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Bolus Ejection: A Method for Removing Sea Urchin Spines

To the Editor:

Sea urchin spine penetration can establish a painful inflammatory tissue reaction that can last for months. Although the spine may resolve, in some people the wound is disabling. Surgical exploration has been recommended for spines that are deeply embedded for a several-week period and for spines that have produced granuloma.[1] Routine open hand surgery can be followed by flexural contraction and limitation of joint movement. Such complications also occur after exploration of sea urchin spines. On these occasions, the surgeon faces a disgruntled patient and possible litigation. A method of spine recovery that is free from these adverse events was suggested earlier and is expanded in this letter.

Radiographic localization of the spine is indicated to determine joint disruption or bony penetration to assess for future sequella. The number of spines and degree of serration are noted to determine the difficulty of removal. Whether future scanning will be a diagnostic help is not known. Tumescent local anesthesia is produced by injecting a 1.5-mL bolus of subcutaneous lidocaine into each lateral surface of the involved phalanx. The apex of the edematous wound or site of maximal discoloration, pain, or potency is nicked with the point of a scalpel blade. Any liquid discharge can be cultured in the appropriate media for mycobacteria, fungi, or aquatic bacteria, and any recovered debris can be examined in the pathology laboratory.

This technique was safely used in the case of an 11-year-old boy.[2] The spine was safely extruded by propulsion initiated by the lidocaine tumescence. It is anticipated that this simple outpatient technique can be adopted and extrapolated successfully for other em-

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1. Baden HP. Injuries from sea urchins. In: Mandojana R, ed. Clinics in Aquatic Dermatology. *5th ed. Philadelphia, PA: JB Lippincott; 1987:112-117.*

2. Burnett JW, Burnett MG. Sea urchins. Cutis. *1999;64:21-22.*

Beyond Left Bundle-Branch Block: Looking for the Acute Transmural Myocardial Infarction

To the Editor:

The recent study of Kontos et al[1] (article #114900; May 2001) is entitled "Can Myocardial Infarction Be Rapidly Identified in Emergency Department Patients Who Have Left Bundle-Branch Block?" However, throughout the paper, the patient population being studied is referred to as emergency department patients who are being evaluated for myocardial ischemia. By blurring the distinction between ischemia and infarction and, furthermore, by failing to distinguish between non–ST-segment elevation infarction and acute ST-segment elevation infarction, the evaluation of the ECG criteria of Sgarbossa et al[2] is fatally flawed.

The Sgarbossa criteria were derived from patients enrolled in the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-I) study. All patients in that study received thrombolytic therapy and, therefore, had symptoms that were thought to be strongly suggestive of acute transmural infarction, or what would be called ST-segment elevation infarction if left bundle-branch block (LBBB) were not present. This presumably means that such patients had ongoing chest pain or equivalent symptoms not easily relieved by conservative treatment. Therefore, it is likely that the ST-segment changes described by Sgarbossa et al are markers for transmural injury, or what would be ST-segment elevation if LBBB were absent.

Kontos et al[1] make no assertion that their patient population had such symptoms. Indeed, of 182 patients studied, only 9 were categorized as "high risk" or as having symptoms warranting urgent catheterization or reperfusion therapy. Only 24 (13%) of these patients were ultimately found to have myocardial infarction, and the median peak creatine phosphokinase level of 340 U/L further suggests that many of these events were the equivalent of non–ST-segment elevation infarctions.

Multiple studies[3] have demonstrated that, when LBBB is absent, thrombolytic therapy decreases mortality only in acute ST-segment elevation myocardial infarction. It is not a great leap of faith to assume that, in patients with LBBB, such therapy would only be effective in patients with transmural injury or the equivalent of ST-segment elevation. Has that been definitively proven? Perhaps not, but mechanistically it makes a lot of sense, and, moreover, any patient with LBBB who was enrolled in a major thrombolytic trial had symptoms consistent with an acute transmural infarction. LBBB has traditionally been thought to preclude ST-segment analysis, and generally, this remains the case. To the clinician, however, the most vexing problem with LBBB is not knowing who will ultimately prove to have an acute coronary syndrome, but rather rapidly identifying patients who might benefit from an emergency revascularization, those with acute transmural injury. In that role, the Sgarbossa criteria might be significantly more sensitive than Kontos et al[1] give it credit for.

