support his spurious concepts by misinterpretations of my own works.

Paul Lunkenheimer, MD
Experimental Thoracic and
Cardiovascular Surgery
University Clinic of Münster
Münster, Germany

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Reply to the Editor:

The role of my basic science lecture was to provide insight into the helix and the heart, with special emphasis on the interaction between nature, the heart, and the human being. Lunkenheimer's letter expands this focus. I credited his remarkable capacity to

add connective tissue spirals to our understanding of the scaffold of organized latticework for ventricular musculature. By intention, my lecture was not limited to the heart, but rather used my awe of the cardiac synthesis of nature's recipe for integration of spirals as a centerpiece to consider structure and rhythm. Claude Bernard,¹ a father of physiology, correctly defined anatomy of dead tissue to align itself with living physiologic function, rather than blindly relying on structure for deduction.

My admiration for the synthesis of spiral structures links closely to the helix described by Torrent-Guasp for the spatial orientation of the ascending and descending components of the helical apical loop and concurs with Lunkenheimer's elegant study of the connective tissue scaffold. Does Lunkenheimer think that the spiral muscular components that are desiccated during his method of transpneumonic spreading and fill this latticework are different from the weave that surrounds them?

Does this arrangement not simply add a new dimension to the hidden cardiac spiral harmony? His anatomic observations in the dead heart blend perfectly with the dual helical spirals of DNA or the planning code, the double helix of actin, myosin, tropomyosin, the spiral configuration of calcium within each cell, and the calcium coil described in my report. Furthermore, Taccardi and coworkers² added the spirals of the conduction system to this symphony. My conclusion is that Lunkenheimer's intrinsic connective tissue framework helps to more solidly integrate dimensions. Lunkenheimer's 1997 monograph³ and his letter show he does not accept the work of Torrent-Guasp.⁴ The literature allows scientists to report observations, make conclusions, and simultaneously let the readers make their own interpretations. My report links the reciprocal spirals defined in computed tomographic scan framework with the apical loop segments displayed by Torrent-Guasp. The *Journal's* readers are also free to render individual interpretations.

Torrent-Guasp's "unique band" may show how transverse fibers of the basal loop become oblique, through the myocardial fold or twist that separates the basal and apical loops (Figure E1, online only). This structural component may alter conventional thinking, because many people (including myself) thought that the heart constricts and dilates, rather than proper

shortening and lengthening in the living organism. This active contraction has overturned the teachings of Harvey, who studied dead anatomy and then presumed for the living. This is a vital lesson, because he was our teacher but not always perfect.

I suspect that recognized embryologic cardiac development may also be questioned, because the fold or turn in developing muscle mass may reflect a more rapid growth of the *ventricle* versus the *bulbus cordis* (Figure E2, online only).⁵ I subsequently learned that Goor and Lillehei⁶ (Figures E3 to E5, online only) and Maner⁷ (Figures E6 to E8, online only) have provided data supporting how the twist evolved. Perhaps anatomists and embryologists must rethink developmental concepts, just as magnetic resonance imaging findings of reciprocal twisting for ejection and suction impose needs for new understanding on clinicians.

Lunkenheimer shows no functional evidence to sustain his jump from anatomic structure to function, a basic premise in Bernard's distinction between anatomists and physiologists. Conversely, our sonomicrometer crystals within reciprocal spiral oblique positions of the living heart (Figure E9, online only) show functional correlation with Torrent-Guasp's anatomic spatial.⁸ Lunkenheimer addresses problems with artifactual dissection as Torrent-Guasp develops the muscular band for myocardial structure by indicating a destruction of the fibrous spatial netting. However, Lunkenheimer defends the correctness of the fiber strand peel-off method (SPOT).⁴ Can anyone follow each individual uncovered fiber, unfold it to show its helical configuration, and then further unravel the helical coil within each fiber to clarify the dual helical configuration of actin, myosin, or tropomyosin? Can someone successfully follow each collagen strand living within the helical connective tissue defined by this exciting and critical computed tomographic scan? Nature made endless coils within coils, and our future role is to discover and to slowly uncover different aspects of them, just as Torrent-Guasp and Lunkenheimer have done in their important studies.

