

to our department for dyspnea was published in 2001.³ A 4-step score of ultrasound comet tail sign appearance was correlated with a corresponding chest x-ray score.⁴ The sensitivity and specificity of ultrasound was 97%, with a positive and negative predictive value of 94% and 98%, respectively. The correlation between ultrasound and radiologic score was significant (0.90).

According to our study and the study of Jambrik et al, we confirm that chest sonography has the potential to evaluate extravascular lung water at bedside in a simple and reliable way.

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LQTS and SIDS Linkage: Clarifying the Record

I read with interest the report of Christiansen et al.¹ documenting an ion-channel mutation as the cause of sudden infant death syndrome (SIDS) in a 7-week-old child. The molecular link between ion-channelopathies such as long QT syndrome (LQTS) and SIDS is an important contribution in providing an etiology for at least a proportion of victims of this still largely mysterious and misunderstood syndrome—undoubtedly with a heterogeneous variety of largely undocumented causes and mechanisms. As the authors have correctly acknowledged, other investigators have established a role for QT interval prolongation in SIDS,^{2–4} including 2 other reported cases of

SIDS due to ion-channel mutations.^{3,4} However, their assertion that the role of LQTS in SIDS was “. . . initially suggested by Schwartz, et al. in the 1970s . . .” is unfortunately quite incorrect. In 1976, I and my colleagues at the National Heart, Lung, and Blood Institute first offered this hypothesis with an electrocardiographic study of QT intervals in parents of SIDS cases⁵ and also in a “near miss” SIDS survivor.^{5,6} Those reports served as an inspiration to my colleague and good friend, Peter Schwartz and his colleagues,^{2–4} >20 years before he went on to publish seminal work on the QT interval and its role in SIDS. Unfortunately, Christiansen et al, failed to recognize our work and Dr. Schwartz also forgot to cite our studies in his ambitious 1998 study² and later in 2000.³ This information is provided here, not to be accusatory, but only to clarify the historical medical record and the published reports in this area.

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Assessing the Quality of Predictive Models for Classification

The artificial neural network (ANN), a computational simulation of the biologic nervous system, has been widely used as a predictive model in medicine with the help of advances in computer-assisted analysis. Therefore, the quality of the chosen ANN models is of increasing concern. To assess the quality for the classification model in clinical investigation, it would be more appropriate to calculate discrimination and calibration concurrently.¹ Common measures used in discriminating diagnostic tests include sensitivity, specificity, positive and negative predictive values, likelihood ratios for positive and negative tests, and the areas under receiver-operating characteristic (AUROC) curves. Allison et al² constructed the ANN models to predict the stenosis of major coronary vessels from the data of stress single-photon emission computed tomography. The authors demonstrated the sensitivity and specificity without mentioning AUROC, which can provide a better index for the performance of each model.

In contrast, although many researchers used the AUROC curve with the best simultaneous sensitivity and specificity to determine discriminatory power of a model, a good discrimination has the possibility of poor calibration when classification outputs are transformed monotonically.³ To avoid this pitfall, calibration using Pearson's chi-square, Hosmer-Lemeshow statistic, or the misclassification rate should be considered. Additionally, inter-rater agreement with κ values among models could be adopted to approach the reproducibility and repeatability.⁴ In the era of evidence-based medicine, a new diagnostic model should be carefully and critically appraised because arbitrary evaluations may lead to wrong conclusions.

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Correction

Two of the column headings of Table 2 in our article “Effect of elevated admission serum creatinine and its worsening on outcome in hospitalized

patients with decompensated heart failure” (*Am J Cardiol* 2004;94:957–960) are incorrect. The correct Table 2 is below.

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Correction

There is an error in the last sentence of the Reader’s Comment, “Will green tea be even better than black tea to increase coronary flow velocity reserve” (*Am J Cardiol* 2004;94:1223). This sentence should actually be: “It is also important to know if the cardioprotective effect of flavonoids from both green and black teas can be attributed not only to **antioxidant**,³ **antithrombogenic**⁶ and **anti-inflammatory**⁷ properties, but also to improvement in coronary flow velocity reserve.”

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Correction

In the article by Gurm et al (Effectiveness and safety of *bivalirudin* dur-

ing percutaneous coronary intervention in a single medical center,” vol. 95, no. 6, March 15, 2005, pp. 716–721), there were several errors that appeared in Tables 1 and 3. In Table 1, the patients receiving the bivalirudin based regimen should read **205 (19.2%)** instead of 205 (18.2%). Also, in Table 1, the patients receiving the bivalirudin based regimen with platelet glycoprotein IIb/IIIa inhibitors should read **206 (19.3%)** instead of 352 (19.3%). In Table 3, column 2, the patients receiving bivalirudin based regimen with restenotic lesions should read **205 (19.2%)** instead of 205 (18.2%). Also, in column 2, the patients receiving the bivalirudin based regimen with platelet glycoprotein IIb/IIIa inhibitors should read **206 (19.3%)** instead of 352 (19.3%). In Table 3, column 5, the patients receiving the bivalirudin based regimen with restenotic lesions should read 175 (20.3%) instead of 175 (24.6%).

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Table 2

Comparison in outcome in patients with and without renal insufficiency (creatinine >1.5 mg/dl)

Outcome	Renal Insufficiency		p Value
	No (n = 266)	Yes (n = 215)	
Mortality (95% CI) at 30 d	5.3% (3.0%–8.5%)	8.8% (5.4%–13.2%)	0.149
Mortality (95% CI) at 6 mo	12.3% (8.6%–16.7%)	37.4% (30.8%–43.9%)	<0.0001
Length of hospitalization (d)	8.2 ± 7.1 (6)*	10.3 ± 8.4 (7)*	0.003
Readmission within 30 d of discharge	17%	27%	0.016

* Numbers in parenthesis represent medium length of stay.

CI = confidence interval.