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1. Kontos MC, McQueen RH, Jesse RL, et al. Can myocardial infarction be rapidly identified in emergency department patients who have left bundle-branch block? Ann Emerg Med. *2001;37:431-438.*

2. Sgarbossa EB, Pinski SL, Barbagelata A, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. GUSTO-I (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) Investigators. N Engl J Med. *1996;334:481-487.*

3. Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomized trials of more than 1,000 patients. Lancet. *1994;343:311-322.*

To the Editor:

Using creatine kinase (CK)-MB as their criterion standard, Kontos et al[1] (article #114900; May 2001) made conclusions about the utility of the Sgarbossa[2] criteria for directing reperfusion therapy. However, because they used CK-MB instead of an angiographic end point, they should have compared the Sgarbossa diagnostic utility with that of standard ST-segment elevation criteria in the absence of left bundle-branch block (LBBB).

Kontos et al[1] found that the presence of any one of the 3 criteria had a sensitivity of 46% for CK-MB–diagnosed acute myocardial infarction (AMI), which they concluded was unacceptably low. In the context of normal conduction, the sensitivity of ST-segment elevation for AMI as diagnosed by CK-MB has been between 45% and 47%.[3-5] If angiogram-proven ongoing coronary occlusion had been used as the criterion standard, the Sgarbossa criteria would have had much better sensitivity. Indeed, in the context of normal conduction, the sensitivity of ST-segment elevation for complete coronary occlusion is much higher than it is for all CK-MB–diagnosed AMI (at least 70% to 92%).

The specificity of ST-segment elevation for AMI in the context of normal conduction ranges from 15% to 95%. Using CK-MB as the criterion standard, one model found an optimum ST-segment elevation model that diagnosed AMI with 56% sensitivity and 94% specificity,[6] compared with 46% and 93% for the Sgarbossa criteria in the study by Kontos et al.[1]

In another study of 797 patients with suspected AMI, LBBB was present in 48 patients, 24 of whom had AMI diagnosed by CK-MB. All 24 patients with negative CK-MB findings had a Sgarbossa score of 0 (specificity and positive predictive value [PPV] 100%). Of those with proven AMI, 21/24 had a score ≥2, for a sensitivity of 88% and negative predictive value (NPV) of 89%.[7]

Thus, the sensitivity and specificity of the Sgarbossa criteria for AMI in the pres-

ence of LBBB appear to be as good as that of ST-segment elevation for AMI in the absence of any BBB. Furthermore, predictive values depend on the pretest probability of AMI. Combining 2 large studies of chest pain patients, 1,962 (16.6%) of 11,805 patients ruled in for AMI by CK-MB.[5,8] In 5 large relevant studies of ischemic symptoms and LBBB, excluding the studies of Kontos et al[1] and Li et al,[9] 188/448 (42%) had AMI.[3,7,10-12] Thus, the pretest probability of AMI is much higher among all patients with ischemic symptoms and LBBB than it is among those without LBBB. It follows that, for any given specificity, the PPV of the Sgarbossa criteria are higher than is the PPV of ST-segment elevation in the context of normal conduction.

The prevalence of AMI in Kontos et al[1] and Li et al[9] combined was 49 (13%) of 372, which is much lower than the other studies of LBBB and ischemic symptoms. Both of the former studies included "all patients presenting to the ED who were evaluated for myocardial ischemia."[19] Thus, Kontos et al and Li et al included many patients at low risk of AMI. This was not the same group of patients with suspected ongoing coronary occlusion who had reduced mortality with thrombolytics (over placebo) in early major trials. Thus, the PPVs derived from the study by Kontos et al do not reflect the true utility of the Sgarbossa criteria for detecting ongoing coronary occlusion.

In conclusion, the study by Kontos et al[1] does not address the relative utility of the Sgarbossa criteria to that of ST-segment elevation in normal conduction. Both are imperfect for diagnosing AMI as confirmed by CK-MB and are much better for identifying patients who will benefit from thrombolytic therapy.

