Myocardial Viability Assessment with FDG Imaging: Comparison of PET, SPECT, and Gamma-Camera Coincidence Detection

TO THE EDITOR: In the November 1999 issue of *The Journal of Nuclear Medicine*, Hasegawa et al. (*1*) reported a direct comparison among PET, SPECT, and dual-head gamma-camera coincidence detection imaging (DCD) to assess myocardial viability with FDG.

We would like to address three issues about the design and methods of the study.

1. The choice of performing the DCD tomography in 32 steps over 360° penalizes this modality by a factor of 2 in the examination duration, compared with 32 steps over 180° with ultra-high-energy general purpose collimators for SPECT. Furthermore, the gamma camera used in the study (Vertex Plus MCD; ADAC Laboratories, Milpitas, CA) is equipped with a 5/8-in. (15.9-mm) thick crystal, which means that the detection efficiency for the photopeak at 511 keV is approximately 21% for a single detector (*2*). Thus, the coincidence detection efficiency for DCD is only 21% × 21% = 4.41% (*2*), which would require more steps to obtain a similar count per image ratio and to allow for a more objective comparison between the 2 modalities. Finally, the 128 × 128-matrix reconstructed data obtained from DCD should have been reduced to a 64 × 64 matrix to obtain the same slice thickness as the SPECT images.

2. The DCD acquisition started 150 min after SPECT acquisition, occurring with 38.56% of the FDG activity that was initially available for SPECT imaging, because of FDG decay (half-life = 109.7 min).

3. The delayed DCD acquisition (210 min after injection and 150 min after SPECT) may be reasonable because of the risk of detector saturation by high counting rate, but it assumes that myocardial trapping of FDG is irreversible. Recently, Herrero et al. (*3*) demonstrated, on a canine model of myocardial glucose usage during hyperinsulinemic–euglycemic clamp, that a reversible myocardial trapping of FDG occurs within the first hour after tracer injection. This delay may therefore alter the myocardial distribution of FDG and consequently the DCD detection in an unpredictable manner.

All these factors acted to penalize DCD and could have influenced the final result of the study: a poor agreement of DCD imaging with PET and a better performance of SPECT. A randomized order for the first acquisition modality (DCD or SPECT) after PET imaging and an optimized DCD acquisition (duration and angle selection) would have been more adequate to sustain the published conclusion.

REFERENCES

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Imaging of Bacterial Infections with ^{99m}Tc-Labeled Human Neutrophil Peptide-1

TO THE EDITOR: Although new techniques for imaging bacterial infections are needed, Welling et al.'s (*1*) criticisms of ^{99m}Tc-ciprofloxacin (Infecton) imaging lack substance and proof. Below, we explore, point by point, issues discussed by Welling et al.

"Low binding affinity of ^{99m}Tc-ciprofloxacin to bacteria": Where are the data to support this? Welling et al. (*1*) cited our article published in *The Lancet* (*2*), but the binding affinity of ^{99m}Tc-ciprofloxacin to bacteria is not discussed in our article. On the contrary, sites of bacterial infection can be imaged up to 24 h after the injection of 370 MBq (10 mCi) ^{99m}Tc ciprofloxacin. This agent shows specific binding to DNA gyrase, an essential enzyme for bacterial division, which is the basis of the therapeutic success of ciprofloxacin. Welling et al. (*1*) failed to mention the greater specificity of Infecton imaging for bacterial infection than radiolabeled white cells (*2*).

"The risk of emerging antibiotic resistant organisms": This is unlikely to be a significant clinical problem because only a tracer amount of ciprofloxacin is present in Infecton (i.e., 2 mg ciprofloxacin, which, being 1/100 to 1/200 of the intravenous therapeutic dose of 200–400 mg, is extremely small). Indeed, no such problems have been encountered in our multicenter study comprising 879 patients worldwide, and no drug toxicity has been observed in that study (*3*).

Reference 5 cited by Welling et al. (*1*) does not address the risk of emerging antibiotic-resistant microorganisms. The cited chapter gives an excellent account of how microbes, particularly those that are intracellular pathogens, have developed strategies to overcome or evade mechanisms that are used by macrophages and polymorphs to kill microorganisms. The chapter includes a discussion about the resistance to defensins (antibacterial peptides, a member of which is human neutrophil peptide-1 [HNP-1]) in *Salmonella typhimurium*. Similarly, other articles have been published that deal with microbial resistance–susceptibility to defensins. It is naive, therefore, to suggest that defensin-resistant bacteria may not emerge easily. The host–microbe interaction is not static but constantly evolving, with each trying to outdo the other. Successful pathogens, many of which are intracellular, have developed systems that enable them to resist and overcome host defense mechanisms, including antibacterial peptides. It would be interest-