Distinction of “Fat Around the Heart”

We were greatly interested by the study by Wong et al. (1) on pericardial fat and atrial fibrillation (AF) outcomes. This study further highlights the association of pericardial fat with cardiac disease. The authors remark that pericardial fat may predict the severity and symptomatic burden of AF and recurrence of AF after conventional AF ablation techniques. The authors have used the term pericardial fat in this study to denote the fat present between the myoepicardium and the pericardium. Others have described this structure more aptly, however, as epicardial fat, thus distinguishing it from paracardial fat that is present outside the parietal pericardium (2). This differentiation is necessary since these 2 types of fat deposits have different embryological origins (3).

Epicardial fat, having similar origin as that of omental and mesenteric fat (3), correlates better with body mass index and metabolic syndrome and is likely to be a better surrogate for cardiac disease (4). Unlike the paracardial fat, the epicardial fat shares a common blood supply with the myocardium and is believed to have paracrine properties that are implicated as a cause of myocardial inflammation due to its proximity to the myocardium. Indeed, autopsy studies have shown that epicardial fat extends into the apposing myocardium (5). Prior studies have used the term pericardial fat to represent both paracardial and epicardial fat (6). It appears that in this study, the authors have used pericardial fat interchangeably for epicardial fat. Furthermore, they have not clearly mentioned whether magnetic resonance imaging is sufficiently sensitive to separate the epicardial fat from the paracardial fat given the temporal motion artifact in magnetic resonance images. Interestingly enough, the authors have calculated epicardial fat in end diastole. A recent review by Iacobellis et al. (2) suggests that epicardial fat should be assessed in end systole during echocardiography. Fat being a compressible structure is better visualized in systole, as there is more space for it to expand.

Nevertheless, the authors have made a significant contribution to our knowledge of the association between pericardial fat and cardiac disease, and should be congratulated on their effort. Future insights into defining the role of epicardial fat in cardiac disease may usher development of interventions to curb the growing epidemic of cardiac disease across its spectrum.

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Reply

We thank Drs. Kaushik and Reddy for their interest in our recently published report on the associations between pericardial fat and atrial fibrillation (1). They correctly point out that pericardial fat is divided into 2 layers—the visceral, epicardial fat layer, and the paracardial fat layer, situated external to the parietal layer of the pericardium. Such a distinction is important as they have different embryological origins, vascular supplies, and potential pathogenic mechanisms (2). In our study, we eventually opted to measure pericardial fat, however, because of difficulties in consistently separating the epicardial and paracardial layers surrounding both the atria and ventricles for analysis. The relevant sentence in our methodology should thus more correctly read: “Pericardial fat was defined as regions of high signal intensity between the myoepicardium and the outermost margin of paracardial fat.” Drs. Kaushik and Reddy also suggest that pericardial fat may be better visualized in systole as opposed to end diastole, as we measured in our report. However, this recommendation was based on echocardiographic assessment of epicardial fat thickness over the right ventricular free wall, as opposed to a volumetric assessment of total pericardial fat and the use of cardiac magnetic resonance imaging (3). Not only are there important differences between the different measurements and imaging modalities, but also, to the best of our knowledge, there have been no comparative studies on epicardial, paracardial, or pericardial fat analysis in systole versus diastole, and no clear consensus yet as to which is best. In our study, we opted to measure pericardial fat in end diastole, given we have previously validated volumetric pericardial fat analysis in end diastole using cardiac magnetic resonance imaging with ex vivo pericardial fat volumes (2). Drs. Kaushik and Reddy have importantly highlighted, however, that differences in the assessment of epicardial, paracardial, and pericardial fat volumes during the cardiac cycle do warrant further clarification in better understanding the relationship between differing
adiposity measurements and the varied mechanisms underlying the substrates supporting atrial fibrillation (4,5).

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Medication Adherence
Is Not Our Problem?

I would respectfully disagree with the decision to exclude medication adherence from the position statement on performance measures for adults with coronary artery disease and hypertension (1). After reviewing the reasons outlined by the writing committee, I am concerned that this decision may have been made without a detailed review of the best available evidence. I believe that medication adherence should be considered an important outcome of care rather than an inevitable destiny for one-half the patients who receive prescription medicines.

Three reasons were provided for excluding nonadherence as a performance measure for physicians. First, “adherence is largely not in the individual physician’s locus of control” (1). On the contrary, a significant body of research suggests that physicians have powerful influence over medication adherence. Although factors underlying the positive elements of this association have not been clearly defined, evidence suggests that patient–physician relationships (2), follow-up visits (3), communication (4), and medical management skills (5) are important determinants. Therefore, it would appear that physicians are not just innocent bystanders in this public health epidemic.

Second, “because patient autonomy is the overriding ethical and pragmatic principle governing the patient–physician relationship, the patient is free to decide whether to take medications as prescribed” (1). Few would argue that patients have the final say in the matter of adherence. However, upholding the principle of patient autonomy does not preclude physicians from helping patients make good decisions. If physicians (or other health care professionals) opt out of the decision-making process, patients are left to navigate the Internet or newspapers to help inform their choice of whether to take medications regularly. At a minimum, engagement in the decision-making process will ensure that physicians are aware of the final decision. Perhaps acknowledging that a final patient decision about adherence is understandable might even improve compliance with the ethical principle of patient autonomy.

The final reason provided was phrased as follows: “a measure of patient adherence could cause physicians to avoid caring for patients with a history of nonadherence or a perceived likelihood of being nonadherent” (1). In contrast, I believe that a measure of adherence could begin to promote the idea that good adherence is an outcome of external factors. I would hope the anecdotal risk for refusing care to patients would be speculative at best, whereas the value of mobilizing a highly skilled profession toward an “age-old” problem would be priceless.

Medication adherence is a complex phenomenon with a multitude of causes that are probably slightly different for every patient. Nonetheless, a physician’s influence is likely an important factor, even under conditions of high copayments or poor employer benefit plans (2). Ultimately, I am concerned that the messages contained in the medication adherence section of the document (1) will reinforce a longstanding myth that physicians have no role to play in addressing this public health problem.

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