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Editorial Comment

Detecting Coronary Artery Disease in Left Bundle Branch Block*

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Left bundle branch block (LBBB) creates a problem in detecting spontaneous or inducible myocardial ischemia or even acute myocardial infarction despite a modification of the ST-segment criteria (1). Although LBBB could be an isolated electrocardiographic abnormality, it could also be, especially in elderly patients, associated with other cardiac diseases such as coronary artery disease (CAD), cardiomyopathy, and valvular heart diseases. The patients could, therefore, be asymptomatic or present with chest pains, heart failure, syncope, or murmurs. Although inter- and intraventricular dyssynchrony cause abnormal splitting of the first and second heart sounds, it is fair to say that, in most patients, the diagnosis is only made with certainty after the 12-lead electrocardiogram is obtained.

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The work-up of a patient with LBBB depends on presentation and age. For those with suspected CAD, the evaluation should answer the following questions: Is there CAD; what is the extent and severity of CAD? Is the LBBB primary or secondary due to scar? (Scar and LBBB are not always cause and effect.) What is the status of viability of the myocardium in those with heart failure and what is the status of left ventricular (LV) function in terms of ejection fraction and remodeling (dilatation, hypertrophy, and secondary mitral regurgitation)?

The treadmill exercise electrocardiogram is not reliable in detecting ischemia according to American College of Cardiology/American Heart Association guidelines (2,3). The treadmill test, however, could provide a plethora of non–ST-segment information that has prognostic relevance. These include exercise metabolic equivalents, heart rate at peak exercise and early recovery, blood pressure response during exercise and early recovery, and presence or absence of ventricular arrhythmias during exercise but especially during recovery (3).

It took some time before it was recognized that exercise myocardial perfusion imaging with single photon emission computed tomography (SPECT) is less than ideal in LBBB because of the occurrence of *reversible* perfusion defects in the anteroseptal region in the absence of stenosis of the left anterior descending artery (LAD) (2). Such defects appear to be heart-rate related and could occur with dobutamine stress because dobutamine, like exercise, increases the heart rate. These defects are not present at rest when the heart rate is in the normal range. The presence of defects at rest and stress (*fixed or irreversible*) in the anteroseptal region is an entirely separate issue and denotes the presence of prior anteroseptal myocardial infarction, which is unrecognized because of LBBB and which could very well be the cause (and not the result) of the LBBB. The reversible defects are only seen in ~40% to 50% of patients with LBBB during exercise and are more frequent with faster peak heart rates. The LBBB-related reversible defects look for all practical purposes like those due to LAD stenosis. Perfusion defects (fixed or reversible), on the other hand, in the inferior and lateral zones of the myocardium are not LBBB-related and signify underlying disease in the left circumflex or right coronary arteries, or both, regardless of the mode of stress modality. The mechanism of LBBB-related anteroseptal reversible defects has been well studied in animal and man and appears to reflect variation in phasic flow in the LAD, and abnormal septal wall stress and metabolism (2,3).

Because of the recognition that high heart rates could produce "false" positive defects (they are not truly false, however), vasodilator imaging with adenosine or dipyridamole has been the accepted stress modality in such patients (4). No exercise should be combined with either agent to avoid high heart rates. Vasodilator SPECT imaging has decreased the false-positive rates (reversible anteroseptal defects) from ~40% to ~5%. Few exceptions are seen and, in my experience, could be explained by unusual increases in heart rate in response to vasodilator challenge.

When regional function is assessed by gated SPECT, there is septal dyskinesia but with normal wall thickening in LBBB (in the presence or absence of reversible

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defects), while wall thickening is also abnormal in those with septal scar. The above principles should apply to other imaging methods such as stress echocardiography and magnetic resonance imaging when assessing wall motion and thickening and perfusion at rest or during stress.

In this issue of the *Journal*, Ghostine et al. (5) examined the role of 64-slice computed tomography (CT) in detecting CAD in 66 patients with LBBB. Heart failure was the most common presenting symptom and was seen in 42% of the patients. Another 12% had silent ischemia although it is not clear how this diagnosis was made. To avoid verification bias, all patients had coronary angiography (CA) after CT. Further, once included, no patient was subsequently excluded based on image quality or other artifacts. The studies were read blindly, and there was excellent agreement between CT and CA; 28 of 29 (97%) patients with CAD and 35 of 37 (95%) patients without CAD by CA were correctly identified by CT. Further, of the 90 stenoses in 990 segments by CA, 68 (72%) were correctly identified by CT. The authors suggest that CT could be an alternative strategy to stress testing.

It should be noted that this study, as many studies, used the 50% diameter stenosis cutoff to define "significant stenosis" even though extensive data clearly demonstrate the unreliability of diameter stenosis as a measure of the physiological relevance of stenosis in terms of impairment in flow reserve (6–9). Moreover, 28% of lesions were missed in those with >1 stenosis. Figure 2 in the work by Ghostine et al. (5) shows an example of such a discordance in the information provided by the 2 methods; the CT missed the downstream lesions, which were severe, but identified the proximal lesions, which were mild. The management of this patient might be entirely different based on the 2 sets of information. The authors indicate that the heart rate (yes, once again the heart rate!) and motion contributed to the less than ideal image quality and misdiagnosis, but that is the reality of clinical practice where exactly the same if not more of such problems could be expected. Did the CT provide sufficient information that could help patient management in terms of the extent and severity of ischemia, LV function, viability, and scar? The other imaging modalities would have provided such information and in addition have a track record of demonstrating the prognostic value of these variables (2). The authors write ". . . multilesion analysis in clinical practice is time-consuming and should refer the patients for CA for revascularization management" (5). It is difficult to accept this argument in deciding who to refer for CA without full information on extent and severity of CAD and status of viability in those with heart failure. The finding of any CAD by CT cannot explain the poor LV function and heart failure, which was present in ~50% of the patients. It is true that the negative predictive value was high, but the pre-test likelihood of CAD could have been different in patients with no CAD compared with those with CAD (not provided by authors), and a separate test was used to assess LV function (in this case, echocardiography).

Where is the role of CT in LBBB? This is unknown at present and is not unlike the role of the CT in other patient groups, even though CT provides exquisitely sharp images. There are patients with LBBB who have contraindications to adenosine or dipyridamole because of bronchospastic lung disease in whom CT will be useful. But it is likely that these patients will not be good candidates for CT either because beta-blockers are not safe or patients cannot hold their breath for sufficient lengths of time to acquire the images. This scenario needs to be tested. Also, the patients with inconclusive or poor quality SPECT studies (with which there is far more experience than with other imaging modalities) will also be candidates for CT.

The imaging community in the U.S. is under scrutiny to abide by guidelines for appropriate utilization of procedure and quality measures (10,11). Quality is far more complicated than simply sharpness of the images (11). The reason is simple; the growth rate (and costs) in imaging has exceeded the growth rates of other procedures (12). Before we adopt new imaging methods to patient care, we should be sure that the test provides sufficient diagnostic and prognostic information, decreases unnecessary downstream utilization of other imaging procedures, and limits the radiation burden to the patients. It is only through well-designed prospective studies that the answers to such questions could be obtained and algorithms of when to use CT versus other imaging procedures or versus no imaging procedures are premature.

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