ACCUMULATED STRESS, RESERVE CAPACITY, AND DISEASE

CONTENTS

INTRODUCTION

The underlying the accumulation of stress affects the reserve capacity of an organism, both in the maintenance of its functional integrity and in the resolution of subsequent exposures to stress. Stress is defined in terms of a reaction resulting from stimuli which sufficiently activate the autonomic nervous system (ANS) and is either resolved or accumulated depending on whether the pre-stimulus baseline is re-established or not.

Accumulated stress profoundly influences the totality of organismic functioning, and is expressed essentially through three bi-polar effector systems: In the realm of the autonomic, the effector system is the sympathetic and parasympathetic visceral outflow. For the somatic, it is paired movers, like extensor/flexors; and metabolically stress is expressed (though less distinctly) by, for example, catabolic/anabolic and inflammatory/anti-inflammatory endocrine reactions. The response to stress is defined as occurring sequentially in tow phases, chare and discharge: When the charging (sympathetic) phase is followed by parasympathetic discharge of equal magnitude, then pre-activation homeostasis is reestablished and the stress is said to be resolved. On the other hand, it is shown that under certain physiologic conditions) and behaviorally where mobilization – i.e., somatic response to stress—is blocked), the charge phase is no longer balanced by rebound. In these cases activation is not resolved and the stress becomes incorporated within the organism, as a diminished adaptational capacity.

In this regard, various holistic systems of healing are seen to focus their efforts towards detecting and treating these accumulation imbalances and reduced capacities even before they become symptomatic and pathologic. It is the view of this work that a wide range of “stress diseases” with varied symptoms and obscure aetiologies are the final—pathologic—expression of this loss in resiliency.

That the accumulation of stress is the underlying stratum in certain disease syndromes is tested by measuring autonomic levels underlying certain blood pressure responses of a hospitalized population. It is not possible, however, to measure the sympathetic and parasympathetic components directly (since they are expressed as a single output vector, blood pressure). For this reason a systems analysis of the cardiovascular system, based on well-known experimental parameters, but with variable set point and gain levels, is constructed. A set of blood pressure response cures is generated and compared with the hospitalized population. The fit of these with the experimental data is surprisingly good. In addition, the prognosis for five groups in the hospitalized population is predicted accurately by the model, whereas no such predictions could be made on the basis of the raw data.

The accumulation of stress, defined in terms of the autonomic nervous systems. The concept of an autonomic hypothalamic “hub” around which behavior is organized and executed is illustrated to clarify some of these extended relationships. Specifically, the hypothalamic links between autonomic-endocrine, as well as somatic mobilizing systems, are examined in the context. In

addition, examples illustrating the potential for the wide and varied symptomatologies of their "mis-integration" (autonomic-endocrine-somatic) in the stress diseases are presented. Some possibilities for pre-symptomatic diagnosis, whereby stress accumulation is detected before the development of debilitating symptoms and tissue pathologies, are investigated as well. These stress diseases are shown, in a selected set of examples, to have underlying patterns of unresolved stress that can be understood in terms of their topologic configurations in catastrophe space.

ACCUMULATED STRESS

The term "stress," despite its universal appearance in the nomenclature of biology and medicine, has been and is used without precise or even consistent definition. This unusual state of affairs must be due to a need in these sciences to describe significant groups of phenomena which simply are not covered adequately by other generic terms or concepts. In Mason's (1976) words: "The controversy over the definition of the term 'stress' does not bear upon the validity of the underlying scientific observations or concepts."

One of the areas where stress has variously been considered is in its relation to disease. The accumulation of "stresses and strains" has in many instances been indicated as a contributory or even primary factor. Diseases such as hypertension, ulcers, asthma, heart conditions, and even various neoplastic growths and certain types of diabetes are widely recognized as having "constitutional" and "emotional" stress components. More and more, these factors have been acknowledged by members of the m medical profession and sciences. Yet there have really been few, if any systematic means to separate and study these stress factors and their cumulative effects.

The use of the concept of homeostasis in the analysis of stress can be useful in eliminating some of the vagueness from the term, and in suggesting a working definition. The mobilizing energy in the anticipation of extreme muscular exertion needed for the "life or death" struggle in these emergency situations. It is of no use for the animal to maintain an internal consistency if it is eaten in the process. On the other hand, survival in the face of emergency, if the organism is unable to return to the previous non-emergency equilibrium, diminishes the capacity for internal regulation.

The basic idea to be built upon here is that activation of emergency response and the functions of efficient cellular activity are often, if not basically, incompatible. Further, they are timed and balanced dynamically to the service of organismic survival, the acute adaptive response of sympatheticoadrenomendullary system having temporarily a higher priority than the ongoing activities of cellular homeostasis.

