

Speckle tracking: distinction of physiologic from pathologic LVH?

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Over the past years, echocardiography and magnetic resonance imaging (MRI) have become important imaging modalities in patients with a broad spectrum of cardiomyopathies [1–20]. Both imaging modalities have been shown to play a pivotal role in the accurate evaluation of left ventricular function particularly in patients with ischemic heart disease and different forms of cardiomyopathy [21–36]. These methods are needed to accurately identify and characterize patients with various manifestations of left ventricular hypertrophy (LVH) [37–49]. The important question should be resolved whether training-induced LVH in athletes is a physiological rather than a pathophysiological phenomenon [50–57]. In a meta-analysis using MRI, involving 59 studies and 1,451 athletes (both endurance-trained and strength-trained athletes), it was reported that the athlete's heart demonstrated normal systolic and diastolic cardiac function, implying that training-induced LVH in athletes is predominantly a physiological phenomenon [58]. With respect to echocardiography, two-dimensional strain has become a novel method to measure strain from standard two-

dimensional echocardiographic images by speckle tracking. Speckle tracking offers the advantage of being less angle-dependent and more reproducible than conventional Doppler-derived strain [59–61]. This echocardiographic imaging technique allows quantification of global and regional myocardial deformation on the basis of tracking of acoustic markers from frame-to-frame.

In the current issue of the *International Journal of Cardiovascular Imaging*, Butz et al. [62] questioned whether two-dimensional strain assessed by speckle tracking was useful as an additional tool in differentiating pathologic from physiologic LVH in high-level athletes. The objective of the study was to identify and characterize global and regional function abnormalities in patients with pathological left ventricular hypertrophy (LVH) caused by non-obstructive hypertrophic cardiomyopathy (HCM), in high-level athletes, and in healthy controls. The authors consecutively studied 53 subjects consisting of 15 patients with HCM and 20 competitive top-level athletes. A control group of 18 sedentary normal subjects was studied by standard echocardiography according to standard guidelines. Global longitudinal strain and regional peak systolic strain were assessed by two-dimensional strain in the apical four-chamber view. It turned out that all components of strain were significantly reduced in patients with HCM when compared to athletes and control subjects. There was no significant difference between the strain values of the athletes and the control group, but in some segments the strain

Editorial comment to the article of Butz et al.
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values of the control group were significantly higher than those in the athletes. A cut-off value of global longitudinal strain less than -10% for the diagnosis of HCM resulted in a sensitivity of 80% and a specificity of 95% . The combination of tissue Doppler imaging (averaged S' , E') and global longitudinal strain cut-off values for the detection of pathologic LVH in HCM demonstrated a sensitivity of 100% and a specificity of 95% . The authors concluded that speckle tracking is a new simple and rapid method to measure global longitudinal strain and peak systolic strain as components of systolic strain.

This novel and very interesting technique offers a unique approach to quantify global as well as regional systolic dysfunction, and might be used as new additional tool for the differentiation between physiologic and pathologic LVH. The present study is one of the first that used the combined and comprehensive tissue Doppler imaging analysis of systolic and early diastolic velocities of the lateral and septal mitral annulus as well as two-dimensional strain analysis for the differentiation between pathologic and physiologic LVH. The main results of this intriguing study show that global strain is significantly reduced in patients with HCM and that strain measurements can therefore be used for differentiation between pathologic and physiologic LVH. Especially, the combination of tissue Doppler imaging and two-dimensional strain assessment allows the differentiation of pathologic and physiologic LVH with acceptable sensitivity and specificity. This differentiation is extremely important in terms of clinical well-being, treatment and prognosis. In physiologic hypertrophy, such as in athletes, LVH reveals normal circumferential, radial, and longitudinal profiles possibly indicating that myocardial strain imaging might be useful as additional echocardiographic modality for the differentiation between athlete's heart and HCM. In pathologic hypertrophy, LVH appears to be associated with subendocardial fibrosis, which might be a potential mechanism for the failure of the hypertrophied myocardium in due time. In another study by the same group [63], peak systolic longitudinal strain of the basal septum and the opposite lateral wall was measured in addition to standard echocardiography in 88 consecutive patients with obstructive HCM who underwent a septal ablation procedure. Regional myocardial deformation was assessed quantitatively by speckle tracking. During a 12-month observation

period, no patient had a severe adverse event. Reduction of left ventricular afterload by elimination of the outflow gradient following a successful septal ablation procedure resulted in improvement of systolic lateral longitudinal function in patients with obstructive HCM.

