(LGE) typical of myocardial infarction and a non–coronary artery disease pattern of LGE, could be distinguished.[2] Analysis of the event-free survival rate suggested that the presence of LGE may also be relevant for future coronary events in marathon runners.[2] Unfortunately, event-free survival data on the control group were not obtained.[3] Thus, any significant difference in the event-free survival rate in runners compared to sedentary subjects cannot be excluded.[3] Moreover, the follow-up period was short, and it is impossible to predict the shapes of these survival curves 5 and 10 years in the future.[3]

At this time, there is not enough evidence to implicate marathon running in the development of a dangerous substrate for coronary events.[3] Nonetheless, there is evidence of delayed enhancement suggestive of the true breakdown of the myocytes,[2] contradicting the results of Mousavi et al.[1] Breuckmann et al[2] performed delayed-enhancement CMR imaging in 102 runners, and 12% had true breakdown of the myocytes. CMR imaging was performed in only 14 participants by Mousavi et al.[1] Consequently, Mousavi et al[1] may have missed delayed enhancement because of the small number of participants in their study. However, given the runners' smoking histories and ages,[2] one cannot be certain that marathon running contributes directly to LGE; rather, it may uncover underlying coronary artery disease.[3]

At present, it can be concluded neither that the increase in cardiac troponin after marathon running is likely due to the cytosolic release of the biomarker[1] nor that the release of cardiac troponin represents the true breakdown of the myocytes,[2] which has been proposed either as pathognomonic of cardiac necrosis or might reflect part of a remodeling process.[4,5]

Arnold Koller, PhD
Wolfgang Schobersberger, MD
Innsbruck, Austria
2 June 2009

1. Mousavi N, Czarnecki A, Kumar K, Fallah-Rad N, Lytwyn M, Han S-Y, Francis A, Walker JR, Kirkpatrick IDC, Neilan TG, Sharma S, Jassal DS. Relation of biomarkers and cardiac magnetic resonance imaging after marathon running. *Am J Cardiol* 2009;103:1467–1472.
2. Breuckmann F, Möhlenkamp S, Nassenstein K, Lehmann N, Ladd S, Schmermund A, Sievers B, Schlosser T, Jöckel K-H, Heusch G, Erbel R, Barkhausen J. Myocardial late gadolinium enhancement: prevalence, pattern, and prognostic relevance in marathon runners. *Radiology* 2009;251:50–57.
3. Yared K, Wood MJ. Is marathon running hazardous to your cardiovascular health? The jury is still out. *Radiology* 2009;251:1–5.
4. Koller A, Schobersberger W. Post-exercise release of cardiac troponins. *J Am Coll Cardiol* 2009;53:1341.
5. Bearzi C, Rota M, Hosoda T, Tillmanns J, Nascimbene A, De Angelis A, Yasuzawa-Amano S, Trofimova I, Siggins RW, LeCapitaine N, Cascapera S, Beltrami AP, D'Alessandro DA, Quaini F, Urbanek K, Michler RE, Bolli R, Kajstura J, Leri A, Anversa P. Human cardiac stem cells. *Proc Natl Acad Sci U S A* 2007;104:14068–14073.

doi:10.1016/j.amjcard.2009.06.001

Sudden Death After Alcohol Septal Ablation in Hypertrophic Cardiomyopathy

Noseworthy et al[1] addressed the risk for sudden cardiac death (SCD) and ventricular arrhythmias after alcohol septal ablation (ASA) for drug-refractory obstructive hypertrophic cardiomyopathy. During a mean follow-up period of 5.0 ± 2.3 years, the investigators examined the rates of SCD in 89 patients treated with ASA and recorded the annual event rate of ventricular arrhythmias in a subgroup of 42 patients with implantable cardioverter-defibrillator (ICDs) or permanent pacemakers. Noseworthy et al[1] concluded that ventricular tachycardia or ventricular fibrillation, cardiac arrest, and/or appropriate ICD discharges affected annually 2.8% of the low-risk population of their study and 13.4% of the high-risk population presenting with ≥1 clinical risk factors for SCD. It is very interesting that 10 patients received procedure-related ICDs (in 7 patients, ICDs were implanted, and in 3, permanent pacemakers were upgraded to ICDs after ASA), meaning that a total of 19 patients received ICDs. Obviously, this number exceeds that of the high-risk cohort. We do not know if the investigators used more exclusion criteria (not mentioned in their report) to eventually form their high-risk group.

We believe that SCD risk in a cohort of patients having undergone ASA can be evaluated only by comparing matched, control patients. Nevertheless, the incidence of malignant ventricular arrhythmias after ASA presented in this study is very high. The major unresolved concern raised after ASA is the risk for arrhythmia-related events attributable to the septal scar generated after the procedure. The potential risk for malignant ventricular arrhythmias and SCD seems to be overcome after surgical myectomy. In a nonrandomized retrospective study, the incidence of SCD in patients with obstructive hypertrophic cardiomyopathy was significantly lower in those who underwent surgical myectomy compared to unoperated patients.[2] In another study, surgical septal myectomy was found to decrease the risk for appropriate ICD discharges in patients with obstructive hypertrophic cardiomyopathy.[3]

In conclusion, the issue of malignant ventricular arrhythmias after ASA should be seriously reevaluated.

