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Hemodynamics I
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Venomotion: Its Effect on Peripheral Circulation

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Observations of rhythmic contractions of peripheral vessels, in a variety of animal species, having periodicities unrelated to heart rate or respiration have inspired an array of theories regarding their function. It has been suggested[1] that venomotion, the rhythmical contraction of muscular venules and small veins, plays a significant role in normal venous return. Two quantities - volume alterations within, and flow out, of these vessels - appear implicated in the pathology of both cardiogenic[2] as well as hemorrhagic[3] shock in man. In this paper the hemodynamic consequences of venomotion on these two quantities are presented.

Passive Physical Properties

The muscular venules and small veins are assumed to be cylindrical vessels of constant length each having the geometric and topological properties reported[4] for the bat wing and internal radius to wall thickness ratio, (a/h), for corresponding vessels in the rat mesentery[5]. The results of a stress analysis indicate that over the physiological pressure range to which these vessels are exposed, the passive, relaxed dimensions are essentially independent of transmural pressure, and approximate the maximum internal radius (a_o) and minimum wall thickness (h_o).

Effect of smooth muscle contraction on Dimension

Using these dimensions as reference, the reductions in small vein radius, a_o, associated with spontaneous contraction of vascular smooth muscle, having force generating characteristics extrapolated from those reported[6] for the arteriole, is calculated. With the assumption of an effective elastic modulus of 10^6 dynes/cm^2 the relaxed internal radius, a_o, is reduced to .7 a_o. It is convenient to define one half this total change in a_o as the venomotion amplitude, A_o. The calculated value of A_o is slightly smaller than one reported[7] observation on a larger diameter vessel and is in very close agreement with observations[8] of similar size vessels.

Effect on Volume

The relationship between A_o and stored volume is determined by approximating the observed continuous venomotion pattern as a cosine function. The result indicates that a loss of venomotion permits an increase in time average small vein stored volume of 21% and 38% corresponding to values for A_o of .1 a_o and .15 a_o respectively.

Effect on Flow

The outflow producing potential associated with the observed longitudinally progressive contraction mode is determined for the case of a small vein with the assumption of no side branches. Fluid inertia is neglected on the basis of the prevailing low Reynolds number flow. Blood is assumed to behave as a modified Bingham plastic but yield stress is retained as a linear factor in the constitutive equation after approximation applicable to a suitable shear rate range. An expression is then derived relating the average pressure difference between two longitudinal reference points to mean flow. The vessel exhibits a contraction wave, hence an active source and time varying flow impedance are included in the considerations. The values calculated for flow in this way, using the measured[8] wave velocity of .3cm/sec over the range of observed venomotion frequencies (.15 to .5 Hz) and amplitudes, are found to bracket the values of average small vein flow, as determined both by extrapolation of observed[9] RBC velocities in capillaries and extrapolation back from large vein flow. The calculated active outflow capability without imposed longitudinal pressure gradient for A_o = .15 a_o is 4×10^{-7} ml/sec and the extrapolated value is 3.5×10^{-7} ml/sec.

The results indicate that, at least in one animal, the bat, there exists an identifiable mechanism, venomotion, the disturbance of which may lead to significant increases in stored blood volume and reduction in venous outflow. Viewed as a control mechanism it appears to possess the operational characteristics to provide a "buffering" action against changes in capillary pressure with less compromise in venous outflow than a passive system. Clarification of the mode and range of control and the interaction with precapillary hemodynamics is presently underway.

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