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Editorial Views

Anesthesia and the Microcirculation

THE MAINTENANCE of adequate tissue perfusion during and after anesthesia is contingent upon an effective microcirculatory blood flow pattern. How anesthetic agents affect the microcirculation, therefore, is of practical concern to anesthesiologists. The report by Harris and co-workers in this issue of ANESTHESIOLOGY of the microvascular responses of the bat wing to pentobarbital and thiopental anesthesia adds important quantitative information about this area of circulatory function.

The anatomy, physiology, and pharmacology of the microvasculature of many tissues have not yet been comprehensively elucidated because of the technical difficulties inherent in *in-vivo* microscopy within the substance of three-dimensional organs. Yet, from study of transparent two-dimensional tissues (such as mesenteries) and the surfaces of solid organs, enough of the structural organization and functional behavior of the microcirculation is known to afford a reliable understanding of its basic operational activities.

It is not enough to point out that the microcirculatory system consists anatomically of small arteries, arterioles, capillaries, venules, shunts and thoroughfare channels, for without question the alignment and architectural organization of these microvessels in any given tissue are intimately related to the nutritional needs, metabolic activity, and homeostatic functions of that tissue. In some microvascular beds the inflow arteries and arterioles may be terminal and in others the supplying vessels are arcuate (interconnected), the various vessels subserving exchange vessels of different quantitative potentials. Thoroughfare channels predominate in some tissues (mesentery–omentum), direct arteriovenous shunts in others (skin, liver). Capillaries can have specialized structural characteristics which permit unusual permeability. Irrespective of these variations in architectural patterns, however, there exists in almost every nutritional bed a basic anatomic microvascular module, about which the system of exchange vessels is organized and which provides a rational basis for understanding the interplay of the factors which regulate blood flow through tissues.[1] This basic unit consists of centrally located muscular precapillary components, arteriole, metarteriole, and precapillary sphincters. Distribution of blood flow in the capillary bed is accomplished by the coordinated vasomotor activity of precapillary muscular components.

The tonic activity of arterioles and sphincters is regulated by nerves, humoral substances, and local control mechanisms. These local and remote regulatory factors, by their interactions, set the level of tone of smooth muscle cells in the small blood vessels. Most dynamic studies indicate that neural control is predominant in small arteries and large arterioles only, and that locally released substances are more critically active in metarterioles, precapillary sphincters and muscular venules. The net effect of these influences on the individual vessels is an integrated response appropriate to the immediate moment. In this way, local blood flow is continuously adjusted to the ever-changing local tissue needs with exquisite precision. This remarkable homeostatic regu-

lation of tissue perfusion is vulnerable, however, to factors such as anesthetic agents, which modify the functional capacity of the regulatory mechanism.

Studies by direct *in-vivo* microscopy demonstrate that anesthetics affect the normal integrated response of the microvasculature in direct proportion to the depth of anesthesia. For example, in the ear chamber preparation in the unanesthetized rabbit,[2,3] in the omentum of the dog,[4,5] and in the mesoappendix of the rat,[6] arteriolar constriction and enhanced vasomotor activity are seen with light ether and cyclopropane anesthesia. This pattern of heightened tone and reactivity also occurs during light halothane anesthesia,[6] and significantly restricts blood flow through thoroughfare channels. With all three agents increasing the depth of anesthesia reduces vasomotor activity, leading to vascular hyporesponsiveness, venular dilatation, and pooling of blood in the capillary bed.[4-6] The prevailing microvascular reaction can also depend on the specific anesthetic. In the rabbit ear and dog omentum, arteriolar dilatation predominates with thiopental.[3,5] Light methoxyflurane anesthesia induces prompt cessation of vasomotion and dilation of pre- and postcapillary muscular microvessels in the rat mesoappendix.[6] The responses of the microvasculature in the intact bat wing also vary according to the anesthetic used, as is reported in this issue. Differences from one species to another and from one tissue to another are to be expected, since tissue environments and neural tone in different vascular beds are not the same.[7] Also of importance to anesthesiologists is the fact that vascular muscle seems to be pharmacologically heterogeneous in its responses to vasoactive drugs and in different vascular beds.[8,9]

Certainly, despite the technical difficulties, much more quantative information about the effects of anesthetics on the microcirculation is needed. Measurements relevant to man must be made as directly as possible with minimum distortion or invasion of the intact organism. This stringent requirement undoubtedly can be met by modern technology, which already offers an array of accurate and sophisticated sensing, measuring and recording techniques that can be used in conjunction with the light microscope. The methods used by Harris *et al.* illustrate this technology. The use of the vasculature of the bat wing, which uniquely among all other species studied demonstrates "vanomotion," as a test model for the anesthetics provides a substantial contribution to our knowledge of the actions of anesthetics on the microcirculation.

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