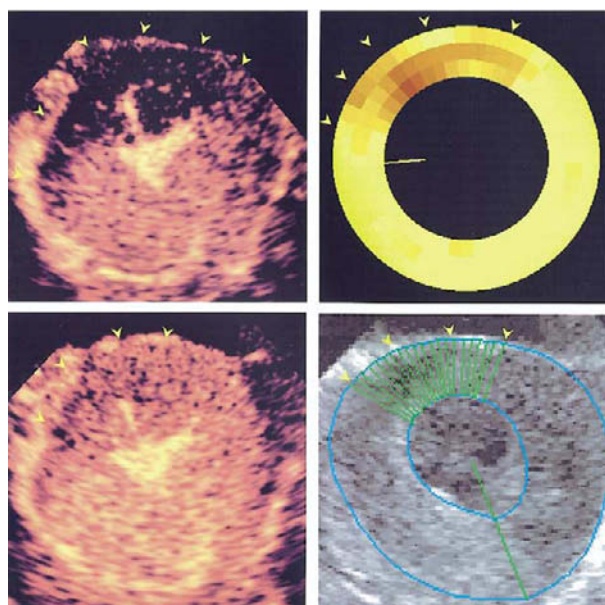


# CORRECTIONS

## CORRECTION

Figure 11 in “Echocardiographic insights into regional flow-function relationships in coronary artery disease” (Kaul S. J Nucl Cardiol 2005;12:216-26) was printed upside down. The corrected figure is reprinted below.



**Figure 11.** Data from a dog with a noncritical LAD stenosis at peak dobutamine dose, where MBF is increased. *Left panels*, Perfusion defects (*arrows*) early (*upper panel*) and late (*lower panel*) after microbubble destruction. The first shows the perfusion territory of the artery, and the second shows how much of it is filled with collaterals. *Right panels*, The *upper panel* shows the radiolabeled microsphere-derived hypoperfused zone (*arrows*), whereas the *lower panel* shows the extent of abnormal WT (defined by *chords* and *arrows*). Note the similarities between the MCE and microsphere data in the *upper panels* and the MCE and WT data in the *lower panels*. (Reprinted from reference 36 with permission.)

## CORRECTION

The tables in “Targeting the vulnerable plaque: The evolving role of nuclear imaging” (J Nucl Cardiol 2005;12:234-46) were printed incorrectly. The corrected tables are as follows.

**Table 1.** Imaging modalities used for assessment of human atherosclerotic plaque

Imaging modality	% Stenosis	Wall	Lipid	Fibrous cap	Thrombus	Macrophage/inflammation	Ca <sup>2+</sup>	Apoptosis
Invasive								
X-ray angiography	*†‡§	—	—	—	(*)	—	*†‡§	—
IVUS	*†‡§	*†‡§	(*†‡§)	(*†‡§)	(*†‡§)	—	*†‡§	—
OCT	*	*	*	*	*	—	*	—
Thermography	—	—	—	—	—	*	—	—
Noninvasive								
US	†‡§	†‡§	—	—	—	—	†‡§	—
MRI	(*)†‡§	*†‡§	†	†	†‡§	(†)	(*)†‡§	—
EBCT	—	—	—	—	—	—	*†‡§	—
MSCT	(*)†‡§	*†‡§	†‡§	†	—	—	*†‡§	—
Nuclear	—	—	†§	—	†§	†‡	—	†

Parentheses indicate that imaging is less than satisfactory.

IVUS, intravascular ultrasound; OCT, optical coherence tomography; US, ultrasound; MRI, magnetic resonance imaging; EBCT, electron beam computed tomography; MSCT, multi-slice computed tomography.

\*Coronary. †Carotid. ‡Aorta. §Ileo-femoral.