In studying factors controlling the adrenal medulla, the control of this gland was carried out by the Autonomic Nervous System (ANS). It was also being discovered that regulation of such automatic control functions as blood pressure, temperature, ventilation, osmolarity, and energy balance were also in the province of the ANS. Since the activity of the adrenal medulla is regulated by the

sympathetic branch of the ANS, this meant that the same division of the ANS participated both in an array of minute, continuous internal adjustments as well as in preparing the organism for flight and fight reactions. Only during these extreme conditions, he reasoned, did the sympathetic division "takeover" and temporarily suppress the normal delicate regulation of the internal milieu and restitution of cellular function (which he felt was served by finely graded reciprocal shifts between autonomic states, i.e., both sympathetic and parasympathetic).

The "shades of grey" wherein the organism may not be able either to fully "mobilize" towards meeting emergency conditions or to make completely the transition back from emergency to "normal" situations with their much smaller and more precise requirements, were not derived in Cannon's era. It is the classification and understanding of these phenomena that is a primary concern of this paper.

This anticipation, which occurs both in humans and in wild animals, is of obvious natural survival value. In humans and laboratory animals, however, the usual mobilization which follows in the wild is suppressed or absent. Only the perception of this reaction, which, in humans, is probably fear, is present as an acute state. Chronic anxiety[1] can have profound autonomic and hormonal influences—a fact fully agreed upon by most clinicians and researchers in the field of psychosomatic medicine.

Thus it will be of great importance in the study of the various stress syndromes, to understand the potential mechanisms for "accumulation of stress," i.e., for the transition from an acute response towards a chronic limitation in overall organismic function and efficiency.

The meaning and scope of "stress" and its relation to health and disease, as it will be used in this dissertation. The facet of stress to be dealt with here is its effect on the autonomic nervous system (Autonomic Stress), the mechanisms by which it accumulates and its relation to an organism's potential or reserve capacity to meet further stress; as well as the eventual pathological breakdown and manifestation of the various symptoms of the so-called "stress diseases" as this capacity becomes sufficiently diminished. This is not to imply an absolute threshold relationship between the accumulation of AS, the eventual breakdown in disease and the manifestation of symptom pathologies. It does imply the existence of lawful processes in the transition between health and disease, which can be understood with a degree of quantitative rigor.

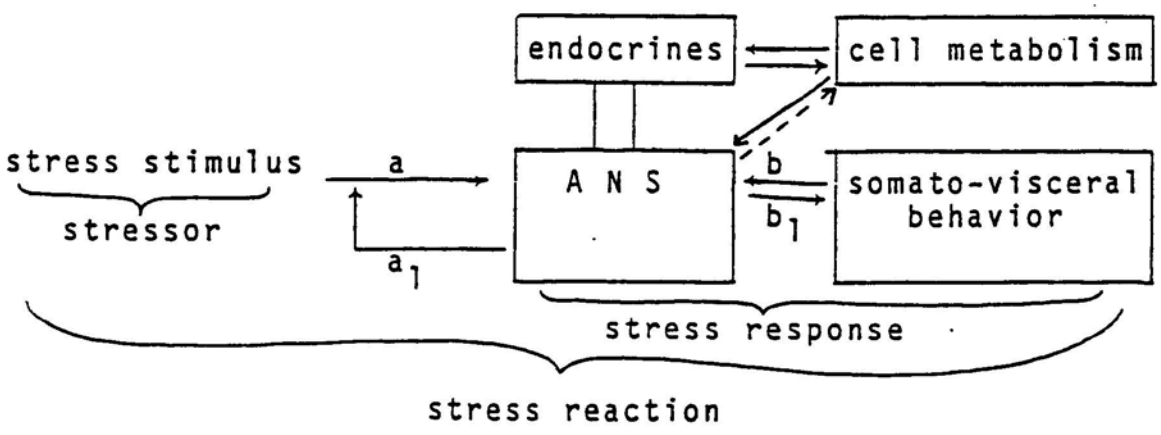
The next step is to formulate and define the phenomenological and neurological mechanisms by which AS accumulates over time, and how that leads progressively to limitation in an organism's capacity to respond appropriately to further stress (dis-ease) and then finally to the appearance of the "stress disease."

Stress is defined as a process whereby a stimulus elicits activation of the autonomic nervous system (ANS) to such a degree that return to the homeostatic balance can be interfered with.

Stress is then further defined in terms of a dichotomy which divides it into two forms: resolved and unresolved or accumulated. In a particular situation it is both the nature of the stressful stimulus and the present "capacity" of the organism to "respond" to this stress. This will determine whether

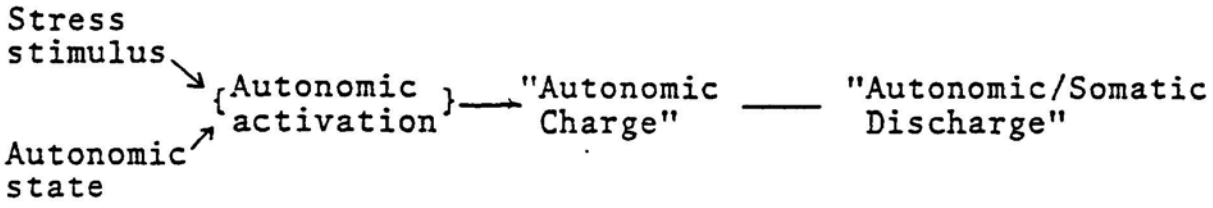
the situation is resolved or whether it becomes "internalized" within the organism as a decreased capacity to resolve future stress.

