In Chapter 2 we reviewed the literature and proposed a model which may be used as a basis for understanding stress as a psychophysiological response process. In Chapter 3 we shall take the logical extension from the psychophysiological stress response itself and discuss how stress may lead to disease. Also in this chapter we shall briefly mention the stress-related disorders which are most commonly encountered by the clinician.

THEORETICAL BASES FOR THE DEVELOPMENT OF STRESS-RELATED DISEASES

As noted in the previous chapter, all the potential stress-response axes detailed earlier cannot become active at once within the same individual. Nor can all of the potential end-organ systems be affected within the same individual during a single stress response. However, the method by which the human body specifically selects which end-organ system to affect and via which stress response axis to do so is currently open to speculation. As a result, there has been considerable theory formulation and research as to the nature of the link between stress and disease.

Lachman’s Model

In a “behavioral interpretation” of psychosomatic disease, Lachman (1972) proposes the following model to explain the stress-to-
disease phenomenon: "In order for emotional reactions to assume pathological significance such reactions must be intense or chronic or both" (p. 70). He goes on to state that which end-organ structure will be affected pathologically depends on:

1. Genetic factors which biologically predispose the organ to harm from psychophysiological arousal.
2. Environmental factors which predispose the organ to harm from psychophysiological arousal. These would include such things as nutritional influences, infectious disease influences, physical trauma influences, etc.
3. The specific structures involved in the physiological reactivity.
4. The magnitude of involvement during the physiological response, which he has defined in terms of intensity, frequency, and duration of involvement of the organ.

Lachman (1972) concludes that the determination of which structure is ultimately affected in the psychosomatic reaction depends on "the biological condition of the structure," (whether a function of genetic or environmental influences), "on the initial reactivity threshold of the organ, and on . . . learning factors" which affect the activation of the organ. He goes on to note that the "magnitude of the psychosomatic phenomenon" appears to be a function of the frequency, intensity, and chronicity of the organ’s activation.

**Sternbach's Model**

In a somewhat more psychophysiologically oriented model, Sternbach (1966) provides another perspective on the stress-to-disease issue.

The first step in Sternbach’s model is *response stereotypy*. This term generally refers to the tendency of an individual to exhibit characteristically similar patterns of psychophysiological reactivity to a variety of stressful stimuli. Sternbach views it as a "predisposed response set.” That such a response stereotypy phenomenon does indeed exist has been clearly demonstrated in patient and normal populations (Lacey & Lacey, 1958, 1962; Malmo & Shagass, 1949; Moos & Engel, 1962; Schnore, 1959).

Response stereotypy may be generally thought of as a form of the "weak-link" or "weak-organ" theory of psychosomatic disease.