Letters to the Editor

Spatial orientation of the ventricular muscle band

To the Editor:

In the August 2001 issue of the Journal, a brief communication was published referring to the “ventricular muscle band.” Its basic premise was that the musculature of the right and left ventricles existed as a continuous band. In support of the concept, the authors cited several works previously published in Spanish and two investigations concerned only with the left ventricle. They failed to cite, however, or to discuss any of the previous investigations which, from Pettigrew onward, have shown that the musculature of the heart is arranged on the basis of a modified blood vessel, rather than in the fashion of a skeletal muscle with discrete origin and insertion. In particular, they neglected to discuss the recent elegant work by Jouk and colleagues, which expanded the concept of geodesic paths initially expounded by Streeter and Torrent-Guasp. Although supporting the basic concept of a geodesic arrangement, Jouk and his colleagues were unable to provide any evidence to support the concept of a muscular band encircling the cavities of both the right and the left ventricles. There is no question but that the anatomic orientation of the muscle fibers within the left ventricular wall is of potential surgical significance. The concept, however, should not be obfuscated by slavish acceptance of a hypothesis that has yet to be confirmed histologically.

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References

Reply to the Editor:

The helical concept of spatial myocardial fiber orientation has been described by Senac, Krehl, and a large spectrum of anatomists over the past 500 years. Three missing links have been the structural origin of the helical left ventricle and septum, why there was a right angle crossing of septal fibers, and the functional meaning of this anatomic configuration. Some background into anatomic contributions appeared in our recent manuscript, including the enormous contributions of Pettigrew, and recognition that Greenbaum and Anderson’s histologic observations further clarified the helical intraventricular formation. We agree that the origin of the heart is from a modified blood vessel.

The helical arrangement of the heart traverses the ventricular musculature and connects with actin, myosin, tropomyosin, and calcium. Efforts to link histology with structure require a pattern of dissection that does not violate underlying structure during unfolding of the helices to prevent disruption. Consequently, fibers cut in a dissected fetus by Jouk, in the orientation used can change the optical properties of birefringent material and not provide a pure optical method. These studies did not reconstruct the 3-dimensional fiber orientation, based on standard tomography methods, and they could not use this 2-dimensional dissection method to see the apex; the site of continuity of twist of car-
Cardiac fibers that links the septal crossing of descending and ascending segments. Some of this is recognized, as they indicate that the final model is conjectural and based on the examiners’ experience with dissection slide reading. Despite this, they see the helical patterns and thus add another coiled observation to cardiac anatomy.

We agree that function is related to the muscular formation of the wrapped tube, and we also recognize that joining of form and function can produce departure between the anatomist and pathologist that observe only the nonfunctional structure, and the physiologist and surgeon that must link function with underlying form. Our recent report of sonomicrometer verification of the functional components along the band, that correlate with magnetic resonance imaging studies of contraction, indicates we must take a new look into the form/function relationship, based on spatial orientation of the ventricular muscular band.

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Alternate explanation of the hypothermic prolonged induction of heat shock protein

To the Editor:

Motoyoshi and associates1 are to be congratulated for their provocative article, titled “Establishment of a Local Cooling Model Against Spinocord Ischemia Representing Prolonged Induction of Heat Shock Protein.” Their method seems reliable as a model to study heat shock protein (Hsp) problems unrelated to temperature, but it seems less reliable as a method to study spinal cord protection from which temperature cannot be unlinked. The explanation of how and why Hsp was induced consistently, in our opinion, is inadequate.

We do not contest that modest hypothermia exerts protective effects.2 However, to claim that it is better not to do anything before ischemia to maximize the beneficial effects of ischemic stress before inducing hypothermia is contradictory to conventional hypothermic and/or pharmacologic protection concepts/approaches.

Systemic hypothermia induced by surface cooling in rabbits was used in studies of spinal cord protection. We found that esophageal temperature measured 3 cm above the gastroesophageal junction before aortic clamping correlated with that of the spinal cord, within 0.1°C to 0.2°C, and therefore was usable as a surrogate site, but rectal temperature was not usable. An esophageal temperature of 29.4°C ± 0.07°C allowed full functional recovery within 5.5 hours of reperfusion after 60 minutes of ischemia in all rabbits, which yielded 6 to 6.2 minutes of ischemic protection for each 1°C, decreased by surface cooling after eucapnic ventilation, equivalent to pH-stat perfusion hypothermia, and rewarmed over a 90-minute period to 34°C to 35°C; however, only 0.5°C higher hypothermia uniformly failed.3,4 The proposed cooling method that results in spinal cord temperature with variability as large as 2°C is unacceptable for investigation or clinical use. Because it lacks other surrogate sites, without measuring actual spinal cord temperature, the exact role or degree of hypothermia required to achieve the reported effect could not be elucidated.

As illustrated in their Figure 1, the model was one of ischemia at 37°C to 36°C during the first 5 minutes and at 35°C to 33°C during the last 10 of the 15 minutes. Fifteen minutes of ischemia could theoretically be protective at 35.8°C induced by surface cooling.

Use of a normothermic group seems inappropriate to support their contention that local cooling is the key element. Instead, rabbits surface-cooled systemically to 36.1°C to 36.3°C before ischemia should have been used. Two rewarming rates should have been studied: a relatively fast rate, using conventional total body rewarming sources, and a rate similar to that of the locally cooled spinal cord. Although the authors did not mention how quickly the animals were rewarmed, in our opinion this information is needed to justify their conclusion, for the rate of rewarming could be the definitive and advantageous feature of local hypothermia.

Whether ischemia-injured neurons die by apoptosis or necrosis depends on the extent of depletion of high-energy-P3, apoptosis-necrosis is a continuum,5 necrosis occurring when depletion is maximal, but in either situation sustained Hsp70 synthesis cannot be supported, as in their normothermic rabbits.

Hsp70 is produced under stressful conditions for protection. If reduced stress was the mechanism of the prolonged induction of Hsp70, as the authors explain, the immunoreactivity should decrease, not increase. In our opinion, timely hypothermia spared enough high-energy-P to preserve the metabolic machinery that enabled continuing synthesis of sufficient Hsp70 for 2 days, but not enough to restore normal function immediately after reperfusion. Two days later, normal function was restored and the presence of Hsp70 was no longer required, thus disappearing by 7 days; apoptosis was averted, as in their hypothermic group.

The proposed strategy is applicable to only short ischemic periods. Ischemic periods lasting long enough to exhaust the high-energy-P store before implementation of systemic or local hypothermia commensurate to the ischemic time will induce a degree of metabolic machinery derangement that could not be protected by the then scarcely available Hsp70, resulting in irreversible injury as either apoptosis or necrosis. To protect the spinal cord during such long ischemic times necessitates implementation of hypothermic and/or pharmacologic preschismic protective means.

The question is not how the hypothermia was induced, but whether it was timely and

References