It is proposed, in other words, that stress be defined in terms of a pattern of autonomic reactions which are not necessarily reversed. When initial conditions are reestablished, the stress is said to be resolved. On the other hand, when the autonomic stress response is evoked but doe not return to its initial state, it is defined as accumulated, and consequently, the autonomic response characteristic to subsequent arousal is fundamentally altered.

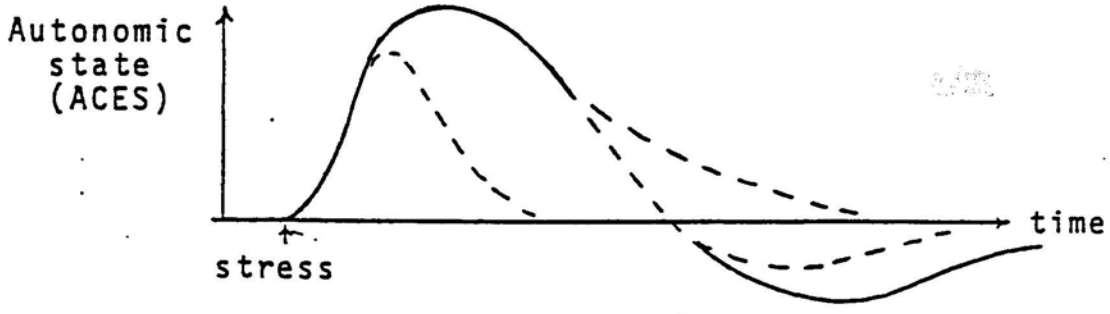


In addition to the involvement of the ANS in the reaction the figure illustrates that not only somato-visceral behavior but endocrine responses participate as well. The two-way arrows allow for more generality. In addition, the two way flow a, a_1 illustrates that the state of the ANS, as well as the nature and magnitude of the stimulus influence one another.

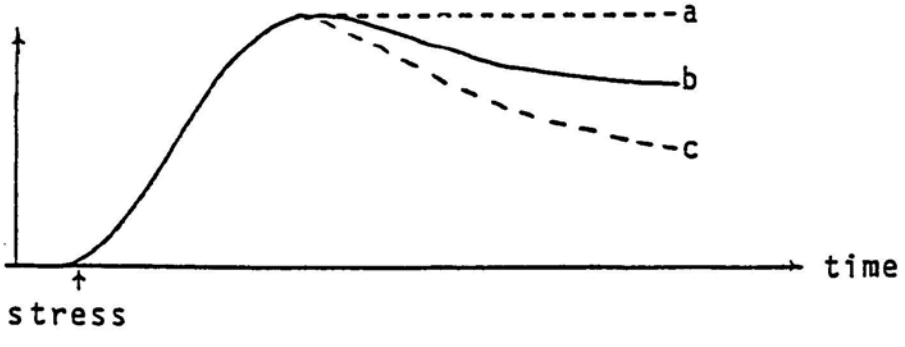
The cycle by which stress is resolved is then defined as follows:



And is portrayed in the following diagrams; for resolved stress, where ACES is taken as a construct of the level of activity in the central autonomic system:



And below for a stress response which is not resolved, i.e., is accumulated:



Curve a represents a totally unresolved stress residual, while b and c are partially accumulated.

In summary, then, for a stress to be resolved the shift of autonomic activity evoked by the stimulus must be restored to the pre-stimulus value. If the level does not return to that baseline, the stress reaction is said to be unresolved and a residual stress accumulates, modifying the baseline of autonomic activity.

The mechanisms by which stress is accumulated are central to the development of this paper, and are intimately related to the fact that autonomic activity is expressed, at the effector level, by the interplay of two component branches, the sympathetic and parasympathetic division.[7]

For reasons which will become clear as the theme of this dissertation is further developed, the autonomic stress response is divided into two primary components, charge and discharge, as shown below:

While stress has been defined in terms of autonomic activities, care should be taken not to think of the autonomic system as a functionally distinct efferent channel isolated from the central nervous or peripheral somatic systems.

As early as 1925 Hess distinguished between 'ergotropic' (E) and 'trophotropic' (T) reactions. The former consisted of sympathetic discharges which were always combined with heightened activity of the somatic muscular system and cortical arousal, while the latter involved parasympathetic discharges and inhibition of somatic and central functions. Indeed, the major function of the autonomic charging was, as Cannon first realized, a preparation and mobilization towards flight or fight. This depended upon the capacity for intense and highly organized motor behavior. As this behavioral response was terminated, a return to the pre-stress autonomic baseline would serve again the ongoing homeostasis.

