Correspondence

Letters to the Editor must not exceed 400 words in length and may be subject to editing or abridgment. Letters must be limited to three authors and five references. They should not have tables or figures and should relate solely to an article published in Circulation within the preceding 12 weeks. Only some letters will be published. Authors of those selected for publication will receive prepublication proofs, and authors of the article cited in the letter will be invited to reply. Replies must be signed by all authors listed in the original publication.

Documentation for the Sake of Documentation?
To the Editor:
In their recent study, Frolkis and colleagues\(^1\) attempt to determine by chart review how often patients with acute myocardial infarction are counseled to quit smoking and encouraged to exercise. They acknowledge that if no such counseling nor encouragement is documented in the chart, the study assumes none was done.

Certainly a patient hospitalized with an acute infarction is unusually likely to be amenable to the idea of quitting smoking and ought to be so encouraged. But many patients are still not interested in quitting. In those cases, most doctors probably do not document a fruitless attempt at counseling in the chart. Why commemorate a waste of breath with a waste of ink? Frolkis and colleagues suggest doing the documentation if only to placate chart reviewers and defense lawyers.

Physician time and patience are finite resources. Suggesting that a patient quit smoking and getting the brush-off from that patient is a bit trying. Having in addition to write in a chart, “Counseled patient to quit smoking, but compliance doubtful,” is considerably more trying on a doctor’s patience. Furthermore, a cursory comment about smoking and lifestyle changes, made mainly to justify a chart entry that “covers” the doctor, is very unlikely to leave a patient with any useful motivation.

The idea that “If it’s not documented in the chart, it wasn’t done,” speaks volumes about anyone who subscribes to it.

David Grant, MD, FACP, FACC
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Response
We were pleased that Dr Grant acknowledges the importance of counseling high-risk patients to quit smoking cigarettes but were puzzled by the intensity of his objection to documenting such an effort. The use of cigarettes represents the largest preventable cause of mortality and health-related expenditure in the United States, accounting for \(\sim 400\) 000 deaths yearly.\(^2\) The data are clear that even brief interventions by physicians and other healthcare professionals can significantly increase quit rates and that the documentation of current tobacco use is a key element of any successful cessation program.\(^3\) Interventions targeted to hospitalized patients have reported major impact, presumably because patients may be more receptive to suggestions regarding lifestyle change in the setting of an acute event or procedure.\(^4\) Unfortunately, many patients report that their caregivers have never urged them to quit smoking.\(^5\)

In addition, as we report, there is good evidence that what is recorded in the hospital record is an accurate reflection of actual physician behavior, so the conclusion that “most doctors probably do not document a fruitless attempt at counseling in the chart” must be viewed with caution. Moreover, we are not encouraging documentation “to placate chart reviewers and defense lawyers,” as Dr Grant states; we are merely reporting the reality of the ways chart notes can be used.

Less clear is what Dr Grant finds so different about documenting advice to discontinue cigarette use from the many other suggestions physicians offer and routinely record in the hospital or office chart. Advising patients to lose weight, increase exercise, reduce alcohol consumption, wear seat belts, practice safe sex, or get yearly mammograms, as well as documenting such advice, provides key information for our own future use or for those colleagues who may encounter our patients at another time or in another setting. Because most patients who eventually quit cigarettes successfully have tried and failed previously, noting one’s effort to help patients with this complicated and sometimes painful process can provide both patient and physician with a useful marker of their joint progress. We are not advocates of defensive medicine or creating unnecessary paperwork. Our concern is that physicians underutilize their considerable influence in helping patients adopt the healthier lifestyles that could decrease the burden of heart disease.

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Postoperative Readmissions to Hospital Do Not Result From Controlled Early Discharge
To the Editor:
Dr J.C. Baldwin’s editorial comment\(^1\) criticized the report by Dr S.J. Lahey et al, “Hospital Readmission After Cardiac Surgery: Does ‘Fast Track’ Cardiac Surgery Result in Cost Saving or Cost Shifting?”\(^2\) as follows: “It does appear that the ‘fast track’ program being used at this institution is a rather conservative one, which may underestimate the real differences in readmission rates and the potential impact of hospitals’ efforts to shorten LOS [length of stay] for given DRGs [diagnosis-related groups].” This means that if the patients in this study had gone home sooner after surgery than they did, more patients would have been readmitted.

On the contrary, evidence indicates the opposite. In previous studies, patients passed exercise tests, met standardized criteria...
of fitness for discharge, and were discharged on the third postoperative day. There was no increase in readmission rates.\textsuperscript{3,4} In one study, all 40 patients discharged on the third postoperative day stayed well and were not rehospitalized in the following 2 years.\textsuperscript{3} If the 417 patients in Lahey’s study who were not rehospitalized had performed exercise tests on the third postoperative day, some of them would have passed the tests and could have been discharged promptly without readmission. So, by identifying and sending home sooner the healthiest and strongest patients, Lahey could have reduced the average hospital stay without increasing readmissions.

