Response—Origin of Vasomotor Waves

Scott Hardin
Ohio State University Hospitals
Columbus, Ohio 43210

Vasomotor waves in the arterial pressure waveform during hypothermic cardiopulmonary bypass are mysterious in that, though clinically common, they have not received very much attention in the curriculae of perfusion schools or in the literature. Perhaps the reason for the academic obscurity of the phenomenon is that conclusive proof as to their nature is not easily obtainable, leaving explanations to the realm of unscientific hypothesizing.

Personally, I am not able to accept the theory that relegates the origin of vasomotor waves to autonomic nervous system activity, and in particular to cerebral ischemic centers. To do so is to ignore the basic physiologic functional characteristics and role of autonomic blood pressure regulation.

Autonomic pathways are designed by nature to regulate blood pressure with responses appropriate in both kind and amount to the sensed deviation from norms. Increased blood pressures stimulate the baroreceptors' efferent impulses to medullary centers, causing parasympathetic stimulation of both the myocardium and vascular smooth muscle. At mean pressures less than 100 mmHg, the efferent impulses to the medulla are at tonic levels. Chemoreceptor activity does not affect vasomotor tone to any large degree, as stimulation of cardiac and respiratory centers is more appropriate for correction of sensed abnormalities. Cerebral ischemic centers are the last line of support for the organism and elicit profound influence on vascular tone during severe crises, reportedly when mean arterial pressure is less than 40 mmHg. The characteristics of vasomotor waves do not match up with the functional role or the level of response of cerebral ischemia centers.

Recognizing that the descriptions of autonomic systems are made in reference to normal physiology, it could be theorized that they could operate outside of their normal range during the extremely abnormal physiologic state of hypothermic cardiopulmonary bypass. If I could believe that assumption, then the oscillatory nature of vasomotor waves would lead me to believe that some form of "thresholding" was being reached with the receptor sites. That is, the receptor sites are alternately "turning on" and "turning off" in response to their sensory input. Their average sensation would be hovering around some minimum standard, which is not to be interpreted as inadequate by the perfusionist. Exceeding the minimum would cause the waves to disappear and the perfusionist really could not say which side of the minimum he or she had swayed to.

I feel that vasomotor waves are caused by inherent properties of vascular smooth muscle. Small artery smooth muscle is of single-unit construction, like the smooth muscle of the intestines and uterus. Membranes of adjacent single-unit cells meet to form gap junctions where the cytoplasms of the cells actually are joined. Some single unit cells are capable of generating spontaneous action potentials without having nervous or hormonal input, much like cardiac muscle cells. These action potentials from so-called pacemaker cells are transmitted to adjoining cells across the gap junctions, resulting in contracture of the entire group as a single unit. The unique structure and function of this type of cell are responsible for such common physiologic phenomenon as peristalsis, childbirth labor contractions, cardiac rhythm and, I propose, vasomotor waves during cardiopulmonary bypass.

Hypothermic bypass provides isolation of vascular tone from other factors involved in blood pressure regulations. Systemic blood flow rates can be constant for relatively long periods of time.
The patient is in a deep resting state and cerebral function is depressed by the cool temperatures. My suggestion is that the conditions of hypothermic bypass favor the visualization of subtle vasomotor contraction that is lost amid other influences under other conditions. It would be interesting to study isolated smooth muscle preparations for effects of temperature and electrolyte imbalance on pacemaker cell impulse rate and on total muscle strength of contraction.

Credence to the theory that vasomotor waves are the result of endogenous smooth muscle activity comes from observations made after the administration of maintenance doses of cardioplegia. At this institution, cardioplegia volumes are absorbed into the systemic circulation. If vasomotor activity is evident, it often ceases shortly after cardioplegia doses are given (see Figure 1). Apparently, the increased serum potassium concentrations arrest pacemaker activities in the smooth muscle. As concentration gradients across the cell membrane equilibrate and resting membrane potential achieved, vasomotor waves become apparent again.

References