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EDITORIAL COMMENT

Cardiologists

Do We Have the Right to Call Ourselves Physiologists?*

Blase A. Carabello, MD, FACC
Houston, Texas

In this issue of *JACC: Cardiovascular Imaging*, Detaint et al. (1) report that accurate quantification of the amount of aortic regurgitation (AR) present in a given patient and the effect of that amount of AR on left ventricular (LV) function (indicated by end systolic volume index) are prognostic of outcome. Furthermore, these measures were superior to qualitative measures of AR—such as AR grade, jet-width, and so forth—in predicting outcome. Their findings make absolute sense, are not surprising, and are totally consistent with previous findings from Enriquez-Sarano et al. (2) regarding mitral regurgitation. You see, this is a group of cardiologists, folks who study the function of the heart by measuring what it does. What is surprising is that some will find their findings surprising. Those surprised are the same people, by the way, who, when asked for an opinion about how much AR a patient has, say, "Well I think it's about 2 to 3+." What in the world does that mean?

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Accurate calculation of cardiac volumes with contrast angiography has been available since the 1960s and was refined to an art form by the 1970s, allowing very accurate calculation of regurgitant volumes (2–6). Indeed such quantification of AR was reported superior to qualitative measures almost 25 years ago (7). I recognize that nothing could be more pointless than to yearn for the good old days when cardiologists knew these techniques or could measure a transvalvular gradient accurately in the catheterization laboratory or knew the limitations of valve area and did not report those areas out to 2 decimal places. But when catheterization laboratories became interventional venues and diagnosis moved to noninvasive laboratories, why did we have to start all over again? Why did it take 25 years to get back to where we were in the invasive era of cardiac diagnosis? To be sure, a whole new diagnostic tool with its clear noninvasive advantage had to be developed, and initially the images available did not lend themselves well to precise quantification. And it was not really until Doppler interrogation arrived that a primarily anatomic technique became empowered to reveal physiology. Still, as a group we seem to avoid quantification and embrace qualitation, even when more sophisticated technology exists.

It is not just in the determination of cardiac volumes that we have fallen down; it seems our ambivalence toward quantification occurs in many other areas of cardiology. Undeniably the most important function of the myocardium is to generate force by which it contracts against a load, propelling blood forward to sustain life. The innate force-generating capacity of the heart, contractility is widely thought to be key to prognosis in heart disease (8). At least 30 indexes have been developed to measure this function, yet ejection fraction, with all its foibles and load dependence, is used ubiquitously to assess cardiac performance, even in valvular heart disease where load is pointedly altered. It was refreshing to see end-systolic volume reaffirmed here as a useful prognostic index in AR (1,9,10). End-systolic volume and/or dimension are dependent upon contractility, afterload, and sarcomere number but are independent of preload. Thus, at least one of the factors confounding ejection fraction is removed when this index is used. Admittedly, many other indexes of LV function are

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From the Department of Medicine, Baylor College of Medicine, and the Veterans Affairs Medical Center, Houston, Texas.

cumbersome to use and difficult to understand, and ejection fraction "works" in many instances. Yet limitations of ejection fraction as a useful tool are often ignored, leading in some cases to poor clinical judgment. How often I have heard that an ejection fraction of 0.55 in a patient with mitral regurgitation indicates normal LV function or that the patient with aortic stenosis and a reduced ejection fraction has LV dysfunction when at the myocardial level neither might be true.

Then there is assessment of coronary stenoses. For decades we have known that 2 highly trained angiographers "eyeballing" the same coronary stenosis might have two very different interpretations of the same lesion or, when seeing the same lesion again at a different time, might assess a different degree of stenosis than when it was examined the first time (11–13). In recognition of this weakness, a large investment was made in developing quantitative angiography, Doppler- and pressure-assessed (fractional) flow reserve, and intravascular ultrasound (14–17). Yet, as noted in the most recent edition of a popular textbook, "In clinical practice, however, the degree of lesion stenosis is usually just estimated visually from the coronary angiogram" (18). Indeed, in my experience, this statement is accurate for most catheterization laboratories.

Why do we behave like this? Is it because we are too busy to measure things accurately? Is it because we do not understand the principles behind quantification? Is it because simpler methods work fairly well most of the time? I suspect it is a little of all of these components that form our practice. To be sure, there are many times when the severity of a lesion is so great or so trivial that quantification is unnecessary. Yet we would rarely accept qualitation in other parts of medicine. Would it be acceptable for our nephrologist to tell us that our patient's potassium was moderately elevated or for our hematologist to tell us that our patient's international normalized ratio was moderately out of range? I doubt it.