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1. Kontos MC, McQueen RH, Jesse RL, et al. Can myocardial infarction be rapidly identified in emergency department patients who have left bundle-branch block? Ann Emerg Med. *2001;37:431-438.*

2. Sgarbossa EB, Pinski SL, Barbagelata A, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. GUSTO-I (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) Investigators. N Engl J Med. *1996;334:481-487.*

3. Rude RE, Poole WK, Muller J, et al. Electrocardiographic and clinical criteria for recognition of acute myocardial infarction based on analysis of 3,697 patients. Am J Cardiol. *1983;52:936-942.*

4. Fesmire FM, Percy RF, Wears RL, et al. Initial ECG in Q wave and non–Q wave myocardial infarction. Ann Emerg Med. *1989;18:741-746.*

5. Rouan GW, Lee TH, Cook EF, et al. Clinical characteristics and outcome of acute myocardial infarction in patients with initially normal or nonspecific electrocardiograms (a report from the Multicenter Chest Pain Study). Am J Cardiol. *1989;64:1087-1092.*

6. Menown IB, Mackenzie G, Adgey AA. Optimizing the initial 12-lead electrocardiographic diagnosis of acute myocardial infarction. Eur Heart J. *2000;21:275-283.*

7. Edhouse JA, Sakr M, Angus J, et al. Suspected myocardial infarction and left bundle-branch block: electrocardiographic indicators of acute ischaemia. J Accid Emerg Med. *1999;16:331-335.*

8. Karlson BW, Herlitz J, Wiklund O, et al. Early prediction of acute myocardial infarction from clinical history, examination and electrocardiogram in the emergency room. Am J Cardiol. *1991;68:171-175.*

9. Li SF, Walden PL, Marcilla O, et al. Electrocardiographic diagnosis of myocardial infarction in patients with left bundle branch block. Ann Emerg Med. *1999;36:561-566.*

10. Hands ME, Cook EF, Stone PH, et al. Electrocardiographic diagnosis of myocardial infarction in the presence of complete left bundle-branch block. Am Heart J. *1988;116:23-31.*

11. Kudenchuk PJ, Maynard C, Cobb LA, et al. Utility of the prehospital electrocardiogram in diagnosing acute coronary syndromes. J Am Coll Cardiol. *1998;32:17-27.*

12. Cannon CP, McCabe CH, Stone PW, et al. The electrocardiogram predicts one-year outcome of patients with unstable angina and non–Q wave myocardial infarction: results of the TIMI-III Registry ECG Ancillary Study. Thrombolysis in Myocardial Ischemia. J Am Coll Cardiol. *1997;30:133-140.*

In reply:

We thank Dr. Waxman and Dr. Smith for their comments. Both indicate that Sgarbossa's criteria (presumably concordant ST-segment elevation and depression, rather than lower specificity discordant ST-segment elevation) can identify patients with myocardial infarction (MI) who benefit from fibrinolysis. We agree. As stated in the discussion in our article, "…these 2 criteria can be used to select patients for fibrinolytic therapy."[1] However, a key point of our study was that these criteria identified only a minority of patients with left bundle-branch block (LBBB) and MI.

Dr. Waxman indicates that fibrinolytics are beneficial only in patients with complete vessel occlusion and may be harmful in patients who have partial patency. As stated in the discussion, treatment with a glycoprotein IIb/IIIa antagonist may be the preferred therapy in the absence of diagnostic ECG changes. A number of considerations temper this assessment. To our knowledge, there are no data that demonstrate that patients with concordant ST-segment elevation or depression have an occluded vessel, that the absence of such changes predicts a patent vessel, or that fibrinolytics are preferentially beneficial in patients with diagnostic ECG changes and LBBB. Until such data are available, it can be only considered a hypothesis. Second, current recommendations do not differentiate on the basis of the presence or absence of ECG changes, but recommend early reperfusion treatment in all patients with presumed MI and LBBB.

Dr. Smith points out that our patients had a lower prevalence of MI than in prior studies, which may have skewed the predictive values. We analyzed the data after excluding the patients at lower risk who underwent perfusion imaging. Of the 107 patients who were considered high to moderate risk, 20 (19%) had MI, of whom 14 were not identified by concordant ST-segment elevation or depression. Predictive values were essentially unchanged.

Dr. Smith asserts that the prevalence of MI in prior studies was substantially higher at 42%. However, this is an overestimate, because this estimate does not include our study, that of Li et al,[2] other studies from Table 1, or Shlipak et al.[3] In addition, Hands et al[4] was derived from patients from Rude et al[5] (which is why it was not included in our Table 1). Inclusion of these studies results in a 27% (256/930) MI prevalence.

