www.leica-microsystems.com/products/novocastra-reagents/primary-antibodies/ <http://bsd.leica-microsystems.com/pdfs/products/5d3-ce.pdf> and <http://www.ncl.ac.uk/business/success/spinout.php/Medical+Devices/Novocastra+Laboratories+Ltd>. Accessed March 28, 2010.

3. Becton Dickinson. Anti-cytokeratin (CAM 5.2) reagent for in vitro diagnostic use 50 tests per vial. Catalog no. 349205, 9/97, 23-3190-01. Material safety data sheet, acc. to ISO/DIS 11014. 1997. Available at: http://www.bdbiosciences.com/external_files/is/doc/tds/Datasheets_RUO/live/web_enabled/23-1336-09-347653-347204-349205-DS-ruo.pdf. Accessed March 28, 2010.
4. Han CP, Hsu JD, Koo CL, Yang SF. Antibody to cytokeratin (CK8/CK18) is not derived from CAM5.2 clone, and anticytokeratin CAM5.2 (Becton Dickinson) is not synonymous with the antibody (CK8/CK18). *Hum Pathol*. 2010;41:616-7.

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Reply to the Editor:

We thank our colleagues Cheng and associates for their commentary with regard to our recently published article, "The Sentinel Node Concept in Adenocarcinomas of the Distal Esophagus and Gastroesophageal Junction."¹ In this study, we used the widely recognized immunohistochemical assay in which antibodies against epithelial-cell proteins are used to detect small clusters of tumor cells in histologically node-negative lymph nodes of patients with esophageal adenocarcinoma. Yao and Han claim that the monoclonal antibody against cytokeratin 8 and 18 (clone NCL-5D3) that we used in the present study should not be mistaken with the CAM5.2 antibody (clone CAM5.2) that appears to be specific for cytokeratin 7 and 8. In our article, we stated that the used antibody CAM 5.2 is specific for intracellular cytokeratin 8 and 18, referring to the original paper of Makin, Bobrow, and Bodmer,² in which it is claimed that (at that time newly developed) antibody CAM5.2 identifies the lower molecular weight cytokeratins (cytokeratin 8, 18 and 19). Other studies that investigated the presence and relevance of micrometastases or isolated tumor cells by using antibody CAM5.2 have also referred to this article.³ However, over the last years, companies producing these antibodies

have changed their products and further investigated the corresponding specificity in reactivity against certain cytokeratins, as pointed out by Yao and Han. Therefore, their statement that antibody CAM5.2 is not specific for intracellular cytokeratin 8 and 18 is correct. Nevertheless, this finding does not affect the conclusions of our study. The presence of cytokeratin 8 and/or 18 (which are both expressed in esophageal adenocarcinoma⁴) as detected with the antibody used in our study (clone NCL-5D3) indicates epithelial cell deposits in lymphoid tissue. Therefore, these cytokeratin deposits will still imply the presence of micrometastatic disease.

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References

1. Grotenhuis BA, Wijnhoven BP, van Marion R, van Dekken H, Hop WC, Tilanus HW, et al. The sentinel node concept in adenocarcinomas of the distal esophagus and gastroesophageal junction. *J Thorac Cardiovasc Surg*. 2009;138:608-12.
2. Makin CA, Bobrow LG, Bodmer WF. Monoclonal antibody to cytokeratin for use in routine histopathology. *J Clin Pathol*. 1984;37:975-83.
3. Buskens CJ, ten Kate FJ, Obertop H, Izbicki JR, van Lanschot JJ. Analysis of micrometastatic disease in histologically negative lymph nodes of patients with adenocarcinoma of the distal esophagus or gastric cardia. *Dis Esophagus*. 2008;21:488-95.
4. Moll R, Franke WW, Schiller DL, Geiger B, Krepler R. The catalog of human cytokeratins: patterns of expression in normal epithelia, tumors and cultured cells. *Cell*. 1982;31:11-24.

doi:10.1016/j.jtcvs.2010.03.004

ANGIOGENESIS AND SURGICAL OR ENDOVASCULAR ENHANCEMENT OF NONCORONARY COLLATERAL CIRCULATION: A NEW RESEARCH FIELD

To the Editor:

We read with great interest the article by Atluri and colleagues,¹ and we

congratulate them for their experiments with myocardial perfusion and contractility improvement using a laser transmyocardial approach. We agree with the authors that the problem with myocardial revascularization in patients who might not benefit from conventional procedures still demands investigative efforts. This was the genesis of a research program we began at Laval University in Quebec and that we are going to start in Europe at Université Paris Sud. This program is aimed at the enhancement of noncoronary collateral circulation (NCCC). Our animal models are dogs (Canada) and pigs (France).

NCCC is a topic that is virtually ignored, with very few publications in existence. This is surprising, considering that it is not rare to find evidence of NCCC, such as during coronary bypass surgery when arterial blood flow comes from the coronary ostia during valve replacement surgery or from the incised coronary artery, despite adequate aortic crossclamping and venting. NCCC consists of a network of small channels that come from mediastinal, bronchial, and pericardial vessels and that enter the heart through the pericardial reflections surrounding the pulmonary and systemic veins, as well as through the vasa vasorum of the aorta and pulmonary artery leading to and from the myocardium. Our studies are premised on the belief that this network is bigger than generally thought and that it might play a role, if adequately enhanced, as an alternative means of myocardial blood supply. We also believe that the normal ventricular function sometimes seen in patients with occlusion of the 3 major coronary vessels can be evidence of myocardial nourishment related to such a collateral source. Previous studies² have shown that vascular connections exist between the internal thoracic arteries (ITAs) and myocardium and that surgical bilateral ligation of the ITAs creates a local hypertensive status, increasing the perfusion pressure within the channels leading to the heart.³