If "aged" is defined as greater than 65 years old, then at the present time approximately 11% of the United States population, or 25 million persons, fall into this category. It may be likely that within their lifetime 50% of these individuals will need anesthesia and surgery. Consequently, it is understandable that there has been a good deal of attention paid recently to the problem of anesthesia in the geriatric population. Although there is somewhat more published data on other aspects of anesthesia and the older patient, as will become evident, there is great sparsity of solid data looking at the effect of anesthesia and anesthetics on cardiovascular function in the aged, and particularly comparing this effect with a younger population. Perhaps the cardiovascular effects of aging are not that important. In fact, it has been suggested that the major diseases interfering with physical activity in the aged population are non-cardiac (1). What then are the cardiovascular effects of aging?

EFFECTS OF AGE ON THE CARDIOVASCULAR SYSTEM

Although it has been said that cardiac output is 35% less at the age of 70 than at 20 years (2) or that maximum cardiac output decreases at a rate of 1% per year from the middle of the fifth decade of life onwards with heart rate decreasing by about half that rate (2), in fact, it would appear that these observations may be a function of disease and environment (3). Non-invasive longitudinal studies have shown that as the healthy individual ages, there is no change in resting heart rate nor in the response to rather substantial work loads. However, the mechanism of maintaining heart rate does vary between the young and old. Whereas the young tend to increase their cardiac output in response to stress by increasing their heart rate with little change in stroke volume or end-diastolic volume, the
elderly predominantly increase cardiac output under these circumstances by increasing their ventricular filling pressures and volumes (13). The mechanism behind these changes is probably two-fold. In the first place, as we age, there are definite anatomic changes in our resistance arteries with increased water and fibrous tissue content and less elasticity leading to progressively increasing systemic vascular resistance. In order to maintain cardiac output, the aged cardiovascular system responds with increasing preload. One of the reasons for this is that there is reasonable evidence in both animals and man that although the activity of the sympathetic nervous system does not change appreciably with age, and in fact may even increase (plasma catecholamines), the response to this sympathetic stimulation definitely does decrease so that even with increasing sympathetic nervous system activation, there is little response of heart rate and cardiac contractility. Of further interest in both aging animals and humans, the observed deterioration in large patient populations in cardiovascular function can be prevented or even reversed by conditioning and exercise programs (3). Of course, as we age there is an increased incidence of cardiovascular diseases (4). Consequently, the effects of aging on the cardiovascular system must encompass not only chronologic age but also the effects of environment and disease.

EFFECTS OF ANESTHETICS ON THE HEALTHY CARDIOVASCULAR SYSTEM

Inhalation Anesthetics. The currently available potent inhalation anesthetics, halothane, enflurane, and isoflurane, have similar effects on the cardiovascular system in healthy humans. In general, with increasing dose, all three anesthetics produce a decrease in cardiac output, stroke volume and systemic vascular resistance. However, there appear to be quantitative differences. Halothane has minimal effects on heart rate whereas both enflurane and isoflurane tend to produce increases in heart rate although not dose related. Although high concentrations of halothane and enflurane can produce a decrease in systemic vascular resistance, clinical concentrations usually have little effect on the peripheral vascular system. On the other hand, there is a dose-related decrease in systemic vascular resistance produced by isoflurane. Partly as a result of this, equi-potent anesthetic concentrations of isoflurane