Georgios K. Efthimiadis, MD
Efstathios D. Pagourelias, MD
Soultana Meditskou, MD
Christodoulos Pliakos, MD
Ioannis H. Styliadis, MD
Georgios E. Parcharidis, MD
Thessaloniki, Greece
8 June 2009

1. Noseworthy PA, Rosenberg MA, Fifer MA, Palacios IF, Lowry PA, Ruskin JN, Sanborn DM, Picard MH, Vlahakes GJ, Mela T, Das S. Ventricular arrhythmia following alcohol septal ablation for obstructive hypertrophic cardiomyopathy. *Am J Cardiol* 2009;104:128–132.
2. Ommen SR, Maron BJ, Olivotto I, Maron MS, Cecchi F, Betocchi S, Gersh BJ, Ackerman MJ, McCully RB, Dearani JA, Schaff HV, Danielson GK, Tajik AJ, Nishimura RA. Long-term effects of surgical septal myectomy on survival in patients with obstructive hypertrophic cardiomyopathy. *J Am Coll Cardiol* 2005;46:470–476.
3. McLeod CJ, Ommen SR, Ackerman MJ, Weivoda PL, Shen WK, Dearani JA, Schaff HV, Tajik AJ, Gersh BJ. Surgical septal myectomy decreases the risk for appropriate implantable cardioverter defibrillator discharge in obstructive hypertrophic cardiomyopathy. *Eur Heart J* 2007;28:2583–2588.

doi:10.1016/j.amjcard.2009.06.017

René Laennec (1781–1826) and the Invention of the Stethoscope

In 1816, René Théophile-Hyacinthe Laennec, a French physician, invented the stethoscope.[1] Laennec was born at Quimper in Brittany on February 17, 1781. At the age of 5 years, his mother died of tuberculosis. His father, a lawyer, turned over the upbringing of René to his uncle Guillaume, dean of the Faculty of Medicine at Nantes. Interested

in medicine, Laennec went to Paris to complete his medical education in 1799. He studied at Hôpital de Charité under Nicolas Corvisart, Napoleon's physician.[2] He studied also pathologic anatomy under Bichat and dissection under Bayle and Dupuytren. Laennec received his medical degree in 1804.

In 1816, Laennec succeeded Bayle as head of Hôpital Necker. In 1761, in Vienna, Leopold Auenbrugger (1722–1809) had introduced percussion as a new method of clinical examination for the detection of lung diseases. In 1808, Jean-Nicolas Corvisart translated into French Auenbrugger's book, *Inventum Novum*. Under Corvisart, Laennec was influenced by this book. In September 1816, in the Louvre's gardens, he observed 2 children who were playing with a long piece of solid wood and a pin. With an ear to one end, a child heard an amplified sound of the pin scratching the opposite end of the wood.[3] The same year, called to examine a young woman but embarrassed because of her age, gender, and corpulence, Laennec used a sheaf of paper rolled into a cylinder to auscultate the heart. By applying one end of the cylinder to her chest and the other to his ear, he heard sounds such as he had never before been able to hear with such clarity. The stethoscope was invented!

The word stethoscope comes from the Greek words "stethos," meaning "chest," and "skopein," meaning "to explore." He termed this indirect manner of listening to the chest "mediate auscultation." Wooden stethoscopes were used until the development of rubber tubing in the 19th century. In 1843, Williams introduced the first binaural stethoscope, using a lead pipe for each piece.

Laennec applied his invention to study cardiopulmonary diseases. He used his stethoscope to correlate bedside findings with autopsy results. Three years later, he produced his great treatise, *De l'Auscultation Mediate*. In January 1823, Laennec became a full member of the French Academy of Medicine and a professor at the medical clinic of Hôpital de Charité. In August 1824, he was made a chevalier of the Legion of Honor. He returned to Kerlournec in Brittany, when his health broke down. Laennec died on August 13, 1826, aged 45 years.

Abdallah Fayssoil, MD

Camden, New Jersey

22 June 2009

1. Bloch H. The inventor of the stethoscope: René Laennec. *J Fam Pract* 1993;37:191.
2. Roguin A. Rene Theophile Hyacinthe Laënnec (1781–1826): the man behind the stethoscope. *Clin Med Res* 2006;4:230–235.
3. Welsby PD, Parry G, Smith D. The stethoscope: some preliminary investigations. *Postgrad Med J* 2003;79:695–698.

doi:10.1016/j.amjcard.2009.06.053

Erratum for Tehrani F, et al. "Value of Medical Therapy in Patients >80 Years of Age With Heart Failure and Preserved Ejection Fraction" *Am J Cardiol* 2009;103:829–833.

The percentages reported in Tables 1 and 3 should be exchanged between the dead and alive column. In Tables 4 all cause and cardiac hospitalization rates for patients on beta blocker therapy should be 5.6 ± 8.8 and 2.3 ± 3.6, respectively. All cause and cardiac hospitalization rates for patients without beta blocker therapy are 5.0 ± 9.7 and 1.7 ± 2.5, respectively.

doi:10.1016/j.amjcard.2009.04.054