Therefore, we stand by our conclusions that "current recommendations that all patients with LBBB should be treated with fibrinolytics will result in administering fibrinolytic therapy to many patients who will not have AMI. Better methods are needed for identifying patients who have LBBB and AMI who will benefit from fibrinolytic therapy."[1]

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1. Kontos MC, McQueen RH, Jesse RL, et al. Can myocardial infarction be rapidly identified in emergency department patients who have left bundle-branch block? Ann Emerg Med. *2001;37:431-438.*

2. Li SF, Walden PL, Marcilla O, et al. Electrocardiographic

diagnosis of myocardial infarction in patients with left bundle branch block. Ann Emerg Med. *1999;36:561-566.*

3. Shlipak MG, Lyons WL, Go AS, et al. Should the electrocardiogram be used to guide therapy for patients with left bundle-branch block and suspected myocardial infarction? JAMA. *1999;281:714-719.*

4. Hands ME, Cook EF, Stone PH, et al. Electrocardiographic diagnosis of myocardial infarction in the presence of complete left bundle branch block. Am Heart J. *1988;116:23-31.*

5. Rude RE, Poole WK, Muller JE, et al. Electrocardiographic and clinical criteria for recognition of acute myocardial infarction based on analysis of 3,697 patients. Am J Cardiol. *1983;52:936-942.*

Electrocardiographic Diagnosis of Acute Myocardial Infarction in the Presence of Left Bundle-Branch Block: Can We "Treat All" Patients?

To the Editor:

Kontos et al[1] (article #114900; May 2001) confirmed that the ECG criteria that we proposed to diagnose acute myocardial infarction (AMI) in the presence of left bundle-branch block (LBBB) were highly specific but lacked sensitivity.[2] We suggested in 1996 that our ECG criteria should not be used to screen patients with LBBB for the presence of AMI[2] and have expressed surprise[3] at subsequent publications that underscored the criteria insensitivity.[4,5] The prevalence of AMI among patients with chest pain and LBBB is less than 20%.[1,5] Because our criteria seem to be present only in 6% of patients with confirmed infarction,[1] their relative usefulness stems from their high specificity. We agree with Kontos et al and Gunnarsson et al[5] that this may apply better to the larger infarcts, which may be electrocardiographically more "visible" and be overrepresented in the Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries (GUSTO-I) study population that we analyzed.[2]

The advantage of using likelihood ratios (LRs) for diagnosis is that they remain stable when either the disease prevalence or "severity mix" change. In his editorial regarding the study by Kontos et al,[1] Gallagher[6] (article #114761) commented that, because our ECG criteria had a high positive LR (of 22 for "either ECG criterion"), when detected they allow to diagnose infarction. Thrombolysis could be offered only to such selected patients with LBBB. However, Gallagher then proposes a perplexing "reperfusion to all" approach. This recommendation seems to be based on unquestioned American Heart Association–American College of Cardiology guidelines and in non-Aristotelian reasoning. First, with an LR of 22, the posterior probability of disease (with a prevalence of <20%) after positive test results is approximately 80%. Thus, our proposed ECG signs detected in a LBBB patient with chest pain confer an approximately 80% probability of infarction, which should provide high confidence. Second, although Gallagher underscores the importance of evidence-based medicine to recommend therapies, he then assumes that the benefit from thrombolysis for "BBB patients" in the Fibrinolytic Therapy Trialists' (FTT) Collaborative Group report must refer to patients with LBBB because "the logic of cardiac conduction argues that suspected AMIs with LBBB rather than right BBB (RBBB) are likely to receive the greatest therapeutic benefit from thrombolysis."[6] (This assumption is also used in Gallagher's sensitivity analysis.) Published studies evidence the opposite: patients with AMI and LBBB have half the mortality risk of their RBBB counterparts.[7,8] Finally, the age of LBBB patients with infarction in the more recent reports is 74 years or older.[1,5] According to Thiemann et al[9] and Berger et al,[10] patients 75 years or older are unlikely to benefit from thrombolysis. Berger et al found that patients with LBBB were more likely to die at both 30 days and 1 year when treated with reperfusion, independent of the strategy selected.

Can emergency physicians and cardiologists still abide by the "reperfusion to all" mandate in patients with chest pain and LBBB? Perhaps these patients should be offered emergency catheterization to identify candidates for surgical revascularization. In settings without these resources, an ECG with positive signs of infarction may be useful to prescribe thrombolysis among the younger minority of patients with LBBB if deemed lifesaving. Measures aimed at treating bradyarrhythmias, electrical instability, or septal asynergy might prove more vital for patients with LBBB.

