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 ventricular 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 colleagues[4] 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 cardiomyocytes 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' article[1] reports evidence collected by application of a bipolar radiofrequency clamp on 8 explanted hearts and postmortem 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

up to the tricuspid annulus would always include the main right coronary artery. The authors conclude that both ablations are unfeasible with bipolar radiofrequency in clinical practice. The only viable options are to cut and sew up to the annulus, which is impractical, or to use an additional unipolar device to complete the last 2 cm on each annulus, which is more expensive and probably less effective.

Our experience led us to a totally different point of view. In regard to left ablation, we share the authors' concern on the risks of ablating a major coronary branch close to the posterior mitral annulus,[2] but rather than impeding left ablation, coronary anatomy should guide the ablation strategy. In patients with right dominance or a codominant system, the coronary-free spot of the AV groove must be actively identified and marked before ablation.[3] In the minority of patients with a left dominant coronary artery, the terminal portion does frequently leave the AV groove obliquely, before the crux cordis,[2] thus potentially leaving once again a safe spot for clamping. In the rare occasions that this safe spot is absent, we would complete the last couple of centimeters with a unipolar probe, reaching the annulus at or around the posteromedial mitral commissure,[2,4] aware there is no safe energy source to ablate across a major coronary vessel.

In regard to mitral clamping, the authors' findings contrast with prior experimental evidence[5] and our clinical experience.[3,4] Complete left ablation with bipolar radiofrequency entailing clamping across the mitral annulus (ie, biting at least 2 mm of the posterior mitral leaflet) has been our technique of choice ever since 2006[3] in now more than 200 patients. One possible explanation of such discrepancy is that, in the clinical setting, clamping across the mitral annulus is invariably performed under cardioplegia, on the flaccid heart, in diastolic arrest. The model used by Castella and colleagues[1] is different: The 9 ''fresh'' hearts were used for the study up to 36 hours after explantation or demise.[1] It is noteworthy that rigor mortis, which affects myocytes earlier than skeletal muscle, leads to an irreversible contraction of the myocardial fibers that is almost complete only 2 hours after death (or explantation).[6] Such contraction is functionally similar to that occurring during systole. Understandably, clamping close to the base of the contracted left ventricle may well turn out to be unpractical. But, although Castella and colleagues' model may reveal the possible shortcomings of the technique on the contracting heart, we think it is poorly representative of the clinical context of everyday open ablation.

On the right side, because, almost invariably, blind clamping across the AV groove would also include the right coronary artery, ablation up to the tricuspid annulus should take a different route, but again, this is definitely feasible. As in Wolf-Parkinson-White surgery, after entering the epicardium adjacent to the right atrial wall, the deepest portion of the AV groove is developed by blunt dissection, thus preparing the way for the external jaw of the clamp down to the plane of the tricuspid annulus.[4] In our preliminary experience this ablation proved routinely feasible, irrespective of the specific coronary anatomy.[4]

Despite Castella and colleagues' report,[1] we are convinced that clamping across the AV valves is still a viable way to perform a complete biatrial set of lines using bipolar radiofrequency alone, thus combining efficacy, swiftness, and cost-effectiveness.

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Reply to the Editor:

We appreciate Benussi and colleagues' comments on our study and recognize their expertise in the field of atrial fibrillation surgery. In their letter, our findings are stated precisely one by one.[1]

On the right side, both atrial and ventricular walls are thin enough to be included within the jaws of a bipolar clamp, so the main issue is to avoid the right coronary artery. We agree with Benussi and colleagues' comments, in which they refer to the technique of dissecting the right coronary artery to introduce the epicardial jaw of the clamp underneath the coronary to reach the tricuspid annulus. This is the only way to perform an ablation not using a monopolar device or the cut-and sew technique.

In regard to the ablation toward the mitral annulus, there are 2 issues: the danger of ablating coronary tissue and the impossibility of reaching the last millimeters of the annulus. We agree with and follow the concept introduced by Benussi and colleagues[2] of guiding