THE TREATMENT OF ACCUMULATED STRESS

The theme was developed that, at least in one particular population, the accumulation of stress gives rise to a spectrum of diverse symptomatologies, the prognosis of which can be ascertained, not on the basis of the symptoms, but by analyzing underlying autonomic response levels and re-presenting them topologically in catastrophe space.

The treatment of "stress disease" is notoriously unsuccessful and unpredictable. One would hope that the theory derived, would be of use in this crucial area by suggesting some different approaches.

Gellhorn (1957), in the epilogue of his book on the hypothalamus, talking about the psycho-physical relation in stress disease, states that "Such disorders (stress disease) require a therapy through which, by physiologic or pharmacological means, brain functions are altered and ultimately restored." He goes on to add that "The so-called shock therapies fulfill this postulate, at least to a certain degree, . . and are appropriate in that they increase central sympathetic activity for a period of time." He tempers this by saying (of shock treatment) that "These procedures are distinguished in principle from an ideal form of therapy (even if the curative effect were far greater than it is) in that convulsions and coma effect the whole brain and are not specifically directed toward those

structural functions which—at least in the theory presented in this monograph—are responsible for the therapeutic effects." (i.e., the hypothalamus)

On the basis of the data presented in his book, Gellhorn proposes that "a physiologic (pharmacologic) therapy. . . can be envisaged. . .that should be directed toward a restitution of autonomic balance."

Gellhorn makes here a tacit assumption which needs to be examined: The parallelism he assumes between the clinical recovery and the Funkenstein test suggests that "autonomic hypothalamic disturbances are not a byproduct but cause the behavior disturbance." But it does not follow, as he implies, that organismic function will be, of necessity, restored by adding excitation to, or sedating, hypothalamic autonomic centers. What must be realized is that the autonomic balance has the function of maintaining homeostasis and the preparedness for emergency through the charging and discharging of the ergotropic and trophotropic systems in appropriate patterns of activity (i.e., changes in motility). Autonomic balance is only a static phase of this response when considered in isolation.

In general, strong stimulation to an already stressed organism will evoke some shift—the symptoms exhibited by this organism would be different—but function could certainly not be claimed to have been restored. The point is that in order to resolve accumulated stress one must restore the function of completing the cycle of activation charge discharge, by appropriate maneuvers. Further, that while no claim can be made that the catastrophe cusp formulation is the appropriate representation, it unquestionably does point to the need for a more integrated view of the relation of accumulated stress biological and social behaviors and in the treatment of the dis-ease process. And that merely stimulating, ergotropically, or attempting to sedate (trophotropic) may well change the behavior of the organism, but that this will restore basic function and a return to homeostasis is not likely. (It may even diminish it.)

To initiate this restorative change in function, again, requires appropriate perturbations of precise magnitude and timing so as to initiate the process of charging to such a degree that it results in discharge with its somatic expression.[32] In this light the use of "shock therapies" may well, in the balance, prove more harmful than beneficial, and should be considered more of a last resort and commentary on our lack of understanding of the substrata of stress than as appropriate treatment. .

A truly integrative approach, then, has to understand the "laws" governing the complex behavior of many systems as well as their interactions. One can, from the utter complexity of this, appreciate the desire to isolate a single localized causative factor in the stress diseases (otherwise it would seem a hopeless and entangled maze), as well as to better appreciate and give more tolerance to certain "unconventional systems" with seemingly less rigorous theoretical basis and practical methodologies. As Gellhorn (1957) remarks, "Man can solve his important problems through action long before he can understand the underlying mechanisms." In any case the need for a basically different approach to the problem does seem necessary. .

The results suggest that the susceptibility to stress disease may be caused, not by a reaction to a specific external or internal agent, but primarily to a reduction in the capacity of the organism to

shift between states of emergency and ongoing internal homeostasis. And the transition from states of health (ease) to those of stress dis-ease are viewed as a gradual diminution in this capacity. .

The theory developed here will be used to outline and orient various approaches which, it is proposed, attempt to prevent and reverse the buildup of stress with its associated loss in reserve capacity and eventual pathosymptomatologies.

STRESS DISEASE

Compendium of Stress Diseases

i. Nomenclature

While almost no one would argue basically against a multifactoral view of disease (incorporating stress, infectious agents, and other epidemiologic considerations); and since, as Selye (1976) points out, “increased corticoid production has been demonstrated in “virtually every pathologic condition of any importance, a certain relationship between stress and the most dissimilar diseases has been suspected.”

Which diseases, though, are primarily stress-induced is not as clear, and if stress is a predominant factor in so many diseases, why they often exhibit such a wide range of varied symptoms and pathogenic sites is by no means apparent.