Perhaps we are entering a new era when our patients demand more precision from us and we will demand more from ourselves. An era when cardiac imaging is so sophisticated that it will yield accurate cardiac measurements more easily. The data presented by Detaint et al. (1) indicate that such precision has real impact on our ability to better gauge prognosis and find the proper timing of surgery. I personally hope their study spurs us to do better not only in valvular heart disease but also in other areas of our profession. However, for this to happen we and our trainees must recognize that, from the time Werner Forssmann placed a catheter in his own antecubital vein and Richards and Cournand used that principle to make intracardiac measurements leading to the Nobel Prize (19,20), part of cardiology is physiology: to understand the heart we must understand how it works, and to understand when it is not working we must be able to measure aspects of its function accurately. If we can remember that we are all physiologists and remember to act like physiologists, perhaps statements like "Well, I think he's got 2 to 3 + AR" will disappear, resulting in better care for our patients. Let's hope that modern cardiac imaging reconnects us with a basic foundation of cardiology that is, in fact, physiology.

Reprint requests and correspondence: Dr. Blase A. Carabello, Veterans Affairs Medical Center, Medical Service (111), 2002 Holcombe Boulevard, Houston, Texas 77030. *E-mail: blaseanthony.carabello@med.va.gov.*

REFERENCES

1. Detaint D, Messika-Zeitoun D, Maalouf J, et al. Quantitative echocardiographic determinants of clinical outcome in asymptomatic patients with aortic regurgitation: a prospective study. J Am Coll Cardiol Img 2008; 1:1–11.
2. Enriquez-Sarano M, Avierinos JF, Messika-Zeitoun D, et al. Quantitative determinants of the outcome of asymptomatic mitral regurgitation. N Engl J Med 2005;352:875–83.
3. Sandler H, Dodge HT, Hay RE, Rackley CE. Quantitation of valvular insufficiency in man by angiocardiography. Am Heart J 1963;65:501–13.
4. Kennedy JW, Trenholme SE, Kasser IS. Left ventricular volume and mass from single-plane cineangiocardiogram. A comparison of anteroposterior and right anterior oblique methods. Am Heart J 1970;80:343–52.
5. Miller GAH, Brown R, Swan HJC. Isolated congenital mitral insufficiency with particular reference to left heart volumes. Circulation 1964;29: 356–65.
6. Carabello BA, Nakano K, Corin W, Biederman R, Spann JF Jr. Left ventricular function in experimental overload hypertrophy. Am J Physiol 1989; 256:H974–81.
7. Croft CH, Lipscomb K, Mathis K, et al. Limitations of qualitative angiographic grading in aortic or mitral regurgitation. Am J Cardiol 1984;53: 1593–8.
8. Carabello BA. Evolution of the study of left ventricular function: everything old is new again. Circulation 2002; 105:2701–3.
9. Borow K, Green L, Mann T, et al. End systolic volume as a predictor of postoperative left ventricular performance in volume overload from valvular regurgitation. Am J Med 1980;68: 655–63.
10. Carabello BA, Williams H, Gash A, et al. Hemodynamic predictors of outcome in patients undergoing valve replacement. Circulation 1986;74: 1309–16.

11. Zir LM, Miller SW, Dinsmore RE, Gilbert JP, Harthorne JW. Interobserver variability in coronary angiography. Circulation 1976;54:627–32.
12. Randall PA. Mach bands in cine coronary arteriography. Radiology 1978; 129:65–6.
13. Arnett EN, Isner JM, Redwood DR, et al. Coronary artery narrowing in coronary heart disease: comparison of cineangiographic and necropsy findings. Ann Intern Med 1979;91:350–6.
14. Spears JR, Sandor T, Als AV, et al. Computerized image analysis for quantitative measurement of vessel diameter from cineangiograms. Circulation 1983;68:453–61.
15. Escaned J, Baptiwsta J, DiMario C, et al. Significance of automated stenosis detection during quantitative angiography: insights gained from intracoronary ultrasound imaging. Circulation 1996;94:966–72.
16. Kern M. Curriculum in interventional cardiology: coronary pressure and flow measurements in the cardiac catheterization laboratory. Cathet Cardiovasc Intervent 2002;54:378–400.
17. Mintz GS, Nissen SE, Anderson WD, et al. American College of Cardiology clinical expert consensus document on standards for acquisition, measurement and reporting of intravascular ultrasound studies (IVUS): a report of the American College of Cardiology Task Force on Clinical Expert Consensus Documents (Committee to Develop a Clinical Expert Consensus Document on Standards for Acquisition, Measurement and Reporting of Intravascular Ultrasound Studies [IVUS]). J Am Coll Cardiol 2001;37:1478–92.
18. Baim DS. Coronary angiography. In: Baim DS, editor. Grossman's Cardiac Catheterization, Angiography, and Intervention. 7th edition. Philadelphia, PA: Lippincott, Williams & Wilkins, 2006:187–221.
19. Forssmann W. Die Sondierung des rechten Herzens [in German]. Klin Wochenschr 1929;8:2085.
20. Cournand AF. Nobel lecture, December 11, 1956. In: Nobel lectures, physiology and medicine 1942–1962. Amsterdam: Elsevier, 1964:529.