# BRIEFS FROM THE LITERATURE

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## ANESTHESIA AND CIRCULATION

This review has five major divisions: effects of anesthetics on the (1) heart, (2) blood vessels and (3) autonomic nervous system; and the over-all effect of these actions on (4) hemodynamics in general and (5) tissue perfusion in specific regions of the body. The effects of general anesthetics on myocardial function include reduced contractile force, and rate of depolarization, reduced contraction rate and increased duration of isometric contraction, slowed relaxation rate and increased mechanical impedance, shortened refractory period and increased threshold to electrical stimulation. Decreased contractile force, although present both in the isolated heart and the intact preparation, is relatively difficult to demonstrate in the latter. This suggests the existence of compensatory reactions which may include: reduced kinetic work by the heart, reduced pressure-volume work (hypotension), tachycardia, increased mean circulatory filling pressure (venous return), increased endocrine secretion (including increased activity of the cardiac sympathetic nerves), and tolerance to narcotics developed at the cellular level. These factors, particularly the role of the sympathetic nervous system, catecholamines of other hormones, are discussed. Under basal conditions, cardiac output is little affected by primary changes in contractile force. This has important implications for homeostasis since it permits maintenance of a normal minute volume in the face of a failing myocardium.

Peripherally, the effect of anesthetics on the caliber of microscopic blood vessels consists usually of mild constriction. Unfortunately, the observation of living blood vessels under a microscope does not lend itself to quantitative expression; it is incapable of sensing such vital information as oxygen tension, hydrostatic pressure, blood vessel permeability, *p*H, and perhaps even the rate of blood flow. The ability of an organism to maintain constant internal conditions despite changes in its environment depends ultimately upon the integrity of its internal communications. It is relatively simple to show that general anesthetics disrupt function in many types of nervous tissue under laboratory conditions, but it is difficult to establish the extent of their interference with a specific function in intact subjects. The extent to which cardiovascular reflexes (carotid sinus, tilt, pressoreceptors, chemoreceptors), afferent transmission, central nervous system activity at various functional levels, and the efferent autonomic nervous system are depressed by anesthetics in animal and man is reviewed. These great numbers of factors which can modify circulatory activity during general anesthesia make it perfectly valid to study hemodynamic alterations to general anesthetics as they are usually administered in the operating room, accepting the possibility that pharmacologic analysis may be impossible. Acceptable data regarding the hemodynamic effects of cyclopropane, diethyl ether, halothane, chloroform, and barbiturate anesthesia are considered. Also considered is blood flow to various organ systems, namely, brain, heart, viscera, kidneys, skin, muscles and subcutaneous tissue. Despite the undesirable effects noted on the circulation, the safety of general anesthetics is apparent from the many instances in which they are administered without obvious evidence of ill effect. This safety clearly results from the ability of the body to cope with toxic actions and not because general anesthetics are innocuous. (*Price, H. L.: General Anesthesia and Circulatory Homeostasis, Physiol. Rev. 40: 187 (April) 1960.*)