In Sweden, as well as in many other European countries, there is growing awareness that psychosocial factors may be important not only to people's general well-being but also to the maintenance of their bodily functions. A dramatic breakdown of human adaptation to psychosocial factors could be myocardial infarction and cardiovascular death. Whereas laymen have always accepted the possibility that psychosocial processes are linked with cardiovascular illness and death, the medical profession has been more hesitating in accepting the idea that there may be a causal link. Establishing causal links may be important both from a legal standpoint, for instance in work compensation cases, and from the standpoint of planning cardiovascular primary prevention. In the present article I do not intend to review all the efforts to establish causal links in this research field but rather ask a few questions of a general nature. Why does our cardiovascular system react as it does to certain challenges and what do some of the associations that have been seen mean in a physiological sense?

The most important question may be: "What is a cause?". Or in other terms: "Is it possible to prove a causal link between psychosocial factors and cardiovascular illness?"

In the case of psychosocial factors we can make the assumption that our general life situation may influence our bodily functions either directly through the brain or indirectly through changes in our life style. This has great importance when we discuss causality. Most medical researchers in the field have focused their attention on direct links for instance between "stres­sors" in the work place and increased risk of coronary heart disease (CHD), that is neurogenic influence on relevant endocrinological and cardiovascular functioning. Indirect links have been disregarded since they have not been...
considered causal. By indirect links I mean the influence of "stressors" on accepted risk factors such as dietary habits, cigarette smoking and blood pressure. According to many researchers, indirect links should not be considered causal (1). But what happens if psychosocial factors cause a change for instance in smoking habits? If that is the case no anti-smoking programme will be effective unless we take account of the psychosocial mechanisms involved in the smoking habit.

What is the overall physiological meaning of some of the pathophysiological processes relevant to CHD development? How do these processes relate to psychosocial factors now and in previous generations?

Every layman has heard of the fight and flight responses to threatening situations. The physiological meaning of these reactions is to provide the body with energy for muscular action and protection against loss of blood (platelet aggregation tendency increases) and salt and water due to sweating (mineralocorticosteroids) which takes place as a response to increased heat production. From a survival perspective the provision of energy is the primary goal. This is achieved by means of catabolism. If the state of alarm is long-lasting the available stores of energy will be emptied and the catabolism will literally dissolve useful protein which is broken down into glucose and waste products. At the same time, anabolism is unfortunately inhibited. Anabolism restores tissues that are being worn out continuously and builds up useful protection against breakdown. Examples are replacement of worn-out epithelial cells in the skin and the gastrointestinal system and leukocytes that are important in the defence against infection. Thus, life could be seen as a continuous battle between catabolism and anabolism. If the catabolic reactions become too longlasting and intense, several of the body systems become "exhausted" (using Selye's terminology (3)) and stress-sensitive. On the other hand intense catabolic periods may be harmless if periods of intense anabolism are interpersed between them.

Physicians frequently assume that cardiovascular disease processes are immune to "stress". In cases of work compensation the argument is often put forward that "stress" may precipitate episodes of myocardial infarction but is not causing atherosclerosis which should be regarded as the underlying cause. However, even if we make the cautious assumption that psychosocial factors are unrelated to atherosclerosis, the matter may not be quite