Reasons for this are undoubtedly extremely complex, involving an intermeshing of specific and non-specific factors. Nevertheless, certain diseases, because of correlated clinical and experimental data, are almost universally accepted to be primarily stress related (not withstanding that the definition of stress may vary from situation to situation, if even defined at all).

Various nomenclature has been proposed to classify the stress diseases, for example, a list by Dr. B. Haynes, from his book Autonomic Dyspraxia (1958).

All of these diseases are diagnosed as “autonomic dyspraxia” on the basis of the following symptoms (in addition to the primary presenting complaints), any two of which, he submits, proports a very good chance of primary autonomic dysfunction.

For example:

Globus hystericus (difficulty in swallowing) is possibly one of the most common symptoms, as are headache, flatulence, flatus and indigestion (even in the case of diaphragmatic hernia he contends that the symptoms are invariably due to a concomitant autonomic dyspraxia).

In the case of headaches persisting over a period of months he quips that "the vast numbers of aspirin preparation consumed daily makes the sum total of all 'organic cases' at our hospitals. . . truly microscopic in comparison." Nevertheless he does feel that "the first step in diagnosis is still to exclude this small but most important group." This, he says, "is possible in the first or second consultation, mainly by clinical methods. In the great majority of cases, multiple tests, and X ray procedures a la May, are not only unnecessary but definitely contraindicated." (Haynes (1958)

Hans Selye (1976), on the basis of an extensive (massive) review of the clinical and experimental literature, presents an even longer list of stress related disease categories. It would serve no useful purpose to review these areas again; discussion is limited to a few cases, as they relate to the concepts derived in this paper.

Eppinger and Hess (1910), in their classic (if not somewhat horrifying) clinical studies on psychosomatic medicine, divided psychosomatic disorders into two broad categories: those associated with sympathetic predominance (sympathetonia) and those with parasympathetic dominance (vagatonia). Various authors, e.g., Gellhorn (1965) have followed this line in classifying stress diseases with an associated dominant or "tuned" autonomic branch.

ii. Hypertension

This dichotomy appears to make sense, for example in the case of essential hypertension, where Folkow and Rubenstein (1966) were able to produce, in rats, sustained heightened blood pressure by daily stimulation, for several months, of the postero-lateral hypothalamus.

This showed that chronic ergotropic (sympathetic) activation is capable of inducing hypertension along with degenerative cardiovascular and renal lesions (Henry, 1967). In addition, pressor responses can be conditioned in man and animals with hypertensives conditioning much more readily (Miasnikof, 1962).

While these and similar observations cannot by themselves be considered proof that the various clinical hypertensive syndromes have, as their basis, prolonged sympathetic tone, they must be considered highly relevant to these pathologies.

Thus the prolonged accumulation of sympathetic cardiovascular stress is at least contributory in a wide range of very serious tissue pathologies.[78]

According to the model, "simple" sympathetic accumulation is an initial stage in the progressive accumulation of autonomic stress. Further accumulation, while perhaps having no more dramatic consequences than those of this initial stage, would be expected to exhibit more complex, varied and divergent behaviors and symptoms. This is because, according to the model, parasympathetic

components begin to enter into the accumulation, progressively, at higher levels, resulting in cusp region catastrophe behavior.

iii. The Ulcer

The duodenal ulcer is a disease often ascribed to a trophotropic imbalance since hypersecretion of hydrochloric acid is associated with vagal activity. (It is even sometimes treated surgically by partial vagotomy.) In addition, ulcer patients are also often know to exhibit such signs and symptoms as low blood pressure and easy fatiguability, indicating a trophotropic pattern. Thus, as Gellhorn (1965) argues, it makes sense to consider the ulcer as a case of parasympathetic tuning. Nonetheless, Simeons (1962) points out that high concentration of hydrochloric acid does not per se cause ulcers, implying vasoconstriction as a necessary factor, too. This would presumably be due to a concurrent sympathetic activation. Also, while many ulcer patients exhibit hypotension and other symptoms of vagatonia they are often (against their doctors' admonitions) involved in highly driven and aggressive behaviors which are more characteristic of ergotropic syndrome. So while trophotropic signs and symptoms appear to predominate, it would be well worth looking for "masked" sympathetic tonus also in these individuals, as well as discontinuous shifts in their behavior and matrices of symptoms.

iv. Vasodepressor Syncope

In this "fainting" syndrome the probability of dual autonomic activation seems somewhat clearer. Vasodepressor syncope is usually precepitated by pain and fear of injury; and as Engle (1962) points out, it is evoked "in a situation where the general circulatory preparation for flight (i.e., the ergotropic syndrome) takes place but for some (italics mine) reason flight is impossible." This "some" reason is reflected in a loss of tone in the skeletal muscles and is quite probably due to parasympathetic inhibition of the gamma efferents.[80]

