Papillary Muscles Do Not Attach Directly to the Solid Heart Wall
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Background—The papillary muscles (PMs) play an important role in normal cardiac function, helping to prevent leakage through the AV valves during systole. The nature of their attachment to the heart wall can affect the understanding of their function. This attachment is conventionally portrayed as a direct connection of their bases to the solid portion of the heart wall. X-ray multidetector CT provides a new, noninvasive way to investigate this connection in vivo.

Methods and Results—With the use of x-ray multidetector CT with interactive 3D reconstruction, the bases of the PMs are seen to attach to the trabeculae carneae lining the ventricular wall rather than directly to the solid portion of the wall, as has been conventionally believed. This is true for both the left and right ventricular PMs.

Conclusions—This new picture of the geometry of the attachment of the PMs to the heart wall may have important implications for the understanding of their function, including the nature of the transmission of the forces between the PMs and the heart wall. (Circulation. 2004;109:3145-3148.)

Key Words: imaging ■ mechanics ■ physiology ■ tomography ■ ventricles

The papillary muscles (PMs) of the heart play an important role in cardiac function. All conventional anatomy and cardiology textbooks and articles depict the PMs as having a broad-based direct connection to the solid portion of the heart wall. Because the mechanical, vascular, and electrical connections of the PMs to the heart wall are through their bases, the nature of this connection can have important functional consequences. X-ray multidetector array CT (MDCT) provides a new imaging method for examining the attachment of the PMs in vivo.

The PMs are elongated, tapered muscles that originate from the inner wall of the ventricles and give rise to the chordae tendineae (connective tissue strands that attach to the edges of the AV valves) at their tips. When the ventricles contract in systole, the PMs also contract and help keep the AV valve leaflets from being inverted or leaking as pressure rises in the ventricular cavity. Dysfunction of the PMs, eg, as a result of ischemia or infarction, can adversely affect cardiac function through resulting AV valvular insufficiency, eg, in the setting of acute myocardial infarction. This attachment is conventionally portrayed as a direct connection of their bases to the solid portion of the heart wall at their ends and run over the inner surface of the ventricular cavity. The trabeculae carneae are present in both ventricles, although they are more prominent in the RV.

In standard cardiology and anatomy textbooks, the PMs are depicted as arising directly from the solid portion of the heart wall, with a broad base of attachment to the wall, much like the thumb emerging from the palm of the hand, and tapering to the origins of the chordae tendineae at their tips. However, conventional imaging methods have until now not had sufficient spatial resolution to study the nature of the PMs’ attachment to the wall in vivo. MDCT with contrast enhancement of the blood is a new tomographic imaging method that permits high-resolution 3D imaging of the ventricular cavity in vivo, with clear visualization of the PMs and the trabeculae carneae lining the cavity at different phases of the cardiac cycle. MDCT was used to visualize the PMs and their relation to the solid and trabecular portions of the heart wall.

Methods

Patient Selection
The 3D image data acquired on 25 consecutive unselected subjects, imaged for possible coronary artery disease using MDCT with...
standard methods, were retrospectively examined under an Institutional Review Board–approved protocol to evaluate the nature of the attachment of the PMs to the heart wall. Because this was a retrospective study, informed consent was not obtained directly from the subjects.

**Imaging Methods**

A 16-row MDCT system (Sensation 16, Siemens Medical Solutions) was used for imaging the subjects. Patients received β-blockers to lower their heart rate, preferably to ≤60 bpm. Contrast enhancement was obtained with 140 mL radiographic contrast agent [Visipaque 320 (iodixanol), Amersham Health] infused intravenously at 4 mL/s; image acquisition was timed to coincide with peak enhancement of blood in the heart. The CT image acquisition/reconstruction was gated to diastole (at an effective time of 350 or 400 ms before the QRS complex of the ECG) to minimize motion effects on the images and to capture the heart in a relatively relaxed state; images were also reconstructed at other effective times in the cardiac cycle. The duration of image acquisition was short enough that the volume of the heart could be covered in a single breathhold. The effective duration of each image set within the cardiac cycle was ~120 ms. Images were reconstructed as 3D data sets with isotropic spatial resolution of 0.75 mm. Image analysis was carried out through interactive 3D reformattting of the image data using the CT manufacturer’s standard image processing workstation and software. Reformatted image planes with an effective thickness of 0.75 mm were interactively chosen for reconstruction of the PMs.