Each letter to the Editor within a scientific journal provides the reader and the writer with the chance to interact about work and then let the reader reach his or her own conclusions. The truth is provided by the

data, not by the belief of the author. The *Journal* editor decides on publication of the questions. Lunkenheimer must advise us how he reached such a pivotal point on the scientific mountain to attain the astounding credibility that frees him to define scientific soundness.

My lecture about nature, the heart, and the human being summarizes my concept of progress. Claude Bernard¹ indicated that "to linger in the anatomic path alone, means lagging behind the progress of science," and that "believing that we can impose scientific principles without experimental verification is a relic of the Scholasticism of the Middle Ages." My basic science report included a description of Lycus of Macedonia, who as noted by Galen did not properly follow the Greek school of science but must be listened to, because he might provide a new and wonderful truth unknown to his predecessors. Lunkenheimer has done this with his unique and creative definition of the spatial collagen scaffolding. I am grateful to him for this contribution. We must now look beyond knowledge of only our focal interest and develop wisdom by synthesizing the interweaving spiral structure of the heart that unveils a cardiac aspect of the astounding spiral harmony of nature.

Gerald D. Buckberg, MD
UCLA Medical Center
Los Angeles, CA 90005

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Mitral valve in ischemic versus idiopathic dilated cardiomyopathy To the Editor:

The interesting data set reported on by Hueb and coworkers¹ in their article "Ventricular Remodeling and Mitral Valve Modifications in Dilated Cardiomyopathy: New Insights From Anatomic Study" has given us some new insights into the pathologic changes seen in ischemic and idiopathic dilated cardiomyopathy (DCM). The differential changes between these two conditions, however, were not fully explored in the article. As compared with the normal heart, the ratio of relative change in posterior leaflet insertion to anterior leaflet insertion is 3.4-fold in ischemic DCM, versus a 1.7-fold with idiopathic etiology. The impact of these pathologic states on the noncongruence of the fibrous annulus and anterior leaflet insertion is also interesting. One can crudely approximate a difference by a right triangle with the long side equal to the fibrous annulus and the hypotenuse equal to the anterior leaflet insertion. In ischemic DCM this distance is *decreased* 5.4%, whereas in the idiopathic DCM the distance is *increased* 11.1%. More detailed studies on the relative positional changes of the fibrous bodies with respect to the commissures will determine whether this is an asymmetric process (occurring more at the posterolateral commissure) or a symmetric one (occurring equally at both).

We also take this opportunity to strongly disagree with a conclusion by the authors, "... these anatomic data support the use of complete mitral annuloplasty techniques in DCM."¹ Conceptually, an "annuloplasty alone" repair for these disease states is performed by remodeling the annulus to effectively reduce the anteroposterior diameter to improve leaflet coaptation²⁻⁴ and not the lateromedial diameter, which does not seem to lead to mitral regurgitation in ischemic DCM

models.⁵ Although the anatomic data in this study demonstrate pathologic increases in both the anterior leaflet insertion and intertrigonal distances, it is not necessary to equate these changes with the need to fixate the anterior border of the annulus. For ischemic disease, the anterior insertion change is relatively minor when compared with posterior elongation, and for idiopathic changes the intertrigonal increase is accompanied by a 20.3% compensatory increase in the anterior leaflet insertion. We strongly advocate that appropriate valve reconstruction can be performed with an aggressive "trigone to trigone" annuloplasty with a device that fixes anteroposterior diameter. With this approach the annuloplasty device is anchored to the trigones, effectively reducing the anteroposterior diameter while still permitting normal dynamic changes along the base of the anterior mitral annulus.⁶

Eugene A. Grossi, MD

Ram Sharony, MD

Stephen B. Colvin, MD

Department of Surgery

New York University School of Medicine

New York, NY

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