CORRESPONDENCE

Keeping Cool in the Operating Room

To the Editor:

In the operating room, the ambient temperature might be uncomfortably warm for the surgical team, for several reasons. During off-pump surgery, the temperature might purposely be kept high. Without use of the heart-lung machine, it is important to avoid overcooling the patient, because rewarming can be difficult and take time. In tropical countries during summer, the atmospheric temperature might be so high that effective indoor cooling cannot be achieved. In some countries, power outages occur frequently, and uninterrupted electrical power is allocated to crucial infrastructure. Meanwhile, the surgical team is gowned and needs to stay focused.

We have devised a simple way to remain cool. A ClearView® Blower/Mister (Medtronic, Inc.; Minneapolis, Minn) is used to direct a jet of oxygen toward the surgeon, at a flow rate of only 2 to 3 L/min. This device—a fixture in our operating room for 10 years now—is kept outside the sterile field, at the anesthesiologist's side.

Apart from the cooling, we think that the high oxygen concentration sharpens our senses and improves our surgical focus.

Harinder Singh Bedi, MCh, FIACS,
Arun Gupta, MD,
Department of Cardiovascular & Thoracic Surgery; and
Nandini Kaul Bedi, MCh,
Paediatric Surgery;
Christian Medical College & Hospital,
Ludhiana, India

http://dx.doi.org/10.14503/THIJ-14-4770

Consider a Mitochondrial Disorder when Left Ventricular Hypertrabeculation/ Noncompaction is Associated with Renal Cysts

To the Editor:

We appreciated the article by Katukuri and colleagues[1] about the 37-year-old man who had left ventricular hypertrabeculation/noncompaction (LVHT) associated with polycystic kidney disease (PCKD). We wish to present some considerations that the authors might have been unable to discuss, given the length criteria for Images in Cardiovascular Medicine publications in the *Journal*.

The authors speculate that the PCKD was caused by mutations in the patient's *PCKD1* or *PCKD2* genes. Polycystic kidney disease can arise from causes other than *PCKD* mutations.[2] Renal cysts might be a phenotypic manifestation of a mitochondrial disorder (MID). Because LVHT has been associated with MIDs, this type of metabolic defect needs to be excluded. It would be enlightening to know whether the patient had cerebral, ocular, otologic, endocrine, gastrointestinal, dermal, or blood cell abnormalities. In addition, was an MID identified in the family history, beyond PCKD and unspecified cardiac disease in the father? When family members of an affected patient are systematically screened genetically and echocardiographically, LVHT can often be found.[3,4]

We wish to point out that LVHT is not necessarily a rare abnormality of cardiac development; the condition can also be acquired.[5] Did the patient have previous echocardiograms that could be reviewed for the presence or absence of LVHT?

We respectfully disagree that cardiac magnetic resonance (CMR) is the gold standard for the diagnosis of LVHT: the condition might be missed on CMR, and the criteria to diagnose LVHT on CMR are vague.

Neuromuscular disorders (NMDs) have also been associated with LVHT.[6] Answers to these questions would be helpful: did the patient or his relatives undergo investigation for NMDs? Did the patient report symptoms indicative of an NMD? Was his creatine kinase or serum lactate level elevated? Did he have a history of sequelae during or after previous general anesthesia?

To our knowledge, at least 9 cases with LVHT and PCKD have been reported.

Overall, this interesting report would have profited from more information with regard to an MID or NMD, because the pathogenesis of LVHT is unclear.

Josef Finsterer, MD, PhD,
Krankenanstalt Rudolfstiftung,
Vienna, Austria; and
Sinda Zarrouk-Mahjoub, PhD,
Laboratory of Biochemistry,
UR "Human Nutrition and Metabolic Disorders,"
Faculty of Medicine,
Monastir, Tunisia

References

1. Katukuri NP, Finger J, Vaitkevicius P, Riba A, Spears JR. Association of left ventricular noncompaction with polycystic kidney disease as shown by cardiac magnetic resonance imaging. Tex Heart Inst J 2014;41(4):449-52.
2. Gurgey A, Ozalp I, Rotig A, Coskun T, Tekinalp G, Erdem G, et al. A case of Pearson syndrome associated with multiple renal cysts. Pediatr Nephrol 1996;10(5):637-8.

3. Finsterer J, Stollberger C, Blazek G, Sehnal E. Familial left ventricular hypertrabeculation (noncompaction) is myopathic. Int J Cardiol 2013;164(3):312-7.
4. Dubourg B, D'Here B, de Vecchi C, Caudron J, Savoure A, Stepowski D, et al. Incidental diagnosis of a familial left ventricular noncompaction on a chest CT angiography. Diagn Interv Imaging 2014;95(1):91-3.
5. Finsterer J, Stollberger C, Schubert B. Acquired left ventricular noncompaction as a cardiac manifestation of neuromuscular disorders. Scand Cardiovasc J 2008;42(1):25-30.
6. Stollberger C, Finsterer J, Blazek G. Left ventricular hypertrabeculation/noncompaction and association with additional cardiac abnormalities and neuromuscular disorders. Am J Cardiol 2002;90(8):899-902.

http://dx.doi.org/10.14503/THIJ-14-4666

This letter was referred to Dr. Neelima Katukuri, who responds as follows:

We thank Drs. Finsterer and Zarrouk-Mahjoub[1] for their interest in our work.[2] In response to their questions, we offer the following:

- We thought about performing genetic testing; however, the patient could not afford it, and his insurance company did not approve further evaluation of any kind.
- The patient's father had died long before and no details were remembered about him. The patient had no siblings or children.
- The patient had no symptoms and had not undergone previous evaluations.
- We agree that several cases have been described in which left ventricular hypertrabeculation/noncompaction (LVHT) was not present at birth or on previous echocardiograms but developed during a patient's lifetime (acquired LVHT).
- The patient reported no symptoms indicative of a neuromuscular disorder and reported no history of complications during or after general anesthesia.
- We checked the patient's creatine kinase and serum lactate levels, and these were within normal limits.
- Because the pathogenesis of LVHT is unclear, any effort to clarify the cause of this enigmatic morphologic abnormality appears to be justified. However, in this case, approval to perform just the magnetic resonance imaging required multiple letters and telephone calls to the patient's insurance company.

Neelima Penugonda Katukuri, MD,
Department of Cardiology,
Oakwood Hospital and Medical Center,
Dearborn, Michigan

References

1. Finsterer J, Zarrouk-Mahjoub S. Consider a mitochondrial disorder when left ventricular hypertrabeculation/noncompaction is associated with renal cysts. Tex Heart Inst J 2014;41(6):677-8.
2. Katukuri NP, Finger J, Vaiktevicius P, Riba A, Spears JR. Association of left ventricular noncompaction with polycystic kidney disease as shown by cardiac magnetic resonance imaging. Tex Heart Inst J 2014;41(4):449-52.

http://dx.doi.org/10.14503/THIJ-14-4869

Letters to the Editor should be no longer than 2 double-spaced typewritten pages and should generally contain no more than 6 references. They should be signed, with the expectation that the letters will be published if appropriate. The right to edit all correspondence in accordance with Journal *style is reserved by the editors.*