Thus it seems that the simultaneous activation of both autonomic components leads to the maladaptive response of so to speak, "being overwhelmed." If, though, the individual, at the moment of fainting, tenses his muscles, the attack can be halted. In terms of Catastrophe topology this would amount to introducing a momentum factor at that critical time, causing behavior to jump back to a higher motility surface. (See figure IX).

v. Anxiety States (General)

Gellhorn (1965), in summarizing a wide variety of physiologic experiments, reports that anxiety states are associated with high degrees of ergotropic activity and simultaneously with "abnormal behaviors," when accompanied also by trophotropic signs. These behaviors appear to often stem from an alternation between opposite extremes of possible action, e.g., attack-retreat, or approach-avoidance. It seems reasonable in studying anxiety experimentally, then, (the experimental neurosis) to look for cusp Catastrophe behavior, and to correlate it with autonomic states. The experimental neurosis, in animals, would provide a unique window in that central sympathetic and parasympathetic activities could be measured directly by electrical recordings.

vi. Anorexia Nervosa

Patients suffering this life threatening illness are characterized by "obsessional" behavior of alternately fasting and goring themselves with food. The disease has been classified in almost every diagnostic category imaginable: Mecklenburg et al.(1974) have investigated hypothalamic function in patients with this syndrome to test the hypothesis that a lesion (their word) in the region is critical to the pathophysiology of this "disease."

Their conclusion is that "patients with anorexia have primary hypothalamic disease of unknown etiology." The data they present, however, support equally if not more strongly the Catastrophe formulation that small (functional) shifts in hypothalamic (autonomic) balance can account for the striking behavioral changes evidenced by this disorder. For example, the following data compare the response to acute hypo-and hyperthermia of normals and anorexics (see tables on next page)

The authors state that "it is possible to clearly separate patients with anorexia nervosa from normal subjects by their responses to heat and cold." Yet clearly, in their response to hypothermia, subjects BB and NP are quite similar, as are EG and RS (more so than most patients within the group, e.g., BP and EG). Similarly, in the response to hyperthermia, normal BB and patient OP are quite alike, as are RS and EG, indicating only small autonomic shifts.

The statement that the pathogenesis is a hypothalamic lesion is supported, the authors claim, by:

…the observations that emotional and behavioral disorders as well as abnormalities of sleep-wake pattern, water balance, thermoregulation, carbohydrate metabolism, and gonadotropin secretion may follow experimental hypothalamic lesions. Patients with syndromes approximating (italics mine) anorexia nervosa have been reported with tumors in the hypothalamic region or following central nervous system infections.

But all of these effects are equally compatible with the diminished adaptational capacity of autonomic imbalance characteristic of concurrent sympathetic/parasympathetic activation. In addition, Zeeman (1976) reports the work of a hypnotic trance approach which appears possibly to be the most effective form of therapy for this disorder, the only one where a substantial portion (though still small in number) report being cured and regain normal weight. Zeeman has been able to model this form of therapy quite elegantly by Catastrophe theory, suggesting again that the model is a promising choice for stress related disorders.

vii. Role of Stress in Primarily Infectious Diseases

The role of stress as a prominent contributory factor even in diseases of less obscure etiologies (infectious disease) is suggested by a fascinating paper by Akiro Saito (1970) of the Tohoku Medical School, Sendai, Japan.

On the basis of more than 15,000 patients, this author has studied the autonomic/hematpoietic relations in several infectious diseases. He summarizes this extensive work as follows:

Thus, observed from the level of autonomic nervous systems, the auto-adaptation mechanism of the human body consists of 2 major antagonistic systems which are composed of many antagonistic links of 2 nerves of the autonomic nervous system—2 phases of mitosis of the neutropoietic system in the bone marrow—2 defense reactions of the blood—2 fields of the blood defense reactions. These 2 major antagonistic systems. . .maintain life in a most suitable and purposeful way . . .The author has found that in a person who has an imbalance of the autonomic nervous system the adaptation of the body to the internal environment loses its suitableness and purposefulness, and an abnormal defense reaction occurs, causing a series of adaptation disturbances from acute to chronic type.

The implications of this are simple and inescapable: The outcome and process initiated by a foreign infective agent are determined perhaps as much by the functioning and dynamic capability of the autonomic system, as by the agent itself.

Some degree of caution, however, is needed, as certain steps in reasoning are not adequately connected in Saito's paper. Nonetheless, what he does offer is the possibility of a rather direct relationship between the accumulation of autonomic stress and the realm of infectious pathogenesis in general.

viii. Aging and Stress

In a provocative paper, V.M. Dilman (1971) at the Petrov Research Institute of Oncology in Leningrad proposes that age changes in the self-regulating systems affect the maintenance of a stable internal environment.[81] The key in this process, he argues, is the decrease, in aging, of hypothalamic sensitivity to feedback suppression. In his words, "This gradually leads to the loss of rhythmic functioning of the main homeostatic systems." He makes further the statement that the existence of this hypothalamic phenomenon per se is sufficient for the age related "switching on and off" of the reproductive cycle, which is "needed for the establishment of relationship between energy and reproductive homeostasis in ontogenesis and to ensure their interaction wit external environmental factors; regulating the density of population, and also for the gradual age-related impairment of homeostasis that finally leads to death from the natural onset of diseases of compensation."