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1. Kontos MC, McQueen RH, Jesse RL, et al. Can myocardial infarction be rapidly identified in emergency department patients who have left bundle-branch block? Ann Emerg Med. *2001;37:431-438.*

2. Sgarbossa EB, Pinski SL, Barbagelata A, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. GUSTO-I (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) Investigators. N Engl J Med. *1996;334:481-487.*

3. Sgarbossa EB, Pinski SL, Wagner GS. Left bundle-branch block and the ECG in diagnosis of acute myocardial infarction [letter]. JAMA. *1999;282:1224-1225.*

4. Edhouse JA, Sakr M, Angus J, et al. Suspected myocardial infarction and left bundle-branch block: electrocardiographic indicators of acute ischaemia. J Accid Emerg Med. *1999;16:331-335.*

5. Gunnarsson G, Eriksson P, Dellborg M. ECG criteria in diagnosis of acute myocardial infarction in the presence of left bundle-branch block. Int J Cardiol. *2001;78:167-174.*

6. Gallagher EJ. Which patients with suspected myocardial ischemia and left bundle-branch block should receive thrombolytic agents? Ann Emerg Med. *2001;37:439-444.*

7. Sgarbossa EB, Pinski SL, Topol EJ, et al. Acute myocardial infarction and complete bundle-branch block at hospital admission: clinical characteristics and outcome in the thrombolytic era. J Am Coll Cardiol. *1998;31:105-110.*

8. Go AS, Barron HV, Rundle AC, et al. Bundle-branch block and in-hospital mortality in acute myocardial infarction. National Registry of Myocardial Infarction 2 Investigators. Ann Intern Med. *1998;129:690-697.*

9. Thiemann DR, Coresh J, Schulman SP, et al. Lack of benefit for intravenous thrombolysis in patients with myocardial infarction who are older than 75 years. Circulation. *2000;101:2239-2246.*

10. Berger AK, Radford MJ, Wang Y, et al. Thrombolytic therapy in older patients. J Am Coll Cardiol. *2000;36:366-374.*

In reply:

Dr. Sgarbossa, who proposed several criteria for the diagnosis of acute myocardial infarction (AMI) in the setting of left bundle-branch block (LBBB),[1] understandably finds the recommendation that "all patients with LBBB and suspected AMI should receive thrombolysis" unsatisfying. I concur. When I undertook an analysis of aggregated data drawn from the 9 studies that comprised the Fibrinolytic Therapy Trialists' systematic review,[2] I had hoped, by combining the Sgarbossa criteria with information on the age of the LBBB, to derive a decision algorithm that performed better than the strategy of administering thrombolytics to all patients with LBBB and suspected AMI. Unfortunately, the data refused to cooperate.[3] My conclusions were disappointingly similar to both the current practice guidelines of the American Heart Association and the American College of Cardiology[4] and to those of another recent decision analysis.[5]

Try as we might, we seem unable to iden-

tify that elusive subset of patients with AMI obscured by LBBB with sufficient accuracy to preclude administration of thrombolytic agents to all such patients. As a management strategy, this is hardly gratifying, knowing that for most patients it constitutes an unnecessary risk of limb- or life-threatening side effects, without discernible benefit. Frustration with our inability to solve this problem has fueled repeated attempts to develop criteria for identification of AMI in the setting of LBBB. Indeed, over the past half century, more than 50 electrocardiographic criteria,[6] of which those of Sgarbossa et al[1] are the most recent, have been proposed to distinguish ischemia from the depolarization/repolarization abnormalities intrinsic to LBBB. Like the Sgarbossa criteria, many showed initial promise, but none ultimately met with success.

The reasons for this are numerous and varied. However, 3 plausible explanations readily emerge from a combined analysis of 2 papers[7,8] on the Sgarbossa criteria accompanied by an examination of the original paper by Sgarbossa et al[1]:

1. Despite excellent specificity (96% [95% confidence interval (CI) 94% to 98%]), the poor sensitivity of the Sgarbossa criteria (20% [95% CI 10% to 34%]) precludes their application as a clinically useful screening ("rule-out") test.[3]

2. Only approximately 3% (95% CI 2% to 6%) of patients with LBBB and suspected ischemia meet any of the Sgarbossa criteria.[3] Thus, their low prevalence among patients with LBBB helps to explain why the criteria are infrequently clinically helpful, high specificity notwithstanding.[3]

3. Finally, validation of the Sgarbossa criteria took place in a cohort of only 45 patients and was associated with a decay in sensitivity of more than 50% compared with that obtained in the derivation cohort.[1] Such marked numerical instability of performance characteristics often signals spectrum bias in the development of a decision rule, resulting in a low probability of generalization to a more heterogeneous population of patients. Indeed, the cohort of patients with AMI from which the Sgarbossa criteria were derived were all obtained from the GUSTO-I trial of thrombolytic therapy.[1]