To summarize, two-dimensional strain analysis by echocardiography is a new simple, rapid, and reproducible method to measure systolic strain. Echocardiographic speckle tracking allows accurate quantitative assessment of regional myocardial deformation. As a result, the speckle tracking technique can be used as additional tool for (1) a comprehensive cardiac evaluation in subjects with physiologic hypertrophy versus pathologic hypertrophy, and (2) an appropriate assessment of the effects of therapy in patients with pathologic LVH.

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References

1. Ten Cate FJ (2009) Cardiomyopathies: a revolution in molecular medicine and cardiac imaging. *Neth Heart J* 17:456–457
2. Kleijn SA, Kamp O (2009) Clinical application of three-dimensional echocardiography: past, present and future. *Neth Heart J* 17:18–24
3. Kamp O (2008) History of echocardiography in The Netherlands: 30 years of education and clinical applications. *Neth Heart J* 16:16–20
4. Nemes A, Geleijnse ML, van Geuns RJ et al (2008) Dobutamine stress MRI versus threedimensional contrast echocardiography: it's all black and white. *Neth Heart J* 16:217–218
5. Cramer MM, De Boeck BW (2007) Three-dimensional echocardiography and left bundle branch block: prime time in cardiology. *Neth Heart J* 15:87–88
6. Schuijf JD, Bax JJ, van der Wall EE (2007) Anatomical and functional imaging techniques: basically similar or fundamentally different? *Neth Heart J* 15:43–44
7. van der Wall, Vliegen HW, de Roos A, Bruschke AV (1995) Magnetic resonance imaging in coronary artery disease. *Circulation* 92:2723–2739
8. van der Wall EE, Siebelink HM, Bax JJ (2010) Evaluation of hypertrophic cardiomyopathy: new horizons for CMR? *Neth Heart J* 18:116–117
9. Holman ER, Buller VG, de Roos A et al (1997) Detection and quantification of dysfunctional myocardium by magnetic resonance imaging. A new three-dimensional method