**Results**

Image sets reconstructed at a range of effective cardiac cycle phases were examined. Images reconstructed near mid to late diastole were best for delineating the PM attachments; near end systole, image blurring and collapse of the blood-filled spaces between the trabeculae carneae made it difficult to see the attachment of the PMs to the trabeculae. In all cases examined, the base of the PMs did not directly contact or join the solid portion of the heart wall. Rather, in all cases, the base of the PMs ended in contact with the network of trabeculae carneae lining the ventricular cavity, above the actual surface of the solid portion of the heart wall. This was true for both LV and RV PMs. Representative images from 1 subject demonstrating this relationship are shown in Figure 1. The absence of PM attachments to solid wall can be seen with contiguous planes of reconstruction through the bases (Figure 2). Image quality was insufficient to assess the PM arterial supply.

**Discussion**

MDCT with 3D reconstruction clearly demonstrates the nature of the attachment of the PMs to the heart wall. The base of the PMs joins to the network of trabeculae carneae lining the ventricular cavity rather than directly to the solid portion of the heart wall, as previously assumed.

**Previous Studies**

There has been only limited discussion of the structure of the PM base in previous articles; clinical interest has focused primarily on the PM blood supply and on variations in the overall location, number, and attachments of the chordae tendineae to variable head shapes. Existence of a “border” between the PMs and the wall in the canine heart has been noted but without further discussion (a diagram in that article shows the standard representation of a broad-based contact of the base of the PMs with the wall); that study also noted an
abrupt change in fiber angle between the solid wall and the PMs. The PMs have been described as “deeply undercut” but apparently without a full appreciation of nature of the attachment of their bases to the trabeculae carneae rather than directly to the solid heart wall. A study of 100 autopsy hearts described approximately half of the specimens as having “equally sessile and intramural” PMs, with the remainder divided between “mostly intramural” (with or without “tip anchored”) and “mostly sessile” but again without a clear description of the attachment of their bases to the wall. Thus, the observation reported here that the PMs attach to the heart wall at the trabeculae carneae rather than directly to the solid portion of the wall appears to be novel.

It may seem surprising that the correct relationship of the PMs to the heart wall has not been appreciated previously. However, several factors have probably contributed to this. Anatomic and pathological studies are usually performed on dead hearts in a strongly contracted state, effectively collapsing the spaces between the trabeculae beneath the base of the PMs. Their base is also hidden from direct view in the usual visual inspection of the ventricular interior, eg, at surgery. In radiographic projection imaging, eg, contrast ventriculography, images of overlying structures can obscure the nature of the attachment of the PM bases. Other tomographic imaging techniques generally have lower spatial resolution than the submillimeter isotropic resolution achievable with current MDCT, making it more difficult to appreciate trabecular structures beneath the PM base. In cardiac MRI, for example, in-plane pixel resolution is typically 1 to 2 mm and slice thickness is ≥5 mm, whereas in echocardiography, resolution along the beam direction is typically ≤1 mm, but resolution across the beam is somewhat worse. Technical improvements will undoubtedly also make this relationship clear with other imaging methods. Finally, the prejudice of expecting to see the “conventional” version of the anatomy at the base of the PMs has undoubtedly led observers to fail to appreciate its true nature.

**Functional Implications**

We can speculate on some functional implications of this new understanding of the relationship of the PMs to the heart wall. Having a broad meshlike rather than pillarlike attachment to the wall may reduce the stress concentration in the wall near the PM bases. On the other hand, stress concentrations at points of attachment between the PMs and the trabeculae may make the base more vulnerable to rupture at those points. Having a broader effective base and multiple points of attachment for the PMs may provide redundancy and thus some protection against full mechanical failure. (Issues related to the effect of leaving the chordae tendineae intact during mitral valve surgery are essentially independent of the nature of the attachment of the PM bases.) Similarly, having the blood supply to the PMs enter from a broader effective base may help provide more potential for collateral perfusion redundancy and thus some protection against ischemia. In addition, a slight delay after initiation of contraction by the ventricular wall before contraction by the PMs, as has been observed experimentally in some studies, might enable the AV valve leaflets to close more freely before tension builds up in the PMs. The small additional conduction time required for the activation wave front to reach the PMs, imposed by a somewhat more circuitous path through the trabeculae rather than directly from the wall, could provide such a short delay.

**Acknowledgments**

Drs Jill Jacobs and James Slater supervised acquisition of the MDCT images.
References


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Circulation. published online June 14, 2004:

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