Dr. Sgarbossa rightly challenges the statement that "the logic of cardiac conduction argues that suspected AMIs with LBBB rather than RBBB are likely to receive the greatest therapeutic benefit from thrombolysis."[3] For this sentence fragment to make any sense, it first needs to be reassembled by reinserting the introductory phrase that underscores its conditionality. The complete sentence then needs to be placed in the context of the paragraph containing it, as well as the paragraphs immediately preceding and following it. It then becomes evident that the "logic" of cardiac conduction simply refers to the difference in the electrophysiologic characteristics of septal and ventricular depolarization in the setting of LBBB versus RBBB. This difference is logically related to the higher probability of an LBBB obscuring the characteristic ECG signature of AMI because of interference with initial electrical forces.[3] Hence, the challenge of identifying occult ischemia in the setting of LBBB.

Closely related to the aforementioned statements is some recent evidence[9,10] offered by Dr. Sgarbossa, suggesting that, despite the comparative ease with which AMI can usually be diagnosed electrocardiographically in RBBB, the latter may actually portend a worse prognosis than LBBB in the setting of acute ischemia. Both references provided to support this contention are instructive to examine because they illustrate the difficulty of drawing valid inferences from associations that may be incompletely adjusted for confounding clinical features.[9,10] In the first paper, written by Sgarbossa et al,[9] the doubling of 30-day mortality associated with RBBB compared with LBBB is seen only in the univariate analyses, disappearing completely when appropriate adjustment is made for covariates such as MI location and Killip classification. In fact, the multivariate odds ratio for BBB (RBBB and LBBB combined) derived from the authors' logistic model is bounded by a CI with a lower limit of the null point of 1.00.[9] In the second paper, which is drawn from a careful analysis of the National Registry of Myocardial Infarction (NRMI)-2, Go et al[10] report that RBBB is a stronger independent predictor of in-hospital mortality than LBBB. Although these investigators adjusted this association for all available confounders, they lacked data on infarct location for half the patients with RBBB and two thirds of those with LBBB. The inability to adjust for location of MI, resulting in residual confounding, may explain the apparent differential mortality.

Finally, Dr. Sgarbossa raises the question of age as a factor in the decision to pursue reperfusion, asserting that patients 75 years of age or older with LBBB are unlikely to benefit from any reperfusion strategy.[11,12] The ability of either reference cited by Dr. Sgarbossa to support this contention is problematic. In the first paper, the investigators clearly state that all patients with LBBB were excluded from analysis.[11] A review of the second citation is equally puzzling because these authors, who included patients with LBBB, report a clinically and statistically significant survival benefit at 1 year for both groups of elderly patients who received thrombolytic agents or primary angioplasty when compared with controls receiving no reperfusion therapy.[12]

When 2 independent quantitative decision analyses[3,5] concur with a third thoughtfully reasoned set of practice recommendations,[4] one must at least entertain the possibility that, unsettling as an inference might seem, it may nonetheless be true. I share Dr. Sgarbossa's frustration with such a recommendation. Evidence, however, is under no obligation to conform to our intellectual preferences or preconceptions, no matter how strongly they may be held.

Thus, in summary, it appears that, until such time as contravening evidence becomes available, the best answer to the question of which patients with suspected AMI and LBBB should receive thrombolytic agents is also the simplest: "All of them."[3]

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1. Sgarbossa EB, Pinski SL, Barbagelata A, et al. Electrocardiographic diagnosis of evolving acute myocardial infarction in the presence of left bundle-branch block. GUSTO-I (Global Utilization of Streptokinase and Tissue Plasminogen Activator for Occluded Coronary Arteries) Investigators. N Engl J Med. *1996;334:481-487.*

2. Fibrinolytic Therapy Trialists' (FTT) Collaborative Group. Indications for fibrinolytic therapy in suspected acute myocardial infarction: collaborative overview of early mortality and major morbidity results from all randomised trials of more than 1,000 patients. Lancet. *1994;343: 311–322.*

3. Gallagher EJ. Which patients with suspected myocardial ischemia and left bundle-branch block should receive thrombolytic agents? Ann Emerg Med. *2001;37:439-444.*

4. Ryan TJ, Antman EM, Brooks NH, et al. ACC/AHA guidelines for the management of patients with acute myocardial infarction: 1999 update: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction). Available at: http://www.acc.org/clinical/guidelines. Accessed August 12, 2001.