In other words, he sees aging as the same process: "loss of hypothalamic sensitivity to feedback-suppression," as that induced by accumulated stress (as in this model), but at a slower time scale (in aging). Thus, behavior which has become restricted to "energetically confined" portions of the space[82] is due to simultaneous sympathetic and parasympathetic activation. This prescribes a "process of disordered homeostatic stability of the internal environment." (See III, B.)

ix. Hyperventilation: Syndrome and Clinical Effects

The mechanisms of hyperventilation are discussed in appendix ii. As a clinical entity:

The syndrome of hyperventilation is one of the most common and yet one of the most infrequently recognized medical disorders (italics mine). This functional derangement of breathing, with the sequela it precipitates, is often regarded as a manifestation of nervousness, yet when organic disease is simulated by the syndrome of hyperventilation, serious consequences by unwarranted restrictions may result from an erroneous interpretation of a patient's symptoms. The syndrome of hyperventilation results from excessive loss of alveolar carbon dioxide caused by increased respiration. By reduction of the partial pressure of alveolar carbon dioxide from its usual value of that of 40 mm. Of mercury to a value less than half that, respiratory alkalosis is induced in the body.[83]

That the syndrome is a manifestation of "nervousness" is only a part of the story: the sensation resulting from hyperventilation is one of suffocation and illness, evoking then further hyperventilation and thus setting up a vicious cycle. It appears that thoracic stretch receptors are particularly effective in evoking sympathetic discharge which will evoke further hyperventilation, which in turn stimulates the sympathetic via these receptors. (See Koizumi & Brooks, 1972.) This process cannot go on unchecked, and it is reasonable to assume that eventual spillover onto the parasympathetic (which decreases respiration) will maintain the system in a "meta-stable state," i.e., the low motility, high activation stasis.

x. Childhood Autism

As previously mentioned, Childhood Autism is characterized by an excessively high internal excitation. Thus this "disease" appears to be similar to the stasis condition described, where behavior is trapped in a cul de sac of low motility, while the autonomic state remains high centrally. This extreme split of behavior and internal set makes for limited and "fixed" modes of emotional expression[84] which characterize these most unfortunate human beings.

The extreme nature of this split between autonomic behavioral components may well be, as Tinbergen argues, a function of the early developmental age (birth or before) at which major unresolvable stresses have occurred.

The work of William Windle reported in his book, The Physiology of the Fetus (1971) is particularly disturbing in this regard: Monkey neonates, delivered by various routine hospital procedures, exhibits gross morphologic and histological brain defects due, he argues convincingly, to asphyxia neonatorium, as compared with spontaneous births. This may have its unfortunate parallel in humans, in whom, he argues, Apgar scores correlate inversely to the degree of hospital involvement; see Ch. 15, on mental retardation. Thus, the combined effects of anesthetics, delivery position, entrapment, etc., may well have profound effects on an organism's potential reserve capacity well beyond the gross damage observed histologically.

Windle states in his introduction that:

To be born undamaged mentally as well as physically is the primary right of every human being. We should inquire from time to time whether all possible provisions are being made to protect this right. The medical scientist has an obligation to seek and evaluate factors that may affect health in

utero and well being after birth. As new knowledge of physiology needs to be considered. Attention will be directed to some of the factors that can damage the brain, oftentimes quite subtly but permanently.

The effect of stress is, in this regard, certainly one of these obligations. The next sentence after the above quote regards, “First, however, a survey of the normal physiology of the fetal organ systems is in order.” Unfortunately this cannot be done in the case of accumulated stress. To understand the mechanisms of stress accumulation and minimize its effects, particularly at crucial developmental periods, is a step towards insuring these primary rights, spoken of by Windle. If this goal is aided by the efforts of this dissertation, then it’s many shortcomings and limitations are more than compensated for by its basic validity.

Pre-symptomatic (Accumulated Stress) Diagnoses

The approach of modern medicine is geared to the appearance of symptoms or debilitation and then—at least in the instance of the “stress disease”—to the treatment of the pathologic expressions, along with advice to the patient to “relax,” to “not worry,” to “exercise,” to “vacation. . . “ In short, not to accumulate any more stress. But it is just the fact that they are no longer able to discharge or resolve stress sufficiently that underlies such patients’ symptoms and pathologies. It is in this direction that the various unconventional systems discussed appear to be most concerned. In particular, they view the detection and correction of these imbalances before they become symptomatic (certainly before they become chronic) as the primary focus. It is in this stage that the organism still has the reserve capacity, the resiliency and strength to begin to resolve, successively, these accumulations before they become debilitating. In addition, when chronic conditions are presented, these systems often treat the underlying imbalance independently of the specific symptoms.