- for quantitative wall-thickening analysis. *Circulation* 95: 924–931
10. van Ruge FP, van der Wall EE, Brusckhe AV (1992) New developments in pharmacologic stress imaging. *Am Heart J* 124:468–485
 11. van Ruge FP, Holman ER, van der Wall EE et al (1993) Quantitation of global and regional left ventricular function by cine magnetic resonance imaging during dobutamine stress in normal human subjects. *Eur Heart J* 14: 456–463
 12. van Ruge FP, van der Wall EE, Spanjersberg SJ et al (1994) Magnetic resonance imaging during dobutamine stress for detection and localization of coronary artery disease Quantitative wall motion analysis using a modification of the centerline method. *Circulation* 90:127–138
 13. Vliegen HW, Doornbos J, de Roos A, Jukema JW, Bekedam MA, van der Wall EE (1997) Value of fast gradient echo magnetic resonance angiography as an adjunct to coronary arteriography in detecting and confirming the course of clinically significant coronary artery anomalies. *Am J Cardiol* 79:773–776
 14. Hoogendoorn LI, Pattynama PM, Buis B, van der Geest RJ, van der Wall EE, de Roos A (1995) Noninvasive evaluation of aortocoronary bypass grafts with magnetic resonance flow mapping. *Am J Cardiol* 75:845–848
 15. Groenink M, Lohuis TA, Tijssen JG et al (1999) Survival and complication free survival in Marfan's syndrome: implications of current guidelines. *Heart* 82:499–504
 16. Oosterhof T, van Straten A, Vliegen HW et al (2007) Preoperative thresholds for pulmonary valve replacement in patients with corrected tetralogy of Fallot using cardiovascular magnetic resonance. *Circulation* 116:545–551
 17. van der Geest RJ, de Roos A, van der Wall EE, Reiber JH (1997) Quantitative analysis of cardiovascular MR images. *Int J Card Imaging* 13:247–258
 18. van der Geest RJ, Niezen RA, van der Wall EE, de Roos A, Reiber JH (1998) Automated measurement of volume flow in the ascending aorta using MR velocity maps: evaluation of inter- and intraobserver variability in healthy volunteers. *J Comput Assist Tomogr* 22:904–911
 19. van der Laarse A, Kerkhof PL, Vermeer F et al (1988) Relation between infarct size and left ventricular performance assessed in patients with first acute myocardial infarction randomized to intracoronary thrombolytic therapy or to conventional treatment. *Am J Cardiol* 61:1–7
 20. van der Wall EE, Vliegen HW, de Roos A, Brusckhe AV (1995) Magnetic resonance imaging in coronary artery disease. *Circulation* 92:2723–2739
 21. Bavelaar-Croon CD, Kayser HW, van der Wall EE et al (2000) Left ventricular function: correlation of quantitative gated SPECT and MR imaging over a wide range of values. *Radiology* 217:572–575
 22. Bleeker GB, SchaliJ MJ, Boersma E et al (2007) Relative merits of M-mode echocardiography and tissue Doppler imaging for prediction of response to cardiac resynchronization therapy in patients with heart failure secondary to ischemic or idiopathic dilated cardiomyopathy. *Am J Cardiol* 99:68–74
 23. Bleeker GB, Bax JJ, Fung JW et al (2006) Clinical versus echocardiographic parameters to assess response to cardiac resynchronization therapy. *Am J Cardiol* 97:260–263