5. Shlipak MG, Lyons WL, Go AS, et al. Should the electrocardiogram be used to guide therapy for patients with left bundle-branch block and suspected myocardial infarction? JAMA. 1999;281:714-719.

6. American College of Emergency Physicians. Clinical policy: critical issues in the evaluation and management of adult patients presenting with suspected acute myocardial infarction or unstable angina. Ann Emerg Med. 2000;35:521-544.

7. Li SF, Walden PL, Marcilla O, et al. Electrocardiographic diagnosis of myocardial infarction in patients with left bundle branch block. Ann Emerg Med. 1999;36:561-566.

8. Kontos MC, McQueen RH, Jesse RL, et al. Can myocardial infarction be rapidly identified in emergency department patients who have left bundle-branch block? Ann Emerg Med. 2001;37:431-438.

9. Sgarbossa EB, Pinski SL, Topol EJ, et al. Acute myocardial infarction and complete bundle-branch block at hospital admission: clinical characteristics and outcome in the thrombolytic era. J Am Coll Cardiol. 1998;31:105-110.

10. Go AS, Barron HV, Rundle AC, et al. Bundle-branch block and in-hospital mortality in acute myocardial infarction. National Registry of Myocardial Infarction 2 Investigators. Ann Intern Med. 1998;129:690-697.

11. Thiemann DR, Coresh J, Schulman SP, et al. Lack of benefit for intravenous thrombolysis in patients with myocardial infarction who are older than 75 years. Circulation. 2000;101:2239-2246.

12. Berger AK, Radford MJ, Wang Y, et al. Thrombolytic therapy in older patients. J Am Coll Cardiol. 2000;36:366-374.

In Defense of Patient Privacy

To the Editor:

I read with keen interest the letter by Dr. Zibulewsky[1] (article #116661) in the August 2001 issue of *Annals* regarding a recent editorial I wrote.[2] It is no surprise that someone who read my editorial at precisely the same moment they were wired to a microphone on a television show that routinely disregards the principle of advance informed consent would defend his own actions. I am quite sure that, if a survey were taken of all physicians who have participated in this, the vast majority would agree that such filming is acceptable, if not desirable, as the author seems to posit. Readers might be surprised to know that several physicians who did participate in these shows have written to me expressing their regret for having done so, and more than one has shared with me that they only participated because their hospital administrators pressured them to do so. In fact, one prominent ethicist in emergency medicine stated to me privately that, after the publication of my editorial, he would now be better able resist the efforts of his administration to pressure him to participate in such shows—unless he could set appropriate guidelines and restrictions.

A careful reading of my editorial reveals that I am not in favor of banning all filming that might portray us in a good light or, more important, that might benefit patients. In fact, my department has on numerous occasions participated in filming, but with the restrictions that I outlined, namely, advanced consent, absent conditions of duress. (The "CNN Perspectives" piece that I alluded to in my editorial—the one in which the producers wanted to enter the trauma rooms and were not allowed to—aired in autumn of 2000. I was not filmed, but I was present throughout the filming to be sure that no patient's rights were violated, and they were not.)

We need not trample on privacy to know that we have "arrived," as is suggested. Cardiac surgeons and neurosurgeons arrived a long time ago and do not allow cameras into their intimate moments with patients without the patients' permission. Equating the television drama *ER* with the reality-based *Trauma: Life in the ER* is a fallacy. *ER* is clearly fiction and perhaps is the vehicle by which to "arrive," if that is even an important goal.

Finally, the author appears not to understand the difference between secrecy and privacy. We clearly do not operate in secrecy whether television cameras are present or not. Everyone knows that the emergency department is a "fishbowl." Secrets can hardly be kept in environments that are wide open and that treat thousands of patients each year, not to mention the families, police officers, clergy, and others who frequently accompany them. We do, however, hold privacy to be a fundamental right, a principle that goes back to the time of Hippocrates. Hopefully, long after people forget *Trauma: Life in the ER,* they will still remember Hippocrates.

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1. Zibulewsky J. Filming of emergency department patients [letter]. Ann Emerg Med. 2001;38:189.

