

41. Corrado D, Basso C, Pavei A, Michieli P, Schiavon M, Thiene G. Trends in sudden cardiovascular death in young competitive athletes after implementation of a preparticipation screening program. *JAMA* 2006;**296**: 1593–1601.
42. Noheria A, Teodorescu C, Uy-Evanado A, Reinier K, Mariani R, Gunson K, Jui J, Chugh SS. Distinctive profile of sudden cardiac arrest in middle-aged vs. older adults: a community-based study. *Int J Cardiol* 2013 May 16, doi:pii: S0167-5273(13)00890-5. 10.1016/j.ijcard.2013.04.207. Published online ahead of print.
43. Drezner JA. Detect, manage, inform: a paradigm shift in the care of athletes with cardiac disorders? *Br J Sports Med* 2013;**47**:4–5.
44. Drezner JA, Levine BD, Vetter VL. Reframing the debate: screening athletes to prevent sudden cardiac death. *Heart Rhythm* 2013;**10**:454–455.
45. Chugh SS. Early identification of risk factors for sudden cardiac death. *Nat Rev Cardiol* 2010;**7**:318–326.
46. Albert CM, Mittleman MA, Chae CU, Lee IM, Hennekens CH, Manson JE. Triggering of sudden death from cardiac causes by vigorous exertion. *N Engl J Med* 2000;**343**: 1355–1361.
47. Siscovick DS, Weiss NS, Fletcher RH, Lasky T. The incidence of primary cardiac arrest during vigorous exercise. *N Engl J Med* 1984;**311**:874–877.
48. Corrado D, Basso C, Rizzoli G, Schiavon M, Thiene G. Does sports activity enhance the risk of sudden death in adolescents and young adults? *J Am Coll Cardiol* 2003;**42**:1959–1963.

## CARDIOVASCULAR FLASHLIGHT

doi:10.1093/eurheartj/eh442

Online publish-ahead-of-print 18 October 2013

### In vivo myofibre architecture in the systemic right ventricle

Jack Harmer<sup>1†\*</sup>, Kuberan Pushparajah<sup>1†</sup>, Nicolas Toussaint<sup>1</sup>, Christian T. Stoeck<sup>2</sup>, Rachel Chan<sup>3</sup>, David Atkinson<sup>3</sup>, Reza Razavi<sup>1</sup>, and Sebastian Kozerke<sup>1,2</sup>

<sup>1</sup>Division of Imaging Sciences and Biomedical Engineering, King's College London, London, UK; <sup>2</sup>Institute for Biomedical Engineering, University and ETH Zurich, Switzerland; and <sup>3</sup>Centre for Medical Imaging, University College London, London, UK

\* Corresponding author. Division of Imaging Sciences, The Rayne Institute, 4th Floor, Lambeth Wing, St Thomas' Hospital, London SE1 7EH, UK.

Tel: +44 207-188 5441; Fax: +44 207 188 5442, Email: jack.harmer@kcl.ac.uk

† Equal Contributors.

Heart failure in the systemic right ventricle (RV) is a common pathway in end-stage disease in patients affected with congenital heart disease. Using state-of-the-art magnetic resonance (MR) diffusion acquisition schemes, we present the first *in vivo* diffusion tensor imaging (DTI) data of the beating heart acquired in an adult with a systemic RV following an atrial switch procedure for transposition of the great arteries. Magnetic resonance-based DTI acquisitions provide information about the predominant direction of structures within each voxel of the acquired image. These aggregates, which are often interpreted as fibres, appear as coherent orientational structures throughout the myocardium. Knowledge of cardiac myocyte architecture has the potential to transform our understanding of cardiac function and the mechanisms behind heart failure. Until recently, direct visualization of myofibre architecture has been limited to *ex vivo* specimens due to cardiac motion. However, recent advances in MRI now allow for robust DTI of the beating heart and can provide *in vivo* knowledge of myofibre architecture. In the data presented here, diffusion tensors are shown across multiple slices and are colour coded to indicate helix angle (Panels A–E). Full 3D reconstructions across the volume of the heart are also shown. Helix angle distributions indicate a predominance of circumferential fibres across the entire healthy LV and in the anterior and inferior segments of the systemic RV. However, in the lateral wall of the systemic RV, helix angles are skewed towards negative values. This indicates a predominance of longitudinal and oblique fibres with a clockwise helix orientation and is likely brought about to adaptation of the RV to systemic pressure and load.

This work was supported by the UK EPSRC (EP/I018700/1), Adult Congenital Heart Disease Service Guy's and St Thomas' NHS foundation Trust, and the National Institute for Health Research (NIHR) Biomedical Research Centre at Guy's and St Thomas' NHS Foundation Trust and King's College London. The views expressed are those of the authors and not necessarily those of the NHS, the NIHR or the Department of Health.

