OR, it details the obstacles to this resolution imposed by the Doctrine of Autonomic Reciprocity. Moreover, we have outlined a more comprehensive conceptual framework for strategic investigations of this problem. Substantive advances in psychophysiology will undoubtedly be facilitated by an increased recognition of the biological origins and control of psychophysiological responses. The details of autonomic space, however, may be indeterminate from common psychophysiological measures of dually innervated organs. Consequently, substantive advances in the psychophysiology of these organ systems may require the deployment of independent measures of sympathetic and parasympathetic actions. Such measures will allow an unambiguous specification of autonomic response, which will likely enhance the resolution of autonomic reactions, and contribute to meaningful investigations of behavioral-psychophysiological relationships.

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