**Table 2.** Radionuclide tracer compounds used to image atherosclerosis

Target mechanism	Target cell/molecule	Tracer	Animal/human	Ex-vivo histologic correlation	Successful in-vivo imaging	Notes
Lipid accumulation	LDL	123-I LDL <sup>35</sup>	Human carotid	✓	✓	Long plasma half-life of tracer necessitates late imaging
		99m-Tc LDL <sup>37</sup>	Human carotid, ileo-femoral	✓	?	Tracer uptake seen in only 4 of 17 patients, but good histologic correlation found between uptake and plaque instability
		125-I LDL <sup>36</sup>	NZW rabbit aorta	✓	N/A	Good correlation with foam cell infiltration
	Ox-LDL	99m-Tc ox-LDL <sup>38</sup>	Human carotid	✓	✓	Rapid plasma clearance (c.f. native LDL tracers)
		125-I MDA2 <sup>39</sup>	Apo E -/- mouse WHHL rabbit	✓	N/A	Also capable of tracking changes in foam cell number <sup>40</sup>
		125-I IK17 <sup>42</sup>	ApoE -/- mouse	✓	N/A	In-vitro staining of human plaques, IK17 localizes to lipid core <sup>42</sup>
		apoB	125-I SP-4 <sup>43</sup>	NZW rabbit aorta	✓	N/A
	123-I SP-4 <sup>44</sup>		WHHL rabbit aorta	N/A	✓	
Macrophage infiltration	Autologous monocytes	111-In monocyte <sup>46</sup>	Human	N/A	✓	Identified 40% of lesions, no histologic correlation
		125-I MCP-1 <sup>51</sup>	NZW rabbit aorta	✓	N/A	Excellent correlation with macrophage number, fast plasma clearance
	Ama	131-I Ama-MoAb <sup>52</sup>	WHHL rabbit aorta	✓	X	Slow plasma clearance, unsuccessful gamma imaging
	GLUT	18-F FDG <sup>65,67-70</sup>	WHHL + NZW aorta Human carotid and aorta	✓	✓	PET tracer, good correlation between tracer uptake and macrophage number, uptake in humans unstable > stable plaque

**Table 2.** Continued

Target mechanism	Target cell/molecule	Tracer	Animal/human	Ex-vivo histologic correlation	Successful in-vivo imaging	Notes
	MMP	<sup>123</sup> I HO-CGS 27023A <sup>55</sup>	apoE -/- mouse carotid	✓	✓	Significant increase in uptake in lesioned carotid (c.f. sham and control), rapid plasma clearance
Apoptosis	PS	<sup>99m</sup> Tc Annexin-V <sup>73,74</sup>	NZW rabbit aorta Human carotid	✓	✓	Colocalisation with apoptotic macrophages, uptake in humans correlates with vulnerable histologic features
Coagulation	Fibrin	<sup>99m</sup> Tc T2G1s Fab <sup>76</sup>	Canine carotid	N/A	✓	Uptake ratio lesion: control = 2:1 in vivo and 4:1 ex vivo
	D-dimer	<sup>99m</sup> Tc TRF1 <sup>80</sup>	Human carotid	N/A	X	Uptake seen in only 5 of 8 patients, no histologic correlate
Platelets	Autologous platelets	<sup>111</sup> In autologous platelets <sup>83,84</sup>	Human carotid	N/A	?	Inconsistent results between studies, may be of use in tracking effects of antiplatelets <sup>81,82</sup>
	GPIIb/IIIa	<sup>99m</sup> Tc P748 <sup>86</sup>	Canine carotid	N/A	✓	
		<sup>99m</sup> Tc P280 <sup>87</sup>	Human carotid	N/A	?	Uptake in 11 of 18 patients, no histologic correlate
		<sup>99m</sup> Tc DMP-444 <sup>88</sup>	Canine carotid	✓	✓	Tracer uptake correlated with platelet number/thrombus weight

*LDL*, Low density lipoprotein; *ox-LDL*, oxidized low density lipoprotein; *NZW*, New Zealand white; *N/A*, not attempted; *Apo E -/-*, apolipoprotein E null; *MDA*, malondialdehyde; *WHHL*, Watanabe heritable hyperlipidaemic; *SP*, synthetic peptide; *CCR*, chemokine receptor; *MCP*, monocyte chemotactic protein; *Ama-MoAb*, amino malonic acid monoclonal antibody; [<sup>18</sup>F]FDG, 18-fluorodeoxyglucose; *GLUT*, glucose transporter protein; *MMP*, matrix metalloproteinase; *PS*, phosphatidyl serine.