 24. Bleeker GB, Holman ER, Steendijk P et al (2006) Cardiac resynchronization therapy in patients with a narrow QRS complex. *J Am Coll Cardiol* 48:2243–2250
 25. Posma JL, Blanksma PK, van der Wall EE, Hamer HP, Mooyaart EL, Lie KI (1996) Assessment of quantitative hypertrophy scores in hypertrophic cardiomyopathy: magnetic resonance imaging versus echocardiography. *Am Heart J* 132:1020–1027
 26. Vehmeijer JT, Christiaans I, van Langen IM et al (2009) Risk stratification for sudden cardiac death in hypertrophic cardiomyopathy: Dutch cardiologists and the care of mutation carriers. *Neth Heart J* 17:464–469
 27. Christiaans I, NannenberG EA, Dooijes D et al (2010) Founder mutations in hypertrophic cardiomyopathy patients in The Netherlands. *Neth Heart J* 18:248–254
 28. ICIN working group on Hereditary Heart Diseases (2010) Genetic diagnostics and genetic counselling in Hypertrophic Cardiomyopathy (HCM). *Neth Heart J* 18:144–159
 29. Pluim BM, Lamb HJ, Kayser HW, Leuijes F et al (1998) Functional and metabolic evaluation of the athlete's heart by magnetic resonance imaging and dobutamine stress magnetic resonance spectroscopy. *Circulation* 97:666–67229
 30. Pluim BM, Beyerbacht HP, Chin JC et al (1997) Comparison of echocardiography with magnetic resonance imaging in the assessment of the athlete's heart. *Eur Heart J* 18:1505–1513
 31. Pluim BM, Chin JC, De Roos A et al (1996) Cardiac anatomy, function and metabolism in elite cyclists assessed by magnetic resonance imaging and spectroscopy. *Eur Heart J* 17:1271–1278
 32. Schroeder J, Peterschroeder A, Vaske B et al (2009) Cardiac volumetry in patients with heart failure and reduced ejection fraction: a comparative study correlating multislice computed tomography and magnetic resonance tomography. Reasons for intermodal disagreement. *Clin Res Cardiol* 98:739–747
 33. Germans T, Nijveldt R, Brouwer WP et al (2010) The role of cardiac magnetic resonance imaging in differentiating the underlying causes of left ventricular hypertrophy. *Neth Heart J* 18:135–143
 34. Hoogsteen J, Hoogveen A, Schaffers H, Wijn PF, van der Wall EE (2003) Left atrial and ventricular dimensions in highly trained cyclists. *Int J Cardiovasc Imaging* 19: 211–21734
 35. Muhl C, Dassen WR, Kuipers H (2008) Cardiac remodeling: concentric versus eccentric hypertrophy in strength and endurance athletes. *Neth Heart J* 16:129–133
 36. Nassenstein K, Breuckmann F, Lehmann N et al (2009) Left ventricular volumes and mass in marathon runners and their association with cardiovascular risk factors. *Int J Cardiovasc Imaging* 25:71–79
 37. van der Wall EE, den Hollander W, Heidendal GA, Westera G, Majid PA, Roos JP (1981) Dynamic myocardial scintigraphy with 123I-labeled free fatty acids in patients with myocardial infarction. *Eur J Nucl Med* 6:383–389
 38. Braun S, van der Wall EE, Emanuelsson S, Kobrin I (1996) Effects of a new calcium antagonist, mibefradil (Ro 40–5967), on silent ischemia in patients with stable chronic angina pectoris: a multicenter placebo-controlled study. The mibefradil international study group. *J Am Coll Cardiol* 27:317–322