2. Geiderman JM. Fame, rights, and videotape. Ann Emerg Med. 2001;37:217-219.

CORRECTION

In the September 2001 issue, the article by Pollack and Gibler ("2000 ACC/AHA Guidelines for the Management of Patients With Unstable Angina and Non–ST-Segment Elevation Myocardial Infarction: A Practical Summary for Emergency Physicians"; pages 229-240), a line was inadvertently dropped during the editing process from the Figure in the box second from the bottom on page 231. The corrected Figure is presented here. The publisher regrets this error.

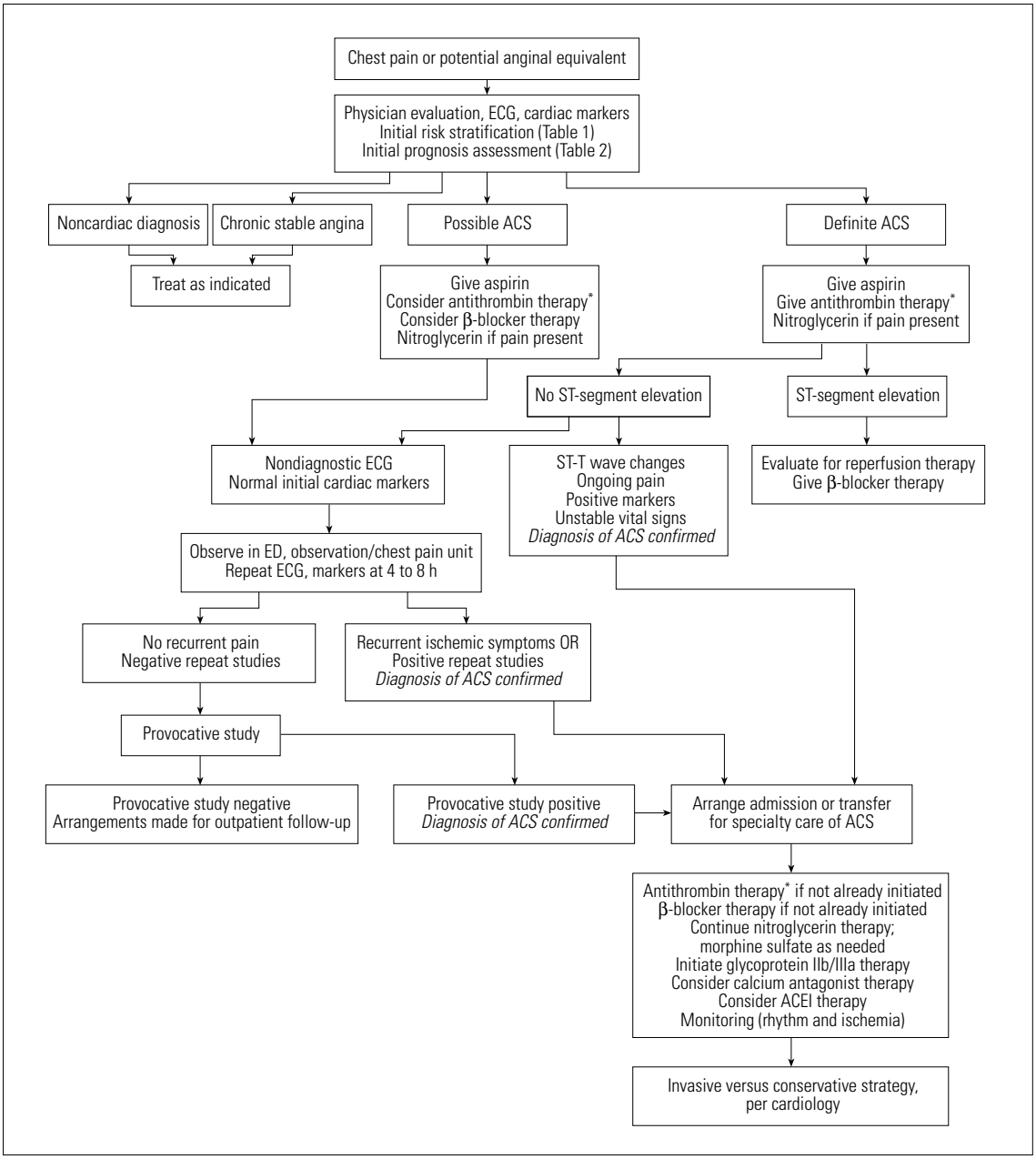

Figure.
Summary of guideline recommendations for evaluation and management of possible ACS pertinent to the ED.[1] ***ACEI,*** *Angiotensin-converting enzyme inhibitor.* **Unfractionated or low-molecular-weight heparin.*