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doi:10.1016/j.jtcvs.2011.11.039

GUIDING THE MOLECULAR DIAGNOSIS OF HYPERTROPHIC CARDIOMYOPATHY

To the Editor:

With interest, we read the recent guideline for hypertrophic cardiomyopathy (HCM) and highly appreciate the useful clinical information.¹ HCM is a pathophysiologically heterogeneous condition. We agree with the authors that there is comprehensive evidence that mutations in 8 genes encoding proteins of, or associated with, the sarcomere can cause HCM. These are, as stated in chapter 6 of the guideline, the genes encoding β myosin heavy chain, myosin binding protein C, troponin T, troponin I, α -tropomyosin, actin, regulatory light chain, and essential light chain. However, only about 40% to 60% of HCM cases can be attributed to these 8 genes.^{2,3}

More recently, a ninth gene was convincingly linked to HCM after a genome-wide linkage analysis. Mutations in *CSRP3* encoding muscle LIM protein were shown to cosegregate with HCM in several families in whom gene mutations in the 8 incriminated sarcomeric proteins had been previously excluded.⁴ All these mutations affected highly conserved residues and were not found in the control genomes. Linkage analysis with a highly significant 5.9 logarithm of the odds score in a single large family provided additional genetic evidence. Physicians and geneticists unaware of the mutation status ensured an unbiased attribution of disease

status and an unbiased genome-wide linkage analysis in that family.⁵ Furthermore, functional data supported the genetic results. Cardiomyocyte disarray was observed in *CSRP3* mutation-positive cardiac tissue from patients with HCM. The findings indicate that *CSRP3* mutations cause "true" HCM and not merely a HCM phenocopy.

Although *CSRP3* mutations in patients with HCM are not common (<5%), we believe that there is convincing, scientific evidence that the gene should be included in the list of confirmed HCM disease genes. *TPM1* and *ACTC* (encoding α -tropomyosin and actin) mutations occur at a frequency not much different than *CSRP3* mutations. The prevalence of HCM disease genes is also related to ethnicity. Such patterns require active surveillance of disease genes and *CSRP3* should be included. To learn more about the ethnic spectrum of disease genes and specific founder mutations we propose including all well-established disease genes into scientific genetic screening analyses. Muscle LIM protein is not confined solely to sarcomeric localizations but can be localized to the sarcolemma and to the cell nucleus. Mechanistically, there is much to be learned about this protein, its gene, and their contributions to HCM. We therefore wish to draw attention to *CSRP3*.

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doi:10.1016/j.jtcvs.2012.01.062

RE: SIMPLIFIED PERFUSION STRATEGY FOR REMOVING RETROPERITONEAL TUMORS WITH EXTENSIVE CAVOATRIAL INVOLVEMENT

To the Editor:

The authors compare beating heart cardiopulmonary bypass (CPB) and hypothermic circulatory arrest (HCA) for removing retroperitoneal tumors with extensive cavoatrial involvement.¹ They included in their series retroperitoneal renal tumors with level III-IV tumor thrombus

extension; 56 (39%) and 88 (61%) underwent CPB and HCA, respectively. Since 1997, we have used liver transplant techniques for mobilization of the liver off the inferior vena cava (piggyback mobilization) to aid in the resection of renal cell carcinoma with level III tumor thrombus.² None of the patients required a thoracoabdominal approach, CBP, HCA or venovenous bypass. Just recently, we published our technique step by step for treating renal cell carcinoma with tumor thrombus, extending our experience to 56 patients with level III and 12 with level IV tumor thrombus, and only 3 patients with level IV required CPB.³ This experience has now been extended to 70 patients with level III thrombus. We also reported our experience removing an adherent⁴ and nonadherent⁵ level IV (intra-atrial thrombus) tumor

thrombus without a thoracoabdominal approach, median sternotomy, or CPB. It is important to describe other experiences of treating renal cell carcinoma with level III-IV cavoatrial involvement in which CPB or HCA can be avoided for most cases, thereby simplifying the operation even more.

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doi:10.1016/j.jtcvs.2011.11.040

Notices of Correction

Re: Akins CW. The ethical dilemma of Thoracic Surgery recertification. *J Thorac Cardiovasc Surg.* 2012;143:521-2.

In the above-mentioned article, the abbreviation for Maintenance of Certification was incorrect throughout. The correct abbreviation is MOC.

Re: Hillis LD, Smith PK, Anderson JL, Bittl JA, Bridges CR, Byrne JG, Cigarroa JE, DiSesa VJ, Hiratzka LF, Hutter AM Jr, Jessen ME, Keeley EC, Lahey SJ, Lange RA, London MJ, Mack MJ, Patel MR, Puskas JD, Sabik JF, Selnes O, Shahian DM, Trost JC, Winniford MD. 2011 ACCF/AHA guideline for coronary artery bypass graft surgery: Executive summary: A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Thorac Cardiovasc Surg.* 2012;143:4-34.

In Section 4.10.3, Central Nervous System Monitoring, in Class IIb recommendation #1, references 449-51 should be replaced by the following references, which should be added to the References section:

316a. Avidan MS, Zhang L, Burnside BA, et al. Anesthesia awareness and the bispectral index. *N Engl J Med.* 2008;358:1097-108.

316b. Hemmerling TM, Olivier JF, Basile F, et al. Bispectral index as an indicator of cerebral hypoperfusion during off-pump coronary artery bypass grafting. *Anesth Analg.* 2005;100:354-6.

316c. Myles PS, Leslie K, McNeil J, et al. Bispectral index monitoring to prevent awareness during anaesthesia: the B-Aware randomised controlled trial. *Lancet.* 2004;363:1757-63.

References 449-451 should be deleted from the References section. These changes are for concordance with the full-length guideline article (*J Am Coll Cardiol.* 2011;58:e123-210; doi:10.1016/j.jacc.2011.08.009).