39. de Roos A, Matheijssen NA, Doornbos J, van Dijkman PR, van Ruge PR, van der Wall EE (1991) Myocardial infarct sizing and assessment of reperfusion by magnetic resonance imaging: a review. *Int J Card Imaging* 7:133–138
40. van der Wall EE, van Dijkman PR, de Roos A et al (1990) Diagnostic significance of gadolinium-DTPA (diethylenetriamine penta-acetic acid) enhanced magnetic resonance imaging in thrombolytic treatment for acute myocardial infarction: its potential in assessing reperfusion. *Br Heart J* 63:12–17
41. van Ruge FP, Boreel JJ, van der Wall EE et al (1991) Cardiac first-pass and myocardial perfusion in normal subjects assessed by sub-second Gd-DTPA enhanced MR imaging. *J Comput Assist Tomogr* 15:959–965
42. Holman ER, van Jonbergen HP, van Dijkman PR, van der Laarse A, de Roos A, van der Wall EE (1993) Comparison of magnetic resonance imaging studies with enzymatic indexes of myocardial necrosis for quantification of myocardial infarct size. *Am J Cardiol* 71:1036–1040
43. Buller VG, van der Geest RJ, Kool MD, van der Wall EE, de Roos A, Reiber JH (1997) Assessment of regional left ventricular wall parameters from short axis magnetic resonance imaging using a three-dimensional extension to the improved centerline method. *Invest Radiol* 32:529–539
44. Bakx AL, van der Wall EE, Braun S, Emanuelsson H, Brusckhe AV, Kobrin I (1995) Effects of the new calcium antagonist mibefradil (Ro 40–5967) on exercise duration in patients with chronic stable angina pectoris: a multicenter, placebo controlled study. *Ro 40–5967 International Study Group. Am Heart J* 130:748–757
45. Portegies MC, Schmitt R, Kraaij CJ et al (1991) Lack of negative inotropic effects of the new calcium antagonist Ro 40–5967 in patients with stable angina pectoris. *J Cardiovasc Pharmacol* 18:746–751
46. van der Wall EE, Heidendal GA, den Hollander W, Westera G, Roos JP (1980) I-123 labeled hexadecenoic acid in comparison with thallium-201 for myocardial imaging in coronary heart disease. A preliminary study. *Eur J Nucl Med* 5:401–405
47. de Nooijer R, Verkleij CJ, von der Thüsen JH et al (2006) Lesional overexpression of matrix metalloproteinase-9 promotes intraplaque hemorrhage in advanced lesions but not at earlier stages of atherogenesis. *Arterioscler Thromb Vasc Biol* 26:340–346
48. Nijveldt R, Beek AM, Hirsch A et al (2008) ‘No-reflow’ after acute myocardial infarction: direct visualisation of microvascular obstruction by gadolinium enhanced CMR. *Neth Heart J* 16:179–181
49. Oemrawsingh PV, Mintz GS, Schalij MJ, Zwilerman AH, Jukema JW, van der Wall EE (2003) Intravascular ultrasound guidance improves angiographic and clinical outcome of stent implantation for long coronary artery stenoses: final results of a randomized comparison with angiographic guidance (TULIP Study). *Circulation* 107: 62–67
50. Ypenburg C, Roes SD, Bleeker GB et al (2007) Effect of total scar burden on contrast-enhanced magnetic resonance imaging on response to cardiac resynchronization therapy. *Am J Cardiol* 99:657–660
51. Ypenburg C, Sieders A, Bleeker GB et al (2007) Myocardial contractile reserve predicts improvement in left ventricular function after cardiac resynchronization therapy. *Am Heart J* 154:1160–1165
52. Ypenburg C, van der Wall EE, Schalij MJ, Bax JJ (2008) Imaging in cardiac resynchronisation therapy. *Neth Heart J* 16:S36–S40
53. Ypenburg C, Westenberg JJ, Bleeker GB et al (2008) Noninvasive imaging in cardiac resynchronization therapy—part 1: selection of patients. *Pacing Clin Electrophysiol* 31:1475–1499
54. Ypenburg C, Schalij MJ, Bleeker GB et al (2007) Impact of viability and scar tissue on response to cardiac resynchronization therapy in ischaemic heart failure patients. *Eur Heart J* 28:33–41
55. Torn M, Bollen WL, van der Meer FJ, van der Wall EE, Rosendaal FR (2005) Risks of oral anticoagulant therapy with increasing age. *Arch Intern Med* 165:1527–1532
56. van der Laan A, Hirsch A, Nijveldt R et al (2008) Bone marrow cell therapy after acute myocardial infarction: the HEBE trial in perspective, first results. *Neth Heart J* 16: 436–439
57. Germans T, Wilde AA, van Echteld CJ, Kamp O, Pinto YM, van Rossum AC (2007) Structural abnormalities of the left ventricle in hypertrophic cardiomyopathy mutation carriers detectable before the development of hypertrophy. *Neth Heart J* 15:161–163
58. Pluim BM, Zwilerman AH, van der Laarse A, van der Wall EE (2000) The athlete’s heart: a meta-analysis of cardiac structure and function. *Circulation* 101:336–344
59. Tops LF, Suffoletto MS, Bleeker GB et al (2007) Speckle tracking radial strain reveals left ventricular dyssynchrony in patients with permanent right ventricular pacing. *J Am Coll Cardiol* 50:1180–1188
60. Tops LF, Schalij MJ, Holman ER, van Erven L, van der Wall EE, Bax JJ (2006) Right ventricular pacing can induce ventricular dyssynchrony in patients with atrial fibrillation after atrioventricular node ablation. *J Am Coll Cardiol* 48:1642–1648
61. Roes SD, Mollema SA, Lamb HJ, van der Wall EE, de Roos A, Bax JJ (2009) Validation of echocardiographic two-dimensional speckle tracking longitudinal strain imaging for viability assessment in patients with chronic ischemic left ventricular dysfunction and comparison with contrast-enhanced magnetic resonance imaging. *Am J Cardiol* 104:312–317
62. Butz T, van Buuren F, Mellwig KP et al (2010) Two-dimensional strain analysis of the global and regional myocardial function for the differentiation of pathologic and physiologic left ventricular hypertrophy: a study in athletes and in patients with hypertrophic cardiomyopathy. *Int J Cardiovasc Imaging*. doi:10.1007/s10554-010-9665-5
63. Faber L, Prinz C, Welge D et al (2010) Peak systolic longitudinal strain of the lateral left ventricular wall improves after septal ablation for symptomatic hypertrophic obstructive cardiomyopathy: a follow-up study using speckle tracking echocardiography. *Int J Cardiovasc Imaging*. doi:10.1007/s10554-010-9678-0