wavefronts (3), resulting in sustained ventricular tachycardia. These two mechanisms may be important in the generation and maintenance of sustained ventricular tachycardia near the papillary muscle.

Clearly separated Purkinje potentials are characteristic findings for endocardial recordings near the papillary muscle (2,3). Successful radiofrequency ablation at these sites (1) suggests that the reentrant wavefronts responsible for idiopathic left ventricular tachycardia are adjacent to or are located within the papillary muscle.

Peng-Sheng Chen, MD, FACC  
Cedars-Sinai Medical Center, Room 5342  
8700 Beverly Boulevard  
Los Angeles, California 90048-1865  
E-mail: chenps@csmc.edu  

Hrayr S. Karagueuzian, PhD, FACC  
Young-Hoon Kim, MD, FACC

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REPLY
We are grateful for the opportunity to respond to the comments by Dr. Chen et al. concerning our recent article in the Journal (1). I was very impressed by the study of Joyner et al. (2), that reported on longitudinal dissociation of ventricular muscle and Purkinje signals in isolated papillary muscle of a dog. According to Figure 1B in their article, pacing from a Purkinje strand inserted into the apex of the papillary muscle resulted in apex to base Purkinje activation, which then excited the ventricular muscle via the Purkinje-muscle junction at the base of the papillary muscle with propagation of the excitation from the apex to the base papillary muscle. Their Figure 1B is similar to our Figure 2A, as Dr. Chen et al. have suggested. According to the results of Joyner et al. (2), our diastolic potential (P1) appeared to be the result of a signal from the local ventricular muscle of the papillary muscle, and our presystolic potential (P2) that from the local Purkinje activation of the papillary muscle. However, there are several differences between our study and the study by Joyner et al. First, while the Purkinje-muscle junction in their study was located at the base of the papillary muscle, the Purkinje-muscle transmission occurred at the infero-apical septum in our study. The position of the octapolar electrode catheter shown in our Figure 1 differed from the site of the posterior papillary muscle. The activation sequences of the Purkinje potential during sinus rhythm may also differ. Because Purkinje fibers enter the anterior and posterior papillary muscles through the trabecular carneae, the activation sequence of the Purkinje potential in the papillary muscle during sinus rhythm must be from the apex to the base. However, our P2 was recorded earlier from the proximal than the distal electrodes during sinus rhythm. We think that P1 is the potential from the trabecular carneae. Trabecular carneae form ridges, bridges and small papillary muscles. Lai et al. (3) proposed a false tendon or interlacing Purkinje fiber as a link between the slow conduction tissue and left posterior fascicle. Gallagher et al. (4) and Thakur et al. (5) have also implied that the left ventricular muscular band may be the anatomic substrate for idiopathic left ventricular tachycardia. However, Lin et al. (6) have shown that the left ventricular muscular band is not the specific arrhythmogenic substrate for this tachycardia. However, trabecular carneae or small papillary muscles cannot be detected by echocardiography. Nevertheless, longitudinal dissociation of the ventricular muscle and Purkinje signals from the papillary muscle and trabecular carneae, and the presence of the Purkinje-muscle junction, are important in considering the reentry circuit of verapamil-sensitive idiopathic left ventricular tachycardia.

Akihiko Nogami, MD  
Division of Cardiology  
Yokohama Rosai General Hospital  
3211 Kozukue, Kohoku  
Yokohama, Kanagawa 222-0036 Japan  
E-mail: akihiko-ind@umin.ac.jp

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The Enigma of Primary Pulmonary Hypertension

Riley et al. (1) observed that exercise increased heart rates and decreased oxygen saturations of arterial hemoglobin abnormally in patients who had primary pulmonary hypertension. They stated that this occurs in subjects with circulatory disease or deconditioning. They did not refer to the studies showing that fast heart rates and desaturation of arterial hemoglobin also occur in well-trained athletes (2,3). They did not measure cardiac outputs in their patients to allow them to calculate stroke volumes, but they concluded that low stroke volume, resulting from pulmonary...
We found that nitric oxide (NO) reduced right ventricular systolic pressure (RVSP) at rest in our patients with PPH, but failed to alter exercise responses. Several factors may have been responsible: 1) Nitric oxide may not have improved cardiac output, despite causing a fall in pulmonary vascular resistance; NO may adversely affect the positive inotropic response to beta-adrenergic stimulation in left ventricular dysfunction (2) resulting in reduced cardiac contractility. 2) Hemodynamic improvement present at rest may not have been sustained during exercise; our study was noninvasive and we could not be sure that NO improved either RVSP or cardiac output during exercise. 3) As proposed by Dr. Krohn, peripheral factors may have limited the benefit of any rise in cardiac output with NO. In patients with chronic left ventricular failure, Wilson et al. (3,4) found an increase in cardiac output with the acute administration of hydralazine or isosorbide dinitrate during submaximal exercise or dobutamine during maximal exercise. However, none of these drugs improved total oxygen consumption, oxygen uptake across the exercising leg or venous lactate. This paradox was attributed to a failure of “nutritive” flow to increase in the exercising muscle. Even though peripheral oxygen extraction is high in patients with PPH (5), it is possible that the peripheral circulation may not have been able to distribute appropriately any additional acute increase in cardiac output caused by NO inhalation.

We agree that the study of neurohumoral mechanisms in PPH may prove fruitful. Endothelin is known to be elevated in patients with PPH (6) and may be important in promoting vascular remodeling. At present it is unclear whether abnormalities of other vasoactive hormones also occur. However, even if neurohumoral abnormalities occur under conditions of circulatory stress, the matter of discerning cause and effect will remain.

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REPLY

We thank Dr. Krohn for his thoughtful comments. The mechanism for the desaturation seen in some elite athletes is likely to be related to the high cardiac output leaving insufficient time for equilibration of oxygen tensions between the alveolus and alveolar capillaries. Oxyhemoglobin desaturation in response to maximal exercise is a very unusual finding in normal adults performing exercise near sea level (1). Cardiac output at peak exercise is low in patients with primary pulmonary hypertension (PPH). Desaturation is unlikely to be due to the same mechanism as that occurring in elite athletes. Arterial oxyhemoglobin desaturation is not a feature of circulatory diseases or deconditioning.