This focus on “pre-symptomatic diagnosis” could be one of the most immediately viable aspects of these systems to be incorporated into modern orthodox diagnostic procedure. (Certainly there can be little doubt that early diagnosis is often related to favorable prognosis.) The problem of diagnostic screening is becoming one of the most complex and alarming issues in medicine today, and radically different inroads certainly seem appropriate. The case of stress disease is painfully obvious. The cure rate of these conditions once they have become established is pathetically low. The cost of administering “care,” for example, to the “mentally ill” is alarming financially but primarily in human suffering. Surely no one would argue against the benefits of preventive medicine, yet systematic approaches to the problem simply do not exist. Beyond the adages, “Early to bed, early to rise,” “sensible nutrition,” and “regular exercise,” etc., there is, in Western medicine, no coherent picture of the ways in which an organism’s capacity to respond to the “stress of life” is maintained, diminished, measured or treated.

It is the view of this dissertation that sufficient knowledge does exist, in the form of cybernetic analysis of physiologic function (both at the unit and systems level), to provide a basis for an integrative approach to human health—to its assessment and maintenance, as well as to its quantitative measurement. It cannot be overemphasized that his will (and must) involve a multiplicity of factors, from the molecular to social and environmental. This dissertation deals only with the "tip of a titanic iceberg" of these factors. In the balance, the ideas, derivations, predictions, etc., derived here depend for their validation on the successful evolution of "integrative medicine."

Since these studies must ultimately, for their empirical validation, involve cross-sectional testing of large populations, the question of measurement is paramount. The mecholyl-noradrenaline test, discussed, has many practical as well as theoretical limitations. Notably, there is a lack of graded sensitivity to subtle autonomic shifts, and awkwardness in administering the test.

The pivotal issue in assessing accumulated stress is the definition of central integrative states on the basis of readily observable peripheral outputs. That this is, in principle, plausible, stems from the fact, discussed throughout this work, that the entire spectrum of central integrative processes is expressed, peripherally, by two opposing systems of output. These outputs in the "unstressed organism" operate in a mutually antagonistic mode, while as stress accumulates, this reciprocity begins to "break down."

This systems view of bipolarly organized adaptive stress behavior was found in several instances to be represented and predicted from the basic principles of Catastrophe theory, and it is expected that this approach would be an excellent one to future studies of the accumulation of stress and its behavioral expression. It should, in principle, be possible to plot an individual's adaptive range in terms of these typographies, and correlate them with the appearance of eventual "breakdown" in disease, as well as to asses a particular course of treatment.

Perhaps one of the most promising tools in the area of pre-symptomatic diagnosis, based on the findings of this thesis, could be time analysis thermography. This highly sophisticated technologic device, perhaps more than any other, is suited ideally, not only for recording sensitively and graphically a wide range of vasomotor responses over the entire body, but as an integrative bridge between systems-topology viewpoints and the observations and principles of the various "archaic" and holistic approaches.

One of the pervasive themes of the holistic approaches is their "energetic perspective." They view behavior as a rhythmic bipolar interplay. When this flow is unimpeded, "harmonious," efficient function will be evident, but when "blocked" or "imbalanced" a disturbance of function (disease) results.

The thermographic motion picture data taken by Frank offer a direct observation at a physiological level of these autonomically mediated energetic phenomena. From his data on Reichian vegetotherapy, one can discern clearly traveling wavelike patterns of vasomotor changes which are modified in their magnitude and speed and may even be reversed in segments where "muscular blocks" occur. Further, as these blocks are "dissolved," the wavelike propagations continue, unimpeded, until they reach another, more distal or deeper "block" (and so on).

If it proves that these propagated waves of vasomotor tone can be related predictably to the neuromuscular patterns recorded by Dr. Hunt, then not only can the relation between autonomic and somatic components in the charging and discharging process be measured, but the response to any given autonomic stress stimulus can be visualized on the Catastrophe surface. Thus, the specific stress configuration of any individual could, in principle, be determined topographically.

It may not be overenthusiastic to hope that this sort of methodology will open entirely new avenues in a integrative medicine. There is, however, a practical limitation in that both the telemetered computer emg and thermographs require highly specialized and expensive equipment, and to have use of both, in the same laboratory setting, may “take some doing.” The actual diagnostic work, however, would eventually be done with the thermographic equipment alone (after the autonomic-somatic relations had been established). And since many hospitals already have computers (or the time shared use of them), the cost of setting up such a facility would not be prohibitive and might even pay for itself in a surprisingly brief period of time.

The cost of not having such a tool for research and medicine, in the opinion of this author, is intolerably expensive. To quote Tinbergen again, at the risk of sermonizing: “It is stress in the widest sense, the inadequacy of our adjustability, that will become perhaps the most important disruptive influence in our society.”

Details and discusses the systems simulation used to derive the independent sympathetic and parasympathetic autonomic components plotted, and which allowed the various.