i.e., the result of interrelationships among genetic, endocrine, neurologic, and immune systems, as well as behavioral and emotional factors.

Stress has been shown to have a significant affect on the cardiovascular and respiratory systems. Functional disorders of the gastrointestinal tract account for 50 per cent of the referrals to a gastroenterologist. A study of air traffic controllers compared to a matched group of U.S. Air Force personnel demonstrated twice the incidence of peptic ulcer disease. Migraine headaches can be precipitated or aggravated by stress. Stress can modulate the activity of the immune system, and depression can compromise specific components of the immunologic apparatus. Surviving spouses are at a higher risk of death than would be expected from standard mortality rates. Stress can precipitate thyroid dysfunction, and control of diabetes mellitus is adversely affected by stress.

Chrousos and Gold[2] report that the main components of the stress system are corticotropin-releasing hormone and locus ceruleus-norepinephrine/autonomic systems and their peripheral effectors, the pituitary-adrenal axis, and the limbs of the autonomic system. Activation of the stress system leads to behavioral and peripheral changes that improve the ability of the organism to adjust homeostasis and increase its chances for survival. This new knowledge has allowed association of stress system dysfunction, characterized by sustained hyperactivity and/or hypoactivity, to various pathophysiologic states that cut across the traditional boundaries of medical disciplines. These include a range of psychiatric, endocrine, and inflammatory disorders and/or susceptibility to such disorders.

Chrousos and Gold also note that shifts in the dose-response curves of stressor versus stress-system activity could reflect a hereditary trait resulting from a genetic defect in metabolism, such as a change in the quantity or quality of expression of a particular gene for a hormone, a receptor, or an enzyme. So the magnitude or duration of the stressor, the critical timing of the event, the actual genetic vulnerability, and the influence of the social environment might ultimately determine the pathogenesis of a syndrome related to dysregulation of the stress system.

In terms of studying coagulation defects, Urano and Cho et al.[3] purposely stressed subjects in order to study the influence of stress on the coagulation and fibrinolytic system. These investigators found that there was enhanced fibrinolysis due to increased tPA levels and decreased PAI-1 levels. Other investigators[4-9] have shown that physical and/or mental stress are able to influence the coagulation and fibrinolysis systems.

Jern and Eriksson et al. from the Coagulation Laboratory in the Department of Clinical Physiology showed the affect of mental