example, physicians look for tests with good sensitivity even if the false-positive rate is a bit high. Computer-aided detection systems are often used to improve sensitivity, usually at a cost of even higher recall rates. Without reasonable sensitivity, many screening programs cannot be cost-effective. Further workup of these patients demands higher specificity to prevent unnecessary invasive testing. The consequences of test errors and prevalence of disease must be weighed in each application to find the best test for a particular application.

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EXPERIMENTAL VENTRICULAR PRESSURE OVERLOAD IN YOUNG ANIMALS INDUCES CARDIOMYOCYTE HYPERTROPHY IN ADDITION TO HYPERPLASIA OF CONTRACTILE AND NONCONTRACTILE ELEMENTS OF THE MYOCARDIUM

To the Editor:

Since the early 1990s, we have been working with experimental right ventric-

ular hypertrophy using young goats aged 4 to 8 weeks to achieve right (subpulmonary) ventricular retraining by means of a balloon catheter or an adjustable pulmonary artery banding system.¹⁻³ We have consistently found a hypertrophic response of the cardiomyocytes. Moreover, we have also demonstrated that the right ventricle responds not only with hypertrophy of the myocardial fibers but also with hyperplasia of the contractile and interstitial components of the myocardium.³ Recently, Leeuwenburgh and colleagues⁴ demonstrated the development of a hyperplastic rather than a hypertrophic myocardial response in young animals submitted to chronic right ventricular pressure overload. Their findings deserve some comments because previous studies also showed a hypertrophic response, even in young animals. Anversa and associates,⁵ studying 30 normal Wistar rats at 1, 5, and 11 days of age, found that left ventricular cardiomyocytes presented both hypertrophy and hyperplasia, attributing these to the physiologic pressure overload imposed on that chamber after birth.

Based on the age of the lambs (2–3 weeks), the duration of pressure overload (8-week period), and the reported data from the study by Leeuwenburgh and colleagues,⁴ we are surprised about the fact that the authors did not find any signal of myocardial fiber hypertrophy. Moreover, even considering the hyperplastic response alone, no mention was made regarding the presence or absence of proliferating (proliferating cell nuclear antigen-labeled) interstitial cells. If, as the authors stated, “more of the same tissue” was found in the trained myocardium, in addition to proliferating cardiomyocytes, they should have found some hyperplasia of cells from the interstitial compartment of the heart. In fact, this was a prominent feature of our experimental model of pressure overload in young animals.³ The number of interstitial and vessel cells labeled by the cell proliferation marker Ki67 in our study was 14 and 18 times greater, respectively, than that of the cardio-

myocytes under 2 different protocols of ventricular systolic overload (continuous and intermittent).³

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CLAMPING THE MITRAL AND TRICUSPID ANNULI WITH BIPOLAR DEVICES

To the Editor:

Castella and colleagues' article¹ reports evidence collected by application of a bipolar radiofrequency clamp on 8 explanted hearts and post-mortem analysis of 1 patient who died hours after surgery. The authors unveil 2 main findings: They state it is not possible to clamp across the mitral annulus because of the increased thickness of the atrioventricular (AV) groove, related to the superimposition of the ventricular mass and fat tissue. They also claim that, although feasible, clamping