

5. Malina RM. Physical activity and fitness: pathways from childhood to adulthood. *Am J Hum Biol.* 2001;13:162-72.
6. González-Gross M, Castillo MJ, Moreno L, Nova E, González-Lamuño D, Pérez-Llamas F, et al. Alimentación y Valoración del Estado Nutricional de los Adolescentes Españoles (Proyecto AVENA). Evaluación de riesgos y propuesta de intervención. I. Descripción metodológica del estudio. *Nutr Hosp.* 2003;18:15-27.
7. Veiga OL, Gómez-Martínez S, Martínez-Gómez D, Villagra A, Calle ME, Marcos A. Physical activity as a preventive measure against overweight, obesity, infections,

allergies and cardiovascular disease risk factors in adolescents: AFINOS Study protocol. *BMC Public Health.* 2009;9:475.

8. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ.* 2000;320:1240-3.

doi:10.1016/j.rec.2011.01.007

## NSTEMI During a Stress Echocardiogram With Dobutamine: Where Is the Problem?

### *IAMSEST durante la realización de un ecocardiograma de estrés con dobutamina: ¿dónde está el problema?*

#### To the Editor,

Few studies have assessed the safety of dobutamine stress echocardiograms (DSE) since they were introduced into clinical practice in 1979. Rodríguez García et al.<sup>1</sup> describe a major complication rate of 1/325 with a dose >20 µg/kg/min, which is comparable to the results from a recently published meta-analysis by Geleijnse et al. (1/475).<sup>2</sup> Acute myocardial infarction (AMI) is a rare complication (0.02%<sup>2</sup>-0.06%<sup>1</sup>) in patients with coronary disease. Although these cited papers do not specify which definition of AMI was used, the first was published in 2001<sup>1</sup> and the second<sup>2</sup> includes studies performed in the nineties. The current AMI definition was published in 2007,<sup>3</sup> and establishes the "central role" of troponin. Meanwhile, dobutamine at doses >20 µg/kg/min is a strong inducer of ischemia due to its effects on arterial tone and double product, and some authors attribute to it increases in the activation and aggregation of platelets.<sup>1,2</sup> For the following case treated at our institution, we highlight the difficulties associated with interpreting isolated increases of troponin in the context of DSE under the current AMI definition.

The patient is a 60-year-old female presenting with obesity, hypertension, and type II diabetes. After 2 months of outpatient care for symptoms of effort angina, she was referred by the emergency room due to prolonged chest pain while at rest, unexplained by electrocardiographic or laboratory testing. Anti-ischemic treatment was prescribed and conventional ergometry and echocardiogram tests were ordered. The echocardiogram indicated dubious segment alterations on the anterior face. Conventional Bruce ergometry tests without medication, in which the patient reached 86% of the theoretical maximum heart rate, 10.2 MET, came back clinically and electrically negative, with an adequate blood pressure response. Given the persistence of symptoms during hospitalization and the echocardiographic anomalies, we decided to use a DSE. Under the conventional protocol, 10-40 µg/kg/min of dobutamine and 1 mg of atropine achieved 90% of theoretical maximum heart rate. After administering the atropine, the patient developed chest pain associated with vegetative reactions that persisted for 3 h, with no registry in the electrocardiogram or echocardiogram. We documented increased troponin I levels (maximum: 0.67 ng/ml at 3 h; normal until 0.04 ng/ml, and range of AMI ≥0.4) with normal creatine kinase. No epicardial lesions were found with the coronariography. Symptoms were attributed to a microcirculatory disease. No coronary vasospasm tests were administered, and the patient was treated with nitrates and diltiazem.

According to the current criteria defining acute coronary syndrome,<sup>3</sup> our patient had a non-ST-segment myocardial infarction with no epicardial coronary lesions. The symptoms indicate

microvascular damage. We did not rule out a possible vasospastic origin, but this was unlikely given that the absence of ST-segment alterations and motility in the acute phase. Furthermore, some studies have concluded that this possibility would be masked if the spasm were "distal".<sup>2</sup>

Some authors have measured troponin after DSE with contradictory results in terms of diagnostic and prognostic usefulness. Pastor et al.<sup>4</sup> found significant lesions in all patients that had an available coronariography, negative DSE, and elevated troponin levels. In addition, they concluded that dobutamine at the levels administered in these tests can produce slight myocardial damage as shown by the elevation in troponin levels, without abnormal motility and angiographic lesions. However, Meluzin et al.<sup>5</sup> and Beckman et al.<sup>6</sup> did not observe increased troponin I or T levels in 27 and 20 patients, respectively, with altered contractility during DSE and a known coronary disease.

The current definition of AMI may be too strict to be applied to spasm provocation tests such as DSE, and the problem could be increased with new "ultra-sensitive" techniques for detecting troponin levels. Furthermore, the incidence of AMI in the medical literature appears to be lower than the real values, probably due to the use of criteria that differ from our current definition. We need studies that evaluate the prognostic value of the increases in troponin levels after stress tests, which would allow their safety to be better assessed.

Juan G. Córdoba-Soriano,\* Juan Carlos García López, Víctor M. Hidalgo-Olivares, and Ana M. Ruíz Tornero

*Servicio de Cardiología, Complejo Hospitalario Universitario de Albacete, Albacete, Spain*

\*Corresponding author:

*E-mail address: jgcordobas@hotmail.com (J.G. Córdoba-Soriano).*

Available online 5 April 2011

## REFERENCES

1. Rodríguez García MA, Iglesias-Garriz I, Corral Fernández F, Garrote Coloma C, Alonso-Orcajo N, Branco L, et al. Evaluación de la seguridad de la ecocardiografía de estrés en España y Portugal. *Rev Esp Cardiol.* 2001;54:941-8.
2. Geleijnse ML, Krenning BJ, Nemes A, Van Dalen BM, Soliman O, Ten Cate FJ, et al. Incidence, pathophysiology, and treatment of complications during dobutamine-atropine stress echocardiography. *Circulation.* 2010;121:1756-67.
3. Thygesen K, Alpert JS, White HD, on behalf of the Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction. Universal definition of myocardial infarction. *Eur Heart J.* 2007;28:2525-38.
4. Pastor G, San Román JA, González-Sagrado M, Vega JL, Arranz ML, Serrador AM, et al. Ecocardiografía de estrés con dobutamina y troponina T como marcador de daño miocárdico. *Rev Esp Cardiol.* 2002;55:469-73.
5. Meluzin J, Toman J, Groch L, Hornacek I, Sitar J, Fischerova B, et al. Can dobutamine echocardiography induce myocardial damage in patients with dysfunctional but viable myocardium supplied by a severely stenotic coronary artery? *Int J Cardiol.* 1997;61:175-81.
6. Beckman S, Boscksch W, Müller C, Scharf M. Does dobutamine stress echocardiography induce damage during viability diagnosis of patients with chronic dysfunction after myocardial infarction? *J Am Soc Echocardiogr.* 1998;11:181-7.

doi:10.1016/j.rec.2010.10.036