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As Americans, We Should Get This Right
To the Editor:

After reading just a fraction of the many articles published on the long-QT syndrome, I became confused about the correct spelling of the distinctive polymorphic ventricular tachycardia identified in this disease. I have seen the following spellings: torsade de pointe, torsades de pointe, torsades de pointes, and torsade de pointes. In the November 24, 1998, issue of Circulation, in the wonderful article by Drs Shimizu and Antzelevitch,\textsuperscript{1} the term “torsade de pointes” was used. In search of the correct way to spell this disorder, I consulted with French medical colleagues. They have informed me that “torsades de pointes” is the right way. The English language dominates so much of medical writings and is usually the presiding language at scientific meetings around the world. It sure seems to me that we could at least get this one, frequently used phrase consistently correct. We owe it to the very beautiful French language. TORSADES DE POINTES.

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Response

We are grateful to Dr Sydney Moise for her kind remarks regarding our recent article in Circulation.\textsuperscript{1}

The spelling of “torsade(s) de pointes” has received a good deal of attention over the years, yet both “torsade de pointes” and “torsader de pointes” continue to be widely used to describe the atypical polymorphic ventricular tachycardia that commonly accompanies the long-QT syndrome (LQTS). A scan of the literature reveals that even among French cardiologists and electrophysiologists, both terms are widely used.\textsuperscript{2,3}

To gain an appreciation for why this is, it behooves us to go back to the origin of the term as described by Dessertenne.\textsuperscript{5,6} Because of the sinusoidal twisting of the QRS around the isoelectric line, Dessertenne termed this arrhythmia “torsade de pointes,” or twisting of the points. It would appear that Dessertenne and many prominent French electrophysiologists who subsequently published on the subject\textsuperscript{2} considered a single twisting of the points as a “torsade de pointes” and repeated twistings or repeated episodes as “torsades de pointes.”

Thereupon rests the distinction. The unit cycle that defines the arrhythmia is a “torsade,” whereas repeated twisting or repeated episodes are “torsades.” Because a single twisting of the QRS around the isoelectric line is sufficient to define the arrhythmia under congenital or acquired long-QT conditions, “torsade de pointes” should suffice to describe the tachycardia entity. Although repeated twisting of the points during a single episode may be more accurately depicted as “torsades de pointes,” this term does not accurately describe a single twisting of the points, frequently observed in clinical cases as well as in experimental models of LQTS. It can be further argued that repeated episodes of torsade(s) de pointes should not be noted in the plural, because in the English language they are not referred to as atypical polymorphic ventricular “tachycardias” but rather as episodes or runs of “tachycardia.” This distinction is due largely to fundamental differences in grammar usage between the 2 languages.

In summary, from our vantage point and with all due respect for the French language, “torsade de pointes,” because it defines the arrhythmia and is all encompassing, might best be used to denote the arrhythmia entity (whether comprised of single or repeated episodes). In documents written in English, “torsades de pointes” might best be reserved for describing repeated twistings during a single episode.

While our intent at the outset was to attempt to arrive at a universally acceptable spelling for torsade de pointes, we suspect that we may have contributed to a further muddying of the water, or is it “waters”? Wasn’t it the French who coined the term “vive la différence”?

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Activation of Nuclear Factor-κB in Unstable Angina
To the Editor:
The recent study by Ritchie\textsuperscript{1} provides evidence of systemic nuclear factor-κB (NF-κB) activation in patients with unstable angina undergoing coronary arteriography and is in agreement with other clinical studies demonstrating systemic immunologic activation.

The major difficulty in interpreting these findings is one of distinguishing cause from effect; rather than reflecting a predisposition to subsequent plaque disruption, NF-κB activation may be the consequence of other subsequent processes, including platelet activation or myocardial ischemia. Increased platelet-leukocyte adhesion can be demonstrated in patients with acute coronary syndromes, and in vitro studies demonstrate NF-κB activation in leukocytes exposed to thrombin-stimulated platelets. NF-κB activation may also occur indirectly as a consequence of myocardial ischemia or necrosis and in response to myocardial synthesis of pro-inflammatory cytokines during ischemia-reperfusion. Prospective studies are needed to prevent these confounding influences and allow an assessment of the true clinical significance of NF-κB activation in patients with coronary artery disease.

Nicholas Jenkins, MB ChB, MRCP
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Response

I thank Dr Jenkins for his response to my article. I agree that it is difficult to distinguish cause from effect with regard to NF-κB activation in humans with unstable angina pectoris, particularly considering the data cited both in Dr Jenkins’ letter and my article showing that events subsequent to plaque rupture can activate NF-κB. However, on the basis of the serendipitous observation noted in the article that 2 patients with clinically stable coronary artery disease who subsequently became clinically unstable with serial angiograms showing acute plaque rupture had marked activation of NF-κB, I am optimistic that NF-κB activation precedes the acute event. Retrospective 5-year follow-up analyses showing a direct relationship between NF-κB levels of activation and events (death, myocardial infarction, and unstable angina), regardless of initial clinical presentation, further support this conjecture (J. Gillespie, unpublished data, 1999). Admittedly, neither article directly addresses the “cause or effect” position of NF-κB. Accordingly, the site of NF-κB activation in the coronary bed in stable and unstable patients, its capacity to predict subsequent coronary artery events, and its effectiveness as a target for therapy are being prospectively analyzed. These experiments should begin to clarify the role of NF-κB in the progression of atherosclerosis, its role in plaque rupture, and most importantly, its clinical applicability.

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Documentation for the Sake of Documentation?
David Grant

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